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Original Research Article

A clinical study of generalised peritonitis and its management in a rural setup

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ABSTRACT

Background: Generalised Peritonitis is a common surgical emergency and its treatment remains a challenge despite advances in surgical techniques, antimicrobial therapy and intensive care support. The commonest etiological factors are perforation of hollow viscus and appendicitis. The aim was to study the most common cause of perforation peritonitis, associated risk factors, modes of clinical presentation, management, postoperative complications, and comorbid conditions influencing the morbidity and mortality in rural set up.
Methods: 50 patients of peritonitis of over 10 years of age managed in our institution from July 2015 to November 2016 were studied and followed up on a three-monthly basis for a period varying from 12 months to 2 years with an average of 18 months.
Results: Appendicular perforation was the most common cause of peritonitis followed by peptic ulcer perforation. Perforation peritonitis constituted 26% of total emergency operations performed with a male to female ratio of 2.84:1 and age between 41-50 years. Patients presenting within 24 hours of perforation had an uneventful recovery whereas those presenting after 24 hours had significant postoperative complications. The serum CRP levels provided as good prognostic marker. It remained high in complicated cases. Out of 56% complication rate, wound infection was the commonest.
Conclusions: Early diagnosis and surgical intervention plays a crucial role in early recovery, though the end result depends on many factors like age of the patient, degree of peritoneal contamination and presence of comorbid diseases. This study also highlights the role of CRP as a serum prognostic marker.

Keywords: Appendicitis, CRP, Perforation, Peritonitis, Peptic ulcer

INTRODUCTION

Peritonitis is inflammation of the serous membrane lining the abdominal cavity and the visceral organs within. Peritonitis may be localized or generalized and is classified into primary, secondary and tertiary. Primary spontaneous peritonitis is rare and monomicrobial due to pneumococci or haemophilus bacteria. Secondary peritonitis is due to spread of infection from intra-abdominal organs or as a result of spillage from gastrointestinal or genitourinary tract. Others causes include exogenous contamination.

Tertiary peritonitis refers to recurrence or reactivation of peritonitis following adequate treatment of initial secondary peritonitis. Sir Cuthbert Wallace quotes “it is better to check than be waiting,” do an early surgery rather than wait in a case of peritonitis of unknown cause.

Pathophysiology of peritonitis

The peritoneal cavity is the largest cavity in the body, with a surface area of about 1.0 to 1.7 m^2 of the total body surface area. Inflammation of parietal peritoneum which is richly supplied by somatic nerves causes severe and localised pain. The visceral peritoneum supplied by autonomic nerves, its irritation causes diffuse pain. After bacterial contamination the events that take place include:

- Removal of bacteria from the peritoneal cavity through the diaphragmatic stomata and lymphatics.
- Pro-inflammatory mediators by peritoneal macrophages promote the migration of leukocytes.
- Mast cells release histamine and other vasoactive products, causing local vasodilatation and extravasation.
- Bacterial opsonisation and promote phagocytosis.
- Sequestration of bacteria limiting spread of infection.

Aims and objectives of this study is to evaluate etiology of peritonitis in rural setting, to understand clinical presentation, investigations findings and intra operative findings in peritonitis and to study the co-morbid conditions.

Review of literature

The Hippocratic Facies of terminal stages of peritonitis is well described since Hippocrates (460BC). Peritonitis was first recognized as a disease entity in 1802 by the young French surgeon Bichat and then followed by Laennec.[1] Opium or cathartics were once used to treat peritonitis.[2] Mikulicz, an assistant of Billroth, in 1881 advocated early laparotomy for peritonitis.[3] At the beginning of the 20th century Surgeons had a relatively clear idea about the host defense in the peritoneal cavity.[4] Veillon and Zuber (1893) showed multimicrobial infection in peritonitis.[5] In 1907 Pawlowsky described bacterial translocation of from the gut.[6] The first time the exact bacteriology of peritonitis was reported was in 1922 by Weinberg.[7] Murphy JB advocated early operation, with no sponging or irrigation, closure with drainage and rectal infusion (Murphy drip).[8] In 1926, Kirschner summarized the principles of peritonitis therapy, thereby reducing mortality rate of peritonitis secondary to perforated appendicitis from 83% to 21%, from 100% to 24% due to perforation, and from 100% to 50% due to small and large bowel perforation.[9]

New scoring systems have been described peritonitis.[10,11] Verma and others in PGI, Chandigarh, compared prognostic factors in peritonitis due to trauma.[12] Fungal pathogens were uncommon isolates in the setting of peritonitis.[13]

