

Eosinophilic Esophagitis, A Rising Pathology

Miranda García M^{1*} and Gutiérrez Teira B²

¹Doctor of Emergency Department, Torrejon University Hospital, Madrid, Spain

²Family Doctor, Center of Health "El Soto", Móstoles, Madrid, Spain

Abstract

Eosinophilic esophagitis is an inflammatory disease of the esophagus, which shows an infiltrate with a high proportion of eosinophils in the pathological exam. The underlying etiopathogenesis is allergic, and its diagnosis is increasing in our population, especially in children and young adults, in the last decade. Its pathophysiology is not completely understood yet. The diagnosis has to be confirmed by endoscopy and biopsies. Differential diagnosis includes gastroesophageal reflux disease, eosinophilic gastroenteritis, Crohn's disease, connective tissue disease, hypereosinophilic syndrome, infections and hypersensitivity response to drugs. There is no curative treatment currently. We present a clinical case, initially evaluated in a Primary Care setting.

Case Report

A 40 year-old man, allergic to penicillins and intolerant of acetylsalicylic acid (ASA), non smoker, with history of rhinoconjunctivitis and asthmatic crises since childhood, and positive allergy tests to grasses and mites.

He had been treated with periodic vaccinations. Asthma crisis worsen in stressful situations and concomitant with common cold symptoms. Occasionally they have prompted his admission to hospital, but at the time of the actual complaint he has achieved a good symptomatic control. There is no family history of atopy.

His medical history also includes cholecistectomy due to acute lithiasic pancreatitis.

The patient showed significant weight loss in 4 months, although still keeping proper the Body mass index (24,5), was diagnosed as allergic symptom exacerbation presented, with wheezing, malaise, arthralgia looking for an emergency room.

In the physical examination his blood pressure was 102/66 mmHg. Weight: 74.2 kg. Height: 1.74 m. Good general condition, well hydrated and perfused, eupneic. Normal pharynx. No lymphadenopathies. Thyroid exploration within normal limits.

Cardiac auscultation: rhythmic, no murmurs. Pulmonary auscultation: vesicular murmur conserved, with wheezing in the lower half of the right lung. Abdomen was soft without tenderness, no masses or organomegaly were found.

Laboratory tests did not show any abnormalities.

The initial investigation showed PPD of 10 mm, although the normal chest x-rays. The patient doesn't remember if he took the vaccine. It's not usual keep positive reaction with 40 years of age, and probably means contact with the tubercle bacilli.

In view of this finding, and to rule out tuberculosis, he was asked to collect three sputum samples, paced out 48 hours, for Ziehl-Neelsen staining, but did not comply. Neither he attended the follow-up visit set to evaluate symptomatic changes and control of weight.

After three months of this episode the patient initiated symptoms of dysphagia to solids, which is a very common symptom in EEO in adults. At this time the high digestive endoscopy showed concentric stenosis in the esophagus, with signs of parakeratosis and microabscessos eosinophilic suggestive of esophagitis eosinophilic.

The patient was initially treated the EEO only with the exclusion of cow's milk protein and Proton pump inhibitor and general measures for the control of asthma.

Just a few months later, in allergist evaluation with allergy tests (it is not possible to know if UNICAP or Prick test) showed if positive for other foods (barley, rye, wheat, egg White, yolk, arizonia, olive and banana) currently has started with fluticasone, prednisone 20 mg per day, desloratadine, terbutaline if necessary and Proton pump inhibitor, and only at this time became asymptomatic. Two weeks later, the next query to the Allergist the patient was subjected to the new test prick which showed positive for nuts and peanuts, which were excluded in his diet. The corticosteroid was reduced to 15 mg per day six more weeks. The patient was able to eat better and swallowing problems disappeared.

At 8 weeks of starting treatment with corticoids, a second endoscopy was carried out and revealed that kept the appearance of EEO without stenosis.

Discussion

Eosinophilic esophagitis is a chronic disease whose diagnosis is increasing in the population (especially in children and young adults) in the last decade. It is most prevalent in young men [1].

Our patient in question has a personal history of allergies like asthma and rhinoconjunctivitis.

Sixty per cent of patients have a history of allergies: allergic rhinitis, asthma or eczema and between 20 and 40% show a family history of atopy.

Only 25% of patients do not show allergic sensitization in usual tests [2].

