Upper gastrointestinal bleeding: a review

Thomas A. Broughan, M.D.
David P. Vogt, M.D.

Department of General Surgery

Despite recent diagnostic and therapeutic advances, the mortality rate for upper gastrointestinal bleeding has remained at 10% for the past three decades.[1] Two factors are probably responsible for this persistently high mortality rate. First, this population is composed of a larger than average proportion of elderly patients who have marked associated medical diseases. Second, advanced techniques in intensive care allow patients who are critically ill or have had multiple traumas to be supported for long periods of time; many of these patients subsequently develop upper gastrointestinal bleeding that may be fatal.

Recent developments in the management of the patient with gastrointestinal bleeding include fiberoptic endoscopy, cimetidine therapy, gastric pH monitoring, and radiographic arterial embolization. Their clinical application is being investigated. Although bleeding episodes are controlled in most cases with medical management, approximately 15% to 20% of patients require surgery.[1]

Etiology

The most frequently encountered causes of upper gastrointestinal bleeding are peptic ulcer disease, acute gastric mucosal lesions, esophageal varices, and the Mallory-Weiss syndrome. Depending on

Table 1. Comparison of causes of upper gastrointestinal bleeding in two series

Etiology	Detroit (Sugawa)[2] (%)	Toronto (Halmagyi)[3] (%)
Duodenal ulcer	11	41
Acute erosive gastritis	42	2
Gastric ulcer	18	11
Mallory-Weiss tear	15	6
Esophageal varices	5	7
Other	10	33

the patient population considered, precise order of frequency varies (*Table 1*).[2,3] Peptic ulcer disease is responsible for approximately 50% of the cases of upper gastrointestinal bleeding in most series. Bleeding is directly responsible for 40% of all deaths encountered in the 20% of patients who bleed as a result of peptic ulcer disease.[4] Although duodenal ulcers are encountered more frequently, gastric ulcers are more virulent because of their greater associated blood loss and greater tendency for bleeding to recur.

The term *acute gastric mucosal lesions* encompasses a wide range of disorders. These include stress ulcers, Cushing's ulcer, Curling's ulcer, and erosive gastritis. Although the exact pathophysiology may vary among these disorders, the lesion that results is the same, mucosal ulceration that does not penetrate the muscularis mucosa. The gross appearance of these lesions may vary from a single bleeding point to a diffuse hemorrhagic gastritis that involves the entire mucosal surface of the stomach.

The pathophysiology is at least partially understood in some of these disorders. In Cushing's ulcer, associated with patients who have had neurological trauma or surgery, increased pressure in the fourth ventricle stimulates the vagal nuclei with a resultant gastric hypersecretion.[5] The erosions resulting from stress, burns (Curling's ulcer), and ulcerogenic agents are in part caused by the destruction of the gastric mucosal barrier.[6] Loss of this barrier allows back diffusion of hydrogen ions into the mucosal cells, which in turn releases vasoactive amines. The injuries caused by the hydrogen ions and vasoactive amines include mucosal congestion, hemorrhagic infarction, mucosal slough, and finally bleeding.

Although a patient may have documented esophageal varices, in about 50% of patients, the bleeding episode may be from another lesion such as an ulcer or hemorrhagic gastritis. Therefore, early investigation is mandatory since treatment of these various lesions differs dramatically. The prognosis for patients with a variceal hemorrhage is considerably worse than for patients with other lesions that may cause upper gastrointestinal bleeding and may approach 50% mortality. The exact mechanism that precipitates variceal rupture and hemorrhage is not clear. No definite correlation has been found between the degree of portal hypertension and the risk of subsequent bleeding.

