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A STUDY OF CLINICAL PRSENTATION AND MANAGEMENT OF 52 CASES PERFORATIVE PERITONITIS, AND THEIR EVALUATION WITH RESPECT TO SOCIODEMOGRAPHIC FACTORS

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BY

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Dr. Hemendra Kumar

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INTRODUCTION

Peritonitis is defined as inflammation of the serosal membrane that lines the abdominal cavity and the organs contained therein. Peritonitis is often caused by introduction of an infection into the otherwise sterile peritoneal environment through perforation of bowel, such as ruptured appendix or colonic diverticulum[1]. The disease may also be caused by introduction of a chemically irritating material, such as gastric acid from a perforated ulcer. Peritonitis secondary to perforation of the gastro intestinal tract, a common occurrence in this country, requires emergency surgical intervention and is associated with significant morbidity and mortality rates.

The first clinical description of perforated peptic ulcer was made by Crisp in 1843. Smoking and use of non steroidal anti inflammatory drugs are important risk factors for perforation[2]. Diagnosis is made clinically and confirmed by the presence of pneumoperitoneum on radiographs. Non operative management is successful in patients identified to have a spontaneously sealed perforation proved by water soluble contrast gastroduodenogram. Operative management consists of time honored practice of omental patch closure, but today this can be done by laparoscopic method. Laparoscopic approaches to closure of duodenal perforation are now being applied widely and may become gold standard in the future especially in patients with perforations less than 10 mm size presenting within the first 24 hours of onset of pain[5] .

Ileal perforation is a common surgical emergency in the tropical countries. It is reported to constitute the 5th commonest cause of abdominal emergencies due to high incidence of enteric fever and tuberculosis in these countries. Despite the availability of modern diagnostic facilities and advances in treatment regimens, this condition is associated with a high mortality and avoidable morbidity[8]**.** In the presence of advanced anaesthesia of today and tremendous improvement of resuscitative measures, every patient diagnosed to have ileal perforation is universally recommended to be treated

surgically. The purpose of operative protocol is to correct the pathology while avoiding any serious accidents and to adopt a surgical procedure which is associated with minimal complications.

Appendicitis was first recognized as a disease entity in 16th century and was called perityphilitis. McBurne in 1889 described the clinical features of acute appendicitis .Open appendicectomy is used since last century. In 1983, a German gynecologist Semm performed the first laparoscopic appendicectomy. Laparoscopic surgery is now a well established and advanced method of performing general surgical procedures. Acute appendicitis results from bacterial invasion usually distal to an obstruction of the lumen. The obstruction is caused by faecolith, seeds or worms in the lumen or by invasion of the appendix wall by parasites. Lymphoid hyperplasia following a viral infection has also been implicated. Untreated, the infection progresses to local peritonitis with formation of an appendicular mass, gangrene of the appendix, perforation and generalised peritonitis.

Acute mesenteric ischaemia, though a relatively rare condition, poses a particular surgical challenge because failure to diagnose it early results in death. It refers to a threatened or established ischaemic necrosis of the major part of the bowel and tends to occur in patients of either sex predominantly those above 50 years of age. The mortality from acute mesenteric ischaemia has not changed during the past two decades and unless we are aware of this entity and in the absence of an accurate diagnostic test, it is unlikely to change in future also. The presence of peritoneal signs mandates surgical exploration, as bowel infarction has probably occurred. Resection of the infarcted bowel as well as embolectomy can be accomplished during the process. In the absence of peritoneal signs, surgical embolectomy is still considered the standard of care. Foregoing surgical embolectomy in favour of a less invasive approach may be appropriate in a patient with appreciable surgical risk. In non occlusive mesenteric ischaemia infusion of an intraluminal vasodilator may be all that is needed to reverse vasoconstriction and prevent bowel infarction.

Colonic diverticular disease was first described in 1700 by Alexis Littre, a French surgeon. More than 90% of diverticula are left sided (sigmoid and descending colon) in western cultures, whereas isolated right colon diverticula are more commonly found in native Pacific Islanders. Crucial to the development of colonic diverticula is a relative lack of dietary fiber, as first described by Painter and Burkitt. Complications of the disease include acute diverticulitis with or without abscess/perforation, chronic diverticulitis (presenting as fistulization, stricture, or obstruction), and diverticular hemorrhage. Overall, complications occur in 15% to 20% of patients with diverticulosis. Diverticulosis is usually asymptomatic, and most often comes to the patient's attention after the clinician discovers it during endoscopic or radiographic evaluation of the colon. Treatment involves education of the patient in regard to the pathophysiology of the condition and its relationship to dietary fiber intake, and institution of a high-fiber diet, fiber supplement, or both. Acute diverticulitis complicated by gross perforation and peritonitis is a surgical emergency and is managed by aggressive volume resuscitation, broad-spectrum antibiotics, and laparotomy culminating in colonic resection and fecal diversion. Surgery in the setting of acute diverticulitis is indicated urgently in cases complicated by gross perforation, purulent peritonitis, or abscess untreatable by other means (antibiotics or percutaneous drainage). Acute diverticulitis with gross perforation is a surgical emergency requiring immediate laparotomy, involving resection and diversion the so called "Hartmann Procedure". Primary anastomosis is not safe in this setting due to inability to mechanically cleanse the bowel and eliminate local contamination. Cases of undrainable abscess or purulent peritonitis (resulting from free rupture of a pericolic abscess) are also managed by the Hartmann procedure.

AIMS AND OBJECTIVES

- Find out the incidence of perforation peritonitis with respect to age group and gender.
- The time of presentation to the hospital after the onset of symptoms.
- To find the relative frequency of anatomical site of perforation.
- To find the relative frequency of causes resulting in perforation.
- Analysis of various symptoms and signs with reference to their diagnostic value.
- Evaluation of reliability of investigation like plain X ray abdomen.
- Outcome with respect to complications, morbidity and mortality.
- Evaluation of sociodemographic factors on the aetiology, site of perforation and outcome.

REVIEW OF LITERATURE

HISTORICAL REVIEW [6, 7]

Surgeons have attempted for 100 years to cure the duodenal ulcer by reducing the secretion of acid and pepsin, and history of surgery for peptic ulcer is a chronicle of their attempts to achieve this aim without producing major disturbance to the functions of alimentary tract. Perforated peptic ulcers as a disease entity has been known since 1670.

1660: Littre (England) first described gastric ulcer perforation as the cause of death of daughter of Charles-I of England.

1726: George Hamberg (Germany) described a duodenal ulcer.

1727: Christopher Rawlinson (England) first described a case of perforated peptic ulcer.

1793: Jacopo Penada (Italy) first recorded a duodenal perforation.

1881: Ludwig Rydygier performed a successful resection of a prepyloric peptic ulcer.

1881: Theodor Billroth, Father of Surgical Audit and Father of Abdominal surgery, performed the excision of distal part of the stomach with ananastomosis of the gastric stump to the duodenum (Billroth I Surgery).

1886: Heineke did the first pyloroplasty.

1888: Mikulicz redefined the pyloroplasty done by Heineke.

1893: Barling, of Great Britain, treated perforated ulcer by closure and vigorous lavage of peritoneal cavity with large quantity of saline.

1893: Codivilla reportedly did the first gastrojejunostomy for a duodenal ulcer.

1896: Bennett suggested sealing a large perforation with omentum.

1899: Kently performed gastric resection for perforated peptic ulcer.

1937: Cellian-Jones and Graham popularized the effectiveness of omental patch for perforation.

1943: Dragsted and Owens introduced bilateral truncal vagotomy.

1948: Franksson of Stockholm first reported selective vagotomy.

1965: Erik Amdrup performed highly selective vagotomy.

1970: Robin Warren reported an association between Helicobacter pylori, gastritis and peptic ulcer perforation.

1985: Barry Marshall cultured Helicobacter pylori.

1985: Johansson B. Gilse H. described a laparoscopic technique for closure of perforated peptic ulcer.

1996: Halkic N. Pescatore P.and Gilleton combined both laproscopic–endoscopic methods using an omental plug for therapy gastroduodenal ulcer perforation.

Anatomy: [9, 10]

The peritoneum is the largest and the most complex serous membrane in the body. It forms a closed sac (i.e. coelom) by lining the interior surfaces of the abdominal wall (anterior and lateral), by forming the boundary to the retro peritoneum (posterior), by covering the extra peritoneal structures in the pelvis (inferior), and by covering the undersurface of the diaphragm (superior). This parietal layer of the peritoneum reflects onto the abdominal visceral organs to form the visceral peritoneum. Hence creating a potential space between the two layers otherwise known as peritoneal cavity.

The peritoneum consists of a single layer of flattened mesothelial cells over a loose areolar tissue. The loose connective tissue layer contains a rich network of vascular and lymphatic capillary channels, nerve endings, and immune competent cells, particularly lymphocytes and macrophages. The peritoneal surface cells are joined by functional complexes, thus forming a dialyzing membrane that allows passage of fluid and certain small solutes.

Peritoneal Cavity: [10]

This is the potential space between the parietal and visceral layers of peritoneum. This consists of –

- The greater sac or general peritoneal cavity.
- The lesser sac or the small omental bursa which is a diverticulum of the peritoneal cavity behind the stomach and adjoining structures. It opens into the greater sac through a slit like aperture the epiploic foramen.

Greater Omentum:

The greater omentum hangs down like a vascular apron from the greater curvature of the stomach, overlying coils of intestine. It is the most vascular part of the peritoneum, and is often called the 'policeman' of the abdomen, since it can move to a site of infection and become adherent to it, bringing protective leucocytes to the area of pathology and 'walling off' the inflammatory region. The greater omentum consists of two closely applied layers of peritoneum enclosing blood vessels and lymphatics (though strictly speaking it is four layers fused together). The greater omentum has a continuous attachment from abdominal oesophagus to duodenum, along the greater curvature of stomach. The part of the greater omentum immediately below the stomach overlies and fuses with the transverse mesocolon and transverse colon and is called as gastrocolic omentum.

Lesser omentum:

The two layers of peritoneum that extend from the liver onto the lesser curvature of stomach and the first inch of duodenum constitute the lesser omentum.

Peritoneal Compartments:

The peritoneum by virtue of its attachments to the posterior abdominal wall and to various viscera, divides the peritoneal cavity into compartments called

- Supracolic
- Infracolic and
- Pelvic

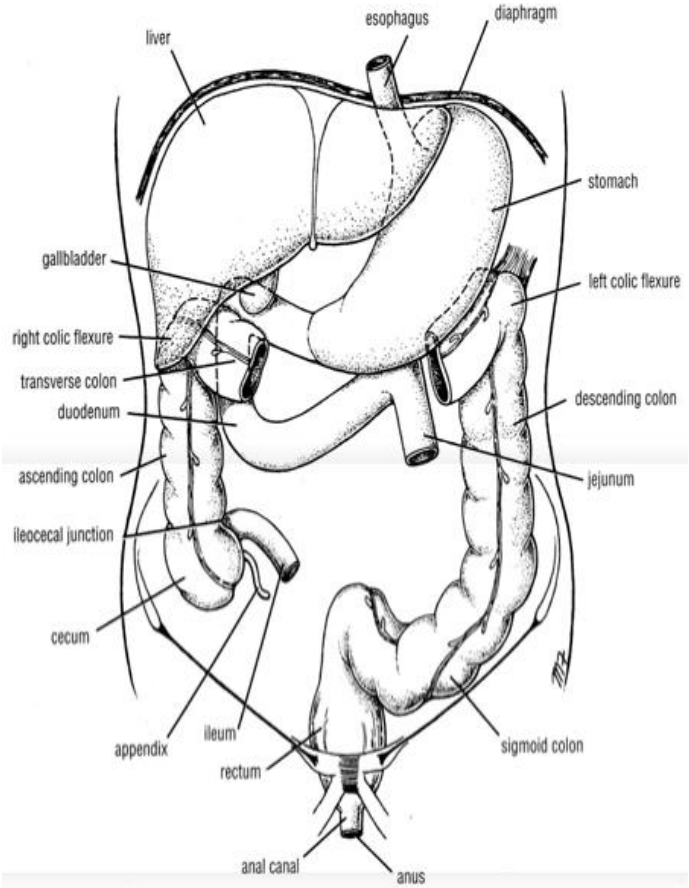


FIG 1: GENERAL ARRANGEMENT OF ABDOMINAL VISCERA

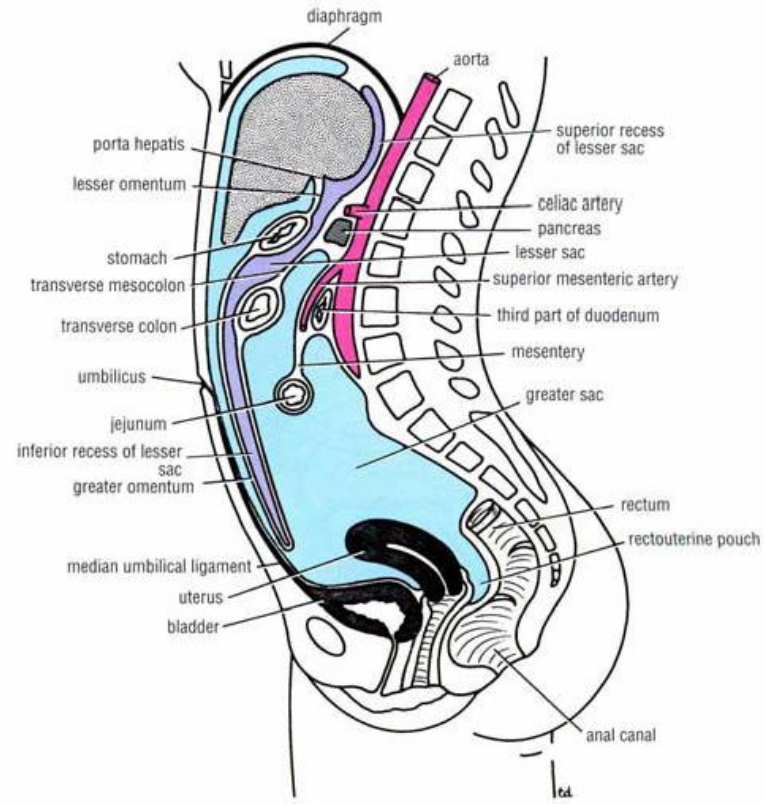


FIG 2: SAGITTAL SECTION SHOWING ARRANGEMENT OF PERITONIUM

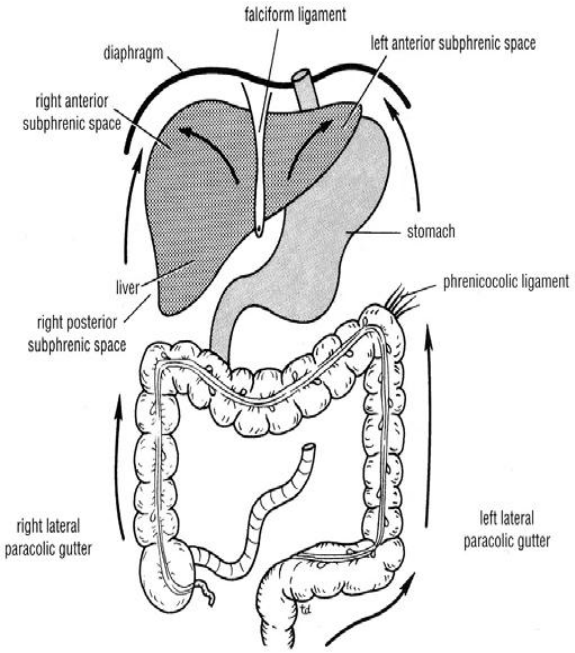


FIG 3: NORMAL DIRECTION OF FLOW OF PERITONEAL FLUID

The Supracolic compartment is subdivided into four compartments

- Right upper or right subphernic (sub diaphragmatic) compartment
- Right lower or hepatorenal pouch (of Morrison)
- Left upper or left Subphrenic (subdiaphargmatic) compartment
- Left lower or left subhepatic compartment.

The infracolic compartment has two parts – Right (upper) and Left (lower).

The dividing line between the supracolic and infracolic compartments is the attachment of the transverse mesocolon to the posterior abdominal wall.

When lying supine, the hepatorenal pouch is the lowest part of the peritoneal cavity (with the exception of the pelvis), and hence is an area where intraperitoneal fluid is likely to accumulate.

Nerve Supply:

Parietal peritoneum is supplied segmentally by the spinal nerves that innervate the overlying muscles. Thus the diaphragmatic peritoneum is supplied centrally by phrenic nerve (C4) and peripherally by intercostal nerves.The remainder of the parietal peritoneum is supplied segmentally by intercostals and lumbar nerves.

The visceral peritoneum has no afferent supply and pain is due to muscle spasm, tension on mesenteric folds or involvement of the parietal peritoneum.

Stomach: [9, 10]

The stomach is the most dilated part of the alimentary tract, interposed between the oesophagus and duodenum in the upper part of abdominal cavity and lying mainly in the left hypochondria, epigastric and umbilical region. Its means capacity varies from 30 ml at birth, but in the adult it may accommodate upto1500 ml or more.

The junction of stomach with the oesophagus is the cardia and lies under the diaphragm, to the left of the midline at the level of T-10 vertebrae.

The distal opening is the pyloric opening, at the gastroduodenal junction. It is about 1.2 cm to the right of the midline in the transpyloric plane, with the body supine and the stomach empty.

The main parts of the stomach are the fundus, body and pyloric part, with the greater and lower curvatures forming the upper and lower borders and joining the anterior and posterior surfaces.

Fundus is the part which projects upwards above the level of the cardia.

The body extends from the fundus to the angular notch (incisura angularis) of the lower part of the lesser curvature.

The pyloric part extends from the angular notch to the gastroduodenal junction, and consists of the proximal pyloric antrum which narrows distally as the pyloric canal. The circular muscle of the distal end of the canal is palpably thickened to form the pyloric sphincter, whose position is indicated on the anterior surface by the prepyloric vein.

Blood Supply:

There are four main arteries supplying to the stomach –

- The left gastric artery arises from the coeliac axis
- The right gastric artery arises from the common hepatic artery.
- The left gastro-epipolic artery arises from the splenic artery.

Blood Supply of the Greater Omentum:

This comes from the right and left gastro epipolic arteries which form an arcade along the greater curvature of the stomach. When mobilizing the greater omentum from the greater curvature of the stomach, it is not necessary to preserve the gastro-epipolic arcade provided the epipolic arcade is preserved.

Venous Drainage:

Veins of the same name accompany the arteries (except that there is no gastroduodenal vein) and drain into the portal vein itself or its splenic and superior mesenteric tributaries. Prepyloric vein (without an accompanying artery) drains into the portal or right gastric veins.

Lymphatic Drainage:

All lymph eventually reaches coeliac nodes after passing through various outlying groups.

Cardiac and most of lesser curvature: left gastric nodes.

Pylorus and distal lesser curvature: right gastric and hepatic nodes.

Proximal portion of the greater curvature: pancreaticosplenic nodes in the splenic hilum.

Distal portion of the Greater curvature: right gastroepipolic nodes in greater Omentum and pyloric nodes at the head of pancreas.

Nerve Supply:

Sympathetic fibres (vasomotor) accompanied by afferent (pain) fibers run with the various arterial branches to the stomach.

The parasympathetic supply is from the vagi which control motility and secretion. The anterior vagal trunk (from the oesophageal plexuses lies in contact with the anterior oesophageal wall, usually nearer its right margin. The anterior nerve of Latarjet, is the termination of he left vagus trunk as it innervates the anterior gastric wall. As the nerve of Latarjet reaches its termination, it divides into 4 or 5 branches in a configuration that resembles a crow's foot. These terminal branches innervate the distal 6-7 cm of the antrum and pylorus.

The posterior vagal trunk lies in a loose tissue a little behind and to the right of the right oesophageal margin. It runs in the lesser omentum behind the anterior trunk (as the posterior nerve of Latarjet), giving off a large celiac branch that runs along with left gastric artery to the coeliac ganglion, and numerous branches to the posterior surface of stomach.

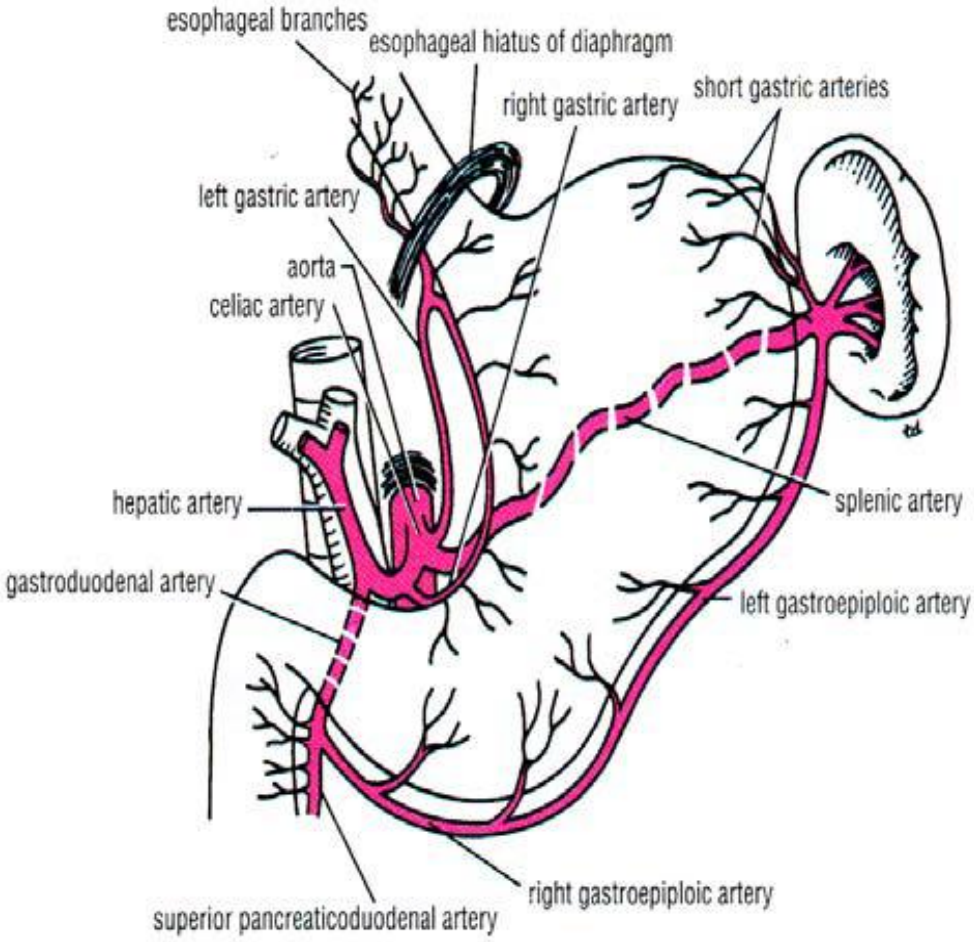


FIG 4: ARTERIAL SUPPLY OF STOMACH

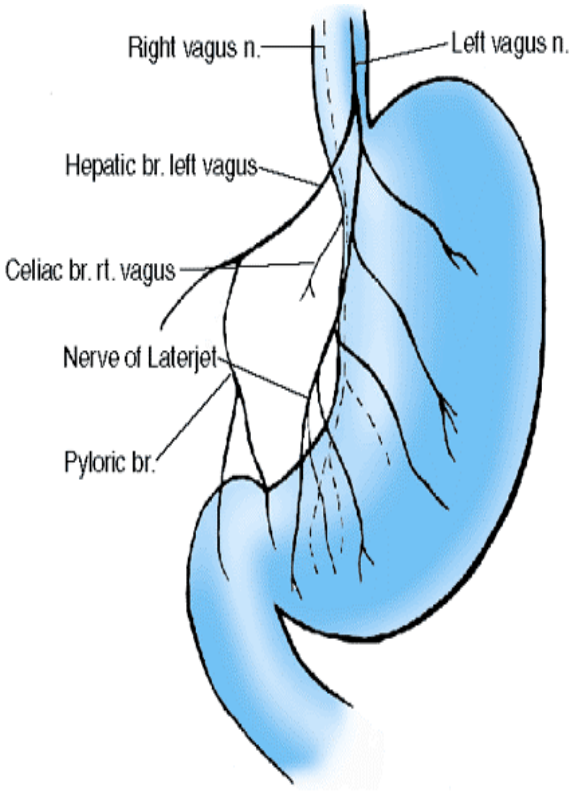


FIG 5: NERVE SUPPLY OF STOMACH

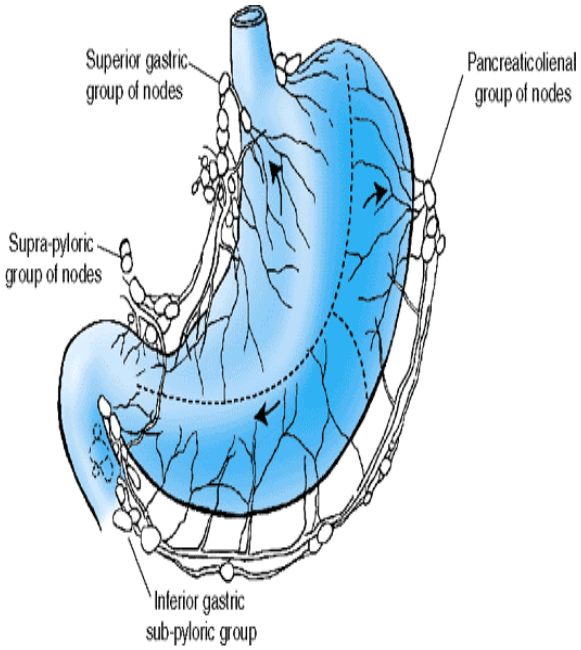


FIG 6: LYMPHATIC SUPPLY OF STOMACH

Small Intestine:

The small intestine consists of the duodenum, jejunum and ileum, and extends from the pylorus to the ileocaecal valve.

Duodenum:

The duodenum is a C-shaped tube curved over the convexity of the forwardly projecting aorta and inferior vena cava. The first 2 cm are contained between the peritoneum, but the remainder is retroperitoneal. It is divided into
4 parts:

- First (Superior): about 5 cm long
- Second (Descending): about 7.5 cm long
- Third (Horizontal): about 10 cm long
- Fourth (Ascending): about 2.5 cm long.

It is the peritoneal attachment which distinguishes duodenum and jejunum. The duodenum is retroperitoneal, the jejunum has a mesentry. The duodenojejunal flexure is fixed to the left psoas fascia by fibrous tissue. It is further supported by the suspensory muscle of the duodenum (ligament of Treitz).

Blood supply:

The duodenum is supplied by the superior and inferior pancreaticoduodenal arteries, but the first 2 cm, receive small branches from a variety of sources; hepatic, common hepatic, gastroduodenal, superior pancreaticoduodenal, right gastric and right gastroepiploic.

Venous drainage drain into splenic, superior mesenteric and portal veins.

Lymphatic drainage:

Duodenal lymph drains to coeliac and superior mesenteric nodes.

Jejunum and Ileum:

The jejunum and ileum together lie in the free margin of the mesentry. Total length varies from about 4 to 6 meters. The jejunum constitutes rather less than half the total length. The jejunum lies coiled in the upper part of the infracolic compartment, the ileum in the lower part thereof and in the pelvis.

