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Clinical assessment of prevalence of peptic ulcer perforation and its outcome in patients from Bihar region

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Abstract
When acute or chronic peptic ulcer perforates into the peritoneal cavity, three components require treatment viz., the ulcer, the perforation and the resultant peritonitis. The perforation and resultant peritonitis are immediate threats to the life; the ulcer in itself is not. The therapeutic priorities thus are treatment of peritonitis and securing the closure of perforation, which may be achieved with surgical procedure. In spite of better understanding of disease; effective resuscitation and prompt surgery under modern anaesthesia techniques, there is high morbidity and mortality. Hence, attempt has been made to analyze the various factors, which are affecting the morbidity/mortality of patients with peptic ulcer perforations.
The present study was planned in Department of General Surgery, Nalanda Medical College, Patna. Total 25 cases diagnosed with the duodenal ulcer proliferation were enrolled in the present study. When acute or chronic duodenal ulcer perforates into the peritoneal cavity, three components require treatment viz., the ulcer, the perforation and the resultant peritonitis. The perforation and resultant peritonitis are immediate threats to the life; the ulcer in itself is not. The therapeutic priorities thus are treatment of peritonitis and securing the closure of perforation, which may be achieved with surgical procedure. In spite of better understanding of disease, effective resuscitation and prompt surgery under modern anaesthesia techniques, there is high morbidity and mortality. Hence, attempt has been made to analyze the various factors, which are affecting the morbidity/mortality of patients with peptic ulcer perforations.
Peptic ulcer perforation is one of the most common acute abdominal emergencies. Early presentation holds a good prognosis. Unfortunately; in developing countries like India, patients usually present late to the hospital with full blown peritonitis, septic shock and multi organ failure. The outcome of the patient depends on the following factors:- age of the patient, associated co-morbidities, time interval between onset of acute abdominal pain and surgery, condition of the patient at the time of surgery like dehydration, contamination and septicaemia. The risk of mortality increases with increase in age, severe contamination of the abdomen observed during surgery leading to post-operative sepsis and increase in time interval between onset of acute pain and surgery. Prompt diagnosis, aggressive resuscitation, early surgery and the quickly performed simple Cellan Jones omental patch repair results in high survival in these moribund patients.

Keywords: outcome, peptic ulcer, perforation, time interval between onset of acute abdominal pain and surgery, etc

Introduction
Peptic ulcer disease can involve the stomach or duodenum. Gastric and duodenal ulcers usually cannot be differentiated based on history alone, although some findings may be suggestive. Epigastric pain is the most common symptom of both gastric and duodenal ulcers, characterized by a gnawing or burning sensation and that occurs after meals—classically, shortly after meals with gastric ulcers and 2-3 hours afterward with duodenal ulcers.
In uncomplicated peptic ulcer disease, the clinical findings are few and nonspecific. “Alarm features" that warrant prompt gastroenterology referral [1] include bleeding, anemia, early satiety, unexplained weight loss, progressive dysphagia or odynophagia, recurrent vomiting, and family history of gastrointestinal (GI) cancer. Patients with perforated peptic ulcer disease usually present with a sudden onset of severe, sharp abdominal pain.
In most patients with uncomplicated peptic ulcer disease, routine laboratory tests usually are not helpful; instead, documentation of peptic ulcer disease depends on radiographic and endoscopic confirmation. Testing for H pylori infection is essential in all patients with peptic ulcers. Rapid urease tests are considered the endoscopic diagnostic test of choice. Of the noninvasive tests, fecal antigen testing is more accurate than antibody testing and is less expensive than urea breath tests but either is reasonable. A fasting serum gastrin level should be obtained in certain cases to screen for Zollinger-Ellison syndrome.
Upper GI endoscopy is the preferred diagnostic test in the evaluation of patients with suspected peptic ulcer disease. Endoscopy provides an opportunity to visualize the ulcer, to determine the presence and degree of active bleeding, and to attempt hemostasis by direct measures, if required. Perform endoscopy early in patients older than 45-50 years and in patients with associated so-called alarm features. Most patients with peptic ulcer disease are treated successfully with cure of H pylori infection and/or avoidance of nonsteroidal anti-inflammatory drugs (NSAIDs), along with the appropriate use of antisecretory therapy. In the United States, the recommended primary therapy for H pylori infection is proton pump inhibitor (PPI)–based triple therapy [1]. These regimens result in a cure of infection and ulcer

healing in approximately 85-90% of cases [2]. Ulcers can recur in the absence of successful H pylori eradication. In patients with NSAID-associated peptic ulcers, discontinuation of NSAIDs is paramount, if it is clinically feasible. For patients who must continue with their NSAIDs, proton pump inhibitor (PPI) maintenance is recommended to prevent recurrences even after eradication of H pylori. [3, 4] Prophylactic regimens that have been shown to dramatically reduce the risk of NSAID-induced gastric and duodenal ulcers include the use of a prostaglandin analogue or a PPI. Maintenance therapy with antisecretory medications (eg, H2 blockers, PPIs) for 1 year is indicated in high-risk patients. The indications for urgent surgery include failure to achieve hemostasis endoscopically, recurrent bleeding despite endoscopic attempts at achieving hemostasis (many advocate surgery after two failed endoscopic attempts), and perforation.

