JAMA Surgery | Review

Management of Perforated Peptic Ulcer
A Review

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IMPORTANCE Perforated peptic ulcer disease (PUD) affects 4 million people annually worldwide, with a lifetime prevalence of 5% to 10%. Of those affected, 5% will progress to the point of perforation. Despite advances in the understanding and treatment, perforated PUD continues to have a high rate of morbidity (48.5%) and mortality (9.3%). This review summarizes the current evidence on management of perforated PUD, including management of failed repairs.

OBSERVATIONS Approaches for repair include primary closure and omental patch closure. Omental patch may be most useful in large perforations with friable tissue. Minimally invasive surgery is the preferred approach in perforated PUD, with improved outcomes compared with open techniques. Leak from the ulcer after repair is seen in approximately 12% to 17% of cases. Approaches to releak include expectant management, radiologic and/or endoscopic intervention, and repeat surgery. Morbidity and mortality after releak are especially high, and complete healing of the leak may take time.

CONCLUSIONS AND RELEVANCE Despite advances in medical management and surgical techniques, perforated PUD continues to have a relatively high rate of morbidity and mortality. Minimally invasive surgery is the current preferred treatment approach.

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Perforated peptic ulcer disease (PUD) was described as early as 167 BC.[1,2] Currently, PUD affects approximately 4 million people annually worldwide, with a lifetime prevalence of 5% to 10%.[3] Of those affected, 5% will progress to the point of perforation. A recent international study of more than 1800 patients with perforated peptic ulcer undergoing surgery from 159 hospitals across 52 countries revealed that the overall 30-day mortality rate was 9.3%.[4] Changes in modern medicine have led to a decrease in the incidence of PUD and a shift from surgical to medical management, although surgery has long been and continues to be the primary treatment for perforation.[5] This review summarizes the current evidence on management of perforated PUD, including management of failed repairs.

Discussion and Observations

Pathophysiology

Peptic ulcer disease is due to an imbalance between the protective and antagonistic factors within the stomach and duodenum. As a part of normal function, the stomach produces an acidic environment to aid digestion while maintaining a mucosal-bicarbonate layer to protect against ulceration.[6] Also involved in this delicate balance are prostaglandins (protective) and pepsin and bile salts (antagonistic). Once this balance is disrupted, PUD may occur, and progression can lead to perforation. Perforations occur most commonly in the proximal duodenum (35%-65%), pylorus (25%-45%), and stomach (5%-25%).[7]

Only in recent decades has the medical community fully recognized the external factors that can disrupt the gastroduodenal environment and contribute to ulcer formation. *Helicobacter pylori*, a flagellated, helical bacterium, is responsible for 50% to 80% of perforated PUD cases. The discovery of *H pylori* and its link to gastritis and PUD in 1982 led to the award of the Nobel Prize in Physiology or Medicine to Barry Marshall and Robin Warren.[8] *H pylori* creates an ulcerogenic state by disrupting the inhibitory control of gastrin release. Nonsteroidal anti-inflammatory drugs also contribute to PUD by blocking prostaglandin synthesis through inhibition of cyclooxygenase 1 enzyme. Other factors that contribute to an ulcerogenic state include smoking, alcohol, steroids, chemotherapy, and illicit drugs.[6]

Clinical Presentation

Approximately 66% to 88% of patients with perforated PUD present with acute onset of severe and generalized abdominal pain.[9] Patients with a known history of PUD (approximately 30% of patients presenting with perforated PUD) may also endorse preexisting gastroesophageal reflux disease; abdominal, chest, and/or back pain; nausea; vomiting; bloating; or belching.[10] During initial presentation, the differential diagnosis is broad and includes cardiac, gastrointestinal, vascular, and other pathologic criteria.

Assessment and Diagnosis

In patients who present for emergency medical attention with a perforated ulcer, vital signs typically include tachycardia and tachypnea as well as fever and hypotension in patients with sepsis. Electrocardiography will help exclude a cardiac event, and

laboratory evaluation will reveal the patient's current inflammatory state and end-organ function, exclude other diagnoses, and allow for preoperative preparations. Plain radiographs may show free air under the diaphragm, although the role of plain radiographs in diagnosis is waning with access to modern cross-sectional computed tomography.