The jejunum is wider, broader, and thicker walled than the ileum. An ileal (Meckel's) diverticulum is present is 2% individuals, 60cm(2 ft) from the caecum and is 5 cm (2 inch) long. It represents the intestinal end of the vitellointestinal duct.

Blood Supply:

Jejunal and ileal branches arise from the left side of the superior mesenteric artery and enter the mesentry. The jejunal branches join each other in a series of anastomosing loops to form arterial arcades. From the arcades straight arteries pass to the mesenteric border of the gut.

The ileal arteries form a larger series of arcades three to five in number and there is more fat in their part of the mesentry.

The veins all correspond to the arteries and they drain to the superior mesenteric vein.

Lymphatic Drainage: Jejunal and ileal lymph drains to superior mesenteric nodes.

Large Intestine:

The large intestine consists of the caecum with the worm- shaped (Vermiform) appendix, the ascending, transverse, descending and sigmoid parts of the colon, the rectum and the anal canal.

Caecum and Appendix:

Caecum is a blind pouch of the large intestine and projects downwards from the commencement of the ascending colon, below the ileocaecal junction.

It's average length is about 6 cm and breadth about 7.5cm. The longitudinal muscle of the caecum is concentrated into three flat bands, the taeniaecoli, between which the circular muscle layer constitutes the sacculated wall of the gut. Internally the ileocaecal junction is guarded by the ileocaecal valve.

The appendix is a worm shaped, blind ending tube varying in length from 2 to 25cm, which opens into the posteriomedial wall of the caecum 2 cm below the ileocaecal valve. The base of the appendix is at the point of convergence of the three taeniae coli. It may occupy one of the several positions. It is connected by a short mesoappendix to the lower part of the ileal mesentry.

TABLE -1

POSITION AND PERCENTAGE OF THE APPENDIX

Positions	Percentage
Retrocolic and Retrocaecal	65.25%
Pelvic	31%
Subcaecal	2.25%
Pre-ileal	1%
Post ileal	0.4%

The appendicular artery is normally a branch of the posterior caecal artery reaches the appendix in the free margin of the mesoappendix and then courses to the appendicular wall.

Colon:

Consists of four parts: ascending, transverse, descending and pelvic colon. Of this the transverse and sigmoid are suspended in mesentery but the ascending and descending colon are plastered on to the posterior abdominal wall, so that they have posterior 'bare areas' devoid of peritoneum.

Ascending Colon:

This first part of the colon, about 15cm in length extends upwards from the ileocaecal junction to the right colic flexure. The taeniae coli lie, in line with those of the caecum, anteriorly, postero laterally and postero medially.Bulbous pouches of peritoneum, distended with fat, the appendices epiploicae, project in places from the serous coat.

The blood vessels supplying them from the mucosa perforate the muscle wall. Mucous membrane may herniate through these vascular perforations, a condition known as diverticulosis.

Transverse colon:

About 45 cm long extends from hepatic to splenic flexure in a loop which hangs down to a variable degree between these two fixed points. It is completely invested in peritoneum. The appendices epiploicae are larger and more numerous than on the ascending colon.

Descending colon:

Less than 30 cm long, this extends from the splenic flexure to the pelvic brim and in the whole of its course is plastered to the posterior abdominal wall by peritoneum. The descending colon is smaller in calibre than the ascending colon.

Sigmoid Colon:

This extends from the descending colon at the pelvic brim to the commencement of the rectum in front of the third piece of the sacrum. It is completely covered by peritoneum and hangs free on a mesentery sigmoid mesocolon. The sigmoid possesses well developed appendices epiploicae.

Blood Supply:

Ascending colon and proximal two thirds of the transverse colon are supplied by ileocolic, right colic and middle colic branches of the superior mesenteric artery.

Rest of the colon is supplied by left colic and sigmoid branches of the inferior mesenteric artery. The marginal artery of Drumond is the paracolic vessel of anastomosis between colic arteries from which arise the terminal arteries to colon (Vasa Recta). It lies 2.5-3.8cm from the bowel wall.

The veins correspond to the arteries and reach the portal vein via the superior or inferior mesenteric tributaries.

Lymph Drainage:

The lymph channel follows the blood vessels so that drainage is to superior or inferior mesenteric nodes.

Nerve Supply:

Up to the splenic (derived from midgut) and from there onwards (hindgut), the parasympathetic supply is partly from the vagi and partly pelvic splanchnic nerves.

Sympathetic supply is derived from spinal cord segments T10-L2. The pain fibres that accompany these vasoconstrictor nerves give rise to periumbilical pain if from midgut derivatives (e.g. appendix) but to hypogastric region if from hind gut.

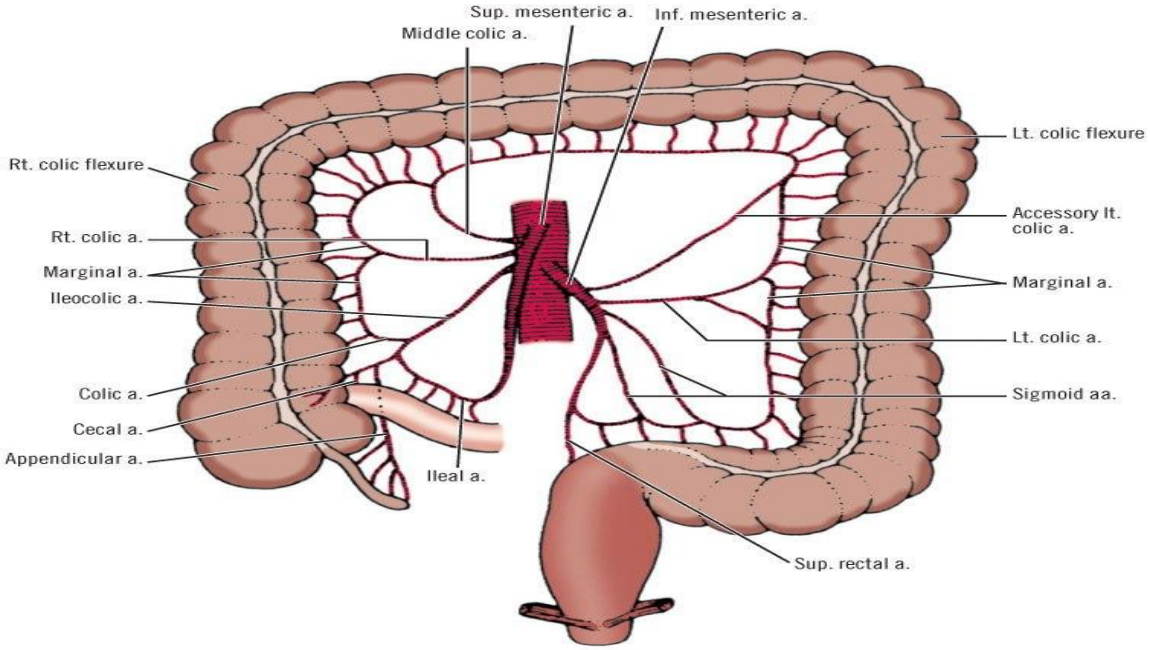


FIG 7: ARTERIAL SUPPLY OF INTESTINE

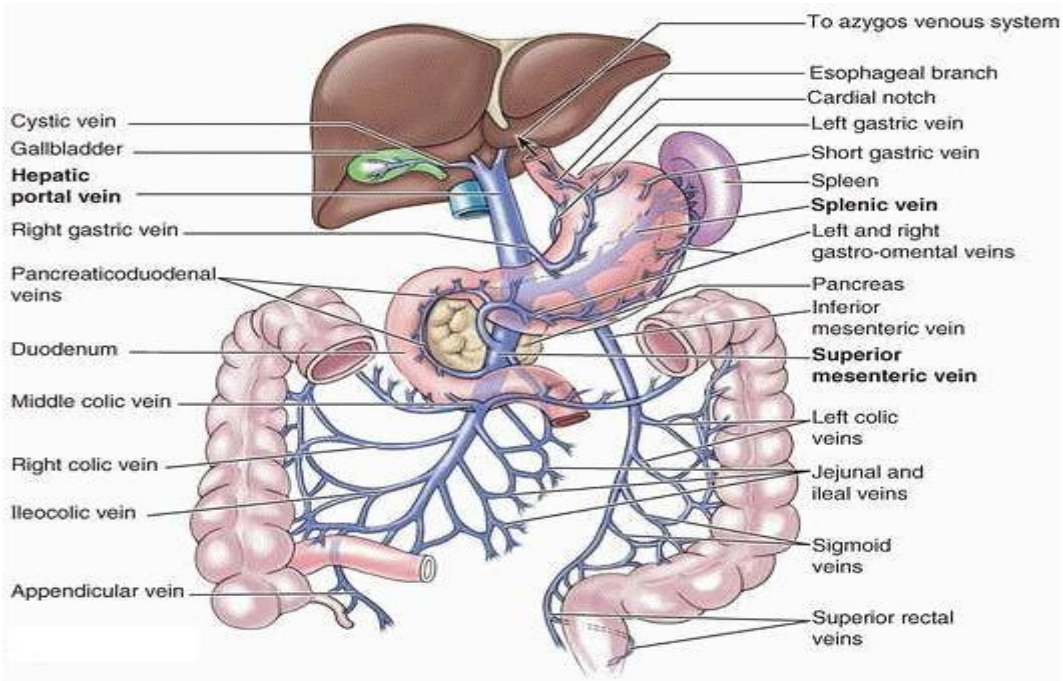


FIG 8: VENOUS SUPPLY OF INTESTINE

Physiology of the peritoneal cavity: [12]

The peritoneum is a single layer of mesothelial cells, with a basement membrane supported by an underlying layer of highly vascularised connective tissue.[13] The surface

area of the peritoneum is extensive, averaging 1.8m^2 (adult male) and is comparable to the surface area of the skin.[14] It has been estimated that a 1mm increase in the thickness of the peritoneum can result in the sequestration of 18 litres of fluid, a fact relevant to the massive fluid shifts associated with diffuse peritonitis.[15]

Under normal condition, < 50 ml of sterile fluid is present within the peritoneal cavity secreted from the visceral peritoneal surfaces; the fluid is circulated through the peritoneal cavity. The cephalad movement proceeds along the paracolic gutter and subhepatic spaces – due to negative pressures in the subphrenic area by diaphragmatic motion. Peritoneal fluid is mostly absorbed into the lymphatic circulation via the parietal peritoneal surfaces, with the remainder absorbed through diaphragmatic lymphatics [16] .The clearance of particulate matter, cells and microorganisms is largely dependent upon diaphragmatic lymphatics.[17]

The diameter of these lymphatic stomata can be varied by diaphragmatic stretching and contraction, from 4 to 12 microns.18,19 In addition, in the presence of inflammation the patency of stomata may be increased to augment the clearance function of the diaphragm.20 At inspiration, contraction of the diaphragm empties the lacunae into efferent lymphatic channels. Negative intrathoracic pressure during inspiration facilitates fluid movement into thoracic lymphatic channels, and ultimately delivered to the central circulation via the thoracic duct. Following the intraperitoneal injection of bacteria, organisms can be recovered from right thoracic duct within 6 min and from blood within 12 minutes. [21]

A number of factors can influence this diaphragmatic clearance mechanism or "pump" –

- Blockage of the stomata by platelets or talc
- Head up position delays appearance of bacteria in the circulation.[21]
- Reducing spontaneous respiration using general anesthesia.[22]
- Application of positive end expiratory pressure.[23]

Clinical observation suggests that the mortality from peritonitis is reduced in patients placed in the semi upright position probably decreases bacterial absorption via the diaphragm. [24, 25]

The second clearance mechanism is by phagocytosis by resident peritoneal macrophages.

Local response to peritoneal infection:

The inflammatory response that occurs within the peritoneal cavity, characterized by hyperemia, the influx of fluid, recruitment of phagocytic cells and fibrin deposition. Any noxious stimulus like endotoxin associated with gram negative bacteria, gram positive bacteria, bacteroides species, irritants such as gastric juice, bile salts and meconium probably incite the inflammatory process by inciting mesothelial cell damage or direct activation of the complement system.

Following activation the peritoneal inflammatory process is composed of changes in blood flow, the enhancement of bacterial phagocytosis and fibrin deposition to contain or trap bacteria.

Systemic response to peritoneal infection:

The systemic response to peritoneal infection emulates the response of the body to other forms of injury such as trauma or surgery. The development of hypovolaemia is a phenomenon central to the systemic response and probably results from the fluid influx occurring in the peritoneal cavity. The subsequent intravascular volume change leads to a

reduction in venous return and cardiac output. Systemic hypotension also may be the result of the secretion of TNF, IL-1, platelet activating factor and nitric oxide.[26,27] Diminished urine flow develops as a result of the effects of increased aldosterone and anti diuretic hormone secretion, reduced cardiac output and interrenal shunting of blood. This is the setting that has been dubbed as "warm" septic shock, characterized by tachycardia, fever, oliguria, hypotension and warm extremities.

Abdominal distention secondary to accumulated fluid within the peritoneal cavity – creates restriction to diaphragmatic mobility and decreases ventilatory volume, creating eventual atelectasis. The accumulation of fluid in the pulmonary interstitium and alveoli decreases pulmonary compliance and decreased work of breathing. Early manifestation is hyperventilation and the development of respiratory alkalosis. With the worsening of the pulmonary edema and alveolar collapse; severe hypoxemia will develop, creating the adult respiratory distress syndrome (ARDS).

Tissue metabolism is severely altered during the response to peritonitis.Tissue hypoxia leads to anaerobic glycolysis leading to metabolic acidosis. The severe loss in the lean body mass that can occur from protein catabolism occurs rapidly and is only partially ameliorated by the use of nutritional support.

Classification, Etiopathogenesis and Pathology: [4]

Peritonitis is classified into three divisions based upon the source and nature of microbial contamination**.**

I. Primary Peritonitis

A) Spontaneous peritonitis of childhood

B) Spontaneous peritonitis of adult

C) Peritonitis with continuous ambulatory peritoneal dialysis

D) Tuberculosis peritonitis

II. Secondary Peritonitis

A) Perforation peritonitis

a. Gastrointestinal tract perforation

b. Pelvipertonitis

c. Peritonitis after translocation of bacteria

B) Postoperative peritonitis

a. Leak of an anastomosis

b. Leak of suture line

c. Stump insufficiency

III. Tertiary peritonitis

I. Primary Peritonitis:

It is defined as an infection, often monomicrobial, of the peritoneal fluid without visceral perforation.

II. Secondary Peritonitis:

It refers to peritoneal infection arising from an intra-abdominal source, majority of these episodes are the result of primary lesions of the stomach, duodenum, small intestine, colon and appendix.[28] It is by far the most common form of peritonitis.

III. Tertiary peritonitis:

It develops following the treatment of secondary peritonitis and represents either a failure of the host inflammatory response or a super infection.

Perforative peritonitis is defined as the end result of a disease process of trauma which extends through the muscular and serosal walls of the gastrointestinal tract, establishing a communication between the lumen of the viscus and the surrounding body cavity and permits free egress of the luminal contents into the cavity.

TABLE -2

CAUSES OF PERFORATIVE PERITONITIS[2]

Source Regions	Causes
Stomach	Peptic ulcer perforation Malignancy (e.g.Adenocarcinoma, lymphoma, gastrointestinal stromal tumor)
Duodenum	Peptic ulcer perforation
Small bowel	Salmonella enteritis Ischemic bowel, Crohn's disease. Meckel diverticulum, intestinal tuberculosis Incarcerated hernia (internal and external) Parasiticperitonitis due to perforation by round worm Closed loop obstruction Malignancy (rare)
Large bowel & Appendix	Ischemic bowel, Diverticulitis, Malignancy, Ulcerative colitis and Crohn's disease, Appendicitis, Colonic volvulus and Amoebic colitis.

History:

Until the end of last century, the intraabdominal infections were treated nonoperatively with a mortality of 90%. Surgical principles were enunciated during the first two decades of this century and have been uniformily applied in the management of peritonitis since 1930. The principles which have by and large remained unchanged are;

i) Elimination of the source of infection and

ii) Removal of the infected material from the peritoneal cavity.

With widespread application of these principles to the treatment of peritonitis, the mortality came down to 40-50%.[29] The further trends of decline in the mortality became visible in 1970's and 1980s. This drop is attributed to the better understanding of the bacteriology of the disease, availability of powerful antibacterial agents against both aerobes and anaerobes seen in peritonitis and better understanding of organ system dysfunction in sepsis and efficient multisystem support the I.C.U. setup.

The declining trend appears to have reached a plateau with the emergence of new problems.For example the problems of microbial resistance in compromised host resulting in peritonitis which may be resistant to number of antimicrobials.[7] Such a scenario is attended upon by a higher mortality.

Bacteriology of Peritonitis:

The insight gained into the bacterial etiology of disease has resulted in significant advances in the antimicrobial therapy of the disease. Although most of the bacteriologic etiology of peritonitis was identified by Freidrich and Heyde in 1920s, the important role of anaerobes remained obscure to most surgeons until 1970s.[30]

The bacteria released into the peritoneal cavity following perforation of a hollow viscus cause secondary peritonitis. The two important facts that have paramount bearing on the treatment of peritonitis are the polymicrobial nature of the infection and mixed

aerobic anaerobic pathogens occurring as the commonest offending bacteriologic combination.[31, 32, 33]

Antibiotic Selection:

When selecting an antibiotic for the patient of peritoneum, the following consideration should be kept in mind –

1) It should be directed against the wellknown typical spectrum of aerobic and anaerobic organisms.
2) It should achieve effective concentration in the blood and peritoneal fluid.
3) It should be safe and devoid of serious toxicities and
4) Should be backed by the results of valid clinical trials.[7]

For most cases of community acquired bacterial peritonitis i.e. appendicitis, diverticulitis, perforated ulcer disease, monotherapy with an agent active against both aerobes and anaerobes is the preferred choice. [34, 35]

PEPTIC ULCER DISEASE:

Epidemiology:

Peptic ulcer disease remains one of the most prevalent and costly gastrointestinal diseases. Elective admission has decreased dramatically while admissions for complications related to ulcer disease have shown little change.[36,37] Peptic ulcer disease has decreased in men and increased in women.[36] Although the reason for the decrease in men is unknown, it may reflect the decrease in smoking among men. It is speculated that the increase in women with peptic ulcer disease was in past due to an increase in smoking and at present due to an increase in NSAID ingestion. On the other hand there has been a consistent increase in the age of the population affected by perforated peptic ulcer in virtually every study worldwide.

H.pylori infection represents the most drastic change in our understanding of peptic ulcer disease and has led many experts to conclude that peptic ulcer disease is in

reality an infectious disease. Moreover, there is a high recurrence rate for peptic ulceration following discontinuation of medical therapy. Thus, there is a renewed interest in operative management of patients suffering from peptic ulcer disease. Although the indications for surgery have not changed dramatically over the last several decades i.e. perforation, bleeding, obstruction, the type of operation has changed in the H.pylori era.[38, 40]

However, recent studies indicate that vagotomy may not even be necessary in some situations such as perforation of the duodenum, provided that H.pylori is eradicated.[40]

Location and type of ulcer:

Peptic ulcer disease can be divided into gastric and duodenal ulcers. Both types tend occur near mucosal junctions. For example, duodenal ulcers usually occur at the duodenal pyloric junction, whereas gastric ulcers tend to occur at the oxyntic antral junction, the antral pylori junction. Duodenal ulcer disease is a disease of multiple etiologies.[41] The only absolute requirements are secretion of acid and pepsin in conjunction with either H.Pylori infection or ingestion of NSAIDS.

Classification of benign gastric ulcers (after Johnson, 1957)

Acute superficial:

- Single or multiple (erosions)

Chronic:

- Type I, usually lesser curve.
- Type II, combined with duodenal ulcer.
- Type III, prepyloric
- Type IV, proximal stomach < 2 cm from oesophageal junction.

PATHOGENESIS:

Helicobacter pylori infection:

Warren and Marshall were the first to identify and isolate the organism for which they received Nobel prize.[42] The organism was found to be a spiral or helical gram-negative rod with 4 to 6 flagella that resided in the gastric type epithelium within or beneath the mucus layers, which protected it from acid. It is now believed that 90% of duodenal ulcers and roughly 75% of gastric ulcers are associated with H.Pylori infection.

Three potential mechanisms for H.pylori induced gastrointestinal injury have been proposed.

1) Production of toxic products to cause local tissue injury.
2) Induction of a local mucosal immune response.
3) Increased gastrin levels with a resultant increase in acid secretion.

The gastric mucosa barrier is distributed by the production of an endopeptidase a powerful mucolytic and by the generation of large amounts of ammonia with an increase in the epithelial surface pH. The latter alters the mucosal charge gradient, cellular permeability and epithelial Na+ K+ - ATPase activity leading to back diffusion of H+.[43] It also causes a local inflammatory reaction in the gastric mucosa and produce chemotactic factors that attract neutrophils and monocytes.[44]

Drugs:

After H.pylori infection, ingestion of NSAIDS is the most common cause of peptic ulcer disease. The increased risk of bleeding and ulceration is proportional to the daily dosage of NSAID. Consequently, the ingestion of NSAIDS remains an important factor in ulcer pathogenesis, especially in relationship to the development of complications and death. NSAIDS increase the risk of gastrointestinal complications approx. 2 to 10 fold. NSAID ingestion not only causes acute gastroduodenal injury but is also associated with chronic gastroduodenal injury. This risk of mucosal injury and or ulceration is roughly proportional to the anti-inflammatory effect associated with each NSAID.[45]

The presence of chronic epigastric pain is more suggestive of ulceration. The acute gastroduodenal lesions typically appear within 1-2 weeks of ingestion, whereas chronic injury typically occurs after 1 month. Again ulcer risk is dose related. In comparison to H.pylori ulcer frequently found in the duodenum. NSAID induced ulcers are more frequently found in the stomach.

The increase in perforation in the elderly might largely be due to widespread use of NSAID in this group. There was a significant association between perforation associated with NSAID use and the lack of need for subsequent definitive surgical treatment.[46]

Corticosteroids have similarly been implicated, and the association with perforation appears just as strong.

Stress:

Psychological stress has been implicated in the etiology of peptic ulcer. Eversince Beaumont's classic observations, a subsequent study from Belfast failed to show any association between psychological stress and perforation of peptic ulcer.

Pathology: [47, 48, 49]

Approximately 98-99% of peptic ulcer occurs in either the duodenum or the stomach at a rate of 4:1. At least 98% of peptic ulcers are located in the first portion of the duodenum or in the stomach. Most duodenal ulcers are generally within a few centimeters of the pyloric ring. The anterior wall of the duodenum is more often affected than the posterior wall; gastric ulcers are located along the lesser curvature. Majority of individuals have a single ulcer. In 10-20% of patients with gastric ulceration there may be coexistent duodenal ulcer.

Peptic ulcers are usually round in shape sharply punched out defects in the mucosa that penetrate at least into the sub mucosa, usually into the muscularis and some times

more deeply. Most are 2-4 cm in diameter; those in the duodenum tend to be smaller. The mucosal margins of the crater are perpendicular and there is some mild edema of the adjacent mucosa. Heaping up of these margins is rare in the benign ulcer but is characteristic of malignant lesion. The base of the ulcer is smooth and clear owing to peptic digestion of any exudates. Scaring may involve entire thickness of the stomach; puckering of the surrounding mucosa creates mucosal folds, which radiate from the crater in spoke like fashion.

Ulceration of the small intestine is a lesion of multifactorial origin. In the tropics; typhoid fever remains the commonest cause of non traumatic ileal perforation.[50,51,52,53,54] Other causes included tuberculosis, amoebiasis, ascariasis and non-specific illness in comparison to west where strangulation of the bowel, diverticula, and foreign bodies, Crohn's disease and radio therapy are common.[55,56,57]

Typhoid Enteritis: [58]

Typhoid enteritis is an acute systemic infection of several weeks'duration caused primarily by Salmonella typhosa. The pathologic events of typhoid fever are initiated in the intestinal tract after oral ingestion of the typhoid bacillus. These organisms penetrate the small mucosa, making their way rapidly to the lymphatics and then systemically hyperplasia of the reticuloendothelial system, including lymph nodes, liver and spleen is characteristic of typhoid fever. Peyer's patches in the small bowel become hyperplastic and may subsequently ulcerate with complications of hemorrhage or perforation. Perforation usually takes place in the 2nd – 3rd week of illness with gradual onset in comparison to that of peptic ulcer and is seen in only 2% of cases.

Pathology: [39, 59]

The terminal ileum bears the brunt of the intestinal infection. Typhoid ulcer is an oval mucosal defect with the long dimension in the axis of the bowel and usually situated in the terminal ileum. There will be hyperplasia and ulceration of the Peyer's patches of the intestine, mesenteric lymphadenopathy and splenomegaly.

Tuberculosis of the intestine: [60, 61, 62]

Tuberculosis accounts for approximately 6% of annual deaths worldwide. Resurgence in the incidence of tuberculosis has occurred in Western countries as a result of AIDS, influx of Asian immigrants and widespread use of immunosuppressive agents after organ transplantation.

The structures involved in abdominal tuberculosis are-

1. Peritoneum
2. Intestines
3. Mesenteric lymph nodes.

INTESTINAL TUBERCULOSIS

Gastrointestinal tuberculosis forms the bulk of what goes by the name of abdominal tuberculosis. It is a localized manifestation of a generalized disease and occurs in two forms-

Primary:

Infection is usually caused by bovine strain of the mycobacterium. and results from ingesting infected milk. It accounts for the 10% of the reported cases.

Secondary

This constitutes majority of the cases and occurs due to swallowing of the sputum containing bacilli by patients with active pulmonary tuberculosis. Ileocaecal and jejunoileal areas are the commonest sites of involvement of gastrointestinal tract. The apparent affinity of tubercle bacilli for lymphoid tissue and areas of physiological stasis make ileocaecal area as the most common site of the disease. There are several mechanisms by which tubercular enteritis may occur

1. From the swallowing of infected sputum in active pulmonary tuberculosis.
2. Ingestion of contaminated milk (bovine strain)

3. By haematogenous spread from active pulmonary tuberculosis, military tuberculosis or silent bacteraemia during primary phase of tuberculosis. Direct extension from adjacent organs (rare).

Active inflammation takes place in submucosa and serosa resulting in thickening because of edema, cellular infiltration, lymphoid hyperplasia, tubercle formation and later on, fibrosis. Mucosal ulcers multiple and transversely placed in terminal ileum, may occur as a result of endarteritis of submucosal vessel. Gross pathological appearance has led to its traditional categorization into four forms that include are

(i) **Ulcerative** (usually in terminal ileum)

(ii) **Hypertrophic** or **hyperplastic** (usually ileocaecal region in patients with high resistance to the organisms.)

(iii) **Ulcerohypertrophic**

(iv) **Ulceroconstrictive**.

Amoebiasis: [47, 59]

Commonest parasitic infestation in the world. It is a disease associated with poor hygiene and as such is more common in the underdeveloped and developing countries in the tropics. This is an infective disease due to the infestation with entamoeba histolytica. It is transmitted mainly in the contaminated drinking water. The ulcers are described as 'bottle necked' because of their undermined edges. The ulcers have yellow necrotic floor, from which blood and pus exude. The most common sites for perforation in amoebiasis are caecum and recto sigmoid area. Usually perforation occurs into a confined space where adhesions have previously formed and a pericolic abscess results. Sometimes a sudden faecal flooding of the general peritoneal cavity occurs. Caecum is more affected than sigmoid colon.

