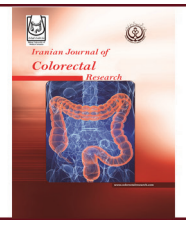
Case Report

Iran J Colorectal Res 2025;13(4):2-7.

Iranian Journal of Colorectal Research



Life-Threatening Mallory-Weiss Tears: From Upper Gastrointestinal Bleed to Multisystem Crisis - A Case Report

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Received: 2025-10-07
Revised: 2025-12-21
Accept: 2025-12-21

Abstract

Introduction: Hypoxic respiratory failure, defined as arterial oxygen tension (PaO_2) <60 mmHg on room air, is commonly caused by pulmonary conditions such as pneumonia, pulmonary embolism, or acute respiratory distress syndrome. Less frequently, systemic factors such as hemorrhagic shock can impair oxygen delivery due to severe anemia and reduced perfusion. Mallory-Weiss tears (MWTs) are longitudinal mucosal lacerations at the gastroesophageal junction, typically caused by vomiting or retching, and may result in significant upper gastrointestinal (GI) bleeding.

Case Description: We report a case of an 81-year-old woman with gastroesophageal reflux disease and a prior cerebral infarct for which she was taking apixaban. She presented with stroke-like symptoms and became hypoxic en route to the hospital, requiring intubation. Laboratory evaluation revealed profound anemia (hemoglobin 2.8 g/dL), metabolic acidosis (pH 6.95, bicarbonate 8 mmol/L), and elevated lactic acid (8.1 mg/dL). She received multiple blood products, reversal of anticoagulation, and supportive care in the intensive care unit. Initial imaging showed a hiatal hernia but no active bleeding. Nasogastric output demonstrated coffee-ground material. Esophagogastroduodenoscopy on day 2 revealed an MWT. She was extubated on day 3 and discharged on day 11.

Discussion: This case emphasizes the importance of considering non-pulmonary causes of hypoxia, particularly severe anemia from occult GI bleeding, even in the absence of overt signs. Hypoxia was primarily driven by reduced oxygen-carrying capacity rather than intrinsic lung pathology. Timely recognition, endoscopic diagnosis, hemostasis, and restoration of hemoglobin reversed respiratory compromise. Clinicians should maintain a high index of suspicion for occult bleeding in unexplained hypoxic respiratory failure.

Keywords: Hypoxic respiratory failure, Mallory-Weiss tear, upper gastrointestinal bleeding, hemorrhagic shock, severe anemia, anticoagulation (apixaban), esophagogastroduodenoscopy, older adult (geriatric)

Please cite this paper as:
Sabharwal S, Young B, Sabharwal D, Clark C, Donnell R. Life-Threatening Mallory-Weiss Tears: From Upper Gastrointestinal Bleed to Multisystem Crisis - A Case Report. *Iran J Colorectal Res*. 2025;13(4):2-7. doi:

Introduction

Hypoxic respiratory failure is defined as a decrease in arterial oxygen tension (PaO_2) to less than 60 mmHg while breathing room air. Hypoxia can be caused by various factors that interfere with gas exchange, such as pulmonary edema, pneumonia, pulmonary embolism, and acute respiratory distress syndrome. Less commonly encountered systemic causes include severe hemorrhagic shock, which can cause hypoxia through reduced oxygen delivery due to decreased hemoglobin content and reduced perfusion.

Mallory-Weiss tears (MWTs) are defined as longitudinal mucosal ruptures that are most commonly seen at the gastroesophageal junction. They are generally caused by sudden increases in intra-abdominal pressure secondary to prolonged episodes of vomiting, retching, or coughing. Although historically attributed to chronic alcohol use, these injuries can occur in a diverse patient population and cause upper gastrointestinal (GI) bleeding of variable severity (1, 2).

The following case documents a patient who presented with clinical hypoxia requiring intubation, which was later found to be due to hemorrhagic shock with a hemoglobin level of 2.8 g/dL, caused by an MWT. This is unique, as to our knowledge, no documented case exists of a hemoglobin level of 2.8 g/dL from an MWT.

