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Emergency Ulcer Surgery

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Introduction

Once one of the most common indications for gastric surgery, the rate of elective surgery for peptic ulcer disease has been declining steadily over the last 3 decades. Data from American surgical training programs and Scandinavian national audits have shown a decrease in the rate of elective ulcer surgery of between 80 and 97% during the 1980's and 1990's^{1, 2}. During this same time period the rate of emergency ulcer surgery rose by 44%. In the United States in 2006 roughly 25,000 operations were performed for bleeding or perforated peptic ulcers³. These time trends mean that the gastrointestinal surgeon is likely to be called upon to manage the emergent complications of peptic ulcer disease in an elderly and ill patient without substantial experience in elective peptic ulcer disease surgery⁴. The goal of this review is to familiarize surgeons with our evolving understanding of the pathogenesis, epidemiology, presentation and management of peptic ulcer disease in the emergency setting, with a focus on peptic ulcer disease associated bleeding and perforation.

Pathogenesis of Peptic Ulcer Disease

The classic understanding of the pathogenesis of peptic ulcer disease is that it represents an imbalance between the toxicity of the gastric injurious forces of acid and pepsin and the mucosal defense mechanisms of the stomach and duodenum. Classically, the dictum was "No acid, no ulcer" and most ulcers were thought to be a consequence of excessive acid secretion caused by smoking, alcohol use, stress or other environmental factors⁵. In this model the pathogenesis was multifactorial, and many of the underlying factors were difficult to modify. Treatment of peptic ulcer disease needed to be chronic and was directed at the

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reduction of acid secretion either by vagotomy and/or surgical elimination of acid secreting gastric mucosa or by chronic use of medications such as H₂ antagonists or proton pump Inhibitors (PPI).

Our understanding of peptic ulcer pathogenesis was revolutionized by the discovery of the presence of the bacterium *Helicobacter pylori* in association with most gastric and duodenal ulcers in the early 1980's⁶. Over the next ten years multiple trials demonstrated that effective eradication of *H. pylori* with a short course of antibiotics and PPI's resulted in relapse free cure of the vast majority of ulcers. This led to an NIH consensus conference in 1994 that recommended treatment of *H. pylori* as the primary target of ulcer treatment⁷. With our increased understanding of the biology of *H. pylori*, it is now clear that infection of the gastric mucosa with *H. pylori* is responsible for most of the observed changes in gastric acid secretion observed in peptic ulcers. Patients with predominantly antral infection have impaired negative feedback of acid secretion, resulting in increased gastric acid production and they develop duodenal and pre-pyloric ulcers. Patients with uniform infection throughout the stomach often have low acid production secondary to inflammation of the gastric body which impair the normal function of the acid secreting mucosal and they frequently develop gastric ulcers. The effects of *H. pylori* infection on acid secretion are beautifully described in a recent review, and nicely explain the observed clinical finding of differential acid secretion in duodenal and gastric ulcers⁵.

The use of aspirin and Non-Steroidal Anti-Inflammatory Drugs (NSAIDS) has long been recognized as an important cause of peptic ulcer disease. These drugs inhibit the production of prostaglandins in the stomach that play a critical role in the mucosal defenses of the stomach against acid and pepsin induced injury⁸. In the stomach prostaglandins stimulate mucus and bicarbonate production, and play an important role in the regulation of gastric mucosal blood flow. By inhibiting mucosal defense mechanisms against acid mediated injury, NSAIDS are able to cause peptic ulceration independently, but also synergize with *H. pylori* infection to cause peptic ulcers⁹. Our current understanding of peptic ulcer disease suggests that *H. pylori* and NSAIDS use, either alone or in combination are the causative agents for the vast majority of peptic ulcers. This new understanding of peptic ulcer disease implies that the great majority of peptic ulcer disease is the result of treatable or modifiable causes. Based on this understanding of the pathogenesis of peptic ulcer disease, the classic surgical approach directed at reducing acid production must be carefully reevaluated.

