Techniques in Gastrointestinal Endoscopy 16 (2014) 26–31

Contents lists available at ScienceDirect

Techniques in Gastrointestinal Endoscopy

journal homepage: www.techgiendoscopy.com/locate/tgie

Esophageal dilation in eosinophilic esophagitis

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ARTICLE INFO

Article history:
Received 12 July 2013
Received in revised form
20 August 2013
Accepted 21 August 2013

Keywords:
Esophageal stricture
Dysphagia
Food impaction
Esophageal dilation

ABSTRACT

Solid-food dysphagia and food impaction are the hallmark symptoms of eosinophilic esophagitis (EoE) and are a result of subepithelial fibrosis, leading to esophageal stricture formation and loss of compliance of the esophagus. This fibrosis can be mechanically disrupted by esophageal dilation, which leads to a significant improvement in dysphagia in more than 80% of patients. The esophageal mucosa is quite fragile in patients with EoE, and dilation frequently leads to deep mucosal tears. The risk of esophageal perforation with dilation in EoE is likely increased over that seen with dilation in other benign esophageal diseases, but this risk has been overestimated in the earlier literature. Dilation in EoE, when done with some precaution, can be done relatively safely and in many cases of small-caliber esophagus, it needs to be done to alleviate symptoms. However, this dilation is frequently associated with postprocedure chest pain. In this article, we discuss the mechanisms of dysphagia in EoE to better understand the indications and timing of esophageal dilation in this disease. Then, we discuss the technique, efficacy, and importantly, the safety of esophageal dilation in EoE.

1. Introduction

The hallmark symptom of eosinophilic esophagitis (EoE) in adult patients is solid-food dysphagia. Food impaction, defined as food stuck in the esophagus for more than 5 minutes, occurs in 57% of adult patients with eosinophilic esophageal infiltration [1] and up to 35% of patients with EoE will require endoscopic food-impaction removal [2].

Eosinophils in the esophageal mucosa can degranulate and secrete multiple proteins. These proteins have been shown to lead to esophageal remodeling, resulting in subepithelial fibrosis [3,4]. This fibrosis leads to esophageal stricture formation and loss of wall compliance, thereby resulting in the symptoms of solid-food dysphagia and food impaction [5]. This fibrosis has been treated for some time with esophageal dilation, which has been associated with relief of dysphagia [6,7]. The early literature highlighted the increased risk of endoscopy and esophageal dilation in patients with EoE [8-11]. These reports of frequent deep mucosal tears and cases of esophageal perforation led to a fear of esophageal dilation in EoE. More recent studies have suggested that dilation can be done safely in experienced centers [6,7,12,13].

In this article, we discuss the mechanisms of dysphagia in EoE to better understand the indications and timing of esophageal dilation in this disease. Then, we discuss the technique, efficacy, and importantly, the safety of esophageal dilation in EoE.

2. Mechanism of dysphagia

An understanding of the mechanisms of dysphagia in EoE allows us to better determine the indications for dilation in this patient group. There are likely several mechanisms for dysphagia in EoE.

(1) *Esophageal stricture*: it is well accepted that esophageal remodeling can lead to the formation of fixed rings or longer esophageal strictures. Food will stick when the size of the bolus exceeds the size of the esophageal lumen. Older studies have suggested that an esophageal lumen having a diameter less than 13 mm is frequently associated with solid-food dysphagia [14]. However, that study suggested that larger boluses of food may stick at esophageal diameters up to 20 mm. Breads and meats are foods often associated with dysphagia in EoE and likely related to stricture formation.
(2) *Loss of compliance*: esophageal remodeling can be associated with loss of compliance and loss of distensibility of the esophagus. This has been shown by Kwiatek et al [6] using the Endoflip device. The Endoflip is a balloon that can be passed into the esophagus and measures balloon pressure, volume, and surface area. Esophageal compliance and distensibility can be calculated. These studies show not only a narrowing of the esophagus in EoE but also a marked decrease

Conflict of interest statement: The author is a consultant for Meritage Pharmacia and Aptalis Pharma. He has research funding from Merck, Meritage Pharmacia, and Aptalis Pharma.

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0049-0172/$ - see front matter © 2014 Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1016/j.tgie.2013.08.009

in compliance in most patients with EoE. Therefore, loss of compliance in the esophagus, in addition to stricture formation, can be a second mechanism that would cause dysphagia with bulky foods, such as bread and meats.

(3) *Mucosal factor*: we suspect that there is also a mucosal factor involved in EoE dysphagia that makes the esophagus sticky or rough. We have measured the esophageal diameter with a structured esophagram in patients with EoE and have found the maximal (as a measure of distensibility) and minimal esophageal diameters to be normal in one-half of patients with symptomatic EoE [15]. Moreover, the minimal diameter was less than 13 mm in fewer than 20% of these patients, thereby suggesting a mechanism other than stricture formation or lack of distensibility in EoE dysphagia. Lastly, the clinical response of dysphagia to treatment with topical steroid is often seen within few days; an interval too short to reverse fibrosis.

(4) *Pill esophagitis*: a secondary pill esophagitis may develop in patients with EoE-related dysphagia as a result of the mechanisms mentioned earlier and lead to a localized esophageal stricture.

