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Overview of the management of patients with variceal bleeding

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INTRODUCTION

Variceal bleeding is a gastrointestinal emergency that is one of the major causes of death in patients with cirrhosis. The outcome for patients with variceal bleeding depends on achieving hemostasis and avoiding complications related to bleeding or underlying chronic liver disease.

A rise in portal pressure (portal hypertension) occurs when there is resistance to outflow from the portal vein. Varices develop in order to decompress the hypertensive portal vein and return blood to the systemic circulation. The formation and progression of varices are discussed separately. (See "Pathogenesis of variceal bleeding in patients with cirrhosis".)

Variceal bleeding is the most common cause of upper gastrointestinal bleeding in patients with cirrhosis and/or portal hypertension. However, patients with portal hypertension can also develop upper gastrointestinal bleeding from sources unrelated to portal hypertension (eg, peptic ulcer disease, Mallory-Weiss tear) [1]. (See "Causes of upper gastrointestinal bleeding in adults".)

This topic presents an overview of the management of patients with variceal bleeding. Strategies for bleeding prevention are discussed separately:

- Preventing the first episode of variceal bleeding (see "Primary prevention of bleeding from esophageal varices in patients with cirrhosis")

- Preventing recurrent variceal bleeding (see "Prevention of recurrent bleeding from esophageal varices in patients with cirrhosis")

The approach to and evaluation of adults with upper gastrointestinal bleeding from any source is presented separately. (See "Approach to acute upper gastrointestinal bleeding in adults".)

DEFINITIONS

The existing literature is confounded by the variable use of terminology across studies. As a result, several definitions were agreed upon during a consensus conference, and this simplifies the evaluation of published studies and renders newer studies more comparable [2]:

- **Time zero** – The time of admission to a medical care facility.
- **Clinically significant bleeding** – Defined by a transfusion requirement of two units of blood or more within 24 hours of time zero together with a systolic blood pressure below 100 mmHg, a postural systolic change >20 mmHg, and/or a pulse rate >100 beats per minute at time zero.
- **Acute variceal bleeding** – The episode of acute bleeding is comprised of the time interval from hospital admission (time zero) to 120 hours (day 5).
- **Treatment failure** – Failure of therapy is defined by any of the following criteria if they occur within 120 hours of time zero:
  - Fresh hematemesis or >100 mL of fresh blood in the nasogastric aspirate >2 hours after the start of a specific drug or endoscopic treatment
  - Development of hypovolemic shock
  - Drop in hemoglobin of ≥3 g/dL (30 g/L) within a 24-hour period
- **Variceal rebleeding** – Variceal rebleeding describes clinically significant bleeding that occurs ≥120 hours after the first hemorrhage, provided that hemostasis was initially achieved [3].

RISK FACTORS

Factors linked to the risk of variceal bleeding include size of varices, appearance of varices (eg, red wale marks or areas of thinning of the variceal wall), and the severity of liver disease.

Predictive factors for variceal bleeding are discussed in more detail separately. (See "Pathogenesis of variceal bleeding in patients with cirrhosis", section on 'Predictive factors'.)

PRINCIPLES OF ACUTE MANAGEMENT

Goals of therapy — Treatment goals during an episode of acute bleeding are to:

- Restore and maintain hemodynamic stability
- Restore and maintain adequate oxygenation
- Control bleeding
- Prevent complications

Management of acute variceal bleeding often requires multidisciplinary care (hepatology, critical care, interventional radiology).

