DISSERTATION OF TEN SURGICAL CONDITIONS

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A dissertation of ten Surgical conditions presented as part of the requirements for the Degree of Master of Medicine (Surgery) of the University of Dar es Salaam



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INTRODUCTION

This dissertation consists of ten different surgical conditions divided into three sections:

Section I: Five General Surgical Conditions

Section II: Three Urological Conditions

Section III: Two Orthopaedic/Trauma Conditions

Except in a few instances, an attempt has been made to cover common surgical conditions met with at Muhimbili Medical Centre. Current concepts, opinions and methods of treatment have been presented in the discussions.

First of all I wish to thank Professor J.K. Shija my supervisor and teacher and my other teachers in surgery including Professor P.J. Phillip, Professor P.R. Hiza, Professor P.M. Sarungi, Dr. B.R. Kirei, Dr. Minja, Dr. S.M. Mlay, Dr. M.R. Aziz, Dr. S.N. Dutta and Dr. D.D. Charles, each for their immense contributions towards my training in surgery. Their guidance helped me to acquire the surgical knowledge I now have.

I thank the Muhimbili Medical Centre authorities for their permission to study the cases submitted in these dissertations.

I also thank the nursing staff of all surgical wards, ward 14 in particular, of the Muhimbili Medical Centre for their assistance and co-operation given to me and the excellent care they offered to the patients.

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CONTENTS

SUBJECT		PAGE
SECTION I: GENERAL SURGERY		
1.	Carcinoma of the Oesophagus	1
2.	Perforated Dudenal Ulcer	26
3.	Carcinoma of the Breast	52
4.	Non-toxic (Simple) Goitre	89
5.	Colo-rectal Carcinoma	117
SECTION II: UROLOGY		
6.	Upper Urinary Calculi	148
7.	Benign Prostatic Hypertrophy	178
8.	Elephantoid Scrotum	201
SECTION III: ORTHOPAEDIC/TRAUMA		
9.	Traumatic Intracranial Haemorrhage	219
10.	Fractures of the Femoral Shaft	247

DAR ES SALAAM MEDICAL

LIST OF ILLUSTRATIONS

Illustration number	Page
1	4
2	4
3	28
4	43
5	44
6	68
7	68
8	113
9	113
10	137
11	164
12	164
13	203
14	203
15	249
16	249

SECTION I

GENERAL SURGERY

CARCINOMA OF ESOPHAGUS

SUMMARY

Seventeen patients with carcinoma of esophagus are presented. One patient is presented in detail. Incidence, etiological factors, pathology, clinical manifestation and management of carcinoma of esophagus are discussed. A review of literature pointing out the difficulties of early diagnosis and treatment of carcinoma esophagus is presented.

INTRODUCTION

Incidence of carcinoma esophagus varies widely thoughout the world being notably high in China, Japan, Scotland, Russia and Scandinavian countries. In South Africa incidence is high especially among the Bantu.[1] Hiza[2] reported 10 adult patients of carcinoma esophagus admitted to Muhimbili Hospital in 1971. Most of the cases were referred from upcountry.

Carcinoma of esophagus is predominantly a disease of males (male to female ratio of 3:1).[1] It commonly occurs between the ages of 40 and 60 years. Carcinoma esophagus is a distressing disease with grave prognosis. Most cases are diagnosed only after esophageal obstruction has developed and the tumour has progressed beyond the anatomical limits of the organ and has involved the regional or distant lymph nodes. In spite of modern methods of treatment only 2 or 3 percent of patients with this disease survive for five years after diagnosis has been made.[3]

In this study seventeen patients of carcinoma esophagus managed by Surgical Firm three in Muhimbili Medical Centre during the period from 1st August 1980 to 8th May 1982 are presented. They were all male patients of age ranging between 29 to 74 years (table 1). Only 3 patients (17.28%) were Dar es Salaam residents. The other 14 patients were referred patients from upcountry as shown in table 4.

CASE REPORT

P.C., I.P. No.275003, a 38 year old male Prison Officer Mhehe by tribe from Iringa was admitted in the Surgical Ward 14 Muhimbili

Medical Centre on 29th December, 1981 with history of progressive dysphagia for 3 months. Initially the difficulty was with swallowing solids but progressively got worse so that at the time of admission even swallowing liquids was difficult. Two months after onset of dysphagia he started experiencing left side chest pain and cough which was initially dry but later became productive. One month prior to admission he started regurgitating the solid foods. He also noticed loss of weight.

He never had any major illness before and this was his first admission in hospital. His usual food was prepared from maize meal (Ugali) and eaten together with meat, fish, beans, cassava leaves, pampkin leaves and sometimes spinach. He was a heavy drinker of the various types of bear and a smoker of about 3 cigarrettes per day. There was no other member of his family who suffered from dysphagia or who had chronic cough.

On examination he was found to be moderately wasted but not pale, not dehydrated and not jaundiced. He had no lymphadenopathy. His temperature was 37°C, pulse 72/min. and blood pressure 120/80.

Mouth and tongue were normal. Abdominal and rectal examinations were essentially normal. Chest was symmetrical, respiratory rate was 18/min. and trachea was centrally placed. Air entry was reduced on the left side of chest and rhonchi were heard on the left side of chest. Cardiovascular and central nervous systems were normal.

Investigations done included full blood picture which was normal. Plain chest X-ray was also normal. Barium Swallow revealed a sharp edged irregular stenosing lession of about 8 cm long in the middle third of esophagus (Illustration 1). There was moderate proximal dilatation. Esophagoscopy revealed a fungating tumour about 26 cm from upper incisors and a biopsy was taken for histology which was reported as poorly differentiated squamous cell carcinoma of esophagus.

The patient was presented to the tumour board for discussion and palliative radiotherapy was recommended. At the end of 3000 rads, dysphagia was greatly relieved but the patient developed severe

productive cough especially after swallowing fluids. Gastrograffin study was requested and revealed left esophagobronchial fistula (Illustration 2). Decision to insert a Celestin tube was made and patient advised accordingly. However, the patient and his relatives refused to give consent for the operation despite explanation given to both the patient and the relatives and the patient was taken home against medical advice.

Rest of the presentation is as follows:

Table 1: Age incidence

Age range in years	No. of patients	Percentages
29 or less	1	5.76%
30 - 39	3	17.28%
40 - 49	3	17.28%
50 - 59	6	34.56%
60 - 69	3	17.28%
70 or more	1	5.76%

They were all male patients.

Table 2: Presenting symptoms as recorded at admission

Presenting symptom	No. of patients	Percentages
Progressive dysphagia	17	100%
Regurgitation	7	23.04%
Chest pain	10	57.64%
Cough	3	17.28%
Loss of weight	11	63.36%

None of the patients presented with cervical lymphadenopathy, jaundice or hepatomegaly.

Table 3: Duration of symptoms at the initial admission

Duration of Symptoms at presentation in months	No. of patients	Percentages
2 or less	4	23.04%
2 - 4	6	34.56%
4 - 6	5	28.80%
6 - 8	-	-
8 - 10	2	11.52%
10 or more	-	-

Most patients were referred from various regions as shown in table 4.

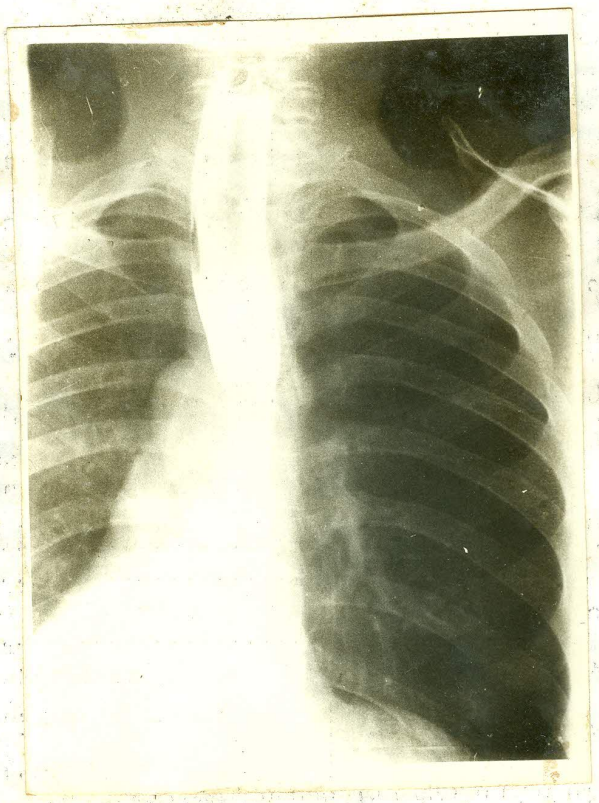


Illustration 1:
Barium swallow shows a stenosis in the middle third of the esophagus with moderate proximal dilatation.



Illustration 2:
Gstrografin swallow shows left oesophagobronchial fistula after palliative radiotherapy.

Table 4: Region of residence

Region of residence	No. of patients	Percentages
Dar es Salaam	3	17.28%
Morogoro	2	11.52%
Mbeya	2	11.52%
Mwanza	3	17.28%
Iringa	2	11.52%
Tanga	2	11.52%
Moshi	1	5.76%
Rufiji	1	5.76%
Lindi	1	7.76%

Table 5: History taken at initial admission included the following:

Positive history of	No. of patients	Percentages
Tobacco smoking	11	63.36%
Alcohol drinks	10	57.64%
Smoked fish eating	5	28.80%

Diagnostic investigations done were:

1. Radiological

   Barium Swallow was performed in only 16 patients and site of lesion as shown by barium swallow was as follows:

Table 6: Radilogical findings

Site	No. of patients	Percentages
Upper third	2	12.5%
Middle third	6	37.5%
Lower third	8	50.0%
Total	16	100%

2. Esophagoscopic examination was done in all the 17 patients and site of the lesion seen by esophagoscope and measured in centimetres from upper incisors was as follows:

Table 7: Esophagoscopic findings

Level of the lesion	No. of patients	Percentages
With 24 cm from upper incisors	2	11.8%
in 24 - 32 cm from r incisors	6	35.3%
in 32 - 40 cm from r incisors	9	52.9%

All biopsies taken on esophagoscopic examination were sent for histology. However only 15 had histology results two on second biopsy one on third biopsy the results were as follows:

Table 8: Histological pattern

Histology	No. of patients	Percentages
Squamours cell carcinoma	13	86.58%
Adenocarcinoma	2	13.32%

Table 9: Treatment was mainly palliative as is shown in table 9

Form of treatment	No. of patients	Results
Surgical:		
1. Curative resection	None	-
2. Palliative procedure:		
(a) Celestin tube insertion	2	One died post-operatively of espiration. The other was discharged able to swallow soft food.
(b) Feeding gastrostomy	3	One patient later received radiotherapy and dysphagia paliated. Gastrostomy was closed and patient discharged.
		Two died before radiotherapy was started.
(c) Feeding jejunostomy	None	-
Irradiation:		
1. Redical curative dose "5000 rads"	2	Both showed good responses. The 2 patients with discharged able to swallow solids.
2. Palliative dose "Range 3000 - 4500 rads"	11	Eight had good initial response and were able to swallow solids before discharge.
		Two had moderate response and were able to swallow fluids.
		One had poor response he had difficulty swallowing fluids at the end of 3000 rads.

DISCUSSION

Etiology:

The cause of carcinoma of esophagus is unknown. However, the known etiological factors include: Dietary, Alcoholic and smoking habits; gastrointestinal diseases like chronic esophagitis, Plummer-Vinson syndrome, stricture of the esophagus, pharyngo-esophageal diverticulum, hiatus hernia, achalasia and after gastric surgery.[4, 5, 6, 7, 8]

Experimental work has shown that polynuclear hydrocarbons like Bensopyrene are carcinogenic.[4] It has been found that melted fat which drips on the hot coals undergoes pyrolysis releasing hydrocarbons which are deposited on the meat as smoke rises. Oral administration of these hydrocarbons to mice, rats and guinea pigs enhances cancers of oral mucosa, esophagus, stomach and small intestine.[4] Thermal irritation of the esophageal mucosa by ingestion of hot food or tea predisposes to carcinoma esophagus.[5] Dimethylnitrosamine has been identified in the solanum incanum fruit which is used to curdle milk by Bantu people of Transkei and esophageal cancer is very prevalent in the Bantu of Transkei.[6]

Heavy alcohol intake particularly whiskey is associated with increased incidence of carcinoma esophagus.[7] The risk of a heavy whiskey drinker is 25 times that for a non-drinker and for a heavy bear drinker about 10 times that of a non-drinker.[7] However, it is not known whether alcohol acts as an independent carcinogen. Or whether it merely promotes the effect of tobacco.[7]

Diseases associated with stagnation of food in the esophagus may allow breakdown products after bacterial and enzymatic action to serve as chronic irritants to the esophageal mucosa and cause cancer.[8] Achalasia of the Cardia is associated with cancer of esophagus usually of squamous type occuring in younger patients and sited above the level of the obstruction. They usually remain asymptomatic for sometime and present with advanced disease. Association between esophageal carcinoma and esophageal hernia has

been reported.[8] However, Mc Keown[9] reported that neither esophageal hernia nor esophagitis predispose to cancer esophagus.

Plummer-Vinson (Paterson - Kelly) syndrome and Malabsorption syndrome have been associated with carcinoma esophagus. It is though that iron and other nutritional deficiencies alter epithelium to become more susceptible to carcinoma.[8] Wynder at al[7] found that onset of carconoma of esophagus was preceded by Paterson - Kelly syndrome in 38 percent of patients.

In this study 5 patients gave history of eating smoked fish, 10 patients gave history of drinking alcohol and 11 patients were tobacco smokers as shown in table 5.

Pathology:

After an esophageal tumour growth has started, there is expansion into the lumen, infiltration of the wall or penetration to the surrounding tissue. Macroscopically three types are recognized: fungating type in which the tumour grows toward the lumen faster than toward the wall; ulcerative type in which the overlying mucosa undergoes necrosis and ulcerates; infiltrative type in which the tumour penetrates the muscularis and thickens the wall, causing sclerosis and narrowing of the esophageal lumen.

The esophagus, is a fibromuscular tube about 25 cm long extending from cricopharyngeal sphincter to the cardia of the stomach, and is considered by surgeons as consisting of three segments; upper, middle and lower third. Site of tumour can be in any of the three segments.

In this study 2 patients (11.8%) had the tumour in the upper third of esophagus, 6 patients (35.3%) had the tumour in the middle third of esophagus and 9 patients (52.9%) had the tumour in the lower third of esophagus as shown in table 7.

Mc Keown[9] reporting on 294 cases treated by excision found segmental distribution as: upper third 8 cases, middle third 108 and lower third 178 cases. Thus because of small number of patients, the finding of more patients with tumour in the lower third as found in this study (table 7), is not significant.

Histological types of malignant tumours of the esophagus include: Squamous cell carcinoma, is the commonest; adenocarcinoma, involving esophagogastric junction and are usually due to the upward extension of primary gastric carcinoma; sacromas mostly leiomysacromas are rare extramucosal tumours which may ulcerate and calcify; carcinosarcoma is uncommon and associated with better prognosis; primary melanomas rarely arise from esophagus although melanoblasts have never been demonstrated in human esophagus.[1] Relative frequency of squamous cell carcinoma is upper third 17 percent, middle third 50 percent and lower third 33 percent.[3]

Carcinoma esophagus is usually a single tumour but accasionally there may be two or more primary tumours in the esophagus. Satellite metastasis may occur, but when two lesions are far apart the possibility of multiple primary tumours must be considered.

In this study histology results of 15 patients were obtained. 13 patients (86.68%) had squamous cell carcinoma and 2 patients (13.32%) had adenocarcinoma as shown in table 8.

Tumour spread can occur by direct, lymphatic and haematogenous routes.[10] Direct submucous spread by burrowing the mucosa occurs both longitudinally and circumferentially and produces a long tortuous stricture. The tendency to submucous permeation is more marked in squamous growth than in adenocarcinoma. Thus safe marginal operation would have to be extended to at least 6 cm for resection of esophageal growths. By direct transverse spread through esophageal wall the tumour invades the surrounding structures or may perforate and lead to mediastinitis. The important structures that may be invaded include; trachea, bronchi, lungs and rarely aorta causing fatal bleeding.

Lymphatic spread occurs by both permeation and embolization. Submucosal lymphatic spread may lead to satellite nodules away from the apparent margins of the tumours. This calls for a very wide excision of the growth so that a recurrence at the anastomosis may

be prevented. Lymphatic spread to the surrounding nodes occurs. Cervical esophageal tumour spreads through lymphatic vessels to cervical nodes particularly the anterior jugular and supraclavicular nodes. Tumours of thoracic esophagus spread early to para-esophageal tracheo-bronchial nodes and occasionally subdiaphragmatic nodes. Tumours of abdominal esophagus commonly spread to lymph nodes along the lesser carvature of the stomach and hence to splenic and coeliac glands. Haematogeneus spread to bones or liver is relatively late. Considering the extent of lesion esophageal tumours can be categorised by TNM classification as follows: T_1 is primary lesion 5 cm or less long with circumference incompletely involved, T_2 is primary lesion of any size with extra-esophageal spread; No when nodes are not palpable with cervical primary or intrathoracic nodes biopsy negative with thoracic primary N_1 when ipsilateral nodes are positive, N_2 contralateral nodes are positive and in N_3 when nodes are positive and fixed; Mo when there is no evidence of distant metastasis and M_1 when distant metastasis are present.[10]

Clinical Manifestation

Late presentation is common partly because carcinoma of esophagus is slow growing tumour with doubling time of about 164 days[11] and the uninvolved esophageal wall is so distensible that unless a considerable proportion of the circumference of the esophagus is involved, obstructive symptoms usually do not develop. It is possible for the adult to swallow well masticated food through an esophageal lumen of 5 cm (French 15) in diameter.[12]

Patient may present with local symptoms like dysphagia; secondary deposits like enlarged neck nodes, hepatomegaly or jaundice; general manifestations of malignant disease like loss of weight, anorexia and anaemia.[12]

Dysphagia is the commonest symptom of carcinoma esophagus. Although sometimes, owing to sloughing of a portion of the growth,

subsiding edema at the narrowed segment or disimpaction of ingestants, for a time, swallowing may become easier, as a rule the dysphagia is progressive.

At first, a large bolus of poorly masticated meat will not pass, then soft food and then even liquids including saliva become difficult to swallow. Dysphagia is present in 96 percent of patients during their entire course of hospitalization.[12] Occasionally there are patients who have never complained of dysphagia simply because carcinoma has not involved the entire circumference of the esophagus before some fatal complication such as fistula to the aorta or trachea develops. Only 40 percent of patients report within 3 months of developing dysphagia.[3] Most patients present when they cannot swallow fluids. By this time the growth has encircled at least $\frac{3}{4}$ of the lumen of the esophagus and loss of weight is obvious.[3]

Palmer[12] found pain to be the second most common symptom present in 15 percent as an initial complaint and 45 percent throughout the hospital course. Initially pain is usually intermittent in the substernal region occuring just after swallowing. It is due to esophagospasm at a level higher than the tumour. Second type of pain is a constant, boring, dull pain deep in the chest due to periesophageal inflammation or invasion of tumour. Carcinoma of the lower third of the esophagus can cause pain not only in the epigastric region but also in the throat depending upon the extent of para-esophageal infiltration. In other words, the site of pain does not necessarily indicate the location of the primary lesion.

Regurgitation occurs in about 18 percent of patients[12] Reflex regurgitation occurs in response to overdistension of the proximal part of the esophagus. The regurgitated material is alkaline, mixed with saliva or streaks of blood the amount is never large unless a tumour develops in an esophagus with achalasia.

Respiratory symptoms are due to aspiration of food or direct involvement of respiratory passages. Severe productive cough is a common complaint and occasionally pleuritic pain, fever and dyspnea are present.

Anorexia is sometimes a feature of growths situated in the lower part of the esophagus.

Other manifestations include; burning sensation in the pharynx as if a foreign body were present, occurs with tumour in the cervical portion of esophagus, hoarseness of voice due to local metastasis and destruction of the recurrent laryngeal nerve, metastatic lymph node mass in the neck may be the only sign. There may be anaemia due to gradual oozing of blood from the tumour.

In this study progressive dysphagia was a presenting symptom in all the 17 patients (100%), 11 (63.36%) complained of loss of weight, chest pain, was a presenting symptom in 10 patients (57.64%). Regurgitation was a presenting symptom in 7 patients (23.04%) and cough was a presenting symptom in 3 patients (17.28%) as shown in table 2.

At time of presentation the duration of symptoms in 11 patients (63.36%) varied between 2 and 6 months. 4 patients (23.04%) had symptoms of less than 2 months duration and 2 patients (11.52%) presented with symptoms of 8 to 10 months duration (table 3).

Complications of esophageal cancer include: Obstruction by bolus of meal or other foreign body and may need removal through esophagoscope; fistula formation occurs when tumour penetrates deeply into surrounding structures and becomes necrotic; choking and productive cough after swallowing occurs and patient usually dies within a short time; mediastinitis with high fever may develop when ulcerative tumour becomes infected; cardiovascular complications include thrombophlebitis of superficial and deep veins, superior venocaval syndrome due to lymph node involvement in the mediastinum and metastasis to the pericardium with or without harassment of the heart; paralysis of recurrent laryngeal nerve leading to

hoarseness and paralysis of the superior laryngeal nerve causing anaesthesia of the laryngeal mucosa and encouraging aspiration of fluid into the trachea is also seen and the patient's condition rapidly runs downhill. Cerebral metastasis causing hemiplegia and other neurological finding is rare.

Differential diagnosis of dysphegia in adults include: Intraluminal-foreign body; in the wall, esophageal stricture (caustic or inflammatory), achalasia, esophageal web (Plummer-Vinson syndrome), pharyngeal pouch; outside the wall; enlarged lymph nodes (malignant or reticuloses), aortic aneurysm, bronchial carcinoma or retrosternal goitre. General causes include; myasthenia gravis, bulbar palsy and hysteria.

Investigations

Diagnosis may be given by history of swallowed caustic liquid in the past. A previous reflux esophagitis suggests peptic stricture. Patients with achalasia tend to be young and the history is often long, usually without loss of weight while malignant stricture has a short history, usually in elderly people and is associated with severe weight loss.

Special investigation include the following: Radiography; plain chest X-ray is usually not informative but occasionally tumour may invade the mediastinum causing a soft tissue shadow or have lymphatic spread to the lungs. Barium swallow and fluoroscopy; radiologist should prepare two types of barium mixture, thick barium of two parts of powdered barium-sulfate and one part of water which leaves a thin coating on the esophageal mucosa and semi-liquid barium, one part of powdered barium sulfate and one part of water to be used when esophageal transit is slow and empting is poor. Sometimes an iodine oil or water soluble contrast medium is used when perforation or fistula of the esophagus is suspected. The films are exposed in the anteroposterior, lateral and oblique views with the patient in the recumbent position. It is important to

expose X-ray films in different views because in the early stage, the neoplasm does not involve the entire circumference of esophageal wall and may be demonstrated only in a certain view. In the fungating type there are irregular images on one wall and destruction of mucosal folds. The opposite wall may be smooth. The ulceration type central excavation of a partly intraluminal mass is the characteristic sign. The niche is rigid and unchanged by passage of barium bolus. In the infiltrating type the tumour grows mainly under the mucosa. There is gradual narrowing of the seophagus without clear transition to the adjacent segments. The contour is smooth but the wall is rigid and is not affected by the barium bolus. The more advanced lesions encircle the esophageal lumen completely. They cause narrowing or complete stenosis of the esophagus. The upper and lower border of the tumour may overhang, producing a sharp angle with the esophageal wall. Unlike the more chronic benign obstructing lesions, carcinoma esophagus usually is not associated with marked proximal dilatation of esophagus.[17]

Radiological diagnosis of carcinoma of the esophagus may be difficult because cicatricial esophagitis and early achalasia may cause similar radiological changes.

Varices at the lower end of esophagus may simulate the broad filling defects of fungating carcinoma. However, the normal distensibility and normal peristalsis of the esophagus would help the diagnosis.

Esophagoscopic examination provides a view of the lesion and opportunity of taking a biopsy specimen and also provides a lumen to facilitate insertion of a Levin tube or a polyethylene tube past the tumour. Multiple biopsy specimens are taken. The difficulties in obtaining positive specimens are: Narrowness of the lumen preventing access of the biopsy forceps to the growth;

deep position of the tumour with overlying mucosa; exfoliative eslophagitis in the proximal portion of the esophagus masking the exact location of the lesion. Perforation rate is reasonably low. Brick[13] reported 2 perforations but no mortality during 1000 esophagoscopies.

Cytologic examination is particularly helpful when biopsy is not practical. After an overnight fast, patient is put in sitting position and a number 18 French Levin tube is passed through the mouth to the esophagus beyond obstruction into the cardia if possible. A 50 ml syringe is attached to the tube. The patient is asked to swallow 100 ml of normal saline while gentle aspiration is maintained with the syringe. The washing fluid is then injected back into the esophagus and is respirated. The tube is then withdrawan for several centimeters and the whole process is repeated. The fluid is collected in a few big centrifuge tubes. The fluid is centrifuged immediately and sediment is smeared on 5 to 6 slides which are put in fixation solution containing 95 percent alcohol and sent to laboratory where Papanicollaon stain is carried out. Johnson et al [14] reported that among 148 cases of proven esophageal carcinoma, cytology was positive in 85 cases and negative in 63 cases, while biopsy was positive in 85 cases. In these 85 cases both biopsy and cytology were positive.

Esophageal washings obtained during esophagoscopy are also good for cytologic examination 10 to 20 ml. of normal sline are injected through the esophagoscope and espirated. The process is repeated until about 100 ml. of aspirate is collected. Another method consists of immediate raising of esophagoscope with 100 to 150 ml. of saline. The rinsing fluid is collected for examination. However, yield by the esophagoscope wash method is inferior to that obtained by the esophageal lavage.

Bronchoscopy should be done routinely and whenever there is cough with expectoration of large quantities of purulent sputum which suggests

involvement of the bronchial tree and possibly a fistula.

Surgical exploration is occasionally required to establish presence or absence of malignant growth when results of diagnostic studies are not definitive.

In this study investigation by barium swallow was done in 16 patients and the findings are shown in table 6. All the 17 patients had esophagoscopy done and the level of the lesion from upper incisors determined. The findings are shown in table 7. At the time of esophagoscopy biopsies of the tumour were taken and sent for histology. The histology results were obtained for 15 patients and are shown in table 8.

Treatment

There are only two methods of treatment which offer benefit to patients with cancer of esophagus, surgical resection and irradiation and the treatment can be curative or palliative.

Aided by modern anaesthesia, use of relaxants and tracheal or bronchial intubation, intrathoracic surgery has gained in safety and precision. It involves excision and reconstruction.

Preparation for operation should aim at improving nutritional state, rehydration, correction of anaemia and assessing the cardiopulmonary state. Good nutrition is important for healing of the anastomosis and incisional wounds. If patient can swallow, minced food should be given with high protein content aiming at giving 3000 calories daily. Supplements like complan, high vitamin plus 500 mg of ascorbic acid per day for two weeks pre-operative. Bailey and Love[3] recommend hourly feeds as much as the patient can take, of fortified milk, 2 eggs, 2 ounces dried milk and 1 ounce of butter to 2 pints of milk.

If the patient is unable to swallow, esophageal lavage and nasogastric tube feeding should be attempted. If it is impossible, intravenous therapy is preferable to the establishment of feeding gastrostomy or jejunostomy though the latter procedures have their advocates.[15, 16]

Intravenous aminoacid preparations like aminosol or laevorotatory (Vlamin) are in use and have superseded use of blood plasma to supply protein. Intravenous fat preparation like intralipid to provide calories and intravenous carbohydrate preparations like sorbitol are in use.

Presence of dehydration may cause haemoglobin levels to be high. After rehydration, a truer estimate is obtained and anaemia corrected by blood transfusion and iron suppliments.

Smoking is forbidden, breathing exercises and postural drainage are started to remove bronchial secretions and rehearse in case of need post-operatively. Sputum culture and appropriate antibiotic will prevent post-operative chest infection.

Cardiac irregularities should be treated with digitalis which prevents post-operative atrial fibrillation.

Patient considered clinically suitable and whose tumour is within the scope of total excision is selected for surgery. However the type of surgery, route of approach and type of replacement depend largely on the level at which the tumour is situated. Patients with lessions is the lower third, 5 cm or less in length and the esophageal circumference incompletely involved are most suited for curative surgery.

The objectives of surgery for growth in the lower ⅓ are radical and curative surgery. The disposition of the lesion enables a block dissection of the growth, its lymphatic field and all adjacent structures to be carried out and even left lobe of the liver may be included in the dissection.[1] Incision is the classical left thoracoabdominal along the 7th rib with the patient lying in the right lateral position. Preliminary exploration of the abdomen through the medial half of the abdominal wound will enable the unresectable cases to be excluded without the added dangers of thoracotomy. This approach provides ideal access for growth in the lower ⅓ below the diaphragm. Wide resection of the distal esophagus and proximal part of the stomach along with spleen, omentum and

regional nodes is done. When local extension demands it, portions of pancreas, diaphragm, transverse mesocolon and colon can be included in the enbloc resection.[1] Esophagogastric anastomosis is performed after mobilizing the second part of duodenum (Kocher's Manoeuvre) to reduce tension at the anastomosis. Since esophagogastrectomy result in bilateral vagotomy and gastroesophageal sphincter mechanism resection, pyloroplasty is done to avoid gastric stasis and esophageal reflux. When the growth is sited at the gastro-esophageal junction, total gastrectomy is most suitable in order to have a complete clearance of the gastric lymphatic glands. Continuity is restored by esophago-jejunostomy employing a Roux - en - Y anastomosis or the jejunal loop with jejunojejunal anastonosis. Although jejunal reconstruction limits the volume of meals taken, biliary regurgitation is rare.

Middle third esophagus lies behind the vital structures of the hilum of the lung and is least easy of access. For growths in this situation a two stage technique is the operation of choice. This procedure is equally appropriate for growths in the lower third above the diaphragmatic hiatus. In this technique the stomach is mobilized through an upper left paramedian incision with great care to preserve its blood supply from the gastroepiploic arch and right gastric artery. The left gastric artery is secured close to its origin and divided between ligatures clearance of peritoneal reflection and the phreno-esophageal ligaments is essential to facilitate the subsequent transfer of the stomach through the diaphragmatic hiatus in the second stage of operation. Mobilization of the second part of duodenum (Kocher's Manoeuvre) is also desirable. If the pyloric canal is narrow, pyloromyotomy as is done in Ramstedt's operation without opening the mucosa is desirable to facilitate gastric drainage since esophageal resection will cause complete truncal vagotomy.

The second stage consists of exposure of the esophagus through a right thoracotomy in the 6th interspace. After esophageal

mobilization the stomach is pulled through the diaphragmatic hiatus into the right chest, so that the affected segment of the esophagus together with the proximal part of the stomach can be excised. Gastro-esophageal anastomosis is made end to side, the opening in the stomach being crescentic in shape, in the front wall of the stomach. Insertion of interrupted vertical mattress silk sutures to produce perfect mucosal apposition is essential and the knots should preferably be tied in the lumen. An outer layer of continuous catgut is desirable and the stomach should be anchored to the pleura to diminish tension at the anastomosis.[9]

However, high middle third growths and growths involving the supra-aortic segment of the upper third resection requires a three stage procedure. The first two stages are as above (Lewis - Tanner procedure). The stomach is however, mobilized with even greater care to preserve an adequate blood supply right up to the fundus. Extensive mobilization of the duodenum and head of the pancreas is essential so that the pylorus can reach the midline and ultimately lie between the crura of the diaphragm when the stomach is transposed into the right chest in the second phase of the operation. Right thoracotomy provides an unobstructed access to the entire thoracic esophagus after the vena azygus has been divided between ligatures. It is important to mobilize the anterior surface of the esophagus from the trachea very completely, not only to preserve the left recurrent nerve but more especially to prevent tracheal damage by kinking when the esophagus is drawn through into the neck in the third stage of the operation.

Exposure of the esophagus in the third stage of the procedure can be carried out on either side of the neck. The left cervical approach carries greater risks because of danger of damage to the thoracic duct or the left recurrent laryngeal nerve. Exposure on the right side of the neck is easier and safer, since after thoracic mobilization the esophagus tends to move to the right.[9]

After exposure of the cervical esophagus between the right lobe of the thyroid and the carotid sheath the entire esophagus containing the tumour is pulled into the nect. The gastro-esophageal junction is stapled and oversewn and the cervical esophagus can be anastomosed without tention to the dome of the gastric fundus. A circular area of about 3 cm in diameter is removed from the fundus of the stomach where the esophagus is anastomosed.[3]

Nakayama[17] recommends a three stage procedure in association with radiotherapy. The first stage consists of laparotomy and removal of coeliac and paraesophageal lymph nodes, ligation of vasa brevia and the establishment of the tumour with a dose of 2000 to 3000 rad, total esophagectomy is carried out through a right thoracotomy and a left sided cervical esophageal fistula is then established. The patient is fed orally with a tube connecting the cervical fistula with the gastrostomy. If no recurrence takes place in six months, the stomach is brought up in front of the sternum and anastomosed to the cervical esophagus. No deaths were reported by Nakayama in 48 cases to treated.[15]

Problem of post operative anastomotic leakage particularly in the thoracic cavity, poses a great danger and total esophagectomy has been carried out to avoid anastomosis in the depths of thoracic cavity. Method used to bridge the gap between the cervical esophagus and abdominal viscera include the following: Anterior presternal reconstruction using a Roux-en-Y jejunal segment brought up in front of the sternum in a subcutaneous tunnel. However, failure of epithelialization between the high columnar epithelium of the jejunum and squamous epithelium of the upper esophagus is associated with stomal leaks and fistulae formation and high jejunal replacement is better avoided. Anterior presternal replacement using left colon or right colon have been recorded.[18] The incidence of cervical leaking and stricutre formation are common after these types of replacement. However, the gastric replacement has shown to be relatively safe. Provided adequate mobilization and

freeing of the phreno-esophageal ligaments are done, the stomach will reach into the neck with ease and anastomosis performed without tension. Epithelial union takes place readly between the squamous epithelium of the cervical esophagus and the mucosa of the gastric fundus. Such union does not take place so easily with jejunal replacement.[18] The disadvantage of utilizing the stomach as a substitute for the esophagus is reflux esophagitis above the anastomotic line. An intrathoracic polythene prosthesis has been used but, in general, it has not been found to be satisfactory.[3]

When at operation the growth is found to be irremovable and the growth is situated at the cardia, the esophagus can be divided above the growth, the lower end closed and esophago-jejunostomy-Roux-en-Y perfomed above the obstruction. If the tumour is higher in the thoracic esophagus, the previously mobilised stomach can be anastomosed to the esophagus above to palliate the dysphagia.[3]

When inoperability is decided upon at a clinical examination, simple palliative procedure assumes an added importance. The introduction of a tube not only allows the patient to take sufficient nourishment by mouth, but relieves him of dysphagia. Celestin tube is safer and consists of a 25 cm shaft carrying an oval barrel-shaped tunnel about the size of a bolus.[19] It is pulled into position by a detachable pilot - bougie. The latter is navigated through the growth under direct vision, no dilatation being required. The tip of the bougie is recovered in the stomach via high gastrotomy. The endo-esophageal tube is then plugged on securely to the bougie and drawn down to position with the tunnel lying above the tumour. It is then tethered to the lesser curve and any excess cut off. Post-operatively the patient is allowed a fairly free diet provided it is chewed thoroughly and frequently washed down with aerated water. The tube may be used in patients receiving high-voltage irradiation or as a pre-operative measure and an important use for prosthetic treatment is to occlude an

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existing esophagotracheal or esophagobronchial fistula to protect the lungs against swallowed water. In this study esophageal intubation was advised for the patient who developed esophago-bronchial fistula, (Illustration 2) but patient refused.

Occasionally it is difficult to insert the esophageal prosthesis, esophagobrancheal fistula seems eminent or the patient has pulmonary problems secondary to aspiration. In such cases cervical esophagostomy may then be performed to divert saliva and esophageal secretions and colostomy bag applied. If the patient is very debilitated, feeding gastrostomy is done and the distal end of the colostomy bag is tied to the gastrostomy tube. The insertion of a gastrostomy tube for nutrition in an individual with an obstructed esophagus does not palliate the inability to swallow saliva and does not extend survival substantially.

Radiotherapy is the treatment of choice for the radio-sensitive squamous cell carcinoma in the upper third of the esophagus and most of the cases with growths in the middle third of esophagus respond to irradiation. However, the collumnar celled adenocarcinoma of the lower third is radio-resistant and irradiation of the liver cannot be avoided, and this makes the treatment dangerous.[3]

Radiation can be radical and curative or palliative. Beatty et al[10] defined radical radiotherapy as radiation of 4000 rads or more in less than 18 fractions, or 4500 rads or more in less than 23 fractions, or 5000 rads or more within 3 months. Palliative radiotherapy was defined as radiation of less than 4000 rads in over 17 fractions or less than 4,500 rads in over 22 fractions. in their study they found that the most important factor in identifying patients who responded to treatment was the size of the primary lesion. Patients whose lesions were 5 cm or less long or incompletely encircled the lumen all responded to treatment Patients with larger primary lesions had a lower incidence of response. All patients with metastatic disease did not respond to radical radiotherapy. They recommended that all patients with carcinoma of

the esophagus who do not have distant metastasis should be treated radically and although most will not be cured, many will respond by leaving prolonged survival. However, patients with metastatic disease should not receive radical treatment of the primary. They also recommended the use of 5000 rads in 20 fractions over 4 weeks using supervoltage radiation through a port of 100 - 140 cm^2 as the optimum radiation therapy for carcinoma of the esophagus.[10]

In this study all patients received palliative radiotherapy alone and most of them showed good immediate response. Eight patients were able to swallow solids at the end of palliative radiotherapy course of about 3000 rads. Two patients had celestin intubation done one of the two patients aspirated and died postoperatively the other was well palliated and was able to swallow soft food at the time of discharge. Forms of treatment carried out in this study are shown in table 9.

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PERFORATED DUODENAL ULCER

SUMMARY

Thirteen patients with perforated duodenal ulcer who were managed initially by simple plication are presented. One of the patients is presented in detail. The incidence, etiological factors, pathogenesis, clinical features and management of acute perforated duodenal ulcer are discussed. A review of the literature pointing out the gradual change over the years in the mode of treatment of perforated duodenal ulcer from a conservative method of simple suture plication to immediate definitive surgery is presented.

INTRODUCTION

Perforation complicates about 10 percent of all peptic ulcers.[1] 90 percent of all perforations occur in duodenal ulcer.[1] The highest incidence is between 45 and 55 years with sex incidence ratio of 19 men to 1 woman.[2] The perforation is usually on the anterior part of the duodenal bulb.

In 80 percent of cases there is a history of peptic ulceration.[2] In 20 percent there is no such history.[2] The incidence is high in alcoholics and in those who take large amounts of acetyl-salicylic acid, phenylbutazone and prednisone.

Peptic ulcer is a world wide disease. The incidence varies from place to place and from time to time. Once considered an un-African disease, it has been found to occur with equal frequency. Badoe[3] recorded 54 cases of perforation between 1965 to 1971 in Accra. Analysing cases of surgical admissions to Muhimbili Hospital Dar es Salaam, Hiza[4] recorded 40 peptic ulcer cases in 1971 with 6 patients admitted as acute perforations.

In this study thirteen patients of perforated duodenal ulcer who were managed by surgical firm three in Muhimbili Medical Centre during the period from 1st August 1980 to 8th May 1982 are presented. The youngest patient was 28 years old male the oldest was 75 year old female.

10 patients (77%) were males and 3 patients (23%) were females (table 1). Christian patients were 3 (23%) and the remaining 10 (77%) were Moslems (table 2). However, the observation on the incidence in relation to religion may not be significant until the population and the religious habits of the people in Dar es Salaam have been studied.

CASE REPORT

M.I., I.P. No.254055, a 37 years old male Post-master, Mhehe by tribe, was admitted in surgical ward 14 of Muhimbili Medical Centre on 19th June, 1981 with the history of acute upper abdominal pain spreading to the back and later to the right iliac fossa for one day. Three days prior to admission he had experienced late morning abdominal pain which he had related to hunger and had passed black tarry stools. He denied any previous history of haematemesis nor waking up at night because of abdominal pain. He also denied drug ingestion but was a drinker of various types of alcohol and occasional cigarette smoker. He was once admitted and treated for typhoid in 1970.

On examination he was ill-looking, mildly dehydrated with a temperature of 37°C, the pulse rate was 100 per minute regular and of good valume, blood pressure was 140/90 and respiratory rate of 18 per minute. He was neither pale nor jaundiced.

The abdomen was generally distended and tender with guarding and rebound tenderness present.

Hepatic dullness was reduced. There was no shifting dullness and the bowel sounds were infrequent. Digital rectal examination revealed a little dark brown stools as seen on the glove.

Auscultatory examinations of respiratory and cardiovascular systems were normal. A clinical diagnosis of perforated duodenal ulcer with generalized peritonitis was made.

An intravenous infustion of normal saline was started and nasogastric tube inserted. The patient was sent for urgent X-ray abdomen (supine and erect) and chest (erect) were specifically asked for. X-ray (Illustration 3) clearly showed gas under the diaphragm.

The patient was prepared for emergency laparotomy and sent to theat
In theatre, under general amaesthesia the patient was cleaned, draped an

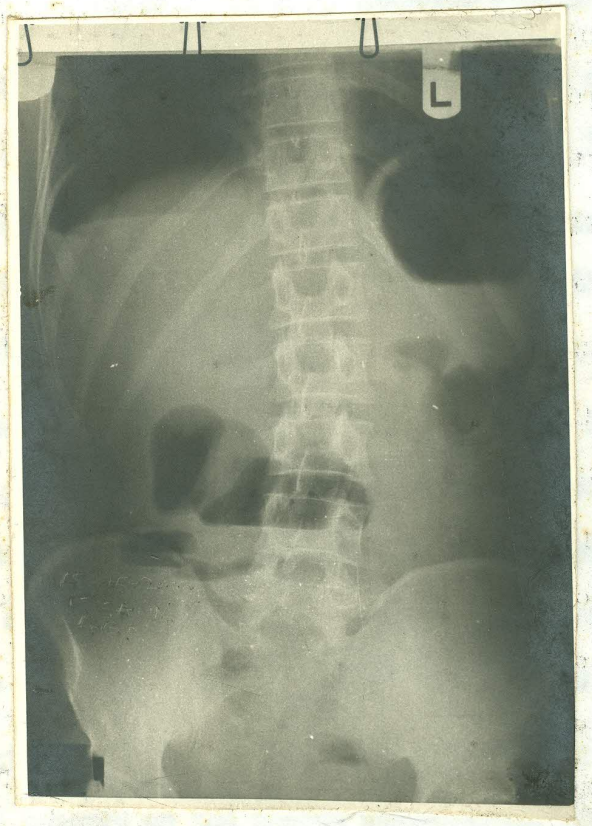


Illustration 3:
Shows gas under the diaphragm following acute perforation of duodenal ulcer.

a laparotomy performed through an upper middline incision. The peritoneal cavity contained a large amount of turbid fluid of about 300 mls which was sucked out. Generally the peritoneum looked inflammed. A perforated ulcer 1 cm large was seen on the anterior of the first part of duodenum with no scarring around it.

The perforation was closed with four sutures of interrupted number 0 silk and a patch of omentum stiched over it.

Peritoneal lavage was done with warm normal saline and after examining other organs the abdomen was closed in three layers with a drain in the Morison's pouch.

Post-operatively the following were ordered: Intravenous crystalline penicillin 2 mega units 6 hourly, chloraphenical 500 mgs 6 hourly; intramuscular pethidine 100 mgs 6 hourly; half hourly nasogastric suction and observation of blood pressure, pulse and respiration. 4 hourly temperature observation was also ordered.

This patient had fairly good recovery. The drain having stopped draining was removed on the second post-operative day. Intravenous drip and nasogastric tube were removed on the fifth day after the bowel sound appeared and the patient was allowed to take oral feeds. Stitches were removed on the seventh day and the patient discharged on antacids to come to surgical outpatient clinic after three weeks for follow-up. He was kept on two tablets of magnesium trisilicate for the first two months and he was also advised to take regular meals. The patient was cooperative and stopped drinking alcohol as well as smoking. He resumed his work. Seen three months later the patient complained of epigastric discomfort especially in the late morning hours. He said he got relieved by taking milk and occasionally magnesium trisillicate.

Rest of the presentation is as follows:

Table 1: Age and Sex incidence

Age range	Sex	No. of patients
20 - 29 years	M F	2 -
30 - 39 years	M F	4 -
40 - 49 years	M F	1 -
50 - 59 years	M F	2 1
60 - 69 years	M F	1 1
70 or more years	M F	1 1

Table 2: Incidence according to religion

Religion	No. of patients	Percentages
Christian	3	23%
Islam	10	77%

Table 3: Presenting Symptoms

Presenting symptoms	No. of patients	Percentages
Abdominal pain	13	100%
Vomiting	9	69.2%
Constipation	7	53.8%
Fever	3	23%
Haematemesis or Melaena	0	-

Table 4: Duration of perforation symptoms at admission

Duration of perforation Symptoms at admission (hrs)	No. of patients	Percentages
12 hours or less	2	15.4%
12 to 24 hours	1	77%
24 to 36 hours	2	15.4%
36 to 48 hours	-	-
48 to 60 hours	3	23%
60 to 72 hours	-	-
72 hours or more	5	38.5%

Table 5: Some associated precipitating factors

Positive pre-perforation history of	No. of patients	Percentages
Peptic ulcer disease	6	46.1%
Drug ingestion	2	15.4%
Alcohol intake	2	15.4%
Smoking	3	23%

Table 6: Findings indicative of perforation

Main findings indicative of perforation	No. of patients	Percentages
Tender and rigid abdomen	13	100%
Presence of pneumoperitoneum (X-rays not done in 3 patients)	10	77%

Table 7: Operative findings

Operative findings	No. of patients	Percentages
Anterior duodenal perforation	13	100%
Peritonitis	9	69.2%
Bilious fluid	6	46.1%
Purulent exudate	7	53.8%

Table 8: Operative management done

Operative management done	No. of patients	Percentages
Simple suture without omental Patch	3	23%
Simple suture with omental Patch	9	69.2%
Peritoneal lavage	7	53.8%
Peritoneal drainage	13	100%

Table 9: Post-operative complications

Complication	No. of patients	Percentages
Mortality	4	30.7%
Wound infection	7	53.8%
Intra-abdominal abscess	1	7.7%
Thrombo-embolism	1	7.7%

DISCUSSION

Aetiology and Pathogenesis

In the majority of cases the cause of perforated duodenal ulcer is obscure. However, there are some known aetiological factors:

(a) Drugs and alcohol intake

Various drugs like salicylates, butazolidine, reserpine, ACTH, steroids, caffeine and histamine are known to cause ulceration by one or more of the following ulcerogenic properties; stimulation of gastric secretion, mucosal irritation and lowered resistance of gastroduodenal mucosa.

Fentress[5] reported three arthritic patients developing perforation on prednisolone therapy and Shields et al[6] reported the first two cases of perforation of peptic ulcer on Butazolidine therapy.

(b) Severe psychological stress

A definite rise in the incidence of perforation of peptic ulcer during air raids in Britain between September 1940 and May 1941 was shown by Spicer et al[7]

(c) Excessive gastric acid secretion

Duodenal ulcer is mainly a disease of excessive acid secretion. This is pupported by the following observations:

1. Duodenal ulcer patients have been found to have a higher mean basal acid output (BAO) and maximal acid output (MAO) than normal controls.
2. Stimulation of gastric acid secretion in experimental dogs is followed by duodenal ulceration.
3. Constant infusion of acid gastric juice in the duodenum is followed by duodenal ulceration.
4. Hyperacidit in gastric outlet zone in dogs by diversion of alkaline pancreatic secretion to another gut (Man-Williamson preparation) is followed by duodenal ulceration.
5. Temporary reduction of gastric acidity by buffers alleviatoms of duodenal ulcer and healing of the ulcer follows permanent reduction of acidity by surgical means.[8]

Factors responsible for gastric hyperacidity include the following:

1. Excessive neural stimulation of gstric secretion

This is based on the fact that complete vagotomy is followed by reduction in gastric hypersecretion. However, this does not prove the theory since vagus nerves in addition to direct and indirect stimulation, renders the parietal cell more reactive to other types of stimuli and such abnormal stimuli that may increase gastric secrettion before vagotomy may be less effective after vagotomy.

