Review Article

Upper Gastrointestinal Bleeding

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Upper Gastrointestinal Bleeding (UGIB) commonly presents with hematemesis (vomiting of blood or coffee-ground like material) and/or melena (black, tarry stools). In comparison, hematochezia (bright red or maroon colored blood or fresh clots per rectum) is usually a sign of a lower GI source (defined as distal to the ligament of Treitz). Although helpful, the distinctions based upon stool color are not absolute since melena can be seen with proximal lower GI bleeding, and hematochezia can be seen with massive upper GI bleeding. [1,2,3]

In this article we will review the etiology, approach to patient with UGI bleed, diagnostic studies, and the various treatment options for various causes of UGIB.

Etiology

Etiology of acute upper gastrointestinal bleeding can be tabulated as follows: [4]

Ulcerative or erosive

Peptic ulcer disease

1. Idiopathic
2. Drug induced
   - Non steroidal anti inflammatory drugs
   - Aspirin
3. Infectious
   - Helicobacter pylori
   - Cytomegalovirus
   - Herpes simplex virus
4. Stress-induced ulcer
5. Zollinger Ellison Syndrome

Esophagitis

1. Peptic
2. Infectious
   - Candida albicans
   - Herpes simplex virus
   - Cytomegalovirus
   - Miscellaneous
3. Pill-induced
   - Alendronate
   - Tetracycline
   - Quinidine
   - Potassium chloride
   - Aspirin
   - Nonsteroidal antiinflammatory drugs

Portal hypertension

1. Esophageal varices
2. Gastric varices
3. Duodenal varices
4. Portal hypertensive gastropathy

Arterial, venous, or other vascular malformations

1. Idiopathic angiomas
2. Osler-Weber-Rendu syndrome
3. Dieulafoy's lesion
4. Watermelon stomach (gastric antral vascular ectasia)
5. Radiation-induced telangiectasia
6. Blue rubber bleb nevus syndrome

Traumatic or post-surgical

1. Mallory-Weiss tear
2. Foreign body ingestion
3. Post-surgical anastamosis
4. Aortoenteric fistula
5. Post gastric/duodenal polypectomy

Tumors

1. Benign
   - Leiomyoma
   - Lipoma
   - Polyp (hyperplastic, adenomatous, hamartomatous)
2. Malignant
   - Adenocarcinoma
   - Mesenchymalneoplasm
   - Lymphoma
   - Kaposi's sarcoma
   - Carcinoid
   - Melanoma
   - Metastatic tumor
3. Miscellaneous
   - Hemobilia
   - Hemosuccus pancreaticus

Approach to patient with UGI bleed

Management of severe upper gastrointestinal bleeding involves the following step

1. Assessment of severity

The initial step is the assessment of the haemodynamic status. A systolic blood pressure less than 100 mmHg identifies a high risk patient with severe acute bleeding. A heart rate over 100 beats/min with a systolic blood pressure over 100mmHg signifies moderate blood loss. A normal systolic blood pressure and heart rate suggest relatively minor hemorrhage. A postural drop >10mmHg, reduced

urine output, pallor, confusion, profuse sweating should alert the physician.[5]

2. Resuscitation and stabilization

All patients with hemodynamic instability (shock, orthostatic hypotension, decrease in hematocrit of at least 6 percent, or transfusion requirement over two units of packed red blood cells) or active bleeding (manifested by hematemesis, bright red blood per nasogastric tube, or hematochezia) should be admitted, preferably to an intensive care unit for resuscitation and close observation with automated blood pressure monitoring, ECG monitoring, and pulse oximetry. Adequate resuscitation and stabilization is essential prior to endoscopy to minimize treatment-associated complications . Two large caliber (16 gauge or larger) peripheral catheters or a central venous line should be inserted for intravenous access.[6] Gastroenterological consultation should be obtained. Surgical consultation should be considered based upon the timing and availability of therapeutic endoscopy and in patients with massive bleeding in whom endoscopy may be less successful.

High-risk patients (e.g., those who are elderly or who have severe co-morbid illnesses such as coronary disease or cirrhosis) should receive packed red blood cell transfusions to maintain the hematocrit above 30 percent. Patients who are elderly or have known cardiovascular disease are at increased risk for a myocardial infarction and should thus be monitored appropriately; consideration should be given to ruling out a myocardial infarction.