Initial measures

Resuscitation and support — General resuscitative and supportive measures for patients with gastrointestinal bleeding who have a history of or are at risk for varices (eg, patients with jaundice or cirrhosis) include [4] (table 1):

- **Intravenous access and fluids** – After intravenous access (eg, two 16 gauge peripheral intravenous catheters or a central venous catheter) is established, fluid resuscitation should begin immediately. The approach to fluid resuscitation in patients who are hemodynamically unstable is discussed in detail elsewhere. (See "Evaluation of and initial approach to the adult patient with undifferentiated hypotension and shock", section on 'Hemodynamic support'.)
- **Supplemental oxygenation and airway protection** – Supplemental oxygen is initially provided by nasal cannula; however, endotracheal intubation to protect the patient's airway is usually performed for patients with hemodynamic instability, altered mental status, and/or ongoing hematemesis [5]. Extubation should be performed when possible after endoscopy because endotracheal intubation may be a risk factor for respiratory infection [6]. Extubation management is discussed separately. (See "Extubation management in the adult intensive care unit".)
- **Additional measures** – For patients with ongoing hematemesis, initial care also includes:

- Nasogastric tube – Nasogastric tube placement may help to remove particulate matter, fresh blood, and clots from the stomach prior to endoscopy. However, we perform nasogastric tube placement cautiously because of the risk of pulmonary infection [6]. (See "Inpatient placement and management of nasogastric and nasoenteric tubes in adults", section on 'Tube placement'.)

- Erythromycin – For patients without contraindications (eg, QTc prolongation), erythromycin may be given prior to upper endoscopy to improve endoscopic visualization [7,8]. The use of prokinetic agents including dosing and administration is discussed in more detail separately. (See "Approach to acute upper gastrointestinal bleeding in adults", section on 'Prokinetics'.)

Blood products — Patients with active bleeding and hypovolemia often require blood products. However, patients with cirrhosis have a form of rebalanced hemostasis, and conventional measures of clotting (eg, prothrombin time) are not particularly reflective of bleeding risk. These issues are presented separately. (See "Hemostatic abnormalities in patients with liver disease".)

Measures to improve hemostasis include:

- **Red blood cells** – We initiate blood transfusion if hemoglobin is <7 g/dL (70 g/L) for most patients, with a goal of maintaining the hemoglobin at a level between ≥7 g/dL (70 g/L) and <9 g/dL (90 g/L). However, for patients at increased risk of adverse events in the setting of anemia (ie, those with unstable coronary artery disease or ongoing active bleeding), our goal is to maintain the hemoglobin at a level of ≥9 g/dL (90 g/L). The patient's intravascular volume status is monitored to avoid volume overload because of the risk of rebound portal hypertension and induction of rebleeding [9-11]. (See "Intraoperative fluid management", section on 'Monitoring intravascular volume status'.)

  Patients who receive more than five to six units of red blood cells may be transfused with plasma and platelets to compensate for losses. Protocols for and complications of massive transfusion are discussed separately. (See "Massive blood transfusion".)

- **Other blood products** – For patients with variceal bleeding and coagulopathy, correcting the coagulopathy may be indicated for selected patients (eg, those in whom endoscopic therapy does not control bleeding). However, laboratory tests such as prothrombin time/international normalized ratio (PT/INR) and activated partial thromboplastin time, do not accurately reflect the hemostatic status of patients with decompensated cirrhosis. We do not routinely use fresh frozen plasma because it does not correct coagulopathy and may lead to volume overload and worsening of portal hypertension. In addition, we do not

routinely use platelet transfusion or fibrinogen supplementation because platelet count and fibrinogen levels have not been associated with the risk of rebleeding or of failing to achieve endoscopic hemostasis [6]. Hemostatic products for patients with active gastrointestinal bleeding in the setting of liver disease is discussed in more detail separately. (See "Hemostatic abnormalities in patients with liver disease", section on 'Bleeding' and "Cryoprecipitate and fibrinogen concentrate".)

- Vitamin K – Patients at risk of vitamin K deficiency (eg, those with cholestatic liver disease) can be treated with vitamin K. (See "Overview of vitamin K", section on 'Vitamin K deficiency'.)

