Original Article

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A Study on Mortality among Admitted Cases of Acute Peritonitis at a Tertiary Care Hospital

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Abstract

Introduction: Primary peritonitis results from bacterial, chlamydial, fungal, or mycobacterial infection in the absence of perforation of the gastrointestinal tract. Secondary peritonitis occurs in the setting of gastrointestinal perforation. Frequent causes of secondary bacterial peritonitis include peptic ulcer disease, acute appendicitis, colonic diverticulitis, and pelvic inflammatory disease. *Methodology:* This study comprises of 50 cases of acute peritonitis coming to Medical College & Hospital. A pre-tested proforma was used to collect the relevant information by history, clinical examination of patients, relevant investigations required and treatment. Patients were admitted as and when they presented with the following inclusion and exclusion criteria. *Results:* Total of 6 patients expired. 4 patients died of septicemia and multiple organ failure. Mortality in duodenal perforation cases is 7.2% and in ileal perforation cases its 50%. Only 1 case of colonic perforation present so mortality cannot be considered. 2 patients of duodenal perforation died of septicemia and multiple organ failure. A case of colonic perforation died of myocardial infarction. *Conclusion*: Mortality is proportional to age, derangement of physiological parameters like hypotension, delay in surgery and as perforation site becomes distal from duodenum to colon.

Keywords: Acute Peritonitis; Mortality; Perforation.

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Introduction

Peritonitis is inflammation of the peritoneum and peritoneal cavity and is most commonly due to a localized or generalized infection. Primary peritonitis results from bacterial, chlamydial, fungal, or mycobacterial infection in the absence of perforation of the gastrointestinal tract. Secondary peritonitis occurs in the setting of gastrointestinal perforation. Frequent causes of secondary bacterial peritonitis include peptic ulcer disease, acute appendicitis, colonic diverticulitis, and pelvic inflammatory disease [1].

Peritoneal infection is usually caused by two or more bacterial strains. Gram-negative bacteria contain endotoxins (lipopolysaccharides) in their cell walls that have multiple toxic effects on the host, primarily by causing the release of tumour necrosis factor (TNF) from host leucocytes.

Systemic absorption of endotoxin may produce endotoxic shock with hypotension and impaired tissue perfusion. Other bacteria such as Clostridium welchii produce harmful exotoxins. Bacteroides are commonly found in peritonitis. These Gram negative, nonsporing organisms, although predominant in the lower intestine, often escape detection because they are strictly anaerobic and slow to grow onculture media unless there is an adequate carbon dioxide tension in the anaerobic apparatus (Gillespie) [2,3].

Most duodenal perforations are initially sterile for up to several hours, and many gastric perforations are also sterile at first; intestinal perforations are usually infected from the beginning. The proportion of anaerobic to aerobic organisms increases with the passage of time.

Mortality Reflects

- Degree and duration of peritoneal contamination
- Age of the patient
- General health of the patient
- Nature of the underlying cause

Secondary peritonitis is a severe clinical situation caused by the rupture of a viscus or abscess that allows free entry of bacteria into the peritoneal cavity, followed bydeterioration of general condition of patient of haemodynamic parameters, follows shock.

Dinesh et al noticed cause of perforation peritonitis was duodenal ulcer (26.4%), ileal typhoid (26.4%), small bowel tuberculosis (10.3%), stomach (9.2%) and acute appendicitis (5%).

Peritoneal clearance of bacteria: Once the bacteria enter the peritoneal cavity, clearance of the offending micro-organisms begins immediately; contaminating material is swept into the subdiaphragmatic space along the paracolic gutters, to large lacunae opening into specialized lymphatic channels in the diaphragm. Decade before it was known that the diaphragm was the predominant site of clearance of bacteria [4,5].

Fowler in 1900 proposed head up pelvis down position for prevention of absorption of toxins from the infected peritoneal cavities.

When the inflammation develops gradually the peritoneum has effective methods of localization, the inflammation quality inhibits the fibrinolytic activity of peritoneal exudate enabling adjacent omentum and loops of bowel to adhere and surround the inflamed organ. When the affected organ perforate, there is an outpouring of serous inflammatory exudate rich in leucocytes and plasma proteins that soon becomes turbid; the turbid fluid becomes frank pus., the pus is localised forming an intraperitoneal abscess. Thus an intraperitoneal abscess may be considered a relatively successful response of the peritoneal defence system [6].

Methodology

This study comprises of 50 cases of acute peritonitis coming to Medical College & Hospital. A pre-tested proforma was used to collect the relevant information by history, clinical examination of patients, relevant investigations required and treatment.

