

A Comparative Study of Typhoid and Non Typhoid Small Bowel Perforations

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ABSTRACT

Introduction: Small Bowel perforations are an important cause of acute abdomen and peritonitis in developing nations. Typhoid fever is the commonest cause in tropical countries, while malignancy and mechanical causes are most common in the west.

Aim and Objectives: The aim of our study was to determine the causes of small bowel perforations and to compare the outcome in patients with typhoid non-typhoid small bowel perforations.

Materials and Methods: This comparative, analytic study was conducted in the Department of Surgery, CAIMS, Karimnagar, between September 2015-2017. Total 56 patients were included in this study. All patients with small bowel perforation who underwent surgical closure of a full thickness small intestinal breach and who satisfied the inclusion criteria were analyzed and followed up until their discharge from hospital or death.

Results: The age of the patients in this study ranged from 9 to 65 years with a mean of 32.54± 13.58 years. Small bowel perforations commonly occurred in the third and fourth decades of life with 55.35% of patients being in that age group. Male to female ratio was 4:1. Typhoid small bowel perforation commonly occurred in second and third decades of life with a mean of 25.12± 11.56 years. Non-typhoid small bowel perforations commonly occurred in the third and fourth decades of life with mean of 35.29± 14.62. Traumatic perforations were found only in men. Twenty one men (37.5%) and 4 women (7.1%) had typhoid small bowel perforations.

Conclusion: We conclude, therefore, that widal and blood cultures alone are not sufficient to diagnose typhoid small bowel perforations. Other tests like DOT EIA, tissue biopsy culture would help to increase diagnostic yield. There appears to be a definite entity of non-specific small bowel perforation, which cannot be attributed to any other cause. Complication rates are much higher in typhoid small bowel perforation.

Keywords: Typhoid, non-typhoid, small bowel perforations

INTRODUCTION

Small Bowel perforations are an important cause of acute abdomen and peritonitis in developing nations. Typhoid fever is the commonest cause in tropical countries, while malignancy and mechanical causes are most common in the west.^[1] The aetiology of small bowel perforations still remains elusive in many patients. These are usually clubbed under the general category of non specific small bowel perforations. Whether these small bowel perforations are a distinct clinical entity or whether they are undiagnosed typhoid perforation is not clear.^[2, 3]

Most authors who have studied small bowel perforations have concentrated on typhoid small bowel perforations.^[4] In all studies, diagnosis of typhoid perforation has been made either on clinical grounds or on Widal, blood culture

or histopathological examination. All these investigations are either positive only in a minority of patients with typhoid fever, or are nonspecific for typhoid.^[5]

AIMS AND OBJECTIVES

This study was planned to elucidate the cause of small bowel perforation in our institute, to establish the diagnosis of typhoid small bowel perforations by performing a battery of tests; and to compare the outcome in typhoid small bowel perforations with non typhoid and non-specific perforations.

MATERIALS AND METHODS

This comparative, analytic study was conducted in the department of Surgery, CAIMS, Karimnagar, between

September 2015 -2017. All patients with small bowel perforation who underwent surgical closure of a full thickness small intestinal breach and who satisfied the inclusion criteria were analyzed and followed up until their discharge from hospital or death.

These patients were ultimately divided into two groups:

Group I: Patients with typhoid small bowel perforations.

Group II: Patients with non-typhoid small bowel perforations.

Exclusion criteria

a) Small bowel perforations occurring secondary to any obstructions.

The patients in the study were initially interviewed with specific regard to certain factors known to be important in the etiology of small bowel perforations. These included age, sex and duration of symptoms before presentation to hospital, the use of steroid medications etc. The attending surgeon's preoperative diagnosis was also noted down, as per the patients record. The vital parameters of each patient, Viz, pulse rate blood pressure were recorded at admission.

Laboratory data obtained in each patient included hemoglobin, total blood counts, differential counts, blood urea and serum creatinine.

The following investigations were done as routine; Widal Test, blood culture, stool culture, urine culture, histopathological examination, DOT Enzyme immunoassay (EIA) (Typhidot) and tissue biopsy culture.

DOT Enzyme Immunoassay (EIA) (Typhidot)

The reaction tray was divided into two columns marked 'M' and 'G' the required number of strips was coded and labeled. The 'M' and 'G' strips for each serum were placed in the appropriate 'M' and 'G' wells, 250 ml of the sample diluents was dispensed into each well, 2.5 ml of control / test serum was also added to achieve a final dilution of 1:100 and the solution mixed well.

