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Duodenal Perforation: Clinical Manifestations, Diagnostic Evaluation, Evidence-Based Management, and Nursing Care Strategies.

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Abstract

Background: Duodenal perforation is a life-threatening gastrointestinal emergency that requires prompt diagnosis and appropriate intervention. Peptic ulcer disease remains a leading cause, although trauma, foreign body ingestion, infections, malignancies, and iatrogenic injuries may also contribute. Delayed treatment can result in peritonitis, sepsis, multiple organ dysfunction, and death.

Aim: This review aims to describe etiology, epidemiology, pathophysiology, clinical manifestations, diagnostic evaluation, treatment approaches, prognosis, complications, and nursing care strategies associated with duodenal perforation.

Methods: A narrative review approach was used to organize and discuss relevant clinical evidence concerning the causes, mechanisms, diagnosis, management, and outcomes of duodenal perforation. Particular attention was given to surgical and endoscopic interventions, risk assessment, postoperative management, and nursing responsibilities.

Results: Clinical presentation commonly includes sudden severe epigastric pain, abdominal tenderness, guarding, and signs of peritonitis. Diagnosis relies on clinical assessment, laboratory investigations, and imaging, particularly computed tomography. Management depends on perforation characteristics and patient stability and may include conservative treatment in selected cases, endoscopic closure, or surgical repair. Prognosis is strongly influenced by treatment delay, advanced age, comorbidities, and preoperative shock. Major complications include sepsis, intra-abdominal abscess, wound infection, pneumonia, and enterocutaneous fistula. Effective postoperative monitoring, infection prevention, nutritional support, early mobilization, and Helicobacter pylori eradication contribute to recovery.

Conclusion: Early recognition, timely intervention, multidisciplinary coordination, and structured nursing care are essential to reduce complications and improve outcomes in patients with duodenal perforation.

Keywords: Duodenal perforation; Peptic ulcer disease; Peritonitis; Diagnosis; Surgical management; Endoscopic treatment; Nursing care; Sepsis.

Introduction

The duodenum is the first and shortest segment of the small intestine. It extends from the pylorus of the stomach to the jejunum and plays an essential role in digestion by receiving gastric contents, bile, and pancreatic secretions. Anatomically, it consists of four segments. The first segment, known as the superior portion or duodenal bulb, connects to the hepatoduodenal ligament, which contains the hepatic artery, portal vein, and common bile duct. The second or descending segment surrounds the head of the pancreas and receives the common bile duct and pancreatic duct. The third or horizontal segment passes across the abdomen, with the superior mesenteric vessels located anteriorly. The fourth or ascending segment continues into the jejunum. Duodenal perforation is a serious surgical emergency characterized by a full-thickness defect in the duodenal wall. This defect allows gastrointestinal contents to escape into surrounding tissues or the peritoneal cavity, potentially causing chemical peritonitis, bacterial contamination, sepsis, and

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multiple organ dysfunction. Although relatively uncommon, duodenal perforation is associated with substantial morbidity and mortality, with reported mortality rates ranging from 8% to 25%.[1,2,3] Its clinical significance depends on the underlying cause, extent of contamination, time to diagnosis, and effectiveness of treatment. The history of surgical management of duodenal perforation includes several important developments. Muralto described a perforated duodenal ulcer in 1688, as reported by Lenepneau.[4] In 1894, Dean documented the first successful surgical closure of a perforated duodenal ulcer.[5] Later, Cellan-Jones introduced an omental patch repair technique in 1929, which Graham subsequently modified in 1937. These techniques contributed to the development of surgical approaches that remain relevant in the management of selected perforated ulcers.[4,6]

Duodenal perforation is classified as either free or contained. Free perforation occurs when gastrointestinal contents enter the peritoneal cavity, resulting in widespread inflammation and potentially generalized peritonitis. In contrast, contained perforation occurs when adjacent structures, such as the pancreas or surrounding tissues, limit leakage and localize the inflammatory process.[4] Peptic ulcer disease is an important cause of duodenal perforation.[7] Patients with duodenal ulcers may experience recurrent epigastric pain, including nocturnal discomfort or hunger-related pain. Perforation typically produces sudden, severe upper abdominal pain, which may progress to generalized abdominal tenderness and rigidity.[8] However, clinical manifestations can be less apparent in older adults and immunocompromised patients, increasing the risk of delayed diagnosis. Early recognition is essential because prolonged leakage and peritoneal contamination can lead to serious complications. Imaging investigations, particularly abdominal computed tomography when available and clinically appropriate, play an important role in identifying perforation and assessing its extent. Prompt resuscitation, appropriate antimicrobial therapy, surgical intervention when indicated, and continuous nursing assessment are central to reducing complications and improving patient outcomes.[9]

