Case report

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Patient with chest pain refractory to proton pump inhibitor: report of a case of eosinophilic esophagitis

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Abstract

Eosinophilic esophagitis (EoE) is the most common form of gastrointestinal disorders associated with eosinophilia. Typically, an inappropriate accumulation of eosinophils is found in the esophageal mucosa. EoE can be well managed and treated with several options that include an elimination diet, drug therapy (oral and topical steroids) and esophageal dilatations.

We herein, report a case of a 49-year-old male affected by EoE associated to chest pain, treated with proton pump inhibitor without clinical response. The patient suffered from long lasting postprandial fullness and gastroesophageal reflux disease-like symptoms. He had a history of episodic asthmatic attacks and allergic rhinitis but had not dysphagia or food impaction. The patient recovered completely after an adequate treatment. ***Clin Ter 2014; 165(1):e70-72. doi: 10.7417/CT.2014.1675***

Key words: *eosinophilic esophagitis (EoE), proton pump inhibitor (PPI), chest pain*

Introduction

Eosinophilic esophagitis (EoE) is the most common form of gastrointestinal disorders associated with eosinophilia, which is characterized by an inappropriate accumulation of eosinophils in the esophageal mucosa (1).

EoE is an emerging underdiagnosed disease in adults, associated with allergic or asthmatic disorders, which typically affect males in the second-fourth decade of life, is often associated commonly with personal or family history of allergy (2). Dysphagia, heartburn and chest pain are the main symptoms present in half of adults patients with food obstruction of the esophagus (3).

Symptoms frequently mimic those of gastroesophageal reflux disease, but the diseases are distinct in their histopathology, gene expression signature, response to therapy, hereditary risk, and association with allergies (4).

The classical endoscopic appearance of the esophagus is polymorphic, including papular elevations, whitish exudates, corrugation, longitudinal furrows, undulated mucosa, reddening, multiple contraction rings and strictures. The esophagus may be normal in 25% of cases (3, 5).

Esophageal manometry often shows non-specific motor disturbances, such as hypoperistaltic and hyperkinetic disorders, or non transmitted, simultaneous contraction waves (5).

Case report

A 49-year-old man with chest pain and long lasting postprandial fullness and gastroesophageal reflux disease-like symptoms was admitted to our Department. There was no history of dysphagia or food impaction. The patient reported episodic asthmatic attacks and allergic rhinitis treated on demand with short courses of steroids and antihistamines beginning at the age of 20 years. Skin prick tests (SPT) previously performed were negative. An esophagogastroduodenoscopy (EGDS) prescribed by another gastroenterologist, showed only mild hyperemia of the distal esophagus, whereas biopsies of the antrum and gastric body showed no alterations. The esophagus was not biopsied. Therapy with omeprazole 20 mg once daily was prescribed. After six months with partial response to proton pump inhibitor (PPI), the patient experienced both worsening of gastroesophageal reflux disease and allergy symptoms. In presence of chest pain, to exclude cardiologic disease, was required a cardiology consult, moreover was increased PPI therapy to 40 mg once daily and was programmed a check-up after three months. The patient came back to our Institute with no cardiac pathology detected and without improvement with PPI treatment. We programmed at our digestive endoscopy service a EGDS with esophageal biopsies of proximal, middle, and distal esophagus. EGDS showed marked hyperemia of the distal esophagus without macroscopic alterations in the stomach and duodenum (Fig. 1).

Histological examination of esophageal biopsies showed chronic esophagitis, marked hyperplasia of the papillae and the basal layer, superficial erosion with significant eosinophi-

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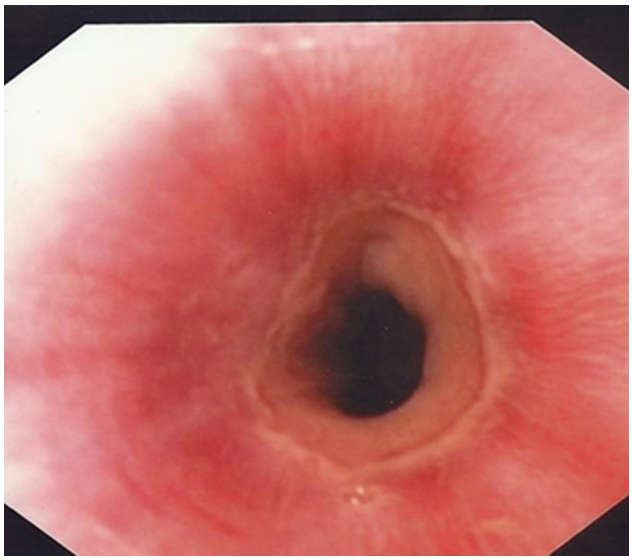


Fig. 1. High hyperemia of the distal esophagus.