Advanced radiographic imaging with computed tomography is the gold standard in making the diagnosis of perforated PUD (98% diagnostic accuracy).[9] The goal is expedient diagnosis to ensure timely access to definitive care of surgery. Every hour delay in surgery in patients with perforated PUD has been associated with a 2.4% decreased probability of survival (adjusted relative risk, 1.024; 95% CI, 1.011-1.037).[11]

Perforated PUD also has a grading system, published by the American Association for the Surgery of Trauma in 2016.[12] This system grades perforated PUD on a 1- to 5-point scale that uses clinical, radiographic, operative, and pathologic criteria. Although clinical utility of this grading scale is currently limited, it provides a uniform set of definitions for the study of outcomes and the potential for future quality improvement guidelines.

Treatment

Nonperforated PUD

Treatment of nonperforated PUD due to *H pylori* infection includes the use of antibiotic and acid-reducing therapy: triple therapy with a proton pump inhibitor (PPI), amoxicillin, and clarithromycin or a variation of this to treat resistant strains. Of note, the first PPI, omeprazole, was only approved by the US Food and Drug Administration in the US in 1989.[13] Additional treatment includes avoidance of contributory medications, such as nonsteroidal anti-inflammatory drugs and steroids. Furthermore, lifestyle modifications, including abstinence from smoking, alcohol, and illicit drugs, may further aid in treatment of nonperforated PUD.

Perforated PUD

Initial Treatment | Once the diagnosis of perforated PUD is made, initial treatment involves resuscitation with large-volume crystalloid therapy, treatment of sepsis with initiation of broad-spectrum antibiotics, careful nasogastric decompression, initiation of acid-reducing therapy with intravenous PPI therapy, and consideration of surgical treatment. In a patient with peritonitis, sepsis, and free air of unknown origin, these measures are also appropriate, even if a definitive diagnosis or perforated PUD as the cause is not made.

Surgical Treatment | With the introduction of PPI medications and the discovery of *H pylori*, the incidence of PUD dropped dramatically. The major resections and reconstructions frequently performed before these discoveries are now rarely performed for peptic ulcers and even less frequently performed in acute cases with perforation.

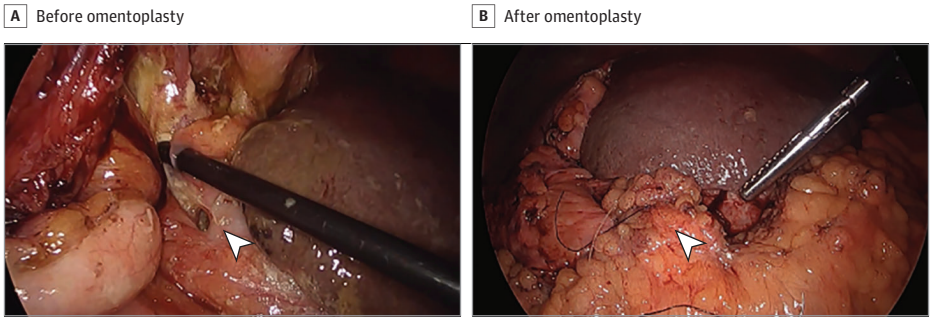
Omental patch, without primary closure of the perforation, was first described by Cellan-Jones in 1929.[14] His criticism of suture closure followed by omental coverage was the constriction of the duodenum. He described "some four to six sutures" traversing the perforation to create an "archway" of suture, under which a tongue of omentum would be secured with an additional suture fixation at the tip of the omentum. Roscoe Graham,[15] to whom omentoplasty is often attributed, later described, in 1937, his simplified technique of 3 sutures traversing the perforation to fixate a piece of free or attached omentum. Modern treatment of perforated PUD is still much the same, although advances in anesthesia, supportive care of the critically ill patient, and minimally invasive operative techniques have greatly improved outcomes.

The goal of surgery is to control ongoing leakage, remove spilled contaminants, and close the perforation. Although it is common practice to irrigate the abdomen with saline, this practice has been demonstrated to be of minimal benefit in appendicitis and possibly perforated peptic ulcer, although the evidence in the latter is limited and of low quality.[16-18] The abdomen should be explored and the perforation identified. If need be, the edges of the perforation should be debrided back to healthy tissue. If the perforation is gastric, a biopsy of the perforation edge is warranted to assess for malignant neoplasms and the presence of *H pylori*.

