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Emergency Assessment, Initial Stabilization, and Definitive Management of Perforated Peptic Ulcer Disease in the Emergency Department-An Updated Review.

Abduallah Hamad Mayuf Alanazi[(1)] , Asma Ali Mayiq Alanazi[(2)] , Mjahd Mufeed Aljawad[(3)] , Ahmed Mofareh Alanazi[(3)] , Khalid Mohammed Saedd Al Jabir[(3)] , Faisal Mukaymin Alanazi[(3)] , Hamoud Nahi Alanazi[(4)]

(1)Saudi Red Crescent Authority, Sector 1, Arar, Saudi Arabia
(2)Ministry of Health, Arar, Saudi Arabia
(3)Saudi Red Crescent Authority, Northern Borders Region, Arar, Saudi Arabia
(4)Saudi Red Crescent Authority, Sakaka, Al-Jouf, Saudi Arabia

Abstract

Background: Perforated peptic ulcer (PPU) is a life-threatening complication of peptic ulcer disease characterized by disruption of the gastric or duodenal wall, resulting in leakage of gastrointestinal contents into the peritoneal cavity. Despite advances in diagnostic imaging, surgical techniques, and perioperative care, PPU remains associated with substantial morbidity and mortality, particularly among older patients and individuals with comorbidities or delayed presentation.

Aim: This review aims to provide a comprehensive overview of PPU, emphasizing its etiology, epidemiology, pathophysiology, histopathological characteristics, clinical presentation, diagnostic evaluation, treatment strategies, differential diagnosis, and prognosis.

Methods: A narrative review approach was used to synthesize the available literature on PPU, focusing on its underlying mechanisms, clinical manifestations, diagnostic methods, and contemporary management principles. Relevant evidence addressing conservative treatment, surgical intervention, antimicrobial therapy, and prognostic determinants was considered.

Results: PPU most commonly develops in association with Helicobacter pylori infection and nonsteroidal anti-inflammatory drug use. Clinical presentation typically includes sudden-onset abdominal pain and signs of peritoneal irritation, although manifestations may be atypical in older or critically ill patients. Diagnosis relies on clinical assessment, laboratory investigations, and imaging, particularly computed tomography. Management requires prompt resuscitation, broad-spectrum antibiotics, acid suppression, and timely surgical assessment. Operative repair remains the principal treatment for most patients, whereas nonoperative management is appropriate only in carefully selected cases without ongoing leakage or clinical deterioration. Delayed treatment, advanced age, shock, and comorbidities are important predictors of adverse outcomes.

Conclusion: Early recognition, rapid resuscitation, appropriate source control, and individualized management are essential to improve outcomes in PPU.

Keywords: Perforated peptic ulcer; peptic ulcer disease; Helicobacter pylori; peritonitis; acute abdomen; surgical management; prognosis.

Introduction

Peptic ulcer disease (PUD) is a gastrointestinal disorder characterized by the disruption of the mucosal integrity of the upper digestive tract, resulting in the formation of ulcers that penetrate beyond the mucosal layer and extend into the underlying submucosa or deeper tissues. These lesions most frequently develop in the stomach and proximal duodenum, although they may occur in other anatomical locations, including the esophagus and, less commonly, a Meckel diverticulum containing ectopic gastric mucosa. The development of peptic ulcers reflects an imbalance between aggressive factors, such as gastric acid, pepsin, and mucosal irritants, and the protective mechanisms responsible for maintaining gastrointestinal epithelial integrity. Disruption of the mucosal barrier, impaired tissue repair, and persistent exposure to injurious factors contribute to ulcer formation and progression. [1] The clinical significance of PUD extends beyond localized mucosal injury because the disease may progress from an asymptomatic condition to a potentially life-threatening gastrointestinal emergency, depending on the depth of ulceration, anatomical location, and occurrence of complications. The clinical presentation of PUD is heterogeneous, and affected individuals may remain asymptomatic for prolonged periods before developing recognizable manifestations. Symptomatic patients commonly report epigastric discomfort, burning pain, postprandial symptoms, nausea, or dyspepsia, although these manifestations

*Corresponding author abdullahhamad@gmail.com (Abduallah Hamad Mayuf Alanazi).
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are neither sufficiently sensitive nor specific to establish the diagnosis independently. The clinical course may be complicated by gastrointestinal hemorrhage, perforation, or gastric outlet obstruction, each of which requires a distinct diagnostic and therapeutic approach. Gastrointestinal bleeding may result from ulceration into an underlying blood vessel, whereas gastric outlet obstruction can develop secondary to inflammatory edema, fibrosis, or scar formation around the pylorus or proximal duodenum. Perforation represents one of the most critical complications because the ulcer penetrates the full thickness of the gastrointestinal wall, allowing gastric or duodenal contents to escape into the peritoneal cavity. This process can precipitate chemical peritonitis, bacterial contamination, systemic inflammatory responses, and, in severe cases, septic shock and multiorgan dysfunction. The potential for rapid clinical deterioration makes early recognition and appropriate intervention essential to reducing morbidity and mortality [1].

Peptic ulcers are conventionally classified according to their anatomical location as gastric or duodenal ulcers, although their clinical characteristics and associated risk factors may overlap. Important etiological factors include Helicobacter pylori infection and the use of nonsteroidal anti-inflammatory drugs, both of which can compromise mucosal defense and contribute to ulcer development. Additional factors, including smoking, severe physiological stress in critically ill patients, and certain comorbid conditions, may influence disease occurrence, healing, and complication risk. From an emergency medicine perspective, perforated peptic ulcer disease warrants particular attention because its initial manifestations may overlap with other causes of acute abdominal pain, potentially delaying definitive diagnosis. Emergency clinicians must rapidly evaluate the patient's hemodynamic status, identify clinical evidence of peritonitis, initiate resuscitative measures, and arrange appropriate imaging and surgical consultation. Effective management requires coordinated multidisciplinary care involving emergency physicians, surgeons, anesthesiologists, and critical care specialists when indicated. Accordingly, understanding the pathophysiology, clinical presentation, diagnostic evaluation, and immediate management of perforated peptic ulcers is fundamental to optimizing emergency care and improving patient outcomes [1][2].

