

Esophageal and Gastroduodenal Hemorrhagic Necrosis: A Unique Finding in the Setting of Septic Shock and Vasopressor Use

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ABSTRACT

Esophageal and gastroduodenal necrosis are rare conditions with poor prognosis. We describe a case that was diagnosed with upper endoscopy in the setting of severe septic shock. To our knowledge, this is the first case in which esophageal and gastroduodenal necrosis occurred simultaneously in this setting. We discuss the pathophysiology, diagnostic approach, and treatment options of this rare entity.

INTRODUCTION

Esophageal, gastric, and duodenal necrosis are extremely rare conditions. They have been described as complications of congenital and acquired conditions such as systemic hypotension, volvulus, thrombosis, embolism, and vasculitis. Septic shock and vasopressor use can elicit profound changes in the splanchnic circulation. Symptoms and laboratory tests are non-specific, and diagnosis can be made on radiology or endoscopy findings. Management includes surgery, and mortality rates are high.¹

CASE REPORT

A 42-year-old man with a history of chronic alcoholism presented with severe shock of unknown etiology. On admission, the patient was febrile (tympanic temperature 38°C), and he was sedated with propofol under invasive mechanical ventilation. Blood pressure was 60/40 mm Hg, and his heart rate was 120 beats/min under vasopressor and inotropic support (noradrenaline 0.5 µg/kg/min and dobutamine 5 µg/kg/min, respectively). On physical exam, a systolic heart murmur was evident on the left sternal border, and the abdomen was soft and had no tenderness to deep palpation. Laboratory findings were significant for leukocytosis (leukocytes 13,100/µL), anemia (hemoglobin 11.2 g/dL, mean corpuscular volume 88.2 fL), prolonged international normalized ratio 5.86, high prothrombin time 71.9 s, acute kidney injury (creatinine 2.3 mg/dL, urea 124 mg/dL), elevated aminotransferases (GPT 2822 U/L, GOT 5068 U/L), hyperbilirubinemia (4.8 mg/dL), and elevated lactate dehydrogenase 4,245 U/L, pro-calcitonin (4.47 ng/ml), N-terminal pro b-type natriuretic peptide (8,018 pg/mL), and troponin (1.10 ng/mL). His arterial blood gas test showed metabolic acidemia and high lactate levels (14 mmol/L).

An urgent transthoracic echocardiogram revealed left ventricle hypertrophy with a moderate-to-severe depression of his global systolic function, severe auricular dilatation with a thrombus in his left auricular appendix, and native aortic valve vegetation. The diagnosis of a severe septic shock with multiple-organ dysfunction secondary to endocarditis was made, and the patient was started on empirical broad-spectrum antibiotics. He

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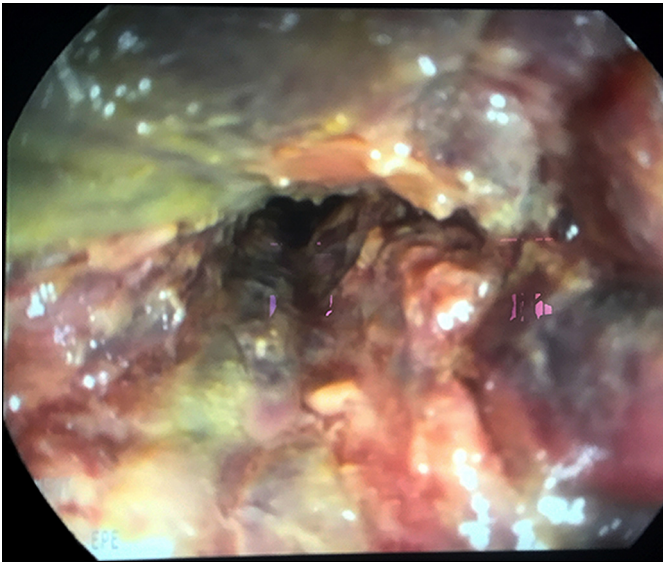


Figure 1. Upper endoscopy showing a circumferential hemorrhagic mucosa, fibropurulent plaques, and violaceous areas of elevated mucosa in the lower third of the esophagus.

was immediately transferred to the intensive care unit of our hospital. His drug abuse history was unknown. Additional investigation was negative for secondary causes of immunosuppression and syphilis. Blood cultures were positive for *Corynebacterium jeikeium*.

