A DISSERTATION OF TEN SURGICAL CONDITIONS

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In partial fulfillment of the requirements for the award of the degree of Master of Medicine (Surgery) of the University of Dar es Salaam.

1990

UNIVERSITY OF DAR ES SALAAM

FACULTY OF MEDICINE

This is to certify that the ten Surgical Conditions presented in this book were managed by Dr. WANDWI during his postgraduate training in Surgery under my direct or delegated supervision.

Prof. B. R. Kirei

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DECLARATION

I 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 been submitted for any other academic award of any other University.

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ACKNOWLEDGEMENT

I wish to acknowledge my sincere appreciation and gratitude to all my colleagues for their tireless advice extended to me which has enabled me to produce this dissertation.

I am especially grateful to Drs: M. R. Aziz and S. M. Mlay my supervisors for their guidance, encouragement and valuable criticisms.

I am also sincerely grateful to my teachers in surgery: Prof. B. R. Kirei, Prof. P. M. Sarungi, Prof. J. K. Shija, Prof. B. M. Minja, Dr. E. M. Kessy, Dr. D. D. Charles, Dr. L.E. K. Lema, Dr. R. E. Kisanga, Dr. C. A. Mkony, Dr. J. Malliwah, Dr. R. Mhina, Dr. L. Mseru, Dr. A. Kinasha, Dr. S. M. Kinunda and Dr. O. Mcharo whom I had the pleasure to work with an learn.

Also I wish to thank the authorities of Muhimbili Medical Centre, the Nursing Staff and all other cadres for their assistance and cooperation.

Furthermore, I am exceedingly grateful to Ms Joyce Gondwe and Esther Jacob for their devoted and valuable secretarial assistance.

Dr. Wandwi, WBC

PREFACE

The specialization course in Surgery in the University of Dar es Salaam covers three years, two of which is devoted to general surgery while one year is spent in orthopaedic and trauma; ENT, Pediatric surgery, and Obstetric and Gynaecology specialities.

This dissertation forms a part of the requirement for the award of master of medicine degree in surgery. It consists of ten surgical conditions seen and entirely or partially managed by the author. It is divided into three parts.

Part one consists of six cases pertaining to general surgery, part two consists of two cases in urology, and part three, two cases in orthopaedic and trauma. All these have been studied during the three years of the course.

A detailed case report of one patient followed by review of characteristic features of the remaining patients in each subject and finally a review of literature is presented.

DEDICATION

To my wife Edda and our children Marinya and Martha, for their help, tolerance and moral family support which has enabled me produce this dissertation.

AND

To my father and mother for sending me to school and taking great concern in my upbringing.

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PERFORATED DUODENAL ULCER

SUMMARY

Eleven patients with duodenal ulcer were treated in three surgical firms of Muhimbili Medical Centre during a two year period from July 1987 to June 1989. There were 9 males and 2 females. Seventy two percent of these patients were aged between 30 and 50 years.

Previous history of peptic ulcer and sudden severe abdominal pain with peritonitis were the main modes of presentation.

Intra-operatively 10 patients were found to have chronic duodenal ulcer and one patient had acute duodenal ulcer.

Simple plication with omental patch and copious peritoneal lavage was done in 10 patients. In one patient truncal vagotomy and pyloroplasty was performed. In follow up of these patients no complication was noted.

INTRODUCTION

Duodenal ulcer is a worldwide disease with frequency varying from place to place[1,2]. In the UK duodenal ulcer complicated by perforation and haemorrhage accounts for 10% to 15% mortality rate in a year[3].

The incidence of duodenal ulcer in the West ranges from 1% to 20% with male preponderance[4]. In Africa duodenal ulcer is not uncommon[5].

In Tanzania, of the 320 patients endoscoped for duodenal ulcer suspicion 26.3% were confirmed to have the disease[6] and 23% of 733 patients who had barium meal studies had evidence of duodenal ulcer[7]. High prevalence of duodenal ulcer has been reported in Kilimanjaro region in Tanzania[8].

The incidence of acute perforation in patients with duodenal ulcer varies from 5% to 10% in patients with active disease process[2]. Thirty percent of patients with perforation have no previous history of duodenal ulcer[2,3].

The aetiology of duodenal ulcer and perforation is unknown; however, it has been associated with stress, salicylates, and alcohol ingestion.

A case of perforated duodenal ulcer is presented in detail and a review of the ten others is presented. The frequence, aetiology pathogenesis, clinical features and treatment of perforated duodenal ulcer is reviewed.

CASE REPORT

Mr. J.K. IP. No. 467126 a 36 years old male was admitted with a 6 hours history of sudden onset of epigastric pain associated with nausea, vomiting and sweating. The pain was gripping in nature. On admission the pain had spread to the right lumbar area. There was no haematemesis, malena or fever prior to the onset of the pain. For the past two years he had been

experiencing epigastric pain, worse with hunger and relieved with meals.

Late at night the pain would wake him up to be relieved with a cup of milk or antacid. During this time he had pain exarcebation lasting up to two weeks followed by remission of two to three months duration.

He had been attended in the peripheral hospital and was told that he had a duodenal ulcer and was started on antacids.

He had no history of weight loss, and denied history of admission due to a similar problem. He was a peasant with no smoking history, alcohol ingestion or acetylsalicylate drug ingestion.

On examination he looked ill and was lying quietly in bed. He was dehydrated, not pale, afibrile with a temperature of 38°C. The pulse rate was 100/min regular and of small volume. His blood pressure was 90/70 mmHg.

The abdomen was moderately distended and was not moving with respiration. There was generalised guarding, and loss of hepatic dullness and bowel sounds were absent. Digital rectal examination revealed distended rectal ampulla and tenderness in both iliac fossae. The other systems were essentially normal.

A provisional diagnosis peritonitis due to peptic ulcer perforation was made, with differential diagnosis of

acute cholecystitis, acute appendicitis, and acute gastritis.

An intravenous line was established using a wide bore cannula, blood samples were collected for serum electrolytes, urea and creatinine, grouping and crossmatch. A nasogastric tube for continuous gastric drainage and urethral catheter were passed.

A chest X-ray together with an erect and supine abdominal x-rays were taken. Both revealed gas under the right hemidiaphragm. On the basis of clinical and X-ray findings a diagnosis of perforated duodenal ulcer was confirmed and decision to operate was made.

The patient was planned for emergency laparatomy. He was given gentamycin 80 mg and metronidazole 500mg intravenously pre-operatively. After one and half hours he had 1.5L and vital signs were stable. Under general anaesthesia the abdomen was cleaned drapped as usual, then opened through an upper midline incision. The findings were purulent bile strained peritoneal fluid which was sucked to reveal an anterior perforation of the first part of the duodenum surrounded by fibrous, exudate. The duodenal wall was oedematous. The perforation was closed with three interrupted 2/0 silk sutures and an omental patch sutured over the already tied stitches.

Copious peritoneal lavage with warm saline was done and the abdomen was closed in two layers with corrugated rubber drain in Morrisons pouch.

Post operatively the patient was monitored half hourly recording pulse rate, blood pressure, and respiratory rate until he gained full consciousness, thereafter he was monitored after every four hours for 24 hours. Urine output and serum electrolytes were normal.

He had one litre of Hartman's solution 0.5 litres normal saline, and 1.5 litres Dextrose 5% for the first 24 hours post-operatively. Pethidine 100mg every 8 hours was given to relieve pain. Gentamycin 80 mg intramusculary for every 12 hours and metronidazole intravenously 500 mg for every 8 hours were given for seven days.

On the first post-operative day, the patient's vital signs were stable, urine output was adequate. The abdomen was soft and dressings were dry with minimal discharge from the drainage site. He was maintained on intravenous fluids for the next 24 hours. The drain was removed after 24 hours post-operative for it was not draining.

On the second post-operative day, he had no cough, and was sitting up in bed. He had passed flatus. The chest was normal and the abdomen was soft. The nasogastric tube and urethral catheter were removed for they were no longer needed. He was allowed to take oral sips and continued with intravenous drip.

On the third post-operative day he was expressing hunger and had passed stools. He had no other

complaints. The intravenous drip was discontinued and removed and patient was ambulated and allowed light feeds.

He fared well and on the eighth post-operative day the stitches were removed and he was discharged on the ninth day with a date to attend follow up clinic for assessment and plan for definitive surgery in three months interval. During this time he was to take antacids.

He was admitted four moths later for truncal vagotomy and pyloroplasty. A week before the operation he was investigated and found to have haemoglobin of 12 g/dl. A day before operation he was fasted and a nasogastric tube inserted before going to theatre. Under general anaesthesia, the anterior abdomen was prepared and drapped. The abdomen was opened through the previous upper midline incision to find lot of adhesions particularly at the site of previous perforation. The distal end of oesophagus was identified, the peritoneum over this part was incised taking care not to perforate the oesophagus while encircling it. With traction on the fundus the posterior left vagus was identified as a taut cord. It was resected between clumps and ends tied. The same was done to the anterior right vagus. The resected vagal stumps were sent for histology. Further palpation with traction at

the fundus of the stomach did not reveal remnant of nerve fibres anteriorly or posteriorly.

The pylorus was identified found to be indurated and fibrotic. Heineke-Miculicz pyloroplasty was performed using catgut 2/0 for seromuscular and silk 2/0 serosal layer. The abdomen was closed in layers. Post-operative nasogastric aspiration half hourly, intravenous fluid dextrose 5%, 2½ litres, normal saline 0.5L, and pethidine 100 mg every 8 hours for analgesia were given. The patient was monitored; blood pressure, pulse, and respiratory rates half hourly for the first 4 hours post-operatively then for every two hours for the next 24 hours.

On the second post-operative day he had no fever, nor cough the abdomen was soft and chest was clear. He continued with nasogastric tube and intravenous fluids. On the third post-operative day he had passed flatus and had no complaints. The nasogastric tube, intravenous drip were stopped. He was ambulant and started oral fluids.

The rest of the post-operative period was uneventful. On the eighth day the stitches were removed. He was discharged and followed up in outpatient clinic three weeks later. He was in good condition. He was discharged from the clinic a year after definitive surgery.

RESULTS

The results of 11 patients with perforated duodenal ulcer is tabulated:

Table 1: Age distribution in 11 patients with perforated Duodenal Ulcer.

Age in Years	No of patients	%age
20 - 30	2	18.2
31 - 40	3	27.3
41 - 50	5	45.5

Table II: Symptoms and signs in 11 patients with perforated Duodenal Ulcer.

Presentation	Frequency in 11 patients
Severe epigastric pain	11
Loss of hepatic dullness	11
Peritonitis	11
Long history of dyspepsia	8
Nausea	5
Fainting	4

Table III: Management of perforated Duodenal Ulcer in 11 patients.

Type of closure	No. of patients
Simple plication and omental patch	9
Simple plication with truncal	1
Vagotomy and pyloroplasty	1

DISCUSSION

Aetiology

There is no known aetiology of duodenal ulceration. Only some associated factors have been suggested like socio-economic status, diet, cigarette smoking, alcohol, drugs and heredity, and Campylobacter, pyloridis renamed as Helicobacter pylori.

Duodenal ulcer develops as a result of acid-pepsin digestion of duodenal mucosa. Ulcerative process is a manifestation of an imbalance between acid pepsin secretion and mucosal resistance.

High hydrochloric acid production is believed to be the cause of duodenal ulcer. Ulcers do not occur in achlorhydric patients. Patients with duodenal ulcer have high basal and maximum acid outputs.

The increased acid output may be due to excessive hormonal gastrin stimulation, excessive vagal impulse, defective acid production, auto regulation; or increased parietal cell mass[1,10]. Not all patients with duodenal ulcer however have high acid levels.

There is an association between high carbohydrate consumption with duodenal ulcer. Smokers have twice the risk of developing duodenal ulcer compared to the general population. Smoking predisposes to both duodenal and gastric ulceration and impair ulcer healing[11,12].

In normal circumstances there is a mucosal barrier maintained by mucus and glycocalyx layer which guards against hydrogen ion reabsorption into the mucosa. In peptic ulceration this protective layer is defective allowing reabsorption of hydrogen ions which stimulates mast cells to produce inflammatory mediators which include histamine.

These leads to mucosal oedema and inflammation both of which make ulceration easier and stimulates more acid production. The pancreatic alkaline secretions and prostaglandin E_2 synthesised by gastric mucosa are also protective[13].

Irritants to gastric mucosa lead to breach of the protective glycocalyx layer predisposing to duodenal ulceration, via increased acid production stimulated from excessive histamine release due to back diffusion of hydrogen ions into the mucosa.

Salicylates, prolonged use of corticosteroids, non-steroidal antinflammatory drugs, alcohol, caffeine, are known to impair the protective mucosal resistance by exfoliative process and epithelial cell damage.

Tobacco smoking is associated with inhibition of pancreatic bicarbonate secretion, increase acid secretory capacity leading to increased acidity in duodenal bulb. Smoking also inhibits ulcer healing in 20% of duodenal ulcer patients[13].

