We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,300

Open access books available

172,000

International authors and editors

190M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Endoscopic Aspects of Eosinophilic Esophagitis: From Diagnosis to Therapy

Alfredo J. Lucendo¹ and Susana Jiménez-Contreras²

¹*Department of Gastroenterology, Hospital General de Tomelloso*

²*Department of Gastroenterology, Hospital Virgen de Altagracia
Spain*

1. Introduction

Eosinophilic esophagitis (EoE) represents a chronic, immune/ antigen mediated disease characterized by esophageal dysfunction and eosinophilic inflammation (Liacouras et al., 2011). The past few years have witnessed a progressive rise in diagnosed cases of EoE, which has become the second most common chronic esophageal disease after gastroesophageal reflux (Lucendo, 2010). In spite of this, EoE remains underdiagnosed in many cases, especially because endoscopic findings are usually much harder to detect than those observed in esophageal growths (such as neoplasms) or erosive disorders. A great variety of endoscopic findings has been described in literature for EoE patient, including an apparently normal esophagus, which suggests that changes in this organ's appearance are not only complex, but also subtle enough to be overlooked by an endoscopist unaccustomed to diagnosing this disease.

At the same time, research efforts aimed at providing efficient therapy for this chronic illness has also intensified. Unfortunately, no treatment strategies have been commonly accepted to date, making adequate management of these patients somewhat controversial (González-Castillo et al., 2010). That being said, 3 different therapeutic approaches have been used effectively in patients with EoE. The first approach involves endoscopic dilation, a technique which is frequently able to solve alterations in the caliber of the esophagus, including a narrowing of the lumen (Schoepfer et al., 2009). From the earliest documented cases, mechanical dilation has been used as a treatment option for EoE, similar to the way it is used in other cases of fibrous esophageal stenosis, such as peptic stenosis or following caustication. The classification of EoE as an immunoallergic disorder has led to a second approach, namely that of treating patients with drugs for bronchial asthma (Furuta & Straumann, 2006). However, because no specifically approved drugs are currently available for EoE patients, these treatment must be due out label.

From the first studies performed on children with EoE, allergies to certain dietary components have been demonstrated to contribute significantly to its pathogenesis; indeed, it is well-documented that both the symptoms of the disease and histology levels improve after eliminating certain foods from the diet (Liacouras et al., 2005). However, while early studies based exclusively on elemental diets showed enormous efficacy in reverting EoE (Kelly et al., 1995), this approach is not plausible in adults or chronic patients.

One important stumbling block to determining the most effective treatment for EoE is the lack of studies directly comparing different treatment strategies for the disease. Such studies will be necessary before the best therapeutic option for EoE can be established.

In this chapter we review the various endoscopic lesions described in EoE to date. This should help relatively inexperienced endoscopists screen for patients suspected of having EoE. We will also discuss the effects and risks of endoscopic treatment by dilation in EoE patients by reviewing the current literature.

2. Diagnosis

EoE is a clinico-pathological disease characterized by symptoms related to esophageal dysfunction. Up to now, esophageal biopsies have been essential for making a diagnosis. For optimal pathological evaluation, multiple biopsies from the proximal and distal esophagus should be obtained and evaluated for a variety of pathological features, the most characteristic being an eosinophil-predominant inflammation with a minimum threshold of 15 eosinophils/ high power field (hpf). However other accompanying findings reinforce the diagnosis and should also be noted by the pathologist. These include: eosinophilic microabscesses, surface layering of eosinophils, extracellular eosinophilic deposits, basal cell hyperplasia, intercellular edema, and lamina propria fibrosis (Furuta et al., 2007).

The effects of EoE are isolated to the esophagus; therefore, eosinophilic inflammation should be absent from both gastric and duodenal biopsy samples (Lucendo, 2010). Furthermore, other causes of esophageal eosinophilia should be excluded, specifically gastroesophageal reflux disease (GERD). This can effectively be excluded if there is a normal pH-metry or if eosinophils persist after treatment with full doses of proton pump inhibitors (PPI). However, the prevalence of patients suffering from both EoE and GERD make the PPI trial the method of choice for diagnosing EoE in these cases (Molina-Infante et al., 2009).

3. Endoscopic findings

The fact that EoE was first identified only 30 years ago is indicative of the frequently subtle and unspecific endoscopic finding present in most patients. In fact, common esophageal diseases causing dysphagia are usually characterized by evident endoscopic lesions, such as peptic erosions, ulcers, protruding masses or stenosis, all of which contrast with the relatively minor findings exhibited by the majority of patients with EoE. Successful diagnosis of the disease thus requires a high level of suspicion on the part of the clinician, who should perform a careful examination of the esophagus accompanied by mucosal biopsies, even if the mucosa appears to be normal.

Many patients diagnosed with EoE have had previous endoscopies for dysphagia or food impaction and received different diagnoses. In fact, one study reported that the average adult EoE patient underwent two endoscopic exams before being diagnosed correctly (Lucendo et al., 2007). Esophageal symptoms in these patients are frequently attributed to various causes, with some reported cases receiving referrals to mental health professionals because psychological rather than physiological disorders were suspected.

Only in past few years has EoE been extensively recognized, leaving behind its status as a broadly misdiagnosed disease (Gonsalves et al., 2005) to become the second cause of chronic esophagitis.

Endoscopy with esophageal biopsy remains the only reliable diagnostic test for EoE. Consequently, in order for clinicians to recognize the disease more easily, a better awareness of the distinct endoscopic features of EoE is essential. Retrospective re-evaluations of the endoscopic appearance of the esophagus in those patients eventually diagnosed with EoE have revealed that esophageal appearance had been described as normal in between one quarter to one third of the cases (Müller et al., 2007; Sgouros et al., 2006; Liacouras et al., 2005). It is important to note, however, that even though the endoscopic findings are subtle, remarkable abnormalities can still be detected in the majority of patients, as we describe below.