In a study by Wani R, Parray F, et al on non-traumatic terminal ileal perforation in 79 cases, the causes for perforation were enteric fever (62%), nonspecific inflammation (26%), obstruction (6%), tuberculosis (4%) and radiation enteritis (1%).[14]

Jhobta RS, Attri AK et al studied 504 consecutive cases in India, reported perforated duodenal ulcer (289 cases) and appendicitis (59 cases).[15] In a study in Pakistan on perforation peritonitis, overall mortality was 10.6%.[16] Chakma S et al studied 490 cases of perforation peritonitis found morbidity and mortality 52.24% and 10% respectively.[17]

METHODS

A clinical study of generalized peritonitis was conducted in the Department of Surgery at Adichunchanagiri Hospital and Research Centre, BG Nagara, located in a rural place in Dist. Mandya, Karnataka. After obtaining Hospital ethical committee clearance 50 cases of peritonitis were studied from July 2015 to November 2016 with a period of follow up from 12 months to 24 months on three monthly basis. Recurrence of symptoms, wound infection, incisional hernia, postoperative intestinal obstruction if any were noted.

Inclusion criteria

- All cases of peritonitis/perforation of hollow viscus
- Patients above 10 years age
- Both males and females.

Exclusion criteria

- Below 10years age
- Immunodeficiency disease
- Peritonitis treated conservatively.

Cases of peritonitis based on investigation or peroperative findings were selected.

Investigations included

Blood

Haemoglobin, coagulation profile, blood grouping and Rh typing, total and differential count, ESR, HIV/HBsAg, blood urea and serum creatinine and serum electrolytes. Serum CRP levels were estimated preoperatively and on day 3, day 5 and day 8 postoperatively. WIDAL test was done in suspected typhoid perforations.

Radiological examinations

Erect x ray of abdomen to detect free gas under the diaphragm and ultrasound of the abdomen and pelvis to detect free fluid and any other associated pathology.

After resuscitation and iv antibiotic, surgery was undertaken. The peritoneal fluid was sent for culture and antibiotic sensitivity. After dealing with the primary

pathology, adequate peritoneal lavage was given using warm normal saline and Intra-abdominal drains were placed in all patients and abdomen was closed in single layer.

RESULTS

Perforation peritonitis constituted 26% of total emergency operations performed in our hospital. Age and sex incidence (Table 1 and 2). Male to female ratio was 8:1 in perforation peritonitis.

Table 1: Age incidence of perforation.

Age in years	No. of cases	Percentage
<20	8	16
21-30	4	8
31-40	8	16
41-50	11	22
51-60	8	16
61-70	8	16
>70	3	6

Table 2: Sex incidence.

Sex	No. of cases	Percentage
Males	38	76
Females	12	24

Of the 50 patients, 38 patients were men and 12 patients were women (2.84:1). The mean age of presentation of perforation was between 41-50 years.

Etiology

Appendicular perforation was common in males with male to female ratio of 1.75:1.

Table 3: Etiology of perforation.

Etiology	Males	Females
Peptic ulcer (D+G)	16	2
Appendicular	14	8
Malignant	4	1
Others	4	1

Figure 1: Appendicular perforation.

3 out of 18 patients of peptic ulcer perforation had previous history of peptic ulcer disease. NSAID abuse was present in 8 out of 18 cases of peptic ulcer perforation. History of cigarette smoking was present in 14 out of 18 cases of peptic ulcer perforation (78%).

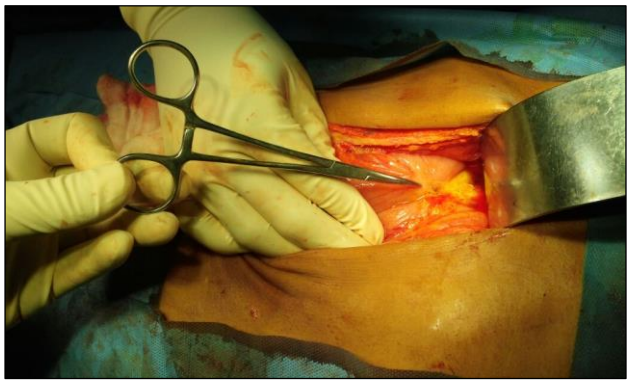


Figure 2: Peptic ulcer perforation.

Figure 3: Ileal perforation.

The peak incidence of perforation was between 41 to 50 years of age. The time delay in presentation was less than 24 hours in 13 patients with a morbidity of 4%, 24-36 hours in 6 patients with a morbidity of 6%, 36-48 hours in 26 patients with a morbidity of 80% and 48-72 hours in 5 patients with a morbidity of 100% (Table 4).

Table 4: Relation between time of presentation and complication rate.