The Epidemiology of Peptic Ulcer Disease

Once relatively common across all age groups, in the 21st century peptic ulcer disease is predominantly a disease of the elderly. Patients presenting with complications of peptic ulcer disease are most commonly in the 7th and 8th decades of life and there is a male predominance with roughly 1.5 times as many cases in men than women^{3, 10}. Overall there has been a marked decline in the incidence of all peptic ulcer disease, with data from multiple countries showing declines in ulcer hospitalization rates of 40–50% over the last three decades^{1, 3, 11}. Duodenal ulcer is more common than gastric ulcer although the largest decreases in ulcer incidence have been seen in duodenal ulcer¹⁰. Despite a declining incidence overall of peptic ulcer disease, the incidence of peptic ulcer disease complicated by either bleeding or perforation has remained constant or in fact even increased. Although the data is inconsistent in different countries, data from Finland and the Netherlands suggest that the rate of ulcer complications and the need for emergent ulcer surgery may have increased slightly over the last 30 years^{1, 11}.

These epidemiologic changes make sense with our new understanding of the pathophysiology of peptic ulcers. The rate of *H. pylori* infection has been decreasing over

time, both as a consequence of improved sanitation, treatment of infection and a cohort effect. This likely explains the decrease overall in ulcer disease and aging of the ulcer patient. At the same time with an aging population and increased use of NSAIDS, the reasons for increase in ulcer complications particularly in elderly seems clear. For the surgeon dealing with patients with ulcer emergencies, this means increasingly being called on to offer surgical therapy to elderly frail patients.

Bleeding Peptic Ulcer

PRESENTATION AND INITIAL MANAGEMENT

Patients with bleeding from peptic ulcer will usually present with hematemesis, melana or both. In the cases of massive bleeding they can occasionally present with hematochezia. Many patients will present with hemodynamic findings of significant volume loss or even shock. Patients may also report a history of syncope prior to presentation that should suggest significant blood loss. The initial management of all non-variceal upper GI bleeding is directed at obtaining IV access, ensuring the availability of blood for possible transfusion, and initiating resuscitation of the patient with either crystalloid solutions or blood if evidence of significant blood loss exists. The primary therapeutic goal in a patient with acute upper GI bleeding is control of bleeding, and the goal of a surgeon in managing a bleeding peptic ulcer is to provide definitive hemostasis. The challenge in managing bleeding peptic ulcers, is that many patients will stop bleeding spontaneously, and only 5–10% of patients with bleeding ulcers will require surgery. To help identify patients likely to require intervention for bleeding control, and those at high risk for re-bleeding and death from bleeding ulcers, several scoring systems based on clinical and endoscopic variables have been developed. The use of the prognostic systems for risk stratification is one of the major recommendations of a recently published international consensus statement on UGIB, and surgeons managing peptic ulcers should be familiar with their use¹². The Blatchford score uses clinical and laboratory data such as hemodynamic parameters, hemoglobin, and BUN level, and co-morbid conditions to assess patients and can accurately identify patients at low risk of requiring intervention. The full scoring system is outlined in Table 1. Based on Blatchford's initial data, patients with a score of 3 or less have a less than 6% chance of requiring intervention for hemostasis while those with a score of 6 or higher have a greater than 50% chance of requiring intervention for control of bleeding¹³.

ENDOSCOPIC INTERVENTION

The most important step in the management of a patient with a bleeding peptic ulcer is to arrange for urgent upper GI endoscopy. Upper GI endoscopy is critical in establishing the etiology of the bleeding, of which up to 60% is related to peptic ulcer disease¹⁴. More importantly in most cases of active GI bleeding, endoscopic hemostatic techniques will be successful in controlling the source of bleeding. Meta-analysis of data in the early 1990's demonstrated that endoscopic therapy is effective at controlling peptic ulcer bleeding and reducing the risk of mortality and the need for surgical intervention^{15, 16}. More recent data has shown that the use of epinephrine injection combined with an additional technique such as thermal contact, sclerosant or clipping improves success in controlling initial bleeding¹⁷. In the hands of a skilled endoscopist, bleeding can initially be controlled in almost all cases. Essentially all patients with bleeding peptic ulcers should undergo upper endoscopy prior to the consideration of surgical therapy. It is important, however for the surgeon to be present at the time of endoscopy, as important anatomic information will be gained during the endoscopic procedure. Failure of initial endoscopic hemostasis attempts is one of the indications for surgery in bleeding peptic ulcers.

