I

A DISSERTATION OF TEN SURGICAL CONDITIONS

BY

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DKANY

A DISSERTATION SUBMITTED IN PARTIAL FULFILMENT FOR THE AWARD OF THE DEGREE OF MASTER OF MEDICINE (SURGERY) OF THE UNIVERSITY OF DAR ES SALAAM

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PREFACE

This dissertation conforms to the requirements for the award of the degree of Master of Medicine in Surgery in accordance with the regulations of the University of Dar es Salaam. It consists of ten studied surgical conditions which have been divided into three parts, Part one consists of five conditions pertaining to General Surgery, part two has two conditions in Urology, and part three has three conditions in Orphopaedics and Trauma. All these cases were studied during the three years of residence in Surgery. In each condition an appraisal of one case has been done in detail followed by a summary of the characteristic features of all the patients in the study population and finally the relevant literature on the subject has reviewed.

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This is to certify that the ten surgical conditions presented in this dissertation were managed by Doctor Peter Clement Adam Nyagawa in the course of his Postgraduate training in General Surgery under my direct or delegated supervision

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DECLARATION

I solemnly do declare that this dissertation is my own work based on patients whose management I have taken an active part, and that this very work has not been submitted and is not currently submitted for any other academic award of any other University.

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DR. PETER C.A. NYAGAWA

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MUCHS, March 1994 **DR. PETER C.A. NYAGAWA**

DEDICATION

To my parents, Adam and Beatrice for their patience, immense love, tireless commitment, and above all for the difficulties endured during my upbringing and all the way through the jungle, leading into this mighty noble profession.

To Dr. Rubera Mafwiri, a honest friend and a man of admirable ability, principles, devotion and courage I'll live to emulate. And, to the Junior doctors whom this work is a real challenge, aiming to inspire the will power.

DR. PETER C.A. NYAGAWA

TABLE OF CONTENTS

ITEM		PAGE
	SECTION 1: GENERAL SURGERY	
1.1.	VOLVULUS OF THE SIGMOID COLON	1
1.2.	TRAUMATIC RUPTURE OF THE SPLEEN IN ADULTS	21
1.3.	THORACIC INJURIES AS SEEN AT MUHIMBILI MEDICAL CENTRE	39
1.4.	MANAGEMENT OF EMPYEMA THORACIS	59
1.5.	BURN INJURY IN ADULTS	85
	SECTION 2: UROLOGY	
2.1	MANAGEMENT OF BENIGN PROSTATIC HYPERLASIA	112
2.2.	MANAGEMENT OF FOURNIER'S GANGRENE	138
	SECTION 3: ORTHOPAEDICS AND TRAUMA	
3.1.	CHRONIC OSTEOMYELITIS IN ADULTS	160
3.2.	MANAGEMENT OF HIP FRACTURES AS SEEN AT MUHIMBILI MEDICAL CENTRE	186
3.3.	TUBERCULOSIS OF THE SPINE AS SEEN AT THE MUHIMBILI MEDICAL CENTRE	213

SECTION ONE
GENERAL SURVERY

1.1: VOLVULUS OF THE SIGMOID COLON

SUMMARY

Between October 1991 to June 1993 a total of 11 patients with Volvulus of the sigmoid colon were attended by the author while in surgical firms II and III.Only one was a female; their ages ranged between 17 and 70 years, whereas 63.6% constituted the age range between 21 to 40 years.

Abdominal pain, distension and constipation were the main presenting features. With the exception of one patient who had gangrene, thus managed by resection and a double barrel colostomy the rest of the patients underwent resection and primary anastomosis. Both were covered with a combination of parenteral antibiotics i.e Flagyl and Gentamycin or Chloramphenicol from the perioperative period to 7 days post operative. One patient died on the 5th postoperative day after apparently showing good postoperative progress. He had an underlying myocardial disease which resulted into Cardiac failure.The average period of hospitalization was 11.4 days with a range of 6 to 20 days.

In this study ten patients had no signs of gangrenous bowel and fared well following resection and primary anastomosis. Only one patient developed wound sepsis, there was no intra-abdominal pus collection.

INTRODUCTION

Sigmoid Volvulus results from the rotation of a segment of the bowel about its mesenteric axis sufficient to produce partial or complete obstruction of its lumen. The understanding

of the natural history of the sigmoid volvulus has been attempted since ancient times. The authors of the Papyrus Ebers from ancient Egypt wrote that either the volvulus spontaneously reduced or the sigmoid "colon rotted"[1] Von Rokitansky in the 19th Century described the various forms of volvulus of the colon. It was a common cause of obstruction among Greeks and Romans.[2]

The incidence of this condition varies geographically. In Tanzania Shija noted that only 4% of all cases of intestinal obstruction were due to sigmoid volvulus.[3] The disease is rare in the U.S.A whereas the rate is higher in areas such as Russia, India, Iran, Norway, East, West and parts of North African countries.[2] Although sigmoid volvulus is frequently found in elderly institutionalized or chronically constipated persons,[4] in this study most patients were between 21 to 40 years (Table I). A male preponderance has been noted world wide.[1–3,6–7,8,11,18]

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 primarily or delayed. Arguments have persisted about the need for staged procedures.[4] A review of the literature on the subject is discussed with special emphasis in our environment.

CASE REPORT

S.I.; IP NO 741356, a 25 year old male, referred to Muhimbili Medical Centre from one of the peripheral hospitals of Dar es salaam, was admitted in the surgical firm II on the

10.03.93 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 fasting period. Pain started over the hypogastrium, gradually increasing in intensity and after one day it became generalized. His abdomen was gradually distending and by the time of admission, he was having difficulty in breathing.

His appetite was good. He had not experienced any nausea, but had vomited twice. Over the previous three days he had not passed stools, whereas no flatus was passed for nearly 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 the history of similar attacks in the past. The patient denied smoking habits or use of alcohol. Cassava, rice and maize diet were the main staple food. He worked as a petty businessman; the rest of his family members were all fine.

General examination revealed a well built man who appeared ill looking and in pain. He was not dehydrated or pale but was slightly dyspnoeic. His temperature was 37°C, had no jaundice and was well oriented to time, place and person. His pulse rate was 80 beats per minute regular and of good volume, the blood pressure was 110/70 mmHg.

The chest was clear. Abdominal examination revealed gross distension all over, there was no visible peristalsis. Had a tense and tender abdomen though no guarding was appreciated. No mass could be palpated. Had a tympanitic percussion note all over the abdomen, there was no evidence of ascites. Bowel sounds were greatly exaggerated.

Rectal examination revealed an empty rectum, no masses. There was no blood on the gloved finger.

Plain abdominal x-rays showed a markedly distended sigmoid colon with thickened but smooth walls. The small bowel was not distended. There were no fluid levels seen on the erect films.Hemogram estimation of haemoglobin recorded a value of 12.5 g/dl.

On the basis of the history, clinical findings and radiological findings a working diagnosis of volvulus of the sigmoid colon was made. While in the ward, passage of a rectal tube was attempted so as to relieve the symptoms to the patient, however the attempt failed and was unrewarding. The patient was then booked and prepared for an emergency laparotomy. The abdomen was shaved and cleaned with antiseptic solution. A nasogastric tube was inserted and intravenous drip of D/Saline was started. Blood samples for grouping and cross match were taken.

General anaesthesia was given and the patient well positioned supine on operating table. Disinfection of the abdomen using Iodine; Spirit and draping was then done. Through a midline incision the abdomen was opened. Immediately after the peritoneum was opened the sigmoid colon eviscerated due to distension. The twist at its base was through 360_o anticlockwise, thus complete obstruction of the colon was obvious though the loop appeared not gangrenous. Detorsion in a clockwise direction was done, then the bowel deflated from below using Ryles flatus tube introduced by an assistant. The viability of the sigmoid loop was finally confirmed. The length of the redundant colon measured about 25 cm. The limbs of the sigmoid loop were close together whereas the mesosigmoid

was noted to be long and freely mobile. The rest of the laparotomy findings were normal.

The redundant colon was clamped using bowel clamps. Resection of the redundant colon was then done and continuity restored primarily by end to end anastomosis in one layer using 2/0 atraumatic interrupted silk sutures. No fecal spillage occurred. Approximation of the mesosigmoid and repair was done using chromic 2/0. No tension was noted at the anastomotic site. Thorough lavage was done using copious amounts of warm saline. No drain was inserted, the abdomen was closed in two layers using catgut 2/0 and Vicryl O to the peritoneum and fascia respectively. The skin was left open. Intra– operatively he was not transfused, the estimated gross blood loss was less than 300 ml. Post operatively, the patient had his pulse, Blood pressure, temperature, respiration rate and urine output observed and charted hourly during the first postoperative 6 hours when he was declared stable, there after taken 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. I/V fluids – D/Saline 3L/24 hours
2. Antibiotics: Chloramphenicol 500mg I/V 8 hourly and Flagyl 500mg I/V 8 hourly. Both were given for 7 days.
3. Injection Pethidine 100mg I/M 8 hourly PRN for the first 24 hours.
4. Nursing to be done in a propped up position.

His post–operative course was uneventful since his vital signs remained stable, had no evidence of early wound sepsis and the abdomen was soft. Furthermore his

general condition fared well. On the third post–operative day the bowel activity had resumed as evidenced by the presence of bowel sound, he was started on ambulation and was able to tolerate oral sips of black tea and water. By the 4th day he had full bowel action, the intravenous drip was discontinued. He started liquid oral diet.The wound site had remained clean, thus secondary suture was done on the 11th day. Was discharged home on the 13th day with a haemoglobin of 12 g/dl. The stitches were removed a week later. He was seen at Surgical clinic a week after discharge and thereafter seen again twice at 2 weeks intervals. He has remained well ever since and gained weight.

The features of the other patients reviewed in this study are summarized in the tables below:

TABULAR SUMMARY

TABLE I: DISTRIBUTION OF THE CASES BY AGE AND SEX (n = 11)

AGE	SEX		
	MALES	FEMALES	TOTAL
10 – 20	1	0	1
21 – 30	5	0	5
31 – 40	2	1	3
41 – 50	1	0	1
51 – 60	0	0	0
61 – 70	1	0	1
TOTAL	10	1	11

- The youngest was 17 years and the oldest 70 years
- 63.% were between 21 – 40 years

TABLE II: CLINICAL FEATURES IN ORDER OF OCCURRENCE IN THIS STUDY

SYMPTOMS	NUMBER OF PATIENTS
Abdominal pain	11
Constipation	10
Abdominal distension	10
Vomiting	7
Dehydration	6
Dyspnoea	1
Fever	1

TABLE III: INTRAOPERATIVE STATE OF THE BOWEL LOOP

STATE OF BOWEL	NUMBER OF PATIENTS
Viable	10
Non viable	1
TOTAL	11

TABLE IV: OPERATIVE PROCEDURES

PROCEDURE	NUMBER OF CASES	DEATHS
Resection with primary anastomosis	10	1
Detorsion and fixation	0	0
Sigmoidoscopic detorsion and elective resection	0	0
Emergency operation detorsion and elective resection	0	0
Hartmann's procedure	0	0
Emergency resection and double barrel colostomy	1	0
TOTAL	11	1

TABLE V: POST OPERATIVE COMPLICATIONS

COMPLICATION	NUMBER OF CASES
Wound sepsis	1
Intraabdominal pus collection	0
Peritonitis	0
Shock	0

DISCUSSION

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. Sigmoid volvulus tends to occur in people with floppy redundant colons and narrow mesosigmoid attachments.[1,2,4] It is probable that congenital and acquired factors work together. In this study the mean age was 33.8 years, the youngest being 17 years old (Table I).

PATHOGENESIS

Two things, whether congenital or acquired must be present before volvulus of the sigmoid colon can be formed:–

(1) A redundant sigmoid with a narrow base. The two limbs are closely approximated forming a narrow stalk.

(2) A long freely mobile mesocolon.

It appears that the two are often coexistent in the same patient.[4,6,8,9] According to

Sutcliffe[12] the bulky sigmoid stretches its mesentery, narrowing its base in the process. The narrowing is accelerated by scarring in the mesentery caused by minor but repeated attacks of twisting. The direction is often said to be anticlockwise as in all of our study patients, but there are several cases with clockwise twist reported[4,8,12] The degree of the twist varies from 180° to 720°. According to Groth[13] as the bowel twists around the mesenteric axis, it also undergoes an axial twist in the direction of the bowel. The axial torsion becomes distributed to both ends of the loop leading to obstruction.

Peristalsis forces gas and fluid into the loop which remains trapped as the proximal end acts as a valve. The loop distends and its intraluminal pressure increases and as it exceeds that of the blood supply to the loop, infarction and gangrene set in. However the sigmoid colon can withstand high intraluminal pressure, thus even though a closed loop obstruction is present, the blood supply usually remains adequate for a few days before it is compromised. This had been the case in ten of the studied patients as seen in table IV. With time, and as more and more twisting beyond 180° occurs, arterial obstruction occurs and anoxia of the bowel becomes severe.[14] This promotes excessive fluid to extravasate into the bowel wall and lumen. This fluid shift compounded with transperitoneal absorption of toxins may lead to severe shock. The process becomes irreversible and spontaneous reduction fails.[14,15] As would be expected, ischemic damage is more severe at the neck of the volvulus where axial torsion in more severe[8,9,15] Forward[16] has noted that detorsion of sigmoid volvulus is followed by watery mucoid discharge per rectum resulting in hypokalemia which can be severe.

CLINICAL FEATURES

Volvulus of the sigmoid colon may present in a variety of ways. There may be an acute episode of sudden complete obstruction and rapid development of gangrene, there may be multiple subacute attacks or there may be a chronic volvulus. Clinical picture may be that of chronic constipation associated with bouts of colicky pain and distension, as was the case in majority of our patients (Table III). Occasionally there may be self–relief by the passage of huge quantities of gas and liquid stools.[17] This was not encountered in our patients.

As the disease progresses, the chances of spontaneous remission becomes remote. Often by the time the patient presents himself for medical attention, the abdomen has already been grossly distended and it is tympanitic. At such stage respiratory embarrassment is frequently obvious. Hypovolemic shock may occur following dehydration and exudation of blood within bowel lumen i.e third space. Bowel necrosis followed by perforation and peritonitis is a late manifestation. In the elderly, obstruction usually develops over a long period ranging from several days to even a week. This was noted in one of our study patients whose age was 70 years old.

DIAGNOSIS

Many patients will have had previous attacks in the past, some relieved spontaneously while others required some procedure to relieve them. In this study, five out of the eleven patients presented with such a history. They had been relieved by rectal tube in hospital. The Acute Fulminating type is usually found in the young patients, usually in severe pain,

simulating a perforated viscus or strangulated bowel. It is characterized by explosive onset with nausea, vomiting, severe abdominal pain and balloon like abdominal distension. There is contouring of loops of bowel, gurgle sound on auscultation of the abdomen and an empty rectum on rectal examination.

The subacute or chronic type is the commonest occurring usually in the older patient. Usually there is history of previous episodes of abdominal pain and associated constipation and distension. Occasionally there can be nausea and vomiting. Plain abdominal X–ray films are necessary in confirming the diagnosis. The sigmoid appears enormously dilated with two fluid levels at its base (in erect). The walls of the loop are thick with loss of haustration. There is usually an absence of gas in the rectum, the walls of the dilated loop may be seen converging in the left iliac fossa simple x–ray films are usually enough to confirm the diagnosis, but when is doubt, Barium enema will reveal the presence of the volvulus as twisted loop with mucosal spiral pattern at the site of the torsion. In this study the plain abdominal x–rays were so diagnostic, hence the need for Barium enema was not that much necessary especially considering the frequent shortage of this diagnostic method at Muhimbili Medical Centre. When gangrene develop the features which may be suggestive include the following:–

(1) Features of peritonitis i.e abdominal guarding and tenderness.

(2) Raised white blood cell count.

(3) Absent bowel sounds

(4) Shock and an ill looking, toxic patient.

(5) Presence of free gas under the diaphragm on plan x-ray.

(6) If sigmoidoscopy is done therapeutically mucosal inflammation, discoloration or ulceration of mucosa/bowel lumen may be suggestive of gangrene.

MANAGEMENT

The management of volvulus of the sigmoid colon has been a controversial subject.[8,9,10] The aim of treatment of sigmoid volvulus however is to achieve reduction and decompression of the twisted colon, to prevent recurrence and to lower the associated high mortality rate. Whereas it is well recognized that spontaneous reduction of the volvulus may occur in some patients, the chief danger from volvulus is the development of impairment of the blood supply with necrosis of the bowel wall and perforation. Although sigmoid resection is the definitive treatment for sigmoid volvulus, two modes of treatment are employed:-

(1) Conservative (Non-operative)

(2) Surgical (Operative)

1. **Conservative Methods:**

Non operative reduction of sigmoid volvulus does not constitute adequate treatment. It is a temporizing measure that allows implementation of definitive operative therapy under improved circumstances.[18,19] The high rate of recurrence and high mortality rate associated with nonoperative reduction have been well-documented.[8,10,18]

The methods used include Barium or Saline enema, proctosigmoidoscopic and a rectal tube reduction and decompression. Recently, the fiberoptic colonoscope has been used

in reducing sigmoid volvulus. Only small series of cases treated with this instrument have been published, however the approach seems to have promising results, although great care and experience are necessary.[20,21] The method should be limited to patients in whom gangrene is not suspected. Usually a long and large rectal tube is inserted after colonoscopic reduction of the volvulus to splint the sigmoid colon for 24 to 48 hours to allow bowel to regain its tone and avoid early recurrence.[20] Non operative methods of treatment are valuable only in the subacute volvulus without gangrene, thus accuracy of diagnosis is highly demanded in a non operative approach.

Once non-operative reduction has been advised elective sigmoid resection should be undertaken preferably within the same hospitalization because of the high morbidity and mortality following recurrent.[18] When non operative reduction cannot be achieved, laparotomy is mandatory.

2. **Operative Methods**

Operative intervention in patients with sigmoid volvulus has taken many forms. The two main indications for emergency procedures are; failure of non-operative procedures and the presence of gangrene.[17,18] This is the only definitive treatment of sigmoid volvulus and remains the only treatment in acute disease and where gangrene is suspected. The viability of the sigmoid colon primarily determines mortality. Large series throughout the world record mortality of 52.9% when bowel is non viable whereas the mortality is 12.3% when the bowel is viable.[21]

Most surgeons prefer to operate on a prepared bowel in "fear" of infection. Different

modifications have been adopted in an attempt to minimize peritoneal soiling during surgery. All agree that gangrenous colon must be resected, but a volvulus of viable colon can be treated by several methods.

I. **When bowel is Viable**

(i) Operative detorsion with or without fixation (colopexy).

Advocated for a viable colon, colopexy has also been used but recurrence is a main threat.[20] However operative detorsion is recommended as a preliminary step for an elective sigmoid resection after bowel preparation. Some authors prefer it to be done within a period of two weeks after detorsion.

(ii) Resection and primary anastomosis.

The best operative therapy for sigmoid volvulus is resection. Sigmoid resection has been generally selected as the procedure of choice. Arguments have persisted about the need for staged procedures.[18] If the loop is non viable, it must be resected. If the loop is viable it is still best resected as recurrent volvulus is common and various means of colopexy are relatively unsuccessful.[19]

In this study, ten out of the eleven patients were definitively managed by primary resection and end to end anastomosis restoring the continuity of the colon (Table V). The outcome has been good and encouraging. This is probably attributable among other factors to the good nutritional status,

good haemoglobin value and preoperative adequate resuscitation measures. Similarly the absence of gangrene and the median age which was 38.8 years favored the good outcome after primary resection and anastomosis. One patient died on the fifth postoperative day for a non surgical condition. He had developed cardiac failure which was precipitated by an underlying myocardial disease. Also there were least complication rate as seen in table VI.

In minimizing or possibly avoiding the high risk of developing post operative infection, all the eleven patients in this study has had perioperative and postoperative antibiotics comprising of parenteral combination of Flagyl with Gentamycin or chloramphenicol. However in order to minimize anastomotic dehiscence other authors advocate one of the following methods:-[22,24]

(1) Exteriorization of the anastomotic site.

(2) Splintage of the anastomotic line with a large rectal tube.

(3) Use of omentum as a pedicle graft at the anastomotic site.

Neither of the above methods were attempted in this study.

II. **When gangrene is present**

A variety of procedures have been recommended. Primary resection and anastomosis with an additional proximal colostomy if the anastomosis appear tenuous.[12] This procedure has a high mortality. The other procedures with less

mortality include:–

(i) Paul – Mickulicz operation.

Involves detorsion of the volvulus, exteriorisation and primary resection of the bowel with establishment of a double barrel colostomy. It is not a feasible procedure when the distal stump is too short to exteriorize.

(ii) Hartmann's Procedure.

This is recommended especially when the distal stump is short but later anastomosis may be done with difficulties after this procedure.One of the patient in this study had gangrene (Table IV), thus resection and double barrel colostomy was the treatment offered. The colostomy was electively closed 3 months later.

CONCLUSIONS AND RECOMMENDATIONS

Although surgery on the colon is, while faeces are present, like stepping into a cesspool,[19] in this study patients fared well following primary anastomosis after sigmoid colectomy for volvulus. Based on this experience and other authors[25,26], resections and end to end anastomosis of viable sigmoid volvulus is safe, acceptable, economical and worth doing in our situation.

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1.2: TRAUMATIC RUPTURE OF THE SPLEEN IN ADULTS

SUMMARY

Over a period of 2 years between 1991 to 1993 a total of 40 patients with traumatic rupture of the spleen were admitted in the three surgical firms of Muhimbili Medical Centre. 9 patients were managed by the author while rotating in General Surgery. Out of these managed by the author there were seven males and two females (Table I). The age ranged between 15 and 61 with a median age of 32.2 years. Of the nine patients, 55.5 percent belonged to the age group between 20 and 35 years (Table II).

All the 9 patients had their injuries being caused by blunt abdominal trauma, the commonest being road traffic accidents followed by falls from heights and assault (Table IV). Diagnosis was based on the history and clinical findings. Delayed splenic rapture was only seen in one patient. However, head injury, cut wounds shoulder dislocation and bruises were there associated injuries (Table VI). Five of the patient's luckily had no associated injuries. Splenic laceration and fragmentation were the commonest types of injury. Splenectomy was the treatment offered in all the patients. There were no deaths.

INTRODUCTION

Although the normal spleen is relatively well protected by the rib cage posteriorly and laterally, and softly cushioned by surrounding organs medially and anteriorly it is one of the most frequently injured organs in cases of blunt abdominal trauma. The relative mobility of the spleen at its pedicle also makes it subject to sudden and forceful dislodgement from its peritoneal attachments by contre-coup, resulting in splenic capsular

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and parenchymal tears.[1] When the spleen is ruptured, the patient is at great risk of exsanguinating hemorrhage.[1,2,3] The task of the surgeon then is to secure hemostasis. However, an increased susceptibility to infection and death from sepsis in infants following splenectomy is reported by King and Shumacker.[4] The same complications has since been reported in older children and adults.[5,6] The organisms commonly involved include the encapsulated pneumococci and meningococci, posing a great threat to the life of the patient.[7]

Thus todate bearing the hematological and immunological functions of the spleen, efforts should be made to conserve all or part of the damaged spleen.[2,3,5,7] Despite the fact that there is a change in the concept of the management of splenic injuries from splenectomy to a more conservative approach, splenectomy is still the treatment of choice in centres where the facilities for monitoring the patient's progress are lacking.

CASE REPORT

S.K., IP NO 746190, a 23 year old man was admitted through casualty, shortly after being involved in a road traffic accident. He complained of left upper abdominal quadrant pain, which was aggravated by deep breath and cough. He had also pain on the left shoulder and impaired movements at the shoulder joint. He did not lose consciousness, however admitted to experience dizziness and excessive thirsty a short moment after the accident. On examination he was found to be fully conscious but was anxious and appeared to be in acute distress. Had pallor and appeared dehydrated but was not dyspnoeic. The radial pulse rate was 120 beats per minute, regular but of poor volume. His blood pressure was

90/50 mmHg in the supine position. The abdomen was noted to have bulging flanks but had no bruises. He had marked tenderness on the left upper quadrant. There was clinical evidence of fluid in the abdominal cavity as demonstrated by the presence of fluid thrill. The bowel sounds were normal. The rectal examination was normal, and the examination of other systems was normal except he had an anterior dislocated shoulder which had already been reduced at the casualty.

A diagnosis of splenic rupture was entertained, thus the patient was resuscitated using intravenous Ringers lactate solution and prepared for emergency Laparotomy. A nosogastric tube and a urethral catheter were inserted, blood taken for grouping and cross matching and consent obtained before laparotomy. Under general anaesthesia, the abdominal skin was disinfected using iodine and spirit. He was draped in the usual manner using sterile drape-sheets. The abdominal cavity was entered through an upper midline incision. There was gross hemoperitoneum, scooping out about 2 Litres of fresh blood. This was filtered using sterile gauze and prepared for autotransfusion. On exploring the abdomen, the spleen was found to have multiple linear lacerations involving the anterior aspect and extending to the splenic pedicle. Had a huge hematoma around the pedicle. The spleen was easily mobilized using blunt dissection and delivered through the wound site. It was of slightly larger size than normal. The other organs were found to be normal. The tail of the pancreas was identified and carefully separated from the splenic hilum. The splenic artery and vein were divided between clamps, ligated and transfixed separately using silk 2/0, achieving hemostasis. Autotransfusion was started at this point.

The abdominal cavity was mopped dry of clots and peritoneal lavage done using 2 L of warm saline. The abdomen was closed in three layers using 2/0 catgut, vicryl 0 and 2/0 silk, for peritoneum, rectus sheath and skin respectively.

After getting 4 units of autotransfusion blood, from the immediate postoperative period, the patient was continued on intravenous fluids; Normal saline 3 L in 24 hours. He was sedated using pethidine 100mg intramusculary 8 hourly a total of 3 doses. No antibiotics were given. The vital signs were monitored half hourly until stable, then 2 hourly for the first 24 hours. Urine output was monitored hourly with a range of between 40–60 ml being noted.

The nasogastric tube and urethral catheter were removed after the second postoperative day since the patients condition was stable, the abdomen was not distended and little drained from the nasogastric tube. He was started on sips of black tea and water. On the third postoperative day he had bowel action, the I.V. drip was discontinued, the patient resumed oral feeds. The patient made an uneventful recovery. His haemoglobin value was 8 g/dl a day prior to his discharge. Skin stitches were removed on day seven postoperatively and the patient was discharged home on oral hematinics. Had no problem when followed up at the surgical clinic for a duration of 6 months.

The following is a summary of the features of the nine patients who all had undergone splenectomy for traumatic rupture.

TABULAR SUMMARY

TABLE I: SEX DISTRIBUTION

SEX	NUMBER OF PATIENTS
Males	7
Females	2
TOTAL	9

TABLE II: DISTRIBUTION OF PATIENTS ACCORDING TO AGE AND SEX

AGE	MALES	FEMALES	TOTAL
15 – 20	3	0	3
21 – 25	0	1	1
26 – 30	1	0	1
31 – 35	2	0	2
36 – 40	0	0	0
> 40	1	1	2
TOTAL	7	2	9

splenectomy sepsis is reported in only 20% of cases in the first-six months, 30% within a year and about 60% in 2 years, prophylaxis may not be most important in the early days. The follow up period was short in this study to be able to give any useful comment. However, it was not seen even in those patients who had splenectomy long ago.

