A 33-year old male patient was referred to the endoscopy unit because of heartburn and dysphagia lasting for 6 weeks. Symptoms improved only slightly after 4 weeks’ treatment with pantoprazole. No information was available regarding his medical history or medication intake.

Endoscopy revealed a pale esophageal mucosa with multiple small white spots, predominantly in the distal esophagus (Fig. 1). Cardia, stomach, and duodenum were normal. A biopsy specimen of the esophagus showed an increased number of eosinophils (peak count 150 per high power field [hpf]), concentrated within the surface epithelium, which is compatible with the presence of eosinophilic microabscesses (Fig. 1).

The symptoms suggestive of esophageal dysfunction, together with the pathological findings of eosinophil-predominant inflammation led to the diagnosis of eosinophilic esophagitis.

Eosinophilic esophagitis is a clinicopathological diagnosis based on the presence of symptoms associated with esophageal dysfunction combined with biopsy findings of eosinophil-predominant inflammation. Common clinical presentations in adults are dysphagia, food impaction, refractory heartburn, central chest pain that is not responsive to antacids, and upper abdominal pain. It is a disease affecting all age groups, with a male predominance [1–3].

Typically, endoscopy reveals esophageal rings (so-called trachealization or feline esophagus) and longitudinal furrows as presented in Fig. 2 (from a patient with long-standing episodes of recurrent food impaction and dysphagia, and a peak eosinophil count of 110/hpf at biopsy). Other endoscopic features include white plaques, strictures, narrowing of the esophagus, and pallor or decreased vascularity [4].

Eosinophil-predominant inflammation on biopsy is generally considered to be present when there is a minimum of 15 eosinophils per hpf on microscopy. It is important to exclude alternative causes of esophageal eosinophilia such as gastro-esophageal reflux disease, celiac disease, Crohn’s disease, infection, hypereosinophilic syndrome, achalasia, drug hypersensitivity, vasculitis, pemphigoid vegetans, connective tissue disease, and graft-versus-host disease [5].

References

Competing interests: None

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Bibliography
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