A DISSERTATION OF TEN SURGICAL CONDITIONS SUBMITTED ON PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE AWARD OF THE DEGREE OF MASTER OF MEDICINE (SURGERY) OF THE UNIVERSITY OF DAR-ES-SALAAM

1991

BY

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This is to certify that the ten surgical conditions presented in this book were managed by Dr. Naboth A. A. Mbembati during his postgraduate training in General Surgery under my direct or delegated supervision.

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ABSTRACT

This dissertation conforms to the requirements of the award of Master of Medicine degree in Surgery of the University of Dar-es-Salaam. It consists of ten case studies on common problems in whose management the author has taken active participation. It is divided into four parts.

Part one is on general surgical problems and this includes Pseudocysts of the Pancreas, Rectal Prolapse, Surgical Cholestasis and Surgical manifestations of AIDS and HIV infection.

Part two consists of Thoracic Surgical problems which include Chest Injuries, Thoracic Empyema and Pericardial Effusion.

Part three deals with Urology and in this section Carcinoma of the Urinary Bladder is discussed.

Part four deals with Orthopaedics and Trauma in which ankle fracture dislocations and fractures of the spine are discussed.

In each topic a case is presented in detail, the rest of the cases are summarized, relevant literature is reviewed and the topic is discussed with particular references to observations made at Muhimbili Medical Centre.

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MANAGEMENT OF PANCREATIC PSEUDOCYSTS AT MUHIMBILI MEDICAL CENTRE

SUMMARY

From June, 1988 to July, 1990 a total of 6 cases of pseudocysts of the pancreas were managed by the author during his rotation as a resident in the three General Surgical firms of Muhimbili Medical Centre. An additional case managed in the department in 1987 is added to the study.

There were 5 males and 2 females giving a male to female ratio of 2.5:1. Five of the patients were below 50 years of age. The most common presenting features were abdominal pain, abdominal mass and weight loss. Only one patient had a preceeding history of trauma. In the rest no definitive history to suggest pancreatitis could be obtained. The diagnosis was mainly clinical and by ultrasound examination. Five patients were managed by transgastric cystogastrostomy, and two patients had cystocutaneous drainage. There were no deaths.

INTRODUCTION

A pancreatic pseudocyst is a localized collection of pancreatic secretions in the region of the pancreas in lacking an epithealia lining, and occurring as a a sequelae to pancratic inflammation.[1] More than three quarters of cystic lesions of the pancreas are pseudocysts.[2] The wall of the cysts is a fibrous wall

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and surrounds a collection of pancreatic juice with or without a blood clot and necrotic or suppurative pancreatic tissue.[1,2] In contrast true cysts of the pancreas are filled with fluid and have a definite epithelial lining.[2]

Most pseudocysts are unilocular, but multilocular cysts have been described. Pancreatic pseudocysts usually occur after acute and chronic pancreatitis and after trauma to the pancreas, but they may also occur following obstruction of the pancreatic duct.[3,4] In some cases, however, no cause can be established. In adults pancreatic pseudocysts are more common in males than females and these cysts have also been described in children in whom they occur following abdominal trauma.[3]

The diagnosis and management of pancreatic pseudocysts is changing over time due to availability of modern diagnostic facilities which allow early and precise diagnosis.

During a period of two years months from June, 1988 to July 1990 six cases of pancreatic pseudocysts were managed by the three surgical firms of Muhimbili Medical Centre. Going through previous medical records as far back as 1985 only one more other case was recorded bringing a collection of seven cases. A case is presented in detail as an index case and the other cases are summarized in the subsequent tables and review of literature on the problem is done.

CASE PRESENTATION

A.K. (Reg. No. 603719) a male patient, aged 25 years was admitted to the surgical ward on 17th March, 1990 with history of abdominal swelling of four weeks duration. He remembered having been kicked on the abdomen five weeks prior to date of admission and about a week later he noted a small mass in the upper abdomen which was slowly but progressively increasing in size. He had not attended any treatment for the kick in the abdomen as he had regarded it as a minor injury. He had no fever, no jaundice and had no vomiting. He had not lost weight although he had lately noted that he became full easily with small amounts of food. He had no pain in the abdomen. He took small to moderate amounts of beer.

Clinical examination revealed a young man who was afebrile, not pale and not jaundiced. His respiratory and cardiovascular systems were normal. Abdominal examination revealed a uniform epigastric mass about 15 x 10 cm in size. The mass was smooth, cystic, non-tender, non-mobile and non-pulsatile nor expansile. It was dull on percussion and there were no bruits on auscultation.

The liver, spleen and kidneys were not palpable. Genital and rectal examinations were normal. A clinical diagnosis of pancreatic pseudocyst was made.

The patient was investigated as follows.

1. Haemoglobin - 11.2 gm/dl
2. A plain abdominal X-ray was taken, but was non-remarkable. There was no evidence of pancreatic calcifications or calcified cyst wall.
3. Barium meal could not be done due to lack of barium sulphate at that time.
4. Serum amylase could not be done due to lack of reagents.
5. Ultrasound of the abdomen was done. This confirmed the presence of cystic mass in the region of the pancreas and the diagnosis of pseudocyst of the pancreas was entertained.

The patient was scheduled for surgery on 4th April, 1990, six weeks from the time of injury because the mass was noted to be increasing in size day after day during the period of hospitalization.

At surgery a huge cyst was found behind the stomach and the transverse colon was displaced inferiorly. On opening the stomach the mass was bulging into the stomach cavity from the posterior wall. Cystogastrostomy was done and about two liters of turbid brown fluid was sucked out from the cyst. A stoma about 5 centimetres in diameter was made between the cyst and the stomach walls which were adherent. Biopsy was taken from the thick cyst wall and marsupialization of the stoma was done. The stomach

was closed and the abdomen was closed in the usual way. The patient had an uneventful recovery and was discharged one week after surgery. He was seen at follow-up clinic three weeks later and he was in good general condition. Further follow-up could not be done as the patient had travelled to his home area in Mtwara. Histology of the cyst wall reported a haematoma in the gastric wall; there was no malignancy and no pancreatic tissue. Probably as a sampling error more of the adherent gastric wall adjacent to the cyst was involved in the biopsy specimen.

The salient features in the other cases are summarised in the following tables:

Table I Age and Sex Distribution

Age Group	Males	Females	Total
Under 20 yrs	0	1	1
21 - 30	3	0	3
31 - 40	0	0	0
41 - 50	1	0	1
Over 50 yrs	1	1	2
Total	5	2	7

Note: (a) The male to female ratio was 2.5:1

(b) Four out of seven patients were young, under 30 years of age.

Table II Clinical Features

Symptom/Sign	Number of Patients
Abdominal pain	6
Weight loss	4
Fever	3
Mass	6
Jaundice	2

Note: The commonest presenting features were abdominal pain, weight loss and an abdominal mass.

Table III Ultrasound Finding

Ultrasound Report	Number
Suggestive of cyst	5
Non-conclusive	1
Not done	1

Note: Ultrasound was conclusive in 5 patients

Table IV Volume of Cyst Contents

Volume	Number
Less then 500 mls	0
500 - 1000 mls	1
More than 1000 mls	3
Volume not stated	3
Total	7

Note: All cysts (where the volume was stated) were big with more than 500 mls of fluid

Table V Surgical Procedure

Procedure	Number
Cystogastrostomy	5
Cystocutaneous	2
cystojejunostomy	0
Total	7

Note: Cystogastrostomy was the commonest procedure done.

PATHOGENESIS

Pancreatitis and trauma are the most important causes of pseudocysts of the pancreas accounting for 75% and 25% of the cases respectively.[2,4] Other rare causes include obstruction of the pancreatic duct by neopasms and parasites. Occasionally no cause can be demonstrated.[2]

Pseudocysts contain fluid which contains all the usual pancreatic enzymes in high concentration and this fluid is mixed with serum, inflammatory debris and sometimes frank blood.[5] The fluid in the cyst comes from acinal cells of the pancreas. It may come from the major duct through a small rupture following necrosis in the surrounding parenchyma, but it is not easy to demonstrate this communication at surgery. Apart from the pancreatic secretions, water may also be attracted by osmosis from the high inflammatory protein content within the cyst adding more volume.[5] When not infected the fluid in the cyst evokes only a mild inflammation which results in a fibrinous reaction on the

surrounding surface and tissues. The fibrinous shell is in a few weeks converted into a fibrous capsule softened in some areas by fat necrosis. There is no endothelial secretory layer hence the term "pseudocyst" in contrast to true cysts of the pancreas.[4,5]

Acinar necrosis may be produced by blunt or penetrating pancreatic trauma, a preceeding acute pancreatitis or rarely behind carcinoma of the head of the pancreas.

The fluid usually collects within the "capsule" of the pancreas or in the lesser sac which becomes closed at the Foramen of Winslow by inflammation.[5]

The fluid collection which increases from continuous secretion from the main ductal system and other sources already mentioned causes increased pressure within the cyst. This collection continues to enlarge in the direction of least resistance until it meets counterbalancing resistance.[4,5] In due course it displaces the contiguous organs hence displacing the stomach anteriorly, superiorly, to the left or to the right. It may displace the transverse colon and the jejunum inferiorly or it may unfold the duodenal loop. Which organ is displaced and in what direction depends on the site of the cyst in relation to the head, body or tail of the pancreas, and also the relation of the cyst to the organ being displaced.

Most cysts ocur in the lesser sac, but they may occur in the pancreas, transverse mesocolon, behind the pancreas or in the mediastinum.[2]

Following acute pancreatitis there are five processes which are likely to cause a mass in the pancreas. These have been categorized by Glazen and Dudley[6] into:

(a) A pancratic pseudocyst which is a non-infected collection or effusion usually in the lesser sac. This may or may not communicate with the pancreatic duct.

(b) A pancreatic abscess. This is a localized collection of pus. It may arise de novo following acute pancreatitis or may develop from an infected pseudocyst.

(c) Pancreatic necrosis with liquefaction. The retroperitoneal space is filled with both pus and slough.

(d) Peripancreatic necrosis; where the gland is still alive and probably secreting so that the peripancreatic necrosis is progressive. This is often associated with multisystem failure.

(e) Pancreatic phlegmon in which there is an oedematous and inflammed pancreas with localized involvement of peripancreatic tissue.

A combination of clinical signs of infection or toxaemia combined with modern imaging techniques will allow the classification of some but not all of the patients in the above categories.

In post traumatic pancreatitis a pseudocyst can appear in one or two weeks after trauma, but it may be delayed

up to two or three months later. A collection of fluid in the lesser sac implies injury to a major pancreatic duct. When there has been a penetrating wound (gun shot wounds or knife wounds) and collection occurs early; the risk of infection, abscess formation and serious haemorrhage are increased; but if pseudocysts occur later the risk of secondary infection and vascular erosion is less.[5]

After blunt trauma the pancreas is usually injured by being crushed against the firm vertebral bodies in the midline. As the pancreas resumes secretions these pour in the midline haematoma, creating a cyst which enlarges slowly. The prognosis in such cases is good since the rest of the pancreas has otherwise been normal previously. Most pseudocysts of the pancreas in children are caused by blunt trauma to the pancreas.[3,5]

So in summary for occurrence of a typical pseudocyst of the pancreas there must be ductal rupture communicating with the potential space, an actively secreting pancreas and interference with normal ductal drainage.[5]

NATURAL HISTORY

The natural history of pancreatic pseudocyst is a function of time. Its understanding is important because it greatly affects the plan of management. With the advent of ultrasound and computed tomography scanning there has emerged a better understanding of the natural history of pseudocysts.

Ten per cent of patients with acute pancreatitis are likely to develop pancreatic pseudocysts.[1,3] Some of these will undergo spontaneous resolution. The resolution rate has been reported as ranging between 15 - 20% in most series.[1,3,6] Agha et al reported a 25% resolution rate

However, in a recent study done by Yeo et al[1] higher figures of spontaneous regression were reported. They found that among patients who were managed conservatively (which was 48% of all patients of pseudocysts) 60 per cent had complete resolution of the pseudocysts while 40 per cent had pseudocysts that remained stable or decreased in size.

These findings are very important as they enable one to plan as to whether to subject patients to surgical or conservative management. Some pseudocysts will however continue to increase in size until surgical intervention is done, while some may progress to complications. These include the following:

(a) Infection

Infection of the pseudocyst will lead to abscess formation. This will lead to fever and persistent leucocytosis.

(b) Obstruction or Pressure Effects

There maybe extrinsic compression onto the stomach leading to failure to tolerate oral intake; or there may be obstructive effects on the duodenum,

jejunum, colon or the biliary tree. Cysts may compress the common bile duct leading to obstructive jaundice.

(3) Spontaneous Rupture

This has been reported to occur in up to 5% of patients in one series.[9] Rupture may occur in the free peritoneal cavity leading to peritonitis. If a slow leak occurs pancreatic ascites may occur.

Rupture may also be transenteric into the stomach, duodenum, jejunum or the colon. These are common sites for cystoenteric communication. When cystoenteric rupture occurs the mortality increases to 50% due to associated gastrointestinal haemorrhage.[1]

Rupture may also occur into adjacent spaces or organs such as the pleural cavity, the portal vein, the oesophagus or the common bile duct.

One of our patients had cyst rupture into the stomach. He went into shock, and after resuscitation he had to be operated on emergency basis.

(d) Haemorrhage

Haemorrhage may complicate pancreatic pseudocysts.

Bleeding may arise from:

1. Oesophageal varices secondary to portal or splenic vein obstruction by the cyst.
2. Contiguous haemorrhage from the gastrointestinal tract.

3. Intracystic

4. Erosion of major arteries such as the splenic, or the gastroduodenal arteries.

Haemorrhage is the most lethal complication, occuring most commonly as a rupture of a pseudoaneurysm. The situation is made worse as clotting mechanisms has been found to be abnormal in a significant number of such patients.[5,6] The high fatality of haemorrhage has also been reported by Kiviluoto et al[10] who reported a mortality of 33% in patients who bled due to erosion and aneurysmal degeneration of splanchnic arteries on the wall of the pseudocyst.

CLINICAL PRESENTATION

Clinical evidence of a pseudocyst usually occurs 2-3 weeks after an attack of acute pancreatitis or trauma. Patients may present with abdominal pain, usually epigastric or on either side of the abdomen. There may be nausea or vomiting from ileus or mechanical obstruction.

Patients also complain of epigastric fullness and are unable to take adequate meals resulting in weight loss. Jaundice may occur if bile duct obstruction occurs.

Sudden rupture of the cyst may lead to shock, while a slow leak in the peritoneum may lead to pancreatic ascites. Secondary infection may lead to abscess formation with fever and leucocytosis. A mass may be palpable in 50% of the cases in the abdomen, or there

may be uniform abdominal distension even if a mass is present.[4,5] Sometimes a cyst can be completely silent, only to be detected by ultrasound.[10]

INVESTIGATIONS

(B) **RADIOLOGY**

(1) A plain abdominal radiograph may show a homogenous mass in the epigastrium or left upper quadrant. There may be obliteration of the psoas or/and renal shadows. It may also show pancreatic calcifications as evidence of chronic pancreatitis or a calcified cyst wall and may also show gall-bladder stones.

(2) A chest radiograph may show an elevation of the left hemidiaphragm and there is usually a pleural effusion in 20-35% of cases.[1]

(3) A barium meal or a hypotonic duodenogram may show an extrinsic mass displacing the stomach and small bowel in 77 -86% of cases.[4,5] The direction of the displacement will depend on the site of the cyst in the pancreas in relation to the displaced organ.

(4) Oral cholecystography may also show gall stones. But these can also be demonstrated by ultrasound scanning.

(B) **HAEMATOLOGICAL AND BIOCHEMICAL STUDIES**

(1) A white blood cell count may show leucocytosis if there is abscess formation.

(2) Serum amylase is elevated in 41-55% of cases,[5] but urinary amylase output expressed as units per hour is more sensitive and urinary amylase is raised in 80% of patients.[5] After the cysts are drained both serum and urinary amylase return to normal levels. Similar results have also been reported by Kiviuloto et al.[10]

(c) **ULTRASOUND**

This is at present one of the best ways of investigating diseases of the pancreas. It has also a very important impact on the investigation and management of pancreatic pseudocysts.[1,2,4,5,7] Its non-invasive nature enables it to be used to pick up even small cysts after an attack of acute pancreatitis and regular follow-up to see whether the cyst is increasing or regressing and hence decide on surgical or conservative management. One of the limitations is that the quality of the report is dependent on the operator and the equipment.[7]

(D) **COMPUTERIZED TOMOGRAPHY (C. T SCAN)**

This is also one of the recent advances in the diagnosis of pancreatic pseudocysts. It enables precise documentation of the size, location, number and wall-thickness of the pseudocysts.[1,7] It is very useful as it can be done repeatedly to document the progression of the size of pancreatic pseudocysts and decide on management plans.[1]

(E) **OTHER DIAGNOSTIC METHODS**

Endoscopic retrograde pancreatography has also been used to demonstrate pancreatic pseudocysts but its use is criticized because it may precipitate further attacks of pancreatitis.[4]

Angiography may be used in detection of pseudoaneurysms prior to surgery.

In our patients the diagnosis was made mainly clinically with ultrasound in 6 patients and Barium meal in two patients. The other patients did not have barium meal examination because they presented at the time when there was no barium sulphate. Likewise we could not estimate serum amylase due to lack of reagents. In the 6 patients who had ultrasound examination done, it was consistent with pseudocysts in 5 patients while it was non-conclusive in one patient.

MANAGEMENT

It is now generally agreed that the management of pancreatic pseudocysts may be conservative or surgical. In the surgical management there are three alternatives external drainage, internal drainage or excision.

The initial phase of management is usually conservative for two reasons:

(a) Conservative management is done in anticipation that spontaneous resolution will occur.

(b) Even if surgery becomes indicated conservative management is instituted while waiting for the

cyst wall to mature so that the patient can be fit for internal drainage which is associated with lower post-operative morbidity. Maturity of the cysts has been found to take about 6 weeks in human beings.[1]

The problem has usually been when to abandon conservative treatment and institute surgery.

Most authors seem to agree that large pseudocysts which persist longer than 6 weeks will probably not regress and therefore persistence of a cyst for longer than 6 weeks is one of the criteria for surgical intervention.[1,2,4,8,]

The size of the cyst has also been used as a factor for determining the need for surgery, so that cysts greater than 5 centimetres in the largest diameter have been considered an adequate indication for surgery.[1,4]

Prior to the advent of ultrasound and computerized tomography the diagnosis and subsequent assessment of patients was mainly clinical, with support of barium contrast studies. Spontaneous resolution was reported to occur in less than 25% of cases, hence the tendency was to subject more patients to surgery.

With the advent of computerised tomography and ultrasound studies, procedures that are non-invasive and can be done repeatedly, the behaviour of pseudocysts has been studied more closely and more precisely so that the plan of management can be done

more objectively. There has been arguments as to which of the two types of studies is more cost-effective. Beebe et al[8] in their study could not demonstrate any superiority of computed tomography over ultrasound scans or vice versa in the diagnosis and follow-up of patients with pseudocysts. Ultimately the most reliable and most cost-effective method will depend on availability of the equipment and individual experience and expertise at the institution.

Based on a study on the natural history of pancreatic pseudocysts followed up by computed tomography by Yeo et al[1] the authors suggested the following general plan of management:

(a) The initial step is verification of the presence of pseudocyst on a suspected patient by computed tomography or ultrasound.

(b) On confirmation of the diagnosis patients are discharged for outpatient follow-up if they satisfy all of the following criteria:

- They remain assymptomatic
- They can tolerate oral intake
- The abdominal pain subsides.

These patients are followed up with repeated computed tomography scans and continue to be managed conservatively provided they continue tolerating oral intake, they remain assymptomatic and the cyst remain stable in size, diminishes or disappears.

Using this programme of follow-up 52% of the patients required operative intervention. The median time period from onset of initial symptoms to date of operation was 75 days.[1] Forty-eight of their patients were treated conservatively. These patients were followed for a mean of one year, and of these 60% had complete resolution of the cysts while in the remaining 40% the cysts either remained stable or decreased in size.[1]

In summary, therefore the management of pseudocysts of the pancreas can be conservative or surgical. Indications for surgery include the following:-

- Non-resolution or increase in the size of the cyst after conservative treatment for more than 6 weeks.
- Persistence of pain related to the cyst or failure to tolerate oral intake.
- A cyst with greates diameter of more than 5 cm.
- Presence of complications which include infection, obstruction, haemorrhage or rupture.

SURGICAL PROCEDURES FOR PANCREATIC PSEUDOCYSTS

Surgery for pancreatic pseudocysts is not completely safe. A summary from seven studies give a mortality rate ranging from 6% to 16%.[5] Mortality seems to be

(c) Patients are operated upon if:

- They have abdominal pain believed to be related to pseudocysts
- They cannot tolerate oral intake
- The pseudocyst enlarges on CT Scan or ultrasound assessment
- The patient develops a complication necessitating surgery.

Following this regimen 48% of their patients were managed conservatively and 52% of the patients needed surgery. Among the patients who satisfied criteria for conservative management 60% had complete resolution of the cysts while in 40% the cysts either remained stable or decreased in size.[1]

So in summary the management of pseudocysts can be conservative or surgical. Indications for surgery include the following:

(1) Non-resolution or increase in size of the cyst after conservative treatment for more than 6 weeks.

(2) Persistence of pain related to the cyst or failure to tolerate oral intake.

(3) Presence of complications which include infection, obstruction haemorrhage or rupture.

SURGICAL PROCEDURES FOR PANCREATIC PSEUDOCYSTS

Surgery for pancreatic pseudocysts is not all that safe. A summary from seven studies give a mortality rate ranging from 6% to 16%.[5] Mortality seems to be

less for internal drainage than for external drainage. High risk is particularly related to associated liver disease and portal hypertension in alcoholic patients. Preoperative evaluation is necessary in all patients who need surgery:

- Liver function tests need to be evaluated including prothrombin time as vitamin K_1 will be necessary to correct it if it is prolonged.
- Plasma albumin may be low necessitating preoperative and postoperative intravenous albumin administration.
- Platelets counts should be done as there may be associated or a co-existent hypersplenism.
- Since the stomach and duodenum may be opened or the cyst infected pre-operative antibiotics should be considered especially in those few patients with obstructive jaundice.

The surgical alternatives are external drainage, internal drainage, and excision.

EXTERNAL DRAINAGE

This procedure is not preferred since it has high postoperative morbidity in terms of prolonged drainage, elecrolyte loss and a high recurrence rate.[10] It is reserved for: extremely ill patients requiring urgent drainage, infected cysts and pancreatic abscesses, and cysts needing drainage before maturity that is before 6 weeks.[2,4,5]

(B) **INTERNAL DRAINAGE**

Internal drainage of some type is the best mode of treatment for most pseudocysts provided they are mature. The fundamental principle is that:

1. The anastomosis must be made to a mature fibrous cyst wall.
2. Dependent drainage is desirable. Selection of a particular type of drainage depends on the site, size and adherence of the cyst to the stomach or small intestine. The alternatives:
    1. Transgastric cystogastrostomy when the lesion is adherent to the stomach.
    2. Transduodenal cystoduodenostomy for cysts located in the head of the pancreas.
    3. Cystojejunostomy to a Roux en Y loop of the jejunum, if the cyst is not adherent to the stomach or if it is broken into.[5]

A small piece of the wall of the cyst should always be taken for histology to exclude the possibility of a cystadenoma or cystadenocarcinoma.[2,4,]

Progressive closure of cystogastrostomy opening has been observed by repeated gastroscopy and has been noted to close in about 6 weeks and recurrences have been observed in 6% of cases.[5] Follow-up has revealed good results after internal drainage without significant difference between cystogastrostomy or cystojejunostomy.[5]

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(C) **EXCISION**

This would theoretically appear to be an ideal method, of treatment, but it is only feasible in the rare small pseudocysts located in the distal pancreas and unattached to adjacent vital structures.

OTHER METHODS

- Drainage through the duct of Wirsung after transduodenal sphincterotomy has been advocated by some authors based on the fact that pseudocysts have major ductal communication.[2] However the procedure is more difficult to perform and is less performed in view of the availability of simple procedures. The primary indication for transphincteric drainage is in patients with gallstone pancreatitis who are undergoing corrective biliary tract surgery.[2]
- Percutaneous drainage has been reported using catheters placed under computed tomography or ultrasound guidance. This seems to be a new method which is becoming popular and has been reported by various authors.[11, 12]

In our patients five were treated by transgastric cystogastrostomy and two patients had cystocutaneous drainage. One of the patients who had cystocutaneous drainage had a pancreatic abscess.

One patient had emergency cystogastrostomy following spontaneous cystoenteric rupture leading to shock. He

was found to have double cysts both of which were drained into the stomach.

CONCLUSION

Pancreatic pseudocysts though uncommon continue to occur in our hospital. A definite history of preceeding acute or chronic pancreatitis seems to be difficult to obtain; but it seems that there are more post-pancreatitis pseudocysts than those following trauma.

With the availability of ultrasound facilities it should be possible to provide adequate objective and precise management to such patients by initial documentation of the presence of the pseudocyst, following the patients adequately during the period of conservative management with repeated ultrasound examination and providing surgical therapy when indications dictate it.

Ultimately patients who have pseudocysts post-alcoholic pancreatitis should be persuaded to stop taking alcohol otherwise recurrence of the cysts is not uncommon.

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RECTAL PROLAPSE AS SEEN AT MUHIMBILI MEDICAL CENTRE

SUMMARY

Between January and September, 1990 6 patients with rectal prolapse were seen and managed in surgical Firm I of Muhimbili Medical Centre. There were 5 males and one female giving a male to female ratio of 5:1. The age range was 22 to 70 years, with 4 of the patients being below 45 years of age.

Three cases presented as emergencies with irreducible rectal prolapse. Two of these were managed by emergency perineal proctosigmoidectomy, the third patient was initially managed by manual reduction of the prolapse, but he died of hypertensive crisis as he was being planned for definitive surgery.

Of the three other patients one patient had anterior sigmoid resection and proctopexy, one had anterior suspension and sacral fixation with marlex mesh and the third one had perineal proctosigmoidectomy.

The results in four patients who were operated upon were good with satisfactory continence of stool and flatus. One patient who had an emergency proctosigmoidectomy developed peritonitis which required a laparotomy, peritoneal lavage and a defunctioning colostomy.

INTRODUCTION

Rectal prolapse is an uncommon problem, but when present it is an embarrassing and disabling problem to

the patient and poses a challenge to the surgeon in terms of management. Both the patient and the surgeon frequently remain dissatisfied because of persistent incontinence, bowel management problems and/or recurrence.[1]

It is difficult to determine the actual overall incidence of rectal prolapse as few authors address themselves to the problem. Literature on rectal prolapse in Africa is scarce although Badoe has described the problem both in children and adults.[2]

From January to September, 1990 the author saw a total of 6 cases of rectal prolapse who were admitted in Surgical Firm I of Muhimbili Medical Centre. Three cases presented with irreducible acute or acute-on-chronic rectal prolapse necessitating emergency management and three cases were managed electively. There were five males and one female giving a male to female ratio of 5:1. One of the cases is described in detail and the other cases are summarized in a table. The topic of rectal prolapse is then discussed in detail with review of relevant literature.

CASE PRESENTATION

M. F. (Reg. No. 603116), a female, aged 70 years was admitted at Muhimbili Medical Centre on 10th March, 1990 with complaints of a mass protruding per rectum

and a mass protruding per vagina all of two years duration.

The patient had been well until two years ago when she noted that whenever she strained at defaecation there was a protrusion of the rectum as well as a mass protruding per vagina. These however, reduced spontaneously. In the past few months prior to admission she had noted that the prolapse appeared whenever she stood up, even without straining and that she had to reduce them manually with her fingers. She also experienced recurrent episodes of loose motions and had lately become incontinent with constant soiling of her underwear either with stools or mucus. She also complained of incontinence of urine on straining. She denied any history of chronic cough or previous perineal operations. She had two normal deliveries and was now postmenopausal for more than 20 years. Her twin sister had suffered from a similar problem and had required surgery.

Clinical examination revealed an old lady who was in good general condition. She was not pale and not wasted. Her pulse and blood pressure was normal. The respiratory and cardiovascular systems were normal. Abdominal examination was also normal.

Perineal and genital examination revealed complete uterovaginal and rectal prolapse. Both the rectum and uterus were easily reducible. The anus was patulous with poor anal grip and squeeze tone. A diagnosis of

complete (3rd degree) rectal and uterovaginal prolapse was made.

After discussion with the gynaecologist it was decided to deal with the rectal prolapse first. In view of her age she was planned for perineal proctosigmoidectomy. Bowel preparation was done with total bowel irrigation on the day prior to surgery and intravenous cefuroxime 1 gram and metronidazole 500 mg at induction on the day of surgery.

The patient was placed in lithotomy position and was cleaned and draped. Stay sutures were placed at 3,6,9 and 12 o'clock 2 cm from the anal verge on the prolapsed rectum. A circular incision was made 2 cm from the anal verge just proximal to the stay sutures and deepened through the muscular layer and peritoneum. The incision was extended circumferentially and on the posterior aspect the vascular pedicle in the mesentery was lifted and transected. The sigmoid colon was then pulled inferiorly to the maximum and the redundant sigmoid was amputated after application of 4 stay sutures corresponding to those applied in the distal stump. The peritoneum was then apposed to the anterior and lateral aspects of the sigmoid colon. End to end anastomosis between the sigmoid colon stump and the rectal stump was done in one layer with 2/o interrupted silk sutures using the stay sutures as guides.

Post-operatively the patient continued with intravenous Gentamycin 80 mg 8 hourly for 72 hours. The post-operative period was uneventful. The patient was continent of both stool and flatus, but she still had occasional episodes of loose motions. Her anus was still patulous and she could hardly grip a finger in the anus.

Four months later she had a vaginal hysterectomy done and she was finally discharged home free of both rectal and uterogavinal prolapse on 6.9.90. Her preoperative condition is illustrated by photographs overleaf and the procedure is also illustrated. The other patients are summarised in the subsequent tables.

