A DISSERTATION OF TEN SURGICAL CONDITIONS

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A dissertation of ten surgical conditions submitted in partial fulfilment of requirements for the award of the degree of Master of Medicine (Surgery) of the University of Dar es Salaam

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A REVIEW OF TEN SURGICAL CONDITIONS

BY:

GABRIEL K. MARIKO M.B.CH.B. (MAK.)

A DISSERTATION SUBMITTED IN PART FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF MEDICINE (SURGERY) UNIVERSITY OF DAR ES SALAAM.

1983.

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DECLARATION

I declare that this work has not been submitted to any other University for the purpose of acquiring a degree or for any other purpose.

Dr. G. K. Mariko

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A C K N O W L E D G E M E N T S

I want to take this opportunity to thank all those who have in one way or another helped me in the preparation of this dissertation.

In particular I want to thank Prof. Philip Hiza, my supervisor in this work, for taking time to go through the manuscripts and offering valuable suggestions and corrections.

Other members of academic staff of the department of Surgery, including Prof. J. Philip Prof. J. Shija, Mr. B. Kirei, Dr. M. Aziz Dr. S. Mlay and others have made my period of residency training in surgery, an enjoyable though hectic experience. Prof. P.M. Sarungi and his staff at the Orthopaedic/Trauma department, and Dr. B.M. Minja and his staff at the Ear, Nose and Throat, Unit, have made valuable contribution to my training in surgery. I want to take this opportunity to thank them all.

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I N T R O D U C T I O N

One of the requirements, for the degree of Master of Medicine in General Surgery is the submission of a disseration of ten surgical conditions, each of different nature, and the author should have participated in the management of patients presented. Of these 10 conditions, five should be of general surgery, three of a surgical specialty of one's choice, and two of orthopaedics and trauma surgery.

The choice of conditions to write has not been an easy one. However in general surgery, I tried to balance between emergency surgery and cold surgical conditions. Therefore I wrote on Volvulus of the Sigmoid Colon, perforated duodenal ulcer and acute appendicitis, to represente emergency surgery and portal hypertension and obstructive jaundice to represent cold surgical conditions.

The choice of a surgical speciality to write on, has equally been difficult. After considering several alternatives, I finally chose to write on surgical oncology. Although surgeons have been treating cancer surgically for several milleniums it is only recently that surgical oncology has come up as a surgical speciality on its own right, in the same level as Urology or neurosurgery. The main reason which prompted me to write on this subject howver is the nature of the conditions we admit in surgical wards at Muhimbili Medical Centre. Despite inadequate facilities, between 30% and 50% of all surgical admissions, and over 80% of all surgical referra ls from up-country hospitals are for cancer patients, and at any single time nearly 40% of all patients in general surgical wards have cancer. So large part of our practice is oncology, and writing on this, as a speciality is in no way, inappropriate.

In Orthopaedics and Trauma Surgery I chose to write on Epiphyseal injuries and traumatic head injuries; two conditions which are very common in our environment.

In these papers I have tried to be as brief as possible; to avoid controversies even at the expense of making discussions less interesting, to avoid making this a long dissertation.

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A. GENERAL SURGERY

1. SIGMOID VOLVULUS

SUMMARY

A case of intestinal obstruction due to sigmoid volvulus in a middle aged male is presented, together with a summary in a tabular form of twelve other cases seen in a two year period (June 1980 - June 1982) in general surgical firm I, Muhimbili Medical Centre. Ten were managed in the emergency situation by laparatomy and primary resection of the redundant sigmoid colon, and end to end anastomosis. In two patients there was gangrene of the sigmoid colon, at operation and general peritonitis. These were managed by resection of the gangrenous sigmoid, closing off the rectal stump and bringing out the proximal portion as an end colostomy in the left iliac fossa. The colostomies were later closed after an average of four months.

Primary resection and anastomosis, of the sigmoid colon in cases of uncomplicated sigmoid volvulus, is defended and advocated in our environment, where follow up of patients post-operatively is at best inadequate and often non-existent. Shepherd's procedure of sigmoidoscopic deflation followed by an elective sigmoid colectomy, is seen as presenting two potential hazards. One is that sigmoidoscopic deflation is not as easy as most people think it is, and in inexperienced hands it is potential very dangerous. Secondly, in a busy department, patients put on an elective list are likely to postponed every now and then, because of the many emergencies and the long waiting lists. So many patients thus managed, will have recurrences, with increased morbidity and mortality. Therefore any form of treatment which solves the patients problems in one session even at a slightly increased risk of mortality is bound to be attractive.

A critical review of the literature on the aetiology, pathogenesis, clinical presentation and management of sigmoid volvulus is presented.

INTRODUCTION

Volvulus is a torsion of the bowel around its mesentery. Sigmoid Volvulus therefore results from a torsion of the sigmoid colon around its mesentery, which causes symptoms of partial or complete obstruction of the bowel and strangulation of blood vessels or both.

Volvulus of the sigmoid colon is the commonest cause of intestinal obstruction in several African Countries, especially in the north-eastern part of the continent, comprising of Ethiopia, Kenya and the Sudan. (Davey W.W. 1968, Burkitt D.P. 1968; McAdam Sir I : 1961; Shepherd J.J. et al 1965, 1966, 1967, 1971; Wambwa 1974).

Worldwide, Sigmoid volvulus is common in the Middle East, Eastern Europe and northern Europe, but less common in Western Europe and the United States of America and Canada (Bruusgard C. 1974; Cohn I. 1972).

In Tanzania, Shija (1975) considers sigmoid volvulus to be a rare entity accounting for only 4% of all cases of intestinal obstruction. However experience in surgical firm I of the Muhimbili Medical Centre, where the author spent two years of general surgical training, at leest four cases of sigmoid volvulus are seen and operated upon every month. Davey (1968) quotes a figure of at least 3 cases every month. This would indicate that incidence of volvulus is on the increase. However, Shija sent a questionnair to 37 hospitals, spanning the whole country, and his statistics are therefore more representative of the incidence volvulus in the whole country.

CASE PRESENTATION

S.J. IP. NO. 259931, is a 40 year old male, admitted via the Casualty Department with a four days history of:-

a) abdominal distension

b) abdominal pain

c) constipation

d) vomiting

The pain was of a colicky nature and has been gradually increasing over the preceding four days, and so was the abdominal distension. The patient had not opened his bowels for the four days of his complaints, although his appetite has been normal. He felt a constant desire to defaecate, but when he goes to the toilet, nothing comes out. Vomitting was not present initially, but he vomitted once, as he was about to come to hospital.

On examination he was an ill - looking middle aged man; who was severely dehydrated with a dry and furred tongue. His blood pressure was 100/60 and pulse 92/min; it was regular small volume. He was afebrile. The abdomen was grossly distended and rigid. The bowel sounds were increased. The digital rectal examination, showed an empty rectum. Other systems were essentially normal.

An emergency abdominal X-ray was ordered for erect and supine views; which showed widely dilated sigmoid colon towards the right flank and air fluid levels.

The patient was prepared for emergemency laparatomy as follows:-

i) An intravenous infusion of normal saline and dextrose was instituted, and 1.5 litres of each infused before operation.

ii) Blood grouping and cross matching were ordered and two units of blood were requested and obtained in preparation for surgery.

iii) A naso-gastric tube was inserted and emptying of gastric contents by aspiration started.

The patient was taken to the operation theatre about four hours after admission.

Details of the Operation

Anaesthesia was induced with thiopentone 250mg intravenously and maintained with nitrous oxide, halothane and oxygen mixture. Flaxedil 120mg intravenously was used for relaxation.

Position: The patient was placed in a supine position and the whole abdomen, from the xiphisternum to the groin, was swabbed and cleansed with an antiseptic colution containing chlorhexidine, and this was followed by flavine-in-spirit.

The patient was then draped in sterile towels, and a generous left paramedian incision was made, extending above and below the umbilicus and abdomen opened.

Findings. A massively distened sigmoid colon was found which has twisted round itself at the base. The bowel was however viable, and was therefore untwisted. Deflation was then done, by the Monks - Moynihan manouver. This is a suction deflation of distended large bowel using a large bone needle (20 s.w.g.) connected to a suction apparatus, through a glass connection and compressible rubber tubing (Dudley H.A.F. 1977).

Resection of the redundant sigmoid colon was done, and end - to-end anastomosis of the remaining part in two payers of interrupted sutures was done. The inner layer was of interrupted chromic catgut

2 - 0 and this was followed by sero muscular Lembert sutures of silk 2 - 0 Peritoneal lavage with warm saline was done and abdomen closed, leaving a corrugated rubber drain which was brought out through a separate stab wound.

The abdomen was then closed in 3 layers; chromic catgut No. 1 - 0 to peritoneum and inner rectus sheath as one layer; prolene 1 - 0 to outer rectus sheath and silk 2 - 0 to skin.

The post-operative course: The patient was given analgesics (pethidine) for post operative pain. Intravenous fluids and naso-gastric suction were continued for five days until bowel sounds reappeared, when these were discontinued and the patient was allowed to drink oral fluids. The post operative course was unremarkable and the patient was discharged on the eight post operative day. He was subsequently followed up at the post operative follow up clinic for two months, and finally discharged in a very healthy condition.

SUMMARY OF 12 CASES OF SIGMOID VOLVULUS

TABLE I AGE AND SEX DISTRIBUTION

AGE GROUP (YEARS)	MALE	FEMALE	TOTAL
Less than 20 years	-	-	-
20 - 29	6	-	6
30 - 39	4	-	4
40 - 49	1	-	1
More Greater than 50	1	-	12

N.B. There were not females in the patients presented

TABLE 2 PRESENTING SYMPTOMS AND SIGNS

SYMPTOMS AND SIGNS	NO. OF PATIENTS	%
Abdominal distension	12	100
Vomiting	3	25
Pain	12	100
Constipation	10	85
Diarrhoea	1	6.5
High pitched sounds	9	75
Empty rectum	10	85

TABLE 3 DURATION OF COMPLAINTS

DURATION IN DAYS	NO.	%
1 - 5 days	11	93.5
6 - 10 days	1	6.5
More than 10 days	-	-

TABLE 4 DIAGNOSIS

METHOD OF DIAGNOSIS	NUMBER OF CASES	%
Clinical diagnosis	10	85
Radiological diagnosis	7	60
Diagnosis at operation	2	17

TABLE 5 TREATMENT OFFERED

TREATMENT	NO.	OUTCOME	MOTALITY RATE
Sigmoidoscopic deflation	-	-	-
Paul-Mickulics exteriorisation	-	-	-
Hartmans operation	2	All survived	-
Primary resection and anastomosis	10	All survived	-

DISCUSSION

AETIOLOGY:

For a volvulus to form two prerequisites must be present. These are:-

i) A long loop of the sigmoid colon with a freely mobile mesentary.

ii) Close approximation of the two limbs at the points where they are tethered to the posterior abdominal wall by parietal peritoneum. Due to its varied distribution, cutting across racial, genetic social and geographic lines, the aetiology of volvulus cannot be pinpointed to a single cause. Several factors are therefore implicated.

a) DIET

A diet poor in animal protein but rich in vegetables and residue, and which is usually taken in bulky quantities predisposes to volvulus (Bruusgard 1947). This type of diet is common in Africa, Asia and Eastern Europe where volvulus is also common. Bruusgard also points out the increase in the incidence of sigmoid volvulus in Northern

and Western Europe, during World War II, when rations had to consist of diets similar to the ones taken in countries where volvulus is common. Shephered while not denying that dietary factors may be important, in the aetiology of sigmoid volvulus, plays down its importance, in a disease which is almost exclusively a male disease in Uganda, although females eat the same diet. However, several authors, including Brumsgard explain away this apparent anomaly, on the generally wider female pelvis, which can easily accomodate the elongated sigmoid loop.

Another important factor in the diet, is the association of volvulus, with the compound 5-Hydroxy-tryptamine which occurs in rather high concentrations in plantain, which is a staple diet of several East African tribes. This substance increases intestinal motility on intravenous injections, and this might predispose to volvulus. Shephered noting this facts, doubts whether 5HT plays any role, as it is easily degraded by digestive enzymes when taken orally with the bananas:-

a) Congenital redundancy of the colon.

Barium enema studies done in Eastern Europe show the sigmoid loop is elongated and redundant from childhood, and this is thought to be a congenital occurrence. While no comparative studies have been studied in the African, Ogilvie (1946) thinks that congenital redundancy of the sigmoid colon is an important aetiological factor, an assertion that is disputed by Shephered et al (1968).

b) Chronic constipation with laxative abuse and mental illness are associated with volvulus of the sigmoid colon, and most patients with sigmoid volvulus in western countries originate

in psychiatric institutions. (Shields I. 1974; Cohn I. et al 1972). Chronic laxative abuse and mental illness are however not associated with sigmoid volvulus in the African.

c) Megacolon: Adamson and Aird (1932) define megacolon as a condition in which there is dilatation and hypertrophy of some part of the colon or whole in the absence of mechanical obstruction. Shephered (1965) recognises at least four categories of megacolon, namely:-

1. Megacolon of habitual constipation which is synonymous with functional megacolon. This is common in children who have had faulty toilet training and in psychotics.
2. Congenital megacolon (Hirchsprung's disease) is due to congenital absence of ganglion cells, in the Auerbach's and Meissner's plexuses, resulting in uncoordinated peristaltic movements and dilatation and hypertrophy of the proximal part. Shepherd did a series of biopsies in patients presenting with sigmoid volvulus but was unable to demonstrate the absence of an aganglionic segment.
3. Megacolon of Chaggas disease. This is a parasitic disease caused by Trypanosoma cruzi, which is commonly seen in Souther America. It attacks the intermurral plexuses of nerves in most parts of the intestines, resulting in dilatation and hypertrophy of the proximal portion. The disease doe not however occur in Africa, and Shepherd in his series was unable to demonstrate histological abnormality in the ganglion cells, serologic tests for Trypanosoma cruzi were negative.
4. Acquired megacolon from other causes. Shepherd and Wright (1965), consider that, the majority of patients treated for sigmoid volvulus in Kampala, have a megacolon as predisposing

factor. They sub-stantiate this point, by radiological examination of patients with sigmoid volvulus who had an initial conservative management by sigmoidoscopic reduction and are awaiting definitive treatment of elective sigmoidectomy. They were able to demonstrate three features:-

i) Dilatation of the pelvic part of the sigmoid colon, with no normal haustrations and inability to contract on evacuation of the gut. At laparatomy it is flabby and does not contract on pinching.

ii) The muscle coat of the same region is thickened and the longitudinal muscle is broad and spread out, instead of being into three narrow bands, taenia coli.

iii) The mesocolon is thickened, elongated and its blood vessels and lymphatics enlarged.

d) Mesocolitis The presence of a marked degree of pelvic mesocolitis leading to a long but narrow pelvic mesocolon has been observed by many workers. (Wright 1928, Bruusgard 1947, Dean & Murray 1952, Andersen 1956, Sinha 1969). This can be attributed to chronic constipation, and recurrent attacks of "Physiologic" volvulus leading to fibrosis of pelvic mesocolon. This produces a tough, thickened and fibrosed axis around which the pelvic colon loop twisted. The presence of fibrosis in the pelvic mesocolon had however not been observed by African workers, and it is debatable whether it is the cause or sequela of sigmoid volvulus.

PATHOPHYSIOLOGY

Physiologic volvulus is formed when there is a clockwise torsion of 180° of the rectal loop under the proximal loop, (Wilms 1906). This usually corrects itself spontaneously and does not usually lead to intestinal obstruction.

However in a counterclockwise torsion of 180°, which can occur, the proximal sigmoid loop may be squeezed between the scybala filled distal sigmoid loop and the posterior abdominal wall leading to a closed loop obstruction and circulatory disturbances in the sigmoid flexure.

The physiologic volvulus, may however proceed to cause symptoms, if the sybala filled distal loop changes its position and falls over the emptier proximal loop, because of body movements, resulting in a 360° torsion. The degree of torsion might progress to 540° or even 720°, but this occurrence is rare.

Groth K.E. (1934) showed that torsion of the sigmoid and its mesenteric axis, is combined with an axial torsion of the bowel itself because the two sigmoid limbs are themselves fixed. Thus axial torsion is twice as great as mesenteric torsion and a 180° torsion will produce a 360° axial torsion, and the occurrence of symptoms depends on whether the torsion is evenly distributed over the loop or not. Stenosis then leads to bowel distension and obstruction. The increased peristalsis forces air and fluid into the loop because the torsion site acts as valve, allowing in air but not allowing it to escape (Bruusgard O. 1947). In a study of 17 patients at Ullevaal Norway, Bruusgard showed that 1 patient had a 540° torsion; 6 had 360° torsion; 1 had a 270° torsion and 9 had 180° torsion; and Frimann - Dahl (1960) gives the approximate percentages of the different degrees of torsion as; 180°, 35%, 360°, 50% and 540° 10%. The direction of torsion is variable, but clockwise torsion is more common (Andersen 1956).

CLINICAL PRESENTATION

Patients with sigmoid volvulus present with colicky abdominal pain and progressive abdominal distension. Vomiting is not an early feature, although it may occur later. The patients are

usually, absolutely constipated for a number of days, and many patients have a previous history of previous attacks. The general condition of the patient is usually suprisingly good despite massive abdominal distension.

In a few patients especially in the younger age group, the disease is more acute, and patients are in a poor condition, with only moderate distension, constipation and vomiting. In these patients strangulation is common. The presentation in this series are shown in Table 2.

Investigations:

The basic work-up of the patient would consist of a haemagram, a urinalysis and serum electrolytes.

To confirm the diagnosis however radiological examination is mandatory, and plain abdominal X-rays in the supine and erect positions are taken. The supine film will show a large inverted-U-shape shadow due to a massive distended sigmoid loop, with no haustration, and is likened to the Greek letter Omega Ω. The supine view may also show three dense lines converging downward from the right hypochondrium to the site of stenosis, the middle line being the most constant, and is produced by the two walls of the loop; Frimann Dahls sign (1960).

In the erect film, there may be an enlarged colon, pushed to the right with, two air-fluid levels, in the two limbs, likened to a pair of scales. These features are pathognomonic of sigmoid volvulus.

If a gangrenous loop perforates, air escapes and may be noticed radiologically under the diaphragm. This however is rare. In elective cases, e.g. in sub-acute cases, a barium enema might be done. A characteristic deformity, resembling a "birds beak" is described

above, the characteristic feature were noted in the erect and supine films as stated above.

According to its clinical presentation volvulus of the sigmoid colon has been classified into:-

a) Acute fulminating type; which tends to occur in younger patients. They have a shorter and more severe clinical course, vomiting, prostration and shock. Abdominal distension is moderate.

b) Chronic type. In contrast, these patients present with marked abdominal distension, with complete obstipation for several days, and only moderate colicky abdominal pain. There is usually a history of similar attacks in the past. A classification based on the presence of absence of gangrene of the bowel was suggested by Boggs and Ratcliffe (1960). Although it is not always possible to tell clinically before operation, whether the bowel is gangrenous or not, the presence of features of peritonitis viz: absent bowel sounds, guarding, prostration, shock and increased white cell counts are indicative of gangrenous bowel. The later classification seems logical since the ultimate mode of treatment is based on the presence or absence of gangrene.

TREATMENT

The aim of treatment is to deal with the emergency and to prevent reccurrence. The choice of method of treatment will depend on the preference and experience of the surgeon, and the condition of the patients and whether his bowel is viable or not.

a) Viable sigmoid loop

i) Sigmoidoscopic reduction followed by elective resection. This was first advocated by Bruusgard (1947), and was successfully used by a number of workers (Pool Dunavent 1951; Andersen 1956 Bolt 1956) and esepcially advocated by Shepherd (1968).

In this method the patient, is put in a left lateral position; knee elbow position or Lithotomy position, depending on the preference of the surgeon, and a well lubricated sigmoidoscope is introduced, slowly, noting the appearance of the bowel mucosa, until, the point of obstruction is reached. Then a wide bore rectal tube is negotiated gently through the kink, and if the procedure is successfull, this would be followed by a gush of gas and liquid faeces, and prompt relief for the patient. The tube is then plastered to the buttock, and patient returned to the ward to be prepared for elective resection of the redundant sigmoid colon. Elective resection has been adviced at intervals ranging from seven days (Andersen 1956, Shepherd 1968) to twelve weeks, after the acute attack.

The main draw - back of this method has been, that a sizable proportion of patients, managed conservatively in the emergency situation, do not turn up for elective resection, and return only when there is a reccurrence of acute obstruction. In Shepherds cases 42%, returned because of recurrence. There is also danger of perforation of sigmoid colon, and the possibility of missing a gangrenous loop with ghastly consequences for the patient.

b) Primary resection and anastomosis:

This method of surgical treatment fulfills both aims of alleviating the acute obstruction and preventing recurrence. The first series of 11 patients were treated by Dean and Murry (1952) with one death, followed by Delafield et al 1953; Shepherd 1968 and Sinha (1969). Sinha (1969) has an impressive record of 149 primary resection and anastomosis of the sigmoid colon in India, but his mortality rate for this specific group of patients was not recorded. Shepherd (1968) reported operative treatment of 35 patients with viable gut with a mortality rate of 14%.

Of these 6 had a primary resection with end-to-end anastomosis with a mortality of 1 (16%) and 29 had a Paul - Mikulicz resection and exteriorisation with 4 deaths (14%).

Whenever possible a method of treatment which fulfils both aims of treatment; relief and permanent cure in a single session, is advocated. This is especially so, in the under - developed countries, where many patients are in the waiting list, and many disappear if they are put in a waiting list for elective resection.

In the series of patients presented above, primary resection and anastomosis was done in ten patients with viable bowel, and there was no mortality.

iii) Operative detorsion of the loop

In this method a laparotomy is done, and the sigmoid loop is restored to its normal lie, after guiding a rectal tube into the loop and deflating it. The mesentery is then fixed to the posterior parietal peritoneum. In Shepherds series simple detorsion without fixation was done in 49 occasion with 8 deaths, and 14 (42%) returned with a recurrent attack after a few weeks. Fixation after detorsion was carried out in 213 occasions with 17 (8% deaths) and 81 (41%) returned with a recurrent attack after a few weeks. Clearly this method has nothing to commend it.

GANGRENOUS BOWEL

If gangrene is suspected, there is no room for conservative treatment, and operation treatment must be performed. If at operation, gangrene of the sigmoid loop is confirmed then three choices of operative procedures are available, primary resection and anastomosis, Hartman's procedure and Paul - Mikulicz resection, and exteriorisation. In Shepherd's series of 30 cases with supervening gangrene of the sigmoid colon, 2 cases had primary resection and anastomosis with 1 death; 20 had Paul - Mikulicz resection 9 deaths, and 8 had Hartman's resection with 1 death. Dean and Murry (1952) did primary resection and anastomosis in 3 patients and 2 died.

stomosis of the colon in the presence of gangrene and peritonitis is a hazardous procedure and should be condemned. In the majority of cases, the rectosigmoid junction is usually involved in the devitalisation process, and bringing this out together with the descending colon in a Paul - Mikulicz procedure, probably caused the high mortality noted by Shepherd. The procedure of choice in this situation is a resection and closure of the distal stump as low down as possible, and bringing out the proximal viable descending colon as an end - colostomy, which would be closed later when inflammation and sepsis have settled down. This was done in two of our patients and they survived.

Volvulus of the sigmoid colon is a life threatening condition and should not be taken complacently, as it is followed by unacceptably high mortality if gangrene of the sigmoid loop superveness.

A number of factors are responsible for the high mortality viz perionitis; endotoxic shock; hypovolaemic shock (every 1 metre of grangrene of bowel is associated with loss of one litre of blood) and electrolyte imbalance especially hypokalemia.

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2. PORTAL HYPERTENSION - SURGICAL TREATMENT

SUMMARY

A case of Portal Hypertension, in a 23 year old female, managed by a conventional splenorenal shunt is described. This patient had a moderately active cirrhosis of the liver. During the same period a 24 year old male was admitted to the same firm with repeated attacks of haematemesis. Subsequent work up confirmed portal hypertension with bleeding oesophageal varices. This patient had a massively enlarged spleen, with a severe degree of hypersplenism, which necessitated that splenectomy be done as an initial procedure. The patient however died later due to progressive hepatocellular failure and uncontrolled haemorrhage from bleeding oesophageal varices.

The patient in whom a splenorenal shunt was done, recovered without hepatic encephalopathy, and was discharged, to be followed up by the referring physician, in a good general condition.

A critical review of the lietrature as it pertains to aetiology, pathogenesis, clinical presentation and modalities of treatment is presented.

INTRODUCTION

Portal hypertension is a common condition in East Africa. (Leather L.H. 1961; Miller J.R.M. 1967; Bagshawe A. 1970, 1973; Carruthers R.M. et al 1978). It's aetiology is however quite different, from that seen in the developed countries of the West. In a series of 30 consecutive cases presenting with portal hypertension with bleeding oesophageal varices in Zambia, 70% were

due to portal fibrosis, caused by schistosoma mansoni infestation (Carruthers R.H. et al 1978). Similar figures were ported in Zimbabwe (Gelfand M. 1974).

In Nairobi, schistosomal hepatic fibrosis was the commonest cause of portal hypertension, followed by idopathic splenomegaly, portal vein thrombosis and liver cirrhosis in that order (Bagshawe A. 1973). Although no similar figures for Tanzania are available, schistosomiasis is as common here, as in the above named neighbouring countries, and there is therefore no reason to assume a different pattern.

The prognosis in portal hypertension is influenced by the associated impairment of liver function. In schistosomal hepatic fibrosis, with oesophageal varices, there is minimal liver damage, and porto - systemic shunt operations are associated with long term survival (Zeegan et al 1970), unlike in liver cirrhosis where overall survival of these patients is uninfluenced by these shunts (Resnick et al 1974).

CASE PRESENTATION

Case I:

M.N. IP. NO. 249627, a twenty three year old Sukuma female, was referred from Bugando Hospital, Mwanza with complaints of:- haematemesis, left hypochondral pain, and a mass in the left flank of the abdomen. Her haematemesis has been three times in the last six months prior to admission to our unit. The bleeding episodes had been severe, and all the time she required blood transfusion.