The solution was incubated for 20 minutes, using a washing buffer. Washing was done 3 times for a total of 15 minutes. 250ml of colour development solution was subsequently added into each well and incubation done for 15 minutes. The results were then interpreted. Only when both dots on the test strip were as dark as or darker than their corresponding dots on the positive control strips would the test be reported as positive.

Tissue Biopsy Culture

Biopsy taken from the edge of perforation was collected

in sterile saline. One portion was inoculated into brain heart broth and incubated for upto 48 hours. Subcultures were made into blood and McConkey agar. Bacteria grown if any, were identified by standard procedure. A second portion was inoculated into selenite F broth and subcultured after 6 hours for 18 hours in deoxycholate citrate agar. A similar procedure was followed for identification of bacteria grown.

In addition, bone marrow aspirate culture was done in all patients whose blood and tissue biopsy cultures were negative.

Management

All patients were resuscitated preoperatively using intravenous fluids and antibiotics. All the patients underwent laparotomy under general anaesthesia. Most of the operative interventions were done by trainee residents under supervision of qualified staff. Intraoperative variables studied included the presence of gross peritoneal contamination; the presence, number and size of perforations; the suture material used and the procedure employed.

The following procedure were Simple Closure, Wedge Resection, Resection Anastomosis and Bypass.

RESULTS

Age and Gender Distribution

The age of the patients in this study ranged from 9 to 65 years with a mean of 32.54± 13.58 years. Small bowel perforations commonly occurred in the third and fourth decades of life with 55.35% of patients being in that age group. Male to female ratio was 4:1.

Typhoid small bowel perforation commonly occurred in second and third decades of life with a mean of 25.12± 11.56 years. Non-typhoid small bowel perforations commonly occurred in the third and fourth decades of life with mean of 35.29± 14.62 . Traumatic perforations were found only in men. Twenty one men (37.5%) and 4 women (7.1%) had typhoid small bowel perforations. There was no significant difference in the occurrence of typhoid perforation ($p=0.538$) between men and women.

Histopathological examination

Histopathological examination was done in 56 patients with small bowel perforations. It was suggestive of typhoid in 15(26.8%) of the patients and tuberculosis in 4(7.1%) patients. Tuberculosis perforations were definitely diagnosed by histopathology. In study, patients with histological feature suggestive of typhoid perforation were not included under typhoid positive group as histopathology features for typhoid perforation are fairly

non-specific.

DOT Enzyme immunoassay (EIA) – (Typhidot)

The typhoid test was done in 56 patients with small bowel perforation and was positive in 21 (31.5%) patients overall. In typhoid positive patients, Typhidot was positive in 84% of patients. This test correlated well with Widal positivity.

Surgical Procedure

Wedge resection and anastomosis was the commonest surgical procedure done in 28(50.0%) patients. Resection anastomosis was done in 20(53.7%) patients, simple closure in 6(10.7%) patients and ileotransverse bypass in 2(3.6%) patients. In this study, 13 (23.2%) patients developed fecal fistula. Leak rate was higher after wedge resection (7; 25.0%) patients than after formal resection (4;20.0%) but this not statistically significant (p=0.752).

The type of surgical procedure did not influence the morbidity and mortality in patients with small bowel perforations. In patients with typhoid perforation, wedge resection anastomosis had a higher mortality and higher complications rate but this was not found to be statistically significant (p = 0.420)

Operative Findings

Peritoneal contamination between typhoid and non typhoid perforations was statistically not significant (p=0.303). Forty eight (85.7%) patients had single perforation. Two perforations were seen in (8.9%) patients. Multiple perforations occurred in 3(5.4%) patients. There was no significant difference in incidence of single and multiple perforation between typhoid and non typhoid groups(p=0.272).

The maximum distance of perforation from the ileocecal valve was 70cm and the minimum was 10cm with a mean of 37.32±14.30cm. 72% of perforations occurred within 45cm of ileocecal junction and 20% occurred within 30cm. The mean distance of perforation from ileocecal valve in typhoid group was 33.0±2.5cms, as compared to the valve of 40.81±2.68cms in non -typhoid group. This difference was statistically significant (p=0.041).

All the anastomosis were constructed in two layers in the studies. Non-absorbable suture are used for both layer in 38 cases, whereas full thickness absorbable with seromuscular non absorbable was used in 14 cases. Leaks were observed in 10 and 3 cases respectively in this group.

This difference was not statistically significant(p=0.424).