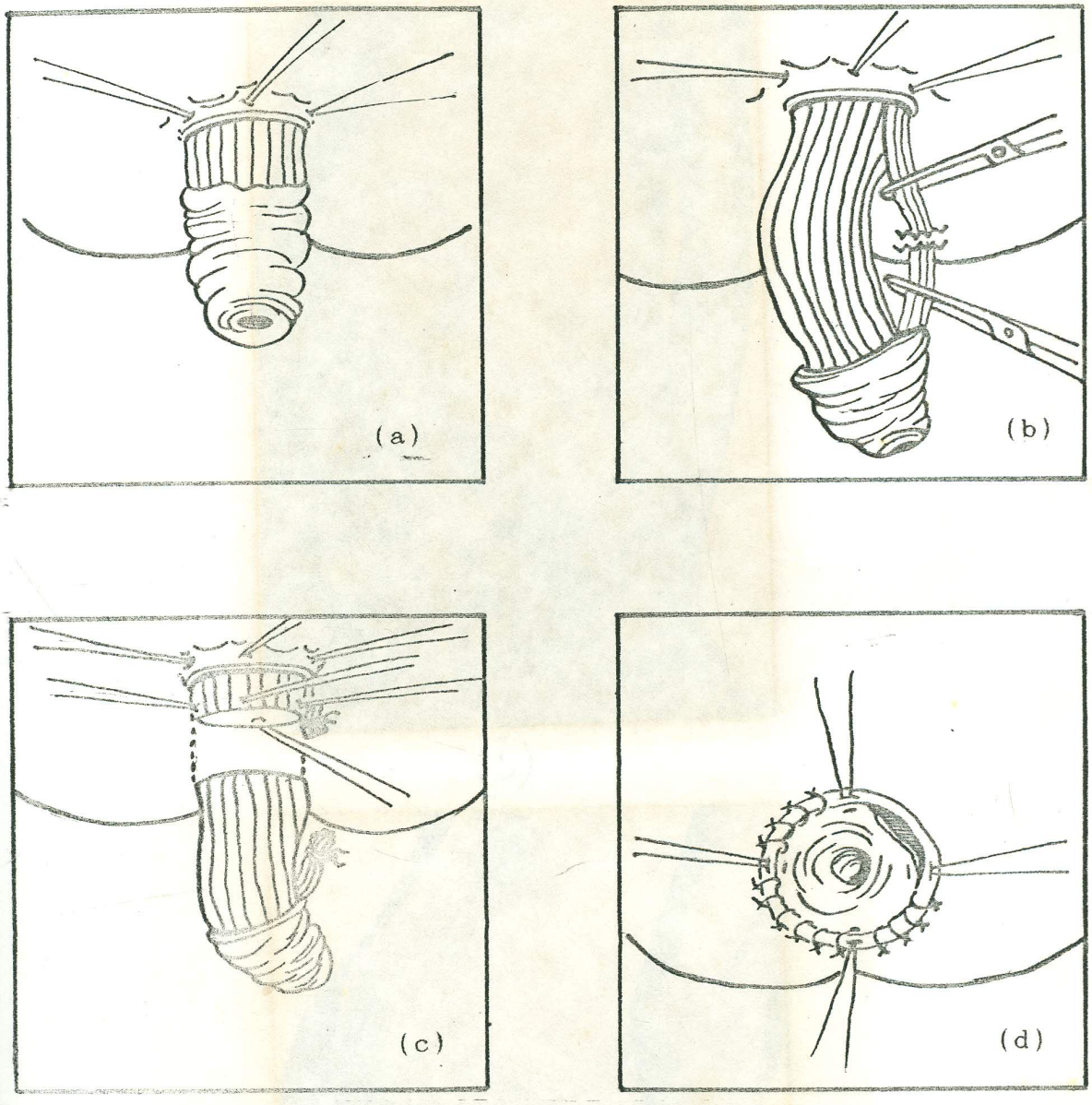
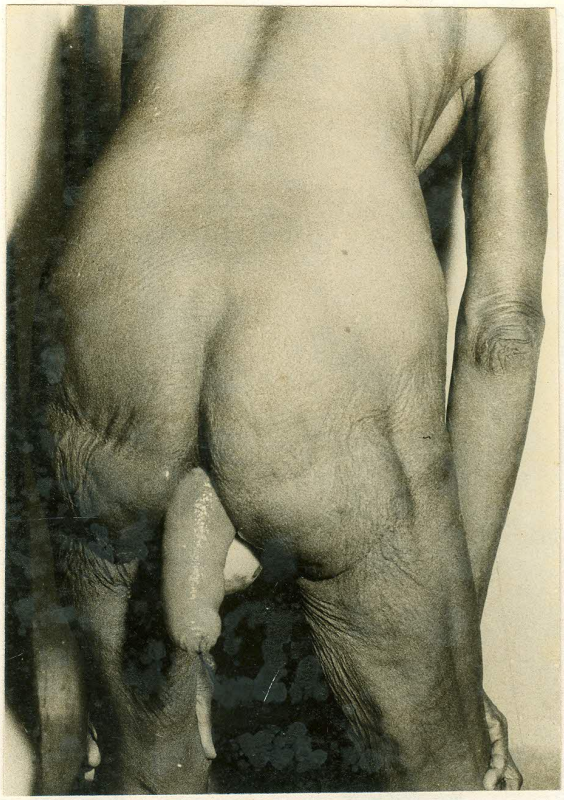
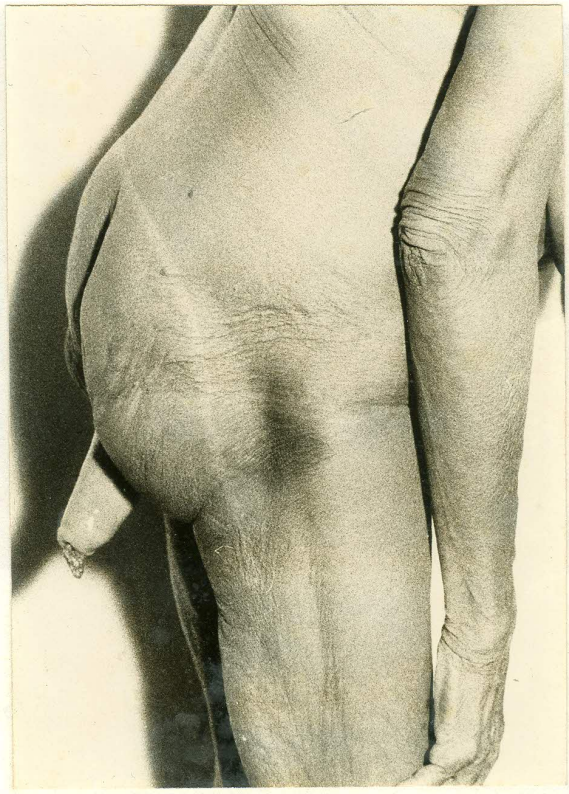


Fig. 1: Steps in Perineal Proctosigmoidectomy.

a - Circular incision is made 2 cm proximal to dentate line.
b - The prolapsed sigmoid colon is pulled to the maximum and mesentery is clamped and divided.
c - The peritoneum is resutured and the sigmoid colon is excised.
d - Anastomosis is made using one layer interupetd silk.



(i)



(ii)



(iii)



(iv)

i-iii - Posterior, lateral and anterior view of rectal prolapse in the index case.

iv - One of the patients with acute rectal prolapse showing oedema and areas of necrosis

Table I: **Age and Sex Distribution**

Age Group	Males	Females	Total
Under 20 years	0	0	0
21 - 30	2	0	2
31 - 40	1	0	1
41 - 50	1	0	1
51 - 60	1	0	1
Over 60 years	0	1	1

Note: (a) The male to female ratio was 5:1

(b) Four of the five males were under 40 years of age

Table II **Treatment Modalities**

Method of Treatment	Emergency	Elective	Total
Manual Reduction	1	0	1
Perineal Proctosigmoidectomy	2	1	3
Anterior Sigmoid Resection and Proctopexy	0	1	1
Anterior Suspension and sacral fixation with marlex mesh	0	1	1
Total	3	3	6

Note: (a) The patient who had manual reduction died of hypertensive crisis as he was being planned for definitive surgery.

(b) One of the emergency proctosigmoidectomies developed an anastomotic leak necessitating a laparotomy and defunctioning colostomy.

DISCUSSION

Prolapse of the rectum is an uncommon but disabling problem in which the rectal wall turns inside out into and through the anal canal.[3,4] The disease is common in old females with peak age incidence of 60-70 years. In males the disease is evenly distributed. The disease is more common in females with a female to male ratio of 5-10:1.[3] The same ratio was found by Watts et al[1] who had 163 females and 16 males in his series giving a female to male ratio of 10:1. However in this series at Muhimbili there were 5 males and one female giving a male to female ratio of 5:1. The number of patients is too small to make any inferences; but it is noteworthy that with the exception of one patient all male patients were young with ages ranging from 22 to 44 years.

Rectal prolapse is also common in children in whom it is sometimes associated with malnutrition, whooping cough, chronic diarrhoea and infestations with strongyloides stercoralis.[2]

In this series children are not included as they are managed by the paediatric surgical unit.

Another related symptom is the descending perineum syndrome which may precede or co-exist with rectal prolapse.[4] In these patients the perineum is abnormally low and most of such patients constantly strain at defaecation. This results in deterioration in the tone of plevic floor muscles and subsequently

descend of the whole pelvic diaphragm occurs.

This condition is more common in young patients and has been found to be present in up to 10% of patients attending rectal clinics in the United Kingdom.[4]

At Muhimbili Medical Centre there is no published literature on the magnitude of the problem, but Hiza[5] reported 7 cases of rectal prolapse being admitted in the hospital in the year 1971.

It is interesting to note however, that among the patients reported in this series two patients have been attending this hospital since 1987, and all the time they have been managed conservatively until this year when they were offered surgical treatment.

Aetiology

There is no single theory that satisfactorily explains the cause of rectal prolapse. Ripstein[6] suggests that there is failure of structures surrounding the rectum to provide adequate support. Goltberg[3] describe various predisposing factors in rectal prolapse which include:

- Diastasis of the levator ani muscles
- Loose endopelvic fascia
- Unusually deep rectouterine or rectovesical pouch.
- Loss of the normal acute anorectal angle which becomes relatively obtuse.

The anorectal angle is maintained by the puborectalis muscle which functions by elevating the lower end of the rectum upwards and forwards towards the pubis. This creates an acute anorectal angle and provides support during straining.[7] Relaxation of the puborectalis sling results in descent of the pelvic floor, obliterating the anorectal angle so that the rectum becomes vertical.[7]

Rectal prolapse may also follow operative procedures such as haemorrhoidectomy, fistulectomy and abdomino-anal pull though, where the sphincter muscle has been divided.[7] Atony of the anal sphincter may be idiopathic or may be secondary to nervous system disorders such as myelitis, cauda equina lesions or tumours.[2]

Though all these factors are mentioned as predisposing factors surgical correction of such factors does not result in cure of the problem and recurrence is common. Thus their actual role in rectal prolapse is difficult to determine.

The other concept which has been controversial is whether rectal prolapse is a sliding hernia, an intussusception or a combination of the two.[1]

Moschowitz in 1912 described rectal prolapse as a sliding hernia through the levator hiatus. He therefore proposed repair of the levator hiatus and obliteration of the cul-de-sac as an important part of

treatment. However, there were recurrence rates up to 48% thus casting doubt on the theory of rectal prolapses being a type of hernia.[1,4,6]

Broden and Snellman[6] by cineradiographic studies demonstrated that rectal prolapse is not a sliding hernia but an intussusception of low and mid-rectum. It was found that in all cases rectal prolapse was initiated by intussusception of the rectum at 6-8 centimetres from the anal verge. A possible sequence of events could be that as a consequence of denervation of the puborectalis (which has been demonstrated in most cases) there is disruption of the anorectal angle. This favors prolapse of the anterior rectal wall into the anal canal, and sets up an intussecepting process that results into extrusion of the rectum through the anal canal.[4,6,11] So the "predisposing" or "associated" factors which include diastasis of the levators, deep cul-de-sac, patulous anus and redundant sigmoid could actually be the results of rectal prolapse rather than its cause.[1,4,6]

The cause of the neuropathic process is still unknown. In some women it could be due to local nerve damage during parturition while in others it may be due to stretch-induced injury related to the descending perineum syndrome. In most cases the cause remains unknown.

Pathophysiology

It is generally believed now that rectal prolapse is an intussusception of the rectum into and through the anal canal.[1,4,6] The theory that rectal prolapse is a hernia has been discarded. The process of intussusception has been described already. As the rectum prolapses it produces further stretching and paralysis of the external sphincter muscles resulting in a wide and patulous anus. There is an elongation and redundancy of the rectosigmoid and sigmoid colon which is thickened, heavy and distorted. The prolapsed rectum is oedematous, hyperaemic and often superficially ulcerated.[8]

Sun et al[10] studied patients with anterior mucosal prolapse, full-thickness rectal prolapse and solitary rectal ulcer. These conditions have been found to be co-related. Solitary rectal ulcer is thought to occur when the redundant rectal mucosa is traumatized and becomes ischemic within the anal canal. Anterior mucosal prolapse may initiate intussusception and progress to full thickness rectal prolapse. So, a common pathophysiology was postulated.

In their study on normal subjects and patients Sun et al[10] found that when normal subjects increased their intra-abdominal pressure in order to blow up a baloon

there was a compensatory increase in external sphincter activity which was accompanied by a rise in anal pressure above the rectal pressure, thus effectively preserving continence. In the patients, however, there was enhanced electrical discharge in the external sphincter muscle, but there was no associated increase in anal pressure above the rectal pressure as in normal subjects. This created conditions that would favour rectal prolapse and anal incontinence. In 61% of the patients there was actually leakage of fluid during the act of increased intra-abdominal pressure.

A similar pattern of results was seen in rectal distension. All patients and normal subjects showed an increase in external anal sphincter activity. But the pressures generated during rectal distension were lower in patients than in normal subjects. In the patients anal pressures were lower than rectal pressures thus favouring incontinence and prolapse. Patients also perceived a desire to defaecate at much lower volumes (of rectal contents) than normal subjects and were more likely to develop rectal contractions in response to rectal distension causing rectal pressure to rise above anal pressure hence leading to incontinence. The increased sensitivity of the rectum may encourage the subject to start to strain when only small volumes of stool are present in the rectum thus predisposing to

rectal prolapse into the anal canal.

So from their results the authors concluded that anterior mucosal prolapse, full-thickness rectal prolapse and solitary rectal ulcer represent different stages of the same basic condition. This is associated with very weak anal sphincter and an abnormally irritable rectum. The weakness in the anal sphincter is thought to be due to pudendal neuropathy as all the patients had abnormal perineal descend. There is also weakness of internal sphincters which is thought to be due to traction effect on the delicate sympathetic nerve supply.[10]

Clinical Features

Patients with rectal prolapse usually presents with history of a mass prolapsing per rectum; initially during defaecation, but later on spontaneously. There is often an associated history of faecal incontinence with soiling of underwear; although some patients may present with constipation.[3,6] Patients may have anorectal pain or discomfort at defaecation. There may be a feeling of incomplete evacuation. Sometimes the prolapse may present as an emergency with strangulation or rupture.[6] This occurred in one of our patients who had gone to toilet and on defaecation the whole rectum suddenly prolapsed and became irreducible. This was the frist time the patient had rectal prolapse. Sometimes mucosal prolapse may be associated with haemorrhoids and polyps.[7,9] In old females rectal prolapse may be

associated with uterovaginal prolapse[7] as occurred in the only female patient in this series. Sometimes the patient may be unaware of the problem which may be discovered during physical examination, and at other times prolapse is suspected but cannot be demonstrated due to constraints of the examination couch. Such patients should be examined straining in a squatting position.[6]

Sigmoidoscopic examination may reveal traumatic proctitis which may be confused with inflammatory bowel disease. Extensive circumferential prolapse in the lumen may be noted.[4] Digital rectal examination confirms poor anal sphincter tone with a patulous anus. Evacuation defaecography can be done in mild cases and this will demonstrate the prolapse.[11]

MANAGEMENT OF RECTAL PROLAPSE

The management of rectal prolapse is difficult and often both the patient and the surgeon are not satisfied with the results because of recurrence, persistent incontinence or bowel management problems.[1] Various modes of treatment have been attempted and more than 50 types of operations have been designed for rectal prolapse.[12] This simply shows that no single treatment modality has as yet been completely satisfactory. Most surgical techniques are a variation of a few basic modes of therapy; and which modality is

used depends on the surgeon's concept of the anatomic defect.[12]

In the era when the concept of prolapse as a hernia was held high treatment aimed at narrowing the anal orifice, obliteration of the peritoneal pouch of Douglas and restoration of the pelvic floor muscles in addition to suspension and fixation of the rectum to the sacrum with resection of the redundant loop of bowel.[3] Nowadays since the theory of hernia has been discarded most treatment modalities do not deal with repair of the cul-de-sac nor restoration of the pelvic floor.

The other factor which influences the type of management is the stage of the prolapse. Griffen et al[13] describe three types or stages of rectal prolapse.

Type I = Mucosal prolapse

Type II = Incomplete protrusion of the descending rectum with a sulcus between the anus and the descending part of the rectum.

Type III= Complete rectal prolapse.

In type I rectal prolapse one would probably be more conservative while in Type III definitely some form of surgery is indicated. In type II other factors such as age, might also influence the type of treatment to be offered.

In the young patient who generally have mucosal prolapse a conservative approach is usually adopted such as injection therapy with other measures to improve bowel function. This is because the condition often resolves spontaneously.[6] If conservative measures fail surgery becomes necessary but even then perineal procedures are preferred to abdominopelvic procedures so as to avoid the risk of damaging the autonomic nerves serving sexual function or to cause infertility in females by instigating pelvic fibrosis.[6] In older age groups surgical treatment is often adopted since there is little to be gained by adopting conservative management.

Conservative Management

In children rectal prolapse is sometimes associated with or occurs as a complication of other diseases such as whooping cough or chronic diarrhoea. These underlying conditions must be treated and meanwhile repeated manual reposition of the prolapsed rectum may be sufficient until the underlying condition improves. In young adults conservative management is advised for reasons mentioned before. These include:

- subcutaneous injection of a sclerosing agent such as phenol in almond oil.[2,3,6]
- Linear cauterization of the anal mucosa.[2]

Surgical management

The objectives of surgical procedures have been changing over time depending on the surgeon's concept as to the cause of rectal prolapse. The objectives also depend on the stage of the prolapse whether it is mucosal, partial or complete rectal prolapse.[13]

At present especially in complete rectal prolapse the objectives of surgery according to Corman[12] include:

(a) Resection of the redundant bowel which may be done trans-abdominally, perennially or trans-sacral.

(b) Suspension or fixation of the rectum to the sacrum, pubis or other structures.

(c) A combination of the above.

The Thiersch Repair

This procedure is used in the old poor risk patients and it can be done under local anaesthesia.[12] In this procedure a silver wire or Teflon or Marlex suture is placed subcutaneously around the anus. A small incision is made in the anterior and posterior position 1 cm outside the anal verge. A suture of the selected material is passed from anterior to posterior direction on either side of the anus in the ischiorectal fossa and tied posteriorly and the knot is buried. The anus is reduced to the size of a Hegar 16 or 18.[2,6,7,12]

This procedure is now being discarded because of complications which are very common and these include:

- Suture fracture if silver wire is used
- Faecal impaction
- Wound infection
- Recurrence of rectal prolapse

There are other Thiersch-type operations but the main difference from the original Thiersch procedure is the type of material used. Apart from low wound sepsis rate the rate of complications is the same.[12]

The Delorme procedure

In this procedure described by Delorme in 1900 the mucosa overlying the prolapsed rectum is excised all around from the anorectal junction to the apex of the prolapse. In a modification done by Monson et al[14] the mucosa excision is extended in the lumen of the prolapsed segment as high as possible. Plicating sutures of number 1 chronic catgut are inserted picking up the distal mucosal cuff, then through the muscular layer, taking generous bites and finally the sutures pick up the proximal mucosa cuff. A series of about 12 sutures is used. When these are tied the rectal prolapse is reduced to a bulbous plug in the pelvis.[14] A recurrence rate of 7-11% has been reported, but the procedure is well tolerated and patients are fully mobile, taking a normal diet within 48 hours. The procedure can be repeated in case recurrence occurs.[14]

Perineal proctosigmoidectomy

This procedure was first described in 1952 and is a single stage perineal procedure.[7] It can be performed in the lithotomy or jacknife position. The prolapsed rectum is exteriorized and a circular incision is made 2 cm. proximal to the anal verge. A circumferential full-thickness incision is made through the outer cylinder of the bowel. The rectosigmoid is mobilized until the redundant bowel cannot be pulled down any further. Then the inner cylinder of the bowel (the sigmoid colon) is amputated and end to end anastomosis is made either with interrupted sutures or with a stapling device.[3,7] This procedure is however associated with recurrence rates varying from 2.8% to 60% depending on the duration of post-operative follow-up.

This procedure was done in 3 patients in this series, two of whom had acute irreducible prolapse and had to be operated on emergency basis. One of them developed anastomotic leak with peritonitis and had to undergo a laparotomy for peritoneal lavage and transverse defunctioning colostomy.

Rectal Suspension

Rectal suspension or rectopexy is based on the concept that the main abnormality in rectal prolapse is an intussusception of the rectum and not weakness of the pelvic floor muscles. In this operation the rectum is

mobilized down to the levator ani muscles. The mesentery of the rectum and the muscularis are secured to the sacral curve.[1,12,15] Fixation may be by simple interrupted non-absorbable sutures, strips of fascia lata or omental pedicles. Recurrence rates of 4.3% at 5 years follow up have been reported by Corman.[7] None of the patients in this series was treated by this method.

Rectal Suspension with Implants

Rectal suspension can be achieved by implanting foreign material.

In the Ripstein procedure a sling of Teflon or Marlex mesh is used to attach and suspend the rectum to the sacrum. This probably the commonest procedure used for treatment of rectal prolapse in the USA.[12] The teflon sling is secured on one side of the sacrum and then with the mobilized rectum held under tension the mesh is sutured to the muscularis of the bowel. It is then resutured to the sacrum on the other side thus encircling the rectum but leaving a defect of about one centimetre posteriorly.[4,12] This procedure is often complicated by sling obstruction and faecal impaction.[1]

In the Well's procedure a sheet of Ivalon sponge wrap of Polyvinyl alcohol, marlex mesh or Teflon mesh is used. The mesh or sponge is fixed to the sacrum in the middle leaving two leaves which are attached to the mobilized rectum on each side, leaving about 25% of the

rectal circumference free anteriorly. This avoids the problem of stenosis, faecal impaction and sling obstruction as seen in the Ripstein procedure.[1,4] Corman[12] reports a recurrence rate of 2.3% and this is related to faulty surgical technique. Faecal impaction may also occur and it could be due to too tight application of the mesh.[7,12]

One of the patients in this series was managed by anterior suspension with sacral fixation by Marlex mesh.

Bowel Resection: Abdominopelvic procedures

This is another method of treatment for rectal prolapse where either the sigmoid colon is resected followed by proctopexy or low anterior resection is performed.[1] The original authors of this approach; Fykmen and Goldberg argued that "of all the weaknesses or abnormalities required to produce rectal prolapse the only factor which can be controlled with certainty is the length of the colon".[1] It is also argued that resection will prevent early recurrence while the mobilized rectum is becoming firmly adherent to the sacrum by fibrosis. Another advantage of resection is the removal of the redundant sigmoid colon which may predispose to sigmoid volvulus.

Resection of the sigmoid can also be combined with sacral fixation of the distal segment (rectopexy). The posterior rectal wall or the intact lateral ligaments

of the rectum are secured to the sacrum by non-absorbable sutures. Usually when resection is done it is safer to avoid implants of foreign material for sacral suspension such as Marlex mesh or Teflon for fear of infection.[1] Recurrence rates after bowel resection have been reported to be as low as 1.9% by Watts and as high as 9% in another report by Corman.[12] In this study one of the patients was managed by anterior sigmoid resection and proctopexy. He had a good outcome.

Conclusion

From the observation that two patients had been coming to hospital since 1987 and being managed conservatively it seems probable that rectal prolapse may be more common in our community than is being documented in operation records.

From the experience gained with the patient who had an anastomotic leak following emergency proctosigmoidectomy for acute irreducible rectal prolapse there is a lesson to learn. In a patient with an acute irreducible prolapse of the rectum with signs of impending gangrene one may consider performing a defunctioning transverse colostomy as a protective procedure. This would have to be done prior to performing the perineal proctosgimoidectomy and it would then be closed when the anastomotic site has healed.

The patients managed in this study benefited from surgery apart from the one who had an anastomotic leak. They were all free from prolapse and were continent. However the small number of the patients and the short period of postoperative follow up limits further comment on recurrence or complications.

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SURGICAL CHOLESTASIS IN ADULTS AS SEEN AT MUHIMBILI MEDICAL CENTRE

SUMMARY

From January, 1989 to June, 1990, 8 cases of surgical cholestasis were managed in the three surgical firms of Muhimbili Medical Centre. There were 5 males and 3 females with age ranging from 33 years to 51 years. Three patients had cholelithiasis with choledocholithiasis, two patients had carcinoma of head of the pancreas and one patient had bile duct carcinoma. One patient had jaundice following cholecystectomy and in one patient the cause could not be determined. The procedures done in the management of these patients included cholecystectomy with common bile duct exploration (three patients) cholecystojejunostomy (two patients) choledochojejunostomy (one patient) and cholangiojejunostomy in one patient. In the last patient only exploration was done.

Investigations of the patient with cholestatic jaundice are discussed with a brief review of literature on the common causes of cholestasis as seen at Muhimbili Medical Centre.

INTRODUCTION

Cholestasis is defined as failure of normal amounts of bile to reach the duodenum.[1]

Patients with surgical cholestasis usually present with jaundice, passage of dark frothy urine, pale stools, pruritus and hepatomegally.[2]

When presented with a jaundiced patient the clinician is faced with several problems, among which is to decide whether the cause of jaundice will need surgical intervention or not.[3] Even when the clinical diagnosis of cholestatic jaundice is made, implying that some surgical intervention may be necessary there is still the question of defining the anatomical site of the problem and its pathology.[4] After anatomical and pathological diagnosis the problem of management needs to be addressed in terms of the type of surgery and whether one is planning a curative or palliative procedure. This is particularly true in patients with malignant disease. So, in general cholestasis is a challenging problem right from the time of diagnosis, pre-operative preparation of the patient and actually in planning treatment best suited to the individual patient. In this paper a case of cholestasis is presented in detail and the management of patients with cholestasis is discussed with review of literature on the most common causes as seen at Muhimbili Medical Centre, Dar es Salaam Tanzania.

CASE PRESENTATION

J.B. (REG 562820) a male teacher aged 49 years was admitted in Muhimbili Medical Centre in February, 1990 having been referred from an upcountry hospital. He presented with yellow colouration of the sclera associated with itching of the body all of 4 months duration. He had noted yellow colouration of eyes which was associated with passing of dark urine 4 months prior to the date of admission. The jaundice was progressive without remission. This was soon followed by itching of the whole body which did not respond to medicines which he was prescribed in hospital. He was passing clay-coloured stools. He had no abdominal pain and had no fever. He had not been given any blood transfusion nor had he been given any parenteral drugs in the past. He had not had any previous abdominal surgery. He had lost 10 kg of his body weight during the four months of illness. He used to drink two to three litres of alcohol occasionally but did not smoke. No other family member or village-mate had similar complaints at the time.

Clinical examination revealed a middle aged man who was deeply jaundiced. He was afebrile with a temperature of 36.5°C. He had scratchmarks all over the skin. He had no tremors, spidae naevi, caput medusae nor palmar erythema. He had normal breathing.

Abdominal examination revealed a soft abdomen which was not distended and non-tender. The liver was palpable 7 cm. below the right subcostal margin along the right mid-clavicular line. It was smooth with a sharp margin and it was tender. The gallbladder and the spleen were not palpable; and there was no ascites. Rectal examination revealed no masses and the examining gloved finger was stained with clay-coloured stool. The respiratory and cardiovascular systems were normal.

A provisional diagnosis of extrahepatic cholestatic jaundice was made.

Investigations done included:

1. Haemoglobin 8.8 gm/dl
2. Urinary bilirubin was strongly positive. Urobilinogen in urine was negative.
3. Serum bilirubin Total 200 millimoles/Litre
   Direct 144 millimoles/Litres
4. Serum Alanine Aminotransferase (ALT) 82 i.u./L
5. Serum Aspartate aminotransferase (AST) 80 i.u./L
6. Serum Alkaline Phosphatase (ALP) 600 i.u./L.
7. Prothrombin time was 28 seconds which was 20% of the control.

Ultrasound scan of the abdomen was done and this revealed gross dilatation of the intrahepatic biliary system. No stones were seen. The gallbladder common bile duct and pancreas were reported as being normal.

A percutaneous transhepatic cholangiogram was recommended; but it could not be done due to lack of contrast media.

In view of the prolonged prothrombin time the patient was given Vitamin K_1 10 mg intramuscularly daily and after a week the prothrombin time was 17 seconds which was 46% of the control. After another week of Vitamin K_1 the prothrombin time was 12 seconds, 85% of the control. He was also given one litre of blood and the control Haemaglobin was 11.4 gm/dL. Other tests which were repeated included:

1. Serum bilirubin: Total 267 millimoles/L
   Direct 188 millimoles/L
2. Serum alkaline phosphatase 853 i.u./L

As the prothrombin time now had improved to near-normal it was decided to operate on him.

The abdomen was opened through a Right Kochers incision. The intra-operative findings were:

- an empty gallbladder
- a tumour in the common hepatic duct extending proximally to the porta hepatis.
- A nodular liver especially the inferior surface - probably from hepatic metastasis.

Since the tumour was inoperable it was decided to perform a bypass procedure to relieve the jaundice. A wedge biopsy was taken from the tumour. Then through

finger fracturing of the liver tissue distended bile ducts were exposed one from the right and one from the left lobe of the liver. These ducts were cleared of hepatic parenchymatous tissue for a considerable distance and each was stented with a small feeding tube size 12. A loop of jejunum was brought up and the two stented bile ducts were anastomosed to the jejunum. The stents were brought out through different openings and were brought out through the left and right lumbar regions and transfixed on the skin. A drain was placed in the subhepatic space and the wound was closed.

The patient was put on intravenous ampicillin 500 mg 6 hourly and gentamycin 80 mg 8 hourly all for 48 hours. He also continued with intramuscular vitamin K1 daily for two more weeks.

On the fifth post-operative day the rubber drain removed and the stents were removed 2 weeks later. The patient started oral intake and the intravenous line was removed. A repeat serum bilirubin 48 hours later had fallen to 240 millimoles (total) and the direct bilirubin to 180 millimoles/litre. Two weeks post-operatively the bilirubin total was 130 millimoles/L and direct was 90 millimoles/Litre.

The jaundice had subsided and the stool regained the brownish colour and itching abated. He was finally discharged home one month post-operatively in good condition. He has not been seen for follow-up.

Histology result from the biopsy of the tumour revealed adenocarcinoma of the bile duct. A report of the hepatic material submitted for histology could not be traced.

Table 1 Age and Sex distribution of patients and diagnosis

Case No.	Age	Sex	Diagnosis
1	33	M	Cholelithiasis and Choledocholithiasis
2	39	F	Cholelithiasis and Choledocholithiasis
3	38	F	Cholelithiasis and Choledocholithiasis
4	48	M	Carcinoma Head of pancreas
5	51	M	Carcinoma Head of pancreas
6	49	M	Bile duct carcinoma
7	41	F	CBD transection
8	30	M	Cause not found

Note:The three patients with carcinoma were all more than 45 years old.

Table II: Clinical Presentation

Presenting Symptoms	Number
Jaundice (yellow eyes)	8
Itching	5
Abdominal pain	4
Fever	3
Passing dark urine	5
Weight loss	3

Note:Weight loss was present in the patients with carcinoma of pancreas or bile duct.

Table III Management

Surgical Procedure	Number
Cholecystectomy + CBD exploration	3
Cholangiojejunostomy	1
Cholecystojejunostomy	2
Choledochojejunostomy	1
Exploration only	1
Total	8

DISCUSSION
ANATOMY OF THE EXTRAHEPATIC BILIARY SYSTEM

The extrahepatic bile duct system originates from the liver as the right and left hepatic ducts each of which is 1 - 2 cm long; and drains the respective hepatic lobe.[5] These join to form the common hepatic duct which is 2 - 4 cm long. The common hepatic duct is joined by the cystic duct to form the common bile duct which is 8 - 15 cm long and 5 - 10 mm in outside diameter.[6] This descends in the hepatoduodenal ligament to the right of the hepatic artery and anterior to the portal vein. It passes posterior the first part of the duodenum, through or behind the pancreas and enters the posteromedial aspect of the second part of the duodenum at the papilla of vater. The choledochoduodenal junction is an oblique passage occupied by the common bile duct and the main pancreatic duct of Wirsung. These two ducts join in a

common channel, the Ampulla of Vater, which opens in the duodenum at the Papilla of Vater. However, the two ducts may join before entering the duodenal wall or they may empty separately. The choledochoduodenal junction is surrounded by the sphincter of Oddi which regulates the flow of bile.

