A DISSERTATION ON

"A STUDY ON NON TRAUMATIC ILEAL PERFORATION"

Dissertation submitted to

THE TAMIL NADU Dr.M.G.R.MEDICAL UNIVERISTY

CHENNAI

with partial fulfilment of the regulations

for the Award of the degree

M.S. (General Surgery)

Branch – I

INSTITUTE OF GENERAL SURGERY,

MADRAS MEDICAL COLLEGE ,

CHENNAI.

APRIL-2015

CERTIFICATE

This is to certify that the dissertation entitled "**A STUDY ON NON TRAUMATIC ILEAL PERFORATION"** is a bonafide original work of **DR KONDBA SHAMRAO MAGHADE**., in partial fulfilment of the requirements for M.S.Branch– I (General Surgery) Examination of the Tamil Nadu Dr. M.G.R. Medical University to be held in APRIL 2015 under my guidance and supervision in 2013-14.

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DECLARATION

I hereby solemnly declare that the dissertation titled "**A STUDY ON NON TRAUMATIC ILEAL PERFORATION"** is done by Me at Madras Medical College & Rajiv Gandhi Govt. General Hospital, Chennai during 2013-14 under the guidance and supervision of Prof.Dr.A.RAJENDRAN, M.S, The dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai towards the partial fulfillment of requirements for the award of M.S. Degree (Branch-I) in General Surgery.

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ACKNOWLEDGEMENT

I express my heartful gratitude to the Dean, **Dr.VIMALA** M.D., Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai-3 for permitting me to do this study.

I am deeply indebted to **Prof.Dr.P.RAGUMANI**,M.S., Director& Professor, Institute of General Surgery, Madras MedicalCollege & Rajiv Gandhi Government General Hospital for his support and guidance.

I am very grateful to **Prof.Dr.A.RAJENDRAN** M.S, Professor of Surgery, Institute of General Surgery, Dr.A.SAGAYA INBA SEKAR M.S, Dr.P.RAMADOSS M.S, Madras Medical College & Rajiv Gandhi Government General Hospital who guided and trimmed my work throughout the period of my study.

I am extremely thankful to all the Members of the INSTITUTIONAL ETHICAL COMMITTEE for giving approval for my study. I also thank all the patients who were part of the study and my Professional colleagues for their support and criticisms.

LIST OF ABBREVIATIONS USED

AIDS	Acquired Immunodeficiency Syndrome
Alb	albumin
ANOVA	analysis of variance
BP	blood pressure
c/s	culture/Sensitivity
CSF	cerebrospinal fluid
dL	decilitre
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
GB	gall bladder
GI	gastrointestinal
Hb	haemoglobin
Hg	mercury
Kg	kilograms
L	litres
mg	milligrams
min	minutes
Misc	miscellaneous
ml	millilitre

mm	millimeter
mmol	millimole
perf	perforation
PR	pulse rate
RR	respiratory rate
SBP	systolic blood pressure
Spp	species
Temp	temperature
TB	tuberculosis

ABSTRACT

Background & Objectives: Ileal perforation is a common problem seen in tropical countries, the commonest cause being typhoid fever. Over the years a definite changing trend has been observed in ileal perforation both in terms of causes, treatment and prognosis.

Aims & Objectives:

- To study the clinical presentation of suspected ileal perforation due to non traumatic cause
- To study different methods of surgical management of non traumatic ileal perforation
- To study the prognosis and outcome in non traumatic ileal perforation.

Methods:

The study was conducted in Institute of General Surgery, Madras Medical College, Chennai from Sep 2013 - Sep 2014. A minimum of 50 patients of ileal perforations included in the study. Patients with traumatic perforations and those who came with a delayed presentation with shock and septicemia whose general condition didn't warrant any operative management have been excluded. Factors were tabulated and statistically analysed to study their contributions.

Results:

In our study the commonest cause of ileal perforation was typhoid followed by non specific causes. Perforation commonly occurred in the third and fourth decade of life with 50% of patients between the ages of 30 and 50. Pneumoperitoneum in chest x-ray and erect abdominal x-ray was seen in 80% of patients. In our study lag period was around 24 to 150 hours with an average of 55 hours. Over 96% of perforations were within 1 feet (30 cms) from the ileocaecal junction. Simple 2-layer closure was the commonest procedure done (50%).

Conclusion: - Typhoid is the most common cause of ileal perforation, followed by non-specific perforation. Other Causes of ileal perforation include non-specific, TB, and meckel's perforation. Widal test is useful in the diagnosis of typhoid fever. Morbidity was significantly influenced by age greater than 50, hypoalbuminemia, azotemia, HB<8, shock and a diagnosis of typhoid as the cause of perforation. Mortality was significantly influenced by age greater than 50, hypoalbuminamia, typhoid and shock on admission. The type of surgical procedure did not influence outcome, either morbidity or mortality

Keywords: Typhoid, Ileal Perforation, Prognosis.

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CHAPTER 1

INTRODUCTION

INTRODUCTION

Ileal perforation is a common problem seen in tropical countries. The commonest cause being typhoid fever. In western countries the causes are malignancy, trauma and mechanical etiology, in the order of frequency [1,2,3].

Over the years a definite changing trend has been observed in ileal perforations both in terms of causes, treatment and prognosis.

Despite the availability of modern diagnostic facilities and advances in treatment regimens, this condition is still associated with a high mortality and unavoidable morbidity.

In the presence of advanced anaesthesia of today and tremendous improvement in resuscitative measures, every patient diagnosed to have ileal perforation is universally recommended to be treated surgically. The purpose of operative protocol is to correct the pathology while avoiding any serious accidents and to adopt a surgical procedure which is associated with minimal complications[4].

This study has been undertaken in order to contribute to the improvement in the knowledge of this disease. This study aims to study clinical features, management, complications and prognostic factors affecting the outcome in ileal perforations[4].

CHAPTER 2

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

The aims and objectives of this study are

- To study the clinical presentation of suspected ileal perforation due to non traumatic cause

- To study different methods of surgical management of non traumatic ileal perforation

- To study the prognosis and outcome in non traumatic ileal perforation.

CHAPTER 3

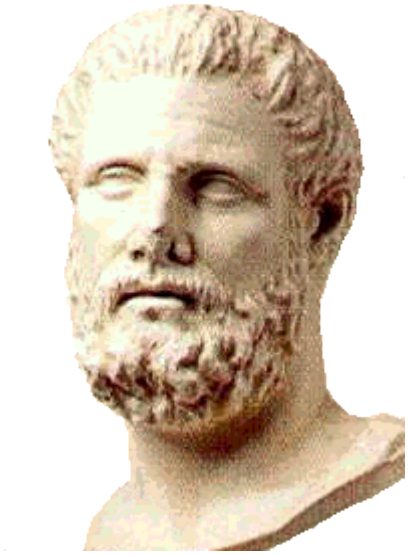
REVIEW OF LITERATURE

REVIEW OF LITERATURE

History

In the Sushruta Samhita, intestinal perforation by sharp objects such as a fish bone or a thorn has been described. It was also recorded that the abdomen was opened and the bowels drawn out. If the intestine was severed or perforated the edges were held together and large black ants allowed to clamp the cut ends with their jaws, prior to their bodies being clipped off. The gut was reintroduced into the abdominal cavity and the incision closed. The text also states that is the intestines were dirty, they were to be rinsed, and then washed with milk and clarified butter . William Cullen coined the term 'peritonitis' in 1776. Benjamin Travers did the first successful closure of an intestinal perforation. The introduction of Lempert's sutures was a significant advancement in the technique of restoring intestinal continuity

Hippocrates first used the term typhus (gr. cloudy) in 460 B.C [(6)]. In 1829 Louis used the term typhoidae and described 150 cases with intestinal perforation, haemorrhage, spleenomegaly, rose spots and mesenteric lymphadenopathy [(7)].



Karl Joseph Ebereth discovered the Typhoid bacillus in 1880. In 1884, Gaffkey first isolated and cultured *Salmonella typhi* [7].Widal described the test to detect agglutinins in the serum of patients suffering from typhoid fever in 1896. The first vaccine for human use against typhoid was made by Pfeiffer and Kalle in 1896 [7].

Anatomy :

Macroscopic anatomy

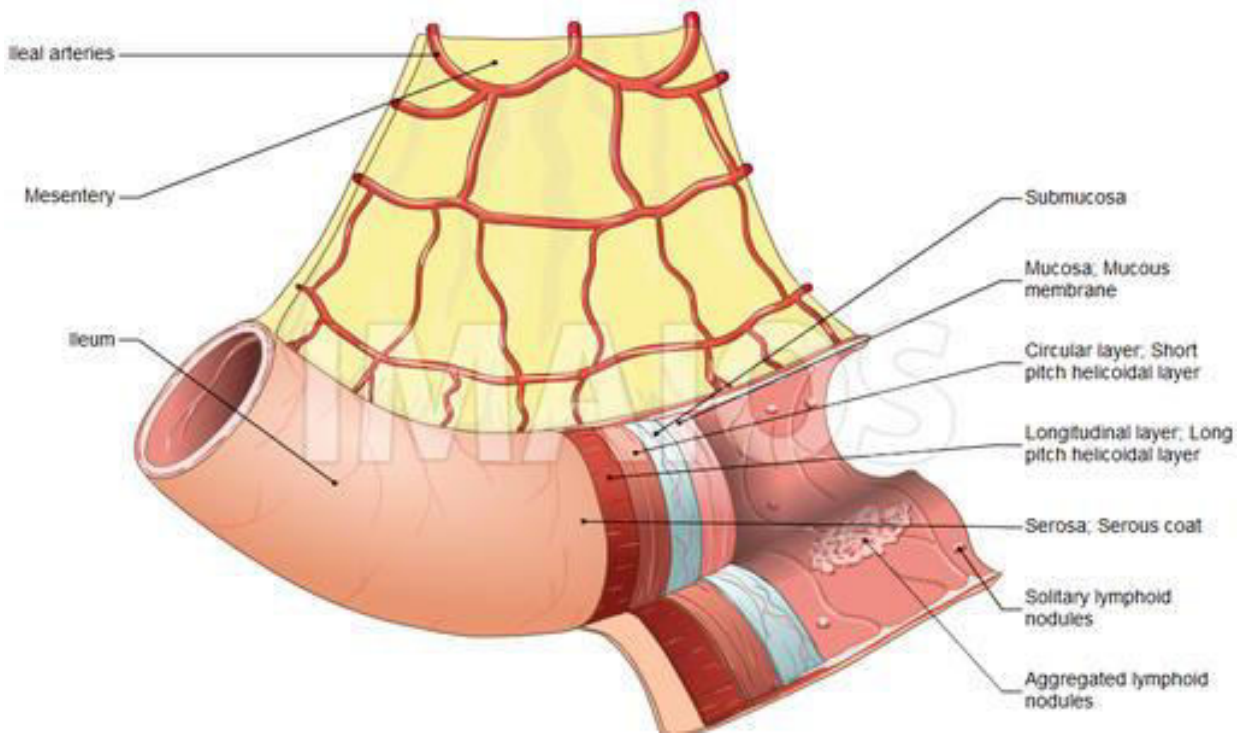
The ileum is the last of the three parts of the small intestine. The transition from the jejunum to the ileum is not sharply marked. At the distal end the ileum opens into the large intestine. At the junction between the ileum and the cecum lies the ileocecal valve (ileal ostium), a functional sphincter formed by the circular muscle layers of both the ileum and cecum. It prevents a reflux of the bacteria-rich content from the large intestine into the small intestine.The ileum makes up about 3/5 of the total length of the small intestine (2.5 to 3.5 meters). Compared to the jejunum the parallel running circular folds in the mucosa (valves of Kerckring) are less prominent. In contrast it is rich in lymphoid follicles. Similar to the jejunum the ileum is attached to the posterior wall of the abdomen by the mesentery and therefore lies flexibly in the abdominal cavity.

About 12 ileal arteries (branches of the superior mesenteric artery) supply the ileum with arterial blood. These form arcades with the other arteries of the small intestine. The venous blood flows from the correspondent veins into the inferior mesenteric vein. Analogous to the jejunum both the coeliac plexus and the superior mesenteric plexus innervate the ileum sympathetically, the vagus nerve (cranial nerve X) parasympathetically.

Microscopic anatomy :

Histologically the ileum has the same basic structure as the jejunum: mucosa, submucosa, muscularis and serosa. The mucosa is lined by simple columnar epithelium (lamina epithelialis) comprising enterocytes and goblet cells. Underneath lies a connective tissue layer (lamina propria) and a muscle layer (lamina muscularis mucosae). Compared to the rest of the small intestine the circular folds are rather flat and the villi relatively short. The submucosa contains blood vessels, lymph nodes and the Meissner's plexus. The muscularis consists of an inner circular and outer longitudinal muscle layer. The ileum is entirely covered by serosa from the outside. It is made up of simple squamous epithelium and a connective tissue layer underneath (lamina propria serosae).

A characteristic feature of the ileum is the Peyer's patches lying in the mucosa. It is an important part of the GALT (gut-associated lymphoid tissue). One patch is around 2 to 5 centimeters long and consists of about 300 aggregated lymphoid follicles and the parafollicular lymphoid tissue. The dome-like bulge above one follicle is called dome area. M cells (microfold cells) are found in the dome epithelium which are counted among the FAE cells (follicle-associated epithelial cells). Their function is to pick up antigens from the intestinal lumen and transport them to the antigen-presenting cells (APC).



Function

The main tasks of the ileum are:

- enzymatic cleavage of nutrients
- absorption of vitamin B_{12} (with intrinsic factor from the stomach), fats (especially fatty acids and glycerol) and bile salts
- immunological function (access and transfer of antigens)

Etiology

Ileal perforation is a serious complication of a variety 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 due to these diseases are seldom encountered in these countries. So much so that the incidence is reported to be one case of perforation per hospital a year. On the other hand in the underdeveloped tropical countries small bowel perforation is quite a commonly encountered surgical emergency [8,9]. Although TB is an important cause, the most important one is the endemic prevalence of typhoid fever in these countries [10].

Wani et al published the causes of ileal perforation as follows [41],

Review Table 1

Etiology	No. of patients (%)
Typhoid	49 (62)
Non-specific	21 (26)
Obstruction	05 (6)
TB	03 (4)
Radiation enteritis	01 (1)

In a series published by Karmakar et al, the causes of ileal perforation were as follows (1).

Review Table 2

Causes	Number
Typhoid	17
Tuberculosis	1
Round Worm	2
Meckel's	1
Blunt Trauma	1
Penetrating Injury	1
Non Specific	7
Total	30

Typhoid fever was the most common cause of ileal perforation in Karmarkar's series, followed by nonspecific perforation [1]. In other studies published by Bhalerao in India is as follows (11)

Review Table 3

Causes	Number
Non Specific	18
Typhoid	8
Tuberculosis	3
Trauma	3
Diverticulitis	2
Total	**34**

In the series by Bhalerao and Karmakar, non-specific and typhoid fever were the commonest Causes [11]. The commonest cause of ileal perforation in the tropics is typhoid fever [1,6,7].

Typhoid Perforation

Typhoid fever, also known as enteric fever, is a potentially fatal multisystemic illness caused primarily by *Salmonella enterica,* Subspecies e*nterica* serovar *typhi* and, to a lesser extent, related serovars *paratyphi* A, B, and C.

The protean manifestations of typhoid fever make this disease a true diagnostic challenge. The classic presentation includes fever, malaise, diffuse abdominal pain, and constipation. Untreated, typhoid fever is a grueling illness that may progress todelirium, obtundation, intestinal hemorrhage, bowel perforation, and death within 1 month of onset. Survivors may be left with long-term or permanent neuropsychiatric complications.

S typhi has been a major human pathogen for thousands of years, thriving in conditions of poor sanitation, crowding, and social chaos. It may have responsible for the Great Plague of Athens at the end of the Pelopennesian War.[1] The name *S typhi* is derived from the ancient Greek *typhos,* an ethereal smoke or cloud that was believed to cause disease and madness. In the advanced stages of typhoid fever, the patient's level of consciousness is truly clouded. Although antibiotics have markedly reduced the frequency of typhoid

fever in the developed world, it remains endemic in developing countries.

The following are modes of transmission of typhoidal salmonella:

- Oral transmission via food or beverages handled by an often asymptomatic individual—a carrier—who chronically sheds the bacteria through stool or, less commonly, urine
- Hand-to-mouth transmission after using a contaminated toilet and neglecting hand hygiene
- Oral transmission via sewage-contaminated water or shellfish (especially in the developing world)

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 [12-16].

