

Surgical treatment of peptic ulcer disease: current indications and techniques

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Abstract

The incidence of peptic ulcer disease has dramatically decreased in the last recent years, mainly due to the knowledge of *Helicobacter pylori* role in the pathogenesis and the eradication treatments. Also, effective acid-decreasing drugs have contributed to the healing of most ulcers. As a result, indications for elective surgery have also dramatically decreased. However, there are some indications for elective surgery such as refractory ulcers and cases of uncertain diagnosis. Also, developing of alternative therapeutic methods such as endoscopy or angioembolization has reduced the need for surgery. Endoscopic therapy is used in the treatment of bleeding ulcers with high rates of success and in cases of gastric outlet obstruction. Angioembolization is used in selected cases of bleeding ulcers. Surgery is today indicated when these procedures fail in hemorrhages of peptic origin. Other indication for surgery is perforated peptic ulcer, since non-operative treatment cannot be considered standard of care. Considerable debate exists concerning the need of adding acid-decreasing procedures to techniques of bleeding control alone in case of hemorrhage. Although the latter are associated with less side effects, the former are associated with lower rates of rebleeding. Simple closure of a perforation, however, appears enough if followed by *Helicobacter pylori* eradication and avoidance of NSAIDs. Importantly, any gastric ulcer must be biopsied to rule out malignancy, before deciding any conservative treatment.

INTRODUCTION

Although the need for surgery has dramatically decreased in the last recent years, some indications still persist. Many cases of refractory ulcers or uncertain diagnosis and gastric outlet obstruction unsuccessfully treated by endoscopy need elective surgery. Also, cases of bleeding ulcers who fail to respond to endoscopic therapy, as well as perforated ulcers, also need emergency surgery.

Controversy exist concerning the need of adding acid-decreasing procedures to simple techniques –such as perforation closure or vessel ligation-, and also concerning which procedure: resection versus vagotomy.

Although the incidence of elective surgery for intractable peptic ulcer disease has decreased [1] due to the development of acid-suppressive medications and the recognition of the role of *Helicobacter pylori* (Hpy) and NSAIDs on its pathophysiology, the number of urgent operations for ulcer complications has remained constant or increased [2].

The purpose of this paper is to review the current role of surgery in the treatment of peptic ulcer disease including elective and emergency procedures. At this point we will discuss the role of minimally invasive techniques.

Pathophysiologic features of peptic ulcer disease.

The vast majority of duodenal and gastric ulcers are caused by NSAID intake and/or Hpy infection [3]. The pathogenic mechanism is the acid-peptic damage of the gastric or duodenal

mucosal barrier, so acid secretion suppression with drugs or surgery is the clue to healing the ulcer and to decreasing peptic disease recurrence [4].

Other agents that have been involved in the development of the ulcer disease and its recurrence are alcohol, smoking, stress, and other drugs such as aspirin, corticoids or cocaine. Some diseases such as Zollinger-Ellison syndrome [5], systemic mastocytosis, trauma, burns and major physiological stress [6] are known to cause peptic ulcer too (Table 1).

Currently, most patients can be managed in a non-operative way, by avoiding NSAID consumption, eradicating Hpy, or by means of endoscopic treatment in case of bleeding [7]. However, there are three main clinical scenarios where surgery can still be necessary: drug-resistant ulcers, uncertain diagnosis and ulcers complicated by bleeding or perforation or, less commonly, gastric outlet obstruction.

ELECTIVE SURGERY

Drug-resistant ulcer or refractory ulcers

Nowadays, most peptic ulcers respond to treatment with antisecretory and antimicrobial drugs against Hpy infection as well as to the avoidance of NSAID intake, but in some patients, the treatment is not effective enough or the peptic disease recurs. Some factors are known to influence the healing or recurrence of peptic ulcer disease such as the continued use of NSAIDs, gastric hypersecretion states due to a gastrinoma, failed eradication of Hpy or an occult focus of carcinoma than mimics ulcer disease.

Although poorly defined, an ulcer can be considered as refractory if the healing, tested endoscopically, is not evident after 8 to 12 weeks of proper medical management - 8 to 12 weeks of treatment with proton pump inhibitors, Hpy eradication therapy, if necessary, and avoidance of NSAIDs [8]. Also ulcers that relapse while on maintenance therapy or after multiple courses of treatment can be included in this category.

The incidence of refractory peptic ulcer disease has decreased in the past five decades. An observational study that included 4032 patients suffering from duodenal ulcer between 1976 and 1993 defined refractory as the persistence of ulceration after 12 weeks of treatment (H2 receptor antagonist era) by an endoscopic examination. The incidence of refractory duodenal ulcer decreased from 33% in 1976-1978 to 7% in 1989-1993 [9]. The study was performed before proton pump inhibitors and Hpy treatment became routine, but there are no other studies that test their mechanism or their role in the natural history of peptic disease.