Prevention of syndrome of "OPSI" can theoretically be achieved by administration of polyvalent pneumococcal vaccine. However, the usefulness of this vaccine is limited since a small portion of the pneumococcal serotype is not sensitive, and still other organisms accounting for one half of the cases of postsplenectomy sepsis are not covered. Sherman[24] suggests patients for splenectomy should preferably be vaccinated before operation, and to be on a safe side repeating the vaccine every 5 years.

Pulmonary complications such as atelectasis arise from varying degrees of bruising of the diaphragm during trauma or splenectomy. Vigorous pulmonary physiotherapy and ambulation prevent this complication.

Paralytic ileus and gastric dilatation which may result from peritoneal irritation by hemoperitoneum can be relieved by nasogastric aspiration postoperatively. Occasionally bone pain occur due to reactionary bone marrow hyperplasia as a result of bone marrow taking over the splenic function.[6]

Splenic vein thrombosis is rare occurring in the second week with unexplained abdominal pain and fever. Propagation of the thrombus in the splenic vein may lead to partial vein thrombosis which is fatal. After splenectomy, malaria attacks may increase or recrudescence of latent infection may occur. This is due to lack of sinusoidal circulation

of the spleen which tends to filter out infected erythrocytes from blood stream.[2]

The splenectomized patient should be warned of these risks and especially that of infection from encapsulated bacteria. Duncombe[25] suggests that such patients should be advised to carry special identification cards and store small supplies of antibiotics at home which could be used when they fell ill.

CONCLUSION

Traumatic rupture of the spleen is a surgical emergency, the diagnosis of which depends on history and clinical features especially in our setting and other Developing countries where diagnostic resources are limited.

The incidence of overwhelming sepsis following splenectomy for trauma is difficult to establish in our centre since most patients are lost soon after discharge, or attend few outpatient clinic visits. Although the follow-up period is not adequate, it seems as though the incidence of OPSI is low in our society, since we have not singled such a complication. This favors splenectomy for traumatic rupture in adults as a safe modality of treatment. In so doing the complications that may arise from splenic salvage, demanding careful monitoring with equipment which are not available are avoided. However non-operative approach is a viable alternative in hemodynamic stable adult patients with splenic injuries due to blunt trauma when intensive monitoring is available.

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1.3: THORACIC INJURIES AS SEEN AT MUHIMBILI MEDICAL CENTRE

SUMMARY

Between October 1991 and December 1992, twenty one patients with thoracic injuries were admitted in the Thoracic unit of Muhimbili Medical Centre. There were 12 men and 9 women who ranged in age from 18 to 73 years (average 30 years). The male to female ratio was 4:3. (Table I) and 14 patients were in the age group of 21 to 40 years.

Blunt chest injuries were the commonest, accounting for 16 of the 21 cases. Automobile accidents, assaults and falls were the major causes of the blunt chest injuries accounting for 13, 2 and 1 cases respectively (Table II). The sharp or penetrating chest injuries were recorded in five patients (Table II). Chest pain, dyspnoea, bruises and bleeding chest wounds were the main mode of presentation (Table V). Of the twenty one studied patients, seven had closed tube thoracostomy for hemothorax, pneumothorax or both. None needed Thoracotomy. All patients recovered well and the mean hospital stay was 5 days (range 1–35).

INTRODUCTION

The chest presents approximately one third of the body surface area to the environment and is therefore vulnerable to injury.[1] High technology including the automobile and weapons capable of hurling high velocity missiles also contribute to trauma. On the other hand the chest is often the target of assault because it contains vital organs.[1,2]

The severity of thoracic injuries especially in blunt injuries may be difficult to determine

soon after injury and both clinical and radiological evaluation at that time may be misleadingly normal.[3] Hypoxia and hypercapnia that may develop following thoracic injuries cannot be tolerated for long by most tissues in 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 a whole.[4] For this reason, thoracic injuries should be promptly recognized and treated so as to regain and maintain the normal physiology and prevent complications.

Although the magnitude of thoracic injuries as an entity has not been well documented in Tanzania, they are among the common causes of the admissions seen at Muhimbili Medical Centre. This prompted the author to take interest in this study. Below is an appraisal of one patient and profiles of the other patients is presented in a tabular summary.

CASE REPORT

M.M., IP No.758799 MMC was a 32 year old female, a pedestrian who was admitted in ward 2, one hour after being knocked down by a fast moving car while attempting to cross one of the city's main roads. She was restless, dyspnoeic, disoriented in time, person and place, and was in agony.

Clinically she was found to have multiple bruises on both upper and lower limbs and the trunk. No evidence of bleeding was seen from the ears, nose or mouth. Had mild pallor, her blood pressure was 80/60 mmHg and the pulse rate was 120/min, feebly felt. Evaluation according to the Glasgow coma scale score revealed, eye movement to pain

stimuli (3 points), inappropriate word response to verbal communication (3 points) and responded by withdrawal of her limbs following inflicted pain (4 points). Total score was 10 points. The pupils were equal, normal in size and reacted normally to light.

There was no obvious chest deformity but the chest movements was reduced on the right side. No flail segment of the chest was noted. There was evidence of subcutaneous emphysema on the right supramammary region. Breath sounds were diminished on the right hemithorax. The apex beat was at 5th intercostal space along the left mid clavicular line, and the heart sounds were normal.

The abdomen was not distended, there was no mass or tenderness. No shifting dullness was elicited. Bowel sounds were normal. On the extremities she had bilateral closed fractures of the distal thirds of the tibia and fibula. Other systems were normal. The following preliminary diagnoses were made:-

1. Severe blunt chest injury with shock.
2. Fractured ribs, pulmonary contusion and hemopneumothorax right hemithorax.
3. Bilateral closed fractures of the distal thirds Tibia and fibula.

The initial management of the patient included grouping and cross matching, institution of intravenous drip using Ringer lactate solution to resuscitate the patient until blood pressure and pulse rate were stable. A quick chest radiograph, frontal view revealed emphysema on the right supramammary and axillary region, simple fracture of ribs 2nd, 3rd, 4th, 5th and 6th right anterior chest. Similarly a small hemopneumothorax right hemithorax was evident. A size 37CH thoracic tube was inserted at the 7th intercostal

space along the mid axillary line into the right hemithorax. About 300 ml of fresh blood was immediately drained and a gush of air escaped. The tube was then connected to the underwater seal. She was started on cloxacillin 500 mg intravenous 6 hourly for 5 days. Hourly urine output through urethral catheter and vital signs were closely monitored.

The fractured lower limbs were immobilized using POP cast below the knee, awaiting internal fixation of one of them when the patients condition was fully stable. The patients general condition stabilized after the first 24 hours. The catheter was then removed to allow self bladder control. The thoracostomy tube was removed after 48 hours, as it was not draining and chest radiograph showed full lung expansion.

She underwent open reduction and internal fixation of the fractured left tibia one week after admission, the indication being multiple fractures. She had an uneventful postoperative period.

The patient was discharged on the third week in a good condition, with a weight bearing POP cast to the right leg. She was seen once at the surgical clinic two weeks after being discharged. Was doing fine and had no complaints.

TABULAR SUMMARY

TABLE I: PATIENTS DISTRIBUTION BY SEX AND AGE

AGE IN YEARS	MALES	FEMALES	TOTAL
10–20	1	2	3
21–30	5	3	8
31–40	4	2	6
41–50	1	1	2
51–60	1	0	1
60+	0	1	1
TOTAL	12	9	21

- Most patients belonged to the age group 21 to 40 years.

TABLE II: NATURE OF INJURY

NATURE OF INJURY		NUMBER OF PATIENTS
Blunt injury	Automobile accidents	13
	Assaults	2
	Falls	1
Penetrating injury	Assaults	4
	Gunshot	1
TOTAL		21

TABLE III: PRESENTING FEATURES.

FEATURE	FREQUENCY IN PATIENTS
Chest pain	21
Difficult in breathing	10
Bruises	8
Bleeding chest wall wounds	5
Subcutaneous emphysema	3
Hemoptysis	0

TABLE IV: CAUSE AND FREQUENCY OF THE ASSOCIATED NON THORACIC INJURIES.

CAUSE OF INJURY	TYPE OF ASSOCIATED INJURY	FREQUENCY
Automobile accidents	Head injury (scalp laceration + brain contusion)	1
	Ruptured spleen	1
	Fractured tibia, fibula	1
	Fractured humerus	1
Assault	Head injury (brain contusion)	1
TOTAL		5

- Head injury, ruptured spleen as a result of automobile accident occurred in the same patient.

DISCUSSION

Thoracic trauma is frequently fatal, involving one or more of the following: thoracic cage, the cardiovascular system, bronchopulmonary system and the diaphragm. A survey of the

literature shows that the thoracic oesophagus and thoracic duct are rarely involved.[1-3,5] The rise in automobile accidents and weapons capable of hurling high velocity missiles contribute to the increasing frequency of chest injuries as a cause of hospital admission.[1,3,6,13] During this study, it accounted for nearly 5% of all admissions in the thoracic unit of MMC, this excluded the less severe cases which had been admitted to other surgical firms. However the actual magnitude of thoracic injuries as an entity has not been well documented in Tanzania.

In the United States, 25% of the 100,000 annual civilian traumatic deaths result from chest trauma.[3] The series by Moussal et al[6] reports thoracic injuries as the third ranking cause of trauma hospital admissions in London after head and lower limb injuries. Many of these deaths can be prevented with prompt diagnosis and correct management.[1,2,6] Despite these high mortality rates, most thoracic injuries do not require thoracotomy, but rather simple life-saving maneuvers such as airway control and tube thoracostomy.[2] In this study no mortality was recorded, this probably could be that some of the more severe cases of thoracic injuries which are usually associated with multiple other injuries died at the scene of accident or before arrival to hospital.

PATHOPHYSIOLOGY

The thorax is responsible for the vital delivery of oxygenated blood to metabolically active tissues. Three pathologic consequences of thoracic injury, either alone on in concert, are responsible for inadequate oxygen delivery:-

(1) Hypoxemia - can follow such divergent etiologies as airway obstruction,

pneumothorax, flail chest, pulmonary contusion, tracheobronchial injury, and diaphragm rupture. Each of these injuries limits the physiologic function of air exchange.

(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.[2,4]

(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 because the clinical manifestations are subtle and the diagnosis is imprecise.[7]

Penetrating injuries following direct cardiac injury and pericardial tamponade may rapidly compromise cardiac function. The severity of chest injuries is influenced by various factors, these also influence the prognosis. They include the following:-

(1) Patients with pre-existing lung disease, chronic bronchitis, emphysema and bronchial asthma or patients with impaired respiratory reserve have a poor outcome.[6]

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

(3) Both extremes of age fair badly.[6] The aged have a more rigid brittle rib chest cage rendering them more prone to multiple fractures, they also have low vital

capacities, reduced lung compliance, impaired cardiac function and increased incidence of thromboembolism. The young age group have highly elastic rib-cage and may develop serious intrathoracic damage without rib fractures.[2,6]

(4) Smokers tend to have poor lung macrophage phagocytic activity, thus with thoracic trauma they are more predisposed to infection and fair badly.

(5) Obese patients.

(6) Patients with scoliosis or kyphosis or both deformities.

(7) Patients who have had previous thoracic surgery such as pneumonectomy or thoracoplasty.

INITIAL EVALUATION AND MANAGEMENT

The initial care of injured patient necessitates two assumptions. The first is that the patient may have more than one injury; the second is that the obvious injury is not necessarily the most important one.[2,3,6] The most important steps are:-

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

2. Cardiovascular support in terms of intravenous fluids with blood transfusion where deemed necessary. Cardio-pulmonary resuscitation with cardiac massage 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 causes 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.

RECOGNITION AND MANAGEMENT OF SPECIFIC INJURIES

RIB FRACTURES

Fracture of the ribs is the most common thoracic injury.[2] 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 imparted on the chest wall.[3,6] 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, the tracheobronchial tree, and the neurovascular structures to the upper extremities.[2,4]

The pain produced by the fracture causes the patient to splint the chest wall causing hypoventilation and accumulation of secretions. These secretions, being rich in proteins,

rapidly become infected and pneumonitis or pneumonia results.[1] A chest film should be obtained to exclude other intrathoracic injuries 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.[1,2,6] In case of narcous the dosage should be adequate to relieve pain but not sufficient to depress respirations or inhibit cough.

Hospital admission for pain relief, cough assistance, and endotracheal suction may be necessary for several days, particularly in the elderly patients.[2] Rib belts and adhesive taping, although once popular, should be avoided because the limitation in motion increases the incidence of retained secretions and atelectasis.

In this study six patients had fractured ribs, all of whom were managed conservatively improved well (Table III). The number might have probably been higher if the other cases admitted in other surgical firms could have been traced and considered.

FLAIL CHEST

This refers to a situation in which the ribs are fractured in more than one part of any particular rib. Unilateral fracture of four or more ribs anteriorly and posteriorly or bilateral anterior or costochondral fracture of four or five ribs, will produce enough instability that paradoxical respiratory motion results in hypoventilation of an unacceptable degree.[2] The Flail chest can also occur with bilateral rib fractures as occurs with "steering wheel" injuries.[1,2] Trauma of this magnitude inevitably contuse the

underlying lung, aggravating the physiologic deficits. Although, usually visually apparent in the unconscious patient, because of splinting, the flail segment may not be readily apparent in the conscious patient. If it is severe and untreated; atelectasis, hypercapnia, hypoxia, accumulation of secretions, and ineffective cough occur.[2,4,6,8] The pathophysiologic effects may be present immediately or may progress over several hours and present as late respiratory decompensation.[2] Many methods have been improvised to stabilize the chest wall with variable results. In addition to providing mechanical stability, operative stabilization provides very efficient pain relief.[9] Internal fixation can be achieved by use of Kirschner wires, intramedullary plates, small fragment plasts or metal clips.[9]

In this study series non of the patients had flail chest.

PNEUMOTHORAX

1. Open Pneumothorax

   A defect in the chest wall provides a direct communication of the pleural space with the environment.[2] Most commonly occurs in combat injuries, though it is being seen more frequently in civilians, especially as a result of shotgun wounds at close range.[4] Inability to generate negative intrathoracic pressure results in lung collapse and marked paroxysmal shifting of the mediastinum with each respiratory effort.[2,4] The resultant hypoventilation and diminished cardia output can become immediately life-threatening. Diagnosis is readily apparent as each inspiration draws air into the intrapleural space, causing the characteristic "sucking chest

wound". Treatment consist of prompt closure of the defect with a sterile dressing, followed by venting of the chest with either a flutter valve or chest tube to treat the possible resultant tension pneumothorax.[2,4,6] In this study two patients had open pneumothorax. Both were managed by wound closure and tube thoracostomy connected to under water seal drainage at the same sitting. Both the two patients fared well, non had complications.

2. Closed pneumothorax

This may result from blunt as well as penetrating thoracic trauma, and is commonly associated with hemothorax.[2,4] As a result of direct or indirect communication of bronchus, bronchiole or alveolus with the pleural cavity, the ipsilateral lung collapses.[1,2] However if not associated with other significant injuries, the lesion causes only mild respiratory embarrassment, since compensation is being maintained by the contralateral lung.[3,4,8,12]

Tension pneumothorax develops when a flap valve leak allows air to enter the pleural space but prevents its escape.[1,2,4] Intrapleural pressure rises, causing total lung collapse and a shift of the mediastinum to the opposite side. This may lead to rapid cardiorespiratory collapse and prove fatal if the raising pressure is not relieved immediately. Tension pneumothorax is a true surgical emergency, requiring immediate diagnosis and chest tube insertion. It is important to remember that in critical situations, an application of a large bore needle introduced into the chest cavity in the second or third intercostal space along the

mid clavicular line can be life saving, as it relieves the intrapleural pressure tension immediately.[1,8]

In this series one patient had simple pneumothorax and another one had tension pneumothorax. Both had chest tube drainage connected to an underwater seal, however the one with tension pneumothorax developed empyema which had to be treated with antibiotics and the tube drainage which continued for five weeks.

HEMOTHORAX

Hemothorax is one of the most common problems following major trauma of the chest due to either penetrating or blunt injury.[2,4] Jurkovich et al[2] suggests that hemorrhage into the pleural space occurs in some quantity in almost every patient with a diagnosable chest injury, however blood loss may vary from slight to extensive.

In 85% of patients, tube thoracostomy with a NO. 32 to 36 French chest tube placed in the sixth or seventh intercostal space on the mid axillary line is the only treatment required.[2] Early tube thoracostomy prevents clotted hemothorax or subsequent fibrothorax in most instances. Needle aspiration is not adequate.[1,2]

In this series two patients had hemopneumothorax, both were successfully managed by closed tube thoracostomy. None developed empyema thoracis as a complication.

PULMONARY CONTUSION

This occurs in most patients with flail chest but can also appear without any evidence of rib fracture.[2] It also commonly results from decelerating or crushing injuries.[6] As a result of lung contusion, fluid and blood from ruptured vessels enter the alveoli, interstitial

spaces, and bronchi producing localized airway obstruction. The extravasation of fluid and blood provides an excellent culture medium especially in debilitate patients, with 50 to 70% developing pneumonia, and out of these 35% will go on to pulmonary abscess, empyema or both.[4] On the other hand, as localized airway obstruction progresses, pulmonary compliance decreases and ventilation becomes more difficult.[2,4] Pulmonary contusion may be confused with the adult respiratory distress syndrome (ARDS), or may even be associated with it.

Physical examination is usually unrewarding. In contrast, radiographic examination eventually demonstrates patchy consolidation, from minimal to lobar. Management involves careful pulmonary support and clearing of secretions, with ventilatory support if arterial blood gas values cannot be maintained in a physiologic range. Prophylactic administration of antibiotics have also been advocated.[4] The use of steroids is controversial. In this study we had four cases of pulmonary contusion (Table III). Both were associated with simple rib fractures. They were managed by antibiotics, antiinflammatory agents and close observation. Non received steroids. They all improved after initial period of pyrexia.

TRACHEAL AND BRONCHIAL INJURIES

Blunt tracheal or bronchial injuries are often due to compression of the airway between the sternum and the vertebral column in decelerating "steering wheel" accidents, with resultant shearing of the right main stem bronchus from the carina or transverse lacerations of the trachea.[2] Alternatively a rupture of the membranous trachea may occur

during chest wall compression with a closed glottis.

The incidence of these injuries is difficult to establish since many sustaining the injury die before reaching the hospital. Patients with tracheal injuries may present with mediastinal and deep cervical emphysema or pneumothorax with a massive air leak. Hemoptysis, hemopneumothorax, subcutaneous crepitance, and respiratory distress are frequent.[2,4] The presence of a tracheobronchial disruption should be considered in any instance in which a significant chest trauma has occurred.[4] The most reliable means of establishing the site, nature, and extent of the lesion is use of bronchoscopy. Bronchography has occasionally been helpful in establishing the diagnosis.[4] Emergency treatment consists of inserting the endotracheal tube beyond the injury, this facilitate ventilation and prevent aspiration of blood. Tube thoracostomy is clearly indicated in the presence of a hemothorax or pneumothorax. If endotracheal intubation is not possible or if the anatomy is significantly distorted, surgical entry into the trachea for placement of a tracheostomy tube may be necessary.[2,6] In this study we had no patient with tracheobronchial injury.

DIAPHRAGMATIC INJURIES

Although traumatic diaphragmatic rupture 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.[4] The mechanisms of injury is through a crushing force producing a sudden increase in the intrathoracic or intra abdominal pressure against the fixed diaphragm.[2,4,10] Penetrating lesions from stabbing or gunshot wounds occuring as high as

4th ICS can also result in diaphragmatic disruption depending on the phase of respiration at the time of injury.[4,10]

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 which severely restricts respiration. 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.[4,10,11] The chest roentgenogram usually confirms the suspected diagnosis, demonstrating abnormal shadows, elevated diaphragmatic outline; and a shift of the mediastinum.

Management is by immediate operative repair, unless precluded by other more serious injuries.[2,4,10,11] The choice of operative approach is somewhat controversial, if the diagnosis is made early, an abdominal approach is usually indicated to exclude associated injuries which is the case in 75% of patient.[2,4,11] 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 then be repaired through the diaphragmatic rupture.[4,11]

In this study one patient had a radial rupture of the left hemidiaphragm, following a road traffic accident. He had sustained also a compression fracture of thoracic vertebra. No.12. The repair was done trans-abdominally shortly after stabilizing the shocked hemodynamic status. Intra operatively no intra-abdominal visceral injury was evident. The bleeding was apparently from the torn phrenic artery. He recovered from operation uneventfully, but

the fractured vertebra had to be managed conservatively using fracture board for immobilization. Was discharged after 8 weeks.

OTHER INJURIES

The following injuries may also occur in both blunt or penetrating injuries of the chest, but none of them occurred in this study.

1. Aortic ruptures and major vessel injuries

   These are not seen commonly, probably most such patients die at the scene of the accident. In one estimate, 80% of such patients die at the scene.[3]

2. Cardiac and pericardial lacerations

   These are lethal injuries, mostly diagnosed at post–mortem. In one series there were 14 cases comprising 2% of all thoracic injuries, and none of them survived.[3,12]

3. Cardiac contusions

These may be minor, and can present later on with arrhythmias and cardiac conduction defects. If diagnosed, myocardial contusion should be treated as myocardial infarction.[3,7]

CONCLUSION

The steady increase in frequency of road accidents and assaults have made chest trauma a common cause of hospital admission at Muhimbili Medical Centre as evidenced by this small scale study. Blunt chest injuries seems to be more common than penetrating chest injuries. This study had no patient with major intrathoracic injury and no death occurred. However it is possible that the severely injured patients died at the scene of the accident

and thus never reached the hospital. By having an efficient ambulance system, these could have been picked and possibly saved. The need for routine postmortem especially in suspected chest injury should help reveal such deaths.

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1.4: THE MANAGEMENT OF EMPYEMA THORACIS

SUMMARY

Twenty cases of empyema of the thorax were managed by the Thoracic Unit of Muhimbili Medical Centre over a period of one year and two months lasting between October, 1991 and December 1992. During this period the author was attached to the unit and actively participated in the management of these patients. 12 were males and 8 females giving a male to female ratio of 3:2. Their age ranged from 11 to 55 years (mean 30.9 years). 75% constituted the age group 11–40 years (Table I). The aetiology of empyema was tubercular in 16 patients (80%) whereas those following thoracic trauma, pneumonia and oesophagoscopy accounted for 10,5,5, percent respectively (Table II). All cases with spontaneous bronchopleural fistula were taken to be tuberculous and some were proven either by sputum studies or pleural biopsy. HIV tests by Elisa and Western blot techniques done in the 16 patients with tuberculosis as the underlying cause was found positive in 5 patients and negative in 8; whereas HIV status was unknown in 4 patients (Table III). Culture results from debris and empyema fluid had variable organisms (Table IV). Closed tube thoracostomy was the mode of treatment in fifteen patients, the other five patients had to subsequently undergo open chest drainage after attempts at closed tube thoracostomy proved futile within a period of six to eight weeks (Table V). The results had been good.

There were two deaths, both were grossly wasted and presented late. The mean hospitalization after open chest thoracostomy was two weeks. The management for most

of the patients has been very challenging due to late presentation with advanced disease.

INTRODUCTION

Empyema is a suppurative infection confined to a natural anatomical space by normal epithelial boundaries, in the thoracic cavity this is the potential space existing between visceral and parietal pleurae. Empyema has been a prominent component of medical literature since the time of Hippocrates.[1] The condition continues to be a problem to reckon with in most developing countries, including Tanzania, since misdiagnosis and inadequate management of patients with pulmonary tuberculosis, pneumonia, chest trauma, subphrenic and liver abscess occur often.[2,3] In 1985 for example thoracic empyema demanded more surgical attention than any other thoracic problem at Muhimbili Medical Centre. In Nigeria thoracic empyema accounted for 30 percent of thoracic surgical cases treated over a period of 6 years.[4]

In Africa where many people do not have easy access to medical care and thus delay in treatment, thoracic empyema remains to be a very distressing and disabling condition which cripples the chest wall and lung function.[5] Whereas in the preantibiotic era, surgical thinking favored early and aggressive drainage of closed space infections, currently the successful treatment depends upon early recognition of the problem, selection of appropriate antibacterial therapy, and adequate drainage of the infected fluid with re-expansion of the underlying lung to completely obliterate the empyema space.[1,2,4] Inspite of limitation of resources and poor clinical condition of most patients, the overall management has significantly influenced the reduction in mortality.

CASE REPORT

J.O. (IP NO: 717636), a 16 year old male was referred from Mwananyamala hospital in August, 1992. On admission to the thoracic Unit he complained of left sided chest pain and breathlessness for one month. Six months prior to the insidious onset of the burning chest pain, he had been experiencing a productive non-irritating cough with a copious, purulent, thick sputum. He denied the history of hemoptysis but admitted the history of associated low grade evening fevers, weight loss and malaise in the course of his illness. The chest pain was said to be situated centrally and being referred to the back. There was no associated periodicity nor aggravating factors. There was no history of tuberculosis contact and denied smoking or drinking alcohol. He denied history of chronic or similar illness in his family.

Clinical examination revealed a wasted, febrile young man with axillary temperature of 38.5^{0}C. He had pallor, was dyspnoeic but was not cyanosed. He was not dehydrated and had no clubbing of fingers or palpable lymph nodes in the neck, axilla and groin. His respiration rate was 30/min, pulse rate 70 beats per minute was regular and of good volume. BP 110/70mmHg. The respiratory system revealed diminished chest movements on the left side, tracheal and apex beat shift to the right side. He had limitation of chest expansion on the left side. There was a stony dull percussion note on the left infrascapular region with a resonant note above it. Tactile vocal fremitus and resonance were both diminished in the left hemithorax; and the breath sounds were absent. The right side of the chest was normal. Other systemic examinations were found to be normal. An

impression of pleural effusion, probably secondary to pulmonary tuberculosis was made. The following investigations were done:-

1. Frontal view chest x-ray: revealed a homogenous opacity with air fluid level in the left hemithorax.
2. Thoracentesis for diagnostic purpose revealed a straw colored fluid.
3. Full blood picture: Haemoglobin 8.9 g/dl Total Wbc-count 8600 and a differential count of: Neutrophils 74%, Eosinophils 2% and lymphocytes 24% ESR was 103mm in the first-hour. Blood film: Anisocytosis, hypochromic Rbc platelet count was reported adequate.
4. Mantoux test was moderately positive (< 10mm).
5. Sputum direct smears for mycobacterium tuberculosis by Ziehl Nielsen stain was positive for acid fast bacilli.
6. HIV antibody screening was positive by both Elisa and Western blot tests.
7. Serum creatinine - 75 micromoles/L.