Antibiotic prophylaxis — Patients with cirrhosis who present with upper gastrointestinal bleeding are given prophylactic antibiotics, preferably before endoscopy (although effectiveness has also been demonstrated when given after endoscopy) [12-15]. We typically use a broad spectrum antibiotic such as ceftriaxone (1 g intravenously daily for seven days). For patients who are discharged before seven days of intravenous antibiotic therapy, we transition to an oral antibiotic, such as ciprofloxacin (500 mg every 12 hours), to complete a total of seven days of antibiotic therapy. (See "Spontaneous bacterial peritonitis in adults: Treatment and prophylaxis", section on 'Choosing a regimen'.)

For patients with cirrhosis and gastrointestinal (GI) bleeding, prophylactic antibiotics reduce the risk of mortality, infections (eg, spontaneous bacterial peritonitis, urinary tract infections), and rebleeding. In a meta-analysis of 12 trials including over 1200 patients with cirrhosis and GI bleeding, antibiotic prophylaxis was compared with either placebo or no intervention, and the benefit of antibiotic use was demonstrated with regard to mortality (relative risk [RR] 0.79, 95% CI 0.63-0.98), bacterial infections (RR 0.35, 95% CI 0.26-0.47), and rebleeding (RR 0.53, 95% CI 0.38-0.74) [13].

Preliminary data suggested that selected patients (eg, those with Child-Pugh class A cirrhosis) may be at lower risk for bacterial infection and may not need antibiotic prophylaxis [16]. However, additional studies are needed to confirm these findings.

We typically use ceftriaxone because most infections are due to gram-negative bacteria and because ceftriaxone was superior to norfloxacin for preventing infection in patients with cirrhosis in a randomized trial [17,18]. Important factors in the choice of antibiotics include individual patient characteristics (eg, prior history of antibiotic exposure or infection) and local patterns of antibiotic resistance. Quinolone resistance, for example, has become an increasing problem in patients with cirrhosis in some centers [19]. The possibility of quinolone resistance is a particular concern in patients who have been receiving prophylactic norfloxacin for the

prevention of spontaneous bacterial peritonitis. In a trial including 111 patients with cirrhosis and GI bleeding, patients receiving oral norfloxacin had higher rates of developing infections (26 versus 11 percent) or spontaneous bacterial peritonitis (12 versus 2 percent) compared with patients given intravenous ceftriaxone [17]. (See "Spontaneous bacterial peritonitis in adults: Treatment and prophylaxis", section on 'Antibiotic resistance'.)

Pharmacologic therapy for bleeding — Pharmacologic therapy for patients with variceal bleeding (eg, octreotide, vasopressin) is discussed separately. (See "Methods to achieve hemostasis in patients with acute variceal hemorrhage".)

Medication adjustments — The decision of whether to continue outpatient medications in patients with cirrhosis and variceal bleeding depends on multiple factors, including the specific medication (eg, antihypertensive agent), the severity of bleeding, hemodynamic status of the patient, the functional status of other organs (ie, kidney, brain), and the presence and severity of underlying cardiovascular disease. Given the multiple variables, medical decision-making is individualized in consultation with the critical care team.

For example, we stop antihypertensive medications (eg, beta blockers) for patients with active variceal bleeding and hemodynamic instability, and we monitor them for signs that such medication may be restarted (ie, cessation of bleeding and hemodynamic stability).

For patients with variceal bleeding, we typically discontinue proton pump inhibitors that were initiated prior to endoscopy after the source of bleeding is confirmed endoscopically. Prolonged use of a proton pump inhibitor (ie, >28 days) has been associated with increased risk for hepatic encephalopathy and for further decompensation [20,21].

Electrolyte and vitamin repletion — We monitor electrolytes (eg, phosphorus, potassium, magnesium) for all patients with variceal bleeding, and we give thiamine to patients with alcohol use disorder. Thiamine is given prior to or along with glucose intravenous infusion to prevent precipitating Wernicke encephalopathy. The management of patients with alcohol use disorder is discussed in more detail separately. (See "Nutritional status in patients with sustained heavy alcohol use", section on 'Supplementation' and "Management of moderate and severe alcohol withdrawal syndromes" and "Wernicke encephalopathy".)