She does not know the exact duration of her other abdominal complaints. She has been brought up in the southern shores of Lake Victoria close to Mwanza town, an area which is hyperendemic for

schistosomiasis. She does not know whether she had ever been treated for schistosomiasis.

On examination, she was an ill looking girl, with severe pallor of conjuctiva and buccal mucosa. She was not however jaundiced and had no cutaneous stigmata of chronic liver disease. The respiratory, cardiovascular and central nervous systems were essentially normal. The abdomen was distended, and the liver and spleen were massively enlarged. There was no caput medusa.

A provisional diagnosis of portal hypertension with oesophageal varices was made and the patient was subjected to a diagnostic work up, to confirm the diagnosis as follows:-

i) A full blood picture, showed hypochromic microcytic anaemia

ii) Upper G.I series showed extensive oesophageal varices in the ~~late 20cm of the~~ distal oesophagus.

iii) Fiber optic oesophago - gastroscopy showed

a) Oesophagus; extensive varices in the distal ~~20~~ 5cm, with no oesophagitis or free bleeding.

b) Stomach; Fundal varices, normal distal stomach

iv)

Liver Function Tests	On admission	After 6 weeks
a) Bilirubin - Total	5.6mg/dl	1mg/dl
Direct	5.2mg/dl	
b) Serum Protein	6.1g/dl	6.4g/dl
Albumin		4.2g/dl
c) Alkaline Phosphatase	319 I.U/l	200 IU/l
D) SGOT	26IU/l	10IU/l

v) Bleeding indices normal

vi) Liver biopsy showed moderately active cirrhosis (HP No. 3831/81)

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As the patient was severely anaemic and weak, her general condition was brought up, by repeated blood transfusion, bed rest and vitamin K injections, in preparation for surgery. She was however considered to be a good risk case, according to Child's criteria (1964) for a portosystemic shunt.

A conventional proximal spleno-renal shunt was planned, and it was done approximately two months after admission.

Operation

The patient was put in a supine position with sand bags at the lumbar spine.

Anaesthesia was induced with 500mg thiopentone intravenously, and maintained with halothane-oxygen-nitrous oxide mixtures. Relaxation was achieved by repeated injections of pavulon.

Operative details

A generous left paramedian incision was made and abdomen opened. There was a markedly enlarged spleen, with multiple areas of infarction; a large cirrhotic liver which was biopsied and there was no ascites.

The spleen was mobilised from its bed, it's vascular pedicle identified and carefully exposed. The splenic artery was double ligated with 2 -0 silk sutures and cut. The splenic vein was held with a bulldog clamp and cut, and the spleen was removed. The splenic vein was further mobilised under the pancreas for about 5cm, ligating and cutting tributaries from the pancreas joining it. The left renal vein was also mobilised, and freed from the covering peritoneum. The left ovarian vein, was ligated and cut flush from the renal vein. After adequate mobilisation was achieved, an end-to-side anastomosis of the splenic vein to the left renal vein was made, using ethilon 5 - 0 in a single layer of continous sutures.

The clamps were then released and the veins were immediately engorged with blood.

Haemostasis was achieved, the abdomen was closed in layers, leaving a corrugated rubber drain to the splenic bed, and out through a separate stab wound.

The post-operative management consisted of:-

1. Control of pain with analgesics pethidine 100mg hourly for 24 hours was used.
2. Blood transfusion. Two 500ml units were given
3. Vitamin K injection and antibiotics
4. Monitoring of vital signs. Respiration, pulse and blood pressure
5. Looking for signs of hepatic encephalopathy viz; mental confusion, fetor hepaticus, and flapping tremors.

The patient continued to make a slow but steady recovery and was ready for discharge, about one and half months after operation. There were no signs of hepatic encephalopathy throughout the post-operative period. She was however discharged with a letter to the referring physician to continued following her up.

Case II:

Z.M. IP. NO. 259263, was a 24 year old male, a Msambaa by tribe, was referred to our unit from Morogoro Hospital with complaints of

i) Haematemesis together with epistaxis and malaena stools

ii) Abdominal discomfort, left hypochondrial pain and extreme body weakness. The problems have persisted for over ten years and the patient has been hospitalised in several hospitals, on and off, over this period. Prior to this admission, he started having nausea followed by vomiting which was not projectile, and the vomitus consisted of previously eaten food and altered blood, about 1 - 2 litres. He also started passing loose dark stools, but terminal stools contained bright red blood. He had a long

standing dull ache in the hypochodrium, associated with dizziness and extreme body weakness.

On examination, he was a young boy who looked small for his age, with extreme pallor of the conjuctiva and buccal mucossa. He had no oedema of the extremities and he was not in cardiac failure. There was a massive splenomegaly and ascites.

Subsequent fiberoptic oesophago - gastroscopy confirmed the diagnosis of portal hypertension with bleeding oesophageal varices. Haematological investigations, showed an iron deficiency anaemia, presumably due to blood loss, but attempts to correct the anaemia with blood transfusion and total dose imferon infusion failed.

This clearly pointed to a presence of hypersplenism. It was therefore decided to remove the spleen as an initial procedure, while bringing up the patient's condition for a portosystemic shunt operation.

Splenectomy was done on the fourth week after admission. It however did not bring the desired effect as the patient made a slow recovery during which he had several episodes of variceal bleeding associated with hepatic encephalopathy of increasing severity. The patients condition continued to deteriorate and he died of sever hepatocellular failure and bleeding oesophageal varices, before a portosystemic shunt could be done.

DISCUSSION

Normal pressure within the portal venous system is about 150 - 180mm of saline, above the right atrium. A rise in portal pressure occurs as a result of impediment of blood flow within the portal

system or its drainage vessels. The impediment to blood flow may be; prehepatic, intrahepatic or post hepatic. It may also result from conditions associated with increased portal blood blow e.g. tropical splenomegaly syndrome.

The rise in portal pressure causes venous dilatation in the portal bed, and blood seeks alternative pathways back to the systemic circulation. The most important collateral channels to open up are those between the left gastric veins and the azygos system at the distal oesophagus and upper stomach; which is also aided by the positive intra - abdominal pressure against a negative intrathoracic pressure. Dilated veins develop within the submucosa; around the oesophagus and they may be seen on gastroscopy or barium swallow.

The other channels to open up as a result of portal hypertension, are veins around the umbilicus due to opening up of the umbilical veins occluded at birth forming the "caput medusa" and the veins connecting the superior haemorrhoidal vein and the inferior haemorrhaoidal and middle rectal veins. This may result in haemorrhoids.

Portal hypertension is important clinically because, rupture of oesophageal varices, could result in massive haemorrhage from the upper gastro-intestinal tract which is life threatening.

Aetiology of portal hypertension

1. Prehepatic causes: there are two main causes of prehepatic portal hypertension - portal vein thrombosis and increased portal blood flow. In contrast to the developed Western countries, portal vein thrombosis is an important cause of portal hypertension in Kenya (Bagshawe A. 1973) accounting to 24% of cases. It is also common in the Indian subcontinent (Basu A.K. et al 1963). The aetiology of portal vein thrombosis, in tropical countries

is obscure, but contributory factors include, umbilical sepsis, the high incidence of chronic intestinal infestations and frequency of severe dehydrating illnesses e.g. typhoid fever, dysentery or even cholera. Other causes are:-

a) Compression due to local glandular involvement in e.g. hepato cellular carcinoma or carcinoma of the head of pancreas.

b) Blood dyscrasias e.g. polycythaemia, thrombotic thrombocytopaenic purpura and secondary to splenectomy. Splenomegaly causes portal hypertension as a result of a greatly increased portal blood flow. Tropical splenomegaly syndrome is common in East Africa and it is associated with portal hypertension, although it does not commonly present with bleeding oesophageal varices. (Leather H.M. et al 1961; Marsden P.D. et al 1965, Miller J.R.P. 1967). Splenomegaly due to other causes e.g. congestive splenomegaly is also associated with portal hypertension.

2. Intra-hepatic portal hypertension

In East Africa, this is most commonly due to schistosomal hepatic fibrosis, followed by cirrhosis (vide supra).

i) Schistosomal hepatic fibrosis; follows infestation with shistosoma mansoni, and it depends on the worm load and duration of the infestation. The ova are deposited in the terminal radicles of the portal vein, surrounded initially by a granulomatous reaction, followed by fibrosis (peri-portal fibrosis) which impedes blood flow. The lesion are patchy in distribution and hepatocellular damage is minimal.

ii) Cirrhosis - In this condition there is destruction of liver cells, followed by fibrosis and regeneration of liver nodules. The portal vascular bed becomes distorted.

This impedes venous outflow from hepatic vein tributaries and sinusoids - causing a post - sinusoidal hypertension. It is the commonest cause of portal hypertension in Britain and other western countries (Dawson J.L. 1977).

3. Post-hepatic portal hypertension

This results from obstruction of venous return to the right side of the heart, and it may arise from any condition causing cardiac failure; in constrictive pericarditis and endomyocardial fibrosis. Congenital suprahepatic vena caval webs are rare but they have been described and they present a potentially curable cause of portal hypertension. (Yamamoto et al 1968). Haemodynamic changes in portal hypertension, occur in three stages:-

i) Portal flow to the liver (hepatopetal flow) is maintained at normal or near normal levels, with little or no escape through collateral circulation.

ii) Portal flow to the liver is significantly reduced and the flow through the collateral circulation is high.

iii) Portal flow is zero or reversed; the portal vein therefore acting as an outflow from the liver (hepatofugal flow), and all the portal circulation escapes through the collaterals. (Dawson J.L. 1977).

INDICATIONS FOR SURGERY IN PORTAL HYPERTENSION

There are two main indications in portal hypertension

i) Haemorrhage - once a patient bleeds from oesophageal varices, surgical treatment should be considered as there is a tendency to recurrent bleeding, with increasing risk of death from each episode.

ii) Hypersplenism - There is mild to moderate pancytopeania in patients with splenomegaly secondary to portal hypertension.

This necessitated a splenectomy in our second patient as an initial procedure as it was impossible to correct anaemia between bleeding episodes. The shunt operation is only indicated however, if the patient has bleed from oesophageal varices.

Type of operation for portal hypertension

There are two types of operations available for portal hypertension. These are:-

i) Shunt operations

ii) Direct operations - these attempt to remove, occlude or thrombose the varices locally. They are attended by a high incidence of recurrent bleeding, and they will only be considered briefly here, as they w are not employed in this centre.

SHUNT OPERATIONS

Patients who have bleed, at least once from oesophageal varices are candidates for shunt operations. However, a preliminary work-up is essential to have an:-

a) Accurate knowledge of the patients liver disease including both biochemical function and histology.

b) Accurate radiographs of the pathological portal circulation.

Assessment of liver disease is usually done, using Child's criteria (Child, 1964). These have been modified by Pugh et al (1973) so that points are used for grading.

Child's grading criteria

	1 Point	2 Points	3 Points
1) Serum Bilirubin			
mg%	2	2-3	>3
2) Serum Albumin			
g%	3.5	3 - 3.5	<3
3) Prothrombin time (seconds prolonged)	2	3 - 5	>5

4)	Ascites	None	Mild/Moderate	Gross
5)	ENCEPHALOPATHY	NONE	MINIMAL	SEVERE

The points are then added and the patient is graded as follosw:-

A = 5 - 7 points

B = 8 - 9 points

C = 10 - 15 points

In addition to liver biopsy is mandatory, to ascertain the type of liver sidease present. If hepatoma is suspected a liver scan using technetium (Tc^{99}) can be done, and serum taken for alfa - feto protein. In 60 - 70% of African patients with hepatomas a- feto protein levels are elevated (Hutt Mew 1973).

The bleeding site should be ascertained By fiberendoscopy and barium studies, as peptic ulcers are more common in cirrhotics than in the general population.

Anatomy of portal circulation, can be ascertained by doing a splenic venogram, which can be done pre-operatively or intra operatively. A pre-operative splenic venogram is convenient and its technique is simple. This involves a splenic puncture and therefore prothrombin time and platelet count should be known before hand. The puncture is made in the tenth intercostal space in the posterior axillary line, using a trocar and cannula to a depth of about 4cm, but it may go deeper. Once the required depth is reached the tracar or needle is withdrawn leaving the cannula in position. The cannula is first connected to a manometer and portal pressure is gauged. This is then withdrawn and 40ml of 60% urograffin injected and films taken. (Bagshawe A. 1973).

Complications of shunt operations

These are major operations, which carry a definite risk to life, which is directly related to the severity of liver disease (Turcoate et al 1969). Hepatic encephalopathy is another compli-

to be caused by a combination of factors namely:-

i) Diversion of portal blood from the liver which contain ammonia and other products of protein degradation by large intestine bacterial flora, which are normally removed or detoxified by the liver.

ii) Deterioration of liver cell function - the more severe the liver disease, the earlier the onset of post shunt encephalopathy. However if a portosystemic shunt is done on a patient with a normal liver, he will eventually develop encephalopathy (McDermott et al 1954; Voorhees et al 1974).

iii) Haemodynamic factors - portosystemic shunts divert significant quantities of blood from the liver in those patient with haemodynamic stage I and II. Patients who have the greatest reduction in hepatic blood flow have increased incidence of encepalopathy. Certain factors are associated with an increased incidence of hepatic encephalopathy. These are:-

a) Age:- the older the patient the higher the incidence of post - shunt encephalopathy. (Hourigan et al 1971; Zeegan et al 1973).

b) G.I.T. haemorrhages:- Patients who develop hepatic encephalopathy after gastro-intestinal haemorrhage are more likely to develop post shunt encephalopathy. (Hourigan et al 1971).

c) Diabetes mellitus - patients with this condition have an incidence of post shunt encephalopathy that is twice that of patients without diabetes mellitus. (Voorhees et al 1970; Hourigan et al 1971).

d) Type of hepatic lesion: Egyptian workers have claimed that patients with severe hepatic schistosomal fibrosis and intractable ascites have a high incidence of post shunt encephalo-

pathy (Hassab 1967). This however is contrary to the experience of other authors. (Carruthers R.H. et al 1978; Zeegen et al 1970).

Management of hepatic encephalopathy

Clinical improvement is achieved by:-

i) Decreasing dietary protein

ii) Changing large intestinal bacterial flora

iii) Prevention of absorption from the colon. Upper gastro intestinal haemorrhage probably precipitates hepatic encephalopathy as a result of protein load provided by blood and should be treated by purgation with magnesium sulphate and neomycin. The change in large intestine bacterial flora can be achieved by using wide spectrum antibiotics e.g. neomycin. This is however associated with overgrowth of opportunistic organism e.g. fungi. Some workers have reported use of large oral doses of lacto-bacillus and/or lactulose which act by lowering the pH of the gut and have a purgative effect (Elkington 1970).

TYPES OF SHUNT OPERATIONS

There are two types of portosystemic shunt operations.

i) End to side anastomosis in which the whole of the portal blood is diverted from the liver into the inferior vena cava.

ii) Side to side anastomosis; between the portal and systemic venous systems. All the other types of portosystemic shunts except end to side portacaval shunts fall into this category.

A) END-TO-SIDE PORTACAVAL SHUNT

This is the most widely used shunt and has the lowest incidence

of occlusion due to the large stoma use. It has the highest incidence of post shunt hepatic encephalopathy.

B) SIDE TO SIDE PORTACANAL SHUNT

The portal vein is apposed to the inferior vena cava

C) CONVENTIONAL SPLENORENAL ANASTOMOSIS

The spleen is removed and the end of the splenic vein anastomosed to the left renal vein. It is technically a difficult operation with a high incidence of anastomotic occlusion by thrombosis. (Hallen-beck et al 1961; Voorhees et al 1970). It should not be attempted if the splenic vein is less than one centimentre in diameter. It's main advantage is that any concurrent hyperplenism is corrected. It is also attended with reduced incidence of post shunt encephalopathy, but this is claimed by some, to be due to the high incidence of shunt thrombosis (Barnes et al 1972). This is the method used in our patient.

D) MESENTERICOCAVAL ANASTOMOSIS

In this method the inferior vena cava is transected and the distal portion is closed off. The proximal portion is then anastomosed to the superior mesenterie vein. (Marion et al 1966; Clatworthy et al 1974). This operation is attended with oedema of the lower extremity in adults but it seem to be well tolerated in children. (Clatworthy et al 1974).

E) DISTAL SPLENORENAL SHUNT (Warren et al in 1967; 1974).

This was first described by Warren et al (1967). It decompresses the portal tributaries of the left upper quadrant of the abdomen while maintaning an adequate portal flow. The left gastric and the right gastro-epiploic veins are transected; the splenic vein is dissected behind the pancreas and transected.

The proximal end is oversewn and the distal end is anastomosed to the left renal vein. This was later modified by the same authors by dividing the left renal vein and anastomosing the proximal end-to-side to the splenic vein. The eplenic vein is then ligated in continuity, between the anastomosis and the superior mesenteric vein. (Warren et al 1974).

RESULTS OF PORTOSYSTEMIC SHUNT OPERATIONS

Porto systemic shunt operations have been in use for over 30 year (Whipple 1945, Blakemore 1948). Several large studies have been done to evaluate the results of the various porto-systemic shunt operations. The results of some of these series are given below.

RESULTS OF END-TO-SIDE PORTACAVAL SHUNTS

Author	No. of patients	Mortality (%)	Occlusion (%)	Encepha-lopathy (%)
Voorhees et al (1970)	271	12	4	34
Zeegen et al (1973)	174	10	2.5	24
RESULTS OF CONVENTIONAL SPLENORENAL SHUNTS				
Voorhees et al (1970)	48	12	19	30
Riddel et al (1972)	29	11	7	38
Zeegen et al (1973)	29	14	20	16

For the end to side portocaval shunt, the operative mortality rate is acceptable, but the hepatic encephalopathy rate is high. The lower rate of hepatic encephalopathy reported in some series of patients who have had a conventional splenorenal shunt, probably reflects the higher incidence of shunt occlusion by thrombosis, as in the series with the least occlusion rate, the encephalopathy rate

is highest. (Riddell et al 1972).

DIRECT OPERATIONS

These operations are attended by a high incidence of recurrent bleeding. They are usually used when portosystemic shunt operations are unsuitable. The common direct operations are:-

i) Oesophageal transection - was first introduced by Walker et al (1960). The lower left chest is opened. The muscle layers of the oesophagus are split longituinally and the mucosal tube together with the submucosal varicees are dissected free for about 5cm and resected. The ends are resutured.

ii) Portoazygous disconnection - in this operation the lower 5cm of the oesophagus and the upper 5cm of the stomach are dissected free of vascular connections and resutured.

iii) Boerema - Crile operation and Boerema button operation

In the Boerema - Crile operation the oesophagus is opened longitudinally into the lumen and the varices are underrun with sutures to stop the bleeding and promote occlusion of the varices. The Boerema button operation involves use of a special device. (The Boerema butoon) to achieve a full thickness oesophageal transection via an abdominal incision.

iv) Gastro - oesophageal decongestion

This operations is extensively used by Egyptian workers, who claim that patients with oesophageal varices due to portal hypertension of extensive schistosomal hepatic fibrosis with Ascites are poor candidates for shunt operations (Hassab 1967). It involves doing a splenectomy with removal of all vascular connections in the lower oesophagus and upper two thirds of the stomach.

EMERGENCY TREATMENT OF BLEEDING OESOPHAGEAL VARICES

In a patient who presents with massive upper gastro intestinal haemorrhage due to bleeding oesophageal varices, the approach should be as follows:-

i) Replacement of blood loss

ii) Minimising the risk of hepatic encephalopathy and coma

iii) Confirmation of bleeding site

iv) Active measures to stop bleeding

To minimise risk of hepatic encephalopathy a purgative e.g. magnesium sulphate, is given orally together with an oral antibiotic e.g. neomycin. Fibre optic oesophago - gastroscopy has proved to be a useful tool to ascertain the bleeding site, since up to 40% of patients with oesophageal varices, bleed from associated peptic ulcers or gastritis, especially in patients with liver cirrhosis (Palmer et al 1969).

Arrest of haemorrhage:-

1. Pitocin infusion - 20 international units of pitocin are added to 100ml of normal saline and infused intravenously over a 15 - 20 minutes period. It causes smooth muscle contraction of the portal vascular bed and the mesenteric arteries, thus reducing portal pressure. The effect is however short lived, and it is only a time buying procedure.
2. Ballon tamponade (Sengstaken Blakemore tube). This triple lumen tube is swallowed by the patient and the oesophageal and gastric balloons are inflated to occlude the varices.

The gastric contents can be aspirated by an inner tube that goes right into the stomach. It also permits ice water lavage, which is claimed by some to help stop haemorrhage from varices by causing smooth muscle contraction. The main disadvantages of the

Sengstaken Blakemore tube are that, it is associated with pressure necrosis of the oesophagus, and aspiration pneumonia. To minimise risk of pressure necrosis, it is adviced that it should be deflated 24 - 36 hours after insertion. It usually achieves the desired purpose, of controlling variceal haemorrhage. (Sherlock S. 1964; Pitcher 1971).

Operative measures to stop variceal haemorrhage:-
These consist of either, a direct assault on the varices or an mergency portosystmic shunt operation.

i) Injection of oesophageal varices: Johnsons and Rodgders (1973) in Belfast (U.K.) had a series of 100 patients whose bleeding oesophageal varices were injected, using a sclerosant (ethylolamine oleate) directly into the varices, followed by insertion of a Sengstaken Blakemore tube to produce tamponade for a few hours. A total of 15 - 25ml of sclerosant are injected in each session. They reported a 90% control of variceal haemorrhage. A modified rigid oesophagoscope incorporating a special needle attachment is used. This method should be more widely used.

ii) Ligation of varices is used by some. A left side thoracotomy is done and the oesophagus opened. The varices are underrun by silk sutures. This method is however associated with a high mortality in a poor risk patient (Rothwell - Jackson et al 1971).

iii) Emergency shunt operations are done by some (Orlof et al 1974), and they claim that the mortality rate for the operation, compares well, with that of other procedures. This however has not been verified by other workers (Rothwell Jackson et al 1971; Pugh et al 1973). It is however difficult to justify, a shunt operation, with its prolonged dissection and adverse haemodynamic effects on the liver, in a critically ill patient who is in shock.

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3. OBSTRUCTIVE JAUNDICE AND GALL-BLADDER DISEASE

SUMMARY

Twenty four patients presenting with obstructive jaundice in all the three general surgical firms in Muhimbili Medical Centre over a two year period (1980 - 1982) are reviewed. Two of them are discussed in detail; the first being an elderly man who had a stricture in the distal portion of the common bile duct. A Choledocho - duodenostomy was done, but the patient succumbed to a chest infection. The other, was a middle aged female who presented with acute cholecystitis, managed conservatively, then had a cholecystectomy as an elective procedure.

She had cholelithiasis. The literature on the subject of gall-bladder disease and obstructive jaundice is reviewed. More emphasis is placed on the conditions as they occur in our environment.

INTRODUCTION

Gall bladder disease and obstructive jaundice are not very common conditions in the African. When encountered, obstructive jaundice in East Africa is more often due to malignancy of either pancreas, bile duct or liver, than gall stones (Ochola Abila 1981; Amollo - Odhiambo B. et al 1976).

In this series of 24 patients, no cause of obstructive jaundice could be found in 8 cases; seven had malignancy; six had gallstones, while the remainder had stricture of the common bile duct and chronic pancreatitis.

Gall stones affect about 15 million people worldwide, necessitating a half a million cholecystectomies every year. They are

more common however in Western Europe, the USA, Canada and India, than in Africa and the Far East (Schwartz 1972).

They form, from the less soluble components of bile - cholesterol, bile pigments and calcium. About 80-90 percent are mixed stones and are multiple. Pigment stones predominate in conditions associated with chronic haemolysis, ascaris lumbricoides and E coli (due to B glucuronidase activity). Cholesterol stones form when there is an imbalance in the concentrations of cholesterol, lecithin and bile salts leading to supersaturation. The presence of infection in the gall bladder aids absorption of bile salts and hence precipitation of cholesterol stones.

We have no experience in this centre of medical treatment for gallstones with chenodeoxycholic acid. It is said to be effective in about 60% of cases (Morehead D.T. & Warren K.W. 1976), but treatment has to be continued indefinitely or stones will reform. Cholecystectomy is the treatment of choice for asymptomatic cholelithiasis, as well as for chronic and acute cholecystitis.

CASE I

A.H. IP. NO. 249542, a 70 year old Asian male presented to us with a three months history of jaundice, right hypochondrium pain, pruritis, nausea and vomiting, polydipsia and polyuria. His illness started with a general malaise and lethargy, associated with a persistent slight fever. This progressed to jaundice of increasing intensity and right hypochondrial pain. He denied history of recent injections and he does not drink or smoke. He has been well before this illness and has not been ill before.

On examination he was an obese elderly man, deeply icteric, ill - looking and afebrile. The liver was palpable four finger-breadth's below the coastal margin. It was smooth. The gall-bladder

was distended but not tender. There was no ascites and no other abdominal organs were palpable. Other systems were normal

A diagnostic work up was as follows:-

1. Bilirubin (serum) Total 14.2mg/dl
   Direct 12.2mg/dl
2. Serum Porteins Total 5.7g/dl
3. Serum Alkaline Phosphatese - 540 IU/l
4. Transaminases - SGPT - 32 IU/l
   SGOT - 56 IU/l
5. A normal Full blood picture
6. Urine - Bilirubin + ve urobilinogen -ve
7. Bleeding Indices
   Bleeding time 2' (30")
   Prothrombin time 20" (control 12")
   Partial Prothrombin time 60" (Control 3C")
8. Plain abdominal X-ray was normal Percutaneous trans-hepatic cholangiagram (PTC) was done two week after admission, using a contrast medium "urovision". It showed gross dilatation of intra-hepatic ducts, a large gall bladder, and a very wide common bile duct in which the contrast medium abruptly stopped in the distal portion. These findings are consistent with an extra hepatic billiary abstruction in the distal common bile duct.