Etiology

Peptic ulcer disease (PUD) is a multifactorial gastrointestinal disorder that develops when the protective mechanisms of the gastric and duodenal mucosa become insufficient to withstand the damaging effects of gastric acid, pepsin, and other injurious factors. Historically, the pathogenesis of peptic ulceration was attributed primarily to excessive gastric acid secretion, dietary practices, and psychological stress. However, advances in gastrointestinal research have established Helicobacter pylori infection and the use of nonsteroidal anti-inflammatory drugs (NSAIDs), including low-dose aspirin, as the principal etiological factors associated with the development of peptic ulcers. These factors can independently or collectively disrupt mucosal defense, impair tissue repair, and increase susceptibility to ulcer formation and subsequent complications. [1][2][3] Although the relative contribution of individual risk factors varies among patients, understanding the underlying etiology is essential for identifying individuals at increased risk, implementing preventive strategies, and selecting appropriate therapeutic interventions. This is particularly important in patients who develop perforated peptic ulcers, in whom the interaction between mucosal injury and impaired healing may contribute to full-thickness gastrointestinal wall disruption. Helicobacter pylori is a Gram-negative, spiral-shaped bacterium that colonizes the gastric mucosa and induces persistent inflammation. Its pathogenic effects involve several mechanisms, including disruption of the mucosal barrier, inflammatory injury, alterations in gastrin and somatostatin regulation, and changes in gastric acid secretion. Depending on the distribution and severity of gastritis, infection may contribute to either increased acid exposure in the duodenum or impaired mucosal protection within the stomach. These processes facilitate ulcer formation and may increase the likelihood of recurrent disease when the infection remains untreated. NSAIDs represent another major etiological category because they inhibit cyclooxygenase activity and reduce the synthesis of prostaglandins involved in maintaining gastrointestinal mucosal integrity. Prostaglandin depletion compromises mucus and bicarbonate secretion, reduces mucosal blood flow, and impairs epithelial repair. Aspirin additionally inhibits platelet function, increasing the clinical consequences of ulcer-related bleeding. The risk of NSAID-associated ulceration is influenced by the specific medication, dosage, duration of exposure, patient age, previous ulcer history, and concurrent use of medications such as anticoagulants or corticosteroids. [1][2][3]

Several additional factors may contribute to the development or progression of peptic ulcer disease. Cigarette smoking can impair mucosal defense and ulcer healing, while alcohol consumption may aggravate gastrointestinal irritation and mucosal injury, particularly with excessive intake. Lower socioeconomic status may be associated with increased exposure to H. pylori through crowded living conditions, inadequate sanitation, and barriers to healthcare access. Nutritional deficiencies and unfavorable dietary practices may also influence general mucosal health, although they are not considered the principal direct causes of most peptic

ulcers. Psychological stress and anxiety have historically been implicated in ulcer pathogenesis; however, they should be regarded as potential modifiers of symptoms, health behaviors, and disease outcomes rather than established primary causes of most cases. Similarly, corticosteroid use may increase gastrointestinal complication risk, particularly when combined with NSAIDs, although corticosteroids alone are less strongly associated with ulcer formation than NSAIDs. Other recognized risk factors include atrophic gastritis, vitamin deficiencies, Zollinger–Ellison syndrome, multiple endocrine neoplasia (MEN) syndromes, and hyperparathyroidism. Zollinger–Ellison syndrome, which is characterized by gastrin-secreting tumors, can produce excessive gastric acid secretion and recurrent, multiple, or treatment-resistant ulcers. Hyperparathyroidism may contribute to acid-related disease through associated metabolic disturbances and its relationship with certain endocrine neoplasia syndromes. Gastric ulcers may also arise in association with factors that compromise local mucosal integrity, including gastric stasis, impaired mucosal perfusion or ischemia, and duodenogastric reflux, in which the retrograde movement of duodenal contents exposes the stomach to bile and other potentially injurious substances. [4] Collectively, these etiological factors demonstrate that peptic ulcer disease results from interactions between infectious, pharmacological, physiological, and patient-related determinants. Identifying these contributors is particularly relevant in emergency settings because recognition of persistent ulcerogenic exposures and underlying disorders can guide definitive treatment, reduce recurrence, and support strategies to prevent severe complications, including perforation, peritonitis, and septic shock [1][2][3].

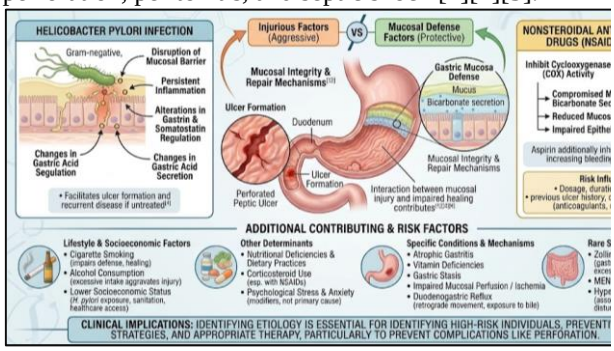


Figure-1: Etiology of Peptic Ulcer Disease (PUD).