Primary Repair vs Omental Patch | Simple primary closure vs omental closure of a perforation continues to be a debated topic. Although primary closure can be performed in isolation, most surgeons perform this with an additional omental patch over the primary closure (hereafter *simple closure*). Simple closure entails closure of the defect with interrupted absorbable suture and use of either the same suture tails or additional pexy sutures to affix a tongue of omentum. Of note, when simple closure is performed, addition of an omental patch has not been associated with improved outcomes, particularly in smaller perforations.[19] Omental closure entails coverage of the perforation with a portion of omentum to plug the defect, secured with suture traversing the defect. Regardless of the method of repair, fixation of the omentum must not only be performed precisely to not strangulate the omentum but also provide adequate attachment and closure of the defect (**Figure 1**). Proponents of the omental closure technique advocate that it provides a tension-free repair without narrowing of the lumen, particularly when tissue is friable or the defect is large. Proponents of the primary closure technique advocate that it is easier to perform and provides a better seal of the defect. In a meta-analysis performed by Demetriou and Chapman[20] that compared primary closure with omental closure, no difference was found in rate of releak (odds ratio [OR], 0.64; 95% CI, 0.26-1.54), mortality (OR, 0.66; 95% CI, 0.25-1.76), or wound infection rate (OR, 0.65; 95% CI, 0.4-1.05).

Minimally Invasive vs Open Repairs | Laparoscopic repair of perforated PUD has been described since the 1990s.[21] Minimally invasive approaches include both laparoscopic and robotic-assisted approaches. Minimally invasive abdominal surgery has well-established benefits of decreased pain, wound complications, and hospital stay but requires additional skill and expertise to perform safely and may prolong operative time. In a 2022 systematic review by Chan et al[22] of 5311 patients from 29 studies, compared with open surgery, the laparoscopic approach was associated with improved morbidity and mortality. Laparoscopy had lower overall 30-day mortality (OR, 0.57; 95% CI, 0.35-0.92), overall morbidity (OR, 0.31; 95% CI, 0.18-0.53), surgical site infection (OR, 0.27; 95% CI, 0.18-0.42), and length of stay (mean difference, −2.84 days; 95% CI, −3.63 to −2.06 days). Releak (OR, 1.06; 95% CI, 0.43-2.61) and need for reoperation (OR, 0.61; 95% CI, 0.36-1.03) after repair was equivalent between open and laparoscopic approaches. Prior studies have shown equivalent mortality between open and laparoscopic approaches.[23,24]

Figure 1. Duodenal Ulcer Before and After Omentoplasty



Arrowhead indicates duodenal ulcer (A) and Graham patch (B).

Figure 2. Endoscopic Vacuum Sponge About to Be Placed Transorally at the Site of a Perforation

Primary open, or conversion to open, procedures may be necessary if a patient is hemodynamically unstable or unable to tolerate pneumoperitoneum, there is a lack of minimally invasive tools or skill set, or tissue destruction is too extensive for a minimally invasive approach. This recommendation echoes recommendations made by the World Society of Emergency Surgery's 2020 guidelines.[19] A tailored approach was also recommended for larger perforations (≥2 cm), which may necessitate a more complex, open, operative approach.

Special Populations | Ulcer perforations may also occur in patients with previous gastrojejunostomy anastomosis, such as patients with Roux-en-Y gastric bypass. In such scenarios, similar closure or repair techniques as previously described may be used.[25] Additionally, perforations that initially appear to be due to PUD may eventually reveal themselves to be secondary to cancer. In these circumstances, operative intervention is dictated by both management of the acute intestinal leakage as well as the additional goal of attempted oncologic resection. Large perforations (>2 cm) may lead to large areas of tissue destruction not amenable to simple patch or repair. Various procedures, such as jejunal serosal patch, resection and reconstruction with Roux-en-Y duodenojejunostomy, Billroth I, II, or Roux-en-Y gastrojejunostomy, pyloric exclusion, and tube drainage, have been described for these situations.[19] In select scenarios of patients with multiply recurrent ulcer disease undergoing maximal medical therapy, with other external factors or cancer ruled out, an acid-reducing operation, such as truncal or selective vagotomy, may be included at the time of addressing perforated PUD.

Posterior duodenal ulcers are more likely to bleed than to perforate. Retroperitoneal perforations present a unique challenge. Contained retroperitoneal perforations may be successfully managed nonoperatively with percutaneous image-guided or endoscopic transluminal or intraluminal drainage procedures.

Peptic ulcers can perforate and bleed, often from what are referred to as "kissing ulcers" on the anterior (perforated) and posterior (bleeding) duodenum. These complex ulcers that perforate and bleed are less common in the modern era.[26] Patients who present with concurrent bleeding and perforation are most likely to undergo suture ligation and Graham patch, probably via laparotomy. A more common scenario is the patient who has perforation, undergoes surgical treatment, and then bleeds shortly after surgery. Endoscopic management, angioembolization, or repeat surgery with suture ligation are all options in patients with severe life-threatening bleeding after perforation. Morbidity and mortality rates are high.