Pathogenesis:

In the lifecycle of E. histolytica 'cyst' is the infective form. The trophozoite phase is responsible for producing characteristic lesions of amoebiasis. Trophozoite enter through the crypts of Lieberkuhn and penetrate directly through the columnar epithelium by their amoeboid activity and by dissolving intestinal epithelial cells with a proteolytic ferment they secrete. They gradually burrow into the submucous coat and form colonies there. With destruction of tissues around the colonies, ulcer develops.

Ascariasis: [61, 62]

Ascarias lumbricoides is the largest intestinal nematode parasite worm in man. The worm has no intermediate host, the infection being transmitted from man to man. Its association with man is as old as the human species itself. Ascaridia infestation is a formidable problem in the younger age groups in some tropical and subtropical countries. The usual habitat of the parasite is the jejunum through it may also be found in the distal reaches of the small intestine and colon.

Pathogenesis:

Ascariasis by itself may not produce ulceration or perforation of the healthy gut. It usually burrows and penetrates through a pre-existing ulcer. At times, the inflammatory edema and the softening of a segment due to long leftover worm mass may lead to weak spots through which the worms extrude.

Acute Appendicitis: [63, 64]

Although appendicitis has been a common problem for centuries, it was not until the early 19th century that appendix was recognized as an organ capable of causing disease. In 1886 Reginald Fitz made a landmark contribution by discussing the appendix as the primary cause of right lower quadrant inflammation. Appendicitis occurs frequently in very young children and elderly persons. The disease has a maximal incidence in patients in their late teens and 20s. There is a slight increased prevalence in males versus females. Currently the mortality rate is 0.25% if all ages are considered.

Pathophysiology:

The inciting event in most instances of appendicitis is obstruction of the appendicular lumen. This may be due to lymphoid hyperplasia, inspissated stool (a fecolith) or some other foreignbody. Obstruction of the lumen leads to bacterial overgrowth as well as continued mucus secretion. This causes distension of the lumen, and the intraluminal pressure increases. This may lead to lymphatic and then venous obstruction. With bacterial overgrowth and edema, an acute inflammatory response ensues. The appendix then becomes more edematous and ischemic necrosis of the appendiceal wall subsequently occurs with translocation of bacteria through the ischemic wall. This is gangrenous appendicitis. Without intervention the gangrenous appendix will perforate with spillage of the appendiceal contents into the peritoneal cavity. If this sequence of events occurs slowly, the appendix is contained by the inflammatory response and the omentum, leading to localized peritonitis and eventually an appendiceal abscess. If the body does not walloff the process, the patient may develop diffuse peritonitis.

Mesenteric Ischemia: [65]

"Occlusion of the mesenteric vessels is apt to be regarded as one of those conditions of which the diagnosis is impossible, the prognosis hopeless and the treatment almost useless" (Cokkinis, 1926). This quote indicates some of the extreme difficulties faced by physician treating acute mesenteric ischaemia. Symptoms are non specific initially before evidence of peritonitis presents. Thus, diagnosis and treatment are often delayed until the disease is far advanced.

AMI is a syndrome in which inadequate blood flow though the mesenteric circulation causes ischemia and eventual gangrene of the bowel wall. The syndrome can be classified as arterial or venous disease –

1. Acute mesenteric arterial embolus (AMAE).
2. Acute mesenteric arterial thrombosis (AMAT)

3. Non occlusive mesenteric ischemia (NOMI)
4. Occlusive mesenteric arterial ischemia (OMAI)

Pathophysiology:

Insufficient blood perfusion to the small bowel and colon may result from arterial occlusion by embolus or thrombosis (AMAE or AMAT), thrombosis of the venous system (MVT) or non occlusive processes such as vasospasm or low cardiac output (NOMI). Embolic phenomena account for approximately 50% of all cases, arterial thrombosis for about 25%, NOMI for rough 20% and MVT for less than 10%. Hemorrhagic infarction is the common pathologic pathway whether the occlusion is arterial or venous .The superior mesenteric vessels are involved more frequently than the inferior mesenteric vessels, with blockage of the latter often being silent because of better collateral circulation.

Damage to the affected bowel may range from reversible ischemia to transmural infarction with necrosis and perforation. Arterial insufficiency causes tissue hypoxia, leading to initial bowelwall spasm. This leads to gut emptying by vomiting or diarrhea. Mucosal sloughing may cause bleeding into the gastrointestinal tract. At this stage, little abdominal tenderness is usually present, producing the classic intense visceral pain disproportionate to physical examination findings. The mucosal barrier becomes disrupted as the ischemia persists, and bacteria, toxins and vasoactive substances are released into the systemic circulation. This can cause death from septic shock cardiac failure or multisystem organ failure before bowel necrosis actually occurs. Bowel necrosis can occur in 8-12 hours from the onset of symptoms. Transmural necrosis leads to peritoneal signs and heralds a much worse prognosis; men might be at higher risk for occlusive arterial disease because they have a higher incidence of atherosclerosis. Acute mesenteric ischaemia (AMI) frequently a disease of people older than yrs. Young people with atrial fibrillation or risk factors for MVT, such as oral contraceptive use or hypercoagulable status may present with AMI.

Inflammatory bowel disease.[66, 67]

Inflammatory bowel disease is the term used to describe the two enigmatic disease processes of ulcerative colitis and crohn's disease. The diseases are related by virtue of having an unknown etiology, common clinical symptoms and overlapping histologic features.

Ulcerative colitis

Ulcerative Colitis is a non specific inflammatory disease involving the mucosa of the colon and rectum. One third of patients undergo operative treatment because of intractability, disease complications and premalignant nature of the disease. Removal of the colon and rectum cures ulcerative colitis.

Epidemiology, etiology and pathogenesis

Ulcerative colitis is more common in developed countries when compared with developing countries, thought to be related, in part, to differences in dietary intake. It has a relatively equal distribution between genders, and it occurs at all ages with peak onsets between the second and fourth decades. Ulcerative colitis is more common in Jewish than in non-Jewish people and in whites than in non whites.

The specific cause of ulcerative colitis in unknown. Current hypothesis suggests that it probably results from a combination of factors, leading to dysfunctional immunoregulation in the intestinal wall. These factors include dietary intake, genetic predisposition, and an imbalance between the normally controlled states of regulated inflammation in the intestinal wall. Family history is the most consistent risk factor for inflammatory bowel disease. Parent-child and sibling relationships are more common than relationships involving distant relatives. Two genetic abnormalities found to be associated with ulcerative colitis are variation in DNA repair genes and class II major histocompatibility complex genes. Peripheral arthritis and ankylosing spondylitis are the most common extra intestinal manifestations of ulcerative colitis and usually improve or

resolve after colectomy. Primary selerosing cholangitis is the most serious extra intestinal manifestation of ulcerative colitis and does not resolve with colectomy.

Peripheral arthritis and ankylosing spondylitis are the most common extra intestinal manifestations of ulcerative colitis and usually improve or resolve after colectomy. Primary sclerosing cholangitis is the most serious extra intestinal manifestation of ulcerative colitis and does not resolve with colectomy.

Pathology

The most common disease pattern in ulcerative colitis is the continuous uninterrupted inflammation of the rectal mucosa that extends to a variable distance into the more proximal colon. Inflammation in ulcerative colitis is confined to the mucosal and sub mucosal layers of the colon. There is infiltration of polymorphonucleocytes and round cells into the crypts of lieberkuhn at the base of the mucosa with multiple crypt abscesses. Confinement of the inflammation to the inner layers of the bowel wall is an important characteristic of ulcerative colitis. However, with the extensive inflammation characteristic of toxic megacolon the full thickness of the bowel wall may be involved, and the process may progress to necrosis and perforation of the colon.

Crohn's disease [64, 65]

It is an inflammatory disease that may affect any segment of the gastrointestinal tract.

Epidemiology, etiology and pathogenesis

The incidence is about 6 / 100,000 population depending on geographic areas. There is a bimodal age distribution with a peak onset between 15 and 30 years and a second smaller peak between 55 and 80 years. It occurs equally between genders and is more common among Jewish people and urban residents.

The etiology of crohn's disease is unknown. The accepted theories suggest that it is probably due to a combination of events. These include a specific infections agent, a

defective mucosal barrier resulting in increased exposure to antigens, and an abnormal host response to intestinal contents. Chromosome 16 and the HLA region of chromosome 6 have been implicated in susceptibility to disease.

Pathology

Crohn's colitis is grossly characterized by a thickened colonic wall and a mucosal appearance of deep, indolent, linear ulcers, cobble stoning, friability, and structuring and apthoid ulceration. Single or multiple strictures may be present in both the colon and small bowel.

Microscopically, there is transmural inflammation, sub mucosal edema, lymphoid aggregation, granulomas and ultimately fibrosis. The pathognomic microscopic feature are the noncaseating granuloma consisting of localized, well formed aggregate of epithelioid histiocytes surrounded by lymphocytes and giant cells.

Diverticular Disease: [39, 59, 68]

Diverticular disease of the colon is a clinical term comprising diverticulosis and its complications. It can be acquired pulsion type or false diverticula form where vessels penetrate the circular muscle to reach the mucosa.

Diverticular disease appears in a population when a diet rich in fiber is exchanged for one containing more refined carbohydrate and meat and less fiber. The sigmoid colon is affected in 95% of patients, and the descending, transverse and ascending portions are involved in decreasing order of frequency. Right sided diverticulosis is common in Asia and rare in West. Complications (diverticulosis and bleeding) eventually develop in 10 to 20% of patients with diverticulosis. Diverticulitis is more likely if diverticula are numerous, involve much of the colon and develop at an early age. It begins as infection in a diverticulum. Generally only one diverticulum is involved at a time, most commonly in the sigmoid colon. Infection extends through the wall into the peridiverticular tissue, causing peridiverticulitis. As a result of the perforation (be it micro or macro) the patient

may develop a pericolic abscess or sinus tract. Abscess can have a varied course – it can drain spontaneously into the colonic lumen, or erode into an adjacent structure causing a fistula, become chronic or rupture into the free peritoneal cavity. Free perforation into the peritoneal cavity may occur as a result of increased intra luminal pressure as well as rupture of an area of peridiverticulitis. The original perforation seals quickly and the pericolic infection is isolated from the colonic lumen.

Meckel's Diverticulum: [68]

Meckel's diverticulum is the most commonly encountered congenital anomaly of the small intestine occurring in approximately 2% of the population. It was reported initially in 1598 by Hildanus and then described in detail by Johann Meckel in 1809. It is located on the antimesenteric border of the ileum 45 to 60 cm proximal to the ileocaecal valve and results from incomplete closure of the omphalomesenteric or vitelline duct. An equal incidence is found among men and women. The usual manifestation is a relatively wide mouthed diverticulum measuring approximately 5 cm in length with a diameter of up to 2 cm. Cells lining the vitelline duct are pluripotent therefore it is not common to find heterotrophic tissue within the diverticulum, the most common being gastric mucosa (present in 50% of all Meckel's diverticulum) Pancreatic mucosa is encountered in approx. 5% of diverticula, less commonly these diverticula may harbor colonic mucosa. The most common clinical presentation is gastrointestinal bleeding, which occurs in 25% to 50% of patients. Another common presenting symptom of Meckel's diverticulum is intestinal obstruction.

Clinical Features: [69]

Peritonitis is an inflammation of the peritoneum either involving a portion or all of the parietal and visceral surfaces of the peritoneal cavity. Perforation of an ulcer into the general peritoneal cavity is a catastrophe that often occurs with dramatic suddenness and unless correctly treated, progresses to the death of the patient. The signs and symptoms produced by the perforation vary according to the time that has elapsed since the rupture

occurred. There are three stages in the pathological process that can be recognized. The symptoms of each stage can be enumerated:

Early (within the first two hours) [69]

- Severe and generalized abdominal pain.
- Anxious countenance.
- Livid or ashen appearance.
- Cold extremities.
- Cold, sweating face.
- Subnormal temperature (95° F or 96° F) .
- Pulse low and weak.
- Shallow respiration.
- Retching or vomiting (slight).
- Pain on top of one or both shoulders.

Intermediate (two to twelve hours)

- Cessation of Vomiting.
- Decreased abdominal pain.
- Normal temperature or slightly elevated temperature.
- Abdominal wall very rigid, tender.
- Tender pelvic peritoneum.
- Diminution of liver dullness.
- Great pain on movement of the body.

Late (after twelve hours)

- Vomiting more frequent but still not profuse.
- Facies of late peritonitis ,classically described as Hippocratic facies
- Abdomen tender and distended.
- Pulse rapid and low; hypovolemic shock may be present.
- Temperature usually elevated.

INVESTIGATIONS

Laboratory Studies:

A complete blood cell (CBC) count with differential count in patients with suspected peritoneal infection. Most patients with intraabdominal infections demonstrate leukocytosis (> 11,000 cells / mm3) with a shift to the immature forms on the differential cell count. But patients who are immune compromised and patients with certain types of infection (e.g. typhoid) may demonstrate absence of leukocytosis and may even demonstrate leucopenia.

Serum amylase and lipase levels in patients with possible diagnosis of pancreatitis.

Urine analysis is essential to rule out urinary tract diseases (E.g. pyelonephritis may mimic peritonitis). However patients with lower abdominal and pelvic infection often demonstrate WBC in the urine and microhematuria. The presence of frank pyuria, large number of red blood cells and bacteria in the specimen suggest a urinary source of patient's symptoms.

Widal test

This is a test for the measurement of H and O agglutins in the patient's sera for typhoid infection. The results are interpreted according to the agglutination titre.The test is taken to be positive if titre is greater than 1/100 for O agglutins and 1/200 or more for H agglutins or rise in titre is demonstrated .

Peritoneal fluid:

A peritoneal fluid should be evaluated for glucose, protein, and lactate dehydrogenase, and gram stain, aerobic and anaerobic culture to rule out peritoneal infection.

A peritoneal fluid amylase should be done if pancreatitis or pancreatic leak is suspected; creatinine level when a urinary leak is suspected. The peritoneal levels should

be compared with serum levels. Routine intraoperative peritoneal fluid cultures in defined acute disease entities (i.e. gastro-duodenal ulcer perforation, appendicitis, and diverticulitis, perforation of the colon caused due obstruction or ischemia) are controversial. Several studies have found no significant outcome in regard to postoperative complication rates or overall outcome. The antibiotic regimen is based on operative culture data in only 8-10% of the time (Bilik, 1998).

Radiographs:[70]

The presence of free, intraabdominal gas almost always indicates perforation of a hollow viscus. The commonest cause is perforation of p1eptic ulcer; other much less common causes are diverticulitis and malignant tumors. About 70% of perforated ulcers will demonstrate free gas, a phenomenon that is almost never seen in cases of a perforated appendix. As little as 1 ml of free gas can be demonstrated on a radiograph, either an erect chest, or a left lateral decubitus abdominal film. Radiographic techniques are important and the patient should remain in position for 5-10 minutes.

The clinical condition of the patient will determine the radiographic technique used. Chest films taken with the patient in an upright position are ideal for demonstrating free air because the x-ray beam strikes the hemi diaphragms tangentially at their highest point.

A lateral decubitus or even a supine radiograph is used in patients who are too ill to be moved. Left lateral decubitus views of the abdomen are also sensitive for detecting small amount of free air interposed between the free edge of the liver and the lateral wall of the peritoneal cavity. Care should be taken to include the upper abdomen, because air rises to the highest point in the abdomen, which frequently is beneath the lower ribs. Films obtained with the patient in the right lateral decubitus position are also helpful, but gas in the stomach or colon may obscure small amounts of the free air. Pneumoperitoneum can be detected in 76% of cases using an erect film only, but when a left lateral decubitus projection is included, a pneumoperitoneum can be demonstrated in

nearly 90% of cases. Reasons suggested for only 76% perforations manifesting as free gas in peritoneum are sealing of perforation, lack of gas at the site of perforation or adhesions around the site of perforation.

TABLE -3

SIGNS OF A PNEUMOPERITONEUM ON THE SUPINE RADIOGRAPH

Right upper quadrant gas:
➢ Perihepatic. ➢ Subhepatic. ➢ Morrison's pouch. ➢ Fissure for the ligamentum teres.
Rigler's or Double wall sign
Ligament visualization
Falciform (ligamentum teres).
Umbilical (inverted 'V' Sign), medial and lateral
Urachus.
Triangular air.
The cupola sign.
Football or air dome.

Pseudopneumoperitoneum: [70]

A number of conditions have been described which simulate free air in the peritoneal cavity i.e. pseudopneumoperitoneum. These are important because failure to recognize them may lead to an unnecessary laparotomy in search of a perforated viscus.These are Chilaiditi syndromes: is distended bowel, usually hepatic flexure of the colon, interposed between the liver and the diaphragm.

- Sub diaphragmatic fat
- Curvilinear pulmonary collapse.
- Uneven diaphragm

- Distended viscus.
- Subphrenic abscess.

Pneumoperitoneum without peritonitis: [70]

Occasionally, asymptomatic patients or those with very minimal signs and symptoms are found to have a pneumoperitoneum.

Causes of pneumoperitoneum without peritonitis are -

i) Silent perforation of a viscus which has sealed itself.
ii) Postoperative setting.
iii) Peritoneal dialysis.
iv) Perforated jejunal diverticulosis.
v) Laparoscopy.

Use of contrast media in suspected perforation:

Not infrequently, a patient presenting with severe upper abdominal pain has equivocal clinical signs and no free gas is demonstrable on plain radiographs. Water soluble contrast medium (about 50 ml) is given by mouth or injected through a nasogastric tube, with the patient lying on his/her right side. The patient can be examined fluoroscopically or the abdominal radiographs can be repeated after the patient has remained in this position for 5 minutes. Duodenal ulcers which have perforated but show no free gas will normally demonstrate evidence of a leak of contrast medium. Patients with pancreatitis may have an oedematous stretched duodenal loop. Ionic water soluble contrast medium should not be given if the patient's clinical state is such that there is risk of it being inhaled and causing pulmonary oedema.

Appendicular perforation: [72, 73, 74]

A ruptured appendix may rarely lead to the development of a small amount of free intraperitoneal air. The obstructed appendiceal lumen prevents larger collection of gas

from escaping into the peritoneal cavity except in case of a ruptured gas containing abscess. It may show a fecolith in the right lower quadrant.

Typhoid: [61]

- Multiple fluid levels.

Ascariasis: [61]

- Whirlpool pattern may be seen.

Ultrasound: [74, 75]

Ultrasound examination allows very rapid screening of patients in suspected patients, for triage of patients who are to undergo more invasive imaging testing. Visualization of an interference echo with a shifting phenomenon is a very strong indication of the presence of free air in the abdominal cavity. This interference echo can be defined as the interruption of echo transmission due to the space between the parietal peritoneum and the surface of the liver. This free air within the peritoneal cavity can be shifted by changing the patient's position. Since the distal stomach and proximal duodenum are the most frequent sites of peptic ulcer disease, focal peritonitis due to perforation usually is located in the right upper quadrant. Unlike free peritoneal fluid, this localised exudate doesn't change shape or location when the patient's position is altered .Other findings are subphrenic or subhepatic collections. Moreover ultrasound can detect ascitic fluid as little as 10 ml. Ultrasound guided paracentesis is safe and will yield a fluid aspirate in nearly 100 % compared to clinical diagnosis with a sensitivity of 58 %.

Computed tomography of abdomen: [76]

The Computed tomography diagnosis of perforation was based on the direct findings of extraluminal air or gastrograffin. Indirect findings are an abscess or inflammatory mass surrounding an enterolith in the region of appendix or a bowel wall related phlegmon or abscess with fluid in the mesentery or surrounding radiopaque foreign body. Computed tomography is a valuable method in the diagnosis of alimentary

tract perforation .The diagnosis can be established rapidly without patient preparation and with a high sensitivity.

Differential Diagnosis:[69]

There are four conditions, sometimes giving rise to symptoms similar to those of perforated ulcer that either do not call for operation or in which operative interference is positively contraindicated. They are:

1) Intestinal Colic:

Diagnosis usually clears on consideration of the patient's history and on careful observation of the condition of the abdominal wall, liver dullness, and the pelvic peritoneum. The radiation of the pain of biliary colic to the subcapsular region and that of renal colic to the groin are sufficiently diagnostic. In ureteral stone colic, the abdominal wall is not usually rigid, and the sufferer may throw themselves about or writhe in agony. After perforation of an ulcer, pain increases on movement and prevents movements. The pain of renal colic is nearly always limited to one side.

2) Right sided or bilateral pneumonia or pulmonary infarction:

The respiration rate will be greater than one would expect with an early peritonitis without distention. A friction rub may be heard on auscultation of the chest. A posteroanterior and lateral chest X-ray will solve the diagnostic dilemma.

3) Acute pancreatitis:

Abdominal rigidity is not so generalized and constant. Cyanosis and slight jaundice are more often seen in pancreatitis, which often occur in obese patients.

Other surgical conditions that are difficult to distinguish from perforated ulcer are:

A) Acute appendicitis:

In the second stage perforated ulcer may be and often is misdiagnosed as appendicitis, as the escaped contents may trickle down and cause pain in right iliac fossa.

This simulates appendicitis closely, for the sequence – epigastric pain, nausea and vomiting, right iliac fossa pain, and fever – may be produced as in appendicitis, but the intensity of initial collapse and the persistence and maximal degree of tenderness over the duodenal area help to differentiate it. In appendicitis the abdominal rigidity is seldom as extensive or as marked as in perforated ulcer and the liver dullness is normal.

B) Intestinal obstruction:

In their late stages, it is difficult to distinguish intestinal obstruction from perforation, for peritonitis is often a complication of late intestinal obstruction and the board like rigidity accompanying a perforated ulcer tends to diminish somewhat as the distention increases. In such cases the history and possibly the character of the vomit may serve to differentiate these conditions.

C) Ruptured ectopic gestation:

The main points in diagnosis are features of hemorrhagic shock such as the blanching of the lips, tongue, nails and the absence of true abdominal rigidity, though the abdomen is tender especially in the lower part.

D) Rupture of an ulcer with formation of localised subphrenic abscess:

Due to previous adhesions, slow leakage of the escaping gastric contents does not flood the peritoneal cavity and the symptoms are modified. The pain may be very great but the initial collapse is not so prostrating, and the abdominal signs will soon be localised to the upper segment of the abdomen and lead to the development of a subphrenic abscess containing gas. If such an abscess develops anteriorly, the local signs of intraperitoneal suppuration are very evident, but when the mischief is high up under the diaphragm, the signs and symptoms take longer to develop. Temperature, rigors, leukocytosis and dullness at the base of the lung consequent on pleural effusion or basal congestion will diagnose a collection of pus under the diaphragm.

Treatment:

Once the clinical diagnosis of peritonitis is made, rapid institution of both physiologic support and aggressive anti-infective therapy are imperative.[12]

Primary objectives in the treatment of peritonitis are –

1. Resuscitation
2. Initiation of antibiotic therapy
3. Elimination of the source of bacterial contamination
4. Reduction of the bacterial inoculum
5. Continued metabolic support.

Resuscitation:

It is an axiom that in all cases of peritonitis, some degree of hypovolaemia is present. The plasma volume must be restored and the plasma electrolyte concentration corrected. Fluid administered must contain both crystalloids and colloids. The effectiveness of fluid replacement can be judged by the normalization of pulse rate, blood pressure and mental status. Placement of a urinary drainage catheter is essential since restoration of urine output is a reliable indicator of adequate fluid resuscitation. Placement of central venous line is imperative for monitoring accurate fluid replacement.

Supplemental oxygen may be necessary and in more extreme circumstances, endotracheal intubation and mechanical ventilation may be needed to preserve oxygenation. Nasogastric decompression using a sump tube should be used in the presence of an ileus to prevent pulmonary aspiration and reduce abdominal distention.

Antibiotic Therapy:

Antibiotic therapy should be initiated as soon as a clinical diagnosis of peritonitis is obtained. The initial selection of antibiotic is empiric. The choice of antibiotics is made with the following considerations–

a) The demonstrated activity of the drug against bacteria that are presumed

to be present based upon the level of gastrointestinal perforation.

b) The bactericidal activity of the antibiotic in the infected tissue.

Presumptive therapy should include coverage for both aerobic gram negative rods and anaerobic organisms. Agents that possess activity against aerobic gram negative bacilli include aminoglycoside, second and third generation cephalosporins and either ampicillin or ticarcillin combined with a beta lactam inhibitor (i.e. sulbactam or clavulanic acid). The optimal duration of antibiotic therapy must be individualized and depends on the underlying pathology, severity of infection, speed and effectiveness of source control.

Traditionally a 10 days therapy has been recommended, although newer studies suggest that a five day therapy may be sufficient. (Mc Quaid; 1999, E.C.Khouser 1997)

Surgical Management:

Surgery remains an important therapeutic modality for all cases of peritonitis. Operative management should be directed towards the control of the source of contamination. This can be accomplished by closure of the perforation, resection of the perforated viscus, or exclusion of the affected organs from the peritoneal cavity.

The secondary goal of operative management is to reduce the bacterial inoculum with the intent to prevent recurrent sepsis. Standard intraoperative techniques to accomplish these goals include swabbing and debriding fibrin, blood and necrotic material and copious irrigation of the peritoneal cavity which are generally accepted and practiced maneuvers. The use of non standard surgical techniques to prevent recurrent sepsis remains controversial. Radical debridement of fragile serosal surfaces may itself cause significant bleeding and fibrin deposition, and at present there is no perceived benefit in the use of this technique.

Planned repeated laparotomy for generalized peritonitis is a technique developed to prevent recurrent sepsis by repetitive abdominal exploration to debride necrotic material and drain abscesses.

Perforated Peptic ulcer:

Peptic ulcer perforation has been classified as 'free perforation' when duodenal / gastric contents spill into the peritoneal cavity. It is called 'contained perforation' when a fullthickness hole is created by an ulcer but free spillage is prevented by contiguous organs resulting in walling off. The term 'penetrating ulcer' has been used to describe perforation in to the pancreas. It is also a type of contained perforation.[77] The incidence of ulcer perforation is 7- 10% per 1 lakh population. Perforation is less frequent than bleeding but more common than obstruction.

Pyloroduodenal perforation occurs six to eight times more commonly than gastric perforation.[78] In a large series, gastric perforation constituted 13% of total perforation. Gastric perforation is more common in elderly. Prepyloric perforation and duodenal perforation occur more often in young men. 90% of perforated duodenal ulcers are seen on anterior wall. 60% of gastric perforations occur on lesser curvature and 40% are distributed all over the stomach. [77] A recent review has shown that 52% of patients of perforation are on ulcerogenic agents. Gastrografin study or CT scan of the abdomen may be required to determine the cause of unexplained abdominal pain.[79]

There are two types of patients with perforation: Acute perforation in whom history of less than 3 months or no history of ulcer symptoms is present and others who have chronic ulcer perforation with symptoms of more than three months duration.

