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IMAGE | STOMACH

Necrotic Gastric Gangrene as Complication of Congestive Portal Venopathy from Portal Vein Thrombosis

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CASE REPORT

A 58-year-old male with past medical history of chronic alcoholism, hepatitis C, and systolic heart failure with low ejection fraction presented to the emergency room with multiple episodes of hematemesis for 24 hours prior to the presentation. On arrival, he was hypotensive and tachycardic with impaired mentation. Laboratory findings revealed anemia (hemoglobin 9.4 gm/dL), thrombocytopenia (platelets of 103,000/mm^3), acute renal failure, and lactic acidosis. The patient was appropriately resuscitated. An emergent esophagogastroduodenoscopy revealed normal esophagus with no esophageal or gastric varices but was noted to have generalized exudative necrosis and edematous mucosa in the body of the stomach and necrotic gastric mucosa in the antrum, appearance suggestive of severe gastric necrosis (Figures 1 and 2). During the procedure, patient developed cardiac arrest, and subsequently cardiopulmonary resuscitation was performed with return of spontaneous circulation after the first epinephrine injection. During the stay in intensive care unit, patient underwent vascular imaging studies, including CT angiogram of the abdomen and Doppler ultrasonography of the abdomen, which identified a large portal vein thrombosis with evidence of portal hypertension and signs of gastric necrosis. Patient was managed conservatively with nutritional support and antibiotics in intensive care unit. Subsequently he was discharged home with resolution of symptoms on follow-up appointment.

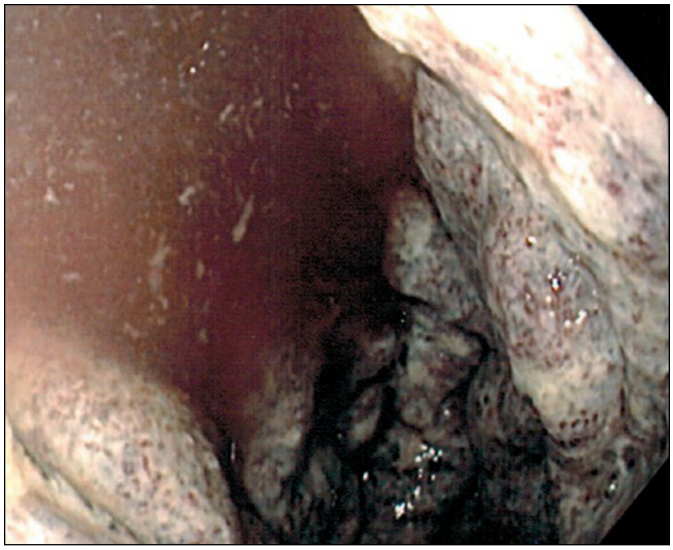

Figure 1. Endoscopic view of the body the stomach showing edematous gastric folds with gastric necrosis.

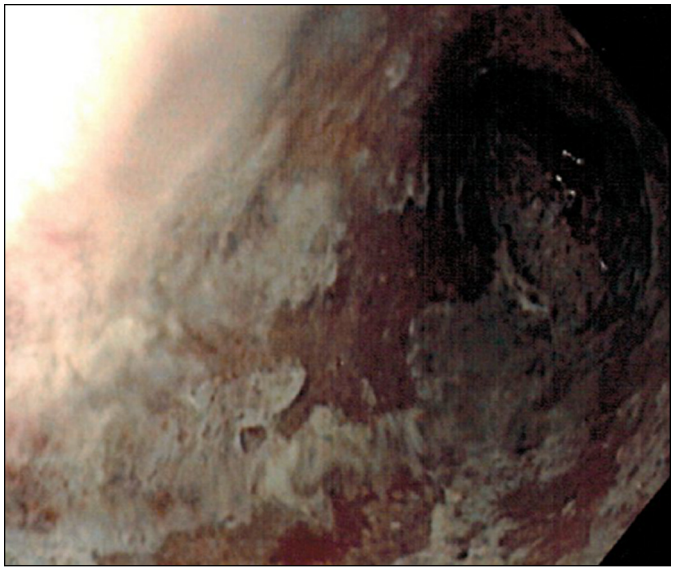

Figure 2. Endoscopic view of antrum showing necrotic exudative gastric mucosa.

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Gastric gangrene is an extremely rare clinical entity, as the stomach has a rich vascular supply from right gastric, left gastric, right and left gastroepiploic, and short gastric, as well as multiple collaterals. Arterial thromboembolism, herniation, volvulus, acute gastric dilatation, bulimia nervosa, trauma, and exposure to caustic materials are listed as possible etiologies for gastric gangrene in the available published literature.[1,2] In 1951, Cohen described that even complete de-arterialization of the stomach in both animals and humans does not result in gastric necrosis, as stomach has rich collateral arterial supply. Interestingly, he postulated complete venous flow disruption to the stomach as pathogenesis of the gastric gangrene.[3] Our patient had severe systolic congestive heart failure and was found to have portal vein thrombosis, which further contributed and worsened back flow venous congestion subsequently resulted in necrotic exudative gastric gangrene. We believe that phenomenon of venous flow disruption compared to arterial thromboembolism contributes more in pathogenesis of gastric gangrene. Venous flow disruption in stomach can lead to congestive gastropathy, which can get infected and leads to dreaded complication like necrotic exudative gastric gangrene.

DISCLOSURES

Author contributions: K. Changela, T. Sunkara, and A. Samarghandi wrote the manuscript, reviewed the literature, and acquired the images. A. N. Culliford wrote and edited the manuscript. T. Sunkara is the article guarantor.

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Informed consent was obtained for this case report.

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