- A. Drugs other than NSAIDs
 - • Acetaminophen
 - • Bisphosphonates
 - • Glucocorticoids
 - • Clopidogrel
 - • Sirolimus
 - • Spironolactone
 - • SSRI: Selective serotonin reuptake inhibitors
 - • Chemotherapy
 - • Cocaine
- B. Hormonal or mediator-induced, including secondary acid hypersecretory states
 - • Gastrinoma
 - • Systemic mastocytosis
 - • Carcinoid syndrome
 - • Basophilia in myeloproliferative disorders
 - • Antral G cell hyperfunction
- C. Recurrent ulceration following gastrectomy for peptic ulcer disease
 - D. Infections non H. Pylori)
 - • Herpes simplex virus type I
 - • Cytomegalovirus
 - • Other infections
 - E. Mechanical causes
 - • Duodenal obstruction
 - F. Radiation therapy
- G. Inflammatory and infiltrating disease

- • Sarcoidosis
- • Crohn's disease
 - • Other
 - H. Idiopathic
- • Idiopathic hypersecretory duodenal ulcer

Table 1. Other causes of peptic ulcer disease.

In refractory or recurrent ulcers persistence of Hpy infection, the continuation of NSAID intake, gastrinoma and other uncommon causes (Table 1) have to be ruled out. As a result, the first step is to confirm with the patient and relatives a real avoidance of NSAIDs.

The second step is confirmation of a true absence of Hpy. Evaluation for Hpy presence is usually done by culture or biopsy – samples taken by endoscopy [10] -, urea breath testing or serology [11], although this has a low accuracy. Although a single positive test is enough to diagnose the infection, at least two negative tests are necessary to accept negativity.

If these factors are excluded, then an endoscopic biopsy must be performed to exclude malignancies or other benign disease [12]. If malignancy is suspected, surgery is needed irrespective of the biopsy result. (See below)

Also, a search for gastrinoma must be performed, even in the absence of family history. This can be accomplished by means of fasting serum gastrin assessment [13] and the secretin stimulation test. Caution must be taken since patients receiving proton pump inhibitors can exhibit elevated serum gastrin levels. In this case, the levels must be repeated one week after drug withdrawal. The secretin stimulation test can differentiate patients with gastrinoma from those having other causes of hypergastrinemia.

Serum gastrin false negatives are possible. If the clinical suspicion of gastrinoma is high, then serum chromogranin A levels must be obtained [14].

If a biochemical diagnosis of gastrinoma has been made, anatomical location must be searched by means of CT, MRI or intraoperative ultrasound, and treated according to location.

After ruling out all the known causes, elective surgery can be indicated.

Uncertain diagnosis

In some cases, a gastric - or less commonly, a duodenal ulcer – has atypical features suggesting it is not a peptic ulcer. They can be caused by a gastric carcinoma or lymphoma and, in the case of the duodenum, by a pancreatic or duodenal carcinoma [12]. They must be upfront biopsied by endoscopy. The same as refractory ulcers, if malignancy is suspected, surgery is needed irrespective of the biopsy result.

Gastric outlet obstruction

The obstruction caused by peptic ulcer disease is today the least common indication for operation in these patients [15]. It is believed to be the result of edema and scars produced by the ulcer, followed by healing and fibrosis, which lead to obstruction of the gastroduodenal junction (usually in the first part of the duodenum).

Nowadays, outlet gastric obstruction due to peptic ulcer disease is exceptional because of the improvement of medical care and endoscopic procedures [16].

The main symptom is vomiting, which typically occurs postprandial, of undigested food, devoid of any bile. A history of previous peptic ulcer disease and loss of weight is also common. In advanced cases, dehydration and nutritional deficiencies are very typical.

Before any kind of treatment, correction of electrolyte disorders and nutritional abnormalities is necessary, as well as emptying the stomach with a nasogastric tube.

A diagnostic endoscopy must be performed to rule out malignancy, taking multiple biopsies followed by endoscopic balloon dilatation in order to resolve the obstacle. This procedure is successful in most cases (80-90%). However, Lau and colleagues demonstrated a high rate of initial endoscopic failure (17%), from negotiating the stenosis, or from balloon induced perforation. They also revealed that, even in patients with initial successful dilatation, at least one half will require subsequent operation. As a result, they recommend balloon dilatation only for high risk patients [17].

Other authors recommend endoscopic balloon dilatation techniques followed by the placement of a stent, but there are no long-term studies comparing these procedures in benign disorders [18].

Csendes and colleagues compared three operations for obstructing duodenal ulcer: highly selective vagotomy (HSV) and gastrojejunostomy, HSV and Jaboulay gastroduodenostomy, selective vagotomy (SV) and antrectomy. They found the first and third procedures significantly better and they recommend HSV and gastrojejunostomy as the treatment of choice for patients with duodenal ulcer and gastric outlet obstruction [19].

In summary, the surgical treatment of outlet gastric obstruction due to peptic ulcer disease would be indicated if the endoscopic procedures failed.

Surgical techniques for elective operations

Elective surgery for peptic ulcer is today uncommon. Understanding the role of H₂ and NSAIDs in the physiology of ulcer disease has reduced the number of surgical procedures and, in case of refractory or recurrence, the surgical procedures are less invasive and have few side effects.

For duodenal ulcers, the procedures are based on acid secretion reduction. This can be accomplished by sectioning the vagus nerve branches (truncal vagotomy, selective vagotomy, highly-selective vagotomy) or by reducing gastrin stimulation (antrectomy) or decreasing the number of acid-producing parietal cells (subtotal gastrectomy).

