

An Unusual Distal Esophageal Finding: An Endoscopic Challenge

Jonatan Merino-Soriano¹  · José Luis Gálvez Romero²  · Andrea Báez Núñez³  · Sayuri Chong Hernández⁴  · Francisco de Jesús García-Mendoza⁵ 

¹Department of Internal Medicine. Hospital Regional de Puebla, Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado.

²Department of Research. Hospital Regional de Puebla, Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado.

³Department of Nephrology. Hospital Regional de Puebla, Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado.

⁴Department of Internal Medicine. Hospital Regional de Puebla, Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado.

⁵Central Research Laboratory. Faculty of Medicine, Benemérita Universidad Autónoma de Puebla, Puebla, México.

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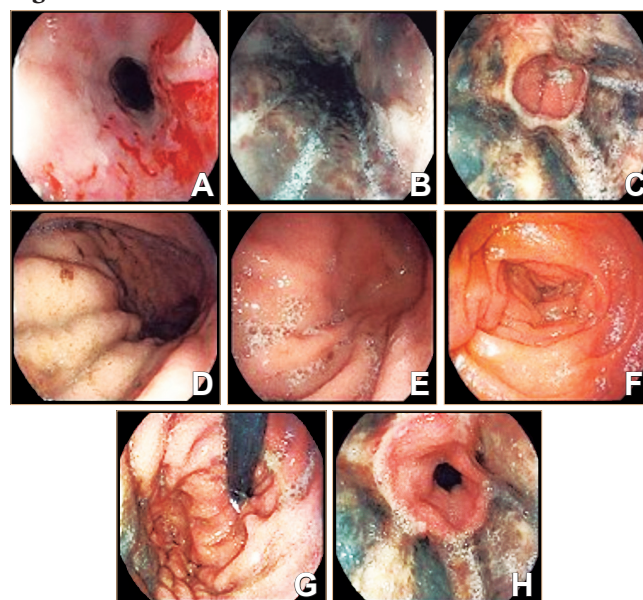
Image presentation

A 66-year-old woman with a history of type 2 diabetes mellitus, hypertension, and advanced chronic kidney disease on peritoneal dialysis was admitted with peritoneal dialysis-associated peritonitis. During hospitalization, she developed intolerance to oral intake, progressive dysphagia, epigastric pain, and coffee-ground emesis. Laboratory evaluation revealed moderate normocytic normochromic anemia, leukocytosis with neutrophilia, hypoalbuminemia (2.1 g/dL), and hyperglycemia (up to 303 mg/dL). Peritoneal fluid analysis showed 1,130 leukocytes/mm³ with 78% polymorphonuclear cells and Gram-negative bacilli on Gram staining, supporting the diagnosis of peritoneal dialysis-associated peritonitis.

Given the suspicion of upper gastrointestinal bleeding, upper gastrointestinal endoscopy was performed on November 22, 2024, revealing extensive circumferential involvement of the distal esophageal mucosa with

a sharply demarcated transition at the gastroesophageal junction (Figure 1). Based on the clinical context and endoscopic findings, what is the most likely diagnosis?

Figure 1.



Solution of the case: An Unusual Distal Esophageal Finding: An Endoscopic Challenge

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Case solution

The image corresponds to acute esophageal necrosis, also known as “black esophagus.” As illustrated in Figure 1B, upper gastrointestinal endoscopy demonstrated diffuse circumferential black discoloration of the distal esophageal mucosa with a well-demarcated transition at the gastroesophageal junction, a hallmark feature of acute esophageal necrosis. This condition is usually associated with severe underlying illnesses and the combination of multiple predisposing factors that contribute to esophageal mucosal injury. In this patient, sepsis secondary to peritonitis, advanced chronic kidney disease on peritoneal dialysis, type 2 diabetes mellitus, anemia, and hypoalbuminemia likely contributed to its development. Conservative treatment was initiated, including bowel rest, proton pump inhibitors, sucralfate, and management of the underlying infection, resulting in progressive clinical improvement. Early recognition of this condition is essential to reduce the risk of complications and guide timely treatment.