Duration	No. of cases	No. of cases developing complications
24	13	2
36	26	18
48	6	3
72	5	5

Comorbid conditions were Diabetes mellitus (24%), hypertension (12%) and COPD (8%).

Table 5: Comorbid conditions.

Comorbidity	No. of cases	Percentage
Diabetes	12	24
Hypertension	6	12
Copd	4	8

Clinical presentation (Table 6 and 7). Pain abdomen, fever, vomiting and distension were the predominant symptoms. Pain abdomen was present in all, vomiting in (55%), fever in 52% and distension of abdomen in 48%.

Table 6: Clinical presentation.

Symptoms	No. of cases	Percentage
Pain abdomen	50	100
Distension	26	52
Vomiting	50	100
Fever	36	72
Constipation	24	48

Table 7: Clinical signs on presentation.

Signs	No. of cases	%
Dehydation	42	84
Shock	8	16
Tachycardia	50	100
Tachypnoea	17	34
Tenderness	50	100
Rebound tenderness	42	84
Guarding/rigidity	32	64
Obliteration of liver dullness	22	44
Absent bowel sounds	28	56

Clinical Signs on presentation (Table 7). Tachycardia was seen in all (100%), Dehydration in 84%, tachypnoea in 34% and 16% presented with shock. Tenderness was present in all (100%), rebound tenderness in 84%, Obliteration of liver dullness in 44% and absent bowel sounds in 56%. Investigations - Pneumoperitoneum in relation to aetiology (Table 8 and Figure 4)

Total count was elevated in all patients. Renal function tests were impaired in 15 patients and all of these 15 patients developed complications in the postoperative period.

Table 8: Pneumoperitoneum in relation to aetiology.

Aetiology	No.of cases	Percentage
Peptic ulcer (D+G)	18	36
Appendicular	0	0
Malignant	5	10
Others	5	10

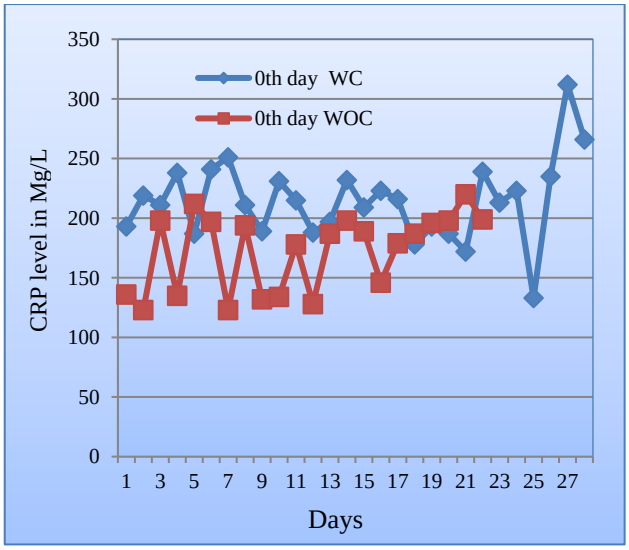
Erect x ray of the abdomen showed evidence of pneumoperitoneum in 28 patients.

Figure 4: Erect X-ray abdomen showing pneumoperitoneum.

All patients had abdomino-pelvic ultrasound scans which was positive for free peritoneal fluid and other features suggestive of peritonitis.

Treatment

The nature of peritoneal exudate was scanty thin seropurulant in early cases and frank pus with fibrinoid adhesions in all patients who presented after 48 hours. After definitive surgery, adequate peritoneal lavage using warm normal saline was given, intra-abdominal drains placed in all and abdomen was closed in single layer.



WC: with complications; WOC: without complications

Figure 5: CRP values on day 0.

Serum C reactive protein levels were estimated preoperatively and on postoperative day 3, day 5 and day 8. The serum CRP levels were significantly elevated in all patients at the time of admission. In patients who had an uncomplicated postoperative recovery, the serum CRP levels gradually decreased over a period of time.

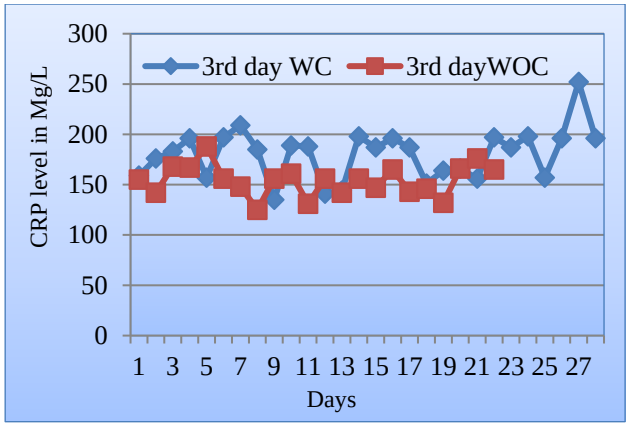


Figure 6: CRP values on day 3.