In the case of gastric ulcers, malignancy can be occult, so the ulcer must be excised. As a result, gastric resection is advocated (antrectomy or subtotal gastrectomy).

Vagotomy

Vagotomy was once commonly performed to treat and prevent peptic ulcer disease. However, with the availability of good acid secretion control with H₂ receptor antagonists and proton pump inhibitors, the need for surgical management of peptic ulcer disease has greatly decreased.

Historically there are three main types of vagotomy techniques:

Truncal vagotomy (TV) consists of denervation of the main trunk divisions of the vagus; therefore, a pyloric drainage procedure, such as pyloromyotomy or pyloroplasty or gastrojejunostomy, is needed. This procedure also denervates the liver, biliary tree, pancreas, and small and large bowel, so is not free of side effects such as dumping syndrome, diarrhea, gastric reflux and a non-negligible ulcer recurrence rate (10%).

Trying to avoid these side effects, Griffith and Harkins [20] developed selective vagotomy which includes the division of the anterior and posterior gastric nerves of Latarjet only (after celiac/hepatic branches have been given off). It does not denervate the liver, biliary tree, pancreas, or small and large bowel but it does require drainage procedure. Selective vagotomy was compared with TV, and they have a similar rate of side effects.

Highly selective vagotomy, also called parietal cell vagotomy, includes denervation of the proximal two thirds of the stomach. This procedure preserves the innervation of the antrum and pylorus and the rest of the abdominal viscera; for these it causes minimal side effects is considered a safe procedure with a low mortality rate. It decreases acid secretion by approximately 65 to 75% which is comparable to TV. The incidence of dumping and diarrhea is insignificant, but although this is a safe procedure, it has a higher recurrence rate [21]. Today it is only indicated in a few cases, if any [22].

Vagotomy is sometimes combined with partial gastrectomy, usually antrectomy, to reduce the rate of recurrence.

Vagotomy and Drainage

Evacuation of solid components is slowed significantly after TV so a gastric drainage procedure must be associated. Gastrojejunostomy or, currently, pyloroplasty are the most used procedures. The most common type, Heineke-Mikulicz pyloroplasty, is performed by dividing the sphincter longitudinally and closing it transversely. Other techniques are the Finney and Jaboulay pyloroplasties, which basically consist of gastro-duodenostomies. All of these can be performed safely but they have a 10% recurrence rate and side effects (dumping syndrome, diarrhea..) in 10%. If an incomplete vagotomy is performed, then a marginal ulceration can appear.

This technique is currently indicated in emergencies (perforation, bleeding) requiring surgery [22] but uncommonly in elective surgery.

Vagotomy and antrectomy

As mentioned above, the addition of partial gastrectomy decreases the rate of ulcer recurrence (6-7, 9%) [23] since by removing the antrum, the number of gastrin-producing cells - as well as some acid-producing parietal cells - is reduced. This technique adds more morbidity and mortality rates, and it is more difficult to perform. The gastric remnant should not be too large because of the risk of marginal ulceration.

All of above described procedures, with similar results to open approach, can be safely performed through a laparoscopic approach, with some technical modifications.

To avoid TV, the Taylor procedure combines a section of the posterior vagus branch with anterior serosal myotomy [24]. In the Gómez-Ferrer technique, the posterior truncal vagotomy is combined with an anterior linear gastrectomy with a stapler [25]. Both procedures proved to be efficient in recurrence prevention and in providing satisfactory functional patient status. In case of stenosis, bilateral truncal vagotomy with stapled gastrojejunostomy can also be performed by laparoscopy [26].

Gastric Resection

In some cases, vagotomy is not sufficient and partial gastrectomy (e.g., antrectomy, subtotal gastrectomy) is necessary. The fundamentals in ulcer treatment are removal of the portion of the stomach containing the ulcer, the gastrin-producing cells that stimulate acid secretion, and most of the acid-producing parietal cells. Gastrectomies, however, are seldom indicated today in the treatment of peptic ulcer disease unless suspicion of malignancy exists. Technical aspects of gastrectomies are beyond the scope of this paper and can be found elsewhere [27].

Reconstruction is performed with gastroduodenostomy (Billroth I), gastrojejunostomy (Billroth II) or by means of a Roux-en-Y technique.

These techniques can also be performed by laparoscopy, since experience treating gastric cancer has shown its feasibility and, by comparison with open surgery, shorter in-hospital stays with similar morbidity and mortality rates [28].

The need of Hpy eradication - if present - after acid reducing surgery remains controversial, since ulcer recurrences can result from incomplete vagotomy or continuation on NSAID treatment [29]. The association of postoperative Hpy persistence with premalignant lesions [30] and the possibility of influencing ulcer recurrence make eradication advisable.

Choice of techniques for refractory ulcers:

Duodenal ulcer: highly-selective anterior vagotomy combined with posterior truncal vagotomy, or seromyotomy combined with TV offer the best combination of acid reduction and preservation of pyloric function. If some degree of stenosis is present, any type of pyloroplasty must be added [31].