Acute perforation of duodenum is now estimated to occur in 5-10% of patients with ulcers most of whom are between the age of 40-50 years of age. A history of peptic ulcer disease is present in 60-70% of patients.

All patients of perforation on NSAID therapy should be operated. The recurrence of ulcer perforation was reported as 7% in case of NSAID users after simple closure. The operation preferred is simple closure followed by 8 weeks of omeprazole therapy. There is no need to add definitive surgery at the time of emergency operation.[80]

Perforated gastric ulcer tends to occur in older patients and may be associated with adenocarcinoma. This leads to higher mortality rates than the routine perforated duodenal ulcer. The operation of choice is gastrectomy as more than 10% of benign looking ulcers may be malignant.[79]

When a patient with peptic ulcer perforation presents to the surgeon, the surgeon has to make 5 therapeutic decisions.[81]

1. Whether an operation is to be performed or not.
2. Whether patient is stable to undergo operation.
3. Whether to do an omental patch closure or a definitive surgery.
4. Type of definitive surgery to be done.
5. Whether availability of new drugs should influence the choice of operation.

Conservative Management:

Most patients with peptic ulcer perforation require operative therapy on rare occasions, conservative management of perforation can be beneficial particularly in those patients who have concomitant medical illness, perforation of more than 24 hrs, systolic pressure less than 100 mm Hg at the time of admission. These risk factors have definitive bearing on mortality rate.[82] If one risk factor is present mortality is about 10%, if two factors are present mortality is about 40%, if three factors are present mortality is about 87%. These patients require close monitoring in intensive care unit as they may deteriorate and need operative therapy. If abdominal findings do not improve in 12 hours then operation is indicated.[84, 85]

TABLE -4

CONTRAINDICATIONS FOR NONOPERATIVE TREATMENT[77]

Age > 70 years
Steroid use
Gastric perforation

Simple closure Vs Definitive operation:

Simple closure was first suggested for patients with gastric ulcer perforation in 1894 and later was popularized by Roscoe Graham in perforated duodenal ulcer in 1937.[81] Longterm follow up of these patients with simple closure has significantly influenced operative management in the past 10-15 years. Simple closure will lead to satisfactory result in 1/3rd of patients. The remaining 2/3rd of patients will need acid suppression therapy or definitive operation for complications. According to Boey and Wong, [83] complications occurred in 52% of these patients (28% had bleeding, 15% had pyloric obstruction, 9% had reperforation). In this group of patients, 40% required reoperation.

Ralph I George followed up 75 patients of simple closure for 5-10 yrs, 14 of these patients were on ulcerogenic drugs; 7% of them had recurrence while 6% patients who did not take ulcerogenic drugs had recurrence rate of 77%, proving that their ulcer diathesis was virulent enough to need definitive surgery.[86]

Boey and associates compared simple closure and closure with P.G.V. in 78 patients with acute perforation, recurrence rate was 34% at 36 months after simple closure, reoperation was required in 43% of this group.[87]

The higher reoperation rate in this group may be due to ethnic and geographic variation.

Surgical technique [88]

- The perforated duodenal ulcer closure was described by Graham.

The two principal techniques used in closure are

a) Simple apposition of the perforation

b) Omental patch technique

Apposition should be performed using three or occasionally four sutures using suture materials such as vicryl, dexon or polydioxonone. The sutures should be through the full thickness of the duodenal wall at least 1cm from the edge of the defect. The omental patch should be used if the perforation is large or if the duodenum is so indurated that it is unlikely to hold sutures. Sutures are placed just to bring about apposition but should not be tied to approximate the ulcer edges. Adjacent omentum should be brought up with an intact vascular pedicle. The sutures are then successively tied from the superior to the inferior side of the perforation, so as to tampon the perforation with the living omental pedicle graft. The disadvantage of sewing the ulcer shut, even if this is technically feasible, is that the omental patch placed over such a closure does not have the surface contact with the anterior duodenal serosal.

In cases of large perforation or the scarred, inflexible duodenal wall that makes simple closure difficult two options are available.

a) Conversion of the perforation into a Heineke- Mikulicz pyloroplasty.

b) Serosal patch with proximal jejunum

Laparoscopic approach: [87, 88]

Laparoscopic techniques have been applied to virtually all abdominal procedures and perforated duodenal ulcer is no exception. It was introduced by Nathanson in 1990.

Two laparoscopic approaches have been developed

a) Suturing technique

b) Fibrin plug technique

Pneumoperitoneum is established by either open or closed method and a 10 mm trocar is inserted at the umbilicus. Exploratory laparoscopy is performed to confirm the diagnosis and to ensure that laparoscopic closure is technically feasible. Working ports are then placed in the right hypochondrium (for grasper), left hypochondrium (for scissors, needle holder) and epigastrium (for suction irrigator).

Primary closure is performed using a 5mm needle holder and a no 2-0 absorbable suture mounted on a half circle needle. The omental patch technique is performed as for open procedure.

The fibrin plug technique involves delivering solution of fibrinogen and thrombin through separate lumina of a double lumen catheter. As the two solution meet at the perforation, a fibrin plug is formed which seals the perforation.

Laparoscopy seems particularly useful for patients without pneumoperitoneum, or for those who present with atypical symptoms and signs. Postoperative wound pain is minimized, allowing early mobilization and a more rapid resumption of daily activities. Laparoscopy is also associated with a significant decrease in the rate of postoperative chest complications.

Perforation associated with hemorrhage:

When perforation of a duodenal ulcer in accompanied by overt gastrointestinal bleeding, a concomitant posterior ulcer should be suspected. Duodenum is opened through the anterior perforation for suture control of the posterior bleeding ulcer. An acid reductive procedure is mandatory two alternatives being truncal vagotomy or proximal gastric vagotomy.

DEFINITIVE OPERATIONS:

Truncal Vagotomy (TV) with pyloroplasty: [79]

It has been used as definitive operation for perforated duodenal ulcer. Advantages:

i) The lesion is removed

ii) Pyloric stenosis is avoided.

iii) Length of operation is only slightly prolonged.

The transverse closure of gastroduodenostomy is performed using an interrupted one layer closure. Operative mortality of emergency Truncal vagotomy with pyloroplasty for perforated ulcers varies from 0-15% in four large series since with recurrence rate of 12-15%.

Truncal Vagotomy with Hemigastrectomy:

The principal disadvantages of truncal vagotomy with hemigastrectomy are the only modest increase in operative time over truncal vagotomy with pyloroplasty, but there is an 8-10% decrease in recurrent ulceration compared with truncal vagotomy with pyloroplasty. This is preferred in cases of perforation in pre-pyloric region. The operative mortality rate for resection is extremely low in properly selected patients

Proximal gastric vagotomy (PGV): [89]

In 1973, Johnston and associates reported first clinical experience with this technique in addition to closure of perforation. Cumulative rate of recurrence was 63% after simple closure, 12% after truncal vagotomy with drainage and only 4% after proximal gastric vagotomy with simple closure in 60 patients over a period of seven years. Proximal gastric vagotomy should be avoided in patients with duodenal scarring. Jordan has suggested that all stable patients with perforated duodenal ulcer without risk factors should undergo proximal gastric vagotomy with closure of perforation.

Post operative follow up and complications:

Perforation may be the end stage in some cases of acute ulcer perforation as in perforation caused by NSAID or ulcerogenic drugs. The patients to be put on omeprazole for eight weeks. H pylori therapy may be added to reduce the recurrence rate.

In acute perforation recurrence rate was 43% and in chronic ulcer perforation was 66 to 87%. 52% may develop complications like bleeding, pyloric obstruction and reperforation. The patients with simple closure will need lifelong acid suppression agents and eradication of H pylori. NSAID, cigarette smoking and alcohol aggravate the disease.

TABLE -5

RECOMMENDED DEFINITE SURGICAL PROCEDURE FOR PERFORATED DUODENAL ULCER

First choice	Second choice	Third choice
PGV with Closure	TV with Drainage	Simple closure

Perforated gastric ulcer

The mortality rate of gastric ulcer perforation is higher as it occurs in older patients and is usually associated with more contamination. A biopsy should always be taken from the gastric ulcer or a partial gastrectomy performed. However, if the patient's general condition is poor, then a simple omental patch closure along with a biopsy may be adequate. Juxta pyloric ulcers behave like duodenal ulcers clinically and are treated by truncal vagotomy and pyloroplasty or by truncal vagotomy and resection.

Benign ulcers in unstable or elderly patients may be treated with excision and closure or closure with omental patch. An Ulcer high on the lesser curvature should be excised and closed. If excision is not possible, the ulcer margin should be biopsied before closure with omental patch.

Perforated stomal ulcer [90 , 91]

Stomal ulcers more commonly penetrate surrounding structures and occasionally perforate into the transverse colon, resulting in a gastrojejunal colic fistula. Perforated stomal ulcers may occur many years after a simple gastroenterostomy. The most effective operation for patients with perforated marginal ulcers is to resect or reresect the stomach including the ulcer and perform a vagotomy if not done earlier. Revagotomy should be done and attention paid to find out the posterior vagus nerve, which is most likely missed. Patients with gastrojejunal-colic fistula are treated by gastric resection, vagotomy, and partial transverse colectomy.

The surgical treatment of a perforated anastomotic ulcer is dictated by the condition of the patient and the operation originally performed. Simple closure can be done, but recurrence rate is 80%.

Perforation of small intestine: [92]

If one is dealing with perforation and associated peritonitis that precludes safe primary anastomosis, a proximal stoma and distal mucous fistula are constructed in close proximity to each other but not so close as to prevent placement of a proper fitting appliance. Once the patient is back to a normal state of health, both stoma and mucous fistula are taken down through and abdominal incision connecting both ends of the bowel. The latter are mobilized and an anastomosis is performed outside the peritoneal cavity. The bowel is then replaced in the peritoneal cavity, the fascia closed, and the skin and subcutaneous tissue left open.

Typhoid Enteritis: 58, 93

Treatment of typhoid fever and uncomplicated typhoid enteritis is accomplished by antibiotic administration. Complications requiring potential surgical intervention include hemorrhage and perforation. Intestinal perforation through an ulcerated Peyer's patch occurs in approximately 2% of cases. Typically, it is a single perforation in the terminal ileum.

Simple closure of the perforation is the treatment of choice.

With multiple perforations, resection with primary anastomosis or Exteriorization of the intestinal loops may be required.

According to Ameh E.A. segmental resection seems to be best treatment for typhoid perforation.[94]

Appendicular perforation:

Generalized peritonitis following perforative appendicitis is the major cause of continuing mortality from appendicitis. This entity requires vigorous treatment. Appendicectomy must be performed in children whether the peritonitis is diffuse or not, since the other course is associated with a prohibitive mortality. But the management of this problem in adults remains a controversy. In patients with diffuse peritonitis after perforative appendicitis appendicectomy is the treatment, as the perforation remains a continuing source of peritoneal contamination.

At operation for free perforation, visualization of all peritoneal surfaces is essential. All purulent and feculent material should be removed and dependent collection of pus should be aspirated, the peritoneal cavity should be repeatedly rinsed with warm saline solution. The placement of multiple prophylactic drains is unwarranted. Not only do these drains fail to improve morbidity and mortality, but there is some evidence to indicate that they may actually increase it.

Intestinal perforation in tuberculosis:

Surgery is the treatment of choice. Early surgery and anti-tubercular treatment are life saving.

a) Simple closure of perforation: It may be done in two layers using nonabsorbable sutures. As tuberculosis strictures are short, it is a quicker treatment for those who

are critically ill. Oval excision of the perforated area with transverse closure, reinforced by an omental patch may also be done.

It is contraindicated when:

i. Perforation has occurred in a segment of bowel where there is much granulation tissue and caseation, where the stitches are liable to cut out as if applied though cheese and the repair is doubtful.

ii. When there is a distal stricture, a blowout of the suture line can occur if the stenosis lies distal to the perforation (as it is in most of the cases). This method has very limited application for very ill patients.

b) Simple closure with bypass of strictures: Simple closure and the bypass of strictures by ileoileostomy or ileotransverse colostomy safeguard the closure against a blow out. Even when bypass is added, fistula formation frequently occurs.

c) Resection of perforated segment: If the disease is limited to a short segment and the patient is fit, the most effective treatment is the resection of the diseased segment. The segment is resected and continuity restored by end to end anastomosis.

d) Perforation at the ileocaecal region: Here closure with ileotransverse anastomosis is the preferred treatment but if the patient is fit a local ileocaecal resection can be performed.

High index of suspicion is essential for early diagnosis and optimal treatment of the patients with tubercular intestinal perforations.

Prognosis:

In a study by Talwar et al., the mortality rate was 29.3%. Adverse prognostic factors were operation beyond 36 hours, multiple perforations and faecal fistula formation. Mortality was least with early resection and end – end anastomosis of the perforated bowel segment.

Perforation in Ascariasis:

"Ascaris lumbricoides has been one of man's most faithful and constant companions since he started domesticating pigs. Wherever soil pollution prevails, if only by toddlers in the backyards, and wherever there is warmth and moisture, infestation by the parasite is a hazard". (J.H.Louw, 1966)

As worm infestation is common in India, its mere presence in a case of intestinal perforation does not necessarily implicate them in perforation of the gut and their presence could merely be coincidental. The perforation therefore may be initiated by worms after producing inflammation, or perforation may be due to some other cause and worms happen to be an incidental finding.

The perforation is usually in the upper reaches of ileum, a site higher than found in most cases of typhoid perforation.

In case of localized intraperitoneal abscess with perforation amidst matted bowel, the bowel is devitalized at places. It is not safe to close the perforation. The correct treatment is to resect the whole affected segment of the bowel.

For large perforations on the antimesenteric border, it is safe to suture if diagnosed early. If the gut is unhealthy the affected bowel should be resected.

If there are multiple perforations, then resection and anastomosis should be the treatment of choice. Otherwise the recent perforation can be tackled by a two layer closure, the outer being the non-absorbable material as the left over worms in the gut, may burrow the suture line.

Perforation in Diverticular disease of small bowel: 97

Small bowel diverticular disease is an uncommon clinical entity. Both acquired and congenital diverticula are frequently asymptomatic and become symptomatic when complicated by infection, perforation, obstruction or hemorrhage.

Duodenal Diverticula:

Duodenal diverticula may be acquired or congenital. Perforation may be secondary to diverticulitis or iatrogenic following endoscopic retrograde cholangio pancreatography. It commonly occurs in the retroperitoneum over the right kidney and posterior to the head of the pancreas and duodenum.

When a perforation is suspected, computed tomographic scan of the abdomen with oral and intravenous contrast is very accurate in confirming the diagnosis and in defining the extent of inflammatory reaction.

Prophylactic resection of an asymptomatic diverticulum is not recommended. In the absence of significant retroperitoneal contamination, primary excision of diverticulum with two layer closure is done. In the case of large duodenal defect, serosal patch technique or a Roux-en-Y duodenojejunostomyis preferred.

In the presence of a perforation with significant edema and contamination, a duodenal diverticulization (e.g. gastrojejunostomy, closure of the pylorus, closure of the perforation, tube duodenostomy, gastrostomy tube and jejunostomy feeding tube) with drainage of the surrounding area.

Jejunal and ileal diverticula:

In the presence of diverticulitis or perforation, resection and primary anastomosis is indicated.

Meckel's diverticulum:

Resection of the diverticulum or resection of the segment of ileum bearing the diverticulum with end to end anastomosis.

Diverticular perforation of colon: [97, 98, 99]

All patients with peritonitis must undergo emergency laparotomy. Patients are resuscitated from shock with intravenous crystalloids. Electrolyte concentration is measured and deficits especially in potassium are corrected. Intravenous antibiotics are

given. A solitary perforation can be managed by resection or exteriorization. The resection need not be definitive but just enough to excise the perforated segment and leave normal colon for colostomy. A mucous fistula (or Hartmann pouch) completes the procedure.

Exteriorization is acceptable provided the involved colon can be brought out tension free through a separate incision in the left lower abdominal wall. The second operation to complete definitive resection and restore colonic continuity is performed in 3 months if the patient has recovered sufficiently.

Perforation in ulcerative colitis: [101, 102]

Perforation of the colon occurs in about 1 to 3 percent of patients with ulcerative colitis. The likelihood of perforation is highest in the initial attack of colitis, and the incidence correlates with the severity of the initial attack and the extent of involvement of the colon. The sigmoid colon is the most common site of perforation; the splenic flexure and transverse colon are next in order. Toxic megacolon precedes perforation in only 1/3rd to 2/3rd of cases; the remaining patients perforate in the absence of recognized colonic dilatation. Corticosteroid therapy was thought to be casual factor at one time, but now disproved, but it masks the symptoms and signs of perforation once it occurs.

The diagnosis of perforation is easy to make in an untreated patient having diffuse abdominal pain, tenderness, rigidity, fever and leukocytosis and free air is seen on abdominal radiographs. In hospitalized patients symptoms and signs are blunted by therapeutic agents.

Immediate laparotomy is mandatory in patients with proved or strongly suspected perforation.

Total abdominal colectomy with end ileostomy and exteriorization of the distal sigmoid as a mucous fistula is the procedure of choice for free perforation. In patients with sealed perforation the multiple blow whole methodof Turnball is an option.

Mortality rate associated with perforated ulcerative colitis is about 20 and 40% if free perforation is recognized and treated by prompt operation. The mortality rate is much lower if sealed perforation is found unexpectedly during elective operation.

Perforation in Crohn's disease:

All patients must undergo emergency laparotomy. Patients are resuscitated from shock with intravenous fluids and antibiotics. A solitary perforation can be managed by resection or exteriorization. Simple closure with proximal colostomy leaving the perforated segment inside should be avoided. Resection need not be definitive but just enough to excise the perforated segment and leave normal colon for colostomy. A mucous fistula (or Hartmann pouch) completes the procedure.

Socio-demographic Factors:

The sociodemographic characteristics were age, sex, religion and area of residence. The risk factors evaluated included; cigarette smoking, alcohol consumption, use of nonsteroidal anti-inflammatory medications, stress, number of meals, serological status for Helicobacter pylori and human immunodeficiency virus.

The study will show socio-demographic characteristics, types and risk factors associated with perforated peptic gastroduodenal ulcers which in turn will help in the following aspects;

- Early identification of patients with peptic gastroduodenal ulcer disease who are at risk of perforation in terms of their age and risk factors associated with perforation.
- May aid in creating follow up clinic for patients with peptic gastroduodenal ulcer disease thus effecting care and preventing complications.
- It will show the need of mobilizing both human resource and equipments to effect preoperative, intra-operative and postoperative care of patients with perforated peptic gastroduodenal ulcer disease.

- The study will have an overall impact in reducing complications (such as perforation), morbidity and mortality as well as the cost of treating patients with peptic gastroduodenal ulcer disease if the identified risk factors are averted.
- The study will contribute in improving the teaching on the matter.

Socio-demographic variables are

- Ages
- Gender(male & female)
- Highest education level(none/primary/secondary/bachelor/higher)
- Occupation(unemployed/employed/self –employed)
- Monthly income(<5000/5000-15000/>15000)
- Area of residence(urban/rular)
- Social behavior (alcohol/smoking/both)

Place of residence:

Meant urban or rural areas, where a participant had been staying for the past year before the date of data collection.

Urban area:

Referred to villages found around towns and the city within the radius of 30 kilometers (km); and

Rural area:

Referred to villages found outside the radius of 30 km away from the towns or city.

Education level meant the educational qualification a male &woman had.

Income:

It was defined as low class or middle class. Livelihoods strategies in Lesotho were defined differently depending on the location one were based. However, low class and middle class categories were used:

Low class:

Included the group of people in society who had less money or education, and the people in society who traditionally did physical work and earned low salaries. These included people whore were self-employed in order to survive such as those who were selling traditional beer, those producing agricultural products from the fields, and those working in the factories; and

Middle class:

Referred to the group of people in society who were educated and worked in professional jobs. Included were teachers, nurses, technicians, and economists.

METHODOLOGY

This study has been based on the analysis of 52 cases of perforation peritonitis admitted to PEOPLE'S COLLEGE OF MEDICAL SCIENCES &RESEARCH CENTRE BHOPAL, INDIA during the study period. Patient admitted with particular criteria fixed during the study period was taken as the universe and cases were selected by Purposive sampling.

A prospective study of 52 patients presenting with clinical diagnosis of perforation peritonitis between November 2012 to November 2014 is done.

INCLUSION CRITERIA:

- Patients admitted with diagnosis of perforative peritonitis.
- Both males and females, of all ages.

EXCLUSION CRITERIA:

- Patients with post-operative anastomotic leak/re-exploration.
- Traumatic cases.
- Patients not undergoing surgery.

Each patient was examined thoroughly, after taking a detailed history & socio-demographic factors were noted. Cases were admitted as emergency and possible immediate investigations were done. The diagnosis was made with history, clinical features and X ray abdomen erect posture to support the diagnosis.

Out of the 52 cases of perforation peritonitis all underwent emergency laparotomy. At laparotomy the site of perforation, its pathological condition and the amount of peritoneal contamination were determined. The procedures adopted in the management were omental patch closure, simple closure; open appendicectomy, resection anastomosis, & resection anastomosis with ileostomy.

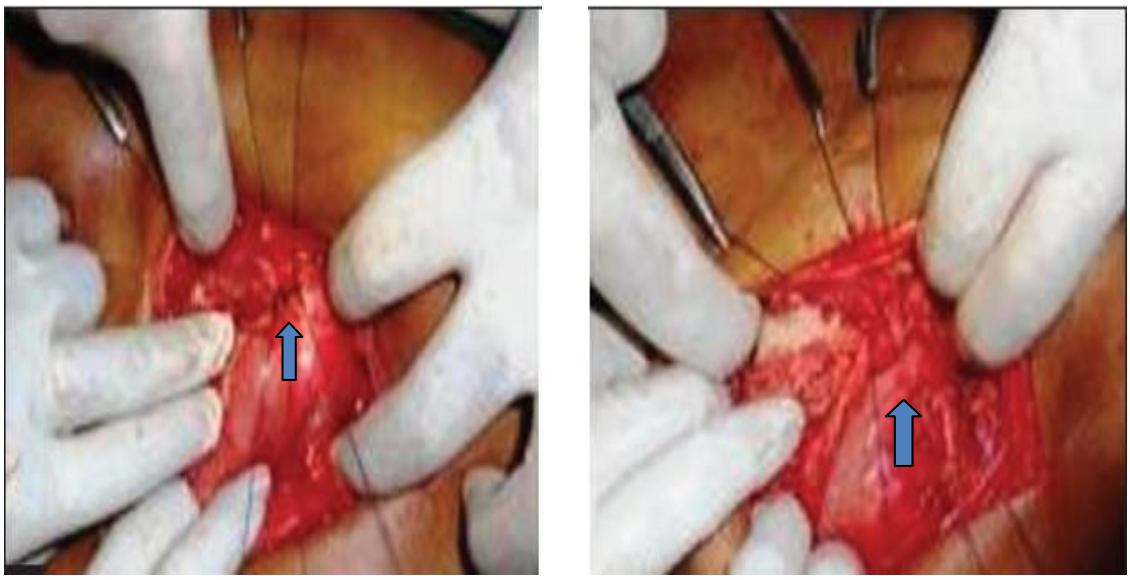


FIG 9 (a) & (b): SIMPLE CLOSURE

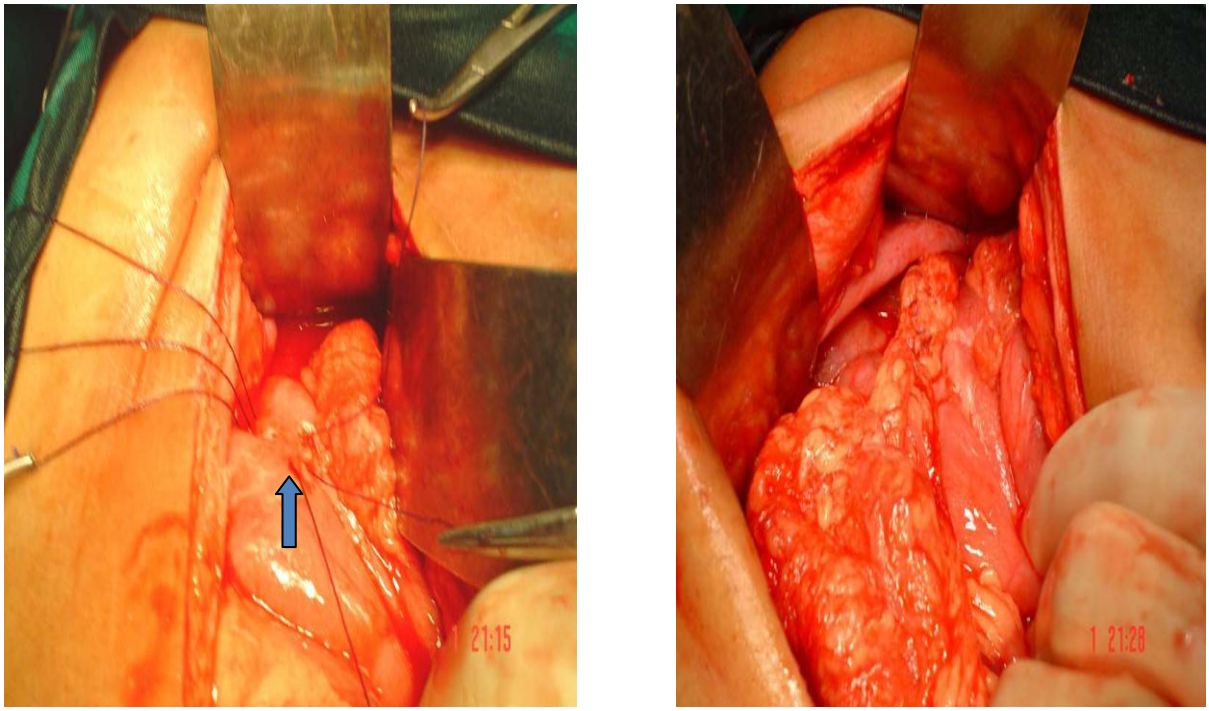


FIG 10 (a) & (b): OMENTAL PATCH CLOSURE

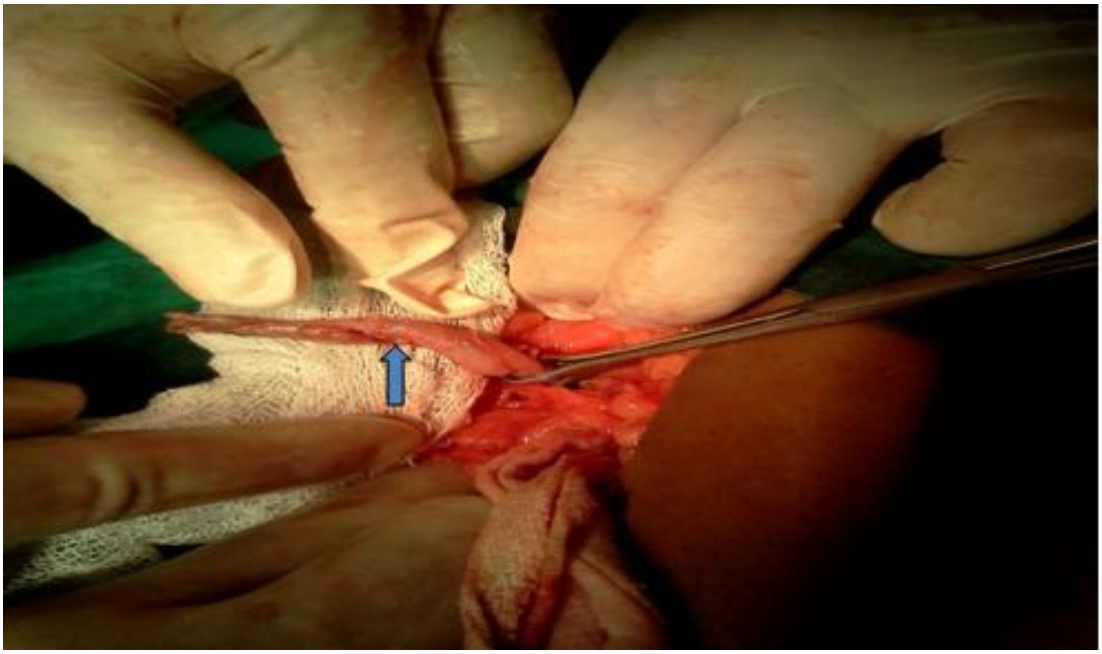
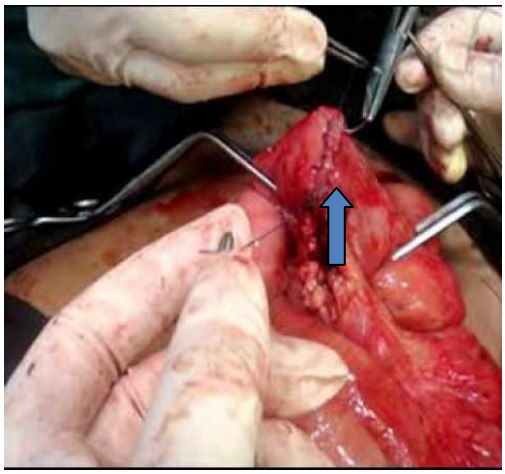


FIG 11: OPEN APPENDICECTOMY



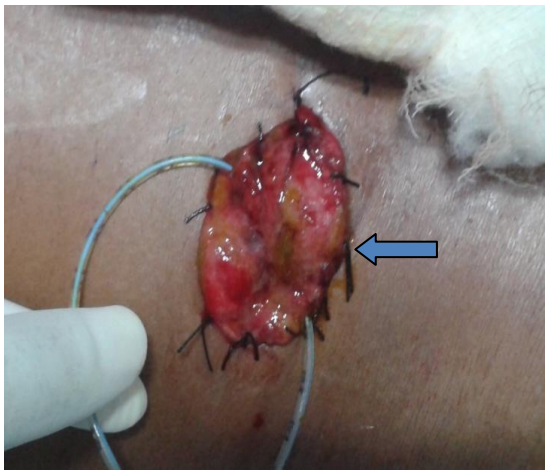


FIG 12: RESECTION ANASTOMOSIS & ILEOSTOMY

Demography /de·mog·ra·phy/ (de-mog´rah-fe) the statistical science dealing with populations, including matters of health, disease, births, and mortality.

