TEN SURGICAL CONDITIONS

BY

DR. WALLACE NG'WINULA NGWESA, MD (DAR)

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I declare that this dissertation is my own work. It has not been submitted for any academic award in any other University.

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

DR. MR AZIZ

SUPERVISOR

DEPARTMENT OF SURGERY
MUHIMBILI MEDICAL CENTRE

DEDICATION

To my Mother, wife and children for their sincere love and patience.

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ABSTRACT

This dissertation is a discussion of ten different surgical conditions seen at Muhimbili Medical Centre. It includes a discussion on patients collected and entirely or partially managed by the author during the period of training (1991 - 1994). The text is divided into three parts consisting of five topics on general surgery, three topics on urology and two topics on orthopaedics and trauma. In each topic one case is presented in detail followed by a discussion and general analysis and review of relevant literature.

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MANAGEMENT OF PANCREATIC PSEUDOCYST

SUMMARY:

A total of six patients with pancreatic pseudocyst who presented to the three adult Surgical wards of Muhimbili Medical Centre between 1992 - 1993, were studied. Two patients were females and four of them were males. Their ages ranged between 23 and 60 years. Four patients gave a history of trauma, three weeks to four months before, one of the patients had history of heavy alcohol intake while one old woman presented with an acute abdomen, later developed an epigastric mass and diabetes mellitus. Upper abdominal mass (6) and upper abdominal pain (6) were the main presenting complaints.

The surgical procedures were; cystojejunostomy(3), cystogastrostomy (1), external drainage followed by cystojejunostomy (1), while one of the patients died a week after being diagnosed to have a pancreatic pseudocyst. No complications were encountered intra/postoperatively. One of the patients studied is presented and a review of literature on the topic follows.

INTRODUCTION:

Pancreatic pseudocysts are localized collections of pancreatic secretions, lacking an epithelial lining, which occur as a result of pancreatic inflammation and ductal

disruption.[1,2] True cysts of the pancreas, which are either congenital or neoplastic, possess an epithelium. Such true cysts constitute only 15% of all cystic lesions of the pancreas[1] and are therefore greatly outnumbered by false cysts (pseudocysts) which are inflammatory in origin. The inflammation may be due to acute or chronic pancreatitis or may follow trauma (external or operative).

D'Egidio and Schein[3] proposed a classification that differentiates between the pseudocysts of acute and chronic pancreatitis, identifying three distinct types. Type 1 or acute 'postnecrotic' pseudocysts occur after an episode of acute pancreatitis. Type II, also postnecrotic pseudocysts, occur after an episode of acute-on-chronic pancreatitis. Type III, defined as 'retention' pseudocysts, occur with chronic pancreatitis. The distinction between acute-on-chronic (type II) and chronic (type III) cysts can be subtle. It often requires operation to show the presence of necrotic debris, indicating a recent flare-up of acute on chronic pancreatitis that may have been missed clinically.

Currently accepted standard management of a pancreatic pseudocyst first involves verification of the suspected diagnosis by imaging studies (sonography, CT or Magnetic resonance imaging). The rest of management plan is determined by the type of pseudocyst, size of the cyst, and presence or absence of complications.[4,5,7]

The options for treating pseudocysts include; observation, percutaneous drainage, endoscopic drainage, endoscopic transpapillary approaches, external drainage and internal drainage. The choice of these options is dictated by the size and site of the pseudocyst, surgeons preference and also presence or absence of complications.[5,8,9]

CASE PRESENTATION:

A 38-year-old peasant, J.L; was admitted to Muhimbili Medical Centre. He gave a history of upper abdominal pain radiating to the back and was being relieved by sitting up. He complained of a mass in the upper part of the abdomen which was increasing in size gradually. He had anorexia and loss of weight. Three months back he was involved in a road traffic accident and became unwell for a few days. He neither smoked cigarettes nor took alcohol.

On examination he was ill looking, not anaemic and not jaundiced. The pulse rate was 80/minute, regular and of good volume and his blood pressure was 120/80 mmHg. There was no abnormality on examination of his respiratory, cardiovascular and central nervous systems. Abdominal examination revealed a mass in the right upper quadrant extending to the midline with the greatest diameter of 12cm, had smooth surface, mobile and was cystic in consistency. The liver and spleen were not palpable.

His haemoglobin was 12.8gm/dl with a white cell count of 11,200/mm.^{3} Lateral view of his barium meal showed anterior displacement of the stomach by a soft tissue shadow. Ultrasonography of the abdomen showed a cystic mass in the lesser sac close to the pancreas. The mass was suspected to be a pancreatic pseudocyst.

The patient was planned for explorative laparotomy with a possibility of internal drainage. At laparotomy, a pancreatic pseudocyst was found adherent to the posterior wall of the stomach. Transgastric cystogastrostomy was performed. The cyst wall was biopsied and sent for histopathology. The fluid was brownish in colour, contained in a unilocular cyst. The patient was maintained on an intravenous drip. He was allowed to take light meals on the fourth postoperative day as his condition was good and he felt hungry. He did well in the postoperative period, the stitches were removed on the seventh postoperative day and he was discharged on the eighth postoperative day.

The biopsy specimen was reported as fibrous tissue wall with granulation tissues and infiltration with lymphocytes, monocytes and plasma cells. There was no reagent for analysing pancreatic amylase in the serum and cystic fluid. He was seen at the clinic two months later and was in good condition.

TABULAR RESULTS

Table 1: Sex distribution.

Sex	Number
F	2
M	4
TOTAL	6

Table II: Age distribution.

Age Group (Years)	Number
Less than 20	0
21 - 30	1
31 - 40	2
41 - 50	2
51 - 60	1
TOTAL	6

Table III: Presentation.

Symptoms, signs.	Number
. Trauma	4
. Alcohol	1
. Upper abdominal mass	6
. Upper abdominal pain	6
. Weight loos	3
. Nausea and vomiting	3

Table IV: Treatment.

Treatment	Number
Cystogastrostomy	1
Cystojejunostomy	3
Eexternal drainage + Later Cystojejunostomy	1
Non operative	1
TOTAL	6

DISCUSSION

Aetiology and incidence:

There are many causes of pancreatic pseudocyst all of which involve the pancreas in one way or another.[3] Studies of Bradley[4] and Yeo[7] showed that the prevalence of pseudocyst after an attack of acute pancreatitis ranged from 16 to 50%, and that in chronic pancreatitis 20-40%. In this study only one patient had a pancreatic pseudocyst following acute pancreatitis. Pseudocysts occurring in the younger age group are secondary to trauma while those in the 4th and 5th decades are due to pancreatitis, gallstones and high alcohol intake.[10] In this study, trauma accounted for 4 out of 6 patients excluding those under 10 years who are admitted to paediatric surgical ward (Table III). And only one patient was known to be an alcoholic. Other series report as follows; O'Malley and Colleagues[12] noted in 69 pseudocysts that; 78% were related to alcohol pancreatitis, 7% to complicated gallstone pancreatitis, 6% were of an unknown causation and 3% each were due to trauma, hyperlipidaemia or a recent surgical operation.[12] A similar study of 83 patients from South Africa included 70% of cysts that followed alcohol-associated pancreatitis, 22% that were due to blunt abdominal trauma and 8% that were idiopathic.[3]

Classification:

A classification proposed by D'Egidio and Schein[3] differentiates between the pseudocysts of acute and chronic pancreatitis. Three distinct types have been identified.

Type I or acute postnecrotic pseudocysts occur after an episode of acute pancreatitis, are associated with normal duct anatomy and rarely communicate with the pancreatic duct. Type II, also post necrotic pseudocysts, occur after an episode of acute-on-chronic pancreatitis. The pancreatic duct is diseased but not strictured, and there is often a duct-pseudocyst communication. Internal drainage is usually required. Type III, defined as retention pseudocyst, occur with chronic pancreatitis and are uniformly associated with stricture and pseudocyst-duct communication. This classification has been supported by Grace and Williamson.[13] However, the distinction between acute-on-chronic (type II) and Chronic (type III) cysts can be very difficult, often requires operation to show the presence of necrotic debris, indicating a recent flare-up of acute-on-chronic pancreatitis that may have been missed clinically. Whenever possible the pancreatic duct should be delineated in patients with chronic pancreatic pseudocyst, since an independent surgical approach may be required to deal with underlying duct pathology.

In this study such a classification was not followed as it has been introduced recently (1992).[3] However, history alone can enable one to predict the type of pancreatic

pseudocyst although differentiation between types II and III might require an operation.[3,13]

Clinical features:

A patient with pancreatic pseudocyst may either be asymptomatic or present with symptoms such as upper abdominal pain and mass, early satiety, nausea and vomiting secondary to gastric outlet obstruction; pruritus and jaundice secondary to obstruction of the common bile duct; alteration of bowel habits and symptoms of obstruction of the gastrointestinal tract secondary to colonic obstruction; variceal bleeding secondary to obstruction of either the splenic or portal vein; arterial haemorrhage secondary to formation of pseudoaneurysm in adjacent visceral vessels, and signs of sepsis secondary to infection of the fluid within the pseudocyst.[7]

In the patients studied, upper abdominal mass and pain were the leading presenting features occurring in all patients. Three patients presented with nausea, vomiting and weight loss (Table III). One patient admitted due to acute pancreatitis, developed an abdominal mass and diabetes mellitus while in the ward and had fever as well.

Investigations:

A pancreatic pseudocyst is suspected from the history and clinical examination.[3,7,12,13] History of abdominal trauma, heavy alcohol intake, and severe upper abdominal pain radiating to the back followed by appearance of an upper abdominal mass and pain, nausea, anorexia, vomiting and fever

suggest a pancreatic pseudocyst.[3,7,13]

Introduction of ultrasound, CT scan, magnetic resonance imaging (MRI) and endoscopic retrograde cholangiopancreatography (ERCP) has improved visualization of pancreatic lesions.[7,13] At our Centre, only ultrasound is done and a barium meal may complement this.

Ultrasound, CT scan and MRI can differentiate solids from cysts which are greater than 2cm in size. Ultrasound can also visualize the biliary tract for presence of gall stones which may be the cause of pancreatitis.[12] All patients in this study had ultrasound done and showed the cyst.

The advent of CT scanning for patients suspected of having pancreatic pseudocyst has allowed precise documentation of the size, location, number and wall thickness of pseudocyst.[7] Computerized CT scan is superior to ultrasonography as is not affected by gas in the bowel but is more expensive and not easily available. Both CT scan and ultrasound have been used to study the natural history of pancreatic pseudocyst.[7,13]

ERCP procedure can display the entire pancreatic duct system. It can show obstruction of the main pancreatic duct or extravasation of the contrast into the cyst.[13] Nealon and colleagues[14] recognized that 9 out of 41 patients thought to have acute pancreatitis with pseudocyst actually had chronic pancreatitis on the basis of ERCP findings. Differentiation of the two types of pseudocyst is important as they are

managed differently.[13] There are no facilities for this investigation at our centre hence no patient in this study was subjected to it.

Serum amylase is elevated in acute pancreatitis and may persist with the pseudocyst. The presence of urinary amylase in a patient with history of pancreatitis suggests the development of a pseudocyst because most of the amylase absorbed into circulation eventually is excreted by the kidneys. In this study there was no reagent to check both serum and urinary amylase.[13] Warshaw and Rattner[8] suggested that the presence of a serum isoamylase, called old amylase, in a patient with a pseudocyst may be a reliable indicator for successful internal drainage.

Cyst wall biopsy confirms the diagnosis, as the pseudocyst will have only fibrous granulation tissue and inflammatory cell infiltrates rather than an epithelial lining.[1] In this study, a cyst wall biopsy was taken at operation and not preoperatively.

Treatment:

Timing of intervention:

The timing of intervention is a critical matter that requires considerable judgement.[13] The pseudocyst of chronic pancreatitis can be drained surgically as soon as it is diagnosed, in the confident expectation of finding a mature cyst wall capable of holding sutures.[7,8] In patients with complications such as infection of the pseudocyst,

haemorrhage, pressure symptoms, or rupture of the cyst into the peritoneal cavity require immediate external drainage by the simplest means available.[8] The optimal timing of intervention in acute or traumatic pseudocyst is more difficult.[13] If the operation is delayed unduly, the incidence of complications such as haemorrhage, obstruction and rupture of the pseudocyst will rise as suggested by some workers.[4,7] However, the consensus is that a minimum of 4-6 weeks from the time of diagnosis of an acute pseudocyst is required for the wall to become sufficiently mature for safe internal drainage.[6,13]

In this study it was difficult to determine the duration of the pancreatic pseudocyst as the majority of patients never reported to hospital immediately after trauma (4), one patient was an alcoholic and was not known as to when he developed the pseudocyst. Only one patient was admitted for acute pancreatitis and later developed a pancreatic pseudocyst. Lack of CT scan to measure the thickness of the wall, made decision making of the drainage procedure be determined intraoperatively.

Options of treatment:

The options of treating pancreatic pseudocyst include external and internal surgical procedures, and non-operative options including observation.[13]

Surgical options:

The operative management of pancreatic pseudocyst aims at

evacuating the cyst contents and preventing the serious complications of cyst rupture, haemorrhage, infection and intestinal obstruction.[5] If the cyst cavity is decompressed, it is likely to become obliterated with time.[7] In pseudocysts following acute pancreatitis there is a choice between external and internal drainage, but in chronic pancreatitis there is also the possibility of cyst resection.[13] The cyst is only one feature of the underlying disease, and the decision between resection and drainage is influenced by features such as the extent of surrounding inflammation, vascular involvement and ductal ectasia.[13]

External drainage is the simplest method of draining a pseudocyst, but carries a mortality rate of 6% and a recurrence rate of 22%.[1] In this study external drainage was the treatment of choice for one patient who at laparotomy was found to have a pancreatic pseudocyst with a friable cyst wall. External drainage is the operation of choice for grossly infected cysts, those associated with haemorrhage or free rupture necessitating emergency laparotomy, and immature cysts with a soft wall that will not hold sutures.[13] These indications are thought to be the cause of high mortality figures, reflecting the severity of pancreatitis in the patients chosen for external drainage rather than the specific shortcomings of the technique.[13] A persistent pancreatic fistula occurs in 10% of patients following external drainage.[15] When fistulae fail to close, usually because of

proximal pancreatic duct obstruction, either distal pancreatectomy or drainage of the fistula by means of a Roux loop of the jejunum may be necessary.[6]

Internal drainage establishes a controlled fistula into the gastrointestinal tract and is the method of choice for all mature uncomplicated pseudocysts.[7,13] Cystogastrostomy and cystojejunostomy are alternative procedures for accomplishing internal decompression.[13]. Internal drainage is associated with a mortality rate of 2% and a recurrence rate of 5%.[1] In this study 4 of the 6 patients underwent cystojejunostomy while cystogastrostomy was the treatment of choice in only one patient. No intra/post operative complication occurred.

Controversy surrounds the question of which viscus should be used for pseudocyst drainage: stomach, duodenum or a Roux loop of jejunum.[13] Anatomical considerations sometimes dictate the choice; for example cystogastrostomy is inappropriate unless the stomach is closely applied to the front of the cyst. A preoperative ERCP has been used by some authors[13,14] to refine operative strategy as this can show pancreatic duct obstruction or pseudocyst communication.[14]

Cystogastrostomy:

Transgastric cystogastrostomy is a relatively simple approach that exploits the fact that many mature cysts are densely adherent to the posterior wall of the stomach. The cyst is approached through the lumen of the stomach, the margins of the posterior gastrostomy being oversewn in the

manner of an anastomosis. Objections to cystogastrostomy are that the cyst is not always contained within the lesser sac nor always adherent to the stomach. The anastomosis may not produce dependent drainage if the cyst is large and extends below the greater curve.[13] Johnson and colleagues[9] noted that the complication rate following cystogastrostomy was directly proportional to the size of the pseudocyst. Three of the four patients with giant pseudocyst (more than 15cm) had life-threatening complications as a result of incomplete emptying of the cyst, and 2 of them died. Moreover, entry of gastric contents into the cyst cavity may occasionally lead to further complications including haemorrhage, pancreatic abscess, gastric abscess, gastric fistula and cyst recurrence.[13]

Cystojejunostomy Roux-en-Y:

Cystojejunostomy with a Roux-en-Y anastomosis is now performed for internal drainage of pseudocyst more frequently than cystogastrostomy.[13,16] The Roux loop can be anastomosed to the lower part of the cyst, using an approach through the transverse mesocolon for a collection in the lesser sac.[13] It is likely that dehiscence of a cystojejunostomy has less serious consequences than that of cystogastrostomy, provided that a Roux loop is used.[16] Newell and colleagues[16] compared the results of the two procedures; cystogastrostomy and cystojejunostomy. There was no significant difference in mortality between the two groups 5% for cystogastrostomy versus 3% for cystojejunostomy. Cystogastrostomy was

associated with a shorter operative time, reduced peroperative blood loss and lower fluid requirements. The hospital stay was also shorter after cystogastrostomy; 11 versus 19 days. By contrast, complications were slightly more frequent; the incidence of cyst recurrence was 10% after cystogastrostomy and 8% after cystojejunostomy, while the incidences of postoperative gastrointestinal haemorrhage were 7 and 2% respectively. In this study cystojejunostomy was done in four of the six patients and no complications were encountered intra or postoperatively. However, the data is too small for relevant comparison purposes.

Cystoduodenostomy:

This procedure is rarely indicated, and is reserved for pseudocysts in the head of the pancreas that impinge on to the duodenal wall.[5] This procedure is likely to be appropriate only in relatively small cysts that complicate chronic pancreatitis. Care has to be taken not to injure the intrapancreatic common bile duct or the gastroduodenal artery. In one study cystoduodenostomy carried a 5% mortality rate and a 5% recurrence rate.[5] In this study no patient underwent cystoduodenostomy.

Pseudocyst resection is an alternative to internal drainage for chronic pseudocyst of moderate proportions, especially those that have largely replaced a portion of the pancreas.[5]

Nonoperative options:

Pancreatic pseudocysts present for less than 6 weeks have a 40% spontaneous resolution rate and a 20% complication rate.[4] Yeo and colleagues[7] noted in the group managed nonoperatively, with a mean follow-up period of one year, that 60% had complete resolution of the pseudocyst and 40% had pseudocyst that remained stable or decreased in size. No patient in this study was managed conservatively as five of the patients presented to hospital more than 6 weeks after noting upper abdominal mass while one patients who developed a pseudocyst following acute pancreatitis died before decision on treatment was made.

Other centres can perform percutaneous drainage under ultrasonographic or CT guidance, endoscopic cystogastrostomy or cystoduodenostomy, and endoscopic transpapillary stenting.[7,13] The above procedures are newly introduced and none of them is performed at our hospital.

CONCLUSION AND RECOMMENDATIONS:

Pancreatic pseudocysts are rare while trauma, pancreatitis and alcoholism have been reported as common causes. Such patients should be followed up for sometime to rule out formation of pancreatic pseudocyst. History and physical examination are important tools for raising suspicion of a pancreatic pseudocyst as roentgenographic studies are not always readily available in Tanzania.

Internal drainage by cystojejunostomy is to be encouraged compared to cystogastrostomy according to reported findings in different series. However, new methods such as non operative drainage options can be encouraged in order to widen choice of procedures in the management of pancreatic pseudocysts.

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TRAUMATIC RUPTURE OF THE SPLEEN

SUMMARY:

A total of ten patients with rupture of the spleen due to trauma were operated in the three adult surgical firms at Muhimbili Medical Centre between January and December, 1993. The age group mostly affected was 31 - 40 years. Those below the age of 20 and above 40 years were infrequently involved.

This study excludes patients who are aged 10 years or less because such patients are admitted to the children's ward. Injuries were sustained from falling from a height (3), road accidents (4) and direct physical abdominal blows (3). Patients presented with symptoms suggestive of intra-abdominal injury with haemorrhage. Five patients had in addition other associated injuries. Splenic rupture was suspected clinically in all patients. At laparotomy, rupture of the spleen was confirmed in all patients. Two patients had laceration of the liver as well and one had an accompanying laceration of the mesentery.

Splenectomy was the treatment of choice in all patients. Splenic preservation procedures such as nonoperative management and splenorrhaphy were not attempted. Major postoperative complications were few and there were no deaths. One of the patients is presented in detail followed by a detailed discussion on the subject.

INTRODUCTION:

The spleen is reported to be most frequently injured organ following abdominal trauma. Thal and associates (1984)[1] reported in a review of several series in the United States that the spleen was injured in more than 26% of patients with blunt abdominal trauma and Nzarubara (1984)[2] in Kampala, Uganda found that the spleen was injured in 32% of patients with abdominal injuries. Mutabihirwa (1984)[3] reported that splenic rupture was the commonest indication for splenectomy at Bugando hospital in Mwanza, Tanzania between the years 1978 - 1982. Road traffic accidents, fall from a height, assaults and sport injuries are the common causes of splenic rupture. Young adults are ones at greater risk due to more vigorous daily activities and travel which expose them to accidents.[4,5]

The modern era for surgery of the spleen following injury began in 1892 when Riegner reported a splenectomy in a 14-year-old construction worker who fell from a height and presented with abdominal pain, distension, tachycardia, and oliguria. This report set the stage for routine splenectomy, which was performed for all splenic injury in the next two generations.[6] Two major milestones were established by this procedure. The first one was definitive laparotomy rather than abdominal compression for refractory intra-abdominal bleeding while the second one established the role of splenectomy as a safe and effective procedure. This success also helped lay to rest the 19th century concept that the

spleen was absolutely necessary for life.[6] Despite early reports by Pearce and by Morris and Bullock,[7] that splenectomy in animals caused impaired defences against infection, little challenge to routine splenectomy was made until King and Schumacker in 1952 reported a syndrome of "Overwhelming postsplenectomy infection (OPSI)".[8] Many studies have since demonstrated the importance of the spleen in preventing infections, particularly from the encapsulated organisms.[9,10,11] The syndrome of OPSI has been recognized as a clear hazard, occurring in 20-50% of splenectomy cases in the first year of life.[8,12] Initial reports estimated the rate of OPSI in adults between 1-2%[13,14] but critical analysis of the data suggest the rate to be 0.5% at most.[12,15]

This has led to introduction of splenic salvage as a popular method of management and can be achieved safely in most patients.[6,16-20] Although several techniques have been found to be effective and safe,[20] they are not commonly practiced at Muhimbili Medical Centre. The aim of this study is to look into the age group at risk of splenic injuries, features, diagnostic procedures and treatment options as seen at Muhimbili compared with studies elsewhere.

CASE REPORT:

JM Reg No. 743231 a 20-year-old girl was involved in a road traffic accident an hour before she was admitted to hospital. On arrival she was restless complaining of upper abdominal pain, left thigh pain and thirst.

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She was examined and found to be pale with a pulse rate of 120/minute, regular but feeble and a blood pressure of 70/40 mmHg. Her respiratory system was normal. An intravenous cannula was introduced and blood for grouping and cross matching taken. Two litres of normal saline were infused rapidly raising the blood pressure to 100/60 mmHg. Further examination showed bruises and marked tenderness over both left and right upper abdominal quadrants. There was shifting dullness in both flanks. Bowel sounds were distantly heard. The middle part of the left thigh was swollen and tender but she could move her toes on the affected side. The left popliteal, posterior tibial and dorsalis pedis pulses were palpable. A clinical diagnosis of traumatic rupture of the spleen, liver and closed fracture of the left femur was made.

The left lower limb was splinted. A chest- x-ray did not show fractured ribs. A plain supine X-ray of the abdomen showed obliteration of the splenic outline and slight displacement of the gastric bubble to the right. It was not possible to take a chest x-ray in the erect position. X-ray of the left femur showed a transverse fracture of the mid one third of the shaft with displacement and an overlap of about 2cm but the alignment was good. The hip and knee joints on the left were normal.

The patient was prepared for an emergency exploratory laparotomy. An indwelling urethral catheter was inserted and clear urine was drained. A naso-gastric tube was introduced and a nonblood stained aspirate was obtained. Prophylactic Ampicillin was given intravenously.

