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Original research article

A study on clinical profile of patients with peritonitis due to peptic ulcer perforation

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Abstract

Peptic ulcers are described as muscularis mucosae-through erosions in the gastric or duodenal mucosa. Increases in the detection and eradication of H. pylori infection, the principal cause of PUD, are probably contributing to the declining incidence and prevalence of PUD. After obtaining approval and clearance from the institutional ethics committee, the patients fulfilling the inclusion criteria will be enrolled for the study after obtaining informed consent. Current study showed that, out of 64 peptic ulcer disease patients, 15(23.4%) of them were admitted to the hospital with ongoing shock with a Blood Pressure of less than 100 mmHg & Heart Rate of more than 100bpm). While remaining 49(76.6%) of patients had no ongoing shock on admission with stable blood pressure and heart rate. This study showed that, out of 64 peptic ulcer disease patients, 52(81.3%) got admitted to hospital after 24 hours of peptic ulcer perforation while 12(18.8%) got admitted to hospital within 24 hours of the peptic ulcer perforation.

Keywords: Peptic ulcers, peptic ulcer perforation, peritonitis

Introduction

Inflammation of the peritoneum and peritoneal cavity is known as peritonitis, and it is typically brought on by a localised or systemic infection. In the absence of GI perforation, primary peritonitis is caused by bacterial, chlamydial, fungal, or mycobacterial infection. Secondary peritonitis develops as a result of GI rupture [1].

Peptic ulcers are described as muscularis mucosae-through erosions in the gastric or duodenal mucosa. Increases in the detection and eradication of *H. pylori* infection, the principal cause of PUD, are probably contributing to the declining incidence and prevalence of PUD [2].

According to the stage of the rupture, the perforated ulcer causes flooding of the peritoneal cavity with stomach and duodenal contents, leading to chemical peritonitis. There are three stages to the clinical course, each with a different duration [3, 4].

Primary stage or the stage of peritonism: The patient experiences sudden, excruciating pain in the right hypochondrium or epigastrium that spreads quickly. The peritoneum is severely irritated by the contents of the stomach and duodenum, which causes the symptoms. They cause neurogenic shock to occur. Vomiting and nausea are rare in the early stages. Diaphragmatic irritation leads to abdominal pain and pain that radiates to both shoulders. Patients' hands and legs are tensely held to the side while they lie almost rigidly. The pulse rate is normal or increased to around 90. Shallow breathing occurs at a higher respiratory rate. Examining the abdomen reveals no breathing-related movements, a large rectus muscle, and inflexible, board-like muscles. Both widespread rigidity and tenderness are present. There are no bowel noises on auscultation. 3-6 hours are spent in this stage. Acute appendicitis-like symptoms, such as right-side-only discomfort and rigidity, can occasionally be caused by fluid seeping from the perforation and trickling down the right paracolic gutter.

Secondary stage or the stage of peritoneal reaction: Depending on the size, location and quantity of peritoneal soiling, the primary stage to secondary stage transition takes 3-6 hours. The spontaneous closure of the perforation may take place at this stage. The patient may progress to the stage of septic peritonitis if there is a severe leaking of stomach content. Rarely does this stage last longer than six hours. In this phase, the pain is noticeably reduced. The patient would have a general improvement in their health. This reactionary stage is sometimes referred to as the Stage of Delusion for this reason. The patient may put off getting medical help as their health improves, and it is at this point that most diagnostic errors occur. There will be variable degrees of abdominal rigidity and pain upon inspection. Occasionally absent or infrequent bowel sounds.

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Tertiary stage or the stage of bacterial peritonitis: This is the diffuse peritonitis stage, which starts around 12 hours after perforation and lasts for about 24 hours before progressing to the paralytic ileus stage at the end. An infectious organism quickly multiplies. Purulent fluid develops in the peritoneum. Gas and liquids gently dilate the intestines. With the development of paralytic ileus, intestinal motions gradually become absent. Similar clinical characteristics apply to generalised peritonitis from any source. Severe pain is accompanied by perspiration and nausea. Dehydration and electrolyte imbalance are made more obvious by the fluid leakage into the peritoneal cavity and the swollen, paralysed intestines. The patient reports feeling extremely thirsty, having a high body temperature, having a dry and coated tongue, a thready pulse, and shallow, quick breathing. Distended abdomen with continued guarding One can see the standard Hippocratic Facies. The body is icy and clammy, with an ashen face. Dehydration, circulatory failure and toxaemia gradually set in for the patient. Death typically occurs four to five days following perforation.

Methodology

Inclusion criteria

1. Patients willing to give informed consent.
2. Patients of either sex aged between 18 and 80 years.
3. All patients presenting with features of hollow viscus perforation with per.
4. Operative finding suggestive of perforated peptic ulcer.