Patients with postoperative complications had significantly higher serum CRP levels preoperatively which persisted in the postoperative period (Figure 5, 6, 7 and 8).

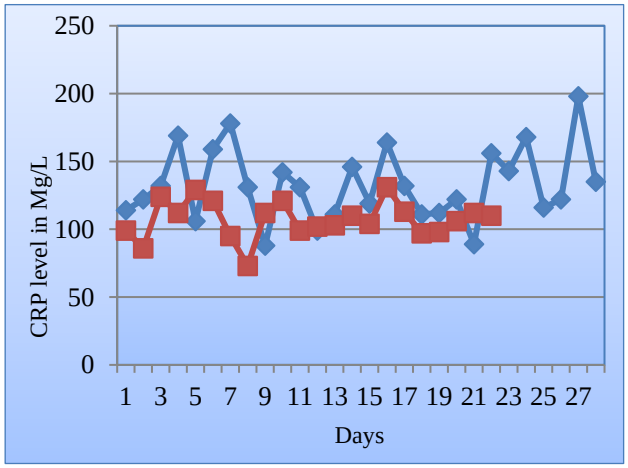


Figure 7: CRP levels on day 5.

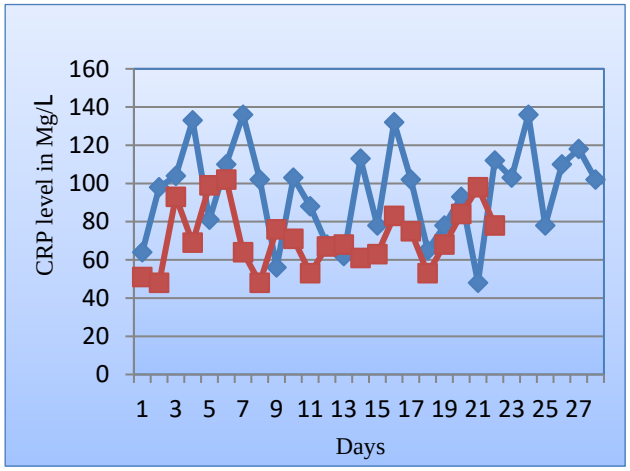


Figure 8: CRP levels on day 8.

Peritoneal culture grew *Escherichia coli* in 37 patients, *Klebsiella* in 29, *Proteus sp* in 9, *Enterococcus* in 8 and *Enterobacter sp* in 5 patients. The mean hospital stay for patients without postoperative complications was 8 days. Among the 50 patients, 8 patients presented with shock at the time of admission. 2 of these patients required postoperative mechanical ventilation and 3 patients required ionotropic support.

Table 9: Postoperative complications.

Complication	Number	%
Wound infection (WI)	23	46
Residual collection (RC)	2	4
Respiratory infections (RI)	12	24
Hypertensive crisis (HTNC)	1	2
Incisional hernia (I)	2	4
Prolonged paralytic ileus (PP)	2	4

28 patients (56%) developed postoperative complications, with more than one complication in some. Wound infection was the most common complication in 23 patients (43%). Respiratory infections in 12 patients (24%), residual collection in 2 (4%), incisional hernia in 2 (4%), paralytic ileus in 2 (4%) and hypertensive crisis in one patient (2%) were other complications

The mean hospital stays for patients who developed complications was 15.3 days. Age >50 years, time of presentation >24 hours, presence of shock and comorbid illness were significant factors associated with postoperative morbidity.

DISCUSSION

26% of emergency operations in our hospital in the department of general surgery were for peritonitis secondary to hollow viscus perforation. This finding is comparable with many other studies. Arveen et al in their study between 2006-08 at JIPMER, reported an incidence rate of 25%.[19] In a study conducted in Netherlands there was a marginal increase in incidence of peptic ulcer perforation.[20]

Etiological factors

A study of 204 consecutive cases of perforation peritonitis conducted by Khanna et al showed that 108 cases were due to typhoid. Others included duodenal ulcer 58), appendicitis (9), amoebiasis (8) and tuberculosis (4).[21]

In a study of peritonitis by Thirumalagiri, Duodenum (52%) was the most common site of perforation followed by ileal perforation (26%) appendicular (14%) and colonic perforation (4%). 84% of the patients were male patients and 16% of the patients were females. Duodenal ulcer (52%) was the most common cause of performative peritonitis followed by small intestinal perforation.[22] Whereas trauma is an important etiological factor of perforation peritonitis in the developed countries.

