

How to Approach Lymphocytic Esophagitis

Anh D. Nguyen¹ · Kerry B. Dunbar²

Published online: 20 April 2017

© Springer Science+Business Media New York (outside the USA) 2017

Abstract

Purpose of Review Lymphocytic esophagitis (LE) is an unusual esophageal condition defined by an increased number of lymphocytes in the esophageal epithelium. With few published studies of LE available, it is unclear whether LE is a truly distinct clinical entity or a histological manifestation of other known gastrointestinal disorders. This review summarizes recent studies of lymphocytic esophagitis.

Recent Findings Studies have suggested that LE may be related to eosinophilic esophagitis (EoE) or a manifestation of gastroesophageal reflux disease (GERD). There is an association between LE and Crohn's disease in children, but not in adults. Patients with LE frequently report symptoms of dysphagia and GERD. Treatment options for LE are limited and involve symptom management similar to treatment of EoE or GERD, including proton pump inhibitors (PPI), swallowed topical steroids, and endoscopic dilation.

Summary With no formal definition and a variety of clinical presentations and endoscopic findings, diagnosis and management of symptomatic LE patients is challenging for clinicians.

Keywords Lymphocytic esophagitis · Lymphocytic oesophagitis · Dysphagia · Gastroesophageal reflux disease (GERD)

Introduction

Lymphocytic esophagitis (LE) was first described in 2006 when Rubio et al. identified 20 patients with increased intraepithelial lymphocytes and few intraepithelial granulocytes. The intraepithelial lymphocytosis was confined to the esophagus as no intraepithelial lymphocytes were found in the stomach, small bowel, or colon [1]. Although 10 years have elapsed since it was first described, the diagnostic criteria and clinical features of LE remain elusive, and it is unclear whether it represents a truly distinct clinical entity or a histological manifestation of other known disorders. This ambiguity may be due in part to the lack of published literature on LE. At the time of this review, there have been 11 published research studies and eight case reports discussing LE. These studies are summarized in Table 1.

Topical Collection on *Esophagus*

✉ Kerry B. Dunbar
Kerry.Dunbar@utsouthwestern.edu; Kerry.Dunbar@va.gov

Anh D. Nguyen
Anh.Nguyen@phhs.org

¹ Division of Gastroenterology and Hepatology, Department of Medicine, University of Texas Southwestern Medical Center, Dallas, TX 75231, USA

² Esophageal Diseases Center, Division of Gastroenterology and Hepatology, Department of Medicine, Dallas VA Medical Center and the University of Texas Southwestern Medical Center, GI Lab—CA 111-B1, Dallas VAMC, 4500 South Lancaster Road, Dallas, TX 75231, USA

Pathogenesis

Part of the difficulty in clarifying the characteristics of LE lies in the rarity of the diagnosis. LE was found in 0.09% of esophageal biopsies obtained by endoscopy in a study of 129,252 adult patients [6]. In children, the prevalence was 5.7% in a study of 545 patients undergoing endoscopy [8].

The pathogenesis and cause of LE is unclear, and several possible causes of LE have been proposed. LE may be an allergic disorder; Purdy et al. found that LE resembled contact dermatitis with a third of their LE study population reporting atopy, though this prevalence did not differ significantly from

Table 1 Summary table of research studies and case reports of lymphocytic esophagitis