Socio-demographic variables are

- Ages
- Gender(male & female)
- Highest education level(none/primary/secondary/bachelor/higher)
- Occupation(unemployed/employed/self –employed)
- Monthly income(<5000/5000-15000/>15000)
- Area of residence(urban/rular)
- Social behavior (alcohol/smoking/both)

Hence, clinical study of peritonitis due to hollow G. I. viscus perforation is undertaken to find out age and gender incidence, types of perforation, correlation between investigation and operative findings, and outcome with respect to morbidity and mortality.

Each case was studied as per the proforma.

Statistical Methods applied

Following statistical methods were applied in the present study

- Chi-square test
- Contingency table analysis
- Independent samples ‘t’ test
- Analysis of variance

Chi-square test

The Chi-Square Test procedure tabulates a variable into categories and computes a chi-square statistic. This goodness-of-fit test compares the observed and expected frequencies in each category to test either that all categories contain the same proportion of values or that each category contains a user-specified proportion of values.

Cross tabs procedure

The Cross tabs procedure forms two-way and multiway tables and provides a variety of tests and measures of association for two-way tables. The structure of the table and whether categories are ordered determine what test or measure to use.

Cross tabs' statistics and measures of association are computed for two-way tables only. If you specify a row, a column, and a layer factor (control variable), the Cross tabs procedure forms one panel of associated statistics and measures for each value of the layer factor (or a combination of values for two or more control variables). For example, if GENDER is a layer factor for a table of MARRIED (yes, no) against LIFE (is life exciting, routine, or dull), the results for a two-way table for the females are computed separately from those for the males and printed as panels following one another.

Independent samples't' test

The Independent-Samples T Test procedure compares means for two groups of cases. Ideally, for this test, the subjects should be randomly assigned to two groups, so that any difference in response is due to the treatment (or lack of treatment) and not to other factors. This is not the case if you compare average income for males and females. A person is not randomly assigned to be a male or female. In such situations, you should ensure that differences in other factors are not masking or enhancing a significant difference in means. Differences in average income may be influenced by factors such as education and not by sex alone.

One-way ANOVA

The One-Way ANOVA procedure produces a one-way analysis of variance for a quantitative dependent variable by a single factor (independent) variable. Analysis of variance is used to test the hypothesis that several means are equal. This technique is an extension of the two-sample t test.

In addition to determining that differences exist among the means, you may want to know which means differ. There are two types of tests for comparing means:

- A priori contrasts and
- Post hoc tests.

Contrasts are tests set up before running the experiment and post hoc tests are run after the experiment has been conducted. You can also test for trends across categories.

The statistical operations were done through SPSS (Statistical Presentation System Software) for Windows, version 10.0 (SPSS, 1999. SPSS Inc: New York).

OBSERVATION

52 patients presenting to PEOPLE'S COLLEGE OF MEDICAL SCIENCES &RESEARCH CENTRE BHOPAL, INDIA with perforative peritonitis were studied.

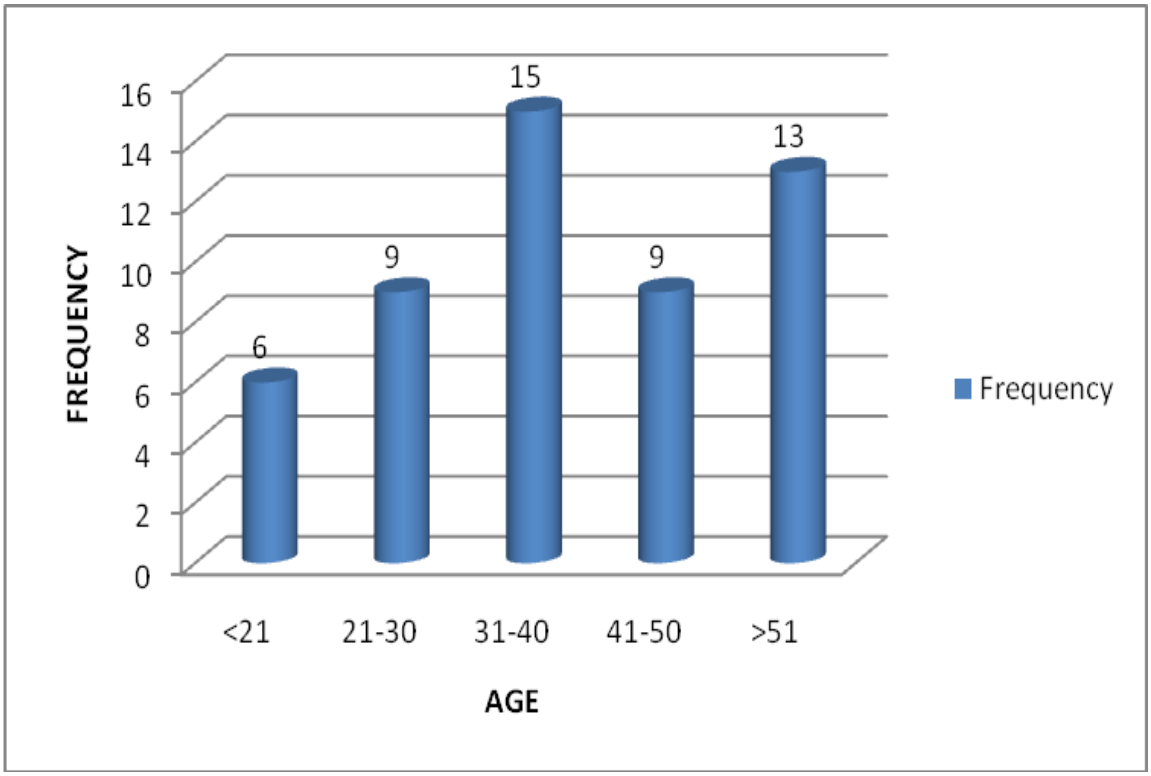
The frequency of anatomical site of perforation is as follows.

TABLE -6

AGE DISTRIBUTION

Age (years)	Frequency	Percent
<21	6	11.5
21-30	9	17.3
31-40	15	28.8
41-50	9	17.3
>51	13	25.0
Total	**52**	**100.0**

GRAPH 1: BAR GRAPH SHOWING AGE DISTRIBUTION



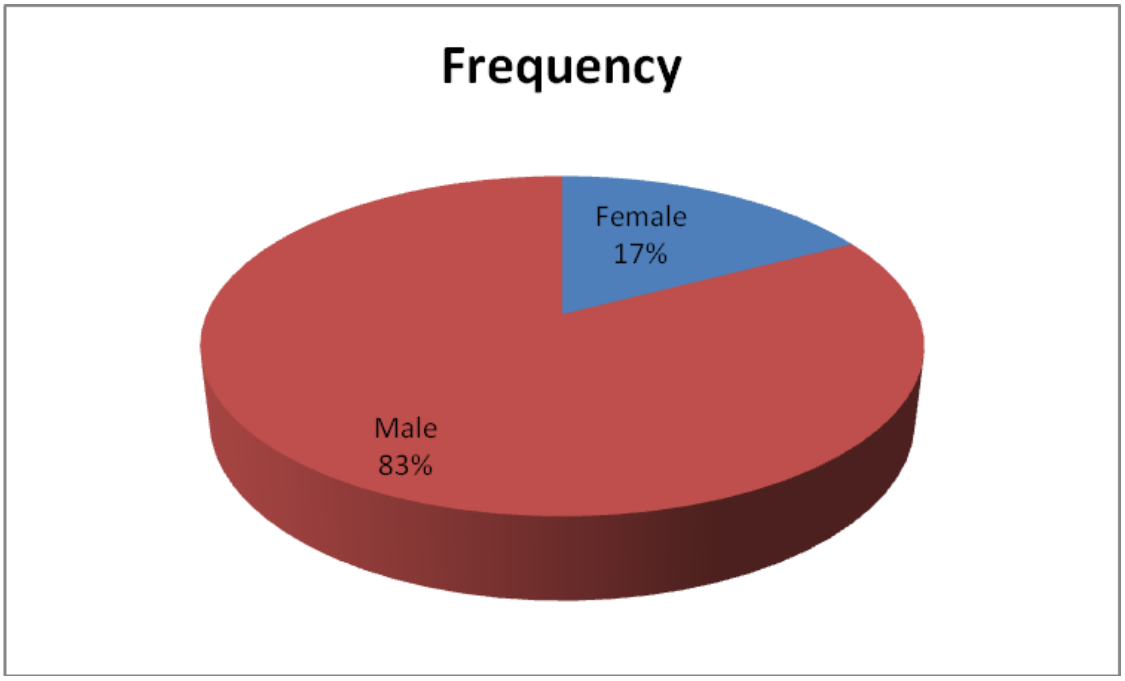
The highest number of patients was seen in the age group above 31-40 years, irrespective of the pathological conditions followed by >51 year age group. In this study youngest patient was 11 years and the oldest was 65 years.

TABLE -7

GENDER DISTRIBUTION

Gender	Frequency	Percent
Female	9	17.3
Male	43	82.7
Total	**52**	**100.0**

GRAPH 2: PIE GRAPH SHOWING GENDER DISTRIBUTION



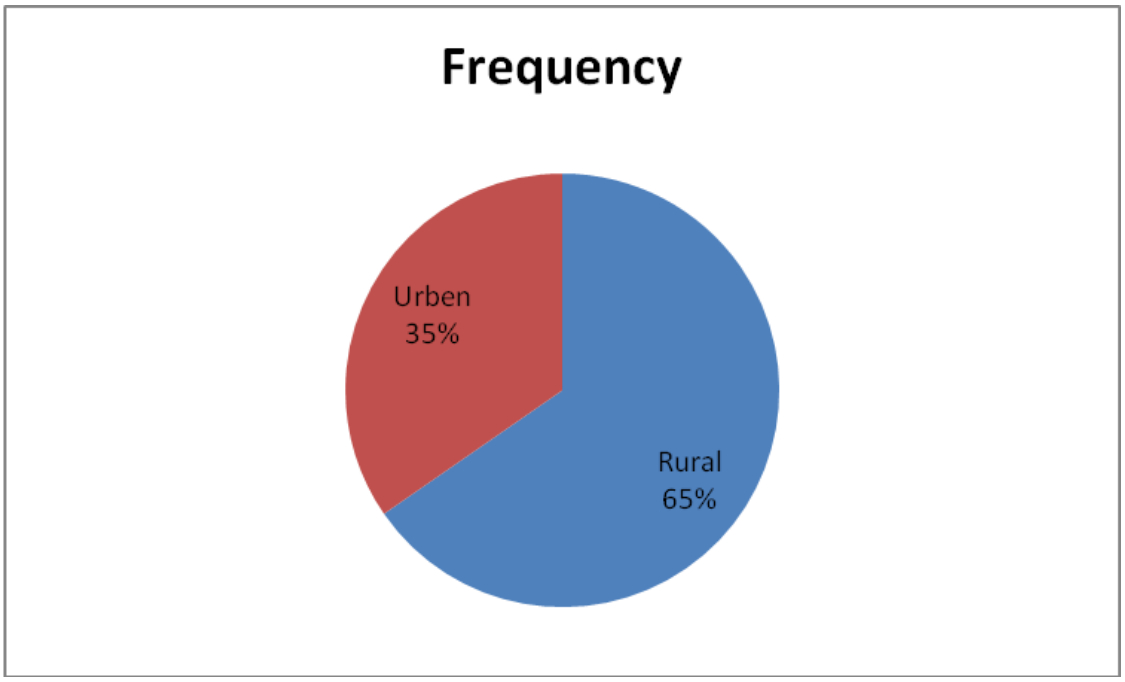
Perforation is more common in males i.e. 43(82.7%)

TABLE -8

POPULATION DISTRIBUTION

Population distribution	Frequency	Percent
Rural	34	65.4
Urben	18	34.6
Total	52	100.0

GRAPH 3: PIE GRAPH SHOWING POPULATION DISTRIBUTION



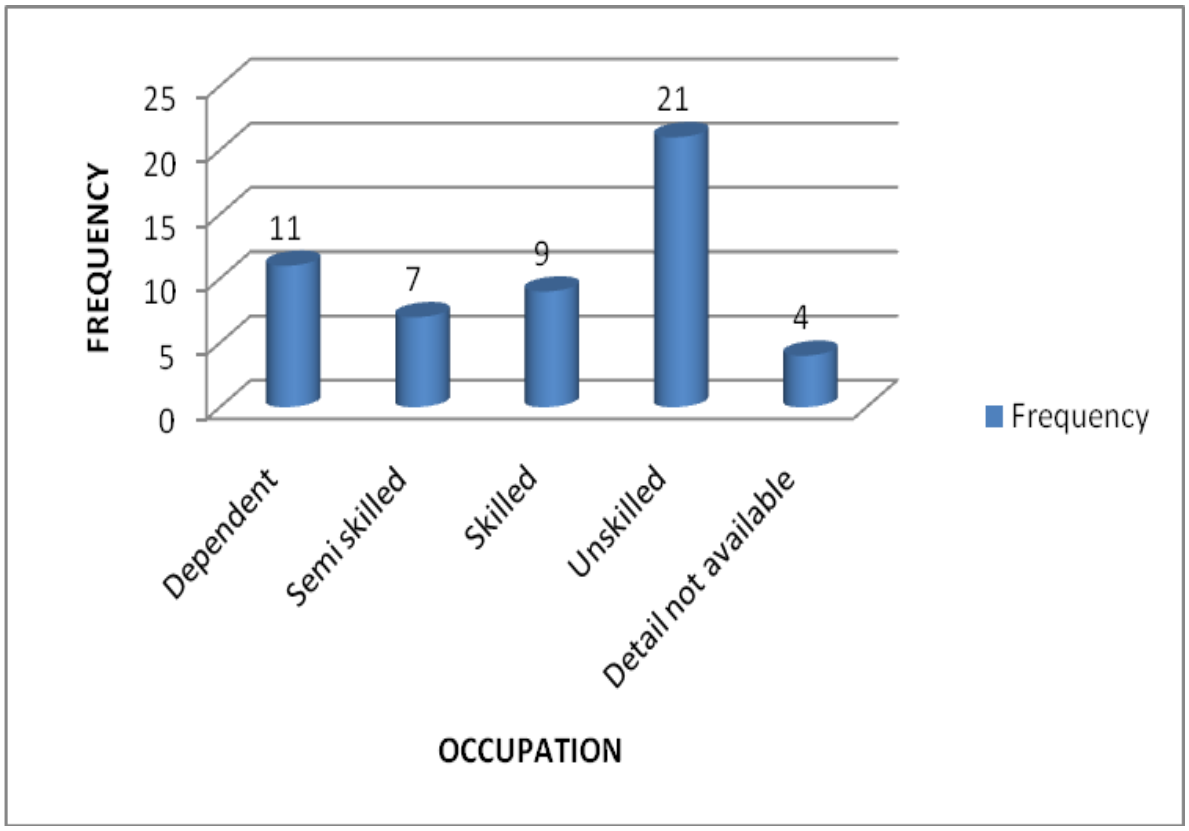
Perforation is more common in rural i.e. 34(65.4%)

TABLE -9

OCCUPATION DISTRIBUTION

Occupation skill	Frequency	Percent
Dependent	11	21.2
Semi skilled	7	13.5
Skilled	9	17.3
Unskilled	21	40.4
Detail not available	4	7.7
Total	**52**	**100.0**

GRAPH 4: BAR GRAPH SHOWING OCCUPATION DISTRIBUTION



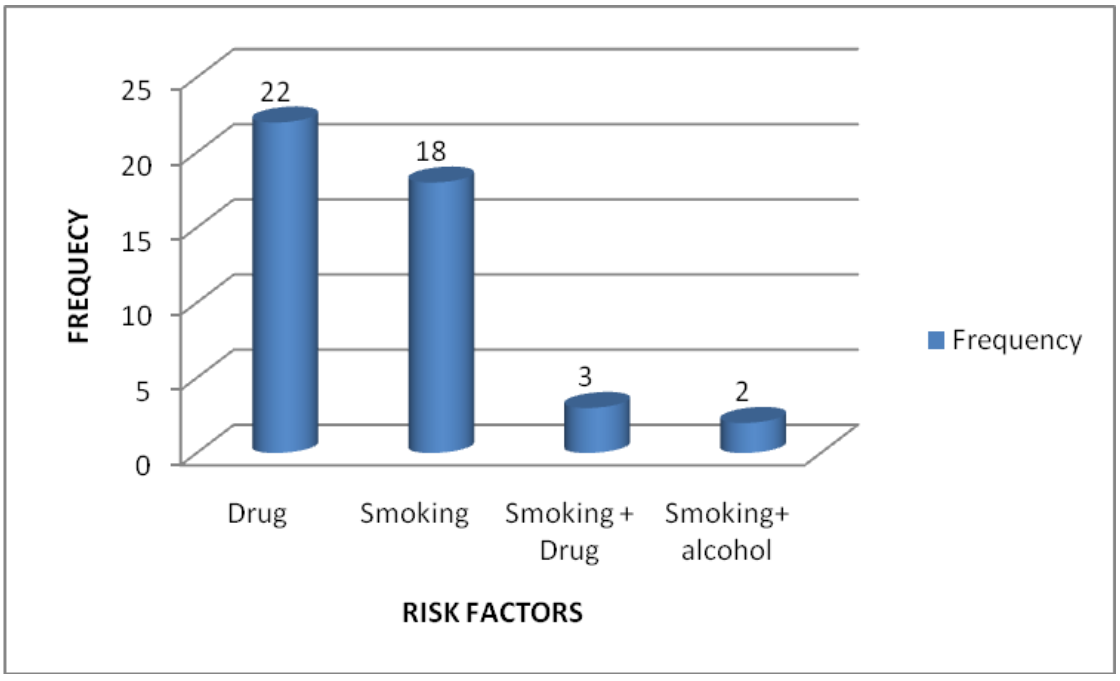
Perforation is more common in unskilled workers.

TABLE -10

RISK FACTORS

Risk factors	Frequency	Percent
Drug	22	42.30
Smoking	18	34.61
Smoking + Drug	3	5.8
Smoking+ alcohol	2	3.8
NO	7	13.46
Total	52	100.0

GRAPH 5: BAR GRAPH SHOWING RISK FACTORS



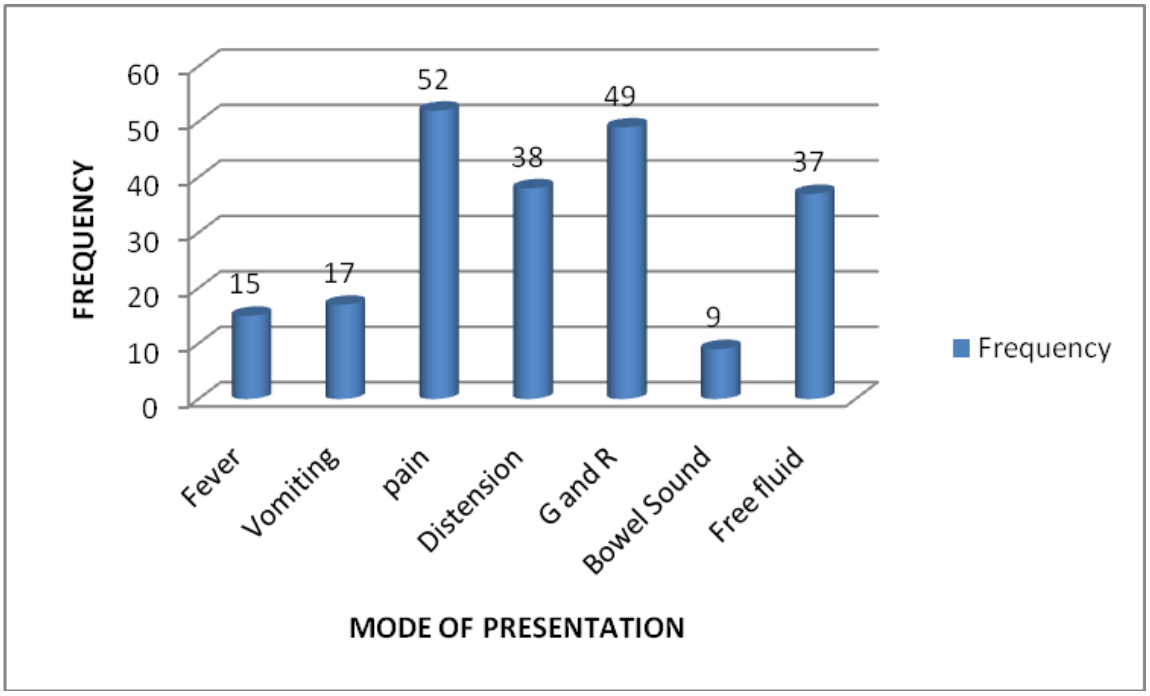
Among 52 patients 22 patients had history of drug followed by 18 patients of smoking.

TABLE -11

MODE OF PRESENTAION : SYMPTOMS & SIGNS

Symptoms	Frequency	Percentage
Fever	15	28.8
Vomiting	17	32.7
Pain	52	100
Distension	38	73.0
G and R	49	94.2
Bowel Sound	9	17.3
Free fluid	37	71.15

GRAPH 6: BAR GRAPH SHOWING MODE OF PRESENTAION



Pain was the presenting symptom in all cases and onset was acute in all of them. In most cases pain was situated at epigastrium , and right hypochondrium.

Guarding and rigidity was also present in most of cases.

Distension was most commonly observed in cases with >24hrs .history of symptoms in all cases after 48 hrs.

Free fluid was present in 37 cases.

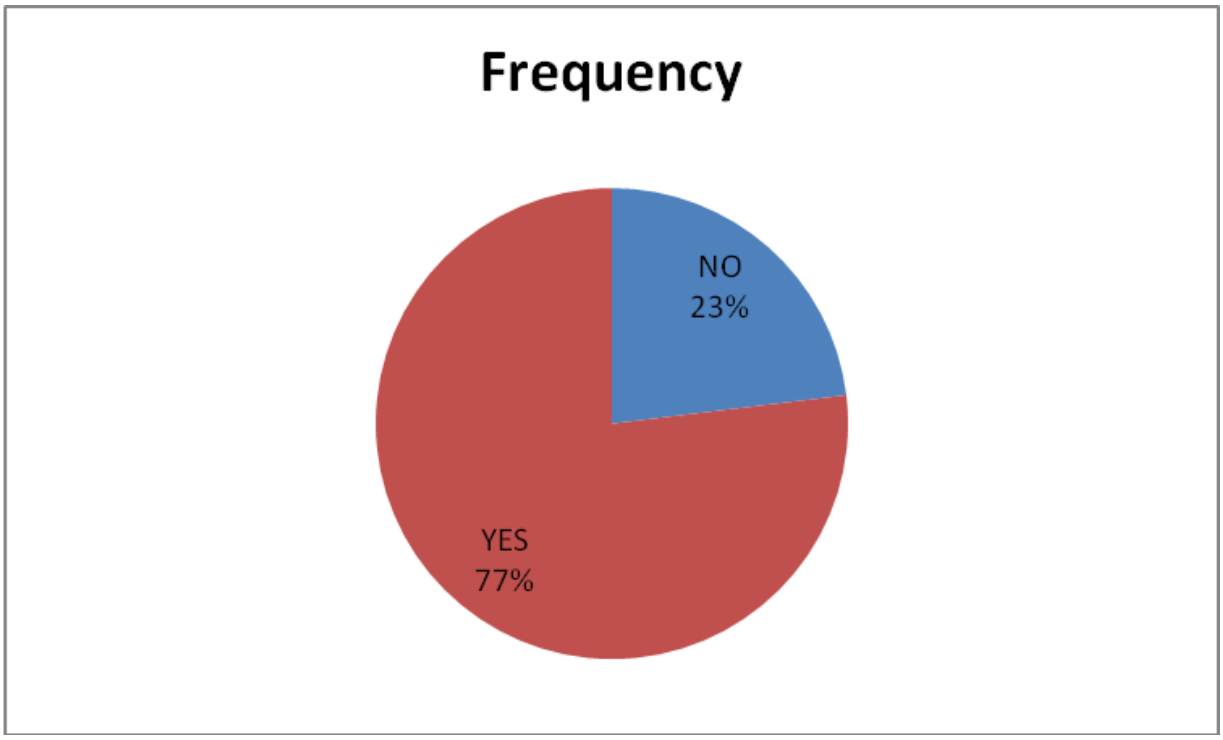
Vomiting was present in 17 cases started along with pain abdomen and contained food particles and bile.

