TEN SURGICAL CONDITIONS

BY

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A DISSERTATION OF TEN SURGICAL CONDITIONS SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF MEDICINE (SURGERY) 1997

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DECLARATION

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

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1997

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

Kisanga.

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ABSTRACT

This dissertation conforms to the Master of Medicine degree in surgery of the University of Dar es Salaam. It is a discussion of ten different surgical conditions seen and managed at Muhimbili Medical Center, during the period of training from 1993 to 1997. The text is divided into four parts, consisting of three topics on general surgery, three topics on thoracic surgery, two topics in Urology and two topics on Orthopaedics and Trauma. In each topic a case is presented in detail, the rest of the cases are summarized, relevant literature is reviewed and the topic is discussed with particular references to observations made at Muhimbili Medical Center Dar es Salaam.

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VOLVULUS OF THE SIGMOID COLON

SUMMARY

A total of thirty patients with sigmoid volvulus were managed in three surgical firms at the Muhimbili Medical Center between October, 1993 and July,1995, while the author was rotating in these surgical firms. There were twenty seven males and three females.

Their ages ranged between 11 and 80 years. The mean age was 34 years. All patients presented with a history of abdominal pain followed by constipation and abdominal fullness. The other features were vomiting(10 cases), dehydration(10 cases), peritonitis (4 cases), and a previous history of a similar attack in (5 cases).

Plain abdominal X-ray examination was done in all patients and radiological features of sigmoid volvulus were present in twenty six patients. Operative procedures performed were as follows; resection and double barrel colostomy (16), resection with primary anastomosis (12), and Hartmann's procedure (2). The operation findings were viable sigmoid volvulus (26), gangrenous sigmoid volvulus (2), and ileosigmoid knotting with gangrenous bowel (2). Postoperative complications were as follows: one post-operative death, wound sepsis (5), colostomy retractions (2), and faecal fistula(1). The average hospitalization was between 2 to 3 weeks, with a

range of 1 to 4 weeks. One of the patient is presented in detail followed literature review.

INTRODUCTION

A desire to understand the natural history of sigmoid volvulus has existed since ancient times[1,10]. The authors of the Papyrus Ebors[1,10] from ancient Egypt wrote that either the volvulus spontaneously reduced or the sigmoid "colon rotted". The first accurate description of volvulus was given by a Viennese pathologist, Rokitansky' in 1837 who defined volvulus as torsion of a section of the bowel on its mesentery causing symptoms by narrowing of the lumen, strangulation or both.[1] Any part of the bowel can undergo torsion but the sigmoid colon is most commonly affected.[3]

Sigmoid volvulus has a marked geographical variation in incidence rates. The disease is common in Eastern Europe, Russia, Scandinavia, Africa and Asia where it constitutes 30-50% of all cases of acute intestinal obstruction.[16] It is rare in Western Europe and North America forming only 1-8% of intestinal obstruction.[17] Observation has shown that there are variations even within one geographical region. One example is that while the disease is common in Kenya, Uganda and Rhodesia where it is reported to be the commonest cause of intestinal obstruction, in Tanzania at Muhimbili only 4% of all cases of intestinal obstruction were due to sigmoid volvulus.[20] while

at Bugando[5] only 20 cases of sigmoid volvulus were recorded in a period of five years.

Although sigmoid volvulus is described in children and infants[8,21,24,] it is commonly a disease of adults mostly affecting males.[21] In the West the peak incidence is in the 6th decade, in Africa it affects a younger age group below 50 years.[21] This study was done in order to look into age and sex distribution, clinical features, diagnosis and the treatment of patients with sigmoid volvulus and compare with studies done elsewhere. In this study the mean age was 34 years. The male to female ratio was 9:1. The diagnosis is usually based on the history, clinical findings, and radiological features. The best operative therapy for sigmoid volvulus is resection of the redundant colon and restoration of bowel continuity, either by primary or delayed anastomosis. Arguments have persisted about the need for staged procedures.[17] A review of the literature on the subject is presented with special emphasis in our environment.

CASE REPORT

M.M. IP No 788, a 63 years old male, referred to Muhimbili Medical Center from one of the peripheral hospitals of Dar es Salaam, was admitted to Surgical Firm III, complaining of crampy abdominal pain, progressive abdominal distension and constipation over the previous three days.

The symptoms had started gradually following a heavy meal during the month of Ramadhan fast. Pain started over the hypogastrium, gradually increasing in intensity and after one day it become generalized. His abdomen gradually distended and by the time of admission, he was having difficulty in breathing. His appetite was good, he had no nausea, but had vomited twice. He had not passed stools for three days and flatus for one day. He did not have any associated fever. There was no history of taking local medications prior to or after the onset of the present condition. This was his first admission. He denied history of similar attacks in the past. The patient denied smoking or use of alcohol. Cassava, rice and maize were the main staple foods. He worked as a petty businessman, and the other family members were all fine.

General examination revealed an elderly, health man who appeared ill looking and in pain. He was not dehydrated, not pale, but was dyspnoeic. His temperature was 37°c, had no jaundice and was well oriented to time place and person. His pulse rate was 100 beats per minute, regular and of good volume and the blood pressure was 100/60mmHg. Abdominal examination revealed gross diffuse distension, but there was no visible peristalsis. The abdomen was tense but there was no guarding. Percussion note was tympanic over the abdomen. Bowel sounds were greatly exaggerated. Hernia orifices were normal.

Digital rectal examination revealed an empty rectum, no masses, and normal rectal mucosa. The examining finger was stained with normal brownish stool.

Plain X-ray films were taken in the erect and supine positions. Gaseous distension of the large bowel extending from the pelvis to the diaphragm in an inverted "U" shaped was demonstrated. Air, fluid levels were visible in two limbs of the sigmoid loop (Balance's Sign). In view of the history, clinical examination and the x-ray findings, a diagnosis of acute large bowel obstruction due to sigmoid volvulus was made. The patient was prepared for emergency laparotomy as follows. Intravenous fluids were given. A urethral catheter was introduced. A nasogastric tube was used to decompress the stomach. Blood samples were sent for grouping and cross-matching, hemoglobin estimation. The haemoglobin was 12.5g/dl. Serum electrolytes could not be determined due to technical reasons. The patient's consent was obtained. The abdomen was shaved, injection Gentamicin and Flagyl were given at the time of operation.

Under general anaesthesia, and routine cleaning with iodine and spirit, draping of the abdomen was done as usual. A midline incision was made to open the abdomen. Immediately after the peritoneum was opened a grossly distended sigmoid colon was evident due to distension. Serous peritoneal fluid

was encountered in the peritoneum cavity. The distended twisted bowel was delivered through the abdominal incision with difficult, the twist at its base was through 360° in anticlockwise direction. The bowel was viable. Detorsion in clockwise direction was done. The viability of the sigmoid loop was confirmed. The length of the redundant colon estimated to be 30cm. The limbs of the sigmoid loop were close together whereas the sigmoid was noted to be long and freely mobile. The rest of laparotomy findings were normal. Mobilization of the redundant twisted sigmoid colon was done. The base of the twisted redundant sigmoid colon was then clamped by applying double intestinal clamps at the proximal and distal limbs. Resection of twisted redundant sigmoid colon was performed between clamps. The distal segment was long enough to exteriorize hence, resection with a double barrel colostomy was fashioned through a separate incision on the left iliac fossa. Warm saline was used to lavage the peritoneum. No drain was inserted, and the abdomen was closed in two layers by using prolene No. 0 and silk no. 2/0, to the peritoneum and rectus sheath and skin respectively. Intra-operatively, blood was not transfused since the estimated blood loss was less than 300ml. Post-operatively, the patient had his pulse, blood pressure, temperature, respiration rate and urine output observed and charted hourly during the first 6 hours. When he was stable, these were then recorded 6 hourly

for 48 hours. He had a nasogastric tube for continuous drainage, and a fluid input/output chart was started. The following were also ordered.

1. Intravenous fluids - D/saline 3L/24hrs
2. Antibiotics. Ampicillin 500gm 8 hourly and Flagyl 500mg 8 hourly for the first 3 days.
3. Injection Pethidine 100mg/im 8 hourly for the first 24 hours.
4. A colostomy appliance was ordered.

His post-operative course was uneventful since his vital signs remained stable, had no evidence of wound sepsis and his abdomen was soft. Furthermore his general condition fared well. On the third day the bowel activity had resumed as evidenced by the presence of bowel sound, and a functioning colostomy. He was mobilized and able to tolerate oral sips of tea and water. By the 4th day he had full bowel action, the intravenous drip was discontinued. His post-operative haemoglobin was 11g/dl. He started liquid oral diet. On the seventh day, the stitches were removed, he was taught how to take care of his colostomy and was allowed home on the tenth day. He was seen at the surgical clinic a week after discharge and thereafter seen monthly. After three months, he was assessed and found fit for colostomy closure as an elective procedure. However, his colostomy closure was done after six months. His haemoglobin level was 12.0g/L, and bowel

preparation was done one day before operation by total bowel irrigation. At night one day before operation, a nasogastric tube was inserted, and through it a total bowel irrigation was done with normal saline at a rate of 100mls per 20min till when the affluent was clear. Ampicillin and metronidazole were given peri-operatively. (at the time of operation and continued 3 days post-operatively).

The operation was performed under general anaesthesia. The colostomy was closed extra peritoneally, the wound was closed in one layer and the skin was sutured. He did well post-operatively and on the 8th day the stitches were removed and he was discharged on 10th day. He was reviewed at the follow-up outpatient surgical clinic twice in a period of three months, found to be in a good general condition and passing stool normally.

Table I: Sex and age distribution.

Age group	Sex		Total number of patients
	Male	Female	
11 - 20	4	-	4
21 - 30	6	2	8
31 - 40	5	-	5
41 - 50	5	1	6
51 - 60	2	-	2
61 - 70	1	-	1
70 - 80	4	-	4
Total	27	3	30

Note: Male/Female ratio was 9:1
Mean age was 34 years.

Table II: Clinical manifestations.

Symptom/Sign	Frequency
Abdominal pain	30
Abdominal distension	30
Constipation	30
Vomiting	10
Dehydration	10
Peritonitis	4
History of suggestive of previous attack	5

Note: All 30 patients presented with abdominal pain, abdominal distension and constipation.

Table III: Surgical procedures done in 30 patients with sigmoid volvulus

Procedure	Number of patients	Percent
Resection and primary anastomosis	12	40%
Resection and double barrel colostomy	16	53%
Resection and Hartmanns procedure	2	7%

Note: Resection and double barrel colostomy was the most preferable surgical treatment.

Table IV: Post operative complications

Type of complications	Type of operation		
	Resection and primary anastomosis	Resection and colostomy	Total
Wounds sepsis	1	4	5
Stitch sinuses	1	2	3
Colostomy retraction	-	2	2
Faecal fistula	-	1	1
Post-operative death	1	-	1
Total	3	9	12

Note: Mortality rate of 8% was recorded in patients who underwent resection and primary anastomosis.

DISCUSSION

Aetiology

The aetiology of sigmoid volvulus is unclear. Various theories have been proposed for centuries, each explaining the prevalence of sigmoid volvulus in different groups of people. Many of these theories, however, can be unified by a single underlying mechanism which is that sigmoid volvulus tends to occur in people with floppy redundant colons and narrow mesosigmoid attachments.[17,22] It is possible that congenital and acquired factors work together.

Two factors, whether congenital or acquired must be present before volvulus of the sigmoid colon can be formed.

1) A redundant sigmoid colon with a narrow base. The two limbs are closely approximated forming a narrow stalk.
2) A long free mobile mesocolon.

The condition is associated with other precipitating factors, which includes

1. Over loaded pelvic colon.[14]
2. Bands of adhesions.[14]
3. A number of diseases such as Chagas's disease, Parkinson's disease and ischemic colitis.[14]

PATHOGENESIS:

Sigmoid volvulus develops when a redundant sigmoid loop with a narrow base of attachment rotates about its mesenteric axis, producing a closed loop type of intestinal obstruction.[12]

Further pathological changes are caused by compression of mesenteric circulation and by rapid and immense distension of the rotated bowel impairing blood supply to the sigmoid loop.[3,17]

Rotation of the bowel starts with the upper limb of sigmoid loop descending in front of lower limb, the twist is either clockwise or counter clockwise.[12]

Sometimes a physiological volvulus of 180° (half turn) may occur without clinical symptoms usually resolving spontaneously. If spontaneous resolution does not occur, subsequent hyperperistalsis, particularly in a loaded colon may cause a further twist of 180° or more in the same direction resulting in a condition reducible with difficulty or one that is irreducible.[3] Increased peristalsis proximal to the obstruction, forces air and fluid into the twisted colon. The site of tension acting as a valve and progressive distension of the sigmoid loop occurs. It is the sustained intraluminal pressure in obstruction that alters or damages the bowel wall[18].

Occasionally bowel contents may be forced into the efferent loop causing intermittent diarrhoea despite predominant symptoms of obstruction.[3,17] In addition to the closed loop obstruction the proximal colon often becomes distended and if ileocecal valve is competent, as is the case in 75% of the patients,[18] no regurgitation is possible and a double closed loop obstruction develops. If there is incompetence, the small bowel becomes distended as well.[18]

Torsion of the mesentery by volvulus causes venous stasis and retrograde thrombosis of the mesenteric vessels with extensive gangrene.[3] Compression of two superior rectal vessels which run a looping course into the mesentery of the sigmoid colon result in gangrene which extends down to the upper rectum.[25] Increased distension of rotated bowel causes vascular hypertension in the involved loop impairing capillary circulation. Submucosal vascular plexuses are particularly vulnerable to the flattening force extorted by pressure within the lumen causing ischemic necrosis.[12] Hemorrhagic infarction of the bowel is sufficient to produce shock. Toxins, bacteria and blood pass into the peritoneal cavity causing peritonitis.[22] In practice, circulatory embarrassment should be assumed if the volvulus has been present for more than six hours.[6] Shepherd[22] found that 40% of patients attended with symptoms of acute sigmoid volvulus within 24 hours, already had gangrene.

In this study 4 of the 30 patients had gangrenous bowel at exploration. These patients presented to hospital three to five days after onset of abdominal pain.

Sigmoid knotting develops when an abnormally mobile small intestines with an elongated mesentery passes around the base of a freely mobile pelvic colon, the net effect of which is formation of the an ileal knot around the base of the pelvic colonic loop producing a double closed loop intestinal obstruction.[24] Early and severe strangulation of the knotted ileum occurs when the trapped loop of pelvic colon becomes engorged and moderately distended with air and fluid.

Gangrene of the sigmoid colon occurs in only 30% with cases of ileosigmoid knotting,[24] while the ileal loop involved in the knot almost invariably become infarcted, except the last 3-5cm of the terminal part which usually remains viable.[24]

In this study two patients out of thirty were found to have ileosigmoid knotting with gangrene of small and large bowels.

CLINICAL FEATURES

In this study all patients had history of abdominal pain followed by constipation and progressive abdominal distension (Table II). The mean age was 34 years. In another study from the tropics the peak incidence of sigmoid volvulus other occurred in the 40-49 years of age group.[25]

Symptoms of sigmoid volvulus often develop insidiously so that a patient may stay for two to three days before reporting to hospital.[17,21] In this study patients reported to hospital after one to seven days of onset of the abdominal pain. A history of previous similar attacks may be obtained in as many as 50% patients.[26] In this study five patients had similar attacks previously which subsided spontaneously. One had laparotomy at which time he underwent mesosigmoidopexy.

The clinical features are mainly due to mechanical bowel obstruction and included mild to moderate colicky abdominal pain, progressive abdominal distention and absolute constipation.

These symptoms occurred in all patients in this study. Diarrhoea may occur consisting of small amount of stool forced into the efferent limb of the sigmoid loop despite the obstruction.[3,17]

Vomiting occurs in some patients usually as a late event.[6] In this study 10 patients out 30 patients presented with vomiting. Early and severe vomiting suggest complicated volvulus associated with strangulation or ileosigmoid knotting.[22,24] When gangrene develop the features which may be suggestive include the following:-

1. Features of peritonitis i.e. abdominal guarding and tenderness.
2. Shock and ill looking toxic patient

3. Absent bowel sounds (silent abdomen)
4. Raised white blood cell count.
5. Presence of free gas under the diaphragm on plain X-ray.

The clinical sign of sigmoid volvulus is mainly the remarkable abdominal distension which may be so gross that the movement of the diaphragm is impaired.[7] There is little abdominal tenderness except with gangrene bowel.

There are two clinical types of sigmoid volvulus described by Hinshaw and Carter,[12] which correspond with the degree of bowel obstruction and strangulation.

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

The second clinical type is subacute sigmoid volvulus which is more common than the acute fulminating type and occurs usually in older patients. It has a gradual onset and often patients give a history of previous chronic constipation. Tremendous abdominal distension is usual and vomiting occurs late in the course of disease, probably due to competence of ileocecal sphincter mechanism. Gangrene develops more slowly and is a result of enormous rise in intra-colonic

pressure in the closed loop. The general condition of the patient is usually good, with a minimal distension of the abdomen.

DIAGNOSIS

The diagnosis of sigmoid volvulus is clinical in more than 80% of patients. This has significant implication particularly in circumstances where plain abdominal X-ray cannot be readily obtained.[15]

Plain abdominal X-ray:

This examination will demonstrate characteristic features of large bowel obstruction with gas shadow extending from the pelvis to the diaphragm, outlining the distended sigmoid loop in the form of an inverted "U". The terms local inner tube sign describes the outline formed by overlap of the sigmoid loop and two air fluid levels at the basic of the sigmoid loops have been likened to a pair of scales'.[16] The edematous double walls of dilated bowel are outlines by lines converging to the left iliac fossa where the mesocolon is twisted (Frimann Dahl's sign).[7] X-ray examination was done in all patients and twenty six of the thirty patients had typical findings.

Barium enema. This is usually not recommended in acute sigmoid volvulus and is absolutely contraindicated if gangrene is suspected.

When performed barium enema examination demonstrates a characteristic "birds beak" sign which shows barium filling the lower part of the rectum terminating at the point of the twisted sigmoid loop.[7,20] If barium enema X-ray examination is done in between attacks it will show a redundant sigmoid loop on a large capacity sigmoid colon.[26] Barium examination was not done in this study because all the patients presented during acute attacks and were managed as emergency cases.

TREATMENT

The management of the volvulus has been a controversial subject.[8,21,24] The aim of treating acute sigmoid volvulus is initially to relieve acute colonic obstruction, followed by measures to prevent recurrence by resection of the sigmoid colon, with minimal morbidity and mortality. Active management is required although spontaneous reduction of acute sigmoid volvulus has been reported.[25] The chief danger from volvulus is the development of impairment of the blood supply with necrosis of the bowel wall and perforation. Two modes of treatment are employed.

1) Conservative (Non-operation)
2) Surgical (operative)

i) Conservative Methods

(a) Spontaneous reduction and use of enema

Spontaneous untwisting of sigmoid volvulus has been known since ancient times. The Egyptians[1,10] wrote in the papyrus Ebers that "The natural history of sigmoid volvulus is either spontaneous reduction or rotting of the bowel". Bruusgaard[3] pointed out that sigmoid volvulus of less than 360° rotation may untwist spontaneously but when the twist is more than 360° the sigmoid loop is held fast between anterior and posterior abdominal walls and becomes less likely to untwist spontaneously. An ordinary or barium enema has been shown to cause reduction of a moderately twisted volvulus.[2] Shepherd[22] reported 9 out of 425 patients with acute sigmoid volvulus who got spontaneous relief while waiting operation. Spontaneous reduction of sigmoid volvulus is unpredictable and the results of enema are often uncertain and temporary. Sigmoid volvulus should therefore be managed actively from the beginning.[2] In this study spontaneous reduction did not occur and enema was not performed for reduction.

b) **Proctosigmoidoscopic or rectal tube reduction:**

Bruusgaard[3] reported a 90% success rate in reducing and deflating acute sigmoid volvulus by rectal intubation with a reduced mortality rate of 14.2%. This technique has been advocated by several other Workers.[2,25]

Some pass a tube through the twist after visualizing it at sigmoidoscopy, while others use the scope itself to open the spiral. Shepherd (1971)[25] extensively practiced the procedure of proctosigmoidoscopy with rectal intubation at Mulago hospital in Uganda and reported 88% success rate. Patients who are successfully decompressed by this means should undergo elective resection after proper bowel preparations and consideration of other medical problems. Arnold and Nance[2] have shown that elective resection of the sigmoid colon with primary anastomosis reduces the operative mortality and eliminates the need for staged operation. According to Shepherd (1971)[16], definitive treatment can be done as early as 7 to 21 days after the relief of the attack. Reduction by rectal intubation is contraindicated in patients with suspected gangrenous bowel. In those cases where sigmoidoscopic detorsion is considered, it is recommended that the procedure be done in theater with the patient well prepared for laparotomy should that need arise. Heavy sedation or anaesthesia should be avoided because pain during or after intubation is a valuable indication of trauma or gangrene which calls for immediate exploration.[25]

Rectal intubation may be complicated by perforation of the colon and also carries the risk of missing a

gangrenous volvulus. Even if deflation is successful, gangrenous bowel may still be missed or may develop later. Hence close monitoring before and after the procedure is emphasized.[22,23] In case non operative reduction fails or there is evidences of gangrene, it is mandatory to perform immediate exploratory laparotomy. In this study proctosigmoidoscopy and rectal tube reduction were not performed, probably because most of the patients came late to hospital, which raised the possibility of gangrenous bowel.

(2) **Surgical or operative detorsion:**

In volvulus of the sigmoid when the colon is viable several treatment option are available.

(i) When the bowel is viable operative detorsion with or without fixation (Colopexy[3]) has been advocated, but recurrence is a major threat. Hall-craggs[11] described a plastic procedure in which the base of sigmoid mesocolon is broadened in an attempt to prevent recurrence after detorsion of sigmoid volvulus. The procedure involves division of mesosigmoid perpendicular to the long axis of the bowel and re-stitching it parallel to the long axis of the bowel. In this way the mesosigmoid is shorter and made broader. The mesocolic fibrous band which provide the axis in which the sigmoid colon rotates is excised. It was an attractive procedure as it provided a means of

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preventing recurrence without the risk of opening the bowel with the advantage the patient did not require a second major operation. However, experience showed that the procedure did result in high recurrence rate of 41%, almost similar to that as simple detorsion.[22] This procedure was not performed in this study.

