TEN SURGICAL CONDITIONS

BY

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This is to certify that the surgical conditions presented in this book were managed by Dr. Petronilla Ngiloi during her postgraduate training in General Surgery under my direct or delegated supervision.

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ABSTRACT

This dissertation consists of ten surgical conditions divided in four parts: General Surgery, Urological surgery, orthopaedics and Traumatology and Paediatric Surgery.

Surgery being a wide subject the ten presented diseases do in no way reflect the frequency of the conditions. In each condition there is a detailed case report of one patient followed by a review of the characteriastic features of the remaining patients. A review of the relevant literature centered on areas of controversy and recent advances in the management of the condition is done and a conclusion drawn.

TABLE OF CONTENTS

ITEM **PAGES**

(i) Abstract.....iii

(ii) Table of Contents..... iv

(iii) Acknowledgement.....v

(iv) Copyright.....vi

(vi) Declaration.....vi

GENERAL SURGERY

1. Lung Cancer..... 1

2. Bronchiectasis..... 39

3. Breast Carcinoma of the Female Breast.. ... 61

4. Ruptured spleen.....114

5. Management of Pancreatic Pseudocyst 146

UROLOGY

1. Benign Prostatic Hyperplasia.....167

2. Management of Male Urethral Stricture at Muhimbili Medical Centre.....212

ORTHOPAEDICS AND TRAUMA

1. Hand injuries.....232

2. Giant cell tumor at Muhimbili Medical Centre..... 260

PAEDIATRIC SURGERY

1. Hydrocephalus..... 279

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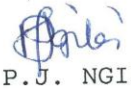
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DR. P.J. NGILOI

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PRIMARY LUNG CARCINOMA

SUMMARY

A total of 15 patients with histologically proven primary lung carcinoma were seen in the thoracic surgical unit, Muhimbili Medical Centre from July, 1988 to August 1990 a period of 26 months. Only patients with histologically proven diagnosis of lung carcinoma were included in this study.

Among the fifteen patients six were females and nine males. The ages of the patients at diagnosis ranged 20 to 65 years, with two thirds of the patients in the 41-60 year age groups.

Diagnosis was made by chest radiographs, bronchoscopy and mediastinotomy helped in the staging of the patients.

Of the 15 patients, 2 had Stage I disease, 4 Stage II, 6 Stage IIIA and 3 had stage IV disease at the time of presentation.

Seven patients had surgery while the other eight patients either refused surgery or were offered palliative treatment due to advanced disease.

Operative mortality was recorded in one out of the seven patients. Three patients died within a period of 2 years after surgery. The other four were lost to follow up.

INTRODUCTION

Primary lung carcinoma is a public enemy number one among cancers in industrialised nations.[1] It is the most common visceral malignancy in males, accounting for approximately one third of all cancer deaths in males and 1/20th of all deaths in both sexes.[1] Although females are less frequently affected, the incidence has been found to be increasing dramatically and breast cancer will soon be overtaken by lung cancer as the leading cause of female cancer death[1,2]

In the U.S the annual number of deaths from lung cancer has increased from 18,000 in 1950 to 121,000 in 1984[1]. In males this has risen from 19.9 to 70 per 100,000 population while in females the death rates has risen from 4.5 to 19 per 100,000[1]. According to cancer statistics registration in England and Wales, 1988 report shows that in 1970 one man out of every nine who died in Greater London did so from lung cancer. Recently, however, there had been a rapid rise in female mortality due to lung cancer to the extent that male to female ratio which was about 5:1 in 1970 was approaching 2:1 in 1988[3]. Reports from other countries are nearly as dramatic and over the past 20 years female deaths from lung cancer have more than doubled in Japan, Norway and Sweden.

Since 1950 several studies have been done and statistical evidence have proven beyond doubt that there is indisputable association between cigarette smoking and all types of primary lung carcinoma[2,4]. The greater the number of cigarettes smoked per day the higher the risk of lung cancer as well as the duration of smoking. Reduced lung cancer rates are found among ex-smokers.[1,2,3,4] In Hongkong, the use of kerosene stoves for cooking in households has been implicated.[5] In developing countries lung cancer is thought to be very rare. In Kenya Karuga[5] found only 19 histologically proven cases in nearly four years, while in Lusaka Bahl[5] found only 41 cases in three years, Stephen and Uraganda in Srilanka reported 84 cases in 10 years as quoted by Kakande.[5] Similar studies from Tanzania have not been published.

The increase in the number of smokers in our society, urbanization and industrialization, an increase in primary lung cancer is anticipated in future. hence there is a need to promote prevention measures now.

CASE PRESENTATION

J.M. a 61 year old male referred from Moshi in Northern Tanzania, where he had been residing throughout his life, was admitted under the care of thoracic surgical unit

His presenting complaints were cough for 9 months, which was initially dry and later became productive with a single episode of haemoptysis. The cough was worse in the mornings and late hours of night. This was later followed by pain localized on the left lower chest, which was pricking in nature and non-radiating. There was no history of fever or loss of body weight. He had been a heavy smoker from 1950 to october, 1988 when he was advised to stop because of his chest symptoms. He was smoking an average of 2 packets of cigarettes per day. He was a retired primary school teacher and he used to take alcohol occasionally. There was no history of bone pains, no headache no palpitation and no joints pain.

He had been residing in the rural areas throughout his life. There was no known history of Tuberculosis contact or chest trauma. He was a married man with 8 children all alive and well, without family history of any chest symptoms.

Examinations revealed an elderly man, wasted, not pale and not jaundiced. There were no palpable lymph nodes and no finger clubbing. The blood pressure was 140/90 mmHg with pulse rate of 82 beats per minute full volume and regular. The heart sounds were heard without any murmurs.

Respiration rate was 18 per minute, chest expansion with respiration was normal and equal both

sides, trachea was central percussion note was of normal resonance, breath sounds were normal, and no added sounds. Chest X-ray film revealed a left sided mass superimposed on the heart shadow on a frontal view. On the lateral view it showed a posteriorly located mass in the superior segment of the lower lobe. There were no lesions on the ribs or vertebral bodies. There was no evidence of pleural effusion, calcifications in the lesion or radiological evidence of hilar and mediastinal nodes enlargement. No tomograms were done.

A clinical diagnosis of carcinoma of the lung was entertained. Several investigations were done as follows:

(a) FBP - Haemoglobin = 10.8 g/dl
- Platelets = adequate
- ESR = 110 mm Westergren in the first hour

(b) Serum creatinine = 82 mmol/l

(c) Serum electrolytes - Na^+ 140 mmol/l
K^+ 4.2 mmol

(d) Urinalysis - Clear yellow
- S.G. 1010
- Reaction acidic
- Sugar nil
- No albumin
- No RBC
- No pus cells

(e) Lung function Tests:

The Physiologist commented that:

There was a predominantly obstructive lung disease affecting the expiratory phase, inspiratory and possibly blood gases might be normal in the patient (no facilities for blood gas analysis at our centre).

The patient was scheduled for bronchoscopy. The bronchoscopic findings were as follows:

- Vocal cords, Trachea and carina were normal.
- Right side main bronchus, upper lobe bronchus, intermediate, middle and lower lobe bronchi appeared normal.
- Left side main bronchus was normal. Tumour was seen in the left lower lobe bronchus causing complete obstruction. The tumour encroached the left upper lobe bronchus, bronchoscopically operable by pneumonectomy.

The tumour was staged as T_2NoMo. Three pieces of tumour tissue were taken for histopathological diagnosis. The pathologists reported a "moderately well differentiated squamous cell carcinoma"

The patient was planned for surgery. A postero-lateral thoracotomy was done. Chest was entered through the upper border of the 6th rib, tumour was found in the lower lobe involving the parietal pleura. Large lymph nodes were found at the hilum, subaortic and subcarina areas. The tumour also

extended to involve the adventitia of descending aorta. The pericardium, diaphragm, and recurrent laryngeal nerve were free of tumour invasion. An extra-pleural strip was done to free the tumour from the chest wall and the area marked with stainless steel wire. Tumour was easily stripped off the adventitia of the Aorta.

The pulmonary artery, superior and inferior pulmonary, veins, were identified doubly ligated and divided. The bronchus was dissected to the carina clamped distally divided and closed with interrupted fine silk sutures ensuring water and airtight closure. The hilar, sub aortic, subcarina lymph nodes were dissected and removed together with the lung and sent for histology. Estimated blood loss of about 1 litre was replaced. The chest wall was closed in layers and the patient recovered well from anaesthesia.

Post-operatively the patient was given crystalline Penicillin 5 megaunits intravenously as loading dose, then 3 megaunits intravenously 6 hourly for 24 hours. Intravenous fluids were also given 1 litre Darrows and 2 litres of Dextrose-saline in 24 hours.

Maintaining of the vital signs blood pressure, pulse, respiration and temperature 1/2 hourly for the first 24 hours was done.

The patient recovered well post-operatively. He had a bout of malaria which was treated with chloroquine injection 200 mg intramuscularly 8 hourly for 24 hours (3 doses), then 4 chloroquine tablets for one day and 2 tablets for the following day.

Histology of the resected specimen was reported as "small cell carcinoma of the intermediate cell subtype". The histology of the lymph nodes also confirmed metastatic carcinoma in the nodes. The patient had otherwise an uneventful recovery and was discharged 24 days after surgery.

In view of the histological results there was no additional treatment given since radiotherapy was considered unfavourable and chemotherapy was not available. On follow up he was seen two weeks after discharge and 3 months thereafter. The patient died at home 6 months after surgery of brain metastases.

RESULTS

Table I: Distribution of 15 Patients with lung carcinoma by Age and sex.

Age	Male	Female	Total
11-20	0	2	2
21-30	1	0	1
31-40	0	0	0
41-50	2	1	3
51-60	4	2	6
Above 60	2	1	3
Total	9	6	15

Most cases were between the 51 to 60 year age group. The ratio male:female 3:2. Two patients were under 20 years of age.

Table II: The histological types of primary lung carcinoma encountered in the study of 15 patients.

Histological cell Type	Number of Patients Smokers	Non-smokers	Total
Squamous cell Carcinoma	5	2	7
Adenocarcinoma	3	2	5
Large cell Carcinoma	0	2	2
Small cell Carcinoma	1	0	1
Total	9	6	15

Squamous cell carcinoma type was the commonest histological type seen in 7 out of the 15 cases.

Table III: The outcome of treatment in relation to disease stage.

Tumour Stage V	I	II	IIIA	IIIB	IV
Number of Patients	2	4	6	0	3
Surgery	2	2	3	0	0
Discharge from hospital	2	2	5	0	3
Operative death	0	0	1	0	0
Death within 2 years ofSurgery	0	2	1	0	2
Lost from follow up	1	2	1	0	1

One of the stage IIIA patient died of stroke one year after surgery.

Those patients lost to follow up are upcountry patients and their follow-up is done at nearby Regional Hospitals. We have no communication from this centre regarding their condition.

Table IV: The different modes of treatment offered

Stage -	Lobectomy	Pneumonectomy	Palliative	Absconded
I	2	0	0	0
II	2	1	1	0
IIIA	0	2	2	2
IIIB	0	0	0	0
IV	0	0	3	0

Seven of the 15 patients had surgery, lobectomy in 4 and pneumonectomy in 3 cases. One of the stage II patients and two of stage III patients refused surgery so they were offered palliative treatment.

Table V: Relationship between histology, stage of disease and survival of the 15 patients

Histology of Disease	Number of Patients	Stage of Disease	Survival in 2 years Alive	Dead	Outcome not known
Adenocarcinoma	2	1	1		1
Adenocarcinoma	2	II	1		1
Adenocarcinoma	2	IIIA	-	2	0
Adenocarcinoma	1	IV	-	-	1
Squamous Cell Carcinoma	0	I	0	0	0
Squamous Cell Carcinoma	2	II	1	0	1
Squamous Cell Carcinoma	3	IIIA	-	1	2
Squamous Cell Carcinoma	2	IV	0	1	1
Squamous Cell Carcinoma + Small cell Carcinoma	1	IIIA	0	1	0

The outcome of the patients depend more on the stage of the disease rather than histological type.

Table VI: Comparison between Pre-operative stage and intra-operative stage.

Number of Patients	Pre-operative Stage	Intra-operative Stage
2	II	II
2	II	III
3	III	III

Only two patients who were clinically staged as stage II were at surgery found to be stage III.

Table VII: Form of Palliative treatment and type of symptoms and signs palliated in the 15 patient

Symptoms andsigns	Stage of Disease	Pleural aspira-	Radio- therapy	Blood trans- fusion	Anage sics
Pleural Effusion	IV	3	-	-	3
Dyspnoea	IV	3	-	-	3
Hoarseness of voice	IIIA	-	2	-	2
Pain	II	-	1	-	0
Anaemia	IV	-	-	1	-

All the stage IV patients had massive pleural effusion, which required aspiration to relieve them of dyspnoea. All the patients except the stage II

patients required analgesics because of chest pain or joints pain.

One patient of stage II disease and two patients of stage III disease had radiotherapy as an adjuvant to surgery. Chemotherapy was not available. So the remaining four patients had surgery alone.

Table VIII: Duration of hospitalization Post-operatively for the 7 patients

Duration of hospital stay(in days)	0-10	11-20	21-30
Number ofpatients	2	4	1

Majority of the patients (i.e. 4) were hospitalized for an average of 2 weeks after surgery. One patient died same day after surgery.

DISCUSSION

Aetiology

(a) There is evidence establishing a positive relationship between cigarette smoking and lung cancer beyond any reasonable doubt.[1,2] Reports from "Royal College of Physicians" and the "United States Surgeon General" gives a collective evidence in both statistics, clinically and experimentally as reported by: Robins and Partridge.[1,2] Cigarette smokers have a 9-10 times risk of developing lung carcinoma

compared to non-smokers.[1] Heavy smokers of over 40 cigarettes per day have 20 times increase risk of developing lung carcinomas than non-smokers.[1]

Ex-smokers who had smoked 20 or more cigarettes daily, after 10 years of not smoking, attain the death rate of lung carcinoma similar to non-smokers.[2,3] In this study out of the 15 patients 9 were cigarette smokers, ranging from those who smoked 10 sticks per day. to 40 sticks per day. Evidence is accruing that those who live or work with cigarette smokers may also be at increased risk of developing the disease.[2,3] Wives of heavy smokers were found to have increased risk of developing lung carcinoma, and a close relation was observed.[2,3]

(b) Industrial hazards: Certain industrial exposures increase the risk of development of lung carcinoma. All types of radiation may be carcinogenic.[1,2] There was an increased incidence of lung carcinoma among the survivors of the Hiroshima and Nagasaki atomic bomb blasts.[1,2]

Uranium miners who are non-smokers have four times the risks of developing lung carcinoma, and the smokers have ten times increased risks than those of the general population.[1] Asbestos dust is another universally recognised carcinogen. Asbestos workers have about five times risk compared to the general

population, and it takes between 10-30 years latent period before developing lung cancer.[1,2] Other agents include nickel, chromates, coal, silver, mustard gas, arsenic, Beryllium, Gold and newspaper workers.

(c) Genetic factors: At one time it was proposed that patients with lung cancer had genetically higher levels of the enzyme aryl-hydrocarbonhydroxilase (AHH) which converts non-carcinogenic, polycyclic hydrocarbons to carcinogenic intermediates.[1]

This hypothesis has not been proven beyond doubt. Decreased immunologic responsiveness occurs in some patients with lung cancer, but whether this is the cause or the effect of carcinoma is unclear.[1]

(d) Scarring: Some lung cancers arise in the vicinity of pulmonary scars and are termed "scar cancers". Histologically these tumours are usually adenocarcinomas. Among the scars incriminated are old and granulomatous infections, such as Tuberculosis.[1]

(e) Pet birds: Pet birds in the home is another speculative association, possibly mediated via damage to alveolar macrophages and a deficiency in humeral and cellular immunity.[2]

(f) Air Pollution: We all swim in a sea of carcinogen and it is conceivable that atmospheric pollutants may play some role in the increased

incidence of primary lung carcinoma.[1,2] However, there is difference of opinion as to whether there is an increased risk of lung carcinoma in urban as opposed to rural areas after populations are corrected for tobacco usage.[1,2] Although most authorities acknowledge the existence of a small urban factor in lung carcinoma incidence, the main culprit, far and away is cigarette smoking.[1,2,3,4]

HISTOLOGICAL CLASSIFICATION

Histological classification of lung cancer is important because some clinical features, prognosis and treatment vary with cell type.[2,3] Numerous histologic classifications of primary lung carcinoma have been proposed,, but the currently popular ones based on classifications of the World Health Organization (WHO) divides these into four major categories.[1,2,3,4]

(a) Squamous cell carcinoma, making up 35-50%.[1,2,3,6] In this study squamous cell carcinoma represented 43.7%.

(b) Adenocarcinoma (including bronchiolo-alveolar carcinoma) make 15-35%. In this study represented 31.25%.

(c) Small cell (including Oat cell) carcinoma make 20-25%.[1,2,3,6] In this study makes 6.25%.

(d) Large cell carcinoma makes 10-20%.[1,2,

There may be overlap in the histologic pattern, even in the same cancer may found two different cell types.[1,3] Thus combined types of squamous and adeno-carcinoma or of small cell and squamous carcinoma are not infrequent.[1] In this study one patient had two histologic cell type within the same tumour, squamous cell carcinoma and small cell carcinoma.

CLINICAL PRESENTATION

Patients with primary lung carcinoma may present with symptoms due to the primary tumour, secondary deposits, para-neoplastic syndromes or a combination of any of these three manifestations.

Symptoms due to primary tumour include: productive cough lasting for more than 2-3 weeks, haemoptysis and or dyspnoea due to partial or complete bronchial obstruction. Chest pain and wheezes are less common symptoms. Sometimes the first symptoms may be due to invasion of the mediastinum causing, pressure on recurrent laryngeal nerve causing hoarse voice, pressure on trachea or main bronchus causing stridor and pressure on oesophagus causing dysphagia.[6,7] Secondary deposits to the brain may manifest as headache, vomiting epileptiform fits weakness of a limb, leading to paralysis. Bone pains, especially in vertebrae, pelvis and long bones due to metastases from lung carcinomas are common. Endocrine related symptoms or

paraneoplastic syndromes such as: osteoarthropathy, arthritic symptoms and finger clubbing are also common. Other paraneoplastic syndromes include carcinomatous neuropathy, inappropriate antidiuretic hormone secretion causing hypokalaemia, (muscle weakness) hyponatremia, hypercalcemia presenting clinically with confusion and drowsiness, gynaecomostia or cushing's syndrome.[5,6] Other symptoms include general body malaise, anorexia and loss of weight.[6,7]

Diagnosis

The diagnosis of lung carcinoma must be based on the study of the histologic features of a tissue sample.[6,7] All the patients in this study had positive histological results for primary lung carcinoma.

Diagnosis and cell typing usually involves either cytological examination of expectorated sputum or bronchoscopy and subsequent cytological examination of bronchial washings, brushings or histological samples. Less commonly patients need percutaneous needle biopsy for peripheral lung lesions. Occasionally a tissue diagnosis is only made by surgical exploration of the mediastinum via a thoracotomy or by biopsy of a metastatic lesion.[7]

Cytological examination of sputum specimens should be a part of most diagnostic work - ups for

suspected lung cancer. The yield with one sputum specimen is reported to be about 40%. Whereas the yield from four properly collected specimens may approach 80-85%. Mid-morning (10 a.m.) sputum samples are recommended for a higher yield.[7]

Bronchoscopy is useful both for obtaining material for tissue diagnosis and, assessment of the extent and operability of the tumour. This technique has improved significantly with development of the fibreoptic bronchoscopy and it has even nearly doubled the ability of the physician to diagnose lung carcinoma with bronchoscopy.[5,7,8] This includes bronchial brushing and cytologic studies of bronchial washings that may be obtained at the same sitting.[8] It is important to remember that cytologic specimens taken at the time of bronchoscopy are less likely to be positive compared to those taken within 48 hours following bronchoscopy.[6] This is due to the irritation caused by the scope, leading to an increase in the shedding of malignant cells. Bronchoscopy is useful both for obtaining material for tissue diagnosis of endobronchial lesions and for the assessment of the extent of the disease as well as its operability.[7]

In the assessment of mediastinal lymph nodes, the use of Gallium (Ga67) scanning has shown promising results in several studies.[2] Although

Gallium (67) scanning and CT scan are complimentary in the assessment of metastasis in the mediastinal lymph nodes.

CT scan is used more often than Ga67 scan.[2] CT scanning will show nodes of 1 cm or more in diameter and nodes with 2 cm or more in diameter in such circumstances certain metastatic disease in 95% of them.[2]

Mediastinoscopy:- In approximately one half of lung cancer patients, mediastinal lymph nodes are already involved with tumour when the patient first comes to diagnosis.[2] For this reason mediastinoscopy has been a very important addition to the staging and diagnosis of lung cancer.

STAGING OF LUNG CANCER

A new international staging system for lung cancer has been developed by members of the Task Force on Lung Cancer of the "American Joint Committee on Cancer staging (AJCC) in co-operation with the "Internation Union Against Cancer."[9]

In this study we used the TNM Classification which has been modified by the new International staging system of 1986. The Classification is as follows:

T= Primary Tumour

Tx= Tumour proven by the presence of malignant cells in bronchopulmonary secretions but not visualized

roentgeno-graphically or bronchoscopically, or any tumour that cannot be assessed as in a retreatment staging.

To = No evidence of Primary tumour

Tis = Carcinoma in situ

T_1 = A tumour that is 3.0 cm or less in greatest dimension, surrounded by lung or visceral pleura, and without evidence of invasion proximal to a lobar bronchus at bronchoscopy.

T2 = A tumour more than 3.0 cm in greatest diameter or a tumour of any size that either invades the visceral pleura or has associated atelectasis or obstructive pneumonitis extending to the hilar region. At bronchoscopy, the proximal extent of demonstrable tumour must be within a lobar bronchus or at least 2.0 cm distal to the carina. Any associated atelectasis or obstructive pneumonitis must involve less than an entire lung.

T3 = A tumour of any size with direct extension into the chest wall (including superior sulcus tumours), diaphragm, or the mediastinal pleura or pericardium without involving the heart, great vessels, trachea, esophagus or vertebral body, or a tumour in the main bronchus within 2 cm of the carina without involving the carina.

T4 = A tumour of any size with invasion of the mediastinum or involving heart, great vessels,

trachea, esophagus, vertebral body, or carina or presence of malignant pleural effusion.

Nodal Involvement (N)

No = No demonstrable metastasis to regional lymph nodes

N1 = Metastasis to lymph nodes in the peribronchial or the ipsilateral hilar region, or both, including direct extension.

N2 = Metastasis to ipsilateral lymph nodes and subcarinal lymph nodes.

N3 = Metastasis to contralateral mediastinal lymph nodes, contralateral hilar lymph nodes, and ipsilateral or contralateral hilar lymph nodes, and ipsilateral or contralateral scalene or supraclavicular lymph nodes.[9]

M = Distant Metastases

Mo = No (known) distant metastasis

M1 = Distant metastasis present.

Previous AICC Versus New International System: Staging comparison.

AJCC Stage		International System Stage
Occult	Tx, No, Mo	Occult
I	Tis, No, Mo	0
	T1, No, Mo	I
	T2, No, Mo T1, N1, Mo	II
II	T2, No, Mo	
III	T3, No, Mo T3, N1, Mo T1, N2, Mo T2, N2, Mo T3, N2, Mo	IIIA
	Any T, N3, Mo T4, any N, Mo Any malignant effusion	IIIB
	Any T, any N, M1	IV

Note: T,N,M descriptions based on definitions of New International Staging System

AJCC = American Joint Committe on Cancer

TREATMENT

SURGERY While lobectomy or pneumonectomy offers the best hope of cure only 8-15% of patients with lung carcinoma have operable disease.[2] If patients with small primary non-small cell carcinomas (NSCL)

without nodal involvement or metastases are treated surgically, a 5 year survival rate between 25 and 50% may be obtained.[2] Unfortunately they comprise a small minority of the total number of patients with lung carcinoma (SCLC) at any stage is widely regarded as a non-surgical disease and yet two reports and a review have drawn attention to the fact that for the minority with this cell type who truly have localized disease surgery offers a prospect of long-term survival.[2]

In this study one patient had two types of histological results, bronchoscopic biopsy revealed squamous cell carcinoma, after pneumonectomy histology reported small cell carcinoma, the patient died after 6 months of surgery. He deteriorated faster after surgery and became more incapacitated until he succumbed after 6 months. One cannot say exactly whether surgery did prolong or shortened his life. It has been reported that for small cell carcinoma if untreated the median survival is 8-12 weeks.[2]

Among the 15 patients in this study 4 had lobectomy and 3 pneumonectomy, 6 had palliative treatment and 2 absconded. The palliative treatment included analgesics to relieve pain, and pleural tape to relieve dyspnoea.

Out of the 7 operated patients one presented to our clinic 3 months later with neurological symptoms of metastatic disease to the brain, and died after a few days. Another patient was followed up for 19 months after which she never turned up to our clinic. The third patient died in the medical ward 8 months later due to cerebrovascular accident. The fourth died 8 hours post-operatively and the 5th died 6 months later with disseminated metastases. The two remaining patients went upcountry to their homes after discharge from our hospital. All the three stage IV patients were offered palliative treatment, two died while still in the ward and one was only seen once in the follow up clinic.

According to the authors opinion surgery did offer a chance of prolonging life, it was unfortunate that most of our patients presented very late. The longest survival had T2,No,Mo and was offered left upper lobectomy with post-operative Radiotherapy to the area where tumour was resected and this was marked with staplers.

The choice of operation of the lung carcinoma traditionally has been either standard pneumonectomy or lobectomy depending on the location of the lesion and the extent of the disease.

TREATMENT OF NON-SMALL LUNG CANCER

As stated previously the treatment of primary lung carcinoma varies depending on the histological type of the tumour; the stage of the cancer, and multiple host factors, such as age, general condition, and the presence of associated disease. In choosing between alternative therapeutic approaches, it is necessary to weigh both the effect of treatment on life expectancy and the quality of that life.[7]

The current modalities of treatment for lung cancer include surgical resection, radiation therapy, chemotherapy, and immunotherapy. Despite aggressive application of these modalities, the five year survival of large groups of patients with lung carcinoma remains in the range 10 to 15%.[7] Resection is the treatment of choice for stage I and II non-small cell lung carcinoma. Broncho-alveolar cell carcinoma, has no effective non-surgical treatment. Some stage III non-small cell lung carcinoma should be resected if there is a reasonable probability that all of the cancer can be removed. This wound include peripheral carcinomas with direct invasion of the chest wall without metastasis to lymph nodes. The need to preserve functioning lung tissue and the lack of a significant difference in the survival of similarly staged patients, lobectomy as opposed to

pneumonectomy is the preferred operation if the entire tumour can be removed and adequate bronchial margins obtained.[7]

When patients are evaluated and felt to be curable by a limited pulmonary resection but have reduced pulmonary and cardiac reserve making the risk of lobectomy prohibitive, segmental or wedge resection has become an accepted form of treatment.[7] Jensik et al as quoted by Anderson and Arentzen showed that in 168 patients with stage I primary lung carcinoma, overall survival following segmentectomy was 53% at 5 years, 37% at 10 years and 25% at 15%.[6,10]

Radiotherapy

U nlike chemotherapy, radiation therapy is a local treatment and in cases of metastatic disease it clearly offers only the hope of symptom palliation.[2] In this role it is particularly successful in controlling haemoptysis and cough, and it is effective in reducing the pain associated with chest wall involvement, skeletal metastases and superior sulcus tumour. It can be helpful in reducing the dysphagia associated with oesophageal compression and in relieving superior vena caval obstruction. If given very promptly it may enable re-expansion of collapsed lung to occur and similarly it may be helpful in cases of partial spinal cord compression

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When patients are evaluated and felt to be curable by a limited pulmonary resection but have reduced pulmonary and cardiac reserve making the risk of lobectomy prohibitive, segmental or wedge resection has become an accepted form of treatment.[7] Jensik et al as quoted by Anderson and Arentzen showed that in 168 patients with stage I primary lung carcinoma, overall survival following segmentectomy was 53% at 5 years, 37% at 10 years and 25% at 15%.[6,10]

Radiotherapy

U nlike chemotherapy, radiation therapy is a local treatment and in cases of metastatic disease it clearly offers only the hope of symptom palliation.[2] In this role it is particularly successful in controlling haemoptysis and cough, and it is effective in reducing the pain associated with chest wall involvement, skeletal metastases and superior sulcus tumour. It can be helpful in reducing the dysphagia associated with oesophageal compression and in relieving superior vena caval obstruction. If given very promptly it may enable re-expansion of collapsed lung to occur and similarly it may be helpful in cases of partial spinal cord compression

CHEMOTHERAPY

For non-small cell lung carcinoma (NSCLC) Chemotherapy should be regarded as experimental (Drug and therapeutics Bulletin) as quoted by partridge. The three most effective agents which are currently being evaluated are: ifosfamide mitomycin C and Cisplatinium reported by Bakowski and Crouch as quoted by Partridge.[2] However, it is too clearly to say whether such regimens have an advantage over radiotherapy or surgery.[2]

Small cell lung carcinoma (SCLC) is overtly metastatic in 50-55% of cases at presentation and micrometastases are likely to be present in most of the rest.[2] Untreated the median survival for SCLC is 8-12 weeks.[2] Elderly male patients with overt metastases,weight loss and low plasma sodium, low albumin and raised alkaline phosphatase levels have the worst prognosis Souhami et al as reported by Partridge.[2] Chemotherapy is the mainstay of therapy in most patients with SCLC and can provide excellent palliation of symptoms such as pain, haemoptysis, breathlessness and superior vena cava obstruction. It can improve survival to a median 14 months in the better prognosis groups.[2] Unfortunately relapse is followed by a less good response to chemotherapy than at initial presentation.[2] The addition of thoracic and cranial radiotherapy to chemotherapy may

respectively reduce the likelihood of recurrence at the primary site and present cerebral metastases but it has not led to a significant increase in survival of patients.[2] Studies of the necessary duration of chemotherapy in SCLC suggest that around six courses is a reasonable optimum duration of initial chemotherapy and that maintenance chemotherapy or further treatment on relapse is not useful (this was reported by Spiro and Souhami as quoted by Partridge.[2]

Current regimens tend to incorporate, etoposide, infosfamide, cisplatin, carboplatin daxorubicin and vincristine in various combinations but it should be noted that single agents may still have a role in the context of palliative chemotherapy.[2] Oral etoposide given to 5 days every 4 weeks to a group of patients aged 70 years and over with SCLC has been shown to be an effective and well-tolerated treatment (Smit et al as reported by Partridge.[2]

Overall however, survival rates have not improved in the last decade and new ways of giving chemotherapy and newer methods of support during such treatment may be necessary before further progress is made.[2]

Immunotherapy

There is relatively limited data available on which to base a judgement on the possible benefits of

immunotherapy, either alone or in combination with chemotherapy in the treatment of patients with lung carcinoma.[7]

Although the immunotherapy of lung carcinoma is certainly in its infancy and reliable clinical results have not yet been reported, sufficient information is available from animal models. Immunologic studies in patients with lung carcinoma and immunotherapy may be trials in patients with other neoplasms tend to suggest that immunotherapy will have role in the future treatment of patients with lung carcinoma.[7]

Other Treatment

In addition to the palliative treatments mentioned above; relief of symptoms due to metastases and non-metastatic manifestations may be necessary. Adequate relieve breathlessness is common.[7] If the latter is due to major airway luminal obstruction the patient may benefit from endoscopic laser resection or cryotherapy or from the newer experimental techniques such as endobronchial irradiation or stenting.[7] Aspiration of pleura effusion may be necessary with consideration of pleurodesis, pleurectomy or pleuroperitoneal shunts for recurrences. For those who are still breathless exclusion or treatment of reversible airway disease or pulmonary emboli is essentially followed by

attempted palliation of residual distress with steroids opiates and nebulized local anaesthetic agents. The last three treatments may also be necessary for the palliation of a distressing cough.[7].

CONCLUSION AND RECOMMENDATION

Primary lung carcinoma is likely to remain a major clinical problem, despite refinements in the selection of patients for surgery and radiotherapy, and reports of impressive remission rates with chemotherapy.[2] In this study no concrete recommendation can be made due to the small sample. Studies done in other places revealed no significant improvements in survival and until. Such improvements are visible we should maximise our efforts at preventing the young from smoking, limiting atmospheric pollution and we should provide caring and empathic palliative medicine to those who already have this distressing disease.[2].

