Approach to the Patient With Gastrointestinal Bleeding

INTRUDUCTION

A cute upper gastrointestinal hemorrhage, which is defined as bleeding proximal to the ligament of Treitz, is a prevalent and clinically significant condition with important implications for health care costs worldwide.

Negative outcomes include rebleeding and death, and many of the deaths are associated with decompensation of coexisting medical conditions precipitated by the acute bleeding event.

This review focuses specifically on the current treatment of patients with acute bleeding from a peptic ulcer.

Epidemiology

The annual rate of hospitalization for acute upper gastrointestinal hemorrhage in the United States is estimated to be 160 hospital admissions per 100,000 population, which translates into more than 400,000 per year. In most settings, the vast majority of acute episodes of upper gastrointestinal bleeding (80 to 90%) have non variceal causes, with gastroduodenal peptic ulcer accounting for the majority of lesions

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A number of studies have suggested that the annual incidence of bleeding from a peptic ulcer may be decreasing worldwide, yet other recent population based estimates have suggested that the incidence is about 60 per 100,000 population, with an increasing proportion of episodes related to the use of aspirin and nonsteroidal antiinflammatory medications.

Moreover, peptic ulcer bleeding is seen predominantly among the elderly, with 68% of patients over the age of 60 years and 27% over the age of 80 years.

Mortality associated with peptic ulcer bleeding remains high at 5 to 10%.

Estimated direct medical costs for the in-hospital care of patients with bleeding from a peptic ulcer total more than $2 billion annually in the United States.

Acute Gastrointestinal Bleed

- Acute Upper GI Bleed
  - 8-10% overall mortality, 30-40% in recurrent bleeders
  - Differential
    - Peptic Ulcer Disease – 50%
      - Bleeding if erodes into lateral wall of a vessel
      - *Helicobacter pylori* and NSAIDs
    - Gastritis / Duodenitis
      - Inflammation or Erosion (<5mm, doesn't traverse the muscularis)
      - NSAID ingestion, Stress (ICU setting), EtOH, Chemotherapy

Acute Upper GI Bleed

- Portal Hypertension
  - Esophageal and Gastric Varices
    - Up to 33% of patients with cirrhosis will have variceal hemorrhage
    - Up to 50% of patients die of their initial esophageal hemorrhage
    - Up to 66% with bleeding varices die within one year
    - Isolated gastric varices may be caused by splenic vein thrombosis
  - Portal Gastropathy
    - Mosaic, snakeskin-like mucosa caused by engorged vessels

Acute Upper GI Bleed

- Miscellaneous Causes
  - Mallory-Weiss Tears – caused by retching, often after EtOH ingestion
  - Esophagitis and Esophageal Ulcers
    - GERD, radiation, infection (Candida & HSV), pill trauma, iatrogenic
  - Vascular Ectasia and Angiodysplasia
    - Advanced age, CRF, radiation, Osler-Weber-Rendu syndrome, watermelon stomach
  - Dieulafoy's lesions
  - Aortoenteric Fistula – after aortic graft surgery
    - "herald" bleed before fatal exsanguination
  - Hemobilia and Humosuccus Pancreaticus
    - Liver or pancreatic source through ampulla of Vater
  - Neoplasm – rare, usually chronic bleed
  - Epistaxis, hemoptysis, oral lesions, factitious blood ingestion
  - No source identified

Workup of Acute Upper GI Bleed

- Resuscitation
  - Shock (resting hypotension) ~ 20-25% volume loss
  - Orthostatic tachycardia/hypotension ~ 10-20% volume loss
  - Two large bore IV's or central line
  - Nasogastric Tube
    - Bright-red aspirate that does not clear with lavage – emergent upper endoscopy as mortality = 30%
    - Coffee grounds – endoscopy once stabilized
    - May negative in up to 25% of upper GI bleeds
  - Colloid - Normal Saline/Lactated Ringers – ASAP
  - Packed RBC's – (only 350cc/unit) – to HCT 30% (elderly), 20-25% (healthy)
  - Platelets, and FFP as needed