(ii) **Resection and Primary anastomosis:**

This procedure involved resection of the bowel and primary anastomosis of proximal and distal ends of the resected bowel. Sutcliffe[26] recommended the above procedure for treatment of acute sigmoid volvulus if the bowel was viable, but reported a mortality rate of 17.6%. This was also observed in another study by Baragani et al[4], who evaluated 31 patient with volvulus, of whom 18 patients were managed by resection and primary anastomosis with a mortality rate of 12.5%. This is contrary to a study of Kocak[27] who analyzed 31 patients with volvulus, of whom 5 patients were managed by resection and primary anastomosis with a zero mortality. In this study twelve out of thirty patients were managed by this method with a mortality rate of 8%. The patient who died was 22 years old male who had uneventful post-operative period untill the 5th day, by which time he was full, ambulated, he suddenly developed abdominal distension, went into shock, and collapsed, before

relaparotomy could be performed. No postmoterm was done, therefore the definitive cause of death was not established.

iii) **Paul-Mickulicz operation**

Another option in managing volvulus is resection and colostomy, which can be performed by colostomy with Paul-Mickulicz operation, Hartmann's procedure or distal mucous fistula. These procedures are later completed by colostomy closure.

Paul-Mickulicz procedure involves immediate resection of sigmoid colon and exteriorization of the proximal and distal ends of the bowel fashioning a double barrel colostomy. Bowel continuity is re-established about three months later as an elective procedures with proper bowel preparation[19]. Interference of blood supply to the colostomy may occur if the ends are held under tension or an injury of the superior rectal vessel which run a looping course at the base of the volvulus can cause gangrene of the rectal stamp.[22] If gangrene extends to the upper rectum, it may not be possible to obtain a long enough rectal stamp for exteriorization in which case a Hartmann's procedure or a colostomy with distal mucous fistula should be performed. Paul-Mickulicz technique allows subsequent closure of colostomy to be performed extra-peritoneally with minimal or no peritoneal

contamination. In this study 16 of the 30 patients underwent resection and double barrel colostomy as in Paul-Micklicz procedure. There was no postoperative death. The procedure is safer than resection and primary anastomosis, as it has lower mortality rate which has been observed in several series.[32,33] Where the mortality rate was 1%. However complications are common and included wound sepsis (4 cases), colostomy retraction (2 cases), and stitch sinuses (3 cases).

iv) **Hartmann's procedure/distal mucous fistula:**

In this procedure, the redundant sigmoid loop is resected the same as in the Paul-Mickulicz procedure, and the proximal end of the bowel is exteriorized fashioning a terminal colostomy at the left iliac fossa while the distal segment is closed and left in the pelvic cavity or exteriorized separately as mucous in the middle. Bowel continuity is re-established after 3 months at a repeat laparotomy[19]

Hartmanns procedure is recommended when there is extensive gangrene involving the upper rectum and when there is technical difficult, in mobilizing enough distal segment for exteriorization. This technique is relatively short hence is suitable for high risk patients.[11,26] Subsequent procedure to close the colostomy involves a laparotomy. Another disadvantage of Hartmanns procedure

is the difficult in locating the distal stump, deep in the pelvis. Hartmann's procedure is reported to be associated with high mortality. This is due to the fact that patients who are managed by this method are complicated cases of volvulus.[11,20] Totte et al[27] in analysis of 37 patients, who were managed by this method observed a mortality of 73%. This was also observed by Baragani[4] et al in a study of 31 patients where the mortality was 33.3%.

In this study, Hartmann's procedure was performed in two patients all of whom had extensive gangrene of small and large bowel, due to ileosigmoid knotting.

CONCLUSION AND RECOMMENDATION

Early diagnosis, with effective resuscitation and prompt surgery are the main treatment options for volvulus. Resection with primary anastomosis as an operative option is reported to have a higher mortality rate compared to resection and colostomy (by Paul Mickulicz) operation. This was observed in this study and other studies.[5,9] Therefore the author suggests that resection and colostomy should be encouraged as the best operation option in managing volvulus.

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BENIGN GASTRIC OUTLET OBSTRUCTION

SUMMARY

Between October, 1993 and July 1995, ten patients presenting with gastric outlet obstruction were managed in three surgical firms at Muhimbili Medical Center, Dar es Salaam. In all cases, gastric outlet obstruction was confirmed endoscopically. The ages of patients ranged from 21 to 70 years with mean age of 48 years, and all of them were males. History suggestive of peptic ulcer disease was present in 8 of the 10 patients. The duration of symptoms at presentation ranged from 1 year to 20 years, with a mean of 10 years. Two patients out of 10 patients had history of ingestion of packets of heroin, which were obstructed in their stomachs for an average of 10 days.

The clinical features consisted of vomiting (80%), loss of weight (80%), epigastric pain and fullness (100%), dehydration (80%), visible peristalsis (60%) succussion splash (80%), and abdominal mass (20%). Truncal vagotomy and gastrojejunostomy was the preferred treatment for most of the patients, and was performed in four patients. Other modalities of treatment were as follows; two patients out of ten patients underwent truncal vagotomy with pyloroplasty. Two patients underwent truncal vagotomy with antrectomy and two patients who had gastric outlet obstruction due to ingestion of packets of heroin were managed by laparotomy with gastrotomy. Two

patients out of ten, were initially managed with anti-H2 receptors prior to surgery. No death was reported in this study.

INTRODUCTION

Benign gastric outlet obstruction is caused by several conditions, peptic ulcer disease being the most frequent cause in adults.[1,22,24] Obstruction is encountered in ulcers involving either the duodenum or prepyloric region, that is why the term benign gastric outlet obstruction is more appropriate than "pyloric stenosis" which is anatomically inaccurate. Although it may produce serious vomiting and fluid and electrolytes disturbances it is rarely a threat to life.[22] Benign gastric outlet obstruction is significantly more common in developing than in developed countries and a recognized complication of peptic ulcer disease in many parts of the developing world.[10]

In developed countries despite recent advances in medical therapy for peptic ulcer disease, complications such as bleeding, perforation, and occasionally obstruction continue to occur. However, Penstom[20], observed that bleeding, perforation, and obstruction may be prevented by maintenance treatment with H2 receptor antagonists. But Gray[9] and associates observed that stenosis may develop for the first time in patients receiving H_2-receptors antagonists and it is conceivable that accelerated healing with these agents may

favor the development of stenosis. Penston[20] and associates in a 5 years retrospective study of 909 patients who were managed by H_2-receptor antagonists observed that the frequency of elective surgery were reduced by 40%, but the frequency of emergency surgery was increased by 72%. Therefore the introduction of H_2-antagonists has altered the role of surgery in peptic ulcer disease.[20,22] It is possible that medical therapy only postpones the need for surgery in certain patients but the natural process of the disease remains unaltered.[19] Presumably, these patients are still prone to ulcers complication. The complications associated with duodenal ulcer and the frequency of elective versus emergency surgery, vary depending on the population studied. Peptic ulcer disease is quite common in Africa. However, it is suggested that the lower incidence reported is probably due to failure of diagnosis rather than actual incidence.[12,27]

A case of benign gastric outlet obstruction is presented, the clinical features are described and discussed with reference to these among other causes of the nature and review of the literature.

CASE PRESENTATION

A.A. Reg. No. 826944, a 70 years male presented at Muhimbili Medical Center being referred from one of the peripheral hospital with a 6 months history of vomiting,

abdominal pain and fullness. Initially vomiting occurred once a day but had increased in frequency to 2 to 4 times a day. It was more marked in the evening and consisted of large volume of bile free fluid, usually contained food eaten in the morning or the previous night. Pain was located in the epigastrium and was constant but relieved by vomiting. He also had a sensation of abdominal fullness particularly towards the evening which was relieved by vomiting. The patient admitted to have lost weight over the past four months. He had no history of hematemesis. He also complained of constipation but there was no history of melaena. He also admitted to have a history of periods of epigastric pain over the past 10 years for which he was taking antacids, and in the past two months he was maintained on H_2-receptor antagonists (cimetidine). He denied smoking or drinking alcohol. He was married with six children. This was the second admission to hospital.

Physical examination revealed a dehydrated man with no pallor, jaundice, enlargement of cervical or axillary nodes. His pulse rate was 100 beats per minute. regular, and a blood pressure of 110/60. Abdomen was asymmetrically distended around the umbilicus with visible peristalsis from left to right. The liver, spleen and kidneys were not palpable, and there was no other palpable mass. Succussion splash was positive. There was no ascites. A digital rectal examination was normal. A Ryles tube was inserted and about 2L of

fermented fluid, free of bile was aspirated. The abdomen distension disappeared following the procedure. The tube was left in situ for gastric aspiration. A provisional diagnosis of gastric outlet obstruction probably due to chronic peptic ulcer disease was made. The following investigations were done. Full blood picture: Haemoglobin was 10gm/dl with normal red cell morphology. White Blood Count was normal. Serum creatinine was 80 micromol/L, electrolytes estimation was not done due to technical reasons. Barium meal was done and revealed a dilated stomach with elements of narrowing at pyloric duodenal region. This was confirmed by gastroscopy which also revealed a dilated stomach with massive fluid retention. The pylorus was deformed, edematous and constricted, and through the pyloric canal an ulcer crater was visualized.

A decision to operate was made and the stomach was lavaged with normal saline until all debris was removed. Blood was taken for serum electrolytes, creatinine, hemogram and four units of blood were crossmatched. Fluid and electrolytes defect was corrected with normal saline and Darrow's solution. A consent from the patient was obtained. In theater, under general anaesthesia, the operating site was cleaned and draped and upper midline incision was made. The left lobe of the liver was retracted and the abdomen was explored. The stomach was found to be grossly dilated with

scarring, oedema and adhesions around the pylorus and the first part of the duodenum. The liver and gall bladder were normal. The regional nodes in the greater and lesser curvature, and the coeliac group of nodes were not enlarged. The small intestines and colon were normal, then they were carefully packed off.

The lower esophagus was identified by palpation of nasogastric tube and a transverse incision was made over the overlying peritoneum. The lower 5cm of oesophagus was then mobilized by gentle dissection, and was hooked by a tape. The stomach was pulled downwards by an assistant, this tensed the vagal trunks which were identified more easily. The main trunks starting with anterior then posterior trunk were divided between clamps and ligated. Specimens of the divided part were taken for histology. A search for additional vagal fibers was done and those found were divided. Then a retrocolic posterior gastrojejunostomy was performed. The greater omentum and the transverse colon were brought out of the wound turned upward and held under traction by an assistant. With a blunt dissector, a vertical opening was made in the avascular mesocolon between the middle colic and left colic arteries. It was gently stretched by fingers to a length of 10cm. The stomach was pulled through the opening. An area of the jejunum was identified about 15cm from the duodenal jejunal flexure. A segment of jejunum was isolated

between intestinal clamps. The posterior wall of the stomach was then exposed. Two stays sutures were placed to the stomach muscular-wall at the extremities of the area selected for anastomosis (the most dependent part of the stomach). The stomach was then anastomosed to the jejunum in two layers with 2/0 catgut (side to side anastomosis). Then a Brown's reconstruction was done 5cm below the anastomotic site. The transverse mesocolon was secured to the stomach by three interrupted sutures on each side of the anastomosis. Packs were then removed and hemostasis was achieved. The abdomen was closed in layers using continuous monofilament nylon N.o 1 for rectus sheath and interrupted silk for skin.

Post-operatively, the patient made an uneventful recovery. By the third day the nasogastric aspirate had decreased considerably and the nasogastric tube and fluids were therefore discontinued and he was allowed light meals. The specimen taken for histology confirmed to be nerve tissue. Stitches were removed on the 8th day and patient discharged on 10th day for follow-up at surgical outpatient clinic. He was followed on monthly basis for 4 months, where his general condition improved. He was eating a normal diet, had no abdominal pain and was gaining weight. He was advised to continue to report at outpatient clinic every 6 months. Characteristics and management of other patients are summarized in the tabular form.

Table 1: Age and sex distribution patients with gastric outlet obstruction

Age group	Males	Females	Total
21 - 30	2	-	2
31 - 40	1	-	1
41 - 50	1	-	1
51 - 60	5	-	5
61 - 70	1	-	1
Total	10	-	10

NB: Male to female ratio was 10:0
Mean age was 48 years

Table 2: Causes of obstruction.

Cause	Number of patients	%
Duodenal ulcer	5	50%
Prepyloric and Pyloric ulcer	2	20%
Combined duodenal and gastric ulcer	1	10%
Foreign body in the stomach.	2	20%

NB: Two patients who had foreign bodies in their stomachs had history of ingestion of sachets of heroin.

Table 3: Clinical features

Sign and symptom	Number of patients
Vomiting	8
Loss of weight	8
Epigastric pain/ Abdominal fullness	10
Previous H/o of PUD	8
Dehydration and wasting	6
Visible peristalsis	6
Succussion splash	8
Abdominal mass	2

NB: The two patients who presented with abdominal mass, had history of ingestion of sachets of heroin.

Table 4: Mode of treatment.

Treatment	Number of patients
Anti H2 receptors prior surgery	2
Truncal vagotomy and drainage (i) Gastrojejunostomy (4) (ii) Pyloroplasty (2)	6
Truncal vagotomy with Antrectomy	2
Laparotomy with Gastrotomy + removal of foreign bodies	2

Note: Truncal vagotomy and drainage was the most preferable surgical treatment.

DISCUSSION

Gastric outlet obstruction is a significantly more common complication of chronic peptic ulcer disease in African countries south of Sahara, a fact documented by numerous reports.[1,10,12,26,27] This pattern is different from that seen in developed countries where bleeding and perforation are common and obstruction is an occasional complication.[10] These differences between developing and developed countries could be due to availability of diagnostic facilities, such as fibre optic endoscopy in the latter facilitating diagnosis. Furthermore, patients with bleeding or perforation may die before reaching hospital and that most reports in Africa could be surgically biased. Tovey[26] observed that it is possible that obstruction develops more frequently and rapidly in developing countries than in developed countries. The average duration in developing countries was 5 years which in developed countries was 10-20 years, from the initial symptoms of peptic ulcer disease to the development of obstruction. In this study the average duration was 10 years.

In general, peptic ulcer disease mostly affects a younger age group between 30 to 50 years.[12,26,27] As would be expected obstruction in African countries occurs at an earlier age. In this study the mean age was 48 years. This compares well with other African countries[1,12,26,27] with a peak age between 45 to 55 years, in contrast to mean age in a British series which is

71 years.[10]

Peptic ulcer disease is predominantly a male disease. The male to female ratio shows great variation around the world. In England it is 2:1, India 2:1 and in Africa it is 9:1.[10,16] In this study the male to female ratio was 10:0

Aetiology

The most common cause of benign gastric outlet obstruction is peptic ulceration. The site of ulceration is duodenal in 75% of patients.[22] In a small number of patients the site of ulceration may be pyloric or prepyloric region. In this study site of obstruction was as follows, in the duodenum 5 patients (50%), prepyloric in two patients (20%), and combined duodenal and pyloric ulcers in one patient (10%). An interesting feature is that two patients out of ten of gastric outlet obstruction, had obstruction due to ingestion of foreign bodies (packets of heroin). One was found to have 47 packets of heroin in the stomach, and the second patient was found to have 37 packets of heroin. They had an obstruction for an average of ten days.

Other causes of benign gastric outlet obstruction include:-

1. Benign tumors: These are rare causes of benign gastric outlet obstruction. Tumors in the antrum may grow to a sufficient size to cause obstruction. It is not uncommon for a pedunculated polyp to prolapse through the pylorus

or present at the gastric outlet-causing intermittent obstruction, eg. lipoma, polyps etc.[4,6]

2. Hypertrophic pyloric stenosis which usually presents in infants many occasionally become manifest in adult life as adult hypertrophic pyloric stenosis.[3]
3. Pancreatic cysts in the pylorus is also are rare cause of obstruction.[6]
4. Intragastric bezoars (trichobezoars and phytobezoars) can grow to size sufficient to cause obstruction.[6]
5. Bouverets syndrome,[5] this is a gastric outlet obstruction by a migrating gallstone into the duodenum.
6. Post gastric surgery complications[2,3,14,18,19]

   Stricture formation after gastric surgery may be formed after the following type of surgery.eg.

   (i) Pyloroplasty (especially Heinke-Mickulicz)

   (ii) Gastrojejunostomy etc.

PATHOGENESIS

Gastric outlet obstruction occurs during the course of ulceration and inflammation in the pyloric channel or duodenal bulb, followed by healing of the ulcer and deposition of scar tissue. Over a period of 10 to 15 years, with repeated episodes of duodenal ulceration, healing and fibrosis, the pyloric channel or duodenal bulb becomes relatively scarred and stenotic with increasing gastric outlet obstruction. An

active ulcer crater is frequently, but not always present. In most patients, the obstruction is incomplete and some food, gastric juice, or ingested barium by roentgenography can be demonstrated to leave the stomach slowly. As stenosis develops the stomach compensates by means of hypertrophy of its muscular wall and a forceful peristalsis in the antrum. Eventually as stenosis become increasingly severe, the stomach becomes atonic, ingested material and gastric secretions gradually accumulate to produce gastric dilation.

In some patients, because of persistent or chronic gastric stasis, gastric ulceration may develop secondary to the duodenal ulcer and may be the indication for operation. In a study by McMahon[17] it was reported that obstruction due to inflammation and edema constitutes one third of patients and due to true fibrosis constitutes about two thirds. In this study it was observed that four patients had obstruction due to inflammation and edema, while other four patients had obstruction due to true fibrosis.

CLINICAL FEATURES AND DIAGNOSIS

A history of previous peptic ulcer disease, vomiting and loss of weight, epigastric pain or abdominal fullness was present in 8 patients out of ten patients. Common physical findings were wasting, dehydration, and a post fasting succussion splash found in 8 patients while Visible

peristalsis was found in 6 patients. Only abdominal mass was palpable in two patients, who had obstruction due to ingestion of packets of heroin. Obstruction is usually followed by progressive vomiting of food eaten the previous day. Vomiting can either be spontaneous or induced and usually relieves pain and abdominal fullness. Patients also present with anorexia and morbid aversion to food.[18] The mean duration of symptoms before obstruction was 10 years in this study, while a duration of 5 years is reported in other African countries[1,12,27] and India.[10] In USA and Europe the duration is much longer ranging to 10-20 years.[10,11,21]

The diagnosis of obstruction is easily made on clinical grounds and can be confirmed by placement of nasogastric tube with finding of resting gastric juice of more than 300 mls or more, containing undigested food residue after an overnight fast.[14]

Radiological features are fairly characteristic. An abdominal plain film may show evidence of gastric stasis with a large gastric shadow and a large amount of gastric fluid. Barium meal examination shows marked gastric dilation with a large gastric residue containing food debris. The barium would normally be emptied from the stomach in less than 2 hours, but in obstruction the barium may be emptied a little or not at all up to 6 hours after ingestion.

Endoscopy is by far the most important investigation. In this study the diagnosis was confirmed by endoscopy in all patients. If the stenosis is the results of duodenal ulceration, the endoscope may pass through a normal pylorus and demonstrate duodenal ulceration and stenosis. However, in prepyloric ulcer stenosis or inflammatory oedema, sometimes the endoscope cannot be negotiated into the duodenum. In this case biopsies must be taken to exclude a malignant gastric outlet obstruction.

Both radiology and endoscopy are reliable methods of diagnosis. However, endoscopy provides access to tissue biopsy and radiology gives more information about the function, severity and length of stenosis.

TREATMENT

The objectives of the management of a patient with gastric outlet obstruction are correction of fluid and electrolytes deficit, and gastric decompression. In obstruction due to ulcer, treatment costs of intensive therapy and eventually surgery to relieve obstruction and permanently reduce acid secretion.

1. **Correction of fluid electrolytes disturbances:**[21,25]

   The volume deficits are estimated on the basis of severity of clinical signs and body weight loss. A mild loss represents a loss of approximately 5 percent of body

weight, and is clinically not detectable and loss of more than 10 percent of body weight is severe deficit and is dominated by circulatory signs. One kilogram is equal to one liter of water. Adequacy of volume replacement is judged by frequent clinical observation. Stabilization of blood pressure and pulse, a urine output of more than 30-50 mls per hour and appearance of alkaline urine, indicate adequate volume replacement.

2. **Gastric decompression:**

While correcting fluid and electrolytes disturbances, gastric decompression is begun to eliminate gastric dilation and restore gastric tone. This is achieved by insertion of nasogastric tube and aspirations every 12 hours usually for 3 to 4 days. Continued gastric aspiration by removing secreted gastric acid, will reduce inflammatory edema and may establish gastric emptying.[18] However, this cannot be achieved in cicatricial stenosis. Gastric emptying may be tested at bed side by saline load test. If more than 400mls of saline remain in the stomach 30 minutes after instilling 750 mls of normal saline, emptying is impaired and surgery should be considered.[14] If there is emptying, oral fluids, caloric food and ulcer therapy should be started.

3. **Ulcer therapy:**

It is usually indicated where obstruction is due only to inflammation and oedema. In this study 2 patients were initially managed by cimetidine 400mg twice daily for 6 weeks. Symptoms improved during the period of treatment. However, all had recurrence of symptoms, a few weeks after stopping treatment. It was then decided to perform surgery as definitive treatment. Gray and associates[9] noted development of stenosis in 4 patients out of 50 within a year of having been given intensive ulcer therapy of 1 g/day of cimetidine for one month followed by 400gm at night as a maintenance dose for 60 months. In this study modality of treatment was as follows; two patients had anti H2 receptor drugs before surgery, six patients underwent truncal vagotomy and drainage procedure (60%), two patients underwent truncal vagotomy with antrectomy (20%), while the remaining two patients underwent laparotomy and gastrotomy for removal of foreign bodies.