Incidence :

The reported rate of bowel perforation in typhoid fever varies from 0.5% to 78.6% [17,18,19,20,21,22]. Various studies have shown the following incidences

Review Table 4

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%
Hisconmez	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 [23,24,25].

Seasonal Variation

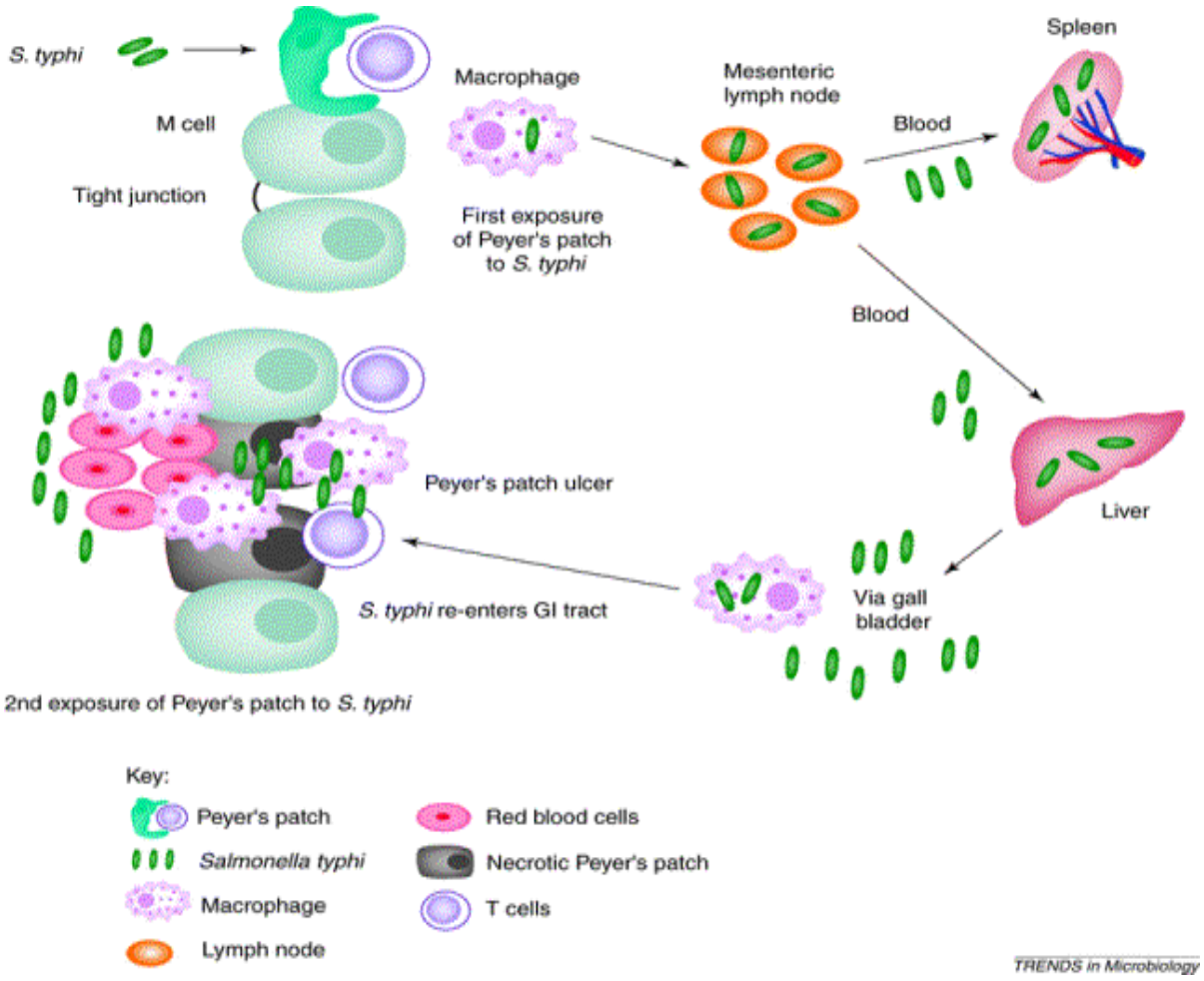
Eggelston reported that over half the cases occurred between July and October [25]. Hadley reported that 58% of cases occurred in the dry season [19]. Tarpley reported that 27% of patients were admitted during the driest quarter of the year and 21% during the rainiest quarter.

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 [20,27,28]. Perforation is reported to occur commonly in the second week following onset of illness[19,20,29,30]. Keenan reported that 88% of patients perforated in the second week [19]. Santillana reported a patient who perforated within 24 hours of onset of clinical illness [20]. The timing of perforation in a series of 59 children reported by Lizzaralde is as follows [30].

Review Table 5

Timing	No.	%
First week	8	13.5%
Second week	32	54.2%
Third week	13	22%
Fourth week	6	10%



Macroscopy

Peyer's patches are swollen and raised. Mesenteric nodes are enlarged. 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 [19]. Tarpley noted that the size of the perforation varied between 1mm and 6cm in size with most being less than 8mm in size(26).

Multiple perforations are seen in 20% of patients. Mock et al reported the following in their series [31].

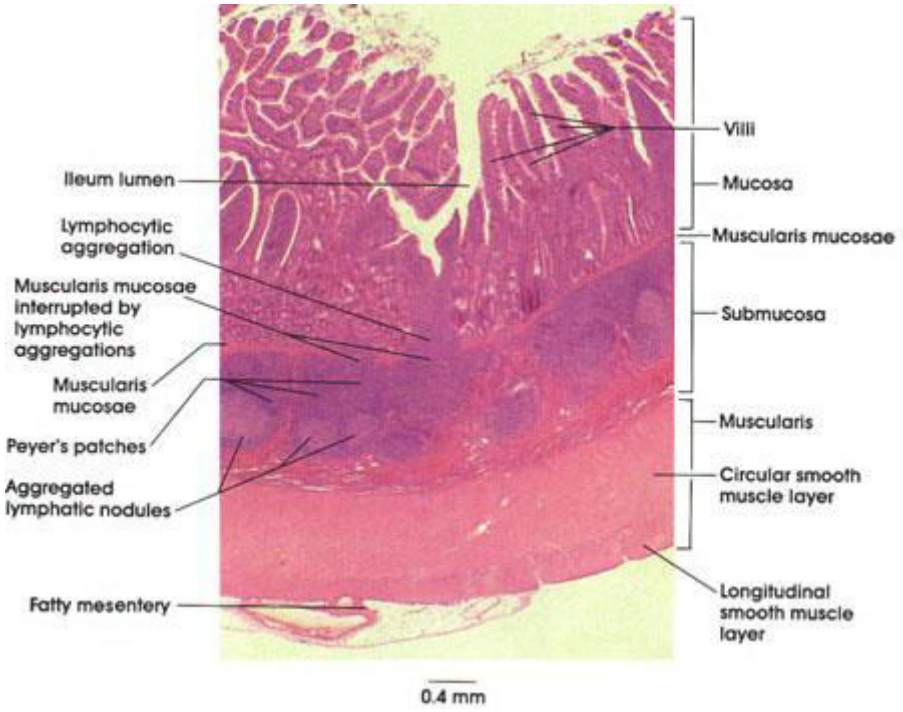
Review Table 6

Single Perforation	78.5%
Two Perforations	13.3%
Three Perforations	4.1%
Four Perforations	4.1%

Most of the perforations occur within 30cm of ileocaecal junction

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 [32].



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 [26].

Review Table 7

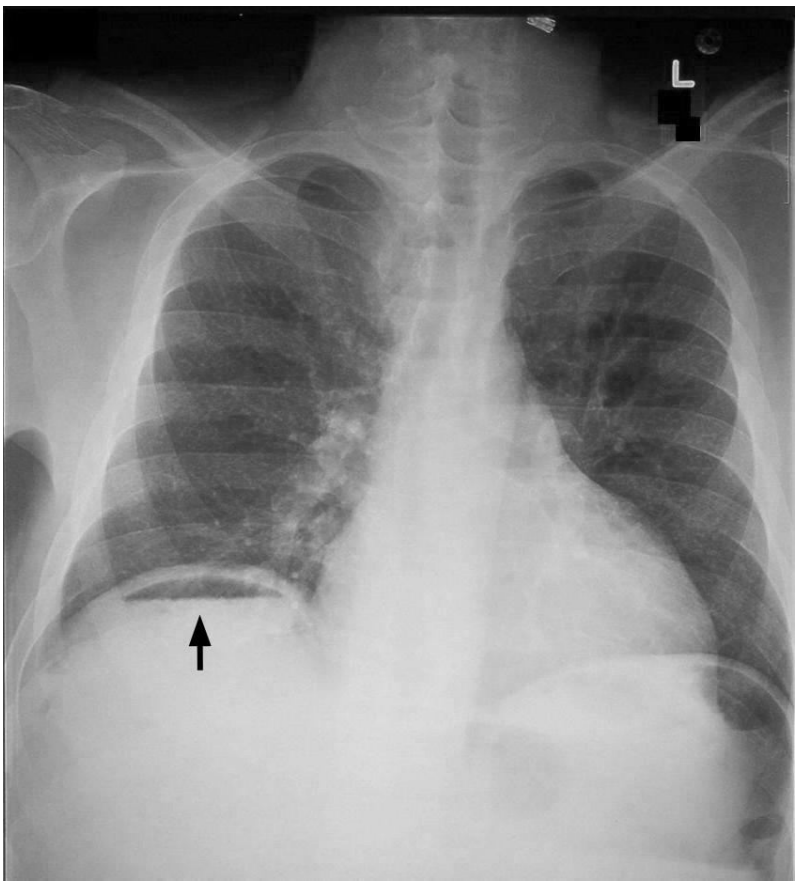
Symptoms		Signs	
Fever	93%	Abdominal Distention	73%
Abdominal Pain	90%	Rectal Tenderness	24%
Vomiting	67%		
Diarrhoea	27%		
Constipation	24%		

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 [25].

Diagnosis

Clinical suspicion is often sufficient for diagnosis in endemic area [19,20,21,33]. Free gas may be present under the diaphragm. Pneumoperitoneum has been reported in 52% to 82% of patients [19,21,34]. Abdominal paracentesis may reveal pus. Bhalerao et al reported positive suprapubic aspiration in all 32 patients with small bowel perforation. Peritoneal lavage might be useful to detect bile or pus [11].

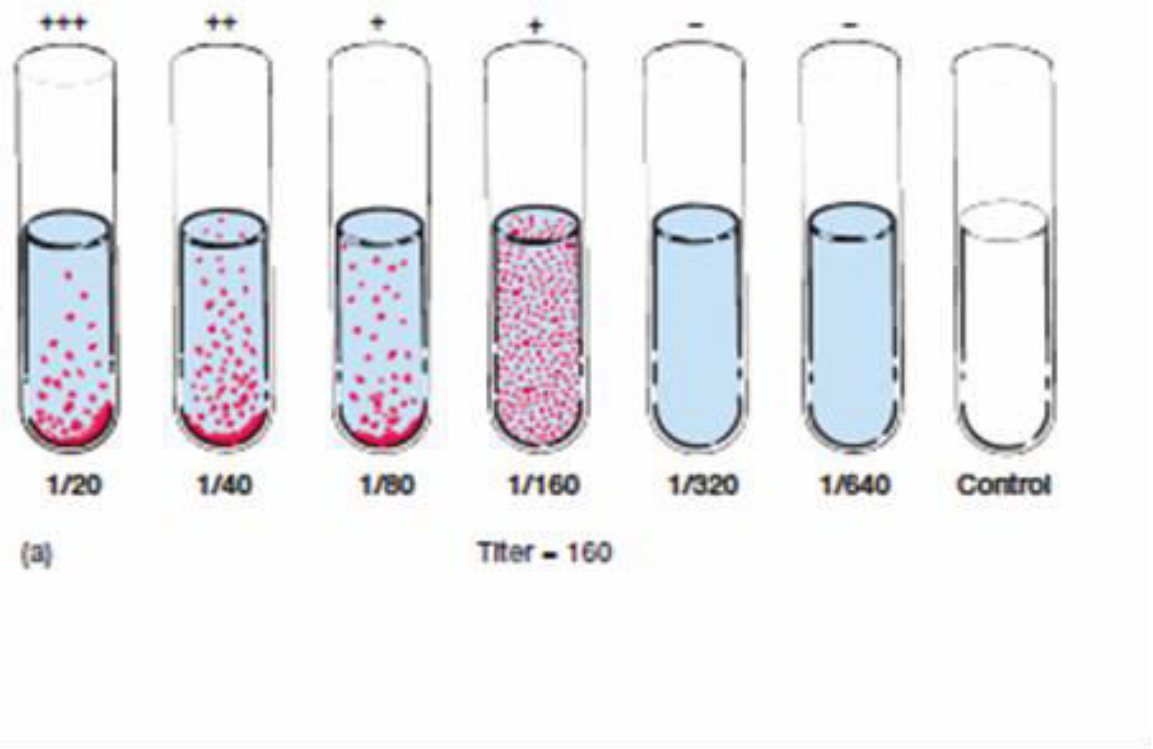
The diagnosis of typhoid fever can be made by Widal test, culture of organism from blood, bone marrow, urine and stools. Newer diagnostic techniques have been introduced to enable rapid diagnosis of typhoid fever. Histopathology of the specimen might reveal etiology of perforation.



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 [35]. 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 [35]. The use of monoclonal antibodies against Salmonella typhi flagellin and DNA probes for detection of Salmonella typhi in blood are promising developments. The newer techniques would enable rapid detection of antibodies or organism [35].

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:

- The terminal ileum is friable and is liable to perforate at more than one spot.
- The friable gut might not hold sutures.
- Chloramphenicol therapy sterilizes bowel contents and adjacent loops might localize the perforation [36].

Hook and Guerrant recommended surgery if there was no localization [37]. Conservative management is associated with a substantial mortality. Presently all authors recommend surgical management [22].

Wani et al described the perforation operation delay in her study [41].

Review Table 8

Perforation-operation delay (in hours)	No. of patients (%)
Within 24 hours	23 (29)
24-48 hours	27 (34)
48-72 hours	11 (14)
72-96 hours	13 (17)
96-120 hours	02 (03)
120-144 hours	03 (04)

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% [22]. Wani et al described the operation performed in her study [41].

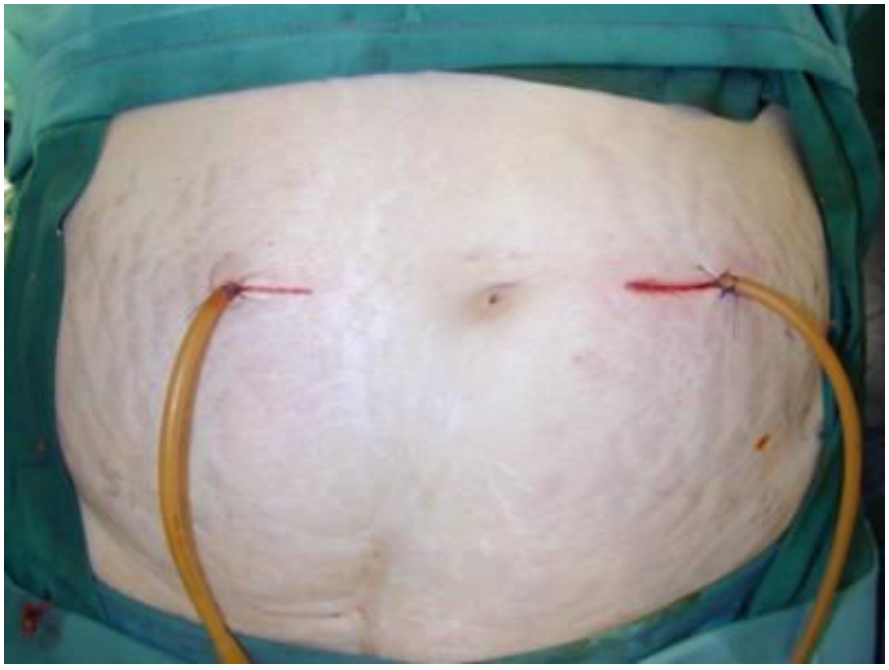
Review Table 9

Operation performed	No. of patients	Death
Simple closure	38 (49)	1
Resection and end to end Ileotransverse	33 (42)	3
Side to side Ileotransverse 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 [42]. 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.