Epidemiology

Peptic ulcer disease (PUD) remains a significant gastrointestinal disorder worldwide, although its prevalence and incidence have declined in many populations over recent decades. The lifetime prevalence of PUD is currently estimated to range between 5% and 10%, reflecting variations in population characteristics, exposure to established risk factors, diagnostic practices, and access to effective treatment. The disease has historically been more prevalent in certain populations with high rates of Helicobacter pylori infection and limited access to healthcare services, whereas lower prevalence has generally been reported in many developed countries. This epidemiological transition has been influenced by improvements in sanitation, changes in the prevalence of H. pylori infection, greater recognition of ulcerogenic medications, and the widespread availability of acid-suppressive therapies and eradication regimens. Nevertheless, the distribution of PUD remains heterogeneous across geographical regions and demographic groups, indicating that the burden of disease continues to depend on the interaction between infectious, pharmacological, socioeconomic, and patient-related determinants. The decline in the overall incidence of PUD has also been accompanied by a reduction in the frequency of associated complications, although serious ulcer-related presentations continue to place a considerable burden on emergency departments and hospital services. [1] Despite these favorable epidemiological trends, complications of PUD remain clinically important because they may require urgent hospitalization, endoscopic intervention, surgery, or critical care. Gastrointestinal bleeding, perforation, and gastric outlet obstruction represent the principal complications responsible for severe disease-related morbidity. In the United States, these complications collectively account for approximately 150,000 hospitalizations annually, demonstrating that the declining incidence of uncomplicated ulcer disease has not eliminated the need for emergency management of advanced presentations. [5] Among these complications, upper gastrointestinal bleeding is the most frequently reported, whereas perforation is generally considered the next most common. The annual incidence of peptic ulcer-related upper gastrointestinal bleeding has been estimated at approximately 19–57 cases per 100,000 individuals, compared with an estimated 4–14 cases per 100,000 individuals for ulcer perforation. [6] Although perforation occurs less frequently than bleeding, its clinical significance is substantial because the escape of gastric or duodenal contents into the peritoneal cavity can lead to peritonitis, systemic infection, hemodynamic instability, and death if definitive treatment is delayed.

Age represents an important epidemiological determinant of complicated PUD. Older adults are disproportionately affected by clinically significant ulcer complications because of the increased prevalence of chronic diseases, greater exposure to medications that impair mucosal protection, and the potential presence of atypical clinical manifestations. Approximately 60% of patients with PUD are reported to be older than 60 years, highlighting the importance of age-related risk assessment in clinical practice. [6] Furthermore, older patients may experience delayed diagnosis because symptoms can

be less pronounced despite substantial underlying pathology. Comorbid cardiovascular, renal, and metabolic disorders may further complicate resuscitation, anesthesia, and postoperative recovery, particularly in patients presenting with perforation and sepsis. The epidemiology of complicated PUD is also closely associated with two major modifiable risk factors, namely H. pylori infection and the use of nonsteroidal anti-inflammatory drugs (NSAIDs). Both are recognized contributors to ulcer-related bleeding and perforation. [6] Persistent H. pylori infection promotes chronic gastric inflammation and compromises mucosal integrity, whereas NSAIDs reduce protective prostaglandin synthesis and impair the physiological mechanisms responsible for maintaining the gastrointestinal mucosal barrier. Concurrent exposure to multiple risk factors may further increase the likelihood of severe complications. Consequently, the epidemiological assessment of PUD should extend beyond estimates of overall prevalence to consider the distribution of complicated disease, the characteristics of vulnerable populations, and the preventable determinants of adverse outcomes. From an emergency medicine perspective, these patterns emphasize the importance of early recognition, prompt diagnostic evaluation, appropriate resuscitation, and timely surgical consultation for suspected perforated peptic ulcer, particularly among older adults and patients with established ulcerogenic exposures.

Pathophysiology

The pathophysiology of peptic ulcer disease (PUD) involves a disruption of the protective mechanisms that preserve the structural and functional integrity of the gastric and duodenal mucosa. Under physiological conditions, the gastrointestinal mucosa is protected against gastric acid and pepsin by several interdependent mechanisms, including the secretion of mucus and bicarbonate, adequate mucosal blood flow, intact epithelial tight junctions, and continuous epithelial regeneration. Prostaglandins play a central role in maintaining these protective processes by promoting mucus and bicarbonate secretion, supporting mucosal perfusion, and facilitating tissue repair. Peptic ulceration develops when these defense mechanisms become compromised and the mucosa is exposed to sustained chemical and inflammatory injury. Infection with Helicobacter pylori and exposure to nonsteroidal anti-inflammatory drugs (NSAIDs), including low-dose aspirin, represent two major mechanisms responsible for disrupting mucosal integrity and initiating ulcer formation. [1] Helicobacter pylori contributes to ulcerogenesis through the combined effects of bacterial virulence factors and the host inflammatory response. Following colonization of the gastric mucosa, the organism induces persistent inflammation that may impair epithelial integrity and alter the regulation of gastric acid secretion. The resulting mucosal injury depends on the distribution of gastritis, bacterial characteristics, and individual host responses. In contrast, NSAIDs and aspirin primarily compromise mucosal protection through the inhibition of cyclooxygenase enzymes, particularly cyclooxygenase-1 (COX-1), which is responsible for producing prostaglandins that support gastrointestinal mucosal homeostasis. Reduced prostaglandin synthesis decreases mucus and bicarbonate production, compromises mucosal blood flow, and impairs epithelial restitution. These effects increase the susceptibility of the gastric and duodenal lining to acid-mediated injury. Once the mucosal barrier is disrupted, gastric acid and pepsin can penetrate the underlying tissues, promoting inflammation, tissue necrosis, and progressive ulceration. If the injurious process persists, the ulcer may extend through the mucosa and submucosa into the muscularis propria and eventually reach the serosal surface. Complete penetration of the gastrointestinal wall results in perforation, permitting gastric or duodenal contents to enter the peritoneal cavity. [1][7]