Workup of Acute Upper GI Bleed

History and Physical – prior disease, interventions, meds, and stigmata

- Risk of NSAID's increases with:
  - Advanced age
  - Prednisone
  - Anticoagulation
  - EtOH
  - Alendronate

- Laboratory Testing
  - CBC, CMP, PT, PTT
    - Initial H/H may be normal because loss of both cells and volume.
    - With volume replacement and equilibration with extravascular fluid this will decrease over 24-72 hours.
    - Azotemia out of proportion to creatinine reflects intestinal absorption of blood and reduction in the GFR due to volume loss.
    - Abnormal LFT's - suspect cirrhosis with portal hypertension

Adverse Prognostic Variables

- Increasing Age
- Increasing number of comorbid conditions
- Cause of bleeding (variceal > others)
- Red blood in emesis or stool
- Shock or hypotension on presentation
- Increasing number of units of blood transfused
- Active bleeding at time of endoscopy
- Bleeding from large ulcers (>2.0cm)
- Onset of bleeding in the hospital
- Emergency surgery

Sleisenger & Fordtran 7th edition – Table 13-3

Clinical Presentation

Initial Management

Hematemesis and melena are the most common presenting signs of acute upper gastrointestinal hemorrhage. Melena is sometimes seen in patients with hemorrhage in the lower gastrointestinal tract (e.g., distal small bowel and colon) and hematochezia in patients with upper gastrointestinal hemorrhage.

Appropriate hemodynamic assessment includes the careful measurement of pulse and blood pressure, including orthostatic changes, to estimate the intravascular volume status and guide resuscitative efforts. Patients who present with acute upper gastrointestinal bleeding and a substantial loss of intravascular volume have resting tachycardia (pulse, ≥100 beats per minute), hypotension (systolic blood pressure, <100 mm Hg), or postural changes (an increase in the pulse of ≥20 beats per minute or a drop in systolic blood pressure of ≥20 mm Hg on standing).

The first priority in treatment is correcting fluid losses and restoring hemodynamic stability.

The insertion of a nasogastric tube may be helpful in the initial assessment of the patient (specifically, triage), although the incremental information such a procedure provides remains controversial.

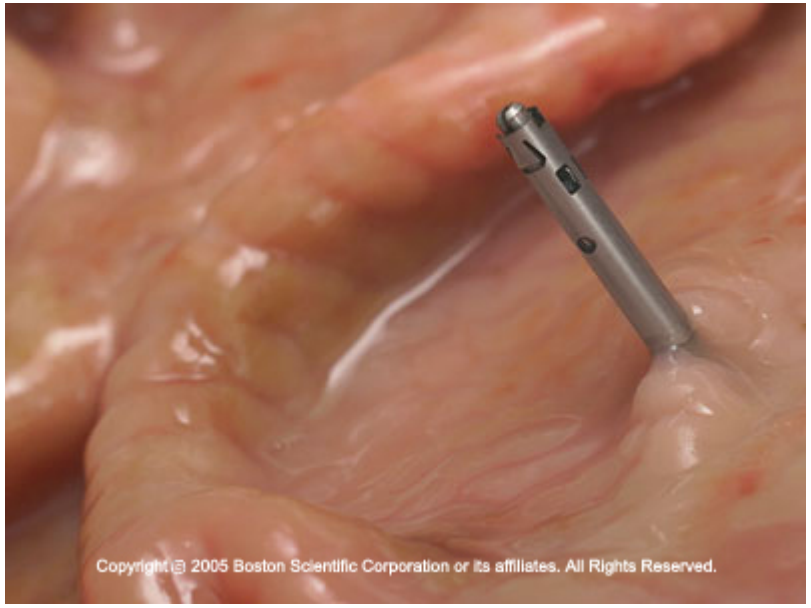
It has been suggested that the presence of red blood in the nasogastric aspirate is an adverse prognostic sign that may be useful in identifying patients who require urgent endoscopic evaluation. However, the absence of bloody or coffee-ground material does not definitively rule out ongoing or recurrent bleeding, since approximately 15% of patients without bloody or coffee-ground material in nasogastric aspirates are found to have high-risk lesions on endoscopy.