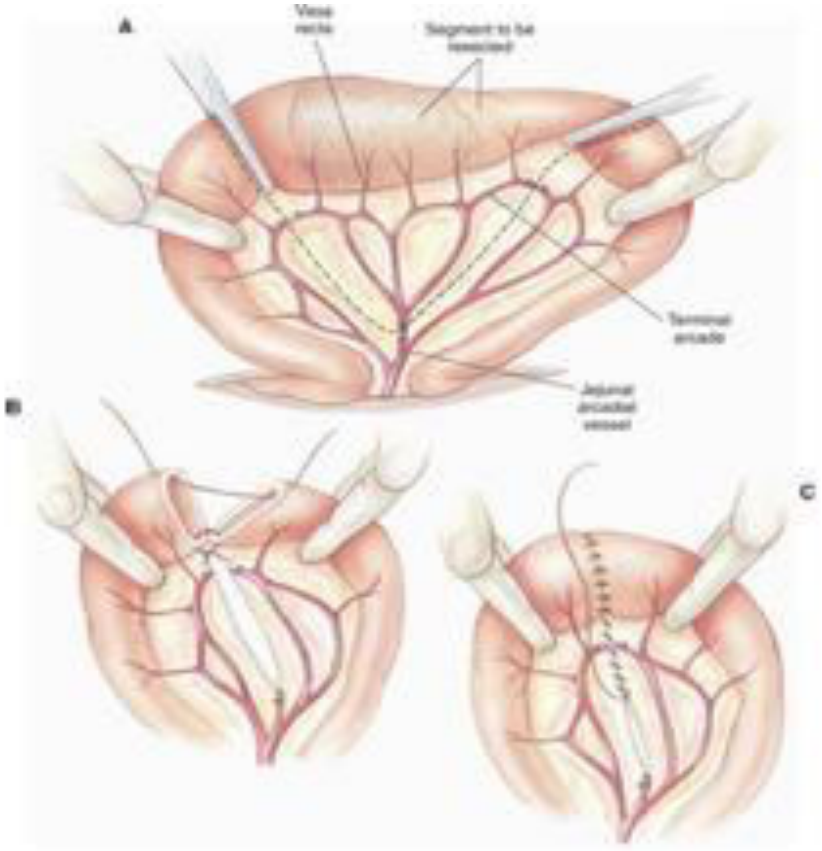
2. SIMPLE CLOSURE

Freshening of the edges and closure has been recommended by Archampong [33]. Primary closure was done in two layers with interrupted sutures. 3-0 vicryl was used for inner layer, while 3-0 silk was used for outer layer.

He reported mortality of 17.3% with this procedure. Talwar et al recommended primary closure and limited surgery [43]. 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 [20,44]. Ameh reported that a wedge resection is associated with a very high mortality rate [44].



4. RESECTION – ANASTOMOSIS

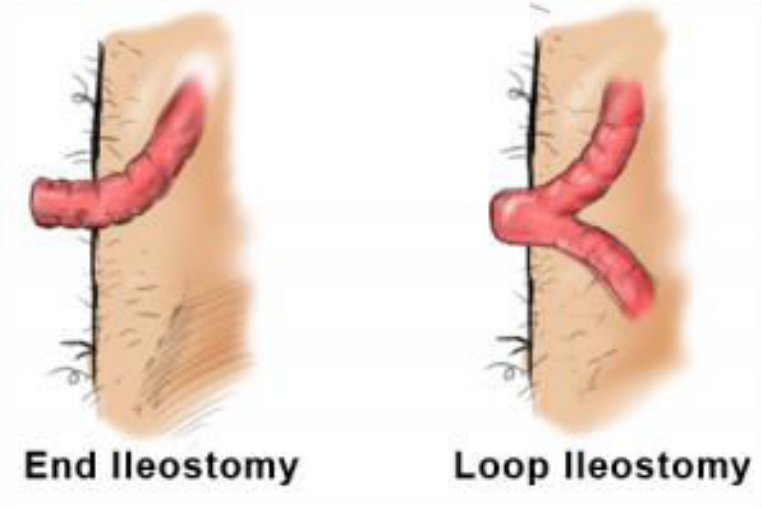
Many workers claimed that segmental resection of the involved bowel may be necessary in the presence of multiple perforations and a severely diseased terminal ileum. The complication and mortality for resection-anastomosis were 37.50% and 21.47% respectively, very much less than that observed in other treatment modality [45]. Jarrett and Gibney recommend resection only for multiple perforations. Gibney recommended resection if there were three or more perforations [28].

5. ILEO-TRANSVERSE COLOSTOMY

Eggleston et al [25] advocated closure of the perforation with end-to-side ileotransverse colostomy; this takes the involved bowel out of the intestinal stream. 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 [46].

6. TUBE ILEOSTOMY

Lozoya [47] introduced tube ileostomy in 1948. 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 ieum (46,48,49). 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 [48] in Vietnam, was reported to have used this method with a very good outcome. Also, Kaul and Ardhanari and Ranqabashyam in India recorded a significant reduction in the mortality rate using tube ileostomy, although Chamber [51] and Lizarralde [46] 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 form they invariably heal on conservative management [7,20]. Good peritoneal lavage and placement of drains to remove pus was recommended. Two-layer closure was recommended to decrease the risk of leakage [29,31] . 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 [52]. 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 [53]. 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 [55,56]. Ciprofloxacin is used in a dose of 200 to 750 mg twice a day. Resistance to this drug is still rare [55]. 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 [55].

Complications

Santillana in his series of 96 patients reported a complication rate of 71.9% (18).

Review Table 10

Author	Year	Complications	%
Santillana	1991	Wound Infection	40.6
		Chest Infection	10.4
		Renal Failure	2.1
		GI Fistula	2.1
		Melena	2.1
		Icterus	2.1
		Septicemia	3.1
		Reperforation	0.7
		Incisional Hernia	0.7
		Pleural Effusion	0.7
		Parotid Abscess	0.7
Keenan and Hadley	1981	Chest Infection	57
		Septicemia	48
		Wound Infection	33
		Reperforation	10
		Recurrent typhoid	5
		GI Fistula	10
		Abdominal Wall Fascitis	5
		Wound dehiscence	5

Mortality

Mortality rates ranging from 20-40% are most commonly reported for typhoid ileal perforation[21,35,57,58]. 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 [20.59.60.61]. 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 [21,62].

The mortality rates are as shown below.

Review Table 11

Author	Year	Country	Percentage
Jarret[42]	1975	Korea	9.9
Eggelston[25]	1979	India	32
Badejo[22]	1980	Nigeria	3
Hadley[19]	1984	South Africa	9
Meier[26]	1989	Nigeria	32
Tacyildiz[34]	1995	Turkey	
Retrospective			25
Prospective			10

The causes of mortality in a series of 68 patients reported by Archampong are follows (21).

Review Table 12

Causes of mortality	Percentage (%)
Toxemia with myocarditis	45.6
Shock and dehydration	23.5
Aspiration Pneumonia	13.2
Bronchopneumonia	7.4
Renal Failure	5.9
Confusional State	4.4

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 [63,64,65,66]. 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 [27,67,68,69]. 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 [21].

Review Table 13

Time Interval	Mortality (%)
< 24 hours	14.1
24-48 hours	22.8
49-72 hours	31.3
>4 days	80

Mock et al reported that increasing number of perforations, generalized contamination of the peritoneal cavity and single layer closure increased the mortality [31]. The relationship between the number of perforations and mortality as reported by Mock is as follows.

Review Table 14

Perforations	Mortality %
1	27
2	31
3	50
>3	80

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 [70]. 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 [43].

Some studies have found mortality to be associated with the type of surgical procedure performed. Ameh reported 50% mortality with simple closure, 62% with wedge resection and 36% with resection and anastomosis [44].

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.

The outcome of surgical intervention; whether death or uncomplicated survival, complications or long term morbidity is not solely dependent on the abilities of the surgeon in isolation. The patient's physiological status, the disease that requires surgical correction, the nature of the operation and the preoperative and post operative support services have a major effect on the ultimate outcome. The systematic approach to quantifying illness in critically ill patients like peritonitis is a recent phenomenon. Early and objective classification of the severity of peritonitis may help in selecting patients for aggressive surgical approach (Bohnen et al., 1983; Giessling et al., 2002; Schein et al., 1983; Farthmann and Schoffel, 1990).

The development of such systems has been specifically the need for methods to compare patient populations and severity of illness, objectively predict morbidity and mortality. Scoring systems like APACHE II, SAPS, MPI have been developed in response to an increasing emphasis on the evaluation and monitoring of health services (Notash et al., 2005; Wisner, 1992). Early evaluation of severity of lesion using Mannheim Peritonitis Index (MPI) allows us to estimate the

possibility of patient survival. The MPI is one of the simplest scoring systems in use that allows the surgeon to easily determine risk during initial surgery. It is a disease specific score based on easy to handle clinical parameters. The recollection of retrospective data is possible and valid, because MPI only requires information routinely found in surgical registers. It takes into account age, gender, organ failure, cancer, and duration of peritonitis, involvement of colon and extent of spread and character of peritoneal fluid. Peritonitis due to perforation of gastro intestinal viscus is the most common surgical emergency in India. Despite advances in surgical techniques, antimicrobial therapy and intensive care support, management of peritonitis continues to be highly demanding, difficult and complex and the spectrum of disease is different from that found in the western world .

THE APACHE SYSTEM

Acute physiological and chronic health evaluation.

The first major attempt at a system to quantity severity of illness in ICU patient was the APACHE system, by Knaus et al in 1981[47].

APACHE I:

In original form, APACHE contained 34 physiological measurement and included many continuous variables. A value of 0-4

was assigned to each variable, according to its degree of abnormality. Shortly after its introduction *APACHE I* system was disfavored, because of practical problem like collection of large number of variable. Also under the rule of APACHE system any unmeasured variable was assumed to be normal and weighted as zero. This gave rise to question about the model general applicability. Another major criticism of original APACHE system was that the variables were chosen by a group of physician and hence there was potential of bias. These inaccuracies in the original APACHE system prevented its widespread use. However, it did serve as a prototype for the development of two subsequent systems.

SAPS:

The simplified acute physiological score was developed from APACHE I system and incorporated 13 variable that had the most discriminate power and were the most frequently measure variables to cover all major organ system. SAPS score is still used but has essentially been replaced by APACHE II in many centers.

APACHE II

Published in 1985 by the same author this is the second version of the APACHE system and it contain refinement based on experience with the original APACHE system. APACHEII has been extensively used and has received for more attention in the literature than any

of the other methodologies for ICU Out comes prediction. It contain 12 continuous variables from the original APACHE system and also takes into account age of the patient, pre- morbid condition and Glasgow coma scale.

DEVELOPMENT OF APACHE II :

Using clinical judgment and documented physiological relationship to choose variables and assign weight remains the essence of APACHE II. The number of variables was reduced from 14 to 12. Infrequently measured variables such as serum osmolality, lactic acid level, and the skin testing for energy were deleted. Serum BUN was replaced by more specific serum creatinine and serum pH was retained in preference to bicarbonate. Many variables crucial in patient care, such as serum glucose, albumin, CVP and urinary output were found to have less explanatory power. Most of these variables were sensitive to variation in therapeutic decision than severity of disease.

Some of the threshold and weight for the physiology variables have been changed e.g. Glasgow coma score, serum creatinine. Also since alveolar- arterial O2 gradient(p [A-a] O2) is heavily dependent on inspired O2 (F1 O2) a direct weighting was given to all paO2 values when FiO2 is less than 0.5

To eliminate the problem of missing values and concerns about the assumption that an unmeasured variables was normal, measurement of

all 12 variables was made mandatory for usage of APACHE II. The recorded value of the variables are based on the most deranged values during the past 24 hours.

Because age and severe chronic health problem reflect diminished physiological reserve, they have been directly incorporated into APACHE II. also, emergency surgery and non operative patient with severe chronic organ system dysfunction were given additional five points in comparisons to elective surgical patient who were given only two points because patient with severe chronic condition are not considered to be candidates for elective surgery.

The maximum possible APACHE II score is 71. in the experience of the author of APACHE II no patient had exceed 55.

The strengths of APACHE II system are

1. It has a well- define outcome (hospital death)

2. It was derived from a database (5815 patient from 13 hospital)

3. Source of bias present in its prototype was understood and corrected.

SHORT COMINGS OF APACHE II SYSTEM

Because of extensive usage, important sources of error and bias in the APACHE II system were revealed. First, APACHE II perform well over all in several ICU population but it is inaccurate when looking at specific disease categories because the data base from which it was derived, though large, did not contain many patient in major disease subsets such as cardiac surgery, oncology etc. second, APACHE II dose not accounts for prior treatment or clinical course before the patient enter ICU, this has been labeled as lead- time bias. Third, APACHE II require determination of a single admission diagnosis, a subjective process prone to bias. Finally despite reduction in number of variables, measurement error from bedside data collection is still on issue.

APACHE II has been recently refined into APACHE O, where O represents Obesity, and this is a better prognosis than APACHE II[48] . another modification of APACHE II is the APACHE III system, which is yet to be applied widely

Adesunkanmi assessed the severity of generalized peritonitis from typhoid ileal perforation using modified APACHE II Score. Modified APACHE II Score ranged from 0-19, with a mean of 8.2+4, 7.6+4 for survivors and 9.4+2 in those who died.

The results are as shown in table [77].

Review Table 15

Modified APACHE II Score	Mortality
0-4	0
5-9	13%
10-14	41.2%
15-19	50%

$P < 0.05$

A high APACHE score was associated with high mortality, but did not predict morbidity rate in patient studied[71].

Manheim peritonitis index

The Manheim peritonitis index is easier to apply for prognostication and is shown in table 15 [51].

It has been found to be most appropriate for Indian settings where access to resources is limited, as in rural areas.

Review Table 16: Mannheim peritonitis index.

Risk factor	Weight age
Age > 50 years	5
Female sex	5
Organ failure*	7
Malignancy	4
Pre-operative duration of peritonitis >24 hours	4
Origin of sepsis not colonic	4
Diffuse generalized peritonitis	6
Exudates	
Clear	0
Cloudy, purulent	6
Faecal	12
Definitions of organ failure	
Kidney	Creatinine level ≥177mmol/l, Urea level ≥167mmol/l, Oligurea < 20ml/h
Lung	PO2 < 50mmHg PCO2 > 50mmHg
Intestinal obstruction	Paralysis ≥ 24 hr. or complete mechanical ileus

Increasing Mannheim Peritonitis Index predicts poor prognostic outcome. Paying close attention in these patients to maximally support vital systems and to prevent complications seems crucial for their eventual prognosis

Trauma :

Trauma is a more common cause of ileal perforation in developed countries. The penetrating injuries are commonly knife stabs or gunshot wounds. Injury to the intra-abdominal structures in blunt injury can be classified into 2 primary mechanisms of injury – compression forces and deceleration forces.[4] Compression or concussive forces may result from direct blows or external compression against a fixed object (e.g. lap belt, spinal column). These forces may deform hollow organs and transiently increase intraluminal pressure, resulting in rupture. Deceleration forces cause stretching and linear shearing between relatively fixed and free objects. As bowel loops travel from their mesenteric attachments, thrombosis and mesenteric tears, with resultant splanchnic vessel injuries can result. Whatever the mechanism, early recognition of these lesions can be difficult. An overlooked bowel injury is very dangerous because of its tremendous infectious potential. Annan in 1837 reported the first case of intestinal rupture secondary to blunt trauma in America.[3] It has been observed in earlier studies that these injuries are seen in the younger age groups and usually occur due to road traffic accidents.[3,5,6] The present study showed similar results. In this study, intestinal injuries occurred in 12.63% patients with blunt abdominal trauma. This figure is consistent with the 5-15% reported in others series, making the intestine the 3rd most commonly injured abdominal organ in blunt trauma.[3,7,8,9,10] Most of the patients in this study presented with

abdominal pain, tenderness and distension. However, the features were vague at initial examinations and became obvious only at repeated abdominal examinations. Delayed presentation or large leakage of bowel contents into the peritoneal cavity results in increased morbidity. This has also been reported in others studies.[6]

As with others studies, the small intestine was also the most commonly injured in the present study.[2,6,11,12] In this study, it was observed that the proximal jejunum and distal ileum were more prone to perforation. This has also been observed in earlier reports.[13,14] But some studies have not supported this view.[3,15] Dauterve et al. in a study of 60 patients, found that less than half of the perforations occurred in these zones.[3] However, according to his study, mesenteric injuries do occur more frequently at these points. Similar results were noted in the present study. Colonic injuries occurred less frequently than small intestinal injuries. This has also been reported in others studies.[2,3,5,6] This is mainly due to its location and the lack of redundancy, which prevents formation of closed loops.

