"A COMPARATIVE STUDY IN SURGICAL MANAGEMENT OF ILEAL PERFORATION BY PRIMARY CLOSURE V/S PRIMARY CLOSURE WITH OMENTAL PATCH

By

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DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA IN PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE DEGREE OF

MASTER OF SURGERY

IN

GENERAL SURGERY

Under the guidance of

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Dr. SRIKANTH TALLURI

LIST OF ABBREVATIONS

AIDS	Acquired Immunodeficiency Syndrome
Alb	albumin
ARDS	Acute Respiratory Distress Syndrome
BP	blood pressure
C.D	Crohn's disease
c/s	culture/Sensitivity
CSF	cerebrospinal fluid
C.T	Computerised Tomography
dL	decilitre
DIC	Disseminated Intravascular Coagulation
DNA	deoxyribonucleic acid
DOA	date of admission
DOD	date of discharge
DOS	date of surgery
E. coli	Escherichia coli
ELISA	enzyme-linked immunosorbent assay
gm	grams
Hb	haemoglobin
Hg	mercury
Kg	kilograms
L	litres
mg	milligrams
min	minutes
Misc	miscellaneous
ml	millilitre
mm	millimetre
mmol	millimole
MODS	Multi Organ Dysfunction Syndrome
perf	perforation
PR	pulse rate
RR	respiratory rate

ABSTRACT

BACKGROUND

Perforation of small bowel especially of ileum is an abdominal emergency faced by the general surgeon. Ileal perforation has an abrupt onset and a rapid downhill course with a high mortality if not treated. Early surgery & adequate resuscitation are the key to successful management of patients with ileal perforation. . The current surgical options include primary double layered closure, primary double layered closure with omental patch segmental resection and end to end anastomosis and primary ileostomy. Though surgery is accepted as the definitive treatment, the choice of exact surgical procedure remains controversial.

AIMS & OBJECTIVES

1. Evaluate outcomes of ileal perforation management with primary closure.
2. Evaluate outcomes of ileal perforation management by primary closure with omental patch
3 .To evaluate which has better outcomes between primary closure and primary closure with omental patch in management of ileal perforation.

METHODS

Study was conducted in R.L Jalappa hospital & research centre from February 2012- June 2013. A total 40 patients randomized to two groups. Multiple ileal perforation cases were excluded. All odd numbered patients were included in group I and underwent primary closure .All even numbered patients were included in group II and underwent primary closure with omental patch. Post operative recovery results analysed and tabulated

RESULTS

In our study commonest cause of ileal perforation was non specific perforation. Perforation commonly occurred in second and third decade of life with mean of age of incidence 34.27 yrs. Most common organism isolated in widal positive cases is Klebsiella species. Incidence of faecal fistula in primary closure group is 25%. Length of hospital stay is significantly lower in primary closure with omental patch group($p < 0.001$)

CONCLUSION

Non specific perforation is the commonest cause of perforation followed by typhoid perforation. Primary closure with omental patch repair has shown better results compared to primary closure alone in terms of wound infection, intra abdominal abscess, burst abdomen, faecal fistula, septicaemia, mortality, length of hospital stay irrespective of site of perforation.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF GRAPHS

INTRODUCTION

Perforation of small bowel is an abdominal emergency faced by the general surgeon. Intestinal perforation is one of the most dreaded & common complication of typhoid fever, remarkably so in the developing countries where it usually leads to septic peritonitis.[1] Perforation of ileum is relatively uncommon in western societies except in regions where typhoid, tuberculosis & parasitic infestations are endemic.[2]

The pre-eminent complication of typhoid is perforation seen in 3rd week. The ileum is the main site of perforation. [3]

Perforated hollow viscus challenges the surgeon's skill and his knowledge of managing severely ill patient.[4] The current surgical options include primary double layered closure, primary double layered closure with omental patch,segmental resection with end to end anastomosis and primary ileostomy.

Though surgery is accepted as the definitive treatment, the choice of exact surgical procedure remains controversial.[5]Studies with controversial outcome have been published & there remains a difference of opinion as to the best surgical procedure in ileal perforation.

This study is undertaken to evaluate outcomes of ileal perforation closure by primary closure and primary closure with omental patch.

OBJECTIVES

1. Evaluate outcomes of ileal perforation management with primary closure.

2. Evaluate outcomes of ileal perforation management by primary closure withomental patch.

3. To evaluate which has better outcomes between primary closure and primary closure with omental patch in management of ileal perforation.

REVIEW OF LITERATURE

Hippocrates first used the term typhus (gr. cloudy) in 460 B.C. [6]

Aristotle was the first to describe intestinal injury as consequence of blunt abdominal trauma.[7]

Typhoid is most common cause of ileal perforation,tuberculosis, trauma and non-specific enteritis follow a close suit. Ileal perforation has an abrupt onset and a rapid downhill course with a high mortality if not treated.[8]Early surgery & adequate resuscitation are key to successful management of patients with typhoid perforation.

An Indian study reported that closure of tubercular perforation with or without bypass procedure has been shown to give poor results & hence resection & anastomosis is recommended. [9]

A recent indian study concluded that repair of typhoid perforation is a better procedure than temporary ileostomy due to its cost effectiveness & absence of complications related to ileostomy.[10]

Primary ileostomy should be given priority over other surgical options especially in those moribund patients who present late in the course of their illness or have multiple perforation with contamination of abdominal cavity.[1]

Recently a study concluded that primary closure with omental patch is a better option when compared to simple primary closure in enteric ileal perforation.[11]

Various factors influence overall prognosis and outcome of surgical treatment such as delayed presentation, adequate preoperative resuscitation, delay in surgery, number of perforations & degree of faecal contamination of peritoneal cavity .[8,12]

GROSS ANATOMY

The length of the alimentary tract in normal humans averages about 453 cm. from the nose to the anus. The duodenum is approximately 21 cm. long, and the colon is approximately 109 cm. long. The combined length of the jejunum and ileum is 261 cm., or about three fifths of the entire canal. The jejunum begins at the duodenojejunal angle, supported by the ligament of Treitz. The proximal of two fifth being jejunum, distal three fifth being the Ileum.

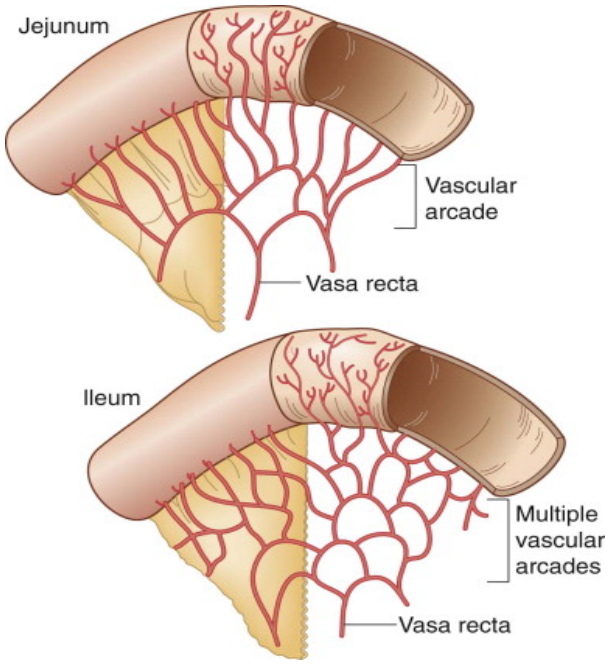
The ileum is thinner than jejunum. It is mainly in hypogastric and pelvic region.

There is no morphological line of distinction between the jejunum and ileum.

Following are some differentiating features.

CHARACTER	**JEJUNUM**	**ILEUM**
WALL	Thicker	Thinner
MESENTERIC FAT	Doesn't reach jejunal wall	Reaches wall of ileum
PEYER'S PATCHES	Present	Absent
VILLI	Larger	Smaller
ARTERIAL ARCADES	1 or 2	2 or 3
	Longer parallel vessels	Shorter parallel vessels

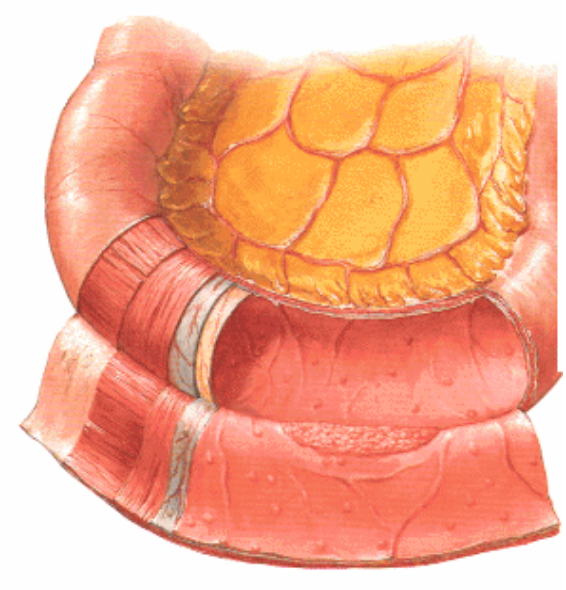
Fig No 1 : Comparision Of Jejunal & Ileal Vascular Arcades



MESENTERY:

The mesentery, a large fold of peritoneum, suspends the small intestine from the posterior abdominal wall. 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 mesentery contains blood vessels, nerves, lymphatics, and lymph nodes, as well as considerable fat. It attaches to the small intestine along the length of one side, the mesenteric border, leaving the remainder of the surface of the bowel covered by its visceral peritoneum, the serosa. The broad-based attachment of the mesenteric base stabilizes the small bowel and prevents it from twisting upon its blood supply.

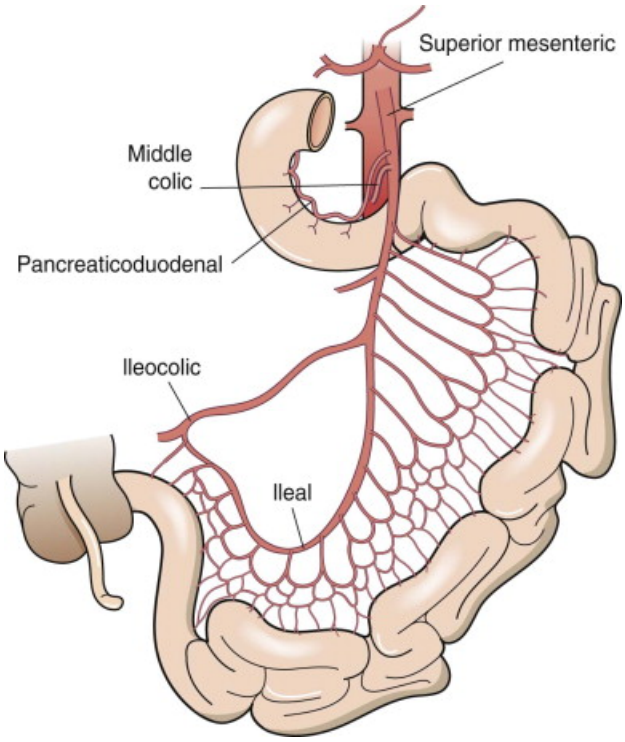
Fig no. 2 : Layers of ileal wall with peyer's patch



BLOOD SUPPLY:

The small intestine receives its blood supply from the superior mesenteric artery, the second largest branch of the abdominal aorta. The superior mesenteric artery courses anterior to the uncinate process of the pancreas and the third portion of the duodenum, where it divides to supply the pancreas, duodenum, and entire small intestine, as well as the ascending and transverse colon. The intestinal arteries branch within the mesentery to unite with adjacent arteries to form a series of arterial arcades before sending small straight arteries to the small intestine. The intestinal arteries contact the small intestine on the mesenteric border, where they pass toward the antimesenteric border, sending small branches into the layers of the intestine. The veins of the small intestine drain into the superior mesenteric vein, a major tributary to the portal vein.

Fig No. 3 : Blood supply of small bowel



LYMPHATICS:

Peyer's patches are lymph nodules aggregated in the submucosa of the small intestine. These lymphatic nodules are most abundant in the ileum. The lymphatic drainage from the small intestine passes into three sets of mesenteric nodes: a first set close to the wall of the small intestine, a second set adjacent to the mesenteric arcades, and a third set along the trunk of the superior mesenteric artery. The superior mesenteric preaortic group drains into the intestinal trunk, which drains into the cisterna chyli. The lymphatic drainage of the small intestine constitutes a major route for transport of absorbed lipid into the circulation.