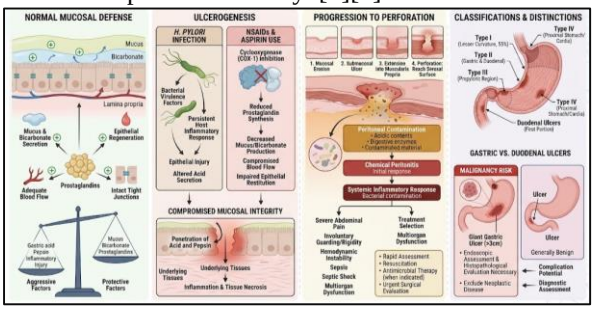


Figure-2: Peptic ulcer pathophysiology.

Perforated peptic ulcer represents a critical progression of the ulcerative process because the release of acidic gastric contents, digestive enzymes, and potentially contaminated intestinal material into the abdominal cavity initiates peritoneal inflammation. The initial response may involve chemical peritonitis, followed by bacterial contamination and an escalating systemic inflammatory response. As the condition progresses, patients may develop severe abdominal pain, involuntary guarding, abdominal rigidity, hemodynamic instability, and manifestations of sepsis. Continued contamination can result in septic shock and multiorgan dysfunction, particularly when diagnosis and definitive treatment are delayed. The severity of the clinical presentation depends on the location and size of the perforation, the volume and nature of the escaped contents, the duration of contamination, and the patient's physiological reserve. These mechanisms explain why rapid assessment, resuscitation, antimicrobial therapy when indicated, and urgent surgical evaluation are central components of emergency management. [7] Gastric ulcers are anatomically classified into four types according to their location. Type I ulcers occur along

the lesser curvature, typically in the antral region. Type II ulcers are associated with concurrent gastric and duodenal ulceration. Type III ulcers are located in the prepyloric region, whereas Type IV ulcers develop in the proximal stomach near the cardia. [8] Gastric ulcers are most frequently identified along the lesser curvature, accounting for approximately 55% of cases, while combined gastric and duodenal ulceration represents another recognized pattern. Duodenal ulcers most commonly occur in the first portion of the duodenum, where exposure to gastric acid and alterations in mucosal defense contribute to tissue injury. Anatomical location is clinically relevant because it influences diagnostic assessment, potential complications, and the selection of treatment.

An important distinction between gastric and duodenal ulcers concerns their potential association with malignancy. Although duodenal ulcers are generally considered benign, gastric ulcers may represent malignant lesions or coexist with gastric cancer. Consequently, appropriate evaluation of gastric ulcers is necessary to distinguish benign ulceration from underlying neoplastic disease. Giant gastric ulcers, conventionally defined as lesions exceeding 3 cm in diameter, have been associated with a reported malignancy risk ranging from 6% to 23%. Previous literature has also documented malignancy rates ranging from 2.4% to 21% among endoscopically diagnosed gastric ulcers, reflecting differences in patient populations, diagnostic criteria, and study methodologies. [9][10] These findings support the importance of endoscopic assessment and histopathological evaluation when indicated. In patients presenting with perforation, emergency stabilization and definitive management take priority, while subsequent investigation should address the underlying cause, exclude malignancy where appropriate, and reduce the risk of recurrent ulceration.

Histopathology

The histopathological features of peptic ulcer disease reflect progressive tissue injury, inflammatory infiltration, and reparative processes involving the gastric or duodenal wall. Microscopically, a typical peptic ulcer demonstrates a combination of acute inflammatory changes, tissue necrosis, chronic inflammation, granulation tissue formation, and fibrosis. These findings vary according to the duration of the ulcer, the severity of mucosal damage, and the stage of healing. Classically, the histological architecture of a peptic ulcer is characterized by four distinct zones extending from the ulcer surface toward its deeper layers. The superficial zone consists primarily of necrotic tissue, neutrophilic infiltration, cellular debris, and, in some cases, bacterial colonies. This layer reflects active tissue destruction and the acute inflammatory response to mucosal injury. Beneath it lies a zone of fibrinoid necrosis involving the ulcer base and margins, indicating substantial tissue damage and the degradation of cellular structures. The third zone contains chronic inflammatory cells, including lymphocytes, plasma cells, and macrophages, accompanied by granulation tissue. This tissue contains proliferating capillaries, fibroblasts, and extracellular matrix components that contribute to tissue repair and ulcer healing. The deepest zone is characterized by fibrous scar formation extending into the muscularis propria, reflecting chronic injury and repeated cycles of inflammation and repair. Blood vessels within this region may exhibit endarteritis obliterans, a pathological process involving inflammatory changes and narrowing of the vascular lumen that can compromise local tissue perfusion. These histopathological changes provide insight into the chronicity and severity of peptic ulceration. Persistent inflammation and vascular compromise may impair healing, promote further tissue destruction, and contribute to the progression of an ulcer into deeper gastrointestinal layers. When ulceration extends through the entire thickness of the gastric or duodenal wall, perforation may occur, resulting in contamination of the peritoneal cavity and the development of peritonitis. Histopathological examination is also important when evaluating gastric ulcers because malignant lesions may occasionally resemble benign peptic ulceration. Therefore, appropriate tissue sampling and pathological assessment, when clinically indicated, help establish the diagnosis, identify underlying malignancy, and guide subsequent management [9][10].