Kaul et al had 10 cases for 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 (1). 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]. Regarding treatment, exploratory laparotomy, drainage of septic peritoneal fluid and wound saline lavage are very important. Prophylactic antibiotics are required.[1] Simple closure is usually adequate for single perforation of the small intestine, but

more extensive injuries such as multiple perforations and gangrene from mesenteric injuries usually require resection and anastomosis. Large injuries may require creation of stoma.

Tuberculosis

Primary intestinal tuberculosis (without pulmonary involvement) is one of the commonest forms of extrapulmonary tuberculosis. The infection is usually caused by ingestion of unpasteurized or contaminated milk that leads to a primary infection of the intestine in the absence of pulmonary disease. Intestinal tuberculosis commonly affects the ileocaecal region because of the following reasons:

1) the terminal ileum is an area of physiological stasis;
2) it has abundant lymphoid tissue; and
3) it has a high absorptive capacity. Thus, after the initial infection occurs in the Peyer's patches, mucosal edema and sloughing occur, leading to the formation of typical tubercular ulcers that lie transversely to the long axis of the ileum. The disease may spread further by dissemination through the lymphatics and by caseation, may heal by fibrosis, or may even remain confined to the area if the host's defence mechanisms are adequate.

   There are the three pathologicl forms of tuberculous enteritis :

- Ulcerative
- Hypertrophic
- Ulcero-hypertrophic

The ulcerative form of the disease is more common than the others, but these ulcers rarely perforate. Fibrosis and the formation of adhesions to adjacent intra-abdominal organs account for the low incidence of perforation seen in tuberculosis. However, if perforation occurs, the patient presents with the signs and symptoms of peritonitis. Although it is well documented that the incidence of perforation in patients with intestinal tuberculosis varies from 1% to 11%, the majority of these perforations (70%–80%) are not truly perforations of such tubercular ulcers, but are 'blow outs' of the small bowel secondary to distension due to distal obstruction (strictures or adhesions). As such, true or 'free' perforations are rare, and only a few cases have been reported to the present in the world literature, with an overall mortality rate of nearly 70%. Recently, vasculitis of the mesenteric vasculature due to tuberculosis has been implicated as a contributory factor, but the exact mechanism by which some patients develop perforation and others is not established.

The treatment of tubercular peritonitis is similar to that for peritonitis due to other causes like resuscitation, nasogastric aspiration, intravenous fluids, antibiotics, and surgery once the patient is stabilized. Tubercular perforation

is rarely diagnosed pre-operatively as the signs and symptoms are similar to those of peritonitis and there are no pathognomic features either on investigation or on clinical examination. Even in patients who are know to be sufferers of the disease, the diagnosis of perforated tubercular ulcers cannot be made with certainty. As this condition is uncommon, it is important to send the margins of any perforation routinely for histopathological analysis, especially in areas where tuberculosis is endemic. We realized that a potentially treatable disease like tuberculosis can be missed by omitting a biopsy, since we consider such perforation is secondary to enteric fever even in our institution. If tuberculosis is suspected intra-operatively, any other suspicious tissues (e.g. lymph nodes, fluid) should also be analyzed, as the combination of histology and culture helps to establish the diagnosis in nearly 80% of the cases. Another important point to keep in mind is the association of tuberculosis with HIV infection, and such patients must always be screened for HIV if the diagnosis of tuberculosis is made.The treatment of the perforation depends upon the condition of the patient and the bowel. Primary closure of the perforation can be considered safe if the patient has presented early and the bowel is healthy, otherwise, exteriorization of the affected bowel as a loop ileostomy is a safer option. If there is a long segment of bowel that is diseased, or there are multiple perforations, resection with either primary anastomosis or exteriorization may be considered. Once biopsy confirms the

diagnosis of tuberculosis of the bowel, anti-tubercular therapy is mandatory.

Free intestinal perforation is an uncommon complication of intestinal tuberculosis because of reactive thickening of the peritoneum and formation of adhesion with surrounding tissues [80]. It account 1-10% of abdominal tuberculosis cases and it has a poor prognosis with mortality rate higher than 30% [81,82].

S. Talwar et al have found 19% of non-traumatic small bowel perforation in 308 patients were due to intestinal tuberculosis [83]. Badoui et al in Switzerland, also reported eleven cases of intestinal tuberculosis perforation, ten of them were immigrants from countries endemic for tuberculosis [84].

Free perforation in intestinal tuberculosis usually occurs in the terminal ileum [85] and it can occur in patient during anti tuberculosis therapy [86]. 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 [87,88,89]. Peritonitis, occurring in a patient with chest radiography indicative of tuberculosis should lead one to suspect a perforated tuberculosis ulcer [86]. 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 [90].

In 90% of the cases, perforation is solitary, but multiple perforations occur in 10-40% of patients[91] 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 [81,83,87]. 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 [83].

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 [27].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[92].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 [2].

Malignancy :

Small intestinal malignancies are very rare accounting for 1-3% of all gastrointestinal malignancies. The reported small bowel tumors in order of frequency are adenocarcinoma, carcinoid, lymphoma and sarcoma. The commonest site is the Ileum. Lymphomas are the commonest small bowel tumors to perforate. Dixon et al in their series of 54 cases had 9 cases of lymphoma and two perforations due to small bowel carcinoma [2].Rajagopalan and Pickleman in their series of 16 patients with free perforation of the small intestine had 2 patients with lymphoma [93]. Lymphomas often involve the bowel wall centrifugally leading to perforation. This may occur in an area of cancerous involvement often secondary to partial or complete distal obstruction [93]. Resection of the segment and the adjacent mesentery is recommended.

Inflammatory Bowel Disease

Crohn's disease is characterized by chronic transmural inflammation of the bowel. The accompanying fibrous reaction and adherence to adjacent organs appears to limit the complication of free perforation. It is generally accepted that 1±3% of patients with CD will present with a free perforation ± initially or eventually in their disease course. Some reports include secondary abscess perforation in their statistics, but this event is not a true free perforation. Free bowel perforation is one of the indications for emergency surgery in Crohn's disease. Massive hemorrhage is rare, abscess formation can be treated non-surgically and is usually not an emergency procedure, bowel obstruction tends to resolve with appropriate medical treatment, and fistula tracts do not require emergency treatment.

Our clinical impression is that free perforation is not as rare as the published estimates of 1±3% and it now presents far more frequently than it did 20 years ago. 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 (2).Chaikof reported 16 cases of Crohn's in the 76 patients of non-traumatic small bowel perforation(92). 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.

Non-specific Perforation

When the etiology of ileal perforation is not identified, it is termed as a non-specific perforation. Dixon et al in their series had such results in 14 out of 54 patients (2). Karmakar et al in their series of 30 patients of ileal perforation had 7 cases of nonspecific perforations (1).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 (95).

Diverticulitis

Meckel's diverticulum is located in the distal ileum, usually within 60–100 cm (2 feet) of the ileocecal valve. This *blind segment* or small pouch is about 3–6 cm long and may have a greater lumen diameter than that of the ileum.[4] It antimesenterically runs and has its own blood supply. It is a remnant of the

connection from the yolk sac to the small intestine present during embryonic development. It is a *true diverticulum*, consisting of all 3 layers of the bowel wall which are mucosa, submucosa and muscularis propria.[5]

As the vitelline duct is made up of pluripotent cell lining, Meckel's diverticulum may harbor abnormal tissues, containing embryonic remnants of other tissue types. Jejunal, duodenal mucosa or Brunner's tissue were each found in 2% of ectopic cases. Heterotopic rests of gastric mucosa and pancreatic tissue are seen in 60% and 6% of cases respectively. Heterotopic means the displacement of an organ from its normal anatomic location.[6] Inflammation of this Meckel's diverticulum may mimic appendicitis. Therefore during appendectomy, ileum should be checked for the presence of Meckel's diverticulum, if it is found to be present it should be removed along with appendix.

A memory aid is the rule of 2s:

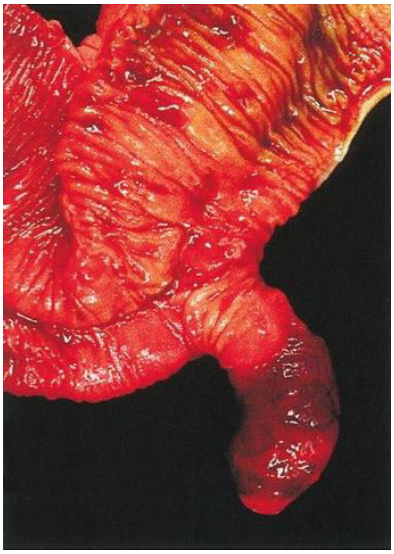
- *2*% (of the population)
- *2* feet (proximal to the ileocecal valve)
- *2* inches (in length)
- *2* types of common ectopic tissue (gastric and pancreatic)
- *2* years is the most common age at clinical presentation
- *2*:1 male:female ratio

However, the exact values for the above criteria range from 0.2–5 (for

example, prevalence is probably 0.2–4%)

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 [96]. Bhalerao et al had two patients with perforated diverticula in their series of 32 patients [11].

Meckel's diverticulum occurs in 0.3% to 2.5% of population. Gastric mucosa is found in up to 38% of Meckel's diverticula [67]. Perforation of an acquired diverticulum is rare. The gastric mucosa in a Meckel's diverticulum may lead to ulceration, which might perforate [96]. Resection of the diverticulum with the adjacent ileum is recommended [92]



Ischemic Enteritis

Ischemic enteritis is a rare cause of ileal perforation. Dixon in his series of 54 cases had 3 due to this cause [2].The gross lesion can be described in four stages

1. Segmental bluish discoloration, edema and mucosal ulceration
2. Circular purple bands with edema of bowel wall
3. Intestinal segment becomes longer, rigid and pipe-like
4. The segment becomes thin and papery

Perforation usually occurs in the fourth stage. Histological picture shows necrosis, the severity of which varies with the stage of the disease [97].

Miscellaneous

The miscellaneous causes reported are roundworm infestations, polyarteritis nodosa, radiation enteritis, steroid dependency, and AIDS [1,2,3].Remine reported 79 patients on steroids at the time of perforation. Patients receiving Prednisolone at a dose greater that 20mg/day had a mortality of 85.1%. Medical problems necessitating steroid therapy were myeloproliferative disorders, connective tissue disorders and metastatic cancer in 62% of patients. The risk of perforation was highest during the first three weeks of starting steroid therapy [98]. Sunke et al reported a patient of AIDS with an

ileal perforation. Cytomegalovirus infection was postulated as the cause in this case[70]. Radiation can lead to perforation due to impairment of blood flow and mucosal inflammation [96].

CHAPTER 4

MATERIALS AND METHODS

MATERIALS AND METHODS

Source of data

Retrospective and descriptive study of patients admitted in Institute of General Surgery, Madras Medical College. A minimum of fifty patients of ileal perforations included in the study.

Inclusion Criteria.

All cases of non traumatic ileal perforations of all age groups

Exclusion Criteria.

- Perforations due to traumatic causes
- Perforations with delayed presentation with shock and septicemia whose general condition didn't warrant any operative management even after resuscitative measures.

Study method

Clinical history, clinical examination, diagnostic and therapeutic biochemical investigations and diagnostic imaging. The data will be entered into a proforma which also includes the therapeutic intervention, course in hospital and follow up.

Clinical history regarding fever, pain, vomiting, abdominal distension,

constipation and treatment prior to admission was taken. Vital signs, hydration, abdominal distension, tenderness, guarding and presence of free fluid were noted. Systemic examination of cardiovascular, respiratory and central nervous system was done.

All patients included in the study underwent investigations in the form of Hb, BT, CT, RBS, blood urea, serum creatinine, CXR, erect X ray abdomen, ECG, blood culture and Widal. Pus culture in case of wound infection.

Cases were resuscitated with IVF and antibiotics. Most cases received ceftriaxone, amikacan and metronidazole antibiotics. In cases of gross contamination inj piperacillin + tazobactum were added. All patients underwent surgery following preoperative preparation.

- Nil by mouth
- Inj TT ½ CC im
- Inj Xylocane test dose
- Preparation of parts by shaving

All patients received one dose of preoperative IV antibiotics – ceftriaxone and metrogyl. All patients underwent laparotomy under GA. Midline laparotomy were employed. The amount and type of peritoneal contamination, number, site and size of perforations and procedure employed were noted. The choice of procedure was based on surgeons preference and condition of the perforation. The following procedures were employed.

- Simple two layered closure
- Resection and anastomosis
- Ileotransverse anastomosis
- Loop or end Ileostomy

For both closure and anastomosis, the inner layer was performed with polygalactin and outer layer with silk.

Antibiotics were routinely given for 5-7 days unless the diagnosis was typhoid in which case antibiotics were continued for up to 10 days. A diagnosis of typhoid was made only if Widal test was positive, or Salmonellae were isolated from blood or urine and if histopathological evidence of typhoid perforation was found. When the etiology of a non-traumatic perforation was not found, it was termed non-specific. Postoperative complications were noted. The factors influencing mortality and morbidity and outcome were assessed. All data will be tabulated, graphical analysis were made and subjected to statistical analysis in the form of ratios, percentages, mean and nonparametric tests like Chi square test.

CHAPTER 5

RESULTS

RESULTS

Fifty patients of Ileal Perforation admitted between September 2013 and September 2014 were included in this study. Patients have been grouped into etiological categories, namely, typhoid, non-specific, tuberculosis and meckels diverticulum.

Etiology

The commonest cause of ileal perforation was typhoid followed by nonspecific, tuberculosis and meckels causing perforation.

The distribution is shown in Table 1.

Table 17: Etiology of Ileal Perforations

Diagnosis	Frequency	Percent
Typhoid	27	54
Nonspecific	14	28
TB	8	16
Meckels	1	2
Total	**50**	**100**

Age and Sex Incidence

The age of patients ranged from 18 to 85 years. Perforation commonly occurred in the third and fourth decades of life with 48% of patients between the ages of 30 and 50. Only six female cases identified in the study. Typhoid perforation commonly occurred in the third and fourth decades with 48% of cases in that age group. Five cases of female typhoid ileal perforations were identified. Non-Specific perforations occurred commonly in a similar age group. The distributions of age and sex in all cases and etiology specific distributions are shown in tables 2 and 3.

Table 18: Age and Sex Incidence in Ileal Perforations

Age	Male	Female	Total	Percent
10 - 20	1	0	1	2
20 - 30	7	2	9	18
30 - 40	11	1	12	24
40 - 50	11	1	12	24
> 50	14	2	16	32
Total	**44**	**6**	**50**	**100**

Table 19: Age and Sex incidence in Typhoid, Nonspecific, TB and Meckel causing Ileal Perforations

Age	Typhoid			Non Specific			TB			Meckel		
	N (%)	M	F	N (%)	M	F	N (%)	M	F	N (%)	M	F
10-20	0	0	0	1(6.25%)	1	0	0	0	0	0	0	0
20-30	6(37.50%)	4	2	2(43.75%)	2	0	1(50%)	1	0	0	0	0
30-40	6(29.10%)	5	1	3(12.50%)	3	0	3	3	0	0	0	0
40-50	7(4.20%)	6	1	3(31.25%)	3	0	1(50%)	1	0	1	1	0
>50	8(8.40%)	7	1	5(6.25%)	4	1	3	3	0	0	0	0
Total	**27**	**22**	**5**	**14**	**13**	**1**	**8**	**8**	**0**	**1**	**1**	**0**

Symptoms and Signs

Most of the patients presented with symptoms and signs of peritonitis. The commonest symptoms were abdominal pain, fever and vomiting. The commonest signs were abdominal tenderness, guarding, intra-abdominal free fluid, distension and dehydration. Most patients of typhoid gave a history of fever. 12 % of patients were in shock. Symptoms and signs are shown in Table 5.

Table 20: Symptoms and Signs in Ileal Perforations

Symptoms	**Number**	**%**
Abdominal Pain	50	100%
Fever	43	86%
Vomiting	27	54%
Constipation	5	10%
Diarrhea	8	16%
Signs	**Number**	**%**
Dehydration	28	56%
Tenderness	50	100%
Guarding	40	80%
Distension	30	60%
Free Fluid	46	92%
Shock	15	30%

Table 21: Symptoms and Signs in each types of Ileal Perforations

Symptoms	**Typhoid N=27**	**Non Specific N=14**	**TB N=8**	**Meckels N=1**
Abd Pain	27	14	8	1
Fever	25	12	5	1
Vomiting	13	10	2	1
Constipation	3	1	1	0
Diarrhea	4	3	1	0
Signs				
Dehydration	16	6	5	1
Tenderness	27	14	8	1
Guarding	24	13	2	1
Distention	16	10	3	1
Free Fluid	25	14	6	1
Shock	9	4	2	0

Investigations

X-Ray: Pneumoperitoneum in chest and erect abdominal x-ray was seen in 56% of patients. Features of intestinal obstruction, including dilated bowel loops with air- fluid levels seen in erect abdominal x-ray.

