# Mallory-Weiss syndrome

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## INTRODUCTION

Mallory-Weiss syndrome is characterized by longitudinal mucosal lacerations (intramural dissection) in the distal esophagus and proximal stomach, which are usually associated with forceful or prolonged vomiting or retching. The lacerations often lead to bleeding from submucosal arteries. This topic will review the epidemiology, pathogenesis, clinical manifestations, diagnosis, and management of Mallory-Weiss syndrome. The management of other causes of upper gastrointestinal bleeding and esophageal perforation are discussed in detail, separately. (See "Causes of upper gastrointestinal bleeding in adults" and "Boerhaave syndrome: Effort rupture of the esophagus".)

## EPIDEMIOLOGY

**Incidence** — The reported incidence of Mallory-Weiss syndrome among patients presenting with upper gastrointestinal bleeding ranges from 8 to 15 percent [1-3]. It is likely that Mallory-Weiss syndrome occurs in a less severe form more frequently than is recognized. However, the incidence of Mallory-Weiss tears in patients without overt gastrointestinal bleeding is not well established.

**Risk factors** — Observational studies have identified specific clinical and demographic features as potential risk factors for Mallory-Weiss syndrome [4-9]. Approximately, 20 percent of patients have no underlying risk factors [10].

- **Alcohol use** – Alcohol use is the most frequently identified risk factor in patients with Mallory-Weiss syndrome [10]. A history of heavy alcohol use leading to vomiting has been noted in 40 to 80 percent of patients with Mallory-Weiss syndrome in case series [3-6,11]. The bleeding is usually more severe when Mallory-Weiss tears are associated with portal hypertension and esophageal varices [7,8].

- **Hiatal hernia** – It has been proposed that retching increases the potential for mucosal laceration by creating a higher pressure gradient in the hiatus hernia as compared with the rest of the stomach. Although a hiatus hernia has been reported in 40 to 80 percent of patients with Mallory-Weiss tears in some case series, a large case-control study found no significant difference in the prevalence of hiatus hernia among patients with Mallory-Weiss syndrome and controls [4,9]. (See "Hiatus hernia".)

- **Age** – Increasing age has been advocated as a predisposing factor of Mallory-Weiss tears [12]. However, most tears occur in patients under the age of 40 years, suggesting that age does not play a major role. Tears have also occurred in children as young as three weeks of age [13].

Precipitating factors include vomiting, straining or lifting, coughing, seizures, blunt abdominal injury, nasogastric tube placement, and gastroscopy [1-3,12,14-18].

## PATHOGENESIS

The pathogenesis of Mallory-Weiss syndrome is not completely understood. It has been proposed that mucosal lacerations develop secondary to a sudden increase in intra-abdominal pressure. Bleeding occurs when the tear involves the underlying esophageal venous or arterial plexus.

## CLINICAL MANIFESTATIONS

Patients usually present with hematemesis (either red blood or coffee-ground emesis). Hematemesis may be accompanied by epigastric pain or pain in the back. Patients often have a history of nonbloody emesis, retching, or coughing prior to hematemesis [11]. Patients with significant bleeding may have signs of hypovolemia and hemodynamic instability (eg, resting tachycardia, hypotension).

## EVALUATION

**Diagnosis** — Mallory-Weiss syndrome should be suspected in patients with hematemesis (either red blood or coffee-ground emesis) and a history of vomiting or retching. The diagnosis is established on endoscopy with visualization of a single, longitudinal mucosal tear at the esophagogastric junction. (See 'Endoscopic therapy for active bleeding' below.)

**Upper endoscopy** — An upper endoscopy establishes the diagnosis, rules out other etiologies, and allows for therapeutic intervention in patients with active bleeding. Mallory-Weiss tears are located in the esophagogastric junction, often within a hiatal hernia; they usually extend downward into the cardia and sometimes upward into the esophagus [5]. Mallory-Weiss tears are usually single and longitudinal. However, multiple mucosal tears have been found in up to 27 percent of cases [5,6]. On endoscopy, the tears typically appear as a red longitudinal break in the mucosa ( picture 1), sometimes extending through the muscularis mucosa ( picture 2), and occasionally covered by a clot ( picture 3). Active bleeding may also be noted ( picture 4). In many instances, the lesions are recognized only after retroflexion of the tip of the gastroscope to view the cardia from below. Most tears heal within 24 to 48 hours in patients without portal hypertension and may be missed if endoscopy is delayed. (See "Approach to acute upper gastrointestinal bleeding in adults", section on 'Diagnostic studies'.)