Treatment of Acute Upper GI Bleed

- Endoscopic Therapy
  - Thermal
    - Bipolar electrocautery, heater probe, laser, argon plasma coagulation
  - Injection
    - Vasoconstrictor (Epinephrine), sclerosant (alcohol or ethanolamine), saline
  - Band Ligation
    - Less complications than sclerotherapy – ulcer, stricture
  - Endoclips
  - In cases of negative endoscopy
    - Scintigraphy (Tagged RBC scan) and Angiography
      - Both require loss >0.5cc/min
      - Abdominal CT or MRI in suspected aortoenteric fistula

Argon Plasma Coagulation

Endoscopic clip



Treatment of Acute Upper GI Bleed

Stigmata of Hemorrhage	Incidence (%)	Rebleeding (%) Endoscopic treatment-untreated
Active Bleeding	18	20-55
Visible Vessel	17	15-43
Adherent Clot	15	5-22
Flat Spot	15	<1-10
Clean Ulcer Base	35	<1

Laine L, Peterson, J: Bleeding Peptic Ulcer. N Engl J Med 331:717, 1994

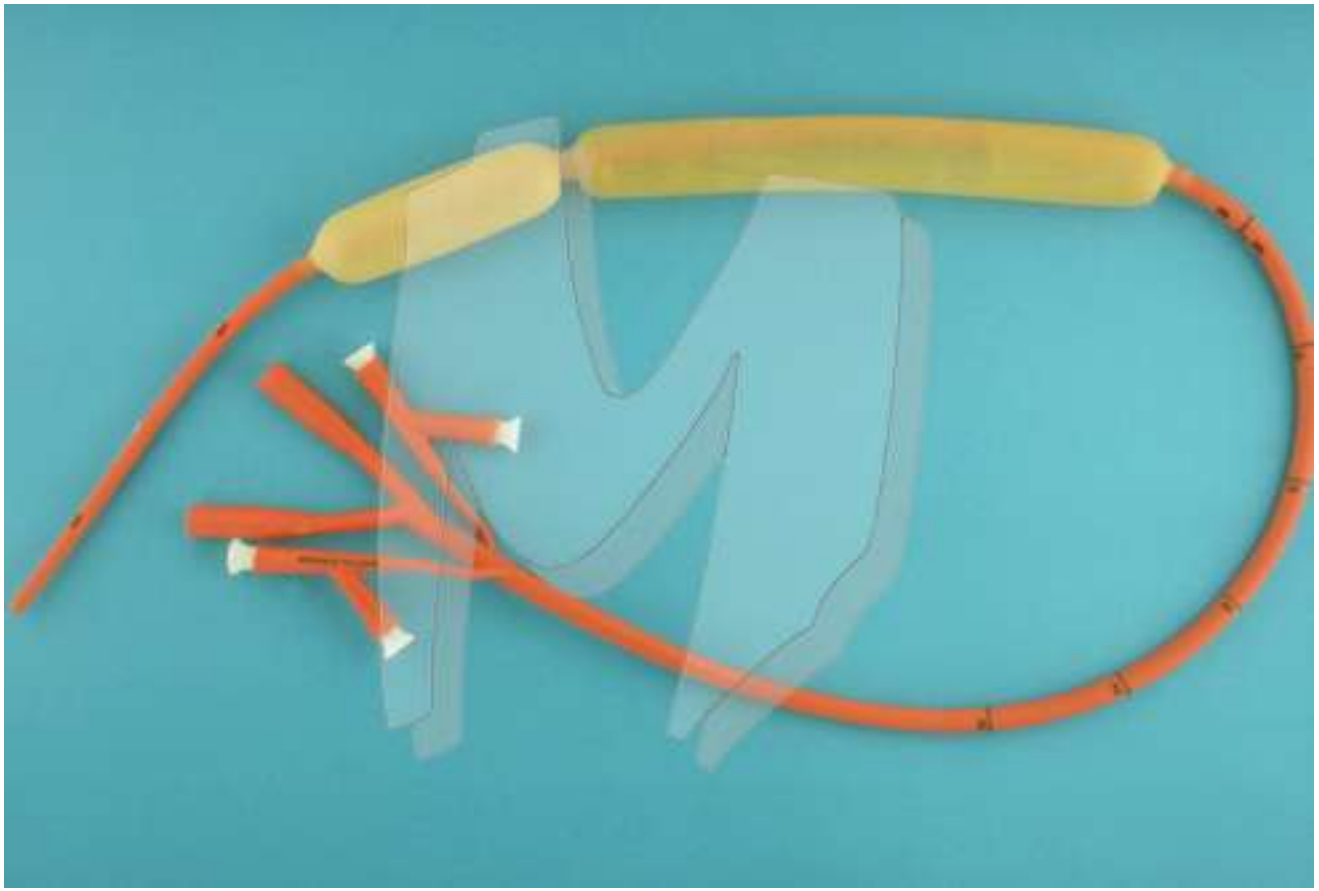
Treatment of Acute Upper GI Bleed

- Medication
  - Proton Pump Inhibitors
    - Treatment of active esophagitis, gastritis, ulcers
  - H2-receptor antagonists
    - Prophylaxis of stress gastritis and duodenal ulcers
  - Sucralfate
    - Stress gastritis prophylaxis when unable to use H2-receptor antagonist (lower rates of nosocomial pneumonia)
  - Antimicrobials
    - In cases of H. Pylori, Candida, and HSV infection
  - Octreotide, Propranolol
    - In cases of portal hypertension

Treatment of Acute Upper GI Bleed

- Mechanical Intervention
  - Balloon Tamponade of varices
    - Sengstaken-Blakemore tube – esophageal
    - Linton-Nachlas tube - gastric
      - 90% effective, rebleed rate high
      - Requires prophylactic intubation
- Therapeutic Angiography
  - Intra-arterial vasopressin, Gelfoam, tissue adhesives, embolization coils