Observation and Nonoperative Management | Nonoperative management was one of the earliest proposed treatments for perforated PUD. In 1843, Edward Crisp proposed that many perforations self-sealed, and modern data confirm high incidence of self-sealing (40%-80%).[21] In the era of minimally invasive operative techniques, however, nonoperative management has a limited role in acute ulcers and may be best suited to stabilized patients who present in a delayed manner and in whom imaging with oral contrast reveals that there is no active leak or extravasation.

The use of endoscopic techniques for perforation closure or drainage, combined with radiographically placed abdominal drains, has also been proposed as a method of nonoperative treatment. One notable trial randomized patients to endoscopy with radiographic drainage vs surgical repair and drainage and found similar rates of successful closure, with less need for general anesthesia and lower morbidity in the endoscopic groups.[27] Endoscopic techniques include defect closure with the use of clips, endoscopic suturing, and/or coverage of defects with use of intraluminal stents. In addition to radiographically placed drains, endoscopic drainage options may include internal drainage with stents traversing from the lumen to the abscess cavity, as well as use of endoluminal vacuum therapy (**Figure 2**). Despite these early successes in nonoperative therapies, these methods are typically reserved for salvage of failed surgical repairs.[28]

Recurrent Leak After Repair | Releak after initial surgical repair has been reported in 12% to 17% of cases. Risk factors for releak include older age, septic shock, larger perforation size (>5 mm), delay in care, hypoalbuminemia, and duodenal ulcer.[29-31] Releak has been associated with higher overall complication rates, need for reoperation, and longer hospital stays. When recurrent leak occurs, options for repair include drainage with expectant management, endoscopic salvage techniques (as described in the Observation and Nonoperative Management section), and reoperation. Reoperation may entail repeat closure, omentopexy, omental closure, or resection with reconstruction. These procedures are difficult for patients to tolerate, and recovery can take time and be resource intensive.

Additional Considerations | The placement of intraperitoneal drains near the site of repair is controversial. Drains may decrease the need for repeat intervention and can identify releak early and possibly allow for nonoperative management. The downside to drains is that they may erode into inflamed tissue and may even contribute to failed repairs. A recent review of administrative data from Japan included 4869 patients from 324 hospitals who were undergoing surgery for perforated PUD over a 7-year interval. Propensity score matching revealed a lower incidence of postoperative interventions than the no-drain group (1.9 vs 5.6%; risk ratio, 0.35; $P = .003$).[32] A more recent randomized clinical trial of drain vs no drain in perforated peptic ulcer revealed that the no-drain group had significantly earlier return of bowel function and oral intake, less pain, and a shorter hospital stay.[33] However, no agreement has been reached regarding the use of drains. All patients who undergo treatment for PUD, perforated or not, should undergo an interval upper endoscopy at 6 weeks to assess for healing and repeat biopsy for bacterial and oncologic testing.[21]

Prognosis

Despite advances in the understanding and treatment of perforated PUD, the rates of morbidity (48.5%) and mortality (9.3%) continue to be high worldwide.[4] The Boey score is the most commonly cited prognostic scoring system and is designed to predict mortality in perforation (**Table**).[34,36] The original 1986 publication on the Boey score concluded that a score of 3 carried a 100% mortality rate; however, a more recent study examining all perforated PUD (not just duodenal) found a mortality rate of 77% with a Boey score of 3, highlighting advances in supportive care in the highest-risk cohort.[35] Recurrence rates are low if ulcers are treated and heal. A systematic review found that the mean long-term recurrence of perforation after healing was 12.2% (95% CI, 2.5%-21.0%).[37]

Table. Comparison of Original and More Recent Mortality Rates for Perforated Ulcer Disease Using the Boey Prognostication Score

	Mortality rate, %	
Boey score	Boey et al[34]	Lohsiriwat et al[35]
0	0	1
1	10	8
2	46	33
3	100	38

Conclusions

This review article summarizes the current state of management for perforated PUD and highlights the contemporary approach to various scenarios that lead to the best patient outcomes. Despite significant advances in medical management and surgical techniques, perforated PUD continues to have a relatively high rate of morbidity and mortality. Approaches for repair include primary closure and omental closure, as well as more extensive resection and reconstruction techniques. Minimally invasive surgery is the preferred treatment approach in perforated PUD, with improved outcomes over open techniques. There are emerging nonsurgical options for primary management or salvage after failed operative repairs as well.

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Concept and design: All authors.
Acquisition, analysis, or interpretation of data: Arshad, Gould.
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Critical review of the manuscript for important intellectual content: All authors.
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