A closed tube thoracostomy was done, the chest tube was inserted in the 7th intercostal space in the left mid axillary line, and the system was connected to an under waterseal. About 700ml of straw-colored fluid was immediately drained with a massive air leakage, thus revealing presence of a bronchopleural fistula. Two weeks later the tube slipped out and had to be reinserted; this time frank greenish pus was drained. Culture isolate from this pus revealed pseudomonas aeruginosa which were sensitive to Erythromycin. The patient was also started on the following anti-tuberculous drugs:-

1. Streptomycin 0.75 g intramuscularly daily.
2. Thiazina 450mg tablets daily
3. Pyrazinamide 1.5g daily.

The chest tube was maintained for six weeks but the empyema could not clear, Similarly the serial chest radiographs revealed insignificant lung expansion. Although the patient was gaining weight – and kept on balloon blowing exercise, his temperature curve was unstable. He was then planned for open thoracostomy which was done on 14.12.92 nearly 4 months since the first admission. A left sided rib resection and open chest drainage was done revealing a huge empyema cavity extending posteriorly to the apex of the left chest, thickened parietal pleura and a raised left hemidiaphragm. The skin flaps were stitched to the parietal pleura and pleural cavity was sucked clean. The window was subsequently dressed with gauze.

The postoperative period was uneventful, the cavity cleared and was then discharged home three weeks after surgery. He was seen at two outpatient visits, his chest window was found to be healing and the cavity smaller with less discharge from the site. He was still on maintenance anti TB therapy using Isoniazid and Thiacetazone. He has since then been lost from follow up and the final outcome is not known.

This case report has illustrated the following points:–

1. That bronchopleural fistula as a complication of pulmonary tuberculosis can lead into empyema of the thorax.
2. The morbidity due to empyema thoracis is substantial

3. It illustrates closed tube thoracostomy and open chest drainage in the management of empyema of the thorax.

The other patient's profiles in this series are summarized in the tables below:-

TABULAR SUMMARY

TABLE I: DISTRIBUTION OF THE PATIENTS BY AGE AND SEX

AGE GROUP (YEARS)	MALE	FEMALE	TOTAL	%
11 – 20	4	1	5	25
21 – 30	1	4	5	25
31 – 40	5	0	5	25
41 – 50	2	1	3	15
> 50	0	2	2	10
TOTAL	12	8	20	100

NB: Children under 10 years were not treated in the Thoracic Surgical Unit.

TABLE II: CAUSE OF THORACIC EMPYEMA

UNDERLYING DIAGNOSIS	MALES	FEMALES	TOTAL	%
Pulmonary Tuberculosis	10	6	16	80
Penetrating Chest Trauma	1	1	2	10
Complicating Pneumonia	1	0	1	5
Following Oesophagoscopy	0	1	1	5
TOTAL	12	8	20	100

TABLE III: HIV SERO STATUS OF THE PATIENTS STUDIED

HIV STATUS	MALES	FEMALES	TOTAL
Negative	4	4	8
Positive	3	2	5
Unknown	3	2	5
TOTAL	10	8	18

- Thirteen patients had their HIV status known 5 of them being positive had tuberculosis asthe underlying etiological factor for the empyema.

TABLE IV: MICROBIOLOGICAL PATTERN

ORGANISM ISOLATED	NUMBER OF PATIENTS
Staphylococcus aureus	4
Coliforms	2
Pseudomonas aeruginosa	1
No bacterial growth	7
NO record	6
TOTAL	20

- Anaerobic cultures were not done.

TABLE V: TREATMENT MODALITIES OFFERED TO THE PATIENTS

TYPE OF TREATMENT	NUMBER OF PATIENTS
Thoracocentesis	0
Tube thoracostomy alone	15
Tube thoracostomy followed by open chest drainage	5
Tube thoracostomy followed by decortication	0
TOTAL	20

- Most patients fared well with thoracostomy alone, few others needed open chest drainage.

TABLE VI: SIDE OF THE EMPYEMA THORACIS

INVOLVED SIDE	NUMBER OF PATIENTS
Right	8
Left	12
Bilateral	0
TOTAL	20

TABLE VII: DURATION OF HOSPITALIZATION

DURATION IN WEEKS	NUMBER OF PATIENTS
Up to 15	12
16 – 30	6
31 – 45	2
46 +	0
TOTAL	20

- Hospitalization ranged from 4 weeks to 45 weeks with mean of 20 weeks.

The other salient features of the studied patients included the following:–

1. Eleven patients had associated brochopleural fistulae.
2. Sixteen patients received antituberculous treatment.
3. Only nine patients among the sixteen with tuberculosis as the underlying cause for empyema thoracis had Acid fast bacilli in direct smear of sputum.
4. All patients were kept on balloon blowing exercise following thoracostomy and serial chest x–rays taken at 1–2 weeks interval to assess lung expansion.
5. Thoracostomy was declared a failure at 6 to 8 weeks if there was no lung expansion and had persistent discharge from the cavity.

DISCUSSION

Empyema of the thorax is a malady which has long plagued mankind, since at least the time of Hippocrates, who first wrote about this problem. It usually occurs secondary to a primary process elsewhere and not a primary pleural infection. Prior to the introduction of antibiotics, it was a frequent, and often difficult, affliction to be managed.[1,3,6] In the

early years empyema complicated pneumonia in 5 to 10 percent of the cases.[1] In the Western World it now occurs in about 1 percent of cases with pneumonia. In Canada an incidence of 0.5 per 1000 admissions has been reported.[7]

However the incidence in developing countries is still high. In Nigeria quoted figures indicated that empyema thoracis represented 30 percent of thoracic surgical cases[4] whereas in Maputo 54.4 percent of adult patient requiring chest drainage had empyema.[8] In Tanzania between 1986 to 1988, Lema et al[2] had managed 63 cases in the Thoracic Unit of Muhimbili Medical Centre. Empyema remains a common thoracic surgical condition seen in this centre.[2,9]

The disease affects all age groups and both sexes, however most of the patients are young. Lema et al[2] and Kamdar et al[10] observed a high frequency of their cases between 11 to 40 years with rates of 68 and 57 percent respectively. Nevertheless, empyema thoracis continues to be one of the major problems in the practice of Medicine and still carries a high mortality and prolonged morbidity despite new antibiotics.

In this study 75 percent of patients were in the age group 11–40 years (Table I). A male to female ratio of 3:2 was noted. This observation is in accord with that made by Lema[2] and Kamdar.[10] Most commonly, the primary condition is a pneumonic process in the underlying lung, which extend to the pleura directly, or by way of the lymphatics, or by hematogenous spread, or by rupture of necrotic pulmonary parenchyma.[11]

PATHOPHYSIOLOGY AND MICROBIOLOGY

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 even fungi since the introduction of penicillinase resistant penicillin.[5,6,13] Today, no single organism predominates in empyema[14] but anaerobic organisms are being isolated more frequently.

Tuberculous empyema though described to be uncommon in the Western world,[5] still remains a significant problem in developing countries.[2,4,8,10] In tuberculous empyema the fluid is thin and serous and tubercle bacilli may be difficult to isolate. Positive cultures are obtained in 50% or less of cases of empyema, presumably because of prior antibiotic therapy or inadequate culture technique, particularly if anaerobes are responsible for the empyema. Lema et al[2] analyzing bacterial isolates from 59 patients found that about half of the patients the cultures were sterile. However, anaerobic cultures were not done. Most of the positive growths had mixed infections.

The course of development of empyema thoracis has been classified by the American Thoracic Society into three phases based on its natural history, and this scheme is useful in planning therapy.[13] These are:-

(1) Exudative stage (early phase)

This is characterized by increased permeability of visceral pleura resulting in formation of a "sterile" exudate.

(2) The Fibrinopurulent stage (transitional phase)

A progression of the untreated empyema characterized by a thicker, more turbid fluid and appearance of fibrin on the pleural surfaces. The lung begins to be trapped by a restrictive fibrin peel.

(3) Chronic phase (Organization stage)

The fibrin peel is organized with an ingrowth of capillaries and fibroblasts from both Visceral and parietal pleural surfaces produces a layer of fibrous tissue that encases the lung. Frequently follows inadequate treatment of the acute phase. Less commonly it results from:–

a) A persistent bronchopleural fistula;

b) A retained foreign body; or

c) An insidious infection due to tuberculosis, fungi or actinomycosis.

"Dry pleurisy" may be the first indication of pleural inflammation although this is quickly followed by the outpouring of fluid rich in protein and polymorphs but frank purulence must develop before an effusion is referred to as an empyema.[15] The empyema may lie free in the pleura 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 shift of the mediastinum to the apposite side. Fluid may be absorbed making the pus more viscous. Fibrin is continuously deposited on the pleural surfaces producing a thickening rind, the deeper layers of which become fibrotic and avascular permitting easy stripping during surgical decortication. The transition from an acute to chronic empyema is arbitrary, periods of between 6 and

12 weeks having been suggested.[15] The continual formation and fibrosis of the pleural rind progressively restricts chest wall and diaphragmatic movement, eventually producing a shrunken, flattened, immobile hemithorax with overlapping 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 achieved.[5,13,15] Occasionally an empyema erodes through the chest wall forming a collar - stud abscess which may break down giving a discharging fistula in continuity with the empyema cavity. This is known as empyema necessitatis.[5,18]

The other fate of empyema is erosion and ejection into the lung and out through bronchi forming brochiopleural fistula (BPF). Due to its persistent air leak BPF potentiates infection by providing a route for constant inoculation of the pleural space and by promoting lung collapse.[1,15]

In this study most of the patients presented with the chronic form of an empyema due to late presentation and 80% were tubercular in origin thus proceeding to a chronic course of the disease. Eleven patients had developed bronchopleural fistula.

CLINICAL PRESENTATION

Empyema of the thorax may present in three forms or stages: acute, subacute or chronic.

(1) Acute empyema

The patient may present with a pyrexia or a respiratory infection which has failed to respond or has relapsed during or after treatment. Fever, rigors, a swinging

temperature, malaise, anorexia, weight loss and pleurisy, followed by a gnawing chest pain, are common but may be modified by antibiotic therapy. Cough is unusual except with a bronchopleural fistula when large quantities of pus may be expectorated. Dyspnoea may arise if there is lung compression. The signs of an empyema lying free within the pleural space are those of a pleural effusion, but encapsulated lesions in the interlobar fissures, mediastinum or subpulmonary area may produce no abnormal signs. Finger clubbing can develop within 2–3 weeks.

(2) Sub–acute empyema

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

(3) Chronic Empyema

This most frequently follows inadequate treatment of the acute phase. It may also occur resulting from failure of diagnosis of the primary condition or other underlying pathology such as a foreign body, actinomycosis, osteomyelitis of ribs with a sequestrum, bronchiectasis, lung abscess or a tumor.

Generalized toxaemia, malaise, anorexia, weight loss, grumbling pyrexia, gnawing chest pain and cutaneous fistulas are common. A persistent cough productive of sputum, particularly when lying on the contralateral side, is a feature of a persistent bronchopleural

fistula. Progressive pleural fibrosis produces a shrunken, immobile hemithorax with scoliosis to the affected side. Finger clubbing is also common. When bronchopleural and cutaneous fistulas coexist air is expelled through the skin sinus on coughing. A normochromic, normocytic anaemia and leucopenia are common. The features of amyloidosis may be superimposed.

In this study of 20 patients; sixteen patients presented with chronic empyema, probably due to late referral and inadequate antibiotic therapy. The other four patients were probably in subacute phase of the disease.

DIAGNOSIS

(1) Conventional chest radiographs:

A free-lying empyema resembles a pleural effusion and, if large, the entire hemithorax may be opaque. Postero-anterior and lateral films are necessary to localize an encapsulated lesion which has different features depending on its site. Multiple views are necessary to accurately assess the size, position, and thickness of the empyema cavity.[9] Interpretation of these films may be difficult in the presence of underlying pulmonary parenchymal pathology.[13]

(2) Imaging:

Imaging by computerized axial tomography and ultrasound are used to localize and diagnose intrathoracic disease.

(3) Thoracentesis:

This makes the definitive diagnosis when clinical presentation, chest x-rays and

imaging are suggestive of empyema thoracis. The obtained material is addressed for:

a) Gram strain

b) Aerobic, anaerobic, fungal and tuberculosis cultures.

c) Cell count, specific gravity and protein content, and

A sample of empyema fluid allowed to stand for 12–24 hours and a sediment fraction of 75% is an indication that the chronic phase has set in.[13] Cultured clear pleural fluid and even pus aspirates may fail to grow organisms and yet pleural infection is present.[5] This may probably be explained by the fact that infection is masked by the antibiotics given while treating the primary disease or of anaerobic organisms which are difficult to grow.[10] There are occasions whereby pus may not be revealed during aspiration, this may be due to the following:–

a) Too fine a needle for pus to pass

b) Selection of the wrong site for aspiration or

c) Presence of fibrin tags or clots which block the needle.

(4) Bronchoscopy:

May occasionally be valuable to exclude the possibility of an intrabronchial tumor, inhaled foreign body, or bronchopleural fistula.[5]

(5) Full blood picture:

In acute and sub–acute empyemas a leucocytosis of 15000–30000 is common whereas leucopenia is a feature in chronic empyema. A low haemoglobin level is

a frequent finding especially in chronic empyema the blood film is usually normocytic, normochromic type.

MANAGEMENT

Pleural empyema is better prevented than treated. However in treating empyema for all stages, the objectives are.[5,11]

1. Control of the primary infection and its secondary manifestation.
2. Evacuation of the purulent contents of the empyema sac and eradication of the sac, to prevent chronicity; and
3. Re-expansion of the underlying lung in order to restore function.

The methods used to achieve objectives depend upon the stage of empyema being treated and the nature of the primary infection or source of contamination. These objectives are realized by the use of appropriate antibiotic therapy, based upon the bacteriologic diagnosis and prompt and adequate drainage.[1,11] Similarly a successful treatment depends upon early recognition of the problem and complete obliteration of the empyema space.[1,11]

The modalities of treatment include the following:-

(1) Thoracocentesis

(2) Thoracoscopy and irrigation

(3) Closed tube thoracostomy

(4) Decortication

(5) Open chest drainage

(6) Thoracoplasty.

I. TREATMENT OF ACUTE AND SUBACUTE EMPYEMA

1. Thoracocentesis

When the pus is thin the pleural space should be aspirated to dryness through a wide-bore needle and a broad-spectrum antibiotic given both intrapleurally and systemically, the antibiotic chosen depending on the sensitivities of the organism. Daily aspirations should be continued until the pus ceases to recollect.[15] Vigorous physiotherapy, including breathing exercises, will aid lung re-expansion and encourage good chest movements. Needle aspiration of an empyema at the time of thoracentesis done for diagnostic purposes may be all that is necessary if the pus is the very thin variety that is seen with streptococcal infections.[11,15]

From large series it has been shown that empyema can be treated adequately in only 9-12 percent of the cases by this method.[1] If repeated aspirations are difficult a wide-bore intercostal tube with drainage to an underwater seal system may be considered.

2. Closed tube thoracostomy

Is indicated in the exudative and fibrin purulent stage of empyema if antibiotics and evacuation have failed to control the empyema or if rapid reaccumulation of fluid occurs or if infection and toxicity are difficult to control.[1,5,11,15] This method is useful to toxic patients 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 bronchopleural fistula.[11] The tube should be of a generous caliber commensurate with the material being evacuated and should not be permitted to become occluded. The ideal tube should also have a radiopaque line and multiple holes. The tube is introduced by one of the three methods.[5]

i) A trocar and canula

ii) A trocar and tube or

iii) A tube with forceps.

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 6 cm from the chest wall, a radiological confirmation is vital that the holes are in the pleural cavity and to a certain the position of the tube. More than one tube may be necessary.[11] The tube should be connected to an underwater-seal system. The patient is then frequently re-assessed clinically and radiologically for signs of lung expansion.

With chest tube drainage, technical difficulties and failures have occurred particularly where there is loculation and multiple or inaccessible collections. In this study five of the patients needed other forms of management as tube thoracostomy was deemed to have failed (Table V).

3. Thoracoscopy and Irrigation

This is a method which is gaining popularity at present.[1] In this procedure thoracoscopy is done under general anaesthesia. Using the thoracoscope loculi are

overlying the most dependent part of the empyema. This technique provides adequate exposure, allowing one to evacuate the pus, break up loculation and adhesions, and assess the need for decortication.[11,15] Biopsies from the cavity wall should be sent for pathological and bacteriological examination.

After washing the cavity, drainage may be achieved by means of:–

(i) A closed system in which a wide–bore tube is left in the most dependent portion and attached to an underwater seal drainage bottle to which gentle suction may be applied if necessary; or

(ii) An open system where a short length of wide–bore tubing discharges into dressings or stoma bag.

Aggressive physiotherapy is essential while antibiotic therapy should be guided by the results of the bacteriological investigations, and given for sufficient time if reinfection is to be prevented.

5. Thoracotomy and Decortication

When the empyema is loculated or if there is an extensive fibrinous rind thoracotomy, excision of the empyema (decortication and removal of all the pus and fibrin clots) will achieve the goals of therapy more efficiently than open drainage.[11,15] Apical and basal drains connected to underwater seal systems ensures adequate drainage and lung re–expansion. Appropriate antibiotic therapy and vigorous physiotherapy are essential.

6. Pleurodesis

The use of talc obliterates the pleural space. Weissberg et al[18] in a study of 5 patients who had empyema of the pleura in the fibrinopurulent stage and did not respond to treatment with tube drainage and antibiotics, treated these patients by talc insufflation to stimulate pleural adhesions. Pleurodesis was achieved in all patients, with complete recovery from empyema. These results suggest that talc pleurodesis may be an acceptable solution to the problem of empyema resistant to other methods of treatment.

II. THE TREATMENT OF CHRONIC EMPYEMA

The treatment of chronic empyema demands a more vigorous and more radical approach.[1,2,8-10] Aspiration is of little value in treating a chronic empyema. Rib resection and drainage can be effective, particularly in improving the condition of severely ill patients prior to thoracotomy.

The treatment of chronic empyema like that in the acute phase aims at establishing adequate drainage in addition to the antibiotics offered.[8] When considering thoracotomy the patients physical state and the condition of the underlying lung are important, since prolonged infection often lead to anaemia and nutritional imbalance. If these are good then decortication and excision of the empyema and any cutaneous sinuses followed by free drainage usually ensures full lung re-expansion although there may be permanent functional impairment due to alveolar destruction and fibrosis.[15]

The management of chronic empyema with bronchopleural fistula or following pneumonectomy is less satisfactory and controversial. Failures are usually attributable of failure to obtain obliteration of the residual pleural space. Adebo et al[4] achieved spontaneous closure of bronchopleural fistula by the Eloesser window only. Occasionally a limited thoracoplasty or a muscle flap may be used to obliterate the residual cavity.[15] Hopkins et al.[16] had a success rate of 73 percent in their series, whereas failure was attributed to inadequate control of infection before thoracoplasty.

In this study only 5 patient out of 20 had open drainage and the results were good (Table V). None had decortication or thoracoplasty as the modality of treatment.

III TREATMENT OF POST-PNEUMONECTOMY EMPYEMA

The development of a leak in the bronchial stump left after pneumonectomy invariable leads to infection of the fluid filling the hemithorax.[12] If this occurs at an early postoperative stage then the leak may be closed surgically at times using a muscle flap. With adequate toilet and drainage of the hemithorax plus appropriate antibiotic therapy this may prevent a chronic empyema developing.[11,12]

CONCLUSION

Empyema of the thorax seems to be still a problem to be reckoned with at Muhimbili Medical Centre as evidenced by this small series of patients. Most of the cases are tubercular in origin. Closed tube thoracostomy was adequate treatment for majority of the

cases in this study, thus young doctors should be conversant with this method of treatment.

In all cases which tuberculosis was the underlying cause, appropriate and early anti TB treatment was instituted. On the other hand thoracoscopy and irrigation is a useful method in treating early empyemas though need for large volume of irrigation fluid can be a limiting factor.

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1.5: BURN INJURY IN ADULTS

SUMMARY

From October 1991 to July 1993, a total of 31 cases of burn injury were managed while the author was rotating in the three surgical firms at Muhimbili Medical Centre. The age range was 15 to 50 years with a mean of 29 years. There were 18 males and 13 females, 71 percent of the patients constituted the age group between 15 and 30 years (Table I). Hot liquids and open flames were the commonest causes of the burn injuries. All the 31 patients had either second or third degree burns with the total burn surface area ranging from 10% to 45%. The mean was 15% (Table II and IV) respectively.

The treatment offered included initial resuscitation with fluid replacement using mainly crystalloid, wound care using the open method and in three of the patients early split skin grafting was necessary. There were 3 deaths thus giving a mortality rate of 9.6%. These involved two females and one male who had more than 35% burns. Staphylococcus aureus was the commonest isolated organism in wound sepsis (Table VII). The duration of hospitalization ranged from 4 to 30 days with a mean of 14 days before discharge.

INTRODUCTION

Burn injury can be one of the most serious and devastating forms of trauma that man can sustain. Millions of people around the world are hospitalized for the treatment of burns each year and thousands die.[1,5] Destruction of the skin by heat results in severe local and systemic physiological alterations with a very marked endocrine response. Knowledge of the burn injury has increased rapidly over the past few years and management of patient

has progressively changed.[1,2,3] Despite these advances, many major burned patients (i.e more than 25% surface area), have a stormy course and complications stemming from the injury as the major cause of illness and death. In managing a burn victim great skills and understanding of the pathophysiology, diagnosis and treatment is required not only of the local skin injury but also of the derangements that occur in the hemodynamic, metabolic, nutritional, immunological and other homeostatic mechanisms.[1,3,4] Proper patient care demands that the burn wound, the accompanying systemic changes and alterations produced by treatment be conceptually viewed as dynamic interrelated processes. Attention to the burn wound must not take precedence over the life-saving support of other systems in the burn wound victim. The first essential in treating a burn victim is to determine the severity of the injury in that individual and then to decide the level of expertise necessary to care for him. The ultimate determinants of the severity of the burn injury are the volume of tissue destroyed and the percentage of surface involved. The daily cost of care for a burn victim remains tremendous, and the successful treatment of the victim means not only survival but also the return to society as a functional human being.[1,3] With these facts in mind and considering burn injury to be one of the common problems in our society, I was prompted to conduct this study.

CASE REPORT

A.M. (IP NO.693858) was a 25 year old female who was admitted in ward 2, two hours after she had sustained thermal injury involving the head and neck, the front of the trunk and the upper limbs. She had been lighting a kerosine lamp when it suddenly explode up,

bursting into open fire. The past medical history was uneventful, and the review of the systems was not remarkable.

On examination, she was in agonizing pain but was alert and fully conscious. She had no pallor; her temperature was 37.4°C. She was noted to be moderately dehydrated. The pulse rate was 110 beats per minute of thin volume but regular. The blood pressure was 85/55 mmHg. There was extensive blistering on the front of the trunk and on both upper limbs. The face was swollen and the skin hyperemic. The areas burnt included parts of the head, neck, upper limbs and the anterior aspect of the trunk were estimated to be 25 percent of the total body surface of the patient. The heart and lungs were clinically normal. The diagnosis of 25% second degree burn with hypovolemic shock was made. Her body weight was 60 kg. Initial resuscitation of the patient was started as follows:– A wide bore cannula was inserted into the right great saphenous vein to establish the route for intravenous fluid administration. The fluid requirement for the first 24 hours post burn was calculated using the modified Brooke formula of 2ml per kg body weight per percentage burn. So the amount of fluid requirement in the first 24 hours came to 3 liters plus 3 other liters for the ongoing and obligatory losses. During the first 8 hours from the time she sustained injury a total of 1.5 liters of Ringer's lactate was given as part of replacement fluid therapy, similarly another 1.5 liters was given in the next 16 hours. In addition to this amount of fluid, 3 liters of 5% Dextrose solution was given to maintain the ongoing and the obligatory losses, the amount was divided equally every 8 hours. The intravenous fluid therapy was monitored by recording the pulse rate blood pressure, and

by the hourly urine output obtained from a urethral catheter in the bladder. The vital signs stabilized within acceptable range and the hourly urine output in the first 24 hours averaged 40 ml per hour.

She was also sedated by using 100mg intravenous Pethidine at intervals of 8 hours PRN for the first 24 hours. A shot of Tetanus toxoid 0.5 ml intramuscularly had been given on admission at the casualty. The burnt area was cleaned with savlon solution, the ruptured blisters were snipped with scissors and the peeled skin removed. Sofratulle gauze were then applied on the burn wounds. A cradle was put over the patient to prevent bedding getting in contact with the burnt area. The patient was similarly nursed under a mosquito net.

Serum electrolytes were estimated after the first 24 hours revealing Na^+ 145, K^+4.5 and Cl^- 104 mEq/L respectively. The haemoglobin value stood at 10.6g/dL. Intravenous fluids were continued for another two days, the requirements being controlled by hourly urine output, aiming at a minimum of 0.5ml/Kg/hour. No colloids or blood were deemed necessary for the patient.

On the third post burn day, the local edema had subsided, the appetite had improved, the drip was discontinued and the catheter removed. The patient was allowed to take a high protein-energy diet and drink copious amounts of oral fluids. Cleaning with salon and sofratulle dressing had been continuing daily in the ward and by the end of the seventh day most of the burn area was drying and healing process proceeded fairly well except-for a small septic area in the right axillary fold. However it also cleared and dried up by

the beginning of the third week. She was discharged on the twentieth day post burn, having good granulation tissue. She was then seen at the surgical clinic two weeks after discharge and all the wounds had healed. Was subsequently discharge from follow up clinic.

The following are the profile for the 31 patients in this study as summarized by the tables below:–

TABULAR SUMMARY

TABLE I: DISTRIBUTION OF PATIENTS BY AGE AND SEX

AGE IN YEARS	S E X		TOTAL NO. OF PATIENTS
	MALE	FEMALE	
15 – 20	7	5	12
21 – 30	4	6	10
31 – 40	4	2	6
41 – 50	3	0	3
50+	0	0	0
TOTAL	18	13	31

- The age group between 15 and 30 years constituted 71% of all patients.

TABLE II: PATIENTS DISTRIBUTION IN ACCORDANCE OF THE CAUSES OF BURN

CAUSES	NUMBER OF PATIENTS		TOTAL
	MALE	FEMALE	
Scalds	10	10	20
Open flame	6	3	9
Electrical	2	0	2
TOTAL	18	13	31

TABLE III: DISTRIBUTION OF PATIENTS IN RELATION TO NATURE OF OCCURRENCE OF THE BURN INJURY

NATURE OF OCCURRENCE	NUMBER OF PATIENTS	PERCENTAGE
Domestic accident	25	80.6
Assault	2	6.5
Road traffic accident	2	6.5
Industrial	2	6.5
TOTAL	31	100

NB: It is pity to note that most domestic accidents occurred due to someones negligence or carelessness.

TABLE IV: PATIENTS DISTRIBUTION IN ACCORDANCE TO ESTIMATED BODY SURFACE AREA OF THE BURN

SURFACE AREA IN %	NUMBER OF PATIENTS
Up to 10	5
11 – 20	20
21 – 30	3
31 – 40	2
40+	1
TOTAL	31

- Wallace's rule of 9 was used to estimate the burnt surface area.
- Mean percentage of surface area burn was 15.