Patient education: Progress will be made in the prevention of carcinoma of the lung when people realize that the use of tobacco products is suicidal.[4] The present epidemic of lung carcinoma in women is a direct result of the increased social acceptance of women smokers.[4] Even smokeless tobacco

is associated with a higher incidence squamous cell carcinoma of the floor of the mouth and tongue.[4]

The Zimbabwe Medical Association (ZIMA) has resolved to challenge the tobacco industry, and the ministry of health to take a more realistic attitude to the completely unrestrained promotion of smoking in that country.[11] Tobacco is big business in Zimbabwe and the industry is a major contributor to government funds through exports and excise duty on local sales. Consequently it is very powerful in government and commercial circles and regularly attacks the policies of WHO and the anti-smoking lobby.[11] However, according to a ZIMA statement doctors are seeing an alarming number of cancers of the lung a high incidence of heart and vascular diseases associated with smoking and more women smokers.[11] The doctors particularly object to advertising campaigns which give the impression that smoking is beneficial by attempting to link it to success in sport. No health warnings have been given on no-smoking areas in public places.[10]

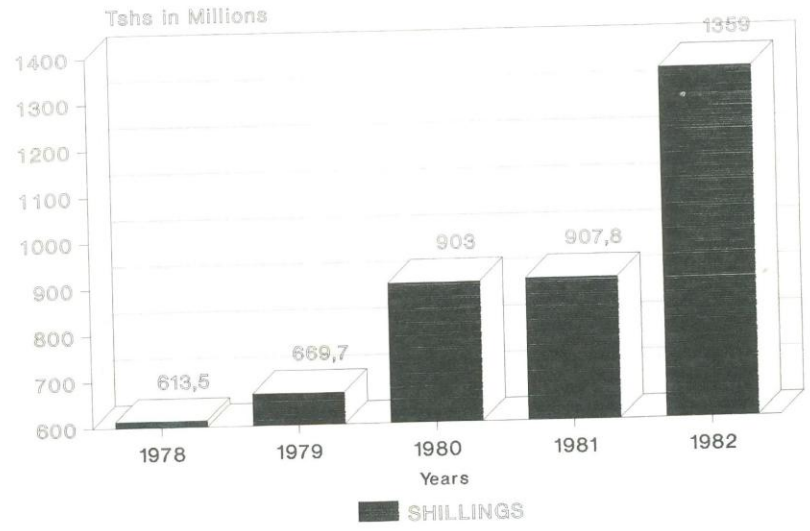
In Tanzania likewise the government earns a big revenue from the tobacco industry. As can be seen in **Figure I.** The rate of cigarette sales is increasing tremendously annually. In 1978 there was 613.5 million shillings in cigarette sales. This doubled by 1982 when it was 1359.0 million shillings i.e a

duration of only four years. Though lung cancer is not yet a major public health problem at the moment, we are bound to realise it in a few years to come.

Cost containment: One of the most effective methods the government could carry out would be to eliminate subsidies for growth of tobacco.

Tobacco advertising should be eliminated or, if it is continued, the warning about carcinoma of the lung should be in as large letters as the name of the cigarette on the packet. In developed countries, tobacco companies are rapidly diversifying into other industries hopefully preparing for the day when they will close their plants permanently. Control of industries pollutants environment protection and other approaches may also decrease the likelihood of carcinoma of the lung. Unfortunately, early detection and screening programmes for occult carcinoma have not been found to be cost effective. Though there was an increase in population during this four year period, it means even the population of smokers rose.

FIG 1: CIGARETTE SALES IN TANZANIA 1978-1982



KCMC SATURDAY PRESENTATION WITH PERMISSION FROM DR. MMUNI

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BRONCHIECTASIS

SUMMARY

A total of 6 patients with diagnosis of bronchiectasis were seen in surgical Firm II during the period of July 1988 to August 1990 (26 months). Their ages ranged from 8 to 54 years, with a mean age of 14 years.

Diagnosis was made by the presenting complaints, physical examination, chest X-ray and bronchoscopy. Bronchography could not be done at our x-ray department, due to lack of the appropriate contrast media during this period.

All the 6 patients were operated 4 had pneumonectomy, 2 had lobectomy (one had left lower lobe lobectomy and the second one had right lower and middle bilobectomy. With exception of one patient who died on the operating table due to cardiac arrest the other five recovered uneventfully and they were free of symptoms after 6 months. Two were seen one year later in healthy, symptom free condition.

INTRODUCTION

Bronchiectasis is a permanent dilatation of the bronchial walls and is usually the result of infection.[1] It was once a common disease of the lung but now reported to occur less frequently in developed countries.[2] Although bronchiectasis may occur in very young and elderly persons, it has

developed most often in teenagers and young adults. The onset occurs in the first decade in about 42 percent of cases and in 64 to 69 percent before age 20.[1,3]

Bronchiectasis was first described by Renè Laennec in 1819. In 1907 Carl Beck wrote "Bronchiectasis i.e., as a rule not a disease per se, but a consequency of various affections of the lungs or bronchi".[3]

Surgeons have been aggressive leaders in the crusade against bronchiectasis, and the development of pulmonary resection was stimulated by this disease.[3] Resection of a lung diseased with bronchiectasis was attempted periodically during the nineteenth century.[3] Due to the high mortality and morbidity, lesser procedures were undertaken, such as cautery excision with diversion of the bronchial drainage to the outside through multiple fistula.[3] Surgical principles underlying one stage lobectomy were proposed by Harold Brunn in 1929. He employed intrapleural suction drainage to remove fluid and air to augment expansion of the remaining lung lobes. Rudolph Nissen performed the first successful pneumonectomy in 1931 on a 12 year old girl with bronchiectasis.[3] Two years later, Cameron Height performed an identical procedure. Evarts Graham made a double advance in the third successful

pneumonectomy in 1933, using a single-stage operation in a patient with bronchogenic carcinoma.[3]

Infectious diseases of the lung and pleura remain the greatest challenge to the thoracic surgeons in developing countries who are often handicapped by inadequate facilities, and lack of drugs. There is also a problem of illiteracy, poverty, superstitious beliefs and poor environmental hygiene.[4] In developed countries, the incidence of infectious diseases of the lung and pleura has declined as a result of effective antibiotics, but in developing countries the incidence has not changed significantly in the past few decades.[4]. Clinically, bronchiectasis is a disease of the basal segments of the lower lobes, the lingula, and the middle lobe.

CASE REPORT

T.H. In patient number 522580, an 8 year old girl was referred to our thoracic, surgical unit from Bugando medical centre Mwanza. She presented with a 10 months history of productive cough, producing a fowl-smelling sputum.

Prior to the above symptom she aspirated a small bead and coughed it up a month later. Since then she had been having recurrent chest pain fevers, cough producing copious sputum on and off, this was also accompanied by a considerable loss of body weight, there was no past medical history of measles,

pulmonary tuberculosis or whooping cough, she was treated with antibiotics several times which gave her slight relief for a short period.

On examination, she was wasted and had finger clubbing; she had no cyanosis or generalised lymphadenopathy she was not pale and she was afebrile. Her pulse was 84 beats per minute regular and of full volume, and the blood pressure was 90/60 mmHg. The heart sounds were heard without any murmurs. Respiration rate was 24 per minute, chest expansion with respiration was normal and equal both sides. Trachea was central percussion note was of normal resonance, she had marked insipiratory crackles on the right lower zone. The sputum was yellowish in colour. Chest X-ray revealed lung consolidation in the right lower lobe and partly the right middle lobe was also affected.

Sputum for pyogenic culture and sensitivity revealed normal upper respiratory flora. Sputum for AFB was negative and Mantoux test was negative. The patient was then scheduled for bronchoscopy. This was done using the rigid Karl Storz ventilating bronchoscope. The findings were:

i) Inflamed trachea and major bronchi, blunt carina

ii) Some thick secretions from left side no foreign body visualized.

iii) Large quantities of pus from the right side particularly from the right lower lobe, no foreign body was visualized

iv) The middle lobe bronchus was not well visualized.

Post bronchoscopic sputum was sent for aerobic culture and sensitivity, the results reported no growth. She then continued on postural drainage. She was scheduled for right lower and middle bilobectomy. She was taken to main theatre and under general anaesthesia she was placed on the left lateral position, with the right arm supported with arm-stand. The skin was cleaned with antiseptic starting with savlon, then Iodine and 70%alcohol and draped.

A right postero-lateral thoracotomy was done; a bronchiectatic right lower lobe was found, and the inflammatory process had crossed the fissure involving the medial segment of the right middle lobe. The rest of the lung appeared normal, there were also some adhesions. The lower lobe was resected together with wedge excision of part of the middle lobe. Then the bronchial stump was closed with 2/0 silk interrupted and it was tested and found to be air tight. Blood loss was estimated to be around 200 - 300 mls. After ensuring thorough haemostasis the chest wall was closed in layers and

chest-tube with underwater seal drainage was left in the chest. Postoperatively the patient was sent to the intensive care unit for close monitoring and continuous active suction. She was also given antibiotic; ampicillin injection 500 mg intravenous intraoperatively and then 250 mg 6 hourly post operatively for 2 days then changed to capsules 250 mg orally 6 hourly for 4 days. She was also given intraveous fluids Dextrose-saline 1.5 litres for the first 24 hours after which she was allowed to feed normally. The next day she was started on chest physiotherapy enhance lung expansion. She recovered uneventfully and a control chest X-ray, was taken on the 4th post-operative day and found to be satisfactory. The chest tube was removed on the 4th day after ensuring good lung expansion clinically and radiologically with no more air leak.

The patient was discharged on the 12th post-operative day after a control X-ray which showed good lung expansion. She was sent back to Mwanza to continue follow up at their paediatric out patient clinic.

The following tables which summarizes the other cases.

Table 1: Age and sex of a 6 patients with Bronciectasis

Age (Years)	Sex Male	Female	Total
8	1	1	2
14	1	1	2
15	1	0	1
54	1	0	1
Total	4	2	6

There were 4 males and 2 females

Table II: The side of the lung affected and operative procedure offered

Number of Patient Left	Right	OPERATIVE PROCEDURE Pneumonectomy	Lobectomy	Total
0	1	1	0	1
3	-	3	-	3
-	1	-	RLL + ML(1)	1
1	0	0	LLL(1)	1
4	2	4	2	6

LLL = Left Lower Lobe

ML = Middle Lobe

RLL = Right Lower Lobe

Out of the 6 patients 4 had pneumonectomy and 2 had lobectomy. The left side appeared to be the most affected.

Table III: The treatment offered, age of the patient outcome, and the follow up after 6 months

Number of Patients	Age (years)	Pneumo-nectomy	Lobe-ctomy	Outcome after surgery	Follow up to 6 months
1	8	1	-	Discharged	Good
1	8	-	1	Discharged	Good
2	14	2	-	Discharged	Good
1	15	-	1	Discharged	Good
1	54	1	-	Died on the table	N/A

Out of the 6 patients operated one patient had cardiac arrest on the operating table. Otherwise the rest were clinically doing well up to 6 months of follow up.

DISCUSSION

Aetiology: This may be divided into two major factors:

I The Acquired conditions

II Congenital or Hereditary conditions

I The Acquired conditions

(a) Infectious diseases: Bronchiectasis is a disease of the pulmonary parenchyma as well as

of the bronchi.[3, 5] Pneumonitis with resultant pulmonary fibrosis may follow pertussis, measles or other viral or bacterial infection.[3] The theory that bronchiectasis is a result of scarring and contraction of lung tissue around bronchi was first proposed by Corrigan in 1938 as quoted by Sabiston D.C. He wrote "If the fingers of the readers' hand, separated one from another, be supposed to represent a bunch of bronchial tubes and at the intervals be supposed to be filled with fibres undergoing a slow contraction, it is obvious that the first tendency of that contractile action will be to draw the sides of the fingers toward one another or if they were tubes to dilate them.[3]"

(b) Bronchial obstruction: This is another causative factor and it can either be:

(i) Extraluminal

(ii) Intraluminal

In Extraluminal - this obstruction occur in childhood, due to glandular enlargement associated with measles, whooping cough, or primary tuberculosis. The bronchial obstruction is followed by infection, which leads to mucosal ulceration, loss of bronchial cartilage and consequent weakening of the bronchial wall.[5] The weakened bronchi later

dilate due to the constant negative pressure on the bronchial wall associated with respiration.

Re-aeration of the collapsed lobe may occur later as the glandular compression subsides but the bronchi remain dilated and infected. The primary diseases are now much less common than 40 years ago and because of this bronchiectasis is now relatively rare.[5] In addition the bronchial infection so often associated with these diseases is now treated much more promptly and efficiently with antibiotics and therefore permanent structural changes are much less likely to develop in the lung distal to the bronchial blockage:[5]

(ii) Intraluminal bronchial obstruction may result from an unsuspected inhaled foreign body, plugs of tenacious mucopurulent material, tumours, under these conditions, the bronchiectasis is localised to the obstructed lung segment.

Bronchiectasis can also complicate diffuse obstructive airway diseases, most commonly atopic asthma and chronic bronchitis.[6] Bronchiectasis is still a prominent feature of mucoviscidosis; The mucus-secreting glands of the respiratory tract as the pancreas produce a scanty, thick and sticky secretion.[3]

Successful management of respiratory problems in these patients is extremely difficult.

Bronchiectasis may occur in heroin addicts and appears to be related to episodes of heroin-induced pulmonary edema and infection.[3]

II <u>Congenital or Hereditary condition</u>

These include:

(a) **Congenital bronchiectasis** This is caused by a defect in the development of bronchi and usually affects a whole lobe or entire lung.[6]

(b) **Cystic fibrosis:** Widespread severe bronchiectasis in this condition is a reflection of the generalised defects in exocrine gland secretions.[3]

(c) **Intralobal sequestration of the lung:** In this condition a part of the lung, usually in the left lower lobe is supplied with blood not by a branch of the pulmonary artery but directly from the systemic arteries.

Characteristically, the airways in the sequestered bronchiectatic lung tissue are separate from those in the adjacent normal lung. The affected part becomes infected and its airways dilate.

(d) **Immunodeficiency states:** Such as congenital IgA deficiency, hypogammaglobulinemia, and alpha- 1 - antitrypsin deficiency. These conditions are associated with heightened susceptibility to

repeated bacterial infections and localized or diffuse bronchiectasis.[6]

(e) **Immotile cilia and Kartagener's syndrome:** Primary ciliary dyskinesia is a well recognized feature of Kartageners' syndrome. This is a syndrome comprising of chronic sinusitis, bronchiectasis and dextrocardia or situs inversus. Mucociliary clearance is an important defence mechanism of the respiratory tract. It depends on effective functioning of the mucus and cilia.[2,7] Mucus is produced by goblet cells which are found lining the tracheobronchial tree. The mucociliary clearance time effectively assesses the function of both the mucus and cilia, while ciliary beat frequently and ciliary ultrastructure specifically assess abnormal glycoproteins in bronchiectasis results in mucus with sub-optimal visco-elastic properties while ciliary beat frequently is slower in pus than in clear secretion.[7] There is currently no single or unifying explanation for abnormal mucociliary action and there is most likely to be an interplay of factors.[7].

(f) **Necrotizing Pneumonia:** Bronchiectasis occasionally follows necrotizing or suppurative pneumonias caused by the tubercle bacillus or staphylococcal or mixed infections. In past

years it was considered to be a sequel of childhood necrotizing infections complicating measles, whooping cough and influenza.[6]

PATHOPHYSIOLOGY

Bronchiectasis may be classified anatomically as:

1. Cylindrical
2. Varicose
3. Saccular

Bronchi involved with bronchiectasis undergo destruction of elastic tissue and muscular coats of the bronchial walls, with replacement by fibrous tissue. Small branch bronchi may become fibrosed and cord-like. The ciliated columnar epithelial lining may show inflammatory change plus squamous metaplasia.

Mucous gland hyperplasia and excess mucus production with stagnation occur and because of the impaired cleansing function of the lung, bacteria that lodge at these sites are not properly cleared.[3] There is extreme enlargement of the bronchial arteries and numerous anastomosis of these vessels with the pulmonary arteries in lung segments diseased by bronchiectasis.[3,5] Because of these rich bronchopulmonary anastomoses bright red blood under systemic pressure may pour into the respiratory tree, with serious or even fatal consequences. In addition to being a troublesome source of haemoptysis, the

expanded bronchial arterial supply to the diseased lung may impose an added circulatory burden on the heart, contributing to pulmonary hypertension and ultimately to the development of corpulmonale.[3] Advanced saccular bronchiectasis of the entire left lung may result in absence of uptake on a pulmonary scan and nonfilling of the left pulmonary artery on a pulmonary angiogram. Dye injected into the descending aorta of such a patient showed marked tortuosity of the bronchial vessels and retrograde filling of the left pulmonary artery.[3]

Bronchiectasis is usually located in the basal segments of the lower lobes and is associated with involvement of the corresponding middle lobe. Ordinarily the superior-segments of the lower lobes are free of disease.[3] The lingula is diseased in 60 to 80 per cent of patients with bronchiectasis of the left basilar segments.[3] Similarly from 45 to 60 per cent of patients with bronchiectasis of the right basilar segments will have involvement of the right middle lobe. There is approximately a 40 per cent incidence of bilateral bronchiectasis. Involvement of the upper lobes is rare but may follow scarring of old tuberculosis; the disease here is a dry type of bronchiectasis.

Clinically patient with bronchiectasis show evidence of chronic disease. They are often thin and

poorly developed. Chest examination may reveal asymmetry, with flattening and decreased motion on the affected side and unilateral disease. Rales are heard over the affected area and may be coarse.[2].

DIAGNOSIS

Roentgenographic findings, such as decreased volume of the lower lobes or segmental or lobal atelectasis, are not specific for bronchiectasis.[2] An association mediastinal shift may be present, but only blurring of the diaphragmatic shadows or heart border with linear streaking or strands may be seen.[2] Honey combing and small air-fluid levels are unusual, except in severe cases.[2]

Laboratory studies may indicate only mild anemia with white cell count elevations, during acute episodes.

Sputum smears and cultures show normal flora. Test for immune system alterations should be carried out and may be positive in some patients, particularly young children.

Bronchoscopy is necessary to rule out foreign bodies, benign tumours, broncholiths, bronchial stenosis and other problems that may exacerbate the disease.[2,3] It should also be done to exclude the rare case of correctable bronchial obstruction or carcinoma, to obtain accurate cultures, and to aspirate the tracheo-bronchial tree. Careful bronchoscopic

pulmonary toilet can achieve surprisingly durable symptomatic benefit.[8]

Bronchography: For diagnosis, localization and determination of extent of the disease, bronchography which is instillation of a radiopaque dye into the tracheo-bronchial tree to visualize the bronchi is necessary. Either general or local anaesthesia[8] is used and a small endotracheal catheter is inserted with fluoroscopic control and image intensifier monitoring, an oily dye, propyliodone (Dionosil) is injected sequentially into all lobes and segments, beginning on the side of dorminant disease.

A complete unilateral bronchogram is required without allowing contrast material to escape into alveoli.

Evaluating each lung on a separate day is safest, but bilateral bronchograms are necessary to make sensible decisions. Following bronchograms, postural drainage and percussion help eliminate the dye quickly. Bronchography should not be performed within 3 months of an acute episode of pneumonia, because any bronchial dilatation may be reversible.[2].

TREATMENT

In order to plan treatment it is essential to have good bronchograms of the whole of both lungs so that the extent of the bronchiectasis can be accurately defined.[5]

(a) **Prophylactic treatment:** Much can be done to prevent the development of bronchiectasis by treating the respiratory complications of measles and whooping cough with antibiotics and breathing exercises, and carrying out bronchoscopy at an early date should atelectasis occur.[5,8]

(b) **Medical treatment:** The majority of patients with bronchiectasis do not require operative treatment.[8] If the bronchiectasis is not too severe it is possible for some patients to keep the disease under control by half hour periods of postural drainage night and morning, together with periods of prophylactic antibiotic therapy, and during the exacerbations of infection.[5,8] Postural drainage is carried out in a position designed to place the affected lobe above the draining bronchi, so that pus will drain by gravity from the lobe into the bronchus from when it can be expectorated.[5,8] Periods of half to one hour twice or three times daily are advisable.[5]

(c) **Surgical Treatment:** When debilitating effects of chronic infection become prominent, the anatomy should be carefully defined with Bronchography, and resection planned.[8] The aim of surgical treatment is to excise the affected segments or lobes and therefore it is essential that the disease be well localized.[5] When extensive saccular disease is

confined to one lobe or segment in a sufficiently symptomatic patient, resection is a clear choice.[8] Removal of the affected area is by pneumonectomy, lobectomy or segmental resection.[3,5] If pneumonectomy is contemplated the contralateral lung must be normal. Bilateral lobectomy or segmental resection may also be undertaken but not more than 7-8 segments should be removed.[5] Resection must be preceded by an adequate period of postural drainage and chemotherapy to reduce infection to a minimum. The results of resection for bronchiectasis are good and the patients' symptoms are greatly relieved. though if there is an associated chronic bronchitis, as is often the case, then the cough will not be abolished completely.[5,8] Care must be taken during naesthesia to prevent spillage of infected secretions into uninvolved segments in the ipsilateral lung.[8] As with operation of lung abscess, the risk of post-operative empyema is higher than for clean surgery, and considerable effort must be expanded in post-operative pulmonary toilet to assure that residual infected secretions are not allowed to pool in the bronchial stump. Surgical treatment of patients with localized bronchiectasis yields excellent results with a very low morbidity and mortality. Procrastination and prolongation of non-operative

therapy in this group of patients seem to increase the risk of morbidity.[3]

RECOMMENDATION

Bronchiectasis is a chronic disease education of the patient and family to prevent or treat acute pulmonary infections promptly is critical. The disease is decreasing in frequency because acute respiratory infections are treated more adequately.[2] Prevention of pulmonary destruction by prompt and accurate antibiotic therapy for acute pulmonary infection should eliminate the disease, except in those individuals who have underlying problems. The early identification of children with immune deficiency, alpha- 1 - antitrypsin deficiency, or broncho-ciliary deficiency should help in promptly treating pulmonary infection and in decreasing the likelihood of progression to a chronic state. In young persons with established bronchiectasis, careful counseling and vigorous control of the disease by conservative means may allow recovery in some. Careful selection of candidates for surgery should help the others.[2]

Cost Containment: The availability of medical care for everyone and excellent specific treatment of pulmonary infection can greatly decrease the frequency, debilitation, and cost of this chronic disease.[2] It is of great importance to try and

decrease the costs of caring for patients with established bronchiectasis.

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BREAST CARCINOMA OF THE FEMALE BREAST

SUMMARY

A total of 55 patients with carcinoma of the breast were seen in surgical Firm II during the period of July, 1988 to August 1990 (total of 26 months). Their ages ranged from 14 to 73 years, with mean age of 45 years. The peak age incidence was between 31 to 50 years. Thirty six cases (65%) of the patients were multiparous with an average breastfeeding period of 2 years. Majority of the patients were in the premenopausal period (29 cases) 52.7%. Quite a number of patients presented with late disease i.e. 19 patients presented with stage III disease and 16 patients stage IV. There was no correlation established between the disease and variables like, age, parity and breastfeeding. The commonest histological type was "Infiltrating ductal carcinoma".

Diagnosis before surgical treatment was made by "frozen section" studies or fine needle aspiration cytology.

Treatment was by a combination of surgery, radiotherapy and or hormonal manipulation. Hormonal manipulation was achieved either by radiation Oophorectomy, surgical oophorectomy, Tamoxifen, or prednisolone. Chemotherapy was not used because of unavailability of drugs. Three male patients with

breast cancer disease were also seen during the period under study, but were not included in this study as it was not part of this study.

INTRODUCTION

Cancer of the breast has been described for centuries. Recognition of "bulging tumors of the breast" is recorded as far back as 1600 B.C. found at Thebes Egypt.[1] Treatment by then was only by two modalities, either to burn the lesion with fire or to remove it with sharpened instruments.[1] Leonides, a Greek physician of the first century AD who worked in the great school at Alexandria, is believed to have been the first to carry out a surgical removal of the breast.[1]

By 1900, the Halsted's radical mastectomy had virtually become established as the primary method of treating all operable cancers of the breast and prevailed for the next 70 years or so.[1] Other methods slowly evolved that challenged its supremacy. These came from improvement in radiotherapy, hormone manipulation, and chemotherapy, and above all the rapid development of pathologic study played one the greatest notes in the management of the disease.[1]

Breast carcinoma is the number one cause of cancer deaths among females and has been called "the foremost cancer" in women in U.S.A.[2] An eloquent justification of this designation was given by H.

Marvin Pollard, President of the "American Cancer Society". It is the most feared of all cancers, the most frequently self discovered and the most controversially treated of all cancers.[2] It ranks first among cancers, in number of surgical procedures, in the number of hormone and chemotherapy administrations. In cancer diagnosis it is the first in the number of biopsies. It is the foremost of all cancers, too, from the standpoint of cost in physician fees and hospital bills.[2]

Breast cancer has been the focus of intensive study relative to its origins, diagnostic methods, and treatment.[2] Despite all the efforts, little ground has been gained and the age-adjusted death rate from breast cancer in females in the United States has virtually remained stable over the past 30 years, now being about 27 per 100,000.[3] It is estimated that one out of every 11 women in the U.S. will develop breast cancer and 43,000 die each year of the disease.[3] It is both ironic and tragic that a neoplasm arising in an exposed organ, readily accessible to self-examination and clinical diagnosis, continues to exert such a heavy toll.[3]

In recent years four major controversies have surfaced concerning breast cancer:

i) The relationship of treatment to the natural history of the disease.

ii) The role of mammography in diagnosis, and especially in screening programmes.

iii) The appropriate operation for treatment.

iv) The use of post-operative chemotherapy as an adjuvant to primary therapy.

CASE REPORT

S.S. IP. number 510181 a 26 year old lady was referred from Tanga, with a 3 year history of painless left breast lump. The patient noticed the swelling when she was pregnant, it did not bother her as it was painless, delivered soon thereafter and was lactating normally.

A year later she had a second pregnancy of which she had spontaneous abortion at 4 months of gestation. Throughout this period the breast swelling persisted and was gradually increasing in size. There was no history of bloody nipple discharge, no chest or bone pains, no history of fever, or weight loss. There was no known family history of breast cancer. On admission she had an amenorrhoea of 5 months, (this was her third pregnancy).

On physical examination she was found to be healthy looking, she was afebrile, had palpable left axillary lymph nodes in the central group measuring about 2 cm in diameter, non-tender discrete, mobile

and hard in consistency. Breast examination revealed the two breasts left and right to be asymmetrical the left being larger than the right one, the nipple of the left breast was lateral to the anterior axillary line, dimpling of the skin near the areolar and an obvious mass on the left upper medial quadrant, without skin ulceration. The mass was hard, non tender and irregular. It was neither fixed to the underlying muscle nor to the overlying skin and measured about 13 cm in its greatest diameter. She was normotensive with blood pressure of 120/85 mmHg and pulse of 75 beats per minute regular and full volume. She had normal chest expansion and the lungs were clear without clinical evidence of pleural effusion. Abdominal palpation revealed hepatomegally 2 cm below costal margin, firm, non-tender, smooth surface and sharp edge, and pelvic examination, revealed a uterine fundal height of about 20 weeks with free adnexa.

A clinical diagnosis of carcinoma of the breast stage T_4N_1Mx, with pregnancy was entertained. Several investigations were done before being booked for incisional biopsy and frozen section examination. The results were as follows:

1) Peripheral blood examination revealed:
   - Haemoglobin of 11.3 g/dl
   - White Blood cell count - normal distribution.

- Platelets were reported adequate.

(1) Erythrocyte sedimentation rate was 40mm in the first hour by Westergren method.

(2) Serum Alkaline phosphatase 121 iu/l

3) Bilirubin Total 12 mmol/l.

4) Serum electrolytes - Na+ 135 mmol/l.-K+ 4.5 mmol/l

5) Serum creatinine 88 mol/l

6) Chest X-ray - normal film. Skeletal survey did not reveal bone lesion.

7) Ultrasound of the liver revealed normal echogenicity no metastatic lesion seen.

The patient was then taken to theatre and the left breast was cleaned and draped with sterile towels, and under local anaesthesia an incisional biopsy was taken and sent for frozen section examination which was reported "infiltrating carcinoma". Following these results simple mastectomy with axillary lymph nodes dissection was decided upon. Patient was given general anaesthesia and left breast, chest and left axilla were cleaned with antiseptics and drapped with sterile towels. Two elliptical incisions were made enclosing the whole breast and extended to the axilla. The incisions were at least 5 cm from the tumour margins. All the subcutaneous fat was dissected from the skin

flaps, and whole breast excised from the pectoralis muscle including the pectoral fascia. Axillary dissection was done and all the fat and lymph nodes up to the level of the axillary vein were removed, en block with the breast. Haemostasis was achieved by coagulation and ligation. The skin was closed without tension with a corrugated rubber drain left in situ. Post-operatively she received two units of blood transfusion, 2 litres of Dextrose-saline in the first 24 hours and Pethidine 100 mg intramuscularly 6 hourly for 24 hours. Antibiotic cover of crystalline Penicillin 2.4 mega units intramuscularly daily for 5 days given as prophylaxis. She recovered uneventfully, the drain was removed after 48 hours and the wound healed well, the skin stitches were removed on the seventh day.

Ten days later we received the histology results of the resected specimen which reported "Papillary carcinoma", and out of the 9 lymph nodes 6 were infiltrated with tumour.

She was discussed in the tumor board were it was recommended that she should be allowed to proceed with her pregnancy and be induced at a favourable time for the baby to survive. Then to be started on Tamoxifen after delivery. The patient was discharged through the antenatal care to be re-admitted after delivery.

At 32 weeks of gestation the patient presented with severe abdominal pain, she was grossly pale, rapid pulse and low blood pressure. She was clinically suspected to have abrutio placenta and in shock. She was transfused 3 units of blood then prepared for emergency laparotomy she was found to have a septate uterus and the pregnancy was in the left horn, with a huge retroplacenta haematoma. A subtotal hysterectomy with post operative blood transfusion, antibiotics and analgesics followed by an uneventful recovery. Two weeks later she was transferred back to our surgical ward.

She was re-discussed in the tumour board and it was decided that she should receive Tamoxifen 20 mg daily as planned previously and after one week she was discharged.

Four months later she came back with chest wall recurrence which was bleeding on and off. She was transfused with 3 units of blood to correct the anaemia as her haemoglobin was only 7 g/dl and then sent for palliative radiotherapy for the bleeding. She received a total of 4,000 rads to the chest wall, and regional lymph nodes, at fractions of 200 rads per day for 20 days. Few days after completing the radiotherapy she suffered pathological fracture of the right neck of the femur. During this period the condition of the patient was too poor to withstand

internal fixation of the fracture. She was put on skin traction and was later decided to transfer her to the nearest hospital for terminal care and medical palliation. Since then there was no feedback information regarding her progress. Analysis of the 55 patients studied.

Table I: The Age incidence of the 55 patients with breast cancer

Age Group	Number of Patients	Percentagein years
11 - 20	2	0.6
21 - 30	8	14.5
31 - 40	17	30.9
41 - 50	14	25.5
51 - 60	11	20
61 and above	3	3.5
Total	55	100

Table II: Menopausal Status of 55 patients with breast cancer

Status	Number of Patients	Percentage
Premenopausal	29	52.7
Perimenopausal	6	10.9
Post-menopausal	20	36.4
Total	55	100%

52.7% of the patients were in the premenopausal status. Followed by Postmenopausal patients 36%.

Table III: Duration of Symptoms at the time of First visit to hospital in 55 patients with breast cancer

Duration of symptoms (months)	Number of patients	Percentage
0-6	21	38.2
7-12	7	12.7
13-18	9	26.4
19-24	6	10.9
25-30	3	5.5
31-36	1	1.8
37-42	2	3.6
43-48	1	1.8
49-54	0	0
55-60	2	3.6
61+	1	1.8
Duration unknown	2	3.6

Over 50% of the patients reported to hospital within one year of noting the symptoms. A number of patients ignored the first sign hoping that the lesion will disappear spontaneous.

Table V: Clinical Stage at Admission Based on TNM Classification as Modified by American Joint Committee on Cancer Staging

Stage of Disease	Number of patients	Percentage
I	5	9.1
II	15	27.3
III_A	9	16.3
III_B	10	18.1
IV	16	29.1

35 (64.5%) patients presented to the hospital with late disease i.e Stage III and IV.

Table VI: The different modalities of Treatment offered at the Different Stages of Disease

Modality of Treatment	Stage of Disease	Number of Patients	Percentage
1. Simple mastectomy axillary node dissection + Radiation + Hormonal manipulation	II-III_A	23	41.8
2. Palliative radiotherapy + Hormonal therapy	III_B-IV	11	20
3. Toilet mastectomy + Axillary node dissection	III_B	9	16.4
4. Simple mastectomy + axillary node dissection	I-II	7	12.7
5. Medical palliation	IV	5	9.1
Total		55	

Radiotherapy was used to treat remnant local disease on the chest wall or regional lymph nodes. It was also used to palliate pain and sometimes uncontrollable bleeding from the tumor.