Despite the high success rates of initial endoscopic hemostasis, roughly 15–20% of patients will experience re-bleeding from their ulcer. Rockall and colleagues identified in 1996 that re-bleeding in patients with peptic ulcer disease is an important contributor to mortality risk¹⁸. Based on a large cohort of patients they devised a clinical scoring system based on patient characteristics and endoscopic findings that could be used to predict mortality and risk of re-bleeding in patients with peptic ulcer disease. Patients with a Rockall score of 3 or less have a risk of re-bleeding of 11% and a mortality rate of less than 5%, while those with a score of 5 or higher have a re-bleeding rate of 25% and a greater than 10% risk of death. The components of the Rockall score are summarized in table 2. Further study of the Rockall score has suggested that it is better at predicting mortality than re-bleeding, and has lead to multiple attempts to better define the risk factors for re-bleeding. In a recent systematic review, six factors were identified as independent predictors of rebleeding: hemodynamic instability, comorbid illnesses, active bleeding at endoscopy, >2 cm ulcer size, and ulcer location in either the posterior duodenum or lesser curvature of the stomach¹⁹.

The role of the surgeon in patients at risk for re-bleeding after endoscopic hemostasis remains an area of controversy. Data from the 1980's before wide spread availability of modern endoscopic techniques for hemostasis could not prove that early operation for ulcers without active bleeding improved mortality, but it did show that it resulted in an increase in the number of patients undergoing operation^{20, 21}. One small trial using modern endoscopic hemostasis techniques that compared early elective operation with endoscopic retreatment per protocol after initial endoscopic control of ulcer bleeding. This trial showed that patients who underwent early elective surgery were less likely to re-bleed, but showed no difference in overall mortality or need for emergency surgery²². It is worth noting that more than 75% of the patients receiving endoscopic therapy achieved definitive hemostasis without surgery. This data agrees with the results of an elegant randomized controlled trial performed by Lau and colleagues that demonstrated that endoscopic retreatment of peptic ulcers that re-bleed after initial endoscopic treatment was successful in nearly 75% of patients and associated with similar mortality and significantly fewer complications than immediate surgery for re-bleeding²³. In this study two factors predicted failure of endoscopic retreatment for recurrent bleeding, an ulcer larger than 2 cm and patients who developed hypotension with the recurrent bleeding. Taken altogether these studies suggest that early elective surgery for bleeding peptic ulcer does not reduce the mortality risk, but it does reduce the risk of re-bleeding. It seems reasonable to consider early elective operative intervention in those patients who are at high risk of recurrent bleeding such as those with ulcers > 2cm, hypotension on presentation, with posterior duodenal or lesser curvature gastric ulcers. This recommendation must be balanced against significant risk of complications and death in this elderly frail patient population, and requires the exercise of careful surgical judgment.

OPERATIVE APPROACH

The primary goal of any operation for a bleeding peptic ulcer is hemorrhage control. Classically, the secondary goal of surgery was treatment of the underlying ulcer diathesis. With our current understanding of the underlying causes of peptic ulcers and the advent of potent acid suppressive medications, the need for surgical reduction of acid secretion is less clear. The preferred operative approach to a peptic ulcer will depend on the location of the ulcer, and for this reason it is important for the surgeon caring for the patient to be present during upper GI endoscopy to obtain precise information on the location of the ulcer.

