

Original Research Article

DOI: <https://dx.doi.org/10.18203/2349-2902.ijssurgery20220322>

Enteric perforation with peritonitis

Sohan Lal Meghwal^{1*}, M. L. Maida², Arjun Lal Kharadi², Mayur Kumar Pargi¹

¹Department of General Surgery, Dr. SN Medical College, Jodhpur, Rajasthan, India

²RNT Medical College, Udaipur, Rajasthan, India

Received: 18 December 2021

Revised: 23 January 2022

Accepted: 24 January 2022

***Correspondence:**

Dr. Sohan Lal Meghwal,

E-mail: dr.sljaipal24@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Typhoid fever, also known as enteric fever, is a systemic infection by *Salmonella typhi* or by the related but less virulent *Salmonella paratyphi*. Patient with perforation present with sign and symptoms of peritonitis complains of pain abdomen along with distension abdomen, obstipation and vomiting. Enteric perforation is managed surgically. Aims and objective: To study various epidemiological factors in relation with enteric perforation with peritonitis and to compare the outcomes of various procedures of enteric perforation with peritonitis.

Methods: The present study was performed on 50 patients of enteric perforation with peritonitis admitted in various surgical wards of RNT medical college hospital, Udaipur. Those patients who underwent conservative treatment or drainage under local anesthesia were not included in this study.

Results: Enteric perforation was more common in young males, maximum cases were in 3rd decade followed by 2nd and 4th. Male:female ratio was 1:77:1. Most common symptoms were acute pain abdomen and fever. Constipation (60%) abdominal distension (40%) and vomiting (52%) were other predominate complains. Simple repair of perforation was done in 25 patients. Repair was done in double layer (inner layer) by continuous vicryl and outer by interrupted silk. Mortality was 14%, almost equal irrespective to procedure chosen for management of enteric perforation peritonitis.

Conclusions: To deal with enteric perforation(s) operative procedure has to be decided to greater caution taking into consideration patient's general condition, gut condition, number and site of perforation and contamination of peritoneal cavity.

Keywords: Typhoid, Enteric perforation, *Salmonella typhi*, Gut, Site of perforation

INTRODUCTION

Typhoid fever, also known as enteric fever, is a systemic infection by *Salmonella typhi* or by the related but less virulent *Salmonella paratyphi*. Since ancient times, these bacteria have thrived during wartime and during the breakdown of basic sanitation. Archeologists have found *S. typhi* in Athenian mass graves from the era of the Peloponnesian Wars, implicating it as the cause of the Great plague of Athens. *S. typhi* persists mostly in developing nations where sanitation is generally poor.¹ Although sporadic outbreaks occur in developed nations,

most individuals with typhoid fever in such areas have recently returned from travel to an endemic region.

Of all *Salmonella* serotypes, only *S. typhi* and *S. paratyphi* are pathogenic exclusively in humans. Typhoid fever is a severe multisystemic illness characterized by the classic prolonged fever, sustained bacteremia without endothelial or endocardial involvement, and bacterial invasion of and multiplication within the mononuclear phagocytic cells of the liver, spleen, lymph nodes, and Peyer patches. Typhoid fever is potentially fatal if untreated.

People are typically infected with *S. typhi* and *S. paratyphi* through food and beverages contaminated by a chronic stool carrier. Less commonly, carriers may shed the bacteria in urine. Individuals may also be infected by drinking sewage-contaminated water or by eating contaminated shellfish or faultily canned meat.²

Salmonella is a genus in the family *Enterobacteriaceae* that has more than 2300 serotypes previously described in the Kauffmann-White schema. *Salmonellae* are gram-negative, flagellate, nonsporulating, facultative anaerobic bacilli that ferment glucose, reduce nitrate to nitrite, and synthesize peritrichous flagella when motile. All but *S. typhi* produce gas upon sugar fermentation.