Hematology and Biochemistry: Haemoglobin was less that 8 g/dL in 12 (24%) of patients and Albumin of < 3.5 g/dL was seen in 6 (12%) of cases. Azotemia as defined as a Blood Urea of > 52 mg/dL and/or Serum Creatinine > 2 mg/dL was seen in 20% of patients.

Microbiology: Blood cultures were done in 30 patients and growth was obtained in 5. Salmonella typhi was grown in all 5 patients. The typhoid growths were sensitive to cefotaxime, ceftriaxone piperacillin and amikacin. Widal test was positive in 12 patients out of 27 patients of enteric perforation (44%)

Histopathology: Pathological examination of either resected specimens or scrapings from the edge of the ulcer was done in all patients. A report suggestive of typhoid was seen in 27 cases. A diagnosis of tuberculosis was made in 8 cases, meckels in 1 case and the rest showed features of non-specific inflammation with no conclusive diagnosis.

Lag Period

It is the time between the onset of pain and the surgical intervention. In our study lag period was between 24 hours and 150 hours with around 52% of cases presenting after 72 hours. There was no significant difference in the mean lag periods of patients of typhoid or non-specific perforations. Traumatic perforation and stab injury though had a significantly reduced lag period.

Table 22: Lag Period of Typhoid, Nonspecific and TB Ileal perforation

Lag	**ileal N=50**	**%**	**Typhoid N=27**	**%**	**Nonspecific N=14**	**%**	**TB N=8**	**%**	**Meckel N=1**	**%**
<24	12	24	6	22	6	43	0	0	0	0
25-48	13	26	7	26	4	28.5	2	25	0	0
49-72	9	18	3	11	2	14.2	3	37.5	1	100
>72	16	32	11	41	2	14.2	3	37.5	0	0
Total	**50**		**27**		**14**		**8**		**1**	

Surgical Procedures

Simple 2-layer closure was the commonest procedure done (82%). Resection and anastomosis were done in 9(18%) patients.

Table 23: Surgical Procedures done for Typhoid, Nonspecific and TB Perforations.

Procedure	Ileal		Typhoid		Nonspecific		TB		Meckels	
Two layer Closure	26	52%	11	40.7%	13	93%	2	25%	0	0
Resection-anastamosis	24	48%	16	59.3%	1	7%	6	75%	1	100 %
Total	**50**		**27**		**14**		**8**		**1**	

Number and Site of Perforation

Multiple perforations occurred in 18% of patients, mostly in typhoid perforations (Table 8). Over 98% of perforations were within 2 feet (60 cms) from the ileocaecal junction and 92% within 30 cms.

Table 24: Number of Perforations

Number	Ileal	Typhoid	Nonspecific	TB	Meckels
1	41	21	14	5	1
2	7	4	0	3	0
3	2	2	0	0	0
Total	**50**	**27**	**14**	**8**	**1**

Complications

Complications occurred in 32 (64%) of all cases. The common complications seen were wound infection, would dehiscence, and respiratory complications. Faecal fistula was seen in 1 case. The highest complication rate was seen with resection- anastomosis and the least with simple closure though this difference was not statistically significant. Patients with typhoid had a complication rate of 70.8% with mortality occurring in 4 patients.

Table 25: Surgical Procedures and their Complication

Complications	Simple closure n=26	Resection-anastomosis n=24	Total N=50
Wound Infection	8(30.7%)	14(58.3%)	22
Wound Dehiscence	4(15.3%)	8(33.3%)	12
Abd Collection	0	0	0
Faecal Fistula	0	0	1
Reperforation	0	0	0
Respiratory	2(7.6%)	2(8.3%)	4
Mortality	3(11.5%)	7(29.1%)	10
Patients with complications	8(30.7%)	14(58.3%)	22(44%)

Table 26: Surgical Procedures and their Complications in Typhoid and Nonspecific Perforations

Complications	Typhoid perforations			Nonspecific perforations		
	simple closure n=11	resection-anastomosis n=16	Total N=27	simple closure N=13	resection -anast N=1	Total N=14
Wound Infection	6(54.5%)	9(56.2%)	15(55%)	2(15.3%)	0	2(14.3%)
Wound Dehiscence	5(45.4%)	5(31.2%)	10(37%)	0	0	0
Abdominal Collection	0	0	0	0	0	0
Faecal Fistula	0	0	0	0	0	0
Reperforation	0	0	0	0	0	0
Respiratory	1(9%)	2(12.5%)	3(11%)	0	0	0
Mortality	3(27.2%)	5(31.2%)	8(30%)	0	0	0
Patients with complications	6(54.4%)	9(56.2%)	15(55.5%)	2(15.3%)	0	2(14.3%)

Out of eight TB perforations, two cases died due to septicaemia. One case of meckels dint develop any complication

Operating Time and Hospital Stay

The average operating time was 87 minutes. Resection and anastomosis took a longer time than simple closure but the difference was not statistically significant. Median hospital stay was 17 days. There was no significant difference in the hospital stay of patients undergoing different surgical procedures.

Table 27: Surgical Procedure and Hospital Stay

Procedure	Mean
Simple Closure	18.65
Resection-Anastomosis	16.33
Total	**17.49**

P > 0.05

Mortality

The mortality rate was 10%. Mortality in patients of typhoid perforations was 16.6%. One patient of TB perforation expired. No patients of perforation with other causes died. Septicemia, faecal fistula and respiratory complications were the other causes of death.

Table 28: Surgical procedures and Morbidity and Mortality

	Simple Closure N=26	Resection Anastomosis N=24	Total N=50
Mortality	3(11.5%)	7(29.1%)	10(20%)
complications	8(30.07%)	14(58.3%)	22(44%)

Complication p > 0.05 Mortality p > 0.05

Table 29: Surgical procedures and Morbidity and Mortality in Typhoid

	Simple Closure N=11	Resection Anastomosis N=16	Total N=27
Mortality	3(27.2%)	5(31.2%)	8(30%)
Complications	6(54.4%)	9(56.2%)	15(55.5%)

Complication $p > 0.05$ Mortality $p > 0.05$

Table 30: Surgical procedures and Morbidity and Mortality in Non Specific Perforation

	Simple Closure N=13	Resection Anastomosis N=1	Total N=14
Mortality	0	0	0
Complications	2(15.3%)	0	2(14.3%)

Complication $p < 0.05$ Mortality $p > 0.05$

Table 31: Mortality Rates in Various Etiological Factors

Etiology	Number	Death	Percent
Typhoid	27	8	29.7%
Nonspecific	14	0	0
TB	8	2	25%
Meckels	1	0	0

Mortality $p > 0.05$

Table 32: Causes of Death in Ileal Perforations

Causes	Typhoid N=8	TB N=2	Total N=10
Septicemia	4	2	8(80%)
ARDS	4	0	2(20%)

Prognostic Factors

Surgical Procedure

The type of surgical procedure did not influence the mortality or morbidity in ileal perforations and also in etiology specific analysis. In patients with typhoid, simple closure and resection anastomosis had statistically similar mortality rates. Resection anastomosis had the highest complication rate but not statistically significant. Simple closure is a better option for single perforation and . Resection anastomosis is better for multiple perforations.

Lag Period

Most patients presented with peritonitis of greater than 24 hours duration. Increasing lag period was associated with increasing mortality and complication rate. The relationship of increasing lag period to both mortality and morbidity in ileal perforations was found to be significant with $p < 0.05$. This was also significant in patients of typhoid perforations with $p < 0.05$ for both mortality and complications

Table 33: Relation of Lag Period to Mortality and Complications.

Lag Period	No of cases	Complications	Death
<24	12(24%)	2(9%)	0
25-48	13(26%)	4(18%)	0
48-72	9(18%)	5(23%)	1(10%)
>72	16(32%)	11(50%)	9(90%)
Total	**50**	**22**	**10**

Complications $p < 0.05$ Death $p < 0.05$

Table 34: Relation of Lag Period to Mortality and Complications in Typhoid Perforation.

Lag Period	No of cases	Complications	Death
<24	6(25%)	1(6%)	0
25-48	7(29.16%)	4(23.5%)	0
48-72	3(25%)	4(23.5%)	1(12.5%)
>72	11(20.83%)	8(47%)	7(87.5%)
Total	**27**	**17**	**8**

Table 35: Relation of Lag Period to Mortality and Complications in Non Specific Perforation.

Lag Period	No of cases	Complications	Death
<24	6(18.75%)	0	0
25-48	4(12.5%)	0	0
48-72	2(43.75%)	1(50%)	0
>72	2(25%)	1(50%)	0
Total	**14**	**2**	**0**

Other factors

Table 36: Risk Factors for Morbidity in Ileal Perforations

Risk Factor	Morbidity n = 22	No Morbidity n = 28	p
Age > 50	**7**	**9**	**<0.05**
Female sex	5	1	>0.05
Male sex	17	27	>0.05
Shock	**9**	**0**	**<0.05**
Hb < 8	**12**	**0**	**<0.05**
Alb < 3.5	**6**	**0**	**<0.05**
Azotemia	**10**	**0**	**<0.05**
Multiple perf	5	4	>0.05
Typhoid	**15**	**12**	**<0.05**

Morbidity was significantly influenced by age greater than 50, hypoalbuminemia, azotemia, HB<8, shock and a diagnosis of typhoid as the cause of perforation.

Table 37: Risk Factors for Mortality in Ileal Perforations

Risk Factors	Mortality n = 10	survivors n = 40	p
Age > 50	**3**	**13**	**<0.05**
Female sex	3	3	>0.05
Male sex	4	40	>0.05
Shock	**6**	**3**	**<0.05**
Hb < 8	2	10	>0.05
Alb < 3.5	**3**	**3**	**<0.05**
Azotemia	3	7	>0.05
Multiple perf	2	7	>0.05
Faecal fistul	0	0	>0.05
Fecal Peritonitis	2	15	>0.05
Typhoid	**8**	**19**	**<0.05**

Mortality was significantly influenced by age greater than 50, hypoalbuminemia, typhoid and shock on admission.

Tables 23 and 24 show the other risk factors for morbidity and mortality in ileal perforations and their statistical significance. For morbidity age > 50 years, reduced serum albumin, Hb, azotemia, shock and an etiology of typhoid were associated with a significantly high morbidity. Multiple perforations and sex did not affect the complication rate. Mortality was affected significantly by age, albumin level, typhoid and the presence of shock on admission. Sex, biochemical parameters, number of perforations and type of peritoneal fluid were not statistically significant.

