



REVIEW OF THE CURRENT LITERATURE ON DUODENAL PERFORATIONS AND DISCUSSION ON THE OUTCOMES OF DIFFERENT TREATMENT STRATEGIES

General Surgery

Dr. Chandra Mauli Upadhyay

Associate Professor, department Of General Surgery, JLNMCH Bhagalpur ,bihar

Dr. Sanjeeda Ali*

1st Year PG , Department Of General Surgery, JLNMCH Bhagalpur ,Bihar. *Corresponding Author

ABSTRACT

Duodenal perforation is one of the common cause of acute abdomen coming in our Emergency OPD. Though the incidence of Peptic Ulcer Disease has diminished due to uses of anti ulcer drugs like PPI and H2 Blockers and eradication treatment for *Helicobacter pylori*. Still the cases of duodenal perforation are not diminishing in number. Duodenal perforation is a potentially life-threatening condition. Duodenal perforations can either be free or contained. Free perforation arises when bowel contents leak freely into the abdominal cavity and causing diffuse peritonitis. Contained perforation occurs when the ulcer creates a full-thickness hole, but free leakage is prevented by contiguous organs such as the pancreas wall off the area. Multiple etiologies are associated with duodenal perforations such as peptic ulcer disease, iatrogenic causes and trauma. The leading causes of perforated peptic ulcers remain NSAIDs use and *H. pylori*. The Iatrogenic cause of Duodenal perforation is ERCP. PUD can be treated with medications. However, PUD may perforate and lead to high mortality risk. The classic triad for PPU(perforated peptic ulcer) is the sudden onset of abdominal pain, abdominal rigidity, and tachycardia. we should be aware that normal upright chest X-ray does not rule out duodenal perforation. only in 65% cases we get free gas in peritoneal cavity. Computed tomography with intravenous and oral contrast is the most valuable imaging technique to identify duodenal perforation. In some cases, surgical exploration may be necessary for diagnosis. Early diagnosis, quick resuscitation, and urgent surgical intervention are crucial to improving outcomes. Specific treatment depends upon the nature of the disease process that caused the perforation, the timing, location and extent of the injury and the clinical condition of the patient. Conservative management seems to be feasible in stable patients with sealed perforations. Immediate surgery is required for patients presenting with peritonitis and/or intra-abdominal sepsis. Minimally invasive techniques are safe and effective alternatives to conventional open surgery in selected patients with duodenal perforations. The gold standard treatment is still exploratory laparotomy and omental patch repair. Once the patients are being discharged they should receive education from the medical care team to change their lifestyles, which includes quitting smoking and avoiding the consumption of NSAIDs as much as possible. Patients with positive *H. pylori* should take medication to eradicate the infection. Here we review the current literature on duodenal perforations and discuss the outcomes of different treatment strategies.

KEYWORDS

INTRODUCTION

Duodenal perforation represents a rare but potentially lifethreatening condition. The mortality rate ranges from 8% to 25% in published studies. The first description of a perforated duodenal ulcer was made in 1688 by Muralto and reported by Lenepneau. In 1894, Dean reported the first successful surgical closure of a perforated duodenal ulcer. Surgery is still the mainstay of treatment for duodenal perforation. Many perforations are repaired using an omental patch, a technique that was first described by Cellan-Jones in 1929 and was later modified by Graham in 1937. The first laparoscopic repair for a perforated duodenal ulcer was reported in 1990.

The incidence of peptic ulcer disease has decreased in recent years. This can partly be explained by the use of proton pump inhibitors (PPIs) and eradication treatment for *Helicobacter pylori*. However, peptic ulcer complications, including perforation, still remain a substantial healthcare problem. This may be related to increased use of non-steroidal anti-inflammatory drugs (NSAIDs) and to the aging population. Furthermore, iatrogenic duodenal perforations are becoming more common following the widespread use of endoscopic procedures, such as endoscopic retrograde cholangiopancreatography (ERCP).