Study	Year published	Number of patients with LE	Gender	Age (years)	Number of intraepithelial lymphocytes	Clinical characteristics	Symptoms	Endoscopic findings	Treatment
Research studies									
Rubio et al. [1•]	2006	20	10 females 10 males	31.3 (range 2–82)	55.1 (range 21–129)	8 (40%) Crohn's disease 4 (20%) GERD 4 (20%) Esophageal cancer	7 (35%) none 4 (20%) GERD 1 (5%) dysphagia	7 (35%) esophagitis 4 (20%) normal 3 (15%) gastroduodenitis 1 (5%) hiatal hernia 1 (5%) ulcer	N/A
Purdy et al. [2]	2008	42	N/A	44 (range 2–81)	70.3 (range 23–133)	2 (10%) Celiac disease 14 (33%) Allergy 11 (26%) GERD 5 (12%) Crohn's disease 4 (10%) H. pylori gastritis	18 (43%) dysphagia 11 (26%) GERD	13 (31%) normal 10 (24%) esophagitis 5 (12%) rings 4 (10%) hiatal hernia	N/A
Basseri et al. [3]	2010	2 (part of a 72 patient study of patients with GERD and EoE)	Female	41, 54	>50	N/A	Globus, abdominal pain, bloating, dysphagia, odynophagia	1 (50%) esophagitis 1 (50%) normal	N/A
Ebach et al. [4]	2011	20	N/A	13.7 (5.9–20.7)	>50	17 (85%) Crohn's disease	10 (50%) none 4 (20%) nausea/-vomiting 2 (10%) abdominal pain 1 (5%) GERD 1 (5%) early satiety	9 (45%) normal 5 (25%) edema 3 (15%) erosions	N/A
Cohen et al. [5•]	2012	81	44 females 37 males	51 (range 19–84)	>20	40 (49%) GERD 28 (35%) Hypothyroidism 11 (14%) IBD	54 (67%) Dysphagia 38 (47%) GERD 36 (44%) chest/-abdominal pain 24 (30%) nausea 10 (12%) odynophagia 30 (71%) dysphagia 11 (26%) GERD 1 (2%) chest pain 2 (5%) Crohn's disease	24 (30%) normal 16 (20%) rings 13 (16%) esophagitis 7 (9%) strictures	PPI (improvement)
Haque and Genta [6•]	2012	42	26 females 16 males	52 (range 20–84)	125	N/A	9 (21%) normal 13 (31%) esophagitis 13 (31%) rings, felinization 4 (10%) strictures 3 (7%) decreased motility	9 (21%) normal 13 (31%) esophagitis 13 (31%) rings, felinization 4 (10%) strictures 3 (7%) decreased motility	N/A

Table 1 (continued)

Study	Year published	Number of patients with LE	Gender	Age (years)	Number of intraepithelial lymphocytes	Clinical characteristics	Symptoms	Endoscopic findings	Treatment
Haque and Genta [6•]	2012	119	53 females 47 males	63	N/A	N/A	1 (2%) sarcoidosis 63 (53%) dysphagia 22 (19%) GERD	40 (34%) felinization, rings, furrows 27(23%) normal 22 (19%) esophagitis 12 (10%) strictures 6 (55%) rings 6 (55%) furrows 4 (36%) stenosis 2 (18%) exudates 2 (6%) erythema 2 (6%) white plaques 1 (3%) stricture	N/A
Tanaka et al. [7]	2013	11	11 males	39 (range 26–82)	>20	N/A	N/A		N/A
Sutton et al. [8]	2014	31	15 females 16 males	9 (range 1–16)	106 (range 50–278)	6 Crohn's disease	19 (61%) abdominal pain 17 (55%) vomiting 10 (32%) diarrhea 2 (6%) dysphagia 28 (62%) dysphagia	15 (33%) normal 6 (13%) rings 6 (13%) esophagitis 4 (9%) strictures	N/A
Xue et al. [9]	2015	45	29 females 16 males	59–65	129–166	16 (36%) Primary esophageal motility disorders	13 (29%) GERD		N/A
Pasricha et al. [10]	2016	27	17 females 10 males	56	4 = 10–20 14 = 21–40 1 = 41–80 6 = >80	14 (52%) GERD 1 (4%) Crohn's disease	19 (70%) dysphagia 7 (26%) GERD 3 (11%) none	10 (37%) strictures 9 (33%) esophagitis 7 (26%) rings 4 (15%) normal	6 PPI (2 improved) 1 swallowed fluticasone (improvement) 1 GI cocktail 1 prednisone taper
Case reports									
Kasirye et al. [11]	2011	1	Female	60	N/A	Stage IV CKD Lichen planus	Dysphagia	Rings, furrows	PPI, H2B Swallowed fluticasone (improvement)
Mandaliya et al. [12]	2012	1	Male	74	N/A	Non Hodgkin Lymphoma	Dysphagia, food impaction	Esophageal stricture, rings	Dilation with injected botulinum toxin
Basseri et al. [13]	2013	1	Male	20	>50	Crohn's disease	N/A	Esophagitis, duodenal nodularity, superficial ulceration and scalloping duodenal mucosa	N/A
Hendy et al. [14]	2013	1	Female	35	N/A	N/A	Chest pain, fever, odynophagia	Normal	Swallowed fluticasone (improvement)
Figueredo et al. [15]	2014	1	Female	30	N/A	Behcet's disease, SLE	Narrowed lumen, ring, strictures		Dilation

