A DISSERTATION OF TEN SURGICAL CONDITIONS PRESENTI

AS PART OF THE REQUIREMENTS FOR THE AWARD OF

THE DEGREE OF MASTER OF MEDICINE (SURGERY) OF

THE UNIVERSITY OF DAR ES SALAAM.

1995

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## DECLARATION

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## ACKNOWLEDGEMENTS

During the 3 year in M. Med course in Surgery, I have had the honour of training under various devoted teachers to whom I feel greatly indebted.

I particularly thank Dr. M. R. Aziz, my supervisor whose guidance, correction and valuable criticism of the protocol, encouragement and supervision has made my three years training a success. I also wish to thank Dr. R. E. Kisanga, the Head of Surgical department for his constant encouragement and guidance throughout the course.

I also owe special thanks to my teachers who gave me direct assistance in the write up of this dissertation. These include Prof. B. R. Kirei, Dr. C. A. Mkony, Dr. E. M. Kessi, Dr. R. I. Mhina and Dr. L. Museru.

I would like to convey my gratitude also to all the teachers in different surgical specialities who have made valuable contributions to my training.

I should also thank all senior and junior colleagues with whom I had great pleasure to work and learn.

To Muhimbili Medical Centre I owe sincere thanks for whatever facilities that were availed to me during the entire period of training.

I thank also my wife Dr. Vida Mmbaga for her love, understanding and encouragement. Finally my sincere thanks to Mr Mponzi P, Ms. Kessy and Jutto for typing this dissertation.

## ABSTRACT

This dissertation is submitted as part of the requirement for the award of the degree of Master of Medicine (Surgery) of the University of Dar es Salaam. It comprises ten surgical conditions divided in three parts. Part one consists of five conditions/pertaining to general surgery. Part two consists of two cases in urology and part three, three cases in orthopaedics and traumatology.

In each topic a case is presented in detail; the rest of the cases are summarized; A review of relevant literature centred on area of controversy and recent advances in the management of the condition done. The topic is discussed with particular references to observations made at Muhimbili Medical Centre.

Surgery is so wide so that its acquisition from daily practice, audio or visual aids is endless. Moreover more facts are accumulating daily.

I am obliged to admit that I will remain a student in surgery until when my life expires.

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# PERFORATED DUODENAL ULCER


## SUMMARY:

Seven patients who had emergency surgery for perforated duodenal ulcer in surgical firm one of the Muhimbili Medical Centre were studied during the period January 1992 to June 1993. There were 5 males and 2 females and their ages ranged between 20 and 67 years. All patients presented with severe upper abdominal pain which became generalized shortly thereafter and was associated with muscle guarding and tenderness on examination. Diagnosis was made on the basis of a typical history and clinical findings and was confirmed by presence of gas under the diaphragm on erect chest radiographs. Simple surgical closer of the perforation, copious peritoneal lavage and drainage of the pouch of Morrison was performed in all cases. Five patients were free of symptoms six months after surgical closure while one required definitive ulcer surgery.

One patient died postoperative, possibly of paralytic ileus or re-leak while one was lost from follow up. Wound sepsis was the commonest complication. In the following review one case of perforated peptic ulcer is reported in detail while the others are summarized in tabular form. The pathophysiology, clinical features and treatment is discussed.


## INTRODUCTION:

Although an initial report of peptic ulcer appeared in 1688, the first unequivocal duodenal ulcer was described in 1799 by Mathew Bailie of London. In

1912, Julius Friedenwald reported a series of 1000 cases of peptic ulcer disease approximately half of them having duodenal ulcer and described its characteristic symptoms[1]. Since the early part of this century duodenal ulcer disease has become recognized as a common chronic disease particularly affecting young men[1]. Duodenal ulcer comprises about 80% of all peptic ulcer disease patients[2]. It is impossible to make a rigid statement of the frequency of duodenal ulcer because there have been and indeed continue to be changes year by year. Two hundred years ago, it was very rare and it has only become common since about 1920. From that time, the frequency has increased to reach a peak in about 1955 and since then there has been a steady decline[3].

There are district geographical differences not only between different countries but also within a country. In the British Isles the highest rate is reported to occur in Scotland[2]. In African countries there is little data available and there is need for other similar studies[4].

Kakande in a prospective study of 240 patients with dyspepsia at a rural hospital in Kenya found 13.7% had peptic ulcer disease[4]. In Tanzania, Dolman et al in a study of 320 patients with dyspepsia who had gastroduodenoscopy found 26.3% had duodenal ulcers[5].

Duodenal ulcer is more common in males than females. The male to female ratio varies between 2 : 1 in England[1] to 9 : 1 in Africa[6] and 18 : 1 in India[7].

One of the major complication of duodenal ulcers is perforation. Badoe working in Accra in Ghana recorded 54 cases of perforated duodenal ulcer within a period of 6 years with an average of 10 cases per year[8]. Hiza in an analysis of

surgical admissions at the Muhimbili Medical Centre in 1971 recorded 40 patients diagnosed as peptic ulcer and 6 patients had perforation[5].

Acute perforation of the duodenum is now estimated to occur in 5-10% of patients with ulcers[9]. On the other hand 30% of patients presenting with a perforation have no previous history of peptic ulcer disease[1].

## CASE REPORT

R. W. IP NO 632114 a 34 years old man was admitted in July 1992 with complaints of epigastric pain for two days and vomiting for one day.

The pain had been gradual, burning in nature, continuous at first in the epigastrium, radiating to the back and then became generalized. On the day of admission the pain had been severe and was associated with a low grade fever, nausea and vomiting. The vomitus was yellowish and non projectile. He had a past history of epigastric pain recurrent for two years but of a lesser intensity. It was exacerbated by taking spices, asprin and beans but relieved by taking antacids. His appetite was normal and he had not lost weight. His bowels were regular and he had not noticed passage of black tarry stool. He had dry cough with chest pain. The rest of systemic review was normal. He had several attacks of similar pain in 1991 and was admitted in one of the hospitals in the city twice that year. He was a primary school teacher and did not smoke cigarettes or drink alcohol. There was no other family member with similar problem and there was no chronic disease in his family.

General examination revealed an ill looking man in pain, restless, moderately dehydrated and not jaundiced. His temperature was 38.2 °C and his respiratory rate was 28/minute. His pulse rate was 96 per minute regular and of good volume and his blood pressure was 120/70 mmHg. He had no pitting oedema and had no

enlarged peripheral lymph nodes. Abdominal examination showed a scaphoid abdomen moving minimally with respiratory movements. There was an old traditional scarification in the epigastrium. The epigastrium and right lower quadrants were markedly tender with rebound tenderness. Liver and spleen were not palpable and liver dullness was absent. Bowel sounds were normal. Rectal examination revealed a tender recto-vesical pouch which was not bulging. The prostate and rectal mucosa were normal. The gloved finger was stained with normal coloured stool. The rest of the systems were clinically normal. The patient was diagnosed to have peritonitis probably due to a perforated peptic ulcer.

He was started on intravenous Hartmann solution and ampicillin 500 mg six hourly. A nasogastric tube for active gastric aspiration and a size 18 three way Foley's catheter for urine output monitoring were inserted. An urgent erect chest x-ray was requested which showed gas under the right hemidiaphragm. He was prepared for emergency laparotomy and a written consent was obtained. Blood was sent for grouping and two units were cross matched.

Under general anaesthesia and endotracheal intubation the abdomen was opened through an upper midline incision. On entering the abdominal cavity there was a gash of gas. The peritoneal cavity was found to have fibrinous exudate and bile stained peritoneal fluid. Small particles of previously eaten food were seen but there was no frank pus. The right lobe of the liver was retracted upward and the stomach pulled gently downwards and to the left of the patient. A perforation measuring 1 cm x 1 cm with oedematous edges was found on the anterior wall of the first part of the duodenum. There was no marked induration around the perforation.

Other viscera were inspected and found to be normal. The perforation was closed by using 3 sutures which were tied snugly over an omental patch, the middle suture being tied last. The peritoneal cavity was lavage with warm normal saline and dried. A corrugated rubber drain was placed in the hepatorenal space of Morrison and the abdomen was closed in layers.

Post operatively the patient had his vital signs charted half hourly for 12 hours. He continued with 5% dextrose two litres and one litre of normal saline over the next 24 hours. He also continued with ampicillin six hourly for 72 hours. He was given Pethidine 100 mg six hourly for 24 hours for pain. An input/output chart was ordered. The catheter was removed after 48 hours. On the third post operative day, normal bowel sound were established and his vital signs remained stable. There was minimal drainage and the nasogastric tube together with rubber drain were removed. He was allowed oral sips and his general condition continued to improve steadily. The wound was infected at the umbilicus and two sutures were removed. The patient was discharged eleven days later in good condition. At follow up one month later, he was symptom free and was asked to report for review after 3 months. Unfortunately he never reported back to the clinic and it was therefore not known for sure if he remained symptomless.

## RESULTS OF THE PATIENTS UNDER STUDY

Table I: Age and sex distribution of the patients with perforated peptic ulcer

| AGE GROUP | NUMBER OF PATIENTS | | TOTAL |
|---|---|---|---|
| | MALE | FEMALE | |
| 20 - 29 | 2 | 0 | 2 |
| 30 - 39 | 2 | 1 | 3 |
| 40 - 49 | 0 | 1 | 1 |
| 50 - 59 | 0 | 0 | 0 |
| 60 - 69 | 1 | 0 | 1 |
| TOTAL | 5 | 2 | 7 |

The majority of patients were at 3rd and 4th decades of life.

Table II: Duration of severe abdominal pain at the time of admission

| TIME IN HOURS | NUMBER OF PATIENTS |
|---|---|
| 1 - 12 | 3 |
| 12 - 24 | 3 |
| 24 - 48 | 0 |
| 48 - 72 | 1 |
| TOTAL | 7 |

The majority of patients presented within 24 hours after acute onset of abdominal pain.

Table III: Clinical presentation

| SYMPTOMS AND SIGNS | NUMBER OF PATIENTS |
|---|---|
| Severe abdominal pain | 7 |
| Vomiting | 6 |
| Nausea | 3 |
| Fever | 1 |
| Tachycardia/hypotension | 2 |
| Abdominal tenderness and guarding | 7 |

In all patients generalized abdominal pain associated with tenderness and guarding

were found.

Table IV: Postoperative complications

| COMPLICATIONS | NUMBER OF PATIENTS | % |
|---|---|---|
| Wound Sepsis | 3 | 43 |
| Death | 1 | 14.3 |

No major complication was seen after simple surgical closer except wound sepsis. One patient who was 60 years of age died 4 days after operation possibly due to re-leak or paralytic ileus with fluid and electrolyte imbalance.

**DISCUSSION**

The gastric and duodenal mucosa is normally protected from the potentially harmful effect of acid-pepsin by ill defined mechanisms of resistance[3]. Surface epithelial cells secrete mucus which provides an unstirred layer over the mucosa to the epithelial surface and an effective barrier to pepsin. Continuous bicarbonate secretion by the gastric and duodenal surface epithelial cells into the overlying mucosa layer results in a pH gradient in which the pH varies from approximately 7 at the epithelial surface to less than 2 in the gastric or duodenal lumen[1]. In addition gastric mucosal cells have apical membranes and tight junctions that are impermeable to the back diffusion of acid into the cells[2]. Other factors important in the maintenance of mucosal integrity include blood flow and the ability of epithelial cells to renew themselves rapidly and to migrate along the basal membranes to areas of minor injury a process referred to as "reconstitution"[1,2]. Furthermore, cytokines and

growth factors play a role in mucosal defence; although their precise role requires further study[3]. It is also known that prostaglandins enhance the mucosal defence factors through the stimulation of mucus and bicarbonate secretion and blood flow. Thus there are numerous interacting factors that serve as defensive mechanisms for the maintenance of gastroduodenal mucosa integrity.

Peptic ulcers are a result of an imbalance between these mucosal defensive mechanisms and the "aggressive" factors primarily the potentially noxious effects of acid and pepsin[2,8]. Peptic ulcers are result of multiple heterogenous factors and there is no single pathological factor that applies to all cases. Hydrochloric acid and pepsin are essential permissive factors in the pathogenetic process of ulcer formation; Schwarz's dictum" no acid no ulcer" therefore still applies[4]. However it is known that only about a third of duodenal ulcer patients are true hypersecretors of gastric acid. The possible mechanisms of increased acid secretion is attributed to an enlarged mass of acid and pepsinogen secreting oxyntic glands[10].

Non steroidal anti-inflammatory drugs (NSAID) are associated with development of mucosal ulceration in 15% to 20% of patients. There is an estimated 8 fold increase in duodenal ulcer among people who use NSAID daily. This arises from the inhibition of cyclo oxygenase resulting in decreased mucosal prostaglandins synthesis and impaired platelet aggregation on the mucosa. It is believed that chronic NSAID users are at least 4 to 7 times likely to develop gastrointestinal perforation[3].

Cigarette smoking, caffeine and corticosteroids have been implicated in reducing mucosal protective barrier, retard ulcer healing and promote ulcer recurrences[1,3].

Genetic predisposition, bile reflux, rapid gastric emptying and underlying medical illnesses (cirrhosis, chronic obstructive pulmonary disease and cystic fibrosis) have been also found to play a pathogenic role in some patients[10].

Helicobacter pylori, a gram negative flagellated rod found in the antrum of more than 95% of duodenal ulcer patients has been implicated in causing peptic ulcer through an inflammatory process with breakdown in mucosal resistance to acid and pepsin. It is believed that duodenal ulcer patients have gastric metaplasia in the duodenum with the presence of the helicobacter pylori at this site in 50%[3,7].

Pathophysiology of perforated duodenal ulcer

When an ulcer extends through the entire wall of the duodenum rapidly enough that the resulting deficit cannot be sealed off by adhesions, duodenal contents leak into the peritoneal cavity. This produces chemical irritation which after the first 6-8 hours becomes secondarily invaded by bacteria[9]. The initial chemical peritonitis is produced by the presence of acid and pepsin within the peritoneal cavity. This reaction is characterized by an increased capillary permeability and transduction of plasma, water and sometimes blood elements. There is also a third space fluid shift and sequestration in the lumen of the gut, the visceral tissues and peritoneal cavity[3]. Progression of this third space leads to a decreased circulating volume and consequently hypovolaemic shock. This then leads to inadequate perfusion, the cell pumps fail and potassium is lost extracelluarly. As oliguria progresses, hyperkalaemia and dilutional hyponatraemia develop. Metabolic acidosis occur as a result of anaerobic metabolism and the accumulation of lactic acid.

With time and continued leakage from the perforation peritonitis become septic due to secondary bacteria invasion producing endotoxin and exotoxin by products of cell breakdown which in him further deleteriously affect the peripheral vascular system and microvasculature of the gastrointestinal tract[3,8,9].

Clinical presentation

Perforation of a duodenal ulcer brings about a remarkable series of dramatic changes. Immediately prior to perforation the patient may be well or within a few minutes of perforation be in severe pain and within an hour be desperately ill.

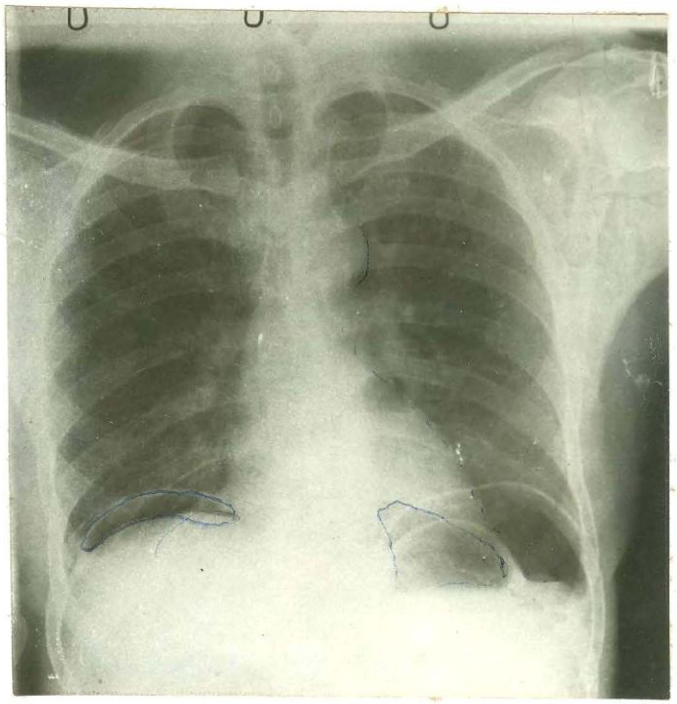
As the ulcer perforates, the patient experiences severe abdominal pain in the epigastrium or in the right upper quadrant which becomes generalized shortly thereafter. At that moment, any movement is painful and the patient lies still in bed with shallow breathing. The pain may be referred to one or both shoulders as a result of diaphragmatic irritation[2]. There is usually associated vomiting. All patients in this study presented with characteristic abdominal pain. Friedenwald[1] reported in 86% of his cases, classical abdominal pain and vomiting in 50% of his series. There was haematemesis in 7.4% of the patients. Six of the 7 patients (85.7%) in this study presented with vomiting but none had haematemesis. Intense epigastric pain radiating to the back, associated with vomiting and retching is also a feature in acute pancreatitis and upper small intestinal obstruction[2].

Physical examination within the first few hours reveals a subnormal temperature with a rapid pulse. The abdomen is characteristically board like rigidity with loss of normal dullness on percussion over the liver. After 3 to 6 hours, the

tenderness and rigidity may decrease due to dilution of the irritant fluid by the peritoneal exudate (the period of illusions). After 6 hours diffuse peritonitis develops accompanied by distension and absent bowel sounds. Enough free fluid may have accumulated in the peritoneal cavity to be detectable[2].

Twenty nine percent of the patients in this study had rapid pulse and a low blood pressure and all patients had a tender abdomen with guarding. A plain erect abdominal and chest x-rays whereby free air is found under the right hemidiaphragm is confirmatory (Figure I) and is found between 59 - 80%[11]. A left lateral decubitus with the right side up or erect chest films are preferred to the erect abdominal film for detection of air[2,11]. Of the seven patients in this study, there was air under the diaphragm on the erect chest films in three patients while in two patients x-rays were not taken and in one patient the x-ray was of poor quality. If free intraperitoneal air is not demonstrable but a diagnosis of perforated peptic ulcer is strongly suspected, a gastroduodenogram using water soluble contrast media can be performed. This method has been found to be 90% accurate in detecting stomach and duodenal perforations[11]. In the remaining cases the perforations may have sealed and with no evident spillage of the contrast[2,8].

Fig 1: Chest X ray film showing gas under both hemidiaphragm a feature suggestive of perforation of duodenal ulcer.



## TREATMENT

1. Conservative treatment

Perforated duodenal ulcer is treated by operation but on rare occasions may be managed conservatively. This method is useful in patients who are old and have associated cardiac, pulmonary or renal disease or those who are in irreversible shock and when facilities for surgical treatment are not available[5]. Wisbur[1] has proposed conservative management that involves correction and maintenance of fluid and electrolyte balance by intravenous infusions, continuous nasogastric tube aspiration, parenteral broad spectrum antibiotics and H2 receptor antagonists. The patient is carefully monitored with regular examination; monitoring temperature, pulse rate, blood pressure and input/output fluid chart. Stable vital signs, absence of generalized abdominal signs and lack of spillage on gastrografin examination indicate that the perforation has sealed[11]. Conservative therapy is abandoned in favour of operation if clinical determination suggests continued leakage and worsening peritonitis.

The disadvantage of this method of treatment is that occasionally more serious causes of peritonitis like strangulated bowel may be missed and wrongly treated. Donovan[8] has reported a mortality of 4.5% in a series of 120 patients with perforated peptic ulcer treated conservatively. There was no patient in this study who was treated by the conservative method.

2. Surgical treatment

Surgical treatment is regarded as the treatment of choice in patients with perforated duodenal ulcer[1]. There are several different modalities of surgical

treatment from simple plication followed by definitive surgery which include:

1. Truncal vagotomy and drainage by pyloroplasty or gastroenterostomy.
2. Vagotomy and antrectomy
3. Highly selective vagotomy after plication.

The outcome of these procedures depends very much on preoperative preparation of patients.

Preoperative preparation

It is essential that before surgery the patient is adequately resuscitated to correct the existing fluid and electrolyte deficit. A nasogastric tube is inserted for gastric decompression and morphine or pethidine for relief of pain and preoperative broad spectrum antibiotics for example cefuroxime are prescribed. Pulse and blood pressure are monitored regularly, a urinary catheter is inserted and hourly urine output is recorded in the presence of shock.

Simple closure method

Simple closure of the perforation is a simple and life saving procedure but has limited value in terms of definitive cure. Most authors use simple closure in patients with acute perforations, in poor risk patients and those with major intraperitoneal sepsis or with intraabdominal abscess. It is also applicable to those who have serious concurrent disease.

Simple closure is done by either suture plication in the case of small less than one centimetre perforated ulcers or by use of an omental onlay patch for large

ulcers; a procedure popularized by Roscoe Graham in 1937[8,12]. The closure of the perforation is performed by inserting three to four absorbable sutures such as catgut or dexon on the edges. A free omental graft is then placed over the perforation and the sutures are tied snugly over it. The central suture which crosses the perforation is inserted or at least tightened last so that it is less likely to cut out the oedematous gut wall. Omental tissue has been shown to enhance fibrin deposition at the site of closure.

Some authors prefer plication by seromuscular interrupted non absorbable Lambert sutures along the transverse axis of the bowel. It is suitable in acute perforations where tissues are still pliable. Narrowing of the duodenum is one of the complication noted with the use of Lambert sutures[13].

Simple closure using 3 sutures aided by an omental patch was the main mode of treatment used in this study. All patients had various degree of peritoneal soiling and sepsis hence definitive emergency surgery was considered to be risky in these patients. Moreover the surgeon dealing with such emergency surgery was usually not experienced enough to perform definitive surgery at the same sitting.

Follow up of patients with perforated duodenal ulcer treated with omental patch or other form of plication leads to a satisfactory long term results in only one third of patients. The remaining two thirds of the patients develop recurrent duodenal ulcer symptoms and between 30-50% of these need definitive surgery[13,14]. In a more recent review by Boey and Ivong; major ulcer complications occurred in 52% of patients (28% bleeding, 15% pyloric obstruction, 9% re-perforation with proved recurrences after simple closure)[13].

The long term recurrence problem is even more impressive when patients undergoing closure only are divided into those with acute perforated ulcers (history less than 3 months, no scarring of the duodenum at surgery) and those with chronic ulcers that have perforated. The re operation rates of 66% and 43% were much higher in chronic perforations than acute ones as reported by Greco et al and Boey et al respectively[14].

Five patients out of seven in this study were followed up for six months without any complication after simple closure. One patient had definitive surgery due to a recurrent ulcer. The other patient who died four days after simple closure was 60 years of age and had reported 3 days after the perforation. All patients were prescribed Cimetidine or Ranitidine after simple plication for six weeks.

H2 receptor antagonist and Omeprazole a proton pump inhibitor are used to heal the ulcer in the early postoperative period in patients with preoperative shock, concurrent medical disease, severe generalized peritonitis or the presence of an intraabdominal abscess. They are also useful for non healing or recurrent ulcers following simple closure. Once healing has occurred, Omeprazole is stopped and H2 receptor antagonist prophylaxis is started as approximately 75% of these ulcers will recur when all medications are discontinued[12].

Definitive Surgery

Boey and coworkers have given the following as indications for definitive surgery during the time of plication of the perforation[5] :

1. Well experienced surgeon.

2. Adequate facilities exist for surgery.

3. The patient's ability to withstand the extra time needed for surgery.

4. Absence of purulent inflammation.

1 (a) Truncal Vagotomy and Drainage

The operation involves division of the anterior and posterior vagal trunks just beneath the oesophageal hiatus to reduce acid and pepsin secretions at the expense of impaired receptive relaxation and diminished antral motility. These undesirable effects lead to significant stasis in the approximately half of the patients subjected to truncal vagotomy alone and for this reason truncal vagotomy is now combined routinely with a procedure to facilitate gastric drainage and prevent stasis[14]. The choice between pyloroplasty and gastro-enterostomy is dictated by the conditions prevailing in the pyloroduodenal area. Where possible the perforation is incorporated in the pyloroplasty but significant stenosis or excessive scarring may mean that simple closure with gastroenterostomy is preferred. In this study, one patient who had recurrent ulcer 3 months after simple closure had truncal vagotomy with gastrojejunostomy as a definitive surgery.

(b) Truncal Vagotomy and Pyloroplasty

This procedure has been in use since its introduction by Douden in 1909[8]. After truncal vagotomy where 3 - 5 cm in length of each major trunk is excised; pyloroplasty is achieved by division of the pyloric ring to provide free drainage. The technique begins with a longitudinal gastroduodenotomy being made through the

anterior perforation followed by excision of a diamond shaped area encompassing the indurated margins of the ulcer. Should there be an associated posterior ulcer, oversewing can be accomplished through the anterior incision. The transverse closure of the gastroduodenotomy is performed using an interrupted one layers closure popularized by Weinberg rather than the traditional two layer closure popularized by Heineke-Mikulicz. The indurated tissues are moved to the superior and inferior corners while the central part is closed using non indurated tissues.

The operative mortality of truncal vagotomy and pyloroplasty for perforated ulcers has been between 0 - 15.4% in four large series reported since 1960[12]. In those series the highest mortality was found in patients with associated medical illnesses. The long term results have been similar to those reported after elective vagotomy and pyloroplasty with a recurrent ulceration rate of 12% to 15%. The advantage of this procedure is that the lesion is removed, pyloric stenosis is avoided, the length of operation is short and the excision of the infected indurated ulcer margins allows healing of normal tissues.

## II. Truncal Vagotomy and Resection

Von Haberer advocated subtotal gastric resection for perforated duodenal ulcers in 1919[8]. This procedure was unpopular until approximately 40 years ago when improvements in anaesthesia, antibiotics and perioperative care appeared. The advantage of this procedure is that it abolishes the hormonal phase of gastric secretions and the rate of recurrent ulceration is decreased in between 8 to 10% as compared to vagotomy and pyloroplasty[12].

The choice of reconstruction after gastric resection (hemigastrectomy) depends on location of the perforation, as well as the extent of duodenal resection, the mobility of the second portion of the duodenum after a Kocher's manoeuvre and the severity of peritoneal inflammation in the subhepatic area. Either a gastroduodenal or a gastrojejunal anastomosis is made. In properly selected patients low mortality rate ranging between 2 and 3.2% have been reported for resection[12,15].

III. Highly Selective Vagotomy

Highly selective vagotomy without a drainage procedure was first used clinically in the late 1960's for intractable duodenal ulcer disease[8]. In 1973, Johnson and associates reported on the first clinical experience with the use of this technique in addition to closure of perforated peptic ulcers[16]. Subsequently, numerous reports have documented the safety and efficacy of this procedure. Sawyers and Herrington had neither operative death nor any recurrent ulcer in 21 patients with perforated duodenal ulcers followed 6 months to 3.5 years after operation[14].

This procedure offers the best alternative with very low mortality rate and side effects. The operation divides individual vagal branches passing to the upper two thirds of the stomach while sparing the vagal supply to the antrum and pylorus so obviating the need for a drainage procedure. This objective is attained by preserving the continuation of anterior and posterior nerves of Latarjet which innervate the distal stomach. The vagal trunks, their hepatic and coeliac branches and nerves of Latarjet are left intact. With preservation of all abdominal vagal innervation except for that supplying acid secretory gastric mucosa, near normal gastric emptying and

extragastric digestive tract functions are maintained. Because the antrum is left intact, it is not surprising that the recurrent ulcer rate is higher than after truncal or selective vagotomy plus antrectomy. The major complication has been ulcer recurrence, as other long term sequelae such as dumping, diarrhoea, bile gastritis, weight loss and malnutrition have been seen in only 3% to 8% of patients[12]. Gastric stasis has been uncommon and most other side effects have been milder than after truncal vagotomy. It has been found that the direct cause of death after this procedure is necrosis and perforation of the lesser curve of the stomach. This carries a mortality of 50% and is most likely due to the direct operative trauma or ischaemia from devascularization[1,15,16].

Highly selective vagotomy has not been found to be efficacious in management of pyloric or prepyloric ulcer[12]. A recurrence rate greater than 30% have been reported. Taylor and coworkers[1] have introduced a modified technique a decade ago consisting of posterior truncal vagotomy with anterior lesser curve seromyotomy of the corpus and fundus. The operation avoids devascularization of the entire lesser curvature of the stomach with achieving similar reduction in acid secretion rates. Several trials have shown good results however more research is needed[1,15,16].

Many patients did not turn up for elective definitive surgery after simple closure and hence there was no patient who had benefited from this procedure.

Several other procedures such as laparoscopic surgery where a combination of posterior truncal vagotomy with an anterior highly selective vagotomy are on evaluation[1]. Nevertheless HSV remains the best elective surgical procedure of choice at present and retains the option of more radical operation should failure occur[1,8].

## CONCLUSION

Out of seven patients treated by simple closure plication, one patient died and three had wound sepsis. The death of this one patient represents a high mortality and may be related to the old age of the patient, delay in reporting to hospital prolonged ileus or re - leak. The high incidence of wound infection may be due to lack of antibiotic cover in the perioperative period. Despite this, simple closure remains a safe procedure even in the hands of experienced surgeon.

Proper selection of patients, adequate fluid and electrolyte resuscitation and perioperative antibiotics may reduce the morbidity. While several surgeons select HSV the best definitive surgical procedure for perforated duodenal ulcer in expert hands, it is recommended that for most general surgeon at the district or regional level, truncal vagotomy and pyloroplasty would be a simpler procedure if definitive surgery is undertaken during the emergency session.

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# CHOLESTATIC JAUNDICE


## SUMMARY:

Eleven patients with cholestatic jaundice were managed in surgical firm I of the Muhimbili Medical Centre in a period of 18 months from January 1992. There were six males and five females with their age ranging between 28 and 81 years. The peak age was 60 - 69 years. The main presenting symptoms were jaundice in 11 patients, right upper abdominal pain 10 patients, pruritis 8 patients, passing dark urine and pale stools, five patients each. Loss of body weight was found in 4 patients with malignancies.

Ultrasonography was used to confirm the presence of an obstruction of the biliary tract. An exploratory laparotomy was performed in all patients. Carcinoma of the head of pancreas, choledocholithiasis and bile duct carcinoma were the commonest causes of cholestatic jaundice in this study. Due to advanced stage of the tumours, the only treatment offered was a bypass surgery to palliate the symptoms through cholecystojejunostomy (6 patients) and choledochojejunostomy (2 patient). Cholecystectomy with bile duct exploration and T tube drainage was done in those patients with choledocholithiasis.