Surgery:

Cicatricial stenosis[4,18,19] is an absolute indication for surgery. Basically a drainage procedure consists of pyloroplasty, duodenoplasty, or gastrojejunostomy. There is no absolute consensus as the most appropriate operative treatment for benign gastric outlet obstruction. Truncal vagotomy and

drainage by either pyloroplasty or gastroenterostomy is the most commonly used procedure in this center. It is favored because of its low operative mortality as most of the patients are in poor condition with respect to nutritional status and electrolytes disturbances. It is also relatively easy to perform with a short operating time. In the series[2] from U.S.A. the mortality rate after 1000 truncal vagotomy and drainage was 0.5%. The recurrence rate from various series which were summarized by Johnson[14] was as high as 27.3%.

The trend nowadays is to perform proximal gastric vagotomy[3,7,16] in patients with benign gastric outlet obstruction due to duodenal ulcer diseases. McMahon and associates[17] in a study involving 23 patients with duodenal ulcer and obstruction who underwent proximal vagotomy and digital dilation and followed from 4 months to 5 years, reported good results in 96% of patients. However, they noted a 4% incidence of failure due to restenosis. Barroso and associates[3] performed duodenoplasties in 43 patients with peptic duodenal stenosis as a complementary procedure to proximal gastric vagotomy. Patients were followed up for 8 years with a 6% recurrent rate of obstruction due to recurrent ulcer.

Vagotomy and antrectomy[15] is widely acknowledged to be the most effective procedure available for curing ulcers since both vagal and gastric drive on parietal cell is removed.[13]

In the series[2] from U.S.A. the mortality after 3500 truncal vagotomy and antrectomy operations was 1.6%, with recurence rate of 1%. The recurrent ulcer rate after operations which were summarized by Johnson[14] was 3.3%. This method was used for two patients with prepyloric ulcer. It has been recommended in recurrent ulcer after proximal gastric vagotomy and in prepyloric ulcers.

Laparotomy with Gastrotomy:

Two patients in this study underwent laparotomy with gastrotomy for removal of packets of heroin, which were obstructed for average period of ten days. Report from the chief chemist proved that these packets contained heroin.

CONCLUSION AND RECOMMENDATION

Duodenal ulcer is the commonest cause of benign gastric outlet obstruction in our environment. Over 80% of patients have a long history suggestive of ulcer disease with a mean duration of 10 years. Clinically it manifested by copious vomiting, loss of weight, epigastric pain and/or fullness. Truncal vagotomy and retrocolic gastrojejunectomy is the preferred treatment as it avoids placing an anastomosis or closure in the scarred duodenum.

The advent of H2-blockers and inhibitors of parietal cell H+, K+ adenosine triphosphatase, and advances in the diagnosis of peptic ulcer disease has radically changed the management

of peptic ulcer diseases.

It would be interesting to study the frequency of complications in the era-of H2 receptor antagonist in our environment. Another area of study would be to see the percentage of patients actually having access to H2 receptor blockers, since this drug is expensive for an average Tanzanian.

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SURGICAL CHOLESTASIS IN ADULTS

SUMMARY

Ten patients with extrahepatic cholestasis were seen and managed at the Muhimbili Medical Center from October, 1993 to July 1995. There were 8 females and 2 males, making a female to male ratio of 4:1. The youngest patient was 30 years old and the oldest was 63 years old, with a mean age of 46 years. Seven patients had carcinoma of the head of pancreas, two patients had a cholelithiasis with choledocholithiasis, and one patient had tumor of the CBD. Diagnosis was confirmed at laparotomy and histopathology.

The main presenting complaints were pain over the right upper abdomen and itching in 8 patient, jaundice in 10 patients, passing dark urine in 6 patients, passing pale stool in 6 patients, weight loss in 8 patients, and fever in 2 patients.

The procedures done in the management of these patients included, cholecystojejunostomy in eight patients (80%), cholecystectomy with choledocholithotomy in two patients (20%). The operative mortality was zero. One patient is discussed in detail with review of literature.

INTRODUCTION

Cholestasis is a condition whereby there is failure of bile flow from the liver into the duodenum[17]. Obstruction may be either intrahepatic (involving the finer radicles of the biliary tree), or extrahepatic (obstruction of the major bile ducts). The former usually results from medical causes, while the latter is usually a surgical problem. Despite fundamental differences the syndrome resulting from cholestasis is essentially similar regardless of the site of obstruction. The distinction of cholestasis is important as an error in diagnosis may result in an operation for suspected extrahepatic bile duct obstruction only to find hepacellular disease or patients may be treated conservatively for lesions that could have been managed by surgical intervention. Patients with surgical cholestasis usually present with jaundice, pruritus, passage of dark frothy urine, pale stools and hepatomegaly.[18] When presented with a jaundiced patient the clinician is faced with several problems, chief among which is to decide whether the course of jaundice will need surgical intervention or not.[19] Even when the clinical diagnosis of cholestatic jaundice is made, implying that some surgical intervention may be necessary, there is still the question of defining the anatomical site of the problem and pathology.[3] After anatomical and pathological diagnosis the problem of management needs to be addressed in terms of the

type of surgery and whether one is planning a curative or palliative procedure. This is particularly true in patients with malignant disease. Cholestasis is a challenging problem right from the time of diagnosis, pre-operative preparation of the patient and in planning treatment best suited to the individual patient. In this study a case of cholestasis is presented in detail and management of patients with cholestasis is discussed with review of literature on the most common causes as seen at Muhimbili Medical Center, Dar es Salaam.

CASE PRESENTATION

M.A. (Reg. 807697), a female aged 55 years was admitted to Muhimbili Medical Center in December, 1994 having been referred from one of the city dispensary. She presented with yellow colouration of the sclera associated with general body itching of 2 months duration. She had noted yellow colouration of the sclera which was associated with passing of dark urine 2 months prior to the date of admission. The jaundice was progressive without remission. This was later followed by itching of the whole body which did not respond to medicines which she was prescribed. She was passing clay-coloured stools. She had abdominal pain which was dull aching in nature localized on the upper abdomen. She also experienced loss of weight. She had no fever. She had not been given any blood

transfusion nor had she been given any parental drugs in the past. She had no previous abdominal surgery or tooth extraction. She denied drinking alcohol. No other family member or village mate had similar complaints.

Clinical examination revealed a middle aged woman who was deeply jaundiced. She was afebrile with a temperature of 37°C. She had scratch marks all over the skin. Cervical and axillary lymph nodes were not enlarged. She had no tremors, spider naevi, caput Medusae nor palmar erythema. She had normal breathing. Abdominal examination revealed a soft abdomen which was not distended and non tender. The liver was palpable about 6 cm below the right subcostal margin along the right mid-clavicular line. It was smooth with a sharp margin and it was tender. The gall bladder was not palpable. There was no ascites and splenomegaly. Rectal digital examination revealed no masses and the examining gloved finger was stained with clay-coloured stool. The respiratory and cardiovascular systems were normal. A provisional diagnosis of extrahepatic cholestatic jaundice was made.

Investigations done included:

1. Full blood picture:
   - Haemoglobin 10.9 g/dl.
   - Normal peripheral film
2. Urinary bilirubin was strongly positive and urobilinogen was negative.

3. Serum bilirubin - total 300 micromoles/liter
   - Direct 208 micromoles/liter
4. Serum Alanine Aminotransferase (ALT) 100 I.U./L
5. Serum Aspartate Aminotransferase (AST) 90 I.U/L
6. Serum Alkaline Phosphatase (ALP) 480 I.U./L
7. Prothrombin time was 28 seconds which was 20% of the control.
8. Hepatitis B. Surface Antigen - Negative

Ultra-sound scan of the abdomen was done and this revealed distended gall bladder, dilated CBD and intrahepatic biliary ducts, and an ill defined mass in the pancreatic region. No stones were seen. A percutaneous transhepatic cholangiogram was recommended but it was not done due to technical problems. In view of the prolonged prothrombin time the patient was given vitamin K_1 10 mg intramuscular daily and after a week the prothrombin time was 17 seconds which was 46% of the control. After another week of vitamin K1 the prothrombin time was 12 seconds, 85% of the control. As the prothrombin time now had improved to near normal it was decided to operate on her.

Pre-operative preparations included the following:

1. Blood grouping and cross-matching 2 units of blood and haemoglobin estimation which was 11.0gm/dl.
2. Fresh frozen plasma

3. Pre-operative antibiotics (Cefuroxine) given 30 minutes before operation.
4. Vitamin K 10 mg im daily for two weeks.
5. Intravenous normal saline 3 litres in 24 hours.
6. Catheterization chart of fluid in and out 24 hours before operation.
7. Insertion of nasogastric tube
8. Signed consent was obtained from the patient

In the operating theater under general anaesthesia routine cleaning and draping was done. The abdomen was opened and explored. The intra-operative findings were:

- Distended gall bladder, a hard mass originating from the head of pancreas extending to the duodenum and lesser omentum, transverse mesocolon and fixity to retroperitoneum tissue. Other organs were free from metastasis. A transduodenal wedge biopsy was taken from the tumor. A point 18 cms from the Treitz ligament on the jejunum was identified and anterior cholecystojejunostomy was done in two layers. The abdomen was closed in layers. Post-operatively the patient was managed with intravenous fluids and cefuroxine 750 Mg IV 8 hourly for 3 days. She also continued with intramuscular vitamin K1 daily for one week.

On the third postoperative day the nasogastric tube was removed. On the fourth day, the intravenous drip was removed

and the patient started on oral intake. On the 8th day stitches were removed. Two weeks post-operatively the bilirubin total was 130 millimoles/liter and direct was 90 millimoles/liter.

The jaundice had subsided and stool regained the brownish color and itching disappeared. She was finally discharged home in good condition two weeks post-operatively. She has not been seen for follow-up. Histology result from the biopsy of the tumor revealed ductal adenocarcinoma of pancreas.

The characteristics and management of other patients is summarized in a tabular form.

Table I: Age and sex distribution among 10 patients with cholestasis

Age in Years	SEX		Total	%
	M	F		
21 - 30	0	1	0	10
31 - 40	1	2	3	30
41 - 50	1	2	3	30
51 - 60	0	2	2	20
61 - 70	0	1	1	10
TOTAL	2	8	10	100

NB: Female to Male ratio was 4:1
The mean age was 46 years

Table II: Aetiology of 10 patients with cholestasis

Aetiology	Frequency among patients
Carcinoma head of pancreas	7
Tumor of common bile duct	1
Cholelithiasis with choledocholithiasis	2

Majority of patients had a malignant disease .

Table III: Age of patients with cholestasis in relation to etiological factors.

Age group in years	Benign	Malignant
21-30	1	0
31-40	1	1
41-50	0	3
51-60	0	3
61-70	0	1
Total	2	8

NB: Young adults (mean age 35 years) had benign disease (gall stone) while malignant disease were the cause in older age group (mean age 50 years).

Table IV: Clinical manifestation

Presenting Symptoms/Signs	Frequency Among Patients
Jaundice	10
Itching	8
Abdominal pain	8
Weight loss	8
Passing dark urine	6
Passing pale stool	6
Fever	2

Table V: Management of 10 patients with cholestasis

Surgical Procedure	Number of Patients	%
Cholecystojejunostomy	8	80
Cholecystectomy and choledocholithomy	2	20

DISCUSSION

Aetiology and pathophysiology

Extrahepatic cholestasis refers to anatomical blockage at a site from beyond the porta hepatis , to the duodenum. It is characterized clinically by jaundice, dark urine and pale stools.[7]

The important causes of extrahepatic cholestasis are carcinoma of the head of pancreas, gallstones in the bile ducts, ampullary tumors, stricture of common bile duct, metastatic carcinoma in the lymph nodes at the porta hepatis, and parasitic infections with ascaris[6] Less common causes are carcinoma of the gallbladder, traumatic pseudo-pancreatic cyst suppurative and sclerosing cholangitis.[2,5,12,17]

In this study, 8 patients out of 10 patients had cholestasis secondary to malignant disease of pancreas, and ccmmon bile dut, while 2 patients had cholestasis secondary to choledocholithiasis. This is contrary to a study of Parks[15] in Europe who analyzed 187 patients with cholestasis and reported that in 144 patients the cause was a benign disease (choledocholithiasis). While in 43 patients it was carcinoma of pancreas.

In this study it was observed that two patients with benign disease were young adults with a mean age of 35 years, while those with malignant diseases were older with a mean age of 50 years. This observation has also been reported by other

authors.[1,4,6] Furthermore it was also observed that the two patients with choledocholithiasis were all females, while in 8 patients with malignant disease, six were females and only two were males.

CLINICAL PRESENTATION OF CHOLESTASIS

Patient develop jaundice slowly but generally feel well, which is in contrast to jaundice associated with hepatocellular disease where malaise and physical deterioration are common.[17] It is important to note whether jaundice is progressive or intermittent since intermittent jaundice may indicate choledocholithiasis. Jaundice and fever may suggest cholangitis. Pruritus is a common presenting symptom. It is often attributed to bile salts deposition in the skin although experimentally this has never been established moreover in terminal liver failure while bile salts may be increased pruritus may disappear.[17] However, the association of pruritus with cholestasis suggests that this is due to a substance normally excreted in bile.[17] The relief of pruritus by cholestyramine, a bile salt chelating agent suggest that bile salts are involved in its pathogenesis. Often when patients note jaundice they also note passage of dark colored urine. This is due to presence of bilirubin in urine. Patients may also note a change in the color of faeces which become pale or clay colored. This is a good indication

Cholestyramine

as to whether cholestasis is total, intermittent or decreasing.[17] Since bile salts are important in the absorption of fats, steatorrhoea may also result, and the severity of steatorrhoea is proportional to the depth of jaundice. In association with malabsorption of fats which leads to deficiency of synthesis of Vitamin K dependent clotting factors these patients may present with spontaneous bleeding disorders. Pain may or may not be present. Biliary colic may be present in three quarters of patients with choledocholithiasis who have cholangitis.[6,17] Pain is also reported to be present in 34% of patients who have pancreatic cancer.[24] Fever when present suggests cholangitis and is present in a third of patients with choledocholithiasis who develop jaundice and cholangitis.[4,24] Weight loss may be one of the major manifestations in patients with pancreatic cancer,[24] and in cancer of the bile duct.[16,22] In this study the main presenting complaints were as follows; pain over the upper abdomen in 8 cases, jaundice in 10 cases, passing dark urine in 6 patients, passing pale stool in 6 patients, weight loss in 8 patients and fever in 2 patients.

Investigation of the jaundiced patient can be classified in two major groups:

A. Serum biochemistry and urine examination

(i) **Urine examination:**

Urine bilirubin is positive. This is also evidenced by the dark color and on shaking the urine becomes frothy. Urine urobilinogen is usually negative, but may be positive depending on the degree of obstruction (partial obstruction).

ii) **Serum biochemistry**

Serum bilirubin is raised, mainly the direct reacting types. When cholestasis is relieved, serum bilirubin falls slowly to normal.[17] The alkaline phosphatase is generally greater than three times normal and the serum transaminases are less than 200 units/ml. Serum albumin and globulins are normal in acute cholestasis. With the development of biliary cirrhosis serum albumin tends to fall with inversion of albumin/globulin ratio.[17] There is prolongation of prothrombin time which is corrected by parenteral administration of Vitamin K_1. This is due to failure of synthesis of Vitamin K related clotting factors resulting from malabsorption of Vitamin K and other fat-soluble vitamins.

B. Imaging of the Hepatobiliary system

1) Non invasive procedures:

- Ultrasonography

It is recommended as the initial screening procedure in patients with obstructive jaundice. Its diagnostic accuracy for dilated ducts is 95%. However, its diagnostic accuracy for the cause of

duct dilation is low (less than 10%).[8] False-negative results can occur in 5 to 20%.

- Technical limitation can be encountered in patients with:
  - i) Early or partial biliary obstruction.
  - ii) Altered hepatic architecture because of scarring from sclerosing cholangitis or cirrhosis.
  - iii) Excessive ascites, adipose tissue, or overwhelming bowel gas.
- Visualization of the hepatobiliary is independent of liver function.

This was the prime imaging method used in this study.

2) Computed axial tomography (CT scan).

Abdominal CT scanning can also be used as a diagnostic investigation in patients with cholestatic jaundice. When compared to ultrasound, CT scan is a more expensive screening test and exposes the patient to small amounts of radiation.[8] Parks[15] assessed the accuracy of ultrasonography (US), computed tomography (CT) and endoscopic retrograde cholangio-pancreatography (ERCP) in distinguishing between benign and malignant causes of cholestasis in which final diagnoses were made

on histology, cytology and operative findings. The calculus nature of the cholestasis was correctly defined by US, CT and ERCP in 22%, 25% and 90% of patients respectively. While accuracy for diagnosis of pancreatic cancer was correctly defined by US, CT and ERCP in 60%, 79% and 60%.

(c) Radioisotope imaging

- Hepatobiliary imaging by use of technetium-labelled organic acid should only be used as a complementary test in evaluating cholestatic jaundice. Its most important contribution is in establishing the diagnosis of acute cholecystitis. The technectium-labelled organic acid is injected intravenously and is removed from the blood by hepatocytes and excreted into the biliary system. With obstruction of the cystic duct (acute cholecystitis) the gallbladder will not be visualized, since the radio-labelled agent is excreted into bile, it is traced down the common bile duct and into the intestine. If there is obstructive jaundice the tracer will not be detected in the intestine supporting the diagnosis of obstructive jaundice. Hepatobiliary imaging studies are not helpful

when patients have a serum bilirubin greater than 2mg/100ml.[22]

II. Invasive procedures

(a) Endoscopic retrograde cholangiopancreatography (ERCP)

Under direct vision, a side viewing endoscope is passed orally to the papilla of Vater. The ampulla is cannulated, and radiographic contrast is injected into the biliary system and into the pancreatic ducts. Standard x-rays are taken after injection of contrast. Successful completion of ERCP depends on the endoscopist. With appropriate expertise completed cholangiography can be expected in about 85% of patients and completed pancreatography in greater than 90%.[8]

Since ERCP does not depend on the duct size for successful completion, it is the procedure of choice for the evalution of an apparent non dilated ducts in the setting of cholestatic jaundice.

ERCP allows cytologic and histologic confirmation of certain lesions.

- In appropriate cases it also allows therapeutic interventions.

(i) Papillotomy with gallstone extraction.

(ii) Common bile duct or pancreatic stent placement.

(iii) Dilation of CBD or pancreatic duct strictures. While performing ERCP the following complication can be encountered, pancreatitis in 1-4%, sepsis in o.8% drug reaction in 0.6% and rarely death.[8]

2. Percutaneous transhepatic cholangiography (PTC)

- A percutaneous cholangiogram is performed by passing a small needle directly into the intrahepatic bile ducts under fluoroscopy. Radiologic contrast is injected to outline the biliary anatomy and standard x-rays are taken. Successful completion of PTC is somewhat dependent on intra hepatic bile duct size. Consequently, this is not the procedure of choice in patients with presumed non dilated ducts and cholestatic jaundice. With dilated ducts the success rate is 85 to 90%. (with non dilated ducts the success rate is 50 to 60 percent).[8] Like ERCP, PTC allows therapeutic intervention such as biliary drainage, cytologic sampling, biliary stent placement and stricture dilation. Unlike ERCP, PTC does not

visualize the pancreatic ducts or the periampullary region. Since the technique involves a percutaneous approach, the procedure is contraindicated in patients with uncorrectable coagulopathies. This method was not used in this study due to technical reasons.

3. Operative cholangiography

This procedure is performed in the operating room at the time of surgery for biliary tract disease with injection of contrast material either in the cystic duct or T.tube placed in the common bile duct[20] The aim is to establish whether there is need to open and explore the CBD.[1,20] Cholangiography could be done selectively, and several criteria have been proposed which are also indications for exploration of the CBD. These include the presence of jaundice, or a recent history of jaundice, acute cholangitis or pancreatitis, raised serum bilirubin and alkaline phosphatase, dilated CBD or cystic duct and a palpable stone in the CBD.

Selective operative cholangiography based on these criteria reduces the need for unnecessary cholangiograms, detects almost all duct stones and avoids the problem of unnecessary exploration of CBD.[20]

Management of common causes of cholestastic jaundice

(i) Carcinoma of the pancreas and malignant neoplasms of the Ampulla of Vater, distal common bile duct, pancreatic duct, the descending dueodenum and head of pancreas are mentioned together for several reasons. They develop within a short distance of each other (the periampullar region), they have similar if not indistinguishable features of presentation and when possible are treated by similar operations.[1,24]

Surgical excision remains the only hope for cure and survival in patients with periampullary tumors and of early cancers of the head of pancreas. However most cases of cancers are inoperable at the time of diagnosis.[4]

The standard surgical procedure is pancreaticoduodenectomy (Whipple's procedure) with enbloc excision of the duodenum, head and neck of the pancreas, antrum of the stomach and upper jejunum, gallbladder and CBD with associated lymph nodes.[4] The major morbidity associated with Whipple's procedure is the pacreaticojejunal anastomosis resulting into the pancreatic fistula in up to 20% of cases.[23]

In order to avoid this complication, total pancreatectomy was advocated by Ross in 1954.[23] Another argument put forward for total pancreatectomy is the multifocal nature of the disease.[4,23] However a mortality of 17% - 27% has been reported.[4,14]

Palliative surgical treatment is performed if the tumor is found inoperable at laparotomy, when endoscopic or percutaneous stenting have failed or the facility is not available locally.[13] This could be done by biliary by pass with or without gastrojejunostomy. Cholecystojejunostomy and choledochojejunostomy seems to be especially effective provided the tumor is below the entrance of the cystic duct. Smith[21] reported a randomized prospective controlled trial comparing endoscopic stent insertion and surgical biliary bypass in patients with malignant low bile duct obstruction of two hundreds and four patients. Technical success was achieved in 96 surgical and 98 stented patients, with functional biliary decompression obtained in 92 patients in both groups. In stented patients, there was a lower procedure-related mortality (3% vs 14%) and major complication rate (11% vs 29%). However jaundice occurred in 36 stented patients and 2 surgical patients. Late gastric outlet obstruction occurred in 17% of stented patients and 7% of the surgical group. Despite the early benefits of stenting there was no significant difference in overall survival between the two groups (median survival; surgical 26 weeks; stented 21 weeks) Endoscopic stenting and surgery are effective palliative treatments with the former having fewer early treatment-related complications and the later fewer late complications. In this study we had seven patients who had inoperable tumor of the pancreas who

were managed by cholecystojejunostomy as palliative procedure with a technical success and functional biliary compression achieved in all patients.

