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A STUDY OF NON – TRAUMATIC ILEAL PERFORATION

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled “A STUDY OF NON – TRAUMATIC ILEAL PERFORATION” submitted by Dr. A.N. Gurumoorthy to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S Degree Branch – I (General Surgery) is a bonafide research work were carried out under his direct supervision & guidance.

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I Dr. A.N. Gurumoorthy declare that, I carried out this work on, “A STUDY OF NON – TRAUMATIC ILEAL PERFORATION” at the Department of Surgery, Govt. Rajaji Hospital during the period of October 2008 to September 2009. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, diploma to any other University, Board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S degree examination in General Surgery.

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INTRODUCTION

In tropical countries like India non traumatic ileal perforation due to enteric fever perforations still occupy a high place in the list of gastrointestinal tract perforations. Typhoid fever is endemic in South India especially among the poor. Next common is tuberculous ileal perforation excluding the vascular causes like bowel gangrene of various etiologies. Perforation due to neoplastic conditions like lymphoma are very rare. Forty five patients were taken up for the study in whom the perforation were established by radiology or laparotomy and the disease confirmed by HPE or blood culture or widal reaction.

Ileal perforations continue to have a high mortality and morbidity rates in spite of extensive efforts made to formulate a more satisfactory outline of management. The management of typhoid or tuberculosis have been a subject of controversy. Surgery has been generally accepted as the treatment of choice but the purpose of surgery and procedure of choice continues to be debated.

REVIEW OF LITERATURE
In the year 1967, at a laparotomy for a suspected duodenal ulcer perforation. Kuruvilla found in the terminal ileum, a solitary perforation. They took specimens at the edges of the ulcer for histopathological examination and closed the lesion. Fortunately all went well. The widal test was positive and the pathologist reported that histopathological examination appearances were compatible with typhoid ulcer.

The early results of operative treatment were uniformly bad and this led to Huckstep’s conservative management of enteric perforation which enjoyed a brief popularity in early sixties. The pendulum has swung back in favour of surgical treatment and besides the standard procedure of simple closure, a number of newer operations are being advocated currently.

Abdominal tuberculosis is one of the commonest forms of extra pulmonary tuberculosis. An early reference to probably intestinal tuberculosis was made in 1643 when an autopsy on King Louis XIII show ulcerative intestinal lesions associated with a large pulmonary cavity.
Gastrointestinal tuberculosis is common in India (S.S. Minhas 1992, Chuttani 1970). 5 – 7% of intestinal perforations have been associated with intestinal tuberculosis. (Bhanasali SK 1967).

Role of vasculities in the natural history of abdominal tuberculosis evaluation by mesenteric angiography was carried by Piriti Sha 1991.

The relation of tuberculosis in AIDS patients were reported by shaver RM Brooklyn, Soriano V, Barcelona 1991. Evaluation of enzyme linked immunosorbant assay using mycobacterium saline extracted antigen for the serodiagnosis of abdominal tuberculosis was carried out by Bargava DK 1902. A change in acute phase proteins in patients with abdominal tuberculosis was studies by Emmanuel C 1990.
AIM OF THE STUDY

1. To determine the common causes of non traumatic ileal perforation
2. To find out the age, sex and seasonal incidence of non traumatic ileal perforation.
3. To assess the adverse prognostic determinates of ileal perforation.

ANATOMY OF THE ILEUM
The ileum is the continuation of jejunum about 2.4 metres away from duodeno jejunal flexure and continues for about 3.7 metres as a mobile bowel end to the caecum, where the mobility of the intestine is again restricted. There is no morphological demarcation between the jejunum and ileum. The ileum is entirely covered with visceral peritoneum except for a narrow strip at its mesenteric border. It is thinner than jejunum and having only few or no circular folds.

In the ileum there are numerous lymph nodules, which are present in the last 2 -10 cms. These form granular patches payer’s patches in the mucosa along the antimesentric border. The ileum is attached to the posterior abdominal wall by root of mesentry, which consists of two layers of peritoneum between which the ileal branches of Superior mesenteric artery enter the mesentry and form a series of arterial arcades. Four or five arcades are produced, and the branches are accompanied by corresponding veins, nerve plexus and lymph vessels, the mesenteric lymph nodes, connective tissue and fat.

**HISTOLOGY**
The serosa of the ileum is formed by peritoneum, which covers the gut completely except along the mesenteric border. The muscle coat is having two layers viz, outer longitudinal layer and inner circular layer. Submucosa is made of loose areolar tissue in which blood vessels and nerves ramify in it. Inner most layer is mucosa containing intestinal glands, villi and lymph follicles.

There are two types of lymphatic tissue in ileum viz., solitary and aggregated. The solitary lymph follicles are scattered all over the mucosa, whereas aggregated lymph follicles occupy the antimesentric border in a lengthwise manner. They are about thirty in number and are larger in the lower part of the ileum. They swell during entric fever. Since they are in the antimesentric border, they perforate the gut, contents of intestine will pass into the peritoneal cavity and set up peritonitis.

ETIO PATHOGENESIS AND PATHOLOGY

TYPHOID:
Typhoid fever is caused by ingestion of Salmonella typhi contaminated water and food and is a systemic infection. The contamination occurs due to infected stools or urine. They use to invade the intestinal lymphatics and mesenteric nodes, thus reach the blood stream. Once the bactremia is established. It leads to the development of secondary areas of inflammation in the liver, gall bladder, spleen and marrow. After one week the bacteria are shed into the small bowel and therefore appears in the stool.

The early change is the hyperplasia of the lymph follicles. The Payer’s patches become swollen and ulcerated. Which can progress to capillary thrombosis and subsequent necrosis. In the second week necrosis and sloughing occurs and ulceration of the follicles leading on to perforation in the third week of disease.

PATHOLOGY:

The organisms cause enlargement of reticulo endothelial and lymphoid tissue throughout the body. Proliferation of phagocytes swells the lymphatic submucosal nodules of the entire gut, mainly Payer’s patches of the terminal ileum. These become sharply delineated plateau like elevations upto 8 mm in diameter bulging into the intestinal lumen. During second week, the mucosa over the
swollen ileal lymphoid tissue is shed, resulting in oval ulcers with their long axis in the longitudinal axis of the bowel.

Bleeding from typhoid ulcers is usually scanty, but it can sometimes become uncontrollable, which has been the cause of fatalities. Once passed the peak of the disease, the ulcers heal slowly and lymphatic structures amazingly regenerate without scarring. Histologically there is accumulation of mononuclear phagocytes, which form nodular aggregates filled with red cells and nuclear debris. Intermingled with phagocytes, there are lymphocytes and plasma cells, but granulocytes are scarce and congregate mainly near the ulcerated surface of Peyer’s patches.