2. Excessive hormonal stimulation of gastric secretion

Higher gastrin levels have been found in duodenal ulcer patients than in normal subjects in response to vagal stimulation.[8] Although G-cell mass in duodenal ulcer patients have greater meal induced gastrin hormone output than normal. This enhanced meal response is probably due to defective regulation of gastrin release. Gastric acid inhibits gastrin release either directly on antral G-cell or indirectly by stimulating gastrin inhibitors from the intestine. Inhibitory effect of acidification is defective in duodenal ulcer. Duodenal ulcer patients are found to have a greater proportion of G-17 the most potent component of gastrin as compared to normal individuals. Hypergastrinemia in Zollinger-Ellison syndrome leads to increased gastric secretion and duodenal ulceration.

3. Excessive capacity to produce acid

Higher rate of acid recretion is highly correlated with large parietal cell mass. Autopsy study by Cox[9] found that duodenal ulcer patients had large stomachs with increased number of perietal cells. This could be inherited trait or as a result of work hyperplasia in response to stimuli or both. Parietal cell mass is found to be more increased in males than females.

4. Defective gastric acid inhibition

Intestinal mechanisms have been shown to inhibit acid secretion. However, acid, fat and hypertonic solutions when instilled into duodenum decrease pentagastrin stimulated acid secretion to the same extent in duodenal ulcer and normal individuals. There is no convincing

evidence that defective inhibition of acid secretion is an important factor in duodenal ulcer disease although there is a lower bulb pH in duodenal ulcer than in normal individual.[10]

5. Gastric emptying

It has been found that duodenal ulcer patients empty gastric contents more rapidly and this reduces the buffering effect of the meal resulting in a greater acid load to the duodenum.[10]

It is therefore clear that duodenal ulcer patients do not only have a greater than normal maximal secretory capacity but more importantly, they secrete at higher than normal rates with submaximal stimuli. A dual defect is present during meal stimulated secretion. Impaired antral inhibition of gastrin release contributes to excessive acid secretion and rapid gastric empting reduces the buffering effect of the meal itself. The result is a large acid load to the duodenum where inhibitory mechanism and pancreatic secretion are not sufficient to control the acid secretion.

Pepsin in gastric acidic juice has proteolytic action and they operate in concert in mucosal destruction characteristic of ulcerogenesis.

(d) Genetic aspects of duodenal ulcers

Duodenal ulceration is more in blood group O than other groups.[8] Secretor status is also significant. Approximately 75 percent of population secrete into saliva, gastric juice and urine water soluble antigens immunologically identical to the blood group antigens on their Red blood cells. They are secretors. The remainder instead secrete Lewis antigen and these are non secretors and are more liable to duodenal ulcer than secretors.[8]

Most duodenal ulcers occur in the first part of the duodenum just beyond the narrow pyloric canal in the duodenal bulb.[2] Perforations are usually on anterior wall of the duodenum.[2] The acute ulcer is seen as small leaking point with slight hyperaemia and chronic ulcer as larger with indurated margins, adhesions and distortion.[11]

In the majority of patients perforation is followed by leakage of air, gastric secretions and food which spread and contaminate peritoneal cavity resulting initially in a chemical peritonitis and later bacterial peritonitis with ileus, toxaemia, fluid and electrolyte imbalance and finally death. In the few cases with slow leak the fluid pools locally or is directed along the right paracolic gutter into the right iliac fossa. Spontaneous closure may occur with omentum sealing off the perforation but in some cases infection may supervene to form a subphrenic abscess which may rupture into the peritoneal or pleural cavity.

Posterior perforation or penetration is rare and duodenal wall is slowly destroyed with the floor of the ulcer being formed by the pancreas, liver or diaphragm. Perforation occurs into the lesser sac.

Clinical features

With large perforations most patients typically present with sudden severe epigastric pain spreading all over the abdomen in some cases radiating to the right or both shoulders. Pain may be accompanied by vomiting and sometimes, haematemesis or melaena. Previous history suggestive of peptic ulcer of varying duration and severity is positive in many cases. However, 20 percent of acute free perforations occur in patients who give no history suggestive of pre-vious duodenal ulcer.[2]

Examination in the early stage of peritoneal irritation, the patient is pale, anxious and loath to move. Temperature may be subnormal but the pulse rate rises to about 80 to 90 per minute. The abdomen is held still, moving little or not at all with respiration. The whole abdomen is tender with board-like rigidity. Sufficient gas may have escaped to reduce liver dullness in the mid-axillary line. Pelvic tenderness may be elicited on rectal examination.

After 3 to 6 hours the pain, tenderness and rigidity may lessen. The temperature rises to normal or higher. However, the pulse remains high, the bowel sounds are absent. This temporary improvement has rightly been called the "period of illusion".[2]

After 6 hours, the stage of diffuse peritonitis develops, accompanied by silent abdominal distension. Enough free fluid may have collected to be clinically detectable. The rising pulse rate marks the progressive deterioration. The patient is extremely toxic and in oligaemic shock.[2]

Atypical presentation occurs mostly in the elderly, the seriously ill and patients with spinal cord injuries. In these, pain may not be a major feature and diagnosis is delayed and mortality increased. In subacute patients pain may be localised to one area of the abdomen and an incorrect diagnosis of pancreatitis, cholecystitis or appendicitis may be made, thus again delaying the right diagnosis and increasing the morbidity and mortality.[12]

Definitive diagnosis is made by the presence of pneumoperitoneum using three radiographic views; erect chest film left lateral decubitus and supine view of the abdomen. Using these views pneumoperitoneum will be detected in 60 to 82 percent.[11] Erect chest film must be taken after patient has been in an upright position for 10 to 20 minutes to allow air to collect under the diaphragm. Left lateral decubitus view may demonstrate as little as a 1 cc of free air. Supine plain abdominal film may show air outlining the bowel wall.[11]

If radiographic diagnosis is uncertain, air or gastrografin can be injected through the nasogastric tube to increase the intraperitoneal air.

Bile-stained fluid may be aspirated from peritoneal cavity; it is alkaline to litmus.[2]

In this study presenting symptoms as shown in "table 3" were as follows: Abdominal pain presented in all 13 patients (100%) vomiting in 9 patients (62.2%); constipation in 7 patients (53.8%) and fever in 3 patients (23%).

6 patients (46.1%) gave positive pre-perforation history of symptoms suggestive of peptic ulcer disease. Pre-perforation drug (Aspirin) ingestion was recorded in 2 patients (15.4%) and alcohol ingestion in another 2 patients and smoking in 3 patients (23%) See table 5.

Main clinical and radiological findings indicative of a perforation were as follows: Tender and rigid abdomen found in all 13 patients (100%) and pneumoperitoneum seen on X-ray of 10 patients (77%). In 3 patients (23%) perforation was confirmed at laparatomy. "See table 6".

Treatment

In all patients treatment is commenced with an intravenous drip for correction of fluid and electrolyte imbalance, a nasogastric tube for decompression and antibiotics for prophylaxis against intraperitoneal and wound infection.

The need of antibiotics increases with increase of post-perforation period supported by the following study findings.[11]

Table 10: Postperforation time and peritoneal infection

Postperforation time	Pos itive peritoneal Cultures	Organisms Isolated
6 hours	21 percent	Streptococci and E-Coli
12 hours	30 percent	"
24 hours	63 percent	"

Therefore prophylactic antibiotics appropriate for streptococci and E-Coli might be of value and should be continued for at least 2 to 3 days post operatively and discontinued if cultures are reported negative or if positive antibiotics are given according to sensitivity report.[11] In this study as shown in table 4, 10 patients (77%) presented with history suggestive of a perforation of more than 24 hours duration. Only 2 patients (15.4%) presented within first 12 hours of perforation and 1 patient (7.7%) presented after 18 hours postperforation. Thus all the patients were started on antibiotics preoperatively. Specific treatment for perforation consists of conservative (Non-operative) or operative methods.

Conservative treatment was introduced by Herman Taylor.[13] They reviewed difference in behaviour between perforations of acute and chronic ulcers both immediately and with incidence of recurrence and

argued that acute ulcers that have perforated if left alone heal without a trace whereas closure distorts the duodenum, introduces a foreign body and interferes with blood supply leading to scarring and recurrence.[13] However, recent opinion reserves this method only for patients with irreversible shock who are Moribund.[14] Steiger and Cooperman[11] advise conservative treatment in two situations:

1. A forme-fruste ulcer, a perforated ulcer that leaked into the peritoneal cavity but was sealed by omentum or surrounding tissues.
2. Patients with other serious illnesses where the operative risk is great such as in acute myocardial infarction. In such cases close continuous observation of the patient should be done. If peritoneal signs progress laparotomy must be undertaken despite high risk. However, mortality rate for such very ill patients is about 90 to 100 percent.

Operative treatment

This is the treatment of choice. But therepeutic dileama exists when deciding which operation should be done; simple suture plication or immediate definitive operation.

Simple suture plication

The perforation may be sutured directly, with or without omental patch. The advantage of this procedure is that it is easy and gives minimal operating time, a significant factor in an emergency. It has proved to be excellent as far as mortality and early morbidity are concerned but some patients will need future medical therapy and some subsequent definitive surgery so as to gain permanent control of ulcer.

Reported mortality rates for this procedure include: 2.1 percent[15] and 5.1 percent.[16] The high mortality usually reflect delayed diagnosis, co-existing disease and/or advanced age. Higher mortality rates are reported in series where good risk patients are assigned immediate definitive operations leaving the high risk patients for plication. 21 percent mortality rate was reported by Jordan at al[15] when most perforated ulcers were treated by definitive ulcer surgery.

In this study 4 patients (30.7%) "table 9) died postoperatively. This was mainly due to late presentation (as shown in table 4) and severe peritoneal sepsis was seen in all the 4 patients who died.

Follow-up studies of patients treated by simple suture plication review the percentage of patients requiring later definitive operation are:

23 percent (Mc Donough)[14]

34.9 percent (Jordan and De Bakey)[15]

37.7 percent (Cassel)[16]

The interval between the initial and second operation varies. Cassel [16] recorded (out of 88 patients) 42 percent of the patients required further surgery within the first year, 81.5 percent within 5 years and all but one patient within 10 years.

Illingworth et al[17] reported similar findings. In contrast Hofkin[18] found that 58 percent required further sugery and that over 50 percent of his patients came to operation after a delay of longer than 5 years.

In this study 1 patient had definitive surgery Truncal Vagotomy and Pyloroplasty done within a 2 year period of follow-up because of Persistant severe symptoms of duodenal ulcer.

Cassel[16] found the following indications for further surgery.

Table 11: Indications for further surgery in 88 patients[16]

Indication	No. of patients
Duration of history and severity of symptoms before perforation	7
Recurrent symptoms	53
Further perforation	10
Haemorrhage from ulcer	12
Pyloric stenosis	6

He found the peak age group of 51 to 60 years and noted that this was probably due to greater number of elderly people in the community he was dealing with. The male to female ratio was 6.25 to 1. This differed from earlier finding of 20 to 1 by Illingworth et al.[19]

The postoperative mortality rate for second perforation was reported by Cassel[16] as 26 percent which was nearly 3 times that of first perforation (9 percent) and advised that definitive surgery should be embarked upon without delay in those who having once perforated develop further symptoms of active ulceration.

Donaldson and Jarret[20] considering factors of age, sex and duration of ulcer symptoms prior to perforation could identify a high risk group of patients. A risk which exceeds 60 percent in male patients between the ages of 50 and 70 years who have had ulcer symptoms for more than 3 months prior to perforation. They advised simple closure of perforation in most women when history of ulcer is less than 3 months, when patients is on steroid therapy and whenever patient is considered a poor risk. However, Jordan et al[15] recommended simple plication only for patients with no previous history of peptic ulcer, patients with generalized purulent peritonitis or those with intra-abdominal abscesses, patients in severe shock and those who have serious concurrent diseases. They believe that in such cases the main aim of an emergency operation is to save life.

The author in his experience in surgical Firm III at Muhimbili Medical Centre has managed cases of perforated duodenal ulcer by simple suture operation and although he agrees that this procedure has no definate effect on peptic ulcer disease and that some patients may need a second operation later, the author found the operation easy and quick to perform and considers this an advantage for an emergency operation in patients who usually present late and when adequate anaesthetic techniques are still lacking.

Immediate definative operation

It is still controversial whether a definative operation should be performed at the time of perforation. Cassel[16] had one third of his patients treated by definative surgery with no increase in mortality rates and believes that provided selection is careful an immediate definative operation is advantageous because it avoids a second operation.

This view is supported by McDonough[14] and Wangensteen.[21]

Definitive operation is particularly necessary where chronicity of an ulcer can be established either by preperforation history or by the appearance at the operation table and granted a patient in a reasonable condition to tolerate additional operation time in a place with adequate facilities.[22]

In this study there was no immediate definitive operation done.

The definitive operations available are:

1. Vagotomy (truncal, selective) + Drainage procedure
2. Vagotomy (truncal, selective) + partial gastric resection
3. Suture + highly selective (proximal gastric) vagotomy
4. Subtotal gastrectomy

The surgeon is guided to choose one type of procedure by the technical problem present, the patient population he is dealing with and his past experience and results.

The assessment of results depends on three principles:

1. Mortality rate
2. Permanent cure of ulcer
3. Side effects

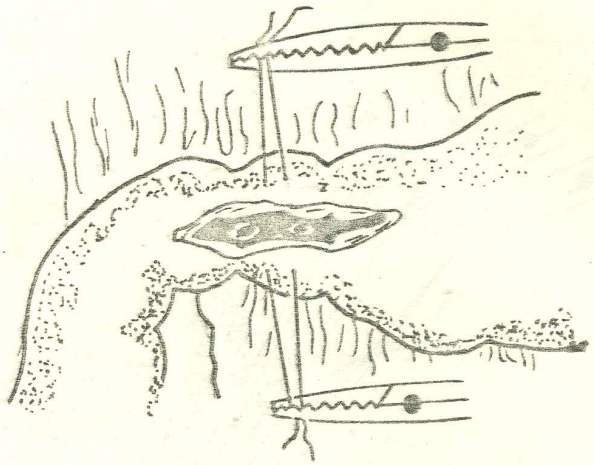
The physiologic basis for vagotomy in the treatment of duodenal ulcer is based on the observation that a hypersecretion of gastric juice of nervous or vagal origin is found uniformly in patients with peptic ulceration. Although Dragstedt[23] found that vagotomy alone often resulted in healing of duodenal ulceration, many patients developed various degrees of gastric stasis which resulted in an augmentation of the humoral (gastrin) mechanism of gastric secretion with persistent or recurrent gastric ulceration. Thus, if vagotomy is to be successful it must be combined with an effective drainage procedure. Gastro-jejunostomy and pyloroplasty are the drainage operations of choice. Highly selective vagotomy which spares the nerve of Latarjet to the antrum has the advantage of not requiring drainage procedure in most cases.

Truncal Vagotomy and pyloroplasty is a simpler and quicker definative operation.[22] The truncal vagotomy deprives not only the stomach, but also the liver, gall-bladder, bile ducts, pancreas, small intestine and proximal half of the large intestine of their perasympathetic nerve supply. It must be complemented by a pyloroplasty or gastro-jejunostomy because it weakens the propulsive power of the antral mill and produces gastric stasis. Pyloroplasty is the drainage procedure of choice because it preserves normal continuity of the pyloroduodenal segment, allowing normal physiologic mixing of duodenal, pancreatic, and biliary secretions with ingested food. It also permits inspection of the duodenal mucosa to determine the degree of ulceration. The frequently used techniques are the Heineke-Mikulicz and Finney.[24] Heineke-Mikulicz pyloroplasty consists of dividing the pyloroduoddenal canal longitudinally and closing it transversely (see illustration 4). In Finney pyloroplasty the duodenum is routinely mobilized and to accomplish this, the lateral peritoneal reflection is incised in a vertical manner. The duodenum is then swept medially and forward by finger dissection until it comes in contact with the stomach. Sutures are applied posteriorly approximating the duodenum and stomach for a distence of about 7 to 8 cm. A horseshoe-shaped incision is then made parallel with the posterior row of sutures on the stomach, pylorus and duodenum. This incision is made about 6 to 7 mm. from the posterior suture line. After controlling bleeding, the inner layer of suture is then made and then the outer anterior layer is completed[24] "See Illustration 5".

The only contraindication to pyloroplasty is obstruction or severe distortion of pyloric canal. In these patients other form of drainage like gastro-jejunostomy must be provided.

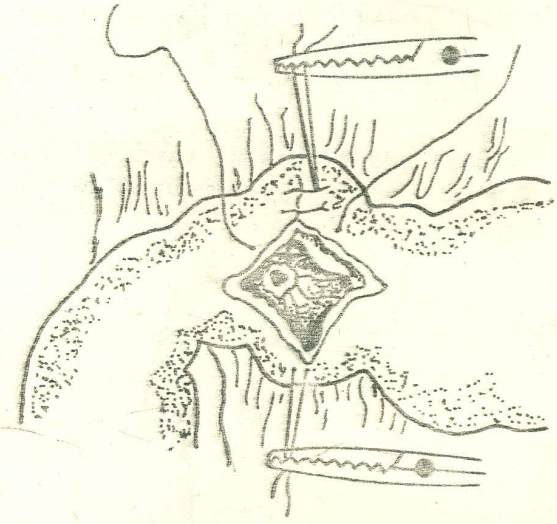
The gastro-jejunostomy can be placed posteriorly or anteriorly. The orthodox posterior gastro-jejunostomy had 50 percent recurrent ulcer rate.[25]

Illustration 4: Heineke - Mikulicz pyloroplasty. The pylorus and the stenosed duodenum are divided longitudinally and sutured transversely.

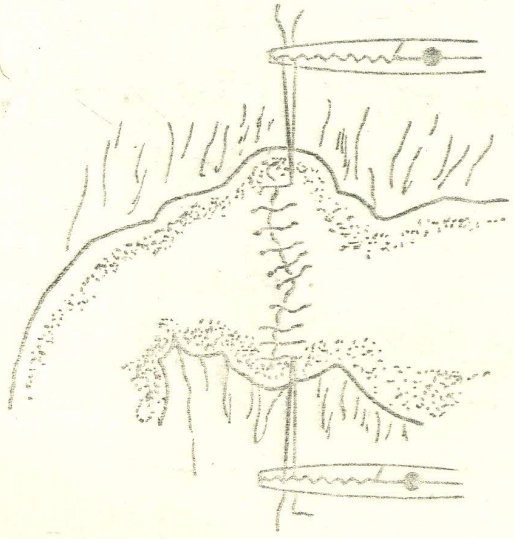


1a.

1a. Shows longitudinal incision of about 4 - 7cm.



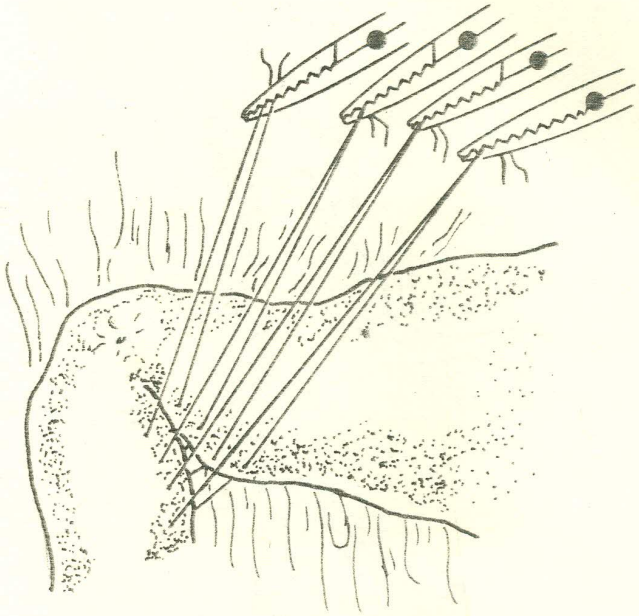
1b.



1c.

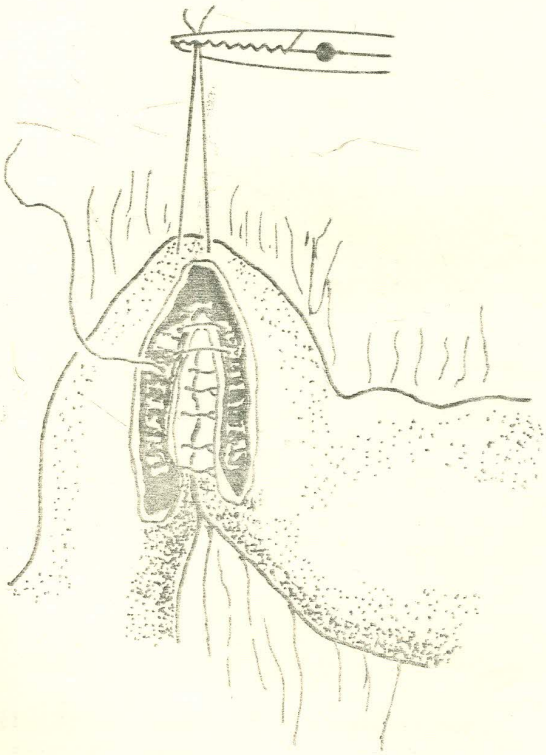
1b and 1c. Shows transverse sutures taking in all layers withminimum of mucosa.

Illustration 5: Finney pyloroplasty.



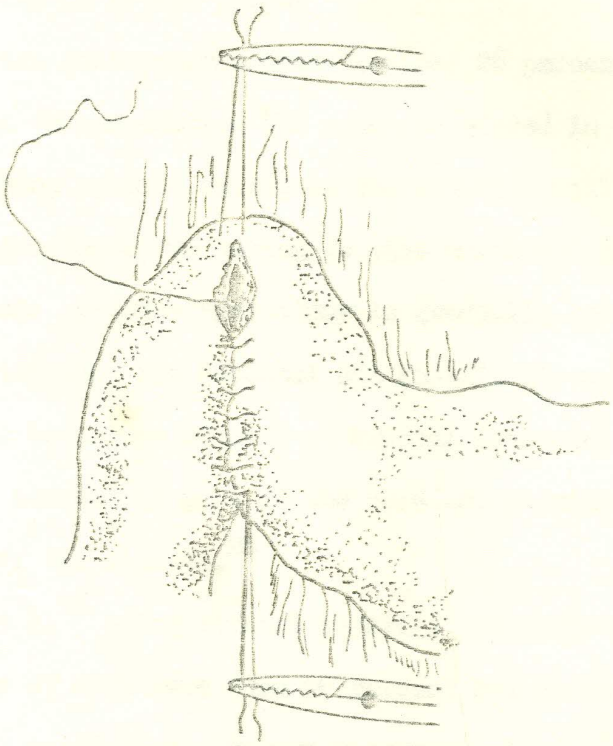
2a.

2a. The adjacent serosal layers of the stomach and duodenum are apposed with sutures.

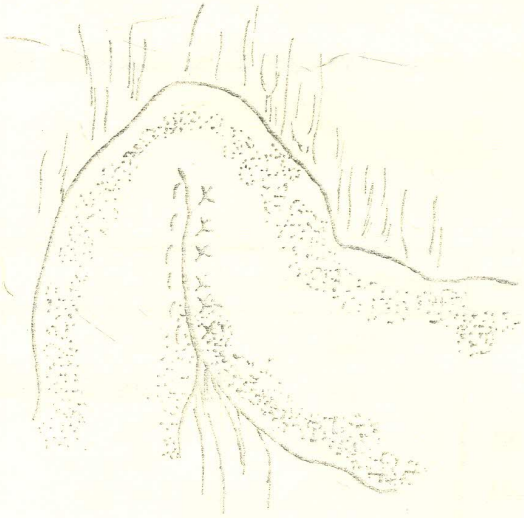


2b.

2b. An inverted U-shaped incision is made extending proximal to the pylorus into the antrum.



2c.



2d.

2c and 2d. The mucosal layers are closed with a continuous catgut suture. A row of interrupted silk sutures completes the two-layer closure.

The site in the posterior wall is the most dependent part of the acid-secreting part of the stomach where the jejunum is constantly bathed in gstric juice.

The anterior juxtapyloric operation had 26 percent recurrent ulcer rate. In this operation the stoma is placed in the alkaline-secreting pyloric antrum on the anterior wall with thedifferent loop near the greater curve hoping to give gravitational drainage in this position. Gastro-jejunostomy is gradually being replaced by partial gastrectomy for duodenal and gastric ulcer. The benefit of this being the removal of humoral (gastrin) stimuli as well as the removal of part of the acid and pepsin-secreting body portions of the stomach.[26]

Hinshaw et al[26] using truncal vagotomy and drainage procedure in 180 patients of whom more than 90 percent had chronic history of peptic ulcer noted that although recurrent ulceration occurs in some patients following pyloroplasty it is a preferred drainage procedure to gastro-jejunostomy which is occasionally associated with bilious vomiting and later nutritional deficiences.

Out of 180 patients with 90% follow-up their results were as follows:

Table 12:

Result	Percentage	Remarks
Mortality	1.1%	
Recurrent ulceration	9%	
Excellent	47%	No ulcer symptoms
Good	27%	Mild ulcer symptoms
Fair	12%	Moderately severe ulcer symptoms
Poor	14%	Severe symptoms

Long term follow-up 15 to 20 years after Truncal Vagotomy and gastro-jejunostomy weight loss and iron deficiency anaemia were found to be very common and 7% of patients had pulmonary tuberculosis.

Selective vagotomy confines vagal denervation to the stomach and preserves vagal innervation to all other abdominal viscera.

Griffith[27] maintains that complete vagal denervation of the stomach is more likely to accomplished by this technique than by truncal vagotomy. Diarrhoea is rare and preservation of vagal innervation to the biliary tract is associated with a reduced incidence of cholelithiasis.[24] However, the main parasympathetic nerve supply to the antral mill is cut and drainage procedure as after truncal vagotomy is required.

Highly selective vagotomy

In this procedure the ulcer is sutured and vagal denervation confined to the body and fundus of the stomach is carried out. The nerve of Latarjet to the antrum is spared. In most cases drainage procedure is not required. Louw and Marks[28] performed the operation on 5 selected patients with no mortality and excellent long term results. Although their series was small they recommend it for perforated duodenal ulcer in a fit patient under the age of 60 years who can withstand extra operating time and in whom history of perforation is not more than 8 hours and peritoneal soiling is minimal. Johnston and Goligher[29] reported operative mortality of about 0.3%. Ischaemic necrosis of the lesser curvature was the cause of death in 0.1%. The only common side effect after highly selective vagotomy is a sensation of post-prandial fullness which is due to failure of receptive relaxation and hence increased pressure in the denervated body of stomach.

Incidence of incomplete vagotomy as shown by the Hollander's test 1 week after operation is high in some centres and in such centres incidence of recurrent ulceration has been as high as 22%. When highly selective vagotomy is completed recurrence is rare.[29]

Vagotomy plus resection is the treatment of choice for a chronic duodenal ulcer with perforation. De Bakey[15] reported on a series of 583 patients of whom 327 had immediate gastrectomy with mortality rate of 2.1% and 208 patients had vagotomy and hemigastrectomy with a mortality rate of 2.5%. Of the 755 patients with a perforated

duodenal ulcer, 37 died. In this series vagotomy and resection gave excellent or good results in 90.6% of patients followed-up. Incidence of recurrent ulceration after truncal vagotomy and antrectomy is only 1%. However, truncal vagotomy and antrectomy should be avoided in high risk patient and when the duodenum poses technical problems. Truncal vagotomy and antrectomy has a high operative mortality than truncal vagotomy and drainage procedure. Thus if one ignores the superiority of truncal vagotomy and antrectomy in terms of recurrent ulceration one, would make truncal vagotomy plus drainage the wiser choice.

Elective Definitive Operation

Harbrecht and Hamilton[22] state that in choosing definitive elective procedure following previous perforation, one considers a variety of procedures acceptable for chronic duodenal ulcer. In their opinion vagotomy and pyloroplasty is preferred and in their experience they had no mortality.

Louw and Marks[28] studed 783 patients subjected to elective surgery for chronic duodenal ulcer and made observations on three types of operations as follows; mortality rates for partial gastrectomy was 0.8%, vagotomy and antrectomy 0.7% and that of vagotomy and drainage procedure was 0.3%. However, risk of recurrent ulcers was higher with vagotomy and drainage procedure "7%". Cure rates were best with vagotomy and antrectomy. In this combined operation, division of the vagus nerves eliminates thecephalic phase of gastric secretion which comprises approximately 60 to 70 percent of acid output in the duodenal ulcer patient while excision of the distal 40% to 50% of the stomach removes the main source of the hormone gastrin which is responsible for humoral phase of gastric secretion.[30]

Controversy exists regarding what constitutes an adequate antrectomy. It is probable that about one-half of the stomach should be removed, including almost the entire lesser curvature, in order to ensure that all antral tissue has been excised. In severe ulcer

diathesis both subtotal gastrectomy, in which 65 to 70% of distal stomach is removed, and vagotomy may be indicated, and indeed total gastrectomy is necessary if a Zollinger-Ellison tumour is present.[24]

Johnston and Goligher[29] recommend highly selective vagotomy as safer, having fewer side effects and long term sequelae than vagotomy and antrectomy although recurrence of ulceration is more after highly selective vagotomy (5 to 10%) than after vagotomy and antrectomy (1 to 2%). In their opinion this equation favours highly selective vagotomy as the initial procedure for duodenal ulcer and that in the 5 to 10% that recurs patients can be salvaged by subsequent antrectomy.

The choice of operation in the treatment of duodenal ulcer may be difficult, and the age, sex, build and clinical history must be taken into consideration in this decision. The maximal histamine test is also of great value in assisting in the selection of operation. The choice of operative procedure also is dictated in part by the specific indication for operation.[24]

The author agrees that Truncal Vagotomy and pyloroplasty is a simpler and quicker definitive operation to do. However, when dealing with recurrent duodenal ulcer disease and in absence of Zollinger-Ellison syndrome, a combined vagotomy and antrectomy is advisable operation to perform.

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CARCINOMA BREAST

SUMMARY

Twenty five patients of breast cancer are presented. One of the patients is presented in detail. Review of literature and discussion of incendence, etiological factors, pathology, clinical manifestation, investigations and management of breast cancer are presented.

INTRODUCTION

Breast cancer was first described in the Edwin Smith Papyrus some 3000 years B.C. Anjelo Nannoni 1946 described an operation that included excision of breast, pectoralis major muscle and the hardened "axillary nodes as specific therapy for breast cancer. Since then many surgeons including Halsted, Patey, McWhirter and others have made variable contributions to the understanding and management of breast cancer.

Breast cancer is a world wide disease. It is perhaps the commonest site of cancer in females. Philip[1] has also noted that the incidence of breast cancer has been increasing by 1% for at least the last 10 years. According to Hubay[2] carcinoma of the breast is six times more prevalent in North America and in Europe than in Asia and in Africa. Some quoted annual incidence of female breast cancer are; in Uganda 6.8 per 100,000, in India 20.4 per 100,000, in United Kingdom 42.5 to 51.1 per 100,000, in U.S.A. 62.3 to 62.4 per 100,000.

In Tanzania Hiza[3] has observed scattered distribution of breast cancer throughout the country with a peak age incidence of 30 to 60 years. He found 328 cases over a 5 year period covering 1967 to 1973 and he noted that carcinoma breast is the most commonly diagnosed tumour in the country.

Carcinoma breast is predominantly a disease of females. Only 0.6% of all cases occur in males.[4] Women between forty and fifty years of age are its most frequent victims, the disease having higher incidence in the unmarried and in the less fertile married women.[4]

In this study all the 25 cases were female. The minimum age recorded was 30 years, the maximum was 80 years (table 1). Only 28% of the patients were residents of Dar es Salaam. The rest (72%) came from other regions as shown in the table II.

CASE REPORT

H.S., Hosp. No. 241760, 30 years old female was admitted by surgical firm III on 6th March, 1981 with history of right breast mass for two months, ulceration of the mass for one month and pain in the right axilla for two weeks. There was no associated fever, no nipple discharge, no cough, no dyspnoea and no backache.

She was married and had one 5 year old child whom she did not breast feed. There was no history of similar breast tumour in her family. She never had any other major illness before.

On examination she was found to be moderately wasted young lady, afebrile, not pale and not jaundiced. Blood pressure was 110/70 mmHg in lying position pulse 72/min. regular and good volume.

The right breast was swollen with extensive ulceration. Area of skin involvement was bigger than the underlying tumour. The tumour was not fixed to the chest wall. Right axillary lymph nodes were enlarged, hard and fixed to the chest wall. The supraclavicular nodes were not palpable. The other breast and axilla were clinically normal. Chest examination was essentially normal, liver was not palpable and no other masses were palpable per abdomen. No abnormal pelvic mass was detected on vaginal and rectal examinations.

Chest X-ray did not show any metastatic lesion. A diagnosis of possible malignant breast tumour clinically stage III (T_3, N_2, M_0) was made. Biopsy of the ulcer and axillary node were taken and sent for histology. It was reported as "anaplastic carcinoma of the breast, lymph node is positive for metastasis". The patient was taken to Tumour Board for discussion. The Tumour Board recommended; toilet mastectomy, course of radiotherapy to the right chest and Oophorectomy. The plan of management was explained to the patient and she agreed.

Preoperatively full blood picture, serum alkaline phosphatase, liver function tests, urine and stool examinations were done and found to be normal. Blood grouping and cross-matching was done. On the day of operation, 3 units of blood group A positive was available for the patient.

Right toilet mastectomy was done through an oblique incision. Skin was undermined and closed with a drain in the wound. She received two units of blood in theatre.

Postoperatively the IV. drip was maintained for 24 hours with 5% dextrose. Chloramphenical 500 mgs. 6 hourly and P.P.F. 2 mega units daily intramuscular injections were given for five days. Pethidine 100 mgs 8 hourly IM for 48 hours was ordered. Blood pressure, pulse and respiration observations were also ordered initially ½ hourly until the patient is fully awake and then 2 hourly for next 24 hours.

Two days postoperatively the wound started draining pus. 2 stitches were removed to facilitate the drainage and pus was sent for culture and sensitivity. It was reported as heavy growth of E-Coli sensitive to Chloramphenical and gentamycin. Gentamycin 80 mgs 8 hourly intramuscular injection was added to the treatment plus daily eusol dressing. The infection was controlled and the wound healed.

The patient was started on right chest radiotherapy and received a total dose of 5000 rad given in an overall time of 43 days by Cobalt machine. Radiotherapy was well tolerated.

Bilateral surgical Oophorectomy was done through midline subumbilical incision. The left ovary was slightly larger and cystic the right ovary appeared normal. The peritoneum and liver appeared to be uninvolved. After acheiving haemostasis, the abdominal wound was closed in layers. The ovaries were sent for histology but results were not found. Postoperatively patient had smooth recovery. The wound healed well and was discharged with a letter to her regional hospital in Morogoro for regular follow-up.

She was also advised to come back to Muhimbili Medical Centre after six months for review but she did not turn up.

Rest of the presentation was as follows:

Table 1: Age incidence

Age range (Years)	No. of patients	percentages
Below 30	-	-
30 - 39	4	16%
40 - 49	3	12%
50 - 59	7	28%
60 - 69	8	32%
70 - 79	2	8%
80 or above	1	4%
Total	25	100%

Minimum age recorded was 30 years. Maximum age recorded was 80 years. All the patients were females.

Table 2: Region of origin

Region	No. of patients	Percentages
Dar es Salaam	7	28%
Morogoro	4	16%
Tabora	3	12%
Tanga	3	12%
Dodoma	3	12%
Songea	2	8%
Mwanza	1	4%
Singida	2	8%
Total	25	100%

Table 3: Symptoms at initial presentation

Symptom	No. of patients	Percentages
Breast lump	25	100%
Breast ulcer	9	36%
Chest pain	4	16%
Fevers	2	8%

Table 4: Parity

Parity	No. of patients	Percentages
Nulliparous	2	8%
Para 1 to 5	9	36%
Para 5 or more	10	40%
Not asked for	4	16%
Total	25	100%

1. 21 patients (84%) said they were breast feeding their children.

2. None of the patients studied used contraceptive pill.

Table 5: Clincal stage at admission

Stage	No. of patients	Percentages
I	2	8%
II	6	24%
III	7	28%
IV	5	20%
Bilateral	2	8%
Recurrent	3	12%
Total	25	100%

Table 6: Treatment offered according to the clinical stage of the disease. Number of patients also indicated.

Treatment	Stage of disease/no. of patients					
	I	II	III	IV	Bilateral	Recurrent
Surgery: (mastectomy)						
Simple	1		4		2	
Radical		1	-	-	-	-
Mod. radical		6	-	-	-	-
Toilet			6	4	-	-
Lumpectomy	-	-	-	-	-	3
Radiotherapy:						
Adjuvant	1	2	5	-	-	-
Alone	-	-	-	-	-	-
Palliative		3		1	1	2
Hormonal						
Manipulation:						
Oophorectomy	-	-	1	2	1	2
Adrenalectomy	-	-	-	-	-	-
Hypophysectomy	-	-	-	-	-	-
Estrogens	-	-	-	-	-	-
Audrogens	-	-	-	-	-	-
Corticostecoids (Prednisolone)		2	1	1		
Antiestrogens	-	-	-	-	-	-
Chemotherapy	-	-	-	-	-	-
Immunotherapy	-	-	-	-	-	-

Table 7: Results of Treatment

1. Death before discharge	-	-	-	-	1	-
2. Poor response at disch. patient weak and wasted	-	-	3	5	1	3
3. Good response with no recurrence up to May 1982	2	6	4	-	-	-

DISCUSSION

Etiology:

The actual cause of breast carcinoma is unknown. However, there are many etiological factors associated with breast carcinoma.

Genetic factors:

Seem to play a role in the development of carcinoma breast. When the mother has had breast cancer the chance in a daughter is 2 to 3 times greater than expected in the general population hence the hypothesis that there is a genotype which has a predisposition to the formation of cancer but which must interact with some nongenetic agent before cancer develops.[6] Patients with positive family histories for the disease are generally younger and have higher frequency of bilaterality than those with negative family history. Anderson[6] found that the risk of breast cancer in relatives of patients with bilateral disease and positive family history is 3 to 4 times higher than that in relatives of patients with unilateral disease. Blood group O, benign breast disease and ovarian cysts and tumours also tend to be more common in patients with early breast cancer. Blood group A, diabetes, hypertension and uterine disorders are common in those with late breast cancer. Hems G.[7] concluded that 'early' breast cancer (mainly of age group 40 to 44 years) appears to be genetically influenced while 'Late' breast cancer (mainly of age group 65 to 69 years) is more associated with environmental factors such as diet. Among women over 50 years of age the risk increases with weight and the relation of weight to height.

Zippin and Petrakos[8] have reported an association between wet cerumen or earwax and breast cancer rates. The association is important because mammary and ceruminous glands are histologically of apocrine type and have many similarities in their secretions. These findings in the apocrine system may influence susceptibility to breast cancer. Cerumen exists in two phenotypic forms, wet and dry, controlled by a pair of genes. The wet type predominates in Western Europeans,

Caucasian Americans and negro Americans and it has been found that breast cancer mortality is associated with wet cerumen. It is found that susceptibility to breast cancer in Negro American women may be a function of the extent of Caucasian genetic contribution to the Negro gene pool and this agrees with the belief that susceptibility may be genetically multifactorial.[8]

Hormonal factors:

Hormonal promoters and inhibitors for any particular type of cell seen to be the same whether the cell is normal or malignant.[5] The hormone first thought to be responsible for mammary tumours was estrogen, but in the regulation of mammary gland function estrogens act indirectly by way of the pituitary. Five of the six anterior pituitary hormones play important parts in mammary development. A pituitary promoter for the mammary cancer cell has not been identified among the pituitary trophic hormones (except perhaps somatotrophin) but possibly all of them are involved.[5]

Estrogen administration under certain conditions causes increased release of pituitary hormones and this effect can be modified by the simultaneous administration of steroids from the target organs like relatively large doses of progesterone combined with moderate doses of estradiol.

In mice with high incidence of spontaneous mammary cancer in-bred females and low incidence in males, the administration of testosterone to females of a high-cancer strain lowers the incidence of tumours. Both testosterone and progesterone increased the latent period for induction of mammary tumours by estrogen in rats and caused temporary regression of tumours that had been established after estrogen. Once cancer cells become autonomous, hormonal factors become less important. Thus the ovarian importance in etiology of human breast cancer is in its influence upon pituitary through ovarian steroids, including estrogens, progesterone and their biosynthetic precursors and metabolites. Oophorectomy before age of 40 years is associated with low risk of breast cancer.[5] Women who have had their

ovaries removed before the age of 40 years escape the climacteric years during which ovarian secretory activity is on the wane and long periods of teetering levels of ovarian steroids are likely to occur and abnormal activity of pituitary - Ovarian axis in this age-group is reflected by irregular menstruation, premenstrual tension, painful problem breast and chronic cystic mastitis which has epidemilogical similarities to breast cancer. Studies of urinary estrogen and pregnanediol in women with these complaints indicate that estrogen is in excessive amounts but progesterone is inadequate. In normal post ovulation situation progesterone is secreted in about 10 to 1 ratio to estrogen and higher ratios of oestrial to oestrone and oestradiol in urine occurs a situation that does not pertain in unovulatory cycles. With regression of corpus luteum, progesterone secretion reduces and when progesterone and estrogen support is withdrawn from breast, swelling of breasts may occur. This reflects increased secretion of pituitary in breast tissue which has been under the influence of ovarian steroids. Periods of estrogen secretion with inadequate progesterone are likely to become chronic in late premenopausal and menopausal years. Women who have had their ovaries removed before age of 40 years escape this period of waning ovarian secretory activity and its retroactive effects upon hypothalamus, adenohypophysis and breasts.

Early pregnancy (before age of 20 years) is associated with low risk of breast carcinoma.[5] It is possible that menarche and adolescence like the late premenopausal and menopausal years are associated with less adequate post-ovulatory biosyntheais and metabolism of the ovarian steroids and hence by less frequent and less prolonged intervals when the release of pituitary trophic hormones stimulated by estrogen, is interrupted. There is lack of adequate suppression by cyclic development of a fully functional corpus luteum. An intervening pregnancy would provide a 9 months period of release from pituitary stimulation. During pregnancy there is increased estrogen production almost entirely by fetoplacental unit and 10 to 20 fold increase in progesterone production by the placenta.

The human pituitary has no gonadotrophic activity during pregnancy and there is decrease in growth hormone secreting cells. Remission of mammary cancer during pregnancy, although still controversial, has been observed and its rapid and often fatal recurrence after parturition, coincident with the pituitary "come-back" of all pituitary trophic hormones is well recognized.

Pregnancy also provides a prolonged release from somatotrophic hormone upon potentially malignant mammary cells. Pituitary secretion of somatotrophic hormone is relatively high in adolescence. Low breast-cancer risk to Japanese is partly due to less pituitary secretion of somatotrophic hormone hence their small stature.[7]

Women who had had their first child at or after 35 years of age experienced breast cancer risks more than twice as high as those who had their first child under 20 years. Low risk of breast cancer is also associated with high parity. However, women who had early first pregnancy usually have high parity.[5] Love and MacMahon[5] found that women who had five or more full term pregnancies were at only about a third (39%) of the risk of breast cancer of multiparous married women. Frequent or prolonged lactation associated with multiparity has relative decrease in risk of breast cancer. However, the low risk of women who marry early is only afforded by their liability to early pregnancy.

High socioeconomic status is associated with high risk of breast cancer.[5] Women who had full time education into their twenties were at a risk of breast cancer that was three times greater than that of women who had left school before age of 17 years. Differences in age at first pregnancy by socioeconomic class account for the variation not lactational experience as was previously thought to be an explanation of socioeconomic or parity differentials in breast cancer risk.

Women taking oral contraceptives do not have an increased incidence of breast cancer.[10] However, the controversy still remains over whether taking oral contraceptives affects the prognosis of the establishe

diagnosed breast cancer not to take contraceptive steroids. Spencer et al[11] reported on 44 patients who had been taking oral contraceptives during the year preceding the diagnosis of breast cancer and compared these women with a control group of patients who had never taken oral contraceptives. They found no evidence that taking such products caused increased progression of the disease. Vessey et al[10] studied 700 women who had not taken oral contraceptives seemed to present with more advanced tumours and that this was reflected in the survival pattern. They also concluded that taking these contraceptives might have a beneficial biological effect on tumour growth and spread. There is certainly no evidence that taking oral contraceptives had an harmful effect, except possibly in patients with close family history of breast cancer. In this study none of the 26 patients used contraceptives. 21 patients (84%) breast fed all their children. 10 patients were multiparous with more than 5 children at the time they developed breast tumour. 9 patients had children ranging from 1 to 5 and 2 patients were nulliporuous. History of parity was not asked for in 4 patients (table 4).

Nutritional factors

Wynder[12] considered dietary fat, obesity, breast size and carbohydrate metabolism as factors associated with the risk of breast cancer. Stokes[13] has reported breast cancer death rates in regions of England and Wales to be positively associated with intake of butter, cheese, liquid milk and green vegetables.

Methylxanthine consumption is associated with development of fibrocystic disease of the breast while incidence of breast cancer is known to be increased in women with fibrocystic disease of the breast.[14] Methylxanthine abstention is associated with resolution of signs and symptoms of fibrocystic disease, and return to normal breast tissue consistency within one month. Chemicals classified as Methylxanthines include caffeine, theophylline and theopromine. All are present in many dietary substances such as coffee,

tea, colas, chocolates and several respiratory and pain medication.[14] In a matched study in Utah, Mormons exhibited significantly lower incidences of most types of cancer including breast cancer than did non-Mormons. Mormon faith forbids consumption of Methylxanthines. Methylxanthines inhibit the action of cyclic AMP and cyclic GMP phosphodiesterase which normally converts cyclic AMP and cyclic GMP to 5 AMP and 5'GMP. The intracellular build up of cyclic AMP and GMP in a cell stimulates protein-kinase activities which direct cellular nucleoproteins and enzymes to respond metabolically. These exaggerated signals may stimulate the overproduction of cellular products, such as fibrous tissue and cyst fluid. There is an increase in severity of fibrocystic disease with time and chronic excessive methylxanthine consumption.

Smoking slows or prevents resolution of fibrocystic disease of the breast because of effect of nicotine on the release of epinephrine producing increased cyclic AMP synthesis.[14]

Viral factor

It has long been known that breast cancer in mice is related to a viral factor transmitted in milk. Moore et al[15] have reported finding in human milk particles with same morphological characteristics as those found to be associated with breast cancer in mice. Of 10 American women with a family history of breast cancer, 6(60%) demonstrated these particles compared with 7(5%) of 146 women without a positive family history. Also 18(39%) of 46 Parsi women in India contained these particles in their milk. Parsi people have endogamous history over centuries resulting in an inbred population. In addition, breast cancer accounts for half of the cancers among Parsi women. Another support of virus etiology for some cases of breast carcinoma is the finding of auto-antibody response against a breast carcinoma tumour-specific antigen. This was found in 6 of 11 breast cancers examined and in one of the controls.[16]

Pathology

Malignant neoplasms, with few exceptions, are adenocarcinomas and Foote and Stewart proposed histological classification as follows:[17]

Paget's disease of the nipple; Carcinomas of Mammary ducts, noninfiltrating or infiltrating which include papillary carcinoma, comedocarcinoma, carcinoma with productive fibrosis, Medullary carcinoma and colloid carcinoma; Carcinomas of Mammary lobules, noninfiltrating or infiltrating; rarely breast sarcoma, carcinosarcoma of Malignant cystosarcoma phylloides are seen.

