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Study Of Factors Involved In Perforative Peritonitis And Its Significance To Mortality And Morbidity

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ABSTRACT

Peritonitis caused by gastrointestinal perforation is one of the most prevalent surgical emergencies worldwide. Early detection and treatment of Perforation Peritonitis may significantly minimise morbidity and death. The result is improved by surgical treatment and good perioperative care. Nowadays, conservative trials have minimal function in perforation peritonitis.

Extremes of age, delayed presentation, distal GI (colonic) perforation, comorbidities, sepsis, faecal peritonitis, and other factors all enhance mortality. Careful clinical examination and post-operative assessment aid in the stratification of high-risk patients in order to offer better treatment for them. [1-2] Peritonitis is a surgical emergency that necessitates patient resuscitation, laparotomy and peritoneal toilet insertion, omental patch application, and, in certain cases, surgery for ulcer management. [3-4] It has been well documented that the majority of perforated peritonitis patients in our subcontinent appear late. Typically, these patients have well-established widespread peritonitis with purulent faecal pollution and septicaemia, increasing the risk of morbidity and death and complicating the challenge of providing appropriate perioperative care. [4]

INTRODUCTION

Despite breakthroughs in diagnosis, surgery, antibiotic treatment, and critical care support, peritonitis remains a potentially lethal condition. Peritonitis caused by hollow viscus perforation is widespread in this nation, and the aetiology spectrum in tropical countries continues to diverge from that in Western countries. The World Society of Emergency Surgery [WSES][7] has produced an evidence-based guideline for the care of such patients, stating that the source of infection for intra-abdominal sepsis should be addressed as soon as possible. The recommended surgical method is determined by the anatomical location of the perforation, the degree of peritoneal inflammation, the generalised septic reaction, the patient's underlying illnesses, and the treatment center's available resources. Early surgical intervention under the cover of broad-spectrum antibiotics, followed by appropriate vigorous resuscitation and electrolyte balance correction, is critical for a positive result. [8]

Objectives

1. To study the etiopathogenesis of perforative peritonitis
2. To study the rate of mortality and morbidity in cases of perforative peritonitis
3. To analyze different factors concerned with perforative peritonitis

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Review of Literature

Surgeons have attempted for 100 years to cure the duodenal ulcer by reducing the secretion of acid and pepsin, and history of surgery for peptic ulcer is a chronicle of their attempts to achieve this aim without producing major disturbance to the functions of alimentary tract.

1726: George Hamberg (Germany) described a duodenal ulcer

1793: Jacopo Penada (Italy) first recorded a duodenal perforation

1881: Theodor Billroth, Father of Surgical Audit and Father of Abdominal surgery, performed the excision of distal part of the stomach with anastomosis of the gastric stump to the duodenum (Billroth I Surgery).

1888: Mikulicz redefined the pyloroplasty done by Heineke

1893: Barling, of Great Britain, treated perforated ulcer by closure and vigorous lavage of peritoneal cavity with large quantity of saline

1937: Cellian-Jones and Graham popularized the effectiveness of omental patch forperforation

1948: Frank son of Stockholm first reported selective vagotomy. 1965: Erik Amdrup performed highly selective vagotomy.

1985: Barry Marshall cultured Helicobacter pylori.

Under normal conditions, < 50 ml of sterile fluid is present within the peritoneal cavity – secreted from the visceral peritoneal surfaces; the fluid is circulated through the peritoneal cavity. The cephalad movement proceeds along the paracolic gutter and subhepatic spaces – due to negative pressures in the subphrenic area by diaphragmatic motion. Peritoneal fluid is mostly absorbed into the lymphatic circulation via the parietal peritoneal surfaces, with the remainder absorbed through diaphragmatic lymphatics. The clearance of particulate matter, cells and microorganisms is largely dependent upon diaphragmatic lymphatics. The diameter of these lymphatic stomata can be varied by diaphragmatic stretching and contraction, from 4 to 12 microns. In addition, in the presence of inflammation the patency of stomata may be increased to augment the clearance function of the diaphragm. At inspiration, contraction of the diaphragm empties the lacunae into efferent lymphatic channels. Negative intrathoracic pressure during inspiration facilitates fluid movement into thoracic lymphatic channels, and ultimately delivered to the central circulation via the thoracic duct. Following the intraperitoneal injection of bacteria, organisms can be recovered from right thoracic duct within 6 min, and from blood within 12 minutes.[16-17]