Exclusion criteria

1. Patient not willing to give informed consent.
2. Patient less than 18 or more than 80 years of age.
3. Histopathology suggestive of malignant ulcer.

Study Methodology

- After obtaining approval and clearance from the institutional ethics committee, the patients fulfilling the inclusion criteria will be enrolled for the study after obtaining informed consent.
- The patients who undergo surgical treatment for perforated peptic ulcer are allotted points according to the POMPP and PULP scoring system after history taking, physical examination, basic pre-operative investigations and radiological imaging. The patients will be allotted points according to both scoring systems which are then compared. The patients will be classified into high risk or low risk categories and followed up accurately to predict the mortality and morbidity within 30 days post operatively.

Statistical Methods

Data was examined using descriptive statistical methods, and all information is presented as Mean, Median, SD, Interquartile Range, Percentages, Tables, and Graphs as needed.

Results

Table 1: Distribution of the Subjects Based on Age Groups

Age groups	Frequency	Percent
18 to 30 yrs	18	28.1
31 to 40 yrs	12	18.8
41 to 50 yrs	8	12.5
51 to 60 yrs	9	14.1
61 to 70 yrs	11	17.2
> 70 yrs	6	9.4
Total	64	100.0

Out of 64 patients included in current study, 5(7.8%) patients belonged to 20 years age group, 13 (20.3%) patients belonged to the 21 to 30 years age group and 12 (18.8%) patients belonged to the 31 to 40 years age group, 8(12.5%) patients belonged to 41 to 50 years age group, 9(14.1%) patients belonged to 51 to 60 years age group, 11(17.2%) patients belonged to 61 to 70 years age group and 6(9.4%) patients belonged to >71 years age group.

Shock on admission: Current study showed that, out of 64 peptic ulcer disease patients, 15(23.4%) of them were admitted to the hospital with on going shock with a Blood Pressure of less than 100 mmHg & Heart Rate of more than 100bpm). While remaining 49(76.6%) of patients had no on going shock on admission with stable blood pressure and heart rate.

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Table 2: Distribution of the Subjects Based on Shock on Admission

Shock on admission	Frequency	Percent
No	15	23.4
Yes	49	76.6
Total	64	100.0

Perforation to Admission >24HRS: This study showed that, out of 64 peptic ulcer disease patients, 52(81.3%) got admitted to hospital after 24 hours of peptic ulcer perforation while 12(18.8%) got admitted to hospital within 24 hours of the peptic ulcer perforation.

Table 3: Distribution of the Subjects Based on Perforation to Admission > 24HRS

Time from perforation to admission > 24 HRS	Frequency	Percent
< 24 HRS	12	18.8
>24 HRS	52	81.3
Total	64	100.0

Serum creatinine: In our study, 48(75%) of peptic ulcer disease patients had serum creatinine levels of ≤1.47 mg/dL while 16(25%) peptic ulcer disease patients had serum creatinine levels of >1.47 mg/dL.

Table 4: Distribution of the Subjects based on Serum Creatinine >1.47 mg/dL

Serum creatinine	Frequency	Percent
< 1.47 mg/dL	48	75.0
> 1.47 mg/dL	16	25.0
Total	64	100.0

Blood Urea Nitrogen: In this study, out of 64 patients, 4 patients had a high Blood Urea Nitrogen level of >45 mg/dl and 60 patients had normal Blood Urea Nitrogen levels ≤ 45 mg/dl accounting for 93.8% of the study population.

Table 5: Distribution of the Subjects Based on Bun > 45 MG/DL

BUN	Frequency	Percent
< 45 mg/dl	60	93.8
> 45 mg/dl	4	6.3
Total	64	100.0

Serum Albumin: Among 64 peptic ulcer disease patients, 9(14.1%) patients had a serum albumin level of ≤1.5g/dl while 55(85.9%) peptic ulcer disease patients had a serum albumin level of >1.5g/dl.

Table 6: Distribution of the Subjects Based on Albumin < 1.5 G/DL

Albumin	Frequency	Percent
>1.5 g/dl	55	85.9
< 1.5 g/dl	9	14.1
Total	64	100.0

Discussion

Although there is no upper age limit for peptic ulcer perforation, we found that the youngest patient in our sample was 18 and the oldest was 85. The third to fourth decade was shown to have the highest prevalence of peptic ulcer perforation (20.3%), followed by the sixth decades (17.2% each) in the study population [5].

In the research by Kocer *et al*. (2007), the mean age of the patients with peptic ulcer perforation was 43 years, whereas in the study by Dakubo *et al*. (2009), it was 41 years. However, a research by Mishra *et al*. (2001) in India found a mean age of 39 years, while a study by Anand *et al*. (2018) found a mean age of 43.2 years. Older age group patients are more frequently impacted, according to recent research like those by Moller *et al*. (2012) and Thorsen *et al*. (2009) (median ages of 71 and 67 years, respectively). This work complements those by Kocer *et al*. (2007) and Anand *et al*., (2018) [6].

