Management of Massive Peptic Ulcer Bleeding

Frances K.Y. Cheung, FRCS, James Y.W. Lau, MD

In an old surgical series, massive bleeding from a peptic ulcer has been defined as blood loss of such a magnitude that the patient is either in shock or bleeding actively and being treated for shock. Blood transfusion would be required not simply to correct anemia but to restore or maintain vital signs. There are also signs of active bleeding from the upper gastrointestinal tract as indicated by hematemesis and melena or passage of blood per rectum, associated with continuing bloody aspirate after gastric lavage. In another study of massive bleeding in duodenal ulcer, Gardner and Baronofsky defined massive bleeding as a recent episode of melena or hematemesis and hemoglobin of 8 g/dL or less or a fall in blood pressure.

Mortality in patients with massive bleeding is unacceptably high. They merit intensive monitoring and aggressive treatment. The National United Kingdom Audit conducted in 1993 was a population-based, multicenter, prospective observational study in 4185 patients presenting with acute upper gastrointestinal bleeding. The Audit reported a crude mortality of 14%. In the cohort, there were 2071 patients with peptic ulcer presenting with acute hemorrhage. In the 251 patients (12%) that came to surgery, mortality was 24%.

CLINICAL ASSESSMENT AND PREPARATION OF PATIENTS

Patients with upper gastrointestinal bleeding require prompt assessment and volume resuscitation. Hematemesis, passage of fresh melena, shock, and a low hemoglobin level signify significant ongoing bleed or a recent significant bleed. Endotracheal intubation should be considered in patients with active hematemesis, unstable vital signs, or altered mental state to minimize the risk of aspiration pneumonia. Coagulopathy exacerbates bleeding and should be corrected with blood products. Massive bleeding mandates emergency endoscopy. Emergency endoscopy is performed as soon as the patient is stabilized after initial resuscitation. In patients with exigent bleeding,
endoscopy can be performed during resuscitation. A more liberal policy in emergency endoscopy should be offered to elderly patients and patients with comorbid illnesses, because they tolerate blood loss poorly and are more likely to suffer from organ dysfunctions consequent to hypotension.

**ENDOSCOPIC TREATMENT**

Endoscopic therapy remains the first treatment modality in the management of bleeding peptic ulcers, even in those presenting with massive bleeding. Endoscopy allows the bleeding source to be localized and excludes varices as the cause of upper gastrointestinal bleeding, as the management is different from that of ulcer bleeding. As endoscopic signs or stigmata of bleeding are prognostic, an early endoscopy enables clinicians to risk-stratify patients. More importantly, endoscopic therapy improves outcomes in patients with actively bleeding ulcers and ulcers with a visible vessel. Sacks and colleagues\(^5\) performed a meta-analysis of 25 randomized controlled trials that compared endoscopic hemostasis to standard treatment. The systematic review showed that endoscopic therapy reduced the risk of recurrent and continued bleeding (69% relative reduction), emergency surgery (62% relative reduction), and mortality (30% relative reduction). Cook and colleagues\(^6\) analyzed data from 30 trials and concluded that endoscopic therapy significantly reduced the rate of rebleeding, surgery, and mortality. The effects were greatest in patients with active bleeding ulcers or nonbleeding, visible vessels. There is also recent evidence to suggest that endoscopic treatment of adherent clots is beneficial. Kahi and colleagues\(^7\) performed a meta-analysis pooling results of six clinical trials with 240 randomized patients (two in abstract form only) and concluded that endoscopic therapy would be more effective in preventing recurrent bleeding when compared with medical therapy alone (rate of recurrent bleeding, 8.2% vs 24.7%).

