

Recurrent Hemorrhage in the course of Mallory-Weiss Syndrome – case report

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Abstract

A 46-year-old patient presented to the department with symptoms suggestive of an exacerbation of chronic pancreatitis. The patient had a history of numerous afflictions associated with alcoholism, such as chronic pancreatitis, hepatic steatosis, erosive gastritis, duodenal ulcers, esophageal hiatal hernia, and Mallory-Weiss syndrome. This case reports on recurrent episodes of intractable hemorrhage in the course of Mallory-Weiss syndrome.

Key words

Mallory-Weiss syndrome, hemorrhage, alcoholism

INTRODUCTION

Mallory-Weiss syndrome (MWS) manifests as an upper gastrointestinal (UGI) bleeding disorder occurring secondary to non-penetrating, longitudinal mucosal lacerations (Mallory-Weiss tears). Mallory-Weiss tears (MWT) are commonly found in proximity to the gastroesophageal junction and the gastric cardia. Episodes of vomiting, retching, or hiccupping are the main culprits in inciting the genesis of this disorder [1,2]. These maneuvers are all fundamentally different but are mutually linked by the fact they generate a sharp rise in intraesophageal/intragastric pressure. Thus, certain demographics such as alcoholics, bulimics, patients receiving gastroscopy, etc. that are known to habitually perform these maneuvers are at greater risk for developing MWS [3,4]. Hematemesis or coffee ground vomiting are the cardinal presenting signs in MWS [5,6]. Most cases resolve spontaneously with a minority of cases requiring significant intervention [1,7]. The severity and number of intractable hemorrhages that occur over a relatively short hospitalization quite uncommon and truly attests to uniqueness of this case.

CASE

A 46-year-old male alcoholic was admitted to the Department of Internal Medicine complaining of severe epigastric pain, nausea, weight loss (BMI 16.9 kg/m²), malaise, as well as lethargy and weakness. Physical examination of the patient revealed tachycardia (100 beats per minute), prehypotensive state (blood pressure 100/70 mmHg), and right upper quadrant tenderness on palpation in the area of the liver. The patient underwent ultrasonography that was significant for hepatic steatosis, pancreatic calcification, and partially dilated pancreatic duct (5 mm in diameter). Upon admission, the patient also underwent a full blood examination which was remarkable for hypochromic, microcytic anemia with

diminished levels of hemoglobin of 5.9 g/dL, hematocrit of 20.5%, mean corpuscular volume of 74.3 fL, mean corpuscular hemoglobin of 21.4 pg (normal MCH 27.0-32.0 pg), mean corpuscular hemoglobin concentration of 28.8 g/dL (normal MCHC 31.0-36.0 g/dL), serum iron – 14.0 ug/dL.

The patient's alcohol-related maladies first appeared when the patient was 40 years old with a bout of gastritis, with inflammation most pronounced in the fundus and the upper portion of the body of the stomach. At the age of 42 the patient was admitted to hospital because of a duodenal ulcer and acute pancreatitis. A few months later the patient returned with exacerbations of the duodenal ulcer and pancreatitis; further investigation revealed the development of an esophageal hiatal hernia. Over the course of the next year and a half the patient visited the hospital four more times with recurrent exacerbations of pancreatitis, bleeding ulcers, anemia, development of steatosis, and development of a Dieulafoy lesion (at the lesser curvature of stomach). It was not until the age of 44 that the patient was first diagnosed with Mallory-Weiss syndrome, and over the course of the next two years the patient was readmitted multiple times due to bouts of pancreatitis and Mallory-Weiss syndrome. Also of note, over the course of six years the patient was documented to have been administered an upper endoscopic examination a total of 11 times.

In this setting, while also taking into account the patient's history, it is highly plausible that hemorrhage is culpable for the iron deficient microcytic anemia (especially considering its severity). It was confounding that the first episode of hemorrhage manifested one and half weeks after admission. During the morning the patient began complaining of abdominal pain with diaphoresis quickly ensuing. Shortly afterwards, the patient collapsed without loss of consciousness, but no palpable pulse could be perceived. The patient was treated empirically with intravenous (IV) fluid resuscitation and 2 units of packed red blood cells. Upon stabilization the following morning the patient underwent a round examinations. Examinations per rectum and stool were unremarkable. But gastroscopy revealed MWTs of up to 3-4 cm in length just below the gastroesophageal junction in the area of the gastric cardia as the origin of bleeding (Fig. 1). During the gastroscopic examination the patient was administered vasopressor solution (NaCl & Epinephrine)

