Black Esophagus: Diagnostic Associations and Management Strategies

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Dear Editor,

It is with great interest that I read an article by Dr. Uyar et al.[1] on the black esophagus (BE) in the latest issue of your journal. The authors describe an interesting case of acute esophageal necrosis (AEN) in a patient with diabetes mellitus, hypertension, and polyarteritis nodosa.

Black esophagus or AEN is a rare clinical syndrome with a classic endoscopic appearance of black discoloration in the distal esophagus with various proximal extensions and abrupt transitions at the gastroesophageal junction. Upper gastrointestinal hemorrhage is the main presenting symptom and a typical patient is an elderly male with multiple comorbidities. Its etiology is multifactorial, a combination of acute ischemic injury, corrosive effects of gastric reflux on the esophageal mucosa, and impaired local esophageal mucosal defenses.

Dr. Uyar briefly mentions the diagnosis of diabetic ketoacidosis (DKA) in the patient prior to developing BE but unfortunately omits it in the discussion section. It is, however, important to note an association of DKA with BE. Previous studies have shown diabetes mellitus to be a risk factor in developing AEN,[2,3] possibly owing to the underlying vasculopathy, coagulability, element of gastric paresis, and increased tissue susceptibility to the injury. Ketoacidosis results in tissue hypoperfusion and cellular damage and may lead to significant multi-organ impairment.[4] In fact, BE has been recently observed in 14% of inpatients with DKA.[5] Increased clinical awareness and prompt endoscopic utilization in setting upper gastrointestinal hemorrhage will likely increase this figure substantially. Furthermore, DKA plays an important prognostic role in patients with BE. The authors mention high early literature mortality rates, but it should be clarified that the development of AEN typically carries a poor prognosis with close to 30% of patients succumbing to the underlying critical illness. However, mortality specific to BE seems to be much lower, at 6%, and known risk factors include esophageal perforation, DKA, and diseases of the immune system compromise.[2,3,6]

Treatment of BE is aimed at hemodynamic resuscitation, nil-per-os restriction, aggressive anti-acid therapy with intravenous proton-pump inhibitors, and management of co-existing medical conditions. The angiographic evaluation may not be helpful but can lead to renal demise in critically ill patients. Long-term complications related to BE are unusual but may include esophageal stricture or stenosis in nearly 10% of the patients, which could be addressed with endoscopic dilatation. Interestingly, it seems that its development is inversely related to the presence of diabetes mellitus – likely a reflection of deficiency in the effective reparative process.[2] Finally, esophageal candidiasis in the setting of BE has been previously described[2] and may be related to underlying DKA. However, in the case presented by Dr. Uyar, it could have been also precipitated by recent steroid use.

The striking endoscopic appearance of BE continues to attract attention in today's medicine, and future reports and studies will aid in a better understanding of this syndrome.

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