Endoscopy has helped identify a great number of esophageal abnormalities in patients with EoE. These include fixed esophageal rings that sometimes reduce the esophageal lumen (a phenomenon known as trachealization) and transient esophageal rings (also called feline folds or felinization). Diffuse nodularity/ granularity of the mucosa has also been described, along with widespread exudative mucosal lesions, either in the form of whitish papulae of varying sizes clustered together (white spots) or as large, white, exudative fibrinoid lesions. These whitish lesions on the mucosa resemble a mild, superficial *Candida* infection, but histopathology shows micro-abscesses made up of eosinophils (Lucendo et al., 2007). Furthermore, a loss of the common vascular pattern of the mucosa has been described (Lucendo, 2007; Straumann et al., 2004). Some of the most common findings are longitudinal furrows (referred to as “corrugated esophagus,” which is an architectural analogy to a grooved column) (Straumann et al., 2004), diffuse esophageal narrowing, and esophageal lacerations induced by passage of the endoscope. Mucosal fragility, also called *crêpe-paper* mucosa (Straumann et al., 2003), is an important feature of this pathology as it may cause tears during upper endoscopy or even if the patient tries to dislodge impacted food by inducing vomiting (Lucendo et al., 2011). However, because all of these endoscopic features have been described in other esophageal disorders, none can be considered pathognomonic for EoE.

To shed light on the varied endoscopic appearances of EoE, we have classified them according to two independent yet complementary aspects: alterations in the caliber of the esophagus and alterations in the appearance of the mucosa (Lucendo et al., 2007).

- Alterations in the caliber of the esophagus, which appear as a consequence of motor esophageal disturbances associated to EoE in children (Nurko et al., 2009) and adults (Moawad, 2011; Lucendo et al., 2007), or after fibrous remodeling of the organ. In this case, multiple simultaneous contraction rings may be observed; these may block the passage of the endoscope while still permitting observation of the distal lumen. Alternatively, the clinician may notice regular concentric strictures, which impede both passage of the endoscope and observation of the distal mucosa (Lucendo et al., 2007). The smaller caliber of the esophagus may go unnoticed in barium contrast radiography and endoscopy (Vasilopoulos et al., 2002). All of these changes may occur without mucosal lesions (e.g. erosions or ulcerations), unlike what happens in peptic disease. Alterations in the caliber are found predominantly in the mid and distal esophageal thirds and can be reverted with treatment; in fact, since motor disturbances can be successfully treated with topical steroid treatment, a functional rather than structural origin of caliber alterations in EoE should be considered.
- Alteration in the appearance of the mucosa. In a study analyzing different endoscopic findings associated with EoE in parallel with the intensity of histological lesions,

density of eosinophils, and cell activation as determined with the aid of immunostaining for Major Basic Protein (MBP) (Lucendo et al., 2007), it was observed that the density of eosinophils increased with the severity of histological changes. Qualitative analysis of the patient biopsies showed a correlation between the intensity of histopathological changes and the diverse patterns of findings from endoscopic exploration of the mucosa. Consequently, four endoscopic-histopathological patterns were defined: 1. Granular pattern: mucosa with relatively defined papular elevations that give it an irregular shape. Histological analysis highlighted changes in eosinophilic infiltration and derived damage, with different intensities in different areas, which implies possible mucosal effects that are not uniform in intensity. 2. Corrugated pattern: linear longitudinal ridges or striae along the folds of the esophagus also affected by mucosal edema. The histology identified edema with growth in intercellular spaces between the epithelial cells, ballooned cells, and spongiosis. 3. Undulated pattern: this may denote contraction of the muscularis mucosae (not evaluable in endoscopic biopsies). It should not be mistaken for simultaneous contraction rings in the internal or circumferential layer of the esophageal muscularis propria, which in this case reduce the size of the lumen. 4. Exudative pattern: different-sized whitish lesions (from slight spotting to squamous lesions), creating epithelial clusters or microabscesses containing eosinophils. These patients had a high density of eosinophils on the surface of the esophagus, destruction and detachment of the most superficial strata, and more intense immunostaining for MBP.

Fig. 1. Images from two patients with eosinophilic esophagitis and spontaneous esophageal tearing, with ring disruption. This occurred as a result of the efforts the patients made to induce vomiting and dislodge impacted food

Several prospective studies have evaluated the utility of endoscopic findings for diagnosing EoE. In 2007, G.A. Prasad and co-workers successfully used endoscopy in conjunction with esophageal biopsies to diagnosis EoE in 15% of 222 patients who were being attended for non-obstructive dysphagia (Prasad et al., 2007). Of the 21 patients who exhibited endoscopic results characteristic of EoE, the diagnosis was confirmed in only 8 cases (38%). However, 10 of the 102 patients (9,8%) with an apparently normal endoscopic examination presented histological evidence of EoE. In 2008, S.H. Mackenzie et al. reported similar findings (Mackenzie et al., 2008). Thus, while 12% of the 261 patients suffering from dysphagia who underwent endoscopy were initially diagnosed with EoE, only 12 of 35 patients (34%) who showed esophageal rings in their endoscopic exams were confirmed to have EoE after esophageal biopsy.

Fig. 2. Several endoscopic aspects of eosinophilic esophagitis: a: Normal-caliber esophagus with a normal appearance mucosal surface; b: Fragile-looking mucosa, with irregular surface and whitish exudates; c: Reduced-caliber, trachealized esophagus with regular mucosal surface, which allows the passage of the endoscope; d: Longitudinal linear furrows and irregular mucosa; e: The esophageal mucosal surface may be covered in cotton-like exudates mimicking candidiasis, but biopsy finds them to be multiple eosinophil-containing micro-abscesses; f: Ringed esophagus with stenosis blocking the passage of the endoscope

In any case, EoE seems to be a very common cause of dysphagia, with a prevalence of up to 22% in patients with the non-obstructive version of this condition (Ricker, 2001). In addition, its incidence rates are significantly higher in men than in women and also in those of European descent than for other ethnicities. These findings underscore the importance of performing routine biopsies to screen for EoE in these patients (Ricker, 2011).