Young and otherwise healthy patients should be transfused to maintain their hematocrit above 20 percent. Patients with active bleeding and a coagulopathy (prolonged prothrombin time with INR >1.5) or low platelet count (<50,000/microL) should also be transfused with fresh frozen plasma and platelets, respectively.[6] It is also essential to send the renal function tests and look for the urea to creatinine ratio. A ratio more than 30:1 should alarm the treating physician of a significant UGIB.

Nasogastric or orogastric tube lavage should be performed to remove particulate matter, fresh blood, and clots to facilitate endoscopy. Elective endo-tracheal intubation in patients with ongoing hematemesis or altered respiratory or mental status may facilitate endoscopy and decrease the risk of aspiration.

Both a gastroenterologist and a surgeon should be promptly notified of all patients with severe acute UGI bleeding.

Somatostatin, or its analog octreotide, which have been best studied in the treatment of variceal bleeding, may also reduce the risk of bleeding due to non variceal causes. It can be used as adjunctive therapy before endoscopy, or when endoscopy is unsuccessful, contraindicated, or unavailable.[7]

3. History taking

The physicians' impression of bleeding source is correct in only 40% of cases; hence history taking is mentioned after more important and imperative hemodynamic assessment and resuscitation. Signs of chronic liver disease implicate bleeding due to portal hypertension but a different lesion is found in 25% of cases with cirrhosis. A history of NSAID use or peptic ulcer disease suggests peptic ulcer. Acute bleed preceded by heavy alcohol ingestion or retching suggests a Mallory Weiss tear, though most patients have neither. Other important elements of history include a history of dysphagia, weight loss, abdominal aortic aneurysm or an abdominal aortic vascular graft.

Diagnostic studies

Upper panendoscopy is the diagnostic modality of choice for acute UGI bleeding.[8,9] Endoscopy is highly sensitive and specific for locating and identifying bleeding lesions in the upper gastrointestinal tract. In addition, once a bleeding lesion has been identified, therapeutic endoscopy can achieve acute hemostasis and prevent recurrent bleeding in most patients.

It is frequently helpful to irrigate the stomach prior to endoscopy to help remove residual blood and other gastric contents. However, despite irrigation, the stomach can be obscured with blood, potentially making it difficult to establish a clear diagnosis and/or perform therapeutic maneuvers. In patients in whom bleeding stopped spontaneously, a second-look endoscopy may be required to establish a diagnosis.

Erythromycin

Erythromycin promotes gastric emptying based upon its ability to be an agonist of motilin receptors. Treatment with erythromycin can be considered in patients who are likely to have a stomach full of blood such as those with severe bleeding. A reasonable dose would be to give 3 mg/kg intravenously over 20 to 30 minutes, 30 to 90 minutes prior to endoscopy. [10]

Risks of endoscopy

Risks of upper endoscopy include aspiration, adverse reactions to conscious sedation, perforation, increasing bleeding while attempting therapeutic intervention. The risks versus benefits of upper endoscopy should be considered in high risk patients, such as those who have had a recent myocardial infarction. [11,12]

Table-1: Endoscopic predictors of recurrent ulcer hemorrhage.[4]

Endoscopic stigmata of recent hemorrhage	Prevalence (%)	Risk of re-bleeding on medical managemnt (%)
Active arterial bleeding	10	90
Non-bleeding visible vessel	25	50
Adherent clot	10	25-30
Oozing without visible vessel	10	10 -20
Flat spot	10	7-10
Clean ulcer base	35	3-5

Other diagnostic tests

Other diagnostic tests for acute UGI bleeding include angiography and a tagged red blood cell scan, which can detect active bleeding.[13,14] UGI barium studies are contraindicated in the setting of acute UGI bleeding because they will interfere with subsequent endoscopy, angiography, or surgery.[8]

Risk stratification

Endoscopic, clinical, and laboratory features may be useful for risk stratification of patients who present with UGI bleeding.[15-21]

Combining this information, several investigators have developed decision rules and predictive models that permit identification of individuals who are at low risk for recurrent or life-threatening hemorrhage. [22] Such individuals may be suitable for early hospital discharge or even outpatient care.

Acid suppression

An intravenous PPI given before endoscopic therapy in patients with UGI bleeding can reduce signs of bleeding and the need for endoscopic therapy. We suggest that patients with upper GI bleeding be started on an intravenous PPI. It can be started at presentation and continued until confirmation of the cause of bleeding after which the need for specific therapy can be determined.