Bile Duct Carcinoma (Cholangiocarcinoma)

Bile duct carcinoma has an incidence of 0.3% with a male to female ratio of 3:2[16,20] The highest incidence is in the 6th and 7th decade.[20] Bile duct carcinoma has been divided according to location in the following group.[22]

(1) Proximal third tumors (56%)

(2) Middle third tumors (17%)

(3) Distal third tumors (18%)

(4) Diffuse involvement (7%)

Treatment of patients with carcinoma of the bile duct may be curative or palliative. Tumors may be resected or by passed by anastomosing the dilated proximal duct system to a segment of small intestine.[20] Therapy should be individualised in each patient taking into account the location and extent of the disease, the condition and the operative risk and the expertise available at the institution.[16] In this study there was one patient who was managed by cholecystojejunostomy as a palliative procedure. A mortality of 22% and a mean survival rate of 17.5 months has been reported.

Choledocholithiasis

Common bile duct stones are found in 12% of patients subjected to cholecystectomy.[11] The majority of stones in the

common bile ducts have migrated from the gall bladder and are associated with calculus cholecystitis.[17,20] Secondary calculi in the bile duct usually follow partial biliary obstruction due to a residual calculus, traumatic strictures, sclerosing cholangitis or congenital abnormalities.[18,20]

The treatment for stones in CBD is surgical removal. Pre-operatively, patients should be prepared with Vit K_1 if the prothrombin time is prolonged. Antibiotics should be administered perioperatively.

Removal of stones in the CBD is facilitated by use of balloon-tipped catheters during conventional CBD exploration. Choledochoscopic removal of bile duct stones is one of the recent advances in hepatobiliary surgery.[1,8] While retained stone rate of 15-20% was observed following conventional CBD exploration, with the use of choledochoscopy it is now lower than 2%.[1]

Indication of CBD exploration after surgery for cholelithiasis include

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

Chemical dissolution is one of the other modes which can be used to remove CBD stones and one of the agents is monoctanonin which is a semisynthetic with vegetable oil and is moderately efficient in dissolving cholesterol stones.[1] It is given as an intraductal infusion through a nasobiliary catheter.[1,8,23] Indication for dissolution of stones are as follows: radiolucent stone since it known that ~~radiopaque~~ radiolucent stones are cholesterol in nature and hence dissolve well. Smooth and faceted stones are also composed of cholesterol. A stone with size of less than 10mm. Patients unfit for surgery eg. Cardiac, pulmonary disease and renal disease. Certaintly contraindications includes, pancreatitis, cholecystitis, cholangitis and liver diseases. Stones may recur after successful dissolution therapy. Thirsthe[1] reported recurrence in 3 of 15 patients followed for 48 months. The commonest complication of monoctanonin is diarrhoea and rarely hepatotoxicity has been reported.[1]

CONCLUSION

Obstructive jaundice is a common surgical problem which is challenging both in investigations and management. The success in management depends on early referral of the patient, proper investigations and correct diagnosis at the first consultation. As in this study most patients came to seek medical attention late, and the majority of the patients

underwent only palliative surgical intervention. The availability of ultrasound scanning is an added advantage in investigating the jaundiced patient, but the lack technology and expertise in ERCP and PTC sometimes make diagnosis difficult. In such cases a high early clinical suspicion and USS results should be enough parameters in exploring a fit patient.

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CHEST INJURIES

SUMMARY

From October 1993 to July 1995 60 cases of thoracic trauma (both penetrating and non penetrating) were managed in the surgical firms at Muhimbili Medical Center. Forty six were males and fourteen were females. The male to female ratio was 4:1 (Table 1). The age range was from 11 to 70 years with a mean age of 33 years. Forty four patients had blunt injuries while sixteen patients had penetrating thoracic trauma. Chest pain, dyspnoea, bruises and bleeding chest wounds were the main mode of presentation. Most patients had closed tube thoracostomy either for haemothorax (12 cases), pneumothorax (10 cases) or pneumohaemothorax (5 cases). None needed thoracotomy. Two patients had laparotomy for other associated intraabdominal injuries whereby a ruptured diaphragm was also repaired. There were three deaths reported, one had severe flail chest injuries died and of cardiopulmonary arrest soon after arrival. The second patient had a penetrating chest stab wound, with perforation of diaphragm and stomach. He died 24 hours post operative. The third patient had multiple cut wounds on the head, penetrating stab wounds of the chest and abdomen, with multiple perforations of the small bowels. He also died 12 hours post-operative.

INTRODUCTION

Chest injuries are a common cause of hospital admission at Muhimbili. In a series from UK they are the third commonest cause of hospital admission due to trauma after head and lower limb injuries.[8] In the United States of America, 25% of annual traumatic deaths results from chest trauma.[12]

The chest represents approximately one third of the body surface area to the environment and is therefore vulnerable to injuries.[13] Technological development and availability of more weapons capable of hurling high velocity missiles increasingly contribute to trauma. On the other hand the chest is often the target of assault because it contains vital organs.[4,13] The severity of thoracic injuries especially in blunt injuries may be difficult to determine soon after injury and both clinical and radiological evaluation at the time may be misleadingly normal.[12] Hypoxia and hypercapnia that may develop following thoracic injuries cannot be tolerated for long by most tissues of the body. The severity of the injury is then related to the degree of pathophysiological changes both in the cardiorespiratory system and in the body as whole.[3] For this reason, thoracic injuries should be promptly recognized and treated so as to regain and maintain the normal physiology. A case history is presented in detail and the characteristics of the other patients is presented in the tabular form.

CASE REPORT

M.E. Reg No, 82284, a male patient aged 22 years was admitted, having sustained penetrating chest injury 7 hours prior to presentation. While fighting, he was stabbed twice on the left aspect of the chest and sustained two penetrating chest wounds. There was no history of loss of consciousness. He had no other injuries.

Clinical evaluation revealed a young man in pain and dyspnoeic (respiration rate was 30 per minute). He was afebrile and not pale. His pulse rate was 90 per minute of good volume with a blood pressure of 100/60 mm/Hg. Laterally on the left side of the chest wall in 7th intercostal space there was a stab wound about 4cms in width. The wound was leaking gastric contents with protrusion of omentum. Another wound was anteriorly on the 6th intercostal space about 2cms in width. The wound was leaking air with blood. There was reduced chest expansion with reduced breath sounds on the upper left hemithorax and bowel sound on the lower zone. The apex beat was not palpable, but the jugular venous pressure was not eleveted.

A diagnosis of penetrating chest wounds with perforation of the diaphragm and stomach was made. A chest radiograph frontal and lateral view revealed the following abnormal shadows: elevated diaphragmatic outline of the left hemithorax and a shift of mediastinum to the right. Under an aseptic

technique and local anaesthesia, a chest tube was inserted in the 6th intercostal space within the posterior axillary line and connected to an under-water seal drain. About 400mls of blood mixed with gastric contents was drained immediately. The patient was prepared for an emergency laparotomy and surgical toilet of the chest wounds. Intravenous Normal Saline was infused. Blood for grouping and cross-matching was taken and 4 units of blood was prepared. A nasogastric tube was introduced and a blood stained gastric contents were aspirated. An indwelling catheter was introduced and a clear urine was drained. A signed consent from the patient was obtained. Injection Zinacef 750mg was given intravenously and the patient was taken to theater. Under general anaesthesia an exploratory laparotomy was done. At laparotomy he was found to have a perforation on the greater curvature of the stomach about 6cm long, herniation of stomach, small bowels and a patch of omentum into the chest cavity with leakage of gastric contents in the chest. Herniated organs were reduced, then a thorough peritoneum lavage was done with warm saline. The stomach perforation was refreshened and repaired in two layers using catgut No.2/0. The laceration in the diaphragm was repaired using prolene No. 0 and a drain was left in situ. The abdomen was closed in layers. The chest tube was re-inserted and surgical toilet of the chest wounds was done.

Postoperatively the patient was given intravenous

dextrose in normal saline 4 liters in 24hrs, Injection pethidine 100gm in 6 hourly for 24 hours and Zinacef 750 mg 8 hourly for 3 days were prescribed vital signs were monitored 1/2 hourly. Twelve hours post operative the patients condition deteriorated. He was febrile, dyspnoeic and tachypneic with a respiration rate of 40 per minute. The under water seal drainage had minimal drainage. His pulse rate was 60 per minute with low volume with a blood pressure of 80/50 mm/Hg. Nasogastric aspiration was 500mls in 24 hours. The abdomen was soft and not distended. Urgent chest x-ray revealed only minimal hemothorax on the left hemithorax with scattered patchy opacities of the left lung.

An diagnous of cardiac tamponade was made. The patient was given oxygen by mask. Intravenous normal saline. Hydrocortisone 200mg 6 hourly was given. Inspite of above resuscitative measures the patient died 24 hours post operative. A postmortem was done and revealed a body of a male African adult with two stab wounds on the left anterolateral aspect of the chest. One wound was anterior in the 6th intercostal space about 2cms in width and 12 cms deep and penetrating the heart. Another wound was lateral in the 7th intercostal space about 4 cms in with penetrating the pleura and diaphragm and penetrating into the stomach. Characteristics of other patients are summarized in a tabular form.

TABULAR SUMMARY

Table 1: Sex and age distribution of the studied cases

Age in years	Male	Female	Total
11 - 20	5	-	5
21 - 30	23	7	30
31 - 40	13	5	18
41 - 50	3	2	5
51 - 60	1	0	1
61 - 70	1	-	1
Total	46	14	60

NB: Majority of patients were young adults in age group 21 to 40 years (78%). Male/Female ratio was 4:1.

Table II: Nature of Injuries of thoracic trauma.

Nature of Injury	Number of patients
Blunt injury:	
Road traffic accidents	35
Assaults	9
Penetrating injury	
Assaults	15
Gun shot	1
Total number of patients	60

Note: Blunt injuries were more commonner (64%), than penetrating injuries (36%) and a majority of these were due to road traffic accidents.

Table III: Clinical manifestations of the thoracic trauma cases studied

Clinical manifestation	Frequency in patients
Bruises with chest pain	20
Fracture ribs	15
Hemothorax	12
Pneumothorax	10
Hemopneumothorax	5
Lung contusion	5
Subcutaneous empyema	4
Flail chest	1
Diaphragmatic rupture	1

Note: Some patients had more than one type of manifestations.

Table IV: Associated injuries

Nature of injury	Number of patients
Splenic laceration	2
Multiple perforation of stomach and small bowel	2
Head injury	1
Total	5

Note: One patient sustained multiple perforation of stomach bowel, and multiple scalp cut wounds died. A second patient with diaphragmatic perforation, and stomach perforation also died in the post operative period.

DISCUSSION

Thoracic trauma is one of the major causes of admission among trauma victims in this hospital[13]. It has also been reported as the third commonest cause of hospital admission due to trauma in a series from U.K[6,18] and accounted for 25% of annual civilian traumatic deaths in the United States.[12] Thoracic trauma is frequently fatal due to injuries to the cage, heart, great vessels and lungs. A survey of literature shows that the thoracic oesophagus and thoracic duct are rarely involved except in missile injuries.[4,12,13,15] The rise in automobiles accidents, weapons capable of hurling high velocity missiles, and abuse of alcohol contribute to the increased frequency of chest injuries as a cause of hospital admission.[8,12,13,14] However, the actual magnitude of thoracic injuries as an entity has not been well documented in Tanzania. In this study, it accounted for nearly 4% of all surgical admissions in the department of surgery.

The distinction between blunt trauma and penetrating chest injuries is useful since it has important diagnostic and therapeutic implications. Most blunt chest injuries result from road traffic accidents[9,13] but could also result from industrial and domestic accidents such as falls, mining accidents or direct blows during assault. Blunt trauma tends to be associated with other multiple injuries such as head injuries, abdominal trauma, pelvic fractures or fracture of

long bones.

Penetrating injuries on the other hand, tend to produce more localized and sometimes predictable injuries, but occasionally lack of precise information on the nature of the weapon used, or on the circumstances and posture of the patients at the time of injury may make it difficult to precisely predict likely damage caused.[8]

In most series[3,5,8] blunt thoracic injuries are more common than penetrating thoracic injuries. In this study forty four patients sustained blunt thoracic injuries, while sixteen patients had penetrating thoracic injuries. Thoracic injuries are associated with high mortality,[11,14] which can be prevented by simple life saving procedures, as airway control and tube thoracostomy.[12,13] In this study deaths occurred, in patients who had associated multiple organ injuries.

PATHOPHYSIOLOGY

Chest injuries have been classified by Reid and Barrd[11] into three classes based on one's ability to cough effectively and maintain satisfactory ventilation.

Class 1: Arterial Po_2 and Pco_2 normal or reduced.

Class 2: Arterial Po_2 and Pco_2 reduced does not cough effectively with impaired level of consciousness.

Class 3: Evidence of respiratory failure with low PH and Po_2 and high Pco_2, evident by respiratory distress and progressive cynosis.

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

Naclerio[9] suggested a "regional" physiological approach to classification. In his classification there are three groups:

(a) Chest wall injuries. These include bony cage and soft tissue injuries, such injuries may result in

(1) Subcutaneous emphysema.

(2) Flail chest

(3) Open pneumothorax

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

(1) Closed pneumothorax

(2) Hemothorax or hemopneumothorax

(3) Lung parenchyma injuries such as lung contusion, lacerations, or aspiration pneumonitis.

(c) Mediastinal injuries. These may result in

(1) Mediastinal Emphysema

(2) Cardiac tamponade

(3) Compression atelectasis

(4) Peri and intracardial laceration etc.

The severity of chest injuries is influenced by various factors,[11] the following being important as they affect the outcome.

(a) Age. Both extremes of age fair badly.[11] The aged patients have a more rigid brittle rib chest cage rendering them more prone to multiple fractures, they also have low vital capacities, reduced lung compliance, impaired cardiac function and increased incidence of thromboembolism. The very young patient have a highly elastic rib-cage and may develop serious intrathoracic damage without rib fractures.[8,9]

(b) Patients with pre-existing lung disease such as chronic bronchitis, emphysema and bronchial asthma or patients with pulmonary fibrosis. In general, patients with impaired respiratory reserve have a poor outcome.[8,9]

(c) Patients with preexisting cardiac disease, as any type of chest injury will in one way or another affect the cardiopulmonary status.

(d) Obese patients

(e) Patients with scoliosis or kyphosis.

(f) Patients who had previous thoracic surgery, such as pneumonectomy or thoracoplasty.

The thorax is responsible for vital delivery of oxygenated blood to metabolically active tissues. Three pathologic consequences of thoracic injury either alone

or in concert, are responsible for inadequate oxygen delivery.

(i) Hypoxemia can follow such divergent etiologies as chest pain, airway obstruction, pneumothorax, flail chest, pulmonary contusion, tracheobronchial injury and diaphragm rupture. Each of these injuries limits the physiologic function of air exchange. Trauma to the parietal pleura, bony structures, and especially, the intercostal nerves is very painful. This pain interferes with ventilatory effort; produces shallow breathing, tachypnea, and hypoventilation, and results in relative increase in dead space, decreased cough effectiveness, and retained secretions. These abnormalities may progress to hypercapnia, hypoxia, and later infection. This chain of events worsens any pulmonary contusion and ventilatory deficiencies and may end in frank adult respiratory distress syndrome. Therefore, control of pain is a major aim in the therapy of chest wall trauma[5]

2. Hypovolemia from intrathoracic hemorrhage is second only to rib fractures as a sequel of thoracic trauma. As much as 40% of the circulating blood can accumulate in a hemothorax, and thoracic bleeding most frequently occurs from injuries to the lung parenchyma or intercostal vessels.[3,9,11]

3. Cardiac pump failure may follow both blunt or penetrating thoracic injuries. Cardiac contusion is estimated to occur in 20% of patients with major thoracic trauma, although many cardiac contusions go unrecognized.[4] Penetrating injuries following direct cardiac injury and pericardial tamponade may rapidly compress cardiac function.

Initial evaluation and management

The evaluation and management of a patient with chest injuries go hand in hand, because some treatment modalities are life saving and need to be instituted immediately. Therefore the initial care of the injured patient necessitates two assumptions. The first is that the patient may have more than one injury, the second is the obvious injury is not necessarily the most important one.[4,8,12] The most import steps are:-

1. Establishment and maintenance of the adequate, clear airway. This may need just proper positioning of the head and neck, or may need endotracheal intubation or even tracheostomy where necessary. Removal of foreign bodies in the oropharynx should be done. Aspiration by nasotracheal,bronchoscopic or through tracheostomy. Oxygen and mechanical ventilation may be necessary.
2. Cardiovascular support in terms of intravenous fluid with blood transfusion where necessary. Cardiac resuscitation

with cardiac message should be instituted immediately if there is cardio-pulmonary arrest.

3. In case of massive hemothorax or pneumothorax with respiratory distress, tube thoracostomy should be done immediately without waiting for x-ray films.

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

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

6. Indications for thoracotomy are usually from observation made during the period of resuscitation and physiological stabilization. Operation is most urgent for patients with massive uncontrollable hemorrhage or massive air leak and those with cardiac tamponade. In general surgical intervention is necessary where blood loss through chest tube exceeds 500ml/hr after 3 hours or more than 180ml/hr after 6 hours.[5,8] In cases of considerable blood loss from the chest tube, the use of autotransfusion should be considered.

Recognition and management of specific injuries

(a) **Rib fractures**

Fracture of the ribs is the most common thoracic injury.[13] The fracture tend to occur at the point of maximum convexity that is the lateral chest wall, and they reflect the large amount of energy that has been imported on the chest wall,[5,8,12] With simple fractures, pain on inspiration is the principal symptom. Localized pain, tenderness, and occasionally crepitus confirm the diagnosis. Fractures of the first rib carry a special significance, since they usually result from severe trauma. This injury should alert the clinician to possible significant injury within the chest or abdomen and dictates a thorough evaluation of the thoracic aorta, subclavian vessels, the tracheobronchial tree and the neurovascular structure to the upper extremities.[1,14]

The pain produced by the fracture causes the patient to splint the chest causing hypoventilation and accumulation of secretions. These secretions, being rich in proteins, rapidly becomes infected and pneumonitis results.[13] A chest film should be obtained to exclude other intrathoracic injuries such as hemothorax or hemopneumothorax, and not necessarily to identify rib fractures. The objective of treatment is to relieve pain sufficiently to restore normal ventilation and the

ability to cough out tracheobronchial secretions. This is best accomplished with narcotics in small amounts, intercostal nerve blocks, and muscle relaxants are usually adequate.[8,11,13]

In case of narcosis the dosage should be adequate to relieve pain but not sufficient to depress respiration or inhibit cough. Hospital admission for pain relief, cough assistance, and endotracheal suction may be necessary for several days, particularly in elderly patients.[9,13] Rib belts and adhesive taping, although once popular, should be avoided because the limitation in motion increases the incidence of retained secretions and atelectasis.[14] In this study 19 patients had fractured ribs, all of whom were managed conservatively improved well, while 5 patients had associated hemothorax or pneumothorax and were managed by tube thoracostomy.

(b) **Flail Chest**

This condition is an unstable chest wall that has a paradoxical motion during respiratory cycle. It is common major injury to the chest wall, occurring in 10 to 20% of trauma admissions.[13] Mortality ranges as high as 50% in some series, higher in patients over 60 years of age.[1] Flail chest usually results from direct impacts, or severe compression, such as being run over by a vehicle. Because of the pliable ribs, the condition rarely occurs

in children. Flailing may be located anteriorly, laterally or posteriorly and it always associated with multiple fractures or dislocations. Therefore it occurs where three or more ribs fracture at two points, or when ribs fracture on both sides of the sternum, the result is a flail segment which moves paradoxically with respiration. This paradox may not be apparent initially, due to muscle spasm.[1] Sometimes impaction of fracture fragments may temporarily stabilize the flail segment, but as the muscle spasm wear off and the segment loosens the flail becomes apparent.[8,9,10] The problems associated with the flail chest includes the following according to Naclerio.[9]

(a) Paradoxical respiration results in useless to and fro movement of air across the carina between the two lungs. This is called the pendiluft phenomenon. Malomey et al[7] challenge this concept. They argue that the respiratory disturbance resulting from flail chest is due to a sharp reduction in the vital capacity, and the inability to drop the intrathoracic pressure to adequate negative level. This concept is also supported by Glinz[17] who says that the forced vital capacity drops to 40% of normal, and it continues to drop in the two days after injury, resulting in significant respiratory

insufficiency.

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

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

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

The management is directed toward maintaining adequate ventilation, decreasing progressive pulmonary damage, and preventing complications.

Therapy may be divided into three levels.

(1) Skeletal splinting by weight, pins and plate.

(2) Careful attention to the pathophysiology of respiration, control of pain, and infection; and

(3) Internal splinting by positive pressure ventilation through an endotracheal tube. Therefore the management of such patients include the followings.

(a) Stabilizing the chest by sandbags or placing the patient on the affected side.

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

(c) Naso-tracheal suction to clear the secretions from the airway.

(d) In severe flail chest endotracheal intubation with intermittent positive pressure ventilation may be necessary till the chest wall stabilizes. This may take 7 to 10 days, and sometimes even up to three weeks.[13] When prolonged orotracheal intubation is needed tracheostomy may be necessary, but is often discouraged since the patient then needs intensive care which is poorly tolerated by old patients or those with chronic bronchitis.[4]

(e) Interest is resurging[17] with internal fixation of the unstable segments. This gets rid of the need for prolonged periods of ventilation, avoids the need for tracheostomy and prevents permanent chest deformities. In addition to providing mechanical stability, stabilization provides pain relief. Internal fixation can be achieved by Kirschner wires, small plates or metal chips.

(f) Epidural analgesia has become one of the cornerstones in the management of flail chest.[17] Interestingly it has frequently been observed that paradoxical movements of the flail segment is diminished or may even disappear completely after epidural analgesia.

(g) Broad spectrum antibiotics are indicated to prevent lungs infection.