Mallory-Weiss syndrome is often considered relatively benign because bleeding is usually self-limited, and severe anemia leading to cardiovascular collapse is rarely attributed to this condition (3). This patient's case was unique in that she presented with hypoxic respiratory failure and neurologic deficits, prompting initial evaluation for stroke. Overt hematemesis was not initially apparent, and recognition of an upper GI bleed relied on indirect findings such as melena and coffee-ground nasogastric aspirate, which may delay diagnosis in older adults. Furthermore, a hemoglobin level of 2.8 g/dL reflects critically reduced oxygen-carrying capacity and can precipitate multisystem organ dysfunction even when respiratory support is adequate. Finally, this severe presentation occurred in the setting of apixaban and aspirin use, highlighting that in select high-risk patients, MWTs can rapidly progress to a multisystem crisis despite anticoagulant reversal and require urgent transfusion support and endoscopic diagnosis (4).

Case Description

Patient Description

The patient is an 81-year-old woman with a history of gastroesophageal reflux disease (GERD), cerebral infarct 6 months prior (for which she was taking apixaban), type II diabetes mellitus, hypertension, and hyperlipidemia, who presented to the emergency department (ED) with a chief complaint of stroke.

Case History

The patient developed left-sided numbness earlier in the day, which prompted emergency medical services to be called. En route to the hospital, she became hypoxic and hypotensive, and she was subsequently intubated.

Physical Examination

The rectal exam showed black stool with presumed occult blood. The patient's vital signs were stable, with a blood pressure (BP) of 113/38 mmHg, heart rate of 79 bpm, and mean arterial pressure of 64 mmHg.

Investigations/Pathology

On arrival at the ED, the patient's hemoglobin was found to be 2.8 g/dL on complete blood count (CBC). Complete metabolic panel (CMP) showed an elevated sodium level of 147 mmol/L, BUN of 48 mg/dL, creatinine of 1.67 mg/dL (baseline ~1.10 mg/dL), AST of 109 U/L, and ALT of 85 U/L. The patient's anion gap was 33 mmol/L.

A subsequent arterial blood gas showed a pH of 6.95, PCO_2 of 36 mmHg, PO_2 of 139 mmHg, and bicarbonate level of 8 mmol/L on mechanical ventilation, with a fraction of inspired oxygen (FiO_2) of 100% and positive end-expiratory pressure (PEEP) of 8 cmH_2O.

A new CBC showed a hemoglobin level of 5.0 g/dL. Lactic acid was elevated at 8.1 mg/dL. The patient was noted to be oozing from every access attempt, and a disseminated intravascular coagulation (DIC) panel was ordered which was negative.

Computed tomography (CT) head scan without contrast and computed tomography angiography (CTA) head and neck scan showed no acute abnormalities. CT of the abdomen and pelvis without contrast showed a medium-sized hiatal hernia. Repeat CBC showed a hemoglobin level of 10 g/dL.

A nasogastric (NG) tube was placed and yielded coffee-ground drainage. A peripheral smear was ordered and showed normochromic, normocytic anemia. Iron studies were within normal limits. The following day, the patient underwent esophagogastroduodenoscopy (EGD), which showed an MWT (Figure 1).

The in-house neurologist was contacted and believed that the patient's symptoms likely represent a recrudescence of a prior stroke in the setting of hypovolemic shock due to a GI bleed.

Treatment Plan

Two units of packed red blood cells (PRBCs) were ordered. The patient was transferred to another ED, where apixaban was reversed with Balfaxar, and desmopressin was given due to her aspirin use. The patient was started on a pantoprazole bolus and infusion for presumed upper GI bleeding, along

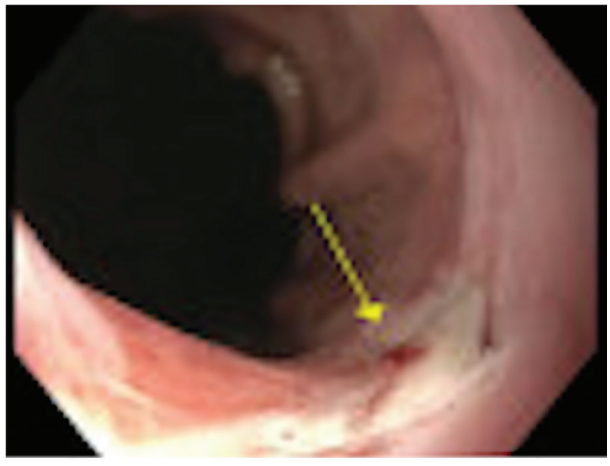

Figure 1: The image is an EGD showing a Mallory-Weiss tear (yellow arrow).

with 4 units of PRBCs, 2 units of liquid plasma, 1 unit of cryoprecipitate, and 1 unit of platelets. The patient was transferred to the intensive care unit (ICU) and started on norepinephrine for BP support. Gastroenterology was consulted in the ICU.