Specific treatment

Specific treatment of patients with upper gastro-intestinal bleeding due to various causes is discussed separately.

Treatment of peptic ulcer disease

Endoscopic treatment

Thermal coagulation: Thermal coagulation achieves acute hemostasis and prevents re bleeding by coaptive coagulation of the underlying artery in the ulcer base.[23] Contact probes which are commercially available include the heater probe (Olympus Corp), Gold probe (Microvasive Corp), and BICAP probe (Circon ACMI).

Injection therapy: Injection therapy with absolute alcohol (98 percent, total volume <1.0mL) or epinephrine (1:10,000 dilution) is inexpensive and effective for acute hemostasis.[24] Addition of a sclerosant (eg, ethanolamine) confers no advantage over injection with epinephrine alone.[25,26] However, the re bleeding rate is high (approximately 18 percent) if epinephrine injections alone are performed.[27]

Saline injection: Injection of saline causes local tamponade, which can be effective in achieving hemostasis. However, saline injection alone was less effective at preventing recurrent bleeding compared to bipolar electro-coagulation in a randomized controlled trial involving 100 patients with high-risk bleeding ulcers .[28]

Combination therapy: Small randomized controlled trials suggest that combination therapy with epinephrine injection followed by mechanical methods of hemostasis (eg, thermal coagulation or placement of a hemoclip) appears to decrease the re bleeding rates compared with epinephrine injection or thermal methods alone for actively bleeding ulcers, those with an adherent clot, or non bleeding visible vessels.[24-28] If endoscopic hemostasis is unsuccessful, emergency surgery may be required.

Fibrin sealant: A relatively new approach involves the use of endoscopically injected fibrin sealant to achieve initial hemostasis and decrease the rate of re bleeding from peptic ulcers. An open label, multi-center, randomized trial of 854 patients with actively bleeding gastro duodenal ulcers compared the safety and efficacy of a single application of fibrin sealant, daily repeated doses of fibrin sealant until the visible vessel disappeared, or a single application of the sclerosant polidocanol. While the safety profiles of all three treatment strategies were similar, the patients who received multiple applications of fibrin sealant had significantly less re bleeding than the polidocanol group (15 versus 23 percent), and had fewer acute treatment failures (8 versus 13 percent).

Endoclips: The endoscopic application of hemoclips (endoclips) provides an alternative to the hemostatic methods described above. Once applied, the clips achieve hemostasis in a manner similar to surgical ligation.

Although experience is relatively limited compared with other hemostatic methods, the available data suggest that endoclips are as safe as other hemostatic methods, and can be considered as an option for patients with ulcer bleeding. Placement of an endoclip can also be of value even if an ulcer is not amenable to endoscopic therapy since it may serve as a radiologic marker for subsequent interventional radiology.

Argon plasma coagulation: Argon plasma coagulation (APC) has a theoretical disadvantage for the treatment of bleeding ulcers since it does not permit tamponade.

Acid suppression

A number of studies have investigated the role of acid suppression with an H2 antagonist or proton pump inhibitor in patients with bleeding ulcers.[23] A meta-analysis of 21 randomized controlled trials evaluating proton pump inhibitors for bleeding ulcers (with or without endoscopic therapy) found a significant and consistent reduction in the risk of re bleeding (OR 0.46, 95% CI 0.33-0.64) and the need for surgery (OR 0.59, 95% CI 0.46-0.76); there was no effect on mortality.[23] In contrast, studies on H2 antagonists have generally produced disappointing results. [27] A meta-analysis concluded that there was a possible minor benefit with intravenous H2 antagonists in bleeding gastric ulcers but no benefit with duodenal ulcers.[29] The relative efficacy of the proton pump inhibitors may be due to their superior ability to maintain a gastric pH at a level above 6.0, and thus protect an ulcer clot from fibrinolysis.