**Differential diagnosis** — The differential diagnosis of Mallory-Weiss syndrome includes other diseases of the esophagus that causes esophageal ulcers (eg, reflux, medication, infections). Mallory-Weiss syndrome can be distinguished from these by history and upper endoscopy.

- **Reflux esophagitis** – Patients often have a history of heartburn, regurgitation, and dysphagia/odynophagia. The ulcerations seen in reflux esophagitis are usually in the distal esophagus, but unlike Mallory-Weiss tears, ulcers may be irregularly shaped and multiple. (See "Clinical manifestations and diagnosis of gastroesophageal reflux in adults", section on 'Upper gastrointestinal endoscopy'.)

- **Medication-induced esophagitis** – Medication-induced esophagitis is suspected by the history (eg, tetracycline or alendronate). Medication-induced ulcerations are usually singular and deep, occurring at points of stasis (especially near the carina), with sparing of the distal esophagus. (See "Pill esophagitis", section on 'Diagnostic evaluation'.)

- **Infectious esophagitis** – In contrast to a Mallory-Weiss tear, which is usually single and at the gastroesophageal junction, ulcers due to infectious esophagitis are usually multiple, punctate, and may be circumferential. In patients with herpes simplex virus esophagitis, ulcers in the distal esophagus are well circumscribed and have a "volcano-like" appearance. In patients with cytomegalovirus infection, ulcers tend to be linear or longitudinal and deep. The diagnosis of infectious esophagitis is established by biopsies

and viral culture. (See "Herpes simplex virus infection of the esophagus", section on 'Diagnosis' and "Epidemiology, clinical manifestations, and treatment of cytomegalovirus infection in immunocompetent adults", section on 'Gastrointestinal manifestations'.)

Other causes of upper gastrointestinal bleeding are discussed in detail separately. (See "Causes of upper gastrointestinal bleeding in adults", section on 'Specific causes'.)

## INITIAL MANAGEMENT

**Assessment of hemodynamic stability and fluid resuscitation** — The initial evaluation of a patient with upper gastrointestinal bleeding starts with assessing hemodynamic stability and determining the need for fluid resuscitation and/or blood transfusion. This part of the evaluation is discussed in detail elsewhere. (See "Approach to acute upper gastrointestinal bleeding in adults".)

**General measures** — Pharmacologic therapy for Mallory-Weiss syndrome includes acid suppression with proton pump inhibitors (PPIs) and antiemetics.

**Acid suppression** — Patients with clinically significant upper gastrointestinal bleeding (ie, signs of active upper gastrointestinal bleeding including hematemesis, melena, or hematochezia, with or without hemodynamic instability or blood transfusion requirement) should be started on a high-dose twice-daily intravenous (IV) PPI prior to endoscopy as part of their initial management. (See "Approach to acute upper gastrointestinal bleeding in adults", section on 'Acid suppression'.)

The twice-daily IV PPI may be switched to an oral PPI (eg, omeprazole 20 mg twice daily) following endoscopy. We continue oral PPI therapy for at least two weeks to stabilize the clot and accelerate mucosal healing. PPIs may promote hemostasis by neutralizing gastric acid and stabilizing blood clots. (See "Overview of the treatment of bleeding peptic ulcers", section on 'Efficacy of proton pump inhibitors'.)

**Antiemetics** — Antiemetics (eg, ondansetron, metoclopramide, prochlorperazine) should be used to treat persistent symptoms of nausea or vomiting. (See "Approach to the adult with nausea and vomiting", section on 'Treatment'.)