(h) After the acute phase of the injury has passed, thoracic and shoulder physiotherapy is indicated. This helps to decrease some of the deformity and restricted pulmonary function, which in various degrees, are a result of flail chest.

In our series we had one patient with flail chest, unfortunately he died of cardiopulmonary arrest while being resuscitated.

(c) **Pneumothorax**

(a) Open pneumothorax

This condition results where a segment of the chest wall has been destroyed such that negative intrathoracic pressure sucks in air directly through chest wall rather than through the trachea and bronchi,[10] such wounds have also been termed "sucking chest wound". When the chest wall wound has a diameter greater than the laryngeal diameter the result is complete lung collapse with marked mediastinal shift with each respiratory effect.[9] The management includes prompt closure of the defect renting the pleural cavity with tube thoracostomy to prevent possible tension pneumothorax.

(b) **Closed pneumothorax**

This results from any direct or indirect communication of bronchus, bronchioles or alveolus with pleural cavity resulting in collapse of the ipsilateral lung.[9] If there is a valvular mechanisms preventing air escape then tension results.[9,10] Effects of tension pneumothorax include.

a) Collapse of the ipsilateral lung.

b) Mediastinal shift leading to angulation and collapse of the great veins. This results in serious interference with cardiac filling.[9] In simple pneumothorax tube thoracostomy needs to be done after radiological documentation, but in tension pneumothorax, tube thoracostomy needs to the done urgently without waiting for chest x-rays. Tension pneumothorax will be recognized by the following clinical features:

1. Progressive cyanosis
2. Respiratory distress
3. Trachea deviation to opposite side.
4. Increased resonance note on the affected side.

5. Absent breath sounds over the affected side.

When these features are seen, then the insertion of a chest tube is an emergency. If this cannot be done immediately then a large bore needle (14 to 16 gauge) can be introduced in the chest cavity in the second, third or fourth intercostal space in the middle clavicular line.[9] This creates a vent for air to escape from the pleural cavity and decreases pleural tension. It can be connected to underwater seal, or if an emergency it can be attached to a flutter valve made from a finger of a glove.[9] In this study 10 patients with pneumothorax, were managed by tube thoracostomy.

(d) **Hemothorax and Hemopneumothorax**

Hemothorax may be secondary to a tear in the lung, and intercostal artery or internal mammary artery but since the pulmonary circulation is a low pressure system, bleeding from the lung parenchyma usually stops with re-expansion, of the lung. Hence bleeding causing hemothorax most commonly follows injury to the chest wall or to internal

mammary arteries.[8,10] The effects of hemothorax are similar to those of pneumothorax, but in addition there is depletion of blood volume.[9] Hemothorax may co-exist with pneumothorax. The presence of hemopneumothorax, doubles the incidence of infection leading to empyema thoracis, and the most common organism in one series was staphylococcus aureus.[2] About 300-500mls of blood may be present in a pleural space before visualization on an erect frontal view chest X-ray and if a supine radiograph is taken up to 1000mls of blood in the pleural space may be over looked.[8,10] A decubitus film could be taken in a very serious patient.[18] Massive hemothorax may be due to injuries of the great vessels of the heart. In management of hemothorax or hemopneumothorax the initial mode of treatment is a thoracic tube insertion in the 6th, or 7th intercostal space in the posterior axillary line, or middle axillary line, with underwater seal drainage (closed tube thoracostomy). This drains both blood and air, and is usually effective in 85% of the cases.[10] Sometimes two tubes can be inserted.[9] The initial volume of blood should be noted

and subsequently hourly record of blood being drained. If the initial blood loss is more than 200-300mls, and shock persists despite blood replacement then thoracotomy is indicated, as there is usually a correctable lesion.[12,13,17]

In pneumothorax or hemopneumothorax a closed tube thoracostomy should always be done prior to administration of a general anaesthetic for other reasons or other injuries. This is to avoid development tension pneumothorax during ventilation! Hemothorax may complicate into clotted hemothorax or later form empyema thoracis, which will need thoracotomy.[9] In this study we had 5 cases of hemopneumothorax all of whom were managed by closed tube thoracostomy. Two of them developed empyema thoracis, and were managed by early decortication.

Lung contusion

This is defined as blunt injury to the lung parenchyma resulting into oedema and intestinal hemorrhage. Lung contusion usually results from decelerating or crushing injuries. It may also occur in most patients with flail chest (49% of all flail chest patients in one series had lung

contusion)[12] and simple rib fractures, but it may be an isolated injury without any bone cage injury especially in children.

As a result of lung contusion, fluid and blood from ruptured vessels enter the alveoli interstitial spaces and bronchi and form a localized air way obstruction. The extravasation of fluid and blood provides excellent culture medium in debilitated patients, with 50 to 70% developing pneumonia. Of these, 30% will go on to develop pulmonary abscess, empyema or both.[3] On the other hand, as localized airway obstruction progresses, pulmonary compliance decreases and ventilation becomes more difficult. Dyspnoea leads increased carbon deoxide, and increased work of breathing, which results in hypoxemia, or frank adult respiratory distress syndrome[5] Pulmonary contusion may be confused with adult respiratory syndrome (ARDs), or may be even associated with it.[1,8,10,13]

Physical examination is usually unrewarding. In contrast, radiographic examination eventually demonstrates, patchy to lobar opacities.

The management of such patients involves clearing of secretions by nasotracheal suction, coughing exercises, avoidance of fluid overload, administration of diuretics, and ventilatory support. Positive and expiratory pressure (PEEP) ventilation may be necessary in severe cases. Prophylactic administration of antibiotics have also been advocated. There

is a controversy as to the severity of interstitial oedema and hemorrhage may be reduced by early administration of methyprednisolone 500mg 6 hourly.[10] In this study we had 5 patients with pulmonary contusion. They were all managed by antibiotics, anti inflammatory agents, coughing exercises and close observation.

Diaphragmatic Injuries

Traumatic rupture of the diaphragm is a relatively rare finding in blunt thoracic trauma. In some series it has been observed to occur in 4 to 5% of patients presenting with thoracic trauma.[6] The mechanisms of injury is through a crushing force producing a sudden increase in the intrathoracic or intra abdominal pressure against the fixed diaphragm.[6,16] Penetrating lesions from stabbing or gunshot wounds occurring as high as 4th interstal space can result in diaphragmatic disruption depending on the phase of respiratory at the time of injury.[15,16] Diaphragmatic rupture usually tends to occur in the weakest parts of the diaphragm along the embryonic fusion line posterolaterally.[12] The most common presenting symptoms include shortness of breath and chest pain. This is due to compression of the lung and mediastinum from the herniated intra abdominal contents. Physical findings include decreased breath sounds on the side of herniation, a contralateral shift of the mediastinum, a marked scaphoid abdomen, and the presence of bowel sounds in the thorax.[6,16]

The chest x-ray usually confirms the suspected diagnosis, demonstrating abnormal shadows, elevated diaphragmatic outline, and a shift of the mediastinum. Management is by immediate operative repair.[6,13,16] The choice of operative approach is somewhat controversial, if the diagnosis is made early, and abdominal approach is usually indicated to exclude associated injuries, which is the case in 75% of patients.[6,12] If the diagnosis has been substantially delayed, where adhesions of herniated contents are suspected, the trans thoracic approach will provide the best exposure and intra abdominal injuries, may be repaired through the diaphragmatic rupture.[13,16] In this study one patient had penetrating chest injury on the left side, with perforation of the diaphragm, and stomach, with hemopneumothorax. The patient was managed by tube thoracostomy, laparotomy with repair of diaphragm and stomach and surgical toilet of the chest wounds. Unfortunately the patient died of cardiac tamponade 24 hrs post operative.

CONCLUSION AND RECOMMENDATION

Chest injuries are a common cause of hospital admission at Muhimbili Medical Center. The increase in number of motor vehicles on the roads and alcohol abuse by drivers, are likely to increase accidents and result in more severely injured patients.

Therefore there is a need that measures be taken to reduce road traffic accidents, which may include:-

1. Legislating strict penalties for drinking and driving
2. Limitation of vehicle speed.
3. Use of vehicle design safety feature.
4. Mandatory seat-belt usage law.

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MANAGEMENT OF THORACIC EMPYEMA

SUMMARY

Between January, 1994 and July, 1995, 35 cases of thoracic empyema were managed by the thoracic surgical unit of Muhimbili Medical Center. There were 29 males and 6 females giving a male to female ratio of about 5:1. The age range was from 7 years to 70 years with a mean age of 32 years.

Pulmonary tuberculosis was the commonest cause of empyema thoracis, accounting for 18 cases. Thirteen out of eighteen patients were ELISA positive for HIV. Five out of 18 cases had thoracic empyema with bronchopleural fistula. other etiological factors were as follows: Chest trauma 10 cases, pneumonia 3 cases, breast cancer 3 cases, and postpneumonectomy space infection one case.

The initial modality of treatment was closed tube thoracostomy for all patients and it was successful in 20 patients. Eight patients were managed by open chest drainage, five patients were managed by decortication and one patient with post pneumonectomy space infection was initially managed by tube thoracostomy, then re-thoracotomy and myoplasty and finally by open chest drainage after failure of the two previous procedures. Hospital stay ranged from one month to nine months with a mode of around 4 months. Two patients died due to fulminating and overwhelming bilateral pulmonary tuberculosis with associated HIV infection.

INTRODUCTION

Thoracic empyema is a common cause of hospital admission at Muhimbili Medical Center (MMC) in both medical and surgical wards. In 1994 and 1995 thoracic empyema demanded more surgical attention than any other thoracic problem at Muhimbili Medical Centre.[14] In Nigeria thoracic empyema accounted for 30% of thoracic surgical cases over a period of 6 years.[1]

Empyema continues to be an important and troublesome problem in Tanzania because many people with pulmonary tuberculosis, pneumonia, chest trauma, lung abscess, liver abscess etc are inadequately managed in the first instance resulting into thoracic empyema.[14,16] In addition it continues to be an important problem in Africa, largely because many people do not have easy access to medical care and therefore are treated late.[1,14] Thoracic empyema is a very distressing and disabling chest condition and cripples the chest wall and lung function.[25] It is important therefore for doctors to be conversant with proper management of this condition so as to reduce the morbidity and mortality and to bring the patient back to a productive life as early as possible. For these reasons, a one and half year experience in its management is hereby recorded.

Case presentation

A.M. (Reg. No. 816292), a 30 years old male referred to Muhimbili Medical Center was admitted to the thoracic unit in January 1995, complaining of cough, fever, night sweat for 5 months, left side chest pain and breathlessness for one month. He had no history of tuberculosis contact or chest trauma. He was referred as a diagnosed patient with pulmonary tuberculosis confirmed bacteriologically and radiologically.. This was complicated by empyema and a bronchopleural fistula. He had been on antituberculosis treatment for 3 months.

General examination revealed a young man wasted, ill looking with temperature of 38°c, dyspnoeic but not cyanosed. Cervical, axillary and inguinal lymph nodes were not palpable. He had no finger clubbing. The blood pressure was 110/70 mm/Hg with a pulse rate of 70 per minute of good volume. He had a chest tube inserted in the left hemithorax connected to the underwater seal drainage with an obvious bronchopleural fistula on the same side evident by presence of gas bubbles in the under water seal drainage bottle. He had limitation of chest expansion on the same side. His trachea was shifted to the right. Apex beat was not felt. There was stony dullness over the lower zone of the left hemithorax, with hyperresonance over the upper zone of the same side. There was diminished vocal fremitus and resonance, and absent breath sounds on the same side. Other systemic examinations were

found to be normal.

The diagnosis was pulmonary tuberculosis complicated with empyema thoracis and bronchopleural fistula. Chest x-ray taken in postero-anterior and lateral views revealed the following.

1. Shift of the mediastinum to the right
2. Left hydropneumothorax
3. Left lung collapse.

Insertion of the chest tube was done under aseptic conditions and local anaesthesia in the 6th intercostal space, and the tube was connected to an underwater seal drainage. Massive air and thick purulent fluid was noted. Before insertion of the tube, aspirates were taken for sugar, protein, Gram stain, Z.N. stain and culture. On the second day a control x-ray was taken and revealed remarkable reduced hydropneumothorax, but with no lung expansion. The patient was also managed with a regime of antituberculosis drugs. (His body weight was 50kg).

1. Streptomycin 1gm daily for 60 days
2. Rifampin 600mg daily for 90 days
3. Pyrazinamide 1600mg daily for 90 days
4. Ethambutol 800mg daily for 90 days

Thereafter to continue with isoniazid 300mg and ethambutol 800mg daily for 5 months.

One month later another control x-ray was taken and revealed remarkable reduced pleural effusion but with no lung

expansion. The bronchopleural fistula healed spontaneously after two months of treatment, whereby another control x-ray was taken and revealed poor lung expansion with a persistence of the cavity. The patient was planned for open drainage. The following are the results of the investigations done.

- Sputum culture was sterile and aspirate taken for culture was sterile. A full blood picture was normal apart from a high ESR of 58 Westergreen in the first hour, Serum creatinine was 80 micromoles per liter the ElISA test was negative

He subsequently gained weight and when his condition was stable he was operated upon in July 1995 about 6 months after admission. Consent for operation was obtained from the patient. In theater under general anaesthesia, and the patient in right lateral position the skin was cleaned and draped. Modified Elosser skin incision was done, and muscle incised after removal of scar tissue. After opening the pleura, a big cavity with no lung tissue was found. The 5th, 6th and 7th ribs segments were resected and removed. Parietal tissue was taken for histological analysis. About 500ml of pus was sucked. The procedure was completed by suturing skin flaps to the parietal tissue. A window approximatelly 10 x 10 cm in diameter. The postoperative period was uneventful. Two weeks post-operative, the patient was discharged on isoniazid 300mg and ethambutol 500mg for 2 months and advised to continue with

follow-up at the regional hospital. The characteristics of other patients is summarized in a tabular form.

Table 1: Sex, Age Relationship among thoracic empyema cases

Age group (yrs)	No of cases		
	Male	Female	Total
1 - 10	2	-	2
11 - 20	1	-	1
21 - 30	10	3	13
31 - 40	11	3	13
41 - 50	3	-	3
51 - 60	1	-	1
61 - 70	1	-	1
Total	29	6	35

Note: Male v/s female ratio 5:1. Mean age was 32 years.

Table 2: Underlying causes among thoracic empyema patients (n = 35)

Underlying cause		No of patients
Pulmonary Tuberculosis		
with BPF	5	
without BPF	13	18
Chest trauma		10
Pneumonia		3
Breast cancer		3
Post pneumonectomy space infection		1
Total		35

NB: BPF = Bronchopleural fistula is defined as communication between the bronchial tree and pleural cavity.
Pulmonary tuberculosis was the commonest cause of empyema thoracis.

DISCUSSION

Thoracic empyema is a collection of purulent fluid in the pleural space.[3] Empyema of the thorax is a disease which has for a long period ~~plagued~~ pledged mankind, since the time of Hippocrates, who first wrote about this problem.[8] It usually occurs secondary to a primary process elsewhere and is not a primary pleural infection. It may be acute or chronic, but a more precise classification is that of the American Thoracic Society which classifies thoracic empyema into the exudative stage, fibrinopurulent stage and organizing stage.[2] This is a useful classification as it is descriptive and has important therapeutic implications.[25]

Prior to introduction of antibiotics, it was a frequent, and often a difficult disease to manage.[14,17,21] In the early years empyema complicated pneumonia in 5 to 10 percent of the cases.[21] In the Western World it now occurs in about 1 percent of cases with pneumonia.[2] In Canada an incidence of 0.5 per 1000 admissions has been reported.[6]

However, the incidence in developing countries is still high. In Nigeria quoted figures indicate that empyema thoracis representes 30 percent of thoracic surgical cases.[1] Whereas in Maputo 54.4 percent of adult patients requiring chest drainage had empyema.[3] In Tanzania between July, 1986 to June 1991, Lema[14,16] et al had managed 147 cases in the Thoracic Unit of Muhimbili Medical Center. Empyema remains a common thoracic

surgical condition seen in this center.

The disease affects all age groups and both sexes, however, most of the patients are young, Lema[16] observed a high frequency of his cases between 21-40 years constituting 70%, with males being affected more frequently than females in a ratio of about 5 to 1 for reasons difficult to explain. This study confirms further the previous reports of males being affected more frequently than females in a ratio of 5 to 1. It also confirms the high incidence of thoracic empyema in the prime age of 21 to 40 years with mean age of 32 years. Pulmonary tuberculosis as an underlying cause of thoracic empyema accounted for 18 cases. The presence of bronchopleural fistula in those with underlying tuberculosis, further complicated the management of these cases.

Thoracic empyema is common in HIV disease, testified by the finding of 13 seropositive cases out of 18 cases tested. This strong association between HIV infection and pulmonary tuberculosis in our environment predisposes to thoracic empyema occurrence. Other etiological factors in this study were as follows: chest trauma 10 cases, pneumonia 3 cases, breast cancer 3 cases and postpneumonectomy space infection one case.

Close tube thoracostomy as the main modality of treatment gave satisfactory results in 20 out of 35 cases. The reasons for a low successful rate include[16]

1. Thick pus and fibrin clots causing tube blockage often necessitating tube re-insertion.
2. Often the empyema has multiple loculations
3. Extensive lung parenchyma destruction by pulmonary tuberculosis making it not expansible.
4. Presence of a bronchopleural fistula

Rib resection and open chest drainage was found to be a suitable procedure for 8 cases with failure of closed tube thoracostomy, decortication was done in 6 cases and one patient with post pneumonectomy space infection was managed by rethoracotomy and myoplasty and finally by open chest drainage.

Pathogenesis and Bacteriology

The bacteriology of empyema has been changing, being predominantly pneumococcal in the pre-antibiotic era, staphylococcal in the antibiotic era, and predominance of gram negative organisms with fungi since the introduction of penicillinase resistant penicillin.[2,17,25] No single organism predominates in empyema[18] but anaerobic organisms are being isolated more frequently.

Tuberculosis empyema though described to be uncommon in the Western World[3], still remains a significant problem in developing countries.[1,12,14] In tuberculosis empyema the fluid is thin and serous and tuberculous bacilli may be difficult to isolate. Lema et al[16] analyzing bacterial isolates from 72

cultures found that about 42 of the cultures were sterile while 20 cultures were positive for tuberculosis. However, anaerobic cultures were not done. Most of the positive growth had mixed infections.

PATHOGENESIS

The course of development of empyema thoracic has been classified by the American Thoracic Society into three phases based on its natural history, and this scheme is useful in planning therapy.[2]

These are:-

a) Exudative stage (early phase) This characterized by increased permeability of visceral pleura resulting in formation of a "sterile" exudate. The visceral pleura and the underlying lung are still able to expand.

b) Fibrinopurulent stage (transitional stage)

It is a progression of the untreated empyema characterized by a thicker, more turbid fluid with Protein and polymorphs and appearance of fibrin on the pleural surface. But frank purulence must develop before an effusion is referred to as an empyema.[5] The empyema may lie free in the pleural surface. The lung begins to be trapped by restrictive fibrin peel.

c) **Chronic stage (organization stage)**

The fibrin peel is organized with an ingrowth of capillaries and fibroblasts from both visceral and parietal surfaces producing a layer of fibrous tissue that encases the lung (frozen chest). It frequently follows inadequate treatment of the acute phase. Less commonly it results from bronchopleural fistula.

The empyema may lie free in the pleural space or be encapsulated by fibrinous pleural adhesions which arise as a local defence mechanism often encouraged by antibiotic therapy. Continued accumulation of pus compresses the lung with a shift of the mediastinum to the opposite side. Fluid may be absorbed making the pus more viscous. Fibrin is continuously deposited on the pleura surfaces producing easy stripping during surgical decortication. The transition of an acute to chronic empyema is arbitrary, periods of between 6 and 12 weeks having been suggested.[5] The continued formation and fibrosis of the pleural progressively restricts the wall and diaphragmatic movement, eventually producing shrunken, flattened, immobile hemithorax with over lapping ribs and scoliosis of the affected side. Adhesions may loculate the viscous pus. At this stage it may be difficult to aspirate pus even for diagnostic purposes. There may be considerable subpleural alveolar destruction and fibrosis leading to permanent functional impairment, even if lung re-expansion is

reaccumulation of fluid occurs or if infection and toxicity are difficult to control.[5,21,22,25] This method is useful in a toxic patient since it allows stabilization of the patient before definitive therapy is undertaken. When intercostal tube is elected, negative pressure may have to be applied to hasten pulmonary expansion especially in the presence of a bronchopleural fistula.[22] The tube should be of a generous caliber commensurate with the material being evacuated.

The chest tube should be placed in the most dependent part of the empyema pocket, with care to avoid perforation of the diaphragm. The last hole should always be along the radiopaque line and about 6cm from the chest wall. A radiological is vital to confirm that the holes are in the pleural cavity and to visualize the position of the tube. More than one tube may be necessary.[22] The tube should be connected to an underwater-seal system. The patient is then frequently reassessed clinically and radiologically for signs of lung expansion. With chest tube drainage, technical difficulties and failures have occured particularly where there is multiple or inaccessible collections. In this study 20 cases were successfully managed by this method.

3. **Thoracoscopy and irrigation**

This is a method which is gaining popularity at present.[4,6,15] In this procedure thoracoscopy is done under general anaesthesia. Using the thoracoscope, loculi are broken down under direct vision. The cavity is then irrigated with an antibiotic solution (eg Noxythrolin or 0.25% Neomycin solution) in normal saline through the thoracostomy tube which is inserted after removal of the thoracoscope until the effluent is clear. The effluent fluid is taken for culture and the irrigation is done twice daily until the effluent culture is sterile.[15] While the procedure may be useful in the management of early thoracic empyema, it may not be applicable in chronic empyema with a thick cortex over an extensively destroyed lung and bronchopleural fistula.[7] The daily irrigation and its requirements of large volumes of irrigation fluid may limit its feasibility. In this study this procedure was not used in any of our patients.

4. **Decortication**

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

The aim of decortication is to remove the fibrous wall of the empyema cavity both from the lung surface, the chest wall and the diaphragm.[23] It is especially useful in post-traumatic thoracic empyema in sub-acute stage, but it is contraindicated in old and frail patients and also in post-pneumonectomy empyema. Beneficial results of decortication as summarized by Sensery,[24] include the following:-

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

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

c) Avoidance of prolonged tube drainage.

