Case Report

Endoscopic ultrasound of isolated gastric corrosive stricture mimicking linitis plastica

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ABSTRACT

Isolated gastric outlet obstruction after 1 month of asymptomatic ingestion of corrosive is a rare phenomenon and rarely reported. In this type of cases, diagnosis is very difficult due to no symptoms at the time of poisoning, and biased history. We report a case of a young male presented with isolated gastric outlet obstruction after 1 month of asymptomatic ingestion of toilet cleaner, which was known to us later, mimicking linitis plastica. On upper endoscopy, the stomach was grossly edematous, antrum edematous and inflamed with reduced distensibility and narrow pyloric canal. Endoscopic ultrasound of the stomach revealed diffuse thickening of the gastric wall, mainly the antrum, involving submucosa and muscularis propria. We propose corrosive injury to be in the differential diagnosis of gastric linitis plastica.

Key words: Corrosive, endoscopic ultrasound, linitis plastica

INTRODUCTION

Most of the time corrosive poisons cause severe local reactions then esophageal strictures and finally gastric injury. Rarely acid may cause only late complication like gastric injury. In this type of cases, diagnosis is very difficult due to no symptoms at the time of poisoning, and biased history.[1] The mode of tissue injury with acids is a process of coagulation necrosis. The coagulum prevents the corrosive agent from spreading transmurally and hence reduces the incidence of full thickness injury.[2] We report a case of isolated gastric outlet obstruction after 1 month of asymptomatic ingestion of toilet cleaner, which was known to us later, with full thickness injury mimicking linitis plastica.

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CASE REPORT

A 35-year-old male presented with intractable vomiting of gradual onset and progressive course for the last 7 days. The frequency of stool passing and urination were reduced. No other history of fever, jaundice, epigastric pain, hematemesis, melena, or headache was reported. On examination, patient was conscious, lethargic, dry tongue, and afebrile. Heart rate was 130/min with all other vitals normal. All other systemic examinations revealed no abnormalities. Biochemical profile was unremarkable. On upper endoscopy, the stomach was grossly edematous, antrum edematous and inflamed with reduced distensibility and narrow pyloric canal. Multiple biopsies were taken and sent for histopathological examination, which revealed chronic non-specific inflammation. Endoscopic ultrasound (EUS) was done, which revealed diffuse thickening of the gastric wall up to 21 mm in the antrum [Figure 1] and 11.5 mm in the gastric body [Figure 2] involving submucosa and muscularis propria. Biopsies were

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repeated again with bite-on-bite technique and sent for histopathological examination which again revealed chronic nonspecific inflammation. After which, patient's father confess that there was a suicidal attempt 1 month ago with a toilet cleaner which passed without any symptoms or complications. The patient was referred for surgical evaluation. Exploration revealed marked thickening of the gastric wall of the distal stomach with marked luminal stenosis, however, the serosa was intact [Figure 3]. Subtotal gastrectomy was done, and the patient passed a smooth postoperative course. Postoperative pathological examination of the resected specimen showed gastric mucosa with focal erosions and submucosa with granulation tissue and dense inflammatory reaction [Figures 4 and 5].

DISCUSSION

The first corrosive induced antral stenosis was reported in 1828.[3] "Acid licks the esophagus and bites the stomach" was the dictum in the past. This principle has been challenged by many authors who have shown that even with acid ingestion esophageal injuries are common.[4] The magnitude of the injury depends upon several factors such as the nature of the caustic agent, volume ingested, concentration, duration of mucosal exposure, age of the patient and the intent (suicidal or accidental) with which the corrosive was consumed.[5] The largest Indian experience, reported 33% of 109 injuries with isolated gastric injury. The majority had prepyloric strictures (83.5%) while the remaining strictures were located in the antrum, body, pyloroduodenal area, or were diffuse.[6] The mode of tissue injury with acids is a process of coagulation necrosis. The coagulum prevents the corrosive agent from spreading transmurally and hence reduces the incidence of full thickness injury.[2] Diffuse gastrointestinal (GI) wall thickening is predominantly observed in the stomach and less frequently, in the esophagus and rectum. Malignant causes include linitis

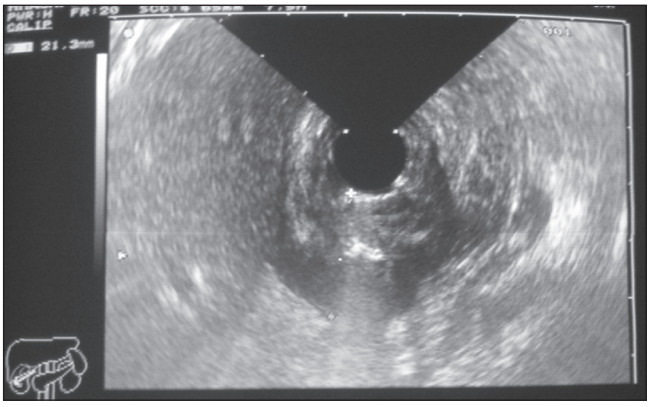

Figure 1. Radial endoscopic ultrasound image showing diffuse thickening of the antral wall (21 mm) involving mainly the submucosa and muscularis propria