INNERVATION:

The parasympathetic and sympathetic divisions of the autonomic nervous system provide the efferent nerves to the small intestine. The parasympathetic preganglionic fibers pass through the vagus nerves to synapse with neurons of the intrinsic plexuses of the intestine. The sympathetic preganglionic fibers arise from the ninth and tenth thoracic segments of the spinal cord and synapse in the superior mesenteric ganglion. The postganglionic sympathetic fibers pass along the branches of the superior mesenteric artery to the intestine. Pain from the intestine is mediated through thoracic visceral afferents, not vagal afferents. Although the vagus contains large numbers of afferent fibers, thoracic visceral afferents, not vagal afferents, mediate pain from the intestine.[13]

HISTOLOGY

The small intestine consists of four layers. From the lumen outward, they are the mucosa, the submucosa, the muscularis, and the adventitia or serosa.

MUCOSA:

The mucosa of the small intestine encompasses the epithelium, the lamina propria, and the muscularis mucosae. The mucosal surface has two important structural features: the villi and the crypts of Lieberkühn. The villi have a columnar epithelial surface and a cellular connective tissue core of lamina propria. Each villus contains a central lymphatic vessel called a lacteal, a small artery, a vein, and a capillary network. Human jejunal villi measure 0.5 to 1.0 mm. high and number 10 to 40 villi per square millimeter of mucosal surface. In addition to the vessels, the villi contain smooth muscle fibers extending from the muscularis mucosae, providing contractility to each villus. The crypts of Lieberkühn, or intestinal glands, reside adjacent to the bases of the villi and extend down to, but not through, the muscularis mucosae. The lamina propria between the intestinal epithelium and the muscularis mucosae contains blood and lymph vessels, nerve fibers, smooth muscle fibers, fibroblasts, macrophages, plasma cells, lymphocytes, eosinophils, and mast cells, as well as elements of connective tissue.[14]

Scanning electron micrographs provide an in-depth perspective of the mucosa with excellent resolution. The villi vary in shape from circular to flattened or finger-shaped. The finger-shaped villi are 0.1 to 0.25 mm. in diameter, are corrugated by deep horizontal clefts, and have holes 3 to 8 m. across on the surface, representing the openings of the goblet cells. The muscularis mucosa is a thin layer of smooth muscle separating the mucosa from the submucosa.[15]

CELLS OF THE EPITHELIUM:

Cells of the Villi. The columnar epithelial cells are responsible for absorption. These cells, 22 to 26m.tall, exhibit a striated luminal border (brush border) and a basally placed nucleus. The microvilli, which are projections 1 m. tall and 0.1 m. wide and are produced by numerous folds in the apical plasma membrane, account for the brush border appearance. The microvilli greatly increase the absorptive surface of the epithelial cell. The membrane of the microvillus is continuous, without fenestrations and it separates the lumen of the gut from the interior of the epithelial cell. The brush border contains high concentrations of digestive enzymes, particularly disaccharidases. The plasma membrane contains 80% to 90% of the disaccharidase activity of the intestinal cell. These findings indicate that the microvilli, besides increasing absorptive surface, perform an important digestive function.

Goblet cells are present in both the villi and the crypts. These cells have cytoplasm filled with mucous granules between the nucleus and the apical brush border. Intestinal goblet cells secrete their mucus by merocrine secretion. [13]

CELLS OF THE CRYPTS:

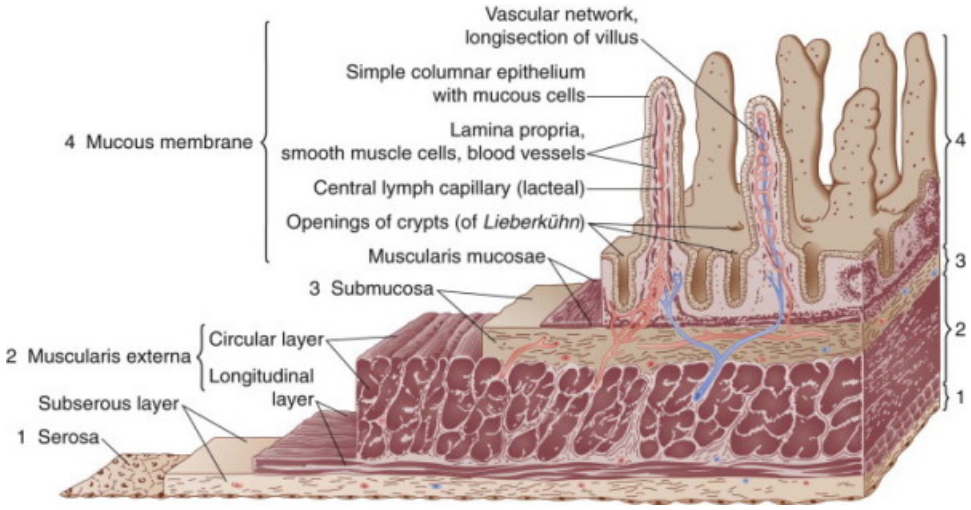
Enterochromaffin cells reside in the crypts of the small intestine and in other parts of the gastrointestinal system as well, including the esophagus, stomach, colon, gallbladder, and pancreas. These cells do not contact the intestinal lumen, and their secretory granules are usually below the nuclei away from the lumen, suggesting secretion into the blood rather than the lumen. The enterochromaffin cells have an endocrine function.

Paneth cells occur in the base of the crypts and are structurally similar to cells known to secrete large amounts of protein, such as pancreatic or parotid acinar cells.

The function of Paneth cells is unknown.

Undifferentiated cells, the most frequent cell in the base of the crypts, multiply and differentiate to replace lost absorptive cells.

Fig No 4 : Histology of Iieum



EPITHELIAL RENEWAL:

The epithelium of the small intestine is a dynamic, rapidly proliferating tissue in which old dying cells are constantly replaced by newly formed cells, thus maintaining the structural integrity of the mucosa. Mitotic division of undifferentiated cells occurs in the crypts. New growth replaces the population of intestinal epithelial cells every 3 to 7 days.[13]

SUBMUCOSA:

The submucosa is a strong fibroelastic and areolar connective tissue layer containing vessels, nerves, and lymph nodules.

Muscular Layer and Intramural Neural Structures

Two distinct layers of smooth or nonstriated muscle, an outer longitudinal coat and an inner circular coat, form the muscular portion of the small intestine. Intestinal smooth muscle fibers are spindle-shaped structures about 250 m. long. The plasma membrane of adjacent cells approximate at points, forming structures called nexuses. The nexuses allow electrical continuity between smooth muscle cells and permit conduction through the muscle layer.

The small intestine has four identifiable neural plexuses: (1) The subserous plexus, noticeable on the mesenteric attachment, forms the transition between the mesenteric nerve fibers and the myenteric plexus. Ganglia occur in the subserous plexus. (2) The myenteric plexus is located between the longitudinal and circular muscle layers and consists of three networks linking various ganglia and ramifying within the muscle layers. (3) The submucosal plexus is a network of nerve fibers and ganglia in the submucosa. (4) The mucous plexus consists of fibers from the submucosal plexus extending into the mucosa. This plexus does not contain nerve cell bodies. [13,14,16]

PHYSIOLOGY

DIGESTION AND ABSORPTION

Carbohydrate:

The digestion of starch by amylase probably occurs predominantly in the lumen of the alimentary tract. Maltose, maltotriose, and dextrin, as well as the dietary disaccharides lactose (glucose-galactose) and sucrose (glucose-fructose), are completely broken down to the constituent monosaccharides by the microvilli.

The intestinal cells actively transport glucose and galactose against a concentration gradient. Glucose and galactose compete for transport in a manner similar to competitive inhibition in other enzyme substrate systems. The active transport of sugars requires metabolic energy as well as oxygen. Sodium ion is important in the transport of glucose and galactose. Glucose and galactose are absorbed by carrier-mediated active transport. The absorption of glucose and galactose depends on Na+ movement into the cell produced by the Na+K+ ATPase, located on the basolateral cell membrane. Fructose, the other significant monosaccharide, is not absorbed by active transport but probably enters the intestinal cells by facilitated diffusion.[13,16]

Protein:

The intestinal enzyme enterokinase converts trypsinogen to trypsin. The activation of trypsinogen is autocatalytic; that is, trypsin also activates trypsinogen. Trypsin likewise activates the other pancreatic proteolytic enzyme precursors. Amino acids are the final product of protein digestion. However, some dipeptides are also absorbed.

Amino acids are absorbed from the intestinal lumen by carrier-mediated active transport. The transport of amino acids requires oxygen and sodium. The sodium pump on the basolateral cell membrane of the intestinal epithelial cells maintains an electrical potential across the brush border. Digestion and absorption of protein are usually 90% completed in the jejunum.[13]

Fat:

The bile salts that occur in humans—glycine or taurine conjugates of cholic acid, deoxycholic acid, or chenodeoxycholic acid—are detergents; they are water-soluble at one portion of the molecule and fat-soluble at the other. In solution, substances produce polymolecular aggregates called micelles,which can dissolve fat.[17]

Lecithin, a phospholipid, greatly enhances the capacity of bile salts to form micelles and dissolve fat. Bile salt and lecithin solubilize lipid in an aqueous environment to produce a micellar solution. This provides an optimal physicochemical environment for the action of pancreatic lipase. Pancreatic bicarbonate regulates the pH of the intestinal lumen to allow lipase to function optimally. An alkaline pH favors ionization of fatty acids and bile salts, which increases their solubility in micelles. An alkaline pH also increases the solubility of bile salts.

Chylomicrons pass from the epithelial cells into the lacteals, where they pass through the lymphatics into the venous system. Medium-chain triglyceride (C 8 to C 10) may be absorbed without hydrolysis and pass into portal blood rather than into lymph via the formation of chylomicrons.

The jejunum absorbs most dietary fat. Although unconjugated bile acids are absorbed in the jejunum by passive diffusion, the conjugated bile acids that form micelles are absorbed in the ileum by active transport. There they are almost completely absorbed and pass via the portal venous blood to the liver, for resecretion as bile.

Water and Electrolytes:

Large quantities of water enter the small intestine. Some water is ingested, but the digestive glands secrete a larger amount to provide the luminal environment for optimal digestion and absorption. Five to 10 liters of water enters the small bowel daily, whereas only about 500 ml. or less leaves the ileum and enters the colon. The small intestine, therefore, absorbs large quantities of water.[18]

The important factors in the movement of water across the intestinal mucosa are diffusion and osmotic filtration caused by osmotic or hydrostatic pressure differences across the membrane. Intestinal cells have a sodium pump (Na+K+ ATPase) on the basolateral cell membrane that moves Na+ out of the cell into the basolateral intercellular space. Movement of K+ into the cell accompanies the Na+ movement. The sodium pump produces a concentration gradient that moves Na+ into the cell from the lumen. This movement of Na+ by the sodium pump also transports glucose, amino acids, and oligopeptide into the intestinal epithelial cells.

In the jejunum, a small portion of sodium absorption is mediated by active transport, but most of jejunal sodium absorption occurs by bulk flow along osmotic gradients. The jejunum effectively absorbs bicarbonate against steep electrochemical gradients.[19]

The human ileum absorbs Na^+, Cl^- against steep electrochemical gradients; this absorption is unaffected by water flow and is not stimulated by glucose, galactose, or HCO_3^-. Potassium is passively absorbed from the intestine according to its electrochemical gradients.

Calcium is absorbed, particularly in the proximal small intestine (duodenum and jejunum), by a process of active transport. This ion is absorbed better from an acid than from an alkaline environment, which may explain the better absorption in the proximal intestine. Vitamin D and parathyroid hormone enhance calcium absorption.