Gastric ulcer: The treatment can differ according to the Johnson classification of gastric ulcers [32] :

- Type I (in the lesser curvature, associated with decreased acid secretion): distal gastrectomy provides a low recurrence rate (0-5%) [31]. Also, good results have been reported with HSV [33].
- Type II (gastric ulcer synchronous with scarring or ulceration in duodenum or pyloric channel): since these ulcers are associated with increased acid secretion, antrectomy with vagotomy is the preferred option.
- Type III (prepyloric ulcer): also associated with increased acid secretion; antrectomy and vagotomy are recommended. Outcomes after highly selective vagotomy have not, however, been as good as in other gastric ulcer types [34].
- Type IV (located high in the lesser curvature, associated with decreased acid secretion): subtotal gastrectomy is the procedure of choice.

MANAGEMENT OF COMPLICATIONS OF PEPTIC ULCER DISEASE

In general, urgent surgery is required in patients with uncontrolled hemorrhage and perforated ulcer with continuous leakage. The election of the technique depends on the ulcer location, the type of complication and the overall status of the patient.

Bleeding ulcer

Thirty percent of upper gastrointestinal hemorrhages are due to peptic ulcer disease, so peptic ulcer bleeding is a common condition. Also, it results in high morbidity and medical care cost.

Most patients can be managed with medical therapy including fluid and blood resuscitation, acid suppression and endoscopic techniques.

Early treatment with intravenous proton pump inhibitors has been shown to reduce rebleeding rates [35]. After endoscopic procedures it decreases the rate of active bleeding and the need of a new endoscopy. The avoidance of NSAIDs and smoking also helps decrease the rate of rebleeding.

Endoscopic treatment decreases rebleeding rates, the need of operation and reduces the mortality rate of the patients with bleeding peptic ulcers. The most effective and less risky endoscopic procedures are thermal contact coagulation and injection of epinephrine. They are initially successful in 75 to 90% of patients. The rebleeding rate is 10 to 30%, most of these episodes occurring within 96 hours. Some factors predict failure of endoscopic therapy [36], such as hypotension and ulcers larger than 2 cm.

Surgery for peptic ulcer hemorrhage is indicated in the following cases:

- (a) Failure of endoscopic techniques to control the hemorrhage
- (b) Hemodynamic instability despite strong resuscitation with uncontrolled bleeding
- (c) Recurrent hemorrhage after a second attempt at endoscopy: the second attempt has a reasonable chance of success and lower risk than surgery. However, if two attempts have failed, additional attempts are unlikely to be successful.

Other indications for early surgery include slow bleeding with transfusion requirements exceeding three units per day, rare blood types, refusal of transfusion, giant ulcers or co-morbid disease.

Angiography can be used in some cases [37]. Embolization of the bleeding vessel has been used in the treatment of patients with prohibitive risk at operation, rebleeding after operation and with uncertain endoscopic diagnosis. Walsh and colleagues published a series of 50 consecutive cases at the Cleveland Clinic. Angiography was successful in 52% of patients. In 17 cases, patients underwent operation as an attempt to control the bleeding [38]. Poultsides in his retrospective study, concluded that the failure of angioembolization can be predicted if it is performed late, following blood transfusion of more than 6 units, or for rebleeding from a previously suture-ligated duodenal ulcer [39].

When non-surgical therapy fails the surgeon should decide two main questions: when to operate and which operation to perform.

Surgical options for the bleeding peptic ulcer are three: oversewing alone or ligation of the bleeding point, oversewing /ligation with definitive non-resective ulcer operation - HSV or TV plus drainage - or gastric resection.

Bleeding Duodenal Ulcer

The number of operations for bleeding duodenal ulcer has decreased considerably due to effective endoscopic therapy and medication. Nowadays, between 5 and 10% of patients require operation. The most common techniques are vagotomy with drainage combined with oversewing of the ulcer, or vagotomy and antrectomy.

Millat and colleagues, and Hunt and McIntyre [40] compare patients undergoing operation for bleeding duodenal ulcer. In summary, they found no difference in mortality but the rates of rebleeding were lower in the resected groups than in the groups treated only with oversewing, even with associated vagotomy 37. It seems, however, that in those non-resective operations, if secure oversewing or ligation of the vessel is performed and proton pump inhibitors are added postoperatively, the risk of rebleeding is quite low. As a result, the current trend is to perform oversewing alone with Hpy eradication treatment and/or long-term proton pump inhibitors [37] [41].

In cases of patients with shock, in order to reduce operative time and mortality, non-resective procedures are recommended, performing only ligation/suture of the bleeding vessel.

When the bleeding control is very difficult, ligation of the gastroduodenal artery can be necessary.

Bleeding Gastric Ulcer

The behavior of the bleeding gastric ulcer is different; smaller submucosal vessels are involved, bleeding can stop spontaneously and in most cases the size of the bleeding vessels is less than 1 mm which explains why endoscopic therapy is effective in approximately 80% of cases.

A bleeding gastric ulcer is best treated by ulcer excision and repairing of the resulting defect [41]. The excision extent can vary from a wedge excision in the greater curvature to a partial gastrectomy if the ulcer is located at the lesser curvature. In the ulcers located near the gastroesophageal junction, biopsy and oversewing can be enough.