After this work-up, the patient was prepared for Laparatomy as follows:-

1. Daily injections of Vitamin K1 2.5mg im for 3 consecutive days prior to operation.
2. Ampicillin 500mg six hourly orally for five days prior to surgery
3. Nasogastric tube and suction of gastric contents in the morning of the operation.
4. Three units of blood were made ready for operation

OPERATION

Anaesthesia was induced with epontol 500mg IV and maintained with halothane/oxygen mixture, and pavulon for relaxation.

With the patient in a supine position, the whole abdomen was cleaned with chlorhexidine solution followed by flavine in spirit. After draping with sterile towels, a right paramedian incision extending above and below the umbilicus was made and the abdomen opened.

Intra-operative findings

A massively distended gallbladder and common bile duct, with a stricture as it enters the retroduodenal space. The head of the pancreas was normal.

The duodenum and the common bile ducts were mobilised. The second part of the duodenum was opened anteriorly and the papilla of Vater inspected and palpated. The stricture had extended to the papilla. The part of the common bile duct proximal to the stricture was transected and anastomosed to the anterior aspect of the duodenum (choledoco-duodenostomy). Haemostasis was achieved and abdomen closed in two layers of non absorbable sutures, after inserting a corrugated rubber drain.

Post-operative course

Patient did not make a slow recovery from the operation. He however died of pneumonia two week after surgery.

CASE II

L.K. IP. NO. 226843 a 55 year old African female, residing at Kimara, Dar es Salaam, presented to the casualty with an acute onset of right hypochondrial pain of one day's duration. The pain was non-colicky, and had been increasing in intensity from onset, until

the time she was seen at the casualty about 7 hours. There was no associated nausea or vomiting, and no fever. She had several similar attacks before which were managed at her local dispensary.

On examination she was an ill looking middle aged lady in severe pain. She was not febrile, jaundiced or dehydrated. She was not obese. The respiratory and cardiovascular systems were normal. There was tenderness in the right hypochondrium with localised guarding in that region. Murphy's sign was positive. The liver, spleen and the kidneys were not palpable. The bowel sounds were present. A plain abdominal X-ray taken at the casualty showed gallstones in the gallbladder.

A diagnosis of acute cholecystitis was entertained. She was managed conservatively by bed rest, intravenous fluid therapy, analgesics and antibiotics (Ampicillin). She was discharged after about seven days in a good condition, free of pain. It was planned that an elective cholecystectomy will be done within three months.

The patient came back after two months for operation. Preliminary investigations included an oral cholecystogram which confirmed gallstones, a full haemogram, urine and stool analysis. The liver functions tests were done and found to be normal.

She was operated on 21.4.81, under a general anaesthetic induced with pentothal 350mg intravenously and maintained by a mixture of halothane, oxygen and nitrous oxide. The abdomen was opened by an upper median incision, from the umbilicus up to the xiphisternum. The gallbladder was tethered to the liver by a few adhesion bands which were divided. The cystic duct and artery were identified dissected and explored up to the junction with the common hepatic duct to form the common bile duct. They were then double ligated with silk - 2 - 0 sutures and transected. The gallbladder was then dissected out of the liver bed to which it was adherent. Loose 2 - 0 catgut sutures were applied to the

bleeding gallbladder site, after it was completely removed. The abdomen was then closed in two layers - prolene 1 - 0 to the rectus sheath and peirtoneum in a single layer of continuous sutures, and silk 2 - 0 to the skin, leaving in a corrugated rubber drain to the removed gall bladder site.

Post operatively she was managed with pethidine intramuscularly for pain. She had a stormy recovery characterised by several episodes of shock and mental confusion. She also had bleeding from the gall-bladder bed, draining to the outside through the corrugated rubber drain and later through a track which has formed after removal of the drain. These episodes were managed conservatively, by fluid and blood transfusion, vitamin K injections and continuous monitoring of blood pressure, pulse rate, fluid input and output. She made a slow but steady recovery and was discharged from hospital after about a month.

She continued to come for follow up at the Surgical clinic for up to one and half years. She was in good health, when last seen.

DISCUSSION

1. Causes of Extrahepatic biliary obstruction in 24 patients

	No. of patients
1. Malignancy	
a) Carcinoma of Pancreas	4
b) Carcinoma of bile duct	2
c) Carcinoma of Gall bladder	1
2. Stones	6
3. Inflammation	
Chronic Pancreatitis	1
Stricture of common bile duct	2
4. Undetermined	8
TOTAL	24

The overall male to female ratio is 3 : 1 but more females had cholelithiasis than males 2 : 1. There were more males presenting with obstructive jaundice due to malignancies.

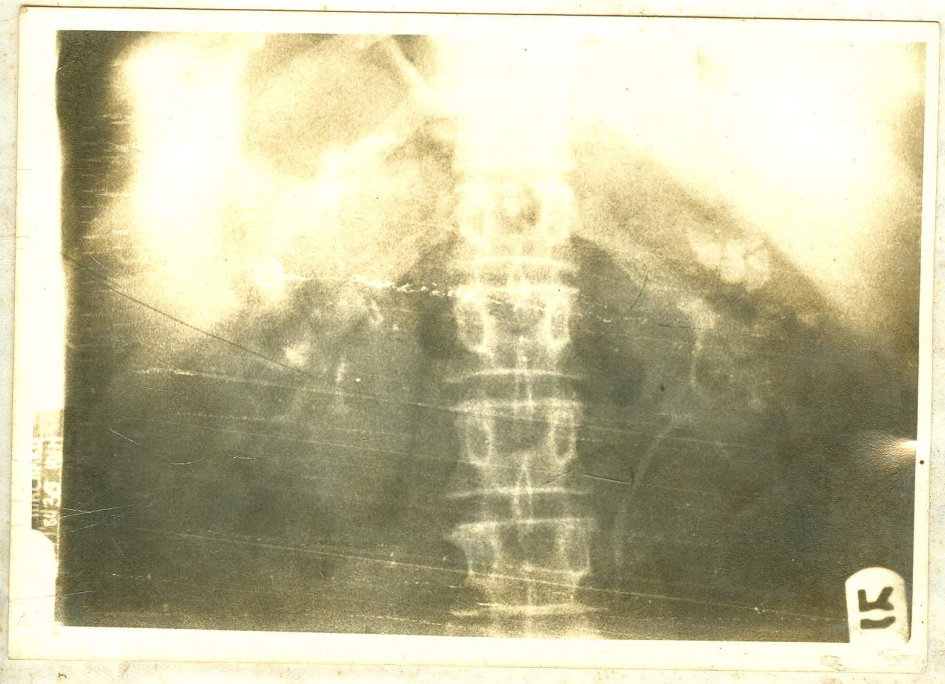
Age incidence - in all the patients in whom the age was recorded, this was greater than 40 years. Similar figures for Nairobi were recorded by Amollo Odhiambo and Kamidigo (1976). In another series from Nairobi, the commonest cause of obstructive jaundice was malignancy (Ochola Abila P. 1981).

2. Treatments carried out in 24 cases.

	No. of patients	Outcome
1. Cholecystectomy	6	All survived.
2. Choledoschoduodenostomy	2	1 survived 1 died.
3. Cholecysto - jejunostomy	4	All died
4. No treatment recorded	12	
TOTAL	24	

The high incidence of malignant disease as a cause of obstructive jaundice in Africans, which is reported to be as high as 71% of all cases of obstructive jaundice in some series (Ochola Abila 1981), makes prognosis a grim one. All the patients who had malignant disease in our series died within six months of operation.

Another feature of obstructive jaundice in Africans, is that a stenosing fibrosis of distal portion of the common bile duct is commonly encountered (Davey W.W. 1968). This may be parasitic in Origin. In areas where ascaris is endemic, patients who undergo biliary surgery, should be given a course of worm expellant as part of pre-operative preparation (Olatoregun and Itayemi 1979). This is



Radio - opaque Gallstone

because operations in the common bile duct and damage to the sphincteric mechanisms predispose to the migrations of ascaris worms into the common bile duct, causing further obstruction to bile flow.

GALLSTONES

a) Aetiology and Pathogenesis

Cholesterol stones are formed when bile which is supersaturated with cholesterol is secreted. A variety of pathophysiologic phenomena increase lithogenicity. These are; dietary factors can lead to an absolute increase in cholesterol secretion in bile; excessive loss of the bile salt pool may decrease the ability of the bile salts to solubilise cholesterol; alteration in entero-hepatic circulations of bile salts may adversely affect solubility by both decreasing bile salt secretion and increasing cholesterol secretion (La Morte W.W. et al 1981).

The main lipids in bile are; cholesterol, bile salts and phospholipids (mainly lecithin). Cholesterol and Lecithin are not soluble in water. However when mixed with bile salts they aggregate to form "micelles" which are soluble in an aqueous environment. The solubility of cholesterol in bile is therefore dependent on the molar concentrations of bile salts, cholesterol and lecithin (Small et al 1980).

Small and associates (1980) were able to demonstrate three stages in the pathogenesis of cholesterol stones.

i) Secretion of supersaturated bile

ii) Precipitation of microcrystals

iii) Growth of macroscopic stone. Secretion of supersaturated bile may be due to a high calorie diet, increased dietary cholesterol or poly - unsaturated fatty acids. In some

individuals, there is an increased activity of hydroxymethyglutaryl-CoA reductase the rate limiting enzyme in cholesterol synthesis. The decrease in bile salts may occur in conditions associated with biliary fistulae so, that ability of the liver to synthesize new bile acids is exceeded by the rate of loss of these acids. Bile is therefore supersaturated with cholesterol, and hence crystalization occurs. Once a nidus of crystalisation forms the growth of the stones becomes phenomenal. Cholesterol stones are not radio opaque.

Pigment stones form as a result of prolonged excessive secretion of certain bile constituents e.g. in chronic haemolytic anaemias, excessive bile pigment, crystallize out if they reach saturation point.

Biliary stasis, causes prolonged storage of bile. This leads to hyperconcentration due to the ability of the gallbladder mucosa to absorb water, and this causes chemical irritation of the mucosa, which predispose to stone formation. Stasis also leads to biliary dyskinesis and dysfuction of the sphincter of Oddi.

Bacterial infection of the gall bladder predispose to stone formation. E coli produces an enzyme B glucuronidase which deconjugates bile. The deconjugated bile combines with calcium to form calcium bilirubinate. The clumps of bacteria and desquamated cells act as nidi upon which crystallisation occurs. Other factors of importance in the formation of biliary stones disease are:-

i) Low inhibitor concentration of bile

ii) Damage to biliary epithelium

iii) Hormonal influence

Sequalae of billiary stone disease:

(i) Acute cholecystitis

This occurs as a result of impaction of a stone in the neck of gall bladder, so that water is reabsorbed and concentrated

is stored. This causes mucosal irritation and if bacterial infection supervences, the patient develops acute cholecystitis.

ii) Mucocoele of the gall bladder occurs when there is an impaction of a stone in the gall bladder neck while the gall bladder is empty. Mucus accummulates in the gall bladder.

iii) Empyema of the gall bladder, with or without perforation - follows bacterial cholecystitis. If pus is not drained, perforation occurs which may lead to bile peritonitis or to a cholecysto - enterostomy.

iv) Obstructive jaundice with biliary Colicus, are caused by impactio of stones in the common bile duct. The characteric signs are fluctuating jaundice and recurrent bouts of pain and fever.

v) Acute pancreatitis:- occurs if stone is impacted at the ampulla of Vater. The pancreatic secretions and bile are incubated together. Deconjugation of bile occurs, with conversion of lecithin to isolecithin by pancreatic phospholipase A. A deconjugated bile and lysolecithin cause destruction of pancreatic tissue. Impacted stones are however found in only 5% of autopsies of patients who die of acute pancreatitis (de Jode 1977).

vi) Carcinoma of the gall bladder; about 6% of patients with gall bladder stones, develop carcinoma of the gall bladder. (Rain et al 1960). Prolonged mechanical irritation of the gall bladder mucosa may be the initiating factor in neoplastic transformation.

vii) Secondary billiary cirrhosis follows long standing biliary obstruction associated with spreading cholangitis. There is necrosis of liver cells around the portal tracts, which heal by fibrosis.

Clinical features of biliary stone disease:

The clinical picture depends upon the size and site of stones. The most commonly involved sites are gall bladder, cystic duct and

common bile duct.

a) Gall bladder stones; up to 15% of gall bladder stones are dasymptomatic (Bailey and Love 1971; Schwartz 1974). The most common presentation however is upper abdominal discomfort, aggravated by fatty meals. This is because fatty meals stimulates secretion of the enzyme pancreozymin - cholecystokinin which causes contraction of the gall bladder.

b) Cystic duct stones: These cause symptoms if stones are impacted. There is biliary colic which starts from the epigastrium of left hypochondrium and radiates to the back. The pain is due to the contraction of the duct muscle around the stones. Distension of the gall bladder may contribute to the pain. If acute cholecystitis develops, the right upper quadrant pain becomes severe and continuous rather than colicky. The temperature and pulse may rise.

c) Common bile duct stones - also cause biliary colicky pain due to the contraction of muscular part of the duct over the stone. This appears before jaundice in 80% of the cases (Cole 1978). Jaundice occurs if there is complete blockage of the common bile duct; characteristically, it is an intermittent type of jaundice.

Fever develops, if there is secondary bacterial infection causing a spreading cholangitis. Itching is a common symptom in obstructive jaundice. It is due to the deposition of bile salts in the skin. It does not occur in haemolytic jaundice.

Investigations in obstructive jaundice

The aim of investigations in obstructive jaundice is two fold. First to ascertain the presence of obstructive jaundice and second to find the cause. To this end, a work up of the patient presenting with jaundice, would be as follows:-

i) Liver function tests will show a high conjugated serum bilirubin and a very high serum alkaline phosphate level.

ii) Urine bilirubin is strongly positive while urobilinogen is negative.

iii) Straight upper abdominal X-ray is mandatory as about 10% of biliary stones are radio - opaque (Rain et al 1960).

iv) Oral cholecystography shows filling defects. Cystic ducts stones with impaction will cause non - visualisation of the gall bladder. It is a useful procedure if total bilirubin is not greater than 2mg% (Schwartz 1974).

v) Intravenous cholangiography. This delineates the extrahepatic biliary system. It is of no use if total bilirubin is greater than 3.5mg%.

vi) Percutaneous Transhepatic cholangiography (PTC) is used in deeply jaundice patients, to differentiate between intrahepatic and extrahepatic biliary obstruction. The bleeding indices must be normal. Potential complications are biliary peritonitis and prolonged bleeding. Image amplification and television monitoring facilitate this study.

vii) Retrograde endoscopic transpapillary cholangiography is an investigation where gastroduodenoscopy is done using a fibre-optic endoscope, the ampulla of Vater is identified and cannulated. The contrast medium is then injected and radiographs taken. It is useful in the investigation of the extrathepatic bile duct systems.

viii) Cholangiomanometry

Bile duct pressure can be gauged either by using fibre - optic endoscopy and cannulation or intra operatively. Normal bile duct pressures range from 15 - 30cm of saline.

ix) Barium meal and Barium enema studies

These radiological investigations help to rule out primary carcinoma from the stomach, head of pancrease and colon, sending a secondaries to the liver causing obstructive jaundice (Berk 1973).

x) Intra operative and post operative T-tube cholangiography

This confirms patency of the bile duct after extraction of stone and before T-tube is removed.

TREATMENT OF OBSTRUCTIVE JAUNDICE AND BILIARY STONE DISEASE

To prevent complications arising from obstruction to bile flow, stated above, it is necessary to decompress, the biliary system before these complications arise. The choice of therapy, and aim of treatment depends upon the cause of obstruction. This has been well illutrated in the two cases described. In the first case decompression was achieved by transecting the common bile duct proximal to the stricture, and anastomosing the proximal stoma to the second part of the duodenum. In the second case, the acute phase of the disease was managed by bed rest, antibiotics, intravenous fluids and naso-gastric suction, followed by an elective cholecystectomy.

In biliary stone disease, due to cholesterol stones there is a choice of treatment between surgical and medical treatment; both aim at relieving symptoms and preventing recurrence.

i) Medical Therapy. Two drugs are currently in use, Chenodeoxycholic acid and ursodeoxycholic acid. (Somerville K.W. et al 1982). These drugs act by increasing the solubility of cholesterol in bile, so that cholesterol stones exposed to such bile gradually get smaller and may even dissolve completely. Treatment has to be continued indefinitely.

Oral cholecystography repeated at six month intervals is the standard method for determining reduction in size (partial success) and complete dissolution of stones (completel success). Recently ultrasonography has been found to be even more sensitive than oral cholecystograms in detecting very small stones and hence delineating patients for post disolution maintenance treatment. Somerville and associates (1982) recommend that complete dissolution of gallstones, should be

reported only if both oral cholecystography and ultrasonographic studies give negative results and progress of patients receiving post dissolution maintenance treatment, be monitored by ultrasonography rather than serial cholecystograms.

ii) Surgical Treatment

Cholecystectomy remains the treatment of choice for cholelithiasis, and for both acute and chronic cholecystitis. The extrahepatic biliary system has many anatomical variations which a surgeon must be aware of, in order to avoid serious errors of technique and injury to other important adjacent structures. Normal anatomical variations of the extra hepatic biliary system are given diagrammatically.

The operative technique is as reported in our second patient. when the aetiology of obstructive jaundice is malignancy of either head of pancreas (commonest in our environment), liver, stomach or bile duct, then one has to be content with palliation only. Decompression is the achieved by a variety of surgical procedures. The commonest and the easiest is cholecystojejunostomy. This was done in four of our patients, and in all the jaundice disappeared, but they all died of cancer.

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4. PERFORATED DUODENAL ULCER

SUMMARY

Twelve patients were operated for perforated duodenal ulcer in surgical firm I, one of three general surgical firms at Muhimbili Medical Centre. The majority were managed by laparatomy and simple closure; some who had chronic symptoms were given appointments for elective definitive surgery after surgery for the acute stage. In a third of these patients, this simple closure proved to have been an adequate treatment as these patients had no symptoms of peptic ulcer at follow up.

Perforated duodenal ulcer was the commonest complication of peptic ulcer necessitating operative intervention in this firm. Out of eighteen operations done for peptic ulcer disease over a two year period twelve (67%) were due to perforation. Other complications of peptic ulcer disease for which surgery was indicated in order of frequency were bleeding (3), intractability (2) and pyloric obstruction (1). This however does not include patients who were admitted and treated medically for acute exacerbation of peptic ulcer disease, symptoms.

One of the patients, presenting with a perforated duodenal ulcer, operated by the author is described in detail, and the subject of peptic ulcer disease is reviewed; as it pertains to aetiology, pathogenesis, clinical presentation, complications and treatment modalities.

INTRODUCTION

Peptic ulcer disease is a common condition in Africans, contrary to views held by earlier workers (Baimbridge 1934, Connel 1937, Trowell, 1960). In a series of 733 barium meal studies, at Muhimbili

hospital, Dar es Salaam, Grech (1965) reported a 26.5% peptic ulcer rate. In another review of surgical admissions to the same hospital in 1971, Hiza (1974) noted 40 admissions out of a total 1871 surgical admissions, 6 of which were due to perforation. If all the admissions to surgical and medical wards, and also patients reviewed at outpatient clinics were taken into account the figure would undoubtedly be much higher. In all reports, there is preponderance of urban over rural populations (Konstam 1954, Shephered et al. 1968).

Incidence of perforation vary from country to country, and it tends to increase during periods of traumatic and stressful situations. Thus during the Battle of Britain of World War II in 1940/1941, there was a marked increase in peptic ulcer perforation due to bombing raids by the German air force.

Perforations are more common in males than females in most series. In the patients reported only one, out of the twelve treated for perforated duodenal ulcer was a female. There are more duodenal ulcer perforations than gastric ulcer perforations in most series (Jamieson 1955, Webster et al 1958, Schwartz 1972).

Patients receiving steroids, aspirin, phenylbutazone, indoles and who excessively consume alcohol are candidates for peptic ulcer disease and perforation. (Du Plessies 1956, Davenport 1969). These substances cause ulceration by inducing alteration in the volume and composition of mucus secreted by gastric and duodenal mucosa. The mucosal barrier to acid pepsin digestion is thus disturbed, and ulcers form. Four of the patients in our series had a positive history of ingestion of aspirin.

CASE PRESENTATION

J.H. IP. NO. 280209 was a 45 year old male lorry driver, from Mtoni in Dar es Salaam who presented with an acute onset of upper abdominal pain at 4.00 p.m. in the afternoon associated with vomiting coffee - ground material. He has previously been well before, except for avague upper abdominal discomfort for 3 years, for which he has not sought any treatment. The acute pain was continuous and was not relieved by anything.

On examination he was an ill-looking middle aged man, in intense pain, was sweating but was not dehydrated nor was he febrile. The pulse was 90 per minute, regular and good volume. Blood pressure was 110/70. The respiratory and cardiovascular systems were essentially normal. The abdomen was rigid, tense and tender all over. Bowel sounds were absent. A chest X-ray in the erect position showed gas in the cupola of the right hemidiaphram.

A diagnosis of perforated peptic ulcer with peritonitis was arrived at. A Nasogastric tube was inserted and suction started. Infusion of normal saline 2 litres was started and blood taken for cross-matching and grouping. The patient was taken to theatre after the preparations for surgery were ready.

Details of the operation

The patient was put in a supine position and general anesthesia was used, with intubation.

The abdomen was opened by an upper median incision extending from the ♦ xiphisternum to the umbilicus. A perforated duodenal ulcer in the first part of the duodenum anteriorly, measuring 2 x 1cm was found. The edges of the ulcer were freshened and interrupted 2 - 0 silk sutures applied to obliterate the ulcer. This was effected with ease. Vagotomy and drainage procedure were not done due to the presence of a severe peritonitis.

Peritoneal toilet was done, using warm saline and suction. The abdomen was then closed in two layers leaving in a corrugated rubber drain. The peritoneum and rectus sheath were closed in a single layer with continuous 1-0 prolene and silk 2 - 0 interrupted sutures to skin.

The post-operative period was not remarkable and the patient was discharged after 7 days from hospital.

DISCUSSION

SUMMARY OT 18 CASES OF DUODENAL ULCER OPERATED IN FIRM I

AUGUST 1980 - FEBRUARY 1982

Complication	M	F	Total	%
1. Perforation	11	1	12	66.7
2. Bleeding	3	0	3	16.6
3. Gastric outlet Obstruction	2	0	2	11.1
4. Intractability	1		1	5.6
Total	17	1	18	100

2. Perforated Duodenal ulcer in relation to age group. 12 cases.

Age group (Year)	Total No. of Cases	%
10 - 19	3	
20 - 29	3	25%
30 - 39	5	41.7%
40 - 49	2	16.6%
50 - 59	1	8.3%
Over 60	1	8.3%
	12	100%

3. Approximate time of perforation, as recorded among the cases studied (12).

Time Range	Number of cases	%
12.00 midnight - 12.00 noon	0	
12.00 noon - 6.00 p.m.	8	66.7%
6 p.m. - 12 midnight	4	33.3%
	12	100%

4. Type of surgery offered for perforated duodenal ulcer in the 12 cases studied.

Type of Surgery	No. of patients	%	Remarks
1. Simple closure	4	33.3	1 died due to peritonitis.
2. Simple closure plus omental patch.	7	58.4	All survived had subsequent definitive surgery survived.
3. Definitive surgery - Vagotomy and Gastrojejunostomy.	1	8.3	Survived
4. Drainage alone	-	-	-
	12	100	-

5. Duration of Hospital stay and mortality for the duodenal ulcer patients studied 12 cases.

Duration	No.	%
10 days	8	66.7
10 - 14 days	3	25%
14 days	-	
Deaths	1	8.3%
	12	100

6. Symptoms, signs and investigations among the 12 patients with perforated duodenal ulcer treated in firm I.

Symptoms/Signs	No. of patients	%
1. Sudden onset of epigastric pain	12	100%
2. A history of previous dyspepsia for 3 months or more.	8	66.7%
3. No history of previous dypepsia	4	33.3%
4. History of ingestion of Apirin phenyl-butazone or alcohol.	4	33.3%
5. Diagnosis from history and gas under diaphragm on X-ray.	9	75%
6. Diagnosis of other acute abdominal conditions made.	3	25%

Aetiology and Pathogenesis of Peptic Ulcer disease:

Peptic ulcer is an ulceration of the gastro-intestinal tract located in the mucosa distal to acid secreting epithelium. It

may be found in the lower oesophagus, stomach, duodenum, jejunum at the margin of a gastrojejunostomy, and in Meckel's diverticulum which frequently contains aberrant gastric mucosa. By far the commonest types are gastric and duodenal ulcers. Although both are considered to be due to acid - pepsin digestion, there are important differences between the two types of peptic ulcers. (Menguy N. 1972). These are:-

a) Sex; both types of ulcer are more common in males than females but the ratios are 2:1 gastric ulcers and 7:1 duodenal ulcers. ~~Thus.~~ Thus comparatively women tend to have more gastric ulcers.

b) Age; Duodenal ulcer is a disease of young men, aged 25 - 45, whereas gastric ulcer incidence tends to increase with age.

c) Familial tendency; Both types of ulcer tend to occur in certain families, but in duodenal ulcer there is a prepo-nderance of subjects with blood group O, whereas, in patients with gastric ulcer, blood group A predominates.

d) Relationship to environmental factors:

Individuals with duodenal ulcer often work in conditions of tension, with increased social responsibility. It tends to occur more often in people of higher social strata e.g. executives, businessman, than gastric ulcer which is more common in poorer social classes in Europe and America.

e) Gastric secretory activity. Duodenal ulcer patients have on the average of higher rate of gastric secretion than the normal population. Individuals with gastric ulcer, have normal or lower levels of hydrochloric acid secretion compared to ulcer free individuals, except in patients with combined gastric and duodenal ulceration.

f) Response to treatment; In general, duodenal ulcer responds favourably to medical treatment, whereas, gastric ulcer does not. Gastric ulcer was once thought to turn malignant in a proportion of cases, but this view is no longer held. (Schwartz 1972). It is now accepted that benign gastric ulcer remain benign and malignant ulcers start de novo.