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

e) Thoracic contractures and scoliosis are avoided.

Decortication has been found to be effective in 80-100% of cases and it shortens hospitalization time.[13] In this study 6 patients out of 35 patients were managed by above method. All patients did well.

5. **Open chest drainage**

In 1935 Eloeser[8] designed a method for treatment of tuberculosis thoracic empyema by creating a "window" on the chest to drain pus. He made a flap of skin 2x2 inches on the skin a rib higher than the presumed "bottom" of

the empyema, removed a segment of the underlying rib, drained the pus and then he sutured the tip of pleura to the skin edges. Various modifications of the Eloeser window are being performed. The modifications are mainly in the size of the window and the number of ribs resected. Takaro et al[25] recommended a window at least 5 cm long with one or two rib resections. Oluwole[1] recommends a linear incision 8-10cm long with excision of 3 ribs. Sampson,[20] makes a vertical incision on the skin. In principle the opening should be large enough to allow incision of the thickened pleura, complete evaluation of empyema contents, and breakdown of all loculi.[13,25]

The procedure is completed by stitching the skin edges to the thickened pleura. The procedure is done under general anaesthesia. The cavity gradually granulates and heals by scarring. In this study 8 of 35 patients were managed by open chest drainage and did well.

6. **Thoracoplasty**

This is a procedure where part of the chest wall is allowed to collapse by resection of segments of ribs, depending on the extent of empyema.[10,25] The aim is to reduce the pleural space.[19] Although the procedure produces deformity and reduces lung function, it may be the only option available to control the problem of dead space in large empyema cavities with or without

bronchopleural fistulae.[9,14,20,25] McCord[19] et al found that among their 65 patients who had thoracoplasty deformity was minimal and movements were almost normal. In this study none of our patients were treated by thoracoplasty.

MANAGEMENT OF POST PNEUMONECTOMY EMPYEMA

One of the most serious complication following pneumonectomy is the development of a bronchopleural fistula (BPF). This predisposes to an infection developing in the space and a resulting empyema. The reasons for formation of a BPF can be.[11]

(i) Early (5 - 10 days post.operatively): usually due to devascularization of the bronchial stump.

(ii) Late (several weeks or months post-operatively);

(a) Usually due tumor recurrence but may also be a complication of radiotherapy in a few patients.

(b) A long bronchial stump.

Management, following early diagnosis is aimed at the prevention of infected fluid spilling over to the contralateral lung.

In cases of early BPF; a closed tube thoracostomy can be inserted initially. If it does not give positive results, then an early thoracotomy is required to evacuate the accumulated fluid. If it is due to a long bronchial stump then reamputation, with reconstruction of the bronchial stump

should be performed. Repair may be achieved by the use of an intercostal muscle flap taken down from an adjacent intercostal space and sutured over the bronchial stamp.

In chronic cases of postpneumonectomy empyema, a simple permanent basal drain may be the last resort. More extensive procedures are aimed at obliteration of the infected space. These include:-

1) Omentum being brought by through chest.

2) The use of a latissimus dorsi muscle flap.

3. Thoracoplasty which is now largely of historical interest. It involves the posterolateral excision of the first ten ribs. The effect is to collapse the infected space. In this study there was one patient with postneumonectomy empyema with BPF and she was initial managed by a closed tube thoracostomy, later re-thoracotomy and myoplasty and finally by open chest drainage.

CONCLUSION

Thoracic empyema remains the commonest thoracic surgical condition at MUhimbili Medical Centre in Dar es Salaam, Tanzania. The rising incidence of pulmonary tuberculosis with HIV desease in Tanzania is a serious and difficult problem to manage under the prevaily socio-economic conditions. A strong and serious national programme against AIDS, early identification and adequate, supervised management of may be some of the steps to try and centre the problem.

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SURGICAL MANAGEMENT OF PATENT DUCTUS ARTERIOSUS

SUMMARY

Between January 1994 and July 1995, closure of patent ductus arteriosus was done in 8 patients in the thoracic unit at Muhimbili Medical Center Dar es Salaam. The ages of patients ranged from 3 years to 11 years with a mean age of 8 years.

Females formed the majority with the ratio of 5:3. The main presenting features were recurrent upper and lower respiratory infections and failure to thrive. The clinical findings were: A systolic thrill in the second left intercostal space and a continuous murmur, over the left precordium otherwise referred to as a machinery murmur. Diagnosis was confirmed at echocardiography. All patients had PDA closure by double ligation through a left thoracotomy. Successful ligation was achieved in 6 patients. However, it was not achieved in 2 patients because these two patients had contraindications for ligation. One patient had other associated cardiac anomalies, while the other patient had a calcified granuloma over the recanalised PDA. The period of hospital stay ranged between 14 to 21 days. There was no mortality.

INTRODUCTION

The ductus is an embryological remnant of the distal left sixth aortic arch, which in the fetus has a diameter equal to the descending aorta. The large amount of smooth muscles in the media is known to dilate with hypoxia and constrict with increased oxygen.[1,3,11] In most infants, the ductus closes within 10-15 hours after birth. Spontaneous closure if not occurring at birth, rarely occurs at 2-3 weeks of age.[1,3,11] Surgical division of PDA has become a standard operation since the first reported case by Gross and Hubbard in 1939,[3,11,16] The single ligature technique has fallen by the wayside to division and oversewing of the cut ends,[11] Division of the PDA is facilitated by a special clamp designed by Potts in 1948, which allows for the curve of the needle, during closure of a divided ductus. The Potts ductus clamps are especially helpful with extremely short PDA.[11] The need for early surgery is reinforced by the finding that PDA is not wholly a benign condition in childhood.[11,16] Many authors[1,7,9,11,12] consider the optimum period to carry out elective PDA closure to be between 2-5 years of age.

The following is a review of 8 patients treated at the thoracic unit. One patient is presented in detail.

CASE REPORT

N.M. Reg. No. 807638, a female child six years of age had presented at the Paediatric cardiac clinic with recurrent respiratory infections and failure to thrive since the age of three years. The mother had noted an abnormal pulsation on the child's chest associated with retardation in physical growth. The child was born after a full term gestation. During the antenatal period the mother did not notice any abnormal rash or undue fevers. The child was delivered in hospital and was a breach presentation. There was a delay in crying at birth. She was however discharged home after three days in apparent good health. She was weaned at four months of age and received all primary immunizations. The child was first born in a family of two. At the time of presentation she was attending a nursery school. The second child was alive and well. On examination she weighed 15kgs. She had a radial pulse of 120 beats per minute which was regular and collapsing. She was not cyanosed or dyspnoeic. There were no finger or toe clubbing. The precordium was not bulging. The apex beat was located in the 5th intercostal space and slightly lateral to the mid clavicular line. There was an apical heave but no parasternal heave. The first and second heart sounds were heard normally. There was a systolic thrill on the left sternal border on the 2nd intercostal space with a continuous machinery murmur maximally heard at the 2nd right sternal

border. The liver and spleen were not palpable. Examination of other systems were essentially normal. A diagnosis of patent ductus arteriosus was made and confirmed by echocardiogram.

Investigation

The results of the following investigations were as follows: Haemoglobin 11.5 g/dl with a normal peripheral film. Serum creatinine was 53 micromol/L. Serum electrolytes estimation were not done due to technical prolems Chest x-ray showed normal findings. Electrocardiogram also revealed normal findings. Echocardiography revealed the followings; left ventricle and right ventricle were normal sized, interventricular septum and interatrial septum were intact with normal motion. The patent ductus arteriosus was evidently seen.

The patient was reviewed by anaestheologist and found to be fit for operation. Blood was grouped and cross matched. Consent for operation was obtained from the parents. In theater the operation site was cleaned and draped. A left lateral thoracotomy through the fourth intercostal space was done. The latissimus dorsi were divided over the fourth intercostal space as was the serratus anterior. The intercostal muscles of the fourth interspace were incised and chest retractor was introduced and opened slowly to expose the thoracic cavity.

The pleura was lifted over the aorta to the level of left subclavian artery. Recurrent laryngeal nerve was identified and retracted anteriorly and the PDA cleared of perivascular tissue. Ligation with linen No. 0 was done at the aortic side and another at pulmonary side. A chest drain was inserted. The pleura was closed with vicryl No 2/0 and the chest cavity was closured in layers. The patient was admitted into the intensive care unit care for monitoring. Post operative pethidine 25mg 6 hourly was given for 48 hours and dextrose in saline was infused at 50mls per hour for 24 hours. After 24 hours a chest x-ray was done, which revealed good lung expansion, the chest tube was thereafter removed in full expansion of the lung. On the sixth day all stitches were removed and the patient was discharged in a good condition. She was followed up subsequently at surgical outpatient clinic, and was found to be in good health.

DISCUSSION

Aetiology and incidence

The following is a tabular summary of age and sex distribution among 8 patients with PDA.

Age groups in year	Sex		Total
	Male	Female	
1 - 5	1	1	2
6 - 10	2	2	4
11 - 20	-	2	2
Total	3	5	8

Note: Female to male ratio was 5:3. The mean age was 8 years.

The ductus is an embryological remnant of the sixth arch. The ductus is a large vessel connecting the pulmonary artery to the ventrolateral aspect of the aorta just to the left subclavian artery.[3,11] Before birth the blood is directed from pulmonary artery through this ductus into the aorta because of the high pulmonary vascular resistance. Soon after birth the alteration in pressure ratio between systemic and pulmonary system is reversed resulting in a left to right shunt. A patent ductus arteriosus is one of the most common congenital cardiovascular anomaly and occurs in approximately one in two hundred live births.[3,11] Female sex predominance compares favorably well with other studies.[8,12,16] In this study female

to male ratio was 5:3. There is known to be higher incidence of persistent patency in premature infants, have high probability, children born with congenital rubella, and individuals born at altitudes above 3,000 meters.[3,11] This high attitude has been attributed to chronic hypoxia due to decreased partial pressure of oxygen. This relation has not been validated in the Caucasian population in USA.[16] More significant is the incidence of congenital heart disease which varies among studies depending on the population studied and diagnostic methods employed. In this study history suggestive of congenital rubella was recorded in two cases.

Over the last 50 years enormous experience in the management of PDA has accumulated making PDA closure a safe procedure with extremely low mortality. The majority of surgeons[11,12,14,15] prefer ligation in younger infants and division and oversewing of ductal ends in older children. In this study 6 patients out of 8 patients underwent double ligation. However, ligation was not achieved in two patients, one had a calcified granuloma over the recanalised PDA, and the other patient had cardiac anomalies of pulmonary vein drainage (supracardial Type 1). This is one of the types of the total anomalous of pulmonary venous connection, whereby there is a connection between the pulmonary veins with superior vena cava, azygos vein or innominate vein.[5,13]

Diagnosis

It is a fact that a history of congenital rubella is frequently associated with PDA. Detailed history about the mode of delivery and other congenital anomalies are helpful facts. Failure to thrive and frequent pulmonary infections are common features. The classical diagnostic finding is a systolic thrill in the second left intercostal space or suprasternal notch, associated with a machinery murmur found in second or third intercostal space. The murmur is characteristic and allows a diagnosis in about 95 percent of cases. The peripheral pulse is strong and collapsing in nature. The heart size may be normal or enlarged. X-ray films of the chest will show increased pulmonary vascular marking. The echocardiogram is the main diagnostic modality in many institutions. The left atrium, aortic ratio (LA/AO) is increased in an infant with a large patent ducts. However, the final word in diagnosis is the visualization on cardiac catheterization of a patent ductus, usually well defined on lateral film, or with the typical picture of the catheter through the ductus visualizing both the descending aorta and pulmonary vessels. This method helps in diagnosing associated cardiac anomalies.

Treatment

Surgical

Surgery is the main modality of therapy. There is no special problem associated with the decision to operate in patients with isolated PDA, apart from age. When the diagnosis is made it is a sufficient reason for closure. In symptomatic patients, closure should be performed when the diagnosis is made. In asymptomatic children intervention can be postponed if desired, but should be done in the pre school years. Older patients should have the ductus closed when the diagnosis is made.[13,16] Infants under 6 months of age carry a greater surgical risk than older babies.[7,9,12] Keith[9] refers to a study where 60 babies under 6 months (with PDA) were operated and the mortality was 8.3 percent. While non of the 148 babies operated between 6 months and 12 years of age died. Many authors[7,11,12] consider an optimum time for elective surgery to be between the ages of 2 and 5 years. In this study the age at operation was between 3-13 years with a mean age of 8 years.

According to Campbell,[3] if the ductus escapes detection in childhood it is been shown that those who are alive after 17 years of age have a life expectancy of that of one half the normal population. He has also shown that 34 percent of patients with ductus are dead by the age of 40 years, and 60 percentage are dead at 60 years. Complications in the adults are:-

- Eisenmenger's syndrome, which is reversed flow in the ductus secondary to pulmonary hypertension and results on vascular changes. Cyanosis and clubbing of the finger and toes are present. These are absolute contraindications for surgery.
- Congestive heart failure, infective endocarditis, calcification, and aneurysm of the ductus make repair of the ductus more difficult. In most instances repair should be completed while on cardiopulmonary by-pass.[11] The ductus is exposed from a posterolateral incision through the 3rd or 4th interspace. The closure of the ductus has undergone great evolution and modification since Gross and Hubbard[11] reported their first ligation. The original technique of simple ligation has been replaced with division and oversewing, due to recanalization in some case.[12,14] In this study 6 out of 8 patients underwent double ligation of the ductus and no death was reported.

CONCLUSION

Elective closure of PDA should carry virtually no risk other than that of anesthesia. Mortality in experienced hands is less than 0.5% percent. However, additional heart anomalies may complicate the procedure.

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TORSION OF THE TESTIS

SUMMARY

Torsion of the testis seems to be uncommonly seen at the Muhimbili Medical Center, Dar es Salaam. In a period of one year, between January and December 1994, the author recorded only twelve cases. The age of the patients ranged between 10 and 30 years with mean age of 14 years. Seven of the twelve patients studied had testicular torsion on the left. All patients had history of sudden onset of pain localized to the scrotum, followed by swelling of the affected testis. One patient had a history suggestive of previous episodes of torsion. Most of the patients reported to hospital more than 24 hours after the onset of their problem (60%). However, exploration of scrotum was done in all cases. Seven patients with gangrenous testes, were managed by orchidectomy of the affected testis and orchidopexy of the opposite. Five patients were managed by bilateral orchidopexy after detorsion of the affected side. Testicular salvage rate was 45% in this series.

INTRODUCTION

Torsion of the testis, or more correctly termed torsion of the spermatic cord, was first reported by Delasiauver[10] in 1940, affecting an undescended testis. The first British case, was recorded in 1881 by Langton[20] at St Barthomew hospital London and this was also the first report of torsion affecting

a normal descended testis. The torsion could not be released and the organ ultimately sloughed.

Torsion of the testis can occur at any age, however, Williamson et al;[24] reported to be commonest around puberty. In the past testicular torsion was reported as uncommon, however, many hospital data from developed countries nowadays show an increasing annual incidence.[23,25,30] The magnitude of the condition in our society is still undetermined, but is been an uncommon condition at the Muhimbili Medical Center.

The difficulty in the diagnosis of testicular torsion and its management as a surgical emergency in young males still persist in our society. The present study has pointed out some of these difficulties and possible ways of managing this condition.

Non-viable testis should be removed to prevent subsequent extrusion through the wound or possible autoimmunization. Beyond 10 hours of torsion greater than 50% of testes atrophy[5,34]. Immediate exploration and fixation of the opposite testis is mandatory. One patient has been presented fully, and a discussion focusing on precipitating factors, incidence, management and the outcome of the testis after torsion is made.

CASE PRESENTATION

J.C. IP No 797314, a 14 years old patient was admitted at Muhimbili surgical ward with complaints of sudden onset of left scrotal pain of one day duration. The pain was sharp, continuous, radiating to the medial aspect of the left thigh and the left inguinal region. As pain persisted, he noticed a swelling of his left testis. He vomited once. There were no associated urethral discharge, trauma to his genitalia, fever, inguinal swelling or history of similar attack in the past. He was a standard seven primary school pupil, second in the family of three children and no other family member with similar experience.

Clinically he was in pain, anxious, afebrile, not dehydrated or pale. He had stable vital signs. His abdomen was not distended and moved well with respiration. There was no organomegaly. His left testis was swollen, tender and non fluctuant. There was no urethral discharge. Digital rectal examination, revealed the following: There was no perianal lesion, normal sphincter tone, normal rectal mucosa and no palpable mass. Examining finger was stained with normal brownish stool color.

From the history and clinical findings a provisional diagnosis of left testicular torsion was made. The patient was therefore planned for emergency testicular exploration. Preoperatively, the patient was advised not to take anything

orally, his haemoglobin was established, and a signed consent was obtained from his father. An intravenous dextrose in normal saline was set, sedation and monitoring of vital signs were started.

In the operating theater, the patient was given a general anaesthesia then the operating site was cleaned with iodine and spirit and draped with sterile towels. Bilateral testicular exploration was performed through a scrotal approach. The affected left side was explored first. A small secondary hydrocele had formed already. The tunica vaginalis was opened and serosanguinous fluid was drained. A torsion of the testis round the mesorchium, one and half turn and gangrenous testes were noted. Detorsion of the left testis was done and a decision was made to do an orchiectomy. The cord and testis were mobilized, divided between ligatures, dissected and then the testis was removed. A drain was inserted. Thereafter the scrotum was closed in two layers using catgut No. 2/0 to the inner layer and silk No 3/0 to the outer layer.

Exploration of the right testis was also done. The right testis and epididymis were found normal. There was a long mesorchium as well. Orchidopexy was done by fixing the tunica albuginea to the lateral scrotal wall, using silk No 2/0 sutures. No drain was left in situ.

Thereafter the scrotum was closed in two layers. Post operatively, a gauze scrotal support was applied and pethidine injection 6 hrly was given for the first 24 hrs. No antibiotics were prescribed. The drain was removed after 24 hrs. He fared well and scrotal sutures were removed on the seventh day post-operative following which he was discharged home.

The patient was seen at the outpatient clinic monthly for the first two visits. His right testis appeared normal in size. He did not have an other attack during this period and was discharged from the outpatient clinic with the advice, that he should report to the hospital immediately if he experienced a similar attack of scrotal pinfor early evaluation and treatment.

DISCUSSION

Aetiology:

Since Astley Cooper[7] first noted that the entire scrotal contents of the neonate could be freely lifted out of the scrotum without tearing any tissue, this mechanism is believed to account for the rare extravaginal torsion which is virtually confined to new born infants.[33] Both Scudder[31] and Keith[13] drew attention to the fundamental anatomical abnormality predisposing to intravaginal torsion, namely a freely mobile testis suspended by a long mesorchium within a

capacious tunica vaginalis. As in the present study, twelve patients had long mesorchium. The high investment of the tunica vaginalis producing the " bell-clapper[1]" testis has been repeatedly described, and Barker and Raper[4] suggested that a normal testis will not undergo torsion unless submitted to severe trauma. It is surprising, in view of the fact that the anomaly is nearly always present on both sides,[1] that bilateral torsion is not a commoner event.

The actual force initiating torsion in a susceptible testis is still not clearly understood. Possibilities include vigorous exercise, direct trauma, a powerful cremasteric reflex and sexual excitement[23,24] In the present study, no obvious precipitating factors were identified. Cold weather could explain seasonal variations in incidence within British Isles[17,25] and geographical variations within the Indian subcontinent.[25] Cold can contract the dartos muscle by direct stimulation or by local reflexes. The capsule of the testis is usually regarded as being composed of fibrous material, but experimental work has shown that it contains contractile tissue that responds to cholinergic and adrenergic stimuli. Similarly, capsular contractility been observed with low temperature.[24,25]

Pathophysiology

Twisting of the spermatic cord may cut off the venous drainage and arterial supply to the testis in question.

Usually, the thick walled arteries remains patent so that intense vascular engorgement and venous infarction follow.[26]

The outcome depends on the duration and severity of the process.[24] The morphologic changes may range from those of merely intense congestion to widespread extravasation of blood into the interstitial tissue of the testis and epididymis. In more extreme instances, hemorrhagic or even anaemic infarction of the testis may occur. In late stages, the testis is markedly enlarged and is converted virtually into sac of soft, necrotic, hemorrhagic tissue.[26]

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

Barker and Raper[4] noted a 90% testicular loss after torsion, 80% by immediate orchidectomy, and 10% by subsequent atrophy. In man, when the duration of torsion exceeds 4 hours some degree of testicular atrophy is almost inevitable, and beyond 12 hours the organ will shrink to less than ½ of its normal size.[34] Kayoga[18] of Mulago, in Uganda reported a study of 18 patients in whom due to delay in diagnosis, 10 testis were necrotic, and orchidectomy was done. Eight patients, reported early and had viable testes which were fixed. Six of

the testes which were viable were operated on within 24 hours, and only two were found viable after a48 hours delay.

Reduced fertility following torsion of the spermatic cord was described by Krarup.[19] The finding has been confirmed by several authors, both in animal experiments and in clinical follow up studies.[5,11] Williamson[23] introduced the term "sympathetic orchidopathia" to describe the condition and compared it with sympathetic ophthalmia. Hormonally mediated contralateral damage has also been suggested[3] and more recently, impaired spermatogenesis has been found in biopsies taken at the time of operation from the contralateral testis suggesting an underlying congenital defect. However, several hypothesis have been put forward to explain reduced fertility after unilateral torsion.

Both testes may be congenitally abnormal and the "bell clapper" anomaly might be associated with defect in spermatogenesia[16] due to testicular dysplasia. This hypothesis is supported by biopsies from the contralateral testis taken at the time of operation. Anderson et al[2] found impaired spermatogenesis, partial maturation arrest and sertoli-cell syndrome in 67 of the 113 biopsies. This theory is some what contradicted by some authors[8,11] who found no significant differences, when they compared biopsies from normal testis with unaffected, contralateral testis from patients with a duration of symptoms less than 5 hours, while changes occurred

in the contralateral testis in patients with a longer duration of symptoms.[8,11] Both testes may be damaged by recurrent subtorsions, since the bell-clapper phenomena is usually bilateral. More than a third of patients report of previous attacks of pain, but these are usually from the side that later undergoes torsion.[24] In a recent study, one patient had history suggestive of two previous attacks.

