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Diagnosis and management of Mallory Weiss syndrome

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Abstract

Mallory-Weiss Syndrome (MWS) is a gastrointestinal condition characterized by longitudinal mucosal lacerations at the gastroesophageal junction, typically following forceful vomiting or sudden increases in intra-abdominal pressure. Although often self-limiting, MWS can lead to upper gastrointestinal bleeding with symptoms such as hematemesis, melena, epigastric pain, and hemodynamic instability. The diagnosis is established through detailed anamnesis, physical examination, and endoscopic confirmation, which remains the gold standard for detecting mucosal tears and assessing bleeding severity. Risk factors include chronic alcohol consumption, retching, hiatal hernia, and gastroesophageal reflux, though some patients may present without identifiable causes. Management primarily involves hemodynamic stabilization, pharmacologic therapy with proton pump inhibitors, and, when needed, endoscopic interventions such as epinephrine injection, thermal coagulation, hemoclips, or band ligation. In rare cases, angiographic or surgical approaches may be indicated for persistent or massive bleeding. Understanding the anatomy and physiology of the esophagus is essential to recognize MWS and differentiate it from other causes of upper GI bleeding, including Boerhaave syndrome. This review highlights the importance of early diagnosis and tailored treatment strategies to reduce complications and improve outcomes in patients with Mallory-Weiss Syndrome.

Keywords: Mallory-Weiss Syndrome; Upper Gastrointestinal Bleeding; Endoscopic Management; Esophageal Laceration; Hematemesis

1. Introduction

Mallory-Weiss Syndrome (MWS) is a condition characterized by the presence of superficial longitudinal mucosal lacerations (Mallory-Weiss tears) in the upper gastrointestinal tract, primarily at the gastroesophageal junction. The symptoms of MWS include abdominal pain, hematemesis, and severe vomiting. The vomitus often contains clotted blood and appears like coffee grounds, while the stool may be dark and tarry (melena). In the United States, MWS accounts for 1% to 15% of upper gastrointestinal bleeding in adults and less than 5% in children [1]. According to a systematic review and meta-analysis, 37% of studies on MWS originate from Asia. However, these reviews did not provide specific data on the incidence or prevalence of MWS in Asia, including Indonesia [2].

MWS is commonly associated with risk factors such as severe vomiting, which may be linked to chronic alcoholism, although it may also arise from severe trauma to the chest or abdomen, chronic hiccups, heavy lifting and straining, inflammation of the gastric or esophagus mucosa, hiatal hernia, or cardiopulmonary resuscitation. Although most Mallory-Weiss tears resolve spontaneously, patients experiencing severe or recurrent bleeding episodes requiring intensive care and interventional endoscopy have been reported [1,2]. Therefore, it is essential for clinicians to understand the diagnosis and management of MWS to ensure appropriate treatment for this condition.

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2. The Anatomy of Esophagus

The esophagus is a tubular organ, part of digestive tract, connecting the pharynx to the stomach. It serves as the conduit through which food passes to reach the stomach for further digestion. The esophagus follows a path behind the trachea and heart, in front of the spine, and through the diaphragm before entering the stomach. It is divided anatomically into three segments: cervical, thoracic, and abdominal. The cervical segment begins at the cricopharyngeus and ends at the suprasternal notch. This segment lies just behind the trachea, connected to it by loose connective tissue. Posteriorly, the prevertebral fascia connects the esophagus to the sixth through eighth cervical vertebrae. The thoracic duct can be found to the left of the sixth cervical vertebra. The carotid sheath and the lower poles of the lateral thyroid glands lie laterally to the esophagus in the lower neck [3,4].

The thoracic segment lies between the spine and the trachea in the superior mediastinum, extending from the suprasternal notch to the diaphragm. As the esophagus proceeds distally, it passes behind the aortic arch at the T4–T5 intervertebral disc level and enters the posterior mediastinum. The abdominal segment travels from the diaphragm to the gastric fundus. This segment descends through the right diaphragmatic hiatus at the level of the T10 thoracic vertebra and enters the gastric cardia at the T11 level [3,4].

Typically, the adult esophagus is about 9 to 10 inches (23 to 25 cm) long, with sphincters located at both its proximal and distal ends, a mucosa-lined lumen with connective tissue, and an outer layer of smooth muscle. The upper esophagus sphincter allows food to pass only in one direction into the esophagus, while the lower esophagus sphincter permits one-way passage into the stomach [3,4].

The cervical esophagus and upper esophagus sphincter receive blood supply from branches of the inferior thyroid artery. The thoracic esophagus is supplied by esophagus arteries from the aorta and terminal branches of the bronchial arteries. The abdominal segment and lower esophageal sphincter receive blood from the left gastric artery and branches of the left phrenic artery, which supply the submucosal layer of the esophagus. Venous blood drains into the superior vena cava via the submucosal plexus. The azygous system drains the proximal and distal segments, while the middle segment is drained by additional vessels branching from the left gastric vein that feeds into the portal vein [3,4].