Etiology

Duodenal perforation may result from several underlying pathological conditions, medical procedures, traumatic injuries, foreign body ingestion, or spontaneous events. Identifying the etiology is essential for selecting appropriate treatment, determining the extent of tissue damage, and preventing recurrent complications. The causes can be broadly classified into underlying duodenal pathology, iatrogenic injury, trauma, foreign body ingestion, and spontaneous perforation. Peptic ulcer disease is the most common cause of duodenal perforation. The two major contributing factors are Helicobacter pylori infection and the use of nonsteroidal anti-inflammatory drugs (NSAIDs).[7] H. pylori damages the protective mechanisms of the gastrointestinal mucosa and promotes chronic inflammation, increasing susceptibility to ulcer formation. NSAIDs interfere with prostaglandin synthesis, reducing mucosal protection and increasing the risk of ulceration. Although the incidence of peptic ulcer disease has declined in recent years, it remains the leading cause of duodenal perforation.[10] Additional risk factors for perforated peptic ulcers include cigarette smoking, physiological stress, previous peptic ulcer disease, and corticosteroid use.[11] These factors may contribute to impaired mucosal protection, delayed tissue healing, or increased susceptibility to ulcer complications. Alcohol consumption can increase gastrin secretion and damage the gastric mucosa. However, available studies have not established alcohol consumption as a direct cause of peptic ulcer disease.[12]

Other pathological conditions can also produce duodenal wall damage and subsequent perforation. Duodenal diverticula may become inflamed or develop complications that result in perforation.[13] Infectious diseases, including tuberculosis, rotavirus, norovirus, and Ascaris lumbricoides infection, have also been associated with duodenal perforation.[14,15,16] Autoimmune and inflammatory disorders, such as systemic sclerosis, Crohn disease, and abdominal polyarteritis nodosa, may contribute through chronic inflammation, vascular injury, or impaired intestinal integrity.[17,18,19] Duodenal ischemia represents another potential cause. Reduced blood supply can result in tissue hypoxia, necrosis, and loss of bowel wall integrity.[20] Other reported causes include gallstones impacted within the duodenum, which may produce local pressure and tissue injury, as well as chemotherapy-associated intestinal damage.[21,22,23] Primary or secondary duodenal tumors may also cause perforation through ulceration, tissue invasion, or necrosis.[24] Iatrogenic duodenal perforation occurs as an unintended complication of medical or surgical procedures. Its recognition has become increasingly important with the widespread use of gastrointestinal endoscopy and minimally invasive surgery.[25] Endoscopic perforations may occur during diagnostic examinations or therapeutic interventions. The risk is generally higher during therapeutic procedures because they may involve tissue resection, dilation, electrocautery, or other instrument-based interventions. Operative injury is another important iatrogenic cause. Surgical instruments may damage the duodenal wall during abdominal procedures. Laparoscopic cholecystectomy can result in duodenal perforation through thermal injury caused by electrocautery or mechanical damage during blunt or

sharp dissection. The reported incidence is approximately 0.015% of procedures.[26,27] Early recognition of these injuries is essential because delayed diagnosis may allow gastrointestinal contents to leak into the abdominal cavity.

Traumatic duodenal perforation may occur following blunt or penetrating abdominal injuries. Isolated duodenal injuries are uncommon because the organ is partly protected by surrounding anatomical structures. However, significant trauma may damage the duodenum alongside the pancreas, liver, biliary tract, or major abdominal blood vessels.[28] Clinical assessment should therefore include evaluation for associated intra-abdominal injuries. Ingestion of foreign bodies can cause direct mechanical injury to the duodenal wall. Thin, sharp objects are associated with a greater risk of penetration and perforation than blunt objects.[29] The severity of injury depends on the object's size, shape, location, and duration of retention within the gastrointestinal tract. Spontaneous duodenal perforation is a rare condition that may occur in neonates without an identifiable underlying cause.[30] It represents a distinct clinical presentation requiring prompt recognition and surgical assessment. Early diagnosis and appropriate intervention are necessary to prevent peritonitis, systemic infection, and other life-threatening complications.