Pathophysiology

After ingestion by host, *salmonella typhi* successfully passing through the stomach, any *Salmonella* sub-species may be phagocytized by the gut's intraluminal dendritic cells, causing inflammation that leads to diarrhoea only. Subspecies *S. enterica* causes severe disease in rest of the body. Its specialized fimbriae adhere to epithelium that over lies Peyer's patches. Peyer's patches are grossly visible aggregates of 5-100 lymphoid follicle in small bowel mucosa. These patches are larger and more numerous distally. In sub mucosa *salmonella* enters macrophage via bacteria-triggered pinocytosis or via macrophage receptor mediated phagocytosis. Infected macrophage provides *salmonella* a vehicle safe from other elements of immune system and in which it can multiply and travel. It passes through mesenteric lymph node into thoracic duct and lymphatics beyond to seed the reticuloendothelial tissue, liver, spleen, bone marrow and lymph node. In these places it multiplies until some critical density is reached. It causes the apoptosis in macrophages and enters the blood stream to attack the rest of body.³

From blood or from liver via bile duct, it infects the gallbladder and reenters the gastro intestinal tract in bile, spreading to other host via stool.

After primary intestinal infection, further seeding of Peyer's patches occurs through infected bile, they may become hyperplastic and necrotic with infiltration of mononuclear cells and neutrophil and forming ulcer that may hemorrhage through eroded vessel or perforate the bowel wall causing peritonitis.

Risk factors

Salmonella has mechanisms against acidic environments, but a pH level of 1.5 or less kills most of the bacilli. People who continually ingest antacids, histamine-2 receptor antagonists (H₂ blockers), or proton pump inhibitors; who have undergone gastrectomy; or who have achlorhydria due to aging or other factors require fewer bacilli to produce clinical disease. Acquired immune deficiencies or hereditary deficiencies in

immune modulators such as IL-12 and IL-23 increase risk for infection, complications, and death.

Sign and symptoms

Classical sign and symptoms of enteric fever are toxemias, delirium, abdominal pain, constipation and hepatosplenomegaly.

Complication of enteric fever

There are several complications like paralytic ileus, typhoid ulcer (Parallel to long axis of gut), cholecystitis, phlebitis, typhoid cystitis, arthritis, osteomyelitis, epiddymorchitis, but intestinal perforation in one of the most dreadful complications of enteric fever. It usually occurs at the end of 2nd week and beginning of 3rd week of disease. Commonest site of perforation is the terminal part of ileum. Intestinal perforations through an ulcerated Peyer patches occur in approximately 2% of cases. Typically, it is single perforation in terminal ileum. The incidence of hemorrhage was reported to be as high as 20%.⁴

Patient with perforation present with sign and symptoms of peritonitis. Patient complains of pain abdomen along with distension abdomen, obstipation and vomiting.

On general examination he usually shows anxious look, tachycardia, tachypnoea, hypotension. Abdominal examination reveals generalized rigidity, guarding, tenderness, liver dullness may or may not be obliterated.

Enteric perforation diagnosed by history clinical examination, radiological investigation and hematological investigation.

But sometime it may be missed because: Pain in not so severe and abrupt in onset, rigidity at times minimal or unconvincing, Leukocytosis is evident in few cases and evidence of pneumoperitoneum is not always obtained.

Enteric perforation in managed surgically, typically there is a single perforation in the terminal ileum and simple closure of perforation is treatment of choice with multiple perforation resection with primary anastomosis or exteriorization of intestinal loop may be required.

Thus, this study is aimed to study various epidemiological factors in relation with enteric perforation with peritonitis and to study and compare the outcomes of various procedures of enteric perforation with peritonitis.

METHOD

The present prospective observational study was performed on 50 patients of enteric perforation with peritonitis admitted in various surgical wards of RNT medical college hospital, Udaipur during the period June

2020 to July 2021 after taking proper approval from the institutional ethical committee.

All patients with primary varicose veins of lower limb due to superficial and perforator incompetence (including long saphenous and short saphenous varicosity) and patients with the following symptoms of varicose veins: ulceration, phlebitis, bleeding, aching, skin changes (eczema, lipo-dermosclerosis, pigmentation) were included in the study.