Because the reliability of endoscopic findings alone for diagnosing EoE does not appear to exceed 40%, few studies deal with finding ways to improve the diagnostic efficiency of endoscopy. Indeed, only one published study has examined the ability of narrow-band imaging (NBI) endoscopy to improve reliability. While this technique proved helpful in detecting mucosal details that go unnoticed in a routine white-light examination, it only managed to identify rings and furrows with fair to good reliability; no other findings were noted. Moreover, there was also great interobserver variability. The researchers thus concluded that endoscopic findings alone were not sufficiently reliable for supporting a diagnosis of EoE or for making treatment decisions (Peery, 2011).

As we have seen, none of the endoscopic features described above is pathognomonic for EoE; however, the presence of more than one of them in a given patient bolsters the case for a diagnosis of EoE. It is our hope that a greater awareness of these subtle characteristics will help clinicians avoid overlooking them to more accurately diagnose patients. Of course, any preliminary diagnosis must then be confirmed through biopsies. Indeed, as we emphasized above, biopsy sampling should also be performed in cases of non-obstructive dysphagia, even when the esophagus appears normal.

4. Endoscopic treatment for Eosinophilic Esophagitis (EoE)

Since the first descriptions of EoE appeared in the literature, the disease has been associated with alterations in the caliber of the esophagus, which specialists have sought to correct by means of endoscopic dilation. In this sense, endoscopic therapy has always been recognized as one of the main treatment modalities in EoE patients, together with systemic and topical steroids and changes in diet.

The efficacy of endoscopic treatment in EoE patients is clear in emergency situations, in which it is needed to resolve food impactions that block the esophageal lumen, and also in scheduled explorations of patients with esophageal symptoms, especially if these are accompanied by a reduced esophageal caliber. The characteristic fragility of the esophageal wall in these patients initially led several authors to consider endoscopic techniques to be a risky treatment option (Lucendo, 2007). We will discuss this assertion in greater depth after reviewing new evidence from the latest studies.

4.1 Emergency endoscopy and food desimpaction

The impaction of food in the esophagus is common in EoE patients and is, together with dysphagia, the clinical hallmark of the disease. Additionally, food impaction is the clinical manifestation which most frequently leads to diagnosis of EoE in adult patients, constituting a complication that must be urgently remedied. In this manner, 43.3% of the 30 adult patients studied in a Spanish series underwent endoscopy as an emergency treatment to resolve food impaction before being diagnosed with EoE (Lucendo et al., 2007). Furthermore, an analysis of 251 Swiss patients with EoE showed that 34.7% required extraction of the impacted bolus with the aid of flexible or rigid esophagoscopy, with the latter causing a 20% rate of transmural perforations (Straumann et al., 2008). Bolus removal by means of rigid endoscopy thus constitutes a high-risk procedure and should be avoided in EoE patients. Food impaction in pediatric forms of EoE seems to be uncommon, with no definitive explanation for this difference.

4.2 Dilation treatment for EoE

From the earliest documented cases, mechanical dilation with through-the-scope hydropneumatic balloons or Savary bougies has been employed as a treatment option for EoE, similar to the way it is used in other cases of rigid or fibrous esophageal stenosis resulting from the cicatrization of prolonged esophageal inflammatory processes such as GERD or after the ingestion of caustic substances. The chronic inflammatory phenomena which characterize EoE seem to cause subepithelial collagen deposition and fibrous remodeling, as recently shown in both childhood (Chehade et al., 2007; Aceves et al., 2007) and adult (Straumann et al., 2010, 2011) forms of the disease as well as in animal models (Mishra et al., 2008). Also, in recent years, various studies have addressed the relationship between EoE and GERD (Spechler et al., 2007), proving that both diseases can coexist in the same patient, causing dysmotility of the distal third of the esophagus, poor acid clearance, and the possibility of lesions – particularly Schatzki rings (Nurko et al., 2004) – from reflux. Several aspects should be considered before defining the real role of endoscopic treatment through dilation in EoE patients:

- There are no universally accepted therapeutic goals for EoE to date. Currently, treatment objectives range from merely controlling the symptoms to resolving the

epithelial inflammatory infiltrate. A group of EoE experts have recommended treating asymptomatic cases of EoE to avoid the potential consequences of fibrous remodeling of the organ (Liacouras et al., 2011), although the long-term consequences are not really known. The experience of each center and the availability of techniques and studies also limit the treatment options and the objectives established in each case. However, we should keep in mind that if left untreated, EoE is a chronic disease involving persistent histological inflammation over time, with detrimental effects on a patient's quality-of-life (Straumann, 2008).

- With regard to what constitutes the best therapeutic option for EoE patients, no studies comparing different therapeutic modalities have been carried out. Moreover, several published EoE case studies involve dilation with concomitant drug therapy (either with steroids or montelukast), which makes it difficult to clearly establish the effect of the individual treatment modalities.
- Additionally, esophageal symptoms are frequently intermittent in EoE patients, who can experience prolonged asymptomatic periods despite the persistence of eosinophilic inflammation. This raises doubts about the convenience of restricting therapy to symptomatic periods only or whether to prescribe a maintenance treatment.
- Narrowing of the esophageal lumen can originate in two ways: by muscle contractions due to motor disturbances secondary to eosinophilic infiltration of deep esophageal wall structures, or by fibrous structures derived from fibrous remodeling and collagen deposits in the subepithelial strata. A combination of both mechanisms may also be possible. In addition, it is difficult to make routine distinctions between patients who have a definite stricture and those in whom it can be reversed through drug or diet therapy.
- A relevant difficulty in assessing the efficacy of individual therapeutic modalities in EoE patients comes from the lack of a validated, commonly accepted score for symptoms in this disease. This makes it difficult not only to extrapolate results from one study to another, but also to objectively evaluate the effect of treatment on clinical manifestations. In this scenario, and with regard to endoscopic treatment, the most valuable criterion for response is the need for repeated dilations.