Patients with cirrhosis may have nutritional deficiencies and thus are prone to electrolyte abnormalities. In particular, hypophosphatemia and hypokalemia may develop during the hospitalization, especially after dextrose infusions, which raise serum insulin concentrations; insulin drives both phosphate and potassium into the cells [22]. (See "Hypophosphatemia in the patient with alcohol use disorder".)

Urgent endoscopy — Upper endoscopy should be performed after fluid resuscitation and within 12 hours of hospital admission [15,23], and endoscopic therapy to control active bleeding is discussed separately. (See "Methods to achieve hemostasis in patients with acute variceal hemorrhage".)

Subsequent measures

Nutritional support — For hemodynamically stable patients, we typically wait a minimum of 48 to 72 hours after an episode of acute variceal bleeding prior to resuming enteral nutrition either orally or via nasogastric tube. Enteral nutrition increases splanchnic blood flow, which may lead to an increase in portal pressure and the risk of rebleeding [24,25]. The goals and delivery of enteral nutrition for critically ill patients is discussed separately. (See "Nutrition support in critically ill patients: An overview" and "Nutrition support in critically ill patients: Enteral nutrition".)

Additional studies — We typically obtain an abdominal ultrasound with Doppler imaging to exclude portal vein thrombosis after the patient has been resuscitated and endoscopy has been performed. (See 'Initial measures' above and 'Urgent endoscopy' above and "Acute portal vein thrombosis in adults: Clinical manifestations, diagnosis, and management", section on 'Abdominal ultrasound with Doppler'.)

If initial pharmacologic and endoscopic therapy fails to stop variceal bleeding, a transjugular intrahepatic portosystemic shunt (TIPS) is a subsequent therapeutic option. The indications and contraindications to TIPS placement, in addition to the role of TIPS in treating patients with variceal bleeding, is discussed separately. (See "Overview of transjugular intrahepatic portosystemic shunts (TIPS)".)

Complications — Control of active variceal bleeding is usually achieved; however, some patients with cirrhosis develop complications during the hospital course. Major complications that can occur and may increase mortality risk include pneumonia, sepsis, acute-on-chronic liver failure, hepatic encephalopathy, and renal failure [18,26]. It is not unusual for multiple complications to develop simultaneously.

Hepatic encephalopathy — Hepatic encephalopathy is managed with lactulose [27], with control of active bleeding, and with correction of other precipitating causes (eg, electrolyte abnormalities, infection). Management of hepatic encephalopathy is discussed in more detail separately. (See "Hepatic encephalopathy in adults: Treatment" and "Hepatic encephalopathy in adults: Clinical manifestations and diagnosis", section on 'Evaluation for precipitating causes'.)

For example, hypokalemia can promote the development of hepatic encephalopathy via increased renal ammonia production. (See "Hepatic encephalopathy: Pathogenesis" and "Hypokalemia-induced kidney dysfunction".)

Renal failure — The risk of renal failure (due either to acute tubular necrosis or to hepatorenal syndrome) can be minimized by adequate volume replacement and by avoiding nephrotoxic drugs (eg, aminoglycosides) and mismatched transfusions. Hepatorenal syndrome that occurs in the setting of gastrointestinal bleeding is discussed separately. (See "Hepatorenal syndrome".)

Other complications — Complications related to the specific therapy used to control variceal bleeding (eg, pharmacologic therapy, endoscopic therapy, TIPS) are discussed separately. (See "Methods to achieve hemostasis in patients with acute variceal hemorrhage" and "Transjugular intrahepatic portosystemic shunts: Postprocedure care and complications".)