Under general anaesthesia, the operation site was cleaned with iodine and spirit and draping was done as usual. The abdomen was opened through an upper midline incision. There was about $1^{1}/_{2}$ litres of fresh blood in the peritoneal cavity and some of it was collected for auto-transfusion. The diagnosis of ruptured spleen was confirmed. There were two deep linear parenchymal lacerations of about 6cm in length on the diaphragmatic surface of the spleen but not extending to involve the hilum. By then there was no active bleeding from the lacerations. Apart from the splenic injury, there was a laceration on the visceral surface of the liver extending from the hilum to about 8cm on the left liver lobe. There was an active bleeder near the hilum and there was minimal bleeding from the rest of the laceration. The active bleeder was picked and tied with catgut and the laceration was repaired by interrupted mattress sutures of catgut 2/0 over haemostatic surgical pack. The spleen was mobilized by excising the gastrosplenic ligament between clamps and lienorenal and lienocolic ligaments by blunt dissection. Hilar vessels were excised between clamps and the spleen was freed and removed. The splenic artery and vein and the short gastric vessels were

ligated separately and in isolation from other tissues using silk no 2/0 suture. The abdomen was once again inspected for injuries to the tail of the pancreas, the stomach, splenic flexure of the colon, small bowels and kidneys. No injury was found to involve the inspected viscera. Peritoneal lavage with warm normal saline was done. After achieving haemostasis, the abdomen was closed in two layers; a single layer of prolene no.2/0 to the peritoneum and rectus sheath and silk no. 3/0 to the skin. No drain was left in situ. The wound was dressed with gauze and then strapped with an elastoplast.

The fractured femur was managed by skeletal traction whereby under the same anaesthesia, a Steinmanns pin was inserted about 2cm posterior to the left tibia tubercle. Intraoperatively the patient received two units of blood. Postoperatively, the blood pressure, pulse rate and urine output were closely monitored. Two litres of 5% dextrose and one litre of normal saline were given for the first 24 hours. That was enough to maintain a stable pulse rate, stable blood pressure and an adequate urine output. Her body temperature remained normal. She was given Pethidine for pain intramascularly only when required for the first 24 hours postoperatively. Ampicillin was continued for the next five days. When the patient recovered from general anaesthesia she was put on skeletal traction of 12 lb which was 10% of her body weight. On the second and third postoperative days the

general condition of the patient remained stable. She had no cough or chest pain, was not pale and had no fever. She had started passing flatus and her abdomen was not distended. Bowel sounds were present and normal. The nasogastrict tube and urethral catheter were removed. The patient was allowed to take sips of water and six hours later the intravenous line was discontinued and the patient could take plain tea. The patient was advised to do isometric exercises.

On the fourth day postoperatively she developed fever, the systemic examination remained normal. A blood slide for malaria parasites was taken and found to be positive. A full course of Chloroquine was prescribed. On the same day she started taking light meals. At the end of one week the abdominal wound was found to be clean and sutures were therefore removed.

A control x-ray of the left femur was taken and overlap of the fragments was still present. A further control x-ray taken four weeks later showed minimal callus formation with the same overlap of fracture fragments. At that juncture it was decided that the patient should be scheduled for open manipulation and application of external fixators. She stayed with external fixators for three months before they were removed. On discharge she was advised to report in case of any febrile illness and to continue with physiotherapy for her left knee joint.

TABULAR SUMMARY:

Ten patients with traumatic rupture of the spleen.

Table I: Age distribution.

Age group (years)	No
11 - 20	1
21 - 30	2
31 - 40	6
More than 40	1
Total	10

Table II: Clinical feature.

Clinical features	No.
History of trauma	10
Abdominal pain	10
Tachycardia	10
Abdominal tenderness and rigidity	10
Pallor	8
Dyspnoea	2
Hypotension	4

Table III: Associated injuries.

Injury	No
Fracture of ribs	1
Fracture of femur	2
Cerebral concussion	1
Laceration of the liver	2
Mesentery tear	1

DISCUSSION;

Clinical features and diagnosis:

Traumatic rupture of the spleen often occurs in young adults, a group that is more exposed to injuries. A history of trauma is often available (Table II) although, the trauma may seem to be trivial.[1] Most patients present with general symptoms and signs of acute haemorrhage and local features of intra-abdominal injury. The clinical features are largely determined by rapidity and severity of bleeding, the interval between injury and examination and the presence or otherwise of associated injuries.[20] Two types of clinical presentation are described:

a) **Acute rupture** is clinically reported in more than 90% of patients[20] and is associated with immediate and heavy intraperitoneal haemorrhage, so that, the patient develops sudden and progressive shock which may become rapidly fatal, particularly when the rupture involves splenic fragmentation or avulsion of the vascular pedicle.[5]

Initial shock is followed by brief period of recovery and clinical features of peritoneal irritation are evident. Progressive pallor, rapid and feeble pulse, tachycardia, sighing respiration and restlessness indicate significant blood loss. Blood pressure may only be slightly reduced but when blood loss is more than 30-40% of total blood volume, sustained marked hypotension is produced.[1] Local bruising and tenderness on the left upper quadrant of the abdomen is highly

suggestive of splenic injury.[5] Abdominal tenderness and rigidity are accurate indications of peritoneal irritation and exploratory laparotomy is indicated without further diagnostic procedure.[4] Abdominal distension due to peritonism and ileus may occur three hours after the injury and Kehr's sign characterized by pain or hyperaesthesia referred to the left shoulder can be demonstrated 15 minutes after elevation of the foot of the bed.[21] Significant intraperitoneal haemorrhage will produce shifting dullness in the flanks but in 25% of patients the dullness may be localized on the left side only because of coagulated blood in the neighborhood of the lacerated spleen.[1,5] In case the patients condition is stable and a clinical suspicion of isolated splenic injury is made, immediate liver-spleen scintillation studies or CT scanning is advised.[22,23]

(b) Delayed rupture of the spleen is reported in less than 1%[6] and 11-15%[20] of patients. Patients develop initial shock followed by recovery and minimal or no serious signs of intra - abdominal injury for up to fifteen days or more. Schweizer and colleagues (1992)[17] reported delayed rupture occurring after 7,10 and 13 days in four patients who were put on nonoperative blunt splenic trauma. Further bleeding is prevented by formation of subcapsular haematoma or blood clot sealing the laceration. When the haematoma bursts or blood clot is eroded, fresh intraperitoneal bleeding occurs and the patient's condition deteriorates suddenly. Every patient with

suspected splenic injury who shows minimal physical signs should therefore be closely monitored with repeated physical examinations at frequent intervals.

Ultrasound examination is useful in doubtful cases whereby an enlarging subcapsular haematoma can be identified by serial ultrasound examinations.[5,22] Plain x-ray examination of the chest and upper abdomen may show fracture of one or more of lower ribs on the left side, and this is reported in 27% of patients with traumatic rupture of the spleen.[5] Other radiological features include elevation of the left side of the diaphragm, obliteration of the splenic outline and indentation or shift of the gastric bubble to the right.[5,20] If the diagnosis of rupture of the spleen is still doubtful exploratory laparotomy is better than continued observation.[24] Abdominal paracentesis with a needle has been found to be a useful diagnostic procedure for patients with suspected intra - abdominal injuries. If blood is aspirated a diagnostic accuracy of 95% is expected but the results become less reliable if non clotting blood is not aspirated, in which case other diagnostic procedures such as peritoneal lavage should be done.[25] In some centres peritoneal lavage is carried out routinely in evaluating patients with suspected intra-abdominal injury.[6,17,18,25] This procedure has a diagnostic accuracy of 98.5% and has been found to be more accurate than clinical examination supplemented by abdominal paracentesis.[25]

More recent methods of investigations for splenic injuries include scintillation studies and CT scanning.[22,23] CT Scanning is performed in search of a splenic or liver injury.[6] It has an added advantage of assessing the degree of splenic injury, and hence enables a surgeon to predict blood loss and the mode of management.[6,17,18,23]

In this study diagnostic paracentesis (DPC) was done in six patients and in all of them a positive tap was obtained. In the remaining patients, diagnosis was basically clinical.

Treatment.

Splenectomy: This is a reliable treatment for a ruptured spleen as a life saving procedure. It is carried out in unstable multiple injured patients who may not be able to withstand the relatively long procedure of splenic preservation. Extensive hilar injuries particularly in grossly enlarged spleen, parenchymal fragmentation and failure to achieve haemostasis by conservative techniques are other indications for splenectomy. It is not advisable to preserve the spleen when the rupture is accompanied by significant peritoneal contamination from concomitant bowel injuries.[1,26,27,28].

Postoperative complications:

Acute haemorrhage may occur if a ligature slips off the splenic artery. This calls for a double ligation of the splenic artery. Rapidly developing hypotension and a rising pulse rate in the postoperative patient is a sign of continued

internal haemorrhage requiring re-exploration.[5] Acute gastric dilatation may occur due to gastric stasis following partial mobilization of the stomach when ligating the short gastric vessels. It can also be associated with inadequately treated ileus. Acute gastric dilation can be prevented and treated by maintaining intravenous fluids and naso-gastric tube drainage for at least 24 hours postoperatively.[5] Injuries to the tail of the pancreas can occur during mobilization of the splenic pedicle and this may produce a localized abscess or a pancreatic fistula if drains were placed.[22]

Several studies show that splenectomy reduces intravascular clearance of particulate matter, including antigens and both humoral and cell- mediated immunity.[22] The spleen is the site of production of opsonin, tuftsin, and properdin, which enhance phagocytosis, and is also the site of many fixed macrophages that remove particulate antigens from the bloodstream.[22] In addition, B lymphocyte maturation and the production of both 1gM and 1gG occur in the spleen. It is a site for maturation of T-helper, T-cytotoxic/suppressor cells, and natural killer cells. The asplenic state leads to a decrease in tuftsin and immunoglobulins so that their defense activities disappear by 8 weeks. The greatest reduction in bacterial clearance is seen with the encapsulated gram-positive bacteria particularly the Pneumococcus although Meningococcus, H.influenza, E.coli, Staphylococcus and Streptococcus have also been implicated.[6,29,30] The impaired

immune response after splenectomy is not limited to intravascular phenomena. Since pneumococcus is an air-borne bacterium, the respiratory route of entry also has been studied. The lungs are probably the source of OPSI in some patients.[6] This impaired immunologic response contributes to the development of OPSI. This syndrome is now well established in children, with an incidence of 20-50% in the first year of life. The clinical features are dramatic and suggest a bacteremia shower[11] which produces constitutional symptoms that are devastating. The gastrointestinal symptoms include nausea, vomiting, and diarrhoea. The general cardiovascular symptoms include overwhelming weakness, progressing to a prostrate state. Worsening headache is associated with increasing fever leading to coma. This overwhelming syndrome may progress from onset to death within hours. Late cardiovascular collapse is associated with a rising pulse and hypotension. Varied electrolyte abnormalities may be encountered and these include hypoglycemia, despite glucose administration and hyponatremia mimicking adrenal insufficiency.

Antibiotics, even when administered early may fail to avert the progressive nature of this syndrome, which has a mortality rate of more than 50%. In fear of OPSI syndrome it is recommended that patients who have had splenectomy or splenic autotransplantation should receive Pneumococcal (Pneumovax), Meningococcal and H.influenza vaccines when

available. The role of prophylactic antibiotics cover at least until 18 years has also been recommended by some authors[20] but due to difficulty in compliance and long term effect of antibiotic use, other authors suggest advice to patients to seek urgent attention from physician at the first sign of febrile illness.[1] In patients who live in malaria endemic areas, prophylaxis against malaria is recommended.[28]

In the cases studied none of the patients got OPSI during a followup period of two months. Only two patients had wound sepsis which healed on removing the skin sutures, dressings and relevant antibiotics.

Splenic salvage: This can either be operative or nonoperative.

Operative:

The importance of splenic preservation has gradually evolved over the last three decades. Since adult patients undergoing splenectomy for trauma are thought to be at risk for developing OPSI,[13] splenorrhaphy is deemed to be the ideal treatment for splenic injuries.[6] Once splenorrhaphy is to be planned, mobilization of the spleen into the midline wound is necessary in almost all patients. Exceptions would be patients with small non-bleeding lacerations or capsular avulsion and little or no blood in the perisplenic area. If careful palpation of the convex lateral surface of the spleen reveals no further injuries, intraoperative packing of the spleen may be justified.[26] Mobilization of the injured spleen in all other patients thought to be candidates for

splenorrhaphy (or splenectomy) is performed by having the surgeon cover the injured spleen with a moist laparotomy pad and apply medial traction with the left hand.[16] Sharp division of the lienorenal and phrenicolienal ligaments with concomitant finger dissection posterior to the attached pancreas will allow partial mobilization of the injured spleen and tail of the pancreas into the midline wound. A complete view of the injured spleen is best obtained by dividing the most superior short gastric vessels that are attached to the fundus of the stomach, as well as the lienocolic ligament inferiorly.

Options for splenorrhaphy continue to evolve but can be divided into; application of topical agents, electrocautery, suture splenorrhaphy, application of meshes and partial resection.[16] All these options are based on the fact that healing of the repaired spleen is relatively rapid and complete[6]. All options outlined have been used with success, however, suture splenorrhapy is the most commonly used type of repair so far. It is easily accomplished with 0 or 00 chromic suture as with hepatic or renal repairs.[16] A continuous suture technique is useful to reapproximate parenchyma and capsule when superficial or moderate-depth (1 to 3cm) lacerations are present. With deeper lacerations, selective ligation of parenchymal bleeders, followed by the insertion of horizontal mattress sutures, will usually be adequate to obtain haemostasis. Should the capsule of the spleen tear as these

large sutures are tied down, the placement of a viable omental pedicle into the laceration or placing the sutures through Teflon mesh may be a valuable adjunct.[26]

Partial or hemisplenectomy is necessary when injuries to hilar vessels or the superior or inferior poles of the spleen are present and is the least commonly used technique of splenorrhaphy in the present series.[26] The short gastric vessels are ligated and divided to allow complete visualization of the hilum. The splenic artery in the hilum most commonly (70%) divides in the shape of the letter Y, so that there are two major branches approaching the edges of the spleen.[26] Each of these branches subsequently divides into smaller segmental arteries at the edge of the spleen itself. A less common configuration (20%) is in the shape of the letter T in which the splenic artery divides into superior and inferior branches at the edge of the spleen. No matter which pattern is present, partial splenectomy is initiated by ligating the segmental branch to the injured, avulsed, or partially devascularized segment.[26] The spleen will clearly demarcate into viable and nonviable fragments, and the latter is removed with the knife or cautery as the surgeon or assistant compresses the edge of the viable fragment. Selective ligation of bleeding points in the raw edge of the viable fragment is performed using either silk or chronic sutures.

One concern with partial or hemisplenectomy is that splenic immunity is maintained only when a critical mass of splenic tissue is left in situ attached to the splenic artery. Available laboratory data suggests that a 33% or larger remnant attached to the splenic artery will protect against an intravenous challenge with type II Streptococcus pneumoniae.[31]

Function of the splenic remnant in situ may be assessed by microscopic observation of the peripheral blood smear (absence of Howell-Jolly bodies and target cells; normal platelet count),[32] and measuring of IgM levels, all of which have been used in the evaluation of autotransplanted splenic remnants after clinical or laboratory splenectomy.[32] Patients with more severe splenic injury requiring a partial splenectomy have a 10% incidence of significant rebleeding requiring reoperation.[26]

Patients with portal hypertension and increased bleeding from all splenic attachments uniformly require splenectomy. Patients with associated splenic disease, such as splenitis, infectious mononucleosis, malaria, lymphoma, or other intrinsic abnormalities seldom have successful splenic salvage.[6]

Splenic autotransplantation:

Autotransplantation of the spleen is done to preserve splenic function after splenectomy.[33] This is accomplished by cutting the excised spleen into small fragments measuring no more than 2mm in largest dimension. These multiple, small

fragments representing at least 50% of spleen mass are placed within omental pockets as the omentum is folded on itself and held in position with through-and-through absorbable sutures.[33]

The natural history of the splenic replanted tissue includes early extensive coagulative necrosis followed by later regeneration with the ultimate return of splenic function being delayed for 5 weeks to 6 months.[34] Rhodes and Coworkers[34] suggested that autotransplantation of the spleen should be avoided in any patient with peritoneal soilage or with the potential for subsequent sepsis from associated conditions. However, subcutaneous tissue splenic implantation has been suggested in intra-abdominal contamination due to associated injuries or pre-existing high risk factors (ascites).[18]

Nonoperative:

More than 100 years ago Billroth suggested that the injured spleen has the ability to heal itself.[11] This modality did not become popular in children until the 1960s or in adults until the latter 1980s.[11] Nonoperative therapy of selected cases of blunt splenic trauma was first advocated in 1968 by Upadhyalia and Simpson who followed 12 children with suspected blunt splenic injuries.[35]

Many authors now support nonoperative management of minor splenic injuries because of the recent advances in the use of CT scan[6,18] for diagnostic and follow up assessment. This modality is not recommended in patients with severe splenic

injury, associated abdominal injury, altered mental status and evidence of continuing intraperitoneal bleeding.[16,18,26] None of the studied patients underwent any splenic salvage technique.

CONCLUSION/RECOMMENDATIONS:

Splenectomy was performed in all ten patients presented in this study. No splenic preservation technique was attempted in any of the patients. It was however observed that while it was justifiable in three patients one of whom had extensive hilar injury and two of then had fragmentation rupture, asplenic salvage could have been done in the other seven patients. Considering that most of the patients were relatively young, attempts to preserve the spleen should have been strongly pursued. Experience elsewhere shows that splenic preservation techniques are feasible and safe bearing in mind that risks to asplenic patients have been well documented. It is the recommendation of this study that splenic preservation in ruptured spleen should be encouraged in training and practice in this country.

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CUTANEOUS MALIGNANT MELANOMA

SUMMARY:

Seven patients with primary cutaneous malignant melanoma of the lower extremities were admitted to Muhimbili Medical Centre, Surgical department between January, 1992 and December, 1993. There were four males and three females. Their ages ranged from 29 to 70 years. All patients were black, presented to hospital 6 to 36 months after the onset of the ulcers. The ulcers were fungating with black colouration. The diagnosis was made on clinical basis and confirmed histologically. The disease at presentation was advanced, in patients was wide excision and groin node dissection. Wound sepsis occurred in three patients. Two of the three patients had sepsis at the primary site and one of the three patients had sepsis at the groin. One of the patients is presented and literature on the topic is discussed.

INTRODUCTION:

Malignant melanoma is a neoplasm of the melanocytes with a potential for rapid invasion, widespread metastasis and subsequent death.[1] The original description of melanoma in Britain was by Norris in 1820.[2]

It has been recognized since 1897 that the melanocyte, which is the cell of origin of melanoma, is also the cell of origin of pigmented, benign lesions.[3] The production of melanin by melanocytes in normal skin affords protection from

sun exposure, as melanocytes replicate in response to ultraviolet light.[4] Melanocytes are also a component of 'naevi' defined as malformations of the skin, containing a 'local excess' of components of normal skin, notably melanocytes, where they are referred to as 'naevus cells'.[4]

Malignant melanoma has a world wide distribution but a high frequency is reported in people with fair skin residing in geographical locations near the equator.[5] The highest incidence of melanoma is reported in the tropical and subtropical regions of Queensland in Australia with a reported annual incidence rate of 32/100000.[6] New Zealand which is further south has a lower annual incidence rate of 14/1000000.[7] The earliest recorded case of melanoma in Africa is found in the notes of Sir Albert Cook (1904)[7] who was working at Mengo Hospital in Uganda. Subsequent studies have showed that the disease is fairly common elsewhere in Africa.[8,9] Several modalities of treatment have been tried with or without success.[10-13]

CASE REPORT:

A.S. (Reg No. 746903) a 70-year-old male peasant from Dodoma, presented with a 10 months history of a non-healing ulcer on the left heel. He had a black pigmented spot which started itching gradually followed by formation of a blister which bursted and did not heal as expected. The ulcer was accompanied by pain and bleeding at times. During the same period, he noticed painless groin swellings. His general

condition remained good with no fevers, loss of appetite or weight. There was no history of either chest pain, cough or abdominal pain. His bowel habits remained normal. There were no neurological deficits.

Physical examination showed that he was in good general health. He was afebrile, not pale and not jaundiced. He had no suspicious skin lesions elsewhere. Local examination showed a fungating, easily bleeding ulcer of about 5x6cm in size on the plantar aspect of the left heel. The ulcer had irregular margins with raised edges and there was black pigmented discharge. The base of the ulcer was fixed to the underlying tissues. The vertical group of inguinal lymph nodes on the left were palpable (4x5cm), hard, mobile and non tender. No abnormality found on examination of the respiratory system, cardiovascular system and the abdomen. Chest x-ray and abdominal ultrasound showed no evidence of metastasis either to the lungs or the liver respectively. A diagnosis of malignant melanoma in clinical stage II was made.

The patient was prepared for a wide excision of the ulcer to be done under general anaesthesia. The operation site was cleaned with iodine and spirit. Draping was done as usual. The ulcer was excised with a clear margin of 2.0 cm and the excision was done deep to the underlying fascia which was excised with the ulcer base. The whole excised tissue with labelled margins was sent for histological examination. Haemostasis was achieved and the wound was dressed with

Vaseline gauze and then pressure bandaging was applied. Post operatively the patient received pethidine for the first 24 hours for pain. He recovered uneventfully. The ulcer was being dressed daily.

The histology results of the primary lesion confirmed the diagnosis of a malignant melanoma. The pathological staging was Clark's level IV of invasion and the excised margins were free of tumour cells.

Left groin lymph node dissection was done 21 days after wide excision of the primary tumour. This was also done under general anaesthesia. At operation with the patient under general anaesthesia, cleaning and draping of the site were done as usual. An oblique inguinal incision was made 2cm below the left inguinal ligament with a vertically downward extension medially and an elliptical incision around the prominent inguinal nodes. Skin flaps were raised, the external oblique aponeurosis was exposed and superficial blood vessels were secured. The long saphenous vein and its tributaries were identified and divided between ligatures 7cm below the inguinal ligament. Fatty and glandular tissue with the elliptical skin attached to the lymph nodes were cleared medially and removed. A corrugated rubber drain was placed and the skin was closed with silk number 2/0 interrupted suture with no tension. Intraoperative blood loss was minimal hence no blood for transfusion was given to the patient. The wound was dressed and a crepe bandage was applied to the left

lower limb. Postoperatively the patient was given, Pethidine for the first 24 hours for pain. On the fourth postoperative day the wound developed sepsis. Sloughectomy and Cloxacillin was prescribed according to culture and sensitivity results. The wounds granulated well hence skin grafting was done to both groin and foot ulcers.

The histology report of the groin nodes dissected confirmed metastatic disease. After two weeks post skin grafting, the patient requested to go home for some social reasons. He was discharged and advised to be reviewed at surgical outpatient clinic two months later. At follow up clinic, both wounds had healed and there was no swelling of the left limb. he was advised to be followed up at his regional hospital.

TABULAR SUMMARY

Seven patients with cutaneous malignant melanoma.

Table I: Age distribution.

Age group (years)	No
<30	1
31 - 40	1
41 - 50	1
51 - 60	1
61 - 70	3
Total	7

DISCUSSION

Pathology:

Melanomas were first described in the 5th Century BC during the times of Hipporates but a fairly accurate histologic description was detailed by Unna at the turn of the 20th Century.[14] It is now evident that cutaneous melanomas arise from epidermal melanocyte and most of the cells contain melanin although some may not have melanin giving rise to amelanotic melanoma lesion which can be characterized by a positive dopa reaction.[12,15]

A histological description of melanoma was given by Clark (1969)[16] stating that, a melanoma starts from a proliferation of atypical melanocyte in radial (or horizontal) phase within the epidermis and after a certain and variable period neoplastic melanocyte proliferation occur in a vertical direction expanding the papillary dermis with invasion of reticular dermis and subcutaneous fat. Increased dermal invasion is associated with increased potential for metastasis.[16] The horizontal and vertical growth pattern is accompanied with unique clinical features from which melanoma is classified into subtypes: Lentigo maligna melanoma (LMM), superficial spreading melanoma (SSM), Acral lentiginous melanoma (ALM), and nodular melanoma (NM).

The depth of invasion of the primary tumour is closely associated with increased risk of distant metastasis and poor prognosis. Currently two microscopic methods are used in

assessing the depth of invasion:

Clark (1969)[16] described levels of dermal invasion of the primary lesion to various histologic landmarks as follows:

Level I - intra-epidermal melanocytic atypism limited to the basement membrane (in situ).

Level II- invading the papillary layer but not the reticular dermis.

Level III- Filling and expanding the papillary layer but not invading the reticular dermis.

level IV- Tumor invading the reticular dermis.

Level V - Tumor invading the subcutaneous fat.