Those patients in Stage II with negative axillary lymph nodes received 4th modality of treatment i.e. they were not irradiated, while those with positive axillary lymph nodes received the 1st modality of treatment.

DISCUSSION

INCIDENCE

Carcinoma of the breast in prepubertal females is extremely rare and with fewer than 25 well documented examples in the literature, its occurrence in the first decade is even rarer. The first Bantu case was reported in 1982 by Philip P.J. and Luande G.J. a carcinoma of the breast in a 6 year old girl which started at the age of 3 years.[4]

Worldwide figures show that 541,000 cases of breast cancer were diagnosed in 1975 and that the number may approach one million by the year 2,000.[5] The Dutch have the highest national mortality of cancer of the breast, with reliable statistics, with an incidence of 3.76 per 100,000 population. The factors leading to this wide range of incidence are

unknown, although studies seeking an answer are in progress.[5]

EPIDEMIOLOGY

Few cancers including breast cancer have been subjected to intensive epidemiologic study. A host of observations has emerged such as striking geographic or racial differences in its prevalence.[2]

Geographical influences: It is five times more common in the United States than in Japan and Taiwan. Sex is certainly an important contributing factor in this disease. As it is rare in males. Being a male is not a complete protection however, there is one carcinoma of the male breast for every one hundred female breast carcinomas.[5]

Genetic predisposition The magnitude of risk is in proportion to number of close relatives with breast cancer and age at which cancer occurred in relatives. The younger the relatives at time of developing cancer and the more bilateral cancers, the greater the genetic predisposition. There are uncommon high-risk families with apparent autosomal dorminant transmission and familial association of breast and ovarian carcinoma.[3,6]

Age: Breast cancer is uncommon before age 20, but then a steady rise to the time of menopause, followed by a slower rise throughout life.[3]

Length of reproductive life: The risk of developing breast cancer is said to increase with early menarche and late menopause.[3]

Parity: Breast carcinoma is more frequent in nulliparous than in multiparous women in this study multiparity did not seem to be protective. Out of the 55 patients 36 (65.5%) were multiparous and 17 (30.9%) nulliparous.

Obesity: Increased risk of breast cancer is attributed to synthesis of Oestrogens in fat keeping obese female at a higher risk.[3]

Exogenous Oestrogens: This is still controversial, but some data show moderately increased risk with high dosage therapy of oestrogens for menopausal symptoms.[3]

There is no clear cut increase risk; attributed to balanced content of oestrogens and progestins in currently used oral contraceptives.[3]

AETIOLOGY AND PATHOGENESIS

Despite extensive studies the causative factors of breast cancer are still unknown.[3] However epidemiologic data and extensive studies of mammary tumors in mice point to three sets of influence which might be important in breast cancer aetiology.

1) Genetic factors
2) Hormonal imbalances
3) Environmental factors

In animals, viruses can clearly interact with these influences to cause cancer, but no such viral aetiology has yet been documented in humans in relation to breast cancer.

Genetic predisposition: It has been shown that there is a markedly increased risk in first-degree relative of breast cancer patients. In mice, high cancer strains can be achieved by genetic in breeding. But how these factors operate to induce cancer is still unknown.[3] Hormonal imbalances: Endogenous estrogen excess is thought to pay a significant role, many of the risk factors mentioned above, like long duration of reproductive life, nulliparity and late age at first child, all imply increased exposure to estrogen peaks during the menstrual cycle. Functioning ovarian tumors that elaborate oestrogens are associated with breast cancer in post menopausal women.[3].

In this study 36 patients i.e. over 65% of the patients were multiparous and all had breastfed their children at least at an average period of two years. Recent studies have confirmed associations among excessive urinary oestrogens, frequency of ovulation, early age of menarche, and increased breast cancer risk.[3]

Environmental influences: The effect of diet is controversial, but there are ardent advocates of the

thesis that high dietary fat constitutes a significant predisposition.

Moderate alcohol consumption is also associated with a 1.5 increased risk of breast cancer.[3]

The role of viruses in human breast cancer, has been pursued since Bitner's brilliant discovery in 1936 that a filterable agent, transmitted through the mother milk, causes breast cancer in suckling mice.[3] The virus, called mouse mammary tumor virus (MMTV) was later identified as a retrovirus. Subsequently there were many hints of the existence of an analogous virus in breast cancer of humans but the findings have not been conclusive. There are also unconfirmed studies reporting the presence of reverse transcriptase or particles with properties of retroviruses in cancerous breast tissue.[3]

NATURAL HISTORY AND PATHOLOGY OF BREAST CANCER

A typical carcinoma of the breast is a scirrhous adenocarcinoma beginning in the upper outer quadrant (40 to 50%), it grows slowly, doubling its volume every 2 to 9 months in 70% of patients. Starting from a single cell, it takes 30 doubling times for a tumor to attain a size of 1 cm diameter, the smallest tumor of the breast normally found on physical examination. Thus even the fastest growing tumor of the common type may require 5 years before it becomes

clinically palpable. The use of doubling times to calculate the preclinical course of tumors of the breast is subject to many errors, the most obvious being that growth rates are not always constant, varying with areas of necrosis within the tumor and hormonal changes in the patient.[5]

Laboratory and clinical observations indicate, however, that growth rates are more consistent than is usually appreciated; especially during the first 30 doublings.[5] The concept of the origin of these tumors in a single cell with increase in size by doubling is a useful model and suggests the long occult period that probably is present in many tumors before they are diagnosed.

A characteristic of malignant cells is the lack of adhehesion to adjacent tissue. As the small mass of tumor increases in size, increasing numbers of tumor cells shed into the intercellular spaces to be taken up by the lymphatics. At about the twentieth doubling, then still tiny tumor cells can now be shed directly into the blood stream.

Fisher as reported by Rush noted that the cells entering the lymphatics network can cross over into the bloodstream by lymphaticovenous communications.[5] Successful implantation of shed cells is another matter, however. Depending on the number of cells shed, special characteristics of the shed cells, and

the resistance of the host, successful implantation of metastatic cells from breast cancer rarely occurs until the primary lesion is larger than 0.5 cm in diameter, or about the twenty seventh doubling. Fisher has also introduced the concept that the appearance of gross tumor in the axillary node is an index of the failure of host resistance and that such nodal involvement indicates a probability of disseminated malignant disease. The chance of dissemination is roughly correlated with the number of nodes involved.[5]

As the tumor increases in size and invades the surrounding glandular tissue, the accompanying fibrosis tends to shorten Cooper's ligaments, producing the characteristic dimpling of the skin. Cords of tumor cells grow out along lymphatics, ultimately invading the skin itself. This invasion is preceeded by localized edema of the skin as many lymphatics are blocked and drainage of fluid from the skin is impeded. Eventually, tumor cells replace the skin, which breaks down to form an ulcer or an ulcerating mass. The tumor increases in size and new areas of skin invasion may occur, seen as small satellite nodules adjacent to the ulcer crater. Blood vessels are invaded, and additional tumor cells seed into the circulation, passing into axillary or intercostal veins to be scattered through the

pulmonary circuit into the lungs or by way of the vertebral veins up and down the vertebral column.[5]

As the breast tumor extends toward the skin, tumor cells simultaneously pass along the lymphatic vessels from the upper outer quadrant to the axillary nodes, where they implant and grow. As the axillary nodes enlarge, they are at first fairly soft, then firm and hard as they are increasingly replaced by tumor. Eventually the nodes adhere to one another in a large conglomerate mass, and as the tumor breaks out of the lymphatic capsule the mass of nodes become choked with tumor, cells are passed along the chain to the supraclavicular nodes, which also enlarge. Other cancer cells pass by way of the right lymphatic trunk or the thoracic duct into the blood stream, heart, and lungs, systemic spread is the rule, and 95% of patients who die of uncontrolled breast cancer have distant metastases. Lung (65%) liver (56%) and bones (56%) are the commonest sites for these deposits.[5]

DIAGNOSIS

Cancers of the breast are usually first discovered by the patient or physician as a solitory, painless mass in the breast.[3] The older the patient with a single breast mass, the more likely is it to be cancer. When first discovered, these cancers are deceptively

freely movable and delimited. Many cancers are first detected by the patient during self-examination. On the average, these lesions are 4 cm in diameter when first found and approximately two thirds have already spread to axillary or other nodes. Intraductal cancers, which rarely produce palpable mass, more often come to attention by the appearance of a discharge which is sometimes haemorrhagic from the nipple.[3]

The normal breast is a nodular structure by virtue of its lobular architecture, and this lobularity may be accentuated during the later portion of the menstrual cycle, pregnancy, or lactation.[5] To the inexperienced examiner the normal nodularity may feel faintly suspicious throughout, although no obvious lesion can be felt. palpation of the typical cancer of the breast normally leaves little doubt in the examiners mind. the lesion is hard, almost cartilaginous; the edges are distinct, serrated and irregular.[5] This is true for the 75 to 80% of breast tumors associated with productive fibrosis. It is the remaining 20-25% of tumors, those associated with little fibrosis and those with a medullary or a colloid element or other less typical lesions, which form the spectrum of neoplasm most difficult to distinguish from benign lesions.[5] Other late stage signs include skin dimpling or

nipple retraction, satellite skin lesions, edema or ulceration and ipsilateral enlarged axillary or supraclavicular nodes involvement, signs of even wider spread may be present such as a history of back or leg pain an enlarged and nodular liver, or perhaps or complaint of dyspnoea associated with physical findings of fluid in the chest.[5]

Since early treatment of localized disease is the best hope of total eradication, enormous effort is being made to identify breast cancers at an early stage.[3] Currently, emphasis is being placed on more frequent, regular medical examinations and mammograpy, Xeroradiography and thermography as screening techniques.[3]

The argument for and against breast screening are well known, but evidence suggests that it may save lives as reported by Reidy and Haskins 1988.[7]

Trials done in Sweden showed a mortality reduction of more than 30%, but there was no benefit under the age 50.[7] For screening to succeed it requires compliance of women and expertise, commitment and enthusiasm of those attending them.[7] The use of screening mammography has increased dramatically, leading to the discovery of suspicious, non-palpable mammary lesion.[8] Mammographic wire localization (MWL) is currently being used to facilitate the biopsy of these lesions.[8]

With the current emphasis on the use of mammography in screening for carcinoma of the breast, clinically occult mammary lesions, identified only by mammography have assumed increasing importance.[8] In 1973, non palpable cancers of the breast were shown to be associated with a survival rate superior to that of palpable cancers of the breast and to have a lower rate of axillary nodal metastases.[8]

The American cancer society currently recommends that all women begin breast self-examination after age of twenty,[5] that they obtain a "base line" mammographic screening thereafter. This fairly cautious recommendation may be extended even further for examinations in the forty-to fifty age group in the light of new findings that radiation exposure involves a risk is virtually zero in the cancer age group.[5] Prospective randomized studies of routine mammographic screening have shown 40% reduction of Stage II and greater cancers in the screened group and a 30% increase in survival in those patients found to have cancer.[5]

Xeroradiography is basically similar to radiographic technique as mammographic, only differs in that image is positive with xeroradiography whereas mammography is negative.[5] One disadvantage of xeroradiography is that in case of a badly exposed Xeroradiography, too poor for proper interpretation, is harder to

recognise than a poor radiomammograph and may fool the unwary physician.[5] It has an advantage in that in a well taken radiograph the image is easier to read.

Thermography The skin over malignant tumors of the breast is usually warmer than the surrounding areas. using special heat scanners it is possible to delineate these "hot spots" on film. Infection may be associated with false positive results.

ULTRASONOGRAPHY

Used in diagnosis of breast lesion is highly effective in differentiating a cystic from a solid mass.[5] It is unsatisfactory for detecting early breast cancers owing to the inability to visualize microcalcification.[5] While ultrasonography is an excellent method for diagnosing a cyst, needle aspiration is much simpler and less expensive.

MRI Magnetic Resonance Imaging and Digital subtraction angiography in breast lesions, are more recent techniques.

Isotope Scanning is also used to detect metastases in the liver.[9]

Breast Biopsy: This is a procedure with little risk and can be done under local anaesthesia. The non-palpable lesion discovered by mammography presents a special problem for the surgeon. Such lesions must be marked before the procedure so that the operator

can be sure that the proper tissue is removed.[5] Marking is done by injection of a dye or insertion of a wise guide under x-ray control.[5]

Needle biopsy of breast masses is done in many institutions and is very satisfactory if the pathologist is familiar with this type of material. Negative needle biopsies have no significance, however, since an adequate sample may not be obtained.[5] These biopsies were mainly done with a trocar like needle such as a Vim-silver man or "True-cut" that yielded a core of tissue that could be fixed and mounted. Currently biopsy with a thin or "skinny" needle, usually 20 to 22 gauge, is gaining popularity. The thin needle is thrust several times through the tumor while suction is exerted on the attached syringe.[5] The material obtained is spread on a slide and fixed. This is a cytologic rather than a histologic technique. Cancer cells are much less adherent to adjacent tissue than normal cells, and the thin needle seems to dislodge them preferentially so that the smaller specimens obtained yielded equivalent and perhaps superior results to a trocar. In addition, patients find the thin needle a less painful and more tolerable method.[5]

It was the general custom to do the biopsy under general anaesthesia and proceed to a definitive cancer operation if the frozen section of the lesion

indicated malignancy. However there is an increasing trend to do the biopsy under local anaesthesia, often in an outpatient setting, and to continue with the larger operation if indicated, a few days later. This delay in definitive operation has no effect on the recurrence or survival rates.[5] A curvilinear incision is made when the lesion is..... small total excision is preferred, but if a large lesion is encountered, a small incisional biopsy of the main mass is done.[5]

STAGING

At present the only acceptable method of classification is a standard system devised by the American Joint Committee on Cancer Staging and End Results Reporting based on a system proposed by the International Union against Cancer in 1958. This system of classification is called the TNM Classification, the T standing for local or primary tumor the N for regional lymph nodes, and the M for distant Metastases. The system is very precise but also complex. This is the staging system which was also used in this study. The staging is as follows:

Primary Tumor (T)

T_X = Tumor cannot be assessed

To = No evidence of primary tumor

Tis = Paget's disease of the nipple with no

T_1 = Tumor 2 cm or less in greatest dimension

T_1a = No fixation to underlying pectoral fascia or muscle (T_1a; Tumor Size < 0.5cm).

T_1b = Fixation to underlying pectoral fascia or muscle (T_1b; Tumor Size > 0.5cm - 1cm) (T_1c; Tumor > 1-2

T_2 = Tumor more than 2 cm but not more than 5 cm in its greatest dimension.

T_2a = No fixation to underlying pectoral fascia or muscle

T_2b = Fixation to underlying pectoral fascia or muscle

T_3 = Tumor more than 5 cm in its greatest dimension

T_3a = No fixation to underlying pectoral fascia or muscle

T_3b = Fixation to underlying pectoral fascia and/or muscle

T_4 = Tumor of any size with direct extension to chest wall or skin

T_4a = Fixation to chest wall

T_4b = Edema (including peau d'orange), ulcer of the skin of the breast, or satellite skin nodules confined to the same breast.

T_4c = Both of the above.

T_4d = Inflammatory Ca

LYMPH NODES (N)

Nx = Regional lymph nodes cannot be assessed clinically

No = homolateral axillary lymph nodes not considered to contain growth.

N_1 = Movable homolateral axillary nodes considered to contain growth.

N_2 = Homolateral axillary nodes considered to contain growth and fixed to one another or to other structures.

N_3 = Homolateral supraclavicular or infraclavicular nodes considered to contain growth or edema of the arm.

DISTANT METASTASES (M)

Mx = Not assessed

Mo = No (known) distant metastasis

M_1 = Distant metastasis present

STAGING ON THE BASIS OF TNM CLASSIFICATION IN COMPARISON

STAGE I = $T_1^{N}{}_oM_o$

STAGE II = $T_{1-2}N_1M_o$ $T_1N_1M_o, T_2N_1M_o, T_2N_oM_o$ $T_2N_{0-1}M_o$

STAGE III_A = $T_{1-3}N_2M_o$ $T_1N_2M_o, T_2N_2M_o$ $T_3N_{0-2}M_o$

$T_3N_oM_o$, $T_3N_1M_o$, $T_3N_2M_o$

STAGE III_B = $T_{1-4}N_3M_o$ Any T_4 with Mo or N_3 with any

$T_4N_{0-3}M_o$

Stage IV = M_1 Disease

HISTOLOGICAL CLASSIFICATION OF CANCER OF THE BREAST ADOPTED FROM THE WORLD HEALTH ORGANIZATION

A. Non-invasive (non-infiltrating)

1. Intraductal carcinoma
2. Intraductal papillary carcinoma
3. Lobular carcinoma in situ (3)

B. Invasive

1. Invasive ductal carcinoma - not otherwise specified
2. Invasive lobular carcinoma
3. Medullary carcinoma
4. Colloid carcinoma (mucinous carcinoma)
5. Pagets disease
6. Tubular carcinoma
7. Adenoid cystic carcinoma
8. Invasive comedocarcinoma
9. Apocrine carcinoma
10. Invasive papillary carcinomas (3)

NONINVASIVE (IN SITU) CARCINOMA

DCIS Intraductal carcinoma - Over 90% of breast carcinomas arise within the ducts.[3] It begins as an atypical proliferation of ductal epithelium that eventually completely fills and plugs the ducts with neoplastic cells. Grossly, the tumor is usually or poorly defined focus of slightly increased consistency caused by the marked dilatation and solidification of the ducts.[3] When the breast is sectioned cord like ducts are found filled with necrotic and cheesy tumorous tissue. This substance can be readily extruded upon slight pressure, hence the designation comedocarcinoma. Histologically, the ducts are dilated and filled with neoplastic anaplastic epithelial cells that plug the lumina;

sometimes there is central necrosis. The growth pattern is sometimes cribriform, indicating the presence of duct like structures within the primary dilated ducts.[3] Rarely, these intraductal carcinoma have a predominantly papillary pattern and are called intraductal papillary carcinomas.

LOBULAR CARCINOMAS IN SITU

This is a histologically unique lesion manifested by proliferation, in one or more terminal ducts, of cells that are loosely cohesive, are somewhat larger than normal, and have rare mitoses and oval or round nuclei with small nucleoli.[3] These lesions can be seen in breasts removed for fibrocystic disease, in the vicinity of invasive carcinoma, or admixed with the foci of intraductal carcinoma.[3] The frequency with which such change may develop into invasive carcinoma.

INFILTRATING (INVASIVE) DUCT CARCINOMA

This is the most common type of breast cancer. Exhibit marked increases in dense fibrous tissue stroma, giving the tumor a hard consistency(scirrhous carcinoma). These growths occur as fairly sharply delimited nodules of stony hard consistency that average 2 cm in diameter and rarely exceed 4 to 5 cm. On palpation, they may have an infiltrative attachment to the surrounding structures with fixation to the underlying chest wall, dimpling of

the skin, and retraction of the nipple. The mass is quite characteristic on cut section. It is retracted below/ the cut surface, has a hard cartilaginous consistency, and produces a grating sound when scraped.[3] Within the central focus, there are small pinpoint foci or streaks of chalky-white, necrotic tumor and small foci of calcification infiltrating duct carcinomas, even in the presence of axillary lymph node metastases. The ten-year survival rate for these tumors is 70 to 90%.

COLLOID OR MUCINOUS CARCINOMA

This tumour tends to occur in older women and grows slowly over the course of may years, producing large, gelatinous masses. The tumor is extremely soft and has the consistency and appearances of pale gray-blue gelatin. It may occur in pure form or in association with other types.

Histologically this tumor usually takes on one or two patterns of growth, which may coexist in a single lesion. There may be large lakes of lightly staining, amorphous mucus that dissect and extend into contiguous tissue spaces and planes of cleavage. Floating within this mucin are small islands and isolated neoplastic cells, sometimes forming glands. Vacuolation of at least some of the cells is characteristic.[3]

The survival rate is greater in pure colloid carcinoma than in the usual infiltrating duct carcinoma, and lymph node metastases are infrequent.

PAGET'S DISEASE

Pagets disease of the nipple is a specialized form of ductal carcinoma that arises in the main excretory ducts of the breast and extends to involve the skin of the nipple and areolar. Careful morphologic study of these lesions has demonstrated beyond doubt that ductal carcinoma, with or without invasion, invariably antidotes the skin change. Since Paget's disease of the nipple implies extension to the skin, the prognosis is somewhat less favourable than in the simple noninvasive ductal carcinoma. About 30 to 40% of these women have metastases at the time of surgery.

The histologic hallmark of this entity is the involvement of the epidermis by malignant cells, referred to as pagets cells.[3]

LOBULAR CARCINOMA

This accounts for about 5-10% of breast carcinomas. Lobular carcinomas are of particular interest for at least two reasons:

(i) They tend to be bilateral more frequently than those arising in ducts (the likelihood of cancer in the contralateral breast being on the order of 20%).

(ii) They tend to be multicentric within the same breast.

Grossly, the tumor is rubbery and poorly circumscribed, but sometimes appear as a typical scirrhous type. Histologically, it consists of strands of infiltrating tumor cells, often only one cell in width (in the form of an "Indian file" loosely dispersed throughout the fibrous matrix.[3]) Differentiation between ductal and lobular infiltrating carcinoma may very often be quite difficult and in some tumors mixed ductal and lobular patterns, as well as intermediate cancers, exist indeed, most duct carcinomas probably arise also from the terminal ductules.

TREATMENT

Breast cancer is the most controversially treated of all cancers. It ranks first among cancers in the number of surgical procedures, radiation therapy treatment as well as hormonal manipulation and chemotherapy administrations.

The treatment of breast cancer is determined primarily by the stage of the disease.[9] In clinical stage I and II, where surgery plays a major role, eradication of local disease can be curative, especially when the axillary nodes are shown to be histologically free. In stage III the local extent of disease usually rules out the possibility of

curative surgery although blood borne metastases are not yet detectable. The low survival figures, indicate that occult deposits are almost always present and even when local treatment is offered, systemic treatment should be recommended.[9]

In stage IV, cancer is widespread: systemic treatment must be considered though the palliative value of locally directed therapy to control disease in the breast, regional nodes, and isolated bone metastases must not be overlooked.[9]

A locally advanced breast cancer remains a challenging disease for current treatment strategies.[10] Management by surgery alone or radiotherapy are associated with a high incidence of early relapse and a poor survival rate 50% of the patients being dead after 2-3 years and even more in case of inflammatory cancer disease.[10] The locoregional failure rate is in the range of 50 to 70 percent at 5 years and a high potential for early dissemination is reflected in the pattern of first relapse which shows involvement of distant areas in almost 70% of the cases. The introduction of systemic therapy (chemotherapy) as part of the primary management of this disease has improved prognosis over conventional treatment with radiotherapy alone.[10]

OPERABLE BREAST CANCER (STAGE I AND II)

Considerable, argument and confusion exists regarding the management of disease at these potentially curable stages. Some authorities advocates surgical operations of varying magnitude, some irradiation and others a combination of surgery and irradiation. Almost all our patients in this study had combination of surgery, radiotherapy and hormonal manipulation.

This continuous persistence of this controversy suggests that there is probably more than one correct method of management. It is essential therefore to have a clear concept of the aim of treatment which are:

(a) To ensure long-term control of disease locally and in the lymph node areas.

(b) To conserve local form and function

(c) To prevent, if possible, the evolution of these occult metastases known to be present in a proportion of patients, no matter how thorough the staging.[9]

THE SURGICAL TREATMENT

1. **Radical mastectomy:** As designed by Halsted. In this procedure the breast and associated structures are dissected en bloc and the excised mass is composed of:

(i) The whole breast, together with the skin overlying the tumour [9,11]

(ii) The entire system of lymphatic glands in the axilla, together with channels which connect them with the breast. These can be removed only by complete clearance of the axillary fat.[9]

The sternocostal part of pectoralis major and its fascial sheath the whole of Pectoralis minor and its fascial sheath, the upper part of the aponeurosis of external oblique, the anterior digitations of serratus anterior, the subscapularis, the exposed portion of the latissimus dorsi, and the upper part of the rectus abdominis. If the growth is situated near the upper margin of the breast the whole of Pectoralis major should be sacrificed.[9,11]

During the operation every effort should be made to preserve:

a) The axillary vein

b) The Cephalic vein

c) The long thoracic nerve (nerve to serratus anterior).

The middle or long subscapular nerve (nerve to latissimus) can be sacrificed without ill effect, and this should be done without hesitation if its division enables a more thorough dissection to be carried out.[9,11]

At the completion of the operation, the wound is drained, preferably using widebore suction tubes.

If a wide area of skin has been sacrificed, it may not be possible to approximate the skin edges completely. A deficiency is left which is treated by immediate or subsequent skin grafting. After operation the arm is supported upon a pillow until the wound has healed, when early movement of the arm is encouraged. The movements of the arm after so extensive surgery of loss of muscle is surprisingly good.[9]

2. **Modified radical mastectomy**

In this operation, as practiced by Patey, Handley and Auchiviclass, the pectoralis major is not sacrificed, but is widely retracted and the pectoralis minor removed. This gives adequate exposure to allow the axillary contents to be cleared as completely as in the Halsted operation. Its main advantage is cosmetic, in that the axillary fold is maintained and there is no hollowing below the clavicle.[11] When there is a deficiency of skin, it provides an ideal bed of muscle on which skin grafts will take very readily. Lastly, it gives a stronger and more useful arm.[11]

SIMPLE MASTECTOMY

This means complete removal of the breast but the axilla is left undisturbed except for the region

of the axillary tail which usually has attached to it a few nodes low in the anterior group. This operation controls the primary site and is adequate for regional control provided the axillary nodes contain no deposits.

In this study most of the patients (52.7%) 29 cases, had simple mastectomy done, but the procedure was combined with axillary node dissection. Only in advanced cases where toilet mastectomy was done to relieve the patients of a fungating smelling ulcer.

SUBCUTANEOUS MASTECTOMY

In subcutaneous mastectomy most of the mammary tissue is removed but the skin of the breast is preserved. The breast contour is restored by inserting a silastic prosthesis into the subcutaneous pocket left by the excision of the breast tissue or now, more commonly, under the pectoralis major muscle.[5] It is impossible to remove all breast tissue by this method, and 1 or 2 percent remains in the subcutaneous layer near the skin even if the nipple is sacrificed. Development of cancer in this residual mammary tissue has been reported, and in view of the multifocal nature of breast cancer, it is expected that some of the foci may be left behind.[5] The risk benefit ratio of this approach remains to be established, and while it is a very attractive alternative for women with in situ cancer of the

breast, the accepted procedure for such lesions in the United States is still total mastectomy or possibly radiation therapy.[5]

SYSTEMIC THERAPY

One of the most important advances in oncology during the past decade has been the acceptance of evidence indicating that most, if not all, patients with primary breast cancer have disseminated disease and that improvement in survival will result only from the use of effective systemic therapy in conjunction with operation.[12] Systemic therapy may be endocrine or cytotoxic. It is indicated when disease is widespread.

ENDOCRINE THERAPY

Some breast cancers respond to manipulation of their hormonal milieu by the administration of hormones or by ablation of endocrine glands.[9] They are said to be hormone dependent and others are hormone-independent.[9] The hormone control of breast cancer is exceedingly complex.[11] About 30-40% of breast cancers are hormone-dependent in that arrest of growth or regression may be obtained by altering the patients hormone balance.[9,11,13] The most striking effects are relief of pain, improvement in nutrition, and regression of local lesions.[9,11] Thus osteolytic lesions may calcify and pathological fractures heal.[11] The effects, however, are only

temporary since, after a varying interval the tumor cells lose their hormone dependence, and thereafter the disease progresses rapidly to a fatal termination.[9,11]

About 60 - 70% of breast carcinomas are composed of cells which require no hormonal stimulus to enable them to thrive, multiply and metastasize. Alteration of the hormonal status has an insignificant effect and usually no effect at all on tumor growth.[9]

MAJOR ENDOCRINE ABLATION

After the synthesis of cortisone in the early 1950's it became possible to undertake the total operation of bilateral total adrenalectomy.[9] This removed an important source of extra ovarian oestrogen and was a valuable method of treatment for hormone dependent breast cancer in the post-menopausal patient or in premenopausal women.[9] The adrenal were removed either via bilateral loin incisions or using the anterior route through a long vertical or transverse incision.[9]

Oophorectomy This was the first treatment designed to effect the endocrine status of patients with advanced carcinoma of the breast. It was first described in 1896 by Sir George Beatson, of the Western Infirmary in Beatson, who published an account of two cases treated in this manner.[9] Oophorectomy or ovarian irradiation is still used

extensively as a treatment of recurrent or advanced breast cancer and is beneficial in approximately 20-30% of cases.[9] It is of value only before the menopause and the remissions produced can last up to two years or longer.[9] Remission of the disease following removal or irradiation of the ovaries implies that the tumor is sensitive to hormone deprivation, and that, when pre-activation of growth occurs, adrenalectomy or hypophysectomy may be beneficial and this is so in about half of the patients.[11] Failure of response to Oophorectomy suggests that further endocrine ablation is unlikely to be rewarding.[11]

ANDROGENS The administration of androgens probably accomplishes the same as an Oophorectomy although it could be argued that the former would also counteract the adrenal secretion of estrogen. The remission rate and duration of remission are similar for the two forms of treatment.[9] Administration is usually by injection, either in the form of testosterone propionate, 100 mg three times a week intramuscularly or one of the anabolic steroids, such as Durabolin 50 mg once a week intramuscularly. Androgens can be administered to all age groups but are usually considered the treatment of choice for patients who are premenopausal or within five years of the menopause.[9]

OESTROGENS

The use of oestogens may seem contradictory to the theories of hormone responsiveness, it is an effective treatment.[9] Oestrogens are given only to post menopausal women, and should be more than five years post menopausal. The response to the drug increases with age and may reach 60% in women over seventy. Overall the remission rate is approximately 40%. Fluid retention and persistent vaginal bleeding may necessitate stopping the drug.[9] Stilboestral is the most commonly used agent, dose 1.0 mg three times a day. There is no proof of increased benefit from higher doses sometimes recommended.[9]

ANTIOESTROGENS

Tamoxifen acts by competition at the cellular level and appears to be effective in both pre and post-menopausal women. Studies have shown that it has a significant prolongation of disease free survival mainly in patients over 50 years of age, with at least less than 4 positive nodes.[14] It has few significant side effects and is considered the agent of first choice. The usual dose is 20 mg daily.

Since the introduction of Tamoxifen, androgens and oestrogens are much less often used; apart from the side effects mentioned they may also occasionally

cause provocation of the disease, the most dangerous manifestation being acute hypercalcemia.