## INTRODUCTION

Cholestasis is failure of bile to reach the duodenum from the site of conjugation of bilirubin in the liver cells[1,2]. Obstruction may be either intrahepatic (involving the finer radicals of biliary tree) or extrahepatic (where there is obstruction of the major bile ducts)[2]. Despite these fundamental differences the

syndrome resulting from cholestasis is essentially similar regardless of the site of obstruction[3,4,5]. The distinction of cholestatic jaundice into intrahepatic or extrahepatic is important to determine the mode of treatment. The common causes of cholestasis are congenital cysts, biliary atresia, stones impacted in the biliary tracts, strictures from infestations, neoplasm of the bile ducts, infection and trauma.

The diagnosis of these conditions depends on history, physical findings and good diagnostic techniques. In recent years wide application of the new imaging techniques has led to improved diagnosis. These include ultrasonography, endoscopic retrograde cholangiopancreatography (ERCP), percutaneous transhepatic cholangiography (PTC) and hepatobiliary imminodiacetic acid (HIDA) scan.

Treatment can be curative or palliative. Complete cure is expected in those causes which are non neoplastic while carcinoma involving the extrahepatic bile duct carries poor prognosis. These tumours are frequently diffuse and sclerotic in nature tending to invade locally and involve the hilum of the liver[5]. As a result the majority of patients are treated by palliative methods. The aim of treatment in cholestatic jaundice is to relieve the jaundice by either removing the obstruction or create a bypass and establish the continuous flow of bile. In this study a typical patient with cholestatic jaundice is presented and a summary of features in the 11 patients is tabulated. The anatomy of extrahepatic biliary system is outlined and literature on the aetiology, diagnosis and management is reviewed.

## CASE REPORT

O. N IP No. 664962 was a male driver aged 50 years, a Tanzanian of

African origin admitted at the Muhimbili Medical Centre as a referral from a district hospital.

He presented with yellow discolouration of the sclera and passing dark urine for two months duration. He had been completely well until two months prior to admission when he noted yellow discolouration of eyes which was progressive. It was associated with passing dark urine. Two weeks after the start of the illness he developed itching of the whole body, which did not respond to medicines prescribed in hospital. His stools had changed to a pale colour and were bulky and offensive. He denied a history of passing blood in stools or passage of black stools. He had vague epigastric pain which was non radiating and had lost significant weight. There was no history of recent injections or blood transfusions. He used to drink two to four litres of alcohol occasionally but had stopped for one year. No family member had a similar illness, diabetes mellitus or hypertension.

Clinical examination revealed a middle aged man with deep jaundice. He was afebrile and had scratch marks all over the body. He was not pale and had no peripheral lymphadenopathy. He had no tremors, spider naevi, caput medusae or palmar erythema. His pulse rate was 72 per minutes regular and of good volume with a blood pressure of 130/90 mmHg. Abdominal examination revealed a soft abdomen with mild tenderness on the right hypochondrium. The liver was palpable 3 cm below the right costal margin along the mid clavicular line. It was smooth with a sharp margin and it was tender. The tip of the spleen was palpable and Murphy's sign was negative. There was no rebound tenderness and bowel sounds were normal. Rectal examination revealed no masses and the examining finger was stained with

pale stools. The respiratory, cardiovascular and central nervous systems were normal. A provisional diagnosis of extrahepatic cholestatic jaundice was made. The results of investigations requested were as follows:

1. Haemoglobin 10.6 g/dl
2. Serum creatinine 92 umol/L
3. Urinary bilirubin was positive.
4. Urobilinogen in urine was negative.
5. Serum bilirubin; Total: 130 mmol/L; Direct: 104 mmol/L.
6. Serum alanine aminotrasferase (ALAT): 561 IU/L.
7. Serum aspartate aminotrasferase (ASAT): 60 IU/L.
8. Serum alkaline phosphatase (ALP): 905 IU/L.
9. Alpha feto protein was negative
10. HBsAg was negative
11. Prothrombin time was 12.5 sec (83%). Control Plasma was 11.5 sec (100%).
12. The bleeding time was 2 minutes, 54 seconds (IVY)
13. The hemogram was normal with adequate platelates and the ESR was 55 mm/ in the first hour.
14. Serum electrolytes: Na 132 mmol/l, K: 4.2 mmol/L were normal.

A plain abdominal x-ray was normal and no gallstones were visualized. Abdominal ultrasound and PTC were not done due to lack of jelly and contrast media respectively. While awaiting surgery the prothrombin time was noted to have prolonged to 16.5 seconds which was 45% of the control. He was given vitamin K,

10 mg intramuscular daily for 7 days. His haemoglobin was noted to have fallen to 8.8 g/dl and he was transfused with three units of fresh blood. Within two weeks his prothrombin time was 12.5 sec (83% of control) with a haemoglobin of 10.9 g/dl.

He was prepared for an exploratory laparotomy. Intravenous infusion was started prior to surgery to provide adequate fluids and glucose. An indwelling catheter was inserted to chart the urine output. Blood was cross-matched for surgery and cephalexim 750 mg IM was given one hour before surgery.

Under general anaesthesia with endotracheal intubation a right subcostal incision was made and the abdomen was opened. A tumour of the head of Pancreas extending to second part of duodenum was seen. The common bile duct was dilated to approximately 2.5 cms in diameter. Lymph nodes were found along the lesser curvature of the stomach. One lymph node was taken for histological examination. The tumour was found to be unresectable and a biopsy from the tumour was taken for histological examination. It was decided to perform a bypass procedure to relieve the jaundice. The jejunum was identified, mobilized at 25 cm from duodeno-jejunal junction and was brought to supraduodenal areas infront of the transverse colon. The common bile duct was cut few centimetres distal to the cystic duct. The proximal stump of the common bile duct was anastomosed to a defunctioned (Roux) loop of the jejunum in two layers using Dexon 3/0. Corrugated rubber drain was left at site of anastomosis and the wound was closed in layers.

Post-operatively the patient was monitored routinely and cefuroxime was given intravenously for 3 days. An indwelling urinary catheter for urine output and nasogastric suction were continued for 3 days. On 6th post operative day the wound

was noted to be discharging pus and the middle two stitches were removed. On 8th post-operative day he was found to be febrile, with an abscess around the wound drain site and leakage of bile at the wound. Incision of the abscess was done and 100 mls of pus was drained. A Foley's catheter was reinserted and remained in situ. Pus was examined for culture and sensitivity which revealed a mixed bacterial growth. He continued dressing the wound with Eusol and measuring daily the bile leakage which was an average of 10 - 15 mls. After two months the fistula closed and the wound healed. He was discharged home, but was lost on follow up.

## TABLE OF RESULT

Table I: Summary of the 11 patients on the basis of Age, Sex and Diagnosis

| Case No | Age | Sex | Diagnosis |
|---|---|---|---|
| 1 | 28 | F | Cholelithiasis and choledocholithiasis |
| 2 | 31 | M | Cholelithiasis and choledocholithiasis |
| 3 | 41 | F | Cholelithiasis and choledocholithiasis |
| 4 | 42 | F | Chronic Pancreatitis |
| 5 | 60 | F | Carcinoma of the gall bladder |
| 6 | 50 | M | Carcinoma of the head of pancreas |
| 7 | 62 | M | Carcinoma of the head of pancreas |
| 8 | 65 | M | Carcinoma of the head of pancreas |
| 9 | 80 | F | Carcinoma of the head of pancreas |
| 10 | 70 | M | Bile duct carcinoma |
| 11 | 81 | M | Bile duct carcinoma |

The majority of patients with carcinoma were above 50 years. Carcinoma of the head of Pancreas and Bile duct were the commonest.

Table II : Presenting Symptoms

| Symptoms | Frequency | Percentage |
|---|---|---|
| Jaundice | 11 | 100 |
| Abdominal pain | 10 | 91 |
| Itching | 8 | 73 |
| Fever/chills | 5 | 45 |
| Dark urine | 5 | 45 |
| Pale, bulky stool | 4 | 36 |
| Weight loss | 4 | 36 |

Jaundice and abdominal pain were the constant symptoms. Weight loss was seen in carcinoma of the head of pancreas and carcinoma of the bile duct.

Table III: Management

| Surgical Procedure | Number | Percentage |
|---|---|---|
| Cholecystojejunostomy | 6 | 54.5 |
| Cholecystectomy + CBD Exploration | 3 | 27.3 |
| Choledochojejunostomy | 2 | 18.2 |
| Total | 11 | 100.0 |

Cholecystojejunostomy was the main modality of palliative procedure for malignant disease.

## DISCUSSION

The anatomy of the biliary system and the related blood vessels has great variation in form, number and position. A good knowledge of the anatomy of the

biliary system and its variation is essential in order to avoid surgical error which may lead to disaster[1,2,6].

Aetiology and Pathology of Cholestasis

Cholestatic jaundice may be intrahepatic or extrahepatic. The clinical picture in both cases is similar[7]. There are numerous causes of cholestasis and some authors suggest classifying them into "Medical" and "Surgical" jaundice, however these terms are generally avoided[1]. The extrahepatic causes of cholestasis according to Decock[2] are; carcinoma of the pancreas, choledocholithiasis, bile duct and ampullary tumours , stricture of the common bile duct, metastatic carcinoma in the lymph nodes at the porta hepatis and pancreatitis. Less common causes are parasitic infestation, sclerosing cholangitis, haemobilia, hepatic abscess and carcinoma of the gall bladder[2].

In this study of 11 patients, it is difficult to make statistical interpretation and conclusion regarding the common etiological factors. However, a five year retrospective study commencing on January 1989, of admission registers of the three Surgical Firms at Muhimbili Medical Centre, the commonest causes of extra hepatic cholestasis were:

(i) Carcinoma of the head of pancreas,

(ii) Cholestasis with cholelithiasis,

(iii) Cholangiocarcinoma of the common bile duct.

Some of these conditions will be considered in more detail, while the rest will only receive brief review.

Clinical features and diagnosis in cholestasis

The clinical presentation of patients with cholestatic jaundice generally includes, jaundice, epigastric pain and itching[1,2,4]. In this study all patients presented with jaundice while 91% presented with abdominal pain and 73% with itching. In one review[8] of 86 patients with cholestatic jaundice, 76% presented with painless jaundice, 68% epigastric pain, and 40% itching. More than half presented with weight loss, generalized malaise, nausea and vomiting.

The pattern of jaundice varies according to the aetiology. A history of intermittent jaundice suggest common bile duct stones; but can also occur in carcinoma of the ampulla of Vater, increasing as the tumour blocks bile flow with relief as the tumour sloughs off due to necrosis[1,2,10]. Progressive jaundice is suggestive of malignancy[1].

Abdominal pain arising from the hepatobiliary system usually occurs in the right upper quadrant or epigastrium and is referred to the right scapula. Pain due to stones in the common bile duct is intermittent and severe where as the pain due to malignancies is dull and at times radiates to the back. Weight loss suggests malignant disease of the pancreas or bile duct and the presence of fever accompanied by chills suggests cholangitis. Tompkins and Saunders[9] in their analysis of 186 patients with bile duct cancers noted that weight loss and pruritis occurred in 50% of patients while fever and chills were noted in 17% of patients. Forty five percent of the patients in this study presented with fever and chills suggesting cholangitis.

Pruritis is often attributed to bile salts deposition in the skin although several experiments done have never proved this[8]. Pale , offensive, bulky stools are

characteristics of cholestatic jaundice[7]. Bile salts are important for absorption of fat and steatorrhoea is proportional to depth of the jaundice[4]. Blimgart[10] in a series of 94 patients with extrahepatic cholestasis due to bile duct noted that 40% of patients presented with pale, offensive, bulky stools. In this study 36% of all patients presented with pale, offensive, bulky stools.

Physical findings in patients with extrahepatic cholestasis include jaundice of variable depth, lymphadenopathy as an indication of metastasis, scratch marks on the skin suggesting cholestasis and wasting which indicates the presence of malignancy.

**INVESTIGATION**

I. Urine Examination

The finding of bilirubinuria in a jaundiced patient suggests that the jaundice is due to the appearance of conjugated bilirubin in the plasma resulting from hepatocellular damage or hepatic obstruction[4]. Bilirubin secreted by the liver into bile is reduced by intestinal bacteria to urobilinogen which is reabsorbed into the portal circulation (enterohepatic circulation). A small amount reaches the systemic circulation and is excreted into the urine. The normal daily excretion in the urine is 0.0 to 4.0 mg per 24 hours; however, altered function of the liver, intestinal bacteria, absorption and renal excretion alter the level of urobilinogen[2]. Rosoff in his study reported the presence of urobilinogen in urine in 0.5 - 5% of patients with cholestatic jaundice[7].

2. Blood Biochemistry

Serum total bilirubin is raised in all forms of jaundice. Raised bilirubin generally indicates hepatocellular disease or cholestasis. In cholestasis the direct

reacting type is usually raised. A fluctuating level suggests biliary stones or tumour of the Ampulla of Vater[4]. When cholestasis is relieved, serum bilirubin falls slowly to normal[6].

Alkaline phosphatases are serum enzymes released by all forms of rapidly multiplying cells especially biliary canaliculi and sinusoidal membranes. Tompkins et al suggest a raised level of alkaliné phosphatase more than three times the upper limit of normal to indicate cholestasis[9]. Sharlock et al proposed a rise of alkaline phosphatase more than 400 iu/l to be most likely due to malignancy[7]. Serum albumin and globulin levels are normal in acute cholestasis. With the development of biliary cirrhosis, serum albumin tends to fall with inversion of albumin/globulin ratio.

Aminotransferases (ALAT and ASAT) are intracellular liver enzymes. Increased serum level is an indicator of current cell damage. Non continuous damage of hepatocytes leads to normal levels within 24 to 48 hours. They are less reliable tests in cholestatic jaundice[2,4]. Alpha feto protein is synthesized by liver cells but may also appear in germ cell tumour and other tumours hence it is a non specific index[4, 7].

3. Haematological Investigation

Most of the coagulation factors are synthesized in the liver. Thus the prothrombin time is prolonged in long standing liver disease due to decrease in factor II, VII, IX and X. These are vitamin K dependent clotting factors[1,2,4]. Vit K absorption is decreased in cholestasis and improvement of prothrombin time after vitamin K injection indicates cholestasis rather than hepatocellular dysfunction[2].

4. Abdominal Ultrasound

This is an initial imaging examination technique which is non invasive and free from risks. It may define the presence of gall bladder stones, dilated biliary tree and abnormalities in the pancreas such as pancreatic cancer[6,9]. Ultrasound however is not a good test for defining calculi in the common bile duct although they can occasionally be demonstrated[11]. Gronmen and Jeffrey in different studies of patients with cholelithiasis reported that ultrasound detected stones in the CBD in 13% of 87 patients and 30% of 53 patients respectively[11]. The patients in this study with stones in the CBD were detected by ultra sound. The inability to visualise the whole gland limits the sensitivity of ultrasound in the diagnosis of pancreatic cancer, although pancreatic tumours greater than 2cm in diameter may be visible[9,12].

5. Percutaneous Transhepatic Cholangiography (PTC)

PTC using the Chiba needle has gained acceptance in the last two decades[2,12]. The investigation should not be undertaken if the patient has a prolonged prothrombin time. A history of cholangitis warrants the use of antibiotic before the procedure. It is the diagnostic procedure of choice when proximal lesions are suspected as it allows better visualization of the proximal biliary tract and proximal extension of a tumour if present[12]. Under fluoroscopy control, a needle (the Chiba Okuda needle) 15 cm long and 0.7 cm in diameter is advanced into the liver through the eighth intercostal space in the mid-axillary line. The stylet is then removed and whilst injecting the medium meglumine iothalamate (60% w/v), the needle is slowly withdrawn until contrast is seen to enter a biliary radical. Tompkins[9] has advocated an extension of the technique in order to place a catheter in the bile ducts so as to

provide external biliary drainage in patients with biliary strictures.

Johnson et al in their series of 572 patients diagnosed 63% of patients with proximal biliary tract neoplasms which is an accuracy that compares favourably with that of Ultrasound which has been shown to diagnose 68% of cases[11]. The procedure has side effects of infection, haemorrhage and bile leakage. Bile is routinely sent for culture and sensitivity after each procedure.

6. Endoscopic Retrograde Cholangiopancreatography (ERCP)

This is a procedure where the duodenal papilla is cannulated with the aid of the fibreoptic duodenoscope and the bile duct and pancreatic duct are visualized after injecting a water soluble contrast. According to Shea[12] the procedure allows the physician also to assess the stomach and duodenum and if there is a malignant lesion a biopsy can be taken. Camloke[7] in his study, found that it is highly accurate in identifying gallstones, strictures and pancreatic carcinoma with sensitivity and specificity rates of 60 - 90% respectively.

In comparing ERCP and PTC, Rothshield[8] found the following complications: haemorrhage 4%; sepsis 2%; bile leakage 1% and pancreatitis 9% while with PTC, the complication rates were: haemorrhage 80%; sepsis 13%; bile leakage 1% and pancreatitis 1%. The use of antibiotics and expeditors surgery have reduced the mortality rate to less than 1%[12].

7. Computerised Tomography (CT SCAN)

This provides similar information as ultrasonography; however it is more informative[1,2,4]. It will delineate dilated bile ducts, define lesions in the pancreatic head and detect metastatic lesions in the liver and stomach. It is useful for those

patients in whom ultrasonography is difficulty e.g. obese patients or those with excessive bowel gas[5].

8. Hepatobiliary Imidoacetic Acid Scan (HIDA)

Within the past decade the HIDA scan has become an important method for investigating cases of right upper quadrant abdominal pain[12]. The biliary system is the normal route for excretion of lipid soluble compounds. Derivatives of Imminodiacetic acid can be chelated with Technetium 99 to form a complex with a high molecular weight. This substance can be substituted with side chain to increase its lipid solubility. These agents are extracted by the liver from both the hepatic arterial and portal venous blood. They are actively transported across hepatocytes bond to protein and secreted into the biliary canaliculi and excreted into the duodenum through the biliary tree. The extraction and secretion of these agents by the liver fail to occur in biliary obstruction. Dilated ducts can be seen by HIDA scan, however ultrasound is more accurate[9,12].

The commonest clinical condition seen in Muhimbili Medical Centre.

The following are three common clinical conditions which contributed to cholestatic jaundice in this study;

1. Carcinoma of the head of Pancreas (4 patients).
2. Choledocholithiasis (3 patients).
3. Carcinoma of the bile duct (2 patients).

1. Carcinoma of the head of Pancreas

It is one of the commonest cause of extrahepatic cholestasis[3,13]. It was also the

leading cause in this small study.

Carcinoma of the head of the pancreas is associated with early compression of the common bile duct leading to dilatation of the upper biliary tract and distension of the gall bladder. Duodenal obstruction may occur and local spread results into involvement of the portal vein and inferior vena cava. Hepatic metastasis occur and retroperitoneal lymph nodes are involved[3].

As in other series, the age at presentation in this study was between 60 and 80 years and males were more commonly affected than females. The commonest symptom was loss of body weight and has also been related to malabsorption. It is reported to occur in 60 - 80% of patients with carcinoma of the head of pancreas[2]. All patients in this study presented with progressive jaundice. All four patients with carcinoma of the head of pancreas had upper abdominal pain which was dull and radiating to the back. Pruritis is a frequent symptom.

Bradley and Zeppa have reported that only 25% of tense distended gall bladder are palpable in tumour of the head of pancreas[2]. In this study none of the patients had palpable gall bladder. Urine may be dark and stool pale and offensive. Other manifestations are diarrhoea, nausea and vomiting.

The only potentially curative treatment is pancreatico duodenal resection of the type described by Whipple. The procedure involves enbloc resection of the head and neck of the pancreas, duodenum, antrum of the stomach, upper jejunum, gall bladder, common bile duct and associated lymph nodes. The procedure has a high morbidity and mortality rates[2,13,14,15].

Palliative cholecystojejunostomy is done to relieve jaundice and pruritis.

Often a gastrojejunostomy is added to prevent late gastric retention should duodenal obstruction occur. Longmire[13] has advocated that a good palliative procedure should relieve jaundice as well as gastric outlet obstruction. All the four patients in this study with carcinoma of the head of pancreas had relief of jaundice after cholecystojejunostomy. Griaco[3] has reported the procedure to be effective, quick and easy with a mortality of 4-5%. The addition of chemotherapy using 5 Flurouracil and Mitomycin C is still debatable[2]. None of the patients in this study had chemotherapy.

2. Choledocholithiasis

In most cases stones in the bile duct migrate from the gall bladder and are found in 8 - 16% of patients with cholelithiasis[4]. Most of these secondary stones are cholesterol in keeping with their high incidence in the gall bladder[16]. Primary common bile duct stones are usually composed of calcium bilirubinate and are always associated with bile duct obstruction[2]. Most stones result from minor alteration in chemical composition of bile. Cholesterol is insoluble in water but held in solution in bile by detergent action of bile salts. Bile salts and phospholipids form a soluble physical state of mixed micelles with cholesterol molecules. A change in concentration of either bile salts, phospholipids or cholesterol would lead to supersaturation of bile with cholesterol. Cholesterol crystallizes and forms a nidus for stone formation. Other factors which predispose to stone formation are biliary tract infection, bile stasis and haemolytic diseases[2].

The aetiology of calcium bilirubinate stones is thought to be due to

precipitation of unconjugated bilirubin with calcium. These stones are ovoid brownish and tend to crumble easily as compared to solitary and round appearance of cholesterol stones. The clinical settings in which they occur include post traumatic biliary strictures, a narrowed biliary enteric anastomosis, stenosis of sphincter of oddi, sclerosing cholangitis and oriental cholangiohepatitis[2]. Primary duct stones are commonly encountered in the tropics where they have been found to occur secondary to biliary tree infestation by ascaris lumbricoides and clonorchis sinensis[15]. All stones found in this study physically appeared to be mixed stones.

Common bile duct stones may be asymptomatic or cause biliary colic, bile duct obstruction, cholangitis or pancreatitis where the patient present with pain, fever and jaundice. The pain may be mild or severe and can not be differentiated from pain arising from the gall bladder. The jaundice may be intermittent if the obstruction is partial and intermittent or progressive if a stone becomes impacted in the distal duct[4,7]. The jaundice is obstructive in type and hence the urine is dark, the stools are pale and there is associated pruritis. Fever and rigors indicate acute cholangitis. The three patients studied presented with progressive jaundice and right upper abdominal pain. None of the patient presented with fever. The early symptoms of choledocholithiasis cannot be distinguished from acute cholecystitis[2,4].

Ultrasonography should be done to detect the presence or absence of gall stone and dilatation of bile ducts. ERCP is indicated for most patients who have bile duct obstruction as manifested by persistence of jaundice or bile duct dilatation as evidenced in ultrasonography. PTC is an alternative, but ERCP allows performance of endoscopic papillotomy and extraction of stones when indicated[16]. The surgical

treatment for common bile duct obstruction by stone is surgical removal after a build up of liver glycogen by a high intake of glucose, correction of clotting abnormality by vitamin $K_1$, fluid and electrolyte balance and antibiotics[1,12].

There are various surgical procedures for removal of stones. Endoscopic papillotomy is a procedure preferred in patients with operation or who had previous cholecystectomy. After ERCP, a diathermy sphincterotome is passed through the endoscope and the papilla of Vater is divided. Then using a Dormia basket or balloon catheter which is passed through the bile duct, stones are extracted.

Operative choledochotomy is another procedure where stones are manoeuvred in a good position. The CBD is opened longitudinally directly over the stone or when one is not felt; a 2cm incision over the common bile duct is made and stones removed by the use of a malleable scoop Desjardins gallstone forceps or balloon catheter may be used to remove and irrigate the duct[2,16]. Choledochoscopy may be employed to confirm that all calculi have been removed[12]. After the common duct is explored, a T tube is inserted prior to closure and drainage is maintained for approximately one week[2]. At this time a T tube cholangiogram is performed in order to demonstrate patency of the duct and absence of retained stones. If these criteria are met, the T tube can then be removed. Intraoperatively, if there is obstruction of the distal duct transduodenal sphincteroplasty and removal of the stone may be carried out[2,4]. Three patients in this study had cholecystectomy and choledocholithotomy.

Rothshield[14] has advocated the use of chemical dissolution especially for the highly risk patients such as the elderly, obese and those with renal or cardiac

disease. Methyl tert-butyl ether (MTBE) and monoctanoin are effective in dissolving cholesterol stones. MTBE is administered via 5 Fr percutaneous transhepatic catheter placed in bile duct while monoctanoin is given as an intraductal infusion through a nasobiliary catheter[13,16].

The use of extracorporeal shock wave lithotripsy which causes fragmentation of stones through mechanical stresses is recommended by some[14].

3. Bile duct carcinoma

The tumour occurs more frequently in males and has the highest incidence in the sixth and seventh decade[1,2,4]. The two patients seen in this study were both male and were 70 and 81 years old.

Carcinoma involving the extrahepatic bile duct carries a poor prognosis with fewer than 30 percent of patients surviving more than 5 years even in selected groups[17]. The tumour is frequently diffuse and sclerotic in nature tending to invade locally and involve the hilum of the liver. This lesion has an average autopsy incidence of approximately 0-3 percent[2,18].

Tumours of the bile duct have been classified as suggested by Longmire into three; proximal tumours above the junction of cystic duct with hepatic duct comprising 56%; the mid duct tumours which occurs between this junction and the pancreatic portion of the common bile duct forming 26% while intrapancreatic common bile duct tumours comprise 18%. In this study the two tumours were found in the middle and distal part of the common bile duct.

The clinical presentation of patients with biliary tract cancer depend on the

site of the tumour and its size. If the tumour causes obstruction of the hepatic duct birfucation or occur in extrahepatic biliary tract, patient present with obstructive jaundice. If the obstruction involves a single right or left hepatic duct, jaundice may be present or absent and a few patients may have cholangitis[2,14]. Abdominal pain occur in half of the patients while pruritis occur in those with obstruction. Weight loss is the most prominent feature. The liver is palpable in 50% of patients and is non tender while the gall bladder is palpable in one third of patients with distal lesion[1,2,4]. The two patients presented had upper quadrant abdominal pain, jaundice and pruritis. According to Guthrie at al jaundice is absent in 2 - 10% of cases at the time of diagnosis of bile duct cancer and many patients admit to long standing abdominal pain or dyspepsia[5].

Ultrasonography is accepted as the initial imaging procedure in jaundiced patients. It may demonstrate bile duct dilation and give an indication of tumour extent or even resectability. More detailed information on the level of obstruction can be obtained by PTC or ERCP. PTC allows visualization of the distal part including the duodenum, the ampullar area and tissue for biopsy can be taken. Good quality computerized tomography (CT) may obviate the need for PTC or ERCP by demonstrating the level of obstruction and involvement of the caudate lobe. It may determine the presence of metastatic invasion of the liver[2,5,6]. The two patients in this study who had bile duct carcinomas were diagnosed only at laparotomy.

The treatment of patients with carcinoma of the bile duct is tailored to each patient depending on location and extent of the tumour, operative risks and surgeons experience; however they are either conservative or operative.

Therapy for proximal tumour is resection if it does not extend beyond the segmental intrahepatic ducts of both right and left lobes, if the hepatic artery proper and portal vein are free of invasion; if the distal extension of the tumour is limited to the common bile ducts above its pancreatic portion and if no metastatic disease is present. Proximal tumours can therefore be resected either by right extended hepatectomy if the tumour involves the segmental right ductal system with preservation of left ductal system or by left hepatectomy when the left intrahepatic segmental bile duct are involved with preservation of the right ductal system[19]. Tumours of the hepatic birfucation that do not extend proximal to the segmental ducts can be removed by skeletonization and resection with or without multiple hepatic jejunostomy and reconstruction is performed using a Roux en-Y loop started preferably with transhepatic tube[19,20]. The resectability rate for hilar cholangiocarcinoma remains low at 15-20 percent even in specialized centres with an interest in this condition and an operative mortality rate of between 0 to 27% is reported[2,4].

When resection is not feasible, biliary decompression may be achieved by surgical bypass method which include cholangiojejunostomy, hepaticojejunostomy and intubation using tubes modified for hepatic drainage. There is still a debate whether bypass surgery is superior to non operative stenting[20]. Liver transplantation has been associated with high recurrence rate[17].

For mid duct tumours which are resectable the procedure mostly used is pancreaticoduodenectomy[20]. Tumours of the distal duct, can be removed by Whipples operation with or without pyloric preservation[13]. Good palliation for mid duct and

distal duct tumours is by hepaticojejunostomy proximal to the site of tumour. The two patients with bile duct cancer were palliated by cholecystojejunostomy and choledochojejunostomy respectively.

## CONCLUSION

Cholestatic jaundice is a common surgical condition which is challenging both as far as investigations and management are concerned. The availability of ultrasound, PTC and ERCP in the advanced centres has improved the diagnostic accuracy to over 90%. In developing countries these tests are either not available or there are few experts to perform these investigations. In this centre facilities for PTC and ERCP are greatly needed. These will facilitate early, accurate diagnosis and management of patients with jaundice.

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# ACUTE APPENDICITIS


## SUMMARY

Acute appendicitis is a common cause of emergency surgical admissions at Muhimbili Medical Centre. Out of 250 emergency operations performed in firm I, in the Department of Surgery at the Muhimbili Medical Centre between January 1992 and June 1993, 60 cases were for acute appendicitis. Of these 60 patients, 20 were studied in details and their findings are presented.

There were 12 males and 8 females with a male to female ratio of 3 : 2. Their age ranged from 14 to 39 years with a peak age between 21 - 30 years. While anorexia and nausea were seen in more than 80 percent of the cases, the classical paraumbilical pain shifting to the right iliac fossa and nausea were seen in 60 percent of the cases. The duration of symptoms prior to presentation to hospital ranged from ten hours to six days.

Three of the 20 patients had an appendicular mass and were treated conservatively first and eventually operated after three months. The remaining 17 were operated as emergencies. The appendix was confirmed to be inflamed in all patients. The hospital stay varied between 3 - 5 days and was longer when complications occurred. Wound infection was the only postoperative complication and occurred in 6 patients (33%).


## INTRODUCTION

Appendicitis refers to inflammation of the vermiform appendix and was first used by Reginald Fitz in 1886 to describe a condition previously thought to be of

caecal origin, known as typhlitis and perityphilitis[1,2]. The first surgeon to correctly diagnose and perform an appendicectomy was Sent in 1889[2]. In the same year Mc Burney described the clinical findings of acute appendicitis prior to rupture, including a description of the point of maximal abdominal tenderness and the incision that is known by his name[3].

Acute appendicitis is considered as a disease of industrialized societies and a rarity among the traditional societies[1,2,4]. However, it has been observed that in Africa and other developing countries as people move into cities and adopt an urban way of life, the incidence of acute appendicitis rises[5]. The primary reason for this appears to be a low residue Western diet with a long intestinal transit time leading to a viscid tenacious stool of high viscosity which cause increased intraluminal pressure in the colon and appendix, mucosa necrosis and infection[1,2,5].

**CASE REPORT**

IP NO 750319 a 20 years old male from Dar es salaam, was admitted with chief complaints of abdominal pain, nausea, vomiting and fever. His illness had stated 18 hours prior to admission with pain in the paraumbilical region. The pain was vague continuous and dull but associated with occasional colic. It was followed a few weeks later by nausea and vomiting. He vomited once and the vomitus was clear. After about 6 hours, the pain shifted to the right iliac fossa. It became very severe and any movement aggravated the pain. He also complained of low grade fever. There was no history of constipation or diarrhoea and his micturition was normal .