Breslow (1970)[17] described a technique of measuring the thickness of the primary lesion directly using a micrometer. In ulcerated lesion the ulcer base over the deepest point of invasion is the reference point. Specific measurements are recorded but for staging purposes lesions are categorized as shown below:

1. 0.75mm or less
2. 0.76mm - 1.5mm
3. 1.51mm - 3.0mm
4. 3.1mm or greater.

Breslow's technique is more accurate and more reproducible but both methods signify that dermal invasion by atypical melanocytes have increased risk of distant metastases and therefore poor prognosis. Apart from tumour thickness and clinical stage of the primary lesion there are other

parameters which influence the prognosis of cutaneous melanoma. Tumours located on the upper back, posterior arm, posterior neck and posterior scalp (BANS) have poor prognosis probably because lesions in these sites are recognized in late stages.[18,19] High mitotic index, presence of microscopic satellites and spontaneous ulceration of the primary lesion are also associated with poor prognosis.[19]

The spread of melanoma is highly unpredictable. The primary lesion may remain dormant for more than five years, but when it enters into aggressive phase rapid invasion of local lymphatics occurs with metastasis to the lymph nodes. Haematogenous spread may give rise to solitary or multiple metastasis to the lung, liver and brain.[12] Metastasis may also occur to unusual sites such as the bowel, heart or spleen.[12] It is now known that lymphovenous anastomoses often bypass nodes and there are naturally occurring channels that allow intact cells to pass through lymph nodes.[20] Thus, cancer cells may disseminate simultaneously by the bloodstream and lymphatic pathways.[20] Furthermore, the demonstration of circulating malignant cells, nodal malignant cells and even distant micrometastases is not invariably associated with clinical metastases or death, hence there may be no inevitable advantage from excising nodes containing micrometastases.[21] The major prognostic determinants of primary melanoma can identify patients at risk of metastases and death, but these criteria do not accurately predict the site of recurrence.[22]

Clinical features:

In this study all five patients presented with advanced lesions which were easily recognized and the clinical diagnosis was not difficult. However, early melanoma lesions may be difficult to detect. Malignant changes, should be suspected whenever a pigmented skin lesion shows changes in colour or growth pattern. Variegation of colour and irregularity of borders of a naevi often with a notch are early signs of melanoma and histological examination is necessary.[12]

Clinical features of cutaneous melanoma can be described according to clinical types: Lentigo Maligna Melanoma (LMM); is a slow evolving pigmented lesion occurring mainly on the exposed cutaneous surface of elderly persons. It constitutes 10% of all melanoma and twice as common in women as in men.[15] Non invasive phase called lentigo maligna evolves from a tan freckle-like lesion that slowly increases in size to 10-20cm or more over years. The site of invasion is marked by brown-black raised area or nodule and is histologically characterized by invasion of the dermis by the atypical melanocyte. At this state the lesion acquires metastatic potential. LMM is the least aggressive type of melanoma.[18]

Superficial spreading melanoma (SSM); commonly arises on the trunk in men and extremities in women and accounts for 60-70% of all cutaneous melanoma.[15] SMM lesions are circular with irregular surface and elevated palpable margins. A distinct

nodule may appear late in the course of the disease characterized by haphazard combination of colours including hues of tan brown, bluish black, pink or dull rose.[18] Histologically there is extensive intradermal spread of atypical melanocyte that may persist for up to five years before vertical dermal invasion occurs.[14] SSM has moderate malignant aggressiveness.[15]

Acral lentiginous melanoma (ALM); describes lesions that appear at the palms, soles of the feet or in the ungual and periungual areas. It also includes lesions in the mucous membranes such as oral, anal and nasal mucosa. Clinically and histologically, acral lentiginous melanoma is similar to lentigo maligna melanoma, with characteristic extensive horizontal growth phase attaining the size of several centimetres in diameter over a relatively diminished dermal elements.[18] ALM is the most common melanoma seen in blacks.[9] Because of its body location the lesions often present in advanced stages with poor prognosis, despite a favourable histology.[19] All patients in this study presented with advanced acral lentiginous melanomas. The lesions were located on the sole of the heel in all five patients. The ulcers were fungating with black pigmented discharge and all patients had enlarged hard inguinal nodes.

Nodular melanoma (NM); is a rapidly developing nodule or plaque that occurs commonly on the back, the head and the neck. It accounts for about 12% of all melanomas and has a

predilection for men.[15,18] Clinically the lesion may appear as a spherical blue berry like nodule with a relatively smooth surface and uniform blue black colour. Histologically the tumour is characterized by minimal epidermal component overlying atypical melanocyte in the dermis. Because of little radial growth the size of the lesion is usually small and the overlying epidermis often shows ulceration.[14,16] Nodular melanoma has an aggressive behaviour with potential for early metastases and carries the worst prognosis.[18]

Non pigmented or amelanotic melanoma accounts for about 2% of all melanomas and has distinctive biologic behaviour.[23] It can develop anywhere on the body and is more common in females. The tumour grows rapidly with 75% of cases presenting with metastases. Approximately 33% of the patients are first seen with occult primary tumour in contrast to 7% in pigmented lesions.[23] Histologically the lesion is composed of authentic melanocyte with complete absence of melanin at both primary and metastatic sites. Characterization of the tumour can be done by a positive 'dopa' reaction.[12] None of the studied patients had amelanotic melanoma.

Diagnosis:

Most lesions of melanoma can be diagnosed clinically but definitive diagnosis should be confirmed by histology. Biopsy specimen can be obtained by excision or incision of the lesion and should include full thickness of the skin and some subcutaneous fat in order to allow full histological

assessment i.e. level of invasion. It has been shown that incisional biopsy prior to definitive surgery does not increase the risk of metastasis and that frozen section diagnosis is reliable.[24]

Regional lymph nodes are usually assessed clinically whereby enlarged hard and fixed nodes are signs of infiltration. Open biopsy of lymph nodes is not recommended to avoid transection of the lymphatics while random biopsy or lymph node sampling by needle aspiration is not reliable. Only positive result should be relied for management plans. Occult metastasis can be detected by using serologic or biochemical assay or tumour surface markers such as melanogen and other by-products of melanin pigment synthesizing pathway such as dopamine and levodopa but this can only detect advanced lesions and often fail to localize the lesions.[25]

Terminal appearances of liver, spleen or brain metastasis can be detected by organ specific radionucleotide studies. Conventional x-ray examination and ultrasound may pick advanced lesions in the lungs and liver respectively.[24]

In this study all patients were provisionally diagnosed as having melanoma. Histological diagnosis of primary lesions was made in all patients from excisional biopsies taken. All patients had enlarged hard inguinal nodes. Histological involvement of the inguinal nodes was made from the dissected nodes. Ultrasound was used to detect liver metastasis and a plain Chest x-ray was taken for each patient to rule out lung

metastasis. None of the patients had liver and lun metastasis.

Treatment:

The primary lesion is currently treated by surgical excision and the regional lymph nodes are dissected in some patients. However, there are controversies regarding the margin of excision of the primary lesion and the time and extent of dissection of regional lymph nodes.

Primary lesions.

Handley (1907)[26] observed that local spread of melanoma and invasion of lymphatic and blood vessels occurred in a centrifugal manner so that wide excision could effectively control the disease before spreading to distant sites.

A number of investigators [27,28,29] reported that thin lesions (measuring less than 0.76mm) rarely recur with margins of 1 to 2cm. A recently reported World Health Organization (WHO) melanoma group trial randomized lesions up to 2mm in thickness of narrow excisions of 1cm versus a 4cm radial margin. There was no statistically significant difference in the local recurrence rate in this randomized trial, but all the observed local recurrences were in the narrow-margin group. Follow-up on this series was short, and such a trial must have a longer follow-up to be accepted as valid.[30] However until the optimum width of excision of a primary melanoma can be determined by longer follow-up of current trials, Marton (1990)[30] and his colleagues have adopted the

following guidelines based on the observation that the risk of local recurrence is directly proportional to the depth of the primary melanoma:

Clark's level I, any margin of excision (minimal)

Clark's level II or III (<0.75mm), excision margin of 2-3cm

Clark's level III or IV (<3.9mm), excision margin of 3-4cm

Clark's level V or (>4mm), excision margin of 5cm.

Excision of the fascia is recommended in all lesions except for thin lesions (<0.76mm). Lesions located in the finger or toe may require amputation or disarticulation of a digit in order to achieve adequate margin. Symes amputation or a below knee amputation may be necessary for excision of advanced lesions of the foot.[31]

In most of the studies, tumour thickness, level of invasion and excision margin have been determined after excision. A more practical approach is proposed by Day et al[32] who recommended that, clinical stage I lesions (Primary lesion with no evidence of regional lymph node or distant metastasis) defined as low risk, excision margin should not be more than 1.5cm. All other tumours require at least 3cm margin of excision.

Regional lymph nodes.

The rationale for elective dissection is based on the hypothesis that cutaneous malignant melanoma spreads, in a stepwise manner, from the primary to the regional nodes and then to distant sites.[33] If this is true, then patients with

occult micrometastases in the regional nodes should benefit from elective lymphadenectomy, because it is performed when the tumour burden is small and before systemic spread occurs. By the time clinically detectable nodal metastases are established, most patients (70-85 percent) will already have distant micrometastases and the 10-year survival rate is around 25 percent.[34] The aim of elective lymph node dissection is, therefore, to provide definitive surgical treatment at an early stage in the natural history of the disease. Balch and Milton contend that the Breslow thickness of the primary melanoma is the major indicator of the group of patients who might benefit most from elective lymph node dissection.[35] They argue that primary melanoma of Breslow thickness 0.76 - 4mm has a high risk of occult regional metastases, up to 60 percent in patients with lesions 1.5 - 4.99mm thick,[33] and a low risk of systemic metastases and that such patients should benefit from elective lymphadenectomy.

There is discussion in literature about the extent and timing of elective lymph node dissection. There is no evidence to confirm whether inguinal or ilioinguinal dissection is more appropriate in the management of lower-limb disease. The major proponents of elective lymph node dissection perform an inguinal procedure[33,36] and the iliac glands are spared unless the inguinal nodes are found to contain metastases. It has been argued that lymphatic stasis, which is common after elective lymphadenectomy, may provide a

favourable environment for the survival and proliferation of melanoma cells.[37] Furthermore, immediate elective lymph node dissection may result in the seeding in transit of intralymphatic cells that might otherwise reach and be contained by the draining nodes, a mechanism that provides a rationale for 'interval' elective dissection. Petersen and colleagues[38] found that interval elective lymph node dissection, i.e. delayed more than 14 days after primary excision, was associated with a lower incidence of locoregional recurrence. Wanebo et al[39] found no benefit from similar approaches and the only prospective study that addresses this question is too small to provide a definitive answer. In the absence of good evidence in favour of interval elective lymph node dissection, those who carry out elective lymphadenectomy tend to do so along with definitive excision on the primary lesion on the same sitting.[35]

Evidence is emerging that may explain why, in malignant melanoma, treatment of the regional lymph nodes fails to influence survival. It is now known that lymphovenous anastomoses often bypass nodes and there are naturally occurring channels that allow intact cells to pass through lymph nodes.[20] Thus, cancer cells may disseminate simultaneously by the blood stream and lymphatic pathways; lymphnode metastases can be considered 'indicators, but not governors' of survival.

Protagonists of elective lymph node dissection often argue[34], that the prognosis of patients who have microscopic regional nodal metastases, diagnosed after elective lymphadenectomy, is better than that of those who undergo therapeutic node dissection for clinically apparent nodal metastases. However, it is not surprising that there is a survival advantage for patients with a microscopic tumour burden compared with those with a clinically obvious malignant lymphadenopathy.[11] Although the overall survival figures for patients undergoing elective lymph node dissection seem favourable,[11] it does not follow logically that such dissection benefits all those who undergo the operation. This is because many such patients undergo unnecessary operation as the excised glands have no demonstrable metastases and prognosis would have been good in any case. Conversely, those who have therapeutic dissection all have clinically apparent nodal metastases and the comparatively poor prognosis is related to the stage of disease.[11]

Adjuvant therapy.

Adjuvant therapy for malignant melanoma has been explored intended to become an integral part of treatment for controlling local disease and associated distant metastases. Patients with regional lymph node metastases have 50-85% chances of developing distant metastases and those with histologically negative regional lymph nodes also have 15-20% chances of developing distant metastases.[13]

Immunotherapy.

Bacille Calmette Guerin (BCG) is an agent which has been used to enhance non specific immune response to control local disease and distant metastasis for melanoma. Earlier results with BCG in non randomized trials showed that BCG was effective in delaying local recurrence and there was improved survival after surgery. However, subsequent carefully randomized studies failed to confirm the usefulness of BCG as an adjuvant treatment for melanoma.[13] Other agents which have been tried include Levamisole, specific active immunization with irradiated neuraminidase treated melanoma cells. None of these have proved to be useful as adjuvant in the treatment of melanoma.

Chemotherapy.

Clinical trials with Dimethyl triazeno imidazole carboxamide (DTIC) and Methyl CCNU have failed to show their usefulness in controlling metastatic melanoma. These drugs which have been extensively used in clinical trials have been discontinued for routine medical use in the treatment of melanoma.[13] Other agents currently under clinical trials include the use of melphalan for isolated limb perfusion technique with hypothermia. So far no effective chemotherapy has been found for treatment of malignant melanoma.

Radiotherapy.

The role of radiotherapy in the primary treatment of cutaneous melanoma is limited because the tumour does not

respond to irradiation and the superiority of surgical excision has been well documented. However there are isolated cases where melanoma has responded to radiotherapy.[40,41] In this study none of the patients received adjuvant therapy.

CONCLUSION/RECOMMENDATION:

Treatment of cutaneous melanoma is successfully achieved when the lesion is still in its early stages. At present surgical excision of such lesions has been proved to give good prognosis and therefore early detection of lesions has to be emphasized. In this study patients presented with advanced disease although lesions were noted earlier in the course. Health education should be given to the general public through news media advising them to seek medical attention early when they notice suspicious lesions or changes occurring on previous skin marks. However, a high suspicion index has to be clear to the medical personnel at all levels to enable early detection of melanoma lesions.

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VARICOSE VEINS IN THE LOWER EXTREMITIES

SUMMARY:

Four patients with varicose veins of the lower extremities were admitted to the surgical department of the Muhimbili Medical Centre between January, 1992 and December, 1993. All patients ranged between 30 and 39 years.

There were three males and one female, all of them presented with prominent vessels and discoloration of the skin. Three patients had non healing varicose ulcers of the lower limbs. One patient had swelling of one leg, cramps in the calf muscles and eczema. On admission, all patients were clinically evaluated by Brodie-Trendelenburg, Perthes' and Linton tests. The patients who presented with ulcers were not subjected to operative procedures until their ulcers had healed. Three patients underwent Trendelenburg operation and stripping of the long saphenous vein (LSV). One of the four patients underwent Trendelenburg operation and sclerotherapy.

One of the four patients is presented in detail and then literature on the topic is reviewed.

INTRODUCTION:

The term varicose is derived from Latin and means dilated.[1] It implies a dilated, tortuous, and elongated vein. The World Health Organization has defined varicose veins as saccular or cylindrical widened superficial veins that can be

circumscribed or segmental.[2] Although varicosities may occur in any venous system, as in the lower oesophageal area, the anorectal area, or the spermatic cord, they most frequently occur in the lower limbs.[1]

Varicose veins are classified into a primary and a secondary form. Primary varicose veins are not associated with deep venous pathology, whereas secondary varicose veins are thought to be a consequence of deep venous insufficiency. Primary varicose veins are by far the most common form.[2]

Varicose veins are a highly prevalent problem that continues to rise in incidence, and affects a large percentage of the adult population. It has been estimated that 24 million people in the United States have varicose veins.[3] The prevalence of varicose vein increases with age and peaks between the fifth and sixth decades of life, it is reported that in the United States, the prevalence increases from less than 1% during the third decade of life, to over 50% in the population over 70 years of age. [2,4] Varicose veins are more common in women, with a female-to-male ratio that varies from 2:1 to 3:1 in different populations[2,4] however, the sex ratio decreases with advancing age, and almost disappears in people over 70 years old. Style of sitting such as chair sitting and ground sitting has been cited as a risk factor for the development of varicose veins .[5] Prolonged standing has also been implicated in the development of varicose veins although there are no solid epidemiologic data that clearly support the

association between standing at work and increased incidence in varicose veins. About 50% of the patients with varicose veins have a family history of the disease.[2]

In this study, four patients were admitted, evaluated and then treated. This was done in order to compare; presenting features, tests used for evaluation,[6] modes of treatment chosen,[7,8] and the outcome, with findings in other centres.

CASE REPORT:

H.V. (Reg.no 725809) a 36 year old male patient was referred to Muhimbili Medical Centre from Kilimanjaro region. He presented with an eight-year history of swelling of the right leg and a two -year history of an ulcer on the same leg. Swelling of his leg was of gradual onset accompanied by prominence of his veins mainly on standing still for some time. He noted discoloration of the skin and that wounds on the affected leg were taking long to heal despite treatment given. He gave no history suggestive of diabetes mellitus. He was married, a social alcoholic drinker and smoked cigarettes occasionally.

On examination, he was in good general health. He was afebrile, not pale and not jaundiced. He had palpable firm, descrete non tender groin lymph nodes on the right. His radial pulse rate was 80/minute, regular, of full volume and a blood pressure of 140/80 mmHg. Pulses of the femoral, popliteal and dorsalis pedis arteries were bilaterally synchronously

palpable. Locally, he had hyperpigmented skin on the distal half of the right leg. He had an ulcer on the medial aspect of the right ankle 3cm at its greatest diameter with slopping edges, the floor was covered with pus and its base was indurated. The long saphenous vein was visible on standing, extending from the ankle up to the groin. Three tests were done; the Brodie-Treandelenburg, Perthes's and Linton. The procedure of these tests is as follows; After inspection of the legs of a standing patient, Perthes' test is performed, this involves application of a tourniquet just above or below the knee and then instructed to walk. If the varices empty, the communicating and deep systems are functioning, thus the diagnosis is primary varicose veins. If the varices do not empty, the patient is instructed to lie down and raise his leg with the tourniquet in situ. This manoeuvre is the Linton test. If the varices now empty, the diagnosis is still primary varicosity but there is evidence that the valves in the communicating vessels are also incompetent. If the varices do not empty, this is evidence for obstruction in the deep system, thus the varices are secondary.

The Brodie-Trendelenburg test is performed by instructing the patient to lie down and elevate his leg until all varices are empty. His greater saphenous vein is occluded at the groin (either by digital compression or by a tourniquet) and then is told to stand up. If the varices do not fill within 30 seconds, the valve of the communicating system are

competent. The obstruction is then removed, and if the varices fill from above down, the valves of the superficial system are incompetent. These tests showed that there was incompetence of the sapheno-femoral and the perforators' valves and that the deep venous system was patent.

Investigations done: Full blood picture (Hb=16.0gm/dl, total count = 7,000/ul. differential count was reported as normal). Pus swab for culture and sensitivity which revealed Staphylococcus aureus sensitive to Erythromycin. His Blood group was established.

In view of the above findings the decision was surgical treatment of the varices after the ulcer had healed. He was advised to rest in bed with his right lower limb elevated on pillows. Daily saline dressing of the ulcer was ordered and a five day course of Erythromycin was prescribed. In three weeks time the ulcer healed and the patient was prepared for high ligation and stripping of the long saphenous vein. Pre-anaesthesia, the patient was made to stand and all varicose veins were marked with a marker.

Under general anaesthesia, the whole right lower limb was cleaned with iodine and spirit. Draping with sterile towels was done as usual. The operation was done in stages as follows; first Trendelenburg operation was done by making an incision 8cm long, below and parallel to the medial half of the inguinal ligament at the level of the saphenous opening which is 3cm below and lateral to the pubic tubercle just

medial to the femoral pulsation. The fat was separated by blunt dissection in the line of the saphenous vein. Then the vein was cleared to a level 8cm below the opening. By using a clamp as a convenient tractor, the upper segment of the vein was raised from its bed and its tributaries were identified. These are; the medial and lateral femoral cutaneous tributaries and the superficial external pudendal, circumflex iliac and inferior epigastric branches. They were clamped, ligated and divided. The superior margin of the saphenous opening was retracted upwards, so as to clear it up to its junction with the femoral vein. A nonabsorbable suture (Silk no 2/0) was used to ligate the upper end of the long saphenous vein. A clamp was applied 2cm away from the ligature and then division of the vein was done. The second step was to introduce a stripper; a transverse incision on the medial aspect of the upper 1/2 of the calf was made to expose the long saphenous vein which was later divided between clamps. A stripper was introduced retrogradely until it emerged through the upper end of the vein in the groin incision. It was drawn upwards until arrested by the acorn-head. A ligature of strong silk (no 2) was then tied firmly around the vein, so that the vessel was anchored against the acorn-head. Lastly, stripping of the long saphenous vein by applying traction to the stripper was performed. The wounds were dressed, the right lower leg was elevated and then an elastic bandage was applied from foot to groin. The maximum pressure was applied on the

foot and was decreased gradually upwards.

Post operatively, the patient was kept in bed with his right lower limb elevated on pillows, Pethidine was prescribed for pain during the first 24 hours. Vital signs were monitored regularly. After the first 24 hours postoperatively, the patient was allowed to walk. On the eighth postoperative day, the wounds were inspected and found clean hence, sutures were removed and then the elastic bandage was reapplied. The patient was allowed home to stay with relatives nearby. He was seen at the outpatient clinic after one month. On examination, there were no varices, no pitting oedema and the skin of the right leg was resuming its normal colour. In view of the above findings, he was discharged back to his home region for follow up.

TABULAR RESULTS

Table I: Sex distribution

Sex	Number
Male	3
Female	1
Total	4

Table II: Presenting clinical features

Signs and symptoms	Number
Prominent vessels	4
Discoloration of the Skin	4
Ulcer	3
Eczema	1
Leg swelling	1
Cramps in the calf muscles	1

DISCUSSION

Pathophysiology:

The pathogenesis of varicose veins has classically been attributed to weakness of the venous wall that yields to venous dilatation, and to valvular incompetence of the main saphenous and perforating veins.[9-11] There is evidence that venous distensibility is higher than normal in patients with varicose veins and that smooth muscle cells from varicose vein walls are capable of phagocyting and decomposing collagen fibrils, characteristics that could account for the venous dilation present in varicose veins.[12-15] Whether the increased distensibility and collagen reabsorption are the cause or the effect of venous dilatation remains unclear.[15]

Primary varicose veins produce an increase in ambulatory venous pressure secondary to the retrograde flow through the main saphenous channel.[16] A study[17] done proved resting interstitial tissue pressure in the deep posterior compartment of the leg is uniformly elevated in patients with varicose veins, regardless of the presence or absence of clinical symptoms. This fact reflects the pathologic accumulation of interstitial fluid secondary to disturbed transcapillary fluid exchange.

On the other hand, greater saphenous vein incompetence has long been considered a prevailing factor in the pathogenesis of varicose veins. It has been suggested that

valvular incompetence develops in a sequential descending fashion and that it preceded venous dilatation.[18] Furthermore, venous dilatation could be secondary to the turbulence associated with retrograde flow in veins with incompetent valves.[19] This theory is reinforced by the fact that patients with varicose veins, when compared to normal individuals have a high incidence of incompetent or absent valves above the saphenofemoral junction.[20]

Doppler investigation of an unselected population of patients with leg varices revealed the presence of partial or complete incompetence of the greater saphenous vein in 95% of the affected limbs.[21] Lesser saphenous vein incompetence was present in 15% of the limbs and 75% of these were associated with an incompetent greater saphenous vein. Perforator vein incompetence was diagnosed in 45% of the studied extremities, and deep venous insufficiency was present in 20% of the limbs.[21] In the same study,[21] 17.6% of the legs with varicose veins had chronic skin changes in the form of hyperpigmentation, lipodermatosclerosis, and healed or active ulceration. In this study all four patients had primary varicose vein. The ulcers and skin discoloration were ascribed to the varicose veins.

Increased oxygen content in the saphenous vein of patients with varicose veins has been documented,[22] suggesting arteriovenous shunting. These arteriovenous communications, however, very rarely drain into the main saphenous trunks, and

most likely would increase pressure in small saphenous tributaries. Therefore, increased venous pressure as a result of pre-capillary arterial blood shunting may play an important role in the pathogenesis of varicose veins.

