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Surgical management of peptic ulcer disease today – indication, technique and outcome

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Abstract *Aims:* The current surgical management of peptic ulcer disease and its outcome have been reviewed. *Results:* Today, surgery for peptic ulcer disease is largely restricted to the treatment of complications. In peptic ulcer perforation, a conservative treatment trial can be given in selected cases. If laparotomy is necessary, simple closure is sufficient in the large majority of cases, and definitive ulcer surgery to reduce gastric acid secretion is no longer justified in these patients. Laparoscopic surgery for perforated peptic ulcer has failed to prove to be a significant advantage over open surgery. In bleeding peptic ulcers, definitive hemostasis can be achieved by endoscopic treatment in more than 90% of cases. In 1–2% of cases, immediate emergency surgery is necessary. Some ulcers have a high risk of re-bleeding, and early elective surgery might be advisable. Surgical bleeding control can be achieved by direct suture and extraluminal ligation of the gastroduodenal artery or by gastric resection. Benign gastric outlet obstruction can be controlled by endoscopic balloon dilatation in 70% of cases, but gastrojejunostomy or gastric resection are necessary in about 30% of cases. *Conclusions:* Elective surgery for peptic ulcer disease has been largely abandoned, and bleeding or obstructing ulcers can be managed safely by endoscopic treatment in most cases. However, surgeons will continue to encounter patients with peptic ulcer disease for emergency surgery. Currently, laparoscopic surgery has no proven advantage in peptic ulcer surgery.

Key words Peptic ulcer disease · Perforation · Bleeding · Obstruction · Laparoscopic surgery

Introduction

Over the past two decades, treatment of peptic ulcer disease has changed dramatically. The first major change occurred after the introduction of histamine H_2 receptor antagonists for gastric acid suppression at the end of the 1970s, followed by proton-pump inhibitors at the end of the 1980s [1, 2, 3]. In addition, the pathophysiological understanding of peptic ulcer disease was completely changed by the discovery that an infective agent, *Helicobacter pylori*, is present in 75–85% of the patients [4, 5, 6]. Today, *H. pylori* is regarded as a causative factor for the majority of duodenal and gastric ulcers encountered in routine clinical practice [2]. *H. pylori* infection and the accompanying inflammation disrupts the inhibitory control of gastrin release by decreasing antral somatostatin, and this is more marked if the infecting organism is a *cag*A-positive strain. The resulting increase in gastrin release and gastric acid secretion is a key mechanism by which the infection induces peptic ulcer disease [7]. About 15–20% of peptic ulcers are related to the intake of non-steroidal anti-inflammatory drugs (NSAIDs).

Rare causes include hypersecretory states like the Zollinger-Ellison syndrome and a variety of uncommon forms of peptic ulcer disease [2, 5, 6].

In the 1960s and 1970s, a large proportion of patients with recurrent peptic ulcer disease were offered a variety of operations as a definitive treatment to reduce gastric acid secretion, mainly highly selective vagotomy with or without antrectomy for duodenal ulcers or gastric resections for gastric ulcers [1, 3, 8]. Although about 85–95% of patients were cured from symptoms of peptic ulcer disease [3, 9, 10, 11], patients had to endure an operation with its inherent risks, and some suffered from postvagotomy or postgastrectomy syndromes [3, 9, 10, 11, 12]. In addition, gastric resection carries an increased long-term risk of gastrointestinal cancers [13, 14].

Today, peptic ulcer disease is mainly looked upon as a disease treated conservatively with H_2 receptor antagonists or proton-pump inhibitors; in the case of *H. pylori* infection, in combination with an antibiotic course for 1 week. Although the problem of antibiotic resistance of *H. pylori* is increasing, combination therapies such as metronidazole with clindamycin or metronidazole with tetracycline can achieve eradication rates of 80% or more [15, 16, 17, 18]. *H. pylori* eradication results in a permanent cure from peptic ulcer disease in most patients, as the re-infection rate seems to be very low [15, 19].

Elective peptic ulcer surgery has been virtually abandoned. In the 1980s, the number of elective operations for peptic ulcer disease dropped by more than 70%, and emergency operations accounted for more than 80% [20]. Today, surgery remains mainly reserved for complications of peptic ulcer disease, which include bleeding, perforation, and gastric outlet obstruction.

The incidence of peptic ulcer disease has been estimated as 1500–3000 per 100,000 inhabitants per year [2]. The lifetime prevalences of hemorrhage, perforation, and obstruction in peptic ulcer patients were estimated to be 15–20%, 5%, and 2%, respectively [21]. Upper gastrointestinal hemorrhage occurs with an estimated incidence of about 100 per 100,000 inhabitants per year [22, 23], half of which are due to peptic ulcer bleeding [21, 24, 25]. Three to four per 100,000 inhabitants per year are operated on due to peptic ulcer bleeding [1], three to ten per 100,000 inhabitants per year due to peptic ulcer perforation [1, 26, 27, 28, 29], and one to three per 100,000 inhabitants per year due to gastric outlet obstruction [1, 30]. The annual incidence of emergency surgery for peptic ulcers and the mortality of peptic ulcer disease have not decreased after the introduction of H_2 receptor antagonists [1, 20, 31, 32, 33], and peptic ulcers are responsible for about 20,000–30,000 deaths per year in Europe [34]. Only recently, possibly due to proton-pump inhibitors and *H. pylori* eradication strategies, is there evidence that the number of emergency operations for peptic ulcer disease is decreasing [29].