Bleeding gastric ulcers are generally best treated by excision of the ulcer and repair of the resulting gastric defect. Excision or biopsy of the ulcer is important, as 4–5% of benign appearing ulcers are actually malignant ulcers²⁴. For ulcers along the greater curvature of the stomach, antrum or body of the stomach wedge excision of the ulcer and closure of the

resulting defect can easily be achieved in most cases without causing significant deformation of the stomach. Gastric ulcers along the lesser curvature of the stomach are more problematic. Because of the rich arcade of vessels from the left gastric artery, wedge excision of these ulcers is more difficult than in other locations, and the subsequent closure of the gastric defect is much more likely to result in deformation of the stomach and either luminal obstruction or gastric volvulus of the resulting J-shaped stomach. For distal gastric ulcers along the lesser curvature in the area of the incisura angularis, a distal gastrectomy with either a Bilroth I or Bilroth II reconstruction is often the easiest method of excising the ulcer and restoring GI continuity. A special case is the proximal gastric ulcer near the gastroesophageal (GE) junction. Wedge excision of these ulcers will often result in compromise of the GE junction and leak. In most patients the easiest approach is an anterior gastrotomy with biopsy and oversewing of the ulcer from inside the gastric lumen. With this approach it is relatively easy to avoid compromising the GE junction. In the event that ulcer excision is necessary, a Csendes procedure, a distal gastrectomy with tongue shaped extension of the lesser curve resection margin to include the ulcer and subsequent Roux-Y esophagogastrorjunostomy is an excellent option²⁵.

The standard approach to a bleeding duodenal ulcer is to perform an anterior longitudinal duodenotomy extending across the pylorus to the distal stomach. The bleeding vessel, often the gastroduodenal artery is ligated in the ulcer crater by placing a figure of eight suture at the top and the bottom of the ulcer crater to control the artery proximally and distally. A third suture is placed as a U-stitch underneath the ulcer to control the transverse pancreatic branches that enter the GDA posteriorly. The transverse duodenal incision is then closed vertically to construct a Heineke-Mikulicz pyloroplasty. Classically a truncal vagotomy is then performed to reduce the risk of recurrent ulceration. The role of the vagotomy in 2011 is unclear. Our modern understanding of the pathogenesis of peptic ulcer suggests that treatment of *H. pylori* and elimination of NSAID use should result in cure of the underlying risk of ulcer. Further, with the advent of PPI's it is now possible to medically eliminate gastric acid production without the side effects of vagotomy. Although level one data exists for perforated duodenal ulcer demonstrating that *H. pylori* treatment eliminates the need for definitive ulcer surgery, there is to date no trial that confirms this finding in the case of bleeding duodenal ulcer²⁶. Despite the lack of level one evidence, surveys of surgeons in the United Kingdom²⁷, and national data from the United States³ suggest that most surgeons no longer perform a vagotomy as a component of operation for bleeding duodenal ulcer.

Although duodenotomy with direct control of the bleeding site with or without vagotomy is the most commonly used approach for a bleeding duodenal ulcer, there is some data to suggest that a more extensive operation may be associated with a lower re-bleeding rate. In 1993 Millat and colleagues published a randomized controlled trial comparing vagotomy and pyloroplasty with gastric resection combined with ulcer excision. They found that the re-bleeding rate was higher (17% vs 3%) with vagotomy and pyloroplasty, but the overall mortality was not different²⁸. The major complication rate, mostly duodenal leaks, was significantly higher after gastric resection. An important caveat to this data is that this study was performed prior to widespread use of PPI's and *H. pylori* treatment, and it is unclear that there is still a place for aggressive surgical treatment of the underlying ulcer disease now that medical therapy has replaced surgical therapy as the mainstay of ulcer treatment. In patients without significant comorbidities, who are not in shock at the time of operation, a more aggressive surgical approach may be warranted in patients with large posterior duodenal ulcers. Given the challenges of dealing with the difficult duodenal stump in a large posterior duodenal ulcer, this approach should only be undertaken by surgeons with significant experience in ulcer surgery.

Despite the best surgical efforts, re-bleeding after vagotomy and pyloroplasty occurs in between 6–17% of cases^{28, 29}. Endoscopic therapy is generally not an option after a recent duodenotomy, leaving two options either reoperation or transcatheter arterial embolization (TAE). Classically reoperation was the procedure of choice for rebleeding after duodenotomy. In the case of reoperation for recurrent bleeding most surgeons have advocated a more extensive operation, usually distal gastrectomy with or without vagotomy and ulcer excision or exclusion. This approach is unfortunately fraught with complications and associated with high operative mortality^{28, 29}. More recently several authors have advocated TAE as a viable alternative to operative treatment for ulcer bleeding refractory to endoscopy. Without a head to head trial, it is unclear whether TAE should replace surgery as a primary approach to bleeding control, but data from two large series suggest that TAE can achieve long-term hemostasis in roughly 75% of patients with recurrent bleeding after duodenotomy and ulcer oversewing^{30, 31}. Given the significant risk of complication or mortality in reoperation for recurrent bleeding, TAE, when available should be the first line therapy for recurrent bleeding after duodenotomy and ulcer oversewing.