Despite initial clinical improvement 9 days after admission, severe hypotension developed and vasopressor support was re-introduced (noradrenaline 1.9 $\mu\text{g}/\text{kg}/\text{min}$). Within 24 hours, blood was seen on the nasogastric tube. An urgent upper endoscopy showed a circumferential hemorrhagic mucosa, fibropurulent plaques, and violaceous areas of elevated mucosa in the lower third of the esophagus (Figure 1). Mucosa of the gastric fundus, body, antrum, and duodenum exhibited loss of vascular pattern, areas of pale, hemorrhagic and violaceous mucosa, multiple erosions, and fibropurulent plaques (Figure 2). Biopsies were consistent with extensive hemorrhagic necrosis (Figure 3). Laboratory findings revealed acute

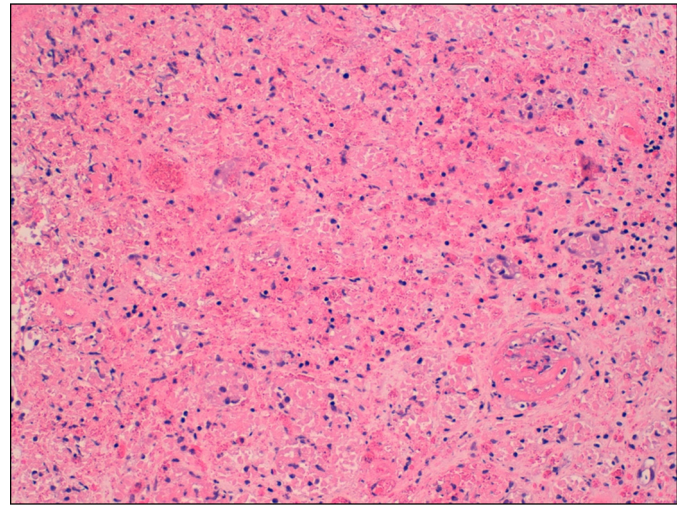


Figure 3. Hematoxylin and eosin stain of the antrum biopsy showing extensive hemorrhagic necrosis.

kidney failure and probable ischemic pancreatitis and hepatitis. The contrast-enhanced abdominal computed tomography performed 2 days before did not show any alterations suggestive of intestinal ischemia or alterations suggestive of chronic liver disease. The patient eventually died hours later due to refractory multiple-organ dysfunction.

DISCUSSION

The gastric blood supply is derived from the common hepatic, left gastric, and splenic arteries arising from the coeliac trunk. The coeliac trunk also provides blood supply to the lower third of the esophagus through the left gastric artery and to the duodenum through the pancreaticoduodenal artery.

Esophageal necrosis is rare, and gastric necrosis is even rarer due to the absence of end arteries and the rich network of collaterals. However, in the setting of septic shock, profound hemodynamic changes can occur and render the gut more susceptible to ischemia. Severe and disproportionate vasoconstriction can lead to a loss of almost 90% of the total