TABLE V: PATIENTS DISTRIBUTION IN ACCORDANCE TO THE LOCATION OF BURNS (SITE)

LOCATION/SITE	NUMBER OF PATIENTS
Upper extremities	12
Lower Extremities	9
Head and Neck	5
Trunk	1
Multiple sites	4
TOTAL	31

- 6 patients had either one or more big joints (knee ankle and elbow) being involved.

TABLE VI: PATIENTS DISTRIBUTION IN ACCORDANCE OF THE DURATION OF TIME BETWEEN ACCIDENT AND ADMISSION

DURATION IN HOURS	NUMBER OF PATIENTS
0 – 2	9
3 – 8	20
9 – 24	1
> 24	1
TOTAL	31

- Most delayed cases had smaller percentage involvement ranging between 8% and 12%

TABLE VII: ISOLATED MICRO-ORGANISMS FROM SWAB CULTURES IN SEPTIC BURNS (n = 15)

MICRO-ORGANISM	NUMBER OF PATIENTS
Staphylococcus aureus	8
Pseudomonas aeruginosa	2
Klebsiella specie	1
Escherichia coli	1
Not reported	3
TOTAL	15

DISCUSSION

Burn injuries are among the most serious injuries man can incur, and remain a major health problem throughout the world.[1,3] Burns are caused by the application of heat to the

body. The most common heat sources are open flame and hot liquid.[3] Other include electrical, cold, chemicals and fumes. In this study thermal injury was the commonest form of injury with scalds and open flame accounting for 29 patients. Only 2 patients sustained electrical injury which however were not of severe type (Table II). No patient had sustained chemical, cold or sun and heat overexposure injuries.

PHYSIOPATHOLOGIC RESPONSES

For easy understanding; the pathophysiology of burn injury can be divided into two major events depending on severity of the injury.[1,6]

I. The local response to burn injury, and

II. The systemic response or reaction to burn injury.

THE LOCAL RESPONSE TO BURN INJURY

Following temperature rise above 40°C tissue destruction and cytologic damage occurs as a result of direct heat transmission and denaturation of protein by rising temperature. However many of the changes are reversible except if temperature exceeds 45°C. At this temperature thermolabile enzyme systems are blocked and when the enzyme activity decreases to 50% of normal, cell death ensues[1]. The response of the cells involved in burns is neither uniform nor static. Since additional trauma, edema, decrease vascular supply and or bacterial invasion will destroy cells that initially survive the thermal insult. In addition to cell damage, there are both immediate and delayed vascular and cellular inflammatory responses at the burned site. Accumulation of fluid in the burn wound is a prominent feature of thermal injury. The fluid and electrolyte shift within the wound

is related to the type and severity of the injury. This appears to be secondary to a series of mediators released following the thermal insult.[1,7,8] They include vasoactive amines, prostaglandins, thromboxanes and leukotrienes. Immediately, a spasm of venules, vasoconstriction of arterioles and dilatation of capillaries occur. Capillaries and venules become permeable in the burned area allowing leakage of fluid, electrolytes, and proteins. The immediate response is followed within 6 hours by a delayed response with venular dilatation and further capillary permeability. The most rapid fluid loss occurs during the first 12 hours post-burn and is accompanied by cell margination and emigration as seen in inflammation from other causes.[1,6] Nonburn tissue edema is attributed to severe hypoproteinemia caused by protein flux into burn injured tissue. Interstitial protein depletion in nonburn tissue also increases the ease of water transport into the interstitial space.[8] Another cause of loss of fluid into the burn wound (besides capillary permeability) is the breakdown of the cell membrane by the thermal injury which injures the sodium pump. This results into an increase in intracellular Na^+, water and an efflux of K^+. There is a local cellular response accompanying the fluid response. Neutrophils emigrate from vessels into the tissue and are attracted by chemotaxis to the site of the injury. Monocytes migrate into the area and tissue macrophages differentiate in the area as needed for repair. In general the local effects amount to impairment of the functional integrity of the skin.

THE SYSTEMIC RESPONSE OR REACTION TO BURN INJURY

As the above discussed events are occurring at the local site of burning, systemic changes

also take place. The functions of all organ systems will eventually be altered due to the effects of a major burn. Some of the changes are related to burning it self, whereas some are in response to the stress of the injury. The reactions involve the nervous system and endocrine activity. They promote metabolic changes, alter cardiovascular mechanisms, and induce effects on renal function, leukocytes, coagulation and fibrinolytic mechanisms, and probably all body systems.[6]

1. Cardiovascular response

Among the most immediate and most severe changes are those in the heart and blood vessels. Although this was originally thought to be due to an unknown myocardial depressant factor, it now appears to be secondary to changes in the interstitium in the wound and adjacent unburned tissue.[1,6] There is an increase in interstitial oncotic pressure due to release of osmotically active cellular elements. When this osmotic gradient occurs across an injured basement membrane, massive oedema can occur.[3,7,8] As blood volume and plasma volume fall, further decreases in the cardiac output occur until levels of as little as 20% of normal are reached. This initial cardiac effect appears to be self-limited, and the cardiac output returns toward normal within 36 hours in patients surviving their burns.[1,8] Added to these losses specific for the burn injury is the obligatory fluid loss by respiration and urine. The fluid loss results in hemodynamic instability or burn shock. Besides the fluid losses progressive anaemia occurs beyond that predicted in response to direct red-cell destruction by heat. Red-cell survival is decreased and the normal

hypoxic stimulus for hematopoiesis appears to malfunction.[1,10] Red-cell trapping by the reticuloendothelial system and losses through capillary permeability and thrombus formation further diminish the red-cell mass.

2. Renal response

Renal function is altered in the burn victim as in any trauma victim.[1] The posterior pituitary releases ADH, causing maximal reabsorption of water in the renal tubules. Maximum sodium reabsorption occurs simultaneously because of aldosterone release from the adrenals. This combination results in excretion of a small amount of concentrated urine containing a decreased sodium concentration.[1,14] Similarly the injured cells in the burn wound increase amounts of myoglobin, haemoglobin and toxic products which are presented to the kidney. If an adequate glomerular filtration rate is not maintained, or if renal ischaemia occurs, these products cannot be effectively removed and acute tubular necrosis ensues. This is the basis of renal failure which was previously a common accompaniment of burn shock. If volume losses are replaced by a similar fluid and volume to that being lost, renal blood flow is maintained. It has been shown that a urine rate of 30-50 ml/hr in an adult and 1.2 ml/kg/hr in a child are adequate for the kidney to remove the toxic breakdown products and maintain function during the period of hemodynamic instability.[1,3,6-9,12-14]

3. Pulmonary response

The pulmonary system is exposed to a lack of oxygen secondary to combustion

and this may be aggravated by inhalation of noxious stimuli at the time of injury. The hypoxia is a stimulus for release of injurious free oxygen radicals as well as arachidonic acid metabolites.[1,9] As the burns victim becomes edematous and his cardiac output falls, the vascular space contracts leading to a ventilation: perfusion imbalance. This leads to inadequate peripheral perfusion and lactic acidosis. The prolonged poor perfusion results in cell damage and thrombosis in the capillaries.[1,6] Unless severe smoke inhalation occurs, there does not appear to be altered permeability in the lungs so lung water is usually not increased. This allows large volumes of resuscitation fluid to be used without risking pulmonary oedema. On the other hand, all burn victims show a degree of hyperventilation. Ventilation is about twice normal with minute volumes up to 14L at a ventilatory rate of 20/min. This is due to increased oxygen needs and a ventilation perfusion imbalance.[1,3] In addition, burns of over 40% total body surface area develop restrictive disease with a mild to moderate decrease in lung volume, a decreased vital capacity and an increase in pulmonary resistance.[6]

4. Neuroendocrine response

After burn trauma, a very marked endocrine response occurs. Their response influences very much the postburn metabolic changes and participates in the integration of the body's response with the nervous and immune systems.[10] Adrenaline and noradrenaline are released in great amounts following thermal trauma.[1,10] This appears to be a protective mechanism, since the mortality is

higher in burn victims who cannot sustain this increased response. The adrenal cortex also responds with increased secretion of 17–hydroxy corticosteroids.[1] These corticosteroids remain elevated as in a standard stress reaction until the patient is healed. Mineralocorticoids, specifically aldosterone, increase to conserve the decreasing Na^+ lost to the third space at the time of injury. This helps to prevent hypovolemia by conserving Na^+.[1,8,10]

5. Metabolic – nutritional response

The metabolic rate of the burn victim is greatly accelerated. Oxygen consumption is increased and Nitrogen losses magnified.[1,6] Associated with this hypermetabolic response are protein catabolism, ureagenesis, lipolysis and increased gluconeogenesis. This is the "flow phase" of metabolism originally described by Curthbertson.[1] Bacterial infection accentuates but does not precipitate the phenomenon.[6] As the water–holding lipid in the skin is destroyed, evaporative water losses increase at a tremendous energy expense. The resultant cooling of the body results in shivering, thus adding to the energy demand.[1] However, these mechanisms do not totally explain the hypermetabolism, and prevention of the evaporative water loss by external heat or impermeable dressings which does not eliminate the hypermetabolism. In burned patients the situation is complicated by anorexia and the calorie requirements for its evaporation. In such circumstances the hypermetabolic state following burning results in profound catabolism with decreased glucagon: insulin ratios and extreme intracellular cation alterations.[1]

Occasionally hyperglycemia persists and gross glycosuria produces osmotic polyuria which may culminate in dehydration, hypernatraemia, and coma.[6] Intracellular Na^+ concentration rises and will remain high unless at least 5000 calories per day are administered. The negative nitrogen balance necessitates the nitrogen requirement to be as high as 20g per m^2 body surface burn/day during the first month postburn.[1] A diet containing a calorie: nitrogen ratio of 100:1 appears to be effective in countering the hypermetabolic response, similarly it can be lessened by administration of high dose morphine, anaesthesia and early wound closure.[1,3,6,9,13]

6. Gastrointestinal response

The initial response of the gastrointestinal tract to a major burn is a reflex ileus due to splanchnic vasoconstriction.[1] If decompression is not instituted, acute gastric dilatation, vomiting and possible aspiration occurs. Gastroduodenal mucosal ulceration is a frequent occurrence in the burns victims. This may not always be clinically evident.[1,2] The ulceration appears not to be due to true hyperacidity. Gastrin does not appear to be increased in the burn patients. Liver function is also altered following thermal injury. These changes are probably due to altered circulation, hypoxia and the effects of toxic waste products.[1,3]

7. Immune response

Immune defenses are activated in response to the burn injury, however, some of these defenses are altered.[6,11] There is no doubt that tissue and cell–mediated

immunity are impaired after burn injury.[6] Neutrophil chemotaxis is compromised by decreased perfusion caused by hypovolemia and formation of microthrombi.[11] Chemotaxis and phagocytosis are dependent on complement components which are unfortunately reduced in patients with large burn wounds. Similarly the neutrophil intracellular killing power is reduced as oxygen delivery to the wound is decreased. Humoral immunity is altered with the drop in IgG levels. Cell mediated immunity is depressed and T cell lymphocyte counts are decreased. Suppressor T cells are generated, and a decrease in interleukin–2.[6,11,15] Sevitt[6] in his extensive review of the complications of burns notes that the actual reason for the immunodepression is uncertain though he is tempted to relate it to the prolonged secretion of cortisol and to a quantitative change in the lymphocyte population.

8. Psychiatric response

With major trauma,victims respond to the psychological stress in individualized patterns. These patterns may be more dependent on the patients pre–burn personality than on a response specific for burning. Situational depression is a uniform response, but true psychosis is unusual.[1,3] Temporary toxic psychotic symptoms accompany the other physiological alterations such as shock, hypoxia, water imbalance, electrolyte disturbance and sepsis. Iatrogenic factors such as inadequate relief of pain and anxiety, injudicious use of drugs and sleep deprivation can all trigger various responses of the psyche.[1]

Finally it is important to remember that the systemic effects are rapid in onset, prolonged in duration, dramatic in intensity and fatal in outcome if not promptly treated. In this study patients presented with most of the features discussed above especially those who had a total burnt surface area of more than 15%. The psychic response was observed only in one patient.

MANAGEMENT

First aid and the initial treatment of the burn victim is contingent upon proper evaluation and understanding of the local and systemic pathophysiological alterations. Early resuscitation with total replacement of the lost fluid and electrolytes form an integral part in the management of burn patients. The treatment of severe burn is usually complex and requires a collective team work of a number of hospital workers.

1. Evaluation and estimation of burn injury

A detailed physical examination should be performed as for any trauma patient, but a few points deserve special attention. The weight of the patient upon admission is very important in assessing fluid status in the next few days. Careful evaluation for associated injuries should be made.[1,12] The extent and depth of the burn should be estimated and documented. This is often more difficult than it appears, especially for those unaccustomed to the task. The distinctions between first, second and third degree burn becomes much clearer with time, so repeated evaluation after one or two days is advisable.[1,2,4,12]

The most commonly used classification of burn depth is one that divides burns

into first, second and third degree. First degree burns, such as sun burn are characterized by red color, small or absent blisters, painful skin, and healing within 3 to 6 days. Second degree burns are usually pink or mottled red with large bullae. They are painful and take 10 to 20 days to heal. The major distinguishing characteristics of third degree burns is that they do not heal spontaneously and require skin grafting. Third degree burns, also called full thickness burns, are usually white, charred, dark red or parchment like. The surface is dry, often with thrombosis of superficial vessels and is insensate. In contrast to full thickness burns, first and second degree burns are referred to as partial thickness burns implying potential for healing.[1-4] The extent of the burn is measured on a percentage of the total body surface area. The simplest method is the "Rule of Nines" as described by Wallace, in which each body region is counted as a multiple of nine percent total body surface area.[1,12] Each arm and the head are 9, each lower extremity is 18, the anterior chest is 18 and the back is 18. The genitalia is 1 percent.

2. Emergency and resuscitative fluid management

Establishing a patent airway, providing adequate ventilation, oxygenation, and supporting circulation are the initial priorities. After addressing any immediate life threatening problems, other steps in resuscitation may begin.[1,2,12] Minor burns can be totally treated in an ambulatory setting.[1] For these patients, the treatment consists of tetanus prophylaxis, burn wound infection prophylaxis and

analgesia.[1,2,4] The wound needs only to be cleansed with a bland soap and carefully dressed. Topical antibacterials are unnecessary.[1,2,4]

Moderate and major burns require hospitalization. Immediate treatment of the impending vascular collapse is begun by introducing a large bore canula into a peripheral or central vein, preferably through unburned skin. Patients with burns of 15% or more should have fluid resuscitation as soon as possible.[2,4] If a cut down is necessary, the area should be carefully prepared and the integumentary wound left unclosed to prevent infection. A nasogastric tube should be inserted to prevent gastric distention, and a urinary catheter should be placed to quantitate urine output on hourly basis.

Fluid resuscitation is one of the major components of initial burn management. Initial guidelines to the amount and rate of fluid replacement are provided by the weight of the patient and percentage of the total body surface area injured.[1,2,4,12,13] To determine the resuscitative fluid replacement, one must recall that the greatest loss of fluid occurs during the first 8–12 hours postburn and then continues more slowly over the next 12–16 hours.[1–4] Because of the capillary permeability, colloid replacement seems to be of no benefit in the immediate postburn period. Osmotic pressure cannot be built up over a freely permeable membrane. Therefore, since sodium seems to be the ion that is lost to the circulation in disproportionate amounts, Na^+ and not colloid appear to be the key to resuscitation.[1]

A number of fluid formulas have been developed such as Evans, Brooke, and Parkland formulas. Each has been used successfully on a large number of patient.[1-4,8,12-14] All formulae aim to give approximately 0.52 mmol of sodium/kg body weight per percentage body burn.[1] In order to compensate the obligatory loss from the vascular compartment this must be given at a rate exceeding 4.4ml/kg/hour. When the sodium ion is replaced in this amount and at this rate, cardiac output returns to normal by 24 hours postburn.[1,2,4]

The Parkland formula, probably the simplest to use,is calculated by multiplying 2-4 times the weight in Kg's times the burn size. The number obtained is the amount in ml to be given in the first 24 hours.[1,2] One half should be given in the first eight hours and one half should be given in the second sixteen hours.

It is important to recognize that these formulas are estimates, thus adequacy of fluid and proper electrolyte resuscitation is also determined by clinical observation of the patient's vital signs, urinary output and general condition.[1,12] A urine output of 30-50 ml/hour in the adult or 1.2ml/kg/hour in the child in the best guidance and monitoring parameter. If the urine output does not reach these levels the fluid input must be increased. Urine flow in excess of these parameters prior to mobilization of the oedema reflects fluid overload.[1]

Following return of the cardiac output at 24 hours, there remains a plasma gap. This amounts to approximately 0.35-0.5 ml/kg/% body burn.[1,2,4] By 24 hours capillary integrity returns and the starling's hypothesis appears to be restored.

Therefore, colloid can be used to replete the plasma volume.[1] By 30 hours, both the cardiac output and plasma volume should be returned to normal and effective resuscitation completed.

3. **Metabolic and Nutritional needs**

To overcome the negative nitrogen balance attendant with the major burn at least $20g/m^2$ of nitrogen per body surface area burn/day during the first month post injury and approximately 15g of nitrogen/m^2/day during the second month must be given to the patient.[1,2] Rapid wound closure decreases this loss since once this is achieved, the protein loss falls to $4g/m^2$/day. In addition to the protein needs, non protein calories must be provided. These can be determined by giving 25 Kcal/kg body weight plus 40kcal/% body surface area burn to the adult. If more than 5000 cal/day are required, a tube is placed in the stomach and, as soon as the ileus is resolved. Otherwise parenteral hyperalimentation is to be considered. Vitamin supplementation and fat must also be provided.

4. **The burn wound care**

Attention should be directed to the burn wound itself only after resuscitation has effected hemodynamic and respiratory stability.[2] However the proper treatment of the wound begins with doing no harm to the injured cells so that any tissue still viable after the initial thermal event can survive.[1,2]

Full thickness excision and coverage of burn wounds above 15–20% have been reported.[1–4] However, these are associated with increased blood loss, frequent

episodes of sepsis and, except in rare reports, a high morbidity and mortality.[1] As the burn wound exceeds 35–40%, full thickness excision and autografting become an arithmetic problem. Available donor site for coverage decrease as the size of excision increases. The alternative to full–thickness is the intradermal or tangential excision. This method has the following advantages:–

i) Removal of only necrotic tissue.

ii) Salvage of injured tissue that would otherwise have progressed to necrosis.

iii) Preservation of the biological properties of the dermis and prevention of contracture by immediate skin grafting.

Tangential excision unfortunately offers nothing if the burn wound is large and full–thickness.[1] Infection prophylaxis of the burn wound is part of the local treatment of every wound. It aims at preventing an increase in colonization from exogenous transient flora or proliferation of resident flora to levels sufficient to cause burn wound sepsis. Two prophylactic measures have been mentioned. Tetanus immunization and systemic penicillin therapy during the oedema phase are both given to prevent infection. The next decision must be how to dress the burn wound. For this the options include:–

i) Excision with skin dressing as previously discussed.

ii) Occlusive dressings

iii) Exposure

iv) Semi open technique with topical chemotherapy application.

To be effective, the dressing must be carefully constructed and applied using meticulous aseptic technique. Following careful cleansing and debridement of the wound, an inner layer of fine mesh or impregnated gauze is placed next to the burn to allow drainage. The dressing remains intact to protect the wound and is only changed if wound exudate soaks through it or odour, similarly pain or fever demand wound inspection.

Exposure method which was introduced by Wallace in 1949, has several major disadvantages which prevent its universal acceptance.[1] As with the occlusive dressing method, the only bacteria from which the victim is protected are those from exogenous sources and these are not the major offenders.[1,2,4] Similarly the eschar develops cracks which break the theoretical barrier. In addition burn wounds treated by exposure method are speculated to become deeper over the first 24–48 hours.[1]

For the reasons above, the semi–open technique with topical chemotherapy has evolved, the goal with this method is to prevent the numerical proliferation of bacteria. Topical chemotherapy seems to be the only rational approach to prevent the numerical proliferation of microorganisms and therefore is the mainstay of infection prophylaxis in most centres today.[1–4,12,13] The present three most commonly used topical chemotherapeutic agents worldwide are:– Silver sulphadiazine, Silver nitrate solution, and Mafenide acetate. Regardless of the

chosen topical agent, its efficacy should be monitored by constant surveillance of the bacterial flora of the burn wound. The use of skin grafting to cover the wound surface is the best means of minimizing fluid and heat losses and protecting the body from infection. In cases where the donor site is very small as a result of extensive burn, other biological dressings may be needed. Isograft and homograft including amniotic membranes have been tried.[2,4] Circumferential wounds require more attention because the eschar can act as a tourniquet as it dries compromising the blood supply distally. Escharotomy in such cases is then required. Burn wounds of the joints, fingers and toes are better dressed singly with vaseline gauze or sofratulle, and active movements should be encouraged.[16]

5. Preservation of Function

Contracture is a normal component of wound healing.[1-4] The principles of prevention of contracture and preservation of function are practiced immediately upon admission. These include splinting at all time in the optimal position to avoid contracture. Positioning, splinting and range of motion exercises are begun immediately and continued until wound closure. However severe contracture are generally difficult to manage.

The development of hypertrophic scars may occur after discharge. The resultant scar overgrowth may inhibit function and often causes disfigurement, and ugly look. Pruitt et al[2] has reported reduction of hypertrophic scar formation following the application of conforming Isoprene splints and or elastic garments during the

convalescent period. Such splints and elastic dressings are recommended for use following hospital discharge for at least 6 months to prevent delayed development of hypertrophic scar. If Keloid and contructures develop then plastic surgery should be considered early to prevent psychological trauma to the patient. The scars should be protected from repeated trauma to avoid development of Marjolins ulcer, which is a possible complication.

CONCLUSION

Burns are a major cause of injury and are "fairly common" in our society. They are devastating injuries that presents a formidable management problem to most surgeons and physicians. The management of burned patients requires great skills and understanding of the burn wound. The treatment of patients with large burns is much more than a matter of fluid therapy and skin grafting, though these of course are very important.

Treatment is both long and expensive and the prognosis is closely related to the amount of injured tissue present. Surgery and intensive care are well established in the management of burns, however the fight against infection and septicemia is still difficult. In our centre the need for special burn unit and special trained personnel should probably be looked into, to ensure the best care of these patients.

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SECTION TWO
UROLOGY

2.1: MANAGEMENT OF BENIGN PROSTATIC HYPERPLASIA

SUMMARY

Over a period of two years commencing October 1991, to September, 1993 at total of 17 patients with benign hyperplastic prostate were seen and managed by the author in the three surgical firms at Muhimbili Medical Centre. All of the patients were elderly, with age range between 53 and 75 years. It was observed that the prevalence increased with age (Table I). Acute urinary retention and features of prostatism were the main mode of presentation (Table II). Initial management included catheterization to relieve retention, 13 patients had urethral catheter through, whereas in the other 4 patients a suprapubic cystostomy plus catheterization had to be employed.

Due to lack of adequate investigation facilities and increasing patient load, most of the patients had delay to definitive treatment which ranged from 0–18 months with a mean period of 6 months (Table V). Open prostatectomy was the main procedure employed mainly for training purpose to the residents; only six of the patients underwent endoscopic resection of the prostate. The result of surgery were good with both methods (Table VI). The immediate post operative complications comprised mainly of hemorrhage, wound sepsis and blocked catheter (Table IV and VII) respectively. There was one death which was not attributed to surgery. Histopathological studies of the adenoma was consistent with benign prostatic hyperplasia in 12 of the patients; 4 results could not be traced, and an incidental carcinoma of the prostate was found in 1 of the patients (Table VIII).

INTRODUCTION

Benign prostatic hyperplasia (BPH) is a common non malignant, age–related condition that affects most men who enjoy a long life. This disorder is most frequently seen in men in the 7th, 8th or 9th decades of life, but also occurs in the 6th and even 5th decades.[1] Its incidence increases with age such that nearly 85% of all men over the age of 50 years have an enlarged prostate[1–3] Although a great deal of work has been accomplished in the attempt to determine the cause of BPH, todate only theories exist the real aetiology is not known.[1,3–5] The importance of BPH is that it can cause urinary outflow obstruction which in turn leads to infection and other changes in the urinary tract. Acute retention of urine is frequently the symptom for which the patient first seeks medical attention. The diagnosis of this condition is mainly clinical, radiological studies help demonstrate the effects of obstruction.

The standard therapy for symptomatic prostatic obstruction remains prostatectomy by transurethral resection (TURP) or, by open surgery. In this study a patient with symptomatic BPH is presented and a review of literature on the subject discussed. Similarly the profiles of the seventeen patients are given in tabular summary.

CASE REPORT

P.M. IP NO. 724668, a 69 year old man from Mbezi Temboni was admitted to hospital with a history of inability to pass urine and lower abdominal pain of one day duration. Over the previous six months he had noticed that he was waking up at night more often to void and had progressively worsening dysuria, urgency and hesitancy. The urinary

stream was gradually noted to be thin and coiled, could not be improved by straining. There was no history of colicky loin pains, feverishness or hematuria. He denied having had urethral discharge or trauma to his urethra in the past.

On examination he was restless and in pain, was not anaemic or dehydrated and was afebrile. Was free of uraemic frost. His blood pressure was 120/70 mmHg with a pulse rate of 80 beats per minute of good volume. The abdomen was symmetrically distended over the suprapubic area extending to just below the umbilicus. It was tense, tender and dull on percussion. This was consistent with a full bladder. On rectal examination, the rectum was empty and the prostate was found enlarged, smooth, non-tender, firm in consistency with a freely movable rectal mucosa over it. There was no blood on the gloved examining finger. Examination of the external genitalia revealed no evidence of meatal stenosis or stricture of the urethra. The other systems were normal.

1% of Xylocaine Jelly was instilled into the urethra. Using sterile technique a 14CH well lubricated, Foley's catheter was gently negotiated into the bladder and 1,200 ml of clear urine emptied. A repeat rectal examination re-affirmed the enlarged lateral prostatic lobes. A catheter urine specimen was obtained and sent for routine examination and for culture and sensitivity test. The catheter was connected to a urine bag for continuous drainage. An impression of Benign prostatic hyperplasia with urine retention was made. The following investigations were done on outpatient basis.

1. Catheter urine analysis showed no sugar, albumin(+) no ova seen. The leucocyte count was 20/HPF.

2. Catheter urine culture yielded a significant growth of Escherichia coli sensitive to Gentamycin and Nitrofuradantoin, resistant to Ampicillin, Tetracyclines and Cotrimoxazole.

3. Full blood picture revealed

- Hb 10.6 g/dl
- Total white cell count 5700/ml
- Neutrophils 68%, Eosinophils 4% Lymphocytes 28%
- Normochromic normocytic blood film.

4. Serum creatine: 84 mmol/L

5. Serum alkaline phosphates 92 IU/L, and acid phosphates 10.0 IU/L.

6. Plain X-rays of the chest and pelvis were both normal.

7. ECG: sinus rhythm.

After the above work up the patient was booked and admitted for prostatectomy. His blood was typed and found to be group AB positive and 3 units of blood were requested for the operation. A pre-operative review by the anaesthetist declared the patient to be fit for the operation.

