Complications of Peptic ulcer disease

Zainab Aws Mohammed

Abstract

The word "peptic ulcer" describes an acid-peptic injury to the digestive tract that reaches the submucosa through a mucosal breach. Peptic ulcers can occur in the oesophagus or Meckel's diverticulum, but they are more commonly found in the stomach or proximal duodenum. The two main risk factors for duodenal and stomach ulcers are H pylori and NSAID or aspirin use. Nonetheless, the development of peptic ulcer disease is rare in those with H pylori infection, NSAID use, or aspirin use. This suggests that the onset of mucosal damage is dependent on an individual's vulnerability to bacterial virulence and medication toxicity. Hospitalization data can be used to identify the complications, however it can be challenging to identify the denominator of individuals at risk for peptic ulcers. Patients with peptic ulcers of any cause may experience complications. There may be an increased risk of complications with giant ulcers and pyloric channel ulcers. PUD can result in five primary complications: bleeding, perforation, penetration, blockage, and gastric CA. The primary PUD problems will be covered in details in this review.

Introduction

The word "peptic ulcer" describes an acid-peptic injury to the digestive tract that reaches the submucosa through a mucosal breach. Peptic ulcers can occur in the oesophagus or Meckel's diverticulum, but they are more commonly found in the stomach or proximal duodenum. Peptic ulcers in the duodenum or stomach are referred to in this review as peptic ulcer disease. The majority of peptic ulcer disorders were previously believed to be caused by hypersecretory acidic environments, food, or stress; however, the identification of Helicobacter pylori infection and the extensive use of nonsteroidal anti-inflammatory drugs (NSAIDs) during the latter half of the 20th century have altered this understanding (1).

It has been estimated that the lifetime prevalence of peptic ulcer disease in the general population is between 5 and 10%, with an annual incidence of 0 to 0.3 percent. The incidence and prevalence of peptic ulcer disease, however, are likely now lower than these estimates globally, particularly in high-income nations, as epidemiological studies have demonstrated a sharp decline in the disease's rates of hospital admissions, mortality, and incidence over the previous 20–30 years. These declining numbers may result from the development of novel treatments or from a cohort trend for which there are no adequate known explanations (such as H pylori infection and NSAID use) (2).

The two main risk factors for duodenal and stomach ulcers are *H pylori* and NSAID or aspirin use. Nonetheless, peptic ulcer disease only rarely develops in those with H pylori infection, NSAID use, or aspirin use, indicating that individual vulnerability to bacterial virulence and medication toxicity is crucial to the start of mucosal damage (3).

The risk of complications from peptic ulcer disease is increased by four times for NSAID users and by two times for aspirin users as compared to non-users.

Comorbidity and aging are the main causes of problems, in addition to the use of NSAIDs, aspirin, and H pylori infection. Upper gastrointestinal bleeding is significantly increased when NSAIDs or aspirin are used together with corticosteroids, aldosterone antagonists, selective serotonin reuptake inhibitors, or anticoagulants (4).

Peptic ulcer disease symptoms are non-specific, which limits their predictive value. Individuals who suffer from duodenal ulcers often experience nighttime abdominal pain or hunger. In contrast, those suffering from stomach ulcers experience weight loss, nausea, vomiting, and postprandial abdominal pain. Untreated peptic ulcer patients usually experience recurring symptoms as a result of spontaneous healing, which happens as long as the underlying cause (such as a H pylori infection or NSAID use) is present. Peptic ulcer disease often presents with few or no symptoms in elderly patients (5).

Anti-secretory therapy may be initiated as part of empirical treatment if clinical signs point to a potential peptic ulcer illness and no alarm signals are observed. Additionally, as H. pylori is frequently the cause of PUD, individuals under the age of 55 should employ a test and treat approach that includes a non-invasive H. pylori test (stool antigen or urea breath test) (6). Endoscopy is advised to make a diagnosis in elderly patients and those exhibiting warning symptoms. A family history of upper gastrointestinal cancer, iron deficiency anemia, weight loss, early satiety, dysphagia or odynophagia, or new upper GI symptoms in patients over 55 are alarm indicators. For the diagnosis of PUD, esophagogastroduodenoscopy (EGD), often known as upper endoscopy, is the gold standard. It is can be used to detect *H. pylori* with gastric biopsies and can also rule out malignancy (7).