Clinical features:

Patients with varicose veins often complain of aching, heaviness, itching moderate swelling, marked discoloration and frequently unsightly appearance of their legs. In rare instances skin eczema, ulceration or bleeding of the varices are reasons for reporting to hospital.[1]

Fibrinogen escapes through large pores in the venules of those patients with venous hypertension secondary to venous insufficiency of the lower limb. The accumulating fibrin cannot be removed because of inadequate blood and tissue fibrinolysis.[1] This accumulation acts as a barrier to diffusion of oxygen, and other nutrients, hence thick, hard, tender subcutaneous tissue (liposclerosis) develops. This changes of stasis dermatitis characterized by brawny oedema, subcutaneous fibrosis, pigmentation, and cutaneous atrophy may lead to tissue death and ulceration.[23]

In this study four patients had discoloration of the skin. Three of the four patients had leg ulcers. In Almgren and colleagues' study 17.6% of the legs with varicose veins had chronic skin changes in the form of hyperpigmentation, lipodermatosclerosis and healed or active ulceration.[21] A chronic venous ulcer is the end stage of lipodermatosclerosis.

It is typically situated in the retromalleolar region on the medial side of the gaiter area, just below the calf perforators. The ulcer is usually single, large and shallow with a base of unhealthy granulation tissue and is surrounded by an area which is pigmented, indurated and eczematous.[1]

In this study prominent blood vessels were found in four patients. Eczema, cramps in the calf muscles and swelling of the leg were present in only one patient (table II). The data was too small to compare with other series.

Diagnosis:

A careful history should be taken, particularly noting thrombotic episodes, previous vein surgery, arterial symptoms, diabetes mellitus, locomotor problems and allergies.[1]

Assessment of the patency and valvular competence of the deep and superficial venous systems is the most important aspect in the evaluation of patients with varicose veins. The valvular competence can be assessed on physical examination with the Brodie-Trendelenburg test and the Perthes test can roughly estimate the presence of deep venous obstruction.[1]

In this study, only physical examination was done to evaluate the valvular competence of the superficial venous system. A detailed history ruled out deep venous thrombosis. Hence it was assumed that the deep venous system was patent as patients with varicose veins and no signs or history of deep venous thrombosis are most likely to have patent deep system. The tests done were; the Brodie-Trendelenburg, Perthes and the

Linton tests.

However, evaluation of the venous system by physical examination, although useful, is unreliable and unable to provide much information about the deep system. Accurate documentation of deep venous patency is best done by Doppler flow detection, duplex scanning or plethysmographic techniques. Duplex scanning is favoured because of its capability to identify nonobstructing or floating thrombus, and sometimes to differentiate between fresh and old thrombus. Saphenofemoral valve incompetence can be demonstrated by detecting reverse flow in the saphenous system with a Doppler probe after releasing a thigh tourniquet[6]. Another investigation which can be done is varicography performed by injecting a low osmolality contrast. This can sometimes be a useful method for delineating the anatomy of complex or recurrent varicose veins, for the planning of surgical excision.[24] Varicography was not done in this study due to lack of facilities.

Treatment:

Both surgical and nonsurgical methods are used in the management of varicose veins.[1] In general, the surgical removal of incompetent veins is the more definitive treatment, because the results are more satisfactory and lasting.[1] Nonsurgical methods are generally reserved for patients who have medical contraindications to surgical treatment, deep venous insufficiency or very minimal varicosities. The

nonsurgical methods include sclerotherapy, elastic support, periodic elevation of the lower extremity, and exercise of the leg muscles.

Surgical treatment:

The general indications for surgical treatment are;

. Symptoms of aching, heaviness and cramps,

. Complications of venous stasis such as pigmentation, dermatitis, induration, superficial ulceration and thrombosis of varicosities,

. Large varicosities subject to trauma,

. Cosmetic concern.

The objectives of surgical treatment are:-

. To relieve symptoms,

. to alleviate stasis complications,

. to restore normal venous physiology,

. to improve cosmetic appearance.

Ideal candidates for surgery are those patients who have a dilated and incompetent greater saphenous vein with a patent deep system, and competent valves in the deep communicating veins.[25] In these patients high ligation and stripping offer excellent long-term results.[25] However, in one-third of patient with varices in the greater saphenous vein, valvular incompetence is limited to a segment of the vein. Therefore with the aid of Doppler ultrasound, the incompetent segments can be mapped and selective venous stripping carried on, with results comparable to those of standard stripping.[26] This

approach has the advantage of preserving segments of healthy saphenous vein as a potential vascular conduit, and of minimizing the incidence of saphenous and sural nerve injuries. [26,27] However, saphenous nerve injury can also be prevented by stripping the vein caudally from the lower incision, rather than from the groin.[25] Furthermore, stripping of the greater saphenous vein limited to just below the knee level may achieve results similar to those of standard stripping in many cases.[25,27]

In this study three of the four patients underwent stripping of the long saphenous vein to just below the knee, No postoperative complications encountered probably because of a relatively short postoperative follow up and the small data collected.

The need for saphenous vein stripping for the treatment of varicose veins had been challenged in a study where high ligation offered results comparable to those with vein stripping suggesting that saphenous removal is unnecessary as long as incompetent perforators have been ligated.[28] Removal of varicose tributaries by avulsion is necessary in many cases, as a complement to saphenous vein stripping or as an isolated procedure.[2]

Sclerotherapy:

Sclerotherapy is most effective for the treatment of small-calibre varices without underlying valvular incompetence.[1] Superficial venular dilatations,

telangiectasias, and venous spiders are best suited for this form of therapy.[29] With appropriate patient selection, sclerotherapy can achieve excellent cosmetic results and relief of symptoms.[29] Sclerotherapy is a good adjunct for the treatment of residual tributary varices, following high ligation and stripping or other forms of surgery.[29]

The aim of sclerotherapy is to inject a small volume of an effective irritating substance into the variceal lumen that produces an inflammatory reaction and eventual fibrotic occlusion of the vessel.[1] The injected sclerosant destroys the venous intima. Compression therapy following injection of small varices is probably unnecessary; however, when treating medium-sized and large lesions, compression therapy seems to be an important factor for the success of the procedure.[1,30] If compression is not applied, a large thrombus forms which soon recanalizes. This process may result in destruction of valves so that the venous pathology is actually worsened.[1] If the sclerosant is injected into an empty vein and external compression is maintained until permanent fibrosis had obliterated the lumen, good results may be obtained.[1]

The sclerosing agents currently used include; Sodium tetradecyl sulphate hypertonic dextrose, sodium chromate in glycerin, hypertonic sodium chloride and ethanolamine. In this study only one patient underwent sclerotherapy by using ethanolamine following high saphenous vein ligation.

Optimal results with sclerotherapy depend on excellence the execution of vein injection and leg bandaging. The varices should be marked while the patient is standing, and the injection made with the patient in horizontal position. When large varices are injected, however, these are emptied by leg elevation prior to injection. Fine needles (25 to 30 gauge) are used, and small volumes (0.25 to 0.5 ml) of sclerosing agent are injected while the vein is collapsed by digital pressure. Injection is only made if there is absolute certainty that the needle is in the vein lumen, and compression is immediately applied with a cotton ball held with paper tape over the puncture site.[31] The number of injections should be limited to no more than 20 to 25 in a session.[31] On completion of all the injections, the leg is rapped with elastic bandages that should be regularly reapplied to maintain adequate compression during the prescribed period of time, which ranges from 1 to 6 weeks. Alternatively, graded high-compression stockings can be placed immediately after sclerosis. Small veins (<1 mm) probably do not require post injection compression but it is believed that the larger the varices, the longer the period of compression that should be prescribed.[31] In this study an elastic bandage was used and was being regulated at intervals to achieve adequate compression.

There are two main sources of failure with sclerosing therapy. These are; inappropriate technique and inadequate

patient selection. Recurrence of the varices is normally secondary to vein recanalization due to persistence of proximal valvular incompetence or to variceal thrombosis, rather than sclerosis followed by lysis of the thrombus. Therefore in the presence of saphenous vein incompetence, sclerosis of the main trunk will carry a very high recurrence rate.

Although unusual, allergic reactions to the sclerosing agent are a potential hazard that can manifest as a local reaction at the injection site or more rarely as generalized anaphylaxis. These reactions never occur with the use of hypertonic saline solution.[30] Extravasation necrosis is the most serious local complication, and can lead to adverse cosmetic results secondary to skin necrosis.[30] Extravasation necrosis is unrelated to the type of sclerosing agent used, therefore fastidious technique is its best preventive measure. For the ethanolamine used in one patient, no short term complication occurred. Transient leg cramping can occur following injection of hypertonic saline solution but it disappears spontaneously in a matter of minutes.[30] Skin hyperpigmentation at the injection site occurs secondary to variceal laceration that allows extravasation of blood with subsequent haemosiderin deposition.[32] Meticulous injection technique and adequate compression minimize this complication. Thrombophlebitis following sclerotherapy is frequent, and pulmonary embolism is rare.[30]

Compression therapy:

Varicose veins tend to develop progressive dilatation, lengthening and tortuosity. The lack of competent valves in varicose vein makes them unable to empty properly despite vigorous leg exercise. However, chronic blood pooling and hypertension in the superficial system can be decreased by applying constant compression on the superficial veins. Therefore, elastic compression therapy might prevent further progression of varicose veins by counteracting the underlying haemodynamic derangement.[33]

In this study compression therapy was not used as a separate mode of treating varicose veins. The main reason is that, the patients studied had varicose veins with complications at the time of admission. Hence compression therapy was undertaken as part of postoperative management following surgical therapy of varicose veins.

CONCLUSION/RECOMMENDATION:

Varicose veins are not uncommon in our community. The patients who are seen in referral centres are those with complicated varices. Those with uncomplicated varices probably are managed in regional and district hospitals, dispensaries and the Maternal and Child Health (MCH) Clinics.

Evaluation of the varices is important as the treatment for primary and secondary varices is different. The tests which can be used even in other centres are the non-invasive

ones such as; the Brodie-Trendelenburg, Perthes' and the Linton tests. The medical personnel who see the patients first should be aware of such a problem and refer them to proper centres for further evaluation and proper treatment. This is important because venous wall and valvular damage is progressive in untreated varicose veins and to prevent complications.

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MANAGEMENT OF SIGMOID VOLVULUS

SUMMARY:

A total of twelve patients with sigmoid volvulus were managed in the three surgical firms of Muhimbili Medical Centre between January, 1992 and July, 1993. There were nine males and three female patients. Their ages ranged between 23 and 65 years. The majority of patients were aged above 30 years. All patients presented with a history of abdominal pain followed by constipation and abdominal fullness. The rest of the features were; vomiting (8), dehydration (4), shock (2), peritonitis (2) and fever (2).

Plain abdominal x-ray examination was done in all patients and radiological features of sigmoid volvulus were present in ten patients. Laparotomy was performed in all patients following initial resuscitation. Operative procedures performed are as follows; resection and double-burrel colostomy (9), Hartimann's procedure (2) and resection and primary anastomosis (1). The operative findings were; viable sigmoid volvulus (8), gangrenous sigmoid volvulus (3) and ileosigmoid knotting with gangrenous ileum (1). There were two operative deaths and four patients with wound sepsis. One of the patients is presented and then literature on the topic is discussed.

INTRODUCTION:

The first accurate description of volvulus was given by a Viennese pathologist, Rokitansky in 1837 who defined volvulus as torsion of a section of the bowel on its mesentery causing symptoms by narrowing of the lumen, strangulation or both.[1] Any part of the bowel can undergo torsion but the sigmoid colon is most commonly affected.[2]

Sigmoid volvulus has a marked geographical variation in incidence rate. The disease is common in Eastern-Europe, Russia, Scandinavia, Africa and Asia where in some regions it constitutes 30-50% of all cases of acute intestinal obstruction.[3] It is rare in Western Europe and North America forming only 1-8% of intestinal obstruction.[4] Observation has shown that there are variations even within one geographical region. One example is that while the disease is common in Kenya, Uganda and Rhodesia where it is reported to be the second commonest cause of intestinal obstruction[5] it forms only 4% of cases of intestinal obstruction in a neighbouring country-Tanzania.[6] Makwani(1984)[7] reported only 20 cases of sigmoid volvulus in a period of five years in a consultant hospital in Tanzania.

Although sigmoid volvulus is described in children and infants[8,9,10] it is commonly a disease of adults mostly affecting males.[8] In the West the peak incidence occurs in the 6th decade but studies in the tropics show that it affects younger age group mostly below 50 years old.[8] This study was

done in order to look into the sex distribution, age distribution, clinical features, diagnosis and the treatment of patients with sigmoid volvulus and compare with studies done elsewhere.

CASE REPORT:

H.K. a 36 year old male patient was admitted to Muhimbili Medical Centre with a history of sudden onset of colicky abdominal pain which was progressive, followed by progressive abdominal distension and absolute constipation of 36 hours duration. There was history of vomiting twice prior to admission. The vomitus contained previously taken food material. He gave no history of similar attacks in the past.

On examination, he was in pain, afebrile, not pale. He was moderately dehydrated with a pulse rate of 100/minute which was regular and of normal volume. His blood pressure was 120/60mmHg and a respiratory rate of 26/minutes which was regular. The abdomen was irregularly distended being more along the left iliac fossa, left lumbar region, in the epigastrium, right hypochondrium, right lumbar region and in the right iliac fossa. It was moving slightly with respiration and there were no visible peristaltic waves. He had generalized abdominal tenderness and his bowel sounds were not heard. No abnormality could be detected by digital rectal examination.

Plain x-ray films were taken in the erect and supine positions. Gaseous distension of the large bowel extending from the pelvis to the diaphragm in an inverted 'U' shape was demonstrated. Air fluid levels were visible on two limbs of the sigmoid loop (balance sign). In view of the history, clinical examination and the x-ray findings, a diagnosis of acute large bowel obstruction due to sigmoid volvulus was made. The patient was prepared for an emergency laparotomy as follows; An intravenous cannula was set and was used to pump in five litres of Hartmanns solution within $2^{1}/_{2}$ hours for correcting electrolytes and fluid loss. A urethral catheter was introduced to monitor urine output and as a means of decompressing the bladder intra-operatively. A naso-gastric tube was used to decompress the stomach. Blood sample for grouping and cross-matching was taken during the process of inserting an intravenous cannula for fluids administration. The machine for measuring serum electrolytes was out of order hence, the level of serum electrolytes was not known. The site for operation was prepared and patient's consent was obtained.

After rehydrating the patient to a urine output of about 40 mls/hour, he was taken to theatre for a laparotomy under general anaesthesia and sterile conditions. After routine cleaning with iodine and spirit, draping of the abdomen was done as usual. A midline incision was made to open the abdomen. Serosanguinous peritoneal fluid was encountered in

the peritoneal cavity with a grossly distended gangrenous sigmoid colon. The distended twisted bowel was delivered through the abdominal incision with difficult. The base of the twisted sigmoid colon was clamped by applying double intestinal clamps at the proximal and distal limbs of the sigmoid loop just beyond the gangrenous parts. Resection of the gangrenous sigmoid colon was performed between clamps. The distal segment was long enough to exteriorize hence, resection and a double burrel colostomy was fashioned through a separate incision on the left iliac fossa. Warm saline was used to wash the peritoneum. After ensuring haemostasis the abdomen was closed in one layer with prolene No.1 and the skin was not closed, instead was dressed with saline.

The patient was transfused with two units of blood postoperatively, Pethidine 100mg I.M. 6 hourly for 24 hours, Ampicillin 500mg I.V. 6 hourly and Metronidazole 500mg I.V. 6 hourly slowly for 5 days were prescribed. The nasogastric tube, intravenous fluids and urethral catheter were continued. He made a good recovery and the colostomy started functioning within the second postoperative day. His vital signs remained stable,, the abdomen was not distended and there was very little drainage from the naso-gastric tube which was therefore removed on the third postoperative day. The urethral catheter was removed on the third day and the patient was allowed to take sips of water and plain tea. After tolerating orals, intravenous fluids were discontinued. The abdominal wound

granulated well and was sutured on the seventh postoperative day.

The patient was taught how to take care of his colostomy and was allowed home on the ninth postoperative day. He was advised to go to a nearby hospital for removal of sutures and to report for check up three months later.

After three months, he was assessed and found fit for colostomy closure as an elective procedure. His haemoglobin level was established pre-operatively, total bowel irrigation with saline solution through a naso-gastric drip and by saline enema to the distal stump was performed and then Ampicillin and Metronidazole were given pre-operatively.

The operation was performed under general anaesthesia and sterile conditions. The colostomy was closed extra-abdominally and dropped into the peritoneal cavity, the abdomen closed in one layer and the skin was sutured. He did well post operatively and was allowed home on the seventh post operative day after establishing good bowel function. He was instructed to go for removal of skin sutures at a nearby dispensary. He was reviewed at the follow-up outpatient clinic twice in a period of three months, found to be in good general health and passing stool normally.

TABULAR SUMMARY:

Table I: Age distribution:

Age group (years)	Number
20 - 29	1
30 - 39	3
40 - 49	5
50 and above	3
Total	12

Table II: Presenting features:

Symptoms/signs	Number
Abdominal pain	12
Abdominal distension	12
Constipation	12
Vomiting	8
Dehydration	4
Shock	2
Peritonitis	2
Fever	2

DISCUSSION:

Pathology:

Sigmoid volvulus develops when a redundant sigmoid loop with a narrow base of attachment rotates about its mesenteric axis, producing a closed loop type of intestinal obstruction.[15] Further pathological changes are caused by compression of mesenteric circulation and by rapid and immense distension of the rotated bowel impairing blood supply to the sigmoid loop.[2,4]

Rotation of the bowel starts with the upper limb of the sigmoid loop descending in front of the lower limb, twisting in either a clockwise or counterclockwise direction accompanied by axial tension of the bowel.[15] Sometimes a physiological volvulus of 180° (half - turn) may occur without clinical symptoms usually resolving spontaneously. If spontaneous resolution does not occur subsequent hyperperistalsis, particularly in a loaded colon may cause a further twist of 180° or more in the same direction resulting in a condition reducible with difficulty or irreducible.[2] Increased peristalsis proximal to the obstruction, forces air and fluid into the twisted sigmoid loop, the site of tension acting as a valve and progressive distension of the sigmoid loop occurs. Occasionally, bowel contents may be forced into the efferent limb causing intermittent diarrhoea despite predominant symptoms of obstruction.[2,4] In addition to the closed loop obstruction, the proximal colon often becomes distended and if ileocaecal valve is competent, as is the case in 75% of the patients, no regurgitation is possible and a double closed loop obstruction develops. If the valve is incompetent, small bowel becomes distended as well.[4,15]

Torsion of the mesentery by volvulus causes venous stasis and retrograde thrombosis of the mesenteric vessels with extensive gangrene.[2] Compression of the superior rectal vessels which run a looping course into the mesentery of the sigmoid colon may result in gangrene which extends down to the

upper rectum.[16] Increased distension of the rotated bowel causes vascular hypertension in the involved loop impairing capillary circulation. Submucosal vascular plexuses are particularly vulnerable to the flattening force exerted by pressure within the lumen causing ischaemic necrosis.[15] Haemorrhagic infarction of the affected segment of the bowel may occur with loss of blood sufficient to produce shock. Toxins, bacteria and blood pass into the peritoneal cavity causing peritonitis.[17] In practice, circulatory embarrassment should be assumed if the volvulus has been present for more than six hours.[18] Shepherd[8] found that 40% of patients attended with symptoms of acute sigmoid volvulus within 24 hours already had gangrenous bowel.

In this study 4 of the 12 patients had gangrenous bowel at exploration. These patients presented to hospital three to five days after the onset of abdominal pain. Two of the four patients had been taking local medicine at home for the abdominal pain prior to admission to hospital.

Ileosigmoid knotting develops when an abnormally mobile small intestine with an elongated mesentery passes around the base of a freely mobile pelvic colon, the net effect of which is the formation of an ileal knot around the base of the pelvic colonic loop producing a double closed loop intestinal obstruction.[10] Early and severe strangulation of the knotted ileum occurs when the trapped loop of pelvic colon becomes engorged and moderately distended with air and fluid.

Gangrene of the sigmoid colon occurs in only 30% with cases of ileosigmoid knotting[19] while ileal loops involved in the knot almost invariably become infarcted, except the last 3 - 5 cm of the terminal part which usually remain viable.[10]

In this study only one patient was found having ileosigmoid knotting with gangrene of the ileum. In this patient, the sigmoid loop was oedematous and greatly distended with gas and haemorrhagic fluid.

Clinical features:

In this study all patients had history of abdominal pain followed by constipation and progressive abdominal distension (Table II). Five of the twelve patients studied were in the age group of 40-49 years, a finding reported in other studies in the tropics where the peak incidence of sigmoid volvulus occurs in the 40-49 years age group.[8]

Symptoms of sigmoid volvulus often develop insidiously so that a patient may stay for two to three days before reporting to hospital.[4,18] In this study patients reported to hospital after one to five days of onset of the abdominal pain. However, four of the twelve patients were given local herbs orally 24-48 hours before reporting to hospital and the rest were delayed at either dispensary or district hospital level. A history of previous similar attacks may be obtained in as much as 50% of patients.[17] In this study three patients had similar attacks previously which subsided spontaneously.

The clinical features are mainly due to mechanical bowel obstruction including mild to moderate colic abdominal pains, progressive abdominal distension and absolute constipation. These symptoms occurred in all patients in this study (Table II). Diarrhoea may occur consisting of small amount of stool forced into the efferent limb of the sigmoid loop despite the obstruction.[2,4] Vomiting occurs in some patients usually as a late event.[10] Early and severe vomiting suggests a complicated volvulus associated with strangulation or ileosigmoid knotting.[10,12]

The clinical signs of sigmoid volvulus are mainly remarkable abdominal distension which may be so gross that the movement of the diaphragm is impaired.[20] There is little abdominal tenderness except with gangrenous bowel.[3]

There are two clinical types of sigmoid volvulus described by Hinshaw and Carter (1957),[15] which correspond with the degree of bowel obstruction and strangulation:

The first clinical type is acute fulminating sigmoid volvulus which is more common in younger patients and characterized by sudden onset and a rapid course. Severe back pain and vomiting occurs early and there is diffuse abdominal pain and tenderness with marked prostration. Early gangrene development is due to occlusion of mesenteric blood supply and this accounts for the fulminating course.

The second clinical type is subacute sigmoid volvulus which is more common than the acute fulminating type and

occurs usually in older patients. It has a gradual onset and often patients give a history of previous chronic constipation. Tremendous abdominal distension is usual and vomiting occurs late in the course of disease, probably due to the competency of ileocaecal sphincter mechanism. Gangrene develops more slowly and is a result of enormous rise in intra-colonic pressure in the closed loop. The general condition of the patient is usually good for the amount of distension of the abdomen.

Such a categorization was not used in this study. However, Sutcliffe[17] once suggested that suspicion of presence or absence of gangrene is more important in providing the basis for treatment rather than basing on this clinical categorization.

Diagnosis:

The diagnosis of sigmoid volvulus is clinical in more than 80% of patients. This has significant implication particularly in circumstances where plain abdominal x-ray examination cannot be readily obtained.[7]

Plain abdominal x-ray: This examination will demonstrate characteristic features of large bowel obstruction with gaseous shadow extending from the pelvis to the diaphragm, outlining the distended sigmoid loop in the form of an inverted 'U'. The term 'local inner tube sign' describes the outline formed by overlap of the sigmoid loop and two air fluid levels at the base of the sigmoid loops have been

likened to a 'pair of scales'.[3] The oedematous double walls of delated bowel are outlined by lines converging to the left iliac fossa where the mesocolon is twisted (Frimann Dahl's sign).[20] X-ray examination was done in all patients and eight patients had typical findings.

Barium enema: this is usually not recommended in acute sigmoid volvulus and is absolutely contraindicated if gangrene is suspected. When performed, barium enema examination demonstrates a characteristic 'birds beak' sign which shows barium filling the lower part of the rectum terminating at the point of the twisted sigmoid loop.[17,20] If barium enema x-ray examination is done in between attacks, it will show a redundant sigmoid loop or a large capacity sigmoid colon.[17] Barium examination was not done in this study because all the patients presented during acute attacks and managed as emergency cases.

Treatment:

The aim of treating acute sigmoid volvulus is initially to relieve acute colonic obstruction followed by measures to prevent recurrence such as operative fixation and resection of the sigmoid colon. Active management is required although spontaneous reduction of acute sigmoid volvulus has been reported.[16] Some surgeons recommend attempting reduction and evacuation by proctosigmoidoscopic rectal intubation or reduction by using barium enema as initial management of acute non strangulated volvulus.

