A Mallory–Weiss tear treated with transarterial embolization complicated by disseminated intravascular coagulation

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 (1:10,000) 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 (1:10,000) 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.

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 (1:10,000) 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 (1:10,000) 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.
Competing interests: None

Youn Jung Choi, Moo In Park, Seun Ja Park, Won Moon, Sung Eun Kim, Hye Jung Kwon, Jae Hyun Kim, Seong Joo Kang
Department of Internal Medicine, Kosin University College of Medicine, Busan, Korea

References
5 Skok P. Fatal hemorrhage from a giant Mallory-Weiss tear. Endoscopy 2003; 35: 635

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

Bibliography
DOI http://dx.doi.org/10.1055/s-0034-1377366
Endoscopy 2015; 47: E247–E248
© Georg Thieme Verlag KG Stuttgart · New York
ISSN 0013-726X

Corresponding author
Moo In Park, MD
Department of Internal Medicine
Kosin University Gospel Hospital
34 Amnam-dong, Seo-gu
Busan 602-702
South Korea
Fax: +82-99-05055
mipark@ns.kosinmed.or.kr