Heredity appears to be important since duodenal ulceration runs in families. There is a three times increased risk in siblings compared to the general population. Those with blood group O and non secretors blood group A, B, O (H), are more predisposed to duodenal ulcer diathesis[13,14].

Duodenal ulcer is common in all social classes. That hard working executives are more susceptible to duodenal ulcer due to stress seems to have small effect[11].

Extragastric conditions such as pulmonary affections like empyema, liver disease, endocrine abnormalities like phaechromocytoma and hyperparthydoidism have been associated with duodenal ulcer. Systemic mastocytosis due to excessive histamine production is associated with duodenal ulcer. In Zollinger Ellison syndrome there is gastric hyperacidity due to gastrin secreting tumour from the pancreas. Males are more affected than females with ratios of 5:1[6,8,15], within the age range of 30 to 50 years[8,16] (Table I).

Most of our patients were between 30 and 50 years. No specific aetiological factor was noted in them (Table II).

Pathology

Perforation is seen in 10% of duodenal ulcer patients[13]. Ulcers on the anterior wall of the duodenum perforate while those in posterior wall leads

to penetration into pancreas. This may erode the gastroduodenal artery leading to haemorrhage, presenting as haematemesis and or malena.

In acute perforations, a patient has no previous symptoms of duodenal ulcer disease or has history of less than two months. Acute perforation account for 20% to 30% of all perforations and at surgery there is hyperaemia with no scarring, or fibrosis around the perforation.

In chronic duodenal ulcer there is history of more than three months duration suggestive of duodenal ulcer disease and during surgery, the margin of the perforation is indurated with adhesions on the serosa and fibrosis, scaring and a distorted first part of duodenum.

After perforation there may occasionally be spontaneous sealing off by omentum and fibrin plug. Failure of this leads to continued duodenal content spillage into the peritonium. This leads to chemical peritonitis resulting from digestive action of hydrochloric acid and pepsin on parietal and visceral peritonium. This result in increased capillary permeability and exudation of fluid and electrolytes into the gut lumen. There is sequestration of fluid in the inflammed peritoneal cavity and visceral wall leading to a fall in blood volume and consequent hypovalaemic shock. As

peritonitis progresses sepsis sets in from infected gastric content spillage.

Ileus associated with vomiting contributes to fluid and electrolyte imbalance as fluid sequestration continues this leads to circulatory failure, infection and finally death.

CLINICAL FEATURES

Characteristically perforated duodenal ulcer presents as sudden sharp epigastric pain which progressively gets worse, to become generalised involving the whole abdomen. There may be pain radiation to one or both of the shouders.

Occassionally in some patients pain may remain localised at the epigastrium if free diffusion of duodenal contents is restricted by inflammation and adhesions. Spread of the contents to the right paracolic gutter may cause pain that is difficult to differentiate from acute cholecystitis and appendicitis[13,17].

Pain is due to peritonitis and in the elderly and immunocompromised patients pain is minimal. This causes delay in diagnosis and leads to poor prognosis in this group of patients.

There is usually a previous history suggestive of duodenal ulcer or a patient may even be a known peptic ulcer patient on treatment. About 20% of duodenal ulcer patients are however asymptomatic, only to be

discovered when complicated by perforation.

On examination the patient appear dehydrated and is febrile particularly if he presents late. There is rise in pulse rate, which is feeble. The blood pressure may be low depending on the degree of hypovolaemia.

The abdomen distends and become tense with guarding and board like rigidity. Hepatic dullness is lost and bowel sounds are absent due to ileus[10].

The diagnosis is confirmed by the demonstration of pneumoperitonium on a plain abdominal x-ray erect or left decubitus position. Pneumoperitonium is demonstrable in 75% to 80% of patients with duodenal ulcer performation[15]. To be demonstrable the perforation should have occurred at least three hours prior to radiological examination[18]. X-ray are taken after the patient has been in the erect position for a duration of between 10 to 20 minutes[2,19]. Left decubitus film has an advantage of detecting smaller volumes of pnemopentonioum.

In perforated duodenal ulcer there is raised serum amylase but this is a non-specific laboratory finding.

In this study all 11 patients presented with severe abdominal pain and had peritonitis with loss of hepatic dullness. A long history suggestive of duodenal ulcer was evident in 8 out of the 11 patients (Table II).

TREATMENT

Resuscitation with intravenous fluids and electrolytes, nasogastric decopression of the stomach and antibiotics are the first line measures in the treatment of duodenal ulcer perforation. Specific treatment is divided into conservative and operative measures.

Conservative treatment consists of continuous nasogastric aspiration, intravenous antibiotics, fluids and electrolytes, coupled with frequent observation and evaluation for relief of abdominal pain, symptoms and signs of peritonitis.

This approach is based on the assumption that spontaneous closure of some small duodenal perforation by the undersurface of the liver, fibrin plug, and adjacent omentum may occur[5, 17].

However, this approach is associated with high mortality rate of 10% to 50% and has thus been left for those patients who cannot withstand surgery who are in irreversible shock, the very old and debilitated patients with organic disease such as cardiac failure, or those refusing surgery[12,20-22]. None of the patients in this study was managed conservatively.

Operative procedure is the best treatment in perforated duodenal ulcer for it is difficult to predict which patient will close spontaneously by conservative management. The choice lies between simple plication

or immediate definitive surgery. In the patients studied in this presentation only one had immediate definitive surgery and 10 had simple plication (Table III).

Simple suture plication is done with or without an omental patch. The omental patch has an extra advantage in closure of the perforation and leakage is minimised. The omental patch can be taken as free graft or as a pedicle.[18]

Although simple plication has no definitive effect on duodenal ulcer diathesis, it has been noted that a third of the patients after the procedure are asymptomatic and do not need any other treatment,[15,18] a third will be symptomatic needing additional surgery,[18,21] and the remaining third will be controlled by medical therapy.[15,20-24]

Simple plication is a life saving procedure and is adequate treatment in patients with acute perforation. In poor risk patients those with massive intraperitoneal sepsis or serious concurrent disease, the procedure can be accomplished quickly with minimal operating time[25]. The mortality associated with this procedure is 2% to 10%, and complications increase with age over 50 years and associated medical diseases. Delay of over six hours from perforation to time of surgery increases the mortality[21].

In cases where the perforation is found to be well sealed only peritoneal lavage and drainage of Morrison's pouch is done.

Immediate definitive surgery is done in selected cases. It carries increased mortality compared to simple plication only[22]. The patients meets the criteria for definitive surgery, definitive operation should be thus performed to avoid the second operation[22,26].

Criteria for definitive surgery in perforated duodenal ulcer is that the patient should be in reasonably good condition and ideal surgical and anaesthetic facilities are available. Definitive surgery is performed in the following situations:[16].

i) Where there is combined perforation and haemorrhage.

ii) In cases where perforation is associated with pyloric stenosis.

iii) Where chronic duodenal ulcer is evident clinically or intraoperatively.

iv) In cases when the patient's condition is clinically assessed and found fit to withstand an additional operative time.

v) In situation where duodenal and gastric ulcers are present with one of them perforated.

Elective definitve surgery is indicated in those patients who receive simple surgery alone at the time of perforation and have constantly had persistent pain,

or evidence of chronic duodenal ulceration findings inraoperatively. The modes of definitive surgery available are:

i) Truncal vagotomy with drainange.
ii) Truncal vagotomy with antrectomy.
iii Selective vagotomy with drainage.
iv) Highly selective vagotomy.
v) Or partial gastrectomy.

The choice of the type of definitive surgery is determined by mortality and recurrance rate, post operative complications, and a surgeon's experience with a particular technique.

The aim of definitive surgery is to suppress ulcer diathesis, achieved with low risk of side effects, mortality and recurrance rate.

Truncal vagotomy and drainage is the simplest, commonly practised procedure. It takes less operative time and mortality rate is in the range of 1% to 5%[2,3,27]. The recurrence rate is 3% to 5% over 5 to 10 years. The only aspect associated with this procedure that benefits the patient is the reduction of acid and pepsin with ulcer healing, the rest is detrimental. Truncal vagotomy and drainage causes loss of antral mill and propulsion, dumping, increased risk of gallstones, and impairment of pancreatic enzymes ouput in response to a meal. There is also alteration of glucose insulin metabolism with increased faecal fat[3].

The drainage procedure is done as single or double layer, Heineke-Mikulicz, Finney or Jabouley type of pyloraplasty. There is no advantage of any one of the type of pyloroplasty procedures chosen[23].

The alternative drainage is gastrojejunostomy, but this has high risk of stomal ulcers, billious vomiting, dumping and it is thus less popularly used.

All out 11 patients had truncal vagotomy and Heineke Mickulicz pyloroplasty.

Truncal vagotomy and antrectomy has the effect of removing the cephalic phase of gastric secretion with the least recurrence rate of 1% to 2% compared to all other procedures[23]. It caries a higher mortality of 5 to 15%[26] and has 30% chance of gallstone complication[30]. This procedure is ideal for those patients with prepyloric ulcer or duodenal ulcer with gastric outlet obstruction or those patients who had truncal vagotomy and pyloroplasty in the past.

Highly selective vagotomy has low mortality of 0.3% and minimal side effects. The ulcer recurrence rate with this proceeding is 5% to 10%[3,13,30] and the procedure takes a longer operative time. Dumping, diarrhoea, reflux oesophagitis, and billious vomiting are less common but post-prandial fullness due to failure of receptive relaxation of the denervated body of stomach is common.

The high failure rate in highly selective vagotomy is due to inadequate denervation. The lesser curvature may become inchaemic due to seromuscular denervation procedure in this technique.

Partial gastrectomy is indicated where perforation co-exists with gastric outlet obstruction. In this operation two thirds distal stomach is removed[13]. The procedure has a highest mortality of over 15%, compared to other procedures.

All patients after definitive surgery require up to five years follow up to evaluate the efficacy of the procedure done, and looking for complications.

Complications of duodenal ulcer perforation surgery are mainly intraperitoneal sepsis, wound sepsis, and the side effects associated with definitive surgery.

Intraperitoneal sepsis accounts for most of post-operative mortality due to peritoneal soiling by duodenal contents and the incidence increases with the longer the perforation time up to surgery. After 24 hours 64% of peritoneal cultures become positive compared to 20% after 6 hours. Copious peritoneal lavage with warm saline and drainage of Morrison's pouch, associated with perioperative antibiotics against upper gastrointestinal organisms limits sepsis[26]. In our patients with perforation, 6 developed wound sepsis. In the two years follow up of these patients no complication associated with definitive treatment was noted.

CONCLUSION

Perforated duodenal ulcer is a surgical emergency needing urgent attention. The earlier the perforation is closed and peritoneal lavage done the better is the prognosis. Simple plication and omental patch is the ideal procedure in all emergency perforations. This is followed by elective definitive surgery if patient persists with symptoms of duodenal ulcer diathesis. Immediate definitive surgery should be reserved to experienced hands and in healthy yough patients preferably with acute perforation presenting very early with short time between perforation and surgery.

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PORTAL HYPERTENSION

SUMMARY:

Five patients with portal hypertension were managed by the author between the period covering July 1987 and June 1989. The age range was 30 to 50 years with male to female ration of 3:1. The commonest presentation was haematemesis and splenomegaly.

Three patients had history of jaundice in the past, and the other two had previously been treated for Schistosoma mansoni. The sizes of the spleen varied from 6 to 10 centimeters below the costal margin. Ascitis was present in two patients. Endoscopy revealed lower third oesophageal varices in all patients. The portal pressure was recorded in three patients at laparatomy and ranged between 30 to 36 centimeters of normal saline.

INTRODUCTION

Portal hypertension is not uncommon; it is life threatening due to massive upper gastrointestinal bleeding from oesophageal varices[1]. 60% of patients die within a year of haemorrhage and 60% of the survivors rebleed within a year of first bleeding episode[2]. The common causes of portal hypertension in theory are preventable; liver cirrhosis due to alcohol or hepatitis B infection, can be prevented by decreasing alcohol intake, and vaccination against hepatitis B virus respectively.

Equally, schistosomal infestation control will lower the incidence of pipestem fibrosis and hence portal hypertension. Furthermore, hygienic delivery will decrease the incidence of omphalitis which predisposes to portal vein thrombosis and hence portal hypertension[1,3,4].

In UK the incidence of portal hypertension presenting with bleeding oesophageal varices accounts for 2.5% of patients admitted in hospital due to haematemesis. In U.S.A. the incidence is higher 10% to 15%. The mortality from bleeding oesophageal varices secondary to portal hypertension is 25% to 59% in patient needing intensive care management for resuscitation, control of bleeding, and confirmation of the bleeding site[5]. There are no statistics available in Africa as regards to the magnitude of this condition[2]. The magnitude, treatment of portal hypertension and its complications as seen at Muhimbili Medical Centre is reviewed in this study.

