Black Esophagus with Superimposed Candida in a Patient with B Cell Lymphoma: A Case Study.

Christopher Lenivy DO
Lehigh Valley Health Network, Christopher.Lenivy@lvhn.org

Patrick Hickey DO
Lehigh Valley Health Network, Patrick.Hickey@lvhn.org

Adam Peyton DO
Lehigh Valley Health Network, Adam_L.Peyton@lvhn.org

Follow this and additional works at: https://scholarlyworks.lvhn.org/medicine

Part of the Gastroenterology Commons, Internal Medicine Commons, and the Medical Sciences Commons

Published In/Presented At

This Poster is brought to you for free and open access by LVHN Scholarly Works. It has been accepted for inclusion in LVHN Scholarly Works by an authorized administrator. For more information, please contact LibraryServices@lvhn.org.
Black esophagus, also known as acute esophageal necrosis (AEN), is an uncommon condition associated with a variety of critical illnesses. The pathophysiology is poorly understood, but most clinicians suggest a multifactorial etiology centered around an ischemic insult. Though AEN itself does not often lead to further complications, the overall prognosis for these patients is typically poor.

A 68 year-old male presented to the emergency department with severe back pain and persistent vomiting. He had a history of diffuse large B cell lymphoma with response to chemotherapy. After 8 years of remission, the patient had a relapse confirmed by excisional biopsy of a supraclavicular node. Three days prior to the patient’s presentation he had finished the second infusion of chemotherapy. The patient stated that since his chemotherapy session he vomited so many times at home he lost count. He complained of severe mid-thoracic back pain and only mild hoarseness and throat discomfort. Physical exam was unremarkable. CT scan of the chest showed diffuse mural thickening of the esophagus.

On the first day of admission, the patient developed rapidly progressing dysphagia and odynophagia. Endoscopic exploration revealed blackened mucosa with circumferential involvement of the middle and distal thirds of the esophagus. This finding did not extend beyond the esophagogastric junction. A clean-based ulcer was also identified in the body of the stomach. Biopsies confirmed extensive necrosis along with abundant fungal organisms present, morphologically consistent with Candida. The patient was treated with systemic fluconazole, sucralfate, and a proton pump inhibitor. After five days of treatment, the patient’s odynophagia improved significantly and he was restarted on a regular diet.

The discovery of black esophagus is a unique finding in a patient complaining of odynophagia and/or dysphagia. The patient exhibited two severe risk factors for AEN, and despite this fact his status improved rapidly upon addition of systemic antifungals and PPI therapy. The mortality rate in these patients is high (13-35%) and usually linked to the underlying disease. Although treatment is largely supportive, the importance of early and accurate diagnosis is of paramount importance.