Perforation is due to the result of rupture of necrotic Peyer’s patches, caused by distention of bowel or by excessive peristalsis. The spleen is enlarged and soft. Microscopically marked histiocytosis and reticulo endothelial proliferation. Sometimes spleen may rupture. The liver shows scattered foci of parenchymal necrosis in which hepatocyte is replaced by phagocytic mononuclear aggregate called as ‘Typhoid Nodule’ which can also occur in bone marrow. Gall bladder colonization produces a carrier state often requires cholecystectomy to
eliminate bacterial shedding.

**ABDOMINAL TUBERCULOSIS**

The causative organism, mycobacterium tuberculosis was discovered by Koch 1882. It is a rod shaped, non-sporing, gram positive, acid fast organism. There are many types 1. Human 2. Bovine 3. Avian 4. Urine 5. cold blooded. But only human and bovine stains are known to produce lesions in human being.

Intestinal tuberculosis was thought to be the result of infection with bovine strain in USA and UK when unpasteurised milk was being consumed.

The commonest sites of involvement in abdominal TB are terminal ileum and ileoacaeal regions followed by jejunum and colon (VK Kapoor LK Sharma K 1998). Approximately 85% of the lesions are located in the ileocecal region (Boyd 1961).

The focus of infection to occur is in areas of

1. Increased physiological stasis
2. Regions with more lymphoid tissues
3. Areas of increased rate of absorption
4. In areas where the small bowel contents are more completely digested permitting closer contact with acid bacilli with the mucosal surface.

Common sites involved is terminal ileum where slow peristalsis favours a prolonged period of close contact between infected gut contents and ileal mucosa (HS Shukla 1986).

The bacilli become localized in the depths of mucosal glands and initiates an inflammatory reaction, the bacilli is then carried from the epithelial layer by phagocytes to submucosa where the initial characteristic lesions form in the lymph follicles of peyers patches.

As a result of mucosal swelling and mucosal end arteritis leading to mucosal oedema, sloughing ensues and ragged ulcer is formed which is shallow with undermined edges, the base of the ulcer may be formed by the submucosa, the mucularis or the serosa.

In the process of ulcer healing there is accumulation of collagenous tissue which subsequently contracts and may cause intestinal narrowing. The tubercle bacilli are carried along the lymph channels to invade the mesenteric nodes. These nodes show complete range of changes from hyperplasia to caseation necrosis and
calcification producing the pathological lesion called the “Tabes mesenterica”.

**Histopathology:**

The histopathological pattern in human tuberculosis is apparently the result of relationship between the virulence of the organism and degree of immunity and the allergic reaction of the host. Polymorphonuclear leucocytes are the first to arrive in response to invasion by tuberculous bacilli and this response is much more active if the patient is previously infected.

**Routes of infections of Gastrointestinal tuberculosis:**

GIT tuberculosis is divided into primary and secondary forms. It is called primary form, when there is no focus of tuberculosis seen anywhere else in the body and secondary from when there is established lesion found in lungs or elsewhere in the body. There are possible routes.

**Direct contact:**

a) Most of the investigators believed that the greater number of intestinal tuberculosis is due to direct contact between the mucosa and contaminated intestinal contents.
b) Consumption of infected milk from tuberculous cattle, in western countries, or ingestion of infected food from human sputum as a probable cause of primary intestinal tuberculosis.

c) The serial section studies of involved intestine by Gardner (1928) showed that the initial lesions were at the tip of the lymphoid follicles.

2. Enterogenous – direct swallowing of infected sputum (Gardner 1930 KL Wig and BNTandon).

3. Hematogenous – blood born infection from a distance focus through mesenteric arteries (patterson 1920 and KLWig BN Tandon)

4. Lymphatic and direct spread.

5. Intestinal involvement can occur from infected female genitalia (cullen JH 1940).

6. Peritoneum is infected from intestinal lesions, female genitalia, mesenteric nodes tuberculosis, and from lungs and pleura through the lymphatics passing through the diaphragm and from bronchial lymphnodes.

**DIAGNOSIS**

**SIGNS AND SYMPTOMS:**
In making diagnosis and deciding the criteria for inclusion in the study, great emphasis has been given to clinical signs and symptoms. The symptoms of headache, fever, vomiting and abdominal pain and the signs of abdominal tenderness, guarding and rigidity, distension, absent intestinal sounds, presence of free fluid and obliteration of hepatic dullness were considered most important.

According to Franklin (1963) the most prominent clinical features were the prolonged debilitating feverishness with diarrhea and on examination, generalized abdominal tenderness with rebound tenderness were more marked on the right side of abdomen.

In typhoid perforation the classical test like widal and blood culture are of little value in preoperative diagnosis as the results are obtained only after few days. Therefore history of fever and physical examination with signs and symptoms suggestive of perforation assume importance.

ERYTHROCYTE SEDIMENTATION RATE

E.S.R. is a non-specific reaction like leukocytosis and is a general manifestation of disease or tissue destruction. It is therefore, not of specific diagnostic value in tuberculosis. The rate reflects only
the degree of activity of the infections process. It is an useful
prognostic test.

WHITE CELL COUNT:

In general leucocytosis is a feature of peritonitis but typhoid
fever is associated with leucopenia. Hence in the enteric fever
perforation there is either high normal or leucocytosis is seen. Saphaha
et al (1970) recorded leucocytosis of over 15,000 in 16% of cases in
their series. Leucocyte count is having no significant value in Typhoid
perforation.

An average count of 8000 to 10,000 leucocytes with moderate
rise of lymphocytic count may be observed.

SPUTUM

Sputum for tuberculous bacilli examination is significant review
of aetiological relationship of swallowed positive sputum to intestinal
tuberculosis.

WIDAL TEST

After an attack of typhoid fever, antibodies appear as early as
fifth day viz., H-antigen (lg G) and O-antigen (lg M) agglutinating
antibodies. The antibody levels rise gradually reaches the maximum in
second to third week. These H and O agglutinins can be estimated by Widal test. The rising titre of agglutinin against s.Typhi H&O Ag (More than 1:200 dilutions) in patient’s serum is considered as positive.

The widal reaction was more often positive than cultures. Since the antibodies develop in the second week onwards, Widal reaction give better results when compared to blood culture. At the same time administration of Ciprofloxacin prior to appearance of agglutinins has a profound effect on antibody response. In typhoid fever cases prone to develop perforation also have an increased circulating cortisol levels which also lowers the antibody production.

Chauhan et al obtained positive widal reaction in 70.1% cases. Kaul (1975) showed a positive widal test in only 33% cases. Hence the widal test is to be taken up as a supportive and not the diagnostic test. Saphaha(1970) recorded a positivity in 80% cases.

Shah et al obtained negative widal reaction in 92% cases and high mortality in their series. Here the cases with overwhelming infection after perforation, which depresses the reticulo endothelial activity and therefore Widal test may be negative. However Gandhi
(1975) has recorded cent percent positive Widal reaction in 45 cases of Typhoid perforation.