We now have evidence that the addition of a second modality to injection therapy further improves patients' outcomes. A Cochrane systematic review pooled data from 17 trials that compared epinephrine injection to epinephrine injection and a second treatment method in 1763 high-risk patients.\(^8\) The addition of a second treatment conferred a reduction in rate of recurrent bleeding from 18.8% to 10.4%, emergency surgery from 10.8% to 7.1%, and mortality from 5% to 2.5%, regardless of which second procedure was applied. We favor the use of hemoclips or a 3.2-mm heater probe. We believe that firm tamponade of the bleeding artery and its coaptive coagulation with a contact thermal probe produce secure hemostasis. The use of hemoclips is closer to surgical ligature in hemostasis. We performed a pooled analysis of 15 randomized trials that compared hemoclips to the use of a heater probe in nonvariceal bleeding.\(^9\) The rate of definitive hemostasis was high with either treatment modalities (81.5% vs 81.2%, respectively). There was no difference in the rate of recurrent bleeding, surgery, and death. In clinical practice, successful placement of clips is particularly difficult in fibrotic ulcer with a tangential position, where many difficult ulcers occur. In a randomized trial, 10% of patients randomized to a hemoclip group did not receive hemoclip placement due to technical failure.\(^10\) The use of either modality should not be mutually exclusive.

**WHAT IS THE LIMIT TO ENDOSCOPIC THERAPY?**

A big bleed is often consequent to a big eroded artery. Blood flow is proportional to the fourth power of the vessel diameter; a small increase in diameter would greatly increase flow. Swain and colleagues\(^11\) studied 27 gastrectomy specimens in patients who underwent urgent surgery for bleeding gastric ulcers. He used thin-barium
angiography to study the bleeding artery underneath these ulcers. The study predated the widespread use of endoscopic therapy. It was not entirely clear if the ulcers studied had been treated by endoscopic means. The bleeding artery had a mean external diameter of 0.7 mm (0.1–1.8 mm). In 13 ulcers, the arteries were subserosal and were technically outside the stomach wall. The other bleeding arteries were smaller than 1 mm in size and were submucosal in disposition. In about half of the arteries, there were aneurysmal dilatations at the bleeding point. Larger penetrating ulcers are more likely to erode into larger subserosal arteries. Swain and colleagues published only in an abstract the size of arteries in patients who died after a major bleed from their peptic ulcers. The mean diameter of the bleeding artery was 3.75 mm.

In a canine mesenteric artery model, Johnston and colleagues studied the limits of endoscopic thermocoagulation in securing hemostasis. The authors emphasized the need for firm compression onto the artery by a contact probe. Due to the limit in the size of the endoscope channel, a 3.2-mm contact thermal probe is arguably the best available hemostatic device. The use of a 3.2-mm contact thermocoagulation device was shown to consistently seal arteries up to 2 mm in size. Findings of an in vitro model in a real clinical situation may not be as applicable as conditions are often less ideal.

Elmunzer and colleagues systematically reviewed 10 prospective series that evaluated predictive factors for endoscopic failure (Table 1). Two of them employed epinephrine injection as a single modality of therapy. Most commonly identified preendoscopic factors were hemodynamic instability and the presence of comorbid illnesses. During endoscopy, active bleeding, large ulcer size, location of ulcer at posterior bulb duodenum, and lesser curve were identified as predictors for endoscopic failure. The author remarked that on the basis of consistency and statistical strength, hemodynamic instability, active bleeding, large ulcer size, and posterior duodenal location appear to be the most important predictors of recurrent bleeding. Larger ulcers located at the posterior bulb duodenum and lesser curve are likely to erode into large arterial complexes—the gastroduodenal artery complex and the left gastric artery proper or its branches. The arteries are often sizable. Bleeding from these arteries exceeds the limit of what endoscopic devices can secure.

### ROLE AND TIMING OF SURGERY

Surgery remains the commonest salvage method for the few patients in whom bleeding cannot be controlled at endoscopy. In the published literature, surgery is often defined as an end point to trials evaluating endoscopic therapy. The rate of operative intervention has decreased dramatically over the years with the advent of endoscopic therapy. Nonetheless, surgery has an important gate-keeping role, and it often represents the last defense against exsanguination.