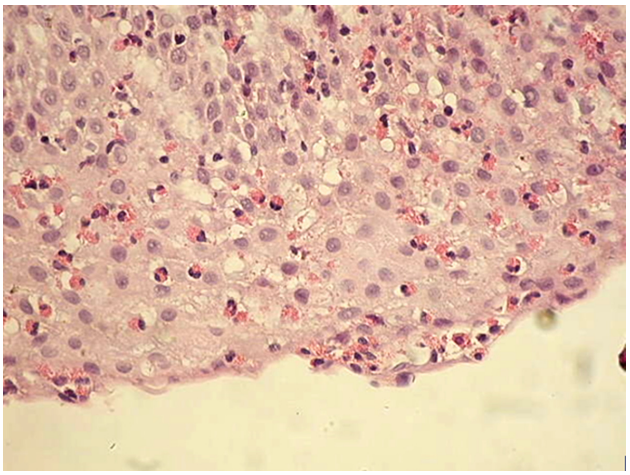


Fig. 2. Massive eosinophilic infiltrate with clusters of eosinophils at epithelial surface. H-E, x250 (original magnification).

lia, a number of intraepithelial eosinophils (20/hpf) formed clusters mainly located in the upper half of the epithelium and eosinophilic microabscesses on the surface (Fig. 2).

Colonoscopy with biopsies was also performed to exclude eosinophilic colitis and was both macroscopically and histologically negative. The patient was diagnosed with eosinophilic esophagitis. In accordance with the allergologist the patient was treated with fluticasone 250 mcg, two puffs swallowed, two times daily for three months. After this therapy, the patient came back to us with complete resolution of EoE and allergic symptoms. We have planned in agreement with the patient a new control, three months after discontinuation of therapy, to decide according to his state of health, the beginning of a new cycle of steroids.

Discussion

The esophagus is the only segment in the gastrointestinal tract that is normally devoid of eosinophils, thus their presence is pathological. EoE is a commonly observed medical problem and is well documented in pediatric patients. The adult form has only recently recognized as a distinct entity. Eosinophilic inflammation at esophageal level was first reported by Dobbins in 1977, followed by the description of patients with gastro-esophageal reflux symptoms resistant to PPI treatment who presented eosinophilic infiltration of the esophageal mucosa. However, only in 1993 EoE was defined as a clinical disorder independent by other eosinophilic diseases of the gastrointestinal tract (6). EoE has been described in patients with ethnic backgrounds, including Caucasians, African-Americans, Latins and Asians. There is evidence of a genetic predisposition in EoE, specifically at the level of the gene encoding for eotaxin-3 (7). A large proportion of patients have familial and/or personal antecedents of atopy (rhinitis, asthma, food allergy or atopic dermatitis) (8). It still remains unclear whether EoE is associated with an ethnic or racial predilection. The etiology of EoE is not clear, though allergy has been suggested as playing an important role in the development of the disease. The most common presenting symptoms are intermittent dysphagia, food impaction, gastroesophageal reflux disease-like symptoms, chest and abdominal pain. In the present case, EoE was diagnosed in a patient with chest pain and gastroesophageal reflux disease-like symptoms only partially responding to PPI therapy. Our case confirms the need for multiple biopsies in all portions of the esophagus, despite the presence of macroscopically normal esophagus, in particularly when symptoms like gastro-esophageal reflux prove resistant to PPI treatment and/or if asthma and allergic rhinitis are concomitant. In our case, we should underline that the patient had already undergone a EGDS without biopsies of the esophagus which was mildly hyperemic in the distal portion. Mimicking gastro-esophageal reflux disease, EoE is thus likely underdiagnosed.

In conclusion we should remember that EoE and gastro-esophageal reflux disease generally present independently but this conditions may coexist in some individuals underlining that the treatment options for EoE are different and include an elimination diet, drug therapy (oral and topical steroids) and esophageal dilatations (9, 10).

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