

Traumatic Gastrointestinal Perforation- An Overview of Types and Methods of Management

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I. Introduction

The Gastrointestinal perforation is one of the commonest surgical emergencies. It presents, as the acute abdomen, which refers to the clinical situation in which an acute change in the condition of the intra abdominal organ, usually related to inflammation and infection, demands immediate and accurate diagnosis with acute abdominal pain present for less than 6-8 hours.

Gastrointestinal perforation can be divided into upper and lower gastrointestinal perforation. Upper GI perforation include those perforation of the bowel upto the ileocaecal junction, lower perforation include those of the colon and rectum.¹

Duodenal perforation is the commonest of all in GI perforation. Small intestine perforation occurs commonly due to trauma. The large intestinal perforation are commonly caused by trauma.²

The case history and the art of the physical examination are of the great importance in the diagnosis of the GI perforation. Assessment of the patient also culminates in deciding whether the patient should be subjected to the risk of GA required for laparotomy.

Success in the treatment of such cases depends largely upon early investigations, early diagnosis, early intervention and good postoperative care,

Henle's aphorism in acute abdominal emergencies, the difference between the best and worst surgery is definitely less, than between early and late surgery and greatest sacrifice is the sacrifice of time.

The aim of this study is to know age and sex related incidences, variety of modes of the presentation of GI perforation with evaluating effect of delay in the treatment on the postoperative progress.

II. Aims And Objectives Of Study

1. To evaluate all traumatic perforation of the Gastro- intestinal tract.
2. To identify the common sites of the perforation.
3. To study the various factors responsible for this perforation.
4. To study the clinical manifestation and postoperative management of this patients.

III. Review Of Literature

- Anatomy of gastrointestinal tract
- Gastrointestinal tract is divided into
- Oesophagus
- Stomach
- Small intestine
- Large intestine

Oesophagus¹:

Surgical anatomy-

The oesophagus is a muscular tube, approximately 25 cm long, mainly occupying the posterior mediastinum and extending from the upper oesophageal sphincter (the cricopharyngeus muscle) in the neck to the junction with the cardia of the stomach. The musculature of the upper oesophagus, including the upper sphincter, is striated. This is followed by a transitional zone of both striated and smooth muscle with the proportion of the latter progressively increasing so that, in the lower half of the oesophagus, there is only smooth muscle. It is lined through-out with squamous epithelium. The parasympathetic nerve supply is mediated by branches of the vagus nerve that has synaptic connections to the mesenteric (Auerbach's) plexus. Meissner's submucosal plexus is sparse in the oesophagus. The upper sphincter consists of powerful

striated muscle. The lower sphincter is more subtle, and is created by the asymmetrical arrangement of muscle fibres in the distal oesophageal wall just above the oesophagogastric junction.

Stomach²

Gross Anatomy

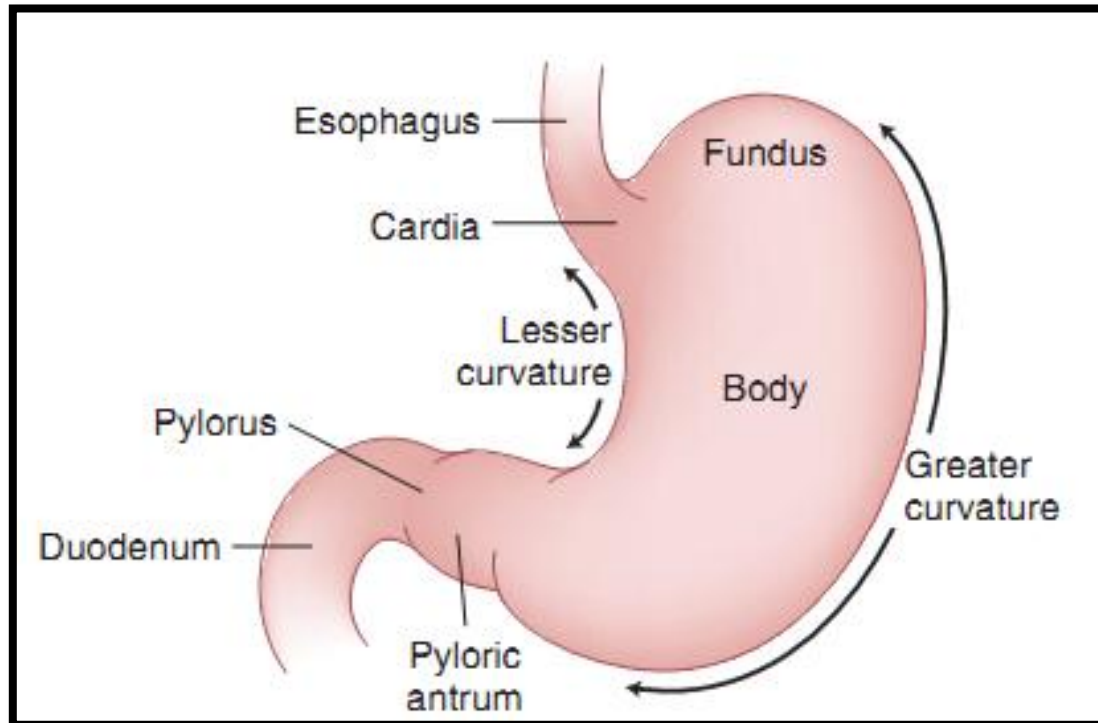


Figure 1- divisions of stomach²

Divisions

The stomach begins as a dilation in the tubular embryonic foregut during the fifth week of gestation. By the seventh week, it descends, rotates, and further dilates with a disproportionate elongation of the greater curvature into its normal anatomic shape and position. Following birth, it is the most proximal abdominal organ of the alimentary tract. The most proximal region of the stomach is called the cardia and attaches to the esophagus. Immediately proximal to the cardia is a physiologically competent lower esophageal sphincter. Distally, the pylorus connects the distal stomach (antrum) to the proximal duodenum. Although the stomach is fixed at the gastroesophageal (GE) junction and pylorus, its large mid portion is mobile. The fundus represents the superior most part of the stomach and is floppy and distensible. The stomach is bounded superiorly by the diaphragm and laterally by the spleen. The body of the stomach represents the largest portion and is also referred to as the corpus. The body also contains most of the parietal cells and is bounded on the right by the relatively straight lesser curvature and on the left by the longer greater curvature. At the angular incisure, the lesser curvature abruptly angles to the right. The body of the stomach ends here and the antrum begins. Another important anatomic angle (angle of His) is the angle formed by the fundus with the left margin of the esophagus (Fig. 1).

Most of the stomach resides within the upper abdomen. The left lateral segment of the liver covers a large portion of the stomach anteriorly. The diaphragm, chest, and abdominal wall bound the remainder of the stomach. Inferiorly, the stomach is attached to the transverse colon, spleen, caudate lobe of the liver, diaphragmatic crura, and retroperitoneal nerves and vessels. Superiorly, the GE junction is found approximately 2 to 3 cm below the diaphragmatic esophageal hiatus in the horizontal plane of the seventh chondrosternal articulation, a plane only slightly cephalad to the plane containing the pylorus. The gastrosplenic ligament attaches the proximal greater curvature to the spleen.

