CLINICAL STUDY AND MANAGEMENT OF

PERFORATED DUODENAL ULCER

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MASTER OF SURGERY (BRANCH I)

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CERTIFICATE

This is to certify that the dissertation entitled **"CLINICAL STUDY AND MANAGEMENT OF PERFORATED DUODENAL ULCER "** submitted by **Dr.SATHISH** to the faculty of surgery, The Tamil nadu Dr. M.G.R. Medical university, Chennai in partial fulfillment of the requirement for the award of **M.S. Degree in GENERAL SURGERY** is a bonafide work carried out by him during the period of September 2007 – November 2009 under my direct supervision and guidance.

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DECLARATION

I, Dr.SATHISH solemnly declare that the dissertation titled **"CLINICAL STUDY AND MANAGEMENT OF PERFORATED DUODENAL ULCER"** has been prepared by me.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of Master of Surgery, **(Branch I) General Surgery** Degree Examination to be held in March 2010.

Place : Madurai

Date :

Dr.SATHISH

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CONTENTS

INTRODUCTION

Duodenal ulcer is a very common disease in southern part of India. In the earlier years the main treatment of duodenal ulcer was primarily surgical, but now it's out of vogue due to advent of effective medical line of treatment. However the important complication of duodenal ulcer which is perforation is still seen commonly and surgical intervention is considered the treatment of choice.

Perforation of duodenal ulcer is one of the most serious and, life threatening complication of peptic ulcer. Perforation of duodenal ulcer leads to contamination of peritoneal cavity with duodenal and gastric secretions which may initiate catastrophic cascade of events which until stopped in its track can be detrimental to the life of the patient. Perforation can occur in a patient with past history of peptic ulcer but it is known occur in about 20% of patient without, any relevant preceding symptoms. It is estimated that roughly 1.3% of population above the age of 20 years have had some degree of peptic ulcer activity during any annual period. Duodenal perforation is one of the dreaded complications that is known to occur in 15% of all recognized duodenial ulcer cases. Thus perforation of duodenal ulcer has become a pestilence that threatens to cripple the economic and social life of significant section of society if un attended to.

Meticulous history taking regarding symptoms of patient and accurate clinical examination of the patient with assistance from radiological investigation should be the aim to arrived at diagnosis of duodenal ulcer. Without second thought surgical intervention is treatment of choice.

Conservative treatment is not suitable for routine use as incidence of morbidity and mortality is very high. And also if the condition is not diagnosed

properly and adequately. It progresses in a definitive manner leading to death due to peritonitis. Mortality increases with delay in operating and the mortality rate almost approaches zero if operated within 6 hrs., from 6-12 hrs the rate is 5-10% and 12-24hrs is a 23% or higher and in the course of 3 day or after the surgery is seldom successful. Hence, It is all the more important that the condition be diagnosed as early as possible since early institution of treatment will definitely decrease the mortality. Here the role of either general practitioners in identifying the disease and immediate referral to a major surgical center is very important.

Lord Moynihan has stated as follows, “Perforation of duodenal or gastric ulcer is one of the most serious and most overwhelming catastrophes that can befall a human being”.

AIMS AND OBJECTIVES

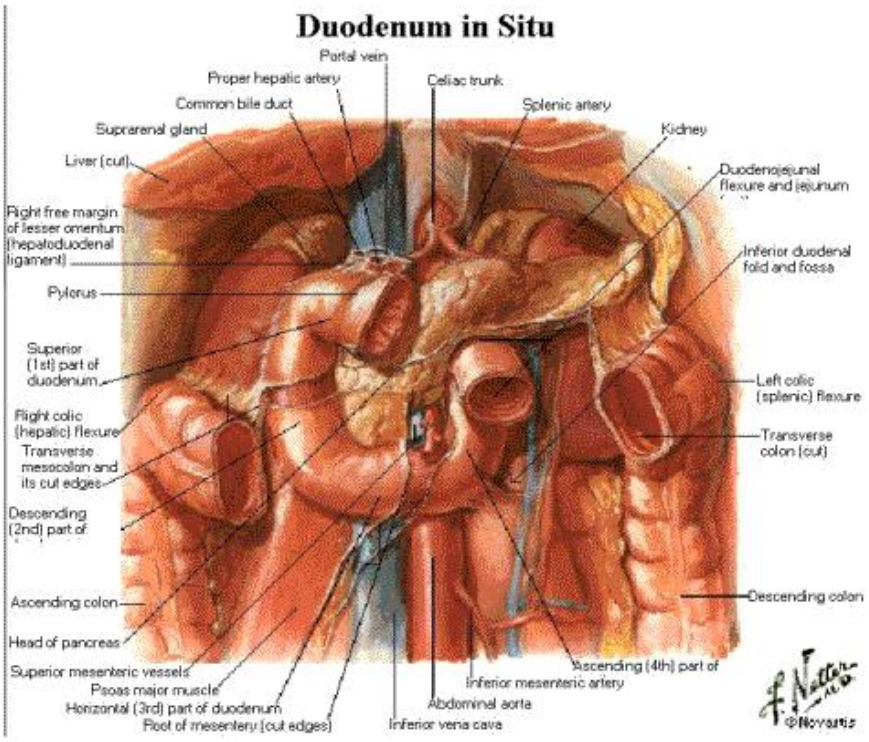
1. To study the clinical aspects of perforated duodenal ulcer.

2. To study the results of different modes of treatment.

3. To compare mortality and morbidity of simple closure of perforation.

SURGICAL ANATOMY OF DUODENUM

The duodenum is the first, shortest, widest and most fixed part of small intestine, it measures 10 inches in the length. It has no mesentery and is partially covered with peritoneum.



It extends form the pylorus to the duodenojejunal flexure forming a “C” shaped curve where concavity extends towards the left and upwards. The “C” shaped curve is occupied by the head of pancreas. Since it forms a “C” shaped curve the beginning and the ends of the duodenum are close together. It lies on the posterior wall of the abdomen above level of umbilicus and almost wholly in the right half (of the abdomen).

The duodenum, which begins at the pylorus, passes backwards, upward and to the right for 2.5 cm under cover of the posterior part of the quadrate lobe of the

liver, to the neck of gall bladder varying slightly in directions, according to the degree of distension of the stomach. It then makes a sharp curve known as superior duodenal flexure and descends for about 7.5 cm in the front of the medical part of right kidney, generally to level of the 31 lumbar vertebra, lying immediately medial to the lateral plane. Here it makes a second bend known as inferior duodenal flexure and passes almost horizontally from right to the left across the vertebral column just above the level of the umbilicus, having a slight inclination upwards, it then ascends in front and to the left of the abdominal aorta for about 2.5 cm and ends opposite the body of the 2 lumbar vertebrae in the jejunum. At its union with jejunum it turns abruptly forwards, forming the duodenum-jejunal flexure, which is, situated 2.5 cms to the left of the median plane and below the transploric plane. The principal changes of directions of "C" shaped curve arc made use of to divide the duodenum into 4 parts for convenience of description.

I Part - The Superior part

It is 5 cms long and is the mobile part of the duodenum. It begins at the pylorus in the transpyloric plane about an inch to the right of the median plane and passes backwards and upwards in close relation with the live and ends at the neck of the gall bladder where it bend sharply to become the second part.

Second Part- Descending part

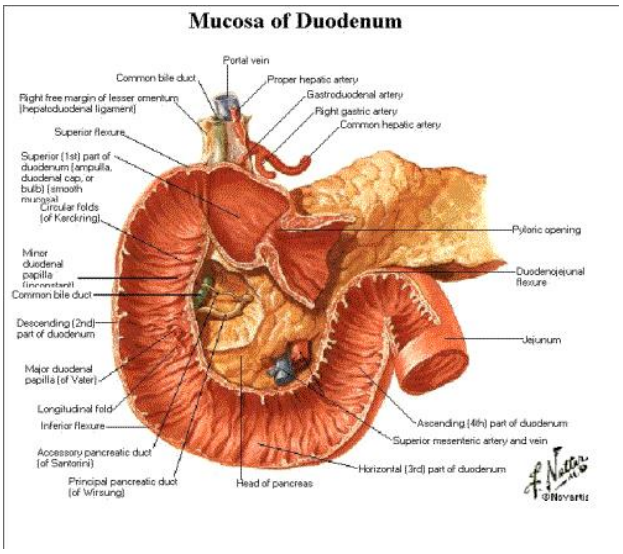
It is about 8 to 10 cms long descends from the neck of the gall bladder along the right side of vertebral column as low as lower border of the body of third lumbar vertebra. It is crossed by transverse colon. The parts above and below in the transverse colon are covered in front by peritoneum.

Third part-Horizontal Part

It is about 10 cms long; begins at the right psoas major muscle at the level of the lower border of third lumbar vertebrae and passes horizontally towards the left across the inferior venacava and aorta and then bends upwards to become the fourth part.

Fourth part-ascending part

It is about 2.5 cms and is the shortest part of the duodenum. It curves upwards, along the left side of the aorta and head of the pancreas on the left psoas muscle, It ends about 2.5 cms to left of the midline from the level of the second lumbar vertebra by bending sharply forward to form duodeno-jejunal flexure where it is continuous with jejunum.



Blood supply

The blood supply of duodenum is derived from the right gastric artery; supraduodenal; right gastroepiploic, superior and inferior pancreaticoduodenal arteries. The superior part of the duodenum receives a leash of small branches from the Hepatic artery proper and a similar leash of vessels from gastroduodenal artery.

Surgical importance of Blood supply

The surgical importance with regard to internal supply is that the area of mucous coat supplied by the duodenal branch of hepatic artery is especially liable to the formation of duodenal ulcer". This is thought to be due to the fact that artery has poor anastomosis is with its neighbouring arteries. The veins end in splenic; superior mesenteric and portal veins, which shows that the area has a poor vascular supply compared to that of rest of duodenum and hence has increased chances of perforation.

In the living the pre-pyloric veins of Mayo are helpful as landmarks in distinguishing pyloric canal form the 1 part of duodenum.

Longitudinal folds

The circular folds begin about an inch from pylorus. The lower half of the second part contains a longitudinal fold near its posteromedial border. If this longitudinal fold is traced upwards it leads to a small round eminence called "Duodenal Papilla". On the summit of the papilla there is a small opening which is the opening CBD and pancreatic ducts.

Nerve Supply

The sympathetic from T9, T10 and the parasympathetic from vagus nerve pass through coeiliac and superior measentric plexus and supplies the arteries.

Lymphatic drainage

The lymph vessels end in the nodes that lie between the duodenum and the head of the pancreas form where lymph is carried to the coeiliac and superior measentric nodes.

PATHOLOGY

Perforation is due to sudden sloughing of an unsupported portion of the floor of an ulcer secondary to slow process of devascularization. The devascularization is most often progressive. It is easier to explain a blow out of the vascular floor of chronic ulcer than to account for the sudden perforation of all components of the visceral wall in acute ulcer.

Acute perforation may occur in acute or chronic duodenal ulcers. More than 95% of cases of ulcer perforation is of chronic variety. In acute perforation an embolic phenomenon resulting in the formation of a disc shaped infarct has been suggested. Loacalized vasospasm has also been suggested as a cause, Whatever may render the area vulnerable; auto digestion may by the final factor to decide for perforation to occur, Free acid is usually present before perforation occurs.

In an acute presentation sudden rupture of the base of the duodenal ulcer takes place with result that the contents of duodenum escape free into general peritoneal cavity. In certain instances the perforation might become sealed off and thus the spillage may be circumscribed. Duodenal ulcer which perforates into the general peritoneal cavity are situated in the anterior or antero superior walls of the duodenum, most commonly on the antero superior surface of 1st part of the duodenum within an inch from the pylorus. Peptic ulcer perforation is rare in 2nd and third part of the duodenum. But the possibility of a retroperitoneal perforation in the 2nd part of the duodenum has also been recognized. Perforations on the anterior, surface of the duodenum leads to wide spread, peritoneal soiling. Localized adherence of the parietal peritoneum or omentum or other viscera is unusual with anterior ulcers.

Posterior- ulcers may cause free leakage into the lesser sac through foramen of Winslow. But more often such ulcers pnetrate the neighbouring structures such as pancreas or liver to which the are adhered by local peritoneal reaction.

An encircling ulcer perforates into the pancreas in it's posterior and may also perforate in it's anterior part. Two duodenal ulcers; one anterior, which has perforated, and a posterior penetrating are commonly found in operation when haemorrhage coexists with perforation and it is the posterior one, which bleeds.

Rarely a posterior ulcer may perforate extra peritoneally and the extravsated fluids may collect in the region around the kidney. If the fluid cracks downward still further, a mass may appear in the right iliac fossa simulating appendicular mass. The drainage of such an abscess may result in duodenal fistula.

The size of the perforation varies from 3 mm to 10 mm in diameter. Perforation of more then 2.5 cm have been reported. In majority of cases the site of perforation is obvious during operation. The veins of Mayo always helps to know whether it is a DU perforation of GU perforation. In some cases the anatomy of the pylorus and of the duodenal bulb may be disturbed. It may be impossible to identify the veins of Mayo which marks the dividing line between duodenum and stomach; and the region be so obscured by oedema and adhesions that it may be difficult to make sure whether the ulcer is prepyloric or duodenal.

At operation or at autopsy, in case of perforated DU; the site of perforation was found to be on the anterior wall of the duodenum in 92% and on the posterior wall in 2% and on or about the pyloroduodenal junction (classified DU) in 6% of cases. In most instances, the second perforation may be overlooked at operation immediately after the perforation has occurred, chemical peritonitis develops as a

result of irritating action of the contents of the stomach and duodenum; the irritants being acid, biliary secretions and pancreatic secretions. It is difficult to determine how long it takes for chemical peritonitis to develop into frank bacterial peritonitis. Theoretically it should depend on the following factors.

1. Size of the perforation
2. Magnitude of spillage
3. Reaction and composition of duodenal contents
4. General condition of the patient
5. Resistance of the patient to infection

Neutral or alkaline contents delay the onset of septic peritonitis for a few hours. When the escaped contents are strongly acidic and actively bactericidal, the infection is considerable inhibited. The bacteria are derived from many sources. The flora of the mouth and nasopharynx may be swallowed. The contents of the stomach and duodenum may be heavily infected in conditions of chronic obstruction or large ulcers favour the retention of many organisms, Ingested food and drink have again a considerable bacterial content. Finally the infected small intestinal contents my regurgitate into the site of perforation. Then the paralytic ileus with peritonitis becomes a marked feature. Organism therefore vary in type and may include streptococci, staphyococci, coliforms, candida species and neumococci.

Rarely an unusual specific organism streptothrix may cause peritoneal infection. Infection is unusual with gas producing organisms. In an average case it may be assumed that during the first 6 hrs the peritonitis is less infective. When malignant ulcer perforates, even after 8-12 hrs peritoneal fluid will be infective

in character. There is an immediate reaction to the sudden and gross soiling of peritoneum after rupture. Hyperaemia, oedema, exudation and fibrin deposits with pus formation occur in varying degree according to the chemical nature of the fluid; and type and virulence and the number of organisms. Davidson et al in 1930 studied 34 cases and found that 75% positive culture in the 2d six hours after perforation. Paralytic ileus and intestinal obstruction supervene it, bacterial peritonitis becomes established and then the clinical picture will be similar to that in the peritonitis from other cause.

Heliobacter Pylori

Over the last 20 years this organism has proved to be of overwhelming importance in the etiology of a number of common gastroduodenal diseases such as chronic gastritis, peptic ulceration and gastric cancer. The organism had unquestionably been observed by a number of workers since Bachet's first description in 1874. But it was not until 190, that Warren and Marshall injested the organism to confirm that Koch's postulate could he fulfilled with respect to gastrics that they succeeded in causing themselves. Eradication therapy was then employed with mixed success.

Organism is spiral shaped and is fastidious in its requirements. One of the characteristics of the organism is its ability to hydrolyse urea resulting in production of ammonia a strong alkali effect of ammonia on ANTRAL G cells is to cause the release of gastrin via the previously described negative feed hack loop.

The urease activity is very specific to detect of organism.

Widely used tests are 13C & C breath tests and CLO test commercially available urease test kit) which is performed on gastric biopsy.