Nonoperative reduction success has been reported in 70% to 90% of patients with complications in the range of 1-2%. Emergency surgery is often necessary for the treatment of acute sigmoid volvulus but it carries a 38% - 50% mortality rate for resection while simple operative detorsion and fixation has high rate of recurrence.[12,17,21] The prognosis after surgery depends on successful initial resuscitation measures, which include correction of fluid and electrolytes and sometimes blood transfusion in patients with gangrenous bowel. Prophylactic antibiotics should be given to all patients and since large bowel is likely to be opened metronidazole and Gentamycin is recommended.[12]

Spontaneous reduction and the use of enema:

Spontaneous untwisting of sigmoid volvulus has been known since ancient times. The Egyptians wrote in the papyrus Ebers that 'The natural history of sigmoid volvulus is either spontaneous reduction or rotting of the bowel'.[1] Bruusgaard (1947)[2] pointed out that sigmoid volvulus of less than 360° rotation may untwist spontaneously but when the twist is more than 360° the sigmoid loop is held fast between anterior and posterior abdominal walls and become less likely to untwist spontaneously. An ordinary or barium enema has been shown to cause reduction of a moderately twisted volvulus.[21] Shepard[12] reported 9 out of 425 patients with acute sigmoid volvulus who got spontaneous relief while awaiting operation. Spontaneous reduction of sigmoid volvulus is unpredictable and the results

of enema are often uncertain and temporary. Sigmoid volvulus should therefore be managed actively from the beginning.[22] In this study spontaneous reduction was not established and the use of enema was not performed.

Proctosigmoidoscopic/rectal tube reduction:

Bruusgaard (1947)[2] reported a 90% success rate in reducing and deflating acute sigmoid volvulus by rectal intubation with a reduced mortality rate of 14.2%. This technique has been adopted by other workers[16,21] some of whom pass a tube through the twist after visualizing it at sigmoidoscopy while others use the scope itself to open the spiral. Shepherd (1971)[21] extensively practiced the procedure of proctosigmoidoscopy with rectal intubation at Mulago hospital in Uganda and reported 88% success rate. Patients who are successfully decompressed non operatively should undergo elective sigmoid resection after proper bowel preparations and consideration of other medical problems often seen in these patients. Arnold and Nance[21] have shown that elective resection of the sigmoid colon with primary anastomosis reduces the operative mortality and eliminates the need for staged operation. According to Shepherd (1971),[16] this definitive treatment can be done as early as 7 to 12 days after the relief of acute attack.

Reduction by rectal intubation is contraindicated in patients with suspected gangrenous bowel. In those cases where sigmoidoscopic detorsion is considered, it is

recommended that the procedure be done in theatre with the patient well prepared for laparotomy should that need arise. Heavy sedation or anaesthesia should be avoided because pain during or after intubation is a valuable indicator of trauma or gangrene which calls for immediate exploration.[16]

Rectal intubation may be complicated by perforation of the colon and also the non-operative procedure carries the risk of missing a gangrenous volvulus. Even if deflation has been successful, gangrenous bowel may still be missed or may develop later after the procedure, hence close monitoring before and after the procedure is emphasized.[16] In case non operative reduction fails or there is evidence of gangrene it is mandatory to perform immediate exploratory laparotomy.

In this study proctosigmoidoscopic and rectal tube reduction were not performed, probably because most of the patients came late to hospital, which raised a possibility of gangrenous bowel.

Operative detorsion.

Some authors[15,17] recommend immediate exploratory laparotomy in every patient with suspected sigmoid volvulus because increased mortality has been associated with delay in active treatment. Surgical exploration in acute stage is indicated if rectal detorsion fails and in case gangrene of the bowel is suspected.[17] Intraoperatively if the sigmoid colon is viable, simple untwisting and deflation of the sigmoid loop has been recommended in patients who are in

surgically poor condition. Following untwisting, continued decompression is assured by passing a flatus tube up from the anus and left for 48 hours.[17] Resection of a redundant sigmoid colon can be done six to twelve weeks after the initial operation, under elective conditions.[15,17,22]

Operative detorsion provides absolute certainty of the diagnosis and offers minimal necessary surgical procedure suitable for life saving in high risk patients.[4,17] In practice however, the procedure is associated with high mortality rate probably because it is commonly performed in the high risk patients.[17] It has also been pointed out that after untwisting the bowel, distension may still persist causing respiratory embarrassment and loss of fluid and electrolyte into the distended bowel, accounting for the observed mortality rate.[17]

Simple detorsion of the bowel is associated with high rate of recurrence. Shepherd[12] reported a recurrence rate of 42% following operative detorsion and noted that this may be partly due to reluctance on the part of the surgeon and the patient alike to embark on a second major operation to resect the redundant loop after the first operation. In this study simple detorsion of the bowel as an isolated procedure was not performed.

Laparotomy and fixation.

Hall-craggs[23] described a plastic procedure in which the base of the sigmoid mesocolon is broadened in an attempt to

prevent recurrence after detorsion of sigmoid volvulus. The procedure involves division of mesosigmoid perpendicular to the long axis of the bowel and restitching it parallel to the long axis of the bowel. In this way the mesosigmoid is shorter and made broader. The mesocolic fibrous band which provide the axis in which the sigmoid colon rotates is excised. It was an attractive procedure as it provided a means of preventing recurrence without the risk of opening the bowel and also the patient did not require a second major operation. However, experience showed that the procedure did result in high recurrence rate of 41%, almost similar as for simple detorsion.[12] This procedure was not performed in this series.

Resection and primary anastomosis.

Sutcliffe[17] recommended resection of the sigmoid loop and primary anastomosis of the bowel for the treatment of acute sigmoid volvulus if the bowel is viable. This procedure effectively prevents postoperative abdominal distension and improves respiratory function. Fluid and potassium lost into the lumen as occurs after simple reduction is also prevented. It is reported[17] that, the success rate for this procedure is 82% with a mortality rate of 17.6% compared to the success for simple operative detorsion which is 75%.

Despite good results which have been reported by other workers,[9] the wisdom of performing resection and primary anastomosis on unprepared bowel is often thought to be

hazardous due to complications associated with breakdown of anastomosis.[24] The use of intraoperative colonic irrigation techniques has been shown by some workers[25] to reduce the risk of anastomotic complications in left colonic surgery. Some workers[24] still hesitate to perform immediate anastomosis after resection of acute sigmoid volvulus in unprepared bowel.

In this study, resection and primary anastomosis was performed in only one patient who had an acute sigmoid volvulus and intra-operatively had a viable and moderately distended bowel. He had an uneventful postoperative period.

Paul-Mickulicz operation.

Paul-Mickulicz procedure involves immediate resection of sigmoid colon and exteriorization of the proximal and distal ends of the bowel fashioning a double barrel colostomy. The technique is commonly recommended for gangrenous sigmoid volvulus. Bowel continuity is re-established about three months later as an elective procedure with proper bowel preparations.[26] Interference of blood supply to the colostomy may occur if the segments are held in tension and injury of the superior rectal vessels which run a looping course at the base of the volvulus can cause gangrene of the distal stump.[12] If gangrene extends down to the upper rectum, it may not be possible to obtain a long enough rectal stump for exteriorization in which a Hartmann's procedure should be done. Paul-Mickulicz technique allows subsequent closure of colostomy to be performed extra-peritoneally with minimal or

no peritoneal contamination. High mortality sometimes reported with this procedure[17] is probably associated with the underlying gangrene rather than the surgical technique. In this study nine of the twelve patients underwent resection and double burrel colostomy as in Paul-Mickulicz procedure. There was one postoperative death which occurred in a patient who had gangrenous bowel.

Hartmann's procedure.

In this procedure, the redundant sigmoid loop is resected the same as in the Paul-Mickulicz procedure. And the proximal end of the bowel is exteriorized fashioning a terminal colostomy at the left iliac fossa. The distal segment is closed and left in the pelvic cavity or exteriorized separately as a mucous fistula. Bowel continuity is re-established after 3 months in a repeat laparotomy.[26]

Hartmann's procedure is recommended in extensive gangrene involving the upper rectum and when there is technical difficulties in mobilizing enough distal segment for exteriorization. The technique is relatively short hence, is suitable for high risk patients.[17,23] Subsequent procedure to close the colostomy involves a laparotomy which is a disadvantage because it increases the risk of peritoneal contamination. In this study, Hartmann's procedure was performed in two patients all of whom had extensive gangrenous bowel. There was one operative death which occurred in a patient who had gangrenous bowel.

CONCLUSION AND RECOMMENDATIONS:

Sigmoid volvulus is not rare in Tanzania as reported in literature. Male preponderance was evident in this study as reported in other studies elsewhere. The prognosis of sigmoid volvulus postoperatively is good but it becomes poor when patients present in shock or are found to have gangrenous bowel.

Proper resuscitation and the use of antibiotics pre-operatively is the recommended management of acute sigmoid volvulus. The specific procedures depend on operative findings. In fit patients, resection and primary anastomosis is to be encouraged in order to avoid expense in the care of a colostomy, its complications and the need for a second operation.

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TESTICULAR TORSION

SUMMARY:

Ten patients with testicular torsion admitted to the three surgical firms at Muhimbili Medical Centre between July, 1992 and July, 1993 were studied. The age of the patients ranged between 15 and 33 years. Most of the patients were under 25 years of age. Seven of the ten patients studied had testicular torsion on the right. Most of the patients had history of sudden onset of pain localized to the scrotum followed by swelling of the testis in question. Two patients had intermittent scrotal pain with previous episodes of the same complaints. Of the two patients one had similar attacks affecting the opposite testis leading to its gradual disappearance. Most of the patients reported to hospital more than 24 hours after the onset of their problem. However, exploration of the scrotum was done in all cases. In only one patient orchidopexy was the treatment of choice for the affected testis and nothing was done to the other side as there was no palpable testis within the scrotum. For the rest of the patients, orchiectomy of the gangrenous testis and orchidopexy of the opposite testis was the treatment offered. Sepsis of the scrotal incision site was the commonest postoperative complication and only one patient developed a scrotal haematoma postoperatively. One case is presented in detail followed by a detailed discussion on testicular torsion.

INTRODUCTION:

Torsion of the testis, or more correctly termed torsion of the spermatic cord, was first reported by Delasiauve in 1940[1], affecting an undescended testis. The first British case, 4th overall, was recorded in 1881 by Langton[2] at St. Bartholomew's Hospital, London, this was also the first report of torsion affecting a normally descended testis. The torsion could not be released and the organ ultimately sloughed.

Torsion of the testis can occur at any age, however, William and colleagues[3] showed that it is commonest around puberty. Testicular torsion has not been studied in Tanzania, however, the age group at risk, presenting features, operative findings and the outcome of testicular torsion as compared with other studies[4-6] were the factors considered in this study. Non-viable testes should be removed to prevent subsequent extrusion through the wound or possible auto-immunization.[3,4] Beyond 10 hours of torsion greater than 50% of testes atrophy.[7] Immediate exploration and fixation of the opposite testes is mandatory. The entire management of torsion is challenged by the recent discovery that at least half of the patients with unilateral torsion are subfertile on subsequent testing.[7,8]

CASE REPORT:

N.S. IP No. 751073, a 33 year old male patient reported to hospital with complaints of sudden onset of intermittent right scrotal pain which started about 16 hours prior to

admission. The pain was radiating upwards along the groin and was not associated with nausea, vomiting, urethral discharge, dysuria and inguinal swelling. The point which made him report to hospital is that, he had a similar attack a year back involving the left side. He never went to hospital at that time and later on he noted gradual decrease in size of the left testis until he could not feel it in the scrotum anymore.

He was single and no other family member with a similar experience. Clinically he was young, not in obvious pain. The right testis was palpable, measured 3x2cm, was not tender to touch between the attacks but was tender during the attacks. The epididymis was normal in size. The left testis was not palpable in the scrotum. There were no inguinal hernias. The rest of his systems were essentially normal.

From the history and clinical findings a provisional diagnosis of torsion and spontaneous detorsion of the right testis was made. The patient was therefore planned for surgical exploration of the right testis. Preoperatively, the patient was advised not to take anything orally, his haemoglobin was established, his written consent was obtained and he was educated about the condition especially the possible complications. No preoperative antibiotics were given to the patient. An intravenous drip of normal saline was set to keep an intravenous line open.

In the operating theatre, the operation site was cleaned with iodine and spirit, draped with sterile towels. Local anaesthesia was infiltrated into the spermatic cord and along the scrotal incision site. A longitudinal scrotal incision was made. The tunica vaginalis was also opened longitudinally. The testis was found to be viable. There were frequent twisting and untwisting of the testis observed intraoperatively. In view of that observation the operative diagnosis was intermittent torsion and detorsion of the right testis. The decision was to do orchidopexy of the testis in question.

The tunica vaginalis was sutured. Orchidopexy was done by fixing the tunica albuginea to the lateral scrotal wall, using three silk No. 2/0 sutures. No drain was left in situ. Thereafter the scrotum was closed in two layers using cat gut No. 2/0 to the inner layer and silk No. 3/0 to the outer layer. Post operatively a gauze scrotal support was applied and pethidine injection (100mg) 6 hourly was given intramuscularly only when required, for the first 24 hours. No antibiotic was prescribed. He was discharged from the ward after changing the initial dressing 24 hours later. He faired well and scrotal sutures were removed on the seventh day postoperatively.

The patient was being seen at the outpatient clinic monthly for the first two visits. His testis appeared normal in size and the induration was subsiding. He never had any

other attack during that period. The patient was discharged from the outpatient clinic with the advice that he should report to hospital immediately whenever he experienced such scrotal attacks for early evaluation and treatment of his testis.

TABULAR SUMMARY:

Table I: Testicular torsion according to age groups.

Age groups (Years)	Number
15 - 19	3
20 - 24	4
25 - 29	1
30 or more	2
Total	10

Table II: Presenting features.

Symptoms/Signs	Number
Sudden scrotal pain	8
Testicular swelling	8
Transversely positioned testis	4
Vomiting	2
Intermittent scrotal pain	2
History of trauma	0

DISCUSSION

Aetiology:

Sir Astley Cooper[9] first noted that the entire scrotal contents of the neonate could be freely lifted out of the scrotum without tearing any tissue, and this mechanism is believed to account for the rare extravaginal torsion which is

virtually confined to new born infants.[10] Both Scudder[11] and Keith[12] drew attention to the fundamental anatomical abnormality predisposing to intravaginal torsion, namely a freely mobile testis suspended by a long mesorchium within a capacious tunica vaginalis. The high investment of the tunica vaginalis producing this 'bell-clapper' testis has been repeatedly described, and Barker and Raper[13] suggested that a normal testis will not undergo torsion unless submitted to severe trauma. Where the operative records in the present series included an adequate anatomical description, these anomalies were almost universally present.[3] It is surprising, in view of the fact that the anomaly is nearly always present on both sides,[14] that bilateral torsion is not a commoner event.

The actual force initiating torsion in a susceptible testis is still not clearly understood. Possibilities include vigorous exercise, direct trauma, a powerful cremasteric reflex and conceivably sexual excitement (when torsion develops during sleep).[3,4] Cold weather could explain seasonal variations in incidence within the British Isles[5,6] and geographical variations within the Indian subcontinent.[5] Cold can contract the dartos muscle by direct stimulation or by local reflexes. The capsule of the testis is usually regarded as being composed of inert and fibrous material, but experimental work has shown that it contains contractile tissue that responds to cholinergic and adrenergic stimuli.

Similarly, capsular contractility may be observed with low temperature.[5]

Pathophysiology:

Twisting of the spermatic cord may cut off the venous drainage and the arterial supply to the testis in question. Usually, the thick-walled arteries remain patent so that intense vascular engorgement and venous infarction follow.[15]

The outcome depends on the duration and severity of the process.[3] The morphologic changes may range from those of merely intense congestion to widespread extravasation of blood into the interstitial tissue of the testis and epididymis. In more extreme instances, haemorrhagic or even anaemic infarction of the entire testis may occur. In late stages, the testis is markedly enlarged and is converted virtually into a sac of soft, necrotic, haemorrhagic tissue.[15]

The leukocytic reaction is very variable and depends on the free access of blood via the arterial system. Usually the blood flow is so impaired that leukocytic infiltration is not a prominent feature and the process essentially resembles one of pure coagulation necrosis without considerable inflammatory reaction.

Reviewing the literature in 1964, Barker and Raper[13] discovered a 90% testicular loss after torsion - 80% by immediate orchiectomy and 10% by subsequent atrophy. In man, when the duration of torsion exceeds 4 hours some degree of testicular atrophy is almost inevitable, and beyond 12 hours

the organ will shrink to less than $^1/_2$ of its normal size.[16]

Reduced fertility following torsion of the spermatic cord was described by Krarup in 1978.[17] The finding was later confirmed by several authors, both in animal experiments and in clinical follow up studies.[18,19] In 1984 Williamson[4] introduced the term "sympathetic orchidopathia" to describe the condition and compared it with sympathetic ophthalmia. Hormonally mediated contralateral damage has also been suggested[8] and more recently, impaired spermatogenesis has been found in biopsies taken at the time of operation from the contralateral testis suggesting an underlying congenital defect. However, several hypotheses have been put forward to explain reduced fertility after unilateral torsion. Both testes may be congenitally abnormal and the bell clapper anomaly might be associated with some defect in spermatogenesis.[36] This hypothesis is supported by biopsies from the contralateral testis taken at the time of operation. Anderson et al (1986)[20] found impaired spermiogenesis, partial maturation arrest and Sertoli-cell-only syndrome in 67 of the 113 biopsies. The theory is somewhat contradicted by some authors who found no significant differences when they compared biopsies from normal testis with unaffected, contralateral testis from patients with a duration of symptoms less than 5 hours, while changes occurred in the contralateral testis in patients with a longer duration of symptoms.[18,21] Both testes may be damaged by recurrent

subtorsions, since the bell-clapper phenomenon is usually bilateral. More than a third of the patients report of previous attacks of pain, but these are usually from the side that later undergoes torsion.[3] The prophylactic fixation of the opposite testis could be injurious, but Krarup (1978)[17] found no differences in semen analysis in patients with or without contralateral fixation.

The finding of antisperm autoantibodies in animal experiments and the reported beneficial effect of immunosuppressive treatment led to the theory that immunological factors may play a role in the reduction of fertility.[22] However, some authors have concluded that the poor sperm counts observed following unilateral testicular torsion seem to owe more to an associated underlying defect in spermatogenesis than to sympathetic orchidopathia.[18,20]

Clinical features:

The sudden onset of scrotal pain and swelling in an adolescent nearly always indicates testicular torsion.[3] Occasionally pain radiates to the abdomen, perineum and the loins. Other highly suggestive features are vomiting (present in 35 - 50% of cases),[3] a past history of similar attacks (37 - 47%)[3] and a horizontal lie (34%),[3] best detected in the opposite testis when the patient is examined standing. History of recurrent attacks of scrotal pain is not uncommon.[3] Confusion can arise when the pain is mild, gradual in onset or ectopic (even leading to inappropriate appendicectomy).[3] Mild

urinary symptoms accompany 5-7 percent of testicular torsions,[3] scrotal erythema and pyrexia may develop after a few hours.[3] Important differential diagnosis before puberty are torsion of a testicular appendage (best explored in any case), strangulated inguinal hernia (especially with undescended testis), acute hydrocoele, idiopathic scrotal oedema, acute epididymitis, haematocoele, tumour and viral orchitis.[3]

Prehn (1934)[23] suggested that scrotal elevation relieved the pain of orchitis but aggravated that of torsion. Most authors, however, have found this sign notoriously fallible, and a much more reliable guide is the presence or absence of urinary infection.[24] Epididymo-orchitis is very uncommon in adolescent boys in the absence of underlying urinary tract infection and is usually a manifestation of more generalized urogenital infection.[25]

In the ten patients studied the presenting features were; scrotal pain of sudden onset (8), vomiting (2), intermittent scrotal pain (2), testicular swelling (8) and physical examination revealed transversely positioned testes in (4) patients (Table II).

Diagnosis:

The overall accuracy of the preoperative hospital diagnosis for torsion of the testis was found to be 90% in this study, diagnosis was made from history and physical examination. Confirmation was made according to the operative

findings. Four methods can be used to supplement clinical diagnosis.

A computer aided diagnosis: The development and evaluation of the computer program requires four steps,[26] construction of data base by using previous literature, application of Baye's theorem (this provides a method of combining probabilities for various indicants' i.e. clinical signs, symptoms or tests, to give an overall estimate of the probability of a specific event occurring), clinical details of the parameters described and analysis by using a computer which prints out the percentage probability for each of the three conditions of epididymitis, torsion of the testis and torsion of an appendage. According to Gaulbourne[26] and colleagues a computer provided the correct diagnosis retrospectively in 89% of 236 patients with torsion. Surgical registrars were even more successful (92%), albeit at the cost of overdiagnosing torsion and exploring 37 cases of acute epididymitis. The computer was very good at predicting epididymitis (98% accurate) and very poor at predicting torsion of an appendage (19%). The technique is inexpensive, rapid, immediately available, non-invasive and easy to use. However this technique is not used at Muhimbili Medical Centre.

Aspiration: Needle aspiration of a bloody secondary hydrocoele favours a diagnosis of torsion,[23] but the technique is seldom practised.

Doppler ultrasonography: This offers a simple method for determining testicular blood flow; unfortunately the examination is both uncomfortable and rather insensitive in acute torsion,[16,27] The precise technique of the Doppler examination is of critical importance if misdiagnosis is to be avoided. Most if not all, of the reported cases of misdiagnosis can probably be attributed to faulty technique. Testicular torsion can be diagnosed incorrectly as epididymitis if a pulsatile signal, originating from either the inflamed scrotal skin or the testicular artery proximal to the torsion, is mistakenly interpreted as arising from the testis. These errors can be avoided by applying a funicular compression test and by pointing the transducer towards the lower pole of the testis.[27] Doppler examination is rapid, simple and easy to learn.

Dynamic scrotal scintiscanning: This reliably and rapidly differentiates testicular torsion from epididymitis.[27] Acute torsion, late torsion and resolved torsion give different patterns of isotope uptake, but the method is useless in detecting torsion of an appendage. A testicular torsion shows the 'halo' sign characterized by a central 'cold' spot with a 'hot' perimeter of markedly increased uptake of isotope. however, some tumours demonstrate a halo sign, with central necrosis and a peripheral increase in blood supply hence can be confused with late torsion.[16] This technique is more accurate than Doppler examination.

Treatment:

Torsion of the spermatic cord is an acute urological emergency and delay in resolution jeopardizes the viability of the testes.[19] Experimentally and clinically, testicular survival has been correlated with the interval from onset of symptoms to surgical detorsion.[3] Most testes are salvaged if surgical relief occurs within 6 hours; conversely, progressively more testes are lost as the period of ischaemia lengthens.[3,19]

The fastest way for a physician to alleviate the testicular ischaemia is by manipulative detorsion at the initial physical examination.[28] The first case of manipulative reduction of torsion was reported by Nash[29] in 1893, though the testis subsequently underwent partial atrophy. Two years later Van de Poel (1895)[30] described the case of a 25-year-old doctor with recurrent torsion who learnt to reduce his own testis. Sparks in 1971[6] underlined the value of manipulative reduction as a first aid measure in his practice as medical officer at Rugby School. He showed that the direction of torsion is usually away from the Septum dartos, possibly owing to the line of pull of the cremasteric muscle fibres. Cattolica[28] found 7 of 8 patients with torsion occurring in a medial direction. The findings indicate that the direction of torsion can be medial or lateral.[28]

The technique of manipulative reduction of torsion of the testis is as follows; the operator sits at the foot of the bed

facing a comfortable, warm supine patient. If pain is intolerable an intravenous injection of Pethidine 75mg may be advisable, but this is usually unnecessary. The affected testis is gently manipulated until the patient feels relief. The correct direction of manipulative rotation to relieve torsion has to be found in every case by experiment.[6] Cattolica[28] recommended that the initial direction of detorsion manoeuvres should be lateral. Correct rotation brings immediate relief, incorrect rotation increases the pain, therefore rotation should be continued until the patient is free of discomfort.[6] One to three turns usually are sufficient to relieve the torsion.[6,8] Return of the anatomy to normal as judged by physical examination is required in addition to relief of pain before detorsion is considered complete.[28]

Surgical exploration within six hours of manipulative reduction is advisable to confirm the efficacy of the manoeuvre, and to carry out bilateral orchidopexy, and so to prevent further episodes of torsion.[6] Many testes can be salvaged if manipulation is done within 24 hours.[28] Frazier and Bucy[31] reported testicular salvage in four of the four testes and Sparks[6] in 73 percent. Of the ten patients in this study none of them underwent preoperative manual detorsion of the spermatic cord.