The gallbladder is a pear-shaped organ covered by peritoneum and attached to the undersurface of the liver. It is about 7 - 10 cm long and 3 - 5 cm in diameter.[6] This is drained by the cystic duct which is 4 cm long. The cystic duct joins the right lateral aspect of the common hepatic duct to form the common bile duct.

The blood supply to the common bile duct comes from the retroduodenal artery which is a branch of the gastroduodenal artery. The gall-bladder is supplied by the cystic artery, a branch of the right hepatic artery.[5] The cystic artery divides into anterior and posterior branches. During operation the cystic artery is usually found in the Carlots triangle which is bounded by the liver, the common hepatic duct and the cystic duct. It contains the cystic artery, a lymph node and the right branch of the portal vein. Venous drainage from the extrahepatic biliary system is in the portal vein. The lymphatic drainage of the gall bladder is to the lymph nodes in the porta hepatis and thence in the free edge of the lesser omentum to the

coeliac group of pre-aortic lymp nodes.

The most important aspect of the anatomy of the extrahepatic biliary system is that it has a lot of variations.[6] For this reason surgery on the biliary system requires an extremely careful technique bearing in mind the possibility of anatomical variations. Injuries to the bile ducts usually result from failure to recognize the variation in the anatomy of the biliary tree and its blood supply. The consequences of such injuries are often serious, sometimes leading to obstructive jaundice, the very problem that one wished to correct.

PHYSIOLOGY OF BILE PIGMENT METABOLISM

Bilirubin is one of the end-products of haemoglobin degradation. When red blood cells are destroyed either effete red blood cells or prematurely by pathological causes haemoglobin is split into haeme and globin.

The haeme ring is opened to give iron and a tetraphyrrole ring. This is transformed into biliverdin which is green.[7]

Biliverdin is reduced to bilirubin which is yellow. The bilirubin which is so formed binds tightly to albumin to form a stable protein-pigment complex which is transported to the liver. This is called "indirect reacting bilirubin" since it does not react with the Van de Bergh diazo reagent unless treated with alcohol.

It is also poorly soluble in water and it is not excreted in urine. In the hepatocytes the indirect reacting bilirubin is conjugated with glucuronic acid to form a diglucuronide which is water soluble, it is excreted in urine and it reacts with the diazo reagent and is therefore called "direct reacting bilirubin." The conjugated bilirubin is excreted via the biliary system into the intestines where it is acted upon by bacteria to form urobilinogen. Some of the urobilinogen is reabsorbed through the intestinal mucosa into the blood. Most of this is re-excreted by the liver into the gut, but some (about 5%) is excreted by the kidneys into the urine.[7]

When there is excessive accumulation of large quantities of bilirubin in the extracellular fluids either due to excessive production or impaired excretion, jaundice occurs. When faced with a jaundiced patient the first step is to try to determine whether it is due to cholestasis or not, since if it is cholestatic there may be a surgically correctable lesion. If it is cholestatic, the next step is to determine the anatomical level of the obstruction. This is the objective in investigating the jaundiced patient.

CLINICAL PRESENTATION OF CHOLESTASIS

The patient develops jaundice slowly and he generally feels well, which is in contrast to jaundice associated with hepatocellular disease where malaise and physical deterioration are common.[1] It is important to note whether jaundice is progressive or intermittent since intermittent jaundice may indicate choledocholithiasis. Jaundice and fever may suggest cholangitis.

Pruritus is a common presenting symptom. It is often attributed to bile salts deposition in the skin although experimentally this has never been established; and in terminal liver failure while serum bile salts may still be increased pruritus may abate.[1] However, the association of pruritus with cholestasis suggests that this is due to a substance normally excreted in bile.[1] The relief of pruritus by cholestyramine, a bile-salt chelating agent suggests that bile salts are involved in its pathogenesis; and the disappearance of pruritus with liver failure suggests that probably the agent leading to pruritus is manufactured by the liver.[1] Often when patients note jaundice they also note passage of dark coloured urine. This is due to presence of bilirubin in urine. Patients may also note changes in the colour of faeces which become pale or clay coloured. This is a good indication as to whether cholestasis is total, intermittent or decreasing.[1] Since bile salts are

important in the absorption of fats steatorrhoea may also result, and the severity of steatorrhoea is proportional to the depth of jaundice.[1]

Pain may or may not be present. Biliary colic may be present in three quarters of patients with choledocholithiasis who have cholangitis. Pain is also said to be present in 34% of patients who have pancreatic cancer.[8,9]

Fever when present also suggests cholangitis. It is present in a third of patients with choledocholithiasis who develop jaundice and cholangitis.[8]

Weight loss may be one of the major manifestations in patients with pancreatic cancer[9] and in cancer of the bile duct.[10]

INVESTIGATIONS OF THE JAUNDICED PATIENT

1. **Urine Examination**

   Urine bilirubin is positive. This is also evidenced by the dark colour and on shaking the urine becomes frothy. Urine urobilinogen is negative.

2. **Blood biochemistry**

   Serum bilirubin is raised, mainly the direct reacting type. When cholestasis is relieved serum bilirubin falls slowly to normal.[1]

   Serum alkaline phosphatase, is raised usually more than three times the upper limit of normal.

Serum albumin and globulins are normal in acute cholestasis. With the development of biliary cirrhosis serum albumin tends to fall with inversion of albumin/globulin ratio.[1]

3. **Haematological investigations**

There is prolongation of prothrombin time which is corrected by parenteral administration of Vitamin K1. This is due to failure of synthesis of Vitamin K related clotting factors resulting from malabsorption of Vitamin K and other fat-soluble vitamins.

4. **Abdominal Ultrasound**

This has emerged as being an initial imaging examination technique because of its great advantage of being non-invasive and free from risk.[3] It may define presence of gall bladder stones, dilated biliary tree, abnormal hepatic anatomy and abnormalities in the pancreas such as pancreatic cancer.[1,3,11,12]

Ultrasound imaging will detect dilated ducts despite absence of jaundice.[3] It is also helpful in detecting occult cancer since jaundice does not interfere with its interpretation.[3] It should be noted that ultrasound is not a good test for defining calculi in the common bile duct although they can occasionally be demonstrated.[3] Also inability to visualise the whole gland limits the

sensitivity of ultrasound in the diagnosis of pancreatic cancer, although pancreatic tumours greater than 2 cm diameter may be visible.[12] However, endoscopic ultrasound in which a small transducer is incorporated into the end of a gastroscope may enable ultrasonic examination of the whole pancreas from within the stomach thus enabling detection of small neoplasms.[12]

5. **Percutaneous Transhepatic Cholangiography (PTC)**

Percutaneous transhepatic cholangiography (PTC) using the Chiba needle has gained acceptance in the last two decades.[3] It is the diagnostic procedure of choice when proximal lesions are suspected as it allows better visualization of the proximal biliary tract and proximal extension of a tumour if present.[10] When on the basis of ultrasound scan a distal lesion is suspected because of dilated gall bladder, dilated extrahepatic bile ducts or there is a possibility of a peri-ampullary tumour, then endoscopic retrograde cholangiopancreatography (ERCP) would be the procedure of choice.[10]

ERCP and PTC are complementary[3,12]. About 60% of non-dilated ducts are visualized by PTC.[3]

6. **Endoscopic Retrograde Cholangiopancreatography (ERCP)**

When on basis of information from ultrasound or computed tomography scan a distal obstruction is suspected because of a dilated gall bladder or extrahepatic biliary system, or a periampullary lesion is suspected, endoscopic retrograde cholangiopancreatography should be the procedure of choice.[10] The following are some of the advantages of ERCP according to Shea:[3]

- It allows the physician to assess the stomach and duodenum, and if there is a malignant lesion biopsy can be taken.
- The Ampulla of Vater can be assessed critically to rule out periampullary carcinoma.
- The common bile duct can be assessed and if there are stones endoscopic papillotomy can be carried out, and if cancer is present a stent can be placed.

ERCP is a highly accurate method in identifying and excluding gall-stones, strictures of benign and malignant origin and may achieve a specificity and sensitivity of greater than 90%.[4] It is also said to be the most sensitive test in diagnosis of pancreatic carcinoma.[13] Complications of ERCP include sepsis, bile leakage, haemorrhage and

pancreatitis. Following introduction of ERCP complications occured in upto 3% of patients being examined. However, morbidity and mortality have now fallen to less than 1% and 0.1% respectively.[4] When performed together ERCP and PTC are complementary.[3,10]

7. **Computerized Tomography (CT)**

This is becoming more popular and obstructive jaundice is an appropriate indication.[3] It will delineate dilated bile ducts and it will define lesions in the pancreatic head, metastatic hepatic lesions and gastric lesions.[3] It is the most informative non-invasive technique currently available for the diagnosis of pancreatic cancer.[14] When combined with ultrasound scan the sensitivity is greater than for either test alone.[14]

8. **Hepatobiliary Iminodiacetic Acid Scan (HIDA)**

Within the past decade HIDA scan has become an important method for investigating the causes of right upper quadrant abdominal pain.[3] The biliary system is the normal route for excretion of lipid soluble compounds. Derivatives of iminodiacetic acid can be chelated with technetium 99^{m} to form a dimer with high molecular weight. This substance can be substituted with side chains to increase

its lipid solubility. These agents are extracted by the liver from both the hepatic arterial and portal venous blood. They are actively transported across hepatocytes, bound to intracellular proteins secreted into the biliary canaliculi and excreted into the duodenum through the biliary three.[3] With biliary obstruction the production and secretion of these agents will be stopped.[3] So, the most important application of HIDA scan is in the area of suspected obstruction and bile stasis. They are also used in studying gall-bladder function and intestinal transit of bile.[3] Demonstration of a patent biliary tree is closely related to absence of obstruction, dilated ducts can be seen by HIDA scan, but ultrasound is probably more useful in this aspect.[3]

9. **Operative Cholangiography**

This procedure is performed in the operating room at the time of surgery for biliary tract disease with injection of contrast material either in the cystic duct or T-tube placed in the common bile duct.[7] The aim is to establish whether there is a need to open and explore the common-bile duct.[7,15] Cholangiography could be done selectively with several criteria which are also indications for exploration of the common bile duct.

- Jaundice present, or a recent history of jaundice.
- Acute cholongitis or pancreatitis
- Raised serum bilirubin and alkaline phosphatase
- Dilated common bile duct or cystic duct
- Palpable stone in the common bile duct

A selective operative cholangiography based on these criteria reduces the need for unnecessary cholangiograms, detects almost all duct stones and avoids the problem of unnecessary explorations of the common bile duct.[15]

Common causes of cholestatic jaundice as seen at Muhimbili Medical Centre

In the period of study eight cases of cholestatic jaundice were seen and their aetiological distribution was as follows:

1. Choledocholithiasis 3 patients
2. Post-operative complications 1 patient
3. Carcinoma of the pancreas 2 patients
4. Carcinoma of the bile duct 1 patient
5. Unknown cause 1 patient

Each of these causes will now be discussed briefly.

CHOLEDOCHOLITHIASIS

Common bile duct stones are found in 12% of cases subjected to cholecystectomy.[7] The majority of stones in the common bile ducts have migrated from the gall

bladder and are associated with calculous cholecystitis.[7,8] Secondary calculi in the bile duct usually follow partial biliary obstruction due to residual calculus, traumatic strictures, sclerosing cholangitis or congenital biliary abnormalities.[8] The stones may be single or multiple and tend to impact in the ampulla of Vater and may project into the duodenum. Patients with Clonorchis Sinensis have a tendency to forming bile duct stones.[7] Stones may pass into the duodenum, but more commonly they are arrested in the lower end of the common bile duct which is narrower, and they exert a ball and valve mechanism leading to intermittent jaundice.[7,8] Edema, fibrosis and spasm of the distal bile duct secondary to irritation aggravate the obstruction.[7] The stagnant bile is readily infected probably from the intestines leading to ascending cholangitis.[8] The bile becomes opaque and dark brown (bile mud). Escherichia Coli is the commonest infecting organism.[7,8] Infection may extend to the liver leading to hepatic abscess.[7,8] Acute or chronic pancreatitis may result from stones in the ampulla of Vater with bile regurgitating along the pancreatic duct.

Bile duct stones may be assymptomatic, they may present with acute cholangitis and jaundice, or they may be residual, detected post-cholecystectomy. When they are symptomatic they present with abdominal pain in the

right upper quadrant radiating to the right shoulder with intermittent jaundice accompanied by passage of pale, clay-coloured stools and dark urine.[7] In contrast to patients with neoplastic obstruction of the common bile duct or ampulla of Vater, the gall bladder is usually not distended because of previous fibrosis according to the Courvoisier's law.[7]

The investigations used for diagnosis of bile duct stones include:

1. Ultrasound scanning.
2. Endoscopic Retrograde cholangiopancreatography
3. Percutaneous transhepatic cholangiography.

The treatment for stones in the common bile duct is surgical removal.[7] Pre-operatively patients should be prepared with vitamin K if the prothrombin time is prolonged. If cholangitis is present antibiotics should be administered.

Removal of stones in the common bile duct is facilitated by use of balloon-tipped catheters during conventional CBD exploration. Choledochoscopic removal of bile duct stones is one of the recent advances in hepatobiliary surgery.[15] This can be used as a final check up procedure after conventional CBD exploration or when the technique is mastered it can be done as a primary procedure using the choledochoscope as the exploratory instrument.[15] While retained stone rate of 15-20% was observed following conventional CBD

exploration, with the use of choledochoscopy it is now lower than 2%.[15]

Indication for CBD exploration after surgery for cholelithiasis include:

1. Palpable stones within the CBD
2. Dilated CBD
3. History of jaundice
4. Recent investigations showing stones in the bile duct
5. Positive operative cholangiogram
6. Presence of facetted stone.

The optimal time for CBD exploration is during the initial procedure of cholecystectomy.[7]

Chemical dissolution is one of the other modes which can be used to remove CBD stones and one of the agents is Monoctanoin (Moctanin) which is a semi synthetic vegetable oil and is moderately efficient in dissolving cholesterol stones.[15] It is given as an intraductal infusion through a nasobiliary catheter.[15] Extracorporrheal shock-wave lithotripsy can replace most of the other techniques in future.[15]

BILE DUCT CARCINOMA (CHOLANGIOCARCINOMA)

Bile duct carcinoma has an average autopsy incidence of 0.3%, with a male to female ratio of 3:2[7,10] The highest incidence is in the 6th and 7th decade.[7] There is no clear-cut causative factor although a

highest incidence is in the 6th and 7th decade.[7] There is no clear-cut causative factor although a relationship with ulcerative colitis, clonorchis sinensis and some congenital anomalies such as choledochus cysts have been suggested.[10]

Bile duct carcinoma has been divided according to location in the following groups:[11]

1. Proximal third tumours (58%)
2. Middle third tumours (17%)
3. Distal third tumours (18%)
4. Diffuse involvement (7%)

At the Lahey clinic 75% of the tumours are located at or around the hepatic duct bifurcation.[10] The tumour usually involves the whole thickness of the duct resulting in complete obstruction in about a third of cases.[7] The most frequent type of cancer is adenocarcinoma with other rare types being squamous cell carcinoma, leiomyosarcoma and mucoepidermoid carcinoma.[10] The liver and regional lymph nodes have been reported to be involved in metastases in about three-quarters of autopsy cases[7] but only one third of patients were found to have liver, lymph node or peritoneal metastases at laparotomy from one series at Lahey clinic.[10]

Clinically carcinoma of the bile ducts may present in one of the three ways:

(a) Obstructive jaundice which is abrupt, associated with pruritus.[7,10,12] Weight loss is nearly always present and abdominal pain is reported in

half of the patients.[7,10] Cholangitis may be present and the liver is palpable in 50% of patients. The gallbladder is palpable in a third of patients. This is the most common presentation.[7,10].

(b) In patients who have obstruction of a single left or right hepatic duct there may be no jaundice but patients will present with abdominal pain with or without cholangitis.[7,10]

(c) The third group may present with features mimmicking cholecystitis.

Diagnostic tests according to Tompkins et al[10] include:

- Ultrasound or computerized tomography scans which will confirm presence of dilated ducts or identify a tumour or metastatic disease.
- If proximal biliary tract tumour is suspected basing on ultrasound scan then PTC is the next procedure of choice.
- When on the basis of ultrasound or CT scan distal obstruction is suspected, then ERCP is done as the next procedure of choice. It allows assessment of the duodenum and the periampullary area and biopsy can be taken.

Treatment of patients with carcinoma of the Bile duct may be curative or palliative. Tumours may be resected or bypassed by anastomosing the dilated proximal duct system to a segment of the small intestine.[7] Therapy

should be tailored to each patient individually taking into account the location and extent of the disease, the condition and the operative risk of the patient and the expertise available at the institution.[10] In general distal third tumours are managed by radical pancreaticoduodenectomy. Middle third tumours are resected and biliary drainage established by Roux en Y hepaticojejunostomy. Removal of upper third tumours may require resection of the hepatic hilus and anastomosing the right and left hepatic ducts to a Roux en Y limb of the jejunum.[7]

Resectability of these tumours is 15-20% and cure rate is low; but the 5 year survival and quality of life is better in patients undergoing resection rather than drainage alone.[7]

In this study one patient had proximal bile duct carcinoma and a bypass procedure (cholangiojejunostomy) was done. Although longterm follow-up is unknown he had immediate relief from symptoms.

CARCINOMA OF THE PANCREAS

Carcinoma of the pancreas is the fourth leading cause of death from cancer in the United States, exceeded only by carcinoma of the lung, colon and breast.[13,14] It is responsible for 3-6% of all cancer deaths in Western Europe, United States and Japan.[13] The incidence increases with age and 75% of the patients

are more than 60 years at the time of diagnosis.[13] It is more common in males with a male to female ratio of 1.6:1.[13] The aetiology of carcinoma of the pancreas is unknown, but exogenous carcinogens have been implicated including dietary fats, coffee consumption and cigarette smoking.[7,13,14]

Distinction must be made between periampullary and pancreatic cancer because the prognosis is different.[13] Periampullary cancer (that is duodenal, ampullary or lower common bile duct) is usually well-differentiated and presents early with localized disease. It is often resectable, carrying a good prognosis if the regional lymph nodes are not involved.[13] Carcinoma of the ampulla of Vater and the duodenum are frequently difficult to differentiate, both being collumnar cell adenocarcinoma.[7]

Both present with intermittent jaundice due to recurrent sloughing of the central portion of the tumour.[7]

In 90% of cases pancreatic carcinoma is of ductal origin. Seventy-five percent are mucinous adenocarcinoma, but often the tumour is undifferentiated.[13] Cystadenocarcinomas of the pancreas are rare lesions, but their importance lies in the fact that growth is slow and metastases occur late even when the tumour is large.[7] Also these tumours should not be confused with pancreatic pseudocysts and

be subjected to cystoenteric drainage.[7]

Pancreatic carcinoma initially presents with vague symptoms, hence delaying diagnosis. The most common definitive symptoms are weight loss, jaundice and pain.[13,14] Weight loss is the earliest feature and is present in 70-80% of patients; regardless the location of the tumour.

Abdominal pain is present in 50-85% of patients.[7,13,14] progressive jaundice follows and is present in 70-80% of patients with carcinoma of the head of pancreas and the incidence of jaundice decreases as the location of the tumour progresses to the tail.[14] Intermittent jaundice is observed in 10% of patients. Pruritus is common and may be very disabling.[7] Sudden unexplained maturity onset diabetes mellitus may be the first indication of the disease.[13] The liver is palpable in 50-70% of patients while the gall bladder is palpable in a third of patients.[7,14]

Hypotonic duodenography may help in defining ampullary or duodenal carcinoma but it is not specific for carcinoma of the pancreas.[14]

Ultrasound and CT scan are useful and complementary in diagnosis of pancreatic cancer.[7,13,14]

Invasive tests include PTC and ERCP.[7,13,14] ERCP is said to be the most sensitive test in the diagnosis of pancreatic carcinoma and is superior to any other method in the differentiation between pancreatitis and

carcinoma.[13] Cytologic diagnosis can be obtained pre-operatively by fine needle aspiration under ultrasound or computerized tomography guidance.[13] Laparoscopy is said to be useful in establishing a definitive diagnosis and detection of advanced disease obviating unnecessary laparotomy.[13]

The management of carcinoma of the pancreas is influenced by the location of the disease, its stage and the general condition of the patient. Surgical excision remains the only hope of cure for longterm survival in patients with periampullary tumours and the rare instances of early cancers of the head of the pancreas, but most cases of cancer of the body and tail are inoperable at the time of diagnosis.[13]

The standard surgical procedure is pancreaticoduodenectomy (Whipple's procedure) with en-block removal of the duodenum, head and neck of the pancreas, antrum of the stomach and upper jejunum, gallbladder and common bile duct with associated lymph nodes.[13]

The major morbidity associated with Whipple's procedure is the pancreaticojejunal anastomosis resulting into a pancreatic fistula in up to 20% of cases.[14] In order to avoid this complication total pancreatectomy was advocated by Ross in 1954.[14] Another argument put forward for total pancreatectomy is the multifocal nature of the disease.[13,14] The other alternative type

of resection is regional pancreatectomy which involves resection of the pancreas, the regional nodes, the spleen and the retropancreatic segment of the portal vein. The vein is then reconstruced by end to end anastomosis[13],

However, Cushieri[14] argues that there is no justification for total or regional pancreatectomy since from various studies there is no difference in the 5 year survival rates between Whipple's procedure and total pancreatectomy.

Palliative surgical treatment is performed if the tumour is found inoperable at laparotomy, when endoscopic or percutaneous stenting have failed or the facility is not available locally.[13] This could be by biliary bypass with or without gastrojejunostomy. Cholecystojejunostomy and choledochojejunostomy seem to be especially effective provided the tumour is below the entrance of the cystic duct.

The development of high energy radiotherapy machines with the ability to deliver sufficient dosage to the pancreatic region have made radiotherapy an adjunctive treatment for pancreatic cancer.[14] It has effected a mean survival rate similar to that of bypass with associated relief from pain.

Chemotherapy has been tried and a combination of 5 Fluorouracil with Mitomycin C or Methyl-CCNU seems to yield a better result than single drug chemotherapy.[14]

In our study we had two patients who had an inoperable

carcinoma of the pancreas and were managed by cholecystojejunostomy as a bypass procedure. One died of metastatic disease; the other was discharged in good condition but was lost from follow-up.

JAUNDICE FOLLOWING ACCIDENTAL BILIARY DUCT INJURIES DURING SURGERY

The variations of anatomy of the cystic duct and cystic artery are numerous predisposing these structures including the common bile duct to accidental injuries. The most devastating complication of cholecystectomy is injury to the bile duct system.[16] According to Hermann[17] injury to the bile duct system occurs in 0.5% of cholecystectomies. Often the surgeon is unaware and the first indication of such an injury is the development of jaundice in the early post-operative period.[16] Injuries may be:

- Complete transections of the common bile duct or hepatic ducts
- Partial transections
- Fibrosis of the common bile duct following repair of an avulsed cystic duct.

Transections of the common hepatic or common bile duct are the ones likely to lead to cholestatic jaundice. For complete transections end to end anastomosis with stenting is the preferred treatment. The stent should stay for 10 - 15 days.[17] If reconstruction is impossible choledochojejunostomy or

choledochoduodenostomy should be done.[17] Even after successful repair close follow-up is necessary because bile duct stenosis can occur as a late complication.[17] In our study we had one patient who developed jaundice after cholecystectomy and at re-laparotomy it was found that she had complete transection of the common bile duct with complete retraction of the proximal segment. She had cholecystojejunostomy done with a Roux-en Y loop and she improved.

CONCLUSION

Obstructive jaundice is a common surgical problem which is challenging both in investigations and management. The availability of ultrasound scanning is an added advantage in investigating the jaundiced patient. However, facilities for percutaneous transhepatic cholangiography (PTC) and endoscopic retrograde cholangio-pancreatography (ERCP) are needed. These would greatly facilitate in the proper investigations and management of patients, especially in cases where stents are indicated. This would eliminate the necessity of doing unnecessary laparatomy sometimes in debilitated patients simply to perform a bypass procedure to relieve jaundice.

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AN ANALYSIS OF HIV SEROPOSITIVE PATIENTS IN THE SURGICAL WARDS AT MUHIMBILI MEDICAL CENTRE

SUMMARY

A total of 49 patients who were HIV positive by ELISA were seen in the surgical wards either for diagnostic or therapeutic surgical procedures between June, 1989 and July 1990, This case collections includes patients who fulfilled the World Health Organization case definition of AIDS and those who did not have all clinical criteria but were ELISA positive for HIV.

The most common surgical manifestations included tuberculosis (tuberculous thoracic empyema, tuberculous pericardial effusion, and tuberculous lymphadenitis) which accounted for 28.6% of cases, Pyomyositis (19%), Kaposis sarcoma (9.5%) and lymphadenopathy (unspecified) in 9.5%. Other common conditions included cutaneous ulcers, anorectal conditions and dermatoses.

Tuberculosis patients were given antituberculous drugs in addition to the appropriate surgical procedures. Patients with pyomyositis were treated with antibiotics and surgical drainage while patients with Kaposis Sarcoma were treated at the Tumour Centre with radiation therapy. Patients with lymphadenopathy were managed according to the underlying cause of the adenopathy.

INTRODUCTION

The Acquired Immunodeficiency Syndrome (AIDS) was first described in the United States of America (USA) in 1981 and since then the incidence has been rising rapidly all over the world. By the end of May 1989 157,191 cases had been reported to the World Health Organization (WHO) from all continents[1] and by June 1990 there were 263,051 cases reported to WHO.[2]

The disease was first noted in Tanzania by physicians in Kagera Region in 1983 and the first case was confirmed at Muhimbili Medical Centre in 1985.[3,4] The disease is seen in every discipline of medical practice including Surgery. Much has been written on AIDS in every clinical discipline but it is generally taken as a "medical problem" and there is relatively less literature on AIDS and Surgery. There are, however, common surgical manifestations of the disease. Carswell[5] has described some initial observations of HIV infected patients in surgical wards as seen in Uganda.

This is a descriptive study in which an attempt is made to analyse HIV Seropositive patients as seen in the surgical wards. The findings should be interpreted within the limitation that this is not a case-control study. It is hoped that this will be a stimulus to further case control studies to evaluate surgical manifestations of AIDS.

CASE PRESENTATION

S.M. (Reg. No. 603324) a male peasant, aged 36 years, was admitted to the surgical ward on 12.3.90 with

history of multiple skin swellings of 4 months duration.

He had developed multiple skin swellings involving the face, trunk and both upper and lower extremities all of which were 4 months duration. He had also noted swellings in the oral cavity. He had on and off fevers and had lost much weight. He had no history of diarrhoea. In the week prior to admission he had noted shortness of breath but he had no cough nor paroxysmal nocturnal dyspnoea. He was single, but he admitted to having multiple female sexual partners.

Clinical examination revealed a febrile and pale young man with a temperature of 38.5°C, a pulse rate of 100 beats per minute, and a blood pressure of 110/70 mmHg. He had generalized cervical, axillary and inguinal lymphadenopathy. The lymph nodes were rubbery in consistency varying in size from 0.5 - 2 cm and in some areas they were matted. He had woody oedema in the right leg. He had mixed skin lesions some nodular and florid lesions and some were plaques. They extended from the face, the trunk, the upper and lower extremities and the scrotum. There were some lesions in the gums and the palate which were purple in colour. He had no oral thrush.

The respiratory system and the heart sounds were normal. Abdominal examination revealed hepatomegally of 3 cm below the right subcostal margin but the spleen

was not palpable. Rectal examination was normal.

A diagnosis of aggressive Kaposis Sarcoma with AIDS was entertained. Investigations done included;:

(1) ELISA for HIV was positive and the Western Blot test was positive.

(2) The haemogram was as follows:

- Haemoglobin 9.4 gm/dl
- White Cell count total 3,600/cumm

  Differential count:

  Neutrophils 56%

  Eosinophils 2%

  Basophils 1%

  Lymphocytes 41%

(3) Blood slide for parasites was negative for malaria

(4) Biopsy from one of the skin nodules revealed Kaposis Sarcoma.

As the patient was being worked up for definitive treatment he developed respiratory distress and died.

This case serves to illustrate the multiplicity of lesions in one patient, the multiple region/organ involvement and the rapid progression of aggressive Kaposis' sarcoma in contrast to the endemic Kaposis' Sarcoma.

The other cases are summarised in the following tables:

Table II Age and Sex Distribution of the Patients

Age Group	Males	Females	Total	Percentage
Below 20 years	2	2	4	8.2
21-30	14	5	19	38.8
31-40	12	4	16	32.6
41-50	4	2	6	12.2
Above 50	3	1	4	8.2
Total	35	14	49	100.0

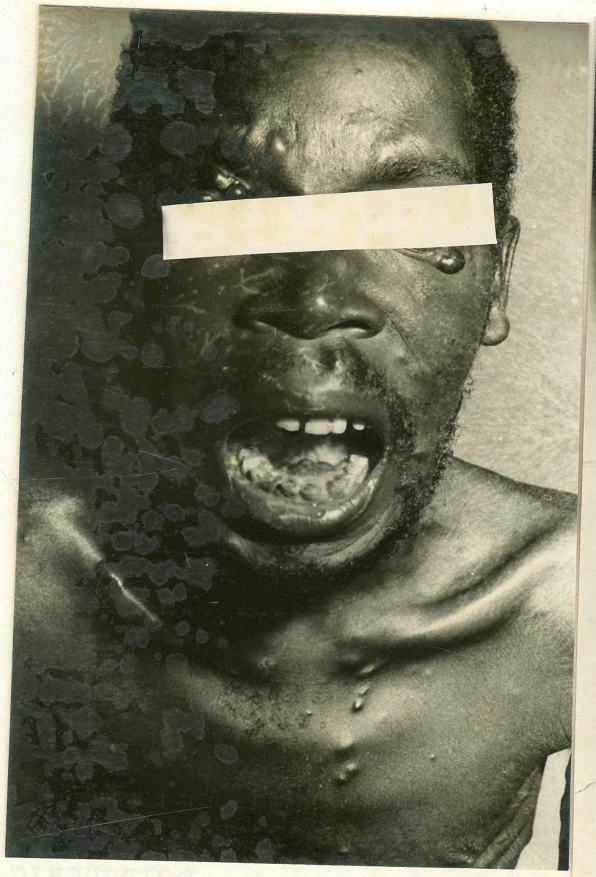
Note: 71.4% of cases were in the 21-40 years age group

Table II: Surgical Presentation

Presentation	Number	Percentage
Pyomyositis	12	19.0
Kaposis Sarcoma	10	15.8
Pericardial effusion (Tubercoulous)	6	9.5
Lymphadenopathy (other than Kaposi sarcoma)	8	12.8
Cutaneous ulcers	6	9.5
Empyema Thoracis	4	6.4
Others	12	19.0
Total number of conditions	63	100

Note: (a) Some patients had more than one presenting symptom/problem.

(b) The most common presentations were Pyomyositis, Kaposis sarcoma and Lymphadenopathy.





