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IMAGE | ENDOSCOPY

A Giant Syphilitic Gastric Ulcer

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CASE REPORT

A 66-year-old man presented with coffee ground emesis for 2 weeks progressing to hematemesis over the previous day and refused to undergo upper endoscopy. He had a medical history significant for alcohol abuse, tobacco abuse, schizophrenia, gastroesophageal reflux disease, hepatitis C, lower back pain, cerebrovascular accident, syphilis, cocaine use, and upper gastrointestinal bleeds.

The complete blood count showed microcytic anemia with hemoglobin 7.1 g/dL, mean corpuscular volume 64.8 fL with underlying iron deficiency (ferritin 4.4 ng/mL, serum iron $<$ 10 mcg/dL). He was given intravenous iron. An esophagogastroduodenoscopy (EGD) was performed. EGD showed a 3-cm wide deep-cratered ulcer with an adherent clot at the incisura, which was very friable, necrotic with a firm base (Figure 1). The clot was removed, and ulcer was treated with epinephrine and bipolar cautery. Biopsies were obtained from the edge and base of this giant ulcer along with random gastric biopsies (Figures 2 and 3). Pathology revealed chronic inflammation with plasma cells, histiocytes, lymphocytic infiltration, and presence of *Helicobacter pylori* and *Treponema pallidum* by immunohistochemistry (Figure 3). Immunohistochemistry was negative for CD3, CD20, CD45, CD79A, AE1/AE3, BCL2, BCL6, BER EP4, and CAM 5.2. The area of ulcer highlighted the extensive presence of granulocytes by CD43. *Treponema pallidum* were spirochetes seen on immunohistochemistry testing (Figure 3). Periodic Acid Schiff-Alcian blue stain was negative as well. A diagnosis of syphilitic gastric ulcer was made.

The patient was treated with penicillin for syphilis and with bismuth-based quadruple therapy for *H. pylori*. A repeat EGD performed 18 months later showed that the ulcer had shrunken to a clean-based $<$ 1 cm ulcer without any deep cratering. Repeat biopsies for *H. pylori* and spirochetes were negative.

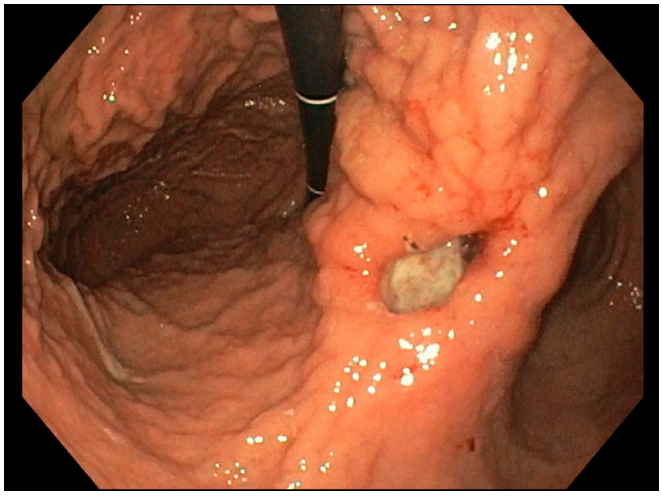

Figure 1. Retroflexed endoscopic view showing a giant incisural ulcer with an adherent clot on esophagogastroduodenoscopy.

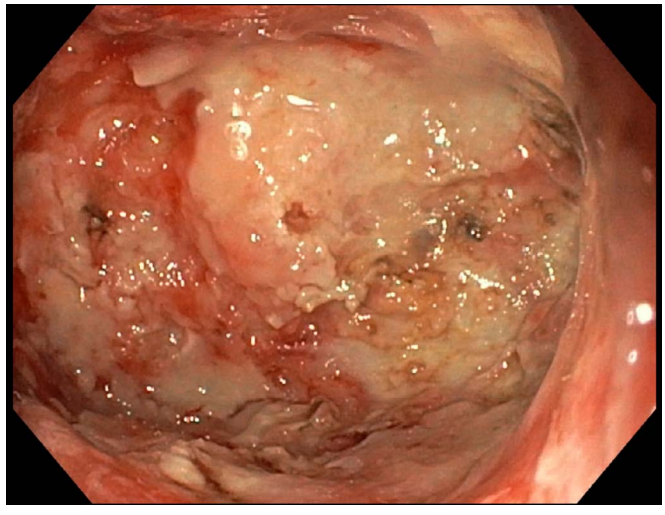

Figure 2. Closer view of incisural ulcer showing its base after paring the adherent clot and the area of endoscopic biopsy.