Those patients who underwent conservative treatment or drainage under local anesthesia were excluded from this study.

All patients of enteric perforation peritonitis were evaluated by detailed history, clinical examination, and radiological and laboratory investigation. After initial resuscitation patient were treated by different operative procedures. Postoperatively daily progress reports and morbidity and mortality were recorded.

Complete history, clinical features, investigation, operative procedure, post operative management, morbidity and mortality were recorded.

General physical examination

On admission of a patient a detailed clinical examination was carried out in every patient, vitals were recorded like pulse rate, blood pressure, temperature, respiratory rate and urinary output. Signs of toxemia and dehydration were specially looked for.

Local examination

Attention was paid to following signs of perforation peritonitis. Generalized tenderness / rigidity / guarding, Obliteration of liver dullness, shifting dullness, bowel sounds, rectal and vaginal examination were done.

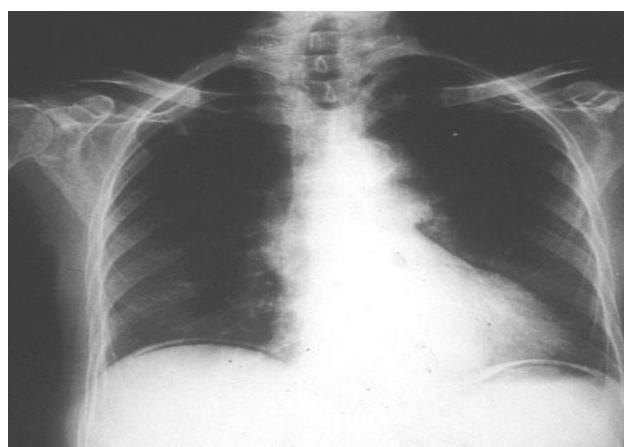


Figure 1: X-ray chest (PA view) of gas under both dome of diaphragm.

Treatment

The provisional preoperative diagnosis was made on the basis of history, clinical examination and radiological findings

A clinical and biochemical assessment of patients for dehydration, electrolyte imbalance, toxæmia and renal function was made.

Patients were kept NBM and good intravenous line was secured to administered fluid and electrolyte solution to correct hydration and electrolyte imbalance.

Nasogastric decompression was provided by continuous Ryles tube aspiration, during resuscitation a combination of broad-spectrum antibiotics (Usually a third-generation cephalosporin, metronidazole and an aminoglycoside) was given parentally to cover gram negative aerobic and anaerobic infections.

After appropriate resuscitation patients underwent surgery. Surgery was conducted under general anesthesia. Abdomen was explored through either right paramedian incision or lower midline incision. The amount quality and contamination of the peritoneal fluid was looked for.

Various operative procedures used were: simple repair by single or double layer, excision of ulcer margin and simple repair, simple repair and illeo-transverse anastomosis, repair of distal perforations and loop ileostomy from proximal perforation in cases of multiple perforations, loop ileostomy, end ileostomy and mucous fistula and postoperatively some patients were kept NBM with continuous Ryles tube aspiration till 5th or more postoperative day.

Broad spectrum antibiotics were given which include combination of a third-generation cephalosporin, metronidazole and an aminoglycoside. A strict output and input chart was maintained during post-operative method.

During postoperative period patients were intensively observed for development of complications like wound infection, repair or anastomotic leakage pulmonary complication, toxæmia, renal failure, intraperitoneal abscess etc.

Ryles tube was taken out on 5th postoperative day when there was no abdominal distension, patient had passed flatus, bowel sounds were present drains, were removed on postoperative day 5 or 6 when there output was less than 100 ml and it was of serous in nature.

After removal of Ryles tube the patients was given liquid diet on the same day, semisolid diet was started on the next day and solid diet was started when patient is tolerating the semisolid diet well.

Patient with ileostomy were normally re-admitted after about 6-8 weeks, when the patient general condition, had improved and end to end anastomosis were performed either in single layer or double layer.

RESULTS

The present retrospective and prospective study was carried in 50 cases of enteric perforation peritonitis admitted to R.N.T. Medical College Udaipur, in various surgical wards during the period 2004-06.