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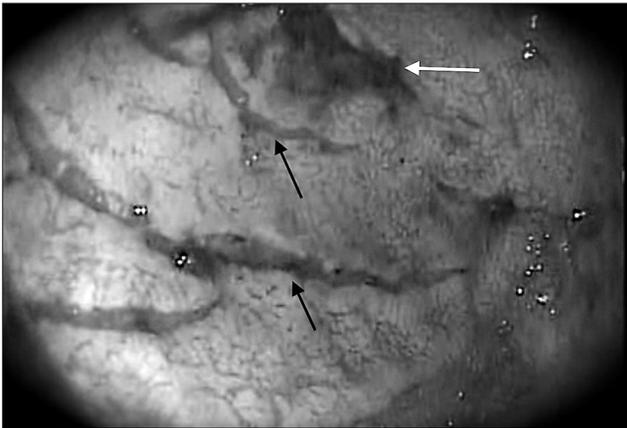


Figure 1. Linear Mallory-Weiss tears of the mucosa (black arrows). Site of fresh clot (white arrow).

in the areas adjacent to the MWTs. Later that same day, an even heavier episode of hemorrhage precipitated with stools consisting of dark blood mixed with fresh blood. A state of shock was validated by the presence of symptoms such as diaphoresis, severe hypotension (60/40 mmHg), and tachycardia (100/min). The patient was aggressively managed with IV fluid resuscitation, 1 unit of packed red blood cells, and Controloc (Pantoprazole). Overt signs (weakness, diaphoresis, hypotension (80/60 mmHg), and abdominal pain) consistent with a relapse of shock, developed again. Laboratory tests also demonstrated severe anemia with a hemoglobin level of 4.6 g/dL. Immediate colonoscopy and gastroscopy were performed. Colonoscopy revealed chyme mixed with blood in the cecum above the ileocecal valve, as well as in the ascending colon. Gastroscopy revealed the presence of a sliding esophageal hiatal hernia just inferior to the gastroesophageal junction, in addition to the MWTs previously diagnosed. Again, the patient was aggressively managed with IV fluid resuscitation and units of packed red blood cells. Over the course of the patient's hospitalization, varying regimens were used including No-Spa (Drotaverine), Polprazole, Cyclonamine, Tramadol, Nitrazepam, Lipancrea, Metoprolol, Ferrous Sulfate, Clemastine. The patient experienced remission of symptoms and generalized improvement and was able to be discharged. The patient was also counseled to cease consuming alcohol immediately and indefinitely.

DISCUSSION

Upper gastrointestinal (UGI) bleeding is generally classified into variceal bleeding (esophageal in 89% of cases and gastric in 11% of cases) and non-variceal bleeding (i.e. from peptic ulcers, erosive gastroduodenitis, gastroesophageal reflux, Dieulafoy lesion, MWS, etc.). The separation is based clinically on presentation, management, modes of treatment, etc. In both, cardinal signs of hematemesis or coffee ground vomiting are commonly present (in 68-85% of cases) as evidenced in our patient. Any patient presenting with these signs should be promptly administered an emergency UGI endoscopic examination (gold standard) [1,5,6,8]. Variceal bleeding most commonly originates in the distal esophagus and/or proximal stomach. Elevated pressure in the portal venous system (i.e. due to hepatic cirrhosis, Budd-Chiari syndrome) causes the dilation of veins [1]. It also commonly presents

with red nasogastric tube aspirate, and concomitantly with signs of cirrhosis. If UGI bleeding is suspected/determined to be from esophageal varices, empirical treatment with vasoactive agents (i.e. somatostatin, octreotide, terlipressin, etc.) should be commenced [8]. It is estimated that 15-20% of all UGI hemorrhages are due to varices [1]. Acute variceal hemorrhage carries a high risk of becoming fatal, with a 15-20% mortality rate [9]. Even though our patient was diagnosed with hepatic steatosis, which is widely known to precede liver cirrhosis, there were no esophageal varices present during any gastroscopic examinations. In contrast, non-variceal bleeding commonly presents with a history of ulcerogenic drug use (i.e. salicylates) or comorbid diseases such as dyspepsia, gastroesophageal reflux, hiatal hernias etc. It is generally managed with Proton-pump inhibitors and conservative/palliative treatment [8]. 90% of UGI hemorrhages resolve spontaneously with the residual 10% of cases requiring significant intervention, including: transfusion, endoscopic hemostasis (with hemoclips, injection of a solution of ethanol/polidocanol/epinephrine, electrocautery, or by banding, which is widely used in the case of esophageal varices) [1,7,10,11]. Our patient was among the unfortunate minority of cases who required significant interventional measures after each hemorrhagic episode.

