**Eosinophilic Esophagitis: A Rare Cause of Dysphagia: About A Case**

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**Abstract**

**Background:** Eosinophilic esophagitis is a chronic, inflammatory disease characterized clinically by symptoms related to esophageal dysfunction and histologically by a marked eosinophilic infiltrate in the esophageal mucosa. It predominantly occurs in young men with a history of atopy. Dysphagia and food impaction are the most common presentations. **Case presentation:** A 35-year-old man with known history of recurrent allergic rhinitis, presented with recurrent dysphagia revealing food impaction. Esophageal biopsies showed eosinophilic polynuclear exocytosis. Avoiding the responsible allergens, as well as topical corticosteroid therapy improved the patient’s symptoms. **Conclusion:** Eosinophilic esophagitis is still poorly understood. It is more common in young men, and there is usually a marked atopic component. It is the leading cause of dysphagia and feeding impaction in young men. Depending on the case, patients may require multidisciplinary treatment by gastroenterologists, allergists, immunologists and nutritionists. **Keywords:** Eosinophilic esophagitis, Dysphagia, corticosteroid therapy.

**Introduction**

Eosinophilic oesophagitis is a chronic inflammatory pathology characterised by isolated dense infiltration of the oesophageal mucosa by eosinophils, associated with high digestive symptoms, unrelated to any other cause [1].

The pathogenesis is still poorly known, but it is an allergic-type disease, the allergens can be either food or aeroallergens. It is the immune response mediated by Th2 lymphocytes that seems to be responsible for this pathology, with the increase in pro-inflammatory mediators (interleukin 4, 5, 13 and eotaxin) recruiting eosinophils [2].

**Observation**

A 35 year old man with a history of recurrent allergic rhinitis episodes presented with iterative and paroxysmal episodes of high dysphagia for two years.

Dysphagia occurred after food intake and predominated over solids. Clinical examination and standard laboratory tests were normal.

Esophageal endoscopy found a foreign food body upstream of a narrowed aspect of the esophageal lumen in the middle third of the esophagus. An extraction of food residue was carried out to complete the assessment.

A biological assessment was requested and did not show any abnormalities. A cervicothoracic scan showed a corrugated and isolated arrangement of the middle oesophagus. Esophageal manometry did not identify any motor disorders.

The patient was then lost to follow-up before returning to the emergency room for a worsening dysphagia. On questioning, the patient reported a predominantly solids dysphagia in solids, with worsening episodes lasting 2 years.

The endoscopy revealed esophageal food foreign body impaction, after extraction of food residues in the middle third; we revealed an erythematous and congestive esophageal mucosa. The gastroduodenal examination was normal.
Staged esophageal biopsies showed eosinophilic polynuclear exocytosis at a rate of 30 intraepithelial eosinophilic polynuclear cells per field at high magnification which led to the diagnosis of eosinophilic esophagitis.
A biological assessment was requested and revealed an Hb: 14.1 g / dl GB: 12000 ele/mm3 with hyperereosinophilia at PNE: 540 ele/mm3 and PLQ: 250000 ele/mm3, renal function as well as the ionogram blood were without abnormalities.

The blood dosage of Total IgE returned slightly increased to 250 kU/L. The diagnosis of eosinophilic esophagitis was retained. In addition, the patient was referred to allergology in order to search for a possible allergen responsible for the aggravation of the symptoms. The prick test and patch test revealed sensitivity to mites, pollens and cockroaches.

The management of our patient consisted first of all in avoiding the responsible allergens, moreover he was put on topical corticosteroid therapy with fluticasone (250 g, two puffs per day) was started for an initial period of eight weeks. The patient remained asymptomatic after 4 months of follow-up, and reported an improvement in his rhinitis.

**DISCUSSION**

Long considered a rare disease, eosinophilic esophagitis is experiencing a resurgence of interest due to an increase in its prevalence in industrialized countries, from 0.1% in 2001 to 1.9% in 2005 in the United States [3].

Adult eosinophilic esophagitis can occur at any age and in all ethnic groups, but predominates in Caucasian males between 30 and 40 years of age [4].

The male / female ratio is 3 to 1. We often find an atopic or allergic ground, as well as a genetic predisposition [5, 6].

