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Review

Diagnostic approaches and treatment of eosinophilic esophagitis. A review article

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HIGHLIGHTS

- EoE is an eosinophilic influx into the esophagus epithelium.
- It is an allergic reaction of esophagus to food particles and allergens.
- Adults present with dysphagia and reflux-like symptoms whereas children with vague abdominal complaints.
- Clinical presentation, endoscopic findings and pathology determine the definitive diagnosis.
- Treatment starts with an elimination diet and followed by orally swallowed inhaled steroids.

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ABSTRACT

Eosinophilic Esophagitis (EoE) is a condition that involves eosinophilic influx into the esophageal epithelium. It affects both children and adults; Adults present with dysphagia whereas children with vague abdominal complaints. The clinical symptoms as well as pathologic features of EoE and gastro esophageal reflux disease (GERD) are similar. Since eosinophilia in the esophagus is a non-specific finding, the clinical presentation in conjunction with endoscopic findings and pathology, is crucial in determining a differential diagnosis. Infections such as parasites, allergic phenomenon, Crohn's disease, malignancies, medication, and chemotherapy are all associated with eosinophilia.

A primary endoscopic difference to note between EoE and GERD is that EoE often involves long segments of the esophagus, could be patchy or focal and frequently involves the proximal esophagus. GERD, however, typically involves the distal much more frequently than the proximal esophagus. Because of the similarity between them, GERD should be excluded by using high dose proton pump inhibitor (PPI) treatment or through evidence of a normal pH by esophageal testing, prior to treatment with an elimination diet or steroids. Until further research establishes different diagnostic tests and criteria, clinical and pathological response to therapy is considered to be the absolute confirmation of this diagnosis. The following is a more detailed discussion of this entity.

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1. Introduction

Forms of esophagitis have been recognized for hundreds of millennia. The Sumerians and Greeks have words describing this phenomenon. The romans used calcium carbonate for its treatment [1]. From the mix of milk, cream and antacids taken before the availability of histamine blockers in the mid-70s, to the later appearance of PPIs in the late 80s, more and more treatments have become common use for this entity. In an unusual chain of events in medicine, however, our real knowledge of the phenomenon was acquired after treatments were readily available, mostly due to fiber optic advances and wide use of endoscopy and biopsy of the esophagus which revealed pathology well beyond what was known beforehand.

Today with the advances in food technologies and widespread availability of cheap processed food products (possibly containing new and improved allergens), esophageal disease is more and more prevalent. Up to 37% of certain populations can suffer from esophageal symptoms on a weekly basis [2].

Inflammatory diseases of the esophagus are numerous and may include: infections such as Candida; allergic responses involving an eosinophilic infiltrate; medication-induced, for example Bisphosphonates; reflux-induced; infiltration by lymphocytes; and bullous skin disease extensions into the esophagus. This review will focus on eosinophilic esophagitis, which is a diagnosis made more and more in clinical practice. It is important to remember that EoE is different from eosinophils in the esophagus, which is a non-specific pattern that can happen because of a variety of non-connected etiologies.

2. Definition

EoE is a chronic immune/antigen-mediated esophageal disease characterized clinically by symptoms related to esophageal dysfunction and histologically by eosinophil-predominant inflammation [3]. EoE is used for abbreviation of eosinophilic esophagitis since erosive esophagitis is abbreviated as EE. There should be gastrointestinal eosinophilia limited to esophagus causing characteristic symptoms, while other etiologies for eosinophilia have been ruled out, for physicians to refer to it as EoE. Overall the pathogenesis involves genetic, environmental and host immune system factors interacting as will be discussed.

3. History and epidemiology

There are reports from the 60s and 70s that describe entities that could have possibly been cases of EoE. These patients with multiple esophageal rings were classified as GERD complications based on their biopsies showing basal zone hyperplasia, papillary lengthening and intraepithelial eosinophils [4]. However, these are not common findings in GERD. The response to acid suppression therapy was nowhere as successful as GERD in these patients. Severe complications such as deep mucosal tears or occasional perforations usually followed [5]. Interestingly, these cases were considered to be “extreme” GERD cases and end of GERD spectrum. The true pathological identification of the entity happened in the 90s and separated it from GERD.

EoE has been reported everywhere in the world other than the African continent, albeit there are regional variations depending on socioeconomic and climate zones. Lower socioeconomic populations in cold and arid areas seem to be more prone to EoE. The incidence of EoE is increasing, undeniably from widespread knowledge of the entity, but also because cheap, processed, modified food is universally accessible and abundantly overused. Prevalence has been estimated to be about 12.8–55 per 100,000 [6] with most of, many of affected adults being men in their 20s and 30s. EoE is predominantly associated with food and environmental allergies, asthma, atopy and celiac disease.

4. Genetics

Family clustering has been noted in some 30 families with EoE [7]. A positive family history can be found in at least 7% of the affected. There is a strong sibling ratio and the risk is 50-fold for siblings compared to general population. The risk is even higher in patients with advanced stenotic EoE.

Several genetic markers have been identified. Although some are common genetic variants, others appear to be specific for EoE. Eotaxin-3, 5q22 (involving gene encoding Thymic stromal lymphopoietin or TSLP), filaggrin gene, transforming growth factor or TGF receptors have been associated with EoE [8]. The earliest works revealed dysregulated expression of 1% of the genome constituting an EoE genetic signature. RNA sequencing on esophageal biopsies showed long-noncoding RNAs. EoE susceptibility locus was also found at 5q22 (related to TSLP coding) and 2p23 (related to a protein expressed in esophagus). 2p23 is upregulated in EoE and induced by IL-13 in esophageal epithelial cells.

Although these are helpful clues in our understanding of the disease, they really don't give us an exact insight into why this phenomenon is happening. Many of these abnormalities have been reported in other pathologies as well.

A 96-gene diagnostic panel based on a study of EoE biopsies and genetics have been produced and is commercially available. It is in effect a genetic testing for EoE molecular transcriptome and might