The goal of treatment is often to identify the causes of PUD. Eradication alone will result in ulcer healing and stop additional mucosal damage in PUD linked to H.

pylori. But therapy is getting harder since H. pylori is becoming more resistant to antibiotics. A proton pump inhibitor (PPI), clarithromycin, amoxicillin, or metronidazole (for individuals allergic to penicillins) are the first line treatments for eliminating H. pylori. These treatments are administered for seven to fourteen days. PPIs and antibiotics combine to completely eradicate H. pylori. In many nations, triple therapy's efficacy is now below 70% as a result of rising antibiotic resistance. When local rates of clarithromycin resistance are more than 15%, clarithromycin-based regimens should be avoided since susceptibility testing is frequently unavailable in clinical practice. Clarithromycin resistance rates are high (>20%) across the United States (8).

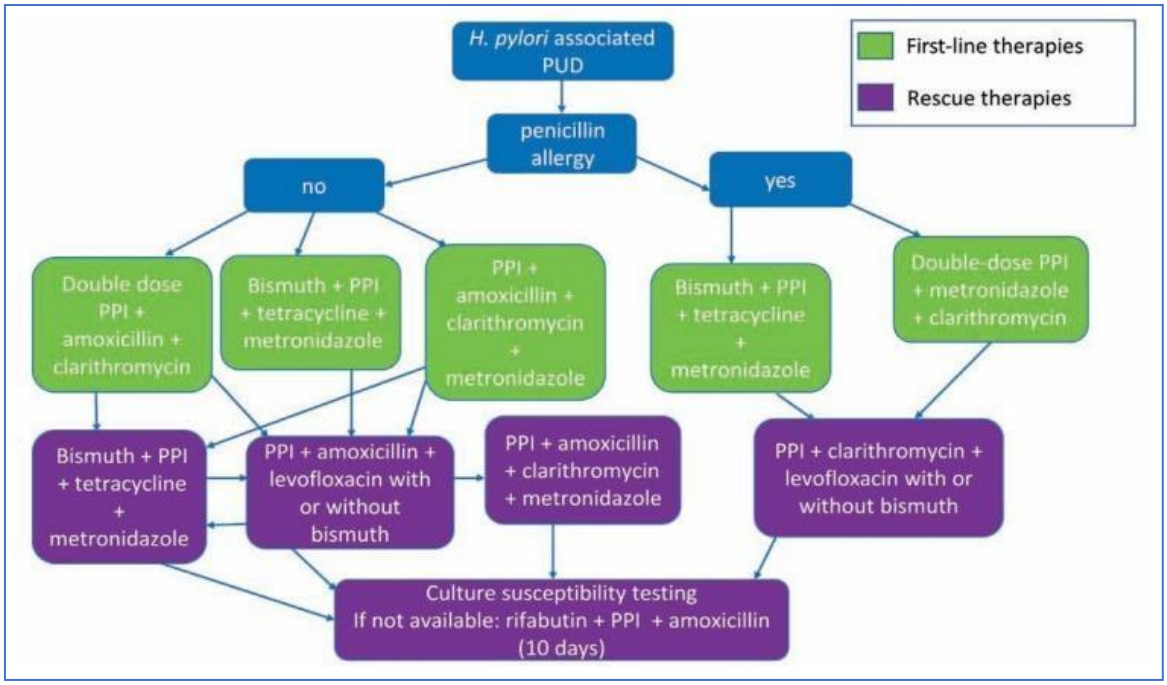

Figure 1. medical treatment of H. pylori

If the offending medication is stopped, ulcers linked with PUD caused by NSAIDs or aspirins typically heal after 6–8 weeks of PPI therapy. With continuous NSAID use, ulcer healing is still possible but will take longer. Patients using aspirin

may begin anti-secretory medication to avoid PUD. PPIs are significantly more successful than other medicines in treating NSAID-associated PUD, while they can also be used in conjunction with misoprostol, sucralfate, and H2 blockers. Sucralfate is not useful in treating or preventing NSAID-associated stomach ulcers, although it is successful in treating NSAID-associated duodenal ulcers. Misoprostol's adverse effect profile, which includes gastrointestinal distress and abortifacient reactions, sometimes limits its use in addition to its lackluster effectiveness (9).