History and Physical

The clinical assessment of patients with peptic ulcer disease (PUD) requires a systematic evaluation of presenting symptoms, previous gastrointestinal complaints, relevant medical history, medication exposure, and physical examination findings. Although approximately 70% of individuals with PUD may initially remain asymptomatic, perforation typically produces an acute clinical presentation that requires urgent assessment and intervention. [5] Obtaining an accurate history may be challenging in certain populations, particularly young children, older adults, immunocompromised individuals, and patients with altered consciousness or cognitive impairment. In these circumstances, information from family members, caregivers, emergency personnel, and available medical records may help establish the onset and progression of symptoms. A detailed history should explore previous episodes of dyspepsia, upper abdominal pain, nausea, vomiting, gastrointestinal bleeding, and known peptic ulcer disease. Particular attention should be given to the use of nonsteroidal anti-inflammatory drugs (NSAIDs), aspirin, corticosteroids, anticoagulants, and other medications that may increase the risk of ulceration or its complications [5]. Dyspepsia and upper abdominal

pain are among the most frequently reported manifestations of uncomplicated PUD. Patients may describe epigastric burning, aching, discomfort, or pain that varies in intensity and duration. The pain may remain localized to the epigastrium or extend toward the right or left upper abdominal quadrants. Traditionally, gastric ulcer pain has been associated with eating or may worsen after meals, whereas duodenal ulcer pain may occur several hours after food intake, commonly within approximately two to five hours. However, these patterns are variable and cannot reliably distinguish gastric from duodenal ulceration without further investigation. Patients with ulcer-related bleeding may report nausea, vomiting of blood, or melena, characterized by black, tarry stools. In cases of substantial and rapid upper gastrointestinal hemorrhage, hematochezia or maroon-colored stools may occur because of accelerated intestinal transit. Associated manifestations, including dizziness, weakness, syncope, and palpitations, may indicate clinically significant blood loss and hemodynamic compromise [11].

Perforated peptic ulcer typically presents with the abrupt onset of severe epigastric pain, which may initially be localized but subsequently becomes generalized as gastric or duodenal contents enter the peritoneal cavity. The sudden onset and intensity of pain reflect acute peritoneal irritation. Patients may report that movement, coughing, deep inspiration, or changes in position aggravate their symptoms. Lightheadedness or syncope may accompany hypotension resulting from hemorrhage, fluid shifts, systemic inflammation, or sepsis. In some cases, pain may temporarily diminish several hours after the initial presentation, but this improvement does not necessarily indicate resolution of the underlying pathology. When perforation remains uncontained and medical attention is delayed, progressive peritoneal contamination may lead to abdominal distension, persistent pain, systemic inflammatory response syndrome (SIRS), sepsis, and circulatory instability. [5][11] A comprehensive physical examination is essential for identifying peritonitis and assessing the severity of the patient's condition. Initial evaluation should prioritize airway patency, respiratory status, circulation, mental status, and vital signs, with repeated measurements to detect deterioration. Typical findings in perforated PUD include diffuse abdominal tenderness, involuntary guarding, and abdominal rigidity. These signs indicate peritoneal irritation and should prompt urgent surgical evaluation. Reduced or absent bowel sounds may occur because of inflammatory ileus, while abdominal distension may develop as the condition progresses. Tachycardia, hypotension, tachypnea, fever, pallor, and altered mental status may indicate systemic inflammation, dehydration, sepsis, or shock. However, fever and pronounced peritoneal signs may be absent during the early stages or in older and immunocompromised patients. [5]

Additional examination findings may help identify associated complications. A digital rectal examination may reveal melena or blood in the stool, although it should be performed when clinically indicated and should not delay resuscitation or definitive investigation. Laboratory evaluation may subsequently identify anemia, leukocytosis, electrolyte disturbances, elevated lactate, or evidence of organ dysfunction, depending on the severity and duration of the disease. Nevertheless, neither normal vital signs nor initially unremarkable laboratory results reliably exclude perforation. The integration of symptom chronology, medication history, hemodynamic assessment, abdominal examination, and appropriate imaging is therefore essential for early diagnosis. In emergency practice, recognition of sudden severe abdominal pain accompanied by peritoneal signs should trigger prompt resuscitation, timely surgical consultation, and expedited diagnostic evaluation to minimize delays in definitive management and reduce the risk of sepsis and mortality [5][11].

Evaluation

The evaluation of suspected perforated peptic ulcer (PPU) constitutes a time-sensitive component of emergency care because delays in diagnosis and definitive treatment are associated with increased morbidity and mortality. The primary objectives are to establish the likelihood of gastrointestinal perforation, assess the severity of physiological compromise, identify associated complications, and exclude alternative causes of acute abdominal pain. Although the clinical history and physical examination may strongly suggest perforation, diagnostic investigations are essential to support the diagnosis, evaluate the patient's overall condition, and guide subsequent management. The initial assessment should proceed alongside resuscitation rather than delay it, particularly in patients presenting with hypotension, tachycardia, altered mental status, or signs of generalized peritonitis. Early involvement of the surgical team is warranted when clinical findings raise substantial suspicion of perforation, even before all diagnostic results become available [5][7]. Laboratory investigations provide important information regarding systemic inflammation, blood loss, metabolic disturbances, coagulation status, and possible organ dysfunction. A complete blood count (CBC) should be obtained to assess hemoglobin concentration, hematocrit, leukocyte count, and platelet count. Leukocytosis may support the presence of an inflammatory or infectious process, whereas anemia may indicate concurrent gastrointestinal bleeding or pre-existing hematological abnormalities. However, normal leukocyte counts and hemoglobin levels do not

exclude perforation, particularly during the early stages of presentation. A serum chemistry panel is required to evaluate electrolyte concentrations, renal function, glucose levels, and metabolic abnormalities that may influence resuscitation and perioperative management. Liver function tests may assist in evaluating alternative hepatobiliary causes of abdominal pain, while serum lipase is useful for investigating acute pancreatitis, which can produce overlapping clinical manifestations. Coagulation studies are indicated to identify abnormalities that may increase procedural bleeding risk or require correction before surgery. Blood group determination and antibody screening should also be performed because patients with severe gastrointestinal complications may require blood transfusion or urgent operative intervention [12].