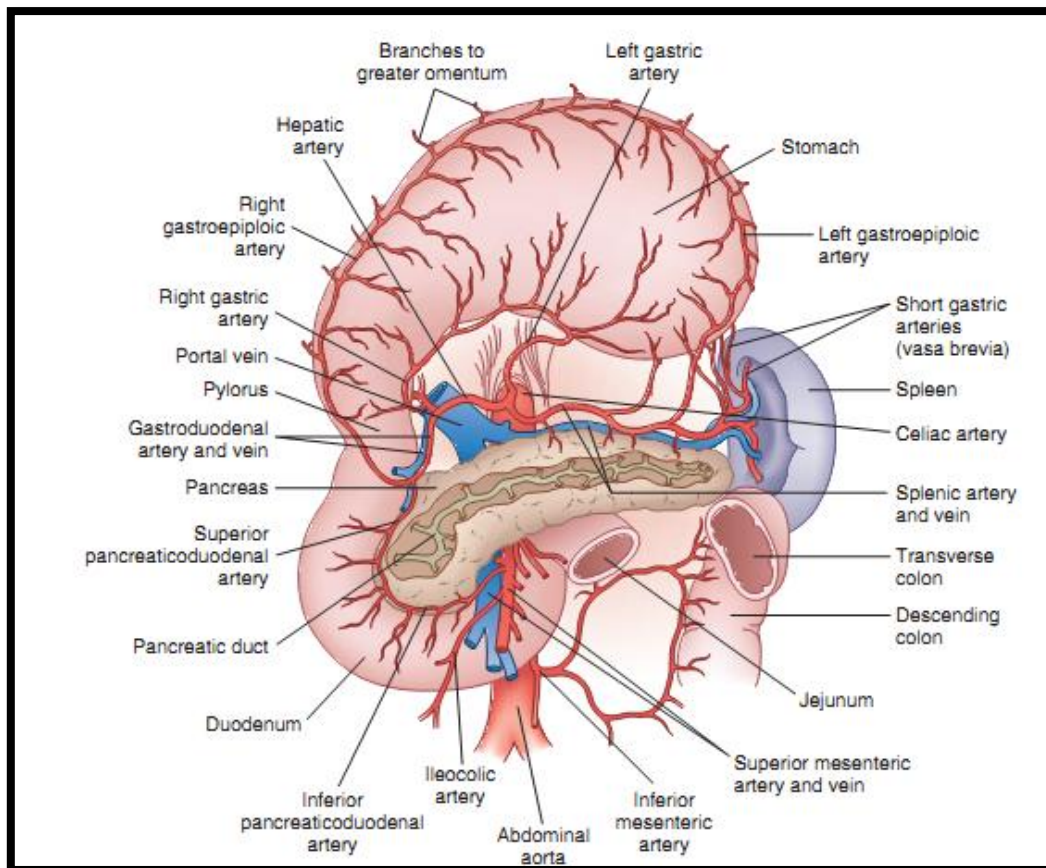


Figure 2- Blood supply to the stomach and duodenum showing anatomic relationships to the spleen and pancreas. The stomach is reflected cephalad²

Blood Supply

The celiac artery provides most of the blood supply to the stomach (Fig. 2). There are four main arteries—the left and right gastric arteries along the lesser curvature and the left and right gastroepiploic artery along the greater curvature. In addition, a substantial quantity of blood may be supplied to the proximal stomach by the inferior phrenic arteries and by the short gastric arteries from the spleen. The largest artery to the stomach is the left gastric artery; it is not uncommon (15% to 20%) for an aberrant left hepatic artery to originate from it. Consequently, proximal ligation of the left gastric artery occasionally results in acute left-sided hepatic ischemia. The right gastric artery arises from the hepatic artery (orgastroduodenal artery). The left gastroepiploic artery originates from the splenic artery, and the right gastroepiploic artery originates from the gastroduodenal artery. The extensive anastomotic connection between these major vessels ensures that in most cases the stomach will survive if three out of four arteries are ligated, provided that the arcades along the greater and lesser curvatures are not disturbed. In general, the veins of the stomach parallel the arteries. The left gastric (coronary) and right gastric veins usually drain into the portal vein. The right gastroepiploic vein drains into the superior mesenteric vein, and the left gastro-epiploic vein drains into the splenic vein.

Lymphatic Drainage

The lymphatic drainage of the stomach parallels the vasculature and drains into four zones of lymph nodes. The superior gastric group drains lymph from the upper lesser curvature into the left gastric and paracardial nodes. The suprapyloric group of nodes drains the antral segment on the lesser curvature of the stomach into the right suprapancreatic nodes. The pancreaticolienal group of nodes drains lymph high on the greater curvature into the left gastroepiploic and splenic nodes. The inferior gastric and subpyloric group of nodes drains lymph along the right gastroepiploic vascular pedicle. All four zones of lymph nodes drain into the coeliac group and into the thoracic duct. Although these lymph nodes drain different areas of the stomach, gastric cancers may metastasize to any of the four nodal groups, regardless of the cancer location. In addition, the extensive submucosal plexus of lymphatics accounts for the fact that there is frequently microscopic evidence of malignant cells several centimeters from gross disease.

Innervation

The extrinsic innervation of the stomach is parasympathetic (via the vagus) and sympathetic (via the coeliac plexus). The vagus nerve originates in the vagal nucleus in the floor of the fourth ventricle and traverses the neck in the carotid sheath to enter the mediastinum, where it divides into several branches around the esophagus. These branches coalesce above the esophageal hiatus to form the left and right vagus nerves. It is not uncommon to find more than two vagal trunks at the distal esophagus. At the GE junction, the left vagus is anterior, and the right vagus is posterior (L ARP)

Small intestine³:

Gross Anatomy

The entire small intestine, which extends from the pylorus to the ileocaecal junction, measures 270 to 290 cm, with duodenal length estimated at approximately 20 cm, jejunal length at 100 to 110 cm, and ileal length at 150 to 160 cm. The jejunum begins at the duodenojejunal angle, which is supported by a peritoneal fold known as the ligament of Treitz. There is no obvious line of demarcation between the jejunum and the ileum; however, the jejunum is commonly considered to make up the proximal two fifths of the small intestine, and the ileum makes up the remaining three fifths. The jejunum has a somewhat larger circumference, is thicker than the ileum, and can be identified at surgery by examining mesenteric vessels (Fig.3). In the jejunum, only one or two arcades send out long, straight vasa recta to the mesenteric border, whereas the blood supply to the ileum may have four or five separate arcades with shorter vasa recta (Fig. 3) The mucosa of the small bowel is characterized by transverse folds (plicae circulares), which are prominent in the distal duodenum and jejunum.

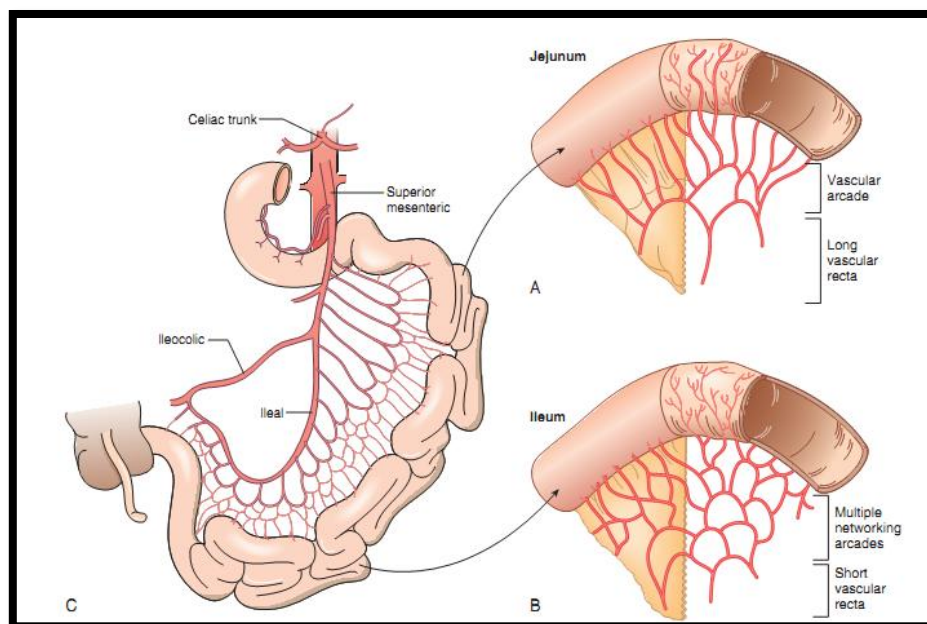


Figure 3- A, The jejunal mesenteric vessels form only one or two arcades with long vasarecta. B, The mesenteric vessels of the ileum form multiple vascular arcades with short vasarecta. C, The superior mesenteric artery, which courses anterior to the third portion of the duodenum, provides blood supply to the jejuno-ileum and distal duodenum. The coeliac artery supplies the proximal duodenum.³