BLOOD CULTURE:

Most of the blood cultures in typhoid fever are positive only in the first week of illness. Moreover many a cases of fever are treated with antibiotics, sometimes including cotrimoxazole and ciprofloxacin. But we come across typhoid perforations most commonly in the second or third week. At that time the blood cultures are less sensitive when compare to the first week. Kaul (1975) noted no positive culture in a study of nine cases.

RADIOLOGICAL DIAGNOSIS:

Abdominal erect view X-rays are taken up to find out pneumoperitoneum in enteric fever perforation cases. The incidence of pneumoperitoneum is varying from one series to other. Pneumoperitoneum was demonstrated in 44 out of 62 cases in whom the abdominal x-rays were taken. The percentage of pneumoperitoneum varies from 45-95% cases.

CT SCAN:

Can identify ileal perforation. Also useful in diagnosis to find

1. Diffuse lymphadenopahty
2. Low density lymph node with multilocular appearance following intravenous contrast.

3. High density ascitis.

4. Mottled low density masses in the omentum thickening of the bowel wall adjacent to mesentery.

5. Hepatic pseudo tumors.

ULTRASONOGRAPHY

May be useful in making diagnosis in abdominal tuberculosis with enlarged lymphnodes in pancreatic, mesenteric, perivascular and hepatic pedicle area (Beliaiche 1986). Hypoechoeic area in splenic tuberculosis (Pedro Botet 1991). Mass lesion and omental adhesions and ultrasonographic guided needle biopsy may give a compliment to the diagnosis.

HISTOPATHOLOGICAL EXAMINATION:

The specimens of the edges of the ulcer for histopathological examination, showed the appearances, compatible with typhoid fever (Kuruvilla 19780). Histological study reveals areas of necrosis with surrounding plasma cells, lymphocytes, macrophages containing abundant cytoplasm with bacteria and red cells termed as Typhoid cells and monocytes. Biopsy of the ulcer edge was performed in 15 cases.
and evidence of typhoid inflammation was shown by chronic non-specific enteritis and mononuclear infiltration.

100% accuracy in needle biopsy of peritoneum (Levine 1968). Culture of AFB in Lowenstein Jenson medium takes about 6 – 8 weeks.

ETIOLOGICAL FACTORS:

AGE INCIDENCE:

In general, age range for typhoid perforation was 2 to 80 years (E.G. Archampong 1976). But most of the typhoid perforation occurs in the second and third decade of life (Kuruvilla 1978). The age range was 8-57 years. Most perforations occurred in the age group 10-19 followed by 20-29 years age of group. In venkataramani Seetharam series the youngest patient was 13 years and the oldest was 65 years. 60.5% of their patients were in the second and third decades of life.

In TB perforation, the early adulthood 11-20 years is the commonest age of involvement and between 21-30 years is the most affected group.

SEX INCIDENCE:

Typhoid ideal perforation occurs more commonly in male than female population. M.J. Kurvillia showed the total of 31 patients with
perforation, of which 25 were male (9). In Venkataramani (1990) series there were 106 males out of 124 cases of perforation. Forrest et al obtained 62 male cases in 85 perforation cases. The male to female ratio is 2:4:1 in Kim series, 9:1 in Gandhi et al and 2:3:1 in Archampong series (1976). The reason for the male preponderance is not known, although it is possible that men have an increased risk of exposure to typhoid fever.

Females are more often attached than males (VK Kapoor and LK Sharma 1988). In India the disease has a higher incidence in female (Wig 1961 Tandon and Prakash (1972). Many affected females had history of contact with open cases of pulmonary tuberculosis (Chutani 1971).

SEASONAL INCIDENCE:

Typhoid perforation occurs more common in summer or autumn (Kim et al 1975). In Swadia et al series (1978) there is increased incidence of typhoid perforation in summer as well as in autumn. No seasonal predilection seen with TB ileal perforation.

SITE OF PERFORATION:

Since the Peyer’s patches are more in the terminal ileum the incidence of typhoid / TB perforation is also more in the terminal
ileum. Kim (1975) obtained 86% cases of perforation occurred in the last 60cm of ileum. Of which 72% perforations were within the last 40cm. In Kuruvilla series (1978) perforations were confined to the last 30cm of the terminal ileum. Purohit et al, noted all the perforations occurring within 40cm from the ileocaecal junction.

GEOGRAPHICAL VARIANCE:

In Ghana successive studies over the past ten years, have found a impressive increase in the incidence of perforation from 15% reported by Badoe 1966 to 35% by Archampong (1974). Ikyrub et al (1972) obtained the phenomenal incidence of 33.6% from the same centre. The high incidence of typhoid perforation in the west Africa has yet to be explained. In other parts of Africa although typhoid fever is commonly encountered, this complication is seldom seen.

TB ileal perforation is more common in the Indian subcontinent where as the diseases and its complications are less common in the western world. The incidence has increased in the last few decades with the prevalence of AIDS.
**TREATMENT AND COMPLICATIONS**

In general ileal perforation is a surgical emergency and the treatment should be prompt and energetic. The role of conservative treatment is limited. The surgical procedure depends upon the general health of the patient and the extent of the ileum involved. The mortality in active intervention is low, so does undue delay.

In 1960 R.L. Hucktep advocated a non surgical approach for typhoid perforation. He proposed management of typhoid perforation on the lines similar to Norman Taylor regimen. In 1963 Li Frankin raised the following objections against the rationale of conservative management.
1. Although ileum is oedematous and friable if handled gently, the stitches do hold.

2. With adequate gastrointestinal suction with specific antibiotic therapy further perforation is unlikely.

3. Evacuation of pus from the peritoneal cavity reduces the toxaemia and enhances the recovery of the patient. The patient is ill to tolerate the surgery, but he is too ill to fight continuing toxemia from the presence of pus in the peritoneal cavity.

4. As observed by Wofford et al (1960) commonly, there is nosuggestion of localization of the inflammation, the perforation is usually not sealed by omentum spontaneously and peritionitis is almost always generalized.

**MANAGEMENT OF PERFORATION:**

Patient was put on naso gastric aspiration while intravenous fluids and drugs were administered parenterally. Electrolytes and fluid balance are meticulously maintained. Any substantial drop in the hemoglobin level was combated by prompt blood transfusion. Care was taken to ensure good urinary output. The appropriate operative procedure was decided at the time of laparotomy and depended upon the general condition of the patient and state of the ileum.
SIMPLE CLOSURE OF PERFORATION:

The standard surgical management consists of simple closure of perforation. Laparotomy was done and bowel loop bearing perforation is sought out. The perforation is closed with atraumatic needle in two layers using 3-0 vicryl for full thickness and 3-0 silk for sero muscular layers. After thorough peritoneal lavage, peritoneal cavity is mopped and tube drain placed in the pelvis and hepato renal pouch. Abdomen is closed in layers.