Because many surgical procedures for peptic ulcer disease entail some type of vagotomy, a discussion concerning the vagal innervation of the abdominal viscera is appropriate (see image below). The left (anterior) and the right (posterior) branches of the vagus nerve descend along either side of the distal esophagus. As they enter the lower thoracic cavity, they can communicate with each other through several cross-branches that comprise the esophageal plexus. However, below this plexus, the 2 vagal trunks again become separate and distinct before the anterior trunk branches to form the hepatic, pyloric, and anterior gastric (also termed the anterior nerve of Latarjet) branches. The posterior trunk branches to form the posterior gastric branch (also termed the posterior nerve of Latarjet) and the celiac branch.

The parietal cell mass of the stomach is segmentally innervated by the terminal branches from each of the anterior and posterior gastric branches. These terminal branches are divided during highly selective vagotomy. The gallbladder is innervated from efferent branches of the hepatic division of the anterior trunk. Consequently, transection of the anterior vagus trunk (performed during truncal vagotomy) can result in a dilated gallbladder with inhibited contractility and subsequent cholelithiasis. The celiac branch of the posterior vagus innervates the entire midgut (with the exception of the gallbladder). Thus, division of the posterior trunk during truncalvagotomy may contribute to postoperative ileus.

Peptic ulcers are defects in the gastric or duodenal mucosa that extend through the muscularis mucosa. The epithelial cells of the stomach and duodenum secrete mucus in response to irritation of the epithelial lining and as a result of cholinergic stimulation. The superficial portion of the gastric and duodenal mucosa exists in the form of a gel layer, which is impermeable to acid and pepsin. Other gastric and duodenal cells secrete bicarbonate, which aids in buffering acid that lies near the mucosa. Prostaglandins of the E type (PGE) have an important protective role, because PGE increases the production of both bicarbonate and the mucous layer. In the event of acid and pepsin entering the epithelial cells, additional mechanisms are in place to reduce injury. Within the epithelial cells, ion pumps in the basolateral cell membrane help to regulate intracellular pH by removing excess hydrogen ions. Through the process of restitution, healthy cells migrate to the site of injury. Mucosal blood flow removes acid that diffuses through the injured mucosa and provides bicarbonate to the surface epithelial cells.

Under normal conditions, a physiologic balance exists between gastric acid secretion and gastroduodenal mucosal defense. Mucosal injury and, thus, peptic ulcer occur when the balance between the aggressive factors and the defensive mechanisms is disrupted. Aggressive factors, such as nonsteroidal anti-inflammatory drugs (NSAIDs), H pylori infection, alcohol, bile salts, acid, and pepsin, can alter the mucosal defense by allowing back diffusion of hydrogen ions and subsequent epithelial cell injury. The defensive mechanisms include tight intercellular junctions, mucus, bicarbonate, mucosal blood flow, cellular restitution, and epithelial renewal.

The gram-negative spirochete H pylori was first linked to gastritis in 1983. Since then, further study of H pylori has revealed that it is a major part of the triad, which includes acid and pepsin that contributes to primary peptic ulcer disease. The unique microbiologic characteristics of this organism, such as urease production, allows it to alkalinize its microenvironment and survive for years in the hostile acidic environment of the stomach, where it causes mucosal inflammation and, in some individuals, worsens the severity of peptic ulcer disease.

When H pylori colonizes the gastric mucosa, inflammation usually results. The causal association between H pylori gastritis and duodenal ulceration is now well established in the adult and pediatric literature. In patients infected with H pylori, high levels of gastrin and pepsinogen and reduced levels of somatostatin have been measured. In infected patients, exposure of the duodenum to acid is increased. Virulence factors produced by H pylori, including urease, catalase, vacuolatingcytotoxin, and lipopolysaccharide, are well described.