On examination the patient was ill looking lying quietly in bed. His

nutritional status was good; with no pallor or jaundice. He had a temperature of 38.5 $^{0}$ centigrade, a pulse rate of 96/min regular and the blood pressure was 130/80 mm Hg. The abdomen was not distended and moving with respiration. Palpation of the abdomen revealed marked tenderness in the right iliac fossa. The rest of the abdomen was soft and non tender. Rovsing's sign and the obturator test were positive while psoas test was negative. Bowel sounds were normal. Digital rectal examination revealed marked tenderness in the right side of the rectovesical pouch. The respiratory, cardiovascular and central nervous system were essentially normal. A provisional diagnosis of acute appendicitis was made and the patient was prepared for emergency appendicectomy. An intravenous infusion of normal saline was established and metronidazole 500 mg was given intravenously. Two hours after admission he had been given two litres of normal saline and was taken to theatre.

Under general anaesthesia and endotracheal intubation the abdomen was opened through a right grid iron incision. A slight turbid but odourless fluid gushed out as the peritoneum was opened and was sucked away. The caecum was easily identified and the taenia coli were followed downwards until the base of the appendix which was in the pelvic position. It was dull red in colour and swollen. The appendix was delivered at the wound, isolated from the peritoneal cavity by abdominal packs and held up by means of Babcock forceps. The messoappendix was clamped divided and transfixing ligatures of silk 2/0 applied. Using an artery forceps the base of the appendix was crushed and tied with chromic catgut 2/0. The artery forceps was applied a little more distally, a swab was placed beneath the appendix and divided. The appendix, the swab and knife were placed in a kidney

dish and removed from the operation field. The stump was not buried. The abdominal incision was closed in layers except the skin and the appendix was sent for histology.

Post operatively, 2,500 mls of dextrose saline were given intravenously in the first 24 hours and intravenous metronidazole 500 mg eight hourly was continued for 72 hours. Blood pressure, pulse and respiration were monitored hourly and temperature eight hourly for 24 hours and then twice daily until discharge. After 48 hours, the patient was allowed oral fluids and mobilized. He was discharged home on the 5th post operative day and the stitches were removed at the 7th post operative day. He was seen twice at 3 monthly intervals at the outpatient clinic and found to be doing well. The histology of the removed appendix showed infiltration of the whole thickness of the appendix with polymorphonuclear neutrophils and exudate formation.

## ANALYSIS OF THE 20 PATIENTS WITH ACUTE APPENDICITIS

Table I: Age and sex distribution

| Age Group (Years) | Number of patients | | TOTAL | % |
|---|---|---|---|---|
| | Male | Female | | |
| 11 - 20 | 2 | 4 | 6 | 30.0 |
| 21 - 30 | 10 | 3 | 13 | 65.0 |
| 31 - 40 | 0 | 1 | 1 | 5.0 |
| Total | 12 | 8 | 20 | 100.0 |

The majority of the patients were in their 3rd decade of life with male over female predominance.

Table II: Presenting symptoms

| Symptoms | Number of Patients |
|---|---|
| Anorexia | 18 |
| Nausea | 16 |
| Paraumbilical pain shifting to RIF | 12 |
| Vomiting | 12 |
| Right iliac fossa pain(RIF) | 10 |
| Fever | 8 |
| Constipation | 4 |
| Diarrhoea | 3 |

Anorexia and nausea were common presenting symptoms in over 80 percent of the patients while paraumbilical pain shifting to RIF was seen in 60% of patients.

Table III: Duration of Symptoms

| Duration in days | Number of patients | % |
|---|---|---|
| Less than 1 | 4 | 20 |
| 1 - 2 | 6 | 30 |
| 3 - 4 | 8 | 40 |
| 5 - 6 | 1 | 5 |
| 7 - 8 | 1 | 5 |
| Total | 20 | 100 |

Eighty percent of the patients reported to hospital more than 24 hours after the onset of illness.

Table IV: Type of incision

| Incision | Number of patients | | Total | % |
|---|---|---|---|---|
| | Male | Female | | |
| Gridiron | 6 | 6 | 12 | 75.00 |
| Right Paramedian | 2 | 1 | 3 | 18.75 |
| Rutherford Morrison | 0 | 1 | 1 | 6.25 |
| Total | 8 | 8 | 16 | 100 |

Seventy five percent of the patients had classical Gridiron incision at Mc Burney's

point while 18.75% of the patients with equivocal symptoms had right paramedian incisions.

Table V: Operative findings

| Findings | Number of patient | % |
|---|---|---|
| Gangrenous perforated appendix<br>Simple catarrh appendicitis (Mucocele)<br>Appendicular mass | 10<br>7<br>3 | 50<br>35<br>15 |
| TOTAL | 20 | 100 |

Fifty percent of the patients had gangrenous appendix with the perforation on admission.

Table VI: Flora cultured from cases of Perforated appendicitis

| Organisms cultured | Number of patients | % |
|---|---|---|
| E. coli | 6 | 60 |
| Klebsiella | 2 | 20 |
| Enterobactor | 0 | 0 |
| Mixed growth | 2 | 20 |

N.B Anaerobic cultures were not done. The flora in the pus was predominantly of the enterobactericcae.

## DISCUSSION

Prevalence

Acute appendicitis is predominantly a disease of Western civilization and is uncommon in Africa and Asia[5]. The disease is related to life style, especially diet.

In fact, even in Europe it was rare in the last century and has increased with a rise in socio economic status. In Western countries it is the commonest cause of acute abdomen[1,2]. In African countries the condition is becoming more common because of their adoption of low residue Western style diet especially in urban areas[5].

At Mulago Hospital in Uganda 23 cases of appendicitis were admitted in 1964[6] and there was evidence of steady increase in 1970's . In Kenya there were over 100 cases in 1964 and this formed the major abdominal emergency[6] . Kakande has however reported acute appendicitis as contributing 41 percent of hospital emergencies in Nyeri Kenya, in 1990[7]. In Lusaka Zambia acute appendicitis accounted for 16.8% of admissions for 'acute abdomen ' over one year[5]. In an analysis of surgical admissions to Muhimbili Medical Centre in 1971, Hiza reported that acute appendicitis was quite common and affected 119 patients out of 1812 surgical admissions in that year[8]. In this study out of 250 emergency surgical operations performed, 60 cases (24%) were due to acute appendicitis in one of the surgical firms over a period of 18 months.

Acute appendicitis occurs at all ages, but is commonest in the second and third decades[1,2,3]. Its rarity in the very young is probably because the lumen is so narrow that obstruction is unlikely[3,5]. The peak age in this study was between 21 - 30 years which conforms with other studies elsewhere[3,5]. Most reports indicate that acute appendicitis is commoner in males than females[3,5]. Out of 20 patients studied 12 were male and 8 were females; showing a male preponderance. Arbjorson had postulated that female sex hormones play a protective role in acute appendicitis after puberty[9].

Aetiology

Theories on the aetiology of acute appendicitis include dietary, environmental and genetic factors[1,2,3]. According to Burkitt, the essence of the dietary factor is a change from high to low residue diet which comes with excessive refining of cereals[3,10]. This was proposed by Rendle Short in 1920 and Burkitt added to it the increase in sugar consumption which goes with improvement in socio-economic status. A high protein/low residue diet produce viscid tenacious stools which have a long transit time especially in the colon. The high viscosity of the stool of this low residue diet causes increased intraluminal pressure, delaying the emptying of the appendix lumen. This increases the chance of pathogenic bacteria multiplying leading to inflammation[3].

Wangensteen and coworkers demonstrated that obstruction to the appendix lumen predispose to the development of acute appendicitis[2]. Obstruction, usually in the form of faecalith and less commonly a calculus tumour or ball of worms (oxyuris enterobius vermicularis) can be demonstrated in 50 to 80% of inflamed appendices[1,2]. With obstruction, there is increased intraluminal secretion of mucinous fluids which cause increased intraluminal pressure causing eventually collapse of the veins of drainage. Ischaemic injury then favours bacteria invasion to add an element of inflammatory oedema and exudation.

Pathology

It is believed that increased intraluminal pressure brought about by luminal obstruction is important in the pathogenesis of acute appendicitis[3]. Following obstruction, there is increased intraluminal mucinous secretions with a build up of

intraluminal pressure which leads to occlusion of the vascular supply. Initially the arterioles continue to fill resulting into vascular congestion and oedema. Thrombosis and inflammation develop resulting in inflammation and eventually gangrene. Once the peritoneum covering the appendix is inflamed, the parietal peritoneum is involved causing the pain to be localized in the right iliac fossa. As time passes the appendix will perforate through autolysis of a gangrenous foci and peritonitis occurs. Loops of bowel and omentum can "wall off" the inflammation resulting into an appendicular mass and later an abscess is formed. These complications are commoner in children and the elderly[1,2].

A build up of pressure of 5 - 15 cm of water is sufficient to cause vascular congestion and ischaemia in the appendicular wall within 6 - 12 hours. It is unusual for the appendix to perforate within 12 hours of onset of inflammation[1,11]. However, Field has reported appendicitis in infants in which the appendix perforates within 8 hours of the onset of inflammation[1]. The ingestion of purgatives or an enema tends to hasten gangrene and perforation of the inflamed appendix. Early perforation is also common in the elderly. Schwartz has explained the mechanism to be similar to that which occur in an incarcerated hernia and being basically due to poor blood supply[2].

Clinical features and Diagnosis

Acute appendicitis has been reported by various authors as the most common cause of acute abdomen[1,2,4,5]. Despite the high frequency with which the condition is encountered, the diagnosis of acute appendicitis in some cases can be challenging.

In the classical presentation of acute appendicitis, the prime symptom is

diffuse paraumbilical pain which shifts to the right lower abdomen within 12 hours usually four to six hours. In some patients the pain begins and remains in the right lower abdomen[2]. In this series of 20 patients, the classical presentation of pain was found in 60 percent of the patients while the atypical pain localized in right iliac fossa was seen in 35 percent. In a series of 55 patients, Udeh reported a corresponding figure of 60% while in 20% the pain started in the right lower abdomen[12]. The initial pain is visceral due to distension of the appendix and is always paraumbilical. The final pain is somatic which is localised in the right lower abdomen. Anorexia is an important symptom in acute appendicitis so much so that its absence should cast serious doubt on the diagnosis of acute appendicitis[1,2]. Several studies have also reported that three quarters of patients with acute appendicitis present with vomiting, usually not more than once or twice[1,2,4,13]. These three important symptoms were seen in this study in the following order of frequency; anorexia (90%), vomiting (80%) and paraumbilical abdominal pain (60%). Other symptoms include fever, constipation, diarrhoea, abdominal distension and dysuria[7]. While fever was observed in 40 percent of patients, constipation and diarrhoea were found in less than 25 percent of patients in this study. It has been observed that tenesmus and diarrhoea occur when the appendix is localized in the pelvis. Diarrhoea quite often indicates irritation of bowel; a sign of perforation[1]. Jaundice is seen only if there is severe portal pyaemia a complication which is indeed rare[11].

The physical signs depend on the anatomical position of the inflamed appendix and whether any complication has occurred[2]. Vital signs are not changed very much by uncomplicated appendicitis. Temperature elevation is rarely more than

$1^0$C and the pulse rate is normal or slightly elevated. Changes of greater magnitude usually mean that complications have occurred or that another diagnosis should be considered. The classical maximal tenderness at MC Burneys point is only to be expected if the appendix is on preileal position. A positive Rovsing's sign is another indicator of inflammation in the right iliac fossa, as are direct and referred rebound tenderness. Early, in the course there may be cutaneous hyperaesthesia on the right side in the area supplied by T 10,11,and 12. Muscle rigidity is at first voluntary, later on reflex guarding. The classical signs of tenderness in the right iliac fossa, guarding and cutaneous paraesthesia in the Sheren's triangle and presence of psoas and obturator signs were found in 80% of the patients in this study. A positive psoas sign and obturator sign each indicates inflammation adjacent to the muscle[1,13].

Some studies have shown that 20 percent of patients with acute appendicitis have microscopic pyuria and haematuria especially if the appendix was abnormally located[3]. Albuminuria is reported in 20% of patients; but when this is combined with 20 WBC or 30 RBCs per field in a centrifuged specimen, urinary tract infection is a more likely diagnosis[1,2]. Leucocytosis is commonly present but the degree does not correlate with severity of the disease. It could be absent in 4 percent of young and very old people. Moderate leucocytosis ranging from about 10,000 to 18,000/mm$^3$ and accompanied by a moderate polymorphonuclear predominance is the rule in acute uncomplicated appendicitis. With normal total and differential white blood cell counts, the diagnosis of appendicitis is in question though not ruled out. If the white cell count is greater than about 18,000/mm$^3$; perforated appendicitis on an acute inflammatory disease of greater magnitude is probable[11]. Although it should

be possible to perform the above test in emergency, at the Muhimbili Medical Centre none of the test results were available early enough to be useful.

Radiological investigation is a useful diagnostic tool when other investigations are non conclusive [5,8]. The plain abdominal X-ray in acute appendicitis may show loops of distended bowel in the right lower quadrant, air fluid levels, altered right psoas shadow, and gas filled appendix. The use of laparoscope, ultrasonography and computed tomogram for patients with indefinite findings and when diagnosis is obscure is being more commonly used for diagnostic purposes[5]. Special problems of diagnosis occur mostly to the paediatric, geriatric and pregnant women. The presentation is atypical and the rate of perforation with peritonitis is high[11,14]. In infants less than one year old, perforation is always present while for children of less than two years old it occurs up to 70 - 80 percent of cases[1,14]. In adults the rate of perforation is reported to be 20 percent[1]. This is in conformity with the findings in this study in which perforations occurred in 5 out of 20 patients. In these patients gross signs of diffuse peritonitis are found and the diagnosis is made intraoperatively.

During pregnancy the disease is infrequently encountered. Several studies have shown that when appendicitis does occur, it is more common in the third trimester. During this period, the enlarged, frequently contracting uterus displaces the omentum. The position of the appendix is also disturbed; frequently displaced cranially. This factor attributes to the development of peritonitis and leads to late diagnosis[1,2,9]. In young males acute appendicitis is to be differentiated from right ureteric colic, acute regional enteritis, torsion of the testis and acute epididymides.

In adults, the differentials are acute cholecystitis, pancreatitis and perforated duodenal or gastric ulcer[3,5]. Women in child bearing age may present with mittelschmerz, endometriosis, torsion of an ovarian cyst or ectopic pregnancy. In children, ilea-caecal syndrome in leukaemic patients is difficult to differentiate from appendicitis[1,2].

## TREATMENT

In the early days,lack of antibiotics and poor perioperative care made conservative management popular because surgery was associated with high mortality rate and severe morbidity[1]. Nowadays, early surgery is well accepted as the best approach whenever the disease is suspected. The merits of early operative intervention were well documented by Mc Burney who had excellent results with the method[4] .

Surgical management is started with preoperative care which include; fluid and electrolyte balance by intravenous infusion using crystalloid, 5% Dextrose saline or Ringers lactate. Salicylate are prescribed in case of high fever. Prophylactic administration of antibiotics is given preoperatively to reduce the incidence of sepsis and operative mortality. A single large dose of antibiotics appropriate for the predicted bacterial flora, and administered intravenously on induction of anaesthesia is most convenient and effective method recommended nowadays. Since the bacterial flora customarily found in acute appendicitis is a mixed colonic flora with both aerobic and anaerobic organisms; metronidazole alone or in combination with aminoglycosides like gentamycin has good record for success[15]. In heavily

contaminated surgery and emergency surgery three additional doses at 8 hourly intervals postoperative may confer further benefit[6,14,20].

The commonest abdominal incision used for acute appendicitis is a grid iron incision. Most authors recommend it because its anatomical centre is at the Mc Burney's point[1,2,5,7]. Though said to give limited access to the appendix, it can easily be enlarged to Rutherford Morrison, Weirs incision or abandoned and another wider incision made. Rutherford Morrison incision is recommended for removal of retrocaecal appendix. A horizontal variety of the Mc Burney's incision is called a Lanz incision, is more cosmetic[1,2]. A right paramedian incision was used in 3 out of 20 patients in whom the diagnosis was uncertain. It is useful because of wide exposure. However access is difficult especially when the inflamed appendix is retrocaecal[2]. Weirs incision has been criticized due to incision of the rectus sheath causing dissemination of infection[3]. In young females, some authors have recommended Pfannenstiel incision as it gives good access to the appendix, reproductive organs and the general peritoneal cavity. It also gives a good cosmetic scar[1,2].

Surgery for acute appendicitis involves the removal of the inflamed appendix, ligation of stump and closure of the wound. The stump is treated in various ways i.e. ligation without burying, ligation with burying and burying without ligation. All stumps of the operated patients in this study were not buried and there were no serious consequence observed. Opinions differ on the best way to treat the stump of the appendix. Buried stumps have been found to form microabscesses and fistulae[11]. In some occasions the buried stump form a polypoid mass which is

difficult to differentiate clinically and radiological from a malignant lesion later on. A stump not cut flush with the caecum can lead to "recurrent appendicitis"[1,5]. Burying of the stump into the caecum has the advantage of peritonization and securing haemostasis. Walter in 1984, showed no significant difference between the two methods of treatment of appendix stumps with respect to wound infection and morbidity in 103 patients[5].

There is divided opinions whether the appendix should be removed when it is found to be normal. While Mcray suggested removal of the appendix after 40 years because of the increased risk of developing malignancy, removing a normal appendix opens the colon and increase the risk of contamination of an otherwise sterile operation hence many authors object to removal of a normal appendix[2,8,14]. Surgical technique and choice of suturing materials have vital role to play in the prevention of wound sepsis[15,16]. When infection is present good results have been obtained by applying delayed sutures. Absorbable sutures such as catgut are suitable in approximating the peritoneum and adjacent layer of the abdominal wall while non absorbable sutures such as linen, silk or monofilament nylon are suitable for superficial layers of the abdominal wall in elderly patients, the obese or those liable to put strain on the wound postoperatively as a result of coughing. Leaving a wound open up to five days or more has been found to reduce wound contamination, allows granulation and leads to acceptable scar[2]. Four of the patients in this series who had generalized peritonitis following perforation of the appendix had their wound left open .

Three patients out of 20 in this series had appendicular masses and were

managed conservatively. The "Ochsner Sherren" regime[2,14] of conservative management implies readiness to intervene surgically if the patients condition deteriorates. The pulse is charted hourly, the temperature every four hours and vomiting is reported if it occurs. The patient is given intravenous fluids and may have 30mls of water every hour. Parenteral antibiotics such as metronidazole is started. This regime give ways to operative treatment if; the pulse is rising, the size of the mass is increasing, there is vomiting or copious constant gastric aspirates and worsening of the abdominal pain. In the three cases, the masses resolved with this regime and interval appendicectomy was performed three months after complete resolution of the masses.

Six out of 17 patients who had emergency appendicectomy developed wound sepsis which contributed to long stay in hospital despite leaving the skin open. The wound pus isolates were mainly gut flora. This confirms the role of endogenous contamination in post appendicectomy sepsis. It has been shown that the organisms causing wound infection in acute appendicitis are almost invariably endogenous and cultures have shown 85% of them to be coliforms[1,2,11]. Three out of five patients in this study were E. Coli. Preoperative use of antibiotics have been shown to reduce the rate of wound sepsis. Barnes in 20 year series involving 7810 patients of acute appendicitis found that systemic antibiotics in normal dosage had no suppressive effect, but in heavy doses they cut down wound infection[17]. Since the bacterial flora found in acute appendicitis is mixed colonic flora with both aerobic and anaerobic organisms, metronidazole in high dose together with either gentamycin or a cephalosporin is recommended. The drugs are given parentally one hour before the

operation as a single dose; however may be followed by two further doses at eight hourly intervals[2].

In a study employing a mixture of topical antibiotic powders, no decrease in infection rate occurred but the topical use of tetracyclines dissolved in saline (1g of tetracyclines per litre of saline) was found to be very effective reducing down the infection rate from 18% to 2%[15,17]. The addition of antibiotics to peritoneal lavage fluid especially the last before wound is closed has also shown to cut down the infection rate[2,15,17].

## CONCLUSION

Acute appendicitis is a common surgical problem in Muhimbili Medical Centre and frequently affects those in young age group. Eighty percent of the patients presented to hospital more than 24 hours from onset of illness when more complications are expected. The only complication however noted, was wound infection which may be related to non availability of antibiotics prior to surgery or a poor choice of drugs or skin closure of contaminated wound. Twenty five percent of patients studied had perforation probably due to delay in reporting to hospital or performing surgery.

A surgeon is advised to balance the risk of surgery against danger of not operating in doubtful cases since waiting until signs of peritonitis appear is generally associated with higher morbidity and mortality.

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# CARCINOMA OF THE OESOPHAGUS


## SUMMARY:

Thirty eight patients with carcinoma of the Oesophagus admitted in one of the three surgical firms at Muhimbili Medical Centre between January and December, 1992 were studied. There were 27 males and 11 females; the male: female ratio being 5 : 2. The peak age was in 51 - 60 year age group. On an average, patients reported to hospital 5 months after the onset of symptoms; at which the disease was advanced and they already had difficulty in swallowing fluids. Progressive dysphagia and loss of weight were the most common clinical symptoms.

Barium swallow, oesophagoscopy, chest x-ray and ultrasound were the main diagnostic procedures which showed advanced disease in all cases. Multiple biopsies were obtained for tissue diagnosis whereby squamous cell carcinoma was the predominant type. The commonest anatomical site involved by the disease was the middle third of the oesophagus accounting for approximately 50% of cases. Palliative radiotherapy was the only modality of treatment given. Five patients died in hospital before and during treatment; the rest completed their treatment. Evaluation of results of therapy was a problem because once discharged patients returned to their home regions and hence lost for follow up.


## INTRODUCTION:

Carcinoma of the oesophagus occurs in different parts of the world with striking variation in incidence. These variations occur even within short distances in

the same country[1,2]. In some parts of the world like the Caspian Littoral of Iran, Linsken province in Northern China, certain parts of Russia and the Transkei in South Africa, the incidence is extremely high[3,4]. Low incidence has been reported in the United States of America and West Africa[4,5]. A sharp variation between regions occurs in some parts of Africa. In Uganda high incidence has been reported along the shores of Lake Victoria, in East and West Buganda; while it is rare in the Northern Province[6]. In Tanzania carcinoma of the oesophagus is the seventh among malignant conditions encountered. It is preceded by breast, skin and thyroid malignancies, lymphoma, cervical and bladder cancer[7]. It forms 1.8% of all tumours as recorded by the Tanzania Cancer Registry[5,7]. In this study the pathology, clinical features, diagnosis and treatment is discussed and review of literature presented. One case is discussed in detail.

## CASE REPORT

M. A. Reg. NO. 677633 a 71 years old man from Morogoro presented with a seven months history of progressive difficulty in swallowing and loss of weight. The symptoms started gradually with difficulty in swallowing solid foods and made a little easier with sips of water. Three months after onset of his illness he was unable to swallow solid food at all and could take only liquids. He was also experiencing disturbing regurgitation with excessive salivation. There was no pain on swallowing, cough, chest pain or difficulty in breathing. He had no hoarseness of voice. He denied a history of oesophageal instrumentation or swallowing corrosives. He had lost weight considerably since the onset of the illness. He took

moderate amounts of local brew made from maize and sugar but had stopped one year earlier. He did not smoke.

On examination the patient was wasted considerably, moderately pale and was not dehydrated. He had no thrush or ulcers in the mouth. He had no palpable cervical, axillary or inguinal lymph nodes. His neck was normal. Abdominal examination revealed no mass, hepatomegaly or ascites. The respiratory, cardiovascular and central nervous system were essentially normal. A provisional diagnosis of carcinoma of the Oesophagus was made. The patient had a haemoglobin level of 9.5 g/dl and normal chest x-ray film. Barium swallow showed a filling defect with irregular stricture of the oesophagus at the lower third. Oesophagoscopy was done with a rigid scope under general anaesthesia. A lot of food residue was found and sucked out. The scope was advanced up to a distance of 33cm from the upper incisor teeth where a friable obstructing tumour was visualised. Several pieces of tissues were taken using biopsy forceps and were submitted for histopathological examination. The report confirmed a moderately differentiated squamous cell carcinoma. His urinalysis, stool and W.B.C. count were reported normal.

He was planned for palliative radiotherapy and received radiation to a total of 4500 rads over ten days delivered by a telecobalt machine. The patient tolerated treatment well and could swallow solids after four weeks of treatment. At 5 weeks he was found to be anaemic with a haemoglobin of 4 g/dl and was transfused two units of blood. He was later discharged home with a haemoglobin of 10.5g/dl and called for review after three months but has never reported back.

## TABLE OF RESULTS

Table I: Age and Sex distribution of patients with Carcinoma of the Oesophagus.

| Age groups (Years) | Male | Female | Total | % |
|---|---|---|---|---|
| <30 | 0 | 1 | 1 | 2.6 |
| 31 - 40 | 0 | 1 | 1 | 2.6 |
| 41 - 50 | 6 | 4 | 10 | 26.3 |
| 51 - 60 | 10 | 2 | 12 | 31.6 |
| 61 - 70 | 6 | 3 | 9 | 23.7 |
| 71 - 80 | 5 | 0 | 5 | 13.2 |
| Total | 27 | 11 | 38 | 100.0 |

The majority of patients were over 40 years of age.

Table II: Clinical presentation

| Symptoms/Signs | Number of patients | % |
|---|---|---|
| Dysphagia | 38 | 100.0 |
| Weight loss | 30 | 79.0 |
| Regurgitation | 24 | 63.2 |
| Chest pain | 20 | 52.6 |
| Cough | 15 | 39.5 |
| Anaemia | 12 | 31.6 |
| Anorexia | 10 | 26.3 |
| Dehydration | 9 | 23.9 |

Dysphagia with loss of weight were main presenting symptoms.

Table 111: Duration of symptoms

| Duration (Months) | Number of patients | % |
|---|---|---|
| Less than 3 | 2 | 5.2 |
| 4 - 6 | 24 | 63.2 |
| More than 6 | 12 | 31.6 |
| Total | 38 | 100.0 |

Majority of the patients presented over 4 months after onset of symptoms.

Table IV: Associated Risk factors:

| Risk factors | Number of patients | % |
|---|---|---|
| Smoking cigarettes/ chew tobacco | **16** | 42.1 |
| Drink local brews | **12** | 31.6 |
| None | **10** | 26.3 |
| Total | 38 | 100 |

Smoking/chewing tobacco and drinking local brew were the risk factors detected.

Table V: Location of tumour in the oesophagus by oesophagoscopy

| Tumour site measured from the upper incisor tooth | Number of patients | % |
|---|---|---|
| Upper (<23 cms) | 3 | 7.9 |
| Mid (23 - 32 cms) | 19 | 50.0 |
| Lower (>32 cms) | 16 | 42.1 |
| Total | 38 | 100.0 |

The disease is most common in the middle third of the oesophagus followed by the distal third.

Table VI: Histological type of tumour

| Histological type | Number of patients | % |
|---|---|---|
| Squamous cell carcinoma | 32 | 84.2 |
| Adenocarcinoma | 3 | 7.9 |
| Anaplastic | 1 | 2.6 |
| Others | 0 | 0 |
| Histology not available | 2 | 5.3 |
| Total | 38 | 100.0 |

Squamous cell carcinoma was the commonest histological type followed by adenocarcinoma of the oesophagus.

## DISCUSSION

The aetiology of carcinoma of the oesophagus is unknown. However several factors that increase the risk of cancer of the oesophagus have been suggested[8,9]. A striking geographical and sex variation in different parts of the world has been reported. Highest incidence is found in Northern China, Japan, Scotland, Russia, Iran and the Scandinavian countries while low incidence is found in Western Europe and America[2]. The incidence in some areas of sub Saharan Africa especially those with the native Bantu and in East Africa is among the highest in the world while in West Africa this tumour is very rare[5]. Within East Africa, the frequency has been reported as low in Tanzania and Uganda and high in Kenya[5,7]. Moreover, even in countries where it is common, sharp variations in frequency between different regions occur[2,4].

The disease is more common in men than women with sex ratio ranging from 12 : 1 reported in Kenya[1] and Tanzania[3] to 1.6 : 1 in Uganda[5]. In this study a male to female ratio of 5 : 2 is reported suggesting that in Tanzania now more females are affected than before. Blacks throughout the world are more affected than whites[8].In the United States of America blacks have a four fold increased incidence compared to the whites[9]. This would indicate a genetic predisposition. Similarly people with tylosis, Torrens Syndrome, Plummer Vinson Syndrome and the Chinese with blood group A have increased risks in developing cancer of the oesophagus[2,8].

Alcohol consumption has been reported to increase the risk for carcinoma of the oesophagus. A fifteen to twenty five fold increased risk has been reported in

France, North America and Western Europe among heavy drinkers with an average alcohol intake of more than 80 g/day. On the other hand it is surprising that incidence of cancer of the oesophagus in Iran and India where alcohol consumption is prohibited is still quite high[6,8]. Different studies in Africa have shown the association of cancer of oesophagus and consumption of local brew[5,8]. The high prevalence has been associated with some chemical contaminants like polycyclic hydrocarbon, fused oils and nitrosamines which are thought to be carcinogens or cocarcinogens present in brew containers, drums and pipes[10]. In this study more than 30% of patients had a history of drinking local brews and / or hard liquor for more than five years.

Habitual smokers especially tobacco pipe smokers have been shown to have six to ten fold risk of having carcinoma of the oesophagus[6,8]. Forty two percent of patients in this study gave history of smoking cigarette.

Other causes are achalasia which is considered to increase the risk by 5 - 10% while Barret's oesophagus increases the risk to 10 - 20% [2,8]. Fungal contamination and aflatoxin have also been implicated[2,8]. Consumption of hot food and beverages have also been implicated, however this has not been substantiated.

Pathology:

Histologically more than 95 percent of oesophageal malignancies are squamous cell carcinomas[11]. Adenocarcinomas arise from the submucosal glands in the oesophageal wall. Adenocarcinomas may originate in glandular epithelium, in Barrets' mucosa associated with reflux oesophagitis or in the ectopic gastric mucosa[7,8]. The middle third of the oesophagus is involved in 50%, the lower third

in 33% and the upper third in 17% of cases[2,7,8,12]. In this study the middle third was predominantly involved in 50% of cases, the lower third in 42% while the upper third was involved in 7.9% (Table V).

Macroscopically three lesions of the oesophageal cancer are recognized. The polypoid tumour mass is the most common (60%) growing into the oesophageal lumen, progressively causing obstruction. The other type is the epitheliomatous necrotic ulcer (25%) with raised and everted margins at the same time excavating the floor eroding the muscular wall. The third type is the annular stenosing lesion (10%) characterized by diffuse infiltration of muscular wall[8,11].

