Atypical gastric ulcer with characteristic eschar appearance

Mohit Girotra, Sudhir K Dutta, H Jeffrey Schwartz

Abstract:
Cocaine is a known potent vasoconstrictor known to cause various complications ranging from nasal septum perforations to myocardial ischemia. Many gastrointestinal effects of cocaine are reported, including bowel ischemia and gangrene. The knowledge on endoscopic appearance of cocaine-induced gastric ulcers is limited, mainly due to presentation of patients with frank perforations. We report a case of 48 year old male, a non-smoker but chronic substance abuser, who presented with abdominal pain, mainly epigastric with radiation to the back. Abdominal CT scan was normal, and endoscopy showing a single chronic non-bleeding ulcer at the incisura. The ulcer due to large sized, round in shape with irregular borders and thick eschar appearance is characteristic of cocaine-induced ulcer. It is important for physicians to remain cognisant of gastrointestinal complications of cocaine, recognise these ulcers endoscopically and prevent perforations in these subsets of patients.

Case report
A 48-year-old African-American male, with no significant medical history but long-standing cocaine abuse, presented with abdominal pain. Pain was of 1-week duration, constant and sharp, localised to epigastrium, moderate in intensity and radiated to the back. The patient was also bothered by sour taste in his mouth and some nausea. He also had single episode of coffee ground emesis and dark coloured stools. He denied any alcohol or smoking but was increasingly using cocaine in the last 9 months. There was no previous history suggestive of peptic ulcer disease or non-steroidal anti-inflammatory medication intake.

Initial labwork including complete blood count, renal and liver function tests were unremarkable, except positive stool haemoccult. Electrocardiogram and troponins ruled out myocardial event and chest X-ray did not suggest any pulmonary process. Abdominal CT was normal. Upper endoscopy revealed a single chronic non-bleeding ulcer at the incisura, with an adherent clot which could not be washed away.

The ulcer was large (2.5-cm diameter), round with irregular borders and thick black eschar rim (Figure1). Biopsy from the ulcer returned as chronic active gastritis but negative for *Helicobacter pylori*. The patient, since not perforated or actively bleeding, was not considered a surgical candidate and was managed medically with proton pump inhibitors. His repeat endoscopy after 4 weeks showed improvement in ulcer.
Discussion

The image shows a large 2.5-cm ulcer, round and irregular borders, with thick black rim of eschar like tissue. This atypical appearance can be considered characteristic of cocaine-induced ulcer, especially in the background of absence of other ulcer forming risk factors.

Cocaine is a known potent vasoconstrictor and many gastrointestinal effects of cocaine are reported, including bowel ischemia and gangrene. There is impressive literature on cocaine-induced duodenal ulcers, described as large pre-pyloric ulcers, which present usually with perforation and undergo patch closure\textsuperscript{1,2}. The knowledge
on endoscopic appearance of cocaine-induced gastric ulcers is limited, mainly due to presentation of patients with frank perforations. This is however, a unique description of cocaine-induced ulcer in gastric region based on authors’ multiple experiences with similar patients.

The black eschar is formed by tissue necrosis caused by vasoconstrictive effects of cocaine. Hence, it is most advantageous for clinicians to remain cognisant of the gastrointestinal complications of cocaine and identify them at ulcer stage with their characteristic eschar appearance, to prevent further and more dreaded complications like perforation and massive haemorrhage. Anti-ulcer and anti *Helicobacter pylori* therapies do work, but abstinence from cocaine is the mainstay of therapy.

**Competing interests:** None known.

**Author information:** Mohit Girotra, MD, Department of Internal Medicine, The Johns Hopkins University/Sinai Hospital Program in Internal Medicine, Baltimore, Maryland, USA; Sudhir K Dutta, MD, FACG, Professor of Medicine, University of Maryland School of Medicine, Division of Gastroenterology, Sinai Hospital of Baltimore, Maryland, USA; H Jeffrey Schwartz, MD, Consultant Gastroenterologist, Division of Gastroenterology, Sinai Hospital of Baltimore, Maryland, USA

**Correspondence:** Mohit Girotra, MD, Johns Hopkins University/Sinai Hospital Program in Internal Medicine, 2401 W. Belvedere Ave, Baltimore, MD 21215, USA; email: mgirotr1@jhmi.edu

**References:**
