

HSV-1 Ulcers as an Infrequent Cause of Hematochezia

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ABSTRACT

We report a 62-year-old woman in the intensive care unit who developed hematochezia. Her endoscopic findings revealed diffusely bleeding esophageal ulcers related to herpes simplex virus. The bleeding was treated successfully with Hemospray.

INTRODUCTION

Herpes simplex esophagitis is a well-known complication in immunosuppressed patients. Major symptoms include dysphagia, esophageal pain, and in severe cases, upper gastrointestinal (GI) bleeding. The endoscopic appearance of herpetic esophagitis is superficial punctate ulcerations. We report a case of herpes esophagitis causing hematochezia and successfully treated with Hemospray (Cook Medical, Winston-Salem, North Carolina). We find that herpes esophagitis may be a previously lesser-known cause of hematochezia.

CASE REPORT

A 62-year-old woman with a medical history of congestive heart failure, type 1 diabetes, and hypertension presented with progressive dyspnea and encephalopathy. Owing to acute respiratory failure, she was admitted to the intensive care unit and intubated for airway protection. She was positive for influenza A complicated by bacterial pneumonia. On hospital day 2, she developed acute respiratory distress syndrome and septic shock requiring broad-spectrum antibiotics and vasopressors. Trans-thoracic echocardiogram revealed an ejection fraction of 20%, concerning associated cardiogenic shock. An Impella device (Abiomed, Danvers, MA) was placed for cardiac support, extracorporeal membranous oxygenation was initiated for respiratory support, systemic heparin was started, and she was transferred to the cardiac intensive care unit. On hospital day 8, continuous renal replacement therapy was started because of anasarca and oliguria. Because hemodynamic status improved, the Impella support and vasopressors were slowly weaned off by hospital day 12.

In addition, on hospital day 12, the patient had 1.5 L of hematochezia from her rectal tube. Her hemoglobin level (Hb) was 6.4 g/dL (compared with 10.3 g/dL at the time of admission and 8.5 g/dL the day before). Owing to concerns regarding upper and lower GI bleeding, she was started on a pantoprazole drip, intravenous fluid resuscitation, and transfused packed red blood cells. Esophagogastroduodenoscopy (EGD) revealed numerous superficial punctate esophageal ulcers (3–4 mm) in the mid and distal portion with oozing blood and clotted blood in the gastric fundus (Figure 1). As likely, the hematochezia was due to the bleeding esophageal ulcers. The patient continued to have hematochezia, and a repeat EGD was done on hospital day 13 after receiving metoclopramide. It revealed numerous punctate, superficial, oozing esophageal ulcers and superficial, linear gastric ulcerations again (Figure 2). A biopsy was not performed on the esophageal ulcers because the patient was on heparin infusion, and Hemospray was applied to the oozing ulcers on the gastric fundus, body, and antrum with the cessation of bleeding (Figure 3). The patient's hematochezia stopped and Hb stabilized.

A few crusted vesicular lesions were noted on the patient's upper lip and lateral aspect of her tongue. Owing to the suspicion of herpes simplex virus (HSV) as the cause of esophageal and gastric ulcers, the patient was empirically started on IV acyclovir. A buccal ulceration was swabbed, and HSV polymerase chain reaction (PCR) was positive for HSV-1. HSV-1 antibody IgG was 4.97 index,

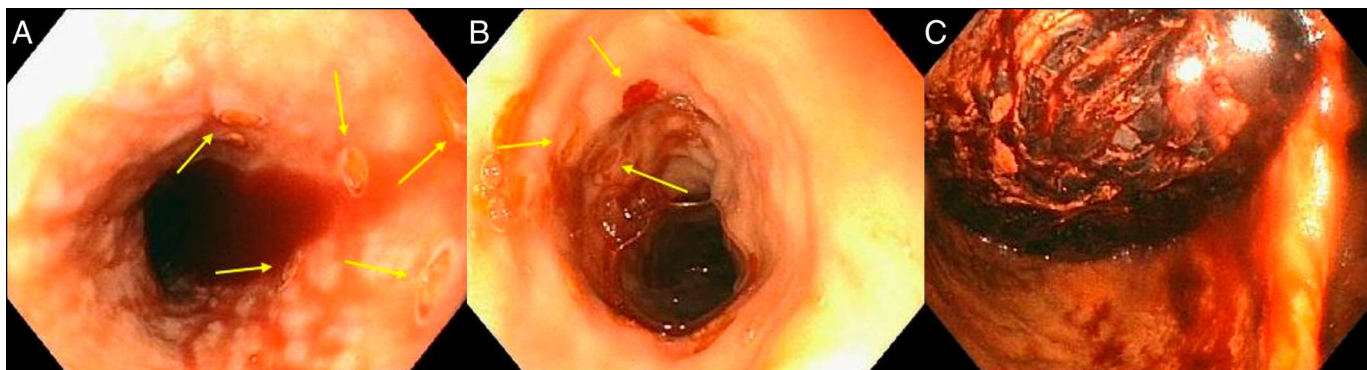


Figure 1. Esophagogastroduodenoscopy showing (A) ulcers (arrows) from the middle third of the esophagus, (B) punctate, oozing ulcers (arrows) from the lower third of the esophagus, and (C) clotted blood in the gastric body.

although HSV IgM immunoglobulin was not detected. In addition, *Helicobacter pylori* stool antigen and serologies were negative. The patient's hematochezia slowly stopped, and her blood counts stabilized within a few days. A few weeks later, after the patient finished acyclovir, a repeat EGD was performed revealing normal esophageal mucosa.