Complications

Complications occurred in 30 (53.9%) of patients. The common complications were wound infections in 27 patients (48.2%), anastomotic leak in 13(23.2%), intra-abdominal collection in 6(10.7%) and wound dehiscence in 3(3.6%). Three patients had respiratory infections. Overall complication rate was significantly higher in typhoid compared to non-typhoid small bowel perforations(72%Vs 38.7%; p=0.013).

Wound infection (64%),anastomotic leak (36%), intra-abdominal collection (20%) were significant higher in typhoid group compared to non-typhoid group (p=0.34, 0.042, 0.044 respectively).

Complication rate was not significant different between typhoid group and non-specific group, except for anastomotic leak which was significantly higher in typhoid group (p=0.030).

Pus culture from postoperative wound infections was done in 27 patients and grew organisms in 18 patients. The common organisms isolated were Esch.coli, Proteus and Staph.aureus. Most of these were sensitive to ciprofloxacin and cefotaxime but resistant to penicillin and ampicillin.

Mortality

The mortality rate was 8 out of 56(14.3%). Six patients died in the hospital and 2 patients went against medical advice (AMA) at terminal stage and were included in the mortality group. The mortality in patients with typhoid and non-typhoid small bowel perforations were 5(20%) and 3(9.7%) respectively but the difference was not found to be statistically significant (p=0.780).

The mortality in patients with typhoid and non specific perforations were 5(20%) respectively, but difference was not found to be statistically significant (p=0.295). The only patients with radiation enteritis had anastomotic leak and subsequently died. All but one of the deaths were associated with anastomotic leak.

Table 1: Test for Typhoid Diagnosis

TEST	POSITIVE No. %
Typhidot	21(84.0)
Widal	12(48.0)
Tissue biopsy culture	6(24.0)
Stool culture	1(4.0)
Total	25 (44.64)

Table 2: Comparison of Typhidot with other tests for detection of Typhoid Fever

Test	Typhidot Positive N=21	Typhidot Negative N=35
Widal Positive	11	1
Biopsy Culture Positive	3	3
Stool Culture Positive	1	0
Blood/ Bone Marrow/Urine Culture	0	0

the entity of non-specific perforations by doing an exhaustive battery of tests for typhoid fever, and to compare outcome of typhoid and non-typhoid small perforations.

Aetiology

In western series, the common causes of small perforation includes mechanical causes,^[6,7] lymphoma,^[6] malignancies^[7,8] etc. The incidence of typhoid perforation

Table 3: Surgical procedures and their complications in small bowel perforations

COMPLICATIONS	SIMPLE CLOSURE (n=6) (%)	WEDGE RESECTION (n=28) (%)	RESECTION ANASTOMOSIS (n=20) (%)	ILEO-TRANSVERSE BYPASS (N=2) (%)	TOTAL No.(%)	'p'
Wound infection	2 (33.0)	18 (62.3)	6 (30.0)	1 (50.0)	27 (48.2)	0.107
Wound dehiscence	0	2 (7.1)	1 (5.0)	0	3 (3.6)	0.889
Intra-abdominal collection	1 (16.6)	3 (10.7)	2 (10.0)	0	6(10.7)	0.925
Anastomotic leak	1 (16.6)	7 (25.0)	4 (20.0)	1 (50.0)	13 (23.2)	0.773
Respiratory	1 (16.6)	2 (7.1)	0	0	0	0.402
Mortality	0	4 (14.3)	3 (15.0)	1 (50.0)	8(14.3)	0.652
Number of patients with complications	2/6 (33.6%)	19/28 (67.9)	8/20 (40.0)	1/2 (50.0)	30/56 (53.9)	0.189

*Some patients had more than one complication

Table 4: Complications of small bowel perforations in Typhoid versus Non-Typhoid Group