In present study of 50 cases, peritonitis due to appendicular perforation was found to be the highest constituting 44% of the cases (Table 3) This result was similar to that Noon et al and Akcay et al who reported that 21% and 18% of their cases were due to appendicular perforation respectively.[23]

This is in contrast to many other studies from India which conclude proximal gastroduodenal tract perforation to be more common. Jhobta et al, reported 57.4%, Chakma et al and Afridi et al 54.29% and 45% and Yadav et al reported 29% cases of perforation among their studies.[24]

The next common cause of perforation peritonitis in present study was peptic ulcer perforation (36%), out of which gastric ulcer perforation constituted 8% and duodenal ulcer perforation constituted 28%. (Table 3) Duodenal to gastric ulcer ratio was 4.5:1 in this study which is in comparison to other studies from India (Jhobta et al duodenal to gastric ulcer ratio of 7:1).

Other causes in present study were malignant perforation (10%) and perforation due to other causes (10%) which included ileal (4%) of unknown aetiology, jejunal diverticula with perforation (2%) and sigmoid volvulus with perforation (4%). (Table 3) Gastric carcinoma presenting with perforation peritonitis accounted for 6%. Colonic cancer-causing perforation was present in 4% of our patients Yadav et al in their study reported a rate of 2.6% due to malignant perforation.

NSAID abuse was present in 8 out of 18 cases of peptic ulcer perforation accounting for 44% cases. Bali et al reported that 15% of their cases had a positive history of NSAID abuse for more than 6 months.[25] Only 16% of the patients with peptic ulcer perforation had past history of chronic Peptic ulcer disease. Ugochukwu, A. et al in their study reported that 31.6% of their patients had a past history of chronic PUD. [26] The lack of prior diagnosis of PUD may be due to the negligence of warning symptoms.

Age incidence

In present study, the maximum incidence of perforation irrespective of the aetiology was found to be between the age of 41-50 years accounting for 22% of the cases. In a study conducted by Arveen et al, the mean age of the patients was reported to be 43.4 years.

Jhobta et al in their study reported a mean age of 36.8 years, while Ugochukwu et al reported a mean age of 39.5 years. In study conducted by Chakma et al on 490 cases of perforation peritonitis, they reported the mean age of the patients to be 48.28 years.

Peptic ulcer perforation was more common between the age group of 41 to 50 years, (Table 3 and Figure 3) which is in agreement with other studies. Kocer B et al reported an overall mean age of 43.41±18.66 years.[27] Tas et al in their study reported a mean age of 51.7±20 years.[28]

Sex incidence

The ratio of male to female cases irrespective of the aetiology of perforation was 2.84:1 Yadav et al n their study reported a male to female ratio of 4.9:1. In another study conducted in Pakistan the male to female ratio was 2.1:1. These findings are in comparison to present study.

Clinical presentation

Pain abdomen was the predominant symptom in present study. Pain abdomen was seen in all cases (100%). In a study conducted by Ugochukwu et al pain abdomen was the most common symptom present in 90.8% of their patients. In another study conducted by Sivaram et al pain abdomen was present in 100% of their patients.[29]

Vomiting and fever were the next most common symptom present in 55% and 52% respectively in present study which is comparable to the findings by Jhobta et al and Sivaram et al (59% and 48.5% respectively) and fever was present in 25% and 33.7% of their cases respectively. Distension of abdomen was present in 48% of our patients compared to 44% and 73.9% in studies conducted by Jhobta et al and Yadav et al respectively.

Time of presentation

Ugochukwu. et al and Chakma. et al reported that the postoperative morbidity and mortality was high in patients presenting late. In present study, 52% patients presented between 36-48 hours after onset of symptoms, less than 24 hours in 26%, 24-36 hours in 12% and 48-72 hours in 10%. Patients presenting within 24 hours of perforation had an uneventful recovery whereas those presenting after 24 hours had significant postoperative complications, which is comparable to the study conducted by Unver M et al and Noguiera C et al.[30,31]

In present study the mean time lag between onset of symptoms and definitive treatment was 42.72 hours which explains the high morbidity rate of 56%. This finding is comparable to studies conducted by Jhobta et al and Chakma et al who reported a complication rate of 49.8% and 52.24% respectively. Chakma et al reported a mean duration of presentation of 57.4 hours.