A considerable number of patients still has to be operated on for peptic ulcer disease even today. Surgeons will continue to encounter patients with peptic ulcer disease in the operating room, with the questions arising whether the current understanding of peptic ulcer disease or new laparoscopic techniques have changed the surgical management of peptic ulcer disease. In the following sections, we will give an overview of current trends in the surgical management of peptic ulcer disease.

Surgical management of recurrent peptic ulcer disease

The reduction of elective surgery for peptic ulcer disease after the extensive use of potent gastric acid suppressive drugs has been stopped in some centers by the introduction of laparoscopic techniques for surgical treatment of duodenal ulcers [35, 36]. In a multicenter study, the outcome of 136 patients treated by laparoscopic posterior vagotomy and anterior linear gastrectomy along the lesser curve of the stomach in order to cut the gastric vagus branches was investigated. The term “anterior linear gastrectomy” is somewhat misleading; it is supposed to describe the cutting and stapling along the lesser curvature of the stomach with an endo-stapling device. The mean operating time was 65 min, the mortality zero, the perioperative morbidity 3%, and the mean hospital stay 3 days. Gastric acid secretion was reduced by about 80%, and only one patient had an asymptomatic ulcer recurrence during a mean follow-up of 2 years. Four patients had a reduced quality of life as evaluated by the Visick score (Visick grade III or IV). The results of this procedure, which is relatively easy to perform, seem to be quite favorable. However, the indication for surgery was not clearly defined, and no data regarding *H. pylori* infection were given [36].

In about 10–20% of patients, *H. pylori* eradication fails [15, 16, 17], and in few patients, a peptic ulcer recurs despite *H. pylori* eradication [15]. Some of them are young and do not want to stay on life-long acid-suppressing medication. In these cases, laparoscopic surgery to reduce gastric acid secretion might offer an alternative treatment. However, as long as the indications for elective laparoscopic surgery and the long-term results are not defined, a general recommendation for this procedure cannot be given at the moment.

Surgical management of complicated peptic ulcer disease

As already mentioned above, today, surgery for peptic ulcer disease is mainly reserved for complications of peptic ulcer disease. About two-thirds of operations for complicated peptic ulcer disease are due to perforations

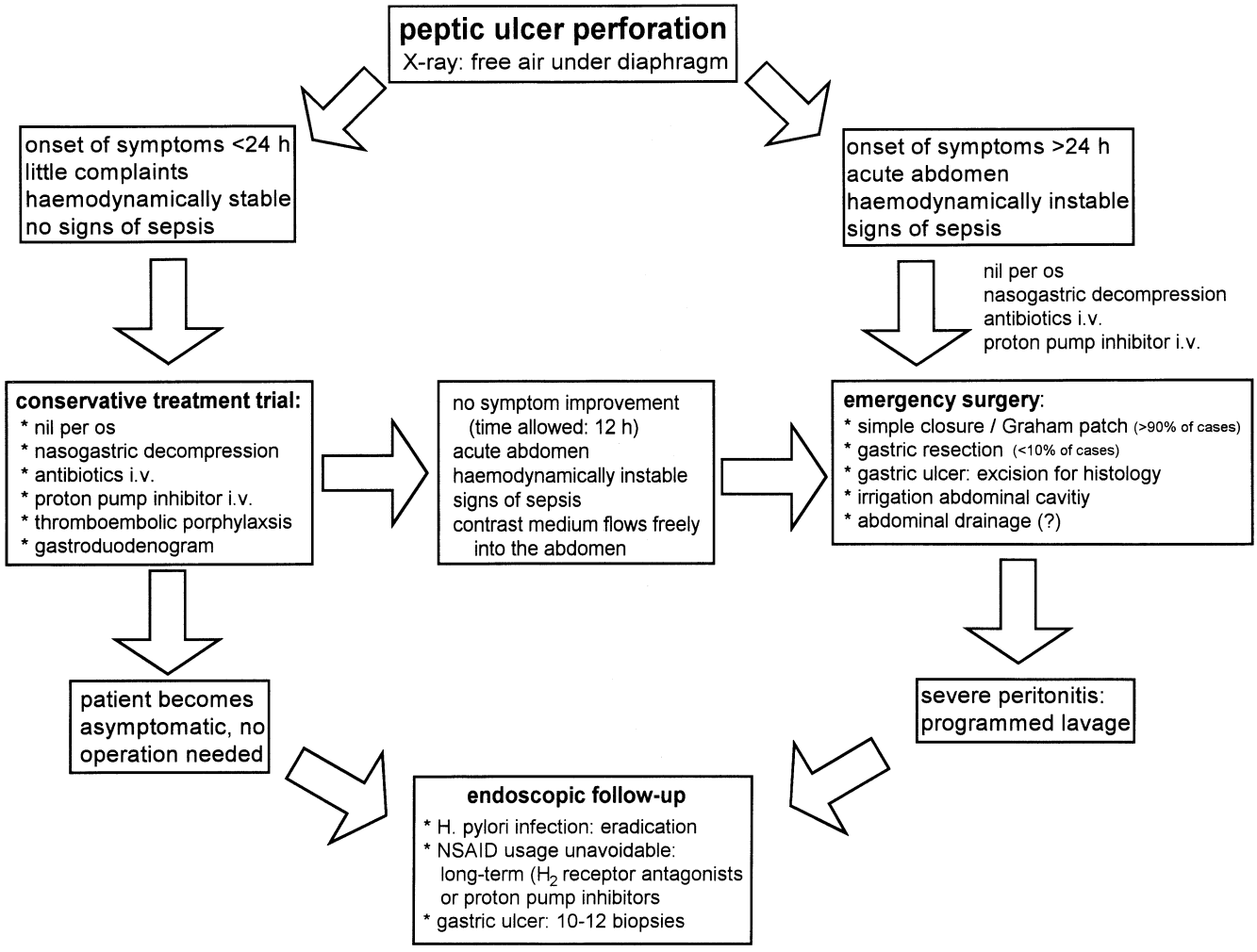

Fig. 1 Perforated peptic ulcer treatment strategy. By reviewing the current literature, a flow chart was developed (details and references see text)