Chauhan et al (1985) treated 138 cases of typhoid perforation surgically. In these patients, the principal operation was closure of the perforation and peritoneal drainage. The overall mortality of surgical treatment was 58.7%. Solitary perforations were treated by simple closure in several patients of Kuruvilla et al (1978).

Where there were only one or two perforations simple closure had the advantage of being quick and easy. (Archampong) (1976) noted that the more insidious the perforation, the greater are the bowel inflammation and oedema at the time of perforation, causing greater friability and increased difficulty in handling and suturing the bowel during surgery.
After vigorous resuscitation, simple closure of the uncomplicated solitary perforation and peritoneal lavage will suffice for most of the cases. Of simple closure, double layer closure of perforation lowered the mortality rate in comparison with single layer closure. Forrest et al (1981) treated forty three patients of typhoid perforation with simple closure.

In Swadis (1978) study of 112 cases, 104 cases were treated by simple closure with only two cases of developing post operative leakage and fistula. Thus the incidence of leakage was quite low and possibility of reperforation or leakage after simple closure have been unduly exaggerated. Hence uncomplicated solitary perforation without external evidence of other ulcers was treated by simple closure without undue tension.

The operative procedure depended on general condition of the patient and on surgeon’s preference. But kim (1975) treated 103 cases with simple closure and had mortality of only 10% Whereas Purohit et al (1976) treated ten patients of typhoid perforation by simple closure after trimming the edge and had cent percent success and claims it to be the ideal operation with the following reasons.

1. Quick and easy
2. Satisfies requirement of wound healing
3. Avoids inverting second layer stitches which often cut through the friable ileal wall.
4. Facilitates confirmation of diagnosis by biopsy of trimmed tissues.

The average mortality of about 25% in Typhoid ileal perforation treated by simple closure led various authors to device various techniques to overcome the high mortality in an attempt to find out on ideal operation which would prevent re-perforation and other complications.

Why Have alternative procedures been thought of at all?

The reason is surgeons are still striving hard to reduce further mortality of enteric perforation. The main argument in favor of larger magnitude of surgery stems from the possibility of postoperative leakage from the sutured site after simple closure or re-perforation (Welch 1975). Such a mishap is supposed to be due to friable ileal wall and hence for the resection of affected segment itself.

**ALTERNATIVE OPERATIVE PROCEDURES:**

- Resection of the affected segment with an end to end anastomosis.
Simple closure after excision of seromuscular layer surrounding the perforation (Purohit 1978).

RESECTION OF THE MOST OF THE AFFECTED SEGMENT WITH END TO END ANASTOMOSIS:

In typhoid ileal perforation, where there were multiple perforations or multiple ulcers or where ileal segment looked unhealthy or where perforation was accompanied by hemorrhage resection becomes the operation of choice. To start with, the veins were tied off and the segment intended for resection was clamped. Manipulation was kept to the minimum and care was taken to see that both sides of anastomotic site had adequate blood supply. Its advantages are:

1. Resection prevents re-perforation
2. It also prevents further perforation of nearby ulcer.

M.J. Kuruvilla (1978) treated 11 cases of their series with of anastomosis. Mulligan et al (1972) write ‘at operation, resection seems rationale on feature to do more than merely repair the perforation. Kim
(1975) obtained lower mortality in his series treated by resection when compared to simple closure.

**SIMPLE CLOSURE AFTER TRAJECTING THE EDGES OF PERFORATION:**

It consists of simple closure of perforation generally after refreshing the edges combined with or without oversewing areas of apparent impending perforation. Purohit treated 10 patients of perforated ileum by simple closure after trimming the edges and had cent percent success rate. In forest (1981) series, 43 patients were treated by simple closure after refreshing the edges combined with oversewing the areas of impending perforation. But their series showed 30.2% of mortality rate.

**BLIND DRAINAGE;**

It is nothing but bilateral flank drainage under local anesthesia without laparotomy which could not be performed because of very poor general condition of the patient. Who could not tolerate the great surgical manoeuvre. Kuruvilla M.J. (1978) treated three cases with
blind drainage because of poor general condition, of which two patients died in two days after drainage.

Blind drainage may help those patients who are too ill to withstand laparotomy. In most of the series, blind drainage procedure claimed a 100% mortality rate. Kala et al (1978) and Badoe. E. Series obtained 60% and 100% mortality rate respectively with blind drainage. Kim et al of Korea showed 20% of mortality rate in their study with blind drainage.

All cases of TB ileal perforation were started on category I ATT after confirmation with HPE and complete recovery from surgery.

All cases are enteric fever ileal perforation were given Inj Ceftriaxone for 5 days followed by Tab. Ciprofloxacin for 2 weeks.

**ANAESTHESIA FOR SURGICAL PROCEDURES OF ILEAL PERFORATION:**

In most of the cases of typhoid perforation, spinal anaesthesia is preferred for surgical treatment. Those with hypotension or those who needed only blind drainage because of poor general condition, we can use local anaesthesia. In few cases with otherwise good health, general anaesthesia is used.
COMPLICATIONS OF SURGERY IN ILEAL PERFORATION:

Various complications can occur following surgical treatment in ileal perforation Viz.

EARLY COMPLICATIONS:

Toxaemia, Respiratory infections, Paralytic ileus, Thrombophlebitis, Transfusion reaction, Uraemia, Meningism, Acute parotitis, Cardiac arrest, Reperforation.

LATE COMPLICATIONS:

Wound infection, Burst abdomen, Faecal fistula, Decubitus ulcer, Intestinal obstruction, incisional hernia.

Apart from the wound infection, the most common abdominal complication is wound dehiscence occurring in upto 30% (Shah-1967). This is the reflection of both the high incidence of infection and debility of the patient. As like as wound dehiscence, the development of faecal fistula is catastrophic. This may be the result of reperforation, perforation in another area or the results of suture line break down. Its incidence varies from 4% to 21% . It has been reported in every type of surgery.
MATERIALS AND METHODS

From Sep 2008 to Aug 2009 45 patients underwent surgery with a clinical diagnosis of nontraumatic perforation of the ileum. The diagnosis was confirmed in these cases either by blood culture or widal reaction or biopsy. Routine investigations like Hb%, Haematocrit value, Blood urea, Serum electrolytes, Plain x-ray abdomen in erect posture were done. Biopsy were taken in all patients to confirm the diagnosis for the cause of perforation, ultimately the line of management for all these cases were noted down and followed up post operatively for any complication.

Exclusion criteria:

Ileal perforations secondary to bowel gangrene of various etiologies like strangulated hernia, volvulus, necrotising enteritis, mesentric ischemia etc. were excluded from the study

SURGICAL METHODS ADOPTED
• Simple closure of the perforation in 35 cases

• Resection of affected segment of bowel and end to end anastomosis in 6 case.

• Blind drainage in 4 cases.