Photographs of the index case showing:

- scattered nodular lesions on the trunk and face
- Florid lesions on the gums and floor of the mouth
- woody oedema on the right leg

Other Salient features:

(a) The "others" in the table above included:

- Herpes zoster - 5 patients
- Mediastinal masses - 2 patients
- Oropharyngeal candidiasis - 2 patients
- Bladder tumour - 1 patient
- Fournier's Gangrene - 2 patients

(b) Urine for sugar was tested in 11 patients with suppurative conditions, none was positive.

(c) Blood sugar was done in 4 patients and it was normal in all patients.

(d) Mantoux test was done in 4 patientsit was positive in one and negative in the other three patients.

(e) Two of the Kaposi Sarcoma patients were presenting with recurrent disease having been treated with radiotherapy and achieved remission 2 and 3 years back.

DISCUSSION

Since the recognition of AIDS cases in 1981 there is an enormous amount of literature and research on HIV infection and AIDS in all branches of medicine; but there is relatively little research on AIDS and surgery. As Sim and Dudley[6] put it "Surgeons voices have been little heard in the discussions and controversies over infection with the human immunodeficiency virus. Yet there is bound to be a growing number of patients infected with HIV who will come in contact with surgeons."

Sim and Dudley categorized AIDS patients who come into contact with surgeons in three categories:[6]

(a) Those who have AIDS and surgical complications or who need invasive treatment.

(b) Those who present with specific surgical problems related to their lifestyle for example anorectal diseases in homosexuals.

(c) Those patients with injuries or problems unrelated to HIV infection that require surgical management.

These authors also cautioned that surgeons come in contact with blood in the least controllable circumstances.[6]

In this presentation it is the patients in the first category that will be discussed as seen at Muhimbili Medical Centre; with a brief review of literature on the virus itself, pathogenesis, immunology and clinical features.

AETIOLOGY

AIDS is caused by a retrovirus which was initially called Lymphadenopathy Associated Virus (LAV), later on it was called the Human T-cell lymphotropic virus type III (HTLV-III) by Ballo et al.[1] Subsequently in 1986 the International Committee on Viral Nomenclature named it the Human Immunodeficiency Virus (HIV).[1,8]

HIV is a retrovirus, and retroviruses are RNA viruses.[8] The name retrovirus refers to the fact that RNA transcription proceeds in a reverse direction (RNA to

DNA) before the viral genome can be incorporated into the host genome and viral replication to commence. This essential step is dependent on a viral enzyme reverse transcriptase. Pharmacological inhibition of this enzyme inhibits replication of HIV.[7]

The structure of the HIV has been determined by electron microscopy. The viral membrane is acquired from the infected cell as the virus buds from the cell membrane. This membrane (or envelope) contains an envelope glycoprotein, gp 41. Inserted in this envelope are protein molecules coupled to carbohydrates called gp 120. Both gp 41 and gp 120 are essential for viral infectivity and probably play a role in the pathogenesis of HIV.[1,8]

Inside the membrane is a core protein shell called core protein p18. It has a viral nucleocapsid composed of many sub-units each weighing 24 kilodaltons.[1]

This p18 shell contains the viral RNA and the reverse transcriptase enzyme.[1] HIV must infect a permissive cell before replication can take place. From various experiments it has been found that the receptor binding side for CD4+ lymphocytes resides on gp 120 as will be elaborated later.[8]

RECEPTORS AND CELL TROPISM

In 1984 Klatzman[8] reported that HIV selectively infects CD4+ lymphocytes in mixed cell cultures, and later on

he and Dalgeisch[8] demonstrated that the receptor for HIV binding to the cell surface may be the CD4 antigen. These cells are principally the helper or inducer subsets of T-lymphocytes. Other macrophages which express the CD4 antigen and are therefore susceptible to HIV infection include the following:

1. In the peripheral blood: Tlymphocytes, B-lymphocytes and monocytes.
2. In the lung: alveolar macrophages (type II pneumocytes)
3. In the skin. Langerhans cells
4. In the central nervous system - Glial cells (microglia)
5. In the gut - argentaffin cells
6. In the extracellular fluid HIV has been found in semen, cervical fluid, tears, saliva, cerebrospinal fluid and breast milk.

This may explain the HIV infection manifesting itself in such sites as the skin, lungs and the nervous system.

The route of transmission of the virus are intromissive sexual intercourse (either heterosexual, or homosexual), blood transfusion and vertically from mother to child.[1]

IMMUNOLOGY

Many of the clinical manifestations of HIV infection can be ascribed to the profound immuno-deficiency that occurs in these individuals. The main target of the virus is the T-helper-inducer cells. These cells carry the glycoprotein molecule called CD4. This protein has

been shown to bind to gp 120 of HIV. There is destruction of CD4+ lymphocytes which are particularly susceptible to the cytopathic effect of HIV as are other cells that have the CD4 molecules on their surfaces (for example macrophages, langhan's cells and glial cells etc).

Another possibility is that the envelope gp 120 on the virus may interfere with the normal function of the CD4+ lymphocyte. There is therefore a decrease in both the number and function of T-helper/inducer cells. The secretion of particular cytokines is also reduced.[9]

B-lymphocytes become non-specifically activated in HIV infection and the result is hypergammaglobulinaemia. The mechanism is unclear. The cytotoxic effects of natural killer cells (NK) is also reduced. There is also a defect in the chemotactic and phagocytic function of the monocyte-macrophage system.[1,8,10]

There is also an impaired response to recall antigens on skin-testing, that is an impaired delayed-type hypersensitivity.

The overall effect of all these defects is an increased susceptibility to infections. Some authors have also implicated an autoimmune mechanism but there is no evidence of autoantibodies to support the concept.[10]

Patients are susceptible to opportunistic infections such as candida and protozoa, but the course of other non-opportunistic infections may be accelerated.

Infections with organisms such as streptococcus pneumoniae, staphylococcus aureus and mycobacterium tuberculosis are more severe in AIDS patients as compared to other individuals.[12]

SURGICAL CLINICAL MANIFESTATIONS

(a) **TUBERCULOSIS**

Patients with tuberculosis needing some surgical intervention either in diagnosis or treatment formed the most common presentation of AIDS and HIV infection in the surgical wards. Fourteen (14) cases with various types of tuberculosis were seen which is 28.6% of all AIDS cases in this series. They included tuberculous pericardial effusion (6 patients), tuberculous lymphadenitis (4 patients) and tuberculous thoracic empyema (4 patients).

The association of tuberculosis and AIDS has been described by various authors from different series. It is known that patients with AIDS have a high susceptibility of developing tuberculosis due to depression of cell-mediated immunity. In these patients tuberculosis may occur either as a reactivation of a previous latent disease or as a new infection as a result of immunodeficiency.[13] It has also been found that in these patients tuberculosis tends to cause higher morbidity and mortality as compared to TB in other non-AIDS patients. In these patients tuberculosis tends to be extrapulmonary,

mainly miliary, lymphadenitis, tuberculous pericarditis or central nervous system infections resulting in tuberculous brain abscesses.[1,12,13] It is these extrapulmonary cases which end up in the surgical wards.

In Africa tuberculosis has been reported to involve as many as 20% of HIV infected patients.[1] Series from Kinshasa in 1985 revealed that 33% of tuberculosis patients were HIV seropositive.[16] In Tanzania Cegielski et al[3] found an increase in the number of patients with effusive pericardial disease in most of whom it was tuberculous in nature.

The fourteen patients seen in our study were treated with the standard antituberculosis drugs, that is streptomycin, isoniazid and thiacetazone. For those in whom acid fast bacilli were demonstrated in sputum the short course chemotherapy was instituted according to the National Tuberculosis and Leprosy Programme, that is streptomycin, Rifampicin, Isoniazid and pyrazinamide. They responded well to treatment, but some succumbed either of overwhelming disease or other opportunistic infections related to AIDS. In addition to drugs these patients were given surgical treatment accordingly that is subxiphoid pericardiostomy for those with pericardial effusion and tube thoracostomy or open chest drainage for those with thoracic empyema.

(b) **PYOMYOSITIS**

This was the next most common surgical manifestation of AIDS accounting for 19% of the patients admitted to the surgical wards. There was a tendency to multiple muscle group involvement or one muscle group being involved after another. Staphylococcus aureus was the most common organism isolated from pus cultures and the organisms were sensitive to erythromycin and cloxacillin. There was a very slow recovery of these patients even after drainage of their pus collections and appropriate antibiotic therapy. They tended to develop pus collections elsewhere in the body.

Adeloye in his description of pyomyositis stated that among the predisposing factors included chronic ill health, debility and trauma.[11] In our patients it was mainly the previously healthy young patients who were affected. Carswell in Uganda also noted a high incidence of pyomyositis among AIDS patients.[5]

However pyomyositis does not feature high as a manifestation of AIDS in series from other countries, although it is known that AIDS patients tend to suffer from organisms traditionally considered as "non-opportunistic". These include streptococcus pneumonia and staphylococcus aureus.[12] This would explain the high incidence of pyomyositis in AIDS patients. However, in the same period there were 9 pyomyositis patients who were HIV seronegative. So there is no significant difference in the incidence of pyomyositis among seronegative and seropositive patients in this study.

(c) **KAPOSIS SARCOMA**

This was the third most common presentation accounting for 9.5% of the patients. It is known that Tanzania is one of the countries where endemic Kaposis Sarcoma is prevalent.[11] The endemic disease is usually a disease of the middle aged and the elderly. It is slowly progressive, its evolution taking up to two years. Its prognosis is usually good except in the lymphdenopathic type in children and the florid and visceral type in adults which have a poorer prognosis.[11]

The HIV related Kaposis Sarcoma is aggressive, it tends to affect younger age group, its lesions are more widespread in both lower and upper extremities as well as the trunk and the mucous membranes; and all types of lesions may be present in the same patient. It has a higher mortality also.[1,7,11,12] In the United States aggressive Kaposis sarcoma was more commonly seen in HIV positive homosexuals, but in Africa no such a relationship has been found.[1] In Africa, Kaposis Sarcoma tends to be rapidly progressive associated with opportunistic infections and marked weight loss.[1] Nodular manifestations are common on the trunk, both upper and lower extremities and on the hard palate. Generalized lymphadenopathy due to Kaposis Sarcoma as well as gastrointestinal tract involvement are more common. A comparison of the endemic and the epidemic

HIV related Kaposis Sarcoma is presented in the next table.

Comparison between Endemic and Epidemic aggressive Kaposis Sarcoma

Factor	Endemic Type	Epidemic Aggressive Type
Age Group	30-40 years or older	Younger age group
Distribution of lesions	On skin of extremities, mainly legs	Extensive: On skin of extremities, trunk, face, mucous membranes.
Rate of Progression	Slow	Rapid
Lymph node involvement	Rare except in children	Common
Lung involvement	Rare	Common
Gut involvement	Rare	Very common
Prognosis	Fair	High mortality
Response to treatment	Fair	Short remissions with recurrences
Recurrences after treatment	Rare	Common

PATHOGENESIS OF KAPOSIS SARCOMA

Kaposis sarcoma is generally agreed to be a malignant tumour arising from the endothelial cells and it is multifocal rather than metastatic.[1,7,11,12] However the exact cell of origin is still unknown and the exact cause is still unknown, but it may be multifactorial with constitutional and exogenous factors playing a

role.[1,12] The role of cytomegallovirus has been implicated. Hence a combination of cytomegallovirus (CMV) infection and immunodeficiency predispose one to getting Kaposis Sarcoma[1,14] and CMV may act as an oncogenic virus.[17]

In this set of case collections most of the patients were young with two patients having had previous treatment for Kaposis sarcoma with radiotherapy and now presented with relapses. Most patients had multiple lesions including oral lesions with generalized lymphadenopathy and nodules on both the trunk and the extremities.

Patients with aggressive Kaposis Sarcoma are said to die either from overwhelming disease especially if they have pulmonary involvement or they may die from opportunistic infections.

One of our patients had Kaposis sarcoma and tuberculosis (pulmonary). A similar case has been reported from South Africa where a patient was found to have tuberculosis, Kaposis Sarcoma and lymphoma, all demonstrated in one lymph node.[15] All the three diseases are related to AIDS.

All our patients with Kaposis Sarcoma were sent to the Tumour Centre for radiotherapy. The response to therapy is generally good, but short term relapses are common.

(e) **LYMPHADENOPATHY**

Generalized lymphadenopathy is the commonest finding in patients with HIV infection, although often it is not noticed by the patients.[1] It is said that about a third of HIV infected patients fulfill the criteria for persistent generalized lymphadenopathy as defined by the centre for Disease Control (CDC), that is "enlarged lymph nodes equal to or more than 1 cm in diameter in two or more extrainguinal sites lasting for at least 3 months where other causes have been excluded".[1] Usually the nodes are non-tender, symmetrical and mobile.[1] Mindel[1] and Haye et al[15] argue that in the absence of any constitutional symptoms in HIV positive patients there is no indication for doing biopsy on such nodes. Lymph node biopsy is recommended, however, to exclude other important causes of lymphadenopathy which are now more common in AIDS patients. These include:

(1) Tuberculous lymphadenitis

(2) Lymphadenopathic Kaposis Sarcoma

(3) Lymphomas.

In lymphadenopathy due to AIDS one finds non-specific reactive hyperplasia.[7,10] However, sometimes one finds follicular depletion and this is said to be a poor prognostic sign.[12] When this type of reaction is seen in patients with persistent generalized lymphadenopathy rapid progression to AIDS follows.[12] The indications for lymph node biopsy as stated by Mandel[10] include:

(1) Presence of constitutional symptoms such as night sweats and fever.

(2) Painful nodes

(3) Assymetrical lymph node enlargement.

(4) Sudden increase in the size of the nodes

(5) Presence of Hilar lymphadenopathy on X-rays.

However I think that each case should be considered individually especially in developing countries where follow-up is difficult. One may miss the only chance of making an early diagnosis only to see the patient again with advanced disease.

In this series we had 12 patients presenting with lymphadenopathy, referred to the Surgical Department for biopsy. Of these patients the tissue diagnoses were equally distributed between Kaposis Sarcoma, tuberculous lymphadenitis and reactive lymph node hyperplasia. None was diagnosed as having a lymphoma. This fact has also been reported by Biggar[16] who noted that in Africa there is no association between AIDS and lymphoma despite the high incidence of Burkitts Lymphoma. Biggar postulated that this could be due to the fact that Burkitts lymphoma is a disease of childhood when HIV infection is uncommon. However in USA AIDS has been found to be associated with Hodgkins Lymphoma and Burkitts lymphoma.[17]

After histology results patients were treated according to the diagnosis.

Patients with tuberculosis were given the standard treatment according to the National Tuberculosis and leprosy control programme. This included streptomycin, Isoniazid and Thiacetazone.

Those who had kaposis Sarcoma were treated with radiation therapy at the tumour centre.

(f) **CUTANEOUS ULCERS**

There is scanty literature on cutaneous ulcers and AIDS. We had 6 patients who were admitted to the surgical wards with cutaneous ulcers. All ulcers were in the lower limbs having developed from small blisters. These patients were young and otherwise healthy looking, but their ulcers took a long time to heal. None of these ulcers were grafted. In three of the patients Pseudomonas pyocyaneus was isolated from wound swab cultures. None of them revealed fusobacterial or Vincents bacilli as commonly occur in Tropical ulcers. Patients with tropical ulcer are usually poor with poor nutritional status. Friction and minor trauma are described by Adeloye[11] as important predisposing factors. One wonders whether this is a variety of tropical ulcers but none of our patients had any predisposing factors mentioned above.

(d) **ANORECTAL CONDITIONS**

We had 6 patients who had various anal symptoms. Three patients had perianal ulcerations, one had pain on

defaecation but proctoscopic examination and digital rectal examination did not reveal anything although he kept on complaining of pain. Such cases have also been reported in literature where patients may present with non-specific anal and perianal complaints.[1] These are usually homosexual patients. Some forms of anal manifestations include:

(1) Anal discharge

This is usually due to proctitis being caused by gonorrhoea, chlamydia and herpes. Patients may even present with haematochezia due to perianal herpes.[1]

(2) Pain

This may be due to fissures, perianal haematomas, herpes or syphilitic chancres.[12,14]

(3) Perianal masses.

These include warts, skin tags, and perianal haematomas. Pre-existing perianal warts may become florid with HIV infection.[1,12,14]

MISCELLANEOUS CONDITIONS

Other conditions noted in our patients included:

(1) Herpes zoster which was in some cases recurrent

(2) Oropharyngeal candidiasis

(3) Skin rashes; especially in patients presenting with pyomyositis. In some cases the rash was

typical of infected scabies and responded to treatment with emulsion of Benzyl Benzoate. In most cases it was non-specific and refractory to treatment. A wide range of dermatoses are known to occur in AIDS patients.[1,12]

CONCLUDING REMARKS

We cannot escape from the fact that AIDS and HIV infection is a surgical problem, and we will continue, as surgeons, to see such patients.

Much more research is needed to answer many unanswered questions in terms of surgical manifestations of AIDS; but also in other aspects of the disease and surgery. For example what is the effect of major surgery on AIDS patients? What is the healing potential in these patients? Patients with cutaneous ulcers in this series were not grafted, but what would be the potential of graft take? What of organ transplant surgery in the era of AIDS? One hope that with time these questions will find answers.

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CHEST INJURIES AS SEEN AT MUHIMBILI MEDICAL CENTRE

SUMMARY

From January to December, 1989 14 cases of thoracic trauma (both penetrating and non-penetrating) were managed in the thoracic surgical unit of Muhimbili Medical Centre. Thirteen were males and only one was a female. The age range was 23 to 66 years with 12 patients being in the 20 - 40 year age group.

Nine cases had penetrating injuries while 5 cases had blunt thoracic trauma. Most patients had closed tube thoracotomy either for haemothorax haemopneumothorax or pneumothorax. None needed thoracotomy. One patient had laparatomy for other associated intraabdominal injuries where ruptured diaphragm was also repaired. All patients recovered and there were no deaths.

INTRODUCTION

Chest injuries are a common cause of hospital admission in our hospital and in some series from London they are the third commonest cause of hospital admission due to trauma after head and lower limb injuries.[1] In the United states of America 25% of annual traumatic deaths result from chest trauma.[2]

In the year 1989, 14 cases were admitted and managed in the thoracic unit of Muhimbili Medical Centre. This excludes paediatric cases which are managed by paediatric surgical unit and other chest injuries which did not need special care which were managed by other

surgical firms. A case history is presented in detail and the characteristics of the other patients is presented in the subsequent tables.

CASE HISTORY

J.H.C. (Reg No 587660) a male patient aged 26 years was admitted in hospital on 5/11/89 having sustained penetrating injury 6 hours back. He was a suspected bandit and was shot in the back of the chest by the police. He had not lost consciousness and had no breathing difficulties nor haemoptysis. He had no abdominal pain and had no other injuries. Clinical evaluation revealed a young man who was not pale but in pain. His pulse was 80 beats per minute, of good volume and his blood pressure was 100/70 mmHg. His respiration rate was 24 per minute and regular.

The trachea was central and the chest expansion was bilaterally symmetrical. There were two penetrating wounds anteriorly in the 5th intercostal space and posteriorly in the 6th intercostal space. The anterior wound was 2 x 3 cm in size, while the posterior wound was less than a centimeter. There was a dull percussion note on the left infrascapular region with reduced air entry in the same region.

The abdomen was soft, non-tender with no sign of free intraperitoneal fluid.

A diagnosis of penetrating chest wound with haemothorax or haemopneumothorax was made. The initial management was to cover the wounds with sterile gauze pads. A chest radiograph, frontal view, revealed minimal haemothorax on the left side; but no fracture ribs.

The patient was taken to the operating theatre where surgical toilet was done to the wounds, the muscles and subcutaneous tissues were closed but the skin was left open. A chest tube was inserted at the same sitting and 500 mls of blood was drained immediately.

On the second day the patient developed abdominal pain which was tender with guarding and rebound tenderness, but the blood pressure and the pulse remained stable. The patient was taken to the theatre and explorative laparatomy was done. At laparatomy he was found to have haemoperitoneum, a lacerated spleen , a lacerated left lobe of the liver which was no longer bleeding, and a laceration in the left dome of the diaphragm about 3 x 3 cm size. So splenectomy was done, the laceration in the diaphragm was repaired with nylon suture number '0' the liver laceration was left alone. The abdomen was irrigated with copious amounts of normal saline after sucking out all blood, and the abdomen was closed. The patient received one litre of blood intraoperatively.

The post-operative period was uneventful. The chest tube was removed after 72 hours as it was not draining

and chest radiograph showed full lung expansion. The patient was discharged 10 days later in good condition. He was seen one month later in the outpatient follow-up clinic and he had not problems.

This case serves the importance of suspecting intraabdominal injuries in all penetrating wounds below the 4th intercostal space. An explorative laparatomy should have been made on the first day.

There were 13 other cases of thoracic trauma (both blunt and penetrating). They are presented in the subsequent tables. There were 13 males and one female with age ranging from 23 years to 66 years.

Table I Nature of Trauma

Nature of Injury		Number of patients
Penetrating:	Bullet	3
	Assault	6
Blunt		5
Total		14

Note. There were more penetrating than non-penetrating injuries.

Table II: Number of Cases in relation to manifestations

Diagnosis	Left Side	Right Side	Total
Haemothorax	2	3	5
Pneumothorax	2	1	3
Haemopneumothorax	2	2	4
Flail chest	0	2	2
Diaphragmatic injury	1	0	1
Rib fractures	0	3	3
Fracture Clavide	1	0	1
Lung contusion	2	2	4
Total	10	13	23

Note:

(1) Some patients had more than one type of manifestation.

(2) Some cases of haemothorax or pneumothorax had no radiological evidence of chest cage injuries, that is no evidence of rib fractures.

Table III Other associated injuries

Nature of injuries	Number of Cases
Splenic laceration	1
Liver laceration	1
Fracture tibia/fibula	1
Total	3

Note: Splenic laceration and liver laceration occurred in the same patient.

DISCUSSION

Thoracic trauma is one of the major causes of admission due to trauma in this hospital. It has also been reported as the third commonest cause of hospital

admission due to trauma in a series from London[1] and accounts for 25% of annual civilian traumatic deaths in the United States.[2] With the increase in use of motor vehicles and alcohol abuse the incidence of thoracic trauma is likely to increase.

The distinction between blunt trauma and penetrating chest injuries is useful since it has important diagnostic and therapeutic implications. Most blunt chest injuries result from road traffic accidents[1,2,3] but could also result from industrial and domestic accidents such as falls, mining accidents or direct blows from assault. Blunt trauma tends to be associated with other multiple injuries such as head injuries, abdominal trauma, pelvic fractures or fracture of long bones.[1,2]

Penetrating injuries on the other hand, tend to produce more localized and sometimes predictable injuries, but occasionally lack of precise information on the nature of the weapon used, or on the circumstances and posture of patients at the time of injury may make it difficult to precisely predict the likely damage caused.[1]

Most of penetrating injuries result from assaults with sharp weapons or gun-shot wounds.[1,2]

In most series blunt trauma is more common than penetrating trauma[1,5] but in this study we had more penetrating injuries than blunt trauma; and most cases were due to assaults, either gun-shot or penetrating

sharp instruments. It could be that some of the more severe cases of blunt thoracic trauma which, are usually associated with other injuries died before arrival in hospital; and hence the low incidence; but it could also be that some cases were treated in other units since the thoracic unit does not handle all the chest injury cases in the hospital. More studies (including autopsy studies of all accident deaths) would help to get the true incidence.

PATHOPHYSIOLOGY

Chest injuries have been classified by Reid and Baird[6] into three classes based on one's ability to cough effectively and maintain satisfactory ventilation:

Class 1 - Arterial PO_2 and PCO_2 normal or reduced
- can cough effectively

Class 2 - Arterial PO_2 and PCO_2 reduced
- does not cough effectively
- impaired level of consciousness

Class 3 - Evidence of progressive respiratory failure with low pH and PO_2 with high PCO_2.

This classification is useful as it aids in planning management of the patient, but its limitation is that in some centres it may not be possible to do blood gases analysis.

Naclerio[4] suggest a "regional" classification which he calls a physiological approach. In this classification there are three groups:-

(a) Chest-wall injuries. These include bony cage and soft tissue injuries. Such injuries may result in:

1. Subcutaneous emphysema
2. Flail chest
3. Open pneumothorax

(b) Injuries to the pleura and lungs. These will result in:

1. Closed pneumothorax
2. Haemothorax or haemopneumothorax
3. Lung parenchyma injuries such as lung contusion, or aspiration pneumonitis.

(c) Mediastinal injuries. These may result in:

1. Mediastinal Emphysema
2. Cardiac Tamponade
3. Compressional atelectasis

The severity of chest injuries is influenced by various factors, the following being important as they adversely affect the outcome.

(a) Age. Both extremes of age fair badly.[1] The aged have a more rigid brittle rib chest cage rendering them more prone to multiple fractures, they also have low vital capacities, reduced lung compliance, impaired cardiac function and increased incidence of thromboembolism. The very young have a highly elastic rib-cage and may develop serious intrathoracic damage without rib fractures.[1,4]

(b) Patients with pre-existing lung disease, chronic bronchitis, emphysema and bronchial asthma or patients with pulmonary fibrosis. In general, patients with impaired respiratory reserve have a poor outcome.[1,4]

(c) patients with pre-existing cardiac disease; as any type of chest injury will in one way or another affect the cardiopulmonary status.

(d) Obese patients

(e) Patients with scoliosis or kyphosis

(f) Patients who have had previous thoracic surgery such as pneumonectomy or thoractoplasty.

The immediate pathologic effect of most thoracic injuries is hypoventilation resulting in tissue hypoxia and acidosis diminished cardiac output and pulmonary shunting. To what degree the respiratory function is deranged depends on the type of injury and the pre-morbid condition of the patient; for example a patient with multiple ribs and flail chest will have a more severe hypoventilation than a patient with simple rib fractures.

PATIENT INITIAL EVALUATION AND MANAGEMENT

The evaluation and management of a patient with chest injuries go hand in hand; because some treatment modalities are life saving and need to be instituted immediately. The most important steps are:

1. Establishment and maintenance of an adequate clear airway. This may need just proper positioning of the head and neck, or may need endotracheal intubation or even tracheostomy where necessary. Removal of foreign bodies in the oropharyngeal cavity should be done. Aspiration may be nasotracheal, bronchoscopic or through tracheostomy. Oxygen and mechanical ventilation may be necessary.

2. Cardiovascular support in terms of intravenous fluids with blood transfusion where necessary. cardio-pulmonary resuscitation with cardiac massage should be instituted immediately if there is cardiopulmonary arrest.

3. In case of massive haemothorax or pneumothorax with respiratory distress tube thoractostomy should be done immediately without waiting for X-ray films.

4. Any open chest wall injury must be closed. In such cases it is important to put a chest tube in order to prevent the development of tension pneumothorax after the open wound is closed.

5. Other management steps should follow the general management of any injured person including tetanus immunization.

SPECIFIC INJURIES

A. RIB FRACTURES

Rib fractures tend to occur at the point of maximum convexity that is the lateral chest wall, and they reflect the large amount of energy that has been imparted on the chest wall.[1,2] When the first rib or scapula are fractured one should suspect injury to the deeper structures including the great vessels and the bronchi, since such fractures occur when a very great force has been imparted on the chest.[4]

Patients with rib fractures usually present with localized pain on inspiration and localized tenderness. A chest radiograph is usually done not necessarily to confirm the presence of fractured ribs but mainly to rule out other intrathoracic injuries such as haemothorax or haemopneumothorax.[2,3]

Treatment includes analgesics and intercostal nerve blocks with local anaesthesia. Patients should be assisted to cough but any form of strapping is contraindicated.[3]

In this series we had only three cases of simple rib fractures, all of whom were managed conservatively with improvement. It is probable that most patients with simple rib fractures were either treated as outpatients or were managed in other surgical wards.

B. FLAIL CHEST

When three or more ribs fracture at two points, or when ribs fracture on both sides of the sternum the result is a flail segment which moves paradoxically with respiration. This paradox may not be apparent initially due to muscle spasm.[1]

Sometimes impaction of fracture fragments may temporarily stabilize the flail segment, but as the muscle spasm wears off and the segment loosens the flail becomes apparent.[1,3,4] The problems associated with the flail chest include the following according to Naclerio[4]:

(a) The paradoxical respiration results in useless to and fro movement of air across the carina between the two lungs. This is called the pendeluft phenomenon. Maloney et al[7] challenge this concept. They argue that the respiratory disturbance resulting from flail chest is due to a sharp reduction in the vital capacity; and the inability to drop the intrathoracic pressure to adequate negative level. This concept is also supported by Glinz[8] who says that the forced vital capacity drops to 40% of normal, and it continues to drop in the two days after injury, resulting in significant respiratory insufficiency.

(b) The mediastinum swings from side to side causing a decreased venous return and hence a reduction in cardiac output.

(c) Obstructive and compressive atelectasis occurs in the poorly ventilated lung.

(d) Lung contusion also commonly occurs in flail chest. Accumulation of secretions and ineffective coughing aggravate the situation.

The management of such patients include the following:

(a) Stabilizing the chest by sand-bags or placing the patient on the affected side.

(b) Pain relief by intercostal nerve blocks or epidural anaesthesia should be provided.

(c) Nasotracheal suction toclear the airway of secretions.

(d) In severe flail chest endotracheal intubation with intermittent positive pressure ventilation may be necessary till the chest wall stabilizes; which may take 7 to 10 days, and sometimes even up to three weeks.[1] When prolonged orotracheal intubation is needed tracheostomy may be necessary, but is often discouraged since the patient then needs intensive care which may be costly. In addition it is poorly tolerated by old patients or those with chronic bronchitis.[4]

(e) Interest is resurging in internal fixation of the unstable segments. This gets rid of the need for prolonged periods of ventilation, avoids the need

for tracheostomy and prevents permanent chest deformities.[1] In addition to providing mechanical stability operative stabilization provides very efficient pain relief.[8] Internal fixation can be achieved by Kirschner wires, intramedullary plates, small fragment plasts or metal clips.[8]

(f) Epidural analgesia has become one of the cornerstones in the management of flail chest.[8] Interestingly it has frequently been observed that paradoxical movement of the flail segment is diminished or may even cease completely after epidural analgesia.

In our series we had two patients with flail chests. One patient had an open pneumothorax also. The wound was closed and tube thoracostomy was done. He managed well without the need for positive pressure ventilation. However, he later developed empyema thoracis for which he continued to have antibiotics and the chest tube. This was removed when pus stopped draining and lung expansion was achieved as seen on control x-rays. The other patient was also managed conservatively and she improved.

PNEUMOTHORAX

Pneumothorax may be open or closed.

(a) Open pneumothorax

This condition results when a segment of the chest wall has been destroyed such that negative intrathoracic pressure sucks in air directly through the chest wall rather than through the trachea and bronchi.[3] Such wounds have also been termed "sucking chest wounds".

When the chest wall wound has a diameter greater than the laryngeal diameter the result is complete lung collapse with marked mediastinal shift with each respiratory effort.[4]

The management includes prompt closure of the defect with sterile dressing and venting the pleural cavity with tube thoracostomy to prevent possible resultant tension pneumothorax.

In this series one of the patients with flail chest had an open pneumothorax. He was managed by wound closure and tube thoracostomy with underwaterseal drainage. The three other cases had open pneumothoraces following penetrating chest injuries. All of these were managed by closure of the penetrating wounds and tube thoracostomy at the same sitting.