Table 1 (continued)

Study	Year published	Number of patients with LE	Gender	Age (years)	Number of intraepithelial lymphocytes	Clinical characteristics	Symptoms	Endoscopic findings	Treatment
Maejima et al. [16]	2015	1	Male	68	N/A	GERD	GERD, odynophagia, food impaction	Stricture, furrows	Swallowed fluticasone (improvement) PPI Dilation (improvement)
Zhang et al. [17]	2016	1	Female	66	N/A	Bipolar disorder	GERD, dysphagia, food impaction Dysphagia, epigastric pain	Rings	PPI BID (improvement) Dilation (improvement)
Niewiarowski and Stoll [18]	2016	1	Female	82	N/A	N/A	Dysphagia	Web in the cervical esophagus Rings	Dilation (improvement)

that of an unmatched control population [2]. A separate case report identified mast cells on histological examination, suggesting that LE may be due to an allergic or hypersensitivity reaction [17]. Another case report proposed an autoimmune phenomenon when LE was identified in a patient with common variable immune deficiency [19].

It has also been suggested that LE is not an independent clinical entity but rather a histologic process existing along the eosinophilic esophagitis (EoE) or reflux esophagitis spectrum [20]. Lymphocytes are also involved in the pathogenesis of gastroesophageal reflux disease (GERD). In one study of patients with GERD, discontinuation of proton pump inhibitor (PPI) therapy led to lymphocytic infiltration of the esophageal mucosa as GERD symptoms developed [21]. Another study of patients with GERD found 5% of reflux esophagitis specimens met the criteria for LE [3]. Thus, LE may be a manifestation of GERD, related to EoE, or associated with allergic disease.

Diagnostic Criteria

As LE is a histological diagnosis, the gold standard for diagnosis is biopsy of the esophageal mucosa. The changes of LE can be found throughout the esophagus but is identified most frequently in biopsies of the distal esophagus, followed by the proximal and mid esophagus [2, 8]. The pattern of biopsies may impact the diagnosis of LE. In a study of 81 patients, >90% of LE cases were diagnosed through random esophageal biopsies compared to 9% of LE cases diagnosed by targeted biopsies of abnormal mucosa or strictures [5•]. While some commentators have suggested that LE is a transient response to an unknown insult, two studies have found LE to be a chronic process with LE found on biopsies from repeat endoscopies [2, 5•].

Histological changes in LE include an increased number of intraepithelial lymphocytes in the peripapillary areas of the esophageal epithelium with few or no intraepithelial granulocytes. Spongiosis, the dilation of intercellular spaces, has also been reported [6•, 8]. In contrast, lymphocytes are found primarily in the interpapillary area in other causes of esophagitis such as radiation, reflux, and candidiasis. While the normal number of lymphocytes in the esophageal epithelium is 10–12 per high-powered field (HPF), the exact number of lymphocytes required for a diagnosis of LE has not been rigorously defined [9, 22]. Research studies have used a wide range of lymphocyte densities to define LE, most commonly >50 lymphocytes per HPF (range 10–50 lymphocytes per HPF), though some studies only report the lymphocyte counts per HPF without defining a specific cutoff value [1•, 2, 5•, 8, 13, 16, 23]. Haque and Genta advocate against using numeric lymphocyte densities as part of the diagnostic criteria for LE due to its often patchy distribution. Rather, they propose that

lymphocytes' peripapillary location and associated spongiosis are more reliable determinants of the diagnosis [6•]. There is also no consensus regarding the presence of intraepithelial granulocytes in patients with LE. Some investigators have suggested the definition of LE should include the absence of granulocytes, which serves to exclude other conditions such as EoE [18].

Spongiosis is also seen in patients with LE. Rubio et al. examined the intercellular spaces in four LE patients using transmission electron microscopy (TEM). In addition to intraepithelial lymphocytosis, TEM identified marked spongiosis and regressive changes of the squamous cells, ranging from cytoplasmic vacuolization to cell disintegration. In this study, the cause of the spongiosis was unclear, as none of the four types of spongiosis previously described in the pathology literature were identified in the LE patients. Rubio proposed that the combination of hampered cell nutrition due to spongiosis and production of noxious molecules lead to injury of the squamous cells and lymphocyte infiltration [24].