Distal gastric resection to include the bleeding ulcer has been associated with the lowest rebleeding rates [37]. A TV can be added if the patient is stable. Vagotomy and drainage with oversewing and biopsy or minor resection of the ulcer, followed by long-term acid suppression is a reasonable alternative to major operation [42].

Perforated ulcer

Perforation of a peptic ulcer is one of the most severe events since it is associated with considerable morbidity and mortality [43]. Less than one-third of patients have antecedents of peptic ulcer disease before perforation [44].

Duodenal

The perforation takes place in 5-10% of patients with chronic peptic ulcer disease. It is more common in men aged between 40 and 60 years. Upon diagnosis, prompt surgery must be performed, since this is currently the standard of care [41].

Surgical treatment can be limited to closure of the perforation, or a definitive treatment of ulcer disease can be performed. Simple closure is the treatment of choice in the management of perforated duodenal ulcers [41]. The main perforation closure techniques are simple suture, epiploplasty with omental pedicle flap epiploplasty or free omental plug –Graham patch- without suture of perforation edges and perforation suture covered by epiploplasty [45] [46].

Two randomized trials found that simple closure followed by Hpy treatment reduced recurrence rates of duodenal, prepyloric, and pyloric ulcers from 30-38% to 4.8-6.1% [47] [48]. Our group found in a prospective study with 92 patients having had perforated peptic ulcer, that simple closure followed by Hpy eradication is associated with a low rate of recurrence and no re-perforation in case of duodenal, pyloric, or pre-pyloric perforated ulcers, although this is not acceptable for perforated gastric ulcers [49]. Other studies found similar results [50] [51]. Importantly, successful postoperative eradication must be checked.

Simple closure can be performed laparoscopically by most surgeons, if properly trained. The procedure is usually performed with mortality or morbidity rates similar to or even lower than through an open approach [52] [53]. However, current evidence does not clearly support superiority of laparoscopy over the open approach [54]. Indeed, there are several factor associated with poor outcomes of the laparoscopic approach – shock, delayed presentation, advanced age, ASA III-IV – which, if present, should guide towards laparotomy [46].

On the other hand, definitive treatment (partial gastrectomy or vagotomy) is associated with diarrhea, disturbing in some cases. Trying to avoid this adverse effect, highly selected vagotomy techniques were developed, but the technical difficulties of these methods make them hard to apply in cases of emergency.

Although conclusive evidence is lacking, if continuation of NSAIDs is predictable - e.g. in case of chronic arthritis -, a surgical procedure to decrease acid secretion may be necessary. Conservative treatment of the duodenal perforation has been described in the literature (Taylor's method). It consists of measures such as nasogastric aspiration, proton pump inhibitors or other acid-suppressives, antibiotics and close monitoring. A few patients could benefit from this conservative treatment: those patients who are fasting at the perforation moment, with abdominal pain for less than 24 hours, and those showing clinical improvement with this approach (decrease of the pain and no other clinical abnormalities). However, this method has a high rate of failure (16-28%), morbidity and a non-negligible mortality (5-8%). A randomized trial from 1989 [55] compared conservative treatment with surgical perforation repair and found a 28% failure rate in the conservative approach. Yet the mortality rate was 5% in both groups with a non-significant difference in morbidity. The worst outcomes were for patients over 70 years. Two perforations were due to carcinoma.

A more recent paper [56] prospectively studied conservative management with surgery only in case of failure of improvement after 24 hours. It was found that more than 50% of patients

responded to conservative treatment and that the association of some criteria (size of pneumoperitoneum larger than the size of the first lumbar vertebra, heart beat over 94 bpm, pain at digital rectal exam and age) made emergency surgery necessary.

Percutaneous drainage associated to conservative treatment has also been reported [57].

The difficulty to select patients who will improve without surgery and the possibility that the perforation is not from a peptic ulcer make, in most cases, early surgical intervention necessary.

Gastric ulcer

Patients having a perforated gastric ulcer are usually older and tend to have associated diseases leading to a poorer prognosis. As a result, overall mortality rate ranges from 10-40% [57] [58]. Since the risk of occult malignancy is always present, the theoretical procedure of choice is a partial gastrectomy, including the ulcer. However, the usual scenario is the case of a patient in shock, dehydrated or with a poor general condition due to co-morbidity. In these cases, biopsy of the ulcer margins and simple closure is an acceptable procedure [45]. This approach is, nevertheless, associated with high recurrence rates, even with reoperation [49]. After recovery, proper study to rule out any occult carcinoma must be performed. A recent paper reported non-operative treatment of gastric ulcer, which was successful in 78% [60]. In the group for emergency surgery due to unsuccessful conservative treatment plus the patients primarily operated on, gastric-preserving surgery –simple closure or minor resections- had no different complication rate or hospital stay compared with major gastric resection. Concerning long-term outcomes of patients not treated by gastrectomy, 4% had ulcer recurrence after a mean follow-up of 36 months [60]. In this paper, the Japanese guidelines regarding patient selection for conservative treatment for PGU are reported: within 24 h of onset; stable condition without severe comorbidities; signs of peritoneal irritation localized within the upper abdomen; and small amount of ascites.

In the cases where the perforation clearly is located in a tumoral area, the options are emergency gastrectomy –doubtfully oncologic- or simple closure and to perform oncologic gastrectomy at a later stage [45].

