CLINICAL VIGNETTE

Dysphagia in Eosinophilic Esophagitis: A Case Report

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Case Report

A 23-year-old male presented for a second opinion regarding ongoing reflux symptoms. He had been having symptoms of heartburn his "whole life." He described symptoms of reflux with substernal burning, which worsened with fasting and recumbency. He noted frequent episodes of dyphagia and occasional food "sticking" without significant changes to bowel movements. He had tried over the counter therapies including calcium carbonate, famotidine and currently was taking pantoprazole without significant improvement in his symptoms. He noted that dairy products worsened his symptoms and had eliminated this food group from his diet. He rarely used non-steroidal anti-inflammatory drugs and drank one cup of coffee daily. He drank alcohol two to three times a week. Past medical history was significant for asthma as a child and allergic rhinitis in the spring. Family history was significant for reflux in his father, as well as asthma in his sister and brother. He had previously been tested negative for H. Pylori. He had not undergone prior endoscopy. Other than the pantoprazole, he was not taking any medications. On examination, he had evidence of post nasal drip, with bilateral tympanic membrane scarring. He had epigastric tenderness to palpation without rebound or guarding. His stool was negative for occult blood. A complete blood count was normal, as was a comprehensive metabolic panel. Review of his previous history confirmed negative serum H. Pylori antibody testing.

The patient was referred to gastroenterology where he underwent esophagogastroduodenoscopy. This revealed esophageal rings. A biopsy was performed which showed an increased number of eosinophils. He was started on inhaled fluticasone twice daily with resolution of his symptoms.

Discussion

Eosinophilic esophagitis is disease mediated by antigens resulting in proliferation of esoinophils within the esophageal mucosa. The incidence of the disease is gradually increasing with most recent data showing an overall incidence of 55 cases per 100,000. The highest prevalence is among men with an average age of onset of 34 years[1].

The pathogenesis of eosinophilic esophagitis is thought to be the result of T cell immune recruitment of eosinophils, and there is a strong association between eosinophilic esophagitis and allergies, such as asthma and eczema. In addition, there are familial clusters of the disease, suggesting a genetic predisposition[2]. Once recruited to the esophageal mucosa, the inflammatory response results in esophageal irritation and the hallmark symptom of the disease, dysphagia.

The clinical presentation is very similar to that of gastroesophageal reflux. Patients complain of heartburn, epigastric pain and dysphagia. The severity of the dysphagia--often associated with food impaction--and the lack of response to antacids are indicators that a patient may be suffering from eosinophilic esophagitis rather than reflux[3]. Nonetheless, differentiating between the two disorders is difficult, resulting in the long average interval from symptom onset to diagnosis--over 4 years. The strongest associated symptom of eosinophilic esophagitis is food impaction, occurring in over 50% of patients[4].

The diagnosis is made through laboratory testing, endoscopic appearance, and biopsy. Serum levels of IgE and peripheral circulating eosinophils are elevated in the majority of cases of eosinophilic esophagitis. On endoscopy, certain characteristics are highly suggestive of the disease. These include a hypervascular subepithelium, linear furrows, white papules, and circumferential rings. On biopsy, histology shows a high concentration of eosinophils. It is important to remember, though, that the diagnosis can only be made in the setting of a positive biopsy and the typical clinical characteristics, as other disorders (such as reflux or Crohn's) can result in eosinophils being found on biopsy[5].

Treatment for eosinophilic esophagitis focuses on allergy elimination or mitigation of the atopic response. Patients should be referred for allergy testing for both environmental and food allergens[6]. Additionally, elimination diets have been shown to be effective in reducing symptoms. The mainstay of medical therapy is topical fluticasone. It is administered via inhaler and patients are instructed to spray directly into the mouth without inhaling, then swallow the medication. The patient should not drink for 30 minutes after administration. Therapy should be titrated to the lowest effective dose. The majority of patients will relapse after discontinuation of treatment, so ongoing therapy is recommended[7]. Proton pump inhibitors can also be used and are effective for symptom relief in a subset of patients. If patients are not responsive to PPI therapy after 4 weeks, it can be discontinued. It should be noted that antihistamines are ineffective in treatment of eosinophilic esophagitis. Lastly, in patients with severe dysphagia who have not responded to medical therapy, esophageal dilation is a treatment option. However, some studies have shown an increased risk of mucosal tears and possibly perforation[8].

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