Neurovascular-Lymphatic Supply

The small intestine is served by rich vascular, neural, and lymphatic supplies, all traversing through the mesentery. The base of the mesentery attaches to the posterior abdominal wall to the left of the second lumbar vertebra and passes obliquely to the right and inferiorly to the right sacroiliac joint. The blood supply of the small bowel, except for the proximal duodenum, which is supplied by branches of the coeliac axis, comes entirely from the superior mesenteric artery (Fig. 3). The superior mesenteric artery courses anterior to the uncinat process of the pancreas and the third portion of the duodenum, where it divides to supply the pancreas, distal duodenum, entire small intestine, and ascending and transverse colons. There is an abundant collateral blood supply to the small bowel provided by vascular arcades coursing in the mesentery. Venous drainage of the small bowel parallels the arterial supply, with blood draining into the superior mesenteric vein, which joins the splenic vein behind the neck of the pancreas to

form the portal vein. The innervation of the small bowel is provided by parasympathetic and sympathetic divisions of the autonomic nervous system that, in turn, provide the efferent nerves to the small intestine. Parasympathetic fibers are derived from the vagus; they traverse the coeliac ganglion and effect secretion, motility, and probably all phases of bowel activity. Vagal afferent fibers are present but apparently do not carry pain impulses. The sympathetic fibers come from three sets of splanchnic nerves and have their ganglion cells usually in a plexus around the base of the superior mesenteric artery. Motor impulses affect blood vessel motility and probably gut secretion and motility. Pain from the intestine is mediated through general visceral afferent fibers in the sympathetic system. The lymphatics of the small intestine are noted in major deposits of lymphatic tissue, particularly in the Peyer's patches of the distal small bowel. Lymphatic drainage proceeds from the mucosa through the wall of the bowel to a set of nodes adjacent to the bowel in the mesentery. Drainage continues to a group of regional nodes adjacent to the mesenteric arterial arcades and then to a group at the base of the superior mesenteric vessels. From there, lymph flows into the cisterna chyli and then up the thoracic ducts, ultimately to empty into the venous system at the confluence of the left internal jugular and subclavian veins. The lymphatic drainage of the small intestine constitutes a major route for transport of absorbed lipid into the circulation and similarly plays a major role in immune defense and also in the spread of cells arising from cancers of the gut.

Large intestine⁴:

Anatomy

The large intestine extends from the ileocecal valve to the anus. It is divided anatomically and functionally into the colon, rectum, and anal canal. The wall of the colon and rectum comprise five distinct layers: mucosa, submucosa, inner circular muscle, outer longitudinal muscle, and serosa. In the colon, the outer longitudinal muscle is separated into three teniae coli, which converge proximally at the appendix and distally at the rectum, where the outer longitudinal muscle layer is circumferential. In the distal rectum, the inner smooth muscle layer coalesces to form the internal anal sphincter. The intraperitoneal colon and proximal one-third of the rectum are covered by serosa; the mid and lower rectum lack serosa.

Colon Landmarks

The colon begins at the junction of the terminal ileum and cecum and extends 3 to 5 feet to the rectum. The rectosigmoid junction is found at approximately the level of the sacral promontory and is arbitrarily described as the point at which the three teniae coli coalesce to form the outer longitudinal smooth muscle layer of the rectum. The cecum is the widest diameter portion of the colon (normally 7.5–8.5 cm) and has the thinnest muscular wall. As a result, the cecum is most vulnerable to perforation and least vulnerable to obstruction. The ascending colon is usually fixed to the retroperitoneum. The hepatic flexure marks the transition to the transverse colon. The intraperitoneal transverse colon is relatively mobile, but is tethered by the gastrocolic ligament and colonic mesentery. The greater omentum is attached to the anterior/superior edge of the transverse colon. These attachments explain the characteristic triangular appearance of the transverse colon observed during colonoscopy. The splenic flexure marks the transition from the transverse colon to the descending colon. The attachments between the splenic flexure and the spleen (the lienocolic ligament) can be short and dense, making mobilization of this flexure during colectomy challenging. The descending colon is relatively fixed to the retroperitoneum. The sigmoid colon is the narrowest part of the large intestine and is extremely mobile. Although the sigmoid colon is usually located in the left lower quadrant, redundancy and mobility can result in a portion of the sigmoid colon residing in the right lower quadrant. This mobility explains why volvulus is most common in the sigmoid colon and why diseases affecting the sigmoid colon, such as diverticulitis, may occasionally present as right-sided abdominal pain. The narrow caliber of the sigmoid colon makes this segment of the large intestine the most vulnerable to obstruction.

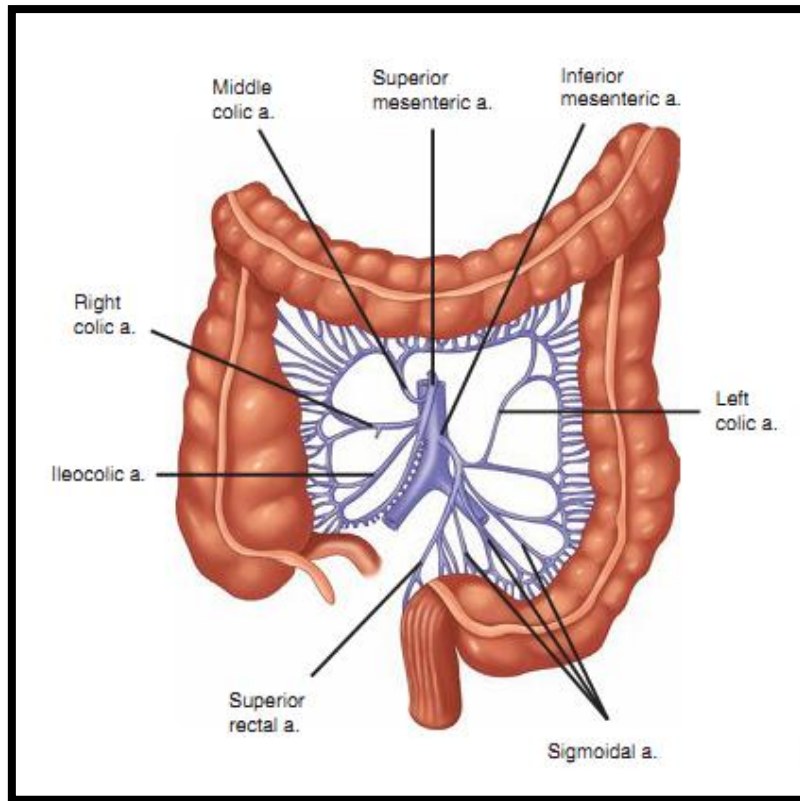


Figure 4- Arterial blood supply to the colon⁴

Colon Vascular Supply

The arterial supply to the colon is highly variable (Fig. 4). In general, the superior mesenteric artery branches into the ileocolic artery (absent in up to 20% of people), which supplies blood flow to the terminal ileum and proximal ascending colon; the right colic artery, which supplies the ascending colon; and the middle colic artery, which supplies the transverse colon. The inferior mesenteric artery branches into the left colic artery, which supplies the descending colon; several sigmoidal branches, which supply the sigmoid colon; and the superior rectal artery, which supplies the proximal rectum. The terminal branches of each artery form anastomoses with the terminal branches of the adjacent artery and communicate via the marginal artery of Drummond. This arcade is complete in only 15% to 20% of people.

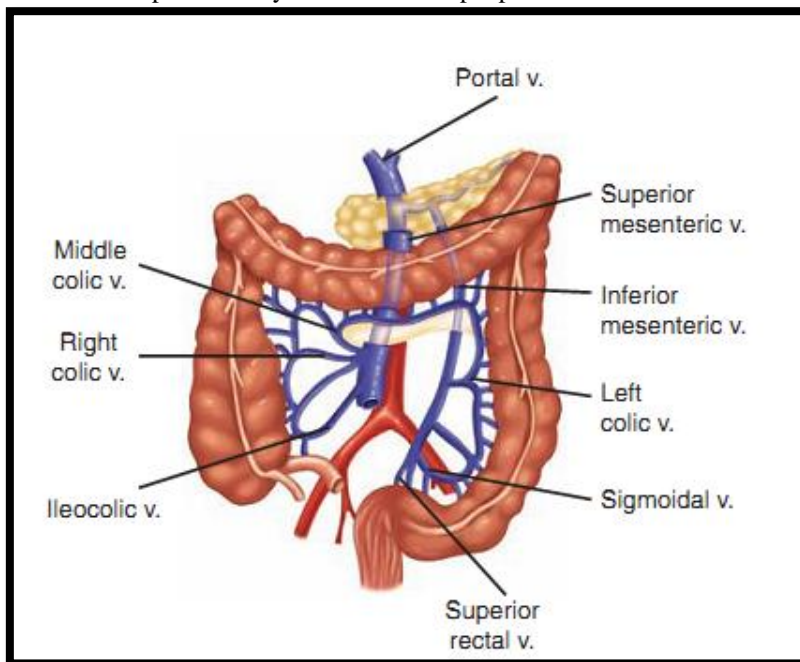


Figure 5-Venous drainage of the colon⁴

Except for the inferior mesenteric vein, the veins of the colon parallel their corresponding arteries and bear the same terminology (Fig. 5). The inferior mesenteric vein ascends in the retroperitoneal plane over the psoas muscle and continues posterior to the pancreas to join the splenic vein. During a colectomy, this vein is often mobilized independently and ligated at the inferior edge of the pancreas.