with ensuing peritonitis. About one-third of operations are necessary to stop peptic ulcer bleeding [37], despite endoscopic treatment options like injection therapy with adrenaline, polidocanol or fibrin glue, or coagulation therapy with heater probes or argon plasma coagulation [25, 38, 39]. In rare cases, peptic ulcer penetration requires surgery [29, 37]. In addition to the emergency cases of perforation and bleeding, 1–2% of patients with peptic ulcer disease might require surgery for gastric outlet obstruction as a sequelae of recurrent peripyloric ulcers with scarring and narrowing of the pylorus [40].

Surgical management of peptic ulcer perforation

Indication

There is an ongoing debate whether perforated peptic ulcers generally need to be operated on or not. It has been estimated that about half of the perforations seal by themselves [41], and a prospective trial comparing conservative treatment with surgical treatment in perforated peptic ulcer disease has shown no advantage of surgical treatment with regard to morbidity and mortality [42]. The results of this trial have been confirmed by the evaluation of a protocol for the non-operative management of perforated peptic ulcer in a general hospital over several years. Possibly, conservative treatment can be safely tried in about two-thirds of patients [43]. However, it has been shown that delaying the time point of an operation beyond 12 h after the onset of clinical symptoms will worsen the outcome in peptic ulcer perforation [44, 45, 46]. Therefore, it is crucial not to pass the time when a laparotomy is definitely indicated [41, 42, 44, 46].

In cases in which the patient has only little discomfort and is hemodynamically stable, conservative treatment can be tried under strict clinical surveillance of a senior surgeon. With nasogastric decompression, substitution of fluids and electrolytes, a proton-pump inhibitor, thromboembolic prophylaxis and antibiotic treatment, the patients' symptoms should improve within 12 h [42,

43]. The diagnostic procedure of a gastroduodenogram with a water-soluble contrast medium should be added. In most of these cases, the leakage, proven by air under the diaphragm, has already been sealed, and surgery remains unnecessary. If abdominal tenderness increases, the patient becomes hemodynamically unstable, or the contrast medium freely flows into the abdomen, then laparotomy is indicated to irrigate the abdomen and close the leakage [41, 42, 43] (Fig. 1).

In cases in which the patient presents more than 24 h after the onset of symptoms or shows the clinical signs of an acute abdomen, hypotension, or sepsis, laparotomy should be performed immediately [41, 43]. Patients 70 years or older are less likely to respond to conservative treatment [42]; these patients might profit from an early decision for surgery [43]. Drains are usually placed along the Morrison pouch and in the pelvis, although its use has been questioned recently [47]. Postoperatively, the surgeon has to decide whether a programmed lavage is needed to treat peritonitis.

In about 3% of patients with peptic ulcer perforation, the ulcer penetrates into an adjacent anatomic structure, namely the pancreas, the gallbladder, or the liver [29, 37]. As these cases are rare, no general recommendations about indication for surgery and surgical technique can be given, and no reports on outcome in significant patient populations are available. In general, problems like pancreatitis or biliary peritonitis might arise, requiring surgical treatment. However, each case must be handled separately, and surgery is not necessary in all cases.

Technique

Perforated peptic ulcers are mainly located in the first part of the duodenum, accounting for about 35–65% of cases. The pylorus harbors about 25–45% and the stomach about 5–25% of perforated peptic ulcers [29, 37, 48, 49]. In up to 90% of the cases, simple closure with or without an omental patch (Graham patch) [50] is sufficient to treat peptic ulcer perforations [28, 29, 37, 46, 48]. Definitive ulcer surgery, performed in the past decades at the time of perforation in most of the patients [51], is no longer needed, as ulcer recurrence rates have dropped dramatically due to *H. pylori* eradication and acid suppressing drugs [2, 15, 19, 28].

In the stomach, the ulcer should be excised to obtain tissue for histological investigation, as gastric cancer occasionally presents as perforated gastric ulcer [42, 43, 45, 52]. Gastric resection, mainly Billroth-II resection, either with or without vagotomy, is needed in less than 10% of cases [28, 29, 37, 46, 48], but has been performed with a frequency of up to about 25% of cases in previous studies [51, 53]. In Japan, high rates of gastric resection (in more than 80% of cases) have been reported, while simple closure was performed in less than 5% of cases. Since no explanation has been provided, the reason for this high rate of resections is unclear, but the outcome in these patients was similar to that reported from Western countries [33, 45]. As most patients with perforated peptic ulcer disease do not need a definite operation to reduce gastric acid secretion [54, 55], this approach is unjustified in our opinion.