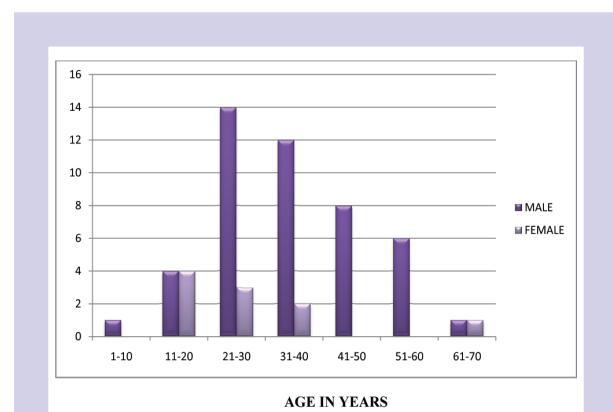
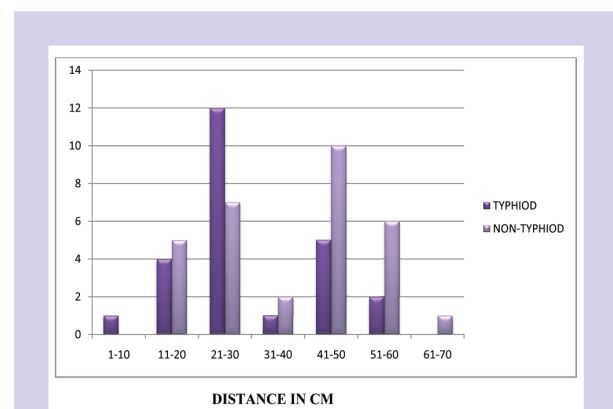
Compilcations	Typhiod N= 25(%)	Non-Typhiod N=31(%)	Total N =56(%)	'p'
Wound Infection	16 (64.0)	11(35.0)	27 (48.2)	0.034*
Wound Dehiscence	2 (8.0)	1 (3.2)	3 (5.4)	0.430
Intra-Abdominal Collection	9(36.0)	4 (12.9)	13 (23.3)	0.042*
Anastomotic Leak	5 (20.0)	1(3.2)	6 (10.7)	0.044*
Respiratory	2 (8.0)	1 (3.2)	3 (5.3)	0.430
Mortality	5 (20.0)	3 (9.7)	8 (14.3)	0.780
Total	18 (72.0)	12 (38.7)	30 (53.9)	0.013*

*Significant p value

** Many patients had more than one complication

DISCUSSION

Small bowel perforations are an important cause of acute abdomen and peritonitis in developing nations. The aetiology of small bowel perforations still remains elusive in many patients. These are usually clubbed under the general category of nonspecific small bowel perforations. Whether these small bowel perforations are a distinct clinical entity or whether they are undiagnosed typhoid perforation is not clear. Our present effort was conducted to clarify causes of small bowel perforations, to confirm

**Graph 1 : Age and Gender distribution of small bowel perforations****Graph 2 : distance of perforation from ileocaecal valve in typhoid versus non-typhoid**

ranges from 0.5% to 78.6%.^[12,13] Typhoid fever is the commonest cause of small bowel perforations in tropical countries. In a previous studies in our institute the incidence of typhoid perforation was 47.8%. In this series, the diagnosis of enteric fever was made by widal and/or blood culture.^[11] A few patients underwent bone marrow culture. In our present study, typhoid small bowel perforation was seen in 44.6% cases. An exhaustive battery of tests was performed to diagnose typhoid (as discussed earlier).

Non- specific perforation was the second common cause of small bowel perforation in our series accounting 39.4% of lesions. No cause for perforation could be demonstrated in any of these patients after detailed history and investigations. Non- specific perforations was the second common cause of small bowel perforations in the series by Chatterjee.^[11] Non-specific perforation was the commonest cause of small bowel perforation in the series by Bhalarao^[9] and Dixon.^[6] It is possible that we have less number of non specific perforations, because of the extensive tests we performed to diagnosed typhoid.

8.5% cause of ileal perforation published by Karmarkar were due to trauma.^[14] Trauma accounted for 4.3% of cases in the study by Chatterjee from our institute.^[11] Trauma accounted for 7.1% of cases of small bowel perforation in our series. Traumatic perforations have therefore shown an increase in this institute but incidence was similar to Karmarkar's series.^[15]

Tuberculosis accounted for 7.1%of cases of small bowel perforations in this series compared to 9.3%of cases published by Bhalarao.^[9]

Age and Gender Distribution

The age of patients in this study ranged from 9 to 65 years with a mean of 32.54± 13.58 years. Others like Bose et al¹⁰ reported age ranging from 14 to 72 years with mean 33.5 years similar to our study. However Argkun reported 19age between 3-76 years with mean age of 27 years, which is much lower compared to our study. Other studies have reported higher mean age of 51.8± 21.8 years.^[7]

Symptoms and Signs

In our study, most common symptoms were pain abdomen and vomiting and the commonest signs were abdominal tenderness and guarding . Other studies have also showed similar presentation.^[16, 17, 18, 19]

Perforation has been reported to commonly occur in the second week following the onset of illness.^[20, 21] Keenan reported that 88% of patients perforated in the second week. Lizzaralde reported that 54.2% of patient perforated in the second week.^[20] Chatterjee et al^[11]

reported that 45% of patients perforated in the first week in the third phase of their study. Similarly, we have found that perforation occurred earlier with 45% of patients have perforation within one week of onset of fever.