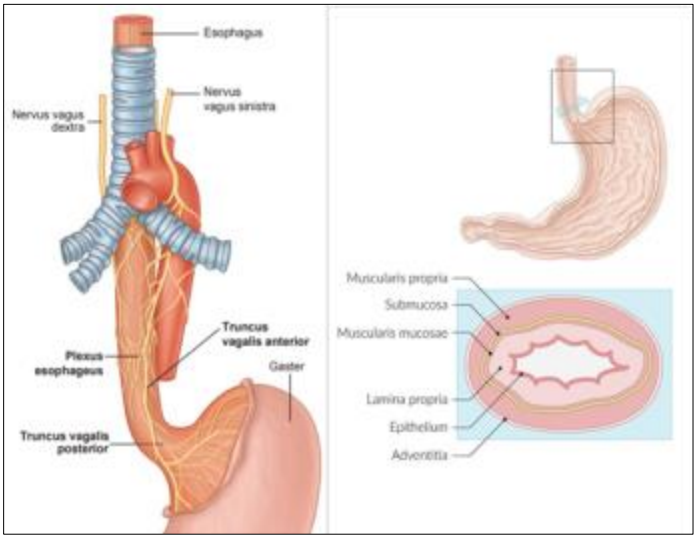

Figure 1 Anatomy of Normal Esophagus [3,4]

The lymphatic vessels and lymph nodes collectively provide lymphatic drainage for the esophagus. These vessels begin as endothelial-lined sacs or protrusions and then form larger lymphatic vessels that run longitudinally along the esophagus. The direction of flow is determined by paired semilunar valves. These vessels merge in various regions to drain into their respective regional lymph nodes. Drainage occurs in three esophageal segments, with significant interconnections. Lymph from the proximal esophagus drains into deep cervical lymph nodes and then into the thoracic duct. The middle third drains into superior and posterior mediastinal nodes. The distal third ultimately drains into the gastric and celiac lymph nodes.[3,4]

Esophageal innervation involves both the sympathetic and parasympathetic nervous systems, primarily via the vagus nerve and spinal nerves (T1 to T10) through the cervical and thoracic sympathetic trunks. The vagus nerve is responsible for parasympathetic motor function of esophageal muscles and glands. The thoracic and cervical sympathetic chains contribute to vasoconstriction, sphincter contraction, relaxation of muscular walls, and increased glandular activity and peristalsis [3,4].

3. The Physiology of Esophagus System

The primary function of the esophagus is to secrete mucus and transport food that enters the mouth through the pharynx into the stomach. The upper esophageal sphincter (pharyngoesophageal sphincter) is a ring-like muscle tissue that normally remains closed in a contracted position. During swallowing, these muscles temporarily relax to allow the passage of food, drink, mucus, and saliva from the laryngopharynx into the esophagus. Subsequently, the bolus moves down the esophageal body. Peristaltic movements push the bolus downward through primary and secondary peristalsis. During the pharyngeal phase of swallowing, the pharyngeal muscles contract, initiating a strong primary peristaltic wave that sends the bolus through the upper esophageal sphincter with kinetic energy. This wave continues along the esophagus as the primary peristaltic wave. If the primary peristalsis is inadequate, leading to continued esophageal distension, a secondary peristaltic wave is initiated to complete the bolus transit into the stomach [3,4].

The lower esophagus sphincter (cardiac or gastroesophageal sphincter) is located just over one inch (approximately 3 cm) from the junction of the esophagus and stomach. Similar to the upper sphincter, it usually remains contracted and closed, mainly to prevent gastric contents from refluxing into the esophagus. This sphincter is controlled involuntarily and is triggered to open during esophageal peristalsis, allowing the bolus to enter the stomach and fulfill the esophagus's primary function [3,4]. Nonetheless, substances transmitted to the stomach via the esophagus may also be expelled back through vomiting, eructation, or activation of the vomiting reflex. However, these functions are generally undesirable as they may lead to malnutrition and esophageal damage if persistent [3,4].

4. Risk factors

Mallory-Weiss Syndrome (MWS) has several risk factors, with heavy alcohol consumption being the most significant, as approximately 50% to 70% of patients diagnosed with MWS have a history of substantial alcohol intake. The severity of upper gastrointestinal bleeding in MWS is also reported to be higher in the presence of portal hypertension and esophageal varices. Furthermore, the association between hiatal hernia, a protrusion of an organ, usually the upper part of the stomach, into the thoracic cavity through the diaphragmatic esophageal opening and MWS remains debated. While hiatal hernia has been observed in several MWS cases, a case-control study found no difference in the incidence of hiatal hernia between MWS patients and control groups [1].

Another frequently observed risk factor is persistent hiccups. Previous studies found that patients presenting to the hospital with MWS symptoms and complications often experienced continuous hiccups. A meta-analysis also revealed a correlation between hiccups and the development of MWS [2].

Other notable risk factors include bulimia nervosa, hyperemesis gravidarum, and gastroesophageal reflux disease (GERD). These conditions all involve the regurgitation of gastric contents into the esophagus. However, in a significant proportion of patients approximately 25%, no identifiable risk factors are found. MWS may also be triggered by repeated episodes of sudden increases in intra-abdominal pressure such as retching, vomiting, straining, coughing, cardiopulmonary resuscitation (CPR), or blunt abdominal trauma [1,2].

Iatrogenic MWS may also occur, though rarely. It can develop as a complication of invasive procedures, such as upper gastrointestinal endoscopy or transesophageal echocardiography (TEE). Upper gastrointestinal endoscopy itself has a very low complication rate for inducing MWS, ranging from only 0.07% to 0.49% [1,2,5].