**Endoscopic therapy for active bleeding** — An upper endoscopy establishes the diagnosis, rules out other etiologies, and allows for therapeutic intervention [19]. Endoscopic therapy is indicated for treatment of stigmata of recent hemorrhage as they are associated with an increased risk of recurrent bleeding. Stigmata include active bleeding (spurting or oozing

hemorrhage), a visible vessel, and an adherent clot. Evidence to support the use of endoscopic therapy in patients with Mallory-Weiss syndrome are extrapolated from patients with bleeding peptic ulcers. Mallory-Weiss tears without stigmata of recent hemorrhage at the time of endoscopy can be managed with acid suppression alone ( algorithm 1). (See "Overview of the treatment of bleeding peptic ulcers".)

- **Choice of endoscopic therapy** – Patients with active bleeding may be treated with 1:10,000 epinephrine local injection to reduce or stop bleeding through vasoconstriction along with thermal coagulation, endoscopic clips, or in some cases, endoscopic band ligation or argon plasma coagulation [16,20-28]. The choice of treatment often depends upon the preference of the endoscopist. Few studies have directly compared these modalities [23,29,30]. In a randomized trial in which 41 patients with actively bleeding Mallory-Weiss tears were assigned to endoscopic band ligation or endoscopic clip placement, there were no differences in the rates of primary hemostasis or rebleeding between the two groups [30]. Hemostatic sprays can be used to control active gastrointestinal bleeding in a variety of contexts, particularly when traditional endoscopic techniques fail to control massive gastrointestinal bleeding.

  Epinephrine should not be used as monotherapy due to an increased risk of recurrent bleeding. In a retrospective study that included 168 patients with Mallory-Weiss tear, endoscopic clip-based therapy and band ligation were associated with higher success rates in preventing rebleeding as compared with injection therapy alone (96, 89, and 71 percent, respectively) [29].

- **Important technical considerations** – Thermal coagulation should be performed with less tamponade force and lower total energy (eg, bipolar probe at 15 watts, mild tamponade pressure, and one second pulses) in patients with Mallory-Weiss syndrome as the esophagus lacks a serosa and may be very thin at the tear site and the underlying artery is small. Repeated coagulation should be avoided because of the risk of transmural injury and perforation [31]. The techniques for endoscopic clips, endoscopic band ligation, and injection with epinephrine are similar to patients with peptic ulcer bleeding. Detailed discussions of the endoscopic techniques used for hemostasis are presented elsewhere. (See "Contact thermal devices for the treatment of bleeding peptic ulcers" and "Endoscopic clip therapy in the gastrointestinal tract: Bleeding lesions and beyond" and "Overview of the treatment of bleeding peptic ulcers", section on 'Injection therapy'.)

**Persistent bleeding despite endoscopic therapy**

- **Transarterial embolization** – Transarterial embolization (TAE) for angiographic control of bleeding is performed if endoscopic therapy has failed. In a meta-analysis of 13 observational studies that included 1077 patients with nonvariceal upper gastrointestinal bleeding, patients who underwent TAE had higher rates of rebleeding as compared with those who underwent surgery (OR 2.44, 1.77–3.36), but had fewer major complications (OR 0.45, 0.30–0.67). There was no significant difference in mortality between the two groups ( algorithm 1). (See "Angiographic control of nonvariceal gastrointestinal bleeding in adults".)

- **Surgery** – Surgical management involves oversewing of the bleeding vessels and is usually reserved for patients who fail angiographic therapy [1,3,32].

## POST-ENDOSCOPIC MANAGEMENT

**Rebleeding risk** — Mallory-Weiss syndrome tears heal rapidly in the absence of portal hypertensive gastropathy. Approximately 7 percent of patients have recurrent bleeding, which usually occurs within 24 hours of the initial episode and usually in patients with an underlying coagulopathy or on antithrombotics [33]. Rebleeding can usually be managed endoscopically. In the absence of rebleeding, patients do not require repeat (second look) endoscopic evaluation to document healing.

### Indications for hospitalization

We typically hospitalize and observe patients at high risk for rebleeding and its complications for 48 hours. This includes patients with any one of the following:

- Risk factors for recurrent bleeding (portal hypertension coagulopathy).
- Endoscopic stigmata of recent bleeding (eg, active bleeding at the time of endoscopy, nonbleeding visible vessel, or adherent clot).
- Severe upper gastrointestinal bleeding (hemodynamic instability, hematochezia, blood transfusion requirement).
- Increased risk for complications should bleeding recur (eg, significant coronary artery or cerebrovascular disease, age over 65 years).