The Mallory-Weiss syndrome has been recognized more frequently with the routine use of fiberoptic endoscopy in studying patients with upper gastrointestinal hemorrhage. Atkinson et al[7] have described an increased transmural pressure gradient in the stomach and esophagus that may occur with protracted vomiting or any other condition that markedly increases intra-abdominal pressure. This increased pressure gradient seems to focus on the area of the gastroesophageal junction and may result in mucosal laceration in this region. Risk factors include alcohol or

aspirin abuse, and the presence of a hiatal hernia. An endoscopic study reported by Knauer[8] observed that most tears involved either the gastroesophageal junction or the proximal stomach.

Clinical presentation

Although a good history and physical examination are mandatory in studying the patient with upper gastrointestinal bleeding, they are of limited value in identifying the source of bleeding. Allan and Dykes[9] found the history to be diagnostically helpful in only 57% of cases. Twenty-five percent of patients with chronic dyspepsia do not have a bleeding peptic ulcer, and conversely, up to one third of patients with a bleeding peptic ulcer deny any previous symptoms.[10] Although esophageal varices are the most likely source in a patient with classic signs of chronic liver disease, other lesions must be considered. A thorough oropharyngeal examination should be conducted to eliminate this area as a possible source of the bleeding. Most patients present either with melena or hematemesis; several associated symptoms and signs are shown in *Table 2*. Another important reason to obtain a thorough history is that patients with critical associated medical diseases are at higher risk and therefore should be identified. *Table 3* shows a series reported by Halmagyi[3] in which 65% of patients had at least one associated disease and 50% had more than one.

Table 2. Signs and symptoms of upper gastrointestinal bleeding

	Percent
Melena	87.0
Hematemesis	61.6
Pain	57.2
Dizziness	46.8
Tachycardia	28.2
Heartburn	24.0
Vomiting	22.6
Shock	17.9
Anorexia	17.7
Fainting	13.5
Weight loss	12.4
Bloating	4.7
Pyloric obstruction	3.0

Table 3. Associated diseases in patients with upper gastrointestinal bleeding[3]

	Percent
Cardiovascular	31.5
Liver	13.2
Skeletal	8.9
Chest	8.0
Central nervous system	8.0
Renal	6.3
Psychiatric	6.0
Multiple system failure	6.0
Malignancy	5.3
Hormonal	3.9
Pancreas	1.2
Septicemia	1.0
Blood dyscrasia	0.7

Diagnosis

Fiberoptic endoscopy is the procedure of choice in identifying the source of bleeding in a patient with upper gastrointestinal hemorrhage. Sugawa et al[2] reported a 97% accuracy rate with emergency endoscopy, compared to a 33% accuracy rate with emergency upper gastrointestinal fluoroscopy. It is important to perform endoscopy early because its accuracy decreases from 90% to 33% after 48 hours.[9]

Four early studies in the 1970s cast doubt on the efficacy of emergency endoscopy in decreasing morbidity and mortality associated with upper gastrointestinal bleeding.[9, 11–13] Although these studies had several errors in experimental design, no subsequent prospective, randomized study has yet clearly shown that emergency endoscopy as the single variable decreases the morbidity and mortality of upper gastrointestinal

bleeding. However, Griffiths et al[14] identified a subset of patients who did benefit from early endoscopy. These patients all had a visible vessel in the base of the ulcer associated with an 86% incidence of bleeding recurrence, and a 14% incidence of uncontrolled hemorrhage. All of these patients required surgery. Despite the unproved benefit from early endoscopy, this aggressive attitude remains. An emergency upper gastrointestinal barium study has many shortcomings. The barium may obstruct the view of the endoscopist, and this study fails to identify most of the superficial mucosal lesions. More importantly, barium in the gastrointestinal tract may obscure any subsequent attempts at emergency diagnostic angiography.

When a rapidly bleeding lesion makes accurate endoscopy impossible, angiography is the procedure of choice. Experimentally, a bleeding rate of at least 0.5–1.0 ml/min is necessary to demonstrate a bleeding site radiographically. In practice, however, a bleeding rate of 3–4 ml/min is probably necessary. Under these circumstances, the bleeding vessel may be identified in 50% to 70% of cases.[15] In addition to its diagnostic capabilities, angiography has useful therapeutic modalities. These include the intra-arterial infusion of vasopressin and embolization of the bleeding vessel.