5. Pathophsiology

The exact mechanism of Mallory-Weiss Syndrome (MWS) is not fully understood; however, it has been proposed that when there is a sudden and significant increase in intra-abdominal pressure, gastric contents under pressure flow proximally into the esophagus. Mallory-Weiss Syndrome (MWS) is one of the common causes of acute upper gastrointestinal bleeding and is characterized by superficial longitudinal mucosal lacerations (Mallory-Weiss tears). These tears typically occur at the gastroesophageal junction and may extend proximally to involve the lower to middle esophagus or distally to involve the proximal stomach (Figure 1) [1]. This excessive pressure leads to longitudinal

mucosal lacerations that may reach the submucosal arteries and veins, causing upper gastrointestinal bleeding (UGIB) [6,7]. The location of the tear is clinically significant because it helps differentiate MWS from other conditions, such as Boerhaave syndrome. In contrast, the tear in MWS is non-transmural (Figure 2) [3,4].

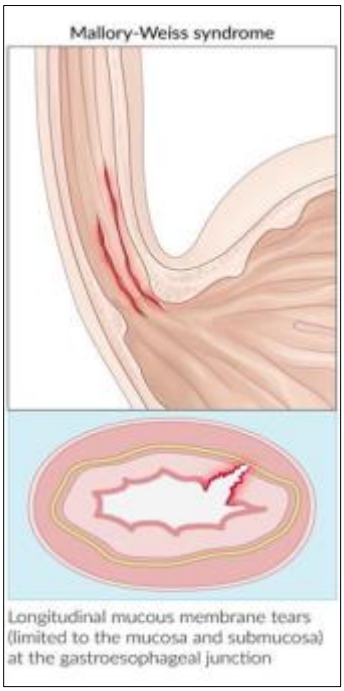

Figure 2 Anatomy of the esophagus in Mallory-Weiss Syndrome [3,4]

6. Diagnosis

Diagnosis of MWS is established through a combination of patient history, physical examination, and supporting investigations.

6.1. Anamnesis

MWS may be asymptomatic in mild cases. In approximately 85% of cases, the main presenting symptom is hematemesis, ranging from blood-streaked mucus and massive bright red bleeding to dark-colored vomitus [1,6,8]. When the blood is fresh, it appears bright red. Patients with MWS typically experience forceful vomiting followed by hematemesis. The volume of hematemesis may be small, such as a tablespoon, or large enough to cause signs of hemodynamic instability. Epigastric or chest pain may also occur in MWS due to intense vomiting [6,7].

In cases of massive bleeding, other symptoms such as melena, dizziness, or syncope may appear, although these are rare, especially among patients with a history of excessive alcohol consumption [1,2,8]. Melena, or black tarry stools, results from partially digested blood originating from a Mallory-Weiss tear. As the tear bleeds, the blood enters the stomach and is broken down, resulting in a tar-like appearance in the stool. Melena is a classic sign of UGIB, including esophagus bleeding in MWS [6,7].

Fatigue may also occur as a symptom of anemia. Although bleeding from a Mallory-Weiss tear may stop spontaneously in some cases, it can recur, leading to anemia. Patients with MWS may show secondary signs of anemia such as fatigue, shortness of breath, dizziness, and pallor [6,7]. Epigastric pain is usually present and may indicate a predisposing condition like gastroesophageal reflux disease (GERD) [1,2,8].

A history of alcohol use is also important to assess, as heavy alcohol consumption is a key predisposing factor, reported in about 50% to 70% of MWS cases. Additionally, aspirin use has been observed in up to 30% of patients with MWS, thus a medication history including aspirin use should be obtained [1,2,8].

6.2. Physical Examination

Following initial history taking, a thorough physical examination should be performed, especially if the patient presents with hematemesis, as management priorities depend on the severity of the bleeding. Physical signs specific to MWS may not be evident. Symptoms of MWS resemble those of other hemorrhagic shock conditions. Some patients may have significant internal bleeding, and therefore, clinical signs of massive hemorrhage and shock should be assessed, including tachycardia, thready pulse, hypotension, dehydration, decreased skin turgor, and delayed capillary refill, with urgent intervention as needed. Digital rectal examination may be performed to confirm signs of melena [1,8].

Both the Glasgow-Blatchford score (GBS) and shock index have been successful in predicting the need for blood transfusion. The GBS is more accurate than the shock index. However, due to its complexity, the shock index may be more useful for rapid assessment of transfusion needs. GBS appears to have the highest area under the curve when predicting the need for endoscopic intervention, followed by the shock index, while the AIMS65 score has the lowest predictive value. GBS and AIMS65 may yield high scores in some individuals due to underlying gastrointestinal conditions rather than the severity of MWS. The Rockall Score is used to assess the risk of death and rebleeding in patients with upper gastrointestinal bleeding, including Mallory-Weiss syndrome. Although Mallory-Weiss tears generally have a good prognosis, this score is helpful in assessing the severity of bleeding. Thus, rockall, GBS and AIMS65 are most effective when used following the algorithm shown in Figure 3 [9,15].