Infection with H. pylori may be the most common human infection. The incidence of infection within a population increases with age and in some populations infection rates are 80 to 90% are not unusual. The infection is acquired in childhood and the possibility of infection is inversely related to socio-economic group. The means of spread has not been identified by it can occur in faeces and feaco oral route spread seems most likely. Known since 1984, that H. Pylori infection is amenable to treatment with antibiotics the profound hypochlorhydria produced by PPI combined with antibiotics is also effective in eradicating the organism. Commonly used eradication regimens include a PPI and two antibiotics such as Metronidazole and Amoxycillin. Higher eradication rates in the region of 90% can be achieved. Re-infection following successful eradication appears rare but incomplete eradication is more important clinical problem.

According to Boey and colleagues in 1982 more than half the cultures of peritoneal fluid taken at the time of operation for perforation were sterile. Antacids and H2 antagonists raise the intragastric pH and thus may allow bacterial growth in the stomach in duodenal ulcer patients. Bacterial contamination may be highest in patients who were taking these drugs before perforation.

As a result of perforation, generalized and diffuse peritonitis develops in a matter of few hours. The development of diffuse peritonitis can be considered in the following 3 stages.

a) Early stage

b) Intermediate stage

c) Late Stage

a) Early stage

This is the stage of diffuse or spreading peritonitis. Pain, which commences in one part of abdomen at the time of onset of perforation become more widespread. Later the patient develops vomiting which becomes frequent and bile strained and most often effortless. The patient lies supine with knees flexed. The temperature is usually raised but in late fulminating cases it maybe subnormal. A rising pulse rate shown by recording an hourly pulse rate is an indication that the peritonitis is advancing. Peritonitis further lead to the development of paralytic ileus, which may be progressive. At the outset the peristaltic activity ceases as a normal response to prevent dissemination but afterwards the bacterial toxins prevent the normal activity of the nerve plexuses. When the bowel begins to recover, the early feeble peristaltic waves may not be able to overcome the obstructive effect of the newly formed slender adhesions between the adjacent loops of intestines. This further leads to quiescence of activity of intestinal musculature.

Further vomiting leads to loss of sodium chloride and also potassium leading to fluid and electrolytic imbalance. Hypokalaemia is also responsible for the development of paralytic ileus. The development of paralytic ileus can be recognized by

a) Distension of abdomen

b) Vomiting

c) Absence of normal intestinal peristaltic sounds on auscultation

b) Intermediate Stage

This stage develops after 72 hours after the onset of diffuse peritonitis. In those cases, which may improve, there will be fall in the pulse rate but in those cases where no improvement occurs the pulse rate continues to rise. The abdominal rigidity may pass of and give place to increasing abdominally distension. The whole abdomen will be acutely tender. The amount of fluid removed by gastric aspirations also increases.

c) Late Stage

If by the 4^{th} and 5 day after the occurrence of perforation, no localization of infection occurs the patients conditions becomes extremely grave. The whole abdomen will be grossly distended, The pulse becomes rapid and thready. The eyes will be sunker, the nose may appear pinched, and the tongue will be dry and shriveled. The forehead and hand will be cold and clammy and lace is drawn and anxious. The classical facies has been described as “Hippocratic facies”. Finally the patient goes into a state of semi-consciousness, which might lead to complete loss of consciousness and death. The amount and nature of the peritoneal fluid will have an important bearing upon the prognosis. If there is more fluid and if it is thick containing particles of food, the outlook may be worse and mortality rate higher.

Some perforations leak continuously and some are sealed off by fibrous and omental adhesions. Few are sealed permanently by the natural methods alone. It is possible that a limited volume of irritant and infected material is diluted and neutralize by reactive peritoneal exudates rich in polymer and antibodies.

When large accumulations are found, the intestines, which are submerged in the turbid fluid, are unable to form defensive adhesions or limit the spread of contamination.

If the perforation is small and stomach is empty the patient is in fact more fortunate in that the perforation may be sealed off or at best a localized abscess may form. When pus tracks up towards diaphragm a subphrenie abscess may develop. When it gets shut off in the pelvis a localized pelvic abscess-may travel down. Further tracking behind the peritoneum to the right illac fossa may produce a swelling, which may be indistinguishable from that of an appendicular abscess. When such abscess is drained an external duodenal fistula results.

Clinical Picture

Usually, there is a previous history of dyspepsia and the patient may know from the earlier investigations that he has an ulcer. However, patients suffering from a perforation may be in no fit state to give an account of previous investigation. Such information may only be obtained further questioning during convalescence. Rarely perforation may take place during a first attack of acute ulceration. An untreated patient passes through 3 clinical phase following perforation of an ulcer with the pathological process and clinical picture tend to overlap.

1. Stage of chemical peritonitis or primary stage
2. Stage of reaction or illusion or secondary stage
3. Stage of bacterial peritonitis or tertiary state

1. Stage of chemical peritonitis

This stage follows immediately upon perforation and lasts for six hours. The symptoms which arise with dramatic suddenness are due to the intense irritation of the peritoneum produces the immediate reflex effect on the circulatory and nervous system, commonly referred to as primary neurogenic shock. Usually, however, this state of so-called shock is transient and most patients when first seen in hospitals have a relatively normal pulse and blood pressure although they are obviously inconsiderable distress. In the early stage, nausea and vomiting are uncommon although retching may be troublesome in addition to abdominal pain, there may be referred pain felt over one or both shoulders as a result of diaphragmatic irritation. The on examination it will be seen that the patient lies almost rigid and in supine position with his legs drawn up and his hands held tensely to his side; is afraid to move for the slightest movement aggravates the pain. In new instances the patient may be extremely restless and again he may be curled up in bed in a position of flexion with his hands grasping the epigastrium. The face is pale and sweating and the expression is one of anxiety or fear. The extremities are pale, cold or moist with sweat.

The temperature during the primary stage may be subnormal as low as 95-96°F or normal. Occasionally it is slightly raised. Respiratory rate is always increased and respiratory excursions are shallow or thoracic in nature owing to the immobility of diaphragm.

On palpation, the muscles are tensely rigid and board like. This rigidity is universal and extends into the flank, there is marked tenderness and usually rebound tenderness, which likewise extends to all the parts. In old people and

debilitated patients the rigidity may be less marked, but tenderness is no less marked. The abdomen is often tympanic from the escape of gas on percussion, the liver dullness may be diminished or absent, the sign is less reliable than is radiological counterpart. The patient suffering is obviously so great that it is rarely justifiable to elicit shifting dullness. Sometimes, the fluid escaping from a perforated DU may tickle down the right paracolic gutter producing signs suggestive of acute appendicitis with tenderness and rigidity limited to the right side of the abdomen.

On rectal examination, there may be tenderness. The time taken for transition from primary to secondary stage from the moment of perforation is usually 2-6 hours depending upon the site of perforation and magnitude of peritoneal soiling. Also during this stage there may be sealing of perforation spontaneously but if there is gross leakage of gastric and duodenal contents the patient may rapidly pass to the stage of septic peritonitis. The chance of recovery depends upon the correct diagnosis at this stage and great attention should be paid to the early features of this catastrophe.

2. Stage of reaction or illusion or secondary stage

This stage of reaction rarely exceeds 6 hours. The pain, which was most intense at the moment of perforation and during peritoneal irritation; tends to ease off during this stage. This is due to dilution of the irritants by the peritoneal exudates. This stage has been called as **"STAGE OF DELUSION OR DECEPTIVE STAGE".** Here there is a general improvement in the patient's condition. He feels better and thinks the crisis has passed. Sharp edge of pain has been dulled: patient feels warm and with improvement of the general condition.

He may be still be sweating but the extremities are no longer cold. Temperature is normal or slightly raised: pulse rate shows only a little change in rate.

Patient is often thirsty; the symptoms may be relieved. The signs will persist and on careful examination, it will be seen that ala nazi are working vigorously. Respiration is still shallow; laboured and costal in time and the patient lies completely motionless. Tenderness and rigidity are still present in a marked degree.

Most important physical signs that appear during this stage are

1. Shifting dullness
2. Obliteation of normal live dullness in mid axillary line

Abdomen is silent on auscultation,

Per rectal examination may reveal tenderness in the rectovesical pouch and rectovaginal pouch and rectovaginal pouch in male and female respectively.

Plain X-ray of the abdomen in the erect posture may show the gas shadow under the right cupola of diaphragm. However negative X-ray finding does not rule out the diagnosis of perforation. In long standing cases of duodenal perforation with peritonitis there may be sympathetic pleural effusion on one side or both as demonstrated chest x-ray.

Stage of peritonitis[20,21]

This stage develops 6-12 hours after perforation. This stage is essentially same as that of generalized peritonitis from any other cause. Pain is less severe and vomiting more frequent while hiccough may further distress the patient.

In this stage dehydration and electrolytic depletion become more evident and may be due to presence of sweating, vomiting and outpouring of fluid into peritoneal cavity and distended intestine. Fever is usually present and is above 100^0F and the body dry and flushed while the lips and tongue are dry and coated. The patient has an anxious look and shrunken eyes, the so called **"HIPPOCRATICAL FACIES".** Pulse rate steady raises and becomes small and thready and respiration is shallow and rapid. Blood pressure begins to fall indicating that hypovolaemic shock with circulatory failure has supervened.

Examination of the abdomen shows following findings

Abdomen is distended; abdominal muscles show guarding limiting distension. Tenderness is still generalized. On auscultation occasional obstructive tinkle is heard.

Characteristic picture of intestinal obstruction due to paralytic ileus with effortless regurgitation of dark and faecal smelling fluid but meteorism will take 36-48 hours do develop. Terminal stage is characterized by toxaemia dehydration, paralytic ileus; oligaemia and hypovolaemic shock with circulatory failure. Fatal outcome is inevitable. The patient may become delirious followed by coma or may remain acutely conscious. Death usually take place 4-5 days after perforation in an untreated case.

Subacute Perforation

An ulcer may perforate and the perforation may seal rapidly before there is spillage of gastric and duodenal contents into the general peritoneal cavity. Patients preset with sudden onset of acute upper abdominal pain often more severe in right upper quadrant. It may radiate to the back; to the precardium or to the left scapular

region. Respiration shall be shallow and deep inspiration may be associated with abrupt catch in the breadth. Examination may show local tenderness and rigidity but the rest of the abdomen soft to palpation and non-tender. Plain X-ray in the erect posture will reveal a small amount of gas under the diaphragm. The condition closely mimics acute cholecystitis. There will no fever and a history of chronic peptic ulceration is present. After an hour or two with bed rest the pain will usually subside. Rarely signs of acute perforation may develop.

Chronic Perforation

Perforation of an ulcer with walled off adhesions are by adjacent viscera will lead to formation of chronic abscess and thee will be considerable confusion in diagnosis. These patients do not have any signs and symptoms of generalized peritonitis and seldom are diagnosed. The common sites for an abscess to form are Morrison's Pouch, right infrahepatic subphrenic space. Diagnosis is made only on a exploratory Laparotomy drain the abscess.

Perforation associated with Haemorrhage[20]

Association between perforation and massive haemorrhage is grave but a rare complication. This may be present in the following ways.

a. Haemorrhage and perforation occurring concomitantly
b. Haemorrhage following recently sutured perforation
c. Perforation occurring during medical treatment for haemorrhage

Posterior duodenal ulcer penetrates the pancreas/liver/ left gastric pedicle. Ulcer ruptures where it is relatively unsupported, but the bleeding arises from the erosion of large vessel like gastro duodenal artery; splenic artery of left gastric artery. The middle celiac artery or one of its branches may be the site in case of

perforation of peptic ulcer with signs of haemorrhage. Patient is usually pale in severely shocked with rapid pulse and low blood pressure.

Perforation and pyloric stenosis

This combination is extremely rare.

Etiological Factors

Exact reason for the perforation of DU are not certain. But some etiological factors help in considering the condition.

1. Sex incidence
2. Age incidence
3. Occupational incidence
4. Seasonal incidence
5. Geographical incidence
6. Trauma
7. Perforation in association with well defined clinical states.

Other factors that are responsible are

1. Physiological and emotional factors
2. Relation to food
3. Iatrogenic Perforation
4. Relation to diet
5. Relation to barium meal examination
6. Trauma during endoscopy
7. Relation to blood groups
8. Genetic factors
9. Environmental factors

1. Sex Incidence

Most notable feature is that there is a great preponderance of males over females. But recent literature has shown that increasing proportion of females. Cause for this is difficult to determine but in the last forty years, there is an increased incidence in women taking more responsibilities from men; occupations which are strenuous, emotionally taxing and a higher incidence of alcohol and smoking in women. Perforated DU is common in both sexes but a higher proportion of females have gastric ulcer. In males 83.6% are DU perforation, whereas 12 % of cases of gastric perforation found and die comparable figures in females being 70.6% and 25% respectively.

2. Age Incidence

Age incidence in peptic ulcer perforations has definitely shifted to older age groups. Although acid secretion decreases with age; the DU increases. The production of duodenal bicarbonate decreases with age suggesting a breakdown of mucosal defense mechanisms, which may be responsible.

Also in older age[22] group individuals are more likely to develop medical conditions like arthritis and chronic obstructive pulmonary diseases, which may preclude them in long term, use of painkillers and other medications, which may be one of the factors responsible for perforation during this age group.

Highest incidence was found in 45-55 years. Perforations very rarely occur in an infant of a few days or in extreme old age.

3. Occupational Incidence[23,24]

Since 1950, both gastric and duodenal ulcers have been more frequent in lower socio-economic groups in UK and USA. Heavy manual work, lifting of

weights and strenuous exercise, which causes a rise of intra-abdominal pressure, predisposes a patient suffering form peptic ulcer for perforation.

In India, most of the perforated peptic ulcers are among the labour class particularly farmers and people of poor class. The reason for this is poor socio-economic conditions in these people. They are more prone to develop perforation. The general resistance to the people to the states of stress and strain will be much less compared to the middle and rich class of people.

4. Trauma

Trauma to the abdomen for eg. Forcible below over the epigastric region may precipitate perforation. This is said to play a major part in as many as 4% of perforations. But its importance is probably not great not for muscular strain reduces gastric mobility.

5. Seasonal Incidence

In winter writers have sought to show that there is an increase in ulcer symptoms and in ulcer perforations in winter.

According to Shanmukeshwara Rao[44], the number or perforations occur during November, December and January in India. This may be due to the fact that the work of cultivators with more during winter season.

6. Geographic Incidence

There are great variations in incidence of perforations in various part of the world. It is common particularly among westernized civilizations and practically unknown among more primitive populations such as Buntus in South Africa. When primitive people move to working areas of western civilization and adopt western social habits they become more likely to peptic ulceration and perforation. In India

too this trend seems to true. As the incidence is more in southern parts of the country and there is also an increased incidence of perforation in lower socio-economic class people who have migrated to urban areas and who have adapted to the life style of stress and habitat of the urban areas are more prone for perforation.

7. Perforation of Peptic Ulcer in association with other Clinical States

The following well-defined clinical states may be associated with perforation.

a. Burns
b. Neurological injury
c. ZE syndrome
d. Aortic aneurysm surgery
e. Cardiac transplantation
f. Rental transplantation

10. Other factors

A. Emotional stress and psychological factors

It is common experience to observe that individuals who perforate will admit recent worry or over work.

B. Relation to food

Beans found that 90% perforate more than 2 hours after a meal and only 10% immediately after food. Acute distensions immediately after the food might be expected as a factor, but De Bakey in his extensive survey suggest that the perforation is unusual within 3 hours after a meal.

C. Iatrogenic perforations

Various authors suggest that if excessive doses of steroids are given to patients suffering from chronic duodenal ulcer they may develop manifestations of acute exacerbations and also the perforation of the duodenum. Steroids by their anti-inflammatory property prevent fibrous tissue formation at the site of ulcer and predisposes for the perforation. Also there is increased acidity during cortisone therapy and cause perforation of peptic ulcer. Regular use **NSAIDS**[22,23] was found in 30% of patients with perforated ulcers.