FIGURES

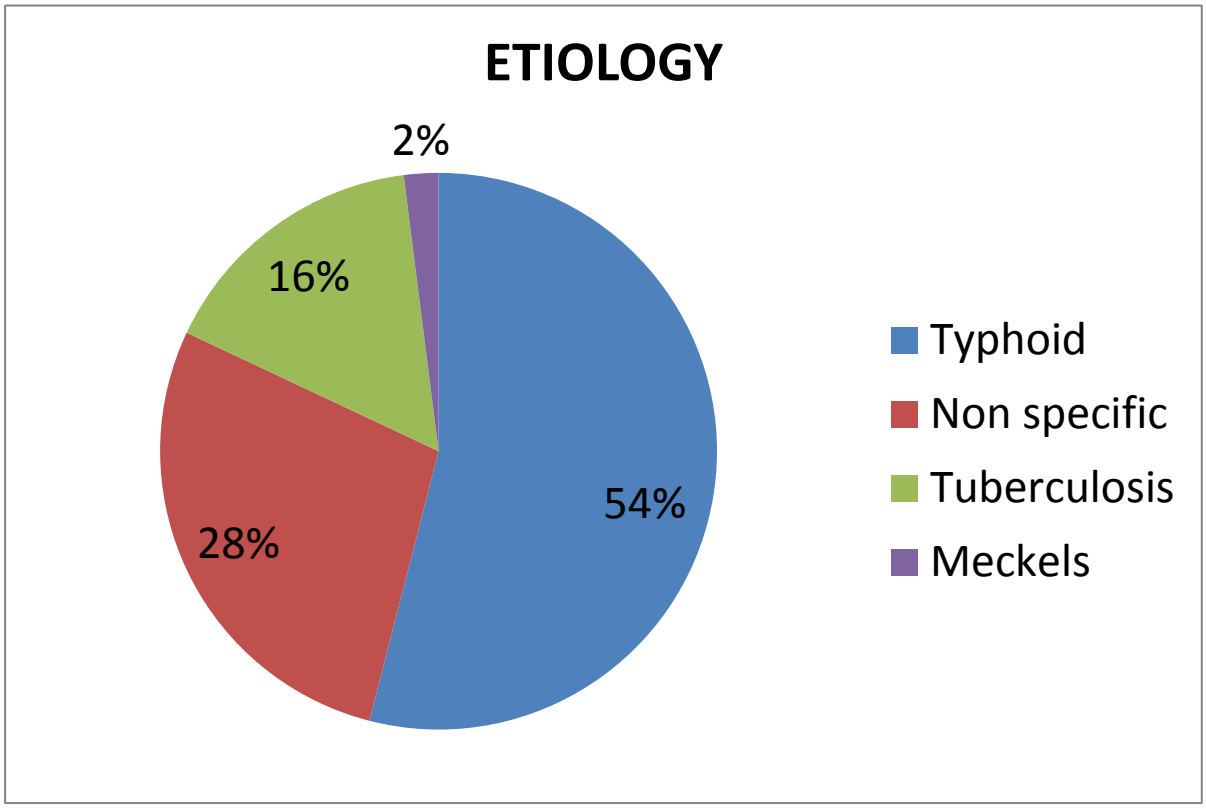


FIGURE 1: ETIOLOGY OF NON TRAUMATIC ILEAL PERFORATIONS

FIGURE 2: AGE DISTRIBUTION IN ILEAL PERFORATIONS

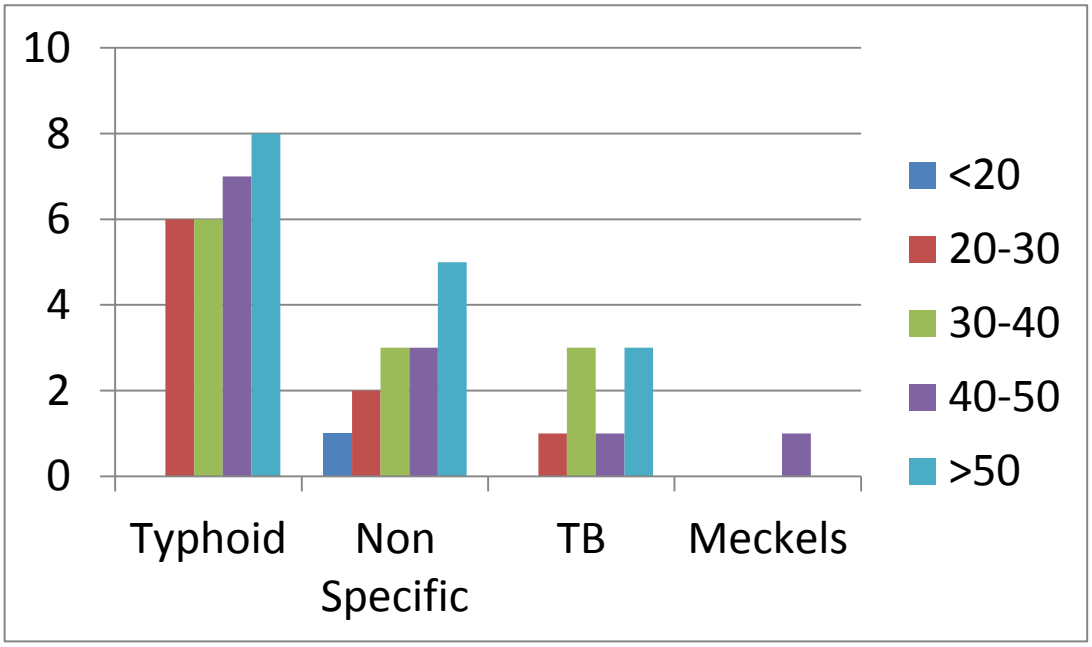


FIGURE 3: SYMPTOMS OF ILEAL PERFORATIONS

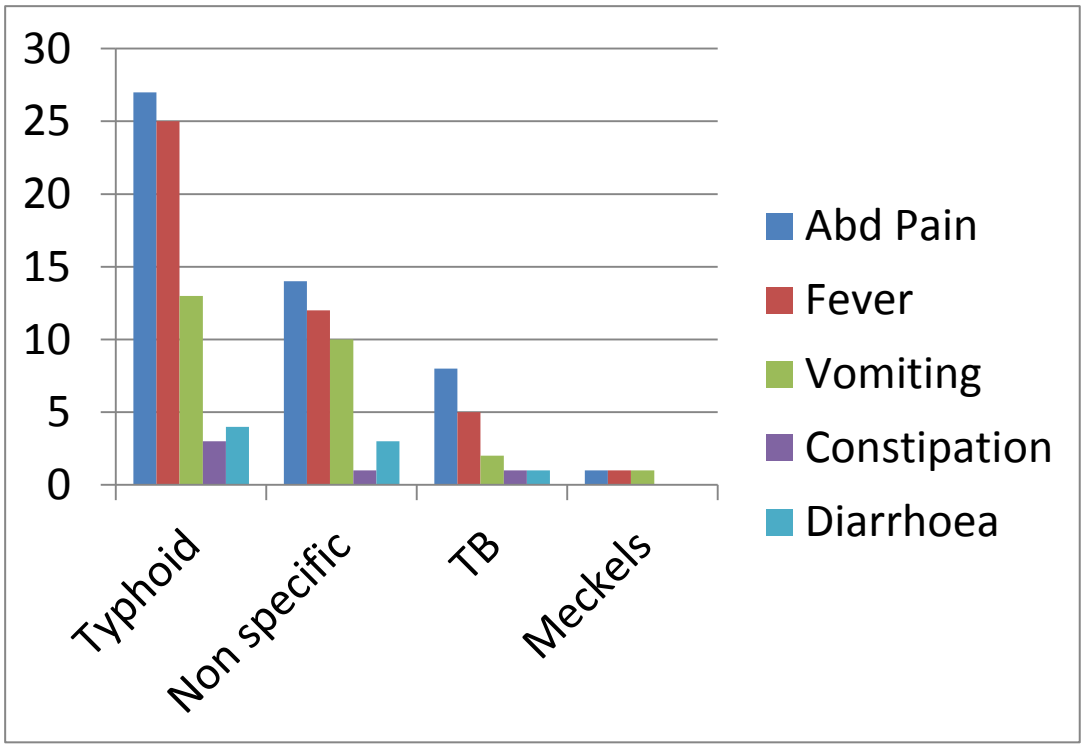
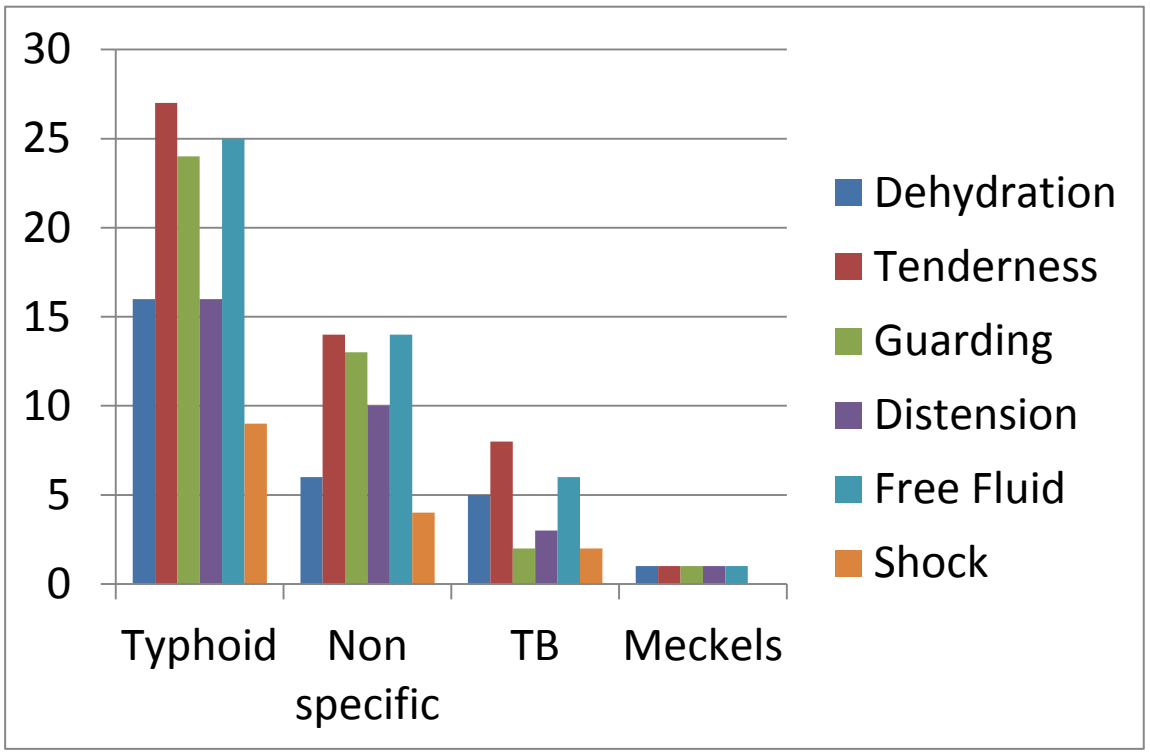