Fig. 3. Concentric esophageal short stricture, with fibrous appearance because of the absence of vascular pattern, before (a) and after (b) endoscopic dilation using a trough-the-scope balloon. A deep mucosal tear can be observed

In this context, endoscopic dilation can be restricted to two well-established subgroups of EoE patients: those unresponsive to medical therapy and those with a persistent or definitive stricture (Schoepfer et al., 2008). The identification of such patients should be made prior to endoscopic therapy, which in clinical practice implies not using endoscopic dilation as an initial treatment.

4.3 Safety of esophageal dilation in EoE patients

A review of the literature indicates that esophageal dilation is an efficient treatment for EoE, providing immediate relief of symptoms (Zuber-Jerger et al., 2006; Roberts-Thomson, 2009), which is why many authors regard it as a front-line treatment (Vasilopoulos et al., 2002; Straumann, 2010). However, initial reports on the use of esophageal dilation in EoE patients also found a high rate of complications ranging from chest pain to esophageal perforation, which appeared in 7% and 5% of all reported cases, respectively (Furuta et al., 2007; Hirano, 2010). These rates are substantially higher than those for esophageal dilation for other benign strictures. Most described cases of esophageal perforation (spontaneous or after endoscopic procedures) only led to pneumomediastinum (Eisenbach et al., 2006; Rajagopalan & Triadafilopoulos, 2009), but in some cases, an emergency esophagectomy by means of thoracotomy or esophagogastroplasty was required (Lucendo et al., 2011; Riou et al., 1996; Liguori et al., 2008). Although no patient fatalities have been reported to date, the seriousness of these complications has led some researchers to warn that endoscopic dilation poses a higher risk of complications in patients with EoE. That, along with the efficacy and proved safety of dietary modification and topical steroids for this disease, has caused several authors to recommend that dilations not be performed until the presence of an eosinophilic infiltrate has been ruled out (Lucendo & De Rezende, 2007). A trial with corticosteroids before dilation has been also proposed in order to reduce active inflammation and the risk for complications (Sgouros et al., 2006).

The exact cause of the extreme fragility described for esophageal mucosa in EoE has not been clearly established, but it seems to be directly related to the inflammatory infiltrate and the cytotoxic effect of eosinophils. These eosinophils contain several cytotoxic proteins in their cytoplasmic granules capable of damaging tissues (Rothenberg et al., 2001), the risk of which is likely to be higher in patients with a high density of eosinophils and long-term symptoms (Straumann et al., 2008). Multiple evidence obtained from patients (Landres et al., 1978) and from animal models of EoE (Mishra et al., 2001) has shown that the inflammatory infiltrate penetrates deeply into the esophageal wall, reaching the muscle layers. Indeed, fibrous remodeling of the esophageal wall, which reduces the elastic properties of its components, has also been described in EoE patients (Aceves et al., 2004; Straumann et al., 2011). In this sense, esophageal distensibility, which alters the mechanical properties of the esophageal wall (Kwiatek et al., 2011), has been shown to be significantly reduced in adult EoE patients in comparison to controls. Accordingly, both the resistance and distension of the organ may be impaired in EoE, leading to increased fragility during endoscopic dilation procedures (Lucendo & De Rezende, 2007) and in traction movements around the gastroesophageal junction in cases of nausea and vomiting. Thus, a simple brush of the endoscope may give rise to mucosal rents, with cases of spontaneous esophageal perforation (Prasad & Arora, 2005) and Boerhaave's syndrome (Lucendo et al., 2011) having been reported in EoE patients after the mere passage of the endoscope (Kaplan et al., 2003). For these patients especially, then, the various endoscopic procedures must be performed gently.

Two recent retrospective, uncontrolled studies developed in adult EoE patients and published in 2010 and 2011 attempted to assess the safety of esophageal dilation with

bougies or through-the-scope balloons in a total of 363 dilation procedures (Dellon et al., 2010; Jung et al., 2011). In the first study, Dellon and coworkers observed an overall symptom improvement of 83% with a concomitant increase in esophageal caliber. The authors also observed a 7% complication rate, with 2 deep mucosal rents and 3 episodes of chest pain, but no transmural perforations. In the second study, Jung's group found that 9.2% of patients suffered deep mucosal tears while major bleeding and immediate perforation occurred in 0.3% and 1.0% of the patients, respectively. Complication rates from these two studies contrast with the high rates of perforation reported in earlier EoE literature. Moreover, none of the perforations reported in these two studies required surgical intervention (Table 1).

Several predictive factors for complications during dilation have been identified, including a long evolution of dysphagia, the existence of esophageal stenosis, and a high density of eosinophils (Cohen et al., 2007). Complications were also significantly associated with younger age and repeated procedures (Dellon et al., 2010), along with luminal narrowing in the upper and middle esophageal thirds, a luminal stricture incapable of being traversed with a standard upper endoscope, and the use of Savary bougies (Jung et al., 2011).

4.4 Sustained efficacy of endoscopic dilation in EoE patients

In spite of these data, it should be noted that because endoscopic dilation is a mechanical procedure with no effect on the underlying inflammatory process (Schoepfer et al., 2010), its efficacy is probably limited over time. In the case studies published to date, the duration of the effect cannot be appropriately estimated owing to the short monitoring period, although it usually ranges from 3 to 12 months. Still, it is common for patients to undergo repeated dilations, in some cases up to 9 times, to control their symptoms (Schoepfer et al., 2008; Dellon et al., 2010; Pasha et al., 2007). Also noteworthy is the fact that a proportion of patients undergoing endoscopic dilation also receive concomitant drug therapy, which may mask the clinical effect of endoscopic therapy in and of itself (Dellon et al., 2010).

Taking all this into account, endoscopic dilation should be considered as an alternative treatment for patients with EoE and esophageal stenosis when other measures (especially topical steroid treatment) have failed. It is also advisable that the procedure be used together with other therapy modalities in order to avoid complications derived from active eosinophilic inflammation of the organ. Further studies should be carried out to determine which patients are the best candidates for this kind of treatment due to their better clinical results and/or lower complication rates. This will probably require the definition of different patient subgroups or phenotypes according to several variables which are as yet unidentified.