PERFORATED ULCER

The therapeutic goal in a perforated peptic ulcer is to repair the hole in the GI tract and treat peritoneal contamination. Unlike in the case of bleeding duodenal ulcers, surgery is the mainstay of treatment for perforated peptic ulcers. The majority of perforated ulcers occur in the duodenum and pyloric channel; in an analysis of 40 trials of perforated peptic ulcer disease, perforation was most common at the duodenal bulb (62%), followed by the pyloric region (20%), and the gastric body (18%)³². Although the majority of patients who present with ulcer perforation have no prior history of ulcer disease, risk factors for perforation include the prior history of ulcer disease or use of NSAIDs³³. In patients on NSAID therapy, there is a greater risk of ulcer perforation with a history of prior ulcer, age greater than 60 years, concomitant use of alendronate, selective serotonin reuptake inhibitors, steroids, or anticoagulants^{34–37}.

PRESENTATION

Classically, the presentation of a perforated peptic ulcer is described as a three-stage process³⁸. Initial symptoms, occurring within 2 hours of perforation, include the abrupt onset of abdominal pain. The pain may initially be focused at the epigastrium, but it can quickly become generalized. Between 2 and 12 hours of perforation, the abdominal pain worsens and there may be significant pain with palpation of the hypogastrium and right lower quadrant secondary to drainage of succus from the perforation. 12 hours after perforation, in addition to increasing pain, the patient may have fever, signs of hypovolemia, and abdominal distention.

EVALUATION

It is important to quickly diagnose a perforated peptic ulcer. The prognosis is improved if treatment is provided within 6 hours of perforation, whereas a delay in treatment beyond 12 hours following perforation is associated with an increase in both morbidity and mortality^{38, 39}. A prospective study of patients with duodenal ulcer perforations, Boey and colleagues identified that perforations >48 hours, pre-operative shock, and concurrent medical illness were associated with an increase in mortality⁴⁰. In a patient with an appropriate history, if free air is present on an upright chest or abdominal x-ray or computed tomography (CT) scan, no additional testing is required before proceeding with treatment. However, direct findings of perforation are not identified in 10–20% of patients with a perforated duodenal ulcer⁴¹. An upper GI study or abdominal CT scan with oral contrast may be performed to confirm the diagnosis.

The patient should be evaluated for *Helicobacter pylori* infection as knowledge of a patient's *H. pylori* status can play an important role in treatment decisions. *H. pylori* infection is present in 70–90% of duodenal ulcers and 30–60% of gastric ulcers and antibiotic therapy is very effective at eradication⁴². Non-invasive testing options include urea breath testing, stool antigen testing, and serology. Stool antigen testing is a modern and rapid method of gaining information on a patient's *H. pylori* status in the preoperative period. A monoclonal stool antigen test has a 94% sensitivity, 97% specificity, and is processed in an hour⁴³. A rapid stool antigen test may be processed within 5 minutes; however the sensitivity is 76% and specificity 98%⁴⁴.

TREATMENT

Medical management of a perforated peptic ulcer consists of fluid resuscitation, nasogastric decompression, acid suppression, and empiric antibiotic therapy. Antibiotic therapy should cover enteric gram-negative rods, anaerobes, oral flora and fungus^{45, 46}. A non-surgical treatment plan consisting of only the aforementioned medical management has been proposed for patients with contained perforation at high risk for operative complications⁴⁷. Despite the appeal of non-operative therapy in high risk patients, the application of this strategy is likely limited, as was demonstrated in a randomized, controlled trial of non-operative treatment for perforated peptic ulcers in which patients over 70 years of age were less likely to improve with conservative management⁴⁸.