Since the first reports in 1990 [56, 57], laparoscopic surgery has been used in a variety of institutions to treat peptic ulcer perforations. The technique itself seems to be feasible in most of the cases, the conversion rate to open surgery was 0–25% [52, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69]. All studies showed an increased operation time compared with open surgery [60, 61, 66, 68], and operation time was even doubled in some reports [58, 59, 63, 69]. The need for postoperative analgesic treatment was reduced in several studies [58, 60, 61, 66, 67, 68], while others noted no difference [59, 62, 69]. Only one study reported a reduced time to return to a normal diet, a shorter hospital stay and an earlier return to work [67]. No differences regarding blood loss, the stress response as determined by endotoxemia, bacteremia and inflammatory markers, the need for nasogastric decompression, postoperative gastric emptying, the start of oral food intake, the length of hospital stay, or the return to normal activities were observed in a variety of studies [58, 59, 60, 61, 62, 63, 66, 68, 69]. Morbidity and mortality were comparable between laparoscopic and open surgery [58, 59, 61, 66, 67].

There is only one prospective randomized trial comparing laparoscopic surgery with open surgery for perforated peptic ulcer; in this study, no differences in most of the criteria mentioned above could be found between laparoscopic and open surgery. Only the need for pain medication was decreased and the operation time was increased in the laparoscopic treatment group. The authors stated that “the overall advantages of laparoscopic over open repair are minor, and laparoscopic repair has the main disadvantage of long operation time” [58].

Very large perforations, a posterior location of the perforation, or a poor general state of health are considered to be contraindications of laparoscopic therapy for peptic ulcer perforations [52, 58, 62, 68]. A further limitation of the laparoscopic approach might be higher costs, as laparoscopic surgery might be more expensive than open surgery. However, no cost analysis has been done in the studies published so far. Taken together, there is currently no evidence that laparoscopic repair improves the results of surgical treatment of perforated peptic ulcer disease.

Outcome

Conservative treatment might be possible in about 60% of cases, while about 30% of patients with perforated ul-

Table 1 Mortality risk factors in perforated peptic ulcer

		Mortality risk increase	Reference
Delay to laparotomy	>6 h vs <6 h	5-fold	[28]
	>12 h vs <12 h	4-fold	[44]
	>12 h vs <12 h	6-fold	[45]
	>12 h vs <12 h	3.5-fold	[46]
	>24 h vs <24 h	9-fold	[44]
Age	>50 years vs <50 years	6-fold	[44]
	>50 years vs <50 years	16-fold	[45]
	>70 years vs <70 years	2.5-fold	[69]
	>70 years vs <70 years	4-fold	[45, 46]
	>70 years vs <70 years	10-fold	[29]
	80+ years vs 70–79 years	2-fold	[28]
Concurrent medical illness		3-fold	[46]
Ulcer location	Gastric vs duodenal	2-fold	[28, 37, 45, 48]
	Gastric vs duodenal	2.5-fold	[44]
	Gastric vs duodenal	4-fold	[46]
Shock on admission		?[a] (3 of 5 patients died)	[45]
Renal failure		?[a] (3 of 4 patients died)	[45]
Liver cirrhosis		?[a] (2 of 3 patients died)	[45]
Immunocompromised state		?[a] (2 of 2 patients died)	[45]

[a] Low patient numbers do not allow risk calculation

cers go to the operating room immediately. In about 10% of cases, conservative treatment fails and patients are proceeded to surgery secondarily [43]. Morbidity and mortality were comparable between non-operatively and operatively managed perforated ulcer patients [42, 43]. However, in clinical practice, adherence to protocol guidelines regarding conservative treatment was poor. The reason might have been that non-operative treatment of perforated peptic ulcer remains controversial and has not been widely adapted. Further, the lack of opportunity for performing definitive ulcer surgery and an individual surgeon's preference for surgical treatment might have contributed to protocol violations [43]. In any perforated peptic ulcer patient treated successfully by non-operative management, endoscopic follow-up is mandatory to test for *H. pylori* infection and to exclude gastric malignancy [43].

Emergency operations for peptic ulcer perforation carry a mortality risk of 6–30% [20, 28, 29, 37, 44, 45, 48, 53, 69]. A variety of factors have been identified to adversely affect outcome. Shock on admission, renal failure, delaying the time point of an operation beyond 12 h, concurrent medical illness, age, liver cirrhosis, and a immunocompromised state have all been identified as risk factors (Table 1) [20, 28, 29, 37, 44, 45, 46, 53, 69]. Delaying the time point of an operation by more than 12 h and 24 h after the onset of symptoms increased mortality by about threefold and ninefold, respectively [44, 46]. Concurrent medical illness, mainly of cardiovascular or pulmonary origin or diabetes mellitus, is present in about 40–60% of patients with perforated peptic ulcer [29, 42, 46] and entails a mortality of up to 50% [37]. Age above 70 years dramatically increases mortality [29, 37, 69]; this is particularly relevant since the majority of patients are currently in this age group [28, 29, 37, 69, 70, 71]. If preoperative shock, longstanding perforation (>24 h), age above 70 years, and severe medical illness are all present, the mortality has been shown to reach 100% [53, 69]. While younger patients with perforated ulcers are predominantly male, the ratio changes to a female preponderance in patients above the age of 65 years [26, 29, 48, 49, 70]. Increased use of NSAIDs and a longer life expectancy of women have been suggested as explanations [71, 72, 73, 74], since a correlation between NSAID prescription, NSAID usage, age, and peptic ulcer perforations has been shown [26, 73]. Sepsis accounts for about 20% of fatal cases in elderly patients [37].

The mortality risk seems to be related to the ulcer location. Perforated gastric ulcers have been shown to carry a two- to threefold increased mortality risk relative to perforated duodenal ulcers [28, 37, 44, 45, 46, 48]. In the situation of a perforated peptic ulcer, the mortality of gastric resections seems to be higher than that of simple closure [46].