**CLINICAL DATA**

**AN OBSERVATION:**

45 cases of non traumatic ileal perforations were taken up for study. Ileal perforation secondary to vascular causes like mesentric vascular occulusion, strangulated hernia, volvulus were excluded from the study. All cases of ileal perforations were treated surgically by various above said techniques. Thorough clinical examinations were done in all the 45 cases. Typhoid perforation has occurred in 57.78% male, and in 23.40% female with ratio of 2.5 : 1. Tuberculous perforation has occurred in 15.55% male, and in 2.23% female with ratio of 7:1. (Table-I)

In this study, most cases belong to second and third decade. The youngest patient in the series was 13yrs. and the oldest was 77years. 50% of the patients were in the second and third decades of life. In venkataramani series (1990), it was 60.5%. (Table-I)
The symptom complex of prodromal fever, headache, vomiting, abdominal pain, distension, constipation, with tenderness and varying degree of guarding and rigidity were noticed. The incidence and duration of these symptoms in each patient were recorded. Abdominal pain was the most prominent symptom in 95.55% cases as like as in other series. Like this bowel sounds were absent in 86.66% cases and fever present in 86.68% cases. (Table – III)

Among the signs, abdominal tenderness was present in 84.44% patients and guarding in 80% cases. The association of intestinal obstruction with protracted fever should always alert the clinician to the possibility of Typhoid, but even this reliable feature may be absent.

The diagnosis was more difficult however in two categories of patient, a small group of patient in whom perforation occurred while under medical treatment and patients with protracted illness reaching several days after perforation. In the former group the signs of perforation were in abeyance because the complication developed insidiously. In the latter group gross abdominal distension over shadowed the other signs of perforation. 3 out of 8 patients TB ileal perforation were known cases of pulmonary TB who had irregular anti tuberculous therapy previously.
WHITE CELL COUNT:

Leucopenia was seen in 25% cases. The white cell was within normal limits 13.88 in cases. But total leucocyte count towards higher side of normal in 20 cases (55.55) and actually had leucotytois in 5.5% cases. (Table - IV)

BLOOD CULTURE:

The blood culture was performed in 25 cases and was positive salmonella typhi only in 28% cases. (Table - VI)

WIDAL REACTION:

The widal reaction was more often positive than blood cultures. (17) The diagnosis was confirmed by Widal reaction in 89.19% cases. (Table - VI)

RADIOLOGICAL DIAGNOSIS:

Abdominal erect view x rays were taken up in all cases. Pneumoperitoneum was confirmed in 73.33% cases.

HISTOPATHOLOGICAL EXAMINATION:
Biopsy of the edges of the typhoid fever perforation was performed in all cases. The diagnosis was confirmatory in TB ileal perforation in all cases. (Table - VII)

AETIOLOGICAL FACTORS

SEX INCIDENCE:

It occurs more common in male as in other series with 73.34% male and 26.67% female. (Table – I)

AGE INCIDENCE:

In this series, age range for typhoid perforation was between 13 and seventy seven years and between 22yrs & 60yrs for TB perforation. Most of the perforations occurred in the second and third decade with 42.22. (Table - I)

SEASONAL INCIDENCE:

Typhoid fever occurs more commonly in summer or autumn. No seasonal predilection noted in TB perforation. (Table – II)

SITE OF PERFORATION:

From the ileo caecal junction perforation were confined to the last 25 cm in 53.33% cases and between 25 and 50 cm in 40%, and >50cm in 6.66% of cases. (Table - VII)
FEVER – PERFORATION INTESTINAL:

In this study, in most of the cases, perforation occurred early in the course of the disease and it has been recognized by others. 88.88% patients who perforate during the first two weeks. Of illness appear to have a better prognosis in typhoid ileal perforation. (Table – XIV)

TREATMENT:

Various surgical methods were adopted for the present series.

SIMPLE CLOSURE OF THE PERFORATION

Since the perforations were solitary one in 75.56 patients the principal operation was closure of the perforation and a flank drainage. Following simple closure 10 patient out of 35 perforation patients died, due to various complications showing the mortality rate of 28.55%. (Table - XI)

RESECTION AND END – END ANASTOMOSIS:

Resection of the affected segment of ileum was performed in 4.4% cases of typhoid perforation in which there were multiple perforation as well as multiple ulcers with 50% mortality. (Table - XI)

BLIND DRAINAGE:

Because of poor general condition, bilateral flank drainage was performed under local anesthesia in 8.8% cases. who would not
tolerate great surgical monoeuvre. In this series, the mortality rate with blind drainage was 50% (Table - XI)

**POST OPERATIVE COMPLICATION:**

In the present series, wound sepsis is the most common complication in 55.6% cases whereas the post-operative period was uneventful in 31.32% cases. Other complications like toxaemia and paralytic ileus were developed in 24.4% and 17.7% cases respectively. (Table - IX)

Our observations contradict Forrest’s view that wound dehiscence occurring in 8.81 % cases, whereas in Forrest et al series, wound dehiscence was occurring in upto 30% so also in Shah (1967) et al series.

The incidence of faecal fistula was 6.6% approaching to Swadia (1978) series. Its incidence was high upto 21% in Olurin series and Forrest series. The high fistula rate is the result of both poor wound healing and the nature of the lesion in the intestine. Other complications included in this series are transfusion reaction- Nil, Thrombophlebitis (6.6%), Pelvic abscess (6.6%), and respiratory complications(11.1%)(Table - IX)
LATE COMPLICATION:

Only one case developed intestinal obstruction (2.2%) six months after simple closure of perforation. (Table - IX) That patient underwent laparotomy again and the adhesions were released.

MORTALITY RATE:

The overall mortality rate of Typhoid perforation in the present series was 32.43%. of the TB perforation in this series was 25%. Among the post operative deaths, the mortality rate following treatment with various surgical techniques was 29.76% and it was 50% with blind drainage. (Table - XI)
### RESULTS

#### TABLE I

<table>
<thead>
<tr>
<th>Age group</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
<th>Typhoid</th>
<th>Total</th>
<th>Tuberculosis</th>
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<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>0-10</td>
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<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<td>11-20</td>
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<td>51-60</td>
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<td>61-70</td>
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<td>71-80</td>
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<td>2.22</td>
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<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>33</td>
<td>73.34</td>
<td>12</td>
<td>26.67</td>
<td>45</td>
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<th>Month</th>
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<th>Death</th>
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</tr>
<tr>
<td>OCTOBER 2008</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>NOVEMBER 2008</td>
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<td>DECEMBER 2008</td>
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<td>2</td>
</tr>
<tr>
<td>JANUARY 2009</td>
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<tr>
<td>FEBRUARY 2009</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>MARCH 2008</td>
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<td>1</td>
</tr>
<tr>
<td>APRIL 2009</td>
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<td>0</td>
</tr>
<tr>
<td>MAY 2009</td>
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<tr>
<td>JUNE 2009</td>
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<td>2</td>
</tr>
<tr>
<td>JULY 2009</td>
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<td>1</td>
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### TABLE III
**SIGNS & SYMPTOMS**