Several factors have been associated with development of peptic ulceration, and one or more of them may be acting in any individual case. These are:-

i) Impaired blood supply; ischaemia of the gastric and duodenal mucosa.

ii) Impaird mucosa barrier; Gastric and duodenal mucosa contain mucus with physical and chemical properties suited to a protective function. It is a viscous gel which completely covers the mucosa; it is spongelike, hydrophilic and it may act like an ion exchange resin (Avery Jones 1977). It is secreted by Goblet cells, and chondroitin sulphate in gastric mucosa has been identified with evolutionary development of secretion of hydrochloric acid to aid digestion. Mucus barrier efficiency is reduced by a variety of factors notably alcohol, salicylates and spices.

iii) Altered cell kinetics; the mucosal cells in the gastro intestinal tract degenerate fast and are replaced every 48 - 72 hours. Factors which reduce or increase this activity lead to peptic ulceration. The most notable of these is stress. Stress causes release of acetylcholine which further stimulates release of histamine and heparin from mast cells. These are vasoactive substances which produce a local inflammatory reaction with mucosal oedema.

Heparin also reduces mitotic activity with reduction in the secretion in the secretion of mucopolysaccharide. Stress also increases release of adrenaline and glucocorticoids which stimulate chalone activity, that is decreased mitotic activity (Avery Jones Sir F. 1977). Chalones are biological substances found especially in epidermal and mucosal cells with "anti-mitotic" activity.

iv) Increased ulcerogenic factors.

i) Increased acid secretion; this may be due to:-

a) Increased neural stimulation; vagal stimulation increases secretion of hydrochloric acid and also renders parietal cells more reactive to other stimuli. (Schwartz 1974). This is the reason why vagotomy reduces or completely abolishes gastric hypersecretion.

b) Excessive capacity to produce acid. Patients with duodenal ulcer have more parietal cells than normal individuals. This may be a genetically determined factor or a result of work hyperplasia due to an unknown stimulis. (Schwartz 1974). The effect of this, is increased secretion of hydrochloric acid. Since males generally have more parietal cells than females, this explains the disparity in the incidence of duodenal ~~an~~ ulcer in the two sexes.

ii) Pepsin; As far as ulcerogenic activity is concerned, gastric hydrochloric acid and pepsin act in concert. Tissue digestion is due to proteolytic activity of pepsin which is only possible at a pH lower than 4. Therefore when the amount of H^+ secreted exceeds the buffering capacity of duodenal secretion, the pH of the gastric outlet zone and first part of the duodenum

remains within the range of pepsin activity for a long period.

iii) Genetic factors in peptic ulcer disease. More individuals with blood group O are found among patients with duodenal ulcer, than normal individuals, and also inability to secrete blood group antigens A, B, and H in gastric juice and saliva occurs in about 25% of the population is associated with duodenal ulcer. The patient who is blood group O who is also a non secretor of these blood group antigens in his saliva and gastric juice has a 2.5 times greater chance of developing duodenal ulcer than an individual with blood group A or B.

iv) Ulcerogenic tumors of the Pancreas: A causal relationship between non-insulin producing islet cell tumors of the pancreas and peptic ulcer disease was first reported by Zollinger and Ellison in 1955. This was later named the Zollinger - Ellison Syndrome and consists of a triad of:-

(a) Marked gastric hypersecretion

(b) Recurrent ulceration despite intensive medical or surgical therapy.

(c) Presence of a non beta islet cell tumor of the pancreas

The potent hormone secreted by these tumors was identified as gastrin by Gregory and Tracy (1960). Multiple endocrine adenomatosis is also associated with peptic ulceration and the Zollinger Ellison Syndrome. The only possible treatment for this condition is a total gastrectomy or Total/Partial Pancreatectomy.

Pathology and clinical features:

Acute perforation may occur in acute or chronic duodenal ulcers. The base of the ulcer suddenly ruptures with the result that duodenal content escape into the general peritoneal cavity. This initiates a train of events which if not intervened may lead to a general bacterial peritonitis. Infrequently, if the duodenum is relatively empty the perforation may become sealed off and the

peritoneum may be able to deal with the contamination, or the perforation occurs into the lesser sac, resulting in a localised peritonitis, sub-phrenic abscess and abscesses in the paracolic gutter or hepatorenal pouch of Rutherford Morrison.

The size of the perforation varies from 3mm to over 1cm in diameter, and the larger the perforation, the higher the morbidity and mortality. Depending on the extend of peritoneal soiling perforated duodenal ulcer may be divided into 3 types:-

(a) Acute perforation; the ulcer perforates and the general peritoneal cavity becomes flooded with duodenal and gastric contents. In up to 30% of these patients there is no antecedent history of chronic peptic ulcer symptoms. (Desmond et al. 1969). From the moment of perforation the clinical course falls into three stages

i) Primary stage or stage of peritoneal irritation follows immediately upon perforation. It's symptoms are due to intense irritation of the peritoneum by duodenal contents which consists of hydrochloric acid and proteolytic enzymes. This sudden and violent irritation of the peritoneum produces a reflex action which shock, intense abdominal pain which may be referred to the back or shoulders, and retching, although actual nausea and vomiting is rare.

The patient lies with his legs drawn up and flexed; is afraid to move in fear of aggravating the pain. He is pale, anxious and sweating, with cold and moist extremities. The temperature may be normal, but the respiratory rate is always increased and it is shallow and thoracic in nature due to the immobility of the diaphragm.

The abdomen is tense and rigid, with marked tenderness. There is also a rebound tenderness; on auscultation, and the bowel sounds are absent.

ii) Secondary stage or stage of peritoneal reaction this follows 3 - 6 hours after perforation depending on the size and site of the perforation. There is profuse peritoneal fluid secretion which tends to dumpen the irritation with the result that pain becomes less, but all the other features described above remain unchanged.

An upper abdominal X-ray, including the lower part of the chest, taken in the erect position demonstrates a pneumoperi-toneum in almost all cases. This is pathognomomic perforated peptic ulcer, and the air collects under one or both cupola or the diaphragm. (Kozoll and Mayer 1962).

iii) Tertiary stage is the stage of bacterial peritonitis, and it becomes increasingly likely after 12 or more hours of perforation. The clinical features are essentially those of a general peritonitis from any other cause. There is less pain and profuse vomiting. The patient is severely dehydrated with marked electrolyte depletion, as a result of vomiting and marked outpouring of fluid into both the general peritoneal cavity and the distended paralytic intestines. Fever also occurs in this stage.

The abdomen is distended, tender and there is guarding of the abdominal muscles. The abdomen is silent on auscultation. If no treatment is instituted, the terminal phase is characterised by a complex clinical picture of toxaemia, dehydration, shock, circulatory failure and finally death.

(b) Sub-acute perforation; in these cases, perforation occurs, but is immediately sealed off, before there is spillage of duodenal contents into the general peritoneal cavity. The clinical picture is that of a sudden acute upper abdominal pain which may radiate to the back or scapula. There is local tenderness in the right upper quadrant of the abdomen, but the rest of the abdomen is soft and non tender. An upper abdominal X-ray

may reveal gas under the diaphragm.

(c) Chronic perforation; this occurs when an ulcer perforates into an area sealed off by adhesions or adjacent viscera, resulting in a chronic abscess. These abscesses may be in the subphrenic spaces, Rutherford Morrison pouch and in the omental bursa. Diagnosis is usually made at laparotomy to drain the abscess.

TREATMENT OF PERFORATED DUODENAL ULCER

The treatment of perforated duodenal ulcer is operative. Non operative treatment is adopted only for those patients considered not fit to withstand an abdominal operation. Some time should be spent in resuscitating the patient prior to the operation. An appropriate pre-operative regimen would be:-

i) Nasogastric intubation and suction with no fluids per mouth

ii) Intravenous fluids, starting with normal saline, while blood is taken for grouping and cross-matching, blood counts, electrolytes and serum amylase.

iii) Pain is relieved by a strong analgesic intramuscularly or intravenously provided the diagnosis is not in doubt.

iv) Blood pressure and pulse rate chart commenced

Surgery:

Simple closure of the perforation is the treatment of choice, but in selected cases a more radical treatment such as vagatomy and drainage procedure may be undertaken without increasing morbidity and mortality.

Simple closure:

Can be effected by placing interrupted sutures across the perforation, using chronic catgut or silk sutures (Graham R. 1937). This is a simple and effective procedure which does not require experience or skill in abdominal surgery. It's aim is to remove the immediate danger of death in a seriously ill patient and not to cure the ulcer. Mortality from this procedure varies from 4-12%. In our series one, out of the twelve patients (8.3%) died.

A general anaesthetic with muscle relxation is prefered.

incision as it is quicker and easier to close in potentially infected area. In most cases, the perforation is easily seen; it may be oval or circular and of variable diameter. The escaping fluid is bile stained. The perforation is best closed by placing interrupted sutures of number 0 - 0 chromic catgut or silk and tying them gently in place. An omental patch can be laid over the perforation. Peritoneal toilet should follow this procedure. All the fluid is sucked out from the general peritoneal cavity, and a specimen taken for bacteriological study. A drain is left in situ, in the ulcer site and through a separate stab wound.

Simple closure of the perforation in the presence of severe duodenal stenosis would aggravate the stenosis. Thus a gastro-jejunostomy should be done, either alone or in combination with a vagotomy.

Emergency radical treatment for perforated ulcer; several series have been reported more extensive procedures being performed, with the aim of curing the ulcer, in which both mortality and morbidity are not higher than in simple closure of the perforation (Siegrist 1957; Nuboer 1951; Lowden 1952; Debakey M. 1963). There is no doubt that, these curative operations can be safe in capable hands, with the provisos that:-

1. Adequate facilities must be available
2. The surgeon must be experienced in these procedures
3. The patient must be in a reasonably good condition to withstand an extensive operation.

The operations commonly done are:-

i) Vagotomy and drainage

ii) Parital gastrectomy. Vagatomy and drainage procedure is a procedure of choice for duodenal ulcer. It is done as in elective procedures, except that it must be accompanied by a meticulous peritoneal toilet in a perforated duodenal ulcer.

The method of drainage depends on the preference of the surgeon, but when there is gross induration and oedema of the duodenum a gastro-jejunostomy would be less risky than a pyloroplasty.

Post operative complications:

1. Peritonitis is the commonest cause of mortality in perforated peptic ulcer.
2. Subphrenic and pelvic abscess. These are less common when a radical operation is performed than in simple closure of the perforation.
3. Duodenal fistula
4. Haemorrhage is common after simple closure
5. Wound infection rate is high especially in patients who present late for treatment.

The post operative care after simple closure of the perforation should be as follows:-

1. Nasogastric intubation and suction continuously until bowel sounds return.
2. The patient is nursed in a sitting position, must have intravenous fluids as dictated by the degree of dehydration and daily serum electrolytes estimations.
3. An accurate fluid balance chart is kept
4. Antibiotics are given if the results of cultures taken at operation, indicate them, or if there is a chest infection.
5. In elderly patients regular breathing exercises to promote chest expansion and bronchial drainage under the supervision of a physiotherapist is recommended.

Prognosis: in a perforated duodenal ulcer depends on:-

(a) Size of the perforation; the larger the perforation, the greater the extent of peritoneal soiling and the worse the prognosis.

(b) Chronicity of the ulcer, the more chronic the ulcer the worse the prognosis.

(c) The age of the patient; elderly patients are less able to stand the stress of the perforation.

(d) Time factor; the time which elapses between perforation and treatment is an important factor in prognosis. The longer the interval between rupture and treatment, the worse the prognosis.

(e) Associated haemorrhage:- when a perforated duodenal ulcer is associated with haemorrhage, the prognosis becomes wores.

Conservative treatment of perforated duodenal ulcer

Indications for this form of treatment are few. It consists of patients who are poor operative risks, in aged patients and in whom diagnosis is not certain.

It consists of continuous effective gastric aspiration, antibiotics, adequate intravenous fluid administration and suction. No fluids per mouth are permitted for at least 48 hours.

This form of treatment has few adherents for the following reasons.

i) A perforated gastric carcinoma may be mistakenly treated by this method.

ii) A gastric ulcer may have perforated, and this responds poorly to non surgical treatment.

However its adherents like Taylor H. (1957) report a mortality rate of 9.6% which is comparable to that of simple closure of a perforated duodenal ulcer. This method however is not used in our centre.

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5. ACUTE APPENDICITIS

SUMMARY

From July 1980 to April, 1982, 32 cases of appendicitis were operated in surgical firm I, one of the three general surgical firms in the Muhimbili Medical Centre, Dar es Salaam. Acute appendicitis was the second commonest surgical emergency encountered in this firm, obstructed inguinal hernia being the first. One of these patients is reported in detail. He was a 20 year old male, with generalised peritonitis due to a perforated gangrenous appendix. A right lower paramedian incision was made, appendicectomy done followed by peritoneal toilet with warm saline and suction. Post operatively he was treated with large doses of parenteral antibiotics, gastric suction and intravenous fluids. He recovered and was well at follow up, for up to six months. He was not seen after this period. Wound sepsis was the commonest complication observed.

The aetiology, pathogenesis, clinical features and treatment are discussed.

INTRODUCTION

Acute appendicitis as a clinical entity was first reported in the first part of the 19th century. The first appendicectomy was performed by Fitz in 1886. From then onwards, the incidence of this condition has continued to rise, first in Europe and North America and later, in the less developed countries of Asia and Africa.

In Europe and America, it is the commonest abdominal surgical emergency (Burkitt 1971). Earlier workers in Africa, considered acute appendicitis to be rare, (Miller 1955) but it is now on the increase (Burkitt 1971), and in Dar es Salaam Hiza (1974) and Shija (1975)

convincingly demonstrated this to be a common surgical problem, especially in urban areas.

The disease commonly affects older children and young adults. Twenty six of the patients peesented were under 30 years, and none was above 40 years.

CASE REPORT

Y.V. IP. NO. 234414 was a 20 year old male, a Dar es Salaam resident who presented to us with a 3 days history of abdominal pain, fever and vomiting. The abdominal pain first started in the umbilical region, then settled in the right iliac fossa. There was accompanying anorexia, together with nausea. Initially there was no vomiting. Later the pain spread to the whole abdomen, and he started vomiting. The pain was constant and non radiating. He vomited four times on the day of admission.

On examination he was an ill looking young man, febrile and midly dehydrated. The pulse rate was 120 per minute, regular and adequate in volume. Blood pressure was normal. The abdomen was tender with severe guarding. There was rebound tenderness. On auscultation there were no bowel sounds, heard. A rectal examination was normal.

A provisional diagnosis of general peritonitis, due to a perforated appendix was made, and the patient was prepared for operation as follows. An intravenous line was started and normal saline was given. Two litres were run in before operation. A naso-gastric tube was inserted and gastric suction was started. The patient was shaved and was taken to theatre after about three hours.

A general anaesthetic with intubation was used. A lower right paramedian incision was made and abdomen opened. There was gene-

ralised peritonitis with a gangrenous perforated appendix. The appendix was divided at the base between two chromic catgut 2 - 0 ligatures placed half a centimetre apart and was removed. The appendicular mesentery was also gangrenous and it was removed, it's base clamped and tied with chromic 2 - 0 catgut. The stump of the appendix was not buried because the wall of the caecum was oedematous and inflammed.

The general peritoneal cavity was swabbed with gauze packs and fluid sucked out. It was then lavaged with warm saline and was sucked out. Crystalline penicillin powder was put in and a drain placed in the appendix site, and out through a separate stab wound. The abdomen was then closed in layers, chromic catgut 2 - 0 to the peritoneum and prolene 1 - 0 to the rectus sheath. The skin was closed with prolene 2 - 0 leaving a corrugated rubber drain to the peritoneal cavity. The patient was put on intravenous fluids, gastric suction and antibiotics.

COMPLICATIONS

The patients temperature continued to rise to 40°C, and pus discharged from the wound by the third day. Some stitches were removed and pus swab taken for culture and sensitivity testing. E. coli sensitive to gentamycin, cotrimoxazole and ampicillin were grown. The patient was put on gentamycin 80mg twice daily and wound dressed with eusol twice daily. The temperature dropped and wound healed and the patient was discharged after 15 days in hospital.

He was followed up at the outpatient clinic for six months and was finally discharged from follow up in a good condition.

The results of the 32 patients treated in surgical firm I between July 1980 and April 1982 are tabulated and summarised below ~~on the following page.~~

1. AGE DISTRIBUTION AMONG PATIENTS STUDIED

AGE IN YEARS	NO. OF PATIENTS
11 - 15	2
16 - 20	8
21 - 25	11
26 - 30	5
31 - 35	4
36 - 40	2
Above 40	-
TOTAL	32

2. PATHOLOGY AND OPERATIVE FINDINGS AMONG THE CASES STUDIED

	PATHOLOGY	NO. OF PATIENTS
1.	Normal Appendix (Elective appendicectomy)	0
2.	Chronic Appendicitis (Elective Appendicectomy)	5
3.	Acute Appendicitis	18
4.	Perforated Appendix and Pelvic Peritonitis.	3
5.	Gangrenous Appendix, Adhesions and mass	1
6.	Gangrenous perforated appendix and appendicular abscess.	3
7.	Gangrenous perforated appendix and generalised peritonitis.	2

3. COMMON SYMPTOMS AMONG THE PATIENTS STUDIED

	SYMPTOMS	NO. OF PATIENTS
1.	Classical shifting abdominal pain	5
2.	Pain starting in the right iliac fossa.	27
3.	Anorexia	32
4.	Nausea and Vomiting	25
5.	Fever	25

4. INCISIONS MADE

	INCISIONS	NO. OF PATIENTS
1.	McBurney Gridiron incision	26
2.	Rutherford-Morrison incision	2
3.	Right lower paramedian	4
		32

5. POST OPERATIVE COMPLICATIONS

	COMPLICATIONS	NO. OF PATIENTS
1.	Wound infections	23
2.	Pelvic abscess	2
3.	Septicaemia	3
4.	No complications	4
		32

DISCUSSION

Aetiology:

The following have been considered as possible aetiological factors in acute appendicitis.

(a) Racial and dietary factors; Rendel Short (1920) noted an increased incidence of acute appendicities from the latter quarter of the 19th century in Europe and North America following increased consumption of white polished wheat flour, as a result of improved milling facilities. Burkitt (1971, 1972,

1973) noted a similar rise in incidence in urban African population, and postulated that this corresponded with increased consumption of a low fibre diet similar to that consummed in Europe.

Dietary, rather than racial factors, seem to be the major causative factor, as incidence in Black Americans, does not differ from that in whites living under similar conditions. Walker and Burkitt (1971) observed differences in intestinal transit times of food among individuals who live on a high residue diet, and those who live on a low residue diet. There was a residue marked difference between the two a very short transit time for those who consume a high residue diet, and a long transit time for those who consume a low residue diet. Altered bowel behaviour, would lead to altered bacterial flora of the large bowel and hence possible acute appendicitis. Also a long transit time would predispose to increased absorption of water, resulting in hardened faeces, which might cause impaction in the appendicular secretions and increased bacterial growth thus leading to acute appendicitis.

(b) Viral infections. Particles of coxsackie B viruses and adenovirus has been found in specimens taken from patients undergoing surgery for acute appendicitis. Similarly high titers of the same virus antibodies in some patients with acute appendicitis (Tobe 1965) have been demonstrated.

(c) Lumen obstruction - obstruction of the proximal part of the appendicular lumen is found in the majority of patients presenting with acute appendicitis. This may be caused by a faecolith, fibrous bands, parasites, carcinoid tumor and carcinoma of appendix (Fitz 1886; Wilks 1914, Shija 1975).

Shija (1975) reported 17 cases of appendicular lumen obstruction by schistosoma mansoni granuloma, a fact frequently observed by the author while working in Lake Victoria islands of Ukerewe, a district where this infestation is holoendemic. In areas where ascaris Lumbricoides is common this may cause lumen obstruction.

(d) Bacterial infection:

This commonly follows obstruction to the appendicular lumen. There is stasis of appendicular secretions and proliferation of normal bacterial flora to the extend that they becomen invasive. The common organisms involved are gram negative enteric bacteria, staphyloccus aureus, streptococcus faecalis and anaerobic organisms e.g. bacterioides.

PATHOGENESIS

Proximal lumen obstruction leads to the accummulation of secretions with consequent distension of its lumen. Normal resident bacteria proliferated to the extent that they become invasive. As a result of distension of the lumen venous and capillary drainage diminish and may even cease. Vascular congestion continues to increase as arterial flow continues and acute inflammation sets in. It extends to involve the muscular layers, the serosa and even the adjacent parietal peritoneum.

When blood supply is compromised, there is sloughing of the mucosa, which may extend to the muscular layers and serosa. Gangrene and perforation may occur. Fever, tachycardia and leucocytosis result from absorption of dead tissue and bacterial toxins. (Bowers 1939).

Inflammation is reversible in some cases leading to healing by fibrosis and scarring. However reccurence is common. (Befeller 1964). The omentum and small bowel may adhere to the inflammed appendix to form an appendicular mass, which if not treated may lead to an appendicular abscess.

Failure to localise infection, will result in a local or generalised peritonitis and even septicaemia. Ileo-caecal veins may be involved in an ascending thrombophlebitis, leading to portal pyemia, liver abscess and septicaemia (Steward Wallace 1936; Lett 1974). The incidence of perforation, gangrene and peritonitis is common in the extremes of life, probably due to reduced blood supply and lowered immunity. Perforation rate is young adults is 10-15% while in those over 50 years is 50% and in infants is 80% (Campbell and McPhasil 1958; Jackson 1963).

CLINICAL FEATURES

Classically, acute appendicitis present as pain, starting in the umbilical region which shifts to settle in the right iliac fossa. This is however, not, invariable and the site of pain varies with the location of appendix which is frequently variable. A pelvic appendix gives rise to flank and back pain.

Anorexia is common to all patients with acute appendicitis, while nausea and vomiting may occur in up to 75% (Bruce 1939; McPhasil and Campbell 1958). There is usually a slight fever, with temperature raised by about 1° celsius, and an increased pulse rate. There is tenderness in the right iliac fossa, with rebound tenderness and guarding if the parietal peritoneum is involved in the inflammatory process.

Rovsing's sign, that is deep palpation of the left iliac fossa, causing pain in the right iliac fossa, may be positive. Retrocaecal and pelvic appendices may present with few abdominal signs, but may give rise to psoas and obturator signs respectively. A rectal digital examination elicits tenderness in an inflammed pelvic appendix. A mass in the right iliac fossa in a patient presenting with symptoms and signs of acute appendicitis as described above may have an appendicular abscess or a peri-appendicular mass.

These symptoms and signs are atypical in the extremes of life, and the disease often progresses to gangrene, perforation and peritonitis. (Morrison 1901; Bruce 1939; Campbell and McPhasil 1958, Jones 1974).

There is usually a leucocytosis of more than 10,000/ul in 90% of cases (Sasso et al 1970). A plain abdominal X-ray may show:-

(a) An air-fluid level in the caecum and ileum

(b) A radio-opaque faecolith

(c) An increased soft tissue density in the right iliac fossa in cases of peri-appendicular mass (Brooks and Killen 1965). These radiological features were not noticed in all the cases presented.

Differential diagnosis:

Acute appendicitis must be differentiated from a wide array of common acute abdominal conditions. There include acute pelvic inflammatory disease, twisted ovarian cyst, ruptured Graaffian follicle, and ruptured ectopic pregnancy in the female. Also acute gastro-enteritis, regional enteritis, acute cholecystitis, acute pancreatitis and a perforated peptic ulcer must be ruled out in both sexes. In children, acute pyelonephritis, mesenteric ly-

mphadenitis and acute intussuception must also be ruled out. (Barnes et al 1962, Jones 1962; 1974). While this list is by no means comprensive, it attempts to show the range of common abdominal conditions which a surgeon must take into account before declaring that a patient has got acute appendicitis.

TREATMENT

The treatment of acute appendicitis is immediate removal of the insult by the operation of appendicectomy. In an uncomplicated acute appendicitis, this simple operation has gratifying results.

The most common approch is by an oblique incision through McBruney's point and perpendicular to a line joining the anterior superior iliac spine to the umbilicus. The incision is 8 - 10cm long and one third of its lies above this line and two thirds below. The external oblique aponeurosis is divided in the line of the incision. The internal oblique is separated in the directed fibres to resemble a grid-iron. The peritoneum is then opened in the direction of the original incision. This approach was first described simulataneously by McBurney and McArthur in 1894, and it affords adequate exposure and has few complications.

A similar, but muscle cutting incision was described by Rutherford and Morrison in 1901. It provides adequate access, and is especially useful in obese and stout individuals (Taylor et al 1935).

A Lanz incision is a transverse incision, following a skin crease and it is performed mainly for cosmetic reasons.

A right lower paramedian incision is preferred by some especially in females, when diagnosis is uncertain and if there is peritonitis.

Gentleness with tissues, especially in a gangrenous appendix, is necessary to avoid complications such as perforation. The appendix

mesentery is clamped, cut and tied between No. 2 - 0 silk ligatures. The appendix base is then clamped, tied and cut. The rest of the appendix is then removed. The stump may be buried by a purse string suture, but this has been observed by some to cause an abscess in the closed cavity created (Mayo 1943), and some surgeons prefer to leave the stump unburied (Kingsley 1969).

Systemic broad spectrum antibiotics are useful in a perforated appendix and peritonitis (Macarey et al 1971). Drainage of the appendix site is necessary if:-

(a) There is an appendicular abscess

(b) There is doubtful integrity of the caecal wall

(c) There is turbid peritoneal exudate (Winnicombe 1964).

Complications of appendicectomy

Wound infection is the commonest complication noted in this and other series, followed by peritonitis and pelvic abscesses. The commonest organisms cultured were E. coli, staphylococcus aureus, proteus spp., Klebsiella and pseudomonas (Winnicombe 1964).

