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PEPTIC ULCER DISEASE

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Peptic ulcers are defects/sores in the gastroduodenal mucosa. These ulcers wreak havoc by causing stomach pain and often lead to gastrointestinal (GI) bleeding. Peptic ulcer disease (PUD) refers to ulcerative disorders of the lower esophagus, upper duodenum, and lower portion of the stomach. The lifetime prevalence of PUD is approximately 10%, affecting about 4.5 million people annually in the United States alone (Prasad & Friedman, 2018).

Incidence of PUD increases with age, with most ulcers occurring between 25 and 64 years of age (Akhtar, Shelton, & Dinh, 2019). Most patients with uncomplicated PUD can be treated successfully, if properly evaluated (Anand & O'Katz, 2018). Early detection of those at risk for and/or with PUD through nursing assessment can help prevent related complications.

Pathophysiology

Under normal conditions, physiological balance exists between gastric acid secretion and gastroduodenal defense. Injury to the mucosa causing peptic ulcer(s) occurs with disruption of the stasis between aggressive factors and defensive mechanisms. The term "peptic ulcer" refers to injury to the digestive tract caused by peptic acid (Anand & O'Katz, 2018). Peptic ulcers form when acid erodes the GI tract lining (Crowe, 2018). This injury causes the gastric mucosa to break, creating ulcerations (Anand & O'Katz, 2018).

Historically, a hypersecretory acidic environment mixed with dietary factors and/or stress was suspected to be the most causative factor of PUD. However, the discovery of *Helicobacter pylori* (*H. pylori*) infection coupled with increasingly widespread use of non-steroidal inflammatory drugs (NSAIDs) has developed new PUD causation perceptions. Use of NSAIDs and *H. pylori* infection are the two main risk factors for peptic ulcers, yet

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relatively few people with *H. pylori* infection who are taking NSAIDs develop PUD, suggesting that personal susceptibility to bacterial virulence and drug toxicity may be essential to the initiation of mucosal damage (Lanas & Chan, 2017).

Interplay between bacterial and host factors likely determines outcome(s) of *H. pylori* infection. An increasing incidence of PUD in families can likely be attributed to familial clustering of *H. pylori* infection, which is thought to spread through fecal oral route as well as responsive inherited genetic factors (Lanas & Chan, 2017). Although *H. pylori* infection and long-term NSAID use remain the two most common risk factors for PUD, additional current risk factors include the following:

- Gastrinoma;
- Medications (i.e., bisphosphonates, corticosteroids [high-dose and/or prolonged use], potassium chloride, chemotherapeutic agents);
- Tobacco use;
- Family history of ulcers;
- Alcohol use; and
- Stress (following intense physical injury/illness, i.e., acute sickness, ventilator support, head injury, burns).

Peptic ulcers are most commonly located in the stomach and proximal duodenum, but they can also occur in the esophagus or Meckel diverticulum (Akhtar et al., 2019; Lanas & Chan, 2017).

TABLE 1. Peptic Ulcer Disease Symptoms

Duodenal ulcer • Mid-epigastric pain • Gnawing or burning, nonradiating, reoccurring, episodic pain • Pain is typically relieved by food or antacids
Gastric ulcer • Mid-epigastric pain • Pain is typically aggravated by food and relieved by antacids
Nonspecific dyspeptic symptoms • Indigestion • Epigastric fullness • Nausea and vomiting • Loss of appetite • Heartburn
Alarm symptoms • Symptom onset after 55 years of age • Progressive dysphagia • Persistent of recurrent vomiting • Severe abdominal pain • Weight loss and/or anorexia • Family history of gastric malignancy • Blood in stool, melena, hematemesis, and/or anemia

Note. Nonsteroidal anti-inflammatory drug-induced ulcers are often silent with perforation or bleeding as initial presentation. Adapted from "Peptic Ulcer Disease," by F. Akhtar, P. Shelton, and A. Dinh, 2019, In F. Domino, R. Baldor, J. Golding., & M. Stephens (Eds.), *The 5-Minute Clinical Consult* (27th ed., pp. 748–749), Philadelphia, PA: Wolters Kluwer.

Presentation

Although some patients are asymptomatic, the most common presenting PUD chief complaint is dyspepsia or epigastric pain. The pain is often centered in the epigastrium. Discomfort associated with PUD is typically described as aching, burning, sharp, and/or gnawing pain that occurs 2–5 hours after eating and/or in the middle of the night. Patients with PUD often report that the pain is relieved by antacids or food; however, the symptoms are recurrent, with episodes lasting hours to months. Peptic ulcers may also be associated with food-provoking symptoms (i.e., pain with food ingestion, postprandial belching, fullness, nausea/vomiting, fatty food intolerance). Changes in pain intensity, location, or duration may indicate ulcer penetration/perforation (Akhtar et al., 2019; Glynn, 2017).

Physical Examination

Physical examinations for uncomplicated PUD may be nonspecific, minimal, and/or absent (Akhtar et al., 2019). With advancement of PUD, more prominent physical findings may be appreciated. Physical assessment should explore the following possible signs/symptoms (Table 1):

- Vital signs (hemodynamic stability);
- Conjunctival pallor (anemia);
- Epigastric tenderness (absent in 30% of older patients);
- Guaiac-positive stool (from occult blood loss); and
- Melena (black or maroon stools resulting from GI bleeding).

Examination of the abdominal quadrants should be performed in the following order:

- Inspection;
- Auscultation;
- Percussion; and
- Palpation. (Weber & Kelley, 2018)

Peritoneal signs (rebound abdominal tenderness, guarding, and rigidity) may signify a perforated ulcer (Prasad & Friedman, 2018).