CASE PRESENTATION

Miss PC No.482059 33 years old was admitted into the surgical department ward with main complaint of vomiting blood. This was her third episode. She spontaneously vomited about a litre of altered blood. There was no previous history of epigastric pain or passage of black stools. She denied taking drugs prior to this complaint.

About five years ago she had developed yellow eyes for a week which was associated with fever malaise and pain in the right hypochondrium.

She did not take alcohol nor smoke had no significant social and family history. On examination she was pale, not jaundiced and well oriented to person, time and place. The BP was 100/60 mmHg, pulse rate was 90/min. feeble with cold clammy extremities. She had no skin hyperpigmentation, foetor hepaticus, no palmar erythema or Duputryens contractures.

The abdomen, was slightly distended, with no distended veins. On palpation the abdomen was soft, with splenomegaly of 8 centimeters below the costal margin; the liver was not palpable. Shifting dullness was positive. Bowel sounds were normal. Digital rectal examination revealed malena. A provisional diagnosis of portal hypertension with bleeding oesophageal varices was made. An intravenous line was established with a wide bore cannula and blood for grouping and crossmatch taken. She got 2.5L Hartman's solution rapidly. The pulse and blood pressure were monitored half hourly. Nasogastric tube was introduced and aspiration revealed altered blood. Lavage with cold saline was done till clear fluid was evident. Repeated aspiration did not show active bleeding.

A urethral catheter was passed and drained about 200 mls. of concentrated urine. It was left in situ to

duodenum were normal. The patient had five points (Grade A) under modified Child-Garder criteria by Pugh (1973) (Table IV). Neither the Sangstaken tube or sclerotherapy facility was available.

On the basis of her good hepatic functional reserve, her age and the recurrent severe episodes of haemorrhage, an elective decompressive shunt operation after correction of anaemia to haemoglobin of 12g/dl was planned.

Operation: The abdomen was opened through left paramedian incision, which was extended obliquely to give optimal exposure. Findings were: yellowish ascitic fluid; a cirrhotic liver, an enlarged spleen, and engorged mensenteric veins. The rest of the organs were normal.

Portal pressure, measured by cannulation of the left gastroepiploic vein and advancing the catheter to portal vein was 30 centimeters of normal saline.

A decision to do proximal spleno-renal-shunt was made. Splenectomy and mobilisation of splenic vein was done. The left renal vein was identified and anastomotic area prepared. End to side anastomosis was done with prolene 4/0 between vascular clamps. A wedge liver biopsy was taken. Haemostasis was achieved. The post shunt portal pressure was recorded to be 21 centimeters of normal saline. The abdomen was closed in layers.

Post operatively: the patient was given intravenous fluid normal saline half a litre and dextrose 5% 2.5

Table II: Signs in five patients with portal hypertension

Sign	Frequency
Splenomegaly	5
Anaemia	4
Ascitis	4
Caput medusa	1

Table III: Grades of liver functional reserve in five patients using Child-Pugh Classification

Grade	No. of patients
A	3
B	1
C	1
Total	5

DISCUSSION

Aetiology

Portal hypertension results from the obstruction to the flow of portal blood[1,2,4], or when there is increased blood flow into the splanchinic bed[4]. The normal pressure ranges from 5 to 25cm of normal saline though majority of patients have pressures below 8cm of normal saline. Davey in Nigeria found most Africans undergoing laparatomy to have portal pressure average of 14.4cm. of normal saline with a range of 8.5cm to

21.5cm. normal saline, while in Uganda the average was 16.7cm. and this was associated with big spleen.[2]

The cause of portal hypertension can broadly be classified as prehepatic, intrahepatic, or post hepatic[6] and that due to increased hepatopetal flow without obstruction.[3,4]

Prehepatic (Infrahepatic) obstruction accounts for 20% of all cases.[7] Portal vein occlusion may be by thrombus, extrinsic pressure, or congenital absence (artresia) of portal vein.

This aetiological group of portal hypertension is not associated with hepatocellular dysfunction.[3] The patients are usually children or adults of young age. Omphalitis is the main route of infection to the umbilical vein and finally to portal vein causing thrombosis.[3]

Dehydration and umbilical vein cannulation have been implicated as causes of portal hypertension by leading to thrombosis of the vein in children, but this is rare.[4] Cavernous malformation is another rare causes of portal hypertension.[3] Blunt abdominal trauma, pregnancy and pancreatitis, predisposes to thrombi of the portal vein, while pancreatic neoplasia causes extra hepatic obstruction of portal vein [4,7]. Patients with prehepatic portal hypertension have very high portal pressure and they bleed readily than those with portal hypertension from other causes.[4]

Intrahepatic causes accounts for about 80% of causes of portal hypertension.[3,7]

Intrahepatic presinusoidal block is when the lesion is within the portal space with increased portal hypertension while wedged hepatic pressure is normal. This is seen in Schistosomal infestation. Schistosoma ova reach small venous tributaries in portal tract, and causes granulation and obstruction of portal space just like, in leukaemia; Hodgkin's lymphoma and other reticulosis; congenital hepatic fibrosis; and sarcoidosis.[1,4]

In intrahepatic sinusoidal portal hypertension, liver cirrhosis of any type is the main aetiological factor world wide. It accounts for 30% of cases of portal hypertension in East Africa. The regenerated liver nodules distort the sinusoids with venous obstruction [1,2,4].

In posthepatic causes of portal hypertension, the obstruction to hepatic blood flow is in the hepatic vein, or there is right sided heart failure.

Increased portal blood flow as in massive splenomegaly where there is 10% to 20% increased blood flow, leads to portal hypertension. This is seen in hepatic artery-portal venous fistula or in splenic artery-

portal venous fistula, and in cases of intrasplenic enlargement as in tropical splenomegaly.

The last aetiological factor in portal hypertension is, idiopathic type where no obstruction can be demonstrated radiologically in portal venous system and primary liver disease is not evident. Histology shows only deranged lobule structure with portal tract fibrosis, despite lack of clinical and biochemical disease.

In the patient presented above the presence of positive hepatitis B surface antigen indicates that there is chronic intrahepatic lesion leading to the portal hypertension. Biopsy confirmed cirrhosis as liver pathology leading to portal hypertension.

Pathophysiology

The portal pressure is a dynamic fluctuating force, with variations depending upon position of patient and phase of respiration.

Whenever there is an increase in intra-abdominal pressure as in defaecation, coughing or muscular contraction the portal pressure rises. The portal pressure depends on blood flow and pressure transmitted to portal bed. The effect of the raised portal pressure has two main effects:- the development of splenomegaly by simple congestion, and development of collateral circulation[1,3], which though numerous are of small calibre and do not effectively decompress the

portal system. The higher value of portal pressure is recorded in patients with marked collaterals.[2] This is in effort of trying to find the way to the heart, and this occurs in four areas.

Inferiorly - at the anal canal where anastomosis between the superior rectal vein with middle and inferior rectal veins occur. This may present as haemorrhoid.[6]

Anteriorly - through the recanalised umbilical vein when the left branch of portal vein is patent connects to the inferior and superior epigastric veins around the umbilicus. This type of anastomosis is responsible for Cruveihier Baumgarten syndrome characterized by dilated abdominal wall veins, venous hum and thrill at the umbilicus. This is always associated with intrahepatic obstruction.[6]

At the posterior abdominal wall collaterals develop between lumbar veins and retroperitoneal vessels.[4,6]

Superiorly, at distal third of the oesophagus, the left gastric and short gastric veins are connected to azygous vein draining the oesophagus. This is the most important surgical collateral, forming varices which when bleeding require surgical intervention.[6,2]

Clinical Features

The major manifestations of portal hypertension are; oesophageal varices, ascitis, splenomegaly,

hypersplenism and hepatic coma.

Patients with portal hypertension may remain latent for months or years or may not have symptoms for life. The commonest presentation is gastrointestinal bleeding and ascitis.[6] Oesophageal varices rupture and bleeding constitutes the most significant complications of portal hypertension and is a major indication for surgical decompression.[3] Though there is no good relation of portal pressure and variceal bleeding, no varices bleed with portal pressure below 10 mmHg.[4] Predisposition to bleeding episode is related to size of the varices; factors which increase pressure in the varices; ulceration secondary to oesophagitis; or drug and alcohol erosions[3,4]. Thrombocytopaenia due to hypersplenism is not implicated.[3] In adults, portal hypertension is usually secondary to intrahepatic disease, and 90% of patients with portal hypertension have varices.[8] In children varices are secondary to portal vein obstruction, 70% bleed before the age of seven and it is extremely rare for them to die of first haemorrhage.[9]

Over 80% of patients with portal hypertension rebleed massively within two years.[3] In children profuse haemorrhage almost always originates from bleeding varices and acute haemorrhage represents the first manifestation of portal hypertension in childhood; as in contrast to adults where a quarter to one third of

massive bleeding is due to oesophageal varices.[1,3] The time from the initial diagnosis to the first haemorrhage varies and it is extremely rare for bleeding to occur within two years of initial observation of the lesions.[3] Survival of patients who did not bleed at the time of diagnosis of varices was identical to that of patients in whom varices, were discovered after bleeding, indicating little improvement in rate of survival in all groups despite aggressive variceal decompression therapy. The cause of death is haemorrhage and hepatic failure.[11]

In cirrhotics, oesophageal varices cause bleeding in 50% of patients while gastric and duodenal ulcer accounts to 25% and 15% respectively. Correlation of the lesions with severity of bleeding reveals that patients bleeding from varices have severe haemorrhage compared to those due other cause of bleeding.[3]

Mortality rate associated with bleeding is different in different causes of portal hypertension. That of cirrhotic patients is higher than that from extrahepatic portal vein thrombosis. Mortality is directly related to a number of haemorrhagic episodes with cirrhosis reaching 50% after five bleeding episodes and non-cirrhotics only reaching 10%.[3] Patients dying within a year after initial haemorrhage range from 60% to 80%.

Ascitis and splenomegaly present as abdominal swelling noticed by the patient. Ascitis to be clinically detected has to be more than two litres. When it is severe, the patient will develop respiratory embarrassment. The splenic size and ascitis are not corralated directly with the level portal pressure. Rarely is there hypersplenism where reduction of one or more cellular elements of the blood is evident.[3] Failure to palpate the spleen in a patient with hematemesis is a good indicator that bleeding is not coming from the varices.[3,13]

Hepatic coma presentation is rare in children below age of twelve year,[3] while coma and blood loss are major causes of death in cirrhotic patient with upper gastrointestinal bleeding.[13]

Patient present with pyschiatric symptom, impaired; motor response, consciousness, deep tendon reflexes, with flapping tremor and foetor hepaticus.[3] Foetor hepaticus in liver failure, suggests an extensive collateral circulation in diseased liver which bypasses sinusoids and join the hepatic vein and the amount of blood passing through this shunt varies greatly.[14,15,16]

Diagnosis

Commonly a patient presents with a history of heamatemesis. Other features include a history

of pain in upper left quadrant, because of splenomegaly, abdominal distension due to ascitis, and marked engorged abdominal veins. On examination the patient is pale and depending on the volume of blood loss, might present with shock. On abdominal examination, ascitis, and splenomegaly are evident. There may be visible abdominal veins. The other signs of liver failure might be evident, like palmar erythema, spider naevi, jaundice and hyperpigmentation due to excessive melanin deposition.

Investigations are helpful in diagnosis of portal hypertension and in planning management of the patient. Liver functions tests, where serum bilirubin, transaminases, alkaline phosphatase, and serum albumin are measured. Prothrombin time is also estimated to determine impairment of Vitamin K, dependent clotting factors. Liver biopsy and Hepatitis B surface antigen are also done.

Barium swallow demonstrate the varices as rounded, filling defects projecting from the wall of oesophagus. Endoscopy is essential to locate the level and extent of varices. This is usually done as an emergency in the first twenty four hour in a patient presenting with acute gastrointestinal bleeding to rule out other causes of bleeding.

Stool is examined for malena and ova. Protosigmoidoscopy and rectal biopsy will exclude

Schistosimasis. Manometry gives the portal pressure levels. This can be done through different methods and it is important that at the same sitting portal venography be performed to establish the patency of the portal vein which is essential before doing any type of elective portal systemic ∫hunts. Ultrasound of the abdomen with emphasis on the liver, portal and splenic vein diameter help to diagnose portal hypertension.

Prognosis of the patients with varice is related to the cause. Portal hypertension due to cirrhosis, with varice whether they had bled or not has poor prognosis.[11] Undecompensated liver carries better prognosis and once bleeding has occurred the prognosis is related to severity of bleeding and degree of loss of hepatic function.[11] Renewed bleeding cause 70% of deaths in patient with good liver function. Patient with non cirrhotic presinusoidal portal hypertension have better prognosis than cirrhotics, and benefit more by correcting pathological component that i the high gastroesophageal pressure.