Additional laboratory investigations should be selected according to the patient's clinical presentation and physiological status. In patients with suspected sepsis or systemic inflammatory response syndrome (SIRS), blood cultures should be obtained when feasible before antimicrobial administration, provided that this does not delay treatment. Serum lactate measurement can help identify tissue hypoperfusion and assess the severity of circulatory compromise. An elevated lactate concentration may indicate inadequate tissue oxygen delivery, impaired clearance, or other causes of metabolic stress and should be interpreted alongside hemodynamic findings and repeated measurements when clinically indicated. Urinalysis may assist in the differential diagnosis when urinary tract infection, nephrolithiasis, or another genitourinary disorder is suspected. Additional tests, including blood gas analysis, inflammatory markers, and renal or hepatic function measurements, may be appropriate in patients with shock, respiratory compromise, or suspected organ dysfunction. Laboratory results should be interpreted collectively because no single test can independently confirm or reliably exclude a perforated peptic ulcer [12]. Imaging plays a central role in confirming perforation and determining the extent of intra-abdominal contamination. Imaging should be performed as soon as the patient's condition permits, without postponing necessary resuscitation or urgent surgical intervention. Plain abdominal radiographs or erect chest radiographs may demonstrate free intraperitoneal air, also known as pneumoperitoneum, beneath the diaphragm. Although this finding can strongly support the diagnosis, plain radiography has limited sensitivity, and the absence of visible free air does not exclude gastrointestinal perforation. Computed tomography (CT) of the abdomen and pelvis is generally the preferred imaging modality in hemodynamically stable patients because it offers greater diagnostic sensitivity and can identify small volumes of free air, localized fluid collections, inflammatory changes, and potential sites of perforation. CT may also help distinguish PPU from other causes of acute abdominal pain, including bowel perforation from alternative causes, pancreatitis, and intra-abdominal inflammatory conditions [5][7].

Intravenous or oral contrast is not invariably necessary to demonstrate pneumoperitoneum. Nevertheless, intravenous contrast may improve the assessment of abdominal organs, inflammatory changes, vascular abnormalities, and alternative diagnoses in patients with undifferentiated abdominal pain or peritonitis. The choice of contrast should account for the diagnostic question, renal function, and clinical urgency. Ultimately, evaluation requires integration of clinical findings, laboratory results, and imaging evidence. In patients with hemodynamic instability or convincing generalized peritonitis, diagnostic procedures must not create avoidable delays in definitive surgical management. A coordinated approach involving emergency physicians, radiologists, surgeons, and critical care specialists facilitates timely diagnosis, appropriate resuscitation, and early intervention, thereby reducing the risk of progressive peritoneal contamination, septic shock, and adverse outcomes [12].

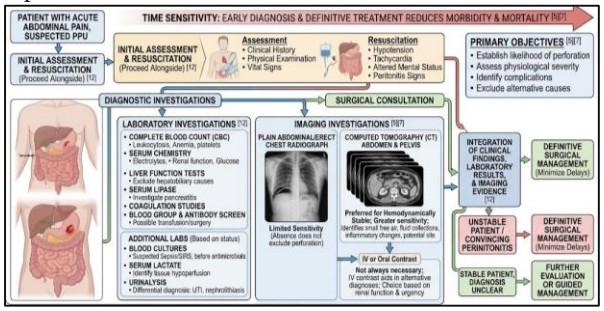


Figure-3: Emergent Evaluation of peptic ulcer disease.

Treatment / Management

Perforated peptic ulcer (PPU) is a life-threatening surgical emergency associated with substantial morbidity and mortality, with reported mortality rates approaching 30% in certain patient populations. The clinical outcome depends on several factors, including the time elapsed between perforation and definitive treatment, the extent of peritoneal contamination, the presence of sepsis or shock, and the patient's age and comorbidities. Early surgical intervention, effective resuscitation, antimicrobial therapy, and prompt management of sepsis constitute the principal components of treatment. [7] Initial management should begin immediately upon clinical suspicion and should proceed concurrently with diagnostic evaluation. Patients presenting with generalized peritonitis require urgent surgical consultation, even before definitive imaging confirmation, because unnecessary delays may allow progressive contamination of the peritoneal cavity and deterioration into septic shock. The initial emergency assessment should prioritize airway patency, respiratory function, circulation, mental status, and vital signs. Intravenous access

should be established promptly, and isotonic crystalloid solutions should be administered according to the patient's hemodynamic condition, with repeated assessment of blood pressure, heart rate, peripheral perfusion, urine output, and response to fluid resuscitation. Analgesics and antiemetics may be administered to control symptoms, while patients should generally remain nil by mouth in anticipation of possible surgery. Once perforated peptic ulcer is strongly suspected or confirmed, treatment should include early intravenous broad-spectrum antibiotics, intravenous proton pump inhibitor (PPI) therapy, and appropriate gastrointestinal decompression when indicated. A nasogastric tube may be inserted to aspirate gastric contents, reduce gastric distension, and minimize ongoing accumulation of gastrointestinal secretions. Urinary catheterization may be necessary in patients with shock or severe systemic illness to facilitate continuous monitoring of urine output as an indicator of renal perfusion and resuscitation adequacy. Electrolyte abnormalities, acid–base disturbances, and associated organ dysfunction should be identified and corrected. Blood cultures should be obtained before antibiotic administration when sepsis is suspected, provided that doing so does not delay treatment. Patients with significant hemorrhage or anticipated major operative intervention may require blood grouping, crossmatching, and transfusion according to clinical indications. These initial measures are intended to stabilize the patient, limit ongoing physiological deterioration, and prepare for definitive management.