DISCUSSION

Herpes simplex virus type 1 (HSV-1) is known to cause vesicular lesions in the oral mucosa but can also cause disease in places including genitalia, the central nervous system, and the GI tract. Immunocompromised patients are especially at risk for HSV-1 extension from the oral mucosa to the oropharynx causing HSV esophagitis.¹

HSV esophagitis lesions are discrete, well-circumscribed ulcers with raised edges. They may also be punched-out or volcano-like in appearance. HSV may also be associated with erosive esophagitis.² The gold standard for diagnosis is histopathologic confirmation of HSV esophagitis.³ However, the virologic diagnosis of HSV-1 DNA by PCR has been shown as a more sensitive and rapid test.⁴ In our patient, because the esophageal ulcer was not biopsied, the confirmation of HSV-1 was performed through the buccal lesion via HSV PCR. HSV esophagitis is the result of the reactivation of HSV by extension of oropharyngeal infection into

the esophagus.⁵ Although the gold standard for oral HSV is through viral cultures, PCR for the detection of HSV is shown to have improved sensitivity and specificity compared with cultures.⁶ Intravenous acyclovir 5 mg/kg every 8 hours for 2 weeks should be initiated in immunocompromised patients with HSV esophagitis.⁷ In patients who do not respond to parenteral therapy because of likely acyclovir-resistant viral strains, foscarnet may be an option.⁸

GI bleeding from HSV esophagitis is an uncommon complication. A review of 53 patients by Canalejo et al reported that only 3 cases involved GI bleeding.⁹ The bleeding HSV esophageal ulcers in our patient were effectively treated with Hemospray. It is a relatively new endoscopic therapy to achieve hemostasis; it is a mineral powder that forms a barrier over the blood vessel wall and increases local clotting factors enhancing clot formation.¹⁰ Owing to its ability to cover large diffuse areas of bleeding, it has great utility in treating cancer-related GI bleeding, vascular ectasias (specifically gastric antral vascular ectasia), and hemorrhagic gastritis.¹⁰ Patients who need endoscopic therapy for GI bleeding on antithrombotic therapy present a challenge and Hemospray may emerge as an effective, safe option.^{11,12} Because the patient described above had diffuse esophageal and gastric bleeding while requiring heparin infusion (stopped only intermittently for procedures), she was an ideal candidate for Hemospray use.

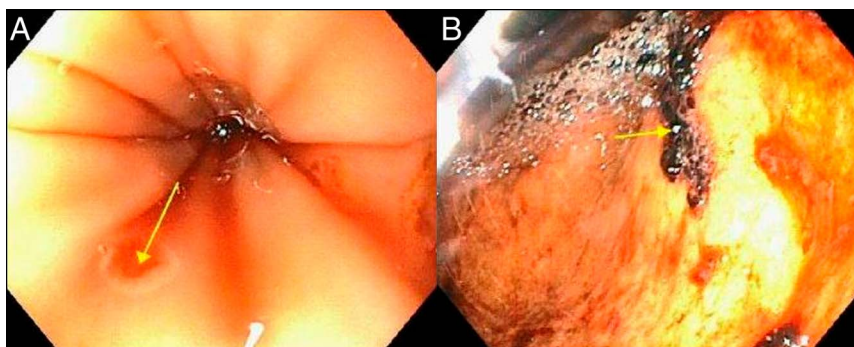


Figure 2. Esophagogastroduodenoscopy showing (A) esophageal ulcer concerning for HSV lesion (arrow) in the middle third of the esophagus and (B) gastric linear ulcer (arrow) in gastric fundus. HSV, herpes simplex virus.



Figure 3. Hemospray applied to the oozing ulcers on the gastric fundus and body.

It is important to consider HSV infection in the differential for immunocompromised patients in atypical cases of hematochezia. Although HSV infection is a rare cause of hematochezia, HSV esophagitis may be the cause in patients who have oral or buccal ulcerations concerning for HSV. Likely, the bleeding in our patient was exacerbated by a multisystem organ failure and anticoagulation with heparin. It is essential to manage these patients with supportive care, appropriate antiviral medications, and endoscopic treatment of the bleeding. In addition, the use of Hemospray may be a potential life-saving therapeutic option in these patients.

DISCLOSURES

Author contributions: Z. Ahmed wrote the manuscript and is the article guarantor. M. Narayanan and C. Hachem revised the manuscript for intellectual content and approved the final manuscript.

Financial disclosure: None to report.

Informed consent was obtained for this case report.

Received May 15, 2019; Accepted March 2, 2020

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