Table 1: Age and sex incidence.

Age (years)	Male	Female	Total
11-20	8	6	14
21-30	14	4	18
31-40	4	4	8
41-50	-	2	2
51-60	2	-	2
61-70	4	2	6
Total	32	18	50

The maximum no. of patients were in 3rd decade followed by 2nd, 4th decade. Pediatric age group were not included in this study.

Table 2: Perforation hospital admission interval.

Duration in days	No. of cases
1-2	24
3-5	20
6-8	4
9-10	2

The 48% of cases presented within 2 days of perforation. 88% of patients presented within 5 days of perforation.

Table 3: Symptomatology and signs typhoid perforation (50 cases).

Symptoms	No. of patient	Percentatge of patient (%)
Fever	40	80
Pain abdomen	50	100
ABD distension	20	40
Vomiting	26	52
Constipation	30	60
Distension of abdomen	20	40
Guarding/rigidity/	50	100
Tenderness		
Shifting dullness	35	70
Obliteration of liver dullness	33	66
Absent bowel sound	46	92

Most common symptoms were pain abdomen and fever present in 100% and 80% of cases respectively. Abdomen distension and constipation were other presenting symptoms.

On clinical examination, presence of guarding/ rigidity/ tenderness/ distended abdomen, absent bowel sounds. Shifting dullness were the major findings.

Primary (simple) repair of perforation was done in 50% of cases. Freshening of margin of perforation and repair was done in 30% cases. 12% of cases were managed by resection and anastomosis 4-4% cases were managed by ileostomy and closure and by-pass respectively.

Table 4: Comparison between various operative procedure.

Procedure	N	Hospital stay	Complications					Mortality (%)
			Wound (infection)	FF	Wound dehiscence	Septicemia	Pulmonary complication	
Simple repair	25	19.5	14	1	-	2	2	4 (16)
Freshening margin repair	15	11.3	2		6	-	-	2 (13.2)
Closure and by pass	2	13.5	2		-	-	-	-
Resection astomosis	6	12	2	1	2	-	-	1 (16.6)
Ileostomy	2	19	-		-	-	-	-

Table 5: Mortality in relation to number of perforations.

No. of perforation	No. of cases	Mortality (%)
One	36	4 (11.11)
Two	8	2 (25)
Three	-	-
>Three	6	1 (16.6)

Mortality was higher in cases with resections and anastomoses as compared on simple repair. Overall complication rate was seventy-six percentages in cases of simple repair while 83.3% in cases of resections anastomoses. Average total hospital stay was almost same in simple repair and ileostomy and it was about nineteen days.

Max. mortality was seen in perforation was 2 and it was 25% while 16.6% mortality was seen in >3 perforation.

Table 6: Mortality in relation to duration of perforation.

Duration of perforation (days)	N	Mortality (n)	Mortality (%)
1-2	24	2	8.3
3-5	20	2	10
> 5	6	3	50

Mortality was more in patients presented with longer duration of perforation. Majority of cases presented within 1-2 days old perforation and mortality was least in these patients.