However, the agonistic and antagonistic oestrogen properties of Tamoxifen are not fully known and may have consequences for morbidity in patients treated with the substance for several years.[5] There is little information on late side effects including the frequency of secondary malignant tumors. Oestrogens have been associated with the development of some types of cancer e.g. breast endometrial and hepatic cancer.[15]

The increased frequency of endometrial cancer limits the usefulness of Tamoxifen for benign indications. On the other hand in early breast cancer there is improvement of recurrence-free survival with adjuvant Tamoxifen and probably also an overall survival benefit.[15] Furthermore, endometrial cancers usually have a good prognosis.[15] The clinical benefit with Tamoxifen probably outweighs the increased frequency of endometrial cancer.[15]

The role of hormonal receptors in predicting the effect of adjuvant endocrine therapy is debatable. Although in postmenopausal women with advanced breast cancer the highest response rates to Tamoxifen are achieved, in patients with the highest oestrogen concentration the results from adjuvant trials that have investigated this question are equivocal.[15]

that is probably attributable to a direct correlation with metastatic adenopathy and lymph node metastases (0,1 to 3 and 4 or more), as well as by the actual drugs and hormonal therapy used.[6]

None of the single alkylating agents is recommended for therapy today, because the long term salvage is unsatisfactory and better treatments have been developed. The significant better effects of combination chemotherapy on survival without disease justify the extracaution necessary in its administration and the side effects encountered.[16]

CMF (Cyclophasphamide, Methotrexate and Fluorouracil showed significant superiority over surgery alone in premenopausal women with one to three nodes or four or more nodes.[16] This superiority was apparent not only in disease free interval but also in survival. Local recurrence was sharply reduced in the chemotherapy group thereby eliminating this manifestation as an indication for regional radiotherapy. Other combination chemotherapy which have been tried is: CMFVP regimen (Cyclophasphamide, methotrexate, 5 Fluorouracil, Vincristine and L-PAM). The CMFVP which is a five drug combination therapy showed a superior efficacy to that of CMF in advanced disease.[14]

CMFVP is significantly superior to CMF in disease free survival and in overall survival

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CMFVP is significantly superior to CMF in disease free survival and in overall survival

cancer.[17] Doxorubicin-containing combinations have produced higher response rates and longer response duration in many although not all, reported trials; while acute toxicity is manageable many oncologist believe that the most serious toxic effect of this drug and the one limiting its use for extended periods is the appearance of cardiomyopathy.[17] After reaching a total cumulative dose of 500 mg/M^2 additional treatment with doxorubicin produces a rapidly increasing incidence of clinically significant and often fatal, cardiomyopathy. Several studies have shown that administration of doxorubicin at lower weekly doses, or by slow intravenous infusion, reduces considerably the incidence and severity of cardiomyopathy.[17]

RADIOTHERAPY

The development of supervoltage radiation has made it possible to give effective doses as well as the skin sparing required for satisfactory cosmetic results.[13] The discovery of radium has made it possible to develop iridium implants that can be used for a booster dose to the tumor area rather than external radiation.[13]

In trying to evaluate the efficacy and safety of primary radiotherapy and to determine whether it is a justifiable alternative to selective mastectomies and

restorative procedures, a number of things have to be considered including:

1. At least one third of breast cancer are radioresistant, raising the question of the effectiveness of radiation in such cases.[13]

2. Radiation therapy must be preceeded by some type of local tumor surgery. Tumor persistence or recurrence is very high when only biopsy without tumor removal is done, being reported as 30-40% by Bataini et al and even higher by Calle, et al as quoted by Leis et al had a rate of 55% for cancers larger than 3 cm or with clinically positive nodes.[13]

Local recurrence rates are directly proportional to the extent of excision, being lowest with partial or segmental mastectomies and highest with limited excisions.[13] Increasing tumor size requires larger excision, more radiation, and results in a less attractive cosmetic appearance.[13]

4. Radiation administration is time consuming, requiring daily treatment for five days a week for a approximately five and a half weeks not including the tumor area booster dose by external radiation or iridium implant. It has to be given in a centre with personnel highly skilled in the technical management of radiation and with dedicated equipment. It requires a large dose of radiation about 5000 rads to

the breast and regional nodes and a tumor area booster dose of about 1500-2000 rads.[13] The cost, including the tumor excision and axillary dissection is equivalent to that of a modified mastectomy and reconstruction.[13] In additional, a considerable number of patients have to bear the additional cost of a salvage mastectomy because of radiation failure.[13]

Early complications related to radiation are less frequent with supervoltage and improved techniques but they still occur, including atrophy and shrinking, fibrotic bands and thickening, pneumonitis supraclavicular thickening, telangiectasis and pigment changes, dermatitis and ulcers, edema of the breast and arm, chest wall and arm pain, and various types of bone and joint damage, especially rib fractures.[13] The frequency and severity of complications vary with different reports and relate to the extent of local tumor surgery and axillary gland dissection proceeding the radiation and to the radiation dosage and technique used.[13]

CONCLUSION

Despite all treatment studies, breast cancer is the most feared cancer, the most frequently self discovered and the most controversially treated of all cancers. What has been achieved so far is not very encouraging. Since screening of at risk women

has shown good results as it helps in early detection and treatment for better prognosis. This means that all suspicious lesions in the breast must be considered as possible cancer until proven otherwise. On this basis the consensus would favour immediate biopsy and investigations of all such lesions. This alarmist attitude may result in a certain number of "unnecessary" surgical procedures but in the long run the greatest good will be done for most patients.

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RUPTURED SPLEEN

SUMMARY

During the period July 1988 to August 1990 a total of 26 months, 22 patients were admitted in surgical firm II due to traumatic rupture of the spleen. There were twenty males and two females in the age range of 16 years to 59 years.

The causes of trauma were motor traffic accident in 14 patients, three other patients due to assault, and one was attacked by a bull.

One patient was referred to our hospital ten (10) days after injury. The rest were referred on the same day of the injury.

Diagnosis was based on history of abdominal trauma, associated with tenderness, pale mucous membranes and conjuctiva, shifting dullness and haemoperitoneum.

All patients were treated by splenectomy. Out of the 22 patients 2 died due to irreversible hypovolemic shock, and these patients had suffered multiple injuries.

INTRODUCTION

The spleen arises by mesenchymal differentiation along the left side of the dorsal mesogastrium in the 8 mm embryo.[1] The weight of the spleen in the healthy adult weights an average of 200 grams, decreasing somewhat with age.[1] The organ is located

in the left upper quadrant, having a superior relationship to the under surface of the left leaf of the diaphragm and protected anteriorly, laterally, and posteriorly by the lower portion of the rib cage. Its position is maintained by several suspensory ligaments, the major ones being the splenophrenic, splenorenal, splenocolic and gastrosplenic ligaments. The gastrosplenic ligament normally contains the short gastric vessels, while the remaining ligaments are generally avascular, except in patients with portal hypertension when collateral veins become apparent.

For more than 2500 years knowledge of the biologic functions of the spleen has been elusive.[2] It has long been known, however, that removal of the spleen is compatible with life.

The Asclepiad, Galen of Pergamon (130-200 A.D.) described the spleen as an organ of mystery (Mysterii pleniorganon.) Aristatle(384-322 B.C.) assumed that the spleen was not necessary for life.[2]

In 1919 Morris and Bullock as quoted by Sheldon et al performed the first experimental studies in animals that suggested an essential role for the spleen in host defense. Their conclusion based on experiments in splenectomised rats infected with the bacillus of rat plague, was that "the spleen aids tremendously in resisting infectious processes and

its removal temporarily robs the body of its resistance.[2] The first major challenge to the concept of the dispensable nature of the spleen was King and Shumacker's (1952) as quoted by Sheldon et al who reported an increased susceptibility to infection in infants following splenectomy.[2]

The first recorded splenectomy was performed in 1549 by Adrian Zaccarelli. Numerous partial and total splenectomies were performed as surgery became established as a speciality in the nineteenth and twentieth centuries.

Billroth 1818, as quoted by Sheldon et al postulated that the spleen was capable of repair after injury, a conclusion based on an autopsy performed on a trauma victim with a contained splenic injury who died of a head injury.[2]

Functions of the spleen as known today are as follows:

(i) **Haemopoesis** During the early fetal development, the spleen produces red and white blood cells.[2]

(ii) **Phagocytosis and Filtration.** Free macrophages remove effete red and white cells and effete platelets and micro-organisms from the blood. Simple filtration of particulate matter e.g. micro-organisms also occur.

(iii) **Immunity** The spleen is involved in specific and non-specific immune responses. Properdin

and tuftsin, which are synthesized in the spleen are opsonins. Tuftsin binds to granulocytes to promote phagocytosis, whereas properdin can initiate the alternative pathway of complete activation to stimulate destruction of bacteria as well as foreign and abnormal cells.

(iv) **Hormone regulation of bone marrow activity:** It has been suggested that the spleen produces a hormone which inhibits the production of platelets and while cells by the marrow. The evidence for this is not generally accepted and the presence of such a hormone remains very much in doubt.

(v) **Storage of Red cells:** In most mammals red cells can be discharged into the circulation to increase its oxygen carrying capacity. The ability of the human spleen to do this is minimal owing to the paucity of smooth muscle fibres in its connective tissue framework.

CASE REPORT

M.M. IP. No. 579517 a 51 year old male Zaramo, was referred from Kibaha one of the District hospitals in the Coast region. He presented with 10 days history of being involved in a motor traffic accident, and he sustained multiple minor injuries. He was admitted at Kibaha hospital for 3 days under observation and then discharged. While at home he

started experiencing abdominal pain which was later accompanied with dizziness and general body weakness. The abdominal pain persisted, it was sharp in nature and more marked on the left upper abdomen, but by the time he arrived at our hospital he said the pain was all over the abdomen. The pain was radiating to the left shoulder joint, and was aggravated by movements and relieved by lying still. He also noted distension of his abdomen. He had normal mircturition but he was constipated, he vomited once one day prior to admission in our hospital. There was no history of dyspepsin, fever, or similar problems before the accident.

He was married with 7 children all were alive and well. There was no chronic illness in the family.

Examination revealed a middle aged man in pain, he was afebrile grossly pale, he had no jaundice, had a blood pressure of 100/50 mmHg and the pulse rate was 102 beats per minute regular and thready. The chest was normal.

The abdomen was distended with fullness on both flanks. There was no visible peristalsis and it did not move with respiration. There was a bruise on the left hypochondrium. There was a generalized tenderness over the abdomen, but more marked on the left hypochondrium. Kehr's sign was positive, there

was rebound tenderness, muscle guarding and shifting dullness. Bowel sounds were reduced- Digital rectal examination revealed a normal rectal wall filled with hard stools in pellet form, and a soft bulging mass in the rectovesical pouch could be palpated. No other lesion was detected.

No pelvic fracture or any other major injury was noted. An abdominal tap after patient was asked to lie on his side for 3 minutes revealed an altered non-coagulating blood, consistent with haemoperitoneum.

Musculoskeletal system: He had minor bruises on the extremities which had healed without long bone fractures. The rest of the other systems were essentially normal.

A diagnosis of intra-abdominal visceral injury leading to haemoperitoneum was made and the patient was prepared for emergency laparatomy. An intravenous line with normal saline was inserted, blood for grouping and cross match was taken. A nasogastric tube and urethral catheter were inserted and was shaved.

Patient was transfused 2 units of blood pre-operatively then was taken to theatre for a laparotomy.

OPERATION

The patient was given general anaesthesia, then the abdomen was cleaned with antiseptics and sterile drapes applied. The abdomen was opened through an upper midline incision, and a massive haemoperitoneum of altered blood with clots was found. After sucking out the blood, the spleen was inspected and found to be covered by the omentum. The omentum was mobilised it was also adherent to the anterior abdominal wall. A laceration of about 4 cm in length was found on the hilum of the spleen, and there was slight active bleeding on releasing the omentum.

The spleen was grasped with the left hand and retracted medially, then the splenic pedicle was clamped, the splenocolic and lienorenal ligaments were clamped and ligated with silk number 0. The splenic vein and artery were also clamped separately and ligated with silk number 0, taking care not to injure the tail of the pancreas. Finally the gastrosplenic and splenophrenic ligaments were also ligated with silk number 0. Then the spleen was delivered, the tail of pancreas was again inspected there was no injury, then thorough exploration of the whole abdomen including, bowel, mesentery, liver, both kidneys, bladder were inspected and found to be normal. The abdomen was then closed in two layers, peritoneum with catgut, rectus sheath with prolene

number 1 and skin with silk number 0. Blood loss was estimated to be 2 litres and the patient received a third unit of whole blood intraoperatively.

Post-operatively the patient continued with nil orally, intravenous fluids Dextrose-saline was given at a rate of 3 litres in 24 hours. He was also sedated with Pethidine 100 mg intramuscularly given every 8 hours in the first 24 hours.

The vital signs remained stable. Urine output in the first 24 hours postoperatively was 1.5 litres of clear urine. The patient recovered uneventfully, the skin stitches removed after 8 days and he was discharged home the same day. He was again seen at the surgical outpatient follow-up clinic after one month and then after 3 months, after which he was discharged from the clinic. He was then told to report to hospital immediately whenever he gets fever.

The following are tables summarizing the results of the other patients in the study:

Table I: Age and Sex relationship among the studied patients

Age in years	Male	Female	Total	Percentage
0 - 10	0	0	0	0
11 - 20	6	1	7	31.8
21 - 30	10	0	10	45.5
31 - 40	2	1	3	13.6
41 - 50	1	0	1	4.5
51 - 60	1	0	1	4.5
Total	20	2	22	

Most of the patients (77.3%) are below 40 years of age. Out of the 22 patients there were only 2 female patients.

Table II: The Different causes of injury

Cause of injury	Number of Patients	Percentage
Motor traffic accident	14	63.6
Fall from a height	4	18.2
Assault	3	13.6
Others	1	4.5
Total	22	100

Motor traffic accidents appear to be the main cause of ruptured spleen which accounted for 14 patients, followed by falls from a height.

Table III: Other Associated Injuries

Associated injuries	Number of Patients	Percentage
Head injury	4	18.2
Pelvic fracture	3	13.6
Contused urinary bladder	3	13.6
Lacerated liver	2	9.1
Fractured ribs	2	9.1
Fractured Femur	1	4.5
Urethral injury	1	4.5
Lacerated Transverse colon	1	4.5

All patients who sustained bony pelvis fracture, had urinary bladder contusion and one of them had urethral injury as well.

Table IV Outcome of the Treated Patients

Number of Patients	Associated injuries and Outcome of treatment	Further Treatment	Duration of Hospital stay (days)
13	Recovered well, no associated injuries	Discharged home	7-14
4	Head injury:Discharged through neurosurgical clinic	Analgesics	10-20
3	Transferred to trauma ward to continue treatment of fractured pelvis	Trauma ward	21-70
1	Transferred to trauma ward to continue treatment of fractured Femur	Trauma ward	56
1	Transferred to urology ward due to urethra injury	Urology ward	30
1	Was discharged with colostomy. Had to come back after 3 months for colostomy closure	Surgical Outpatient clinic	17

DISCUSSION

AETIOLOGY

The spleen may be ruptured by penetrating wounds or closed injuries.[4,5] It may be damaged alone or in association with other abdominal, chest organs or skeleton. An enlarged spleen is more prone to injury than a normal one and is also less well protected by the overlying ribs.

The spleen is a rather friable vascular organ, suspended by ligaments that are attached to an adherent capsule, and even relatively minor trauma can result in avulsion of the splenic substance or tearing of vessels in suspensory ligaments, causing profuse bleeding.[6]

No injury to the spleen should be considered trivial, since delay in diagnosis and institution of the proper therapy can result in serious consequences.[6]

The reported mortality of approximately 10% in isolated splenic rupture can be reduced by early diagnosis and prompt treatment.[6]

Splenic rupture may be classified into 4 groups:

(i) Penetrating trauma
    (a) Transabdominal
    (b) Transthoracic
(ii) Blunt Trauma
    (a) Immediate rupture
    (b) Delayed rupture
(iv) Spontaneous rupture.

i) **Penetrating injury:**

Penetrating injury from high speed missiles occasionally can be a diagnostic problem for the unwary physician, particularly if the missiles follows an erratic course, after entering the skin. Missiles such as bullets quite frequently do not

follow a straight trajectory after entering the body and injury to any organ, including the spleen, should be considered in all such patients.[6]

Haemopneumothorax in association with splenic injury indicates penetration of the lung, pleura and diaphragm and a transthoracic approach for splenectomy is often preferable in order to assess and repair the damaged thoracic structures. Such an approach is used, careful consideration should be given to abdominal exploration which usually requires a separate abdominal or a thoraco-abdominal incision.[6]

(ii) **Non-penetrate trauma**

Automobile accidents are the most common causes of blunt trauma to the spleen even in children in whom sliding and bicycle accidents are often the cause of splenic rupture. The spleen can be avulsed from its pedicle, ruptured through the capsule and parenchyma, or ruptured beneath an intact capsule to give rise to a subcapsular haematoma. Bleeding is usually profuse with the first two, although a completely avulsed splenic artery can temporarily close with spasm, particularly in children. Delayed exsanguinating haemorrhage can occur when spasm ceases and the thrombus is pushed out. Delayed rupture, which occurs in 15% of non-penetrating splenic injuries, usually results from a subcapsular

haematoma, which become manifested any time from a day to several weeks after an injury.[6] This could be what might have happened with the patient presented in this study. Delayed rupture not infrequently is heralded by sudden, profuse bleeding and shock, after an episode of minor trauma is forgotten. Delay in diagnosis leads to relatively high mortality rates.

(iii) Spontaneous rupture is rare in a normal spleen; it is more often associated with diseases spleen. Rupture with minimal trauma or spontaneous rupture, has been reported in malaria, mononucleosis (usually in the second to fourth week of the disease), Sarcoidosis, acute and chronic leukemia, congestive splenomegally, polycythaemia rubra-vera, and acquired hamolytic anemias.[5] Rapid, early splenectomy gives an excellent prognosis of survival.

(iv) Operative trauma to the spleen necessitating "incidental" splenectomy occurs in a significant number of operations in the left upper quadrant.[6] Such injury is most common in operations involving the stomach and splenic flexure of the colon.2

Some people have reported an iatrogenic injuries of the spleen ranging from 16-40%.[7] Though a large number of accidental splenectomies are secondary to operations on the colon, the actual incidence of splenic injury during colorectal operation has not

been previously reported.[7] In this study non of the patients had iatrogenic injury of the spleen.

CLINICAL MANIFESTATIONS OF RUPTURED SPLEEN

Although some patients, on admission may be moribund in severe haemorrhagic shock, others may be at the other extreme relatively asymptomatic. The condition of most patients falls between these two extremes. There is usually some evidence of hypovolemia particularly tachycardia and orthostatic hypotension. Most patients have varying degree of generalized abdominal pain, often accompanied by nausea. Only about a third of patients with splenic rupture without other associated abdominal injuries are their pain localized to the upper left-quadrant. pain radiating into the left shoulder (Kehr's sign) is relatively frequent occurring in 15 to 75% patients in most series, and this can often be experienced by placing the patient in the Trendelenburg position. There is usually tenderness in the left upper quadrant, and often it can be elicited as rebound tenderness-[6] In this study (5) 22.7% of the patients had Kehr's sign positive as shown in table V below.

Occasionally there is a fixed mass or fixed area of dullness (Ballance's sign) in the left upper quadrant due to subcapsular or extracapsular haematoma surrounded by omentum.

Shifting dullness and a "doughy" feel to the abdomen are uncommon signs or extensive intraperitoneal bleeding. If clinical and laboratory evaluation does not demonstrate the splenic injury, repeated examinations are often valuable. The importance of continued clinical evaluation in suspected splenic injury cannot be overemphasized.

Table V: Presenting features of the patients with ruptured spleen.

Presenting Feature	Number of Patients	Percentage (%)
Gross pallor	18	81.8
Abdominal distension and tenderness	16	72.7
Shifting dullness	9	40.9
Shock	6	27.7
Positive Kehr's sign	5	22.7

Over 80% of the patients presented with gross pallor, followed by abdominal distension and tenderness.

DIAGNOSIS OF SPLENIC RUPTURE

Diagnosis of splenic rupture may be difficult to establish. The signs and symptoms of splenic rupture will vary according to the severity and rapidity of the haemorrhage in the presence of other injuries. Rupture of the spleen should be suspected on the basis of a history of severe upper abdominal pain,

left scapular or shoulder pain. The pain may be relieved in the recumbent position. It is aggravated by deep breathing or coughing.[8]

Physical examination discloses signs of intraperitoneal bleeding such as a rigid abdomen (more marked in the upper abdomen.[5,8] The abnormalities on physical examination have been described by many observers and are directly related to irritation of the peritoneal cavity by blood.[5] Signs such as referral of pain to the left shoulder (Kehr's sing), dullness to percussion that changes with patient position (Ballance's sign) and neck pain related to irritation of the phrenic nerve (Saegesser's sign) are seen infrequently and are certainly not indications by themselves for either conservative therapy or exploration.[5,6] Physical examination can also be helpful if there are multiple rib fractures on the left side, usually present in about 20% of splenic injuries or areas of ecchymosis or bruising in the left upper quadrant. There are, however, a significant number of patients with splenic injuries who have absolutely no external manifestations of trauma.[5]

Other diagnostic aids include: Radiologic tests and laboratory tests:

- The white blood cell count often is increased to levels of 15,000 to 20,000.

- An initial hematocrit is often misleading, but serial determinations usually show a fall, because haemodilution will by then have taken place.
- Radiologic examinations are often not conclusive, but occasionally will show an enlarged spleen, without sharp outlines, that compresses the stomach medially and the transverse, colon inferiorly in case of splenic haematoma.[6] The left kidney and psoas shadows may be in distinct, and sometimes the left diaphragm is elevated.
- Arteriograms of the splenic artery can be diagnostic in difficult cases but are seldom needed.
- Scanning: There are various techniques for scanning the spleen.

(i) Ultrasound is the investigations of choice when there is difficulty in diagnosing a ruptured spleen or a subcapsular haematoma.[9]

(ii) C.T, scanning may be useful in diagnosing splenic haematoma.[9]

- Paracentesis with an 18 or 20 gauge needle, or a small polyethylene catheter, can be helpful in difficult diagnostic situations. The presence of blood from four quadrant tap does much to confirm the diagnosis of ruptured intra-abdominal viscera, and may be expected in as many as 50% of cases with splenic rupture. The procedure is not without some

danger, particularly when there are dense peritoneal adhesion or abdominal distension, and a negative tap does not exclude injury. In reality there is little justification for routine 4-quadrant taps in all cases of abdominal trauma but rather the procedure should be limited to situations with questionable indications for abdominal exploration.[6] In this study out of the 22 patients only one had abdominal tap to aid in diagnosis of haemoperitoneum.

Radionuclide imaging has become extremely popular in the last several years in evaluating the severity of splenic injury and for use as a possible indication for conservative treatment.[6] Extravasation of radionuclide material or disruption of the normal appearance of the spleen on imaging may be diagnostic of significant splenic injury.[6] In addition many feel that scanning may be preferable to angiography because it has a lower morbidity rate but equal accuracy.

THE TECHNIQUE OF SPLENECTOMY

Removal of the spleen basically involves the division of its ligaments attachments and its vascular pedicle. To accomplish these manouevres the operative-exposure is planned with care.

The stomach must be depressed prior to induction of anaesthesia.[8,9] This is accomplished by an indwelling nasogastric tube which may be removed at

the termination of the procedure.[8,9] An inadequately depressed stomach will obscure the surgeons view of the gastrolienal ligament and the phrenicolienal ligaments.[8]

Adequate exposure for splenectomy may be obtained either through a midline incision or an extended right or left subcostal incision. The disadvantage of the latter two incisions may be the treatment of other associated injuries in a patient who has undergone multiple trauma. The midline incision has the advantages of extended exposure, rapid entrance into the peritoneal cavity, and ease of closure.[5] The presence of large amounts of blood in the left upper quadrant when the peritoneal cavity is entered should alert the surgeon to the possibility of a ruptured spleen. Other significant injuries, such as gastric injuries and injuries to the left lobe of the liver, may produce bleeding in the left upper quadrant, and blind delivery of the spleen into the wound may result in unnecessary splenectomy. It may be advantageous when a large amount of blood is present in the left upper quadrant to place packs on the left hemidiaphragm while evaluating the blood loss to assess the injured organ.[5]

During an emergency splenectomy for splenic rupture and haemorrhage, it is necessary to have the

assistant grasp the hilum of the spleen with his fingers, thus controlling the haemorrhage while the splenic artery and vasa bravia are ligated. Any adhesions between the anterior abdominal wall and the spleen are now divided as is the lienocolic ligament. Firm retraction of the costal margin by the assistant coupled with gentle retraction of the spleen medially by the surgeon, gradually rotates the spleen medially and progressively exposes any diaphragmatic adhesions. Such adhesions should be divided under direct vision since they may also contain blood vessels. This procedure will expose the lienorenal ligament, which is the last remaining attachment of the spleen. The posterior lienorenal ligament is incised near the spleen, beginning inferiorly following the outline of the greater curvature of the spleen. Since this ligamentous structure occasionally is found to contain blood vessels, dissection must be done under direct vision.[8] At the superior pole of the spleen is the phrenicolienal ligament. Division of the phrenicolienal ligament is extended to join the medial incision in the gastrolienal ligament, so that the spleen is now free of ligamentous attachments and is maintained only by its hilar vascular pedicle. The hilum of the spleen is now freely visible and the vessels of the hilum are identified and divided, care is taken not to

damage the tail of the pancreas. The splenic vessels at the hilum have usually divided into branches they may range from 6 to 30 in number. These vessels should be isolated individually and doubly ligated with stout non-absorbable sutures.[8] When the last vessel has been divided the spleen will be fee and may be removed from the abdomen. The splenic bed is carefully inspected to ascertain that adequate hemostasis has been secured.

The abdomen is again searched for bleeding points. The areas most prone to haemorrhage are the tail of the pancreas, the greater curvature of the stomach, and the diaphragmatic surface of the divided splenic ligament.

The decision whether or not to drain the splenic bed continues to be controversial. The advocates of drainage state that it may reduce the number of intra-abdominal infection, whereas the opponents state that the drain may in reality cause several of the wound infections or subphrenic abscesses or may result in erosion of a hollow viscus.[5,8] In this study drains were not used, thorough haemostasis was achieved and peritoneal lavage was done with warm normal saline. It is of my opinion not to use drains as they increase the risk of infection, its important for the surgeon to ensure thorough haemostasis rather than relying or drains.

CHANGING CONCEPTS IN THE TREATMENT OF RUPTURED SPLEEN

In recent years the spleen's important role in cellular and humoral immunity has been clarified, and the danger of overwhelming bacterial infection in a splenic patients has been established. Operative techniques for splenic preservation have been developed, and a concept for non-operative management of selected splenic injuries is evolving.[10,11]

Conservatism in the management of splenic injury has extended beyond repairing and preserving an injured spleen when possible. Bleeding from splenic trauma appears to be more self limited in children than in adults, non-operative therapy requires a stable patient who is found by diagnostic tests to have an isolated splenic injury.[10]

Successful non-operative management of blunt splenic injuries in children was first proposed two decades ago by Lepadhyaya as reported by Pachter H.L. et al.[13] Subsequent series have established non-operative management as the most commonly used form of treatment in children with blunt splenic injuries.[13] In adults the applicability of this approach has been uncertain. Frequently cited concerns were that success rates in adults would not parallel the experience reported in children because of differences in both size and texture of the spleen; concomitant intra-abdominal injuries might be

overlooked; transfusion requirements would be excessive increasing the risk of hepatitis and more recently acquiring the human immunodeficiency virus.[12,13]

There seems to be a uniform agreement that patients must meet the following criteria to be considered for non-operative treatment.

(i) Haemodynamic stability must be present or initially maintained by modest infusions of crystalloid solution.

(ii) The patient must be alert so that frequent abdominal examinations can be performed.

(iii) CT Scan documentation of a grade I,II or III splenic injury, in the absence of associated intra-abdominal injuries must be present.

(iv) Transfusion requirements must be less than three units. Recent data from eight institutions with a dedicated interest in the non-operative treatment of blunt splenic injuries, revealed a mean non-operative treatment of blunt splenic injuries, revealed a mean non-operative rate of only 18.[13]

Patients who are selected for non-operative management are admitted to the surgical intensive care unit for 48 to 72 hours of bed rest and observation. Replacement of initial blood loss of up to 1000 ml (or an equivalent amount in children) over the first few hours after injury may be required.

Evidence of continued blood loss is an indication for a laparatomy. Adults who remain stable are kept in the hospital with limited activity for 7 - 10 days.[14] After discharge patients are restricted from vigorous physical activity but may return to non-strenuous daily routines. Children who remain stable after splenic injury are managed similarly to adults. Total hospitalization, however is usually for 2-3 weeks because restriction of activity of younger children after discharge may be more difficulty than restriction of adults.[14,15] Follow up splenic imaging before discharge and at monthly intervals indicates resorption of subcapsular hematomas or healing of splenic fractures within 3 months after injury in almost all patients manages as outlined.[14,15]

During the past 5 years advances in imaging techniques have been the single most important factor responsible for a greater willingness by surgeons to consider non-operative treatment of blunt splenic injuries in the adults.[13]

Computed tomography scanning has been invaluable in assessing both the anatomy and severity of the sustained splenic injury.[13,16] The danger of non-operative treatment seems to be far outweighed by the lifetime sentence of potential post-splenectomy overwhelming sepsis.[16,17]

Recently splenorrhaphy has been advocated especially in children.[10] Several techniques of repairing the torn spleen have been described, but this should not be attempted unless the surgeon has experience with the technique.[10]

As experience and confidence with segmental resection; splenorrhaphy ligation of segmental vessels, capsular repair, and the use of microfibrillar collagen for hemostasis have grown, large numbers of patients have had operative preservation of the injured spleen.[14]

Control of parenchyma bleeding during partial splenic resection can be provided by temporary occlusion of the splenic artery. Individual ligation or clipping of intrasplenic vessels in intersegmental planes can be accomplished.[13] Through and through 2/0 catgut sutures are adequate for hemostasis.[18] Bleeding from raw surface can be controlled with microfibrillar collagen. splenic artery ligation may be needed for control of haemorrhage and will not lessen viability of the spleen.[14] Another more recent technique of using stapler as an alternative to the sutured partial splenectomy has been developed and results are good.[19]

Repaired spleens are not at increased risk for future injury but undergo healing with preservation of viable tissue. It appears that if at least 50% of

the normal splenic tissue can remain viable, essential foundations of the spleen can be preserved. All patients with penetrating wounds would be managed by laparatomy and splenic repair when possible.[13,18]

Splenic autotransplantation

Several studies have been done on splenic autotransplantation and some have concluded that, although autotransplantation does provide a method for preserving some splenic function they concluded that it was interior to partial splenectomy. Splenic autotransplantation in animals models have shown to reduce Haemophilus influenzae meningitis in asplenic rats and gave some protection from pneumococcal infection in mice. These studies are still experimental no human trials have been done yet.[20]

POST-OPERATIVE COMPLICATIONS

(i) The most common complication is left lower lobe atelectasis.

(ii) A more serious complication is postoperative haemorrhage from thrombocytopenia or inadequate haemostasis. Continued bleeding requires prompt re-exploration.

(iii) Other Post-operative complications include left subphrenic abscess, pancreatitis.

(iv) There is a continued risk of overwhelming postsplenectomy infection.[14]

Thrombocytosis following splenectomy, when present, usually does not cause thrombotic complications. However, in some instances (e.g. myeloproliferative disorders) when the platelet count rises over 1 million. Thrombosis of peripheral and splanchnic veins may occur. Specific antiplatelet therapy should be used to prevent thrombotic complications in patient who have markedly elevated platelet counts.

Overwhelming post-splenectomy infection (OPSI). The evidence of the immunologic role of spleen provides an explanation of fulminating postsplenectomy infection. When the spleen encounters relatively few-blood-borne organisms (especially encapsulated bacteria) it effectively and rapidly mobilizes an immune response.[14] The spleen initiates rapid specific antibody synthesis or releases the antibody in high local concentration into the slow-moving splenic pulp, where efficient phagocytosis occurs. The way is thus prepared to deal effectively with the subsequent phases of the bacteremia that determine the outcome of infection.

Without the spleen the patient is deprived of an organ capable of both recognizing small amounts of particulate antigen and of phagocytosis in the absence of antibody would not reach to small antigenic challenge presented by the few organisms in

the circulation in the early phases of bacteremia. Because the liver is relatively inefficient in removal of poorly opsonized bacteria compared with the spleen, increasing numbers of organisms can circulate freely, producing fulminating sepsis.

Although low the precise incidence of severe post splenectomy infection is not known.[14] However the risk is greater in children than in adults, which is higher after splenectomy for hematologic reasons than for trauma is increased after incidental splenectomy during abdominal operations for malignancy, and is present for the life time of the patient of more importance is the fact that asplenic persons are much more susceptible to death from infection with encapsulated organisms.

The characteristic syndrome of severe sepsis seen in asplenic persons has been called"overwhelming post-splenectomy infection.[14] It is unlike fulminating bacteremias and septicemias in persons with normal splenic function. The course of this overwhelming sepsis in asplenic persons from good health until death often lasts only 12 to 14 hours.[14]

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MANAGEMENT OF PANCREATIC PSEUDOCYST

SUMMARY

Seven patients with pancreatic pseudocysts were seen in the three surgical firms during the period of July 1988 to August, 1990 (26 months). Their ages ranged from 17 to 61 years, with a mean age of 46 years. There were five males and two females.

The most frequent presenting complaints of these patients were abdominal pain, abdominal swelling, vomiting, constipation and loss of weight. Three patients had history suggestive of pancreatitis while two patients gave history of blunt abdominal trauma and one patient gave history of excessive alcohol indulgence.

All seven patients were operated upon, six had transgastric cystogastrostomy and one patient had cystojejunostomy with Roux-en-Y reconstruction. There was no death.

INTRODUCTION

A pancreatic pseudocyst is a collection of pancreatic secretions localized in the lesser sac, which accumulates following pancreatic inflammation and ductal disruption, and the wall lacks epithelial lining.[1,2,]

Most pseudocysts are unilocular and are located in the lesser sac.[3] In order of frequency the

pseudocyst projects between the stomach and the transverse colon, between the stomach and the liver behind or below the transverse colon.[1] They occasionally occur within the pancreas (as retention cyst) and even more rarely may be found behind the pancreas or within the mediastinum.[3] Since a pseudocyst usually results from disruption of the ductal system of the pancreas, the ultimate form and location of the lesion are dependent upon the position and the extent of pancreatic injury and the secretory pressure of the pancreas distal to the part of ductal damage.