The clinical appraisal of inflammatory markers (CRP and Procalcitonin) as prognostic markers has gained considerable interest. Reith and colleagues in their study reported that serum CRP and PCT levels were superior to TNF-α, IL-1 and IL-6 levels in predicting prognosis in 246 patients with abdominal sepsis.[32] The serum CRP levels were significantly elevated in our patients at the time of admission (Figure 5). In patients who had an uncomplicated postoperative recovery, the serum CRP levels repeated on day 3, day 5 and day 8 gradually decreased (Figure 6, 7 and 8) but remained high in those with complications. High postoperative levels corresponded with high preoperative levels (Figure 5, 6, 7

and 8). Shelton J et al reported that a CRP level of >150mg/L is associated with increased complication rate postoperatively.[33] Suh S et al reported that serum CRP levels are sensitive markers to differentiate a perforated from non-perforated appendicitis. They found mean CRP levels in patients with perforated appendicitis was 43.4 mg/L while in non-perforated appendicitis was 11.88 mg/L.[34] Kaya B et al reported that high CRP levels help to differentiate between phlegmonous appendicitis from perforated appendicitis.[35]

Management

Perforated appendicitis with peritonitis underwent appendicectomy and peritoneal toilet (44%). Patients with duodenal ulcer perforation were managed by simple closure of the perforation with omentopexy (28%) which was also stated by Khalil et al and Plummer JM et al.[36] In patients with gastric ulcer perforation, biopsy of the ulcer site was taken followed by simple closure and omentopexy (8%). All our patients had an adequate peritoneal followed by closure in single layer after placing drains. Anti H pylori therapy was administered to all peptic ulcer perforation patients for a period of 14 days in the postoperative period.

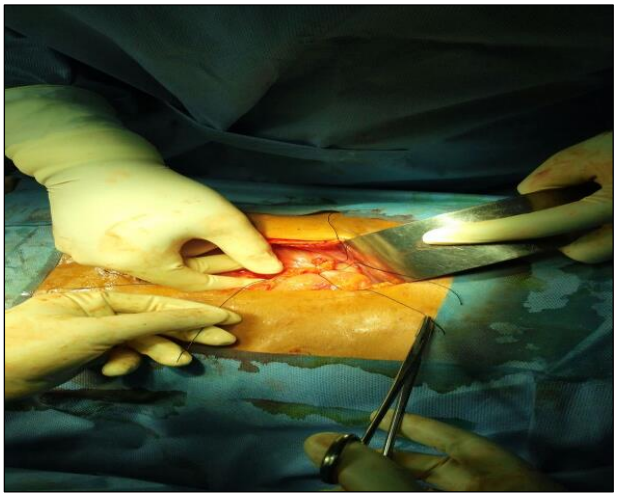


Figure 9: Graham's patch repair.

No definitive antiulcer surgery was undertaken. Peritoneal soiling precludes any type of definitive antiulcer surgery, which also has been stated by Ugochukwu et al and Khalil et al. Patients with malignant perforation underwent limited or definitive resection. Distal radical gastrectomy was done for malignant gastric ulcer perforation in one patient.

In other two patients plugging of perforation with momentum and anterior gastrojejunostomy was done as the growth was found to be unresectable. One Colonic malignancy underwent radical right hemicolectomy with ileostomy and in other splenic flexure growth was found unresectable, so biopsy, closure of perforation, peritoneal toilet and ileostomy was done. Subsequently the patient underwent definitive surgery after neoadjuvant chemotherapy. One jejunal diverticula with perforation underwent resection and end to end anastomoses. Two other patients with ileal perforation of unknown aetiology underwent resection with end to end anastomoses and proximal enterostomy.

Seropurulent exudate was seen in 45 patients (90%) who presented within 48hours of perforation whereas frank pus with fibrinous adhesions was found in 5 patients (10%) who presented after 48 hours of perforation and they had higher morbidity (100%). Similar results were observed by Ugochukwu et al who reported a complication rate of 63.2% in those presenting late.

Spectrum of organisms

Peritoneal culture was *Escherichia coli* in 37 patients, *Klebsiella* species in 29, Proteus sp in 9, *Enterococcus* in 8 and *Enterobacter sp* in 5 patients. Boueil A et al in their study reported Escherichia coli (81%), *Streptococcus milleri* group (12%), and *Pseudomonas aeruginosa* (12%).[37]

Postoperative morbidity

The overall complication rate in our patients was 56%. Jhopta et al from India had a complication rate of 49.8% which is comparable to present study. Wound infection was the most common complication in this study in 43% (Table 9 and figure 8), which is comparable to another study having 39.5% postoperative complications.[38] Study by Chaiya et al reported a surgical site infection rate of 48%.[39] Respiratory infections in our 12 patients (24%) was comparable to a rate of 28% in the study conducted by Jhobta et al.