An important electrolyte absorbed by the small intestine is iron. One of the important functions of the small intestine is to regulate the body pool of iron.[13,16]

MOTILITY

There are several types of visible small intestinal muscular activity. The segmenting contraction is a localized circumferential contraction of the circular muscle over a length of about 1 cm. of the small intestine. Segmenting contractions divide the luminal content within the area of contraction. Their rhythmic segmenting activity occurs in the proximal small intestine at about nine contractions per minute. Segmenting contractions occurring regularly and rhythmically in adjacent portions of the small intestine divide and subdivide the intestinal content, mixing it and exposing it to larger areas of mucosa, which facilitates digestion and absorption. Peristalsis consists of intestinal contractions passing aborally at a rate of 1 to 2 cm. per second through several centimeters of intestine. Peristalsis is slower in the distal than in the proximal small bowel. The major function of peristalsis is the distal movement of intestinal chyme.

THE MIGRATING MYOELECTRICAL COMPLEX (MMC):

During the interdigestive period, there are cyclically occurring contractions that move aborally along the intestine every 75 to 90 minutes during fasting. Most of these fronts of activity begin in the stomach or duodenum, last about 4½ minutes, and pass along the gut at about 6.8 cm. per minute. The MMC is thought to sweep or cleanse the intestine during the interdigestive period.Motilin may regulate the MMC.[16, 20]

REGULATION OF SMALL INTESTINAL MOTILITY:

1. Myogenic Factors:

Two types of electrical activity can be recorded from the small intestine. Slow-wave electrical activity begins in the longitudinal muscle layer of the duodenum and is propagated distally. This phenomenon, called the basic electrical rhythm (BER), is independent of the intrinsic neural plexuses, and is unrelated to motor activity. Intestinal spike potential may occur spontaneously during depolarization or from stretching of the bowel, and it is associated with motor activity.

2. **Neurogenic Factors:**

Intrinsic neural regulation is initiated by stimulation of the mucosa, particularly by distention, which causes contraction of longitudinal and circular muscle, propelling luminal content distally. The intrinsic nerve supply regulates rather than initiates motor action. In general, sympathetic activity inhibits motor function, whereas parasympathetic activity stimulates it. Epinephrine inhibits small intestinal motor activity, whereas acetylcholine stimulates it. Distention of the small intestine can inhibit small intestinal motility by the intestinointestinal inhibitory reflex.

Distention of the ureter, renal pelvis, or biliary system or peritoneal irritation may inhibit intestinal movements. [13]

3. . **Hormonal Factors:** Gastrointestinal hormones may be important in regulating intestinal motility. Gastrin stimulates gastric and intestinal motility and relaxes the ileocecal sphincter. Cholecystokinin-pancreozymin (CCK-PZ) also stimulates intestinal motility and may decrease intestinal transit time. Secretin and chemically similar glucagon inhibit intestinal motility.

IMMUNOLOGIC FUNCTION OF THE INTESTINE

The intestine is a source of immunoglobulin, particularly IgA.[21] It is believed that this immunoglobulin arises from plasma cells in the lamina propria, and after linkage with a protein synthesized by epithelial cells, it is secreted into the lumen. Secretory IgA contains antibody activities, the exact roles of which are not yet known.

ILEAL PERFORATION

Etiology

Ileal perforation is a serious complication of a spectrum of diseases. In developed countries these perforations are reported to be mostly because of foreign bodies,radiotherapy, crohns disease, drugs, malignancies and congenital malformations. Due to rare incidence of typhoid fever and TB, perforations because of these diseases are seldom encountered in these countries. On the other hand in the underdeveloped tropical countries small bowel perforation is quite a commonly encountered surgical emergency .[22,23] The most important cause is endemic prevalence of typhoid fever in these countries .[24]

Wani et al published the causes of ileal perforation as follows [25]

TABLE NO. 1

ETIOLOGY	NO.OF PATIENTS(%)
Typhoid	49(62)
Non-specific	21(26)
Obstruction	05(6)
TB	03(4)
Radiation enteritis	01(1)

Typhoid fever was the most common cause of ileal perforation in Karmarkar's series,followed by nonspecific perforation.[26]

In the series by Wani and Karmakar, non-specific and typhoid fever were the commonest Causes . The commonest cause of ileal perforation in the tropics is typhoid fever.[26,6,27]

Typhoid Perforation

Typhoid fever is endemic in poor and underdeveloped countries of the world causing fatal complications such as intestinal perforations, which leads to generalized peritonitis, septicaemia, fluid and electrolyte derangements.Typhoid intestinal perforation is a common cause of surgical acute abdomen in our environment.

The incidence of perforation varies considerably with the west African subregion having one of the highest perforation rates in the world (15-33%)., and reasons for this remain speculative. Despite decades of improvement in patient care, the morbidity and mortality of typhoid perforation remain high, and this is related to multiple variable factors.[28-32]

INCIDENCE

The reported rate of bowel perforation in typhoid fever varies from 0.5% to 78.6%.[33,34,35,36,37,38] Various studies have shown the following incidences

Table.No. : 2

Author	Year	Country	Number
Purohit	1976	India	0.5%
Archampong	1976	Ghana	20.5%
Thakkar	1979	India	3.77%
Arigbabu	1980	Nigeria	78.6%
Hadley	1984	South Africa	4%
Santillana	1991	Peru	7.8%
Hisconnez	1992	Turkey	0.58%

SEX AND AGE

There is a male preponderance in typhoid perforation. It predominantly occurs in younger age groups. It has been reported in patients from age 2 to 76 years. Perforation predominantly occurs in the second and third decades of life.[39,40,41]

SEASONAL VARIATION

Eggelston reported that over half the cases occurred between July and October.[23]

Hadley reported that 58% of cases occurred in the dry season .[35]Tarpley reported that 27% of patients were admitted during the driest quarter of the year and 21% during the rainiest quarter.[41]

PATHOLOGY

Typhoid fever is caused by a Gram-negative bacillus Salmonella typhi. The organism passes through the Peyer's patches without causing inflammation. Multiplication occurs in the reticuloendothelial system for 10-14 days. Seeding occurs into the blood stream corresponding to the clinical onset. During the second week of illness bacteria reach the gut and localize in Peyer's patches. Ulceration and mesenteric adenitis occurs. Necrotic areas appear in lymphoid tissue. This might lead to perforation of Peyer's patches .[36,42,8] Perforation is reported to occur commonly in the second week following onset of illness.[35,36,43,44] Keenan reported that 88% of patients perforated in the second week .[35]Santillana reported a patient who perforated within 24 hours of onset of clinical illness .[36]

MACROSCOPY

Peyer's patches are swollen and raised. The terminal ileum and caecum are affected. Ulceration occurs in the long axis of the bowel.

Perforation diameter varies with a mean of 5mm. Hadley reported that most of the perforations are smaller than 5mm .[35]Tarpley noted that the size of the perforation varied between 1mm and 6cm in size with most being less than 8mm in size.[41]

Multiple perforations are seen in 20% of patients.

MICROSCOPY

There is marked proliferation of reticuloendothelial cells of the lymphoid follicles locally and systemically. There is accumulation of mononuclear phagocytes. The macrophages form small nodular aggregates filled with red cells (erythrophagocytosis). The bacteria are sometimes visualized .

CLINICAL FEATURES

The onset of perforation is heralded by sudden increase of abdominal pain, vomiting and distention. Meier et al reported the following symptoms and signs of typhoid ileal perforation .[41]

Table No. :3

Symptoms		**Signs**	
Fever	93%	Abdominal distension	73%
Abdominal pain	90%	Rectal tenderness	24%
Vomiting	67%		
Diarrhoea	27%		

Eggleston reported that most of the patients had fever, malaise and sudden increase in abdominal pain. Examination revealed signs of toxemia and acute abdomen. Hyper resonance was present over the liver in 70% of patients and paralytic ileus in 68% of patients. 19.2% of patients were in shock .[23]

DIAGNOSIS

Clinical suspicion is often sufficient for diagnosis in endemic area.[35,36,37,12] Free gas may be present under the diaphragm. Abdominal paracentesis may reveal pus. Peritoneal lavage might be useful to detect bile or pus.[47]

The diagnosis of typhoid fever can be made by Widal test, culture of organism from blood, bone marrow, urine and stools. Newer diagnostic techniques have been introduced to enable rapid diagnosis of typhoid fever. Histopathology of the specimen might reveal etiology of perforation.

SEROLOGY

Widal test measures antibodies against the flagellar and capsular antigens of the causative organism. A positive diagnosis can be made from seventh to tenth day. This test is of less value in low endemic regions. Salmonella typhi can be isolated from blood, bone marrow aspiration stool and urine. In untreated patents blood culture is positive in 80% of the patients in the first week declining to 20-30% during later stages. Bone marrow might yield Salmonella typhi in the absence of a positive blood culture.[48] Stool cultures are frequently negative during the first week but are positive in 75% by the third week. The frequency of positive urine culture parallels that of stool culture.

NEWER METHODS

Currently several newer methods of diagnosis are under evaluation. Indirect heamagglutination, indirect fluorescent Vi Antibody and ELISA are more specific and sensitive when compared to the Widal test .[48] The use of monoclonal antibodies against Salmonella typhiflagellin and DNA probes for detection of Salmonella typhi in blood are promising developments. The newer techniques would enable rapid detection of antibodies or organism.[48]

Treatment

Appropriate management of typhoid perforation was controversial till 1960. Huckstep advocated conservative treatment in 1959. He proposed management of typhoid perforation on the lines of the Oschner-Scherren regimen. His reasons for this were:

1. The terminal ileum is friable and is liable to perforate at more than one spot.
2. The friable gut might not hold sutures.
3. Chloramphenicol therapy sterilizes bowel contents and adjacent loops might localize the perforation .[49]

Hook and Guerrant recommended surgery if there was no localization.[50]

Conservative management is associated with a substantial mortality. Presently recommended treatment is surgery.[38]

SURGICAL TREATMENT

Patients are resuscitated preoperatively with intravenous fluids and antibiotics. Tacyildiz et al reported that preoperative resuscitation, antibiotic therapy and total parental nutrition reduced mortality from 28.5% to 10% .[38]

Wani et al described the operation performed in her study.[25]

Table no. : 4

Operation performed	**No.of patients(%)**	**Death**
Simple closure	38(49)	1
Resection and end to end Ileo transverse anastomosis	33(42)	3
Side to side ileo transverse anastomosis	02(03)	1
Resection anastomosis	05(06)	1
Ileostomy	01(01)	-

The various surgical options are

1. DRAINAGE OF PERITONEAL CAVITY

It is done in moribund patients during resuscitation and preparation for surgery.[51] Flank drains are inserted under local anaesthesia. As the only procedure it carries an unacceptably high mortality. It may be used as a temporary measure or as a preliminary step prior to surgery in moribund patients.

2. SIMPLE CLOSURE

Freshening of the edges and closure has been recommended by Archampong.[12] He reported mortality of 17.3% with this procedure. Talwar et al recommended primary closure and limited surgery.[52] Excision of edge and simple transverse closure, either in a single layer or in two layers, have been widely practised by many workers.

3. WEDGE RESECTION AND CLOSURE

A wedge of ileal tissue is resected around the perforation and the defect is closed transversely in two layers. Mortality rates between 2.3% to 6.2% have been reported.[36,53]Ameh reported that a wedge resection is associated with a very high mortality rate.[53]

4. RESECTION – ANASTOMOSIS

Many workers claimed that segmental resection of the involved bowel may be necessary in the presence of multiple perforations and a severely diseased terminal ileum. The complication and mortality for resection-anastomosis were 37.50% and 21.47% respectively, very much less than that observed in other treatment modality.[54] Jarrett and Gibney recommend resection only for multiple perforations. Gibney recommended resection if there were three or more perforations.[8]

5. ILEO-TRANSVERSE COLOSTOMY

Eggleston et al advocated closure of the perforation with end-to-side ileotransverse colostomy; this takes the involved bowel out of the intestinal stream.[23] Although the mortality rate has not been improved by this method, a lowering of the morbidity rate has been achieved. The need for a second operation to restore ileal continuity has made the procedure less popular, and thus some workers prefer the use of side-to-side ileotransverse colostomy.[44]

6. TUBE ILEOSTOMY

Lozoya introduced tube ileostomy in 1948.[55] Many workers have carried out this procedure using a size 24FG Foley catheter, passing it through either the perforation or the stab wound in the least inflamed and edematous part of the ileum .[44,45,56] The procedure has been described as quick to carry out, simple and effective in decompressing the bowels; also it prevents further contamination of the peritoneal cavity from either reperforation or fresh perforations. Maloney in Vietnam,has reported to have used this method with a very good outcome.[45] Also, Kaul and Ardhanari and Rangabashyam in India recorded a significant reduction in the mortality rate using tube ileostomy, although Chamber [57]and Lizarralde[44]used the same method with mortality ranging from 25 to 35%.