(b) **Closed pneumothorax**

This results from any direct or indirect communication of bronchus, bronchiole or alveolus with the pleural cavity resulting in collapse of the ipsilateral lung.[4] If there is a valvular mechanism preventing air escape then tension pneumothorax results. This is enhanced by forced expiration against a closed glottis for example during coughing or sneezing, all of which push air through the valve into the pleural space and raise the intrapleural pressure to more than the atmospheric pressure creating tension pneumothorax.[3,4] Effects of tension pneumothorax include:

(a) Collapse of the ipsilateral lung

(b) Mediastinal shift leading to angulation and collapse of the great veins. This results in serious interference with cardiac filling.[4]

In simple pneumothorax tube thoracostomy needs to be done after radiological documentation, but in tension pneumothorax tube thoracostomy needs to be done urgently without waiting for chest X-rays. Tension pneumothorax will be recognised by the following clinical features:

1. Progressive cyanosis
2. Respiratory distress
3. Tracheal deviation to opposite side
4. Hyper resonant percussion note on affected side
5. Absent breath sounds on the affected side.

When these features are seen then the insertion of a chest tube is an emergency. If this cannot be done immediately then a large bore needle (14 to 16 gauge) can be introduced in the chest cavity in the second or third intercostal space in the mid-cavicular line.[4] This creates a vent for air to escape from the pleural cavity and decreases pleural tension. It can be connected to underwater-seal; or in an emergency it can be attached to a flutter valve made from a finger of a glove.[4]

In this series we had no cases of simple or tension pneumothorax.

blood and air; and is usually effective in 85% of the cases.[3]

Sometimes two tubes can be inserted.[4] The initial volume of blood should be noted and subsequently hourly record of blood being drained should be recorded. If the initial blood loss is more than 1500 mls and the subsequent hourly blood loss is more than 200 - 300 mls, and shock persists despite blood replacement then thoracotomy is indicated; as there is usually a correctable lesion.[1,3,9]

In pneumothorax or haemopneumothorax a closed tube thoracostomy should always be done prior to administration of a general anaesthetic for other reasons or other injuries. This is to avoid development of tension pneumothorax during ventilation.[1]

Haemothorax may complicate into a clotted haemothorax which will need thoracotomy.[4] In this study we had four cases of haemopneumothorax all of which were managed by closed tube thoracostomy. None needed thoracotomy.

LUNG CONTUSION

This is defined as blunt injury to the lung parenchyma resulting in oedema and interstitial haemorrhage. Lung contusion usually results from decelerating or crushing injuries.[1] It may also occur in most patients with

flail chest (49% of all flail chest patients in one series had lung contusion)[2] and Simple rib fractures, but it may be an isolated injury without any bone cage injury especially in children.

Fluid and blood from ruptured vessels enter the alveoli, interstitial spaces and bronchi, and produces localized airway obstruction. Pulmonary compliance is reduced leading to hypoventilation and hypoxia.[2]

Pulmonary contusion may be confused with adult respiratory distress syndrome, or may co-exist with it. Infection and atelectasis may complicate pulmonary contusion and worsen the condition.

Physical examination is usually unremarkable, but on X-ray examination there are fluffy opacities or areas of patch consolidation. These findings in a patient presenting with haemoptysis should lead to the diagnosis of pulmonary contusion.

The management of such patients involves clearing of secretions by nasotracheal suction, avoidance of fluid overload and ventillatory support.[1,3] Positive end expiratory pressure (P.E.E.P) ventillation may be necessary in severe cases.[3]

There is a controversy as to the role of steroids but some authors claim that the severity of interstitial

oedema and haemorrhage may be reduced by early administration of methylprednisolone 500 mg every 6 hours.[1]

In this study we had two cases of lung contusion; one was associated with simple rib fractures and the other patient had multiple rib fractures with flail-chest. None of the patients received steroids, but both received antibiotics. One patient (the one with flail chest) developed empyema thoracis; the other patient developed pyrexia, but this subsided after completion of the antibiotic course.

TRACHEOBRONCHIAL INJURIES

These are rare but serious injuries. Many patients with disruption of the tracheobronchial tree do not survive and hence the true incidence is difficult to determine.[2] Three theories of causation have been proposed by Kirsch and Sloan[2]

(a) Forceful compression on the thorax pulls the lungs apart producing a tear by traction on the trachea and carina.

(b) High intrabronchial pressure with a closed glottis predisposes to rupture of the bronchial tree with a sudden impact on the chest.

(c) Shearing forces between the relatively static area of the cricoid cartilage and the carina may lead to tears with decelerating forces and steering wheel injuries.

Patients may present with:

- Features of airway obstruction due to accumulated blood and secretions
- Pneumomediastinum and cervical emphysema
- Haemoptysis
- Massive atelectasis which may be lobar or whole lung collapse.

Incomplete tears may heal with bronchial stricture formation, presenting later with recurrent atelectasis, infections and bronchiectasis.[4] Diagnosis is confirmed by bronchoscopy.[1]

Small tears can be managed without operation if the air leak is small. Tube thoracostomy should be done in presence of pneumothorax.

Large or complete tears require surgical repair and tracheostomy is often necessary. This aids in tracheobronchial toilet but may also facilitate bronchoscopic examination.[4]

In this study we had no patient with major airway injury.

DIAPHRAGMATIC INJURIES

Traumatic rupture of the diaphragm is a relatively rare finding in blunt thoracic trauma. In one series it accounted for 4% of all thoracic injuries.[2] It is most commonly due to motor traffic accidents; and it occurs as a result of abrupt increase in intraabdominal

pressure. It occurs in the weakest parts of the diaphragm along the embryonic fusion lines posterolaterally.[2]

Rupture of the diaphragm will lead to herniation of intraabdominal viscera into the chest cavity and this may be sudden or slowly.[2,3]

However, injuries to the diaphragm may occur following penetrating trauma. In such cases a high index of suspicion is needed; as the presence of other injuries may make one miss the diaphragmatic injury. Penetrating wounds as high as the 4th intercostal space could penetrate the diaphragm depending on the phase of respiration at the time of injury.[8,11] Small diaphragmatic injuries with herniation have a high chance of strangulating the herniated contents than a larger wound; and when strangulation occurs the mortality ranges from 25 - 60%.[11]

Chest X-rays may be normal, but in some cases they may reveal pneumothorax or herniated abdominal viscera. Most cases of blunt ruptures of the diaphragm occur on the left side, since the right dome is protected by the liver.[2]

Management is by repair mainly by abdominal approach. This has the advantage that one deals with other abdominal injuries at the same time, but thoracotomy is necessary for right sided diaphragmatic injuries or in late operations where adhersions of the herniated contents are suspected.

In this study we had one case which has been presented as an index case.

He had a diaphragmatic laceration on the left hemidiaphragm. This was repaired by abdominal approach where a lacerated left lobe of the liver and a ruptured spleen were also found. Splenectomy was done, the liver was left alone. He recovered uneventfully.

OTHER INJURIES

The following injuries may also occur in both blunt or penetrating injuries of the chest; but none of them was recorded in our study.

(1) Aortic ruptures and major vessel injuries. These are not seen commonly. Probably most such patients die at the scene of the accident. In one estimate 80% of such patients die at the scene.[2]

(2) Cardiac and pericardial lacerations. These are lethal injuries, mostly diagnosed at post-mortem. In one series there were 14 cases comprising 2% of all thoracic injuries; and none of them survived.[2]

(3) Cardiac contusions. These may be minor, and can present later on with arrythmias and conduction defects. If diagnosed, myocardial contusion should be treated as myocardial infarction.[2]

CONCLUSION

Chest injuries are a common cause of hospital admission at Muhimbili Medical Centre with penetrating chest injuries being more common than blunt chest injuries. It is possible that the most severely injured patients never reach the hospital; but adequate and efficient ambulance services are neccesary if we are to save those patients dying at the scene. Also efficient initial management at the casualty reception is important to save lives of those who reach the hospital alive. From the present study most patients needed tube thoracostomy only. Still one needs to be prepared for major surgery for the occasional patient who comes with severe injuries.

With the increase in the number of motor vehicles on the roads, we are likely to see increasing accidents and more severely injured patients.

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THORACIC EMPYEMA AS SEEN AT MUHIMBILI MEDICAL CENTRE

SUMMARY

Between January and December, 1989, 22 cases of thoracic empyema were managed by the thoracic surgical unit of Muhimbili Medical Centre. There were 18 males and 4 females giving a male to female ratio of about 5:1. The age range was from 18 to 61 years with 15 patients being in the 21-40 year age group. Nine patients had thoracic empyema with bronchopleural fistula. Pulmonary tuberculosis was the most common cause accounting for 13 cases.

The initial modality of treatment was closed tube thoracostomy for all patients. Six patients had open chest drainage and one patient with post-traumatic empyema had decortication after failure of tube thoracostomy. Those patients treated by open chest drainage and decortication did well. There were no operative deaths. Six patients died either of severe tuberculosis or overwhelming sepsis in the first one to three months of treatment. Hospital stay ranged from one to 12 months with 11 patients having stayed in hospital for at least 3 months.

INTRODUCTION

Thoracic empyema is a common cause of hospital admission at Muhimbili Medical Centre (MMC) in both medical and surgical wards. In 1985 thoracic empyema demanded more surgical attention than any other thoracic problem at

MMC.[2] In Nigeria thoracic empyema accounted for 30% of thoracic surgical cases treated over a period of 6 years.[3]

Many cases of thoracic empyema are due to inadequately treated pulmonary tuberculosis, pneumonia, chest trauma, liver abscesses and subphrenic abscesses.[2,4] In addition it continues to be an important problem in Africa largely because many people do not have easy access to medical care and therefore are treated late.[4] Thoracic empyema is a very distressing and disabling chest condition and cripples the chest wall and lung function.[5] It is important therefore for doctors to be conversant with proper management of this condition so as to reduce the morbidity and mortality and to bring the patient back to productive life as early as possible.

CASE PRESENTATION

G.M. (Reg. No. 572585) a male patient aged 42 years was referred to the thoracic unit in June, 1989 with one month history of right sided chest pain and breathlessness. He was referred as a known patient with pulmonary tuberculosis confirmed by sputum examination and had been on antituberculosis treatment for the previous nine months. One month prior to admission to the thoracic unit he had developed right-sided chest-pain associated with difficulty in breathing. He had on and off fever in the evening with

loss of weight. He had no history of haemoptysis and there was no history of tuberculosis contacts.

Clinical examination revealed a febrile wasted young man with a temperature of 38°C, dyspnoeic but not cyanosed. He had neither cervical nor axillary lymphadenopathy and had no clubbing of fingers. His trachea was shifted to the left, but the apex beat was not palpable. He had limitation of chest expansion on the right side. There was stony dullness on the right infrascapular region above which there was a resonant percussion note. There was diminished tactile vocal fremitus in the whole of the right hemithorax. There was diminished vocal resonance and absent breath sounds in the same region. He had a tender hepatomegally but the spleen was not palpable. The rest of the abdomen was normal.

A diagnosis of pulmonary tuberculosis with hydropneumothorax was entertained and this was confirmed radiologically. A diagnostic chest aspiration revealed straw coloured fluid.

Tube thoracostomy was done, the chest tube was inserted in the 7th intercostal space in the right axillary line, and this was connected to an underwaterseal system. Straw-coloured fluid was drained with a lot of air and a bronchopleural fistula was suspected to be present. Three weeks later the tube fell off and had to be reinserted; and this time frank pus was drained. Culture from this pus revealed klebsiella species. The

second tube fell off again after 6 weeks and the thoracostomy site went on leaking pus. Radiologically there was no lung expansion. The patient was planned for open chest drainage.

Meanwhile investigations done included:

1. An initial and subsequent radiographs of the chest (frontal view) which confirmed the hydropneumothorax.
2. Sputum examination by Ziehl Nielsen stain for acid fast bacilli was positive
3. A haemogram which was within normal limits apart from a high ESR of 120 mm in the first hour.
4. Serum creatinine - 118 micromoles/Litre
5. ELISA for HIV antibodies which was positive and Western Blot test which was also positive.

The patient was meanwhile put on anti TB drugs which included:

- Streptomycin 0.75 grams intramuscularly daily (as his weight was less than 50 kg)
- Isoniazid tablets 300 mg daily
- Rifampicin tablets 450 mg daily
- Pyrazinamide 2 grammes daily

He subsequently gained some weight and when his condition was thought to be relatively stable he was operated upon on 6.10.89 about 4 months since the first admission. A right sided rib resection and open chest drainage was done. Pus was drained and the skin flap was sutured to the thickened parietal pleura. The

window was subsequently dressed with gauze. It was refashioned one month later as it was threatening to close before complete obliteration of the cavity.

The patient continued with anti-TB drugs and he subsequently started to gain weight although he frequently succumbed to episodes of fever on and off due to other opportunistic infections as a result of the underlying HIV infection. Finally he was discharged to continue with outpatient treatment, having stayed in hospital for 5 months. He was seen at two outpatient follow-up visits when he was coming to collect his isoniazid and thiacetazone tablets as part of maintenance anti-TB therapy. His chest window was found to be healing well and the cavity was becoming smaller. Since then he was lost from follow up so the final outcome is unknown.

This case serves to illustrate several points.

1. Empyema thoracis with bronchopleural fistula can complicate pulmonary tuberculosis.
2. It illustrates one of the several modalities of treatment of thoracic empyema'
3. The morbidity due to thoracic empyema is substantial.
4. Concommitant HIV infection aggravates the long ill-health that these patients suffer from, that is it increases the morbidity.

Table 1: Age and Sex Distribution

Age Group	Male	Female	Total
11-20 years	1	1	2
21-30	7	2	9
31-40	6	0	6
41-50	3	0	3
Over 50 years	1	1	2
Total	18	4	22

Note: 1. Children under 10 years were not treated in the thoracic surgical unit.

2. More than half of the patients were in the 21-40 age group.

3. The male to female ratio was about 5:1.

Table II Cause of thoracic empyema

Underlying Diagnosis	Number
Pulmonary Tuberculosis	13
Complicating pneumonia	3
Penetrating chest trauma	2
Following thoracocentesis	2
Carcinoma Oesophagus	1
Ruptured amoebic liver abscess	1
Total	22

Note: Tuberculosis was the most common cause

Table III Microbiological pattern

Organism isolated	Number
Staphylococcus Aureus	7
Proteus and Klebsiella	5
Klebsiella alone	2
No bacterial growth	4
No record	4
Total	22

Note: 1. Staphylococcus aureus, proteus and klebsiella species were the most common organisms.

2. Anerobic cultures were not done.

Table IV: Treatment modalities

Mode of Treatment	Number
Thoracocentesis	0
Tube thoracostomy alone	15
Tube thoracostomy followed by open chest drainage	6
Tube thoracostomy followed by decortication	1
Total	22

Note: Tube thoracostomy and open chest drainage were the most common modalities of treatment used.

The other salient features among the patient included the following:

1. Nine patients had associated bronchopleural fistulae and one patient had an oesophagobronchopleural fistula following carcinoma of the oesophagus.
2. Seven patients were positive for HIV by ELISA, nine patients were negative and there was no record in six patients.
3. Thirteen patients received antituberculosis treatment.
4. Six patients died of overwhelming sepsis, or severe advanced tuberculosis. One of the deaths was the patient with oesophagobronchopleural fistula from carcinoma of the oesophagus.
5. Hospital stay ranged from 1 - 12 months with eleven patients having stayed in hospital for at least 3 months.
6. For those who had open chest drainage the outcome was good. There were no operative deaths. All the six deaths occurred in the first 1 - 3 months of treatment, none of them had open chest drainage.

DISCUSSION

Thoracic empyema is a collection of purulent fluid in the pleural space.[5] It may be focal for example interlobar, meadiastinal or intrapulmonary, or it may

involve the entire pleural space.[5] It may be acute or chronic, but the more precise classification is that of the American Thoracic Society which classifies thoracic empyema into exudative stage, fibrinopurulent stage and organizing stage.[5] This is a useful classification as it has descriptive and has important therapeutic implications.[5]

AETIOLOGY

Empyema of the thorax is usually secondary to a primary process elsewhere and not a primary pleural infection.[5] The aetiology of thoracic empyema according to Takaro et al [5] include:

(a) Chest wall lesions such as penetrating wounds or osteomyelitis of ribs.

(b) Pulmonary lesions such as pneumonia lung abscesses, bronchiectasis, tuberculosis or a new growth.

(c) Post-operative infection (postpneumonectomy or any other thoracotomy).

(d) From the oesophagus e.g. carcinoma of the oesophagus or oesophageal perforations.,

(e) From an intra-abdominal process e.g. amoebic liver abscess rupturing into the chest or a subphrenic abscess rupturing into the chest.

(f) From spinal column lesions such as paravertebral abscess or osteomyelitis of the vertebral bones.

(g) Secondary to systemic infections such as septicaemia.

A similar pattern of aetiological factors has been elaborated by several authors.[1,2,4]

Sometimes empyema may follow an infected tube thoracostomy for drainage of a non-purulent pleural effusion, haemothorax or for treatment of pneumothorax.[6] Post-pneumonectomy empyema is a specific entity since even its treatment poses specific problems due to the large empyema cavity.[7] However, if empyema is discovered when it is fully developed the primary pathology may be masked and difficult to identify.[5]

PATHOGENESIS

Thoracic empyema has been said to pass through three stages.

(a) **Exudative Stage**

This is the earliest stage. There is exudation of thin fluid with low cellular content. The visceral pleura and the underlying lung are still able to expand.

(b) **Fibrinopurulent stage**

This is also called the transitional stage. There is fibrin deposition both on the visceral and parietal pleura; and the fluid is rich in polymorpho-nuclear leucocytes. In some cases there may be adhersions or fusion of the lung to the chest wall. The fibrin deposits may be invaded by blood vessels from the

adjacent lung or chest wall with formation of granulation tissue and later on fibrous connective tissue converting the stage to the next one.

(c) **The organizing stage**

Here the exudate is quite thick. Fibroblasts have invaded the thickened fibrous layer on the visceral and parietal pleural membranes. So a mature or organized thoracic empyema consists of visceral and parietal layers of fibrous tissue on the lungs and chest wall respectively with pus in between them. There is usually a good cleavage plane between the wall of the empyema and the visceral pleura which allows decortication to be achieved.

Contraction of the fibrous tissue leads to loss of motility of ribs which are also drawn close together; the diaphragm is elevated and fixed and the mediastinum is drawn towards the affected side. The lung is encased in a rigid fibrous covering, is immobile and functionless (frozen chest).

In penetrating injuries suppuration is uncommon unless foreign material is retained in the pleural cavity; such as clothing or organ foreign bodies.[5]

However, presence of haemopneumothorax almost doubles the likelihood of developing empyema thoracis.[8]

Oesophageal perforation from a spontaneous rupture, instrumentation or anastomotic leakage leads to

mediastinitis which rapidly progresses to empyema. Subdiaphragmatic infection or sepsis usually leads to serous effusions, but these may progress to empyema. Sometimes an amoebic liver abscess or a subphrenic abscess may extend directly through the diaphgram or may spread through lymphatics to cause thoracic empyema.

An encapsulated empyema may burrow externally through the chest wall, this is then called empyema necessitatis.[5] Sometimes it can erode the lung or an old or fresh postpneumonectomy bronchial stump leading to a bronchopleural fistula. The reverse is also true, that a bronchopleural fistula from other causes may be complicated by an empyema, and when both are present it may be difficult to establish which started before the other. A bronchopleural fistula due to its persistent air leak potentiates infection by providing a route for constant innoculation of the pleural space and by promoting lung collapse.[9]

BACTERIOLOGY

The bacteriology of thoracic empyema has shifted from pneumococcus and streptococcus in the preantibiotic era to staphylococcus and gram negative organisms such as Pseudomonas, Klebsiella and Proteus.[5,9] In tuberculous empyema the fluid is thin and serous and tubercle bacilli may be difficult to isolate. In post-

pneumonectomy empyema similar organisms are isolated. Ereola et al[7] and Virkulla et al[11] found that staphylococcus aureus and Pseudomonas aeruginosa were the most common isolates in post-pneumonectomy empyema. Lema et al[2] analysing bacterial isolates from 59 patients found that in about half of the patients the cultures were sterile. However, anaerobic cultures were not done. Most of the positive growths had mixed infections.

In our patients the most common organisms were staphylococcus aureus and mixed infections of Klebsiella and Proteus species. In four patients there was no bacterial growth. However, anaerobic cultures were not done. In four patients there was no report of pus swab culture. In the patient with a ruptured amoebic liver abscess complicating into thoracic empyema the diagnosis was made on clinical grounds and the characteristic chocolate coloured pus. He also responded to treatment with metronidazole in addition to tube thoracostomy.

CLINICAL PRESENTATION

Thoracic empyema may present in three forms or stages: acute, sub-acute or chronic.

(a) **Acute Empyema**

The patient presents with

- Fever
- Cough with pleuritic pain
- Rapid, shallow respiration

- patient may be toxic.
- Chest signs are those of fluid in the pleural cavity with decreased chest expansion, stony dullness, decreased vocal fremitus and vocal resonance and absent breath sounds on the affected side.

The diagnosis is confirmed by chest aspiration.

(b) **Sub-acute Empyema**

This is a less severe form of presentation in a patient who has been on antibiotic treatment for pneumonia. It should be suspected in patients with slow resolution, slow convalescence or persistent fever following a pneumonic illness or where physical signs are inconclusive. Clinical chest findings are those of fluid in the pleural cavity as above.

(c) **Chronic Empyema**

This usually results from mismanagement of the acute stage. It may also occur resulting from failure of diagnosis of the primary condition or other underlying pathology, for example:

i) In the lungs; bronchiectasis, lung abscess or a tumour.

ii) In the pleura - a foreign body or actinomycosis.

iii) In the chest wall - ostemyelitis of ribs with a sequestrum.

The presentation may be as a localised empyema, a bronchopleural fistula discharging either continuously or intermittently through a bronchus; or empyema

necessitatis discharging either continuously or intermittently through the chest wall.

In this series 20 of the 22 patients were seen in the thoracic unit with chronic empyema; probably having had treatment in the acute or sub-acute stages in the medical wards or in the referring hospitals. Two patients with post-traumatic empyema were seen probably in the subacute phase, one of them was treated by decortication. Nine patients had associated bronchopleural fistula.

DIAGNOSIS

Chest X-ray films (frontal and appropriate lateral views) will show evidence of fluid but the definitive diagnosis is made by aspiration of pus from the pleural cavity. Failure to find pus may be due to:

i) too fine a needle for pus to pass
ii) selection of the wrong site for aspiration
iii) presence of fibrin tags or clots which block the needle.

Aspirated pus should be sent for microbiological studies to get bacteriological diagnosis and to get antimicrobial sensitivity tests done. The possibility of tuberculosis, actinomycosis or fungal infection should be considered if:

i) The response to treatment is slow
ii) a patient presents with chronic empyema or a discharging sinus

iii) the pus contains sulphur granules[5]

In amoebiasis complicated by thoracic empyema the "anchovy sauce" pus may be drained from the pleural cavity, as was the case in one of our patients.

Sometimes bronchoscopy may be necessary to exclude the possibility of an intrabronchial tumour as an underlying pathology.[5] The presence of a bronchopleural fistula may be confirmed by injecting methylene blue in the empyema cavity and observing the expectorated material for the blued dye[5] but in our case we made the diagnosis of bronchopleural fistula clinically when there was massive air leak in the underwaterseal bottle with the patient coughing or blowing.

MANAGEMENT OF THORACIC EMPYEMA

There are three objectives in the management of thoracic empyema[5]:

1. Control of the causative agent of the primary infection by the appropriate antimicrobial, antifungal or antiprotozoal agent.
2. Evacuation of the contents and obliteration of the empyema space.
3. Restoration of normal lung function by complete expansion of the underlying lung.

The modalities of treatment include the following:

1. Thoracocentesis
2. Thoracoscopy and irrigation

3. Closed tube thoracostomy
4. Decortication
5. Open chest drainage
6. Thoracoplasty 7. VATS - recent.

THORACOCENTESIS

This is usually the initial step in the management of thoracic empyema especially in the acute phase.[1,5] This procedure enables one to establish the diagnosis, to obtain a specimen for identification of the causative agent and to evacuate the contents. In children it may be the definitive treatment.[5,6] The needle should be of a wide bore and all the fluid as far as possible should be aspirated. A specimen of the aspirated fluid should be sent for microbiological studies including the special CO_2 filled tubes for anaerobic culture. If fluid re-accumulates one additional aspiration may be done, but if further reaccumulation occurs tube thoracostomy should be resorted to.[5] Thoracocentesis alone is said to provide adequate treatment in 9-12% of patients.[9]

THORACOSCOPY AND IRRIGATION

This is a method which is gaining popularity at present.[9] In this procedure thoracoscopy is done under general anaesthesia. Using the thoracoscope loculi are broken down under direct vision. The cavity is then irrigated with an antibiotic solution (eg Noxythiolin or 0.25% Neomycin solution)[10] and normal saline through

the thoracostomy tube which is inserted after removal of the thoracoscope until the effluent is clear. The effluent fluid is taken for culture and the irrigation is done twice daily until the effluent culture is sterile.[10] While the procedure may be useful in the management of early thoracic empyema it may not be applicable in chronic empyema with a thick cortex over an extensively destroyed lung and bronchopleural fistula.[7] The daily irrigation and its requirements of large volumes of irrigation fluid may limit its feasibility. In this study this procedure was not used in any of our patients.

CLOSED TUBE THORACOSTOMY

This is used for management of exudative and fibrinopurulent stage if antibiotics and thoracocentesis have failed to control the empyema.[5,9] The tube must be connected to an underwater seal to prevent pneumothorax and lung collapse. With this procedure accurate localization of empyema is even more important than in thoracocentesis.[5] The lowermost and most dependent part with the patient in the recumbent position is identified. X-rays in the appropriate planes are important and fluoroscopy with image intensifier if available is the most appropriate method for accurate localization.[5] The extent of the cavity

can, if desired, be established by injecting a small amount of contrast medium into the space and move the patient in various positions to displace the contrast medium by gravity and take X-ray films.[5]

Local anaesthesia is usually adequate. It is infiltrated in the subcutaneous tissues and the intercostal muscles. The track should be along the superior border of the rib to avoid injury to the neurovascular bundle in the inferior border. A skin incision 1.5 - 2cm long is made. The tube is then introduced in one of the three methods[5]:

i) a trocar and cannula

ii) a trocar and tube

iii) a tube with forceps.

The ideal tube should have a radiopaque line and multiple holes. The last hole should always be on this line so that one can confirm radiologically that all the holes are in the pleural cavity. The tube should be connected to an underwater-seal system. The patient is frequently re-evaluated clinically and radiologically for signs of lung expansion.

Tube thoracostomy was the initial form of management in all our patients. If after 6 weeks there was persistence of empyema and there was no adequate lung expansion other modalities of treatment were considered. Seven patients needed other forms of management as tube thoracostomy was deemed to have failed.

DECORTICATION

In chronic empyema the thick peel (cortex) that forms over the visceral pleura may trap the underlying functional lung to produce respiratory embarrassment. The peel may also cause failure of the empyema to resolve due to its rigid lining. In such cases decortication may improve function and lead to healing of the empyema.

The aim of decortication is to remove the fibrous wall of the empyema cavity both from the lung surface, the chest wall and the diaphragm.[11] It is especially useful in post-traumatic thoracic empyema where the underlying lung is basically normal.[11] It is also indicated in sub-acute empyema, but it is contraindicated in old and flail patient and also in post-pneumonectomy empyema.

Beneficial results of decortication as summarized by Senserig[12] include the following:

(a) Achievement of lung expansion after removal of the visceral peel

(b) Achievement of costal and diaphragmatic motion after removal of the parietal peel.

(c) Avoidance of prolonged tube drainage.

(d) Removal of additional pus pockets which may may not have been removed by tube thoracostomy.

(e) Thoracic contractures and scoliosis are avoided.

Decortication has been found to be effective in 80-100% of cases and it shortens hospitalization time.[9] In our cases one patient with post-traumatic empyema was treated by decortication and he did well.

OPEN CHEST DRAINAGE

In 1935 Eloeser[13] designed a method for treatment of tuberculous thoracic empyema by creating a "window" on the chest to drain pus. He made a flap of skin 2 x 2 inches on the skin a rib higher than the presumed "bottom" of the empyema, removed a segment of the underlying rib, drained the pus and then he sutured the tip of the flap to the thickened parietal pleura.[13] Various modifications of the ~~Eleoser~~ Eloeser window are being performed. The modifications are mainly in the size of the window and the number of rib resections. Takaro et al[5] recommend a window of at least 5 cm long with one or two rib resections. Oluwole[14] recommends a cuvillinear incision 8 - 10 cm long with excision of 3 ribs. Sampson[15] makes a vertical incision on the skin. In principle the opening should be large enough to allow incision of the thickened pleura, complete evaluation of empyema contents and breakdown of all loculi[5,9]. The procedure is completed by stitching the skin edges to the thickened pleura.

The procedure may be done either under local or general anaesthesia. If there is a bronchopleural fistula it

is best to do the procedure under local anaesthesia with the patient sitting in order to prevent empyema contents entering the opposite lung.[5] If the procedure must be done under general anaesthesia then an endo-bronchial tube should be used. The cavity is irrigated with saline and wet dressing applied. The cavity gradually granulates and may heal by scarring, or may be grafted either by pedicle graft or free skin grafts.[7]

Open chest drainage has been found to give satisfactory results by Lema et al[2] and Oluwole[14] in management of chronic thoracic empyema. It has also been found effective in management of post-pneumonectomy empyema by Ereola et al.[7]

In our study six patients were treated by open chest drainage and all did well. Follow up of patients treated previously by this procedure has revealed that the patients are in good general health without any residual disabilities.

THORACOPLASTY

This is a procedure where part of the chest wall is allowed to collapse by subperiosteal resection of segments of ribs usually the second to the sixth.[5.14] The aim is to reduce the pleural space, so a relatively conservative procedure can be done.[15] Although the procedure produces deformity and reduces lung function it may be the only option to control the problem of

dead-space in large empyema cavities with or without bronchopleural fistuale.[1,2,5,14] However, McCord and Vieira[14] found that among their patients who had thoracoplasty deformity was minimal and movements were almost normal.

In this study none of our patients was treated by thoracoplasty.

CONCLUSION

Empyema thoracis is a common problem and most cases are due to pulmonary tuberculosis. In our circumstances closed tube thoracostomy forms the main modality of treatment, and is adequate for most cases. Open chest drainage should be reserved for those cases which do not respond to closed tube thoractostomy.

In all cases the underlying disease should be treated appropriately.

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PERICARDIAL EFFUSION AS SEEN AT MUHIMBILI MEDICAL CENTRE

SUMMARY

From January to December, 1989 a total of 14 cases pericardial effusion were managed in the thoracic surgical unit of Muhimbili Medical Centre. The age range was from 11 years to 43 years with more than half of the patients (9 patients) being in the 20-40 year age group. There was a male preponderance with a male to female ratio of 6:1. Eleven cases were found to be due to tuberculosis either by histology from pericardial biopsy or from mycobacterial culture from pericardial tissue or fluid or both. Six patients were HIV. positive by ELISA and Western Blot. All cases were managed by subxiphoid pericardiostomy.

A case history of one of the patients is presented in detail, the other cases are summarized and literature on pericardial effusion is reviewed.

INTRODUCTION

Pericardial disease may manifest in one of the following ways: dry pericarditis, pericardial effusion, or constrictive pericarditis[1].

Pericardial effusion may develop in any type of acute pericarditis and it may be unnoticed until the onset of cardiac tamponade[2].

While much literature on pericardial effusion is available in developed countries minimal information is

available in developing countries and Tanzania in particular. However, over the past three years there has been an increase in the number of patients admitted with effusive pericarditis in the medical wards of Muhimbili Medical Centre[3].