CONCLUSION

In summary, to choose the best operation for peptic ulcer disease it is necessary to consider the features and the clinical status of the patient, the surgeon's experience and the published evidence [61]. The main recommendations are summarized in Table 2.

Refractory ulcer*	
Duodenal	Highly-selective anterior vagotomy combined with posterior truncal vagotomy or seromyotomy combined with posterior truncal vagotomy
Gastric	Type I: Distal gastrectomy Type II: Antrectomy with vagotomy Type III: Antrectomy with vagotomy Type IV: Subtotal gastrectomy
Uncertain diagnosis*	The same than in case of refractory ulcer
Gastric outlet obstruction*	Highly-selective vagotomy with gastrojejunostomy (if balloon dilatation fails)
Bleeding duodenal ulcer	Partial gastrectomy (less rebleeding rate) Suture oversewing (less long-term side effects)
Bleeding gastric ulcer	Ulcer excision (variable from wedge excision to partial gastrectomy)
Perforated duodenal ulcer	Simple closure Acid secretion reduction procedure if continuation of NSAIDs is predictable
Perforated gastric ulcer	Partial gastrectomy Biopsy and simple closure in case of patient poor condition

Table 2. Indications for surgery and recommended techniques. *After excluding diseases other than peptic ulcer

References

1. Sarosi G, Jaiswal K, Nwariaku F, Asolati M, Fleming J, Anthony T. Surgical therapy of peptic ulcers in the 21st century: more common than you think. *Am J Surg*. 2005;190:775-9 [pubmed](#)
2. Louw J. Peptic ulcer disease. *Curr Opin Gastroenterol*. 2006;22:607-11 [pubmed](#)
3. Chan F, Leung W. Peptic-ulcer disease. *Lancet*. 2002;360:933-41 [pubmed](#)
4. Yuan Y, Padol I, Hunt R. Peptic ulcer disease today. *Nat Clin Pract Gastroenterol Hepatol*. 2006;3:80-9 [pubmed](#)
5. Annibale B, De Magistris L, Corleto V, D'Ambra G, Marignani M, Iannoni C, *et al*. Zollinger-Ellison syndrome and antral G-cell hyperfunction in patients with resistant duodenal ulcer disease. *Aliment Pharmacol Ther*. 1994;8:87-93 [pubmed](#)
6. Stollman N, Metz D. Pathophysiology and prophylaxis of stress ulcer in intensive care unit patients. *J Crit Care*. 2005;20:35-45 [pubmed](#)
7. Liang M, Marks J, Berman R, Carter J. Management of complicated peptic ulcer disease. *Arch Surg*. 2005;140:914-5; author reply 915 [pubmed](#)
8. Banerjee S, Cash B, Dominitz J, Baron T, Anderson M, Ben-Menachem T, *et al*. The role of endoscopy in the management of patients with peptic ulcer disease. *Gastrointest Endosc*. 2010;71:663-8 [pubmed](#)
9. Bardhan K, Nayyar A, Royston C. History in our lifetime: the changing nature of refractory duodenal ulcer in the era of histamine H2 receptor antagonists. *Dig Liver Dis*. 2003;35:529-36 [pubmed](#)
10. Cohen H, Laine L. Endoscopic methods for the diagnosis of *Helicobacter pylori*. *Aliment Pharmacol Ther*. 1997;11:3-9 [pubmed](#)
11. Malfertheiner P, Megraud F, O'Morain C, Bazzoli F, El-Omar E, Graham D, *et al*. Current concepts in the management of *Helicobacter pylori* infection: the Maastricht III Consensus Report. *Gut*. 2007;56:772-81 [pubmed](#)
12. McColl K. How I manage *H. pylori*-negative, NSAID/aspirin-negative peptic ulcers. *Am J Gastroenterol*. 2009;104:190-3 [pubmed](#) [publisher](#)