Colon Lymphatic Drainage

The lymphatic drainage of the colon originates in a network of lymphatics in the muscularis mucosa. Lymphatic vessels and lymph nodes follow theregional arteries. Lymph nodes are found on the bowel wall (epicolic), along the inner margin of the bowel adjacent to the arterial arcades (paracolic), around the named mesenteric vessels (intermediate), and at the origin of the superior and inferior mesenteric arteries (main). The sentinel lymph nodes are the first one to four lymph nodes to drain a specific segment of the colon and are thought to be the first site of metastasis in colon cancer. The utility of sentinel lymph node dissection and analysis in colon cancer remains controversial.

Colon Nerve Supply

The colon is innervated by both sympathetic (inhibitory) and parasympathetic (stimulatory) nerves, which parallel the course of the arteries. Sympathetic nerves arise from T6-T12 and L1-L3. The parasympathetic innervation to the right and transverse colon is from the vagus nerve; the parasympathetic nerves to the left colon arise from sacral nerves S2-S4 to form the nervierigentes. Anorectal Landmarks. The rectum is approximately 12 to 15 cm in length. Three distinct submucosal folds, the valves of Houston, extend into the rectal lumen. Posteriorly, the presecral fascia separates the rectum from the presacral venous plexus and the pelvic nerves. At S4, the rectosacral fascia (Waldeyer's fascia) extends forward and downward and attaches to the fascia propria at the anorectal junction. Anteriorly, Denonvilliers' fascia separates the rectum from the prostate and seminal vesicles in men and from the vagina in women. The lateral ligaments support the lower rectum.

The anatomic anal canal extends from the dentate or pectinate line to the anal verge. The dentate or pectinate line marks the transition point between columnar rectal mucosa and squamous anoderm. The anal transition zone includes mucosa proximal to the dentate line that shares histologic characteristics of columnar, cuboidal, and squamous epithelium. Although the anal transition zone was long thought to extend only 1 to 2 cm proximal to the dentate line, it is known that the proximal extent of this zone is highly variable and can be as far as 15 cm proximal to the dentate line. The dentate line is surrounded by longitudinal mucosal folds, known as the columns of Morgagni, into which the anal crypts empty. These crypts are the source of crypto-glandular abscesses. In contrast to the anatomic anal canal, the surgical anal canal begins at the anorectal junction and terminates at the anal verge. The surgical anal canal measures 2 to 4 cm in length and is generally longer in men than in women. It begins at the anorectal junction and terminates at the anal verge.

In the distal rectum, the inner smooth muscle is thickened and comprises the internal anal sphincter that is surrounded by the subcutaneous, superficial, and deep external sphincter. The deep external anal sphincter is an extension of the puborectalis muscle. The puborectalis, iliococcygeus, and pubococcygeus muscles form the levatorani muscle of the pelvic floor (Fig. 5).

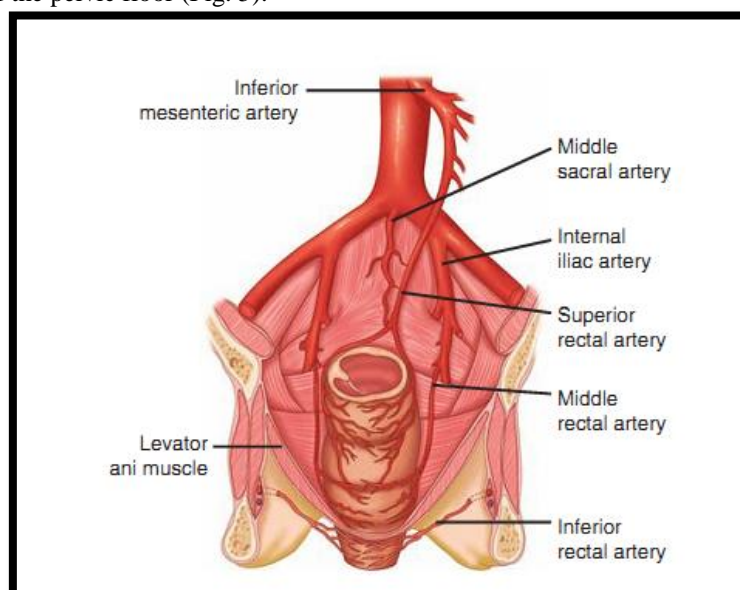


Figure 6- Arterial supply to the rectum and anal canal.⁴

Anorectal Vascular Supply

The superior rectal artery arises from the terminal branch of the inferior mesenteric artery and supplies the upper rectum. The middle rectal artery arises from the internal iliac; the presence and size of these arteries are highly variable. The inferior rectal artery arises from the internal pudendal artery, which is a branch of the internal iliac artery. A rich network of collaterals connects the terminal arterioles of each of these arteries, thus making the rectum relatively resistant to ischemia (Fig.6).

The venous drainage of the rectum parallels the arterial supply. The superior rectal vein drains into the portal system via the inferior mesenteric vein. The middle rectal vein drains into the internal iliac vein. The inferior rectal vein drains into the internal pudendal vein, and subsequently into the internal iliac vein. A submucosal plexus deep to the columns of Morgagni forms the hemorrhoidal plexus and drains into all three veins.

Anorectal Lymphatic Drainage: Lymphatic drainage of the rectum parallels the vascular supply. Lymphatic channels in the upper and middle rectum drain superiorly into the inferior mesenteric lymph nodes. Lymphatic channels in the lower rectum drain both superiorly into the inferior mesenteric lymph nodes and laterally into the internal iliac lymph nodes. The anal canal has a more complex pattern of lymphatic drainage. Proximal to the dentate line, lymph drains into both the inferior mesenteric lymph nodes and the internal iliac lymph nodes. Distal to the dentate line, lymph primarily drains into the inguinal lymph nodes, but can also drain into the inferior mesenteric lymph nodes and internal iliac lymph nodes.

Anorectal Nerve Supply.

Both sympathetic and parasympathetic nerves innervate the anorectum. Sympathetic nerve fibers are derived from L1-L3 and join the preaortic plexus. The preaortic nerve fibers then extend below the aorta to form the hypogastric plexus, which subsequently joins the para-sympathetic fibers to form the pelvic plexus. Parasympathetic nerve fibers are known as the nervus erigens and originate from S2-S4. These fibers join the sympathetic fibers to form the pelvic plexus. Sympathetic and parasympathetic fibers then supply the anorectum and adjacent urogenital organs. The internal anal sphincter is innervated by sympathetic and parasympathetic nerve fibers; both types of fibers inhibit sphincter contraction. The external anal sphincter and puborectalis muscles are innervated by the inferior rectal branch of the internal pudendal nerve. The levator ani receives innervation from both the internal pudendal nerve and direct branches of S3 to S5. Sensory innervation to the anal canal is provided by the inferior rectal branch of the pudendal nerve. While the rectum is relatively insensate, the anal canal below the dentate line is sensate.

Gastric perforations following blunt abdominal trauma are rare, accounting for about 2% of all blunt abdominal injuries. Isolated blunt gastric ruptures are uncommon. They are usually associated with other solid visceral injuries. Injuries to this to match are associated with the highest mortality of all hollow viscus injuries. Severity of the injury, timing of presentation and presentation following the last meal as well as concomitant injuries are important prognostic factors. Imaging modalities may be unreliable in making a diagnosis and thus clinical vigilance is mandatory.