Wound contamination is frequent in the presence of a gangrenous appendix or peritonitis. Use of packs minimises this complication. Leaving heavily contaminated wounds unsutured or leaving in a drain reduces wound sepsis. In the experience of Polk and Lopez-Mayor (1969), drainage of the wound site reduced wound sepsis from 14.6% to 2.3%. Use of antibiotics, also reduces wound sepsis to a considerable extend.

In the last 15 years, infections following coloerectal and genitourinary surgery including acute appendicitis have been found to be caused by non-spore forming anaerobi bacteria (Willcie 1967). It has therefore been recommended by a study group on wound infection following surgery, that metronidazole be used for both prophylaxis and treatment. (Study group 1974 1975, 1976). This group has noted that prophylactic use of metronidazole reduces

wound sepsis rate following appendicectomy from about 20% to practically nil.

For patients undergoing appendicectomy for acute appendicitis, a dose of 100ml (500mg) given intravenously with premedication and continued post operatively at 8 hourly intervals for at least three days is recommended. It is commonly combined with gentamycin or Kanamycin to reduce chances of sepsis from aerobic organisms. (Willis 1979).

A pelvic abscess presents with an intermittent fever, mucus discharge per rectum and diarrhoea. It can be drained per rectum and an in-situ rubber or tube drain is unnecessary.

Appendicular mass and abscess

An appendicular mass forms when an inflammed appendix is walled off by adhesions and omentum. It is s considered to be a less virulent form of the disease, and is an attempt by the body to localise the infection.

The standard approach to the treatment of a periappendicular mass is conservative, a method first recommended by Oschner (1901). The patient is admitted to hospital and given intravenous fluids, gastric suction, pareneral antibiotics and ~~fear~~ a four hourly monitoring of pulse and temperature.

If fever fails to settle and the mass remains the same in size or increases after 12 hours, then operative intervention is necessary. The success of the method is indicated by a drop in temperature, a diminished mass size and an improvement in the general condition of the patient. The treatment is continued until there is no more pain, and interval appendicectomy is recommended after 6 - 13 weeks (Barnes et al 1962).

Some surgeons prefer to operate on all cases, irrespective of whether, there is a mass or not, contending that it is difficult to differentiate clinically between a mass and an appendicular abscess. If an abscess is encountered it is drained and appe-

ndicectomy may be done at the same sitting or later, and if an appendicular mass is encountered, the adhesions are separated, and appendicectomy done.

An appendicular abscess must be drained without delay, to avoid the high mortality rate following rupture of the abscess into the general peritoneal cavity (Barnes et al 1962). A Rutherford Morrison muscle cutting incision is recommended for this, and it is extended as far laterally as possible. If possible the abscess should be drained extra-peritoneally, and the wound either left unstitched or loose stitches applied with a rubber drain.

Diffuse peritonitis

The approach to this is the same as that to peritonitis due to other causes. Immediate appendicectomy is performed, followed by an adequate peritoneal toilet, and drains left in-situ. A Pus swab is taken for both aerobic and anaerobic cultures and an adequate antibiotic cover given.

Mortality

There has been a decrease in mortality from acute appendicitis, in Europe and the U.S.A. over the last 30 years (Contrell and Stafford 1955); Lazarian 1970). The mortality for perforated appendices have also shown a similar downward trend. This has been attributed to a much wider use of antibiotics and early operation. However appendicular abscess still carries a high mortality. (Barnes et al 1962).

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B. SURGICAL ONCOLOGY

6. CARCINOMA OF RECTUM

SUMMARY

During the period between June 1978 and June 1982, twelve cases of rectal carcinoma were seen and treated in firm I, one of the three general surgical firms, at the Muhimbili Medical Centre. Among the patients treated, only one out of the twelve presented in an early stage (Duke stage A), where a curative surgical excision could be done. Nine patients presented in late stages (Duke stage C_1 C_2 and D) and the aim of treatment in these cases, surgical or otherwise was palliative. The main symptoms encountered were, bleeding per rectum, feeling of incomplete evacuation, loss of weight, pain, incontence of stools, and alteration of bowel habits. All patients had a proctoscopy and biopsy done. Barium enema, examination under anaesthesia, cystoscopy, intravenous urograms, and chest radiographs, were done as indicated in Table V. Two patients had liver and bone scans, during the last 4 months of the period under review, as these special investigations became available at that period. The treatment of the cases presented, was as indicated in table VI. Out of the six patients who had an abdomino - perineal excision, only three were alive and well, at one and half years after surgery. Follow up of patients has been a difficult process, as most of them come from up-country stations and transport to and from Dar es Salaam is difficult.

The aetiology, pathology and treatment modalities of rectal carcinoma, are reviewed quoting extrensively from the world literature on the subject.

INTRODUCTION

Although large bowel cancer is not as common in Africans, as it is in the developed countries of the West, recent reviews show that its incidence is on the increase. (Hiza 1976; Ssali 1977; Sande 1975; Ojara 1980). Large bowel cancer in Africans also tends to occur in the younger age groups, compared to Western societies (El Masri S.H. and Boulos P.B. 1975).

In East Africa, rectal carcinoma is more common in males, and a male to female ratio of 2 : 1 have been reported in Tanzania (Hiza 1976) and Uganda, Ssali (1977). Similar observations were made in Kenya (Sande 1975; Ojara 1980). In the patients presented were 8 males and 4 females, and all but one were below sixty years of age.

In a retrospective study, base on material gathered by the Tanzania cancer registry between 1969 and 1973, undertaken by Hiza (1976), large bowel cancer was shown to constitute about 15% of all alimentary tract cancers with carcinoma of the rectum forming 60% of the large bowel carcinomas. In Ssali's series (1977), 41 patients with large bowel carcinoma were seen in a 7 year period, of which 64% were rectal carcinomas. These figures are similar to those observed in the Western countries.

Pathology ~~and clinical~~ and clinical presentation of rectal carcinoma are the same as that observed in the literature from advanced countries. However patients with malignant disease in general and rectal carcinoma in particular tend to present late for treatment in Africa, resorting first to herbs and other medications from traditional healers.

CASE PRESENTATION

F.A. IP. NO. 253811) was a 30 year old Zigua female who presented to us with a two year history of:-

a) Painless swelling in the anus

b) Bleeding on defaecation

c) Wasting and general body weakness

d) Slight lower abdominal pain. Since there was initially little pain, she did not seek medical advice; she instead went to traditional healers and she was given some herbs, which she took for about two years without noticing any improvement.

She reports that, the mass has now increased in size and has ulcerated. It was now painful and there was more bleeding. In addition her general condition and weight have continued to decline over the same period. She has been well, prior to the onset of these symptoms and had not been admitted to hospital for any serious disease before. She is married with three children, the last being five years old.

On examination, she was ill-looking, generally wasted and anaemic. There was no inguinal lymph node enlargement, or icterus. The respiratory, cardiovascular and central nervous systems were normal. Per abdomen, there was no organomegally nor ascites. On rectal examination there was an ulcer around the anal orifice with everted margins. It was mostly in the anterior aspect, and extending for up to five centimetres into the anal canal. The anal sphincter tone was absent and faeces were passing freely. There was also a large recto-vaginal fistula.

Biopsy was done, and it was reported as a well differentiate adenocarcinoma of the rectum. (Histology report No. HP. 3302/81). A full blood picture showed iron deficiency anaemia, due to chronic blood loss. This was corrected by blood transfusion and iron therapy (total dose imferon).

After the correction of the anaemia, a two stage operative treatment was planned for this patient:-

a) An initial laparatomy to assess the operability of the tumor, and to fashion a colostomy.

b) If tumor was found to be operable, then an abdominal - perineal excision of the rectum and anal canal together with the posterior vaginal wall be done, at a later stage.

Initial laparotomy was done and the tumor was found to have not spread beyond and peritoneal reflection, and was therefore operable. A double barreled sigmoid colostomy was made bringing out the proximal protion in the left iliac fossa as an end colostomy and the distal portion in the midline, below the incision as a mucus fistula. She was taken back to the ward and her ulcer which was infected, was treated with Sitz-baths twice daily and antibiotics, orally.

After two weeks the patient was ready for the abdomino-perineal resection. This was done under spinal analgesia.

Abdominal phase: With patient in a supine position a below umbilicu s median incision was made, excising the old scar and freeing the mucus fistula which was clamped with a Zachary-Cope clamp. The abdomen was opened. The dissection of the pelvis was done stage by stage starting posteriorly with the sigmoid mesocolon, until the peritoneal reflection was reached; then the lateral pelvic walls, ligating and cutting the superior rectal vessels. The ureters were identified and preserved. The anterior aspect was also dissected free of the uterus until the posterior formix of the vagina was reached. At this stage, the, abdominal dissection was stopped, and patient put in a lithotomy position for the perineal dissection.

Perineal phase: With patient in a lithotomy position a purse - string stay suture was put into the anal opening. A wide elliptical incision was made, extending from the coccyx to the vestibule on both sides of the anal canal. This was deepened to meet the abdominal

pelvic dissection; the posterior wall of the vagina was excised with the rectum and anal canal. The whole mass comprising, the anus, rectum and posterior vaginal wall were removed en-block through the perineal wound.

The bleeding vessels were secured with silk, catgut and diathermy, depending on their size. The pelvic floor was reconstituted with chromic catgut 2/0 and the remaining part of the posterior vaginal wall stitched to it with chromic catgut 2 - 0. The perineal wound was then packed with sofra tulle and gauze and patient returned to the original position.

The pelvic peritoneum was stitched with chromic catgut, and the abdominal wound closed in two layers. The rectus sheath and peritoneum were closed in one layer of prolene No. 1 and skin with prolene No. 2-0.

The post-operative course was unremarkable and perineal wound healed well, filling up with granulation tissue. The patient was discharged approximately a month after operation in a good general condition. She was followed up, at the outpatient clinic for about 1½ years, after which she was lost to follow up.

SUMMARY OF THE 12 PATIENTS REVIEWED

TABLEI, AGE GROUPS IN 12 PATIENTS

Age in years	No. of patients
21 - 30	2
31 - 40	2
41 - 50	4
51 - 60	3
- 60	1
TOTAL	12

Age range 24 - 65 years; males 8;

females 4 2 : 1.

TABLE II DUKES STAGING IN 12 PATIENTS STUDIED

Stages	No. of patients
A	1
B	2
C_1	2
C_2	6
D	1
TOTAL	12

TABLE III HISTOLOGICAL GRADING IN 12 PATIENTS STUDIED

GRADING	NO. OF PATIENTS
Well differentiated	2
Moderately differentiated	4
Poorly differentiated	2
Anaplastic Carcinoma	4

TABLE IV SYMPTOMS OBSERVED IN 12 PATIENTS STUDIED

SYMPTOMS	NO. OF PATIENTS
Bleeding per rectum	12
Feeding of incomplete evacuation	6
Loss of weight	5
Incontinence	3
Pain	4
Alteration in bowel habits	3

TABLE V; DIAGNOSTIC WORK-UP IN 12 PATIENTS STUDIED

INVESTIGATION	NO. OF PATIENTS
Proctoscopy and biopsy	12
Cystoscopy	5
Barium enema	8
Examination under anaesthesia	7
Intravenous urogram	2
Chest X-ray	12

In addition to the above investigations, Haemogram, liver function tests, blood urea and creatinine, urine sugar and culture, were done in all patients. Towards the period under review liver and bone scans were available and this was done in two patients. They showed metastases in both patients.

TABLE VI TREATMENT AND FOLLOW UP IN THE 12PATIENTS STUDIED

TREATMENT	NO.	FOLLOW-UP
1. Abdomino - perineal excision	6	3 were well and alive at 18 months; others were lost to follow up.
2. Pelvic exenteration with colostomy and ileal conduct.	1	Died
3. Radiotherapy and Palliative colostomy.	2	One died after 1 year 1 alive
4. Refused treatment	3	1 died after 2 months 2 were lost of follo w u

TABLE VII COMPLICATIONS FOLLOWING ABDOMINO - PERINEAL EXCISION IN 6 PATIENTS

	COMPLICATION	NO. OF PATIENTS
1.	Perineal wound infection	6
2.	Abdominal wound infection	4
3.	Bed sores, Septicaemia and death	1
4.	Urinary retention	4
5.	Impotence (males)	4

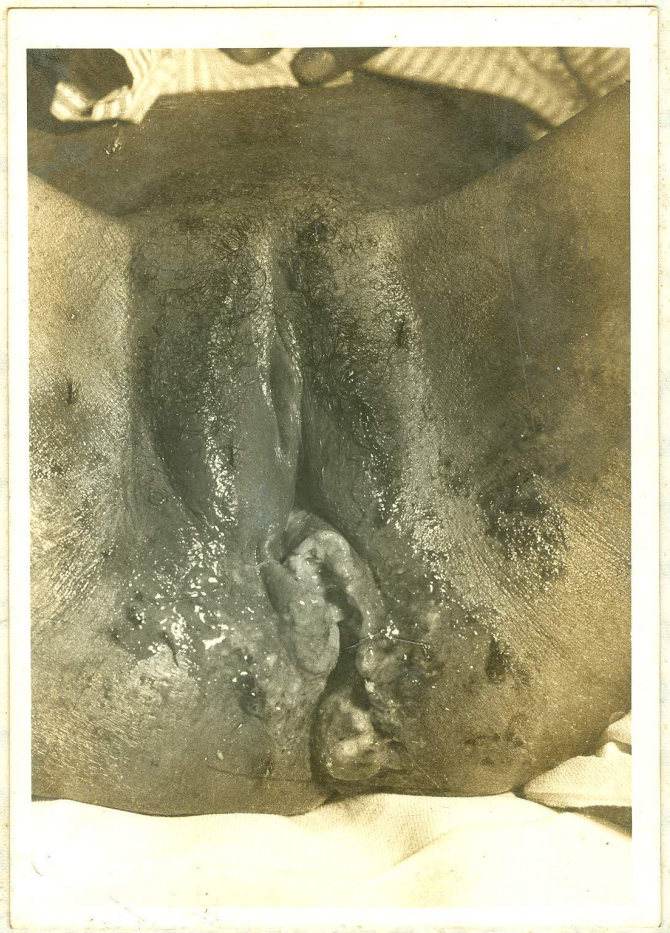
DISCUSSION

Aetiology

Like many other human malignancies, the aetiology of colorectal cancer is unknown. However several factors are implicated. These are:-

1. Diet:

In societies, where a low residue diet is consummed, with it's prolonged intestinal transit time, carcinoma of the rectum is common. (Burkitt 1971; Walker 1970). This is presumably because prolonged intestinal transit time exposes colon and rectal mucosa to dietary carcinogens for a longer time. Also enteric bacteria break down bile acids to form carcinogenic polycylic hydrocarbons, which exert their action for a longer time in individuals with prolonged intestinal transit times. Hill and associates (1975), identified these bacteria as anaerobic organisms, notably "clostridum paraputrificum", and were further able to show that, healthy subjects from areas of colorectal cancer had a high concentration of fecal acids and more dehydrogenating clostridia, than matched controls from low incidence areas.



Ca Rectum ulerating though the anus

A high fat diet, common in western societies with a high incidence of colorectal cancer, depresses levels of mucosal enzymes, especially benzepyrene hydroxylase, which breaks down carcinogenic polycyclic hydrocarbons e.g. benzepyrene to harmless products. (Schwartz 1972, Burdette 1970).

2. Genetic factors:

Colorectal carcinoma is much more commoner in patients with: familial polyposis coli, ulcerative colitis, adenomas of the colon, than in normal individuals. There is also evidence that carcinoma of the rectum occurs in families. However, no pattern of genetic transmission has been proven. (Schwartz 1972).

PATHOLOGY

About 70% of colorectal cancer is found in the distal colon and rectum; and 90% of these are palpable by a finger in the rectum. (Schwartz 1972). From gross macroscopic apperance, rectal carcinoma is described as; polypoid, nodular, ulcerating, scirrhous or colloid. Microscopically, carcinoma of the rectum resemble normal rectal mucosa, to a varying degree; from well differentiated tumor cells grouped in acinar clusters, simulating normal rectal mucosal glands to anaplastic cells with no resemblance to normal rectal cells. A tumour grading system, attributed to Broader, classifies tumors on the basis of the number of cells, resembling glandular cells from which they are derived. Thus a well differentiated rectal carcinoma will have 75%; a poorly differentiated adenocarcinoma 25 - 49%; and anaplastic carcinoma less than 25% of the cells resemble normal rectal mucosa cells. When taken together with Dukes clinical staging, this correlates well with prognosis.

Dukes classification of colorectal cancer (Duke 1932, 1940), iss based on the extend of the malignant process, and is a useful tool to the surgeons as he can gauge the extend of the tumor at the operating

table. Dukes A lesion is confined to the mucosa; B-1 extends into but not through the muscularis propria; B-2 penetrates the muscularis propria but with negative nodes; C-1 lesion is limited to the bowel wall but with positive nodes. A C-2 lesion extends through all layers and positive nodes. A type D lesion has been added to the original classification to denote lesions with distant matastases.

More recently, Wood et al (1981) proposed a system of staging which emphasizes extent of tumor invasion to adjacent tissues or organs. The proposed staging is as follows:-

1. Tumor confined to bowel wall with no distant metastases
2. Microscopic tumor penetration of the bowel wall, but no macroscopic local invasion and no distant metastases.
3. Tumor penetration of the bowel wall with macroscopic local invasion to adjacent tissues or organs and no distant metastases.
4. Distant metastases, unresectable tumors. Peritoneal seedlings; lymph node metastases beyond the line of surgical resection.

While this system, is a controversial deviation from existing staging systems for colorectal cancer, it highlights local tumor invasion as a significant prognostic determinant, and it's authors argue that it is intended to complement rather than replace current classifications.

Spread of rectal carcinoma

Carcinoma of the rectum spreads by direct invasion, lymphatic spread, blood stream dissemination and by implantation at operation. Significant extension by continuity is by penetration of the bowel wall, but some intramural extension principally in the submucosa may occur, but it rarely exceeds 4cm distal to the tumor and 7cm proximal to the tumor (Dukes and Bussey 1958).

Spread to regional lymphatics is the commonest form of metastasis for rectal carcinoma.

Above the level of 2.5cm from the anal orifice, lymphatics spread of rectal carcinoma is strictly upwards (Dukes and Bussey 1958). Spread along the middle rectal vessels to lymph nodes along the internal iliac vessels also occurs in growths arising from the lower and middle third of the rectum. Anal growths drain to the inguinal lymph nodes.

Vemuous spread and extra mural spread to pelvic organs, as well as peritoneal dissemination are late manifestations.

CLINICAL FEATURES AND INVESTIGATIONS

The symptoms of carcinoma of the rectum vary according to the duration of growth. The most constant symptoms are:-

i) Alteration of bowel habitis

ii) Passage of blood, stained mucus or stool per rectum

iii) Abdominal pain

iv) Sense of imcomplete evacuation of the rectum

Rectal bleeding occuring mainly at the end of defaecation, is the most constant symptom. The sense of incomplete evacuation is observed mainly in tumors arising from the rectal ampulla, and alteration of bowel habits, presenting with constipation alternating with diarrhoea and tenesmus is characteristic of growths in the rectosigmoid junction and ampulla. The main symptoms observed in our series is given in Table IV.

The majority of rectal carcinomas can be felt by a finger in the rectum, and a bimanual examination may reveal a higher growth in the rectosigmoid junction. In females a bi-digital rectal and vaginal examination may give further information.

Protoscopy and sigmoidoscopy will show the tumor, its extend and other characteristics, as well as enabling a biopsy to be taken. A barium enema and air contrast studies will show the upward extent

of the tumor and other lesions which may have been missed at sigmoidoscopy.

Other investigations include, haemogram, erythrocyte sedimentation rate, albumin/globulin ratio, serum proteins, bilirubin, blood, urea nitrogen and creatinine, intravenous urogram, a radioisotope liver scan and a chest X-ray. The investigations done in our patients are listed in table V.

The differential diagnosis include, benign papillomas, adenomas, inflammatory strictures, amoebic and schistosomal granulomas, granulomas, lymphogranuloma venereum, endometriosis, tuberculous ulcers, leiomyomas and carcinoid tumors. These conditions are however, not common. We have seen one case of leiomyoma of the rectum in a two year period and despite the hyper-endemicity of schistosomiasis in our area, we have not encountered a case of schistosoma granuloma of the rectum.

TREATMENT

The mainstay of treatment of rectal carcinoma is surgical excision, and the most commonly performed operation is abdomino-perineal excision of the rectum with a permanent colostomy. (Lloyd Davies 1938). This is now a standardised procedure, which in suitably selected patients, aims at radical cure. Other forms of radical excision which aim at curative resection but avoiding a permanent colostomy, are determined by the distance of the tumor from the anal varge, the size of the tumor, and its degree of differentiation.

ABDOMIN-PERINEAL RESECTION

The indication for this operation is Dukes stages A, B and C tumors with a margin of less than 7 - 8cm from the anal verge. For growths above this margin, abdomino - perineal resection may also be performed if an anterior resection is found to be difficult to

perform or dehiscence of the anastomosis is thought to be likely. (Lloyd Davies 1938; Lockhart Mummery et al 1972; Goligher 1975).

The operation involves the removal of the anorectum, anal sphincters and the fascia propria surrounding the rectum, the sigmoid loop, the whole of the pelvic mesocolon with its lymphatic drainage and the peritoneum lining the floor of the pelvis. A permanent colostomy is also established.

It can be performed by a single surgeons doing the abdominal part first, then the perineal part (Miles 1908). The synchronous combined abdomino-perineal excision is performed by two surgical teams in the lithotomy - Tendelenburg position (Loyd Davies 1938) It has several advantages; namely, it obviates the need to turn the patients, and considerably shortens operating time.

Bowel preparation

Mechanical bowel cleansing is important because bacteria form about 2% of fecal mass. (Goldrin 1975). A low residue diet is recommended for 4 - 5 days prior to the operation and an enema given 48 hours before operation, followed by colonic irrigation with normal saline, in the evening and morning before operation. An interesting development in the last few years in mechanical preparation of the bowel, for large bowel surgery, was an attempt by Hewitt et al (1973) at total gut irrigation in a period of 2 - 3 hours.

The irrigation is delivered into the stomach via a naso-gastric tube while the patient sits in a commode. The irrigation solution contains sodium chloride (6.14g), potassium chloride (0.75g), and sodium bicarbonate (2.94g) in distilled water (1000ml), warmed to 37°C. It is pumped into the stomach at a rate of 75ml per minute. Bowel action usually starts at 40-60 minutes after the start of the irrigation and a clear fluid is passed at about

90 minutes and the irrigation is continued for an hour after this stage. Up to 10 litres of this irrigation fluid may be used. The irrigation is well tolerated by patients and it produces a most effective cleansing of the bowel for large bowel surgery, colonoscopy and contrast medium radiological studies. However, its margin of safety, still needs to be defined. (Goligher 1977).

The use of antibiotics and other antibacterial agents, in preparation for large bowel surgery is advocated by many. (Polk and Lopez Mayer 1969; Evans and Pollock 1973, study Group 1974, 1975, 1976). Stokes et al (1974) advocate combined therapy against commonly encountered organisms. They recommend gentamycin against gram negative aerobic organisms, combined with Lincomycin or metronidazole for their effect against non-spore forming anaerobic bacteria which inhabit the large bowel, 1gm metronidazole may be given as a bolus in the last pre-operative day, followed by 200mg, eight hourly until pre-operative starvation starts, or given with pre - medication intra - venously (500mg) and continued for three days post operatively at eight hourly intervals. This is combined with gentamycin 50mg twice daily. (Goldring 1975; Willis 1979).

Non absorbable oral antibiotics, such as neomycin or a sulphonamide have been used for a long time, but they are occasionally associated with complications such as pseudo-membranous colitis. (Collins 1970).

Technique operation

A left paramedian incision extending above the umbilicus if necessary is recommended. The pelvic colon is mobilised by incising the lateral peritoneal reflection, identifying and securing the left ureter from injury. The inferior mesenteric artery is divided and tied just below its origin from the abdominal aorta, the blood supply to the remaining left colon being met by the marginal artery. The dissection is continued until the pelvic colon and the rectum have

been completely mobilised from the lateral pelvic walls, the sacrum, and anteriorly. The site for a colostomy is now chosen. Some surgeons still prefer to place it, in the left iliac fossa, but the tendency is now to place it higher up to the umbilicus level lateral to the umbilicus, as this permits easy application of an appliance and reduces chances of herniation (Maingot 1969).

The perineal dissection is carried out by placing a purse - string suture around the anus, which when tied invaginates the anus and the distal anal canal. An eliptical incision is made on both sides, around the anus, starting from the a sacro-coccygeal joint to the perineal body, and this is deepened to divide the ischio-rectal fat close to the bones and the medial part of the gluteus maximus muscles. The sacrococcygeal joint is opened and the coccyx excised out. The incision is then deepened to communicate with the abdominal dissection and the whole mass, comprising the anus, the rectum and the pelvic colon together with the surrounding fascia and peritoneum are removed. In females the posterior vagina wall is also removed. (Butler 1971).

Management of the perineal wound is still a controversial issue. Some surgeons still prefer to pack the wound with non-adhesive dressings, but Irving and Goligher (1975) recommend primary closure and drainage with suction, through a separate stab wound. Various authors have reported rates of primary healing with primary closure of the perineal wound ranging from 60 - 90 percent (Ruchley, Smith and Balfour 1970; Hulten et al 1971; Denker et al 1973; Broader et al 1974, Walter and Malik 1974; Altemeiyer 1974; Goligher 1980). On the basis of the experience of these authors, it is now recommende that primary closure with suction drainage be employed, unless it is specifically contra-indicated, e.g. by inadequate control of bleeding or gross contamination of the perineal wound.