Intra-abdominal abscess was seen in 2 patients (4%), incisional hernia in 2 patients (4%), prolonged paralytic ileus in 2 patients and hypertensive crisis in one patient (2%). These were amongst the other complications in our patients.

The high incidence of postoperative complications in this study may be attributed to the late presentation of patients (>24 hours). This is comparable to a study conducted by Chakma et al who reported a complication rate of 52.24% and a mean duration of presentation of 54.7 hours. The sex of the patient did not have any influence on the complication rate in present study. Other factors for this high complication rate included age more than 50 years, shock at the time of presentation and presence of comorbid illness. 32% of our patients had Diabetes mellitus, hypertension and COPD (Table 5).

This is in agreement with a study conducted by Sivaramet al, Noguiera et al and Montalvo et al.[40] 16% of the patients in this study presented with shock. It was found that the presence of shock increased the need for postoperative mechanical ventilation and ionotropic

support thus increasing morbidity and hospital stay. This finding has also been earlier reported by Tas et al in their study. Abnormal renal function tests at the time of admission was an additional risk factor for postoperative morbidity and longer hospital stay (Sivaram et al).[29]

CONCLUSION

The spectrum of generalised peritonitis secondary to hollow viscus perforation continues to vary from one part of the world to another. In the developing countries the proximal gastrointestinal tract is the most common site of perforation whereas in the developed countries perforation of distal gastrointestinal tract of traumatic etiology is most common.

In rural setting like ours Appendicitis causing peritonitis was commoner than ulcer perforation and is due to delay in presentation resulting in perforation peritonitis. This study also highlights the role of serum prognostic markers like CRP to assess the prognosis of the patients. The need for early recognition and prompt surgical intervention to reduce the morbidity and mortality cannot be overemphasized.

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REFERENCES

1. Carson H. The evolution of the modern treatment of septic peritonitis. Lancet. 1923;201(5203):1035-8.
2. Price M. Peritonitis, from a surgical standpoint. J Am Med Assoc. 891;17(5):173-6.
3. Curtis BF. The surgical treatment of peritonitis. Ann Surg. 1887;5:22-31.
4. Durham HE. The mechanism of reaction to peritoneal infection. J Pathol. 1897;4(3):338-82.
5. Steinberg B. Peritoneal exudate: a guide for the diagnosis and prognosis of peritoneal conditions. JAMA. 1941;116(7):572-8.
6. Pawlowsky AD. The fate of some pathogenic (primarily pyogenic) microbes when they enter the animal organism from the joints, the pleura, the eye, the oral cavity, the intestinal canal and the vagina. Medical Microbiol Immunol. 1909;62(1):433-52.
7. Weinberg M, Prevot AR, Davesne J, Renard C. Research on the bacteriology and serotherapy of acute appendicitis. Ann Int Pastor. 1928;42:1167-241.
8. Murphy JB. IV treatment of perforative peritonitis: general free suppurative. Ann Surg. 1908;47(6):870.
9. Kirschner M. The treatment of acute purulent free peritonitis. Arch Klin Chir. 1926;142:253-311.
10. Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. Critical Care Med. 1985;13(10):818-29.
11. Ohmann C, Yang Q, Hau T, Wacha H. Prognostic modelling in peritonitis. Peritonitis Study Group of the Surgical Infection Society Europe. Eu J Surg.1997;163(1):53-60.
12. Verma GR. Gastro-intestinal injuries in abdominal trauma. Trop Gastroenterol 1990;11(4):206-10.
13. Salvaggio MR, Pappas PG. Current concepts in the management of fungal peritonitis. Current infectious disease reports. 2003;5(2):120-4.
14. Wani RA, Parray FQ, Bhat NA, Wani MA, Bhat TH, Farzana F. Nontraumatic terminal ileal perforation. World J Emerg Surg. 2006;1(1):7.
15. Jhobta RS, Attri AK, Kaushik R, Sharma R, Jhobta A. Spectrum of perforation peritonitis in India-review of 504 consecutive cases. World J Emerg Surg. 2006;1(1):26.
16. Afridi S, Malik P, Ur-Rahman S, Shamim S, Samo K. Spectrum of perforation peritonitis in Pakistan: 300 cases Eastern experience. World J Emerg Surg. 2008;3(1):31.
17. Chakma SM, Singh RL, Parmekar MV, Singh KG, Kapa B, Sharatchandra KH, Longkumer AT. Spectrum of perforation peritonitis. J Clin Diagnostic Res: JCDR. 2013;7(11):2518-20.
18. Shaikh A, Shaikh SF, Shaikh SM. Common factors leading to re-laparotomy in secondary peritonitis. Rawal Med J. 2012;37(2):161-4.
19. Arveen S, Jagdish S, Kadambari D. Perforated peptic ulcer in South India: an institutional perspective. World J Surg. 2009;33(8):1600-4.
20. Noon GP, Beall AC, Jordan GL, RIGGS S, DeBakey ME. Clinical evaluation of peritoneal irrigation with antibiotic solution. Surg. 1967;62(1):73.
21. Khanna A, Misra M. Typhoid perforation of the gut. Postgraduate Med J. 1984;60(706):523-5.
22. Thirumalagiri VR, Chandra H. Acute peritonitis secondary to hollow viscous perforation: a clinical study. Int Surg J. 2017;4(7):2262-9.
23. Akcay I, Okoh A, Yalav O, Eray I, Rencuzogullari A, Dalci K. The prognostic value of Pro-Calcitonin, CRP and thyroid hormones in secondary peritonitis: a single-center prospective study. Turkish J Trauma Emerg Surg. 2014;20(5):343-52.
24. Yadav D, Garg P. Spectrum of perforation peritonitis in Delhi: 77 cases experience. Indian J Surg. 2012;75(2):133-7.
25. Bali R, Verma S, Agarwal P, Singh R, Talwar N. Perforation peritonitis and the developing world. ISRN Surg. 2014;2014:1-4.
26. Ugochukwu A, Amu O, Nzegwu M, Dilibe U. Acute perforated peptic ulcer: on clinical experience