There is a considerable postoperative morbidity, which may concern up to two-thirds of patients. Up to 30% of patients suffer from pneumonia, 10–15% may have a wound infection or an abdominal abscess, and urinary tract infections are often observed [37, 48]. Cardiac problems such as myocardial failure or arrhythmias often complicate the postoperative course in elderly patients [37, 48, 69], and the proportion of patients with associated diseases who died increased from 27% to 85%

over the last six decades [48]. Delaying the time point of an operation beyond 24 h after the onset of symptoms increases the risk of complications sixfold and the length of hospital stay [44]. Perforated gastric ulcers seem to carry a higher risk of postoperative morbidity than duodenal ulcers [45, 48].

The outcome of laparoscopic repair for perforated duodenal ulcer has been comparable with open surgery, with a mortality and morbidity rate of 0–30% each [49, 52, 59, 60, 61, 62, 66]. Some studies observed mortality and morbidity rates lower than those known from open surgery [52, 62, 64, 68], so that the question arises whether a patient selection contributed to the results reported.

The influence of *H. pylori* infection in duodenal ulcer perforations has been evaluated in a variety of studies. There is a 50–100% prevalence of *H. pylori* infection in perforated duodenal ulcers [4, 33, 75, 76, 77, 78] and, in about 40% of cases, *H. pylori* can be found in the mucosa and throughout the ulcer wall [78]. However, a direct association between *H. pylori* infection and duodenal ulcer perforation has been negated [4, 75]. *H. pylori* infection is associated with increased rates of recurrent epigastric pain, positive endoscopic findings, and ulcer bleedings postoperatively [52, 79], and recurrent duodenal ulcer rates of 18–40% have been reported postoperatively [54, 55, 79]. As a consequence of the high rates of clinical symptoms and endoscopic findings observed, postoperative endoscopy should be performed and eradication of *H. pylori* infection is recommended in patients with perforated peptic ulcer [43, 77, 78, 79, 80].

The most substantial impact of *H. pylori* eradication on life expectancy has been calculated in complicated peptic ulcer disease, the increase ranging from 5 years to 26 years, depending on the patient's age [81]. Although perforation, bleeding and gastric outlet obstruction were not differentiated further, the available data suggest that *H. pylori* eradication could considerably increase life expectancy in patients with perforated peptic ulcer disease.

The long-term mortality in survivors of peptic ulcer perforation was increased relative to that of the general population for at least 30 years after the initial perforation, and the excess death rate was caused by cardiorespiratory diseases and non-gastric malignancies, but not by further ulcer complications [82]. Smoking prevalences of about 80–85% have been reported in patients with ulcer perforation [75, 83], and current smoking was related to ulcer perforation by a tenfold increased risk and a dose–response relationship [83]. Since smoking is known to increase the prevalence of cardiorespiratory diseases and lung cancer, smoking might be the link to the long-term increase in mortality in patients with peptic ulcer perforation.

Surgical management of peptic ulcer bleeding

Indication

Peptic ulcers remain the most common cause of gastrointestinal bleeding [22, 25]. Fortunately, about 80–85% of bleeding ulcers stop bleeding spontaneously [3, 25, 84]. In experienced hands, initial hemostasis in bleeding peptic ulcers can be achieved by endoscopic treatment in 95–99%, and 85%–95% of peptic ulcer bleedings can eventually be handled by endoscopic treatment [85, 86, 87]. Endoscopic treatment of bleeding peptic ulcers has been shown reduce the rates of re-bleeding, surgery, and mortality and to be cost effective relative to medical-surgical therapy [25, 38, 88, 89].

About 1.5–2% of bleeding peptic ulcers continue to bleed despite endoscopic treatment, and these patients go to emergency operation directly [85, 87]. About 15–20% of peptic ulcers re-bleed after endoscopic treatment. About 5–10% of bleeding peptic ulcers need emergency operation, and the overall mortality should be below 5% today [5, 25, 39, 87, 90, 91]. Bleeding duodenal ulcers seem to have a lower re-bleeding rate and mortality than gastric ulcers [21]. The risk of re-bleeding increases with ulcer size (>2 cm), age, concurrent coronary heart disease, and shock on admission [5, 25, 87, 92, 93], while peptic ulcer bleeding due to NSAID usage reduced the risk of re-bleeding by almost 50% [87, 93]. Re-bleeding in the absence of a Doppler signal from the base of the ulcer is rare, and patients with a clean-based ulcer virtually never require urgent intervention for recurrent bleeding [24].

There are some features of bleeding ulcers that predict ongoing bleeding or a high probability of re-

Table 2 Risk of bleeding in patients without endoscopic therapy according to the ulcer appearance (adapted from [25])

Forrest classification	Endoscopic appearance	Prevalence % (range)	Re-bleeding % (range)	Surgery % (range)	Mortality % (range)
I a, I b *n*=3194	Active bleeding	18 (4–26)	55 (17–100)	35 (20–69)	11 (0–23)
II a *n*=1647	Visible vessel	17 (4–35)	43 (0–81)	34 (0–56)	11 (0–21)
II b *n*=1420	Adherent clot	17 (0–49)	22 (14–36)	10 (5–12)	7 (0–10)
III *n*=1288	Flat spot	20 (0–42)	10 (0–13)	6 (0–10)	3 (0–10)
III *n*=2869	Clean base	42 (19–52)	5 (0–10)	0.5 (0–3)	2 (0–3)