In our study, pulse rate per minute at presentation between typhoid and non-typhoid did not differ significantly ($p=0.0638$). Mean systolic blood pressure also not differ between typhoid and non typhoid small bowel perforations (98±10 vs 102±15 respectively) ($p=0.363$). Meier reported an average systemic blood pressure of 104mm Hg and an average pulse rate of 121/min in patients with typhoid small bowel perforations. The mean blood pressure in his study was similar to ours, but the average pulse rate was higher in his series.^[16]

In our study, the mean white cell count was 10164±1784 cells/mm³, mean hemoglobin was 9.54±1.67 gm/dl, mean neutrophil differential count was 70±3% and mean lymphocyte count 23±5%. Several studies have shown similar to our study^[16] other have shown lower total white cell counts ranging from 5100 to 8200/cu mm.^[22] We found a mean blood urea of 37.71±14.50 mg/dl and serum creatinine of 0.612±0.568 mg/dl. Kaul reported higher blood urea values (60-150mg/ dl) in comparison to our series.^[23]

Radiology

Chest X-ray is a useful investigation to detect hollow viscus perforation. Pneumoperitoneum was found in 75% of patients with small perforation in our series. Pneumoperitoneum has been reported in 52% to 82% of patients in the earlier series.^[24, 25] Pneumoperitoneum was present in 639% of patients with small bowel perforation in the series by Chatterjee.^[11]

Serology

Widal test was positive in 48.0%of patients with typhoid small bowel perforation in the present series. Widal test was reported positive in 30% of patients with typhoid perforation in series by Kaul.^[23] Sentillana^[26] reported 46.1% of patients with widal positive similar to our study. Noorani also reported widal test positive in 47.3% of cases.^[19] Widal test was reported positive in 75.5% of patients by Jarrett^[27] and 73% of patients by Vaidyanthan.^[24] Both series showed higher rate of positively compare to our study. In the earlier study done by Chatterjee^[11] in our institute , Widal test was positive in 72% of patients with typhoid ileal perforations. Hence, it is observed that Widal test is not a good indicator of typhoid small bowel perforation and other tests are necessary to get a higher diagnostic yield.

Histopathology

Other series have been used histopathology as one of the

diagnosed tests for typhoid small bowel perforation.^[28] Histopathology was suggestive of typhoid perforation in 26.7% of patients with small bowel perforation in the present series. However, histopathology diagnosis is based on non-specific appearance like the presence of macrophages containing bacteria, red blood cells (erythrophagocytosis), and nuclear debris from small nodular aggregates in Peyer's patches with intermingled lymphocytes and plasma cells.^[29] Hence, we feel that histopathology is not a reliable indicator of small bowel perforation. Ravinder Bal has also reported that biopsy of every perforated terminal ileum should be correlated with serological tests for typhoid fever.^[28] Tuberculosis and malignant perforation were the other disease definitely diagnosed by histopathology in our series.

Cultures

Culture yields were generally poor in our study. In the present study none of the blood culture grew Salmonella typhi. Hadley reported positive blood culture in 27.2% of patients and Santillana in 48% of patients. Chatterjee et al reported blood culture in 11% patients with typhoid perforation. Prior antibiotic therapy was probably responsible for the low isolated rates in all studies.^[20] Other causes might be inadequate samples. One stool culture in our study grew salmonella typhi which was sensitive to ciprofloxacin and ceftazidime and resistant to other antibiotics. All other stool/urine cultures were negative. Gilman et al reported stool cultures positive in 37% and urine culture positive in 9% of patients with typhoid fever.^[30] Badajo reported stool culture positively in 4.4% and urine culture positively in 1.1% of patients with typhoid fever.^[14] All stool/urine culture were reported negative in typhoid perforation patients in several studies.^[31]

Salmonella typhi was grown in 6 (24%) patients with typhoid small bowel perforation in whom tissue biopsy culture was done in our study. No previous studies have so far performed tissue biopsy culture for diagnosis to the best of our knowledge. Tissue biopsy culture will improve culture diagnosis of typhoid and should be done routinely.

