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GI BLEEDING: IDENTIFYING THE SOURCE

When a patient presents with gastrointestinal (GI) bleeding, the first priority is to assess the patient's hemodynamic status and, if necessary, begin resuscitative measures. Only after this is done should one attempt to find the cause of the bleeding and try to stop it.

Upper GI bleeding stops spontaneously in 80% of patients who present with it; however, the mortality rate has remained constant at approximately 10%. Factors that predict the patient's risk of death are the severity of the bleeding, age over 60, concurrent illness, and onset of bleeding in the hospital. Patients rarely die of exsanguination; rather, they generally die of decompensation from another illness.

UPPER GI VS LOWER GI BLEEDING

Hematemesis signifies upper GI bleeding, melena is also almost always due to upper GI bleeding, and hematochezia usually means lower GI bleeding. However, the color of blood in the stool relates to how long the blood has been in the GI tract, not to the origin of the bleeding: blood must remain in the GI tract for 14 hours before melena develops. The blood urea nitrogen concentration may be increased in upper GI bleeding because of volume loss and because of protein absorption from the small intestine.

If the site of bleeding is uncertain, a nasogastric tube should be inserted and the aspirate checked for blood. Presence of blood in the aspirate confirms an upper GI source, but absence of blood does not rule it out: a nonbloody aspirate can be seen in 3% to 16% of patients with upper GI bleeding, usually from a duodenal ulcer. A report of bile in the aspirate does not rule out duodenal bleeding either, since physicians often are incorrect in their assessment.

ENDOSCOPY EMERGING FOR DIAGNOSIS, THERAPY

Endoscopy is emerging as a means to find and treat the source of upper GI bleeding. In contrast, no medical therapy is documented effective in stopping active bleeding or preventing recurrent bleeding. Lavage is no more effective than doing nothing for stopping bleeding, although it can be used to clean the stomach before endoscopy. There is no evidence that antacids given via the nasogastric tube or intravenous H_2 blockers make any difference in the outcome.

DIAGNOSING UPPER GI BLEEDING

Ulcers account for 50% of acute upper GI bleeding, varices account for 10% to 30%, and Mallory-Weiss tears account for most of the rest. Gastritis and cancer are uncommon causes of major GI bleeding.

Barium studies of the upper GI tract are less accurate than endoscopy, as they miss gastric ulcers, Mallory-Weiss tears, hemorrhages, erosions, and varices. Also, they do not permit one to actually view the lesion. Finally, one should never use barium in acute upper GI bleeding because it may interfere with subsequent studies such as angiography or endoscopy.

The only disadvantage of endoscopy is its expense. Early studies that showed no benefit from endoscopy illustrate the dilemma of an accurate diagnostic tool for a condition that has no effective treatment. However, more recent studies do document its efficacy.

ULCERS: APPEARANCE DETERMINES RISK

It is not enough to document that an ulcer is present: most management decisions are based on the appearance of the ulcer base. White-based ulcers hardly ever rebleed, and patients with these can be sent home immediately after stabilization. The incidence of rebleeding is higher if there is a flat spot or

clot in the ulcer base: 5% to 20%. These patients can be admitted to a regular hospital unit instead of an intensive care unit. In contrast, patients with a nonbleeding visible vessel in the ulcer have a 40% to 45% chance of rebleeding. If left untreated, one third will require urgent surgery.

Endoscopic therapy has provided major advances in treating bleeding ulcers. The bipolar electrocoagulation probe and the heater probe have supplanted the yttrium-aluminum-garnet (YAG) laser. These thermal contact devices can stop bleeding 90% of the time and should be used only in patients with active bleeding or a nonbleeding visible vessel. They are not indicated for patients at lower risk. Another endoscopic approach is to inject epinephrine or sclerosing agents directly into the base of a bleeding lesion.

All these approaches control bleeding equally well, and a meta-analysis of trials of therapeutic endoscopy for nonvariceal upper GI hemorrhage showed a significant effect on mortality in patients with active bleeding or with nonbleeding visible vessels. Afterward, patients should receive maintenance therapy with an H_2 blocker to prevent recurrence, and most experts recommend eradicating *Helicobacter pylori* if it is present.

VARICEAL HEMORRHAGE: TIP OF THE ALCOHOLIC ICEBERG

Although less common than ulcers, varices necessitate more blood transfusions and cause more rebleeding, longer hospital stays, and a higher death rate. Many are due to alcoholism, but this may be the tip of the iceberg. No more than 15% of heavy drinkers get cirrhosis, only half of people with cirrhosis get varices, and no more than a third of people with varices bleed from them. The short-term mortality rate is 15% to 40%, and the long-term survival rate may not be better than in many cancers.

Intravenous vasopressin is a standard treatment, but it has not been shown to prolong survival nor even definitely to control bleeding. Further, it has considerable cardiac side effects, and nitroglycerin should be used with it to improve its efficacy and decrease its cardiovascular side effects. Octreotide, a new analog of somatostatin, may be beneficial and is safer than vasopressin or balloon tamponade. Balloon tamponade stops bleeding at least 90% of the time, but bleeding usually recurs. This uncomfortable therapy can cause major side effects such as esophageal rupture, aspiration pneumonia, and airway obstruction.

One should never empirically treat a patient with vasopressin or balloon tamponade, even if the patient has cirrhosis and varices, because there is a good likelihood the varices are not the source of the bleeding. I would recommend performing endoscopy first to establish the diagnosis, and then perhaps performing endoscopic therapy.

Long-term sclerotherapy is more effective than conservative therapy. Patients must return weekly for sclerotherapy until the varices are eradicated, and then every 3 to 6 months for the rest of their lives for endoscopy and possible retreatment if the varices recur.

Ligation therapy should produce fewer complications than sclerotherapy. This recently introduced technique involves placing an elastic band around the base of the varix. In studies to date, ligation therapy has been at least as effective as sclerotherapy, and it requires fewer sessions. Unfortunately, the endoscope must be withdrawn to reload after each band is placed.

Transjugular intrahepatic portasystemic shunting has become popular, but it has disadvantages, and no controlled studies of its effectiveness have been published. Its main role may be as a bridge until the patient can undergo liver transplantation. It relieves portal hypertension, but hepatic encephalopathy is a danger. These shunts tend to become stenosed, and up to 50% need to be redone by 6 to 12 months.

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SUGGESTED READING

Cook DJ, Guyatt GH, Salena BJ, Laine LA. Endoscopic therapy for acute nonvariceal upper gastrointestinal hemorrhage: a meta-analysis. Gastroenterology 1992; **102**:139–148.

Laine L. Rolling review: upper gastrointestinal bleeding. Aliment Pharmacol Ther 1993; **7**:207–232.

Laine L, el-Newihi HM, Migikovsky B, Sloane R, Garcia F. Endoscopic ligation compared with sclerotherapy for the treatment of bleeding esophageal varices. Ann Intern Med 1993; **119**:1–7.