Refractory bleeding Although the majority of bleeding ulcers can be controlled endoscopically, some patients have refractory bleeding. [30,31] In one report, gastric ulcers along the lesser curvature and duodenal bulbar ulcers in the posterior wall appeared to be at greater risk for severe bleeding or re bleeding compared with ulcers in other locations because of their proximity to large underlying arteries (left gastric and posterior gastro duodenal arteries, respectively).[30] In addition, patients who presented with active hemorrhage, shock, and the lowest hemoglobin concentrations did less well than those without these risk factors. Factors that did not predict outcome of endoscopic therapy were a history of non steroidal anti inflammatory drug (NSAID) or aspirin use, coagulopathy, previous peptic ulceration, and concomitant cardio-respiratory disease. In another report, severe bleeding, active bleeding, fresh blood in the stomach and large ulcers were independent risk factors for therapeutic failure after injection of epinephrine plus heater probe treatment. [31]

Interventions such as surgery or angiography are indicated in patients who have active bleeding that is not stopped or slowed down significantly with endoscopic therapy. We perform repeat endoscopy and retreat the bleeding source for lesions that were initially controlled by endoscopic therapy. The patient is referred for surgery if the bleeding persists or re bleeding occurs after two therapeutic endoscopies.

Surgery

In addition to failure of endoscopic therapy, other indications for surgery for peptic ulcer hemorrhage include: Hemodynamic instability despite vigorous resuscitation (more than a three unit transfusion) Recurrent hemorrhage after initial stabilization (with up to two attempts at obtaining endoscopic hemostasis) Shock associated with recurrent hemorrhage Continued slow bleeding with a transfusion requirement exceeding three units per day.

Secondary or relative indications include rare blood type, difficult cross match, refusal of transfusion, shock on presentation, advanced age, severe co morbid disease, and chronic gastric ulcer as the origin of hemorrhage. These criteria also apply to elderly patients in whom prolonged resuscitation, large volume transfusion, and periods of hypotension are poorly tolerated.

Surgical treatments for peptic ulcer disease include oversewing of the artery plus truncal vagotomy and pyloroplasty, antrectomy, and gastrojejunostomy (Billroth procedure), and highly selective vagotomy. Emergency surgery for bleeding peptic ulcer disease involves oversewing of the ulcer (to ligate the bleeding artery) plus truncal vagotomy (to decrease acid secretion) and pyloroplasty (drainage procedure). More time consuming procedures, such as highly selective vagotomy, can be performed either at standard laparotomy or laparoscopically for non-emergency ulcer surgery.

Risk of recurrence

The risk of recurrent ulceration and bleeding depends upon characteristics of the ulcer, use of

endoscopic therapy, and the extent to which risk factors such as use of NSAIDs and H. pylori infection.

Treatment of Mallory Weiss tear

Although 40 to 70 percent of patients with bleeding Mallory-Weiss tear require blood transfusions, most tears heal spontaneously.[32] Endoscopic therapy is the first-line treatment of actively bleeding lacerations. Several hemostatic methods have been used to control bleeding. The results obtained with any modality depend upon technique, experience, and practice.

Injection therapy of various agents, including epinephrine,[32] ethanol, and other sclerosants have been used as monotherapy or in combination with thermal devices. Injection of epinephrine (1:10,000 to 20,000) diminishes arterial blood flow by a combination of vasoconstriction and edema permitting more effective thermal therapy. Epinephrine has also been used successfully in combination with polidocanol (a sclerosing agent). Bipolar or multipolar electrocoagulation (e.g., a bipolar probe at 15 watts, mild tamponade, and one second pulses) is the most popular thermal therapy. It is effective, safe, and inexpensive and most endoscopists have had experience with this technique.

Thermal coagulation should not be performed in patients with portal hypertension and esophageal varices since it may precipitate or worsen bleeding; sclerotherapy or endoscopic variceal ligation is preferable in such cases. Another limiting factor is that the esophagus lacks a serosa and may be very thin at the tear site. Thus, repeated coagulation should be avoided because of the risk of transmural injury and perforation.[33]

Another approach that has been described in case reports and small controlled trials is the use of endoscopic band ligation. Similar to variceal band ligation, the affected mucosa is suctioned into the ligating device after which the band is applied.[34]

Successful hemostasis using hemoclips has also been reported. Hemoclips were as effective as epinephrine injection in patients with spurting vessels or oozing lesions in a small controlled trial. Almost all patients will respond to endoscopic hemostatic therapy. Intravenous infusion of vasopressin, esophageal balloon tamponade, and angiographic arterial embolization have been used occasionally to control severe or refractory hemorrhage. Surgery, with over sewing of the bleeding vessel, may be necessary in those rare instances when bleeding cannot be controlled.