TABLE -12

X-RAY (PNEUMOPERITONEUM)

X-ray (PNEUMOPERITONEUM)	Frequency	Percentage
NO	12	23.1
YES	40	76.9
Total	52	100.0

GRAPH 7: PIE GRAPH SHOWING X-RAY (PNEUMOPERITONEUM)



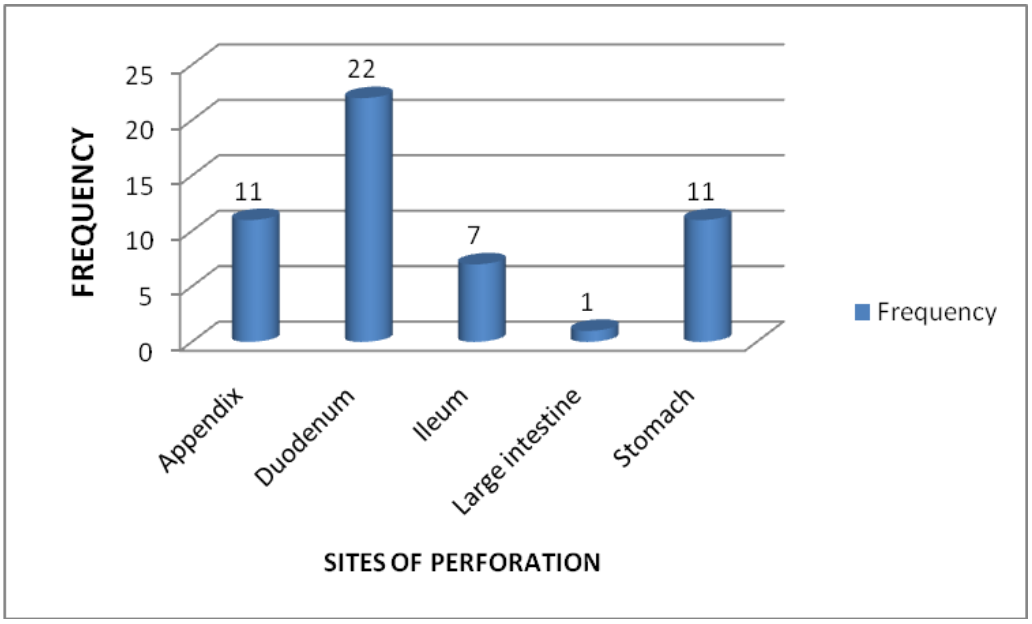
In the present study gas under diaphragm was seen in 40 cases (77 %)

TABLE -13

ANATOMICAL SITES OF PERFORATION

Anatomical site	Frequency	Percent
Appendix	11	21.2
Duodenum	22	42.3
Ileum	7	13.5
Large intestine	1	1.9
Stomach	11	21.2
Total	52	100.0

GRAPH 8: BAR GRAPH SHOWING ANATOMICAL SITES OF PERFORATION



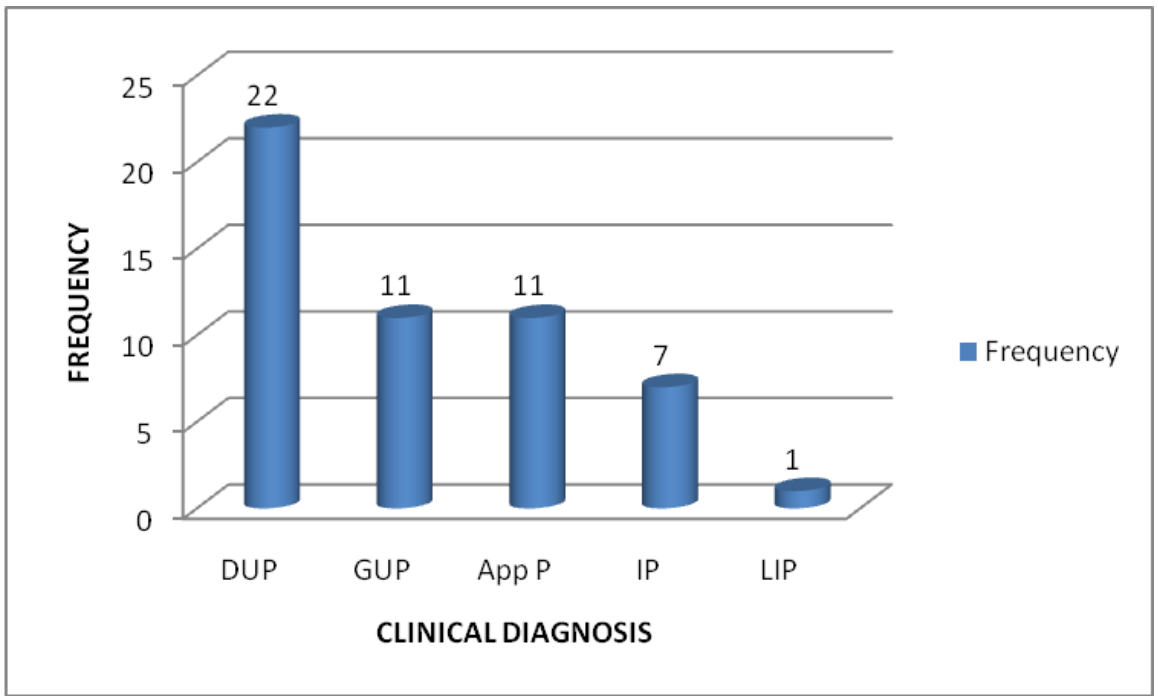
The commonest site involved in gastrointestinal perforation in this study was duodenal ulcer perforation (42.3%) . appendicular and ileal perforation were equal in frequency (21.1%).

TABLE -14

CLINICAL DIAGNOSIS

DIAGNOSIS	Frequency	Percent
DUP	22	42.3
GUP	11	21.2
App P	11	21.2
IP	7	13.5
LIP	1	1.9
Total	**52**	**100**

GRAPH 9: BAR GRAPH SHOWING CLINICAL DIAGNOSIS



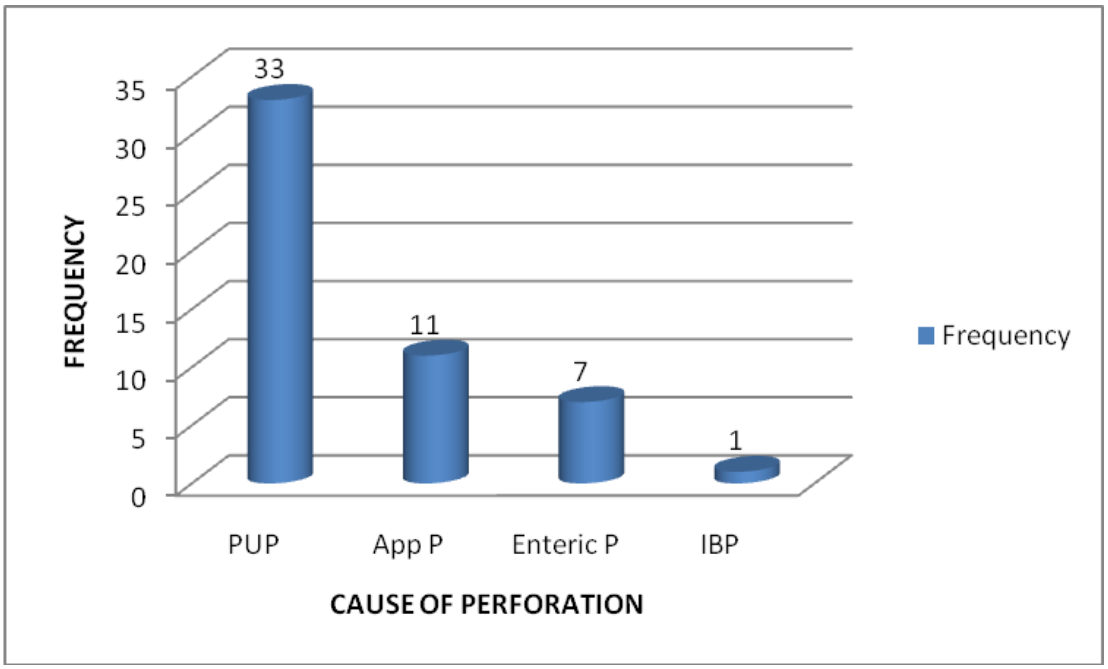
Most common clinical diagnosis were duodenal ulcer perforation (42.3%) followed by equal cases of Gastric & Appendicular perforation (21.2).

TABLE -15

CAUSE OF PERFORATION

CAUSE OF PERFORATION	Frequency	Percent
PUP	33	63.5
App P	11	21.2
Enteric P	7	13.5
IBP	1	1.9
Total	**52**	**100**

GRAPH 10: BAR GRAPH SHOWING CAUSE OF PERFORATION



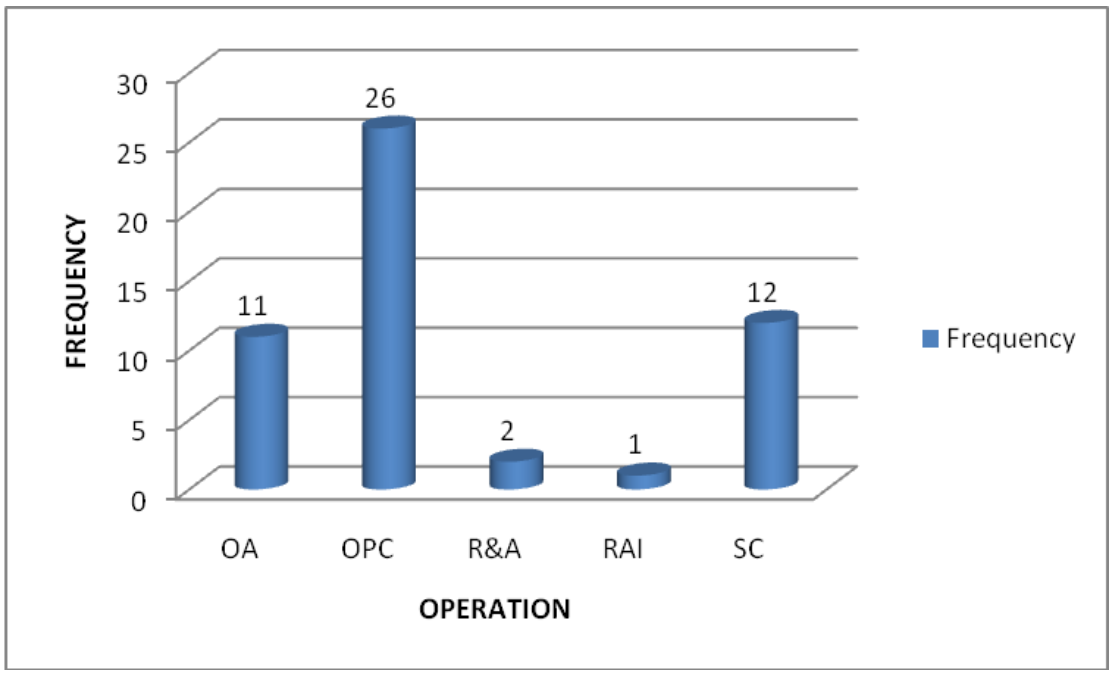
In this study peptic ulcer is the commonest cause of perforative peritonitis followed by appendicitis and typhoid fever.

TABLE -16

OPERATIONS

OPERATIONS	Frequency	Percent
OA	11	21.2
OPC	26	50
R&A	2	3.8
RAI	1	1.9
SC	12	23.1
Total	**52**	**100**

GRAPH 11: BAR GRAPH SHOWING OPERATIONS



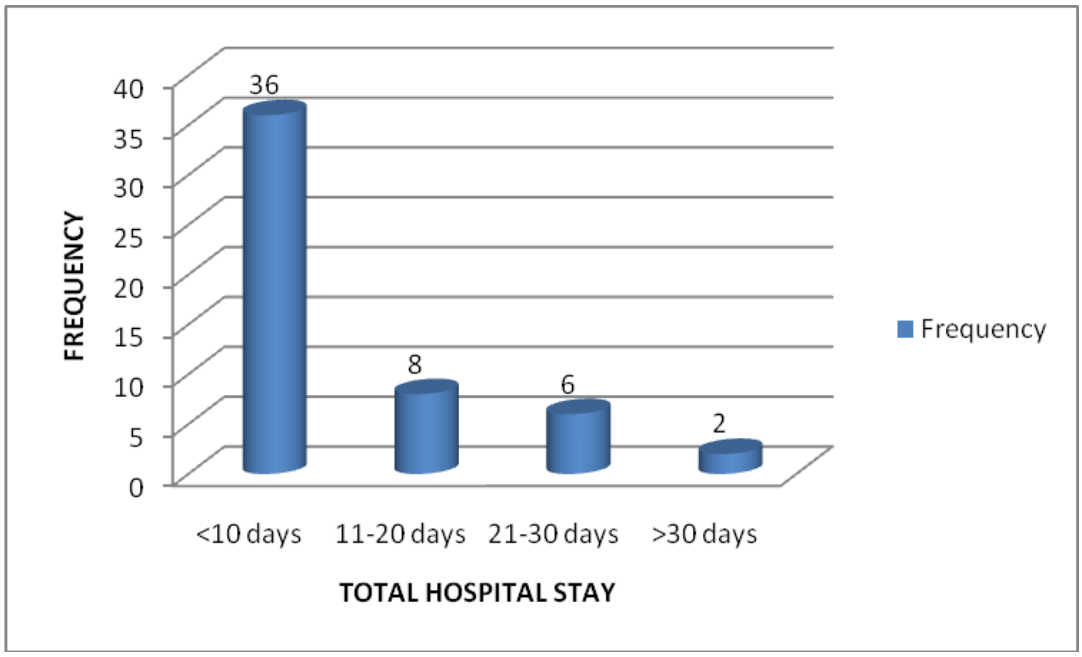
More number of cases were operated through omental patch closure (50%) and followed by simple closure (21.2%) and least number of cases was operated by resection anastomosis with covering ileostomy.

TABLE -17

DURATION OF STAY

Total hospital stay in days	Frequency	Percent
<10	36	69.2
11-20	8	15.4
21-30	6	11.5
>30	2	3.8
Total	52	100.0

GRAPH 12: BAR GRAPH SHOWING TOTAL HOSPITAL STAY



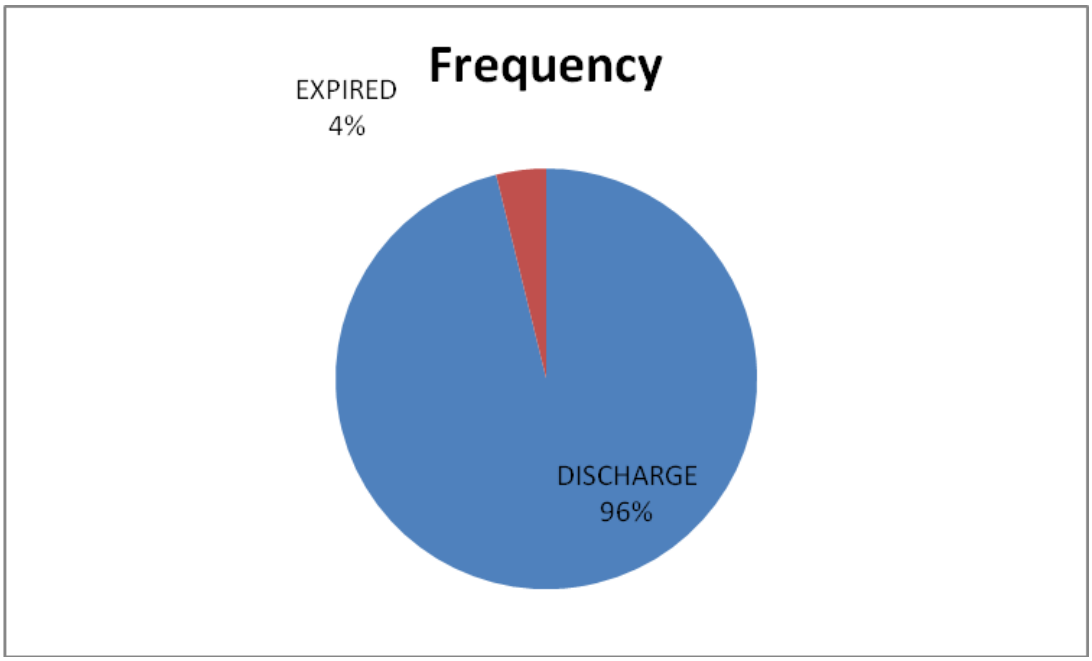
Poor risk patients, stayed for prolong period in their postoperative period, because of various complications.

TABLE -18

OUTCOME

Outcome	Frequency	Percent
Discharge	50	96.2
Expired	2	3.8
Total	**52**	**100**

GRAPH 13: PIE GRAPH SHOWING OUTCOME



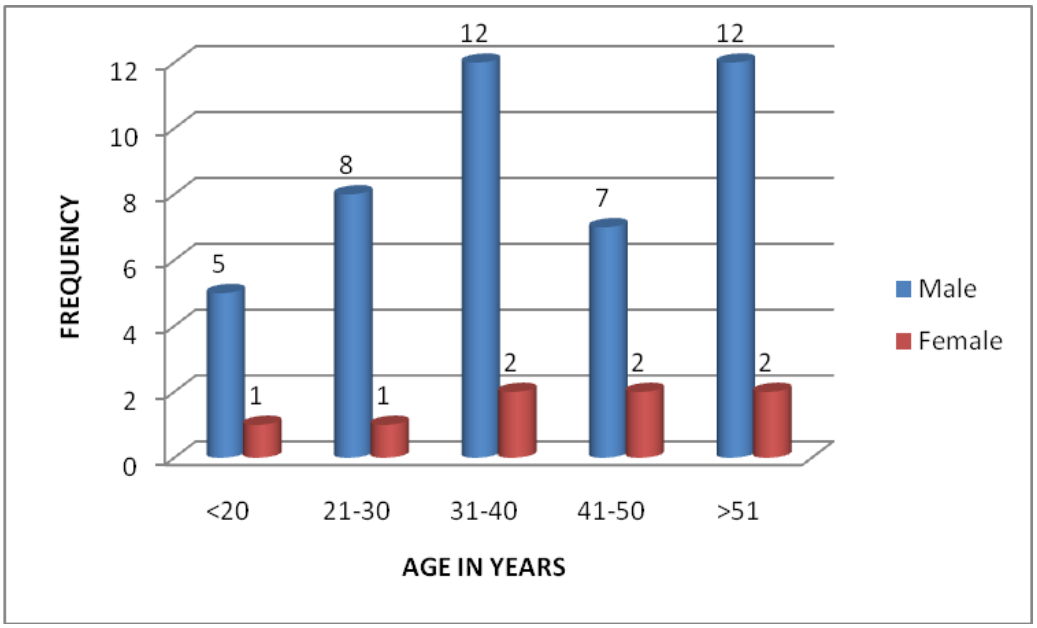
In this study the overall mortality rate irrespective of site and pathology of perforation was 3.8 %. Out of the 52 cases, 2 patients which expired, had ileal perforation.

TABLE -19

DISTRIBUTION OF SAMPLE BY AGE AND GENDER

Age group(yrs)		Male	Female	Total
<20	Frequency	5	1	6
	Percent	11.3	12.5	11.5
21-30	Frequency	8	1	9
	Percent	18.1	12.5	17.3
31-40	Frequency	12	2	14
	Percent	27.2	25	26.9
41-50	Frequency	7	2	8
	Percent	15.9	25	15.4
>51	Frequency	12	2	15
	Percent	27.2	25	28.8
Total	**Frequency**	**44**	**8**	**52**
	Percent	**100**	**100**	**100**

GRAPH 14: BAR GRAPH SHOWING DISTRIBUTION OF SAMPLE BY AGE & GENDER



The highest number of patients was seen in the age group above 31-40 years, irrespective of the pathological conditions followed by >51 year age group. In this study youngest patient was 11 years and the oldest was 65 years. A non significant association (CC-.0.1103, P<.998) between age groups and sex.

TABLE -20

DISTRIBUTION OF AGE & CAUSE OF PERFORATION

Age	Cause Of Perforation				Total
	PUP	App P	Enteric p	IBP	
<21 Years	2	4	0	0	6
	33.30%	66.70%	0.00%	0.00%	100.00%
21-30 Years	5	4	0	0	9
	55.60%	44.40%	0.00%	0.00%	100.00%
31-40 Years	11	2	2	0	15
	73.30%	13.30%	13.30%	0.00%	100.00%
41-50 Years	7	1	1	0	9
	77.80%	11.10%	11.10%	0.00%	100.00%
>51 years	8	0	4	1	13
	61.50%	0.00%	30.80%	7.70%	100.00%
Total	**33**	**11**	**7**	**1**	**52**
	63.50%	**21.20%**	**13.50%**	**1.90%**	**100.00%**

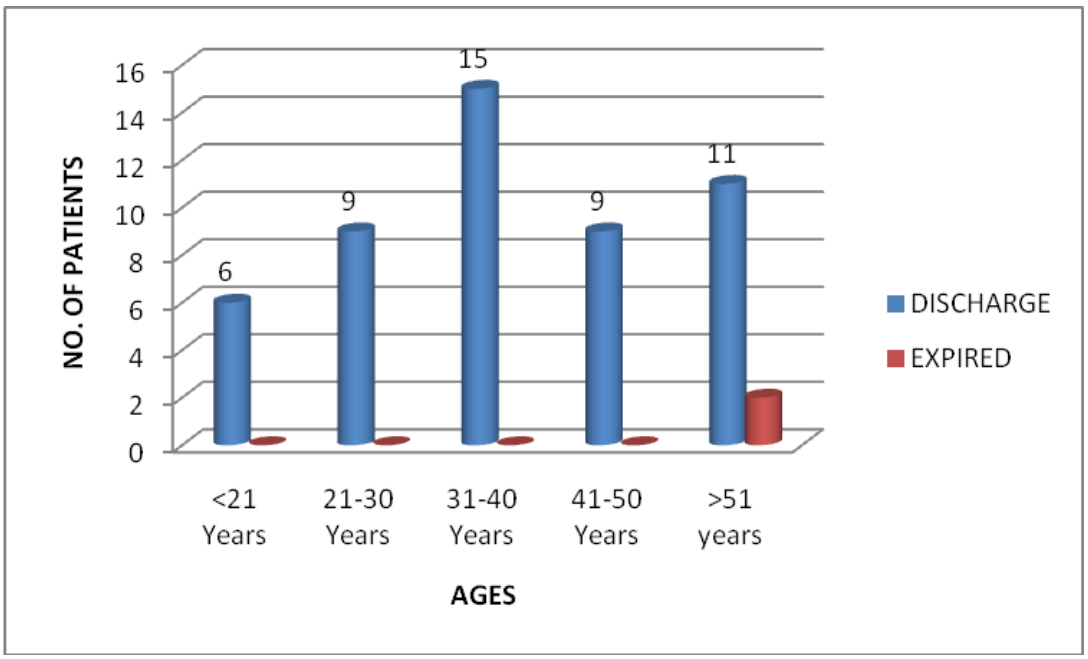
The chi – square statistic is 21.221. The P-value is 0.047 (significant)

TABLE -21

CORRELATION BETWEEN AGE & OUTCOME

Age(years)	Outcome	
	Discharge	Expired
<21 Years	6	0
21-30 Years	9	0
31-40 Years	15	0
41-50 Years	9	0
>51 years	11	2
Total	50	2
Percent	96.20%	3.80%

GRAPH 15: BAR GRAPH SHOWING CORRELATION BETWEEN AGE & OUTCOME



The chi – square statistic is 6.240. The P-value is 0.182 (Non significant)

TABLE -22

CORRELATION BETWEEN AGE & POST OF COMPLICATION

Age	POC					Total
	ARDS+ARF+S	LRTI	WD	WI	WI+ARF+S	(Percent)
<21 Years	0	0	0	0	0	0(0.00%)
21-30 Years	0	0	0	0	0	0(0.00%)
31-40 Years	0	1	2	0	0	3(5.78%)
41-50 Years	0	2	1	1	0	4(7.7%)
>51 years	1	2	0	1	1	5(9.61%)

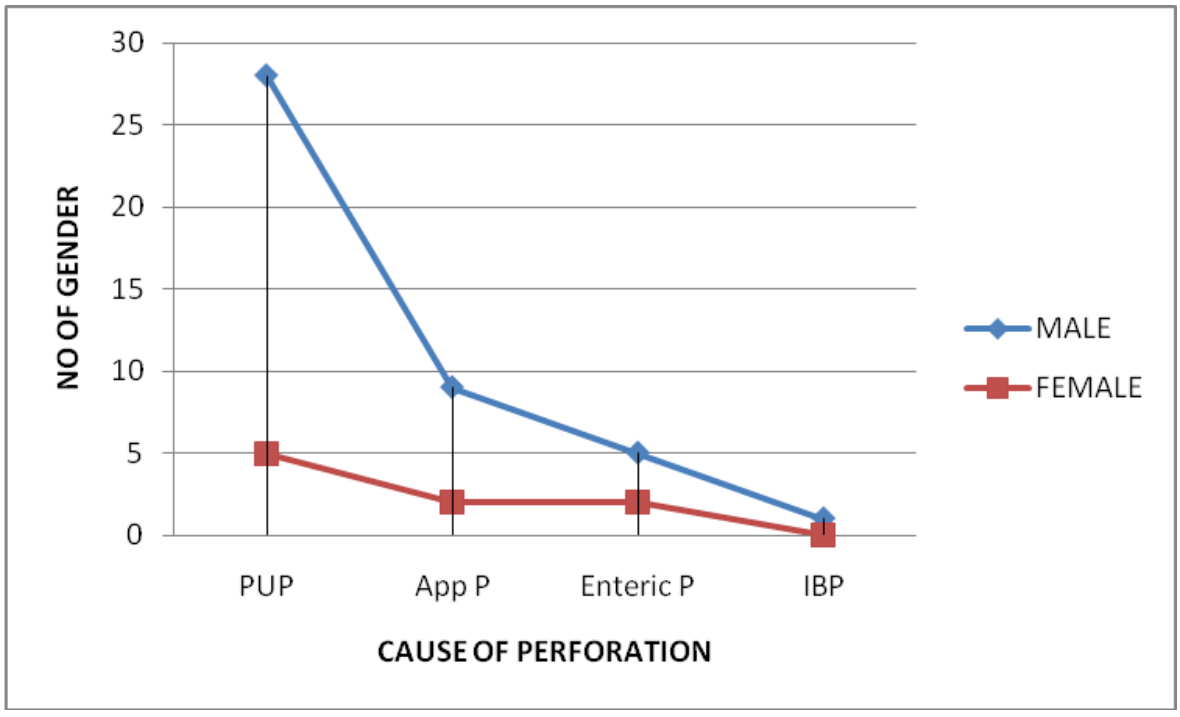
The chi – square statistic is 17.944. The P-value is 0.591 (nonsignificant)