Ulcer appearance according to the Forrest classification [94]: *Forrest Ia* spurting arterial bleeding; *Forrest Ib* oozing venous bleeding; *Forrest IIa* visible non-bleeding vessel; *Forrest IIb* adherent blot clot; *Forrest III* flat spot or clean base

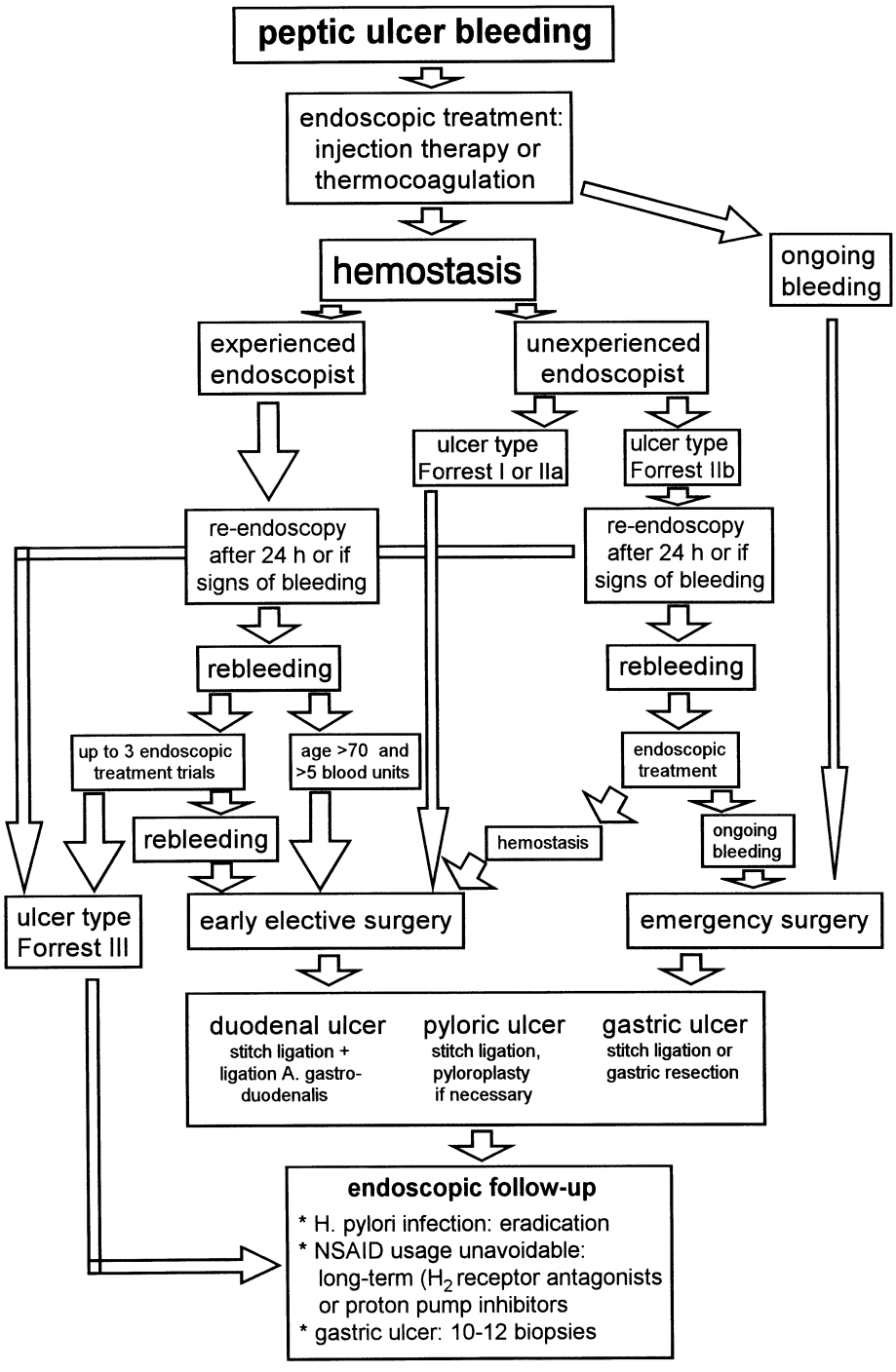

Fig. 2 Bleeding peptic ulcer treatment strategy. By reviewing the current literature, a flow chart was developed (details and references see text)

bleeding. These are mainly the ulcers with spurting arterial or oozing venous bleeding, classified as Forrest Ia and Forrest Ib ulcers, or Forrest IIa ulcers with a visible non-bleeding vessel [94] (Table 2). If a non-bleeding visible vessel is Doppler positive, the risk of re-bleeding is 80% [92]. When endoscopic treatment fails or recurrent bleeding occurs, a decision has to be made whether re-endoscopy or emergency surgery is indicated.

In the past decade, some centers have advocated early elective surgery in those patients with a high risk of re-bleeding [84, 92, 95], as re-bleeding in the hospital increased mortality by 6- to 12-fold [84]. This approach reduced mortality to below 10%, while it was previously around 25% in these high-risk patients [84, 92, 95, 96]. However, repeated endoscopic treatment after initial endoscopic bleeding control has been shown to reduce the

need for surgery, morbidity, and mortality relative to a single endoscopic treatment trial, and permanent bleeding control can be achieved by repeated endoscopic treatment even in high risk ulcers in more than 80% of cases [85, 87, 90]. Accordingly, re-endoscopy should be attempted. If more than three endoscopic treatments are needed, the probability of permanent bleeding control drops below 50%, so that surgery is indicated [85]. If more than five blood units are needed, especially in the case of concomitant medical illness or increased age, mortality increases and surgery should be considered [92] (Fig. 2).