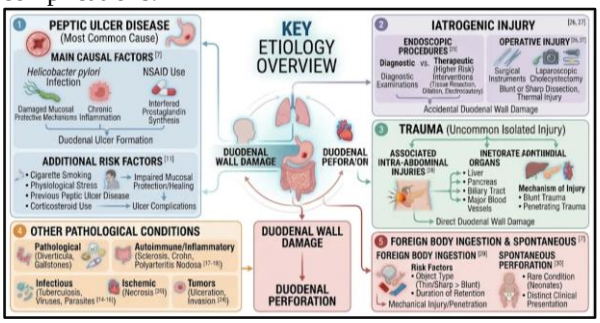


Figure-1: Etiology of Duodenal Perforation.

Epidemiology

Duodenal perforation is an uncommon but potentially life-threatening gastrointestinal emergency. Peptic ulcer disease (PUD) remains its leading cause, despite the decline in PUD incidence observed over recent decades.[10] Worldwide, PUD affects approximately 4 million people annually, with a reported incidence ranging from 1.5% to 3%.[31,32] The reduction in PUD incidence has been attributed mainly to the widespread use of proton pump inhibitors and effective eradication therapy for Helicobacter pylori infection. Nevertheless, perforated peptic ulcers continue to represent a significant clinical and surgical challenge because of their association with peritonitis, sepsis, and substantial mortality. Several factors contribute to the continued occurrence of peptic ulcer perforation. Population aging has increased the number of individuals exposed to chronic diseases and medications that affect gastrointestinal mucosal protection. The frequent use of nonsteroidal anti-inflammatory drugs (NSAIDs), particularly among older adults, represents an important risk factor. Concurrent use of NSAIDs with selective serotonin reuptake inhibitors or corticosteroids may further increase the risk of gastrointestinal complications.[3,33] H. pylori infection also remains strongly associated with perforated duodenal ulcers. Studies have reported that its prevalence ranges from 50% to 80% among patients presenting with this complication.[34] Some studies have additionally identified a higher frequency of ulcer perforation during morning hours, potentially reflecting circadian variations in gastric acid secretion.[9]

Iatrogenic duodenal perforation represents another important epidemiological category. Following endoscopic retrograde cholangiopancreatography (ERCP), the reported incidence of duodenal perforation ranges from 0.09% to 1.67%.[35,36] The risk is influenced by several patient-related and procedural factors, including older age, sphincter of Oddi dysfunction, precut sphincterotomy, anatomical abnormalities, and contrast medium injection.[37,38] Perforation or leakage following endoscopic interventions also varies according to the procedure. Reported leakage rates after endoscopic suturing of duodenal ulcers range from 1% to 13%, while rates following endoscopic mucosal resection range from 6% to 13%.[39] Traumatic duodenal injuries are relatively uncommon because of the organ's protected anatomical position. Fewer than 2% of traumatic abdominal injuries involve the duodenum, although these injuries may occur alongside damage to other abdominal organs.[40] Foreign body-related gastrointestinal perforation is also rare, occurring in fewer than 1% of ingested foreign bodies. Sharp and elongated objects account for a greater proportion of these complications.[41] The epidemiological characteristics of duodenal perforation demonstrate the importance of recognizing medication-related risks, persistent H. pylori infection, procedural complications, and traumatic injuries. Understanding these patterns can support preventive strategies, early diagnosis, and timely surgical management.

Pathophysiology

Duodenal perforation develops when the integrity of the duodenal wall is disrupted, resulting in communication between the intestinal lumen and surrounding tissues or the peritoneal cavity. The underlying mechanisms vary according to the causative condition, but they commonly involve mucosal injury, inflammation, tissue necrosis, impaired healing, or direct mechanical damage. Peptic ulcer disease is the leading pathological cause of duodenal perforation, with nonsteroidal anti-inflammatory drug (NSAID) use and Helicobacter pylori infection representing the two major contributing factors.[10] NSAIDs contribute to gastroduodenal mucosal injury by inhibiting

cyclooxygenase enzymes, particularly cyclooxygenase-1 (COX-1), which reduces prostaglandin synthesis. Prostaglandins normally maintain gastrointestinal mucosal protection by supporting mucus and bicarbonate secretion, preserving mucosal blood flow, and promoting epithelial repair. Their reduction weakens the protective barrier against gastric acid and digestive enzymes, increasing susceptibility to inflammation, ulceration, and subsequent perforation. The risk of NSAID-associated mucosal injury is greater among individuals older than 65 years and those with heart disease, previous peptic ulcer disease, or concurrent use of antiplatelet medications, corticosteroids, or anticoagulants.[42] Prolonged exposure and high NSAID doses further increase this risk. Piroxicam and ketorolac have been associated with substantial gastroduodenal mucosal injury among NSAIDs.[43]