The drugs implicated in precipitating perforations are[22,23,25]

a. Aspirin
b. Phenylbutazone (Now banned in the throughout the world)
c. Colchicine
d. Adenocortico Trophic hormones in excessive doses
e. Oral administration of streprtokinase and streptodornase cause perforation

This is explained by their mucolytic and fibrinolytic actions.

D. Relation to Dietary Factors

Dietary difficulties particularly vitamin deficiency is common in India. Mac Canson has provided experimental evidence to suggest that in India, vitamin deficiency is at fault. Deficiency of Vitamin A is also being blamed.

E. Relations to Barium Meal examination

It has been mentioned that administration of barium meal radiological examination may be a merely a coincidental factor with the occurrence of' the perforation otherwise imminent.

F. Trauma during Endoscopy

Internal trauma, which might be caused during specialized investigation like gastroscopy rarely, induces perforation.

G. Relations to Blood Groups

Since duodenal ulcer is more common in presence of blood group "O" the incidence of DU perforation is correspondingly high. Persons with blood group O are about 3 times more likely to develop a peptic ulcer than persons of other group.

H. Environmental Factors

During the last 50 years there is a remarkable increase in the frequency of incidence of peptic ulcer. This is attributed to alterations in environment. One reaction to such environments, the increase in wear and tear of life in this age of "rush and hurry" is the factor responsible for the ulcer to develop. The same reason also holds good of occurrence of perforations.

Diagnosis

Diagnosis of perforated DU is arrived at by

1. Careful elicitation of history
2. Clinical Examination of the Patients —both general and local
3. Confirmation of diagnosis by necessary investigations

Differential Diagnosis[21]

The differential diagnosis falls mainly into two categories;

1. Intraabdominal conditions

a. Acute gastric ulcer perforation
b. Acute ileal perforation-typhoid and non-specific
c. Acute Pancreatitis

d. Acute appendicitis
e. Acute choloecystitis
f. Acute intestinal obstruction
g. Vascular accidents like mesenteric infarction and ruptured aneurysm
h. Ruptured ectopic gestation
i. Acute peritonitis from any other cause
j. Peptic ulcer in Meckel's diverticulum.

2. Intrathoracic conditions

a. Acute myocardial infarction
b. Pneumonia
c. Pleurisy
d. Acute pericarditis

According to Kozak and Meyers a significant number of perforated DU patients showed evidence of anaemia. Hb needs correction if the patient is severely anaemic. Due to marked fluid loss, the peritoneal cavity, some of the patients may show haemoconcentration.

2. ECG in 12 Leads

Especially in the patients over the age of 40 years, this is very important. It helps to exclude the conditions where there will be characteristic ECG changes, which simulated DU perforation and rule out underlying cardiovascular diseases in a case of DU perforation.

3. X-RAY of Chest PA view Diaphragm

This is routinely practiced for three reasons.

a. To exclude cardio thoracic conditions which simulate DU perforation
b. To rule out underlying cardiorespiratory diseases
c. To identify any sympathetic effusion leading to cardiac respiratory distress secondary to DU perforation.

4. Urine

It is examined routinely for sugar, albumin and microscopy.

5. Plain X-ray of the abdomen to confirm the diagnosis of the peptic ulcer perforation and cases, this is one of the most important investigation. Radiograph can be taken either in erect posture or left lateral decubitus.

Presence of air under the diaphragm is revealed through plain x-ray-the right cupola of the diaphragm involved in about 70% of the cases. It is very essential that the patient waits for 5-10 minutes either in the erect posture or in the left lateral decubitus to get a positive finding radiologically. In the earlier stages of perforation the plain x-ray of the abdomen may not reveal the presence of air under the diaphragm. In such cases, repeated x-rays should be taken after waiting for 1-2 hours.

The air may also collect under the left cupola of the diaphragm. Since normally the fundal gas shadow is seen on the left side, it may be difficult to distinguish the collection of the air under the left dome. But if carefull observed a collection of air would be visualized, well above the level of air shadow in the fundus to the stomach. 20-30 ml of air can be injected into the stomach through the nasogastric tube in these case where radiologically, it will not be possible to confirm the presence or absence of perforation after aspirating gastric contents.

Radiograph is taken in the sitting or erect posture after the patient lies on the left side for a few minutes. In these circumstances, if a perforation is present, the cresentic translucent area will be seen under the right dome of the diaphragm in a very high percentage case. The presence of air under the right dome of the diaphragm is both diagnostic and prognostic and prognostic importance. The amount of air collected may possibly give a clue not only regarding the size of perforation but also regarding the probable duration of perforation. It also affords useful information regarding the line of treatment to be adopted in cases where there is minimal collection of gas under the diaphragm, there is a chance for conservative line of treatment whereas operative line of treatment is mandatory if huge collection of gas is present.

At times when the patient is not in a position to stand or sit. x-ray of the abdomen is taken in supine positions. In this position gas may be outlined lying free in the peritoneal cavity. A careful watch is necessary to differentiate gas shadow in the abdomen, which have got a definite contour of gastrointestinal tract. There are chances of missing the gas under the right dome of the diaphragm. If the x-ray taken is not sufficiently high up at least to the level of 7 or 8 of the costol cartilage. Free gas under 1 both halves of the diaphragm is not necessarily indicative of perforated DU. Free gas under the diaphragm may be demonstrated under the following conditions.

1. All small bowel ulcer perforations which include perforation of typhoidal ulcer, non specific ulcer and traumatic ulcer.
2. All large bowel ulcer perforations
3. Perforation of gall bladder due to stones or due to trauma

4. Interposition either small bowel or large bowel between dome of the diaphragm and funds of the diaphragm
5. Liver abscess with gas forming organisms involving the upper most portion of the liver in which gas bubble may be demonstrated.
6. Surgical procedures
    a. After recent Laparotomy,
    b. Peritoneoscopy
    c. Pneumoperitoneal as a therapeutic measure in collapsing the cavity in lungs in pulmonary tuberculosis
    d. Rubin's test- to know the tubal patency n cases of infertility
    e. Retro peritoneal air insufflations in certain diagnostic procedures Cases have been recorded in western countries where radiological evidence of gas under the diaphragm particularly in 1adies, engaged chronically in sweeping the floors. In such cases speculations have been made that an entry might have occurred through the genital tract possibly facilitated by the knee-chest exercises. It is not possible to distinguish between perforated gastric and perforated DU radiographically.

Upper gastrointestinal study with gastrograffin

To distinguish between gastric and duodenal perforate it has been suggested that instillation of radio opaque material and gastrograffin by indwelling nasongastric tube is helpful. But pylorospasm may prevent the entry of dye into the first part of the duodenum, which is the commonest site of perforation. The

manipulation required for the additional diagnostic radiography are an added strain to a shocked patient and are rarely justified.

Serum Amylase Estimation[25]

Serum amylase is one the most used single means for recognizing certain acute abdomen cases especially acute pancreatitis. The normal level of serum amylase ranges from 62 to 170 units. The normal according to Somogyi units in 80 to 150 units serum amylase activity is also expressed in forms of Wohi goneth units, normal being 3 to 12 units. But Somogyl units are more reliable than others.

The reasons to why serum amylase is increased in perforated duodenal ulcer is due to the escape of pancreatic juice and absorption of its ferments. It must be emphasized that it is exceptional to obtain a very high levels of serum amylase in perforated peptic ulcers.

Urinary Amylase Estimates

Estimation of the total amount of amylase in a 24 hour. Urine sample is more accurate than in the simple measurement of the concentration of the enzyme in the urine; blood or the serum lipase.

The normal values of urinary amylase (diastase) expressed are 35 units in 24 hours collection of urine and in casual specimen upto 50 units may be taken as normal. After 12-24 hours it often rises to 100 units or more. Sometimes may be as high as 500 units. The urinary amylase level remains elevated after serum amylase level has returned to the normal. Later it too will decline usually to reach normality between 3rd and 5th days.

Diagnostic Peritoneal Tap (DPT)

Bile stained fluid is characteristic of perforated DU, the fluid is turbid and contains food particles and may be cloudy due to debris; mucus and pus.

A smear frequently yields yeast cells and may have a wide variety but lack of abundance of bacteria (BMJ Vol. Dec 1958). The most important observations is that the fluid was never acidic when tested with litmus paper.

The fluid was neutral 1 or alkaline.

Differential diagnosis of bile stained fluid

1. Perforated DU
2. Perforated gastric Ulcer
3. Perforated gall bladder
4. Perforated bile duct
5. Spontaneous Billiary peritonitis.

Culture Sensitivity Of Peritoneal Fluid

A higher incidence of positive culture is obtained by collecting some of the peritoneal fluid for implantation in the culture medium, then when a swab dipped in the exudates is submitted. For the first 10 hrs. the cultures are often sterile because of the bacteriostatic effect of hydrochloric acid from the. stomach, peritonitis at this time although often intense, is a chemical peritonitis. After 12 hrs, inhibition due to the acid is no longer present. The following organisms are often encountered most frequently staphylococci and strepto cocci and less frequently colon bacilli, pneumococci, anaerobes and yeast cells.

Ultrasound scan of abdomen and pelvis

This investigation in duodenal ulcer is to rule out other condition. The usual finding s are presence of free fluid in the peritoneal cavity and in patients presenting late may also show thickened collections in the peritoneum. Also in very big perforations there may be evidence of gas under one or both lobes of diaphragm.

Prognosis

The prognosis depends on the following factors

1. Age and general condition of the patient

The prognosis is good in young adults and middle aged individuals when compared to the older people and children. After 60 years the prognosis worsens with each increasing years of life.

2. Sex

The postoperative mortality is higher in men.

3. Time of occurrence of perforation and time factor

The time which has elapsed between perforation and treatments one of the most important factors in prognosis. The longer the interval between the rupture and the operation the higher the mortality. After 12 hours the death rate rises steeply. The golden time for treatment is between 6 hours and 12 hours after the perforation. If the perforation occurs in the day time better medical care can be obtained rather than in perforation occurring in night and odd ours; particularly those residing at mofussil areas. Also the prognosis is such cases is decided better than a case of perforation occurring in full stomach.

4. Condition of the stomach whether full or empty at the time of perforation

5. Size of Perforation

Smaller the perforation, less the spillage will be into the peritoneal cavity and better would be the prognosis.

6. Chronicity of Ulcer

It is exception for a patient with a perforated acute ulcer not to recover. Great majority of deaths occur when chronic ulcer perforates.

7. Prognosis of Ulcer

Prognosis is better with anterior ulcer perforation of the duodenum than with those occurring in posterior ulcers. The higher the ulcer is situated in the lesser curvature the greater the postoperative majority.

8. Associated Haemorrhage

Associate haemorrhage considerably prejudices the patients chances of recovery. The incidence is 2% according to State (1957) and Avery James et al (1953). A more radical surgical approach and substitution of partial gastrectomy for simple suture has reduced the mortality considerably, but this association is probably the gravest factor in prognosis.

9. Associated Diseases

A high proportion of older patients will have serious disease such as hypertension or cardiovascular and pulmonary conditions and will thus less able to withstand the rigors of perforation and its associated operations. Many will die of pulmonary or cardiac complications; although recovery from the perforation is pathologically complete.

10. Efficiency of Local General Practitioners to Diagnose the conditions

Pre and Post Operative Management

The marked reduction in overall mortality is due to the following factors

- Improved methods of preoperative treatment especially gastric suction; of fluids, electrolyte replacement, antibiotic therapy and blood transfusion.
- Improved methods of anaesthesia especially muscle relaxants and positive pressure pulmonary ventilation.
- Better post operative management particularly greater control by antibiotics.
- Improved operative technique and radical approach to the treatment of perforated ulcer.

Line of Treatment adopted

Prognosis also depends on the line of treatment adopted either conservative or operative. Results of operative treatment if the general condition of the patient permits is better than conservative treatment. The conservative one is a blind procedure wherein we presume that the perforation would be sealed by omentum etc. Whereas in operative line of treatment we can directly see the pathology and can do the best.

Treatment

In most hospitals, the treatment of perforation is operative. Although non operative management role is limited, immediate operation has the significant advantages of enabling the surgeon to confirm the diagnosis, close to perforation, evacuate the peritoneal cavity and if necessary perform definitive ulcer surgery.

Operative Line of Treatment

A surgeon has 3 problems to treat when operating for a perforated ulcer.

a) Peritoneal contamination

b) Perforation

c) Underlying ulcer

The management of the peritoneal contamination is straight forward. However there is considerable controversy over the best management of the perforation and the underlying ulcer disease.

The following operative procedures have been described for the treatment of perforated duodenal ulcer.

Simple closure of perforation[20,50]. This is a basic procedure for closure of perforation. It is usually associated with various technical modifications in order to make the closure a perfect one.

The following modifications are usually undertaken.

i) Simple closure of perforation[1,20] in two layers. Mucosal layer using a absorbable synthetic suture material eg. Vicryl 2-0 and n second laycr of seromuscular layer of unabsorable Mersilk 2-0. This is an ideal procedure for a very small perforation.

ii) Simple closure of perforation in two layers as described above and reinforcing it with a patch of omentum which is a anchored to the sue of perforation. This can again be done it two ways.

ii) a. Taking a isolated piece of or patch of omentum used as free graft which is separated form main body of omentum immersed in Norma Saline for a few minutes and then layered over the closure and sutured with Mersilk 2-0.

ii) b. Taking on adjacent omental patch[1,26,28,50] with an intact vascular pedicle, overlying it over the perforation closure and tying the underlying layer of Mersilk suture which was put in seromuscular layer and tying it over the perforation site Here again there is another technical modification (where the sutures which are initially tied to approximate the edges of the ulcer and omentum placed above the knots leads to a potential space between duodenal serosa and omentum) and hence some surgeons use this technical modification described below.

Here the omentum is closely applied to the serosa and after taking a bite through the wails of duodenum and plugged with omentum[27]. This ensures a watertight closure and in healing, the Cicatix forms a viable omental bed and mucosa rapidly covers the Cicatrix.

This technique also is suitable when the duodenal wall [27,39,50] is very much friable and the bites taken over the adjacent wall may cut through the walls leading to loosening or opening of perforation or taking bites very far off on the healthy wall of duodenal mucosa may lead to subsequent stenosis on healing.

The other options available for closure of perforation are

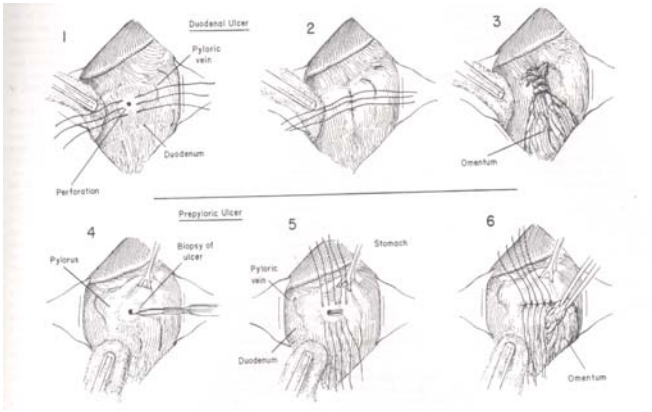
a) In some special circumstances falciform ligament can be used for closure of perforation instead of omental patch.

b) In some cases omentum may be pulled through the perforation site using a Ryle's tube No.18 and omentum can be anchored with a absorbable suture material. When suture material dissolves in 10 days the omentum will have formed a we tight seal over the perforation. Later the Ryle's tube which was inserted per nasally can be pulled out.

Definitive ulcer operations for perforated duodenal ulcer

Simple closure of perforation with drainage procedure like gastroenterostomy with or without vagotomy.

a) Simple closure of perforation with drainage procedure like gastroenterostomy with or without vagotomy
b) Simple closure with PGV [29,31,32,33,34,49]
c) Gastric resection with or without vagotomy[28]
d) Laparoscopic closure of perforation



Pre-operative management

It is mandatory that a short time be spent on resuscitation of the patient before operation. Precipitate surgery often leads to death, whereas preliminary attentions to restoration of fluids, electrolytes and blood with control of septicaemia, makes surgery much safer and prevents possible complications

The following regimen is suggested.