Typically, patients with duodenal ulcers have nocturnal abdominal pain or feel hungry. If perforation occurs, it usually can cause sudden onset of severe pain in the upper abdomen. However, in immunocompromised or elderly patients, the clinical signs can be undetectable and lead to delay diagnosis.

Imaging has an essential role in diagnosis, and subsequently, for early resuscitation. Appropriate selection of therapeutic alternatives and risk-assessment can decrease the risk of morbidity and mortality.

Optimal methods for the management of duodenal perforations remain controversial. The diagnosis is often delayed leading to decreased survival. There are few randomized controlled studies and management strategies often rely on data from observational studies, or even case reports. One area of controversy includes the role of non-operative management. In patients that need surgery, there is still ongoing debate regarding type of repair, open or laparoscopic

technique and the role of gastric diversion procedures, such as pyloric exclusion.

In this review, we provide an overview of duodenal perforations and potential management strategies based on available data.

Etiology

Underlying duodenal pathology

1) Peptic ulcer disease (PUD): *H. pylori* infection and non-steroidal anti-inflammatory drugs (NSAIDs) are two major causes for PUD and subsequently duodenal perforation. Although the incidence of PUD has decreased in recent years, it is still the main cause of duodenal perforation.

Smoking, physiological stress, previous history of PUD, and corticosteroids are other risk factors for a perforated peptic ulcer (PPU).

Alcohol consumption is known to cause to increase gastrin secretion and damage gastric mucosa. However, studies did not show that alcohol can cause PUD.

- 2) Duodenal diverticula
- 3) Infectious disease (TB, rotavirus, norovirus, *Ascaris lumbricoides*)
- 4) Autoimmune conditions, including scleroderma, Crohn disease, and abdominal polyarteritis nodosa.
- 5) Duodenal ischemia
- 6) Impacted gallstones in the duodenum
- 7) Chemotherapy
- 8) Tumors

Iatrogenic: Due to the widespread use of endoscopic procedures, it is becoming more common. The incidence of endoscopic perforations is higher for therapeutic procedures. The rate of duodenal perforations after ERCP ranges from 0.09 to 1.67%. The Stapfer classification has been developed to categorize ERCP-related perforations. Type I perforations are large lateral or medial duodenal wall perforations usually caused by the endoscope itself. Type II perforations, also known as peri-Vaterian injuries, are related to the sphincterotomy. Type III perforations represent distal bile duct injuries caused by wire

or basket instrumentation, while type IV perforations represent retroperitoneal air alone on imaging and are often asymptomatic. Risk factors for ERCP-related perforations have been reported to include old age, sphincter of Oddi dysfunction, precut, intramural injection of contrast medium and anatomical abnormalities, such as Billroth II gastrectomy.

1) Endoscopic perforations: It includes diagnostic and therapeutic procedures. However, the incidence of perforation is higher in therapeutic procedures.

2) Operative injury: Surgical instruments can cause duodenal perforation. Laparoscopic cholecystectomy can perforate the duodenum (0.015%) during thermal burns by electrocautery or blunt or sharp dissection.

Trauma: Isolated duodenal injuries are uncommon and usually occur with other organ injuries. Less than 2% of traumatic abdominal injuries involves the duodenum.

Foreign bodies: Thin and sharp foreign bodies correlations with higher perforation risk.

Spontaneous perforations: It occurs in neonates with the unknown underlying causes.

Pathophysiology

Duodenal perforation can be either free or contained. Free perforation arises when bowel contents leak freely into the abdominal cavity, causing diffuse peritonitis. Contained perforation occurs when the ulcer creates a full-thickness hole, but free leakage is prevented by contiguous organs such as the pancreas wall off the area. In duodenal perforation, initially, gastric acid juice leaks into the peritoneal cavity, which leads to profound chemical peritonitis. During this period the patient develops severe pain abdomen. It remains for about 6 hours. It is called phase of chemical peritonitis. Due to peritoneal reaction a large amount of fluid collects in peritoneal cavity. It dilutes the chemical and pain in abdomen diminishes. Patient gets relief for some times. It is called phase of illusion. After few hours bacterial peritonitis develops leading to abdominal symptoms.