***Corresponding author:** Miranda García M, Servicio de Urgencias, Hospital Universitario de Torrejón, Madrid, Spain, Tel: 91 626 26 00; E-mail: mariomiranda7@hotmail.es

Received November 11, 2013; **Accepted** January 27, 2014; **Published** February 10, 2014

Citation: García MM, Teira BG (2014) Eosinophilic Esophagitis, A Rising Pathology. J Genet Syndr Gene Ther 5: 215. doi: [10.4172/2157-7412.1000215](https://doi.org/10.4172/2157-7412.1000215)

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Up to date, there is no evidence regarding a higher prevalence in certain geographical areas.

The pathophysiology is not completely established yet. Various studies have shown two possible mechanisms. One is the extrinsic or IgE-dependent, related to high serum IgE levels, atopic predisposition and the high percentage of patients positive for intradermal sensitivity tests. The second one is the intrinsic or IgE-independent mechanism, with the apparent involvement of T cells.

Some studies suggest there may be a family association, but it is difficult to determine whether this is due to a genetic predisposition or exposure to similar environments [3].

Other studies have showed elevated levels of eotaxin - 3 in eosinophilic esophagitis. In the long term, this cytokine expression and its genetic variation may be markers to distinguish eosinophilic esophagitis from other etiologies of esophagitis [4].

The symptoms can be quite diverse and sometimes overlap with those corresponding to gastroesophageal reflux.

In young children, vomiting or food refusal is prominent. When only one or two foods are associated, sometimes symptoms resolve in the first years of life.

The most often associated foods milk, eggs, peanuts, soy, wheat, nuts, fish and shellfish.

There can be weight loss and failure to thrive in children, while dysphagia and food impaction are more common in adults.

Chest or abdominal pain is also a clinical feature of this disease. Gastroesophageal reflux symptoms that may be present do not resolve with drugs that suppress acid production at gastric level.

The diagnosis needs to be confirmed with endoscopy and biopsies, whose histopathological exam show more than 15 eosinophils per field. The most common endoscopic findings are: linear esophageal creases, esophageal rings, whitish granulate and esophageal stenosis. The whitish plaques are associated to the presence of eosinophilic microabscesses and areas of great density of eosinophilic infiltrate [5]. Differential diagnosis includes gastroesophageal reflux disease, eosinophilic gastroenteritis, Crohn's disease, hypereosinophilic syndrome, infections and hypersensitivity response to drugs [6].

In summary, the criteria needed to diagnose eosinophilic esophagitis are symptoms of esophageal dysfunction, over 15 eosinophils per microscopic field, lack of response to Proton pump inhibitor therapy and normal pH monitorization in distal esophagus.

Our patient presented symptoms of esophageal dysfunction. The first high digestive endoscopy showed concentric stenosis in the esophagus with microabscessos eosinophilic, the patient was initially treated with the exclusion of cow's milk protein, Proton pump inhibitor and general measures for the control of asthma, but there was a lack of response with this treatment. When he started treatment with corticosteroids he became asymptomatic.

Depending on the individual case, the patient may be followed and treated jointly by digestive and allergy or immunology specialists. When certain foods are associated, a nutritionist should also be engaged.

There is no a curative therapy yet. Standardized protocols for the therapy don't exist.

The treatment is based on removing foods found positive in allergy

tests from the diet. Formulas based on amino acids can be used [7]. Once the symptoms have disappeared, a food challenge test may be implemented, introducing a food every 4-7 days to identify the responsible and guide the diet therapy in the long term.

Steroids can be administered topically. Fluticasone propionate is used in doses of 220 µg (2-4 puff swallowed every 12 hours) for 4 to 6 weeks, or beclomethasone.

Systemic corticosteroids may be required in some cases, which bring about a symptom improvement in one or two weeks. Methylprednisolone is given in doses of 0.5 to 1 mg/ kg/day (up to 16 mg/day) for 6 months, followed by gradual tapering [8].

Prednisone is given in doses of 30 mg/day for 2 weeks, followed by gradual tapering for 6 weeks [9]. It is difficult to establish the duration of the treatment for lack of studies.

Montelukast is another treatment option. Its use is associated with symptomatic improvement without histological changes. Early relapses have been observed after the end of the therapy [10].

Endoscopy is valuable to resolve food impaction in the esophagus or to dilate seriously stenotic esophagus. It is also useful for the follow-up of the disease.

In cases resistant to the aforementioned treatments, studies are being conducted with mepolizumab, an anti-IL-5 biological agent administered in 3 monthly infusions of 10 mg/kg (maximum 750 mg) doses.

Patients with dysphagia associated with allergic symptoms should be investigated for the possibility of EEO, an entity with rising prevalence in actuality.

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