Occasionally, auscultation may reveal a succession splash approximately 4 hours after meals, indicating a duodenal or pyloric channel ulcer—resulting in gastric outlet obstruction (Table 2). If a perforation has occurred, a rigid abdomen and generalized rebound tenderness are likely to be noted. A rectal examination should also be included with melena testing (Glynn, 2017).

TABLE 2. Peptic Ulcer Disease Description

Peptic ulcer types
Duodenal ulcer Most common peptic ulcer Typically located in the proximal duodenum Multiple ulcers and/or ulcers distal to the second portion of the duodenum raise clinical suspicion of gastrinoma (Zollinger–Ellison syndrome)
Gastric ulcer Less common than duodenal ulcer in the absence of NSAID usage Typically located along the lesser curvature of the antrum
Esophageal ulcers Located in the distal esophagus; usually secondary to gastroesophageal reflux disease Seen also with gastrinoma
Ectopic gastric mucosal ulceration Can develop with Meckel diverticulum
Commonly associated conditions
Gastrinoma (Zollinger–Ellison syndrome) Multiple endocrine neoplasia (Type 1) Carcinoid syndrome Chronic illness: COPD, Crohn disease, chronic renal failure, hepatic cirrhosis, cystic fibrosis Hematopoietic disorders (rare) myeloproliferative disease, hyperparathyroidism, systemic mastocytosis, polycythemia rubra vera
Differential diagnosis
Functional dyspepsia Gastritis GERD Biliary colic Cholecystitis Gastroenteritis Crohn disease GI malignancy Cardiac ischemia Intestinal ischemia

Note. COPD = chronic obstructive pulmonary disease; GERD = gastroesophageal reflux disease; GI = gastrointestinal. Adapted from "Peptic Ulcer Disease," by F. Akhtar, P. Shelton, and A. Dinh, 2019, In F. Domino, R. Baldor, J. Golding, and M. Stephens (Eds.), *The 5-Minute Clinical Consult* (27th ed., pp. 748–749), Philadelphia, PA: Wolters Kluwer.

Diagnostic Workup

In most patients with uncomplicated PUD, routine laboratory tests are often unhelpful (Prasad & Friedman, 2018). Although a complete blood cell count can exclude anemia, there is no reliable blood test to confirm PUD. In addition, the following tests may aid in PUD workup:

- Blood chemistries for assessment of electrolyte levels and liver function;
- Fecal occult blood test to rule out GI bleeding;
- Testing for *H. pylori* through stool antigen test, urea breath test, rapid urease test, serology, or histology:
  - Stool antigen is the most accurate test for diagnosing *H. pylori* with 96% sensitivity, 83% specificity, and 91% accuracy compared with serology, which has 50% sensitivity, 54% specificity, and 52% accuracy (Kazemi, Tavakkoli, Habizadeh, & Emami, 2011);
- Fasting serum gastrin (to rule out gastrinoma) if multiple or refractory ulcers are present; and
- Esophagogastroduodenoscopy (or upper endoscopy).

Endoscopy is the gold standard for PUD diagnosis and remains the most accurate PUD diagnostic test. Because *H. pylori* infection is the causative factor of most types of PUD, a test-and-treat strategy with a noninvasive test to exclude infection is recommended to rule out infection in patients younger than 55 years (Moayyedi et al., 2017) who present with no alarm symptoms in geographical areas where gastric carcinoma is uncommon and *H. pylori* prevalence is greater than 20%. Geographic prevalence of *H. pylori* varies worldwide depending on the socioeconomic status and sanitation conditions, with a prevalence of less than 40% in developed countries and more than 80% in developing countries (Kazemi et al., 2011). In older patients, endoscopy is recommended to rule out PUD (Lanas & Chan, 2017). Although GI symptoms are common in children, PUD is relatively rare (Fashner & Gitu, 2015).

Clinical Pearls

In some cases, peptic ulcers may heal without treatment. However, untreated ulcers are more likely to reoccur (Crowe, 2018). Most patients with PUD are successfully treated with cure of *H. pylori* infection and/or avoidance of NSAIDs, as well as appropriate use of antisecretory therapy. Management of *H. pylori* gastritis should include diagnosis, treatment, and eradication confirmation by a noninvasive test such as fecal antigen no sooner than 4 weeks after completion of therapy (Romano & Cuomo, 2004), while keeping in mind that proton pump inhibitors (PPIs) can cause false-negative results on *H. pylori* testing and should be held 2 weeks before testing (Mayo Clinic Staff, 2017).

In patients with NSAID-induced peptic ulcers, discontinuation of NSAIDs is critical if clinically feasible. For those who must continue NSAIDs, PPI therapy should be initiated to prevent future recurrences, even following eradication of *H. pylori* (Anand & O'Katz, 2018). All patients and especially those at risk for PUD should be advised to discontinue smoking (Prasad & Friedman, 2018).

Because a small percentage of gastric ulcers are actually ulcerated gastric carcinomas, all gastric ulcers must be carefully assessed to properly distinguish benign from malignant ulcers. Endoscopy should be repeated to ensure

gastric ulcers (unrelated to NSAID use) are healing, particularly if the ulcer(s) is greater than 2 cm in size (Prasad & Friedman, 2018). Indications for urgent surgery include failure to achieve hemostasis with endoscopy, recurrent bleeding despite endoscopic attempts, and ulcer perforation (Anand & O'Katz, 2018).

Conclusion

Complications of PUD remain life threating. Endoscopic and pharmacological therapy advances have not substantially reduced mortality associated with gastric bleeding, as comorbidities are now a major cause of death in patients with severe PUD (Lanas & Chan, 2017). Prevention of PUD is the best course of action. Proper nursing evaluation of patients at risk for and/or with PUD and identification/treatment of subsequent sequela remain critical.

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