The fluid within a pseudocyst varies from clear and colourless to a brown or greenish material, depending upon the amount of blood or necrotic pancreas present.[3]

As recently as 1972, the preoperative objective assessment of a clinically impalpable pancreatic pseudocyst was limited to upper gastrointestinal radiologic studies, which were very insensitive, depending as they did on distortion by the pseudocyst of adjacent structures. The natural course of pancreatic pseudocyst at that time was poor and in retrospect, often incorrectly understood.[4]

Since 1972, on the diagnostic front, there has been application of essentially non-invasive tools such as ultrasonography (US), computed Tomography

(CT), amylase-isoenzyme measurements, and the relatively non-invasive endoscopic retrograde cholangiopancreato-graphy (ERCP) which have facilitated early, accurate sequential, and reproducible tracking of pseudocysts to an impressive degree, showing their evolution regression, growth and complications.

CASE REPORT

M.A. Inpatient number 568279, a 45 year old man was referred to our hospital with five months' history of abdominal swelling and pain. One month prior to the onset of the above problems he presented with acute abdomen to a certain private hospital, where he had an emergency laparatomy and found to have haemorrhagic pancreatitis. One month after the operation he noted his abdomen was increasing in size, this was accompanied with pain which was colicky in nature and localized in the epigastrium. He also had, a sense of abdominal fullness making him eat a smaller amount of food. He had occasional vomiting soon after eating, with no history of jaundice before the emergency laparatomy. He denied history of abdominal trauma. He had normal bowel motion and he was passing a normal colour urine.

On examination he was found to be a middle aged man, wasted, he was not jaundiced, not pale and he was afebrile. His cardiovascular and respiratory

systems were normal. Abdominal examination revealed an epigastric mass, which measured about 8 cm in greatest diameter, cystic, not mobile, smooth, ovoid and non-tender. The rest of the abdomen was soft, no guarding or tenderness. Spleen was also palpable about 3 cm below costal margin non-tender, liver and kidneys were not palpable.

Digital rectal examination revealed a normal rectal wall filled with normal colour stools. A clinical diagnosis of Pancreatic Pseudocyst was entertained. Several investigations were done.

1. Haemoglobin 10.8 g/dl.
2. Plain abdominal X-rays revealed no pancreatic calcifications.
3. Barium-meal could not be done as there was no Barium sulphate.
4. Ultrasound was done, it was reported as "9 x 15 cm monocystic mass between the stomach and liver. The mass had thick wall with pancreatic silhouette prominent at the head." Concluded to be a pancreatic pseudocyst.
5. Serum amylase was not done as there was no reagent.
6. Some liver function tests were also done:
   (i) Bilirubin - Total 62 mmol/l
   - Direct 44 mmol/l
   (ii) Protein - 76.6 g/dl

Alb. 36.3 g/dl

α_1 = 4.0 g/dl

α_2 = 8.1 g/dl

β = 24.2 g/dl

(iii) Alkaline phosphatase = 504 iu/l.

(iv) Fasting blood sugar = 4.8 mmol/L

7. Bleeding indices:

Prothrombin Time

PT 12" = 84%

Control 11" = 100%

PTT = 40"

Control = 36

8. HBsAG = positive

9. Alphafeto Protein = Negative

The patient was scheduled for laparatomy as the mass was increasing in size and the patient was loosing weight. The abdomen was opened through the midline incision after cleaning the skin with antiseptic and draping the patient with sterile drapes. The laparatomy findings were:- A huge pseudopancreatic cyst behind the stomach in the lesser sac, there was "scarring" of the first part of the duodenum but there was no obstruction. Other findings included a dilated and tortuous common bile duct and dilated cystic duct. There were no stones in the common bile duct or in the gall bladder.

The anterior wall of the stomach was opened and a small opening measuring about 4 cm was made in the posterior wall of the stomach creating a stoma into the cyst and 2 litres of dark greenish fluid was sucked from the cyst. Then marsupilization of the stoma was done and the anterior wall was closed in two layers using silk number 2/0. The abdomen was explored no other cyst or lesion were seen, the abdomen was irrigated with warm normal saline and closed in layers.

The patient recovered well post-operatively, he was discharged one week after surgery. He was seen again one month later in the outpatient clinic, he was in good condition and had no complaints. The other cases are summarized below:

Table I: AGE AND SEX DISTRIBUTION

Age Groups (Years)	Male	Female	Total
10-20	0	1	1
21-30	1	0	1
31-40	0	0	0
41-50	2	0	2
51-60	1	1	1
>60	1	0	1
Total	5	2	7

There were two females and five males. Only two patients were below 40 years.

Table II: PRESENTING COMPLAINTS

Complaints	Number of Cases
Abdominal pain	5
Abdominal swelling	5
Weight loss	4
Vomiting	3
Constipation	2
Fever	1

The commonest presenting feature was abdominal pain, abdominal swelling and weight loss.

Table III: PREDISPOSING FACTORS

Predisposing Factor	Number of patients
Pancreatitis	3
Trauma (abdominal)	2
Alcohol (Alcoholic Pancreatitis)	1
Unknown	1

Pancreatitis had the highest frequency and two patients had history of blunt abdominal trauma.

All patients were operated upon with no deaths. Six patients had cystogastrostomy and one had cystojejunostomy.

DISCUSSION

AETIOLOGY

Pseudocyst may follow traumatic injury to the abdomen with direct damage and haemorrhage in the pancreas.[1,5]

In acute pancreatitis about half of all cases are complicated by the formation of pseudocyst, but 20-40% of these resolve spontaneously.[1] In acute alcoholic pancreatitis ultrasound studies have demonstrated an incidence of 11 to 18 percent of pseudocyst formation and 8 percent incidence of spontaneous resolution.[2,6] Pseudocysts are also associated with other causes of acute pancreatitis (beside alcohol) and with chronic pancreatitis, pancreatic trauma either external or operative.[2,7]

Pancreatic cysts may develop either as a result of ductular obstruction leading to the formation of a retention cyst or following superimposed acute inflammation, resulting in pseudocyst formation. Retention cysts are demonstrable in 25% of patients, and approximately half of these, are small intrapancreatic cysts, which do not require treatment.[7] Cyst formation may result in compression of surrounding structures (such as the common bile duct and colon or result in increasing pain, necessitating surgical decompression.[7] Pseudocysts that complicate acute exacerbations of chronic

pancreatitis are similar to those that develop in acute pancreatitis and 25% resolve spontaneously.[7] Other causes are unknown.[8]

In this study 4 patients had previous history suggestive of pancreatitis one of whom admitted to be an alcoholic, he had been taking hard drinks for over 10 years taking an average of 2 litres/day. Another patient who had acute haemorrhagic pancreatitis was diagnosed at laparatomy 5 months prior to admission in our hospital with a pseudocyst.

PATHOGENESIS

Pseudocysts are distinguished by their content of pancreatic juice. Although this juice is diluted by serum, inflammatory debris, and sometimes frank blood, the cyst fluid contains all the usual pancreatic enzymes in high concentrations. When in doubt about the nature of a mass, it is easy to measure its fluid amylase, which ranges 3 - 50 times the normal serum values.[9] Lipase, trypsin, chymotrypsin, carboxypeptidase, and other enzymes are all present, as well as bicarbonate, which is neutralized by serous exudate resulting in a normal pH.

When not infected, this fluid evokes only a mild inflammatory reaction, producing a shaggy fibrinous exudate on surrounding surfaces.[9] This shell of fibrin is converted in one to several weeks into a

tough fibrous capsule which may be softened in places by fat necrosis and inflammatory reaction. There is no central layer of secretory endothelium as in other cysts - hence the name "pseudocysts".[9]

The pancreatic juice in the cyst comes from the acinar cells in the main pancreas. Often this juice comes directly from the major duct, pouring out through a small rupture created by necrosis in the surrounding parenchyma.[9] This communication is hard to find at operation but often may be demonstrated later by injection of X-ray contrast material into the tube draining a cyst cavity.

The localized acinar necrosis may be produced by blunt or penetrating trauma or by a preceding attack of acute pancreatitis. Pseudocysts also appear in the course of chronic relapsing pancreatitis, and in a few cases behind carcinoma in the head of the pancreas. These generally grow more insidiously without a prior acute abdominal episode and have been termed retention cysts.

Three elements are necessary for creation of a typical pseudocyst:

a) Ductal rupture communicating with ~~the~~ a potential space.

b) an actively secreting pancreas

c) Interference with normal ductal drainage.[9] The pressure of pancreatic secretion from the main

ductal system forces the pseudocyst to enlarge until it meets counterbalancing resistance.[9] Further water may be attracted osmotically by the inflammatory protein content of the pseudocyst.

CLINICAL FEATURES

The patient with a pancreatic pseudocyst may be asymptomatic or may present with upper abdominal pain. Other symptoms and findings related to pseudocyst include early satiety, nausea and vomiting secondary to gastric outlet obstruction, pruritus and jaundice secondary to obstruction of the common bile duct; edema of the legs and weight gain secondary to compression or obstruction of the inferior vena cava: Alteration of bowel habits and symptoms of obstruction of the gastrointestinal tract secondary to colonic obstruction; variceal bleeding secondary to obstruction of either the splenic or portal vein; arterial haemorrhage secondary to formation of pseudoaneurysm in adjacent visceral vessels, and signs of sepsis secondary to infection of the fluid within the pseudocyst.[2]

In this study abdominal pain and swelling had the highest frequency of presentation. None of the patients presented with sepsis. Two patient presented with a tinge of jaundice, but none had pruritus. One of the patient during operation after evacuating the pseudocyst there was another swelling which was thought to be a

cyst but when opened it bled a lot this later confirmed to be an aneurysm, which was most likely a traumatic aneurysm. It was closed successfully and the patient recovered well, the same patient was also HIV antibody positive by ELISA serotest.

Pseudocysts have presented in an amazing variety of ways both with and without evident masses. In chronic pancreatitis,cysts within the head of the pancreas too small to be recognized by upper gastrointestinal series have been found at operation by needle aspiration.[9] Cystoduodenostomy has relieved this source of abdominal pain. Similar lesions have produced typical obstructive jaundice without other signs of a pseudocyst.

The pressure of pancreatic secretion aided by its potent enzyme content can burrow surprising distances, forming distinct tracts surrounded by fat necrosis, debris and wood inflammation. These follow by chance or proximity, one of three favourite routes.

i) The tract may extend upward through the oesophageal hiatus into the posterior mediastinum where a true pseudocyst may develop. This tract may occassionally extend further and present as an abscess in the neck.

ii) The right lateral lumbar gutter is a second area for tract formation from the head of the pancreas.

iii) The left lumbar gutter is involved from the tail. Tracts dissecting downward and laterally on the

right have presented as abscesses in the groin or even through the groin or even through the groin into the scrotum.[9]

Cysts may form anywhere along the course of such tracts, and where pressure builds up adjacent viscera may be eroded. Colonic necrosis has been encountered, often at the hepatic or splenic flexures where the colon is less mobile, resulting in a colopancreatic fistula. This infects the entire cyst or lesser sac with colonic organisms and can be a disastrous complication of undrained pancreatic juice.[9]

Chronic or recurrent pleural effusions of pancreatic juice are probably caused by similar burrowing and erosion.[9]

DIAGNOSIS

1. Diagnosis of a pseudocyst is based on clinical history and physical examination. Physical examination discloses a mass in only half the patients even though a large cyst is present hidden under the rib margin. Other less specific physical signs are epigastric and left castovertebral angle tenderness or elevated lung bases.[9]

Usually history will establish one of the three situations.

i) The mass has followed shortly after a sudden severe attack of acute pancreatitis or post operative pancreatitis.

ii) There has been trauma to the upper abdomen, either penetrating or crushing, such as a steering wheel injury.

iii) The onset has been insidious without any recognisable sudden pancreatic injury. The presenting symptoms could be persistent pain, fever and ileus during or after an attack of pancreatitis.[3,9]2.

Radiology:

i) A chest X-ray may reveal elevation of one or both hemidiaphragms, and a pleural effusion 20-35% which may be bilateral but is more commonly seen on the left side.[11]

ii) Plain abdominal X-ray film may show a soft tissue mass in presence of pancreatic pseudocyst. Pancreatic calcifications may be present in chronic pancreatitis, particularly alcoholic.[10]

iii) An upper GI series is the mainstay of diagnosis and will show the mass displacing the stomach or duodenum in 77-88% of patients. It may push the stomach or duodenum in a number of different directions.[9]

iv) An intravenous pyelogram may show a downward displacement or distortion of the left kidney.[9]

iv) A barium enema may show the transverse colon thinned out and pushed downward by the mass.[9]

3. Ultrasonography is of value in detecting a pseudocyst, especially as a preliminary to the CT scan.[10]

4. CT Scan the most helpful radiologic examination usually shows a mass adjacent to the pancreas displacing the stomach anteriorly and superiorly. Occasionally the lesser curvature of the stomach may be flattened by a high lying pseudocyst.

5. Serum amylase is elevated in only 41-55 percent. A much more sensitive test of abnormal amylase activity is the urine amylase output. This is measured in a timed urine specimen and expressed in units per hour to eliminate fluctuations with changing urine concentrations.

When done in this way the urine amylase output has been elevated in 80 percent of pseudocyst subjects.[9]

In this study diagnosis was made by clinical examination, and ultrasound. There was no contrast media for Barium meal and there was no reagent for serum amylase estimations.

TREATMENT

Treatment for pancreatic pseudocyst can either be conservative or operative.

(i) <u>CONSERVATIVE</u>

It is necessary to distinguish between acute and chronic pseudocyst formation. Acute pseudocyst commonly arise after severe attacks of pancreatitis. About 20-40 per

cent of these will resolve without treatment and so no action should be taken during the first weeks after they have been recognised unless there is clinical evidence that the cyst has become infected. Where however, infection has supervened or when the cyst has been present for 2-3 months or is over 6 cm in diameter, operative treatment is recommended because of the risk to life from spontaneous rupture.[1] In this study one patient had spontaneous rupture, while being investigated. An emergency laparatomy was done and he recovered well.

(ii) OPERATIVE TREATMENT

Surgical treatment is recommended for pseudocyst of the pancreas for two reasons:

(a) Complications such as secondary infection, severe haemorrhage, or rupture into an adjacent viscus or into the free peritoneal cavity may occur if a pseudocyst remains untreated.

(b) These lesions rarely resolve once a thick fibrous wall has developed.[2,3]

A patient who develops a mass during an attack of acute pancreatitis should be observed for several weeks, should the mass enlarge or the patient become more ill during this period, immediate operation is indicated. If physical and radiologic examination show complete disappearance of the mass, operation may be postponed indefinitely, but pseudocysts that persist for 4 to 6

weeks are unlikely to resolve spontaneously and should be treated aggressively.[3]

The mainstay of surgical treatment is the drainage of the cyst. There is no single procedure which is appropriate for every pseudocyst of the pancreas.[3] The various procedures are:

(a) External drainage: This is done in extremely ill-patients requiring urgent drainage in whom more extensive procedures might endanger life. If the pseudocyst has a thin flimsy wall, external drainage may be the only means of treatment.

(b) Internal drainage: This is the best treatment for most pseudocysts. Transgastric cystogastrostomy when the lesion is adherent to the stomach or transduodenal cystoduodenostomy if the cyst is located in the head of the pancreas, have produced mortality rates of 2.5 to 5 percent with approximately the same low rate of recurrence.[3] In this study patients had internal drainage, six had cystogastrostomy and one patient had cystojejunostomy with the Roux-en-y reconstruction. In all cases a piece of cystic was sent for histological analysis and none reported malignancy.

(c) Drainage through the duct of Wirsung after transduodenal sphincterotomy has been advocated by Doubilet and Mulholland, based on the principle that pseudocysts have major ductal communications. The primary indication for transphincteric drainage is in

patients with gallstone pancreatitis who are undergoing corrective biliary tract operation. In these cases, common duct exploration is frequently performed, making transsphincteric drainage a rather simple matter and recurrence of the pancreatitis is less likely.

(d) Extirpation: theoretically this is an ideal method of therapy. Only the rare, and very small pseudocyst located in the distal pancreas and unattached to vital structures can be excised. Extirpation is only recommended under these special circumstances as the mortality associated with it is high.[3]

(e) Pancreatic Resection: Most authors agree that resection of the pancreas with a pseudocyst is, in certain cases indicated. Without dilatation of the main pancreatic duct, multiple pseudocysts that constitute the head of the pancreas are not appropriate for the internal drainage procedure, and resection of the proximal portion of the pancreas is often the only possibility. In such situations the method of choice is usually pancreaticoduodenectomy (Whipple's procedure) or duodenum saving near to total pancreatectomy. Resection is also often indicated, when the patient already has severe pancreatic endocrine insufficiency.

COMPLICATIONS

The complications encountered in most studies are: haemorrhage which is the major and commonest complication.[1,2,3,12] Gastric outlet obstruction,

pancreatic fistula which tend to occur following this occur in external drainage leading to loss of fluids and electrolytes. Other complications include secondary infection; rupture into an adjacent viscus or into the free peritoneal cavity.[3]

RECOMMENDATION

Pancreatic pseudocyst is probably not a common problem in our environment. According to our study it was not easy to establish history of acute or chronic pancreatitis. All our patients had obvious or clinically palpable cysts. This means that, there is a possibility that we are missing patients with small cysts and which most probably resolve spontaneously. We hope with the availability of modern facilities like ultrasound and may be later CT Scan will be able to detect more patients and this will also help in the follow up of these patients.

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BENIGN PROSTATIC HYPERPLASIA

SUMMARY

During the period July 1988 to June 1990, 55 patients were admitted in surgical Firm II with clinical features of benign prostatic hyperplasia. All 55 patients were operated, and they all had open prostatectomy with exception of two patients who were referred to the Urological unit for Transurethral resection of the prostate.

Most patients presented with poor stream 40, acute urinary retention 38, urgency 36, frequency 18.

The commonest associated medical condition was hypertension and anaemia.

INTRODUCTION

Benign prostatic hyperplasia is an extremely common disorder in men over 50 years of age.[1,2] It is a benign tumour that originates from the periurethral prostatic tissue.[2] It also ranks second among commonest urological admissions at Muhimbili Medical Centre Dar es Salaam Tanzania.[3] Hiza as quoted by Philip and Nkulila recorded 79 cases out of 1812 patients admitted in General Surgical Department in 1971.[3] Philip and Nkulila recorded benign prostatic hypertrophy as one of the commonest urological problem and second only to hydrocele during the period 1977 to 1979.[3]

It is remarkable that although vesical calculi had been treated by surgery for over 2,000 years, the role

of the enlarged prostate in causing bladder outlet obstruction was fully understood, only in the later half of the last Century and its treatment by prostatectomy established only after the turn of the century.[4]

The word "prostate" is derived from the terms used in the fourth century B.C. by Herophilus of Alexandria who according to Galen, referred to the seminal vesicles as "adenoicides prostatai (glandular prostate and to the ampullae of the Vasa as Kirsoides prostatai" cirsoid prostate, the word denoting the position of these organs at the bladder neck.[4]

The origin of prostatic hyperplasia from the periurethral glands was suggested by Jones in 1894 and was established by the studies of Albarran and Motz in 1902 and Perearnall in 1905. Subsequently, Reischauer in 1925 demonstrated that the glandular proliferation was preceded by the development of fibromuscular nodules.[4,5]

Although reports vary slightly, a careful survey of the prostate in an unselected series of post-mortem disclosed nodular hyperplasia in approximately 50-60% of men aged 40-59 years, and more than 95% of those over 70 years.

In this study it was found that the highest prevalence was at the age between 50-60 years, for those who came with symptoms of prostatism. The reason could be that not all patients with prostatic hyperplasia

presents with symptoms of obstruction. It has been noted that only about 5-10% of men with this condition will present with symptoms and require surgical treatment.[1]

It has been noted that after the age of 40, androgen production diminishes and glandular hypertrophy and hyperplasia of the prostate occur; progressing with advancing age.

CASE REPORT

S.M. IP. No. 546511 a 66 year old Zaramo, presented to our outpatient department with a painful right inguinal swelling for about 6 hours duration. He had noticed the reducible swelling for a period of 2 years but this particular day it was painful and irreducible. He was diagnosed as having obstructed right inguinal hernia and an emergency herniorrhaphy was done. Two days post-operatively he complained of urge incontinence and increased frequency of micturition. On examination he was found to have a full bladder palpable up to the umbilical level. He admitted history of poor stream of urine and frequency of micturition for about one year. He was catheterized and digital rectal examination revealed an enlarged prostate grade II, it was firm non-nodular, non-tender and rectal mucosa moved freely over it.

Clinically he was healthy looking he was not pale, and his blood pressure was 120/80 mmHg.

He was investigated as below:

(a) Urinalysis - Clear yellow urine
- Acidic pH
- Proteins, ketones and red blood cells were absent
- Sugar was negative

(b) Serum for: - Alkaline phosphatase 92 IU/l
- Acid phosphatase 6.0. IU/l
- Creatinine 92 µmol/l

(c) Haemogram - Revealed Heamoglobin 13.0 g/dl and a normal blood film.

(d) Chest x-ray was normal

(e) Intravenous Urogram: reported as normal except for a left side duplex drainage system, and a prostatic impression on the cystogram.

(f) Cystocopy: Revealed a normal bladder with normal ureteric orifices. No bladder diverticulum. It showed both lateral and median lobes enlargement.

The patient was scheduled for open prostatectomy, four weeks after herniorrhaphy .

OPERATION

Patient was given general anaesthesia the indwelling urethral catheter was removed. The abdomen was cleaned with antiseptic (savlon, Iodine then spirit) then drapped with sterile drappings.

A midline subumbilical incision was made the muscles were retracted aside and the bladder was exposed

extraperitoneally. With the help of a self retaining retractor and 2 stay sutures the bladder was opened through a longitudinal incision. The prostate was found to be enlarged, the bladder wall was normal without intravesical calculus or diverticulum. A split of the anterior commissure was made by the index finger and the prostate was enucleated through the cleavage created by the finger between the capsule and the adenoma. The prostatic bed was packed with a hot pack for 4 minutes. An attempt to achieve haemostasis by placing catgut sutures at obvious bleeders was done. The ureteric openings were inspected and ensured that they are patent and urine flowing out of each ostia.

A size 20 French Gauge 3 way Foley's catheter was introduced per urethra into the bladder, its balloon inflated to 20 mls of normal saline and placed in the prostatic bed. The bladder was closed water tight with chromic catgut in two layers. The abdominal wall was also closed in layers with Dexon suture and skin with nylon No 2/0. The catheter was connected to urinal bag and a continuous bladder irrigation was stopped after 48 hours when the urine was clear with no clots. The urethral catheter was removed after 14 days. The patient was encouraged to start ambulation after the 4th day. He recovered uneventfully and was discharged 14 days postoperatively after removal of the urethral catheter. He was seen at our outpatient clinic one

month later with no complaints and he was voiding normally.

The other patients are summarized in the following tables:

RESULTS

Table I: The number of patients in relation to age groups

Age Groups in years	Number of Patients	Percentage (%)
Below 50	0	0
51 - 59	19	34.5
60 - 69	17	30.9
70 - 79	14	25.4
80 and above	5	9.1
Total	55	100

The peak age incidence of prostatism was between 51-69 years, constituting 65% of all cases.

Table II: Some of the presenting clinical features of prostatism

Clinical Features	Frequency	Percentage (%)
Urine retention	47	85.4
Poor stream	43	78.1
Frequency	38	69.1
Urgency	26	47.2
Hesitancy	21	38.1
Dysuria	13	23.6
Haematuria	11	20.0
Stone formation	5	9.1
Bladder diverticulum	2	3.6

Urine retention which was accompanied with gross bladder distension appears to be the commonest problem which prompted the patient to seek medical help. The other symptoms were: poor stream, frequency, urgency, hesitancy and haematuria.

Table III: Other associated medical conditions besides the problem of prostatism are shown in the this table.

Other associated Medical conditions	Number of Patients	Percentage (%)
Hypertension	23	41.8
Anaemia	12	21.8
Urinary Schistosomiasis	5	9.1
Cardiomyopathy	1	1.8
Obstructed Ing. Hernia	1	1.8

It was noted that quite a number of patients had hypertension followed by anaemia.

Table IV: Surgical approach for prostatectomy among the studied cases

Surgical approach	Number of Patients	Percentage
Transvesical Prostatectomy	48	87.2
Retropubic	5	9.1
TURP	2	3.6
Transperineal	0	0

Almost all patients had open prostatectomy; except 7 patients, of these seven, five had retropubic prostatectomy and one had cardiomyopathy was offered Transurethral resection and another patient was a very elderly patient and fragile was also offered Transurethral Resection Prostatectomy.

Table V: Type of Anaesthesia offered during Prostatectomy

Type of Anaesthesia	Number of Patients	Percentage
General anaesthesia	37	67.3
Spinal anaesthesia	11	20
Epidural anaesthesia	7	12.7

The choice of anaesthesia was decided by the anaesthesiologist, depending on the condition of the

patient and convenience of anaethesiologist in monitoring the patient.

Table VI: Post-operative complications

Post- Operative Complications	Number of Patients	Percentage
Wound sepsis	31	56.3
Haemorrhage	26	40.0
Urinary incontinence	21	38.1
Suprapubic leak	7	12.7
Pneumonia	3	5.5
Cardiac arrest	1	1.8

Wound sepsis was the commonest complication especially to those patients with long standing indwelling catheter, this was followed by haemorrhage which was mainly reactionary haemorrhage.

Table VII:Duration of hospital stay after prostatectomy

Duration of hospital stay (days)	Number of Patients
5 - 10	4
11 - 15	20
16 - 20	17
21 - 25	9
26 - 30	3

Majority of the patients were discharged 2 weeks post-operative after removal of the urethral catheter. Others stayed a little longer mainly because of wound sepsis and other complications.

Out of the 55 patients only one patient died of cardiac arrest intra-operatively.

DISCUSSION

PATHOGENESIS

One of the pathologic processes to involve the prostate is hyperplasia which is characterized by an enlargement of the gland, secondary to a cellular increase in both its fibromuscular and acinar components. Architecturally the mature prostate gland consists of a group of multicentric, inner periurethral glands and an outer group of paraurethral or true prostatic gland with their fibromuscular stroma.[5]

Hyperplasia originates in the inner periurethral group of glands compressing the outer paraurethral glands into a surgical false capsule.[5]

AETIOLOGY

Throughout the years a number of aetiological theories have been advanced to explain the development of hyperplasia. "When the hair becomes grey and scanty, when specks of earthy matter begin to be deposited in the tunics of the artery, and when a white zone is formed at the margin of the cornea, at this same period the prostate gland usually. I might perhaps say

invariably becomes increased in size" (Sir Benjamin Brodie 1849).[4,6]

Benign enlargement of the prostate usually occurs in men over 50 years of age; most often between 60 to 70 years.

In Indians prostatic enlargement is less frequent, and occurs more often in a younger age-group. The reason for these discrepancies is unknown.[6]

Theories of causation of benign enlargement of the prostate have been put forward. Mastofi has written an extensive review in Campbell's urology outlining the investigative work that has taken place in each of the present theoretical etiologic principles.[4]

I **HORMONE THEORY**

Since the prostate is a secondary sex gland the hormonal theory of hyperplasia has been the most favoured explanation for its development.[7] However conclusive evidence to support this theory has been difficult to collect because of the non-specific effect of sex hormones on target tissue, the synergistic relationships of proper ratios of sex hormones, the inhibitory effect when these proper ratios are altered, the in vivo transformation or conversion of one sex hormone to another and the deficiency of information regarding prostate hormonal relationships during the life span of the normal human male.

Hyperplasia of the prostate develops only in the presence of intact testes.[1] It has been noted that in castrated young dogs it is possible to induce prostatic hyperplasia by administration of 17-beta estradiol thus pointing to possible synergism between androgens and estrogens.[1]

II The Neoplastic theory

Postulates that the enlargement is a benign neoplasm.[6] As the prostate is composed essentially of fibrous tissue, muscle tissue, and glandular tissue, the neoplasm is a fibro-myo-adenoma.[6]

III Inflammation

Various inflammatory processes have been incriminated over the years.

John Hunter, as quoted by Murphy in 1786, thought that the prostate become enlarged in persons "of scrotufous habit". Gonorrhoea and non-specific prostatitis were causes put forward by Petit (1774) and Desault, and Syphilis was thought by Amussat and Swediou to be an aetiological factor.[4] Ciechanowski in 1901 as quoted by Murphy that hyperplasia was the end result of chronic prostatitis which caused scarring of the prostatic ducts, with resulting dilatation of the gland and cyst formation. Moore (1943) pointed out that "the changes interpreted as evidence of inflammation occurred in other organs, such as the thyroid and were evidence of endocrine control rather than infection.[4]

bIV Arteriosclerosis

Launois (1885 as quoted by Murphy considered that enlargement of the prostate was caused by the local effects of generalised arteriosclerosis. [4] Guyon as quoted by Murphy confirmed that most patients with bladder neck obstruction suffered from atheroma but thought that this was not the only causative factor.[4]

Flocks in 1937 and Moore, in 1943 were unable to correlate vascular changes with the occurrence of hyperplasia.[4]

V Congestion of the prostate

Venous congestion of the pelvis is one of the oldest theories concerning the causation of enlargement of the prostate.[4] Lose (1898) quoted Reliquet's observations of Haematuria and retention of urine being caused by the venous congestion of liver disease.[4]

VI Neoplasm

This resemblance of the nodules of prostatic hyperplasia to uterine fibro-myomata led to the theory that the two diseases were analogous, especially as it was erroneously believed that the prostate was the homologue of the uterus.[4]

In 1947, Charles Huggins as quoted by Murphy stated that "Benign prostatic hypertrophy in man consists of multiple spheroidal neoplastic nodules in the medullary portion of the prostate."

VII Endocrine theories

The demonstration by John Hunter as quoted by Murphy that hyperplasia of the prostate does not occur in "Castrates was the basis of the attempts of White (1893), Moulin (1892) and others to treat the condition by castration and vasectomy.[7]

Wilgmeister (1937) as quoted by Murphy maintained that oestrogen production was reduced in later life and thought that a preponderance of androgen might cause hyperplasia.

Hormonal imbalance is accepted by most as a factor in prostatic hyperplasia, but attempt to correct this have had little success.[7]

Recently, interest in the possible hormonal treatment of prostatic enlargement has been aroused by the report of Geller which reported relief of symptoms and atrophy of the prostate following long-term therapy with hydroxyprogesterone in a small series of cases.[7]

VIII Age

Age is accepted as the chief agent in the development of hyperplasia of the prostate, and little has been added to the statements of the master-surgeons of the last century.[7] "The enlarged prostate is the consequence of age and not disease (Sir Astlley Cooper 1831) as quoted by Murphy".[4]

CLINICAL PRESENTATION AND FINDINGS

The clinical picture of prostatic hyperplasia parallels these pathophysiologic changes. In those patients with early or very gradual prostatic enlargement, the disease produces no symptoms because the bladder is capable of compensating for the increased outlet resistance.[7]

There are no alterations in micturition, the bladder empties completely on voiding, and there are no symptoms of urinary tract difficulties. However, on digital rectal examination there is enlargement of the prostate gland. Such a situation is called "silent prostatism" and is discovered only inadvertently during patient evaluation for unrelated problems.[7]

The predominant symptoms associated with hyperplasia that prompt the patient to seek medical care are those of disturbances of urination secondary to the distortion and obstruction of the bladder neck and proximal urethra. Characteristically the patient complaints of poor stream, which worsens on straining, hesitancy in initiating voiding, a feeling of incomplete emptying of the bladder and occasionally, the inability to urinate. Incomplete evacuation of the bladder may be heralded by other symptoms. Such as increased urinary frequency, incontinence, nocturia, and as symptoms worsen nocturia may awaken the patient hourly.[5,7]

Residual urine predisposes to secondary infection with symptoms of painful mircturition or strangury becoming part of the clinical picture.[6] Likewise, rupture of dilated vessels coursing over the surface of the enlarged prostatic lobes produces gross haematuria, a less common but occasional integral part of the clinical picture.[7]

Depending upon whether the source of bleeding is an intravesical or intra-urethral prostatic lobe, the haematuria may be total, initial or terminal in nature. The enlarged prostate in itself produces few, if any, symptoms, however a large benign prostate, simply because of its size, may produce perineal aching or pain.[7]

A third group of patients present with symptoms that represent the secondary effects of the bladder neck obstruction rather than those of the local pathophysiologic changes discussed above. The clinical picture of this group of patients may be so unrelated to the urinary tract that it suggests primary disease in other body systems.[7] Asymptomatic bladder neck obstruction with bilateral hydronephrosis frequently mimics gastrointestinal or neurologic disease with its symptoms of nausea, vomiting, poor appetite weight loss, malaise, apathy and stupor, superimposed infection upon the chronically obstructed upper tracts produces

additional symptoms of chills, fever, general body aching, and upper abdominal or flank pain.