Expected Outcome

The patient continued to improve with volume resuscitation, and her elevated liver function tests, likely secondary to hypoperfusion, began to decrease.

Actual Outcome

The patient was extubated on day 3 of hospitalization and discharged on day 11 to skilled nursing on pantoprazole 40 mg twice daily for 8 weeks, then 40 mg once-a-day thereafter. She was also advised to continue taking aspirin but stop taking apixaban due to her recent blood loss anemia.

Consent

Consent for publication was obtained from the patient.

Discussion

Upper GI hemorrhage, as represented by an MWT, can lead to hypovolemic shock and severe anemia, significantly impairing oxygen transport and tissue perfusion. In older adults with chronic comorbidities such as cerebrovascular disease, the resulting hypoxia has the potential to exacerbate neurological dysfunction or mimic acute neurologic events such as stroke. While the individual effects of GI bleeding on systemic oxygenation and hemodynamics are well documented, the specific interplay between MWTs, hypoxia, and neurologic decline in this population is not extensively described in the literature. Available case reports and series do, however, underscore the serious physiological consequences of upper GI bleeding, including hemodynamic instability and respiratory compromise, suggesting that this may be a less recognized but clinically significant relationship (5-7).

Although MWTs are often regarded as a relatively benign cause of upper GI bleeding, recent multicenter data suggest that outcomes can be more serious, particularly in patients with active bleeding or comorbidities, as the 30-day mortality for MWT was comparable to that of peptic ulcer bleeding despite a lower rebleeding rate (8). Patients in the critical care setting may present with more serious GI illnesses or coexisting comorbidities. Endoscopic assessment enables monitoring and treatment (9).

MWT is caused by a sudden increase in intra-abdominal pressure that is usually accompanied by vomiting, retching, or coughing. The tear occurs at the gastroesophageal junction and can cause significant hemorrhage, especially if it is large or in proximity to a blood vessel (1). Contributing factors include alcohol use, varices, ulcers, GERD, and anticoagulant use (6, 10, 11). The presence of a hiatal hernia, as identified in this patient, has been found to be a structural factor that increases mucosal susceptibility (12).

Hemorrhagic shock secondary to MWT is caused by significant intravascular volume loss, which subsequently reduces cardiac output and oxygen-carrying capacity. A hemoglobin level of 2.8 g/dL is far below the normal range of 12-16 g/dL and severely compromises oxygen delivery (DO2), which can be expressed by the following equation:

[DO_2 = Cardiac Output x (Hemoglobin x 1.34 x SaO_2 + 0.0031 x PaO_2)]

Tissue hypoxia occurs when DO_2 falls below a critical threshold of approximately 8-10 mL O_2/min/kg, which results in anaerobic metabolism, lactic acidosis, and multiorgan dysfunction (13). If we assume 100% arterial oxygen saturation (SaO_2) and a normal PaO_2 of 100 mmHg, at 2.8 g/dL, DO_2 is drastically reduced, even with compensatory increases in cardiac output, triggering the above pathologic events.

Hypoxic respiratory failure in this clinical scenario may have resulted from:

1. Respiratory Muscle Fatigue: Severe anemia reduces oxygen delivery throughout the body, including to the respiratory muscles. This reduces their endurance, causing hyperventilation and fatigue (14).

2. Pulmonary Aspiration: MWT is often caused by vomiting, leading to aspiration of blood or gastric contents, impairing gas exchange and exacerbating hypoxemia (5).

3. Shock-Induced Lung Injury: Hemorrhagic shock may cause acute lung injury secondary to systemic inflammation and hypoperfusion, subsequently reducing lung compliance and oxygenation (13).