Medical management

A patient who presents with melena or hematemesis must undergo immediate simultaneous evaluation and resuscitation. While the history is being obtained and the physical examination performed, appropriate blood samples must be drawn, intravenous lines started, and a nasogastric tube passed. Other monitoring modalities that may be warranted include a mean arterial line, a central venous pressure line, a Foley catheter, and perhaps a Swan-Ganz catheter. A patient who has suffered a major bleeding episode should be admitted to an intensive care area and initially resuscitated with crystalloid solutions. If indicated, whole blood should be given after proper blood typing and crossmatching has been performed. In addition, fresh frozen plasma should also be available for patients with liver disease or for those who will require several units of blood. Once initial evaluation and resuscitation are underway, arrangements should be made for urgent endoscopy to delineate the source of bleeding.

Passing either a large nasogastric tube or an Ewald tube is helpful for many reasons. Return of a bloody gastric aspirate suggests that the bleeding point is proximal to the ligament of Treitz. More importantly, evacuation of clots from the stomach facilitates endoscopy. Thorough gastric lavage controls the hemorrhage in approximately two thirds of cases. Although iced saline is the most frequently used irrigating solution, Ponsky et al[16] advocate the use of room-temperature solutions. If saline lavage alone fails to control the hemorrhage, small amounts of norepinephrine may be added to the irrigating solution. Kiselow and Wagner[17] reported a 54% success rate with this technique.

The best prophylaxis for stress-induced upper gastrointestinal hemorrhage is yet to be determined. Antacids and cimetidine, either alone or in combination are the two most frequently used agents. The work of Priebe et al[18] and Hastings et al[19] demonstrated the efficacy of antacid therapy if the gastric pH was monitored hourly and maintained at 3.5 or above. Only one clinical trial has generated data to suggest that

antacids alone are more effective than cimetidine alone for bleeding prophylaxis.[20] A further implication of this study was that their combined use was additive. Therefore, in a patient who is at risk for stress, upper gastrointestinal bleeding should be treated with either of these agents alone or more preferably in combination.

The management of an acute variceal hemorrhage continues to be challenging. After the stomach has been thoroughly lavaged and the diagnosis of bleeding esophageal varices has been confirmed by endoscopy, a Pitressin intravenous drip is usually begun. If this does not control the hemorrhage, either a Sengstaken-Blakemore tube or a Minnesota tube is passed into the stomach in an attempt to effect tamponade at the bleeding site. Aspiration is less of a problem with the latter tube because a fourth lumen has been added to aspirate any blood that may accumulate in the esophagus. If inflation of the gastric balloon does not control the bleeding, the esophageal balloon is inflated to a pressure of 35–40 mm Hg. If this controls the bleeding, the esophageal component is deflated after 24 hours, and if the bleeding remains controlled, the gastric balloon is deflated after a subsequent 24 hours. If the patient remains stable after this, the tube is removed. In Pitcher's series, in 92% of patients, bleeding stopped with the first tamponade, and in 62% of those in whom bleeding resumed, it stopped with a second tamponade.[21] Balloon tamponade is no longer indicated in the treatment of a Mallory-Weiss tear for fear of converting a partial-thickness tear into a full-thickness perforation.[22]

The endoscope is no longer simply a diagnostic tool. The rigid endoscope has been used for several years to sclerose esophageal varices. In 1973, Johnston and Rodgers[23] reported a 15-year experience with this technique in which hemorrhage was controlled in 93% of 117 patients with a 12% mortality. More recently, the fiberoptic endoscope has been used for this same problem with similar results. Endoscopic electrocoagulation and laser photocoagulation are new techniques that seem to be promising. In Gaisford's experience, electrocoagulation was successful in controlling hemorrhage in 92% of 71 patients.[24] Of the six patients in whom bleeding recurred, only two did not respond to a second application of this technique. Laser photocoagulation is currently being studied at this institution.