RETROPUBIC PROSTATECTOMY

Under spinal anaesthesia, the patient was placed on the operating table in the supine position with a slight Trendelenburg tilt. The abdomen was cleaned and draped; light directed to the lower abdomen. A lower midline incision was employed to develop the retropubic space and expose the bladder extraperitoneally. The bladder was identified by

the numerous blood vessels running over it and the peritoneum was pushed gently cephalad. Hemostatic stitches of "O" catgut were placed to vessels encountered in the anterior surface of the prostatic capsule and ligated. The capsule was then opened transversely using a diathermy knife in between the stay sutures. Through this incision a cleavage plain between the adenoma and the capsule was established and subsequently using the index finger, the adenoma was enucleated by sweeping the finger all around the circumference, taking care to keep in the plane between the capsule and adenoma. The prostatic urethra was nipped with the finger just proximal to the verumontanum and the prostate removed. Hemostasis was achieved through hot tamponade, coagulating diathermy and ligation. Having secured hemostasis, the bladder cavity was inspected and found normal. A three way 22CH haematuric Foley's catheter was then introduced per urethra; ballooned with 30 ml of water and impacted in the prostatic cavity. Irrigation was started through one of the openings and the other one connected to a urine bag. The prostatic capsule was then repaired water tight using number "O" chromic catgut continuous suture. A corrugated rubber drain was inserted into the cave of Retzius, then the wound closed in layers using catgut and interrupted 2/0 silk to the skin. Estimated blood loss was about ml. Post-operative management included monitoring of vital signs 1/2 hourly, continuous bladder irrigation using normal saline and sedation with pethidine 100mg I.M. 8 hourly for the first 24 hours. He had also two units of blood transfusion and thereafter continued with intravenous Dextro-saline drip 3 L in 24 hours to ensure a good diuresis. Prophylactic Ampicillin was given intravenously, just before surgery and

was continued for 48 hours after surgery. The drip was discontinued on the second post-operative day, whereas irrigation was continued for 4 days. The drain was removed on the third day and the skin stitches on the seventh day; the wound had healed well.

The urethral catheter was removed on the eighth day and the patient was discharged on the tenth day.

At follow up for a period of six months, the patient had no complaints. Histological report on the specimen confirmed nodular hyperplasia of the prostate.

The following is a tabular summary of the features of all the 17 patients studied:

TABULAR SUMMARY

TABLE I: DISTRIBUTION OF THE PATIENT BY AGE (n = 17)

AGE (YEARS)	NUMBER OF PATIENTS	PERCENTAGE
40 – 50	0	0
51 – 60	4	21.4
61 – 70	6	42.8
71 – 80	7	35.7
TOTAL	17	100

- No patient was aged below 50 years.

TABLE II: PRESENTING SYMPTOMS

SYMPTOM	NUMBER OF PATIENTS
Acute retention	15
Frequency	15
Poor stream	14
Hesitancy	8
Urgency	6
Dysuria	5
Hematuria	2
Chronic retention	2

- Most patients presented with acute retention and symptoms of prostatism.

TABLE III: ASSOCIATED DISEASES/CONDITIONS

DISEASE/CONDITION	NUMBER OF PATIENTS
Hypertension	4
Inguinal Hernia	3
Uremia	1
Bladder Stone	1
Heat failure	1
TOTAL	10

TABLE IV: TIME OF SURGERY AFTER INITIAL PRESENTATION (n = 17)

TIME (MONTHS)	NUMBER OF PATIENTS
0 – 6	11
7 – 12	4
13 – 18	2
TOTAL	17

- Most of the patients were operated within six months of presentation to hospital.

TABLE V: TYPE OF OPERATION AND OUTCOME (n = 17)

OPERATION	OUTCOME		TOTAL
	CURED	DIED	
Endoscopic resection (TURP)	6	0	6
Retropubic	5	1	6
Transvesical	5	0	5
Transperineal	0	0	0
TOTAL	16	1	17

- Operative results were good for both Endoscopic and open prostatectomy.

TABLE VI: COMPLICATIONS OBSERVED

COMPLICATION	MODALITY OF TREATMENT			TOTAL
	TURP	RETROPUBIC	TRANSVESICAL	
Hemorrhage	1	3	3	7
Wound sepsis	0	2	2	4
Blocked catheter	1	2	2	5
Stricture	1	0	0	1
Recurrence	1	0	0	1

- Hemorrhage and blocked catheter were the commonest complications.

TABLE VII: FINAL HISTOLOGY OF THE GLAND (n = 17)

RESULT	NUMBER OF PATIENTS
Benign hyperplasia	12
Carcinoma	1
Not available	4
TOTAL	17

- Incidental carcinoma occurred in one patient which had been thought to be clinically a "benign" prostate.

DISCUSSION

INCIDENCE AND AETIOLOGY

Diseases of the prostate constitute much of the work load of the urological surgeon, none

more so than benign prostatic hyperplasia.[7] The development of benign prostatic hyperplasia is a worldwide problem; usually occurs in men over 50 years of age, most often between 60 and 70.[1,5-8] In Indians, prostatic enlargement is less frequent, and occurs more often in a younger age group.[6] Robbins[8] reports a 50–60% incidence in men between 40–59 years and more than 95% chance in those over 70 years of age. Hiza[9] noted in 1971 that prostatic hypertrophy presenting in the form of acute urinary retention was the second commonest cause of emergency admissions in Muhimbili Hospital. The condition is rare among the chinese males they have an incidence of only 6% after the age of forty.[10] The reason for these discrepancies is unknown. In this study 50% of the patients were between 70–75 years, however the youngest was 53 years whereas the median age was 67 years. (Table I).

Despite a vast literature, the aetiology of benign prostatic hyperplasia remains elusive. However a number of etiologic theories including Hormonal, Neoplastic, Inflammation and Arteriosclerosis have been advocated to explain the development of hyperplasia. The most favored of these is the hormonal theory.[3,4,6,11] According to this theory, as the age advances androgen production diminishes while the quantity of the oestrogenic hormone is not decreased equally. The prostate enlarges because of predominance of the oestrogenic hormone, thus the enlargement is regarded to be due to a disturbance of the ratio and quantity of the circulating androgens and oestrogen.[4,6,11]

The neoplastic theory postulates that the enlargement is a benign neoplasm.[6] As the prostate is composed essentially of fibrous tissue, muscle tissue, and glandular tissue, the

neoplasm is a fibromyoadenoma. The other postulates include that of inflammation and arteriosclerosis. Whereas chronic inflammation due to infection, or chemical irritation is speculated to cause retention of prostatic secretions leading to hyperplasia, the arteriosclerosis which can occur with advancing age is speculated to interfere with the prostatic blood supply to the peripheral one third leading to atrophy of the posterior lobe. This leads into compensatory hyperplasia of the inner two thirds.[8,12]

PATHOPHYSIOLOGY AND CLINICAL FEATURES

The clinical picture of prostatic hyperplasia parallels pathophysiologic changes. The disturbances which result from prostatic hyperplasia are secondary to its effects on the urethra, bladder and kidneys.[3,6,7,8,12] Prostatic enlargement may develop silently and its evidence of enlargement may be apparent only when a rectal examination is carried out for some other reasons. In those patients with early or very gradual prostatic enlargement, the disease produces no symptoms because the bladder is capable of compensating for the increased urethral resistance.[2,3,14] There are no alterations in micturition, the bladder empties completely on each voiding, and there are no symptoms of urinary tract difficulties. Such a situation is called "Silent prostatism" and is discovered only inadvertently during patient evaluation for unrelated problems.

The predominate symptoms associated with hyperplasia that prompt the patient to seek medical attention are those of disturbances of urination secondary to distortion and mechanical obstruction of the bladder neck and proximal urethra.[3,6,14] Characteristically the patient complains of diminution in the caliber and force of the urinary stream, and as

obstruction progresses there is hesitancy in initiating voiding, a feeling of incomplete emptying of the bladder and occasionally inability to urinate.[3,14] Incomplete evacuation of the bladder may be heralded by other symptoms such as increased urinary frequency, nocturia or incontinence. Residual urine predisposes to secondary infection with symptoms of painful micturition or strangury becoming part of the clinical picture. Nocturia is a similar index of the mechanical pressure of the enlarging prostate. It should be noted that nocturia normally occurs in the aged partially as a result of the inability of the kidney to concentrate urine. This results into excretion of larger nocturnal volumes. However, nocturia more than once or twice nightly in the elderly male suggests mechanical pressure of the prostatic enlargement as well as the possibility that the bladder is emptying incompletely.[3] This internal sphincter is stretched by the upward growth of the middle lobe and dribbling results. In its effort to overcome the obstruction, the bladder hypertrophies and sacculation form. This progresses to diverticula formation as urothelium herniates through widely spaced bundles of detrusor muscle. These diverticulae do not empty completely, thus dribbling suggests both residual urine and pooling of urine within the prostatic urethra.[8,14]

Urinary bleeding may first bring the patient to the attention of the physician. Hematuria may result from prostatic enlargement with engorgement of the small mucosal vessels covering the adenomatous gland, ruptured as a consequence of straining to urinate. Depending upon whether the source of bleeding is an intravesical or intraurethral prostatic lobe, the hematuria may be total, initial, or terminal in nature.[3,14] Only two of the study

our patients presented with hematuria, whereas most of the patients presented with acute urinary retention and features of prostatism (Table II).

The patient with residual urine secondary to Begin prostatic hyperplasia is always in danger of acute retention if he defers urination until the bladder is so distended that a detrusor contraction cannot be initiated. The diuretic and analgesic responses to alcohol and the effects of anesthesia and of anticholinergic and sympathomimetic drugs also increase the likelihood of acute retention.[2]

The presence of residual urine favors formation of vesical calculi as noted in one of the study patient (Table III). These may be associated with sharp twinges of pain, the "Curbstone" symptom, when the patient descends a stairway or steps down from the curb.[2,3] Some patients may present with symptoms that represent the secondary effects of the bladder neck obstruction rather than those of the local pathophysiologic changes discussed above. The clinical picture of this group of patients may be so unrelated to the urinary tract that it suggests primary disease in other body systems.[14] Asymptomatic bladder neck obstruction with bilateral hydronephrosis frequently mimics gastrointestinal or neurologic disease with its symptoms of nausea, vomiting, poor appetite, weight loss, malaise, apathy and stupor. Superimposed infection Upon the chronically obstructed upper tracts produces additional symptoms of chills, fever, general body aching, and upper abdominal or flank pain.[3,14]

Residual urine of 1000 ml or more may produce a palpable lower abdominal mass before the patient experiences any particular symptoms, and it is not uncommon to observe

bilateral hydroureteronephrosis and evidence of impending renal failure with azotemia and electrolyte imbalance.[3] Although the above features are commonly associated with hyperplasia, they are also found in other disease entities. This is particularly true of increased frequency of voiding and dysuria.

INVESTIGATIONS

These should include evaluation of the patient as a whole, because most of the patients with prostatic enlargement are elderly and so prove to have other associated medical illness (Table III). Cardiovascular, Respiratory systems, should be examined to exclude the cardiorespiratory complications of obstructive uropathy.

Electrocardiogram, fasting blood sugar, a blood urea estimation, a blood count are basically essential investigations. The urine should be examined for evidence of infection, and cultured. Also tested for presence of glucose. Blood urea, serum creatinine, serum electrolytes assess for renal status, while acid and alkaline phosphatase are necessary in suspicion of prostatic carcinoma with bone metastases.

Intravenous urogram may reveal evidence of hydronephrosis. It will also show the contour of the bladder and whether trabeculation, sacculation or a diverticulum is present. A film after micturition may reveal a significant residual urine. In the presence of renal failure urography is unlikely to be helpful.

With the progress made in ultrasound examination over the last few years, the size of the prostate can be assessed through a rectal transducer. Should doubt exist following ultrasound examination the topography of the urinary tract may then be demonstrated with

a urogram. A plain abdominal x–ray should never be omitted as may show the presence of a calculus in the kidney or the bladder. It will also show if there is bony metastasis in case of prostatic carcinoma.

Cystourethroscopy allows for inspection of the urethra, the prostate and the urothelium of the bladder which should always be made before prostatectomy regardless of the method. Presence of a urethral stricture, a bladder carcinoma and the occasional non radiopaque vesical calculus can be excluded by this method.

Before surgery the patient's blood group must be ascertained and matching for at least two units of fresh blood done. It is also worth while doing platelet count, bleeding and clotting time. In this study the main associated pathologies included, hypertension, Inguinal hernia, uraemia, bladder stone and heart failure. (Table III).

TREATMENT

The standard therapy for symptomatic prostatic obstruction has been prostatectomy by transurethral resection (TURP), or frequently, by open surgery.[1,2,3,6,7,14–17] However many types of nonsurgical therapy including balloon dilatation hyperthermia, adrenergic blockade, hormonal therapy and stetting have been used for the treatment of BPH. Patients deciding between different treatment choices need to know the likely outcomes of these alternatives, including chances for symptom responses and improved functional health and well–being.[3,7,16–18] It is important to note that some degree of nodular hyperplasia occurs in the prostate of every man over the age of 40, but only one in 10 ever needs an operation or call for any action, by way of either investigation or

treatment.[3,16,17] The crucial factor is outflow obstruction to the bladder. It is this which affects the health of the patient, and for this that treatment is needed. Before definitive treatment the patient receives symptomatic treatment depending on the mode of presentation.

The Management of Retention

Acute urinary retention is distressing and painful. It requires decompression of the bladder by the passage of a urethral catheter, or with its failure, a suprapubic cystostomy plus catheter. For patients with chronic urinary retention, which is painless, careful assessment is necessary; patients who do not have symptoms suggestive of co existent infection and who have a normal serum creatinine, do not necessarily required a catheter before proceeding with a prostatectomy. For those who are uraemic, decompression of the bladder is mandatory. Controversy exists over whether the bladder should be drained completely or decompressed slowly when dealing with chronic retention. Occasionally, bleeding can occur from rapid decompression of a chronically overdistended bladder but regrettably the diligent management necessary of slow bladder decompression is often not exercised, and the patient is left in a state of continuing retention under the misapprehension that good is being done. The use of a "gate" clip is only to be condemned.

Controlled drainage can be effected by joining the catheter to a sterile collecting bottle placed slightly higher, about 20cm above the patient. Drainage in this position is effected when there is increased intra abdominal pressure as in coughing, or straining. Thereafter

gradually the bottle is lowered for 2cm every hour till it is at a lower level than the patient. This offers an excellent gradual bladder decompression.

Uraemic patients with chronic retention are often dehydrated at the time of admission. Due to the chronic back pressure on the distal tubules within the kidney, there is loss of their ability to reabsorb salt and water. The result, following release of this pressure is an enormous outflow of salt and water which has become known as a post obstructive diuresis. It is for this reason that a careful fluid chart, daily measurements of the patient's weight and serial estimations of the urea and electrolytes are mandatory. Intravenous fluid replacement is required if the patient is unable to keep up with this fluid loss. These patients are usually anaemic and may require a blood transfusion once the fluid balance is stabilized.

In acute retention, after bladder decompression the catheter is removed and the patient is observed for urine retention. However more usually a catheter is left in situ. The catheter is then connected to a closed system drainage bag.

Relieving acute retention by catheterization has a risk of introducing infection into the bladder which may lead to urinary tract infection; thus strict adherence to aseptic technique needs to be employed. In this study 13 patients out of 17 had retention relieved by urethral catheterization. Only four patients had suprapubic cystostomy + catheterization as urethral catheter failed to pass through.

The treatment of prostatic hyperplasia is prostatectomy, which is only required if there is equivocal subjective or objective evidence of obstruction.[7,16,17] The natural history of

the disorder is unpredictable and it may improve or get no worse without treatment, and any form of prostatectomy may be followed by complications.[7,16]

Indications for operation include [3,6]

1. Prostatism – increasing difficulty in micturition, with considerable frequency day and night, delay in starting, and a poor stream.It must be stressed that frequency alone is never an indication for prostatectomy.
2. Acute retention which is unrelieved by passing a catheter, emptying the bladder, and immediately removing the catheter.
3. Chronic retention – a residual urine of 200ml or more.[6] A raised blood urea, hydroureter, or hydronephrosis demonstrated on urography, and uraemic manifestation.
4. Complications – including stone, infection, and diverticulum formation, which are often present in varying combinations.
5. Hemorrhage – occasionally, venous bleeding from a ruptured vein overlying the prostate will not stop with a catheter drainage, so prostatectomy must be performed as an emergency measure.

Operative treatment

The prostate can be approached:

(1) Transurethrally.

(2) Through the bladder (transvesical),

(3) Retropubically,

(4) From the perineum.

The transurethral approach is ideal for small glands up to 70 grams or median lobe enlargement.[6,7] Strips of tissue are cut from the bladder neck down to the level of the verumontanum. Cutting is established by a high-frequency diathermy current which is applied across a loop mounted on the hand-held trigger of the resectoscope. Coagulation of bleeding points can be accurately achieved and damage to the external sphincter is avoided provided one uses the verumontanum as a guide to the most distal point of the resection.

Hyponatremia is avoided by using 1.5% isotonic glycine for irrigation. The "chips" of prostate are then removed from the urinary bladder using an Ellik evacuator. A three – way, self retaining catheter irrigated with isotonic saline is then introduced into the bladder at the end of the procedure to prevent any further bleeding from forming blood clots. Irrigation is continued until the outflow is pale pink and the catheter usually removed on the 3rd postoperative day. The procedure has low mortality and morbidity thus it is well tolerated by the elderly debilitated patients. It also represents an excellent operation for the young patient who values sexual potency. The postoperative stay is usually 3 to 5 days in contrast to approximately 7 days for open prostatectomy.[2] The disadvantage of transurethral resection is that it requires a certain expertise which is not easily acquired and needs constant practice.[7] The patient is usually seen post operative at intervals to evaluate the character and frequency of voiding, since recurrence of symptoms as a result of fibrotic stricture or regrowth of the adenoma is a possible set

back. In this study one patient had recurrence necessitating a repeat procedure (Table VII).

* Transvesical prostatectomy is historically the first procedure to be generally used in benign prostatic hyperplasia.[3,14] It is performed through pfannenstiel or low midline incision. The exposed bladder extraperitoneally is opened longitudinally between stay sutures. This method has the advantage of exposure of lesions within the bladder cavity and dealing with them at the same sitting. With the index finger in the prostatic urethra the anterior commissure is broken, a cleavage plane is found and enucleation of the adenoma is done by sweeping a finger on each side taking care of the verumontanum. Hemostasis is achieved by applying ligatures to the bladder neck at 5 and 7 O'clock.

* In retropubic prostatectomy, the bladder is approached as in the transvesical method, the retropubic space is then bluntly dissected and developed and the prostate exposed. The capsule is then opened transversely between stay sutures using a diathermy knife, Then using the index finger the prostatic adenoma is enucleated. Hemostasis is achieved easily in this procedure and all the obstructive tissue is removed under direct vision. It has a low mortality and morbidity as compared with the other approaches of open prostatectomy.[3,7,15]

* Perineal prostatectomy is done through an incision made in the perineum infront of the anus. The rectum is retracted backwards to expose the posterior aspect of the prostate above the triangular ligament. This is incised and the adenoma enucleated. Perineal enucleation of the hyperplastic prostate is particularly suitable to the large, low-lying prostate.[3] The procedure offers the advantage of good control of bleeding and it is

thought that the extreme lithotomy position promotes venous return and minimizes operative vascular complications. Although the perineal procedure carries a lower mortality and morbidity than suprapubic prostatectomy, the duration for hospitalization is somewhat longer.[13,14,15] Rectourethral fistula complicating this procedure has an incidence of 1–3% and is most distressing.[3] The approach is obsolete in various centres and is unsuitable for high lying prostate. It is not popular in Britain because of the incidence of incontinence and delay in regaining normal control.[6] In this centre it is also never practised and the author has never seen one being done.

Alternatives to prostatectomy

Recently, interest in developing non surgical alternatives for the management of BPH has surged, and patients understandably are seeking less invasive approaches to their medical problems.[17,20] However the alternatives to prostatectomy are still limited. They include the following[16,17,19,20]

1. Balloons dilation.
2. Stainless steel or titanium stents to maintain the lumen of prostatic urethra
3. Vaporizing energy sources i.e. thermotherapy
4. Endoscopically guided laser systems
5. Use of 5 alpha reductase agent which interferes with conversion of testosterone to dihydrotestpsterone.
6. Use of selective alpha 21–adrenergic inhibitors.
7. Cryosurgery.

All these approaches seem to be associated with less pain and perhaps less morbidity than in a standard surgical procedure.[17,20] None of the above methods is currently available at Muhimbili Medical Centre.Physicians and patients must resist the temptation or the pressure to embrace these new treatments until safety, efficacy, and cost benefits have been clearly demonstrated. For patients who cannot stand an operation even with the safe modern anaesthesia, permanent catheterization is recommended. The catheters should be narrow, soft and changed frequently to reduce the risk of infection.

COMPLICATIONS

Complications following prostatectomy are many and varied and some follow the respective types of operations while others are general.

Primary hemorrhage was the commonest complication, noted in seven patients and five of the patients had catheter blockage due to clot retention. Syringing was successfully done and irrigation continued. It is noted in the literature that, hemorrhage can be caused by excessive fibrinolysis.[7,19] Secondary hemorrhage may occur any time after a week to six weeks and is due to infection. None was noted in this study. Wound infection occurred in 4 patients, the culture isolates revealed Enterobacilli which were sensitive to Septrin and Gentamycin. Wound infection is thought to be due to infected urine especially in long standing retention. However wound sepsis is high in patients who have had continuous bladder drainage than those who had intermittent drainage.[21]

Another complication associated with prostatectomy is stricture or bladder neck contracture mostly seen in transurethral prostatic resection. The degree of contracture is

dependent on the tissue destruction at the bladder neck.[8,19] This was noted in one patient who eventually had a repeat TURP.

All patients having a prostatectomy should be warned that they are likely to suffer from retrograde ejaculation; this occurs once the bladder neck is rendered incompetent. Some authors put an incidence of between 4–40% of patients as becoming impotent following prostatectomy.[3,19]

The absorption of water into the circulation at the time of a transurethral resection can give rise to congestive cardiac failure, hyponatremia and hemolysis. This is known as post–TURP syndrome.

CONCLUSION

Benign prostatic hyperplasia is a common problem since the number of geriatric patients increases significantly worldwide. Most of the patients we see present as emergencies with acute urinary retention.

Although endoscopic resection of the symptomatic BPH is the standard procedure, at this centre, residents have to be competent with open prostatectomy since very few up country hospitals are equipped with resectoscopes. Nevertheless TURP is a difficult to learn and difficult to teach, and if done badly gives bad results. The developing nonsurgical alternatives for the management of BPH should pass the test of time until safety, efficacy, and cost benefits have been clearly demonstrated.

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2.2: FOURNIER'S GANGRENE AS SEEN AT MUHIMBILI MEDICAL CENTRE

SUMMARY

Between October 1991 and July 1993; 8 patients with Fournier's gangrene were seen and managed by the author while rotating in the three surgical firms. Their ages ranged from 45 to 70 years with a mean of 50 years (Table 1). Each of the patient had nonspecific symptoms before the gangrene became evident. Sudden onset of scrotal pain and swelling accompanied with fever were the main mode of presentation(Table II). This was rapidly followed by gangrene with a demonstrable typical demarcation line between viable and dead tissue.

History and examination of the patients could not reveal the aetiology. The diagnosis was based on the fulminating clinical progression of scrotal gangrene and in six of the patients presence of multiple pathogenic organisms with Pseudomonas aeruginosa, streptococcus faecalis and Escherichia coli predominating from the culture (Table V).

Management included surgical debridement, daily wound toilet, antibiotic therapy using combination of chloramphenicol and Metronidazole, or depending on sensitivity pattern from culture isolates. In 3 of the patients subsequent skin grafting was necessary to cover the skin defect (Table VI). No death was encountered. Early diagnosis and aggressive medical and surgical attention is of utmost importance so as to avoid extensive tissue destruction, and possible complications of the disease such as toxaemia and shock.

INTRODUCTION

In 1883, Jean Alfred Fournier a French venereologist presented 5 cases of scrotal gangrene that were characterized by an idiopathic involvement of the genitalia in otherwise healthy young male patients. Since then this syndrome has borne his name.[1,2,3] Since that time many cases have been described and the syndrome has come to encompass all necrotizing subcutaneous infections of the male genitalia whether primary (Idiopathic) or secondary, i.e caused by pathological processes in the anorectal area, lower urinary tract, and scrotal or perineal skin.[1,3]

Fournier's gangrene is a rare but serious infection with toxicity that destroys soft tissues, initially in a single tissue plane.[1] The process starts from a devitalized fascia, undermines the skin and leads to progressive necrosis of subcutaneous tissues. Gangrene of the skin ensues late.[1,4,5] Other terms for this syndrome include "Synergistic gangrene of the scrotum and penis", and "Necrotizing fascitis of the male external genitalia.[1]

Most of the cases seen today differ from the classical description[1,2,3] in that, the patients are generally older, the onset less abrupt and the course more gradual(Table I and II), hence the use of the term necrotizing fasciitis of the genitalia would be more appropriate terminology.[4] Although necrotizing fasciitis is an extremely morbid disorder, the majority of patients will survive if diagnosed early, treated promptly and aggressively with antimicrobial therapy combined with immediate, and if required, repetitive surgical debridements.

The following gives an account of a patient with Fournier's gangrene admitted and managed in surgical firm II at Muhimbili Medical Centre.

CASE REPORT

S.K. was a 50 years old male patient referred from one of the city dispensaries. His presenting complaints on admission to ward 11 was swelling and burning pain in the genital area for the past three days. The pain had started suddenly in the scrotum and root of the penis. This was then accompanied by fever and progressive swelling of the genitals. He had taken some aspirin tablets but could not get relief and a few hours prior to admission he had two bouts of vomiting and was now anorexic. He denied previous history of trauma or discharge from the urethra and had no history of dysuria or hematuria. He had no problems with opening bowels. There had never been history of serious illness before such as Diabetes mellitus, TB, Schistosomiasis, Filariasis or hypertension. This was his first attack. He was a peasant, married to three wives and had a number of children and grandchildren.

On examination, the patient was ill looking, febrile and sweaty. Had mild pallor, was not dehydrated or dyspnoeic. Did not appear toxic and was not jaundiced. His axillary temperature read 39^0C, had a pulse rate of 120 beats per minute regular and good volume. The blood pressure was 110/70 mmHg.

The scrotum was swollen, appeared, shiny was hot and tender to touch. Inguinal hernial orifices were free, inguinal lymph nodes were not palpable.The perineum looked normal, rectal examination revealed a normal rectal mucosa, a normal size prostate, no rectal

masses, the gloved finger was stained with normal fecal matter. Examination of the other systems including the chest, abdomen and extremities were found to be normal.

A tentative diagnosis of Fournier's gangrene was entertained with a differential diagnosis of infected hydrocele. Aspiration with a wide bore needle revealed a clear serous fluid, which was set for cell and bacterial analysis. The patient was empirically started on chloramphenicol 500mg and Flagyl 500mg both orally, 8 hourly regime respectively, also scrotal support, analgesics and bed rest. Investigations taken included:–

1. Full blood picture: Hb 9g/dl, Total white cell count 10,000, Neutrophils 80%, Eosinophils 5%, lymphocytes 15% Hypochromic microcytic blood film.
2. Culture of blood and scrotal aspirate revealed no organisms.
3. Urinalysis: clear, Albumin (–), Sugar (–), Leucocyte 3/ HPF.
4. Blood slide for malaria revealed no parasites
5. Random blood sugar 5mmol/L.