Occasionally the bladder neck obstruction is detected when the distended bladder or hydronephrotic kidneys are accidentally discovered by the patient or the physician during abdominal palpation.[7]

In summary, these three categories of patients those with no symptoms, those with local symptoms, and those with distant symptoms, comprise the composite clinical picture of bladder neck obstruction secondary to benign prostatic hyperplasia.[7]

Although these symptoms are commonly associated with hyperplasia they are equally pathognomonic of other disease entities. This is particularly true of increased frequency of voiding and dysuria. All too often disastrous results follow prostatectomy in patients whose symptoms stem from inflammatory disease rather than obstruction. Therefore it is extremely important that diagnostic measures be employed to establish the presence of hyperplasia and the role it plays in the patients clinical picture.[7]

DIAGNOSIS

On physical examination i.e. digital rectal examination; there is alteration in the palpable characteristic of the prostate gland. In this parameter one can evaluate the size, shape and consistency of the gland, the degree of tenderness, the contents of the

prostatic secretions, and the tone of the rectal sphincter. Hyperplasia produces smooth, firm, elastic enlargement of the gland.[7]

However, it is not possible to predict the degree of bladder neck obstruction from the size of the gland, particularly when the pathologic processes are located primarily in a subcervical variety of the median lobe. Obliteration of the median sulcus is usually an indication of median lobe involvement. Uncomplicated hyperplasia is non-tender although superimposed inflammation may render the gland sensitive to rectal palpation.[7] The spastic unihibited neurogenic bladder frequently presents with symptoms similar to those of hyperplasia. Failure to differentiate between these lesions often results in unfortunate post-prostatectomy results.[7]

Urinalysis In most cases is normal an abnormality occurs primarily with the development of secondary infection in the obstructed urinary tract. The urine is normally alkaline and white blood cells, red blood cells and bacteria appear in the urinary sediments. The urine is also used for evaluating the effects of BPH upon renal function through such tests as specific gravity, concentration, or dilution tests, excretion of phenosulfophthalein or other dyes, and the creatinine clearance.[7] Similarly examinations of the blood are useful in determining either primarily or secondarily

the level of renal function, through examining the blood urea, creatinine, calcium, phosphorus and various serum electrolytes i.e.: Na^+, K^+, Ct etc.[7]

Radiological examination of prostatic hyperplasia entails:- various techniques which are helpful in establishing the obstructive nature of the prostatic enlargement and its effect on the tract proximal to the point of obstruction, the tests are:

(a) **Cystourethrogram and Excretory urogram**

Various distortions of the prostatic urethra and bladder neck can be identified with either voiding or injecting technique provides better visualization because a greater concentration of contrast media can be instilled than that obtained by the excretory route.[7] This technique consists of four films: an anterior - posterior cystogram, an oblique air cystogram, and an oblique-opaque injections urethrogram. No roentgenographic examination that depends upon the use of contrast materials should be performed without a plain film to serve as a baseline reference point. In this particular examination, it also provides information regarding the degree of bladder distension, the presence of radiopaque bladder distension, the presence of radiopaque bladder or prostatic calculi, as well as abnormalities in the bony structures. Intravesical protrusions of the enlarged hyperplastic

prostate are visualized best on the air cystogram while intra urethral encroachment are seen.[7]

Three types of prostatic urethral distortions which occur with prostatic hypertrophy are: lateral lobe enlargements flattens the urethra in the lateral dimension but increase the anterior-posterior diameter so that an oblique view demonstrates a spreading of the prostatic urethral shadow.[7]

Secondly there is lengthening of the prostatic urethra, resulting from an increase in the superior-inferior dimensions of the prostate.[7]

Thirdly the roentgenographic alteration in the prostatic urethra results from hyperplastic enlargement of the median lobe, producing a forward angulation or anterior tilting, therefore one or more urethral distortions accompanying hyperplasia may be seen, depending upon the lobes involved in a given patient.[7]

The Cystourethrogram provides rough estimation of renal function as well as a visualization of such obstructive morphology as urinary changes as hydronephrosis, hydroureter, vesical trabeculation, and vesical diverticulum. If a sufficient dense contrast material collects in the bladder avoiding urethro-gram can be obtained at the end of excretory urogram following which can appraisal of residual urine; can be made from the retained contrast material.[7]

Residual urine can also be estimated by catheterizing the patient after he has made an effort to completely evacuate his bladder. The absence of residual urine, however, does not eliminate bladder neck obstruction since residual urine is an expression of bladder decompensation which appears only after the bladder has made an effort to overcome the obstruction through hypertrophy of the detrusor muscle.[7]

Cystoscopy Direct endoscopic visualization of the prostatic enlargement and vesical changes is extremely helpful in evaluating the degree of bladder neck obstruction and in selecting subsequent therapeutic measures.[5]

The administration of anticholinergic preparations to the patient with an unihibited bladder improves, in contrast to the patient with bladder neck obstruction secondary to prostatic hyperplasia, who develops greater difficulty in voiding or progressive increase in the residual urine that ultimately ends incomplete retention.[5]

- Ultrasonography is useful in that it will evaluate the amount of residual urine and give information regarding prostate size.[5] The sonogram also detects renal pathologic conditions and hydronephrosis.

- IVP (Intravenous Pyelography) is another classic method for evaluating patients with severe prostatism

and residual urine can easily be estimated with a post-voiding film.[5]

The kidneys and ureters are nicely evaluated by intravenous Pyelography, and hydronephrosis or any upper tract pathologic lesions can be detected.[5] The intravenous pyelography should be avoided in patients whose creatinine levels are above 3mg/dl because excretion of the contrast material will be poorly visualized and the information obtained inadequate. In addition, the contrast material may have some toxic effect on the renal tubules.[5]

TREATMENT

Conservative and medical measures of managing benign prostatic enlargement with bladder outlet obstruction are generally unsuccessful.[2]

Prostatic massage and urethral dilatation are of little value unless there is demonstrated substantial congestion and stricture formation respectively. Anticholinergic drugs and antihistamine should be avoided as they may precipitate urinary retention.

Occasionally oestrogen in small dosage may include minimal improvement in the urinary stream, presumably through the mechanism of some prostatic shrinkage but in general hormonal measures have been ineffective in BPH.[2]

SURGERY

The decision for surgical intervention in BPH is reached after evaluation of the following factors. Indications for surgery include:

(a) Residual urine more than 100 ml particularly when there is associated azotemia of any degree.[2,6]

(b) persistent or recurrent urinary infection refractory to usual therapeutic methods.[2] This problem was amongst the commonest in our patients, especially those who required an indwelling catheter.

(c) Gross haematuria on more than one occasion. Occasionally venous bleeding from a ruptured vein overlying the prostate will not stop with catheter, so prostatectomy must be performed as an emergency measure.[2,8] In this study 11 patients presented with haematuria, and out of the eleven two had gross Haematuria which necessitated blood transfusion. No emergency prostatectomy was done. These patients were fully worked up and underwent an open prostatectomy.

(d) Acute or chronic urinary retention with overflow dribbling or hydroureter or hydronephrosis demonstrated on urography and uraemic manifestations.[2,6,8] In this study this was amongst the commonest presenting symptoms, 47 patients presented with urine retention.

Other complications included stone formation, infection and diverticulum formation. There are four standard surgical procedures for removal of the obstructing enlarged portion of the prostate gland.[2] None of these procedures constitute total prostatectomy; all of them are designed for removal of the adenomatous hyperplastic portion of the gland.[2] Hence these procedures should most properly be termed prostatic adenoctomy rather than prostatectomy, since the true prostate, compressed laterally into a fibromuscular and acinar surgical capsule, is retained after removal of the central adenomatous elements.[2]

TRANSVESICAL AND RETROPUBIC PROSTATECTOMY

Prostatectomy by way of the transvesical and retropubic approach is still a standard procedure for the urologic surgeon.[9]

Open prostatectomy may be indicated when the weight of the adenoma is greater than 65 grams. Other indications are the presence of vesical calculi that cannot be crushed by lithotripsy or bladder diverticula that require surgical repair.[9] Orthopaedic deformities, particularly diseases of the hip joint, may mandate an open approach in so far as the patient cannot be placed in a sufficient position of abduction for transurethral resection.[9]

OPERATIVE TECHNIQUE

TRANSVESICAL PROSTATECTOMY

The patient is placed in a supine position with elevation of the proximal sacrum to tilt the pubis inferiorly and open the retropubic space.[8]

A vertical midline subumbilical incision is made through the skin, subcutaneous tissue, and linear alba. The rectus abdominis muscle is refracted laterally and the prevesical and retropubic spaces developed by gently sweeping the peritoneum and fatty tissue superiorly to avoid injury to the numerous veins coarsing on the bladder and prostatic capsule.[8] A vertical cystostomy is made large enough to allow inspection of the bladder and introduction of the enucleating fingers.[8]

The enucleating finger is inserted in the prostatic urethra to fracture the thin anterior prostatic adenoma against the undersurface of the symphisis pubis and develop the enucleation plane laterally.[8] The digital enucleation continues laterally, posteriorly, and anteriorly until only the membraneous urethra prevents delivery of the adenoma.

The adenoma is then retracted superiorly and the membraneous urethra transected with blunt-nosed scissors to prevent avulsion of the membraneous urethra or laceration of the prostatic capsule.[8]

The prostate is now withdrawn from the wound and inspected to determine if all the adenoma has been

removed. The prostatic fossa is packed with hot sponge to control bleeding while the bladder neck bleeding points are controlled with suture ligatures to 2/0 chromic catgut. These hemostatic sutures are placed routinely at e, 5, 7 and 9 O'clock. Whether or not the vessels are seen because the major arterial blood supply to the prostate enters here. The prostatic fossa sponges are removed and the remaining bleeding points suture ligated or coagulated.[8]

The prostatic fossa can be trigonalized by suturing the distal trigone to the midportion of the posterior surgical capsule. With interrupted 2/0 chronic catgut sutures. This decreases the area of denuded epithelium.[8]

A 22 F 30 cc Foley urethral catheter is inserted and the balloon inflated in the bladder.[8] A 24 F malecot suprapubic tube is placed through a stab wound in the dome of the bladder and brought out through a stab wound lateral to the abdominal incision.[8] The cystostomy is closed with running locked 2/0 chronic catgut or polyglycolic acid sutures, the closure checked for leaks and a prevesical Renrose drain brought out through a stab wound lateral to the abdominal incision.

Patients in whom poor wound healing is anticipated such as kidney transplant recipients, require a second layer of interrupted, absorbable seromuscular bladder sutures to reinforce the initial closure.[8] Wound is

irrigated free of clots and urine and the muscle and fascia are closed with interrupted nonabsorbable sutures No. 0. The subcutaneous tissue is closed with 3/0 absorbable sutures, the skin is approximated with monofilament nylon, and the drain and suprapubic tube sutured to the skin with nonabsorbable sutures.[8]



Figure 6.I shows a prostate which weighed 300 grammes

RETROPUBIC PROSTATECTOMY

The patients position and exposure of the prostate and bladder are the same as described for the transvesical prostatectomy.[8]

An incision is made vertically from the midportion of the prostate up to the bladder neck. There, may be immediate capsular bleeding requiring sutures plane of enucleation is then developed on either side by inserting the tip of the dissecting scissors into the line of separation between surgical capsule, and adenoma and spreading the tips of the scissors outward to develop the plane. The forefinger is then introduced and the line of enucleation developed on either side of the vertical incision. The adenoma is then digitally enucleated distally, laterally, and posteriorly.[8]

One must be careful to avoid tearing the surgical capsule, particularly in fibrous or carcinomatous glands. Undue difficulty in separating the adenoma should increase the suspicion that there is indeed a malignant element in the prostatic enlargement.[8] After retraction of the adenoma upward, its attachments to the membraneous urethra are transected with blunt nosed scissors. The adenoma is then delivered through the capsulotomy and the cone of attached bladder neck mucosa is incised, completing the enucleating.[8] In large prostate it may be advisable to remove first one lateral lobe and then the other. The median lobe, if present,

usually remains attached to one or the other of the lateral lobes. Absorbable hemostatic neck sutures are placed at 3,5,7 and 9 O'clock and prostatic fossa bleeders are coagulated or suture-ligated.[8]

At 22 F 20 cc Foley catheter is inserted into the urinary bladder and the balloon inflated. The prostatetomy is closed with running locked 2/0 chromic catgut or polyglycolic acid. The wound is then drained and closed as in the transvesical prostatectomy.

III TRANSURETHRAL RESECTION

The technique of transurethral prostatectomy was developed in the 1930's largely at the University of Iowa, the Mayo clinic, and the University of Michigan and was perfected and accepted as a useful operation by the early 1940's. During the ensuing years it has gradually displaced the open procedures in most instances.[10] At our centre it is the operation of choice in the Urology unit.

As with all surgical procedures trans-urethral resection of the prostate is not the operation of choice in all patients with prostatism.[10] Since a satisfactory prostatectomy must remove all of the second growth of the prostate to the surgical capsule. The size and character of the gland may require an open technique.[10] Glands large than 75 gm, to be comfortably excised, should be removed by an open operation.[10]

The technique of transurethral resection is probably the most difficult of all surgical procedures to master and the resident and teacher require great patience as it cannot be learned by watching, as it requires coordinated use of the eyes, hands and feet. The reward of mastery is an operation with exceedingly minimal morbidity and mortality and excellent results.[10]

SURGICAL PROCEDURE

At the time of surgery the operating table should have the instruments in an orderly fashion to facilitate the surgical procedure.[10] The fibreoptic lighting system has been a great step forward and more recently, the Hopkins lens system gives a better view of the operative field.[10] Unless contraindicated by spinal or neurologic diseases, the anaesthetic. The relaxation of the perineum and pelvis is far superior to other anaesthetics, and the anaesthetist can communicate with the patient to be sure that an unrecognised problem such as a myocardial infarction, rupture of the bladder or abnormal absorption of fluid with secondary hyponatremia and disorientation has not occurred.[10]

In this study quite a number of patients had spinal anaesthesia 17, and 11 had Epidural anaesthesia.

The commonly used fluid for resection is isotonic 1.5% glycine; this is a fluid used also at our centre. The surgical technique of resection may use a three stage procedure.

First resecting the vesical neck down to the fibres of the internal sphincter then the mid fossa, and finally the apex of the prostate removing all the abnormal tessile to the external sphincter. Rarely is it necessary to do a second stage resection, but if one has misjudged the size of the prostate or there are other externuating circumstances, the resection can be stopped at any time and carried out later at a better time.[10]

IV PERINEAL PROSTATECTOMY

This procedure was never used in this study. Crude perineal prostatic surgery was, however, not displaced by more precise techniques until the start of the twentieth century.

Proust 1901 and Young 1905 provided the basis for the improvements embodied in the methods of Haun 1936 and finally, Belt 1939 as quoted by Hudson.[11]

In perineal prostatectomy a four man surgical team is the most ideal i.e.

- third assistant stands on the patients left side in order to hold the left lateral retractor in his right hand and the rectal retractor in his left hand.[11]
- second assistant stands on the patients right side to direct the right lateral retractor with his right hand. In the phases of the dissection, he rests his left wrist on the patient's symphisis to keep the curved prostatic tractor immobile as a guide and

counter pressure for the surgeons entering dissection and exposure of th dorsal prostate.[11]

- First assistant stands on the side opposite the instruments nurse, at the discretion of the operating surgeon. He assists in maintaining exposure and directing the lesser-assistant, as well as participating in the excise and reconstruction.

The surgeon performs the operation and notifies the assistants of his anticipated needs.[11]

The surgical technique

After positioning the patient so that the perineum almost horizontal and with the buttocks extending several inches off the surgeon's end of the operating table. After sterile preparation and draping of perineum and genitalia are left exposed as the visible operative field.

Next the curved prostatic retractor is introduced through the urethra, opened and held at approximately 30°degrees angle to the abdominal horizontal plane in order to place the prostate on tension toward the perineum and immobilise it when the surgeon is satisfied with the position of the retractor, it is then held in the left hand of the second assistant until he is instructed to remove it later in the operation.

The semicircular surgical incision is then made through the skin with a small round bladed surgical scapel. The subcutaneous layers of fascia and fat are

divided in the line of incision. Then the central tendon is identified, elevated and divided in a systematic way that rarely requires modification. This makes it possible to identify the distal border of the maroon coloured deep portion of the external anal sphincter. The sphincter is elevated to expose the levator ani muscles and ventral rectal wall. After the levator ani muscles have been separated in the median line, they are retracted laterally to expose the dorsal prostate. Prostatetomy, either with or without a biopsy specimen, is then performed. After this, the line of cleavage is begun between the dorsal prostate and the tissue to be removed. Control of bleeding under direct vision contributes to the safety of the operation. With the prostatic enlargement partially freed, the prostatic urethra is identified and divided. The curved urethra retractor is removed and a straight prostatic retractor substituted for it the surgical specimen is freed at its distal tip. Fibers of the bladder neck or internal sphincter are identified, and hemostatic sutures are placed to prevent bleeding.[11] In many cases, this step completes the excisional phase of perineal prostatectomy. However if the median prostatic lobe has an extension beneath the trigone, retraction of the bladder neck is readily achieved to permit safe removal of the subtrigonal hyperplastic prostatic tissue.[11]

After removal of the prostate then the perineum is reconstructed. First the ventral suture to approximate the urethra to the bladder neck is placed and tied.[11] Similar sutures are placed and tied after theurethral catheter has been advanced into the bladder. Finally, the dorsal prostate is closed. Closure of the surgical incision is performed by reuniting the levater ani muscles in the median line, then the more superficial structure.[11]

Other modalities of treatment are: Surgical removal of the obstructing prostatic tissue has been in widespread use for over 50 years and remains the principal therapeutic choice. In recent years it has been increasingly recognised that conventional surgery is not without its dangers, particularly in the elderly, unfit patients.[12]

A number of alternative, minimally invasive therapeutic options have been explored in recent years, but none of these has so far been as effective.[12]

This is of particular importance in the elderly infirm patient who may not be able to comply with or tolerate pharmacological therapy and where the only previous alternative has been on long-term catheter drainage.[12,13]

Some recently reported techniques used to relieve bladder outlet obstruction are:

1. Urethral stent: this is implanted in the urethral.[12,13,14,15] The stent is introduced using a delivery system, local anaesthetic jelly is inserted into the urethra with a concurrent use of a sedative.[12,13,14,15]

The patient is placed in the left lateral position and transrectal ultrasound probe is inserted per rectum. A flexible cystoscope is carried out to exclude bladder tumours and stones and to confirm the position of the bladder neck and distal sphincter mechanism. On the ultrasound pictures. The distance between the bladder neck and sphincter mechanism is then measured.[16]

A guide wire is passed into the bladder and the flexible cystoscope withdrawn. The stent is available in varying sizes. the length chosen depending on the distance between the bladder neck and distal sphincter mechanism on the ultrasound image. A mesh prosthesis of appropriate length constrained in its compressed form on the delivery catheter, was then passed over the guide wire. The stent is initially positioned. With its proximal end just within the bladder and the delivery catheter is then pressurised with normal saline through a side arm to 3 atmospheres to separate the 2 layers of the plastic membrane holding the stent in its compressed state. The outer layer of the plastic membrane is withdrawn approximately 2 cm, thereby enabling the proximal end of the stent to open, and further opening

of the prosthesis is further arrested by depressuring the apparatus.[12,13,14,15]

The stent is then cautiously withdrawn over the guidewire, allowing its partially open proximal end to be accurately positioned at the bladder neck, under ultrasound control. This partial opening of the stent allows it to grip the tissues of the bladder neck and thus provide anchorage. When the operator is satisfied with the positioned of the stent, the membranes are repressurized and the outer sheath is fully withdrawn allowing full release of the stent. The delivery catheter is then removed. The unconstrained calibre of the stents is 14 mm. If the stent did not open to 12mm diameter during this procedure, balloon dilatation is carried out using a 12 mm diameter balloon passed over the guidewire. The position of the stent is then confirmed endoscopically using the flexible scope.[12,13,14,15]

Another procedure is the use of prostatic balloon dilation uses a low compliance, fixed diameter balloon inserted transurethrally and positioned under radiological control or under direct vision using cystoscope.[13,14] Once in the prostatic urethra the balloon is expanded to a pressure 300 - 400 kPa for periods of 10 - 15 mins. This is said to lead to disruption of the prostatic commissures and possibly to stretching of the prostate capsule with resultant

decompression of the prostatic urethra.[13,14] The exact mechanism of action is questionable.[13,14] Operative commisurotomy which could be expected to produce the same effect, is claimed to produce improvement in flow rates in 91% of patients, but this does not hold truth for prostatic dilatation.[13,14] It has been demonstrated that splitting of the commissures did not correlate with relief of retention or improvement of urinary flow.[13,14] Neither tissue necrosis nor atrophy appear to be important factors in the reduction of prostatic resistance obtained by dilatation with only small reduction in prostatic volume being noted following dilatation.[13,14]

Beside the stents and balloon are prostatic spirals.[14,16,17] This consists of a tightly coiled metallic spring which lodges in the prostatic urethra. It has a tail which traverses the external sphincter allowing the device to be grasped should it need to be removed.[14,16] In contrast to the necroporous mesh the device does not become epithelialized and thus remains a foreign body in the urethra, as long as it remains in place. The internal diameter of the spiral is narrower than that of the mesh and does not allow endoscopy of the bladder when in situ.[14,16]

The spiral is inserted endoscopically under local anaesthesia with visual positioning.[14] Nordling et al as quoted by Mcloughkin J. reported that it is the most

recently used ultrasonically guided placement.[14] The spiral can easily be removed, also under local anaesthesia, by grasping the tail with cystoscopic forceps.[14, 17]

Local deep microwave hyperthermia has been introduced recently for the treatment of Benign Prostatic hyperplasia.[17] Few studies have been done with few outcome results not conclusive yet.[18]

COMPLICATIONS OF PROSTATECTOMY

(i) **Local Haemorrhage:** Reactionary haemorrhage is the most tiresome complication following prostatectomy whatever the surgical approach.[7] If the blood is not adequately washed out clot retention will ensue. One should always check that the bladder is adequately decompressed for the distended bladder and a catheter that is not draining indicate that a clot is blocking the eye of the catheter. The bladder should be promptly washed out using strict aseptic technique. Only rarely is it necessary to return the patient to the operating room.

In this study 26 patients had reactionary haemorrhage, and 10 of them required extra blood transfusions, the rest were resuscitated with i.v. fluids and bladder decompression and responded well.

(ii) Secondary haemorrhage: This occurs around the tenth post-operative day, and is usually associated with the patient over-exerting himself or the presence of a

urinary infection.[7] If the bleeding is not severe and there are no clots the patient is simply advised to rest; and encouraged to have a high fluid intake and is given a broad spectrum antibiotic. In this study none of the patients had secondary haemorrhage. Primary haemorrhage is common with prostatectomy also.

(iii) **Perforation**: Perforation of the bladder or the prostatic capsule can occur at the time of transurethral surgery.[7] This usually occurs in association with a large prostate or heavy blood loss.[7] If the field of vision becomes obscured by heavy blood loss it is often prudent to achieve adequate haemostasis and abandon the operation.[7]

In this study only two patients had transurethral resection and none of them had complication of perforation neither haemorrhage.

(iv) **Infection**: Infection whether in the bladder epididymis or the kidneys should be treated with appropriate antibiotic.[7] Wound infection following open prostatectomy is common if a urethral catheter has been in situ for a number of days before the operation. In this study wound sepsis was the commonest complication as majority of our patients had indwelling catheters for at least more than three weeks before prostatectomy. Most of them had urine culture before operation so that after surgery they were given antibiotics according to sensitivity pattern of the culture bacteriology.

(v) **Incontinence**: Incontinence is inevitable if the external sphincter mechanism is damaged. The bladder neck is rendered incompetent by these operations and therefore an intact external mechanism is essential for continence.[6] Drainage to the sphincter may occur at open prostatectomy and following transurethral surgery if the resection extends beyond the verumontanum. The outcome for these patients is naturally disastrous. Urodynamic investigation has shown that not all of these patients with incontinence have sphincter damage. Some will have unstable bladders with inappropriate bladder contractions contributing to the incontinence. The use of powerful anticholinergic agents or imipramine may help.[6]

In this study 21 patients had urine incontinence which stopped within a period of 1 - 6 months.

(vi) Retrograde ejaculation and impotence. All patients having a prostatectomy should be warned that they are likely to suffer from retrograde ejaculation; this occurs once the bladder neck is rendered incompetent.[6] A proportion of men will also claim that the operation is the final insult on their already waning sex life.[7]

(vii) **Stricture**: Stricture may occur secondary to prolonged catherization, the use of an unnecessarily large catheter, clumsy instrumentation or secondary to the presence of the resectoscope in the urethra for too long a period. An early stricture can usually be

managed by simple bourginage but later on it may be necessary to cut the densely fibrotic stricture with the optical urethrotome. Many surgeons claim the routine use of an otis urethrotomy reduces the incidence of Post-operative stricture.[6]

(viii) **Bladder neck contracture**: Justoccasionally a dense fibrotic stenosis of the bladder neck occurs following the resection of a small prostate. It may be due to the over use of a coagulating diathermy. Re-resection of the scar tissue is necessary.[6]

(xi) **General-Cardiovascular**: Pulmonary atelectasis, pneumonia, myocardial infarction congestive cardiac failure, and deep venous thrombosis are all potentially life threatening conditions that can effect this elderly and often frail patients.[6]

It is particularly for this group of complications that the transurethral route of surgery which saves the need for a painful abdominal scar and permits early ambulation, has proved of benefit.[6]

(x) **Water intoxication**: The absorption of water into the circulation at the time of a transurethral resection can give rise to congestive cardiac failure, hyponatremia and haemolysis. Accompanying this there is frequently contusion and other cerebral events often mimicking a stroke. The incidence of this condition has been reduced since the introduction of isotonic glycine for performing the resection and the use of isotonic

saline for post-operative irrigation. The treatment consists of diuretics for the fluid overload and digoxin may be necessary. Cautious replacement of intravenous fluids should be with isotonic saline.[6]

(xi) Osteitis pubis: May occur but rare. This is possibly due to pricking of the periosteum during operation or to infection in the prevesical space.The patient presents with pain and tenderness at the pubis with inability to walk.[6]

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MANAGEMENT OF MALE URETHRAL STRICTURE AT MUHIMBILI MEDICAL CENTRE

SUMMARY

A total of 98 patients with urethral stricture were attended in the urology unit at Muhimbili Medical Centre from October, 1990 to September, 1991, a period of 12 months.

The age range was 3 years to 80 years, with mean age of 40 years.

Out of the 98 patients 52 were due to inflammatory causes, 29 traumatic, 11 iatrogenic and 6 due to congenital causes.

Diagnosis was made by history, clinical examination and urethrogram.

Treatment included bourginage, meatal dilatation, meatotomy and urethroplasty. Out of the 98 patients 18 required a multistage urethroplasty.

INTRODUCTION

Urethral stricture is one of the commonest urological conditions seen at the urology unit. As noted in this study during a period of one year 98 patients were seen with the problem of urethral stricture.

Strictures according to literature may be congenital, iatrogenic, traumatic or inflammatory.[1] Inflammatory stricture is a rare problem in developed

countries, but it still poses a problem in developing countries.

Congenital urethral stricture occasionally occurs in male infants.[2] A congenital stricture may be single or located at multiple sites anywhere along the urethra and is reported to be due to embryologic narrowing of the channel or failure of tube formation.[1]

The two most common are in the region of the corona (fossa navicularis) and in the membranous urethra.[2]

The literature on urethral stricture in boys is sparse suggesting that the condition is uncommon. In this study there was a higher prevalence in the middle age group ranging from 30 to 50 years.

In the female, the urethra is rarely injured except during child birth or surgery.[3] **Diagnosis:** Specific investigation such as urethrography helps in locating the site and length of the stricture. Urethroscopy renders the diagnosis of urethral stricture very precise, at the same time its dilatability can be determined.

Treatment of the urethral stricture varies from dilatation to urethrotomy (external or internal) to urethroplasty.

Complications of urethral stricture include: urine retention, urethral diverticulum, periurethral abscess, urethral fistula, hydronephrosis resulting from back pressure.

CASE REPORT

M.M. Inpatient number 658598 was a 16 year old boy referred from Zanzibar. Two weeks prior to admission he presented to Zanzibar hospital complaining of difficulty to pass urine. Urethral catheterization failed and he had suprapubic cystostomy done, then he was referred to our hospital.

The patient gave a history of falling from a mango tree injuring his perineum five months prior to the onset of the above problem. He had slight urethral bleeding for two days then the bleeding stopped. He was treated with antibiotics at a nearby dispensary. Three months later he noted his urine stream was becoming poorer and occasionally he had to strain to improve the stream, this problem was progressive until five months later when he had acute retention of urine.

On physical examination he was a healthy-looking boy, he was not pale and he was afebrile, his cardiovascular and respiratory system were normal. He had a suprapubic Catheter, normal genitalia, he had no urethral discharge or obvious lesion.

Several investigations were done including:

- Haemoglobin which was 12.8 g/dl
- Urinalysis - S.G 1.010
  - No pus cells
  - No red blood cells
- Urethrogram was done:

Revealed a tight stricture at the bulbar urethra. No extravasation of the contrast.

The patient was then taken to theatre for trial of bourginage. He was given general anaesthesia under aseptic condition. Bourginage was tried with filiform bourge size 6 which was the smallest, but could not pass through, even the pilot could not pass through. The stricture was noted to be impassable at the bulbo-urethral level. Another bougie was passed through the suprapubic cystostomy and could be felt in the perineum. He was then planned for urethroplasty.

He was later taken to the main theatre and was given spinal anaesthesia. Under aseptic conditions a U bougie was introduced into the suprapubic stoma and the tip palpated in the perineum. Lister's bougie was introduced through the urethra up to the point where it got stuck. With the guidance of the two bourgies in place an inverted U incision was made in the perineum. The bulbospongiosus muscles were identified. The proximal portion of the urethra was opened with a guidance of the bouge. Then the skin flap was sutured on the distal end of the proximal urethra and the sides of the flap were also sutured around the whole opening with Dexon suture No. 3/0.

Two catheters were left in situ after one week the distal catheter was removed and the suprapubic catheter

spigotted and patient was instructed to avoid using the perineal stoma.

Post operatively he was covered with Erythromycin tablets 500 mg 8 hourly for 5 days and Flagyl 400 mg tablets 8 hourly for 5 days.

He recovered well, four months later he was scheduled for 2nd stage urethroplasty.

The patient was given spinal anaesthesia. The urethra was inspected and patency was confirmed by Hegars dilators. An oval incision was made around and including the two stomas, skin undermined on either side of the incision, then closed in 3 layers over a urethral catheter.

Post-operatively the patient faired well. The suprapubic catheter was removed 1 week post-operatively after ascertaining that the second operation has healed well. The suprapubic stoma closed spontaneously. The urethral catheter was removed after 2 weeks, he was voiding normally. He was discharged, and when seen again at the out-patient clinic 2 months later he was doing well.

The following is the analysis of the 98 patients.

Table I: AGE DISTRIBUTION TABLE

Age (Years)	Number of Patients	Percentage
1 - 10	10	10.2
11 - 20	8	8.2
21 - 30	14	14.3
31 - 40	16	16.3
41 - 50	13	13.3
51 - 60	15	15.3
61 - 70	17	17.3
71 - 80	5	5.1
Total	98	100

There was a higher frequency after the age of 20. This is due to regard to the high frequency of trauma to the urethra.

DISCUSSION

AETIOLOGY

In the male, injuries of the urethra may be caused by foreign bodies, external trauma (mechanical), internal trauma (instrumental), or spontaneous rupture and extravasation proximal to a urethral stricture. The natural history and treatment required in these types of injury differ considerably.[3]

i) **Inflammatory:** Strictures are considered infectious when they occur after clinical urethritis without any history of trauma or instrumentation. An infectious

narrowing is commonly found in the penile or bulbous part of the urethra because there are many crypts and glands in these areas where bacteria may lodge and multiply.

ii) **Iatrogenic strictures**: These are strictures occurring after endoscopy, surgery, or traumatic catherization. Such strictures are usually located distal to the bulbous urethra.

iii) **Pelvic Fractures:** These may be caused by a car accident resulting in a stricture of the membranous urethra, while straddle injuries cause a stricture of the bulbar urethra.[1]

iv) **Prostatic Surgery**: Urethral stricture is the commonest late complication of transurethral prostatectomy, the incidence in published series varying between 4 and 12% (data by Warres [4] 1958). Several measures have been suggested to prevent stricture formation including perineal urethrotomy pre-operative urethral dilatation and internal urethrotomy. Although internal urethrotomy is widely practised, there have been no controlled trials published to assess the efficacy of this procedure.[4]

In this study the aetiological factors are shown in Table II below

Table II: Aetiology of urethral stricture at Muhimbili Medical Centre

Aetiology	Number of patients	Percentage
Inflammatory	52	53.1
Traumatic	29	29.6
Iatrogenic	11	11.2
Congenital	6	6.1
Total	98	100

In this study the commonest aetiological factor was inflammatory, over 50% of the patients, and trauma ranked second, 29.6% of the patients.