Operative intervention is almost always indicated in the treatment of perforated peptic ulcers. Unfortunately, emergency surgery for a perforated peptic ulcer has a 6–30% risk of mortality³⁹. In the setting of emergency surgery for perforated peptic ulcer, several variables have been independently associated with an increase risk of mortality, including age, American Society of Anesthesiologists (ASA) class, shock on admission, hypoalbuminemia on admission, an elevated serum creatinine, and pre-operative metabolic acidosis⁴⁹. Unfortunately most of these adverse prognostic factors are not modifiable, and despite substantial advances in medical care, there has been little change in the mortality of perforated ulcer over the last 15 years³.

The choice of operation will depend on the site of perforation found at exploration. Duodenal and pyloric channel perforations are the most common site of ulcer perforation and are functionally grouped as duodenal perforations. The most common technique for the management of a perforated duodenal ulcer is a patch repair with an omental pedicle commonly referred to as a Graham patch or omentopexy⁵⁰. In this technique the ulcer is not closed, but instead a pedicle of vascularized omentum is sutured over the perforation site with multiple interrupted sutures. These repairs may be performed by a laparoscopic or open approach, but ulcers over 10 mm in size appear to increase the risk of conversion to open surgery. In a randomized controlled trial of 121 patients with perforated peptic ulcer disease Siu and colleagues demonstrated significantly lower analgesic requirements, post-operative hospital length of stay, and time away from work in patients receiving a laparoscopic repair. Importantly, there were no significant differences between the groups receiving an open or laparoscopic repair in terms of mortality, incidence of reoperation, or in the identification of post-operative intra-abdominal fluid collections⁵¹. Classically repair of a perforated duodenal ulcer was accompanied by a definitive ulcer operation either a vagotomy and pyloroplasty or a patch repair and a parietal cell vagotomy. However with our improved understanding of the pathogenesis of peptic ulcers, it appears that definitive ulcer surgery is no longer necessary in most cases. Patch repair of the perforation with concomitant medical therapy is often sufficient for patients with ulcer disease secondary to *H. pylori* infection or NSAIDs. A randomized study of 99 patients with perforated duodenal ulcers infected with *H. pylori* treated with a patch repair demonstrated that successful treatment of *H. pylori* significantly decreased ulcer recurrence from 38% to 5%, leading the authors to conclude

that a definitive ulcer procedure is not necessary in this setting⁵². In light of this data knowledge of a patient's *H. pylori* status prior to surgery cannot be understated.

In the rare patient with a history of *H. pylori* negative peptic ulcer disease or those who are unable to stop NSAID therapy, a definitive ulcer procedure may be performed if the patient is hemodynamically stable and has minimal intra-abdominal contamination. In this setting, a truncal vagotomy and pyloroplasty, omental patch and parietal cell vagotomy, or an antrectomy with truncal vagotomy have all been advocated as suitable repairs. Vagotomy and pyloroplasty is the easiest operation to perform, but has an 10–15% ulcer recurrence rate and exposes the patient to all of the complications of dumping and post vagotomy syndromes. Omental patch with parietal cell vagotomy avoids most of the complications of dumping and post vagotomy syndrome, but is a more challenging operation and high ulcer recurrence rates have been reported in inexperienced hands⁵³. The benefits of a vagotomy with antrectomy are that the procedure may be applied to a variety of situations, and that the ulcer recurrence rate is very low. The disadvantages are that the operative mortality is higher than either of the other procedures, and the surgeon is forced to deal with an often chronically scarred duodenal stump and the complications of duodenal stump leak or anastomotic failure. The choice of definitive operation should depend on the experience of the surgeon, but in the absence of significant experience with ulcer surgery, vagotomy and pyloroplasty or not performing definitive surgery in the emergent setting seems prudent.