Carcinoma of the oesophagus spreads by direct extension, by the lymphatic and haematogenous routes[12,13]. Direct extension occur transversely and longitudinally eroding the muscular wall and invading surrounding structures in the neck and chest. This is facilitated by the absence of serosa and muscularis layer which is rich in lymphatics[13]. The tumour also spreads in loose connective tissue underlying the epithelium. The tendency to burrow under the mucosa leads to submucous spread well beyond the apparent margins of the growth. Tumours of the upper two third of the oesophagus tend to involve the tracheo-bronchial tree with fistula formation, recurrent laryngeal nerve and erosion of the aorta which is fatal. Two patients in this study had trache-oesophageal fistula and did not survive long.

Tumours of the lower oesophagus invade the diaphragm, pericardium or the stomach. The extensive mediastinal lymphatic drainage which communicates with central and abdominal collateral is responsible for mediastinal, supraclavicular or coeliac lymph node metastases which occurs in at least 75% of patients with

oesophageal carcinoma[2,8]. Blood bone metastases occur usually to the liver and lungs[8]. The UICC / AJCC has been recognised by W. H. O and is recommended to be used World wide. The following table gives a UICC / AJCC oesophageal cancer staging which gives the best correlation between the extent of the disease and prognosis.

T = Tumour N = Nodes M = Metastases

| TUMOUR | DESCRIPTION |
|---|---|
| TX | Primary tumour cannot be assessed. |
| To | No demonstrable tumour in the oesophagus. |
| Tis | Carcinoma in situ. |
| T1 | Tumour invades lamina propria or submucosa. |
| T2 | Tumour invades muscularis propria (Muscle involvement). |
| T3 | Tumour invades perioesophageal tissues. |
| T4 | Tumour invades adjacent structures. |

NODES

| | |
|---|---|
| NX | Regional nodes cannot be assessed. |
| No | No regional node metastases (Oesophageal nodes are not available at bedside for palpation). |
| N1 | Regional nodes metastases. |

METASTASES

| | |
|---|---|
| Mx | Distant metastases cannot be assessed. |
| Mo | No evidence of distant metastases. |
| M1 | Distant metastases present. |

Note: N : Nodal involvement. For cervical oesophagus the regional lymph nodes are the cervical and supraclavicular nodes. Whereas regional lymph nodes for the upper, mid and lower thoracic oesophagus are not ordinarily accessible for clinical evaluation. Ultrasound of the oesophagus usually show oesophagus nodes, if involved.

**STAGES**

| | |
|---|---|
| 0 | Tis No Mo |
| I | T 1 - 2 No Mo |
| IIA | T 2 - 3 No Mo |
| IIB | T 1 - 2 N1 Mo |
| III | T3 N1 Mo, T4 N o - 1 Mo |
| IV | T? N? M1 |

## CLINICAL FEATURES AND DIAGNOSIS

The most common symptom of oesophageal cancer is dysphagia. However, dysphagia can occur with mechanical or functional disorder which interferes with peristalsis. Regrettably dysphagia is a late manifestation of the disease because the early stage progresses silently. At the time dysphagia sets in 60 percent of the oesophageal circumference has been surrounded by the tumour[14]. The sensation that the patient feels is progressive and persistent. It is progressive in the sense that the patient first notices difficulty in swallowing solid foods, later semisolid and ultimately liquids[12,14]. The progressive occlusion of the oesophageal lumen by tumour may be marked if the patient subconsciously restricts intake to avoid such unpleasant

sensation and therefore careful attention is paid to the consistency of ingested food when enquiring about oesophageal symptoms. Dysphagia is usually persistent, present at all meals rather than intermittently as it might be in oesophageal spasm, achalasia and other motility disorders[12]. In this study all 37 patients had dysphagia (Table II). The mean duration of dysphagia in series of Tanzanian patients has been found to be 3 months[3,5,7]. Another common complaint was regurgitation. This was recorded in 24 patients in this series. Regurgitated food material is usually alkaline mixed with saliva and may be streaked with blood from the malignant growth. The pain associated with oesophageal cancer is usually steady, substernal, occasionally radiating to the back. Sometimes patients report odynophagia (painful swallowing) which may be related to a tumour of the upper third of oesophagus[8]. Pain is an unfavourable prognostic symptom because it may represent involvement of adjacent structures[11,13]. In this study 45% of patients presented with non radiating chest pain. Anorexia and weight loss are non specific symptoms related both to presence of disease in general and the problem created with eating. Most patients are cachectic (defined as 10 percent or more loss in body weight within six months[12,13]. In this study 85% of patients presented with loss of weight.

Chronic cough is another symptom and is present in most patients[14]. This is an unfavourable prognostic symptom as it may indicate oesophageal, bronchial or trache-oesophageal fistula, abscess or chronic over flow and aspiration of saliva or food into the lung resulting from obstruction[8,14]. Fifteen patients (46%) in this study presented with cough. Pulmonary involvement is not uncommon as nearly half of patients with oesophageal cancer have findings of bronchopneumonia at autopsy[14].

Haemoptysis occurs when the trachea or bronchi are invaded[4]. None of the patients in this study had haemoptysis.

Hoarseness of voice is another symptom of prognostic value indicating vocal cord paralysis from recurrent laryngeal nerve involvement with tumour or chronic laryngitis. Aphonia with persistent irritation may accompany hoarseness[21]. No patient in this study had hoarseness of voice or aphonia. Decomposition of material stuck in the oesophagus gives a malodour. Almost all patient have malodour noticeable when one gets closer to them. Involvement of sympathetic nerve trunk on either side or both sides produce initially a dilatation of pupil on corresponding side and slight degree of exophthalmos and later paralysis associated with enopthalmos and finally the pupil become contracted and fixed. Hiccups signify tumour involvement of the diaphragm and the phrenic nerve[8].

Physical signs do not contribute greatly to the diagnosis. The only signs are cachexia, anaemia with severe dehydration.

A chest x-ray is essential in every patient since it determines the prognosis. The normal oesophagus is not visible on chest x-ray. Air fluid level on chest x-ray suggests total oesophageal obstruction while a large soft tissue mass may represent tumour, pulmonary metastasis, lung abscess, pneumonitis or hilar lymphadenopathy. The trachea shadow may show compression, irregularity with displacement. A barium swallow characteristically shows irregular ragged mucosa with luminal narrowing, severe stenosis with slight proximal dilatation in infiltrative lesion, whereas multiple filling defects are seen in fungating lesions. Other typical appearance includes annular constriction and "shouldering" which may be seen at the

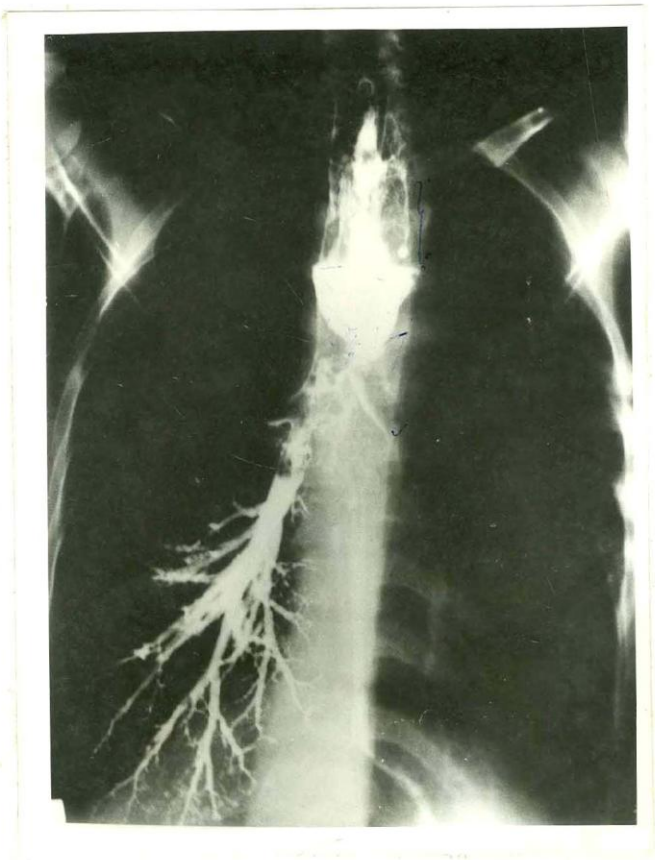
proximal end of the filling defect[8,13], (Fig II). Sometimes the tumour presents as a tight stricture such that barium swallow cannot pass. Use of double contrast using barium followed by water in upright position is more diagnostic[14].

Another diagnostic method is the use of radioactive isotopes such as phosphorus and followed by passing a Geiger counter down the oesophagus by which means the isotope accumulated by a normal and malignant tissue structure can be compared[8,14]. With Computerized Axial Tomography (CAT) the normal oesophagus is shown as a thin walled structure, often containing air and delineated from other structures. In carcinoma the walls have undergone irregular thickening with obliteration or destruction of its lumen. CAT makes it possible to assess extra oesophageal spread into airway, mediastinal, intraabdominal lymph nodes as well as hepatic and adrenal involvements[8].

Oesophagoscopy is mandatory in all patients with dysphagia. It helps in tumour location and obtaining samples by brushing for cytological examination as well as biopsy for tissue diagnosis.

Epidermoid tumours usually stimulate an inflammatory response with scar formation at the advancing margin, hence taking a biopsy at the margin may simply demonstrates inflammatory tissues. The same applies to an area narrowed by submucosal spread and inflammatory oedema which may give a negative histological results. It is therefore advised that biopsies should be taken from 4 quadrants deep down within the obstruction and sometimes after bouginage[14]. A single biopsy is positive in only 70 - 80 percent of cases where as multiple biopsies aided by endoscopic brush cytology increase the diagnosis by 96 to 100 percent[8,15].

Figure II: An Oesophagogram and a brochogram showing hold up of barium swallow at the middle third of the oesophagus, proximal dilatation, shouldering with distal irregular stricture formation, features consistent with carcinoma of the oesophagus. However there is oesophageal bronchial fistula as a complication.



Lavage exfoliative cytology is an alternative to endoscopic brush cytology in case of stricture. It involves inserting a radiopaque nasogastric tube through the oesophageal stricture under fluoroscopic guidance and aspiration of fasting gastric content with prompt fixation and immediate processing. The diagnostic yield of this procedure exceeds 95 percent when done properly[14,15].

Bronchoscopy is useful in excluding extension into the airway and is done preferably before oesophagoscopy at the same sitting.

Detection of cancer at cytocellular stage in high prevalence areas in China is reported using endoscopy with Lugols iodine dye. This has improved survival rate following oesophagectomy with 5 year survival rates of 85 to 90 percent[14]. The differential diagnoses include benign peptic stricture, cancer of the gastric fundus or oesophago - gastric junction and achalasia. Endoscopy and biopsy usually confirm the diagnosis[8].

## TREATMENT

The philosophy of surgical management of all gastrointestinal malignancy is total excision of the lesion and regional nodes and re-establish intestinal continuity (effective cure)[1,15]. However the advanced state of the disease that is seen in most patients makes palliation a reasonable goal and must be considered as the primary objective in most instances[15].

### SURGERY

The outlook of patients with cancer of the oesophagus has improved over 10

years[12]. The operative mortality rate for resection has been less than 10% and is now attainable in most centres in the world[8]. With better surgical technique and better preoperative care mortality rates between 1 - 8% have been reported with 5 year survival after curative resection between 20 - 40% while for early lesions T1 - T3 No the 5 year survival rate of between 40 - 80% has been achieved[6]. The common surgical procedures for treatment of carcinoma of the oesophagus include radical oesophagectomy, palliative resection and reconstruction, bypass procedures and oesophageal intubation[1,9]. Thorough evaluation of the patients for evidence of systemic spread, wall penetration, nodal metastases are important before deciding what procedure is to be performed.

Preoperative preparation

A high incidence of chronic pulmonary disease is seen in patients with oesophageal carcinoma. Although it is caused by long term use of tobacco, chronic aspiration pneumonia is not infrequent[15]. Almost all patients are nutritionally depleted and dehydration is common. The red cell mass is frequently low although haematocrit might be deceptively high because of coexisting dehydration[15]. Although prolonged period of preoperative preparation cannot be justified in a disease in which survival is often short, intensive preoperative treatment is initiated. The blood volume, fluid and electrolyte balance should be normal. Infusion of colloid and blood if necessary should be instituted. Hyperalimentation preferably using parenteral route is necessary. Assessment of the colon for reconstruction by barium enema and bowel preparation is indicated preoperatively[8,9]. The use of mediastinal tomography and CAT scan may afford greater preoperative staging information and hence smooth

surgery[8].

Total oesophagectomy

This is a procedure aimed at eradicating the disease[1,15]. Initially radical oesophagectomy was done, however a sufficient wide margin above the tumour of about 10 cm was not obtained due to submucosal microscopic spread[15]. The posterior mediastinal tissues surrounding the oesophagus are removed intact. Total oesophagectomy with anastomosis of the fundus of the stomach to the oesophagus in the neck (Mckeown three stage operation) has been advised for growths in the upper half of the oesophagus[16]. The first two stages are those of the Lewis operation with complete mobilization of the stomach and thoracic oesophagus. The cervical oesophagus is then exposed through an incision along the anterior border of the right sternomastoid. The omohyoid muscles, middle thyroid vein and inferior thyroid artery are divided and the oesophagus and stomach drawn up into the wound. This procedure is done on right side as the thoracic duct may be damaged on the left. The oesophagus and stomach are transected and anastomosis is done. In selected case, left thoracic approach is preferred. Mckeown has reported 253 oesophageal resection with a 12 percent operative mortality and a 24 percent 5 year survival rate[16]. The only complication feared is anastomosis leak, however, if it occurs in the neck, the consequences are fewer and healing is good[1].

Palliative Oesophagectomy

Up to 40% of oesophageal cancer patients have lymph node metastasis in the neck, mediastinum or abdomen. This is not necessarily an indication that the disease

is locally advanced and oesophagectomy is sometimes advised even when systemic metastases are present[2]. Palliative oesophagectomy effectively removes the fungating necrotic lesion which is the cause of obstruction and the source of sepsis and anaemia.

Reconstruction of the oesophagus after resection is done by using iso-peristaltic colonic segment from the stomach or jejunal segment. The left colon at the splenic flexure and left part of the transverse colon based on the ascending branch of left colic artery is highly suitable because it has a reliable blood supply and is near the oesophageal hiatus. The diameter of this colonic segment closely approximates that of the oesophagus. Colonic interposition is associated with less frequency of reflux, bleeding or stricture formation[4]. Two principal approaches to oesophagectomy are commonly used:

i) Transthoracic oesophagectomy

The procedure involves an abdominal operation for mobilization and resection of colonic segment, (commonly used) mobilization of the stomach and performing intra - abdominal colonic anastomosis and gastro - colic anastomosis. A right postero - lateral thoracotomy is then performed where the oesophagus is resected and in its place the colonic segment along with its blood supply is brought into the mediastinum and oesophago - colic anastomosis is performed in the chest or neck[8].

ii) Transhiatal oesophagectomy without thoracotomy

This involves resection of the oesophageal tumour through an upper abdominal and cervical incision. The oesophagus is removed largely by blunt

dissection through the oesophageal hiatus. In this place the stomach tube is commonly used and oesophago - gastric anastomosis is performed in the neck. It is a palliative procedure where the dissection or posterior mediastinal tissues cannot be achieved. It is indicated in elderly and poor surgical risk patients who cannot tolerate Transthoracic approach. It is a rapid procedure. Anastomosis of this procedure is performed at the neck and anastomosis leak which is a major factor determining surgical mortality and morbidity can more easily be managed. Oesophagectomy using this method has been practised by Orrringer[18]. In 100 patients he operated using this procedure, the post operative mortality was 6% with a 5 year survival rate of 17%. In comparison between thoracotomy and non thoracic procedure, the Transthoracic procedure enables systematic dissection of posterior mediastinal tissues and is therefore suitable for radical operations however, it is a long procedure involving two intra abdominal anastomosis and intrathoracic anastomosis whereas Transhiatal oesophagectomy does not involve systemic mediastinal dissection of lymph nodes[15]. The survival benefit however with the two procedures do not significantly differ.

Bypass procedures

When the growth is inoperable because of the general condition of the patient or presence of metastases or is found not to be resectable at thoracotomy or laparotomy then a palliative procedure is carried out to enable the patient to swallow[4]. This may be by surgical bypass or by intubation. However, greater judgement is needed to ensure that palliative procedures don't end by causing the patient extra misery[4]. Some of the methods used to bypass the oesophagus is transposition of the stomach or a segment of colon to a substernal position. The

oesophagus is excluded by dividing and closing the proximal and distal ends. The mortality rate for colon bypass of oesophageal carcinoma is at least 15 to 25 percent while the retrosternal gastric bypass of oesophageal carcinoma is 25 to 40 percent with the incidence of anastomotic disruption high and an average survival rates of less than 6 months[19].

Vikkular et al proposed the use of reversed pectroralis skin pedicle flap and free skin graft to create extra thoracic conduit from the oesophagus to the stomach, however this still need further research.

Oesophageal intubation

This is suitable in patients with unresectable tumours, recurrence after oesophageal resection or in tracheo - oesophageal fistula. An intra oesophageal tube in malignant tracheo - oesophageal fistula occludes the oesophageal side of the fistula and permits oral alimentation for several months of the remaining life[3]. Pulsion tubes (e. g. Souttar) can be pushed through the tumour from above with the aid of an oesophagoscope while traction or pull through tubes (e. g. Celestin) can be pulled into the place by traction with access into the stomach obtained through gastrotomy[20]. Although the ability of the patient to handle saliva is improved by intubation of the oesophageal tumour, oral intake should be restricted to food of a consistency compatible with passage though the rigid indwelling oesophageal conduit and palliation is therefore not optimal. A favourable experience on ten patients managed by oesophageal intubation has been shown by Hiza at Muhimbili Medical Centre and that the procedure does not need a specialized unit[3]. In several studies the overall

mortality as shown by Ferguson, Appelgnst and Stone et al is between 11 and 21 percent respectively with survival of less than 6 months[15]. Some of the complications of these procedures are perforation of the oesophagus, obstruction of the tube by food or tumour overgrowth and migration of the tube[19]. None of the patients in this study was treated surgically or by intubation.

Radiotherapy

Radiotherapy is a good palliative treatment for pain but is less effective for palliating dysphagia[15]. Cure seldom results from radiation treatment. Distant intramural spread and radioresistant tumours like adenocarcinoma will not be cured by radiation[21]. Squamous cell carcinomas respond best to radiation but cure is not usual. This is limited to a small number of patients with small localized lesions[21]. Pearquin in a study of 100 patients found significant palliation with clinical improvement of dysphagia in 65 - 75% however, despite treatment with 6900 rads and 8700 rads over 5.5 weeks and 8 weeks split course respectively, the recurrence rate was 80%[13].

The attractiveness of radiotherapy is its safety and that it can be stopped when the patient's condition deteriorates. However, serious complications can occur related to the tumour extent, treatment volume and radiation dose. These includes fistulae, radiation pneumonitis, pulmonary fibrosis, pericarditis, myocardial damage and injuries to the spinal cord. When there is malignant infiltration of the atrium or aorta or if there is oesophago - trachea fistula, radiation is likely to worsen the situation[21].

In this study only 17 patients (46%) completed the planned dose of

irradiation; 14 patients had definitive improvement on dysphagia, 5 patients died in hospital, 3 of them before the start of the treatment. In 16 patients dysphagia did not improve on radiation. Four of the patients presented with recurrent disease after initial treatment.

The aim of preoperative radiotherapy is down staging and has recently revealed significantly improved resectability rates however, is associated with increased postoperative mortality[11,12,13]. Due to controversy of benefits of radiotherapy, it has been suggested that radiotherapy should be reserved for unresectable tumours or for patients unwilling to undergo surgery or for upper third tumours in which surgery is difficult and dangerous[12].

Chemotherapy

This differs from radiotherapy and surgery in that its effects are systemic[15]. Unfortunately it has not found a definite place in the treatment of oesophageal carcinoma but it can increase the chance of curative treatment in early disease when used in combination with surgery or radiation or it may improve palliation in advanced disease[8,12]. Some benefits in oesophageal carcinoma have been noted with 5 fluouracil, Bleomycin, Mitomycin C and Cis - platinum in combination[8]. Greater response has been shown when combination, chemotherapy is used but recurrence rates of up to 50% have been reported[4,8]. Chemotherapy in combination with radiation has been reported to be superior in palliation than radiation alone[15]. Similarly combined surgery, radiation and chemotherapy offer the best palliative result than one alone.

## CONCLUSION

Over a period of 5 years, an average of 5 patients per month were admitted to one of the three surgical firms at Muhimbili Medical Centre. Most patients present late when the disease is advanced and the only modality of treatment offered is palliative radiotherapy. Effective radiotherapy and facilities for intubation should be given special consideration for palliation of dysphagia that accompanies this distressing disease. In view of the high frequency of this malignancy in Tanzania, surgery, radiation, and chemotherapy in combination should be considered as a future palliative treatment modality.

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# KAPOSI'S SARCOMA


## SUMMARY:

Twenty patients with histologically proven Kaposi's Sarcoma were seen in one of the surgical firms at Muhimbili Medical Centre between January 1990 to December 1992. The male : female ratio was 4 : 1 and their ages ranged between 10 and 69 years. The most affected age group was the 30 - 39 years. (Children less than ten years were excluded from the study).

The most frequent presenting symptoms were cutaneous skin lesions and oedema of the limbs. The commonest morphological types were nodular and florid lesions while mixed cellularity was the commonest histological finding. The most affected parts of the body were the lower limbs. Forty percent of patients presented with atypical type of Kaposi's Sarcoma and were HIV positive by ELISA. Sixty percent of the patients were treated with half body radiation with good short term response. Follow up remains a problem because once discharged patients returned to their home regions and hence lost for follow up.


## INTRODUCTION:

Kaposi's Sarcoma is a multifocal, idiopathic angiosarcoma which was first described by a Hungarian dermatologist Moritz Kaposi in 1872[1]. During that time Kaposi's sarcoma was described as affecting mainly the Jews, Italians and Africans around the equator. It occurred commonly in males in 5th to 7th decade of life. Kaposi's sarcoma of this type was referred to as Endemic Kaposi's sarcoma (EKS)[1,2].

The frequency of the disease started increasing in 1978 when cases of Kaposi's sarcoma were reported among a group of young homosexual men. By 1983, an aggressive form of Kaposi's sarcoma was reported to the Centre for Disease Control (CDC) from more than 30 states in the united state[2,3]. Since then more cases of Kaposi's sarcoma have been reported with a fatally aggressive behaviour. This formed the basis of classification of Kaposi's sarcoma into two categories Endemic type (classical) and Epidemic type (aggressive, atypical). Homosexuality, drug abuse and haemophilia were found to be the associated risk factors in Europe and America a finding which is uncommon in Africa[1,4]. However, one common feature of all patients with the aggressive form of Kaposi's sarcoma was that they were invariably immunocompromised.

The EKS presents with lesions that had classical pattern (i.e nodules concentrated on extremities starting with the lower limbs and then the upper limbs, a long standing history not less than nine months since onset with a good prognosis. The AKS is characterised by disseminated atypical appearance of nodules, fast progress, early occurrence in lymph nodes and / or internal organs such as liver, spleen and intestines, is refracting to treatment and has a poor prognosis.

The diagnosis of Kaposi's sarcoma is confirmed at histopathology[4,5]. Radiotherapy is the main modality of treatment and chemotherapy is promising while surgery plays a very little role. Most patients with EKS have good response to treatment, while those with AKS have poor response and most of them die due to intercurrent opportunistic infections[5].

In this study an interest has been focused on determining the various clinical

presentations of Kaposi's sarcoma as seen at Muhimbili Medical Centre over a period of 3 years. One patient is presented in detail while literature is reviewed on epidemiology, pathology, clinical presentation and treatment.

**CASE PRESENTATION**

A male patient E . S aged 34 years IP No 768613 was admitted in one of the Surgical firms in November 1993 as a referral from Bukoba with complaints of progressive multiple swellings of both legs and feet for two years. The swelling started as a bruise that gradually darkened and became raised as a firm nodule on the right foot. A year from the onset of the disease a similar swelling appeared on the other foot. Initially he incised one of the nodules with a razor blade, resulting in profuse bleeding. He was attended at a nearby dispensary but later used traditional herbs without improvement. Multiple crop of similar swelling were noted increasing upward from the feet up to the region below the knees. They were painful and at times he had pricking pain in both feet when walking over long distances. He also noted swelling of both lower limbs. He denied a change in bowel habit and his appetite was good. However he had lost weight. He was not married. He had a history of unprotected sex with multiple partners. He did not smoke cigarettes or drink alcohol. He denied a history of drug abuse. There was no similar disease in the family.

His general health was fair and he was not pale icteric or febrile. His pulse rate was 88 / min regular and of full volume. His blood pressure was 130/70 mmHg. He had cervical, axillary and inguinal lymph node enlargement and these were

mobile, firm non tender and measured in average 1 - 3 cm in their largest diameter. He had non pitting oedema confined to both legs. Detailed examination of the lesions on the lower extremities showed hard woody oedema below knees with multiple hyperpigmented nodules. They were of variable sizes from (0.2 - 1 cm in diameter), pink to black in colour some ovoid while others round in shape and were non tender. The few ulcerative lesions in between toes were foul smelling and easily bleeding to touch. The thighs and upper extremities were normal. The oral cavity was normal. Examination of the alimentary , respiratory, cardiovascular and urogenital system was normal. A diagnosis of Kaposi's sarcoma (endemic type) was made and several investigations were done.

The haemoglobin was 12.6 g/dl with a normal white blood cell count. Urine and stool examinations were normal. Chest x-ray was normal and abdominal ultrasound did not reveal bowel involvement. HIV ELISA test was negative. Biopsy taken from one of the nodules showed spindle cells, vascular space with macrophages and leucocytes characteristic of Kaposi's sarcoma. The patient was planed for local radiotherapy with 800 rads. It was given in fraction of 200 rads daily which the patient tolerated well. He had a normal white cell count post radiotherapy and was discharged to be followed at six months. He had not reported back by end of this study.

## PATIENTS ANALYSIS

Table 1: Age and Sex distribution of Kaposi's sarcoma cases

| Age group | Number of Patients | | Total |
|---|---|---|---|
| | Male | Female | |
| 10-19 | 1 | 0 | 1 |
| 20-29 | 3 | 1 | 4 |
| 30-39 | 9 | 2 | 11 |
| 40-49 | 1 | 1 | 2 |
| 50-59 | 0 | 0 | 0 |
| 60-69 | 2 | 0 | 2 |
| Total | 16 | 4 | 20 |

The commonest age group was 30 - 39 years majority being male.

Table II: Clinical Presentation

| Symptoms | Number of Patients |
|---|---|
| Cutaneous lesions | 14 |
| Oedema of limbs | 10 |
| Emptying sign to nodules | 12 |
| Itching | 6 |
| Pain | 6 |
| Progressive loss of weight | 5 |
| Pyrexia of unknown origin | 4 |
| Generalized lymphadenopathy | 4 |
| Respiratory symptoms | 3 |
| Facial oedema and herpes Zoster | 2 |

Cutaneous skin lesions with oedema of the limbs were the most frequent presenting symptoms.

Table III: Type of Kaposi's Sarcoma lesion

a) Morphological type

| Type of lesion | Number of Patients | % |
|---|---|---|
| Nodular | 9 | 45 |
| Plaque | 2 | ~~30~~ 10 |
| Florid | 6 | ~~15~~ 30 |
| Lymphoglandular | 3 | ~~10~~ 15 |
| Total | 20 | 100 |

Nodular and florid type of lesions were the commonest morphological types.

b) Histological types

| Histological types | Number of Patients | % |
|---|---|---|
| Mixed cellularity | 10 | 50 |
| Spindle cell | 4 | 20 |
| Anaplastic | 3 | 15 |
| Unspecified | 3 | 15 |
| Total | 20 | 100 |

The commonest histological type was mixed cellularity while 3 specimen were unspecified.

Table IV: Distribution of Kaposi's sarcoma lesion

| Site of lesion | Number of patients | % |
|---|---|---|
| Lower limbs only | 12 | 60 |
| Upper limbs only | 2 | 10 |
| Upper and lower limbs | 4 | 20 |
| Generalized ( Limbs and trunks ) | 0 | 0 |
| Oral cavity, vagina and rectum ( Mucosal involvement ) | 2 | 10 |
| Total | 20 | 100 |

The lower limbs were affected mostly.

Table V: Modality of treatment

| Mode of Treatment | Number of patients | % |
|---|---|---|
| Half body Radiation | 14 | 70 |
| Extended local irradiation | 3 | 15 |
| Irradiation combined with chemotherapy | 2 | 10 |
| Combined chemotherapy ( Adriamycin, Bleomycin and Vincristine ) | 1 | 5 |
| Total | 20 | 100 |

The main mode of treatment was Radiotherapy.

## DISCUSSION

Incidence

Kaposi's sarcoma was first described in 1872[1]. There has recently been a marked change in age, sex, geographical distribution and disease pattern[1,6] Before 1979 Kaposi's sarcoma was mostly described in elderly men in the age range between 40 and 89 years of either Italian or Eastern Europe, Jewish in origin. In the U. S. A. the incidence was reported to be around 0.02 per 100,000 population. The form of the disease was the classical endemic type which pursues a rather indolent course[7]. Kaposi's sarcoma was found to be endemic also in North East Congo and North West Uganda accounting for approximately 10 percent malignancies reported in theses areas at that time[8]. Native Blacks were more commonly affected than non Blacks[5].

Since 1981 when the Centre for Disease Control (CDC) reported an outbreak of an aggressive form of Kaposi's sarcoma in young male homosexuals in America,

there has been subsequent increase in newly reported cases sometimes doubling every six months to reach an epidemic proportion[4]. The rapid rise coincided with the AIDS epidemic. Most patients with Kaposi's sarcoma were also noted to have an acquired immunodeficiency syndrome ( AIDS ). Furthermore patients with immunodeficiency states were also found to have increased incidence of the tumour than the general population. Twenty to thirty percent of patients with AIDS have been found to present with Kaposi's Sarcoma as a principal symptom[9].

Since the AIDS epidemic, the disease pattern of Kaposi's Sarcoma have changed[9]. Those patients at risk for AIDS like homosexuals, heterosexuals, intravenous drug abusers, transfusion recipients such as haemophiliacs are also at risk of acquiring AIDS related Kaposi's Sarcoma[4,6]. It was noted however that patients with AIDS acquired through blood transfusion had a lower risk of developing Kaposi's Sarcoma compared to those with sexually acquired AIDS[2,3,6]. Patients affected are younger with an average age of 36 years[6]. While pre- AIDS Kaposi's Sarcoma was classically known as predominantly male disease, with a male : female ratio of up to 12 : 1[1]; the ratio has changed in some parts to a ratio of 2 : 1 suggesting increase in female affected by the disease[5,7].