We place all patients on a proton pump inhibitor, although the benefit for preventing re bleeding in Mallory-Weiss syndrome has not been well-studied.

General principles of management of variceal bleed

The general principles of support of patients presenting with variceal bleeding such as transfusion, prevention of aspiration, and use of recombinant factors is presented separately.

The American Association for the Study of Liver Diseases (AASLD) has issued guidelines for management of variceal bleeding.[35] Acute GI hemorrhage in a patients with cirrhosis is an emergency that requires prompt attention with intravascular volume support and blood transfusions, being careful to maintain a hemoglobin of approximately 8 g/dL.

Antibiotics for patients with cirrhosis

Bacterial infections are present in up to 20 percent of patients with cirrhosis who are hospitalized with gastrointestinal bleeding; up to an additional 50 percent develop an infection while hospitalized. Such patients have increased mortality. According to AASLD short-term (maximum 7 days) antibiotic prophylaxis should be instituted in any patient with cirrhosis and GI hemorrhage. Oral norfloxacin (400 mg twice daily) or intravenous ciprofloxacin (in patients in whom oral administration is not possible) is the recommended antibiotic. In patients with advanced cirrhosis, intravenous ceftriaxone (1 g/day) may be preferable, particularly in centers with a high prevalence of quinolone-resistant organisms.

AASLD guideline

Pharmacological therapy somatostatin or its analogues octreotide and vapreotide; terlipressin), should be initiated as soon as variceal hemorrhage is suspected and continued for 3 to 5 days after diagnosis is confirmed. EGD, performed within 12 hours, should be used to make the diagnosis and to treat variceal hemorrhage either with EVBL or sclerotherapy. TIPS is indicated in patients in whom hemorrhage from esophageal varices cannot be controlled or in whom bleeding recurs despite combined pharmacological and endoscopic therapy. Balloon tamponade should be used as a temporizing measure (maximum 24 hours) in patients with uncontrollable bleeding for whom a more definitive

therapy (e.g., TIPS or endoscopic therapy is planned.

Intravenous vasopressin and its analogs

Intravenous vasopressin (0.4 U bolus followed by 0.4 to 1.0 U/min as an infusion) directly constricts mesenteric arterioles and decreases portal venous inflow, thereby reducing portal pressures. A number of studies have evaluated the role of vasopressin in the management of active hemorrhage. Vasopressin can achieve initial hemostasis in 60 to 80 percent of patients, but has only marginal effects on early re bleeding episodes and does not improve survival from active variceal hemorrhage.

However, the benefit of bleeding cessation may be counterbalanced by enhanced mortality due to extrasplanchnic vasoconstrictive properties and resultant myocardial, cerebral, and bowel and limb ischemia. Furthermore, there is evidence that the portal hypotensive effect of vasopressin is attenuated when variceal hemorrhage occurs. 36 These considerations plus the seemingly greater benefit with somatostatin or an analog have reduced the use of vasopressin in the management of variceal hemorrhage. Vasopressin is rarely used for the management of variceal hemorrhage.

The systemic vasodilator nitroglycerin has been concurrently administered in an attempt to avoid the adverse effects related to vasopressin-induced systemic vasoconstriction. Nitroglycerin is a potent coronary dilator and a systemic venodilator. When used in combination with vasopressin, it further accentuates the portal hypotensive actions of vasopressin, while reversing its systemic hemodynamic effects. Based upon data, intravenous vasopressin should be combined with intravenous nitroglycerin (10 to 50 microg/min). Nitroglycerin given transdermally may not be as effective when given with vasopressin.

Terlipressin: Terlipressin (triglycyl lysine vasopressin) is a synthetic analog of vasopressin that is released in a slow and sustained manner, permitting its administration via intermittent injections. At least 20 clinical trials have evaluated its efficacy. A meta-analysis found a statistically significant reduction in all cause mortality compared with placebo (RR 0.66, 95 percent CI 0.49 to 0.88). 37 Only a few studies directly compared terlipressin to somatostatin or endoscopic treatment but these suggest that terlipressin has similar efficacy in control of acute bleeding.

Somatostatin and its analogs

Somatostatin inhibits the release of vasodilator hormones, such as glucagon, indirectly causing splanchnic vasoconstriction and decreased portal inflow. It has a short half-life and disappears within minutes of a bolus infusion; octreotide is a long-acting analog of Somatostatin.