Technique

Bleeding peptic ulcers requiring surgery are mainly located in the first part of the duodenum, accounting for about 75% of cases, while bleeding gastric and pyloric ulcers account for about 20% and 5%, respectively [37].

In most patients, stitch ligation of the ulcer is sufficient to control peptic ulcer bleeding. Extraduodenal ligation of the gastroduodenal artery is necessary to stop bleeding of large duodenal ulcers and should always be done [97]. About 25% of patients require some form of pyloroplasty, and about 10% of patients undergo gastric resection, mainly Billroth-II resection [37]. The presence of an ulcer with a diameter of more than 2 cm increases the likelihood that a gastric resection must be performed [98].

Whether an emergency operation for bleeding peptic ulcer should be combined with a vagotomy for permanent control of gastric acid secretion is questionable. In the past, emergency surgery for bleeding peptic ulcer was often combined with some form of vagotomy [3, 8, 37, 92, 98]. However, in the light of a decreasing experience with this form of surgical treatment, the availability of potent drugs to suppress gastric acid secretion, and the current pathophysiological understanding of peptic ulcer disease as an infectious disease in most cases, we would refrain from doing so.

Bleeding of duodenal ulcers not controlled endoscopically have been treated recently in six patients by minilaparotomy, pyloroplasty, oversewing of the arterial bleeder, and laparoscopic bilateral truncal vagotomy. Bleeding was controlled in all patients, and no ulcer recurred [99]. The technique seems to be feasible, but superiority to open surgery is unproven. In addition, vagotomy to reduce gastric acid secretion is unnecessary today, and truncal vagotomy should no longer be used, as the re-operation rate due to postvagotomy symptoms approached 20% in open surgery [100]. It is unlikely that a study will compare these two techniques in the future, as emergency surgery for bleeding peptic ulcer becomes increasingly rare [85, 87]. From the lessons we have learned from truncal vagotomy in open surgery [12, 100], we would recommend not using this procedure.

Outcome

Emergency operations for bleeding peptic ulcers carry a mortality risk of 26–30% [20, 37, 86]. The most important predictors of mortality are age above 70 years, concurrent medical illness, mainly cardiorespiratory diseases, and shock on admission. In these cases, a mortality of more than 50% has been reported [86]. Mortality increased if more than five blood units were needed [92]. The APACHE II score has been used to estimate the mortality risk in bleeding gastric ulcers, and patients with a score of 15 or higher had a mortality of 55%, compared with a mortality of 5% with a score lower than 15 [86]. About half of the fatal cases eventually die due to sepsis, of which about 20% are related to pneumonia [37].

Mortality was below 10% if initial hemostasis was achieved by endoscopic treatment and patients with a high risk of re-bleeding proceeded to early elective surgery [84, 95, 96]. Similar mortality rates have been reported by repeated endoscopic treatment, but the need for surgery was reduced [85, 87, 90].

The postoperative morbidity in emergency surgery for bleeding peptic ulcer is high. More than half of the patients suffer from infections of the lung, the urinary tract or the wound [37, 86]. The high incidence of fatal infectious problems provides a rationale for antibiotic therapy in bleeding peptic ulcer patients. There is a considerable risk of re-bleeding in patients operated for bleeding gastric ulcers. The incidence is around 15%, and these patients have a more than 50% mortality risk [86]. NSAID usage increased the risk of gastrointestinal bleeding by two- to threefold, the risk for gastrointestinal surgery by seven- to eightfold, and the mortality risk by four- to fivefold [74]. However, NSAID usage was not correlated with an increased risk for emergency surgery or mortality in bleeding ulcer patients [73, 101], and gastric ulcer bleeding due to NSAID usage had a reduced risk of re-bleeding postoperatively [86].

The results of trials investigating the effects of H_2 receptor antagonist or proton-pump inhibitor treatment on re-bleeding, emergency surgery, and mortality rates are inconclusive. A meta-analysis of H_2 receptor antagonist treatment identified only marginal effects on the probability of re-bleeding, surgery, and death, and these effects were solely attributable to effects on gastric ulcers [102]. When no endoscopic treatment for hemostasis was performed, proton-pump inhibitors have been shown to reduce the risk of re-bleeding and emergency surgery in one study [5] but not in another [103]. When initial hemostasis was achieved by endoscopic treatment, proton-pump inhibitor treatment reduced the re-bleeding rate in one study [104] and not in another [105], while the need for emergency surgery was reduced in both. Finally, proton-pump inhibitor treatment did not improve mortality in all four studies [5, 103, 104, 105]. However, as gastric

acid suppression has been shown to accelerate ulcer healing and to increase ulcer healing rates [2], it is reasonable to initiate therapy to promote healing [25].

H. pylori infection is present in 55–75% of patients with bleeding peptic ulcer, so that the prevalence in bleeding peptic ulcers might be 15–20% lower than in non-bleeding ulcers [6, 21, 25, 33]. *H. pylori* infection has been shown to be an independent risk factor for peptic ulcer bleeding and re-bleeding [6, 21, 25]. Two uncontrolled and two randomized controlled trials showed that patients treated for *H. pylori* infection had no recurrent episodes of hemorrhage in the subsequent year, compared with a 30% re-bleeding rate in those not eradicated [18, 106]. Even in ulcer bleeding attributable to NSAID usage, *H. pylori* infection increased the risk of bleeding by about 80% [107]. As a consequence, eradication of *H. pylori* infection in patients with bleeding ulcers is strongly recommended [21, 25, 80, 106, 107].