Paget's disease of the nipple forms 1% of breast carcinomas. It is slow growing, eczematoid tumour arising from mammary ducts of the nipple. Histology shows a large binucleated cell with clear vacuolated cytoplasm. If noninfiltrating, 5 year survival is 100%, if infiltrating survival is about 64%. Noninfiltrating mammary duct carcinoma constitutes 1% of breast carcinomas. Histology shows epithelium thrown up into papillae and pleomorphic, mitotic cells with no invasion of basement membrane. Comedocarcinoma type will extrude small cores of tissue from ducts on gross section when it is squeezed. Infiltrating papillary carcinoma is the late stage of noninfiltrating papillary lesion which is slow growing and may reach large size before metastasizing to the axilla. Dimpling and skin oedema are uncommon. Another index of late infiltration of the lymphatics and of failure to stimulate a fibrous response.

Infiltrating comedocarcinomas comprise about 5% of all breast cancers and are often found in association with other forms of adenocarcinomas.

Infiltrating duct carcinoma with productive fibrosis constitutes 78% of specimens seen. These lesions have been termed scirrhous carcinoma, fibrocarcinoma or sclerosing carcinoma. The desmoplastic response to the invading cancer cells accounts for remarkable hardness of the average breast cancer. Grossly, lesions have uneven serrated edges, cut with resistance and gritty feeling. Histologically the lesions vary from scattered, well-differentiated adenomatous clusters and massive amounts of fibrosarcoma to dense cellular aggregates with only minor amounts of fibroplasia. Electron

microscopic examination of these tumours indicates that they originate in the myoepithelial cells of the mammary duct.

Medullary breast carcinoma constitute about 5%. On physical examination the tumours are soft, freely mobile and may be misdignosed as cysts or fibroadenoma. Grossly necrotic areas are usually present. Histology shows polygonal cells with abundant cytoplasm arranged in plexiform masses anastomosing with one another. Electron microscopic and histochemical evidence suggests that these cells originate in ductal epithelium. There is abundant lymphoid infiltrate and plasma cells are sometimes prominent. Axillary metastases occur less frequently (in about 40%) and often only involve a single node.

Colloid carcinoma constitutes about 1% of all breast cancers. Clinically are soft, ill defined and bulky. Gelatinous on cut section. Histology shows large mucinous lakes in which epithelial aggregates float. Have better prognosis.

Carcinoma of mammary lobules arise from cells of acini and terminal ducts. Initially insitu subsequently becomes infiltrative giving rise to regular scirrhous carcinoma. Multicentricity (35 to 59% of contralateral breast) are important features of lobular carcinoma. Simple mastectomy done when the tumour is insitu stage is curative.[17]

Sarcoma of the breast is rare, of these cystosarcoma phylloides is commonest but only 1 in 10 is truly malignant, majority being a benign. Cystosarcoma phylloides occurs at an average age of 40 years. It has more cellular stroma than fibroadenoma. Clinically the tumours average 5 to 10 cm of firm, rubbery consistancy and may have bosselated surface. Cut surface shows a discrete capsule, small lesions present leaflike intracanalicular protrusions and larger lesions have cystic spaces in which project densely packed polypoid masses. The malignant variant metastasizes commonly to the lungs, bones and subcutaneous tissues. Axillary metastasis is so uncommon that simple mastectomy is adequate procedure for both the benign and the malignant forms.[17]

Mastitis carcinomatosa constitutes about 2% of breast cancer and commonly occurs during pregnancy or lactation.[4] The diseased breast is often painful, warm and has reddened skin as if it is inflammed. Extensive cutaneous oedema occurs indicating blockage of subdermal lymphatics with carcinoma cells. Enlargement of axillary nodes is common. It is a galloping form of mammary cancer with increased proliferation of cancer cells with very little fibrous reaction. It is differentiated from breast abscess by abscence of pyrexia and leucocytosis and failure to respond to antibiotics.

Spread of breast carcinoma can occur by locally invading other portions of the breast and may involve skin, penetrate pectoral muscles or chest wall. Lymphatic spread occurs by either embolization of cancer cells in the lymph stream or permeation by columns of cancer cells growing along the lumina of lymphatic channels and may also reach the skin or pectoral muscles. Lymphatic vessels of the breast are as follows: Cutaneous lymphatics draining the skin communicate with sub-areolar plexus of Sappey and, by means of vessels accompaning the ligaments of Cooper, with the lymphatics of gland stroma. The sub-areolar plexus of sappey also communicates with vessels accompanying the ducts, plexuses around glandular acini and with vessel on the deep surface of the gland.[18] From superficial, subareolar and deep plexuses lymph channels pass mainly to the regional lymph nodes accompanying veins. However, some cutaneous lymphatics communicate across the midline with the cutaneous lymphatics of the opposite breast, and unilateral disease may become bilateral by this route. The infero-medial quadrant of the breast is close to xiphi-sternum and lymphatics of the subjacent fascial plexus communicate through the upper thin part of the linea alba, falciform ligament and peritoneal cavity; secondary deposits may appear on the surface of the liver or cancer cells, traversing the peritoneal cavity, may give rise to metastases in the pelvis.

All five axillary groups of lymphanodes directly or indirectly receive lymph from the breast. The anterior group lies under cover of pectoral muscles forming anterior wall of the axilla. Posterior group lies on the posterior wall along subscapular vessels. Lateral group lies along the upper part of the humerus on the medial side of axillary vessels. The central group lies in the fat in the central part of axilla receives afferents from all three groups of nodes. The apical group is at the apex of the axilla, in the space between pectaralis minor and clavicle and covered by clavi-pectoral fascia. Afferents from other four groups reach the apical group and also receives some vessels directly from the breast and may be the first to become involved in carcinoma. Afferents from the apical glands drain mainly to the blood stream at the origin of the innominate vein, but some pass to the supra-clavicular glands which also receive from internal mammary glands. The supraclavicular glands lie in the lower posterior triangle and belong to deep cervical glands.

The internal mammary gland (anterior mediastinal or parasternal glands) lie along side the artery of the same name, and receive afferent vessels originating in the lymphatic plexus of deep fascia and passing deeply along the course of perforating branches of the internal mammary artery. Their efferents pass mainly to the innominate vein but some pass to supraclavicular nodes.

Distant metastases, usually seen at late stage of the disease, are generally regarded as being due to blood spread. They develop most commonly in the lungs, pleura, bones (spine, pelvis, femur, ribs and skull) and also to the liver and brain. The cancer cells may also travel in retrograde manner along venous channels. Leaving the mammary region by posterior intercostal veins or by lymph vessels accompanying them) they gain access to the rich paravertebral plexus of veins running the whole length of the spinal column. From these veins they may pass in a retrograde direction along anastomosing

veins which drain the lungs and pleura and the vertebral bodies. As the result of upward and downward spread within the paravertebral plexus, metastatic deposits may develop in the skull, pelvis or femur.

Clinical features

About three quarters of patients present with history of painless lump found accidentally or following minor trauma to the breast.[19] 60% occur in the upper outer quadrant, 12% in the upper inner quadrant, 12% areolar area, 10% in the lower outer quadrant and 6% in the lower inner quandrant.[4] About 10% of patients have nipple discharge and another 10% have mild discomfort and tingling sensation. Areolar irritation and blood stained nipple discharge may indicate Pagets disease. There may be eczymatous changes on the nipple. As the tumour invades the surrounding fibrous ligaments of ducts, retraction of overlying skin or nipple occurs. The inflammatory type of carcinoma produces diffuse induration rather than a discrete mass and is associated with erythema and oedema. Extensive plugging of lymphatic channels by tumour leads to stasis of lymph edema of the breast and proliferation of in-transit tumour cells to produce satellites within the dermis. Ulceration may eventually occur.[20]

Metastases resulting from both vascular and lymphatic dissemination, can occur while the primary lesion is still clinically occult. 20% of carcinomas even as small as 1 cm in diameter, already will have metastasized when they are discovered.[20] Osseous involvement usually is lytic, but may be blastic and leads to pathologic fractures, hypercalcemia and anaemia when bone marrow is extensively replaced. Lungs, liver, and brain are also sites of symptomatic metastases. Pulmonary lesions, often associated with pleural effusions, may be nodular and discrete, but lymphangitic spread in the parenchyma is not uncommon, and produces respiratory insufficiency with its distressing symptoms of dyspnoea and cough.[20] Liver involvement may result in right upper abdominal discomforts, anorexia and jaundice. Deep jaundice suggests presence of metastasis

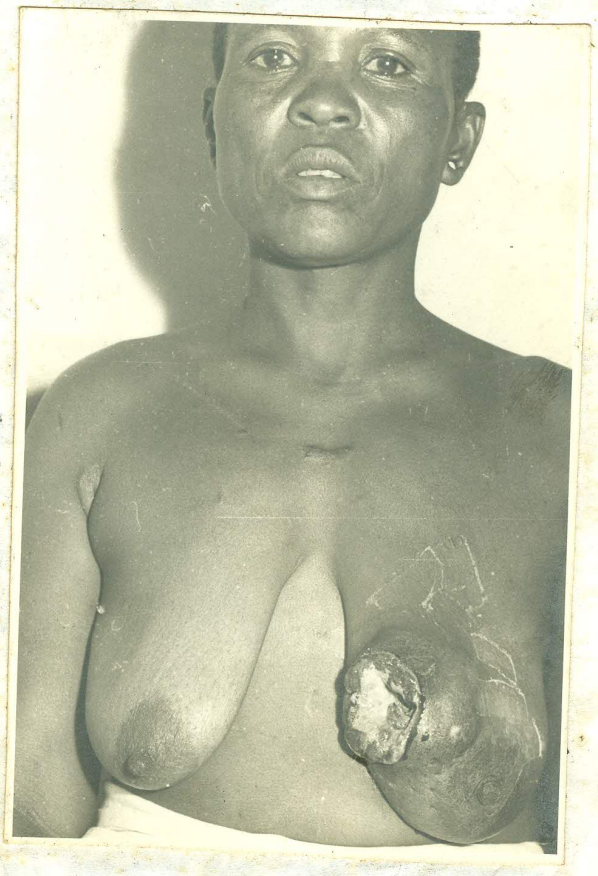


Illustration 6:
Shows ulcerated left breast tumour involving medial upper quadrant.

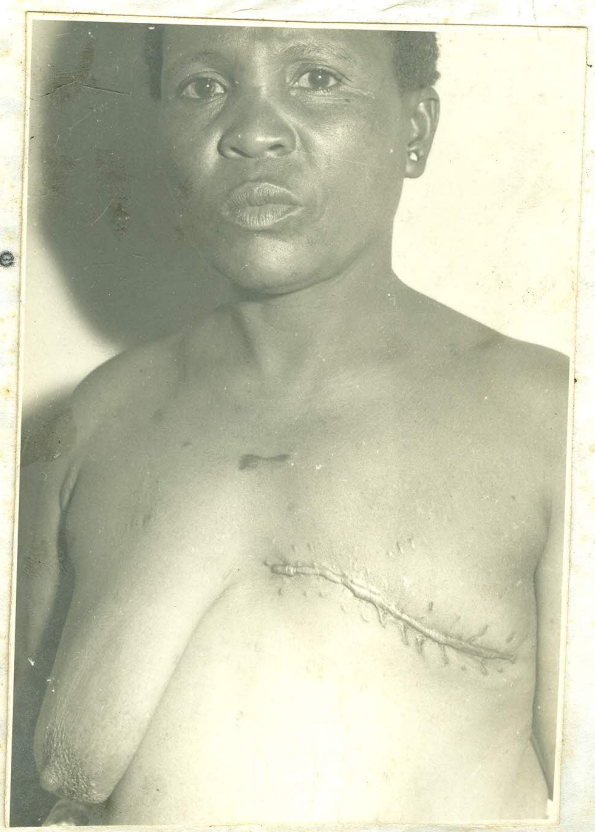


Illustration 7:
Shows the same patient in Illustration 6 after simple left mastectomy was done.

in porta hepatis obstructing the extrahepatic bile ducts. Abdominal distension may occur from ascites.[19] Brain metastasis are followed by a variety of neurologic signs.[20] Dermal and subcutaneous deposits appear randomly or at a mastectomy site. Local recurrence is usually a sign of wide spread disease. Swelling of the arm 6 months postoperative is most likely the result of recurrent disease in the axilla. In this study 25 patients (100%) presented with a breast lump. Ulceration had occurred in 9 patients (36%). 4 patients (16%) complained of chest pain and 2 patients (8%) complained of fever at the time of presentation (table 3). Illustration 6 shows one of the patients with ulcerated left breast tumour involving medial upper quadrant. Illustration 7 shows the same patient after simple left mastectomy was done. She also had Oophorectomy done.

On examination, the site and size of breast tumour and/or ulcer must be determined and evidence for spread to the skin and/or deep structures, pectoralis muscles and chest wall, is looked for. The axilla and supraclavicular region are palpated for nodal involvement. Swelling in the fingers signifies extensive axillary nodal infiltration and that prognosis is poor. Edema of the skin overlying the entire breast or satellite skin nodules signifies that only palliative treatment is indicated.

Patient should also be examined for distant metastasis. Tenderness over bone especially the ribs, sternum and vertebrae suggests bony metastasis. Abnormality in breath sounds suggests pleural effusion from lung metastases while liver metastasis will cause hepatonegaly and jaundice. The occasional metastasis in the pelvis produces a rectal shelf or ovarian enlargement can be detected by vaginal or rectal examination.

Neurologic abnormality indicate cerebral metastasis and signs of Addison's disease would suggest metastasis in the adrenal glands.

Staging is valuable in planning the treatment and making a prognosis. Manchester classification is based on spread of the primary tumour toward the skin or chest wall, the state of the regional nodes and presence or obsence of distant metastasis and is as follows:

Stage I: Growth confined to the breast. An area of adherence to the skin smaller than the periphery of the tumour, even ulceration does not affect staging. Stage II: Same as Stage I but there are affected mobile lymph nodes in the axilla. Stage III: Skin involvement larger than tumour and/or lymph nodes one fixed and/or tumour fixed to pectoral muscle and/or affected lymph nodes in supraclavicular fossa. Stage IV: The same as Stage III plus distant metastases either blood borne or lymph borne; this includes metastases to opposite breast, axilla, supraclavicular fossa, or satellite skin nodules.

Stage I and II are taken as suitable for attempted curative treatment. Those in Stage III and IV are advanced and are given palliative treatment. 5 year survival is approximately 85% in Stage I, 50% in Stage II, 30 in Stage III and 10% in Stage IV.

The international T.N.M. classification is as follows: (T(tumour size); 1, 2, 3, or 4; thus 1 to 2 cm, 2 - 5 cm, 5 - 10 cm, 10 cm or more in maximal diameter respectively.

N(Nodes); 0, 1, 2; no axillary nodal involvement, nodes are involved and are mobile, nodes are involved and fixed respectively.

M(Metastasis - distant): 0, 1 or X; absent, present, uncertain respectively.

Stage I (T_1, N_o, M_o): Breast mass that is 2 cm or less in its maximal dimension, no palpable axillary lymph nodes and no metastasis. There is 80% chance of 10 year survival.[21]
Stag II (T_1, N_1, M_o; T_2, N_o, M_o or T_2, N_1, M_o): Breast mass less than 2 cm in size but associated with mobile axillary lymph node metastasis or breast mass that is 2 to 5 cm in size with or without clinical evidence of axillary lymph node metastasis. There is about 60% chance of 10 year survival. Stage III: Primary breast tumours that are greater than 5 cm in the greatest dimensions or have extended to the skin characterized by retraction dimpling, ulceration or presence of satellite skin nodules or the tumour that has extended to and fixed to the chest wall, fixed axillary nodes. Palpable surpraclavicular nodes or edema of arm indicate Stage III and

chance of 10 year survival is less than 30%. Presence of distant metastasis, regardless of condition of primary tumour and regional lymph nodes makes it Stage IV.[21]

Early diagnosis

This requires regular breast examination, self or by physician particularly those in the high risk group; those 50 or more years old, with breast lump, localized pain in the breast, nipple discharge, chronic mastitis, family history of breast cancer, previous breast cancer, no children or birth of first child after age of 30 years, early menarche (before 11 years), late menopause or postmenopausal obesity. Small tumour of less than 1 cm diameter cannot be palpated particularly in those with large pendulus breast and in such, screening tests like mammography are helpful. Mammographic film - screen technique which deliver 0.2 to 0.3 rads to the skin per exposure is safe. In Xeromammography, lower radiation levels are used and a mammographic image on paper with edge enhancement obtained. It is thought that palpation plus mammography would reduce death rate by at least one third.[22] Other screening methods include thermography which detects increased heat measured by heat sensors and translated into a photographic image out false positives are common.

Specimen of breast lumph should be submitted for histological confirmation and this can be by aspiration cytology and/or biopsy (needle or open). Open biopsy requires anaesthesia. It has the advantage that estrogen receptor assays are easily performed. Frozen section can be done so that mastectomy may proceed if indicated. Urgent paraffin section can also be done.

Occult metastases

Folkman[23] has demonstrated experimentally that tumours can live by diffusion alone until they are composed of approximately 100,000 cells. Survival beyond this stage required vascularization and once vascularization has occurred, metastases are theoretically possible. The incidence of occult metastases varies considerably with clinical stage of disease: Stage I: 20% of patients have

occult metastasis. Stage II; 40% of patients have occult metastasis and Stage III; 70% of patients have occult metastasis. These occur commonly in the lungs, liver and bone and screening procedures for metastases regardless of stage of the disease should include: Chest X-ray film; scrutinize for radiographic evidence of nodular parenchymal lesions, interstitial infiltrates, mediastinal widening, pleural effusion, osteolytic or osteoblastic lesions.

Serum alkaline phosphatase; this isoenzyme originates from liver, bone, intestine, kidney and placenta. It is elevated in 58% to 90% of patients with liver metastasis. Significant elevation of serum glutamic oxaloacetic transaminase are suspicious enough of bone or liver metastasis to indicate need for additional studies of skeleton and liver like bone scan, liver scan, carcinoembryonic antigen and in some cases tomograms. The commonest type of osseous metastasis is osteolytic lesion seen on X-ray when 50% of bone mineral is destroved. There is also increased osteoclastic activity initiated by bone distruction. This produces an increased accumulation of radioacticity (^{99}Tc) on bone scan. However 5 to 10% of bone metastasis of breast cancer are osteoblastic producing thickening of trabecular giving radiographic evidence of irregular dense masses. Some osseous metastatic lesions produce no radiographic or scan evidence of metastasis. These are intertrabecular metastases produced by malignant cells growing into the bone marrow without damage to the adjacent bone. Bone metastasis remains asymptomatic until the metastatic lesions extend to the periosteum causing pain and tenderness in the area. Liver scans are obtained by injecting a sulphar colloid labelled 99 technetium which are taken up by Kuppfer cells of the liver and the radioactivity provides a means of screening the liver. Metastatic lesions destroy or displace kuppfer cells and are detected as filling defects. Liver scans are indicated when serum alkaline phosphatase is elevated. Better results are found with combined liver scan and serum carcinoembryonic antigen (CEA) a glycoprotein which although not

detectable in all breast cancer patients, is a valuable tool for clinical monitoring of the patient. High titers of C.E.A. indicate poor response to treatment, high frequency of disease recurrence, and/or presence of disseminated disease especially hepatic metastases. All patients with positive liver scan and C.E.A. greater than 9ng/ml had liver metastasis. Focal defects on the liver scan that are not associated with significant elevation of C.E.A. should be further evaluated by percutaneous needle biopsy, peritoneoscopy or laparotomy.

Treatment

There is at present a disquiteting uncertainty as to the best treatment of breast cancer.[18] Surgery, radiotherapy, hormonal manipulation, chemotherapy and immunotherapy or various combinations of these have been used. Radical mastectomy and simple mastectomy with radical radiotherapy have each their advocates, and reports do not suggest that the results are very different, provided the neoplasm can be categorised as belonging to stages I or II. When stages III and IV are reached, the interests of the patient are best served by simple mastectomy, radiotherapy and, provided the tumour is hormone stimulated, endocrine surgery.[4]

Surgery

The primary aim of oncologic surgery is to reduce the tumour burden to a number of viable cells which are entirely destroyable by host immunological factors, systemic anticancer agents or a combination of both.[24] In case of breast cancer, at the end of the spectrum of opinion, there are those who, in addition to performing the classical radical mastectomy, remove the internal mammary glands after thoracotomy and the supra-clavicular glands after division of the clavicle. At the other end of the spectrum there are those who are content to remove only the actual tumour (Lumpectomy) together with a reasonable margin of breast tissue and to follow this with radiotherapy.

Radical mastectomy

In 1894, William Halstead described 50 cases of breast cancer operated on at the Johns Hopkins Hospital and introduced radical mastectomy. The operation is based on the assumptions that axillary nodes may contain metastases in the absence of supraclavicular, internal mammary or hematogenous metastases.[19] The classical radical mastectomy (Halstead) involves removal of the breast itself with a large portion of the skin overlying the tumour including the nipple, all the fat and fascia from the lower border of the clavicle to the upper quarter of the rectus abdominis including its sheath and from the sternum to the anterior border of the latissmus dorsi, the pectoralis major with its fascial sheath, the pectoralis minor and the clavipectoral fascia and all the fat and fascia and lymphnodes of the axilla. The axillary vein, cephalic vein and the long thoracic nerve of Bell to serratus anterior, are specifically preserved.

Handley and Thackray[25] described involvement of the internal mammary nodes at or even before involvement of the axilla and described the technique of interval mammary node removal. In another study of 500 cases, Handley[26] showed that with the tumou r in the inner quadrant 31% central part 47% of outer quadrant 19% and involvement of the internal mammary nodes. Further more when axillary nodes were positive, the involvement of internal mammary nodes rose to 51%, 59% and 28% respectively. It was, therefore, suggested that the operation of radical mastectomy was inadequate because it ignored internal mammary node involvement and subsequently extended radical mastectomy was developed.

Extended radical mastectomy

Involves division of the second, third and fourth costal cartilages close to the sternum and then half an inch laterally and through a trapdoor thus fashioned, the internal mammary vessels are ligated and dissected out together with the lymphatics. It is extensive mutilating procedure carrying high mortality rate and damage to the pleura is common.

Modified radical mastectomy

In this operation developed by Patey,[27] pectoralis major is not removed but retracted and the pectoralis minor removed. The axillary contents are cleared as in the Halstead operation. Its main advantage is cosmetic in that the axillary fold is maintained and there is no hallowing below the clavicle. When there is a deficiency of skin, it provides an ideal bed of muscle on which skin grafts will take readily. It also gives a stronger and more useful arm.[20]

Hagensen[28] recommended preliminary trippled biopsy of the breast, apex of the axilla and internal mammary nodes, which if positive indicated that the tumour was inoperable and simple mastectomy followed by early radiotherapy was done.

Simple mastectomy

Indications for simple mastectomy include carcinoma-in-situ, cystosarcoma phylloides and fibrosarcoma. In these it is important to totally remove the breast tissues to avoid recurrence. McWhirter[29] pioneered the procedure of simple mastectomy plus radiotherapy and hypothesized that in early cases when the disease has not spread to the axilla, dissection of the axilla is unnecessary. On the other hand, 48% of patients who have axillary lymph node involvement will have either the supraclavicular or internal mammary lymph node involvement and in such patients axillary clearance is illogical. He therefore, advised simple mastectomy to remove the primary neoplasm and radiotherapy to treat axillary, supraclavicular and internal mammary lymph node involvement. In such patients Iodine preparation of skin and postoperative adhesive strapping of the wound should be avoid because skin irritation produced lowers skin tolerance to irradiation, skin removal should be minimal to avoid wound tension or skin grafting in order to give adeuate irradiation, undermining of skin flaps should be minimal, pectoral fascia should not be removed unless the tumour is adherent to it. This will help avoid post irradiation

fibrosis in the muscle. Drainage of the wound should be carried through the lower end of the main wound not through the stab wound to confine the area of irradiation. In stage III cases, where the tumour is fixed to the pectoral muscle, part of the muscle should be removed along with the breast.

Bruce[30] has shown that there was no difference in survival rates between radical mastectomy and simple mastectomy followed by radiotherapy. Haaur[31] considers that lymphnodes far from being inert traps infact play such a vital role in the host response to cancer that their removal when free from tumour may be detrimental to the host.

Simple mastectomy is also indicated in a patient with carcinoma of the breast and early distant metastases, who has a primary lesion that is ulcerated and foul or is threatening to ulcerate and patient will benefit by palliative removal of the breast sometimes called toilet mastectomy. Such local management can contribute to the patients comfort and her social acceptability. In this instance it is not necessary to remove the breast in its entirety. It is important that primary closure is possible and that gross tumour will not be transected, for little is gained if prompt primary healing is not achieved.[20]

Simple mastectomy plus radiotherapy is contraindicated in; very obese or aged patients, those with arteriosclerosis, those with tuberculosis and those with primary neoplasm in the axillary tail of the breast.[4]

Lumpectomy plus radiotherapy

There may be a place for segmental excision in those with early tumours.[19] However, it has been found that 61% of patients after lumpectomy developed local recurrence while only 16.9% with radical mastectomy with or without radiotherapy has local recurrence. Thus lumpectomy is an inadequate procedure.[19]

Radiotherapy

This is usually given by a high voltage machine, a linear accelerator or by a cobalt^{60} unit. Although it may be used alone in

those women who adamantly refuse mastectomy, in general it should always be combined with local tumour excision if a reasonable chance of local control is to be expected. Adjuvant radiotherapy after mastectomy may be given immediately to the skin flaps and lymph node areas or the development of recurrent disease may be a waited. For patients with stage I disease there is no indication for radiotherapy nor is there any indication for radiotheray to the axilla after adequate surgical clearance, but irradiation of the internal mammary chain in patients with involved axillary nodes is indicated. Handley and Thackray[25] showed that about 33% of patients with involved axillary nodes had involved internal mammary nodes. For stages III and IV irradiation of the primary site leads to considerable regression of the tumour. Some stage III tumours may be made operable after pre-operative radiotherapy. It may also be used for isolated bone metastases but not for pulmonary metastases as it produces extensive fibrosis.

Flap recurrence is closely related to nodal status and radiotherapy should be considered where more than four nodes are involved. Therapeutic radiotherapy after surgery may be required for recurrent disease. For large exillary nodes appearing after simple mastectomy, surgical resection provides more certain control of the disease. The reluctance to use radiotherapy is based on its considerable morbidity both early and late. Edelstyn[32] described phenomenon of radiation - enhanced local recurrence in about 2% of patients. However, with improved technology, in recent years it has been demonstrated that 6000 to 7000 rads might be given without unacceptable toxicity to normal tissue and obtain excellent local control by radiotherapy or lumpectomy plus rediatherapy (a less than 7 to 10% rate of recurrence in patients in clinical stage I and II) with good cosmesis and a low incidence of radiation pneumonitis and radiation induced fractures.[21]

Lee S.H.[33] studied the estrogen receptor activity of individual cancer cells in frozen sections using a flourescent steroid histochemical technique and found that most human breast cancers are composed of heterogenous tumour cell populations in terms of estrogen receptor level. In the majority of cases the predominant malignant cells are estrogen receptor negative. The number of estrogen, receptor positive cancer cells is zero to 9% in about half of the tumours. There are no 100% hormone - responsive tumours in human mammary carcinomas although 15% of tumours are 100% hormone - independent. Only about a quarter of cases have 50% or more of the total cancer cells estrogen positive. This explains the clinical observation. Primarily hormonal manipulation includes; exogenous administration of estrogen, Oophorectomy, adrenalectomy, hypophysectomy and administration of anti-estrogen drugs. Adrogens, progesterones and corticosteroids are generally reserved as second line therapy.

When estrogens are used, pharmacologic doses, such as 15mg/day of diethyl stilbesterol or 3mg/day of ethinyl estradiol are usually given. The highest response rates are seen in patients who are more than 5 years beyond menopause and almost no responses occur in women treated less than 1 year after the last menstrual period.[34] Similarly, premenopausal women only rarely respond even when large doses are given. Patients in whom the disease progresses after unequivocal response to estrogen therapy may have a second response when estrogen is discontinued. This had been observed in 9 to 30% of patients who respond to estrogens.[34]

Oophorectomy

In theory, elimination of ovarian production of estrogen in the premenopausal women might delay growth of disseminated cancer cells and such stimulus may allow host defense mechanisms to contain growth or induce cytotoxic effects. After oophorectomy or the administration of estrogen, the fraction of cells synthesizing D.N.A. falls in patients who respond to the endocrine therapy, but not in

patients who fail to respond.[34] Histologic evaluation of biopsy specimens taken from these patients suggests that death of tumour cells has occurred and that the shrinkage of mass lesions after hormone therapy cannot be adequately explained as merely the entry of tumour cells into a resting state.[18] Thus regression of local lesions and relief of pain occur. Osteolytic lesions may calcify and pathological fractures heal.[18]

Oophorectomy is effective in about 30 to 50% of pre-menopausal patients and has been shown to delay the course of cancer and provide the patient a longer interval of free clinical disease, delays the onset of recurrence but does not delay death. Patients younger than 35 years of age reportedly have a lower response rate as do women in the first post-menopausal year. The response rates in women castrated more than one year after menopause are almost all less than 10%[35] Suppression of urinary estrogens is as complete after ovarian irradiation as it is after surgical oophorectomy, but there is a latent period of 3 to 6 months before the lowest levels are achieved. Ovarian irradiation may be as effective as surgical castration.[36]

Remission of the disease following removal or irradiation of the ovaries implies that the tumour is sensitive to hormone deprivation, and that, when re-activation of growth occurs, adrenalectomy or hypophysectomy may be beneficial and this is so in about half of the premenopausal patients who have had response to oophorectomy. The response rate is lowest in the first 4 to 5 years after menopause.

Adrenalectomy and hypophysectomy

Assessment of breast cancer tissue for presence of both estrogen and progesterone receptors is important before any of these major endocrine ablation is done.[37]

Both adrenalectomy and hypophysectomy are thought to be effective after menopause or after oophorectomy because they remove persistent, low levels of estrogen available to the growing breast

tumour. After menopause for oophorectomy there is an abrupt fall in urinary estrogen but these levels may rise gradually to premenopausal levels five years later.[38] There is little evidence that the two procedures affect human breast cancer differently and there is little to choose between them. However, both are major operations associated with a considerable mortality and morbidity.[18] Although they offer a 50 percent chance of relief of pain, there is no more than a 30% prospect of a worthwhile remission of the disease. After adrenolectomy patient should be maintained on cortisone while after hypophysectomy, 25 to 80% of patients require cortisone, thyroxine and vasopresin. One hesitates to advise an operation which may have so little to offer in a patient who has probably only a few months to live. Hypophysectomy may be incomplete and ineffective. The operation may be complicated by rhinorrhea, meningitis or diabetes insipidus. Neither adrenolectomy nor hypophysectomy was done in any of the patients in this study.

Anti-estrogen drugs

(1) Tamoxifen is a nonsteroidal anti-estrogen that acts by competing with estrogen for binding sites within the cytoplasm of the cancer cell and when transferred into the nucleus, inhibits vital cellular processes. Tamoxifen is associated with minimal toxicity.[21] Ingle et al[40] preferred tamoxifen as the initial hormonal therapy for postmenopausal women with advanced cancer of the breast. However, he states that other authors suggest that tamoxifen should be considered the endocrine therapy of choice for both premonopausal and postmenopausal patients.

(2) Aminoglutethimide

Is a derivative of the hypnotic glutethimide and blocks estrogen biosynthesis at two separate sites, the adrenal cortex and extra-adrenal tissue.[41] In the adrenal, aminoglutethimide interrupts the conversion of cholesterol to pregnenolone and conversion

of cholesterol to pregnenolone and subsequently reduces production of the major estrogen precursor androstenedione (Medical adrenalectomy). In peripheral tissues, aminoglutethimide blocks the formation of estrogens by inhibiting the aromatizing enzymes responsible for conversion of androstenedione to estrone and this has been reported by Santen et al[42] as the main effect of aminoglutethimide. It is relatively safe. Transient lethargy and skin rash may develop but soon disappear without stopping the aminoglutethimide. Hypothyroidism may develop requiring thyroid replacement therapy. Small doses of Corticosteroid must be administered to inhibit the reflex rise in ACTH secretion which would occur following aminoglutethimide alone.[41] Aminoglutethimide and hypophysectomy produce similar results. The effects of aminoglutethimide are readly reversible and this is important since most women will later require multiple drug chemotherapy for treatment of advancing disease. The stress induced by these cytotoxic agents might be tolerated better in patients with intact pituitary-adrenal axis than in those compromised following hypophysectomy or adrenalectomy.[41] Aminoglutethimide is a reasonable second therapy in postmenopausal women.

Androgen therapy is of doubtful value and has been largely discarded.[18]

Tumour regression with corticosteroid alone is generally accepted to be only about 10 to 20% and large doses are required, frequently producing cushinoid side effects.[41] Adjuvant castration together with prolonged administration of prednisolone could be used in patients with estrogen receptor positive tumours particularly if they refused chemotherapy.

Chemotherapy

This is effective in both estrogen receptor positive and estrogen receptor negative tumours. The principle chemotherapeutic agents used are: the alkylating agents, cyclophosphamide, melphalan,

thio-Tepa; antimetabolites; 5-Flourouracil, methotrexate and others like vincristine, adriamycin and prednisolone. These have been used separately and in various combinations.

Combination of cyclophosphomide methotrexate and 5-Flourouracil (C.M.F.) is the preferred chemotherapy for premenopausal women. Five year results for C.M.F. randomized study carried out in Milan have confirmed the usefulness of the combined modality over radical mastectomy alone in terms of recurrence free survival and total survival and its use is advised in all patients with histologically positive lymph nodes.[43] It is a safer combination to use and alopecia requiring temporary use of a wig occurs in less than 10% patients. However, combination containing adriamycin like, cyclophosphomide, adriamycin and 5-Flourouracil produce frank alopecia and prolonged administration of regimes containing adriamycin has been associated with cardiomyopathy and congestive heart failure. Adriamycin containing regimens are therefore reserved only for very high risk subjects like those with more than 3 positive nodes.

Hoogstratren et al[44] used weekly repeated combination of cyclophosphamide, 5-Flourouracil, methotrexate, vincriatine and prednisolone; the same drugs in a course of 5 days repeated every four weeks. For 283 patients the response rates were 59% for weekly combination 40% for adriamycin. Drug toxicity was the limiting factor in all regimens.

Immunotherapy

Active non-specific immunotherapy with BCG has been used for breast cancer alone or in combination with chemotherapy. Gutherman et al[4] reported remission rate of 75% with chemoimmunotherapy using cyclophosphamide, adriamycin and 5-Flourouracil with BCG by scarification. Controlled trial with or without BCG showed that remission and survival for patients with chemoimmunotherapy was higher than chemotherapy alone.

Corynebacterium Parvum given 4mg weekly in combination with chemotherapy has shown that survival for patients is superior to

chemotherapy alone.

Levamisole given after radiation in a dose of 150mg orally three times weekly every alternative week has also shown to increase the duration of remission and survival rates.

Terminal care is needed for the very advanced or recurrent breast cancer patients and the objective of such treatment is improving the quality of life by symptomatic relief. This has been summarised by Baum.[31] as follows:

Problems	Action
Skeletal pain	Adequate analgesia Radiotherapy to solitary metastases
Pathological fracture	Internal fixation, Radiotherapy
Dyspnoea: (i) Effusion (ii) Lymphangitis (iii) Carcinomatosis	 Tube thoracostomy, intrapleural Cytotoxic drugs Corticosteroids
Hepatic metastases: (i) Pain (ii) Anorexia	 Corticosteroids Corticosteroids
Cerebral metstases	Dexamethasone, Radiotherapy
Anaemia (Bone marrow infiltration)	Blood transfusion, Androgens, Corticosteroids
Hypercalcaemia	Forced diuresis, Corticosteroids, Calcitonin, Phosphates (Mithramycin)
Progressive local/Regional Recurrences	Local Radiotherapy
Lymphoedema arm	Elastic bandage, Physiotherapy, Pneumatic Compression

Other Considerations

Of all breast cancers, 2.8% are associated with pregnancy. Breast enlargement during pregnancy tends to shield breast masses from the examining fingers and accompanying vascular and lymphatic changes often allow early growth and dissemination before the disease can be detected. These changes are associated with poor prognosis. Haagensen[28] advised that with early pregnancy it is reasonable to abort. However, White[46]

states that abortion does not benefit the subsequent course of breast cancer. Most opinion on third trimester pregnancy suggests that a decision to proceed to premature delivery by cesarean section or induced labour depends on the patient's wishes for a child and the imminence of fetus. There is no threat to the unborn child since cancer cells that traverse the placenta and involve the fetus are practically unheard of.[46] Chemotherapy however, is harzardous to fetal development and there is a strong risk of fetal anomalies with both antimetabolites and alkylating agents. Therefore, if mastectomy is done early in pregnancy and positive nodes necessitate adjuvant chemotherapy, the pregnancy should be terminated.

Bilateral breast cancer is occasionally encountered. However, it is sometimes of the contralateral breast, most of these are non-invasive. He concluded that using these other modalities for detection beyond clinical examination would seem to indicate that the incidence of bilateral simultaneous primary breast cancer could be as high as 12%. The incidence of subsequent (Metachronous) primary cancer in the contralateral breast is about 7%.[47]

Incidence of bilateral breast cancer is particularly high in patients with positive family history of breast cancer particularly if it was bilateral in a premenopausal mother or sister or if the cancer of the first breast was multicentric or there is evidence of atypical changes found on biopsy of the remaining breast. Development of cancer in the second breast almost halved the expected survival of the patient. Pack[48] advocated routine prophylactic contralateral mastectomy. However, Leis[47] believes this should be reserved for women with a high risk for developing cancer in the remaining breast and it should be delayed for 3 to 6 months after the initial mastectomy to reduce emotional reaction and operative morbidity.

Some authorities believe that the other breast has a strong psychological support for many who cherish it as a sign of feminity and an important psychosexual symbol. In this context there is no

place for bilateral mastectomy or prophylactic mastectomy. mastectomy with radiation for an early lesion in the remaini or if the disease is already advanced than radiation, chemothe with or without hormonal manipulation have been suggested.[48] It been possible to do bilateral breast reconstructions following bilateral mastectomy with satisfactory results.

In this study treatment, was offered according to the clinical stage. Most patients presented late. 68% were having stage III, IV, bilateral or recurrent breast cancer at presentation (table 5). Palliative measures include; Toilet mastectomy done in 10 patients, lumpectomy in 3 patients, of these, radiotherapy was added for 9 patients. Oophorectomy was done in 6 patients and prednisolone was administered to 2 patients. Of 32% operable patients with stage I or II breast cancer, modified radical mastectomy was done in 6 patients, radical mastectomy 1 patient. Radiotherapy was added to 6 patients, prednisolone was administered to 2 patients (as shown in table 6).

None of the patients received chemotherapy or immunotherapy.

One patient who had a advanced bilateral ulcerated breast cancer died post-operatively. Good response to treatment (without recurrence by May 1982) was seen in 8 patients who presented with stage I or II breast cancer, and 4 who had stage III. 12 patients who had advanced breast cancer (stage III, or bilateral or recurrent) showed poor response. They were weak and wasted at time of discharge (table 7).

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NON-TOXIC SIMPLE GOITRE

SUMMARY

Ten patients of non-toxic goitre are presented. One of the 10 patients is presented in detail. Discussion of incidence, etiological factors, pathology, clinical manifestation, investigations and management and review of literature on non-toxic goitre are presented.

INTRODUCTION

Non-toxic goitre is an enlargment of thyroid gland which maintains euthyroidism. An enlarged thyroid gland is palpable and for the adult, it weighs more than 30 grams.

Goitre can occur at any age in a person living in an endemic area. In areas where the incidence is high it is not uncommon for babies to be borne with goitre. However, the highest incidence of endemic goitre in most goitrous areas occurs in girls 12 to 18 years of age, and in boys 9 to 13 years of age.[1] Adesola recorded a female to male ratio of 10:1.[2]

Non-toxic goitre can be diffuse or nodular (multinodular or uninodular) and either form is found endemically (prevalence in excess of 10% of population) or sporadically, the difference being statistical and arbitrary.[1]

In Tanzania goitrous areas have been reported and include, mountainous regions of North-Western Tanzania bordering Zaire and Ruanda, Mwezi area in the highland area of Western Tanzania.[3] Kelly and Snedden[4] reported large central part of Tanzania as goitrous area. Latham[5] reported on Ukinga division of Njombe district in Southern Tanzania and found a goitre incidence of 75.9%.

In this study of 10 non-toxic goitre patients; age incidence ranged from 15 years old (youngest) to 43 year old (oldest) 'table 1'. 7 patients (70%) were between 20 years to 40 years of age. This reflects more of late presentation other than the age of disease onset since most patients (90%) presented with grade 3 thyroid enlargement (table 6).

There were 8 females and 2 males, a ratio of 4:1 (table 2).

CASE REPORT

A.H., I.P. No.215277, a 30 year old female, Mngoni by tribe from Songea was seen at the outpatient department with history of anterior cervical swelling of 18 years duration. The swelling which was noticed when she was 12 years old, progressively increased in size, disfiguring the neck and causing difficulty on breathing for the last 6 months and difficulty or swallowing solids for 4 months.

There was no other member of her family having a similar illness. He usual diet included: rice, meat and beans. She did not smoke and did not drink alcohol.

She denied history of sudden increase in size of the swelling. She gave no history of heart palpitations, increased appetite or loss of weight. She also denied history of bone pain anywhere in the body.

The patient had been to many hospitals for treatment. She was given various medications but did not have any previous operation on the swelling. When she came to Dar es Salaam she was advised to come to Muhimbili Medical Centre for treatment.

On examination she was found to be generally in good condition. She was well naurished and calm. She was not pale, not jaundiced and had no lymphadenopathy. Blood pressure on admission was 110/70, pulse 72/min. regular, temperature 37°C, respiration rate was 16 per minute.

A big anterior cervical swelling measuring about 10 cm by 7 cm was seen mainly on the right side. The swelling moved with swallowing. Skin over it looked normal.

On palpation the swelling was found to be nodular, non-pulsatile and non-tender. It was firm in consistancy and mobile. The trachea was slightly shifted to the left of the midline. Cervical glands were not palpable.

There was no bruit detected on auscultation.

She had no tremors, no exophthalmos and no lid-lag. Reflexes were normal.

Cardiovascular, respiratory, gastrointestinal, musculo-skeletal and urinary systems were normal.

A diagnosis of non-toxic goitre (grade 3) of the right lobe was made and because of the associated local pressure symptoms and uncosmetic appearance surgical treatment was advised.

The preoperative investigations included X-ray of the neck and thoracic inlet, full blood picture was requested for and revealed Haemoglobin of 10.7Gm%, white blood cell of 4,900 with normal differential count and, was reported as nomocytic normochromic and platelet count as adequate.

Routine stool and urine examinations were normal.

Preoperative laryngoscopy was done and vocal cords were found to be functioning normally.

Blood grouping and cross match of 2 units of blood was done.

In the theatre, under general endotracheal anaesthesia, the patient was positioned with a sand-bag under the shoulders and a head ring to keep the neck extended. Using a silk thread, the incision outline was marked at about 4 cm above the sternal notch and extending to the sternocleidomastoid on each side. The skin and platysma were incised and using the subplatysmal plane the upper flap was reflected up to the superior notch of the thyroid cartilage, the lower flap down to the level of sternal notch. The skin flaps were retracted with self retaining retractor.

The investing layer of the deep fascia was incised and the infra-hyoid muscles were separated and retracted. The pre-tracheal fascia was incised to expose the thyroid gland.

The right lobe was found enlarged and a nodule in the isthmus was also seen. The lateral surface of the right lobe was gently cleared. The middle thyroid vein was identified, ligated and divided. The right recurrent laryngeal nerve was identified lying posterior to the inferior thyroid artery in the tracheo-esophageal groove. The artery

was ligated and divided while safe-guarding the nerve. The upper pole of right lobe was mobilized and cleared. The superior vascular pedicle was clamped and doubly ligated and divided close to the gland. The lower pole was then mobilized and the inferior thyroid vein ligated and divided. The freed right lobe was medially rotated and posteriorly the right parathyroids (superior and inferior) were identified and preserved. The isthmus was mobilized and excised between clamps thus removed together with the right lobe.

The left lobe was found to be normal and left behind.

Small bleeders were cauterized using diathermy and the wound closed in layers with a corrugated rubber drain placed on the right lateral end of the wound. Chromic catgut 2/0 interrupted suture was used for subcutaneous tissues and fascia. The skin was closed with metal clips. Patient received one unit of blood during operation. After removing the endotracheal tube, the cords were examined and noted to be normal.

Postoperatively the patient was nursed in propped up position and intravenous drip was maintained with 2.5 litres of 5% dextrose for 24 hours and she received Pethidine 100mgs 8 hourly intramuscular injection for 24 hours. An hourly observation of pulse, blood pressure and respiration were ordered.

The patient recovered well. The drain was removed after 48 hours. The metal clips were removed on the 7th postoperative day when the patient was discharged from the ward and advised to attend outpatient clinic for follow-up.

The patient was seen after 2 weeks. She had no complaints and her condition was good. She never came back for second review. Probably she went back to Songea region.

Rest of the presentation is as follows:

Table 1: Age incidence

Age range (years)	No. of patients	Percentages
20 or below	2	20%
20 - 30	4	40%
30 - 40	3	30%
40 or above	1	10%
Total	10	100%

The youngest was 15 years old.
The oldest was 43 years old.

Table 2: Sex incidence

Sex	No. of patients	Percentages
Female	8	80%
Male	2	20%
Total	10	100%

Table 3: Presenting symptoms

Presenting symptoms	No. of patients	Percentages
Anterior cervical swelling	10	100%
Dyspnoea	2	20%
Headache	3	30%
Dysphagia	1	10%

Table 4: Duration of thyroid enlargement

Duration (years)	No. of patients	Percentages
5 years or less	4	40%
5 to 10 years	1	10%
10 to 15 years	3	30%
15 to more years	2	20%
Total	10	100%

Table 5: Forms of thyroid enlargement

Form of thyroid enlargement	No. of patients	Percentages
Diffuse (smooth)	1	10%
Nodular: (a) Multinodular	7	70%
(b) Solitary nodule	2	20%

Table 6: Grade of thyroid enlargement

Grade	No. of patients	Percentages
1	-	-
2	1	10%
3	9	90%
Total	10	100%

Table 7: Indications for surgery

Main indication	No. of patients	Percentages
Unconsmetic cervical swelling	6	60%
Solitary thyroid nodule	2	20%
Local pressure effects (dysphoea, dysphagia +)	2	20%

Table 8: Surgery performed

Operation	No. of patients	Percentages
1. Left lobectomy	2	20%
2. Right lobectomy	1	10%
3. Right lobectomy + isthmus excision	3	30%
4. Left lobectomy + isthmus excision	1	10%
5. Thyroid adenoma excision	1	10%
6. Subtotal thyroidectomy	2	20%

The indications for each opeation were as follows:

1. Left Lobectomy was done on 2 patients. One complained of uncosmetic cervical mass the other had a firm solitary nodeule.
2. Right lobectomy was done on 1 patient who complained of uncosmetic cervical swelling.
3. Right lobectomy and isthmus excision were done on 3 patients. All the 3 patients complained of uncosmetic cervical swelling.
4. Left lobectomy and isthmus excision were done on 1 patient who complained of cervical swelling which was associated dyspnoea and dysphagia of solids.
5. Thyroid adenoma excision was done on 1 patient who had a slitary thyroid nodule.

6. Subtotal thyroidectomy was done on two patients who complained of large uncosmetic cervical swelling.

DISCUSSION

Aetiology

Goitre is produced by a mechanism in which the anterior lobe of the pituitary is concerned as well as the thyroid gland which is acted on by thyroid stimulating hormone (TSH).[6] Decrease in thyroid hormone secretion brings about increased secretion of TSH which in normal subjects re-establishes the production of the thyroid hormones in normal amounts. In turn the thyroid hormones inhibit the production of (TSH). Such a self-regulating mechanism ensures a physiological balance in the secretory activity of the thyroid only if the latter responds adequately to TSH which activates the biosynthesis and secretion of the thyroid hormones. A permanent falling off in thyroid activity results in hypertrophy and hyperplasia of the gland which are initially compensatory processes.[6] These changes may lead to development of simple colloid and nodular goitre which are all stages in the same pathological process, the basic defect being thyroxine deficiency.