  - Transjugular Intrahepatic Portosystemic Shunt (TIPS)
    - Stent between Hepatic and Portal veins
- Surgery – 5-10%

Sengstaken-Blakemore Tube



Patient Triage and Risk Stratification

The clinical Rockall score and the Blatchford score are useful prognostic tools in patients presenting with acute upper gastrointestinal hemorrhage, since the two tools have selected common features, including a determination of the patient's hemodynamic status and coexisting illnesses, and may reduce the need for urgent endoscopic evaluation in patients who are deemed to be at low risk.

Additional risk-stratification tools have been proposed.

The use of such validated tools as adjuncts to clinical evaluation and the judgment of the medical practitioner is encouraged in clinical practice.

The endoscopic appearance of a bleeding ulcer can be used to predict the likelihood of recurrent bleeding on the basis of the Forrest classification, which ranges from IA to III.

Highrisk lesions include those characterized by active spurting of blood (grade IA) or oozing blood (grade IB), a nonbleeding visible vessel described as a pigmented protuberance (grade IIA), and an adherent clot (which is defined as a lesion that is red, maroon, or black and amorphous in texture and that cannot be dislodged by suction or forceful water irrigation) (grade IIB) (Fig. 2A through 2D).

Low-risk lesions include flat, pigmented spots (grade IIC) and clean-base ulcers (grade III) (Fig. 2E and 2F).

The interobserver variation in diagnosing these endoscopic stigmata is low to moderate

At initial endoscopy, high-risk lesions are seen in approximately one third to one half of all patients, with rebleeding rates of 22 to 55% if the ulcer is left untreated endoscopically.