Pathogenesis and Pathology

A severe degree of stenosis causes changes typical of obstruction. These include:

a) Dilatation of the urethra proximal to the stricture.[2]

b) Ectasia of the prostatic ducts

c) Compensatory changes in the bladder musculature.

d) Hydroureteronephrosis secondary to hypertrophy of the ureterotrigonal complex or vesicoureteral reflux.

Because of stasis, infection occurs that may cause periurethral abscess, urethrocutaneous fistulas, prostatitis, cystitis and pyelonephritis. If the organism splits urea, vesical or renal calculi may form.

Urethral stricture, then may cause severe damage to the urinary tract.[2]

Post-gonorrheal stricture, which has become very much less common since the introduction of the antibiotic treatment of gonorrhoea, is situated most frequently (a) in the bulbar (70%) (b) the peno-scrotal junction. (c) in the distal part of the spongy urethra, in that order. The membranous and prostatic parts of the urethra are exempt.

In this study the commonest site of stricture was at bulbar urethra 41.8% followed by multiple sites which was 21.4% as shown in table III below.

Table III: SITE OF STRICTURE

Site of Stricture	Number of patients	Percentage
Bulbar	43	41.8
Multiple	21	21.4
Penile	12	12.4
Meatus	10	10.2
Prostatic	7	7.1
Membranous	5	5.1

CLINICAL FEATURES

The commonest symptom is gradual diminution of the force and caliber of the urinary stream. Sudden urinary retention may occur if an infection at the site

of stricture is exacerbated. A history of urethral injury or severe untreated gonorrhoea can usually be obtained. Symptoms of cystitis may be noted. There may be fever secondary to prostatitis or pyelonephritis.[2]

Signs: Include periurethral induration which may be found at the site of the stricture. A tender mass may be present if periurethral abscess has developed. Perineal urinary fistulas may be noted. A visible palpable bladder may be found if urinary retention has supervened. Prostatic massage and culture of the secretions may reveal evidence of prostatitis.[2]

INVESTIGATIONS

Some of the basic investigations done in patients, with urethral strictures, which are also commonly done at our hospital include:

i) **Urinalysis**: In case of infection will show pus cells, bacteria and also the white blood cells will be elevated. If there is renal damage then the urinary Phosphate excretion may be diminished.

ii) **Radiogical**: examination include urethrogram and voiding cystourethrogram which will reveal the site and degree of the stricture.

Fistulas may also be demonstrated. After urethral dilatation a cystogram may show a thickened, trabeculated bladder and possibly, ureteral reflux. Excretory urograms may reveal urinary calculi or changes 217compatible with healed pyelonephritis.[2] If the

urethra does not accept a catheter, retrograde urethrogram should be performed.[1,2] Which will indicate the site and sometimes the degree of injury.[1,2,3]



Figure 7.1 A urethrogram showing urethral stricture



Figure 7.2 The same photograph as in 7.1 showing persistant narrowing at bulbous urethra

Instrumental examination The diagnosis of urethral stricture may also be confirmed by the passage of a metal bougie or catheter. This is likely to be arrested in the region of the bulb, when the characteristic "grating" sensation will be felt from contact with the hard fibrous tissue of the stricture.[5]

Differential diagnosis

It is important to know the other conditions which present with similar symptoms and signs as urethral stricture. For example prostatic or bladder neck obstruction may cause similar symptoms but doesnot

impede passage of a urethral catheter. An enlarged gland is usually found on rectal examination. Another condition is carcinoma of the urethra which can mimic urethra stricture, but panendoscopy and biopsy of the visualised tumor will establish the proper diagnosis.[2]

TREATMENT

The multitude of procedures in use for the treatment of urethral stricture indicates that none is fully satisfactory. Dilatation brings only temporary relief, carries the risk of urinary infection and subjects the patient to regular treatment and supervision. The operative methods for the treatment of urethral strictures are complicated andsubject to complications. In particular there is the risk of impotence if the operation is performed from the perineum.[6]

Several studies have been done by several authorities concerning urethral repair. And people have come up with different opinions concerning optimal time for stricture repair. For example Turner-Warwick[7] reported good results with immediate exploration and primary urethral repair, others like Blandy as reported by Kernohan et al had equally good results by immediate suprapubic drainage and late urethroplasty after traumatic injury.[7]

The four components of the urethra:- It is important to recognise the four regions of the urethra

each require different consideration in their repair; the glans-meatus, the penile, bulbar, and the sphincter - active posterior urethra.[8]

The reconstruction or the creation of a cosmetically and functionally perfect glans-meatus requires meticulous attention to detail. The reconstruction of the pendulous, free draining penile component of the spongy urethra is relatively simple compared with that of the bulbar region which is both more difficult technically and more critically demanding.[8] Reconstruction of the posterior prostato-membranous) urethra is further complicated not only by the problem of surgical access but also by the fact that its whole length is sphincter-active: a real video-urodynamic understanding of the mechanisms and an appropriate adaptation of procedures are therefore fundamental to a satisfactory functional reconstruction in this area.[8]

The four regional procedures can be used in appropriate combinations so that full-length urethral reconstruction can be achieved when necessary.[8]

(a) **Bouginage** Before subjecting the patient to operative treatment many of our patients were tried on bouginage.

The use of graduated bougies in dilating the urethral stricture is a standard treatment at the present time.[5] The metal bougies are usually preferred.

Owing to their rigidity the beak is under absolute control, and when used with the gentleness that is essential they are not more traumatizing than plastic bourgies.[5,9]

Other types of bougies include Gum-elastic bougies (French bougies), filiform bougies varying in size from 1 to 3 French. By means of gum-elastic bougies a stricture is dilated up to the size of 14 French. Thereafter metal bougies are to be preferred.[10]

Some of the strictures dilated by bougies were very tight strictures it was not possible to negotiate even the smallest bougies. These patients were subjected to urethroplasty. For those whose strictures could be bouged were dilated at an interval of 2 to 3 times a month to one year.

(b) **Urethrotome** As early as 1854 an urethrotome had been developed by Maisonneuve and later by Otis in 1872, which are still used in their original form.[6] Transurethral urethrotomy has been frequently critisized mostly because it was done without direct vision. The consequence was often on incomplete incision of urethral strictures as well as secondary injuries. To avoid the complications of "blind" urethrotomy, the incision of strictures under vision was tried very early. Ravasini reported this method in 1957, using transurethral diathermy incision for circular strictures.[6]

Transurethral incision is indicated for every kind of urethral stricture (even with children) and it is also suitable for the treatment of total urethral obstruction, as is occasionally observed after complete urethral rupture.[6]

(c) **Urethroplasty** Urethroplasty is applicable to all strictures of the bulbar and penile portions, and is the treatment of choice if simple measures fail.[10]

Johanson's urethroplasty: The urethra is exposed by a mid-line incision in the skin overlying the stricture. A wheelhouse staff is used to open the normal urethra for 2 cm distal to stricture. Three to four months after the procedure when wound contraction is complete then the urethra can be reconstructed. A dorsal relieving incision may be necessary.[10]

Where the stricture is in the bulb: the first stage is the same as above. The urethra floor is divided up and into normal urethra. In many cases this entails splitting the urethra through the external sphincter up into the wide prostatic urethra. In order to stitch skin between the edges, either a perineal skin flap has to be constructed at the time of skin incision (Blandy), or a scrotal tunnel (Turner-Warwick) displaced backwards to be sutured to the open edges of the urethral defect. a second stage closure is simple, a complete tube being reconstructed. A small catheter should be left in the urethra after these operations for 10-14 days. In this

study the treatment offered to the various patients is summarised in Table IV below:

Table IV: TREATMENT OFFERED

Treatment	Number of Patients	Percentages
Bouginage	92	93.9
Urethroplasty	18	18.4
Meatal dilatation	10	10.2
Meatotomy	6	6.1

COMPLICATION

1. **With Bouginage:** False passages may be created especially the meatal bourgies resulting from unskillful attempts to pass a bourgie.[10]

2. **Urethroplasty:** The complications of urethroplasty may be very serious and include impotence, infection, unsatisfactory cosmetic results. In addition, urethroplasty is taxing for the patient involving long periods of anaesthesia and hospitalization, especially with two stage procedures. By comparison the complications of transurethral incision are quite insignificant and this fact should be adequately considered.[6]

3. **Other complications include:** Catheter-balloon complications in a recently reconstructed urethra.

(a) Spontaneous balloon deflation may result in the premature removal of the catheter and this, or the need to replace it, may compromise some reconstructive procedures.

(b) Partial deflation of the balloon may result in it slipping, still oversized into the area of a reconstruction and damaging it.

(c) Inadvertent severe traction may withdraw a fully inflated balloon into the reconstructed area and disrupt it.

(d) Even when fully deflated, residual corrugations, of an overstretched balloon surface may be sufficiently rough to disturb a skin graft urethroplasty. Therefore when a urethral catheter is used it is best retained by a transvesical sling stitch to a crush button on the abdominal wall.

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HAND INJURIES

SUMMARY

A total of 37 patients were admitted in the orthopaedic and trauma ward, following hand injuries, in the period of seven months. (from 1.8.90 to 28.2.91). During the same period total admission including other injuries was 382 patients.

Seventeen (40%) patients sustained hand injuries while operating machines in factories and industries, whereas ten (27%) patients were injured in motor traffic accident. Six (16%) were assaulted and four (10%) had various domestic injuries. The injuries ranged from superficial cuts, partial loss of digits to loss of the whole hand.

The magnitude of trauma sustained was evaluated clinically and with aid of X-rays.

Surgical toilet to all patients were done within hours of injury and some required skin grafting later on. Secondary skin grafting was carried on some of the patients who had extensive skin loss during the time of accident. Results were better for those patients who reported to hospital in less than 12 hours of injury. There were lesser septic wounds for those who sustained injury following motor traffic accident, than those of machine injury.

INTRODUCTION

Injuries to the hand are extremely important. The majority of hand injuries occur to the otherwise young and healthy individual and thus have enormous implications for occupations, home life and future career choices.[1]

Mutilating injuries of the hand commonly occur as a result of industrial accidents, negligence and inexperience are main contributory factors to these accidents.[2] Domestic power tools are now common. Failure to recognise the risks involved in the use of such appliances has become a hazard, particularly to the junior members of the household, with disastrous consequences, Arabi and Sabet as quoted by Cardoso et al reported 22 cases of severe mincer injuries of the hand in children in Saudi Arabia.[2]

The quality of the primary care, more than anything done subsequently will determine the final result. The best example of this is the patient with a flexor tendon injury at "no man's land" no secondary reconstruction no matter how skillfully done and sophisticated, can recover the function irrevocably lost by inappropriate primary treatment.

Although often considered to be minor surgery, the so called "finger tip" injuries are in reality of great importance to the patient. They can happen to anybody, at any place, and in any circumstances connected with

the use of the hands. As a matter of fact, the extremities of the digits are the most exposed parts of the hand and are prone to all kinds of accidental injuries. Industrial workers, mechanics, butchers and carpenters are particularly vulnerable.[3] This anatomic region is the most precious part of a digit, as it not only completes the grasp in prehension, but also contains sensory nerve endings that allow the blind to read with the pulps of the fingers and musicians to transmit harmony by their skill, for then, the slightest sequela to an injury is important: Skin loss, poor healing, painful scars, deformities of the distal phalanx, and persistent deformity of the nail considerably impair the function of the affected finger and may jeopardise use of the hand professionally.[3]

Consequently, not only specialized hand surgeons and general surgeons working in an emergency service, but also general practitioners should be interested in the care of such lesions. In the case of partial or total amputation, microsurgical techniques may be applied.

The pulp has an excellent arterial vascularisation with the two collateral arteries joined by a transverse arch from which many arterioles branch out distally (rete arteriosum). There is also a dorsal branch for the nail bed with transverse branches, one along the matrix and arcus unguicularis proximalis) and a second

one crossing the middle of the nail bed (arcus unguicularis distalis). Between them are numerous arteriolar junctions and small distal arterioles, which secure an excellent blood supply to this region. The sensibility of the pulp is furnished by two or three branches of the collateral nerve, which can be followed by microsurgical dissection along a few millimeters.[3]

The most frequent of all injuries, those to the hand, commonly involve tendons. When inadequately or inappropriately treated, tendon injuries lead to serious incapacity of the involved hand.[3] In his classic book on surgery of the hand Sterling Bunnel writes that "one of the most baffling problems in surgery is to restore normal function to a finger in which the tendons have been injured". In the 35 years since those words were written, there has been much improvement in the management of tendon injuries; yet several problems remain to be solved. Tendon gliding is a complex, the solution of which may involve the control of still unknown mechanical and biochemical factors.[4]

CASE REPORT

M.S. IP. No. 640497 was a 26 year old male patient who presented to the casualty department with history of injury to his right hand while operating a machine about 3 hours prior to arrival at hospital. Soon after injury he received first aid treatment at his dispensary, where they bandaged the arm to stop bleeding.

The patient was a machine operator in a cooking oil factory. His arm was compressed by a moving part which is normally operated manually. One presses a certain part with a foot and puts in metal sheets which are embedded by the machine, so one presses the foot part after putting in the sheet properly. In this case, the patient pressed the foot part before removing his hand and sustained cuts to his fingers. There was no history of other injuries, he reported to have bled considerably but there was no history of syncope.

The patient had been working in another factory for seven years before joining the present one, four months prior to the injury. He admitted that the machines he used to work with in the previous factory were more modern with automatic safety gadgets, as compared to the latter where their machines are old with no safety mechanisms.

The patient learnt his job through working experience as he had not received any formal education.

On physical examination he was a young man, in pain, he was not pale and was afebrile. His pulse was 96 beats per minute regular and full volume, his blood pressure was 110/70 mmHg. Heart sounds were normal and he had no murmurs. Local examination of the hand revealed that the second third and fourth fingers of the right hand were completely crushed, the skin and muscles were completely denuded and all the distal and

middle phalanges were sticking out. The rest of the hand appeared normal with exception of slight edema.

The patient was admitted immediately and prepared for surgical toilet in main theatre.

In the main theatre he was given general anaesthesia and the hand was inspected thoroughly under aseptic condition. The hanging skin from the distal third of the second third and fourth fingers, was found to be non-viable, blood vessels were completely crushed, the bones of the distal phalanx of the ring finger and the distal third of the middle phalanges of the second and third fingers were also crushed. Debridement of the dead tissues and excision of the exposed crushed bones was done. The remaining wound was thoroughly cleaned with normal saline alternating with hydrogen peroxide. Then the stumps were sutures with proline number 3/0 covering the bones completely. The wounds were covered with sterile gauze, and a ball of sterile gauze was made so that the hand was dressed in a ball holding position.

Post-operatively the hand was held in an elevated position to reduce the edema. The wound was inspected two days later and found to be healing well, and after one week the patient was discharged to rest at home and was instructed to do flexion and extension exercises several times a day to prevent contractures. He was seen again at our outpatient clinic fortnightly until he recovered well, at least he could still write, eat and

pick something with the same hand, he was also accepted back to his job after two months.

For the other patients, Table I below shows their age and sex distribution.

Table I Age and Sex of the Patients

AGE (YEARS)	SEX F	M	TOTAL NUMBER OF PATIENTS	PERCENTAGE
0 - 10	0	0	0	0
11 - 20	1	3	4	10.8
21 - 30	4	14	18	48.6
31 - 40	0	14	14	37.6
41 - 50	0	1	1	2.7
Total	5	32	37	100.0

Majority of the patients were between 21 - 40 years and there were more males than females.

DISCUSSION

AETIOLOGY

In this study the commonest aetiological factors causing hand injuries were machine crash, this involved industrial workers and accounted for 46% of the hand injury patients as shown in Table II below. The next frequent aetiological factor was motor traffic accident which accounted for 27%, followed by assault 16.2% the remainder 11% were minor domestic injuries such as cuts

with knives, brick wall falling on the patient and crash with a hammer.

Table II: Nature of Injury sustained

NATURE OF INJURY	SEX F	M	TOTAL NUMBER OF PATIENTS	PERCENTAGE
Machine crush	0	17	17	46
Motor traffic accident	2	8	10	27
Assaulted	2	4	6	16.2
Domestic accident	1	3	.4	11

Most of the patients sustained hand injury following machine crush.

In other studies aetiological factors causing hand injuries include industrial machines, butchers, carpentry.[3] Others include farm machinery accidents that are usually complicated by deep tissue destruction, home washing machines were friction burns occur most severely at points of increasing size of the extremity, the metacarpophalangeal joint level, the thenar area and base of the hand.[1] Also meat mincer injuries is common especially in children,[2] few people in our community have meat mincer hence the difference. Other crush syndromes of the hand occurs in drug addicts during the camatose state the weight of the patients body upon an extremity will compromise the vascularity resulting in

erythema induration, ulceration, vesiculation and paralysis.[1] Another factor is a short working experience and lack of formal education this was noted in this study as shown in Table III, about 70% of the patients had working experience of less than one year.

Table III Working experience and duration of employment in that particular factory

Working experience in months	Number of Patients	Percentage
0 - 6	7	41.1
7 - 12	5	29.4
13 - 18	3	17.6
19 - 24	1	5.8
25 - 30	0	0
31 - 36	1	5.8
37 - 42	0	0
>48	2	11.7

For those patients who sustained machine crush injury, more than 70% had a working experience of less than one year.

MANAGEMENT

1. General Considerations:

The proper management of the patient with amputations of the fingers, and hand is dependent upon a knowledge of the normal structure and function of the

hand and its response to the abnormal. The basic function of the hand for prehension reflects its anatomical development and is dependent upon the integrity of the bone and joint apparatus and the controlling neuromuscular system.[5] "A slight injury to a finger or to the hand may rob a young man of his livelihood and alter the whole course of his life. Is there any condition which is more deserving of our care, and of all the skill at one command." (commented by Lorenz Böhler as quoted by Rintoul.[6]

The hand which has been injured may loose the capacity for infinite pattern of action but may still retain some basic prehensile functions. These gross functions should be the goal in the care of the patient with multiple digit loss.[5]



Figure 8.I A machine crushed hand

The well-established tenets of surgery of the hand in maintaining a system with good architectural structure, mobile joints, balance between extrinsic and intrinsic musculature and with a durable, flexible skin cover which has good sensibility, are paramount in the care of the hand subjected to amputation surgery.[5] The patient suffering from a partial hand amputation has usually lost only structural parts or their arrangement. The control apparatus is usually intact, as differing from the spastic or paralysed hand. The usefulness of

the reconstructed partial hand and the substitutionary functional adaptations and prehensile skills to be acquired will, in great part, depend on the mobility of the system.

The amazing prehensile skills seen in the congenitally malformed, partial hand are a demonstration of substitutionary patterns that can be attained in the pain-free, mobile hand.[5]

2. Primary treatment of the wounded hand:

When the hand is acutely injured, the purpose of treatment is to restore its function; thus measures are necessary: to prevent infection, to promote primary healing, and to salvage injured parts. Re-aligning fractures and suturing nerves and tendons may be considered and sometimes may even be done during this primary treatment, but these repairs, except for fracture reduction are secondary in importance to cleansing the wound of foreign matter and devitalized tissue and to providing complete skin coverage even if this requires skin grafting. The surgeon must therefore by history and examination, personally appraise the injury to decide what procedures can be safely done primarily and what secondary procedures may be necessary.[7]

In this study a thorough surgical toilet was done to all wounds and tendons, nerves were also sutured primarily, and fractures fixed with Kirschner wires.

The surgeon who first sees the badly traumatized hand has an opportunity and a responsibility to preserve as much viable tissue as possible. The main objectives in surgery of the disabled hand are to salvage all remaining digits and digital remnants and save as much length as possible to preserve palm, to provide a functional thumb and an opposing pole for pinch and grasp.[5] In this study all patients with hand injuries, the clinical examination and surgical toilet was done under general anaesthesia for proper and thorough evaluation, with the exception of those who have very obvious superficial minor cuts.

According to table IV below 21 patients had partial or total loss of digits. The injury ranged from loss of one digit to the whole hand.

Table IV: Number of digits lost and extent of Hand injury

Number of digits lost partial or total	Number of Patients	Percentage
0	16	43.2
1	8	21.6
2	3	8.1
3	5	13.5
4	2	5.4
5	1	2.7
>5	2	5.4

Most patients had partial injury two patients lost the whole hand.

Roentgenograms of a severely injured hand are made routinely to reveal fractures or metallic foreign bodies. The patient is also examined for other injuries and his general condition is evaluated. Antibiotics sedation, blood transfusions, tetanus antitoxin, and other measures are started as indicated.[7] The patient should be advised as to the extent of his injuries and especially with regard to any possible amputation.[7]

In loss of digital substance, it is necessary to provide a cleft for two opposing units. Osteotomy of the skeletal units for better opposition and parallel adduction may be indicated. A trained hand surgeon can

in certain instances, by understanding the principles of reconstruction facilitate the end result during the primary stage. Secondary reconstructive procedures will then be done in shorter order. The importance of having adequate tissues to work within the reconstructive stage of treatment cannot be overstressed. Careful alignment of fractures with consideration of the arches of the hand should be the rule. Firm internal fixation with kirschner wires. The dressings should not be depended upon to hold fractures. At our centre there is no hand surgeon yet, so most of the hand injuries which involves tendons, nerves and fractures are taken care by orthopaedic and trauma surgeon. We also lack the proper instruments e.g. microplates and small drills for the small bones of the hand.

Proper operative and post-operative care to avoid the secondary problems of drainage and swelling and the institution of early mobilization may make the difference between a stiff and secondary deformed hand and a useful, prehensile mechanism capable of substitutionary prehensile functions.[5]

3. CONSIDERATION FOR SKIN CLOSURE

Primary skin closure is desirable and can usually be done in all incised tidy wounds. The purpose of primary skin closure is to obtain early healing and to avoid infection, granulation tissue, and excessive scar production. Misjudgement leads to delayed healing

resulting from hematoma, excessive swelling, and possible infection, any of which may require re-opening the wound for drainage and an even greater delay in healing. Certain wounds should never be closed primarily; these include the severely contaminated or crushed wounds caused by farm machinery, human bites, tornado missiles,war wounds, such as high-velocity missile wounds and wounds contaminated with human excreta or fertilizer, also late wounds more than 8 hours should not be closed primarily.[7]
When in doubt, the wound should be left open after careful debridement under anaesthesia.

Within 24 to 48 hours the wound should be reinspected, and if it is sufficiently clean, it can be closed by direct suture or by skin graft. A wound should not be left open to granulate and heal by scar unless it cannot be rendered clean, but should be closed if possible within five days after injury.[7]

4. TENDON INJURIES

The posture of the hand often provides clues as to which tendons are severed.

Flexor tendons: When both flexor tendons of a finger are severed the finger lies in an unnatural position of hyperextension.

Extensor tendons: Is presumed to be divided between the proximal and distal interphalangeal joints when active extension of the latter joint is lost; initially a gross

mallet finger deformity may be absent because the surrounding capsule and other soft tissues have not yet been stretched by the strong flexor digitorum profundus.

TENDON REPAIR

The purpose of tendon suture is to approximate the ends of a tendon or to fasten one end of a tendon to adjoining tendons or to bone and to hold this position during healing. When the tendons are being sutured, management should be delicate causing as little reaction and scarring as possible.

TECHNIQUE OF TENDON SUTURES

i) **End to end suture** The most classic technique of end to end suture is the Bunnell crisscross stitch. It is not commonly used now because the rippling created by tension tends to make the ends of the tendons avascular.

ii) **End to side anastomosis:** This is frequently used in tendon transfers when ONE motor must activate several tendons.

iii) **Double right-angled sutures (modified Kessler)** To suture the several ends of a tendon together without shortening, a double right-angled stitch can be used. This method is easier and is more frequently used when multiple tendons are severed.

iv) **Attachment of tendon to bone:** To attach a tendon to bone, use a small osteotome or dental chissel to roughen the site of insertion or raise a small area of cortex to accept the tendon. If several tendon ends are to be

fixed to bone, they are best inserted into a large hole drilled in the bone.

INDICATIONS FOR PRIMARY TENDON SUTURE

When a wound is caused by a sharp object such as a knife and is reasonably clean, some tendons of the hand should be repaired at the time of primary wound closure. Some factors determining which tendon may be so repaired are as follows:-

1. the time since injury
2. the area of the hand affected
3. the number of tendons divided
4. the severity of wound contamination
5. the presence or absence of unstable fractures.
6. the general condition of the surrounding tissues.

In the following instances divided tendons are usually not sutured primarily but should be left secondary repair.

SECONDARY REPAIR OF TENDONS

Before tendons are secondarily repaired, certain requirements must be met:

1. Wound erythema and swelling suggest inflammation or potential infection and should not exist.
2. Skin coverage must be adequate
3. The tissues in which the tendon is expected to glide must be relatively free of scar;
4. the alignment of bones must be satisfactory and any fractures must be healed or fixed securely;

5. Joints must have a useful range of passive motion; and
6. Sensation in the involved digit must be undamaged or restored, or it must be possible to suture damaged nerves at the time of tendon repair.

When all these requirements are met, the tendon may be repaired as late, as two weeks after injury; however, whether it should be repaired then mainly depends on the condition of the wound itself or the associated tissues.[7]

FRACTURES

The principles of fracture treatment applying elsewhere in the body also apply to the hand, except that, since the tolerance of this part is low, fractures here must be more accurately reduced to restore function - closed treatment by manipulation and splinting in the position of function is indicated for almost all stable fractures of the metacarpals and phalanges; this cannot be emphasized too much.

Internal fixation is indicated in the following instances:

1. When a displaced fracture with a fragment too small to manipulate involves a joint; exact reduction is necessary to restore smooth joint motion.
2. When a fracture is displaced so severely that interposition of soft tissue prevents realignment by manipulation.



A



B

Figure 8.II A and B. This patient's hand was crushed by a brick. Figure 8.IIA shows X-ray of the hand before mobilization and Figure 8.IIB after internal fixation.

NERVE INJURIES

Injuries of nerves are even more difficult to evaluate than those of tendons and are often overlooked during the preliminary examination. Digital nerve injuries are frequently overlooked because these nerves are so small or because they are not thought of even when the wound is inspected during the operation. When a flexor tendon in the finger is cut, at least one digital nerve is usually cut also.[7]

In 1984 the clinical Assessment Committee of the American Society for surgery of the Hand issued a report on nerve function evaluation. The committee recommended four areas to evaluate in the progress of peripheral nerve injuries and repair; sensibility testing, motor testing, subjective evaluation and pseudomotor function.

SENSIBILITY EVALUATION

The basic minimum, tests recommended are the stationary two-point discrimination and moving two-point discrimination test.

Technique: The hand should be warm and the instrument at room temperature. Apply a blunt caliper distally over the digital pad until blanching occurs. Test each area three time, going progressively from longer distances to shorter distances.

The moving two-point discrimination test is done in a similar fashion. The caliper is applied in an axial discretion and moved from proximal to distal aspects

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along the digital pad. Two out of three correct answer are considerable proof of perception with either test.

DISLOCATIONS

Most dislocations of the joints of the hand can be treated by closed means.

Exceptions are:

1. Unstable dislocations of the carpometacarpol joints of the thumb or finger.
2. Dislocation of the metacarpo-phalangeal joint of the thumb with a complete rupture of the ulnar collateral ligament, which renders pinch weak and unstable.
3. Dislocation in which a tendon is trapped.
4. Old undiagnosed dislocations.

CONCEPTS OF AMPUTATION OF INDIVIDUAL DIGITS

The Thumb: The thumb is considered as 40% of the hand because of its polar position and its oppositional ability. Its effectiveness is in direct proportion to its length, mobility and placement. The thumb web space provides freedom of action and also a cleft for the grasping space.[5]

The fingernail is essential for fingernail pinch. The short thumb can be a very functional digit as long as the other digits can reach it and the cleft is deep enough. Retention of the base of the phalanx may be of importance in function as long as there is a tactile pad

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covering. Amputation to the proximal portion of the proximal phalanx is disabling and marked improvement may be obtained by deepening the cleft. Amputation at the metacarpophalangeal joint is considered as 100 percent loss of the thumb. The neurovascular skin pedicle may be used for restoring critical sensibility to the thumb with a scarred asensory tip.[5]

The index finger

The index finger is considered to represent 20 percent of the hand and is most important in providing an opposite pole for the functions of grasp and pinch. it provides spread for grasp, opposition to the thumb for palmar pinch, fingertip pinch and lateral pinch. It is important in percussion adaptations of the hand. Maintenance of length is of great importance in this finger. It is the only digit other than the thumb that needs have its length maintained by cross finger or thenar skin pedicle coverage. When amputation occurs proximal to the distal interphalangeal joint, pinch is usually transferred to the middle finger. When amputation occurs at or proximal to the interphalangeal joint, the stump acts mainly in widening the palm. Some lateral pinch can still occur and may be of functional importance. However in the presence of other fingers, it may be better to resect the ray at its base subperiosteally and to transfer the first dorsal interosseous muscle to the base of the proximal phalanx

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of the middle digit. This procedure will open up the web space and improve the functional relationship between the thumb and middle finger.[5]

The Middle Finger

The middle finger is considered as 20 percent of the hand because of its contribution to strong grasp, pinch, hook and percussion action. It reinforces the index fingers action and may substitute for it completely. Loss of the middle finger also results in loss of cup action or scoop action of the hand. Small objects fall through the closed hand. Amputation through the metacarpal results in loss of the stabilizing effect of the transverse metacarpal ligament. Adjacent fingers roll away from the missing finger. The metacarpal also provide an insertion for the strong adductor pollicis muscle.[5]

The Ring Finger

The ring finger fills in the finger span and is considered to represent 10 percent of the hand. Along with the little finger, it provides the palmar arch with mobility. Metacarpal loss results in rotary deviation of adjacent fingers.[5]

The Little Finger

The little finger is considered as 10 per cent of the hand. It adds to the width and strength of the grasp. Its presence is especially important in loss of the other fingers. It is frequently taken as a spare in

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multiple loss. Amputation through the metacarphus is best carried out obliquely at its base to avoid a cosmetically and functionally deterring hump on the ulnar aspect of the hand.[5]

The table below shows the digits injured most frequently as depicted in this study.

Table 4: DIGITS INJURED FREQUENTLY

DIGIT	FREQUENCY OF INJURY	%
Thumb	7	18.7
Index finger	10	27.0
Middle finger	9	24.3
Ring finger	7	18.9
Little finger	2	5.4
Whole hand	2	5.4

The index finger had the highest frequency followed by the middle finger.

MULTIPLE DIGIT AMPUTATION

When there has been multiple major digit loss, the relative length of each digit assumes greater importance. It is of prime importance that as much length as possible be maintained.

Skin grafts and atypical flap closures to cover finger tips assume greater importance, and resection of much bone to obtain smooth stump closure should be

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MANAGEMENT OF GIANT CELL TUMOR AT MUHIMBILI MEDICAL CENTRE

SUMMARY

A total of 8 patients with Giant-cell tumor (osteoclastoma) were seen in the orthopaedic and trauma, during the period of 1st August, 1990 to 28th February 1991 (total period of 7 months). There were 6 females and two males. The age ranged from 21 to 40 years.

Out of the 8 patients who were confirmed histologically, five were operated,two refused surgery and one patient was offered palliative treatment. The surgery offered was either curettage, amputation or bone resection with bone grafting. None of the patients received radiotherapy.

The distal third of the femur showed to have a high predilection.

Diagnosis was confirmed radiologically and histologically.

The only complication encountered was severe sepsis in one of the patients whose curettage was not adequate. He had refused amputation. The other patients faired well and were able to return to work within a period of six months.