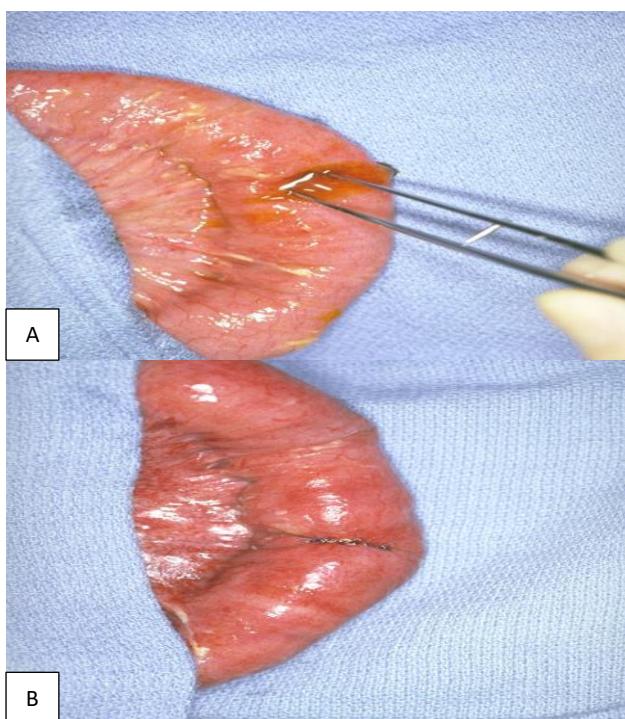


Figure 2 (A and B): Enteric perforation in the ileum, closure of enteric perforation in the ileum.



Figure 3: Closed abdomen after repair of enteric perforation with drains placement.

DISCUSSION

Typhoid fever is an endemic disease in Indian subcontinent. It is caused by gram negative bacilli, *Salmonella typhi*. It is transmitted by feco-oral route. Poor water supply and sanitation facilities are the main cause of prevalence of the disease in developing countries. Intestinal perforation and bleeding are two main complication of typhoid fever and are responsible for majority of deaths.

In our study enteric perforation was more common in young males. In our series maximum cases were in 3rd decade followed by 2nd and 4th decade. Same was reported by Olurin et al, Eggleston et al and Kohli et al.⁵⁻⁷ However Edino et al reported a mean age of 21.7 year.⁸

In previous published studies Baliga et al reported M:F ratio of 3.8:1 whereas Shah et al¹⁰ reported M:F of 9:1 where as in study by Edino et al from Nigeria on 50 patients M:F was 4.5:1. Male and female ratio in our study was 1:77:1.^{8,9}

Most common symptoms were acute pain abdomen and fever which were present in almost all patients. Only in 10 patients there was no history of fever. Constipation (60%) abdominal distension (40%) and vomiting (52%) were other predominate complains.

According to Vyas and Prakash et al fever and pain abdomen was present in all cases abdominal distension, constipation and vomiting were other predominant symptoms in other series as well. Guarding, rigidity and tenderness were present in 100% of patient while abdominal distension was present in 40% and bowel sound was absent in 92% cases.^{11,12}

Diagnosis of perforation peritonitis was mainly based upon the history and clinical findings. Presence of free air under diaphragm on upright chest X-ray and Widal test. Another investigation was total leucocyte count and Hb. In our study free gas under diaphragm was found in all cases and Widal test was positive in 68% cases. Despite severe peritonitis 38% patient had, either normal total leucocyte count (18%) or leucopenia 20%. Free gas under diaphragm was reported in different series of enteric perforation peritonitis viz in 75% by Purohit et al and 60% by Prakash et al.^{12,13}

Mehta et al found Widal test positive in only 24% cases, Prakash et al found it positive in 73% cases while Eggleston and Santosh⁶ in 95% cases.^{12,14} Mehta stated there is leucopenia in enteric fever as such as but as the perforation peritonitis occurs leucocytosis occurs.¹⁴

As soon as the patients, were received in the ward, assessment of dehydration and electrolyte imbalance was made preoperative antibiotics were given and correction of fluid and electrolyte imbalance was done Chauhan and Pande et al emphasized necessity of fluid and electrolyte resuscitation of patient with enteric perforation.¹⁵

In our study simple repair of perforation was done in 25 patients. Repair was done in double layer (inner layer) by continuous vicryl and outer by interrupted silk. In 15 patient simple repairs in double layer with freshening of margins of perforation was done. Faecal fistula developed in 2 patients in our series. It followed in one patient after simple closure while other patient had resection and anastomosis. Mortality in our series almost same, irrespective to procedure choose for management of enteric perforation peritonitis.

Overall mortality in our series was 14% (7 patients died out of total 50). Akgun et al, Shah et al, Sahoo et al reported mortality rates of 28.5%, 21.47% and 10% respectively.^{16,10,17} Lower mortality rate in present study may be due to availability of better antibiotics and better preoperative preparation of patient.