Hospitalized patients can start a clear liquid diet soon after endoscopic therapy and advance as tolerated after 24 hours.

All other low-risk patients can be discharged from the hospital on oral proton pump inhibitor therapy once the effects of procedural sedation have worn off, provided that the patient is

reliable and can promptly receive medical care should bleeding recur.

**Resumption of anticoagulants and antiplatelet agents** — Data are limited with regard to the appropriate timing for resuming anticoagulation or antiplatelet agents following successful endoscopic hemostasis. The timing will depend on the patient's risk of suffering a thromboembolic event while off of the medication(s). When to resume these agents after hemostasis has been achieved is discussed elsewhere. (See "Management of anticoagulants in patients undergoing endoscopic procedures", section on 'Resuming anticoagulants after hemostasis' and "Management of antiplatelet agents in patients undergoing endoscopic procedures".)

## PROGNOSIS

Active bleeding at the time of initial endoscopy and a low initial hematocrit have been associated with a higher rate of rebleeding, need for angiography or surgery, or death [10]. The mortality in patients with Mallory-Weiss syndrome is comparable to patients with peptic ulcer disease. A retrospective study comparing the 30-day mortality in 281 patients with endoscopically confirmed Mallory-Weiss syndrome and 1530 patients with peptic ulcer bleeding found that the mortality rate was 5.3 percent for patients with bleeding Mallory-Weiss syndrome and 4.6 percent for patients with peptic ulcer bleeding [34]. Mortality was significantly higher in patients over 65 years of age and those with significant overall comorbidities.

## 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".)

## SUMMARY AND RECOMMENDATIONS

- **Etiology and pathogenesis** – Mallory-Weiss syndrome is characterized by longitudinal mucosal lacerations in the distal esophagus and proximal stomach secondary to a sudden increase in intra-abdominal pressure. Bleeding occurs when the tear involves the underlying esophageal venous or arterial plexus. Risk factors include alcohol use and hiatus hernia. Mallory-Weiss syndrome may be precipitated by vomiting, straining or

lifting, coughing, seizures, blunt abdominal injury, nasogastric tube placement, and gastroscopy. (See 'Epidemiology' above and 'Pathogenesis' above.)

- **Clinical features** – Patients usually present with hematemesis (either red blood or coffee-ground emesis). Hematemesis may be accompanied by epigastric pain or pain in the back. Patients often have a history of nonbloody emesis, retching, or coughing prior to hematemesis. (See 'Clinical manifestations' above.)

- **Diagnosis** – Mallory-Weiss syndrome should be suspected in patients with upper gastrointestinal bleeding and a history of vomiting or retching. The diagnosis is established on endoscopy with visualization of a single, longitudinal mucosal tear at the esophagogastric junction. An upper endoscopy establishes the diagnosis, rules out other etiologies, and allows for therapeutic intervention. (See 'Evaluation' above and 'Endoscopic therapy for active bleeding' above.)

- **Management**

  - **General measures**

    - Patients require urgent assessment of hemodynamic stability and may require supportive care including fluid resuscitation and/or blood transfusions, and antiemetics for management of persistent nausea or vomiting.

    - In general, a high-dose twice-daily intravenous proton pump inhibitor (PPI) is administered to all patients with suspected clinically significant upper gastrointestinal bleeding prior to endoscopy as part of their initial management. In patients with Mallory-Weiss syndrome, we continue an oral PPI (eg, omeprazole 20 mg twice daily) in all patients for at least two weeks. (See "Approach to acute upper gastrointestinal bleeding in adults", section on 'Acid suppression'.)

  - **Endoscopic evaluation and therapy** – In patients with Mallory-Weiss tears with stigmata of recent hemorrhage on upper endoscopy, we recommend endoscopic therapy and acid suppressive therapy rather than acid suppressive therapy alone (**Grade 1B**). Stigmata of recent hemorrhage (active bleeding, visible vessel, or adherent clot) increase the risk of recurrent bleeding. Patients may be treated with thermal coagulation, endoscopic clips, or endoscopic band ligation (with or without epinephrine injection) ( algorithm 1). (See 'Endoscopic therapy for active bleeding' above.)