On the 2nd day of admission gangrene set in and it was evident in the scrotum especially on the most dependent part, and was spreading towards the root of the penis. Two days later the gangrenous part was demarcating, it involved nearly two thirds of the anterior scrotal skin. The penis remained swollen but was not involved with gangrene. Debridement was done, excising all the necrotic and compromised tissue. The area was then cleaned with hydrogen peroxide and copiously irrigated with saline before a dressing with Eusol could be applied. Thereafter the patient had daily B.D. soaks of the exposed

testes in a warm soapy water lasting 10–15min, the intention being to achieve mechanical cleansing of the exposed testes and the wound area. Each day the testes were inspected, looked healthy and free from the gangrenous process.

The necrotic tissues were sent for culture and sensitivity and the patient was maintained on same antibiotics since the isolates were predominantly pseudomonas aeruginosa and Escherichia coli. Pain, scrotal swelling and fever subsided on the 1st week of treatment and the patient regained good appetite. On the 14th day following debridement, a healthy granulation tissue had formed and the patient was planned for skin grafting.

However this could not be done due to short–comings at our operating theater, subsequently the edges of remaining scrotal skin gradually started closing. At 10th weeks it had almost closed and the patient was then discharged home. On follow up, the patient had no complaints and the wound healed completely. The following tables below give summary of the eight patients studied:–

TABLE I: DISTRIBUTION OF THE PATIENTS BY AGE (n = 8)

A G E	NUMBER OF PATIENTS
< 45	0
45 – 50	4
51 – 60	3
61 – 70	1
TOTAL	8

TABLE II: PRESENTING FEATURES ON ADMISSION (n = 8)

SYMPTOM/SIGN	FREQUENCY
Scrotal pain	8
Fever	8
Scrotal edema	5
Cellulitis	4
Crepitus	3
Necrosis	1

- Most patients presented with scrotal pain and fever.

TABLE III: DURATION OF SYMPTOMS AT THE TIME OF ADMISSION

NUMBER OF DAYS	NUMBER OF PATIENTS
1 – 2	0
3 – 10	7
> 10	1

TABLE IV: PRESUMED RISK FACTORS

FACTORS	NUMBER OF PATIENTS
Alcoholism	2
Immunosuppression	1
Diabetes mellitus	1
No identifiable factor	4
TOTAL	8

TABLE V: BACTERIOLOGY

MICRO-ORGANISM	FREQUENCY IN PATIENTS								TOTAL
	P1	P2	P3	P4	P5	P6	P7	P8	
S. faecalis	+	+	+	+	+	+	x	x	6
P. aeruginosa	–	+	+	+	+	+	x	x	5
E. coli	+	–	–	+	+	–	x	x	3
NOT DONE							x	x	2

- NOT DONE = x

TABLE VI: MANAGEMENT OF THE GANGRENE

MODALITY	NUMBER OF PATIENTS
Debridement alone	3
Debridement & skin grafting	3
Debridement & secondary suturing	2
Recovery without surgical interference	0
TOTAL	8

TABLE VII: DURATION OF HOSPITALIZATION

DURATION IN WEEKS	NUMBER OF PATIENTS
< – 3	1
3 – 8	4
9 – 10	2
> 10	1
TOTAL	8

- Hospitalization lasted from 20 – 85 days with a mean of 45 days.

DISCUSSION

INCIDENCE AND AETIOLOGY

The doubtful aetiology of the condition and the limited number of studies carried out on its clinical features, complications and treatment are all reflected in conflicting published reports. Since Fournier described the condition in 1883, more than 500 cases have been reported.[1] Akinola[2] quotes Pande and Mewara in 1976 describing this ailment as ineffective gangrene, whereas Kearney and Carling in 1983 described it as necrotizing fasciitis.

The disease seems to be more prevalent in African and Asian countries, although the scarcity of recorded cases does not reflect its actual incidence.[1,3,4,5] In his series Akinola[2] attributes the ailment to low socio-economic communities where adequate social amenities are lacking; and amongst those with poor genital hygiene. This concurs with earlier observation by Fahal et al.[4] It needs no mention of the importance to search

thoroughly for the underlying cause so that this too may be treated appropriately as part of the immediate management of the case. A diagnosis of "Idiopathic" Fournier's gangrene should be accepted only after all possible underlying causes have been excluded.[2,3,7,9,15] In this study no apparent cause was found, however in few of the patients alcoholism, diabetes mellitus and immunosuppression were the predisposing or accompanying factors (Table IV).

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.[2,6,9]

Earlier reports suggested that synergistic gangrene was most often secondary to a combination of microaerophilic streptococcus and staphylococcus aureus. In 1945, Main reported that 30 of 44 patients yielded one or both of these organisms.[6] Recently, most investigators have isolated gram-negative bacteria (Escherichia coli, Klebsiella, and Pseudomonas) in combination with anaerobes, including especially the Bacteroides.[1-4,6,9] Usually the first gram stain shows a polymicrobial flora with predominating Gram-negative rods and gram-positive cocci. Bacteroides species, especially Bacteroides fragilis and Peptostreptococci are often cultured. In his series Bahlmann[3] had Bacteroides fragilis as the most common pathogen accounting for 88% followed by Escherichia coli and anaerobic Peptostreptococcus, respectively.

Other commonly cultured aerobes include Enterococcus, Staphylococcus, Streptococcus, Proteus, Pseudomonas and Klebsiella. Fahal and Hassan[4] in a study of 9 patients had Proteus as the commonest isolated organism. With improved culture techniques, an average of four organisms per patient are reported to be isolated.[6] This is in accord with other author's experience.[2,3,7] However, there is need for high index of suspicion for the anaerobes as they may constitute more danger and spread along the fascia planes and eventually, death.[2] It is important to remember that Clostridium organisms may also be responsible, particularly following trauma, or postsurgical infection. However according to Martti[1] and Pessa[8] Clostridia are only rarely seen in these infections. A gram stain displaying gram-positive rods with little inflammatory reaction should suggest the possibility of Clostridial infection.[6,8] Anaerobic bacteria act in synergism with the aerobes. It was first demonstrated by Meleney for staphylococcus aureus and microaerophillic streptococcus in progressive synergistic gangrene.[1] Staphylococcal hyaluronidase was one of the major factors in the progressive nature of the disease. Subsequently, bacterial synergism has been demonstrated for a variety mixtures of aerobes and anaerobes. 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.[1] 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 fasciitis which is responsible for subsequent tissue necrosis and gangrene.

The portal of entry of microorganisms is not always known, however most cases occur as a result of one of the three mechanisms:–

(1) Trauma to the area, which provides access of organism to the subcutaneous tissues.

(2) Extension from a urinary tract infection probably most commonly periurethral gland infection with dissection along fascial planes to involve the penis and scrotum.

(3) Extension of an infection from the perianal area or less commonly the retroperitoneal space along fascial planes to involve the penis and scrotum.

In all cases, rapid proliferation of bacteria occurs at the superficial fascial layer, spreading along fascial planes and producing an obliterative endarteritis of the overlying skin and subcutaneous tissue.[6,9]

Bacterial synergism leads to extension of the infection and further proliferation within the subcutaneous tissue, thus causing extensive subcutaneous necrosis.[1,9] Spreading takes place along fascial planes and does not involve the underlying muscle. However the 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.[1] If the process remains untreated the small subcutaneous end arteries undergo thrombosis which is a histologic hallmark of necrotizing fasciitis.[9] This results into ischemia which contributes to the 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 bacterial 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 visible gas in the tissues.[9] This was elicited in 3 patients (Table II).

With local trauma including surgery, organisms present on the skin can enter the subcutaneous tissues and cause dermal and epidermal coagulative necrosis, subcutaneous necrosis and infection.

Whereas in patients with urinary tract infection it may occur in several ways. In those 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 probably for many apparently idiopathic cases.[10,11]

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.[6,12] Whatever the pathway, organisms reaching the subcutaneous tissues 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

Although the first symptoms are usually pain or itching in the genital region,[1] it may be

surprising to see a patient presenting with total genital gangrene within hours or few days of onset.[2] In his series Akinola[2] noted in over 50 per cent of patients who had presented with warning signs of its onset which included itching and peppery–sensation of the genitalia. This is refuted by Martti[1] who feels the initial symptoms are slight and do not warn the patient of the rapid and often dramatic development of the condition. Fahal et al[4] in analyzing 9 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 1 day to a week, with a mean interval of 2 to 3 days.[3,6] However several early authors described a dramatic explosive nature of the disease taking the patient from robust health to severe prostration within a few hours. Recently cases are noted to have an insidious course, with symptoms often presenting for several days before hospitalization.[3,5,6] In this study the symptoms took 3 to 14 days, with most patients averaging between 5 to 7 days. (Table III).

Early in its course, Fournier's gangrene may resemble 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.[6] The condition is usually associated with fever and chills. Rarely a patient may show evidence of severe toxaemia.[2–4,6] The scrotum often enlarges to several times its normal diameter, the skin becomes hyperemic and later ominous black spot ensues.[1,9]

There is local crepitation in more than half of the patient due to subcutaneous emphysema

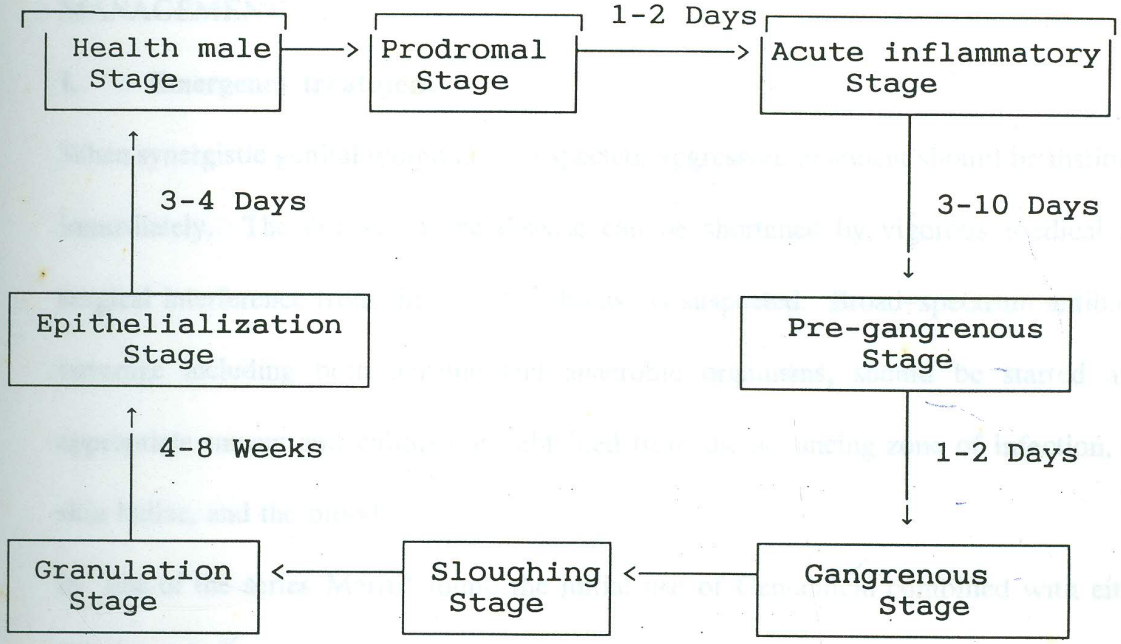
of scrotum.[1,2,6,9] In this series only 3 out of the eight patients, crepitus was demonstrated clinically (Table II).

In severe cases, systemic effects of infection may manifest as severe prostration, nausea and vomiting.[1,5,10] In this study the patient presented had signs of systemic infection. Occasionally delirium and adynamic ileus due to reflex sympathetic activity via the splanchnic nerves, and elevated levels of circulating catecholamine which inhibit peristalsis, may also be noted. In untreated cases, the scrotal and penile skin develop gangrene. At this stage pain almost subsides, probably owing to destruction done to sensory nerves. The involved area sloughs off. With this sloughing, the systemic symptoms and signs commonly abate as a result of naturally occurring drainage.[10,12] The area between the viable and dead skin becomes clearly demarcated and the slough separates in about 3 days time.[12,13] The testes, bared of their tunica vaginalis hang suspended by their cords, shamefully exposed through remarkably, free of gangrene, inured and oblivious to their new surroundings.[3]

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 initial contact included scrotal pain, fever and edema. Others had cellulitis, crepitus and necrosis. Thereafter gangrene and sloughing set in (Table II).

The following is a diagrammatic summary of the natural history of Fournier's gangrene as described by Bejanga.[13]



DIAGNOSIS

The diagnosis is based on the clinical picture of a fulminant progression of the disease, and on presence of multiple pathogenic organisms in the primary gram stain or culture. A high index of suspicion for Fournier's gangrene as one of the possible causes of acute scrotal pain can be valuable in early diagnosis of this condition and subsequently successful treatment of the patient.

Plain radiographs can aid in demonstrating subcutaneous emphysema before sloughing has set in.[1,3,4,9] Feculent odour of the wound can be an inference to presence of anaerobic organisms, this is important where laboratory facilities are inadequate to isolate the organisms involved.[4]

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MANAGEMENT

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 interference 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 advancing zone of infection, the skin bullae, and the blood.

In one of the series Martti[1] found the initial use of Gentamicin combined with either Clindamycin or sometimes with Chloramphenicol provided standard antibiotic treatment. Ampicillin is suggested as an addition to this basic regimen to cover suspected enterococci if the source of infection is genitourinary. Bahlmann et al[3] in another study had most cultured organisms sensitive to Chloramphenicol and Metronidazole. No other antibiotics has had a better spectrum according to the sensitivity tests in that series. They never used the antibiotics for more than seven days, however they found it logical in the initial regime to add penicillin G. The use of co-trimoxazole alone was found to be the drug of choice in a small scale study where the commonest isolated organism was Proteus.[4] It is apparent that until antibiotics were used in the management of Fournier's gangrene, the mortality ranged between 20 and 30 percent[1,9,13.] In the acute inflammatory stage most authors recommend early coverage with broad spectrum antibiotics, this will cover both aerobic and anaerobic bacteria pending modification of the agents after gram

stain and culture results are obtained. In this study a combination of Chloramphenicol and Metronidazole either orally or parenterally for duration between 7 to 10 days had good response. Patients with prominent systemic manifestation, were initially started on parenteral antibiotics with intravenous fluids until the acute stage of the disease subsided. The expeditious debridement of nonviable tissues following adequate resuscitation remains the mainstay of treatment.[1-6,9-13] However there are two school of thoughts regarding extent and or frequency of surgical debridement. These are:-

(1) Limited debridement - The 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 the repetitive excision of necrotic tissue during the ensuing one to two weeks as the margins between viable and non-viable tissue become demarcated. Akinola[2] quotes Warner and Falk in 1964, Kearney and Carling in 1983 and Badejo in 1985 concluding that limited debridement was very effective and had no mortality.

(2) Radical debridement - Initial debridement is usually extensively done and should extend generously to healthy tissue. Proponents of this approach believes that the only way to control the fulminant and rapidly progressing infection and gangrene is by aggressive debridement. However Akinola[2] quotes Lamb and Juler in 1983 experiencing a high mortality of 33 percent in their series.

are among the causes of negative culture, the search for fungi should also be undertaken in negative cultures since it has been observed also to cause osteomyelitis.[4,8]

In this series the commonest isolated organism was staphylococcus aureus which is in accord to findings of other authors.[1,7,3,4,6,7] The other isolated organisms included Klebsiella, proteus and E. Coli (Table III).

PATHOGENESIS

Infection of the bone depends on the interaction between the host and the offending microorganism. The host considerations include the anatomical properties of the bone, humoral and cellular immune mechanisms and the reparative power of the damaged area. Bacterial properties involved include the portal of entry, virulence, number, localization and colonization of the area.

The routes by which bacteria can reach the bone include extension from an adjoining soft tissue infection, from a source external to the bone and by the hematogenous route from a distant focus of infection. In this study, 9 patients had the disease set in spontaneously, probably following hematogenous route and in the other 6 patients the osteomyelitic process was post-traumatic in origin. Although the initial changes at the site of localization of infection have not been extensively studied in man, the inflammatory reaction in bone does not differ essentially from that in other tissues.[8] In hematogenous osteomyelitis the organisms enter the bone through the nutrient or metaphyseal vessels and localize in the venous sinusoid. Among the factors favouring bacterial proliferation includes a sharp bend epiphyseal plate which widens into sinusoid. This slows blood

flow producing a likely site for bacteria to settle.[3,4] On the other hand the endothelial cells in the affected metaphyseal vessels have no or insufficient phagocytic action, or urgumenting the process.

As exudative inflammation with increased vascularity, oedema and proliferation of polymorphs set in, the intramedullary pressure increases. This results into thrombosis and obliteration of the vessels, thus ischemia and bone necrosis occur. The increase in pressure in the intramedullary cavity forces pus through the Haversian and Volkmann canals. This results into stripping of the periosteum from the bone. The cortical bone is therefore isolated from its blood supply and produces more dead bone.[3,4,8,12] Pus may also perforate the periosteum escaping into the soft tissues or the neighbouring joint. Similarly, pus may re-enter the medullary canal through the cortex at a distance.

In a society like ours where drugs are scarce and limited choice, combined with the low literacy rate of our people, at this stage the chance of acute osteomyelitis proceeding to chronicity is even higher.

The focus of inflammation is encapsulated by a wall of granulation tissue and later by dense fibrous tissue which is avascular forms an impenetrable wall around the infected area. The primitive mesenchymal cells are stimulated to form new bone called the involucrum, which further isolates the dead and infected bone. The dead bone, seeded with bacteria can then act as a permanent reservoir for bacterial growth and invasion. This causes inflammation to flare up from time to time. Although the sequestrum reabsorbed by phagocytic cells slowly, more bone is destroyed and a vicious cycle may set in.[3,4,12]

No antibiotic can then penetrate the depth of this sequestrum, and hence infection can not be eradicated by this mode of therapy at this stage.

In sickle cell disease the devitalization of the tissue that arises as a result of intravascular sickling facilitates the hematogenous spread of the organisms, and usually the infecting agent is Salmonella[12] Hypoxia of diaphyseal marrow sinusoid facilitates sickling and thrombosis, thus explaining both the localization and multiplicity of the lesions. The increasing inflammatory oedema in a non–yielding living bone is associated with severe pain that leads to fixed flexed deformity of adjacent joints. Also loss of bone mineral occurs as a result of prolonged immobilization and inflammation, this further render the bone vulnerable to destruction and stress fractures.[3,12] In osteomyelitis occurring as a result of traumatic bone injury, the most important factors, in pathophysiology depends on the following[10]:–

(1) The extent of soft tissue damage and impairment of blood supply;

(2) The inoculation of bacterial flora;

(3) The instability of the fracture area; and

(4) The general defensive condition of the organism.

Tissue damage is caused by the injury itself and by the crushing forces that impact on the body. In addition, it is intensified by the operation and by hematoma and oedema. These factors together lead to increasing pressure on the tissue, which disturbs the microcirculation sufficiently to cause of compartment syndrome in some cases. Tissue necrosis ensues. Necrotic tissue itself acts as an ideal culture medium for bacteria. In

presence of synthetic material such as that implanted for osteosynthesis or internal fixation, additional non–living surfaces are available. In particular, large areas of destroyed bone constitute a risk. In such a field of debris after comminuted fracture it must be expected that a major part of bone pieces are devitalized and necrotic. Bacteria can settle there out of reach of the defense systems of the organism.[15] Such comminuted fracture areas are also unstable. The blood supply to the periosteum is disturbed further by traumatic reduction technique and by the operation itself. It remains of utmost importance to rember that rough manipulations cause additional lesions of the surrounding tissue, and that the risk of infection to a disturbed and devitalized bone area is very high.[16]

The other factors which are important in pathogenesis of osteomyelitis as mentioned above influence each other and are interdependent. The influence is progresive, intensifying to a vicious circle of post–traumatic osteomyelits.[10,15,16]

CLINICAL PICTURE AND DIAGNOSIS

Chronic osteomyelitis is characterized by its protracted course and by the rarity of spontaneous recovery. It is rare for spontaneous expulsion or reabsorption of a sequestra to lead to spontaneous cure. The clinical features depending on how advanced is the osteomyelitic process. They include swelling, local tenderness, erythema and increased warmth at the affected area. Chronic osteomyelitis should be suspected in patients whom acute osteomyelitis was previously diagnosed and received inadequate therapy. Recurrence is one of the features of chronic osteomyelitis. Recurrences after periods of

up to 80 years have been reported.[3,5] The average duration is several decades and often several surgical interventions and long courses of antibiotic treatment are necessary. The affected part often has deeply retracted scars and one or several discharging sinuses. The soft parts around the osteomyelitic focus are usually scarified and the skin dystrophic. The presence of a sinus may cause severe psychic stress. Most patients hardly feel any pain in chronic osteomyelitis, however pain may be a symptom due to early closure of a sinus resulting into increased pressure of pus in the sinus, or due to a flare up. When pain develops in a discharging sinus, the possibility of malignant degeneration of the epithelium of the fistulous duct should be considered.[4] Fever is not a feature of chronic osteomyelitis. It is usually found during an exacerbation of the disease.

The diagnosis can be reached purely on clinical grounds, however the following investigations are important:–

(1) **Hemogram**

Sickling test and Hb–electrophoresis are very important. A low haemoglobin may be observed in sicklers and in chronic osteomyelitis. Usually there is leucocytosis with a shift to the left although in vertebral and chronic osteomyelitis, these parameters may be normal.

ESR is usually high but is not specific for bone infection.

(2) **Microbiology**

Blood culture is known to be positive in acute hematogenous osteomyelitis in half the cases i.e 50%.[8] The same organisms are usually grown in material aspirated

from subperiosteal space. In chronic osteomyelitis, a pus swab can be taken from a discharging sinus.

(3) **Radiology**

The roentgenographic features of chronic osteomyelitis include a soft tissue swelling, sequestrum– involucrum process, periosteal reaction, irregular bone cortex, osteoporosis and Brodies abscess. The radiographic abnormalities usually appear 3 to 4 weeks after the onset of the symptoms. Only in less than one–third of the patients radiographic changes can be seen seven to ten days after the onset of the symptoms.[18]

Changes in bone blood flow and bone reaction occur very early. These can be detected by Technetium 99 radionuclide scanning.[18] Scintigraphy scan is believed to reveal abnormalities sooner than x–ray and recently it is being employed in the diagnosis of osteomyelitis.[19]

Use of sinogram in the presence of a sinus should be considered if possible. It shows the site and direction of the lesion and aids in planning the surgical approach. However it can be a way to disseminate infection due to increased pressure during the procedure.

TREATMENT

Chronic osteomyelitis is a disease that can cause frustration to both the patient and the surgeon alike because of its protracted course and the poor results or recurrence after treatment. Prevention is better than cure hold true in case of this disease, among others

Acute osteomyelitis should be treated vigorously once it is diagnosed, similarly the basic principles in the 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.[2,4] The organisms thrive in avascular bone and scar tissue and frequently the scar tissue acts as an impenetrable membrane to antibiotic therapy.[5] 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.[4,5] The hematocrit value should aim at above 40%. Hypoproteinemia and vitamin deficiency particularly vitamin C should be corrected. Splintage or immobilization in plaster of paris and rest are adjunct to treatment of chronic osteomyelitis. Immobilization minimizes the chances of disseminating the infection, reducing deformity and pathological fracture.

2. **Debridement**

   The old principle of Surgery that states where there is pus it should be evacuated holds. However opening an abscess though appropriate, opening of bone in the hope of preventing bone destruction, is controversial. The mainstay in surgery of chronic osteomyelitis is radicality. Surgery should be as radical as possible without regard to remaining defect.[2,4,5] 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 vital bone is essential so that the radicality is combined with maximum conservation of the skeletal continuity. The identification of non viable tissue is done by several methods using:–

(1) Disulphide blue given intravenously, this stains vital structures green and the sequestra remain white, however a bloodless field is required for proper staining. When injected through a sinus it stains avascular tissue.

(2) Tetracycline given orally prior to surgery is incorporated in vital bone and can be visualized by ultraviolet light.

(3) In the experienced, viable bone can be visualized by the actual bleeding from the bone.

Following the above, surrounding scars are resected generously. Saucerization is done and where bone sequesters are present these are removed regardless of the amount of instability that arises.[5,10] At the end of the debridement, all tissues must show bleeding points. The operation is done without tourniquet. Finally the cavity is cleaned by irrigation using copious amounts of normal saline and hydrogen peroxide. Recently the bone resection and transport using the Ilizarov's technique is gaining increasing popularity. This method has the advantage that infected bone is excised en bloc, and stability of the remaining fragments is achieved using the Ilizarov's external fixator apparatus, thus the patient ambulates considerably early.[20] New bone gradually forms filling the defect, eventually through bone

distraction and transport with time the bone loss is finally recovered. However this method is unsuitable in pan osteomyelitis. It is worthy remembering that the most frequent cause of unsuccessful treatment is failure to remove all the involved tissue especially at initial debridement, this is due to poor surgical technique.

Chronic infection of the bone in the face of open fracture may present a difficult problem. If a fracture is in a satisfactory position, excellent results may be obtained by providing drainage of any pus that may accumulate, rigid uninterrupted and prolonged immobilization plus appropriate antibiotics.[10] In such situations the fracture will heal usually by periosteal reaction on one side while draining on the opposite side. When internal fixation is used, the material is not removed as long as it is providing rigid internal fixation. However other authors advocate removal of the internal fixation material because it is ordinarily surrounded by chronically infected granulation tissue.[5] The resulting instability is then managed by cast immobilization or external fixator.

Post-traumatic osteomyelitis can also be best managed using excision and transport of bone by the Ilizarov's technique as described previously.[20]

3. **Chemotherapy**

In the treatment of chronic osteomyelitis antibiotics can in no way replace surgery regardless of the method of administration.[10,21] 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.[6,21,22] Local antibiotic use when added to systemic antibiotic therapy is claimed to accelerate the formation of new bone and localization of sequestra.[25] Antibiotic irrigation of wound can be done in an open wound or in the form of closed irrigation – suction technique. Irrigation has the advantage of mechanically washing away microorganisms and debris and reaching high concentrations. However leakage of irrigation solution onto the bedding further increase the danger of infection.[3,4] Insufflation of antibiotic powder and antibiotic dressing have similar advantage of reaching high local concentration, and may prevent superinfection. However the danger of development of resistant strains remains a real threat.[22]

Gentamycin in the form of beads consisting of polymethyl methacrylate impregnated with gentamycin sulphate are being used more often in the treatment of chronic osteomyelitis. In our centre Bactgrass is often used since the later is not readily available. The antibiotic is slowly but steadily released and diffuses into the tissues where the beads are implanted. The chains are removed after two weeks before they are firmly fixed in the bone, thus allowing also for inspection of the wound and bone.