INTRODUCTION

Giant cell tumor is usually seen in patients over 20 years of age.[1] It is a relatively uncommon neoplasm

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INTRODUCTION

Giant cell tumor is usually seen in patients over 20 years of age.[1] It is a relatively uncommon neoplasm

and the great majority occur between the ages of 20-40 years, with a slight female preponderance.[1,2] The classical location is the epiphysis of a long bone, from which it may spread into the metaphyseal area, break through the cortex invade intermuscular septa, or even cross a joint space.[1,2]

The sites most commonly affected in order to frequency are the:

i) Distal end of the femur) 55%
ii) Proximal end of the tibia)
iii) Distal end of the radius - 10%
iv) Sacram - 8%
v) Pelvis and head of femur - 7%
vi) Distal end of tibia - 7%

Exceptions do occur e.g. giant cell tumors have also been seen in metaphysis of the radious, patell, fibula, humerus and sphenoid bone. Involvement of the bones of the hands and feet, jaw and vertabrae is distinctly unusual.[1]

Three aspects of these neoplasm are of particular interest:

i) Their cell of origin
ii) Their differentiation from other giant cell bearing lesions.
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difficult. Multinucleate giant cells are feature of many non-neoplastic as well as neoplastic lesions of bone. They are particularly prominent in so-called "brown tumors" seen in Osteitis fibrosa cystica related to hyperparathyroidism, and in the giant-cell granuloma (also called epulis) of the gingiva and jaw bones. Multinucleate giant cells are also encountered in many benign and malignant bone tumours. Chondroblastoms in particular pose a challenging diagnostic problem because they have many multinucleated giant cells and moreover they often arise in the epiphyseal ends of bones, where giant cell tumors arise.[2] Since chondroblastoma is rarely malignant compared to giant cell tumors which prove to be malignant in 3-10% of cases, the importance of the differential is evident.[2]

CASE REPORT

A 35 year old patient (IP NO. 623055) was referred from Iringa with one and a half years history of painful swelling in the left wrist joint on lifting a heavy object. The pain was pricking in nature and localised to site of swelling which after 6 months became painless. The swelling was gradually increasing in size, and by the time she presented to us she was unable to utilise her left hand fully.

There was no history of trauma or other affections. Initial treatment at nearby hospital was immobilization in a POP cast for two weeks without relief. She

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There was no history of trauma or other affections. Initial treatment at nearby hospital was immobilization in a POP cast for two weeks without relief. She

resorted to the traditional healer, who tattooed the swelling with some herbs as in the hope of controlling the swelling but with no relief of symptoms. She then attended Iringa Regional hospital where she narrated history specific to the lesion except for a small simple goitre which was apparent since her menarche.

Examination revealed a healthy looking. afebrile young woman who was not pale, and without lymphadenopathy. The left forearm revealed a globular swelling the wrist joint with therapeutic marks over it. Measuring about 6 cm in greatestdiameter the swelling was irregular in nature, firm and not pulsatile. Some areas felt like a cracking egg shell.

X-ray of the wrist joint revealed an expanded rarefied lesion destroying the whole of the distal one-third of the left radius, there was also thinning of the overlying cortex which was partly intact, and partly destroyed. The carpal bones were spared. An impression of Giant-cell tumor was entertained. Chest x-ray was normal.

- Haemogram was reported normal with HB. 12.2 g/dl.

She was then scheduled for radical excisional biopsy of the tumor and grafting by the tibia for bridging the anticipated defect. There were two teams of surgeons where one team was resecting a bone graft from the middle portion of the left tibia, another team was excising the tumor. An S incision was made over the

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tumor on the overlying skin on the dorsum of the left wrist joint, extending to the distal third of the forearm. The skin was dissected out and the tendons overlying the tumor retracted. a well capsulated tumor of the distal third radius destroying the whole bone, infiltrating the extensor tendons was found.

The distal third of the radius was resected with a margin of 2.5cm proximal to the tumor, sparing the extensor tendons, a portion of the capsule of the proximal carpal bones was excised. The defect left by the resected bone was grafted by the piece of the cortical tibial bone which measured about 8 cm, and it was held in position 6 hole plate. The wound was closed after irrigating with normal saline. The operation took about 1 hour.

The patient was given sedation - Pethidine 100 mg intramuscularly 8 hourly for 24 hours. Post operatively no prophylacti antibiotic was given. She recovered uneventful and was discharged after 10 days. She was instructed to attend physiotherapy as she was unable to flex or extend the fingers.

The resected bone was sent for histological analysis and the results reported: " A tumour characterized by tissue with necrotic foci and haemorrhages consisting of spindle-shaped, avoid cells with easinophilic granules and presence of numerous giant cells of osteoclastic type, this morphologic

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The patient was seen in the outpatient clinic after one month with improvement, she had than a reasonably strong grip, but could not flex or extend the hand as the wrist joint was arthrodosed.



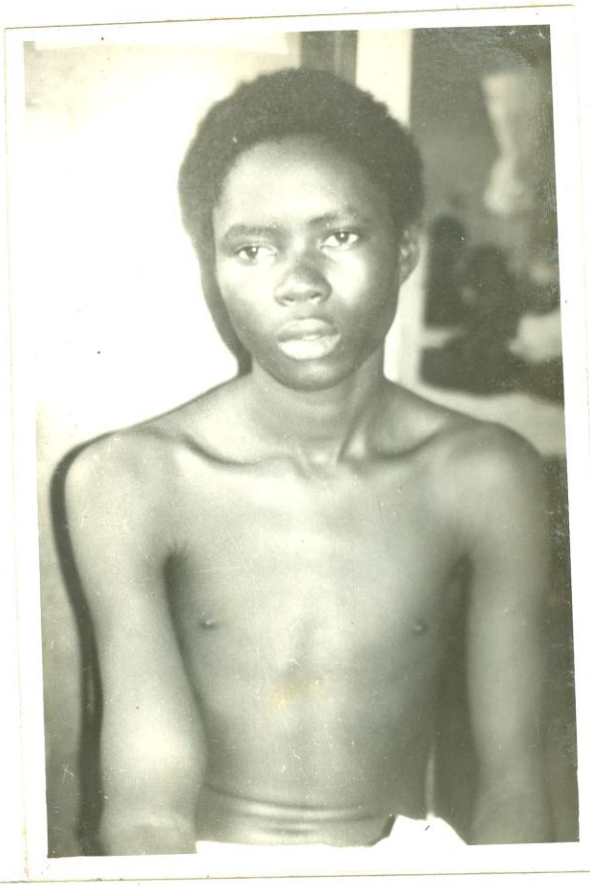
Figure 9.I X-ray of patient with Giant Cell tumor. Distal end of the humerus.

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Figure 9.I X-ray of patient with Giant Cell tumor. Distal end of the humerus.

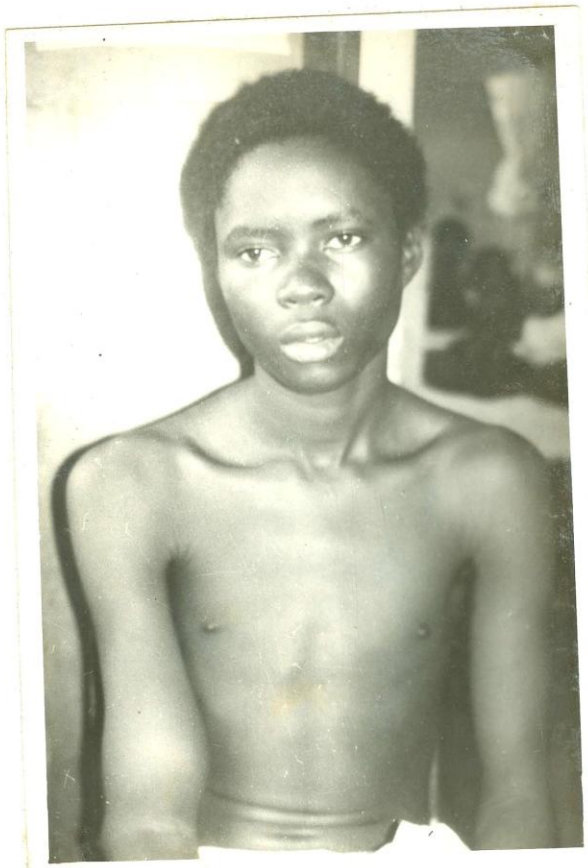


A



B

Figure 9.IIA. Photograph of a patient with Giant cell tumour right arm. Figure 9.IIB. The same patient after surgery.



A



B

Figure 9.IIA. Photograph of a patient with Giant cell tumour right arm. Figure 9.IIB. The same patient after surgery.

For the other patients the age range was between 21 to 40 years. With a preponderance of five patients in the age range of 21-30 years.

The commonest site of lesion was the distal third femur which accounted for 50% followed by distal third humerus which accounted for 25% and then distal third of radius and tibia 12.5% each.

DISCUSSION

Aetiology:

The aetiological factors of this tumor just like many other tumors is unknown.

PATHOLOGY

i) **Morphology:** Giant cell tumours nearly always arise within the epiphysis, but may then spread into the metaphysis or even through the articular cartilage into the joint space. Over half of these lesions occur above the knee in the distal femur, proximal tibia or proximal fibula but virtually any other bone may be involved, including sacrum, pelvis, long bones of the forearm, and the small bones of the hands and feet.[2]

Characteristically, they are multilobular and red-brown on cut surface, with prominent areas of haemorrhage, cyst formation, and foci of pale, yellow-white necrosis.[2] Most tumors are large and have undergone fairly rapid increase in size with clublike expansion.

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The cortex may be thinned or even eroded. Rarely the tumors penetrate into the surrounding soft tissue, but despite this invasive behaviour they tend to grow as cohesive masses along broad lobulated fronts.[2]

ii) **Histology**

Giant cell tumors are composed mainly of nondistinctive conocunclear cells having round oval or spindle-shaped nuclei remarkably uniform in size. Mitotic figures are often numerous even in lesions that later prove to be entirely curable by local resection. Scattered within this background are numerous giant cells having as many as 100 benign-appearing nuclei resembling those in the mononuclear cells. There may be foci of necrosis, hemorrhage, chondroid foci are unusual and when prominent strongly suggest the diagnosis of chondro-blastoma. In the unusual giant cell tumors that are malignant from the outset, the stromal mononuclear cells are anaplastic.[2]

The stromal cells are mononuclear and in general resemble young connective tissue cells.[3] They are spindle shaped or ovoid in varying proportions. Correspondingly, the nucleus, which occupies much of the cell body is longish or roundish. It contains a moderate amount of chromatin and a more or less central nucleolus. The outlines of the individual stromal cells cannot always be traced completely, but it can almost regularly be seen that the cells have cytoplasmic

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processes. The collagenous intercellular material in the giant-cell tumor is most abundant where the stromal cells are very spindle and loosely arranged. Where they are ovoid and closely packed, the collagenous material is sparse.[3] The specimen in this study had similar features.

GRADING OF GIANT-CELL TUMOURS

If one adheres to a strict definition of what should be regarded as giant-cell tumor and strips away all the alleged variants, what is left constitutes a formidable neoplasm.[3] Most of them in recent years have been aggressive or potentially aggressive tumours. An appreciable number have manifested local recurrence after surgical extirpation, either in the surgical wound or in the operative field after resection.[3] There appears to be substantial agreement, among pathologist at least, are successfully treated, by thorough currettement or irradiation, many are undoubtedly aggressive and prove to recur, and a few behave like frank sarcomas, either when initially observed or, more often after one or more local recurrences.[3]

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The treatment and outcome of the patients is as shown in the table below.

MODE OF TREATMENT AND OUTCOME WITHIN SIX MONTHS

Mode of Treatment	Number of patients	Developed recurrence	Discharged in good condition
Curettage	2	1	1
Amputation	1	0	1
Bone resection	2	0	2
Refused surgery	2	0	0
Analgesics Palliative	1	0	0
Total	8	1	4

For those operated only one patient who was curetted developed recurrence within a period of 6 months.

As a guide to therapy and with a view to forecasting prognosis within certain limits, it was suggested as early as 1940 that giant-cell tumors be subdivided into three grades, designated numerically as I, II and III and showing respectively, insignificant, moderate, and pronounced atypism of their stromal cell.[3]

GRADE I: This seems to make up about half of any representative series of giant-cell tumors of bone, and the cytologic features indicate the lowest degree of aggressiveness and hence, a relatively favourable prognosis.[3] The tumor delegated to the other two group

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show successively more ominous cytologic features, those of Grade III being definitely malignant.[3] In connection with grading, the details of th stromal cells should be closely scrutinized under high magnification; and one should judge the tumor by its most ominous-looking areas rather than by areas selected at random.[3] It follows from this, that for a relatively safe judgement more material is usually needed than is obtained in a needle or a punch biopsy specimen.[3] Previous irradiation of the growth, too is likely to be a factor in the grading, since it may have induced fibrosis, hyalinization, and calcification as well as distortion and atypism of the stromal cells.[3]

In Grade I lesion it must not show appreciable atypism of its stromal cells. Though these cells are abundant, they are not densely compacted in a tumor of this grade. Also one finds an occasional stromal cell that has a hyperchromatic or abnormally large nucleus or perhaps two nuclei. In regard to prognosis, recurrence can be expected in some instances though not in the majority.

In this study out of the five patients operated in this study only one had recurrence.

Furthermore, if it recurred more than once, a Grade I tumor may finally come to exhibit characteristics that actually put it in the next grade.[3]

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Grade II

Constitute a somewhat varied group ranging from those in which the stromal cells show only slight atypism to those in which they are striking atypical though not yet sufficiently so to justify placing them in the category of the frankly malignant tumours. The stromal cells are abundant and closely compacted and may even show plainly an irregular whorled arrangement. In some cases they tend on the whole to be spindle shaped, though in some areas they may be plumper or ovoid, while their nuclei are large and swollen in proportion to the cell as a whole, often exhibiting atypism. Specifically, in the latter connection the nuclei may vary considerably in size, many of them may be hyperchromatic, and some of them may be multiple, two or three being present in a given cell. The giant cells are likely to be abundant in some or in many areas, though they may be sparse in others. Their nuclei may reveal atypism similar in a general way to that of the stromal cell nuclei and running more or less parallel to it.

The compacted stroma may be found poorer in vascular channels than it is in the tumors of Grade I.[3]

As to prognosis, it is to be noted that the tumor of Grade II tend strongly toward recurrence, and that some of them eventually undergo malignant transformation, although usually only after an interval

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of several years.[3] In this study none of the 8 patients was in this grade.

Grade III

This constitute the small group of giant cell tumors of bone that are frankly and obviously malignant, possessing a sarcomatous type of stroma and a capacity for metastases. Their stromal cells are abundant and closely compacted and tend to present an irregular whorled arrangement. Their nuclei exhibit atypism more often, however a malignant giant cell tumor is that originally belonged to Grade I or II but that has become more and more aggressive in the course of years. One of the patient in this study presented with grade III tumor, which had completely destroyed the distal half of the femur, the chest x-ray revealed pulmonary metastases. This patient was offered supportive treatment was given analgesics for the bone pain.

The other characteristic of this grade they also show increased tendency of the growth is normally inherent in it from the beginning. The tumors of Grade III metastasize sooner or later, particularly to the lungs. In their metastases, at least they do not show up as osteogenic sarcoma, if there has been no prior irradiation. In some instances, one can recognize the metastases as those of giant-cell tumor, although the giant-cells may be scant and diverted; in others. They may look like undifferentiated fibrosarcoma. In a

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sense, a malignant giant-cell tumor (that has not been previously irradiated) may be regarded as a special kind of fibrosarcoma.

Clinical Presentation

As it has been noted in other studies some giant cell tumor develops insidiously and usually has attained appreciable size before presenting to hospital, only one patient her tumor was noted radiologically. The rest had obvious deformity of the site involved. Even the patient whose tumor was first noted radiologically the whole proximal end of the tibia was completely destroyed.

Just as it is reported in other studies, clinically these patients present because of local pain, tenderness and loss of function around the joint, which may even suggest a possible inflammatory lesion or a swelling of an asymmetric nature.[2,5] Sometimes they may present with pathologic fractures. However in many instances the tumor grow insidiously to massive size to produce externally palpable masses before discovery.[2,3]

Pronounced expansion and a sense of crackling of the cortical shell surrounding the lesion are manifestations of a far advanced neglected tumor.[3] Six of our patients presented with lesions which on palpation felt like a broken egg shell. In these 6 patients 5 patients the lesion were resectable and only

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one patient his tumor was too advanced with metastases and one refused surgery.

In case of some few giant-cell tumors, one may elicit a sense of pulsation reflecting great vascularity, but this finding too is rather unusual.[2]

INVESTIGATIONS

I **X-rays:** Radiologically the tumor is osteolytic and expands the bone with thinning, often perforation, of the cortex to cause a pathological fracture in some cases. In this study only one patient presented with pathological fracture. The bone is destroyed irregularly so that the tumor is traversed by remnants of the original bone and comes to lie in a cavity with heavily trabeculated walls. As a consequences, it has a typical soap bubble radiological appearance.[2] It rarely invades soft tissue but may do so when fracture occurs.[2]

II **Biopsy:** Diagnosis should be confirmed by biopsy.[4]

TREATMENT

As noted previously these tumors have been regarded as relatively benign, but recently it has been found that up to 30% behave with malignant characteristics despite a benign histologic appearance.

In view of these varying degrees of malignancy, excision, resection or thorough curettement is indicated, With curettage there is up to 50% local recurrence rate.[5] Treatment is therefore directed to a complete and total local resection at all possible.

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Amputation is occasionally indicated in primary tumor whose biopsy discloses evidence suggestive of malignancy e.g. large stromal cells with mitotic activity amputation is undoubtedly the safest treatment and should not be delayed.[4] For tumors in the most common situation immediately above or below the knee this should be carried out at mid-thigh level.[4]

A complete excision entails surrounding shell of healthy bone, and the loss can be replaced by bone-grafting.[4] In this study all those who had bone resection about 2.5 cm margin of the normal bone was taken with the tumor, and where bone resection was not possible, amputation was done.

RADIATION

With the development of super-voltage apparatus, radiotherapy may have some place in preventing recurrence.[4] There seems to be good reason to hold empirically that x-ray irradiation per se is capable of effectively controlling a certain number of giant-cell tumors particularly the less aggressive ones.[3] At present, the tide appears except as a modality of last resort, because of its complications (sloughing of the wound infection, and pathologic fracture) and the real hazard of post irradiation sarcoma which is reported to occur in as many as 20% of the patients.[3,6] None of our patients was irradiated.

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Cryosurgery

Marcove et al reported by Lichtenstein, have advocated cryosurgery as a promising approach, but this recommendation will require critical clinical confirmation.

Chemotherapy

No chemotherapy offered due to shortage of the cytotoxic drugs.

Surgery: Treatment offered in this study was mainly surgery either bone resection, amputation or curettage. Out of the 5 operated patients one who was curretted had recurrence within a period of 3 months. The rest were free of the disease during the 6 month period of follow up.

CONCLUSION

Giant cell tumor though uncommon condition at our environment, its importance lies in the virtual fact that it mainly affects young people. As shown in this study the age range was 21 to 40 years. When it comes to an individual this is the most productive age. And also sometimes it entails loosing a limb in a such benign lesion.

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SUMMARY

Twenty-eight children with hydrocephalus were managed at the paediatric surgical unit of Muhimbili Medical Centre between 1.8.1991 to 31.6.1991.

Eighteen patients were new cases and ten were readmission with complications or routine follow up cases.

Their ages ranged from one month to 8 years. Fourteen patients were below the age of 6 months.

Diagnosis was clinical. Serial daily measurements of the occipital frontal circumference to determine whether the hydrocephalus was progressive or not were taken. The presence or absence of neurological deficits were recorded.

Out of the 28 patients 16 were operated upon while 12 were not. Operations was not performed in those with advanced hydrocephalus, severe malnutrition or severe neurological deficits.

The commonest aetiology of hydrocephalus was meningitis followed by congenital causes.

Most of the children had quite advance hydrocephalus, either because of the ignorance of the parents in knowing that the condition was treatable. Also it took time for parents to note that the child's head is growing abnormally until it was quite advanced.

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Although hydrocephalus is not a very common problem but it is quite significant in terms of admissions in our paediatric surgical unit. Hence the need to include Occipital Frontal Circumference as an important parameter in Maternal and Child Health Care (MCH) services so that this problem can be detected early.

INTRODUCTION

Hippocrates (460-377 B.C.) recognized that water accumulating in the head caused it to swell and may have performed ventricular puncture for its treatment: Herophilus and Erasistratus knew of the existence of the ventricular cavities and recognized that they contained fluid.[1] Oribasuis mentioned cases in which fluid accumulated over the surface of the brain, but did not mention enlargement of the ventricles.

The first clear description of internal hydrocephalus was that of Vesalius, who described the brain of a child with internal hydrocephalus whose ventricles were so distended that they contained about 9 pounds of water.[1] In this case there was concomitant marked enlargement of the head. Two centuries later, Morgagni noted that this condition could occur in adults without enlargement of the head, a fact not generally appreciated at that time. Magendie showed by animal experimentation that the fluid in the ventricles and subarachnoid space was in free communication via the midline fourth ventricular foramen which now bears

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his name. This finding was confirmed by Luschka who in addition, described the paired lateral exists of the 4th ventricle.[1]

Hydrocephalus is commonly seen at Muhimbili Medical Centre. This is partially because it is the only centre offering treatment for this pathology. This study looks into age and sex distributions and management of hydrocephalus at Muhimbili Medical Centre. A typical case is presented in full while literature review on embryology, pathophysiology, etiology, treatment, and treatment complication is presented.

CASE REPORT

S.D. Inpatient number 629515 a 4 month old male baby, presented to our outpatient clinic with history of progressive enlargement of the head since birth. Antenatal history was uneventful and the child was delivered normally. He had no history of convulsions, no fever, no history of trauma or vomiting.

On examination he was a healthy looking baby and he was afebrile. The cardiovascular and respiratory systems were normal. He had a large head with bulging anterior fontanelle with occipital frontal circumference 55 cm i.e. 13 cm deviation from the 50th percentile. (Welcome Classification). He had head lag, the suture lines were widely open, could turn head towards wounds. He could hear well and he could watch his own hand

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Table I: Age and Sex Distribution of 28 Children with Hydrocephalus

Age (Months)	Sex		
	Male	Female	Total
0 - 6	8	6	14
7 - 12	3	3	6
13 - 18	1	2	3
19 - 24	1	0	1
25+	2	2	4
Total	15	13	28

There were 15 males and 13 females. 50% (14) of the patients were less than 6 months old. Only 4 patients were above 2 years of age.

Table II: The management of 28 patients with Hydrocephalus

Management	Number of patients	Percentage
Operative	16	57
Non operative	12	43
Total	28	100

More than half (57%) of the patients were operated, and those who were not operated due to advance hydrocephalus, severe malnutrition, or severe neurological deficits.

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Figure 10.1: This 9 months old child had a very advanced hydrocephaus with occipital frontal circumference of 91cm.

DISCUSSION

EMBRYOLOGY

The central nervous system develops as an indentation of ectoderm, this infolding becoming deeper and the outer parts gradually migrating towards one another and finally fusing to form the primitive neural tube. Fusion commences in the middle portion and extends cranially and caudally. Failure of completion of this process results in many of the structural abnormalities of the CNS found in the fetus and the neonate.[2]

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Various congenital anomalies may occur which will lead to hydrocephalus. These anomalies include: Aqueductal stenosis, aqueductalatresia, fourth ventricle outlet obstruction and the Arnold-Chiari malformation.[1]

PATHOGENESIS OF HYDROCEPHALUS

The current knowledge concerning the morbid anatomy of hydrocephalus was put on firm ground in 1949 with the publication of observations on the Pathology of hydrocephalus by Dorothy Russell. It was noted that almost every case of hydrocephalus there is a pathological obstruction at some point along the pathways of cerebrospinal fluid circulation.[3] By convention hydrocephalus has been divided into two broad categories: Communicating and non-communicating hydrocephalus. In non-communicating hydrocephalus there is an obstruction to the flow of CSF from inside the ventricular system. In communicating hydrocephalus the obstruction is outside the ventricular system.[3]

Communicating hydrocephalus: Is characterized by panventricular enlargement with obstruction to the flow of CSF in the subarachnoid space, that is distal to the outlets of the fourth ventricle. Hence, there is free communication between the ventricles and the subarachnoid space.[3]

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Non-communicating hydrocephalus: This is obstruction within the ventricular system or at the outlets of the fourth ventricle. However, patients with non-communicating hydrocephalus may present with exactly the same symptoms and signs as those with the communicating variety, in particular obstructive lesions of the 3rd ventricle (By giant Pituitary adenoma or colloid cyst) basilar artery ectasia, aqueduct stenosis, Arnold-chiari malformation, and after multiple shunt revisions. In both types patients may present solely with symptoms of hydrocephalus with none referable to the primary pathology. The two forms may produce symptoms of raised intracranial pressure or have apparently normal cerebrospinal fluid or have apparently normal cerebrospinal pressure. Both may arrest spontaneously.[4]

EPIDEMIOLOGY AND AETIOLOGY

The overall incidence of hydrocephalus in the general population is not known. Since the condition occurs in association with a large number of childhood intracranial diseases, it is obvious that the reported incidence of infantile hydrocephalus, namely 3 to 4 per 1000 live births, is grossly understated. The incidence of hydrocephalus occurring as a single congenital disorder is given as 0.9 to 1.5 per 1000 births, and that of hydrocephalus occurring with spina bifida and myolemeningocele varies from 1.3 to 2.9 per 1000 births.[1]

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Whereas the causes of acquired hydrocephalus are often apparent, the aetiology of hydrocephalus in neonate is not entirely clear. The most commonly associated abnormality a neural tube defect. The Arnold-Chiari malformation and stenosis of the aqueduct of sylivius were considered the causes of hydrocephalus for sometime but these are related malformations rather than aetiological factors. [2]

Primary hydrocephalus may result from congenital infections. e.g. toxoplasmosis, rubella, cytomegalovirus, herpes, maternal malnutrition, toxins, syphilis or from a cerebrovascular accident such as thrombosis, embolism or haemorrhage. The last occurs more commonly intrapartum and post-natally. Rarely a choroid plexus papilloma, prenatal tumors, or an arteriovenous malformation with aneurysm of Galen's vein may be the cause. An inheritable form of aqueductal stenosis has been described that occurs only in males and is transmitted from one generation to the next by a female carrier (X-linked hydrocephalus). This is an extremely rare condition and probably accounts for less than 2 percent of all cases of congenital hydrocephalus.

Aetiological factors of acquired hydrocephalus, include trauma, intracranial bleeding, meningitis, trauma, tumors and cysts.

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such patients. Their distribution is as shown in Table III below:

Table III: The Aetiological Factors of Hydrocephalus in 28 patients at Muhimbili Medical Centre

Aetiology	Number of Patients	Percentage
Post meningitis	12	43
Congenital	10	36
Idiopathic	6	21
Total	28	100

The commonest cause of hydrocephalus in this study was due to meningitis.

DIAGNOSIS

The diagnosis of hydrocephalus is usually based on clinical and simple radiological features, but more sophisticated investigations like pneumoventriculography, ultrasonography and CT scan may be needed to distinguish subdural effusions, macrocephaly and other cephalic abnormalities.[3,5]

Ultrasonography The ultrasonography of the neonatal brain and its value in the diagnosis of hydrocephalus and intracerebral and ventricular haemorrhage is now well established. The low cost, ease of examination and improved diagnostic accuracy have contributed to the clinical acceptance of cranial ultrasound and a

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reduction in the requirement for CT-scanning of the head in the very young infant. The highly reflective nature of the choroid plexus makes it a most striking ultrasonic landmark in the infant's brain. This feature, together with the location of choroid plexus tumours and the associated ventricular dilatation, makes ultrasound the obvious initial investigation of these lesions.[6] Early ultrasound examination of the head is possible so long as the anterior fontanelle is patent. Also ultrasound unlike the CT Scan machine can be taken to the infant rather than the infant to the apparatus. It helps to distinguish more accurately the infants with hydrocephalus at birth from those who have intraventricular haemorrhage with secondary hydrocephalus.[2] The small infant is at greater risk and the increasing numbers of low birth weight group more are surviving and subsequently presenting with hydrocephalus.

CT-scanning: The pattern and extent of ventricular enlargement is clearly shown together with gross widening of the cerebral sulci and fissures. Any space occupying lesions such as tumours are readily apparent. Even ectatic basilar arteries sufficiently large to obstruct the 3rd ventricle are revealed after contrast enhancement.[4]

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In this study non of the patients had CT-scanning because this machine is not yet available at our hospital. Diagnosis was mainly by clinical examination and serial measurements of the occipital frontal circumference to determine whether the hydrocephalus was progressive or not.

TREATMENT

The main objective of operative treatment is to prevent or reduce impairment of cerebral development, particularly during the crucial first year of life, by either removing the cause of the disease (which is rarely possible) or by providing a suitable shunt to the flow of CSF which is often feasible.[5]

Many ingenius operations attempting to control progressive hydrocephalus were performed earlier in the century but the modern era commenced in the UK with the introduction of the Holter ventriculocaval shunt valve by Macnab in 1958. Since then there has been proliferation of the drainage systems and techniques: however, problems remain in maintaining adequate control of the hydrocephalus and there may also be complications due to superimposed infection.[2] Various shunting procedures have been developed and refined and replaced the older operations of third ventriculostomy and ventriculo-cisternostomy.

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hydrocephalus. The shunt crosses enormous anatomic barriers, the plastic does not grow, and the valve system treats a volume transfer problem by pressure regulation. These situation indicate the need for continued care of these patients as well as the need for advances in treatment modalities.[8]

The shunting procedures are of two general varieties:

(a) **Intracranial shunts:** These connect the ventricles to other intracranial areas such as the basal cisterns. Ventriculocisternostomy, in which one or both lateral ventricles are connected to the cisterna magna by polythene catheters, was devised by Rorkildsen for treating obstructive lesions in the region of the aqueduct of sylvius.

(b) **Ventricular or lumbar shunts:** In this type there is diversion of the cerebrospinal fluid from the lateral ventricle into a body cavity or into the right side of the heart. Most surgeons nowadays use the ventriculo-atrial shunt utilising one of the several valves now available for this purpose.

These valves have a similar unidirectional flow of cerebrospinal fluid from the ventricles to the heart. The most popular of these are the Pudenz and Holster valves.[8,7]

The shunt tube has a valve which is pressure regulated to low pressure, medium pressure normal

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pressure or High pressure, so that it only allows normal level cerebrospinal fluid. In infants low pressure valve is preferred because the sutures have not closed. So that it is regulated at 4.5 cm to 7 cm H_2O so that if it increases above 7 cm then the valve closes.

Another modality of diagnosis and treatment which was not employed in this study, but worthwhile mentioning is the "intrauterine shunting". But it is still experimental with improvement in ultrasound equipment and refinement of interpretation it has become possible to diagnose many malformation during the second trimester of pregnancy.[9] Most such anomalies do not require treatment in-utero. Only those malformations that cause progressive damage to the fetus and which are diagnosed at a gestational age precluding elective premature delivery should be considered for in-utero treatment. The hydrocephalus is readily detectable in the fetus by ultrasound because of the abnormal fluid collections in the fetal head.[9] The placement of shunts from within the fetal head to the amnion is technically relatively simple and entails little risk to the mother. Obstructive hydrocephalus was among the first fetal malformation treated in utero.[9]

Fetal hydrocephalus is easily identified on ultrasound due to the abnormal enlargement of the cerebral ventricles. Since ventricular size can be measured with ultrasound it is possible to diagnose

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fetal ventriculomegaly before there has been any increase in biparietal diameter. some fetuses have enlarged ventricles in early pregnancy and then demonstrate either no progression or even a gradual improvement in the ventriculomegally. Serial observations are important in evaluating fetal ventriculomegally in order to document progressive worsening of the hydrocephalus or at least its failure to decline in the second trimester of pregnancy.

Outcome of Treatment

Results of treatment depend on patients' selection. Often times at our hospital patients come very late, when the brain mantle is very thin and the brain has been completely squashed. In this study 5 (17.8%) came with over 20 cm deviation from the 50th percentile, as shown in Table IV below. Out of the 5 one had an outstanding occipital frontal circumference of 91 cm.

Table IV: The 50th percentile of Occipital Frontal circumference deviation of the 28 patients

Occipital Frontal Circumference deviation (cm)	Number of Patients	Percentage
1 - 10	11	39.3
11 - 20	12	42.9
21 - 30	4	14.3
31 - 40	0	0
41 - 50	0	0
51+	1	3.6

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17.8% of the patients presented with very advanced hydrocephalus over 20 cm deviation from the 50th percentile of the hydrocephalus.

Sometimes the hydrocephalus develops since the 8th week of intrauterine life, therefore those born with hydrocephalus of intrauterine origin fair very badly. The size of head reflects the amount of brain damage. Other factors of importance to note are the degree of mental retardation from the primary disease and other associated neurotube defect.

COMPLICATIONS

Common complications encountered include: Peritoneal absorptive failure, shunt breakage and peritoneal cyst formation.[3]

Shunt infection: The incidence of shunt infection has declined with improved aseptic techniques.

In this study non of theoperated patients developed infection. All were covered with prophylactic antibiotic intraoperatively and the immediate post-operative period.

- Following the rapid or relatively rapid decompression of the hydrocephalic brain by a shunting procedure, there may be dramatic collapse of the cerebrum, tearing bridging vessels, with subsequent formation of subdural haematomas. Even when these veins are not torn,

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- Penetration of an abdominal organ although rare, may be difficult to diagnose because of a lack of abdominal symptomatology with intermittent fever indicative of infection.

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