Bhalerao et al recommended exteriorization of suture line, which prevents contamination of the peritoneal cavity in case of leak. Santillana recommended exteriorization in moribund patients. If fistulae forms they invariably heal by conservative management.[27,36] Good peritoneal lavage and placement of drains to remove pus was recommended. Two-layer closure was recommended to decrease the risk of leakage.[43,45]

A midline or Para median incision was commonly used. Talwar.et.al recommended Rutherford Morrison incision in the presence of a confirmed preoperative diagnosis of perforation. If there is fulminant sepsis in the abdominal cavity due to the formation of faecal fistula or any other cause laparostomy might be done. Laparostomy is defined as a laparotomy without reapproximation and suture closure of abdominal fascia and skin. The abdominal cavity is left open. It helps drainage of pus and prevents deleterious rise of intra-abdominal pressure. The wound can be closed after control of sepsis. The disadvantages are that the exposed intestine might perforate and formation of an incisional hernia. It may be combined with continuous postoperative peritoneal lavage.

MEDICAL THERAPY

The antibiotic of choice for S.typhi infection is chloramphenicol. The recommended dose is 3-4g/day or 50-70mg/kg for children. The dose may be slowly reduced to 2g/day or 30mg/kg once the patient is afebrile. The duration of treatment is 2 weeks.[58] Combination of chloramphenicol with agents more effective against anaerobes (metronidazole or clindamycin) and against aerobic gram negative bacilli (aminoglycosides) would improve the spectrum of coverage needed by patients with ongoing typhoid fever and faecal peritonitis.[59] Results with the combination of chloramphenicol and parenteral metronidazole have been encouraging. There was a significant improvement in survival when either metronidazole, gentamicin or both were added to the chloramphenicol. The addition of both would be logical. There might be a tendency to reserve additional antibiotics for more grossly contaminated cases. Improvement in survival was most marked, however, for minimally contaminated cases.

With the advent of resistance to chloramphenicol, quinolones have replaced chloramphenicol as the drugs of choice.[60,61] Ciprofloxacin is used in a dose of 200 to 750 mg twice a day. Resistance to this drug is still rare.[60] Ceftriaxone may be used as an alternative. The dose is 3-4g/day for 3 days in adults and 80mg/kg/day in two divided doses for 5 days in children.[60]

COMPLICATIONS

Santillana in his series of 96 patients reported a complication rate of 71.9%.[36]

MORTALITY

Mortality rates ranging from 20-40% are most commonly reported for typhoid ileal perforation.[37,48,62,63] Rates as low as 3-9% have been reported from areas in the development world with better economic conditions. Such mortality rates have been achieved by the addition of close electrolyte and blood gas monitoring, intensive care unit nursing, central venous pressure monitoring and use of total parenteral nutrition.[36,51,8,64] Most of these measures are beyond the reach of the majority of hospitals in the developing world, especially in rural areas. Variables that can be manipulated to improve survival in such locations include more aggressive fluid and electrolyte resuscitation, the type of surgical procedure and antibiotic regimen.[37,65]

PROGNOSTIC FACTORS

Typhoid ileal perforation is still very common in tropics, with high morbidity and mortality. The mortality ranges between 9 and 43% with survivors having severe wound infection and history of long hospital stays.27,[66,67,68,]Many factors such as late presentation, adequate preoperative resuscitation, delayed operation, the number of perforation and the extent of fecal peritonitis have been found to have a significant effect on the prognosis.[42,69,70,71] The sex and age distributions had no effect on the postoperative outcome.

Adesunkanmi reported that late presentation, delay in operation, multiple perforation and drainage of copious quantities of pus and fecal material from peritoneal cavity adversely affect the incidence of fecal fistula and the mortality rate. The development of fecal fistula significantly affected the mortality rate. Early presentation, single perforation and moderate amounts of pus/fecal matter drainage of the peritoneal cavity enhanced the development of wound infection, wound

dehiscence and residual intraabdominal abscess. Surviving for more than 10 postoperative days tends to give a better chance of recovery.

Archampong reported that the duration of illness, perforation-operation interval, urinary output before surgery, blood urea and serum potassium influenced survival.Survival was independent of hemoglobin level, presence of peripheral circulatory failure, sickling status and number of perforations.[37]

Bose et al reported that mortality in small bowel perforation was significantly influenced by perforation-operation interval, presence of multi-organ system failure and septic shock. Mortality was not influenced by haemoglobin, serum electrolyte levels, age and sex of the patients. Patients were stratified in to four groups depending on their general condition.

Group 1 - Patients with normal parameters

Group 2 - Patient is conscious, afebrile, PR 90-110/min, SBP 90-110mm Hg, Urine output > 30ml/hour

Group 3 - Patient is febrile, moderately dehydrated with PR 110-130/min,BP 80-90mm Hg, Urine output 20-30ml/hour

Group 4 - Patient is disoriented, BP < 80 mm Hg, febrile or hypothermic, Urine output<20ml/hour.

There was no mortality in the first two groups whereas groups 3 and 4 had a mortality of 19.29% and 53.8%, respectively.[72]Talwar and Sharma reported that increasing the time interval between the perforation and surgery and feculent peritonitis increase the mortality. Mortality was least with early primary closure.[52]

Early prognostic evaluation of abdominal sepsis can easily be done by various scoring systems. Acute Physiology and Chronic Health Evaluation score (APACHE II) and Manheim peritonitis index predict the outcome of peritonitis.

TRAUMA

Trauma is a more common cause of ileal perforation in developed countries. The injury may be due to blunt or penetrating trauma. The vast majority of traumatic perforations are from automobile or road traffic accidents. The penetrating injuries are commonly knife stabs or gunshot wounds. Kaul et al had 10 cases of ileal perforation in a series of 24 traumatic bowel perforations.[73]Karmakar et al had two cases of traumatic perforations in their series of 30 cases of ileal perforations.[26] Scully et al in their series of 20 cases of small bowel rupture following blunt trauma, had 2 cases of ileal perforation.[74]Blumgart reported small bowel injuries in patients involved in high-speed traffic accidents.[75] The mechanisms of injury postulated are, 1. Crushing or pinching of bowel between the spine and a blunt object 2. Rupture of a closed loop due to increased abdominal and intraluminal Pressure.[74]

Paran reported two patients with perforation of the terminal ileum in whom abdominal complaints evolved only 24-48 hours after trauma.[76]They proposed a mechanism involving damage to the bowel wall leading to late rupture up to 48 hours after trauma. The diagnosis of injury is based on clinical findings, X-ray and abdominal paracentesis. X-ray might reveal free gas under the diaphragm. Four-quadrant needle aspiration was positive in 21 of 24 cases of small bowel perforation reported by Koul.[73] Diagnostic Peritoneal Lavage may reveal blood or bile.[75] Marshal Orloff recommended debridement and closure for small bowel perforations while recommending resection-anastomosis for large wounds or multiple perforations in a segment of bowel. Mortality should be less than 5% in the absence of injury to other organs or systems.[76]

TUBERCULOSIS

In the past, abdominal tuberculosis caused by ingestion of milk contaminated by Mycobacterium bovis frequently complicated extensive pulmonary tuberculosis, but the rate of intestinal tuberculosis in the absence of recognizable pulmonary disease has increased.[75] The incidence of intestinal tuberculosis is increasing in the west due to immigration from third world countries, aging population and increasing incidence of human immune deficiency (HIV) infection.[77]

Free intestinal perforation is an uncommon complication of intestinal tuberculosis because of reactive thickening of the peritoneum and formation of adhesion with surrounding tissues.[78] It accounts 1-10% of abdominal tuberculosis cases and it has a poor prognosis with mortality rate higher than 30%.[79,80]

S. Talwar et al have found 19% of non-traumatic small bowel perforation in 308 patients were due to intestinal tuberculosis.[81]Badoui et al in Switzerland, also reported eleven cases of intestinal tuberculosis perforation, ten of them were immigrants from countries endemic for tuberculosis.[82]

Free perforation in intestinal tuberculosis usually occurs in the terminal ileum [83] and it can occur in patient during anti tuberculosis therapy.[84] Specific diagnostic investigations are not available. Plain x-ray has shown free air in only 25-50%. Fifty percent of the extra pulmonary tuberculosis patients have normal chest radiography.[85,86,87] Peritonitis, occurring in a patient with chest radiography indicative of tuberculosis should lead one to suspect a perforated tuberculosis ulcer.[84] In patients with intestinal tuberculosis who presented with generalized peritonitis should have exploratory laparotomy. However, in equivoval cases computed tomography helps in identifying the perforation. Makanguola has shown that computed tomography can provide a diagnosis of intestinal tuberculosis in 81% of the cases.[88]

In 90% of the cases, perforation is solitary, but multiple perforations occur in 10-40% of patients[89] and are associated with a poor prognosis, therefore immediate operative intervention is needed to be undertaken. Resection of the affected small bowel segment and end to end anastomosis proved to be the best method of treatment.[79] Simple repair of the perforation is not recommended because of the high incidence of leak and fistula formation. High mortality and morbidity reported (more than 29.3%) but the rate was significantly less in patients operated within 36 hours of perforation.[81]

MECHANICAL CAUSES

When the perforation occurs secondary to a distal obstruction due to causes such as hernias, bands, volvulus, intussusception and obstructing growths it is considered to be due to a mechanical cause. The cause is vascular strangulation following obstruction either by a hernia or a band. And gangrenous segment of bowel ruptures possibly as a result of delayed surgical treatment.[42]Increased intraluminal pressure may also lead to perforation. Mechanical causes are the one of the commonest causes of bowel perforation in the western world. These were responsible for 18 out of 76 cases of small bowel perforation as reported by Chaikof. The causes were adhesions in 12 patients, hernia in 4 and obstructive carcinomas in 2 patients.[90]Dixon et al in their series of 54 patients reported 13 cases due to mechanical causes – adhesions in 8, colonic cancer in 2, gall stones in 2 and small bowel volvulus in one patient.[91]

NON-SPECIFIC PERFORATION

When the etiology of ileal perforation is not identified, it is termed as a non-specificl perforation. Dixon et al in their series had such results in 14 out of 54 patients.[91] Karmakar et al in their series of 30 patients of ileal perforation had 7 cases of nonspecific perforations.[26]Many of these cases may be due to undiagnosed typhoid or other non-specific causes such as diet, drugs, viral or parasitic infections and infestations. It was earlier attributed to undiagnosed typhoid but these patients have different outcomes when compared to those with typhoid perforation. It has been proposed that sub mucosal vascular emboli may be responsible for such perforations.

Drugs such as potassium tablets may cause ulceration and subsequent small bowel perforation.[92]

DIVERTICULITIS

Perforation of diverticula is a rare cause of small bowel perforation. Huttunen et al in their series of 24 patients of perforation had this as the etiological factor in 4 patients, one with perforated ileal diverticulum, two with divertuculitis and one with ectopic gastric mucosa in a perforated Meckel's diverticulum.[93] Bhalerao et al had two patients with perforated diverticula in their series of 32 patients.[47] Meckel's diverticulum occurs in 0.3% to 2.5% of population. Gastric mucosa is found in up to 38% of Meckel's diverticula.[69] Perforation of an acquired diverticulum is rare. The gastric mucosa in a Meckel's diverticulum may lead to ulceration, which might perforate.[93] Resection of the diverticulum with the adjacent ileum is recommended.[90]

INFLAMMATORY BOWEL DISEASE

Free perforation is a rare complication in Crohn's disease. Ileum is the commonest site of perforation in this disease. Steinberg et al in their series of seven patients of Crohn's with free perforation of small bowel had five with ileal perforation.[94] Dixon et al in their series of 54 patients had 5 with Crohn's disease.[91] Chaikof reported 16 cases of Crohn's in the 76 patients of non-traumatic small bowel perforation.[90]

Perforation in Crohn's disease occurs during an acute exacerbation and is usually associated with distal obstruction. Simple closure is inadequate and has poor results. Menguy recommends primary excision and creation of a double-barrelled ileocolostomy with closure of stoma at a later date.

