

Mallory-Weiss Syndrome with Oesophageal Tear: A Case Report

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ABSTRACT

A 67-year-old male patient presented to the hospital with complaints of mild-grade fever, gradually progressive cough, and frequent episodes of vomiting. The initial diagnosis was acute gastritis with severe dehydration. The patient had a medical history of Type 2 Diabetes Mellitus (T2DM) for 2 years and Hypertension (HTN) for 20 years, for which he was already receiving treatment. Due to continuous bleeding, an urgent endoscopy was performed, revealing a linear tear in the mucous membrane of the oesophageal region, leading to a diagnosis of Mallory-Weiss Syndrome (MWS). The patient was treated with antibiotics, antipyretics, antiemetics, and antacids. Subsequently, the patient was symptomatically improved, leading to his discharge from the hospital.

Keywords: Mallory Weiss Syndrome (MWS), Hypertension (HTN), Type 2 Diabetes Mellitus (T2DM)

INTRODUCTION

Mallory-Weiss Syndrome (MWS) is a condition characterized by longitudinal, non-perforating superficial lacerations at the gastroesophageal junction. It is commonly observed in cases of upper gastrointestinal bleeding. The syndrome was first identified by George Kenneth Mallory and Soma Weiss, who were renowned medical professionals. Males have a slightly higher susceptibility to MWS, with a prevalence of 2 to 4% compared to women. The condition is more frequently seen in individuals between the ages of 40 and 60.^[1] In men, the pathology often arises from forceful

vomiting, causing the rupture of the pathologically altered mucous membrane in the upper stomach. Conditions such as gastritis, cholecystitis, and tumors can contribute to changes in the gastric mucosa.^[2] MWS commonly affects the duodenum and stomach and is characterized by various symptoms such as hematemesis, dyspnea, epigastric pain and melena. It can sometimes lead to anemia or even shock due to severe bleeding. There are several mechanisms involved in MWS, including the obstruction of the gastric pylorus, which increases pressure in the stomach and causes the urge to vomit. Other mechanisms include reduced motility, between the mucosa and submucosa, loss of collagen fibers in the mucosal membrane, and decreased elasticity and motility of the gastro-intestinal tract associated with aging.^[1] Excessive alcohol consumption is found in a significant percentage of patients diagnosed with MWS ranging from 40-80%.^[3] Diagnosis of MWS is typically made through endoscopy, chest X ray for radiological examination and sometimes detection of blood in the stool. It can also occur as a complication of upper endoscopy known as Iatrogenic Mallory Weiss tear.^[1] Conservative medical treatment, including fasting, bed rest, antiemetics, sedation, intravenous antacids, somatostatin, and blood transfusion, can benefit patients with MWS, as the hemorrhage is typically mild and self-limited. However, certain patients with risk factors such as active bleeding), recurrent bleeding signs and comorbid

conditions may require interventional endoscopy or other hemostasis procedures. Various techniques, such as surgery, balloon tamponade, transcatheter embolization, and arterial infusion of vasopressin, have been used to control active bleeding in these cases. Endoscopic methods, including electrocoagulation, injection therapy, haemoclipping, and band ligation, have also been employed to treat Mallory-Weiss syndrome-induced upper gastrointestinal bleeding. [4]

CASE DESCRIPTION

A male patient of age 67 years came to S.S.I.M.S & RC, Davangere, with chief complaints of fever which was mild grade in nature and cough which was sudden in onset gradually progressive in nature, since yesterday along with complaints of 10-15 episodes of vomiting since 2 days and was diagnosed as acute gastritis with severe dehydration. Patient was a known case of T2DM since 2 years and HTN since 20 years and on medication Tab Glycomet Trio 2 (Glimepiride 2mg+Metformin 500mg+Voglibose 0.2mg) 1-0-1, Tab. Amlodac (Amlodipine 5 mg) 0-0-1. On the initial day of admission, the patient was administered Inj. Silaxone SB (Ceftriaxone + Salbactam) 1g BD, Inj. Silpan (Pantoprazole) 40 mg, and Inj. Ondem (Ondansetron) 4mg TID, along with oral intake of Syp Sucrafil-O (Sucralfate + Oxetacine) 10ml BD. The physician recommended conducting laboratory tests for complete blood count, liver function, and serum electrolyte levels. On the following day, due to the complaints of bleeding, the physician recommended Upper Gastro-Intestinal (GI) Endoscopy which illustrates a linear mucosal tear along the Oesophageal Gastric Junction (OGJ) to distal oesophagus and expressed as Mallory Weiss Syndrome (**Fig 1**). Furthermore, the patient was prescribed additional medications: Inj. Pause (Tranexamic Acid) and Inj. Vitamin K (Phytomenadione). On the third day, the patient did not report any specific issues, and the physician advised