in an urban tertiary hospital in south east Nigeria. Int J Surg. 2013;11(3):223-7.
27. Kocer B, Surmeli S, Solak C, Unal B, Bozkurt B, Yildirim O. Factors affecting mortality and morbidity in patients with peptic ulcer perforation. J Gastroenterol Hepatol. 2007;22(4):565-70.
28. Tas I, Ulger B, Onder A, Kapan M, Bozdag Z. Risk factors influencing morbidity and mortality in perforated peptic ulcer disease. Turkish J Surg. 2015;31(1):20-5.
29. Sivaram P, Sreekumar A. Preoperative factors influencing mortality and morbidity in peptic ulcer perforation. Eu J Trauma Emerg Surg. 2017;44(2):251-7.
30. Unver M, Fırat Ö, Ünalp Ö, Uğuz A, Gümüş T, Sezer T, et al. Prognostic factors in peptic ulcer perforations: a retrospective 14-year study. Int Surg. 2015;100(5):942-8.
31. Noguiera C, Silva A, Santos J, Silva A, Ferreira J, Matos E, et al. Perforated peptic ulcer: main factors of morbidity and mortality. World J Surg. 2003;27(7):782-7.
32. Reith H, Mittelkötter U, Wagner R, Thiede A. Procalcitonin (PCT) in patients with abdominal sepsis. Intensive Care Med. 2000;26(15):S165-9.
33. Shelton J, Brown J, Young J. Preoperative C-reactive protein predicts the severity and likelihood of complications following appendicectomy. Ann Royal Coll Surg England. 2014;96(5):369-72.
34. Suh S, Choi Y, Park J, Kim B, Cha S, Park S, et al. Clinical factors for distinguishing perforated from nonperforated appendicitis. Surgical Laparoscopy, Endoscopy Percutaneous Tech. 2011;21(2):72-5.
35. Kaya B, Sana B, Eris C, Karabulut K, Bat O, Kutanis R. The Diagnostic Value of D-dimer, Procalcitonin and crp in acute appendicitis. Int J Med Sci. 2012;9(10):909-15.
36. Plummer JM, McFarlane ME, Newnham. Surgical management of perforated duodenal ulcer: the changing scene. West Indian Med J. 2004;53:378-81.
37. Boueil A, Guégan H, Colot J, D'Ortenzio E, Guerrier G. Peritoneal fluid culture and antibiotic treatment in patients with perforated appendicitis in a Pacific Island. Asian J Surg. 2015;38(4):242-6.
38. Ohmann C, Yang Q, Hau T, Wacha H. Prognostic modelling in peritonitis. Peritonitis study group of the surgical infection society Europe. Eu J Surg.1997;163(1):53-60.
39. Chalya P, Mabula J, Koy M, Mchembe M, Jaka H, Kabangila R, et al. Clinical profile and outcome of surgical treatment of perforated peptic ulcers in north western tanzania: a tertiary hospital experience. World J Emerg Surg. 2011;6(1):31.
40. Montalvo-Javé EE, Corres-Sillas O, Athié-Gutiérrez C. Factors associated with postoperative complications and mortality in perforated peptic ulcer. Cirugia Y Cirujanos. 2011;79(2):141-8.

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