In recent years, new angiographic techniques have been added to our therapeutic regimen for the treatment of upper gastrointestinal bleeding. These include intra-arterial infusion of vasopressin and therapeutic embolization. The former modality has been shown to be effective in controlling the bleeding from Mallory-Weiss tears and acute gastric mucosal lesions approximately 80% of the time. However, the intra-arterial infusion of vasopressin has been successful in controlling duodenal ulcer bleeding in only 40% of patients.[25] The much lower success rate in this latter group reflects the difficulty in controlling the bleeding from the gastroduodenal artery. Intra-arterial embolization has a reported success rate of 65% to 70% in the distribution of the gastroduodenal artery. Autologous blood clot, Gelfoam, polyvinyl alcohol, and the Gianturco coil are but a few of the agents used for embolization. Athanasoulis[25] recommends infusing vasopressin into the left gastric artery to control acute gastric mucosal lesions, and embolization of the gastroduodenal artery to control bleed-

ing from a duodenal ulcer. Few complications have been associated with these techniques.

Surgical management

Several authors, including Palmer,[26] and Himal et al,[27] advocate aggressive management of upper gastrointestinal bleeding. This includes urgent endoscopy, and deciding early which patients are likely to continue bleeding or are at higher risk, and therefore should be considered for prompt operative intervention. Factors favoring early surgery include hemodynamic instability after three units of blood, the loss of five to six units of blood, a transfusion requirement of three units of blood per day, gastric ulcer, or bleeding after initial stabilization.[28] A further indication for aggressive management includes significant associated medical diseases. Using this approach, Himal et al[27] reported a reduction in mortality rate from 12.5% to 6%. In contrast to bleeding ulcers, acute gastric mucosal lesions are generally treated conservatively. Relatively few of these patients require an operation to control bleeding. Unfortunately, in most series, the mortality rate for emergency surgery still varies from 10% to 50%, depending on the lesion treated, age of the patient, and coexisting medical problems.[28]

Truncal vagotomy, pyloroplasty, and oversewing the bleeding ulcer constitute the most frequently performed operation for a bleeding duodenal ulcer. An alternative procedure is a truncal vagotomy and antrectomy. However, in a bleeding and unstable patient, the former procedure is much faster and associated with less morbidity and mortality. These two procedures were compared in terms of operative mortality, morbidity, and the incidence of bleeding recurrence.[29] The average operative mortality rate for gastric resection was 20%, twice that for vagotomy and pyloroplasty. Also, rates of bleeding recurrence for these two operations were not dissimilar, ranging from 8% to 33% for vagotomy and pyloroplasty, and 5% to 26% after gastric resection. Some authors[30, 31] have favored the removal of giant posterior, penetrating duodenal ulcers because of a 30% bleeding resumption rate after vagotomy and pyloroplasty.

It is more difficult to determine the best operation for a bleeding gastric ulcer than for a bleeding duodenal ulcer. In the good-risk patient who has not suffered a marked blood loss and is hemodynamically stable, removal of the ulcer by a hemigastrectomy and vagotomy is probably the best operation.[10] However, patients who are poor risks, or hemodynamically unstable, or have an ulcer high on the lesser curvature, are not amenable to gastric resection without a marked increase in operative morbidity and mortality. Therefore, wedge-excision or oversewing of the bleeding point in the ulcer in addition to a vagotomy and pyloroplasty is recommended in this group of patients. In a review by Sapala and Ponka,[32] the recurrent ulcer rate after a gastric resection varied from 0% to 17%, and was 1% to 15% after vagotomy and pyloroplasty. Schiller's[33] series confirms the higher mortality associated with resection. He reported no mortality after a vagotomy and pyloroplasty, 10% after a Billroth I resection, and almost 20% after a more extensive resection.