Endemic Kaposi's Sarcoma which is common in East and Central Africa has been noted to increase in prevalence as one approaches the equator from both the north and south. The highest frequency has been reported in Zaire[6] (12%), Uganda[8] (9%) and Tanzania[10] (4%).

In Tanzania Kaposi's Sarcoma has been existing for a long time. In 1969, Slavin et al[10] reported it as accounting for 4% of all cancers on mainland Tanzania. Bayley[11]

has reported an increasing incidence and changing pattern in North West Tanzania during the AIDS era.

It is the explosion in the number of AIDS patients and the consequent increase in the number of patients with AIDS-KS world wide that has led to the epidemic-KS.

Aetiology and Pathogenesis

The etiology and pathogenesis of Kaposi's Sarcoma is not clearly known. Various factors have been associated with Kaposi's Sarcoma, including geographical and environmental factors, oncovirus, genetic factors, various disease conditions, drugs and the state of immunity. The finding of high incidence of Kaposi's Sarcoma among Jews whether born in Russia or U. S. A. is in favour of racial influence. A high incidence of Kaposi's Sarcoma in Eastern Zaire, Western Uganda and North Western Tanzania tend to suggest probable geographical as well as environmental factors.

HLA - DR5 a major histocompatibility antigen noted to be present with high frequency among Africans, Italians and Jews compared to other ethnic groups also suggesting a genetic inheritance[2,4]. Scully et al[12] demonstrated that HLA - DR5 occurred over 60% in patients with epidemic Kaposi's Sarcoma associated with immunodeficiency syndrome against a control of 23%.

Single or repeated viral infections have been implicated in the aetiology of Kaposi's Sarcoma, particularly the epidemic AIDS associated type[13]. Viral oncogenesis is supported by the finding of high level circulating antibodies to several viruses including the cytomegalovirus, Epstein Barr virus (EBV), Herpes virus (HV)

and hepatitis A and B viruses. Likewise elevated levels of cytomegalovirus - RNA, cytomegalovirus - DNA and cytomegalovirus antigen have been isolated in the serum of Kaposi's Sarcoma patients and in the tumour cells[13,14]. It is postulated that AIDS results in immunosuppression allowing induction of Kaposi's Sarcoma by agents such as cytomegalovirus[14]. Kaposi's Sarcoma has been reported to develop in 8 - 15% of HIV - seropositive individuals and in 30% of patients suffering from AIDS (50 - 70%) for those with lesions in the oral cavity)[6]. It has been reported to occur in association with primary lymphoma of the central nervous system, bone and bowel in 42%, 33% and 17% respectively[6,13].

In the West, Kaposi's Sarcoma is reported to occur with higher frequency in homosexual men and intravenous drug abusers, haemophiliacs, children whose mothers were proven HIV seropositive, those with recurrent sexually transmitted diseases such as syphilis and exposure to drugs such as amyl nitrates[13].

An underlying state of compromised immunity appears to play a significant role in the genesis of Kaposi's sarcoma. The increased incidence of malignancies has been well documented in patients on immunosuppressive drugs e.g. cytotoxic ones, long steroids therapy as well as in kidney transplant recipients[13]. These drugs have an indirect effect by altering the immunological surveillance. The suppressed immune system then loses its policing function of destroying neoplastic cells as they arise. For those patients on cytotoxic drugs the tumour resolves spontaneously with reduction or discontinuation of the medication thus implicating an immune deficiency factor[14,15].

## CLASSIFICATION OF KAPOSI'S SARCOMA

Kaposi's sarcoma can be classified into histological, morphological type and on clinical basis:

### 1. Histology

O'Connel[16] attempted to classify Kaposi's sarcoma into three histological types; spindle cell, mixed cell and anaplastic. Harawi has added two other histological types, angiomatous and inflammatory types.

Mixed type is the commonest in all epidemic forms of Kaposi's sarcoma and the most easily recognized. O'Connel found the mixed type of Kaposi's sarcoma in 80% of cases reported in Malawi[16]. In this study 50% of the patients had a mixed type of lesion (Table IIIb). This lower percentage might be explained by the small number of patients in the study. In this type there is equal proportion of spindle cell and vascular slits. The spindle cell are arranged in bundles with red blood cells endothelial cells, plasma cells and mitotic figures are few. It is the most favourable histological type[16].

In the spindle cell type, the spindle cell predominate the histological picture. Some authors call it monomorphic or pleomorphic Kaposi's sarcoma. It is the second commonest variant. In this type vascular component and other cells are few, mitotic figures are frequent while plasma cells infiltration is more marked. Twenty percent of the patient in this study had spindle cell type; a finding which corresponds to other studies[6,8].

In the anaplastic type; There is cellular pleomorphism; the spindle cells

being of variable shapes and sizes and mitotic figures most marked. It has the worst prognosis.

Angiomatuos type is a rare variety composed predominantly of well formed usually thin walled large and small vascular space. It is difficult to differentiate this from angiosarcoma or anaplastic type. The inflammatory type consist of angioproliferative process with arborizing vessels. The vessels may have hyalinized wall and associated with a prominent lymphoplasmocytic infiltrate[16].

2. Morphological type

Morphologically there are five types of Kaposi's sarcoma; Nodular, Plaque, Florid, Lymphoglandular and visceral. These are important in clinical diagnosis. Nodular type: Is commonest in adult and second most favourable[16,17]. It represents the tumour that evolves from the early lesion and commonly manifest as a well defined nodule ranging from a few millimetres up to 3 cm in diameter oval or round shaped pink, blue, brown or purple in colour. Emptying sign is pathognomonic of Kaposi's sarcoma nodules. Although predominantly dermal nodular Kaposi's may involve the viscera and solitary nodules have also been found on the penis or in the lacrimal gland. A solitary nodule on the hand or foot usually heralds progressive disease.

Nodules that are not palpable can be detected by a soft tissue x-ray. It is found that nodules do not ulcerate unless traumatised. The nodular type of Kaposi's sarcoma is preceded in over 80% of all patients by pitting oedema which become firm and finally hardens. Onchocerciasis, leprosy, malignant melanoma, jiggers and scabies may sometimes be confused with this type of lesion. The disease progresses

slowly and centrally with formation of spreading to lymph nodes and viscera. Both the classical and some endemic types of Kaposi's sarcoma presents in this way.

In this study 45% of patients presented with nodular type of Kaposi's sarcoma while Taylor from Uganda found it to occur in 30% out of 37 patients . Hyperpigmented and flattened lesions but which remained elevated above the skin surface also occur forming plaques. In many untreated patients after several years; The nodules or plaques may grow rapidly; coalesce and ulcerate forming florid tumours.

Florid type: These presents as fungating ulcerative masses that invade the subcutis and easily bleed. They may infiltrate the deeper tissues and invade bones. This infiltration induces fibroblastic reaction resulting in swelling and woody induration of the affected limb making it difficulty to recognise as Kaposi's sarcoma. This type accounts for 35% of Kaposi's sarcoma in Africans[16]. It is rare in the Caucasians and has not been described in association with AIDS[8]. Sometimes it is indistinguishable from syphilis, fungal and tropical ulcer except by histology[7,8]. In this study 30% had a florid lesion, a finding which is low compared to 43% in a study reported by Taylor[8]. A probable explanation of the difference might be the aggressiveness of the disease and small number of patients studied.

Lymphadenopathic type: This type was commonly seen in children below the age of ten years in the pre-AIDS era. The disease had a fulminating cause with death within a year. After AIDS epidemic it is the commonest presentation in adult with atypical type of Kaposi's sarcoma[6]. Fifteen percent of patients in this study had lymphadenopathic type, a finding similar to the one reported by Taylor in Uganda[8].

Visceral type: This sometimes is the only manifestation in young adults. Any internal organ can be involved; the stomach, bowel, mesentery, liver, and the suprarenal gland. Lungs and brain may be primarily or secondarily involved. Bayley[5] found 10% of patients had visceral involvement in a group not affected by AIDS while 50% of patients with AIDS in the same study had visceral involvement[3,5]. These patients presented with haemorrhage, perforation, obstruction and protein losing enteropathy. She further recommended endoscopy for detection of gastrointestinal involvement. None of the patients in this study were diagnosed as having visceral involvement.

Mixed type: This implies any combination of the above clinical types. Safai[3] et al noted that patients with immunodeficiency have a mixture of nodular and lymphadenopathic types.

None of the patients in this study had a full skeletal survey for diagnosis. ( Periosteal elevation, cortical erosion and cystic changes may be seen ).

Clinical types: There is no universal clinical classification of Kaposi's sarcoma. Initially it was classified into indolent form which include, plaque, nodular, mixed and aggressive type consisting of florid, bone, Lymphoglandular, anaplastic and visceral type. Since the description of AIDS in 1983 it has been classified into Typical ( classical, endemic) and Atypical ( aggressive, epidemic ) type.

Typical Kaposi's sarcoma includes nodular, plaque and florid cutaneous lesions on the limbs in an adult man of 45 years and above in Africa and 60 years and above in Europe with no associated opportunistic infection, malignant lymphoma, squamous cell carcinoma of the tongue or anus and with a negative HIV

test[3]. The progress of the disease is slow with good response to treatment.

Atypical Kaposi's sarcoma is characterised by fulminating wide spread and atypical location of the lesion both cutaneous and visceral, rapid and generalised lymphadenopathy resulting in severe oedema of the limbs and face. This can virtually involve any organ or system in the body. The HIV test is usually positive. Dawning[18] et al reported 35 to 45 percent of patients with atypical Kaposi's sarcoma to be HIV seropositive while Bayley[11] in Zambia and Taylor[8] in Uganda have reported 90% of patients with atypical Kaposi's sarcoma to be HIV seropositive.

Clinical Presentation

Fifty percent of patients in this study had painless patches, plaque or nodules while in 20% the lesions were preceded by pricking pain and itching. The remaining 30% presented with loss of weight, fever and generalised lymphadenopathy. Pain has been reported to occur late as a result of superinfection or trauma.

Woody oedema of the limbs was seen in 70% of patients while facial oedema was observed in 10% of the patients in this study. Facial oedema is common in aggressive type of Kaposi's sarcoma and was evident in 2 patients (10%) in this study. Generalised oedema is thought to develop due to rapid growth of nodules or plaques invading the subcutaneous tissues and blocking the lymphatics or due to generalised lymphadenopathy. Forty percent of patients who had severe itching, oedema of the lower limbs up to the inguinal ligament and facial oedema were HIV seropositive in this study. This is comparable to a study by Johnson[1] et al where out of 70 patients; 55% had itching, generalised oedema and were HIV seropositive.

There is sequential involvement of the disease whereby the lesions start in the lower limbs and spread over 6 - 12 months and in another 4 - 6 months the upper limbs affected. Patients with oral Kaposi's sarcoma present with flat raised or plaque of dark red or purple colour occurring most commonly on hard palate or tonsils and less often on the gums or posterior pharyngeal wall[3,18]. Presence of nodules in the oral cavity predict presence of similar nodules in the gastrointestinal system from the oesophageal mucosa to the rectum[11,15]. The gastrointestinal tract is involved in 40-50% of patients in epidemic cutaneous sarcoma. Patients presents with abdominal pain, vomiting, diarrhoea, perforation or bleeding[1,19]. In this study no patient presented with oral lesion or gastrointestinal symptoms. Extensive lesion were seen in the anorectal region and genitalia in 2 patients in this study.

Pulmonary manifestations occur late in the disease and heralds onset of terminal phase. It manifests as infiltration endobronchial nodules or as pleural effusions[19].

## TREATMENT

Various treatment modality have been tried but the following seems effective.

### SURGERY

The role of surgery in Kaposi's sarcoma is very minimal. Bayley[11] has claimed an improved general health on removing bulky tumours by either local excision or amputation[18]. Surgery is also indicated for removing a destroying disease -riddled part of a limb for toilet purposes. Occasionally there is a need for laparotomy to deal

with complication such as bowel perforation, peritonitis or mechanical bowel obstruction.

Preoperative radiotherapy or chemotherapy is recommended when contemplating local excision or amputation. In this study none of the patient had surgical intervation as a treatment modality.

Radiotherapy.

This is the main mode of treatment in patients with local disease where the tumour is radiosensitive[20]. The form of treatment can be local radiotherapy, extended field radiotherapy, half body radiotherapy and total body radiotherapy. Local radiotherapy is directed to local Kaposi's sarcoma where a cure rate of 35 to 80 percent has been reported[20]. When used in palliation of obstructing lymphadenopathy, ulcerative or fungating extensive skin lesion, a response rate of 30 percent has been achieved[1,20]. Local radiotherapy is also indicated in solitary lesion or palliation a bleeding or badly destroyed part of a limb.

Extended field radiotherapy involves the irradiation of at least half a limb. The proximal margin at least 15 cm beyond any lesion or oedema. This distance is arbitrary and it does not demonstrate the upper safe limit of the tumour. The dosage used is 800 rads which gives comparable results with higher practitioned doses and carries less complications[20].

The use of half body radiation has been reported by Fitz Patrick and Rider for treatment of advanced cancer[3]. They recommended the use of 800 rads as a single dose to the lower half of the body. The upper half can be irradiated 6 weeks later by 600 rads as single dose. Half body radiation is recommended for lesion

involving more than one limb. In this study 14 patients (60%) were treated with half body irradiation while 3 (15%) were managed by extended body radiation.

Chemotherapy

Chemotherapy is an alternative when a radiotherapy is contraindicated or not available. Taylor[8] claimed good response in same patients given Trenimon 2, 3, 5 Ethylene Imino 1 - 4 benzoquinone, particularly for nodular type while Actinomycin D has been effective in the florid type. Cytotoxic drugs can be given as a single drug or in combination. The ones currently in use include Actinomycin D, Vincristine, bleomycin, Vinblastine, Methotrexate, Cyclophosphamide and Nitrogen Mustard.

Combination chemotherapy has been found to be more effective but is associated with side effect such as immunosuppression and development of opportunistic infection[15]. Extra precaution are necessary when chemotherapy is given to HIV seropositive patients.

Indomethacin and ascorbic acid have been reported to be useful in treatment of atypical Kaposi's sarcoma[18]. It follow a report that high concentration of prostaglandins are found in this tumour cells. Prostaglandins are known to promote tissue permeability and reduce immunity. The two drugs are reported to act by concentrating the prostaglandins activity; however this needs more research[8,18].
Six patients in this study who were treated with combined chemotherapy of Actinomycin D, Vinblastine and Bleomycin showed evidence of tumour regression and disappearance of enlarged lymph nodes.

Immunotherapy

Various immune modulators are currently in use including interferon, thymic hormones, interleukin 11, Gammaglobulin and bone marrow transplantation. Some have shown good results while others have proved futile but all need further research.

Prognosis:

There were four hospital deaths all of them being HIV seropositive. The rest were lost to follow up after initial treatment. Patients presenting with endemic Kaposi's sarcoma have a good chance of responding to treatment ( radiotherapy or chemotherapy ) with a life expectancy of some 8 - 13 years[1,3]. Patients with Kaposi's sarcoma alone without opportunistic infections have about 70% chance of survival for longer than two years, while those with opportunistic infections have less than 50% chance of surviving more than one year.

Endemic Kaposi's sarcoma in children and Epidemic Kaposi's sarcoma particularly in HIV seropositive patients in all age groups have poor prognosis. There is poor response to all forms of treatment and rapid progression of the disease.

Until todate there is no universal acceptable clinical prognostic markers. However, the depletion of $CD_4$ lymphocytes, the decline in antibody such as $P_{24}$ indicate a poor prognosis.

## CONCLUSION

Endemic Kaposi's sarcoma affects mainly elderly men more than 45 years where

epidemic type of Kaposi's sarcoma affects the young men is on rise in Africa in association with AIDS. Endemic Kaposi's sarcoma commonly affects the limbs with a slow progressive course and good prognosis while the epidemic type affect an organ in the body, is rapidly progressive with poor prognosis. The actiology of both endemic and epidemic type is unknown. The recognition of Kaposi's sarcoma in AIDS posses a World wide challenge for epidemiologic, virologic, toxicologic and genetic research.

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# CARCINOMA OF THE URINARY BLADDER


## SUMMARY:

During the period of January 1992 to December 1993, a total of fifteen patients with carcinoma of the urinary bladder were managed by the author at Muhimbili Medical Centre. Eleven of them were males while 4 were females. Their ages ranged from 24 to 72 years, majority being between the 6th and 7th decade. The most frequent presenting symptoms were suprapubic pain, total painless haematuria and frequency of micturition. Eight of the 15 patients studied had previous history of schistosomiasis. All but one patient had T3 or T4 lesions. One was too sick to withstand anaesthesia but vaginal examination had revealed a huge mass anteriorly.

Histology was obtained in 13 patients and 8 of these were squamous cell carcinoma, 3 transitional cell carcinoma; one adenocarcinoma and one anaplastic carcinoma. Most of the patients came from regions around Lake Victoria and Coast region endemic for urinary schistosomiasis. Radiotherapy was used as a palliative therapy to T3 and T4 lesions which accounted for majority of the tumours. Combined surgery and Radiotherapy was only used on two patients. Two patients died before any form of therapy could be instituted.


## INTRODUCTION

Carcinoma of the urinary bladder is a world wide disease. Its incidence varies between different parts of the world[1]. In USA it is the second most common urological malignancy after carcinoma of the prostate[2]. In Malawi, it represents

about 5 - 10% of all malignant tumours[3]. In Tanzania carcinoma of the urinary bladder is the commonest urological malignancy accounting for 40% of all urological cancers and 3.3% of all registered malignancy[4].

While world wide the commonest histological type of cancer is transitional cell type, there seems to be a geographical variation[2]. In Tanzania the most common histological type is squamous cell carcinoma accounting for up to 65% of all cases. Transitional cell carcinoma accounts for 12% of all the cases[4]. Similar histological distribution has been found by Ibrahim[5] in Sudan and Lucas[3] in Malawi. In all these areas squamous cell carcinoma has been found to account for 50 - 80% of all bladder carcinomas. Squamous cell carcinoma of the bladder is common in areas endemic in urinary schistosomiasis which tend to suggest a causal relationship[3,5,6,7]. Exposure to chemicals is associated with transitional cell carcinoma[2]. In most African countries patients report late when disease is advanced and therefore mortality is high[4,5]. In Tanzania many patients with haematuria are repeatedly treated as urinary schistosomiasis at peripheral health units hence the carcinoma is often discovered at a late stage[4,6].

Control of urinary schistosomiasis by mass health education about the consequences of the disease, effective management of the patients and close follow up will reduce the incidence of the urinary bladder carcinoma. In this study, one patient with carcinoma of the urinary bladder is presented in detail. The main features of the 15 patients studied are summarised followed by a discussion and review of literature on pathology, clinical features and management with emphasis on epidemiology so as to establish preventive methods.

**CASE REPORT**

E. M. IP. No. 748792; a 71 years old male patient was referred to Muhimbili Medical Centre from Rukwa Regional Hospital with 8 months history of total painless haematuria, increased frequency of micturition and burning sensation experienced during and after micturition. He had urgency but no incontinence. Two months after the above symptoms he started experiencing dull, non radiating lower abdominal pain with no relieving or aggravating factors. He had no history of bony pain but had lost weight considerably and his appetite was poor. He had urinary schistosomiasis which was treated during childhood. He gave no history of major surgical or medical illnesses apart from the current illness and this was his second admission to hospital. He was a peasant who neither smoked nor took alcohol.

General examination revealed him to be an ill looking, elderly wasted patient, moderately pale but well hydrated. He was not jaundiced and had no pedal oedema. His pulse rate was 88 beats /minute, regular with good volume and the blood pressure was 160/90 mmHg in supine position. He was afebrile with no peripheral lymphadenopathy. The only abnormal finding in the abdomen was suprapubic tenderness. On rectal examination a firm grade one prostate with smooth surface was found. A mass separate from the prostate which could be arising from the bladder was palpable at the finger tip. The gloved finger was stained with normal colour stool. The other systems were examined and appeared normal. A provisional diagnosis of carcinoma of the urinary bladder was made and the patient was investigated as follows:-

1. Urine: A midstream specimen of urine for microscopy revealed numerous

red blood cells with no schistosoma ova. Culture of the urine yielded heavy growth of E. Coli sensitive to Nitrofurantoin, Augmentin and Gentamycin. Cytology revealed malignant cells.

2. Blood: Hemogram: His haemoglobin was 7.4 g/dl and ESR of 35 mm/in the first hour ( westergren ). Biochemistry; gave a serum creatinine of 79 ug/dl and blood urea of 3.5 mmol/l. Serum electrolytes were not measured because the machine was out of order. The above findings are within normal limits.
3. X - ray of the chest, abdomen and pelvis were all normal.
4. Intravenous urography: The kidney and ureters appeared normal. The cystogram revealed a filling defect in the bladder.
5. Cystoscopy: The urethra appeared healthy and the scope could be introduced easily. The median lobe of the prostate was enlarged. There was an ulcerating growth on the lateral wall of the bladder which was easily bleeding. Two biopsies were taken and the tumour was staged T3a Nx Mx on bimanual examination under anaesthesia. Histological results from the biopsies taken revealed well differentiated squamous cell carcinoma of the urinary bladder.

After correction of anaemia by blood transfusion the patient had preoperative radiotherapy. He received a total of 3000 rads given at 300 rads per fraction. He tolerated the treatment well. A follow up cystoscopy after radiotherapy showed the tumour had shrunken considerably and there was no palpable tumour on bimanual palpation. The patient looked weak; had dysuria with increased frequency of micturition. He was also anaemic with haemoglobin of 4.8 g/dl. It took several weeks to correct the anaemia. His condition continued deteriorating progressively

with time. He refused to consent for cystectomy. He was hence discharged for terminal care after his haemoglobin rose to 10 g/dl.

**RESULTS**

The following tables summarise the main features of the patients studied.

Table I: The age and sex distribution

| Age Group (Years) | NUMBER OF PATIENTS | | |
|---|---|---|---|
| | MALE | FEMALE | TOTAL |
| 20-29 | 1 | 0 | 1 |
| 30-39 | 2 | 0 | 2 |
| 40-49 | 2 | 1 | 3 |
| 50-59 | 1 | 0 | 1 |
| 60-69 | 3 | 2 | 5 |
| 70-79 | 2 | 1 | 3 |
| TOTAL | 11 | 4 | 15 |

Fifty three percent of the patients were above 60 years.

Table 2: Presenting Symptoms

| SYMPTOMS | NUMBER OF PATIENTS | % |
|---|---|---|
| Total painless haematuria | 11 | 73.3 |
| Increase frequency | 9 | 60.0 |
| Suprapubic pain | 8 | 53.3 |
| Weight loss | 6 | 40.0 |
| Passing necrotic tissue | 6 | 40.0 |
| Urgency | 4 | 26.7 |
| Dysuria | 3 | 20.0 |
| Incontinence | 2 | 13.3 |

The commonest symptoms were total painless haematuria, increased frequency of micturition and suprapubic pain.

Table 3: Stage of tumour at cystoscopy and examination under anaesthesia.

| STAGE | NUMBER OF PATIENTS |
|---|---|
| Tis | 0 |
| T1 | 0 |
| T2 | 1 |
| T3 (a or b) | 8 |
| T4 (a or b) | 5 |
| Unstaged | 1 |
| TOTAL | 15 |

The majority of the patients had advanced tumour on admission.

Table 4: Histological types

| Histological types | Number of Patients | % |
|---|---|---|
| Squamous cell carcinoma | 8 | 53.3 |
| Transitional cell carcinoma | 3 | 20.0 |
| Adenocarcinoma | 1 | 6.7 |
| Anaplastic carcinoma | 1 | 6.7 |
| No histology | 2 | 13.3 |
| Total | 15 | 100.0 |

Histology was obtained in 13 of the patients and 8 of them ( 53.3% ) were squamous cell carcinoma.

Table 5: Predisposing Factors

| FACTORS | Number of Patients | % |
|---|---|---|
| Urinary Schistosomiasis | 8 | 53.3 |
| Smoking or chewing Tobacco | 5 | 33.3 |
| None | 2 | 13.3 |
| Total | 15 | 100.0 |

Over 50% of patients were treated previously for urinary schistosomiasis.

Table 6: Mode of the treatment.

| TREATMENT | NUMBER OF PATIENTS |
|---|---|
| Curative Radiotherapy(RT) | 0 |
| RT+ Total Cystectomy | 2 |
| Palliative Radiotherapy | 10 |
| Chemotherapy + RT | 1 |
| No treatment | 2 |
| TOTAL | 15 |

The majority of the patients had palliative radiotherapy due to advanced cancer.

## DISCUSSION

Bladder cancer is distributed all over the world. It is more common in industrialized countries than developing ones[7]. There are some 38,000 new cases of bladder cancer each year in the United States with a death rate of 3% of all cancer deaths[2]. This forms about 2% of all cancers in the white population of North America and Europe[1,2]. In Tanzania urinary bladder cancer accounts for about 40% of all urological malignancies and constitutes 3.3% of all malignancies[4,6]. In Zimbabwe[7] and Zambia[8] it constitutes 10.6% and 9.7% of all malignant tumours respectively while in Egypt it is the commonest malignant disease accounting for 20-40% of all malignant tumours[9]. In Africa, a high frequency of bladder cancer was reported from Ghana[3], Nigeria[1,2], Mozambique[4] and in the Bantu in South Africa. Sudan and Kenya have lower prevalence of 3.1% and 0.75% of all reported cases respectively[5,10].

The mean age incidence of bladder cancer is 46 years with a peak incidence in the fifth decade in Malawi and 73% of cases are below the age of 50 years[3].

However bladder cancer is rare if ever, encountered below the age of 20 years. The young age incidence is remarkable compared to non schistosomiasis cancer in which the mean age is 63.5 years with only 12% of cases under the age of 50 years. In this study the peak was between 6th and 7th decade with a mean age of 52 years. This is comparable to a study by Ndoguitha[14] in Kenyata National Hospital where the peak age was the same but the mean age was 49.8 years. The mean age for the bladder cancer in Uganda is 54.8 years; Malawi 44.9 years, while in Europe is 61.2 years[1,3,10]. It has been found that bladder cancer occur about a decade and a half earlier in Africa than in Europe, a finding probably attributable to the association with schistosomiasis in Africa[2,3,5].

The male to female world wide ratio varies from 3 : 2 to 8 : 1 with an average of 5 : 1[2,5]. In our series the male to female ratio was 2 . 8 : 1 as compared to the one found in Kenya which was 4.4 : 1[7]. This difference with a male predominance is probably related to increased exposure of males to schistosomal infection as they work in the fields more than females in some parts of Africa. However in Mozambique, Protas found a ratio of 1 : 1.06 and had a thought that women worked in the field more than males[8].

The causal relationship between schistosoma haematobium and bladder cancer has been speculated since 1911 though a true etiological relationship remains unproven[7]. In Africa areas endemic for urinary schistosomiasis appear to have a high incidence of urinary bladder cancer. The tumour commonly found is well differentiated squamous cell carcinoma[3,8,10]. This association defines a distinct clinicopathological entity quite different from experienced western world where

approximately 90% are basically transitional cell carcinoma and only 5% are squamous cell carcinoma[7]. Some other authors believe that even transitional cell carcinoma and anaplastic carcinoma are all associated with schistosomiasis[7].

Aetiology

A number of environmental factors increase the risk of bladder cancer. Occupational exposure to industrial aromatic amines like beta naphthylamine; 4 aminobiphenyl, 4 nitrobiphenyl and 4, 4 diaminobiphenyl have been clearly demonstrated to be carcinogenic since 1894 by Rehn in Germany. The particular industry involved are the dyeing industries like aniline dye; rubber and electric cable industrics, printing and plastic industries[1].

Bladder cancer occurs among workers after a mean exposure of approximately 20 years accounting for up to 50 fold increased incidence in those exposed. Certain metabolites of tryptophan, kynurenine and some of its close related compounds are excreted in some amount in patients with bladder cancer. Metabolites of Tryptophan enhances chemical induced carcinogenesis. In the body the carcinogens are bound in the liver to glucuronic acid which renders it non carcinogenic. In the urinary bladder it is deconjugated by glucuronidase enzymes and the carcinogens are released in urine. Chemical carcinogenesis in the urinary bladder is important in industrialized countries where the predominant tumour type is transitional cell carcinoma. Chemical carcinogenes play little part in tropical countries and the tumours are predominantly ( up to 75% ) squamous cell carcinoma. A report by Ibrahim[5] et al from Sudan and by Golvan[9] from Cairo are exceptional in that transitional cell carcinoma is commoner than squamous cell ???

carcinoma ( 57.7 and 46.6% respectively ); in these areas with endemic schistosomiasis.

Tobacco tar in cigarette smoke has also been implicated in the aetiology of bladder cancer but this has not been proved. Morison et al on their research also have found no correlation between bladder cancer and smoking[2]. Cyclophosphamide and immunosuppressive agents used in immune diseases cause haemorrhagic cystitis and atypia in the urothelium. This increases the risk of bladder cancer by close to ten times when the agent are used for long time[1]. Phenacetin containing mixtures used as analgesic have been implicated to cause transitional cell carcinoma of the bladder common to patients with "Analgesic abuse Nephropathy"[1,2].

Chronic mechanical stimulation from chronic schistosomiasis infestations or calculosis or chronic irritation by infections may also be involved in the aetiology of bladder cancer. There are several theories put forward to account for the high incidence of urinary bladder cancer associated with schistosomiasis. These are mechanical irritation of bladder epithelium by ova of schistosoma haematobium, chronic inflammatory theory, schistosoma toxins produced by the worms and miracidia and urine retention theory which suggest that carcinogens in urine could have longer time to act due to incomplete emptying as a result of the destruction of the detrusor muscles by schistosoma cystitis. Schistosoma haematobium induce severe squamous metaplasia and proliferative mucosal epithelial changes in the urinary bladder. Sceptics however, have postulated the association to be coincidental.

Beta glucuronidase is one of the hydrolytic enzymes which converts substances in urine into carcinogens. It is increased in renal and vesical infection;

renal cyst, benign enlargement of prostate and urolithiasis hence can be used for diagnostic purposes; however Kalet et al claimed that this enzyme was elaborated by urothelial cells that have been damaged therefore was of no diagnostic significance.

In this study 8 out of 15 patients had sandy patches with few nodules at cystoscopy, a finding which is suggestive of previous schistosoma infestation[1,2].

Pathology

Carcinoma of the urinary bladder may be classified histologically into transitional cell, squamous cell, adenocarcinoma and anaplastic carcinoma[1].

All of these tumours originate from the epithelium. Sarcoma, carcinosarcoma and leiomyosarcoma which are non epithelial or mesenchymal occur rarely in the urinary bladder[1]. In childhood epithelial tumours are rare and when they occur, they are usually benign[3]. In this study 8 patients had squamous cell carcinoma, 3 transitional cell carcinoma, 1 adenocarcinoma and the other one anaplastic carcinoma.