In the case of a perforated gastric ulcer, either ulcer excision and repair of the defect or biopsy and omental patch are the most expeditious approach in the emergency setting. Because malignancy has been reported in 4–14% of gastric perforations, biopsy or excision of the ulcer when feasible is important⁵⁴. For a gastric ulcer located along the greater curvature, antrum or body of the stomach, simple wedge excision of the ulcer is easy to perform, often with a single fire of a linear stapler, simultaneously obtaining tissue for biopsy and closing the perforation. Although no trials have been conducted comparing the techniques, this could be performed either with an open or laparoscopic approach. As with bleeding ulcers, ulcers along the lesser curvature of the stomach are more challenging because of the left gastric artery arcade, and the GE junction in high lesser curve ulcers. For distal lesser curve ulcers distal gastrectomy can be performed with similar mortality to that seen with patch or simple excision⁵⁵. The operative approach to perforation of an ulcer located next to the esophagogastric junction may include a subtotal gastrectomy to include the ulcer with a Roux-en-Y esophagogastric jejunostomy as described above for bleeding ulcers.

A particularly challenging clinical scenario is the perforated giant duodenal ulcer. With a duodenal ulcer perforation greater than 2 cm, there is an increased risk of repair failure with omental patch repair, with leak rates of up to 12% reported⁵⁶. In the setting of a giant perforated duodenal ulcer there is no standard management. Recommendations for repair include: omental patch, controlled tube duodenostomy, jejunal pedicled graft, jejunal serosal patch, pedicled omental plug, partial gastrectomy, and gastric disconnection^{56–58}. The pedicled omental plug is an intriguing and easy option for this problem. In this procedure an NG tube is passed out through the perforation and a tongue of omentum sutured to the NG tube. This is withdrawn back into the stomach and the omental plug is then sewn to the edges of the ulcer. In a single randomized trial comparing omental plug with standard omental patch, plug repair was associated with a lower recurrent leak and duodenal stenosis rate⁵⁶. The choice of repair should be influenced by the patient's clinical status, the size of the perforation, the degree of intraperitoneal contamination, and the surgeon's experience.

Summary

Our current understanding of peptic ulcer disease as an infectious disease caused by *H. pylori* infection, or a side effect of NSAID use has almost eliminated elective surgery for peptic ulcer disease. However complications of peptic ulcer disease either bleeding or perforation still frequently require surgical intervention. Although bleeding peptic ulcers can usually be treated with non-surgical means, 5–10% will require emergent surgery for hemostasis. With effective medical therapy for peptic ulcer disease, surgical therapy is now focused on obtaining hemostasis and not the underlying ulcer diathesis. Almost all perforated peptic ulcers will require surgery, but the focus of surgery has changed to a damage control approach rather than one directed at definitive surgical therapy. Although the surgeon does not have to master acid reducing surgery any longer, emergency ulcer surgery remains high risk surgery and surgeons must be familiar with the many options for managing this challenging problem.

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Table 1

Blatchford Score

Blood Urea Nitrogen (BUN mg/dl)	
18.2–22.4	2
22.4–28	3
28–70	4
> 70	6
Hemoglobin for Men (g/dl)	
12–13	1
10–12	3
<10	6
Hemoglobin for Womens (g/dl)	
10–12	1
< 10	6
Systolic Blood Pressure mmHg	
100–109	1
90–99	2
<90	3
Heart Rate >100 Bpm	1
Presentation with Melena	1
Presentation with Syncope	2
Hepatic Disease	2
History of Heart Failure	2

Data adapted from Blatchford O, Murray WR, Blatchford M. A risk score to predict need for treatment for upper-gastrointestinal haemorrhage. *Lancet* 2000;356(9238):1318–1321.

Table 2

Rockall Score

Variable	0	1	2	3
Age	<60	60–79	80	
Shock	No Shock	Tachycardia	Hypotension	
Comorbidity	None		Cardiac Disease	Liver or Kidney Failure
Diagnosis	Mallory-Weiss or no diagnosis	All other Diagnoses	Upper GI Malignancy	
Stigmata of recent Hemorrhage	None or Dark Spot		Blood in GI tract, bleeding or visible vessel, adherent clot	

Data adapted from Rockall TA, Logan RF, Devlin HB, et al. Risk assessment after acute upper gastrointestinal haemorrhage. *Gut* 1996;38(3):316–321.