Mallory-Weiss tears without stigmata of recent hemorrhage can be managed with acid suppression alone. (See 'Acid suppression' above.)

- **Hospitalization in patients at high risk for rebleeding and complications** – We typically hospitalize and observe patients at high risk for rebleeding and its complications for 48 hours. This includes patients with any one of the following:

  - Risk factors for recurrent bleeding (portal hypertension, coagulopathy).
  - Endoscopic stigmata of recent bleeding (eg, active bleeding at the time of endoscopy, nonbleeding visible vessel, or adherent clot).
  - Severe upper gastrointestinal bleeding (hemodynamic instability, hematochezia, blood transfusion requirement).
  - Increased risk for complications should bleeding recur (eg, significant coronary artery or cerebrovascular disease).

  Patients without risk factors for rebleeding, endoscopic stigmata of recent bleeding, or clinical features indicating severe bleeding can be discharged following endoscopy. (See 'Post-endoscopic management' above.)

- **Patients with persistent bleeding** – We suggest that patients who fail endoscopic therapy undergo transarterial angiographic embolization rather than surgery (**Grade 2C**). (See 'Persistent bleeding despite endoscopic therapy' above.)

- **Prognosis** – Mallory-Weiss syndrome tears heal rapidly. Patients do not require repeat endoscopic evaluation to document healing. Approximately 7 percent of patients with Mallory-Weiss syndrome have recurrent bleeding. Rebleeding can usually be managed endoscopically. The mortality rate is approximately 5 percent and depends upon patient age and the presence of coexisting medical conditions. (See 'Rebleeding risk' above and 'Prognosis' above.)

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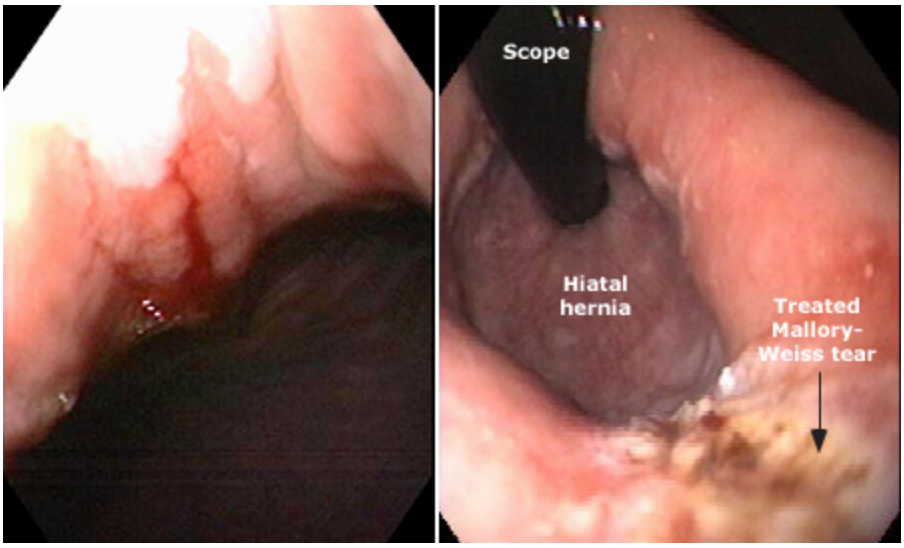
## GRAPHICS

### Mallory-Weiss tear




Endoscopic diagnosis and treatment of Mallory-Weiss tears. Left panel: A large esophageal laceration extending to the lower esophageal body. Right panel: A large esophageal laceration below the gastroesophageal junction seen by retroflexion of the tip of the gastroscope which also visualized a hiatal hernia; previous bleeding was controlled by multipolar electrocoagulation.