Peptic ulcers (up to 44% of cases) are a frequent source of UGI hemorrhages, commonly located in the duodenum (16-30%) and stomach (20-30%) [1,6]. They both share a strong correlation to *H. Pylori* infection, implicated in approximately 95% of duodenal ulcers and 85% of gastric ulcers. The bacteria possesses virulence factors such as the CagA (encoded by cytotoxin-associated gene) and VacA (vacuolating cytotoxin) which facilitates in the colonization of the gastric mucosa, and subsequently seem to modulate the host's immune system [12]. Another important virulence factor is urease which allows for survival in the highly acidic gastric lumen via production of ammonia. It is also valuable as a non-invasive diagnostic measure (urease breath test) [12]. Peptic Ulcers are frequently associated with prandial-related abdominal pain concomitantly with weight gain or weight loss, depending on the timing of the pain.

Erosive diseases, such as erosive esophagitis and gastroduodenitis, are common etiologies of UGI bleeding, responsible for 5-10% and 20-30% of cases, respectively [1]. These diseases are often due to an incompetent lower esophageal sphincter, NSAIDs, and/or *H. Pylori* that allow acid to damage the mucosa of the UGI. With just 12 weeks of aspirin therapy, 47% patients may develop erosions due to the suppression of prostaglandin synthesis, which normally exert a gastrointestinal-protective effect [13,14]. Long-standing erosive esophagitis can lead to metaplasia, strictures, anorexia, Barrett's esophagus, and esophageal adenocarcinoma. This is why the disease commonly presents with progressive dysphagia, odynophagia, dyspepsia, abnormal tastes, and weight loss [1,15].

Dieulafoy lesion is a dilated aberrant submucosal vessel eroding into the mucosal surface in the absence of an ulcer [16]. Although originally speculated to be the result of an aneurysm or ischemia, the current consensus seems to be that it is caused by an abnormally large-caliber persistent tortuous submucosal artery [17]. Typically, it is located along the proximal portions of the lesser curvature and/or fundus of the stomach [16,17]. It is often the cause of recurrent hemorrhages occurring from a pinpoint non-

ulcerated arterial lesion, which often makes it difficult to discover and diagnose [17]

Mallory-Weiss syndrome has been found to be the cause of 5-10.3% of all (UGI) hemorrhage cases originating above the ligament of Treitz. MWS has been seen in a varying degree of conditions and diseases. It is most notoriously documented in connection with alcoholism. In 21-38% of MWS cases, alcohol abuse (with binges often preceding hemorrhagic episodes by several hours) has been reported. According to Clain et al., up to 38% of these episodes of hemorrhage are preceded by retching and vomiting [10,18]. On imbibing just 180 ml of alcohol after a standard meal a normal individual may experience impairment of esophageal propulsive motor activity and reduction of lower esophageal sphincter (LES) pressure [19]. Further investigations by Keshavarzian et al. found that acute ethanol increases esophageal contraction amplitude (ECA), while chronic ethanol consumption and withdrawal from ethanol increases ECA. This seems to be a compensatory mechanism in chronic alcoholics, leading to high-pressure esophageal contractions during withdrawal [20]. The higher intraesophageal/intragastric pressures generated are thought to be the main inciting factor in the development of MWS. There have been a significant number of cases that have linked salicylate ingestion to MWS, attributed to approximately 36% of cases of Mallory-Weiss syndrome [10]. MWS has even been implicated as an iatrogenic risk/complication from an esophageal endoscopy (incidence rate 0.07-0.49%) [3,21]. MWS has also occurred in those with eating disorders such as bulimia. This seems apparent as this condition is based on recurrent self-induced purging (i.e. vomiting) which commonly follow episodes of bingeing [1,4]. Less commonly, MWS has been attributed to hyperemesis of pregnancy, which generally occurs during the gestational period, but on rare occasions has occurred postnatally as a result of vomiting due to caudal epidural anesthesia [22]. MWS may be accompanied by a hiatal hernia (seen in approximately 7% of cases) [10]. Up to 10.7% of MWS patients can experience recurrent hemorrhagic episodes, often requiring greater subsequent transfusions, or rarely requiring surgical intervention [23]. The information presented suggests Mallory-Weiss syndrome is a highly plausible cause of recurrent upper gastrointestinal hemorrhage.

CONCLUSION

Our patient had a history littered with many diseases that are a frequent source of upper gastrointestinal hemorrhage. The hemorrhages that occurred during our patient's hospitalization, directly observed by gastroscopic examination, originated from the Mallory-Weiss tears and not from any other lesions present in our patient (i.e. duodenal ulcers, erosive esophagitis, gastroduodenitis, or Dieulafoy lesion). In patients suffering from very severe and chronic alcoholism, as witnessed in our case, it seems almost incontrovertible that there is a strong risk for developing recurrent hemorrhages in the course of Mallory-Weiss syndrome. We also believe that the number and extent of alcohol-related diseases developed by our patient,

superimposed with their rapid fruition (in just a few years), were major factors influencing the severe/intractable nature of the hemorrhages.

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