

Acute esophageal necrosis syndrome

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Published online: 11 October 2011
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Editor,

I read with great interest the Case Series by Singh et al. on acute esophageal necrosis (AEN) [1]. The authors describe a retrospective series of cases of AEN recorded over a 3-year period. Black esophagus or AEN is a rare entity characterized by a diffuse circumferential black-appearing mucosa extending proximally from the gastroesophageal junction and may involve the length of the entire esophagus. AEN is seen in the setting of hemodynamic or vascular compromise, overflow with corrosive gastric contents, and decreased esophageal mucosal defense mechanisms. An important endoscopic finding is the presence of duodenal pathology, commonly ulcerations in over 25% of cases [2]. This may result in gastric outlet obstruction which, in turn may cause direct injury to the esophagus as well as potentiate the initial vascular hypoperfusion insult to the distal esophageal mucosa. It would be, therefore, important to know about endoscopic findings in the duodenum in the case series.

A recent case report described black esophagus in a patient with alcohol abuse and achalasia [3]. It would therefore be of interest to find out how the second patient in Singh et al's series was diagnosed with achalasia.

The authors mention that "reflux esophagitis" was noted on endoscopy in two patients. Severe gastroesophageal reflux, in a correct clinical setting, may lead to the

development of black esophagus. However, the classic appearance of the AEN heavily outweighs endoscopic description of the reflux esophagitis.

Response from the authors

We thank Dr Gurvits for his comments [1].

All the patients with AEN had black esophagus on endoscopy. There were no patients with black duodenum or any changes suggestive of ischemia involving the duodenum, except for one patient with duodenitis.

Achalasia was assumptive diagnosis, since the secretions were pooled in the esophagus and the scope did not traverse the lower esophageal sphincter (LES) on first endoscopy. On repeat endoscopy few days later, the scope traversed with a "pop" and patient responded well to botulium injection to the LES. The authors feel that black esophagus may cause achalasia by damaging the LES with resultant tightening of the LES; however, this is merely a hypothesis.

Reflux esophagitis was seen on subsequent endoscopies in 2 patients. The resolution of black esophagus may heal slowly (evolving from black to mild esophagitis to complete resolution). The stage of the disease picked up on endoscopy merely depends on the timing of the EGD.

The author has no commercial associations that might be a conflict of interest in relation to the submitted manuscript.

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References

1. Singh D, Singh R, Laya AS. Acute esophageal necrosis: a case series of five patients presenting with “Black esophagus”. *Indian J Gastroenterol.* 2011;30:41–5.
2. Gurvits GE, Shapsis A, Lau N, Gualtieri N, Robilotti JG. Acute esophageal necrosis: a rare syndrome. *J Gastroenterol.* 2007;42:29–38.
3. Lee JK, Bhargava V, Mittal RK, Ghosh P. Achalasia, alcohol-stasis, and acute necrotizing esophagitis: connecting the dots. *Dig Dis Sci.* 2011;56:612–4.