TABLE -23

DISTRIBUTION OF CAUSE OF PERFORATION & GENDER

Cause of Perforation	Gender				
	Male	Percent	Female	Percent	Total
PUP	28	65.10%	5	55.60%	33
App P	9	20.90%	2	22.20%	11
Enteric P	5	11.60%	2	22.20%	7
IBP	1	2.30%	0	0.00%	1
TOTAL	**43**	**82.69%**	**9**	**17.30%**	**52**

GRAPH 16: LINE GRAPH SHOWING DISTRIBUTION CAUSES OF PERFORATION & GENDER



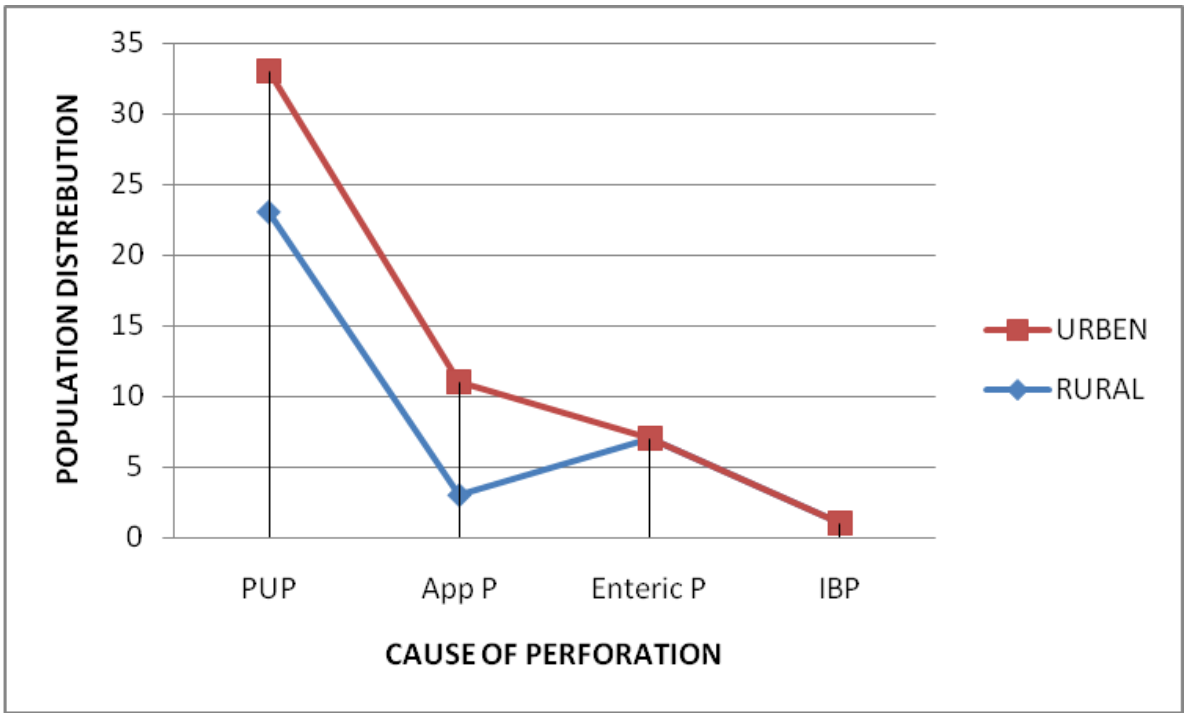
The chi – square statistic is 0.943. The P-value is 0.815 (nonsignificant)

TABLE -24

CORRELATION BETWEEN POPULATION DISTRIBUTION & CAUSE OF PERFORATION

Cause of Perforation	Population Distribution				
	Rural	Percent	Urban	Percent	Total
PUP	23	67.6%	10	55.6%	33
App P	3	8.8%	8	44.4%	11
Enteric P	7	20.6%	0	0.00%	7
IBP	1	2.9%	0	0.00%	1
TOTAL	**34**	**65.38%**	**18**	**34.16%**	**52**

GRAPH 17: LINE GRAPH SHOWING CORRELATION BETWEEN POPULATION DISTRIBUTION & CAUSE OF PERFORATION



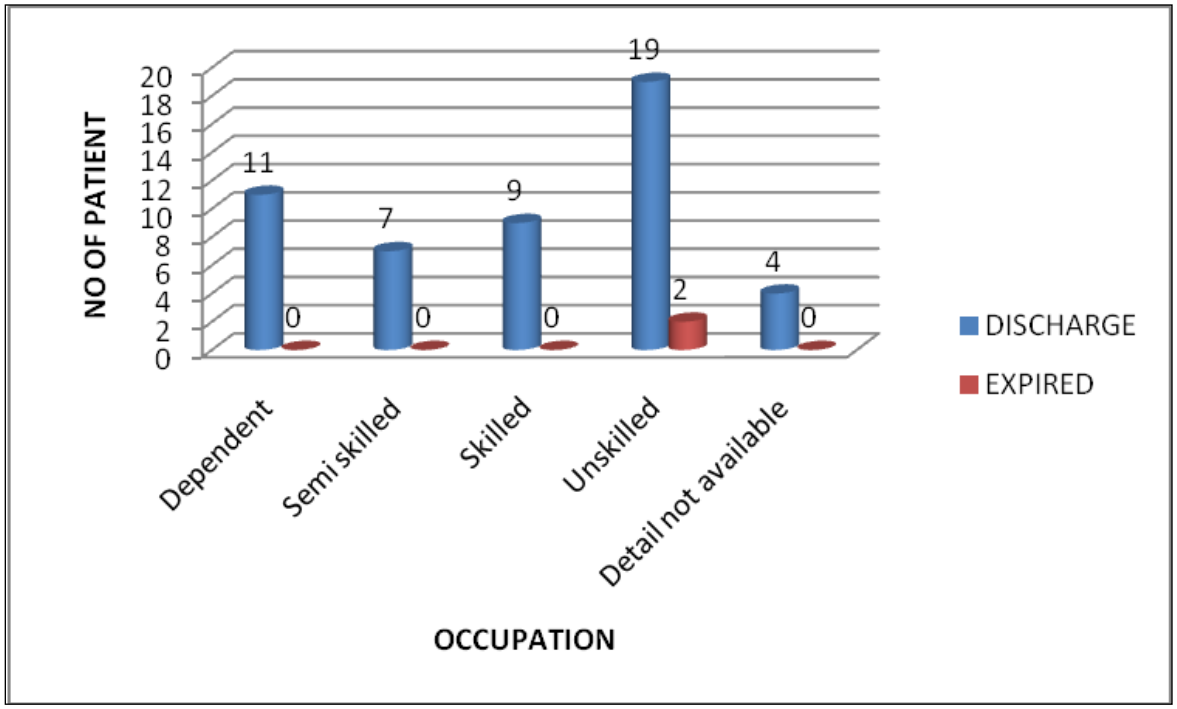
The chi – square statistic is 11.566. The P-value is 0.009 (significant)

TABLE -25

CORRELATION BETWEEN OCCUPATION & OUTCOME

Occupation	Outcome	
	Discharge	Expired
Dependent	11	0
Semi skilled	7	0
Skilled	9	0
Unskilled	19	2
Detail not available	4	0
Total	50	2

GRAPH 18: BAR GRAPH SHOWING CORRELATION BETWEEN OCCUPATION & OUTCOME



The chi – square statistic is 3.070. The P-value is 0.546 (Non significant)

TABLE -26

CORRELATION OF DURATION OF SYMPTOMS & MORBIDITY AND MORTILITY

Duration of symptoms	Number of patients	No. of Morbidity& Mortality	% of morbidity& Mortality
<12 hours	11	0	0
12 -24hours	16	0	0
24-48 hours	15	1	6.6
48-72 hours	8	3	37.5
>72 hours	2	2	100
Total	52	6	11.5
Inference	Incidence of morbidity and mortality is significantly associated with duration of symptoms>48 hours with p<0.001		

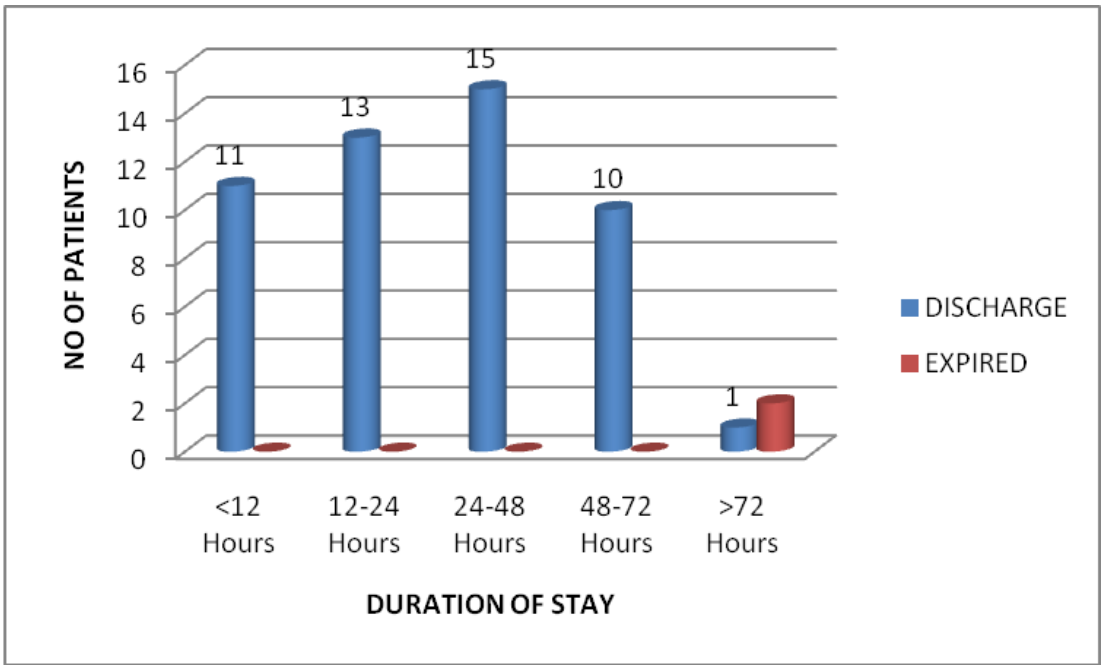
Large group of patients had delayed presentation, which had significant impact on increased morbidity and mortality of the patient.

TABLE -27

CORRELATION BETWEEN DURATION OF PRESENTATION & OUTCOME

	Outcome	
Duration Of Presentation	**Discharge**	**Expired**
<12 Hours	11	0
12-24 Hours	13	0
24-48 Hours	15	0
48-72 Hours	10	0
>72 Hours	1	2
Total	50	2
Percent	96.20%	3.80%

GRAPH 19: BAR GRAPH SHOWING CORRELATION BETWEEN DURATION OF PRESENTATION & OUTCOME



The chi – square statistic is 33.973. The P-value is 0.000 (significant)

TABLE -28

DISTRIBUTION OF SITE OF PERFORATION WITH POST OPERATIVE COMPLICATIONS

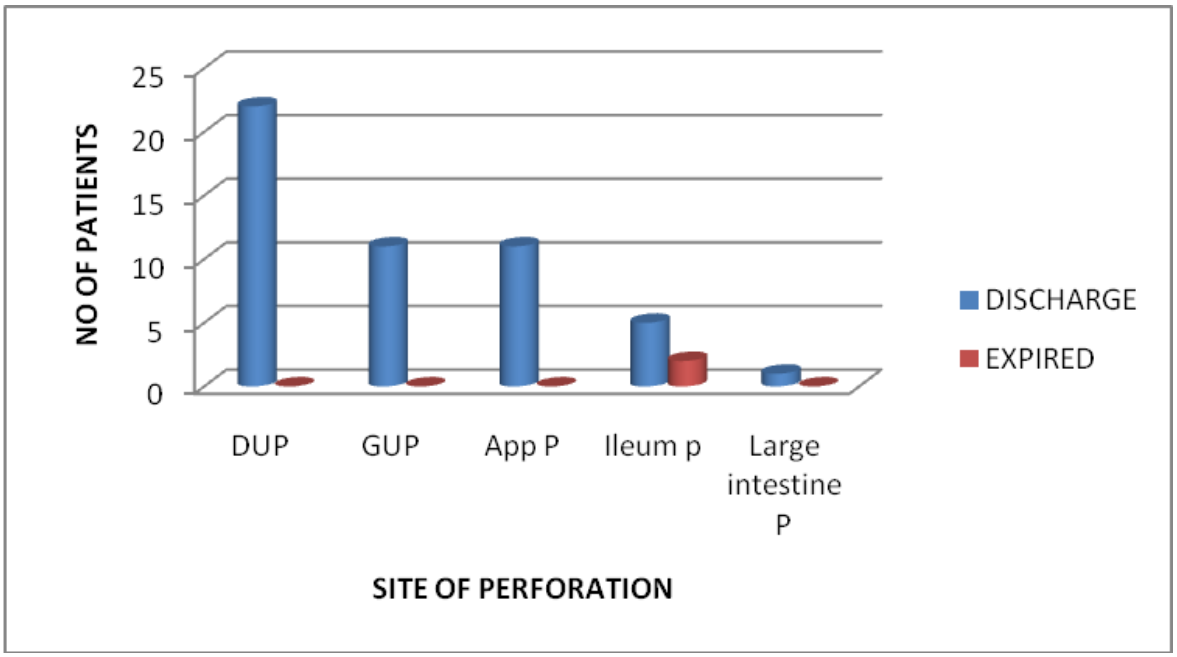
Post op complications	DUP	GUP	App P	Ileum P	Large intestine P	Total
WI	0	0	1	1	1	3
LRTI	4	1	0	0	0	5
ARF	0	0	0	2	0	2
WD	0	0	0	2	0	2
ARDS	0	0	0	1	0	1
Septicemia	0	0	0	2	0	2
Nil	22	10	8	1	0	41
TOTAL	**26**	**11**	**9**	**5**	**1**	**52**

TABLE -29

CORRELATION BETWEEN SITE OF PERFORATION & OUTCOME

Site Op Perforation	Outcome	
	Discharge	Expired
DUP	22	0
GUP	11	0
App P	11	0
Ileum p	5	2
Large intestine P	1	0
Total	**50**	**2**

GRAPH 20: BAR GRAPH SHOWING CORRELATION BETWEEN SITE OF PERFORATION & OUTCOME



The chi – square statistic is 13.371. The P-value is 0.010 (significant)

Post operatively 9 patients had one or other complications.

Lower respiratory tract infection (LRTI) was the most common complication observed. LRTI patients presented post operatively with fever, cough with expectoration and on chest X ray showed consolidation changes.

Wound infection was present in 2 cases of appendicular perforation large intestine perforation, manifested by pain at the wound site and discharge. Patient was managed by draining out the subcutaneous collection and antibiotics.

Wound dehiscence was observed in 1cases. Secondary suturing was done after controlling wound infection.

One patient of ileal perforation developed ARF with septicaemia was a 58 year old male. Post operatively patient was put in intensive care unit with ventilator support but expired.

One patient of ileal perforation developed wound infection, Acute Respiratory distress syndrome (ARDS) & later developed wound dehiscence and had signs and symptoms of septicaemia with renal failure was a 65 years old male known case of chronic obstructive pulmonary disease (COPD) and a chronic smoker .Post operatively patient was put in intensive care unit with ventilator support but expired.

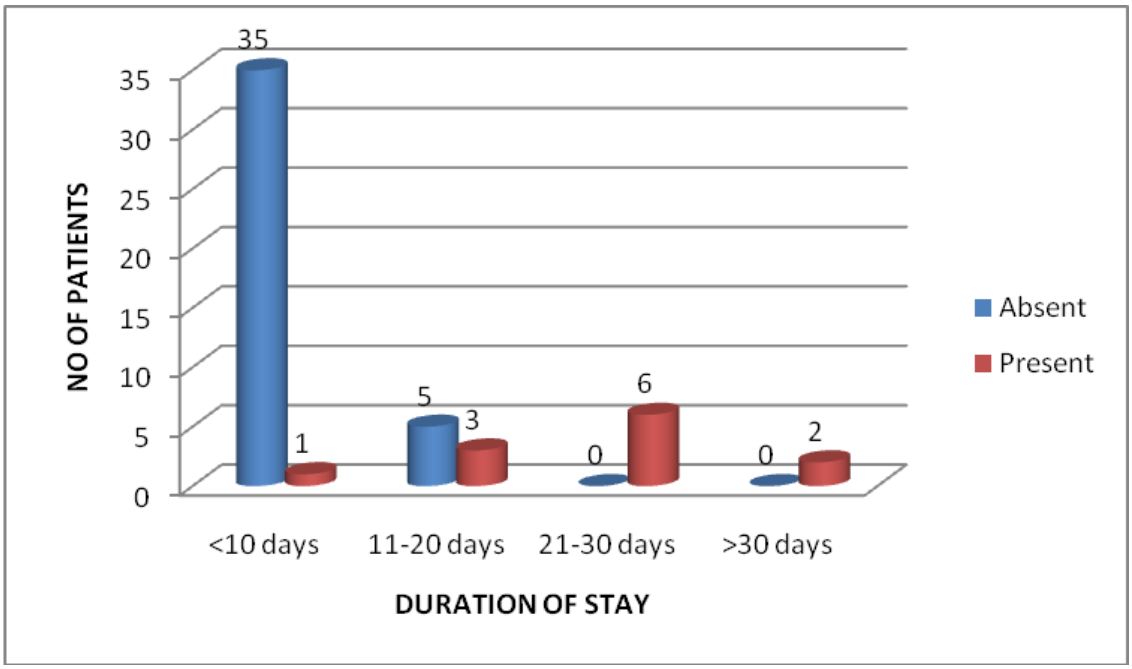
In this study the overall mortality rate irrespective of site and pathology of perforation was 3.8 %. Out of the 52 cases , 2 patients which expired, had ileal perforation.

TABLE -30

CORRELATION BETWEEN DURATION OF STAYAND POSTOPERATIVE COMPLICATIONS

Duration of hospital stay	Complications	
	Absent	Present
<10 days	35	1
11-20 days	5	3
21-30 days	0	6
>30 days	0	2
Total	**40(100.0%)**	**12(100.0%)**

GRAPH 21: LINE GRAPH SHOWING CORRELATION BETWEEN DURATION OF STAY AND POSTOPERATIVE COMPLICATIONS



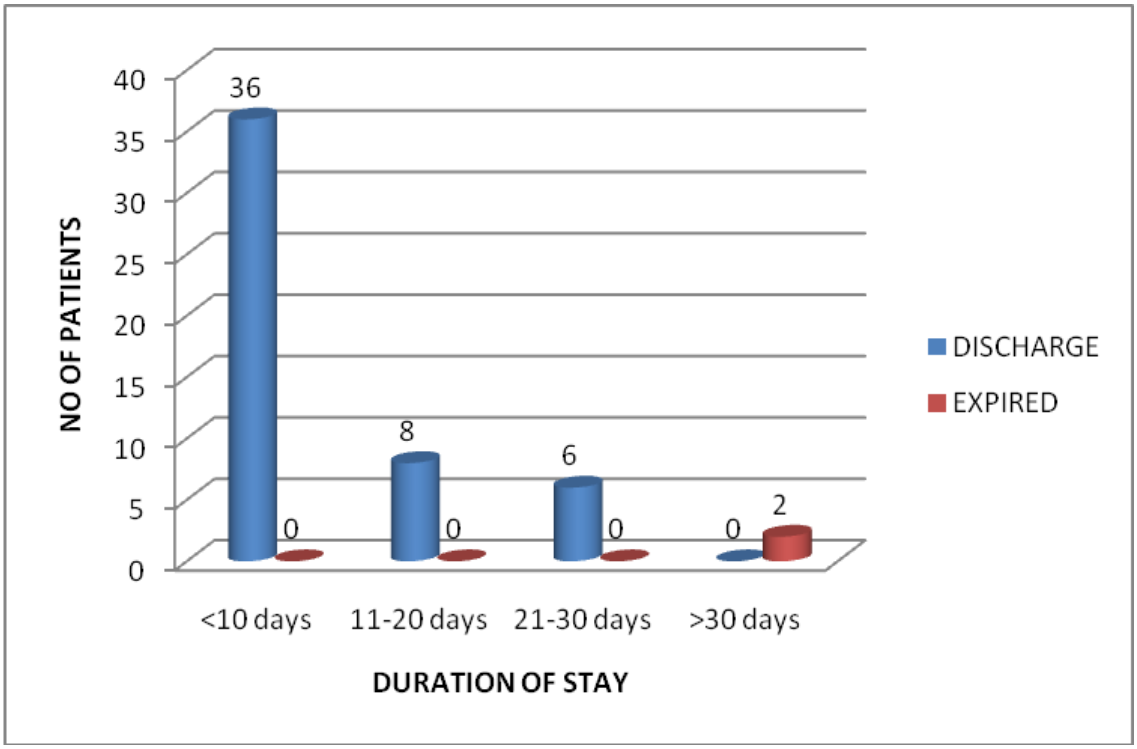
Duration of hospital stay is significantly more in patients with complications with p<0.001

TABLE -31

CORRELATION BETWEEN DURATION OF STAY & OUTCOME

Duration of Stay	Outcome	
	Discharge	Expired
<10 days	36	0
11-20 days	8	0
21-30 days	6	0
>30 days	0	2
Total	**50**	**2**

GRAPH 22: BAR GRAPH SHOWING CORRELATION BETWEEN DURATION OF STAY & OUTCOME



The chi – square statistic is 52.00. The P-value is 0.000 (significant)

TABLE -32

COMPARISON OF THE PRESENT SERIES WITH OTHER STUDIES

Author	Year	Mortality Rate (%)
Swayer	1977	6.7
M.C.Dandapat	1991	10.5
Ramesh C.Bharti	1996	4
Present series	2014	3.8

Our study was showing mortality 3.8% which is equal to Ramesh c. bharti study.

QUALITATIVE ANALYSIS

All patients of perforative peritonitis were treated as a surgical emergency. Preoperatively all patients had broad spectrum antibiotic coverage, nasogastric suction and management of fluid and electrolyte imbalance. Anemic patients required blood transfusion. Post operatively parenteral antibiotics were continued and after that oral antibiotics were given for 5 days.

Perforation peritonitis is more common in 3rd & 5th decade of age but age is no bar for perforation to occur. In this study youngest patient was 11yrs & the oldest was 65 yrs. Perforation is more common in males than females. Also perforation was more common in rural and unskilled population.

Drug (NSIDS) & smoking were leading risk factor of perforation irrespective to site of perforation.

26 cases of perforation underwent closure as described by Graham (Omental patch closure).Size of the perforation measured from 5-10mm. Double layer closure of the enteric perforation was done in 12 cases using 2-0 vicryl and 2-0 silk. One case of multiple ileal perforation underwent resection and anastomosis with covering ileostomy

One case of gangrene bowel with perforation was met within the study. Patient presented with 4 days history of periumblical pain, with history of diarrhoea and vomiting (so treated as gastroenteritis) but after 4days patient developed distension with guarding. At laparotomy there was gangrene of terminal ileum with perforation. Resection of terminal ileum with end to end anastomosis was done.

Of the 11 cases of perforative appendicitis underwent open appendicectomy.

In all cases of peritonitis thorough lavage was given with 0.9 % saline and drains were kept in the pelvis ,which was usually removed on the 2nd post operative day or drainage < 100 ml .Nasogastric tube was usually removed on the 4th -5th postoperative

day and started orally on the 5th day depending on bowel sounds. All patients were started on chest physiotherapy from the first post operative day.

DISCUSSION

This study was conducted in P.C.M.S. &R.C, Hospital, BHOPAL. A total of 52 patients admitted with particular criteria fixed during the study period were taken as the universe and cases were selected by Purposive sampling.

Age seem to be the important factor in determining the outcome. Extremes of age had increased mortality rates. Factors like decreased functional reserve, concomitant other illness such as diabetes, hypertension seem to be the cause of increase mortality in elderly patients. In our studies male to female ratio is 4.7%.

SHOWING PEAK AGE INCIDENCE BY VARIOUS AUTHORS

Authors	Peak age in years
Palanivelu et al (2007)	20 –30
Philipo.l. Chalya et al (2011)	40 – 50
Present series	31-40,>50

SavnesC[83] has reported that the lethality is higher in the elderly reported that age of a patient, rather than the type of surgery which influences the mortality in perforation peritonitis.

In our studied series 82.7%were males and %were females, and the male-female ratio being 4.8:1. Perforation is more common in males than females, because males were subjected to more stress and strain of life and female sex hormone offer some security against perforation as claimed by Skovgaard (1997).[86] High prevalence of perforation is more is in male society as they are more in stress as compared to their female counter parts said by Zahid Amman (2008).[87]

It is fact that enteric fever is more common in males, possibly because of more exposure to infection. Enteric perforation is common in the 2nd and 3rd decade of life. Enteric perforation usually occurs in the second and third week of fever .In the present series the maximum incidence of perforation was in the second week of fever followed by those in the first week .Dickson and Cole, Olurin[15] and Purohit[16] reported the majority of perforations in the first week of fever while Eggleston and Santoshi[17] reported 33% in the second week of fever.

SHOWING GENDER INCIDENCE BY VARIOUS AUTHERS

	Male : female ratio
Primose N. Jhon (Baily and Love)	2 : 1
Palanivelu et al (2007)	12.3 : 1
Chalaya et al (2011)	1.3 : 1
Present series	4.8 : 1

It is believed that perforation occurs in those people who are engaged in heavy manual labour. Wair(1996) in relatively 1390 cases in Scotland, found highest incidence in fishermen, farm labourers and heavy manual worker. Less than half the number was professional sedentary occupation.

In our studies, it is noticed that perforations occurred in the patients belonging to poor socioeconomic status and more so in the rural population, who are manual workers (unskilled workers).

The incidence of perforation in urban class was less, because of effective medical treatment and early surgery they seek whenever they suffer from perforation peritonitis.

Hermansson M and Ekedhal A et al[89] showed that smoking increased the risk of perforation to 10-fold in the age group of 15-74 years, and there was highly significant dose-response relationship. Savensetal[83] concluded that smoking is a casual factor for perforation and accounts for a major part of perforations in the population aged>75years. Smoking is a definite risk factor for peptic ulcer perforation. Peritonitis is a life threatening complication. Smoking and use of non steroidal anti inflammatory drugs are important risk factors for perforation.In our study, 18 patients out of 52 patients were smokers.

INCIDENCE OF CAUSAL FACTORS

Authors	Smoking	NSAIDS
Palanivelu et al (2007)	72.5 %	10 %
Chalya et al (2011)	64.3 %	10.7 %
Present series	34.61%	42.30 %

In the present study, pain was present in all cases of perforation, indicating that the pain was the most common symptom. Guarding and Rigidity was present in 94.2% of cases. Out of 52 patients only 9 had a bowel sounds.

Gas under diaphragm in X –ray abdomen standing is an important finding, and helpful in diagnosis.

In this study, the leading cause for peritonitis was peptic ulcer perforation, 33cases (22 cases duodenal ulcer perforation & 11 cases gastric ulcer perforation). This is due to the habit of consumption of high spicy food, smoking, analgesic drugs & alcohol in this region.

The second leading cause was due to appendicular perforation (11 cases). The third most common cause is ileal perforation (7 cases).