Local Reponse to peritoneal Infection

Any noxious stimulus like endotoxin associated with gram negative bacteria, gram positive bacteria, bacteroides species, irritants such as gastric juice, bile salts and meconium probably incite the inflammatory process by inciting mesothelial cell damage or direct activation of the complement system. Following activation the peritoneal inflammatory process is composed of changes in blood flow, the enhancement of bacterial phagocytosis and fibrin deposition to contain or trap bacteria.

Systemic Response to Peritoneal Infection

Abdominal distention secondary to accumulated fluid within the peritoneal cavity – creates

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restriction to diaphragmatic mobility and decreases ventilatory volume, creating eventual atelectasis. The accumulation of fluid in the pulmonary interstitium and alveoli decreases pulmonary compliance and decreased work of breathing. Early manifestation is hyperventilation and the development of respiratory alkalosis. With the worsening of the pulmonary edema and alveolar collapse; severe hypoxemia will develop, creating the adult respiratory distress syndrome (ARDS). Tissue metabolism is severely altered during the response to peritonitis. Tissue hypoxia leads to anaerobic glycolysis leading to metabolic acidosis. The severe loss in the lean body mass that can occur from protein catabolism occurs rapidly and is onlypartially ameliorated by the use of nutritional support.

Clinical features of perforative peritonitis :

The signs and symptoms produced by the perforation vary according to the time that has elapsed since the rupture occurred. There are three stages in the pathological process that can be recognized. The symptoms of each stage can be enumerated as: early stages present as severe and generalized abdominal pain, Hypothermia, Pulse low and weak, Shallow respiration, Retching or vomiting (slight), Pain on top of one or both shoulders.

In their late stages, it is difficult to distinguish intestinal obstruction from perforation, for peritonitis is often a complication of late intestinal obstruction and the board like rigidity accompanying a perforated ulcer tends to diminish somewhat as thedistention increases. In such cases the history and possibly the character of the vomit may serve to differentiate these conditions.

The main points in diagnosis are features of hemorrhagic shock such as the blanching of the lips, tongue, nails and the absence of true abdominal rigidity, though the abdomen is tender especially in the lower part. Rupture of an ulcer with formation of localized subphrenic abscess: Due to previous adhesions, slow leakage of the escaping gastric contents does not flood the peritoneal cavity and the symptoms are modified. The pain may be very great but the initial collapse is not so prostrating, and the abdominal signs will soon be localized to the upper segment of the abdomen and lead to the development of a subphrenic abscess containing gas. If such an abscess develops anteriorly, the local signs of intraperitoneal suppuration are very evident, butwhen it is high up under the diaphragm, the signs and symptoms take longer to develop. Temperature, rigors, leukocytosis and dullness at the base of the lung consequent on pleural effusion or basal congestion will diagnose a collection of pus under the diaphragm.

Treatment:

Once the clinical diagnosis of peritonitis is made, rapid institution of both physiologic support and aggressive anti-infective therapy are imperative. Primary objectives in the treatment of peritonitis are : Resuscitation -> Initiation of antibiotic therapy -> Elimination of the source of bacterial contamination -> Reduction of the bacterial inoculums -> Continued metabolic support.

Antibiotic Therapy:

- Antibiotic therapy should be initiated as soon as a clinical diagnosis of peritonitis is

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obtained. The initial selection of antibiotic is empirical.