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*Courtesy of Moises Guelrud, MD.*

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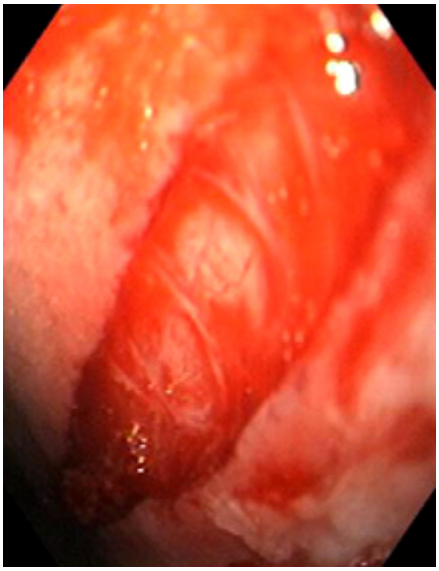
Graphic 57049 Version 1.0

## Mallory-Weiss tear



Endoscopy shows a mucosal tear extending into the muscularis mucosa at the distal esophagus.

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*Courtesy of Moises Guelrud, MD.*

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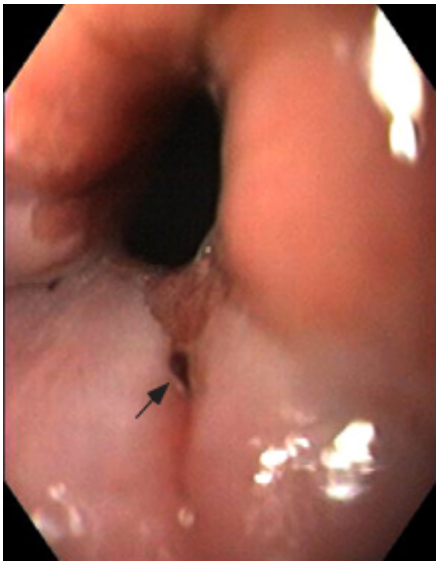
Graphic 76980 Version 1.0

## Mallory-Weiss tear



Endoscopy shows a Mallory-Weiss tear with an adherent clot at the apex of the tear.

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*Courtesy of Moises Guelrud, MD.*

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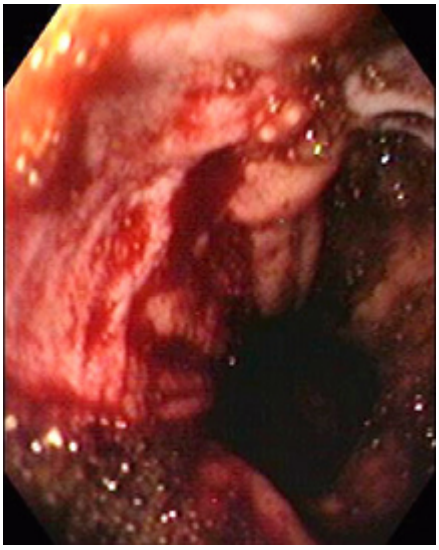
Graphic 68239 Version 2.0

## Mallory-Weiss tear



Endoscopy shows an actively bleeding Mallory-Weiss tear at the gastroesophageal junction.

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*Courtesy of Moises Guelrud, MD.*