History and Physical examination

Typical symptoms for a perforated peptic ulcer (PPU) are defined by sudden onset of abdominal pain, which never completely subsides even with premedicated remedies. The classic triad in PPU patients is tachycardia, sudden onset of abdominal pain, and abdominal rigidity. Abdominal tenderness with rigidity and tachycardia are common clinical signs.

Three phases define the clinical manifestations of PPU. Within two hours of onset (initial phase), tachycardia, epigastric pain, and cold extremities are characteristic. Within 2 to 12 hours (second phase), pain becomes generalized and increases with movement. Fluid moving along the right paracolic gutter can lead to typical signs such as right lower quadrant tenderness and abdominal rigidity. More than 12 hours (third phase), can manifest as abdominal distension, fever, and hypotension. Patients with retroperitoneal perforation present more indolently without peritoneal signs.

Diagnosis

Laboratory tests: Usually used to rule out differential diagnoses.

Raising in serum amylase less than four times than normal levels can be associated with PPU.

Serum gastrin levels are useful in patients who have a history of recurrent ulcers to establish the diagnosis of Zollinger Ellison syndrome.

Leukocytosis and high C-reactive protein level indicate existing of inflammation or infection. Blood cultures are necessary before starting antibiotics.

An arterial blood gas measures the level of metabolic compromise in septic patients.

X-ray chest erect posture is advised to see for free gas under right dome of diaphragm. It is present in 65% cases.

CT Scan of abdomen-- Double-contrast computed tomography (CT) scan is the most valuable method for diagnosing duodenal perforation.

It should be performed whenever there is a clinical suspicion and the patient does not need immediate surgery. CT features of perforation include discontinuity of the duodenal wall and the presence of extraluminal air or extravasated oral contrast. Other CT findings include duodenal wall thickening, fat stranding and periduodenal fluid collection.

Treatment

Management of duodenal perforations includes conservative, endoscopic and surgical strategies. The main goals of treatment are resuscitation, control of infection, nutritional support and restoration of gastrointestinal tract continuity.

Management of duodenal perforations depends on the type of perforation.

In Contained perforations Conservative management is done. Conservative management consists of intravenous fluid therapy, nil per oral (NPO), intravenous proton pump inhibitors (PPIs), broad-spectrum antibiotics, H. pylori eradication, and repeated clinical assessment. Non-operative management of perforated duodenal ulcers is feasible in selected patients. Perforated ulcers may seal spontaneously with fibrin, omentum or by fusion of the duodenum to the underside of the liver between the gallbladder and the falciform ligament. Approximately, 50–70% of patients with perforated peptic ulcers respond to conservative treatment without surgery. For patients undergoing conservative treatment, a gastroduodenogram may be performed soon after admission to investigate if there is any contrast extravasation. Conservative management seems to be safe if the gastroduodenogram shows self-sealing. Operative management is usually recommended if there is free leakage of contrast medium into the peritoneal cavity. Progressive abdominal signs or intra-abdominal sepsis should warrant surgery.

In high-risk patients, who cannot tolerate surgical treatment, conservative management may also include percutaneous drainage of fluid collections. The essential components of conservative management of duodenal perforation are gatherable as "R"s: (1) Repeated clinical examination; (2) Radiologically undetected leak; (3) Repeated blood investigations; (4) Resources for monitoring; (5) Respiratory and renal support; and (6) Readiness to operate. In contained perforation patients, studies reported the mortality rate with conservative management was 3% as opposed to 6.2% with surgical management.

Minor non contained perforation : Endoscopic or simple surgical repair are two primary management strategies for this group. Endoscopic management includes through-the-scope clips (TTSC), over-the-scope clips (OTSC), detachable snare loops with clips, and self-expandable metal stents (SEMS). TTSC management is favorable for linear perforations less than 1 cm while in perforation with a range from 1 to 3 cm OTSC, detachable snare loop with clips, and SEMS are suitable management options. Simple surgical treatment is another option for the management of minor non-contained perforation. The surgeon can perform it with or without an omental patch. Alternatively, a free omental plug (Graham patch) or a pedicled omental flap, which is known as a Cellan-Jones repair, can be sutured into the perforation. Studies did not show any benefit for the placement of drain after surgical repair.