Antimicrobial therapy is a central component of treatment because bacterial contamination of the peritoneal cavity can lead to intra-abdominal infection, systemic inflammatory response, and sepsis. Sepsis accounts for approximately half of reported mortalities associated with perforated peptic ulcers, highlighting the importance of early recognition and effective infection control. [7] Empirical antibiotics should provide coverage against enteric Gram-negative organisms and anaerobic bacteria, with the regimen selected according to the severity of illness, local resistance patterns, allergies, renal function, and institutional protocols. A third-generation cephalosporin combined with metronidazole represents one reasonable approach, while beta-lactam/beta-lactamase inhibitor combinations, such as ampicillin-sulbactam or piperacillin-tazobactam, may be appropriate alternatives in selected settings. [12] Broader antimicrobial coverage may be required in patients with septic shock, healthcare-associated infection, previous antibiotic exposure, or substantial risk of resistant organisms. Treatment duration should be guided by the adequacy of source control, clinical response, microbiological findings, and the presence of persistent infection. Definitive control of the perforation remains essential because antibiotics alone may not adequately control ongoing contamination. Intravenous PPI therapy is commonly administered to suppress gastric acid secretion and support ulcer management. By increasing intragastric pH, PPIs may improve the conditions required for platelet aggregation and clot stability, mechanisms that are particularly relevant when bleeding accompanies ulceration. However, the clinical efficacy of intravenous PPIs specifically for sealing a perforated ulcer has not been firmly established. Their use should therefore be regarded as an adjunct to comprehensive management rather than a substitute for definitive treatment. [1][13] Subsequent treatment should also address the underlying cause of ulceration, including testing for Helicobacter pylori and providing eradication therapy when infection is identified. Ulcerogenic medications should be discontinued or reviewed whenever clinically feasible, and appropriate gastroprotection should be considered when continued NSAID or aspirin therapy is necessary.

Early operative intervention remains the principal treatment for most patients with perforated peptic ulcer, particularly those with generalized peritonitis, significant pneumoperitoneum, ongoing leakage, or hemodynamic instability. Evidence indicates that mortality increases when surgery is delayed, emphasizing the importance of timely source control. [14] The operative procedure commonly involves identification of the perforation, closure of the defect with sutures, and reinforcement with an omental patch, frequently referred to as a Graham patch repair. Peritoneal lavage is performed to remove contaminated fluid and reduce the burden of intraperitoneal contamination. The volume of irrigation fluid is determined by operative findings and surgical judgment. Drains may be placed selectively according to the extent of contamination, the adequacy of source control, and the surgeon's assessment; routine drainage and a fixed removal schedule are not universally required. The abdomen is then closed, and the patient is monitored for postoperative complications, including persistent leakage, intra-abdominal abscess, wound infection, pneumonia, and organ dysfunction. Surgical repair may be performed through an open approach or laparoscopically, depending on patient stability, perforation characteristics, available expertise, and institutional resources. Laparoscopic repair may offer advantages in postoperative pain and recovery in appropriately selected patients, whereas open surgery may be preferable in patients with profound instability, extensive contamination, or circumstances requiring rapid access. The choice of technique should be individualized, as clinical outcomes depend on both patient characteristics and timely, effective source control.

Nonoperative management may be considered in a carefully selected minority of

patients, but it should not be regarded as routine treatment. Conservative management is appropriate only when the patient is hemodynamically stable, has no evidence of generalized peritonitis or uncontrolled sepsis, and investigations indicate that the perforation has sealed without ongoing leakage. The decision must be made by the surgical team with the capacity for continuous monitoring and immediate operative intervention if deterioration occurs. Traditional conservative management, historically associated with the Taylor regimen, includes intravenous fluids, broad-spectrum antibiotics, gastric decompression, acid suppression, serial abdominal examinations, and monitoring of vital signs, urine output, and laboratory parameters. [7][13] Persistent pain, worsening peritoneal signs, fever, hemodynamic deterioration, or evidence of ongoing leakage should prompt urgent reassessment for surgery. Age alone should not determine eligibility for conservative treatment, and patients with delayed presentation or severe symptoms generally require a more cautious approach. Following recovery, long-term management should focus on preventing recurrence and identifying the underlying cause of ulceration. Patients should undergo H. pylori testing and receive appropriate eradication treatment when indicated, followed by confirmation of eradication. Medication review, smoking cessation, and avoidance of unnecessary NSAID exposure are important preventive measures. Follow-up upper gastrointestinal endoscopy, commonly arranged at approximately 6–12 weeks when clinically indicated, is particularly important after gastric ulceration to assess healing and exclude malignancy. [9][10] Duodenal ulcers do not carry the same inherent malignancy concern, so the need for repeat endoscopy depends on the clinical course and specific indications. A coordinated approach involving emergency physicians, surgeons, anesthesiologists, infectious disease specialists when necessary, and postoperative care teams is essential to optimize recovery and reduce the risk of recurrent ulceration and serious complications.