Treatment

Patients usually present with upper gastrointestinal bleeding and the initial treatment is resuscitation with fluid, blood replacement and close monitoring of vital signs followed by identification of the bleeding site by emergency endoscopy.

Cold saline gastric lavage through a nasogastric tube

and if bleeding continues then tamponade using Sengken-Blakemore tube is applied within six hours of onset of bleeding for 12 to 24 hours, being 90% effective.[17,18] Aspiration, oesophageal rupture, and 6% to 60% rebleeding risk after removal of the tube are drawback in its use.

Vasopressin (pitressin) has been used to reduce portal pressure and contract oesophageal musculature. It can be given as bolus, by continued infusion, or selectively into inferior mensenteric artery. Conventional bolus dose of 20 units given in 200mls of 5% dextrose intravenously over 10 to 20 minutes does not permanently stop bleeding. It is effective in 20% to 50% of patients. Low dose 0.4 units per minute infusion of vasopressin stops bleeding in 80% of cases. Intra-arterial injection of Vasopressin has no advantage compared to bolus doe. Vasopressin is avoided in patients with coronary heart insufficiency due to it cardiac output and pulmonary blood flow decreasing effect. Abdominal cramps and flushing indicate optimal dose of vasopressin.[19] Glycepressin, an analogue of vasopressin is preferred due to its prolonged vasomotor effect and is more effective than vasopressin. Also somatostatin which reduce splanchnic blood flow and portal pressure has been tried, given as an infusion for 12-24 hours controlling the bleeding. These two drug are however not commonly used due to cost. Propranolol at a dose of 20-180mg twice daily

threatening condition.

Management of these patients entails emergency diagnotic endoscopy and arrest of bleeding by sclerotherapy. Unfortunately these facilities are not readily available. Replacement of blood loss by crystalloids and blood transfusion continue to be the only hope in managing these patients. The future should see development of endoscopy facilities at least at the regional and national hospitals and these should go a long way in control of life-threatening haemorrhage from varices.

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CHOLELITHIASIS

SUMMARY

The author participated in the management of 11 patients with gallstones admitted in the Department of Surgery in Muhimbili Medical Centre during a period covering July 1987 to June 1989.

There were 10 females and one male patients. The peak age was 30 to 40 years. Most of the patients presented with right upper quadrant pain and dyspepsia.

Abdominal ultrasound was the single most diagnostic investigation. Elective cholecystectomy was the mode of treatment for all patients. No anatomical anomaly of the biliary tree was noted in this series.

INTRODUCTION

Cholelithiasis is more frequent in Western world than in developing countries[1]. Its prevalence varies from country to country. In UK 10% to 15% of adult population have gallstones[2], while in USA it is up to 20%;[1,3] in both places cholesterol being the main stone constituent. In Africa few cases are reported,[4] however, improved living standards, increased longevity of life, with western dietary habit acquisition will make gallstone disease a relatively common disorder. Pigment or mixed stones predominate in developing countries.[5]

Gallstones are either symptomatic or asymptomatic. The incidence of asymptomatic stone varies. It is highest

in UK (70% to 80%)[6] and lowest in USA (4%).[7] Symptomatic gallstones disease presentation in tropical countries is similar to that in developed countries. The difference is in predisposing factors, type of stones, diagnostic methods but not treatment.[8] Cholecystectomy is the standard procedure when the gall bladder is diseased or contains calculi.[9] In this study the pathogenesis, stages of gallstone formation, predisposing factors, clinical presentation, complications and mode of treatment of cholelithiasis is reviewed.

CASE REPORT

Mrs E.N. IP NO. 530131 30 years African was admitted with sudden onset of right hypochondrial pain, radiating to the scapula. The pain started three hours after supper consisting of fatty food. It was colicky and she persistently rolled on the bed with no relief. There were no aggravating or relieving factors of the pain noted. She was nauseated but was not vomiting. In the past she had experienced intolerance to fat. There were no history of haematemesis or malena. She had no history of jaundice, fever nor chills. She did not smoke nor drink. She worked as a secretary, and had two children. She was not on the pill. Systemic review was insignificant. On examination, she was obese, afebrile, in pain, not jaundiced and not pale.

Her blood pressure was 120/80 mm of mercury. Pulse rate was 100/min regular and of full volume. The abdomen was not distended and was moving with respiration, there was no distended veins. Palpation revealed a soft abdomen with no organomegaly. The right hypochondrium was slightly tender. Murphy's sign was negative. There was no ascitis and the bowel sounds were normal. Digital vaginal and rectal examination were normal.

Other systems were normal. A provisional diagnosis of billiary colic was made and the patient was offered bed rest, injection pethidine 100mg, atropine 0.6mg intramuscularly stat, and asked not to take anything orally. The pulse, blood pressure and respiratory rate were monitored every two hours. The patient felt better after four hours, she had no pain then and the vital signs remained normal. She was investigated as follows:

Investigations	Results
Urinalysis	No bilirubin
Stool	Brown formed stools no ova or cyst
Serum Bilirubin (umol/1)	15
ALAT (IU/L)	20
ASAT (IU/L)	25
Alkaline phosphatase (IU/L)	90
White blood cells	
- Total (mm^3)	9500
- Differential	Leukocytosis
Serum Electrolytes mmol/1	Na^+ 143, K^+ 3.1, Cl^- 107

On the basis of these findings a diagnosis of cholelithiasis was reached. The patient was informed, cholecystectomy was planned and she was prepared for the operation with her consent six weeks after the attack of billiary colic.

Operation

With the patient in supine position, general anaesthesia was given and the abdomen prepared and drapped in the usual way. Through a right Kochers incision the abdomen was opened and on inspection the abdominal viscera was found to be normal except the gallbladder which had three palpable gallstones. Cholecystectomy was done, beginning from the hilum, after exposing the cystic duct and artery. The cystic artery in Calot's triangle was ligated with silk 2/0 and divided between ligatures. The cystic duct was identified confirmed and was then divided in the same way. Then the gallbladder was dissected from its liver bed. A sample of bile was aspirated for culture and sensitivity before starting cholecystectomy. The gallbladder after cholecystectomy was opened and found to contain three black stones of equal sizes about half a centimeter in diameter, bathed in greenish bile. The gall stones were sent for biochemical analysis and the gall bladder for histopathological study. Gall bladder bed haemostasis was achieved, the abdomen closed with

0-nylon in single layer with drain in Morrison's pouch and skin with prolene 3/0. Postoperative, pethidine 100mg intramuscularly every 8 hours; intravenous fluids 5% Dextrose, 2 litres alternating with normal saline 1.5L in 24 hours were given.

Vital signs, pulse, blood pressure, and respiratory rate were monitored four hourly. The patient made uneventful recovery. Bile sample revealed sterile culture. Stitches were removed on the seventh day, and patient was discharged with an eight weeks out-patient appointment. Two months later she was seen and found to have no complaint. The histological results of the gallbladder indicated chronic inflammation and no malignancy. Biochemical analysis of the gallstones revealed mixed, calcium, bilirubinate stones.

RESULTS

Salient features: type of stones, and mode of treatment, sex, age, parity, clinical presentation, diagnosis, in the 11 patients is presented in Tables (I - VII).

Table I: Sex distribution in 11 patients with cholelithiasis

Sex	No. of patients	Percentage
Male	1	9
Female	10	91
Total	11	100

Table II: Age distribution in 11 patients with cholelithiasis

Age group in (years)	No. of Patients
15 - 20	1
25 - 30	1
31 - 35	5
36 - 40	2
41 - 45	0
46 - 50	2
Total	11

Table III: Parity of 10 female patients with cholelithiasis.

Number of children	Number of female patients
0	1
1	0
2	5
3	3
4	0
5	1
5+	0
Total	10

Table IV: Clinical presentation of 11 patients with cholelithiasis

Symptoms	Frequency
Right hypochondrial pain	11
Dyspepsia	4
Vomiting	3
Fat intolerance	2
Signs	
Right hypochondrial tenderness	11
Jaundice	1
Fever	4

Table V: Diagnostic investigation in 11 patients with cholelithiasis

Investigations	Results			
	Positive		Negative	
	No.of Patients	%age	No.of Patients	%age
Plain abdomen X-ray	2	18	9	82
Oral Cholelystography*	7	87.5	1	12.5
Abdominal ultrasound	11	100	-	-

*Three patients had no visualisation of the gall bladder

Table VI: Type of stones in 11 patients

Type of stone	Number of Patients	%age
Mixed	9	81
Pigment	1	9
Cholesterol	1	9
Total	11	100

Table VII: Mode of treatment in 11 patients with cholelithiasis

Type of treatment	No. of Patients
Cholecystectomy	9
Cholecystectomy & choledoctotomy	2
Total	11

DISCUSSION

Stone composition

In the developed world the site of stone formation is invariably in the gallbladder, while in Orients, pigment stones form mainly in the bile duct.[5] There

are three types of stones namely: cholesterol stones, pigment, and mixed stones.[2,3,5]

Cholesterol stones are common in developed world and only a small number are composed of pure cholesterol monohydrate.[3] Mixed stones are common, predominantly containing cholesterol plus about 60%, calcium salts[7] bile acids, bile pigments, fatty acids, proteins and phospholipids.[2,7]

Black pigment stones are composed of calcium bilirubinate. Brown pigmented stones seen in developing world and in far East are associated with bacterial infection,[6] sometimes Salmonela typhi bacteria are cultured from the nucleus of these stones.[1,2,6] In our patients most gall stones were of mixed type (Table VI).

Pathogenesis

Cholesterol containing stones are related geographically to other diseases characteristic of western civilisation like heart disease, diabetes mellitus and diverticular disease of the colon.[1,3]

These stones are formed from the three constituents of bile, as a result of cholesterol precipitation after imbalance in concentration of these constituents, namely bile salts, phospholipids and cholesterol. The bile salts are water soluble forming miscelle in acqueous solution. Lecithin and cholesterol which are

insoluble in water are kept in solution in incorporation forming fixed misceles. The relative proportion of these three components in bile determine whether cholesterol is maintained in miscellar solution or precipitated to form microcystals which aggregate to form gallstones.[6,10,11]

The two mechanism appearing to be responsible for cholesterol gall stone formation are: increased hepatic cholesterol secretion and decreased bile acid pool.[6,11] Increased hepatic cholesterol secretion is the main cause of saturated bile. The enzyme hydroxymethy glutaryl Co-A reductase is important in cholesterol synthesis, its hyperactivity leads to excess cholesterol secretion by the liver causing supersaturation, and precipitation of micro-cystals of cholesterol. This is because there is excess cholesterol present in relation to concentration of two solubilisers of cholesterol that is lecithin and bile salts.[3,6,11,12]

Bile salts deficiency in patients with decreased circulating bile acid pool reduces cholesterol dissolving capacity. This deficiency can be caused by the decreased activity of hepatic cholesterol 7a-hydroxylase enzyme which is rate limiting in bile acid synthesis. This can be congenital or acquired by eating refined carbohydrates.[3,6]

Pigment stones, are formed by precipitation of free insoluble bilirubin in bile. The origin of unconjugated bilirubin is from enzymatic action of B glucoronidase which deconjugates bilirubin diglucuronide to free bilirubin which combines with calcium to form calcium bilirubinate. Usually the normal gall bladder contains glucaric acid an inhibitor of B-glucuronidase and it is thought that pigment stones are formed when the equilibrium between B-gluconidase and its inhibitors are offset.[3]

In some cases the liver has abnormally excessive secretion of unconjugated bilirubin leading to pigment stones.[10]

In gall stone formation, there are four stages through which all types of stones undergo before they are clinically evident.[3,8,14,15]

First is a metabolic defect or predisposing factor leading to increased cholesterol or decreased bile salts in case of cholesterol stones. In pigment stones there is an increase in unconjugated bilirubin.

Second is chemical stage where bile is saturated but has no crystals of cholesterol or bilirubin. Saturation is a prerequisite but does not in itself cause formation of stones, many people without stones have saturated bile especially after prolonged fasting.[10]

In the third or nucleation stage, bile is saturated and contains microscopic crystals. Solid crystals occur by nucleation which can occur in two forms: Coalescence of cholesterol or pigment molecules forming nidus for further precipitation. This is seen in highly supersaturated bile. Second form of nucleation is by heterogenous nucleation in which precipitation occurs around some particle other than crystals of pure cholesterol or bilirubin. So particles in bile like mucus, bacteria or calcium acting as nidus.

Generally there is rapid nucleation in patients with gallstones than in persons without gallstones.[3,11]

Fourth is growth stage where the so formed stone gradually increases in size. Decreased evacuation of gall bladder, or concentration and any condition leading to decreased in bile salts enhances growth of gall stones.

Predisposing factors

Heredity, obesity, female sex, lithogenic drugs, haemolytic diseases, liver cirrhosis and ileal diseases or resection do predispose to cholelithiasis. Infection of gall bladder is also a predisposing factor.