In this study the choice of systemic antibiotics had limitation due to lack of required antibiotics. Most frequently we had to use Cloxacillin, Ampicillin, Chloramphenicol, Flagyl or Gentamycin. In such a state the outcome is expectedly

poor and, recurrence is frequent. The other problem experienced was the choice of route, frequently we had been forced by the prevailing circumstances to use oral antibiotics since parenteral ones could not be available. In 5 of our patients who had no culture and sensitivity results antibiotics had to be given empirically based on experience that the commonest isolate is staphylococcus aureus, however this violates regulation for antibiotic prescription. This came as a result of shortage of swab sticks or transport media for taking the culture specimen.

4. **Elimination of the dead space**

After debridement, small wounds can be left to granulate and heal. Secondary wound closure and split thickness skin grafting are other means to fasten recovery.[23] Wide local excision however may result into failure to obliterate the dead space due to the scarred and fibrotic tissue structures. This may however be overcome by use of local muscle flaps or transfer of free vascular muscle flaps.[4,5,23,26] The procedures require great skill and dilligency but results are encouraging.[26] The indications are:-

(1) When adjacent soft parts cannot obliterate the dead space remaining after debridement.

(2) The dead space is located in the area that permits transposition of a local muscle flap without compromising its neurovascular pedicle.

(3) The debridement procedure has not compromised the mechanical integrity of the osseous structures and

(4) Gross purulence has been eliminated by debridement and antimicrobial therapy.

Bone defects created by debridement are bridged by cancellous or cortical bone graft. Similarly grafts consisting of muscle with the overlying skin and the bone sharing the same blood supply have been used with success to bridge the bone defect and at the same time eliminate the dead space.[26] This method however was not used in any of our patients.

Esterhai[23] quote Papineau who described the treatment of chronic osteomyelitis by bone grafting using the open cancellous technique. This method is reported to be successful and is carried out in the three stages: debridement and excision of devitalized tissues (Papineau I); bone grafting (Papineau II); skin coverage (Papineau III). However bone grafting is contradicted in aggressive purulent osteomyelitis. Infection should be controlled first otherwise the survival of the graft will be limited.[23]

PREVENTION

Chronic osteomyelitis is often a complication of acute osteomyelitis. An important aspect in the prevention of chronic osteomyelitis is the correct diagnosis and treatment of acute osteomyelitis. The place of surgical exploration and drilling in acute osteomyelitis is still controversial. While some authors advocate surgical decompression as part of the treatment of acute osteomyelitis if pus is obtained on aspiration, others recommend surgical exploration as being mandatory in all cases of acute osteomyelitis regardless of

whether there are clinical signs of abscess or not.[4,5,8] Health education to the mass regarding early reporting to hospital for treatment is also an integral part in prevention of acute osteomyelitis proceeding to a chronic form.

For open fractures, the basic principles in the management should be strictly adhered to. These consist of:–

(a) Simple coverage with sterile dressing soaked in hydrogen peroxide.

(b) An open fracture should never be reduced by dragging the exposed bone into the depths of the wound. All exposed tissues should be debrided carefully and cleaned before reduction.

(c) During the preparation of the operative field, the wound should be kept covered. Nothing can be gained by probing the depths of the wound until the surrounding skin is prepared thoroughly i.e. Social toilet, followed by debridement.

(d) Harsh disinfectants like iodine and alcohol should never be allowed to come in contact with soft tissue exposed, because this will produce tissue damage. Debridement must be accomplished thoroughly. Tight closure of the wound may cause tissue ischaemia and encourage formation of hematoma which is an ideal culture medium for bacteria.

(e) Use of suction irrigation is frequently indicated. Immobilization is essential to minimize oedema, tissue damage, hemorrhage and sepsis.

(f) At the time of operation the surgeon should observe maximum sterility and strictly adhere to the aseptic technique especially during bone surgery.

CONCLUSION/RECOMMENDATIONS

Chronic osteomyelitis is increasingly becoming a common problem in our society and continue to be a challenge to the surgeon in our environment. It remains true that considerable number of cases of acute osteomyelitis progress to the chronic form as has been the case in 9 of the studied patients. This is probably due to a low index of suspicion to the condition at our peripheral hospitals and also due to shortage or lack of appropriate antibiotics. Similarly most patients are ignorant of the condition and most of them present very late to a medical care. The long periods of hospitalization and the unavoidable prolonged use of costly antibiotics have a significant socio–economic impact. Radical surgery at the first attempt is the cornerstone to success in treating chronic osteomyelitis. The good surgical work should be supplemented by relevant antibiotics depending on culture and sensitivity results.

It is high time we look into a number of issues which might help in the control of this dreadful condition. The following are suggested:–

(1) Provision of health education at community level so as to help increase awareness of both acute hematogenous osteomyelitis and chronic osteomyelitis hence promoting early recognition and reporting to a health facility.

(2) Provision of update knowledge of the disease, its prevention and treatment to Dispensary, Health Centre and District Hospital personnel this will influence and accelerate decision making i.e early referral.

(3) Increase the emphasis to this condition during teaching of medical students.

(4) Embark to a National Policy in regard to the epidemiology, bacteriology, sensitivity and hence standardization of treatment.

(5) Start serial radio programmes to educate the mass of the condition.

However chronic osteomyelitis remains to be a very frustrating condition as far as cure is concerned, since failure and recurrence are frequently met.

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3.2: THE MANAGEMENT OF HIP FRACTURES AS SEEN AT MUHIMBILI MEDICAL CENTRE

SUMMARY

Between July and November, 1993, a total of 19 patients with fractures of the hip were managed while the author was rotating in the department of Orthopaedic and Trauma of Muhimbili Medical Centre. Ten of the patients were elderly ranging between 54 to 75 years (mean 66.4 years). These sustained femoral neck fractures from trivial injuries. In this group there were 6 females and 4 male.The other 9 patients were in the age range between 14 and 50 years of age (mean 36 years). These had a variety of hip fractures; cervical, intertrochanteric and subtrochanteric. The causes in this group were mostly motor traffic accidents, falls from height and gunshot injuries. Pain, shortening and loss of function were the main mode of presentation and only one patient had open fracture from a gunshot.

Management comprised of non operative approach in 16 patients, this included all patients in the age range between 60 to 75 years. 3 patients had been treated by open reduction and internal fixation. There was only one death but the morbidity had been considerable. The cause of death was a sudden cardiac arrest, in a 71 years old man. The cause of this cardiac arrest was not established.

INTRODUCTION

Fractures of the hip usually refer to those involving the proximal femur, including the

head, neck and trochanteric regions.[1] Fractures of the acetabulum are usually considered pelvic fractures. Proximal femoral fractures are a common and increasing cause of hospital admission in the elderly (i.e persons over 70 years of age). The increase cannot simply be accounted for by the rising number of elderly people in the population, as the number of younger patients admitted to hospital with these fractures is also increasing.[2] In any fracture, healing depends not only on fixation or immobilization, but, also on blood supply. The blood supply in this case is marginal and more subjected to damage to the neck or the portion of the femur that is intracapsular, which makes the prognosis of these fractures much worse than for those occurring below the intertrochanteric line outside the capsule.[2,3] Similarly in intracapsular fractures, bleeding occurs within the hip joint, and the resultant intracapsular pressure rapidly exceeds the pressure in capsular vessels, causing thrombosis.

The management of these fractures is therefore difficult especially in our setting where resources and facilities are limited. Nevertheless fractures of the proximal femur commonly results in the need for long-term institutional care, and the social and economical implications are considerable.[3,4] This prompted the author to pip into the look of management of these cases in our setting.

CASE REPORT

S.E. IP NO 759762 was a 70 year old lady who was admitted to one of the wards of the Orthopaedic Department of Muhimbili Medical Centre, following a trivial slip injury to the left hip while trying to stand up. She then started experiencing sharp pain to the

affected hip and was unable to stand or walk without support. Denied history of loss of consciousness nor does she drink alcohol. She had no history of drug abuse, or that of chronic illnesses such as TB or Diabetics mellitus, however she is a known case of Hypertension who has been on treatment for several years. Had no history suggestive of epilepsy.

Clinical examination revealed an elderly lady, fully conscious and well oriented to time, place and person. She was not pale looking and was afebrile. Her pulse rate was 80 beats per minute with good volume, the blood pressure was 180/120 mmHg. Examination of the lower limbs revealed the left lower limb to be in an attitude of slight abduction and external rotation. The left hip was swollen and was tender especially on deep palpation. The femoral head could not be appreciated on palpation. Active movements at the left hip joint were impaired, she was not examined for passive movements. There was no shortening of the affected limb. Examination of the right lower limb and other systems were normal.

The diagnosis of hip fracture left limb was made, with the femoral neck fracture considered as the most likely type of the fracture. A plain antero-posterior radiograph of the left hip revealed undisplaced Basicervical fracture of the left femur, with a slight varus position. The patients's limb was immobilized using skin traction and was positioned in abduction, 3 kg was suspended to effect the traction. The limb was also supported on a Braun's splint. She was also continued on oral Aldomet 500 mg T.D.S to control hypertension.

Control x-rays of the left hip were done serially after every 2 weeks. At 4th week of traction there was minimal callus formation. At 6th week, the callus had stabilized and the patient was started on ambulatory physiotherapy using crutches. Was discharged home in the 7th week with crutches bearing no weight until the 10th week. She was then seen twice at follow-up clinics and was doing fine.

TABULAR SUMMARY

TABLE I: DISTRIBUTION OF PATIENTS BY AGE AND SEX

AGE IN YEARS	NUMBER OF PATIENTS		TOTAL
	MALES	FEMALES	
14 - 20	4	0	4
21 - 30	1	1	2
31 - 40	0	0	0
41 - 50	3	0	3
51 - 60	2	4	6
61 - 70	0	2	2
71+	2	0	2
TOTAL	12	7	19

TABLE II: CAUSES OF THE FRACTURE

CAUSE	NUMBER OF PATIENTS
Trivial, Trauma (Minor Trauma)	10
Falls (height/slip/sports)	5
Road traffic accident	3
Gunshot	1
TOTAL	19

TABLE III: DISTRIBUTION OF PATIENTS IN RELATION TO THE ANATOMICAL LOCATION OF THE FRACTURE

LOCATION	NUMBER OF PATIENTS
Head of femur	0
Femoral neck	10
Intertrochanteric	5
Subtrochanteric	4
TOTAL	19

TABLE IV: DURATION OF PERIOD FROM TRAUMA TO THE TIME REPORTED TO OUR CENTRE

DURATION IN DAYS	NUMBER OF PATIENTS
Up to 1	11
2 – 4	4
5 – 7	2
8+	2
TOTAL	19

TABLE V: TREATMENT OFFERED

MODALITY	NUMBER OF PATIENTS
Closed reduction + skin traction in abduction	15
Dynamic hip screw	1
Angled nail plate	1
Girdlestone operation	1
Simple parallel supporting screws	1
TOTAL	19

TABLE VI: COMPLICATIONS

COMPLICATIONS	FREQUENCY
Avascular necrosis	9
Shortening of the affected limb	9
Decubitus ulcers	4
Pneumonia	3
Malunion	3
TOTAL	28

- Nine patients had more than one complication.

DISCUSSION

INCIDENCE AND AETIOLOGY

Fractures of the hip usually refer to those involving the proximal femur, including the head, neck and trochanteric regions.[1,3,] Both hip fracture incidence and life expectancy are known to have increased during the last decades, they are therefore primarily fractures of the aged.[2,3,4]. In his series Hofeldt[4] found that the absolute number of hip fractures is large, increasing and related to osteoporosis as well as other factors. He observed further that by 65 years of age 50% of women will exhibit bone mineral below the fracture threshold; by age 85, 100%. Many other authors have observed that the frequency of osteoporosis and hip fractures increases with age.[5,6,10,11] However Wicks et al[12] in studying patients with hip fractures in Australia found neither osteoporosis nor osteomalacia to be

a major factor, although vertebral fractures were invariably associated with osteoporosis. His findings are in accord to another study of fifty one patients on the effect of age on bone composition and viability in the femoral head.[7]

However osteocyte death which is believed to be associated with the normal age–related loss of bone, which could contribute to hip fracture was not assessed in his study.

"Trivial" trauma from falls is frequently the cause of Hip fractures in the elderly who often loose the renal ability to 1 alpha hydroxylate 25–OH, consequently loose the ability to absorb calcium. This renders the bone osteoporotic.[4,11,13] In his series Finsen[9], attributed the increase in incidence of hip fracture to be probably accounted for partly by the increased life span of the infirm. However, he further suggested that increases in life expectancy among a majority of the elderly also entail better general health at any particular age, and that this may lead to a higher level of physical mobility which raises the risk of falls and fractures. The later is denied by Cooper[6] who suggests that physical inactivity and concomitant muscle weakness are probably important determinants of the risk of hip fracture.

Stress fractures of the proximal femur should be thought in all military personnel with bone pain after exertion.[14] Whether this reflects the amount of military endurance it remains un answered question of interest.

Few literature discuss about the incidence of pathological fractures of the proximal femur. They can occur secondary to bone tumors, destructive processes following inflammatory bone and joint diseases such as third stage tuberculosis of the hip and severe chronic

osteomyelitis of the proximal femur.

In this study the causes predisposing to the proximal femoral fractures are shown in table II. Ten elderly patients had sustained the fracture following a trivial trauma, most of them had radiological evidence of osteoporosis.

CLASSIFICATION

Hip fractures can be broadly divided into 2 general categories[1,2,13,] intracapsular and extracapsular (or trochanteric). This division into anatomic areas is more than just of academic interest, as fractures occurring in each of these locations have their own specific problems in regard to treatment, healing and complications.[1] Nevertheless the AO has its own classification of hip fractures which seems to be gaining increasing popularity.[21]

1. **Intracapsular Fractures**

   These are fraught with more complications than any of the other types of hip fractures, and thus require special attention. Although controversy exists as to further subclassification of intracapsular fractures these fractures can, for those who care, be divided into thirds, namely the subcapital area, the transcervical area, and the base of neck or basicervical area.[1] Subcapital fractures occur just distal to the articular margin of the head of the femur, whereas those passing through the neck are referred to as transcervical fractures. Fractures occurring at the junction of the neck and the shaft are referred to as base of neck (basicervical) fractures. Intracapsular fractures can also be classified according to the degree of displacement of the fragments.

Stanley et al[2] quotes Garden[15](1964) grading the development of a complete fracture into four grades or Garden's:–

Grade I: Incomplete fracture; the fracture is impacted or abducted with preservation of the medical group of trabeculae.

Grade II: Complete fracture without displacement; the trabecular pattern remain's undisturbed.

Grade III: Complete fracture with partial displacement; the fracture fragments remain connected by the posterior retinaculum attachment, but the trabeculae are malaligned.

Grade IV: Complete fracture with total displacement; the proximal component lies normally in the acetabulum, while the distal component is displaced anteriorly.

At one time emphasis was placed on the direction of hip fractures and its relation to fracture healing. Shultz[1] quotes Pauwell who established a classification in which intracapsular fractures were classified by their direction, noting the angle of the fracture line with the horizontal, as measured in the anteroposterior radiograph and basing this angular measurement on the post reduction x–rays. According to this classification it is graded into the following:–

Class 1 fracture: forms an angle of 30° or less

Class 2 fracture: an angle of 30°–70° and,

Class 3 fracture: an angle of 70°– 90°.

The premise was that those fractures with a more horizontal fracture lie would be subject to impacting forces with weight bearing and thus offer a better prognosis for healing than did more oblique fractures, which would be subjected to shearing forces. The Pauwell's classification is no longer commonly used but should be known by those interested in these fractures.

2. **Extracapsular fractures**

These include intertrochanteric fractures and subtrochanteric fractures which by definition occur between the lesser trochanter and a point 5 cm distally.[2] Some are inherently stable and others tend to be quite unstable.

Depending on the degree of medial and posterolateral comminution of the fracture which determines stability. Stanley et al[2] quotes the classification done by Jansen which has the following:-

I. Stable intertrochanteric fractures:-

   a) Undisplaced 2-fragment fracture

   b) Displaced 2-fragment fracture

II. Unstable intertrochanteric fractures:

   a) 3-fragment fracture without posterolateral support.

   b) 3-fragment fracture without medial support.

   c) 4 - fragment fracture.

3. **AO Classification**

The AO/ASIF foundation has recently classified Hip fractures considering the

arrangement in an ascending order of severity according to morphologic complexities of the fractures. Also the classification considers the difficulties in their treatment and the prognosis.[21] This classification divides Hip fractures into three categories namely, Trochanteric area fractures; Neck fractures and Head fracture with neck fracture.

In this study table III shows the location distribution of hip fractures. Out of the 10 femoral neck fractures the subtyping distribution was 2,4,4, for subcapital, transcervical basicervical respectively. And according to Garden's classification; 3 patients had grade I, 2 patients grade II and 5 patients had grade III. None had grade IV fracture.

CLINICAL FEATURES AND DIAGNOSIS

The following are important features in a patient with a fractured hip:–

1. Pain around the hip, however occasionally the only complaint may be soreness or stiffness following a fall. Pain is pronounced on movement but in most impacted fractures, the patient can move the leg about freely, and he may have walked on it with minimal pain.
2. Anatomic deformity – the characteristic deformity of the leg in a patient with deformity of the leg in a patient with a fractured hip is one of adduction, external rotation and shortening. Various degrees of anatomical deformity are found, however as a rule, the patient will be unable to lift the leg. The amount of external rotation in femoral neck fractures is usually of the magnitude of 30°–40°, since the intact capsule prevents the leg from achieving the extreme external

rotation that is associated with trochanteric fractures that occur beyond its limits.

3. Variation from the classic deformity of a fractured hip may occur. In a patient who has fallen directly on the greater trochanter, the findings may reveal a slight increase in the length of the leg, a slight knock-knee deformity and a local hematoma over the greater trochanter. The trochanter may be lower and less prominent and external rotation deformity may be absent.

4. An unusual deformity associated with femoral neck fractures may be internal rotation of the extremity. This position may be due to displacement of the femoral neck behind the head of the femur, or it may indicate a base of the neck fracture in which the centre of rotation is at the anterior capsule.

5. In case of fracture of the neck of the femur with impaction, the loss of internal rotation may indicate impaction in external rotation, whereas loss of external rotation may indicate impaction in internal rotation.

6. In trochanteric fractures, findings on initial physical examination will closely parallel those associated with intracapsular fracture, as pointed above, except that there may be more shortening with a bruise over the greater trochanter. Since the joint capsule is proximal to the line of the fracture and does not tether the distal fragments, the leg will often lie in as much as 80° or 90° of external rotation.

DIAGNOSIS

The following aids in reaching the diagnosis:–

1. **History and clinical features**

Although fractures of the hip are usually associated with a fall, patients with intracapsular fractures often have an indirect mechanism of injury. The history in such patients suggest that the hip broke first from a twist and then the patient fell. In patients with trochanteric fracture, the injury is generally the result of a direct blow to the trochanteric area. However fractures of the lesser trochanter almost never occur by direct trauma but are the result of avulsion forces.

Other precipitating causes of intracapsular fractures besides falls and muscular effort are tumors, radiation therapy, metabolic disease and other diseases such as osteomyelitis and bone Tb. These should be sought in the history. Trying for crepitus is not indicated.

2. **Conventional radiographs**

The surgeon should rely on proper and adequate X–rays studies to confirm the diagnosis anteroposterior view is most important but lateral view is also equally important in trochanteric fractures and in cases of comminuted fractures.

MANAGEMENT

The management of fractures of the proximal femur continues to be one of the most challenging areas in the field of trauma. Many patients with hip injuries do not survive the fracture and disability due to unsatisfactory healing is common.[2,13]

These are fractures primarily of the aged, in whom the incidence of serious associated disease is high and tolerance for prolonged immobilization and bed rest poor, similarly the aged do not have the same potential for recovery as younger patients to.[2]

I. **Intracapsular fractures**

First aid and emergency treatment:

As soon as a fracture is suspected, the affected limb should be splinted with a pillow bound between the limbs to protect the soft tissues and to minimize the possibility of further trauma to the vessels of the posterior retinaculum lying against the femoral neck. Splinting not only relieve the discomfort and much of the apprehension of the patient but it also reduces the possibility of further hemorrhage within the joint, with accompanying increase in the intra–articular pressure, a condition that may contribute to the development of aseptic necrosis.[2,3,13]

Conservative management:

This is useful method especially when the fracture fragments are in anatomical position or slightly valgus on the anteroposterior radiograph. Otherwise minimal reduction should be attempted, and then the limb immobilized in abduction position using a Russel's Skin traction method. Aufranc et al[13] suggests that for the first 10–14 days of traction the patient should be on strict bed rest, if the fracture is doing well i.e absence of pain then a total period of 3–6 weeks of bed rest is completed followed by crutch protection until union is completed usually 4–6 months period. The patient should have serial radiographs at least once weekly or after 2 weeks.[16] The use of local injected anaesthetic agent such

as Celestone has role in the conservative management and control of local pain. This contains betamethasone sodium phosphate and betamethasone acetate in a sterile buffered and preserved vehicle. It controls pain for a prolonged period of time and by the time its action is over, fibrotic tissue has formed around and will thus help to cushion the pressure effect, and the osteoarthritic process has had sufficient time to heal.

Operative management:

Many methods of internal fixation have been proposed for undisplaced intracapsular fractures (Garden I and II), this is because if left untreated, up to 20% will displace.[2] As successful outcome depends, however, not only on the device chosen, but on it being carefully and accurately inserted to produce rigid fixation.[2,16,17] Often the use of image intensifier helps in achieving the right goal.

The following are methods commonly advocated, including their merits and demerits:–

i) **Single screw fixation**

Is inadequate, regardless of the angle of screw insertion. A horizontal screw position predisposes to deformity from weight bearing forces, while a low angle position is subject to lateral rotation deformity.

ii) **Cross Garden Screw fixation**

The use of two screws gives fracture stability, provided that the screws are inserted so that they cross.[2] The horizontal screw is inserted first, and should pass through the anterior half of the greater trochanter towards the inferior part of the femoral head. The second, more obliquely placed screw is inserted posteriorly so



as to skirt the calcar with the tip of the screw lying at the midpoint of the articular margin on the femoral head. Poorly inserted screws fails to resist the deforming forces and may cut out through the bone. Parker et al[19] in studying results of treatment is 242 patients with intracapsular fractures found that those treated using parallel screws had superior outcome if the reduction was good.

iii) **Multiple screw fixation:**

Involves the use of two or three cancellous screws (e.g.AO cancellous screws). These should be places symmetrically and peripherally in the neck and head of the femur, and should be parallel to each other so that fracture impaction can occur.[2,13] To ensure maximal proximal fixation, the tips of the screws must lie in the subchondral bone of the femoral head with only the screw thread in the proximal fragment.

iv) **Sliding screw and plate fixation**:

Is used for basicervical fractures.[1,2] The implants most commonly used are the Richard's compression screw and the Dynamic Hip screw. Both screws provide firm fixation of the femoral head to the femoral shaft, and so neutralize the lateral rotation forces at the fracture site. In addition, the screw is able to "back out", ensuring continuous impaction of the fracture fragments under the action of surrounding muscles.

The ideal position of the screw in the femoral head remains controversial. Whereas central placement is favored, others recommend that the screw be

positioned inferiorly on the anteroposterior radiography and slightly posterior on the lateral radiography.[2] Whichever position is chosen, however, the screw should be advanced to within 5mm of the articular margin of the femoral head.[1,2,13,17]

v) **Prosthesis:**

In younger patients with subcapital fractures, every effort must be made to preserve the femoral head.[2] In the elderly, the complication rate from aseptic necrosis, non–union and fixation failure is such that prosthetic replacement of the femoral head is usually the treatment of choice. Prosthetic replacement avoids the specific complications and has the added advantage of allowing immediate mobilization with weight bearing.

The displaced fractures (Garden III and IV) can be managed using the following methods:–

a) **Hemiarthroplasty**

The Austin Moore and the Thompson prostheses are the most commonly used types of femoral head replacement.[1,2,17,18] Although the incidence of loosening is less with the cemented Thompson prosthesis than with the uncemented Austin Moore, the use of cement is not without problems and can make revision, if necessary, extremely difficult. Acetabular erosion can occur with both types of hemiarthroplasty, and this has led to an increasing use of bipolar prostheses.

b) **Bipolar femoral head prostheses**

Were designed to overcome the problem of acetabular erosion because, at least in theory, most movement should occur at the low friction inner bearing rather than at the high friction outer head of acetabular surface.[1,2] However it has recently been shown, that the behaviour of bipolar implants depends on the condition of the acetabular cartilage. If the cartilage is well preserved, as often occurs with femoral neck fractures, the outer head slides easily on the acetabular cartilage and the implant functions as if it were a hemiarthroplasty.[2] However, as the acetabular cartilage is eroded, the increase in frictional force between the outer head and the acetabulum may result in a change of the centre of movement to the inner bearing. The implant would then begin to function as a bipolar prosthesis with arrest of the acetabular damage. Stanley et al[2] notes that these types of implant have the theoretical advantage that if acetabular erosion occurs, the bipolar head can be removed and, without revising the femoral component, can be converted to a total hip replacement with a cemented acetabular cup.

c) **Total hip replacement**

This is valuable as a salvage technique in the treatment of complications of femoral neck fractures, and has limited specific indications in the management of acute fractures.Franzen et al[20] in studying the rate of

revision in 84 consecutive total hip replacements performed for failed osteosynthesis of femoral neck fractures had good results. He recommends internal fixation as the primary procedure, and total hip replacement should be reserved when osteosynthesis fails. The argument is also supported by Stanley et al[2] who views internal fixation as simpler and quicker than total hip replacement, thus place the elderly patient at less risk than when a total hip arthroplasty is performed.

(v) **Other salvage procedures**

This include the girdlestone operation which has value when the head of femur has undergone some degree of avascular necrosis thus its surface roughened and irritant, leading to a constant source of pain. This procedure will control pain by excising the useless remaining head and arthrodezing the proximal part of the femur to the acetabulum. This was done to one of the study patient who has had severe pain to the affected hip following a subcapital fracture of the left femur and had poor response to use of local injection of anaesthetic agent (Celestone). The other salvage procedure used to control pain is joint toilet which include excision of inflamed tissue between the head and acetabulum, and interposition of soft tissue pad such as the gracilis muscle. This helps to cushion the direct pressure thus controlling the pain.

II. **Extracapsular fractures**

Unlike intracapsular fractures; these fractures have a large surface area of cancellous bone and consequently, providing accurate reduction, and fixation to be achieved. Fracture union is seldom a problem.[2,13] However depending on the stability of the fracture the management can be as follows:–

i) **Stable fractures**

Some form of Russell traction is the best means of regaining length and correcting deformity.[13] Traction must be continued for 12–14 weeks in many cases, and even at the end of that time, varus deformity may recur. The limb is normally positioned in slight abduction. One problem is that the skin may not tolerate the prolonged use of the adhesive tape needed for effective traction. In such a case, a threaded kirschner wire through the tibial tubercle is advisable. Use of a threaded wire eliminates sliding of the wire in the osteoporotic bone so frequently found in aged patients.

ii) **Unstable Fractures**

Numerous devices for holding the fracture fragments are available. Treatment of these fractures is most effectively accomplished using either the Richard's compression Screw or the Dynamic Hip Screw. Neither of these devices, however, can withstand cyclical loading in the presence of an unstable fracture which has been inadequately reduced and fixed.[2] The strength of the fracture fragment assembly is dependent upon the Bone

quality, Fragment geometry, Reduction, and similarily Implant design and placement.