1. As soon as the presumptive diagnosis of perforation is made, pain should be relieved by appropriate dosages of narcotic analgesics

2. A large bore nasogastric tube should be passed and stomach is emptied as completely as possible.
3. Nothing is allowed by mouth, blood should be drawn for grouping and Rh typing and a biochemical profile including serum amylase. An IV infusion should be commenced.
4. A urinary catheter should be inserted; urinary retentions is common in patients with acute peritonitis and it is also necessary to monitor the urinary output closely.
5. It is advisable to obtain a chest x-ray and ECG. Cardiovascular monitoring appropriate to the conditions of the patient should be instituted.
6. Antibiotics should have an appropriate spectrum for the aerobic and anaerobic organisms commonly found in GIT.
7. Consent for definite ulcer operations should be obtained.
8. Preparation of the abdomen is better postponed till the patient is anaesthetized.
9. Blood pressure and pulse rate are recorded at half hourly interval.
10. If the patient is not immunized against Tetanus; injection tetanus toxoid should be given.

Simple closure of perforation together with technical Modifications, as use of Omental Patch[26,27,28,30,31,38]

This operation has been the mainstay of surgical treatment of perforation in most centers and caries a low mortality rate compared to other definitive surgical procedures.

The first operation of consisted of a simple closure of the perforation with two rows of Lembert sutures; washing out the peritoneal cavity with warm water or antiseptic lotions and inserting rubber or glass drainage tubes down to the site of ulcer; to the pelvis and sometimes to the locus.

Bennett (1896) suggested that in some cases in which perforation was very large and the opening difficult to suture owing to the flexibility of the points, omentum could be used to plug the defect. Today it is found more satisfactory to introduce 3 interrupted sutures, one at the top; one in the middle and one at the bottom of perforation and after bringing up a portion of greater omentum and laying it over the defect; to tie them; in order to hold it in position.

Cellan Jones (1929) and R. Graham (1937) emphasized the simplicity and effectiveness of this procedure and maintained that it never produced duodenal stenosis.

This procedure aims solely at warding off the immediate danger in a patient who is seriously ill and whose life is threatened; while subjecting him to the minimum amount of operative trauma. The object is not to cure the ulcer, this can be deferred to a later date. Nevertheless cure may be achieved in 85% of cases by this simple technique. If the ulcer is acute and in 25% if it is of chronic ulcer.

Ilingwoth in 1946 noted that relapse after perforation closure was more frequent in younger patients regardless of their duration of pre-preforation history.

Techniques of simple closure[26,27,28,40]

Anaesthesia

Operations can be undertaken under general anaesthesia. Muscle relaxants have added greatly to the performance of the operations and the peritoneal toilet

and wound closure is simplified. Premedication with morphine and atropine 0.6 mg is ideal.

Incision

Surgeon has a choice between two epigastric incisions; midline or paramedian. The former is the most rapid method of entry. Moreover it is easy to close and sheath. On opening of the peritoneal cavity there is often an escape of gas; a sort of “hissing noise” or “muffled pop” of escaping gas- could be heard if listened carefully. This is diagnostic of perforation of hollow viscus.

Location of Perforation[26,27,28]

The edges of the wound are gently retracted and the right lobe of the liver is drawn upwards with a suitable retractor so that to bring the lesser curvature of stomach, pylorus and the first part of duodenum into view. The stomach is drawn down by applying gentle traction to the greater curvature with a moist abdominal pack.

In most cases the perforation is readily seen (8 out of 1). It maybe oval or circular and punched out and of variable diameter. Through this opening the stomach contents will be seen to pour intermittently or continuously. In duodenal perforation the escaping fluid is usually bile stained and somewhat frothy. The gut around the perforation is infected and edematous in many cases. In acute ulcer the duodenum is mobile and there are no adhesions.

In long chronic ulcers the first part of the duodenum seems to be part of a chronic inflammatory mass consisting of greater omentum, pancreatic head and lower portions of the stomach and sometimes the liver and hepatic flexure of the colon. In many the perforation will be sealed at times of laparotomy and may not

be obvious without a careful search. Occasionally the omentum, sometimes viscous or the anterior abdominal wall scales off the hole and only separation of one or other of these structures will reveal perforation. If the perforation is so obscured gentle dissection with the finger will produce some walling up of fluid and this reveals it.

A careful search should be made for second perforation elsewhere and for evidence of ulcer disease in other sites, such as posterior gastric ulcer that has ruptured into the lesser sac. Occasionally peroration of an ulcer of the 2nd part of duodenum may be unmasked by mobilization of duodenum by Kocher's method[38].

For quick location of perforation when it is not found early, 30-60 ml 1% solution of methylene blue may be injected down the Ryle's tube and the site of perforation becomes readily apparent.

Closure Of perforation[26,27,28,29,40]

There are many ways of closing perforation. The perforation having been found, retraction of the abdominal wall and traction upon the stomach are so arranged as to bring the perforation into best positive view. Among the many ways of closing perforations, simple closure of perforation with interrupted sutures is the ideal method. The suture material use should be a synthetic monofilament sutures such as polydioxanone (PD5). The absorbable characteristics of this material are more predictable than those of catgut. Non-absorbable material such as silk should be avoided; because these materials cause silk ulcers, which may bleed and produce pain. The term placation is used for simple closure method.

The first stitch is taken over the perforation

2nd below the perforation and

3rd through the opening

Cutting out of the sutures were prevented by taking considerable bite of the duodenal wall which is prone to occur especially in odematous tissues.

The first and second stitches should be tied and held in forceps before the center stitch is drawn tight to complete the closure of perforation.

The line of closure should now be reinforced with interrupted seromuscular (Lambert) stitches, which bury the previous suture line. The ends of one or two of these sutures should be utilized to suture a tag at omentum over the closure.

In cases wherein duration is so marked that all stitches tend to cut out, then perforation is closed with omentum along. Omentum is plugged[27] into the hole and is secured with sutures.

It is recommended that the closure sutures be placed to avoid narrowing the lumen of the bowel.

In duodenal ulcer perforation the sutures should be placed in long axis of the gut. If however the tissues are friable or the hole is larger and the sutures cut out, sutures can be placed in the axis which permits the safest closure. It is rare for actual stenosis to be caused by the operation and it can be always treated by gastroenterostomy; 7 to 10 days later when patient overcomes the immediate danger. The aim of operation is to close the perforation securely.

Peritoneal Toilet

It is often stated that general condition of patient improved once peritoneal fluid is aspirated. The exact mechanism is not known. The peritoneal toilet should be done meticulously as a fixed routine with the use of suction.

Drainage

In most recent perforations i.e., within 6 to 8 hours provided that the exudates is little in amount and is not markedly bile stained, it is advisable to close peritoneal cavity without drainage. In perforation beyond 6 to 8 hours where the exudates is usually copious and may be frank pus, it is advisable to provide drainage though a separate incision the Right flank, below the tip of the 11^{th} rib either on one side or both sides.

Looking For A Second Perforation[28]

It is worthwhile making a practice of looking for second perforations. Several examples of simultaneous perforations of two ulcers have been reported. If a second perforation is found; it should be closed as in done for the first one.

Closure of Abdomen

After meticulous haemostasis, the abdominal incision is closed in layers. When there is a gross contamination of the wound; it may be advisable to close the linea alba and to use delayed primary closure of the skin wound.

Different Operative Procedures

1. Simple closure of perforation with gastrojejunostomy and vagotomy.

If the patient is young with good general condition and perforation of less than 24 hours duration; gastro jejunostomy and total truncal vegatomy is the right choice after closure of perforation. The fear of mediastinitis following vagotomy have not been fulfilled. The operation of choice for perforation is associated with stenosis is gastrojejunostomy away from the site of perforation together with total truncal vegatomy.

2. Pyloroplasty and Vagotomy

Pyloroplasty with Vagotomy has been carried out successfully as an emergency procedure in perforate duodenal ulcers. Pyloroplasty and vagotomy is the method of choice in two situations.When perforation is very weak and it is not possible to close the perforation by simple suture or omental patch. If the patient is young the operation is completed by vagotomy. When perforation is associated with haemorrhage.

Haemorrhage in association with perforation is usually due to posterior wall ulcer or penetrating ulcer. It is often convenient to enlarge the perforation by converting it into a pyloroplasty with an incision in the long axis of the stomach; so that suture ligation of the bleeding point in posterior wall of the duodenum can be carried out. The operation may be completed by truncal vagotomy[44,45]. It is easier to convert perforation into pyloroplasty and then add vagotomy if general condition permits. This procedure is less time consuming, easy takes care of the basic pathology lying behind the ulceration and gives long term results as and when performed actively.

The techniques needed in these difficult ulcers have been well described by Herrington and Davidson in 1987.

Simple Closure Of Perforation With Proximal Vagotomy (HSV OR Parietal Cell Vagotomy) [1,30,32,42,43]

This operation denervates the acid-pepsin secreting mucosa but spares the innervation of antrum and pylorus so that a drainage procedure is not required. This operation carries the least mortality of only 0.3% compared to the total truncal vagotomy and gastrojejunostomy

The only disadvantage with PGV is highest recurrence rate of about 2-l5% almost equal to total truncal vagotomy and drainage procedure. PGV carries the lowest incidence of side effects such as dumping syndrome, biliary vomiting and distention. It can be argued that recurrence of ulcer after PGV is easier to deal with than severe dumping and diarrhea. It is clear however that success of PGV depends upon the experience of the surgeon. If a definitive ulcer operation is deemed an appropriate addition to simple closure of a perforated DU PGV is the procedure of choice.

Gastric Resection with Or Without Vagotomy

The estimated overall risk of recurrence is least compared to other methods. It has less than 1% for antrectomy and truncal vagotomy and 2-5% after truncal vagotomy and drainage or PGV is about 4-15%.

However gastric resection carries the highest mortality. This surgery requires an experienced surgeon. Perforation is frequently and "out of hours" emergency and the operation is performed by a trainee surgeon. Partial gastrectomy is a more radical procedure, which requires an experienced surgeon, ideal facilities and a patient with good general condition.

There are other methods, which are safe, and can be done by a trainee surgeon with supervision and less time consuming. Gastric resection is no longer for perforate DU. Gastric resection is advocated provided the patient's general condition is good in cases of perforation associated with Zollinger -Ellison syndrome. Perforations were associated with 23% of the cases of ZE syndrome.

In summary it may: be said the whilst simple suture still remains the most commonly employed method; radical treatment of perforated DU by antrectomy and truncal vagotomy is justified in the hands of experienced surgeon.

Omental closure is indicated for perforation in poor risk cases and for acute ulcer associated with drug ingestion or acute stress. H2 blockers and latest proton pump inhibitors[4,29,35,36] used post operatively goes a long way in reducing the incidence of recurrent ulcers. Elective surgery may be offered to patients who relapse after closure. Only in fit patients with acute ulcer perforation; when an experienced surgeon is available, PGV is undertaken as an ideal operation.

Postoperative Management

The postoperative management play a vital role in the final outcome. The adoption a careful postoperative regime has led to a considerable reduction in mortality and unpleasant Sequelae and complications have become less frequent.

Important Steps In Post Operative Management[4,29]

1. **Position in bed:-** Foot end of the bed is elevated when the patient is returned after operation. This position is maintained for 12-24 hrs till the pulse and blood pressure are stabilized. In order to avoid the risk of vomitus being aspirated the patient is placed in a semi-porne position till the consciousness is regained. Old people are propped up in order to allow maximum pulmonary ventilation in order to reduce future pulmonary complications.
2. **Fluid replacement:-** Patients require intravenous fluids viz., dextrose, dextrose saline and Ringer lactate. About 3 liters are required during 24 hrs. However while calculating the fluid requirement for the day one must take

into account the condition of the patient, quantity of nasogastric aspiration, urine output, perspiration, associated respiratory conditions like chronic bronchitis, pneumona etc. Additional allowances of fluid are made depending upon individual requirement. Overhydration has to be avoided.

3. **Monitoring urine output:-** This is one of the important aspects of post operative care. Patients are usually catheterized by a no. 18 Foley's catheter with strict aseptic precautions. The fluid requirement per day is calculated and started to achieve an adequate urine output of 0.5 ml/kg/hr and calculated for 24 hrs.
4. **Nasogastric aspiration:-** The nasogastric aspiration should be started from the time of surgery and should be continued at regular intervals of 1-2 hrs post operatively. An wide bored 18 size Ryle's tube is inserted. This is the most important aspect of the postoperative care. A correct record is kept about the quantity, colour and the nature of aspiration every time. A very good nasogastric aspiration decompresses the stomach and increases the recovery of the operated site. When bowel sounds appear after 48-72 hrs and when the quantity of aspiration decreases; the Ryle's tube is removed but may have to be kept as long as necessary.
5. **Antibiotics:-** Initially either ciprofloxacillin or cefotaxime with Metronidazole is given parenterally. If peritoneal fluid is sent for culture and sensitivity then further antibiotics are administered depending on the report, for a minimum of 5-7 days from the day of surgery. Injection crystalline penicillin can be given in postoperative respiratory complications.

6. **H2 antagonists[4,29,35]:-** are started parenterally immediately after operation, if only simple closure is done. Now-a-days[4,29,35] proton pump inhibitors are better option.
7. **Blood transfusion -** If the patient is anaemic or perforation is associated with haemorrhage, blood transfusion is advised.
8. **Bowel action:-** After 48-72 hours, a small glycerine enema is given to ensure bowel movement. Drastic purgatives must be avoided.
9. **Diet:-** Oral fluids are started after the removal of Ryle's tube and after the appearance of bowel sounds, if there s no vomiting or distension of the abdomen. The diet is gradually increased through the stages of fluids semisolids and gradually is switched onto normal diet. Proteins and vitamins should be supplemented for quick recovery. It is modern practice to regulate the dietary process by needs and reactions of the patient, rather than by arbitrary rules of the older days.
10. **Exercise and period of rest:-** During recent years, the principles of early ambulation have been more and more widely accepted. The patient is helped into chair by 2nd or 3rd postoperative day and thereafter allowed to start walking with support.

Postoperative care after simple closure[4,29,35,41]

Patient must be explained that the perforation has been closed but the underlying ulcer disease has not been treated. H2 receptor antagonists and proton pump inhibitors should be administered intravenously and then, for a period of 8 weeks orally in full dosage as soon as the patient is able to take orally. After 8

weeks a follow up upper GI endoscopy is performed to ensure that the ulcer has healed.

Mortality rate associated with perforation

Although the mortality rate associated with perforated ulcer has declined over 50 years; still it is substantial because of the increasing number of elderly patients with this problem. Mortality rate varies from one study to the other. In patients without any of comorbid factors present, the mortality rate was 4.2% raising to 6.7% with all three risk factors present. The other factors, which increase the mortality rate, are

Position of the Ulcer- Gastric ulcer perforations carry greater mortality than DU perforation. Higher the ulcer greater is the mortality.

Associated haemorhage

Age - Most authors include extreme age as a risk factor.

The scope of laparoscopic surgery is rapidly expanding. Perforated DU is often repaired by laparoscopic surgery with or without suture. A sutureless method of Laparoscopic treatment of perforated DU using Gelatin sponge plug and fibrin sealant can be done. This procedure is quick, simple secure and well suited for the laparoscopic environment. A laparoscopic patch repair using omental patch to be securely sutured under laparoscopic vision offers an alternative to open surge and avoid the dangers of conservative treatment. Peritoneal lavage can also be done with the same incision. This avoids Laparotomy and decreases hospital stay (average 6.7 days).

Conservative Line Of Treatment (Non Operative Line)[28]

Conservative line of treatment is justified in

a. High risk group of patients with perforations who are likely to die and patients in whom operation is unlikely to be beneficial.
b. As an expectant measure when the diagnosis is uncertain in early acute cases.
c. In cases where perforation is a very small one i.e. a leaking ulcer with correspondingly slight peritoneal reaction.
d. Sub-acute perforation

Non-operative treatment of these patients with modern methods such as ultrasonography. CT scanning to monitor progress and in combination with percutaneous aspiration of fluid collection and abscess deserve revaluation. Of the many advocates of non-operative treatment Herman Taylor (1946) is a forceful representative. Conservative treatment is based on the assumption that clinical and radiological diagnostic method are accurate, the leakage may be controlled by or diminished by gastric section; that fluid and electrolyte loss can be corrected and that peritoneal infection is minimal in early hours after perforation and can be controlled by antibiotics. Taylor believes that this method facilitates natural healing process; whereas suturing of perforation may make ulcer become chronic and intractable. If medical line of treatment is undertaken and operative line is withheld, one of the three results may be expected:

1. The abdominal tenderness and rigidity may gradually disappear and the perforations close.

2. A localized abscess-perigastric, periduodenal, and subdiaphragmatic collection may form.
3. The Patient may die of septic peritonitis.