Both medication compliance with PPI use and unintentional NSAID use should be investigated in cases of refractory ulcers. To assess healing, a follow-up endoscopy is necessary for all stomach ulcers after six to eight weeks. Biopsies must be obtained during a repeat endoscopy if a stomach ulcer has not healed in order to rule out gastric cancer (10).

In this short review, we will discuss in details the most common complications of peptic ulcer disease and its impact on general health condition.

Complications of peptic ulcer disease

The proportional frequency of the various sequelae is not well-documented, although estimates place their incidence between 1% and 2% per ulcer patient every year of follow-up. The hospitalization data can be used to identify the problems, however it can be challenging to identify the denominator of individuals at risk for peptic ulcers. Peptic ulcer patients of any origin may experience complications. Complication rates may be increased in cases of pyloric channel ulcers and giant ulcers. Peptic ulcer disease (PUD) is more common in older adults now than it was a few decades ago (11).

Hemorrhage

The most common PUD consequence is bleeding, which is becoming more common than stenosis and perforation. Nonsteroidal anti-inflammatory medications (NSAIDs) or Helicobacter pylori-induced peptic ulcers can be extremely dangerous if they rupture or hemorrhage into the duodenum or stomach. A life-threatening amount of bleeding can occur in up to 15% of patients with ulcers. Compared to ulcers generated by H. pylori, NSAID-induced ulcers are more prone to bleed. Those with significant diseases like heart difficulties and the elderly are the populations most at danger (12).

When a blood artery in the stomach lining is eroded by an ulcer, it might result in bleeding from the stomach. Blood in stools or possibly black, sticky-looking feces may be seen when this happens. Given that gastrointestinal bleeding can be fatal, this should be treated as an emergency if it occurs. Acute bleeding in the upper gastrointestinal tract (AUGI) is a common, expensive, and possibly fatal condition. To avoid negative consequences, it needs to be handled quickly and correctly.

Hospital admissions for AUGI hemorrhage outnumber those for DVT or congestive heart failure (12).

An upper endoscopic examination is necessary for all AUGI patients in order to identify the bleeding lesion, evaluate the risk it poses, treat it, and lower the chance of further bleeding. The standard of care is thought to be endoscopy within the first twenty-four hours of AUGI. More than 90% of patients have their bleeding stopped by endoscopic therapy; nevertheless, 10% to 25% of individuals experience recurrent bleeding. For endoscopic hemostasis, any significant coagulopathy must be reversed using platelet or fresh frozen plasma infusions. However, it does not seem that coagulopathy during the first bleeding and endoscopy is linked to increased risks of recurrent bleeding after endoscopic treatment for nonvariceal AUGI (13).

Perforation

NSAID use is the primary cause of perforated peptic ulcers, which are currently most commonly observed in elderly people, especially women. Clinical signs of sepsis and even shock may be present, as well as a history of abrupt onset upper abdomen pain. But in the case of a little leak or a hole from the stomach's posterior wall into the smaller sac, the symptoms could be far more modest, necessitating a high index of suspicion. A plain erect chest X-ray will only show apparent sub-diaphragmatic gas in about 50% of individuals (14).

Although rarely to the same degree as in a patient with acute pancreatitis, serum amylase levels may be elevated. CT is always diagnostic in situations where diagnosis is challenging, if the patient is healthy enough. With a few exceptional exceptions, such as severe septic shock coupled with pre-existing frailty or if the patient is well with few symptoms, the patient should be revived and made ready for

surgery. Only then should a trial of conservative management with nasogastric tube drainage and intravenous antibiotics be taken into consideration. Closing or covering the ulcer defect and performing a complete peritoneal lavage to lessen peritoneal contamination with gut luminal contents are the cornerstones of surgical intervention. This is most often performed through an upper midline incision, covering of the defect with an omental patch; a technique first described by Graham in 1937 (15).