Pharmacodynamics: Following a bolus injection of either somatostatin or octreotide, portal venous inflow, portal pressures, azygos flow, and intra variceal pressures decrease within seconds. Of these effects, the changes in portal pressures, as measured by wedged hepatic pressures, are most variable and the decrease in collateral flow (azygos flow) are most consistently observed.

Variceal hemorrhage is associated with an increase in intestinal blood flow, presumably mediated by pathways that are activated by the presence of blood, a high protein substance, in the gut. Octreotide can blunt this response for at least 48 hours. In addition, activation of somatostatin receptors may decrease the rebound increase in portal venous pressure that occurs when blood enters the gastrointestinal tract and during correction of hypovolemia.

Endoscopic treatment

Endoscopic therapy is currently the definitive treatment of choice for active variceal hemorrhage. [38] It can be performed at the same time as diagnostic endoscopy at the bedside by virtually all trained gastroenterologists. Two forms of endoscopic treatment are available: sclerotherapy and variceal band ligation. Sclerotherapy involves injection of a sclerosant solution into the varices using a freehand technique. A number of sclerosant solutions are available and one has not been found to be superior to another; the volume and frequency of injections also vary widely. We use 1 to 2 mL of 5 percent sodium morrhuate for a total of 12 to 20 mL per session. Variceal band ligation is similar to hemorrhoidal banding; it involves placing small elastic bands around varices in the distal 5 cm of the esophagus

What to do when endoscopic treatment fails

Definitions: Failure of endoscopic treatment is generally considered within two time frames according to consensus definitions:[39] Within six hours - when the following factors are present:

a) transfusion of four units of blood or more, and

b) inability to achieve an increase in systolic blood pressure of 20 mmHg or to 70 mmHg or more, and/or

c) a pulse reduction to less than 100/min or a reduction of 20/min from the baseline pulse rate.

After six hours - when the following factors are present:

present:
a) occurrence of hematemesis,
b)reduction in blood pressure of more than 20 mmHg from the six hour point and/or
c)increase of pulse rate of more than 20/min from the six hour point on two readings one hour apart,
d) transfusion of two units of blood or more (over and above the previous transfusions) required to increase the hematocrit above 27 percent or Hb to above 9g/dL.
These criteria do not require documentation of bleeding and can potentially be met without continued bleeding. Thus, it is necessary to exercise clinical judgment.
Any bleeding that occurs more than 48 hours after the initial admission for variceal hemorrhage and is separated by at least a 24 hour bleed-free period is considered to represent re bleeding. Re bleeding that occurs within six weeks from the onset of active bleeding is considered to be "early re bleeding" while re bleeding episodes at later time points are referred to as "late re bleeding". The highest risk for failure to control bleeding or early re bleeding is in the first 72 hours after the onset of bleeding.
Approach: Emergent endoscopic treatment fails to control bleeding in 10 to 20 percent of patients. These patients are at high risk for exsanguination and other complications related to active bleeding. Most patients have already received a trial of pharmacologic treatment by the time a diagnosis of failed endoscopic treatment is established. There are no data to support the use of higher doses of octreotide or somatostatin in those who have failed endoscopic treatment. A second attempt at endoscopic hemostasis can be made (e.g., band ligation for failed sclerotherapy), an approach supported by widely accepted guidelines.[40] If bleeding is not quickly and effectively stopped, more definitive therapy must be instituted immediately with balloon tamponade or some type of porto systemic shunt.

Balloon tamponade

Balloon tamponade is an effective way to achieve short-term hemostasis. Three balloons have been used: the Sengstaken-Blakemore tube (which has a 250 cc gastric balloon an esophageal balloon and a single gastric suction port), the Minnesota tube (a modified Sengstaken-Blakemore tube with an esophageal suction port above the esophageal balloon), and the Linton-Nachlas tube (which has a single 600 cc gastric balloon).[41] Balloon tamponade also appears to be less successful in patients who have failed pharmacologic therapy and in patients with early re bleeding.