Table 1 Rockall, GBS and AIMS65 [9,15]

	Rockall Score[a]			
	0	**1**	**2**	**3**
Age	< 60 years	60 – 79 years	≥ 80 years	
Shock	"No shock": SBP > 100 mm Hg and HR < 100 bpm	"Tachycardia": SBP > 100 mm Hg and HR > 100 bpm	"Hypotension": SBP < 100 mm Hg	
Comorbidity			IHD, CHF, any major comorbidity	Renal failure, liver failure, disseminated malignancy
Diagnosis[b]	Mallory-Weiss tear or no lesion observed	Peptic ulcer disease, erosive esophagitis	Malignancy of UGI tract	
Stigmata of recent hemorrhage[b]	Clean-based ulcer, flat pigmented spot		Blood in UGI tract, clot, visible vessel, bleeding	

	Glasgow – Blatchford score[c]					
	0	**1**	**2**	**3**	**4**	**6**
Blood urea nitrogen, mg/dL	< 18.2		≥ 18.2 to < 22.4	≥ 22.4 to < 28	≥ 28 to < 70	≥ 70
Hemoglobin, men g/dL	≥ 13	≥ 12 to < 13		≥ 10 to < 12		< 10
Hemoglobin, women g/dL	≥ 12	≥ 10 to < 12			-	< 10
Systolic blood pressure, mm Hg	≥ 10	≥100 to< 109	≥90 to < 99	< 90		
Other markers		Pulse rate ≥ 100 bpm; melena[4]	Syncope[d]; hepatic disease[d]; heart failure[d]			

AIMS65 score[4]						
Albumin <3.0 gg/dL	1					
INR > 1.5	1					
Altered mental status	1					
Systolic blood pressure < 90 mm Hg	1					
Age > 65 years	1					

SBP, systolic blood pressure; HR, heart rate; bpm, beats per minute; IHD, ischemic heart disease; CHF, congestive heart failure, UGI, upper gastrointestinal; INR, international normalized ratio. [a] Maximum score in the postendoscopic Rockall score: 11 points; maximum score in the pre-endoscopic Rockall score: 7 points. [b]Variables not included in the pre-endoscopic Rockall Score. [c]Maximum score in the original Glasgow-Blatchford score: 23 points; maximum score in the modified Glasgow-Blatchford score: 16 points. [d]Variables not included in the simplified Glasgow-Blatchford score. [e]Maximum score: 5 points

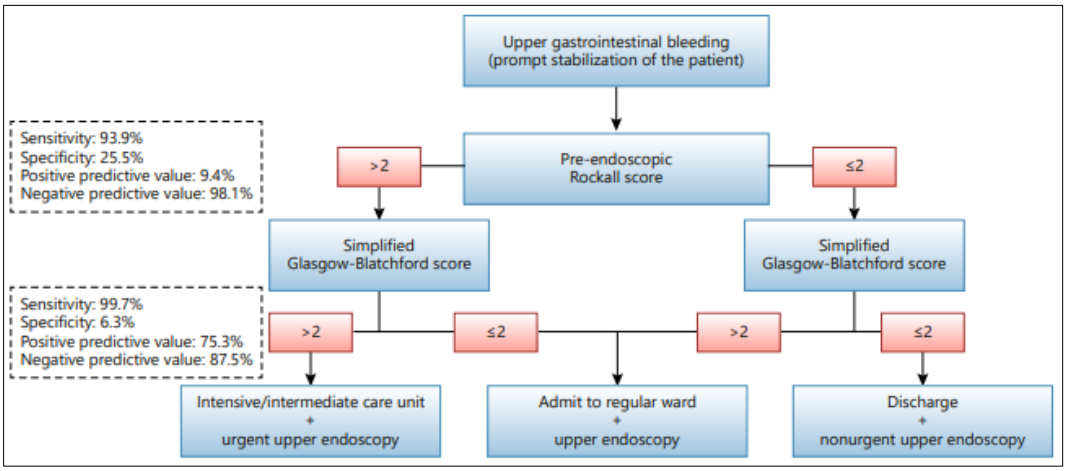

Figure 3 Bleeding Condition Risk Assessment Algorithm [9]

6.3. Suporting Examination

Laboratory examinations include a complete blood count, hemoglobin and hematocrit levels, and coagulation profiles such as bleeding time (BT), prothrombin time (PT), activated partial thromboplastin time (aPTT), and platelet count. Coagulation profiles are used to assess for severe thrombocytopenia and coagulopathy. If thrombocytopenia is observed along with relevant clinical symptoms, a diagnosis of Mallory-Weiss Syndrome should be considered, as chronic alcoholism is one of the major predisposing factors that can result in reduced platelet counts [1,8].

Renal function tests are also necessary to evaluate for renal failure by measuring blood urea nitrogen (BUN) and creatinine levels. If renal failure is detected, it is most likely due to prerenal azotemia unless there is a pre-existing history of chronic kidney disease. Ischemia or myocardial infarction should also be excluded through electrocardiography (ECG) and measurement of cardiac enzymes [1,8].

Upper gastrointestinal (UGI) transnasal esophagoscopy is the gold standard for definitively diagnosing a Mallory-Weiss tear and managing active esophagus bleeding. During endoscopy, active bleeding, clots, or fibrin crusts may be observed over the tear. In most MWS cases, a single linear tear is typically found at the proximal portion of the lesser curvature of the stomach just below the cardia. UGI endoscopy is also helpful in identifying other sources of bleeding, such as esophagus varices, gastric ulcers, or duodenal ulcers. Most Mallory-Weiss tears measure approximately one inch in length [1,8].

Radiological examination using barium contrast is not recommended in these cases due to its low diagnostic value and potential to interfere with endoscopic diagnosis. Angiography may be indicated for identifying and stopping active bleeding from a tear when endoscopy fails or is not feasible [1,8].