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<th>No.</th>
<th>Clinical Features</th>
<th>Number of Cases</th>
<th>Percentage</th>
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<td><strong>Symptoms</strong></td>
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<tr>
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<td>Fever</td>
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<td>86.68</td>
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<td>Abdominal Pain</td>
<td>43</td>
<td>95.55</td>
</tr>
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<td>Head Ache</td>
<td>25</td>
<td>55.55</td>
</tr>
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<td>4</td>
<td>Vomiting</td>
<td>15</td>
<td>33.33</td>
</tr>
<tr>
<td>5</td>
<td>Distension</td>
<td>35</td>
<td>77.77</td>
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<td>6</td>
<td>Bowel Habits</td>
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<td></td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>-</td>
<td>-</td>
</tr>
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<td></td>
<td>Constipation</td>
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<td>73.33</td>
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<td></td>
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<td>7</td>
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<td>6</td>
<td>13.33</td>
</tr>
<tr>
<td>8</td>
<td>Anuria</td>
<td>2</td>
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<td></td>
<td><strong>SIGNS</strong></td>
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<td></td>
</tr>
<tr>
<td>9</td>
<td>Abdomen Tenderness</td>
<td>38</td>
<td>84.44</td>
</tr>
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<td>10</td>
<td>Distension</td>
<td>33</td>
<td>73.33</td>
</tr>
<tr>
<td>11</td>
<td>Guarding</td>
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<td>73.33</td>
</tr>
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<td>12</td>
<td>Rigidity</td>
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<tr>
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<td>Free Fluid</td>
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</tr>
<tr>
<td>14</td>
<td>Obliteration of Liver</td>
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<tr>
<td></td>
<td>Dullness</td>
<td></td>
<td></td>
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<tr>
<td>15</td>
<td>Bowel Sounds</td>
<td></td>
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<td>Normal</td>
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<td>Absent</td>
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<td>86.66</td>
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<td>Increased</td>
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### TABLE – IV

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<th>S.No.</th>
<th>Laboratory Investigation</th>
<th>No.of patients</th>
<th>Percentage</th>
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<tr>
<td>1</td>
<td>Leuko paenia (&lt;4000/cumm)</td>
<td>9</td>
<td>25.00</td>
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<td>2</td>
<td>Normal Total Leucocytes Count (4000 – 7000/cumm)</td>
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<td>13.88</td>
</tr>
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<td>3</td>
<td>High Normal Total Leucocytes(7000-11,000/cumm)</td>
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<td>55.55</td>
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<td>4</td>
<td>Leucocytosis(&gt;11,000/cumm)</td>
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<td>5.55</td>
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### TABLE – V

<table>
<thead>
<tr>
<th>S.No.</th>
<th>Serum Potassium ion Level</th>
<th>No.of Cases Pre-operative</th>
<th>No. of Cases Post operative</th>
<th>Deaths</th>
<th>% of mortality %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Normal Level</td>
<td>22</td>
<td>36</td>
<td>6</td>
<td>16.67</td>
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<td>2</td>
<td>Low Level</td>
<td>23</td>
<td>9</td>
<td>8</td>
<td>88.89</td>
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### TABLE – VI

**SEROLOGICAL TESTS**

<table>
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<tr>
<th>S.No.</th>
<th>Name of the test</th>
<th>Positive Cases</th>
<th>Percentage</th>
<th>Negative Cases</th>
<th>Total</th>
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<tr>
<td>1</td>
<td>Widal Test</td>
<td>33</td>
<td>89.19</td>
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<td>37</td>
</tr>
<tr>
<td>2</td>
<td>Blood culture</td>
<td>7</td>
<td>28</td>
<td>18</td>
<td>25</td>
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### TABLE – VII

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<th>S.No.</th>
<th>Test</th>
<th>Total case</th>
<th>Positive Cases</th>
<th>%</th>
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<tr>
<td>1.</td>
<td>HPE</td>
<td>8</td>
<td>8</td>
<td>100</td>
</tr>
<tr>
<td>2.</td>
<td>Sputum</td>
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<td>2</td>
<td>25</td>
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### TABLE – VIII

**SITE AND NUMBER OF PERFORATION**

<table>
<thead>
<tr>
<th>Site of perforation from ileocaecal jn</th>
<th>No of cases</th>
<th>Death</th>
<th>%</th>
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</thead>
<tbody>
<tr>
<td>0 – 25 cm</td>
<td>23</td>
<td>8</td>
<td>34.78</td>
</tr>
<tr>
<td>25 - 50 cm</td>
<td>16</td>
<td>4</td>
<td>25</td>
</tr>
<tr>
<td>&gt; 50 cm</td>
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### TABLE IX

**COMPLICATIONS**

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<th>No.</th>
<th>Complications</th>
<th>No. of cases</th>
<th>Percentage</th>
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<tr>
<td>1</td>
<td>Sepsis</td>
<td>25</td>
<td>55.6</td>
</tr>
<tr>
<td>2</td>
<td>Wound dehiscence</td>
<td>4</td>
<td>8.8</td>
</tr>
<tr>
<td>3</td>
<td>Toxaemia</td>
<td>11</td>
<td>24.4</td>
</tr>
<tr>
<td>4</td>
<td>Paralytic ileus</td>
<td>8</td>
<td>17.7</td>
</tr>
<tr>
<td>5</td>
<td>Respiratory</td>
<td>5</td>
<td>11.1</td>
</tr>
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<td>6</td>
<td>Faecal fistula</td>
<td>3</td>
<td>6.6</td>
</tr>
<tr>
<td>7</td>
<td>Transfusion reaction</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>8</td>
<td>Thrombophlebitis</td>
<td>3</td>
<td>6.6</td>
</tr>
<tr>
<td>9</td>
<td>Pelvic abscess</td>
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<td>6.6</td>
</tr>
<tr>
<td>10</td>
<td>Encephalitis</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>11</td>
<td>No infection</td>
<td>20</td>
<td>44.4</td>
</tr>
<tr>
<td>12</td>
<td>Late complication</td>
<td></td>
<td></td>
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<td></td>
<td>Intestinal obstruction</td>
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<td>2.2</td>
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### TABLE – X

#### CAUSE OF DEATH

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<thead>
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<th>S.No.</th>
<th>Causes of Death</th>
<th>No. of Cases</th>
<th>Percentage</th>
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<td>1</td>
<td>Toxaemia</td>
<td>8</td>
<td>57.14</td>
</tr>
<tr>
<td>2</td>
<td>Respiratory Complication</td>
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<td>21.43</td>
</tr>
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<td>3</td>
<td>Faecal fistula</td>
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<td>4</td>
<td>Circulatory failure</td>
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<td>14.285</td>
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### TABLE XI