Aboobakar et al. Concluded that severity of the injury, timing of presentation and presentation following the last meal as well as concomitant injuries are important prognostic factors. Prompt diagnosis and timely intervention greatly limits mortality and morbidity associated with blunt gastric injuries. Majority of which are septic in nature.⁵

Tan et al. suggested that Gastro-intestinal tract (GIT) perforation following blunt trauma poses numerous challenges for surgeons worldwide. The authors advocate the usage of computed tomography (CT) scans for further assessment of the abdomen in hemodynamically stable patients. Any abnormal CT scan either warrants surgery or close monitoring. Prompt and early surgery is advised.⁶

Mouton et al Gastro-intestinal tract injuries (GITI) are found in 5% to 17% of laparotomies performed for blunt abdominal trauma. Motor vehicle accidents play an important role. Less than one half of the GITI's caused by blunt abdominal trauma are reported to have enough clinical findings to indicate laparotomy. Laparotomy based on clinical signs alone shows a negative exploration rate of up to 40%. Clinical signs have an important role in combination with radiological examinations and observations. Haemo-dynamically unstable patients undergo immediate laparotomy. In hemodynamically stable patients focused abdominal sonography for trauma (FAST) is recommended as an initial investigation.⁷

Aguirre et al suggested that Traumatic origin injury of the gastrointestinal tract is rare, being less than 1%, however, mortality is 20% in the case of intestinal perforation. The clinical manifestations are varied and depend on the composition of the extravasated contents. This is usually secondary to sudden deceleration and compression by the seatbelt.⁸

Polychronidis et al concluded that Blast trauma can result in injuries to peritoneal organs. In contrast to bombs and other explosive devices, the association with blunt force trauma secondary to the blast effects

of penetrating shotgun wounds is very rare. Penetrating extraperitoneal shotgun wounds, and even tangential gunshot or shotgun injuries of the abdominal or chest wall, can result in damage to abdominal organs despite an intact peritoneum and diaphragm.^{1,2} Delayed or missed diagnosis of blast intestinal injury, because of the lack of clinical findings, are frequently associated with high morbidity and mortality rates.^{1,3} Were poor there in a rare case of a penetrating but extraperitoneal shotgun wound resulting in perforation of the large bowel, presumably from primary blast effect.⁹

Etiology

Traumatic Gastro-intestinal perforation may have the etiological factors as follows.

Traumatic perforation:

Blunt injuries-

Solid organs are more likely to be compressed from the blunt trauma than are the hollow viscera. Thus the kidneys, liver, spleen and pancreas are especially vulnerable, while intestines and stomach are less likely to do so. Among which more commonly affected are small intestine especially upper jejunum.¹⁰

Penetrating injuries-

These injuries involve the viscera approximately in proportion to the volume of the space each of them occupies. Thus, there is great increase in frequency of wounds of the small intestine and then large one.¹⁰

Pathological perforation¹¹:

Stomach and duodenum:

Peptic ulcer: perforation may occur in acute or chronic cases. It is the commonest cause of the G-I perforation. More common in the duodenum than in stomach.

Carcinoma of the stomach: perforation may be the first manifestation of carcinoma. Biopsy from edge of the perforation confirms the diagnosis.

Small intestines:

1. Typhoid ulcers: it is the commonest cause of the perforation in the small intestines. Ulcer is usually single and along the parallel to the long axis of the gut. It is common in lower 1/3rd of the ileum.
2. Tuberculous ulcers: The multiple ulcers in the terminal ileum characterize it. The long axis of the ulcer lie transversely.
3. Meckel's diverticulum: may perforate due to lodging of the coarse food residue or sharp foreign body or acid secreting mucosa.
4. Ascaris lumbricoides: it may cause primary or secondary perforation.
5. Inflammatory bowel diseases: Crohn's disease or Ulcerative colitis. more commonly in Crohn's disease.
6. Miscellaneous- Amoebic dysentery, Bacillary dysentery, Scleroderma, Reduplicated ileal loop syndrome.

Large intestine:

1. Carcinoma: may present as perforation.
2. Amoebic ulcer: the most common sites are caecum and rectosigmoid junction. If perforation occurs then it is commonly in the confined space causing abscess.
3. Diverticulitis: Diverticulitis may perforate if proper antibiotics and NSAID is not advised.

Pathophysiology

Perforation requires full-thickness injury of the bowel wall; however, partial-thickness bowel injury (eg, electrocautery, blunt trauma) can progress over time to become a full-thickness injury or perforation, subsequently releasing gastrointestinal contents. Full-thickness injury and subsequent perforation of the gastrointestinal tract can be due to a variety of etiologies, commonly instrumentation (particularly with cautery) or surgery, blunt or penetrating injury.

Traumatic intestinal perforation

Small intestine may be contused or perforated or transected due to traumas to anterior abdominal wall. It may or may not be associated with mesenteric injury or mesentery may get damaged alone. The viability of the intestine depends on the extent of mesenteric injury. Injury abdomen can be penetrating or blunt.

Blunt Abdominal Injury-

The small intestine may be confused or by direct violence applied to abdominal wall. A small intestine fixed by adhesions are more prone for this injury. The upper jejunum and lower ileum suffer most commonly because of their fixity.

Penetrating abdominal injury-

External injury can be anywhere on abdominal wall. If the perforated intestine is inspected immediately after injury, there is tight spasm and the mucosa is everted through perforation. Later the injured bowel dilated and due to local oedema edges become swollen and because of dilatation and oedema edge tear. It leads to increase in gap. It is followed by suppurative peritonitis.

Small bowel may accidentally be injured during few surgical procedures like, laparoscopy, vaginal surgery, uterine curettage etc.

Perforation peritonitis

Intestines perforate at the site of the weakness either by further invasion of the disease, raised intraluminal pressure.

A normal intestinal wall can tolerate intraluminal pressure up to 150-200cms of the water.

The case of the peritonitis of whatever cause, initiates a sequence of the responses involving the peritoneal membrane, bowel and the body fluid compartments, which then produce secondary responses by endocrine, respiratory, renal and metabolic systems. In some case of the peritonitis the most notable is the acute suppurative peritonitis due to the presence of the infection.

Primary responses of the peritonitis:

Membrane inflammation: peritoneum acts as a two way street for the transudation and toxin to give rise to systemic responses. Peritoneum reacts by the transudation of the interstitial fluid in the peritoneal cavity across the inflamed peritoneum. It is shortly followed by the exudation of the protein rich fluid containing large amount of plasma proteins and fibrin, which help in agglutination of the loops of the bowel and other viscera. This response helps to confine the source of peritoneal contamination and dilution of the irritants. Dilution of the irritant might give temporary relief of the symptoms, which gives rise to the following stage in the development of the perforation peritonitis- Stage of the acute perforation, stage of the reaction and stage of the definitive peritonitis.

Bowel response: initially there is hypermotility, but after a short time motility is depressed and nearly complete adynamic ileum soon develops. Bowel becomes distended and air-fluid accumulates into it.

Hypovolemia: it is caused by the outpouring of plasma like fluid from extracellular interstitial and intravascular compartment into the peritoneal space as an exudate. The atonic dilated gastrointestinal tract also accumulates fluid derived from extracellular compartment.

- Secondary responses to peritonitis:
- Endocrine response
- Cardiac response
- Respiratory response
- Renal response
- Metabolic response
- Suppurative peritonitis:

A group of specific response due to the presence of over-whelming number of bacteria is superimposed on general responses. The magnitude of the specific response to generalized spreading peritonitis is in part determined by:

The virulence of the bacteria

Extent of contamination- which again depends upon:

- Size of the perforation
- Site of the perforation
- Number of perforation
- Duration of the contamination
- Partial or complete flooding of the peritoneum
- General condition of the patient
- The capacity of resistance of the patient to the infection.

In peritonitis during first 6 hours, the peritonitis is noninfective. Where the perforation is existed for more than 8-12 hours, the peritoneal fluid will be infective in character. There is an immediate reaction to the sudden and gross soiling of the peritoneum after perforation by hyperemia, oedema, transudation and exudation. Fibrin deposition and pus formation occurs to a varying degree according to the chemical nature of the fluid, type, virulence and number of organisms.

Perforation leak continuously and some are sealed by fibrin and omental adhesions. Few are sealed by natural method alone. It is possible that a limited amount of irritant and infected material is diluted and neutralized by reactive peritoneal exudates rich in polymorphs and antibodies. When large collection are found, the intestines are found submerged in this fluid and are unable to form defensive adhesions.^{10,11}

Clinical manifestation

- Patient present with the following complaints
- Pain in abdomen
- Vomiting
- Fever
- Distention of abdomen
- Constipation
- Malena

Post operative management of the patients

The postoperative management is equally important as it plays a vital role in final outcome of this abdominal surgery. The postoperative led to a considerable reduction in mortality and complication may become much less frequent.