MATERIALS AND METHODS

MATERIALS

This prospective group comparative study included 40 patients with ileal perforation admitted in Sri Devaraj Urs Medical College and attached teaching hospitals, Kolar, from February 2012 to June 2013 satisfying all the inclusion criteria mentioned below after clearance from the ethical committee .

INCLUSION CRITERIA

All cases admitted with peritonitis diagnosed to have ileal perforation .

EXCLUSION CRITERIA

1. Multiple ileal perforations.
2. Patients not consenting for surgery.

METHODS

1. Informed consent will be obtained from the patients under study.
2. Investigations done are routine blood investigations, Widal test , blood culture , x-ray erect abdomen. ultrasound abdomen, abdominal paracentesis.

3 Patients are grouped into 2 groups

a) Group I - 20 patients

b) Group II - 20 patients

All odd numbered patients will be included under **group I** will undergo primary closure.

All even numbered patients will be included under **group 2** will undergo primary closure with omental patch.

4).Outcomes are compared with respect to wound infection, early post operative obstruction, Burst abdomen, intra abdominal abscess, development of faecal fistula, septicaemia, & mortality.

5) Follow up is for a period of one year.

6) Student t test and fisher exact test are used for analysis

OPERATIVE PHOTOGRAPHS

Fig No. 5 : Ileal Perforation

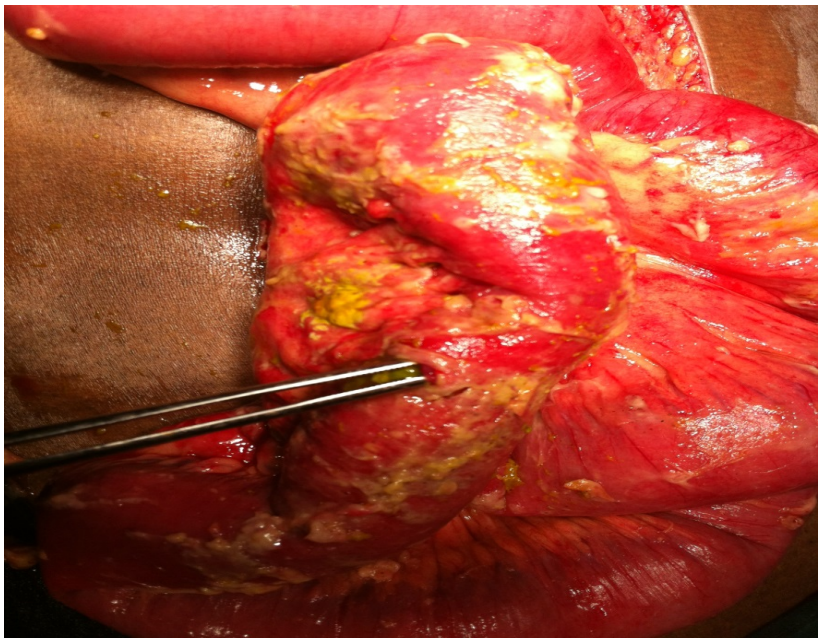


Fig No. 6 : Primary Closure Of Ileal Perforation

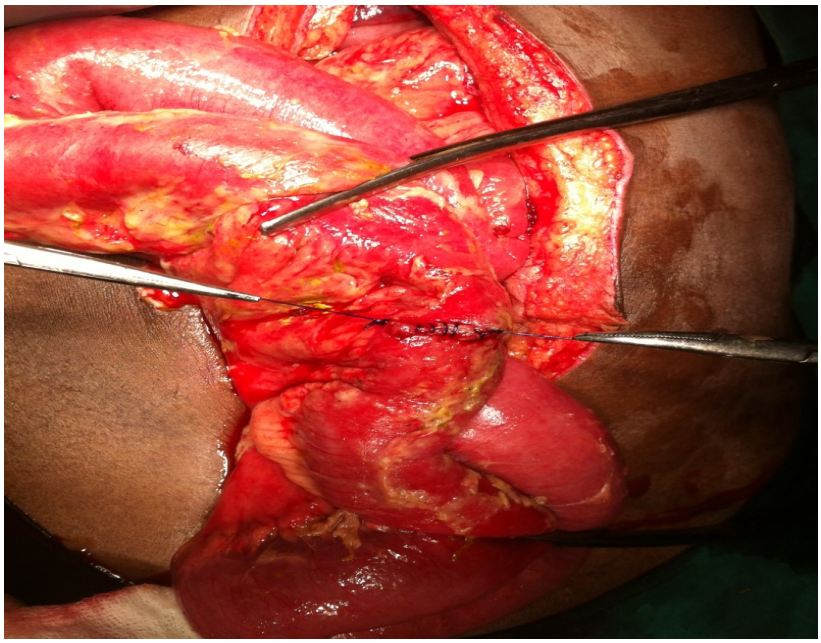


Fig No 7 : Omental Patch Closure

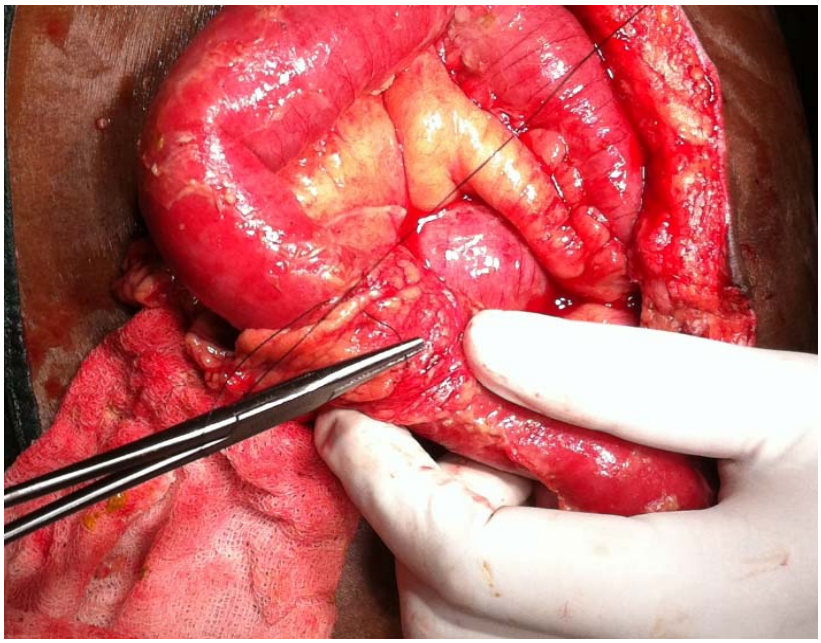
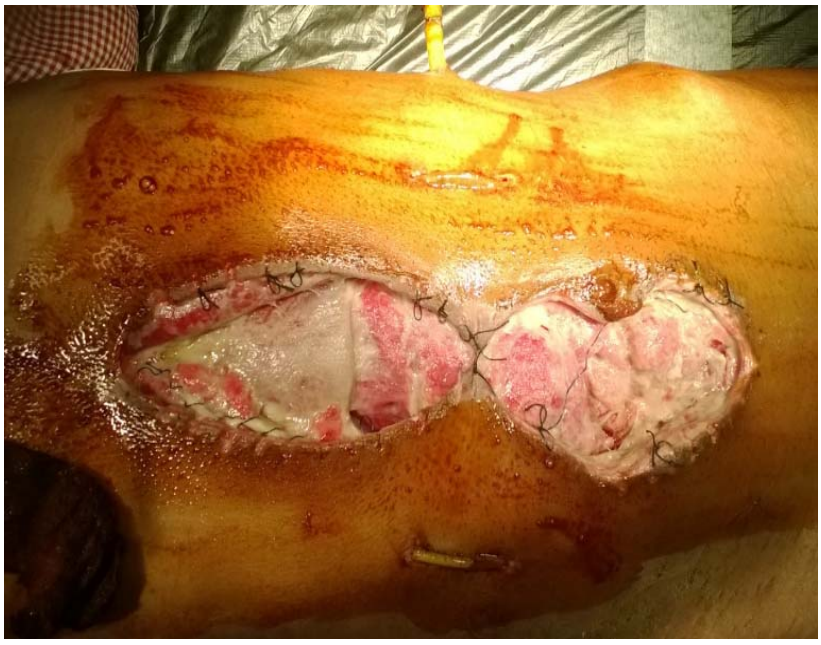


Fig No. 8: Burst Abdomen Following Primary Closure Of Ileal Perforation



RESULTS AND OBSERVATIONS

During the present study between February 2012 and june 2013 , a total of 40 patients with ileal perforation were divided into two groups of 20 patients each.

GROUP I – underwent primary closure alone

GROUP II - underwent primary closure with omental patch

Results were analysed and are as follows

1) AGE DISTRIBUTION

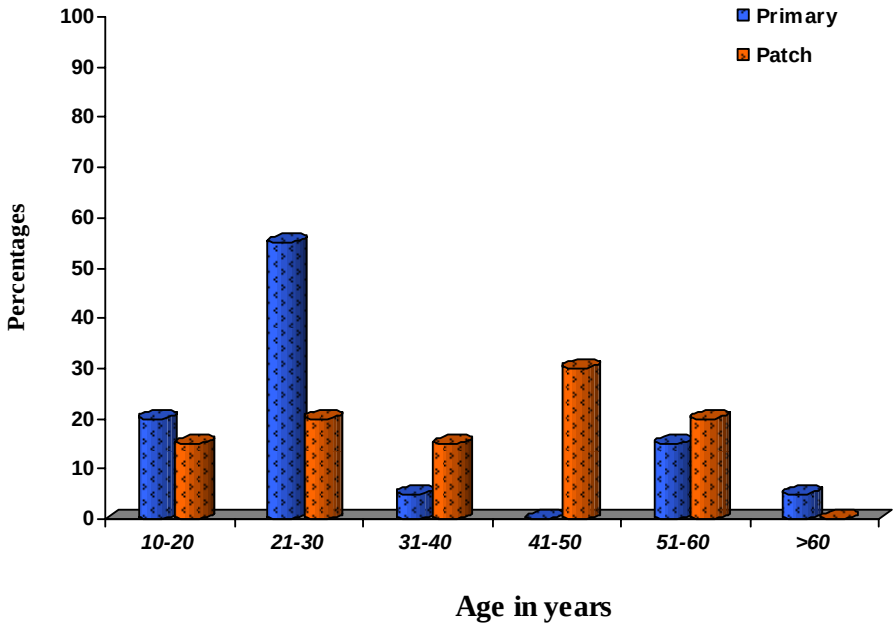
TABLE NO. 5 : Table showing age distribution

Age in years	Primary		Patch	
	No	%	No	%
10-20	4	20.0	3	15.0
21-30	11	55.0	4	20.0
31-40	1	5.0	3	15.0
41-50	0	0.0	6	30.0
51-60	3	15.0	4	20.0
>60	1	5.0	0	0.0
Total	**20**	**100.0**	**20**	**100.0**
Mean ± SD	**30.7±15.34**		**37.85±15.27**	

p=0.148 – Insignificant

In our study ileal perforation is most commonly seen in age groups of 10-20 & 21-30 years. Mean age of presentation was 34.27 yrs. Mean age was 30.7 yrs with a standard deviation of 15.34 in primary closure group. Mean age was 37.85 yrs with a standard deviation of 15.27 in primary closure with omental patch.