As aforementioned, the operative mortality following failed endoscopic therapy is substantial, ranging from 15% to 25%. There are several reasons. First, unsuccessful endoscopic therapy leads to more blood loss and patients are left in a poorer shape after episodes of hypotension. Operative mortality increases with number of episodes of recurrent bleeding and endoscopic attempts. Second, we are managing an aging population with bleeding. Third, ulcers not amenable to endoscopic treatment are “difficult” ulcers. These are large chronic ulcers in difficult positions. Following the near extinction of elective ulcer surgery, experience in dealing with these difficult ulcers is patchy. How to decide on the optimal timing of operation in the right patient remains a challenging clinical decision.
## Table 1
Independent predictors of rebleeding after endoscopic therapy

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Study</th>
<th>% Rebleeding in Entire Study Population</th>
<th>% Rebleeding in Patients With Predictor</th>
<th>% Rebleeding in Patients Without Predictor</th>
<th>Odds Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemodynamic instability</td>
<td>Guglielmi</td>
<td>20 (86/429)</td>
<td>41.1 (30/73)</td>
<td>14.8 (54/366)</td>
<td>3.68 (1.99–6.81)</td>
</tr>
<tr>
<td></td>
<td>Wong</td>
<td>8.3 (94/1,128)</td>
<td>19.2 (35/182)</td>
<td>6 (56/946)</td>
<td>2.21 (1.40–3.48)</td>
</tr>
<tr>
<td></td>
<td>Thomopolous</td>
<td>22 (86/390)</td>
<td>47.1 (24/51)</td>
<td>16 (54/339)</td>
<td>2.31 (1.33–4.97)</td>
</tr>
<tr>
<td></td>
<td>Brullet (DU)</td>
<td>16.7 (17/102)</td>
<td>32.0 (8.25)</td>
<td>12.3 (10/81)</td>
<td>3.53 (1.27–4.1)</td>
</tr>
<tr>
<td></td>
<td>Park</td>
<td>20 (25/127)</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Comorbid illness</td>
<td>Villanueva</td>
<td>24.5 (57/233)</td>
<td>36.5 (42/115)</td>
<td>12.7 (15/118)</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td>Saeed</td>
<td>12 (8/69)</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Likelihood ratio 7.63, P = 0.005</td>
<td></td>
</tr>
<tr>
<td>Active bleeding</td>
<td>Guglielmi</td>
<td>20 (86/829)</td>
<td>20.3 (39/192)</td>
<td>18 (45/247)</td>
<td>14.47 oozing, 13.38 spurting</td>
</tr>
<tr>
<td></td>
<td>Wong</td>
<td>8.3 (94/1,128)</td>
<td>12.1 (71/587)</td>
<td>4.2 (23/541)</td>
<td>1.65 (1.07–2.56)</td>
</tr>
<tr>
<td></td>
<td>Chung</td>
<td>25.2 (35/139)</td>
<td>NR</td>
<td>NR</td>
<td>6.48 (1.88–22.49)</td>
</tr>
<tr>
<td></td>
<td>Thomopolous</td>
<td>22 (86/390)</td>
<td>48.9 (46/94)</td>
<td>10.8 (32/296)</td>
<td>2.45 (1.51–3.93)</td>
</tr>
<tr>
<td></td>
<td>Brullet (GU)</td>
<td>13.1 (23/175)</td>
<td>26 (13/50)</td>
<td>8 (10/125)</td>
<td>2.98 (1.12–7.91)</td>
</tr>
<tr>
<td>Ulcer Type</td>
<td>Treatment</td>
<td>Rebleeding Rate</td>
<td>Failure Rate</td>
<td>95% CI</td>
<td></td>
</tr>
<tr>
<td>-------------------------------</td>
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<td></td>
</tr>
<tr>
<td>Large ulcer size (≥ 2 cm)</td>
<td>Guglielmi</td>
<td>20 (86/429)</td>
<td>31.3 (40/128)</td>
<td>14.1 (44/311)</td>
<td>4.61 (2.20–9.64)</td>
</tr>
<tr>
<td></td>
<td>Wong*</td>
<td>8.3 (94/1,128)</td>
<td>14.8 (36/244)</td>
<td>6.6 (58/884)</td>
<td>1.80 (1.16–2.83)</td>
</tr>
<tr>
<td></td>
<td>Brullet± (GU)</td>
<td>13.1 (23/175)</td>
<td>23.9 (16/67)</td>
<td>6.5 (7/108)</td>
<td>3.64 (1.34–9.89)</td>
</tr>
<tr>
<td></td>
<td>Brullet± (DU)</td>
<td>16.7 (17/102)</td>
<td>36.3 (8/22)</td>
<td>12 (10/84)</td>
<td>2.29 (1.13–10.9)</td>
</tr>
<tr>
<td>Large ulcer size (&gt;1 cm)</td>
<td>Villanueva</td>
<td>24.5 (57/233)</td>
<td>42.0 (34/81)</td>
<td>15.1 (23/152)</td>
<td>NR</td>
</tr>
<tr>
<td>Posterior duodenal ulcer</td>
<td>Thomopolous±</td>
<td>22 (86/390)</td>
<td>43.2 (16/37)</td>
<td>17.6 (62/353)</td>
<td>2.48 (1.37–7.01)</td>
</tr>
<tr>
<td></td>
<td>Park</td>
<td>20 (25/127)</td>
<td>44 (11/25)</td>
<td>13.7 (14/102)</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td>Villanueva</td>
<td>24.5 (57/233)</td>
<td>57.1 (20/35)</td>
<td>18.7 (37/198)</td>
<td>NR</td>
</tr>
<tr>
<td>Lesser gastric curve ulcer</td>
<td>Brullet± (GU)</td>
<td>13.1 (23/175)</td>
<td>22.9 (16/70)</td>
<td>6.7 (7/105)</td>
<td>2.79 (1.01–7.69)</td>
</tr>
<tr>
<td></td>
<td>Park</td>
<td>20 (25/127)</td>
<td>35 (7/20)</td>
<td>16.8 (18/107)</td>
<td>NR</td>
</tr>
</tbody>
</table>