Follow-up and outcomes

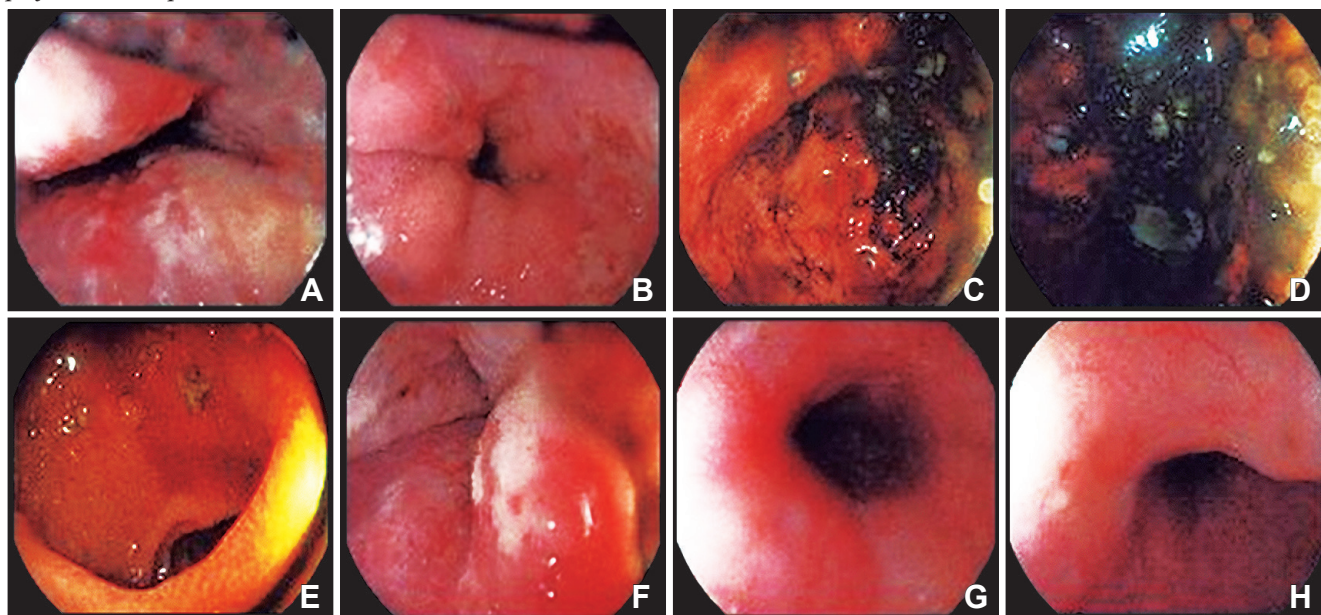
The patient showed progressive clinical improvement during hospitalization after initiation of conservative management. Coffee-ground emesis, dysphagia, and epigastric pain gradually resolved, and oral intake was progressively reintroduced. A liquid diet was initially tolerated and subsequently advanced as symptoms improved. No clinical evidence of early complications

of acute esophageal necrosis, including esophageal perforation or mediastinitis, was identified.

Given the sustained clinical improvement, resolution of upper gastrointestinal bleeding symptoms, progressive tolerance of oral intake, and absence of signs suggestive of esophageal perforation or other complications, repeat upper gastrointestinal endoscopy was not performed during the same hospitalization. The patient was discharged with outpatient follow-up by the Gastroenterology and Nephrology departments. During outpatient follow-up, an upper gastrointestinal endoscopy requested by the Gastroenterology Department was performed on September 23, 2025. The examination showed no residual endoscopic features of acute esophageal necrosis. Instead, severe Los Angeles grade D erosive esophagitis was identified, with linear, flat erosions greater than 5 mm involving approximately 90% of the esophageal circumference and along with remnants of old blood in the stomach (Figure 2). Proton pump inhibitor therapy was therefore continued.