Thyroxine deficiency may be due to: (1) Insufficient dietary iodine; (2) Inability of the gland to incorporate iodine which may be caused by goitrogens, metabolic defect in the cells from an inherited enzyme defect, autoimmune disease process, radiation and thyroid trauma. (3) Increased renal clearance of iodine. (4) Increased demand for thyroxine for e.g. at puberty or pregnancy.[6]

Insufficient: Dietary Iodine

Iodine defficiency is the chief factor involved in endemic goitre.[6] In sporadic goitre, however, the cause is usually unknown.[7]

A mean dietary requirement of iodine of 70 µg has been proposed. Ocean water contains approximately 50 µg of iodine per litre and air borne iodine is precipitated in the soil with rain. Factors leading to soil enrichment include; vegetation, iodine absorbing colloids like clay and humus, acidity, fossil and atmospheric precipitation. Thus regions where air, water and soil and therefore, food stuffs are poor in iodine are found to be areas of endemic goitre. This is especially true in the mountainous rural areas where people rely on locally produced food.[6] Iodine deficiency state in these areas is shown by the finding of higher uptake of radio-iodine by the thyroid in endemic areas (74%) than in the nonendemic areas (37%) and there is low urinary excretion in the endemic areas (15 - 40mg/day) than in the non-endemic (over 100mg/day).

In Mwezi area of Western Tanzania, McGill and Taylor[3] found low excretion of stable iodine in urine with low levels of plasma inorganic iodine indicating the role of iodine deficiency there.

Dietary iodine is absorbed in the form of inorganic iodide and is either excreted by the Kidneys or taken up by the Thyroid gland and a normal thyroid is able to concentrate inorganic iodides in a ratio of about 20:1. Failure to do so will limit the rate of synthesis of thyroid hormone. The uptake is under the influence of TSH which also stimulates every step in the thyroxine synthesis and secretion and is an autoregulatory mechanism.

Cell membrane transport is energy dependent, and can be depressed by thocynates and perchlorate and saturated by iodides.

The trapped iodide is then oxidized to iodine under the influence of peroxidase and takes part in the substitution reaction replacing the hydrogen atoms at the positions 3 and 5 in tyrosine giving rise to 3-monoiodo -L- tyrosine (MIT) and 3, 5 - diiodo -L- tyrosine (DIT). This process can be blocked by thiourea derivatives.

Condensation of two 3, 5 - diiodo -L- tyrosine residues gives rise to an L- thyroxine residue, and condensation of a 3 - mono-iodo -L- tyrosine with a 3, 5 - diiodo -L- tyrosine residue gives a 3, 3',

5 - triiodo -L- thyronine residue. The greater part of the organic iodine is stored in the form of thyroglobulin, in the colloid substance of the thyroid follicles. The iodinated amino-acids are set free by hydrolysis of thyroglobulin by a thyroid protease.

Thyroxine (T4) and 3, 3', 5 - triiodo -L- thyronine (T3) are the only iodinated amino-acids constantly circulating in the blood under normal physiological conditions. Free 3 - monoiodo -L- tyrosine and 3, 5 - diiodo -L- tyrosine are diodinated by enzyme action to form L- tyrosine and iodide within the thyroid gland where the iodides are again used for a fresh cycle of hormone synthesis.

When the thyroid does not secrete an adequate supply of hormone, it is stimulated by TSH to become hyperplastic and such stimulation occurs, irrespective of the cause of the defficient output.

Goitrogens

Two types of antithyroid substances, both goitrogens, are recognized in terms of their action on the metabolism of iodine; (a) the thiocyanates and perchlorates, whose goitrogenic action can be reversed by iodides, indicating that they must inhibit thyroid concentration of iodides; (b) thiourea, thiouracil and its derivatives, numerous other thiocarbomide derivatives (aminothiazoles and mercaptoimidazoles), aniline derivatives (sulfaguanidine, para-aminosalicylic acid etc) and numerous polyphenols (resorcinol, phloroglucinol etc) all these have a hyperplastic and hypothyroidogenic action which can be reversed by L- thyroxine or excision of the pituitary, but not by the administration of large amounts of iodide.

Barker[8] observed that goitre appeared in patients who had been receiving thiocyanate over a long period for the treatment of hypertentio Thiocyanates and perchlorates do not completely inhibit the penetration of iodide into the thyroid; they appear merely to inhibit the active concentrationmechanism. The incorporation of iodide in organic compounds is not prevented.[9] The thyroid hormones are synthesized normally, but only in small amounts. On the other hand, the

antithyroid substances in the second group do affect the transformation of inorganic iodine into organic iodide and they inhibit hormone synthesis. They act as thyroid peroxidase inhibitors and prevent the formation of iodothyrosine residues in the thyroglobulin. Propylthiouracil also inhibits the iodination of 3 - monoiodo - L- tyrosine by acting on iodotyrosine linkage in iodothyronine.[10]

In any case, the reduction or inhibition of hormone biosynthesis results in a decrease in the level of circulating hormone and an increase in the secretion of pituitary TSH, giving rise to goitre.

Dietary goitrogens

Discovery in 1928 of goitre in rabbits fed on cabbage was proof of existence of goitrogens.[6] It was later established that other vegetables of the genus Brassica such as cauliflower, turnip and Brussel sprouts have goitrogenic properties as well. The goitrogenic effects of soya beans and Groundnuts has been shown in young rats. Presence of a goitrogen was confirmed by isolating 1 - 5 Vinyl 1 - 2 thio-oxarodine from the root and seeds of turnip and from the seed of cabbage, kale and rape.[11]

Clements and Wishart[12] showed that in certain parts of Australia and Tasmania, children drinking milk from cows fed on Marrowstem kale develop goitres which cannot be prevented by the administration of 10mg of potassium iodide weekly. It was later proved that thiocyanate is present in a high concentration in this milk about six times as much as in milk from grazing animals and showed that consumption of the milk leads to the release of radioiodine from the thyroid of rats previously treated with propylthiouracil.[6]

Latham[13] considered cabbage as a factor of endemic goitre in Ukinga, Njombe district in Tanzania but while he related the response to iodine therapy, there was no effect on withdraw of cabbage for 12 weeks.

Drinking of hard water containing large amounts of calcium is an important etiological factor.[6] Calcium is thought to decrease the

metabolic effects of thyroxine and cause a relative iodine deficiency by increasing its demand. It has been shown that a relatively high iodine content of the gland is not necessarily incompartible with the existence of goitre.

Other goitrogenic substances that may cause sporadic form of goitre include: aniline derivatives like sulphaguanidine, para-amino salicylic acid (PAS); polyphenols like resorcinol ointment; thiocarbamide derivatives like aminothiazoles and mercaptoimidazoles, and thiocynates substances which may be ingested by a pregnant mother in the form of medication and result in the development of an infant with congenital goitre.

Enzyme defect

This is inherited as an autosomal recessive trait and the metabolic defect result in impairment to synthesize the thyroid hormone resulting in goitre. Depending on type of specific enzyme defect, the following types are recognized.

Type 1: this follows iodide transport defect in terms of genetic control of active cellular transport mechanism and is believed to be a defect on the synthesis of a specific cell surface protein the iodide permease.

Type 2: is associated with full iodide organification defect.

Type 3: there is partial iodide organification defect associated with nerve deafness (Pendred's syndrome) and the biochemical defect is the formation of hydrogen perioxide.[14]

Type 4: there is iodotyrosine coupling defect.

Type 5: there is iodotyrosine deiodinase defect.

Type 6: there is thyroglobulin iodinated polypeptide defect.

Type 7: there is peripheral target organ refractoriness to thyroid hormone.

Type 8: there is a circulating substance with chromatographic mobility characteristic of T3.

Puberty and pregnancy

Change of hormonal environment occurs during puberty, menstruation and pregnancy which may result in physiological goitre developing. The levels of estrogen may lead to increased iodine clearance while changes in the serum proteins especially in the site for thyroxine may be contributory factor.

Pathology

When the thyroid fails to secrete an adequate amount of hormone, it is increasingly stimulated by TSH and cellular hyperplasia proportional to the degree of defficiency occurs. The pathological changes vary, depending on the severity and duration of stimulation. The enlargement can be hyperplastic, colloid or nodular.

1. Hyperplastic state

The gland is hyperaemic and diffusely enlarged, the epithelium is tall columnar with small follicles containing scanty colloid.

2. Colloid stage

This develops when stimulation is chronic, some follicles undergo process of involution becoming enlarged and accumulate colloid. The epithelium is now low cuboidal like that of a normal gland. The enlargement is diffuse and smooth. This type of diffuse colloid goitre, constitutes a very small proportion of goitre in the tropics.[2]

3. The nodular stage

As the process develops over the years, many follicles become further distended with colloid while others are micro-follicular with mild hyperplasia. Eventually some of the enlarged follicles rupture and fibrosis occurs. In addition the distortion and pressure causes infarction, haemorrhage and necrosis. This combination of degenerative changes leads to nodularity which involves the entire gland. The nodules vary in size and consistency, and may be firm soft or cystic.

The solitary nodule probably represents the first nodule in the gland which subsequently becomes multinodular or may be a dominant nodule in a multinodular gland. Such a nodule may be functional, the so called "hot" nodule and may be autonomous.

The importance of a nodule is in its differential diagnosis from benign adenomas on the one hand and a carcinoma on the other. Nearly half of such clinically solitary nodules are benign.

The weight of the adult thyroid in Europeans and Americans varies between 20 and 35 grams.[15] In the Nongoitrous region in the Congo, DeSmet[15] found the average weight of the normal adult thyroid was 15 to 25 grams. In Nigeria Taylor[16] found the weight of adult thyroid varied between 7 and 21 grams, about half that reported in other parts of the world. It is believed that the low thyroid weight of the negro is partly due to their low body weight and living in a hot climate which reduces the risk of compensatory hyperplasia of the thyroid gland.[17]

Complications of simple goitre include: Trachial obstruction due to gross lateral displacement and compression; haemorrhage into a nodule with acute respiratory obstruction; secondary thyrotoxicosis; and carcinoma occuring in (4 - 17%).[18]

In this study trachial obstruction causing dyspnoea was presented in 2 patients (table 3).

Clinical manifestations

Goitres whose primary cause is a thyroid deficiency are seen most often in pubescent girls. The goitre, often subsides spontaneously during adolescence but may recur during pregnancy and lactation and subside again soon afterward. In areas of severe iodine deficiency goitre tend to develop in early childhood or even before birth in the children of goitrous mothers. Sporadic goitres develop in area where goitre is not common but are clinically indistinguishable from endemic goitres.

In spite of the enormous size of some thyroid glands there are remarkably few symptoms and signs. Pressure effects such as dyspnoea, dysphagia, and pain are uncommon. Sometimes retrosternal extension of the thyroid may result in venous obstruction. In most patients the cosmetic effect of an enlarged thyroid gland is the only reason for the patient reporting to a doctor.

In this study of 10 patients, 8 patients were females and only 2 males (see table 2) the commonest presenting symptom was uncosmetic anterior cervical swelling which was a presenting symptom in all 10 patients. However, additional complaints included; dyspnoea in 2 patients, headache in 3 patients and dysphagia in 1 patient. Varying duration of symptoms before presentation is shown in (table 4).

Clinical examination

This is best done with the patient in a sitting position because palpation of thyroid is more difficult in recumbent position.

A visible goitre is easily recognized but on inspection it is also necessary to assess whether the goitre is symetrical nodular or localized. By palpation its consistency is noted. The surface may be diffuse, lobulated, nodular or present a discrete nodule. Any displacement of the trachea is determined as well as the presence or obsence of enlarged lymph nodes. The goitre or the nodule may be small and deeply placed or hidden by thick and tense overlying prethyroid and sterno-mastoid muscles. It is therefore, essential to flex the head on the neck to enable the exploring fingers to pass under the relaxed sterno-mastoid muscles in order to gain a clear impression of the size, consistency and outline of the mass. By this method the presence of a deeply placed and apparently impalpable thyroid swelling can be demonstrated, whilst the upper margin of a hidden retrosternal mass may be palpated as it rises on swallowing. If displacement of the trachea has been demonstrated, the patient is asked to breathe deeply through the partly opened mouth. Stridor may then be elicited, suggesting the presence of tracheal pressure.

Perez C.[19] recommended the following classification (grading) for endemic goitre:

Group 0: Persons without goitre

Group 1: Goitre is palpable with the head in normal position, but not visible; visible with the head fully extended.

Group 2: Goitre is visible with the head in normal position.

Group 3: Goitre is grossly visible and prominent at a considerable distance.

A specific note is also made for signs of tracheal compression, venous obstruction, eye signs for toxicity, recurrent laryngeal nerve involvement, horners syndrome and presence of lymph nodes. Systemic examination is made for hypothyroidism and hyperthyroidism.

In this study one patient (10%) was found to have diffuse, smooth thyroid enlargment, 7 patients (70%) had multinodular thyroid enlargement at the time of initial presentation and two presented with solitary nodule both were in the right lobe of the thyroid. Forms of enlargement as seen in this study are shown in table 5. 9 patients (90%) had very large thyroid enlargement visible at a distance and disfiguring (grade 3) at time of presentation. Only one patient (10%) presented with visible but moderately enlarged thyroid of grade 2.

Investigations include the following:

1. X-ray of chest and neck to show a substernal or intra-thoracic goitre, calcification and tracheal compression or deviation, barium swallow is indicated for dysphagia.

2. Laryngoscopy is done to examine the vocal cords prior to operation since recurrent laryngeal nerve involvement occasionally occurs with large goitres even without malignant change.

3. Laboratory investigations for thyroid function include: Basal Metabolic Rate (BMR), protein bound iodine (PBI); plasma cholesterol, radioactive iodine tests like thyroid uptake, thyroid clearance rate, primary excretion of radioactive, iodine, and radioactive protein bound iodine; mapping of the thyroid gland with radioactive iodine by means of a scintillation counter.

Thyroxine (T4) and triiodo thyronine (T3) serum levels can be measured.

4. Thyroid biopsy using Vim-Silverman needle or excision biopsy is performed differentiating a nodular goitre from suspected carcinoma.

In this study clinical assessment, pre-operative radiological investigation and laryngoscopy were done in addition to routine investigations like full blood picture, stool and urine microscopy.

Treatment

Prophylaxis

It is generally accepted that the immediate cause of simple goitre is failure of the thyroid gland to obtain a supply of iodine sufficient to maintain its normal structure and function.[20] However, in certain places a goitrogenic substance with a capacity to inhibit the utilization of iodine by the thyroid may be responsible for the occurence of endemic goitre.[1] In such situations treatment with iodide may be of no avail until the source of goitrogen from the diet has been removed.[20]

Large scale programmes of goitre prevention should be preceeded by study of aetiology and the degree of iodine deficiency in that area and treatment with iodine is indicated when goitre has been shown to be due to absolute or relative deficiency of iodine.

The basic principle in mass prophylaxis of endemic goitre is to increase the intake of iodine within safety limits. The most practical method of providing iodine is iodization of salt. Other methods include iodization of water supplies and bread, the periodic administration of iodide tablets and sweets. The level of iodization of salt to be adopted in any area must consider the normal requirements of iodine the degree of environmental deficiency, the presence of goitrogenic factors and the daily consumption of salt per person. A concentration of 1:100,000 (Approx. 72 µg of iodine per day) has been recommended by WHO.[20]

In the tropical countries; the salt is better treated with potassium iodate, not iodide, which is not stable under tropical condition.[2]

Latham[13] recommends use of iodized salt at the national and local level, administration of lugols iodine through schools, dispensaries and other centres.

Complications of iodine therapy include, iodism, iodine thyroiditis and enlargement of the goitre with or without hypothyroidism.

Iodism

Is an allergic reaction to iodine. Acute reaction is manifested by angioneurotic symptoms, from urticaria to haemorrhagic exudates.

Chronic iodism presents like common cold with chronic rhinitis, enlargment of salivary glands and various acneiform exanthemas. It often observed among people over-sensitive to iodine and if therapeutic dose is larger.

Iodine thyroidis

Painful enlargement of the goitre can occur about the seventh day of therapy of large doses of iodine administration.[20] This complication follows the temporary hypersecretion of thyrotropic hormone which causes the sudden formation of iodine-containing colloid in the follicles with a decreased release of thyroid hormone into the blood-stream due to the blocking effect of iodine. It is transitory harmless phenomenon.[20]

Enlargement of the goitre with or without hypothyroidism

Iodine-induced goitre arises because in some people iodine in large doses inhibit the release of thyroid hormone into the blood-stream the shortage of thyroid hormone in the blood, sometimes down to the level of hypothyroidism, leads to a compensatory increase in the secretion of thyrotropin (TSH) which produces enlargement of the thyroid. This type of goitre disappears spontaneously after the administration of iodine has been discontinued and the concentration of iodine in the blood has decreased.

These complications are rare and do not occur when recommended dosage is used.

Medical treatment

Iodine therapy

This is useful in endemic goitre. The simplest prescription is a solution of potassium iodide in water (K.I. 30 µg in 20mls distilled water) given in 4 to 6 drops in a glass of water daily. The widely known preparation is Lugols iodine (Iodine 5mgs: KI, 10.0gm to 100mls of water) which contains 30 µg in one drop; is too high a dose for routine use.

Treatment has to be continuous and a therapeutic effect is usually observed after 6 to 8 weeks of therapy. If there is no response even after 3 months then iodine should be replaced by thyroid preparations.

Thyroid preparations

Therapeutic use of thyroid preparations is indicated for:

1. Adults with large goitres of the diffuse or nodular type containing in addition to hyperplastic and macrofollicular parenchyma, degenerated, haemorrhagic, cystic and fibrotic tissue.

2. Simple sporadic goitre whether due to an innate defect in iodine metabolism, excessive intake of goitrogenic substances of an unknown cause.

3. Thyroid therapy becomes imperative if either endemic or sporadic goitre is associated with manifestations of hypothyroidism.

4. It is particularly indicated in newborn babies with goitre, since iodine is rather slow in its action and may occasionally induce temporary enlargement of the goitre thus aggravating the pressure symptoms.

5. Pregnant women with goitre. It has been shown that the heightened activity of the thyroid gland and the raised serum level of protein bound iodine (PBI) during pregnancy are related to the increase in the thyroxine-binding protein which is induced by the high estrogen concentration in the blood.[21] Miscarriages are common in women with a protein bound iodine level below 6.0 µg per 100ml and it has been observed that when thyroid preparation was administered during pregnancy to a goitrous woman, her goitre diminishes in size and a healthy baby with a normal thyroid is born.[20]

Treatment may be commenced with 50 mg per day, the amount being increased by weekly increments up to a maximum of 300mg per day.

Diffuse hyperplastic goitres respond more readily and completely to thyroid than do nodular goitres.[22] In the series of 50 goitrous

cases treated by Greer and Astwood,[22] 87% of diffuse goitres, 67% of multiple nodular goitres and 67% of single nodular goitre found corresponding figures for the complete disapprearance of the goitres as 44%, 33% and 39% respectively.

Observations during treatment should include pulse rate, size of the thyroid gland, basal metabolic rate, blood cholesterol and serum protein bound iodine. The therapy should be discontinued for 14 days if toxic symptoms develop (thyrotoxicosis factitia) and thereafter resumed with a dose of 25 to 50mg smaller and continued until the goitre diminishes in size. In about 50% of people, the withdrawal of thyroid therapy result in a recurrence of the goitre after about the same period of time.[20] In some patients, goitre may develop again despite iodine therapy, particularly in women of child-bearing age and people with a biologically inferior thyroid. Relapse is also common with sporadic goitre.[20] In such cases thyroid therapy should be continued for the rest of the patients life. Therapy is continued in cases of hypothyroidism with dosage adjusted as is required.

Development of hypothyroidism (Farquharson's phenomenon)[20] may occur after withdrawal of thyroid treatment. This is due to temporary but prolonged inhibition of the secretion of thyrotropin after the exogenous thyroid hormone has been used up. Similarly, hyperthyroidism after withdrawal of thyroid therapy is due to the rebound hypersecretion of thyrotropin, followed by hyperactivity of the thyroid due to the lag in the adjustment of thyrotropin secretion.

In patients who vomit after taking thyroid, thyroxine or triiodothyronine can be used instead of dessicated thyroid. According to Werner,[23] 0.1gm of thyroid is approximately as effective as 300 μg of L thyroxine sodium or 75 μg of L triiodo thyronine orally and represents the average maintainance dose for treatment of myxoedema. The maximum dose for the therapy of myxoedema with L thyroxine sodium is about 500 μg and with L triiodo - thyronine about 125 μg per day.[23] In this study oral L thyroxine was given postoperatively to one patient

who had subtotal thyroidectomy. The patient was stable and has been maintained on 0.1mg /- thyroxine daily. The other patients did not need thyroid replacement therapy in the immediate postoperative.

Surgical treatment

Surgical treatment is indicated in goitre:

(a) When there are signs of compression

(b) When thyrotoxicosis supervenes on a nodular goitre

(c) When there is malignant transformation of the goitre and

(d) For cosmetic reasons.

Signs of compression depend upon the part which is being compressed. There may be compression of the larynx and trachea, leading to dislocation of these organs with dyspnoea, cough, stridor and hoarseness. In children and adolescent, the trachea is relatively soft and narrow and even a small colloid goitre can induce signs of compression. There may be compression of the blood vessels; the internal jugular and subclavian veins and, in the case of right-sided substernal goitre, the superior vena cava as well. In such cases there will be dilatation of the subcutaneous veins, cyanosis and oedema of the face, neck or extremities and a feeling of congestion in the head upon bending.

Dysphagia develops when there is compression of the esophagus but this is rare.

The goitre may press on the adjoining nerves; on the recurrent laryngeal nerves producing a low husky voice, on the cervical sympathetic producing Horner's syndrome, on the vagus producing paroxysmal bradycardia and syncope, cervical and brachial nerve plexuses may be compressed, leading to occipital headaches, pain in the right shoulder paraesthesia and weakness in the right arm.

In endemic goitre areas, hyperthyroidism (toxic adenoma) in patients with pre-existent goitre is said to be common and to account for about 20% of all patients with hyperthyroidism[20] and surgery in the best treatment for such toxic adenoma.[20]

The major arguement concerning the management of nodular thyroid is related to the possibility of carcinoma in the gland. Sokal,[18] estimated the incidence of malignancy in nodular goitre to be between 4 and 17%. The criteria for malignancy are:[24]

(a) Signs and symptoms of compression

(b) Progressive growth or recent rapid growth in one region

(c) Fixation of the gland to the skin or other neighbouring structures.

(d) Vocal cord paralysis

(e) Horner's syndrome and

(f) Hard cervical lymph nodes.

Operation is also indicated for solitary nodules especially in children and over the age of 40 years in men. Radioactive scintillation scan may also contribute to the diagnosis. Robinson,[25] found the incidence of carcinoma in operated cases for hot nodules as 6.8% and for cold nodules as 31.5%.

However, most patients come to the hospital concerned with cosmetic reasons and surgery is often indicated for moderate to large goitre.[2]

Surgery usually consists of

1. Subtotal thyroidectomy when an amount of gland equal to the normal gland is left on each side.

2. A lobectomy and isthimusectomy is sufficient when only one lobe is affected.

3. Single nodule enucleation (excision) is not satisfactory and is associated with high incidence of recurrent goitre.[2]

4. Occasionally total thyroidectomy is done.

Thyroid surgery is better done under general endotracheal anaesthesia. A sandbag should be placed under the patient's shoulders this will help extend the neck and neck structures become easier to expose.

An adequate incision is positioned approximately two fingers above the sternal notch but higher if the gland is markedly enlarged and is made from one sternocleidomastoid muscle to the other. The incision is deepened through the platysma muscle and a plane is developed subplatysmally to raise the upper flap upward to the superior notch of the thyroid cartilage and lower flap to the level of the sternal notch. The fascia over the medial edge of the sternocleidomastoid muscles is incised.

A midline longitudinal incision is made to separate the prethyroid strap muscles (sterno-hyoid and sternothyroid). These muscles are divided transversely high across the upper third to avoid injury to the muscle's nerve supply (ansa hypoglossi).

Mobilization of the gland is further carried out after incising the lateral attachment of the pretracheal fascia to the lateral border of the gland. The thyroid is then displaced toward the midline and countertraction is produced by lateral traction on the sternocleidomastoid mascle and the pretracheal layer of deep carvical fascia becomes stretched. Opening of the fascia permits entrance to the post-thyroid space and the gland falls into the operative field. The middle thyroid vein is usually encountered and is ligated and divided.

Recurrent laryngeal nerve should be identified and protected. The nerve usually travels in the trachec-esophageal groove posterior to the inferior thyroid artery but may be anterior or interwined in the between the branches of the inferior thyroid artery. The artery may have to be ligated and divided a way from the gland to avoid injury to the nerve.

The superior pole, including the superior thyroid artery, is exposed by downward and inward retraction of the thyroid gland. The superior vascular pedicle contains three branches of superior throid artery (anterior, posterior and arcuate) and each arterial branch is accompanied by two small veins. The external branch of the superior

laryngeal nerve is intimately related to the superior vascular pedicle. The pedicle should be doubly ligated close to the superior pole of the thyroid to avoid injury to the nerve. Superior laryngeal nerve innervates the cricothyroid muscle and if it is injured, the voice becomes hoarse and weak.

The inferior pole of the thyroid is mobilized and then the inferior thyroid vein is ligated and divided.

Parathyroids must be identified and preserved. Downward and medial rotation of the mobilized thyroid lobe brings into view the posterior aspect of the thyroid capsule. The superior parathyroids are usually seen near the junction of the upper and middle sections of the thyroid gland; the inferior parathyroids are usually close to the junction of the inferior thyroid artery and the recurrent laryngeal nerve.[26]

Excision of the thyroid gland can be done when the thyroid lobe has been mobilized, its major vessels divided, recurrent nerve exposed, parathyroids preserved and isthmus divided between clamps. After determining the amount to be removed, haemostats are placed along the posterolateral capsule of the thyroid, outlining the amount of thyroid tissue to remain and the thyroid to be removed is excised. Haemostasis is secured and the lateral thyroid capsule of the remaining thyroid is sutured to the pretracheal fascia. Both lateral thyroid lobes are removed in the same manner, with the pyramidal lobe being excised together with the left lateral lobe.[26]

Prethyroid muscles are approximated with mattress sutures and skin, together with the platysma, is closed with metal clips with a drain brought put at the angle of the wound.

In this study, 6 patients were operated for cosmetic reasons alone, 2 other patients had, in addition to uncosmetic anterior cervical swelling, local pressure effects of the swelling. Solitary nodule was indication for surgery in two patients as shown in table 7.

Operations done are shown in table 8; left lobectomy was done in 2 patients, right lobectomy in one patient, right lobectomy with

isthmusectomy was done in 3 patients, left lobectomy with isthmusectomy was performed in one patient, thyroid adenoma excision in one patient, subtotal thyroidectomy in two patients and one of the two patients had a huge multinodular goitre (see illustration 8). The patient was dyspnoeic at presentation. Illustration 9 shows the same patient 4 weeks after subtotal thyroidectomy.

Haemorrhage

This can be immediate or delayed. Immediate haemorrhage occur when the patient emerges from anaesthesia. Vigorous coughing, straining and vomiting produce increased venous pressure, allowing insecure ligatures to dislodge or insignificant vessels to bleed profusely. Delayed bleeding may occur 2 or 3 days after operation and is usually the result of oozing from small veins.[26] Haemorrhage is manifested by blood from the drain or wound, swelling of the neck or respiratory difficulty. If hemorrhage produces respiratory obstruction, the wound must be opened immediately to allow respiration and haemorrhage can be controlled.

Respiratory obstruction

This may be as a result of haemorrhage but more often is the result of laryngeal edema producing an inadequate airway. Occasionally, respiratory obstruction may be the result of vocal cord paralysis secondary to injury of the recurrent laryngeal nerves. Tracheal collapse may also occur following excision of a huge goitre. Impending obstruction is heralded by stridor and this must be recognized early and immediate measures to ensure an adequate airway either by intubation or tracheostomy are carried out whatever the cause.[26]

Parathyroid Insufficiency

This is temporary in about 0.94% and permanent in about 0.5% of the patients,[20] the former is caused by surgical trauma, the latter the result of removal of all parathyroid tissue. Hypoparathyroidism

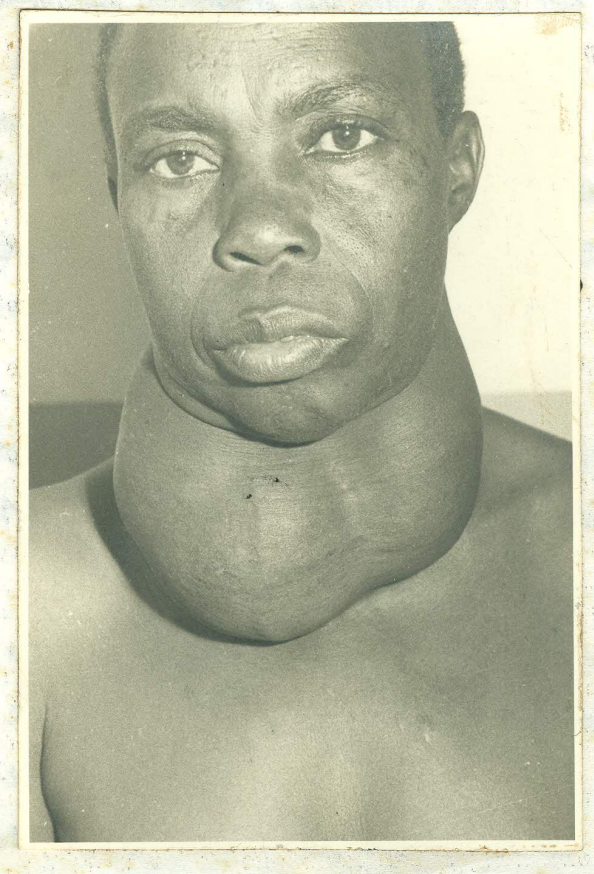


Illustration 8:

Shows a huge multinodular goitre involving both right and left lobes of the thyroid.

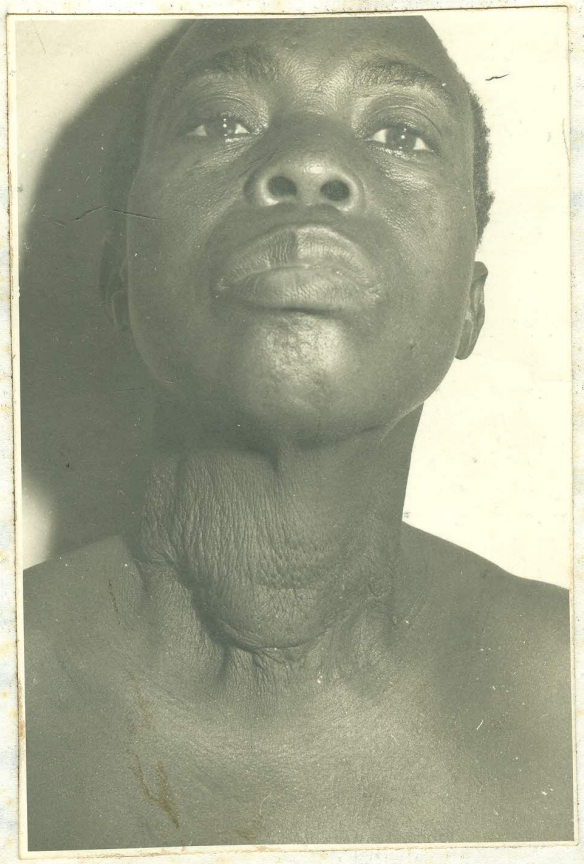


Illustration 9:

Shows the same patient in in the illustration 8, 4 weeks after subtotal thyroidectomy.

is manifested early by paresthesia, numbness, and tingling of the hand and carpopedal spasm. A positive chvostek's or Trousseau's sign may be elicited. Low serum calcium confirms the diagnosis. Symptoms may be relieved immediately by the intravenous administration of calcium. Patients with permanent parathyroidism are maintained on large doses of vitamin D and calcium lactate.

Infection

This may occur as brawny edema of the skin flap, which requires little treatment and more often follows operation for thyroiditis.

Hypothyroidism

This may follow if too much gland has been removed. It occurs in about 9% of cases.[27] It is usually controlled with 2 to 3 grains of thyroid extract a day or its equivalent.

None of these postoperative complications was encountered in this study.

Other methods of treatment

X-ray and radium therapy have been applied to endemic goitre but no signficant results have been obtained and this approach does not fit in with the conception that goitre is a compensation for the shortage of thyroid hormone.[20]

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COLO-RECTAL CARCINOMA

SUMMARY

Ten patients with carcinoma of the colon and rectum are presented. One patient is presented in detail. Incidence, etiological factors, pathology, clinical manifestation and management of carcinoma of the colon and rectum are discussed and a review of literature presented.

INTRODUCTION

Colo-rectal carcinoma is a world wide disease forming about 15% of all malignant neoplasms.[1] The incidences vary greatly in different countries but usually countries with high incidence of cancer of the rectum have also a relatively high incidence of cancer of the Colon.[2] In the United States of America, Wynder and Shigematsu[3] found higher incidence in white population than in the non-white population this perhaps is due to relatively higher socioeconomic level of the whites.

About 70% of cancer of the large bowel occurs in the rectosigmoid. The rest is distributed almost equally through out the remaining colon with slightly higher incidence in the caecum.[4] The disease occurs at all ages but most frequently between 50 and 80 years.[4] Men are attacked more frequently than women (3:2) although cancer of the ascending colon is encountered more often in women.[5]

In this study age incidence ranged from 38 years to 64 years. There were 5 males (50%) and 5 females (50%) (tables 1 and 2).

Cancer of the right colon was found in 4 patients (3 in the caecum, 1 in the ascending colon). Cancer of the transverse colon was found in 1 patient. Cancer of the left colon was found in 3 patients (all in the sigmoid colon). Rectal cancer was seen in 2 patients (table 3).

CASE REPORT

S.W., I.P. No. 263278, 56 year old female Mvidunda by tribe was admitted in Surgical Ward 21 of Muhimbili Medical Centre on 25th October, 1981 with history of intermittent bleeding per rectum for one year. She noted that red fresh blood was passed more often. However, she occasionally noticed dark blood staining on the surface of stools. She also complained of low abdominal pain and backache. The pain was not associated with any aggravating factors and was not radiating to any other site of the body. Analgesics (aspirin) helped to relieve the pain. She had occasional constipation but denied any history of diarrhea or weight loss.

Review of respiratory, cardiovascular and central nervous systems was normal. She never had any major illness in the past. She was a peasant farmer by occupation. She occasionally drank alcohol but did not smoke. There was no other member of her family having similar symptoms.

On examination she was found to be healthy looking and fat. She was not pale, not jaundiced and had no lymphadenopathy. Temperature was 37°C, Blood pressure was 130/80 and pulse was 68 per minute regular.

Mouth and tongue were normal. Abdomen was not distended. On palpation the abdomen was soft nontender and no masses or organomegaly detected. It was normal on percusion and bowel sounds were normal.

On rectal examination a mass was felt in the rectum at about 6 cm from the anal verge. Blood was seen on the glove of the examining finger. Clinical diagnosis of rectal carcinoma was made. Patient was prepared and taken for proctosigmoidoscopy and biopsy of the mass. Histology report was well differentiated adenocarcinoma of the rectum.

The patient was discussed in the tumour Board and Management by abdominoperineal resection with permanent colostomy was advised. The nature of treatment to be done was explained to the patient and close relatives. They agreed with the plan.

Preoperative investigation done included full blood picture which was reported as normocytic normochromic with haemoglobin of 15.7Gm%. Stool microscopy, urinalysis and random blood sugar were normal. Grouping and cross match of 4 units of blood were ordered.

Bowel preparation were started four days before operation and the following were ordered, low residue diet, oral flagyl 400mgs 8 hourly for 7 days, intramuscular daily injection of procaine penicillin for 7 days, intramuscular daily injection of Vitamin K 5mg for 5 days. 2 days before operation patient was restricted to fluid diet, oral neomycin 1gm 8 hourly was ordered and enema was given twice per day. The patient was ordered nil by mouth from midnight on the day before surgery and she was started on intravenous drip of 2 litres 5% dextrose, and 1 litre normal saline. On the day of operation, enema was given in the morning. Patient had nasogastric tube and urethral catheter inserted.

In the operating theatre, the patient was given general anaesthesia and put in lithotomy-Trendelenburg position, cleaned and draped.

The operation was carried out by two teams of surgeons (abdominal and perineal surgeons). The abdominal lower left paramedian incision was made first and after finding the tumour operable, the perineal dissection was also started by the perineal surgeon after closing the anus with an encircling silk stitch.

The abdominal surgeon after extending the incision about 2 inches above the umbilicus, mobilized the sigmoid colon, identified the left ureter and swept it to safety away from the vascular pedicle. The artery was isolated and double ligated with silk and divided close to its origin from the aorta. The inferior mesenteric vein was also ligated and divided. The lymph nodes were checked and found to be clinically uninvolved. The rectosigmoid mesentery was mobilized and lifted up. Blunt dissection downwards and backwards was carried out until the presacral space was entered to meet the perineal dissection posteriorly. The rectal vessels were ligated

and divided. Strands of pelvic fascia were divided with scissors, taking care not to injure the ureters.

After haemostasis was achieved, the abdominal operators prepared the remaining portion of the sigmoid colon and passed out to form a terminal colostomy in the left iliac fossa. The pelvis was irrigated with normal saline and the peritoneum from lateral walls and iliac fossae was mobilized and sutured with continuous chromic catgut. The abdominal wound was closed in layers without drain. The colostomy was completed by suturing the edges of the 2 cm colon projecting above the colostomy wound to the surrounding skin with interrupted 2/0 chromic catgut sutures to form a stoma.

The perineal surgeon partially closed the wound and put in a pack and pressure bandage applied.

During the operation the patient received one and half litres of blood.

Postoperatively the patient was maintained on a drip with 2 litres of 5% dextrose in 24 hours and was started on; intravenous crystalline penicillin 2 mega units 6 hourly for five days, chloramphenical 500 mgs 6 hourly for 5 days and intramuscular pethidine 100 mgs 6 hourly for 24 hours.

Continous bladder drainage was maintained as well as nasogastric tube suction. Observation of blood pressure pulse, temperature and input/output chart were ordered, one hourly for first 24 hours and 6 hourly thereafter.

On second postoperative day colostomy started working and bowel sounds were present. The nasogastric suction was stopped and patient allowed to take oral liquids.

On third postoperative day the perineal pack was removed and dressing of the perineal wound twice daily was ordered. The I.V. drip was stopped and antibiotics were given by intramuscular route.

Stitches were removed from abdominal wound on the 10th day but continued with daily sit baths until the perineal wound was healed. She was trained how to manage her own colostomy and she was

discharged from the ward four weeks after operation.

She regularly attended follow-up. She remained symptom free and had no sign of recurrence up to 5th May, 1982.

The Rest of the presentation is as follows:

Table 1: Age incidence

Age range (Years)	No. of patients	Percentages
30	-	-
30 - 40	3	30%
40 - 50	2	20%
50 - 60	3	30%
60 - 70	2	20%
70	-	-
Total No. of patients	10	100%

The youngest patient was 38 years old. The oldest patient was 64 years old.

Table 2: Sex incidence

Sex	No. of patients	Percentages
Male	5	50%
Female	5	50%

Table 3: Site of the tumour as found at operation

Site of the tumour	No. of patients	Percentages
Right colon	4	40%
Transverse colon	1	10%
Left colon	3	30%
Rectum	2	20%
Total	10	100%

1. Right Colon included caecum, ascending colon and hepatic flexure.
2. Left Colon included splenic flexure, descending colon and sigmoid colon.

Presenting symptoms in relation to the site of the tumour and number of patients involved are shown in Table 4.

Table 4:

Presenting Symptoms	Site of the tumour				Total No. of patients
	Right Colon	Transverse Colon	Left Colon	Rectum	
Abdominal pain	4 patients	1 patient	3 patients	2 patients	10(100%)
Low backache	3 patients			2 patients	5(50%)
Constipation		1 patient	3 patients	2 patients	6(60%)
Bleeding per rectum				1 patient	1(10%)
Passing blood stained stools			2 patients		2(20%)
Diarrhoea			1 patient		1(10%)
Passing mucoid stools				1 patient	1(10%)
Weight loss	1 patient				1(10%)
Vomiting	1 patient				1(10%)

Site and extent of tumour spread (Duke's classification) as seen at first operation are shown in Table 5.

Table 5:

Tumour Site	Extent of lession by Duke's classification			
	A	B	C	D
Right Colon			2	1
Transverse Colon				1
Left Colon		1	1	2
Rectum		2		
Total number of patients	-	3(30%)	3(30%)	4(40%)

Treatment

Treatment offered for carcinoma of the colon (7 patients) is shown in Table 6.

Table 6:

Treatment	No. of patients	Percentages
(a) Surgery:		
Right hemicolectomy	2	25%
Sigmodectomy	1	12.1%
Left hemicolectomy	2	25%
Right transverse colostomy (palliative)	2	25%
Terminal Ileostomy (palliative)	1	12.5%
(b) Adjuvant chemoimmuno-therapy	2	25%
(c) Adjuvant Immunotherapy	1	12.5%

Rectal Carcinoma

The two patients who had rectal carcinoma were treated by abdominoperineal resection with permanent left side colostomy. They both had rectal tumour situated at a level about 6cm from anal verge.

DISCUSSION

Etiology:

The cause of colo-rectal cancer is unknown but there are predisposing and premalignant conditions with which it has been found to be associated.

Diet

Epidemiological studies strongly indicate that the disease is produced by environmental factors probably consumed with food. Burkit[6] feels that the refined, fibre free, carbohydrates eaten in the Western countries increase the intestinal transit time and the bacterial content of the intestine. The resultant bacterial fermentation and putrefaction are more intense and may well be important factors in the causation of colonic cancers. Any carcinogens ingested or formed in the intestines would be not only present in a more concentrated form but also held in contact with the mucosa for a more prolonged timein constipated colon. He also noted that the rapidly changing dietary pattern in Japan especially in the urban areas,

is associated with an increase in the large bowel disease. Walker[7] using plastic pallets has shown a faster transit time in the Bantu population and Globes et al[8] have shown that weight of stool in the high cancer Japanese population in Hawai is significantly less than those in the low risk group. Wynder et al[2] have also shown that incidence of colonic cancer is associated with the total fat content of the diet which influence the fecal bacterial content. Aries[9] has shown that the faeces from healthy English adult subjects on a diet of mixed animal and vegetable food contained significantly more bacteroids than feces from Ugandan adults on a high carbohydrate vegetarian diet. Bacteroids are very active in degrading bile salts and in the laboratory it has been shown possible to convert deoxycholate into a potent carcinogen 20-methylocholanthrene. It is thus postulated that high dietary fat increases concentration of bile acid in the large bowel with subsequent metabolism by the bacteroids to carcinogens.

It is known that bacterial flora in the colon is altered in high risk populations as in colon cancer patients to an elevated ratio of anaerobes to aerobes. The resident bacteria may contribute to the development of colonic cancer by; enzymatically removing a detoxifying group from a carcinogen whose group had been covalently added by the hepatic system, may conduct a limited activation of a procarcinogen introduced into the colon in the diet and may metabolize an endogenous substance to either a carcinogen or more likely to a promoter. On the other hand microbes may remove endogenous procarcinogen or carcinogen for the colonic contents thus sparing the intestinal epithelium from the toxic action. It is well established that β-glucuronidase activity is markedly elevated in the feces of high-risk colon cancer populations.

The inactive glucuronide conjugates formed within the liver would be excreted by the biliary system into the colon and intestinal microbial flora can hydrolyze glucuronides releasing the phenol at the colonic mucosal site. Further metabolism by the flora or by the mixed

function oxidases of the mucosal cells would lead to the production of mutagenic and/or carcinogenic substances. That β-glucuronidase - catalyzed hydrolysis of a benzoapyrene phenol glucuronide can result in formation of a DNA binding benzoapyrene derivative has been shown.[10]

Administration of the colon procarcinogen, 1, 2 dimethylhydrazine elevates the level of intestinal microbial β-glucuronidase which might reflect the increased content of methylazoxymethanol glucuronide formed in the liver and introduced into the bowel through the bile. The microbial enzyme could consequently release methylazoxymethanol from the inactive conjugate and therefore expose the colon to this highly reactive carcinogenic metabolite of diamethylhydrazine.[10]

Two stage mechanism of large bowel carcinogenesis[10]

In this mechanism, the initiator may arrive into the colon by the diet or the bile secondary to hepatic metabolism. The initiator leads to alkylation of the colonic DNA, a process composing at least a segment of the neoplastic transformation. The initiated cell, however, would require the additional stimulus of a promoter in order to yield sufficient progeny to compromize a distinct and sizeable neoplasm.

Bile acids, formed in large amounts following high-fat diet could save as promotors or promotors could be introduced directly by the diet. The mechanism of action of promotors could lead to intracellular production of cyclic nucleotides which constitutes part of promotion or the interaction of the promoter with colonic cell membrane might lead to the activation of prostaglandin synthetase particularly PGF_2 which subsequently activates cyclic nucleotide synthesis and the stimulation of division of the initiated cell.

Drugs

Second proposal for the presentation of activated carcinogens to the colon involves the participation of the hepatic drug metabolism systems and the fecal bacteria. Activation and conjugation of the activated metabolite with glucuronic acid occurs primarily in the liver and then deconjugation of the activated metabolites by fecal

bacterial enzymes such as B-glucuronidase. In this way activated carcinogens might reach the colonic mucosa. Alternatively, drugs and carcinogens could also be activated in the colon directly by a colonic drug metabolism system analogous to that found in the liver.

Premalignant conditions

Conditions of the large bowel that have been found to be associated with malignancy include; adenomatous polyps, familial polyposis and ulcerative colitis.[1]

In about 30% of cases of colorectal carcinoma, operation specimens show that in some part of the bowel that has been removed, in addition to the carcinoma, there are one or more adenomas or papillomas.[5] It is known that the incidence of colonic cancer is high in relatives of patients with colonic cancer, those with multiple polyposis and patients with ulcerative colitis.[12]

In this study one female patient was known case of ulcerative colitis.

Pathology

Macroscopically:

There are four types of colorectal cancers; annular, tubular, ulcerative and cauliflower types.[5] The annular type is a hard small, encircling tumour with little proliferation and much fibrosis that tends to obstruct the lumen; the ulcerating type has a raised edge a necrotic center and often bleeds and the cauliflower or polypoid type is usually found in the right colon or the rectum it may ulcerate and bleed.

Microscopic types

Although other malignant tumours such as leiomyosarcomas, fibrosarcomas, lymphomas and carcinoid tumours can be found in the colon, approximately 98% of colo-rectal cancers are columnar cell adenocarcinomas in their varient forms.[1]

Medullary adenocarcinoma is the commonest form and is usually a sessile mass projecting into the lumen of the bowel with its edges

indurated and nodular. In the cecum it assumes fungoid appearance and oozes blood freely. Scirrhous adenocarcinoma has much more fibrous than epithelial or glandular tissue, feels hard and often causes intestinal obstruction. It often occurs in the left colon. The papillary or villous adenocarcinoma arises from papillary or villous adenomas. The villous projections from the surface give it a warty appearance and usually ulcerates. It is mostly found in the left colon. Mucoid adenocarcinoma richly contains mucinous material and forms about 5% of adenocarcinoma of the colon and rectum. They occur mostly in the rectum, rectosigmoid, cecum and ascending colon.[1]

Histologically the tumour can be a well differentiated, a poorly differentiated or an anaplastic adenocarcinoma. The Broder's system grades tumours into 4 categories based on the degree of differentiation. Grade I, there are 75 to 100% differentiated cells; in Grade II 50 to 75% differentiated cells; and grade III 25 to 50%. The more and grade IV 25% or less. The more the cells are undifferentiated, the more malignant is the tumour. This Grade I and II tumours have a relatively good prognosis whereas Grade III and IV tumour have increasingly poorer prognosis.