Cholesterol gallstones have a high familial incidence and show high incidence in certain racial groups with the American Indians having a 70% chance of developing cholesterol stones.[8,11,14] The prevalence of

gallstones is higher in first degree of relatives with gallstones.

The abnormality in cholesterol stones lies in the hydroxymethyl glutaryl Co A reductase enzyme hyperactivity or cholesterol 7a - hydroxylase deficiency.

The method of inheritance is not yet known.

In obesity, fat people have increased body fat and hence increased liver synthesis and excretion of cholesterol. A contributory factor is the lithogenic drugs like clofibrate used to reduce cholesterol and weight. Diet rich in cholesterol also causes excessive cholesterol excretion and hence ending up with gall stone formation.

Female sex has high incidence of developing cholelithiasis than males about two to three times commoner than in males.[6,8,14] This is because of suppression of hepatic bile acid secretion by female sex hormones.

Pregnancy, oestogen therapy and use of oral contraceptives all increases biliary cholesterol secretion and susceptibility to gall stones.[1,4]

Haemolytic diseases are predisposing factors in pigment stones. Sickle cell disease and other haemolytic conditions have increased incidence of pigment stones due to increased indirect bilirubin excretion into bile

by the liver.[5,8,16] Mechanism of increased gall stone formation in patients with cirrhosis is unknown.[3]

Clinical conditions that cause excessive bile acid loss as in extensive ileal disease, or ileal resection or jejunal ileal by pass operations has increased tendency of gallstone formation.[14] The effect of defective feedback regulation of bile acid synthesis in which bile acid synthesis is suppressed by abnormally high small quantities of bile salts returning to the liver through enterohepatic circulation.[8,10,11] Accelerated intestinal transit time increases recycling by speeding delivery of bile salts to site of absorption.[10]

Delayed gall-bladder emptying has high incidence of gall bladder stone formation as seen in truncal vagotomy in treatment of duodenal ulcer. Delayed gall bladder emptying, hyperallmentation and fasting leads to sludging and supersaturation.[3,10]

Drugs that lead to loss of bile salt, like cholestyramine decreases bile acid pool and predisposes to cholelithiasis.

Infection predisposes gall stones in that, the infective organisms e.g. E. Coli. is rich in B-glucuronidase enzyme which act on conjugated bilirubin, deconjugating it and frees birubin leading to formation

There occurs symptoms in more than 50% of patients with cholelithiasis, and calculus obstruction of the cystic duct leads to billiary colic or acute inflammation of the gall bladder.

Calculous obstruction of the common bile duct occurs in 10% to 20% of patients causing incomplete and intermittent obstruction.[3,14]

In patients with asymptomatic gallstones 50% have risk of being symptomatic and some of them develop severe complications with advancement of age.[3,6,8,17]

Billiary colic is the commonly encountered symptom in both calculous obstruction of gall bladder outlet and common bile duct. The pain is usually located in right upper quadrant with radiation to right scapula.[3,8] The pain occur at unpredictable times and characteristically it begins suddenly after a fatty meal and persists for one to four hours and is not intensified by movement. There is relief after passage of stone. Pain persisting more than five hours is likely to complicate acute cholecystitis. Nausea, dyspepsia and vomiting accompany cholelithiasis.

Jaundice is noted in 25% of patients with gallbladder outlet calculi obstruction probably due to oedema or external compression of the common bile duct.[3]

In common bile duct calculi obstruction there is jaundice which can be intermittent. Fever is present in acute cholecystitis and cholangitis as a result of obstruction in cystic duct and common bile duct, predisposing to infection secondary to stasis.

The abdomen is usually tender with local guarding. Deep inspiration during palpation of the right upper quadrant produces pain and inspiratory arrest. (Murphy's sign).

White blood cells count is raised with left shift, and serum bilirubin, alkaline phosphatase, and transminases is usually raised in 30% to 40% of the patients.[3,19]

Acute cholecystitis is the commonest complication of prolonged or persistent calculous obstruction of the cystic duct. The inflammation results from four factors:[5,14]

i) Obstruction causes epithelial injury and increased intraluminal pressure, which enhances inflammation.

ii) Ischaemia results from increased intraluminal pressure.

iii) Chemical injury contributed by prostaglandins and lysolecithin, released as a result of phospholipase-A action on Lecithin.

iv) Bacterial proliferation in stagnant bile causes further inflammation. Sixty percent of positive bacterial cultures are recorded in gall bladders removed for acute cholecystitis.

Perforation of gall bladder, fistula formation, gallstone ileus, porcelain gall bladder, hydropic gall bladder, while: pancreatitis, intrahepatic stones, secondary cholangitis, empyema, and bleeding tendency due to Vit. K dependent coagulation factors deficiency are the complications of common bile duct stone.[2,3,12-14]

Acalculous cholecystitis develops after trauma, burns, or any surgical procedure. It runs similar clinical course as calculous cholecystitis. Acute post traumatic cholecystitis is seen in young men after injury while acute post operative cholecystitis is evident in elderly men after abdominal operation. In our series billiary colic and dyspepsia were the main symptoms while fever and right upper quadrant tenderness and fever were the common signs (Table IV).

Investigations

Investigations include white blood cells, total and differential, this is abnormal if there is an element of infection as in cholangitis or acute cholecystitis.

Serum alkaline phosphatase, bilirubin and transaminases are usually abnormal in case of common bile duct obstruction and if there is pancreatitis complicated by choledocholithiasis serum amylase is raised. Urine analysis and stool analysis will be supportive in assessing the degree of common bile duct obstruction by absence of urobilinogen in urine and stercobilinogen in stools, while where will be excess direct bilirubin spillage in urine. In a jaundiced patient prothrombin time is mandatory for it assess the degree of impairment of Vit. K dependent coagulation factors. Diagnostic investigations are those able to show stones in the billiary free.

Plain radiography of the abdomen may be able to show opaque gallstones in 10% to 20% of patients.[2,19] Asymptomatic stones are visualised on the X-ray taken for investigating other conditions. However the majority of gall stones, about 80% are not radiopaque so are missed. Plain abdomen radiograph is helpful in diagnosis of emphysematous cholecystitis showing intraluminal air, and porcelain gall bladder or limy bile.[3]

The prime use of oral cholecystograph is for visualisation of radioluscent gallstone, assessment of

gall bladder function and differentiation of opacities in right hypochondrium.[2,19] In 95% oral cholecystography accurately demonstrates the presence of gall stone with 5% giving false negative.[3] A fatty meal is given and stimulates evacuation of gall bladder.

Failure to opacify can be due to diarrhoea, vomiting, or impaired absorption of the contrast.

Acute inflammation of gall bladder leads to poor concentration of contrast causing poor or non visualisation of the gall bladder. About 10% to 30% of gall bladders that do not become opacifiable with first dose become visible with second dose.[3,19] If the direct bilirubin is more than 35 umo/L there is non concentration of the contrast and non visualisation of the gall bladder.

Intravenous cholangiography is indicated in patient with upper quadrant pain with non visualisation on oral cholecystograms. A normal opacifying duct with non filling of gall bladder is suggestive of acute cholecystitis in patients with acute abdomen, or cystic duct obstruction in patient with palpable gall bladder. The patient must be well hydrated and the contrast needs a normal hepatocyte to be excreted. It is given slowly intravenously. In cases of liver disease or

obstructive jaundice with raised bilirubin there is no dye excretion.

Ultrasound is 95% sensitive and most accurate. It is however less accurate in diagnosis of acalculous cholecystitis or bile duct stones.

From the investigations carried out in our patients ultrasound was 100% diagnostic and oral cholecystography had about 12.5% false negative. Plain abdominal x-ray was only helpful in 19% (Table V). The results of abdominal x-ray and ultrasonography compares well with literature.[3,12,13,19] An ultrasound is cheap and free from side effects compared to oral cholecystography. It may be used as a diagnostic test of gall bladder disease in developing countries.[20]

TREATMENT

Surgical

Cholecystectomy is the mode of treatment of cholelithiasis unless there are risk factors to surgical intervation.

Asymptomatic and symptomatic gall stones are treated equally. The silent stones have 50% of chance of becoming symptomatic needing cholecystectomy within 10 years.[8,3] So early surgery is a treatment of choice. The timing of cholecystectomy in case of acute cholecystitis is not uniformly agreed upon. There are

those advocating conservative or delayed operation in which cholecystectomy is performed in six weeks to three months. Usually acute cholecystitis resolves in about 48 hours and emergency operation amidst inflammation which may obscure anatomy and lead to technical error; and congested vessels in inflammation lead to spread of infection. A patient is prescribed bed rest, nasogastric tube, opiate anaelgesics and atropine to relax sphincture of odd.

The other school of management is that advocating eraly cholecystectomy. The main reason for urgent operation is fear of perforation and that most of the risks advanced above, regarding inflammation usually are not encountered in emergency cholecystectomy.[7,14] Early operation is advocated in this centre. The use of antibiotics preoperatively against bacterial isolates in gall bladder is continued postoperatively depending on culture and sensitivity to prevents septiscaemia and wound sepsis.[22]

The complications of cholelithiasis are also managed by cholecystectomy which may be performed either from the hilum moving to the fundus or fundus first.

The previous approach is best, for in ligating the cystic artery and cystic duct, the risk of gallstone migration is minimized, and the operation is less bloody. However in fibrosis of hilum due to inflammation and in very fat patients, the fundus first

approach can be used. Drainage of foramen of Winslow is essential to safeguard billiary leak from liver bed of gallbladder.

Cholecystotomy is indicated in very sick patients who will not stand further time in cholecystectomy. The gallbladder is opened and gallstone and bile is evacuated and then drained percutaneously. This procedure is sometimes a standard treatment in very old patients who have short life span that they will not live long enough to form more stones.[7,9,14,23]

Choledocholithotomy is indicated in presence of gall stones in common bile duct. Preparation of patients for common bile duct exploration includes correcting bleeding indices, by giving Vit. K injection intramusculary 10mg daily until prothrombine time is at least about 80% of normal. Also antibiotics are indicated due to chronic cholangitis and prevention of wound sepsis.[22]

The following are the indications for common bile exploration:[14]

Absolute indications are:

i) palpable gallstone in common bile duct (is 99%) reliable)

ii) jaundice with cholangitis (is 97% reliable)

iii) demonstration of gallstone by cholangiography (is 85% reliable)

iv) dilatation of common bile duct more than 12mm (is 35% reliable)

Relative indications are:

v) obstructive jaundice, recent or recurrent

vi) biliary enteric fistula involving the common bile duct

vii) small stone in the gall bladder

viii) single faceted stone in gall bladder

ix) pancreatitis

In this respect then the common bile duct must be exposed by mobilising the duodenum by Kocherisation and the surgeon palpates the entire common bile duct.

Technical aspect of exploration involves intra-preoperative cholangiography where the tube is inserted through the cystic duct and intraoperative cholangiography done. Some do routine intraoperative cholangiographs for three reasons:[3]

i) ruling out choledocholithiasis,

ii) locating bile duct culculi to avoid blind choledocholithotomy,

iii) to decide if or not to explore a patient who has relative indication of exploration.

After exploration the stones are removed by Dormia basket or Desjardins forceps, then completion cholangiography is done through a T- tube. To make sure there are no stones left in common bile duct intra opertive billiary endoscopy may be performed.[24]

The T-tube in common bile duct is used for post operative cholangiography. In cases of retained stone,

after 14 days it is removed and the formed tract acts as a guide through which a tube is introduced and the stone removed by Dormia basket, or choledoduonoscopy is performed with removal of the stone.[7,14,24]

In cases of impacted common bile duct stones, sphincterotomy of the sphincture of Oddi and flushing the common bile duct with normal saline or heparinised saline leads to passage of stone.[14] Sphincterotomy can be done through endoscopy with removal of impacted stone.[25,26]

Gallstone lithotripsy[27] and medical treatment by resolution of cholestrol stone using chenodeoxycholic or ursodeoxycholic acid[28] are other modes of treatment.

In 11 patients we managed, cholecystectomy was the mode of treatment and only two patients had common bile duct exploration after palpating gallstone in common bile duct in one patient; while the second one had a relative indication for exploration due to intermitent jaundice. (Table VII). No intraoperative cholangiography was done. Post operative cholangiography was normal in both patients. T-tube was removed on the 9th day and fistula closed after a further four days.

CONCLUSION

Cholelithiasis is increasingly diagnosed in our environment. Mixed and pigment stones are commonly encountered.

The introduction of ultrasound has possibly increased the frequency of diagnosis of cholelithiasis. Cholecystectomy still remains the best method of management. Unfortunately ERCP, percutaneous transhepatic cholangiography facilities are not available in Tanzania. Yet these would go a long way in the diagnosis of patients with jaundice.

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TRAUMATIC RUPTURE OF THE SPLEEN

SUMMARY

Fifteen patients who had undergone splenectomy for traumatic rupture of the spleen at Muhimbili Medical Centre under the care of the author between July 1987 and June 1989 are presented.