Graph No.1 : Graph Showing Age Distribution



2) SEX DISTRIBUTION

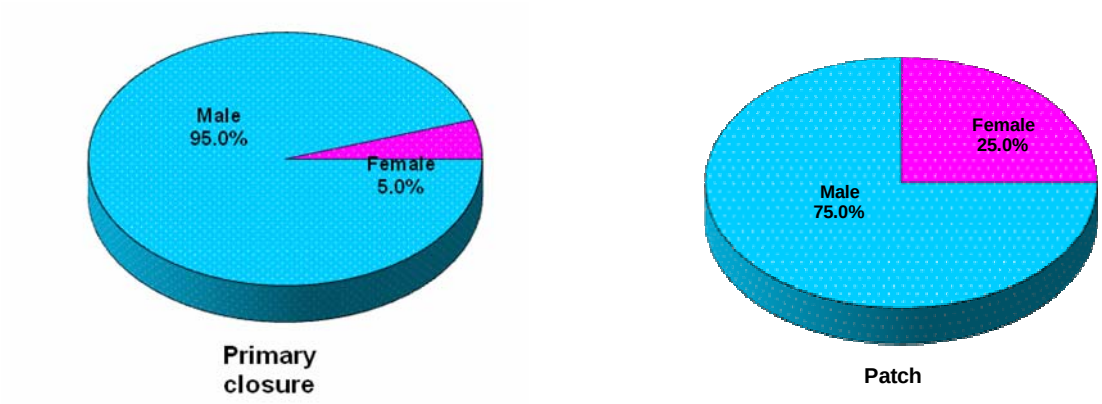
TABLE NO. 6 : Table showing sex distribution

Gender	Primary		Patch	
	No	%	No	%
Male	19	95.0	15	75.0
Female	1	5.0	5	25.0
Total	20	100.0	20	100.0

p=0.182

In this study 85% were males, 15% are females. In primary closure group 95% are males & 5% are females. In primary closure with omental patch group 75% are males & 25% are females.

Fig 9: Pie Diagram Showing Sex Distribution



3) DISTRIBUTION OF SYMPTOMS & SIGNS

TABLE NO. 7 : Table showing distribution of symptoms & signs

History	Primary (n=20)		Patch (n=20)		p value
	No	%	No	%	
Fever					
No	6	30.0	8	40.0	**1.000**
Yes	13	65.0	11	55.0	
Pain abdomen					
No	0	0.0	0	0.0	**1.000**
Yes	20	100.0	20	100.0	
Constip.					
No	16	80.0	15	75.0	**0.047***
Yes	4	20.0	5	25.0	
Vitals					
No	0	0.0	0	0.0	**1.000**
Yes	20	100.0	20	100.0	
Tenderness					
No	0	0.0	0	0.0	**1.000**
Yes	20	100.0	20	100.0	
Rigidity					
No	3	15.0	4	20.0	**1.000**
Yes	17	85.0	16	80.0	
Distension					
No	14	70.0	17	85.0	**0.451**
Yes	6	30.0	3	15.0	
Bowel sounds					
No	11	55.0	5	25.0	**0.470**
Yes	9	45.0	15	75.0	

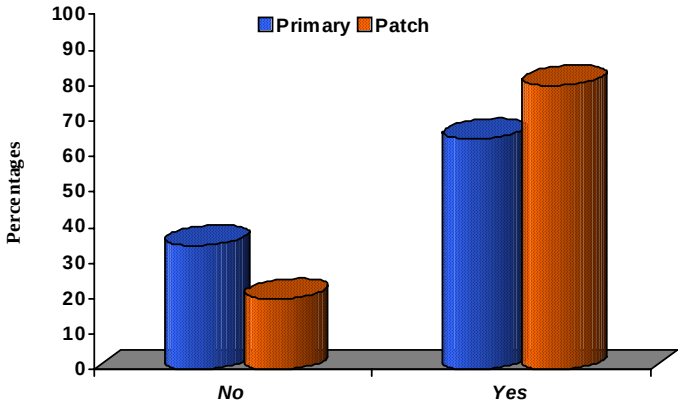
In our study 60% of patients had fever. 100% of patients had pain abdomen. Features suggestive of peritonitis such as tenderness is seen in 100% of patients. Rigidity is seen in 82.5% of patients.

4) INVESTIGATIONS

TABLE No. 8 : Table showing distribution of investigative work up

Investigations	Primary (n=20)		Patch (n=20)		P value
	No	%	No	%	
X ray erect abdomen					
No	7	35.0	4	20.0	**0.288**
Yes	13	65.0	16	80.0	
Ascitic fluid c/s					
No	4	20.0	9	45.0	**0.487**
Yes	16	80.0	11	55.0	
Pus c/s					
No	10	50.0	12	60.0	**0.512**
Yes	10	50.0	8	40.0	
Blood c/s					
Negative	20	100.0	20	100.0	**1.000**
Positive	0	0.0	0	0.0	
Widal test					
Negative	8	45.0	12	65.0	**0.487**
Positive	11	50.0	7	30.0	
Not done	1	5.0	1	5.0	

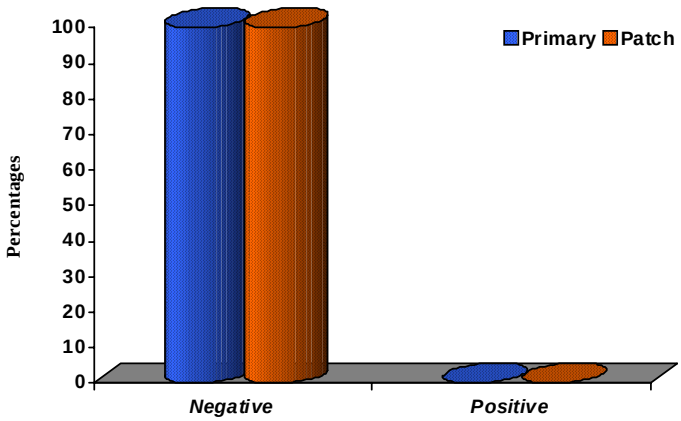
Graph No 2: Graph showing results of x ray erect abdomen



X ray erect abdomen

X- RAY ERECT ABDOMEN- In present study erect abdomen x-ray showed air under diaphragm in 65% of patients in primary closure group and 80% of patients in primary closure with omental patch group. Both groups together air under diaphragm on x-ray erect abdomen was seen in 72.5% of patients.

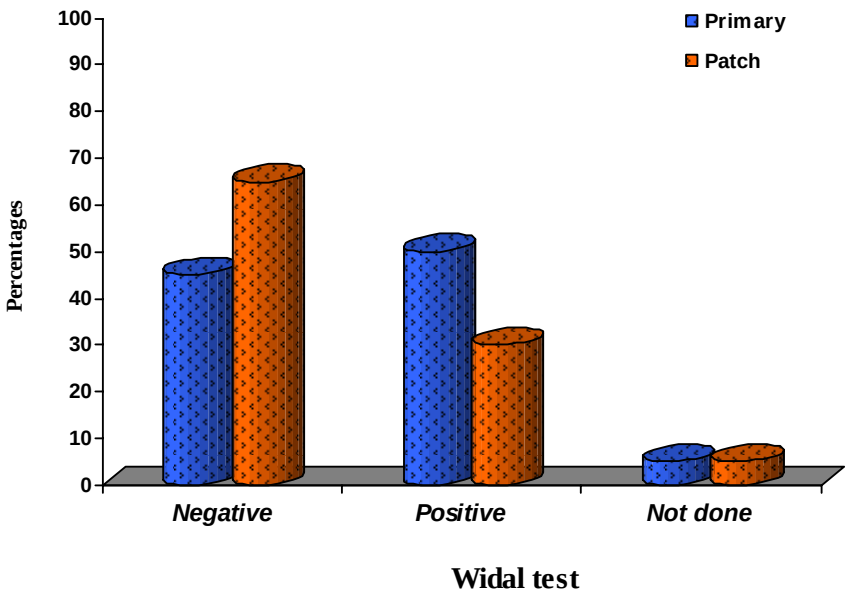
GRAPH NO. 3: Graph showing results of blood c/s



Blood c/s

BLOOD C/S- Negative in all patients in both the groups.

Graph No 4 : Results of Widal Test

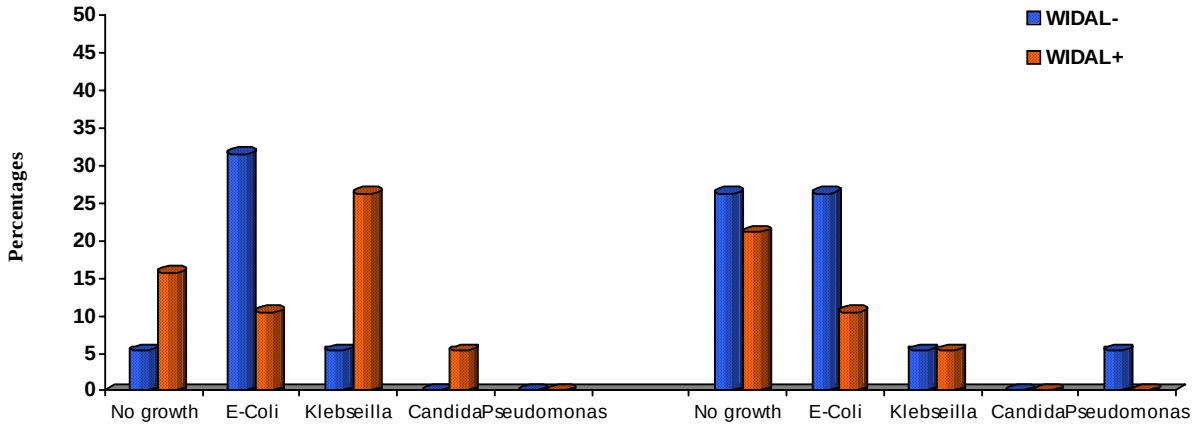


WIDAL TEST- widal test was positive in 50% of cases in primary closure group and 30% of cases in primary closure with omental patch group.

ASCITIC FLUID ANALYSIS

TABLE NO.9 : Results of ascitic fluid analysis

ASCITIC FLUID	Primary (n=19)		Patch (n=19)	
	WIDAL-	WIDAL+	WIDAL-	WIDAL+
No growth	1(5.3%)	3(15.8%)	5(26.3%)	4(21.1%)
E-Coli	6(31.5%)	2(10.5%)	5(26.3%)	2(10.5%)
Klebsiella	1(5.3%)	5(26.3%)	1(5.3%)	1(5.3%)
Candida	0	1(5.3%)	0	0
Pseudomonas	0	0	1(5.3%)	0
p value	0.090+		0.883	



Graph No 5 : Results of ascitic fluid analysis

In our study most common organism isolated in widal positive cases is Klebsiella species. It is seen in 26.3% of patients in primary closure group and 5.3% of patients in primary closure with omental patch group.

Most common organism isolated in widal negative cases is Escherichia coli. It is seen in 31.5% of patients in primary closure group and 26.3% of patients in primary closure with omental patch group.

5)ETIOLOGY

TABLE NO. 10 : Analysis of etiology of perforation

ETIOLOGY	**PRIMARY** **No.=20**	**PATCH** **No.=20**
TYPHOID	11(55%)	7(35%)
NON SPECIFIC	8(40%)	12(60%)
TRAUMA	1(5%)	1(5%)

In our study most common cause of ileal perforation is non specific perforation seen in 50% of patients both groups put together.

Typhoid perforation is seen in 45% of patients.

Ileal perforation secondary to trauma is seen in 5% of patients.

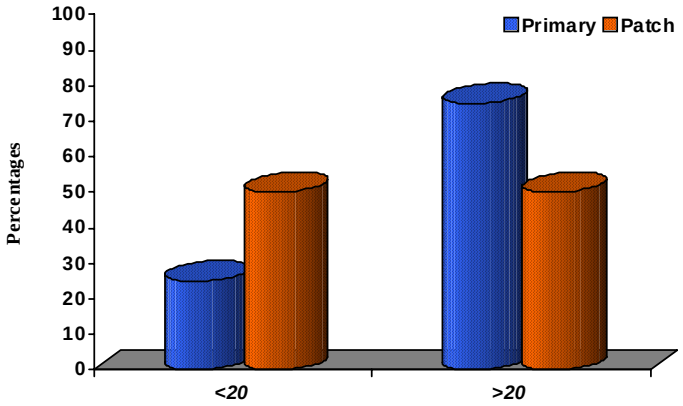
5) OPERATIVE DETAILS

TABLE NO.11 : Analysis of operative findings

Operative details	Primary (n=20)		Patch (n=20)		p value
	No	%	No	%	
No. of perf.					
1	20	100.0	20	100.0	**1.000**
2	0	0.0	0	0.0	
Site from ICJ					
<20cm	5	25.0	10	50.0	**0.047***
>20cm	15	75.0	10	50.0	
Size of perforation					
<10mm	12	60.0	16	80.0	**0.168**
>10mm	8	40.0	4	20.0	

NO.OF PERFORATIONS- In both groups all cases had single perforations

Graph No 6: Analysis Of Patients According To Site Of Perforation

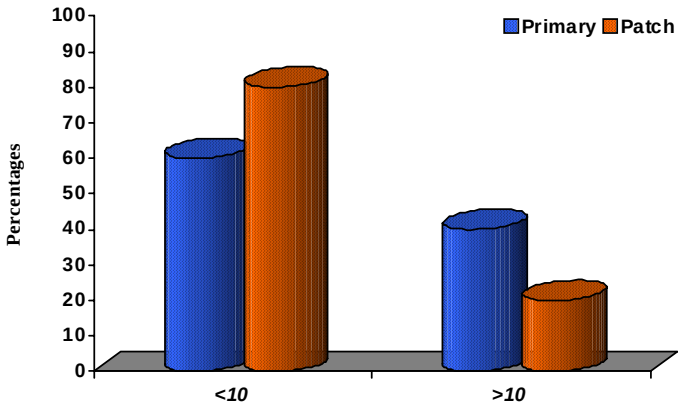


Site from ICJ

SITE OF PERFORATION FROM ICJ- study showed perforation within 20cm proximal to ICJ found in 75% of patients in primary closure group and 50% of patients in primary closure with omental patch group.