**Abbreviations:** DU, Duodenal ulcer; GU, Gastric ulcer; NR, Not reported.

* CI = 3.27–64.05.

** CI = 2.69–66.66.

± Percentage of patients experiencing rebleeding was not available. Percentage of patients experiencing overall failure (defined as the failure to achieve initial hemostasis and recurrent hemorrhage) is reported.

The timing of surgery was a subject of intense debate in the 1980s when endoscopic therapy was not available or widespread. There were two randomized studies. The Birmingham trial randomized 104 patients with bleeding peptic ulcer to early or delayed surgery.\textsuperscript{18} Criteria for early surgery were four units of blood or plasma expander needed to correct acute blood loss in 24 hours, one rebleed, and endoscopic stigmata or one previous bleed with 2 years of dyspepsia. Criteria for surgery in the delayed group were eight units of blood or plasma expander in 24 hours, two rebleeds, and persistent bleeding requiring 12 units of blood in 48 hours or 16 units in 72 hours. In patients younger than 60 years of age, there was no death in either group, but the early surgery policy led to an unacceptably high operation rate (52\% in the early and 5\% in the delayed group). For those older than 60 years of age, the operation rate was 62\% in the early group and 27\% in the delayed group. There were three deaths in 48 patients (6\%) in the early group and seven deaths in 52 patients (13\%) in the delayed group. On an intention-to-treat analysis, there was no statistical difference. There was subgroup difference in favor of early surgery in elderly patients with bleeding gastric ulcers only on per protocol analysis (0 in 19 of the early group vs 5 in 21 of the delayed group, $P<.01$). The trial was criticized for allowing ongoing bleeding in elderly patients. Saperas and colleagues\textsuperscript{19} randomized 69 patients older than 50 years of age in whom emergency endoscopy showed nonarterial bleeding or signs of recent hemorrhage without a visible vessel to receive either immediate surgery or expectant management. In the latter group, 23 of 34 (68\%) patients had no further bleeding, whereas 11 patients were operated upon for further bleeding. Mortality in those who underwent surgery was 14.7\% (5 in 34 patients) and 2.9\% (1 in 34 patients) in patients who were managed expectantly. The outcome of both trials leads us to conclude that a routine policy offering early surgery should not be instated particularly when no major endoscopic stigma is evident as shown in the second trial. It is, however, difficult to interpret both trials in the context of endoscopic therapy. Endoscopic therapy should be attempted in patients with massive bleeding from a peptic ulcer. Often endoscopic hemostasis is possible. In patients in whom exigent bleeding cannot be controlled at endoscopy, surgery is clearly indicated. Whether to consider a second endoscopic attempt or immediate surgery in patients in whom bleeding recurs after initial endoscopic control is more controversial. Such patients are often elderly and at high surgical risk, and they are likely to benefit if endoscopy is repeated with satisfactory results. Conversely, the hypotension and delay in reestablishing hemostasis from repeated but unsuccessful endoscopic attempts are likely to adversely affect their survival. We compared endoscopic retreatment with surgery in patients in whom bleeding recurred after initial endoscopic control.\textsuperscript{20} In a 40-month period 1169 patients with bleeding peptic ulcers were treated by epinephrine injection followed by thermocoagulation to the vessel by a 3.2 mm heater probe. Hemostasis was not achieved in 17 patients, and they went directly to surgery. The rate of recurrent bleeding after endoscopic hemostasis was 8.7\%. Ninety-two (mean age, 65 years; 76\% men) were randomized; 48 were allocated to endoscopic retreatment and 44 to surgery. With intention-to-treat analysis, the endoscopic retreatment and surgery groups did not differ in mortality at 30 days (10\% vs 18\%, $P = .37$), duration of hospitalization (median, 10 vs 11 days; $P = .59$), need for or length of intensive care unit stay (5 vs 10 patients; median, 59 days for both; $P = .16$), or units of blood transfused (median, 8 vs 7 units $P = .27$). Patients who underwent surgery were more likely to have complications (7 vs 16; $P = .03$). Endoscopic retreatment was able to control bleeding in three-quarters of patients. In a logistic regression analysis of a small subgroup of patients, ulcers measuring 2 cm or more and hypotension at rebleeding were two independent factors predicting failure with endoscopic
retreatment. Findings from this trial led us to conclude that we can be selective in offering treatment to patients during their first rebleed. Cases of recurrent bleeding after initial endoscopic hemostasis can be broadly divided into those with suboptimal primary endoscopic treatment and those with arteries deemed to fail endoscopic treatment. Endoscopic retreatment is often successful and worthwhile in smaller ulcers. The pertinent message to the subgroup analysis is that initial control in ulcers predicted to fail may represent a window of opportunity for more aggressive treatment before its first bleed. Our clinical trial should prompt us to investigate the role of early intervention after initial endoscopic hemostasis in selected high-risk ulcers.