H. pylori infection contributes to duodenal ulcer formation through several mechanisms. The organism induces chronic gastric inflammation and alters normal gastric acid regulation. It may promote gastric metaplasia within the duodenal mucosa, allowing bacterial colonization and subsequent local inflammatory injury. Increased gastric acid secretion, immune-mediated tissue damage, and reduced mucosal defense mechanisms contribute to ulcer development and progression. When tissue destruction extends through the entire thickness of the duodenal wall, perforation may occur.[44] Iatrogenic perforation involves direct mechanical or thermal injury during diagnostic or therapeutic procedures. Following endoscopic retrograde cholangiopancreatography (ERCP), the Stapfer classification identifies four types of duodenal perforation. Type I involves the lateral or medial duodenal wall and usually results from direct endoscope trauma. Type II refers to periampullary perforation associated with sphincterotomy. Type III involves the distal bile duct and generally results from guidewire or basket instrumentation. Type IV is characterized by retroperitoneal air detected on imaging and is usually asymptomatic.[45] Management depends on the injury type, clinical condition, and evidence of leakage or infection. Selected type III and IV injuries can be managed conservatively, while other cases may require surgical or endoscopic intervention.[46]

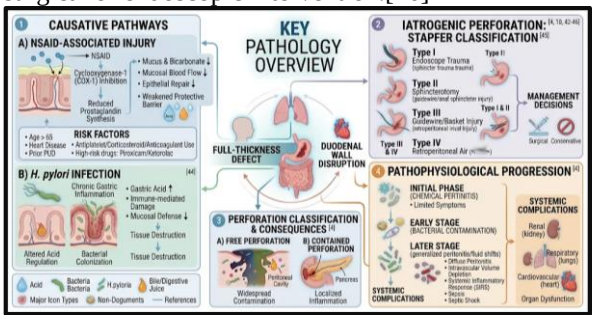


Figure-2: Pathophysiology of duodenal perforation.

Duodenal perforations are further classified as free or contained. Free perforation occurs when a full-thickness defect allows gastrointestinal contents to escape directly into the peritoneal cavity. This produces widespread contamination and diffuse peritonitis. In contained perforation, adjacent structures, including the pancreas and surrounding tissues, restrict the leakage and localize inflammation around the defect.[4] The pathophysiological progression of perforation involves several stages. Initially, gastric acid and digestive secretions enter the peritoneal cavity, causing chemical peritonitis and intense local inflammation. During the early phase, clinical manifestations may be limited or absent, which can delay recognition. If the defect remains open, intestinal contents, food particles, and microorganisms enter the abdominal cavity, resulting in bacterial contamination. The inflammatory response subsequently becomes more extensive and may lead to generalized peritonitis, fluid shifts, intravascular volume depletion, systemic inflammatory response, sepsis, and septic shock.[4] Progressive inflammation can impair intestinal function and reduce tissue perfusion. In severe cases, inadequate circulation contributes to organ dysfunction, including renal, respiratory, and cardiovascular complications. The severity of these effects depends on the size and location of the perforation, the amount of contamination, the patient's physiological reserve, and the time elapsed before treatment. Understanding these mechanisms is essential for nurses and other healthcare professionals because early recognition of deterioration, prompt resuscitation, antimicrobial therapy, and timely surgical management can reduce complications and improve patient outcomes.

Evaluation

The evaluation of duodenal perforation requires rapid clinical assessment, laboratory investigations, and appropriate imaging studies. The primary objectives are to confirm the presence of perforation, identify its location and extent, assess the degree of peritoneal contamination, and evaluate the patient's physiological condition. Early diagnosis is essential because delayed treatment increases the risk of generalized peritonitis, sepsis, septic shock, and multiple organ dysfunction. The selection of investigations depends on the patient's clinical stability, suspected underlying cause, and availability of diagnostic resources. Laboratory investigations provide important information about inflammation, infection, metabolic disturbances, and organ function. Serum amylase levels may be elevated in patients with perforated peptic ulcers, although the increase may remain below four times the upper limit of the reference range. This finding can support clinical assessment but is not specific to duodenal perforation and should be interpreted alongside other investigations.[47] Serum gastrin measurement may

be indicated in patients with recurrent or multiple peptic ulcers when Zollinger-Ellison syndrome is suspected. This condition is associated with excessive gastrin secretion and increased gastric acid production.[12]