1. Position in the bed: when returned to bed after operation the patient should be placed in the semi prone position, in order to avoid the aspiration of vomitus into the respiratory tract. The foot end of bed is usually raised in the first 12 hours till the pulse and blood pressure are stabilized. Old people are allowed maximum ventilation in order to minimize the pulmonary complication.
2. Fluid requirement: Patient requires intravenous fluid therapy for the first two or three days. Ringer lactate, normal saline and dextrose were administered by intravenous infusion. About 2000 to 2500ml may be required in the course of 24 hours. However for the calculating the fluid requirement for the day one must take into account, the quantity of nasogastric aspiration, urine output etc. The only thing that should be kept in mind is that the patient should not be overhydrated.
3. Nasogastric aspiration: the nasogastric aspiration should be started soon after the patient returns to the ward and it should be continued at a regular interval of one hour. The colour and the nature of fluid should be recorded.
4. Antibiotics: Initially a combination of ceftriaxone+salbactam 1.5gmi.v. should be given 12hourly a day, amikacin 500mg i.v. should be given 12hourly a day and metronidazole 500mg i.v. should be given 8hourly a day. If the peritoneal fluid sent for culture and sensitivity, further antibiotics should be given depending upon the culture and sensitivity report.
5. Blood transfusion: if patient is anemic a blood transfusion should be given. If blood is not available a plasma expander such as dextran can be infused.
6. Bowel action: after 48 hours to 72 hours a small glycerine suppository may be given to ensure bowel movement.
7. Diet: oral fluids are started when the aspiration reduces in quantity, the bowel sounds are well heard and the patient has passed flatus. If there is no vomiting the diet is changed over to solid or normal diet. Proteins and vitamins should be given for quick recovery.
8. Exercises and period of bed rest: the patient is mobilised after 2nd or 3rd postoperative day. Tell patient to take deep breath to help the secretion in the lungs to be expectorated. Complete body activity should be carried out after the sutures removal on 8th to 10th day, the patient can discharge from hospital.¹²

IV. Materials And Methods

Type of study- prospective study

Sample Size- 50 cases.

Duration of study-

Study place-

Institutional Ethics Committee approval obtained on 07-04-2016. Study was started after obtaining the approval letter from Ethics Committee.

Source of Data- data will be obtained from the patient getting admitted to the M.G.M Medical College, Navi Mumbai. All patients were explained about the study. Written Informed Valid consent was obtained from each patient before conducting the study. With history of trauma along with pain in abdomen, vomiting, fever, distention of abdomen, tenderness, in whom trauma to gastro-intestinal tract is sustained.

Inclusion criteria

All traumatic Gastro- Intestinal tract injuries.

Exculsion criteria

All the patient <14 years.

Methodology collection of Data

Study sample was taken from the patient getting admitted to the M.G.M Medical college, Navi Mumbai with the history of trauma in whom injury to gastro-intestinal tract is suspected. These patients with sudden onset of pain inabdominal pain, vomiting, fever, were examined. They found to have examined clinically for pulse, blood pressure, abdominal distension, tenderness, guarding, rigidity. The patients having trauma to abdomen and operated consecutively with findings of gastrointestinal perforations were also included for the evaluation.

Investigation required

- Blood investigation: Complete heamogram
- Random blood sugar (RBS)
- liver function test (LFT)
- Renal function test (RFT),
- HbsAg, HIV, HCV.

Radiological investigation:

- X-ray chest PA view
 - X-ray abdomen standing
 - Ultrasonography abdomen and pelvis.
 - Image-Guided minimally invasive procedure machine used was Philips HD 1.5 & 11, probe curvilinear with 5 MHz frequency.
- Patient positioned on the examining table in supine position.

Adequate preoperative preparation and diagnosis was established in each case and subjected for the laparotomy. The nature and cause of the perforation were noted at laparotomy and was followed up for the postoperative complication.

These patients underwent laparotomy and result were compared with postoperative diagnosis. The etiology, age and sex, pulse rate and blood pressure, severe abdominal tenderness and distension of abdomen were analysed.

V. Observation And Results

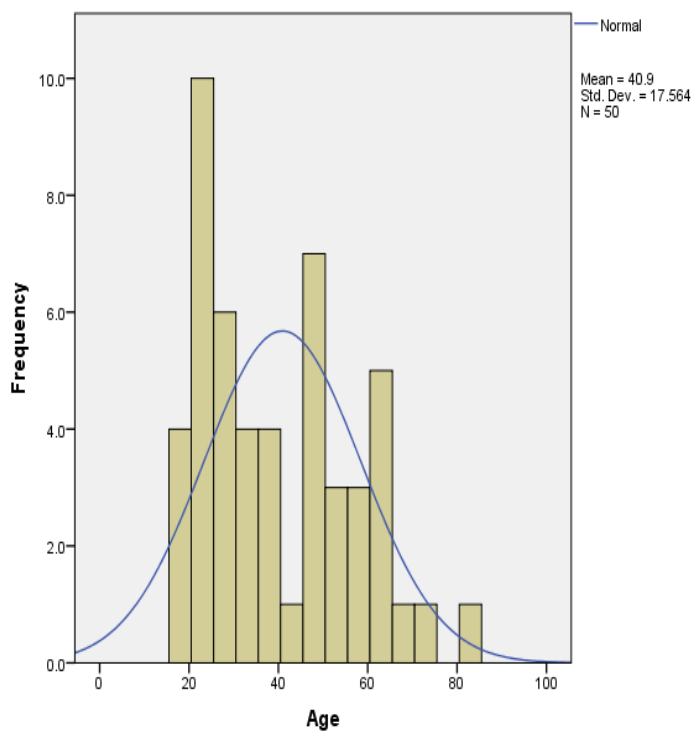
1.Age related incidence:

Table 1- Age related incidence Descriptive Statistics

Age Group (in years)	No. Of cases/ 50	In percentage
14-20	4	8%
21-30	16	32%
31-40	8	16%
41-50	8	16%
51-60	6	12%
61-70	6	12%
>70	2	4%

Interpretation:

It is clearly seen from the above table that, in our study of 50 patients the maximum number of cases were from the age group 21-30 years and next in incidence is the 31-40 years and 41-50 years of age group respectively.



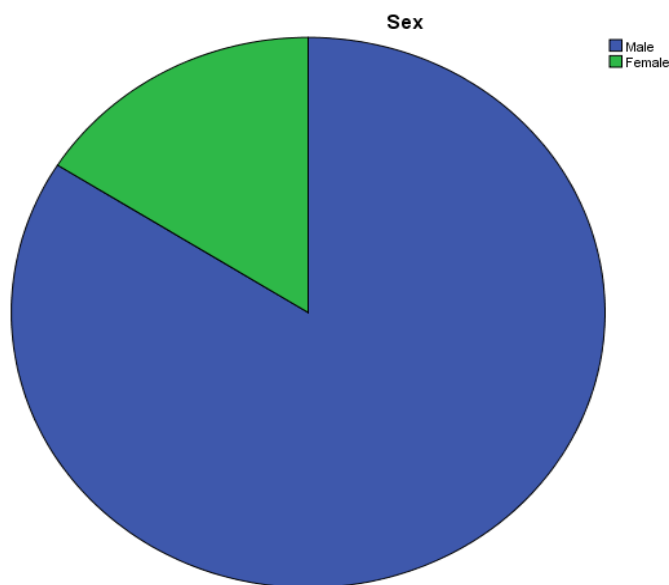
Graph 1- Age related incidence

2. Sex related incidence:

Table 2- sex related incidence Sex

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Male	42	84.0	84.0	84.0
	Female	8	16.0	16.0	100.0
	Total	50	100.0	100.0	

In our study male constituted 42 cases and that of female only 8 with sex ratio, 8.4:1.6 (male:female).



Graph 2- sex related incidence

3. Incidence of the symptoms:

Table 3- pain in abdomen related incidence Presenting Complaints Pain in abdomen

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Present	50	100.0	100.0	100.0

Interpretation:

In all 50 cases acute pain in abdomen was the main presenting complaints with incidence of 100%.

Table 4- vomiting related incidence Presenting Complaints Vomiting

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Present	35	70.0	70.0	70.0
	Absent	15	30.0	30.0	30.0
	Total	50	100.0	100.0	

Interpretation:

In all 50 cases vomiting as the second most common symptoms with incidence rate of 70%.

Table 5- fever related incidence Presenting Complaints Fever

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	45	90.0	90.0	90.0
	Present	5	10.0	10.0	100.0
	Total	50	100.0	100.0	

Interpretation:

In all 50 cases Fever as the third most common symptoms with incidence rate of 10%.

Table 6- Distension of abdomen related incidence

Presenting Complaints Distension of abdomen

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	50	100.0	100.0	100.0

Interpretation:

In all 50 cases Distension of abdomen incidence rate of 0%.

Table 7- Constipation related incidence Presenting Complaints Constipation

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	50	100.0	100.0	100.0

Interpretation:

In all 50 cases Constipation incidence rate of 0%.