It has been observed that bladder cancers in urinary schistosomial endemic areas differ in clinical presentation, type of tumour, degree of invasion, site of involvement and age of onset from non endemic areas. Cancers from endemic areas are commonly of squamous cell type; the tumour being broad based, nodular, fungating and infiltrative. Majority of these arise directly from the bladder mucosa or are papillary carcinoma in which metaplasia has taken place or from leucoplakia. Twenty five percent have multiple tumours and rarely arise from the trigone. The majority occur in the 4th and 5th decades of life[1,2].

Cancers which occur in non endemic area for schistosomiasis usually are

transitional cell type; the tumour are pedunculated and papillary projecting into the bladder cavity. Most often they arise from the trigone and the majority occur in the 6th or 7th decade of life; one to one and half decade later than cancers occurring in endemic areas[1,2].

Cytologically it is possible to follow the changes of malignant transformation from hyperplasia - dysplasia stage through carcinoma in situ to invasive tumour[1]. Classification of bladder cancer varies from one country to another. Every country adopts a system which will help them to select treatment modalities and determine prognosis[11].

The extent the tumour cells have differentiated correlates very closely with the degree of malignancy. Grading of the tumours classifies them according to the degree of anaplasia and this influences the prognosis. The malignancy can be graded as well differentiated (G1), moderately differentiated (G2) and poorly differentiated (G3) or undifferentiated[11].

The well differentiated tumour cells have reverted least from normal epithelium and have good prognosis while the poorly differentiated tumour cells have deviated most from normality and have poorest prognosis. Moderately differentiated tumour cell are in between. Staging of the tumour is important for determining the treatment and prognosis as well as for comparison of results from different institutions. The urinary bladder tumours are staged at cystoscopy and bimanual palpation under anaesthesia. There are several staging procedures, started by Jewet and Strong (1946), modified by Marshall (1973) and the TNM classification.

A comparison of the above staging systems and their correlation to clinical palpation is as summarised.

| UICC /TNM | Biopsy finding | Bimanual palpation | Jewet/ Strong 1946 | Marshall 1973 |
|---|---|---|---|---|
| Tis | Ca in situ (Pre-invasive). | Normal bladder. | O | O |
| T1 | Tumour doesn't extend beyond the lamina propria (Confined to submucosa). | Small mobile bladder mass. | A | A |
| T2 | Tumour involving superficial detrusor muscle. | Mobile mass; indurated bladder wall. | B1 | B1 |
| T3a | Tumour involving deep muscle (full thickness of detrusor muscle). | Distinct nodular hard/Firm, indurated mobile mass. | B2 | B2 |
| T3b | Tumour extension to involve paravesical tissue. | Distinct nodular hard/firm, indurated mobile mass. | C | C |
| T4a | Tumour invading adjacent structure, prostate, seminal vesicle, rectum, uterus, vagina. | Tumour fixed to the prostate, vaginal or pelvic wall. | D | D1 |
| T4b | Tumours fixed to the pelvic wall or infiltrating to the anterior abdominal wall. | Tumour fixed to the prostate, vagina or pelvic wall. | D | D2 |

Lymph node involvement is classified as follows:

No : No lymph node involvement

N1: Homolateral lymph node involvement

N2: Contralateral or bilateral lymph node involvement.

N3: Fixed mass on pelvic wall separate from the bladder.

N4: Involvement of iliac and paraaortic lymph nodes.

The metastatic status is classified as follows:

MO: No evidence of metastases.

M1: Metastases present.

MIa: Occult metastases

MIb: Single metastases in a single organ

MIc: Multiple metastases in a single organ

MId: Metastases in multiple organ sites.

Bladder tumour spreads in four ways namely; tentacular invasion, enbloc invasion, lymphatic and vascular invasion. In tentacular invasion, the carcinoma infiltrates normal structure in the form of small strands with scattered pattern. In enbloc invasion the tumour form large cohesive carcinoma and is associated with a more favourable prognosis[1]. Lymphatic and angioinvasion are commonly observed in high grade tumours and indicate poor prognosis. The lymphatic spread involve the hypogastric; external iliac, common iliac and aortic nodes first followed by paraaortic and mediastinal lymphnodes. In haematogenous spread; the cancer cells present in blood are swept in the lungs; liver, bones and brain. In some instances blood borne metastases occur earlier than lymphatic spread.

Clinical presentation and Diagnosis

Haematuria is a cardinal symptom in patients with bladder cancer. Blandy has reported haematuria to be present in 80% of all patients with bladder cancer. Total painless haematuria, increased frequency of micturition and suprapubic pain were the most frequent presenting symptoms where there were 11, 9 and 8 patients

respectively out of 15 studied. Similar findings have been reported by Ndoguitha in Kenyata National Hospital[10]. Other symptoms in order of decreasing frequency were weight loss, pyuria, passing necrotic tissue, urgency and incontinence. Frequency and urgency are due to reduced bladder capacity and sometimes superadded infection causing chronic cystitis[11].

The majority of the bladder tumours grow in close proximity to the ureter. Urinary tract infection is hence common leading to fever, loin pain with renal angle tenderness. Obstruction at the ureteric opening may lead to hydroureter, hydronephrosis and uraemia[10].

Some patients have a lower abdominal swelling at presentation and in advanced disease the patient may present with features related to distant metastases. In early cases, physical findings are minimal since the tumour remains within the bladder. In this series most patients presented late when they had anaemia, wasting and dehydration. Seventy five percent of the patients had an obvious palpable suprapubic mass, a finding similar to the one reported by Retchie and Skinner[16]. Vesicovaginal fistula is very rare and was not found in this study. Cuscheri consider it common to local tentacular spreading types of tumour[11]. Urine for microscopic analysis and culture determine the presence of infection, microscopic haematuria and schistosoma ova.

Exfoliative cytology is superior to urine cytology in screening and follow-up of the patients[12]. Cystourethroscopy is the cornerstone in the diagnosis of bladder cancer[1,5]. It allows visualization of the whole bladder cavity. A record of position, size, number of tumour, degree of fronding, irregularity and shape can be obtained.

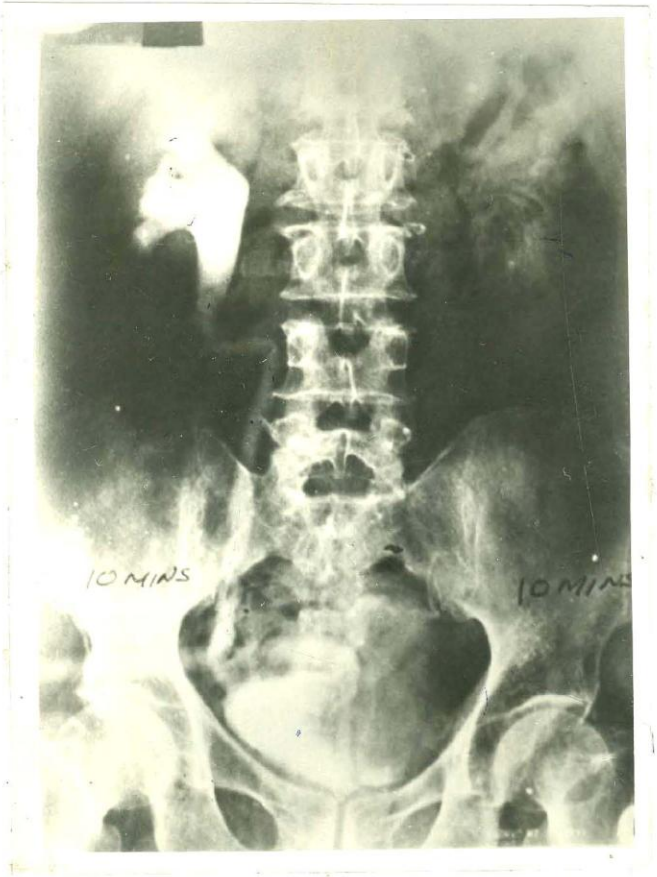
Biopsies taken will confirm the histological diagnosis. Multiple biopsies are taken at cystoscopy in different suggestive sites as postulated by Blandy[1]. He further suggested that a negative biopsy in the presence of an apparent carcinoma should be repeated. Bimanual palpation of the empty bladder under general anaesthesia is done after cystoscopy to determine the size and extent of the tumour. This enables one to stage the bladder cancer and make a plan for therapy.

Serum creatinine is important to assess renal function in case of obstruction, a common complication in bladder cancer. A plain abdominal x-ray may reveal a suspicious soft tissue mass or calcification of bladder due to chronic schistosomiasis.[9] Currenthers reported bladder calcification in 47% of patients with chronic schistosomiasis[13]. No patient was found to have calcification of the bladder even among those with previous history of schistosomiasis in this study. The chest x-ray may demonstrate lung metastasis or other lesions.

The intravenous urogram (I.V.U) is an essential investigation. However in our centre it is limited to selected patients due to shortage of facilities. It may show bladder tumour, diverticulum, dilation of the upper urinary tract ( hydroureter and hydronephrosis Fig. III ), ureteric tumours or damage to the kidneys. IVU's showed unilateral or bilateral hydronephrosis in two patients in this series. Currenthers[13] has reported normal kidneys and ureters in 10% of his cases while Retchie[11] found 4 out of 37 IVU's were normal in his series.

The degree of muscle infiltration and staging of the tumour can be done by ultrasonography and computerized tomography. For evaluation of distant metastasis; liver and bone scans are of value. Carcinoembryonic antigen (CEA) can be used as

Figure III: IVU in a patient with cancer of the urinary bladder.




There is hydronephrosis, hydroureter and kinking on the right side. The left side show no excretory function. There is a filling defect in the bladder, the probable cause of ureteric obstruction.

a tumour marker for early detection of cases, surveillance and detection of recurrences[12]. High levels of carcinoembryonic antigens are found in urine of patients with urinary bladder cancer. It has been shown that clinical assessment, radiological and laboratory investigations are best used together for diagnosis rather than relying on any one of them in isolation[10,11]. This will enable one to differentiate carcinoma of the bladder from other conditions which resemble it in presentation like urinary schistosomiasis, bladder stones, tuberculosis of the bladder, chronic interstitial cystitis and secondary bladder tumours which spread directly from adjacent organs such as the prostate, cervix and rectum.

**TREATMENT**

The treatment of bladder cancer can be curative or palliative depending on the stage of the tumour, the grade and histological type of the tumour, the number of lesions, the age of the patient, and general condition of the patient[2]. There are various modality of treatment which include surgery, radiotherapy, chemotherapy, immunotherapy, thermal and mechanical therapy. It is unfortunate that none of the different modalities of treatment offer an outstanding success as a single entity except in combination. For the purpose of determining treatment, bladder cancer can conveniently be grouped into three categories: Carcinoma in situ (Tis), Superficial (non-invasive) carcinoma, invading no further than lamina propria (T1) and invasive carcinoma (T2 - T4).

(I) Carcinoma in Situ (Tis)

The treatment of carcinoma in situ still remains controversial because its

natural history is unpredictable. Several studies have shown it to have high recurrence rates up to 80% after treatment. The study done on various site of the bladder after cystectomy have shown that the epithelium display atypia with mucosal hyperplasia, the changes which are sometimes not visualized by endoscopy[12].

An extensive study on treatment of carcinoma in situ was done by Utz, Farn and Sejura[14]. They treated their patients by transurethral resection and fulguration. Seventy five percent of their patients had subsequent invasion while more than half died within 5 years. Remission rates ranging from 50 to 90 per cent have been achieved using intravesical chemotherapeutic agents like Thiotepa, Epodyl, Mitomycin, Cisplastin or Doxorubicin hydrochloride[15]. Due to variability of latency interval before invasion of the cancer some surgeons prefer conservative management using intravesical chemotherapy rather than radical treatment such as cystectomy.

Skinner and colleague have finally put the following recommendations for treatment of carcinoma in situ of the bladder based on studies by Utz and his colleagues[16].

(i) Electrofulguration and intravesical chemotherapy

This combination of treatment is limited to small lesions in the bladder which does not involve the prostatic urethra, ureteric orifice or bladder neck. It is then followed by intravesical chemotherapy using either of the following; Thiotepa, Epodyl, Mitomycin C, Bleomycin or Doxorubicin[16]. Routine follow up is done for recurrences at three months intervals for the first year, six monthly for the next two years and annually for the following years.

(ii) Radical cystectomy and urinary diversion

This is recommended when there is poor response to above therapy evidenced by persistence of symptoms, positivity of the cytology and evidence of extension to the bladder, neck, prostatic urethra or ureteric orifices.

(iii)Intravesical BCG Immunotherapy

Bacillus Calmette Guerin (BCG) instilled intravesically has shown favourable therapeutic response on diffuse carcinoma in situ of the bladder. Maaz did a study on 139 patients by giving 200 ampoules of 150 mg of BCG dissolved in 75 mls of sterile normal saline instilled into the empty bladder via a urethral catheter. It was a six month course and followed them 3 - 6 monthly for 2 years. Complete response up to 5 years was seen in 52% of the patients while no response was observed in 48% of the patients[17].

BCG enhance host defence to malignancy. The effectiveness however, depends on ability of the host to react on mycobacteria antigen, size of tumour load, adequate number of living bacilli and closeness of their contact with tumour cells. Bladder irritation, haematuria, fever, nausea, arthritis, BCG pneumonitis and hepatitis are some of the side effects. Most of the patients studied rarely presented early with carcinoma in situ where they could benefit from the above regimen.

(II) Superficial bladder tumours (Non invasive (T1)

These are the tumours which do not extend beyond the lamina propria and are limited to submucosa(T1). It is the commonest stage of presentation in non bilharzia bladder cancer and carries a good prognosis. Most of these tumours can be treated by endoscopic resection or electro-coagulation. All visible or suspicious

lesions are resected up to the level of bladder mucosa in order to assess microscopically the presence or absence of infiltration. This enable one to have histological tumour characteristic of the resected part which is of prognostic significance. Recurrence rate of up to 80% have been reported especially to those with multiple tumours following transurethral resection[12,15]. The survival rate is just 70% at 5 years[15].

Intravesical Thiotepa or Epodyl have been reported to produce complete regression in 40% of small superficial tumours; if this fails, then cystourethrectomy can be employed and this has a 60% five year survival[15,17]. Immunotherapy with intravesical BCG has been shown to be effective in these tumours also[17]. For tumours that are inaccessible for resection or patients unfit for surgery; the use of external beam radiation with 6000 rads given in six weeks have shown good results[18]. The use of Doxorubicin with urokinase, a plasminogen activator which enhance cytotoxic drug penetration and promotes intracellular release of lysozymes hydrolase which destroy internal cell structure have shown good results in recurrences[19]. There was no patient seen at this stage of disease in this study series.

(III)Invasive carcinoma (T2, T3 and T4)

These are the tumours which have infiltrated the superficial muscle layer (T2), deep muscle (T3a), paravesical tissue (T3b) and involve adjacent organs/pelvis (T4). Over 80% of the patients studied were at stage T3 - T4 while 6.7% were having T2 lesion.

Several studies done on treatment of invasive bladder tumours have shown the response to be generally poor where 50% of patients die within 2 years[20]. When

surgery or radiotherapy is used separately the 5 year survival rates for each therapy ranged from 15 to 35%, but when used in combination, the 5 year survival ranged between 30 - 50%[16].

At our centre the two main modalities of treatment available are surgery and external radiation. Thirteen out of 15 patients in this series were treated either by surgery, radiotherapy or in combination. Follow up was inadequate because after treatment they were discharged back to their home regions and most of them did not return back for follow up.

T2 Bladder tumours

There are various modalities of treatment, but the most accepted one is that advocated by Badoe and colleagues where there are three modalities[12].

(i) Partial segmental cystectomy

This involves resection of a localized tumour with a good margin of not less than a centimetre of normal tissues leaving a functional bladder capacity. It does not involve the bladder base hence has a high chance of inadequate tumour clearance. Pre-operative irradiation is advocated to prevent tumour recurrence.

(ii) Simple cystectomy

This is advocated in large tumours or in those tumours located in the bladder base with grade I and II. Preoperative irradiation should be given to Grade III tumours.

(iii) Radical radiotherapy

The results of this treatment alone is not encouraging and have shown severe

side effects like diarrhoea, nausea, perforation of the bowel and contraction of the bladder[16].

T3 Bladder tumours

This is the bladder carcinoma which has involved the whole muscle layer and perivesical fat. Skinner and Ridlles have advocated three modalities of treatment which is accepted in many places.

(i) Radical Cystectomy

This involves total cystectomy with pelvic node dissection. The surgery in males include, prostatectomy with removal of seminal vesicles with / without urethrectomy. In females, the operation includes hysterectomy with removal of the major part of the vagina. The five year survival rates after radical Cystectomy has been reported to be 25%[16].

(ii) Radical radiotherapy

Similar survival rates of 25% has been claimed[20].

(iii)Combined radical cystectomy and radiotherapy

The five year survival rate has been shown to be more than 50% in combined radical cystectomy and radiotherapy than when one mode of treatment is used alone. Experience from the above mentioned mode of therapy is that; radical cystectomy should be followed by urinary diversion which is associated with high morbidity and mortality. New operative technique such as radical cysto-prostatectomy combined with radiotherapy have improved the condition. Also, preoperatively irradiated patients have shown poor healing of their ureters. A new method of bringing the ureters to the skin instead of internal anastomosis has

improved the situation although skin excoriation, encrustation, chronic irritation and hyperkeratosis with stomal stenosis is a problem. The technique of urinary diversion via a continent ileal reservoirs (koch pouch) is favoured[20].

In our centre radical cysto-prostatectomy with ureterosigmoidostomy or ureter anastomosed to the rectum is practised despite the potential for the complications of pyelonephritis, ureteric obstruction, nephrolithiasis, electrolyte imbalance, systemic acidosis and primary neoplasm at the site of uretero-sigmoid anastomosis.

T4 Bladder tumour

This is the bladder carcinoma which has spread to adjacent organs such as the prostate, rectum, vagina or bony pelvis. It is an advanced stage in which whatever modality of treatment given is aimed at palliating symptoms and making the patient as comfortable as possible[21].

Local palliative radiotherapy can be used to arrest bleeding and local bone pain. Local radiation however causes contraction of the bladder and frequently makes the condition worse. Some authors treat T4 bladder tumours as T3 especially those involving the prostate but not other viscera or pelvic wall.

Forty percent Formalin instilled in the bladder for 15 minutes followed by 10% alcohol and normal saline have been used to control intractable haematuria. Helmstein balloon therapy has been used for the same purpose.

Good results in this stage of disease have been obtained by combining radiation and chemotherapy. There is however no agreement on which agents are the most effective. Adriamycin, 5 Fluorouracil, Mitomycin C and Methotrexate have been used with success and give some hope for the future.

## CONCLUSION

Cancer of the urinary bladder is a common problem in Tanzania especially in areas endemic for schistosomiasis. Most patients present late to hospital where they end up with palliative treatment in the form of radiotherapy. Doctors are cautioned to have a high index of suspicion in those patients who present early with haematuria and symptoms of bladder irritation. Such patients should be investigated thoroughly including urine for cytology and cystoscopy. Emphasis should be given on prevention of schistosomiasis which is the major contributing factor. Radiotherapy has proved to be good in advanced bladder cancer.

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# PRIAPISM


## SUMMARY:

During the period of January 1992 to December 1992, six patients with priapism were studied. Their age ranged from 16 to 62 years. One patient was suffering from sickle cell disease (SCD). In 4 patients the predisposing factors could not be established and one patient was found to have AIDS. The period from onset of symptoms to presentation to hospital ranged from 6 hours to 7 days. Four patients were treated by Cavernoso-Spongiosal shunt using Tru-Cut biopsy needle while the other 2 patients had Carvenoso-Saphenous shunt.

Among the 3 patients who were available for follow up to six months, one regained erectile potency. Complications were encountered in 2 patients. One patient died from severe sepsis with gas gangrene seven days after shunt procedure and was proved to be HIV seropositive. The other had urethro-cutaneous fistula and necrosis of part of the glans penis.


## INTRODUCTION

Priapism is defined as a state of sustained penile erection quite often painful without any evident sexual desire or ejaculation[1,2,3]. The penile erection fail to subside, even after climax[4,5]. This medical condition was named after Priapus, a Greek who was generally regarded as a God of fertility. Recently, increased appreciation of the pharmacology and hemodynamics of erection, and the introduction of intracavernous injections have compelled a broadening of this definition to persistence of erection for more than four to six hours[6].

These pathological changes in the corpora cavernosa have been initiated by changes in pH and oxygenation[6]. Prolonged unrelieved priapism almost inevitably leads to subsequent permanent impotence from fibrosis of the corpora cavernosa[1]. It is a condition which requires immediate treatment. A wide range of surgical and medical treatments has been attempted for priapism. Because of development of fibrosis which prevents subsequent erections, surgery has now an ever diminishing place and medical means are becoming more popular[1]. In this paper a series of 6 patients were seen. One of the patients is selected for presentation. A review of literature on the aetiology, pathophysiology, clinical presentation, investigation and treatment is also presented.

## CASE REPORT

Z. P. Hospital number 737059, a 31 years old male was seen on consultation in the medical ward where he had been admitted with complaints of persistent painful erection of the penis for one week.

This was his first episode and was not preceded by coitus, trauma or fever. He had no history of taking alcohol or any drugs. He was married with two children. He did not drink alcohol or smoke cigarettes. There was an attempt to achieve detumescence by local herbs and sedatives, all proving unsuccessful.

On examination, his general condition was fair. He was asthenic and had moderate dehydration. He was not pale, not icteric and was afebrile. There was no evidence of cyanosis, lymphadenopathy or generalized oedema. The respiratory, central nervous and cardiovascular systems were normal; although slight tachycardia

was noted with a pulse rate of 100 beats per minute, regular and of normal volume. His blood pressure was 140/90 mmHg. The abdomen was scaphoid, moving with respiration. There was slight suprapubic tenderness. The liver and spleen were not palpable. There was no other mass felt in the abdomen. Bowel sounds were normal. He had an erect penis which was oedematous and tender. The scrotum and testes were normal; and a rectal examination revealed normal findings. A diagnosis of Priapism was made. The treatment given in the medical ward consisting of Pethidine, Diazepam and ice water enema was unsuccessful. Preparation was started immediately for emergency shunt procedure. An intravenous drip was started and prescription made for infusion of 2 litres of normal saline in 2 - 3 hours. Blood was drawn at the same time for haemoglobin estimation.

Under general anaesthesia with endotracheal intubation, the patient was cleaned, draped and catheterized. An incision of about 8cm in length was made below and parallel to the medial half of the inguinal ligament at the level of the saphenous opening. Fat was separated by blunt dissection in the line of saphenous vein. The vein was cleared up to 10 cm below the opening. Each of the tributaries from saphenous trunk was careful ligated and the vein was distended with heparinised saline while making sure that any fascial band on its adventitia was not obstructing the lumen. A tunnel was made under the fat between the fossa ovalis where the saphenous vein enters the femoral vein and the root of the penis. In this area, through a separate incision, Buck's fascia was exposed and a window was cut away. The distal free end of the saphenous vein was anastomosed to an elliptical window on corpus cavernosum using 5/0 Nylon after the black current like blood had

been squeezed out and the corpus irrigated with normal saline. The shunt was proved to be functioning and the skin was closed with silk 3/0.

Post operatively the patient was monitored routinely and he was given Ampicillin for five days. His recovery was uneventful. On the third post operative day, the penis was semiflaccid and the catheter was removed. On the eight post operative day; he had improved significantly and his penis was becoming soft. He was discharged home on the tenth post operative day after removal of stitches. He was followed up two weekly for the first three months after discharge where he was found to be doing well except potency was not established. He was later lost from follow up.

## PATIENT ANALYSIS IN SUMMARY

| Patient Hosp. No. | Age in Years | Presenting symptoms /signs | Duration from onset of symptoms to time of admission hrs/days | Treatment | Complication / follow up |
|---|---|---|---|---|---|
| 698473 | 52 | PPE+UR +WL | 7 days | Cavernoso-Saphenous shunt | Sepsis with gangrene, died 7 days after operation. HIV positive. |
| 737059 | 31 | PPE | 5 days | Cavernoso-Saphenous shunt | One week semi flaccid penis, 3 months had not established potency. Lost from follow up. |
| 750387 | 62 | PPE | 4 days | Cavernoso-Spongiosal shunt | Had not established potency after one week. Asked a referral back to Bukoba for social reason. |
| 730311 | 16 | PPE+UR | 7 days | Cavernoso-Spongiosal shunt | One week semi flaccid penis, 6 months had not established potency. Had urethrocutaneous fistula. |
| 768815 | 17 | PPE | 6 hours | Cavernoso-Spongiosal shunt | Had flaccid penis after two weeks, 6 months had not established potency. |
| 677059 | 22 | PPE | 6 hours | Cavernoso-Spongiosal shunt | Detumescence was achieved within 5 days. He was known to have sickle cell disease. Potency was established in 3 months. |

**KEY**

**PPE**: Persistent painful erection **UR:** Urine retention **WL:** Weight loss

All patients presented with persistent painful erection. Two of them had urine retention. Complications noted in 2 patients were sepsis with gas gangrene in AIDS patient and urethrocutaneous fistula.

## DISCUSSION

Aetiology and Pathology

The initiating event in priapism whether neural, vascular or mechanical is not known[2,5,7]. What may be described, however, are the many clinical settings in which this condition has been observed. Congenital neonatal priapism has occurred idiopathically and has association with birth trauma, forceps delivery, umbilical artery catheterization, polycythemia, respiratory distress syndrome and congenital syphilis as reported by Hinmen et al[1,3]. Some authors postulate the cause to be increased viscosity as a result of vascular stasis and increased carbon dioxide tension.

Inflammation and irritation of the urethra, prostatitis and mumps orchitis are documented causes of priapism[4]. It is believed that distension of the seminal vesicles is a compromising factor. Urethral instrumentation and prostatic massage have been mentioned as initiators of priapism. Testicular inflammation may lead to oedema of the connective tissue, dilatation of the blood vessels and thrombosis of prostatic plexus thereby resulting in congestion and tumescence. Some cases of priapism have been noted in association with phlebitis or in pelvic abscess[9].

Haematological disorders such as leukaemia, thalassaemia and sickle cell anaemia have been known to cause priapism[9]. Macciota observed priapism in a ten years old boy with myelocytic leukaemia, the condition having existed for five weeks and having increased by voiding. The cause of priapism in leukaemic patients has been postulated to be due to haemorrhage into cavernous tissue or mechanical obstruction of the blood vessels of the corpora cavernosa and dorsal vein of the penis

with leukaemic cellular debris. Levant and Strept reported 10 cases in 102 male Negroes admitted in hospital with sickle cell anaemia[7,8]. Deformation and eventual destruction of the red cells leads to anaemia and vascular occlusion which is responsible for priapism.

Muro in his series found 10 percent of patients with priapism secondary to spinal cord injuries. He postulated further that the higher the lesion, the more commonly priapism occurs ranging from 14 percent in the cervical spine injuries to 3 percent in cauda equina injuries[7].

Priapism secondary to infiltration of the base of the penis by carcinoma has been reported. Hann and Weinberg on reviewing 55 cases found 5 cases of priapism due to infiltration of the penis by cancer[4,7]. The primary site commonly were from the bladder, rectum, kidney, testis and lungs.

Pryor and Pelouse have mentioned the possibility of cerebral malaria with resultant cerebral thrombosis as an etiological factor. This is due to the association between minimal brain damage and abnormal erectile activity[6,9]. This theory however needs further research.

Overdosage with certain drugs notably methyl testosterone and testosterone propionate have been noted to cause priapism. The other agents implicated are administration of cantharis and anticoagulant drugs like heparin and coumarin[10].

In Sweden alcohol abuse is reported to be one of the commonest etiological factors of priapism[11]. Some patients on Total Parenteral Nutrition (TPN) have also been noted to get priapism. Klein et al in 1985 postulated that a fat emulsion stronger than 10% (especially 40%) accelerates thrombin production leading to a

short coagulation time. Increased erythrocyte adhesiveness and aggregation lead to decrease or even absent capillary blood flow[12].

In most of the patients in this study with priapism, none of the etiological factors are operative except in one patient with sickle cell disease. Hinman Jor has termed them "idiopathic priapism". They usually have a previous history of prolonged erection that have subsided. The presenting bout is precipitated by continued and vigorous intercourse. However, none of the patients presented volunteered history of intercourse preceding the priapism.

Clinical features and Diagnosis

Priapism can occur at any age owing to the varied etiological factors. The main symptom is painful erection. There may be a short period at the onset where the erection is pleasurable but evernescence of the sensation is rapid[2,3,4]. Urinary difficulties are common in cases where erection of the corpus spongiosum is included in the process. Grace[3] in his appraisal of management of 23 patients found all presenting with persistent painful erection and 30% with urine retention. In our study all patients presented with persistent painful erection, while 2 out of 6 had urine retention. Prolonged pain especially perineal pain which occurs in priapism is associated with urinary difficulties[8]. Thrombotic crises can occur in patients with sickle cell disease and sometimes affect the penis causing priapism as evidenced in one case of the patients studied. Similar findings have been reported by Haq[2].

Diagnosis depends on history and physical findings together with laboratory examination. Haematological investigations include total white blood cells count and differential count where leukaemia can be diagnosed. It is confirmed by bone

marrow aspiration with analysis of the cells. Sickling test with Hb electrophoresis is important in diagnosis of sickle cell anaemia[3]. VDRL test is indicated when congenital syphilis is suspected[21]. Urine for culture and sensitivity is beneficial when priapism is suspected to be caused by urinary tract infection. Haq and Mesink in their series were able to establish the cause of priapism by the above investigations[3,6]. Special investigations such as Radioactive fibrinogen test and phlebography to confirm venous thrombosis have been used elsewhere with good results though they have side effects. Cavernosogram is another investigation used to trace the possible outflow obstruction due to thrombosis or metastasis. Blood gas analysis from the corpora cavernosa is important to establish the severity of priapism or the degree of ischaemia. Van Driel et al in their series of 8 patients studied 6 of them who were found to be acidotic and there were ischaemia of the corpora cavernosa[6]. The blood gas analysis is of prognostic value in establishing erectile function.

## TREATMENT

Priapism is a surgical emergency[1]. The immediate goal is to improve venous drainage of the corpora cavernosa permitting detumescence of the penis and preventing ischaemia, fibrosis and impotence[1]. Since the outcome of treatment is uncertain; it is important to counsel the patient on the possibility of failure to preserve potency. There are two modalities of treatment; conservative and surgery.

### Conservative therapy

Since some of the causes of priapism are known, the first step is to correct

any underlying cause[14]. Patients with sickle cell disease should be treated with oxygen, infusions for hydration, alkalinization using sodium bicarbonate and transfusion if necessary. Packed red cell transfusions have largely been successful because of dilution of sickle cells with normal cells. One patient in this series who had sickle cell disease could not improve on conservative means.

Patients with priapism secondary to leukaemia often respond promptly to chemotherapy and splenic irradiation. Haar et al postulated that direct penile irradiation is often effective for priapism due to malignant infiltrates[10]. Systemic infection is usually controlled by appropriate antibiotics while pelvic infection in addition may require debridement and drainage.

