

## Upper Gastrointestinal Bleeding in a Patient with Liver Cirrhosis: Gastroesophageal Varices Are not Always to Blame

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A 45-year-old male with active alcoholism and liver cirrhosis was brought to the emergency room with hypovolemic shock in the context of diarrhea and melena. He presented hemodynamic instability (blood pressure: 33/22 mmHg, heart rate: 120-130 bpm), hyperlactatemia (lactate level: 20 mmol/L) and anemia (hemoglobin: 5.8 g/dL). Aggressive supportive therapy with vasopressor support, octreotide, antibiotic prophylaxis, packed red blood cell transfusion and intra-venous proton pump inhibitor was started. Upper digestive endoscopy revealed diffuse circumferential black discoloration of the middle and distal esophagus with areas of linear ulceration and mucosal sloughing, consistent with acute esophageal necrosis (AEN) (Fig. 1). There were blood clots and bright blood in the lumen but no apparent active bleeding. There was no evidence of esophageal varices. Gastric and duodenal mucosa were normal. Abdominal computed tomography scan revealed concomitant extensive bowel ischemia involving the small (Fig. 2) and large bowel (Fig. 3). Despite supportive measures, the disease had a fulminant evolution and the patient died after a few hours.

Acute esophageal necrosis is defined endoscopically by diffuse and circumferential black mucosal discoloration of the distal esophagus with abrupt transition at gastroesophageal junction and variable proximal extension. It is more common in older males with general debilitation and multiple comorbidities and typically presents with hematemesis or melena. Pathophysiology involves esophageal ischemia, gastro-esophageal reflux and debilitated physical states [1]. An association with liver cirrhosis is well established, malnutrition decreasing esophageal mucosal defense and impairing

regenerative ability [2]. This case demonstrates that, although gastroesophageal varices and peptic ulcer bleeding are the most common sources of gastrointestinal bleeding in cirrhotic patients [3], AEN must also be considered, particularly in the setting of hemodynamic instability.

To our knowledge there are no previous cases of liver cirrhosis in association with extensive ischemia of the small and large bowel, probably reflecting severe hypoperfusion secondary to hypovolemic shock. It is possible that splanchnic vasoconstriction secondary to vasoactive therapy could be another factor involved in intestinal ischemia. Therefore, the benefit of these agents must be balanced with the risk of ischemic insult in hemodynamically unstable patients and immediately discontinued if there is any evidence of ischemia.

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**Conflicts of interest:** None to declare.

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