A complete blood count may demonstrate leukocytosis, reflecting an inflammatory or infectious response. Elevated C-reactive protein levels also indicate inflammation and may help assess disease severity and response to treatment.[48] Blood cultures should be collected before initiating antimicrobial therapy whenever possible, particularly in patients with suspected sepsis. However, obtaining cultures should not delay the administration of antibiotics in critically ill patients.[49] Arterial blood gas analysis helps evaluate acid-base disturbances, oxygenation, and metabolic compromise. Metabolic acidosis and elevated lactate levels may indicate tissue hypoperfusion and severe systemic illness.[9] Additional investigations, including renal function tests, electrolytes, liver function tests, coagulation studies, and serum lactate, may help assess the patient's overall condition and guide resuscitation. Imaging plays a central role in confirming duodenal perforation. Urgent upright chest radiography is an important initial investigation in patients presenting with acute upper abdominal pain and suspected perforation. It may demonstrate free intraperitoneal air beneath the diaphragm, a finding identified in approximately 75% of patients.[50] The presence of subdiaphragmatic free air in a patient with compatible abdominal symptoms strongly supports the diagnosis of a perforated peptic ulcer. Nevertheless, a normal radiograph does not exclude perforation, particularly when imaging is performed shortly after symptom onset or when the amount of leaked air is small.

Abdominal radiography may demonstrate additional findings associated with pneumoperitoneum. The Rigler sign refers to visualization of both sides of the intestinal wall because of gas within the bowel lumen and the peritoneal cavity. The football sign describes a large, oval collection of intraperitoneal gas. Free air may also outline anatomical structures, including the falciform ligament and liver margin.[51] Computed tomography (CT) is the preferred imaging investigation for stable patients when clinical suspicion remains despite normal or inconclusive radiographic findings.[4] Its diagnostic accuracy for detecting perforated peptic ulcers is approximately 98%.[52] CT performed with oral and intravenous contrast provides detailed information about the perforation site and surrounding tissues.[53] Important findings include duodenal wall thickening, extraluminal air, leakage of oral contrast, inflammatory changes in surrounding fat, and localized periduodenal fluid collections. Noncontrast CT may be useful when intravenous contrast is contraindicated, including in selected patients with acute kidney injury. It can demonstrate free intraperitoneal air and other findings suggestive of perforation. Oral diatrizoate may also help identify leakage when CT is available but upright chest radiography is inconclusive. However, the absence of contrast extravasation does not exclude perforation because the defect may have sealed spontaneously.[54] Nurses contribute significantly to diagnostic evaluation by monitoring vital signs, assessing abdominal pain and tenderness, documenting changes in consciousness, and observing for signs of shock. Accurate documentation of laboratory results, fluid balance, and clinical deterioration supports timely medical decisions and appropriate surgical intervention.

Treatment / Management

The management of duodenal perforation depends on the location, size, and severity of the defect, the extent of peritoneal contamination, and the patient's general clinical condition. Perforations are classified as contained or noncontained. Noncontained perforations are further categorized as minor or major according to the extent of tissue damage and the complexity of the required intervention. The primary objectives are to control leakage, prevent infection, restore gastrointestinal integrity, and maintain physiological stability.

Contained Perforations

Contained perforations occur when adjacent structures, such as the pancreas and surrounding tissues, restrict the leakage of gastrointestinal contents into the peritoneal cavity. Selected patients may be managed conservatively when imaging confirms that the perforation is sealed and there is no ongoing leakage. A water-soluble contrast study using diatrizoate may be performed before initiating conservative management to confirm the absence of extravasation. Conservative treatment includes intravenous fluid replacement, keeping the patient nil per os (NPO), intravenous proton pump inhibitors, and broad-spectrum antibiotics to control infection and prevent bacterial complications. H. pylori eradication therapy should be provided when infection is identified or strongly suspected. Patients require repeated clinical examinations, laboratory investigations, and monitoring for signs of deterioration.[4] Somatostatin may also be considered in selected cases because studies suggest that it can facilitate enterocutaneous fistula closure.[55] Reported mortality rates among patients with contained perforations were 3% with conservative treatment and 6.2% with surgical treatment. However, treatment selection should depend on individual clinical circumstances rather than these figures alone.[56] Conservative management follows the six Rs: repeated clinical examination, radiological confirmation of no leakage, repeated blood investigations, adequate resources for monitoring, respiratory and renal support when required, and

readiness to proceed with surgery if the patient's condition deteriorates.[12]