All were males, and 12 (80%) of these patients were aged between 10 and 25 years. Assaults and falls from heights were the commonest cause of splenic injury followed by road traffic accidents.

Abdominal pain, bruises on the abdominal wall, abdominal distension and shifting dullness were the main mode of presentation.

Head injury was the commonest association. Splenectomy was the only mode of treatment offered to these patients. Splenic laceration and fragmentation were noted to be the commonest types of splenic injury. There was no death.

INTRODUCTION

For over 2000 year the spleen has been interesting in that it was regarded as an organ full of mystery. It has been thought to contribute to imaginative functions, anger, speech of an individual and even influencing the speed of running.[1] Melpighi and Clarke demonstrated in animal studies that the spleen was not vital for life, after noting normal life in splenectomised animals.[1]

A report of increased susceptibility to infection after splenectomy in children[2,3] and adults[4] prompted the studies of the function and role of the spleen in certain infections. It is established that the spleen has a role in both cellular and humoral immunity. It elaborates immunoglobulin M antibodies and is a major site for production of opsonins and tuftsin and it is important in phagocytosis especially of capsulated organisms.[5]

This has changed the concept in the management of splenic injuries from splenectomy to a more conservative approach consisting of non operative management to splenic sparing operation. However splenectomy is still the treatment of choice of ruptured spleen, at centres lacking monitoring facilities. In Kampala Uganda ruptured spleen was noted to be a main indication for splenectomy.[5]

The normal spleen in an adult is a friable vascular organ, rich in lymphoid tissue, playing vital haematological and immunological functions. It is located in the left upper quadrant of the abdomen, weighing 18 gm and is 12 cm in length.[1]

The small size and location below the rib cage appear to protect the spleen from injury. However despite this protection the spleen is the commonest intra abdominal organ injured in blunt abdominal trauma.[1,2]

In this study of 15 patients one patient is presented

in detail and the salient features, review of literature and management of splenic injury is given.

CASE REPORT

Mr. W.C., a 22 years old patient was admitted to hospital early in the morning after being assaulted by unknown people the previous night. He remembers to have been kicked in the abdomen and hit on the head before he lost consciousness. During admission he was concious, complaining of thirst and upper abdominal pain referred to the left shoulder. The pain was more felt wherever the patient coughed or laid down. He denied history of vomiting or bleeding from the ears or nose.

Physical examination revealed bruises on the face, left forearm, and thigh. He was pale, afebrile, with cold sweating extrimities. The pulse rate was 105/min feeble and the blood pressure was 90/70mmHg. His respiration rate was 28/min. The abdomen was distended with decreased movement with respiration. There were bruises over the left hypochondrium. Palpation revealed generalised tenderness in the left hypochondrium. The liver was not enlarged. There was shifting dullness and bowel sounds were diminished. Digital examination revealed no abnormality.

A preliminary diagnosis of intra-abdominal haemorrhage due to splenic rupture was made. The patient was resuscitated and planned for emergency laparatomy.

An intravenous line with a wide bore cannula was established. Blood was taken for grouping and crossmatch, and the patient was started on intravenous infusion of Ringer lactate. A nasogastric tube and a urethral catheter were inserted, and 200 mls of clear concentrated urine was drained. The patients vital signs were monitored every fifteen minutes. He had 2.5L Ringer lactate fast unitl the blood pressure rose to 100/80 mmHg before laparatomy.

Under general anaesthesia the abdominal wall was cleaned and drapped. The abdomen was opened through an upper midline incision. There was 1.5L of blood in the peritonium which was collected for autotransfusion. Abdominal exploration revealed an enlarged lacerated spleen, the laceration was located on the costal surface.

The spleen was delivered at the wound after division of the lienorenal, lienocolic, and gastrosplenic ligaments. The hillum of the spleed was exposed posteriorly. The splenic artery and vein were divided between clumps separately and transfixed with silk 2/0. After achieving haemostasis the abdominal cavity was mopped dry of the clots and peritoneal lavage with warm saline done. The abdomen was closed in layers.

Post-operatively the patient was given two units of blood and continued with intravenous infusion, dextrose 5% 1.5L and Ringer lactate one litre for 24 hours.

He was given pathidine 100mg intramusculary 8 hourly for 24 hours. Vital signs were monitored every quarter an hour until he was awake thereafter every two hours for 24 hours.

On the first day postoperatively he had normal urine output and the vital signs were stable. The urinary catheter and nasogastric tube were removed. Forty-eight hours after splenectomy the intravenous drip was stopped and the patient started on oral feeds.

He had an uneventful recovery. Stitches were removed on the seventh day and he was discharged in good condition. He was followed up for one year and had no problem.

RESULTS

The features of patients with traumatic rupture of the spleen in this study is summarized in Table I to V. All the patients were male and all had undergone splenectomy.

Table I: The age distribution in 15 patients with ruptured spleen.

Age in Years	Frequency	Percentage
10 - 15	4	26.7
16 - 20	5	33.3
21 - 25	3	20.0
26 - 30	2	13.3
31 - 35	-	-
> 35	1	6.7
TOTAL	15	100

Table II: Type of splenic injury noted at laparatomy in 15 patients.

Type of injury	No. of Patients	Percentage
Laceration	9	60.0
Fragmentation	5	33.3
Subcapsular haematoma	1	6.7
TOTAL	15	100

Table III: Causes of splenic injury in 15 patients.

Cause	No. of Patients	Percentage
Assault	6	40.0
Falls from height	5	33.3
Motor traffic accident	4	26.7

Table IV: Clinical features in 15 patients with ruptured spleen.

Symptoms	No. of Patients	Percentage
History of trauma	15	100.0
Upper abdomnal pain	14	93.3
Signs		
Raised pulse	15	100.0
Tender left hypochondrium	15	100.0
Palor	12	80.0
Low blood pressure	12	80.0
Left upper hypochondrial bruise	10	66.7
Shifting dullness	10	66.7
Abdominal distension	8	53.3
Positive paracentesis	8	53.3

Table V: Associated injuries in 15 patients with traumatic rupture of the spleen.

Injury	Frequency in 15 patients
Bruises	15
Cutwounds	9
Head injury	2
Fracture pelvis	1

DISCUSSION

Aetiology

Splenic rupture implies splenic injuries in which there is disruption of the organ parenchyma, capsule or blood supply.[6] The spleen is the commonest intra-adominal organ injured in blunt abdominal and left chest injuries[1,2] despite that it is seemingly well protected by the ribs.[1,2,6-8] The spleen can rupture beneath an intact thin capsule forming a subcapsular haematoma. Rupture through the parenchyma and capsule or avulsion at the pedicle can also occur.[9]

Other causes of splenic rupture include penetrating trans-abdominal or thoracic trauma; operative trauma and spontaneous rupture.

In this series of 15 patients, all were males and 12 patients (80%) were between 10 and 25 years (Table I). Parenchymal laceration and fragmentation were the main type of splenic injury noted.(Table II).

Blunt abdominal or lower thoracic trauma leads to splenic rupture by compression between the vertebra column or between the anterior abdominal wall and posterior ribs cage of the left side.[6] The spleen can also be dislodged from its peritoneal attachments by a big force and through counter-coup mechanism.[8]

Automobile accidents are a major cause of traumatic splenic rupture. Other causes include fall from a height, direct blow as in assault or sports.[9-11] A diseased enlarged spleen as is the case in holoendemic malaria, infectious monoculeosis, or polycythaemia vera is prone to rupture.[6,8] Enlargement places the spleen out of the protection from the rib cage making it more exposed to external forces on the anterior abdominal all. However this has not been supported by Abdulmunium[11] who noted that in rural Egypt despite a high frequncy of hepatosplenomegally due to malaria and schistosomiasis the incidence of ruptured spleen was not particularly high. This was also noted in West Africa.[9]

In this study all 15 patients had a splenic rupture as a result of blunt abdominal trauma, substained by kicks from assaults (six patients), and falls from height (5 patients) and motor traffic accident (4 patients= (Table III).

It is my finding that assaults contributed more to ruptured spleen. This may be explained by an increased

violence, assilants robbing their victims cash and any valuables as a result of economic hardship.

Penetrating injury as seen with gun shot wounds, and stab wounds through the spleen, cause rupture of the spleen and other intra-abdominal or thoracic cage organs. In this study, there was no patient with a penetrating injury. Penetrating trauma may become an important cause of splenic trauma with emerging wave of assaults and armed robberies in this country.

Operative trauma as a result of operations on an adjacent viscus such as the liver, stomach, pancreas and diaphragm can cause injury of an enlarged spleen.[2,6,10] Retraction or accidental ligamentous tear may also cause the injury. In USA 10% to 20% of all splenectomies performed are due to operative injury.[11] No operative trauma to the spleen was noted in this study despite high incidence of enlarged malarial spleen in this area.

Spontaneous or delayed rupture occurs in patients with no history of trauma. It is questionable whether a normal spleen ever ruptures spontaneously.[13] Delayed rupture presents days or weeks after a trivial trauma that is usually forgotten by the patient. Without a detailed history these may be interpreted as spontaneous cases of splenic rupture.

In these cases, after a trivial injury haemorrhage may be subcapsular or the omentum may seal and localise the

splenic injury preventing the bleeding and appearance of generalised symptoms. When this protection or subcapsular haematoma ruptures this lead to internal heamorrhage. Males are more prone to traumatic rupture of the spleen than females.[9,14]

Clinical Presentation

The clinical presentation of splenic rupture can be acute or delayed depending on the severity of the injury and the time taken to reach the hospital. The site of rupture determines the degree of haemorrhage. A tear of the vascular pedicle or the concave surface of the spleen is associated with profuse haemorrhage and shock.[14]

Acute rupture is seen in 80% to 90% of cases and results in uncontrolled bleeding manifesting as symptoms and signs of hypovolaemia.[15,16] Delayed rupture occurs in 10% to 20% of splenic injuries. It is delayed as a result of contained bleeding in subcapsular haematoma or omental sealing and localising of small ruptures. Subcapsular rupture and break of omental seal or clot leads to intraperitoneal bleeding with symptom of hypovolaemic shock.[9,12,14]

Most patients have a history of trauma. However, a negative history of trauma does not exclude the possibility of a ruptured spleen. Abdominal pain localised to the left hypogastriun is usually present, and my be referred to the left shoulder as a result of

haemoperitonium irritating the under surface of the diaphragm. Nausea, fainting and weakness may be present.

In this study all patients presented with a history of upper abdominal trauma and 14 complained of diffuse upper abdominal pain (Table IV).

Pallor, tachycardia, hypovolaemia and evidence of external violence on the anterior abdominal wall are the main signs.[17] Kehr's sign is reported to be present in 15% to 50% of the patients.[6] Abdominal distension, deep tenderness or guarding may be present localised in the left hypochondrium. Shifting dullness and positive paracentensis may be demonstrable.

Hypovolaemia rapid pulse and left hypochondrial tenderness were the main findings in the 15 patients in this study (Table IV).

Diagnosis of ruptured spleen in most cases is made from history and physical examination alone. In penetrating injury it may be obvious from the location of the wound. In some cases particularly in those with associated multiple injuries, however, the diagnosis cannot be made with certainly. Those with a small splenic injury and minimal haemorrhage are also difficult to diagnose. These minor degrees of rupture which pass undiagnosed are followed by formation of traumatic cysts or calcifications.[18]

Special investigations help in the diagnosis of unclear cases which are not acute, affording time for clinical observation and investigation. A four quadrant paracentesis with wide bore needle, may demonstrate haemoperitoneum. A negative tap however does not exclude splenic injury and the associated risk of the procedure are false negative results and injury to the intestine, limiting its application. It is also unhelpful in intrasplenic haematoma. Peritoneal lavage however is more useful particularly in unconcious patients and paraplegics.[19] It is regarded positive if there is recovery of fresh nonclotting or clotting blood in the dialysis catheter, or recovering grossly bloody fluid with lavage. In clear lavage fluids the microscopic presence of 100,000 red blood cells per milliter indicates the presence of intra-abdominal bleeding.

The serial determination of haematocrit, with a fall suggests haemorrhage. In traumatic rupture of the spleen there is unexplained leucocytosis.[6,7,8]

Angiography and radionuclide scanning are helpful inthe diagnosis of splenic rupture and can help to assess the magnitude of splenic injury. Major injuries are shown as splenic fragmentation or extravasation of the contrast media in the peritoneal cavity.

Minor injuries are shown as minimal parachymal disruption.