PREVENTING RECURRENT BLEEDING

Patients who recover from acute variceal bleeding are at risk for rebleeding, and interventions to prevent recurrent bleeding are discussed separately. (See "Prevention of recurrent bleeding from esophageal varices in patients with cirrhosis".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Gastrointestinal bleeding in adults".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5^{th} to 6^{th} grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10^{th} to 12^{th} grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: GI bleed (The Basics)")
- Beyond the Basics topics (see "Patient education: Esophageal varices (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- **Background** – Variceal bleeding is a gastrointestinal emergency that is one of the major causes of death in patients with cirrhosis. The outcome of an episode of variceal bleeding depends on achieving hemostasis and avoiding complications related to bleeding or underlying chronic liver disease. (See 'Introduction' above.)

  Factors linked to the risk of variceal bleeding include size of varices, appearance of varices (eg, red wale marks or areas of thinning of the variceal wall), and the severity of liver disease. (See 'Risk factors' above and "Pathogenesis of variceal bleeding in patients with cirrhosis", section on 'Predictive factors'.)

- **Goals of therapy** – For patients with acute variceal bleeding, goals of treatment are (see 'Goals of therapy' above):
  - Restore and maintain hemodynamic stability
  - Restore and maintain adequate oxygenation
  - Control bleeding
  - Prevent complications
- **Initial measures**
  - General measures – For patients with cirrhosis and gastrointestinal bleeding, general initial measures include intravenous access, intravenous fluids, and supplemental oxygen, and airway protection. A table outlining the clinical features and emergency management of acute severe upper gastrointestinal bleeding in adults is provided (table 1). (See 'Resuscitation and support' above and "Approach to acute upper gastrointestinal bleeding in adults".)

- Antibiotic prophylaxis – For patients with cirrhosis who present with gastrointestinal bleeding, we recommend prophylactic antibiotic therapy rather than no antibiotics because antibiotic prophylaxis reduces the risk of mortality, infection, and rebleeding (**Grade 1B**). We typically use a course of ceftriaxone (1 g intravenously daily for seven days). (See 'Antibiotic prophylaxis' above.)

- Pharmacologic therapy – The use of pharmacologic therapy to control variceal bleeding is discussed separately. (See "Methods to achieve hemostasis in patients with acute variceal hemorrhage", section on 'Pharmacologic therapy'.)

- **Endoscopy** – For patients with cirrhosis and gastrointestinal bleeding, upper endoscopy should be performed after fluid resuscitation and within 12 hours of hospital admission. Endoscopic therapy to control active bleeding is discussed separately. (See 'Urgent endoscopy' above and "Methods to achieve hemostasis in patients with acute variceal hemorrhage".)

- **Adverse events** – For most patients with variceal bleeding, hemostasis is achieved; however, patients with cirrhosis are at risk for complications during the hospital course. Major complications include pneumonia, sepsis, acute-on-chronic liver failure, hepatic encephalopathy, and renal failure. (See 'Complications' above and "Acute liver failure in adults: Management and prognosis".)

- **Preventing recurrent bleeding** – Patients who recover from acute variceal bleeding are at risk for rebleeding, and interventions to prevent recurrent bleeding are discussed separately. (See "Prevention of recurrent bleeding from esophageal varices in patients with cirrhosis".)