4.5 How endoscopic dilations should be done in EoE?

As noted above, endoscopic dilation constitutes an effective treatment for EoE and should therefore be considered in those patients exhibiting a reduced esophageal caliber and persisting esophageal symptoms despite topical steroid treatment and/or dietary modifications. Dilation should preferably be done when the active inflammatory infiltrate has been banned or significantly reduced (Sgouros et al., 2006). Endoscopic dilation should be carried out by experienced endoscopists and under sedation to avoid provoking Boerhaave's syndrome if the technique is tolerated badly (Nantes et al., 2009). In order to minimize complications, the procedure should be carried out gently with medium-sized bougies, gradually increasing the caliber and never dilating fully to the larger calibers used in the treatment of other forms of stenosis.

Author and year	Patients dilated	Efficiency	Repeated sessions	Perfora- tion	Other complications
Riou PJ et al., 1996	1 patient	Stenotic esophagus despite dilation	No	Yes	Pneumomediastinum and early mediastinitis, requiring subtotal esophagectomy.
Morrow JB. et al., 2001	16 adults	16 clinically improved	1 required repeated dilation	No	Deep mucosal tears Increased post endoscopy analgesia. Difficulty in inserting the endoscope.
Vasilopoulos S. et al., 2002	5 adults	5/ 5 clinically improved	Yes (4 of them)	No	2 extensive esophageal tearing, chest pain and overnight hospitalization.
Straumann A. et al., 2003	11 adults	A single dilation of 7 patients 50% reduction in symptoms 1 patient did not show improvement of symptoms	Yes (in 4 patients)	No	Severe mucosal tearing.
Croese J et al., 2003	17 adults	16/ 17 improved clinically	Mean 3.4 dilations per patient, range 1-13)	No	Tears were recorded in 13 (87%).
Straumann A. et al., 2003	5 adults	5 asymptomatic for 3 to 24 months	No	No	Development of disquieting lesions in response to the procedure.
Nurko S. et al., 2004	7 children	5 total symptomatic relief 2 partial response	Not specified	No	No.
Potter JW. et al., 2004	13 adults	7/ 13 showed transient (<3 months) improvement	Repeated in 6 patients at least twice over the following year	No	Extensive esophageal trauma. Moderate chest pain. Overnight hospitalization.
Langdom DE. 2005	11 (not specified)	Not specified	Not specified	Yes	2–3 day hospitalization, severe chest pain and odynophagia.
Zimmerman SL. et al., 2005	8 adults	8 temporary relief of dysphagia.	Four patients with recurrent dysphagia (mean number of procedures, 2.5; range, 2–4) over an average period of 4.5 years (range 1–10 years).	No	No.

Author and year	Patients dilated	Efficiency	Repeated sessions	Perfora- tion	Other complications
Cantù P. et al., 2005	2 adults	Both cases	No	No	No.
Eisenbach C. et al., 2006	1 adult	Asymptomatic	Repeated esophageal dilation	Yes	No.
Zuber-Jërger I. et al., 2006	1 adult	Clinical improvement for 3 years	Yes, after dysphagia recurred.	No	No.
Pasha SF. et al., 2007	13 adults	11/ 13 clinically improved	Mean number of dilations was 2 (range, 1-5)	No	Superficial mucosal tears occurred in 31% of dilations.
Schoepfer AM. et al., 2008	10 adults	10/ 10 clinically improved over an average 6-month period	Mean number of dilations was 2.7 (range, 1-5)	No	Transient postprocedural odynophagia for 1-3 days.
Rajagopalan J et al., 2009	1 adult	Symptoms improved for 6 months	Two dilations in a 6-week period	No	Severe pain during the subsequent 24-48-hour period.
Dellon ES. et al., 2010	36 patients	Overall clinical response in 20 (83%)	Mean no. of dilations per patient 1.9 (range 1-9).	No	5 complications reported: 2 deep mucosal rents and 3 episodes of chest pain, on of them needing hospitalization.
Jung KW. et al., 2011	161 patients	Not specified	Mean no. of dilations per patient 1.8±1.4	Yes	Deep mucosal tear in 9,2% of dilations and major bleeding in 0,3% of dilations.
Swan MP. et al., 2011	29 patients	Not specified	Mean of 2.07 dilations per patient	No	2 cases admitted with postdilatation pain.

Table 1. Summary of published cases of dilations, their results and complications

No definitive data exist with regard to which dilation technique(s) should be used. Some clinicians prefer using through-the-scope balloons to dilate EoE patients since this method allows the endoscopist a direct visualization of the mucosa during the procedure (Dellon et al., 2010), but the use of Savary bougies has also been reported to be a safe method (Swan et al., 2011).

Multiple strictures are also possible in patients with EoE, but a common strategy in such cases has likewise yet to be established. Inflating a balloon segmentally in multiple areas can dilate the entire esophagus quickly if necessary while maintaining direct visualization at all times (Dellon et al., 2010), but the final method employed should preferably depend on the endoscopist’s experience.

5. Conclusion

Eosinophilic esophagitis is a rapidly emerging disease which has become a common pathology in clinical practice. A wide range of endoscopic findings typical of EoE has been described in the literature, but none of them is pathognomonic for the disease. If a patient

presents more than one typical finding, a diagnosis of EoE can be proposed with a certain amount of confidence, but the definitive diagnosis must be confirmed through biopsies, which should also be performed on patients with compatible clinical data, even if their esophagus appears to be normal.

Endoscopic dilation should only be considered in cases in which symptoms and/ or a reduced esophageal caliber persist despite topical steroid or dietary therapies. The procedure should be carried out gently under sedation with medium-sized hydropneumatic balloons or bougies, and only up to smaller calibers than those used in other forms of strictures.