7. Differential Diagnosis

MWS should be differentiated from other causes of UGIB. It is crucial to distinguish MWS from Boerhaave syndrome, as both involve esophagus injury. MWS is characterized by non-transmural esophagus tears, whereas Boerhaave syndrome involves transmural perforation of the esophagus. Esophagus rupture in Boerhaave syndrome is thought to result from a sudden increase in intraluminal pressure during vomiting due to poor neuromuscular coordination and failure of the cricopharyngeal muscle to relax [6,7]. Peptic ulcers are the most common cause of UGIB, with characteristic clinical features and definitive diagnosis via endoscopy. Esophagus varices that dilated, tortuous veins around the lower esophagus, commonly secondary to portal hypertension may coexist with MWS. Additionally, arteriovenous malformations, esophagus or gastric neoplasms must also be considered in the differential diagnosis of MWS [1,8]. In Boerhaave syndrome, the entire thickness of the esophagus wall is torn, also known as a transmural tear (Figure 4) [3,4].

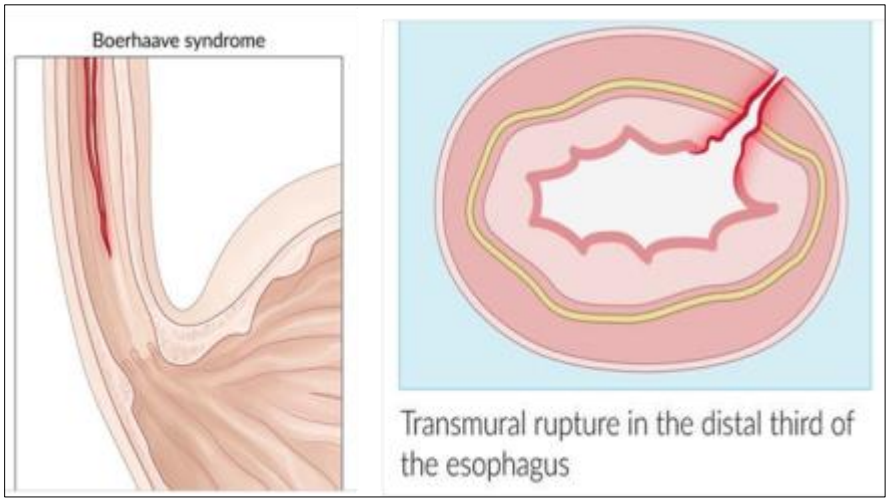

Figure 4 Anatomy of the esophagus in Boerhaave syndrome [3,4]

8. Management

Mallory-Weiss Syndrome is largely self-limiting, with recurrences being rare. Initial management aims to stabilize the patient's general condition. Immediate resuscitation is necessary for patients presenting with active bleeding upon admission. Hemodynamic stability should be assessed through airway, breathing, and circulation (ABC) protocols. Establishing good central or peripheral intravenous (IV) access (preferably two lines) alongside fluid resuscitation is crucial and may be lifesaving in cases of massive hemorrhage. Packed red blood cell transfusion is indicated when hemoglobin levels fall below 8 g/dL or when signs of shock or massive bleeding are present [1,8].

Nasogastric decompression using a nasogastric tube may be considered, especially in patients suspected of having esophagus varices, prior to gastric lavage. Any electrolyte imbalances should be promptly corrected. Coagulation factors should be optimized before proceeding with endoscopy. Most patients managed conservatively are typically hospitalized until hemostasis is achieved and symptoms are resolved [1,8]. Treatment may include:

8.1. Pharmacology Therapy

Proton pump inhibitors (PPIs) and H2 receptor antagonists are administered to reduce gastric acidity, as increased acidity impairs mucosal healing of the stomach and esophagus. Intravenous PPIs may be given to patients undergoing endoscopy. Additionally, antiemetics such as promethazine and ondansetron may be used to control nausea and vomiting [1,8].

8.2. Endoscopic Therapy

While most patients can be managed with observation or conservative measures, some cases require endoscopic or surgical intervention. For patients with risk factors such as portal hypertension or coagulopathy, intensive care including endoscopic hemostasis is recommended. In patients with active bleeding from a Mallory-Weiss tear, endoscopic evaluation is essential for both diagnosis and treatment (e.g., arterial or diffuse bleeding). Early endoscopic

findings such as visible vessels without bleeding or adherent clots may not necessitate endoscopic therapy unless there is recurrent bleeding or associated coagulopathy. In such instances, endoscopic hemostasis is advised [1,8,10].

As shown in Figure 5, endoscopic findings on the left display active pulsatile bleeding from the esophago-cardial junction (arrow), while the right side shows the post-hemostasis condition following cauterization and thrombin spray (10,000 IU) [7].

Esophagogastroduodenoscopy (EGD) is the preferred procedure in all UGIB cases. If bleeding has ceased at the time of endoscopy, no further intervention is usually required. In cases of ongoing or recurrent bleeding, various endoscopic modalities are available. These include local epinephrine injection for vasoconstriction, multipolar electrocoagulation (MPEC), sclerosing agent injection, contact heat treatment, argon plasma coagulation (APC), hemoclip placement, and band ligation, commonly used for bleeding Mallory-Weiss tears [1,8].