Surgery

A vast body of literature exists on the use of surgery for the control of variceal hemorrhage. There are two basic types of operations: shunt operations and non shunt operations. Shunt operations can be categorized as follows:
Nonselective: those that decompress the entire portal tree and divert all flow away from the portal system, such as porta caval shunts**.**
Selective: those that compartmentalize the portal tree into a decompressed variceal system while maintaining sinusoidal perfusion via a hypertensive superior mesenteric-portal compartment, such as a distal splenorenal shunt**.**
Partial: those that incompletely decompress the entire portal tree and thereby also maintain some hepatic perfusion**.**
The ideal patient for surgical therapy is one with well preserved liver function who fails emergent endoscopic treatment and has no complications from the bleeding or endoscopy. The choice of surgery is usually dependent on the training and expertise of the surgeon. Although a selective shunt has some physiologic advantages, it may significantly exacerbate marked ascites. Thus, a porta caval shunt would be preferable in patients with marked ascites.

Transjugular intrahepatic portosystemic shunts

The transjugular intrahepatic portosystemic stent shunt (TIPS) is created by passing a needle catheter (Colapinto catheter) via the transjugular route into the hepatic vein and wedging it there. The needle is then extruded and advanced through the liver parenchyma to the intra hepatic portion of the portal vein. TIPS function like side-to-side surgical porta caval shunts without general anesthesia or major surgery.
Large-scale, randomized prospective trials are needed to establish the roles of surgery and TIPS in average risk subjects with active hemorrhage who have failed endoscopic and medical treatment. At present, TIPS is generally preferred in patients who are poor risks for surgery but the relative roles of TIPS and surgery in those who are good candidates (and where highly-trained surgeons are available) is less clear.

Gastric varices

The AASLD guidelines recommend the following:[35]
In patients who bleed from gastric fundal varices, endoscopic variceal obturation using tissue adhesives

such as cyanoacrylate is preferred, where available. Otherwise, EVL is an option. A TIPS should be considered in patients in whom hemorrhage from fundal varices cannot be controlled or in whom bleeding recurs despite combined pharmacological and endoscopic therapy.

Successful hemostasis and obliteration of gastric varices has been reported with intra variceal injections of sclerosant, absolute alcohol, fibrin glue, and cyanoacrylate.

Another promising approach is the intra variceal injection of thrombin.[42] One of the largest series included 52 patients with bleeding gastric varices who were treated by intravariceal injections of bovine thrombin (average of 10-70 IU administered during two treatment sessions). Initial hemostasis was achieved in 94 percent. Bleeding related mortality within 72 hours of the index bleed was 6 percent. After six weeks of follow-up, 9 of 49 surviving patients (18 percent) re bled, and one further patient died.

Angiograghic control of bleeding

In the overwhelming majority of patients with upper or lower GI bleeding, the bleeding either resolves spontaneously or can be controlled endoscopically. However, some patients have persistent bleeding that requires angiographic intervention either to locate or control the source of bleeding. Arterial GI bleeding can be controlled by the selective arterial infusion of vaso constrictive drugs, by embolization of particulate matter into the bleeding artery, or by a combination of these techniques.

Intra arterial vasopressin initially controls app. 70 to 80 percent of cases of gastric arterial hemorrhage, regardless of whether the infusion is administered via the left gastric or celiac arteries. Most patients will respond to a vasopressin infusion of 0.2 pressor units/minute. Repeat angiography is performed 30 minutes later: if bleeding persists, the rate is increased to 0.4 units/minute; if bleeding is controlled, the infusion is continued in an intensive care setting for 24 to 36 hours, and then tapered over 24 hours.[43] Patients not responding to an infusion rate of 0.4 pressor units/minute are unlikely to respond to higher doses and require alternative therapies.

Embolization should only be performed by interventional radiologists experienced with this technique because of the risk of causing bowel wall ischemia and infarction. The vessel must be accessible for selective catheterization of the bleeding site.

Outcome and recommendations

The outcome of angiographic therapy largely depends upon the source of bleeding. The respective success rates with vasopressin and embolization are: Mallory-Weiss tears 74 and 82 percent.[44] Severe gastritis and stress ulcers 69 and 78 percent.[45] Gastric lesions 62 and 66 percent.[44] Lower gastrointestinal lesions as high as 90 and 100 percent.[46]

The choice between vasopressin and embolization should be individualized for each patient, also considering angiographer experience. Vasopressin infusion is generally preferred because of ease of use in the absence of one of the indications for embolization noted above. This is particularly true in hemorrhagic gastritis because of its diffuse peripheral vaso constrictive effect.

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