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GRAPHICS

Upper GI bleeding in adults: Rapid overview of emergency management

Major causes*
Peptic ulcer, esophagogastric varices, arteriovenous malformation, tumor, esophageal (Mallory-Weiss) tear
Clinical features
History
Use of: NSAIDs, aspirin, anticoagulants, antiplatelet agents
Alcohol abuse, previous GI bleed, liver disease, coagulopathy
Symptoms and signs: Abdominal pain, hematemesis or "coffee ground" emesis, passing melena/tarry stool (stool may be frankly bloody or maroon with massive or brisk upper GI bleeding)
Examination
Tachycardia; orthostatic blood pressure changes suggest moderate to severe blood loss; hypotension suggests life-threatening blood loss (hypotension may be late finding in healthy younger adult)
Rectal examination is performed to assess stool color (melena versus hematochezia versus brown)
Significant abdominal tenderness accompanied by signs of peritoneal irritation (eg, involuntary guarding) suggests perforation
Diagnostic testing
Obtain type and crossmatch for hemodynamic instability, severe bleeding, or high-risk patient; obtain type and screen for hemodynamically stable patient without signs of severe bleeding
Obtain hemoglobin concentration (note that measurement may be inaccurate with acute severe hemorrhage), platelet count, coagulation studies (prothrombin time with INR), liver enzymes (AST, ALT), albumin, BUN, and creatinine
Nasogastric lavage may be helpful if the source of bleeding is unclear (upper or lower GI tract) or to clean the stomach prior to endoscopy
Treatment
Closely monitor airway, clinical status, vital signs, cardiac rhythm, urine output, nasogastric output (if nasogastric tube in place)
Do **NOT** give patient anything by mouth
Establish two large bore IV lines (16 gauge or larger)

Provide supplemental oxygen (goal oxygen saturation ≥94% for patients without COPD)
Treat hypotension initially with rapid, bolus infusions of isotonic crystalloid (eg, 500 to 1000 mL per bolus; use smaller boluses and lower total volumes for patients with compromised cardiac function)
Transfusion:
For severe, ongoing bleeding, immediately transfuse blood products in 1:1:1 ration of RBCs, plasma, and platelets, as for trauma patients
For hemodynamic instability despite crystalloid resuscitation, transfuse 1 to 2 units RBCs
For hemoglobin <8 g/dL (80 g/L) in high-risk patients (eg, older adult, coronary artery disease), transfuse 1 unit RBCs and reassess the patient's clinical condition
For hemoglobin <7 g/dL (70 g/L) in low-risk patients, transfuse 1 units RBCs and reassess the patient's clinical condition
Avoid over-transfusion with possible variceal bleeding
Give plasma for coagulopathy or after transfusing four units of RBCs; give platelets for thrombocytopenia (platelets <50,000) or platelet dysfunction (eg, chronic aspirin therapy) or after transfusing four units of RBCs
Obtain immediate consultation with gastroenterologist; obtain surgical and interventional radiology consultation for any large-scale bleeding¶
Pharmacotherapy for all patients with suspected or known severe bleeding:
Give a proton pump inhibitor:
Evidence of active bleeding (eg, hematemesis, hemodynamic instability), give esomeprazole or pantoprazole, 80 mg IV
No evidence of active bleeding, give esomeprazole or pantoprazole, 40 mg IV
Endoscopy delayed beyond 12 hours, give second dose of esomeprazole or pantoprazole, 40 mg IV
Pharmacotherapy for known or suspected esophagogastric variceal bleeding and/or cirrhosis:
Give somatostatin or an analogue (eg, octreotide 50 mcg IV bolus followed by 50 mcg/hour continuous IV infusion)
Give an IV antibiotic (eg, ceftriaxone or fluoroquinolone)
Balloon tamponade may be performed as a temporizing measure for patients with uncontrollable hemorrhage likely due to varices using any of several devices (eg, Sengstaken-Blakemore tube, Minnesota tube); tracheal intubation is necessary if such a device is to be placed; ensure proper device placement prior to inflation to avoid esophageal rupture

COPD: chronic obstructive pulmonary disease; GI: gastrointestinal; INR: international normalized ratio; AST: aspartate aminotransferase; ALT: alanine aminotransferase; BUN: blood urea nitrogen; IV: intravenous; RBC: red blood cells.

* An important but uncommon cause of gastrointestinal hemorrhage is vascular-enteric fistula, typically aortoduodenal fistula related to erosion of a prosthetic aortic graft.

¶ Minimally invasive techniques to control bleeding include sclerotherapy, embolization, and other vascular occlusion techniques. For patients with massive hemorrhage, resuscitative endovascular balloon occlusion of the aorta (REBOA) can be used to limit blood loss and support perfusion of vital organs until the sites of bleeding can be directly controlled.

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