As in patients with perforated peptic ulcer, the long-term mortality in patients with bleeding peptic ulcers is increased relative to that of the general population. The increased mortality was predominantly due to smoking-related diseases such as lung cancer or benign respiratory and cardiovascular diseases, while deaths from recurrent peptic ulcer complications were not responsible for the excess death rate [101, 108, 109].

Surgical management of gastric outlet obstruction

Indication

Surgery for gastric outlet obstruction due to peptic ulcer disease is rather rare, although the incidence in Finland has been estimated as 1–3 per 100,000 inhabitants per year [30]. Approximately 1–2% of patients with peptic ulcer disease develop gastric outlet obstruction [40], and about 80% of gastric outlet obstructions due to peptic ulcer disease are caused by duodenal ulcers [30]. In Japan, a decline in the number of operations for stenotic ulcers has been described recently [33]. Patients operated on for stenotic ulcer do not differ from those operated on for perforated or bleeding peptic ulcer with respect to gender distribution or age. Recurrent peptic ulcer disease is more prevalent, while stress-related or NSAID-related ulcers are less often observed [33].

Patients present with vomiting and the inability to maintain body weight, as fluid and caloric intake are increasingly restricted. A gastroduodenogram or endoscopy reveal narrowing of the pyloric region, which cannot be passed by the endoscope. Malignancy has to be excluded by endoscopically retrieved biopsies, as gastric outlet obstruction is most often caused by malignant tumors [40, 110]. However, endoscopic biopsy to detect malignant obstruction has been shown to have a poor sensitivity of less than 40%, so that surgical exploration has been recommended [111]. Computed tomography or endoscopic ultrasound might be indicated to judge the extraluminal peripyloric region [40].

Some patients present with an acute inflammatory edema and an impaired antral motility. Such patients are usually treated successfully with nasogastric tube decompression, intravenous hydration, and gastric acid suppression by proton-pump inhibitors for 3–5 days, as reversible outlet obstruction will almost always be apparent within this time period [3].

In patients with chronic gastric outlet obstruction due to recurrent peptic ulcer, the pylorus is usually very rigid due to extensive scarring. Endoscopic treatment can be tried, as repeated dilatations can be performed safely. To date, no criteria have been identified that are useful to identify patients as likely to succeed or fail endoscopic balloon dilatation. There are no controlled studies comparing endoscopic balloon dilatation and surgery as treatment for gastric outlet obstruction. Considering the morbidity, the mortality, and the costs of surgery, endoscopic balloon dilatation should be attempted initially. If endoscopic dilatation fails or symptoms frequently recur, surgery can be performed without compromising the patient outcome [40].

Technique

A minimum of 72 h of nasogastric tube decompression should be used to minimize the postoperative period of gastric atony, before surgery for gastric outlet obstruction is performed [3]. Due to extensive scarring, pyloroplasty is often not feasible. Gastric resection with some form of vagotomy has been used as a definitive treatment to reduce gastric acid output and to remove the physical obstruction. Simple gastrojejunostomy has not been recommended, as ulcer recurrence rates of up to 50% have been reported [3]. However, these data stem from the period before H_2 receptor antagonists or proton-pump inhibitors became available or before *H. pylori* infection was identified as the main causative agent of peptic ulcer disease. Possibly, gastrojejunostomy and eradication of *H. pylori* or long-term prescription of a proton-pump inhibitor might be sufficient today. As this issue is unresolved, no general recommendation can be given at this time.

Outcome

The long-term results of endoscopic treatment have been rather disappointing. Endoscopic balloon dilatation resulted in a short-term technical success in 83–100%, and the morbidity, mainly perforation, was only 2% [40, 112]. About 30% of patients were permanently relieved from symptoms by a single dilatation [40, 112], but ob-

jective improvement lasted for more than 3 months in 38% of patients only, and 33–84% of patients needed repeated dilatations [40]. Stent placement has been tried in the pyloric region to obviate repeated dilatations, but no reports on larger series or long-term observations are available yet [40].

When initial endoscopic treatment was successful, subsequent failures of endoscopic dilatations occurred in 10% of cases, necessitating surgery immediately. Another 20% required surgery after repeated endoscopic balloon dilatations, so that about 30% of patients with benign gastric outlet obstruction initially treated by balloon dilatation eventually require surgery [112].

When surgery for gastric outlet obstruction is necessary, morbidity and mortality should be below 15% and 5%, respectively, as elective surgery is possible [20, 30]. In patients treated by antrectomy or Billroth-I resection, re-stenosis occurs in 5–8% [30]. Proximal selective vagotomy in combination with a pyloroduodenal dilatation should not be used any more, as high ulcer recurrence rates and re-stenosis rates of more than 40% have been reported [3, 30].

H. pylori infection has been observed in 45–69% of patients operated for gastric outlet obstruction [33, 112], which is significantly less than in patients with perforated or bleeding peptic ulcers. *H. pylori* eradication has been strongly recommended in patients with recurrent peptic ulcer disease [16, 18, 80], which is the case in almost all patients with gastric outlet obstruction. However, whether this improves outcome is not known yet. In one series, nine patients with gastric outlet obstruction and *H. pylori* infection were treated by endoscopic balloon dilatation and *H. pylori* eradication. On follow-up, only one of these patients needed surgery (11%), while in 12 of 31 patients, in which *H. pylori* status was unknown (n=27) or negative (n=4), 39% of patients eventually needed surgery after endoscopic balloon dilatation was initially successful [112].

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