Nakeeb A and Fikrya (2002) analyzed that time interval between onset of acute symptoms and surgery was less than or equal to 24 hours mortality rate is 12% and if more than 24 hours the mortality rate is 21%. The mortality risk for a patient who is operated on more than 24 hours after the onset of acute symptoms is 4.9 times to that of a patient operated within 24 hours. So the interval between the time of perforation and surgery has a very strong significance in deciding the mode of treatment i.e. type of surgery to be planned and outcome of the disease (DurrHR, WeisC, 1992).

In our studies most of morbidity & mortality seem in patient reached hospital > 48 hrs after onset of symptoms.

Conventional simple closure of a perforated peptic ulcer necessitates an upper abdominal incision to perform a simple repair. Spontaneous ileal perforation is a serious complication of a variety of diseases. In the developed countries these perforations are mostly because of foreign bodies, radiotherapy, drugs, Crohn's disease, malignancies and congenital malformations .In tropical countries small bowel perforation is commonly encountered surgical emergency .Although tuberculosis is an important cause, the most important is typhoid fever.

In Enteric perforation cases, leucopenia (<4,000 /cu.mm) was present in the majority (61 %) of cases, due to bone marrow depression by enteric toxaemia. Enteric perforations are best managed surgically as it prevents further peritoneal contamination by intestinal contents. Repair of perforation should be the choice of

treatment in enteric perforation because this is a simple, quick and cost effective procedure[18] .Ileostomy should be considered selectively in patients with multiple perforations and unhealthy gut. Resection however may be necessary for multiple perforations. The best possible way to decrease the morbidity and mortality of typhoid perforation is to prevent typhoid fever by improved sanitation and immunization programmes.

Appendiceal perforation is associated with increased morbidity and mortality compared to nonperforating appendicitis. The rate of perforation varies from 16-40%, with a higher frequency occuring in younger age groups (40-57%) and in patients older than 50 years (55-70%) in whom misdiagnosis and delayed diagnosis are common. The majority of the cases in the study were in the young age group(100%). Most of the patients presented with right iliac fossa pain (100 %). In the diagnosis of perforated appendicitis, gray-scale Ultasound is also valuable despite the fact that the perforated appendix may not be visualized in the RLQ. Irregularity and damage of the appendix contour with the presence of periappendiceal fluid and hyperechoic prominent pericecal fat are diagnostic of perforation. In our study the ratio was 4.5:1(male-9 & female -2).

Acute mesenteric ischaemia (A.M.I.) is an abdominal catastrophe that carries high morbidity and mortality rates. Leucocytosis and elevated serum lactate levels are common [22, 23]. Acute abdominal pain is the initial symptom in 85% of patients with A.M.I. A.M.I is characterized by pain that is out of proportion to physical findings. Generalized peritonitis and eventually shock develops if treatment is delayed. Elevated serum amylase is non-specific. Metabolic acidosis occurs only after advanced ischaemia. Plain abdominal X-rays are also non-specific. An ileus pattern, diffuse distension with air-fluid levels, evidence of bowel wall oedema, or even gas in the bowel wall or within mesenteric or portal veins are some of the findings that may allow

a presumptive diagnosis of mesenteric ischaemia. CT scan is not a specific diagnostic study of choice but it is often used to rule out other pathology. However, it exhibits sensitivity and specificity that is found to be higher than conventional radiography. Duplex ultrasonography[24] in experienced hands can be accurate in assessing flow in proximal visceral arteries as well as superior mesenteric and portal veins. The most important diagnostic modality is angiography[25] it confirms the clinical diagnosis and aids in planning specific therapy. However, some argue that it is time consuming, delays treatment and does not provide information that cannot be determined at laparotomy. Due to the short ischaemic tolerance time of the intestine, diagnostic and therapeutic decisions have to be made under extreme time pressure. The principles of treatment are adequate rehydration, broad-spectrum antibiotics and early surgical intervention. Various studies have shown an improved survival following early diagnosis and aggressive management. Surgical techniques involve revascularization techniques and/or bowel resection. At laparotomy, the appearance of the bowel wall may vary from pallor to haemorrhagic infarction. Established infarcted bowel should be resected and a second look procedure planned 24-48 hours later. Revascularization techniques include isolated embolectomy, thrombo endarterectomy bypass techniques and intra-arterial thrombolysis[26-30]. The main difficulty at operation is to predict intestinal recovery and to accurately assess amount of bowel that needs to be resected. Clinical assessment relies on colour, contractility and capillary bleeding, all of which are insensitive[31]. Several methods have been described which can help judge viability. Doppler ultrasonic flow meter may be helpful but results with the laser Doppler system prove to be promising[31].

In our study period we had 2 deaths (3.8%). Mortality is high in patients who was taken late for surgery. Delay in surgery (due to late hospitalization) , advanced age & extensive contamination of the peritoneum were most important factors for mortality.

Follow up:

Most of the patients in our study did not turn up after 3 months followup. So long term outcome of procedure could not be made out.

CONCLUSION

- The prospective study was done on 52 patients in P. C. M. C. & R. C. BHANPUR, BHOPAL(M.P).
- Various factors affecting both mortality and morbidity in peritonitis patients were studied.
- Duodenal ulcer perforation the most common cause of acute abdominal catastrophe, followed by gastric perforation, with male preponderance.
- In our study, we found that perforation peritonitis is a multifactorial disease with smoking (34.6%) and drugs (NSAIDs, 42.3%) being common causative factors.
- The factors significantly related to increased morbidity and mortality are delayed presentation, elderly age group (43%) and size of perforation.
- . Overall male female ratio is 4.8:1.
- Omental patch repair either conventional or laparoscopic remains gold standard treatment for perforation.
- Duodenum (42.3%) is the most common site of perforation followed by gastric perforation (21.2%), appendicular (21.2%), & Ileal perforation (13.5%).
- Peptic ulcer (63.5%) is the most common cause of perforative peritonitis followed by appendicular perforation (21.2%) forms the next commonest cause of perforation. The small intestinal perforation due to perforated typhoid ulcer (13.5%).
- Diagnosis is made clinically and confirmed by the presence of pneumoperitoneum (76.9%) on radiographs.
- Socio-demographic profiles referred to place of residence, education level, occupation, and income which show relation with high incidence and morbidity & mortality of perforation peritonitis.
- Cases of peritonitis carry a high mortality which can be reduced by early diagnosis, risk stratification, appropriate treatment based on risk score.
- Delayed presentation which has important effect on both mortality and morbidity

is beyond our control. Only adequate Health education, proper referral mechanism can help in reducing this.

- Peritonitis and it sequel management involves lots of skill, expensive modalities of monitoring and treatment which has to be utilized judiciously based on risk stratification.

SUMMARY

In the series, 52 cases of perforation peritonitis were studied during the period from November 2012 to November 2014 at PEOPLE'S COLLEGE OF MEDICAL SCIENCES &RESEARCH CENTRE BHOPAL, INDIA.

Duodenal ulcer perforation was the most common cause of perforation in perforative peritonitis, next commonest was appendicular perforation followed by enteric perforation. Colonic perforations are rare. Duodenal ulcer perforation was more common in the 50 years and above age group.

The highest number of patients was seen in the age group 40 years and above, irrespective of the pathological conditions.

Perforation was more cmmon in male than female. 82.7% male & 17.3% female.

Majorities of patient were belonging to rural area with poor socioeconomic status and were unskilled workers.

Smoking and use of non steroidal anti–inflammatory drugs are important risk factors for peptic ulcer perforation.

Most of the patients presented within 24-48 hrs of the clinical symptoms. But delay hospitalization shows high morbidity & mortality.

Presence of gas under the diaphragm confirms the diagnosis, but their absence does not exclude the diagnosis.

Sudden onset of abdomen pain, was constant symptom. Vomiting and nausea were also seen.

Tenderness, rigidity & guarding are important signs. Absence of bowel sounds is one of the early sign of perforation peritonitis.

Resuscitation & preoperative management of the patient is as important as the surgical procedure.

Risk factors for operation of perforation was old age, duration of perforation, size of perforation & presence of preoperative shock.

Laparotomy with closure of the perforation and omental patch closure to be the commonest method of surgical management in perforative peritonitis.

Typhoid and appendicular perforation were more common in 20 - 40 years age group. History of fever is one of the most useful clinical criteria to differentiate typhoid from other perforations. Simple repair of perforation in two layers is the treatment of choice for typhoid perforations.

Post operative morbidity occurred in 19.2 %(major complication only) of cases and mortality of 3.8%.

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PROFORMA

Case No.	IP No.	Unit	Ward
Name:	Age:	Sex	Religion
Occupation:		Address:	
Date of admission:		Date of operation:	
Date of Discharge / Death:			

1. COMPLAINTS:

a. Pain Abdomen

i) Duration ii) Mode of onset

iii) Site of pain iv) Nature

v) Radiation vi) Shifting of pain

vii) Aggravating / Relieving factors

b. Vomiting

i) Duration ii) Frequency

iii) Vomitus: Bilious / Blood / Faecal / otherwise.

c. Fever

i) Duration ii) Degree

iii) Type: Remittent / continuous / Intermittent

iv) Chills / Rigors

d. Distension of abdomen

i) Present / absent:

ii) Duration (if present):

e. Bowel symptoms.

i)Diarrhoea ii)Constipation

iii) Alternating diarrhoea / constipation

iv) Melaena

f. Other complaints (if any):

2. PAST HISTORY:

a) Pain abdomen
b) Haematemesis / Melaena.
c) Previous operation
d) Drug history
e) Other complaints (if any)

3. PERSONAL HISTORY:

a) Diet: Mixed / Vegetarian
b) Appetite: Good / Impaired
c) Bowel / Bladder habits:
d) Habits: Smoker / Alcoholic

4. FAMILY HISTORY:

5. GENERAL PHYSICAL EXAMINATION:

a) Vital signs:

Pulse: BloodPressure:

Temperature: Respiratory rate:

b) Built and Nutrition:

c) State of hydration

d) Anaemia / Jaundice / Pedal edema / Lymphadenopathy

6. EXAMINATION OF THE ABDOMEN

a) Inspection

1. Shape 2. Umbilicus
3. Skin 4. Movement with respiration
5. Visible veins 6. Visible pulsation
7. Hernial orifices 8. External genitalia
9. Distension

b) Palpation:

1. Site of tenderness
2. Guarding / Rigidity
3. Whether liver / spleen palpable

c) Percussion

Liver dullness obliterated

Evidence of fluid in the peritoneal cavity

d) Auscultation

Bowel sounds: Present / Absent

e) Per rectal examination

7. EXAMINATION OF OTHER SYSTEMS

a. Cardiovascular system

b. Respiratory system

c. Nervous system

8. INVESTIGATION

1. Blood investigations:

Hb% TC Blood Group

2. Urine :

Micro Alb Sug

3. Others

Special: X-ray abdomen (Erect / lateral decubitus)

USG abdomen

9. CLINICAL DIAGNOSIS

10. MANAGEMENT

- Operative Management
- Pre operative treatment
- Operative details
- Post operative management

11. FINAL DIAGNOSIS

12. COMPLICATIONS

13. OUTCOME

ETHICAL CONSIDRATIONS

1. Informed written consent will be taken from the patient.
2. Investigations will be done using aseptic precaution.
3. Treatment will be given as per protocol.
4. They will be given from any kind of harm.
5. Protection will be given from any kind of harm.
6. Full confidentiality of data will be maintained.
7. No religious issues involved. All religions and custon will be respected.
8. Study will be conducted under supervision

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PARTICIPANT INFORMED CONSENT FORM (PICF)
(English)

Protocol / Study number :_____
Participant identification number for this trial: _____
Title of project:_____
_____
Name of Principal Investigator: _____Tel.No(s).____
The contents of the information sheet dated that was provided have been read carefully by me / explained in detail to me, in a language that I comprehend, and I have fully understood the contents. I confirm that I have had the opportunity to ask questions.

The nature and purpose of the study and its potential risks / benefits and expected duration of the study, and other relevant details of the study have been explained to me in detail. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal right being affected.

I understand that the information collected about me from my participation in this research and sections of any of my medical notes may be looked at by responsible individuals from P.C.M.S,Bhopal. I give permission for these individuals to have access to my records.

I agree to take part in the above study.

---------- Date:
(Signatures / Left Thumb Impression) Place:

Name of the Participant: _____
Son / Daughter / Spouse of:_____
Complete postal address: _____

This is to certify that the above consent has been obtained in my presence.

Signatures of the Principal Investigator Date:
Place:

1) Witness – 1

Signatures

Name:

Address:

2) Witness – 2

Signatures

Name:

Address:

NB Three copies should be made, for (1) patient, (2) researcher, (2) Institution
(Investigators are advised to prepare the translation in simple understandable Hindi on their own.)

KEY OF MASTER CHART

F	Female
M	Male
RR	Rural
U	Urban
US	Unskilled Worker
SW	Skilled Worker
SS	Semiskilled Worker
D	Dependent
DNA	Detail Not Available
Ep	Epigastric
Rt I	Right Ileac
Rt H	Right Hypochondrium
U	Umbilicus
Drg	Drugs
Smk	Smoking
Alc	Alcohol
Diff	Diffuse
RIF	Right Iliac Fossa
I	Ileum
A	Appendix
D	Duodenum
S	Stomach
LI	Large Intestine
GUP	Gastric Ulcer Perforation
DUP	Duodenal Ulcer Perforation
EP	Enteric Perforation
AP	Appendicular Perforation

IP	Ileal Perforation
PUP	Peptic Ulcer Perforation
LIP	Large Intestine Perforation
OA	Open Appendicectomy
OPC	Omentopexy Closure
SC	Simple Closure
R&A	Resection Anastomosis
RAI	Resection Anastomosis With Ileostomy
ARDS	Adult Respiratory Distress Syndrome
WI	Wound Infection
WD	Wound Dehiscence
LRTI	Lower Respiratory Tract Infection
S	Septicemia

MASTER CHART

SL No.	Name	DOA	Age	Sex	POPULATION DISTRIBUTION	OCCUPATION	IP No.	Symptoms				PAST H/O PAIN	Risk Factor	DEHYDRATION	Signs					X-ray (PNEUMOPERITONEUM)	SITE OF PERFORATION	Clinical diagnosis	CAUSE OF PERFORATION	OPERATIONS	POC	Duration of stay	Outcome
								DURATION OF PRESENTATION	SITE OF PAIN	FEVER	VOMITING				DISTENSION ABDOMEN	SOT	G&R	FRE FLUID	Bs									
1	HALKA SINGH	22/06/2012	65	M	RR	US	1866855	>72 hr	Ep	Yes	No	No	Smk	Yes	Yes	Diff	Yes	Yes	No	Yes	I	IP	EP	RAI	ARDS+ARF+S	32	EXPIRED
2	FIZA	23/06/2012	18	F	U	SW	186853	<12 hr	Rt I	Yes	Yes	No	No	No	No	RIF	No	No	Yes	No	A	AP	AP	OA	No	6	DISCHARGED
3	USHA SHAU	1/7/2012	58	F	RR	US	187472	12-24 hr	Ep	Yes	No	No	Drg	YES	Yes	Diff	Yes	Yes	No	Yes	D	DUP	PUP	SC	LRTI	22	DISCHARGED
4	HUKUM SINGH	10/7/2012	55	M	RR	US	188286	24-48 hr	U	Yes	Yes	Yes	Drg	Yes	Yes	Diff	Yes	Yes	No	Yes	D	DUP	PUP	SC	No	16	DISCHARGED
5	MANESH	11/7/2012	50	M	RR	SS	187830	48-72 hr	Ep	No	Yes	Yes	Smk +Drg	Yes	Yes	Diff	Yes	yes	No	Yes	S	GUP	PUP	OPC	LRTI	20	DISCHARGED
6	SARITA VISHAVAKARMA	18/08/2012	30	F	U	SW	194715	<12 hr	Rt I	Yes	Yes	No	Smk	No	No	RIF	Yes	No	Yes	No	A	AP	AP	OA	No	8	DISCHARGED
7	BABULAL	7/9/2012	40	M	U	SS	1980294	24-48 hr	Rt H	Yes	Yes	Yes	Alc+ Smk	Yes	Yes	Rt H	Yes	Yes	No	Yes	D	DUP	PUP	SC	LRTI	24	DISCHARGED
8	PAPPU	13/09/2012	40	M	RR	US	193671	48-72 hr	Rt I	Yes	No	Yes	Smk	Yes	Yes	RIF	Yes	Yes	No	Yes	I	IP	EP	SC	No	15	DISCHARGED
9	JAWAHAR SINGH	20/9/12	50	M	RR	US	194399	48-72 hr	Ep	No	Yes	Yes	Drg	Yes	Yes	U	Yes	Yes	No	Yes	D	DUP	PUP	OPC	No	10	DISCHARGED
10`	GOVIN PRASAD	1/10/2012	44	M	U	SW	195274	12-24 hr	Ep	No	Yes	No	Drg	No	No	Ep	Yes	Yes	Yes	Yes	S	GUP	PUP	OPC	No	10	DISCHARGED
11	SONU	18/10/12	16	M	U	DP	197868	<12 hr	Rt I	Yes	Yes	No	No	No	No	RIF	No	No	Yes	No	A	AP	AP	OA	No	8	DISCHARGED
12	ISHAN	2/11/2012	11	M	U	DP	199087	6-12 hr	U	Yes	No	No	Drg	No	No	U	No	No	Yes	No	A	AP	AP	OA	No	6	DISCHARGED
13	HEMLATA	4/11/2012	38	F	RR	DNA	199271	48-72 hr	Rt I	Yes	No	No	Drg	Yes	Yes	Diff	Yes	Yes	No	Yes	I	IP	EP	SC	WD	21	DISCHARGED
14	ASHUTOSH	4/11/2012	19	M	RR	DP	199275	6-12 hr	Rt I	Yes	Yes	Yes	No	No	No	RIF	Yes	No	Yes	No	A	AP	AP	OA	No	8	DISCHARGED
15	SOMAT SINGH	1/12/2012	30	M	U	SW	200698	6-12 hr	U	No	Yes	No	Smk	No	No	RIF	Yes	No	Yes	No	A	AP	AP	OA	No	10	DISCHARGED
16	KAJAL	10/1/2013	53	F	RR	US	205588	24-48 hr	Rt H	Yes	No	Yes	Drg	Yes	Yes	U	Yes	Yes	No	Yes	S	GUP	PUP	OPC	No	9	DISCHARGED
17	KAMLESH	19/01/2013	59	M	RR	US	206060	>72 hr	Rt I	Yes	Yes	YES	Smk+ Drg	YES	Yes	Diff	Yes	Yes	No	Yes	I	IP	EP	R&A	WI+ARF+S	34	EXPIRED
18	MONU DHAKAD	24/01/2013	22	M	U	DP	207031	12-24 hr	Rt H	No	Yes	No	Drg	No	Yes	Rt H	Yes	Yes	No	Yes	D	DUP	PUP	SC	No	10	DISCHARGED
19	SAHARA	30/01/2013	44	F	RR	US	207144	48-72 hr	U	Yes	Yes	No	Smk	Yes	Yes	Diff	Yes	Yes	No	Yes	I	IP	EP	SC	WD	18	DISCHARGED
20	RAJENDRA	11/2/2013	39	M	U	SW	207770	12-24 hr	Ep	No	Yes	YES	Smk	No	No	Rt H	Yes	Yes	Yes	Yes	S	GUP	PUP	OPC	No	12	DISCHARGED
21	PRATHVI SINGH	28/02/2012	17	M	U	DP	209423	24-48 hr	Ep	Yes	Yes	YES	No	No	No	U	Yes	No	Yes	Yes	S	GUP	PUP	OPC	No	7	DISCHARGED
22	OMPRAKASH	9/3/2013	55	M	RR	US	210392	48-72 hr	U	No	No	YES	Smk	YES	Yes	Diff	Yes	Yes	No	Yes	LI	LIP	IBP	R&A	WI	21	DISCHARGED
23	KAMLESH	21/03/2013	35	M	U	DNA	210470	12-24 hr	Ep	Yes	Yes	No	Smk	YES	Yes	Diff	Yes	No	No	Yes	S	GUP	PUP	OPC	No	8	DISCHARGED
24	BRAJ MOHAN	11/4/2013	60	M	RR	SS	212286	48-72 hr	Rt H	Yes	No	Yes	Drg	No	Yes	U	Yes	Yes	No	Yes	D	DUP	PUP	SC	LRTI	10	DISCHARGED
25	ANJUNIK	27/04/2013	20	M	U	D	214076	12-24 hr	U	Yes	Yes	YES	Drg	No	No	U	Yes	No	Yes	No	S	GUP	PUP	OPC	No	8	DISCHARGED
26	SHRIRAM KUSHWAHA	2/5/2013	42	M	U	SW	214365	<12 hr	RIF	Yes	Yes	Yes	Smk	No	No	RIF	Yes	No	Yes	No	A	APP	APP	OA	WI	14	DISCHARGED
27	ZAHID ALI	7/5/2013	49	M	RR	DNA	214918	48-72 hr	Rt H	No	Yes	No	Smk	YES	Yes	Rt H	Yes	Yes	No	Yes	D	DUP	PUP	SC	LRTI	22	DISCHARGED
28	BHAIYA LAL	4/6/2013	38	M	RR	SS	1693	<12 hr	RIF	Yes	Yes	No	No	No	No	RIF	No	No	Yes	No	A	APP	APP	OA	No	7	DISCHARGED
29	REKHA TIWARI	17/06/2013	44	F	U	SW	2306	<12 hr	U	No	No	No	No	No	Yes	U	Yes	Yes	Yes	Yes	D	DUP	PUP	OPC	No	10	DISCHARGED
30	SAMUNT SINGH	28/06/2013	22	M	U	D	3499	24-48 hr	RIF	Yes	Yes	No	Smk	NO	Yes	Diff	Yes	No	Yes	No	A	APP	APP	OA	No	8	DISCHARGED
31	BHAGMAN SINGH	18/07/2013	62	M	RR	US	4992	48-72 hr	Ep	No	Yes	Yes	Drg	YES	Yes	Diff	Yes	Yes	No	Yes	S	GUP	PUP	OPC	No	9	DISCHARGED
32	SANJEEV GUPTA	22/07/2013	30	M	RR	D	5442	12-24 hr	Rt H	Yes	Yes	No	Smk	NO	Yes	Diff	Yes	Yes	No	Yes	D	DUP	PUP	OPC	No	10	DISCHARGED
33	SACHIN KUMAR	12/8/2013	33	M	U	D	5521	<12 hr	Ep	No	Yes	No	Drg	No	No	Diff	Yes	Yes	Yes	Yes	D	DUP	PUP	OPC	No	7	DISCHARGED
34	SAVITRI BAI	30/08/2013	40	F	RR	UW	8164	24-48 hr	U	Yes	Yes	Yes	Smk	No	No	U	Yes	Yes	Yes	Yes	D	DUP	PUP	OPC	WD	22	DISCHARGED
35	RAJ NARAYAN	3/9/2013	55	M	RR	US	7599	12-24 hr	RIF	Yes	No	No	Drg	YES	Yes	RIF	Yes	No	No	Yes	I	IP	IP	SC	No	11	DISCHARGED
36	RAM KISHAN	5/9/2013	60	M	RR	US	9019	12-24 hr	Ep	Yes	Yes	No	Drg+Smk	YES	Yes	Ep	Yes	Yes	No	Yes	D	DUP	PUP	SC	No	9	DISCHARGED
37	SURAJ BHAN SINGH	22/09/2013	49	M	RR	US	10440	24-48 hr	Ep	No	No	No	Drg	Yes	No	Ep	Yes	No	No	Yes	S	GUP	PUP	OPC	No	9	DISCHARGED
38	AZHEEM KHAN	28/09/2013	29	M	U	D	10969	<12 hr	RIF	No	Yes	No	Smk	No	No	RIF	Yes	No	Yes	No	A	APP	APP	OA	No	8	DISCHARGED
39	PUNEET	16/10/2013	47	M	RR	US	12337	12-24 hr	Rt H	No	No	No	Drg	No	No	U	Yes	No	No	Yes	D	DUP	PUP	OPC	No	7	DISCHARGED
40	AYUSH RAO	19/10/2013	55	M	RR	SS	12613	24-48 hr	Ep	No	No	Yes	Smk	Yes	No	Diff	Yes	No	No	Yes	D	DUP	PUP	OPC	No	7	DISCHARGED
41	HAFEEZ	6/1/2014	62	M	RR	DNA	18311	24-48 hr	Rt H	Yes	No	No	smk+alc	Yes	Yes	Diff	Yes	Yes	No	Yes	I	IP	IP	SC	No	11	DISCHARGED
42	RAJA	27/03/2014	31	M	RR	US	25343	12-24 hr	RIF	Yes	No	No	Smk	Yes	No	RIF	Yes	No	No	Yes	D	DUP	PUP	OPC	No	7	DISCHARGED
43	SAGAR	23/05/2014	30	M	RR	US	31028	24-48 hr	Rt H	No	No	Yes	Drg	Yes	Yes	Diff	Yes	No	No	Yes	D	DUP	PUP	OPC	No	7	DISCHARGED
44	KAMAL SINGH	12/6/2014	33	M	RR	D	33082	12-24 hr	RIF	No	Yes	Yes	No	No	No	RIF	Yes	No	No	Yes	D	DUP	PUP	OPC	No	7	DISCHARGED
45	KISHANLAL	18/07/2014	35	M	RR	US	36125	48-72 hr	U	No	No	No	Smk	No	No	Diff	Yes	No	No	No	A	APP	APP	OA	No	8	DISCHARGED
46	DEEPAK	31/07/2014	35	M	RR	SS	38145	24-48 hr	Ep	No	No	Yes	Drg	Yes	Yes	Diff	Yes	Yes	No	Yes	D	DUP	PUP	OPC	No	7	DISCHARGED
47	LOKNATH	1/8/2014	29	M	RR	US	33315	24-48 hr	Ep	No	No	No	Drg	Yes	Yes	Diff	Yes	No	No	Yes	D	DUP	PUP	OPC	No	7	DISCHARGED
48	BASANT	5/8/2014	56	M	RR	US	36232	24-48 hr	RIF	Yes	No	No	Smk	Yes	Yes	RIF	Yes	No	No	Yes	S	GUP	PUP	OPC	No	9	DISCHARGED
49	HEERALAL	15/08/2014	40	M	RR	SW	39657	12-24 hr	Ep	No	No	No	Drg	No	No	Ep	Yes	No	No	Yes	S	GUP	PUP	OPC	No	9	DISCHARGED
50	LAXIMI BAI	29/08/2014	28	F	U	SW	41090	24-48 hr	U	Yes	Yes	No	Drg	No	No	RIF	Yes	No	No	Yes	D	DUP	PUP	OPC	No	7	DISCHARGED
51	RADHYA GOVIND	12/9/2014	40	M	RR	US	47652	>72 hr	Ep	No	Yes	No	Drg	Yes	No	Ep	Yes	No	Yes	Yes	D	DUP	PUP	OPC	No	12	DISCHARGED
52	RAM CHARAN	3/10/2014	52	M	RR	US	50045	48-72	Ep	No	Yes	No	Drg	Yes	No	Ep	Yes	No	Yes	Yes	D	DUP	PUP	OPC	No	14	DISCHARGED