Bladder catheterization is often necessary as some patients present with urine retention. Two patients in our study needed catheterization. A variety of non invasive measures other than the above mentioned have been practised with some success. These include, prompt sedation without analgesics initiated by Hasen and Raines[10,15].

Hot or cold enema (ice water) have been reported to be beneficial[15]. Haisen and Raines did several studies on spinal caudal and general anaesthetic drugs and have found that Ketamine hydrochloride cause rapid detumescence in priapism. This was noted during repair of hypospadiasis. They showed that Ketamine hydrochloride was successful in reversing early cases of priapism in approximately 50% of the time[10]. Some other authors have observed that spinal or caudal anaesthesia are ineffective and have lost favour[13].

Pelouse[9] suggested the use of anticoagulant therapy to minimize the risk of venous thrombosis. Intravenous administration of Heparin with a 'loading' dose of

100mg followed by 50mg six hourly is suggested while others insist on irrigation with a mixture of Heparin and Saline[15]. Some other authors disagree because there is apparently no evidence to suggest clot or thrombus formation with conversion of fibrinogen into fibrin[12]. The use of fibrinolytic agents, thrombolysis, vasodilators, dextran and stilboestrol are controversial and need further study. Other drugs which have been proved to be useful are antiandrogens and adrenocortical hormones.

Van Driel et al have advocated the use of Adrenaline injection into corpora cavernosa of the penis[6]. An injection of 0.01mg Adrenaline diluted in 10mls of 0.9% sodium chloride is slowly injected intracavernously, while monitoring pulse rate and blood pressure. Aspiration is usually done before injecting Adrenaline. In 6 cases out of 8; the priapism was successfully controlled after drainage followed by injection of the Adrenaline solution. Adrenaline is freely available, however its potential disadvantages is that it has both alpha adrenergic and beta adrenergic effect which may cause increase in the pulse rate and extrasystole. That is why some authors discourage its use, and prefer the use of purely alpha adrenergic sympathomimetic drugs such as phenylephrine[14,15]. Adrenaline has however the advantage of having beta adrenergic properties on venous wall where it promotes outflow.

Surgery

Surgical treatment, if all else fails, is effective in relieving stress. There are several surgical methods:

1. Aspiration

This is the procedure where viscous stagnant blood is aspirated using a large

bored needle placed directly at the corpora. While aspirating the corpus is irrigated with plain or heparinized saline or hyaluronidase enzyme. It is a preferred technique in emergency since it is simple and avoids scar formation[6]. Harrow recommends that aspiration of the corpora cavernosa at the base of the penis should be combined with gentle massage of the shaft towards the base to express the venous blood, followed by application of a T binder[16]. Other authors are against the use of heparin because it has been implicated in the pathogenesis of the disease[15]. Using this method; Harrow[16] in his series of 30 patients with priapism was able to achieve detumescence in 50% of the patients seen within 6 to 8 hours after onset of priapism. No patient in this study series was treated by this method.

2. Cavernoso-Spongiosal Shunt

This technique was described by Winter in 1978. It is done by using a Tru-Cut (Travenol laboratories, Dearfield, Illinois, USA) biopsy needle which is passed through the glans penis to remove a circle of fascia covering the obstructed bulging part of the corpus cavernosum[16,17]. It is a simple procedure creating a communication between the tense and distended corpus cavernosum and the flaccid glans and its adjacent corpus spongiosum. It allows drainage of the trapped venous blood. The procedure is repeated on the other side. Al Ghourab proposed a modified procedure utilizing a rongeur under direct vision permitting the creation of a large shunt[2]. Bueschen in a series of 55 patients used this shunt procedure with a success rate of 77.5%[7]. Three patients in our series were treated by this method and in all detumescence was achieved.

3. Cavernoso-Saphenous Shunt

Grayhack and associates have devised an operation corpus cavernosum-long saphenous vein shunt which relieves the venous stasis and prevents initiation of long series of events leading to corpora cavernosa fibrosis and impotence. The procedure is as follows[10,15];

The sapheno-femoral junction is exposed through an incision 5 to 8 cm in length below and parallel to the medial half of the inguinal ligament, 3 - 4 cm below and lateral to the pubic tubercle. Fat in deep fascia is separated by blunt dissection in the line of the saphenous vein. The vein is dissected out for 10 cm inferiorly, its tributaries being carefully separated and ligated. The vein is distended with heparinized saline while making sure that any fascial bands on its adventitia are not obstructing the lumen. A tunnel is made under fat between the fossa ovalis where the saphenous vein enters the femoral vein and the root of the penis. There, through a separate incision, Bucks fascia is exposed, a window is cut away and the end of the vein is anastomosed to the fascial edge with 5/0 Tevdek or 7/0 Proline. The penis is irrigated thoroughly with heparinized saline to eliminate the accumulation of thick sludge before anastomosis. The immediate effect of this procedure if done within 36 to 48 hours of priapism, is to allow the penis to collapse. There is however no guarantee that the procedure will work the first time and hence one must be prepared for a repeat procedure or use contralateral saphenous vein. Two patients out of six in our series were treated by this method and detumescence was achieved in one.

After this procedure usually the penis remain thickened and semirigid. Fibrosis occurs in the septa of the corpus cavernosum and the normal erectile system

may never function properly again. Usually the bypass become blocked within a week but occasionally it is patent and may have to be tied off to allow normal erection to return[10].

4. Corporo - Corporal Anastomosis:

This is another shunt procedure where a short midline incision is made near the base of the penile urethra[15]. Dissection is done in the plane between the corpus spongiosum and the hard, distended corpus cavernosum. A generous window about 1 cm x 1 cm 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 both windows are anastomosed using very fine non absorbable suture material eg. 5/0 Tevdek (Davis and Geck) or 7/0 Proline (Ethicon, UK). Skin is closed with interrupted 4/0 catgut mattress sutures. Blandy[1] in his series of 11 patients with priapism, 3 achieved detumescence with the procedure. No patient in this study who was treated by this method.

Complications.

Some of the complications of these procedures are: Urethrocutaneous fistula, urethrocavernous fistula, painful spongiosum on erection, pulmonary embolism, gangrene or necrosis of the distal penis, skin necrosis, haematoma, wound infection, abscess and erectile deviation. These complications are also evident in other shunt procedures[10,15,16]. Two patients in this study developed complications. One patient had urethrocutaneous fistula and the other developed necrosis of the distal glans

penis with gas gangrene.

Potency is a major concern in evaluating the results of treatment of priapism. The length of time from onset is of paramount importance[13]. Cosque et al found an impressive sixty one percent of 75 patients who regained potency with no complications in either of the shunt procedures. In this study, erectile potency after treatment was maintained in one of the three patients available for follow up to six months. Other surgical procedures used in some other places are ligation of internal pudendal nerves and ischiocavernous muscles when the above procedure fails or amputation of the penis if associated with malignant disease of the genitalia.

## CONCLUSION

Priapism although infrequent, when it occurs causes serious consequences. Most African people relate it to witchcraft especially when someone had sexual relation with someone else's wife and this causes delay in reporting to hospital. Although it is difficult to predict the outcome, early treatment gives better results than when treatment is delayed. Among the above shunt procedures, covernoso-Spongiosal shunt is advocated by many urologist. It is simple easy to perform and hence every general practitioner and surgeon is advised to learn it.

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# CHRONIC OSTEOMYELITIS


## SUMMARY:

For a period of four months commencing August to November 1993, twelve patients with chronic osteomyelitis were seen and managed at Muhimbili Medical Centre, department of orthopaedic and trauma where the author was attached. There were 10 males and 2 females. The age range was from 11 to 50 years with a peak in the 11 - 20 years age group. This group accounted for 58.3% of all cases. The femur and humerus were the bones most frequently affected by the disease. The disease was of spontaneous onset in 6 patients. In 5 patients it followed open fractures while in one it occurred in a closed fracture.

Of the positive pus swab cultures; 7 had staphylococcus aureus, 2 had escherichia coli and one had proteus vulgaris. All staphylococcus aureus isolated were sensitive to cloxacillin and erythromycin. The main mode of treatment was surgery which consisted of debridement, saucerization and sequestrectomy. One patient is presented in detail, the data on the twelve patients is analysed and the literature reviewed with emphasis on etiology, pathogenesis and management.


## INTRODUCTION

Chronic Osteomyelitis is a disease especially common in developing countries; extra ordinarily difficult to cure and often leading to permanent disabilities. Wheres in Western World the prevalence is decreasing due to early diagnosis and treatment; in the developing countries it is increasing due to late

presentation for treatment[1]. Chronic osteomyelitis many times arises as a complication of acute osteomyelitis which is inadequately treated[2]. This often occurs as a result of inadequate dosage of drugs, inappropriate drug therapy and late presentation to hospital. Late presentation to hospital results into radical operations and long term use of expensive systemic antibiotics.

Since the discovery of antibiotic and their wide spread use, the incidence and course of the disease has changed[1,2]. The emergency of resistant organisms have further complicated the management of chronic osteomyelitis. Surgery is mandatory for proper treatment of chronic osteomyelitis[3]. Measures to prevent road traffic accidents, prompt and adequate surgical toilets to open fractures and adequate treatment of acute osteomyelitis may reduce the incidence of chronic osteomyelitis[3].

**CASE REPORT**

A. B. in patient number 625943 was a seventeen years old male referred to Muhimbili Medical Centre with complaints of pain and swelling of the left upper arm for three months. He had history of throbbing pain for three months on the left upper arm. The pain was non radiating. It was associated with a slowly progressive swelling at the site with a pus discharging sinus. He had experienced similar attack 12 months previously. He underwent an operation following the first attack, after which he improved but one year later the disease reappeared. There was no history of injury to the upper arm prior to the onset of the illness. He had intermittent fever of high grade with generalized malaise. No history of cough or chest pain. He was a primary school leaver, unemployed and staying with his relative. He had been

treated at various dispensaries and hospitals without remarkable improvement.

He was a young man in good general condition not pale or icteric and was afebrile. There was no palpable peripheral lymphadenopathy. He had a pulse of 80 beats per minute regular and of good volume with a blood pressure of 110/70 mmHg. The patient's left upper arm was swollen mildly and there was a lateral longitudinal scar. There was a discharging sinus at the lower end of the incisional scar with a small bone fragment sticking out of the sinus. There was no appreciable warmth or tenderness and the shoulder could be put through the normal range of motion. The other limbs were normal. The respiratory, cardiovascular, gastrointestinal and central nervous systems were found to be normal. A clinical diagnosis of chronic osteomyelitis of the left humerus was made. It was confirmed by x-ray of the left humerus on anteroposterior and lateral views which showed a small sequestra with a big involucrum in the proximal third of the humerus. The left shoulder and elbow joints were normal. The chest x-ray was normal. Other investigations done were the haemoglobin which was 11.2 g/dl and ESR 72 mm/in the first hour. Other features of the blood picture were normal. A pus swab revealed staphylococcus aureus on culture, sensitive to cloxacillin and erythromycin.

The patient was booked for sequestrectomy which was done one week later. He was started on cloxacillin 500 mg six hourly four days prior to surgery. The operation was done under general anaesthesia and aseptic condition. An incision was made around the sinus and the loose bone removed. The sinus tract was excised down to the bone. The surrounding scar was also excised leaving healthy normal tissues. Using an osteotome the bone marrow cavity was opened, from which

another sequestrum was removed and a large amount of pus evacuated. All necrotic materials were curetted out, leaving healthy bleeding bone. The wound was thoroughly irrigated with normal saline and hydrogen peroxide. The exposed bone and wound were then covered with vaseline gauze. Compressive dressing were applied and the limb was supported by a triangular armsling.

Postoperatively the patient continued with cloxacillin for a period of two weeks. Dressing were changed on the fourth post operative day. The wound was cleaned with hydrogen peroxide and normal saline on alternative days and dressed with vaseline gauze.

He was discharged on the seventh post operative day and was advised to continue with eusol and vaseline dressing on alternative days at the nearest dispensary or hospital. Three weeks later, he reported in the out-patient clinic with a clean wound which had good granulation tissues. At 6 weeks following surgery, the wound was completely closed.

## DISCUSSION

### Incidence

Chronic osteomyelitis is a common cause for admission of patients to the orthopaedic wards at our hospital. Out of 590 consecutive admission, 30 were cases of osteomyelitis. This formed nearly 5% of all the admissions during the four months period as compared to 10% reported by Kessi in 1978[4]. Osteomyelitis occurs in all age groups. Nine patients out of twelve were under 30 years as seen in the table 1 below:

TABLE 1: The age and sex distribution of the patient with chronic Osteomyelitis

| AGE GROUP (YEARS) | NUMBER OF PATIENTS | | TOTAL | % |
|---|---|---|---|---|
| | MALE | FEMALE | | |
| 11 - 20 | 5 | 2 | 7 | 58.3 |
| 21 - 30 | 2 | 0 | 2 | 16.7 |
| 31 - 40 | 2 | 0 | 2 | 16.7 |
| 41 - 50 | 1 | 0 | 1 | 8.3 |
| Total | 10 | 2 | 12 | 100 |

The age range was from 11 - 50 years with 58% of patients less than 20 years. Waldogel[2] found more than 85% of patient less than 20 years old. Our figure is different from Waldogel as children below 10 years of age were not included in our study since they were admitted in the Paediatric wards. Males are affected 3 times more than females[1,2,3]. In this study the male to female ratio was 5 : 1.

Pathogenesis

In our study 50% of the patients the disease started spontaneously while the rest were associated with fractures; 5 cases open fractures and one closed fracture (Table 11 ). Failure of treatment of acute osteomyelitis by immediate, adequate and uninterrupted chemotherapy for one month or longer is a common sequela of chronic osteomyelitis[4]. This was clearly illustrated in our cases.

Table II: The causes of Osteomyelitis

| CAUSES | NUMBER OF PATIENTS |
|---|---|
| Spontaneous (Haematogenous) | 6 |
| Open fracture | 5 |
| Closed fracture | 1 |
| Total | 12 |

Chronic osteomyelitis is virtually a chronic infection of bone by micro-organisms commonly bacteria or fungi. The infection and clinical presentation of the disease depend on site of entrance, localization, virulence of the invading organisms, colonization in term of finite number of bacteria per unit volume and the resistance of the host which includes bone properties at the site, e. g. blood supply, humoral and cellular immunity and reparative power of the damaged area[1,5]. The routes for bacteria invasion to the bone include direct infection through an open fracture or open wound after surgery or extension from nearby soft tissue or joints or through bacteraemia[6,7]. Location of the infection on osteomyelitis varies with the age of the patients[6].

In new born, the umbilical cord is the potential source of entrance. The micro-angiopathic radiological studies have shown that vessels on metaphysis do cross the growth plate to epiphysis up to the joint. Nade in his study detected bacteria colonies in the epiphysis hence concluded that spread of infection to adjacent joints occurs[8].

In children, the medulla of diaphysis and metaphysis is supplied with blood from nutrient arteries whereas epiphysis receive its blood from a separate route. The branches of nutrient arteries in metaphysis of children below sixteen years end as straight narrow capillaries which take a hair pin turn back on themselves at the growth plate and then terminate in veins with a much wider caliber than the capillaries. These veins in turn drain into the venous sinusoids of the marrow. The large calibre of the metaphyseal vein enhance stasis and if bacteria emboli are present thrombosis of the vein will occur[1,8]. The occlusion of the capillaries followed

by transudation and marrow necrosis provide good environment for growth and colonization by the bacteria. The area has low phagocytic function which favour infection. The infection then travels through the harvesian canals of the bone cortex to form subperiosteal exudate.

In infants, their immunity is low especially if unprotected by maternal antibodies. When infection of bone set in, is usually rapid, first localize in metaphysis but frequently has early involvement of the epiphysis due to free anastomotic channels of the vascular system between the two growth plates. This is known as Pan Osteomyelitis. Pus penetrate through the periosteum to form an extra periosteal abscess[5]. Patients with high tissue resistance or low grade infection or both are associated with a chronic sclerosing osteomyelitis. This is a sclerosing form of osteomyelitis described by Garre in 1893[9]. It is associated with distension and thickening of bone but no suppuration, sequestration or fistula formation.

Post traumatic bone injuries may result into chronic osteomyelitis[7]. Tissue damage depend on applied forces which cause blunt or sharp injuries. In open fractures especially of second and third degree or in bone surgery there is increased chance of innoculation of bacteria at the moment of injury, during operation in the theatre or during wound dressing[7]. The amount of tissue contamination at the time of injury, the length of time between injury and treatment, the duration of operation and the handling of tissue during surgery determine the infection rate. Blood is known to be a very good media for bacteria growth. Haematoma and oedema which occur after injury increase pressure on tissues which disturb the microcirculation sufficiently to cause a "compartment syndrome" in some cases. Tissues necrosis

follows, which act as the ideal culture media for bacterial growth[1,7]. The blood supply to the periosteum is disturbed by the operation. In most of the time the periosteum cannot be spared unless an atraumatic operation technique is applied. Pressure from tenaculum and rough manipulation cause additional lesions of the surrounding tissues.

The aim of closed or open reduction is stability at the fracture site. If this is not achieved the blood supply is disturbed and the necrotic tissue area become more extensive. Under these circumstances the tissue and bone pieces are not able to be revitalized in the fractured areas. Devitalization of tissues provide a good media for bacterial growth. This is primary instability[10]. Loosening of a primary osteosynthesis results into secondary instability. This is as a result of weak devices or the effect of an inflammation. The use of immobilizing agents externally or internally for fixation do destroy or interfere with blood supply and spread the infection from external i.e. skin to bone marrow or from the fracture site on the bone marrow to proximal and distal areas as in intramedullary nailing[7,10].

The use of drainage agents such as rubber or tubes are important for evacuation of any collection of blood, however their use for long periods is not indicated. They usually block within two to three days and the drains become the source of bacteria transport from outside[7].

The healthy status of the patient is another important factor for the pathogenesis of osteomyelitis. Those individuals with low humoral and cellular, immunity will succumb more to infection than the ones with good immunity[10]. The young and old age groups have lower immunity than the middle energetic people[5,6,8]. Similarly

individuals with AIDS are at risk of infection than the normal individual. Those individuals with associated systemic diseases like diabetes mellitus and angiopathy are more predisposed to infection and complications than those without it[2,3].

In 90% of cases of chronic oteomyelitis; the commonest organism is staphylococcus aureus[5,6]. Waldvogel et al however have pointed declining role of the organisms in recent years to only 50% of cases[3]. Fifty eight percent of patients seen with chronic osteomyelitis in this series were having staphylococcus aureus (Table IV). Fox et al have reported a shift from staphylococcus aureus to haemolytic streptococcus in haematogenous osteomyelitis. Streptococcus infection is frequently found in infants than adults[5]. Salmonella is the commonest organism isolated in patients with sickle cell disease in more than 50% of cases. Infarction, thrombosis and necrosis in sicklers have been found to favour growth as stipulated by Underhill[11]. Other organisms that have been found to cause osteomyelitis include haemophylus influenza, E. coli, staphylococcus albus, proteus, pseudomonas, diphtheroides, brucella, mycobacteria and treponema[7].

Pathology

Chronic osteomyelitis is normally regarded as a result of inadequately treated acute haematogenous osteomyelitis[1]. The disease process does not merely involve infection of bone alone but also the skin, muscle, fascia and periosteum[1,5]. The commonest sites involved are the distal end of femur, proximal part of tibia and humerus while in radius, it occurs on the distal part[5]. Similar findings were observed in this study where the femur and humerus were involved in 41.7% and 33.3% of cases respectively (TableIII). The disease was involving one part of the limb only.

Table III: The site involved by the disease

| SITE | NUMBER OF PATIENTS |
|---|---|
| Femur | 5 |
| Humerus | 4 |
| Tibia | 1 |
| Ulna | 1 |
| Radius | 1 |
| Total | 12 |

Three quarters of the cases had femur and humerus affected. Similar frequency was observed by Mc Allister[12].

Salmonella Osteomyelitis has been implicated to involve multiple limbs in about 60% as compared to 10% in other part of skeleton system[11]. The upper limbs are more affected in sicklers in about 60% as compared to 10% in non sicklers[13]. In children the infective process starts at the metaphysis of rapidly growing long bones especially the proximal part of femur, tibia, humerus and radius. Extension of infection proximally is prevented by the epiphyseal plate and joint capsule[5]. In infants this protective barrier is lacking hence septic arthritis is common especially to the hip and shoulder. There are usually vessels that cross the growth plate. Damage to the epiphysis is irreparable and interfere with growth. The elasticity and osteogenic power in infants leads to profuse involucrum formation but this disappears in time and leaves no trace later in life[5,8].

In adults, the metaphyseal vessels communicate with epiphyseal vessels hence infection can extend and damage the joint[2,4]. Spread of infection from the metaphysis involves bone resorption which takes place by both osteoclastic and non osteoclastic

processes. Some studies done have suggested that staphylococcus aureus might be able to synthesize the prostaglandins that accelerate bone destruction.

In absence of treatment, death of some of the physis and metaphyseal bone occurs after organisms lodge in the medullary vessels. This dead bone, seeded with bacteria can then act as permanent reservoir for bacteria growth and invasion. No antibiotics can penetrate the depth of this sequestrum and hence cannot eradicate infection at this stage. The spontaneous or surgical drainage of pus is followed by repair of favourable cases, through apposition of new bone. The cortex may become thickened by the newly formed bone of the repair process. Where the blood supply has been inadequate, necrotic bone persists as osteoclastic resorption can only take place on living bone. The area devoid of blood supply undergo more ischaemic necrosis and forms a sequestrum[1,2]. Defects on the periosteal new bone may develop allowing infectious material to pass through dissecting fascial planes to the skin surface presenting as chronic discharging sinuses. The sequestra surrounded by the involucrum and fibrotic soft tissue with poor blood supply become the seat of persistent and recurrent infection for many years[2].

Sometimes the pus with necrotic material has no direct communication with the exterior hence a "Brodie cold abscess" is formed[2,4,6]. At times small abscesses are formed and are replaced by cellular or fatty bone marrow while large abscesses are transformed into cystic cavities. It has been found that in 25% of cases the pus is sterile. Whereas up to 50% the commonest organism isolated is staphylococcus[2,3]. The spread of infection to surrounding osseous tissue shut off more vessels and causes death of more bone. Destruction of bone cortex can lead to pathological

fractures. The presence of residual abscess or sequestra sets the stage of recurrent or chronic osteomyelitis. In long standing chronic osteomyelitis, with discharging sinuses, 0.5% of cases are associated with epidermoid carcinoma[3].

Clinical presentation

Chronic osteomyelitis affects all age groups, however, the commonest age groups are children and young adults[5,8]. This was also reflected in our study where the peak age group was 11 - 20 years. Waldvogel found that 85% of his cases were less than 16 years old[3]. Fifty percent of the patients studied presented with local bone pain, swelling and febrile illness. This is comparable to a study by Morse whereby 65% of his cases presented with the same symptoms[5]. Many authors have suggested that acute osteomyelitis should be suspected in young patients with febrile illness and localised bone pain[14]. However, in chronic osteomyelitis patients are accustomed to the disease and hence difficult to obtain relevant information.

Anderson et al reviewing 217 patients suggested that "Any unwell infant or child with an area of local bone tenderness has osteomyelitis until proved otherwise[14]. In older children and young adults he suggested clinical diagnosis to be made when having local bone tenderness and decreased mobility or in the absence of the later; the clinical findings of increased body temperature, local body tenderness, local limbs swelling and at least one of the local erythema, increased skin warmness and decrease in range of adjacent joints movement[5,14].

The presence of a pus discharging sinus from a bone is an indication of chronicity. Scrobie[8] had 80% of his patient presenting with discharging sinus whereas only 70% of our cases had discharging sinus. This might be explained by

the small sample group studied.

Long standing sepsis predispose the patient to anaemia. Low oxygen carrying capacity in anaemia predispose the patient to poor healing. They also have a raised ESR which does not drop until when sequestrectomy is done if a sequestra is present. White blood cell count is not always raised but a shift to the left is a constant feature. Those patients with acute osteomyelitis who do not have a raised leucocyte count are the ones whose disease tend to proceed to chronicity[2]. Clawson supports the investigations as an important and useful guide to the treatment response. Pus swab or blood cultures are useful diagnostic index. A positive blood culture is more likely to be due to acute than chronic osteomyelitis[14].

Table IV: The organisms isolated from pus on culture

| ORGANISMS | NUMBER OF PATIENTS |
|---|---|
| Staphycoccus aureus | 7 |
| E. Coli | 2 |
| Proteus species | 1 |
| No Growth | 2 |
| TOTAL | 12 |

Eighty three percent of the patients studied had positive pus swab cultures. Staphylococcus aureus was cultured in 7 out of 12 patients investigated. They are the commonest organisms isolated in chronic osteomyelitis upto 80% in most series[6,7,8]. E. Coli were isolated in 2 patients and proteus species in one patient. No growth was reported in two patients. The positive pus cultures may be due to organisms obtained on the surface or contaminants and not the one responsible for bone

infection. The specimen for culture may have to be obtained surgically, and routine exploration for diagnostic as well as therapeutic purposes is recommended by some authors. If there is no pus as such, the tissue fluid can be taken for culture. Sometimes cultures may be negative because of prior administration of antibiotics or anaerobic infection whose culture was not studied. The two patients reported having no growth may have had anaerobes.

In one third of patients, radiographic changes take seven to ten days from onset of symptoms, but in the majority it takes 3 - 4 weeks[14]. Radiological changes include local bone destruction manifesting as lytic changes and new bone formation (involucrum). Sclerotic changes on x-ray indicate that the disease process has been going on for more than one month because these changes take time to appear. The x-ray changes are also delayed during healing. All patients in this study had positive radiological features of osteomyelitis after an average of three weeks from onset of illness. Few authors discourage dependence on x-ray findings only [13,14]. According to Waldvogel, 30% of patients show worsening radiological changes while they are improving clinically[3].

According to Pearson sinograms demonstrate more extensive lesions than are seen on plain x-rays[15]. Tomography and bone scanning can also give useful information in chronic osteomyelitis[14]. Sometimes the radiological signs cannot distinguish between chronic osteomyelitis and Ewing's tumour and hence a biopsy is required.

Chronic osteomyelitis and sickle cell disease have similar clinical and radiological findings. Sickling test and haemoglobin electrophoresis have to be done

to differentiate these conditions. The diagnosis in this study group was based mostly on history, physical examination, radiological findings, white blood cells both total and differential, erythrocyte sedimentation rate and swab culture results.

**TREATMENT**

Since chronic osteomyelitis is often a complication of acute osteomyelitis an important aspect of the management of chronic osteomyelitis is through prevention by proper treatment of acute osteomyelitis. Timely treatment with adequate doses of the right antibiotic is very important in acute osteomyelitis and the treatment should be prolonged and uninterrupted for an average of four to six weeks. By clinical judgment one can determine whether surgical intervention is required in addition to chemotheraphy. Drilling of holes has been discouraged in chronic osteomyelitis because often no pus is found in the medullary cavity and may lead to severe haemorrhage. The hole also becomes the route of infection[16]. The value of immobilization is also emphasized to minimize the chances of dissemination of infection, reduce deformity and prevent pathological fractures[5]. Administration of effective antibiotics in the management of chronic osteomyelitis is mandatory. Therapy is guided by culture and sensitivity tests of pus obtained from the base of the discharging sinuses. However, one of the problems is that antibiotics may not reach the organisms in the sclerosed avascular bone. Hence, in the treatment of the disease emphasis has been laid on the excision of sclerotic bone and diseased tissue. Antibiotics are to be started preoperatively to provide an effective blood level during the operation. The choice of antibiotics varies depending upon the changing spectrum

of causative organism and sensitivities. The commonest organism at our centre and in this study is staphylococcus aureus sensitive to erythromycin and cloxacillin. In sicklers, the commonest organism is salmonella sensitive to chloramphenical.

Gilespore and Mayo have found that bacteriocidal antibiotics like cloxacillin, have smaller failure rate than bacteriostatic antibiotics[17]. Nade has advocated the use of combination of antibiotic for single or mixed growth[5]. A combination of drugs like cloxacillin or erythromycin with sodium fusidate have been recommended. They are of low toxicity, show synergism and broaden the range of antibiotics coverage[5,17]. Eighty percent of the patients in this study series were treated from seven to fourteen days with antibiotics as an adjuvant to surgery. Single drug according to culture and sensitivity was used. Vertebral pyogenic osteomyelitis have been implicated to be resistant to antibiotic therapy and hence the use of antibiotics with gamma globulin have been recommended[12]. It is followed by clinical and radiological review of patients at 6 weeks interval. Failure of conservative therapy leads to surgery.

## SURGERY

The cure of chronic osteomyelitis depends on adequate excision of the diseased tissues, obliteration of the dead space by plastic construction, bone fixation and bone grafting in cases of non union and skin coverage. The first aim of therapy is to resect all sequestra and dead materials. Then debridement is done radically and without regard to the remaining defects. Some of the radical procedures include saucerization and sequestrectomy, papineue method and sometimes bone resection.

Table V: The surgical procedure

| PRODUCE | NUMBER OF PATIENTS |
|---|---|
| Sequestrectomy/Saucerization | 7 |
| Sequestrectomy + Bone graft | 3 |
| Saucerization + External Fixator | 1 |
| Incision + Drainage + Antibiotics | 1 |
| TOTAL | 12 |

Most of the patients with chronic osteomyelitis were treated surgically by sequestrectomy and saucerization.

(i) Saucerization and Sequestrectomy

These are procedures done to the infected focus in the bone which contain sequestra or dead bone embedded in infected granulation tissue which is responsible for persistent or recurrent draining sinuses[12]. Antibiotics are started from four to seven days preoperatively to provide an adequate and effective blood level during operation. Sometimes the chronic infection takes the form of diffuse sclerosis with multiple sinus tracks but no large cavities or sequestra. Nade[8] advocate the use of dye such as Methylene blue which is injected to the sinus track one day before surgery to outline the sinus track and dermacate dead tissues. All the procedures are done using tourniquets unless there are contraindications like sickle cell disease.

Under aseptic technique, the sinus tracks are excised along with poorly vascularized soft tissue up to the bone. After opening the cavity, it is saucerized, that is completely deroofed using osteotomes and gouges while the margins are bevelled as extensive as may be required to produce a crater whose opening is wider than its

base. Sequestra which are usually easily identified by their white and crenated appearance are lifted out and the wall of the cavity closely examined for other loculi or communicating sinuses. Release of the tourniquet is of help to locate unexcised dead tissue which do not bleed freely.

The cavities are curettaged and cleaned with hydrogen peroxide[4]. They are then irrigated with copious amount of saline to remove all loose tissues. A local flap of muscle is then mobilized with care to preserve its blood supply and this is used to fill the cavity. If no muscle is available; the wound is dressed and left open[4]. A few days later when clean and granulating, a split skin graft may be used to cover the cavity. At a later date when all signs of infection have subsided, the cavity may be filled by cancellous bone[16].