#### METHOD OF TREATMENT AND MORTALITY RATE

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<th>Method</th>
<th>Alive</th>
<th>Death</th>
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</thead>
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<tr>
<td></td>
<td>No.of Cases</td>
<td>%</td>
</tr>
<tr>
<td>1. Simple Closure</td>
<td>35</td>
<td>24</td>
</tr>
<tr>
<td>2. Resection&amp; Anastamosis</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>3. Flank drain</td>
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<td>2</td>
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<tr>
<td>Total</td>
<td>45</td>
<td>31</td>
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### TABLE XII

#### PROGNOSIS OF SURGERY Vs CONSERVATIVE MANAGEMENT

<table>
<thead>
<tr>
<th>Parameter</th>
<th>No. of Cases</th>
<th>Alive</th>
<th>Death</th>
<th>No. of Cases</th>
<th>Typhoid</th>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Alive</td>
<td>Death</td>
</tr>
<tr>
<td>Surgery</td>
<td>41</td>
<td>29</td>
<td>12</td>
<td>34</td>
<td>23</td>
<td>11</td>
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<td></td>
<td></td>
<td>70.73</td>
<td>29.68</td>
<td></td>
<td>67.64</td>
<td>32.35</td>
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<tr>
<td>Flank Drain</td>
<td>4</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50</td>
<td>50</td>
<td></td>
<td>66.67</td>
<td>33.33</td>
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### MORTALITY IN RELATION TO ONSET OF PERFORATION AND SURGERY INTERVAL

<table>
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<tr>
<th>S.No.</th>
<th>Perforation – surgery interval</th>
<th>Alive</th>
<th>Deaths</th>
<th>Total</th>
<th>Percentage</th>
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<tr>
<td>1.</td>
<td>24 hrs</td>
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<td>2.</td>
<td>24 – 48 hrs</td>
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<td>13</td>
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<td>3.</td>
<td>48 – 72 hrs</td>
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<td>20.00</td>
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<td>4.</td>
<td>72 – 96 hrs</td>
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<td>5.</td>
<td>Above 96 hrs</td>
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<td>5</td>
<td>8</td>
<td>62.50</td>
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### TABLE – XIV

**PATTERN OF TYPHOID FEVER COMPLICATION (ILEAL PERFORATION)**

<table>
<thead>
<tr>
<th>S.No.</th>
<th>Duration</th>
<th>No.of Cases</th>
<th>Death</th>
<th>% of total</th>
<th>% of mortality</th>
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<tbody>
<tr>
<td>1</td>
<td>0-5 Days</td>
<td>7</td>
<td>2</td>
<td>18.91</td>
<td>28.57</td>
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<td>2</td>
<td>6-10 Days</td>
<td>21</td>
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<td>56.75</td>
<td>23.80</td>
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<td>3</td>
<td>11-15 Days</td>
<td>6</td>
<td>3</td>
<td>16.22</td>
<td>50.00</td>
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<tr>
<td>4</td>
<td>&gt;2 Weeks</td>
<td>3</td>
<td>2</td>
<td>8.11</td>
<td>66.67</td>
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<td>37</td>
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### TABLE – XV

**NUMBER OF PERFORATION**

<table>
<thead>
<tr>
<th></th>
<th>No of cases</th>
<th>Death</th>
<th>%</th>
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<tbody>
<tr>
<td>Single</td>
<td>44</td>
<td>13</td>
<td>29.54</td>
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<tr>
<td>Multiple</td>
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<td>100</td>
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DISCUSSION

The present series have found typhoid ideal perforation occur in both sexes and in all age groups. As in other series this study also found it to be more common in men during second and third decades. The reason for this male predominance is yet to be identified although it is possible that men have an increased risk of exposure to typhoid fever.

In this study, in most of the cases, perforation occurred early in the course of the diseases and it has been recognized by others. Proof of typhoid was found in more than 80% cases.

The important investigations carried out to confirm the diagnosis are plain X ray abdomen erect view for evidence of pneumo peritoneum, Widal test, Blood culture and biopsy of the ileum at the site of perforation. Widal test was positive in 75.55% and was more useful in confirming the diagnosis than blood culture which was positive only in 36% cases.

Incidence of positive blood culture was low due to the fact that perforation occurred mostly in third week and is due to indiscriminate use of ciprofloxacin by Practitioners before confirmation of the diagnosis.
The management of typhoid perforation is still disputed. Huckstep advocated a conservative approach. (Albeit with certain exceptions). Hook Guerrans recommended surgery only if there was no localization. Rains and Ritchie felt the results of surgery to be poor to advise against it. However, the localization is unusual in most of the cases and the mortality reported from non-surgical management varies from 60-100%.

Typhoid perforation producing fulminating generalized peritonitis. Often frankly faecal in character. At laparotomy these perforations were rarely found sealed, nor has omentum migrated to the area. If facilities are available for surgery, it is better to go for surgical management rather than the conservative management.

This series showed that simple closure with ileo transverse colostomy has been encouraging with less morbidity and mortality. This study recommend, when patient’s general condition is good, simple closure alone is adequate in uncomplicated solitary perforation. But resection is necessary if there is bleeding or if multiple ulcers with more than one perforation are present in one segment. Blind drainage may be of help in those patients who are too ill to withstand the laparotomy.
Most of the ulcers in our patients were confined to the last 30cm of ileum.

Unfortunately no matter what procedure has been done, post operative morbidity has been extremely high. Other than wound infection, the most common abdominal complications is wound dehiscence in Forrest’s view, whereas in this study next to wound infection is paralytic ileus and toxaemia. Some of the post operative complication is beyond the control of the surgeon.

Another dangerous abdominal complication is wound dehiscence occurring in 8.8% cases. This is the reflection of both the high incidence of infection and debility of the patient. Just like wound dehiscence, the development of faecal fistula is catastrophic. This may be the result of reperforation, perforation in another area or the result of suture line break down. It has been reported in every type of operation. The incidence is 6.6% in the present series.

The most significant finding of the present study is the absence of any appreciable change in the mortality over the two year period. This is also the experience of other centres such as Mulligan (1972), Olurin (1976), Kuruvilla et al (1978), Chauhan (1982), Mock C.N. (1992). I have not seen any other acute intraabdominal lesion due to
typhoid, unlike Miguei, who recorded four gall bladder perforations in his series and Stuart who had reported two perforations of appendix. Fortunately I have never had intra abdominal abscess formation a complication reported to be more common in typhoid perforation.

**MORTALITY RATE AND METHOD OF TREATMENT**

The incidence of mortality rate following blind drainage was 50% which is higher when compared to perforation closure (31.43%) and resection anastomosis (16.67%)

**MORTALITY RATE IN RELATION TO ONSET OF PERFORATION AND SURGERY INTERVAL**

The mortality rate was absolutely nil in 5 patients in whom onset of perforation and surgery interval was less than 24 hours. Its incidence in 24-48 hours and 48-72 hours were 15.38% and 20% respectively. After 72 hours the mortality rate sharply rises.
MORTALITY RATE WITH PATTERN OF TYPHOID FEVER COMPLICATION:

In the present series, if the fever i.e. typhoid fever onset and perforation interval was more than two weeks, the incidence of perforation was increased. But the mortality rate was more than 60% where as it was 27.5% if the perforation and the onset of typhoid fever interval was less than two weeks. Patients who were perforated during the first two weeks of the illness appear to have a better prognosis.