Two important factors that have an important bearing on operative treatment are the extent of local spread and the lymphatic drainage.[1]

Duke's classification: This is based on the extent of spread and correlates well to the prognosis and is as follows:

Table 7:

Class	Extent of the lesion
A	Lesion confined to the mucosa
B_1	Lesion limited to the muscularis propria
B_2	Lesion penetrating the muscularis propria
C_1	Lesion limited to the bowel wall
C_2	Lesion penetrating all layers of the wall. Both C_1 and C_2 have positive lymph nodes
D	Distant spread

Adenocarcinomas of the colon spread mainly by direct invasion, lymphatics or peritoneal implantation.

Direct invasion around the circumference and within the bowel may occur. Spread within the wall of the rectum or rectosigmoid is rather limited and division of the bowel 2.5 cm below the tumour will remove the intramural portion of a well differentiated tumour of a grade I or II of Broder's classification; while a resection of 6 cm or more is needed for anaplastic tumours.[1]

By regional lymphatics, metastasis usually occurs first to the epicolic nodes on the wall of the colon. From there it progresses to the paracolic nodes along the marginal artery, to the mesocolon nodes along the colon and sigmoid arteries and the principal nodes at the origin of the superior and inferior mesenteric arteries.[13] The ultimate lymphatic drainage is to the ileolumbar chain and to the thoracic duct.[13] In general the flow of lymph is from the epicolic to the principal nodes, but some nodes may be by-passed and some nodes receive lymphatic drainage from more distant segments of the bowel. The nodes and their connecting lymphatics lie chiefly along the arteries.

The lymphatics of the rectum and anal canal above the pectinate (mucocutaneous) line drain along the vessels within the pelvis and those below pectinate line drain to the superficial inguinal nodes. Upward drainage along the superior haemorrhoidal vessels is the easier pathway and both downward and lateral metastasis of rectal carcinoma occurs only as a result of blockage of the channels, leading upwards. Retrograde lymphatic spread of carcinoma from rectosigmoid area occurs only in one percent of cases.[14]

The lymphatics are the most important route by which adenocarcinoma of the colon metastasizes and about 60% of patients have metastases to lymph nodes when operated upon.[13]

In this study 7 patients (70%) had advanced disease with lymph node involvement at the time of first operation "table 5". Only 3 of these 7 patients were resectable.

Clinical Manifestations

The symptoms and signs of cancer of the colon and rectum mainly

bowel at the site of the tumour. The main function of the right or ascending colon is absorption and of the left colon is storage. Cancers arising from the right colon or the absorbing portion tend to be larger and fungating because the intestinal content is more liquid, the bowel wall thinner and the diameter much bigger than in the left colon where cancers tend to be scirrhous, annular and characterized frequently by obstruction during the course of the growth. In general, symptoms in patients with cancer of the left colon occur earlier than those of the right.[15]

Abdominal pain

Abdominal pain or discomfort is the main symptom of cancer of the colon and rectum.[16] It is present as the first symptom in 76% of patients with cancer of the right colon, in 41% of the left colon and in 24% of the rectum.[16] Colicky or cramp-like pain is the most common variety associated with lesions in the right and left colon although distension, intermittent dull or constant ache is also encountered. Pain usually radiates to the back in cases of sigmoidal neoplasms and to the lower part of the abdomen and perineum in cases of rectal cancer. Cancers elsewhere in the colon, the radiation of pain occurs rarely. Pain simulating appendicitis may occur in patients with right colon cancer. The cancer may obstruct the appendiceal lumen or interfere with the vascular supply of the appendix. Sometimes cancer obstructs the ascending colon and the appendix becomes inflamed because of retrograde pressure which prevents the emptying of the organ. Appendisectomy is sometimes done for lower right quadrant pain only to find the correct diagnosis is caecal cancer[16] when cancer obstructs the left colon, peritonitis may result due to perforation of a proximal ruptured diverticulum above the tumour.

In this study two patients had previous appendisectomy done. One had appendisectomy done 3 weeks before the diagnosis of carcinoma of the colon involving the transverse colon was made. The other had appendisectomy 6 months before diagnosis of advanced carcinoma of ascending colon was made.

Tenesmus

This is a painful desire for frequent but ineffectual bowel monements. It is commonly a late sign and suggests incomplete obstruction of the rectum.

Blood in stools

Gross bleeding occurs in 80% of cancers of the rectum, in 49% of those of the left colon and 30% of those of the right colon.[16] Frequently blood in stool is attributed to an anal lesion particularly hemorrhoids and about 15% of patients with cancer of the colon and rectum have had a prior haemorrhoidectomy done.[16]

Obstruction of the superior haemorrhoidal veins by the rectal tumour may lead to formation of haemorrhoids which the patient presents with or the patient may mistake the symptoms of the rectal cancer for those of the haemorrhoids.[5]

Occult blood occurs much more frequently. In cancer of the right colon, the occult or dark blood is mixed in the stool, while in cancer of the left colon or rectum, the blood is red and noted on the outside of the stool on the paper or in the bowl.

In this study (see table 4) one of the two patients of carcinoma rectum complained of frank bleeding per rectum while two other patients with carcinoma of left colon complained of passing blood stained stools.

Change in bowel habits

This occurs commonly with cancer of the left colon and rectum. Changes may be from constipation to diarrhoea, diarrhea to constipation or from normal habit to alternating diarrhea and constipation.

In this study, constipation was a presenting symptom in 6 patients (60%) 3 of these 6 patients had carcinoma of left colon, 2 patients had carcinoma of rectum and 1 had carcinoma of transverse colon (table 4).

Intestinal obstruction

This is often a late sign of advanced cancer. Marked abdominal distension, pain, tenderness, nausea and vomiting are the predominant manifestations of obstruction.

In this study two patients had complete intestinal obstruction at the time of admission. One of the two patients had carcinoma of the caecum and presented with history of abdominal pain and vomiting (table 4) and the other had carcinoma of the sigmoid colon and complained of complete constipation with abdominal distension.

Weight loss

This is frequently found when neoplasm is in the right colon, probably because the lesion is more advanced when the diagnosis is made.[12]

This was a presenting symptom in one of the patients in this study who had carcinoma of the right colon (table 4).

Anaemia

From 4 to 20% of patients have anaemia on admission and usually occurs with cancer of the right colon and caecum.[15] It is caused by blood loss and depression of hematopoietic system by the toxin from the tumour.

In this study pallor was noted at admission in two patients. One with carcinoma of transverse colon the other had carcinoma involving the caecum.

On examination a mass may be palpated on the abdomen particularly when the tumour arises from the right colon when patients usually present with a far advanced lesion.

Concomitant diseases like; haemorrhoids inguinal hernia may also be found and digital rectal examination will detect most of rectal carcinomas.

In this study the two patients who had rectal carcinomas were detected on digital rectal examinations. In both cases the tumours were about 6 cm from anal verge.

Diagnostic investigations

These may include, occult blood test in stools, proctosigmoidoscopy, barium enema, exfoliative cytology, search for cancer cells in blood and radioimmunoassay of circulating carcino-embryonic antigen.

Occult blood test

Patient should be put on a meat-free, high residue diet for 3 days before the test is done. The test can detect as little as 0.5mg of haemoglobin per ml. of aqueous solution. 3 to 6 specimens of stool should be tested.[17]

Proctosigmoidoscopy

By this procedure, it is possible to detect the 70% of cancers located in the last 25 cm of the large bowel.[12] Palmer[18] stresses that the prostosigmoidoscopy can be done without prior enema preparation of the bowel and that enemas irritate the mucosa, causing hyperemia and edema, surface exudate is washed off making proper interpretation difficult.

Rigid sigmoidoscope is quite safe, easy to insert and gives a more clear image. However, the flexible fiberoptic sigmoidoscope makes visualization of colonic mucosa for 50 cm or more from anal orifice possible and patient accepts it much better.[19]

Proctosigmoidoscopy is a safe procedure. However, occasionally perforation of the colon, bacteremia and haemorrhage may be encountered.[18]

Rigid proctosigmoidoscopy is a common procedure done in surgical firm III at Muhimbili Medical Centre particularly on patients with haemorrhoids or suspected of having colo-rectal cancer. The author has not encountered or withessed any of the above complications so far.

Barium enema

This is usually done in patients who present with symptoms suspicious of malignancy or when sigmoidoscopy discloses the lesion and one wishes to be sure that there is no secondary tumour above it. A prepared colon free of residual feces and mucus will give best results. The patient should be given 30 cc of castor oil before noon and a tap water enema in the evening of the day before the barium enema is scheduled. On arrival at the Radiology Department, the patient is given subcutaneously 0.25% to 1mg of atropine and an enema containing both water and bisacodyl tannex.[20]

Exfoliative cytology

Preparation of the bowel is done as for a barium enema. In this procedure, a sigmoidoscope is inserted to the 15 cm mark and through the scope, a No.28 Ewall tube is inserted. The scope is withdrawn and 800 cc of 0.9 percent of normal saline injected. The abdomen is massaged and the material is siphoned out through the Ewald tube and prepared for Papanicolaou stain.

Using this procedure Raskin and Pleticka,[21] reported an accurancy of 85% in 569 examinations.

Search for cancer cells in blood

In this procedure bovine fibrinogen has been used to sediment rapidly most of the erythrocytes. Then bovine albumin solution is added and the cancer cells are collected in the interface between the plasma and albumin solution and examined with Papanicolaou stain. Using this procedure cancers have been found in the blood of patients with colonic and rectal cancer.[22] However, the procedure is still in experimental stage.

Radioimmunoassay

Successful use of radioimmunoassay in detecting circulating carcino-embryonic antigen of adenocarcinoma of the colon has been reported.[23] In this procedure, the patients blood is mixed with goat's milk containing antibodies and radioactively tagged carcino-embryonic antigen in a test tube. The antibodies were produced after the goat was injected with colon cancer cells. If, after centrifuge, the Geiger counter shows that all of the radioactive carcino-embryonic antigen originally put into the tube are now at the bottom, it means that there were no antigens in the patients blood to complete for combination with antibodies. On the other hand, if the counter finds only a portion of the radioactive carcino-embryonic antigen at the bottom, then the nonradioactive carcino-embryonic antigen must have come from the patient's blood. 35 of 36 patients with adenocarcinoma of colon and rectum had detectable levels of circulating carcinoembryonic antigen.[28] The test became

negative after tumour resection and only remained positive if there were persistent cancer tissue left behind.[23]

Non of the last three investigations was done in this study.

Treatment

Surgery:

Primary surgical resection of the tumour and its lymphatics offers the only hope of cure of malignant neoplasms of the colon and rectum with a five year survival rate of over 50% of patients so treated.[24] The Turnbull's no touch isolation technique of dissection is the ideal method.[25]

Resection for cure is done in the absence of demonstrable metastases to the liver or lung, but with or without malignant invasion of the immediate regional nodes within surgical reach. Adjacently involved movable organs such as a loop of small intestine, the appendix, the uterus, the adnexae may be included in the resection for cure. Resection for palliation on the other hand is done with definate evidence of inextirpable cancer which is left behind at the time of the operation. Colonic cancer should be removed even in the presence of metastases because they grow, become infected ulcerate, bleed or perforate. In case of obstruction, proximal colostomy, usually with plans for later resection or local radiotherapy, is done.

Preoperative preparation

In order to prepare the patient adequately for surgery, he has to be hospitalized for a minimum of 5 to 7 days.

1. Nutritional preparation

This is often required and oral feeding with a pure amino acid diet combined with simple sugars, vitamins, electrolytes and essential fats; available as a water soluble powder (Vivonex) is advised.[24] It is rapidly absorbed in the small intestine, gives a low residue and seems to be able to sterilize the large bowel of most of its bacteria.[24] Intravenous alimentation can also be done.

Some patients particularly those with caecal carcinoma present with anaemia. Those with haemoglobin below 10 grams should have preoperative blood transfusion and blood should be avilable during surgery.

2. Mechanical bowel preparation

Clean and sterile bowel is of utmost value to ensure a normal performance and healing of the anastomosis. Upon admission to the hospital, the patient should be given a low residue regime e.g. with VIVONEX. Castor oil, 45 to 60ml is given 48 hours prior to surgery and on the day before surgery. The patient is kept on clear oral liquids e.g. glucose water with citric acid, on the final preoperative day. Cleansing saline enemas are given in the evening before surgery and on the morning of surgery.

3. Chemotherapeutic bowel preparation

It is generally accepted that the bowel cannot be completely sterilized but the bacterial flora can be markedly suppressed by antibiotics. Various antibiotics have been used Cohn[26] advises use of Kanamycin. He repoted on 560 patients who were given Kanamycin orally 72 hours before surgery in 4 hourly doses of 1 gram each followed by doses of 1 gram every 6 hours until surgery, he reported only 3 patients with side effects, 12% with septic wounds and 1.6 percent with peritonitis.

Patients with large bowel obstruction present special problems. The bowel cannot be cleansed mechanically and oral antibiotics are not effective maximally. In such patients, abdominal distention should be decompressed by a long tube. Fluids, electrolytes, vitamins and blood are given. The patient is explored and in absence of strangulation, left-sided large bowel obstruction is decompressed with a right side transverse colostomy or occationally a cecostomy.

In this study both mechanical and chemotherapeutic bowel preparations were done preoperatively, low residue diet was ordered 4 days prior to surgery, purgative (Magnesium sulfate) was usually

ordered 2 days prior to surgery, enemas were ordered a day before surgery and in the morning on the day of operation. Patient was put on either oral flagyl or neomycin or both in the course of preparation. Flagyl was usually started 4 days prior to surgery while Neomycin was started 48 hours before surgery. Gentamycin 80mgs intravenous was started early in the morning on the day of operation and was maintained for about 4 days postoperatively.

One day prior to surgery patient was restricted to oral liquids only and from midnight of the day prior to surgery patient was not allowed to take anything by mouth and was put on intravenous drip.

This preparation worked well with only one patient (10%) developing sepsis postoperatively.

Carcinoma of the colon

All lesions of the colon should be resected preferably by the "No-touch isolation technique" as described by Turnbull.[25] This technique is designed to minimize haematogenous dissemination of cancers by the trauma which always follows surgical removal. The lesion itself is not touched until all the blood supply in the messentery has been divided and the anastomosis is completed. Turnbull and his associates[25] have reported a five years survival of 69% of patients resected for cure by this technique, compared with a 52% survival in a comparable group of patients resected by conventional techniques at the same clinic.

The amount of bowel which should be removed varies with the situation of the growth and with the extent of lymphatic spread. Extent of resections for carcinoma of the colon is shown in illustration 10.

1. Right-sided hemicolectomy

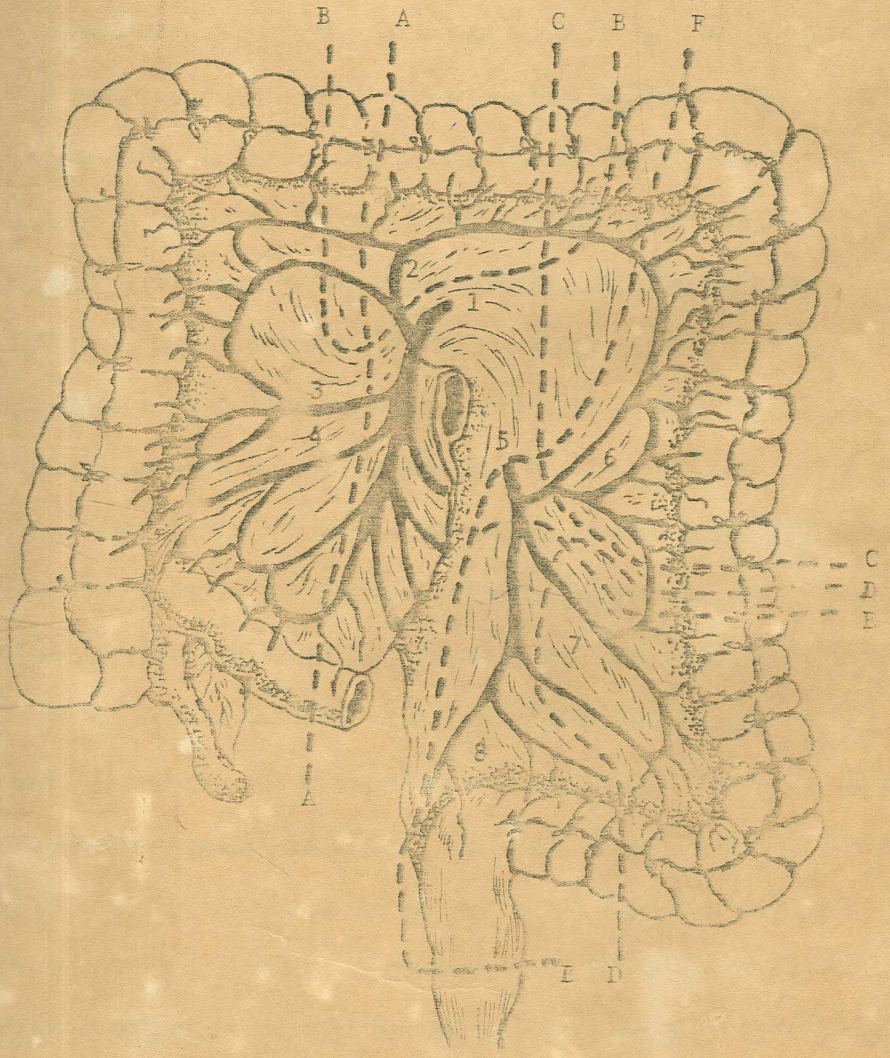
This operation is carried out in the treatment of carcinoma in any part of the right side of the colon.[27] It implies removal of the terminal 20 cm of ileum, the caecum, the ascending colon and the proximal one-third of the transverse colon thus AA in illustration 10.

Illustration 10:

Drawing to show the extent of resections for carcinoma of the colon.

AA - Right hemi-colectomy. BB - Resection for growth in middle of transverse colon. CC AND DD - Limited resections in left side of colon. EE - A more radical resection with removal of upper rectum. FE - A still more radical resection (left hemi-colectomy).

Note relative position of arteries: 1 - superior mesenteric; 2 - middle colic; 3 - right colic; 4 - ileo-colic; 5 - inferior mesenteric; 6 - upper left colic; 7 - lower left colic; 8 - superior rectal.



During mobilization of the bowel, small bowel is packed off to the left side of the abdomen and the greater omentum is detached from the proximal one-third of the transverse colon by dividing it between ligatures. The caecum and ascending colon are drawn towards the midline, and the peritonium of the posterior abdominal wall is incised where it is reflected from the lateral side of the bowel, the incision is carried downwards below the caecum, and upwards around the hepatic flexure. The bowel is elevated from its bed and swept medially until it hangs loosely by its attached mesenteric leaf containing the blood vessels and lymphatic structures. Care is taken to avoid injury to the duodenum, the ureter and the genital vessels, which lie on the posterior abdominal wall. The ileo-colic and right colic vessels are identified, and are divided between ligatures close to their origin from the superior mesenteric. The mesentery is divided in a line extending to the ileum 20 cm from its lower end and to the junction of the proximal and middle thirds of the transverse colon. At the levels of mesenteric section the bowel is divided between clamps and the separated gut together with its mesentery is removed. If side to side anastomosis is to be performed, both ends are closed and invaginated. The ileum is brought up along side the transverse colon and a lateral anastomosis (iso-peristaltic) is carried out close to the blind ends. If end to side anastomosis is preferred the colon alone is closed and the ileum is implanted into the side of the blind end. The third alternative of end to end anastomosis may also be considered.

Resection and anastomosis carried out as a one-stage operation in the procedure of choice. However, when a considerable degree of obstruction is present, side to side ileo-transverse anastomosis may be performed first, to be followed by the excision 2 to 3 weeks later. The ileum and transverse colon are divided alone to the anastomosis the cut ends are closed and infolded.[24]

In this study right hemicolectomy was done in two patients (both had carcinoma of the caecum (table 6).

Transverse colon resection

When the gorwth is situated in the middle third of the transverse colon, resection is planned so that 7 to 8 cm of healthy bowel is included on each side of the tumour (BB in illustration 10). The greater omentum is detached from the segment of colon to be removed, and the mesocolon is divided between clamps in a V-shaped manner; the limbs of the V extend to the bowel at the levels selected for resection, and the apex will usually include the main trunk of the middle colic artery which is divided between ligatures. The hepatic and splenic flexures are usually mobilized to allow end to end anastomosis without tension.[27] A safe anastomosis depends essentially on the integrity of its blood supply, and this is seriously endangered by continuous sutures or by too many interrupted sutures. Catgut should not be used, because in the colonic wall it may be absorbed before the anastomosis is firm. Carefully spaced interrupted sutures of nonabsorbable material; silk, linen or stainless steel wire; should be used. Two layers of suture are normally employed. The knots of the first layer transfixing the whole thickness of the gut wall, should be tied on the mucous surface; the second layer consists of sero-muscular (lembert) sutures. After the junction has been completed the appendices epiploicae should be utilised to reinforce the suture line. A drain is placed down to the region of the anastomosis, so that any leakage which may occur will have free egress to the exterior, and will not contaminate the peritoneal cavity.

Resections for growths in the left colon

1. Conservative resection

In case of early growths of the left side of the colon, with no sign of spread to glands alongside the main trunk of the inferior mesenteric artery, conservative resection may be adequate. For early

tumours of the splenic flexure and descending colon, a segment of bowel between the left part of the transverse colon and the middle or lower part of the pelvic colon will normally be removed, and the whole of the upper left colic branch of the inferior mesenteric artery will be included in the resection. (CC in illustration 10). In the case of pelvic colon growths the resection may comprise only a relatively short segment 7 to 8 cm of healthy bowel, on each side of the tumour with inclusion of two or three sigmoid arteries.[27] (DD in illustration 10).

2. Radical resection of the pelvic colon

In this resection, the inferior mesenteric artery is divided either below the origin of its upper left colic branch or at its origin from the aorta and the gut is resected down to the upper one-third of the rectum. End-to-end anastomosis is then effected between the distal part of the descending colon, which is usually well vascularised by the marginal anastomosis and the remaining rectum which derives adequate blood supply from the middle and inferior rectal arteries.

3. Left-sided hemicolectomy

This resection may be done for tumour in any part of the left colon. However, it is specially indicated in the cases of tumours of the splenic flexure or descending colon. It implies the removal of gut from the left part of the transverse colon to the upper one-third of the rectum. The inferior mesenteric artery is divided flush with the aorta, so that all the glands related to it may be removed (FE in illustration 10). The transverse colon is then anastomoised to the remaining rectum.

In this study left hemicolectomy was done in two patients. One had a tumour in the descending colon the other had a tumour in the upper part of sigmoid colon.

Carcinoma of the rectum

The choice of the operative procedure mainly depends on the site of the tumour. However, other factors to be considered are;

patient's general condition tumour size and fixity, and surgeon's experience in rectal surgery.

Operations commonly used for carcinoma of the rectum and rectosigmoid junction include the following.[28]

1. Excision of rectum with preservation of the anal sphincters
   (a) Anterior resection
   (b) Pull through procedures
   (c) Abdominosacral resection
2. Radical excision of the rectum with permanent colostomy
   (a) Abdominal resection (Hartman's procedure)
   (b) Abdomino-perineal resection (Miles procedure)
3. For inoperable cases
   (a) Colostomy
   (b) Electrocoagulation of cancers of the rectum (Madden's procedure)

Anterior resection

This is an operation of choice for carcinoma of rectum situated 12 cm or above from anal margin. The rectum and sigmoid are mobilized. A portion of rectum, rectosigmoid, sigmoid colon are removed together with superior haemorrhoidal vessels and lymphatics up to the origin of the inferior resenteric artery. The inferior mesenteric vessels are divided and the lateral ligaments partially divided. A point on the descending colon and preferably a point 6 cm below the tumour (particularly for anaplastic tumour) or in extreme cases at least 4 cm below the lower gross border of the lesion are chosen for the end to end anastomosis in two rows of interrupted fine silk sutures. The no-touch isolation technique is used. A great advantage of this procedure is that fecal continence is resumed.[24]

Using this procedure, Dixon[29] reported on 340 anterior resection with a 5.9% mortality, a 74% five year cure in patients without nodal involvement and 58.5% when the nodes were involved.

Pull through procedures

This procedure may be used for lesions between 5 and 10 cm above the dentate line.[30] The operation is carried out with the patient in

a lithotomy - trendenburg position. The bowel after mobilization is removed at least 5 cm below the growth. The anal sphincter is stretched and the ano-rectal stump everted and drawn outside. The proximal colon (about 7.5 cm) is pulled down through the everted ano-rectal stump and anastomosis performed, the redundant proximal colon is excised and mucosal stitches applied. The anastomosis retracts spentaneously into the pelvic cavity in 10 to 14 days.[30]

Abdominosacral resection

In this operation the anastomosis is performed through a posterior approach after resecting the coccyx and the distal portion of the sacrum. About 30% of patients develop partial dehiscence and posterior fistula formation. The operation should be strictly limited to small middle rectal, low grade carcinomas.[24]

Abdominal resection (Hartmann's proceedure)

This operation aims at removal of the carcinoma, its lymphatic drainage and at least 5 cm of healthy bowel below and the major portion of the sigmoid colon. A segment of the anal canal, the rectum, the sigmoid loop and a portion of descending colon are removed en bloc and a colostomy is made lateral to the abdominal incision.[27] The rectal stump may be oversewn and the pelvic floor reconstructed or the operation may be extended to remove the rectum down to the anal canal. The operation is easier to perform and less shocking than abdominoperineal resection.[24] Perineal wound is avoided.

Miles abdominoperineal operation

Miles of England introduced this operation in 1907.[24] It is the most radical of all operations for rectal cancer. It removes the rectum, the sigmoid colon together with blood vessels and lymphatics; the pelvic musculature, the external sphincter, the ischiorectal fat and the perianal skin. A retroperitoneal sigmoid colostomy through the left rectus sheath and muscle in a position favourable for the application of a colostomy appliance completes the procedure.[25]

However, when necessary a loop of small bowel adherent to the tumour, the posterior wall of the vagina, a portion of the bladder, uterus or prostate may be resected concomitantly.

The operation can be carried out either by one or two teams (as in Lloyd - Davies procedure).[28] In general, mortality rate varies from 5 to 10 percent. The five-year survival rates without lymph node involvement are about 85% and 35% for those with nodal involvement.[24]

In this study two patients had abdominoperineal operation done. Two teams of surgeons were involved in these operations. In both cases the rectal tumour was about 6 cm from the anal verge. Postoperatively one patient died. The other patient recovered well and remained recurrence free upto 5th May 1982.

When carcinoma of the colon or rectum is non-resectable, bypass procedure like lateral colocolostomy may be done. An iliac colostomy may be performed for non-resectable obstructive tumours of rectosigmoid.[24] Madden's procedure of electrocoagulation of cancers of the rectum may be used in inoperable adenocarcinomas of the rectum.[24]

In this study two cases with Non-Resectable carcinoma of the colon (Left Transverse and Left Colon) had right transverse colostomy done. One patient who had non-resectable carcinoma of the ascending colon had terminal Ileostomy done (see table 6).

Radiotherapy

Preoperative administration of 2000 to 2500 rads has been associated with an improved overall five year survival; 34.6% compared with 28.3% for patients treated with surgery alone.[31]

In the study of Withers[32] the postoperative administration of approximately 5000 rads reduced the local recurrence rate from 21% for a surgically treated control group of 168 patients to 4.4% for 45 patients receiving irradiation.

Radiotherapy has also been found beneficial for palliating painful localized perineal recurrences and solitary skeletal metastasis.[32]

None of the patients in this study received pre or postoperative radiotherapy.

Chemoimmunotherapy

5 Fluorouracil produces significant tumour remission in only 10 to 20% of patients with advanced colo-rectal carcinomas. However, the remission is usually brief (less than five months).[32]

The addition of immunotherapy with Bacillus Calmette-Guerin (BCG) scarifications to 5FU was found to produce slightly higher response rate (35% Versus 21%) and longer survival (Median, 140 versus 73 days) in a group of 23 patients as compared with 24 patient receiving chemotherapy alone.[32]

In this study two patients who had inoperable metastatic carcinoma of the colon were put on two weekly 5FU 500mgs in 250mls of 5% dextrose slow infusion daily for 4 days and weekly BCG vaccination. Beneficial effect was noted (for 6 weeks) in one patient who managed to receive 6 courses of the drug. One other patient received postoperative immunotherapy (BCG) alone.

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SECTION II

UROLOGY

UPPER URINARY TRACT CALCULI

SUMMARY

Seven patients with renal and/or ureteric calculi are presented. One of the patients is presented in detail. Detailed investigations including analysis of stones of five patients who attended regular postoperative follow-up have also been presented. Incidence, etiological factors, composition specific risk factors, clinical features and management of various upper urinary tract calculi are discussed.

INTRODUCTION

Incidence of renal lithiasis varies in different geographical areas, ethnic groups within the same area and may alter at different times within the same area. In Western countries the incidence is about 2 percent.[1] Social groups and occupation play a part, as shown in the Royal Navy and high incidence in the medical and dental professions.[2] Rise in the standard of living and changes of diet structure and other factors of modern living may be important.[1]

50 percent of patients with renal calculus present between. the ages of 30 and 50 years with a male to female ratio of 4:3.[2]

In this study seven patients were managed by surgical firm III in Muhimbili Medical Centre during the period from first August 1980 to 8th May 1982. The youngest patient was 20 years old the oldest was 75 years old. Five were males two females, as shown in table 1.

CASE REPORT

A.A., I.P. No.282041, 31 years old male electrician of Afro-Arab racial origin, was admitted in ward 16 the Muhimbili Medical Centre on 22nd February, 1982 with history of recurrent right hypochondrial pain of 5 years duration. He was experiencing about four attacks of pain in a month. The pain was colicky in nature and radiating to the right testicle and right thigh. It was not associated with haematuria nor vomiting. However, during childhood he had developed haematuria and diagnosis of renal stones was made. He denied any history of prolonged drug ingestion. His causin is having similar

problem of renal stones, is being managed by the same surgical firm and she is included in this study.

On examination the patient was found to be in pain, pulse was 82 per min. regular, blood pressure 120/80, temperature 37°C. He was not jaundiced and the conjunctiva was pink.

There was right renal tenderness without a palpable mass. All other systems were essentially normal.

Plain abdominal X-ray showed right renal stone. Intravenous urogram was later done and confirmed the presence of a stone in the right renal pelvis with moderate right hydronephrosis. The obstruction was incomplete as the distal ureter was visualized. The left kidney, ureter and bladder were normal. Blood urea was 18mg/dl, white cell count was 11.5 thousand with neutrophilic leucocytosis. He had haemoglobin of 12.1g% percent and red blood cells were reported as normocytic, normochromic. Urine culture showed no significant bacterial growth. Urinalysis reviewed no sugar nor albumin in urine. The pH was reported as acidic. Urine deposit microscopy showed leucocytes of more than 100/HPF and few epithelial cells but red blood cells, casts, ova were not seen. Patient was grouped (B+) and X-matched 3 units of blood ready for pyelolithotomy.

In theatre, after endotracheal anaesthesia was administered, the patient was placed and secured in the left lateral position cleaned and draped. Classical loin incision was made, upper ureter identified and a tape passed around it. Further dissection was done to mobilize the lower pole of the kidney. Adhesions were encountered and released around the pelvis in which the stone was palpated. While steadying the mobilized pelvis with ureteric sling, a linear incision in the line of the ureter was made in between stay sutures over the palpable stone. The stone was mobile and difficult to remove because it was able to retract up into dilated calyx. However, the stone was removed with stone forceps. Irrigation with normal saline

was done to ensure complete removal of all calculi. Haemostasis was achieved and ureteric catheter was freely passed down the ureter before pyelotomy was closed with 4/0 chromic catgut. Corrugated Portex drain was placed through a separate stab incision down to the renal pelvis and abdomen was closed in one layer with 0/0 dexon. Skin was closed with 3/0 Nylon simple interrupted sutures.

Postoperatively 1 hourly blood pressure, pulse and input-output chart were oldered as well as 4 hourly temperature observation. The patient was put on pethidine 100mgs 6 hourly for 24 hours and ampicillin 500mgs 1/m 6 hourly for four days.

Recovery was smooth stitches were removed on the 8th day and the patient was discharged on the 10th day. On chemical analysis the stone was found to contain calcium oxalate and phosphate. Postoperative radiological investigations including intravenous pyelogram showed no residual or recurrent stones in the right kidney and the left kidney ureters and bladder remained essentially normal. The patient was one of the five regular follow-up attendants and the author was able to carry out postoperative radiological, haematological, biochemical and dietary investigations as shown in table 5.

Table 1: Rest of the presentation of the seven patients was as follows:

Patient's Identity	B.G. 243434	A.Z. 259937	O.A. 282932	K.R. 198397	H.R. 272526	A.K. 21849	A.A. 282041
Age Sex	37 years Female	32 years Male	20 years Male	38 years Male	30 years Male ?	75 years Male	31 years Male
Residence	Kariakoo D'Salaam	Kariakoo D'Salaam	Mtoni D'Salaam	Kibaha Coast	Dodoma	Tabora	D'Salaam
Occupation	Tea lady, Tans. Tea Blenders	Businessman	Businessman	Road worker driver	Housewife	Peasant farmer	Electrician
Religion	Islam	Islam	Islam	Islam	Islam	?	Islam
Racial Origin	Afro-Arab	Afro-Arab	Afro-Arab	Af. [illegible]	Af. [illegible]	Af. [illegible]	Afro-Arab
Nationality	Tanzanian	Tanzanian	Tanzanian	Tanzanian	Tanzanian	Tanzanian	Tanzanian
Duration of renal stone symptoms	2 years	4 years	14 years	10 years	3½ years	1 year	5 years

151

Table 2: Presenting symptoms at the initial admission

Presenting Symptom	No. of patients	Percentages
Abdominal pain	7	100%
Recurrent fevers	2	28.5%
Haematouria	2	28.5%
Dysuria	2	28.5%
Increased frequency	1	14.2%
Nocturia	1	14.2%
Pyruria	1	14.2%

Table 3: Associated previous diagnosis and treatment

Previous dignosis and treatment	No. of patients	Percentages
Appendicitis	2	28.5%
Cystolithotomy	2	28.5%
Gout	2	28.5%
Peptic ulcer	1	14.2%
Cholecystitis	1	14.2%
Pelvic inflammation	1	14.2%
Depression	1	14.2%
Urethrolithotomy	1	14.2%
Right renal injury	1	14.2%
Ano-stenosis	1	14.2%

Table 4: Family history of renal calculi

Family history of renal calculi	No. of patients	Percentages
Negative	6	85.7%
Positive	1	14.2%

Past history of prolonged drug ingestion (Allopurinal) was given by one patient.

Radiolcgical investigation confirmed the diagnosis of renal calculi in all seven patients. However, two patients were lost to follow-up. One after surgical removal of the stone the other before surgical removal of the stone.

The five patients who regularly attended follow-up were investigated in detail and results obtained are shown in table 5.

Table 5: Postoperative radiological, haematological, biochemical and dietary investigations

Patient identity	B.G. 243434	A.A. 282041	A.Z. 259937	O.A. 282932	K.R. 198397
Stone analysis	Calcium oxalate	Cacium oxalate phosphate	Calcium oxalate phosphate	Calcium oxalate	Calcium oxalate
Radiology: Plain X-ray abdomen	Calcified mesenteric nodes at L3 left side seen	Right renal calculi	Right renal calculi	Faint radiopaque shadow in the right kidney	Right renal calculi
I.V.P.	Right hydronephrosis and filing defect in lower third of right ureter	Stone in right renal pelvis and hydronephrosis	Non-functioning right kidney and hydronephrotic left kidney	Non-function right kidney and left hydronephrosis	Stone in the right renal pelvis and hydronephrosis left kidney normal
Retrograde	Confirmed the filling defect	Not done	Obstruction of right ureter at L5 level	Right hydronephrosis with calculi in the renal pelvis	Not done
X-ray of hands	Normal	Not done	Not done	Normal	Normal
Urine microscopy:					
W.B.C.	11 - 15/HPF	Few	>100/HPF	Nil	16/HPF
R.B.C.	10 - 12/HPF	Nil	20/HPF	Nil	Nil
Casts	?	Nil	?	Nil	Nil
Ova	?	Nil	Nil	Nil	Nil

Table 5: (Cont'd)

Patient identity	DG. 243434	A.A. 282041	A.Z. 259937	O.A. 282932	K.R. 1983
Urine: Culture	No significant growth	No bacterial growth	Mixed growth of Kelbsiella Pseudamonus, E-Coli and staphylococcus	No bacterial growth	Klebsiella species
Sensitivity	-	-	Nitrofurantain Nalidixic and Gentamycin Polymyxin B	-	Nalidixic acid
24 hour Urine: Analysis					
Volume	1.45 litres	1.80 litres	2 litres	2.2 litres	900mls
PH	5.5	Neutral	Acidic	6	5
Calcium	4.5 mmol/l	6 mmol/l	2.4 mmol/l	2 mmol/l	7 mmol/l
Uric acid	1.05g/24 hrs.	420mg	390mg	480mg	5.4mg
Phosphorus	0.66g	611mg	611mg	183mg	90mg/24 hrs.
Blood: Urea	14mg/dl	18mg/dl	31mg/dl	29mg/dl	22mg/dl
Creatinine	1.1mg/dl	0.8mg/dl	1.7mg/dl	-	1.0mg/dl
Calcium	2.9 mmol/	2.35 mmol/	2.2 mmol/	2.8 mmol/	2.3 mmol/
Phosphorus	0.6mg/dl	3.5mg/dl	2mg/dl	2.6mg/dl	3.7mg/dl
Uric acid	8.6mg/dl	7.1mg/dl	8.6mg/dl	4.5mg/dl	8.5mg/dl
Total protein	7.1 g/dl	7.3mg/dl	-	6.5 g/dl	7.6 g/dl

Table 5: (Cont'd)

Patient identity	B.G. 243434	A.A. 282041	A.Z. 259937	O.A. 282932	K.R. 198397
Diet pattern Breakfast: About 7 - 9 a.m.	2 cups of black tea with sugar	2 cups of white with sugar and bread	1 cup of white tea with sugar and 2 eggs	1 cup of white tea with sugar and bread	2 cups of black tea with sugar
Lunch About 1-2 p.m.	Rice + meat, fish, cabbage or spinach salf spieces used	Rice + meat, cassava leaves, salt and spieces used	Rice + meat, fish orange or banana	Rice + meat + potatoes	Ugali (maize meal) + meat, fish cabbage and spinach
Evening dinnes	Bread with soup	Bread with meat and 2 cups of black tea	Bread and 1 cup of tea	Rice or potatoes meat, beans 2 cups of white tea	Rice with meat, fish, cabbage or spinach 2 cups black tea
Miscellaneous	Cocacola orange, banana	Smokes 20 cigarrettes per day	Smokes about 20 cigarrettes a day	Pepsicola pineapple juice, smokes 10 cigarrettes a day	-

DISCUSSION

Aetiology:

In the majority of cases upper urinary tract stones form without apparent explanation. However, many anatomical and biochemical predisposing factors are known and these include; inadequate drainage, excess of normal constituents in the urine and the presence of abnormal constituents.[3]

Impaired drainage from the kidney may be congenital or acquired and these include; caliectasis, hydronephrosis, megaureter, horseshoe kidney, recumbency and medullary cystic disease (sponge kidney). Permanent caliceal dilatation may result from reflux nephropathy, chronic pyelonephritis, papillary necrosis and tuberculosis. Caliceal cysts as well as hydronephrosis may also follow calculus formation. Medullary cystic disease is rare but important anatomical abnormality marked by the dilatation of the terminal collecting ducts in the pyramids which are in turn expanded as are the calices into which they drain. The condition is bilateral in three quarters of the cases and usually contain multiple calcium phosphate calculi. Immobilization of the whole patient like a long-standing orthopaedic or paraplegic case is associated with stone formation. In these cases mobilization of calcium from the skeleton is an additional factor.

Sluggish urine facilitates crystallization and increases the risk of infection.

The other group of anatomical causes of renal calculus formation is calcification within kidney substance as nephrocalcinosis, Randall's plagues and Carr's concretions.

Nephrocalcinosis usually associated with hypercalcaemia and hypercalciuria which may be caused by hyperparathyroidism (primary or seconday), renal tubular acidosis, malignant disease associated with bone destruction or secretion of parathyroid hormone like substance, myelomatosis, lymphoma and leukaemia, Paget's disease of bone, hyperthyroidism with bone destruction, hypothyroidism in children vitamin D intoxication, Sarcoidosis, milk-alkaline syndrome and increased intestinal absorption of calcium. It may also occur in

damaged renal tissue as dystrophic calcification.

In clinical practice the majority of cases of nephrocalcinosis are caused by primary hyperparathyroidism and renal tubular acidosis but others have no obvious causation and are termed idiopathic. The increased parathyroid hormone in hyperparathyroidism is associated with increased bone resorption, increased intestinal calcium absorption as a result of increased activity of 1.25 di-hydroxyvitamin D_3, decreased renal tubular resorption of phosphate and increased renal tubular resorption of calcium. These lead to hypercalcemia, hypophosphatemia and phosphaturia developing. In renal tubular acidosis in which distal tubule fails to maintain a normal hydrogen ion gradient between tubular cells and urine results in a low excretion of titratable acid. Systemic acidosis, with urinary pH about 6 develops and there is hypercalcaemia, hypercalciuria, decreased urinary citrate resulting in nephrocalcinosis. The disease is inherited as an autosomal dominant trait of varying penetrance.

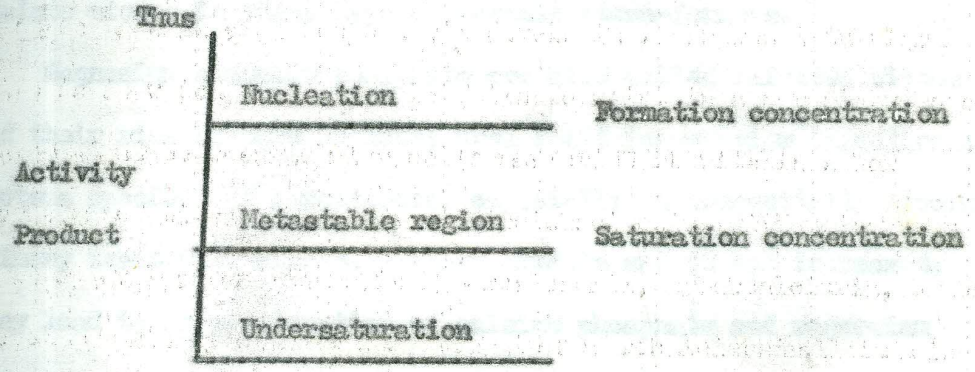
Calculogenic material of uric acid salts and cystine may also be deposited in Kidney substance.

Randall's plagues are focal calcified lesions in the medulla initially subepithelial but may ulcerate the epithelium and become exposed to urinary calcium or uric acid salts facilitating growth.[4] Vermooten[5] found plagues to be present in 17.2% of South African whites and only 4.3% of Bantus who rarely formed stones.

Carr[6] suggested that renal calculi formed when normal lymphatic drainage becomes impaired. This could occur either because of overloading with microliths or by an impairment of drainage secondary to inflammation and fibrosis. However, the finding of intranephronic calculi has led to the suggestion that both Randall's plagues and Carr's intra-lymphatic calculi represent intranephronic calculi that have, because of the associated inflammation and obstruction, migrated to a subepithelial or intralymphatic position.

Biochemical analysis of renal stones categorises stones into four main groups; calcium, magnesium, ammonium phosphate, uric acid and cystine. Most theories state that in stone urine there is an excess of agents resulting in precipitation or lacks those agents which tend to inhibit crystallization.

The urine can be undersaturated, supersaturated or oversaturated and stone formation occurs in saturated solution and involves crystal, nucleation, growth, and aggregation.



Crystal growth rates are a function of solution supersaturation with regard to the crystalloid ions. The greater the level of supersaturation the greater the growth rate. About 90 percent of recurrent calcium stone formers have aggregated crystals in the freshly voided urine filtrate, whereas less than one fifth of normal subjects have similar findings. It is thought that stone formers lack naturally occuring urinary substances like, pyrophosphates, citrates, diphosphates and magnesium which can inhibit crystal formation and growth. Stone formation may also be secondary to metabolic abnormalities like hyperparathyroidism, hyperoxaluria and cystinuria or in presence of abnormal constituents of the urine like bacteria, epithelial sloughs and foreign body like unabsorbable sutures. Hyperkeratosis of urinary epithelium which occur with vitamin A deficiency provides the debris upon which stones may form.[3]

Types of renal stones and their specific risk factors:

Calcium oxalate and calcium phosphate are the most common urinary stones.[7] Their urinary risk factors include: increased urinary calcium,

oxalate, increased pH and reduced urine volume these lead to supersaturation of calcium oxalate and/or calcium phosphate while reduced acid mucopolysaccharides and increased uric acid lead to reduced inhibitory activity. Robertson et al[8] showed that male, idiopathic calcium oxalate stone-formers have reduced levels of inhibitory activity in their urine and 50 percent of these patients have reduced inhibitory activity due to hyperuricosuria with anti-inhibitory effect. Allopurinol reduces the incidence of calcium oxalate stones in these hyperuricosuric stone-formers.[9]

Magnesium ammonium phosphate are also called infected stones and their risk factors include; urea splitting organism infection like proteus species and streptococci especially in congenitally abnormal urinary tract with stasis, urinary ammonia and pH are increased. They lead to supersaturation of calcium phosphate and magnesium ammonium phosphate, abnormal crystalluria and stone formation. Bacterial degradation also causes reduction in urinary pyrophosphate and citrate thus reducing inhibitory activity for calcium phosphate crystallization. Urinary citrate reduction also occurs in renal failure. Inflammation of urinary tract increases urinary mucoproteins with increased tendency of nucleation and crystalluria of calcium phosphate and magnesium ammonium phosphate.

Uric acid stone formers are commonly found in industrialized areas of the world and about 20 to 25 percent of these patients have primary or secondary gout.[10]

There is also an increased tendency to form uric acid stones after ileostomy. However, majority of uric acid stone-formers do not have high excretion of uric acid and are classed as indiopathic. Low pH is the common abnormality found in these patients.[11]

Since uric acid has a dissociation constant (pKa) of 5.46, at pH values below this figure most of uric acid is in the undissociated form and below pH 5.3 spontaneous precipitation and crystalluria may occur. The mean 24 hour urinary pH of idiopathic uric acid stone

formers is 5.32. Patients with hyperuricosuria, an excessive dietary intake of purines is generally found to be the cause.

Additional pre-urinary risk factors include abnormal purine metabolism, ammonia production, purine ingestion and reduced fluid intake particularly in hot environment will lead to reduced urinary valume and supersaturation, uric acid crystolluria and stone formation.

Cystine stone-formers suffer from an autosomal recessive disease characterized by a defect in the tubular re-absorption of cystine, lysine, arginine and ornithine.

Cystine is normally excreted in the range of 10 to 100 μmol/ℓ. In patients with cystinuria this is increased to 20 - 600 μ mol/ℓ in heterozygotes and to 1,400 - 4,200 μ mol/ℓ in homozygotes.[12] within the pH range 5 to 7 at 37°C is about 1,250 μ mol/ℓ urine from homozygous cystinuria is excessively supersaturated with cystine and this accounts for the frequent crystalluria and stone formation of this group.

Xanthine calculi formers have a congenital defect in purine metabolism caused by a deficiency of xanthine oxidase which catalyses the oxidation of hypoxanthine to uric acid via xanthine.[13] One third of these patients form xanthine calculi usually beginning in infancy. Calculi arise in the collecting system when excessive supersaturation of the urine occurs. Myopathy may occur as a complication due to deposition of hypoxanthine and xanthine crystals in striated muscle.