There is no increase in neoplastic risk or impact on life expectancy, despite an impact on quality of life [7].

Symptoms of eosinophilic esophagitis vary greatly with age. In small children, there is difficulty feeding and gaining weight. The older child complains of abdominal pain and vomiting, while adolescents, like adults, mainly complains of dysphagia with solids (70%) and symptoms suggestive of gastroesophageal reflux (40%) [8].

As with our patient It is often accompanied by episodes of food impaction. The other signs are hyper salorrhea, vomiting, abdominal or chest pain, and heartburn. There is a long delay between the onset of symptoms and diagnosis [1, 9, 10].

In the majority of cases, it is associated with atopic manifestations (asthma, rhinoconjunctivitis, eczema), another allergic pathology or food allergies. A family history of allergy is present in more than 40% of patients [11].

The biological l results are usually normal in patients with eosinophilic esophagitis. However, a few studies show a hypercosinophilia greater than 300–350 / mm3, which can affect up to 40–50% of patients. In this case, the age of the patient must be taken into account, and the possible presence of concomitant allergies (seasonal, airborne allergens). This slight rise in eosinophils is normalized under effective treatment [1].

The first examination requested should be a gastroscopy with biopsies, as the barium swallow does not make the diagnosis. Gastroscopy is therefore indispensable and must be accompanied in all cases by...
biopsies of the esophagus, even if the endoscopic appearance is normal [12].

In eosinophilic esophagitis, endoscopy is normal in about 10% of cases [13].

There are characteristic, but not pathognomonic, images of eosinophilic esophagitis. The esophagus may present with stenosis, circular ring appearances, reduced size of the esophagus, or unspecific whitish spots not suggestive of candidiasis. Finally, there may be some granularity of the mucosa or longitudinal striations, but the mucosa may also be completely normal. Biopsies should be taken from the lower and upper oesophagus. The pathologist will make the diagnosis by revealing a significant infiltration by eosinophils throughout the esophagus (more than 15 per field, at magnification x 400) [12].

![Figure 10-12: Endoscopic features suggestive of eosinophilic esophagitis](image)

Figure 10-12: Endoscopic features suggestive of eosinophilic esophagitis A. Whitish deposits with streaky appearance. B. Ring aspect. C. Whitish appearance of the gastroesophageal junction [14]

![Figure 13: Histological features suggestive of eosinophilic esophagitis](image)

Fig-13: Histological features suggestive of eosinophilic esophagitis. HES A. Multiple eosinophils scattered in the superficial layers of the esophageal epithelium, HES 100. B. Microabscess of eosinophils on the surface of the esophageal epithelium, HES 400 [14]

It is sometimes difficult to tell the difference between eosinophilic esophagitis and genuine gastroesophageal reflux disease. In most studies, the distinction is based on the clinical and histological aspect. However, some forms of esophageal eosinophilia respond to treatment with a proton pump inhibitor and therefore should no longer be considered as eosinophilic esophagitis [15].
Treatment consists of eliminating allergens when they are identified. It combines topical corticosteroid therapy with budesonide (2 mg / day) or fluticasone (500 to 1000 mg / day) for six to eight weeks [1, 10, 11, 16].

Systemic corticosteroid therapy with prednisone is an alternative in case of failure or severe forms. The dosage is 1 to 2 mg / kg per day for eight weeks and then a gradual decrease over six weeks.

Other molecules are used in severe or corticosteroid-resistant forms: they are leukotriene antagonists (montelukast), immunosuppressants (azathioprine) and biotherapy (mepolizumab) [17].

**CONCLUSION**

Eosinophilic esophagitis is a recently recognized entity for which interest is growing. It is the leading cause of dysphagia and feeding impaction in young men.

The anatomic clinical forms are diverse and varied. It is important for gastroenterologists to think about when faced with any unexplained upper digestive tract symptoms, especially dysphagia. The performance of staged biopsies, in healthy and damaged areas, is of paramount interest in the diagnosis of eosinophilic esophagitis. Corticosteroid therapy is the reference treatment. Complications are rare, but recurrences are frequent. Regular endoscopic monitoring is not essential [17].

**REFERENCES**