Although manipulative reduction can be attempted, operative detorsion remains the treatment modality of

choice.[3,20] The technique is as follows; under either general anaesthesia or local anaesthesia, the scrotum is explored through a low transverse incision and the tunica vaginalis is opened widely in the transverse axis. Detorsion is performed after extruding the testis through the tunica, which is then reflected upwards and retained at the root of the spermatic cord. If the viability of the testis is in doubt at operation after the torsion has been reduced, the organ should be wrapped in a warm moist pack and reinspected after 5 to 10 minutes. Bright bleeding following incision of the tunica albuginea is a promising sign.[3] It is obviously wise to retain any testis in which there is a possibility of residual function, particularly if the period of torsion has been less than eight to ten hours and the cord has not been twisted more than $1^1/_2$ times. If the testis is infarcted, orchiectomy is done to prevent subsequent extrusion through the wound[3] or possible auto-immunization.[4] The scrotum is then closed leaving a drain in situ. If the testis is salvageable, orchidopexy is then done, fixing the tunica albuginea to the lateral scrotal wall, with no intervening tunica vaginalis, using three non-absorbable sutures. The tunica vaginalis should be everted, plicated or excised. Contralateral orchidopexy is then carried out and the scrotum closed without drainage. Nine out of the ten patients in this study underwent orchiectomy of the affected side and orchidopexy of the contralateral testis. Eight out of the ten patients

reported more than 24 hours after the onset of the symptoms.

Post operative complications were found in this study, such as scrotal haematoma (1) and infection of the scrotal incision site (5). Presence of auto-antibodies, and fertility determination were not included in this study. Patients followed up for the first three months never showed testicular atrophy of the remaining testis. Reduced fertility following torsion of the spermatic cord was described by Krarup[17] and later confirmed by other authors.[7,18,21] Testicular atrophy is another complication following testicular torsion.[6]

CONCLUSION/RECOMMENDATIONS:

Torsion of the testis is not an uncommon condition. It can occur at any age with a high peak at adolescence. The condition is a real surgical emergency requiring proper clinical diagnosis and urgent relief of the torsion. It has to be differentiated from some other causes of an acute scrotum such as acute epididymitis, orchitis, torsion of a testicular appendage etc. Some other diagnostic measures can be used to supplement clinical diagnosis such as a computer program, needle aspiration, Doppler ultrasonography and a dynamic scrotal scintiscanning. These should not delay an urgent operative exploration which answers most of the questions. Since most of the patients report late, leading to loss of their testes, the public should be educated about the importance of early reporting to hospital for urgent treatment

and that the medical personnel who see these patients should think of testicular torsion as the first differential diagnosis unless proved otherwise. In some centres where an urgent operative exploration cannot be performed, a manipulative detorsion should be attempted especially for those who report less than 24 hours after the onset of the symptoms and where the scrotum is not adherent to the testis, taking all of the precautions not to cause more harm than good.

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PRIAPISM IN ADULTS

SUMMARY:

A total of three patients with priapism were admitted to Muhimbili Medical Centre in adult surgical wards, between July 1992 and July, 1993. Their ages ranged between 35 and 58 years. All patients presented to hospital more than 24 hours after the onset of priapism. All patients presented with persistent erection which was painful and physical examination confirmed priapism. Surgery was the treatment modality of choice in all three patients. One of the patients developed gangrene of the penis, later developed septicaemia and finally died. He is the one where caverno-saphenous anastomosis was done. Penile pain was another complication encountered, which occurred in one of the patients who underwent caverno-spongiosum shunt by using a Tru-cut needle. One of the three patients is presented and literature on priapism is discussed thereafter.

INTRODUCTION:

In Greek mythology Priapus was the god of horticulture, viniculture, apiculture and stock farming, and in general the god of fertility. His image was characterised by an enormous phallus!. Priapus lent his name to denote a medical condition-priapism-characterised by a persistent erection that is accompanied by pain and tenderness and fails to subside,

even after climax. This type of erection may last days, weeks or months.[2]

Priapism frequently occurs in the 30-40 age group and is therefore an uncommon occurrence in pre-adolescent males.[2] According to an american study, the number of patients aged under 16 years with priapism represented 3.6% Of total admissions.[3] It is reported to be more common in East african than in Europe.[4]

Priapism is classified as primary (idiopathic) or secondary. Several causes of secondary priapism have been reported such as haematologic, neurogenic, trauma, inflammations and infections, chemicals, drugs and toxins, carcinoma and congenital diseases.[5]

A wide range of surgical treatment has been attempted for priapism, including proximal ligation of the internal pudendal artery, anastomosis of the corpus to the saphenous vein, cavernous spongiosum shunt, cavernosa dorsal vein shunt, incision and irrigation of the corpora cavernosa, percutaneous aspiration, division of the pudendal nerves and ischiocavernosus muscles, amputation of the penis if associated with malignant disease of the genitalia and so forth.[6]

This study was done in order to have a look into the age distribution, causes, presenting features, management and the complications of priapism as seen at Muhimbili Medical Centre compared to findings at other centres.

CASE REPORT:

H.K., a peasant, aged 35 years, was referred to Muhimbili Medical Centre with an extremely painful erection which had persisted for three days. He woke up at night with that problem which he reported to be his first experience. He denied history of taking any medication, trauma or sexual intercourse prior to onset of his problem. There was no history suggestive of sickle cell disease in his family. He had a wife and three children.

Clinically, he was anxious, afebrile and was not pale. He had a pulse rate of 100/minute, regular, of full volume and a blood pressure of 130/70 mmHg. His respiratory system abdomen and digital rectal examination were normal. Local examination revealed an erect oedematous penis. Both corpora cavernosa were tense. The glans penis, which is the terminal part of the corpus spongiosum was pliable. From the history and examination a diagnosis of priapism was made, thereafter the patient was put on Valium and Panadol tablets. Blood specimen was taken for haemoglobin checking, sickling test, haemoglobin electrophoresis and leucocyte count (total and differential). Sickling test was negative and haemoglobin electrophoresis was AA. His haemoglobin level was 14.0 gm/dl. The leucocyte count; total and differential were reported as normal. No improvement was observed on subsequent examinations on the day of admission thus was scheduled for cavernospongiousum shunting in theatre as an emergency. Blood

for grouping and cross-matching was taken.

In the theatre, under general anaesthesia and sterile conditions a Tru-cut biopsy needle was used to create one shunt between the flaccid glans and the distended corpora cavernosa on either side of the urethra. Care was taken not to damage the urethra when introducing the Tru-cut biopsy needle by avoiding directing it more medially and obliquely. Blood was squeezed out manually until the corpora cavernosa softened. Normal saline was used to irrigate the corpora cavernosa. No heparin was available for irrigation.

Post operatively, gauze bandaging was applied loosely around the penis to allow blood to drain freely. Squeezing out blood and change of dressings were being done twice a day. Subsequent haemoglobin readings remained within normal range. The penis was softening gradually and on the seventh day he was allowed off to stay with relatives nearby. Dressing of the wounds was being done on outpatient basis. He turned up for follow up two weeks later, on review was found to be in good general condition and the wounds were healing. Two weeks later he requested to go home to solve some social problems and promised to be back after two months but never did so. The patient never reported any experience of penile erection before he went home.

TABULAR SUMMARY:

Table I: Age distribution.

Age group (years)	Number
30 or less	0
31 - 40	1
41 - 50	1
51 - 60	1
TOTAL	3

Table II: Presenting features.

SYMPTOMS/SIGNS	Number
Persistent erection	3
Painful penis	3
Oedematous and tender penis	3

Table III: Treatment modality.

Treatment	Number
Carveno-spongiosum shunt	2
Caverno-saphenous anastomosis	1

Table IV: Postoperative complications.

	Number
Gangrene of the penis	1
Septicaemia	1
Persistent postoperative penile pain	1

DISCUSSION:

Aetiology:

Inflammation and irritation of the urethra, prostatitis and mumps orchitis are documented causes of priapism.

Urethral instrumentation and prostatic massage have been mentioned as initiators of priapism. Testicular inflammation may lead to thrombosis of the pelvic and prostatic plexuses, thereby resulting in congestion and tumescence. Some cases of priapism have been noted in association with phlebitis.[7]

Overdosage with certain drugs, notably methyltestosterone and testosterone propionate cause priapism.[8] Oral administration of Cantharis[9] and anticoagulant therapy with heparin and coumarin is also cited.[10,11] Other cases of recurrent nocturnal priapism are of unknown pathogenesis, but humoral activity may be responsible.[12]

Haematological disorders such as leukaemia, thalassemia and sickle cell anaemia have been known to cause priapism. Many cases of priapism have been recorded in leukaemia patients, either from haemorrhage into the cavernous tissue,[13] or as a result of actual mechanical obstruction of the blood vessels of the corpora cavernosa and dorsal veins of the penis with leukaemic cellular debris.[10] Sickle cell anaemia is one of many hereditary haemorrhagic anaemias, characterized by an abnormal type of haemoglobin. Sickling of the erythrocytes occurs in the presence of low oxygen tension, low plasma pH concentration or high carbon dioxide tension. Priapism in sickle cell disease may result from vascular stasis following: normal physiological nocturnal erection, dehydration, relative metabolic acidosis associated with sleep, all of which may induce erythrocyte sickling within the corpora cavernosa.[14]

The commonest cause of priapism in Nigeria is sickle cell anaemia which affects 1.5% of her paediatric population. In these patients, priapism occur during vascular thrombotic crises.[15] Other factors reported to be the cause include carcinomatoses, neuro-psychiatric disorders,[16] stuttering spasmodic disease,[17] and idiosyncratic drug reactions.[5,16]

One of the largest series of priapism is a report of 48 patients treated during a 22-year period.[18] The study showed that idiopathic priapism and sickle cell disease accounted for 81% of all patients. There were 37 adults and 11 children. Idiopathic priapism occurred in 26 (70%) of the adults, and sickle cells disease occurred in 7 (63.3%) of the children. Other aetiologic factors in the adults were sickle cell disease (4), solid tumours (4), trauma (2) and leukaemia (1). Other aetiologic factors in the children were leukaemia (2) and idiopathic (2). In the three patients studied, no aetiological factor could be established, probably because the series was very small.

Pathophysiology:

Normal human penile erection depends on integration of afferent signals in the thoracolumbar or sacral centres, intact autonomic nerves to the penis, normal erectile tissue, and an adequate blood supply to the penis.[19] Pollsters(valve like structures) are present in vessels and control the flow of blood through the corpora cavernosa. The pollsters are controlled by parasympathetic fibres (S2-4) and sympathetic

fibres (thoracolumbar). The sacral erection centre is activated by either psychic or local reflexogenic stimuli, and the thoracolumbar erection centre is activated by psychic stimuli.

In sicklers, the haemoglobin S molecule has the peculiarity of being less soluble in deoxygenated blood, it undergoes crystallization. A sludging effect is then created in the small vessels by the climping action of the sickled erythrocytes. With the increase in blood viscosity and resultant stasis, mechanical occlusion of the vascular system occurs. Haemostasis is further complicated by perivascular oedema, which leads to pain and swelling of the involved organs due to tissue hypoxia.[13]

Hinman et al (1960), studied the histologic changes that occurred in seven patients with idiopathic priapism, demonstrating evidence of oedematous septae and subsequent fibrosis.[20] Electro-microscopic studies have shown unequivocally that ischaemic priapism in particular requires immediate treatment.[21] Otherwise endothelial lesions will occur after 12 hours, and a few hours later scattered necrotic smooth muscle cells may be seen in the trabecular network of the corpora cavernosa. After 24-48 hours the endothelium shows very severe damage, and diffuse necrosis develops. Multiple thrombi and damaged nerve endings are found. After a few days to weeks, the corpora becomes fibrotic and irreversible erectile impotence results.[10,20] All of the three

patients in this study reported to hospital more than 24 hours after the onset of priapism.

Clinical features and investigations:

Patients with priapism present in a state of having sustained penile erection, quite often painful, without any evident sexual desire or ejaculation. It may last days, weeks or months. In case of secondary priapism, patients may have features due to an underlying disease in question.[2,3] For example; fever, jaundice, anaemia and painful joints in sicklers.[2]

The state of the penis will depend on the duration of the condition. In prolonged cases gangrene and septicaemia might be the findings on admission. Acute urinary retention is one of the reported complications of priapism.[2] Local examination reveals a swollen, tender, erect and oedematous penis. Both corpora cavernosa are tense. The glans penis (the terminal part of the corpus spongiosum) is usually pliable.[2,22] All of the patients in this study presented with persistent painful erections. None of them had history of preceding sexual intercourse or drug intake (Table II).

A good history and physical examination are the two important points in reaching to a diagnosis of primary priapism. Supportive investigations are important to rule out secondary priapism.[2,5] The cause of secondary priapism influences the choice of treatment modality.[2] For example, surgical treatment is not helpful for the patient with

priapism due to direct infiltration with malignant disease.[5]

Sickling test and Hb-electrophoresis can be done to rule out the presence of sickle cell disease or trait.[2] White blood count (total and differential) might reveal the presence of leukaemia. Van Driel and colleagues reported a patient with priapism who was found to be having myeloid leukaemia. He was successfully treated with hydroxyurea.[22] Needle puncturing of both corpora cavernosa is done to obtain blood for determination of pH and blood gas values in order to establish the severity of the priapism or the degree of ischaemia.[10,22] Coagulation tests and hormone profile are also important investigations.[12,22] A cavernosogram is made in order to trace possible outflow obstruction due to metastases[16] or thrombosis.[15,22]

In the patients studied only the following investigations were done; sickling test, Hb-electrophoresis, leucocyte count and peripheral blood smear.

Treatment:

Priapism is a medical emergency. It is essential to administer first aid to achieve detumescence. Even though it sometimes subsides spontaneously, there may be danger of acute urinary retention and venous thrombosis in prolonged priapism. Fibrosis of the corpora cavernosa often follows with the development of chordee and complete impotence. Due to this effect, cold compresses (ice packs), sedation (diazepam, chlorpromazine), analgesics and catheterization should be

employed. Hot enemas have been reported to be useful. Amyl nitrate has been noted to cause relaxation during the early phase.[7] Other medical treatment include deep X-ray therapy, enzymatic lysis of thrombosed blood in the corpora cavernosa, local or systemic anticoagulant irrigation, ketamine, terbutaline, physostigmine, steroids, female hormones, use of hypotensive agents to reduce blood pressure, general and spinal anaesthesia, curare, application of a pneumatic cuff around the penis and more recently intracavernous injection of sympathomimetic drugs (metaraminol, dopamine, phenylephrine, noradrenaline or adrenaline).[6,22,23,24]

Lue et al (1988),[25] in a survey showed that several different sympathomimetic drugs were used to treat priapism.[25] The ideal drug has yet to be identified. In actual practice, adrenaline is freely available but its potential disadvantage is that it has both alpha-adrenergic and beta-adrenergic actions. If it enters the systemic circulation the beta-adrenergic effect many cause a marked increase in pulse rate and extrasystoles may occur. This is why a purely alpha-adrenergic sympathicomimetic drug such as phenylephrine is to be preferred on theoretical grounds.[23] Others advocate the use of adrenaline simply because of the beta-adrenergic properties, since beta-activity may have some added benefit by its action on the venous walls promoting outflow.[26]

The form of therapy should be in accordance with the suspected aetiology. In the case of sicklers, fresh blood

transfusions reduce the concentration of sickle cells in the blood-stream and inhibit the activity of the bone marrow. By raising the haemoglobin concentration, suppression of erythropoiesis will occur. The suppression of circulating sickle cells would reduce the tendency for clumping and subsequent occlusion of the venous channels. Thus normal venous drainage is returned to the penis.[3] Packed red cell transfusions have largely been successful because of the dilution of sickle cells with normal cells. In sicklers having high haematocrit levels, partial exchange transfusions are advocated: 20mls per kg of packed red cells is suggested in an attempt to double the red cells mass,[27] but care has to be taken to avoid iron overload.[2]

Some authors insist that anticoagulant therapy is necessary to minimize the risk of venous thrombosis. Intravenous administration of heparin, a loading dose of 100mg followed by 50mg four times in a day is suggested. Others, however, disagree because there is apparently no evidence to suggest clot or thrombus formation with conversion of fibrinogen into fibrin.[27] Thus, the use of fibrinolytic agents, thrombolysins, vasodilators, dextran and stilboestrol are controversial.[10]

Grayhack and associates,[28] initiated the surgical treatment of priapism with a cavernosaphenous shunt following unsuccessful non operative treatment. Hinman[20] studied the histologic changes that occurred in patients with idiopathic

priapism. He postulated that the basic reasons for failure of the various non surgical treatments are that they do not restore venous drainage and that they do not eliminate progressive damage to the local erectile mechanisms.

A wide range of surgical treatments has been attempted for priapism, including proximal ligation of the internal pudendal artery, anastomosis of the corpus to the saphenous vein, cavernous spongiosum shunt, cavernosa dorsal vein shunt, incision and irrigation of the corpora cavernosa percutaneous aspiration, division of the pudendal nerves and ischiocavernosus muscles.[6]

Waterhouse's and Winter's procedures are similar except that in Waterhouse's manoeuvre, a sharp knife is used to cut through the glans and the fascia covering the obstructed, bulging part of the corpora. In Winter's procedure a Tru-Cut biopsy needle is used to remove a circle of the fascia in the hope of keeping the communication open between the tense and distended corpora cavernosa and the flaccid glans and its adjacent corpus spongiosum.[4,29,30,31]

Winter (1978)[29] advised the creation of one or two such shunts on each side via one puncture in the tips of the glans penis after initial aspiration and irrigation of the corpora with heparinized saline using an 18-gauge needle. The needle was also inserted through the tip of the glans. The brisk bleeding, mainly of dark blood from the corpora cavernosa, which follows such biopsies can be controlled either by very

gentle pressure on the puncture site in the glans or by a fine catgut suture. There should be rapid detumescence of the corpora cavernosa, but the glans and corpus spongiosum may look fuller than normal because they are now draining blood from the corpora cavernosa. Catheterization should not be necessary. The procedure can be performed under local anaesthesia but a general anaesthesia is advisable, at least on the first occasion. It requires considerable force to push the biopsy needle through the fascia covering the corpora cavernosa and the effect required could prove distressing for both the surgeon and a conscious patient. This technique has further advantage that it avoids the risk of entering the urethra and causing a fistula which is a recognized complication of the caverno-spongiosum shunt. Furthermore, it preserves the saphenous vein.[4,29]

Complications of these open shunt operations occur in about 15% of patients.[5] Complications are; urethrocutaneous fistula, urethrocavernous fistula and a painful spongiosum on erection. Pulmonary embolism has also been reported to follow cavernospongiosum shunt. Following either procedure gangrene, or necrosis of the distal penis, penile skin necrosis, haematoma, wound infection or abscess, erectile deviation and wound sepsis may occur. Penile skin necrosis tends to occur following application of pressure dressing; therefore, a pressure dressing should never be used following any of the therapeutic procedures.[5]

Because Winter's method is so straightforward, it would seem reasonable to use it as the primary method of treating idiopathic priapism, preserving other shunt procedures for patients in whom sustained detumescence is not obtained.[5,29]

If Waterhouse's and Winter's procedures fail, the next step is corporocorporal anastomosis. It is reported to be a quick and easy operation. A short midline incision is made over the shaft of the penis at any convenient site. Dissection is done in the plane between the corpus spongiosum and the hard, distended corpus carvenosum. A generous window, about 1cm x 1cm, is cut away from the fascia covering the spongy tissue of each corpus. The sludge from the corpus cavernosum is irrigated using heparinized saline until bright 'arterial' blood appears and the erect penis is emptied out by firm pressure. The fascial edges of each window are anastomosed using very fine non-absorbable suture material, eg. 7/0 Prolene.[31]

CONCLUSION AND RECOMMENDATIONS:

Priapism is a medical emergency. A detailed history and thorough examination are required to rule out neurologic, haematologic, systemic or local mechanical causes. Full haematological investigations are required to rule out sickle cell disease, leukaemia and also to tell the severity of priapism and ischaemia. It is essential to administer first aid to achieve detumescence as early as possible to avoid

complications of prolonged ischaemia to the erectile penile tissue.

Since most of the patients report late after the onset of the disease, it is reasonable to educate the community about priapism. Those with predisposing diseases such as sickle cell anaemia and leukaemia should be informed about the possible occurrence of priapism during their lifetime, and that they should report to hospital as soon as possible.

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MANAGEMENT OF CARCINOMA OF THE PROSTATE AS SEEN AT MUHIMBILI MEDICAL CENTRE

SUMMARY:

Ten patients with carcinoma of the prostate admitted to one of the three surgical firms at Muhimbili Medical Centre between July, 1992 and July, 1993 were studied. Their ages ranged between 50 and 79 years. The majority of the patients were 60 years or older (Table I). The clinical features at presentation were mainly due to urinary outflow obstruction and pain. Seven out of ten patients had hard and nodular prostate on digital rectal examination (Table II). Histological diagnosis of prostate cancer was made from prostatic needle biopsy in seven patients while in three patients diagnosis was made incidentally from prostatectomy specimen removed for benign prostatic hypertrophy (BPH). Distant metastases were documented in four patients and three patients had localized disease (stage A). Three patients presented with locally advanced disease with haematuria but no distant metastases were documented and metastasis to regional lymph nodes was not evaluated. These patients were classified as stage C disease. Treatment given to the patients is summarized in table V. A detailed case report of one of the patients and a discussion on carcinoma of the prostate in general is presented.

INTRODUCTION:

Prostate cancer is the second commonest malignancy in men in Western Society.[1,2] In New South Wales, 1082 new cases were reported in 1982, representing a crude incidence of 41 new cases/100,000 males per year.[3] In the same year, 522 patients died of the disease.[3] Similarly in the United States, the annual incidence is 40-50 new cases/100,000 males,[1] although the figure varies depending upon racial group. Prostate cancer is reported to be commonest in men over the age of 50 years and the incidence increases with advancing age.[4] More than 10% of prostate cancer is discovered incidentally in simple prostatectomy specimens[5] while routine autopsy shows occult malignant foci in the prostate in 80% of men over the age of 70 years who die of other causes[6,7] and this is found uniformly in different races.[7]

In Africa prostate cancer is not uncommon. Davey[8] reported that in elderly men in Uganda carcinoma of the prostate was second most frequent malignancy surpassed only by epithelioma of the penis. Carcinoma of the prostate was the third most frequent registered malignancy (9.6%) at KCMC hospital in North Tanzania between 1971 - 1980.[9]

The techniques available to the urologist for the assessment of the prostate have recently increased with the development of the serum assay of prostate specific antigen (PSA), the wider availability of urodynamics and the introduction of newer imaging techniques. Transrectal

ultrasonography (TRUS), computed tomography (CT) and magnetic resonance imaging (MRI)[10] have now been available for over 10 years and continued development during this period has considerably improved the image quality.

In the early clinical stages, the natural history of carcinoma of the prostate is not well known," so even the treatment also differs to patient with incidental carcinoma.[12] In advanced stage curative therapy is not possible, hence radiotherapy and hormonal manipulation are for palliation.[13]

This study was done in order to look into the clinical features, diagnostic measures and treatment options compared to studies done elsewhere for patients with carcinoma of the prostate.

CASE PRESENTATION:

J.M. a 63 year old peasant, presented to hospital with history of failure to pass urine. Prior to that problem he had been having urgency, frequency and hesistance for one year. He never experienced backache, weight loss or haematuria. Clinically he was not pale, the blood pressure was 150/90 mmHg. His respiratory, cardiovascular, gastrointestinal and neurological systems were essentially normal. Digital rectal examination revealed normal anal verge with an intact sphincter, he had an enlarged grade III prostate which was firm in consistency with no hard areas or nodules. He had an indwelling urethral catheter which was introduced at the

casualty department to relieve the acute retention. Investigations done;

Haemoglobin = 12gm/dl

Serum acid phosphatase = 8iu/L

Serum creatinine = 120umol/L

Chest X-ray = normal

Plain abdominal x-ray (KUB) = normal

Cystoscopy = showed an elongated prostatic urethra, with a normal surface, the lobes were kissing and the median lobe was protruding into the bladder cavity. The trigone was hyperemic while the rest of the bladder was normal.