- The choice of antibiotics is made with the following considerations–
- The demonstrated activity of the drug against bacteria that are presumed to be present based upon the level of gastrointestinal perforation.
- The bactericidal activity of the antibiotic in the infected tissue.
- Presumptive therapy should include coverage for both aerobic gram-negative rods and anaerobic organisms. Agents that possess activity against aerobic gram-negative bacilli include aminoglycoside, second and third generationcephalosporins and either ampicillin or ticarcillin combined with a beta lactam inhibitor (i.e. sulbactam or clavulanic acid).
- Traditionally a 10 days therapy has been recommended, although newer studies suggest that a five-day therapy may be sufficient.

Conservative Management:

Most patients with peptic ulcer perforation require operative therapy on rare occasions, conservative management of perforation can be beneficial particularly in those patients who have concomitant medical illness, perforation of more than 24 hrs, systolic pressure less than 100 mm Hg at the time of admission. These risk factors have definitive bearing on mortality rate. If one risk factor is present mortality isabout 10%, if two factors are present mortality is about 40%, if three factors are present mortality is about 87%. These patients require close monitoring in intensive care unit as they may deteriorate and need operative therapy. If abdominal findings donot improve in 12 hours then operation is indicated.

Contraindications for non-operative treatment

(i) Age > 70 years, (ii) Steroid use, (iii) Gastric perforation

Simple closure Vs Definitive operation:

Simple closure was first suggested for patients with gastric ulcer perforation in 1894 and later was popularized by Roscoe Graham in perforated duodenal ulcer in 1937. Long-term follow up of these patients with simple closure has significantly influenced operative management in the past 10-15 years. Simple closure will lead to satisfactory result in 1/3rd of patients. The remaining 2/3rd of patients will need acid suppression therapy or definitive operation for complications.74 According to Boey and Wong, complications occurred in 52% of these patients (28% had bleeding, 15% had pyloric obstruction, 9% had reperforation). In this group of patients, 40% requiredreoperation. Ralph I George followed up 75 patients of simple closure for 5-10 yrs, 14of these patients were on ulcerogenic drugs; 7% of them had recurrence while 6% patients who did not take ulcerogenic drugs had recurrence rate of 77%, proving that their ulcer diathesis was virulent enough to need definitive surgery. Boey and associates compared simple closure and closure with vagotomy in 78 patients with acute perforation, recurrence rate was 34% at 36 months after simple closure, reoperation was required in 43% of this group.[76] The higher reoperation rate in this group may be due to ethnic and geographic variation.

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Truncal Vagotomy with Hemigastrectomy:
The principal disadvantages of truncal vagotomy with hemigastrectomy arethe only modest increase in operative time over truncal vagotomy with pyloroplasty, but there is an 8-10% decrease in recurrent ulceration compared with truncal vagotomy with pyloroplasty. This is preferred in cases of perforation in pre-pyloric region. The operative mortality rate for resection is extremely low in properly selectedpatients.

Post-operative follow up and complications:
Perforation may be the end stage in some cases of acute ulcer perforation as in perforation caused by NSAID or ulcerogenic drugs. The patients to be put on omeprazole for eight weeks. H pylori therapy may be added to reduce the recurrence rate. In acute perforation recurrence rate was 43% and in chronic ulcer perforation was 66 to 87%. 52% may develop complications like bleeding, pyloric obstruction and reperforation. The patients with simple closure will need lifelong acid suppression agents and eradication of H pylori. NSAID, cigarette smoking and alcohol aggravate the disease.

Perforated stomal ulcer:
Stomal ulcers more commonly penetrate surrounding structures and occasionally perforate into the transverse colon, resulting in a gastrojejuno-colic fistula. Perforated stomal ulcers may occur many years after a simple gastroenterostomy. The most effective operation for patients with perforated marginal ulcers is to resect or re-resect the stomach including the ulcer and perform a vagotomy if not done earlier. Revagotomy should be done and attention paid to find out the posterior vagus nerve, which is most likely missed. Patients with gastrojejuno-colic fistula are treated by gastric resection, vagotomy, and partial transverse colectomy.