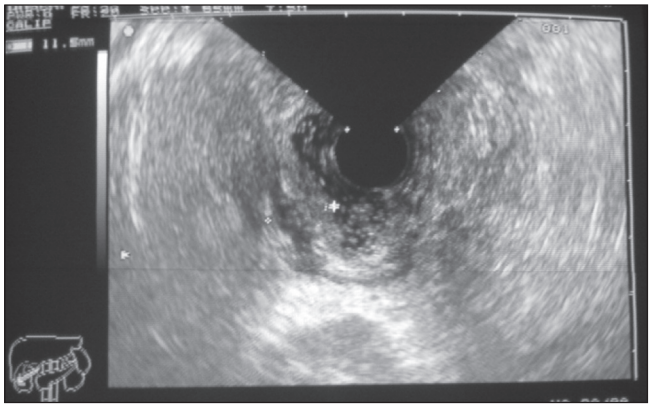

Figure 2. Radial endoscopic ultrasound image showing diffuse thickening of the gastric body wall (11.5 mm) involving mainly the submucosa and muscularis propria

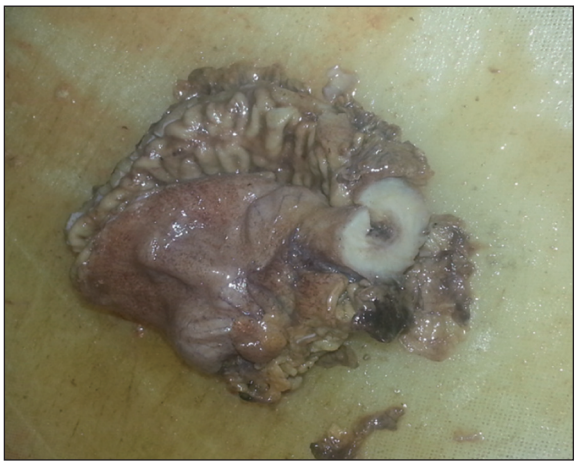

Figure 3. The resected specimen is showing diffuse thickening of the gastric wall mainly the antrum and the pyloric canal

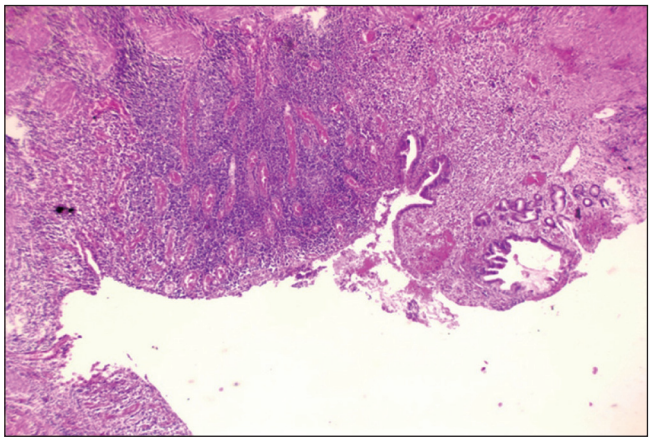

Figure 4. Gastric mucosa with focal erosions and submucosa showing dense inflammatory reaction, H and E, ×200

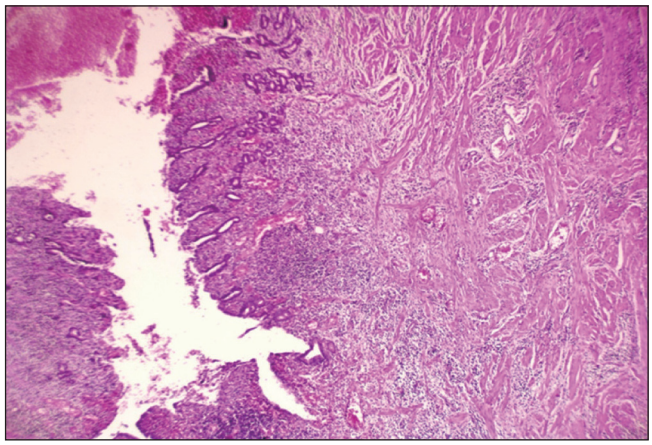

Figure 5. Ulcerated area with granulation tissue and dense inflammatory reaction, H and E, ×200

plastica and less frequently, lymphoma or diffuse metastasis. Benign causes are multiple, including eosinophilic infiltration, Zollinger-Ellison syndrome, Ménétrier's disease, and amyloidosis.[7] In subepithelial infiltrating tumors, standard endoscopic biopsy sampling often yields false negative results, and the diagnostic yield of bite-on-bite biopsy sampling is unknown, although this technique is commonly used.[8] At least in the stomach, EUS without sampling is relatively accurate in discriminating malignant from benign conditions: In a prospective study of 61 patients, the thickening of the submucosa and/or muscularis propria (as opposed to thickening limited to the mucosa) was the single independent predictor of malignancy; the clinical impact of this feature was high because the probability of malignancy was 95% versus 5%, respectively, depending on whether deep wall layers were thickened or not.[9] In a prospective study, the diagnostic accuracy of EUS-fine needle aspiration was significantly lower for diffuse GI wall thickening as compared with all other indications.[10] In this patient, there was thickening of the submucosa and muscularis propria although it is a benign lesion. We propose corrosive injury to be in the differential diagnosis of gastric linitis plastica.

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