continuing the existing treatment. On the fourth day, the patient was discharged with a prescription for medications including Tab. Cefpod CV (Cefpodoxime Proxetil +Clavulanic acid) 125mg 1-0-1 for 5 days, Tab. Dabigatran 110 mg 1-0-0 for 6 weeks, Tab. Metformin 500mg 1-0-0 to continue, Tab. Arbutal Trio (Cilnidipine, Metoprolol Succinate, Telmisartan) 25 mg 1-0-0 to continue, Tab. Nexovas (Cilnidipine) 10mg 0-0-1, Tab. Pan (Pantoprazole) 40mg 1-0-0 for 7 days, Tab. Orofer XT (Ferrous Ascorbate +Folic Acid) 0-1-0 For 60 days. The patient and accompanying individuals received counseling regarding the future treatment plan, were advised to abstain from alcohol consumption, and were cautioned about the importance of adhering to their prescribed medications. Patient was conscious and coherent on examination and vitals of the patient were BP: 150/100mm/Hg, PR :86bpm SPO2: 98% at RA, CVS: S1S2+, P/A: Soft. **Lab Investigations-** Complete Blood Picture: Haemoglobin: 14.4 gm%, Red Blood Cells: 4.6 m/UI , Thousands/cu.mm, Platelet count: 2.6lakhs/cu.mm, Neutrophils: 83%, lymphocytes: 13%, monocytes: 4%, MCV: 91.5 fL, MCH: 31.1 pg , MCHC: 34.%, **Liver Function Test:** Total Bilirubin: 2.7 mg/dl, Direct Bilirubin: 0.5 mg/dl, SGOT: 20 IU/L, SGPT: 24.3 IU/L, Serum Alkaline Phosphatase: 80.1 IU/L, Serum Albumin: 3.5 gm/dl, Globulin: 2.9, Random Blood Sugars: 215mg/dl, Serum Creatinine: 0.92 mg/dl, Serum Sodium: 129.5 mmol/L, Serum Potassium: 4 mmol/L, Serum Chloride: 97 mmol/L, **Endoscopy:** Erosive gastro duodenitis Mallory Weiss tear. The patient, without any existing signs of bleeding, was discharged in a fully healed condition.

DISCUSSION

A Mallory-Weiss tear, accounting for approximately 1.3% to 14% of cases, is a common cause of non-variceal upper gastrointestinal bleeding, representing 3% to 15% of cases [5,6]. In this patient, the presence of Mallory-Weiss tear was found

to be linked with vomiting episodes associated with gastritis. These episodes of retching or vomiting create a significant pressure difference within the stomach, heightening the risk of mucosal laceration.

MWS is a rare cause of gastrointestinal bleeding without varices, typically has a benign course with a favorable outcome in over 90% of cases. However, in some instances, it can lead to a fatal outcome. Several significant risk factors are associated with mortality in patients diagnosed with MWS. These factors include advanced age, presence of shock upon arrival, low initial and minimum hemoglobin levels, prolonged prothrombin time, elevated levels of AST and ALT liver enzymes, endoscopic identification of exposed blood vessels, a history of rebleeding, high volume of blood transfusion, and the presence of the clinical symptom of passing dark, tarry stool. [4]

Moreover, regarding the localization of bleeding, findings indicate that tears and bleeding in the abdominal section of the esophagus stand out due to the highest volume of bleeding. Furthermore, compared to other parts of the esophagus, heavy bleeding is less common in this area, and instead, a milder degree of blood loss tends to prevail. The location of these tears offers advantages in terms of both diagnosing the bleeding and treating it conservatively. Conservative treatment options, such as endoscopic hemostasis using electrocoagulation, can effectively clot small and medium-sized blood vessels. However, this method may not be effective in restoring the integrity of vessels, tissues, and blood circulation. Therefore, resorting to traditional treatment methods for Mallory-Weiss tears becomes necessary. [2] Conversely, endoscopic ligation is a safe technique that can be employed for cases of severe bleeding from MWS, achieving effective hemostasis without complications. [7]

In this patient, MWS was confirmed by both physical examination and upper GI endoscopy. Medications were prescribed for

continued use after discharge. Subsequent follow-up showed significant improvement, with noticeable reduction in body temperature and decreased frequency of vomiting episodes.

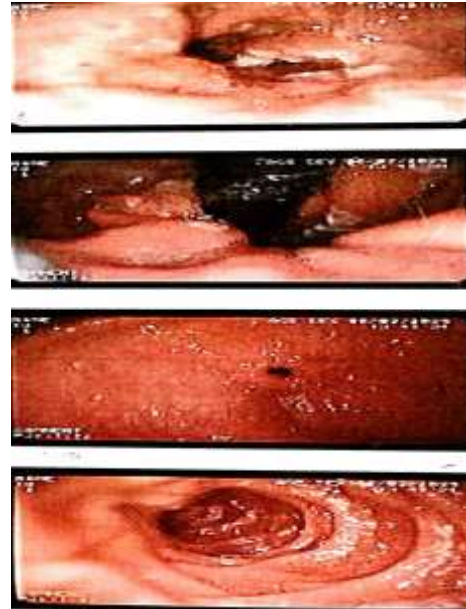


Fig 1: A linear tear has occurred along the junction where the oesophagus and stomach meet, extending towards the distal part of the oesophagus.

CONCLUSION

In conclusion, this case report highlights the diagnosis of MWS in a patient presenting with a linear mucosal tear along the Oesophageal Gastric Junction (OGJ) extending to the distal oesophagus. This study indicates that the development of MWS in this patient can be attributed to the presence of acute gastritis accompanied by vomiting. Treatment consisted of antibiotic therapy, antiemetics and antacids. It emphasizes the importance of comprehensive patient history assessment and prompt diagnosis for effective management of MWS. Neglecting immediate treatment can have severe and potentially life-threatening consequences.

CONSENT FOR PUBLICATION

The patient granted written informed consent for the publication of this case report.

Declaration by Authors

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