Noncontained Perforations

Noncontained perforations allow gastrointestinal contents to enter the abdominal cavity, increasing the risk of generalized peritonitis and sepsis. These injuries require prompt assessment and intervention. Treatment depends on whether the defect is minor or major, with endoscopic procedures and surgical repair representing the principal approaches.[4] Minor noncontained perforations may be managed using endoscopic closure or simple surgical repair. Endoscopic techniques include through-the-scope clips, over-the-scope clips, detachable snare loops combined with clips, and self-expandable metal stents. Through-the-scope clips are generally suitable for linear defects smaller than 1 cm, whereas over-the-scope clips, detachable snare loops, and self-expandable metal stents may be used for defects measuring approximately 1 to 3 cm.[57] Surgical repair may involve direct closure, with or without an omental patch. A free omental plug, known as the Graham patch, or a pedicled omental flap, known as the Cellan-Jones repair, may be sutured over the perforation to reinforce closure.[4,6] Laparoscopic Graham patch repair has been associated with lower morbidity and mortality than open repair in a meta-analysis, although selection bias may have influenced the findings.[58] Studies have not demonstrated a consistent benefit from routine drain placement following surgical repair.[59] Endoscopic vacuum therapy may be considered when perforation produces an associated cavity. This technique involves placing a sponge attached to a nasogastric tube within the defect under endoscopic guidance. Continuous negative pressure helps drain gastrointestinal contents, reduce cavity contamination, and promote granulation tissue formation. The system is generally replaced every 3 to 5 days until closure is achieved.[60] Major noncontained perforations usually require complex reconstructive surgery because extensive tissue damage may prevent simple closure. Surgical options include duodenoduodenostomy as the preferred reconstructive approach, Roux-en-Y duodenojejunostomy as an alternative, and Billroth II reconstruction when necessary.[40] Perforations involving the first or proximal second portion of the duodenum may require Billroth II reconstruction, depending on the extent and location of tissue damage.[61] Throughout treatment, nurses play an essential role in monitoring vital signs, assessing abdominal pain, maintaining fluid balance, administering prescribed medications, and observing for postoperative complications. Early identification of sepsis, bleeding, anastomotic leakage, and respiratory or renal deterioration supports timely intervention and improves patient outcomes.

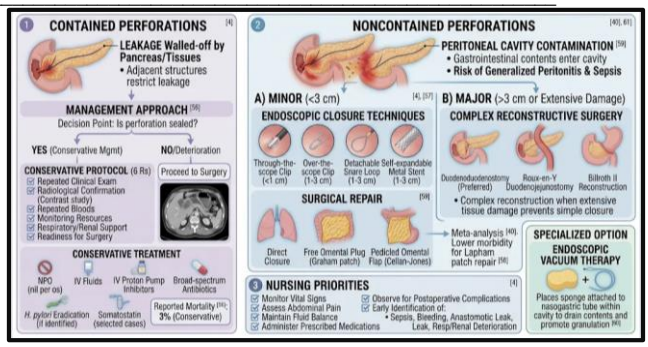


Figure-3: Treatment and Management of duodenal perforation.

Differential Diagnosis:

The differential diagnosis of duodenal perforation includes several gastrointestinal, cardiovascular, hepatobiliary, respiratory, and other abdominal conditions that may present with acute epigastric or generalized abdominal pain. Accurate differentiation is essential because some of these conditions require immediate intervention, and delays in diagnosis can increase morbidity and mortality. Gastrointestinal conditions include acute pancreatitis, gastritis, duodenitis, esophagitis, gastroesophageal reflux disease, cholecystitis, cholelithiasis, hepatitis, diverticulitis, appendicitis, and bowel obstruction. Pancreatitis may produce severe epigastric pain radiating to the back, accompanied by nausea and vomiting. Cholecystitis and cholelithiasis commonly cause right upper quadrant or epigastric pain, sometimes following meals. Appendicitis may initially present with periumbilical or epigastric discomfort before localizing to the right lower quadrant. Small bowel obstruction and volvulus may cause abdominal distension, vomiting, and altered bowel movements. Mesenteric ischemia should also be considered, particularly when severe abdominal pain occurs without proportionate findings on physical examination. Other important diagnoses include Boerhaave syndrome, which results from esophageal rupture and may cause severe chest or upper abdominal pain following forceful vomiting. Foreign body ingestion can produce gastrointestinal injury or perforation. Ventral hernia may cause localized pain and, when incarcerated or strangulated, symptoms of intestinal obstruction. Cardiovascular emergencies must not be overlooked. Abdominal aortic aneurysm rupture, aortic dissection, and acute coronary syndrome can present with epigastric pain and may initially resemble an acute abdominal condition. Pneumonia, particularly involving the lower lung lobes, can also produce referred upper abdominal pain. Clinical assessment should consider pain characteristics, associated symptoms, vital signs, abdominal examination findings, and relevant laboratory investigations. Imaging, particularly computed tomography when indicated, helps distinguish duodenal perforation from these conditions and supports timely treatment.