DOT enzyme immunoassay (EIA) (Typhidot) test has been found to be as sensitive as the Widal test (95% and 98% respectively).^[32] Butta et al reported a sensitivity of 85.9% and specificity of 77.9% for typhidot, and Ismail reported a sensitivity of 90.0% and specificity of 93%.^[31] The dot EIA test was positive in 84% of patients with typhoid perforation in present series. This rate was much higher than the widal positivity rate (48%).

Management

In the management of typhoid perforation, some authors

have earlier advocated conservative management.^[34] Presently there is no controversy with all authors recommending surgical treatment. The choice of surgical procedure in typhoid perforation is controversial. The various modalities employed have been flank drainage of peritoneal cavity, simple closure wedge resection and closure, resection anastomosis ileotransverse bypass and ileostomy. In the present series no patients was treated by conservative management, flank drainage or ileostomy. The surgical procedure employed were simple closure, wedge resection and closure, resection anastomosis and ileotransverse bypass for both typhoid and non-typhoid small bowel perforation. There was no statistically significant difference in the surgical procedure performed between these groups ($p=0.522$)

The type of surgical procedure did not influence the morbidity and mortality in patients with small bowel perforation in our series. In patients with typhoid perforation, wedge resection and closure had a higher complication rates, but the difference was not found to be statistically significant. Egsieston and Santhosi reported that mortality was independent of the surgical procedure done.^[20] Talwar and Sharma reported that mortality least with early primary closure.^[35] Ameh et al reported maximum mortality with wedge resection and least mortality with resection anastomosis. Chatterjee et al reported in first phase simple closure had the highest mortality while wedge resection had the least mortality. In the second phase ileotransverse bypass had the highest mortality. In third phase, resection anastomosis had the highest mortality.

In our study, 48 (85.7%) patients had a single perforation. Two perforations were seen in 5 (8.9%) patients and multiple perforation in 3 (5.3%) patients. There was no significant difference in incidence of single and multiple perforation between typhoid and non typhoid groups ($p=0.272$). The perforations were located on the antimesenteric border of the ileum, ranging from a distance of 10cms to 70 cms proximal to the ileocecal valve, with mean of 36.5 cms. The mean distance of perforation from ileocecal valve in typhoid group 33.0 ± 2.50 cms as compared to the valve of 40.81 ± 2.68 cms in non typhoid group. This difference was statistically significant ($p=0.041$)

Mork et al^[36] reported that most of patients had only one perforation and the distance of perforation from the ileocecal valve was upto 80cms proximally with mean of 24 cms. Santillana^[26] was reported that a single perforation was found in (78%) of the patients. Two perforation were found in (16%) patients and multiple perforations in (6%). Most perforations (63%) were located within 30 cms of ileocecal valve. His results were similar to ours.

In our study, all the anastomosis were constructed in two layers. Non absorbable suture are used for both layer in 38 cases, where as full thickness absorbable with seromuscular non-absorbable was used in 14 cases. There was no difference in leak rates in between the two techniques ($p=0.424$). Singh et al 37 reported two layers closure using full thickness 3.0 chromic catgut for inner layer and 3.0 silk seromuscular suture for the outer layer. He found that the addition of a second layer in closure of the perforation was helpful in lessening the chance of suture line leakage.

Complications

In the present series, 58.9% of patients developed one or more postoperative complications. Chatterjee et al reported that postoperative complication rate 34%, 35% and 51.1% in three phases of their study. The third phase showed complication rate similar to our study. Santillana in his series reported a complication rate of 71%. His series showed higher complication rate compared to our present series. Complication rate between 28.%% have reported in various studies.^[38]

In our study, typhoid perforation was associated with complication rate of 72% and non typhoid perforation with a rate of 38.7%. The total complication rate was significantly higher in typhoid small bowel perforation compare to non- typhoid group ($p=0.013$). The complications encountered in decreasing order were wound infection (48.3%), anastomotic leak (23.2%), abdominal collection (10.7%), wound dehiscence (3.6%) and respiratory complication (3.6%). Similar complication have been reported in various studies.

In the present series, wound infection, anastomotic leak and intra-abdominal fluid collections were significantly higher in typhoid group compared to the (p values of 0.034, 0.042 and 0.044 respectively). Only anastomotic leak was significantly higher in typhoid group compared to patients with non specific perforation ($p= 0.030$). In other studies, wound infection rates have been found to be high for typhoid small bowel perforation ranging from 30% to 79%).