Imhof and colleagues\(^2\) reported results of a multicenter trial comparing endoscopic fibrin glue injection with early elective surgery in peptic ulcer patients with arterial bleeding or a visible vessel of 2 mm or more. After initial endoscopic control of bleeding, patients were randomized to repeated fibrin glue injection or early elective surgery. With an intention-to-treat analysis, bleeding recurred in 50% (16 of 33) of the patients in the endoscopic group and 4% (3 of 28) in the early surgical group. Mortality (two in each group) between groups, however, did not differ. We can only conclude from the trial that surgery is more definitive in hemostasis. In a prospective series using the concept of early elective surgery following endoscopic control in patients with arterial bleeding and a visible vessel of 2 mm or more in size, Imhof and colleagues\(^2\) reported an admirable overall operative mortality of 5%, whereas the mortality of a historical control group in which patients were treated conservatively was 14%.

Indeed, good results can be achieved with early surgery with adherence to defined protocols in dedicated units. Bender and colleagues\(^2\) operated on patients with shock on admissions, age greater than 65 years, ulcer size greater than 2 cm, or with stigmata of recent hemorrhage and previous admission for ulcer complication. About 66 patients (mean age, 58 years) were included in a 5-year period with no mortality. Mueller and colleagues operated on patients with spurting hemorrhage, nonbleeding visible vessels on posterior duodenal ulcers, blood transfusion greater than 6 units in the first 24 hours, and rebleeding in 48 hours. In a consecutive series of 157 patients, the 30-day mortality was 7%\(^2\).

Data from surgical units dedicated to the management of bleeding peptic ulcers support the use of early elective surgery in selected high-risk patients. There was probably publication bias, as the mean age in some of these series was low. These series were mostly uncontrolled trials.

**TYPES OF SURGERY**

The type of surgery to use has also been a subject of debate. In the presence of powerful proton pump inhibitor and *Helicobacter pylori* eradication, the aim of emergency surgery is no longer to cure ulcer diathesis but to secure bleeding. Acid reduction surgery is therefore unnecessary. Two randomized studies published in the 1980s, however, supported a more aggressive approach in surgical management. In a multicenter, randomized, controlled trial reported by Poxon and colleagues\(^2\), comprising 137 patients with bleeding peptic ulcer, patients were randomized to simple intraluminal ligature, plication, or conventional surgery (ulcer excision with either vagotomy and pyloroplasty or gastrectomy). H\(_2\) antagonist was used after surgery. Eight patients were excluded from the study due to misdiagnosis or loss of data. Recurrent bleeding occurred in 6 of the 62 patients who underwent simple plication, whereas none rebled after conventional surgery. The trial was aborted because of
a high rate of fatal rebleeding in those randomized to simple intraluminal ligature or plication alone. A total of 29 patients died—16 (26%) after conservative surgery and 13 (19%), after conventional operations.