A bacterial infection of the ascitic fluid without an intra-abdominal cause of infection, such as a visceral perforation, abscess, acute pancreatitis, or cholecystitis, is known as spontaneous bacterial peritonitis (SBP). SBP can develop in patients with nephrotic syndrome and, less frequently, congestive heart failure, however it is typically linked to cirrhosis [7].

It is believed that bacterial translocation from the GI tract is a crucial stage in the pathogenesis of SBP. Impaired local and systemic immune function limits the efficient clearance of translocated bacteria from the mesenteric lymphatics and circulation, and impaired GI motility in cirrhotics is thought to affect

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normal gut microbiobe. A lack of protein in ascitic fluid prohibits macrophages and neutrophils from effectively opsonizing germs and clearing them from the body. The clinical picture resembles secondary bacterial peritonitis because of the sudden onset of fever, distension in the abdomen, and rebound soreness. But just a quarter of patients experience any peritoneal symptoms at all. Most patients with severe cirrhosis or nephrosis will exhibit clinical and biochemical symptoms [2].

When a patient has low-protein ascites and exhibits abdominal pain, a fever, or leukocytosis, the diagnosis of SBP is initially made by demonstrating more than 250 neutrophils/mm3 of ascitic fluid.

Characteristic findings include leukocytosis, hypoalbuminemia and an extended prothrombin time. The diagnosis is supported by a blood-ascitic fluid albumin gradient more than 1.1 g/dL, a high serum lactic acid level (> 33 mg/dL), or a low pH of the ascitic fluid (7.31). A single enteric organism, most frequently E coli, 32 Klebsiella, or Streptococci, is typically detected when ascitic fluid is cultured and quickly added to blood culture media at the patient's bedside. However, Listeria monocytogenes has been documented in immunocompromised hosts [9].

When a patient has an ascitic fluid infection, broad-spectrum antibiotics, such as a third-generation cephalosporin, are administered right away.

Amoxicillin/clavulanic acid and quinolones like ciprofloxacin are substitutes.

SBP complications such septic shock, gastrointestinal haemorrhage and hypoalbuminemia should be treated appropriately. SBP carries a minimal risk of immediate mortality, especially if it is identified and treated quickly.

Bacterial contamination that originates in the viscera or from external sources causes secondary peritonitis (e.g., penetrating injury). It most frequently happens once a hollow viscus is disrupted.

Depending on the virulence of the pathogens, the bacterial load, the length of bacterial proliferation, and the synergistic interaction of the bacteria, systemic sepsis caused by peritonitis occurs to varied degrees.

Peritonitis is virtually always polymicrobial, with the exception of spontaneous bacterial peritonitis; cultures typically contain more than one aerobic and more than two anaerobic species. The bacterial flora of the affected organ is reflected in the microbiome image.

Stomach perforations are typically sterile or connected to relatively few gram-positive organisms as long as gastric acid output and gastric emptying are normal. Normally, stomach secretions contain about 1000 microorganisms per millilitre. Alpha hemolytic streptococci, lactobacilli, yeasts, and a few oral bacteria are the only obligate anaerobes. There are 100 to 10000 bacteria per ml in the duodenum and jejunum, mostly streptococci, lactobacilli, transient oral flora, and infrequently Enterobacter species [10].

About 30% of times, perforations or ischemic damage to the distal small bowel (such as strangulated hernias) result in an infection with aerobic bacteria, and 10% of times, an infection with anaerobic organisms. 106 to 107 bacteria per millilitre are present. Streptococci, lactobacilli, bacteroids and enterobacter are equally prevalent.

Fecal leakage is particularly hazardous when the bacterial burden is 1012 or more per gramme. Bacteria make up 60% of dry faecal matter. The number of bacteria per milligramme of dry stool is 3.81014.

The flora is composed of streptococci, bacillus species, enterococci, E. coli, bacteroids, clostridia, and anaerobic cocci.

The way adhesions develop around the injured organ determines the clinical course. Inflamed peritoneum loses its glossy appearance and changes to a reddish, velvety appearance. Upon the appearance of fibrin flakes, intestinal loops cling to one another and the parietal wall.

Serous inflammatory exudates that are rich in leukocytes and plasma proteins pour forth and quickly turn turbid; if localization takes place, the turbid fluid turns into true pus. The larger omentum frequently acts as a strong barrier to the spread of infection by encircling and adhering to inflamed tissues.

Conclusion

- Peptic ulcer perforation was found to be most common in the age group of 20 to 40 years with a mean age of 45.5.
- Peptic ulcer perforation was more common in males than females in the ratio 8.3:1. It was observed that the post-operative mortality was 6.2%.
- Shock at the time of admission was also found to be an important risk factor and all patients who died had preoperative shock.

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