Perforation found more than 20cm proximal to ICJ was seen in 25% of patients in primary closure group and 50% of patients in primary closure with omental patch group.

Graph 7 : Analysis of cases according to size of perforation



Size of

SIZE OF PERFORATION- In present study size of perforation is < 10mm in 60% of patients in primary closure group and 80% of patients in primary closure with omental patch group.

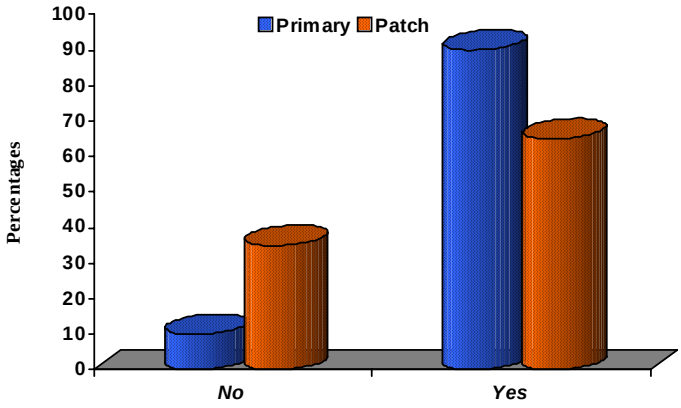
Size >10mm in 40% of patients in primary closure group and 20% of patients in primary closure with omental patch group.

6)POST OPERATIVE COMPLICATIONS

TABLE NO. 12 : Analysis of postoperative complications

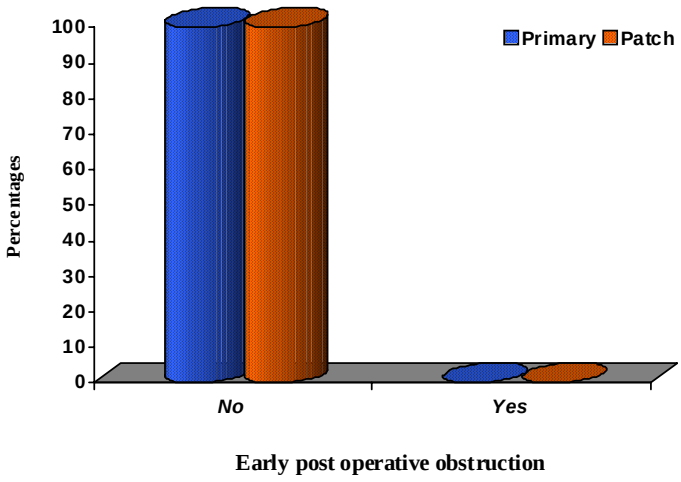
Post op complications	Primary (n=20)		Patch (n=20)		p value
	No	%	No	%	
Wound infection					
No	2	10.0	7	35.0	0.127
Yes	18	90.0	13	65.0	
Early post operative obstruction					
No	20	100.0	20	100.0	1.000
Yes	0	0.0	0	0.0	
Burst abdomen					
No	16	80.0	20	100.0	0.106
Yes	4	20.0	0	0.0	
Intra abdominal abscess					
No	19	95.0	20	100.0	1.000
Yes	1	5.0	0	0.0	
Faecal fistula					
No	15	75.0	20	100.0	0.047*
Yes	5	25.0	0	0.0	
Septicaemia					
No	17	85.0	20	100.0	0.231
Yes	3	15.0	0	0.0	
Mortality					
No	18	90.0	20	100.0	0.487
Yes	2	10.0	0	0.0	
Cause of mortality					
No	18	90.0	20	100.0	0.487
Yes	2	10.0	0	0.0	

Graph 8 : Comparison of wound infection among the two groups



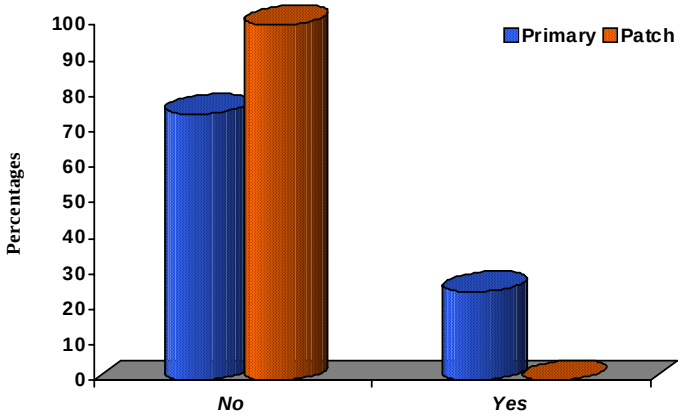
WOUND INFECTION- 90% of patients in primary closure group and 65% of patients in primary closure with omental patch group had wound infection while it was seen in 77.5% of patients in the entire study group.

Graph 09 : Analysis of incidence of early postoperative obstruction



EARLY POST OPERATIVE OBSTRUCTION-In present study it is not seen in both the groups.

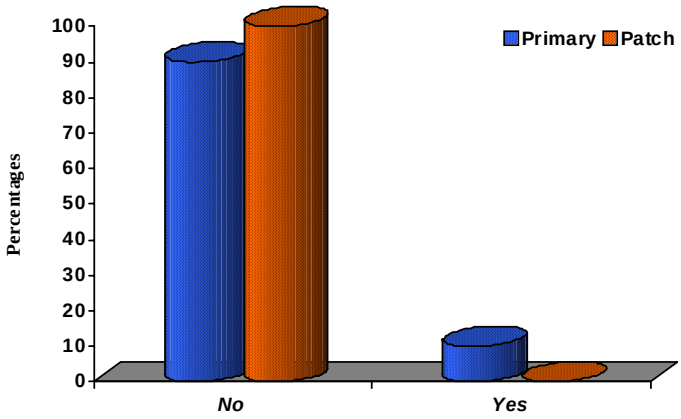
Graph 10 : Analysis of incidence of faecal fistula



Faecal fistula

FAECAL FISTULA- In this study faecal fistula is seen in 25% of cases in primary closure group and no case had faecal fistula in primary closure with omental patch.

Graph 11: Analysis of mortality in study population



Mortality

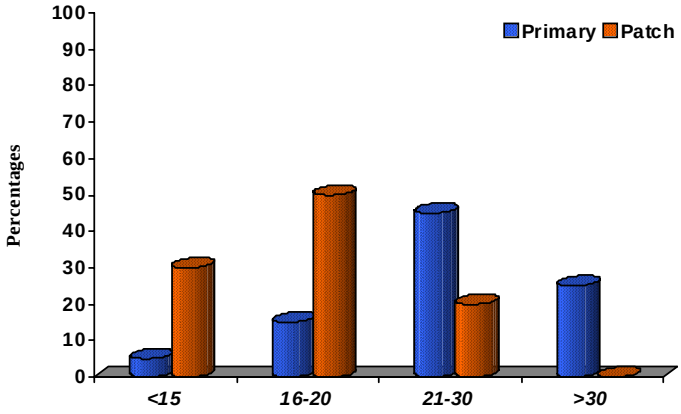
POST OPERATIVE MORTALITY- In our study post operative mortality is seen in 10% of cases in primary closure group and none in primary closure with omental patch group.

7) LENGTH OF HOSPITAL STAY-

TABLE NO. 13 : Analysis of length of hospital stay among the study groups

Length of hospital stays (days)	Primary		Patch	
	No	%	No	%
<15	1	5.0	6	30.0
16-20	3	15.0	10	50.0
21-30	9	45.0	4	20.0
>30	5	25.0	0	0.0
Total	**20**	**100.0**	**20**	**100.0**
Mean ± SD	**25.44±6.94**		**17.05±5.52**	

Graph 12 : Analysis of length of hospital stay



Length of hospital stays (days)

In this study length of hospital stay is 25.4 days with a standard deviation of 6.9 in primary closure group and 17.05 days with a standard deviation of 5.5 in primary closure with omental patch.

DISCUSSION

ETIOLOGY-

Most common cause of ileal perforation is non specific perforation, seen in 50% of patients in the study group. When the etiology of the perforation was not identified it was termed non-specific perforation. Widal test, blood culture were not suggestive of typhoid. Non-specific perforations were the commonest cause of small bowel perforation in the series by Dixon and Bhalerao.[47,91]

Second most common cause is typhoid perforation seen in 45% of patients. Typhoid fever was the commonest cause of ileal perforation in tropical countries. It accounted for 56.6% of cases of ileal perforation in the series by Karmakar [26]

Two cases of ileal perforation were due to trauma in our study accounting for 5% of cases. The rising rate of road traffic accidents and civil violence has contributed to this increased incidence of traumatic perforations.

AGE & SEX

In our study majority of the cases were seen in 2nd & 3rd decade of life. Ileal perforations is common in 2nd & 3rd decade of life as reported by Eggleston.[23]

Males are predominantly affected with 85% of incidence. Sex ratio in the study is M:F=5.6:1

TABLE NO. 14: Comparison of Sex ratios

STUDY	**SEX RATIO**
Chatterjee et al.	2.3 : 1
Swadia et al.	6.1 : 1
Present study	**5.6 : 1**

CLINICAL PRESENTATION

Most patients presented with features suggestive of peritonitis. Patients of both typhoid and non-specific perforations had similar presentation with respect to abdominal signs and symptoms. Examination revealed tenderness, rigidity, distension.

MANAGEMENT

Standard treatment for ileal perforation is surgical management. The various methods in use are flank drains, simple closure, closure with omental patch, wedge resection, resection and anastomosis, ileotransverse anastomosis and ileostomy.[8,44,46,51,52,53]

In present study outcomes following primary closure and primary closure with omental patch were evaluated and compared. Outcomes were compared with respect to wound infection, early post operative obstruction, burst abdomen, intra abdominal abscess, development of faecal fistula, septicaemia, & mortality.

TABLE NO. 15: Comparision of postop complications

POST OP COMPLICATIONS	**PRIMARY GROUP**		**PATCH GROUP**	
	Husain M et.al	**Present study**	**Husain M et.al**	**Present study**
Wound infection	41.1%	90%	36.6%	65%
Early post operative obstruction	0%	0%	0%	0%
Burst abdomen	7.7%	20%	6.6%	0%
Intra abdominal abscess	10%	5%	5.5%	0%
Faecal fistula	7.7%	25%	1.1%	0%
Septicaemia	10%	15%	3.3%	0%
Mortality	3.3%	10%	1.1%	0%

Results are similar in comparison with Husain M et.al.

RELATION TO SITE OF PERFORATION WITH CLOSURE TECHNIQUE AND COMPLICATIONS

Site of perforation

</=10cm from ICJ:-

Primary closure done in 1 patient, had faecal fistula

Primary closure with omental patch done in 3 patients, no major complication

11 – 20 cm from ICJ:-

Primary closure done in 12 patients had complications

Intra abdominal abscess-1

Burst abdomen – 3

Faecal fistula – 3

Septicaemia – 3

Mortality – 2

Primary closure with omental patch done in 10 patients, no major complication

>20cm from ICJ – primary closure done in 7 patients with following complications

Burst abdomen-1

Faecal fistula -1

Primary closure with omental patch done in 7 patients , no major complication .