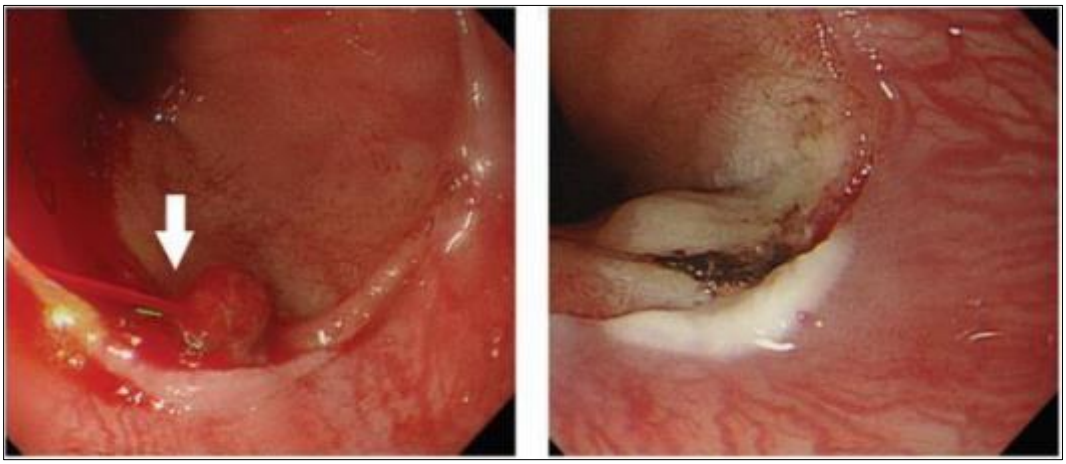

Figure 5 Endoscopic Findings Before and After Hemostasis [7]

There are two types of endoscopic submucosal dissection (ESD) procedures: traditional ESD and endoscopic submucosal tunnel dissection (ESTD), as shown in Figure 6. Figure 6A displays an esophagus lesion after iodine staining, while Figure 6B illustrates the creation of a submucosal tunnel during ESTD. Figure 6C shows the artificial wound after ESTD, and Figure 6D demonstrates a mucosal laceration at the gastroesophageal junction [8].

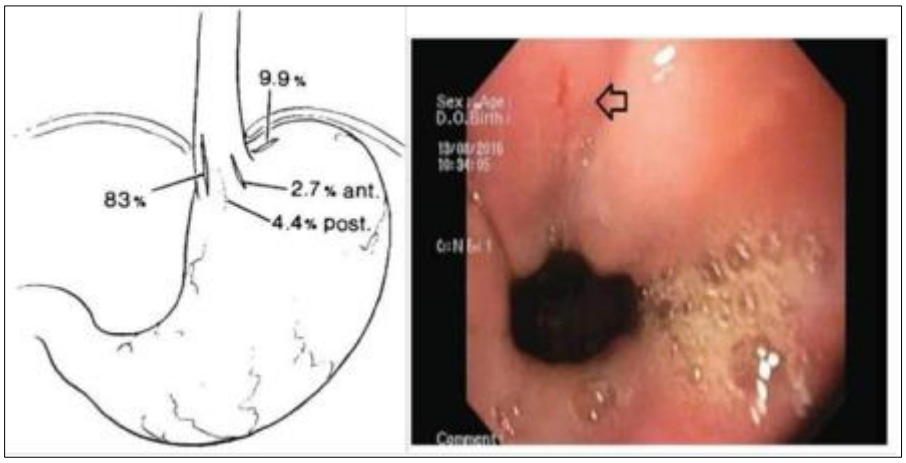

Figure 6 Most Common Locations of Mallory-Weiss Tears [7]

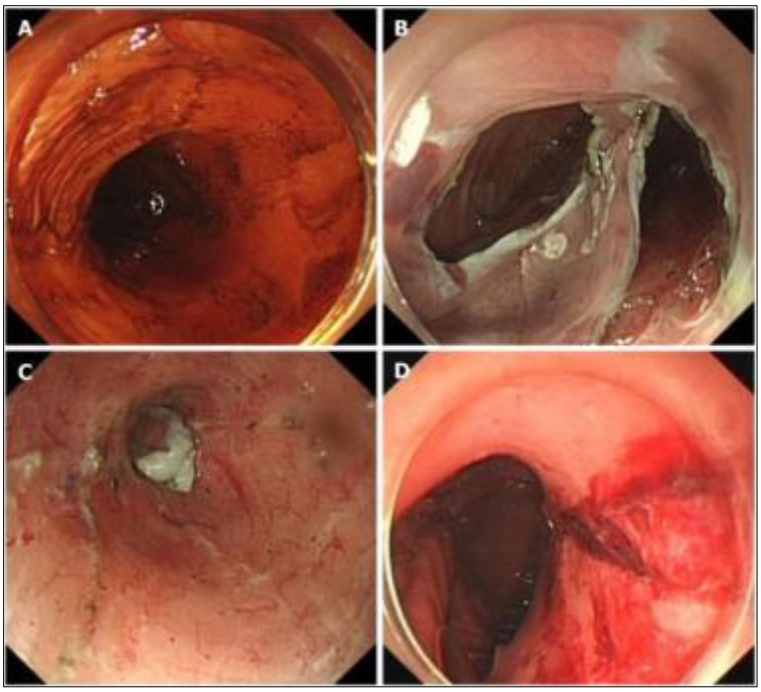

Figure 7 Appearance of Mallory-Weiss Tear on ESTD [8]