Table 8- Malena related incidence Presenting Complaints Malena

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	50	100.0	100.0	100.0

Interpretation:

In all 50 cases Malena incidence rate of 0%.

Table 9- Past history related incidence

Presenting Complaints Relevant past history

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	no Significant abnormalty	43	86.0	86.0	86.0
	known case of diabetes	4	8.0	8.0	94.0
	known case of diabetes and hypertension	3	6.0	6.0	100.0
	Total	50	100.0	100.0	

Interpretation:

In 50 cases of study known case of Diabetes 8% and known case of Diabetes and hypertension 6%.

4.Incidence radiological ultrasonography finding:

Table 10- Ultrasonography related finding Radiological Investigation Ultrasonography

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Haemoperitonium	29	58.0	58.0	58.0
	Minimal Free Fluid	1	2.0	2.0	60.0
	Gross free fluid	10	20.0	20.0	80.0
	Moderate free fluid	7	14.0	14.0	94.0
	Mild free fluid	3	6.0	6.0	100.0
Total		50	100.0	100.0	

Interpretation:

In 50 patients case study the ultrasonography commonest finding is haemoperitonium (58%). Followed by gross free fluid (20%).

5.Incidence of the site of the perforation:

Table 11- site of perforation related finding Site of the perforation

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Small Intestine Perforation	19	38.0	38.0	38.0
	Gastric Perforation	8	16.0	16.0	54.0
	Abdominal perforation	14	28.0	28.0	82.0
	Intestinal perforation	8	16.0	16.0	98.0
	Duodenal perforation	1	2.0	2.0	100.0
Total		50	100.0	100.0	

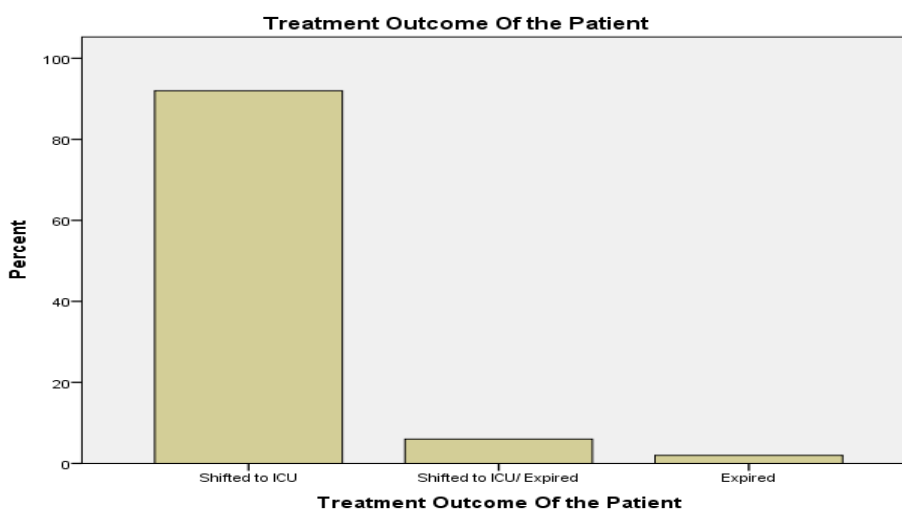
Interpretation:

In 50 patients cases study the commonest site of the perforation is small intestine perforation (38%). Followed by gastric (16%) .

6. Incidence of outcome of the patient

Table 12- postoperative outcome of patient related incidence Treatment Outcome Of the Patient

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Shifted to ICU	46	92.0	92.0	92.0
	Shifted to ICU/ Expired	3	6.0	6.0	98.0
	Expired	1	2.0	2.0	100.0
	Total	50	100.0	100.0	



Graph 3- postoperative outcome of patient related graph

Interpretation: in 50 patients case study patients shifted to ICU than ward than discharge from hospital (92%). Followed by Expired in ICU (6%).

CLINICAL PHOTOGRAPHS

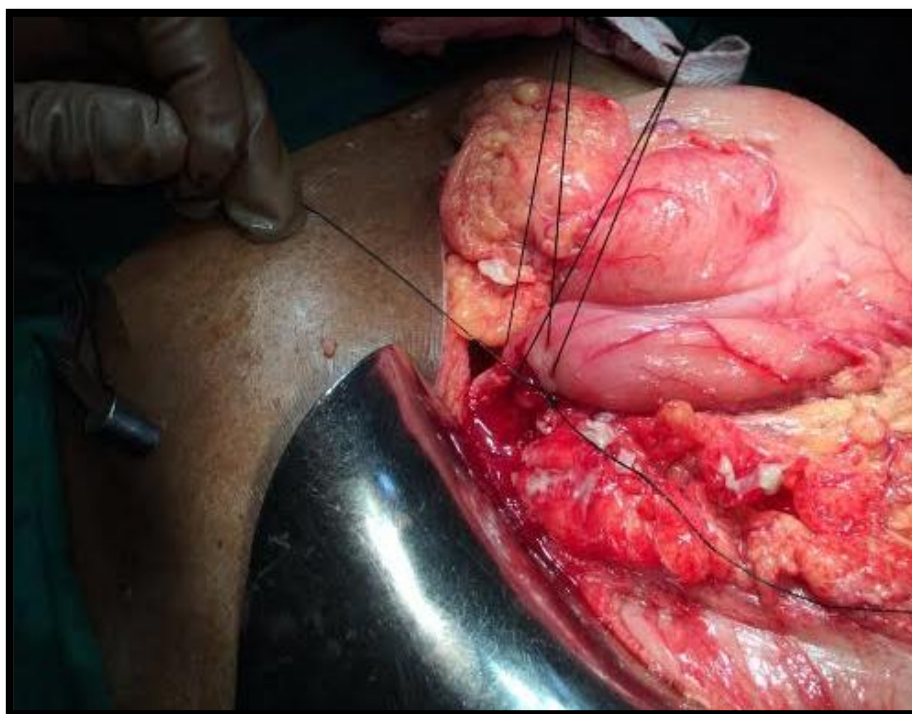


Figure 7- Small intestine perforation (Ileum perforation)



Figure 8- Small intestine perforation (Jejunum perforation)

