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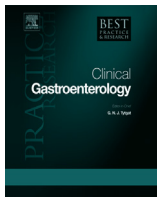

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Esophageal dilation in eosinophilic esophagitis

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A B S T R A C T

Tissue remodeling with scaring is common in adult EoE patients with long standing disease. This is the major factor contributing to their complaints of solid food dysphagia and recurrent food impactions. The best tests to define the degree of remodeling are barium esophagram, high resolution manometry and endoscopy. Many physicians are fearful to dilate EoE patients because of concerns about mucosal tears and perforations. However, multiple recent case series attest to the safety of esophageal dilation and its efficacy with many patients having symptom relief for an average of two years. This chapter will review the sordid history of esophageal dilation in EoE patients and outline how to perform this procedure safely. The key is graduated dilation over one to several sessions to a diameter of 15–18 mm. Postprocedural pain is to be expected and mucosal tears are a sign of successful dilation, not complications. In some healthy adults, occasional dilation may be preferred to regular use of medications or restricted diets. This approach is now supported by recent EoE consensus statements and societal guidelines.
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Introduction

Eosinophilic esophagitis (EoE) is a disease of increasing prevalence and detection [1]. All patients must have greater than or equal to 15 eosinophils/hpf on their esophageal biopsies. The disease in

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children is more of an inflammatory process with common symptoms including failure to thrive, vomiting, abdominal pain and heartburn. Food elimination diets and steroids (both oral and swallowed) effectively treat these complaints by controlling the underlying inflammatory process [1]. In adolescents and adults, the symptoms are primarily solid food dysphagia, odynophagia, and heartburn that can be associated with the fearful complication of food impaction with or without esophageal strictures [2,3]. The more severe the stricture disease, the longer duration of untreated disease suggesting an evolution from pure inflammation to fibrosis, strictures and generalized esophageal narrowing [4,5]. Elimination diets and steroids in adults with EoE are not as successful in treating symptoms as the childhood variant, probably due to the degree and severity of underlying fibrostenotic disease [1]. As with other esophageal strictures such as gastroesophageal reflux disease (GERD), esophageal dilation in EoE patients can markedly improve symptoms for prolonged durations. However, unlike reflux strictures, dilation is commonly associated with pain and sometimes deep tears requiring hospitalization but rarely surgery. This conflicting "risk–benefit" profile for esophageal dilation in treating EoE has resulted in confusing recommendations from the two international consensus reports on EoE [6,7] and reluctance in the GI community for employing this treatment more routinely in clinical practice.

This chapter will review the pathophysiology of esophageal fibrosis and remodeling in EoE and potential diagnostic tests. The history of EoE and esophageal dilation will be summarized from the "dark ages" before 2010 to the new "renaissance" period we are embarking upon. Unfortunately, all of the data is based on case series and personal experience as no comparative studies between dilation and anti-inflammatory drugs/diet are available. In the latter category, I will rely on my experience dilating patients with EoE since the early 1990s and the first case series on esophageal dilation in EoE published in 2001 from our group at the Cleveland Clinic [8].

Esophageal remodeling

The concept of eosinophil-associated tissue remodeling stems from diseases such as asthma and the hypereosinophilic syndrome. Remodeling is defined as tissue changes in target organs which result in organ dysfunction [9]. Remodeling is associated with histologic alterations, such as fibrosis and angiogenesis and may be beneficial or detrimental to the target organ. For example, it can be protective when part of wound healing, but when uncontrolled, due to ongoing inflammation, can have negative consequences to organ function [9]. In EoE, remodeling changes include epithelial basal cell hyperplasia, lamina propria fibrosis, expansion of the muscularis propria and increased vascularity. These tissues changes in combination are the likely mechanisms for EoE associated complications including dysphagia, strictures, food impactions, esophageal rigidity and dysmotility.

Pathogenesis of EoE remodeling

Inflammatory mediators and cells clearly play a role in driving esophageal remodeling. Most of the experimental data is obtained from animal studies, especially in the mouse as human tissue is usually confined to superficial biopsies and rarely do we have full thickness specimens. The pathogenesis of EoE and remodeling is complex and will only be briefly summarized in this chapter. Recommended readings are by S. Aceves and colleagues [9,10] with Fig. 1 summarizing the key interleukins and cytokines with their clinical consequences.

Mice lacking eosinophils or the eosinophilic cytokine interleukin (IL)-5 have much less collagen deposition and fibronectin expression than their wild type littermates [11]. Over expression of IL-13 causes esophageal stricture that is not reversible by subsequent removal of IL-13 [12]. IL-13 is the master regulator in EoE, functioning to increase both IL-5 and eotaxin -3 contributing to basal cell hyperplasia, muscle hypertrophy as well as fibrosis (Fig. 1) [13].

Esophageal eosinophils in EoE produce the profibrotic factor, transforming growth factor beta-1 (TGFB1) which increases the production of collagen, fibronectin and other extracellular matrix proteins [9,14]. Other profibrotic molecules, including CC chemokine ligand 18 (CCL-18) and fibroblast growth factor 9 (FGF-9) are increased in EoE patients and may have TGFB1 independent pathways to

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