Highlights of Conservative Line Of Treatment

1. The patient should be placed in fowler's position.
2. Charts: as routine, pulse rate, blood pressure, respiratory rate are recorded every half hourly or hourly in graphic form on a special chart. The temperature is recorded 4^{th} hourly. A special antibiotic chart and an input and output chart are also necessary.
3. Repeated radiographs- It is an essential part of conservative line of treatment. A perforation becomes sealed off; the subdiaphramatic shadow slowly diminishes in size. Conversely, if the shadow has increased in size, after the passage of Ryle's tube of good size, then operation should be performed without delay.
4. Nasogastric aspiration

   It is the most important part of conservative management. After the stomach has been emptied through wide bore tube; it is kept empty by intermittent or continuous nasogastric aspiration. Aspiration should be continued until flatus is passed, if the volume of aspiration is minimal-300 ml or less in 24 hours. no fluids are permitted by mouth for the first 48 hours.
5. Antibiotics must be started right from the time of admission and continued for 5 to 7 days.

6. H2 receptors antagonists or proton pump inhibitors; ranitidine or omeprazole or pentoprazole should be started parenterally from the time of admission. Usually in bid dose.
7. Simple drainage.

   In those cases of perforation, which might seal by fibrinous exudates; simple drainage of peritoneal fluid may be done by making an incision over one or both flanks. This procedure can be carried out under local anaesthesia. simple drainage helps for the early escape of peritoneal fluid and also the gas. By providing this simple drainage, further complication of development of residual abscesses like subphrenic abscess and pelvic abscess can be prevented.

Contra Indications to conservative line of treatment.

1. These are perforation associated with haemorrhage,
2. Perforation following heavy meal.

Advantages of conservation line of treatment

The period of convalescence and absence from work is relatively shorter after this than following operative line of treatment. However this seems to be a small consideration when weighed against the lessened anxiety and ensured results after a simple closure of perforation.

Disadvantages and dangers of conservative treatment

1. One cannot judge without operation whether the peritoneal contamination is excessive or not. Even if the stomach is kept empty, peritoneal contamination can occur from regurgitation of duodenal and jejunal) contents.

2. A perforated carcinoma stomach cannot be diagnosed
3. Risk of leaving another condition unoperated which may require surgery.
4. Risk of lung complications is higher than with operative methods as also is the occurrence of intraperitoneal and subdiaphramalic abscess.

COMPLICATION DUODENAL PERFORATION

Complication include

1. Subphrenic abscess - occurs in neglected duodenal perforation. The duodenal contents track down to subphrenic space and cause an abscess. Subphrenic abscess is remember by the dictum that if there is no abscess anywhere in peritoneal cavity then it will be in subphrenic space.
2. Duodenal fistula - It is a rare complication and is mostly seen in older age patients with poor general medical condition

AIMS OF STUDY

The present study has been undertaken to evaluate the age and sex incidence and the importance of various mode of presentations. The predisposing factors along with the general condition of patient at presentation, which has a relationship with mortality and morbidity also has been considered. comparative study has been made between the types of treatment and complications associated with it also the time taken for surgery and post op. complications have been analyzed. The role of different types of operations performed and the mortality and morbidity associated with each type of surgery has been studied. Also the socio economic status dietary habits and blood group has been analyzed with respect to the presentation of DU perforation.

MATERIALS AND METHODS

A prospective study consisting of 50 patients of DU perforation presenting as acute abdomen to casualty department of Government Rajaji Hospital, Madurai Medical college from September 2007 November 2009. The cases were collected at random and treated at various surgical units of Govt. Rajaji hospital. All the cases were admitted as emergencies. After admission a detailed history was taken and clinical evaluation was done and possible immediate investigations were done. All the 50 cases were taken up for emergency laparotomy. At laparotorny site of perforation. size of perforation and amount of peritoneal contamination was determined later by different operative techniques like simple closure of perforation with technical modification or pyeleroplasty or serosal patch were performed. The decision regarding the line of treatment and type of surgery to be performed in each case were arrived after consideration of following factors.

1. Age of patient
2. General condition of patient
3. Evidence of shock
4. Clinical findings of distention, guarding, Rigidity were determined
5. Plain X-ray abdomen in erect posture and DPL was undertaken to arrive at a clinical diagnosis.
6. Duration of Perforation, site of perforalion and size of perforation with the state of surrounding tissues was determined
7. Associated medical illness was determined
8. Amount of peritoneal contamination determined

Patients were followed with continuous bed side monitoring of vital data in immediate post operative period. Due attention was paid to not be the development of an complications and suitable and appropriate treatment was instituted from time to time. Patients were discharged after satisfactory improvements with the advice regarding diet rest and drugs to be taken and those to be avoided was advised. The need for periodic check ups and need for upper GI endoscopy and Barium meal was advised.

After studying 50 cases a review of available literature has been made. All the cases wee analysed and results tabulated.

Gastric perforation with clinical findings and X-ray and DPL findings similar to DU perforation were excluded from study and hence not mentioned here.

RESULTS AND DISCUSSION

This discussion is based on analysis of data pertaining to 50 cases. It is summarized as follows:

Age Incidence

In the present series age of the patient varied from <20 years to >60 years. The average age of presentation is 42.74 years, which quite in conformity with the opinion expressed by leading authorities.

Author	Year	Peak age of incidence
Turner	1951	30-40 years
James hardy and walker	1961	30-50 years
Jmieson	1947	20-40 years

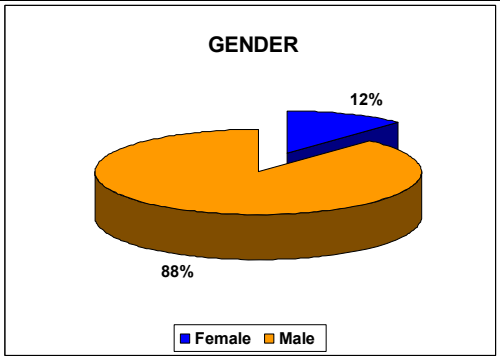
Sex & Regional Incidence

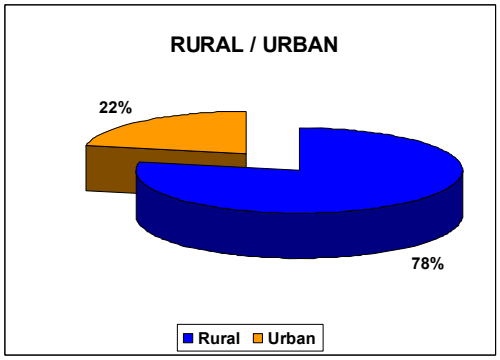
The following table and chart shows that maximum incidence of the cases wherein rural males.

Table 1

Presentation of patients across sex and region

Sex/ Region	Number	Percentage
Sex		
Female	6	12%
Male	44	88%
Region		
Rural	39	78%
Urban	11	22%
Total	**50**	**100**





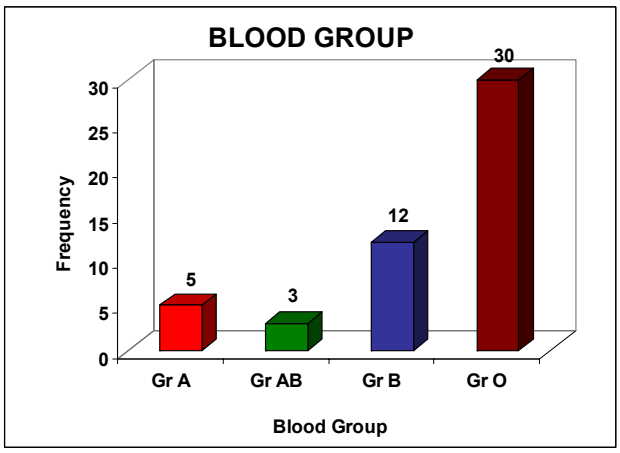
Blood Groups

The following table shows that the incidence of duodenal perforation is highest in blood group O.

Table 2

Presentation of patients in different blood group

Blood group	Number	Percentage
A	5	10%
B	12	24%
O	30	60%
AB	03	06%
Total	**50**	**100**



Occupational Incidence

Incidence of duodenal perforation is highest in lower socio-economic group like farmers, coolies and labourers. This is also similar to views expressed by leading authors.

Predisposing factors

The following table shows the number of patients with predisposing factors like habits and history of APD and history of drug intake.

Table 3
Predisposing factors

Habit	Presentations
No habits	2 (4%)
Tea / coffee / spicy food	29 (58%)
Alcohol	23 (46%)
Smoking	32 (64%)
COPD /Bronchian astama	11 (22%)
APD	10 (20%
Analaagesics / steroids	9 (18%)

Presentation of patients

The following table show that all the patients presented to the hospital with pain abdomen and the maximum incidence of presentation was within 48 hours.

Table 4
Presentation of patients

Pain (in hours)	No of cases	Percentage
With in 24 hours	19	38%
24-48	14	28%
48-72	12	24%
>72	5	10%

Physical Examination of the patient

Most of the patients who presented to the hospital were with no shock or with evidence of mild dehydration.

Location examination of the Patient

All cases presented with distention of abdomen guarding and rigidity. There was obliteration of liver dullness in all the patients. All the patients had

positive DPT and it was bilious in all patients. A few patients who presented late had pus along with bilious aspirate.

Systemic examination

Most of patients presented with no systemic problems and a patient had associated COPD/ osteoarthritis/DM/HTN.

Investigations

Plain X-ray of the abdomen-erect view. In all the patients there was gas under the right dome of the diapharagm (in a few cases it was present under both the domes of the diaphragm with obliteration of liver shadow and ground glass appearance.

Treatment

All the cases in the present series were subjected to surgical management.

Preoperative Treatment

All cases were admitted and thorough clinical work up and resuscitation taken on emergency basis with IV fluids nil orally by mouth. RTA with size 18 tube was undertaken blood drawn for routine investigation like grouping and cross matching. Injectable antibiotics and injection tetanus toxide was given. Parts were prepared for surgery and urine output was monitored.

Operative treatment

All patients were treated with surgery. The following procedures were undertaken based on the size of perforation.

a. Simple closure of perforation with technical modification

b. Definitive surgery like pyloroplasty in some cases

All cases were subjected to peritoneal lavage with normal saline. Intercostal drainage tubes were kept in right paracolic gutter and subhepatic space and in pelvic region in all cases through a separate wound in right flank and left flank respectively. Abdomen closed in layers.

Postoperative treatment

NPO

RTA hourly

IVF as per needs

Injectable antibiotics

In the present series most of the patients received injection, cefotaxime, Inj.Ampicillin and injection Metronidazole.

Injectable H2 receptor blocker or PPI was started. Injection Tramadol was given only if patient complained of severe pain. Continuous monitoring of BP, pulse and urine output done. By about 3rd or 4th day, when quantity of RTA decreased and bowel sounds started returning to normal, RT was removed and Oral fluids allowed. About 5th or 6th day patient allowed soft diet, sutures removed on 8th or 9th day and patient discharged on 10 or 11th day.

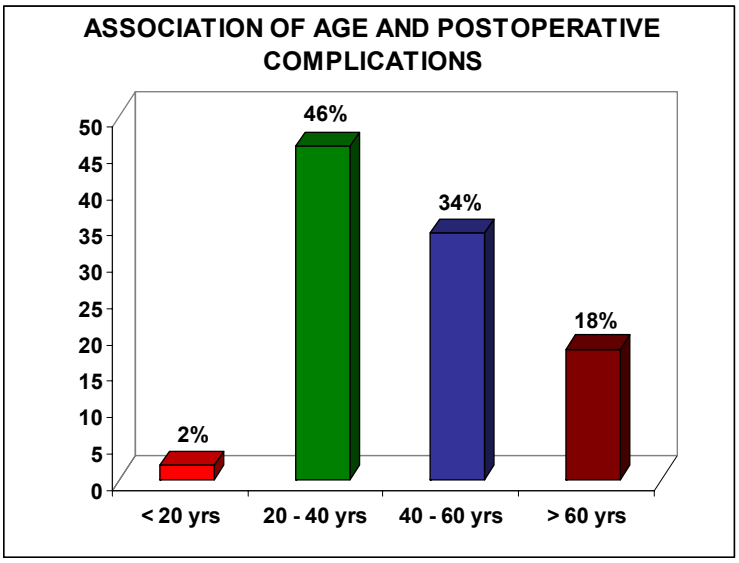
Postoperative Complications

Postoperative complications in relationship with time since onset of pain.

Table 5

Association of age and postoperative complications

Age (in years)	Presentation	Total complications	P value	Minor complications	Major complications
≥20	1 (2%)	0(0)		0 (0)	0(0)
21-40	23 (46%)	4(30.8 %)		4 (40%)	0(0)
41-60	17 (34%)	3 (23%)	0.022	1 (10%)	2 (66.7%)
>60	9 (18%)	18 (46.2%)		5 (50%)	1 (33.3%)
Total	50 (100%)	13 (100%)		10 (100%)	3 (100%)

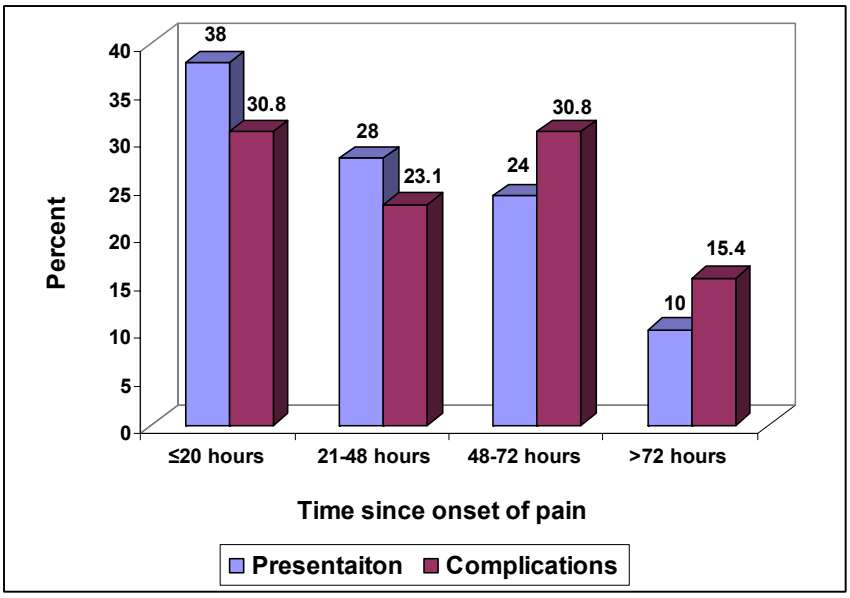


It is observed that for the age group 20-40 years the proportions of minor post op complication is significantly higher compared to any other age groups ($P<0.05$). It is also observed that the major complications increased with the increase of age in years of major complications were observed in the age group of >60 years indicating that older age group are most susceptible for postoperative complications.