Prognosis

The prognosis of duodenal perforation depends on several clinical factors, including the patient's age, general health, severity of peritoneal contamination, presence of sepsis, and time elapsed between perforation and initiation of treatment. Reported mortality rates among patients with perforated peptic ulcers range from 1.3% to 20%.[62][63][64] Other studies have reported a 30-day mortality rate of up to 20%, reflecting the serious nature of this condition and the potential for rapid clinical deterioration.[33] One of the most important determinants of survival is the interval between perforation and definitive treatment. Delays exceeding 24 hours increase the risk of mortality because prolonged leakage of gastrointestinal contents promotes peritoneal inflammation, bacterial contamination, sepsis, and organ dysfunction.[4] Several scoring systems help clinicians estimate the risk of postoperative complications and death. The American Society of Anesthesiologists (ASA) physical status classification and the Boey score are among the most commonly validated tools for predicting outcomes in patients with duodenal perforation.[12] The Boey score considers three major risk factors: severe concomitant medical illness, preoperative shock, and a perforation duration exceeding 24 hours. Each factor contributes one point. Patients with all three factors have a score of 3, which has been associated with a mortality rate of 38% and a morbidity rate of 77%.[65] The ASA classification evaluates the severity of systemic disease and the presence of comorbidities to estimate perioperative risk.[65] Other factors associated with increased mortality include advanced age, shock at admission, metabolic acidosis, acute kidney injury, hypoalbuminemia, smoking, underweight status, female sex, and the need for resection procedures.[66][67] Patients older than 65 years generally experience higher mortality than younger patients, partly because of reduced physiological reserve and the greater prevalence of chronic diseases.[66] Postoperative mortality is estimated at approximately 6% to 10% among patients with perforated peptic ulcers.[68] Treatment delays exceeding 24 hours, age above 60 years, associated medical conditions, and systolic blood pressure below 100 mm Hg are particularly important predictors of poor outcomes.[69] Early diagnosis, prompt surgical or appropriate endoscopic intervention, effective antimicrobial therapy, and careful postoperative monitoring can improve survival and reduce morbidity.

Complications

Duodenal perforation can produce serious complications because gastrointestinal contents, including gastric acid, bile, pancreatic secretions, and intestinal bacteria, may enter the peritoneal cavity. The resulting inflammation can progress from chemical peritonitis to bacterial peritonitis, systemic infection, and multiple organ dysfunction. Sepsis represents one of the most important causes of death in patients with perforated peptic ulcers and accounts for approximately 40% to 50% of fatalities. At the time of arrival in the operating room, approximately 30% to 35% of patients may already have sepsis. Furthermore, more than 25% of patients may develop septic shock within the first postoperative month. Septic shock is associated with a mortality rate of approximately 50% to 60%.[9] Postoperative complications occur in approximately 30% of patients undergoing treatment for perforated peptic ulcers.[70] Their frequency and severity are influenced by patient characteristics, the extent of contamination, perforation size, and the timing of intervention. Age above 40 years, a history of shock, and a larger perforation are recognized risk factors for increased postoperative morbidity.[71] Common complications include pneumonia, wound dehiscence, persistent peritonitis, incisional hernia, enterocutaneous fistula, intra-abdominal fluid collection, abscess formation, surgical site infection, and postoperative ileus. Surgical site infection is among the most frequently reported complications and has been documented in approximately 32% of patients in some studies.[71] Intra-abdominal abscesses may develop when infected fluid or gastrointestinal contents remain within the abdominal cavity after repair. These collections can cause persistent fever, abdominal pain, leukocytosis, and delayed recovery. Enterocutaneous fistula may occur when an abnormal communication develops between the intestinal lumen and the skin, resulting in leakage of intestinal contents and potential fluid and electrolyte disturbances. Respiratory complications, particularly pneumonia, may arise because of postoperative pain, reduced mobility, impaired coughing, and prolonged hospitalization. Wound dehiscence and incisional hernia may compromise surgical recovery and sometimes require further intervention. Ileus can delay the return of normal gastrointestinal function and prolong hospital stay. Endoscopic vacuum therapy may also present specific technical challenges. These include difficulty accessing the perforation, the need for specialized equipment and trained personnel, and displacement of the vacuum device because of intestinal motility.[72] Early recognition of complications through clinical assessment, laboratory investigations, and appropriate imaging is essential. Nursing surveillance plays an important role in identifying deterioration and facilitating timely medical intervention.