Results of surgical treatment for rectal carcinoma, have been analysed by many authors, especially at specialised centres, like the St Marks Hospital for diseases of the rectum and colon

in London. (Gabriel 1932, 1957, 1963; Duke 1940, 1957, Morgan 1965, Bussey 1964, Lockhart Mummery 1969, 1976). The results as analysed by Dukes (1957) show a rising resectability rate, a falling operative mortality and an increasing five year survival rate, between 1928 and 1952, and it has been increasing ever since. (Morgan 1965; Lockhart Mummery 1976). An overall five year survival of 63.8% among 990 patients was reported by Lockhart Mummery et al (1976) and Dukes stage A, have the best 5, 10 and 15 year survival rates of 85.8%, 67.2% and 34.5% respectively. (Lloyd Davies 1969).

Sphincter saving operations

Sphincter saving operations for rectal cancer, have been employed since the last quarter of the nineteenth century in suitable cases. (Hochmneg 1889). They received further impetus, when it was realised that, rectal carcinoma rarely spreads beyond 4cm distally. 30 - 60% of all rectal carcinomas are now treated by sphincter saving procedures (Lockhart Mummery et al 1976; Whittaker and Goligher 1976).

The prime criterion for deciding on a restrorative resection, is the distance of the tumor above the anal verge, as well as the degree of tumor differentiation. This determines the length of normal bowel below the growth that can be removed so as to clear any distal intramural or perirectal spread. A margin of 3-4cm below the tumor is safe (Nickolls 1980). This means that, the lowest tumors that can be treated by restorative resection is 7 - 8cm from the anal verge. Poorly differentiated tumors tend to spread more distally, and therefore an abdominal perineal resection is recommended.

(a) Anterior resection

Maingot (1969), has listed a number of criteria that should be rigidly followed if a successfull anterior resection of a tumor situated in the upper rectum or rectosigmoid junction is contemplated. These are:-

i) Growth should be situated not less than 12cm from the anal verge.

ii) The growth should be small or of moderate size and mobile

iii) Histologically, the tumor should be of low grade malignancy

With the introduction of stapling machines in the 1970's for anastomosing the bowel, very low anterior resections are now possible (Nicholls 1980). The extent of the abdominal dissection is the same as in abdominal perineal excision. The anastomosis made above the peritoneal reflection (High anterior resection) is simpler and has less anastomotic leakage. There is a high incidence of leakage at the anastomotic site, when the anastomosis is made below the peritoneal reflection (Low anterior resection). Anastomotic leakage is manifested by a pelvic abscess or a faecal fistula and a frequency ranging from 5 - 30% of cases. (Fielding et al 1978). Tension and poor vascularity of the distal colon, defects in the suture line and a post operative discharge of a pelvic haematoma through the anastomosis are possible causes.

There is no difference in the dehiscence rate whether a two layer or a single layer technique of anastomosis is used (Everett 1975; Goligher et al 1977). Although anastomosis using a stapling device (e.g. the Russian STPU instrument or the American EEA stapler) is much easier than hand suturing, there is no evidence that these staples reduce anastomotic leakage (Fain et al 1975; Goligher et al 1979). Some surgeons prefer to do a complementary transverse colostomy, in connection with a low anterior resection, to diminish the hazard of faecal contamination, should anastomotic dehiscence occur (Goligher 1977).

Abdominal pull-through resection

In this operation, the resection of the growth is done in the abdomen as in anterior resection, leaving a long colonic and a very short anorectal stump. These stumps are brought into continuity by pulling the colon through the anal canal, with the anorectal stump everted before the colon is drawn through. Complications following

this procedure include anastomotic leakage and necrosis of the bowel, but in experienced hands it has a low morbidity and mortality. (Bacon 1956; Goligher et al 1965; Kennedy et al 1970).

Abdominal transanal resection

This operation was introduced by Parks in 1972. The patient is put in a reversed Trendelenburg position with legs raised. The abdominal dissection is carried out as in nterior resection and the tumor bearing portion excised out. The remaining distal colon is pushed through a previously widely dilated anus held open by a retractor. Anastomosis is then carried out, under direct vision through the anus. Parks (1972) reports favourably on the results of this operation in his hands, but more time is required for a proper appraisal.

Sacro-abdominal resection

The left colon and rectum are fully mobilised but not divided through an abdominal approach. They are pushed down into the pelvis, and the patient is then turned to a Sim's position after the abdomen is closed. The coccyx is excised through a transverse or oblique curved incision, from which the mobilised rectum and distal colon are drawn out as a loop and resected. The colonic and distal anorectal stumps are then anastomosed under direct vision. A complication of this procedure is anastomotic dehiscence with the development of fecal fistula through the sacral wound. Localio (1975, 1978) who has extensive experience in this procedure, now performs a transverse colostomy to protect the anastomosis. Longer periods of follow up of patients who have had this operation are necessary for a proper appraisal.

Abdomino-transphincteric resection

This operation was developed by Yolk - Mason (1972). The abdominal dissection is carried out as in anterior resection, but not resected at this stage, and the abdomen, is closed. The patient

is then turned to a jack - knife position and the rectum is approached through a longitudinal para - sacral incision dividing the anal sphincters completely. Resection is then carried out and anastomosis under direct vision done. The sphincters are then repaired in layers. Mortality rates (2 - 10%) and anastomotic dehiscence rate (5 - 25%) are similar to anterior resection, but this procedure needs more time to be properly evaluated.

Local resection for rectal carcinoma

Tumors that can be resected by local excision are adenomas with malignant change or sessile ulcerative carcinomas. Suitable cases should be selected by strict criteria, which are:-

i) The growth should not have penetrated the bowel wall
ii) The growth should be small and histologically well differentiated
iii) There should be no palpable para rectal lymph nodes
iv) If possible, the resected specimen should be examined by a pathologist (Masson et al 1977).

Diathermy fulguration is advocated by some (Madden and Kandalaft 1971). As much of the tumor as possible is destroyed in the first treatment, and treatment is then repeated at regular six monthly intervals. One possible draw back for this procedure is that, no specimen for pathological examination is available.

RADIOTHERAPY FOR RECTAL CARCINOMA

Adenocarcinoma of the rectum responds to radiation therapy (Nicholls 1980). Radiotherapy can be used both for palliation and cure. Palliative Radiotherapy. Distressing symptoms from inoperable carcinoma can be improved in a high proportion of cases by radiotherapy. Pain and bleeding are the main symptoms and they can be effectively controlled by irradiation. (Williams and Horwitz 1956; Wang and Schultz 1962; Uredanata lafee et al 1972). However, symptoms, tend

to recur. Radiotherapy can also render a locally inoperable tumor operable. (Stevens 1976).

Curative Radiotherapy

Papillon (1975) treated 186 patients by intracavitary low voltage X-rays applied directly through a special protoscope. Three to five applicantions of 2500 - 4500 rad were give over 4 - 6 weeks, to a total of 10,000 - 15,000 rad. Criteria for selection of these patients were similar to those of local excision. Overall 5 - year survival rate was 78%.

Chemotherapy and immunotherapy for rectal cancer

Fluorouracil and it's analogues (e.g. 5 FUDR) give a partial response rate of about 20% in rectal carcinoma, followed by methyl CCNU, mitomycin and razoxane (ICRF - 159) (Nicholls 1980). None of these drugs, alone or in combination are effective in the treatment of rectal cancer; nor as adjuvants to surgery are they superior to radical surgical excision alone. (Grage 1979; Higgins 1976).

BCG, Levamisole (Ketrax) and BCG methanol extract (BCG - MER) have been used to increase non - specific immune response in patients with rectal carcinoma with the hope of improving survival. (Lokitch 1979; Bedikan 1978). No improvement was found. At present, it seems that non specific immunotherapy, does not play a slignificant role in the treatment of rectal carcinoma.

Follow-up of patients with rectal cancer

Wangesteen in the 1950's advocated a routine second look operation 6 months after primary resection, and reported up to 20% of local recurrences to be resectable. However this is associated with mortality and morbidity and these outweigh the advantages accur accruing from early detection of emetastases. Carcino-embryonic antigen monitoring post-operatively is much more accurate in predicting sublinical recurrences. It is able to detect recurrence is over 90% of cases. (Staab et al 1978; Steele et al 1980). Whenever available this is now accepted as the method of choice, of detecting

post operative recurrences.

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7. CARCINOMA OF THE OESOPHAGUS

SUMMARY

Twenty patients presenting with carcinoma of the oesophagus, between July 1980 and June 1982, in surgical firm I at Muhimbili Medical Centre were studied and reported. Epidemiological features, such as age, sex, residence, drinking and social habits were carefully recorded in each case. Important clinical features were noted. All patients had barium swallow radiographic studies done, and nineteen out of twenty had an oesophagoscopy done and histological confirmation of the tumor. Treatment modalities offered were as shown in table IX. The majority of the patients were ~~treatment~~ Treated by radiotherapy, which was at times combined with celestin tube.

This was for palliation only as all patients presented in an advanced stage of the disease. Follow up of thse patients was difficult as many patients came from upcountry stations with transport problems, and most were probably too ill to come for follow up. The literature on this important subject is reviewed.

INTRODUCTION

Oesophageal carcinoma is common in East and Southern Africa, but rare in West Africa. (Aziz and Hiza 1980). It is more common in Kenya than in either Uganda or Tanzania, and in these countries there is great variation within various regions. The peculiar epidemiological pattern, has an aetiological significance which will be discussed under "aetiology".

There are variations in the incidence of the disease in the two sexes, ranging from a male to female ratio of 19 : 1 observed in this series to a ratio of 1 : 1 seen in Transkei (South Africa). Aziz and Hiza (1980) found a male to femal ratio of 5 : 1, in 147

patients seen over a 5 year period. They report male to female ratios for Uganda and Kenya as 1.6 : 1 and 12 : 1 respectively. However Bonelli (1980) working in Kampala, Uganda, reported 54 females among 216 patients with carcinoma of oesophagus, making a male to female ratio of 3 : 1.

The incidence of esophageal carcinoma has sometimes increased over a short period of time in some regions. For example in the high incidence zone of Transkei in South Africa, the disease was rare in the 1930s but it has now reached near epidemic proportions (Gillis and Carter 1977). Thus in epidemiological terms, oesophageal carcinoma exhibits both temporal and spatial variation.

CASE PRESENTATION

F.M. IP. NO. 277172, was a 44 year old male, referred from Iringa, with a four months history of dysphagia, regurgitation and progressive weight loss. He also had a retrosternal chest pain, which is not colicky and not severe. He is a member of the Hehe tribe in Iringa region and he drinks locaLy brewed maize beer and also smokes, cigarettes.

On examination he was in a fair general condition, slightly dehydrated and not cachectic. Systemic examination showed no abnormality. A barium swallow showed a malignant stricture in the middle $\frac{1}{3}$ of the oesophagus. Oesophagoscopy showed the tumor at 26cm from the upper incisors, and a biopsy was taken. Histology report revealed a well differentiated squamous cell carcinoma of the oesophagus.

The patient was first treated with radiotherapy using anterior and posterior fields, extending 5cm above and below the visualised lesion and given in 200 rad fractions daily, four times a week to a total of 5,000 rads. This was achieved in 6 weeks.

Although there was less dysphagia at the end of radiation therapy, this was still significant and it was decided that this patient needed a Celestin tube. Under a general anaesthetic, with the patient in a supine position a mini - laparotomy was done, by an upper median incision and a gastrotomy done. Another team did an oesophagescopy and a pilot bougie was introduced through the oesophagoscope and out through the gastrotomy. The oesophagoscope was removed and the proximal end of the pilot bougie was attached to the Celestin tube using silk 1 - 0 sutures. The pilot bougie was then pulled out through the gastrotomy until the Celestin tube appeared. This was further pulled until there was some resistance, signifying that the ballooned end of the tube was resting snugly on the tumor. The tube was then fashioned to size and stitched to the wall of the stomach using No. 0 - 0 silk sutures. The gastrotomy opening was then closed in two layers of chromic catgut 2 - 0 and the abdominal wound was also closed in layers, using No. 2 - 0 silk to the skin.

Post - operatively, the patient did well. He was able to take oral fluids on the third day, and the stitches were removed on the seventh day. He was discharged on the 10th day to Iringa with a letter to the referring surgeon to continue with follow up and to make arrangements for the patient to come again for review after 3 months. We were unable to trace him, after the end of the three months period.

Summary of 20 cases of carcinoma of the oesophagus studied in a two year period (July 1980 - June 1982).

I. SEX DISTRIBUTION AMONG THE CASES STUDIED

SEX	NO.	%
M	19	95
F	1	5
TOTAL	20	

m : f = 19 : 1

II. AGE DISTRIBUTION AMONG THE CASES STUDIED

AGE GROUP IN YEARS	NO.	%
30 - 39	2	10
40 - 49	8	40
50 - 59	7	35
60 - 69	2	10
70 and above	1	5
TOTAL	20	100

The majority of the patients were in the age group 40 - 59, forming 75% in the patient studied.

III. PATTERN OF ADMISSION

	NUMBER	%
First admission	20	100
Re-admission	0	-
TOTAL	20	100

IV. REGIONAL DISTRIBUTION (REGION OF RESIDENCE) FOR THE CASES STUDIED

REGION	NUMBER	%
Dar es Salaam	3	15
Pwani (Coast)	2	10
Tanga	4	20
Kilimanjaro	1	5
Morogoro	4	20
Mbeya	2	10
Other	4	20
TOTAL	20	100

The majority of patients came from Morogoro, Tanga and Dar es Salaam.

V. THE MAIN SYMPTOMS RECORDED AMONG THE PATIENT STUDIED

SYMPTOM	NUMBER	%
Dysphagia	20	100
Thoracic pain	9	45
Regurgitation	20	100
Weight loss	18	90
Cough	2	10

VI. RECORDED FACTORS ASSOCIATED WITH CARCINOMA OF THE OESOPHAGUS AMONG THE CASES STUDIED.

FACTOR	NUMBER	%
1) Drinking local brew	16	80
2) Drinking local brew and tobacco smoking.	13	65
3) Smoked fish (common among Tanga and Lindi residents)	12	60
4) Previous oesophageal disease	1	5
5) Previous and associated head and neck cancer.	nil	0

VII. LOCATION OF THE TUMOR WITHIN THE OESOPHAGUS

SITE	NO.	%
Upper $\frac{1}{3}$	1	5
Middle $\frac{1}{3}$	12	60
Lower $\frac{1}{3}$	7	35
TOTAL	20	100

VIII. HISTOLOGICAL CLASSIFICATION OF THE TUMOR

TYPE	NO.	%
Squamous Cell	15	75
Anaplastic	3	15
Adenocarcinoma	1	5
TOTAL	19	95

IX. MODALITIES OF TREATMENT OFFERED TO THE CASES STUDIED

MODALITY	NO.	OBJECTIVE RESPONSE WEIGHT GAIN AND RELIEF OF DYSPHAGIA	OUTCOME
1. Radiotherapy alone	14	6 improved	6 discharged
2. Celestin tube alone	2	Dysphagia relieved	8 died
3. Radiotherapy and Celestin tube.	4	All improve	Discharged
4. Surgery (oesophagectomy)	1	Died	
5. No treatment (Refused)	1	-	-

DISCUSSION

Aetiology:

In Africa, the great differences in incidence within short geographic distances suggests an environmental aetiology, although no direct evidence of a particular aetiological factor is available (Langman, 1971). Factors implicated in the aetiology of oesophageal carcinoma are:-

i) Genetic factors: There are ethnic variations in the incidence of oesophageal cancer and familial patterns have been described in high incidence areas (Gills and Carter, 1977). In Europeans, an association between tylosis, (an abnormal thickening of the palms and soles inherited as a dominant characteristic) and oesophageal carcinoma was noted by Howell - Evans et al (1958), but this was disputed by Langman (1971). There is no pisitive evidence of genetic susceptibility to oesophageal carcinoma and exposure to environmental carcinogens is more plausible.

ii) Tobacco and Alcohol:- both alcohol and tobacco consumption, have been noted as important aetiological factors of oesophageal

carcinoma in several parts of the world. (Winder and Bross 1961; Hirayama 1970; Hakulinen et al 1974; Cook 1971). Although chewed tobacco carries a higher risk, there is little difference between various types of smoked tobacco (e.g. cigarretes, cigars and pipes). Cook (1971) undertook a detailed study of African home brewed beers and their relationship to the increased incidence of oesophageal carcinoma. The results from this study suggested that some constituents of maize beer or one of its fermentation products, was involved. However the nature of the carcinogenic agent is not known.

There are situations where correlation between oesophageal carcinoma and alcohol and tobacco consumption cannot be upheld. For example, in the high incidence zone of the Caspian sea littoral especially in Iran, no alcohol is consummed, and although both alcohol and tobacco are heavily consummed in industrialised countries of north America, and both Eastern and Western Europe, the incidence of oesophageal carcinoma is not high.

3. Local oesophageal conditions:

Oesophageal carcinoma has been linked with strictures of the oesophagus, achalasia, and previous gastric surgery, but the nature of these associations is obscure (McDonald 1971; Warwick and Harrington 1973). Habitual consumption of hot fluids, especially hot tea, which is a common practice in the Asian high incidence areas of oesophageal carcinoma may also be important as an aetiological factor. (Hightower and Salem 1958; Kaufman et al 1965).

Most oesophageal cancers are squamous cell carcinomata. Ong (1975) in a series of 181 patients reported 92.8% squamous cell carcinomas, the rest being adenocarcinoma. In this series, histology was available in 19 patients, and squamous cells carcinomas were 15 (77.7%), anaplastic carcinoma 3 (15.8) and adenocarcinoma 1 (5.2%). (Table VIII).

Spread; There is significant submucous spread in the longitudinal axis. Therefore, a curative resection should involve a segment of at least 4 - 6cm of normal oesophagus beyond the tumor in either direction (Miller 1962). Direct extension through the wall occurs easily and may extent to involve adjacent mediastinal structures, the bronchi and the lungs. Lymphatic spread to the regional lymph nodes as well as haematogenous spread to the liver, lungs and bones does occur. (McKewon 1972).

CLINICAL FEATURES

Progressive dysphagia is the commonest symptom, and all the twenty patients presented with this symptom. Loss of weight, regurgitation and a retrosternal chest pain are also common symptoms in patients with oesophageal carcinoma. Patients may present with tracheo-oesophageal fistula with lung infection and choking on swallowing fluids. Erosion of a blood vessel by the malignant process may lead to massive haematemesis and/or haemoptysis.

Investigation

Barium swallow demonstrate the malignant stricture and its extend, but oesophagoscopy is necessary for a more exact estimation

4. Malnutrition. Although it is difficult to characterise malnutrion with precision in relation to aetiology of oesophageal carcinoma, there is an association between poor nutrition, Low economic status, and a tendency to develop carcinoma (Giles and Carter 1977). There are multiple dietary deficiencies which differ qualitatively and quantitavely in different parts of the world. Vitamin A, riboflavine and nicotinic acid deficiency attract attention as these vitamins are concerned with maintaining integrity of squamous epithelium. Iron deficiency predisposes to the Plummer - Vinson (Patterson - Kelly) Syndrome which is associated with oesophageal carcinoma. Malnutrition for a long period, leads to a degree of immunological incompetence involving cell mediated immune processes that are involved in antitumor immunity.

In this series of 20 patients, consumtion of locally brewed alcohol, smoking and chewing tobacco, as well as eating smoked fish, were noted in a number of patients (Table VI).

Pathology: Oesophageal carcinoma is slow growing and it takes a long time before it gives rise to symptoms. To facilitate choice of treatment, the oesophagus is divided into an upper, middle and lower third segments, corresponding to the distances of 15 - 23cm, 24 -31cm and 32 - 40cm from the upper incisors respectively. Carcinoma is commonest in the middle third and in areas of constriction i.e. hypopharynx, under the aortic arch and at the cardia (Gunnlaugson et al 1970, Ong 1975, Azizi and Hiza 1980). The middle third segment, was the most commonly involved site in the twenty patients presented (Table VII).

of the level of the tumor and also for taking a biopsy. A plain chest X-ray may reveal aspiration pneumonitis or pulmonary metastases. Bronchoscopy may show involvement of the trachea and the main bronchi. It is indicated if a tracheo-broncho-oesophageal fistula is suspected, as the use of a thick barium sulphate in the presence of a fistula may lead to airway obstruction.

TREATMENT

Surgery and radiotherapy are the mainstay of treatment for oesophageal carcinoma. They are used for both palliation and cure. Oesophageal bouginage and intubation are used for palliation only.
Resection: The type of surgical resection depends largely on the level at which the tumor is situated (McKewon 1972). The aim of surgical treatment in most cases is relief of dysphagia as only a few patients present early enough for a curative resection.

Lower ⅓ cancer:

Objectives of surgical excision for growths of the lower one third are radical and curative, as it is possible to carry out a block dissection of the growth, its lymphatic fields and all adjacent structures.

The classical approach is by a left thoracoabdominal incision along the line of the 7th rib with the patient lying in a right lateral position. This approach is especially ideal for growths below the diaphragmatic hiatus and the gastro-oesophageal junction, but access is limited for growths above the diaphragmatic hiatus.

Reconstruction using a Roux-en-Y or jejunal loop with jejuno-jejunal anastomosis, combined with a total gastrectomy is the most suitable resection for carcinoma at the gastro-oesophageal junction

as it allows a complete clearance of gastric lymphatic glands. (McKewon 1972). For growths above the gastro-oesophageal junction, preservation of the pyloric antrum and anastomosis with the proximal oesophageal stump is recommended (Collis 1971; McKewon 1972).

Middle ⅓ cancer: A left sided thoraco-abdominal approach is unsuitable for middle third oesophageal carcinomas because the proximity between the oesophagus and the heart, the thoracic aorta and the lungs makes dissection and insertion of sutures difficult. For these growths, a two stage technique first developed by Lewis (1946) and Tanner (1949) is recommended. This approach is also suitable for Lower one third tumors (Franklin 1970). The stomach is first mobilised through an upper left paramedian incision taking care to preserve its blood supply from the gastro-epiploic arch and the right gastric artery. The left gastric artery and the short gastric vessels are cut and tied off and the peritoneal reflections excised. The spleen is removed and the second part of the duodenum is mobilised (Kocher's manouvre). A pyloromyotomy is recommended, to facilitate gastric drainage as section of the parasymphathetic nerve supply is an inevitable consequence of this operation.

The second stage consists of exposure of the oesophagus through a right thoracotomy in the 6th interspace. The oesophagus is mobilised from adjacent structures, ligating and cutting the azygos vein in the process. The stomach is then pulled into the right chest, through the diaphragmatic oesophageal hiatus, so that the affected part of the oesophagus, as well as the proximal part of the stomach are resected en-block. The distal remnant of the stomach is sutured in two layers. The proximal oesophageal remnant

is anastomosed end-to-side to the stomach remnant, using vertical mattress silk sutures to produce a perfect mucosal apposition with knots tied in the lumen. An outer layer of continous catgut is desirable and the stomach should be anchored to the parietal pleura to diminish tension in the anastomosis (McKewon 1972).

Three stage total oesophagectomy:

This was developed because of difficulties encountered in the two stage procedure for growths in the upper oesophagus, involving the upper middle third and the upper third. The first two stages are similar to the Lewis - Tanner procedure, but the stomach is mobilised with an even greater care to preserve adequate blood supply up to the fundus. This is done by cutting the left gastric artery at its point of origin from the coeliac axis to preserve its anastomotic connections up to the fundus. Extensive mobilisation of the second part of the duodenum and head of the pancreas, combined with a pyloromyotomy are essential.

A right thoracotomy is done and after the azygos vein is divided between ligatures the entire thoracic oesophagus is mobilised, taking care to avoid damage to the left recurrent laryngeal nerve anteriorly and the trachea presteriorly.

The third stage involves exposure of thoracic oesophagus. It is easier on the right side of the neck between the right thyroid lobe and the carotid sheath. The gastro-oesophageal junction is oversewn after the entire thoracic oesophagus including the tumor has been pulled into the neck, and excised. The cervical oesophagus or even the hypopharynx can be anastomosed to the gastric fundus.

McKewon (1973) who developed this technique listed a number of advantages over other surgical procedures for carcinoma of the

upper oesophagus. These are:-

i) A total oesophagectomy gives a wide clearence above and below the growth so that the hazard of inadequate excision leading to recurrence is diminished.

ii) Stomal leakage, if it occurs, would be less harmfull if it occurs in the neck, than if it occurs in the thoracic cavity.

iii) Epithelial union takes place easily between the squamous epithelium of the cervical oesophagus and the mucosa of the gastric fundus. This is not so, with jejunal or colonic replacement.

iv) In total gastric replacement there is only one anastomosis between the cervical oesophagus and the fundus of the stomach whereas in colonic replacement there are multiple anastomoses each with a danger of leakage and contamination with colonic bacteria.

v) The patient is able to eat a full meal without distress or discomfort after a three stage total oesophagectomy, and its mortality rate compares favourably with other procedures.

vi) After a cervical anastomosis, post operative radiotherapy can be given to the hilar lymph nodes without the danger of producing post - irradiation necrosis in the region of the gastro-oesophageal anastomosis. The point of anastomosis can easily be identified by Michel clips inserted at the oesophago-gastric junction at the time of anastomosis.

RADIOTHERAPY

Although oesophageal carcinoma has a poor prognosis, there is a 50 - 60 percent chance of relieving symptoms e.g. dysphagia by

radiation therapy. (Henschke U. 1966). Earlier cases, esepcially these involving upper 2/3 of the oesophagus, treated by radiation therapy using megavaltage radiation yield improved results.

A localising barium swallow, film and oesophagescopy and biopsy are mandatory pre-irrdiation procedures. For curative purposes a dose of 5500 - 6000 rads given in 5 - 6 weeks is adequate. For palliation 400 rads given in 4 weeks is enough. Radiotherapy, given in a cobalt - 60 machine, like the one available at Muhimbili Medical Centre, it is advised to use anterior and posterior portals, and the field extending 5cm above and below the visualised defect. In general the field will be 15 - 18cm long and 7 - 8cm wide depending on the localising film (barium swallow).

A three field technique for cervical and upper 2/3 of the thoracic oesophagus is optional. There is one anterior and two postero-lateral fields, thus avoiding spinal cord irradiation.