Typhoid Enteritis:
Treatment of typhoid fever and uncomplicated typhoid enteritis is accomplished by antibiotic administration. Complications requiring potential surgical intervention include hemorrhage and perforation. Intestinal perforation through an ulcerated Peyer's patch occurs in approximately 2% of cases. Typically, it is a single perforation in the terminal ileum. Simple closure of the perforation is the treatment of choice. With multiple perforations, resection with primary anastomosis or Exteriorization of the intestinal loops may be required. According to Ameh E.A. segmental resection seems to be best treatment for typhoid perforation.

Perforation in Diverticular disease of small bowel: Small bowel diverticular disease is an uncommon clinical entity. Both acquired and congenital diverticula are frequently asymptomatic and become symptomatic when complicated by infection, perforation, obstruction or hemorrhage.

Duodenal Diverticula: Duodenal diverticula may be acquired or congenital. Perforation may be secondary to diverticulitis or iatrogenic following endoscopic retrograde cholangiopancreatography.

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It commonly occurs in the retroperitoneum over the right kidney and posterior to the head of the pancreas and duodenum. When a perforation is suspected, computed tomographic scan of the abdomen with oral and intravenous contrast is very accurate in confirming the diagnosis and in defining the extent of inflammatory reaction. Prophylactic resection of an asymptomatic diverticulum is not recommended. In the absence of significant retroperitoneal contamination, primary excision of diverticulum with two-layer closure is done. In the case of large duodenal defect, serosal patch technique or a Roux-en-Y duodenojejunostomy is preferred.

In the presence of a perforation with significant edema and contamination, a duodenal diverticulisation (e.g. gastrojejunostomy, closure of the pylorus, closure of the perforation and jejunostomy feeding tube) with drainage of the surrounding area.

Material and Methods

Study Design: Prospective Observational Study

Study Duration: 18 months

Study Site: Department of General Surgery, KIMSDU, Karad

Study Population: Patients presenting in emergency room of Krishna Institute of Medical Sciences and Research Centre, Karad, between any age group, with symptoms of acute pain in abdomen with gas under diaphragm seen on either chest radiograph or x-ray erect abdomen.

Inclusion Criteria:

1. Patients of either sex all age groups willing to participate in the study with valid consent.
2. Patients presenting with acute abdomen with pneumoperitoneum on X-rays and/or CT scan, USG.
3. Patient presenting with acute abdomen and perforation diagnosed intraoperative.

Exclusion Criteria

1. Iatrogenic perforations
2. Pregnancy and lactation.
3. Patients with perforative peritonitis not willing to participate in the study.

Sample Size Estimation

According to articles the prevalence rate of perforated peritonitis patients was 5% to 10%. Sample size formula used was:

$n= Z^2Pq/L^2$; n≈ 124; n= 130; L= allowable error (3)P= prevalence (5); Q= 100-P (95)

By given formula and reference article, I will study a total of 130 cases.

Study Tools: Mannheim Peritonitis Index

Results

Age distribution: In the present study we assessed the Age distribution among the study subjects.We observed that majority of the study subjects belonged to the age group of 46 to 55 years (31.54%), followed by 36 to 45 years (24.62%), more than 66 years among 29.69% study subjects.

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Table: Age distribution

Age distribution	Number of subjects	Percentage
Less than 25 years	6	4.62
26 to 35 years	15	11.54
36 to 45 years	32	24.62
46 to 55 years	41	31.54
More than 56 years	36	27.69
Total	130	100.00

Age distribution	Number of subjects	Percentage
Less than 50	58	44.62
More than 50	72	55.38
Total	130	100.00

Clinical presentation

In the present study we assessed the Clinical presentation among the study subjects. We observed that Fever was noted among 36.92% study subjects, Vomiting was noted among 9.23% study subjects, Pain was noted among 100.00% study subjects, Distension was noted among 60.77% study subjects, Guarding and Rigidity was noted among 67.69% study subjects, Bowel Sound was noted among 11.54% study subjects, Free fluid was noted among 63.08% study subjects.

Diagnosis

In the present study we assessed the Diagnosis among the study subjects. We observed that Duodenal ulcer perforation was diagnosed among 26.92% study subjects, Gastric ulcer perforation was diagnosed among 34.62% study subjects, Appendicular perforation was diagnosed among 23.08% study subjects, Ileal perforation was diagnosed among 8.46% study subjects, Large intestinal perforation was diagnosed among 6.92% study subjects.