Routine stool analysis was normal. Urinalysis = normal. A clinical diagnosis of benign prostatic hypertrophy was made. Hence the patient was scheduled for open prostatectomy. He was crossmatched with 3 units of blood of his group. After all preoperative preparations he was taken to theatre where, under general anaesthesia a retropubic prostatectomy was done. He had a fairly good recovery post operatively after continuous bladder irrigation with normal saline for 24 hours. He received 2 units of blood transfusion due to intra and post operative loss of blood.

The whole prostate was sent for histological diagnosis and reported as a well differentiated adenocarcinoma of the prostate. The tumour was clinically staged as A1. It was then

recommended that he never required any additional therapy and be followed up at the urology clinic. He was seen on three monthly intervals for two visits. He was doing well, digital rectal examination done did not reveal any abnormality and the prostatic acid phosphatase was normal. He had no urine incontinence and was requested to come for follow up after a period of 6 months.

TABULAR SUMMARY

Table I: Ten patients with prostate cancer.

Age group	Number
50 - 59	2
60 - 69	4
70 - 79	4
80 - 89	0
Total	10

Table II: Clinical features.

Signs/Symptoms	Number
Frequency	10
Dysuria	10
Hard nodular prostate	9
Pelvic/backpain	6
Anaemia	6
Acute retension of urine	5
Haematuria	4
Wasting	3

Table III: Histological diagnosis.

Histology	Number
Well differentiated adenocarcinoma	4
Moderately differentiated adenocarcinoma.	2
Poorly differentiated adenocarcinoma.	4

Table IV: ~~Disease stage and treatment~~ metastatic Site.

Metastatic Site	Number
Lumbar spine	4
Pelvis	4
Ribs	1
Scapular	1
Femur	2
Pulmonary	1

Table V: Disease stage and treatement.

Disease Stage	No. of patients	Treatment given
A1	1	Retropubic Prostatectomy
A2	2	TURP and Radiotherapy
C	3	Subcapsular Orchiectomy (2) Diethy-Stilboestrol-3mg (1)
D	4	Subcapsular Orchiectomy (2) Diethyl-stilboestrol-3mg (1) Palliative-TURP (1)

*TRUP: Trans-Urethral-Resection of the Prostate

DISCUSSION:

Clinical features:

Only a third of patients with prostate cancer present with symptoms but at the time of diagnosis more than 80% have

stage C or D disease.[14] Symptoms caused by local disease include frequency of micturition and nocturia. Other symptoms are dysuria, urinary incontinence, difficulty in voiding and sometimes acute retention of urine may be the first presenting symptom. The presence of haematuria is an indication of locally advanced disease with involvement of the prostatic urethra. Backpain with or without associated weight loss suggests metastasis in an elderly male particularly with suspicious finding on digital rectal examination.[15]

The earliest palpable change of prostate cancer is a discrete nodule of firm or stony hard consistency detected on digital rectal examination. Such a nodule has the probability of being neoplastic in about 50% of patients [16,17] and it was the finding in seven out of the ten patients in this study. In more advanced stages the median sulcus may be obscured by malignant enlargement. Local extraprostatic extension of tumour into the seminal vesicles can also be detected by digital rectal examination.

Groin and supraclavicular nodes may be palpable but these are late events. Oedema of one or both legs and scrotum is a sign of infiltration of pelvic lymph nodes and is a manifestation of extensive disease. Paraplegia or spontaneous fracture are occasionally due to osseous metastasis and may be the first presenting feature. Wasting and anaemia are late features of prostate cancer. These were the features in the patients in this study (Tabale II).

Diagnosis:

Tissue biospy:

The diagnosis of carcinoma of the prostate is confirmed histologically from prostatic tissue biospy. The biopsy can be obtained by using a Tru-cut or Vim-Silvermann biopsy needle through trans-perineal or transrectal approach. Trans-perineal approach avoids the risk of bacterial contamination but carries a risk of injury to the pelvic nerves. A trans-rectal approach is more precise with 70 to 80% accuracy but haematuria and bacterial contamination are some of its complications.[14] A cleansing enema and prophylactic antibiotics prior to biopsy are recommended. The experience with trans-rectal Tru-cut needle biopsy at KCMC shows that the procedure is simple, cheap with few complications and has a diagnostic accuracy of 77%.[18] In nine of the patients in this study, the histological diagnosis was made from prostatic needle biopsy obtained by using a Tru-cut needle through the rectal wall. There were no complications apart from mild pains and brief rectal bleeding.

Needle aspiration cytology is one of diagnostic procedures which can give accurate diagnosis of prostate cancer and allows morphologic assessment of several areas of the prostate with low rate of complications.[19] Higher yield is obtained with the needle guided by a transrectal ultrasound probe. Prostatic chips from trans-urethral resection of the prostate can yield tissue suitable for diagnosis in more than

90% of patients with neoplasms who have significant obstructive symptoms. Open perineal biospy of the prostate is a relatively more invasive procedure and not commonly recommended.[20] Prostate cancer can be diagnosed incidentally from the histology of 10-20% of prostatectomy specimen performed for benign prostatic hypertrophy.[5] Three of the patients studied were diagnosed this way.

Imaging examination:

Transrectal ultrasonoggraphy (TRUS), computed tomography (CT) and magnetic resonance imaging (MRI) have now been available for over 10 years and continued development during this period had considerably improved the image quality.

TRUS of the prostate: The transrectal approach has been most widely used for the ultrasonic examination of the prostate. With modern high frequency transducers, excellent visualization of the internal anatomy of the prostate and seminal vesicles is possible. The examination is usually performed with the patient lying in the left lateral decubitus position with the knees bent. The gland should be scanned in the transverse plane from the base to the apex, noting its overall shape and symmetry, the 'capsular' integrity, and the internal echo pattern. The longitudinal plane provided better visualization of the base and the apex of the gland; it is thus the optimal imaging plane for needle insertion into the prostate and seminal vesicles for biopsy and aspiration.[10] Cancer usually appears ultrasonically as a hypoechoic area,

but both isoechoic and hyperechoic cancers are found. Prostatic cancer generally occurs in the peripheral zone but can also arise in the central and transition zones.[21] It is now accepted that prostatic cancer may be a multifocal process, with separate discrete foci in different areas of the gland. The diagnosis of a hypoechoic area must be established by biopsy as there are many causes of hypoechoic areas within the prostate, including prostatitis, glandular hyperplasia, granulomatous prostatitis and prostatic infarcts as well as prostatic cancer.[10] TRUS can be used to stage the extent of prostatic cancer by assessing the integrity of the capsule and the likelihood of seminal vesicle invasion.[10]

CT of the prostate: CT has been found to be of no value in diagnosing prostatic cancer confined to the gland but has a role in the staging of prostatic cancer, particularly if TRUS and MRI are not available.[10] In a comparative study of 27 patients with biopsy-proven carcinoma of the prostate examined with TRUS, CT and MRI; TRUS was found to be superior to CT and MRI for assessing capsular disruption and MRI was found to be superior to both TRUS and CT for evaluating seminal vesicle invasion. MRI was also slightly better than CT for detecting lymphadenopathy.[22]

MRI of the prostate: In recent years MRI has played an increasing role in the staging of prostatic cancer, particularly before radical surgery.[10]

None of the imaging techniques available, including TRUS,

CT and MRI, are able to identify microscopic disease; they can only detect macroscopic disease.[10]

Roentgenography and Radionuclide scan:

Although pulmonary metastasis occurs in 25% of patients dying of prostate cancer they are often unrecognized on routing chest x-rays because they are micro-nodular and diffuse lymphangitic in nature.[23] When visible, pulmonary metastasis from prostate cancer show multiple opacities resembling miliary tuberculosis. Skeletal metastases are characteristically osteoblastic in nature but mixed osteoblastic and osteolytic lesions occur. Approximately 50% bone must be replaced by tumour before the lesion can be detected on plain x-ray.[6]

Radionuclide bone scans using Technetium-99 is the most sensitive means of detecting metastasis to the bone. It reveals metastasis in 25% of patients with negative findings on routine bone films.[6] Metabolically active bone lesions such as ostemyelitis or traumatic fractures may give false positive results so any suspicious lesion on radionuclide scan should be verified by conventional x-rays or by biopsy.

Intravenous urography (IVU) in prostate cancer may show elevation of the bladder base or irregular distortion of the ureters by enlarged nodes.

Tumour markers:

Prostatic serum acid phosphatase (ACP): For many years, circulating levels of serum acid phosphatase have been

measured to assist in the diagnosis and treatment of prostate cancer.[24] The earliest assays for these proteins were crude and relatively inaccurate. The more recent demonstration of a prostatic isoenzyme of acid phosphatase and the consequent development of a radioimmunoassay for this enzyme have improved the sensitivity and specificity of this tumour marker.[24] ACP is elevated in 60-80% of patients with extracapsular or metastatic disease.[23] Serum levels may also be elevated in patients with localized disease, prostatitis, prostatic infarction, thromboembolic disease, osteosarcoma or pancreatic cancer,[23] thus making the test less specific. However, specificity can be improved by estimation of the prostatic fraction of serum ACP.

Temporary elevation of serum ACP may occur following digital prostatic massage, urinary tract instrumentation or prostatic needle biopsy.[6] The clinical use of ACP is mainly in the staging and monitoring of already diagnosed prostate cancer but not specific enough to be of use in screening for patients with localized disease.[23]

Prostatic specific antigen (PSA): This is a recent innovation which now has a major role in routine management. PSA has been demonstrated in prostatic tissue as a more specific tumour marker: PSA is a single chain glycoprotein with a molecular weight of 34,000, appears to be one of the kallikrein family. It is found in the cytoplasm of prostatic acinar cells and ductal epithelium, and is demonstrable in the normal prostate,

in benign prostatic hypertrophy and in prostatic cancer, but not in other normal or malignant tissues.[24] Elevated levels occur in 20 to 92% of patients with extracapsular prostate cancer depending on the size of the tumour and degree of differentiation. Serum levels are also elevated in 20-50% of patient with benign prostatic hypertrophy (BPH).[23] PSA is one of the most sensitive markers for monitoring progression and response to therapy but again it is not sensitive or specific enough to be used alone as a screening test for prostate cancer.[23]

Serum alkaline phosphatase (ALP): Is normally elevated in patients with prostate cancer who have hepatic involvement or secondaries in the bone. ALP is most useful in the monitoring of bone metastases from prostate cancer, and had only a very limited role in the monitoring of localized prostate involvement or soft tissue disease.

Lymphangiography.

Bipedal lymphangiography may be useful in detecting metastasis in the regional lymph nodes but being an invasive procedure, is used less frequently where CT scan is available. False negative results occur in about 15 to 30%[23] of cases particularly those with microscopic metastases and in lymph nodes that are totally replaced by tumour. Moreover bipedal lymphangiography does not routinely visualize the internal iliac and obturator nodes which are of primary importance in carcinoma of the prostate.[25]

Staging pelvic lymphadenectomy.

Pelvic lymphadenectomy is performed mainly to gain more prognostic information especially for patients who are being considered for radical prostatectomy. In patients who are to receive interstitial radiation therapy lymphadenectomy is an associated staging procedure usually performed at the time of placement of radioactive seeds. Lymphadenectomy as a therapeutic procedure has been found to offer little survival benefit in the treatment of prostate cancer particularly where no active therapy is planned or when the projected treatment is to be palliative.[6] This procedure was not performed in any of the patients in this study.

Clinical staging:

Whitmore's clinical staging system has received wide general usage in the treatment plans and prognostication of prostate cancer because of a direct relationship between the clinical stage and prognosis.

Stage A: Tumour confined to the prostate and not palpable on digital rectal examination.

A1: Local, (well differentiated tumour with very limited involvement)

A2: Diffuse (less differentiated large tumour volume).

Stage B: Tumour confined to the prostate but palpable on digital rectal examination.

B1: Involving less than one lobe

B2: Involving one lobe or more

Stage C: Palpable extra-prostatic tumour without metastasis

C1: Involving lateral sulci only

C2: Involving the base of seminal vesicles

C3: Other than C1, and C2

Stage D: Prostatic cancer with metastasis

D1: Metastasis to regional lymph nodes

D2: Metastasis to distant site.

Other classifications are as follows;

American Joint committee Classification.

Stage A (I) = clinical unrecognized prostatic cancer without evidence of metastases.

Stage B (II) = prostate cancer clinically confined within the prostatic capsule.

Stage C (III) = prostatic cancer with clinical evidence of local extension beyond the prostatic capsule but without clinical evidence of metastases.

Stage D (IV) = Any prostatic cancer with evidence of metastases.

TNM classification:

Tx = requirement for assessment of the extent of the primary tumour have not been met.

To = no tumour palpable.

T1 = intracapsular tumour surrounded by palpably normal prostate.

T2 = tumour confined to the gland but deforming the contour.

T3 = tumour extending beyond the capsule with or without involvement of the lateral sulci and or seminal vesicles.

Nx = requirement for assessment of the regional lymphnode have not been met.

No = no involvement of regional lymphnodes.

N1 = involvement of single regional lymphnodes.

N2 = involvement of multiple regional lymphnodes.

N3 = a fixed pelvic wall mass of regional nodes with a free space between the mass and the primary tumour.

N4 = involvement of juxtaregional nodes.

Mx = requirements for assessment of distant metastases have not been met.

Mo = no recognized distant metastases.

M1 = distant metastases present.

Treatment:

Treatment modalities for prostate cancer include surgical, radiotherapy, hormonal therapy and chemotherapy. Surgical removal of the prostate (radical prostatectomy) and radical radiotherapy are expected to offer long term survival if the disease is still confined to the prostate. In a more advanced disease treatment is aimed at palliation of symptoms.

Stage A and B disease.

The treatment of stage A1 prostate cancer may be controversial. This stage of disease is often cured definitively by the simple prostatectomy at which the diagnosis is made. Studies have shown that life expectancy of

patients with stage A1 disease who are older than 70 years and receive no treatment, is more than 85% of normal longevity for 10 years.[6] Same authors however recommend radical prostatectomy for younger patients.[23]

The recommended treatment for stage A2 disease is even more controversial. Byar et al[26] reported 5 and 10 years survival rate for treated stage A2 that was equivalent to that of age matched men without prostate cancer. However it has been shown that stage A2 tumours may take aggressive course with rapid and widespread metastases. For this reason some authors recommend definitive treatment for stage A2 disease.[6] Radical treatment by surgery or radiotherapy has its clearest indication in stage B disease.[14] Radical prostatectomy and radical (curative) radiotherapy offers similar results for stage A and B disease except that (surgical) radical prostatectomy carries a slight advantage in long term survival while radiotherapy avoids complications of surgery. Some workers therefore recommend radical prostatectomy for patients less than 60 years old who are surgically fit while radiotherapy is recommended for older patients, poor surgical risks and those who refuse surgery.[23]

In radical prostatectomy the entire prostate, the seminal vesicles and periprostatic tissues are removed and the bladder neck is anastomosed directly to the urethra. Kuchler in Germany performed radical perineal prostatectomy in 1866[27] and it was modified by Young in 1904. In 1945 Millins introduced

retropubic approach[28] which allowed access to the pelvic lymph nodes providing more accurate assessment of the tumour.[6,27] Culp and Meyer[28] reported a 10-21 years survival rates of 72% for stage A tumours with 57% of the patients showing no clinical evidence of disease. They also reported a 15-21 years survival rate of 52% with 48% of the patients free of cancer. Walsh reported a 15 years survival rate of 51% in carefully staged B disease patients.[27] Failure of cure in localized disease has been attributed to pre-existing metastases, local tumour extension beyond the limit of excision and tumour embolization occurring during operation. High failure rate is associated with poorly differentiated tumours.[16]

Radical prostatectomy is associated with urinary incontinence and erection dysfunction. More than 90% of patients end up with impotence as a result of injury to pelvic nerve plexus. Nerve sparing modification described by Walsh preserves sexual function in the majority of patients without compromising the adequacy of tumour excision.[27,29]

Radiotherapy:

Radiation therapy for prostate cancer was developed in a desire to avoid complications associated with surgery. Radiotherapy can be deliverd as an external beam irradiation suitable for more extensive lesions and this can be delivered by linear accelerator or Cobalt-60 units or as interstitial irradiation which involves the implantation of seeds of radioactive iodine(I^{125}) or radioactive gold (Au 165) around

the tumour suitable for stage B1 tumours in a well defined lesion of diameter less than 5cm.[30,31] Staging lymphadenectomy is recommended before external beam radiation or during surgical placement of radioactive seed in order to map out the radiation field and dose. Ray et al[30] reported 5 and 10 years survival rate of 72% and 48% respectively for stage B tumours treated by external beam irradition. Sexual potency was preserved in 70% of patients. He also reported 10 years survival rate of 41% in patients with advanced stage B and C after similar treatment. It should be noted that patients selected for radiotherapy are usually those who would not be suitable for radical surgery so these results are therefore comparable or even more superior to surigal prostatectomy and carry fewer complications.

Acute proctitis, tenesmus, acute urethritis and diarrhoea are common side effects of radiotherapy to the prostate but they generally disappear within a month after treatment. Chronic lymphoedema, chronic proctitis, rectal stricture and rectal fistula may occur after full course of external beam radiation.[6]

In the patients studied 3 had stage A disease discovered from prostatectomy specimen performed for benign prostatic hypertrophy. Evaluation showed the disease was localized to the prostate and initial surgery had controlled urinary obstruction symptoms. One patient was 72 years of age with well differentiated tumour (stage A1), no further treatment

was given. The other two patients who had poorly differentiated tumours (stage A2), were treated by radiation therapy with Tele-cobalt machine. They completed treatment course of 4500 rad over five weeks. No major radiotherapy complications were encountered.

Stage C disease:

Treatment options for patients with stage C disease (extra-prostatic tumour extension with no evidence of metastasis) are either no curative therapy, hormonal therapy or whole pelvis irradiation. The tumours are usually biologically aggressive responding poorly to treatment. Studies show that definitive treatment does not alter the rate of development of distant metastasis or survival rate,[6] so treatment for stage C disease is basically palliative. Treatment is withheld until the disease metastasizes and hormonal therapy is initiated for controlling painful symptoms.[6] Urinary outflow obstruction usually respond well to hormonal therapy otherwise, transurethral resection of the prostate should be done.[9] Radical prostatectomy is sometimes contemplated for stage C disease to control morbidity due to local pelvic symptoms, bladder outlet obstruction, haematuria and ureteral obstruction.[14]

Hormonal therapy.

The androgen dependence of adenocarcinoma of the prostate was established over 50 years ago by Huggins and Hodges (1941) by studying the effects of castration, oestrogens and

androgens on disease progression.[32,33] Orchidectomy and Oral Oestrogens became the standard treatments for advanced prostate cancer, although the use of oestrogens subsequently fell into disrepute because of the high associated cardiovascular morbidity and mortality.[26] As a result, bilateral orchidectomy had become the gold standard against which all other therapies must be judged. It produces a favourable response in 70-80% cases for a median duration of 50 weeks.[34] There is considerable debate regarding the use of total androgen blockade and a number of newly developed pharmaceutical preparations are being evaluated in Europe and the USA.[35]

Most treatments, either available or undergoing trial, have been developed on the basis of their ability to deprive the prostate cancer cell of androgens. This may be achieved by interfering with androgen production at one of several points on the hypothalamic-pituitary-gonadal axis or by blocking androgen receptors at the cellular level.[35] It is noteworthy that the adrenal androgen dihydroepiandrosterone and its related compounds constitute approximately 5% of total circulating androgens and are unaffected by conventional methods of androgen ablation.

Despite the apparent hormonal dependence of prostate cancer, the majority of tumours ultimately progress in spite of hormonal manipulation. There are two theories to explain this phenomenon.

The first theory claims that prostate cancers are histologically heterogeneous tumours containing clones of well-differentiated androgen-dependent cells and poorly differentiated androgen-insensitive cells. The latter cells continue to divide in spite of androgen withdrawal, eventually replacing the regressed androgen dependent tumour bulk until it again reaches clinically significant proportions. The response of the tumour to androgen withdrawal should thus depend upon the proportion of androgen-dependent cells present.[35]

The less attractive theory suggests that cells which are initially androgen-sensitive develop androgen resitance and continue to divide after a period of quiescence.[35]

Bilateral orchidectomy reduces serum testosterone to castration levels (<2nmol/litre) within 12 hours.[36] It is a simple procedure and safe to perform under general or spinal anaesthesia. Common complications include hot flushes and impotence (58 and 85% repectively) although Kaisary et al (1991)[34] found 86% of patients to be impotent before treatment. Harmful psychological effects from surgical castration have often been cited although few objective data support this.[35] A palpable intrascrotal remnant can be left by performing subcapsular orchiectomy, and testicular prostheses are available if required. With the advent of non-surgical alternative, patients should be given an informed choice of treatments. Chadwick et al[37] noted that 23 out of 50 patients

chose orchidectomy, more commonly those who were inpatients at the time of consultation.

Stage D disease.

The treatment of stage D disease is palliation of symptoms by surgery, radiation therapy or hormonal therapy. Hormonal therapy by androgen deprivation can give relief to urinary obstruction symptoms and control of painful symptoms. If urinary obstruction symptoms do not respond within three months of hormonal treatment transurethral resection of the prostate is recommended.[9]

When stage D disease becomes refractory to hormonal therapy after an initial period of response to chemotherapy, prednisolone therapy or local irradiation may provide relief of pain. Most patients however die within 6 months after their disease escapes androgen deprivation.

In this study three patients were categorized as having stage C disease and had urinary outflow obstruction symptoms. These patients were treated by hormonal therapy (bilateral subcapsular ochiectomy (2) and diethy/stilboestrol (1). Four patients had stage D disease and were treated by hormonal therapy (Table V).

Chemotherapy.

The indications and effectiveness of chemotherapy in prostate cancer is still not well defined. Currently cytotoxic agents are used primarily to ease the pain of advanced disease in those patients who relapse after initial oestrogen

response. Cyclophosphamide, 5-fluorouracil, cis-platinum, doxorubicin etc, used alone or in various combinations may give pain relief but does not produce significant regression of primary tumour or metastasis.[6] Yagoda[38] observed objective responses in terminal patients after treatment with nitrogen mustard, cyclophosphamide and 5-fluorouracil. He reported lowering of acid phosphatase levels, decrease in osseous metastasis and changes in cell differentiation in 40% of patients. However, these observations were not translated into improved survival. High doses of prednisolone have been found to give relief of pain in the terminal phase of the disease.[6] None of the patients I studied received this type of treatment.

CONCLUSION/RECOMMENDATION:

Early carcinoma of the prostate can be cured by radical prostatectomy. However, only a few patients present with disease which is still confined to the prostate (stage A and B). Modern diagnostic facilities including transrectal ultrasonography (TRUS), computed tomography (CT) and magnetic resonance imaging (MRI) have not managed to improve early detection of prostate cancer. Digital rectal examination remains to be the best predictor of early lesions.

It has been shown that radiation therapy has comparable therapeutic results as radical prostatectomy with fewer side effects, and is therefore recommended for both radical cure

and palliative treatment. Most patients present with advanced disease (stage C and D) requiring, only palliation of symptoms. Hormonal therapy by bilateral orchiectomy or oestrogen administration has been found to be most effective. In Tanzania, bilateral subcapsular orchiectomy should be considered first after a proper counselling because the alternatives such as LHRH analogues and antiandrogens are expensive, may not be readily available in every place in the country, have side effects and require constant supervision.

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MANAGEMENT OF CLOSED PELVIC RING FRACTURE

SUMMARY:

Ten patients with closed pelvic ring fractures were admitted to trauma wards of the Muhimbili Medical Centre over a period of four months; from August, 1993 to November, 1993. The majority of the patients were aged between 21 and 40 years. The causes of injury included; direct trauma by moving motor vehicles (9), and falling part of a wall (1). Five of the ten patients in this study had stable fractures (type A2), those with type B fractures were three and the rest (2) had type C fractures. Those who had type A2 fractures were either put on skin traction, skeletal traction or Book traction. Associated injuries included; head injury (2), ruptured spleen (2), ruptured urethra (1) and were managed accordingly. One of the patients is presented and then literature on the topic discussed.