MORTALITY RATE AND THE CAUSE OF DEATH:

The major cause of death appears to the toxaemia with 57.14%. The longer the perforation and surgery interval, the greater the toxaemia and higher the mortality rate. In the series next to the toxaemia, circulatory failure (14.29%) and respiratory complications (21.43%) and rest of the mortality by faecal fistula(7.14%).

MORTALITY AND SITE, NUMBER OF PERFORATION :

When the perforation was within the 25 cm of terminal ileum from ileo caecal junction, there was increased mortality of 34.78% whereas if the perforation was beyond 25 cm, the mortality was reduced and almost nil beyond 50cm.
It is also noted that although few patients had more than one perforation, who did poorly. Thus this study contraindicated Archampong’s view that multiple perforations do not adversely affect prognosis.

MORTALITY IN RELATION TO WATER AND ELECTROLYTE CHANGES:

The influence of water and electrolyte imbalance on prognosis was remarkable and provided a measure of the degree of transport disturbance across the bowel in this disease. In most patients all the major electrolytes were reduced.

In the present series, when serum potassium levels were low both in the preoperative as well as in the postoperative period, the mortality rate was absolutely quite high with 88.89%.

The estimated fluid requirements of 3.0 to 4.0 litres was based on the measurements of peritoneal exudates as in previous studies of Archampong (1969). The urinary output, which is always a good index of the degree of rehydration achieved in preoperative patients, reflected at the same level of significance the difference in the intravenous fluid therapy between survivors and non survivors. It is
suggested that operations on typhoid patients with perforation should be avoided until the urinary output is above the safety level of 30ml/hr.

In most of the patients, all the major plasma electrolytes were reduced. This is associated with ileus of enteric fever, which results in impaired absorption. It is also likely that the endotoxin of S. Typhi actively depresses the functions of the intestinal mucosa (Norris 1967; Edmonds 1971). The peritoneal exudates represents an important route of electrolytes loss and the remarkable loss of potassium, which is a correct explanation for close association of low plasma potassium with the prognosis (Archampong 1976, Badoe 1973)

The high incidence of Hypokalemia demonstrated in the present series and the low frequency of renal failure noted would, however suggest in many cases of typhoid perforation, potassium deficiency is a dire emergency.

Therefore the potassium could be included in the very first infusions e.g. as Ringer’s lactate solution, even before the full extent of renal function is ascertained. This is particularly important because the serum level provide only an approximate estimate of the total body or interstitial fluid potassium levels, which are particularly depressed in hypokalemic states.
SUMMARY

In the present series, 45 cases of non traumatic ileal perforations were taken up for study. Most of the cases are belonging to 3\textsuperscript{rd} and 4\textsuperscript{th}
decade (53.33%). Widal reaction was positive in 89.19% cases (33 cases) and blood culture was positive only in 28% (7 cases) of patients. 78.57% patients who perforate during the first two weeks of illness appear to have a better prognosis with the mortality rate of 27.50%.

Simple closure of the perforation was performed in 77.78% cases (35 cases) with 31.43% (11 cases) mortality rate. Resection and end to end anastomosis was carried out with 16.67% (6 cases) mortality rate. Perforations were confined to last 50 cm of terminal ileum in 93.33% (39 cases).

Blind drainage was done in 8.8% cases (4 cases) with poor general condition with 50% (2 cases) mortality. Wound sepsis was the most common complication in 55.6% cases (25 cases). One patient who developed intestinal obstruction after six months of perforation closure.

The overall mortality rate in this series was 31.11% (14 cases). In relation to the onset of perforation and surgery interval, the mortality rate sharply rises after 72 hours. If the perforation and fever interval was more than two weeks, the mortality rate increases. Toxaemia was the major cause of death in the post operative period.
When the serum potassium levels were low in the post operative period, the mortality rate was very high with 88.89%. (8 cases) when the perforation was within the 25cm ileo caecal junction in the terminal ileum (23 cases) there was high mortality of 34.78. (7 cases) If there were multiple perforations, the mortality rate was high.

CONCLUSION

1. From this study typhoid fever is the commonest cause of non traumatic ileal perforation, followed by tuberculous perforation
2. There appears to be a trend towards better prognosis in the patients who underwent surgery soon after they perforated irrespective of the cause.

3. Patients who perforate during the first two weeks of typhoid illness appear to have a better prognosis.

4. Resection is necessary if there are multiple perforations or associated strictures and has equally good prognosis as simple closure.

5. Blind drainage may help in those patients, who are too ill to withstand major surgical procedure.

6. There is a close association of low plasma potassium with the prognosis.

In conclusion, bearing in mind that all patient with non traumatic ileal perforation excluding vascular causes of various etiology, admitted to this hospital are included in this study, the final survival figure of 68.88% is very gratifying.

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# PROFORMA

## A STUDY OF NON – TRAUMATIC ILEAL PERFORATION

<table>
<thead>
<tr>
<th>NAME</th>
<th>AGE</th>
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<table>
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Time interval between onset of Fever and Perforation:

Time interval between Perforation & Surgery

### SYMPTOMS & SIGNS

- Fever
- Abd, pain
- Vomiting
- Constipation/ Diarrhoea
- Other symptoms
- Previous H/o ATT
- Dehydratrtion
- Pallor
- P/A
  - Distension
  - Rigidity / Guarding
  - Obliteration of liver dullness
- BS
PR
BP
RR
Temperature
Investigation
Hb
Blood
Urea
Urine
Creatinine
Sugar
Widal test
X –Ray Abdomen erect
X- Ray Chest
USG / CT Abdomen
Operative finding
Procedure done
Post Op Complications
Follow up
Biopsy Report

Electrolytes
Na
K
HCO₃
Cl
Blood Culture
Mantoux Test
Sputum AFB
KEY TO MASTER CHART

S  - Sepsis
T  - Toxemia
D  - Wound Dehiscence
TH - Thrombophlebitis
PI - Paralytic ileus
PA - Pelvic abscess
F  - Faecal Fistula
R  - Respiratory Complication
I  - Intestinal Obstruction
MULTIPLE ILEAL PERFORATION

RESECTION AND END TO END ANASTOMOSIS
STRICTURE ILEOCAECAL JUNCTION (TB)
WITH ILEAL PERFORATION – RIGHT HEMICOLECTOMY
SPECIMEN
ILEAL PERFORATION IN THE
ANTIMESENTRIC BORDER
ILEAL PERFORATION AT THE ANTIMESENTRIC BORDER
BURST ABDOMEN