Table 6

Association of time since onset of pain and postoperative complications

Time since onset of pain	Presentation	Total complications	P value	Minor complications	Major complications
≤20 hours	19 (38)	4 (30.8)		3 (30)	1 (33.3)
21-48 hours	14 (28)	3 (23.1)		2 (20)	1 (33.3)
48-72 hours	12 (24)	4 (30.8)	0.774	4 (40)	0 (0)
>72 hours	5 (10)	2. (15.4)		1 (10)	1 (33.3)
Q	50 (100)	13 (100)		10 (100)	3. (100)

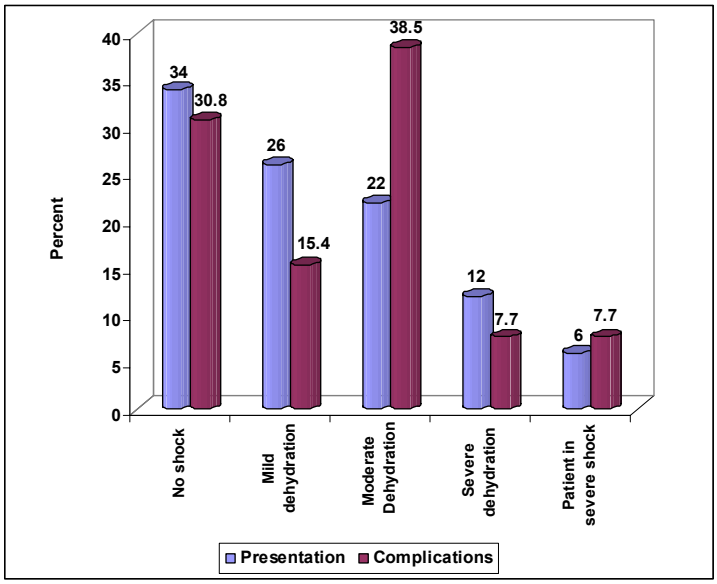


The rate of complications increase with the increasing presentation in hours and this graph shows that there is an increased rate of complications when the presentation (arrival to hospital after the onset of pain) increases to more than 48 hours ($P < 0.05$). Around 66% of patients have presented to hospital within 48 hours.

Table 7

Association of general conditions with the post op complications

General conditions	Presentation	Total complications	P value	Minor complications	Major complications
No shock	17 (34)	4 (30.8)	0.503	3 (30)	1 (33.3)
Mild dehydration	13 (26)	2 (15.4)		2 (20)	0 (0)
Moderate Dehydration	11 (22)	5 (38.5)		4 (40)	1 (33.3)
Severe dehydration	6 (12)	1 (7.7)		1 (10)	0 ()
Patient in severe shock	3 (6)	1 (7.7)		0 (0)	1 (33.3)
Total	**50 (100)**	**13(100)**		**10 (100)**	**3 (100)**



The above table chart shows that 30.0% of the patients presented with no shock and 15% of patients with mild dehydration. The percentage of complications noted in these two groups was significantly lower ($P<0.05$) than patients presenting with the moderate and severe dehydration and severe shock that was associated with significant increased rate of complications ($P<0.05$)

Table 8

Association of habit with postoperative complications

Habits	Presentation n=50	Total complications	P value	Minor complications	Major complications
No habits	2	1	0.932	1	10
Tea / coffee / spicy food	29	7		6	1
Alcohol	23	4		2	2
Smoking	32	6		4	2
COPD / Bronchial astama	11	4		3	1
APD	10	1		1	0
Analaagesics / steroids	9	2		2	0

The above table and graph clearly shows that tea/coffee/spicy food, alcohol, smoking and APD are clear predisposing factors for perforation of D.U. and also associated with significant postoperative complications ($P<0.05$). Also there is a significant relationship between analgesics and steroids with increasing risk of D.U perforation and with postoperative complications.

Table 9

Association of operative procedure with the postop complications

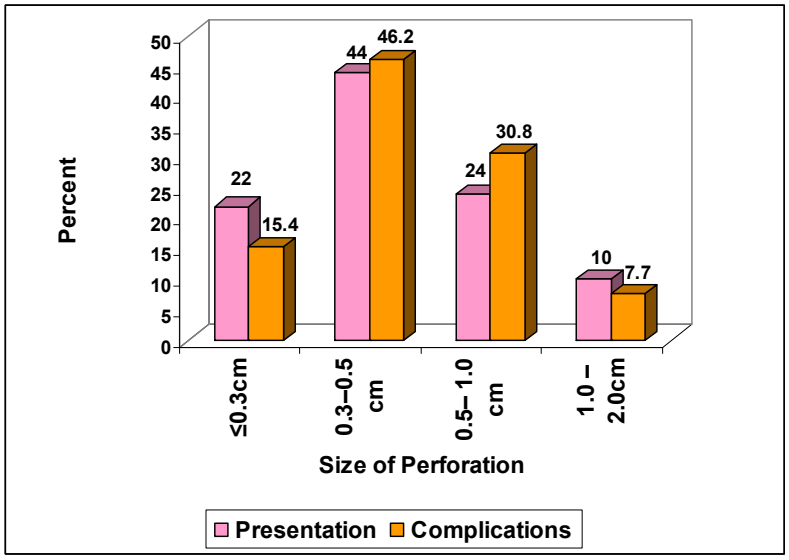
Operative Procedure	Presentations	Total complications	P value	Minor complications	Major complications
Simple closure with live patch of omentum	30 (60)	4 (30.8)		6 (60)	1 (33.3)
Simple closure with free graft of omentum	10 (20)	7 (53.8)		4 (40)	0 (0.0)
Simple closure with of omentum plugging	5 (10.0)	0 (0)	0.467	0 (0)	0 (0)
Simple closure with plugging and pyeleroplasty	2 (4)	1 (7.7)		0 (0)	1 (33.3)
Jejunal Serosal patch	3 (6)	1 (7.7)		0 (0)	-
Total	**50 (100)**	**13 (100)**		**10 (100)**	**3 (100)**

According to this table there is significantly an increased complications noted in simple closure of perforation with free grafting compared to the other procedures, however these findings should be viewed with respect the type of patients selected for undergoing this type of procedure (Most of them were with the increased size of perforation and with surrounding tissues highly friable).

Table – 10

Association of size of perforation with the post op complications

Size of perforation	Presentations	Total Complications	P value	Minor complications	Major complications
≤0.3cm	11 (22)	2 (15.4)	0.850	2 (20)	0 (0)
0.3–0.5 cm	22 (44)	6 (46.2)		6 (60)	0 (0)
0.5– 1.0 cm	12 (24)	4 (30.8)		2 (20)	2 (66.6)
1.0 – 2.0cm	5 (10)	1 (7.7)		10 (0)	1 (33.3)
Total	**50 (100)**	**13 (100)**	**-**	**10 (100)**	**3 (100)**



The above table and graph shows that 44% of presentations with the size of perforation varying from 0.3mm to 0.5mm and there is significant increase in the rate of complications, which is associated with the increasing size of perforation of more than 0.5mm ($p<0.05$).

Association of size of perforation with the general condition of patient (Table 11). This table shows that the size of perforation is related to general condition of the patient and increasing size of perforation is associated with the increased morbidity.

Association of size of perforation with the operation procedure (Table 12): This table shows that simple closure live patch of omentum is the preferred method of closure in mild to moderate size of perforation. Simple closure of perforation with free graft is the procedure which is more likely to help in patients with the size of the perforation not exceeding 0.5 cm. Simple closure with plugging of omentum and simple closure with pyeleroplasty and jejunal serosal partch is the preferred method in increasing size of perforation and in cases where the surrounding tissues are highly edematous and friable.

Table 11

Association of size of perforation with the general condition of patient

Size of perforation	No shock	Mild dehydration	Moderate dehydration	Severe dehydration	Severe shock
< = 0.3cm	4 (23.5)	2 (15.4)	3 (27.3)	1 (16.7)	1 (33.3)
0.3 – 0.5 cm	8 (47.1)	6 (46.2)	4 (36.4)	3 (50)	1 (33.3)
0.5 – 1.0cm	3 (17.6)	3 (23.1)	4 (36.4)	2 (33.3)	0 (0)
1.0-2.0 cm	2 (11.8)	2 (15.4)	0 (0)	0 (0)	1 (33.3)
Total	**17 (100.0)**	**13 (100)**	**11 (100)**	**6 (100)**	**3 (100)**

Table – 12

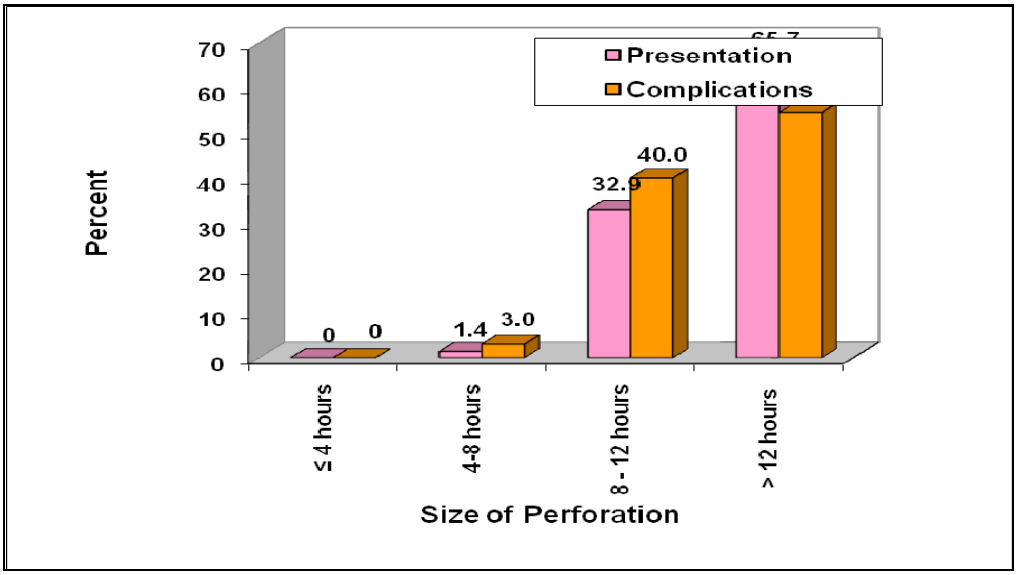
Association of size of perforation with the Operative procedure

Size of perforation	Simple closure with live patch of omentum	Simple closure with fee graft of omentum	Simple closure with plugging of omentum	Simple closure with pyeleroplasty	Jejunal serosal patch
< = 0.3cm	7 (23.3)	0 (0)	4 (80)	0 (0)	0 (0)
0.3 – 0.5 cm	5 (50)	7 (70)	0 (0)	0 (0)	0 (0)
0.5 – 1.0cm	8 (26.7)	3 (30)	0 (0)	1 (50)	3 (100)
1.0-2.0 cm	0 (0)	0 (0)	1 (20)	1 (50)	3 (100)
Total	**30 (100)**	**10 (100)**	**05 (100)**	**2 (100)**	**3 (100)**

Table 13

Association of time gap for surgery since onset of pain with the postoperative complications

Time gap for surgery	Presentations	Total complications	Minor complications	Major complications
≤ 4 hours	0 (0.0)	0 (0.0)	0 (0.0)	0
4 – 8 hours	1 (1.4)	1 (3.0)	1 (3.3)	0
8 – 12 hours	16 (32.9)	8 (40.0)	8 (40.0)	2 (66.7)
> 12 hours	33 (65.7)	12 (54.5)	12 (56.7)	1 (33.3)
Total	50 (100)	21 (100)	21 (100)	3 (100)

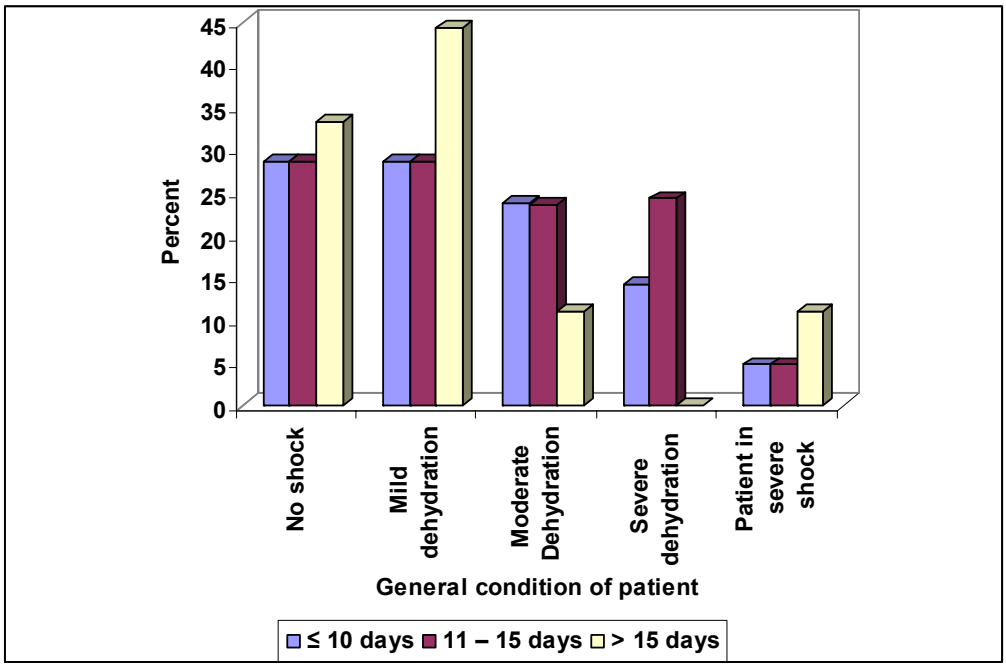


The above table shows that delay in taking the patients for surgery has significant relationship with complications. However the time for taking the patient up for surgery also depends upon the general condition of the patient on presentation and the time interval of presentation and the time interval of presentation to the hospital after onset of pain.

Table 14

Association of general condition of patient with hospital stay

General Condition	Hospital stay		
	≤ 10 days	**11 – 15 days**	**> 15 days**
No shock	6 (28.6)	13 (28.6)	3 (33.3)
Mild dehydration	3 (28.6)	6 (28.6)	4 (44.3)
Moderate dehydration	5 (23.8)	5 (23.6)	1 (11.1)
Severe dehydration	3 (14.3)	3 (24.3)	0 (0)
Patient in severe shock	1 (4.8)	1 (4.8)	1 (11.1)
Total	**20 (100)**	**21 (100)**	**9 (100)**
Average hospital stay	12.9 ± 4.87		



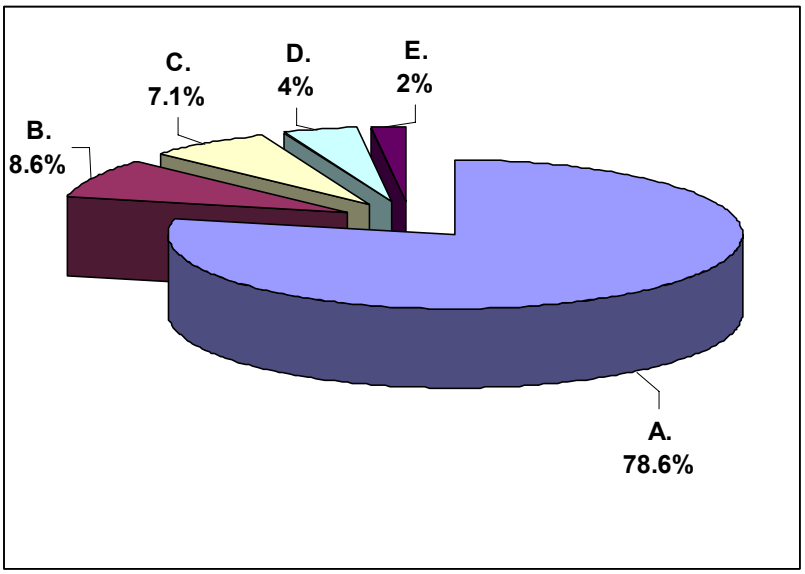
The above table and charts shows that the hospital stay decreases when patient present with no shock or mild dehydration and increases with worsening of general condition of patient. Hence the role of referral hospital and general practitioners is very important in diagnosing this condition at the earliest and

referring to a bigger hospital with minimal resuscitation goes a long way in speedy recovery of patient.

Follow up

Percentage of patients developing complications on follow up

Follow up	No	%
A : uneventful	39	78.6
B: Recurrence of ulcer treated conservatively	4	8.6
C: Recurrence of ulcer treated with definitive surgery	4	7.1
D: Patients with gastric outlet obstruction requiring surgery	2	4
E: Lost for followup	1	2
Total	**50**	**100**



The above table shows that out of the 70 patients 78.6 of patients had no complications on follow up about 8.6% of patients had recurrence of ulcer proved endoscopically and treated conservatively. 7.1% of patients had recurrence of ulcer, which was treated with definitive surgery (TV plus GJ). About 4.3% of patients developed GOO, which required reoperation. One patient was lost for follow up.