Sutureless techniques have also been developed using a gelatin sponge and fibrin glue to seal off the perforation. There seem to be no significant differences in terms of postoperative morbidity and mortality rates when comparing primary closure, omentopexy or tegmentation (without closure). Surgical repair can be performed either with conventional open surgery or with laparoscopy. The results of a recent meta-analysis including seven randomized controlled trials showed a significant benefit for the laparoscopic approach for the treatment of perforated peptic ulcer disease with a significant reduction in postoperative complications and hospital stay.

B) Major non-contained perforations: These types of perforations usually need reconstructive surgery, which includes duodenoduodenostomy (first option), Roux-en-Y duodenojejunostomy (second option), and Billroth II operation. Perforation in the first or proximal second portion requires management by the Billroth II operation.

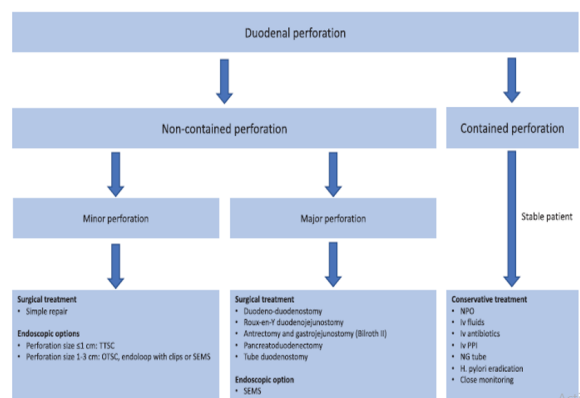


Figure 1. A general management algorithm for duodenal perforations. Abbreviations: NG: nasogastric; NPO: nil per os; OTSC: over-the-scope clip; PPI: proton pump inhibitor; SEMS: self-expandable metal stent; TTSC: through-the-scope clip.

Prognosis

Mortality rate in perforation peptic ulcer (PPU) patients reported with ranging from 1.3% to 20%. Other studies have reported 30 days mortality rate reaching 20%. The main prognostic factor is the time interval between the duodenal perforation and treatment. The interval time greater than 24 hours increases mortality. The American Society of Anesthesiologists (ASA) score and Boey score are the most common validated scoring systems to predict outcomes in duodenal perforation. Boey score factors include concomitant severe medical illness, preoperative shock, and the duration of perforation over 24 hours. When all these factors are positive, the total score count is 3, which predicts the mortality as 38% and morbidity as 77%. The American Society of Anesthesiologists (ASA) score uses the present systemic disease and degree of comorbidity as parameters to predict the outcome of PPU. Significant risk factors that increase the mortality rate are co-morbidities, resection surgery, presence of shock at admission, female, elderly patients, metabolic acidosis, a delay presentation of more than 24 hours, acute renal failure, hypoalbuminemia, smokers, and being underweight. Studies show the mortality rate is higher in patients older than 65-year-old compared to younger patients. The postoperative mortality rate in patients with PPU is estimated to be 6% to 10%. Delays in treatment greater than 24 hours, age more than 60 years old, concomitant diseases, and systolic blood pressure less than 100 mmHg are the main risk factors that increase the mortality rate.

CONCLUSION

Duodenal perforation is caused by a variety of different

mechanisms. Some duodenal perforations can be managed

conservatively, while others require prompt surgical treatment.

The type of treatment should be individualized and depends on the mechanism of injury, the timing, location and extent of the injury and the clinical state of the patient.

Open surgery is still the gold standard for patients that need

surgical intervention and most duodenal perforations can be

managed with a simple repair of the defect. Gastric diversion

procedures such as pyloric exclusion have been used for many years to treat duodenal perforations, but there is little evidence to support any benefit. Minimally invasive treatments are slowly emerging as alternative methods to open surgery in the treatment of duodenal perforation.

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