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Case Report

An interesting case of dysphagia- eosinophilic esophagitis

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ABSTRACT

Eosinophilic esophagitis (EE) is an atopic condition of the esophagus that has become increasingly recognized over the last decade. It is associated with a variety of esophageal symptoms such as dysphagia, food impaction and chest pain. It has a predilection for white males between the age of 30 and 40 years of age. EE is diagnosed based on the combination of typical esophageal symptoms and esophageal mucosal biopsies demonstrating squamous epithelial eosinophil-predominant inflammation. Hereby we present a case of 65-year-old female who presented with dysphagia and gastritis for 12 months duration.

Keywords: Dysphagia, EE, Old female

INTRODUCTION

Eosinophilic esophagitis (EE) which is an inflammatory disorder of the esophagus, has become increasingly recognized over the last decade because of improvement in diagnostic procedures. This disorder is also known as primary EE, allergic EE and idiopathic EE.[1] It is characterized by dense eosinophilic infiltration in the esophageal mucosa with associated with dysphagia, chest pain, epigastric pain and food bolus impaction in adults, while failure to thrive and vomiting is more common in children.[2]

Eosinophils in the esophagus is always considered pathological since eosinophils are absent in the mucosa of healthy individuals.

EE is defined as a pathologic disorder characterized by >15 eosinophils per high power field (HPF) in one or more esophageal biopsy specimens and the absence of pathologic gastrointestinal reflux disease confirmed by a normal pH monitoring study or lack of response to acid-suppression therapy.[3]

Eosinophils that infiltrate the esophagus contribute to tissue damage and chronic inflammation. Studies suggest EE shares many clinical and pathophysiologic characteristics with allergies such as asthma.[4] The etiology of the disease is still poorly understood but the increasing number of recognized cases of EE has caused a dramatic interest and studies to comprehend the etiology of this disorder.

Patients with this condition often have a personal or family history of allergic conditions such as asthma, eczema, atopic dermatitis, seasonal allergies and food allergies.[5] EE is a disease associated with T helper cell (Th)-2 type immune response. The majority of patients with EE have positive skin prick tests, which detect IgE-mediated reactions, and atopy patch tests, which may identify non-IgE-mediated reactions, to foods and/or aeroallergens.[3]

CASE REPORT

A 65-year-old female came to the OPD with the complaints of dysphagia, more to solids than liquids for 1 year, burning sensation in the epigastrium especially after eating food since 1 year, which was associated with epigastric pain, loss of apetite since 3 months duration. The patient was treated before with high dose PPI's, but the symptoms did not improve. The patient is a known case of bronchial asthma since she was 15 years of age and was on

bronchodilators. Patient also has a known allergy to peanuts. Patient has positive family history of atopy as her mother also suffered from bronchial asthma.

Physical examination showed a blood pressure of 130/80 mmHg, pulse of 86 BPM, respiratory rate 18/min, and temperature 98.2°F. On abdominal examination, it was soft but tender on palpation in the epigastric region, without any guarding or organomegaly. Complete blood count of the patient was unremarkable and it did not show any eosinophilia.

The patient was admitted for further workup and upper video esophagogastroduodenoscopy (VOGD).

On VOGD, esophagus was found to have loss of vascular markings (edema), there were presence of multiple esophageal rings and longitudinally oriented furrows (Figure 1). Biopsy of multiple level of esophagus was taken for histopathological examination, which showed inflammation with marked infiltration of eosinophils (upto 60 /HPF) which was suggestive of EE (Figure 2).

After confirming the diagnosis the patient was treated with high dose PPI's as well as oral steroids. Patient was told to eat broths and clear soup. Patient was started on omeprazole and oral prednisone for 8 weeks and was asked for followup. After 6 months, the patient remains asymptomatic.

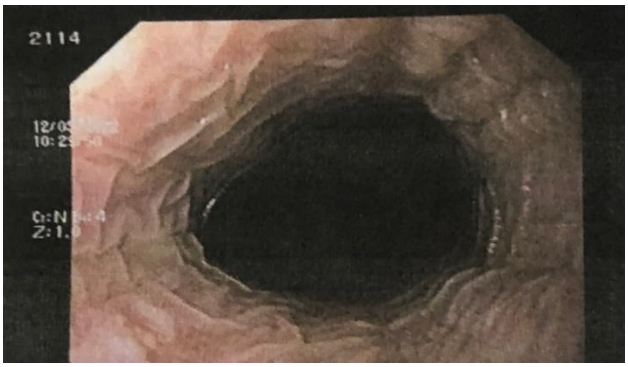

Figure 1: Showing multiple esophageal.