The finding of antisperm autoantibodies in animal experiments and the reported beneficial affect of immunosuppressive treatment led to the theory that immunological factors may play a role in the reduction of fertility.[14] However, some authors have concluded that the poor sperm counts observed following unilateral testicular torsion seems to owe more to an associated underlying defect in spermatogenesis than to sympathetic orchidopathia, which includes congenital testicular dysplasia, OAT's syndrome and sertoli-cell syndrome.[34,35]

CLINICAL FEATURES

The sudden onset of scrotal pain and swelling in an adolescent nearly always indicates testicular torsion.[24] Occasionally pain radiates to the abdomen, perineum or the thigh. Other highly suggestive features are vomiting (present in 35-50% of cases),[24] a past history of similar attacks (37 - 47%). In a recent study, one patient presented with two

previous attacks which were relieved by analgesics, probably spontaneous detorsion took place. The affected testis is swollen, and tender in one third of patients, the testis is usually reported to be in horizontal lie (34%),[24] but it is sometimes difficult to appreciate due to oedema formation and severe testicular tenderness. Confusion can arise when the pain is mild, gradual in onset or ectopic (even leading to inappropriate appendicectomy)[24]. This occurred in three boys in one series.[24] One had appendicectomy done on the day of admission, and right orchidectomy the following day. Mild urinary symptoms accompany 5 to 7 percent of testicular torsions,[24] scrotal erythema and pyrexia may develop in afew hours.

Prehn (1934)[27] suggested that scrotal elevation relieved the pain of orchitis but aggravated that of torsion. Most authors,[1,6,18] however, have found this sign notoriously falliable, and a much more reliable guide is the presence or abscence of urinary infection.

Epididymo-orchitis is uncommon in adolescent boys in the abscence of underlying urinary tract infection and is usually a manifestation of more generalized urogenital infection.[21]

In a recenty study the presenting features were, scrotal pain of sudden onset(12), vomiting(1), recurrent scrotal pain(1), testicular swelling(12).

Diagnosis

The overall accuracy of the preoperative hospital diagnosis for torsion of the testis was found to be 60 to 90%[17]. In this study diagnosis was made from history and physical examination and confirmation at surgery(100%). Four methods can be used to supplement clinical diagnosis.

i) A computer aided diagnosis

ii) Aspiration

iii) Doppler ultrasonography

iv) Dynamic scrotal scintiscanning

1. **A computer aided diagnosis:** The development and evaluation of the computer requires four steps,[17] construction of data base by using previous literature, application of Baye's theorem (this provides a method of combining probabilities for various indicant's i.e. clinical symptoms signs to give an overall estimate of the probability of a specific event occurring), clinical details of the parameters described and analysis by using a computer which prints out the percentage probability for each of the three conditions of epididymitis, torsion of the testis and torsion of an appendage. According to Goulbourne[15] a computer provided the correct diagnosis retrospectively in 89% of 236 patients with torsion while surgical registrars were even more successful with an occuracy of 92%. The computer was very good at predicting

epididymitis (92% accurate) and very poor at predicting torsion of appendage (19%).

The technique is inexpensive, rapid, immediately available, non invasive and easy to use. However, this technique is not used at Muhimbili Medical Center.

2. **Doppler ultrasonography:** This offers a simple method for determining testicular blood flow, unfortunately the examination is both uncomfortable and rather insensitive in acute torsion.[6,34] The precise technique of the Doppler examination is of critical importance if misdiagnosis is to be avoided. Most if not all, of the reported cases of misdiagnosis can probably be attributed to faulty technique. Testicular torsion can be diagnosed incorrectly as epididymitis, if a pulsatile signal, originating from either the inflamed scrotal skin or the testicular artery proximal to the torsion, is mistakenly interpreted as arising from the testis. These errors can be avoided by applying tunicular compression and by pointing the transducer towards lower pole of the testis.[28] Doppler examination is rapid, simple and easy to learn. The diagnostic accuracy is about 82%.[17]

3. **Aspiration:** Needle aspiration of bloody secondary hydrocele favors a diagnosis of torsion,[27] but the technique is seldom practiced.

4. **Dynamic scrotal scintiscanning:** This reliably and rapidly differentiates testicular torsion from epididymitis.[28] Acute torsion, late torsion and resolved torsion give different patterns of isotope uptake, but the method is useless in detecting torsion of an appendage. A testicular torsion shows the 'halo' sign characterized by a central 'cold' spot with a 'hot' perimeter of markedly increased uptake of isotope. However, some tumors demonstrate a 'halo' sign, with central necrosis and peripheral increase in blood supply and can be confused with late torsion.[34] This technique is however more accurate than Doppler examination.

Treatment

The successful management of a patient with torsion depends on prompt reporting to hospital, correct diagnosis at first consultation and urgent exploration.

Torsion of the spermatic cord is an acute urological emergency and delay jeopardizes the viability of the testis.[5] Experimentally and clinically testicular survival has been correlated with the interval from the onset of symptoms to surgical detorsion.[24] Detorsion results in success if performed within 4-6 hours, conversely, progressively more testes are lost as the period of ischaemia lengthens.[5,24]

The fastest way for a physician to alleviate testicular ischaemia is by manipulative detorsion at the initial physical

examination.[9] Although this technique is done as a first aid measure,[24] explorative surgery should always follow. The first case of manipulative reduction of torsion was reported by Nash[22] though the testis subsequently underwent partial atrophy. Two years later Van de poel[35] underlined the value of manipulative reduction as the first aid measure he showed that the direction of the torsion is usually away from the septum dartos, possibly owing to the line of pull of the cremasteric muscle fibers. Cattolica[9] found 7 of 8 patients with torsion occurring in a medial direction. The findings indicate that the direction of the torsion can be medial or lateral.

The technique of manipulative reduction of torsion of the testis is as follows; the operator sits at the foot of the bed facing the patient in supine position. The affected testis is gently manipulated until the patient feels relief. The correct direction of manipulative rotation to relieve torsion has to be found in every case by experiment.[30] Correct rotation brings immediate relief, incorrect rotation increases the pain, therefore rotation should be continued until the patient is free of discomfort.[30] One to three turns usually are sufficient to relieve the torsion.[9] Return of the anatomy to normal as judged by physical examination is required in addition to relief of pain before detorsion is considered complete.[9] Many testes can be salvaged if manipulation is done within 24

hours.[9] Frazier and Bucy[12] reported testicular salvage in all four testes and Sparks[30] in 73 percent. Although manipulative reduction can be attempted, operative detorsion remains the treatment modality of choice.[2,24] Surgery may be performed under either general or local anaesthesia. The scrotum is explored through a low transverse or longitudinal incision and the tunica vaginalis is opened widely in a transverse axis. Detorsion is performed after extruding the testis through the tunica, which is then reflected upwards and retained at the root of the spermatic cord. If the viability of the testis is in doubt at operation after torsion has been reduced, the testes should be wrapped in a warm moist pack and reinspected after 5 to 10 minutes. Bright bleeding following incision of the tunica albuginea is a promising sign.[24] It is obviously wise to retain the testis in which there is a possibility of residual function, particularly if the period of torsion has been less than eight to ten hours and the cord has not been twisted less than 1½ times. If the testis is infarcted, orchiectomy is done to prevent subsequent extrusion through the wound[24] or possible auto-immunization.[3,23] The scrotum is then closed leaving a drain in situ. If the testis is salvageable, orchidopexy is then done, fixing the tunica vaginalis to the lateral scrotal wall using silk No. 2.0 and the scrotum closed without drainage. Seven out of five patients in this study underwent orchiectomy of the affected

side and orchidopexy of the contralateral testis. Five of twelve patients underwent bilateral orchidopexy.

CONCLUSION/RECOMMENDATIONS

The successful management of a patient with torsion depends on correct diagnosis at first consultation and urgent exploration. Therefore the author recommends that it is a good rule that all children, prepubertal patients and adolescents presenting with a sudden testicular pain without fever or urinary tract infection, should have an emergency testicular exploration. Thereafter they ought to followed up closely for long duration with respect to fertility with an ideal of proving the reduced infertility in patients with torsion of testis.

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FOURNIER'S GANGRENE OF MALE EXTERNAL GENITALIA

SUMMARY

An analysis of ten cases of necrotizing cellulitis of the male external genitalia (Fournier's gangrene) seen at Muhimbili Medical Center (MMC) between October, 1993 and July 1995 is presented. Details covering age, extent of gangrene, associated conditions, microorganism involved, serology, forms of surgical treatment offered and duration of hospital stay was analyzed. Their ages ranged between 30 to 70 years with mean age of 48 years.

All patients had similar initial presentation of scrotal pain and swelling in 10 cases and fever in 4 patients. This was later followed by either cellulitis, or by gangrene with a demonstrable typical demarcation line between viable and dead tissue. Gangrene was limited to scrota in 8 cases, and only in two cases gangrene extended up to the perineum. There was no penile or abdominal wall involvement.

Hydrocele was the commonest associated condition and was present in 4 patients. Two patients presented with urethral discharge. Two patients were ELISA positive and another two patients had diabetes mellitus. The diagnosis was based on the fulminating clinical progression of scrotal gangrene, and confirmed by presence of multiple pathogenic micro-organisms from cultures and histological findings.

Management included debridement, daily wound toilet and antibiotic therapy according to sensitivity pattern from culture isolates. Commonly used combination of antibiotics, was chloramphenicol and metronidazole. In six out of ten patients, hydrocelectomy and secondary suture was done while in two patients subsequent skin grafting was necessary to cover the skin defect while debridement alone was successful in two cases. No death was encountered. Early diagnosis and vigorous medical and surgical treatment is necessary to avoid extensive tissue destruction, toxaemia and subsequent mortality in patients with Fournier's gangrene.

INTRODUCTION

Gangrene of the scrotum has continued to be a surgical rarity since the first reports of this mystifying penoscrotal affection in 1764. In 1883, Jean Alfred Fournier[2] a French venereologist presented 5 cases of scrotal gangrene that ~~was~~ were characterized by an idiopathic, involvement of the genitalia in otherwise healthy young male patients. Since then this syndrome has borne his name. Since that time many cases ~~has~~ have been described and the syndrome has came to encompass all necrotizing subcutaneous tissue of the male genitalia whether primary or secondary ie. caused by pathological processes in the anorectal area, lower urinary tract, and scrotal or perineal skin.[2,4,6]

Fournier's gangrene is an aggressive inflammatory disease affecting the perineum. Characterized by necrosis of subcutaneous tissue and skin with a typical dermacating zone, with the histological hallmark of vascular thrombosis and subcutaneous endarteritis.[4] Clearly, it can no longer be considered idiopathic in its origin, as in most cases infection can be localized to a cutaneous, urethral, or rectal source.[5] It presents in a wide age range and has an indolent onset, thus requiring a high index of suspicion. It may be fulminating and progressive in the case of immunocompromised and underlying debilitating illnesses. Despite aggressive antibiotic therapy and debridement, it is sometimes associated with a high mortality rate.[19] The rate has been higher in older patients, those with rectal focus, and diabetics. Initial treatment of Fourniers gangrene includes surgical debridement, antibiotics and treatment of any underlying aetiological factor. New reconstructive efforts such as medial thigh pedicle flap, have improved the cosmetic effect after extensive debridement.[5,7,15]

The following is an account of a patient with Fournier's gangrene. He was managed in the surgical firm III at Muhimbili Medical Center with combination of antibiotics together with debridement and finally secondary suturing of the scrotum. The pathogenesis and management of the condition is described in detail.

Case report

A 50 years old male patient Reg No. 806628, a Dar es Salaam resident was admitted in the male surgical firm III. His main complaint was swelling and pain in the genital area of four days. The pain started suddenly in the scrotum and penis. This was accompanied by fever and swelling of the genitalia which was getting progressively worse. He also developed loss of appetite and vomiting. It was at this time that he decided to seek medical attention and was referred to Muhimbili Medical Center from a nearby dispensary. According to the patient this was the first attack. He had a history of previous urethral discharge which was treated with antibiotics. He denied history of any perianal swelling. His scrotum had been of normal size. His appetite had been good prior to the attack and he had no problems with opening bowels. Other systems were normal. He never had any serious illness before such as chronic cough, hematuria, polyuria or lower limbs swellings. These was no history of previous accident and he had never been admitted before in any hospital for surgery or otherwise. He was a peasant, married to two wives and had a number of children and grand children. He denied smoking, or drinking alcohol.

On examination, the patient appeared to be ill looking with fair nutritional status. He was not dehydrated, not pale and not jaundiced. Peripheral lymph nodes were not palpable,

and he did not have pedal oedema. His temperature was 38.5°c, blood pressure was 120/70 mm/Hg and pulse rate of 100/minutes, regular of good volume. The abdomen was scaphoid, soft and non tender, no masses were palpable.

Hernial orifices were normal. The scrotum and penis were swollen with the scrotum appearing shiny, hot, tender, non fluctuant and irreducible. Rectal digital examination revealed a normal rectal mucosaa normal prostate and no masses or ulcers within the rectum and no evidence of rectal bleeding.

A provisional diagnosis of Fournier's gangrene was reached with the differential diagnosis of epididymoorchitis. Aspiration with a wide bore needle 9-18 was attempted and clear serous fluid was aspirated and sent for cell and bacterial analysis. The patient was subsequently started on a combination of metronidazole 400mg twice daily and chloramphenicol 500mg, 6 hourly orally and bed rest with scrotal support. On the third day of admission gangrene set in the scrotum starting with the most dependent part spreading towards the penis. Two days later the gangrenous part was dermacated and the scrotal skin sloughed off. At this stage a final diagnosis of Fournier's gangrene was made. This process took three days and about one third of the anterior scrotum was involved. The penis remained swollen but unaffected by the gangrenous process. After same time following dermacation, debridement was done. All the necrotic tissue was excised and

the area cleaned with hydrogen peroxide in two sessions. The testes were exposed, healthy and were free from the gangrenous process. The necrotic tissue was sent for tissue culture and sensitivity, and histological analysis. The patient was maintained on the same antibiotics as culture results indicated mixed organisms growth with predominantly E. Coli and staphylococcus. After one week of therapy, fever and pain disappeared and the patients appetite improved. The results of the following investigations were as follows:-

1. Full blood picture;

   Hb 10g/dl

   WBC 12,000

   Neutrophils 80%

   Eosinophils 5%

   Lymphocytes 15%

   Blood film showed normochromic, normocytic red blood cells morphology.

   ESR 35mm/first hour.

2. Urinalysis: clear, albumin (negative), sugar (negative) leucocytes 3/HPF

3. Pus swab for culture/sensitivity, Gram staining revealed, gram positive cocci, gram negative Bacilli and culture isolated staphylococcus aureus and E.Coli, sensitive to chloramphenicol, gentamicin, and

Metronidazole

4. Random Blood Sugar was 4.7mmol/l

5. Serum protein Total 70.5g/l and albumin 25.5g/l

6. Histological analysis revealed: Polymorphonuclear focal tissue necrosis with vascular thrombosis.

Three weeks after the debridement and dressings, healthy granulation tissue had formed. The patient was taken back to theater for secondary suturing which was done under local anesthesia. The skin edges were trimmed and undermined to provide sufficient opposition over the granulation tissue without tension, using silk No. 2.0. Healing went on well and stitches were removed on the seventh day and the patient was discharged after staying for almost one month in hospital. He was reviewed after two weeks at the surgical outpatient clinic and found well with complete healing of the scrotal wound. He had no urinary complications and he still enjoyed his potency.

The characteristics and modality of treatment of other patients are summarized in a tabular form.

Table 1: Age incidence of the 10 studied cases

Age group (years)	Number of cases
31 - 40	1
41 - 50	4
51 - 60	4
61 - 70	1
Total	10

The mean age was 48 years

Table 2: Clinical manifestation of Fournier's gangrene

Symptoms/Sign	Frequency
Scrotal pain	10
Scrotal oedema	10
Fever	4
Cellulitis	4
Necrosis	3

NB: All patients presented with scrotal pain and oedema

Table 3: Associated conditions

Associated condition	No of cases
-Hydrocele bilateral 1 unilateral 3	4
-Diabetes mellitus	2
-HIV infection	2
-Urethral discharge or periurethral abscess	2
Total	10

NB: Hydrocele was the commonest associated condition

DISCUSSION

Incidence and Aetiology

The limited number of studies on the clinical features, complication and treatment of Fournier's gangrene are all reflected in a few conflicting published reports. Since Fournier described the condition in 1883, more than 500 cases have been reported.[1,2] Akinola[1] quotes Pande and Mewara in 1967 describing this ailment as an infective gangrene, whereas Bahlmann[2] and Pess[16] described it as necrotizing fascitis.

The disease seems to be more prevalent in African and Asian countries although the scarcity of recorded cases does not reflect the actual incidence.[2,9,11] In his series Akinola[1] attributes the ailment to low socio-economic state of communities where adequate social amenities are lacking and amongst those with poor genital hygiene. This concurs with earlier observation by Fahal et al.[6] However Efem[5] and associates observed the concept that Fourniers gangrene which was traditionally thought to arise spontaneously, is no longer true. They reported four cases of Fourniers gangrene, all associated with a predisposing causes, two with diabetes mellitus, two secondary to urinary tract pathologies, and one secondary to and infected wound after an inguinal herniorrhaphy.[5] Kamei[9] and associates also support this concept and in a period of two years, five cases of Fournier's gangrene following anorectal disorders were encountered.

Perirectal abscess was the most common associated underlying condition occurring in three patients. E.Coli was the predominant organisms cultured in all cases. In this study the predisposing factor were as follows. Four patients had hydrocele, two patients had diabetes mellitus, two patients had urinary tract infection and another two patients were HIV positive.

Culture results revealed mixed organisms with predominanty of E.Coli in four patients. Fournier's gangrene can be very aggressive if improperly treated and the gangrene can extend upwards to the anterior abdominal wall, or to involve the whole perineum up to the penis. Therefore a diagnosis of "Idiopathic" Fournier's gangrene should be accepted only after all possible underlying causes have been excluded.[1,2,4,8,11]

Bacteriology and pathophysiology

The bacteriology of the gangrene is highly complex partly because of the location of the lesion and the multiplicity of predisposing factors. However, the common denominator in aggressive genital gangrene is synergism of infection. In most cases the causative organisms is usually a combination of aerobic and anaerobic bacteria.[2,7,8] Earlier reports suggested that synergistic gangrene was most often secondary to a combination of microaerophilic streptococcus and staphylococcus aureus. In 1945, Flanigan[7] reported that 30 to

44 patients yielded one or both or these organisms. Recently, most investigators have isolated gram negative bacteria (Escherichia Coli; Klebsiella and Pseudomonas) in combination with anaerobes, including especially the Bacteroides.[4,7,12] The interaction may predispose to vascular thrombosis. Aerobes cause platelet aggregation hence accelerating coagulation by fixing complement. Some anaerobes promote clotting by their production of heparinase.[12] Bacteroides species inhibit phagocytosis and the killing of a variety of aerobic organisms, this favors their proliferation. These factors may explain the prominent vascular thrombosis seen in necrotizing fascitis which is responsible for subsequent tissue necrosis and gangrene.

The port of entry of microorganisms is as follows:-[4,11,16,19]

1. Trauma to the area, which provides access of organisms to the subcutaneous tissue.
2. Extension from a urinary tract infection probably most commonly periurethral gland infection with extension along fascial planes to involve the penis and scrotum.
3. Extension of an infection from the perianal area or less commonly space along fascial planes to involve the penis and scrotum.
4. Wounds sepsis after surgery of the perineum or inguinal region, these includes: vasectomy, herniorrhaphy and hydrocelectomy.

In all cases, rapid proliferation of bacteria occurs at the superficial fascial layer, spreading along fascial planes and producing on obliterative endarteritis of the overlying skin and subcutaneous tissue.[19] Bacterial synergism leads to extension of the infection and further proliferation within the subcutaneous tissue, thus causing extensive subcutaneous necrosis.[4,12] Spread takes place along fascia planes and does not involve the underlying muscle. However, fascial necrosis extends beyond the cutaneous manifestation and proceeds under what appears to be normal skin. The scrotal wall reacts to infection by considerable edema, which may further interfere with perfusion.[12] If the process remains untreated the small subcutaneous endarteritis undergoes thrombosis which is a histological hallmark of necrotizing fascitis.[4] This results in ischaemia which contributes to explosive extension of the infection, necrosis of the involved tissues and secondary dermal gangrene. In addition, ischemia allows the proliferation of obligate anaerobes and promotes the anaerobic metabolism of facultative anaerobes. As a result of the increased anaerobic bacteria metabolism, hydrogen and nitrogen gases which are relatively insoluble are produced and accumulate in the subcutaneous tissues. This usually leads to a subcutaneous emphysema which become clinically apparent as crepitus or with plain radiograph as a visible gas in the tissues.[4] With local trauma including surgery, organisms

present on the skin can enter the subcutaneous tissues and cause dermal and epidermal coagulative necrosis and infection.

In patients with epididymitis, small foci of infection may rupture into the scrotal sac. More frequently, the extension is from infected periurethral glands especially in cases with urethral strictures. This may be responsible for many apparently diagnosed as idiopathic cases, which is no longer accepted.[10,13]

The mechanism of spread from the colorectal area to the genitalia is unclear, but may occur via the interrupted connection of Colle's fascia to the urogenital diaphragm.[7,14] Whatever the pathway, organisms reaching the subcutaneous tissue cause obliterative endarteritis of the branches of the pudendal arteries, testicular and cremasteric arteries which supplies the scrotal skin. This results into an acute dermal gangrene.