Cystathionine stone-formers have a rare familial disorder marked by excessive excretion of cystathionine which can be detected by chromatography of urine. The condition is generally accompanied by a variety of developmental defects, mental retardation and thrombocytopenia.

Silicaseous calculi associated with consumption of magnesium trisilicate for dyspepsia have been reported.[14]

In this study five patients had their renal stones analyzed. Of these five patients, three had calcium oxalate stones and two had calcium oxalate phosphate stones. Rest of the postoperative investigation findings are shown in table 5.

Clinical manifestation

Clinical features of renal calculus are influenced by the effects of urinary obstruction, infection and local irritation. Although some stones (especially phosphate stones) remain dormant for a long period causing progressive renal destruction and uraemia, pain is the leading symptom in 75 percent of cases.[2] Pain depends on size, site and mobility of the stone. Large staghorn calculus usually presents with fixed dull ache in the loin while small mobile stone may enter the ureter and present with acute, agonizing and proxysmal ureteric colic usually of sudden onset. As the stone enters the lower third of ureter, pain is referred along the two branches of genito-femoral nerve to the testis in the male, labium majus in female and thigh in both sexes when the stone enters the intramural portion of the ureter pain is referred to the tip of the penis in male and there is increased frequency of micturition in both sexes. In more than 50 percent of cases the stone is passed spontaneously.[2] However, impaction of the stone may occur at any of the following narrowings of the ureter, uretero-pelvic junction, crossing of iliac artery, justaposition of vas deferens or broad ligament, entering bladder wall and ureteric orifice. When stone becomes impacted attacks of colic pass off and give place to dull pain usually in the iliac fossa in which case misdiagnosis of appendicitis may be made. In this study two patients had appendisectomy done prior to the diagnosis of renal stones.

Haematuria varies from microscopic to frank. Large stone in the renal pelvis may usually be associated with painless microscopic haematuria whereas hard, spiky, small, mobile calcium oxalate stones usually cause frank haematuria by traumatizing urinary epithelium particularly during the attack of ureteric colic. Pyuria may present in varying amounts particulary with infected triple phosphate stone.

Gastrointestinal symptoms like nausea and vomiting may accompany ureteric colic and occasionally diarrhea will be present. A reflex paralytic ileus from retroperitoneal irritation may produce abdominal distention.

History of previous urinary tract stone, infection and prolonged illness involving recumbecy is important as is the family history in some cases of uric acid stone disease and all cases of cystinuria. History of dietary habits especially of foods with high calcium or oxalate content or excessive intake of milk and ingestion of drugs like alkalis is important.

In this study abdominal pain was a presenting symptom in all the 7 patients (100%) as shown in table 2 which also shows other presenting symptoms as seen in this study.

Previous diagnosis and treatment the patients received before the renal stone was diagnosed are shown in table 3.

Family history was negative in 6 patients (85.7%) and positive only in one patient who had calcium oxalate phosphate stone.

On examination the patient in acute attack rolls about in agony. The skin may be cold and clammy, pulse is rapid and temperature may be subnormal. Renal angle tenderness may be present. Pressure on the lateral abdominal muscles of the affected side will provoke and aggravate the pain and muscular rigidity may be present. Occasionally patient may present with hydronephrosis or pyonephrosis with palpable kidney. Anuria is due either to impaction of calculi into the ureter on each side, or blockage of the ureter in a remaining solitary kidney, or occasionally due to reflex anuria with shut down of the normal opposite kidney.

Differential diagnosis should include primary hydronephrosis which can be distinguished by radiology. Right ureteric colic must be distinguished from biliary colic by its history and presence of tenderness under the rib margin and absence of renal angle tenderness. The pain of biliary colic is usually referred to the back and right shoulder. Haematuria is absent. In acute appendicitis, maximum tenderness is over McBurney's point and haematuria is usually absent. Acute intestinal obstruction is differentiated by the presence of general abdominal distension, failure to pass flatus per rectum and radiological findings.

Investigations

Radiological, bacteriological and biochemical investigations are required to establish diagnosis and obtain information about the etiology of the stone and provide a guide to prevention of recurrent stones.

The most important radio opaque constituent is calcium and calcium oxalate calculi are the most opaque on X-rays while those of calculum phosphate are less dense. Cystine calculi are radio-opague while pure uric acid and xanthine calculi are radio-lucent and require intravenous pyelogram for diagnosis.

A plain abdominal X-ray will show the presence of stone in 90 percent of cases. However, before X-ray is taken bowels should be emptied. Radio-opaque renal stone should be differentiated from calcified lumbar or mesenteric lymph node, gall-stone, concretion in the appendix, drug like fersolate or foreign bodies in the intestine, phleboliths, ossified tip of twelth rib, chip fracture of transverse process of a lumbar vertebra and calcified suprarenal gland.

Intravenous urogram helps to confirm the presence, size and position of the stone and whether other stones are present in either the ipsilateral or contralateral renal tract, the degree of function and/or obstruction of each kidney and evidence of any incidental disease in the urinary tract. Exposures made during full inspiration and full expiration and note if opacity moves with the kidney and if measurements from the lower pole of the kidney to the opacity remain constant the shadow is intrarenal. On lateral view intrarenal opacity lies posterior to the vertebral bodies. Nonradio-opaque calculus is demonstrated as a filling defect but similar defect is seen in papilloma of the renal pelvis.[2]

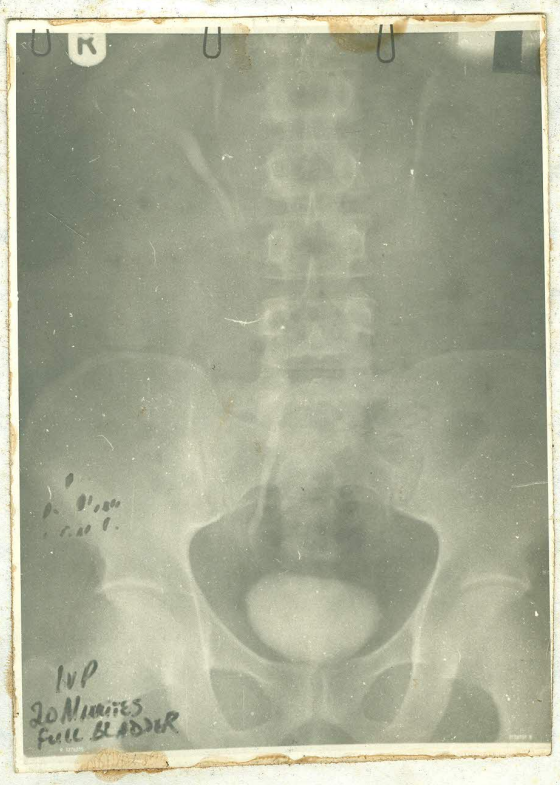
Retrograde pyelograph gives a clear delineation Antero-posterior and oblique posterior views taken with ureteric catheter in place should clearly identify whether or not an opacity is within the ureteric lumen. Illustration 11 of intravenous urogram shows double ureters on the right side and a ureteric stone impacted in the lower third of right ureter with hydroureters.

Illustration 11:

Intravenous urogram. Shows double ureters on the right side. There is impacted ureteric stone in the lower third of the right ureter and hydroureters.



Illustration 12: (The same patient shown in illustration 11) Preoperative urogram showing that the right ureteric stone spontenecously dislodged and hydroureters disappeared.



When the stone in the lower third of ureter cystoscopy shows a patulous ureteric orifice with haemorrhages around it or stone may be seen at the orifice.

Biochemical investigations are carried out initially to establish the type of stone formation involved and to classify the patient into one of the three main groups; infection stone formers, metabolic stones like, uric acid and cystine; calcium-containing stone group which includes idiopathic stone - formers, hyperparathyroidism hyperoxaluria and renal tubular acidosis.

In addition to usual haematological counts, particular attention is paid to the serum calcium, phosphate and uric acid and estimations of serum urea, creatinine and electrolytes should also be measured. Patient who has a calcium stone and hypercalcaemia most often has hyperparathyroidism and if he is not uraemic an associated hypophosphataemia is usually present. Serum uric acid estimation is important both in relationship to uric acid stone and to incidence of calcium oxalate stone formation. Serum urea and creatinine estimations provide a guide to renal function. Estimation of plasma parathormone is done in specialized centers.

Urinary studies include examination of urinary sediment, bacteriology, measurement of the urinary pH and a qualitative measurement for cystine. Microscopic examination of urine will usually show the presence of red and white cells as well as bacteria if infection is present examination of urine sediment after centrifugation may show presence of crystals especially oxalate, uric acid or cystine. Urine culture frequently grows B proteus which is the commonest organism responsible for infection stones but other species which may produce urease include staphylococcal, Klebsiella, pseudomonas and B-Coli these were found in the two positive cultures in this study but not B. proteus. The urea splitting organisms release ammonia increasing the pH to about 8.0.

Stone screen test on 24 hour urine sample includes; calcium (normal is 1.25 - 10.0 mmol in male, 1.25 - 9.5 mmol in female) Phosphate (noamal 16 - 22 mmol), oxalate (normal 0.2 - 0.5 mmol), uric acid (normal 10 - 30mg).

Results of 24 hour urine tests as obtained in this study are shown in table 5.

Other urinary tests are for creatinine, volume, pH. Cystinine quantitative test is done when the qualitative cyanide - nitroprusside test is positive and urine chromatography helps to detect presence of cystathioninuria.

Chromatography, cyanide - nitroprusside test and cystinine quantitative test were not done in this study. Results of the tests done in this study are shown in table 5.

Any stone obtained by surgery or spontaneous passage should be submitted for analysis of its chemical constituents. When stone disease is classified according to chemical composition it is found that about 70 percent are calcium oxalate and/or calcium phosphate,[15] about 20 percent are composed of magnesium ammonium phosphate (infected stones). They are commoner in females (14 percent) than in males (6 percent). Uric acid stones form about 5 percent and cystine calculi form about 2 percent. Rare forms of calculi are composed of xanthine, crysthionine and silica.

In this study all stones analysed were calcium oxalate (3 patients) and calcium oxalate and phosphate (2 patients).

Surgical treatment

Surgery is indicated for; relief of pain, preservation of renal function depressed by outflow obstruction; relief of symptoms of urinary tract infection, correction of any abnormalities that may predispose to further calculus formation.

The common stone situations requiring surgical treatment encountered clinically can be classified into four grades:[16]

Grade I Calculi are small immobile or mobile. Usually solitary but could be multiple particularly in medullary sponge kidney or in renal papillary necrosis. If asymptomatic, they do not require surgical treatment but patients are screened metabolically to exclude any significant biochemical abnormality. About 80 - 90 percent of small mobile stones move rapidly into ureter and pass to the bladder giving rise enroute to classical ureteric colic.[16] However, stones larger than 0.5 cm in diameter may remain trapped in the renal pelvis causing recurrent attacks of loin pain and haematuria as they become intermittently impacted in the pelvi-ureteric junction or upper ureter. Such calculi should be removed by pyelolithotomy.

Grade II stone is large solitary calculus occupying the whole renal pelvis but not involving calyces. Usually patient presents with long standing mild backache but occasionally patient presents with exacerbation of pain and haematuria following abrasion of transitional epithelium by the stone. They may produce hydrocalycosis, parenchymal atrophy with or without infection and should be removed by pyelolithotomy.

Grade III stones consist of large staghorn calculus or diffuse multiple calyceal stones. Staghorn calculi are usually of mixed phosphatic composition caused by persistent urinary infection. The patient often presents with loin pain, recurrent attacks of urinary infection and lower tract symptoms of frequency, dysuria and haematuria. If left untreated, there is progressive loss of renal function from a combination of obstructive hydronephrosis, parenchymal atrophy and pyelonephritis. Such stones should be removed by nephrolithotomy and the associated urinary tract infections treated vigorously to prevent stone recurrence. The diffuse multiple calyceal grade III calculi usually coalesce and form complete staghorn or cast calculus. Such calculus should also be removed by nephrolithotomy.

Grad IV calculi are usually as a result of neglected gradeIII stones. The kidney is severely damaged and little renal function remains. If contralateral kidney is normal nephrectomy is the best

treatment. However, if both kidneys are involved, conservative nephrolithotomy is undertaken preserving the limited renal function sufficient to sustain life and avoid the need for renal replacement therapy.

When surgery is decided, mandatory pre-operative considerations include radiological investigations, renal function tests, examination of the urine and blood grouping and cross match. Thus within one month prior to surgery, intravenous urogram is performed to demonstrate the anatomical structure of the kidney, the position of stones and the functional capacity of the kidneys. If there is any doubt about ureteric patency, a retrograde ureterogram must be performed. Plain film of renal and ureteric area should be taken immediately before operation preferrably on the way to theatre because renal or uteric stone can move dramatically in a few hours. X-rays should be displayed in the operation theatre.

In this study illustration 11 shows right hydroureters due to the stone impacted in the lower third of the ureter. However, the immediate preoperative urogram shown in illustration 12 shows that the ureteric stone spontaneously dislodged.

Renal function tests including blood urea and cleatinine will help to assess overall renal capacity. Estimations of other serum electrolytes are also carried out. High potassium and low bicarbonate levels may indicate that medical correction of serum biochemistry or even dialysis may be required to render the patient fit for surgery.

Urine culture and sensitivity is done and if positive antibiotic therapy is started 24 hours before operation. At least 2 units of cross-matched blood should be available for any renal operation.

Optimal pre-operative manoeuvres include; radioactive renography which helps to estimate relative contribution of each kidney to overall renal function. Where there is bilateral renal damage nephrectomy is is advised if the function of a severely damaged kidney is below 15 percent of overall renal capability. Above this level a conservative technique of nephrolithotomy is advisable.

It is useful to set up intravenous infusion an hour before operation and give at least 1.0 litre of dextrose saline to promote diuresis. At operation and prior to pedical clamping, 20 grams of mannitol in 200 mls of saline as an intravenous infusion provides a further degree of protection to renal tubules.

Operations for upper urinary tract calculi include; pyelolithotomy, nephrolithotomy, nephrectomy Bench surgery and uretero-lithotomy (open or endoscopic).

Pyelolithotomy is indicated when a stone is in the extrarenal pelvis. Lumbar incision is commonly used, lower pole of kidney and upper ureter are exposed. Silastic rubber sling is passed around the ureter and gentle traction helps to elevate the lower pole of the kidney and allow further dissection of renal pelvis. By fitting lower pole forward, renal pelvis is displayed posteriorly and a linear incision made over the palpable stone exposes the stone which is removed with stone forceps. After the stone has been removed irrigation with normal saline using a 14 French catheter and 50 ml syringe will help remove small mobile stones and a bougie is passed through the pelvi-ureteric junction to exclude any obstruction lower down. If the kidney is known to be uninfected the Pyelotomy is closed by 4/0 plain catgut. If the kidney is infected, nephrostomy is performed before closing renal pelvis.[2]

When Staghorn calculus or multiple small calyceal stones are present, extended pyelolithotomy (Gill - Vernet) is performed to remove the stones. After the renal pelvis and upper reter are exposed and stabilized in a sling, dissection is extended using a pair of blunt scissors to incise the sinus plane between the renal pelvis and overlying renal parenchyma and expose calyceal necks. Pyelotomy and removal of stones are done. The open necks of calyces can then be seen and if dilated, peripheral stones are removed with stone forceps or irrigation. If calyceal necks are normal size it is impossible to remove peripheral stones and in such cases the kidney is

fully mobilized so that control of the pedicle can be obtained if necessary and remove stones by direct incision of the parenchyma over the calyx.

Nephrolithotomy may be necessary when approach to the pelvis is difficult like in presence of adhesions. Thermocoupic - regulated cooling of the kidney, by ice chips in polythene bags or liquid nitrogen circulation through coils placed around the kidney, enables the renal pedicle to be clamped to provide a bloodless field and unharried surgery.[2] Incision is made just behind and paralled to the most prominent part of the convex border of the kidney (Brodel's line) where only terminal branches of the anterior and posterior renal arteries meet and the stone is removed, bleeding points tied with 4/0 catgut and renal incision closed by interrupted catgut sutures tied over the oxycel or muscle to enhance haemostasis. Hypothermia of 15 or 20°C will provide about 3 hours of good protection of renal function. However, purine nucleotide inosine in 30ml of diluent as trophycardyl at room temperature injected directly into distal renal artery preserves renal function without cooling as long as ischaemia does not exceed 1½ hours.[17]

On declamping, 40mgs of intravenous Frusemide is given and post-Operative hydration is maintained with 1 litre of dextrose saline given 8 hourly. Lumbotomy patients are able to walk 24 hours postoperatively. Those with loin incision take 2 days. On 7th day a nephrostogram is done if no fragments of calculi seen, it is removed. Skin sutures are usually removed on 8th day and patient discharged. However, when fragments of calculi are seen, irrigation with chemical agents like Renacidin which is a mixture of citric acid, D-Gluconic acid, magnesium hydroxycarbonate, magnesium acid citrate and calcium carbonate is done, to dissolve the fragments. 10 percent solution in 500ml of distilled water is infused through nephrostomy tube 6 hourly for 7 days.

There is controversy as to whether partial nephrectomy should be done at all since it has been shown histologically that calculogenic foci are widespread through the renal parenchyma and not restricted to any one part and the correction of drainage deficiencies of lower calyces by pyelocalycostomy followed by control of any metabolic abnormalities would prevent recurrences.[18]

For grade IV type of calculus disease in severely damaged kidney then nephrectomy is indicated if the other kidney is normal.

Extracapsular mobilization of the kidney is done and ureter traced down to the pelvic brim divided and stump ligated. The renal pedicle is dissected, the artery and vein are doubly and separately ligated and kidney removed.

Bench surgery is advocated in which the kidney is transfered to a secondary operating table where it is perfused with cooled solution and electrolyte fluid (collin's solution) and calculi removed. The kidney is then re-implanted in the iliac fossa. End to end anastomosis of renal artery to internal iliac artery and end to side anastomosis of renal vein to external iliac vein and ureter re-implanted into bladder.

Other method advocated for removal of intra-renal calculi is coagulation pyelolithotomy in which a solution which rapidly forms a coagulum is introduced in the renal collecting system and coagulum mass with the entrapped calculi is removed through a simple pyelotomy.[19]

Ureteric calculi

Expectant treatment of ureteric calculi is adopted because most small ureteric calculi will pass naturally and progress can be observed by regular X-ray (illustration 11, and 12). However, ureteric stone of 0.5 cm or more may impact at any of the following sites: Pelvi-ureteric junction, at the point where the ureter crosses the iliac vessels; at the juxtraposition of vas deferens or at the broad ligament, on entering bladder wall or at the ureteric orifice. Surgical intervention (instrumental or operative) is indicated if; repeated attacks of ureteric colic produce no advance of the stone, the

stone is enlarging and stationary, hydronephrosis is increasing, the urine is infected or, the stone when first seen is thought to be too large to pass.

Endoscopic ureterolithotomy requires use of ureteric catheter, Dormia basket or Davi's nylon loop passed through the cystoscope. Small stone of about 0.5 cm will pass after ureteric catheterisation. Dormia basket is used to dislodge and deliver a stone in the lower third of ureter. There is danger of ureteric avulsion particularly if the stone is larger than ureteric orifice. Davi's nylon loop may also be used to engage ureteric stone and draw it into the bladder.

Removal of low ureteric or intramural stone can be performed, extravesically, intravesically, transperitoneally or vaginally. Low Pfannenstial incision is used in extravesical approach. The ureter is identified, stone palpated and is removed. Most stones in intramural ureter can be visualized by endoscope and when ureteric orifice mucosa is incised stone falls into bladder. There is a postoperative danger of ureteric reflux. Similary the intravesical portion of ureterocele is incised with diathermy knife and calculi fall into bladder where they are removed by irrigation.[20] There is danger of postoperative ureteric reflux and persistent urinary infection. Re-implantation of ureter may be required.

Bose[21] advocated approaching lower ends of ureters transperitoneally after midline subumbilical incision but there is a danger of intraperitoneal urinary leakage, peritonitis and paralytic ileus. Calculus in the lower 2 inches of ureter of multiparous female can be removed vaginally.[22]

In this study pyelolithotomy was done in four patients who had clculi in the renal pelvis. Ureterolithotomy was done in 2 patients who had ureteric stone impacted in the lower one third of the ureter. One patient refused to have pyelolithotomy done for stones in the renal pelvis.

2. Medical treatment

Medical treatment involves use of various medications in an attempt to dissolve stones, prevent further growth of existing stones or as a prophylaxis against future stone formation.

Patients who have idiopathic calcium oxalate stones should avoid calcium rich foods like milk and milk products, drugs like antacids containing aluminum hydroxide, calcium aspirin and large doses of vitamin C. Oxalate rich foods like nuts, spinach and tea be restricted. Treatment is considered effective if urinary oxalate crystals have been made to disappear. Promising results are found with low calcium, low oxalate diet. Patients should be encouraged to drink more water to achieve excretion of 3 litres of dilute urine a day.

In this study dietary history taken from five patients showed high intake of tea, milk and spinach. Stones collected from these patients mainly contained calcium oxalate (3 patients) and calcium oxalate with phosphate (2 patients) "table 5". The patients were advised to take low calcium and low oxalate diet and the patients were encouraged to drink more water.

Table 6 shows drug therapies that the patients with idiopathic hypercalciuria may follow. The most effective are the first three drugs.[23] Patients with primary hyperoxaluria should be treated by pyridoxine.[24] Secondary hyperoxaluria can be treated by stopping ascorbic acid if any and cholestyramine where steatorrhea is present. Low oxalate diet is advisable.

Patients with mixed phosphatic infected stones should be treated by surgical removal, antibiotic therapy, oestrogen therapy to raise urinary citrate and oral aluminium hydroxide to divert phosphate from urinary to alimentary excretion. Urease inhibitor, acetylhydroxamic acid can be used to inhibit alkalinization of urine.[25]

Table 6: Patients with idiopathic hypercalciuria may follow the following drug therapies

Drug	Action	Cost	Side effects	Daily dose
Thiazides e.g. Bendrofluazide	Lowers urine calcium by action on renal tubules	Cheap	D. Mellitus Gout Fatigue	5 - 10 mg
Celluse phosphate	Lowers urine Ca^{++} by blocking obsorption from gut	Expensive	Diarrhea	15 - 30 mg with meal
Inorganic phosphate	Lowers urinary Ca^{++} increases urinary pyrophosphate	Cheap	Diarrhea Oedema	10-20g.
Pyridoxine	Lowers urinary oxalate	Cheap	Nil	10 - 20mg
Magnesium	Inhibits crystal growth	Cheap	Diarrhea	420 1200mg.
Methylene blue	Inhibits Ca^{++} Oxalate and phosphate crystal growth	-	Diarrhea Gastritis	65mg.

Uric acid stone treatment includes diet, akali, water and allopurinol. Diet should omit purine rich foods like liver, kidney and fish. To keep pH above 6.5, sodium bicabonate can be used. Allopurinol blocks the action of Xanthin oxidase in oxidizing hypoxanthine to xanthine and xanthine to urate. Godfrey and Rankin[26] obtained uric acid stones dissolution on giving a combination of allopurinol 100mgs. tds., water and alkalis.

Cystine stones can be dissolved by keeping urine flow rate at 3 litres per 24 hours especially if urine is of alkaline pH. D-Penicillamine or Thiola (Alpha - Mercaptopropionyl glycine) combines with cystein to form a soluble complex.

Administration of alkali, high fluid intake and restriction of dietary purine intake are used to treat xanthine calculi but these are more difficult to treat.

Regular drug therapy as shown in table 6 was not administered to any of the patient during the time of this study.

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BENIGN PROSTATIC HYPERTROPHY

SUMMARY

Thirty patients, with Benign Prostatic Hypertrophy (BPH) causing obstructive uropathy are presented. One patient is presented in detail. Etiological factors, clinical features, treatment and postoperative complications are discussed and Review of literature is presented.

INTRODUCTION

Benign enlargement of the prostate is a common world wide disease of males above 45 years of age. Sir Benjamin Brodie related prostatic disease with the normal process of aging by stating that the prostate gland increases in size as the hair of the head grows grey.[1]

Lissoos[2] reported the incidence of benign prostatic enlargement to be low in Bantus. However in U.S.A. the incidence among negroes is found not to be different from their white counterparts.

The disease is rare among the Chinese and comparatively uncommon among the Japanese. In one hospital in Sudan Kambal[3] recorded 395 cases in 7 years. Hiza[4] recorded 79 cases out of 1812 patients admitted in general surgical department in 1971. Nkulila and Phillip[5] recorded benign prostatic hyperplasia as one of the commonest urological problems and second only to hydrocele during the period from 1977 to 1979.

In this study of 30 patients treated by surgical firm III between July 1980 to May 1982 the minimum age recorded was 48 years, the maximum age was 80 years. The mean age was 55 years. The age incidence is shown in table 1.

CASE REPORT

M.L. I.P. No.257973, 60 year old male, Mkwele by tribe and a resident of Bagamoyo presented with history of acute retention of urine of 3 days duration. He said he had dysuria and poor stream of urine for one year prior to admission. There was gradual reduction in the

force of the stream of urine associated with terminal dribbling. He denied any history of urgency, hesitancy or haematuria. However, he said about 10 days prior to admission he developed retention of urine and he was relieved in Morogoro hospital by catheterization. The retention recurred 7 days after discharge. He denied any other history of major illness in the past.

Examination on the day of admission revealed an ill looking elderly man. He was not dehydrated, not pale and not jaundiced. Blood pressure was 90/60, pulse 80/min. regular and of good volume. He was a febrile. Respiratory, cardiovascular and central nervous systems were normal. The abdomen was distended in the hypogastrium and bladder was palpable up to just below the umbilicus and it was tender. Palpation of renal angles was essentially normal. Digital rectal examination done after catheterization and emptying of the bladder revealed grade III enlargement of prostate which felt smooth, with well marked median groove, firm in consistency and mucosa over it was freely mobile. There was no blood on the glove of the examining finger and the stool on the glove looked normal.

Clinical diagnosis of benign prostatic hypertrophy with urinary retention was made.

Preoperative investigation done included; blood urea = 31mg/dl, serum creatinine = 1.2/dl. Full blood picture was reported as normal. Cystoscopy, serum acid phosphatase and alkaline phosphatase were not done. Stool microscopy revealed presence of hookworm ova. Urine examination was normal.

Patient was treated for hookworm using alcopar 5 grams on alternate days for 3 days. Blood grouping and X-match was done and with preoperative haemoglobin of 11.2gm% patient was operated on 1st January, 1982.

Under spinal anaesthesia, cleaning and drapping were done. Midline subumbilical incision was made to expose the bladder. After applying stay sutures the bladder was opened extraperitoneally. The urine was sucked out and on inspection a bladder stone was found and removed. Enlargment of the prostate was confirmed. Digital

enucleation was done, haemostasis was achieved by applying warm gauze in the prostatic bed, ligating bleeders at about 5 and 7 O'clock positions and using diathermy on small bleeders. 3 way Foley's catheter was passed and its balloon placed in the prostatic bed.

Continuous drainage with normal saline was established and bladder closed in two layers. Corrugated rubber drain was placed in the retropubic space and abdomen was closed in layers.

Postoperatively the patient received two units of blood and was put on antibiotics, analgesics and observation of vital signs.

Recovery was fairly good. Bladder drainage was stopped after the urine was clear on the forth postoperative day. However wound sepsis was noticed on the 8th postoperative day, about 100 cc of pus was drained after removing two stitches at the site of discharge and was sent for culture and sensitivity. Staphylococcus aureus sensitive to cloxacillin, gentamycin and erythromycin was isolated. Patient was put on erythromycin and daily cleaning with eusol was advised. Sepsis was controlled and patient discharged from ward on 20th postoperative day. He has remained symptom free since then.

Summary of presentation of the 30 patients is shown below.

Table 1: Age incidence

Age range (years)	No. of patients	Percentages
50 or below	3	10%
50 to 60	14	46.7%
60 to 70	9	30%
70 to 80	3	10%
80 or more	1	3.3%
Total	30	100%

Minimum age at presentation was 48 years.

Maximum age at presentation was 80 years.

Table 2: Presenting symptoms as seen in this study

Presenting symptom	No. of patients	Percentages
Retention of urine	20	66.6%
Dysuria	18	60%
Frequency	15	50%
Poor stream of urine	13	43.3%
Terminal dribbling	7	23.3%
Hestancy	5	16.6%
Haematuria	2	6.6%
Urgency	2	6.6%
Incontinence	1	3.3%

Associated complications of primary disease (found preoperatively) included the following:

Table 3: Preoperative investigation findings included

Investigation finding	No. of patients	Percentages
Urinary tract infection	10	33.3%
High blood urea	7	23.3%

Table 4: Concomitant conditions encountered in the study included

Condition	No. of patients	Percentages
Bladder tumour	4	13.3%
Bladder stone	2	6.6%
Hypertension	2	6.6%
Inguinal hernia	4	13.3%
Haemorrhoids	1	3.3%

Table 5: Number of patients and age distribution according to type of prostatectomy done

Age range (yrs)	No. of patients and type of prostatectomy done		
	Transversical	Retropubic	Transurethral
≤ 50	2	1	
50 to 60	12	2	
60 to 70	8		1
70 to 80	3		
>80	1		
Total	26	3	1

Thus: 1. 26 patients (86.6%) had transvesical prostatectomy done.
2. 3 patients (10%) had retropubic prostatectomy done.
3. 1 patient (3.33%) had transurethral resection done.

Postoperative complications encountered according to operation done were as follows:

Table 6:

Complication	No. of patients according to type of prostatectomy done		
	Transvesical	Retropubic	Transurethral
Haemorrhage	4	1	
Epididymo-orchitis	2		
Wound infection	9	1	
Suprapubic fistula	4		
Incontinence	2		
Bladder neck stricture	2		
Impotence	1		

Duration of postoperative bladder drainage and hospital stay according to type of prostatectomy done were found as follows (table 7).

Table 7:

Type of prostatectomy	No. of patients and duration of bladder drainage (days)				No. of patients and duration of hospital stay (days)			
	<7 days	7-14	14-21	>21 days	<7 days	7-14	14-21	>21 days
Transvesical	2	19	3	2		16	7	3
Retropubic		2	1				2	1
Transurethral		1					1	

There was no intra or postoperative death.

DISCUSSION

Etiology:

There are various theories postulated and perhaps have a contributory role though none has been accepted as the cause of prostatic enlargement. The theories include: hormonal theory; consitutional, racial and environmental factors; arteriosclerotic theory; inflammatory theory; metabolic and nutritional factors and neoplastic theory. Hormonal theory the most widely accepted of them all.

Hormonal theory

The prostate is under control of androgenic and estrogenic hormones. It has been shown that prepubertal castration or destruction of the pituitary prevents normal development of the prostate, that postpubertal castration or destruction of the pituitary results in atrophy of the prostate. It has been shown that hyperplasia of the prostatic muscle occurs when transplanted hormologously in the eye of the estrogen treated rabbits.[6] Franks[7] has also demonstrated nodular hyperplasia of the stroma around the utricle in Guinea pigs treated with estrogen. It is thus agreed that there is some disturbance of the androgen - estrogen ratio in prostatic hyperplasia probably in favour of estrogen. As age advances the male hormone diminishes while the quantity of the estrogenic hormone is not decreased equally and prostate enlarges because of predominance of the estrogenic hormone.[1] It has been stated that alteration in androgen and estrogen concentrations and possibly changes in receptor responses in the prostatic cells that occur with age are the main factors that allow the gland to hypertrophy in later life.[8]

Constitutional, racial and environmental factors

Moore[6] quates the views of Blatt (1926) who described patients with prostatic hyperplasia to have certain specific physical features that represent feminine characteristics. Moore[6] found that among the European and American males in various countries the incidence of prostatic hypertrophy was similar.

In South Africa, it was found that prostatic hypertrophy occured twice as commonly in the Asiatic than in the African.[9] The disease is also uncommon in the Chinese and Japanese.[6]

Arteriosclerotic theory

It has been shown that the inner two third of the prostate has a separate arterial blood supply from the outer third and the posterior lobe. The posterior lobe undergoes atrophy with advancing age when arteriosclerosis appears and it was proposed that the inner

two third undergoes a compensatory hypertrophy. However, Moore[6] found no histological correlation between the degree of arteriosclerosis and prostatic hypertrophy and noted that it occured even without associated arterial thickening and hypertension.

Inflammatory theory

The theory states that prostatic secretion retention may follow bacterial, mechanical or chemical inflammation of the prostate and that if it is chronic, diffuse and nodular enlargement may result. The presence of lymphocytes in the pathological specimen has been suggested as evidence of inflammatory reaction. However, it has been shown that the lymphocytes are actually lymphoid tissue identical with lymphoid follicles in other organs and not an evidence of inflammation.[6]

Metabolic and nutritional factors

Disturbances of fat metabolism have been suggested to play a role in prostatic hyperlasia. Hypercholestremia and increased beta to alpha lipoprotein ratio has been observed in patients with prostatic hypertrophy. Low protein diet and/or lack of vitamins A, B, C and E have been suggested as a cause for lower incidence in the Asiatic and Bantu. Prolonged undernutrition has thus been suggested to prevent maturation of the testis, reduce circulating androgens and cause atrophy of the prostate.[10]

Neoplastic theory

This theory postulates that the enlargement is a benign neoplasm.[1] This was based on the similarity between the prostatic and uterine leiomyomatous nodules and it was assumed that the prostate and uterus both developed embryologically from the Mullerian duct. However, Moore[6] pointed out that the origin was not homologous and that in fact the prostate arose from the periurethral tissue which is a derivative of the Wolffian duct. He further pointed out that there was no criteria to distinguish whether the process was a benign neoplasia or a focal hyperplasia.

Pathology

In a young man the detrusor fibres continue down through the entire gland. As the gland enlarges with age, at about 40 years the diffuse fibres begin to rearrange to form a septum dividing the gland into an outer and inner zones. The inner zone is principally responsive to estrogens, consists of submucosal and urethral glands and has a tendency to adenomatuos hyperplasia while the outer zone consists of prostatic gland proper and has a tendency to malignancy.

Microscopically five types of hyperplasia are seen and these are; stromal, muscular, fibromuscular, fibroadenomatous and adenomatous hyperplasia. Other variable combinations of chronic inflammatory infiltrates, infarcts, cysts, stones or carcinoma insitu are often seen. Kambal[3] reported 6.5% carcinoma insitu in his series.

The gland may be large and soft or relatively small and firm depending on the proportion of glandular or fibrous tissue.

The hyperplasia can involve part or whole gland producing various forms of hyperplasia including; lateral lobe, median lobe, trilobular median bar hyperplasia, and subcervical median lobe hyperplasia.

The enlargement causes the urethra to become a slit above the verumontanum. The middle lobe enlargment acts as a valve preventing complete emptying or stretch and exaggerate the posterior curve and cause the sphincter to be incompetent. In response to out flow obstruction the detrusor muscle usually undergoes hypertrophy to form trabeculations. In the gaps between the trabeculations the epithelium herniates to form sacculations and diverticuli which do not empty out completely during micturition. In others the bladder shows very little hypertrophy and what is even more common is detrusor hypertrophy succeeded by weakness and thinning of the bladder with development of enormous residual urine.[11] As residual urine increases infection occurs and leads to calculi formation. The ureter in its intramural section is compressed by the hypertrophied muscle and allows reflux. Both these lead to hydroureter and hydronephrosis. Ascending infection from the bladder soon follows up

to the kidney. Renal tissue destruction and impairment of the concentrating power of the tubules lead to obligatory diuresis, salt wasting and dehydration. Infection accelerates impairment of renal function with uraemia.

Compression of the prostatic veinous plexus results in congestion of the veins of the base of the bladder neck which may rupture and bleed.

Clinical features

Frequency is the earliest symptom particularly nocturia. The deranged anatomy in the region of the internal meatus may cause introvesion of the sensitive mucosa of the prostatic urethra into the bladder or may allow urine into the prostatic urethra setting up a desire to micturate. This is particularly worrying to the patient at night as it interferes with his sleep.[13] As the sphincter becomes incompetent escape of urine causes urgency and with increasing residual urine there may be incontinence. Later the frequency is exaggerated by infection and impaired renal function with obligatory diuresis.

The patient also has difficulty in micturition. In the early stages there is usually hesitancy before the flow starts and straining is useless. The stream is poor and intermittent and ends with a dribble. Urgency, hesitancy, poor stream and dysuria are the signs of prostatism.

Occasionally the patient may notice drops of blood but rupture of the prostatic veins or erosion of the prostate may result in massive haematuria. Urinary infection results in cystitis, epididymo-orchitis and the ascending pyelonephritis.

As the obstruction progresses, eventually the bladder may fail to overcome the obstruction and retention of urine results. This may be acute with sudden onset and severe pain or chronic, in which the bladder gradually becomes distended and the patient develops dribbling overflow incontinence, with little or no pain. It is in latter group

that uraemia is likely to occur. In some instances a complete obstruction then supervenes (acute on chronic obstruction). History of drug ingestion particularly drugs like propantheline, imipramine, ephedrine etc which can precipitate acute retention of urine in the elderly should be asked for.[12] History should also include a review of cardiovascular respiratory and neurological symptoms, associated diabetes and intercurrent diseases.

Elebute[14] reported that while frequency of micturition is usual presenting symptom of prostatic obstruction in Europeans, two thirds of African patients in Durban South Africa and three quarters of patients in Lagos, Nigeria delay attending hospital until they are forced to do so by the development of retention of urine.

In this study retention of urine was the commonest presenting complaint recorded in 20 patients (66.6%) while frequency as a presenting symptom was recorded in 15 patients (50%). Dysuria was the second commonest complaint at presentation recorded in 18 patients (60%). Each presenting symptom seen in this study and number of patients who presented with the symptom are shown in table 2 in order of frequency. It is also seen in this study that most of the patients (20) presented with retention of urine.

On examination the patient with an enlarged prostate, if uraemic, is likely to be pale and with a dry furred tongue; he may be mentally confused. Examination of the abdomen may reveal a large bladder which may reach to the umbilicus or even above. If the obstruction is acute the typical globular shape of the bladder and it is dull to percussion. On rectal examination, the size, shape, consistency of the prostate and mobility of the rectal mucosa are noted. Typically in benign enlargement the lateral lobes are enlarged and a sulcus is palpable between them in the midline posteriorly. This is in contrast to the carcinoma which usually involves the posterior part of the gland and obliterates the sulcus. The size of the prostate may appear to be larger than it really is if the bladder is grossly enlarged and pushes the prostate down towards the examination finger.

and before operation. In case of median lobe enlargement digital rectal examination will not reveal adequate information and cystoscopy will be most helpful investigation.

Examination should also be done to exclude other conditions whose symptoms can mimick enlarged prostate. These include urethral stricture, neurogenic bladder and diabetis mellitus as well as cardiovascular complications.

Investigations should include blood urea and serum creatinine estimation as an index of renal function. Serum acid and alkaline phosphatase are usually requested in order to exclude a malignant prostate lesion. Acid phosphatase is raised above 10 King-Armstrong Units in 70% of patients with malignant prostates.[14] Haemoglobin is estimated, since uraemia inhibits the bone marrow and leads to anaemia. The act of micturition should be watched and after micturition catheterization done to measure the amount of residual urine. Residual urine exceeding 100ml is considered significant.[15] Urine analysis, culture and sensitivity should be done to exclude other intravesical lesions like schistosomiasis, systemic diseases like diabetes mellitus; obtain drug sensitivity of the infecting organisms, if present, and give appropriate drug preoperatively and in the immediate postoperative period.

Intravenous urogram gives useful information regarding renal function, evidence of hydronephrosis, hydroureter, enlargment of bladder with chronic retention, or residual urine due to inability to empty the bladder completely. Intravesical enlargement of the prostate is shown by a globular filling defect at the base of the bladder and acute hooking of the terminations of the ureters due to the enlarged prostate pushing up the trigone of the bladder. Diverticuli and other intravesical lesions like calculi can also be seen and can influence the mode of surgical approach. Post micturition cystogram provides useful information on bladder emptying.

Urethro-cystoscopy helps to exclude the presence of intercurrent urethral stricture, bladder neck hypertrophy, malignancy in the urethra and bladder, bladder stones, diverticuli and presence of schistosomiasis. It also helps to ascertain the degree of prostate enlargement and confirms the diagnosis of median lobe enlargement. Such information will also help on deciding the mode of surgical approach.[16] Blood grouping and cross-match should be done and blood should be available on the day of operation. Patient's fitness for surgical procedure should also be ascertained.

In this study 10 patients (33.3%) had preoperative urinary tract infection as a complication of primary disease as shown in table 3. Preoperatively 7 patients (23.3%) had high blood urea (table 3). Bladder stones were found in 2 patients (6.6%). Other concomitant conditions found preoperatively were; inguinal hernia 4 patients (13.3%) and haemorrhoids in 1 patient (3.3%) and hypertension in 2 patients (6.6%) as shown in table 4.

Treatment

In cases of acute retention, especially where there is a history of overholding the urine, as on a long journey or after drinking alcohol, the bladder should be emptied and catheter removed. Micturition may then be re-established.[1] However, this is rarely successful where patients present with advanced prostatic disease and in such patients continuous bladder drainage through an indwelling catheter is necessary until definitive treatment is carried out. Gibbon's catheter, connected to a sterile plastic bag provides a closed drainage carries a low risk of introducing secondary infection into the bladder, should be preferrably used.[14] In chronic retention, particularly when blood urea is above 100 mg%, slow decompression should be the rule. This is obtained by joining the catheter to a bottle and arranging the end of the inlet into the bottle at such a height, 18 to 20 cm, above the pubis so that urine flows over only when the patient coughs or moves, thus raising his intra-abdominal pressure. The bottle can be lowered 2.5 cm every four hours.[1]

This allows kidneys time to accomodate the decreasing tension. Sudden release may result in suppression of urine or haemorrhage from the kidney, ureter or bladder.[1] Closed sterile drainage system should be used and prophylactic antibiotic be prescribed.

When urethral catheterization is not possible, suprapubic drainage of the bladder should be carried out using one of the following three methods; through a formal suprapubic cystostomy, through a catheter introduced with the aid of a troca and cannula and through a fine polyethlyne tube introduced with the aid of a small sized trocar and cannula and connected directly to a sterile plastic bag. The latter two methods can be employed only when the bladder is distended well above the symphysis pubis. The last method is simple, provides drainage at a rate which is adequate without being too fast and reduces considerably the risk of secondary infection of the bladder.[17] When formal suprapubic cystostomy is performed the cystostomy tube should be brought out not less than 5 cm and preferably much higher above the symphysis pubis. If the opening is lower, subsequent suprapubic prostatectomy may be difficult.

Preoperative continuous bladder drainage done in patients with chronic retention and signs of renal damage has shown tramendous improvement on renal function.[12] The few patients who present before they have any signs of retention should be operated upon as soon as possible after they have been fully assessed and investigated.[14]

Indications for operation

1. Prostatism with 'muissance' symptoms of progressive difficulty in micturition with frequency, urgency, hesitancy and poor stream. However, frequency alone is not an indication for prostatectomy. A patient with symptoms for 9 years is unlikely to get worse while a similar degree of symptoms reached in 3 years is a greater indication for surgery.

2. Acute retention which is unrelieved by passing a catheter, emptying the bladder, and immediately removing the catheter. Wilson Hey (1945)

recommended immediate prostatectomy (one stage) in such patients who are usually fit, thus avoiding the dangers of infection by instrumentation and the increased liability to venous thrombosis if catheter drainage and bed-rest are instituted. However, immediate prostatectomy is not practicable in most tropical centers.[14] Most patients reach hospital with established urinary infection, others with acidosis, water and electrolyte imbalance and anaemia which need to be treated before operation. Another reason is that facilities for investigations and blood transfusion are usually not good enough.[14]

3. Chronic retention: A residual urine of 200ml or more, raised blood urea hydroureter, or hydronephrosis demonstrated on urography or uraemic manifestations are indications for operation.

4. Haemorrhage: Occasionally may follow ruptured prostatic vein and may not stop with catheter drainage, so prostatectomy must be performed as an emergency measure.[1]

The choice of surgical approach to prostatectomy depends on, surgeon's skill, presence of other associated bladder pathology e.g. stones, diverticulum etc, and the size of the prostate.

The operations include; suprapubic transvesical prostatectomy, retropubic prostatectomy, endoscopic prostatectomy (Transurethral resection), and perineal prostatectomy.

Transvesical prostatectomy

The indications of this procedure include; presence of big prostate judged to be above 40Gm in weight, presence of vesical calculi, diverticuli and following previous cystostomy (two stage prostatectomy). However, it is not suitable for cancer either as a palliative or a curative measure.[18]

The procedure is performed under general or regional (spinal) anaesthesia. The bladder is washed out with a dilute antiseptic solution and about 250ml of the same solution is left in the bladder. The skin incision is either transverse (pfannestiel) incision 1 inch above the symphysis pubis or vertical midline between the symphysis

pubis and the umbilicus. The rectus sheath is opened vertically and recti separated. The urinary bladder is exposed extraperitoneally and longitudinal incision is made on the anterior aspect in between stay sutures. Bladder is entered, sucked and explored for any other pathology and prostatic enlargment confirmed. With a finger introduced through internal urethral meatus into the prostatic urethra the enucleation is commenced in front at the junction of the two lateral lobes. The mucosa of the anterior commisure of the prostate is split and extended down to the apex of the prostate at its junction with the membranous urethra. The lateral lobe is then freed on each side and the middle lobe usually enucleates in the same line of cleavage within the false capsule.

When the prostate has been delivered, a self retaining retractor is introduced. Tags are resected from the prostatic rim.

Haemorrhage is arrested by packing the prostatic cavity with hot roll of gauze, ligatures may be placed at 5 and 7 O'clock positions thus 30 degrees right and left, while diathermy can be used to coagulate smaller bleeders. The bladder may be drained by a 3 way foleys catheter the balloon of which is inflated in the prostatic bed. Harris (1929) recommended reforming the floor of the urethra and obliteration of the prostatic cavity by stitching the trigonal flap tissue of the posterior part of the surgical capsule to the mucosa of the urethra and he like Wilson Hey, recommended urethral catheter drainage of the bladder. However Freyer (1901) recommended suprapubic catheter drainage of the bladder after transvesical prostatectomy.[14]

Walker (1914) advocated removal of a posterior wedge at the bladder neck, "trigonotomy", to avoid subsequent contracture of the bladder neck.

Continous bladder irrigation is established to prevent intravesical clot formation. The bladder is closed in two layers and with a drain in the retropubic space placed through a separate stab incision, the abdominal wound is closed in layers.

With better control of haemorrhage, urinary infection and ranal failure, the mortality rate is much reduced below earlier recorded 25%.[18] This approach though still associated with higher morbidity and mortality, higher incidence of postoperative calculi formation, suprapubic urinary fistulae, bladder neck contructure it is the one which is feasible in most tropical centers.[14] It is easy to master. It is suitable for most cases and is the method most widely employed at Muhimbili Medical Centre. The author has used this method for all the cases in one of the three surgical firms but also had a chance of assisting in retropubic and transurethral prostatectomies. In this study 26 patients (86.6%) had transvesical prostatectomy 4 of these patients had concomitant bladder tumour and 2 had concomitant bladder stones as shown in table 4.

Retropubic prostatectomy

In this operation the bladder previously emptied is approached by a suprapubic approach through pfannestiel incision about 1 inch above the symphysis pubis. Retropubic space is entered by retracting laterally the recti muscles. The fat is pushed aside with a swab. The veins on the anterior surface of the prostate are under run with catgut stitch and the capsule of the gland is incised preferrably with a diathermy transversely to open the plane of cleavage between the capsule and the adenoma. Using a finger the prostate is dissected off and a wedge of bladder neck fibres together with the middle lobe is cut away and the whole prostate is removed. Bleeders are easily seen and ligated. A urethral catheter is passed and retained. The capsule is closed, continous bladder irrigation started and after placing a drain in the retropubic space, the abdominal wound is closed in layers.

