

Rare cause of whitish gastric mucosal plaque on upper gastrointestinal endoscopy

Srinu Deshidi¹, Gaurav Mahajan², Amrutha Guguloth³, Sreekanth Appasani¹

From ¹Consultant, Department of Gastroenterology, Krishna Institute of Medical Sciences, Hyderabad, Telangana, ²Associate Professor, Department of Gastroenterology, Indian Naval Hospital Ship Asvini, Mumbai, Maharashtra, ³Assistant Professor, Department of Pathology, Government Medical College, Mahabubabad, Telangana, India

A 28-year-old lady with no known comorbidities presented to our outpatient department with a 3-month history of intermittent, mild to moderate upper abdominal pain, which worsened after meals. She denied any history of vomiting, fever, weight loss, jaundice, diarrhea, or abdominal distension. She had been taking pantoprazole 40 mg once daily for 4 weeks and sucralfate 1 g oral suspension 3 times daily for the past 2 weeks. Her general physical examination was normal. Routine blood investigations and abdominal ultrasound were unremarkable. Due to ongoing symptoms, an upper gastrointestinal endoscopy was performed, which revealed a whitish, adherent plaque in the fundal region that was difficult to wash off, with underlying hyperemia (Fig. 1). Biopsy from the plaque revealed ulceration with features of chronic superficial gastritis, and the rapid urease test from the antral mucosal biopsy was positive (Fig. 2). A diagnosis of *Helicobacter pylori*-associated chronic gastritis was made, and she was initiated on triple therapy with esomeprazole 40 mg, amoxicillin 750 mg, and clarithromycin 500 mg, each given twice daily for 14 days. Repeat endoscopy after 1 week showed complete disappearance of the plaque. Her symptoms gradually improved, and she became asymptomatic within 4 weeks. The adherent plaque was most likely caused by sucralfate deposits on the inflamed gastric mucosa.

Persistent dyspeptic symptoms are among the most common indications for gastroscopy in gastroenterology practice. Whitish or yellowish-white plaques in the stomach are uncommon findings during endoscopy. Differential diagnoses for gastric whitish plaques include xanthelasma, *Candida* infection, gastric mucosal calcinosis, gastric metaplasia, etc [1-3]. Sucralfate, an aluminum salt of sucrose octasulfate, is an anti-ulcer agent that forms a stable drug-protein complex on eroded or ulcerated mucosa, protecting it from further injury by

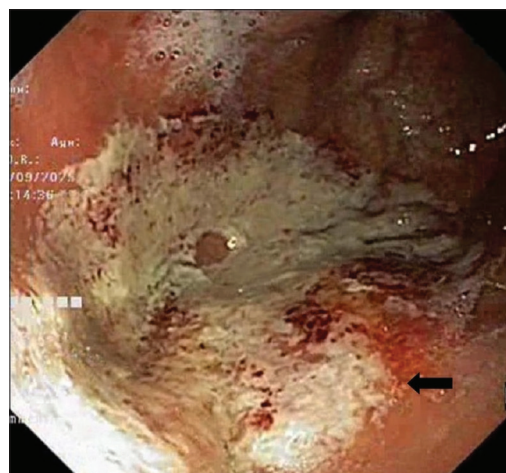


Figure 1: Endoscopic images showing whitish plaque with underlying hyperemia in the gastric fundal mucosa (black arrow)

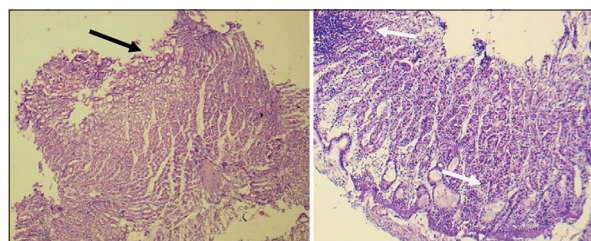


Figure 2: Low power hematoxylin and eosin-stained section of gastric mucosa showing foveolar lining epithelium with ulceration (black arrow) and lymphoplasmocytic infiltration of lamina propria (white arrow)

acid, pepsin, and bile salts. It also promotes the release of endogenous prostaglandin E2 from the gastric mucosa and exerts a trophic effect by activating genes encoding epidermal growth factor and its receptors [4]. However, sucralfate acts on the gastric mucosa in a more general and complex fashion than the formation of a protective “barrier” only on ulcerated surfaces. In normal mucosa, sucralfate can form whitish deposits that adhere firmly to the glandular surface, leading to histological changes such as disruption and exfoliation of some surface epithelial cells, mucosal hyperemia, increased mucus secretion from the surface epithelium, and edema of the

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Correspondence to: Srinu Deshidi, Consultant, Department of Gastroenterology, Krishna Institute of Medical Sciences, Hyderabad - 500003, Telangana, India. E-mail: srinu.deshidi@gmail.com

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lamina propria and submucosa [5,6]. The disruption of certain surface epithelial cells by sucralfate may act as a trophic stimulus for epithelial regeneration and can lead to the release of prostaglandin precursors, which themselves serve as protective agents for the gastric mucosa [6]. We report these rare cases of adherent whitish gastric mucosal plaque caused by sucralfate to raise awareness among gastroenterologists, helping to prevent unnecessary investigations and biopsies.

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