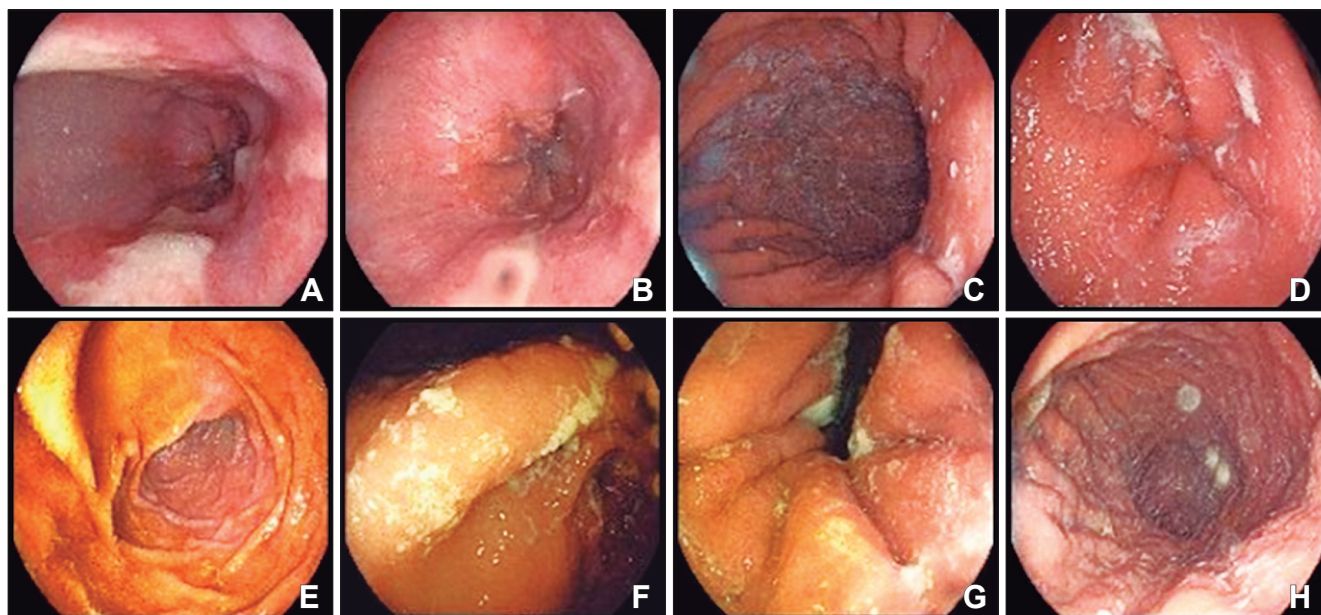
During a subsequent hospitalization for upper gastrointestinal bleeding, imaging studies demonstrated findings consistent with portal hypertension. Upper gastrointestinal endoscopy performed on October 27, 2025, revealed Los Angeles grade C erosive esophagitis and chronic gastropathy, without evidence of recurrent acute esophageal necrosis (Figure 3). The patient showed a favorable clinical response to medical management and was discharged on pharmacological therapy with continued outpatient follow-up.

Figure 2. During outpatient follow-up, a control upper gastrointestinal endoscopy requested by the Gastroenterology Department was performed on September 23, 2025



Panels A – H show the endoscopic findings after resolution of acute esophageal necrosis. Panel F demonstrates linear, flat erosive lesions of the distal esophagus, consistent with Los Angeles grade D erosive esophagitis, involving approximately 90% of the esophageal circumference. No residual black discoloration characteristic of acute esophageal necrosis was observed. The stomach contained remnants of old blood.

Figure 3. Upper gastrointestinal endoscopy performed on October 27, 2025, during a subsequent hospitalization for upper gastrointestinal bleeding



Panels A – H show the endoscopic findings. Panel B demonstrates two longitudinal erosions measuring 15 mm and covered with fibrin in the distal third of the esophagus, consistent with Los Angeles grade C erosive esophagitis. No evidence of recurrent acute esophageal necrosis was identified. The stomach contained whitish residual material, and the mucosa of the fundus, body, and antrum showed moderate edema and mild erythema.

Consent for publication. The authors certify that informed consent was obtained from the patient for the publication of her clinical information and images. The patient

understands that her name and initials will not be published and that all reasonable efforts will be made to protect her identity; however, complete anonymity cannot be

guaranteed. Approval by an Institutional Review Board or Research Ethics Committee was not required for the publication of this case report.

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