More recently the Gamma nail, a short intramedullary nail, has been developed and appears to provide improved rotational and axial stability. Similarly the use of interlocking intramedullary nails, with proximal and or distal locking screws is gaining increased popularity.[2]

In this study of 19 patients the treatment offered in summarized in table (V). Fifteen patients were managed conservatively using skin traction and abduction. Out of the 15, seven has femoral neck fractures and eight had trochanteric fractures. Only 4 patients underwent operative treatment, out of which, three had femoral neck fractures and one had pertrochanteric fracture which was comminuted.

Although most patients managed conservatively had indications for surgery, due to theater shortcomings and lack of the necessary equipments, we were forced to embark on non operative methods. The results were poor in such patients as they developed avascular necrosis non union and limb shortening (Table VI). Mobilization was assisted by using wooden crutches and in some instances relatives were instructed how to assist the disabled elderly patients.

COMPLICATIONS

These can be divided into general complications relating to the patient and those

associated with the fracture. However it is important to remember that the elderly patients who sustains a hip fracture are usually at high risk and tend to have more complications than the younger patients.

1. **General complications**

   Include thromboembolic disease, pressure sores and sepsis.

2. **Complications related to the fracture**

   a) Avascular necrosis of the femoral head develops when its blood supply is interrupted. It is common following intracapsular fractures with the incidence varying between 66–84% in displaced type of fractures.[2]

   b) **Non–Union**

   Occurs as a result of an unsatisfactory reduction, inadequate immobilization or as with avascular necrosis, due to interruption of the blood supply to the head of the femur secondary to trauma.[1,2,13] Although it is difficult to define non–union, most surgeons agree that there should be evidence of healing 6–12 months after injury.[2]

   c) **Other Complications**

   These include osteoarthritis, malunion or fracture myositis ossificans, and peripheral nerve injuries.

The complications observed in this study are shown in table (VI). Avascular necrosis was the predominating complication of hip fractures.

PROGNOSIS

Despite all the possible complications related to surgery, the conservatively treated patients has a significantly higher morbidity and mortality.[1,2,13] Stanley et al[2] has the opinion that careful preoperative assessment, correct selection of implant, and meticulous surgical technique, combined with aggressive post operative rehabilitation, offers the best chance of success in the management of these common fractures.

CONCLUSION

Hip fractures is one of the common injuries see at Orthopaedic and Trauma Department of Muhimbili Medical Centre. These are fractures primarily of the aged, in whom the incidence of other serious associated disease is high and tolerance to prolonged immobilization and bed rest is poor. The management of hip fractures continues to be one of the most challenging areas in the field of trauma, and more so in third world countries which are economically disadvantaged.

In our environment where most osteosynthesis materials are lacking the use of Girdlestone resection should be considered, similarly the use of local steroid injection for the control of pain. As soon as the pain is controlled, patients should be mobilized using crutches or other walking aids.

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3.3. TUBERCULOSIS OF THE SPINE AS SEEN AT MUHIMBILI MEDICAL CENTRE

SUMMARY

Between July to November 93, ten cases of Tuberculosis of the spine were admitted in the Department of Orthopaedic and Trauma of Muhimbili Medical Centre. Six were males and four were females, giving the male to female ratio of 3:2 (Table I). They ranged in age from 20 to 56 years, with mean age of 40.4 years. Two patients had evidence of BCG vaccination, they were both under the age of 30 years (Table I). The main mode of presentation included low backache, inability to walk, kyphosis and paraplegia (Table II). All the ten patients were managed conservatively with antituberculous therapy, which included Thiazina, Streptomycin and or Ethambutol; Rimactazid. They all responded well except for one patient who needed surgery for decompression of a paravertebral abscess which was causing progressive neurological deficit despite eight weeks of antituberculous therapy.

The shortest time of hospital stay was six weeks noted in those with no neurological affection. Two patients stayed in the wards for a period longer than six months, these were those presenting with neurological symptoms (Table V). There was no death.

INTRODUCTION

In the developing countries which usually are economically disadvantaged, Tuberculosis is one of the common conditions.[1] Similarly these countries being densely populated, has

inadequate housing, poor nutrition and public health preventive measures. These factors further worsen the prevalence of Tuberculosis. Tuberculosis of the spine is secondary to pulmonary tuberculosis.[1-3] The incidence of pulmonary tuberculosis has been studied and its prevalence well known, however the magnitude of Pott's disease is not well documented.[2,3] Kochi[2] quote a figure of about 1,700 million people or one third of the world's population as been infected with Mycobacterium tuberculosis. He further suggests that the overall proportion of infected people is similar in the Industrialized and Developing nations. However, 80% of infected individuals in the industrialized countries are aged 50 years or more while 75% of those in developing countries are less than 50 years old.

While pulmonary tuberculosis may be easy to treat at home or ambulantly, spinal tuberculosis and the sequelae of pott's paraplegia are disabling and usually leads to long periods of hospitalization.[1,4,5] Response to anti-tuberculous drug therapy is good and recovery depends much on the initial spinal cord involvement.[3,7] Early identification and proper treatment is therefore the cornerstone for successful treatment and hope for cure. Todate tuberculosis continues to be a disease of much concern worldwide. In 1990 alone for example it is estimated that there were 8 million new cases of tuberculosis in the Developing and Industrialized countries, and 2.9 million deaths occurred.[2] This made tuberculosis the largest cause of death from a single pathogen in the world. Such terrific figures prompted the author to undertake this study.

A typical Pott's disease patient is presented and the pathogenesis, diagnosis and treatment

of Tuberculosis of the spine is discussed. The salient features of the ten patients are also tabulated.

CASE REPORT

G.B. IP No. 759744, was a 30 year old male presented with low backache of six months duration, and inability to walk for a period of two weeks prior to admission. Previously he had been well, before the spontaneous onset of progressive back pain radiating to the left lumbar area. The pain had been aggravated by bending and coughing. No radiation had been experienced to the lower limbs. Two weeks prior to admission he developed fever and difficult in walking. He had to support his trunk with the hands while walking. No numbness was noted to the low extremities. Prior to onset of pain he had experienced night sweats, evening fevers and occasional dry cough. His wife was a known case of open pulmonary tuberculosis on treatment for the past one year. His appetite had been normal but admitted to have lost weight.

Clinical examination revealed a patient who was wasted and had mild pallor. Was not jaundiced, but was slightly febrile, with the axillary temperature reading at 38°C. He had a blood pressure of 110/70 mmHg and a pulse rate of 86 beats per minute which was regular and of full volume. No peripheral lymphadenopathy was noted. Had a gibbus at the level of the twelfth thoracic and first lumbar spine, with the skin at this level having local therapeutic marks. Marked tenderness was elicited at the same level, more so on flexion and extension of the spine. Kerning's and Lasegue signs were both negative. Muscle power and tone were normal and sensation was intact. Both the hip and knee

joints were normal. Respiratory, cardiovascular and abdominal examination were normal. A tentative impression of Tuberculosis of the spine was made with a differential of a vertebral tumor. The following investigations were done:–

1. Full blood picture + Erythrocyte sedimentation rate.
   - Hemogram revealed haemoglobin of 9 g/dl.
   - Total white blood cell count was 10,000/mm^3
   - Lymphocyte count of 40%
   - Neutrophil 60%
   - Rbc morphology – Normocytic normochromic film
   - ESR 105 mm in the first hour (Westergren).
2. Mantoux test was positive (14mm)
3. Plain radiographs:–
   - Chest x–ray was normal
   - Thoracolumbar x–rays revealed narrowing of the disc space between twelfth thoracic and first lumbar vertebrae. There was destruction of anterior part of the end plate of both T_{12} and L_1. There were no features suggestive of new bone formation or healing.
4. Serology tests:–
   - Brucella and Widal test were negative.
   - Elisa – negative
5. Sputum for Acid fast bacilli was not done since the patient was not producing

any sputum.

On the basis of history of low backache, inability to walk, evening fevers and weight loss. Also presence of a gibbus at $T_{12} - L_1$ and radiological features of destruction of $T_{12} - L_1$, a positive mantoux and a highly elevated ESR; Pott's disease was diagnosed. The patient was then started on antituberculous therapy, using streptomycin 0.75g and Thiazina 450mg daily.

Two weeks following the institution of anti-tuberculous therapy, the patient started improving and was discharged after nine weeks of hospitalization. On discharge he was advised that despite the apparent improvement, the treatment must continue using Thiazina for a total of 18 months. Similarly was advised to attend physiotherapy and to avoid heavy weight bearing. Was instructed to perform light duties only until recommended otherwise by the attending physician. Follow up was planned on monthly basis to evaluate his general condition, body weight and ESR plus control thoracolumbar x-rays. On his first month following discharge, he was found to be doing fine, had gained 0.5kg and the ESR had started dropping down. He is still being followed-up.

TABULAR SUMMARY

TABLE I: AGE AND SEX DISTRIBUTION OF PATIENTS

AGE IN YEARS	SEX		TOTAL NUMBER OF PATIENTS
	MALES	FEMALES	
15–20	1	0	1
21–30	1	0	1
31–40	1	1	2
41–50	2	2	4
51–60	1	1	2
TOTAL	6	4	10

TABLE II: FREQUENCY OF PRESENTING FEATURES

FEATURES	FREQUENCY IN PATIENTS
Symptom:	
• Backache	10
• Weight loss and anorexia	5
• Difficulty in walking	5
• Fever	5
Sign:	
• Local tenderness	10
• Gibbus	5
• Paraplegia	5
• Psoas abscess	2
• Paravertebral abscess	2

TABLE III: NUMBER OF PATIENTS IN RELATION TO THE LEVEL OF DISEASED SPINE.

LEVEL OF SPINE	NUMBER OF PATIENTS
Upper Cervical (C_{1-5})	0
Lower Cervical (C_{6-7})	0
Upper Thoracic (T_{1-4})	0
Mid Thoracic (T_{5-8})	2
Lower Thoracic (T_{9-12})	4
Lumbar	4
Sacral	0
TOTAL	10

TABLE IV: NUMBER OF VERTEBRAE INVOLVED IN PATIENTS WITH POTT'S DISEASE

NUMBER OF VERTEBRAE	NUMBER OF PATIENTS
1	0
2	8
3	0
4	2
5	0
TOTAL	10

TABLE V: THE DURATION OF HOSPITAL STAY IN TEN PATIENTS WITH POTT'S DISEASE.

DURATION IN MONTHS	NUMBER OF PATIENTS
< 2	4
2 – 6	4
> 6	2
TOTAL	10

DISCUSSION

AETIOLOGY AND INCIDENCE

Human and Bovine mycobacterium bacilli are the two main causes of tuberculosis of the spine. The bacilli reach the vertebra through venous plexus of Batson from a primary foci in either the lung, kidney or gut.[1,3–5]

Though it has become a rare disease in highly industrialized countries, bone and joint tuberculosis is still frequently encountered in the so–called developing countries, where tuberculosis is still prevalent.[1–3] Contrary other authors notes that the disease has not yet completely disappeared from Industrialized countries.[4,6,9] The spine is the most common location of bone and joint tuberculosis and the most serious location because of its anatomical position and possibility of involvement of the central nervous system.[3]

In most series, the disease is found to be more common in male than in female subjects.[1,2,5,9] This is in accord with the experience in this study (Table I). However

Martini[3] in his series had the disease being common in women than in men by 6%. It is useless to attempt to provide definitive statistics concerning the frequency of tuberculosis of the spine according to location because the figures given in the literature correspond to very different epidemiologic situations.[3] Although all vertebrae can be affected the majority of lesions are located between T_8 and L_3.[1,3,4,5] In this study, the lower thoracic and lumbar vertebrae were the most commonly affected followed by the mid thoracic vertebrae (Table III). Most patients had two vertebrae involved while few had more than two vertebrae (Table IV).

PATHOPHYSIOLOGY

Tuberculosis of the spine is always secondary to tuberculosis somewhere in the body and the patient usually presents with a history suggestive of prolonged tuberculosis infection.[1] The bacilli reach the vertebra through the venous plexus of Batson from a primary foci possibly in either the lung, kidney or gut, thus infection establishes to the vertebral body.[1,3] The tuberculous process nearly always commences in the vertebral body. The initial disease focus is usually located on the superior or inferior surface of the vertebral body immediately adjacent to the layer of hyaline cartilage covering these surfaces and the intervertebral disks.[3] The initial pre-purulent phase of granulation consists of an inflammatory reaction with Langhans giant cells, epithelioid and small inflammatory cells.[1,5] These are initially closely related to the vessels but spreads rapidly thrombosing the vessels and formation of cellular edema. Hypersensitivity and immunological reaction adds in augmenting the inflammatory process.

As infection and tissue necrosis progress, it then spreads to involve the rest of the vertebral body, gradually detaching and rupturing the vertebral cortex, the disk and the cartilage. Thus progressively sequestration occurs and penetrates the bony focus.[3,5,7] A paravertebral abscess is then formed, which is initially small surrounded by edema of paraspinal tissue. With increased tissue destruction the abscess increases in size, stripping further the periosteum off the vertebrae rendering them avascular.[3,7] The loss of substance leads to diminishing height of the disk, which is evident radiologically as narrowed intervertebral space. The extent of which is usually proportionate to the duration of the patients symptoms.[3,5-7]

As the bone destruction and sequestration proceeds, the infected vertebra becomes filled with caseous material, it is then gradually reduced to a flimsy nutshell.[3,5] This enables the development of kyphosis as the infected vertebrae rapidly collapses due to combined action of gravity and muscular contraction or in case of a trivial trauma.[1,3,4] Kyphosis of the spine is more marked when thoracic and thoraco-lumbar regions, are affected. This is due to a convex dorsal curvature which already exists normally. It is less so in the cervical and lumbar regions, where normal lordosis exists. Here the tuberculous lesions lead to straightening of the lordosis rather than kyphosis.[3,10]

The asymmetrical damage in the frontal plane is responsible for a certain degree of scoliosis, however it does not become significant as it tends not to continue increasing as kyphosis does.

Abscesses are the normal anatomical and biological result of large areas of destruction. They tend to spread along anatomical lines of little resistance particularly in tuberculosis of the spine.[3] However the paravertebral abscess may be forced backwards into the spinal canal compressing the cord, and leading to paraplegia.[4,9] The paravertebral abscess contains initially fluid and greenish–yellow pus. Later in the course of the disease it becomes thicker. When the abscess is in contact with the dura, an inflammatory reaction with pachymeningitis results and heals by fibrous tissue. This may strangulate the cord to cause late onset paraplegia.[9,11] The vertebral disc is an avascular structure and is not involved in the disease. This explains why there is absence of bony fusion in spinal tuberculosis foci, as the disco–cartilaginous volume is interposed between the vertebral bodies. However with severe bone destruction above and below, it can become completely detached and lie free in pus. This can then allow the ossification centre of collapsed vertebra to meet and thus new bone formation may result.[1,5] The most serious sequelae of spinal tuberculosis is the development of Pott's paraplegia. Since the establishment of its tuberculous origin, Pott's paraplegia has remained the most problematic form of bone and joint tuberculosis.[1,3,4,6,9,11,18] The pathology of paraplegia in Pott's disease is thought to be through the following mechanisms:–

(1) Segmental spinal vessel thrombosis leading to ischaemia of the cord.

(2) Angulation of the spine resulting in cord compression by internal gibbus. The severe the internal gibbus the higher is the chance of paraplegia.

(3) Sequestration and debris compressing the cord. Pressure may be exerted

posteriorly through posterior longitudinal ligament to compress the cord or by pus tracking through intervertebral foramen.

(4) Pressure of pus from a paraspinal abscess. In such cases resolution of the abscess or its drainage lead to improvement of paraplegia, giving a proof that tension from the abscess is the cause of the paraplegia.

Pott's paraplegia can be graded into four categories depending on the patients ability to walk. These grades carry a significant prognostic value.[1,3,9,11,17,18]

Grade I: In this grade there is exaggerated reflexes but the patient is able to walk unaided.

Grade II: The patient has a marked altered gait and needs external support to walk.

Grade III: The patient is bed ridden.

Grade IV: The patient is totally paraplegic.

Martini[3] notes that the frequency of Pott's paraplegia is the same for adults and children, and that there seems to be no differences in incidence according to sex.

CLINICAL FEATURES

The salient features include anorexia, easy fatigability, weight loss, night sweats or history of close contact with a family member known to have pulmonary tuberculosis.[1,3]

The commonest symptom is localized back pain accompanied with stiffness. Undue or mild localized pain is often the only symptom for weeks or months.[3,5,7] This fact added to the common occurrence of backache in modern life can explain the frequent long delay

in diagnosis. Clinically the patient presents with muscle spasm in the affected region. There is tenderness upon percussion of affected spinous processes, and in particular a slight kyphosis may be present in advanced states of the disease. Local tenderness and a gibbus are common signs and the presence of a gibbus is synonymous with established disease.[1]

If the cord is compressed the presentation depends much upon the level of the compression. Paraparesis or paraplegia is commoner with lower thoracic and lumbar spine involvement.[4,9] The cord compression by a tense paravertebral abscess produces heaviness in the lower limbs, an altered gait, exaggerated reflexes, and mild sphincteric tone changes. Also there may be muscle atrophy in the lower limbs. The indolent nature of Pott's disease makes the patients present late and are probably seen when the backache is so severe that one can no longer continue with normal daily activities.[1,3,4–7,9]

In this study all the ten patient had back pain and local tenderness. Five patients had features of tuberculous infection. Four patients presented with abscess with two paravertebral and two psoas respectively. Gibbus, difficult in walking and paraplegia was the presentation in five of the patients (Table II).

DIAGNOSIS

The mainstay in the diagnosis of tuberculosis lies in the demonstration of mycobacterium bacilli in the sputum, gastric washing or pus material from sinuses. Isolation of the bacilli from an abscess or the other samples indicates an active pulmonary, gastrointestinal, or spinal tuberculous infection. However attempts of mycobacterium identification is not

very rewarding in most instances due to the scanty nature of the bacilli in these specimen.[3] Culture and sensitivity tests takes not less than 6 weeks, which is a long duration of time to be of value in respect to early management of the patient. The importance however lies in indicating the sensitivity pattern of the drugs used in view of resistant strains or atypical species of mycobacteria.[5]

The Erythrocyte sedimentation rate and the lymphocyte count are usually raised in chronic granulomatous infection. The ESR may however be normal in up to 15% of patients with Pott's disease.[1] Thus the findings of these hematological examinations are not diagnostic of Pott's disease but in collaboration with other clinical findings, they become helpful.

Bone involvement can manifest in various other infectious diseases such as Typhoid, Brucellosis and Syphilis, thus special investigations including Widal, agglutination test for Brucella, and VDRL tests are also done to exclude such a possibility. Mantoux test is of little value in a community where a high percentage of the population is expected to be positive as a result of BCG vaccination or primary tuberculosis, as is the case in Tanzania.[8] However a cut off point of 15 mm, with ulceration may be indicative of an active infection.[2,8]

Radiologic findings, which constitute an important part of arriving at the presumed diagnosis, roughly correspond to the clinical findings of the inflammatory and destructive stages.[3,6,12] Antero-posterior and lateral x-rays together with conned view of the suspected area should be taken. Chest x-ray is mandatory to exclude pulmonary tuberculosis as a primary source of Pott's disease.

Spinal x–rays in typical cases of Pott's disease show the disease which commences on the antero superior or inferior aspect of the vertebral body. The loss of disc space at the affected area is an early radiological manifestation. Similarly the absence of sclerosis around the periphery of the lesion, and lack of evidence of new bone formation are typical of tuberculous lesion in contrast to pyogenic lesions of the bone.[1,7] If a paravertebral abscess is present, it produce, a soft tissue shadow usually evident in 30 to 60% of cases.[7] In this study two of the patients had this radiological evidence.

As the collapse of the anterior part of the vertebrae leads to a gibbus, plain radiographs of the spine are then useful in establishing the geometry of the deforming angle thus assessing its severity .[10] The internal gibbus angle as architecture by Dickson[13] is formed by two projected lines passing through the level of the posterior borders of the normal vertebral bodies proximal and distal to the site of the gibbus. The angle increases with severity of the gibbus, and is not affected by vertebral body disappearances. The external angle is made up by horizontal lines passing at the lower border of the first normal vertebra distal to the tuberculous lesion, and that passing at the upper border of the first normal vertebra proximal to the lesion. The external angle decreases with severity of the kyphosis but is increased with improvement of the gibbus. Grading of the gibbus severity according to Dickson[13] can thus be assessed by measuring the internal angle as follows:–

(1) Mild, if the angle is less than 30°

(2) Moderate, if the angle is between 30–50°

(3) Severe, when the angle is more than 50°

Radiological evaluation of kyphosis angle in case of a good response to treatment usually will show a decrease in internal kyphotic angle.[13]

In classical Pott's disease two vertebral bodies are usually involved together with the intervening discs. The tenth and eleventh thoracic vertebrae are highly susceptible, followed by the upper lumbar vertebrae.[1,5]

In this study the lower thoracic and lumbar vertebrae were the commonly involved followed by mid thoracic vertebrae (Table III).

Assessment for multiple involvement of more than a single site should be suspected in conventional radiographs of the spine, since 10% of new patients are reported to have multiple site involvement.[1] This was observed in two patients in whom four vertebrae were involved (Table IV).

Some authors advocate fine needle aspiration for cytology and or for culture and sensitivity, whereas others believe that bony surgical biopsy is best for confirmation of the diagnosis, particularly for surgeons working in countries where tuberculosis is still prevalent.[1,3,5] Surgical biopsy enables the taking of tissue samples which can then be sent for histologic and bacteriologic examination. The other diagnostic methods include the use of Magnetic image resonance and CAT scan. These are more informative than plain radiographs but are expensive to afford.[6,12] Similarly Technetium scanning can detect 5% of the lesions that are not picked on x-rays, and demonstrate multiple foci easily.

Proving the diagnosis in Pott's disease presents a problem since the isolation of mycobacteria is difficult and the sophisticated modern diagnostic techniques mentioned

above are not readily available in most developing countries. In such prevailing situation the history, and clinical features including plain x-ray findings can thus justify institution of antituberculous therapy.[3] In this study, diagnosis was based on clinical and x-ray findings, whereas laboratory demonstration of a raised Erythrocyte sedimentation rate and high lymphocyte count gave high index of suspicion.

MANAGEMENT

The management of the spinal tuberculosis is conservative. It however has some surgical indications, upon which it is a multidiciplinary calling for the general, orthopaedic and neurosurgical expertise.[1,3,6,10,11,18] Deciding on the appropriate treatment is a problem that each experienced surgeon who is aware of his own abilities and means at his disposal must himself resolve.[3]

Methods

1. Conservative – this includes supportive treatment by good nutrition and proper hygiene plus initial rest augments faster recovery.[1,3,6,13-15] The patient is admitted for investigation and once diagnosis is confirmed treatment is started.

   Chemotherapy does not pose any particular problem, treatment for eighteen months with first line drugs or eight months regimen with short course treatment effects cure of tuberculosis of the spine by 90%.[1,3,6,13-15] However Gillespie et al[14] noted in their series that effective chemotherapy in terms of dose, drugs and duration of treatment are key factors to wards successful treatment. In a study of 53 patients on longterm chemotherapy Scrimgeour et al[4] recorded success in 70%

of cases though only 18% were known to have completed 18 months course of chemotherapy.

The anti-tuberculous drugs penetrate well the thick walled paravertebral abscess to reach bacteriostatic levels even in caseous material. This property makes antituberculous drugs ideal in the treatment of spinal tuberculosis.[3,15] In this study one patient with paravertebral abscess was managed conservatively and recovered well (Table II). Drug combinations usually delay emergency of drug resistant strains, and the antituberculous effect is significantly higher than would be a single agent.[3,20] However the combination of three drugs has no added benefit over two drugs.[1,3] Nevertheless in patients who had already been on a long course of chemotherapy, and an initial drug resistance to standard drugs is known or believed to be common in that geographical area a three drug combination is used.[3,21]

Using a three drug combination consisting of INH, Rifampicin and Ethambutol, Moon et al[15] had favorable results in 95% of the 75 cases of spinal tuberculosis. Although the advantages of operative management in certain circumstances are obvious, conservative regimen can be confidently recommended for use in the less privileged countries where adequate facilities for treatment may not be readily available.[3,14,15,21]

2. Operative - combined with chemotherapy, surgery prevents an increase of kyphosis and its life-threatening consequences.

In Pott's disease surgery has a role in the following:–

i) Drainage of paravertebral abscesses and its associated sinuses. It is however noted that, paravertebral abscesses and sinuses in most instances do respond well to chemotherapy and hence rarely need surgical intervention.[3,21] No drainage of a paravertebral abscess should be done unless there is presence of pressure symptoms, radiological evidence of progressive increase of the abscess and development of neurological deficit. Similarly pointing skin abscesses failing to heal by aspiration need drainage.[1,3]

ii) Patients with progressive paresis leading to paraplegia despite antituberculous chemotherapy. Progressive paresis is an absolute indication for surgery.[19]

iii) Instability of the spine where surgical stabilization is indicated.

In treating tuberculosis of the spine surgically the objective is radical excision of the infected and devitalized tissues, decompression of the cord and interspinal neural elements. Likewise to achieve the reduction of spinal deformity and stabilization of the spine.[3,9,16,17–18] The earlier the decompression is done the better the treatment results.[3,6,17,18] In the early stages of paraplegia, treatment by removing the probable cause lead to good recovery whereas in the late stages the paraplegia is irreversible due to cord degeneration.

Antero–lateral approach is the standard approach in decompression in Pott's

disease.[3] The technique however carries a risk of weakening the vertebral column, which may lead to vertebral collapse and cord injury.[3,11,16] Laminectomy is rarely indicated since it destroys the healthy part of neural arch leading to instability of the spine.[11]

It is equally important to remember that Pott's paraplegia leads to prolonged hospitalization and close care is necessary to prevent pressure sores. Early rehabilitation to prevent contracture in a patient is also important. However early lifting weight should be avoided to prevent collapse of already weak vertebrae.

CONCLUSION

Tuberculosis of the spine can present as infectious disease or orthopedic problem to the spine. It is a debilitating disease which may lead to severe incapacitation once treatment is delayed. Confirming the diagnosis is not easy due to scarcity of mycobacteria tuberculosis in bone and joint lesions, thus determining appropriate indications for treatment is relatively difficult.

Chemotherapy makes the basis of treatment, given effectively it is the key to successful treatment. Apart from draining large abscesses or taking biopsy, the place of surgery in the early management of spinal tuberculosis is discretional. In advanced stages of the disease or when complicated with paraplegia and the response to chemotherapy alone is poor, surgery has a role to decompress the spine and radically excise the infected material. Similarly surgery may have value in stabilizing the spine so as to prevent an increase of kyphosis and its consequences.

Prevention of spinal tuberculosis can be achieved through combined efforts, including BCG vaccination in childhood and nonimmune population, plus early adequate and effective treatment of open pulmonary tuberculosis cases.

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