In the year 1989, 14 cases of pericardial effusion were seen and surgically managed in the thoracic unit of the surgical department. A case history of one of the patients is presented in details and the characteristics of the other patients is presented in Tables I to III.

CASE HISTORY

A.H. (Case No. 578849) a 30 year old male watchman was admitted in the medical ward with history of shortness of breath on and off, fever and loss of weight, all of one month duration. He also had non-productive cough. His father had been treated for proven tuberculosis and was cured.

Clinical examination revealed an ill looking young man, dyspnoeic with distended neck veins and pedal oedema. He had a small volume pulse of 126 beats per minute, regular, with pulsus paradoxus. His blood pressure was 100/60 mmHg. The apex beat was not palpable. There was an increased area of precordial dullness, the heart sounds were distantly heard and there was no pericardial rub. The breath sounds were normal. There

was a tender hepatomegally but the spleen was not palpable.

A clinical diagnosis of pericardial effusion was entertained. A chest radiograph, frontal view was done and this revealed an increase in the size of the pericardial shadow and there was no hydrothorax.

An echocardiogram was done which confirmed the presence of a huge pericardial effusion with floating fibrin strands, and there was pericardial thickening especially at the apex.

Other investigations done included the following:

1. Haemoglobin 15.9 g/dl.
2. Serum creatinine 101 micromoles/L
3. ELISA for HIV-negative
4. Blood slide for parasites; this revealed malarial parasites for which the patient was treated with chloroquine 600 mg the first day, 600 mg the second day and 300 mg the third day in form of tablets.

After confirmation of the diagnosis it was decided to treat the patient by subxiphoid pericardiostomy.

The patient was taken to the operating theatre. He was placed on the operating table in a semi-sitting position. The procedure was done under local anaesthesia. The operation field was cleaned and drapped. One per cent lignocaine was infiltrated on either side of the xiphisternum down to the epigastrium. A skin incision 6-8 cm long was made

centred over the xiphoid process. The skin and subcutaneous tissues were deflected aside. The xiphoid process was identified, dissected out, held with Allis forceps and excised.

A plane was developed between the pericardium and the diaphragm. By both blunt and sharp dissection the diaphragm was reflected inferiorly. The sternum was elevated with a small angled retractor.

The bulging pericardium was held with Allis forceps and a piece of pericardium 4 x 4 cm on the antero-inferior aspect was excised. There were extensive fibrin strands attached to the epicardium.

Loculations were broken with a finger or suction nozzle. 1500 mls of haemorrhagic pericardial fluid was sucked out on the operating table. The pericardial cavity was explored for any further loculations. A large drainage tube was inserted in the pericardial sac and brought out through a separate stab incision of the inferior aspect of the main wound. This was connected to an underwater seal drainage bottle. The wound was closed with nylon No. 0". The skin was also closed with nylon No. 2/0 interrupted sutures. The patient was returned to the ward where he was nursed in a semi-sitting position. He got marked relief from the symptoms. The tube was removed 5 days later when it was found that no more fluid was draining out.

The pericardial tissue was sent to the laboratory for histopathology and mycobacterial culture. The pericardial fluid was also sent for cytological and microbiological studies including mycobacterial culture. After histological and mycobacterial culture which confirmed tuberculous pericarditis the patient was treated with anti-tuberculous drugs which included:

1. Streptomycin injection 0.75 grams intramuscularly daily (as the patient had body weight less than 50 kg).
2. Rifampicin tablets 450 mg orally
3. Isoniazid tablets 300 mg orally
4. Pyrazinamide tablets 2 grams orally.

These drugs were given daily. The plan was to continue with all the four drugs for two months after which the patient would continue with isoniazid 300 mg and thiacetazone 300 mg orally daily up to one year.

The patient initially progressed well. However, he later on developed constrictive pericarditis for which pericardiectomy was done with good results.

The characteristics of the other patients who were surgically treated for pericardial effusion are summarized in the next tables.

The main presenting symptoms were cough, shortness of breath and fever. In all patients electrocardiography showed low voltage, chest radiographs (frontal view) showed increased pericardial shadow and echocardiography which was done in all patients

confirmed the presence of pericardial effusion and the thickness of the pericardium.

All patients were offered subxiphoid pericardiostomy under local anaesthesia. The average duration of tube stay was 5 days.

Of the 14 patients, 11 had proven tuberculous pericarditis either by histopathology, Ziehl Nielsen stain or from mycobacterial culture from pericardial fluid and/or pericardial tissue. Seven patients had haemorrhagic fluid, all of whom were proved to have tuberculosis. More than half of the patients had fluid volumes greater than one litre.

In addition to subxiphoid pericardiostomy the patients with tuberculous pericarditis were given antituberculosis treatment. One patient had a very thin pericardium and he was actually found to have cardiomyopathy. One child had purulent pericarditis as a part of disseminated sepsis and pyoarthritis. However culture from the pericardial fluid revealed no bacterial growth (but anaerobic cultures were not done) and mycobacterial culture was negative.

Table I: Age and Sex Distribution

Age Group	Number (Males)	Number (Females)	Total
Under 20 years	2	1	3
21 - 30	7	1	8
31 - 40	1	0	1
41 - 50	2	0	2
Over 50 years	0	0	0
Total	12	2	14

Note:

(a) More than half of the patients (8) were in the 21-30 years age group

(b) The male to female ratio was 6:1 showing a male preponderance.

Table II: Macroscopic appearance of fluid

Type of Fluid	Number
Haemorrhagic	7
Serous	4
Purulent	3
Total	14

Note:
Half of the patients had haemorrhagic fluid, all of whom were tuberculous.

Table III Volume of fluid drained on the table

Volume of Fluid	Number
Less than 500 mls	2
501 - 1000	4
1001 - 1500	3
1501 - 2000	3
More than 2000	1
No record of volume of fluid	1
Total	14

Note:
More than half of the patients had fluid volumes greater than one litre.

Table IV: Summary of individual Cases) (Including HIV status by ELISA)

Case No.	Type of fluid	Volume (mls)	Histology	HIV Status
1	Purulent	2000	Non specific	Neg
2	Serous	1600	TB	Pos
3	Serous	1200	TB	Neg
4	Haemorrhagic	1500	TB	Neg
5	Haemorrhagic	1000	TB	Pos
6	Serous	1300	TB	Pos
7	Serous	No record	cardiomyo-pathy	Neg
8	Haemorrhagic	600	TB	Neg
9	Haemorrhagic	500	TB	Pos
10	Haemorrhagic	600	TB	Neg
11	Purulent	500	Non specific	Neg
12	Haemorrhagic	900	TB	Neg
13	Haemorrhagic	2000	TB	Pos
14	Purulent	2500	Non specific	Pos

Note: 6 out of the patients were HIV positive by ELISA and most of the tuberculous effusions were haemorrhagic.

DISCUSSION

Pericardial effusion may develop in any case of acute pericarditis. The development of pericardial effusion may be a silent process without symptoms,[2] but give rise to cardiac tamponade if the accumulating fluid causes an increase in intrapericardial pressure. The development of symptoms also depends on the rate of accumulation of the fluid in the pericardium.[1,4]

The overall incidence of pericardial effusion is not well documented, but it is described in various papers in Europe and America especially complicating Uraemia and malignant diseases.[1] It has also been described as a common cause of heart failure in South Africa.[5] In Tanzania documented reports at Muhimbili Medical Centre are available only from 1987 where in a period of 18

months 44 cases have been described; and the incidence has risen sharply since the epidemic of the acquired Immunodeficiency Syndrome (AIDS) started.[3]

AETIOLOGY

Pericardial Effusion may accompany acute pericarditis or may occur in a variety of chronic pericarditis.[1,6]

The causes of pericarditis include:

(a) Infectious agents
    1. Viral infections
    2. Tuberculosis
    3. Pyogenic bacteria
    4. Fungal infections

(b) Presumably immunologically mediated:
    1. Rheumatic Fever
    2. Post-myocardial infarction (Dressler's syndrome)

(c) Miscellaneous causes
    1. Uraemia
    2. Neoplasia
    3. Trauma
    4. Radiation

In developed countries viral pericarditis seems to be the most common type of pericarditis and it is usually due to coxsackie A and B virus and Echovirus-A.[1,4] As far as pericardial effusion is concerned the most common cause in these countries include uraemic pericardial effusion, chronic renal failure, metastatic

cancer and post-irradiation pericardial effusion according to most authors.[7,8,9] Alkan et al[7] in analysis of 18 cases of pericardial effusion who underwent subxiphoid pericardiostomy in USA found 9 cases having uraemic pericardial effusion, 5 cases metastatic cancer and 2 cases post-traumatic. Miller, Mansour and Hather[8] in their analysis of 76 cases of pericardial effusion found the most common causes being neoplastic (26 patients) uraemic (21 patients) idiopathic (15 cases) and tuberculous (6 cases). Similar results have been reported by Kottinen and Saklo in Finland[9] who found malignant conditions accounting for 12 out of their 19 cases with pericardial effusion.

However, in Africa literature is scanty except in South Africa where tuberculosis seems to be the most common cause. Hoffmeyer and Purry[5] in their analysis of 52 patients who underwent subxiphoid pericardiostomy found the most common causes as being active tuberculous pericarditis in 40 patients cardiomyopathy in 4 patients and cor pulmonale in 2 patients. Tuberculosis has also been found to be the most common cause of pericardial effusion in Mexico by Perez and Radillo.[10] In Tanzania at Muhimbili Medical Centre Cegielski in 1987[3] found tuberculosis to be the most common cause of pericardial effusion in his analysis of 44 cases. This has also been demonstrated by the present study which

reveals tuberculosis being responsible for 11 out of 14 cases of pericardial effusion.

PATHOLOGY

Acute pericarditis maybe:

1. Serous
2. Fibrinous or serofibrinous
3. Purulent or suppurative
4. Haemorrhagic
5. Caseous

1. **Serous Pericarditis**

This is commonly due to non-bacterial inflammation such as rheumatic fever, collagen diseases, tumours and uraemia,[6] but it may also occur in the early stages of bacterial or tuberculous pericarditis.[6] Viral pericarditis may also present as serous effusion but in many instances the cause of pericarditis remains unknown.

There is inflammatory reaction in the epi- and pericardial surfaces with scanty numbers of polymorphonuclear leucocytes, lymphocytes and histiocytes. Usually the volume of fluid varies from 50 to 200 mls, occurs slowly and rarely causes cardiac tamponade. The fluid resorbs when and if the underlying pathology remits.[6]

2. **Fibrinous and serofibrinous pericarditis**

This type of effusion is serous fluid mixed with fibrinous exudate; and is the most common form of

pericarditis. Common causes include myocardial infarction, uraemia, chest irradiation, rheumatic fever and trauma.[6]

The pericardial space becomes filled by fibrinous masses and on separating the pericardium from the epicardium fibrin is seen to cover both surfaces producing what is called "bread and butter" pericarditis. The fibrin maybe organized to obliterate the pericardial sac. The fibrosis leads to adhersive pericarditis which only rarely restricts cardiac action. The fibrin may be digested with resolution of the exudate. In conclusion serous or serofibrinous pericarditis rarely leads to serious sequelae.[6]

3. **Purulent pericarditis**

This form of pericarditis almost invariably denotes presence of bacterial, mycotic or parasitic invasion of the pericardial sac[6]. The organisms infect the pericardial cavity by the following routes:-

(a) Direct extension of infection from adjacent inflammatory conditions such as thoracic empyema

(b) Haematogenous seeding

(c) Lymphatic extension

(d) Direct introduction during cardiotomy.

Immunosuppression potentiates all of these pathways. The exudate rages from thin to creamy pus with volumes ranging up to 400-500 mls. Microscopic features are those of acute inflammatory reaction. Organization is

the usual outcome of this type of inflammation leading to constrictive pericarditis.[6]

4. **Haemorrhagic pericarditis**

Here the exudate is composed of blood mixed with fibrinous or suppurative effusion. It is most commonly caused by tuberculosis or malignant neoplastic involvement of the pericardial space. It may also be seen in bacterial infections. If the underlying pathology is a tumour malignant cells maybe found in the fluid. Malignant spread is mostly from the lung, breast carcinoma melanocarcinoma, lymphomas and leukaemia.

5. **Caseous pericarditis**

This is, until proved otherwise tuberculous in origin. Mycotic infections may infrequently evoke a similar reaction.

Tubercle bacilli involve the pericardium by direct spread from a tuberculous focus within the tracheobronchial lymph nodes but the infection maybe due to lymphatic or haematogenous dissemination from a non-contiguous focus. This form of pericarditis commonly leads to constrictive pericarditis later on.

In this series we had 11 cases of tuberculous pericarditis, 7 of which presented with haemorrhagic effusion, 3 cases had serous effusion and one case had purulent effusion. Similar observations have been made by Hoffmeyer and Purry in their series.[5] Out of 40

cases of tuberculous pericarditis 21 had haemorrhagic effusion, 11 had straw coloured fluid 4 cases had clear fluid and one had caseous fluid.

Striking in our patients is the fact that large volumes of fluid were drained ranging from 500 mls to 2,500 mls. In most other cases only small volumes were drained, except in series by Hoffmeyer and Purry[5] where 22 out of 40 cases had more than 500 mls drained. It could be that our patients were seen late; or that tuberculous pericarditis leads to more rapid accumulation of fluid as compared to other causes of effusive pericardial disease.

CLINICAL MANIFESTATIONS

Pericardial effusion may cause no symptoms at all.[1] The development of symptoms depends on the rate of accumulation of fluid and hence the rate of increase in pericardial pressure.[1] Some patients present with chest pain or chest-tightness due to stretch of the pericardial sac.[1,4] Patients may present with features of cardiac failure, that is cough, dyspnoea and swelling of legs.[4]

Physical examination may reveal distant or muffled heart sounds and rhales maybe heard over the lung fields secondary to lung compression.[4]

A paradoxical pulse with a fall in systolic blood pressure greater than 10 mmHg during inspiration may be present. This is because during inspiration all blood

vessels of the lungs increase in size due to increased negative pressure in the thorax. Therefore blood collects in the lungs and the stroke volume and pulse strength decreases.[11]

INVESTIGATIONS AND DIAGNOSIS

The presence of pericardial effusion apart from physical findings will be suspected on visualising an increased pericardial shadow on a frontal chest x-ray film. The main condition to be differentiated from is cardiomegally from other causes such as cardiomyopathy. Investigations to be done include:

1. Chest x-ray-frontal view. This will show an enlargement of the cardiac silhouette, but this will be seen when there is at lease 250 mls of fluid in the pericardium. Fluoroscopy may also be helpful.
2. Electrocardiography. This shows a low QRS voltage and flattening of T-waves.
3. Echocardiography. This is the most reliable non-invasive procedure for the diagnosis of pericardial effusion. It will indicate the amount of pericardial fluid, the present or absence of loculations and presence or absence of pericardial thickening.

Microbiological diagnosis can be made from culture of the pericardial fluid and histological diagnosis can be made from studies of the pericardium.

In this series all patients had a frontal chest X-ray film, an electrocardiogram and echocardiogram done before undergoing surgical treatment. In addition pericardial biopsy for histology and mycobacterial culture were done in all cases.

TREATMENT

If an effusion has been diagnosed but there are no significant haemodynamic changes there is no indication for treatment.[1,4] Sometimes the effusion may completely resolve. The patient should be watched for features of cardiac tamponade that is:

- tachycardia
- paradoxical pulse
- elevated jugular venous pressure
- fall in blood pressure
- other signs of deterioration.

When there are features of massive effusion with tamponade then the patient needs to be treated. The management maybe by:

- pericardiocentesis
- pericardiotomy
- pericardiectomy

These procedures are aimed at relieving the tamponade as well as providing a specimen of fluid for diagnosis. In case of pericardiotomy and pericardiectomy, pericardial tissue is also available for histological diagnosis.

PERICARDIOCENTESIS

This can be done either through a left parasternal approach between the 4th and 5th intercostal space or through the subxiphoid route at the costo-sternal angle.[4] It is done under local anaesthesia. An electrocardiographic lead can be attached to the aspiration needle by a small clip. This will indicate on the ECG screen if the heart is accidentally touched.[4]

The needle is directed at 30° - 45° angle posteriorly towards a point midway between the scapulae. If blood is aspirated from the ventricles it will clot. Blood from the pericardium does not clot due to defibrination from constant myocardial contractions.[4]

Laceration may occur to the ventricles, coronary arteries, internal mammary arteries, the lungs or pleura.

Limitations of pericardiocentesis:

Pericardiocentesis has both diagnostic and therapeutic limitations:

(a) Diagnostic limitations

False negative (dry tap) may occur if there is a loculated effusion localized posteriorly.

(b) Therapeutic limitations

It needs to be done repeatedly as fluid may re-accumulate.

PERICARDIOTOMY

This is the creation of a "window" in the pericardium and can be done through various approaches:

(a) **Anterior approach**

This was originally done by resecting the left 4th, 5th or 6th costal cartilages and the pericardium was then opened, but this did not provide adequate drainage of fluid unless the patient laid prone; so the seventh costal cartilage was often removed.[2] Later on it became known that the absorptive capacity of the pleural membrane was 3 times as great as the pericardium. So anterior pericardiotomy was done together with formation of a pleuropericardial window.[2]

(b) **Posterior approach**

This was an attempt to secure an efficient postural drainage and an incision was made in the posterior wall of the pericardium, but this necessitated a lateral thoracotomy. So this type of procedure has not been widely practised.

(c) **Inferior approach (subxiphoid pericardiostomy)**

This method was first described by Larrey in 1929 though he himself did not perform it. In 1970 Fontanelle redescribed it and at present it seems to be the most widely accepted method in management of

pericardial effusion.[2,4,5,7,9,12,14] The procedure is as described in the management of the index case but basically it involves the excision of the xiphoid process, separation of the pericardium from the diaphragm and creation of a pericardial window about 4 x 4 cm. The pericardial fluid is drained and a tube is inserted in the pericardial cavity and is connected to an underwaterseal bottle. This tube stays on average for 4-5 days until when no more fluid is draining.

Several authors have indicated their preference for this procedure in view of its advantages over pericardiocentesis and over anterior or left lateral thoracotomy.[2,5,7,14] The advantages have been well summarized by Fontanelle[14] but have been expressed by other authors as indicated above. They include:

(a) The procedure can be done under local anaesthesia. This is especially advantageous as most of these patients maybe too sick to tolerate general anaesthesia. However if desired general anaesthesia may be used.

(b) The procedure offers an opportunity for direct observation of the effusion, the pericardium and the pericardial cavity. If there are multiple loculations they can be broken under direct vision.

(c) The pericardial cavity can be completely drained without pleural contamination.

(d) A pericardial drainage tube can be easily placed in the pericardial cavity under direct vision. This may or may not be connected to a suction apparatus.

(e) It eliminates the uncertainty as to whether one is in the pericardium and not in a cardiac chamber if blood is aspirated.

(f) It provides an opportunity for obtaining adequate amount of fluid for diagnostic studies; and an adequate amount of pericardial tissue for histological diagnosis.

(g) It can be repeated if necessary and pericardiectomy can still be performed later on if indicated.

(h) It is simple enough to be done by a general surgeon with limited facilities.

(i) Small pericardial effusions are not missed by this procedure; so it can be used in early management of pericardial effusion.

(j) Trauma to the heart is very unlikely as the procedure is done under direct vision. If it occurs it can be treated immediately.

Complications of Subxiphoid pericardiostomy

These are few, but have been described. They include:

(a) Trauma to the heart. This may occur if the pericardial fluid is minimal; or there is an

adherent pericardium to the epicardium.

(b) Pneumothorax. The pleural lining may be accidentally punctured.

(c) Wound sepsis may occur; especially if the tube is left for too long.

(d) Transient arrythmias have been described.

In this series all patients had subxiphoid pericardiostomy. One patient died on the first postoperative day, but no cause of death could be ascertained (autopsy was done) at least none attributable to the procedure.

Three cases developed wound sepsis, they were treated with appropriate antibiotics after pus culture and sensitivity results.

PERICARDIECTOMY

This has been used in the treatment of tuberculous pericarditis and suppurative pericarditis where there is an increased risk of developing constrictive pericarditis.[2] However, it is being performed less and less as it is considered to be too extensive for a condition which can be managed by a less extensive procedure. Nevertheless Takaro et al consider early pericardiectomy the treatment of choice for purulent pericarditis.[15] In this series none of the patients received this form of treatment.

CONCLUSION

Pericardial effusion is an increasing problem, especially now with the AIDS epidemic. Subxiphoid pericardiostomy is a simple method for managing such cases which can be done even at regional level. However; patients need to be followed up so that for those who develop constrictive pericarditis further treatment such as pericardiectomy can be offered. With 11 out of 14 patients having tuberculous aetiology, a question arises as to whether it would be justified to start anti-tuberculosis treatment for all patients with pericardial effusion while waiting for histology or mycobacterial culture results. As this series had only 14 patients further studies are needed before justifying such a decision.

The other lesson to learn is to have pericardial effusion in mind in a patient with AIDS who develops shortness of breath and engorged neck veins. On the other hand AIDS should be ruled out in a patient with pericardial effusion.

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CARCINOMA OF THE URINARY BLADDER AS SEEN AT MUHIMBILI MEDICAL CENTRE

SUMMARY

Between December, 1989 and April 1990 the author during his residency in the Urology unit of Muhimbili Medical Centre attended to ten cases of histologically proven carcinoma of the urinary bladder. Six of these cases were males and four were females giving a male to female ratio of 3:2. The age range was between 21 and 62 years with 5 patients being more than 50 years old. Only two patients presented with T_1 and T_2 disease, the rest of the patients presented with advanced disease between T_3 and T_4. Seven of the ten patients had squamous cell carcinoma, two cases had transitional cell carcinoma and one had a mixed histology of transitional and adenocarcinoma. Patients with T_3 or T_4 disease were treated by palliative radiotherapy and the patient with T_1 disease was treated by transurethral resection.

INTRODUCTION

Carcinoma of the Urinary bladder is the second most common urological malignancy in the USA after carcinoma of the prostate.[1]

It has a peak age incidence of 50-60 years and a male to female ratio of 4.5:1.[1,2]

In the United States bladder carcinoma accounts for 3% of all cancer deaths.[2] In Tanzania carcinoma of the

urinary bladder is the commonest urological malignancy accounting for 40% of all urological cancers and 3.3% of all registered malignancies.[3]

While worldwide the commonest histological type of carcinoma of the bladder is transitional cell type there seems to be a geographical variation. In Tanzania the most common histological type is squamous cell carcinoma accounting for up to 64% while transitional cell carcinoma accounts for 12% of all cases[3] Similar histological distribution has been found by Lucas in Malawi[4] and Cairns in Zambia.[5] In all these areas squamous cell carcinoma has been found to account for 60-80% of bladder cancer. The disease also seems to coincide with the schistosoma haematobium belt.

While in developed countries most patients report early for treatment in most African countries, Tanzania included, most patients report late when the disease is advanced and hence the results of treatment are poorer and the mortality is higher.[3,4,5]

CASE PRESENTATION

J. S (Reg. No. 590800) a male, aged 51 years was admitted in the urology unit of Muhimbili Medical Centre in December, 1989 with history of haematuria of one month duration. The patient had been well till one month prior to the date of admission when he noted that he was passing bloody urine. He was having full-stream

haematuria. There were no shreds, clots or pain. He had no episodes of retention of urine and no frequency. He had no previous episodes of haematuria in the past and had no history of being treated for schistosomiasis. He was a businessman in Mwanza region but he originally came from Moshi, both areas being schistosomal endemic sites. He took moderate amounts of beer but did not smoke.

Physical examination revealed a middle aged man who was not pale and he was afebrile. His blood pressure was 150/90 mmHg and a pulse rate of 80 beats per minute, and he had no peripheral lymphadenopathy. The respiratory and cardiovascular systems were essentially normal. Abdominal examination including the genitalia were normal. Digital rectal examination was normal.

A clinical diagnosis of carcinoma of the urinary bladder was entertained and the patient was subjected to the following investigations:

(1) Urine analysis
   - Specific gravity - 1030
   - Albumin - ++
   - Microscopy revealed leucocytes and red blood cells but no schistosomal ova.
   - Cytology - negative for malignant cells.

(2) Haemoglobin 15.4 gm/dl

(3) Serum creatinine 76 micromoles/L

(4) Intravenous urogram was normal.

(5) Cystoscopy revealed a papillary bladder tumour on the left ureteric orifice. There was no evidence of previous bilharzia.

Bimanual examination under anaesthesia revealed a normal bladder wall and no other palpable masses.

Biopsy was taken at the same sitting. So he was staged as having a $T_1N_XM_X$ bladder tumour.

Histology revealed a papillary structure with normal transitional epithelium with no features of malignancy.

The patient was subjected to transurethral resection of the tumour two weeks later. The tumour was resected leaving a normal bladder margin. The resected material was submitted for histological examination which revealed a transitional cell carcinoma grade I with no features of stromal invarsion. Four weeks later the patient was subjected to cystoscopy where a residual tumour was seen. This was completely fulgurated. He was then discharged and was advised to come back for a repeat cystoscopy in 3 months time. The patient was again seen at follow-up 4 months later. He was assymptomatic and repeat cystoscopy revealed a bladder which appeared normal. He was discharged, and was due for a follow-up cystoscopy every six months for two years and then annually. The other cases are summarised in the subsequent tables.

Table I: Age and Sex Distribution

Age Group	Males	Females	Total
Up to 20 yrs	0	0	0
21-30	0	2	2
31-40	1	1	2
41-50	1	0	1
51-60	2	0	2
Above 60	2	1	3
Total	6	4	10

Note: Half of the patients were more than 50 years of age

Table II: Primary Tumour status at presentation

Tumour Stage	Number of Patients
T15	0
T1	1
T2	1
T3a	1
T3b	2
T4a	3
T4b	2
Total	10

Note: 8 out of 10 patients had more than T3 status

Table III Histological distribution

Histology	Number
Squamous cell	7
Transitional cell	2
Transitional and Adenocarcinoma	1
Total	10

Note: Squamous cell carcinoma was the most common type occurring in 7 out of 10 patients.

DISCUSSION

Carcinoma of the urinary bladder continues to be a problem in Tanzania. Nkulila and Phillip[6] found that carcinoma of the bladder accounted for 4.3% of all urological admissions at Muhimbili Medical Centre in 1977/78 while in 1978/79 it accounted for 8.7%. Kirei[3] in a retrospective study over a 10 year period from 1974-1983 found that carcinoma of the bladder accounted for 40% of all urological cancers and 3.3% of all registered cancers in Tanzania. Cancer of the bladder has also been found to be prevalent along the coast and around Lake Victoria where schistosoma haematobium is also prevalent.[7]

While in the USA and Europe transitional cell carcinoma is the most common type[2,8,9] in Tanzania and most other African countries squamous cell carcinoma seems to be the most common type. This has been reported in series from Egypt, Malawi and Zambia where squamous cell carcinoma accounts for 70 - 80% of all bladder cancers in contrast to the USA and Europe where squamous cell carcinoma accounts for less than 5%.[3,4,5]

Among our patients we found the same situation with 7 out of the 10 patients having squamous cell carcinoma. We found more than half of the patients were in the 50-60 year age group, which is the peak age incidence from most series. The male to female ratio from most series is 3:1. This has also been reported by Kirei in

Tanzania.[7] We had a male to female ratio of 3:2 but the number of the patients studied is too small to form a basis for discussion.

AETIOLOGY

In 1894-1895 Rehn first raised suspicion that bladder tumours could be caused by chemical carcinogens.[10] Since then further studies have established the role of chemical carcinogens in bladder cancer. It is now established that the following are among the aetiological factors for carcinoma of the urinary bladder.

(1) **Industrial Carcinogens**

The most potent among the industrial carcinogens is beta napthylamine but other related compounds are also strongly carcinogenic. These include alpha-napthylamine and benzidine.[10] These chemicals are used in rubber and cable industries. Other carcinogenic chemicals are the intermediate compounds in the synthesis of azo dyes and pigments. Workers in textile and a niline dye industries are exposed to these compounds exposing them to higher risk of developing bladder cancer.[2,10] These chemicals enter the body through the skin and food and their metabolites are excreted into the urine as orthohydroxylated metabolites complexed to sulphates or glucuronic acid. The glucuronides are split in the urinary bladder to release the active carcinogens.[2]

(2) **Environmental exposure to potential carcinogens**

In areas where schistosoma haematobium is endemic carcinoma of the bladder is also exceedingly prevalent and is one of the most common malignancies.[10]

It has also been found that carcinoma of the urinary bladder associated with urinary bilharziasis is the squamous cell type.[2-5,7,10] The following are among the reasons to explain the association between squamous cell carcinoma and urinary bilharziasis:

(a) Mechanical irritation of the bladder epithelium by schistosoma haematobium ova together with chronic inflammation leads to squamous metaplasia.

(b) Bilharzia toxins produced both by miracidia and worms are said to contribute to the process of carcinogenesis.[7]

(c) Urinary retention theory; that is bilharzial cystitis is said to cause destruction of the muscle coat leading to interference with complete emptying of the bladder. Carcinogens in the urine then have longer time to act on the bladder. Recently it has been found that high urinary nitrite is associated with squamous cell carcinoma secondary to urinary schistosomiasis. When urinary infection is associated with high urinary nitrite there is production of nitrossoureas which are proximate carcinogens which lead to production of squamous cell carcinoma.[11]

3. Smoking has also been implicated and smokers have a two to four-fold risk of getting bladder cancer and the greater the number of cigarettes smoked the higher the risk.[2,10]
4. Excessive alcohol consumption, coffee drinking and artificial food sweeteners such as saccharin have all been implicated in the causation of bladder cancer.[2,10]
5. Excessive accumulation of tryptophan metabolites which include 3-hydroxykynurenine and 3 hydroxyanthranillic acid are said to enhance chemical carcinogenesis.[2,3,15]
6. Drugs such as phenacetin and cyclophosphamide have also been incriminated in the causation of bladder cancer.
7. Urethral strictures have also been found to complicate into carcinoma of the urinary bladder.[13]

PATHOLOGY

Carcinoma of the urinary bladder may be transitional cell, squamous cell, adenocarcinoma or anaplastic.

(1) **Transitional cell carcinoma**

This type of bladder cancer may manifest itself as carcinoma in situ, superficial bladder cancer or invasive lesions.

(a) **Superficial bladder cancer**

This type of tumour may manifest macroscopically as exophytic polypoid lesions which grow into the bladder cavity. They may be pedunculated with a stalk but sometimes they may have a broad base[1,2,8] The area around the tumour is usually oedematous with dilated blood vessels. The most common sites for these tumours are the trigone and the lateral walls of the bladder. These tumours have a propensity for recurrence after resection in 50-70% of patients.[18]

The prognosis of these tumours depend on the differentiation. Well-differentiated (G1) and moderately differentiated (G2) tumours have a better prognosis compared to the poorly differentiated (G3) tumours which have a poorer prognosis. In these (G3) tumours the tumours can advance beyond the lamina propria with muscle invasion. The may be invasion of the lymphatics with distant metastasis.

(b) **Invasive transitional cell carcinoma**

These tumours are always solid and they show infiltration or invasion of the basement membrane or other contiguous structures. They are usually large, broad-based, with ugly or ulcerated appearance.[8] They have a higher incidence of associated metastases especially if they are G3

tumours.[8,12]

(c) **Carcinoma in Situ**

This term is used to describe a spectrum of flat and papillary epithelial lesions of the bladder.[12] There is thickening of the mucosa by proliferation of the basement membrane. These cells show moderate to severe degrees of anaplasia. There may be abundant abnormal mitotic figures. There is loss of cell cohesiveness as manifested by widening of intercellular spaces and separation of cells from the basement membrane. This may explain the tendency of carcinoma in situ to shed a large number of cells in urine[12] a fact which can be made use of in examining urine for cytology.