The French Association of Surgical Research trial was a multicenter study that randomized only patients with bleeding bulbar duodenal ulcers. Of 202 eligible patients, 120 entered the study. They were randomly assigned to oversewing plus vagotomy and drainage or partial gastrectomy. The more aggressive approach of gastric resection was accompanied by a lower rebleeding rate (3% vs 17%). Gastric resection was, however, associated with a higher duodenal leak rate (13% vs 3%). On intention-to-treat analysis, the duodenal leak rate, however, was similar between two groups when leak following reoperation for recurrent bleeding in the vagotomy was taken into account (12% vs 13%). The overall mortality was high (22% in vagotomy group and 23% in gastric resection group). Duodenal stump leak is one of the main complications, either in the gastrectomy group or in the patients who rebleed in simple placation group and require conversion to gastrectomy. The authors suggested that when a gastric resection is required, a Billroth type I reconstruction avoiding closure of a duodenal stump is more desirable. A proper ligation of the gastroduodenal artery complex is necessary to avoid recurrent bleeding when a vagotomy and pyloroplasty are considered.

Findings from these two clinical trials suggested that a more aggressive approach would be warranted. They also underlie the fact that a great amount of surgical expertise is required in managing these ulcers. The extinction of elective ulcer surgery also means that there is atrophy in skills in managing these difficult ulcers. Surgeons with experience in dealing with these ulcers should be identified and be an integral member of a bleeding team.

**ANGIOGRAPHIC THERAPY—A LESS INVASIVE OPTION?**

In the 1970s, angiographic therapy was proposed as an alternative to surgery for massive peptic ulcer bleeding before the advent of therapeutic endoscopy. Significant advances have since been made in catheter and guidewire technology. We now have finer microcatheter systems and safer embolization materials such as microcoils. Ischemic complications are less seen today, and vessel occlusion is more long lasting. Ulcers that fail endoscopic hemostasis often erode into large arterial complexes—a lesser curve gastric ulcer often into branches of left gastric artery and a posterior bulbar duodenal ulcer into gastroduodenal or pancreaticoduodenal arteries. Due to abundant collateral circulation in the duodenum, cannulation of the superior mesenteric artery is occasionally required to exclude collateral supply from inferior pancreaticoduodenal artery. In a series by Loffroy and colleagues angiography showed contrast extravasation or false aneurysm at the bleeding site in 25 of 33 patients. Since ulcer bleeding is intermittent, blind therapy is sometimes based on endoscopic localization of bleeding site. Eriksson and colleagues reported the technique of clip placement during endoscopy, which facilitates the localization of the bleeder. Various agents including coils, glue, microspheres, and gelatin particles can be used alone or in combination to occlude feeding vessels after their selective cannulations. Complications including gastric and duodenal infarction, hepatic infarction, and late duodenal stenosis have been reported in early series. Special precaution and technique are required, especially when using glue, due to the risk of ischemia when the glue regurgitates to undesired vessels. Case series reporting the efficacy of angiographic embolization usually comprises patients with a high operative risk, with previous failed endoscopic therapy or with recurrent bleeding after surgery. The reported technical
<table>
<thead>
<tr>
<th>Study</th>
<th>Patient No.</th>
<th>Patient Characteristics</th>
<th>Techniques and Agents</th>
<th>Radiologic Success</th>
<th>Lasting Hemostasis</th>
<th>Complications of TAE</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Larssen and colleagues 2008</td>
<td>36</td>
<td>Failed endoscopic treatment or rebleed, 2 prophylactic</td>
<td>Coils, microcoils, gelfoam</td>
<td>92%</td>
<td>72%</td>
<td>3% no clinical consequence</td>
<td>19%</td>
</tr>
<tr>
<td>Loffroy and colleagues 2008</td>
<td>35</td>
<td>High operative risk patients</td>
<td>Coils, microcoils, gelatin powder, cyanoacrylate glue, microspheres</td>
<td>94%</td>
<td>77%</td>
<td>6% major complication (groin), 8% minimal complication</td>
<td>21%</td>
</tr>
<tr>
<td>Holme and colleagues 2006</td>
<td>40</td>
<td>13 had previous surgery, only include hemodynamically stable patients</td>
<td>Superselective with microcoils (12) or sandwich technique with coils (28)</td>
<td>83%</td>
<td>50%</td>
<td>0</td>
<td>25%</td>
</tr>
<tr>
<td>Toyoda and colleagues 2005</td>
<td>11</td>
<td>Severe comorbidities or malignancy</td>
<td>Coils, microcoils, or gelfoam embolized gastroduodenal, anterior and posterior superior pancreaticoduodenal arteries in 10</td>
<td>91%</td>
<td>91%</td>
<td>No major complication</td>
<td>27%</td>
</tr>
<tr>
<td>Ljungdahl and colleagues 2002</td>
<td>18</td>
<td>5 had previous surgery</td>
<td>Coils, microcoils, or gelfoam</td>
<td>100%</td>
<td>83%</td>
<td>2 patients (11%)</td>
<td>5%</td>
</tr>
<tr>
<td>Walsh and colleagues 1999</td>
<td>50</td>
<td>High operative risk patients</td>
<td>Coils, microcoils, polyvinyl alcohol, gelfoam</td>
<td>92%</td>
<td>46%</td>
<td>4% major leading to death</td>
<td>40%</td>
</tr>
</tbody>
</table>
success rate ranged from 90% to 100%, whereas clinical success rate is lower and ranged from 50% to 83% (Table 2). In these series, those who failed embolization or rebleed after embolization were treated with repeat endoscopy, surgery, or repeat embolization. An overall mortality of 5% to 40% was reported.