Theoretically, esophageal dilation would benefit those with significant esophageal strictures as well as those with fibrosis-induced loss of compliance. How esophageal dilation would benefit those with a normal esophageal diameter and mucosal stickiness or irregularity is less obvious. In reality, multiple, if not all of these, mechanisms are involved in patients with EoE.

3. Indications for dilation

The role of esophageal dilation in EoE is controversial. Dilation will treat the fibrosis of the esophageal wall in EoE, but not the eosinophilia. Most physicians believe that the symptoms and complications of EoE are a direct result of protein products released during degranulation from the activated eosinophil. There are 2 major schools of thought on the long-term treatment of EoE.

3.1. Eosinophilia needs long-term suppression

The eosinophil products stimulate fibrosis, which can lead with time to strictures and small-caliber esophagus, causing symptomatic dysphagia as well as food impaction. Food impaction is not an uncommon occurrence developing in 35% of patients with EoE over 18 years [2]. This can be an unpleasant experience, can require emergent endoscopic removal, and can be associated with esophageal perforation due to either retching or the endoscopic procedure [16,17,2]. In a older series, perforation occurred in 1.5% of patients undergoing endoscopic food-impaction removal [2]. Investigators anxious to avoid these potential complications feel all patients with EoE require long-term treatment with medical or dietary therapy to eliminate significant esophageal eosinophilia. The role of esophageal dilation with this approach is to treat the fibrotic component of dysphagia. In other words, esophageal dilation would be limited to those patients with persistent dysphagia despite resolution of esophageal eosinophilia with medical or dietary therapy, that is, after the elimination of the inflammatory component that might contribute to dysphagia. Notably, the contribution of inflammation to dysphagia is not insignificant. In one series, dysphagia resolved with treatment of proton-pump inhibitor or topical steroid in 55 (89%) of 62 patients with symptomatic esophageal eosinophilic infiltration [18]. Of these patients, 11% had persistent dysphagia despite medical treatment and resolution of esophageal eosinophilia. Ten of these patients had symptomatic response to dilation of fibrotic esophageal strictures. Moreover, all of these patients had a minimal esophageal diameter of <14 mm on structured esophagram, suggesting diffuse fibrotic disease. In this regard, it might be reasonable to dilate patients with a markedly narrowed esophagus, such as <10 smm, at the initial endoscopy before anti-inflammatory therapy, as a patient with this degree of fibrostenotic disease is unlikely to respond to anti-inflammatory therapy alone.

3.2. Eosinophilia does not necessarily need long-term treatment

With this strategy, patients with prolonged response to initial dilation could be treated with periodic dilation between relatively long intervals of symptomatic remission. Indeed, a study found the dysphagia-free interval to be the same between patients undergoing dilation and medical therapy compared with dilation alone [6]. We surmise that although dilation does not treat the underlying etiology inflammatory component of EoE, there is likely a subset of patients with slowly progressive disease and without an advanced-stage fibrotic esophagus in which dilation is just as efficacious as medical therapy without the risks of the latter. This school of thought would support only treating patients who are symptomatic or those with frequent symptomatic recurrences when off medical or dietary therapy. The supporters of this school of thought would support intermittent steroid therapy or intermittent esophageal dilation or both. This approach uses esophageal dilation as a primary treatment of EoE, and not just a rescue therapy for medical failures. It is important to note that in patients with EoE, dilation may be treating both compromises in esophageal lumen and compliance, some might view the latter as diffuse but incomplete stricture formation.

4. Technique

4.1. Which dilator to use?

The 5 largest studies on esophageal dilation in EoE are shown in Table 1. Through the scope balloon dilators (TTS), over the wire Savary dilators (OTW), and unguided Maloney bougie dilators were used. The vast majority of procedures in these studies were performed with TTS or OTW dilators. As they are rarely used and are theoretically risky in patients with EoE, I will not discuss unguided bougie dilators further. In general, several published trials comparing TTS and OTW dilators have found no major differences in efficacy or safety between the 2 techniques for dilation of various esophageal stricturing diseases [19-21]. It is noteworthy that these studies mentioned earlier did not evaluate the use of these dilators specifically in patients with EoE, but as esophageal strictures may represent a common end point of diverse etiologies, these studies suggest that the same principles may apply to patients with EoE. From studies specifically involving patients with EoE listed in Table 1, no differences in efficacy were demonstrated in using TTW vs OTW dilators. However, 2 studies commented on differences in safety between TTS and OTW dilators. Jung et al [13] found the use of OTW dilators to be associated with over a twofold increased risk of complication over the use of TTS dilators. In contrast to this, Dellon et al [12] found all complications in their study (7% of patients) to have occurred in patients undergoing TTS dilation (used in 83% of their procedures). This complication data are somewhat difficult to interpret because the most common complication in these studies was a deep mucosal tear, which many would argue is a desired outcome of successful dilation. It is noteoworthy that in the Jung trial, the greatest risk of complication involved the dilation of a stricture in the proximal location in the esophagus. This is a significant confounding variable, as proximal esophageal strictures are more likely to be treated with an OTW dilator rather than a TTS dilator. It is also likely that longer and more complicated strictures would be treated with Savary dilators. In contrast to visualized luminal tears,