In carcinoma involving the distal third of the oesophagus it is advised to use an L - shaped field which includes coeliac axis and perigastric lymph nodes.

Pre-operative radiotherapy, is recommended, when surgery is contemplated. A dose of 400 rads given daily for 5 days, is followed within a week by surgery. If tumor is found to be unresectable, a further 3000 rads is given in a 3 week period. (Henschke U. 1966).

PALLIATIVE PROCEDURES

Very frequently, patients with oesophageal carcinoma present with lesions which are not amenable to curative excision. The greatest problem of these patients is inability to swallow their own saliva. This leads to accummulation of saliva in the mouth and aspiration if the patient attempts to swallow. A feeding gastrotomy or jejunostomy does not solve this problem; and irradiation or bouginage offer only temporary relief.

A permanent solution to this problem is oesophageal intubation (Schwartz 1972). Several oesophageal tubes have been devised (Souttar 1924; Mouseau - Barbin tube 1956 Celestin tube 1959). The most widely used and the most satisfactory is the Celestin tube. When properly used it gives a most gratifying relief of dysphagia for the patient (Hiza 1974). The Celestin tube is introduced through a pilot bougie, and a mini-laparatomy, as reported in the case described. The patient is given instructions to:-

a) Chew food properly

b) Avoid swallowing lumps

c) To wash down the meal with plenty of water

d) Rinse out daily with diluted hydrogen peroxide (Davey W.W. 1968).

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8 CARCINOMA OF THE BREAST - TREATMENT MODALITIES

SUMMARY

Seventeen patients presenting with breast carcinoma, seen in surgical firm I, were analysed. Among the features recorded were age, parity, stage of the disease at which the patient presented, whether the right or left breast is affected, sex, treatment given and response. These are presented in a tabular form.

One of these patients is presented in detail. She presented with a stage II carcinoma of the right breast. A modified radical mastectomy (Patey's operation) was done, followed by radiotherapy to the chest wall and axilla. After this course of radiotherapy, her ovaries were irradiated, to a total of 1200 rads, as she was premenopausal. She tolerated all treatments well and was in a good condition when last seen at follow up.

Current treatment modalities of carcinoma of the breast are reviewed, quoting from the literature on the subject.

INTRODUCTION

Carcinoma of the breast is a common condition all over the world. It is the second commonest cancer of the adult female in this part of the world. In a review of malignant disease in Tanzania Hiza (1976), found a total of 328 female breast carcinoma registered at the Tanzania Cancer Registry over a 5 year period (1969 - 1973). There were 33 male breast cancers over the same period. The distribution was scattered all over the country.

The choice of treatment for this disease is determined by the assumption that breast cancer spreads in an orderly manner, from a focus on the breast, to regional lymph nodes and then to distant

sites. Also the number of patients treated before the tumor spreads beyond the breast and regional lymph nodes might be increased by early detection and screening programs. This justfies these, otherwise expensive programs. (Henderson C. and Canellos G.P. 1980). Current staging systems have been developed to determine these patients with "local" disease and hence benefit from radical surgery and those in which the disease has spread beyond the confines of the breast and its lymphatic drainage.

Patients with "early" stages of breast cancer i.e. Local disease, with no evidence of spread beyond the breast and its lymph nodes, are treated, in firm I, at the Muhimbili Medical Centre with radical surgery (Halsted's or Patey's Mastectomy) or simple mastectomy and radiotherapy to regional lymphatics. Advanced disease is treated by a combination of limited surgery, hormonal manipulation, anti oestrogens, radiation therapy and chemotherapy. Treatment modalities used in the fifteen patients presented are given in table V.

By far the largest group of patients presenting to us with carcinoma of the breast, come with advanced disease. Therefore to standardise treatment in these patients was not possible and each patient was therefore evaluated and treated on an individual basis. As usual follow up of patients in our situation is a difficult process and, only a handfull of patients were available for follow up, for the two years, under review.

CASE REPORT

T.M. IP. NO. 270518, is a 40 year old woman, who was referred from Kibaha hospital with a complaint of a mass in the right breast which has been slowly increasing in size for the last 7 years. It was not painfull, but it was bringing discomfort, especially when breast feeding. She has had a total of twelve pregnancies, three deliveries having occurred

since she started noting the mass. The average duration of breast feeding was one year. She gets her menstrual period regularly.

On examination she was a healthy looking middle aged female, who was neither anaemic nor dehydrated. She had a mass in the outer lower quadrant of the right breast, which was irregular and measuring 6cm in the largest diameter. There was no pectoral muscle fixation, peau d"orange or nipple retraction. Oedema of the breast, skin ulceration and satellite skin nodules were not present. There were palpable mobile homolateral axillary lymph nodes. Other systems were essentially normal.

A chest X-ray and a haemogram were done in preparation for operation, as well as two units of blood. The patient was taken to theatre for a frozen section biopsy and modified radical mastectomy after the preparations were ready.

Operation:

With the patient lying supine, and under general anaesthesia, a frozen section biopsy was taken, by cutting into the tumor. The results were available after about fifteen minutes. The pathology report was of a scirrhous carcinoma of the breast.

The frozen section incision was sutured with silk 2 - 0, and the surgeons changed gowns and gloves. An elliptical incision was then made from the anterior axillary fold to the sternum at the 7th intercostal space passing above the breast. A smaller incision was made to circumscribe the breast. The incisions were deepened and the breast was dissected out first, and with the arm stretched and the pectoral muscles strongly retracted the axillary tail a d axillary fat lying below the axillary vessels were dissected out. The whole mass, comprising the breast, its axillary tail and fat were removed en-block. It was not necessary to remove the head of pectoralis minor during this procedure.

The wound was then closed in layers, using silk 3 - 0 to skin and leaving a tube drain. The patient made an uneventful recovery from the operation and was prepared for radiation therapy.

Radiation therapy was applied to the regional lymphatic drainage sites; the axilla, and supraclavicular area using a hockey stick shaped field. A total of 5500 rads were given using a Cobalt 60 machine, in 200 rad fractions daily four times a week, over a period of six weeks. Later radiation oophorectomy was done, employing a total of 1200 rads to the pelvis. She tolerated all treatment well. At follow up, she was free of disease up to one year after therapy, when she was last seen.

SUMMARY OF 17 CASES OF CARCINOMA OF BREAST

TABLE I; STAGES OF CARCINOMA OF BREAST AMONG THE CASES SEEN

SEX	STAGE				RECURRENT	BILATERAL	TOTAL
	I	II	III	IV			
Male			1				1
Female	1	3	6	5		1	16
Total	1	3	7	5		1	17

TABLE II; PERCENTAGES OF CASES SEEN IN VARIOUS AGE GROUPS

AGE	NUMBER	%
Less than 30 years	2	12 5
30 - 39 years	4	25
40 - 49 years	4	25
50 - 59 years	4	25
Greater than 60 years	2	12 5
TOTAL	16	100

There was 1 male breast carcinoma

TABLE III; PARITY AMONG THE PATIENTS STUDIED

PARITY	NUMBER	%
NULLIPAROUS	1	6.2.5
FIRST PARITY	3	18.75
MULTIPARITY	12	75.0
TOTAL	16	100

TABLE IV; PERCENTAGE OF THE CASES SEEN IN RELATION TO THE BREAST AFFECTED

BREAST AFFECTED	NUMBER	PERCENTAGE
RIGHT	8	50
LEFT	7	43.75
BILATERAL	1	6.25
TOTAL	16	100

TABLE IV: PERCENTAGE OF THE CASES SEEN IN RELATION TO THE BREAST AFFECTED INSTITUTED AND THE STAGE OF THE DISEASE

MODE OF TREATMENT		STAGE				RECURRENT	BILATERAL
		I	II	III	IV		
NO TREATMENT			1				
SURGERY (Mastectomy)	Simple			1	1		1
	Modified Radical		4	1	2		
	Radical	1	-	1			
	Toilet			1			
RADIOTHERAPY	Alone			1			
	Adjuvant		4	1	3		1
	Palliative				1		
HORMONAL MANIPULATION	Oophorectomy			1	2		
	Adrenalectomy						
	Oestrogens						
	Androgens			1	2		
	Hypophysectomy						
	Anti-estrogens		2				
CHEMOTHERAPY	Alone						
	Adjuvant						
	Additional palliation						

TABLE VI; RESPONSE TO TREATMENT FOR VARIOUS STAGES

RESPONSE	STAGE				RECURRENT	BILATERAL	TOTAL
	I	II	III	IV			
Control of the disease (Objective with no recurrence at April 1982	1	1					2
Recurrence April 1982		2	1	1			4
NO RESPONSE BUT ALIVE AT APRIL 1982		1	3	3			7
DEATHS UP TO APRIL 1982			1			1	2

STAGING

For a proper appraisal and comparison of different types of treatment, a system of classification and staging of the disease is necessary. A standardised system proposed by the International Union against Cancer in 1958, and also adopted by the American Joint Committee on Cancer Staging and End Results, has been widely accepted, and it is the one used in this presentation.

This system of classification is called the T.N.M. classification, where T stands for "tumor", N for "nodes" and M for "metastases". The general outline used in breast cancer is as follows.

T - Primary tumor

T1 Tumor of 2cm or less in 'its' greatest diameter, skin is not involved or involved locally with Paget's disease.

T2 - Tumor less than 5cm in size or with skin attachment (dimpling of skin) or nipple retraction (in sub-areolar tumors), and no pectoral muscle



Carcinoma of the breast

or chest wall attachment.

T3 - Tumor of any size or greater than 5cm with any of the following; skin infiltration; ulceration, peaud'orange, skin oedema, pectoral muscle or chest wall attachment.

N - (Regional lymph nodes)

N0 - No clinically palpable axillary lymph nodes (metastases not suspected).

N1 - Clinically palpable axillary lymph nodes that are not fixed. (Metastases suspected).

N2 - Clinically palpable homolateral axillary or infraclavicular lymph nodes, that are fixed to one another.

M - (Distant metastases)

M0 - No distant metastases

M1 - Clinical and radiologic evidence of metastases, other than to homolateral axillary or infraclavicular lymph nodes.

Summary of clinical staging

Stage I - T1 N0 M0; T2 N0 M0

Stage II - T1 N1 M0; T2 N1 M0

Stage III - T3 N0 M0; T3 M1 M0; T3 N2 M0; T1 N2 M0; T2 N2 M0 and includes any combination of T1, T2 or T3 with N2 and M0.

Stage IV Any stage of the disease with distant metastases (M1)

Histological examination of lymph nodes removed at mastectomy provide more precise prognostic information (Henderson and Canellos 1980). Clinical evaluation of the axilla correctly predicts tumor invasion of the nodes in 67% of all patients, while about one third without palpable nodes will have microscopic evidence of the tumor histologically. (Haagensen 1971; Wallace et al 1972). The absolute number of lymph nodes involved with tumor is the single factor most predictive of 10 year and 20 year survival, and patients with four or more positive

nodes have a worse prognosis than those with less number of positive nodes. (Fisher et al 1975). Other well established prognostic determinants, such as tumor size, local skin involvement, tumor fixation to chest wall, et cetera, are indicated in the TNM staging criteria.

Although generally speaking the patients with stage I and II of the disease survive more than those with stages III and IV, there are exceptions to this general assumption. Patients with early or limited disease (stage I with no nodal involvement), about 25 - 35% of them, will die within 10 years after mastectomy, usually as a direct result of tumor recurrence at distant sites, (Haagensen 1971; Fisher et al 1975), inspite of complete control of the disease in the breast and regional lymph nodes. This implies that the disease had spread to distant organs before detectable spread to lymph nodes.

In another study, which contradicts well established prognostic determinants, patients with large lesions (larger than 6cm) and no histologic evidence of nodal metastases had a 24% recurrence rate at 5 years, whereas those with smaller lesions (1-2cm) and positive nodes had a 44% recurrence at 5 years (Fisher et al 1969; Packard et al 1977). These observations, suggest that there are at least two different populations of patients with breast cancer; one in which the tumor grows large within the breast, but without either regional or distant metastases, and another in which distant metastases occur before there is extensive growth within the breast. It may also be that some tumors are intrinsecially more aggressive or the immune response of the host to the tumor is inadequate. Both factors may be important but the relative contribution of each cannot yet be determined. (Henderson & Canellos 1980; Fegiz et al 1974; Carter R.L. 1977).

Numerous attempts have been made to identify patients with more aggressive disease by means of histologic grading systems, including more recently the determination of hormone receptor status. (Bloom et al 1971; Black et al 1975; Walgren et al 1976; Kister et al 1969; Meyer J.S. et al 1977; Knight et al 1977). Histological grading is based on the degree of tubule formation, nuclear pleomorphism and frequency of mitotic figures. In one study involving 1500 patients, those with "low grade" malignancy had a 62% ten year survival rate compared to only 25% for those with high grade cancer. (Brinkley and Haybittle 1975).

TREATMENT MODALITIES IN BREAST CARCINOMA

Carcinoma of the breast is treated, by surgery, radiotherapy, hormonal manipulation and chemotherapy, usually combining two or three of these modalities. No results of treatment, whether measured in terms of percentage of patients alive 10 years after diagnosis and treatment (60%) or patients free of recurrence (89%) have been better than those achieved by Haagensen in a series of over 1000 patients treated with radical mastectomy between 1935 and 1973. On the basis of these objective data, it is difficult not to accept radical mastectomy as a standard upon which all other proposed therapy should be compared. (Henderson and Canellos 1980; Haagensen 1974).

Whatever mode of treatment chosen; it should ideally

(i) Prevent the development of distant metastases in those patients without evidence of either bloodstream or lymphatic dissemination

(ii) Prevent local recurrence of the disease

(iii) Eliminate extensive local surgery, where it is not strongly indicated (Wilson R. 1972).

a) RADICAL MASTECTOMY

This was developed independently by Halsted and Meyer in the last decade of the 19th century. They stressed certain aspects of technique, namely:

i) en-block dissection of all breast tissues

ii) Removal of pectoral muscles

iii) Axillary node dissection

iv) Wide excision of the skin around the breast tumor.

Haagensen was the first to stress the need for carefull selection, to rule out those who are already incurable, if the best results are to be obtained.

b) MODIFIED RADICAL MASTECTOMY

This was first developed by Patey (1943) and it is sometims known as Patey's operation. It differs from the classical radical Mastectomy only in that the pectoralis major is left intact. Several modifications are done by different surgeons; (Handley 1964; Madden 1965) Patey removed the pectoralis minor with axillary and interpectoral nodes; Auchincloss (1963) preserved both pectoral mucles. Although an en-block removal of axillary lmph nodes is not achievedb by this operation, adequate sampling of the lymph nodes is accomplished, with better arm functional result.

c) EXTENDED RADICAL MASTECTOMY

Handley (1964), found that 21% of outer breast quadrant tumors and 34% of inner quadrant tumors had internal mammary nodes involvement at the time of diagnosis. These patients would definitely not be cured by radical mastectomy alone, and it has led some surgeons, to extend the radical mastectomy to include the internal mammary chain. (Wangesteen 1956; Urban 1959; 1977). Although results of 10 year survival obtained by Urban (1977) and Bucallosi et al (1971) quoted by Henderson and Canellos (1980) are impressive, pooled data from combined studies, failed to show an advantage of this extended procedure except in a group of patients with inner quadrant lesions, and even in these patients, sterilisation of these lymph nodes by radiation therapy would achieve the same results. (Lacour J. Bucallossi P. et al 1976).

d) SIMPLE MASTECTOMY WITH OR WITHOUT RADIOTHERAPY

This is designed to remove only the breast tissue and nipple. Crile (1968) vigorously advocated this procedure in patients with stage I disease, without addition of radiation therapy to lymph node drainage sites. McWhirter (1948) used a combination of radiotherapy to the axilla, the supraclavicular area, the internal mammary chain and simple mastectomy. Since 1948 it has been shown repeatedly that the addition of radiotherapy to any surgical procedure, lowers the incidence of local or regional recurrence, especially in patients with involved axillary lymph nodes. (Weichselbaum et al 1976; Brady et al 1977; Basson 1968; Host et al 1977).

e) RADIOTHERAPY AND TYLECTOMY (LUMPECTOMY)

Treatment of breast carcinoma, by radiotherapy alone was until recently confined to those patients who refuse mastectomy. Wizenbarg and Brickner (1979) presented a review of studies done by Baclesse in Paris, Peters in Canada and Hellman in Boston (U.S.A.), in which incisional or excisional biopsies of the tumor is followed by radiation therapy to the breast and lymphatic drainage sites to a dose of 4500 to 5000 rads. Furthermore a supplementary dose of between 1000 rand 1500 rad is given to tylectomy site using either external beam radiation or interstitial implantation of radioactive Iridium (I^{192}). For stage I and II cancers, there is good local control and an excellent cosmetic result.

All the above are local treatment for a disease in early stages, that is before the disease has spread beyond the confines of the breast and its lymphatic drainage sites. Comparison of different types of local therapy for breast cancer shows that no single type of local therapy is superior to all others. (Henderson and Canellos 1980).

TREATMENT OF ADVANCED BREAST CARCINOMA

Advanced breast carcinoma is a disease which has metastasize d to distant sites, regardless of the size of the primary lesion. The likelihood

of "cure" in these patients is remote, but relief of distressing symptoms can be achieved, using different types of treatment e.g. administration of hormones, endocrine ablation, chemotherapy or a combination of these. "Response" in these cases, means objective or measurable reduction of lesions found on physical examination.

a. Endrocrine Therapy

Oophorectomy was first done for breast cancer by Beatson in 1896, and in most institutions, it is still considered, the initial procedure of choice in premenopausal women with recurrent breast cancer. Surgical oophorectomy or radiation castration are both equally effective, but it takes about two to six months before effect of radiation oophorectomy are objectively seen. There is on the average a response rate of 33% in premenopausal women who undergo castration (Veronesi et al 1975, Puga et al 1976). The response rate in women castrated more than one year after menopause is less than 10%.

Adrenalectomy and hypophysectomy are effective because they remove persistent low levels of oestrogens to the growing tumor, after oophorectomy or menopause. The two procedures can be done alternately, although studies comparing the two procedures show that, the response rate, and duration of response are usually slightly higher after hypophysectomy, but these differences are not statistically significant. Adrenalectomised patients may be maintained with cortisone alone, but patients undergoing hypophysectomy either surgically or by yttrium implantation require replacement therapy with cortisone, thyroxine and vassopression. Other forms of endocrine therapy, involve the use of hormones such as oestrogens, androgens, progesterones and cortisosteroids.

Oestrogens (e.g. silboestrol) or endocrine ablation are usually the first line of therapy, after a relapse, whereas androgen s, progesterons and corticosteroids are reserved for second - line therapy. (Kennedy 1974). The median duration of response is 9 - 10 months after oophorectomy or oestrogen therapy and 12 - 14 months after major

endrocrine ablation.

Complications after endocrine therapy must be anticipated and dealt with. The most common are a transient flare of the disease, and a life threatening hypercalcaemia.

The important discovery of oestrogen receptors in cytosol derived from breast carcinoma by Jensen and associates in 1967 (Baum M. 1980), made prediction of patients who would respond to endocrine therapy possible, although only about 60% of patients with oestrogen receptor content above 5 fmol per mg respond to endocrine manipulation(McGuire 1975). More recently, progesterone receptors have been identified, and when both oestrogen and progesterone receptors are positive, the patients chance of remission on endocrine treatment approaches 100% (Horwitx and McGuire 1977). These techniques are however still largely experimental and are not widely available, to clinicians.

Newer forms of hormone therapy, include the use of anti - oestrogens, and suppression of the adrenals using, aminoglutethimide. The most widely used anti-oestrogen is tamoxifen, administered as a 10mg tablet twice daily. About one third of patients treated with tamoxifen have evidence of objective regression, mostly in post menopausal women. (Henderson and Canellos 1980). It's use is associated with very few side effects.

Aminoglutethimide is thought to effect a medical adrenalectomy, by blocking conversion of cholesterol to pregnenolone in the adrenal gland as well as inhibiting conversion of androstenedione to oestrogen in peripheral tissues. Replacement therapy with corticosteroids must be given concurrently with aminoglutethimide (Santen et al 1977). The response rate to aminoglutethimide is 31% and duration of response is similar to surgical ablative procedures.

CHEMOTHERAPY IN BREAST CARCINOMA

Single agent chemotherapy eg cyclophosphamide induces remission of around 30%, which compares well with endocrine manipulation, but with far greater toxicity. Recently combination of three or more cytotoxic agents, each with a differing mode of action or acting at a different point in the cell cycle, and cyclically given at say, monthly intervals have produced remission rates of the order of 50 - 80%, without increased toxicity. (De Vita et al 1975, Carbone and Torney 1977; Baum 1980). These relative lack of toxicity is partly due to the therapeutic intervals which allow rapidly dividing stem cells to recover.

The agents most commonly used, either alone or in various combination are; cyclophosphamide, adriamycin, methotrexate and 5 - fluorouracil. These drugs are not cross resistant. Cooper (1969), instituted a regimen containing five drugs cyclophosphamide, methotrexate, 5 - FU, Vincristine and predmisone (CMF - VP) given in an intensive weekly induction course, followed by a less intensive maintenance course. He reported a response rate of 90%. Other workers confirmed the effectiveness of this drug combination although at reduced rates.

Other drug combinations have been used, but none has been found to be superior to CMF alone. The toxicity associated, with chemotherapy for breast cancer is not different from that associated with chemotherapy for other malignancies eg. vomiting, myelosuppression, thrombocytopaenia, alopecia and increased fatifuability, and they are reversible.

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ORTHOPAEDICS AND TRAUMA

9. EPIPHYSEAL INJURIES WITH EMPHASIS ON DISTAL FEMORAL EPIPHYSIS

SUMMARY

Eight patients, all aged between 12 and 20 years, were seen in the orthopaedics trauma wards, during a 3 months period with distal femoral epiphyseal injuries. The majority were referred cases from upcountry stations, with no attempt at reduction made. Open reduction was done in 4, and was being planned for another two. The rest were seen soon after injury and immediate reduction and immobilisation using a toe - to - groin complete plaster cast done. All patients who were treated recovered.

One of these patients is reported in detail, from the time he presented at the casualty to the day he was discharged. The period was too short for a conclusive follow up. A general review of epiphyseal injuries, with particular emphasis to injuries to the distal femoral epiphysis is presented.

INTRODUCTION

Epiphyseal plate injuries occur specifically in children. The epiphysis may separate through the epiphyseal plate or it may be crushed by a longitudinal force, or a fracture may cross the epiphyseal plate.

They are common injuries in children, although, the specific injuries to the distal femoral epiphysis are uncommon. (Sharrod W.J. 1979; Reidy J.A. 1965). The epiphyseal plate is weaker than normal tendons, ligaments or joint capsule and an injury that may cause a ligamentous tear or a traumatic dislocation in the adult

is more likely to cause epiphyseal separation in the child. These injuries are best managed in the fresh state by closed reduction and immobilisation with a plaster cast. If this is not possible, open reduction with or without internal fixation is the next line of treatment. (Reidy J.A. 1965). Results of treatment, if properly done are usually gratifying.

CASE REPORT

A.S. IP. NO. 316666, was a 19 year old boy, Zaramo by tribe, who presented to the casualty department, having sustained injury to the right leg just above the knee, while playing football. He was hit by an opponent directly, from the lateral aspect. He fell down and was unable to stand up and move. He was taken to a nearby health centre, and was subsequently referred to Muhimbili after about twenty four hours. On examination he was a healthy looking boy with a swollen, tender right leg just above the knee. The leg was in a hypervalgus position. The dorsalis pedis pulse was present and nerve supply to the distal aspect of the leg was normal. Other systems were normal.

An X-ray of the affected part confirmed the clinical diagnosis of traumatic distal femoral epiphyseal separation, Salter - Harris type II. Because of the massive swelling, closed reduction was not done.

Open reduction was done, under a general anasthesia, and an Esmarch bandage tourniquet applied. The patient was placed in a supine position and an S-shaped anteromedial incision was made. The injury site was reached after a layer by layer dissection. The old haematoma which has accumulated at the site of injury was sucked out. Reduction was then effected with ease, and was maintained by placing two strong staples across the epiphyseal plate anteriorly and medially. The wound was then closed in layers, silk 3-0 being used to appose the

skin. The leg was not immobilised in plaster.

He made a quick recovery from the operation and was discharged on non weight bearing crutches after about four weeks. Gradual weight bearing was allowed after eight weeks. When last seen at the end of the three months period he was able to bear weight on the affected limb. He was planned for removal of staples after 6 months.

SUMMARY OF 8 CASES OF EPIPHYSEAL INJURIES SEEN IN A 3 MONTHS PERIOD

NO.	INITIALS	AGE	SEX	SITE OF INJURY	AETIOLOGY	MANAGEMENT	OUTCOME
1.	A. S.	19	M	Right distal femoral epiphysis.	Direct injury in a football game.	Open reduction stapling	Cured
2.	O. R.	16	M	Right distal femoral epiphysis.	Sustained while playing football.	Open Reduction stapling	Cured
3.	Y.S.	15	M	LEFT distal femoral epiphysis.	Sustained while playing playing football.	Open reduction stapling.	Cured
4.	M. A.	15	F	Right distal femoral epiphysis.	Was hit by a falling sack of rice from a lorry	Open reduction stapling planned.	
5.	M. A.	17	M	Left distal femoral epiphysis.	Fell down froma coconut tree.	Open reduction stapling	Cured
6.	S. J.	14	M	Right distal femoral epiphysis.	Road traffic accident	Closed reduction POP immobilisation.	Cured
7.	J.H.	16	M	Left distal femoral epipysis.	Sustained while playing football.	Closed reduction POP immobilisation.	Cured
8.	A. J.	18	M	Right distal femoral epiphysis.	Sustained while playing football.	Open reduction and stapling planned.	Cured

DISCUSSION

Epiphyseal injuries, occur through a layer of hypertrophic cartilage cells on the epiphyseal side of the zone of provisional calcification, so that growing cells are attached to the epiphysis. (Trueta J. and Amato V.P. 1960). The fate of these growing cells, depends on the damage that may have occurred to the nutrient vessels of the epiphysis. The epiphyseal plate is commonly supplied at a point remote from it, so that they are less likely to be damaged in epiphyseal separation. When they traverse the epiphyseal plate from the metaphysis to the epiphysis, they may be damaged in epiphyseal separation. (Dale G.G. and Harris W.R. 1958). The upper femoral epiphysis and the upper radial epiphysis are valnerable in this way. The lower radial epiphysis is more frequently separated than any other, followed by lower ulnar, lateral humeral condyle, upper end of radius and lower end of radius, in that order. Salter and Harris (1963), classified epiphyseal injuries into five types.