Anastomotic leak was seen in 13 (22.02%) patients in the present series. In patients with typhoid perforation anastomotic leak was seen in 36% and in non-typhoid perforation it was 12.9%. Anastomotic leak rate was significantly higher in typhoid small bowel perforations compared to non-typhoid group ($p= 0.042$). Fistula rates between 3 to 10% have been reported in literature.^[40]

In our patients with anastomotic leak, 6 out of 13(46.2%) patients were managed conservatively with a mortality of 4 (66.6%). Seven out of 13 (53.8%) patients were reoperated among whom two patients had an

exteriorization of the leak site with one death. The remaining five (71.4%) patients underwent freshening of the bowel edges at the sites of the primary anastomotic followed by reanastomosis of whom 2 (40%) patients subsequently died. The mortality rate was not significantly different between conservative and reoperative management group ($p=1.00$). The reason for lower complication rates in non-typhoid group may be because of relatively healthy bowel edge at the perforation site.

Mortality

Eight patients out of the 56 studied died, giving an overall mortality rate of 14.3%. All the deaths but one occurred in the leak group. In patients with typhoid perforation, mortality rate was 20% and in non-typhoid perforation group it was 9.7%. The mortality rate for non-specific perforations was 9.1% in the present study. There was no significant difference in mortality between typhoid and non specific perforation ($p=0.295$), nor between typhoid and non typhoid groups ($p=0.780$). Chatterjee reported a mortality of 7.1% in patients with typhoid perforation. Mortality rates in typhoid perforation ranges from 3-60% in various series.

CONCLUSION

We conclude, therefore, that widal and blood cultures alone are not sufficient to diagnose typhoid small bowel perforations. Other tests like DOT EIA, tissue biopsy culture would help to increase diagnostic yield. There appears to be a definite entity of non-specific small bowel perforation, which cannot be attributed to any other cause. Complication rates are much higher in typhoid small bowel perforation.

CONFLICT OF INTEREST :

The authors declared no conflict of interest.

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REFERENCES

1. Bishop WJ. *The Early History of surgery*. London: Robert Hale Limited; 1966.
2. Meade RH. *Surgery of the small intestine*, In: An introduction to the History of General Surgery. Philadelphia: WB Saunders Company; 1968.
3. Bitas R, Tarpley J. Intestinal perforation in typhoid fever. A historical and state of art review. *Rev infec Dis*. 1985; 7: 257-271.
4. Burch JM, Franciose Rj, Moore EE, Biftl WL, Offner PJ. Single Layer continuous versus two- layer interrupted intestinal anastomosis: a prospective randomized trial. *Ann Surg*. 2000; 231: 832-837.
5. Chambers HF. Antimicrobial agents, protein synthesis, inhibitors and miscellaneous antibacterial agent. In: Joel G. Hardman, Lee