It is generally thought that carcinoma in situ is an irreversible lesion, and it is a precursor of invasive carcinoma; but its biologic and clinical significance is controversial, and the latent period prior to development of invasive disease is unpredictable.[15] The unpredictability of the natural history of carcinoma in situ has a big impact on its management as will be seen later.

(2) **Squamous Cell Carcinoma**

Squamous cell tumours tend to be solid in consistency and are nearly always associated with muscle invasion. They are most common where bilharzia is prevalent

although they can occur secondary to chronic bladder irritation by vesical calculi.[1,8] In squamous cell carcinoma associated with bilharziasis mechanical irritation of the bladder epithelium by the ova, chronic inflammation and toxins produced by the worms and miracidia may account for carcinogenesis.[7] Among the epithelial changes which occur in chronic urinary bilharziasis is squamous metaplasia which leads to carcinomatous change and hence squamous cell carcinoma.[13]

This type of carcinoma is the most common type in most African countries accounting for 60-80% of bladder cancers as per series reported in Tanzania, Malawi and Zambia.[3,4,5,7]

(3) **Adenocarcinoma**

This type of tumour is uncommon accounting for 1% of bladder tumours, and it arises in the fundus of the bladder at the urachal remnants or primarily in the trigone. Adenocarcinomas arising in the trigone of the bladder have to be differentiated from primary prostatic adenocarcinoma invading the bladder.

CLINICAL PRESENTATION

Total painless haematuria is the most common presenting symptom and it should be regarded as indicative of bladder carcinoma in areas where this tumour is common until proven otherwise.[8] Cases with initial and terminal haematuria should also be adequately

investigated so as not to miss any case.[10] Bleeding may be massive with clots leading to clot retention. Infection may lead to symptoms of cystitis with frequency, dysuria and occasionally retention. Sometimes poor-stream and urinary retention may occur due to bladder-neck involvement by the tumour.[1] Tumour necrosis and slough may result in tissue fragments being passed in urine as shreds. If the tumour invades or obstructs any ureteric orifice there may be pain in the lumbar region due to pyelonephritis or hydronephrosis. In bilateral ureteric involvement or in bladder neck involvement patients may present with features of uraemia.

Local spread may lead to rectal or pelvic pains and fistula formation such as cystocolic or cystocutaneous fistulae. Venous and lymphatic obstruction may lead to unilateral limb oedema. In late cases there may be a palpable suprapubic mass which may also be felt on vaginal and rectal examination. Weight loss, anaemia and bone pains may occur with advanced disease. On the other hand flat carcinoma in situ may present only with frequency, urgency and perineal pain. So unless a high index of suspicion is used such cases may be missed.

DIAGNOSIS

This is established by physical examination, laboratory investigations intravenous urography, cystoscopy combined with bimanual examination under anaesthesia and biopsy.

Physical examination may be unrewarding in early cases. In advanced cases one may palpate a suprapubic mass or detect features suggestive of metastatic disease such as unilateral limb oedema.[8,10]

Urine should be examined microscopically to rule out other causes of haematuria such as schistosomiasis. Urine cytology should be done to try to detect presence of malignant cells. Although a negative cytology does not rule out malignancy a positive cytology suggests it. False positive cytology may occur in urinary stone disease.[8] Cytology will pick up bladder neoplasms in up to 60% of cases overall.[14]

Blood should be examined for haemoglobin estimation, white cell count, serum creatinine and urea and serum electrolytes.

Intravenous urography is important. In ureteric involvement hydronephrosis may be demonstrated, and a filling defect in the bladder may be seen in the cystogram phase.[8,10]

Chest X-ray should be taken to rule out metastatic disease and if there are abnormal bone pains skeletal survey radiographs should be done including a bone scan if indicated.[10]

Urethrocystoscopy is the mainstay of diagnosis. The urethra is examined first and then the bladder should

be examined systematically noting with accuracy the site, shape and number of tumours. Adequate biopsy material should be obtained.[10]

After urethroscopy bimanual examination under anaesthesia is done with an empty bladder. The right index finger should be in the rectum in the male patient or in the vagina in the female patient and the four fingers of the left hand push down the anterior abdominal wall in the suprapubic region. The bladder is thus bimanually palpated. This should always be done before and after endoscopic resection or fulguration of these tumours. One should note the presence or absence of infiltration, thickening or masses. If a mass is palpable its size and mobility should be noted. Fixity to the prostate, uterus, pelvic wall or involvement of the rectum and vagina is noted.

The ability to perform bimanual examination comes with experience; and even in best hands the accuracy of clinical staging is not more than 80%.[10] It is nevertheless a very important examination which guides the plan of management.

CLINICAL STAGING

This is a local tumour staging and relies on the cystoscopic and bimanual examination, but histological report aids the clinical staging in distinguishing between flat carcinoma in situ and atypia or chronic

inflammation. Histology also determines the depth of muscle infiltration. The most commonly used staging system is the Tumour Nodes and Metastasis (TNM) system; according to the International Union against cancer (UICC).

Tumour Status (CT)

Bimanual Palpation	Microscopy	UICC T status
Normal bladder	Carcinoma in Situ	T1S
A freely mobile mass may be felt. Should not persist after resection	Tumour limited to mucosa	T1
Indurated bladder wall mass, mobile. No residual induration after resection	Superficial destussor involvement	T2
Nodular mobile mass Persists after resection of exophytic portion of the lesion	Full thickness muscle involvement.	T3a
	Paravesical tissue involvement	T3b
Tumour fixed to prostate vagina, uterus	Prostate, vagina uterus or rectal involvement	T4a
Tumour fixed to pelvic wall or abdominal wall	Bony pelvis involved and beyond	T4b

Nodal status (i.e. Regional Lymph Nodes)

No = No evidence of regional lymph node involvement

N1 = Involvement of single homolateral regional lymph node.

N2 = Involvement of contralateral, bilateral or multiple regional lymph nodes.

N3 = Fixed mass on the pelvic wall with a free space between this and the bladder tumour.

N4 = Involvement of juxtaregional lymph nodes.

M status Distant metastases

M0 = No evidence of distant metastasis

M1 = Distant metastasis present

M1a = Occult metastasis detected by biochemical or other tests.

M1b = Single metastasis in a single organ site

M1c = Multiple metastases in a single organ site.

M1d = Metastases in multiple organ sites.

Nodal status is usually not easy to assess unless lymphangiography or computed tomography scanning is done. Even when lymphangiography is done there is a tendency to underestimate the incidence of nodes which are involved.[14] The primary lymph nodes draining the urinary bladder are the perivesical, internal iliac and obturator nodes.[16] The external and common iliac nodes are regarded as a second line of defense. Normally the internal iliac and obturator nodes are not visualized by pedal lymphangiography. This is the main short coming of pedal lymphangiography in that it does not enable one to visualize the primary lymph nodes draining the urinary bladder.[16] Its value is in determining involvement of nodes above the brim of the pelvis.[16] Computed tomography can also be used to assess nodal involvement.[14]

Another classification which is sometimes used especially in the United States is the Jewett and Marshall classification.[11]

This classification was initially proposed by Jewett and Strong in 1946 and later modified by Marshall. It is based on depth of tumour penetration, local extension and presence of metastasis. This system has stages O, A, B, C and D.

Stage O = Tumour limited to the mucosa. (It includes T1S and Ta in the TNM system)

Stage A = Tumour invades the lamina propria (= T1)

Stage B = Tumour confined to the muscularis

B1 = Less than half of muscularies is involved (T2)

B2 = More than halfway through muscularis is involved (=T3a)

Stage C = Tumour involves the perivesical fat (T3b)

Stage D = Tumour beyond the perivesical fat

D1 = Tumour has metastasis to pelvic lymph nodes below level of sacral promontory (T3bN2)

D2 = Tumour with lymphatic involvement above the aortic bifurcation (T3bN4)

This system has almost been replaced by the TNM system, but it is still commonly used by American Urologists.[11]

MANAGEMENT

It is important to treat the whole patient with carcinoma of the bladder rather than treating the bladder condition, and it is useful to divide the management of patients into those with superficial non-invasive and those with invasive tumour.[10]

The choice of treatment will ultimately be determined by the stage, grade and cell-type of the tumour, the number of lesions, the age and the general condition of the patient.[1]

B. Management of Carcinoma in Situ

The management of carcinoma in situ is difficult because it has a high recurrence rate after local treatment since most tumours assessed as in situ cancer are actually invasive in as high as 70 - 80% of cases.[15] In one series where carcinoma in situ was treated by electrofulguration 82% of the patients had local recurrences and 37 out of 62 patients developed invasive carcinoma.[15] For these reasons some authors have gone ahead and recommended radical treatment such as cystectomy which is claimed to offer the best opportunity for survival in patients with the very early stage of this disease.[1,7,15] Other arguments put forwards for this radical approach include the following:

(i) In some cases carcinoma in situ is extensive involving more than 60% of bladder mucosa.[15]

(ii) Some tumours may involve the distal ureters or the bladder neck and the urethra.[15]

(iii) The natural history of carcinoma in situ is unpredictable and some have a high propensity for developing invasive disease.[15]

Intravesical BCG has also been used to treat carcinoma in situ. Cunning et al [17] reported that the risk of progression of carcinoma in situ has been reduced from 70 -80% to 7% with the use of BCG.

In our locality patients usually come late and it is rare to pick up patients with carcinoma in situ. In this study we had no patients in this stage.

B. **SUPERFICIAL BLADDER TUMOURS**

These are tumours which have not infiltrated beyond the submucosa and are staged as T_1 tumours. There have been various modalities of treatment for such tumours, some of which have been abandoned with advance in technology and endoscopic electrosurgery. Some of these modalities include:

1. Mucosal stripping
2. Local hyperthermia
3. Helmstein Baloon therapy
4. Intracavitary radiotherapy with gold chains or tantalum wire
5. External radiotherapy

Most authors now are in agreement that T_1 tumours can be adequately managed by local electrofulguration or resection[1,10,18-19] Riddle[10] argues that transurethral resection or electrofulguration can control up to 80% of cases. Resection should be done to the level of

bladder muscle to assess microscopic presence or absence of infiltration.

After transurethral resection or fulguration local or topical chemotherapy may be given as an adjunct treatment to prevent recurrences.[1,19] However, Riddle[10] advocates the use of intracavitary chemotherapy only if recurrences occur and are too numerous for electrocautery. The agents used are the same as for carcinoma in situ, and include Thiotepa, Epodyl, Bleomycin and mitomycin C as single agents.

Immunotherapy with intravesical BCG has also been shown to be effective in these tumours.[18] These patients need close follow up by cystoscopy and urine cytology.

A suggested follow-up plan is a three monthly cystoscopy for the first year, then 6 monthly for two years and then annually. Intravenous urograms should also be done 3 yearly due to the panurothelial nature of the disease.

In our series we had one patient who had T_1G_1 transitional cell carcinoma. He had transurethral resection and fulguration of the tumour. His first follow-up cystoscopy done 4 months after transurethral resection showed no recurrence. He is due for another repeat cystoscopy in 3 months.

T2 BLADDER CARCINOMA

This is carcinoma of the bladder with infiltration of the superficial bladder muscle but not through it. It

is a rare lesion, probably due to difficulties in clinical diagnosis. Most often the diagnosis is made from the pathological report after transurethral resection of a T_1 tumour which shows superficial muscle invasion.

There are various alternatives for management of this stage of tumour, advocated by different authors. Riddle[10] advocates three alternatives:

The first alternative is to leave the patient alone (that is after transurethral resection), and arrange for an early follow-up cystoscopy. If at cystoscopy the bladder is found to be normal, then continue with cystoscopic follow-up. If a lesion is found at the resection site then subject the patient to radiotherapy. The second alternative is implantation of radioactive gold at the tumour site and the third alternative is radical external radiotherapy.

Badoe[1] however advocates 3 alternatives which differ with Riddle's approach.

(a) Partial or Segmental cystectomy

This should be limited to tumuors which are not situated in or do not involve the base of the bladder. The resection should include a good margin of not less than two centimetres of normal tissue, and should leave a functional bladder capacity. This might be possible in about 20% of bladder tumours. The disadvantages of this

approach are the possibility of implanting tumour cells in the wound and tumour recurrence due to inadequate tumour clearance. Pre-operative irradiation has been advocated to reduce incidence of tumour implantation and tumour recurrence.

(b) Simple cystectomy could be done for G1 and G2 tumours which are located in the bladder base or are too large for partial cystectomy. Pre-operative irradiation should be given for G3 tumours.

(c) The last alternative is radical radiotherapy alone; but the results of this modality have been disappointing; also they are accompanied by marked side-effects, especially mucositis of the sigmoid colon, rectum and small bowel resulting in nausea, diarrhoea and bowel perforation.

T3 BLADDER CANCER

This is bladder carcinoma with involvement of the muscle layer and perivesical fat. There are three approaches in the management according to Riddle.[10]

(a) **Radical Cystectomy**

This involves total cystectomy with pelvic node dissection. In males this should include prostatectomy with removal of the seminal vesicles with or without urethrectomy.

In females total hysterectomy with removal of major part of the vagina is done. The 5 year

survival rates after radical cystectomy is 25%.

(b) **Radical radiotherapy**

This has been claimed to give similar survival rate as radical cystectomy, that is 25%,

(c) **Combined pre-operative radiotherapy and radical cystectomy**

This seems to be a more popular method. The mortality and morbidity rates are not worse than radiotherapy or surgery alone, but the 5 year survival rate is approximately double that of either radiotherapy or surgery alone.[10] At Muhimbili Mkony and Phillip[22] has used this method for some patients, and it seemed to give promising results.

We had 3 patients with T_3 bladder carcinoma. All were treated by radical radiotherapy. None had cystectomy.

MANAGEMENT OF T4 BLADDER CARCINOMA

This is an advanced disease and whatever treatment given should be aimed at palliating symptoms. So some assymptomatic patients deserve no treatment. Generally palliative radiotherapy is given in hope of achieving tumour regression and arrest of bleeding.[1,10] However, local radiotherapy for bladder pain and frequency often makes things worse.[10] Radiotherapy may be useful for local bone pains due to metastatic disease.[1,10,14] Sometimes intractable haematuria may be controlled by Helmsteins Baloon therapy[10] or by intravesical instillation of 40% formalin diluted in normal saline

as a 1:10 dilution. This is instilled in the urinary bladder via a urinary catheter and wait for 15 minutes then irrigate the bladder with 10% alcohol followed with normal saline.[20]

Systemic chemotherapy has been tried, but results are inferior to radiotherapy and are disappointing.[1,10] Drugs which have been used include 5 Fluorouracil, Mitomycin C, Methotrexate, Bleomycin and Hydroxourea.[21] In our series we had 5 patients with T4 tumours, and four (4) of them were subjected to palliative radiotherapy. One of the patients died before radiotherapy could be started. Another patient died after he had finished his course of radiotherapy and three patients were discharged home and did not come for follow-up.

CONCLUSION

Carcinoma of the urinary bladder is a common problem in Tanzania with many patients presenting late for treatment. Most patients are given palliative treatment in the form of radiotherapy. However doctors should have a high index of suspicion in those patients who present early with haematuria or symptoms of bladder irritation. Such patients should be investigated adequately including urine cytology and cystoscopy. On the other hand we need also to direct our efforts on control of urinary schistosomiasis which is a major contributing factor. The initial good results of preoperative radiotherapy and cystectomy reported at the centre need to be taken seriously and further work on the modality of treatment is necessary.

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ANKLE FRACTURES AND DISLOCATIONS AS SEEN AT MUHIMBILI MEDICAL CENTRE

SUMMARY

From August 1990 to January,1991 a total of 18 cases of ankle fractures and dislocations were seen and managed at Muhimbili Medical Centre, Orthopaedic and Trauma Department. There were 13 males and 5 females. Road traffic accidents were the most common cause of trauma accounting for 9 cases followed by falls from height which accounted for 4 cases. There were 11 cases of closed fractures and 7 cases of open fractures. Nine of the patients with closed fractures were initially managed by closed reduction and plaster cast application but four of these later needed open reduction and internal fixation after failure of closed reduction. Five patients (4 with open and 1 with closed fracture) were managed by calcaneal traction. One patient was treated by primary repositioning non-rigid osteosynthesis. The final outcome was good in all cases.

INTRODUCTION

The ankle joint is an important weight bearing joint in the body. Injuries of the ankle are common both in adults and children.[1] The ankle is usually injured by indirect violence, the foot being inverted, everted, externally or internally rotated on the tibia.[2] Each of these mechanisms produce a characteristic fracture

pattern, so that these fractures can be classified on the basis of the mechanism.

Since the ankle carries all the weight of the body it follows that the management of fractures of the ankle should be meticulous and prompt if one is to avoid chronic pain and disability due to malunited fractures resulting from inadequate reduction or immobilization.

At Muhimbili Medical Centre most ligamentous ankle injuries and some ankle fractures are managed on an outpatient basis. Major ankle fractures and dislocations however, are usually managed as in patients during the initial phase of management. The management of ankle fractures in patients seen between August 1990 and January 1991 is presented and the relevant literature reviewed.

CASE PRESENTATION

U.R (Reg. No. 642119) a male, aged 56 years was admitted to hospital on 15.1.91 having sustained injury to his right ankle after falling from the roof of a container while at work about 8 hours prior to admission. He had landed on his feet and after the fall developed pain and swelling of his right ankle. He did not sustain any other injuries and denied having any other medical problems. His past medical and surgical history was unremarkable.

Clinical examination revealed an old man who was in pain but was otherwise in good nutritional status. He

was not pale. His blood pressure was 130/90 mmHg and pulse was 76 beats per minute. Systemic examination was normal. Local examination revealed a swollen right ankle joint which was also deformed. It was tender on palpation and both active and passive movements of the joint were impossible due to pain.

The patient was given pethidine 100 mg intramuscularly statim, the leg was splinted and then taken for X-ray examination. The radiography revealed fracture of both the medial and lateral malleoli with lateral displacement of the talus. The injury was classified as Weber "C" ankle injury as characterized by:

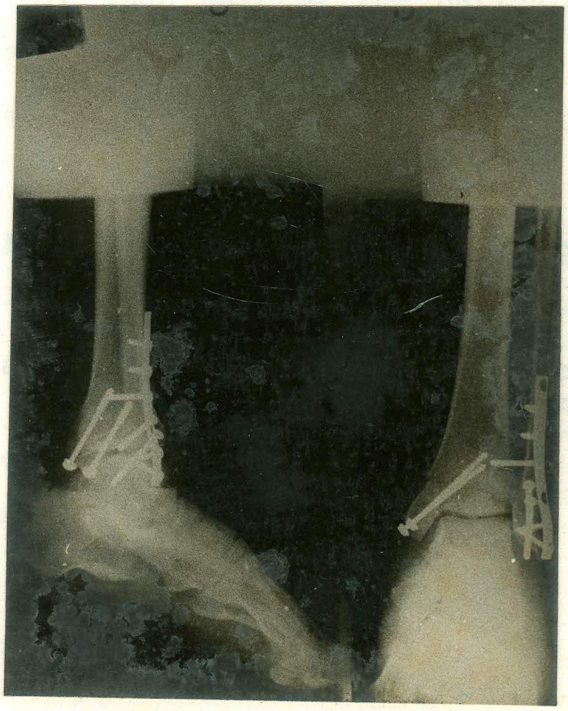
- The fibula was fractured above the level of the syndesmosis
- The medial malleolus was fractured at the level of the mortise and displaced laterally.
- The talus was completely displaced laterally.

An attempt at closed reduction under general anaesthesia was made and after manipulation with reduction a below-knee plaster cast was applied. Post-reduction X-rays however revealed inadequate reduction. He was therefore planned for open reduction and internal fixation which was done on 22.1.91 one week post-injury. The operation was done under general anaesthesia with a tourniquet in situ. A 5 cm

longitudinal incision was made over the lateral malleolus. The fracture site was exposed and cleared of old haematoma and some bone debris, and the fracture fragments were apposed together and fixed in place by two lag-screws. This fixation was reinforced by a plate which was held in place by cortical screws on the proximal fragment and cancellous screws on the distal fragment. The medial malleolus was then exposed through a separate curved incision and the fracture site was exposed and freshened. The fractured malleolus was reduced and fixed with two malleolar screws. The wounds were then irrigated with normal saline and closed. Post-operatively the leg was elevated on pillows and the patient was instructed on dorsiflexion exercised from the second post-operative day. The wounds healed well and the patient was mobilized on non-weight bearing crutches but instructed to continue with dorsiflexion and plantarflexion exercises. A week post-operatively he was discharged and instructed on his subsequent gradual return to weight bearing at the end of 6 weeks. He was seen 8 weeks later and apart from slight swelling in the right ankle he had no other complications and he was weaned from crutches and started to use a walking stick. He was seen 6 weeks later and was found to be free of pain and was able to walk without the walking stick.



(a)



(b)

Preoperative (a) and postoperative (b) photographs of the index case.

Table I Age and Sex distribution

Age Group	Males	Females	Total
10 - 20 yrs	3	0	3
21 - 30	3	1	4
31 - 40	3	2	3
41 - 50	1	2	3
51 - 60	2	0	2
Over 60	1	1	2
Total	13	5	18

Note: (a) More than half of the patients were young, below 40 years of age.

(b) The male to female ratio was 2.6:1

Table II: Aetiological Factors and Type of Fracture

Causative Factor	Open Fracture	Closed Fracture	Total
Road traffic Accident	5	4	9
Falls from height	0	4	4
Misstep	0	1	1
Sports injury	0	1	1
Assault	1	0	1
Object hitting the leg (Impact Injury)	1	1	2
Total	7	11	18

Note: (a) Closed fractures were more common than open fractures.

(b) Road traffic accident was the most common cause of ankle fractures.

Table III Individual bony injuries

Bone involved	Number of Patients
Bimalleolar fractures	7
Epiphysiolysis (Salter Harris 2)	2
Medial malleolus alone	2
Lateral malleolus alone	3
Fracture calcaneus	1
Fracture talus	1
Crush injury - multiple fractures	2
Total	18

Note: Malleolar fractures were the most common type of injuries.

Table IV Management

Modality of Treatment	Number of Patients
Closed manipulation + and plaster cast	9
Open reduction and Internal Fixation	4
Calcaneal traction	5
Primary minimal osteosynthesis	1
Surgical Toilet, Reduction and Plaster cast	3
Total	22

Note: (a) Closed manipulation and Plaster cast was the most common method of treatment.

(b) Four patients required open reduction after failure of closed manipulation.

DISCUSSION

Understanding the anatomy of the ankle joint is important if one is to understand injuries of the ankle joint and their management. The ankle joint is an articulation between the superior, medial and lateral surfaces of the talus with the tibia plafond, the medial and the lateral malleolus.[1] The joint is often called the ankle mortise.[4] The capsule of the joint fits closely around its articular surfaces. It is weak anteriorly and posteriorly but it is reinforced on the medial and lateral aspects by collateral ligaments.[5] The medial collateral ligament include the deltoid ligament which is composed of the anterior talotibial, posterior talotibial and the calcaneotibial ligaments.[6] The lateral collateral ligament includes the anterior and posterior talofibular ligaments. These form the fibular collateral ligament.[6] Laterally the peroneal tendons pass posterior to the fibular malleolus; but these have no deforming effects on ankle fractures.[6] Medially are the posterior tibial tendon and flexor hallucis tendons passing in a groove on the medial malleolus. These tendons may push the distal fracture

fragment anteriorly hence interfering with adequate reduction.

The stability of the ankle is dependent on both the bony and ligamentous structures. The ankle joint is said to be a hinge joint but this is an oversimplification.[1,4] The talus is wider anteriorly than posteriorly so that the fibula must move relative to the tibia in order to keep in contact with the talus throughout its range of movement.[1,5,6] Also the tibial plafond and the talus are saddle shaped; hence even a small loss of congruity between the two articular surfaces will result in loss of a large surface of contact. It has been shown that a lateral shift of 1 mm of the talus relative to the tibia results in a 42% loss of mutual contact.[1]

The anatomical configuration of the ankle mortise allows rotatory, rocking and tilting motion.[6] The ankle is responsible for vertical transfer of body weight over the fixed foot during walking; but it must also transmit considerable rotatory force generated at the hip and the knee. The ankle joint should always be considered together with the subtalar joint because both are responsible for converting torsion about the vertical axis of the tibia to torsion about the longitudinal horizontal axis of the fixed foot.[1]

The use of the terms flexion and extension as applied to the ankle joint varies. When the dorsum of the foot

is made to approach the tibia this is called "dorsiflexion". The opposite movement is called "plantarflexion". Pronation (eversion), supination (inversion) and pure abduction and adduction of the middle part of the foot take place at the subtalar joint.[4]

MECHANISM OF INJURY OF ANKLE FRACTURES

Rowley[1] described the following mechanisms operating in ankle fractures:

(a) Abnormally directed forces which may induce the ankle to move in a direction in which it was not intended to move.

(b) Normally directed forces but of magnitude that the ankle cannot tolerate.

Cadaver studies by Lauge-Hansen have demonstrated rotatory forces as being the most common cause of ankle fractures.[1,4]

The talus is the key to all ankle fractures. Abnormal forces on a supinated foot will usually displace the talus laterally; thus impinging on the lateral malleolus. This could cause an "eversion fracture" of the fibula without the foot itself being everted at the time of injury.[4] Likewise the stability of the ankle joint depends on the integrity of the tibial plateau and the talus. If there is incongruence in the tibial plafond and hence incongruence in talotibial articulation then the injury will lead to instability.[2]

CLASSIFICATION SYSTEMS

There have been several classification systems for fractures of the ankle. Some of them have been descriptive only; for example unimalleolar, bimalleolar or trimalleolar fractures. Most of these are only of historical interest. A perfect classification system should indicate the mechanism of injury; influence the management and if possible predict the outcome.[1] None of the classification systems meets all these criteria. The first classification system which suggests the mechanism of injury was that described by Ashurst and Bromer.[1] Under this system fractures are caused by external rotation, abduction and compression.

The Lauge-Hansen classification[1,4] is based on studies done on cadavers linked with clinical and radiological observations.

This was first proposed by Lauge-Hansen in 1942. Fractures are classified into:

(a) Supination adduction fractures (SA)

(b) Supination-eversion fractures (SE)

(c) Pronation-abduction fractures (PA)

(d) Pronation-eversion fractures (PE)

Each group has a number of subgroups called stages (I-IV) depending on additional injuries to the ankle caused by the main deforming force. The Lauge-Hansen classification is criticized for being too complex and its fine elaborations are based on assumptions that are

scarcely justified.[1] Also there are some fractures which cannot be classified according to this system.[4]

THE DANIS-WEBER CLASSIFICATION

According to this system fracture/dislocations are classified into three classes A, B and C depending on the level of the fibular fracture in relation to the syndesmosis and the talotibial joint.[4] This system is more widely used and hence will be described here in detail:

Weber Type A

1. There is a fracture of the lateral malleolus below or at the level of the ankle joint.
2. The medial malleolus is intact or has an oblique fracture.
3. The posterior edge of the tibia is usually intact.
4. The tibiofibular ligamentous complex is intact.

Mechanism of injury

Supination (inversion) violence is the main force.

Weber Type B

1. lateral malleolus: There is a fracture at the level of the syndesmosis
2. The medial malleolus may be intact or shows an avulsion fracture accompanied by rupture of the deltoid ligament.
3. Posterior edge of the tibia may be intact or there may be an avulsion fracture fragment.
4. The tibiofibular ligamentous complex is torn in 50% of cases.

Mechanism of injury: There is forced outward rotation of the talus

Weber Type C

These are fractures above the level of the syndesmosis:

1. The fibula is fractured between the syndesmosis and head of fibula.
2. The medial malleolus demonstrates an avulsion fracture through the malleolus or an injury to the deltoid ligament.
3. There may be a fracture of the posterior edge of the tibia laterally.
4. The tibiofibular, ligamentous complex is always disrupted.

Mechanism of injury: There is an outward rotatory violence with a component of impacting force.

Impact Fractures

If the foot is exposed to direct external violence for example from a fall from a height; crush injuries or compression, fractures occur with an appearance and functional pathology different from fracture dislocations.[4]

Impact fractures have been divided into three groups by Weber:

A. Crush fracture at distal end of tibia with a fibular fracture. The talar trochlea is intact.

B. Variable mortise injury with simultaneous fracture of the talar trochlea.

C. Crush fracture at distal end of tibia without fibular fracture. The talar trochlea is intact but the syndesmosis is always torn.

Post-traumatic function

(a) Widening of the ankle mortise. This implies altered loading conditions and there is a great risk of incongruence arthritis.[4] It has been demonstrated that lateral displacement of the talar trochlea by one to two millimetres leads to a reduction of the tibiotalar contact area on weight bearing of almost 40-60%.[1,4]

(b) The role of the Syndesmosis.

Injury to the syndesmotic ligaments leads to increased risk of pathological outward rotation of the talus and backward displacement of the lateral malleolus since the syndesmosis plays an important role in maintaining the position of the talus.[4] The talar displacement is worse if the deltoid ligament is also torn.

(c) Shortening of the fibula

When the fibula is shortened it will not reach the bottom of the fibular notch on the tibia. This will result in lateral displacement of the lateral malleolus and widening of the ankle mortise.[4] The axis of motion of the ankle joint is also changed. The result is incongruence osteoarthritis.

Fracture through the posterior margin of the tibia

This usually means that there is a rupture of the posterior tibiofibular ligament which is the strongest ligament of the syndesmotic system.[4] This implies rupture of the syndesmosis and a high risk of widening of the ankle mortise. Posterior tibial margin fracture also means that there is a significant damage to the articular cartilage with a high risk of post-traumatic arthritis.[4]

Diagnosis

The history in ankle injuries can be misleading as quite minor falls may lead to severe injuries especially in the elderly.[1] Physical examination should include palpation of all the ligaments and bony prominences. The whole fibula and the proximal tibiofibular articulation should be examined. Localized tenderness over th anterior tibiofibular ligament often implies a fracture in addition to ligamentous injury.[1] Fractures of the lateral malleous accompanied by tenderness along the medial malleolus should make one suspect a tear of the deltoid ligament and indicate more severe injury than indicated on radiographs.[3]

Radiography

Radiographs should not replace careful clinical examination otherwise many fractures will be missed such as the Maisonneuve's fracture for which

radiographs of the whole fibula are required for diagnosis.[1] Stress radiographs are not routinely necessary for acute injury. Even in chronic injuries they are of questionable value.[1,7] Stress radiographs are difficult to perform without anaesthesia and may be misleading without comparative views of the normal ankle. According to Rowley[1] at least 20% of talar tilt must be demonstrated on stress radiographs before diagnosis of ankle instability is made. If on X-ray the medial joint space between the medial malleolus and the talus is increased by 2 mm or more this would imply rupture of the deltoid ligament.[7]

MANAGEMENT

The management of fractures of the ankle can be considered under two headings:

(a) Management of closed fractures
- . Closed manipulation and plaster cast application
- . Open reduction and internal fixation

(b) Management of open fractures:
- . Surgical toilet, manipulation and plaster cast application.
- . Surgical toilet and minimal osteosynthesis or repositioning non-rigid osteosynthesis

MANAGEMENT OF CLOSED FRACTURES

(a) **Closed Reduction**

All ankle fractures deserve an attempt at closed reduction. But it is important to remember that

only accurate reduction is compatible with good ankle function, and therefore it is essential that the ankle mortise is aligned and the superior surface of the talus must fit into the tibial plafond and the malleoli.[6] Any slight variation will not allow the talus to ride smoothly within the mortise and will lead to pain.