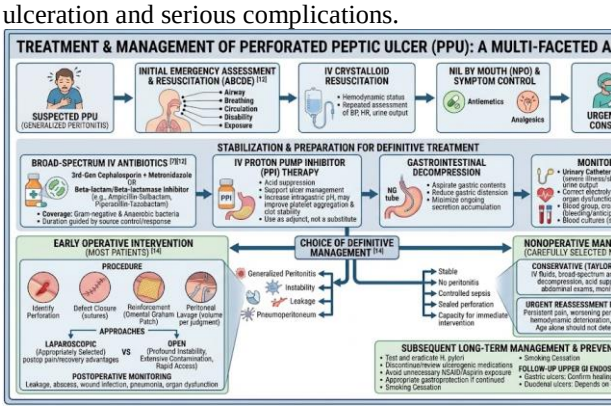


Figure-4: Treatment and management of peptic ulcer.

Differential Diagnosis

The differential diagnosis of perforated peptic ulcer includes a broad spectrum of gastrointestinal, cardiovascular, hepatobiliary, and genitourinary disorders that may present acute abdominal pain, peritoneal irritation, or hemodynamic instability. Gastrointestinal conditions include acute appendicitis, diverticulitis, cholecystitis, cholelithiasis, choledocholithiasis, gastritis, duodenitis, esophagitis, Boerhaave syndrome, small bowel obstruction, volvulus, incarcerated or strangulated hernia, and gastrointestinal neoplasms. Other potential causes include foreign body ingestion and hepatitis. Vascular emergencies, particularly abdominal aortic aneurysm rupture, aortic dissection, and mesenteric ischemia, must be considered because delayed diagnosis can result in substantial morbidity and mortality. Acute coronary syndrome may also present epigastric discomfort or upper abdominal pain, particularly in older adults. Genitourinary disorders, including nephrolithiasis, ureterolithiasis, and pyelonephritis, may produce overlapping symptoms. Accurate differentiation requires a detailed clinical history, focused physical examination, appropriate laboratory investigations, electrocardiography when indicated, and diagnostic imaging. Early identification of life-threatening alternative diagnoses is essential to ensure timely and appropriate treatment.

Prognosis

The prognosis of perforated peptic ulcer (PPU) depends on the timeliness of diagnosis, the effectiveness of initial resuscitation, the extent of peritoneal contamination, and the patient's underlying health status. Despite advances in emergency medicine, antimicrobial therapy, anesthesia, and surgical techniques, PPU remains a life-threatening condition associated with substantial mortality. Its mortality rate has been reported to be approximately ten times higher than that associated with acute appendicitis or cholecystitis. Furthermore, although gastrointestinal bleeding occurs more frequently than perforation, with an estimated ratio of approximately 6:1, mortality associated with perforated peptic ulcer is approximately five times higher than that associated with bleeding peptic ulcer. [7] The estimated 30-day mortality rate following perforation is approximately 24%, underscoring the severity of this complication and the importance of timely intervention. [6] Several patient-related and clinical factors contribute to adverse outcomes. Advanced age, particularly beyond 65 years, is associated with increased mortality because of reduced physiological reserve, a greater prevalence of comorbidities, and increased susceptibility to postoperative complications. Patients with cardiovascular disease, renal impairment, diabetes mellitus, or other chronic illnesses may have a reduced capacity to tolerate severe infection, fluid shifts, and emergency surgery. The presence of sepsis, septic shock, or

hemodynamic instability at initial presentation further worsens the prognosis because these conditions may lead to impaired tissue perfusion and multiorgan dysfunction. [6]

The interval between perforation and definitive treatment is another important determinant of survival. Delayed presentation allows continued leakage of gastrointestinal contents into the peritoneal cavity, increasing inflammation, bacterial contamination, and the risk of systemic infection. Consequently, delayed surgical source control may contribute to prolonged hospitalization, postoperative complications, and increased mortality. Early recognition, appropriate fluid resuscitation, prompt administration of broad-spectrum antibiotics, and timely surgical intervention are therefore essential to improving outcomes. Long-term prognosis also depends on successful ulcer healing and prevention of recurrence. Identification and eradication of Helicobacter pylori, avoidance of unnecessary nonsteroidal anti-inflammatory drug use, and appropriate acid-suppressive therapy can reduce the risk of recurrent ulceration. Follow-up assessment is necessary to evaluate recovery, detect persistent complications, and investigate gastric ulcers when malignancy remains a concern. Overall, early diagnosis, effective source control, and individualized management of associated comorbidities are central to reducing mortality and improving recovery among patients with perforated peptic ulcer.

Conclusion:

Perforated peptic ulcer remains a major surgical emergency requiring rapid diagnosis and timely intervention. Its development is primarily associated with Helicobacter pylori infection and nonsteroidal anti-inflammatory drug exposure, although patient-related risk factors and delayed presentation also influence disease severity. Recognizing characteristic clinical manifestations and using appropriate imaging are essential for establishing the diagnosis and determining the extent of peritoneal contamination. Management should prioritize hemodynamic stabilization, fluid resuscitation, broad-spectrum antimicrobial therapy, acid suppression, and effective source control. Surgical repair is required in most patients, while conservative treatment should be restricted to carefully selected individuals with evidence that the perforation has sealed and who can undergo close monitoring. Advanced age, shock, significant comorbidities, and delays in treatment are associated with poorer outcomes. Therefore, an integrated, multidisciplinary approach involving emergency physicians, surgeons, anesthesiologists, and critical care teams is essential. Prevention through appropriate H. pylori detection and eradication, judicious NSAID prescribing, and gastroprotection in high-risk patients may further reduce the incidence of this potentially fatal complication.

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