Clinical features

The illness typically presents as pain or itching of scrotum or penis. While, Fournier original context refers to a healthy young man, in subsequent published series and also in this study, patients have been in the older age group, more than 40 years. Although the first symptoms are usually mild pain or itching in the genital region,[12] several early authors[3,18] described a dramatic explosive nature of the disease taking the patient from robust health to severe

prostration within a few hours, it may be surprising to see a patient presenting with total genital gangrene within hours or few days of onset.[12] In his series Akinola[1] noted that in over 50% of patients, these were warning signs at the onset which included itching and peppery-sensation of the genitalia. This is refuted by Martti[12] who feels the initial symptoms are slight and do not warn the patient of the rapid and dramatic development of the condition. Fahal et al[6] in analyzing patients found a prodromal period of pyrexia which was followed by scrotal pain and swelling, before emphysema, frank gangrene and sloughing set in. The duration of prodromal symptoms varies from one day to a week, with a mean interval of 2 to 3 days.[2,7] In this study the symptoms took 3 to 7 days, in most patients ranged between 3 to 4 days. Early in its course, Fournier's gangrene resembles acute orchitis, or epididymitis, testicular torsion or strangulated hernia.[1] However, in some instances pain in the involved area is often out of proportion to findings on physical examination, particularly in the early stages of development of the syndrome.[7] The condition is usually associated with fevers and chills. Rarely a patient may show evidence of severe toxaemia.[1,6,7] The scrotum often enlarges to several times it's normal diameter, the skin becomes hyperemic and later ominous black spot ensues.[4,12] This may involve the penis, scrotum or both. Either may assume very large proportions and sometimes

crepitus on palpation may be felt. The inflammation and subcutaneous emphysema may extend superiorly up the abdominal wall as far as the axilla, interiorly to the thighs and posteriorly to involve the perineal tissues. In severe cases, systemic effects of infection manifest as severe prostration, nausea and vomiting.[11,12,17]

In untreated cases, the scrotum and penis develop gangrene. At this stage pain almost subsides, probably owing to destruction to sensory nerves. The involved area sloughs off. With this sloughing, the systemic symptoms and signs commonly abort as a result of naturally occurring drainage.[14,17] The areas between the viable and dead skin becomes clearly demarcated and the slough separates in about 3 days time.[3,14] The testes, bared off their tunica vaginalis hang suspended by their cords.[2] The testes are free of gangrene because of its separate blood supply. Finally with passage of time, the healing process result into formation of a clean granulation tissue. In this study, the main mode of presentation at onset included scrotal pain and oedema in 10 cases and fever in 4 cases.

Others had cellulitis and necrosis, thereafter gangrene and sloughing set in.

Diagnosis

The diagnosis is based on the history of a fulminant progression of the disease, and on presence of multiple pathogenic organisms in the Gram stain or culture. Other investigations includes:-

[1] Plain radiographs can be used in demonstrating subcutaneous emphysema before sloughing has set in.

[2] Color Doppler Ultrasonography B Scan imaging and radio-isotope scanning. Flamigan[7] and associates demonstrated that scrotal pain is initially evaluated by Color Doppler Ultrasonography providing information on the presence or absence of blood flow within the testis and B scan imaging providing complementary information about lesions such as torsion of the appendix testis and testicular abscess. While radioisotope scanning provides similar information to color Doppler about testicular blood flow, it does not demonstrate the anatomy. Therefore, the above three methods helps to differentiate Fournier's gangrene from other causes of pain in the early stage. (Radioisotope and Doppler are available at Ocean Road Hospital but were not used in this study).

MANAGEMENT

Treatment of gangrene of scrotum is two fold. The first stage is recognition and treatment of any underlying etiologic disease and the second stage is surgery.

I. **Emergency treatment**

When synergistic genital gangrene is suspected, aggressive treatment should be instituted immediately. The course of the disease can be shortened by vigorous medical and surgical intervension from the time the disease is suspected. Broad spectrum antibiotic coverage including both aerobic and anaerobic organisms, should be started after appropriate smears and cultures are obtained from the zone of infection. In one series Martti[12] found the initial use of Gentamicin combined with either Clindamycin or Chloramphenicol gave encouraging results. Ampicillin is suggested as an addition to this basic regimen to cover suspected enterococci, if the source of infection is genitourinary.[6] Bahliman et al[2] in another study found most organisms to be sensitive to Chloramphenicol and Metronidazole. Efem[5] managed 21 patients with Fournier's gangrene with Amoxicillin/Clavulanic acid and Metronidazole in addition to daily topical application of honey to the gangrenous scrotum. Fahal[6] recommend the use of cotrimoxazole alone to be a drug of choice in a small

study where the commonest isolated organism was Proteus. In this study a combination of chloramphenicol and metronidazole orally or parenterally for a duration between 7 to 14 days cleared the infection. Patients with prominent systemic manifestation were initially started on parenteral antibiotics with intravenous fluid until the acute stage of disease subsided.

The debridement of nonviable tissue following adequate resuscitation remains the mainstay of treatment.[3,4,12] However there are two schools of thoughts regarding surgical debridement. These are:-

[1] Limited debridement[3,7]. At first excision of necrotic tissue only is undertaken at the initial operation in an effort to preserve as much skin and subcutaneous tissue as possible. Advocates of this approach argue that the degree of tissue loss and the magnitude of subsequent reconstructive procedures may be reduced. However, this approach generally necessitates repetitive excision of necrotic tissue before the margins between viable and nonviable tissue become demarcated.

[2] **Radical debridement:**

Randall in 1920, where involved skin, subcutaneous tissues, fascia, and structures of its wall, come away in a fascial mass. Therefore initial debridement extensively done should extend generously to healthy tissue.

Proponents[1,5,6] of this approach believe that the only way to control the fulminant and rapidly progressing infection and gangrene is by aggressive debridement. However, Akinola[1] et al experienced a high mortality of 33% in their series. To change the pattern in the study both limited and radical debridement were used, depending on the extent of the gangrene. Thereafter the daily dressing continued in the ward to keep area as clean as possible and promote the healing process. The duration of the healing process depends among other factors, on the extent of gangrene. With a small area, a few weeks are sufficient but in large defects several weeks will be required to complete the healing. Generally long hospitalization required for successful management.

The role of suprapubic cystostomy in extensive genital gangrene is unclear. However, if the urethra is disrupted, suprapubic cystostomy may be necessary. In case where gangrene arises from a colorectal source, a diverging colostomy is often required and drainage of the ischiorectal space involved is mandatory.[7,12] Orchidectomy is not routinely needed since necrosis of the testes is rare due to the rich blood supply via the spermatic vessels. Penectomy should be avoided.[12] In patients with hydrocele hydrocelectomy was done

II: **Reconstructive procedures**

Loss of genital skin may take various forms from a small areas of excised tissue to the of total denudation of penile, scrotal-perineal, and even lower abdominal region. Small areas skin loss can be expected to epithelialize over a period of several weeks. On the other hand, if for any reason more conservative treatment is indicated, even small remaining part of scrota skin will form a new scrotum in six to eight weeks or longer. Remarkably large defects in the perineal scrotal area can be covered by split thickness skin grafting, mobilization of scrotal or a combination of both techniques. Otherwise in most instances the scrotal skin can be mobilized and sutured secondarily over the raw area.[7]

In cases of more complete loss of perineal scrotal tissue, reconstruction can be accomplished in one or two stages by means of an inner-thigh pedicle flap supplied by cutaneous and myocutaneous branches of the deep external pudendal artery, the anterior branch of the orbturatory artery, and the medial circumflex femoral artery.[18] Penile reconstruction is generally best accomplished by means of split thickness skin grafting.[9] All distal penile skin should be excised prior to application of the split thickness skin graft so that

distal lymphatic edema may be decreased. In his series Flamigan[7] reports best functional as well as cosmetic result with this method.[7,18] In case of the denuded testes may be implanted into the subcutaneous tissue of the medial thighs, with subsequent reconstruction of a scrotal sac by mean of release of medial thigh tissue.[18] Alternatively they may be covered in one stage by means of skin mobilization from the perineum, thighs, or scrotal remnant.[5] In this study two patients were managed by split skin grafts. The results were good, and acceptable cosmetic wise. Six patients underwent secondary suture. The rest of two patients underwent only debridement. In five patients with hydrocele, hydrocelectomy was done.

CONCLUSION

Fourniers gangrene is an aggressive disease affecting the peritoneum. Clearly it can no longer by considered idiopathic in its origin, as infection can be localized to a cutaneous, urethral, or rectal source. It presents in a wide age range and can have an indolent onset, thus requiring a high index of suspicion. Initial early institution of broad spectrum antibiotics, pending modified antibiotics, therapy after culture isolates and radical debridement as the gangrene establish, constitute the main stay of treatment lines. Often

long hospitalizations is required in managing these patients. However, reconstructive surgical procedures help shorten this duration.

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MANAGEMENT OF CHRONIC OSTEOMYELITIS

SUMMARY

Twenty patients with chronic osteomyelitis were attended and analyzed by the author in the period between September 1995 and January 1996 in the Orthopaedic and trauma department of Muhimbili Medical Center. There were 15 males and 5 females giving a ratio of 3:1. The age range was from 1 to 60 years. The mean age was 22 years. Tibia and femur were the most frequently affected bones followed by humerus, ulna and radius.

Hematogenous chronic osteomyelitis was recorded in 8 patients, while the disease was a complication of open fractures in 8 patients, and the remaining four patients had osteomyelitis as a result of bone Surgery. Chronic discharging sinuses, pathological fractures, contractures and joint ankylosis were the complications encountered. Surgery, inform of sequestrectomy or saucerization supported with antibiotics as guided by Culture Sensitivity results was the mainstay of treatment. ~~While~~ One patient underwent amputation of the leg.

INTRODUCTION

Osteomyelitis refers to an infective process of bone. Chronic Osteomyelitis is a very serious condition that every Doctor should take all measures to prevent. It is a common problem in developing countries, like Tanzania where

ignorance, poverty, late diagnosis, improper management of acute osteomyelitis and open fractures are among the contributing factors[8,11]. It is estimated that as many as 15 to 30% of chronic Osteomyelitis cases, are complicated by persistence of infections[10]. This occurrence is seen in patients whose infection is hematogenous in origin, or introduced at the time of surgery or trauma. In the urban areas where Vehicular accidents are many, chronic osteomyelitis is frequently seen as a complication open fractures of grade 2 or 3 resulting of road traffic accidents.[16] However, the treatment of chronic osteomyelitis is always long term and very expensive, and sometimes it can be lifelong. Often both the patient and the attending surgeon get frustrated when recurrences occur.[9,11] For this matter emphasis should be dedicated on preventive measures than cure, so that all efforts in terms of facilities and skills should be directed to the prevention of chronic osteomyelitis.

CASE REPORT

T.R (IP No 704818), was a 17 year old female referred from one of the peripheral hospitals where she was initially admitted in January, 1994 after presenting with a one week history of swelling of the left leg associated with fever and inability to use the limb. She was initially treated with ampicillin for 10 days and surgery inform of incision and

drainage. She got relief from treatment given but four months later she was readmitted after presenting with swelling of the left leg, a discharging sinus, pain and inability to use the limb. A diagnosis of chronic osteomyelitis and pathological fracture was made and confirmed radiologically. Thereafter she was referred to Muhimbili Medical Center and admitted to the Orthopaedic and trauma ward in May 1994.

Clinical examination revealed a health young girl afebrile, not pale and not jaundiced. Cervical, axillary, inguinal lymph nodes were not enlarged. Her blood pressure was 100/60 mm/Hg and had a pulse rate of 72 beats per minute regular of normal volume. The above findings gave an impression of a chronic osteomyelitis and pathological fracture of proximal third of the left tibia.

The following investigations were done.

(1) Xray of the left tibia and fibula anteroposterior and lateral views showed evidence of osteomyelitis of the left tibia by the presence of a large sequestrum and involucrum on the proximal third of the left tibia bone, with a pathological fracture.

(2) Full blood picture revealed a haemoglobin of 10.3g/dl with a normal blood film and raised ESR of 30mm/hr.

(3) Blood sugar was 5.0mmol/L and mantoux test was negative

(4) Pus swab for culture and sensitivity was also taken.

The patient was initially managed with Gentamicin, daily Eusol dressing, and the limb was immobilized with above knee pop back slab pending surgery. One week after admission she was operated. Under general anaesthesia a tourniquet was applied on the Left thigh, the left limb was then cleaned using iodine and spirit from the foot to the thigh. Drapes were applied such as the leg was left exposed.

A longitudinal incision about 8 cms was made over the tibia anterolaterally extending below the knee joint. Saucerization was done, manipulation for correction of the deformity was done the AO external fixator frame applied. Compressive gauze applied and secured with crepe bandage. Postoperatively the patient was maintained on oral erythromycin, guided by culture results, pethidine was also prescribed in the first 24 hours. Limb to rest on pillows. Dressings were changed on the third post operative day. Three weeks after operation she started ambulation with crutches and she continued dressing with vaseline gauze on alternative days till a healthy granulation tissue was evident and skin defect had contracted significantly. The last serial control x-rays were taken in July thereafter she was discharged home. Then she was seen at our outpatient clinic after every two months. During the follow ups she was found to have an obvious varus deformity of the left tibia and fibula and clinical shortening of the left by leg of about 1.5cms. Control x-rays showed the

tibia has healed very well despite the deformity it has developed. Therefore she was planned for corrective osteotomy. She was prepared for operation, in November 1995 operation was performed. Patient under G/A, tourniquet applied on left thigh, cleaning and draping done. Longitudinal incision done followed by segmental fibula osteotomy and wedge osteotomy of proximal 1/3 of left tibia. Varus deformity corrected and held in position with an AO external fixator applied in T form. The wound was irrigated with normal saline, a drain was inserted and wound closed in two layers. Post operatively Injection pethidine 50mg in 8 hourly for 24hrs was given, ampiclox 1gm 8hourly daily elevation of the left leg on pillows. Control x-ray was taken revealed that, the varus deformity was well corrected. On the tenth day stitches were removed. One month after operation, she was mobilized on crutches after control x-ray showing presence of callous formation. She was discharged six weeks after operation, thereafter she was seen at our clinic after every two weeks where by she was found to be walking in the normal gait.

RESULTS

Tabular summary of other patients

Table 1: Age and sex distribution of patients (n=20)

Age in years	Male	Female	Total
1 - 10	2	1	3
11 - 20	4	4	8
21 - 30	5	-	5
31 - 40	2	-	2
41 - 50	2	-	2
Total	15	5	20

Male to female ratio was 3:1. The mean age was 22 years.

Table 2: Anatomical distribution of cases studied

Bone	Numbers of patients
Femur	7
Tibia	6
Humerus	4
Ulna	3
Radius	3
Fibula	3
Total	26

Note: Some patients had more than one bone involved.

Table 3: Distribution of etiological factors of cases studied (n=20)

Etiological factors	No of patients
Hematogenous	8
Open fracture	8
Bone surgery (ORIF)	4

Note: ORIF = Open reduction and internal fixation.

The infection is limited at each end of the long bone by the metaphyseal-physeal plate junction. It may rupture through the periosteum into the adjacent soft tissue and ultimate to the skin, where it produces a draining sinus tract. The sequestrum may be extruded if the sinus is large enough to extrude it. Subsequent healing may occur with obliteration of the abscess cavity by fibrous tissue. However, this situation rarely occurs, the sequestrum being too large for complete extrusion. Even with complete extrusion the rigid nature of bone prevents collapse of the abscess cavity.

It is estimated that as many as 15 - 30% of acute osteomyelitis cases are complicated by persistence of infection.[6,21,22] This occurrence is seen in patients whose infection is hematogenous in origin or introduced at the time of surgery or trauma. Factors involved in the inability to cure acute infection include delay in diagnosis, inappropriate or inadequate antibiotics and insufficient surgical drainage. Frequently with chronic infection, a cortical sequestrum forms. This occurs when local osteal blood supply is compromised by surrounding infection and vascular thrombosis leaving on island of a vascular necrotic and infected bone.[10]

Certain bacteria posses virulence factors that allow the persistence within bone despite adequate therapy. One of these factors is the glycocalyx, which forms glyco-enclosed microcolonies with bacteria, bacteroides species and

pseudomonas species.[10,13]

Another mechanism of persistence is poor host response. Certain immunologic defects allow for persistent or recurrent bone infections. The classic example is sickle cell anaemia, in which salmonella osteomyelitis has been recognized as a complication infection. Persons with sickle cell anaemia have specific opsonizing defects for salmonella.[7] Foreign bodies[11,12] (eg orthopaedic stabilization devices) may introduce bacteria into sterile bone or may lead to persistence of infection when located in an area of infected bone. Some organisms grow slowly in bone and are characteristically associated with a more indolent course. Include: Mycobacterium tuberculous, brucella species and various fungal. In addition, osteomyelitis associated with vascular insufficiency often presents as a slowly progressing infection.

CLINICAL FEATURES AND DIAGNOSIS

It is difficult to define the true incidence of symptoms and clinical findings associated with chronic osteomyelitis since the origin of infection is quite variable. However, there are certain generalizations that can be made. Chronic bone infections are frequently obscured by other physical findings or present with little or no findings altogether. Hematogenous osteomyelitis, acute disease presents with fever,

local tenderness, and swelling in over 50% of cases.[10,21] On the other hand, sinus tracts and drainage are much more frequent in recurrent illness. Osteomyelitis that complicates fractures may result in nonunion.

The diagnosis can be reached purely on clinical grounds, however the following investigations are important.[17,21]

1. **Hematology**

   ESR is usually high but not specific to bone infection WBC results are difficult to interpret, they may be leucocytosis or normal. However normochromic, normocytic anaemia is often present.

2. **Bacteriology**

   Pus swabs from pus discharging sinuses, abscesses or fistula may provide a satisfactory clue as regards the offending micro organism but negative cultures are not uncommon. One study in this field staphylococcus aureus was isolated in about 7% of cases.[18] The incidence due streptococcus pyogenes is decreasing.[21] Gram-negative microorganisms like hemophilus influenzae has been isolated and salmonella species in patients with sickle cell disease are also isolated.[7]

3. **Radiology**

   a) Simple x-rays are sufficient to obtain a proper diagnosis and plan treatment, the cavities and sequestra being clearly demonstrated.

b) Tomography will give information on the site, number and layout of the sequestra and their place in connection with the cortex.

4. **Histopathology**

Biopsy is quite a useful armamentarium, particularly in situation in which osteomyelitis is to be differentiated from a neoplastic process.

TREATMENT

Chronic osteomyelitis is a disease that can cause frustration to both the patient and the surgeon alike because of it's protracted course and poor results or recurrence after treatment. Prevention is better that cure in case of this disease, among others. Acute osteomyelitis should be treated vigorously once it is diagnosed, similarly the basic principles in management of open fractures should be strictly adhered to. The basis of management lies on the realization that the disease is one of ischaemia more than infection.[8] The organisms thrive in avascular bone and scar tissue and frequently the scar tissue acts as an impenetrable membrane to antibiotic therapy.[9,12,13] Effective treatment therefore consists of: Excision of all dead bone, necrotic tissue and scar tissue, effective antibiotic therapy and elimination of dead space when the wound is closed.

1. **General management**

This includes improvement or correction of anaemia and blood volume. Chronic surface lesions do not heal in presence of anaemia.[12] Hypoproteinemia and vitamin deficiency particularly vitamin C should be corrected. Splintage or immobilization in plaster of paris and rest are adjuvant treatment of chronic osteomyelitis. Immobilization minimizes the chances of disseminating the infection reducing deformity and pathological fractures.

2. **Surgical treatment**

The main-stay in surgery of chronic osteomyelitis is radicality, surgery should be as radical as possible without regard to remaining defect.[8] Saucerization and sequestrectomy are the keystone to the management. All scar tissue including the sinus tract and dead bone must be removed. Identification of the dead bone is essential so that the radicality is combined with maximum conservation of the skeletal continuity.

Patients with considerable bone or soft tissue loss. Bone grafting or skin grafting is advisable.[1,4,14,15] While patients with pathological fractures can be managed by saucerization and external fixation.[8] Amputation[22] may be indicated by years of invalidation with no prospects of improvement in future and in case of malignant change. In this study we had one patient who requested amputation of the leg due to uselessness of it.

3. Chemotherapy

In the treatment of chronic osteomyelitis antibiotics can in no way ~~may~~ replace surgery regardless of the method of administration or type of antibiotic.[2] This is because chronic osteomyelitis is more of ischaemia than infection, thus antibiotics are used to support surgery. The chemotherapy should be prolonged, systemic and locally applied in order to control bacterial growth and prevent persistent infection. Local antibiotic use when added to systemic antibiotic is claimed to accelerate the formation of new bone and localization of sequestra.[16,21] Antibiotic irrigation of wound can be done in an open wound or in the form of closed irrigation suction technique.[21] Commonly used drugs are: Cloxacillin, Chloromycetin, Penicillin, Tetracycline, Erythromycin, Ampicillin, Lincomycin and Gentamicin.

PREVENTION

Chronic osteomyelitis is better prevented rather than cured. From this study about 80% of all patients had chronic osteomyelitis secondary to either acute hematogenous osteomyelitis or open fractures. Therefore an important aspect in the prevention of chronic osteomyelitis is the early consultation of patients to hospital, correct diagnosis and treatment of acute osteomyelitis and open fractures. The place of surgical exploration and drilling in acute osteomyelitis is

still controversial. While some authors[8,18,20] advocate surgical decompression as part of treatment if pus is obtained on aspiration. Others[3,12] suggest surgical exploration as being mandatory in all cases of acute osteomyelitis regardless of whether there are clinical signs of abscess or not.

For open fractures, the basic principles in the management should be strictly adhered to these consist of:-

a) Improving first AID and Ambulance system so as open fractures should initially be properly managed to minimize wound contamination.

b) Early and proper surgical toilet of open fractures. Surgeon should observe maximum sterility and strictly adhere to the aseptic technique during bone surgery. From this study it was observed that all patients who had chronic osteomyelitis secondary to grade 2 or 3 open fractures had surgical toilet done after 8 hours of injury, this delay of time favored bacterial growth.

CONCLUSION AND RECOMMENDATIONS

During the past 30 years, the investigative technology and treatment of chronic osteomyelitis have advanced impressively. Inspite of all above achievement still in our community chronic osteomyelitis is a bi problem, therefore all measures should be taken to prevent acute osteomyelitis and open fractures being complicated to chronic osteomyelitis

as discussed above. Also there is a need of educating the mass about the disease so that they can early consultant medical service.

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