Patient of long history of perforation (>5 days) had very high mortality rate 50% as compared to those who presented within in 48 hour was 9.09%. According to Venketramani et al mortality was 18.9% in those patients who presented within 48 hours as compared to 32% in those who presented after 48 hours.¹⁸ Patient with multiple perforations had greater mortality 21.4% than single perforation 11.11% mortality. In our study complication were present in 76% of patient (38 out of 50). Most commonly being wound infection 60% (20 out of 50) wound dehiscence was seen in 16% (8 out of 50). Mortality was recorded in 14% patient (7 out of 50). Venketramani et al reported mortality rate of 52.6% is patient with multiple perforation as compared to those with single perforation (14%).¹⁸

To deal with enteric perforation(s) operative procedure has to be decided to greater caution taking into consideration patient's general condition, gut condition, number and site of perforation and contamination of peritoneal cavity.

CONCLUSION

Enteric perforation peritonitis is a disease with a high mortality rate 14% despite surgical management. Surgical management of enteric perforation peritonitis is associated with 16% (8 patients) incidence of burst abdomen and 4% incidence of fecal fistula. This highlights the importance of choosing correct operative procedures to deal with enteric perforation peritonitis to prevent postoperative burst abdomen and fecal fistula which is associated with high mortality.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Agrawal SC. Chloramphenical resistance of species in the India 1959-61. WHO Bulletin. 1962;27-33.
2. Ajao G. Typhoid perforation: Factor affecting mortality and morbidity. Int Surg. 1982;67:371-9.
3. Bailey H, Mechnell L. Short Practice of Surgery 14th Ed. 1958;910.
4. Butler T, Knight J, Nath SK. Typhoid fever complicated by intestinal perforation : A persisting fatal Disease requiring surgical management. Review of infections Dis. 1985;7:244256.
5. Olurin EO, Ajay OO, Bohrer SP. Typhoid perforation J. Royal Coll. Surg. 1972;12:253-6.
6. Eggleston FC, Santoshi B. Typhoid perforation, Choice of operation. BJS. 1981;68:341-42.
7. Kohli JS, Singh KP. Choice of surgical procedure in typhoid perforation. JIMA. 1991;89(1):255-6.
8. Edino ST, Mohammed AZ, Uba AF, Sheshe AA, Anumah M, Ochicha O, et al. Typhoid enteric perforation in north western Nigeria. Niger J Med. 2004;13(4):345-9.
9. Baliga AV. Surgical Complication of Typhoid. I J Surg. 1949;11:166-77.
10. Shah AA, Wani KA, Wazir BS. The ideal treatment of the typhoid enteric perforation-resection anastomosis. Int Surg. 1999;84(1):35-8.
11. Vyas PM. A study of 15 cases of intestinal perforation in enteric fever. Indian J Surg. 1964;26:1-8.
12. Prakash A, Mittal RN, Ashray SM. Perforation in typhoid enteritis. Surgical J New Delhi. 1972;19.
13. Purohit PG. Surgical treatment of typhoid perforation. ITS. 1978;40:227-38.
14. Mehta VP. Intestinal perforation in enteric fever based on study of 169 cases. Indian J Surg. 1953;4(15):235.
15. Chauhan M.K. and Pande S.K. A review of 344 patients with typhoid Perforation. Br J Surg. 1982;69:173-5.
16. Akgun Y, Bac B, Boylu S, Aban N, Tacyildiz I. Typhoid enteric perforation. Br J Surg. 1997;84(4):584-5.
17. Sahoo SP, Shukla VK, Chauhan VS, Pandey M, Gautam A. Enteric perforation--single-layer closure. Dig Dis Sci. 2004 ;49(1):161-4.
18. Venketramani Sitararn BV, Moses AS, Khanduri FP. Typhoid perforation A retrospective study. Ann Royal College Surgeon Eng. 1990;72:347-9.

Cite this article as: Meghwal SL, Maida ML, Kharadi AL, Pargi MK. Enteric perforation with peritonitis. Int Surg J 2022;9:350-5.