Table: Diagnosis

Diagnosis	Number of subjects	Percentage
Duodenal ulcer perforation	35	26.92
Gastric ulcer perforation	45	34.62
Appendicular perforation	30	23.08
Ileal perforation	11	8.46
Large intestinal perforation	9	6.92
Total	130	100.00

Discussion

Perforations of the proximal gastrointestinal tract were six times more prevalent than perforations of the distal gastrointestinal tract, according to previous research from India. The

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proximal gastrointestinal tract perforations are the most prevalent in my research, which is consistent to previous studies in India, although distal GI tract perforations prevail in the Western world.

Age Distribution

In the present study we assessed the Gender wise distribution among the study subjects. We observed that majority of the subjects were males (73.85%), and 26.15% were females. The male: female ratio in the current study was 2.82:1.

Clinical presentation

In the present study we assessed the Clinical presentation among the study subjects. We observed that Fever was noted among 36.92% study subjects, Vomiting was noted among 9.23% study subjects, Pain was noted among 100.00% study subjects, Distension was noted among 60.77% study subjects, Guarding and Rigidity was noted among 67.69% study subjects, Bowel Sound was noted among 11.54% study subjects, Free fluid was noted among 63.08% study subjects.

Site of perforation

In the present study we assessed the Site of perforation among the study subjects. We observed that stomach was the commonest site of perforation (34.62%), followed by duodenum (26.92%), appendix (23.08%). Ileum (8.46%) and Large intestine (6.92%) was also involved rarely.

Diagnosis

In the present study we assessed the Diagnosis among the study subjects. We observed that Duodenal ulcer perforation was diagnosed among 26.92% study subjects, Gastric ulcer perforation was diagnosed among 34.62% study subjects, Appendicular perforation was diagnosed among 23.08% study subjects, Ileal perforation was diagnosed among 8.46% study subjects, Large intestinal perforation was diagnosed among 6.92% study subjects.

Summary and Conclusion

Duodenal ulcer perforation was diagnosed among 26.92% study subjects, Gastric ulcer perforation was diagnosed among 34.62% study subjects, Appendicular perforation was diagnosed among 23.08% study subjects, Ileal perforation was diagnosed among 8.46% study subjects, Large intestinal perforation was diagnosed among 6.92% study subjects. Organ failure was reported among 33.08% subjects, Malignancy was present among 14.62% study subjects. Non-colonic origin of sepsis (86.15%), followed by colonic origin among 13.85% subjects.

Majority of the study subjects had pre-operative duration more than 24 hours (78.46%), while 21.54% had it less than 24 hours. Localized peritonitis was reported among 26.92% study subjects, whereas majority had diffuse type of peritonitis (73.08%). In our study, Clear exudate was noted among 9.23% study subjects, Purulent exudate was noted among 48.46% study subjects, Fecal exudate was noted among 42.31% study subjects. 11.54% mortality was observed

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among the present study subjects, while 88.46% study subjects were discharged, while Surgical site infection was observed among 23.85% study subjects, Stomas were observed among 6.9% study subjects, Re- exploration was observed among 6.15% study subjects, Wound dehiscence was observed among 3.08% study subjects.

Cases of peritonitis carry a high mortality which can be reduced by early diagnosis, risk stratification, appropriate treatment based on risk score. Delayed presentation which has important effect on both mortality and morbidity is beyond our control. Only adequate Health education, proper referral mechanism can help in reducing this. Peritonitis and its sequalae management involves lots of skill, expensive modalities of monitoring and treatment which has to be utilized judiciously based on risk stratification. The male: female ration in the present study was 2.82:1 (male preponderence). The majority of perforation peritonitis cases in the study comprised of gastric ulcer perforations followed by duodenal, appendicular and traumatic perforations. Faecal exudates were more ordinarily associated with colonic origin of sepsis, and it was associated with worse outcomes.

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