Most patients with duodenal ulcers have impaired duodenal bicarbonate secretion, which has also proven to be caused by H pylori because its eradication reverses the defect. [5] The combination of increased gastric acid secretion and reduced duodenal bicarbonate secretion lowers the pH in the duodenum, which promotes the development of gastric metaplasia (ie, the presence of gastric epithelium in the first portion of the duodenum). H pylori infection in areas of gastric metaplasia induces duodenitis and enhances the susceptibility to acid injury, thereby predisposing to duodenal ulcers. Duodenal colonization by H pylori was found to be a highly significant predictor of subsequent development of duodenal ulcers in one study that followed 181 patients with endoscopy-negative, nonulcer dyspepsia [6].

Patients with perforated peptic ulcer disease (PUD) usually present with a sudden onset of severe, sharp abdominal pain. Most patients describe generalized pain; few present with severe epigastric pain. As even slight movement can tremendously worsen their pain, these patients assume a fetal position. Abdominal examination findings are usually consistent with generalized tenderness, rebound tenderness, guarding, and rigidity. However, the degree of peritoneal findings is strongly influenced by a number of factors, including the size of perforation, amount of bacterial and gastric contents contaminating the abdominal cavity, time between perforation and presentation, and spontaneous sealing of perforation.

These patients may also demonstrate signs and symptoms of septic shock, such as tachycardia, hypotension, and anuria. Not surprisingly, these indicators of shock may be absent in

elderly or immunocompromised patients or in those with diabetes. Patients should be asked if retching and vomiting occurred before the onset of pain. Obtaining the medical history, especially for peptic ulcer disease, H pylori infection, ingestion of NSAIDs, or smoking, is essential in making the correct diagnosis.

When acute or chronic peptic ulcer perforates into the peritoneal cavity, three components require treatment viz., the ulcer, the perforation and the resultant peritonitis. The perforation and resultant peritonitis are immediate threats to the life; the ulcer in itself is not. The therapeutic priorities thus are treatment of peritonitis and securing the closure of perforation, which may be achieved with surgical procedure. In spite of better understanding of disease, effective resuscitation and prompt surgery under modern anaesthesia techniques, there is high morbidity and mortality. Hence, attempt has been made to analyze the various factors, which are affecting the morbidity/mortality of patients with peptic ulcer perforations.

Methodology

The present study was planned in Department of General Surgery, Nalanda Medical College, Patna.Total 25 cases diagnosed with the duodenal ulcer proliferation were enrolled in the present study. When acute or chronic duodenal ulcer perforates into the peritoneal cavity, three components require treatment viz., the ulcer, the perforation and the resultant peritonitis. The perforation and resultant peritonitis are immediate threats to the life; the ulcer in itself is not. The therapeutic priorities thus are treatment of peritonitis and securing the closure of perforation, which may be achieved with surgical procedure. In spite of better understanding of disease, effective resuscitation and prompt surgery under modern anaesthesia techniques, there is high morbidity and mortality. Hence, attempt has been made to analyze the various factors, which are affecting the morbidity/mortality of patients with peptic ulcer perforations.

Informed consent was taken with all patients included in the study. The aims and the objectives of the present study were conveyed to them. Approval of the institutional ethical committee was taken prior to conduct of this study.

Following was the inclusion and exclusion criteria for the present study.

Inclusion criteria:• Patients with peptic ulcer perforations of size>2cm. • Patients with age 15 to 80 years.

Exclusion criteria:• Patients with peptic ulcer perforation of size 80 years. • Malignant gastric ulcer perforation either suspicious or proven by edge biopsy.

Results & Discussion

Peptic ulcer perforation is a common surgical emergency condition. The advent of better proton pump inhibitors have led to decline in the rates of elective perforation surgery but the emergency perforation rates remain unchanged [7].

Lau and Leow have indicated that perforated peptic ulcer was clinically recognized by 1799, but the first successful surgical management of gastric ulcer was by Ludwig Heusner in Germany in 1892. In 1894, Henry Percy Dean from London was the first surgeon to report successful repair of a perforated duodenal ulcer. Wangensteen *et al* reported that in a patient with perforation but without evidence of pneumoperitoneum, one can safely assume that perforation has sealed off on its own. They advocated a non-operative approach for such patients. However, they too supported operative treatment in patients with perforated ulcer and evidence of pneumoperitoneum. Berne and Donovan emphasized the use of a water-soluble upper GI study to demonstrate spontaneous sealing of the perforation. They demonstrated that as many as 40% of perforated peptic ulcers had no evidence of leak on upper GI contrast studies. Berne and Donovan concluded that these patients can be observed safely as long as peritonitis does not develop. Mortality rates were 6% and 3% in the operative and non-operative groups, respectively. Donovan *et al* proposed dividing patients based on their Helicobacter pylori infection status and recommended non-operative treatment in all patients except those without H pylori infection and those in whom prior treatment of H pylori infection had failed.