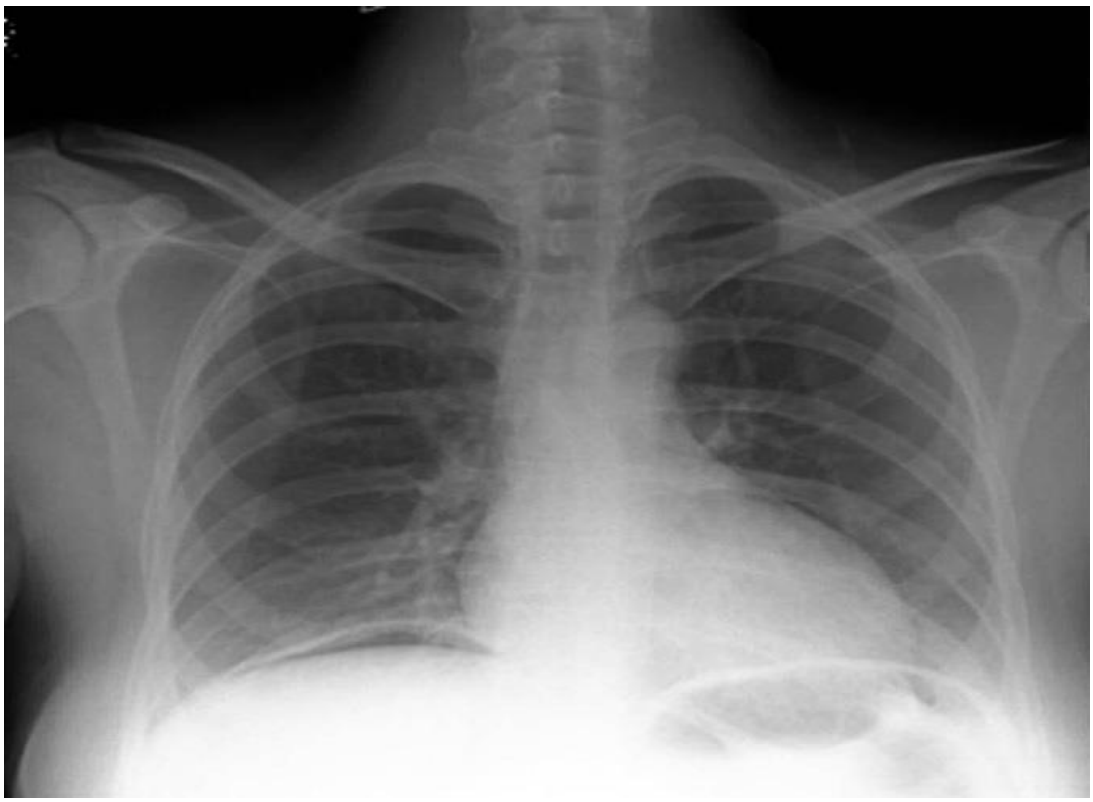

Figure 2. Free sub-diaphragmatic gas on erect postero-anterior chest X-ray in perforated peptic ulcer.

The surgical repair of a perforated peptic ulcers carries a substantial risk of morbidity and mortality, despite its seemingly straightforward idea. Shock at admission, metabolic acidosis, tachycardia, acute renal failure, low blood albumin, a high American Society of Anesthesiologists (ASA) score, and a preoperative delay

of more than 24 hours are preoperative variables linked to increased mortality. The implementation of a multidisciplinary and multi-modal perioperative care strategy can dramatically lower 30-day mortality (by more than one-third), albeit it still remains 17%, according to a recent multicenter randomized controlled trial (RCT). (16).

Pyloric obstruction

Stenosis is a less common complication of peptic ulcers that arises from the disease's progression owing to inflammation, edema, and muscle spasm, followed by a scarring-causing healing phase. The frequency range that has been observed is between 5 and 8%. In specifics, a number of variables, some pathological and some functional, contribute to the development of this problem. The pathogenic variables include inflammation, edema, fibrosis, and scarring and stenosis; the functional factors include spasm, pyloric dysmotility, and loss in stomach motility as a result of peptic ulcer illness. Reversible gastric blockage is caused by the initial dysfunctional phase, which includes inflammation and edema; irreversible obstruction is brought on by the subsequent phase of fibrosis and scarring (17).