Table 1. Management of Acute Bleeding from a Peptic Ulcer, According to Clinical Status and Endoscopic Findings.*

Clinical status

At presentation

- Assess hemodynamic status (pulse and blood pressure, including orthostatic changes).
- Obtain complete blood count, levels of electrolytes (including blood urea nitrogen and creatinine), international normalized ratio, blood type, and cross-match.
- Initiate resuscitation (crystalloids and blood products, if indicated) and use of supplemental oxygen.
- Consider nasogastric-tube placement and aspiration; no role for occult-blood testing of aspirate.
- Consider initiating treatment with an intravenous proton-pump inhibitor (80-mg bolus dose plus continuous infusion at 8 mg per hour) while awaiting early endoscopy; no role for H_2 blocker.†
- Perform early endoscopy (within 24 hours after presentation).
- Consider giving a single 250-mg intravenous dose of erythromycin 30 to 60 minutes before endoscopy.
- Perform risk stratification; consider the use of a scoring tool (e.g., Blatchford score[16] or clinical Rockall score[17]) before endoscopy.

At early endoscopy

- Perform risk stratification; consider the use of a validated scoring tool (e.g., complete Rockall score[17]) after endoscopy.

Approach to Therapy

A multidisciplinary approach with timely involvement of a trained endoscopist and endoscopy assistant is widely recommended. Such involvement may entail after-hours availability, since early endoscopy (performed within 24 hours after presentation of the patient) is the cornerstone of treatment for patients with acute upper gastrointestinal hemorrhage and may improve certain outcomes (the number of units of blood transfused and the length of the hospital stay) for selected patients who are classified as being at high risk. Early endoscopy also allows for the safe and expedited discharge of patients who are classified as being at low risk and reduces the use of healthcare resources.

Goals of early endoscopy are to determine the cause of bleeding, ascertain prognosis, and administer endoscopic therapy, if indicated.

Treatment recommendations have focused on the first 72 hours after presentation and endoscopic evaluation and therapy, since this is the period when the risk of rebleeding is greatest (Table 1).

Patients at High Risk

High-risk patients should be admitted to the hospital and should receive endoscopic therapy. They should then be triaged to a monitored setting or intensive care unit for the first 24 hours of what is usually at least a 3-day hospital stay.

Patients who have bleeding ulcers with highrisk stigmata as determined on endoscopy (active bleeding or a nonbleeding visible vessel) should undergo endoscopic hemostasis, a procedure that has been shown to decrease rates of rebleeding, the need for urgent surgery, and mortality.

Contemporary endoscopic treatments include injection therapy (e.g., saline, vasoconstrictors, sclerosing agents, tissue adhesives, or a combination thereof), thermal therapy (with the use of contact methods, such as multipolar electrocoagulation and heater probe, or noncontact methods, such as argon plasma coagulation), and mechanical therapy (principally endoscopic clips).

Table 2. Proposed Selection Criteria for an Abbreviated Hospital Stay or Outpatient Treatment of Patients at Low Risk.*

Criteria
Age, <60 yr
Absence of hemodynamic instability, which is defined as resting tachycardia (pulse, ≥100 beats per minute), hypotension (systolic blood pressure, <100 mm Hg), or postural changes (increase in pulse of ≥20 beats per minute or a drop in systolic blood pressure of ≥20 mm Hg on standing), or hemodynamic stability within 3 hours after initial evaluation
Absence of a severe coexisting illness (e.g., heart failure, chronic obstructive pulmonary disease, hepatic cirrhosis, hematologic cancer, chronic renal failure, and cerebrovascular accident)
A hemoglobin level of more than 8 to 10 g per deciliter after adequate intravascular volume expansion and no need for blood transfusion
Normal coagulation studies
Onset of bleeding outside the hospital
Presence of a clean-base ulcer or no obvious endoscopic finding on early endoscopy (performed within 24 hours after presentation)
Adequate social support at home with the ability to return promptly to a hospital