There are no specific case reports documenting a hemoglobin level of 2.8 g/dL resulting from an MWT. However, related studies provide context;

for example, a pediatric case of MWT complicated by aspiration pneumonitis reported a hemoglobin level of 8.7 g/dL following significant bleeding, with respiratory failure necessitating mechanical ventilation (5). Therefore, a hemoglobin level of 2.8 g/dL suggests substantially greater blood loss (estimated at 60-70% of total blood volume), consistent with Class IV hemorrhagic shock (15). Studies of severe anemia, such as those involving Jehovah's Witnesses who refuse transfusion, report high mortality without intervention but also document survival at hemoglobin levels as low as 2-3 g/dL, attributed to extreme compensatory mechanisms like hyperdynamic circulation (16). A hemoglobin level of 2.8 g/dL falls below the critical DO_2 threshold of 4 g/dL identified in animal models, indicating imminent risk of cardiovascular collapse (13). In a porcine model of hemorrhagic shock, hemoglobin levels remained stable initially despite 65% blood loss, only decreasing significantly after fluid resuscitation (17). This suggests that a hemoglobin level of 2.8 g/dL in MWT likely reflects either delayed presentation or hemodilution following resuscitation.

The patient was diagnosed with hypoxic respiratory failure secondary to severe hypovolemic shock caused by an acute upper GI bleed due to an MWT. The severe anemia (hemoglobin level of 2.8 g/dL), metabolic acidosis, and high lactate levels signaled imminent tissue hypoxia; subsequent endoscopy confirmed MWT as the cause of the bleed.

Clinically, MWTs typically present as hematemesis after bouts of vomiting; however, unusual presentations such as silent hypotension or isolated hypoxemia may occur in critically ill or older patients. In some cases, iatrogenic tears may be caused by medical procedures such as endoscopy or cardiopulmonary resuscitation (CPR) (2, 18).

Diagnosis of MWT requires differentiating it from other causes of upper GI bleeding, such as peptic ulcers, esophageal varices, Dieulafoy's lesions, and erosive esophagitis. All of these conditions have distinct endoscopic features and require different treatment modalities (19).

Patients with a hemoglobin level of 2.8 g/dL from hemorrhagic shock secondary to an MWT would present with Class IV shock, which is characterized by tachycardia (>120 bpm), hypotension (systolic BP <70 mmHg), altered mental status, and profound pallor (20). Hypoxic respiratory failure would manifest as tachypnea (>30 breaths/min), hypoxemia (PaO_2<60 mmHg), and potentially hypercapnia if respiratory muscles fail.

Identification and reversal of anticoagulant therapy in patients with suspected upper GI bleeding is of great significance, as anticoagulants increase hemorrhage risk and complicate decision-making during therapy. Reversal of factor Xa (activated factor X) inhibitors, such as apixaban, using andexanet alfa, along with adjunctive desmopressin in patients on concurrent antiplatelet therapy, may be important for achieving hemostasis (21).

Prompt volume resuscitation and transfusion strategies are important. Successful management of fluid and blood transfusion in cases of hemorrhagic shock requires proactive management with packed red blood cells, fresh plasma, cryoprecipitate, and platelets. Effective and immediate intervention, including hemostasis, can improve oxygen delivery and reduce the risk of multiple organ failure (22). Given a goal hemoglobin level of >7 g/dL, immediate transfusion of PRBCs in addition to crystalloids is used to restore preload (23). At 2.8 g/dL, rapid transfusion is critical to prevent potential cardiac arrest.

Treatment with PPI infusions has been proven to be effective in stabilizing upper GI bleeding, such as mucosal-vascular lesions, through reduced gastric acid secretion and increased blood clot stability (24).

In elderly patients with high stroke risk but recent severe bleeding, clinicians must carefully assess whether to resume anticoagulation. In this case, apixaban was discontinued, and the patient remained on aspirin alone, aligning with literature indicating the benefit of individualized assessment and treatment (25, 26).

Most MWTs resolve spontaneously, but active bleeding may require endoscopic intervention such as hemoclips, injection therapy, or newer methods such as the tulip-bundle technique (12, 27). Proton pump inhibitors are standard therapy for mucosal healing and prevention of recurrence (28). Consideration must be given to whether anticoagulation should be restarted, modified, or discontinued after bleeding has resolved, particularly in elderly patients with thromboembolic risk (25).

Endoscopy is regarded as the diagnostic test of choice for MWTs owing to its ability to observe mucosal lesions directly and to provide immediate therapy as necessary (29). Clinical features, including acute anemia in conjunction with bleeding, increased lactate, and coagulopathy, are suspicious for active hemorrhage and can prompt immediate endoscopy (28, 30). Risk stratification tools, including the Blatchford score, help identify patients for urgent endoscopy or transfusion (31).