ANGIOGRAPHIC EMBOLIZATION VERSUS SURGERY

To date, there has been no controlled trial that compared angiographic embolization to surgery as a salvage procedure for failed endoscopic therapy. Two retrospective comparisons showed at least similar efficacy in terms of rate of rebleeding, morbidity, and mortality. Ripoll and colleagues41 retrospectively analyzed the outcome of 70 patients with refractory peptic ulcer bleeding. About 31 patients underwent angiographic embolization, and 39 patients were managed with surgery. Although patients receiving angiographic embolization were 10 years older and more had heart disease, the incidence of recurrent bleeding (29% vs 23%) and mortality was similar (26% vs 21%). Another retrospective comparison study by Eriksson and colleagues42 included 40 patients who underwent angiographic embolization and 51 patients who underwent surgery after failed endoscopic therapy. The angiographic embolization group was older with more comorbidity. The 30-day mortality was lower in the angiographic embolization group (3% vs 14%). These results are promising, and we are eagerly awaiting results of randomized, controlled trials to prove the benefits of angiographic embolization.

SUMMARY

Massive bleeding from a peptic ulcer remains a challenge. A multidisciplinary team of skilled endoscopists, intensive care specialists, experienced upper gastrointestinal surgeons, and intervention radiologists all have a role to play. Endoscopy is the first-line treatment. Even with larger ulcers, endoscopic hemostasis can be achieved in the majority of cases. Surgery is clearly indicated in patients in whom arterial bleeding cannot be controlled at endoscopy. Angiographic embolization is an alternate option, particularly in those unfit for surgery. In selected patients judged to belong to the high-risk group—ulcers 2 cm or greater in size located at the lesser curve and posterior bulbar duodenal, shock on presentation, and elderly with comorbid illnesses—a more aggressive postendoscopy management is warranted. The optimal course of action is unclear. Most would be expectant and offer medical therapy in the form of acid suppression. Surgical series suggest that early elective surgery may improve outcome. Angiography allows the bleeding artery to be characterized, and coil embolization of larger arteries may further add to endoscopic hemostasis. The role of early elective surgery or angiographic embolization in selected high-risk patients to forestall recurrent bleeding remains controversial. Prospective studies are needed to compare different management strategies in these high-risk ulcers.

REFERENCES