INTRODUCTION:

The pelvis possesses exceptional inherent strength and major external forces are required to fracture the normal pelvis. The considerable forces that are required to fracture the pelvis also involve other areas of the body, resulting in many associated injuries. Despite these injuries massive haemorrhage within the pelvis has received a great deal of attention as one of the major consequences of pelvic

fractures. This bleeding can originate from exposed cancellous bone and injured soft tissue at the fracture sites, or from pelvic blood vessels. Under normal circumstances the blood is contained within the pelvis between the parietal peritoneum superiorly and the strong fibromuscular and bony walls on the pelvis. If these barriers are violated by the wounding forces or at surgery, the bleeding no longer will be contained and exsanguination can occur.[1]

The case fatality rate of major pelvic fractures, despite improvement in management, continues to be about 10%.[2] Poole[1] and his colleagues reported a mortality rate of 7.6% (18 of the 236 patients), the causes of death being; irreversible shock from exsanguination, abdominal haemorrhage due to massive visceral or vascular injuries, head injury, sepsis, multiple organ failure and respiratory failure.

The mechanisms of injury are reported to be; falls from heights, crush, and road traffic accidents.[1,3] A thorough knowledge of the anatomical structures contributing to pelvic stability is essential to the assessment and treatment of pelvic fractures. The anatomy of the pelvic ring has been described in detail by many authors, who emphasized the weight-bearing posterior structures.[2] Weight-bearing requires an intact femoral-iliac-sacral arch, secured by the strongest ligaments in the body, the posterosuperior interosseous and dorsal sacral-iliac ligaments. Important contributions to the integrity of the arch are made by the sacrotuberous,

sacrospinous, lumbosacral, iliolumbar, and anterior sacro-iliac ligaments, which provide additional restraints to translation and rotation.[2,4] Potential areas of weakness in the ring are created by the neural foramina of the sacrum, which lie between the body and the lateral mass of the sacral vertebrae. Lesions of the anterior part of the ring are less critical to the stability of pelvic ring that is intact posteriorly.[2]

The stability of the pelvic ring is determined by clinical and radiological means. Computer-assisted tomographic scans provide sequential cross-sectional information about osseous and soft-tissue injury and are extremely useful for identification of the sacro-iliac complex and occult fractures.[2,4] Peritoneal lavage by a supraumbilical open technique,[3] intravenous pyelography and cystography in selected patients[1,3] and Technetium bone scans have been recommended for evaluation of associated injuries.[3]

Management of patients include; quick assessment, resuscitation with fluids and blood, provisional stabilization and definitive stabilization. Associated injuries are treated accordingly.[1,2,3]

CASE REPORT:

Z.L. a 35 year old female patient was admitted to Muhimbili Medical Centre trauma ward, with a history of being involved in a road traffic accident an hour previously. She

was complaining of lower abdominal pain, left anterior upper chest pain and was unable to walk. There was no history of loss of consciousness, bleeding from the ears, nose and throat, and had not passed urine before admission. She was in her menstruation period which started a day before the accident.

Clinically, she was fully conscious, not pale and had a pulse rate of 84 per minute, regular and of full volume. Her blood pressure was 120/80 mmHg. The rest of the cardiovascular system and her respiratory system findings were normal. There was distension of the suprapubic region with a swelling equivalent to a pregnant uterus of 14 weeks which was tender and cystic. On catheterization 800mls of clear urine were obtained and the swelling disappeared. The rest of the abdomen was free of any palpable mass and tenderness. There was no shifting dullness and bowel sounds were heard. Rectal examination revealed normal anal verge and sphincter. There was tenderness of both ischial tuberosities. She had hard stool in the rectum and no blood staining of the examination gloves. Compression of the pelvis revealed tenderness over the pubic area. Passive movements of both lower limbs about the hip joints were normal. No neurological deficit was found.

X-ray of the pelvis showed multiple fractures involving the left superior and inferior rami and the right inferior ramus. The sacro-iliac joints were normal. She had symphysiolysis of 2.0 cm (stage I). In view of the clinical

and radiological findings, a diagnosis of Tile's type A2 closed pelvic ring fracture was made.

The baseline haemoglobin of the patient was 12.5gm/dl. Vital signs remained stable for the next 24 hours. She got 2 litres of normal saline and one litre of Hartimanns solution for a period of 24 hours. Her urine remained clear with a normal output. Grouping and crossmatching was done in case she needed blood. Bilateral skin traction for 6 weeks was applied to stabilize the fractured pubic rami and symphysiolysis.

The patient made a good recovery. The skin tractions were removed after 6 weeks. She began mobilisation with crutches, and was allowed home after achieving good radiological healing. She continued to be seen at the outpatient clinic on monthly basis while she was attending physiotherapy once in a week.

TABULAR RESULTS

Table I: Age distribution of patients with closed pelvic ring fractures.

Age group (years)	Number
10 or less	0
11 - 20	1
21 - 30	4
31 - 40	4
41 - 50	1
Total	10

Table II: Types of pelvic fractures observed.

Type	Number
A	5
B	3
C	2

Table III: Treatment modality of patients with closed pelvic ring fractures.

Type of treatment	Number
. Skin traction	1
. Skeletal traction	3
. Book traction	1
. Bed rest only	5

DISCUSSION:

Classification:

Because of the anatomical complexity of fractures of the pelvic ring, numerous and confusing systems have been described for classification of the injuries.[5,6,7] Classification has been based on one or more criteria: the location of the fracture; the stability of the fracture or involvement of the weight-bearing portion of the pelvic ring, or both; the mechanism of the injury and the direction of the force; and whether the fracture is open or closed.

Beginning with the system of Pennal et al.,[8] the emphasis of current classification schemes has shifted toward the mechanism of injury to assist in the acute hemodynamic and skeletal stabilization of these patients.[6,8] Tile[2] modified the system of Pennal et al, identifying fractures that are rotationally or vertically unstable and emphasizing the

continuum of stability into which all fractures must be placed.[2,8] Young and Burgess added a category of combined injuries because few fractures are the result of a single force. Their classification also provides a comprehensive guide to associated injuries, which often are neglected in the acute post-traumatic period.[6] Using a system based on the mechanism of injury, Young and Burgess reported that most pelvic fractures (41 to 72%) are lateral compression, with anterior-posterior compression occuring 15 to 25% of the time, vertical shear 6% and combination injuries 14%.[6,9,10] In this study Tile's classification was used to classify pelvic fractures (Table II).

Table IV: Tile's classification of pelvic distruption.

Type	Description
A	Stable
A1	Fracture of the pelvis not involving the ring.
A2	Stable, minimally displaced fractures of the ring.
B	Rotationally unstable, vertically stable.
B1	Open book.
B2	Lateral compression: ipsilateral
B3	Lateral compression: contralateral (bucket handle)
C	Rotationally and vertically unstable
C1	Unilateral
C2	Bilateral
C3	Associated with an acetabular fracture

Table IV: (Summarizes the types of fractured pelvis).

Type A - stable minimally displaced.

The pelvic ring is stable and the amount of displacement is insignificant. In type A, the ring is not involved. Avulsion fractures of the iliac spines, the ischial tuberosity and isolated fractures of the iliac wing are included in this group. In Type A2, the pelvic ring is fractured, but is undisplaced and stable. In this study five of the ten patients had type A2 fractures.

Type B- Rotationally unstable, vertically stable.

Type B1 - open book: This injury is caused by external rotatory forces resulting in a disruption of the symphysis pubis, but there is vertical stability. The hemipelvis is unstable in external rotation, but can be made stable by internal rotation. Three stages are recognized. In stage 1 the disruption of the symphysis pubis is less than 2.5 cm and there is no posterior lesion. In stages 2 and 3 the separation at the symphysis pubis is greater than 2.5cm. This implies disruption of the sacrospinous ligament and the anterior sacroiliac ligament. This can occur on one side only (stage 2) or be bilateral (stage 3).

Type B2 and B3 - Lateral compression: The hemi-pelvis in unstable in internal rotation, and may rotate inwardly until it impacts with the opposite hemi-pelvis. The posterior sacroiliac complex is commonly impacted, so vertical displacement is not possible. The stages of lateral compression depend upon the sites of the anterior and

posterior lesions. In Type B2 - Ipsilateral the rami are commonly fractured anteriorly and the posterior complex is crushed. Other varieties include those with an overlapped locked symphysis, or with a superior ramus fracture rotated around the disrupted symphysis. In the latter case a fragment may protrude into the perineum, the so-called a "tilt" fracture. In type B3-contralateral (bucket handle) the cause is usually a direct blow on the iliac crest. The major anterior fracture is on the opposite side to the posterior lesion, but all four rami may have fractured anteriorly. The affected hemi-pelvis rotates anteriorly and superiorly like the handle of a bucket so that, even if the posterior structures are relatively intact, the patients may have a major limb length discrepancy. Reduction requires derotation of the hemipelvis rather than traction in the vertical plane. In this study three patients had type B fractures; type B1 (1), type B2 (2).

Type C - Rotationally and Vertically unstable.

There is rupture of the pelvic floor including the posterior sacroiliac complex, as well as the sacrospinous and sacrotuberous ligaments. This injury may be unilateral (Type C1) or bilateral (Type C2). The radiographic signs of instability include posterior displacement of the hemi-pelvis by more than 1cm, avulsion of the transverse process of the 5th lumbar vertebra, and detachment of the bony insertion of the sacrospinous ligament from either the sacrum or the ischial

spine. Diagnosis of the posterior changes associated with gross vertical instability is best made by computerized tomography (CT). In type C3 there is an associated acetabular fracture. Pelvic ring disruptions associated with acetabular fractures should be considered separately, since the prognosis is more dependent on the result of treatment of the hip than upon that for the pelvic ring.

In this study two patients had type C fractures of the pelvis; type C1 (1) and type C2 (1). One of the two patients was run over by a motor vehicle and the other was knocked by a moving motor vehicle.

Diagnosis:

Early management of patients who have sustained trauma must include proper diagnosis of all injuries. A high index of suspicion is essential for the diagnosis of pelvic fracture, particularly when the patient is uncooperative or unconscious due to the injury[3] Mc Carrol et al,[11] noted that 93 of the 200 pelvic fractures that had been sustained by people who died in a pedestrian - motor vehicle accident were unsuspected until the autopsy.

Clinical examination:

Inspection is essential, as abrasions and contusions point to other contiguous structures that may have been injured and they alert the examiner to major skin or soft-tissue injury. The Destot sign (a superficial haematoma above the inguinal ligament or in the scrotum or thigh) may indicate

a pelvic fracture, as may a limb length discrepancy or an obvious rotational deformity of the pelvis or lower extremity.[2,12]

The pelvic ring, including the symphysis, pubic rami, iliac crests, sacro-iliac joints, sacrum and ischial tuberosities, should be palpated in search of tenderness, asymmetry, or crepitus in patients who are haemodynamically stable. Bimanual compression and distraction of the iliac wings, and abduction and adduction of the hip should be done to detect instability or crepitus.[5] Manual traction can aid in the determination of vertical instability.[2,6] Careful rectal and vaginal examinations should be performed in order to determine whether the pelvic injury communicates with these structures.[1,13] During the rectal examination, the examiner can gently palpate the sacrum for tenderness or asymmetry.[7]

Radiography:

Radiographs are the principal diagnostic tool and so-called gold standard in the assessment of patients for suspected pelvic fractures. This is due to their low cost, widespread availability, and familiarity to physicians.[1,4,14] They are used for rapid screening of people who have sustained a traumatic injury, and they provide considerable information on the location stability of the pelvic fractures and associated soft-tissue injuries.[10] A single anteroposterior radiograph of the pelvis of a trauma patient is mandatory for an initial assessment of the pelvic ring stability, in the

emergency evaluation.[2,6] The arcuate, iliopubic, ilioischial, and acetabular tear drop lines, the sacral foramina, and the sacro-iliac joint should be scrutinized carefully and identified in a systematic manner.[7] Young and Burgess stated that 90% of all traumatic injuries to the pelvic ring can be diagnosed on anteroposterior radiographs alone.[9] However, due to shadows cast by soft tissue structures, superimposed osseous structures, and the sagittal inclination of the pelvic ring, inlet and outlet radiographs,[8] should also be made in haemodynamically stable patients to further delineate suspected fractures of the pubic ramus or sacrum and superior, posterior, or rational pelvic displacement.[2,9] Tile considered these radiographs to be more important than anteroposterior radiographs.[2] They can demonstrate 94% of all pelvic fractures.[9] Inlet view clearly, shows posterior displacement and the outlet view clearly shows posterior displacement and the outlet view shows superior migration or rational.[5,8]

It is also important to remember that an unstable pelvic distruption may appear reduced on the initial radiograph. Stress radiographs may be needed to demonstrate this instability in the haemodynamically stable patients. Also it should be remembered that an isolated anterior fracture of the pelvic ring is uncommon. If such a fracture is seen, a second posterior disruption should be sought.[6] Tile stated that if the pelvic ring is broken in one place and the fragments are displaced, there is another injury in the ring.[2]

Computer-assisted tomographic scans:

Computerized tomography (CT) provide sequential cross-sectional information about osseous and soft tissue injury and are extremely useful for identification of the sacro-iliac complex and occult fractures.[5,15] The recent refinement of three-dimentional CT allows clearer visualization of the pattern of injury.[2,7] CT is valuable in the non-emergency evaluation of fractures of the pelvis. CT is also important in evaluating associated intra-abdominal injuries.[1,3]

Technetium bone scans:

These have been useful in the evaluation of patients who have persistent pain in the hip or the posterior part of the pelvis after an apparently isolated injury to the anterior part of the pelvic ring.[16,17] They are particularly helpful for the discovery of sacral fractures in osteoporotic bone, but they have no role in the assessment of high-energy pelvic disruptions. A specific role for the use of magnetic resonance imaging (MRI) in the evaluation of acute pelvic fractures had not been well defined.[7] Other investigations are done to rule out associated injuries which influence management.[3]

Peritoneal diagnostic lavage (PDL):

PDL is recommended on a selective basis in patients with pelvic fractures. The procedure is performed for the following indications; unstable haemodynamic state, signs of peritoneal irritation, and altered awareness due to associated head

injury or intoxication with suspected abdominal injury.

The procedure is important for evaluating intra-abdominal injuries accompanying pelvic fractures. The approach is by supraumbilical incision that exposes the peritoneum. It is reported to be more accurate than the blind introduction of a needle.[1,3] Patients with a red blood cell count of more than 100,000/ml or a white cell count of more than 500/ml, are regarded to be positive.[1]

Intravenous pyelography (IVP) and cystourethrography:

These procedures are also important in a few selected cases. They are used to evaluate the genitourinary tract in patients with haematuria associated with fractures of the pelvis. Blood in the urethral meatus after injury should be investigated by urethrography before inserting a urinary catheter. Patients with gross haematuria should be appropriately investigated immediately after an initial resuscitation.[1,3]

The problems related to the radiological investigations of the genitourinary tract are: 1. Patients with fractures of the pelvis may have associated injuries and often are unstable. The time required to perform urological studies is better utilized to stabilize the circulation or perform other more useful investigations such as angiograms and CT; 2. Exposure to the hyperosmolar contrast material of elderly patients, with or without pre-existing renal disease may further depress renal function; 3. The negligible yield of the

screening test in a large group of patients.[3]

Arteriograph: This investigation can disclose haemorrhage from the pelvic blood vessels. Actively bleeding vessels can be embolized with autologous clot, gelatin sponge or coils.[1]

Radiographs: Additional films of the cervical spine, chest and extremities are taken if clinically indicated. In the patients studied, clinical examination and anterior-posterior radiographic views of the pelvis were the only means used to evaluate fractures of the pelvis.

Treatment: Patients who have high-energy fractures of the pelvis often have accompanying genito-urinary, neurological, vascular, and other skeletal injuries that complicate treatment, increase mortality and morbidity, and necessitate intensive care. The associated injuries are primarily responsible for the death of patients who have severe pelvic fractures.[1,3] The presence of these associated injuries necessitates a consistent and thorough evaluation. The goals of treatment of high-energy pelvic distruptions are the prevention of early death from haemorrhage, the early detection and treatment of all concomitant injuries, and the restoration of the patient to the preinjury level of function.[1,7]

Haemorrhage:

High-energy pelvic fractures can be accompanied by life-threatening haemorrhage with multiple sources of bleeding, including osseous, vascular and visceral structures.[6]

However, advances in resuscitation have markedly decreased the mortality that is associated with haemorrhage from pelvic fractures.[18] The energy of the injury is absorbed only partially by the fractured pelvis; the soft tissues must absorb the remainder. Distortion and disruption of the soft tissues results in haemorrhage. Shock can occur before the haemorrhage is recognized,[19] and the amount of blood that is lost and the subsequent mortality is directly proportional to the severity of the pelvic fracture.[1,2,20,21] While the primary source of bleeding may differ (it can be a major vessel, multiple small vessels, organs or bone), early diagnosis and treatment of haemorrhage is essential.

Fluids and Blood. The first step in the treatment of blood loss is prompt and sufficient replacement of fluid. Three litres of a balanced salt solution should be given initially. Administration of type-specific packed red blood cells or whole blood should follow immediately. Replacement of blood and fluids has been found to be sufficient to achieve haemodynamic stabilization in two-thirds of all patients who have a pelvic fracture and an intact peritoneum posteriorly.[21] When replacement of fluid and blood does not stabilize the patient's vital signs, additional steps must be taken such as diagnostic peritoneal lavage to diagnose intra-abdominal haemorrhage.

Pneumatic antishock garments (PAG).

These have been promoted as techniques to reduce bleeding

from pelvic fractures by providing tamponade, apposing the fracture fragments and reducing the volume of the pelvis.[1,6] However, the use of antishock trousers once the patient has arrived at the hospital is not common. The trousers limit access to areas of the body that may be injured; they decrease expansion of the lungs, with possible resulting respiratory compromise; and they may contribute to the development of compartment syndromes in hypoperfused extremities.[6]

External pelvic fixation.

The use of external pelvic fixation for the management of haemorrhage developed primarily out of concern that the mobile, sharp edges of a fractured bone lead to dislodgement of clotted blood and renewed bleeding. Use of an external fixator allows access to areas that would be covered by antishock trousers. Definitive stabilization of a fracture can be achieved with external fixation in patients who have certain rotationally unstable but vertically stable injuries. The device can be used in the emergency room or the operating room. There has been controversy about the best time to apply the device when an exploratory laparotomy for intraperitoneal haemorrhage is to be performed. However, there have been few clinical studies to confirm reduction of blood loss after placement of the device.[2] This mode of treatment was not used in this study.

Skeletal injury:

Early assessment of pelvic stability is essential for

determination of optimum (non-operative or operative) method of treatment. If the pelvic ring is stable and only minimally displaced (Type A2) symptomatic treatment only is necessary. These patients may be mobilised quickly and the pelvic fracture largely ignored. The definitive management of displaced fractures depends upon their stability and requires clear definition of the risks and benefits of stabilization.[2] Five patients had type A2 pelvic fractures and were put on bed rest and analgesics (tables II and III).

Type B1- open book:

When the symphysis is open less than 2.5 cm (stage 1), no specific stabilization is usually required. A good result may be expected although in some cases pain at the symphysis may persist for months or even permanently.

When the symphysis is open more than 2.5cm (stage 2 and 3) the pelvic ring may be closed by placing the patient in the lateral position. The reduction may be maintained either by a simple external skeletal fixator or by a plate across the symphysis.[2] In this study one patient had this type of injury and book traction was applied (Table III). If the patient needs a laparotomy and there has been no faecal or urinary contamination, some authors favour plating or external fixation.[2,22]

Type B2, B3 - lateral compression injuries:

These are the commonest group of pelvic ring fractures. In most cases of Type B2 (ipsilateral) injuries, the elastic

recoil of the pelvis restores the anatomy to near normal and no form of stabilization is required. In this study two patients had type B2 and were put on skeletal traction.

Type B3- bucket handle injury: In this type of pelvic injury, the posterior complex is commonly compressed, rendering the ring stable. It may be impossible to reduce the displacement by closed manipulation under general anaesthesia, and in most cases, no stabilization is required. It is usually preferable to accept a leg length discrepancy of up to 1.5 cm and some internal rotation of the hemi-pelvis rather than destabilizing the pelvis and producing the need for an external frame or internal fixation.

If the leg length discrepancy is greater than 1.5 cm, or if the pelvic deformity is excessive, more aggressive management may be indicated. Reduction may then be obtained by external rotation of the hemi-pelvis by pins in the iliac crest.[2] When reduction has been achieved, the anterior frame is completed to hold the necessary external rotation. In the rare case of a "tilt" fracture where bone is protruding into the perineum, open reduction and internal fixation may be required.

Type C- Rotational and vertical instability:

In these case, the options for definitive fixation include external frames with or without skeletal traction, and open reduction with internal fixation. Complex frames have a biomechanical advantage over simple frames, but no frame can

restore enough stability to allow the patient to walk without loss of position; since the use of these frames add risk to the procedure without substantial benefit, they are not indicated.

The combination of an anterior frame with skeletal traction provides the surgeon with a safe method of treatment which yields a high percentage or satisfactory results in the right cases.[2,6] To be successful, traction must be maintained for at least 8 to 12 weeks, depending on the rate of radiographic healing. Tile[2] et al favours a supracondylar traction pin in the femur, and up to 20Kg. of traction may be necessary to maintain the posterior sacroiliac complex in the reduced position.

The indications for the use of external fixation have been summarized by some authors[8,22] as follows;

1. resuscitation, to decrease pelvic volume and haemorrhage (a three-centimetre diastasis increases the true pelvic volume by a factor of two).
2. Provisional management, to help to mobilize the patient for other tests, make the patient more confortable in the early post-injury period, and allow for possible later open reduction and internal fixation.
3. Definitive management of certain patients, particularly those who have a vertically stable but rotationally unstable pelvic fracture.

The development of techniques for open reduction and

internal fixation of pelvic fractures has been slow because of concern about violation of the retroperitoneal space with recurrence of haemorrhage and about possible iatrogenic contamination with subsequent infection of the retroperitoneal space, the risks of skin-sloughing (especially posteriorly) and of iatrogenic injury of nerves.[22,23] These possible complications must be weighed against the benefits of decreased morbidity of the patient and the increased stability provided by internal fixation.[2] Tile stated that the role of internal fixation is not clear as good results have been obtained with external fixation and traction.[2] However, biomechamical studies have shown that, of all forms of fixation, internal fixation of both the posterior and the anterior portions of the pelvic ring provides the greatest stability (approximating that of the intact pelvis).[2,22,23].

The indications for open reduction and internal fixation have been summarized by various authors.[2,6,23] They included;

1. pure ligamentous injury posteriorly
2. failed closed reduction or residual displacement after external fixation.
3. multiple injuries
4. associated acetabular fractures
5. open posterior wound without perineal contamination and
6. a vertically unstable fracture.

The current techniques for posterior internal fixation[2,6,23] include anchorage of the injured hemipelvis to

the opposite ileum with sacral bars, use of double cobra plates with supplemental screws across the sacro-iliac joints, anterior fixation of the sacro-iliac joint with a plate, and lag screw fixation of the ileum into the sacrum or across an iliac fracture, with or without supplementation by a plate. Fixation with one or two sacral bars requires that one hemipelvis be intact posteriorly, for anchorage of the opposite unstable pelvic segment. Fixation with double cobra plates is useful in patients who have a bilateral posterior discruption. The disadvantages are; the operative approach is extensive, the implant is large and precise placement of the screw into the sacrum is difficult.

Anterior fixation of the sacro-iliac joint with a plate avoids any contused areas of the buttocks. Excellent visualization and reduction of the discrupted sacro-iliac joint can be achieved, except in very obese patients. Special care must be taken to prevent injury to the 5th lumbar nerve during anterior exposure and fixation of the sacro-iliac joint.

Anterior fixation has been used for disruptions of the symphysis of more than two centimetres, for fractures of the pubic rami with marked displacement, or after a laparotomy.[22,23]

CONCLUSION/RECOMMENDATION:

Pelvic ring injuries are common findings in motor vehicular accidents. Once they occur are associated with

injuries of other parts of the body hence, careful physical examination and radiography are important diagnostic tools. Special care for head injury patients as one can miss a fatal associated injury. Due to lack of other diagnostic facilities such as CT scanning, some of the pelvic injuries such as those involving the posterior weight bearing sacroiliac complex can be missed. In Tanzania most of the orthopaedic and trauma surgeons are in referal hospitals, as a result the majority of trauma patients are managed by general medical practitioners or general surgeons only. These patients are put into a disadvantage as they don't fall into the right speciality hands. In view of that the recommendation to doctors in the districts and regions is to refer early the patients they think can benefit from orthopaedic and trauma speiciality. Also the flying doctors team should visit regional hospitals and teach the available medical team the measures for resuscitating patients with fractured pelvis and the definitive treatment according to the available facilities.

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