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Letter to the Editor

Food Bolus Impaction in Eosinophilic Esophagitis

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Dear Editor, A 32-year-old man presented with complete dysphagia after eating a traditional greek meat lunch ("souvlaki") with yogurt. Past medical history revealed four episodes of food impaction during last 2 years, which were relieved by endoscopic means. No symptoms of dysphagia, chest pain or retrosternal burning suggesting gastroesophageal reflux disease (GERD) were noted between these episodes. There was no history of atopy or allergy. Hematological and biochemical tests were unremarkable. Upper endoscopy demonstrated an impacted meat bolus (Fig. 1) in the lower segment of esophagus, which was removed with a Roth net of large size. Re-endoscopy showed multiple mucosal rings (Fig. 2) with linear furrows in esophageal lumen and normal stomach and duodenum. Histological examination of biopsies taken from esophagus, gastric antrum and 1st and 2nd part of duodenum demonstrated markedly increased number of intraepithelial eosinophils limited to esophageal mucosa. The patient was treated with fluticasone proprionate (2 mg/day per os) with histological improvement and no recurrence of symptoms at the one-year follow up.

Eosinophilic esophagitis (EE) is an increasingly recognized condition in adults, presenting with a variety of esophageal symptoms including dysphagia, atypical chest pain and heartburn not responding to medical therapy or a history of recurrent food impaction.[1,2] Typically, EE is characterized by dense eosinophilic infiltration of the esophageal mucosa (>20 eosinophils/high power filed) in the absence of gastric or duodenal eosinophil infiltrates.[1] To date, the pathogenesis of EE remains unknown, but a personal and family history of atopy and immune-mediated mechanisms triggered by both food and inhalant allergens (aeroallergen hypersensitivity) are implicated.[1-4] Mechanisms involving interleukin (IL)-5, eotaxin-3 and signal transducer and activator of transcription 6 have been considered and appear to represent therapeutic targets.[4] A number of endoscopic features have been described, including mucosal rings (often multiple), linear furrows, multiple white papules (eosinophilic microabscesses), strictures (frequently proximal), mucosal ulceration and small-caliber esophagus.[3,5] Although optimal treatment for EE has not been defined yet, current therapies include specific elimination diets or elemental diets; systemic and topical corticosteroids; oral antihistamines; sodium cromoglycate; anti-inflammatory drugs such as leukotriene receptor antagonists (selective inhibitor of leukotriene D4 receptor), anti-IL-5 (Mepolizumab), and anti-IL-13 monoclonal antibodies; esophageal dilation; and, in cases of co-existing GERD, oral intake of

Figure 1. Endoscopic picture showing impacted meat bolus in the lower segment of esophagus.

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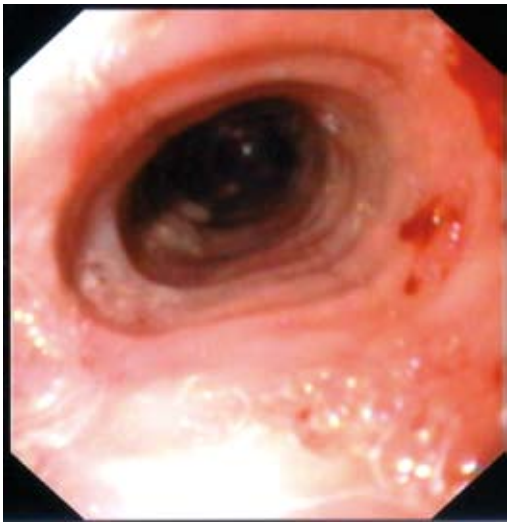

Figure 2. Endoscopic picture, taken a few days after removal of impacted meat bolus, showing multiple mucosal rings with linear furrows in esophageal lumen and normal stomach and duodenum.

proton pump inhibitors.[6,7] Regarding EE natural history, there has been no association with malignant conditions, but there is concern that the chronic, uncontrolled inflammation will evoke irreversible structural esophageal alterations, resulting in tissue fibrosis, stricture formation, and impaired function.[8]

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