We present a 50-year-old woman with human immunodeficiency virus admitted to the hospital for melena and anemia who underwent esophagastroduodenoscopy for evaluation of upper gastrointestinal bleed. She is found to have inflammation of the esophagus with ulcerations and crater formation. A biopsy reveals severe inflammation with lymphocytic infiltrates with civatte bodies suggestive of lichenoid esophagitis with the presence of spirochetes (*Treponema pallidum*). The presence of syphilis in the esophagus causing lichenoid esophagitis is an extremely rare presentation that has never been reported.

**INTRODUCTION**

Syphilis of the gastrointestinal (GI) tract is an extremely rare entity. Our extensive literature search did not reveal any reported cases of esophageal syphilis in the past 3 decades. Hence, we present a case of esophageal syphilis with rare pathological diagnosis of lichenoid esophagitis.

**CASE REPORT**

The patient is a 50-year-old woman with human immunodeficiency virus with a CD4 count of 217 mostly noncompliant with the highly active antiretroviral therapy who presented to the hospital with 4 days of diarrhea, nausea, vomiting, and melena stools. Her medical history is also significant for end-stage renal disease requiring intermittent hemodialysis and cirrhosis secondary to hepatitis C virus. Because of the melena stools, gastroenterology was consulted for the evaluation of upper GI bleeding. On admission, patient’s hemoglobin was found to be 6.7 mg/dL which dropped from 10.1 mg/dL from her previous admission to the hospital 4 months earlier. The patient underwent 2 upper endoscopies with biopsies in the past 6 months secondary to dysphagia which showed nonspecific inflammation and positive for *Candida albicans*, which was treated with 3 weeks of fluconazole.

Owing to the concern of acute blood loss from an upper GI bleed, another upper GI endoscopy showed severe inflammation and ulcerations with crater formation in the midesophagus and distal esophagus. A stricture was present at 35 cm through which the regular scope could not be passed (Figure 1). The examination was completed using a 6-mm slim scope with normal examination of the stomach and the duodenum. Dilation was not performed because of severe inflammation and the risk of perforation. For further evaluation of the underlying esophageal pathology, several biopsies were obtained from the midesophagus and distal esophagus.

The histopathologic examination of the biopsies from the esophageal ulcers showed marked intraepithelial lymphocytes, neutrophils, dyskeratotic keratinocytes (civatte bodies), and dense band-like lymphoplasmocytic infiltrates in the lamina propria, consistent with lichenoid pattern of inflammation along with the presence of spirochetes (*Treponema pallidum*) in the esophageal mucosa diagnosed using immunohistochemistry (Figure 2). No fungal elements or viral inclusion bodies were noted. The tissue was negative for cytomegalovirus and herpes simplex viruses I and II. In light of the histopathological findings, diagnosis of esophageal syphilis was made, causing inflammation leading to the rare diagnosis of lichenoid esophagitis. The patient was found to be positive for Treponemal antibodies and *T. pallidum* particle agglutination assay, hence confirming the diagnosis of syphilis. The patient was initiated on IV penicillin for 14 days, followed by intramuscular penicillin G 2.4 million units on discharge. On a follow-up visit with infectious disease 4 weeks after the discharge, the patient reported resolution of the symptoms. After 3 months, the patient was again admitted at a different hospital for...
complaints of dysphagia. Another upper endoscopy with biopsy showed the presence of inflammation in the esophagus with *C. albicans* but negative for *T. pallidum*. The patient was treated with 3 weeks of fluconazole for candida esophagitis.

DISCUSSION

Extragenital syphilis has been reported as either single case reports or small case series.1 Syphilis of the stomach has been reported in only 1% of the cases.2 Most cases of syphilis involving the GI tract have been reported in the rectum3 and oral cavity.4,5 Ijiri et al in 2016 described syphilis of the whole GI tract, but they did not mention any involvement of the esophagus.6 After extensive research, we found only few case reports that described esophageal syphilis and none in the past 3 decades. Hence, we have very limited information regarding the effects of syphilis on the esophagus. Lichenoid esophagitis is described as lichenoid pattern of inflammation for which no specific histological diagnosis can be made.7 Salaria et al in 2013 described the lichenoid pattern of esophageal injury secondary to lichen planus, drug reactions, or viral infections.7 Syphilis causing lichenoid pattern of injury in the esophagus has not been reported. Lichenoid esophagitis causes extensive inflammation that can lead to bleeding in the GI tract as described in our case and can very rarely cause fatal upper GI bleeding leading to death, as reported by Mitchell and Patrella in 2013.8 Lichenoid esophagitis can lead to dysphagia, superficial sloughing of the mucosa with stricture formation, and ulceration.7,9 Our patient also presented with the similar clinical and endoscopic findings of ulcerations and stricture formation. Apoptotic squamous cells known as civatte bodies are scattered throughout the injured epithelium and are pathognomonic of lichenoid esophagitis.7 Our case describes the presence of civatte bodies, thus confirming the diagnosis of lichenoid esophagitis.

In conclusion, the presence of *T. pallidum* in the esophagus has been described very rarely with the last case report dating back to the 1970s. Lichenoid pattern of inflammation in the esophagus is also considered a rare entity and has never been described to be caused by syphilis. Hence, our case is extremely unique because we strongly believe that in our patient the lichenoid esophagitis was caused by the presence of *T. pallidum* in the esophagus.

DISCLOSURES

Author contributions: S. Siddiqui wrote the manuscript and reviewed the literature. S. Khurana reviewed the literature. Z. Cai provided the pathology images. S. Larson edited the manuscript and is the article guarantor.

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Informed consent was obtained for this case report.

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Figure 1. Esophagoduodenoscopy showing esophageal ulcers with crater formation and stricture at 35 cm.

Figure 2. Biopsy of the esophageal ulcer showing (A) lichenoid pattern of injury with civatte bodies (red arrow) and intraepithelial lymphocytosis (green arrow) (100× magnification) and (B) numerous spirochetes.
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