Thus classification into major and minor injuries may influence treatment. Minor injuries are treated conservatively and surgery is reserved for major ones.[13,20]

Plain abdominal and chest roentgenographs are helpful in that they may show a fractured rib highly suggestive of rupture of the spleen underneath an elevated left hemidiaphragm; enlarged splenic shadow with medially displaced gastric air buble and antral flatening. If there is gastric dilatation the greater curvature of the stomach mucosa is serrated due to dissection of blood into gastrosplenic ligament causing irritation. The colonic splenic flecture is displaced and an impression on the colon is made by the lower pole of the spleen in cases of splenic enlargement or subcapsular haematoma. In ruptured spleen it distends representing localised ileus in response to injury.[21]

In this study only paracentesis was done and it was positive in 8 patients, (Table IV) and negative in 7 patients. the diagnosis was made from clinical and physical examination in all 15 patients.

The above mentioned investigations are helpful in cases of doubt, and when the patients condition allows for delay, offering time for investigations. In this centre only roentegenography and paracentesis are done.

TREATMENT

The treatment of a ruptured spleen may be conservative or operative. The choice depends on experience and availability of facilities for patient monitoring. Splenic sparing procedures were prompted after it was noted that splenectomy increased susceptibility to infection in both children and adults.[3,4]

Conservative non-operative treatment is offered to selected patients under the criteria of stable vital signs and favourable general condition[9]

Centres adopting this approach have facilites for close patient monitoring like angiography and radionuclide scan. There is also need for a good blood bank service and emergency operation is done as soon as it is decided, when the conservative management fails.

Splenorrhaphy or partial splenectomy are methods of operative splenic conservation. Packing with the omentum or microfibrilar collagen of a small injured area to stop splenic bleeding conserves the spleen.

Some workers claim to treat 85% of cases with traumatic rupture of the spleen by splenorrhaphy.[22,23]

The other spleen sparing procedures include splenic arterial ligation to stop bleeding. The colaterals from the short gastric and left gastroepiploic arteris develop to nourish the spleen.[25] This procedure is however not recommended because it has been noted that

there is impaired pneumococcal clearance rate from the circulation by the preserved spleen.[26]

Partial splenectomy or splenectomy plus autotransplantation is practised[4,24] in other centres.[4,5,24]

Total splenectomy is a traditional treatment of traumatic rupture of the spleen adopted to eliminate the risk of post operative haemorrhage. The patient is resuscitated with fluids, and blood transfusion started after ligating the splenic artery.

Autotransfusion is preferred compared to bank blood because of the risk of HIV infection. Blood collected for autotransfusion is ideal only incases where there is no associated visceral injuries like the liver or bowel, and should not be haemolysed. No autotransfusion should be done with blood recovered after penetrating abdominal injuries.

Associated injuries to the patients with traumatic rupture of the spleen commonly noted in this study were bruises and cut wounds. (table V).

COMPLICATIONS

Haemorrhage from the splenic bed occurs in cases of large spleens with adhesions. Early splenic artery ligation, prevents bleeding. Reactionary haemorrhage is avoided by double transfixing the splenic artery and vein separately.

Pulmonary complications like atelectasis arise from varying degrees of bruising of the diaphragm during trauma or splenectomy. Breathing exercises and ambulation prevent this complication.

Subphremic abscess occur following infected haematoma in the splenic bed associated with gastric or splenic trauma. A drain occasionally acts as a source of infection.

Paralytic ileus and gastric dilatation results from peritoneal irritation by haemoperitonium. This is relieved by nasogastric aspiration post-operatively.

After splenectomy there is leucocystosis[27], and thrombocytopaenia.[27,28] Blood viscosity is increased predisposing to venous thrombosis. There is also an increase in circulation of abnormal red blood cells with Howell Joly bodies.[20]

Occasionally bone pains occur due to reactionary bone marrow hyperplasia as a result of bone marrow taking over the splenic function.[30]

Post splenectomy sepsis or overwhelming post splenectomy infection is characterised by septicaemia, meningitis, or pneumonia, occurring days to years after splenectomy.[3,23,31]

Post splenectomy sepsis is more common in children.[31] In adults the rate is between 1% and 2% and it is higher in children, fatal complications occurring any time after splenectomy.[24]

In the first six months post splenectomy sepsis is reported in only 20% of cases, 30% being reported within a year and 60% in 2 years.[30]

The immune alteration after splenectomy leads to an overwhelming infection, because of loss of splenic functions; like clearance of particulate matter, elaboration of specific immune response by opsonins and tuffsin. Encapsulated bacteria, pneumococcus being the commonest.[31] Haemophilus, Neisseria, Pseudomonas, fungi and viruses are also implicated.

In the treatment of post splenectomy sepsis, early cultures of body fluids and approapriate antibiotics is mandatory. Prevention is achieved by close follow up of the patient for the first two years, so that any infection is diagnosed and treated early. Polyvalent pneumoccocal vaccine and Haemophilus vaccines with long acting penicillin (Penadur) prophylaxis may be given for at least two years. The ideal way to avoid post splenectomy sepsis is by splenic preserving procedures in cases of ruptured spleen.

In the patients presented, 10 were followed up for one year and none was noted to have any complication. The other five were lost for follow up, and their fate is therefore unknown.

CONCLUSION

Traumatic rupture of the spleen is a surgical emergency whose diagnosis depends on history and clinical acumen. Splenectomy is the treatment of choice in our centre and close follow up of these patients with prophylactic antibiotics in children may prevent post splenectomy sepsis.

In the increase of AIDS incidence autrotransfusion is preferred compared to bank blood.

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PANCREATIC PSEUDOCYST

SUMMARY

Eight patients with pancreatic pseudocysts were managed in the surgical department of Muhimbili Medical Centre during the period covering July 1987 to June 1989. There were five males and three females patients with peak age of 30 and 40 years. Alcohol and trauma were the main associated aetiological factors in most patients.

They presented with painful upper abdominal swelling which was tender on palpation. Ultrasound and barium meal studies established diagnosis in all cases.

Internal drainage was the mode of treatment in all cases. None of the cysts was associated with complication and the post operative course was uneventiful.

INTRODUCTION

Pseudocyst is the most common cystic lesion of the pancreas accounting for 75% to 80% of cysts in the gland.[1] Pseudocyst is a sequalae of an inflammatory process within the pancreas. Common causes of pancreatitis included trauma, alcohol and billiary tract diseases.[1,2] Pseudocysts have been recognised more frequently in Europe in the last decade probably due to increased incidence of alcohol intake and the advent of widespread use of ultrasound and computerised tomography.[3,4]

The incidence of pseudocyst in developed world is between 1% and 4.5% of all patients with pancreatitis regardless of the cause.[2] There is high association of pancreatic pseudocyst with alcohol, with figures ranging from 60 to 90% [2,5,6]. Males are more commonly effected with peak age in the fourth and fifth decades. 26% of all pancreatic pseudocysts are complicated with obstruction, rupture, infection, or haemorrhage.[7] The last three are fatal if not diagnosed early and managed properly.[2,8-11] Trauma is frequent in our environment, and this study looks at its association with pseudocyst. The clinical presentation, pathogenesis, complications, diagnosis and management of pancreatic pseudocyst is presented.

CASE PRESENTATION

Mr. W.M. 38 years old was admitted with a painful progressive abdominal swelling of four months duration. This was treated in peripheral hospital with analgesics without relief. Six months, before admission he was involved in road traffic accident. As he was crossing the road he was knocked down from anterior by a fast moving vehicle. He was treated for minor bruises on the abdomen and discharged. A month after the accident he started to experience a painful upper abdominal swelling, that was gradually increasing in size.

There was no history of jaundice, vomiting blood, or haematuria. He was nauseated and felt a sense of fullness that prevented him from eating well. He denied a history of cough, difficulty in breathing, but admitted to slight weight loss. On examination he was found to be moderately wasted, not pale, afebrile and not jaundiced. His blood pressure was 130/80mmHg, and pulse rate of 80/min regular and full volume. The abdomen was moving with respiration, with no visible distended veins, and slight fullness on the left hypochondrial area was noted. On palpation a firm smooth oval slightly mobile, non pulsatile intraperitoneal mass was confirmed. The mass was extending to the epigastrium and measured about five centimeter below the left costal margin.

Cardiovascular and respiratory systems were normal. A provisional diagnosis of pancreatic pseudocyst was reached.

Investigation revealed he had haemoglobin of 13g/dl, white blood cells (WBC) total count was 10,000mm^3 and differential count was normal.

. Serum creatinine was 100/umol/1

. Serum electrolytes Na^+150 Mmol/1

K^+ 3.0 Mmol/1

Cl^- 108 Mmol/1

. Serum protein 7g/dl

. Fasting blood sugar 3 Mmol/1

. Alkaline phosphatase 90 iu/1

. Serum bilirubin 15 umol/1

. Urinalysis and stool were normal

. Serum amylase there were no reagents

. Prothrombin time: patient 13 seconds and control 12 seconds

. Chest X-ray was normal

. Barium meal. Anteriorly displaced stomach with indentation of the greater curvature of the stomach by an extragastric mass on a lateral film. The abdominal ultrasound showed no stones in gallbladder, normal common bile duct, normal splenic and portal veins, and pancreatic cyst was visualised.

With the diagnosis of pancreatic pseudocyst confirmed the patient was planned for surgery. This was explained to him and he gave his consent.

At operation, the abdomen was opened through a left paramedian incision. A thick walled cystic swelling was found in the lesser sac measuring about 10cm in diameter adherent to the posterior wall of the stomach and arising from the body of pancreas. The viscera were found to be normal. A decision was therefore made to perform a cystogastrostomy.

An anterior wall gastrotomy directly opposite the point chosen for entering cyst was done. The posterior wall of the stomach was opened through a four centimeter longitudinal incision after aspirating fluid for

culture and sensitivity and ascertaining safety of entering the cyst. The locules in the cyst were broken after suction of 250mls. of colourless cystic fluid. Part of the pseudocyst wall was taken for histology. Haemostasis at the incision edge was achieved by continuous suture with dexon 2/0. The gastrotomy was closed in two layers using 2/0 chromic catgut seromuscular layer and silk 2/0 second layer. After saline lavage the abdomen was closed in single layer using prolene 'O' suture. Skin was closed with silk 3/0.

Post operatively the patient was on I.V. 5% Dextrose 2 litres alternating with Normal saline 1½ litre for 24 hours, injection pethidine 100mg 8 hourly for 24h, injection i.m gentamycin 80mg twice daily for 5 days; nasogastric tube aspiration every two hours, input output chart every 4 hours. Monitor of blood pressure, pulse rate, temperature, every half hours for the first 4 hours and two hourly in the next 24 hours. On the first post operative day the patient had maintained normal vital signs and had no complaint. He was maintained on intravenous fluids and to continue with nasogastric decompression for next 24 hours.

On this third post operative day he was afebrile, had passed flatus and was well hydrated, the dressing were dry and nasogastric aspirate was very minimal. He was

allowed oral fluids, intravenous fluids and nasogastric tube were removed.

The cystic fluid culture and sensitivity results revealed sterile culture. Gentamycin was stopped. On the eighth day all stitches were removed and the patient discharged with a date to attend surgical outpatient clinic. Two weeks later he was seen at the clinic, he had no complaint and appeared brighter, the surgical scar had healed and no mass was palpable. The cyst wall biopsy revealed chronic non specific inflammation with marked fibrosis.

RESULTS

Results of the patients studied is presented. The age, associated factors, presenting symptoms and signs are tabulated.

TABLE I: Age distribution in eight patients with pancreatic pseudocyst.

Age in years	No. of patients	%age
30	1	12.5
31 - 34	2	25.0
35 - 39	3	37.5
40 - 44	1	12.5
45 - 49	1	12.5
TOTAL	8	100.0

TABLE II: Aetiological factors in pancreatic pseudocyst N = 8

Aetiology	Frequency	% age
Alcohol with trauma	3	37.5
Alcohol	2	25.0
Trauma	2	25.0
Billiary tract disease	1	12.5

TABLE III: Presenting symptoms and signs in eight patients with pseudocyst.

Symptoms	Frequency	% age
Upper abdominal pain	8	100.0
Abdominal swelling	6	75.0
Weight loss	1	12.5
Nausea	1	12.5
Vomiting	1	12.5
Signs		
Left hypochondrial Mass	8	100.0
Left hypochondrial tenderness	8	100.0
Ascitis	-	-
Jaundice	-	-

In all 8 patients ultrasound and barium meal were diagnostic, and all were treated by cystogastrostomy.

DISCUSSION

Aetiology

Pancreatic pseudocyst is a collection of inflammatory debris sometimes bloody and fluid rich in pancreatic enzymes in a cavity without epithelial lining.[1,2,11,12] Pancreatitis secondary to either alcohol, billiary tract disease, trauma or any other cause are the main aetiological factors.[1,3,5,6,11,13]

The pseudocyst appears one to two weeks after acute pancreatitis, but is more often seen two weeks to three months.

The incidence of acute or a chronic pancreatitis which is associated with alcohol varies greatly in different parts of the world. It accounts for 25% in Scotland and 50% to 90% in France and USA respectively.