6. References

- Aceves, SS., Newbury, RO., & Dohil, R. (2007). *J Allergy Clin Immunol. Esophageal remodeling in paediatric eosinophilic esophagitis*, Vol.119, No.1, (January 2007), pp. 206-212, ISSN 0091-6749
- Chehade, M., Sampson, HA., & Morotti, RA. (2007). *J Pediatr Gastroenterol Nutr. Esophageal subepithelial fibrosis in children with eosinophilic esophagitis*, Vol.45, No.3, (September 2007), pp. 319-328, ISSN 1536-4801
- Cheung, KM., Oliver, MR., & Cameron, DJS. (2003). *J Pediatr Gastroenterol Nutr. Esophageal eosinophilia in children with dysphagia*, Vol.37, No.4, (October 2003), pp. 498-503, ISSN 1536-4801
- Cohen, MS., Kaufman, AB., & Palazzo, JP. (2007). *Clin Gastroenterol Hepatol. An audit of endoscopic complications in adult eosinophilic esophagitis*, Vol.5, No.10, (October 2007), pp. 1149-1153, ISSN 1198-1206
- Dellon, ES., Gibbs, WB., & Rubinas, TC. (2010). *Gastrointest Endosc. Esophageal dilation in eosinophilic esophagitis: safety and predictors of clinical response and complications*, Vol.71, No.4, (April 2010), pp. 706-712, ISSN 0016-5107
- Eisenbach, C., Merle, U., & Schirmacher, P. (2006). *Endoscopy. Perforation of the esophagus after dilation treatment for dysphagia in a patient with eosinophilic esophagitis*, Vol.38, Suppl.2, (November 2006), pp. E43-E44, ISSN 1068-9249
- Furuta, GT., & Straumann, A. (2006). *Aliment Pharmacol Ther. Review article: the pathogenesis and management of eosinophilic esophagitis*, Vol.24, No.2, (July 2006), pp. 173-182, ISSN 1365-2036
- Furuta, GT., Liacouras, CA., & Collins, MH. (2007). *Gastroenterology. Eosinophilic esophagitis in children and adults: a systematic review and consensus recommendations for diagnosis and treatment*, Vol.133, No.4, (October 2007), pp. 1342-1363, ISSN 0016-5085
- González-Castillo, S., Arias, Á., & Lucendo, AJ. (2010). *J Clin Gastroenterol. Treatment of eosinophilic esophagitis: how should we manage the disease?*, Vol.44, No.10, (December 2010), pp. 663-671, ISSN 1539-2031
- Hirano, I. (2010). *Gastrointest Endosc. Dilation in eosinophilic esophagitis: to do or not to do?*, Vol.71, No.4, (April 2010), pp. 713-714, ISSN 0016-5107
- Jung, KW., Gundersen, N., & Kopacova, J. (2011). *Gastrointest Endosc. Occurrence of and risk factors for complications after endoscopic dilation in eosinophilic esophagitis*, Vol.73, No.1, (January 2011), pp. 15-21, ISSN 0016-5107

- Kaplan, M., Mutlu, EA., & Jakate, S. (2003). Clin Gastroenterol Hepatol. *Endoscopy in eosinophilic esophagitis: 'feline' esophagus and perforation risk*, Vol.1, No.6, (November 2003), pp. 433-437, ISSN 1198-1206
- Kelly, KJ, Lazenby, AJ, & Rowe, PC. (1995). Gastroenterology. *Eosinophilic esophagitis attributed to gastroesophageal reflux: improvement with an amino acid-based formula*. Vol.109, No.5, (November 1995), pp. 1503-1512, ISSN 0016-5085
- Kwiatek, MA., Hirano, I., & Kahrilas, PJ (2011). Gastroenterology. *Mechanical properties of the esophagus in eosinophilic esophagitis*, Vol.140, No.1, (January 2011), pp. 82-90, ISSN 0016-5085
- Korsapati, H., Babaei, A., & Bhargava, V. (2009). Gut. *Dysfunction of the longitudinal muscles of the oesophagus in eosinophilic oesophagitis*, Vol.58, No.8, (August 2009), pp. 1056-1062, ISSN 1468-3288
- Kwiatek, MA., Hirano, I., & Kahrilas, PJ (2011). Gastroenterology. *Mechanical properties of the esophagus in eosinophilic esophagitis*, Vol.140, No.1, (January 2011), pp. 82-90, ISSN 0016-5085
- Landres, RT., Kuster, GGR., & Strum, WB. (1978). Gastroenterology. *Eosinophilic esophagitis in a patient with vigorous achalasia*, Vol.74, No.6, (June 1978), pp. 1298-1301, ISSN 0016-5085
- Liacouras, CA., Spergel, JM., & Ruchelli, E. (2005). Clin Gastroenterol Hepatol. *Eosinophilic esophagitis: a 10-year experience in 381 children*, Vol.3, No.12, (December 2005), pp. 1198-1206
- Liacouras, CA., Furuta, GT., & Hirano, I. (2011). J Allergy Clin Immunol. *Eosinophilic esophagitis: Updated consensus recommendation for children and adults*, Vol.6, (April 2011), Epub ahead of print, ISSN 0091-6749
- Liguori, G., Cortale, M., & Cimino, F. (2008). World J Gastroenterol. *Circumferential mucosal dissection and esophageal perforation in a patient with eosinophilic esophagitis*, Vol.14; No.5, (February 2008), pp. 803-804, ISSN 1007-9327
- Lucendo, AJ (2007). Med Clin (Barc). *Eosinophilic esophagitis*. Vol.128, No.5, (April 2007), pp. 590-597, ISSN 0025-7753
- Lucendo, AJ, & De Rezende, L. (2007). Endoscopy. *Endoscopic dilation in eosinophilic esophagitis: a treatment strategy associated with a high risk of perforation*, Vol.39, No.4, (April 2007), pp. 376, ISSN 1068-9249
- Lucendo, AJ, Castillo, P., & Martín-Chávarri, S. (2007). Eur J Gastroenterol Hepatol. *Manometric findings in adult eosinophilic oesophagitis: A study of 12 cases*, Vol.19, No.5, (May 2007), pp. 417-424, ISSN 1473-5687
- Lucendo, AJ, Pascual-Turrión, JM., & Navarro, M. (2007). Endoscopy. *Endoscopic, bioptic and manometric findings in eosinophilic esophagitis before and after steroid therapy: a case series*, Vol.39, No.9, (September 2007), pp. 765-771, ISSN 1068-9249
- Lucendo, AJ (2010). Scand J Gastroenterol. *Eosinophilic diseases of the gastrointestinal tract*, 2010, Vol.45, No.9, (September 2010), pp. 1013-1021, ISSN 1502-7708
- Lucendo, AJ, Frigal-Ruiz, AB., & Rodríguez, B. (2011). Dis Esophagus. *Boerhaave's syndrome as the primary manifestation of adult eosinophilic esophagitis. Two case reports and a review of the literature*, Vol.24, No.2, (February 2011), pp. E11-E15, ISSN 1442-2050