- E, Limbird, eds. *The Pharmacological Basis of Therapeutics*, 10th edn. McGraw Hill, 2001; 1246-1247.
6. Dixon JM, Lamusden AM, Paris J. Small Bowel perforation. *J R Coll Surg Edinb*. 1985; 30: 43-46
 7. Chsikot. Non traumatic perforation of small. *Am J Surg*. 1987; 153: 355-358.
 8. Orringer RD, Collier JA, Veidhenheimer MC. Spontaneous free perforation of small intestine. *Dis Colon Rectum*. 1983; 26: 323-326.
 9. Nadkarni KM, Shetty SD, Kagzi RS, Bhalarao RA. Small bowel perforation. A study of 32 cases. *Arch surg*. 1981; 116: 53-57.
 10. Bose SM, Anantkumar, Chaudhary A, Indudhar A, Gupta NM, Khanna SK. Factors affecting mortality in small intestine perforation. *Indian J Gastroenterol*. 1986; 5:261-263.
 11. Chatterjee H, Jagdish S, Pai D, Satish N, Jayadev D, Reddy PS. Changing trends of typhoid ileal perforations over three decades in Pondicherry. *Trop Gastroenterol*. 2010; 22:155-158.
 12. Purohit. Surgical treatment of typhoid perforation. *Indian J Surg*. 1978; 40: 227.
 13. Keenan JP, Hadley GP. The surgical management of typhoid perforation in children. *Br J Surg*. 1984; 71:928-929.
 14. Choo KE, Oppenheimer SJ, Asma B, Ismail A, Ing KH. Rapid serodiagnosis of typhoid fever by dot enzyme immunoassay in an endemic area. *Clin Infect Dis*. 1994; 19:172-176.
 15. Ameh EA, Dago PM, Attah MM, Nmadu PT. Comparison of three operations for typhoid perforation. *Br J Surg*. 1997; 84:558-559.
 16. Meier DE, Imediogwu OO, Tarpley JL. Perforated typhoid enteritis: Operative experience with 108 cases. *Am J Surg*. 1989; 157:423-427.
 17. Eggleston FC, Santhosh B, Singh CM. Typhoid perforation of bowel. *Ann Surg*. 1979; 190:31-35.
 18. Gibney EJ. Typhoid perforation. *Br J Surg*. 1989; 76:887-889.
 19. Noorani MA, Sial I, Mal V. Typhoid perforation of small bowel: A study of 72 cases. *JR Coll Surg Edinb*. 1997; 42:274-276.
 20. Lizarralda EA. Typhoid perforation of the ileum in children. *J Pediatr Surg*. 1981; 16: 1012-1016.
 21. Singh S, Singh K, Grover AS, Kumar P, Singh G, Gupta DK. Two layer closure of typhoid ileal perforation. A prospective study of 416 cases. *Br J Surg*. 1995; 85:1253.
 22. Kayabali I, Gokcor H, Kayabali M. A contemporary evaluation of enteric perforation in typhoid fever: analysis of 357 cases. *JPMI*. 2001; 11:1-3.
 23. Kaul BK. Operative management of typhoid perforation in children. *Int Surg*. 1975; 60: 409-410.
 24. Vaidyanathan S. Surgical management of typhoid ileal perforation. *Indian J Surg*. 1986; 335-341.
 25. Laurence DR, Bennett PN. *Clinical Pharmacology, Infection I: Chemotherapy*, 7th edition, Edinburgh: Churchill Livingstone, 1992;157.
 26. Santhillana M. Surgical complications of typhoid fever. *World J Surg*. 1991; 15:170-175.
 27. Kim JP, Oh SK, Jarrett F. Management of ileal perforation due to typhoid fever. *Am Surg*. 1975; 181:88-91.
 28. Bal R, Uppin VM, Godhi AS. *Correlation between serological investigation and histopathological studies in cases of ileal perforations*. Karnataka Lingayat Education Society's Hospital and District Hospital, Online publication, August 2004.
 29. Samuelson J. *Infectious disease*. In: Cortran RS, Kumar V, Collins T, Robbins L. eds. *Robbins pathologic Basis of disease*. 6th edn. Philadelphia: WB Sounder's Company, 1999:356.
 30. Gilman RH, Terminal M, Levine MM, Hernandez MP, Hornick RB. Relative efficacy of blood, urine, rectal swab, bone marrow and rose-spot cultures for recovery of salmonella typhi in typhoid fever. *Lancet*. 1975; 1 (7918):1211-1213.
 31. Seshadri V, Natarajan TS, Johnson J, Jayaraj SD, Gnanvendan. Efficacy of bone marrow culture in enteric fever. *J Assoc Phys India*. 1977; 25:561.
 32. Choo KE, Oppenheimer SJ, Asma B, Ismail A, Ing KH. Rapid serodiagnosis of typhoid fever by dot enzyme immunoassay in an endemic area. *Clin infect Dis*. 1994; 19:172-176.
 33. Butta ZA, Mansurali N. Rapid serologic diagnosis of pediatric typhoid fever in an endemic area: a prospective comparative evaluation of two dot enzyme immunoassays and Widal test. *Am J Trop Med Hyg*. 1999; 61:654-657.
 34. Eggleston FC, Santhosh B, Singh CM. Typhoid perforation of bowel. *Ann Surg*. 1979; 190:31-35.
 35. Adesunkanmi ARK, Ajao OG. The prognostic factors in typhoid ileal perforation. *JR Coll Surg Edinb*. 1997; 42:395-399.
 36. Mock CN, Amaral J, Visser LE. Improvement in survival from typhoid ileal perforation. *Ann Surg*. 1992; 215:244-249.
 37. Singh S, Singh K, Grover AS, Kumar P, Singh G, Gupta DK. Two layer closure of typhoid ileal perforation. A prospective study of 416 cases. *Br J Surg*. 1995; 85:1253.
 38. Keenan JP, Hadley GP. The surgical management of typhoid perforation in children. *Br J Surg*. 1984; 71:928-929.
 39. Talwar S, Sharma RK, Mittal DK, Prasad P. Typhoid enteric perforation. *Aust AZ J Surg*. 1997; 67:351-353.
 40. Akgun Y, Bac B, Boylu S, Aban N, Taeyildiz I. Typhoid enteric perforation. *Br J Surg*. 1995; 82:1512-1515.