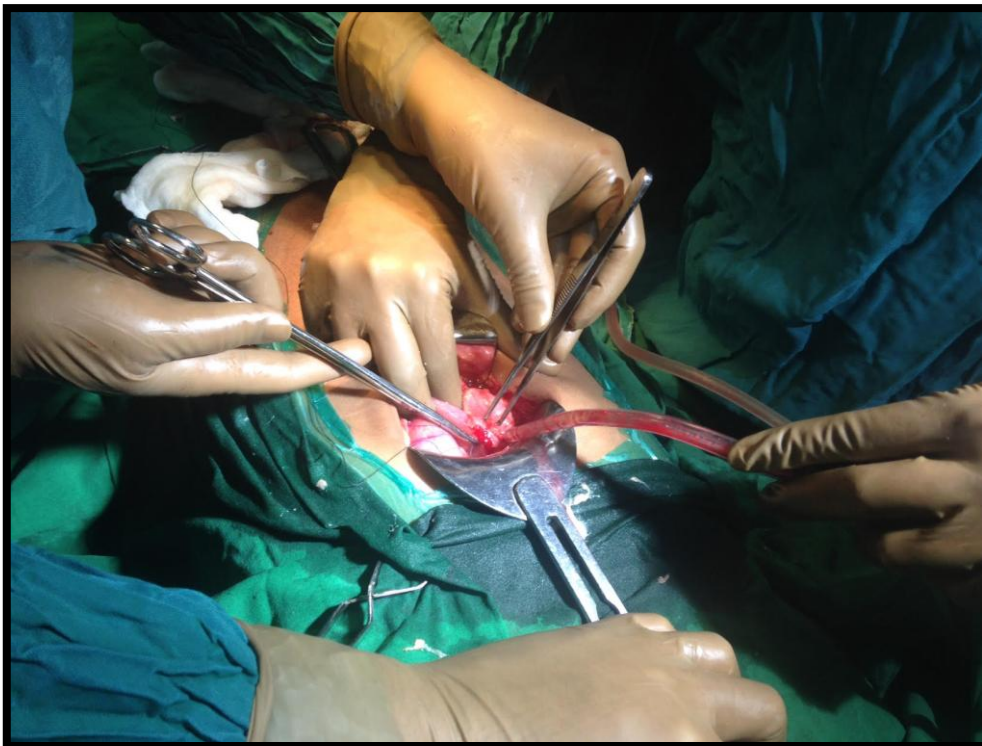


Figure 9- Duodenal perforation

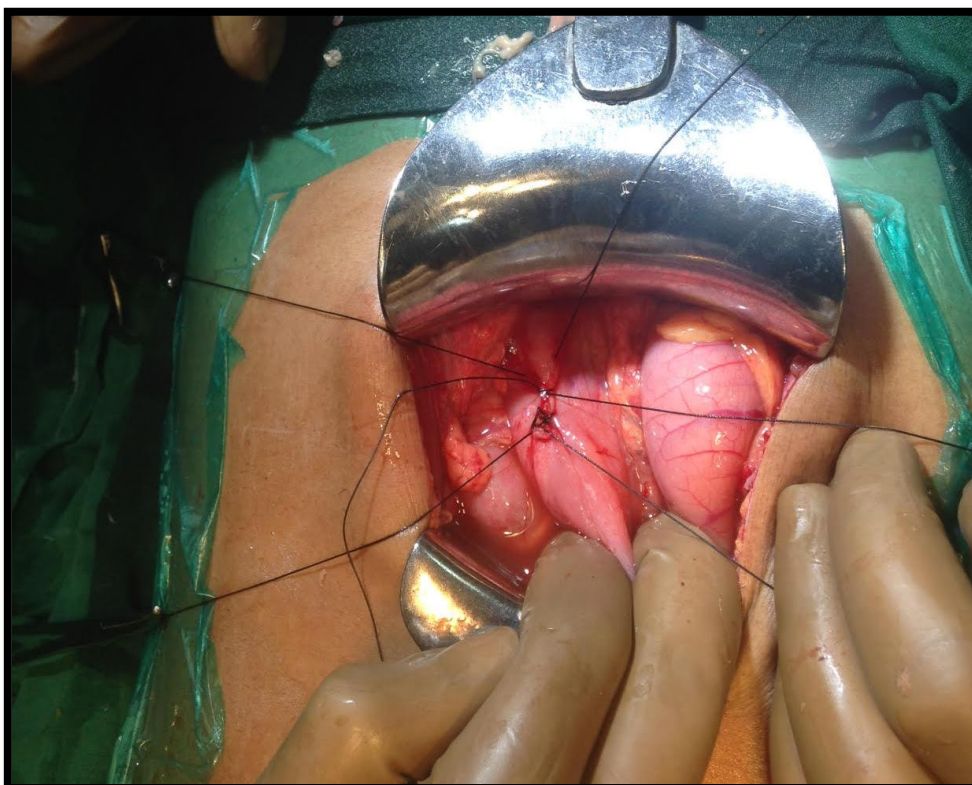


Figure 10- Gastric perforation

VI. Discussion

The result obtained in the present study compared with previously conducted similar studies.

1. Etiology related incidence.

Amuthan J, Vijay A., Pradeep C., Anandan H., et al common cause of blunt trauma to abdomen was (road traffic accidents) RTA (68%) and the second common cause was fall from height(22%). Other causes were hit by blunt objects (6%) and assaults(4%). In our study of 50 patients common cause of blunt trauma to abdomen was road traffic accidents.¹⁹

Table 13- Etiology related incidence

Etiology	Amuthan J ¹⁹ et all	Present study 50 cases
Road traffic accidents	68%	100%
Fall from height	22%	0%
Hit by blunt object	6%	0%
Assault	4%	0%

2. Age and sex related incidence.

Dauterve A., Flancbaum L., Cox E., et al study of the 60 patients sustaining major intestinal injuries, the average age was 33.2 years. The male-to-female ratio was 7.5:1. In our study of the 50 patients sustaining major intestinal injuries, the average age was 40.9. the male-to-female ratio was 8.4:1.6.²⁶

Table 14- age related incidence

Age in years	No cases	Percentage
14-30	20	40%
31-50	16	34%
>50	14	28%

Table 15- sex related incidence

		Sex			
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Male	42	84.0	84.0	84.0
	Female	8	16.0	16.0	100.0
	Total	50	100.0	100.0	

3. Incidence pulse rate and blood pressure relation.

Hughes T., Elton C., Hitos K., Perez J., McDougall P., et al study Thirty six patients (48.6%) had a heart rate <100bpm, five (13.9%) of whom had a systolic BP equal to or <90mmHg. 38 (51.4%) patients had a heart rate of 100bpm or more, five (13.2%) of whom had a systolic BP equal to or <90mmHg. In our study of the 50 patients (14%) had a heart rate <100bpm, (16%) of whom had a systolic BP equal to or <90mmHg. (86%) patients had a heart rate equal to or <90mmHg (84%) had a heart rate equal to or >90mmHg.³³

Table 16- General examination finding related incidence

Examination Vitals Descriptive Statistics

	N	Range	Minimum	Maximum	Mean	Std. Deviation	Skewness		Kurtosis	
	Statistic	Statistic	Statistic	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic	Std. Error
On Examination Vitals Pulse Rate beats/min	50	74	56	130	82.68	15.442	.723	.337	.619	.662
On Examination Vitals Blood Pressure	50	16	1	17	8.38	4.379	.000	.337	-1.031	.662
On Examination Vitals Respiratory Rate cycles/min	50	14	14	28	19.40	2.259	.717	.337	3.374	.662
On Examination Vitals Temperature F	50	2	98	100	98.44	.416	3.064	.337	8.129	.662
On Examination Vitals Saturation	50	3	1	4	1.72	.991	1.126	.337	.035	.662
On Examination Vitals RBS mg/dl	50	172	96	268	124.44	28.597	2.956	.337	12.396	.662
Valid N (listwise)	50									

4. Incidence severe abdominal tenderness and distension of abdomen relation.

54 (73%) patients had moderate to severe abdominal tenderness, diffuse peritoneum being noted in 29 cases. Five (6.7%) patients had mild or absent tenderness. 11 (14.9%) patients had marked abdominal distension, nine of whom had associated injuries to the pelvis or chest that could have contributed to the

distension. Mild or moderate distension was present in 19 (25.7%). Abdominal distension was absent in 44 (59.4%) cases. In our study of 50 patients (100%) severe abdominal tenderness . Abdominal distension absent in (100%).³³

Table 17- Distension of abdomen related incidence Presenting Complaints Distension of abdomen

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	50	100.0	100.0	100.0

VII. Conclusion

From our study of 50 cases of gastrointestinal perforation following can be concluded.

1. The commonest cause of GI perforation is small intestine perforation. The second commonest cause is stomach perforation.
2. The peak age incidence of perforation is early adulthood years of age group.
3. Males are more vulnerable for the perforation with male: female ratio of 8.2:1.6.
4. Small intestine is the commonest site of perforation while stomach and duodenum are second and third in the rank respectively.
5. Small intestine perforation is more common than in stomach with small intestine: gastric ratio of 6:4.
6. All patients (100%) present with symptom of acute abdominal pain and vomiting (70%) as the second common complaint and fever (10%) as the third common complaint.
7. Patients presents with per abdominal tenderness in (100%).
8. Use of X-rays erect abdomen helps in clinching the diagnosis.
9. Many of the patients presented within 24 hours after the trauma.

VIII. Summary

Traumatic injuries of gastrointestinal tract due to blunt and penetrating injury is on rise due to excessive use of motor vehicles more common in early adulthood and male. It poses a therapeutic and diagnostic dilemma for the attending surgeon due to wide range of clinical manifestations like vitals sudden onset of abdomen pain, vomiting, fever ranging from no early physical findings to progression to shock. Hence, the trauma surgeon should rely on his physical findings like distension of abdomen, abdomen tenderness, guarding, rigidity, in association with the use of modalities such as X-ray abdomen and USG abdomen and pelvis. Hollow viscus perforations are relatively easy to pick on X-ray. However, solid organ injuries are sometimes difficult to diagnose due to restricted use of USG Abdomen and pelvis but it is easy, quick and cheap to help to give diagnosis in emergency condition.

In our study of 50 cases of traumatic injuries of G-I tract during operative procedure we find common site involve of G-I perforation and during postoperative management of patient with proper care, proper instruction and proper intravenous fluid with antacid, analgesic, sensitivity to patient antibiotic should give good outcome.

In our study we find we compared the incidence related age, sex, abdominal finding, investigation finding, common site of perforation and postoperative management of this patients. we conclude that in hemodynamically stable patients with solid organ injury, conservative management can be tried and non-operative management is associated with less complication and morbidity.

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