Millin[19] published the first paper on the retropubic approach for postatectomy. The operation has the advantage in that the dissection of the gland and the control of bleeding are carried out under direct vision, the bladder is not opened and, therefore,

a suprapubic urinary fistula is avoided.[14] However, the operation is difficult to execute on obese patients and patients with deep and narrow pelves.[18] The operation must be preceded by cystoscopy and in tropical areas this approach is made more difficult because of high incidence of inflammatory reaction around the prostate glands, particularly so in many African patients, with consequent partial obstruction of the retropubic space and firm adherence of the adenoma to its capsule.[9]

Jacobs[20] in a review of 500 cases who had retropubic prostatectomies, had relatively low mortality rate of 6% associated with speedy convalescence, minimum morbidity and excellent results. He considered the operation as the "modern surgical ideal".

In this study three patients (10%) had retropubic prostatectomy done shown in table 5. Although the number is small, the author appreciated the fact that this operation gives better exposure to prostatic bed and haemostasis can easily be achieved under direct vision.

Transurethral prostatectomy

Endoscopic resection of the prostate is considered safe, less painful and requires lesser time in the hospital for the patient. It is particularly helpful to the aged debilitated subjects where there is a high surgical risk with other methods of approach, even more useful in those whose renal function is to some extent compromised and who would have been denied chance of a prostatectomy and carry a life long suprapubic cystostomy or urethral catheter.[16] This operation was initially devised for small fibrous prostate, middle lobe enlargement and small adenomatous prostates ideally up to 30mg. The procedure becomes more difficult with larger prostates.[21] However, modifications on the resectoscope has made it quite suitable for all sizes of prostates now.[12]

In transurethral resection the essential landmark distally is the verumontanum and proximally the fibres of the bladder neck. Resection is carried out by retracting the loop electrode downwards

from the bladder neck towards the prostatic urethra, the resection being confined to the part of the gland above the verumontanum which should itself be preserved from injury. First the middle lobe tissue in the midline is partially cut then the lateral lobes on each side systematically and finally the residual tissue on either side of the verumontanum and in the region of the anterior comissure is removed. This stage is followed by stopping all the bleeding, evacuating any loose fragments and draining the bladder by an indwelling catheter.[21]

In the United Kingdom over 90% of the patients receive this form of treatment. Thomson reported on a series of 26, 463 cases who underwent transurethral resection with postoperative mortality of 1.04% (29 deaths). This procedure is technically difficult and difficult to teach. When wrongly done the results can be bad.[22]

In this study only 1. patient (3.3%) had transurethral resection and had good immediate results. He had minimal bleeding and did not need blood transfusion. The patient was an elderly man with mild heart failure and unsuitable for transvesical or retropubic prostatectomy.

Perineal prostatectomy

This operation was described by Young in 1903 and was used extensively for tuberculosis and carcinoma of the prostate in those days. It is now the least popular. It is difficult operation and there is no indication for it when much easy techniques are available.[12]

Cryoprostatectomy

New approach introduced in 1966 and not yet widely practised. The prostate is subjected to freezing transurethrally using liquid nitrogen at a temperature of -160° for about 13 minutes. Hansen et al[23] in 1972 in his series had 84% good results, 10% failure rate, 6% recurrence needing reoperation. The operation is associated with a high degree of postoperative infection due to the sloughs which are expelled gradually and vasectomy is recommended in all the cases to avoid severe epididymoorchitis.[23]

Finally indwelling catheter may be used in an old patient with other associated serious disease who is unsafe for any operation.

Complications of prostatectomy

Haemorrhage: This can be primary reactionary or secondary haemorrhage. It is the commonest and most serious complication. During operation it can be controlled by hypotensive anaesthesia.[24] Vasoconstrictors have the danger of reactionary haemorrhage afterwards and are not advised. During operation, haemostasis as already discussed above, should be achieved properly and blood loss replaced. Fibrinolysis is suspected when clots which have formed disappear when the blood is left to stand and can be confirmed by plasma fibrinogen estimation. Such cases should be treated by whole blood, fibrinogen and a specific fibrinolyte inhibitor, epsilon - amino-caproic acid (EACA).[25]

Secondary haemorrhage may occur at any time upto 6 weeks and is due to infection. It is occasionally severe enough to warrant blood transfusion or reopening and evacuation of clots from the bladder.[12]

Infection

This generally results from carelessness with aseptic conditions or failure to manage preoperative urinary tract infection which majority of cases have at the time of presentation. Catheterization may add to the risk of infection. Mid-stream urine or catheter specimen should be sent for culture and sensitivity. However, many patients at the time of surgery still have infected urine despite preoperative antibiotics and this predisposes to wound infection postoperatively as well as persistent urinary tract infection particularly if clots are retained in bladder.

Bacteraemia is the most dreaded hazard of transurethral resection or transvesical prostatectomy.[12] Post operative antibiotic should be given according to the recent preoperative culture and sensitivity results to avoid this complication and with use of postoperative antibiotics, epididymitis is so rare that routine vasectomy is no longer necessary.

Deep vein thrombosis (DVT)

Calf is the commonest site where deep vein thrombosis occurs first mainly on 3rd to 6th postoperative days. Blandy[12] reports it as a common complication after open prostatectomy (transvesical or retropubic) and rate after transurethral resection. Nicolaides.[26] reported an incidence of 47.6% following retropubic prostatectomy and 6.6% after transurethral resection. However, generally the incidence of DVT is low in the African Compared to their European counterparts.

Early ambulation is advised to minimize the risk of DVT.

In this study none of the patients developed this complication.

Urinary incontinence

This may occur after transvesical, retropubic as well as transurethral resection.[27] It is as a result of injury to the bladder neck, external sphincter or a combination of the two.

In this study two patients (6.6%) complained of postoperative urinary incontinence. Both had transvesical prostatectomy done for large prostates of about 40 grams of weight.

Bladder neck contracture

This may occur following transvesical and retropubic approaches. Wedge resection from the posterior lip of bladder neck minimizes the complication.

Urethral stricture

Singh and Blandy[30] reported that urethral trauma following catheterization or instrumentation as well as after transurethral resection may heal with significant fibrosis causing urethral stricture. Urethritis occurs most often due to the inlying urethral catheter while allergy to the rubber or its components may be one cause of urethritis.[12]

Osteitis pubis

This may occur following open prostatectomy particularly retropubic prostatectomy and presents 4 to 5 weeks after operation with pain on

walking and tenderness over the symphilis pubis.[12] Radiologically it is evident as decalcification on either side of symphysis pubis. It is due to infarction or infection and necrosis of the symphysial cartilage which heals with dense sclerotic bone. The healing can be accelerated by curating the dead cartilage out. This complication was not seen in this study.

Impotence

Post prostatectomy reduced libido may occur. Caine[28] reported 46.2% in his series complaining of diminished sexual activity and this happened less in the relatively younger patients and that it was not common with transurethral resection. However, Blandy[12] reports it as a rare complication. In those who have not lost libido and if their vas deferens are intact, preservation of fertility depends upon the extent to which the bladder neck can prevent the backward ejaculation of the semen.

Incisional hernia

This may rarely occur following lower midline incision particularly when complicated by post operative wound infection making the final scar very weak.

Other rare complication include calculi formation either in intravesical or in the prostatic cavity and recurrent benign prostatic enlargement partcularly after transurethral resection.[12]

Postoperative complications encountered in this study are shown according to the operation done in table 6. Wound infection was the commonest complication following transvesical prostatectomy seen in 9 patients and 1 patient had similar complication following retropubic prostatectomy. Haemorrhage requiring reopening of the bladder or more blood transfusion was encountered in 4 patients who had transvesical prostatectomy and 1 patient who had retropubic prostatectomy. Transvesical prostatectomy was also complicated by suprabic urinary fistula (4 patients) incontinence (2 patients), Bladder neck stricture 2 patients and impotence (1 patient). These complications largerly influenced the duration of bladder drainage and hospital stay as shown in table 7.

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ELEPHANTOID SCROTUM

SUMMARY

Ten patients with elephatoid scrotum are presented. Some elephantoid changes were noted on penile skin in six of the ten patients. One patient is presented in detail. The aetiology, pathology, clinical features, investigations and treatment are discussed.

INTRODUCTION

The incidence of elephantiasis is almost exclusively in people who live or have lived for an appreciable period of time in filarial infested regions. However, despite much work which has been done on the life cycles of the filarial worms, Wuchereria bancrofti and Brugei Malayi, there is little direct evidence on the role played by filarial worms in causing elephantiasis.[1]

In Tanzania, Hasham[2] reported that Tropical elephantiasis of the lower limbs and genitals resulting from filarial infection (Wuchereria bancrofti) is endemic.

In this study most patients came from the coastal areas where filarial infection is believed to be common. 7 of the 10 patients came from Dar es Salaam region as shown in table 2. The age incidence ranged from 27 years to 75 years. 4 patients had their ages between 50 years to 60 years as shown in table 1.

CASE REPORT

I.M., I.P. No.246140, a 55 year old male peasant from Rufiji who presented at surgical outpatient clinic with history of scrotal swelling of 14 years duration.

The patient said that he noticed a small scrotal enlargement about 14 years back and he had several attacks of painful scrotal swelling accompanied by itching, fever and skin changes. The swelling gradually increased in size. He knew of many other males in Rufiji who had similar scrotal swellings and he was not, therefore, very worried of urgently going to hospital for treatment.

Four years prior to admission the swelling became so big that the whole penis became included in the scrotal mass. He could not perform sexual intercourse and could only walk with difficulty because of the weight of the scrotal mass.

He was married and had four children. The last born child was about 9 years old. However, the wife has deserted him for past 2 years because of the disease he has had.

During childhood he had suffered from poliomyelitis and developed postpolio paralysis of the left leg but was never admitted for any other major illness prior to this disease. There was no history of chronic cough or contact with tuberculous patients.

On examination, he was an elderly man with a stooping posture. He walks with a wide gait. His left leg was smaller but there was no leg edema. He was pale, the inguinal nodes were moderately enlarged and hard in consistency. The hernia orifices were normal.

The scrotal swelling was huge extending to just above the knees (Illustration 13). It measured about 66 cm perineal to symphysis pubis and 35 cm from midline posterior to midline anterior at the mid point on the right side and 62 cm by 36 cm on the left side. The median scrotal skin was hypertrophied and rugose in some areas. The pubic skin and that of the base of the scrotum posteriorly and laterally looked normal.

The penis was completely included into the mass. Only the stretched out opening near the midline which was wet indicated the site of the penis. It could be palpated within the scrotal swelling.

A diagnosis of elephantiasis of the scrotum and the penis was made.

The patient was admitted for investigations and management.

The investigations included:

1. Night blood smears for microfilaria which were all three reported as negative.
2. Urine microscopic examination revealed schistosoma haematobium.
3. Stool microscopy revealed hookworm ova.
4. Haemoglobin on admission was 9.5 gm% and ESR of 30mm/hour. Red cells were reported as hypochronic microcytic.

Illustration 13:
Preoperative anterior view of a huge elephantoid scrotum with the penis completely included into the mass.

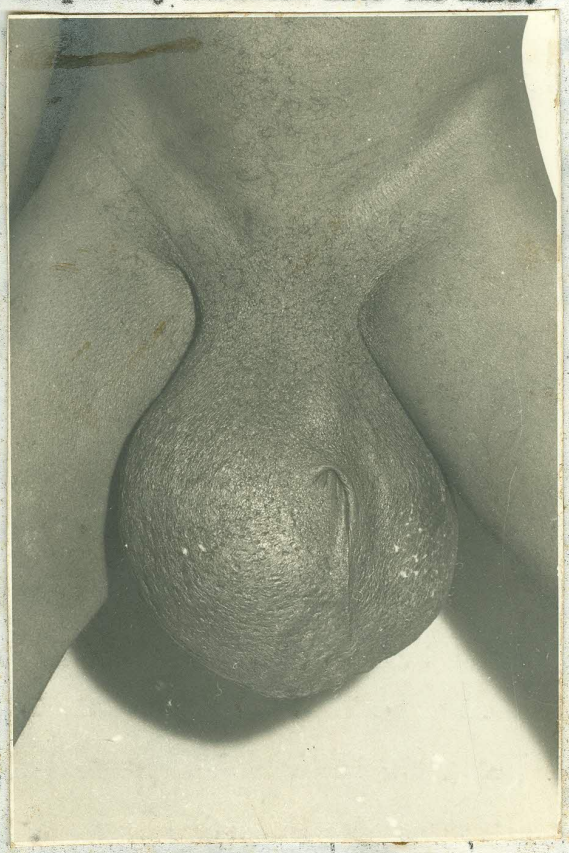


Illustration 14:
Postoperative anterior view of the same patient six months after operation.



Treatment

Initial treatment included bed rest, deworming, high protein diet and oral haematenics. After two weeks check haemoglobin was reported as 10gm%. Scrotoplasty was planed and was done as follows:

Three units of whole blood were grouped and cross matched ready for the operation. The urethral catheter was passed.

The operation was done under spinal anaesthesia with the patient in lithotomy position. Thorough cleaning with antiseptic solution was done before oblique skin incision was made along the right spermatic cord which was identified and separated. Further dissection was carried out to deliver out the right testis. The right hydrocelectomy was done by excising the tunica vaginalis and after achieving haemostasis, the testis with its spermatic cord were wrapped in a wet saline gauze and kept to safety over the lower abdomen. The same procedure was carried out on the left side.

The penis was checked and preputial skin was normal. However, some of the penile skin was elephantoid.

Radical excision was made to remove all the elephantoid skin of the scrotum following the demarcation of normal and the elephantoid skin and going deeply through all the layers. Posteriorly the dissection was extended to the normal perineal skin but all the time watching on the urethra as guided by the urethral catheter in place. The elephantoid skin of the penis was similarly excised but the normal preputial skin was re-served to cover the penile shaft.

After achieving haemostasis, the margins of the wound were checked for residual oedematous skin which was excised together with its subcutaneous tissue.

Skin flaps were prepared from the marginal normal skin. The testes were placed back in perineum and artificial pouch was fashioned by stitching the normal subcutaneous tissue using 2/0 chromic catgut interrupted sutures across the wound. The skin edges were approximated without tension using silk mattress sutures after inserting two drains coming out of each corner of the wound.

The preputial skin was used to cover the shaft of the penis and skin graft was not necessary.

The wound was dressed and scrotal support applied and patient taken back to the ward.

Postoperatively the patient was put on ampicillin 500mgs 6 hourly I/M for 7 days. Observation of vital signs was ordered. On the 4th day wound sepsis was noted and cleaning with hydrogen peroxide followed by eusol dressing was advised.

The drains and urethral catheter were removed on the 7th postoperative day. The stitches were removed on the 10th day.

The patient was discharged from ward at the end of 3rd week postoperative and advised to come back for review after three months. He was seen in the surgical outpatient clinic six months later and had healed quite well (Illustration 14).

Rest of the presentation is as follows:

Table 1: Age incidence

Age range (years)	No. of patients	Percentages
<30	1	10%
30 - 40	1	10%
40 - 50	2	20%
50 - 60	4	40%
>60	2	20%
Total	10	100%

Table 2: Place of residence

Place of residence	No. of patients	Percentages
Dar es Salaam:		
(a) Manzese	3	30%
(b) Magomeni	2	20%
(c) Kariakoo	2	20%
Rufiji	1	10%
Ruvu	1	10%
Kagera	1	10%
Total	10	100%

Table 3: Presenting symptoms and their duration

Symptom	Duration	No. of patients	Percentages
1. Bilateral scrotal enlargement	< 5 years	3	30%
	5 - 10 yrs.	3	30%
	10 - 15 yrs.	2	20%
	>15 years	2	20%
2. Unable to perform sex	>2 years	5	50%
3. Completely buried penis	>2 years	5	50%

Table 4: Average size of elephantoid scrotum at the time of admission

N.B. Measurements were done from perineum (P) to symphysis papis (SP) and the findings were as follows:

"P-SP" Measurement (cm)		No. of patients	Percentages
25 to 45 cm	Right	2	20%
	Left	2	20%
45 to 65 cm	Right	5	50%
	Left	4	40%
65 to 85cm or more	Right	3	30%
	Left	4	40%

Table 5: Investigation

Night blood smears for microfilaria were as follows:

Result	No. of patients	Percentages
Positive	2	20%
Negative	8	80%
Total	10	100%

Table 6: Treatment

Surgical treatment	No. of patients	Percentages
Excision of elephantoid scrotum	10	100%
Excision of elephantoid penile skin	6	60%
Scrotal reconstraction from skin flaps	10	100%
Skin grafting of the penis	2	20%
Preputial skin used to cover the penis	4	40%
Perineal wound drainage	10	100%
Medical treatment:		
Preoperative hetrazan given	3	30%
Postoperative hetrazan given	2	20%
Prophylactic antibiotics given	10	100%

In general good results were achieved. Complications encountered in this study are discussed below:

Table 7: Postoperative complications encountered

Complications	No. of patients	Percentages
Haematoma collection	4	40%
Wound infection	6	60%

DISCUSSION

Aetiology

Filariasis: Filariasis and elephantiasis are traditionally associated but it is difficult to obtain evidence that they are cause and effect.[3, 4] It is known that the microfilariae of Wuchelaria bancrofti can cause lymphatic obstruction. The nematodes reach the lymphatics of the skin from a bite from an infected mosquito which is an intermediate host. From the skin lymphatics, the worm will penetrate to the deep lymphatics and thence to the lymph nodes which constitute its natural habitat. The lymphatic channels become blocked and there is proliferation of the endothelium of the lymph vessels. Around dead worms there develops a granulomatous reaction with eosinophils, lymphocytes and giant cells. Calcification may also be a feature.

At the onset, lymphatic obstruction may be intermittent and following repeated attacks of lymphangitis, the scrotal skin becomes thicker owing to the chronic edema, progressive fibrosis, loss of

elasticity and contractility of the tissues. The intervals between these attacks may vary from a few weeks to several months or years.[5]

Secondary bacterial infection

Secondary infection of tissues already oedematous from obstruction of the flow of lymph through the lymph gland with streptococci or other pyogenic organisms has been suggested as cause of repeated attacks of lymphangitis.[5] This view has been supported by finding of such organisms in edematous tissues in some of the patients and that suppuration in elephantoid tissues is also not uncommon. Another explanation is that the fever with chills and rigors is an allergic reaction to the antibodies from dead filarial worms and that the bodies of dead microfilariae which must perish within the patient's body in myriads could also give rise to antibodies causing allergic reactions. However, Bowesman[1] suggests that it is the presence of degenerating filarial worms in the vascular supply of the glands which permits a leakage of blood serum from the blood vascular system into the lymphatic channels. The serum escaping is of a much higher cosmetic pressure and density than the normal lymph, it therefore descends passing down the lymphatics to the base of the lymphatic field that is in the reverse direction to the normal flow of lymph. The serum in the lymphatic field causes allergic lymphangitis and also exerts an osmotic effect which ultimately produces gross lymphoedema and elephantiasis.

Schistosomiasis

During the migratory phase of this disease there may be massive deposition of the flukes of schistosoma haematobium or mansoni in the tissues of the scrotal skin. This may produce chronic edema and fibrosis leading to elephantiasis.[6]

Other infections associated with scrotal elephantiasis include; lymphogranuloma inguinale, inguinal tuberculous adenitis and actinomycosis.[6]

Neoplasm

Tumour (secondary or primary) involving pelvic or inguinal lymphatics may cause obstruction and lymphoedema. This may be aggravated by radical inguinal gland dissection and by postoperative radiotherapy.[6]

Congenital factors

Occasionally congenital form of elephantiasis is seen. Such primary lymphoedema is caused by a congenital abnormality of the lymph vascular system.[7] Allen[8] subdivided primary lymphedema into congenital lymphedema, appearing from birth to puberty and lymphedema precox, appearing betwen puberty and 35 years of age. Kinmonth et al[9] described a third group, lymphoedema tarda, presenting for the first time after 35 years of age and by lymphangiographic studies showed that the abnormality of the lymphatics in primary lymphoedema may be aplasia, hypoplasia or hyperplasia.

If the condition is hereditary and congenital it is called Milroy's disease.[10] The disease is inherited as an autosomal dominant of variable penetrance.

Pathology

Obstruction to the flow of lymph through a lymphatic gland dams back the lymph in the lymphatic vessels at their entry into the lymphatic gland. Although this lymph pressure would not be transmitted directly back a long the lymphatic vassel because of its valves, the obstruction to the flow of the lymph would raise the pressure of the lymph sufficiently to close the valves at the distal ends of each section of the lymphatic vessel, starting at the section nearest the lymphatic gland and going on to the most distal segment.[11]

Extracellular fluid (ECF) entering the most distal segment of the lymphatic vessel would now have to accumulate sufficiently to open the valves at the proximal ends of each segment against the pressure closing the valve from the lymph already held back in each section. The back pressure of the lymph would be still greater when dilatation of the

lymphatic vessels made the valves incompetent. The hindrance from this to the passage of lymph along the lymphatic vessel would eventually hinder the entrance of extra cellular, fluid into the distal section of the lymphatic vessel. This hindrance would be greatest at the distal end of the lymphatic taking the longest course to the lymphatic gland especially when this distal end was also at the most dependent point. The accumulation of extracellular fluid would be the oedema formation which heralds elephantiasis."

When edema starts in the subcutaneous tissues the edema fluid will compress the lymphatic vessels and obliteration of the distal segments of the lymphatic vessels will prevent the extracellular fluid from entering these lymphatic vessels. This perpetuates the oedema. The skin overlying the subcutaneous tissues stretches when these tissues become filled with oedematous fluid and the resistance of the skin to stretching will drive the extracellular fluid from the oedematous subcutaneous tissues into adjacent subcutaneous tissues. The oedema fluid thus squeezed into the adjacent tissues will in its turn compress the lymphatics of this area and extracellular fluid will be prevented from entering the most distal segments of these compressed lymphatic vessels. The accumulation of extracellular fluid from this compression increases the edema at these adjacent sites and spread of edema continues. The retention of fluid with a high protein content in the interstitial spaces stimulates the production of new connective tissue and fibrosis. The edema is of a solid and non-pitting character. The march of the events is: subcutaneous oedema leads to subcutaneous lymphoedema; and then cutaneous oedema develops followed by cutaneous lymphoedema.

The great bulk of elephantiasis is in the subcutaneous tissue and elephantiasis is essentially a disease of subcutaneous tissue. The skin become elephantoid only where it overlies such subcutaneous tissue. The tissues deep to the deep fascia. retain their normal texture and translucency."

Elephantiasis of the scrotum begins as a subcutaneous oedema at the fundus of the scrotum which is the most dependent part and ascends towards the root.

Elephantiasis of the penis starts as edema of the distal end of the foreskin and extends from there back to the root of the penis.[11, 12] Elephantiasis starts as subcutaneous oedema around the distal ends of lymphatics taking the longest course to their regional nodes. Thus giving evidence that the obstruction must be at the level of lymphatic glands.

The distribution and incidence of elephantiasis of the lower legs, scrotum and penis is dependent on the lymph nodes draining these organs that are distinct from each other. The scrotum and part of the penis as drained by vessels to the superficial inguinal nodes. The lymph vessels of the glans penis and prepuce pass to the deep inguinal and external iliac groups of lymph nodes.[12]

The cut surface of the section shows oedematous fibrous tissue filling the scrotum from the epidermis inwards and this tissue is most massive at the fundus which gives the scrotum a pyriform shape. If there is no associated elephatiasis of the skin of the penis, the skin of the penis flares out from the root of the penis as the anterior wall of the enlarging scrotum advances forwards. The flared out skin becomes flush with the skin of the scrotum. Then the advancing anterior wall of the scrotum has opened out the skin of the whole of the penis, the skin of the prepuce which is continuous with it also opens out until finally the prepuce opens on the anterior wall of the scrotum. Further advancement of the anterior wall of the scrotum stretches and elongates the tube of preputial mucous membrane. The downward drag of the heavy scrotum pulls the preputial orifice down the anterior wall of the scrotum for several inches and this further elongates the preputial tube of mucous membrane. The skin of the anterior abdominal wall at the symphysis pubis does not move when the scrotum drags down and elongates the skin between the preputial orifice and the symphysis pubis.

The movement backward of the posterior wall of the enlarging scrotum lifts off the skin of the perineum and this skin becomes the skin of the posterior wall of the scrotum at its root. The perineal skin lifts off as far back as the base of the urogenital triangle and stops. There is no distortion or interference with the anal orifice no matter how heavy the elephantoid scrotum becomes.

The elephantiasis of the scrotum reaches its maximum size in the cases in which there are no elephantoid changes in the skin for it is when the skin retains its normal suppleness that it is able to increase in area by stretching to accomodate the increasing bulk of the elephantoid tissues immediately subjacent to it.[11] In the cases in which the skin is elephantoid, the thickened inelastic skin resists stretching and this limits the scrotal enlargement.

Histologically in the early stages there is atrophy of the epidermis, dilatation of the lymphatics in the dermis with surrounding plasma cells, lymphocytes and occasional eosinophils. The blood vessels are compressed. Interstitial edema and fibrous tissue proliferation occurs. Eventually there is excessive hyperkeratosis and acanthosis with scarring and loss of elasticity in the dermis, leading to nodular and fissured lesions. Occasionally giant cell systems may be present.

Clinical features

The patients usually present with history of scrotal enlargements of varying size and duration.[3] The patients who have hydrocele fluid in the tunica vaginalis, have an increased sensation of weight and heaviness in the scrotum. When the fluid within the hydrocele sac is under great tension it produces a dull testicular pain with irradiation along the spermatic cord toward the corresponding lumbar region. Some patients give history of repeated attacks of funiculo-epididymo-orchitis and/or acute attacks of scrotal swelling. The penis may be involved by the elephantiasis and/or buried in the elephantoid scrotum at the time of presentation, such patients are usually

unable to perform sexual intercourse.

On physical examination the bulky solid enlargement of the scrotum as well as the less common bulky enlargment of the penis with evidence of lymphoedema are found. There may be associated palpable regional lymph node enlargement.

Investigations

Night blood smears for microfilaria are frequently negative in patients with established elephantiasis. It is believed that the fibrosis around the adult worms traps the microfilaria which would otherwise enter the bloodstream.[12] The infectivity of the patients with elephantiasis therefore diminishes with time.

Biopsy of regional nodes most often shows a granulation tissue with chronic inflammatory cells and fibrosis. There may be the remains, sometimes calcified, of the adult worm. A foreign body giant cell reaction is frequently seen.[12] At times tuberculous caseation is found.

A lymphangiogram of the scrotum shows dilatation of the vessels and a free communication between the two halves of the scrotum with abrupt termination of the vessels due to obstruction at the inguinal nodes. In the late stages, lymphangiography is usually impossible because of the dense fibrosis in the tissues.

In this study, all the ten patients presented with bilateral scrotal enlargement. Seven patients (70%) had the enlargements of more than 5 years duration. The other 3 patients (30%) had the enlargement of less than 5 years duration (table 3). 5 patients (50%) had scrotal enlargements which had buried the penis for more than two years. These patients were unable to perform sexual intercourse for almost same duration of time (table 3).

By the time of admission three patients had been divorced because of the scrotal disease.

Elephantoid scrotal enlargements were of varying size in different patients and the two sides of the scrotum on the same patient, as

shown in table 4. The author noted the relationship that the more elephantoid the skin is the less the size of scrotal enlargement is and the less the elephatoid change on the skin the larger was the scrotal enlargement.

Night blood smears for microfilaria were taken for all the ten patients in this study. Two patients (20%) were reported to have night blood microfilaria positive the other 8 patients (80%) were reported negative (table 5).

Surgical treatment

The surgical treatment of elephantiasis by amputation has been widely practised from the early days of aseptic surgery. Total amputation of elephantoid scrotum has given uniformly good results and it is still the one and only operation for elephantiasis at this site.[2]

No matter how massive elephantoid scrotum can be, there is always free loose cellular tissue around the testis, spermatic cord, the body of the penis and around the tube formed by the mucous membrane of the prepuce.

Thus the external spermatic fascia, which ensheathes the testis with its spermatic cord is not fixed to the elephantoid tissue in the scrotum because of the existence of the free loose cellular tissue in between.

When incisions are made through the skin, the cord can be identified on either side and dissected clear to the testis which is then delivered with the accompanying hydrocele where it is present. Small hydroceles can be treated by inversion of the tunica vaginalis but when the vaginal sac is large and redundant, it is better resected to prevent pocket formation and extensive fibrosis.[5,13] When the amount of hydrocele fluid is expected to be greater than 500cc it is advisable to aspirate some of the fluid a few days before surgery in order to avoid circulatory shock following sudden evacuation of the fluid.[5] After hydrocelectomy the testis and its cord on each side are laid in a moist pack across the respective iliac fossae.

Resection of elephantoid scrotum must be as radical as possible removing all the elephantoid skin, fibrous and oedematous tissues.[5] A line of demarcation is always visible on the posterior and lateral aspects of the scrotum, where healthy skin is present.

The scrotal skin flaps can be made to cover the testicles. Occasionally however, the flaps left are too small to cover the testis in this case the testis can be temporarily burried in the thigh skin pocket on each side and transferred later when good scrotal skin has evolved.[5]

In elephantiasis of the penis the reflected layer of the prepuce seldom becomes edematous or fibrotic and it may be utilised to cover the penile shaft in most cases.

In occasional cases skin grafting of the penis is required. In such cases two lateral longitudinal incisions from the glans to the root of the penis are made and the whole elephantoid skin is completely removed including the prepuce leaving the glans and shaft bare. Half-thickness free graft is obtained from the lower halves of the thighs with a Humbey Knife and the graft is applied encircling the penis completely. It is sutured superiorly to the corona and inferiorly to the skin at the root of the penis. Urethral catheter is maintained for continuous bladder drainage.

Immediate postoperative orders include the following:

1. Complete bed rest
2. A liquid diet and administration of constipating agents
3. Administration of prophylactic antibiotics and urinary antiseptic

<u>Immediate postoperative complications include:</u>

1. Haematoma formation. To avoid this complication, the scrotal mass must be excised with careful haemostasis.[3] When haematoma forms, it must be evacuated promptly.

2. Infection: Measures that help to prevent this complication include: Thorough pre-operative cleaning of the scrotal skin; provision of a 15 to 20 minute one percent cetavlon (cetramide) hip bath in the theatre immediately prior to operation is satisfactory.[3] Control of urinary stream by using a Foley's or a Gibbon's self-retaining catheter and administration of constipating agents will minimize wound contamination.[1] Prophylactic antibiotics are given routinely.

On the sixth postoperative day laxatives are administered and sutures are removed after twelve days.

In this study, total excision of elephantoid scrotum was performed in all the 10 patients. Scrotal reconstruction from the remaining skin flaps was possible in all the 10 patients (table 6).

Excision of elephantoid penile skin was done on six patients (60%). In two of the patients the penile shaft was covered by partial thickness skin grafting. Preputial skin was used to cover the shaft of the penis in the other 4 patients (table 6).

Long term results

1. Sexual activity. Bowesman[1] observed that in those patients with burried penis the return to sexual activity remained poor even after two years. Erection of the penis occurred but was not maintained for long periods. In those in whom the penis was not burried in the filarial mass, improvement in libido was observed. Chordee may occur in a few cases because of breakdown of the penile skin graft and regrafting is done. Persistent priapism occurs in some patients. The priapism can be relieved with clot aspiration from the crura, sedation and anticoagulants.

Sperm count may remain normal at the end of two years.

2. Lasting satisfactory results are obtained in those cases in which a collateral lymphatic circulation is established and the progress of the disease is arrested.[5]

In this study three of the five patients who had, preoperatively, the penis completely burried in the scrotal mass and who turned up for postoperative review after 3 months reported that they were able to erect and were seeking medical advice to perform sexual intercourse. It must be stated that because of failure on the part of the patients to turn up for regular follow-up and the short time involved in the study of these patients the long term results of scrotoplasty can not be adequately assessed.

Medical treatment

Hetrazan (diethylcarbamyl-4-methyl-piperazine) produces prompt reduction of the microfilarial count, and it has possible action against the adult worms.[14] However, far-advanced manifestations resulting from extensive tissue damage may not be expected to regress completely.[14]

Hetrazan is administered orally in tablet form in doses of 0.5 to 2mg per kilo of body weight three times a day for three weeks.

It is a safe drug and can be used in the mass treatment of individuals living in an endemic area.

In this study Hetrazan was given preoperatively to 3 patients (30%) and postoperatively to 2 patients (20%) (table 6). Prophylactic antibiotics were given in all the 10 patients (100%).

Immediate postoperative complications included haematoma collection which was seen in 4 patients (40%). This occured despite the perineal wound drainage in all the 10 patients (table 6). The hematomas were evacuated as soon as they were found. Postoperative wound sepsis was noted in 6 patients (60%) table 7. However, by daily cleaning and dressing and systemic administration of antibiotics sepsis was soon controlled.

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SECTION III

ORTHOPAEDIC/TRAUMA

TRAUMATIC INTRACRANIAL HAEMORRHAGE

SUMMARY

Six patients with traumatic intracranial haemorrhage are presented. One of the patients is presented in detail. The various mechanisms of injury commonly associated with intracranial haemorrhage, basic pathology, clinical features, diagnostic investigations and management of traumatic intracranial haemorrhage are discussed.

INTRODUCTION

The incidence of traumatic intracronial haemorrhage varies from one place to another. In general, cerebral compression from intracranial haematoma occurs in some 6% of patients with head injury admitted to hospital.[1]

Haemorrhage within the skull following injury may be; extradural, subdural (acute, chronic), subarachnoid, intracerebral or intraventricular.[2] The increasing frequency of violent acceleration - deceleration injuries incurred in traffic accidents has increased prevalence of intracranial haemorrhage, particularly acute subdural haematoma, in the population of the head injured patients.[3]

In this study six patients of head injury with intracranial haemorrhage who were managed by orthopaedic and trauma department in Muhimbili Medical Centre during the period from 1st January 1982 to 30th September 1982 are presented.

The youngest patient was 11 years old the oldest was 43 years old. 5 were male patients (83.3%) and 1 was female patient (16.6%) as shown in table 1. Two patients (33.3%) sustained head injury following motor car accident, one (16.6%) fell from a height and three patients (50%) were assaulted as shown in table 2.

CASE REPORT

J.Z., I.P., No. 300258, a 11 years old male patient was admitted in the Orthopedic and trauma ward 9 of Muhimbili Medical

Centre on 17th July 1982 at 2 p.m. with hostory of having been involved in motor accident.

The patient remained unconscious from the time of accident up to admission.

On examination at the time of admission the patient would only respond to painful stimuli with non-purposeful and ineffective movement. He had deep bruises and a swelling over the temporal area on the right side of the head. He was not bleeding from ears or nose. His right pupil was dilated and sluggishly reacting to light; the left pupil was slightly constricted and was reacting to light.

The blood pressure was 110/50, pulse 96/min regular and of good volume respiration rate was 20/min. and the airway was patent.

Examination of the neck, chest, abdomen, pelvis, dorsal and lumbar spine and lower limbs were essentially normal. He had tender swelling of left elbow joint.

Examination of central nervious system revealed left sided hemiparesis, reflexes on both lower limbs were slightly exaggerated. Plantar reflex showed extensor response (Babinski's sign positive) on both feet.

Clinical diagnosis of head injury with acute subdural haemorrhage was made.

Urgent X-rays of skull and left elbow joint were taken. The skull X-ray showed linear fracture involving the right temporal bone. X-ray of the elbow showed an incomplete fracture of left ulnar.

The patient was prepared for emergency craniotomy.

Blood grouping and cross-match was done and the patient was taken to the theatre after shaving the whole head.

In theatre the patient was put under general anaesthesia, cleaned and draped. The horse-shoe shaped scalp incision was made on the right temporal area centred at a point about 2 cm above the mid-point of the zygomatic arch where the initial burr-hole was made.

Extradural haematoma was found and evacuated by suction and irrigation with normal saline after enlargement of the burr-hole to form a craniectomy. The bleeding branches of middle meningeal artery were ligated and bleeding from skull edge of the craniectomy was controlled by the bone wax. Elevation of depressed temporal bone fracture was done. A small incision was made to open up subdural space where clear fluid was found and sucked out.

The dura was closed and with rubber drain placed in the extradural area the scalp was closed in layers.

Postoperatively patient was put on triple antibiotics (crystalline penicillin, sulphadimedine and chloramphenical), for 7 days, diuretic (40mgs lasix I/M 6 hourly for 48 hours) and dexamethasone 4mgs intravenously 6 hourly for 5 days.

He made gradual improvement and was discharged from the ward on the 23rd August, 1982.

Rest of the presentation was as follows:

Table 1: Age and Sex incidence

Age range (years)	Sex	No. of patients	Percentages
< 20	Male	1	16.7%
	Female	-	
20 - 30	Male	2	33.3%
	Female	1	16.7%
≥ 30	Male	2	33.3%
	Female	-	

Table 2: Mode of injury

Mode of injury	No. of patients	Percentages
Motor car accident	2	33.3%
Falling from a height	1	16.7%
Assault	3	50%
Total	6	100%

Table 3: Preoperative examination findings in the 5 patients who were operated.

Level of consciousness	Patient				
	1	2	3	4	5
Collin's stage 1-3					
Collin's stage 3-5	+				+
Collin's stage 5-7			+	+	
Collin's stage 7-9		+			
Collin's stage 9-10					
Pupil Size:					
Right	dila-ted	dila-ted	normal	dilated	dilated
Left	dilated	constr-icted	normal	constr-icted	normal
Pupillary reaction to light:					
Right	slugg-ish	absent	normal	slugg-ish	slugg-ish
Left	slugg-ish	absent	normal	normal	normal
Hemiparesis or Hemiparalysis:					
Right	+	+			
Left		+	+	+	+
Reflexes:					
Right	incre-ased	-	incre-ased	incre-ased	incre-ased
Left	incre-ased	-	incre-ased	incre-ased	incre-ased
Babinski sign:					
Right	exten-sor	-	exten-sor	exten-sor	negative
Left	extensor	-	extensor	extensor	negative
Blood pressure Pulse rate	250/170 88/min.	110/60 76/min.	140/100 68/min.	110/50 96/min.	160/110 72/min.

The sixth patient, presented with history of headache, photophobia and neckache for about two weeks. He said that 15 days prior to admission he fell down from a height and sustained a bruise on the back of the head. This was associated with loss of consciousness for a short time. For the first 13 days after the head injury he had no major complaints except for mild headache. On the 14th day, he developed severe headache, neckache and photophobia and decided to come to hospital.

On examination the patient was conscious, responding to spoken words, cooperative and well oriented in time, place and person. The bruise on the occiput had healed. The pupils were equal and reacting to light. He was afebrile but had neck stiffness and a positive Kernig's sign.

The lumber puncture done revealed blood stained cerebrospinal fluid confirming the clinical diagnosis of subarachnoid haemorrhage.

After one week of bed rest and analgesics the meningeal irritation and headache subsided and patient was discharged and advised to come to outpatient for follow-up after two weeks which he did not do.

Table 4: Diagnosis

Diagnosis	No. of patients	Percentages
Epidural haematoma	3	50%
Subdural haematoma		
i. acute		
ii. subacute		
iii. chronic	1	16.6%
Subarachnoid haemorrhage	1	16.6%
Intracerebral haematoma		
Intraventricular haematoma		
Epidural plus subdural haematoma	1	16.6%

Plain skull X-rays showed a fracture in all 4 patients who had epidural haematoma.

Table 5: Operative findings and management of the five patients

Craniotomy	Patient				
	1	2	3	4	5
Right temporal		+	+	+	+
Left temporal	+				
Bitemporal					
Other site					

Table 5: (Cont'd)

Craniotomy	Patient				
	1	2	3	4	5
Haematoma:					
Epidural	+	+	+	+	
Subdural	+				+
Subarachnoid					
Intracerebral					
Intraventricular					
Evacuation by:					
Suction	+	+	+	+	+
Irrigation with saline	+	+	+	+	+
Haemostasis by:					
Coagulation	+	+	+		+
Ligation	+			+	
Bone wax	+	+	+	+	+
Closure of wound:					
With drain		+		+	+
Without drain	+		+		

Table 6: Postoperative treatment; included the following

Dehydration therapy	No. of patients	Percentages
Urea	-	
Mannitol	2	33.3%
Glycerol	-	
Lasix	4	66.6%
Steroid therapy:		
Dexamethasone	5	83.5%
Antibiotic therapy (Triple)		
Crystalline penicillin	5	83.5%
Sulphadimadine	5	83.5%
Chloramphenical	5	83.5%

Table 7: Results of treatment

	No. of patients	Percentages
Mortality: i. Preoperative	-	
ii. Intraoperative	-	
iii. Postoperative	2	33.3%
Recovery:	4	66.6%

DISCUSSION

Mechanism of injury

1. Epidural haemorrhage: Epidural haemorrhage may result from injuries of the anterior or posterior branches of the middle meningeal artery.[4] In such cases injuring force comes from a lateral direction and strikes the thin bone of the temporal plate inflicting a fracture which drives the dura inwards and in the case of anterior branch bleeding snaps the meningeal artery at a point where it leaves the bony canal in the pterion and crosses to gain attachment of the dura mater.

Blood from the torn vessel may pass downwards into the middle fossa, upwards over the motor cortex or outwards through the fracture to form a boggy swelling under the temporal muscle. In case of posterior branch bleeding, the clot is situated at a distance from the motor cortex and there may be a steady arterial haemorrhage from the ear when the fracture line involves the middle ear.

Epidural haemorrhage of venous origin may be produced by fractures which injure major sinuses (longitudinal, or transverse), middle meningeal veins or lacerated mastoid or occipital emissary veins.[4]

In this study three patients (50%) had epidural haematoma. The 4th patient had both epidural as well as subdural haematomas. All the four patients had skull fractures shown on plain X-rays taken on admission (table 4).

Subdural haemorrhage

A blow applied to the front or back of the head may cause rupture of the veins passing from the cerebral hemispheres to the venous sinuses as the result of displacement of the brain inside the skull and lead to subdural haemorrhage.[5] It is also frequently seen in acceleration - deceleration injuries incurred in traffic accidents.[3]

Usually the superior cerebral veins are ruptured, producing haemorrhage over, the convexity of the hemispheres. The superior cerebral veins pass from the convexity of the hemispheres and pierce

the arachnoid membrane before crossing the potential subdural space to join the inner aspect of the dura 2.5 cm or more from the lower compartment of the superior longitudinal sinus. One end of the vein is fixed to the dura the other to the mobile hemisphere. Sudden displacement may snap the vein at the level of the arachnoid allowing blood to pass downwards into the potential subdural space between the arachnoid and dura. Corresponding veins on both sides are affected in 50% of cases.[5]

The haematomas are often large, bilateral collections of up to 60ml a side or unilateral collections of 120ml being quite usual.[5]

Rarely veins from the temporal lobe to the sphenoid or petrosal sinuses are ruptured and produce clots which collect on the under-aspect of the brain.[5]

In this study two patients had subdural haemorrhage. One developed subacute form of subdural haematoma while the other had chronic subdural haematoma. Both patients sustained head injury following an assault.

One of the two patients was found to have both epidural and subdural haematomas and has already been discussed above.

Subarachnoid haemorrhage

Cerebral laceration with effusion of blood into the cerebrospinal fluid may occur during brain displacement.[4] Lacerations are common on the inner aspect of the hemisphere against the falx, on the under aspect of the frontal lobe and tip of the temporal lobe against bony ridges and less commonly at the occipital poles. Surface injuries may also be situated at a point diametrically opposite the site of impact (Contre-Coup).

In this study the one patient who sustained subarachnoid haemorrhage presented 15 days after he had fallen down from a height and hit the occiput on the concrete floor. The case has been presented in detail above.

Intracerebral haemorrhage

Post-traumatic intracerebral haemorrhage is often seen in patients with severe head trauma and may be produced by arterial bleeding from an area of surface laceration.[6] It may be found in conjunction with acute subdural haematoma especially in patients injured in high speed vehicle accidents.[6] A clot may form under the corter or as a confluence of small haemorrhages in a contused area of the brain. Intracerebral haematomas are most commonly found in the anterior third of the temporal lobe where the temporal lobe may strike the sphenoid bone and less frequently in the tips of the frontal or occipital lobes. They can also be seen in any area of the cerebrum or cerebellum.

None of the six patients in this study was found to have sustained intracerebral hematoma.

Intraventricular haemorrhage

Haemorrhage into a ventricle may occur from tearing of the choroid plexus at the time of injury or rupture of an intracerebral clot into the ventride. It is usually part of an overwhelming head injury.[2]

Intraventricular haemorrhage was not encountered in this study.

Pathology

As the hours pass, the expanding intracranial haematoma compresses the surface of the brain and interfering with local cerebral circulation, gives rise to beginning cerebral oedema, which in turn increases the overall mass effect. Severe headache, vomiting and weakness of the contralateral limbs are present at this stage and most important the patients state of consciousness begins to deteriorate.[4]

With further enlargement of the mass, the temporal lobe is displaced inward until its most medial portion, the uncus is forced medially and downward over the free edge of the tentorium to impinge on the adjacent third nerve and brainstem. This mechanism, designated

as uncal herniation or tentorial pressure cone, is the common denominator of death from any rapidly expanding supratentorial mass lesion and it is fatal unless the brainstem compression can be relieved quickly.[4]

As the medial temporal lobe herniates over the tentorial edge, it displaces the occulomotor nerve, paralyzing the pupillo-constrictor fibers. The ipsilateral pupil becomes widely dilated and nonreactive to light. Further displacement of the uncus brings it into contact with blood vessels supplying the midbrain, impairing its circulation and, finally, the midbrain itself is compressed and then displaced against the opposite third nerve and the unyielding contralateral tentorial edge. As these events occur, the hemiparesis becomes a hemiplegia, coma progressively deepens and the patient develops decerebrate rigidity, first on the side opposite the mass, then bilaterally. The opposite pupil becomes dilated and fixed to light.

Midbrain haemorrhages secondary to compression and circulatory obstruction (duret haemorrhages) extend rostrally and caudally along the axis of the brainstem destroying centers concerned with cardio-respiratory regulation and death follows quickly.[4]

The syndromes of traumatic intracranial haemorrhage may arise without any significant brain injury and, hence, no initial loss of consciousness.[7] However, when the initial impact is sufficient to cause a concussion with brief unconsciousness, the patient revives and remains awake (lucid interval) until the compressive effects of the expanding hematoma become evident. This is the only clinical setting in which a pure Lucid interval is present.[4]

The patient with a moderately severe brain injury (contusion) may remain stupporous from its effects for hours, then begin to lighten, only to deteriorate from the later expansion of the hematoma. There is also acute brain swelling and oedema in areas of infarction around contusions producing further compression.[5] At the far end of the spectrum is the patient with extensive cerebral and/or

brainstem injuries, comatose at the outset and worsening steadly from the superimposed brain compression.[5]

Clinical features

Progressive neurologic deficit, the most common being progressive loss of consciousness, following head trauma should always be assumed to be due to intracranial haemorrhage until proved otherwise.[6]

An immediate neurological examination is important to determine the base line and careful observation in a head injury is centred on the need to detect the patients who are developing features of cerebral compression. These features include: the level of consciousness, the size and reaction of the pupils; the blood pressure, pulse and respiration; and other central nervous system signs.[2]

Level of consciousness

The level of consciousness should be charted according to the patient's response to stimuli.[2] Staging of the level of consciousness according to Collin's ten stages is as follows: 1. A patient responds to spoken words, is alert, cooperative and oriented in time, place and person. 2. The patient responds to spoken words but is confused and obeys simple commands only. 3. The patient responds to spoken word only after receiving painful stimuli of supraorbital pressure, 4. The patient does not respond to auditory stimuli before or while receiving painful stimuli but has purposeful, and effective motor response to painful supraorbital and sternal pressure. 5. The patient responds to painful supraorbital and sternal pressure with purposeful but non-effective motor movements. 6. The patient responds to painful supraorbital and sternal pressure with nonpuposeful movement. 7. The patient responds to painful stimuli only with alteration in pulse and respiratory rate or with decerebrate posturing. 8. The patient does not respond to painful stimuli but cough and gurg reflexes are present. 9. There is no response to stimuli. 10. Lowest level of unconsciousness the only step below this is death.