- Mackenzie, SH., Go, M., & Chadwick, B. (2008). *Aliment Pharmacol Ther. Eosinophilic oesophagitis in patients presenting with dysphagia-a prospective analysis*, Vol.28, No.9, (November 2008), pp. 1140-1146, ISSN 1365-2036
- Mishra, A., Hogan, SP., & Brandt, EB. (2001). *J Clin Invest. An etiological role for aeroallergens and eosinophils in experimental esophagitis*, Vol.107, No.1, (January 2001), pp. 83-90, ISSN 0021-9738
- Mishra, A., Wang, M., & Pemmaraju, VR. (2008). *Gastroenterology. Esophageal remodeling develops as a consequence of tissue specific IL-5-induced eosinophilia*, Vol.134, No.1, (January 2008), pp. 204-214, ISSN 0016-5085
- Moawad, FJ (2011). *Dig Dis Sci. Esophageal Motor Disorders in Adults with Eosinophilic Esophagitis*, Vol.18, Epub ahead of print, (March 2011), ISSN 1573-2568
- Molina-Infante, J, Ferrando-Lamana, L., & Fernandez-Bermejo, M. *Am J Gastroenterol. Eosinophilic esophagitis in GERD patients: a clinicopathological diagnosis using proton pump inhibitors*, Vol.104, No.11, (November 2009), pp. 2856-2857, ISSN 1572-0241
- Müller, S., Pühl, S., & Vieth, M. (2007). *Endoscopy. Analysis of symptoms and endoscopic findings in 117 patients with histological diagnoses of esinophilic esophagitis*, Vol.39, No.4, (April 2007), pp. 339-344, ISSN 1068-9249
- Nantes, Ó., Jiménez, FJ, & Zozaya, JM. (2009). *Endoscopy. Increased risk of esophageal perforation in eosinophilic esophagitis*, Vol.41, Suppl.2, (July 2009), pp. E177-E178, ISSN 1068-9249
- Nurko, S, Teitelbaum, J, & Husain, K. (2004). *J Pediatr Gastroenterol Nutr. Association of Schatzki ring with eosinophilic esophagitis in children*, Vol.38, No.4, (April 2004), pp. 431-441, ISSN 1536-4801
- Nurko, S, Rosen, R., & Furuta, GT. (2009). *Am J Gastroenterol. Esophageal dysmotility in children with eosinophilic esophagitis: a study using prolonged esophageal manometry*, Vol.104, No.12, (December 2009), pp. 3050-3057, ISSN 1572-0241
- Pasha, SF., DiBaise, JK., & Kim, HJ (2007). *Dis Esophagus. Patient characteristics, clinical, endoscopic, and histologic findings in adult eosinophilic esophagitis: a case series and systematic review of the medical literature*, Vol.20, No.4 (2007), pp. 311-319, ISSN 1442-2050
- Peery, AF., Cao, H., & Dominik, R. (2011). *Clin Gastroenterol Hepatol. Variable Reliability of Endoscopic Findings with White-Light and Narrow-Band Imaging for Patients with Suspected Eosinophilic Esophagitis*, Vol.4, Epub ahead of print, (March 2011), ISSN 1542-7714
- Prasad, GA., & Arora, AS. (2005). *Dis Esophagus. Spontaneous perforation in the ringed esophagus*, Vol. 18, No.6 (June 2005), pp. 406-409, ISSN 1442-2050
- Prasad, GA., Talley, NJ, & Romero, Y. (2007). *Am J Gastroenterol. Prevalence and predictive factors of eosinophilic esophagitis in patients presenting with dysphagia: a prospective study*, Vol.102, No.12, (December 2007), pp. 2627-2632, ISSN 1572-0241
- Rajagopalan, J, & Triadafilopoulos, G. (2009). *Dis Esophagus. Ring(s)-related esophageal meat bolus impaction: biopsy first, dilate later*, Vol.22, No.5 (November 2009), pp. E14-E16, ISSN 1442-2050
- Ricker, J, McNear, S., & Cassidy, T. (2011). *Therap Adv Gastroenterol. Routine screening for eosinophilic esophagitis in patients presenting with dysphagia*, Vol.4, No.1, (January 2011), pp. 27-35, ISSN 1756-2848