In this study 10 out of 12 patients were treated surgically by excision of the scarred tissue, saucerization and sequestrectomy for those who had sequestra (66.7%). The exposed bones and wounds were covered with vaseline gauze or sofratule initially because the remaining wounds were not amenable to other means of obliteration. Pressure dressing were then applied before applying either a plaster cast with a window or inserting external fixator to prevent against pathological fracture when the left bone is found to be weak (Table V). The wound was regularly dressed till when granulating tissue occurred where partial thickness skin grafting was done. Mathes has reported that the use of grafted vascularized muscles or muscle and skin arrest progression of necrosis and improve tissue oxygenation as well as leucocyte and antibody delivery necessary for healing[18].

(ii) Papineue method

This is an extensive surgical procedure which involve sequestrectomy and

saucerization with stabilization of the bone, bone grafting and finally closure of the wound by skin grafting or muscle pedicle. The aim during treatment is to stabilize the bone by fixation, remove fragments of infected dead bone (sequestra) and to open abscess cavities by chiseling away the overlying bone. Sometimes it is possible to obliterate a cavity with a flap of muscle or to exteriorise it and cover the walls directly with split skin grafts. Mathes has advocated the use of myocutaneous flap to cover the wounds after saucerization[18]. Grafted vascularized muscles and skin, arrest progression of necrosis in septic wound and improve circulation. Casablanka has obtained good result using the open cancelllous bone grafting technique[19]. The transposition of the upper fibula into a defective upper tibia in growing children was well accepted by many authors.

The use of sliver or strip grafts from iliac crest have promoted union in un-united cavities in bones[3,16]. Sometimes a segment of bone which is severely infected is resected and a fresh bone is grafted. No patient in this study had the papineue method of treatment.

Amputation of a limb is indicated following failure of the above treatment. Amputation in chronic osteomyelitis is indicated in malignant changes in chronic sinus which occur in 0.5% of the cases, where there is arterial insufficiency especially the distal part of the lower limbs, major nerve paralysis, joint contracture with very severe stiffness and on patients request after several futile operations[8].

(iii) Local antibiotics therapy

Local instillation of antibiotics in order to achieve high local concentration of antibiotics agents with minimal systemic toxicity have been practised for long time

now[17]. It was used in prolonged infection. Local antibiotics were given in form of beads embeded with antibiotics or irrigation into the cavities. Cloxacillin and Fusidic acid have been recommended for irrigation whereas Gentamycin chain of beads have commonly been used. The method of local antibiotic therapy with Gentamycin containing Polymethyl Methacrylate (PMMA) beads in combination with radical excision of lesion and primary wound closure have been suggested by Klemm to have greatly simplified the treatment and is suitable for use in developing countries although very expensive[20]. In his study of 27 patients given Gentamycin beads after radical after radical surgery, 19 wounds healed within one months as compared with other reports where it took longer periods[20]. The advantage of this method is that the wound is closed primarily, frequent dressing is not required, the patient gets up after 24 hours and the antibiotics are used for short period ( one to two weeks). During the period of study there were no Gentamycin embeded chain at the Muhimbili Medical Centre (MMC) because they are expensive and not freely available from the local pharmacies.

Complications

Osteomyelitis rarely causes death. However, several complications do occur including recurrence. The rate of recurrence is between 15 - 20% in children, while in proximal or distal third of Tibia and Fermur is between 20 - 30%[8]. The spine and upper limb bones are not usually associated with recurrence. Involvement of an adjacent joint leads to suppurative arthritis particularly the shoulder and hip joints. Destruction of the epiphyses and joint dislocation can also occur due to effusion and septic necrosis. Metaphyseal infection on the germinal cells at the epiphyseal edge

of the growth plate leads to unequal growth producing deforming or shortening.

Irregular laying down of bone during the repair process leads to angulatory deformity whereas pathological fracture has been reported to occur during resorption of bone.

Long standing discharging sinus tract may undergo malignant change. The other implicated complication is amyloidosis. In three patients (25%) in this study; the disease was recurrent while two patients (33%) had pathological fractures.

## CONCLUSION

Chronic Osteomyelitis is a disease in which cure without any complication cannot be guaranteed unless concrete measures are taken during the initial phase of the disease. Its incidence however can be brought down by arousing recognition and institution of adequate therapy in acute osteomyelitis, prevention of traffic and other accidents and avoidance of sepsis in bone surgery.

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## MANAGEMENT OF FRACTURE OF THE HUMERUS SHAFT

**SUMMARY:**

During the period of August to November 1993 twelve patients with humerus shaft fractures were studied by the author. Eight of these patients were males while four were females. The age range was from 11 years to 65 years. Five of these patients were injured following a Motor Traffic Accident (MTA); while four were injured following a fall from a height. Two patients sustained their injuries after being assaulted while the last patient had a sport injury. Six patients had open fractures while the rest sustained closed fractures. Seven patients sustained fractures in the distal third of the humerus shaft while the middle and proximal third of the humerus were involved in four and one patient respectively. X-ray revealed transverse fracture in three patients while four patients had spiral fractures with butterfly fragments and five had oblique fractures. Two patients had other associated injuries.

While conservative treatment using hanging casts extending from the axilla to the wrist joint were used in seven patients, five patients required open reduction with internal fixation of the fractured humerus shaft primarily, using plates and screws. The commonest complication observed after fracture of the shaft of the humerus was non union. Other complications were wound infection and radial nerve injury. While non union was treated by open reduction with internal fixation using plates and screws after cancellous bone graft, wound sepsis was treated by antibiotics guided by culture and sensitivity. One patient who was having neurotmesis of the radial nerve had secondary radial nerve repair by end to end anastomosis.

## INTRODUCTION

The humerus is the most versatile bone in the body due to its articulation at the shoulder joint[1,2]. It is commonly fractured due to twisting forces to the arm or direct blow over the shaft of humerus. The former injury produces a spiral fracture which may include 'butterfly' fragment; the latter produces a transverse fracture[2,3]. These injuries may also result from a fall on the hand[3]. The proximal third of the humerus is a common site for pathological fractures secondary to metastatic carcinoma or various bone disease[4]. The fracture occurs in adults of any age but seldom in children[2,4]. Displacement of the fragment is in variable degrees depending on direction and intensity of the fracture forces, muscle action, gravity and position especially of the forearm. In most cases there may be marked angulation or overlapping of the fragments[1]. The objective of fracture treatment is to restore function of the arm to its pre-injury condition. Most humeral shaft fractures are treated by conservative methods such as a U slab, hanging cast or shoulder spica[1]. The other method is operative by internal fixation. It is particularly useful for fracture of the greater tuberosity, supra or intracondylar fracture of the elbow and avulsion fractures. These methods give better chance of fracture healing by achieving reduction and adequate immobilization[4,5].

Humerus shaft fracture are complicated by neurovascular bundle injuries especially radial nerve, delayed union, malunion and non union[1,6]. In this paper a series of twelve patients were studied. One patient is presented as an index case and the rest are summarized in the text. A review of anatomy, mechanism of injury and treatment is also highlighted.

**CASE REPORT**

J. J. IP. No. 739631, a 30 years old African male was referred from Tanga with a history of failure of union of the fracture in the distal third of the right upper arm. The injury was due to motor traffic accident he had sustained three months before hospital admission. The limb was immobilized in a long complete POP cast since the date of injury without any improvement. He had other multiple soft tissue injuries which healed well after being treated. He denied having suffered from other ailments in the past. The patient was a peasant, married with two children. He did not smoke cigarette nor drink alcohol.

Systemic examination was normal. Local examination of the right upper limb showed an obvious deformity with dorsal angulation of the distal third of the humerus. The overlying skin was normal and there was no increased warmth. There was some movements at the fracture sight with slightly tenderness. The radial pulse was normal. The motor function and sensation of the forearm and hand were normal. A diagnosis of the fracture of the distal third of the Humerus complicated by Non Union was reached. It was confirmed by X-ray taken on Front and Lateral view. Apart from fracture at the distal third of the humerus, the bone ends were rounded, sclerotic with no bridging callus. The patient was planned for open reduction and internal fixation. He had a normal chest x-ray; a haemoglobin of 10.8g/dl and a normal urine analysis.

Three weeks after admission, he was operated under general anaesthesia with endotracheal intubation; a tourniquet was applied on the right upper arm. A lateral longitudinal incision was made over the distal humerus. The radial nerve identified,

taped and isolated. The fibres of the triceps muscles were separated and the fracture site exposed. The bridging fibrous tissue were cleared on both fragments at contact ends. Reduction with immobilization in a six holes plate was done. Before fixation, spongiosa bone grafting obtained from the right iliac crest was done. The wound was closed in layers.

Post operatively the patient had his vital signs charted half hourly for 12 hours and then four hourly for 24 hours. He was prescribed Cloxacillin 500 mg six hourly for five days which is sensitive to staphylococcus aureus. The right upper limb was elevated. A control x-ray of the right humerus in two views taken immediately after operation showed satisfactory reduction. The post operative period was uneventful. The drain was removed after 48 hours. The wound healed without sepsis and stitches were removed on the eighth postoperative day. He was discharged from hospital on the tenth postoperative day. He was seen two weeks later at outpatient clinic free of pain with good health. He was referred back home to Tanga with instruction to avoid lifting heavy objects for three months. He was to come for follow up after six months.

## DISCUSSION

### Surgical Anatomy

Humerus shaft fractures are those involving the part between the surgical neck and the epicondyles[1]. The humerus is the most versatile long bone in the body. The shaft is cylindrical proximal and broaden distally. The major neurovascular structures are located medially except the radial nerve which courses laterally. The

humerus bone receives its blood supply from a nutrient artery which enters the bone in a downward inclination usually on the medial surface at the junction of the middle and lower third of the humerus. The artery has other accessory nutrient arteries ranging from one to four[1]. The nutrient artery originates from the subclavian artery[7]. When it is single; it divides in the medullary cavity into a large ascending and small descending branches. Sometimes the division occur before reaching the bone. In most of the time the artery is constant in position hence easier to avoid during surgical procedure[1,7].

Fracture of the shaft of the humerus may be complicated by injury to the radial nerve in the radial groove of the humerus[1,8]. The nerve injury is usually no more than a contusion seldom a complete division. It occurs in 5 - 10 percent of all fractures of the distal third of the humerus[8]. Szyckowitz et al have reported radial nerve lesion accounting to between 7 - 9% in patients with fracture of the humerus[9]. In this study 2 patients (16%) had radial nerve injury. The course of the radial nerve make it susceptible to injury. In the upper arm, it passes obliquely across the back of the humerus first, between the lateral and medial head of the triceps accompanied by the profunda bronchii artery to reach the lateral side of the humerus where it pierces the lateral intermuscular septum and enter the anterior compartment of the upper arm[6].

Some authors have postulated that the radial nerve is injured frequently because the intermuscular septum holds the radial nerve tightly and constant in position[1,8]. The radial nerve then descends between the brachialis on one side and the brachioradialis muscle above the extensor carpi radialis longus below on the other

side up to the level of lateral epicondyles. At this region; it divides into two terminal branches, the superficial and the deep branches[1]. Whitson has reported a small group of people (less than 10%) where the radial nerve does not make any contact with the bone on the radial groove except on the lateral supracondyle ridge. These are the groups which can sustain severe comminuted humeral fracture at the radial groove without injury to the nerve[8,10].

The median nerve which passes lateral to the branchial artery initially and later descend medially to the cubital fossa is very mobile and rarely injured. On rare occasion it can be injured in severe supracondylar fracture of the humerus or dislocation of the elbow joint. It is commonly injured at the wrist joint where it is very superficial[5,9]

The ulnar nerve runs on the medial side of the axillary artery up to the middle of the humerus when it pierces the intermuscular septum to reach the posterior compartment of the upper arm. It passes infront of the triceps muscle and finally reaches the elbow joint where it lies behind the medial epicondyle accompanied by the superior ulnar collateral artery. The ulnar nerve may be injured in fracture of the medial epicondyle of humerus or supracondylar fracture or by callus formation at the fracture site[8,10].

Incidence

Seventy five percent of the patients were at 11 - 30 years age group in this study[1] (Table I below). Similar findings have been reported by Adams et al who considered the young age group as being the most active and therefore involved in sports and other energetic activities[6]. Some authors consider the old age group as the most

affected because of osteoporosis and metastatic carcinoma[4,6]. However majority have the opinion that fractures of the humerus shaft have no age predilection[2,3,6]. The incidence is higher in males than females. Our study showed a male to female ratio of 2 : 1. Such findings have been reported elsewhere[1,2,3]. The reason for the higher male preponderance is probably due to the more vigorous physical work undertaken by males[1,3,6].

Table I: Distribution of the patients according to age and sex

| AGE GROUP (YEARS) | NUMBER OF PATIENTS | | TOTAL |
|---|---|---|---|
| | MALE | FEMALE | |
| 11 - 20 | 4 | 1 | 5 |
| 21 - 30 | 3 | 0 | 3 |
| 31 - 40 | 0 | 1 | 1 |
| 41 - 50 | 1 | 1 | 2 |
| 51 - 60 | 0 | 0 | 0 |
| 61 - 70 | 0 | 1 | 1 |
| TOTAL | 8 | 4 | 12 |

Pathology and clinical features

Table II: The aetiology

| CAUSATIVE FACTOR | NUMBER OF PATIENTS | % |
|---|---|---|
| Motor traffic accidents | 5 | 41.7 |
| Fall from height | 4 | 33.4 |
| Sports injury | 1 | 8.3 |
| Assault - Human | 1 | 8.3 |
| - Animal | 1 | 8.3 |
| Total | 12 | 100.0 |

Most fractures of the humerus shaft are caused by direct violence[3,15]. The commonest cause in this study were motor traffic accidents and falls from heights contributing 75% of the patients. Other causes were sports injuries and assaults from human and animal (one patient each). The type of fracture of the humerus and displacement depend on intensity of the causative force, muscle action, gravity and position of the forearm[6]. Half of the patients had closed fractures while the rest had open fractures.

The fracture line can be transverse, spiral, oblique or comminuted depending on the causative force[3]. Twisting forces give rise to spiral fractures. Adam has postulated that any bending movement causes transverse fracture whereas torsional forces cause spiral fracture[6]. It has been found that pathological fractures occur commonly in the proximal third of the humerus shaft and fractures of the distal third of the humerus are common in children[4,5]. Fifty eight percent of the fractures were in the distal third of the humerus while 33.3% were in the middle third. Only one patient (8.4%) had fracture in the proximal third of the humerus following a fall from a height.

Table III: Site of injury at the Humerus shaft

| SITE | NUMBER OF PATIENTS | % |
|---|---|---|
| Distal third | 7 | 58.3 |
| Middle third | 4 | 33.3 |
| Proximal third | 1 | 8.4 |
| TOTAL | 12 | 100.0 |

The commonest site of injury was the distal third of the humerus shaft.

Fracture of the humerus shaft is sometimes accompanied by injury to the neurovascular bundles. The radial nerve is commonly injured at the junction of the middle and lower third of the humerus in 5 - 10% of cases[8]. When injured, it results into paralysis of the extensor group of muscle of the wrist, fingers and thumb (wrist drop[1]). Paralysis of brachioradialis and supinator muscles also occur. The only sensory change is in the small area of the anaesthesia on the radial side of back of the hand[5,8]. Median and ulnar nerves are rarely injured in fracture of the shaft of the humerus[3]. Vascular injuries occur in 3 - 5% of cases[1,6].

After fracture, the muscles of the arm and gravity act on mid shaft fracture to produce shortening and varus angulation[1]. The degree of displacement depends on the transmitted force, muscle action, gravity and position of the forearm[6]. Fractures occurring above the insertion of the pectoralis major muscles usually result into abduction and internal rotation of the proximal fragment. These are the results of action of the rotator cuff and subscapularis muscles respectively. Fractures occurring between the insertion of pectoralis major and deltoid muscles result into adduction of the proximal fragment and abduction of the distal fragment. It is an action of pectoralis major and deltoid muscles respectively[1]. The deltoid, biceps, triceps, and coracobrachialis muscles are responsible for abduction of the proximal fragment with or without shortening in fractures occurring below the deltoid insertion[11].

The diagnosis of fracture of the humerus shaft is simple. The physical signs of swelling, deformity, instability, abnormal movements and tenderness are obvious and confirmation is done by radiographs. In children marked loss of function, unwillingness to use the affected limb and local pain and tenderness are amongst the

cardinal signs used to suggest fractures[1,3].

**TREATMENT**

Immediately after injury, first aid measures should be started. This prevents further injuries to the soft tissues and vessels. The patient is in pain and unable to use the limb. So he requires gentle handling and effective immobilization of the fractured limb to minimize pain and anxiety. As injuries occur in different surroundings where no medical facilities are available, a satisfactory splint using pieces of wood which are placed, one on medial side and the other on lateral side are used. At times strapping of the affected limb on patients chest has proved effective.

On arrival to a hospital a quick assessment of the general condition is made as some may be in shock. Measurement of the blood pressure, pulse, inspection of mucous membrane for pallor and assessment of the patient is made for a possibility of associated injuries to the head, neck, chest, abdomen and in the other limbs. A wound in the fracture site may indicate an open fracture. Replacement of fluid is done using crystalloid fluids and restoration of blood volume is done by blood transfusion when indicated. Open fractures are treated as emergencies with immediate surgical toilet[1]. Similarly injuries to major blood vessels result into ischaemia which may become irreversible if not handled promptly.

Exploration to determine the nature of injury is done if all effort to relieve occlusion like removal of external splint or bandage or reduction of the grossly displaced fracture fail.

Nerves can be damaged at the times of injury resulting into axonotmesis or

neuropraxia where it can be cured by reduction of the fracture whereas if the nerve is severed (neurotmesis), it can be treated primarily or secondary by surgical reconstruction. Most of the humerus shaft fractures are treated conservatively; however if fractures fail to heal, they are treated by open reduction and internal fixation.

The aim of fracture treatment is to restore function of the affected limb to its pre-injury condition by achieving good reduction. This involve anatomical re-alignment of the fracture site and immobilization to achieve proper healing. Conservative method entails prolonged immobilization and may lead to disadvantages of joint stiffness, muscle atrophy, osteoporosis, vascular insufficiency, malunion, delayed union and non union[6].

Fracture of the shaft of the humerus is said to be the most easily reducible of any long bone fracture[9]. The hanging position of the humerus and gravity of the arm aid in maintaining position and alignment. The gravity acting on the arm exerts steady traction which tend to correct any angulation and overriding that may occur[12]. Because the shoulder is a multiaxial joint any angulation, which may persist at the fracture site does not interfere significantly with function. Klenerman has postulated that an angulation of up to 20 degrees at the united fracture site doesn't produce any obvious clinical deformity[3].

Conservative therapy

Numerous methods are used as conservative treatment of humerus shaft fracture which include; collar and cuff sling, U-slab, hanging cast and the use of

shoulder spica cast[1,3].

Six out of twelve patients in this study were treated by the hanging cast method. This involved the use of light weight cast extending from a distance of 2.5 cm above the fracture site to midpalm. Several authors have concluded that the weight of cast should vary according to the general muscle build of the patient however shouldn't exceed one kilogram. If exceeded, it results into distraction[9,12].The hanging cast method has been reported to produce excellent and good results in 93.5% of patients treated by it[11]. The hanging cast is used in conjunction with an armsling[2,12]. Angulation at the fracture site is corrected by adjusting the length of the armsling[12]. Backward angulation is corrected by lengthening the sling and forward angulation by shortening it. To correct lateral angulation the suspension loop on the wrist is placed on the dorsal surface and to check medial angulation, the hook is moved on the volar surface of the wrist. If the cast used is too heavy distraction will ensue and can lead to delayed or non union.

During the three weeks of treatment the patient is kept in an upright or semisitting position because the weight of the arm and the cast provides traction and aligns the fracture. Pendulum exercises are initiated with plaster cast still attached to maintain shoulder motion. During treatment it is advisable to make a control x-ray at least once a week[3,6]. It has been found that most humerus shaft fracture unite in approximately 8 weeks. The disadvantages of this method is that the elbow joint cannot be mobilized and occasionally it is not possible to obtain a satisfactory alignment.

Another method is the use of U shaped slab of plaster of negligible weight

applied to the upper arm and held in place with an elastic cotton bandage. The bandage can be tightened every few days as the swelling subsides. It is used with an armsling and the patient makes regular exercises of the wrist and fingers[12]. After four to six weeks; if stable union is demonstrated clinically and radiologically, the slab is removed and only the armsling is retained for two to three weeks, during which exercises of the shoulder, elbow, wrist and fingers are encouraged[3,12]. Twenty five percent of patients in this study were treated with this method with good result except one who needed open reduction and internal fixation due to non union.

In children and other patients who cannot cooperate these methods are not applicable. A thoraco brachial cast which is applied to the arm and the chest to obtain immobilization may be employed in such patients. After three to four weeks the cast is removed and replaced by a less rigid form of immobilization, where upon circumduction exercises are begun[4,6]. A shoulder spica is used in the treatment of grossly unstable open fractures and complex fracture with radial nerve paralysis[8].

Sarmiento has reported good result with a plastic sleeve brace which is applied on the arm over the fracture and allows ample exercises during treatment[13]. It allows controlled movements at the fracture site and encourage formation of a large callus and so enhances healing. Spak has described a method of treatment in which only an armsling is used to support the hand[2]. This treatment was successful in all 59 patients in whom it was used. It avoids immobilization of the shoulder, elbow and wrist with their consequences. The patient can perform circumduction exercises from start and does not need hospitalization. One patient with fracture of the proximal third of the humerus was treated with this method; however had nc

improvement which necessitated open reduction with internal fixation.

Thus, there are numerous methods of non operative treatment to choose from. Treatment should be individualized because there is no one method which is good for all patients. However for the uncomplicated case the hanging cast method appear to be the best, provided it is used with necessary caution[12]. Simple treatment with an armsling alone is also very attractive particularly in a very cooperative patient[2]. Unless complicated, up to 70% of humerus shaft fracture can be treated by conservative method and only 30 % need operative treatment[2,3].

Operative treatment

Open reduction and internal fixation is reserved for open fractures, intraarticular fractures or some oblique fractures of the distal third of the humerus shaft which fail to reduce, unstable badly displaced fractures and those occurring in patients with multiple injuries[10]. Others are those fractures associated with neurovascular injuries and pathological fractures due to malignant diseases. Failure of conservative treatment is another indicator for operative treatment[9,11].

There are various methods for osteosynthesis, plating, intramedullary pinning, intramedullary nailing (interlocking or not) and the use of external fixators[14]., Szyszkowitz[9] et al have advocated the use of external fixators to severely contaminated wounds in presence of vascular bone fragments and where there is high chance of infection. Two patients with severely contaminated fractured wounds (third degree fracture with extensive muscle injury) were treated by the "A-O" external fixator.

The transverse fractures are usually associated with soft tissue interposition as was found in 3 patients in this series. They had internal fixation after failure of conservative method. Similarly Schippinger[9] et al had to treat 33% of their patients with transverse fracture by open reduction and internal fixation after failure of conservative method. The treatment given include the use of broad plate and screwing or intramedullary fixation either with a nail or 3 - 4 flexible pins[9]. The use of Hakethal retrograde stacked pinning for undisplaced or reducing pathological fractures have shown good result in 75% of cases[1,6]. The point of entry is at the distal part above the olecranon fossa. In larger humerus defects and more unstable fractures, plating in combination with a crylic cement is indicated[9].

In intraarticular fracture extending into the humerus head or elbow, osteosynthesis with plates (T plate proximal and a reconstruction plate distally should be applied. The procedure can also be done in the so called "floating elbows" where the shaft fractures of the humerus and forearm are combined[6]. Most of the patients who had midshaft fracture who needed open reduction (5 patients) were treated using plates and screws. There are several views on the treatment of radial nerve injury which occur in 7 - 9% of cases[9]. Schippinger and Seggl advocate early exploration of the radial nerve because with increasing callus formation, revision is more difficulty and nerve recovery is unlikely. If however, there is partial radial nerve lesion without other problems which need surgery; they justify observation for about 3 weeks because at that time the neuropraxia will have recovered[8]. Others prefer an even longer period up to 7 - 8 weeks where if there is a neuroma, it will delineate and complete excision can be done successfully[5,8]. Delay in exploration prevents

interference in fracture healing and avoid unnecessary operation to a radial nerve lesion which could recover spontaneously after 3 weeks. Two patients who had multiple injuries were treated by open reduction and internal fixation primarily as advocated by Connoly et al to facilitate early rehabilitation of the patient as a whole[1].

There are two approaches for open reduction and internal fixation of the fractured humerus; the anterolateral and posterior approach[9]. The anterolateral incision using the Henry approach, between the triceps and biceps muscles and then splitting longitudinally the brachialis muscle and proceed to the humerus is preferred for the upper two third fractures. This approach is extendible at both ends and is suitable in reducing shaft fractures extending into the humerus head. Since the radial nerve lies posteriorly in the spiral groove close to the bone; it is at risk of damage in the middle half of the diaphysis when using the Henry approach. Four patients with fracture of the midshaft of humerus were operated using this method.

In the posterior approach; the triceps aponeuroses is split down in the middle[14]. Care must be taken to find the radial nerve in the proximal part of the incision and follow it up carefully avoiding damage to the muscle branches. In doing so, the deep head of triceps is split longitudinally in line with its fibres. This approach is useful in fractures of the distal third of the humerus, intraarticular fractures to elbow and when a radial nerve lesion exists. Care should be taken to prevent damage to the radial nerve when fixing a broad plate dorsally. Screw holes which crosses the nerves should be recorded. Lag screw are used to secure interfragmental compression preferable through the staggered holes of a broad low contact dynamic compression plates. Semitubular plates are useful to distal diaphysial

fracture.

Fractures of the humerus usually unite solidly in 2 - 3 months and plates need to be removed if they give rise to symptoms[3,13]. In most of the patients they remain in situ. If in exceptional circumstances the plate is removed, the patient should abstain from heavy lifting for a period of 6 - 8 weeks.

Complications

Table IV: The complications encountered:

| COMPLICATIONS | NUMBER OF PATIENTS |
|---|---|
| Non union | 3 |
| Infection | 1 |
| Malunion | 1 |
| Neuropraxia of the Radial Nerve | 1 |
| Severed Radial Nerve (Neurotmesis) | 1 |
| TOTAL | 7 |

The commonest complication encountered was Non union.

1. Radial nerve Injury

The radial nerve may be injured in open fracture or fractures at the middle and distal third of the humerus where it is at close proximity. It may be injured in the course of treatment during manipulation or at the time of healing due to entrapment in the callus or fibrous tissue. In most of the time injury occur as a result of stretching or bruising and recovery occur spontaneous within several weeks up to six months[6,7]. As discussed before, the treatment is controversial. Show[8] has

advocated to wait for at least 3 months to determine whether regeneration will occur. He preferred the use of electromyogram to demonstrate early regeneration. Out of 25 patients who had complete radial nerve paralysis; 15 regained full function spontaneously with an average of 4 months. In two patients with radial nerve paralysis one was treated by conservative method while the other was explored. When the nerve is found to be severed and no regeneration; the nerve is explored, and repaired by end to end anastomosis. If the healing doesn't occur, nerve grafting is done; however the results are unpredictable.

Some surgeons prefer tendon transfer after failure of repair of the nerve[6,11]. The pronator teres is transferred to extensor carpi radialis brevis to extend the wrist. Flexor capi ulnaris or flexor carpi radialis is transferred to the extensor digitorum and extensor pollicis longus to extend the fingers and thumb. The palmaris longus is transferred to the abductor pollicis longus to provide abduction of the thumb away from the palm. In our series, one patient whose radial nerve was severed improved after end to end anastomosis eight weeks after injury. No patient had tendon transfer.

2.Non Union

Fracture of the midshaft of the humerus is recognized as the most resistant to healing especially to transverse fractures[9]. Non union of a fracture become established radiologically if the fragments become dense, rounded and sclerotic. Operative treatment is then indicated. Some of the commonest cause of non union were interposition of soft tissues, infection, loss of apposition between fragments,

and inadequate blood supply to the distal fragment in one patient with severe injury[14].

The treatment offered to these complications were open reduction with internal fixation and cancellous bone graft. The fracture site is laid open, the fracture surface refreshened and the two ends decorticated for 2 - 3 cm on each side. The fracture is fixed internally by intramedullary rod or occasionally by a compression plate[1,14]. Cancellous iliac bone fragments are impacted between the bone ends and cancellous strips are laid over the decorticated surface of both ends. The three patients in this study with non union were treated with compression plates. Two had an additional cancellous iliac bone graft and the fractures healed well.

3. Infection

Infection is virtually confined to open fractures especially of third degree in which the wound is contaminated by organisms carried in from outside the body[11]. Closed fracture may become infected when converted into an open fracture by operative intervention. Wound infection occasionally, remains superficial and the bone escapes, but more often the infection extends to the bone giving rise to osteomyelitis. It is frequently associated with delayed union and non union and presents as significant problems of management. The treatment programme is often lengthy and consist of excision of dead and infected bone, stabilization of the fracture (usually by an external fixator), administration of appropriate antibiotics parenteral in high dosage, cancellous bone grafting and early provision of full thickness skin cover if a skin defect is present.

## CONCLUSION

Fractures of the humerus shaft occur commonly following events like motor traffic accidents, sports and fall from height. In some cases they are associated with other multiple injuries and so the treatment of these patients is tailored according to individual need. The aim of treatment however should be to restore the limb to a normal functional result as possible. This can be done by using conservative or operative means. Awareness of complications and the predisposing factors inherent in such fractures will act as a guide in achieving better results.

Conservative treatment using hanging cast method has proved to be the most practical treatment of fractures of the humerus shaft unless the fracture is open or complicated. This is where open reduction with internal fixation is required.

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# THE MANAGEMENT OF SPINAL TUBERCULOSIS

**SUMMARY**:

Twelve patients with tuberculosis of the spine were treated in the Orthopaedic/Trauma department of Muhimbili Medical Centre in a period of five months from August 1993. Nine of them were male while three were females with an age range of 12 to 60 years. Presenting features recorded in order of frequency were back pain at the site of affection in 10 patients, neurological deficit in 6, constitutional symptoms such as fever and weight loss in 4 and cold abscesses in 2 patients. The lesions were localized in the thoracic and lumbar spine in 6 and 4 patients respectively, and in the cervical and thoracolumbar region one patient each. Radiological investigations were the main stay of diagnosis. A raised erythrocyte sedimentation rate and positive mantoux test though non specific were significant diagnostic findings. Antituberculous chemotherapy was administered inform of streptomycin, thiacetazone, isonicotinic acid hydrazide in seven patients while two groups of two patient each, were given streptomycin, rimactazid and pyrazinamide, another given streptomycin, isoniazid, pyrazinamide and ethambutol.

Two patients had adjuvant surgical decompression procedure. Five patients who had flaccid paralysis did not show obvious signs of paraplegic recovery while the other five had partial recoveries in muscle power, tone and tendon reflexes of the lower limbs. One patient who had early onset of paraplegia with paraspinal abscess recovered completely after decompression of spinal cord under antituberculosis chemotherapy cover.