A case of gastric ischemic necrosis

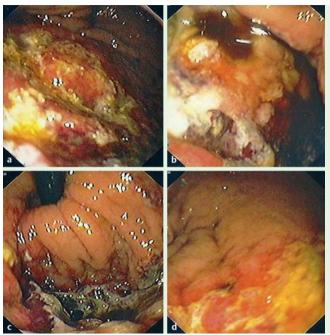


Fig. 1 Upper-gastrointestinal endoscopy. a, b A hemorrhagiculcerative lesion which involves almost the entire major curvature of the stomach. Note the dark gastric mucosa with hemorrhagic and necrotic tissue due to ischemia. c, d The cardia and the antrum are unaffected.

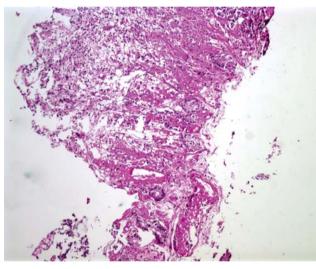


Fig. 2 An overview of the necrotic gastric mucosa, with infarction-like features. (Hematoxylin and eosin stain; original magnification × 4.)

Gastric ischemic necrosis (GIN) is a rare condition, usually seen postoperatively or after therapeutic embolizations [1]; in the absence of such surgical interventions, diagnosis can be delayed, a delay that can be fatal [2]. Patients are treated conservatively unless signs of perforation or sepsis develop,in which casegastrectomy is warranted [3].

We report a case of GIN in a patient without prior surgical or other invasive interventions. A 78-year-old man with a history of diabetes mellitus, hypertension, and atrial fibrillation presented with coffee-ground emesis and epigastric pain. He was in severe distress with a heart rate of 82 beats per minute, arterial blood pressure of 90/50 mm Hg, and a respiratory rate of 20 breaths per minute. His abdomen was tender with involuntary guarding and decreased bowel sounds. Pathological tests included an increased white blood cell (WBC) count (11950/mm³), decreased hemoglobin (10.6 g/dL), elevated blood urea nitrogen (59.8 mg/dL), and hypoalbuminemia (2.7 g/dL). Radiographs showed no subdiaphragmatic free air. Abdominal computed tomography (CT) revealed thickened gastric folds; the major abdominal vessels had atherosclerotic lesions but no occlusion. Esophagogastroduodenoscopy revealed a hemorrhagiculcerative lesion of the major curvature (**p** Fig. 1).

Biopsies showed granulomatous interstitial nephritis (GIN) (**Fig. 2**).

The patient was treated with intravenous fluids and broad-spectrum antibiotics and was discharged after 12 days in good condition.

This case highlights a challenging entity, GIN, suggesting the importance of endoscopy in its prompt diagnosis. Although the patient had risk factors for gastric hypoperfusion (diabetes, hypertension, atrial fibrillation), he reported no abdominal interventions, and CT scans showed no vascular thrombosis or occlusion (although atherosclerosis and hypotension were probably responsible). This drew differential diagnosis away from GIN until endoscopy provided the diagnosis.

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