Mechanism of cystic formation is uncertain but postulates are: **Alcohol** causes pancreatic hypersecretion and spasms of the sphincture of Oddi, together with duodenitis both of which block the flow of pancreatic

juice leading to pancreatitis. The alternative theory is that there is reflux of duodenal contents into the pancreatic duct as a result of increased intraduodenal pressure from prolonged vomiting in chronic alcoholism. While malnutrition in alcoholism leads to easy susceptibility to inflammation.[10,15]

Billiary tract disease

The incidence of cholelithiasis in patient with pseudocysts varies from country to country, but in UK it is present in 50% of patients who develop pancreatitis.[15]

The majority of patients have a short common bile duct and pancreatic duct opening where a stone impaction at the lower end of bile duct may oclude the pancreatic drainage or promote reflux of bile into pancreatic duct system. In 5% to 10% of pancreatitis, gallstones are found in the common bile duct and in 80% cases of pancreatitis stones, ranging from 1 to 12mm are recovered from stools a few days following onset of pancreatitis. The attacks of pancreatitis decreases when the billiary tract disease is eradicatted.[15-16]

Trauma. Post traumatic cases of pseudocyst are associated with blunt or penetrating abdominal injuries to the pancreatic tissue and duct. Pancreatic secretions are activated leading to pancreatitis.

Acute pancreatitis or trauma preceeds clinical discovering of pancreatic pseudocysts in 9% to 10% of patients.[3,11,16,17] 75% to 80% of pseudocysts

complicate acute pancreatitis and 25% pancreatic trauma.[2,13,15,18] In this study of 8 patients alcohol was the main aetiological factor in 5 patients, and trauma in five patients (Table II).

Pseudocysts in children are similar to those occuring in adults except in them, 60% are largely due to trauma. Mumps and cocksakie virus infections, kwashiokor, steroid therapy and L-asparagnase used in treatment of acute leukemia have led to development of pseudocysts in children.[11]

Pathogenesis:

In the formation of pseudocyst three elements are necessary.[2,10,12,14]

i) Ductal rupture communicating with patent space;

ii) An actively secreting pancreas, and

iii) Interfearance with normal ductal drainage i.e blockage of duct.

The exocrine pancreas secretes the enzymes in a proform except for amylase, and lipase which are secreted in active from.[15] The activation of these enzymes within the substance of the pancreas leads to autodigestion and tissue distruction, including pancreatic acinar cells. Ductal rupture caused by necrosis in the parenchyma leads to seepage of pancreatic fluid in the cavity evolking an inflammatory response, so that the body reaction in an effort to seal off the cause of irritation creates a cystic mass of shaggy fibrinous

exudate on the surrounding surfaces which is converted in one to several weeks into a tough fibrinous capsule which may be softened in places by fat necrosis and inflammatory reaction. If situated in the lesser sac the wall is thus formed by fibrotic parietal and visceral peritonium together with the wall of adjacent organs.

There is no secretory endothelium hence the name pseudocyst. The cystic fluid is thus a result of leakage of pancreatic juice from a damaged duct and an exudate due to inflammation.

The cyst contains necrotic material, fluid rich in pancreatic enzymes with amylase 50 to 100 times more than serum levels. The necrotic material is produced as a result of digestive action of pancreatic enzymes and surrounding tissues.[4,12,14]

Actively secreting pancreas, is essential for the continuous cyst growth as result of secretion pressure of the ducts, which is about 30mm of water after a meal. The osmotically active cystic fluid further accounts for the cystic growth. These two factors contribute to different sizes of the cysts ranging from few millimeters to several centimeters in diameter.

Ductal occlusion appears to be a major factor in the pathogenesis of pseudocyst. It has been noted that pseudocysts disappear in three to six months after blocked ductal system is unblocked. This continued

ductal blockage provides the pancreatic secretion into the cyst enhancing inflammatory process hence existance of pseudocyst.[12,13,]

Pseudocyst develops from or in the head, body or tail of pancreas in ratio of 1:2:1, respectively.[6]

The cyst contains blood, pancreatic enzymes, tissue fluid and debris from pancreas which may develop after a period of days or weeks after onset of pancreatitis, and communicate in the necrotic portions of pancreas in lesser sac.[10,18] Usually the pseudocyst spreads in the region of least resistance leading to unusual presentations in the abdomen or intrathorax.[8,18] Commonly it occurs in the lesser sac.[12,14,18]

The majority of pseudocysts are single, however 25% present as multiple cysts.[11,15] In the course of evolution 10% to 40% of pseudocysts resolve spontaneously.[7,11,19,-23] of the persisting pseudocysts 26% to 40% are complicated preoperatively.[5,7,10,22]

Complications

Complications of pseudocyst occurs twice as often as spontaneous resolution.[24] Complications include bleeding, rupture, pressure effects, and infection.[2,8,9,10].

Bleeding is a serious complication with mortality rate approaching 60%.[10,14,18] Mechanism through which bleeding is brought about are probably due to derranged clotting mechanism, erosion of vessels or via

lysolecithin and enterokinase action. Clotting mechanism is deranged in significant number of patients as a result of hypoprothrombinaemia secondary to prolonged starvation after vomiting or prior hepatic injury associated with, alcoholicm pancreatitis, and pseudocyst.[14] There is multiple bleeding sites, in the wall of the cyst accounting for 5% of bleeding cases. Erosion of the major artery by autodigestion process leads to arterial bleeding into the cyst forming a pseudoaneurysm or bleeding outside the cyst. Erosion occurs more commonly in presence of sepsis as in primary pancreatic abscess or secondarily infected pseudocyst.[10] There may be concomitant bleeding of duodenal or gastric ulcer due to irritation process by cyst wall on adjacent visceral wall.[10,14]

Lysolecithin and enterokinase play part in causing bleeding from the stomal margins after internal drainage. The conversion of lecithin to lysolecithin within the cyst and in emptying into the stomach leads to digestion and inflammation of gastric mucosa.[25] Enterokinase enters the pseudocyst leading to continuous activation of pacreatic enzymes within the cyst structure.[10] This presents as upper gastronestinal bleeding.

Sudden increase in size of the pseudocyst, pulsation and development of bruit over the mass associated with

blood pressure fall and high pulse, indicates bleeding.[10,14,18]

Repture of pseudocyst is uncommon. If it ruptures into the peritoneal cavity it is associated with 50% mortality,[5,10,14] but if it ruptures into the gastrointestinal tract it may not require surgical intervention for the fistula that is formed leads to therapeutic internal drainage.[10] It can rupture into the stomach, duodenum, colon or jejunum. The mechanism of rupture of pseudocysts is not known. Increased cystic pressure or diminished wall thickness appears not to be the only mechanisms.[26]

Sudden rupture in the peritonium is heralded by shock, with death being inevitable unless the pancreatic secretions are drained. The small leakage of pseudocyst leads to ascitis. 13% to 16% of patients present with ascitis as a result of slow accumulation of fluid in the peritoneal cavity.[10] This condition is difficult to diagnose for it occurs in middle aged alcoholic in whom ascitis from hepatic disease is expected.

Increased ascitic amylase 346 to 9700 somogyi units and protein 2.5 to 6g/l differentiates ascitic fluid of pseudocyst origin from other causes of ascitis. At surgery the leak may be difficult to find.

Pressure effects: Local expression of pseudocyst leads to pain which is present in 80% to 90% of patients.

The pain is persistent and may occur even with small pseudocysts demonstrable only by ultrasound and not clinically palpable.[5,10]

The effect of space occupying lesion of the pseudocyst involves other organs. Pseudocyst is palpable in 50% of cases[5] and displaces the upper gastrointestinal tract as demonstrated on barium meal. This may present as a sense of fullness causing limited meal capacity if it stretches the stomach. There may be gastric outlet obstruction if the cyst arises from the head of the pancreas. The common bile duct if also occluded will present with jaundice and elevated alkaline phosphatase. Portal vein and splenic compression tends to cause stasis and thrombosis. This can lead to portal hypertension.[5,27]

Infection: The pseudocyst may be converted into an abscess spontaneously. Cystocolic fistula formation with subsequent invasion by coliform bacteria, or iatrogenically following aspiration may also occur. The abscess is seen two to six weeks after an attack of pancreatitis. An abscess presents with high fever, leukocytosis, and multiple blood culture identify the offending organism. In 50% to 60% of cases the organisms being coliform from the gastrointestinal tract.[6,10,14]

Diagnosis: Patients present complaining of epigastric pain associated with nausea and vomiting secondary to

paralytic ileus and mechanical obstruction caused by the cyst.[18] Depending on pressure effects to the common bile duct or duodenum the patient may be jaundiced and have gastric outlet obstruction. (Table III).

The history will establish if the mass was preceeded by the known aetiological factor of pseudocyst, like acute pancreatitis, upper abdominal trauma, cholelithiasis, or if the patient is a known alcoholic. In some cases the development of pseudocyst may be incidous without recognisable predisposing factor.

The affected age group is between 40 to 50 years, with prepondrance of males being three times more affected.[2,10,14] Table I).

Abdominal examination will reveal left hypochondrial fullness with palpable slightly tender firm mass which may be mobile. Ascitis may be present if there is slow leak from the cyst. 50% of the patients present with mass, anaemia, and carbohydrate intolerance may be present.[10]

Serum amylase and urine amylase are elevated in 71% and 90% respectively.[2,10,14] The amylase crtatinine clearance ration is always above normal of between 1% to 4%.[15] Alkaline phosphatase is increased if there is common bile duct obstruction. There may be anaemia due to folic acid deficiency or persistent bleeding from

upper gastrointestinal tract or as a result of chronic disease.

The raised serum amylase may be high in other conditions like, intestinal obstruction, perforated duodenal ulcer, pyelonephritis, salivary gland disease and ruptured ectopic pregnancy.[14]

However urine amylase is more reliable than serum amylase. The cause of raised urine amylase in pancreatitis is unknown but is is thought to occur as a result of decreased tubular reabsorption of amylase in pancreatitis.[10]

Indirect radiological studies which include barium meal, and chest X-ray provide a clue to the underlying effect of the pseudocyst. Chest X-ray may show elevated hemidiaphragm and pleural effusion. 10% of patients have suggestive radiological findings on chest x-ray.[10] Abdominal x-ray may show evidence of chronic pancreatitis like pancreatic calcifications which occur in 31% of patients.[10,14] And obliteration of iliopsoas shadow or homogenous mass in the epigastrium, or displacement of renal shadow may be present. Visualisation of soap bubble sign is pathognomic of retroperitoneal abscess. Barium meal demonstrates a mass in 60% of cases, as deviation of portions of stomach, duodenum and other parts of bowel, or retained gastric secrections with thick oedematous ruggae of stomach or duodenum.[10,14] Cyst in the head of pancreas

widens the duodenal loop, displaces the antrum upwards and flattens mucosa. That in the body displaces the stomach anteriorly and upwards while those in the tail displaces the stomach medially and also the proximal jejunum and splenic flexture.

Oral chroloangiogram and ultrasound are essential to demonstrate goal bledder caliculi. Intravenous urography helps to differentiate the abdominal mass from a kidney lession and to see if the kidney has been displaced.

Direct radiological investigations like Ultrasound is able to demonstrate the fluid in cyst, and pseudocyst diagnosis is simpler. It is diagnostic in 85% to 90% of patients. False positive results are reported in 5% to 10% of cases.[12,13,14] Ultrasound can give the thickness of cystic wall; which is important in the timing of surgery.

Endoscopic retrograde cholongio-pancreatography (ERCP) is 83% diagnostic.[13] It is useful for demarcation of multiple cysts, presence of fistula to mediastenum or retrocolic space. ERCP however is dangerous for it causes further pancreatitis. If the contrast enters in the cyst, surgery should be performed within 24 hours without delay because of risk of infecting the cyst.

Angiography is diagnostic in that it shows stretching of vessels indicating presence of cyst.

Computerised Axial Tomography has a diagnostic accuracy of 92% to 98%

In this study of 8 patients ultrasound and upper gastrointestinal tract series were diagnostic in all cases. Angiography, and ERCP are not available in our centre.

TREATMENT

About 10% to 40% of pseudocysts resolves spontaneously needing no treatment at all[7,11,14,20,21] and this occurs in the first two to three weeks from the onset of pacreatitis or upper abdominal trauma.[21,22] Patients seen early during onset of disease should be observed and treated medically with close serial ultrasound observation of size of cyst, and amylase creatinine ratio. Prolonged observation increases risk of complication. In one series, 13.5 weeks was man duration from formation of cyst to emergency of complication.[10] The conservative approach should be abandoned in case of cyst complication like bleeding, rupture or obstruction.[10,14,23] There is 60% mortality if definitive surgery is performed before six weeks as compared to 6% after six weeks.[14]

Complications occur more within three weeks or less after pseudocyst formation when the cyst wall is less than five millimeters in thickness. Chronic cysts are those with duration of more than six weeks. Their wall is usually more than five millimeters thick, and have