- Riou, PJ, Nicholson, AG., & Pastorino, U. (1996). *Ann Thorac Surg. Esophageal rupture in a patient with idiopathic eosinophilic esophagitis*, Vol.62, No.6 (December 1996), pp. 1854-1856, ISSN 1552-6259
- Roberts-Thomson, IC. (2005). *J Gastroenterol Hepatol. Images of interest. Gastrointestinal: Eosinophilic esophagitis*, Vol.20, No.8, (August 2005), pp. 1299, ISSN 1440-1746
- Roman, S., Hirano, I., & Kwiatek, MA. (2011). *Neurogastroenterol Motil. Manometric features of eosinophilic esophagitis in esophageal pressure topography*, Vol.23, No.3, (March 2011), pp. 208-214, ISSN 1365-2982
- Schoepfer, AM., Gschossmann, J., & Scheurer, U. (2008). *Endoscopy. Esophageal strictures in adult eosinophilic esophagitis: dilation is an effective and safe alternative after failure of topical corticosteroids*, Vol.40, No.2, (February 2008), pp. 161-164, ISSN 1068-9249
- Schoepfer, AM., Gonsalves, N., & Bussmann, C. (2009). *Am J Gastroenterol. Esophageal Dilation in Eosinophilic Esophagitis: Effectiveness, Safety, and Impact on the Underlying Inflammation*, Vol. 105, No.5, (May 2009), pp. 1062-1070, ISSN 1572-0241
- Sgouros, SN., Bergele, C., & Mantides, A. (2006). *Eur J Gastroenterol Hepatol. Eosinophilic esophagitis in adults: a systematic review*, Vol.18, No.2, (February 2006), pp. 211-217, ISSN 1473-5687
- Sgouros, SN., Bergele, C., & Mantides, A. (2006). *Endoscopy. Eosinophilic esophagitis in adults: what is the clinical significance?*, Vol.38, No. 5, (May 2006), pp. 515-520, ISSN 1068-9249
- Spechler, SJ, Genta, RM., & Souza, RF. (2007). *Am J Gastroenterol. Thoughts on the complex relationship between gastroesophageal reflux disease and eosinophilic esophagitis*, Vol.102, No.6, (June 2007), pp. 1301-1306, ISSN 1572-0241
- Straumann, A., Rossi, L., & Simon, HU. (2003). *Gastrointest Endosc. Fragility of the esophageal mucosa: a pathognomonic endoscopic sign of primary eosinophilic esophagitis?*, Vol.57, No.3, (March 2003), pp. 407-412, ISSN 0016-5107
- Straumann, A., Spichtin, HP., & Grize, L. (2004). *Digestion. Eosinophilic esophagitis: red on microscopy, white on endoscopy*, Vol.70, No.2 (September 2004), pp. 109-116, ISSN 1421-9867
- Straumann, A. (2008). *Gastrointestinal Endosc Clin North Am. The natural history and complications of eosinophilic esophagitis*, Vol.18, No.1, (January 2008), pp. 99-118, ISSN 1052-5157
- Straumann, A., Bussmann, C., & Zuber, M. (2008). *Clin Gastroenterol Hepatol. Eosinophilic Esophagitis: Analysis of Food Impaction and Perforation in 251 Adolescent and Adult Patients*, Vol.6, No.5, (May 2008), pp. 598-600, ISSN 1542-7714
- Straumann, A. (2010). *Gastrointest Endosc Clin N Am. The natural history and complications of eosinophilic esophagitis*, Vol.18, No.1, (January 2010), pp. 99-118, ISSN 1052-5157
- Straumann, A., Conus, S., & Degen, L. (2010). *Gastroenterology. Budesonide is effective in adolescent and adult patients with active eosinophilic esophagitis*, Vol.139, No.5, (November 2010), pp. 1526-1537, ISSN 0016-5085
- Straumann, A., Conus, S., & Degen, L. (2011). *Clin Gastroenterol Hepatol. Long-term budesonide maintenance treatment is partially effective for patients with eosinophili esophagits*, Vol.28, Epub ahead of print (January 2011), ISSN 1542-7714
- Swan, MP., Donnellan, F., & Kandel, G. (2011). *Gastrointest Endosc. Safe and effective dilatation in eosinophilic esophagitis: the tale of two subgroups*, Vol.72, No.2, (February 2011), pp. 408, ISSN 0016-5107

- Vasilopoulos, S., Murphy, P., & Auerbach, A. (2002). *Gastrointest Endosc. The small-caliber esophagus: an unappreciated cause of dysphagia for solids in patients with eosinophilic esophagitis*, Vol.55, No.1, (January 2002), pp. 99-106, ISSN 0016-5107
- Zuber-Jerger, I., Ratiu, N., & Kullman, F. (2006). *JGastrointestin Liver Dis. Long-lasting effect of endoscopic dilation of an esophageal stenosis due to eosinophilic esophagitis*, Vol.15, No.2, (June 2006), pp. 167-170, ISSN 1842-1121

IntechOpen

IntechOpen

Gastrointestinal Endoscopy

Edited by Prof. Oliviu Pascu

ISBN 978-953-307-385-9

Hard cover, 272 pages

Publisher InTech

Published online 19, July, 2011

Published in print edition July, 2011

Endoscopy has had a major impact in the development of modern gastroenterology. By using different data it provided a better understanding of pathogenic mechanisms, described new entities and changed diagnostic and therapeutic strategies. Meanwhile, taking advantage of many technical advances, endoscopy has had a developed spectacularly. Video-endoscopes, magnification, confocal and narrow-band imaging endoscopes, endoscopic ultrasounds and enteroscopes emerged. Moreover, endoscopy has surpassed its function as an examination tool and it became a rapid and efficient therapeutic tool of low invasiveness. InTech Open Access Publisher selected several known names from all continents and countries with different levels of development. Multiple specific points of view, with respect to different origins of the authors were presented together with various topics regarding diagnostic or therapeutic endoscopy. This book represents a valuable tool for formation and continuous medical education in endoscopy considering the performances or technical possibilities in different parts of the world.

How to reference

In order to correctly reference this scholarly work, feel free to copy and paste the following:

Alfredo J. Lucendo and Susana Jiménez-Contreras (2011). Endoscopic Aspects of Eosinophilic Esophagitis: From Diagnosis to Therapy, Gastrointestinal Endoscopy, Prof. Oliviu Pascu (Ed.), ISBN: 978-953-307-385-9, InTech, Available from: <http://www.intechopen.com/books/gastrointestinal-endoscopy/endoscopic-aspects-of-eosinophilic-esophagitis-from-diagnosis-to-therapy>

INTECH
open science | open minds

InTech Europe

University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
www.intechopen.com

InTech China

Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821

© 2011 The Author(s). Licensee IntechOpen. This is an open access article distributed under the terms of the [Creative Commons Attribution 3.0 License](https://creativecommons.org/licenses/by/3.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

IntechOpen

IntechOpen