The best time to reduce any fracture is immediately after it occurs. No ankle fracture is too swollen to be reduced.[6] Persistent swelling may jeopardize the skin over the malleoli and fracture blisters may develop particularly in delayed reduction.[6] But swelling usually subsides after adequate reduction. If there is extensive swelling a compression dressing or an Esmarchs bandage can be applied with the patient under general anaesthesia for 10 -15 minutes. Manipulation and reduction are then done and a plaster cast is moulded. During application of plaster of Paris the foot should be held in an angle of function and the plaster application should conform to the shape of the foot and ankle and should be smoothly moulded around the Archille's tendon, the heel and the malleoli.[6]

The leg should be immobilized in plaster for 6-12 weeks to allow both soft tissues and bones to heal. Weight bearing may be gradually permitted if the ankle is in neutral position.

At Muhimbili Medical Centre closed reduction and plaster cast immobilization was attempted in all cases of closed ankle injuries except in one case where the patient had an almost complete crush injury and the surgeon felt that the fracture fragments could not be held in place by Plaster of paris cast. This patient had calcaneal traction. In four cases there was failure of closed reduction and open reduction had to be done.

OPEN REDUCTION AND INTERNAL FIXATION

The most certain way of restoring normal function to a fractured/dislocated weight bearing joint is to obtain a perfect reduction of the joint surfaces.[8] With meticulous care this can be achieved by closed manipulation and plaster of paris application. In some cases, however, maintenance of reduction is difficult because of the changing dimensions of the ankle due to a decrease in oedema. In such cases open reduction may be needed. Operative treatment leads to a higher proportion of anatomically reduced fractures of the ankle as compared to closed methods. This leads to lower risk of developing osteoarthritis, pseudoarthrosis and disabling pain.[9] With advances in internal fixation techniques it is becoming increasingly common to operate on fracture/dislocations of the ankle.[1] The indications of open reduction according to Rowley[1] and Cave[6] include:

1. Failure of closed reduction
2. When the posterior tibia is fractured and the fragment is greater than one third of the tibial plateau.
3. Widening of the mortise in bimalleolar fractures.
4. Shortening of the fibula in simple fibula fractures
5. Presence of an intra-articular fragment or ligaments that prevent the talus from assuming a good weight bearing position under the tibia.

In open reduction previously more importance was given to correct reduction and internal fixation of the medial malleolus. But after studies done by Yablon et al[10] it has now been demonstrated that the lateral malleolus is the key to anatomical reduction of displaced malleolar fractures and establishment of stability of the joint. This has also been supported by Ward et al[11] who describe the importance of late lengthening of the fibula for malaligned ankle fractures.

Yablon and colleagues[10] found through cadaver experiments and clinical studies that in fractures of the lateral malleolus or in bimalleolar fractures the talus tended to follow the lateral malleolus if this was displaced. These findings suggest that in bimalleolar fractures or in fractures where the deltoid

ligament is torn the talus remains attached to the lateral malleolus.[3,10] So if the posterior tibia is intact the lateral malleolus should be fixed first if possible by interfragmentary compression screws supplemented by a laterally placed buttress plate.[1] The medial malleolus can then be fixed by a malleolar screw or screws.

Post-operatively the ankle is elevated preferably with a leg suspension sling to above the level of the heart if it is to be effective.[3]

After 48 hours the patient should start dorsiflexion exercises and then he can be ambulated on crutches. A below knee POP cast or brace can be applied. Anti-inflammatory medication and elastic bandaging are provided.[3]

THE MANAGEMENT OF OPEN FRACTURES

The management of open fractures at the ankle region is complicated by absence of adequate loose skin around the ankle and yet it is important to cover the wound without tension.[6] Ngcelwane[12] poses two more problems in management of open fractures of the ankle:

(a) In some open fractures dirt is often found in the depth of the joint in the region of the syndesmosis. This dirt is lodged in this area by suction effect during he mechanism of dislocation.

(b) In severely displaced fractures there is often extensively soft tissue injuries especially the deltoid ligament.

While reduction may be easy maintenance of reduction is usually difficult. So in the management of such fractures all the three problems mentioned above must be tackled. After adequate surgical toilet and reduction of the fracture fragment attempts should be made to close the skin without tension. This may necessitate making release incisions, sometimes at the expense of inadequate reduction which can be done later when wounds are healed.[6]

For the severely contaminated fracture dislocations in order to adequately deal with the dirt which has been sucked deep in the syndesmotic area Ngcelwane[12] advocates making a counter-incision opposite the main wound and cross-irrigation with large amounts of irrigation fluid. Thereafter metallic fixation can be done, which provides stable fixation and allows regular dressing of the wound if it had been left open.[12]

There is a controversy on the necessity to repair the deltoid ligament in such injuries. Zeegers and VandeWerken[13] argue that if the ankle mortice is adequately reduced anatomically there is no need to repair the deltoid ligament because even when it is not repaired it does not lead to medial instability.

Johnson and Hill[7] on the other hand argue that reduction and internal fixation without repair of the deltoid ligament results in severe disability and instability in about 45% of patients. They therefore

advocate repair of the deltoid ligament at the time of internal fixation of the associated fractures.

In our environment the management of open fractures is complicated by various problems:

(a) patients often come late from the time of injury and by this time there is extensive swelling of the soft tissues which make primary wound closure futile due to the time factor and soft tissue swelling.

(b) Most of the open wounds are heavily contaminated hence the method advocated by Ngcelwane[12], that is of making a counter incision for wound irrigation would be useful in such cases.

As seen in this study some cases of open fractures were managed by calcaneal traction in an attempt to put the fracture fragments in alignment. Probably such case could have benefited from primary repositional non-rigid osteosynthesis. Of the 7 cases of open ankle fractures 3 were treated by surgical toilet and closed manipulation with plaster cast application, 3 cases were treated by calcaneal traction after surgical toilet as it was found that to maintain reduction with a plaster cast would be impossible. One patient was treated by surgical toilet and repositional non-rigid osteosynthesis with Kirschner wires for both medial and lateral malleoli.

INDIVIDUAL FRACTURES

1. **Isolated fractures of the lateral malleolus.**

These are said to be the commonest injuries seen in the emergency room according to Yablon and Segal.[3] Usually they are caused by supination adduction force. If the ligaments are intact such fractures are stable. But they will be unstable if associated with ligamentous injuries. One has to look for tenderness over the medial malleolus; since tenderness in this area may imply tear of the deltoid ligament. If it is torn it will lead to instability and will cause a talar tilt.[3] Treatment includes closed reduction and a below-knee POP cast for 6-8 weeks. We had 3 such cases and all were treated by Plaster of Paris cast.'

2. **Isolated fractures of the medial malleolus**

These fractures occur in road traffic accidents and sports injuries; being caused by pronation-eversion and pronation-abduction injuries. Usually there is no displacement. Treatment consists of a short leg-walking POP cast.[3] We had two such cases all being treated by a POP cast.

3. **Fractures of the Talus**

These fractures are important for two reasons:

(a) The talus is wholly intra-articular and three-fifths of its surface is covered by articular cartilage, articulating with the

os calcis, navicula, tibia and fibula. Since any fracture of the talus will involve a joint the risk of traumatic arthritis is high.[6]

Most of the nutrition of the talus is received through the neck of the talus from a branch of the dorsalis pedis artery. so fractures through the neck of the talus are likely to lead to avascular necrosis. In the management of talar fractures impacted fractures can be treated conservatively. But fractures with displacement that do not respond to closed manipulation should be reduced surgically. Then POP is applied for 8 weeks after which a walking boot is applied for one month.[6] In our study we had one patient with fracture of the talus which was managed conservatively.

4. **Fracture of the calcaneum**

Os calcis fractures are of two types: those that do not enter the joint such as avulsion fractures and "beak" fractures, and those that involve the subtalar joint such as crush or comminuted fractures. Most fractures are due to falls from height and landing on heels. The majority can be managed conservatively by "moulding" the os calcis and a boot POP. But some may need open reduction;

and if this is impossible the possibility of subtalar arthrodesis should be considered.[6] We had one patient who had a beak fracture which was managed conservatively by POP.

Fractures of the Tibial plafond

These fractures are also called "pilon" fractures; and they are fractures which involve the articular surface. In the management of such fractures the goals should be to restore anatomic reduction of the joint surface, to achieve rigid fixation of the fracture fragments and to have early mobilization of the joint.[3] It is not always possible to achieve all goals; but one should aim at achieving as close to perfection as possible. Open reduction is the treatment of choice. One should try to restore the length of the tibia by pulling down the tibial fragments, then fix or reconstruct the tibial articular fragments and then fill any gaps with bone grafts. The distal bone fragments may be held with Kirschner wires, but finally a butress plate on the medial side and interfragmentary compression screws will be necessary.[3]

In our case we had one patient with a pilon fracture who was treated by calcaneal traction. Though brought in alignment the congruence of the articular surface was not perfect; so he may need arthrodesis later on.

Epiphyseal injuries

In children bimalleolar fractures do not usually occur. Epiphyseal injuries usually occur at the distal epiphyseal plate. Any type among the Salter Harris groups may occur from Salter Harris (SH) I to V.[3] In such cases adequate reduction should be done in order to avoid growth deformities. Kirschner wire fixation may be used. In our study we had two cases which were classified as Salter Harris II epiphyseal injury. Both were treated by closed reduction and plaster of Paris cast, but one patient developed fracture blisters. The cast had to be removed and a back slab was applied to allow management of blisters which had progressed to ulceration. He finally healed well: but he is due for long term follow-up in view of detecting any long-term complications.

CONCLUDING REMARKS

Ankle fractures and fracture-dislocations are a common cause of orthopaedic admission at Muhimbili Medical Centre; and their management is challenging. It is important that patients get the correct management right from the beginning. Thorough surgical toilet should be done for the open fractures, and while some of these cases were managed by calcaneal traction there is probably a place for minimal repositioning non-rigid osteosynthesis. This would minimise the length of hospital stay and would enhance early return to productive life.

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FRACTURES AND FRACTURE DISLOCATIONS OF THE SPINE AS SEEN AT MUHIMBILI MEDICAL CENTRE

SUMMARY

Between August, 1990 and January, 1991 a total of 21 cases of fractures or fracture-dislocations of the spine were managed by the Orthopaedic and Trauma department of Muhimbili Medical Centre. All were males with age ranging from 15 years to 80 years with 12 patients being in the 20-40 year age group. Falls from height, mainly from coconut trees and road traffic accidents were the most common causes. The thoracolumbar spine and the cervical spine were the most commonly affected regions having 9 and 6 cases respectively. Nine patients had no neurological deficits while 12 patients had some degree of neurological deficit on admission.

All patients were managed conservatively and all improved with the exception of 4 patients who remained with residual complete paraplegia.

INTRODUCTION

Fractures of the spine are a common cause of hospital admission due to trauma. The great majority of fractures of the spine involve the cervical, lower thoracic and upper lumbar vertebrae.[1] In civilian practice dorsolumbar fractures are usually flexion injuries either from falls from height, forward flexion

in decellerating car crash or a heavy object falling onto the shoulders.[1] Open fractures of the spine are unusual except in times of war when they are due to gun-shot injuries.[1] The cervical spine is usually injured in flexion such as falls upon the head in diving or acute flexion in decellerating injury but hyperextension injuries are also common.[2]

Documented literature on the incidence of spinal injuries in Tanzania is scanty, but Kivambe[3] found that fractures of the spine accounted for 1.6% of all fracture cases admitted at Bumbuli Hospital in Lushoto. The incidence is likely to be higher in larger cities and even in other towns especially those along highways which will be receiving road traffic accident victims. Between August, 1990 and January, 1991, 21 cases of fractures of the spine were admitted at Muhimbili Medical Centre, Orthopaedics and Trauma Department. A case history is presented in detail and a brief review of literature on cervical and thoracolumbar fractures being the most commonly observed is done.

CASE PRESENTATION

S.T. (Reg.No. 624412) a 30 year old man was admitted on 2.9.90 with post-traumatic weakness and numbness in both upper and lower extremities 6 hours post-injury. He was a clerical assistant in a customs ware house and on that particular day he was at his work-table when a crane which was lifting fertilizer bags gave way and

bags of fertilizer fell onto his right shoulder. He fell down onto his abdomen as more bags fell onto his back. Since then he was unable to walk and and was numb in all extremities. He had not lost consciousness. He had not passed urine since the time of injury and did not feel the desire to do so. He denied having taken alcohol on the day of injury and was not on any medications for any chronic illness. His past medical history was unremarkable. He was single and non-smoker.

Clinical examination revealed a young man who was fully conscious, well oriented in time, place and person. His pulse was 80 beats per minute and the blood pressure was 80/40 mmHg. He was not pale. The head was normal. There was no obvious tenderness over the neck. The spinal column was non tender. Respiratory system examination revealed a predominantly abdominal type of breathing with bilateral limitation of chest expansion. The trachea was centrally situated. Air entry was normal and there was no evidence of haemo- or pneumothorax. Neurological examination revealed loss of pain sensation up to the nipple line bilaterally with flaccid paralysis of both upper and lower limbs. Abdominal examination was normal apart from a distended urinary bladder. Genital examination revealed a turgid penis.

A clinical diagnosis of fracture of the cervical spine was made X-rays of the cervical spine revealed a compression fracture of the sixth cervical vertebra with a tear-drop fragment.

The patient was put on a neck collar and a regime of paraplegic nursing was instituted. This included:

. Two hourly turning on a fracture bed with a firm surface

. Skin care to prevent pressure sores

. intermittent 6 hourly urinary cathetererization.

The patient was re-evaluated 24 hours later and was found to have a return of sensation. He subsequently started to regain muscle power slowly but steadily. By the third week he had a muscle power grade 4 in most muscle groups in both upper and lower limbs. Muscle strengthening physiotherapy was started and at 8 weeks he was able to walk with crutches. He was then discharged. He was seen 4 weeks later, that is 12 weeks post-injury when he was found to be able to walk unsupported. He was weaned off the collar over a period of 6 weeks and he was last seen 6 months post injury and was found to have fully recovered apart from a residual weakness of the right arm.

Table I **Age distribution of patients**

Age Group	Number
11 - 20	3
21 - 30	9
31 - 40	3
41 - 50	4
More than 50	2
Total	21

Note: - All patients were males

- Children below 10 years were not treated in the orthopaedic trauma department
- More than half (15 patients) were young, below 40 years of age.

Table II **Causes of Spinal Injuries**

Cause of Injury	Number
Falls from height	12
Road traffic accidents	6
Hit by falling heavy objects	3
Total	21

Note: Falls from height and road traffic accidents were the most common causes.

Table III **Level of injury**

Level of fracture	Number
Cervical spine	6
Thoracic spine (T1 - T10)	2
Thoracolumbar (T11 - L2)	9
Lower Lumber (L3 - L5)	4
Total	21

Note: Cervical and thoracolumber fractures were the most common.

Table IV: **Neurological status at admission**

Neurological Status	Number
No neurological deficit	9
Quadriplegia	1
Paraplegia or paraparesis	9
Monoplegia or monoparesis	2
Total	21

Note: - More than half (12) of the patients had some degree of neurological deficit.

- Three patients with paraplegia were referred from regional hospitals already paraplegic.

DISCUSSION

Aetiology

Fractures of the spine commonly occur in road-traffic accidents following forced flexion or extension with or without rotatory strain. This is particularly common in the cervical and thoracolumbar region.[4] Other causes include falls from heights either on the head, buttocks or feet.[4]

Heavy falling objects could also cause forced flexion and/or rotation of the spine leading to fractures.[4] While generally road traffic accidents are said to cause most of the fractures of the spine, Trafton and Boyd[5] analysing thoracolumbar fractures found that falls from height caused 3/4 of all the injuries. Falls onto the head in diving is a common cause of fractures of the cervical spine.[1] In this series of case collections falls from height was the most common cause of fractures of the spine accounting for 12 of the 21 patients. It is also interesting to note that 8 of the 11 patients fell from coconut trees either while harvesting coconuts or during tapping alcohol(tembo). Only two of the patients agreed to have taken alcohol. Two patient fell from mango trees and one patient fell from the roof of a container.

Ambeva[6] found that fractures of the spine accounted for 11% of all injuries sustained by coconut climbers in Kenya. He analysed the possible causes of falls from

coconut trees to include:

- Fatigue after climbing many trees
- Drunkenness on climbers who tape alcohol
- The figure of 8 rope they use for the feet may snap
- Strong winds may cause loss of balance at the top of a swaying coconut tree
- The supporting branch may break
- Climbers may meet tree snakes at the top and fall from fright.

As an aid to prevention of such injuries Ambeva suggested the use of safety belts as those used by electricians working on electrical poles.[5]

Mechanism of Injury

According to Holdsworth[7] forces causing fractures of the spine may be grouped into five types: pure flexion, flexion and rotation, extension vertical compression, or direct shearing force. Sometimes there may be a combination of the pure mechanics. Pure flexion violence leads to direct longitudinal pull on the posterior ligamentous complex. The body of the vertebra is crushed anteriorly resulting in a wedge fracture which usually remains stable.

In flexion-rotation violence the posterior ligamentous complex ruptures and the upper (proximal) vertebra swings over the inferior vertebra. In the cervical region this usually results in an unstable pure

dislocation since the articular processes easily slide off each other. Sometimes locked facets may lead to a stable injury. In the lumbar region pure dislocation is rare due to the large vertical articular processes, but a slice fracture occurs more commonly and this is an unstable fracture.[7]

Extension injuries mostly occur in the neck and may lead to rupture of the disc and tear of the anterior longitudinal ligament resulting in a dislocation. The posterior ligaments are unaffected. This type of dislocation is stable in flexion.[7]

Vertical compression usually occurs in the cervical and lumbar spines. In such areas vertical compression leads to a "burst" fracture which is caused by the nucleus pulposus of the disc being forced into the vertebral body. The ligaments are not damaged, hence the fracture is stable. However, it is very painful due to multiple fracture fragments which are impacted.[7]

Shearing violence occurs when a powerful force is applied directly to a part of the back, mainly in the thoracic region. The proximal part is sheared directly over the inferior vertebra with fracture of the articular processes and rupture of all ligaments.[7] These fractures are made stable by the rib cage. Holdsworth[7] also introduced the concept of stable and unstable fractures.

Dennis[8] however, in an attempt to precisely determine stability of fractures of the vertebrae described the three column concept of the spine:

(a) The posterior column as described by Dennis is the posterior ligamentous complex described by Holdsworth. It is formed by the posterior bony complex (posterior arch), the supraspinous ligament interspinous ligament and the ligamentum flavum[8].

(b) The middle column is formed by the posterior longitudinal ligament, posterior part of the annulus fibrosus and the posterior wall of the vertebral body.

(c) The anterior column is formed by the anterior longitudinal ligament, anterior part of the annulus fibrosus and the anterior half of the vertebral body.

Basing on the three column concept one can more precisely determine the stability of a fracture depending on which column or columns are involved in the fracture (vide infra).

Dennis[8] also classifies significant fractures of the spine into 4 groups and describes them according to the three column concept.

(a) **Compression fractures**

These are described as wedge fractures by Holdsworth.[7] There is compression of the anterior column but the middle column is intact and this prevents subluxation of the spine, compression of the neural elements or retropulsion of the fragments of the posterior wall

into the neural canal. Clinically there is no palpable gap. Radiologically there is compression of the anterior part of the vertebra with intact end plates and no significant separation of the spinous processes.[7,8]

In this series there were seven cases of compression fractures, six of which were due to falls from coconut trees.

(b) **Burst fractures**

In this type of fracture there is failure under axial load of both anterior and middle column originating at the level of one or both end plates of the same vertebra.[8] The vertebra is shattered and there is outward, displacement of the fracture fragments. Radiologically the spinous processes are not separated. There is loss of height of both the anterior and posterior edge of the vertebral body on the lateral view. The anteroposterior view shows an increase in interpedicular distance. There may be retropulsion of fracture fragments into the spinal canal.[8] According to Holdsworth[7] these fractures are essential stable; however Dennis[8] classifies them into five subgroups depending on whether there is superior, inferior or both end plates involvement and whether there is a rotational or flexion component.[8] Hence there is a variable degree of stability.

(c) **Seat-belt injuries**

There is failure of both the posterior and middle columns caused by forces in flexion with the anterior column acting as a hinge.[8] Sometimes even the anterior column may fail. This type of fracture was called shear fracture by Holdsworth.[7] Radiologically there is a horizontal split of the transverse processes as well as the pedicles.[8] If there is no displacement these fractures are stable. The "chance" fracture and the Hangman's fracture are in this group.[7,8] There was no patient with this type of fracture in this series.

(d) **Fracture/Dislocations**

In this type of fracture there is failure of all the three columns under compression, tension, rotation or shear.[8] This is the most unstable of all injuries.[7,8] Radiologically there is subluxation or dislocation as seen in the lateral or anteroposterior views.

The effect of flexion and rotation varies with the level of the injury. In cervical spine where the articular facets are almost horizontal the injury may entirely be ligamentous resulting in a pure unstable dislocation. In the lumbar region where the articular facets are vertical and interdigitate more effectively these forces fracture the articular processes causing fracture dislocation.[9]

In this study there were 6 cases of fracture and/or dislocations. In the cervical spine there were 3 cases

of pure dislocations while the rest were in the thoracolumbar region; and these were fracture dislocations.

Definition of Instability

An unstable spinal injury is that which may lead to increased deformity with potential possibility of causing an increased neurological deficit.[8] Dennis[8] using the three column concept classified instability into 3 degrees.

1st degree instability

This is mechanical instability with risk of chronic kyphosis. This applies to severe compression fractures with posterior column disruption.

2nd degree instability

This is neurological instability. It includes the so-called stable burst fractures where there may be retropulsion of bone into the neural canal.

3rd degree instability

This is both mechanical and neurological instability. This includes fracture-dislocations and unstable burst fractures with or without co existing neurological deficits.

However, assessment of instability must be based on sound clinical judgement and not by some arbitrary rules or unproven radiological parameters.[10]

DIAGNOSIS

Apart from the history which may suggest the mechanism of injury the diagnosis of spinal injuries rests on a thorough, meticulous and systematic examination of the patient.[1,7]

During inspection the attitude of the extremities and the position of the neck may give a clue to cervical spine fractures.[11] Occasionally rotation of the head to one side may indicate unilateral facet joint dislocation on that side.[11] Note presence of abrasions lacerations or gibbus on the back. Note also the type of respiration. A predominantly abdominal respiration suggests paralysis of intercostal muscles and hence a possible cervicodorsal spine injury.[12] Note presence of a distended urinary bladder and priapism all of which suggest injury to the spinal cord.[12]

Palpation of the back is very important. A palpable gap between affected spinus processes is looked for and when present it almost always means an unstable spinal injury even when roentgenograms are equivocal.[7]

Muscle power, reflexes and sensation are tested and recorded accurately and the neurological level of injury is therefore determined. It is important to note, however, that in the first 24 hours findings of paraplegia may be due to spinal shock and therefore

such an evaluation should be repeated after 24 hours, and also after 72 hours; because it has been found that patients with features of motor and sensory sparing at that time can be given a good optimistic prognosis.[13] But the final functional outcome depends on motivation and training and the amount of spasticity present.[13]

After clinical examination X-rays of the spine are done, and two views are generally required (an anteroposterior and a lateral) although for the cervical spine a good quality lateral radiograph may be sufficient to reveal cervical fractures between C1 and C7.[11] However, to obtain such a radiograph the surgeon should accompany the patient to the X-ray department and position him. It may be necessary to pull the shoulders caudally to get a good exposure. When the shoulders cannot be pulled down a swimmer's view can be taken. X-rays with an open mouth may be necessary for demonstrations of fractures of the atlas and odontoid peg.[11]

On the frontal view X-rays one should look for and note:-

- The centrality, displacement or absence of the spinous processes.
- The presence and symmetry or asymmetry of the articular facets.
- Any lateral displacement of the vertebral bodies.

On the lateral view one should note:

- Any separation of the spinus processes
- The height of the posterior and anterior margins of the vertebral bodies
- The superior and inferior end plates
- Any anteroposterior subluxation or dislocation.

Currently computerized tomography (CT) where available has got a place in the evaluation of injuries of the spine.[3,5] According to Straughton[5] and McAffee et al[14] the value of CT scan include the following:

- It allows the assessment of the degree of retropulsion of the vertebral body fragments or annulus fibrosus into the spinal cord.
- It allows identification of unstable burst fractures by illustrating facet joint subluxation or disruption of the neural arch.
- It enables objective planning of any surgical management.

By CT scan evaluation spinal canal narrowing correlates well with neurological deficit, and when there is a 50% decrease in mid-saggital diameter of the spinal canal there is a risk of neurological compression.[5]

Indications for CT scan include:

1. Spine injuries with neurological deficit
2. Spine injuries with possible or definite disruption of all the three columns as assessed by plain radiographs

3. Severe deformities especially with fractures at multiple levels.

Where CAT scans are not available myelography should be performed.

MANAGEMENT OF FRACTURES OF THE SPINE

(a) **Cervical Spine fractures**

Management depends on stability of the fracture. Stable fractures require a surgical neck collar or skull traction to control pain.[11] Such stable injuries include fractures of the atlas and simple extension injuries with "tear-drop" fractures of the inferior edge of the vertebral body.[11]

Unstable injuries require reduction of any displacement and immobilization which should continue until healing of fracture and torn ligaments has taken place. This usually requires at least 6 weeks.[9,10] Such unstable fractures include burst fractures, hangman's fractures, odontoid fractures and fracture-dislocations.

Reduction may be achieved by simple traction through skull-clippers which may also be used for immobilization if it is maintained until bone union has taken place.[11] If it is desirable and possible for the patient to be ambulant then immobilization can be continued in a "halo" vest.[11]

Reduction of complete dislocations can be achieved by heavy and increasing traction through skull callipers,

or more rapidly by manipulation under general anaesthesia.[9,11] Once reduction is achieved traction is maintained for 6 weeks when spontaneous stabilization can be anticipated. If at that time there is no evidence of stabilization on X-rays in flexion and extension then surgery is indicated such as bone grafting.[9,11]

In this study there were 9 patients with cervical injuries, three had dislocation of cervical vertebrae. All were managed conservatively with cervical collars. One patient with a dislocation of the fifth over sixth cervical vertebra had an initial period of traction, with Glissons sling for three weeks followed by a cervical collar. The patients stayed with their neck collars for a minimum of 12 weeks.

(b) **Thoracic spine injuries**

Between T_1 and T_{10} the thoracic rib cage provides adequate stability to the spinal column. Fracture-dislocations in this region are rare, and if they occur they are usually from very severe shearing forces at right angles to the spine. Paraplegia invariably results and treatment is aimed at managing the paraplegia since reduction is usually impossible.[9] In this study there was no patient with fractures in the thoracic spine.

(c) **Thoracolumbar and lumbar fractures**

Management of fractures in this region depends on their degree of stability.

(i) Stable Fractures

These should be treated by bed-rest for three to six weeks, then the patient is encouraged to sit up with back-muscle strengthening exercises and he is finally mobilized. This includes simple anterior wedge fractures, or fractures of the transverse or spinous processes only.[7, 9, 15]

(ii) Unstable fractures or fracture dislocations

Patient should be recumbent in bed for 6 to 12 weeks. They may be treated in a plaster of Paris bed for 6 weeks and then in a plaster jacket until bony fusion is evident.[7,9] If reduction is not achieved then open reduction and plating is probably appropriate. However, most authors now prefer open reduction and instrumentation for unstable thoracolumbar fractures.[7,9,10,15,16]

Indications for surgical intervention include:

1. Complete dislocation
2. Development of kyphosis move than 45°
3. Unstable fractures in a patient with multisystem trauma where stabilization facilitates total patient care.
4. If CT scan shows a mid-saggital diameter reduction of more than 50%.

5. a wedge compression fracture of more than 40% as it may compress further.

Currently most authors advocate posterior, stabilization of the spine by Harrington's rods.[4,5,10,14,15] It is also generally agreed that laminectomy does not have a place in the management of fractures of the spine. In fact it is said that laminectomy may further destabilize the spine.[5,7,15,16] In this series we had 9 patients with thoracolumbar fractures, 4 of whom had stable wedge compressions fractures which were treated by bed rest for 6-8 weeks, after which they were mobilized and discharged on crutches. Three patients with fracture dislocations were discharged with corsets after an initial period of bed rest on fracture boards. Two patients were completely paraplegic, these were managed conservatively and finally discharged home for care by relatives.

(d) **Thoracolumbar fractures with neurological deficits**

In unstable fractures with paraplegia the management is centred on prevention of the complications of paraplegia. Immediate complete paraplegia due to cord damage in which no recovery of motor power or sensation within 24-48 hours will not recover, as it has been found that neurological trauma to the cord seems to be determined at the moment of impact.[7,9,15,16] The management involves:

(a) Intense nursing care programme

- Skin care - needs creaseless bed sheets
- Two hourly turning of the patient day and night to prevent bed sores
- Intermittent bladder catheterization every 6 hours.

(b) Physiotherapy

- Prevention of contractures by doing full range of motion for all joints
- Chest physiotherapy to avoid hypostatic pneumonia
- Development of unparalyzed muscles
- Final mobilization on a wheel-chair, tricycle or crutches.

(c) Occupational and psychological therapy to increase the morale of the patient and to enable him to earn some bread.

In this study there were 4 patients with complete paraplegia, 3 patients having been referred from nearby regional hospitals and one patient was admitted with severe fracture dislocation. These were managed conservatively and finally discharged home after discussion with and education for the patients' relatives in preparation for home-care.

CONCLUDING REMARKS

All patients in this study were managed consevatively either with neck collar or Glisson's sling traction for

cervical fractures and recumbent bed rest for thoracolumbar and lumbar fractures. None was managed surgically either because surgery was not indicated or probably due to inadequate facilities.

For fractures of the cervical spine availability of a Halo traction device may be a better alternative to a Glisson's sling since with Halo traction one can control the position of the neck and it is easier to turn the patient without disturbing the spine. The use of a Halo vest or a halopelvic frame also allows earlier mobilization of the patient.[9,17] Surgical stabilization either by posterior approach or anterior approach with bone grafts and wiring could be done for selected cases and this would have similar advantages of shortening hospital stay in addition to allowing a more precise reduction in fracture dislocations and offering sufficient stabilization.[9]

Likewise for fractures of the lumbar spine surgical stabilization allows decompression of neurological structures and early mobilization of patients regardless of neurological deficit while protecting the neurological structures from further injury.[10] Certainly open reduction should not be resorted to without adequate indications but where facilities allow and when indicated this would aid in achieving early mobilization of patients thus avoiding all the complications associated with prolonged recumbency for

months especially considering the pressure of beds existing in the hospital.

There has been a big problem in discharging paraplegic patients. since relatives are reluctant to take patients home. To overcome this problem patients' relatives must be involved in the rehabilitation programme of the patient right from the beginning. They must be educated about the complications of paraglegia and how to avoid them. They must be educated also how they can assist in rehabilitating the patient so that he can be productive (occupational therapy). They should also be educated and given time to prepare the home environment ready to receive the patient when he is discharged. If relatives are involved in planning the care of the patient they are more likely to accept him rather than when they are told to take the patient home without adequate preparation as to how they are going to take care of him.

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