CONCLUSION

The following list of conclusions drawn after study of 50 cases.

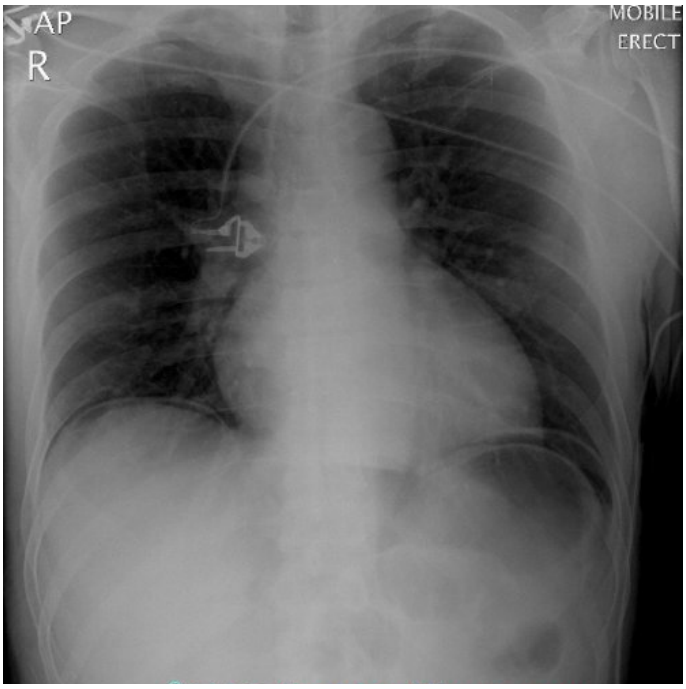
DU perforation is one of the commonest acute abdominal emergencies between 2007 to 2009. Peak age incidence of 42 years shows that duodenal ulcer perforation has a predilection of young and middle age adults. Now-a-days, there is a increased incidence of DU perforation seen in aged indiciduals and these are the population at risk for morbidity and mortality. It is more common in low socio-economic status, rural males. Majority did not have any previous history suggestive of chronic duodenal ulcer. In those present it varied from 1-2 years. Most patients presented with sudden onset of acute pain abdomen in epigastrium and presented to the hospital within 48 hours. General condition of the patient was stable at the time of admission in those patients who came within 48 hours and in those who were referred form other hospitals or general practitioners. All patients had generalized guarding, rigidity and diffuse tenderness with obliteration of liver dullness. All cases showed gas under diaphragm in one or both the domes of diaphragm with ground glass appearance. Diagnostic peritoneal tap was positive in all cases. It was bilious and in few cases pus was aspirated. Maximum number of cases were seen in blood group O. Most of the perforation cases underwent simple closure of perforation with technical modifications. A few cases underwent pyloroplasty due to edematous and friable surrounding tissues around the perforation site along with simple closure of perforation. Four cases had gaping at the site of skin over sutured wound, which was closed with the secondary suturing.

Time of presentation – General condition of patient, size of perforation and time gap for surgery were all related to post operative complications. Increasing size of perforation with the type of closure performed had significant relationship with postoperative complications. There was one death, two were lost for follow up and one patient was discharged against medical advice.

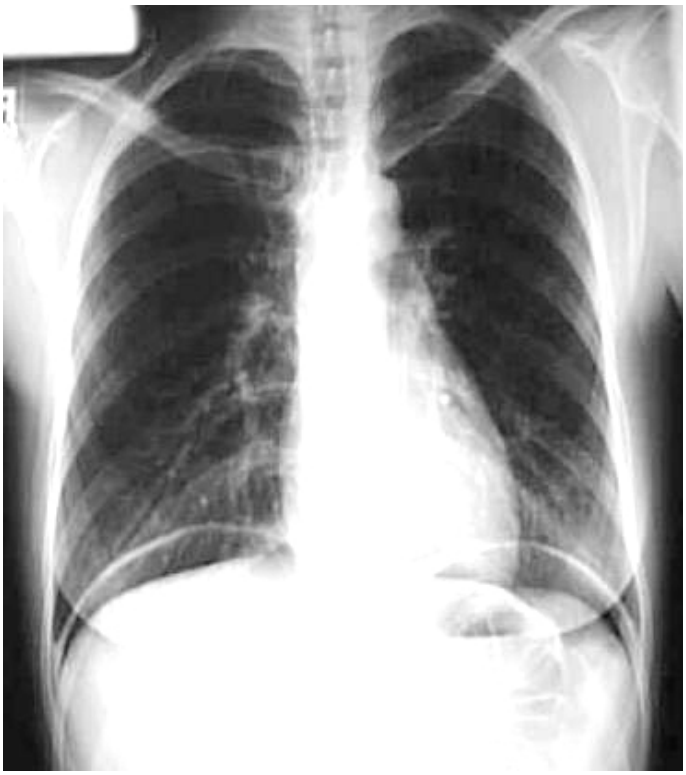
Simple diagnostic work up like plain x-ray abdomen-erect, chest and DPT with routine investigation of blood are quite sufficient in making a correct diagnosis and patients benefit from early presentation to the hospital.

The operative treatment of simple closure's perforation with technical modifications like closure of perforation with live pedicle of omentum or pedicel patch (patch graft) or omental plugging along with post operative PPIs or triple therapy is still the best method of treatment perforated duodenal ulcer. This is a cost effective method in our set up where majority of patients presenting are from poor, rural population in and around madurai city.

Clinical diagnosis is still the best method of arriving at a correct diagnosis of perforated duodenal ulcer and patients benefit from early presentation to the hospital.

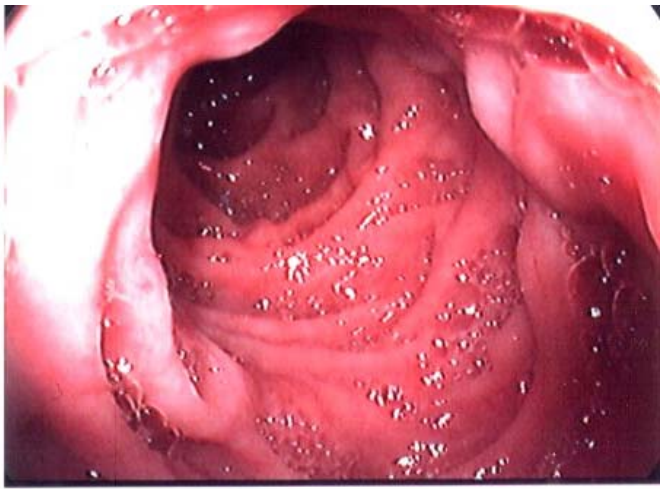


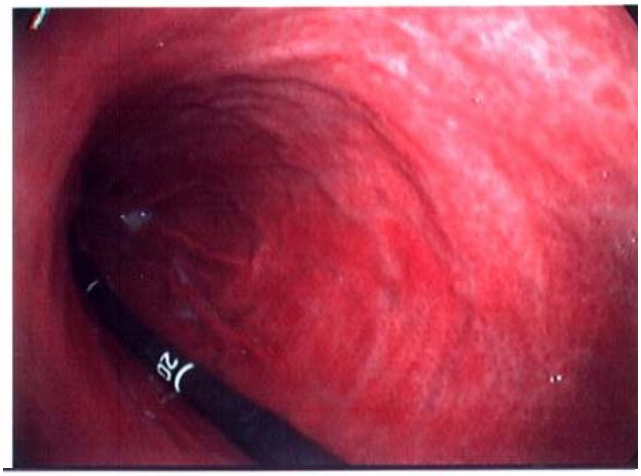
Plain X-ray of abdomen erect view showing gas under both lobes of the diaphragm with obliteration of liver shadow and ground glass appearance

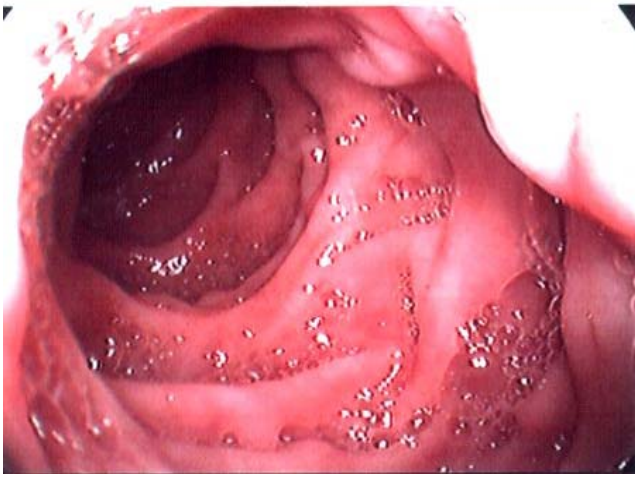


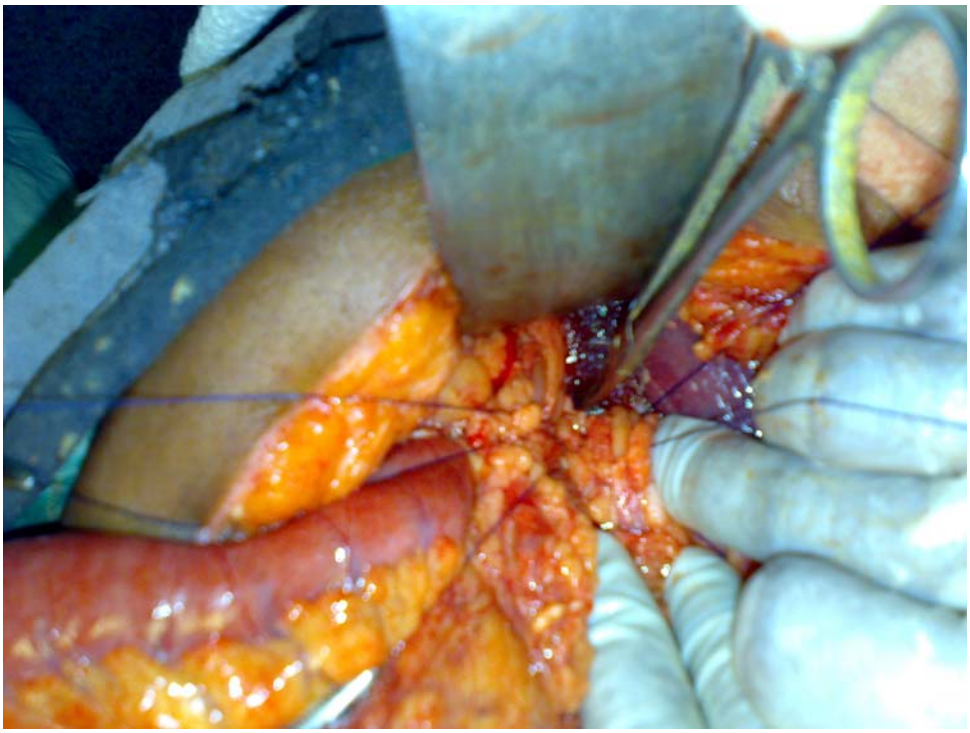
Plain X-ray of abdomen erect view showing gas under both lobes of the diaphragm with obliteration of liver shadow and ground glass appearance