In view of above results irrespective of the position of perforation, primary closure with omental patch has better outcomes compared to primary closure alone .

LENGTH OF HOSPITAL STAY

It is significantly lower in primary closure with omental patch group($p<0.001$) because of lesser postoperative complications.

SUMMARY AND CONCLUSION

- Most common cause of ileal perforation is non specific perforation followed by typhoid fever.
- Irrespective of causes, ileal perforation is predominantly seen second and third decade of life with preponderance in male sex.
- Klebsiella species is commonest organism seen in peritoneal fluid analysis of typhoid perforation cases
- Primary closure with omental patch repair has better outcomes compared to primary closure alone in terms of wound infection, intra abdominal abscess, burst abdomen, faecal fistula, septicaemia, mortality irrespective of position of perforation from ICJ.
- Primary closure with omental patch group had better and early recovery thus reducing length of hospital stay.

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ANNEXURE

PROFORMA

A COMPARATIVE STUDY IN SURGICAL MANAGEMENT OF ILEAL PERFORATION BY PRIMARY CLOSURE V/S PRIMARY CLOSURE WITH OMENTAL PATCH

SERIAL NO. : **GROUP :**

NAME :

AGE:

SEX:

ADDRESS:

IP/OP NO:

CHIEF COMPLAINTS:

HISTORY OF PRESENT ILLNESS:

FEVER:

Pain Abdomen:

Vomiting:

Trauma

H/o bleeding per rectum:No ☐ yes ☐

PAST HISTORY:

H/O Similar complaints in the past: No ☐ Yes ☐

H/O typhoid, tuberculosis.

H/O surgical procedures

H/O medical illness

FAMILY HISTORY: No ☐ Yes ☐

PERSONAL HISTORY:

H/o smoking: No ☐ Yes ☐

H/o alcohol intake: No ☐ Yes ☐

GENERAL PHYSICAL EXAMINATION:

Built:

Pallor/icterus/cyanosis/clubbing/lymphadenopathy/edema.

Vitals: P.R- B.P-

SYSTEMIC EXAMINATION:

ABDOMINAL EXAMINATION:

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM:

CNS:

Per Rectal examination:

CLINICAL DIAGNOSIS:

INVESTIGATIONS:

X-ray erect abdomen:

Ultra sonography of abdomen & pelvis :

Complete blood counts: Hb: TLC: Differential count: N: L: E: B:

RBS: B.U: S.CREAT:

Serum electrolytes: Na^+: K^+:

Widal test: Day of widal test after onset of fever :

HIV: HbsAg:

INTRA OPERATIVE FINDINGS:

PROCEDURE DONE:

POST OPERATIVE COMPLICATIONS:

Wound infection:

Early post operative obstruction:

Wound dehiscence:

Intra abdominal abscess:

Faecal fistula:

Septicemia:

POST OPERATIVE RECOVERY:

Day of onset of bowel movement:

Day on which liquid diet started:

Day on which semisolid diet started:

OTHER DETAILS:

MASTER CHART

NAME	HOSP. NO	HISTORY					EXAMINATION					INVESTIGATIONS					OPERATIVE DETAILS				POST OP COMPLICATIONS								
		AGE IN YEARS	SEX	FEVER	PAIN ABDOMEN	CONSTIP.	VITALS	TENDERNESS	RIGIDITY	DISTENSION	BOWEL SOUNDS	X ray ERECT ABDOMEN	ASCITIC FLUID C/S	PUS C/S	BLOOD C/S	WIDAL TEST	NO.OF PERF.	SITE FROM ICJ	size of perf.	procedure	WOUND INFECTION	EARLY POST OPERATIVE OBSTRUCTION	BURST ABDOMEN	INTRA ABDOMINAL ABSCESS	FAECAL FISTULA	SEPTICAEMIA	MORTALITY	CAUSE OF MORTALITY	LENGTH OF HOSPITAL STAY
Krishna murthy	758737	26	M	10 d	5 d	nil	st	+	+	+	sl	AU	NG	-	NG	pos	1	20cm	5mm	primary	+	-	-	-	-	-	-		17d
Thimma reddy.k	768583	65	M	nil	1 d	nil	st	+	+	-	-	AU	E.coli	-	NG	neg	1	15cm	10mm	primary	+	-	+	+	-	-	-		33d
chinnapaiah	770979	35	M	10 d	7 d	2 d	st	+	+	-	-	AU	E.coli	-	NG	neg	1	20cm	4mm	primary	+	-	-	-	-	-	-		23d
Rajendra	784957	23	M	nil	1 d	nil	st	+	-	-	sl	-	E.coli	-	NG	neg	1	40cm	5mm	primary	+	-	-	-	-	-	-		22d
Ambrisha	785544	26	M	14 d	1 d	nil	st	+	+	-	+	AU	NG	-	NG	pos	1	15cm	10mm	patch	-	-	-	-	-	-	-		10d
Girija	788419	35	F	nil	1 d	nil	st	+	+	-	sl	AU	Klebsiella	-	NG	neg	1	30cm	5mm	patch	-	-	-	-	-	-	-		15d
Rama chandrappa	789933	45	M	nil	1 d	nil	st	+	+	-	sl	AU	NG	-	NG	neg	1	40cm	5mm	patch	-	-	-	-	-	-	-		10d
Ramesh	791154	28	M	3 d	1 d	nil	st	+	+	+	-	AU	NG	-	NG	neg	1	50cm	5mm	patch	+	-	-	-	-	-	-		20d
Narayana swamy	792149	46	M	14 d	3 d	nil	st	+	-	-	sl	AU	E.coli	E.coli	NG	neg	1	5cm	4mm	patch	+	-	-	-	-	-	-		24d
Manjunatha.G	795413	20	M	10 d	4 d	nil	st	+	+	-	-	AU	Klebsiella	Klebsiella	NG	pos	1	50cm	10mm	primary	+	-	-	-	-	-	-		28d
Govindappa	797123	56	M	trauma +	2 d	nil	SH	+	+	-	-	AU	Pseudomo	-	NG	-	1	45cm	5mm	patch	+	-	-	-	-	-	-		24d
Harisha	739294	23	M	nil	1 d	nil	st	+	-	-	sl	AU	E.coli	MRSA,Kleb	NG	neg	1	20cm	5mm	primary	+	-	-	-	+	-	-		36d
Yarab pasha	825890	22	M	7 d	1 d	nil	st	+	+	-	sl	AU	NG	-	NG	pos	1	15cm	5mm	patch	+	-	-	-	-	-	-		16d
Nagaraj	836287	28	M	1 d	2 d	nil	st	+	+	-	-	-	E.coli	E.coli	NG	neg	1	20cm	5mm	primary	+	-	-	-	-	-	-		15d
Anjineya reddy	838486	24	M	nil	1 d	nil	st	+	+	+	-	AU	NG	-	NG	neg	1	25cm	5mm	primary	+	-	-	-	-	-	-		11d
Nagappa	872835	60	M	nil	2 d	3 d	st	+	+	-	sl	AU	NG	Klebsiella	NG	neg	1	25cm	5mm	patch	+	-	-	-	-	-	-		24d
Jagadish	886693	20	M	4 d	1 d	nil	st	+	+	-	sl	AU	NG	-	NG	pos	1	15cm	7mm	patch	-	-	-	-	-	-	-		10d
Govindappa	887024	30	M	nil	2 d	nil	st	+	+	-	-	AU	Klebsiella	Klebsiella	NG	neg	1	25cm	10mm	primary	+	-	-	-	-	-	-		25d
Suresh.L	912676	25	M	10 d	1 d	nil	st	+	+	-	+	-	Candida	-	NG	pos	1	20cm	5mm	primary	+	-	-	-	-	-	-		34d
Anjum	913911	14	F	10 d	3 d	2 d	st	+	-	+	-	-	E.coli	E.coli	NG	pos	1	10cm	10mm	patch	+	-	-	-	-	-	-		30d
Soma sekhar	922159	23	M	15 d	2 d	nil	st	+	+	-	-	-	Klebsiella	Klebsiella	NG	pos	1	15cm	5mm	primary	+	-	+	-	-	-	-		28d
Akram	946304	18	M	5 d	3 d	3 d	st	+	+	+	sl	AU	E.coli	E.coli	NG	pos	1	15cm	10mm	primary	+	-	+	-	+	+	-		32d
Venkateshappa	948845	25	M	7 d	2 d	nil	st	+	+	-	sl	AU	NG	-	NG	pos	1	20cm	15mm	primary	-	-	-	-	-	+	+	ARDS	
Basavaraj	814315	21	M	10 d	3 d	1 d	st	+	+	+	-	-	E.coli	E.coli	NG	neg	1	15cm	10mm	patch	+	-	-	-	-	-	-		10d
Naveen	807015	12	M	6 d	3 d	nil	st	+	+	+	-	AU	Klebsiella	Klebsiella	NG	pos	1	30cm	5mm	primary	+	-	+	-	+	-	-		32d
Changamma	820041	60	F	trauma +	3 d	nil	st	+	+	-	+	-	E.coli	E.coli	NG	-	1	60cm	10mm	primary	+	-	-	-	-	-	-		26d
Venkata ramana	773882	15	M	7 d	4 d	nil	st	+	-	+	-	-	NG	-	NG	pos	1	30cm	5mm	primary	-	-	-	-	-	-	-		30d
Lakshmamma	865317	45	F	-	2 d	1 d	st	+	+	-	sl	AU	E.coli	E.coli	NG	neg	1	20cm	5mm	patch	+	-	-	-	-	-	-		20d
Narayana swamy	886777	50	M	-	1 d	nil	st	+	-	-	+	AU	NG	-	NG	neg	1	15cm	5mm	patch	+	-	-	-	-	-	-		18d
Anjanappa.N.K	861374	55	M	7d	2 d	1 d	st	+	+	-	-	AU	Klebsiella	Klebsiella	NG	pos	1	10cm	5mm	primary	+	-	-	-	+	-	-		25d
Ramappa.R	812302	58	M	14d	1 d	nil	st	+	-	-	sl	AU	Klebsiella	-	NG	pos	1	25cm	5mm	patch	-	-	-	-	-	-	-		17d
Shilpa	829136	10	F	3d	3 d	nil	st	+	+	-	sl	-	E.coli	-	NG	neg	1	15cm	10mm	patch	+	-	-	-	-	-	-		13d
Krishna murthy	791162	40	M	-	2 d	nil	st	+	+	-	sl	AU	Pseudomo	Pseudomo	NG	neg	1	15cm	5mm	patch	+	-	-	-	-	-	-		20d
Byramma	766467	35	F	10d	2 d	1 d	st	+	+	-	-	AU	E.coli	-	NG	pos	1	20cm	8mm	patch	-	-	-	-	-	-	-		14d
Ramakrishnappa	769517	56	M	7d	3 d	nil	st	+	+	-	+	-	NG	-	NG	pos	1	10cm	5mm	patch	-	-	-	-	-	-	-		15d
Ambrisha	868962	27	M	12d	2 d	1 d	st	+	+	-	-	AU	Klebsiella	-	NG	pos	1	20cm	10mm	primary	+	-	-	-	+	+	+	septic shock	
Sriramappa	856030	45	M	-	2 d	nil	st	+	+	-	sl	AU	E.coli	E.coli	NG	neg	1	15cm	7mm	patch	+	-	-	-	-	-	-		15d
Manjunath	837452	25	M	-	3 d	nil	st	+	+	+	sl	AU	E.coli	-	NG	neg	1	15cm	5mm	primary	+	-	-	-	-	-	-		20d
Muniyappa	834852	45	M	-	2 d	nil	st	+	+	-	sl	AU	NG	Klebsiella	NG	neg	1	25cm	5mm	patch	+	-	-	-	-	-	-		16d
Ramakrishna reddy	799262	55	M	11d	2 d	nil	st	+	+	-	sl	-	E.coli	E.coli	NG	pos	1	20cm	10mm	primary	+	-	-	-	-	-	-		21d