A 53-year-old woman presented to the emergency department with persistent vomiting followed by hematemesis. She reported heavy alcohol consumption on the previous day. Her blood pressure was 100/80 mmHg and her pulse rate was 131 beats/minute. The results of laboratory studies revealed a hemoglobin level of 8.0 g/dL.

On esophagogastroduodenoscopy (EGD), two mucosal lacerations measuring approximately 30 × 4 mm were identified at the gastroesophageal junction (GEJ) in the 3 o’clock and 11 o’clock positions. Endoscopic hemostasis was attempted using 13 mL dilute epinephrine (1:10000) and four hemoclips; however, bleeding persisted (Fig. 1). Therefore, transarterial angiography was performed, which revealed a pseudoaneurysm and extravasation of contrast from a branch of the left gastric artery. The gastric artery was selectively embolized with gelfoam and a microcoil (Fig. 2).

Over the next 72 hours, the patient received a total of 12 units of packed red blood cells, 18 units of platelets, and 3 units of fresh frozen plasma (FFP). The results of subsequent laboratory tests showed a platelet count of 14,000/mL, D-dimer level >20 μg/mL, fibrinogen level <60 mg/dL, and fibrin degradation products (FDPs) of 65.5 μg/mL. During the next 48 hours, she received an additional 18 units of platelets, 9 units of FFP, and 12 units of cryoprecipitate. On rechecking, her hemoglobin was 4.6 g/dL.

A further EGD was performed, which revealed a 20 × 5-mm oozing ulcer in the cardia (Fig. 3). Hemostasis was achieved with 6 mL injected epinephrine and 10 mL topical epinephrine (1:10000) sprayed onto the area. After 2 weeks, the patient was discharged without bleeding, and she is now under outpatient follow-up.

Mallory–Weiss tears are mucosal lacerations at the GEJ [1]. The combination of persistent vomiting and alcohol consumption is a well-established cause of Mallory–Weiss tears [2]. The management of these lesions is for the most part supportive [3]; however, in rare cases, fatal hemorrhage can result [4,5]. In this case, the patient’s severe bleeding was controlled using embolization after endoscopic treatment was unsuccessful. However, bleeding from an ulcer in the gastric cardia occurred 3 days after hemostasis had initially been achieved. It is possible that this ulcer was induced by ischemia secondary to inadequate collateral blood flow after embolization. In a previous study, ischemic ulcers occurred primarily in patients who had undergone a previous operation [6]. In this case, however, the ischemic ulcer with bleeding occurred without a previous operative history. The development of disseminated intravascular coagulation (DIC) thereafter was most likely due to the massive bleeding and subsequent transfusions.

**Fig. 1** Views during esophagogastroduodenoscopy (EGD) showing: a two mucosal lacerations measuring approximately 30 × 4 mm at the gastroesophageal junction in the 3 o’clock and 11 o’clock positions; b the appearance after endoscopic hemostasis had been achieved with injection of 13 mL dilute epinephrine (1:10000) and placement of four hemoclips.

**Fig. 2** Images from celiac angiography showing: a, b a pseudoaneurysm at the gastroesophageal junction and extravasation of contrast from a branch of the left gastric artery; c gelfoam and a microcoil (arrow) positioned to selectively embolize the gastric artery, leading to a diminishing of both the pseudoaneurysm and the extravasation of contrast (arrow head).
Competing interests: None

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Fig. 3 Repeat esophagogastroduodenoscopy (EGD) showing a 20 × 5-mm oozing ulcer in the gastric cardia.