Patients were admitted as and when they presented with the following inclusion and exclusion criteria.

Inclusion Criteria

In study, all the cases that were provisionally diagnosed with acute peritonitis and subjected to relevant investigations and underwent surgery were included.

Exclusion Criteria

- Cases who were ruled out after investigations
- Cases in paediatric age group (<15 years) as they come under superspeciality.
- Cases that were treated conservatively.
- Cases who refused surgery
- Cases unfit for surgery

Cases clinically diagnosed as peritonitis underwent X-ray erect abdomen, and blood investigations like CBC, Blood urea, serum creatinine, urine routine and microscopy. Serum amylase and widal test was done if pancreatitis or enteric fever was suspected respectively.

After stabilization, patients were taken up for surgery. Laparotomy was done under general anaesthesia or epidural anaesthesia.

Results

60% of patients had duration of illness more than 24 hours by the time they were taken for surgery. It was mainly because of late presentation to the hospital.

Table 1: Duration of Illness

Duration in hours	No. of Cases	Percentage
≥ 24	30	60
≤ 24	20	40

Table 2: Postoperative complications — General

Complications	No. of Cases	Percentage
Respiratory	2	4
Septicemia	6	12
Renal	2	4
Cardiac	1	2
Total	**11**	**22**

12% of patients had persistent septicemia in post operative period. They were managed with antibiotics, IV fluids and blood transfusions.

Two cases developed acute renal failure and needed dialysis. One case had cardiac complication in the form of ischemic changes

Two patients had respiratory complications. All were smokers and developed pneumonia and diagnosed clinically and on chest – x-ray.

Total of 6 patients expired. 4 patients died of septicemia and multiple organ failure. Mortality in duodenal perforation cases is 7.2% and in ileal perforation cases its 50%. Only 1 case of colonic perforation present so mortality cannot be considered. 2 patients of duodenal perforation died of septicemia and multiple organ failure. A case of colonic perforation died of myocardial infarction.

The group of patients in whom onset of symptoms was present more than twenty four hours before surgery, had higher number of death. Even case fatality rate was higher in this group which was 16%. It was more than double of the group in whom surgical intervention was done early.

Mean age of survivors is 35 years while mean age of patients who expired is higher i.e. 51 years.

Table 3: Mortality in relation to cause

Cause	Mortality	Percentage
Gastric perforation	0	0
Duodenal perforation	2	7.2
Jejunal perforation	0	0
Ileal perforation	2	50
Appendicular perforation	0	0
Colonic perforation	1	100
Intestinal gangrene	1	25

Table 4: Mortality in relation to duration of illness

Duration in hours	No. of Cases	Death	CFR%
≥ 24	30	5	16
≤ 24	20	1	5

Table 5: Mortality in relation to age of patients

No. of cases	Mean age	Range
Expired	51years	28-65
Survivors	35 years	15-74

Discussion

In present study mortality rate, was 12%, while in LA Desa [7] series it was 24.8%, and in Kachroo series it was 8.8%.

In present study mean age of patients expired was higher than mean age in survivors i.e. 51 years and 35 years respectively.

Hunt [8] in a study of 54 patients with generalized peritonitis found that average age of patients who expired was 62 years compared to 49 years in those who survived.

In study LA Desa [7] mean age of those who succumbed was 49.2 years.

In present study patient in whom duration of illness was more than 24 hours, case fatality rate was double than those who were operated early, i.e., 16% and 5% respectively. Patients presented late because of poor socioeconomic status, poor transportation facility and lack of surgical facility in near by areas.

In present study four patients died of septicemia and multiple organ failure.

Bolon [9] in a study of 176 patients with peritonitis found that in patients with one organ failure and delay in surgery for more than 24 hours of onset of peritonitis, the mortality was 88%.

In present study mortality rate from duodenal ulcer perforation, intestinal gangrene and ileal perforation was 7.2%, 25% and 50% respectively. There was no mortality in gastric ulcer perforation and appendicular perforation patients.

A Study by R. Goris [10] reported a mortality rate of 9% to 40% in perforated gastric ulcer or duodenal ulcer cases.

Miller and Wich in a study of 118 cases of free perforation of colon reported mortality of 30%.

Conclusion

Mortality rate was 12% in this study with septicemia and multiple organ failure being the commonest cause of mortality.

Mean age of patients who expired was 51 years while those of survivors were 35 years.

Mortality was clearly more in patient in whom surgery was delayed for more than 24 hours.

Hence from my study it can be concluded that prompt resuscitation and early surgical intervention can reduce morbidity and mortality associated with peritonitis.

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