FIGURE 4: SIGNS OF ILEAL PERFORATIONS



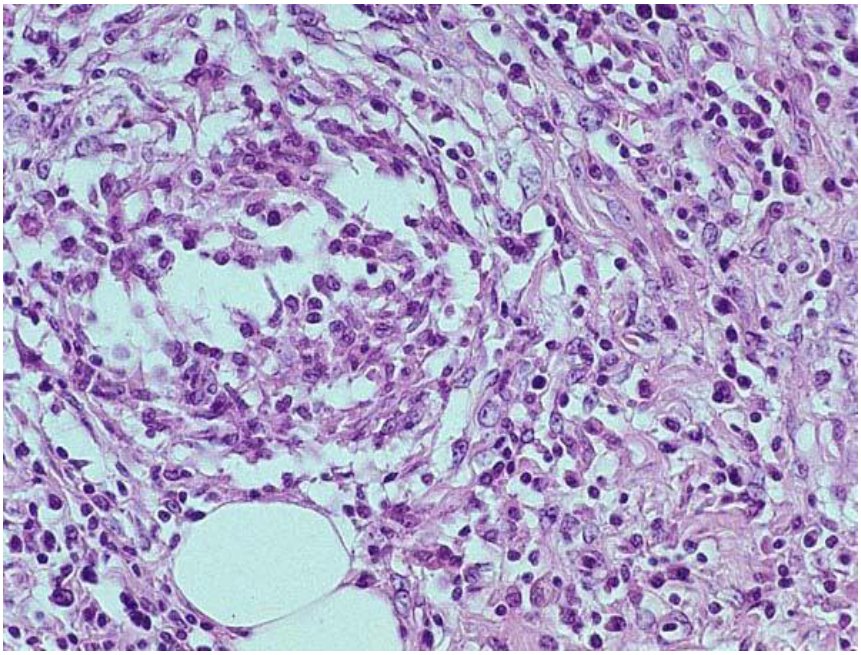


FIGURE 5: HISTOPATHOLOGY IN TYPHOID ILEAL PERFORATION

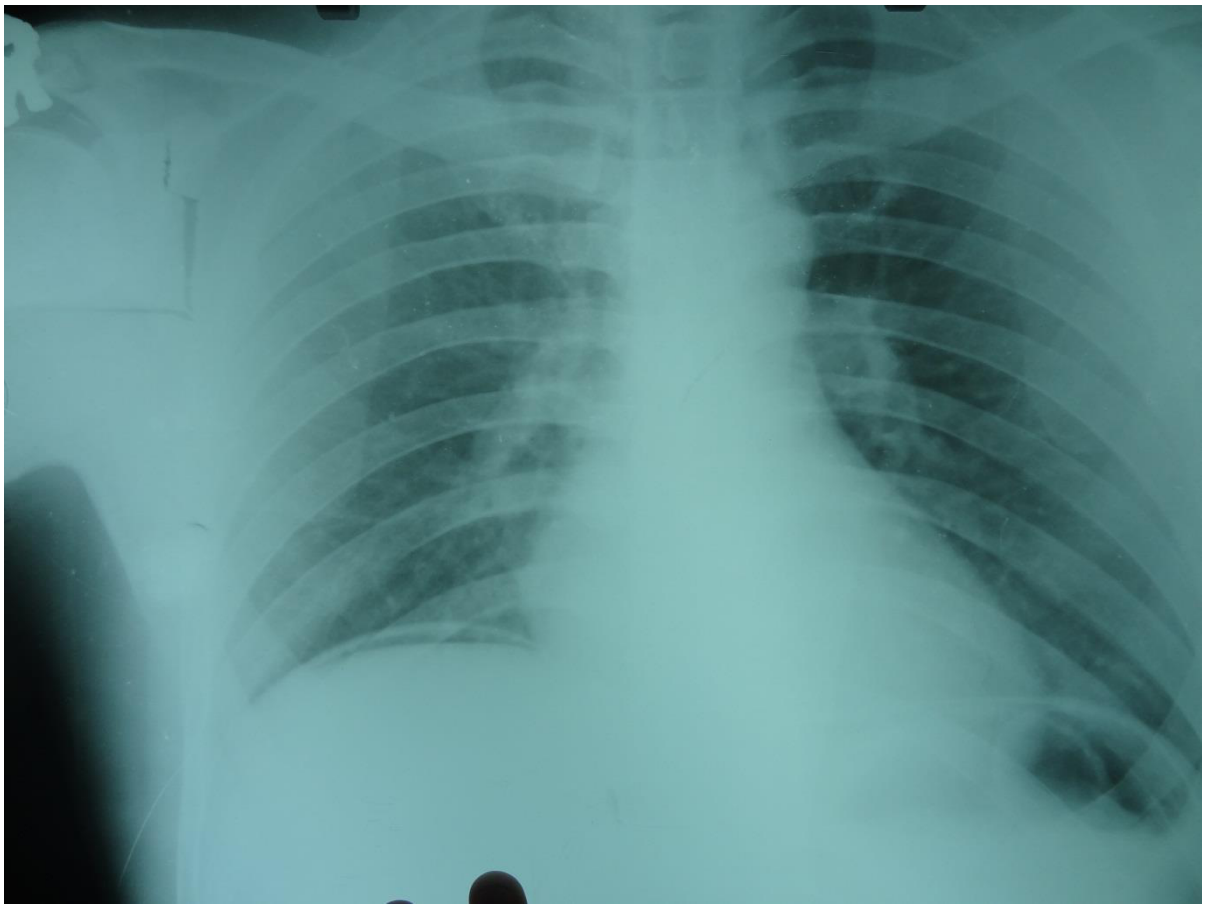


FIGURE 6: AIR UNDER DIAPHRAGM

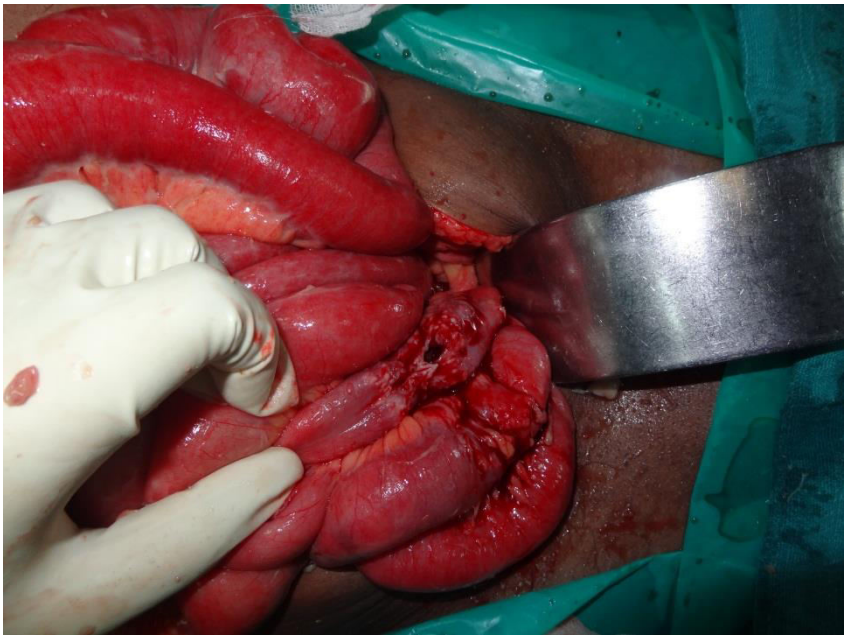


FIGURE 7: ILEAL PERFORATION

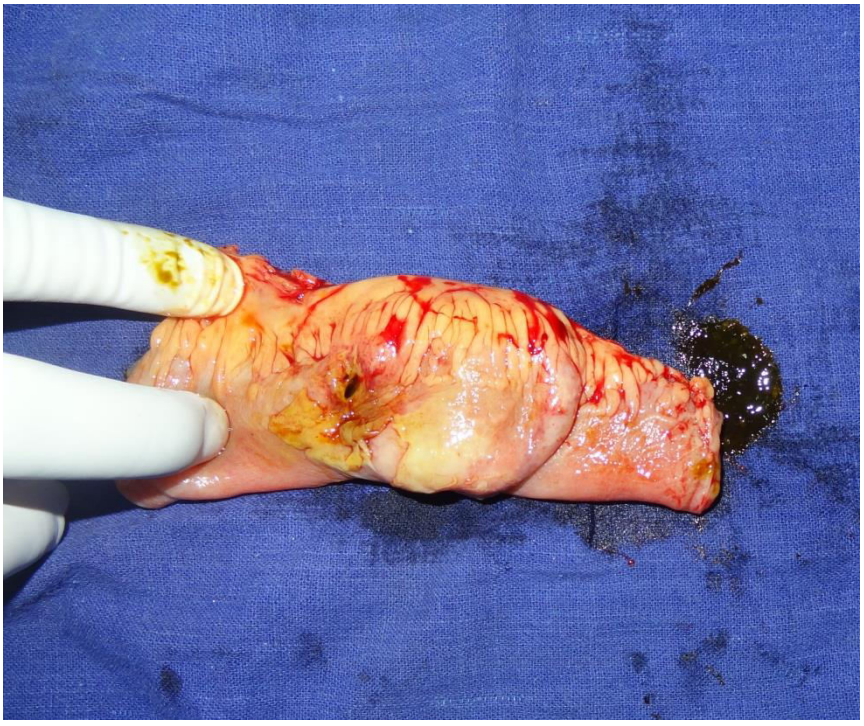


FIGURE 8: MECKELS DIVERTICULAR PERFORATION

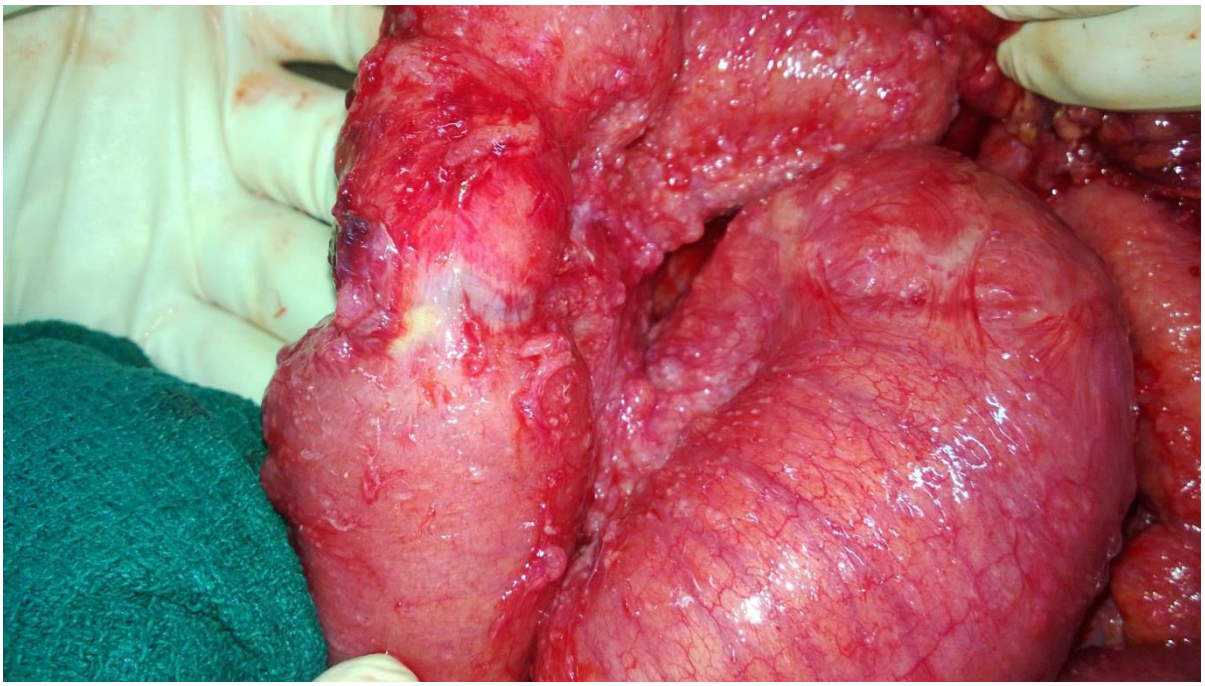


FIGURE 9: TB ABDOMEN

FIGURE 10: DOUBLE BARELL ILEOSTOMY

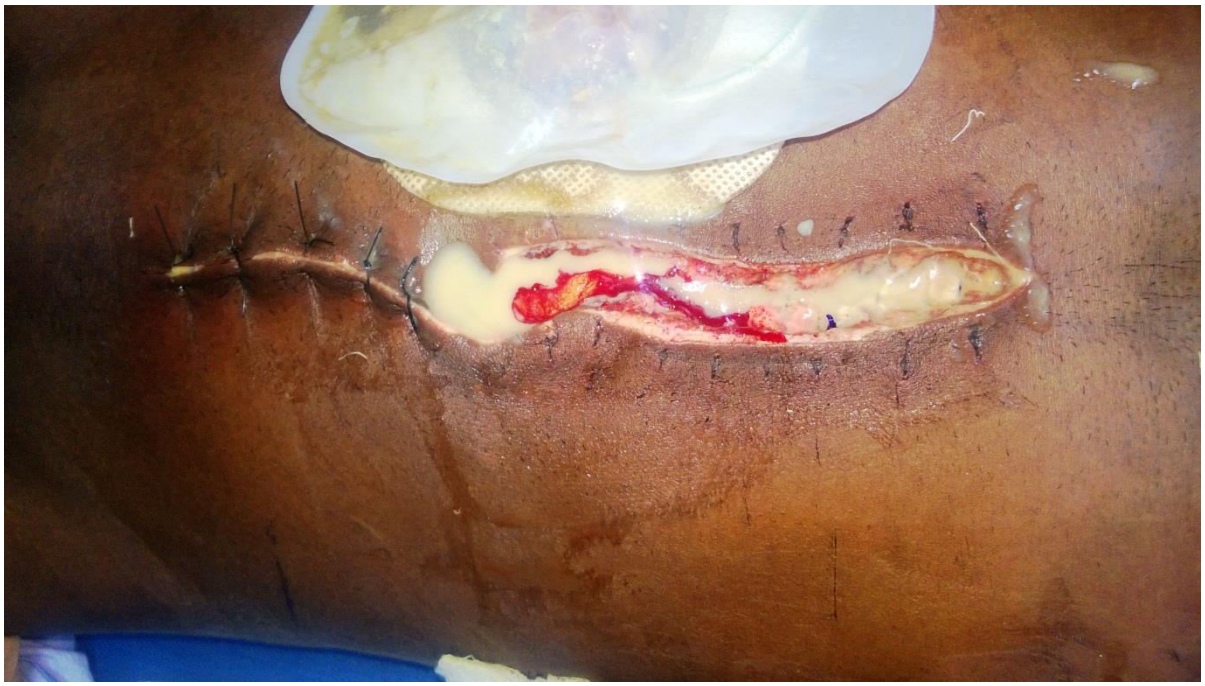


FIGURE 11 : WOUND INFECTION WITH DEHISCENCE

CHAPTER 6

DISCUSSION

DISCUSSION

The commonest cause of ileal perforation in the series was typhoid fever accounting for 54% of cases. Typhoid fever was the commonest cause of ileal perforation in tropical countries. Typhoid fever accounted for 56.6% of cases of ileal perforation in the series by Karmakar [1]. Mechanical causes and malignancy are the commonest causes of small bowel perforation in the western world. Mechanical causes and lymphomas accounted for 40.7% of perforations in the series by Dixon [2]. Malignancy was the commonest cause in the series by Orringer [3]. There were no cases of typhoid perforations in either series [2, 3].

When the etiology of the perforation was not identified it was termed non-specific perforation. Non-specific perforation was the second commonest cause in this study accounting for 28% of cases. Twelve patients of non-specific perforation had fever prior to onset of abdominal symptoms. Widal test, blood culture and histopathology were not suggestive of typhoid. These cases may be undiagnosed cases of typhoid. Non-specific perforations were the commonest cause of small bowel perforation in the series by Dixon and Bhalerao [2, 11].

TB accounted for 16% of cases of ileal perforations in this study. Mortality rate is 25% in our study. 4.9% of intestinal TB undergoes perforation [78]. It

has a poor prognosis with mortality rate higher than 30% [81,82] . Talwar et al have found 19% of nontraumatic small bowel perforations were due to intestinal TB [83]. In 90% of the cases, perforation is solitary [91]. Multiple TB perforations are associated with poor prognosis. In our study both cases had solitary perforation.

There was a male preponderance with the male: female ratio in this study being 22:3. Five cases of typhoid perforations were seen in females. Published literature shows a similar finding with reported ratios from 2.3:1 to 6.1:1 [24, 18].

Typhoid perforations as reported by Eggleston occurred in the second and third decades of life [25]. In this study 37% of typhoid and 43% of nonspecific perforations were in a similar age group.

Most patients presented with features suggestive of peritonitis. Patients of both typhoid and non-specific perforations had similar presentation with respect to abdominal symptoms and signs. Patients with typhoid perforation had fever, abdominal pain and vomiting. Examination revealed tenderness, guarding, distension and intraperitoneal free fluid. 9 patients were in shock on admission. Eggleston reported that most patients had fever, malaise and sudden increase in abdominal pain in typhoid perforation. Examination revealed signs of toxemia and acute abdomen [25].

Perforation was commonly seen to occur in the second week following onset of illness [19, 20, 29, 30]. Keenan reported that 88% of patients perforated in the second week [19]. Lizzaralde reported that 54.2% of patients perforated in the second week [30]. In this series the perforation was earlier with a majority occurring within a week of onset of fever.

Chest X-ray is a useful investigation to detect hollow viscus perforation. Free gas was seen under the diaphragm in 56% of perforations and in 63% of typhoid perforation.

Abdominal X-ray revealed gas of features suggestive of ileus. Pneumoperitoneum has been reported in 52% to 82% in studies by Hadley, Archampong, Tacyildiz and Vaidhyanathan [19, 21, 34, 56].

Widal was positive in 24% of tested cases and in 44% of patients of typhoid perforation. Widal was reported positive in 30% of patients with typhoid perforation by Kaul and in 46.1% of patients by Santillana [20, 34]. It was reported positive in% of cases by Jarrett and in 73% by Vaidyanathan [44, 56].

Salmonella typhi was grown in 5 patients with typhoid ileal perforation in whom blood cultures were done. All cultures were sensitive to piperacillin, Cefotaxime and ceftriaxone. Hadley reported positive cultures

in 22.2% and Santillana in 48% of patients [19, 20]. Prior antibiotic therapy was probably responsible for the low isolation in the study [19, 21]. Another cause may be delay in plating the samples.

Tuberculosis was diagnosed definitively by histopathology. Histopathology was suggestive of typhoid in all enteric perforation patients. The presence of erythrophagocytosis virtually confirms the diagnosis of typhoid perforation [32].. Both the TB cases were confirmed by HPE. Widal along with HPE is the most useful test for typhoid. It is easily available and is less susceptible to prior therapy when compared to blood culture. This usefulness was confirmed by Jarrett [42].

In this study most patients of confirmed typhoid were treated with piperacilin or ceftriaxone and metronidazole. In the management of typhoid perforation some authors advocated conservative Management [36, 37, 38]. Presently there is no such controversy in the treatment of typhoid perforation with the current recommendation being surgical management [22]. The various methods in use are local drains, simple closure, closure with omental patch, wedge resection, resection and anastomosis, ileotransverse anastomosis and ileostomy [28,30,34,42,43,44]. In this study patients underwent simple closure or resection anastomosis. No patients were treated by conservative measures, wedge resection, omental patch

repair ileotransverse anastomosis or ileostomy. Resection was employed in typhoid perforations wherein multiple perforations were found and in meckel's perforations. Orloff recommended debridement and closure in patients of traumatic perforation where the injury was small and resection-anastomosis in patients with large wounds or multiple perforations [72].

The overall complication rate for all patients in this series was 44%. Typhoid perforations are associated with a high morbidity rate with literature reports between 28.5% and 81% [19,20,29,34]. Santillana in his series reported a rate of 71.9% in 96 patients. In this series typhoid perforations had a complication rate of 70%. The common complications were wound infection, wound dehiscence, and respiratory complication which compare with published reports [19,20,26,31].

In patients of typhoid perforation the mortality was 29.7%. Though this rate has been on the decline, reported rates are between 3% and 60%. In non specific perforations no mortality was found. This difference shows a trend towards significance on statistical analysis. Typhoid perforations in this study thus showed a poorer prognosis than the other etiologies.

The surgical procedure did not influence either the morbidity or the mortality in patients irrespective of etiology. Resection-anastomosis was found to have a higher complication rate but this was not statistically significant. Eggleston reported that the procedure done did not influence

outcome [25]. Talwar and Sharma reported that mortality was least with early primary closure and Ameh et al found mortality was highest with wedge resection and least with resection and anastomosis [43, 44].

Lag period has been known to influence both mortality and morbidity. Regression analysis showed that the mortality and morbidity increased with increasing lag period.

This association was also found in patients of typhoid perforations. Increasing lag period was associated with increased mortality in series by Archampong, Eggleston, Bose and Talwar [21,25,43,72].

In patients of ileal perforation the significant factors influencing mortality are age greater than 50, female sex, feculent peritonitis, raised blood urea or creatinine as per the Manheim peritonitis index. In this study age greater than 50, shock at presentation, albumin<3.5 and typhoid were significant factors influencing mortality. Factors increasing morbidity are age>50 years, shock at presentation, Hb<8 gms%, albumin<3.5, azotemia and typhoid perforations.

Archampong reported that urine output prior to surgery, blood urea and serum potassium affected survival in patients of typhoid perforation. Survival was independent of haemoglobin level, shock, sickling status and

number of perforations [19]. Mock reported that increasing number of perforations, generalised contamination of the peritoneal cavity and single layer closure influenced survival [21]. Eggleston in his series of 78 patients reported the shock, uremia, encephalopahy, fecal peritonitis and postoperative fecal fistula were predictors of mortality [25].

CHAPTER 7

SUMMARY AND CONCLUSIONS

SUMMARY AND CONCLUSIONS

This study was conducted from September 2013 to September 2014. It includes fifty cases of ileal perforation admitted to Institute of General Surgery, Madras Medical College in that period. Etiology, presentation, management and outcome of patients with non traumatic ileal perforations were studied with emphasis on typhoid, non-specific, TB, meckels and the factors that influenced the prognosis.

- Typhoid is the most common cause of Ileal perforation, followed by non- specific perforations.
- Patients have a male preponderance and are usually in the third and fourth decades of their lives.
- Widal serology is a useful test in the diagnosis of typhoid fever. Histopathology is useful in the diagnosis of tubercular perforations and very useful in the diagnosis of typhoid.
- Typhoid perforations have a significantly higher morbidity rate than non- specific meckels, and tuberculosis.
- Mortality is more in case of typhoid ileal perforation.
- The type of surgical procedure did not influence outcome, either morbidity or mortality.
- Lag period significantly influenced outcome. This was true for

cases of ileal perforation irrespective of etiology and significant when typhoid perforations were separately considered.

- Morbidity was significantly influenced by age greater than 50, hypoalbuminemia, azotemia, HB<8, shock and a diagnosis of typhoid as the cause of perforation.
- Mortality was significantly influenced by age greater than 50, hypoalbuminemia, typhoid and shock on admission.

CHAPTER 8

BIBLIOGRAPHY

BIBLIOGRAPHY

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CHAPTER 9

ANNEXURES

PROFORMA

Name: **Age:** **Sex:**

IP No. :

DOA : **DOS :** **DOD :**

Chief Complaints :

Presenting Complaints :

Co Morbid Illness :

Past Surgical / Medical History :

CLINICAL EXAMINATION:

General Condition :

VITAL SIGNS

Pulse: **BP:** **Temp:**

SYSTEMIC EXAMINATION

CVS :

RS :

ABDOMEN ;

INSPECTION ;

PALPATION ;

AUSCULTATION ;

PR;

CLINICAL DIAGNOSIS

INVESTIGATIONS :

Hemogram:

Renal Function Test:

Liver Function Test;

Ultrasound:

CXR:

Xray Abdomen :

CECT abdomen;

Blood C/s :

Widal :

HPE :

PEROPERATIVE FACTORS :

1. **Date of operation :**
2. **Time of Starting :**
3. **Lag Period :**
4. **Duration of operation :**
5. **Findings :**
6. **Procedure Done :**

POST OPERATIVE PERIOD :

POST OPERATIVE COMPLICATIONS :

CONDITION ON DISCHARGE

FOLLOW UP;

MASTER CHART

S. NO.	NAME	AGE	SEX	IP.NO	NO. OF PERFORATION	SIZE OF PERFPRATION (cm)	DISTANCE FROM IC VALVE (cm)	PROCEDURE DONE	LAG PERIOD	DURATION OF SURGERY	Death (days)	WIDAL	BLOOD C/S	HPE REPORT	Coll	RS	Inf	Deh	Fistula	Reperf
1	SATISH	22	M	79731	Sin	1 X 1	20	PC	<24	1-2hrs		Negative		NSP						
2	DEVENDRAN	44	M	31848	Sin	3 x 1.5	15	LRA, LI	<72	>2hrs		Negative		MD						
3	MURUGESAN	38	M	41279	Mul	2x2	20,50,60	LRA	>72	>2hrs		Positive	+	EF						
4	SASIKUMAR	36	M	61143	Mul	2x2.5	15,20	LRA	>72	>2hrs		Negative		TB			+			
5	DILLI	18	M	61320	Sin	1 X 1	25	PC	<48	1-2hrs		Negative		NSP						
6	SAROJINI	45	F	69295	Sin	1 X 1	25	PC	>72	1-2hrs	1	Positive		EF			+	+		
7	KUMARESAN	46	M	87886	Sin	1 X 1	20	PC	<24	1-2hrs		Negative		NSP						
8	ANBU	35	M	89154	Sin	.8 x .6	30	PC	<48	1-2hrs		Positive		EF			+	+		
9	JEEVITHA	20	F	90584	Mul	2x1	15,20,25	LRA, LI	>72	>2hrs		Positive	+	EF			+	+		
10	ANAND	34	M	98637	Sin	1.5 x 1	20	PC	>72	>2hrs		Negative		NSP						
11	KASI	60	M	98757	Sin	1 x 0.8	25	PC	<24	1-2hrs		Negative		NSP						
12	SIVA	25	M	102891	Sin	1 X 1	20	PC	<48	1-2hrs		Negative		NSP						
13	MUTHUSAMY	61	M	107732	Sin	1 X 1	40	PC	>72	1-2hrs	5	Positive		EF		+	+			

14	KUMAR	40	M	108525	Sin	1 X 1	20	PC	<48	1-2hrs		Negative		EF						
15	GANESH	40	M	109637	Sin	1 x 0.8	30	PC	<24	1-2hrs		Negative		NSP						
16	ALAGHUKUMAR	25	M	111779	Sin	1 X 1	25	PC	<48	1-2hrs		Negative		EF						
17	KOVALAN	45	M	122412	Sin	1 X 1	20	PC	<24	1-2hrs		Negative		NSP			+	+		
18	KRISHNAN	66	M	1541	Mul	2x2	10,20	LRA, LI	>72	>2hrs	2	Positive		EF			+	+		
19	VENGAIYAH	25	M	3840	Sin	1 X 1	25	PC	<48	1-2hrs		Negative		EF			+			
20	MEENATCHI	85	F	8704	Sin	2 x 2	15	LRA	<72	1-2hrs		Negative		EF			+			
21	JOTHY	37	F	10986	Sin	1.5 x 1	25	PC	>72	1-2hrs	2	Positive	+	EF		+	+			
22	BANKAJAVALLI	65	F	123706	Sin	1 X 1	30	PC	<24	1-2hrs		Negative		NSP						
23	KUNAL	35	M	4313	Sin	2 x 2	25	LI	>72	1-2hrs	2	Negative		TB			+	+		
24	YUVENDRAN	33	M	13564	Sin	.5 x .5	40	PC	<48	1-2hrs		Negative		NSP						
25	RAMADOSS	63	M	111173	Mul	3x2	25	LRA	<72	1-2hrs		Negative		TB						
26	RAMASAMY	70	M	112214	Sin	3 x 2	20	LRA, LI	>72	>2hrs	3	Positive	+	EF		+	+			
27	MOORTHI	60	M	4427	Sin	1 X 1	15	PC	<72	1-2hrs		Negative		EF			+	+		
28	ADHINARAYANAN	55	M	18338	Sin	2 x 2	30	LI	>72	>2hrs		Negative		EF						
29	PAKKINI	40	M	21233	Sin	3 x 2	25	LRA, LI	<24	>2hrs		Negative		EF						