13. Berna M, Hoffmann K, Serrano J, Gibril F, Jensen R. Serum gastrin in Zollinger-Ellison syndrome: I. Prospective study of fasting serum gastrin in 309 patients from the National Institutes of Health and comparison with 2229 cases from the literature. *Medicine (Baltimore)*. 2006;85:295-330 [pubmed](#)
14. Tomassetti P, Migliori M, Simoni P, Casadei R, De Iasio R, Corinaldesi R, *et al*. Diagnostic value of plasma chromogranin A in neuroendocrine tumours. *Eur J Gastroenterol Hepatol*. 2001;13:55-8 [pubmed](#)
15. Zittel T, Jehle E, Becker H. Surgical management of peptic ulcer disease today--indication, technique and outcome. *Langenbecks Arch Surg*. 2000;385:84-96 [pubmed](#)
16. Cherian P, Cherian S, Singh P. Long-term follow-up of patients with gastric outlet obstruction related to peptic ulcer disease treated with endoscopic balloon dilatation and drug therapy. *Gastrointest Endosc*. 2007;66:491-7 [pubmed](#)
17. Lau J, Chung S, Sung J, Chan A, Ng E, Suen R, *et al*. Through-the-scope balloon dilation for pyloric stenosis: long-term results. *Gastrointest Endosc*. 1996;43:98-101 [pubmed](#)
18. Wadhwa R, Kozarek R, France R, Brandabur J, Gluck M, Low D, *et al*. Use of self-expandable metallic stents in benign GI diseases. *Gastrointest Endosc*. 2003;58:207-12 [pubmed](#)
19. Csendes A, Maluenda F, Braghetto I, Schutte H, Burdiles P, Diaz J. Prospective randomized study comparing three surgical techniques for the treatment of gastric outlet obstruction secondary to duodenal ulcer. *Am J Surg*. 1993;166:45-9 [pubmed](#)
20. Griffith C, Harkins H. Selective gastric vagotomy: physiologic basis and technique. *Surg Clin North Am*. 1962;42:1431-41 [pubmed](#)
21. Maddern G, Vauthey J, Devitt P, Britten Jones R, Hetzel D, Jamieson G. Recurrent peptic ulceration after highly selective vagotomy: long-term outcome. *Br J Surg*. 1991;78:940-1 [pubmed](#)
22. Lagoo J, Pappas T, Perez A. A relic or still relevant: the narrowing role for vagotomy in the treatment of peptic ulcer disease. *Am J Surg*. 2014;207:120-6 [pubmed](#) [publisher](#)
23. Heberer G, Teichmann R. Recurrence after proximal gastric vagotomy for gastric, pyloric, and prepyloric ulcers. *World J Surg*. 1987;11:283-8 [pubmed](#)
24. Taylor T, Lythgoe J, McFarland J, Gilmore I, Thomas P, Ferguson G. Anterior lesser curve seromyotomy and posterior truncal vagotomy versus truncal vagotomy and pyloroplasty in the treatment of chronic duodenal ulcer. *Br J Surg*. 1990;77:1007-9 [pubmed](#)
25. Gomez-Ferrer F, Ballyque J, Azagra S, Bycha-Castelo H, Castro-Sousa F, Espalyeu P, *et al*. Laparoscopic surgery for duodenal ulcer: first results of a multicenter study applying a personal procedure. *Hepatogastroenterology*. 1999;46:1517-21 [pubmed](#)
26. Palanivelu C, Jani K, Rajan P, Kumar K, Madhankumar M, Kavalakat A. Laparoscopic management of acid peptic disease. *Surg Laparosc Endosc Percutan Tech*. 2006;16:312-6 [pubmed](#)
27. MUTTER II: Mutter D, Marescaux J. *Gastrectomies pour lésions bénignes*. EMC (Elsevier Masson SAS, Paris) Techniques chirurgicales – Appareil digestif, 40-320, 2007.
28. Kim E, Seo K, Yoon K. Laparoscopy-assisted distal gastrectomy for early gastric cancer in the elderly. *J Gastric Cancer*. 2012;12:232-6 [pubmed](#) [publisher](#)
29. Turnage R, Sarosi G, Cryer B, Spechler S, Peterson W, Feldman M. Evaluation and management of patients with recurrent peptic ulcer disease after acid-reducing operations: a systematic review. *J Gastrointest Surg*. 2003;7:606-26 [pubmed](#)