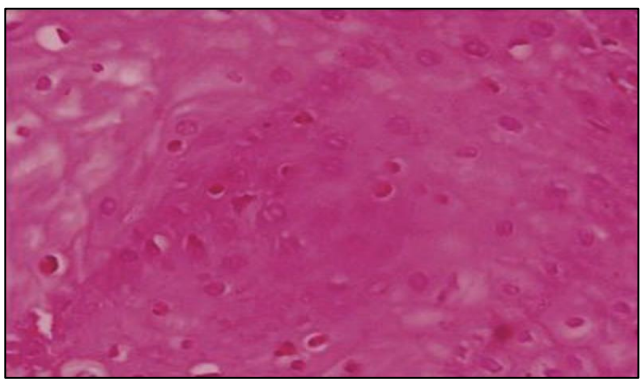

Figure 2: HPE of inflammation with marked infiltration of eosinophils.

DISCUSSION

The normal mucosa of esophagus does not contain eosinophils, so any eosinophils found in the esophagus can be considered pathological. Noel et al conducted a study of 103 patients, 0 to 19 years of age, who had EE. It reported a predominance of the male gender (71%). Half of them had histories of atopy, and 70% of them had family histories of atopy. Our patient had a personal and family history of atopy.

Personal histories of atopy are found in 60% of cases while family histories are found in 20% to 40% of cases.[6] Some patients with EE are asymptomatic and suspicion of the disease is based upon incidental findings at endoscopy that is performed for other indications. As a result, diagnosis for EE has been delayed in the past.[6]

There is a considerable overlap between GERD and EE. Therefore, trial of PPI's as well as esophageal pH testing has to be carried out before thinking about the possibility of EE. A study by Desai et al has found that EE was responsible for 50% of esophageal food impacts in outpatient visits.[7]

Endoscopy can aid in the diagnosis of EE but it is not a diagnostic tool. The characteristic endoscopically identified esophageal findings include loss of vascular markings (edema), multiple esophageal rings, longitudinally oriented furrows, and punctate exudate.

Endoscopic mucosal biopsy remains the most important diagnostic test for EE. Two or more biopsies are recommended regardless of the gross appearance of the mucosa, and specimens should be obtained from both the proximal and distal esophagus as well as areas showing endoscopic abnormalities in order to obtain high sensitivity for the detection of EE.[6]

The case of EE presented here was diagnosed in an older female which is quite unusual. Endoscopic as well as histopathological findings in our patient was consistent with the diagnosis of EE.

Treatment recommendations include diet restrictions, medications (systemic or topical steroids) and esophageal dilation. Diet restrictions such as avoiding egg, wheat, soy, cow's milk protein, seafood, peanuts or allergy testing such as skin prick test or atopy patch test have shown promising results.

Liacouras et al observed that the combination of steroids and diet significantly improved symptoms and histological findings, as in the case of our patient.[8] Teltelbaum et al and Konikoff et al recommend using steroids systemically or topically for four to six weeks.[9,10]

Endoscopic dilation is a treatment option for those patients with esophageal narrowing that do not respond to medication therapy.

More recently, therapies that act against specific substances identified within the inflammatory cascade have been proposed. These include mepolizumab, a humanized monoclonal antibody that blocks interleukin 5 (IL-5).[11]

CONCLUSION

EE consists of inflammation of the esophagus that is characterized by a large number of eosinophils in histological studies. It is an allergic disorder whose frequency is increasing in the population because knowledge about it has improved, and more people are developing allergic tendencies secondary to the environmental changes. Its pathophysiology is not known with certainty, and there is no definitive treatment. Even though the common presentation of EE is between 3rd and 4th decade of life in white male patient, our case was presented as an EE in an elderly female. Therefore, diagnostic suspicion should always be there in patient presenting with gastritis and dysphagia not getting relieved by PPI's.

There is also a considerable difference between treatment of GERD and EE, as the main treatment of EE lies in the admission of steroids whereas they are contraindicated in GERD. Therefore, it is very important to differentiate between the two.

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