In this study preoperative level of consciousness of each of the five patients who were surgically treated is shown in table 3. Five patients had level of consciousness within Collin's stages 3 to 5. Another two patients had levels of consciousness within Collin's stages 5 to 7 and one had level of consciousness within Collin's stages 7 to 9.

Pupil size and reaction to light

As a result of increasing pressure upon the third cranial nerve, the phenomenon of Hutchinson's pupil passes through the following stages.

1. Pupil on the side on which compression commenced is slightly contracted but reacts to light. The opposite pupil is normal.
2. Pupil on the side on which compression commenced is moderately dilated and reacts to light sluggishly. The opposite pupil is normal.
3. The pupil on the side on which compression commenced is widely dilated and does not react to light. The opposite side pupil is moderately dilated and reacts to light.
4. The pupil on the side on which compression commenced is widely dilated, insensitive and occasionally ptosis is present. The opposite side pupil is also widely dilated and insensitive. This may indicate severe midbrain damage on implication of midbrain cone.

Dilatation of both pupils which are sluggishly reacting to light may indicate cerebral oedema while changing pupillary sizes indicate disturbance at the third nerve nuclei.

In this study pupil preoparative examination findings are shown in table 3. The first patient had bilateral pupil dilatation with sluggish reaction to light. Craniotomy (table 5) revealed left epidural and subdural hematomas. The second patient had right dilated and fixed pupil. The left pupil was constricted and fixed. Craniotomy revealed right epidural hematoma (table 5). The third patient had bilateral normal pupils which reacted well to light.

Craniotomy revealed right epidural haemorrhage. The fourth patient had dilated right pupil which showed sluggish reaction to light and craniotomy revealed right epidural hematoma. The fifth patient had dilated right pupil which was reacting well to light. He had right subdural hematoma. Thus the pupillary signs were unreliable indicators of the site of the hematoma in some cases like the first and third patients in this study.

Blood pressure, pulse and respiration

With increasing cerebral compression the blood pressure rises, pulse slows and respiration may become stertorous and eventually Cheyne Stokes in nature (Cushing phenomenon).[9] However, this phenomenon is not only unreliable physiologic response but may be absent in some cases.[10]

In this study preoperative examination findings of blood pressure and pulse rate are shown in table 3. The significant rise in blood pressure was noted in three patients. Two patients had significantly increased pulse rate. In the other patients, both blood pressure and pulse rate remained within normal range as shown in table 3.

Other Central Nervous System Signs include: hemiparesis (or hemiparalysis), focal epilepsy or fits, aphasia, Babinski's sign and decerebrate rigidity.

Hemiparesis

Progressive paresis (or paralysis) of the lower half of the face spreading to the arm then to the lower limb may occur first on the opposite side of the lesion. If the pressure is not relieved, displacement of the brain stem at the tentorial opening forces the opposite crus against the rim of the tentorium, producing further hemiplegia on the side of the haemorrhage.[5] Similarly sensory loss appears first on the opposite side of the lesion.

In comparatively early cases the arm is always more affected than the leg.[8] Corroborating signs such as absent abdominal reflexes,

increased triceps jerks, and a positive Babinski's sign help to determine the presence of hemiplegia.

In this study preoperative examination findings on hemiparesis, reflexes and Babinski sign are shown in table 3. Right hemiplegia was found in one patient who had left epidural and subdural hematomas (table 5). The second patient had tetraparesis and was found to have right epidural hematoma (table 5). The other three patients who had left hemiparesis were found to have right sided intracranial hematomas.

Focal epilepsy

Progressive unilateral Jacksonian epilepsy is a useful localizing sign and usually appears first on the opposite side of the lesion.[2]

Non of the patients in this study developed epilepsy.

Aphasia

Difficulty in speech may be the first lateralizing sign in a conscious patient who is right handed and has left - sided lesion.[8] In right-handed people Broca's area usually is left-sided.[2]

However, in this study aphasia was not a reliable sign because all the patients except one were in the range of level of consciousness of Collin's stage 3 - 9 as recorded on preoperative examinations (table 3).

Babinski's sign

Is significant and most frequently present in cases of intracranial haemorrhage. It usually denotes a haematoma on the opposite side of the brain.[8]

In this study extensor response of Babinski's sign was bilateral in three patients, negative in two as shown in (table 3).

Decerebrate rigidity

The patient with decerebrate rigidity has all antigravity muscles facilitated. The extremities are extended and internally rotated. The neck is extended and the back is arched. The decerebrate rigidity may first develop on the side opposite the mass, then

bilaterally. It is a severe form of midbrain compression.[4]

In this study decerebrate rigidity was not seen in any one of the patients.

Extradural haematoma

In about three fourths of cases, the site of the haematoma is in the middle fossa, but it can be el-sewhere and the rule is that such a haematoma will be found in relation to the fracture line and an area of scalp bruising.[1]

When the haemorrhage is of arterial origin, the signs of cerebral compression may develop within a few hours of injury. There may also be a superficial swelling forming under the temporal muscle. In case of bleeding from posterior branch of the middle meningeal artery, the clot is situated at a distance from the motor cortex, the surface bruising is situated farther back, or there may be a steady arterial haemorrhage from the ear when the fracture line involves the middle ear. The level of consciousness deteriorates but there may be no motor paralysis for several days or the paralysis appears on the same side of the body as the haemorrhage as the result of brain shift and impaction of the contralateral crus.[5]

When the haemorrhage is of venous origin produced by fractures which injure the major simuses like superior longitudinal sinus, a massive subgaleal haematoma forms causing deterioration of consciousness with generalized epilepsy if the clot lies over the frontal areas or unilateral or bilateral leg weakness with extensor plantar responses if the clot is over the upper and of one or both motor cortices.[5] Haemorrhage, from transverse sinus occurs into the posterior fossa and deterioration of consciousness may occur at an interval of days after the injury. Signs of cerebellar compression may occur and these include reduction of tone and reduction of reflexes on the side of compression.

Subdural haemorrhage

An acute subdural haematoma is usually associated with underlying cerebral contusion and/or laceration.[1] Patients with this

injury are therefore commonly unconscious from the outset. In these circumstances evidence of cerebral compression has to be sought from detailed observation and examination for further deterioration. In others, however, there may be a more obvious lucid interval and here the subdural haematoma may have been produced by rupture of the veins passing from the cerebral hemispheres to the venous sinuses as the result of displacement of the brain inside the skull without associated severe cerebral injury.

Usually the superior cerebral veins are ruptured, producing haemorrhage over the convexity of the hemispheres. Normal pressure in these veins is so low that venous bleeding by itself could never compress the brain and overcome arterial pressure. However, vomiting after injury may force blood out at a high pressure and account for the acute onset of pressure occurring in a period of hours.[5]

In the subacute or chronic cases, the pressure increases as a result of the osmotic traction of the blood in the subdural space drawing fluid from the cerebrospinal fluid system through the semipermeable arachnoid so that the cyst increases steadily in size and produces compression symptoms in days or months after the injury. The symptoms include; headache, mental apathy later merging into stupor. When this phase passes on to come as the result of the formation of a mid-brain pressure cone, the operative mortality rises from nil to 30%.

Subarachnoid haemorrhage

Blood in the cerebrospinal fluid is an irritant and produces sterile meningitis.[8] The patient complains of intense headache frequently associated with vomiting and photophobia. Meningism with a neck stiffness and positive Kernig's sign may be a feature.

However, in doubtful cases, X-rays of the cervical spine must be obtained to rule out fracture-dislocation of a cervical vertebra before testing for neck rigidity.[8]

Some patients may present with manical behaviour weeks or months after head injury. Subarachnoid haemorrhage may also cause blockage of the basal cisterns leading to stasis in and distension of the lateral ventricles, which nevertheless remain in communication with the subarachnoid spaces.[5] The progressive communicating hydrocephalous leads to progressive dementia and motor apraxia.

Intracerebral haemorrhage

The surgically significant intracerebral hematomas present with progressive decrease in consciousness and/or progressive focal neurologic deficit. Most frequently focal deficits are third nerve palsy and hemiparesis. These symptoms may develop within a few hours or a few days.

Intracerebral haemorrhage can also be associated with extra or subdural haematoma.[8]

Investigations

Radiography: Urgent plain radiography of the skull in two planes should be undertaken in all cases of head injury.[5] Presence or absence of fracture of skull, intracranial air or shift of the calcified pineal gland should be noted. In about 90% of adults with epidural haematoma X-rays reveal a fracture line crossing the middle meningeal groove.[5] Calcification of the pineal gland or habenular commissure region is seen in about 80% of adult cases while in about 50% of cases calcified choroid plexus is seen.[11] Unilateral supratentorial expanding lesions such as subdural, epidural hemotomas or intracerebral haemorrhage may displace these structures.

Angiography

Cerebral arteriography helps to demonstrate the site and size of any intracranial clot.[2] Percutaneous common carotid angiography is the commonest type of contrast study to be used in the head-injured patient.[11] However, the contrast can be injected into small arteries

such as internal carotid, external carotid or vertebral arteries. The common carotid angiography has the advantage of filling of the middle meningeal artery but there is a disadvantage of not showing the posterior cerebral artery as often as internal carotid injection.

Most often the angiograms of the head-injured patients show epidural, subdural or intracerebral haematomas or a combination of these lesions. Occasionally however, non-traumatic lesions such as aneurysms, arteriovenous malformations and tumours are seen on the angiograms. These lesions may have been the cause of the fall that produced the head injury.

Extracerebral masses displace the surface of the brain indicated angiographically by the terminal branches of the middle cerebral artery from the inner surface of the skull and, unless bilateral, shift the pericallosal artery and internal cerebral vein from their normal midline positions toward the opposite side. Sometimes the middle meningeal artery can be seen displaced inward with dura, enabling one to distinguish the lesion from a subdural collection.

Preoperative angiography is particularly important in cases of acute subdural haematoma because they tend to be bilateral. However, in emergency situation or where facilities for cerebral angiography are lacking, the only safe procedure is rapid burr-hole exploration of the skull over the suspected site of the clot collection.

In this study skull X-rays were taken on admission on all the 6 patients. Four patients who had epidural hematomas showed fracture of skull. Non of the patients had preoperative or postoperative angiography done. Other investigations that can be done include echoencephalography, air-studies and brain scan. Non of these was done in this study.

Echoencephalography: This is helpful in the detection of midline shift.[2] However, in cases with bilateral clots there may be no shift detected.[12]

Air-studies: Ventriculograms or subdural air injections are usually done after burr-hole exploration of the cranium.[13] If the burr-holes reveal subdural or extradural blood, it is aspirated and the space formerly occupied by blood can be allowed to fill passively with air, X-rays in prone and supine projection will then show the extent of the hematoma space. If the burr-holes revealed no epidural or subdural haematomas, a needle may be passed into a lateral ventricle and the ventricular fluid replaced by gas allowing a very precise diagnosis to be made.

Occasionally lumbar encephalography may be carried out and in case of chronic subdural haematoma or subdural hygroma, the ventricular deformity is that of a laterally situated parietal mass lesion. In some cases the sulci on the surface of the hemisphere fill with gas and can be seen displaced away from the inner table of the skull.

Brain scan

External localization of intracranial lesions after the intravenous injection of radioactive compounds (isotope) is valuable in slowly developing subdural haematoma.[14] Repeated scanning may prove informative and is permissive particularly if technetium is used.[15]

Treatment

Surgical: Early removal of the hematoma before transtentorial herniation has occurred is the key to a favourable outcome.[16] In a series of 167 patients with extradural hematoma, Jamieson and Yelland[16] reported a 77% mortality among patients who were decerebrate prior to surgery whereas there was only one fatality among the 74 patients who were conscious at the time of operation. The earliest tangible physical findings; lessening of consciousness from a previously established baseline or the development of any lateralizing signs such as hyperactive reflexes, limb weakness, Babinski sign or enlarging pupil are clear cut danger signals indicating an expanding intracranial clot and call for immediate

definitive action without delaying for angiography or even plain skull films.[4]

Epidural haematoma

The initial burr-hole should be placed over the side of bruising, the side of skull fracture and the side of the initial dilatation of the pupil.[5] Otherwise a low temporal burr-hole will disclose the majority of extradural clots. The burr-hole can be enlarged with rongeurs to form a craniectomy and in most cases the entire haematoma can be removed by suction and irrigation. When the clot is solid and difficult to remove, a subtemporal craniotomy can be made by turning down an osteoplastic bone flap. This maneuver improves the exposure, facilitates haemostasis and obviates the need for future cranioplasty.[4]

Haemostasis is achieved by first occluding the proximal main trunks of the middle meningeal vessels and then the major peripheral branches to prevent back-bleeding within the exposed area of the dura. Occasionally the artery is ruptured near the foramen spinosum and the dura must be separated from the floor of the middle fossa, tracing the artery to the foramen spinosum where it can be coagulated. Oozing from the dura can be stopped by light coagulation or application of absorbable haemostatic cellulose. Bone bleeding from the edges of the craniectomy is stopped by application of bone wax. Finally fine silk sutures placed through the outer layer of the dura and the inner temporalis muscle are tied snugly to tent the dura against the edges of the bony defect to control dural oozing near the bone edge.[4] In case of haemorrhage from a sinus, bleeding is arrested by holding a muscle graft firmly against the aperture whilst it is secured by an overlying letter 'N' stitch.[5]

Once haemostasis is absolute, a small dural incision is made to exclude coexistent subdural hematoma. If opened, the dura is closed, followed by layer closure of the scalp. Where haemostasis is uncertain, drainage of the extradural space by a sialastic catheter

drain led out through a separate incision and connected to low suction for 24 to 48 hours is advisable.[4]

Postoperatively the patient should be kept lying flat in order to encourage the brain to re-expand. If the level of consciousness does not improve rapidly and the patient shows signs of persistent coning with dilated pupil a diagnostic lumbar puncture should be performed.[5] If the pressure is abnormally low, a sterile saline should be injected intrathecally in order to raise the pressure to the normal level of 120mm of cerebrospinal fluid or slightly above and so produce a free rise and fall on jugular compression. This may necessitate an injection of 90 or 100ml of saline; when successful it will occasionally relieve cone formation and result in rapid restoration of consciousness. It probably does so by floating the brain stem up into its normal position, thus straightening the vessels supplying the stem. Re-exploration with additional burr-holes on the same and opposite sides in search of second haematoma may be necessary in some cases.

The mortality from epidural haematoma remains approximately 50%, partly because of the difficult in recognizing the syndrome and delay in control of the haemorrhage.[6]

In this study three patients had epidural haematoma. The fourth patient had both epidural and subdural hematomas. In all the 4 patients the hematomas were evaluated by suction and saline irrigation through a temporal craniectomy. To achieve hemostasis, coagulation of bleeders was used in three patients ligation was applied in two patients and bone wax was applied in all the four patients as shown in table 5. Wound closure with a drain was done in two patients. In the other two patients scalp wound closure was done without a drain.

Subdural haematoma

The majority of acute subdural hematomas are accompanied by severe intrinsic brain injury and the expanding blood clot is only

part of the problem. Traumatic intracerebral hematoma and/or contusion or lacerations of the cerebrum, which may be contre-coup or bilateral, are common findings as are impact-produced brain-stem contusions.

The seriousness of this constellation of injuries is reflected in the 70% to 90% mortality reported by most investigators[17, 18] when operation is required during the first 12 to 24 hours after injury. In Jamieson and Yelland's Series[19] 89% of the patients with decerebrate rigidity died, and when fixed dilated pupils were also present the mortality rose to 95%.

In this study two patients had subdural hematoma. One of the two patients had a combination of opidural and subdural hematomas. They were evacuated by suction and saline irrigation through the temporal craniectomy as shown in table 5. One patient died postoperatively.

Approximately 20% of acute subdural hematomas are bilateral. Therefore, unless preoperative angiography has excluded this possibility, exploration of both sides is necessary. Hematoma is usually found on the side of the dilated pupil.[4] However, McKissock[20] found 9% of acute hematomas contralateral to the dilated pupil. The position of the hematoma was even less reliably predicted by a hemiparesis, 29% were ipsilateral to the hematoma. A finding probably explained by direct injury to the opposite cerebral pedicle or by compression of the pedicle contralateral to the clot against the edge of the tentorium.

None of the patients in this study had acute subdural hematoma.

Because acute hematomas are formed by solid clots, large osteoplastic craniectomy is required to remove the clot and control the source of bleeding. After the hematoma has been removed, areas of contusion are noted and intracerebral clots sought by passing a ventricular needle into the tips of the frontal and temporal lobes.

If an intracerebral hematoma is located, a cortical incision is made in a silent area and carried down into the cavity to permit aspiration of the clot and surrounding necrotic brain. Tapping of the lateral ventricles is usually necessary and helps to decompress the expanding oedematous brain. If brain swelling is still uncontrollable, exploration for hematoma on the opposite side should be carried out. Further internal mechanical decompression can be achieved by performing temporal or frontal lobectomy or by performing bifrontal craniotomy.[21] The dura must be closed with a graft if necessary.[21]

Chronic subdural haematoma

About 20% of chronic subdural hematoma are bilateral.[4] Preoperative angiography is therefore, necessary to determine the presence of a clot on the other side. They are usually liquid hematomas and are easily aspirated through two or more burr-holes or small craniectomy. If hematoma spurts out under pressure, it is important to tamponade the burr-hole with a finger and allow the blood to escape very slowly over several minutes to avoid abrupt changes in intracranial dynamics.[4]

After the haematoma has been completely removed, the edges of the highly vascular outer membrane are coagulated to prevent postoperative recurrence of the hematoma which is as high as 20%. Number 10 French red rubber drains are placed in the subdural space and left in place for 24 to 48 hours. As the dura is being closed, the subdural space is filled with Hartman's artificial cerebrospinal fluid solution and finally the scalp is sutured with non-absorbable material.

Postoperative nursing of the patient in 20 degrees of Trendelenburg position and generous hydration are two postoperative measures to enhance brain expansion.[5]

In this study one patient had chronic subdural hematoma evacuated through temporal craniectomy and haemostasis achieved by

coagulation and bone wax applied to the bone edge. The wound was closed in layers with rubber drain in the subdural space. The drain was removed after 24 hours and patient recovered well and was discharged two weeks after operation.

Non-Surgical adjuncts to management

Hyperventilation: Underventilation with relative hypercapnia and hypoxia, is a common respiratory abnormality in patients with brain injuries even though the airway may be satisfactory. The hypercapnia and hypoxia are potent stimuli to vasodilatation and enhance cerebral oedema and intracranial hypertension.[4] This can be modified favourably by hyperventilation, provided that irreversible vasomotor paralysis has not yet occurred.

Lowering arterial Pco_2 to 30 mmHg and raising Po_2 and pH by controlled alveolar hyperventilation 25 to 50% above rated minute volume with a negative pressure expiratory phase and frequent blood gas monitoring usually produce a modest lowering of intracranial pressure that can be sustained for hours.[22]

Pharmacologic agents

Two major classes of drugs effective in reducing cerebral edema and lowering the increased intracranial pressure are the osmotic diuretics and the adrenocortical steroids.

Osmotic diuretics

These are used to temporarily reverse signs of decompression from increased intracranial pressure, thus gaining time for more definitive therapy. The duration of action is brief (3 to 6 hours). However, in uncritical hands, there is a possibility of masking the early focal signs of an expanding hematoma by temporarily lowering intracranial pressure.[4]

Mannitol is given intravenously as a 20% solution over 15 to 45 minutes. The recommended dose is 1.5 gm/Kg body weight. It creates on osmotic gradient that favours the passage of water from the brain into the blood stream and eventual excretion by the kidneys.

Reduction in intracranial pressure is usually apparent within 20 to 60 minutes and lasts upto 6 hours.[4]

Urea was the first hyperosmolar agent to achieve extensive clinical use. It is given as 30% solution in dosage of about 1 gm/Kg body weight. It is now used less frequently because of its somewhat shorter duration of action (3 to 4 hours) and because of the rebound rise in pressure sometimes seen several hours after administration, when the urea concentration in brain tissue exceeds that in the extracellular space, reversing the osmotic gradient.[4]

Glycerol, a trivalent alcohol is absorbed from the gastrointestinal tract and can be given by tube or take orally when flavoured with lemon. It is suitable for more chronic use in situation similar to those in which steroids ordinarily are employed when, because of side-effects, these latter agents are contraindicated. The usual oral dose is 1 to 2 gm/Kg body weight in six divided doses.

In this study dehydration therapy was used in all the six patients (table 6). Two patients received mannitol. The other four patients received Lasix because osmotic diuretics were out of stoke at the time.

Glucocorticoids: The glucocorticoids suppress elevated intracranial pressure over longer periods and commonly are used for days, weeks or rarely months. However, the action is not rapid, and beneficial effects seldom are seen before 24 hours. The steroids act by decreasing the abnormal cerebrovascular permeability thought to be the basic defect in vasogenic brain oedema and thus retard expansion of the extracellular space and astroglial swelling.[23] They are more effective in preventing the development of brain edema than correcting it once it has occurred and it is advisable to administer a large loading dose to seriously injured person when they are first seen unless compelling contraindications such as active peptic ulcer, herpetic Keratitis or tuberculosis are present.[4]

Initial dose 10mg dexamethasone or 40 to 80mg methylprednisolone are given intravenous to adults. The usual maintainance dose of dexamethasone 4mg (0.5mg/Kg) is given every 6 hours thereafter. Duration of therapy is guided by the response of the patient to the over-all program of management.[4]

In this study steroid therapy (dexamethasone) was given to five patients as shown in table 6.

All the five patients received postoperative prophylactic triple antibiotics crystalin peniccillin, sulphadimadine and chloramphenical as shown in table 6.

Results of treatment are shown in table 7. There were two postoperative deaths and four patients recovered after treatment (66.6%).

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FRACTURES OF THE FEMORAL SHAFT

SUMMARY

Ten patients with fracture of shaft of femur are presented. One of the patients is presented in detail. Mechanism of injury commonly associated with fracture of shaft of femur, clinical features, investigations and treatment and complications are discussed.

INTRODUCTION

Fractures of the femoral shaft occur at any age, from severe violence.[1] When major trauma is involved, associated injuries of head, chest and abdomen are common and should be the first consideration.[1]

In this study of 10 patients with fractures of femoral shaft, 7 patients (70%) were involved in motor traffic accident, 2 patients (20%) fell from a height and 1 patient (10%) was assalted (table 3). Age incidence and sex incidence are shown in tables 1 and 2 respectively.

CASE REPORT

S.S., I.P. No.290713, a 47 year old female was admitted in trauma ward 2 of Muhimbili Medical Centre one hour after she fell down and sustained injury to the left thigh. She complained of painful and swollen left thigh. There was no history of loss of consciousness. She gave history that 15 years ago she was involved in motor accident and sustained injury to the same thigh for which she was operated.

On examination her general condition was good. She was fully conscious and cooperative. Her blood pressure was 110/70; pulse 80/min regular and of good volume. Examination of the patients head, neck, chest and abdomen showed no abnormalities. Her left thigh was swollen and there was an old scar on the lateral aspect of the left thigh. There was tenderness on palpation and movement of the left thigh. The circulation and neurological examination of the extremities were within normal limits. Clinical diagnosis of simple fracture of the lower third of left femur was made.

X-ray of the left femur showed complete transverse fracture at the lower end of the plate which was used to fix the mid-shaft fracture of the left femur (Illustration 15). Peripheral blood picture, urine and stool examinations showed no abnormality. The patient was kept on skin traction and 3 units of blood were grouped and cross-matched ready for operative treatment of the fracture.

On 15th July 1982 the patient was taken to theatre for operation. After giving the patient general anaesthesia, cleaning of the left thigh and draping were done. A lateral incision was made on the thigh after excising the previous scar. The fracture and the plate were exposed. An attempt to remove the entire plate was made but the proximal two screws could not be removed and the plate was cut leaving a small piece behind.

The fracture ends were cleared and the fragments accurately reduced. The AO blade plate was used to fix the fracture (Illustration 16). The area was irrigated with normal saline. Streptomycin was locally applied and the wound was closed in layers.

Post-operatively the patient was put on ampicillin 500mgs 6 hourly intramuscular for 5 days and streptomycin 1 gram daily intramuscular for 5 days. The wound healed well and on 8th postoperative day, stitches were removed and patient mobilized on crutches. On the 10th postoperative day she was discharged from the ward on crutches and advised to attend Orthopaedic and Trauma clinic for review after one month.

Rest of the presentation is as follows:

Table 1: Age incidence

Age range in years	No. of patients	Percentages
20 years or below	1	10%
20 - 30	4	40%
30 - 40	3	30%
40 - 50	2	20%
50 years or above		
Total	10	100



Illustration 15:
Shows X-ray of the left femur with a complete transverse fracture at the lower end of the plate which was used to fix the mid-shaft fracture of the left femur 15 years ago.

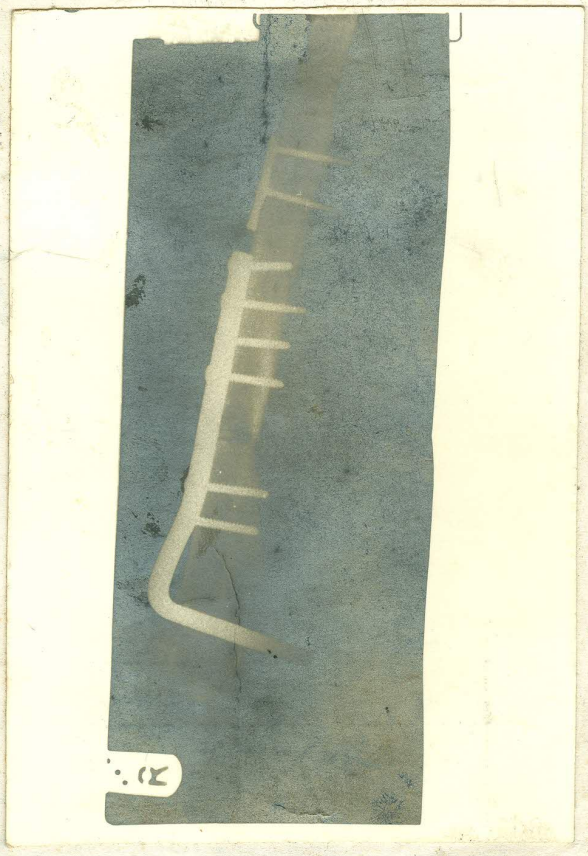


Illustration 16:
Shows the A.O. blade plate used to fix the fracture shown in illustration 15. A small piece broken from the previous plate is also seen.

Table 2: Sex incidence

Sex	No. of patients	Percentages
Male	8	80%
Female	2	20%

Table 3: Mode of injury

Mode of injury	No. of patients	Percentages
Motor accident	7	70%
Falling from a height	2	20%
Assalt	1	10%

Table 4: Site of fracture

Site of fracture	No. of patients	Percentages
Right femur:		
1. Proximal third	1	10%
2. Middle third	4	40%
3. Distal third	2	20%
Left femur:		
1. Proximal third	2	20%
2. Middle third		
3. Distal third	1	10%

Table 5: Type of fracture

Type of fracture	No. of patients	Percentages
Simple:		
1. Transverse	4	40%
2. Oblique	1	10%
3. Comminuted	2	20%
Compound:		
1. Transverse	2	20%
2. Oblique		
3. Commimuted	1	10%

Table 6: Associated injuries

Associated injury	No. of patients	Percentages
Head injury	2	20%
Fracture of rib	1	10%
Fracture of clavicle	1	10%
Fracture of humerus	1	10%
Dislocation of hip joint	1	10%
Fracture of tibia and fibula	1	10%

Table 7: Treatment of fracture of shaft of femur

Treatment	No. of patients	Percentages
Conservative:		
1. Manipulation + skin traction	2	20%
2. Manipulation + skeletal traction	3	30%
Operative:		
1. Intramedullary nailing (Kuntscher nail)	3	30%
2. Plating (AO blade plate)	2	20%

Table 8: Complications encountered

Complication	No. of patients	Percentages
Malunion	1	10%
Delayed union	1	10%
Non-union		
Infection	1	10%

DISCUSSION

Fractures of femoral shaft are usually the result of severe violence such as in automobile accidents, fall from a height, but may also occur from less severe indirect torsional stress applied to the leg.[2]

Anatomic considerations

The femur is the largest bone in the body and its shaft is composed of dense cortical bone with sufficient strength to withstand stresses and fractures of femoral shaft usually result from major violence.[2]

The muscles and fascial layers enveloping the femur serve as tension braces which, during normal activity, reduce the bending stresses on the femoral shaft. These layers also tend to obscure the extensive soft tissue injuries that are associated with the fracture of femur.

After fracture of shaft of femur the massive muscles of the hip and thigh tend to cause angulation and displacement of the fracture fragments.[2] When the fracture is in the proximal third of the shaft, the proximal fragment tends to abduct, flex and externally

rotate due to the action of the gluteus medius and minimus, the iliopsoas and the short rotators respectively. The distal fragment tends to displace medially due to the effect of the adductor and gracillis muscles and to move proximally under the influence of the long muscles arising from the ilium and ischial tuberosity; the hamstrings, rectus femoris, tensor fascia lata and sartorius.

In middle third and lower third fractures, the deformity is one of backward angulation of the distal fragment and shortening. Gastrocnemius muscle tends to flex the distal fragment, producing posterior angulation of the fracture site.

Femur has good blood supply. The four perforating branches of the profunda femoris artery which course around the posterior and lateral aspects of the femoral shaft are particularly vulnerable to injury. Even the femoral artery itself may be ruptured in association with fracture of the shaft. The nutrient arteries to the femoral shaft arise from one or more branches of the bone along the linea aspera. The inner two thirds of the cortex is supplied by the medullary circulation.[3]

Clinical features: The patient with fractured femoral shaft complains of severe pain which is aggravated by any attempt at motion of the affected extremity.[1] The deformity, usually anterior or lateral angulation, is obvious. There is also abnormal rotation and shortening of the affected leg with prominent swelling of the thigh. The deformity mainly depends on site of the fracture as explained above. There may be an associated wound on the thigh when the fracture is compound.

Bone crepitus may be felt as the patient is moved or has painful muscle spasms in the thigh.[2] Circulation and neurological examination of the extremities should be carefully done. If the patient has good pedal pulses with normal sensation and motor power in the foot, major arterial injury is unlikely. However, when signs of ischaemia are present, exploration of the vessels is indicated.[1]

When major trauma is involved, associated injuries of the head, chest and abdomen are common and should be the first consideration.[1] Massive haemorrhage into the soft parts of the thigh, accompanied by hypotension and falling hematocrit is not unusual. However, a patient with a fracture femur who develops shock should be suspected of having other injuries. Thus priorities must be established for the treatment of life threatening visceral or other injuries while the fractured femur is treated in traction. At the same time, other skeletal injuries such as a dislocation or fracture dislocation of the ipsilateral hip should not escape notice since their presence may be masked by the more obvious fracture shaft.[2]

In this study seven patients (70%) sustained fractures of right femoral shaft; one had fracture involving proximal third, four had fractures involving middle third and two had fractures involving distal third of right femoral shaft. Left femoral shaft was fractured in three patients (30%). Two had fractures involving proximal third and one had a fracture involving distal third of left femoral shaft as shown in table 4.

Other associated injuries encountered in this study included; head injury in two patients, fractured rib in one patient, fractured humerus in one patient, dislocated hip joint in one patient and fracture of right tibia and fibula another one patient as shown in table 6.

Investigations

1. Radiological: The diagnosis of fracture shaft of femur is usually confirmed radiologically.[4] All types of fractures occur in the femoral diaphysis; transverse, oblique, spiral and comminuted. There may be a third, "butterfly" fragment or merely a crack in one of the major fragment not visible on X-ray. Such a crack may make intramedullary fixation unstable if additional fixation is not used or if the medullary canal is not reamed so that a large snug-fitting rod can be inserted.

A severely comminuted or shattered fracture usually signifies an injury associated with high energy absorption and hence extensive damage to all tissues of the thigh. This damage may impair wound healing and lessen resistance to infection after open reduction and internal fixation.[2]

2. General investigations include peripheral blood picture, urine and stool examinations.

In this study there were seven simple fractures of femoral shaft and three compound fractures. The fractures were all confirmed radiologically. There were four simple transverse fractures, one simple oblique, two simple comminuted fractures, two compound transverse fractures and one compound comminuted fracture as shown in table 5.

Treatment

Treatment of fracture of shaft of femur is aimed at achieving reduction (restoration of alignment and of length), immobilization of the fracture and prevention of knee stiffness.[4] The treatment can be either conservative or operative.

Conservative treatment

This involves manipulative, closed reduction under anaesthesia and maintainance of the correction with skin or skeletal traction usually using a Thomas Splint.[5] The reduction should preferably be done within a few hours of injury under general anaesthesia with relaxant. At the same time the surgeon should apply skin traction or insert a skeletal-traction pin. This is done with the assistant holding the foot and pulling strongly and steadly upon the limb so that it can be moved and raised without injury to the soft tissues from sharp fragments of bone.

In general, skin traction is applied if the patients skin and subcutaneous tissues are healthy and not more than 10 lbs (4.5kg.) of pull should be applied to achieve the required results. If traction for a prolonged period is anticipated, especially if heavy

weights will be needed, skeletal traction is required. A threaded Kirshner wire or Steinmann pin can be inserted through the tibial tubercle, supracondylar region of the femur or the OsCalcis. The tibial tubercle is the safest and most satisfactory site becuase the bone is dense and there are few soft tissues to be traversed.[2]

When overriding of the fragments has been corrected by traction and the fragments have been replaced in apposition by direct pressure of the surgeon's hands, the ring of the Thomas splint is guided over the limb which is supported on strips of bandage slung between the side bars of the splints. The slings should be padded with a length of cotton wool. The knee joint should be in a position of slight flexion in order to avoid stretching of the posterior ligaments and causing genu recurvatum. Local splintage can be applied with gutter splints, plaster slabs or by pressure pads bandaged to the opposite side bar of the Thomas splint.

Methods of traction

A distinction should be made between fixed traction and balanced or sliding traction. In adults the counter-thrust of the Thomas splint in the region of the ischium must be reduced by fixing the lower end of the splint to the foot of the bed which is raised or better still by attaching a weight to the end of the splint (combined fixed and balanced traction). In balanced traction the cord attached to the skin or skeletal pin is passed directly over a pulley at the foot of the bed to the weight of about 10 lbs (4.5kg.) suspended from it and the foot of the bed elevated to give counter-traction. The balanced traction offers the advantage of allowing earlier movement of the knee joint.

Other methods of suspension traction

1. Fisk traction:[6] This makes use of the proximal half of Thomas splint with a Pearson flexed-knee attachment. The lower half of the bars of the Thomas splint are cut off and from the sawn ends, just above the level of the knee, suspension cords are tied and passed

over a pulley to a 4 lbs (1.8kg.) weight near the head of the bed which the patient can seize and pull, exercising the hip and knee joints while still depending on the continous traction of about 10 lb (4.5kg.) from a skeletal pin in the upper end of the tibia.

This form of traction may be of advantage in a patient whose knee has been injured or in elderly patients whose arthritic knee joints may need early active mobilization.[5]

Russell traction[7]

In this method, no splint is used. A system of cords, pulleys and weights permits the limb to be supported in a comfortable position with continuous traction in the line of the femur.[7]

The method is not advised for the routine treatment of femoral shaft fractures because it makes no real provision at all for protection of the bone fragments against backward sagging or lateral angulation occurring. However, it may be useful in the later stages of treatment.[5]

Perkins traction

This method depends entirely upon skeletal traction over pillows to control deformity and allows immediate knee movement.[8] However, the method gives even less support for the fracture than Russell traction. With up to 30 lb (13.5kg.) of traction recommended, delayed union is usual and mal-union is seen in a number of cases.[2]

Skeletal-traction in a Bohler-Braun Splint

This method offers no control of alignment of the fragments except by the degree of traction. The weight needed to maintain proper alignment causes distraction, whereas if the weight is reduced to keep the fragments in contact, it is not enough to prevent angulation. The method is not recommended for the routine treatment of femoral shaft fractures.[5]

In this study five patients (50%) were treated conservatively. Two patients had manipulation and skin traction applied. Three patients

had manipulation done and skeletal traction applied to the steinman pin inserted through the tibial tubercle (table 7).

In all these cases, Thomas splints were used and balanced traction applied. In two of the patients plaster casts were applied to the thigh to prevent angulation of the fracture fragments.

Duration of immobilization

In adults traction must be maintained for about 12 to 16 weeks. During this time, great care must be exercised to avoid distraction particularly, in transverse midshaft fractures once alignment has been obtained, the weight should be reduced gradually as guided by periodic X-rays in order to avoid loss of position.

The duration of traction and hospitalization may be reduced by placing the patient in a plaster spica once there is clinical union and the fracture gap is bridged by early callus. However, adults often tolerate recumbency in a plaster spica poorly. A walking spica is a better alternative especially with distal third fractures. A cast boot is wone on the fractured side and a raised shoe on the normal side. Ambulation with crutches is quite feasible.[5] However, a plaster spica does not permit knee motion.

Another alternative is to decrease the amount of traction gradually until the leg is simply suspended. During this time, the patient is encouraged to move the knee and gain muscle control. Once muscle control is satisfactory and union appears sufficiently solid, the limb may be removed from the suspension apparatus. Crutch walking with minimal weight bearing on the affected leg is started when there are 40 to 60 degrees of active knee motion and the patient has good muscular control.

Full weight bearing should not be allowed until mature healing is evident usually about 6 months after fracture.

Cast brace or Hip spica

The cast brace method is based on the concept that walking on a limb with an incompletely healed long-bone fracture is a positive

factor in that it encourages more rapid and secure fracture healing and maintains knee joint function. The method is applicable to fractures below the midshaft level. It is unsuitable for fractures above the midshaft level since they tend to angulate. It is also unsuitable if wound is present on the thigh.

Initially the patient is treated with skeletal traction with a Kirschner wire through the tibial tubercle. Accurate alignment should be obtained and maintained until the fracture is clinically stable i.e., until the fracture site is nontender and rubbery and no shortening occurs when traction is removed.

With the patient under sedation, an assistant holds the traction bow to support the fracture as total-contact cast is applied to the thigh over a spandex sock. Elastic plaster of paris bandages are used for the first 2 layers, followed by rolls of conventional plaster. The plaster cyclinder must be applied snugly and molded meticulaously to provide a completely smooth inner surface and total contact. The thigh component should extend as far distally as possible and still permit knee flexion to 90 degrees when the cast brace is completed. As the thigh is compressed within the rigid conical external support, the resulting cylinder of compressed soft tissue surrounding the fractured femur then tends to maintain length and alignment of the femur.

The plaster boot is applied extending from toes to the knee and after it is set, the hinges are applied and secured with plaster bandages to the thigh and boot components. Check X-rays are obtained and if the position of the fragments is not satisfactory the thigh portion of the cast may be wedged by a single cut open wedge technique to achieve proper position.

An elastic bandage is applied to the knee and exposed soft tissue between the two components of the brace and the patient is started on gait training with crutches. The cast brace is usually worn for 10 to 12 weeks until the patient can walk with full weight

bearing without distress and has active use of the knee at the functional level.

Treatment by traction and a cast brace has some advantages. The risks of open reduction and internal fixation are avoided, there is continuing muscle and joint function, there is shortened hospitalization and convalescence compared to traction followed by immobilization in a spica cast. However, movement of the fragments has been noted as the patient walks, although the snug-fitting cast controlls the rotational movement quite well.[5]

The cast brace method of treatment was not used in any of the patients in this study. Occasionally, however, the walking hip spica has been used in the department of Orthopaedic and Trauma at Muhimbili Medical Centre.

Operative treatment

Although it has been emphasized that the routine treatment of fractures of the femoral shaft should usually be by traction in a Thomas splint, there is sometimes difficult in the managment of fractures of shaft of femur by the conservative method.[5]

The strong abductor muscles of the hip which are attached to the trochanteric region on the lateral side, and the strong abductor muscles which are inserted into the lower shaft of the femur on the medial side, account for powerful deforming influence which always tends to angulate the fragments. This is particularly common with fractures of the upper third and middle third of the shaft of femur. In fractures with slow union, even though in perfect alignment for 12 to 14 weeks, the imbalance of muscle power often causes late deformity with outward bowing.

There are fractures which cannot be reduced and in which, despite repeated manipulation, a wide gap remains between the fracture surfaces. These are certain indications of soft tissue interposition between the bone ends and such fractures require early open reduction. There are also circumstances in which it may be impossible to maintain even

a simple fracture in a reasonable position by conservative methods. Such circumstances include; severe head injury with marked muscle spasm, traumatic paraplegia requiring constant turning of the patient to avoid sores; multiple injuries necessitating neutralization of the femoral fracture, femoral shaft fractures complicated by a major vascular injury requiring arterial surgery and fractures of the mid-shaft of the femur with patellar fractures on the same side should also be treated by nailing. In fractures with a large segmental third fragment the treatment of choice is open reduction and Kuntscher nailing, sometimes with additional wiring of fragments.[5]

In most fractures of the distal third of the shaft of femur an intramedullary rod will not provide adequate fixation. Such fractures are best fixed by use of plates.[2]

Intramedullary nailing

Intramedullary nails of trefoil section were widely used by Kuntscher who was faced with war emergencies from bombed hospitals and the difficulties of nursing.[10]

Intramedullary nailing can be done by closed or open method.

Closed intramedullary nailing

This is done under X-ray control without exposing the fracture site. An almost perfect reduction is essential.[11] With the aid of an image intensifier a guide-wire is driven from the trochanter through the medulla of the proximal fragment into the distal fragment. The intramedullary cavity is reamed using a cannulated reamer and a nail is threaded down the guide-wire. Kuntscher and many of his surgical colleagues in Europe have always advocated the technique of closed intramedullary nailing.[11] The emphasized advantage for this technique is the low infection rate compared with open nailing. However, the technique has its complications which include; danger to the hands of the surgeon and his assistants from X-rays exposure, danger of failure to engage the medulla of the distal fragment, and damage of the femoral artery.

Blind intramedullary nailing is a difficult operation and should only be carried out in a fully equipped theatre.[5]

Blind intramedullary nailing was not done in any of the patients in this study. However, percutaneous multiple pin fixation incorporated in cylinder plaster of Paris with early weight bearing has been occasionally used in the department of Orthopaedic and Trauma at Muhimbili Medical Centre.[12] By using this method, loss of hematoma at the fracture site is prevented, damage to the periosteum is reduced, risk of infection is reduced and hospitalization time is also reduced.

Open intramedullary nailing

Most surgeons prefer the technique to open exposure of the fracture, so that the fragments can be seen as they are replaced with accuracy and the nail can be seen as it passes from the proximal to the distal fragment.

The fracture is usually exposed through a postero-lateral incision. The bone surfaces are cleared and if the fracture is more than a few weeks old the young callus is removed and the medulla of the fragments opened with long-handled drills or reamers.

From the site of the fracture a guide-wire is then passed up the medulla of the proximal fragment until resistance is felt against the trochanter and measurement of the length is taken. The guide-wire is then pushed still further, and a short incision is made over the point of its emergence from the trochanter. A flanged nail, already known to be correct in both width and length is driven from the trochanter over the guide-wire until the point is seen to appear at the site of fracture. The guide-wire is removed. It is important that the length of the nail finally chosen should be about 1 inch (2.5 cm) less than the measurements shown by the exploration of the guide-wire because it is far better that the point of the nail should lie about 1 inch (2.5 cm) short of the femoral articular surfaces than that it should be so long as to penetrate the joint surface.[5]

An alternative method of inserting the nail is to introduce it retrograde from the fractured site hammering it through the trochanter until it appears in the buttock where it can be exposed through a short incision.[5] If the nail is used in this way it is important for it to have extractor holes at either end just in case it becomes impacted in the first stage.[5] The fragments are then held in correct apposition and alignment while the nail is driven into the distal fragment.

In this study open intramedullary nailing was done in three patients (table 7). Two patients had fracture of upper third of shaft of femur and one had fracture involving middle third. In all the three patients fractures were exposed through the postero-lateral incision. After clearing fracture surfaces and openning up of medullary cavity by using reamers, Kuntscher nail of correct length and width was introduced by retrograde method in each case.

Although many surgeons have relied entirely on the intramedullary fixation of a nail and have allowed patients to move freely in bed or even to resume weight bearing activity within a few days of operation, it is generally better, to supplement the internal fixation of a nail with the external support of a Thomas splint for about 4 weeks. Without such external support and with entire reliance on internal fixation there may be loosening of the nail in some cases resulting in non-union. After about four weeks, active exercises at the knee joint can be practised and shortly after the weight-bearing with the support of crutches may begin.

It is quite safe to leave an asymptomatic intramedullary nail in position indefinately. However, the nail can be removed as soon as the fracture is readiographically united. Most surgeons advise removal at one year.[5]

Recent studies comparing the results of closed treatment by skeletal traction and a spica cast with those of medullary nailing indicate that better results were achieved after intramedullary nailing.[13]

The best results after nailing are obtained with non-comminuted fractures located 5 cm or more below the lesser trochanter and 15 cm or more above the femoral condyles. Stable fixation of such fracture can be achieved consistently.[2] However, because the medullary cavity of the femur widens in its lower third, Orthodox Kuntscher nailing gives inefficient fixation of fracture in the lower third of the femoral shaft. If conservative treatment cannot be used for this fracture it should be fixed by plating.

Plating

In this study plating of fractures in the lower third of shaft of femur was done in two patients. In both cases the fracture was exposed through the lateral incision. After clearing the fracture ends and achieving reduction the fracture was fixed by A.O. blade plate in each case.

Fixation of fracture of the femoral shaft by 1 or 2 plates and screws or botts has been used since the early days of internal fixation. Enthusiasm for this type of fixation has waxed and waned with each modification starting with the Lane plate and progressing to the slotted plate of Eggers and more recently to the A.O. group.

It is now well established that fixation sufficiently strong to eliminate the need for immobilization cannot be achieved with a single plate. If dual plates are used maximal strength is provided by parallel placement on the medial and lateral aspects of the shaft.[14] However, the technical problems of such placement are great, so that anterior and lateral plates at 90 degrees to each other, have been used by most proponents of double plating.[15] The disadvantages inherent in this method include, the extensive exposure required to apply the plates with consequent impairment of the periosteal circulation and increased risk of postoperative infection, the stress concentration at the ends of the plates that may result in secondary fracture at these levels unless the plates are removed, and the fact that if both plates are removed together, the risk of refracture is high. Accordingly the plates must be removed separately

with enough time in between to permit remodeling of the bone in the segment of shaft previously supported by two plates.

If postoperative sepsis occurs after plating, treatment may be difficult indeed. With an infection after medullary nailing the nail is unlikely to break, and it is feasible to wait until union slowly occurs and then treat the infection by removal of the rod, debridement, sequestrectomy and appropriate antibiotic.[16] With dual compression plates the same procedure may be followed, but the fixation is less reliable. If the plate breaks or the screws pull out before healing occurs, the situation is complicated. In this even the metal must be removed, clear the infection with patient immobilized for many months in a spica or remove the plates and insert a medullary rod despite the sepsis in the hope of getting union first and eliminating the infection at a later date.

A spiral fracture in the proximal or distal third of the shaft in which adequate reduction is not achieved by closed method and an intramedullary rod will not provide adequate fixation, use of an A.O. condylar blade plate and an anterior plate will provide stable fixation.[2]

Plating can also be used to fix an ununited fracture previously treated by closed or open methods. Although there are theoretical objections to the use of plates after an intramedullary rod has failed, because both the medullary and periosteal circulation are then damaged, use of compression plates under these circumstances is often successful.[2]

When there is inadequate fixation after insertion of an intramedullary nail, the addition of a compression plate with screws penetrating only 1 cortex gives good fixation.[17] Similarly a nail supplemented by a plate may provide adequate fixation for fractures too far proximal or distal for fixation by the plate alone.

Complications encountered in this study included malunion which required corrective osteotomy in one patient (10%) delayed union which

required prolonged immobilization was seen in one patient and infection of fracture of shaft of femur was seen in one patient who had compound fracture. The infection was successfully controlled by debridement and administration of antibiotics. None of the patients developed non-union (table 8).

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