Most patients with symptoms of obstruction of the gastric outlet have a history of peptic ulcer disease. The symptoms that define the clinical appearance include vomiting, nausea, early satiety, anorexia, and epigastric pain. Weight loss and a decline in overall health follow this protracted untreated clinical condition. Reduced antiacid medication efficacy is a common symptom. This is a hint that suggests a situation of changed acid stomach secretion: Pyloric blockage causes stasis when the pH of the stomach rises due to a rise in the gastrin problem and excessive acid output (18).

Typically, endoscopy, endo-biopsy, and imaging tests like CT scans and regular radiography are used to diagnose stenosis and rule out malignancy. Restoring hydroelectrolytic equilibrium and stomach decompression by nasogastric tube for 48–72 hours constitute the foundation of the first medical management. These treatments sometimes make it possible to resume an oral diet and regain nutritional status. A few studies indicate that treating the H. pylori infection has a beneficial effect on clearing the obstruction in the outflow (19).

Penetration

This consequence arises when an ulcer penetrates the wall of the stomach or duodenum, but instead of freely entering the peritoneal cavity, it burrows into an organ that is nearby. 15% of stomach ulcers and about 25% of duodenal ulcers have it. In most cases, the pancreas, liver, or omentum are adjacent organs that expand. While an uncomplicated ulcer may resemble the clinical presentation, the pain is typically more intense and long-lasting. No amount of food can ease pain, and in fact, it can exacerbate it and cause the patient to wake up at night more frequently. Pain radiating to the back usually indicates a gastrohepatic omentum penetration, which can also cause pain to the right upper quadrant or pancreas. Rarely, penetrating peptic ulcers may form fistulas between the duodenum and bile duct (choledoco-duodenal fistula) or between the stomach and colon (gastro-colic fistula) (20).

Gastric cancer

Since Cruveilhier first made a clear distinction between chronic ulcer and cancer in 1839, both clinically and pathologically, the relationship between peptic ulcer and stomach cancer has been contested. Later, in the 1940s and 1950s, there was discussion on the distinctions between duodenal and stomach ulcers. Although there was a tendency for these disorders to be mutually exclusive, no explanation

was provided for the observed disparities. With the identification of Helicobacter pylori by Warren and Marshall in 1982, the understanding of gastroduodenal disease was completely transformed, and it is now widely acknowledged that this bacteria is responsible for at least 60% to 70% of gastric ulcers and 95% of duodenal ulcers (21).

Although 2% of patients with a diagnosis of gastric ulcer also had a concomitant stomach cancer, the malignant degeneration of gastric ulcers seems to be uncommon. Chemically generated tumors in rodents typically form in or around ulcers, while the incidence of tumors in rats does not rise when ulcers are present. Therefore, even though gastric ulcers are most likely not the cause of stomach cancer, the fact that the two conditions are positively correlated shows that they share some risk factors (22).

On the other hand, there is a strong inverse relationship between stomach cancer and duodenal ulcer disease; however, the majority of the evidence for this relationship comes from small studies or case series. The question of whether peptic ulcers and stomach cancer are associated is still relevant, especially in light of the identification of H. pylori as a potential key player in the emergence of duodenal, gastric, and stomach cancers. By causing chronic gastritis, which results in the generation of free radicals by inflammatory cells, nitric oxide, nitrates, and nitrosamines by macrophages, and accelerated cell turnover, H. pylori infection may have an impact on the initial phases of gastric carcinogenesis. It appears likely that a few variables influencing duodenal ulcer patients alter the H. pylori-related risk of stomach cancer. One indicator of a potential protective factor is the finding that patients with multifocal atrophic gastritis are more likely to develop stomach cancer and gastric ulcers. Less is known about the relationship between this kind of gastritis and duodenal ulcers (23).