Despite strong arguments favouring non-operative treatment of patients with perforated PUD, delaying the initiation of surgery more than 12 hours after presentation was demonstrated to worsen the outcome. Therefore, when definitely indicated, a laparotomy should be performed as soon as possible.

Table 1: Age & Sex of Cases

Age	No. of Cases
Less than 20 years	1
21 – 30 years	5
31 – 40 years	5
41 – 50 years	6
51 – 60 years	3
Above 60 years	5
Total	25
Sex	
Males	21
Females	4
Total	25

Table 2: History of Peptic Ulcer Diseases

History	No. of Cases
Positive	20
Negative	5
Total	25

Table 3: Outcome of the Perforation

History	No. of Cases
Cured	23
Expired	2
Total	25

Males in the age group 31-40 years were most commonly affected outnumbering females in the ratio 4.1:1 as also reported from another Indian study [8] but in complete contrast from a study in Norway [9] where the male to female ratio was equal and most of the patients were above 50 years of age. Smoking, alcohol and NSAID abuse accounted for most of the cases due to peptic ulcer but probably more than one factor contributed to the disease as also reported in other studies [10]. Tests for H. Pylori not being commonly available their exact contribution in the etiology of peptic ulcer in this study is not known. Abdominal pain, distension, tachycardia, abdominal guarding and rigidity were the commonest symptoms and signs on presentation as has been reported in most other studies [11].

Both alcohol and smoking is collectively contributed a risk, probably this could be a risk factor for higher male preponderance. Alcohol causes mucosal damage by stimulating acid secretion and increasing gastrin level [13]. Smoking inhibits bicarbonate secretion from pancreas which results in increased acidity in the duodenal bulb and also inhibits healing of the duodenal ulcer [12, 14]. It is observed that taking NSAIDs is a risk factor since NSAIDs inhibit the synthesis of prostaglandins which reduce mucosal blood flow, vulnerable to peptic ulceration and its perforation. The incidence of taking NSAIDs in this study is little higher than the other developing countries, as coated by Phillipo L Chalya *et al* from Tanzania in their study. However the incidence of taking NSAIDs is more in the developed countries [15].

Perforation is the most common complication of peptic ulcer disease. We did not get a single case of bleeding ulcer case in our study group, however we had one case of pyloric stenosis. Perforation is more common in 4th to7th decades of life. Pain epigastric region, vomiting and distension of the abdomen are cardinal symptoms. Tenderness, rigidity and guarding along with absence of bowel sounds are the cardinal signs of perforation. Patients who presents late may have fever, cardiovascular instability with septicaemia and may have organ failure like renal and respiratory failure. Plain X-ray erect abdomen is most important diagnostic aid which shows pneumoperitoneum i.e. presence of gas under the diaphragm. Contrast CT may be useful when sealed up perforation is encountered. Simple closure perforation is the treatment of the choice with omental patch. Open method is preferred than the laparoscopic procedure, since operating time is shorter in open method and most of the patients presents with cardiovascular instability. Surgical site infection is most common post-operative complication followed by septicaemia and leak from sutured site of perforation. Leak sometimes demand reopening and closure. Time of hospital admission is crucial factor; late presentation may lead to bacterial peritonitis and septicaemia sometime death. Elevated leukocyte count with purulent peritoneal fluid is an indirect evidence of bacterial peritonitis, herald increased morbidity and mortality.

Tsugawa K *et al* reviewed that three risk factors: pre-operative shock, delay to surgery over 24 hours and medical illness, was shown by the progressive rise in the mortality rate with the increasing number of risk factors (Hepatogastroenterology). [16]Boey John *et al*. [18] revealed concurrent medical illness, pre-operative shock and delayed operation (>48hours) as significant risk factors that increase mortality in patients with perforated duodenal ulcers (1982) [17].

Conclusion

Peptic ulcer perforation is one of the most common acute abdominal emergencies. Early presentation holds a good prognosis, unfortunately in developing countries like India patients usually present late to the hospital with full blown peritonitis, septic shock and multi organ failure. The outcome of the patient depends on the following factors:- age of the patient, associated co-morbidities, time interval between onset of acute abdominal pain and surgery, condition of the patient at the time of surgery like dehydration, contamination and septicaemia. The risk of mortality increases with increase in age, severe contamination of the abdomen observed during surgery leading to post-operative sepsis and increase in time interval between onset of acute pain and surgery. Prompt diagnosis, aggressive resuscitation, early surgery and the quickly performed simple Cellan Jones omental patch repair results in high survival in these moribund patients.

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