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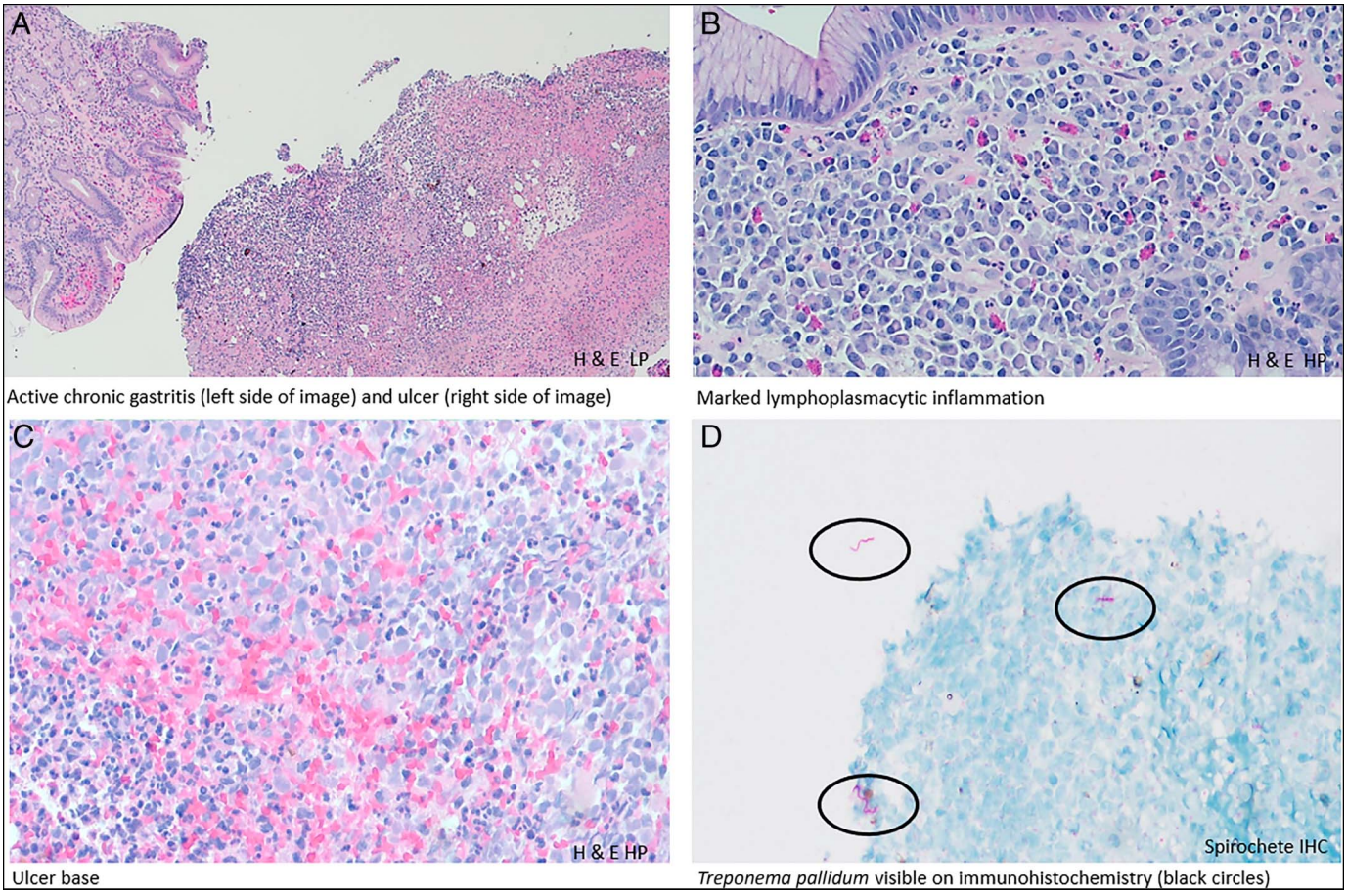

Figure 3. (A) Hematoxylin and eosin (H&E) stain (low-power magnification ×10) showing chronic gastritis on the left and ulcer on the right. (B) H&E stain (high-power magnification ×100) showing marked lymphoplasmacytic infiltration around the ulcer. (C) H&E stain (high-power magnification ×100) showing diminished mucosal glands and presence of *Helicobacter pylori*. (D) Spirochetes seen on (low-power magnification ×10) gastric ulcer biopsy (black circles) found to be *Treponema pallidum* on immunohistochemistry.

Syphilis is considered the master masquerader capable of infecting almost every organ system with a myriad of manifestations.[1–4] Gastrointestinal manifestations of secondary syphilis include stomach ulcers (epigastric pain, vomiting, and weight loss), upper gastrointestinal bleeding, anemia, gastroparesis, rarely obstruction and perforation, colitis, rectal mass/ulcer, hematochezia, anal pain, and hepatitis/hepatomegaly. Diagnosis is made by performing a treponemal and/or nontreponemal test and immunohistochemistry for *Treponema pallidum* on biopsies. Fortunately, despite being around for centuries, *Treponema* is still sensitive to penicillin and responds well to intramuscular penicillin G. Although rare, gastroenterologists should be aware of various gastrointestinal manifestations of secondary syphilis under appropriate clinical settings.

DISCLOSURES

Author contributions: R. Verma and C. White took care of the patient and obtained history. T. Oza and A. Azouz reviewed and provided the pathology images. MA Elshafey performed the literature search. All authors contributed to writing and approving the manuscript. R. Verma is the article guarantor.

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