30. Giuliani A, Galati G, Demoro M, Scimò M, Pecorella I, Basso L. Screening of *Helicobacter pylori* infection after gastrectomy for cancer or peptic ulcer: results of a cohort study. *Arch Surg.* 2010;145:962-7 [pubmed](#) [publisher](#)
31. Harbison S, Dempsey D. Peptic ulcer disease. *Curr Probl Surg.* 2005;42:346-454 [pubmed](#)
32. Johnson H. Gastric ulcer: classification, blood group characteristics, secretion patterns and pathogenesis. *Ann Surg.* 1965;162:996-1004 [pubmed](#)
33. Tekant Y, Goh P, Low C, Ngoi S. Pyloric channel ulcers: management and three-year follow-up. *Am Surg.* 1995;61:237-9 [pubmed](#)
34. Jordan P. Type I gastric ulcer treated by parietal cell vagotomy and mucosal ulcerectomy. *J Am Coll Surg.* 1996;182:388-93 [pubmed](#)
35. Leontiadis G, Sharma V, Howden C. Systematic review and meta-analysis of proton pump inhibitor therapy in peptic ulcer bleeding. *BMJ.* 2005;330:568 [pubmed](#)
36. Brullet E, Calvet X, Campo R, Rue M, Catot L, Donoso L. Factors predicting failure of endoscopic injection therapy in bleeding duodenal ulcer. *Gastrointest Endosc.* 1996;43:111-6 [pubmed](#)
37. Lu Y, Loffroy R, Lau J, Barkun A. Multidisciplinary management strategies for acute non-variceal upper gastrointestinal bleeding. *Br J Surg.* 2014;101:e34-50 [pubmed](#) [publisher](#)
38. Walsh R, Anain P, Geisinger M, Vogt D, Mayes J, Grundfest-Broniatowski S, *et al.* Role of angiography and embolization for massive gastroduodenal hemorrhage. *J Gastrointest Surg.* 1999;3:61-5; discussion 66 [pubmed](#)
39. Poultsides G, Kim C, Orlando R, Peros G, Hallisey M, Vignati P. Angiographic embolization for gastroduodenal hemorrhage: safety, efficacy, and predictors of outcome. *Arch Surg.* 2008;143:457-61 [pubmed](#) [publisher](#)
40. Hunt P, McIntyre R. Choice of emergency operative procedure for bleeding duodenal ulcer. *Br J Surg.* 1990;77:1004-6 [pubmed](#)
41. Lee C, Sarosi G. Emergency ulcer surgery. *Surg Clin North Am.* 2011;91:1001-13 [pubmed](#) [publisher](#)
42. Nguyen L, Brunicardi F, Dibardino D, Scott B, Awad S, Bush R, *et al.* Education of the modern surgical resident: novel approaches to learning in the era of the 80-hour workweek. *World J Surg.* 2006;30:1120-7 [pubmed](#)
43. Nogueira C, Silva A, Santos J, Silva A, Ferreira J, Matos E, *et al.* Perforated peptic ulcer: main factors of morbidity and mortality. *World J Surg.* 2003;27:782-7 [pubmed](#)
44. Bertleff M, Lange J. Perforated peptic ulcer disease: a review of history and treatment. *Dig Surg.* 2010;27:161-9 [pubmed](#) [publisher](#)
45. Mutter D, Marescaux J. Traitement chirurgical des complications des ulcers gastroduodénaux. EMC (Elsevier Masson SAS, Paris) Techniques chirurgicales – Appareil digestif, 40-326, 2007.
46. Søreide K, Thorsen K, Søreide J. Strategies to improve the outcome of emergency surgery for perforated peptic ulcer. *Br J Surg.* 2014;101:e51-64 [pubmed](#) [publisher](#)
47. Ng E, Lam Y, Sung J, Yung M, To K, Chan A, *et al.* Eradication of *Helicobacter pylori* prevents recurrence of ulcer after simple closure of duodenal ulcer perforation: randomized controlled trial. *Ann Surg.* 2000;231:153-8 [pubmed](#)
48. El-Nakeeb A, Fikry A, Abd El-Hamed T, Fouda E, El Awady S, Youssef T, *et al.* Effect of *Helicobacter pylori* eradication on ulcer recurrence after simple closure of perforated duodenal ulcer. *Int J Surg.* 2009;7:126-9 [pubmed](#) [publisher](#)
49. Rodríguez-Sanjuán J, Fernández-Santiago R, García R, Trageda S, Seco I, la de Torre F, *et al.* Perforated peptic ulcer treated by simple closure and *Helicobacter pylori* eradication. *World J Surg.* 2005;29:849-52 [pubmed](#)
50. Bose A, Kate V, Ananthkrishnan N, Parija S. *Helicobacter pylori* eradication prevents recurrence after simple closure of perforated duodenal ulcer. *J Gastroenterol Hepatol.* 2007;22:345-8 [pubmed](#)
51. Yetkin G, Uludag M, Akgun I, Citgez B, Karakoc S. Late results of a simple closure technique and *Helicobacter pylori* eradication in duodenal ulcer perforation. *Acta Chir Belg.* 2010;110:537-42 [pubmed](#)
52. Kuwabara K, Matsuda S, Fushimi K, Ishikawa K, Horiguchi H, Fujimori K. Community-based evaluation of laparoscopic versus open simple closure of perforated peptic ulcers. *World J Surg.* 2011;35:2485-92 [pubmed](#) [publisher](#)
53. Sanabria A, Villegas M, Morales Uribe C. Laparoscopic repair for perforated peptic ulcer disease. *Cochrane Database Syst Rev.* 2013;2:CD004778 [pubmed](#) [publisher](#)
54. Lunevicius R, Morkevicius M. Management strategies, early results, benefits, and risk factors of laparoscopic repair of perforated peptic ulcer. *World J Surg.* 2005;29:1299-310 [pubmed](#)
55. Crofts T, Park K, Steele R, Chung S, Li A. A randomized trial of nonoperative treatment for perforated peptic ulcer. *N Engl J Med.* 1989;320:970-3 [pubmed](#)
56. Songne B, Jean F, Foulatier O, Khalil H, Scotte M. [Non operative treatment for perforated peptic ulcer: results of a prospective study]. *Ann Chir.* 2004;129:578-82 [pubmed](#)
57. Saber A, Gad M, Ellabban G. Perforated duodenal ulcer in high risk patients: is percutaneous drainage justified?. *N Am J Med Sci.* 2012;4:35-9 [pubmed](#)
58. Jordan P. Surgery for peptic ulcer disease. *Curr Probl Surg.* 1991;28:265-330 [pubmed](#)
59. Hodnett R, Gonzalez F, Lee W, Nance F, Deboisblanc R. The need for definitive therapy in the management of perforated gastric ulcers. Review of 202 cases. *Ann Surg.* 1989;209:36-9 [pubmed](#)
60. Tanaka R, Kosugi S, Sakamoto K, Yajima K, Ishikawa T, Kanda T, *et al.* Treatment for perforated gastric ulcer: a multi-institutional retrospective review. *J Gastrointest Surg.* 2013;17:2074-81 [pubmed](#) [publisher](#)

61. Jamieson G. Current status of indications for surgery in peptic ulcer disease. World J Surg.
2000;24:256-8 [pubmed](#)
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