8.2.1. Endoscopic Injection Therapy

Endoscopic injection therapy utilizes various drugs, with epinephrine (dilution 1:10,000 to 1:20,000) being the most commonly used. This approach is simple and cost-effective. Regarding rebleeding, length of hospital stay, and transfusion requirements, epinephrine injection therapy has shown better outcomes compared to supportive measures alone. However, due to systemic absorption, epinephrine may cause ventricular tachycardia, so it should be avoided in patients with a history of coronary artery disease [1,8,11]. as shown in Figure 7

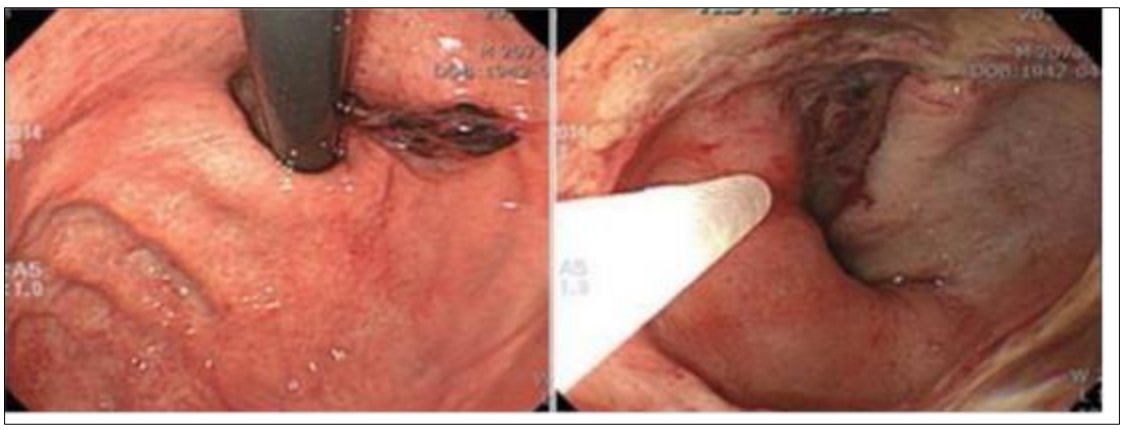

Figure 8 Endoscopic Injection Therapy [7]

8.2.2. Endoscopic electrocoagulation

lectrocoagulation allows the simultaneous application of heat and pressure to the bleeding lesion. However, coagulation is less effective in moist areas like bleeding sites due to rapid heat dissipation by fluids. Accurate device placement is particularly challenging at the lesser curvature of the cardia. Multipolar electrocoagulation significantly improves hemostasis and reduces the need for surgery in bleeding Mallory-Weiss tears, though minor complications may occur. Repeated coagulation increases the risk of transmural injury and perforation because of the thin esophagus wall and absence of serosa at the tear site [1,8,11].

Another available coagulation method is argon plasma coagulation (APC), where the probe is positioned at a distance from the bleeding site. A high-frequency electric current with a low argon gas flow rate (1 L/min) generates coagulation. The absence of direct contact between the catheter and tissue creates a surface burn that minimizes unintended tissue damage and perforation [6,7].

8.2.3. Endoscopic Hemoclip Implantation

Endoscopic hemoclip placement is a straightforward treatment option for bleeding lesions in non-fibrotic tissue such as Mallory-Weiss tears and Dieulafoy's ulcers. Due to the location of Mallory-Weiss tears at the gastroesophageal junction, hemoclip application can be technically challenging. A newly developed rotatable mechanism in the delivery catheter allows controlled adjustment of the hemoclip and easier access to the gastroesophageal junction. Clip dislodgement at this site is common because of large-amplitude contractions in this anatomical region. In cases of deeper lacerations, such as in Boerhaave syndrome, endoclip placement may approximate both edges of the tear and close the perforated lesion [1,8,11].

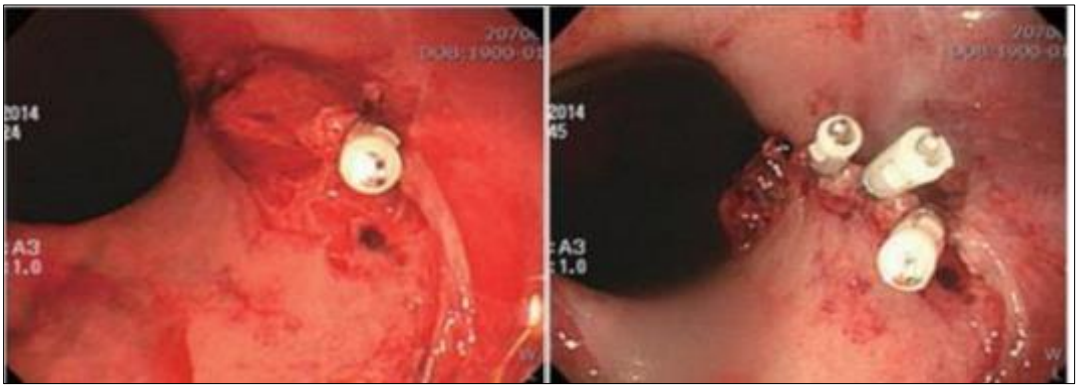

Figure 9 Endoscopic Hemoklip Implantation [7]

8.2.4. Endoscopic Band Ligation

Endoscopic band ligation (EBL) offers significant technological advantages over other hemostatic techniques. Lesions are clearly visualized tangentially during EBL due to direct pressure from the transparent ligation cap. When esophagus perforation occurs, EBL is highly effective for treating bleeding in non-fibrotic tissue. The transparent cap facilitates EBL by immobilizing the bleeding site and preventing movement caused by peristalsis or belching. Deeper portions of visible vessels are ligated, resulting in permanent hemostasis and secure band placement. Large Mallory-Weiss tears can be treated with a single band ligation. There is no significant difference in efficacy or safety between band ligation and epinephrine injection based on a short randomized prospective study involving 34 patients with actively bleeding lesions. Thermal therapy is not recommended for Mallory-Weiss tears associated with portal hypertension or gastric varices; thus, band ligation is preferred [1,8,11].

