Discussion: Downhill esophageal varices cause less than 0.1% of all variceal bleeds. Occlusion of the SVC results in increased pressure to the azygous and hemiazygous systems responsible for drainage of the venous plexuses of the upper two thirds of the esophagus. If the occlusion does not interfere with azygous drainage to the SVC, only the upper two thirds of the esophagus will be involved, as in the present case. Occlusion of the azygous drainage into the SVC results in varices throughout the esophagus. Treatment of downhill varices focuses on restoring venous drainage. Options include balloon angioplasty, SVC stenting and open surgical therapies. Endoscopic options include proximal banding. Sclerotherapy is avoided due to concerns over embolization.

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Esophageal Mucosal Calcinosis Complicated by Upper GI Bleeding
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Calcium deposition in the gastrointestinal tract in the setting of hypercalcemia, while rare, is most commonly localized to gastric mucosa. We present, to our knowledge, the first reported case of esophageal mucosal calcium.

The patient is a 51 year old female with past medical history significant for chronic kidney disease secondary to lupus nephritis (on immunosuppression), coronary artery disease s/p stent placement, abdominal aortic aneurysm s/p repair, pericarditis, transfusion-related hepatitis C, recurrent deep venous thrombosis (DVT- on warfarin), primary hyperparathyroidism and osteoporosis who was initially admitted for acute right lower extremity (RLE) DVT and two large RLE hematomas despite a therapeutic INR, s/p IVC filter placement, as well as a lupus flare requiring rituximab therapy. During her hospital course, she developed epigastric pain, dysphagia (initially to solids and progressing to include liquids), and odynophagia. Physical exam was significant for epigastric tenderness. Pertinent labs included creatinine 2.14 mg/dL and calcium (corrected) 11.0 mg/dL. EGD revealed severe acute esophagitis with extensive necrotic exudate in the distal esophagus, characterized by white, plaque-like lesions, as well as erosive gastritis with minor oozing in the gastric antrum and body. In addition to a twice daily proton pump inhibitor (PPI), fluconazole was started for presumed candidal esophagitis. Pathology did not show evidence of fungal infection. Symptoms did not improve and repeat EGD was performed, demonstrating a white, circumferential, plaque-like lesion with areas of ulceration, necrotic exudates, inflammatory granulation tissue and focal small calcific deposits. Pertinent labs included phosphorous 3.3 mg/dL, intact PTH 187 pg/mL, Vitamin D 25-OH 14. Fluconazole was discontinued. A few days later, the patient had massive hematemesis and rectal bleeding with hemoglobin 4.1 g/dL, tachycardia, and hypotension in the setting of a supratherapeutic INR. Emergent EGD revealed active arterial bleeding in the distal esophagus, stopped with epinephrine injection and two hemoclips. Interventional radiology then embolized a left gastric artery branch supplying the corresponding area of bleeding. Neck imaging failed to show evidence of a parathyroid adenoma. The patient had no further bleeding, her symptoms improved on the PPI and she was discharged home on calcium and vitamin D supplementation. She planned to follow-up with endocrine surgery for parathyroidectomy evaluation.

This is a unique case of hypercalcemia from untreated primary hyperparathyroidism causing esophageal calcium deposition, ultimately leading to inflammation, necrosis, and bleeding.