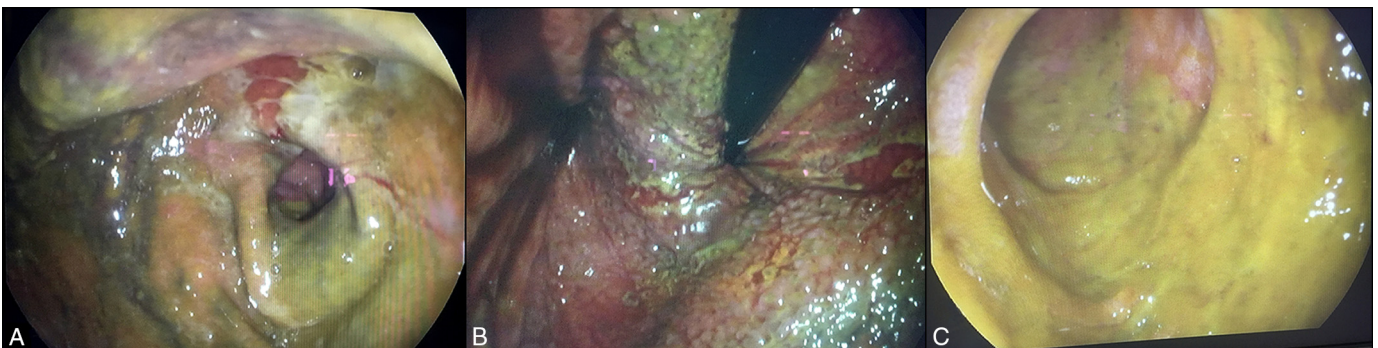


Figure 2. Upper endoscopy showing loss of vascular pattern, areas of pale, hemorrhagic and violaceous mucosa, multiple erosions, and fibropurulent plaques in the (A) antrum, (B) fundus, and (C) duodenum.

gastric blood flow.² Myocardial dysfunction, hypoxia, and hypovolemia also seem to contribute to further reduce blood supply. In addition, supportive measures for septic shock, such as mechanical ventilation and vasoactive agents, can further exacerbate these changes. The latter act by shifting the blood from the splanchnic system into systemic circulation due to pronounced effects on both α_1 and α_2 receptors.¹ In our case, the concomitant finding of acute kidney failure, probable ischemic pancreatitis, and hepatitis suggest hypovolemia as a main cause. It is possible that other organs supplied by the splanchnic system were also affected.

These events seem to be under-reported in the literature; in addition, considering the frequency of septic shock, these events are most likely underdiagnosed. In fact, symptoms of acute esophageal or gastroduodenal necrosis such as nausea, dysphagia, vomiting, gastrointestinal bleeding, and abdominal distension can be a problem in the critical care patient due to deep sedation. In suspicious cases, endoscopy could provide an early and probably safe diagnosis with a proper assessment of the extent and severity of ischemia, and, in an appropriate setting, endoscopy could help guide surgical plans. Suggestive endoscopic findings are areas of pale mucosa, loss of mucosal vascular pattern, friable mucosa, erosions, ulceration, and the presence of adherent fibropurulent exudates as seen in our case.²⁻⁴ In extreme cases, the esophagus can have a striking circumferential black appearance.⁵ Imaging of the abdomen can also suggest the diagnosis. A computed tomography scan can show a non-enhancing thickened stomach wall and intramural gas with eventual complications.^{1,4}

There is no specific treatment approach. Supportive measures include adequate fluid resuscitation, nasogastric intubation and suction, gastric acid suppression with high-dose intravenous proton pump inhibitors, none per oral, parenteral nutrition, and broad-spectrum antibiotics.^{2,3} In cases of severe

necrosis or complications such as perforation, surgery is recommended with a surgical consultation and multidisciplinary management.² Our patient's clinical condition was so severe that, after multidisciplinary consultation, only supportive measures were pursued.

In the setting of intensive care, the pathological insult for ischemia is probably multifactorial. In case of suspicion, an early diagnosis with endoscopy and early supportive measures could lead to improvement of the patient outcome. There is still a poor prognosis overall.

DISCLOSURES

Author contributions: J. Carvão and V. Magno Pereira wrote the manuscript. M. G. Faria reviewed the literature and collected the images. C. Sousa Andrade, N. Fernandes, L. Jasmins, and J. J. Nóbrega edited the manuscript. J. Carvão is the article guarantor.

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