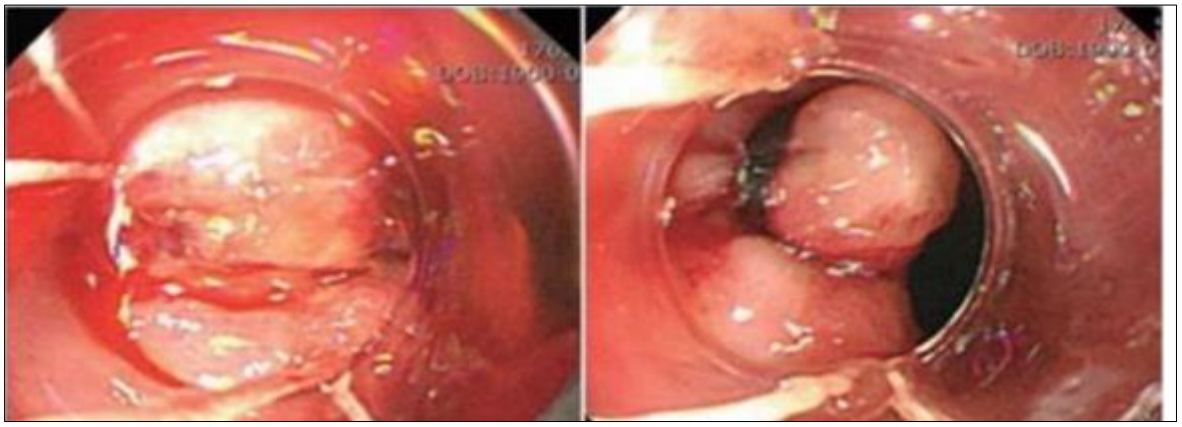

Figure 10 Endoscopic Band Ligation [7]

8.2.5. Angiotherapy

Angiographic interventions, such as injection of vasoconstrictor agents (e.g., vasopressin) or transcatheter embolization using gel foam, may be considered when endoscopy is unavailable or unsuccessful. These approaches aim to obliterate the left gastric artery or superior mesenteric artery to control bleeding [1,8].

8.2.6. Surgical Management

Surgery is rarely required and is considered a last resort following failed endoscopic or angiographic interventions. In patients with severe UGIB where a definitive diagnosis cannot be established prior to surgery, surgeons should explore all potential causes. If no duodenal or gastric lesions are found, a wide gastrostomy is performed via laparotomy. Blind gastrectomy is not recommended at this stage, as esophagus or cardial bleeding may be missed and left untreated. The bleeding source is typically identified once the anterior wall of the stomach is opened 5 to 7 inches using gauze packing. Mallory-Weiss lacerations at the gastric cardia are readily visible and can be repaired using running catgut sutures. If bleeding originates from the esophagus, more extensive exposure or visualization is required. Endoscopy-guided laparoscopic suturing of the tear has demonstrated excellent outcomes [1,8].

9. Conclusion

Mallory-Weiss Syndrome (MWS) is a condition caused by superficial longitudinal mucosal lacerations in the upper gastrointestinal tract, particularly at the gastroesophageal junction. Although MWS is relatively rare, it can have significant clinical implications, especially in cases with active or recurrent bleeding. As a known cause of upper gastrointestinal bleeding (UGIB), MWS requires careful diagnosis and appropriate management to achieve favorable outcomes.

A comprehensive understanding of esophagus anatomy, including its structure, vascular supply, and innervation, is essential in recognizing how Mallory-Weiss tears develop and why they can cause substantial bleeding. Identifying risk factors is also important for early recognition and potential prevention. Diagnosis involves thorough history taking, physical examination, and supportive investigations, with endoscopy playing a central role in confirming the tear and evaluating bleeding severity. Management of MWS depends on the severity of bleeding and the patient's clinical condition. Patients with active bleeding should undergo hemodynamic resuscitation, stabilization, and prompt endoscopic evaluation for hemostasis. Pharmacologic therapy, particularly with proton pump inhibitors, plays a role in reducing gastric acidity and facilitating mucosal healing. Endoscopic treatments such as epinephrine injection, electrocoagulation, hemoclips, and band l igation are selected based on bleeding severity and endoscopic findings. It is important to note that the majority of MWS cases resolve spontaneously without recurrence. Thus, management should be tailored to each patient, balancing conservative approaches with intervention when indicated. A multidisciplinary team is often essential in providing accurate diagnosis and effective care. Ultimately, a comprehensive knowledge of MWS from anatomy and risk factors to diagnosis and management, enables clinicians to optimize care, reduce complications, and support rapid patient recovery.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

Ethical clearance and informed consent were not applicable, given that the study is a literature-based review.

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