Acute gastric mucosal lesions vary from a single bleeding ulcer to diffuse hemorrhagic gastritis, and the proper operation must be tailored to each case. For instance, performing a vagotomy, pyloroplasty, and oversewing the bleeding point is appropriate for an isolated

Table 4. Choices of operation for acute gastric mucosal lesions

	Mortality (%)	Bleeding Recurrence (%)
Vagotomy and pyloroplasty[35]	27	29
Hemigastrectomy with vagotomy[35]	35	45
Gastric devascularization[36]	38	9.5
Near-total gastrectomy[34]	100	0

erosion, whereas a nearly total gastrectomy may be necessary to control the hemorrhage from a diffusely eroded stomach. Because of the high incidence of postoperative bleeding in these critically ill patients, some authors favor more aggressive resections, despite the higher mortality rate.[34] These workers maintain that the higher operative mortality is more acceptable than the risk of a second or third operation for bleeding. A comparison of bleeding recurrence and mortality rates for the various procedures is shown in *Table 4.*

Although intravenous vasopressin and balloon tamponade, either alone or in combination, initially control acutely bleeding esophageal varices in 85% of patients, the remainder fail to be controlled with these modalities and require emergency operation. Either a portosystemic shunt or ligation procedure may be performed. A standard portacaval shunt or a mesocaval shunt are the two most frequently performed diversion procedures in the acute setting. A ligation procedure consists of oversewing the bleeding varices, interrupting the column of varices in the distal esophagus either by hand suture or by the stapling device, devascularization of both the greater and lesser curvatures of the stomach, and occasionally a splenectomy. Although most patients survive the operation, at least 50% die in 2–4 weeks, predominantly of liver failure. This high operative mortality reflects the limited hepatic reserve of the cirrhotic liver in these patients. Moreover, an emergency operation for bleeding esophageal varices is performed only if the patient continues to bleed despite maximal nonsurgical measures.

In most instances, bleeding from a Mallory-Weiss tear can be controlled by medical management. However, surgical intervention is occasionally necessary. In these instances, a large gastrotomy is necessary to expose the distal esophagus and the gastroesophageal junction. The bleeding point is suture ligated and additional tears are sought. Also, the stomach is inspected for any additional mucosal lesions, since they have been reported in 80% of patients requiring an operation for a Mallory-Weiss tear.[8]

Summary

A review of the current management of acute upper gastrointestinal hemorrhage raises many questions. For instance, prospective studies have not supported the hypothesis that early endoscopy lowers the mortality and morbidity associated with upper gastrointestinal bleeding. Whether antacids or cimetidine should be used alone or in combination is under investigation. Finally, because acute gastric mucosal lesions are so diversified, a single operation of choice cannot be proposed. Until these issues are resolved, an aggressive attitude must be maintained.

A patient with an acute upper gastrointestinal hemorrhage must have a rapid and thorough simultaneous evaluation and resuscitation. This includes

assessing hemodynamic stability, replacing volume with crystalloid solution and blood, thoroughly lavaging the stomach, and arranging early endoscopy. In many instances, the patient may require monitoring available only in an intensive care unit. The gastric pH should be kept at ≥4.0 with antacids, and cimetidine may be given intravenously. Variceal hemorrhage that does not cease spontaneously is first treated by intravenous vasopressin. If the bleeding continues, a Sengstaken-Blakemore tube is inserted and the balloons are inflated. An emergency operation, either a portosystemic shunt or ligation procedure, is attempted only if these measures fail. When endoscopy is not diagnostic because of the rate of bleeding or some other technical problem, angiography is helpful both from a diagnostic and therapeutic standpoint. Intra-arterial vasopressin and embolization are valuable therapeutic modalities, especially in poor-risk patients. Early surgery should be advocated for patients with lesions that are not likely to stop bleeding with medical management or who have significant associated medical diseases. Finally, acute gastric mucosal lesions and Mallory-Weiss tears should be treated conservatively.

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