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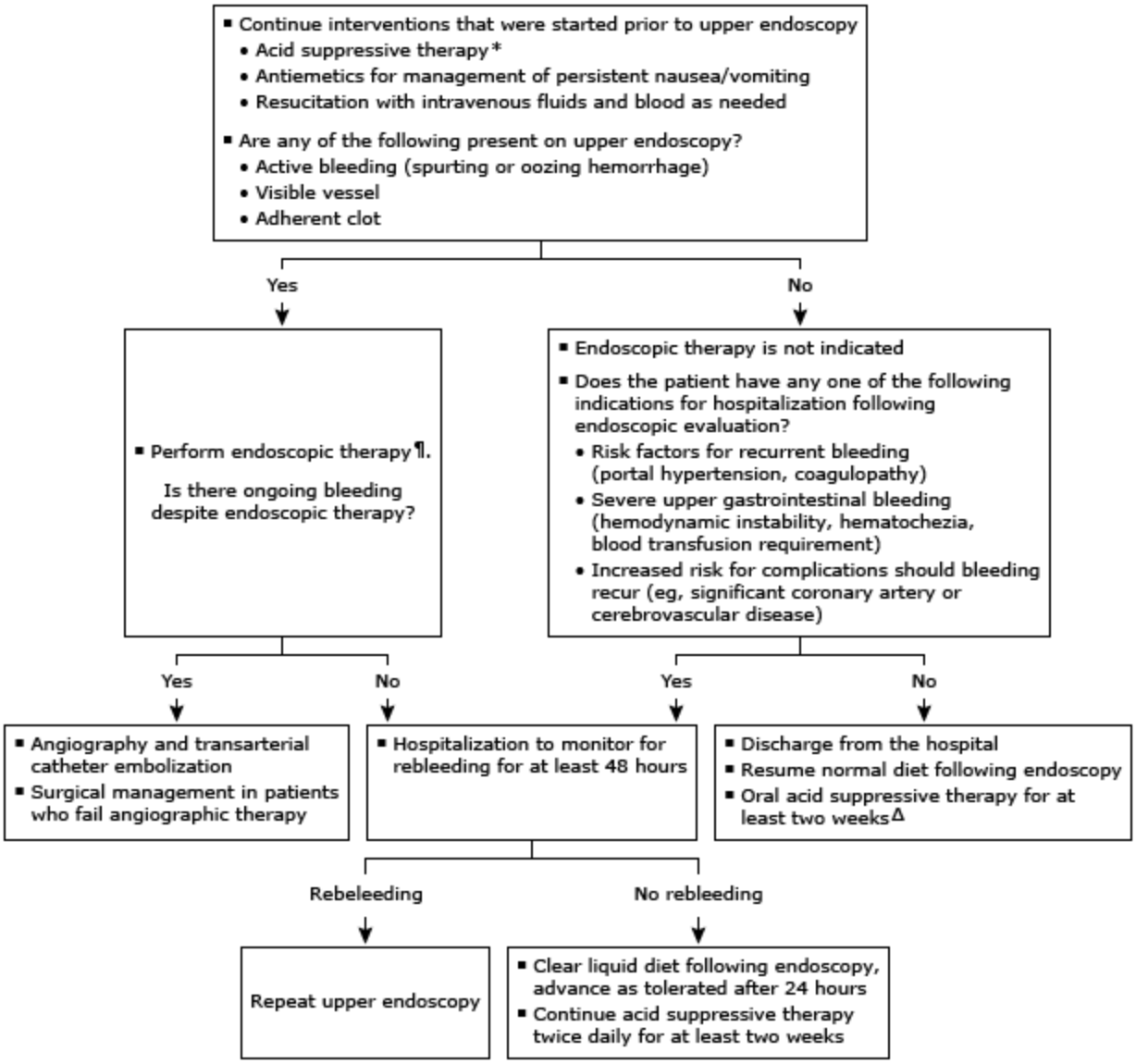
Graphic 71690 Version 1.0

## Approach to the management of an adult patient with a Mallory-Weiss tear on upper endoscopy




IV: intravenous; PPI: proton pump inhibitor.

* Patients with clinically significant upper gastrointestinal bleeding (ie, signs of active upper gastrointestinal bleeding including hematemesis, melena, or hematochezia, with or without hemodynamic instability or blood transfusion requirement) should be started on a high-dose twice-daily IV PPI prior to endoscopy as part of their initial management. The twice-daily IV PPI may be switched to oral PPI following endoscopy. Refer to UpToDate topics on management of upper gastrointestinal bleeding.

¶ The choice of treatment often depends on the preference of the endoscopist. Options include thermal coagulation with or without epinephrine local injection, endoscopic clips, endoscopic band ligation, and argon plasma coagulation. Hemostatic spray may be used in patients when these endoscopic treatments fail to control massive gastrointestinal bleeding.

Δ Oral PPI (eg, omeprazole 20 mg twice daily).

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Graphic 141350 Version 2.0

## Contributor Disclosures

**Andres Gelrud, MD, MMSc** Consultant/Advisory Boards: AbbVie [Pancreatic exocrine insufficiency]; Ariel [Genetic testing]; National Pancreas Foundation [Ad honorem]. Speaker's Bureau: AbbVie [Pancreatic insufficiency]. All of the relevant financial relationships listed have been mitigated. **John R Saltzman, MD, FACP, FACG, FASGE, AGAF** No relevant financial relationship(s) with ineligible companies to disclose. **Shilpa Grover, MD, MPH, AGAF** No relevant financial relationship(s) with ineligible companies to disclose.

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