30	DEVENDRAN	44	M	31848	Sin	3 x 2	30	LRA, LI	>72	>2hrs	2	Negative		TB			+	+		
31	KAMATCHI	22	M	36808	Mul	1.2x1	20, 25	LRA	<24	>2hrs		Negative		EF			+			
32	MURUGESAN	45	M	37340	Sin	2 x 2	20	LRA	<24	1-2hrs		Negative		EF						
33	KRISHNA	85	M	42030	Sin	2 x 2	15	LRA	<48	1-2hrs		Negative		TB			+			
34	SIVAKUMAR	38	M	61143	Mul	1 X 1	25	LRA	>72	>2hrs	7	Positive		EF			+	+		
35	CHINNAPPA	68	M	61364	Sin	1 X 1	25	PC	<72	1-2hrs		Negative		NSP						
36	KANNIYAPPAN	30	M	76766	Sin	2 x 2	40	LRA	<48	1-2hrs		Negative		EF						
37	GAJENDRAN	37	M	83400	Sin	1 X 1	25	PC	>72	1-2hrs		Negative		NSP						
38	PATCHIADOSS	30	M	59259	Sin	1 X 1	20	PC	<72	1-2hrs		Negative		TB						
39	BALASUBRAMANI	42	M	64636	Sin	2 x 2	30	LI	<72	>2hrs	2	Positive		EF			+	+		
40	VINAYAGAM	30	M	68512	Sin	2 x 2	20	LRA, LI	<48	>2hrs		Negative		EF		+	+			
41	ESWARAMMAL	52	M	75106	Sin	2 x 2	25	LRA	<48	1-2hrs		Negative		NSP						
42	BHARATHI	25	F	78799	Sin	2 x 2	20	LRA, LI	>72	>2hrs	1	Positive	+	EF			+	+		
43	ULAGANATHAN	55	M	39819	Sin	1 X 1	20	PC	<72	1-2hrs		Negative		NSP			+			
44	NAGARAJ	55	M	49453	Sin	1.5 x 1	30	PC	<72	1-2hrs		Negative		TB						
45	SHANMUGAM	26	M	53357	Sin	2 x 2	50	LI	<48	1-2hrs		Positive		EF						

46	DHANSEKAR	25	M	59868	Mul	1 X 1	30	LRA	<48	1-2hrs		Negative		TB			+	+	
47	SUBRAMANI	62	M	59368	Sin	1 X 1	20	PC	<24	1-2hrs		Negative		EF					
48	KRISHNAN	41	M	35781	Mul	1 X 1	20, 25	LRA	>72	>2hrs		Negative		EF					
49	PANNEER	45	M	75529	Sin	1 X 1	20	PC	<24	1-2hrs		Negative		EF					
50	SADHASIVAM	62	M	91395	Sin	2 X 1	15	PC	<24	1-2hrs		Negative		EF					

S. NO.	NAME	IPNO	AGE	SEX	SYMPTOMSM		HISTORY				EXAMINATION							INVESTIGATIONS						DIAGNOSIS
					Fever	Pain	Fever	Vom	Cons	Diar	Pulse	SBP	Dehy	Dist	Tender	Free Fluid	BS	Hb	Azo	Alb	Gas	Ileus	Tap	
1	SATISH	79731	22	M	7	2	+	_	_	_	110	80	+	+	+	+	_	+	_	_	+	+	+	NSP
2	DEVENDRAN	31848	44	M	3	3	+	+	_	_	140	78	+	+	+	+	_	_	_	_	+	+	+	MD
3	MURUGESAN	41279	38	M	_	1	_	+	_	_	100	110	+	+	+	+	_	+	_	_	+	+	_	EF
4	SASIKUMAR	61143	36	M	3	2	+	+	_	_	90	110	+	+	+	+	_	_	_	_	_	_	+	TB
5	DILLI	61320	18	M	_	1hr	_	_	_	_	92	122	+	+	+	_	+	_	_	_	_	+	_	NSP
6	SAROJINI	69295	45	F	7	2	+	+	+	_	130	78	+	+	+	_	_	+	_	+	+	+	+	EF
7	KUMARESAN	87886	46	M	_	4	_	+	+	_	120	80	+	+	+	+	_	+	+	+	_	+	+	NSP
8	ANBU	89154	35	M	5	2	+	+	+	_	96	110	+	+	+	+	_	_	_	_	+	+	+	EF
9	JEEVITHA	90584	20	F	14	2	+	_	+	_	88	120	_	+	+	+	_	_	+	_	+	+	+	EF
10	ANAND	98637	34	M	14	1	+	_	_	_	120	116	+	+	+	+	_	_	_	_	+	+	_	NSP
11	KASI	98757	60	M	—	1	_	+	_	_	119	120	+	+	+	+	_	_	_	_	+	+	_	NSP
12	SIVA	102891	25	M	20	1	+	+	+	_	80	110	_	+	+	+	_	_	_	_	+	+	_	NSP
13	MUTHUSAMY	107732	61	M	7	1	+	_	_	_	120	110	+	+	+	+	_	_	—	_	+	+	_	EF
14	KUMAR	108525	40	M	4	3	+	_	_	_	126	76	_	+	+	+	_	_	_	_	+	+	+	EF
15	GANESH	109637	40	M	10	2	+	+	_	+	96	112	+	+	+	+	_	_	_	_	+	+	+	NSP
16	ALAGHUKUMAR	111779	25	M	14	1	+	+	_	_	90	150	_	+	+	+	_	_	_	_	+	+	_	EF
17	KOVALAN	122412	45	M	5	2	+	_	+	_	110	110	+	_	+	+	_	+	_	_	_	+	+	NSP
18	KRISHNAN	1541	66	M	4	2	+	+	_	_	120	96	+	_	+	+	_	_	+	_	+	+	+	EF
19	VENGAIYAH	3840	25	M	10	1	+	_	_	_	90	120	_	_	+	+	_	_	_	_	_	+	_	EF
20	MEENATCHI	8704	85	F	4	2	+	_	+	_	88	116	_	_	+	+	_	_	_	_	_	+	+	EF
21	JOTHY	10986	37	F	2	1	+	+	_		76	130	_	_	+	+	_	_	_	_	_	+	_	EF
22	BANKAJAVALLI	123706	65	F	7	5	+	_		+	80	124	_	_	+	+	_	_	_	_	_	+	+	NSP
23	KUNAL	4313	35	M	_	2	_	_		_	84	120	_	_	+	+	_	_	_	_	_	+	+	TB

24	YUVENDRAN	13564	33	M	4	2	+	+	+	_	116	98	+	_	+	+	_	+	_	_	+	+	+	NSP
25	RAMADOSS	111173	63	M	5	1	+	_	+	_	122	96	+	_	+	+	_	+	_	_	_	+	_	TB
26	RAMASAMY	112214	70	M	7	3	+	_	+	_	106	78	+	_	+	+	_	_	—	_	_	+	+	EF
27	MOORTHI	4427	60	M	6	3	+	+	_	_	92	112	_	_	+	+	_	+	_	_	_	+	+	EF
28	ADHINARAYANAN	18338	55	M	3	3	+	+	+	_	88	118	_	_	+	+	_	_	_	_	+	+	+	EF
29	PAKKINI	21233	40	M	_	2	_	_	+	_	86	124	_	_	+	+	_	_	_	_	_	+	+	EF
30	DEVENDRAN	31848	44	M	6	2	_	_	+	_	90	106	_	_	+	+	_	_	_	_	_	+	+	TB
31	KAMATCHI	36808	22	M	7	3	+	_	_	_	100	110	+	+	+	+	_	_	+	+	+	+	+	EF
32	MURUGESAN	37340	45	M	7	3	+	+	_	+	84	140	_	_	+	+	_	_	_	_	_	+	+	EF
33	KRISHNA	42030	85	M	25	4	+	+	_	_	100	110	+	+	+	+	_	_	+	+	+	+	+	TB
34	SIVAKUMAR	61143	38	M	_	7	_	+	_	+	110	110	+	+	+	+	_	_	_	_	+	+	+	EF
35	CHINNAPPA	61364	68	M	3	1	+	+	_	+	112	80	+	+	+	+	_	_	+	_	_	+	_	NSP
36	KANNIYAPPAN	76766	30	M	3	9	+	_	_	_	92	120	+	+	+	+	_	—	_	_	_	+	+	EF
37	GAJENDRAN	83400	37	M	1	1	+	+	_	_	86	122	_	_	+	_	+	_	_	+	_	+	_	NSP
38	PATCHIADOSS	59259	30	M	1	3	+	+	+	_	92	110	_	_	+	+	_	_	+	_	+	+	+	TB
39	BALASUBRAMANI	64636	42	M	4	4	+	_	_	+	92	130	_	+	+	+	_	_	+	_	+	+	+	EF
40	VINAYAGAM	68512	30	M	3	1	+	_	_	_	140	118	+	+	+	+	_	_	_	_	+	+	_	EF
41	ESWARAMMAL	75106	52	M	_	4	_	+	_	_	122	80	+	+	+	+	_	_	_	+	+	+	+	NSP
42	BHARATHI	78799	25	F	_	1	_	_	_	_	90	110	_	_	+	_	+	_	_	_	+	_	_	EF
43	ULAGANATHAN	39819	55	M	_	3	_	+	_	+	100	150	_	+	+	+	_	+	_	_	_	+	+	NSP
44	NAGARAJ	49453	55	M	3	3	+	+	_	_	104	110	+	+	+	+	_	+	+	_	+	+	+	TB
45	SHANMUGAM	53357	26	M	3	3	+	+	_	+	86	110	_	+	+	+	_	_	_	_	+	+	+	EF
46	DHANSEKAR	59868	25	M	10	4	+	+	_	_	90	110	_	+	+	+	_	_	_	_	_	+	+	TB
47	SUBRAMANI	59368	62	M	10	4	+	+	_	_	110	100	+	+	+	+	_	+	+	_	+	+	+	EF
48	KRISHNAN	35781	41	M	_	1	_	_	+	_	118	94	+	_	+	+	_	+	_	_	_	+	+	EF
49	PANNEER	75529	45	M	5	2	+	_	+	_	116	98	+	_	+	+	_		_	_	_	+	+	EF
50	SADHASIVAM	91395	62	M	14	2	+	+	_	_	86	122	_	+	+	+	_		_	_	+	+	_	EF

KEY TO MASTER CHART

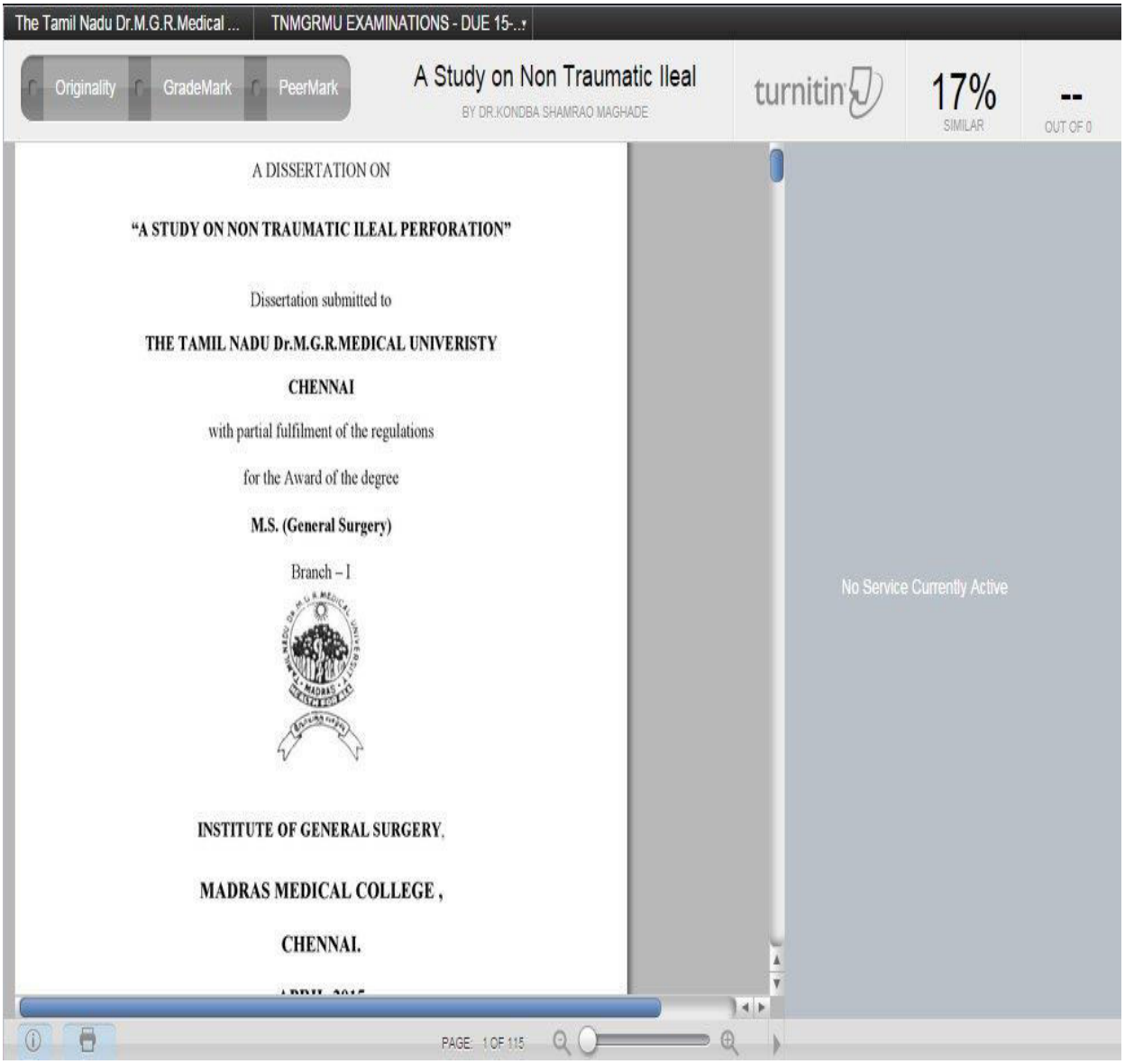
M	-	Male
F	-	Female
Sin	-	Single
Mul	-	Muliple
PC	-	Primary Closure
LRA	-	Limited resection and anastomosis
LI	-	Loop ileostomy
EF	-	Enteric Fever
NSP	-	No specific pathology
TB	-	Tuberculosis
MD	-	Meckels Diverticulum
Vom	-	Vomiting
Cons	-	Constipation
Diar	-	Diarrhea
SBP	-	Systolic Blood Pressure
Hb	-	Hemoglobin
Azo	-	Azotemia
Alb	-	Albumin
Tap	-	Peritonial Tap
Gas	-	Gas under diaphragm
HPE	-	Histopathology
Dehy	-	Dehydration
Dist	-	Distention
Tender	-	Tenderness
BS	-	Bowel Sounds
Coll	-	Collection
RS	-	Respiratory System

Deh - Dehiscence

Inf - Infection

Fist - Fistula

Reperf - Reperforation


The Tamil Nadu Dr.M.G.R.Medical ...
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A Study on Non Traumatic Ileal
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A DISSERTATION ON
"A STUDY ON NON TRAUMATIC ILEAL PERFORATION"
Dissertation submitted to
THE TAMIL NADU Dr.M.G.R.MEDICAL UNIVERISTY
CHENNAI
with partial fulfilment of the regulations
for the Award of the degree
M.S. (General Surgery)
Branch – I
INSTITUTE OF GENERAL SURGERY,
MADRAS MEDICAL COLLEGE ,
CHENNAI.
No Service Currently Active
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