LONGITUDINAL FOLDS OF DUODENUM

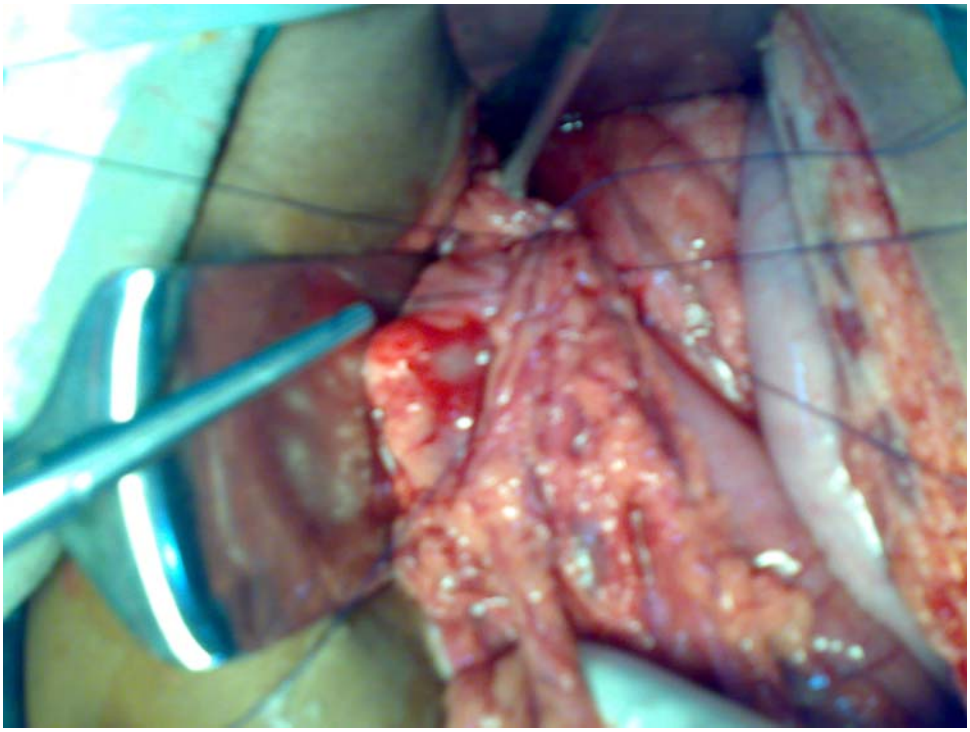




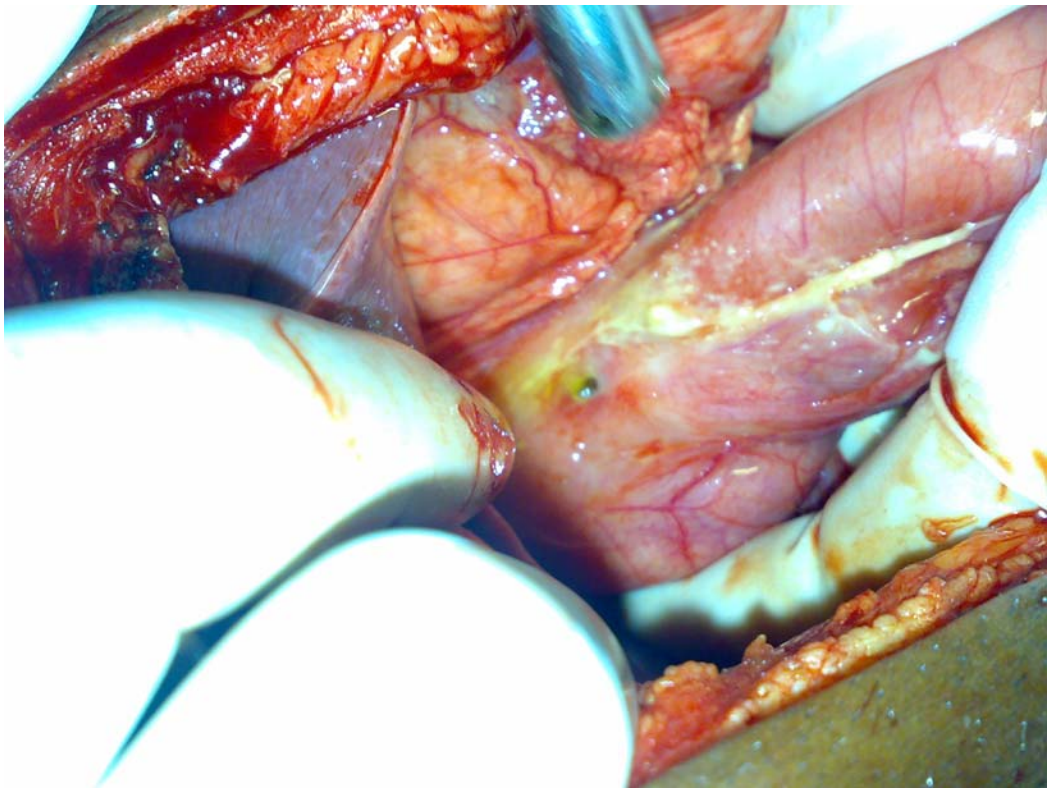




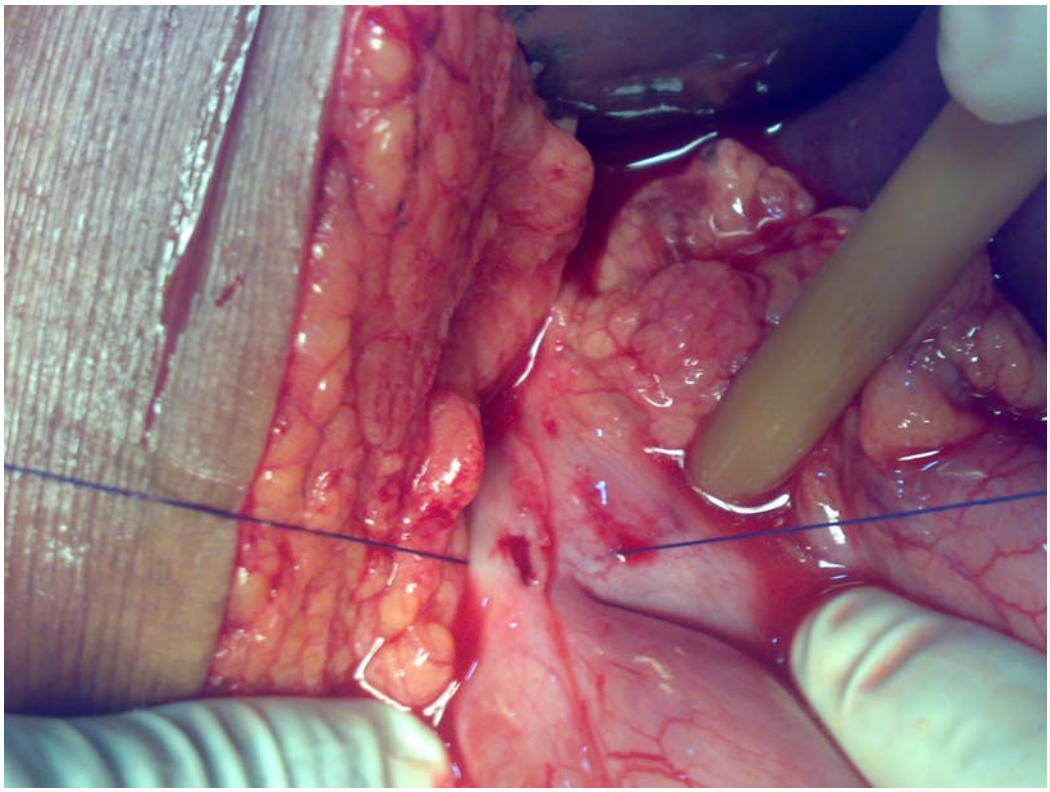
Simple closure of perforation with live patch of omentum



Simple closure of perforation with sutures in place

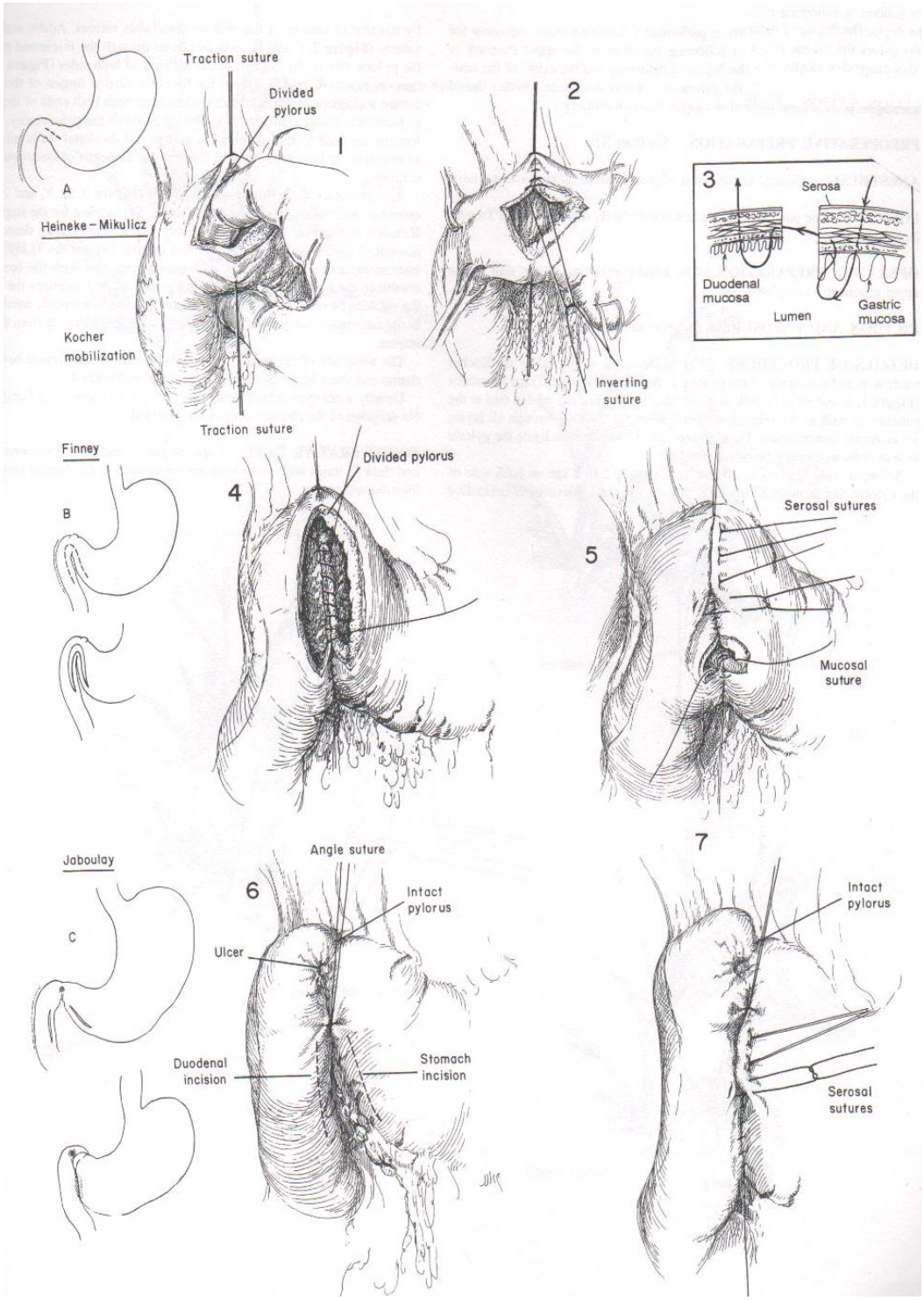


DU perforation – exposure of the site of perforation

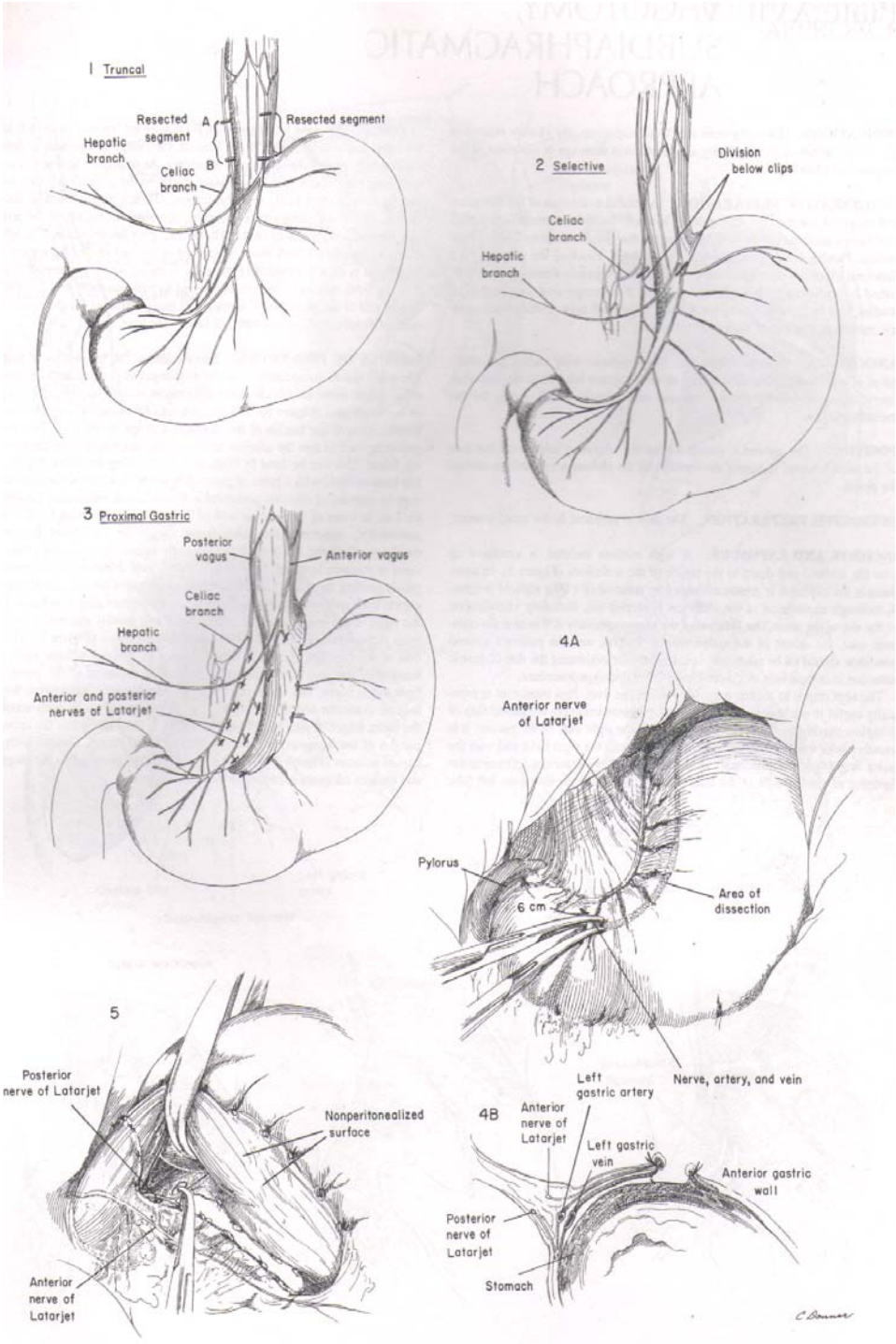


Simple closure of perforation with sutures in place

PYLOROPLASTY



TRUNCAL VAGOTOMY



PROFORMA

Name: Age:

I.P.No.

Address Sex.

Blood group:

Occupation

DOA:

DOS:

DOD:

Socioeconomic status

Presenting symptoms

1. Pain abdomen

a. Time of onset and duraüon
b. Mode of onset
c. Site of pain
d. Type of pain Any radiation
f. Aggravating/Relieving factors

2. Distension

3. Vomiting

a. Frequency

b. Type

c. Contents

4. Haemetemesis and malena

5. Bowels

6. Mlcturition

7. Any history of medications Eg. Asprin/Steroids

Past History

Any previous history of peptic ulcer.

Any treatment received

Personal History

a. Appetite

b. Diet

c. Sleep

d. Habits

e. Coffee/Tea/Spicy food/Alcohol/Smoking/Betal nut chewing

Family History

Any history of peptic ulcer or perforation

Examination

1. General Examination

a) Applearance: Comfortable/Pain Shock
b) Pulse
c) Blood Pressure
d) Temperature
e) Tongue

f) Jaundice
g) Lymphadenopthy
h) Cyanosis
i) Other signs of shock: Sunken Eyes! Cold Extremities / oligurial Dehydration

2. Local Examination

Inspection

a. Contour of abdomen
b. Shape of abdomen
c. Movement with respiration
d. Hernial orifices
e. Skin over abdomen
f. Distention of borne

Palpation

a. Hyperaesthesia
b. Tenderness
c. Guaarding
d. Muscular rigidity
e. Rebound tenderness

Percussion

a. Shifting dullness! Obliteration of liver dullness

Pauscultation

a. Bowel sounds: Heard Not heard
b. External genitalia

Per rectal examination

Systemic examination

a. CVC/ RS /CNS/ Spine
b. Clinical impression

Investigation

Blood: Hb /BT /CT / RBS /Blood urea) Serum creatinine Serum electrolytes.

Urine: Albumin/ sugar/ Microscopy

Blood grouping and cross matching

Plain x-ray abdomen, erect posture / chest x- ray PA View

Diagnostic peritoneal tap

Operative diagnosis

Operative findings

Operative procedure

1. Simple closure of perforation with technical modifications

a. Simple closure with a live patch of omentum
b. Free patch (Pedicle Patch) of omentum
c. Omental Plugging
d. Jejunal serosal patch

2. Pyloroplasty

All were followed with peritoneal wash.

3. Post operative Treatment

4. Post operative course and diagnosis

5. Condition on discharge

Advice

With proton pump inhibitors for 8 weeks

6 Follow up

a. By personal interview- General Condition and examination of abdomen
b. By postal communication
c. Date of visit and finding

Any recurrence of pain abdomen, patients were advised to undergo upper GI endoscopy and based the findings, were advised to undergo definitive ulcer surgery like Bilaterai, Truncal Vagotomy and astrojejunostomy and diet modification.

Any complications in follow us especially pain abdomen / vomiting with features of gastric outlet obstruction were advised to undergo upper GI endoscopy and if presence of GOO present were advised to undergo barium meal study and based on that to undergo surgery like pyloroplasty.

GUIDE TO MASTER CHART

I. Operative Procedure

a. Simple Closure of perforation with live patch of omentum (Live patch) in two layers.

b. Simple closure of perforation with pedicle patch (free graft or pedicle graft) of omentum in two layers.

c. Simple closure of perforation with omental plugging.

d. Jejunal serosal patch

e. Pyleroplasty

II. Habits

a. No habits

b. 1-Tea Coffee! Spicy foo

c. 2-Alcohol

d. 3-Smoking

e. 4-COPD with bronchial asthama

f. 5-APD

g. 6-Analgesics- NSAJD/ Steroids

III. General condition of the patient of presentation

a. 1-No Shock

b. 2-Mild dehydration

c. 3-Moderate dehydration

d. 4-Severe dehydration

e. 5-Severe shock

IV Other Investigations

X-ray of abdomen / DPT/ US of abdomen n and pelvis

+ present;- absent

V. Post operative complications

Minor complications

1. Uneventful/NO Complications
2. Cough
3. Post operative collections in abdomen with fever and loose motion
4. **Major complications**
5. Releak

VI. Any associated Medical Conditional

a. Normal

b. COPD with bronchial asthma

c. Hypetension

d. Diabetes mellitus

e. Arthritis

VII. Time gap for Surgery

1. 0-4 hours
2. 4.8 hours
3. 8-12 hours
4. >12 hours

Age

1. <20 years
2. 21-40 years
3. 41-60 years
4. 61-75 years

WNL -Within normal limits

Resuscitation done

IV fluids injectable anatibiotics/ Ryle's tube aspiration

Triple THerap- Combinatio of proton pump inhibitors (Orneprazole/

Pantazoprazole)

Plus amoxicillin 1 gm bd plus Metronidazole 400 mg tid.

Follow up

I. Stable

ABBREVIATIONS USED

+	-	History Present
-	-	History absent
Chr	-	Chronic
Clo	-	Closure
DM	-	Diabetes mellitus
DOA	-	Date of Admission
DOS	-	Date of Surgery
Upper GI	-	Upper Gastrointetinal
DPT	-	Diagnostic Peritoneal tap
H/o	-	History of
IP No.	-	In Patient No.
LD	-	Left dome
Mod	-	Moderate
NAD	-	Normal
GJ	-	Gastrojejunostomy
TV	-	Truncal vagotomy
P/R	-	Per rectal examination
RD	-	Right dome
REC.UL	-	Recurrent ulcer

S1.No.	-	Serial No.
Yrs	-	Years
RT	-	Ryle’s Tube
RTA	-	Ryle’s tube aspiration
IVF	-	Intravenous fluids
GOO	-	Gastric outlet obstruction