TYPE I

The epiphysis separates completely from the metaphysis, along the whole of the epiphyseal line, without any metaphyseal fracture. Displacement is minimal.

It is usually due to a shearing, torsion or avulsion force. It is found typically in birth injuries. Reduction if at all is necessary is usually not difficult, if done sufficiently early, and prognosis for subsequent growth is good.

TYPE II; is the commonest. The line of epiphyseal separation, extends along the epiphyseal plate for variable distance and then passes obliquely into the metaphysis to produce, a triangular shaped metaphyseal fragment. It is due to lateral displacement force which tears the periosteum on

one side and leaves it intact on the other side.

Reduction is not difficult and excessive reduction is prevented by intact periosteum.

TYPE III is due to an intra articular shearing force producing a verticle split from the joint surface to the epiphyseal plate, and then along the plate to the periphery. The fragments may or may not be displaced.

This type of epiphyseal injury is not common and is seen most frequently at the lower tibial epiphysis. Accurate reduction is essential for the restoration of congruent joint surfaces. Prognosis for growth is good provided the blood supply of the separated portion of the epiphysis is intact.

Type IV is a fracture extending from the joint surface, through the epiphysis, the epiphyseal plate and the metaphysis. It is commonly seen at the lower end of the humerus.

Accurate repositioning of the fragment is needed to restore good alignment to the joint and to prevent the possibility of malunion or premature epiphyseal closure which may arise from fusion between the bone of the epiphysis and the metaphysis of the main bone.

TYPE V is uncommon but serious injury due to a severe crushing injury applied to the epiphysis. There is minimal displacement but prognosis for growth is poor. Premature cessation of growth or deformity may occur.

Distal femoral epiphyseal injuries:

These are rare injuries, occurring, almost exclusively in young boys aged between 12 and 20 years. (Reidy J.A. 1965). This particular injury is associated with two important complications.

(a) Injury to the popliteal neurovascular bundle

(b) Damage to the epiphysis and the resultant deformity with continued growth. Damage to the neurovascular bundle may occur at the time of injury, from persistent deformity when swelling increases or at the time of reduction of the

deformity. Immediate attention to these is mandatory. Deformity should be reduced as soon after the injury as possible under a general or spinal anaesthesia. In Salter - Harris type I - III lesions reduction is usually easy. The leg should be immobilised from the toes to the groin in a long plaster cylinder following reduction, with the knee in extension. Circulation and sensation should be closely monitored, for the first 24 hours after reduction. If there is evident neurovascular damage, it will be necessary to expose the vessels in the popliteal region, and to render appropriate treatment.

The leg should be immobilised for 6-8 weeks followed by gradual mobilisation of the knee and the patients moves about on crutches.

Full function should be possible in 12 - 16 weeks. If there has been a delay in reduction or failure, a skeletal traction by means of a Kirschner wire through the tibial tubercle may be done. Traction is first applied in the line of deformity, followed by gradual correction of the valgus or varus and flexion deformity, and the leg suspended in a Thomas Splint with Pierson's knee attachment. If reduction is effected, usually in 10 - 14 days, traction is removed the leg is immobilised in plaster (Reidy 1965).

If the fracture has been neglected for a long time, and a closed reduction is not possible, an open reduction should be carried out. An antero-medial parapatellar incision is recommended to expose the supra patellar region and the knee joint. The fragments could then be readily visualised and adequately freed and reduced. Internal fixation with staples may or may not be done. It is the practice in the orthopaedics/trauma department at Muhimbili Medical Centre, to always immobilise using strong staples, which are removed after 6 months (Sarungi P.M. 1982 - personal communication). We have not seen any

complication following this procedure.

Staples were originally devised to control bone growth, and in many centres, they are mainly used for epiphysiodesis (Blount W.P. and Clark G.R. 1949). They should be well made, strong and non-reacting. They must be accurately placed, to produce the desired effect. In epiphyseal injuries, they are used to stabilise a separated epiphysis, and are usually removed after they have served that purpose.

Complications following epiphyseal stapling include, ex extrusion, broken staples, laxity of knee ligaments and deformities. (Broadway et al 1954; Hogberg N, et al 1957; Pilcher M.F. et al 1962; Poirier H. 1968). Complications are less common, in stapling the distal femoral epiphysis compared to others (Pilcher M. 1968).

Complications of epiphyseal injuries:

Apart from injuries to vessels and nerves and other complications associated with fractures in general, there are specific complications to epiphyseal injuries which include.

i) Cessation of growth - is produced by a Salter - Harris type V lesion in which there is a compression force upon the epiphysis. Parent should be warned of this possibility and a carefull watch upon epiphyseal growth is kept for 6 - 12 months.

ii) Angular deformity - occurs when there is a crushing injury to one side of epiphysis producing partial closure of the epiphyseal line. The angular deformity, so produced is progressive until the end of the childs growth (Salter and Harris 1963). Osterman (1972) showed experimentally that a similar deformity could be produced in animals and then corrected by removal of a bony drige, which is replaced by a transplant of cartilage ot fat. Langenskiold (1975) excised the crushed portion of the epiphyseal

line and transplanted fat into the defect. He showed that cells from the unaffected part of the epiphysis will grow laterally and restore the normal width of the epiphyseal line, which in addition will produce correction of the angular deformity.

iii) In Salter - Harris type IV lesions, in which the fracture, has crossed the epiphyseal plate, if accurate reduction is not effected, so that both fragments remain separated, production of a double ended bone, with eventual arrest of growth can occur. (Siffert et al 1966).

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10. HEAD INJURIES

SUMMARY

Twenty five consecutive cases of acute traumatic head injuries were seen in a 3 months period in the orthopaedics - trauma department from September to November 1982. Five had subdural haematoma which was evacuated; six had cerebral injuries two of whom died on admission or soon afterwards. The rest had cerebral concussion and were admitted for a period of observation of two days before discharge. Aetiology and pattern of head injuries as seen in Dar es Salaam are discussed.

One of the patients with chronic subdural haematoma is presented in detail. He was a young man, who was assaulted while walking at night and hit on the head. He was able to get up and walk home, but developed fluctuating levels of consciousness 3 weeks after injury. A carotid angiogram showed a space occupying lesion on the left temporo-parietal region. A craniotomy was done and haematoma evacuated. The patient made a complete recovery from the operation and was very well about 3 months after surgery.

INTRODUCTION

In most published series, craniocerebral injuries, account for approximately, half of the fatalities from trauma, and less severe head injuries take a lot of hospital beds and days out of work. (Pitt's L.H. and Martin N. 1982). In the U.S.A., head injury is second only to stroke as a cause of death from neurologic disorders.

The majority of head injuries occur as a result of road traffic accidents, although a significant number arise from other causes of craniocerebral trauma (Table II).

Emergency treatment of head injury cases, should follow a general pattern of cardiopulmonary resuscitation, with tracheal intubation if necessary, control of haemorrhage and treatment of shock. The head and neck must be kept in a neutral position, until

cervical spine X-rays exclude instability of the spine. Other associated injuries should then be looked for and dealt with. Indeed, in no other aspect of emergency surgery is the well known adage revive, review, repair and rehabilitate in that order, so appropriate as in head injuries.

CASES PRESENTATION

S.A. IP. NO. 310406, was a 35 year old male who presented to the casualty with fever, haedache and drawsyness of two days duration. He had been assulted and hit on the head three weeks prior to admission. He lost consciousness for a short time but was able to get up and walk home.

On examination he was a healthy young man, drewsy but conscious, and responds to commands, and eyes open spontaneously. He had a temperature of 38°C. Systemic examination was normal. A blood smear, showed malaria parasites, and the patients was started on a course of chloroquine. The fever subsided.

A skull X-ray, showed a calcified falx cerebri which has shifted to the right, indicating a space occupying lesion on the left side. Bilateral carotid angiograms confirmed the presence of a space occupying mass in temporo- parietal region on the left side. The patient was prepared for a craniotomy, as his condition continued to deteriorate while he was being worked up.

Under a Local anaesthetic, with the patient lying supine a 3cm incision was made in the left temporo-parietal junction, right to the periosteum. All the layers of the scalp were then retracted using a self retaining retractor. A single burr hole was drilled and the dura was found to be bulging. When it was opened, a liquefied haematoma gushed out under pressure. About 200ml of altered blood was drained. A soft tube drain, connected to a closed system was

put in and the wound was closed in layers, using silk 3-0 to skin.

Post operatively the patient was put on dehydration therapy with mannitol, and antibiotics empirically. The drain was removed on the third day. He made a remarkable recovery and was in a very good general and mental state when last seen at the follow up clinic.

SUMMARY OF 25 HEAD INJURY PATIENTS SEE IN A 3 MONTHS PERIOD

TABLE I TYPE OF HEAD INJURY

TYPE	NO. OF PATIENTS	PERCENTAGE
Cerebral concussion	10	40
Cerebral Contusion	6	24
Brain Laceration	2	8
Subdural Haematoma	6	24
Epidural Haematoma	1	4
TOTAL	25	100

TABLE II CAUSE OF HEAD INJURIES IN 25 PATIENTS

CAUSE	NO	%
Road Traffice & Accidents	15	60
Assaults	6	24
Fell from a Height	2	8
Hit by a missile	2	8
TOTAL	25	100

TABLE III OUTCOME IN 25 PATIENTS WITH HEAD INJURIES

OUTCOME	NO.	%
1. Complete recovery	15	60
2. Hemiparesis/Hemiplegia	0	-
3. Epilepsy	3	12
4. Dementia	2	8
5. Vegetative state	2	8
6. Death	3	12
TOTAL	25	100

TABLE IV INDICATIONS FOR CRANIOTOMY IN 8 PATIENTS

INDICATION	NUMBER OF PATIENTS
1. A rapidly expanding Subdural haematoma	5
2. Epidural haematoma	1
3. Open head injury with brain laceration and dural tear.	2
TOTAL	8

DISCUSSION

Head injuries include injury to the scalp skull and brain. Skull injuries include fracture of the facial bones; the cramial vault and basal fractures. Brain injuries are our main concern.

Brain injuries can result in a concussion, contusion and laceration of the brain with resultant brain oedema and/or intracranial haemorrhage. There are four types of intracramial bleeding

depending on the layers involved (Jennet 1977). These are epidural, subdural, sub-arachnoid and intracerebral haemorrhage. Subdural haematoma is further divided into acute, subacute and chronic. Sometimes a chronic subdural haematoma liquefied, the cellular elements phagocytosed b and a chronic subdural hygroma forms. (Collin's 1972).

Mechanism of injury and basic pathology:

In civilian life, most head injuries are due to diffuse forces caused by a moving head striking a static surface (usually the road), or by the head being struck by a moving object. The resulting damage to the brain, depends on the rapid decleration when a moving head comes abruptly to rest on the road or acceleration when a stationary head is struck.

In early stages, neuronal lesions occurring at the moment of impact, are due to displacement and distortion of cerebral tissue, occuring during impact. The superior cerebral veins supend the brain in cerebrospinal fluid, allowing greater antero-posterior movements. The midline septum, the falx cerebi is rigid and does not allow lateral movements, and this means that lateral forces cause less diffuse injuries. (Bailey and Love 1971).

Antero-posterior forces mainly cause torsion between, the cerebrum, brainstem and hypothalamus as well as the two portions of the cerebrum at the corpus callosum. Loss of consciousness is due to two factors. These are:-

(a) Shearing effect and injury to brainstem excitatory pathways. Temporary paralysis or permanent damage to these pathways is thought to be the cause of instant loss of consciousness, following the initial impact.

(b) Damage to grey and white matter, with resultant defective memory and personality changes. Retrograds and antegrade amnesia is very common following head injury. These mechanisms results

in the clinical conditions of cerebral concussion, contusion and laceration. When the effect of alteration in total function of the nervous system is superimposed, changes in respiratory function, vasomotor control and other clinical syndromes, seen in head injuries can be explained.

Histopathological findings:

The main source of information is from pathological specimens of brain tissue in fatal head injuries. The main findings are rupture and degeneration of axons and pyknosis of nerve cells. Bleeding into the perivascular spaces of Robin - Virchow is seen commonly at the commissures, the corpus callosum, the third ventricle and the brain stem.

CARE OF HEAD INJURY PATIENTS

This is discussed in two stages.

a. First aid - should be instituted at the site of injury by ambulance personel. It consists of:-

(i) Maintainance of cardio respiratory function by suction of the airway, laryngoscopy and endotracheal intubation or tracheostomy. Use of an ambu bag, for assisted respiration may be necessary

(ii) Control of haemorrhage and shock

(iii) Ryle's tube should inserted and gastric suction instituted. The patient should be put in a semiprone position to avoid aspiration pneumonia.

b. EVALUATION; by a thorough general physical examination should follow initial therapy. Establishment of baseline data such as; date and exact time of injury, the duration of post - traumatic amnesia, the pulse and respiratory rates is of paramount importance in head injuries as repeated subsequent examination should be compared with the initial baseline.

Injuries to the scalp and skull fractures are not usually a diagnostic problem. Basilar skull fractures are diagnosed on the basis of clinical findings e.g. "Raccoon eyes" or peri-orbital ecchymoses are due to intra-orbital bleeding from orbital roof fractures. Bleeding from the ears, indicates fracture to the lateral portion of the temporal bone. Battles sign, an area of ecchymosis behind the pinna represents blood dissecting to the skin from a mastoid fracture. It is usually delayed for 12 - 24 hours after injury.

Cerebrospinal fluid leakage through the nose or ears, indicates meningeal disruption at the site of the basilar fracture. Early onset facial nerve palsy indicates direct facial nerve injury at a temporal fracture site and requires early surgical intervention. Late onset facial nerve palsy resolves spontaneously.

Other associated injuries should be sought, and dealt with, starting from the neck, chest, abdomen, pelvis and the extremities. Neurological examination; A rapid but complete neurological examination provides a baseline for determining patient improvement or deterioration, on subsequent examinations. The examination is standardised, and it is designed to assess the level of consciousness and to detect signs of brainstem dysfunction. The level of consciousness is a measure of the severity of diffuse brain injury. The Glasgow Coma Scale has been widely adopted in many trauma centres as a standard for the examination of the level of consciouness. (Pitts & Martin 1982).

The GLASGOW COMA SCALE

Eye opening	Best motor response	Best verbal response
4 Spontaneous	6 Follows commands	5 Oriented
3 To voice	5 Localised painfull stimulus.	4 Confused
2 To pain	4 Complex arm movement	3 Inappropriate words
1 None	3 Reflex flexor posturing	2 Incomprehensible sounds.
	1 Flaccid	1 None

This simple scheme, which is highly reproducible, records the patient's response to verbal and painfull stimuli and provides a valuable tool for following a patients improvement or deterioration. It can also be charted graphically and at a glance the patient's level of consciousness can be assessed. Alternatively the level of consciousness can be ascertained by using, Collins Levels of consciousness numbers one to ten, where level one is the best response, and level ten is the lowest level of consciouness, with death being the next step. (Collins W.F. et al 1972). The intermediate levels, depend on the response to various stimuli, like voice and pain.

Signs of brainstem compression must be sought. This includes assessment of the integrity of the midbrain, pons and medulla by examination of cranial nerves. Pupillary light reflex allows evaluation of optic and oculomotor nerves. Oculocephalic reflex ("Doll's eyes") consists of conjugate eye deviation, contralateral to the direction of head rotation. It depends on intact connections between the pontine and brainstem nuclei of the sixth's cranial nerve and third cranial nerve. In patients with cervical fractures or comatose patients, the function of the pontine gaze centre and its connection to the 6th nerve can be evaluated by caloric testing (ocular vestibular reflex) by which cold water is irrigated into the external auditory canal; there is conjugate deviation of the eyes to the irrigated ear. The 7th cranial nerve can be tested in a patient with altered level of consciousness by noting facial grimace to supra orbital pressure. Assessment of the the respiratory pattern, cough and gag reflexes, provide information as to the integrity of medullary function and the lower cranial nerves.

Diagnostic work up in head injuries:

1. Radiographic Studies: A chest X-ray and cervical spine X-rays are indicated to rule out injuries to the chest and cervical fractures. Skull X-rays will show, fractures of the skull, or pineal gland shift in intracranial space occupying lesions.

2. Computerised tomographic scanning; (CT scan)

of the head has revolutionised the care of head injuries and has largely replaced carotid angiography and ventriculography in the diagnosis of traumatic intracranial lesions. Epidural, subdural and intracerebral haematomas, cortical contusions, cerebral oedema, skull fractures and intracranial foreign bodies are all visualised with precision.

3. Cerebral angiography; This used to be the mainstay in the radiologic evaluation of severe head injury before the advent of computerised axial tomography. It is still a valuable test where computerised tomographic scanning is not available. It can demonstrate extracerebral and intracerebral space occupying lesion, but the exact nature of the lesion eg haematoma, contused necrotic brain or focal oedema, cannot be obtained from angiograms.

4. Air ventriculography - The lateral ventricles are cannulate via a burr hole. It permits localisation of mass lesions, by demonstrating shifts of the ventricular system. It also offers the additional benefit of allowing measurement of intra cranial pressure.

Treatment of head injuries

After initial emergency therapy, clinical evaluation and diagnostic work-up, a therapeutic plan must be instituted for each patient. Pitt's and Martin (1982), identified four degrees of severity of head injury, each with a specific diagnostic and therapeutic plan. Patients are grouped into four categories and each category has a specific action to be taken. These are tabulated below.

GROUP	FINDINGS ON EXAMINATION	ACTION
1	Brain stem compromise	Diagnostic burr holes
2	Focal deficits	CT scan or angiogram
3	Non focal deficits	Observation or CT scan
4	No deficits	Observation for 4 hours then discharge

This scheme, simplifies an otherwise complex problem of adequate care to these patients and it is adopted for discussion in this presentation.

Group I; The brainstem dysfuction is due to compression by an expanding intra-cranial mass eg epidural or subdural haematoma. The medial aspect of the temporal lobe, the uncus, is shifted medially compressing, the upper brainstem with in the tentorial notch - transtentorial herniation. This manifests clinically as a trial of:-

i) Abnormal pupillary light reflexes

ii) Drepression of the level of consciousness and

iii) Asymmetric motor response Oculomotor nerve palsy with pupillary dilatation results from stretch of the nerve as it traverses the tentorial notch by the shifted uncus. Pupillary dilatation is a reliable localising sign and it is ipsilateral to the space occupying lesion in 95% of cases. Progressive impairment of consciouness is due to failure of the reticular activating system in the compressed upper brainstem, as already discussed. Motor asymmetry reflects either direct compression of the motor cortex or focal pressure on the ipsilateral cerebral peduncle by the hermiating temporal lobe. This manifests as weakness in the contralateral arm and leg. In some cases however, the brainstem shifting, during herniation causes the contra lateral cerebral peduncle to be compressed against the edge of the tentorial notch resulting in

motor weakness ipsilateral to the lesion. Therefore motor weakness is not as accurate as pupillary abnormalities in localizing the site of the lesion. It is contra lateral to the site of the lesion in 80 - 85% of cases (Pitts and Martin 1982).

The management of transtentorial herniation syndrome must be immediate and vigorous, and it includes the following measures.

i. Immediate first aid measures as already discussed

ii. Use of hyper - osmotic agents eg Mannitol (1-1.5gm) per kilogram as a bolus, and can be repeated.

iii. Carefull monitoring of arterial, blood pressure with avoidance of hypotension. Arterial hypertension should ~~be~~ not be treated because this may be a response to elevated intracranial pressure, and lowering it may cause a dangerous fall in cerebral perfusion pressure with resultant cerebral ischaemia.

iv. Operative intervention:-

Exploratory burr holes is search of an extracerebral space occupying mass lesion eg epidural or subdural haematoma should not be delayed. Computerised tomographic scanning or carotid angiograms, before surgery have the advantage of accurate localisation of the lesion before operation, and helps to remove dangers of unnecessary surgery. These advantages are however outweighed by the delay incurred in these investigations. If a haematoma is identified, a full craniotomy is done to evacuate the clot.

v. Steroids are not effective in improving, outcome from severe head injury and in high doses, they increase the chances of intracranial infections. (Pitts and Martin - 1982). They are therefore no longer recommended in the treatment of severe head injury.

vi. Hyperventilation therapy with addition of oxygen is helpfull in preventing anoxia which causes vasocontriction and brain ischaemia.

Group II

Patients with focal neurological deficits e.g. visual, speech or motor defects, with absent signs of brainstem compression (especially pupil dilatation) should be evaluated by appropriate investigations, e.g. CAT scans or carotid angiography. If there is a diminished level of consciousness, mannitol should be administered while a definitive diagnostic work-up is being done. This should only be a temporary measure and definitive therapy must be given, depending on the outcome of investigations.

Group III

For patients who have no neurologic deficit, but only a diminished level of consciousness, carefull serial observations may be employed. Mannitol may be given. If no improvement is detected or the patient shows any deterioration within several hours, appropriate investigations should be done. This is especially important in elderly people with cerebral atrophy because they can harbour expanding intracranial haematomas that produce no focal deficit until they become very large.

Group IV; These are patients who are neurologically normal following head injury. They are unlikely to develop later complications. Patients who develop delayed complications (weeks or months after injury) of chronic subdural haematoma are an exception to this principle.

Amnesia due to injury impact is very common, in cerebral concussion, and short periods of retrograde or antegrade amnesia are of no prognostic importance. But prolonged amnesia manifested by continuing confusion and inability to answer simple questions is a significant neurologic deficit and requires close observation.

INDICATIONS FOR SURGERY IN HEAD INJURIES

The decision to operate on a head injury case, depends on:

i. Deterioration on the level of consciousness with localising signs eg pupillary reaction or hemiparesis.

ii. Depressed fracture of the skull with an instep of more than 0.5cm

iii. Compound skull fracture - debridement and surgical toilet are done.

iv. Cerebrospinal fluid leakage. If CSF rhinorrhoea persists for more than 10 -14 days the CSF fistula has to be found and closed.

v. Signs of internal hydrocephalus:-

If these develop, then a lateral ventriculostomy and CSF shunt into the superior vena cava via the internal jugular vein is done. A Holter valve is used to maintain ventricular pressure at 50 - 70mm of water.

Post operative care:

Supportive care following initial resuscitation and surgical intervention, is a critical phase in the care of these patients, that is optimally provided in the intensive care setting.

1. Management of raised intracranial pressure

Increased intracranial pressure is most pronounced in the first 48 - 72 hours after trauma. Cerebral blood flow depends on erebral perfusion pressure (CPP) which is calculated as mean arterial pressure mims mean intracranial pressure. Intracranial pressure may be controlled using a combination of the following modalities:-

(a) Surgical decompression by removal of space occupying lesions.

(b) CSF drainage if hydrocephalus has formed

(c) Head elivation, enhances venous drainage and lowers venous pressure

(d) Control of body temperative below 37°C. The range of 33 - 37°C is optimal. It reduces brain metabolism.

(e) Hyperosmotic therapy: Mannitol is the most widely used agent; it is given in a dose 1.5 - 2g per kilogram body weight. Repeated use is best guided by actual measurement of intracranial pressure. Lower doses of mannitol (0.25 - 0.5gm per kilogram body weight), every three to four hours may be given for chronic control of intracranial hypertension.

Other agents used are ueaurea and glycerol as well as diuretics such as frusemide (lasix 0.5 - 1mg per kilogram).

(f) Hyperventilation therapy

Cerebral vessels constrict in response to hypocapnia. Cerebral blood volume decreases during hyperventilation, thus lowering intracranial pressure.

ii) Fluid and Electrolyte balance:-

It is important to monitor fluid input and output, and serum electrolytes. Low plasma sodium is not uncommon following head injury, due to inappropriate secretion of anti diuretic hormone. If this is proved of an to be the case, appropriate therapy with antidiuretic hormone is given.

iii) Prevention of seizures:-

Anticonvulants are advised in patients with head injury that produce coma for more than 24 hours. Phenytoin or Phenobarbitone may be given, but the duration of therapy that would effectively prevent development of epilepsy is not known.

OUTCOME IN HEAD INJURIES

Post - concussion syndrome, characterised by bothersome but non-specific symptoms, including dizziness, headache, and poor memory and concentration, is the most common sequala of head injury. There is no specific therapy. If there has been significant subarachnoid haemorrhage, resorption of cerebrospinal fluid will be impaired resulti in a communicating hydrocephalus. Other sequalae may be permanent or temporary cranial nerve dysfunction, resulting in anasmia, blindness, a other cranial nerve palsies.

Whatever the sequalae it is useful to evaluate the overall social outcome for the patient who has survived an episode of brain damage. The Glasgow Outcome Scale (Jennet 1979) enables this to be done in a simple way.

THE GLASGOW OUTCOME SCALE

5	Dead	
4	Vegetative Disabled	Sleep/wake but not sentinent
3	Severely disabled	Conscious but f dependent
2	Moderately disabled	Independent but disabled
1	Good recovery	May have minor requalae

Vegetative states refers to a survivor of brain damage, who is breathing spontaneously, but remains unresponsive and speechless. (Jennet 1977). Severe disability is due to a combination of mental and physical disabilit He is unable to get through the day without some sort of dependence on someone else. Moderately disabled people, have a wide range of handicap but are able to cope on their own, but at a reduced level. Good recovery does not necessarily mean complete recovery, but ability to undertake work and leisure activities similar to those previously possible.

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