Postoperative and Rehabilitation Care

Postoperative care following duodenal perforation focuses on maintaining physiological stability, preventing infection, supporting gastrointestinal recovery, and reducing the risk of recurrent ulceration. Management should begin immediately after the procedure and continue

throughout hospitalization and subsequent follow-up. A nonrandomized study demonstrated that implementing a structured treatment protocol from admission through the first three postoperative days following laparoscopic repair reduced 30-day mortality from 27% to 17%. The protocol emphasized minimizing surgical delays and preventing, identifying, and treating sepsis.[9] These findings highlight the importance of coordinated perioperative management and continuous monitoring. During the initial postoperative period, healthcare professionals should monitor vital signs, oxygen saturation, urine output, fluid balance, abdominal findings, pain, and mental status. Particular attention should be given to hypotension, tachycardia, fever, reduced urine output, increasing abdominal pain, and altered consciousness, as these findings may indicate bleeding, persistent leakage, infection, or sepsis. Laboratory investigations, including complete blood count, renal function, electrolytes, inflammatory markers, and acid-base assessment when indicated, help evaluate recovery and detect complications. Intravenous fluids, antimicrobial therapy, analgesics, and proton pump inhibitors should be administered according to the prescribed treatment plan. Gastrointestinal recovery is another important component of postoperative care. Patients may initially require bowel rest and intravenous nutritional support depending on the severity of the perforation and the type of repair. However, prolonged fasting and unnecessary use of tubes may delay recovery. Small randomized clinical trials conducted in Türkiye found that early removal of tubes and initiation of oral intake reduced hospital stay by approximately three days among patients with ASA physical status scores of I or II.[73] Decisions regarding oral intake and tube removal should therefore consider the patient's clinical stability, gastrointestinal function, and evidence of successful repair. Helicobacter pylori eradication is essential when infection is identified because persistent infection increases the risk of recurrent peptic ulceration. A meta-analysis by Wong et al. demonstrated that eradication therapy significantly reduced recurrent ulcer incidence at both eight weeks and one year among patients who underwent surgical treatment for perforated duodenal ulcers.[74] Patients should also receive appropriate counseling regarding medication adherence, avoidance of unnecessary nonsteroidal anti-inflammatory drugs, smoking cessation, and recognition of recurrent ulcer symptoms. Rehabilitation includes gradual mobilization, respiratory exercises, nutritional support, and restoration of daily activities. Nurses should encourage appropriate movement, assist with coughing and deep-breathing exercises, assess wound healing, and provide education about discharge instructions. Follow-up endoscopy is generally not recommended after duodenal ulcer perforation because these patients have a low risk of malignant neoplasms.[9] Follow-up should instead focus on symptom resolution, treatment adherence, confirmation of H. pylori eradication when appropriate, and prevention of recurrent ulcer disease.

Conclusion:

Duodenal perforation is a serious surgical emergency associated with substantial morbidity and mortality, particularly when diagnosis and treatment are delayed. Peptic ulcer disease remains its principal cause, although several traumatic, infectious, inflammatory, neoplastic, and iatrogenic conditions may also produce perforation. Accurate diagnosis requires careful clinical assessment supported by laboratory investigations and appropriate imaging. Treatment should be selected according to the patient's clinical condition, perforation size, and extent of contamination. Surgical repair remains essential in many cases, while endoscopic and conservative approaches may be appropriate for selected patients. Early intervention, infection control, Helicobacter pylori eradication, and effective postoperative monitoring improve patient outcomes. Nursing care is central to detecting complications, maintaining physiological stability, supporting recovery, and educating patients about preventive measures. A coordinated multidisciplinary approach is therefore necessary to reduce mortality, prevent recurrence, and improve quality of care.

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