The occurrence of MWTs in patients on anticoagulation therapy is correlated with worse outcomes and is associated with increased risks for hemorrhage, rebleeding, and comorbid conditions. One study reported that mortality can be as high as 23.5% in patients with MWTs after transesophageal echocardiography, depending on age and ongoing anticoagulation (32).

Treatment with anticoagulant drugs such as apixaban significantly increases the rate and severity of GI hemorrhage in older adults with comorbid conditions. Evidence shows that this population has increased susceptibility to severe bleeding and reduced survival rates in case of hemorrhage (33).

Anticoagulation management for this patient population requires a careful balance between stroke prevention and bleeding risks, with recent studies calling for the use of reversal agents and investigation into alternative treatment methods post-discharge (25, 26).

Severe GI bleeding can cause respiratory failure secondary to hypovolemic shock and decreased oxygen-carrying capacity. Subsequently, the resultant tissue hypoxia impairs the function of critical organs, including the neurologic and hepatic systems. The arterial blood gas in this case, showing a pH of 6.95, a bicarbonate of 8 mmol/L, and an elevated lactate, highlights the severity of hypoperfusion. These findings align with current literature that emphasizes the need for urgent resuscitation, hemostasis, and ventilatory support in cases of hypoxia secondary to GI bleeding (22). In rare cases, upper GI bleeding can lead to severe aspiration pneumonitis and respiratory failure, as seen in a reported case of MWT in an infant requiring mechanical ventilation and nitric oxide therapy for respiratory support (5).

The key challenge in warfarin therapy management is to balance rebleeding risk with the need for anticoagulation in patients with higher stroke and cardiac event risk. Evidence guiding the optimal timing and strategy for resuming anticoagulation is still lacking; therefore, decisions are made on a case-by-case basis (26, 32).

There is a clear need for longitudinal studies that evaluate outcomes for patients managed for MWT while on anticoagulation and that establish evidence-based guidelines for restarting anticoagulation in GI bleeding.

The literature suggests that MWT rarely causes such extreme anemia (reflected by a hemoglobin level of 2.8 g/dL), as it is considered a benign condition; bleeding typically arrests spontaneously in 80-90% of cases (34). Therefore, coexisting factors (such as coagulopathy or aspiration) and diagnostic accuracy (i.e., an alternative bleeding source) must be considered. Furthermore, the established narrative of MWT as a benign condition must be challenged in such extreme cases, as it underestimates the potential for mortality.

Conclusion

This case describes an unusual and potentially fatal presentation of a MWT, complicated by hypoxic respiratory failure in an elderly patient undergoing anticoagulation therapy. Hypoxic respiratory failure secondary to hemorrhagic shock from an MWT, with a hemoglobin level of 2.8 g/dL represents a critical emergency. The severe anemia significantly impairs oxygen delivery, precipitating respiratory and systemic failure. The condition was successfully managed through endoscopic diagnosis, reversal of anticoagulation therapy, and prompt resuscitative measures. The literature highlights the need for urgent endoscopic diagnosis as well as individualized management strategies, particularly regarding anticoagulation in high-risk patients. In general, the majority of MWTs are best managed conservatively, whereas more severe cases require intensive, multidisciplinary treatments. Although rare, this scenario highlights the need for prompt recognition, endoscopic control, and transfusion. Further study is needed to refine management strategies for such outliers, guide long-term anticoagulation regimens, and improve risk stratification in elderly patients presenting with GI hemorrhage.

Authors' Contribution

Sahil Sabharwal, Brandyn Young and Deepak Sabharwal wrote the main manuscript. Sahil Sabharwal prepared the figures. Sahil Sabharwal wrote the abstract. **Christopher Clark and Robert Donnell guided in writing and reviewing the manuscript.**

- **Funding**: None of the authors have any funding to declare.

- **Competing Interests:** There are no competing interests to declare
- **Data Availability:** This manuscript does not report data generation or analysis
- **Third Party Material/Consent for Publication:** Patient consent was obtained. All of the material is owned by the authors and/or no permissions are required. Case reports are not considered Human Subject Research by the UAMS IRB and do not require IRB Review or Approval.
- **Dual publication:** The results/data/figures in this manuscript have not been published elsewhere, nor are they under consideration by another publisher

Conflict of Interest: None declared.

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