Summarizing the available data, the current diagnostic criteria for LE include an increased number of lymphocytes (≥ 50 per HPF) in the peripapillary space with associated spongiosis and few to no granulocytes. However, no formal definition for LE has been proposed as the histological criteria are still evolving. Thus, clinicians should consider discussing equivocal cases with a pathologist.

Clinical Characteristics

Most published studies of LE are small, the largest to date having included 119 patients [6•]. In terms of demographics, several studies of LE have reported a slight female preponderance with the highest frequency in the fifth and sixth decades of life [5•, 6•, 9, 10]. Common symptoms in LE patients include dysphagia and GERD. Dysphagia has been reported as the predominant complaint in two studies, with a frequency of up to 70% in patients with LE [10, 23]. In the study by Haque and Genta, dysphagia was as frequently reported by LE patients as by those with EoE [6•]. Patients with LE appear to have lower rates of food impaction than EoE patients, however [5•, 25].

GERD symptoms are also frequent, occurring in 20–25% of LE patients [1•, 2, 5•]. Chest and abdominal pain were reported in 44% of LE patients in one study [5•]. Spontaneous esophageal perforation was described in a single case report [14]. Esophageal motility abnormalities, including hypocontractile and hypercontractile patterns, have also been reported in one-third of LE patients. One study examined the association of CD4+ and CD8+ T lymphocytes with esophageal dysmotility and found that patients with CD4+ lymphocyte-predominant LE were twice as likely as patients with CD8+ lymphocyte-predominant LE to have primary

motility disorders identified by esophageal manometry or barium esophagram [23].

Numerous studies have searched for an association between LE and other diagnoses. Specifically, there have been several research studies evaluating the association between LE and Crohn's disease in children and adults. In the initial study of LE by Rubio et al., 40% of LE patients were found to have Crohn's disease, but almost all patients with both LE and Crohn's disease were under 17 years of age [1•]. In two further pediatric studies, Ebach et al. found LE in 28% of all children with Crohn's disease undergoing upper endoscopy as compared with 4.4% without Crohn's disease, while Sutton et al. found LE in 12% of children with Crohn's disease compared to 5% without Crohn's disease. Both studies suggest a significant association between these two processes in the pediatric population [4, 8]. However, subsequent studies in adults have not found any association between Crohn's disease and LE [2, 5•, 6•, 10, 23]. One study of adults with inflammatory bowel disease (IBD) reported that increased esophageal peripapillary lymphocytosis was associated with higher inflammatory marker levels and an increased IBD disease activity index, suggesting that LE may be a marker of IBD severity in the adult population [13]. No association between celiac disease and LE has been identified [2, 6•].

Based on the available data, LE has a benign clinical course and good overall prognosis. In a study by Cohen et al., 70 of 81 patients with LE were alive after a median follow-up interval of 3.3 years. Among the 29 patients for whom follow-up survey data were obtained, 22 were found to have ER visits in the preceding 5 years, with 41% (9/29) of visits attributable to a gastrointestinal complaint. Despite this finding, 59% of patients reported improvement in their symptoms over time, usually after PPI initiation, with greater than 60% of patients reporting satisfaction with their gastrointestinal health [5•].

Endoscopic Findings

The endoscopic findings reported in patients with LE vary widely by study. Pasricha et al. found that 82% of the 27 LE patients evaluated had abnormal endoscopic findings. In contrast, Purdy et al. found no difference in endoscopic appearance between LE patients and controls [2, 10]. Similarities between the endoscopic appearance of EoE and LE have also been reported. In one study, 34% of LE patients had findings similar to EoE such as rings, furrows, plaques, and strictures [6•]. In another study, LE patients and EoE patients had comparable rates of esophageal rings, but LE patients had lower rates of strictures [5•]. The cause of the endoscopic findings seen in LE is unclear. One study proposed that the endoscopic findings common to EoE and LE may develop due to inflammatory cell infiltration (eosinophils in EoE and lymphocytes in LE), leading to muscularis mucosa contraction and

esophageal wall thickening [17]. Endoscopic ultrasound in at least one patient with LE has demonstrated thickening of the esophageal mucosa and submucosa [16]. The endoscopic findings seen in LE have also been evaluated with narrow band imaging (NBI). Using NBI, 90.5% of LE patients were found to have beige discoloration of the mucosa, an increased number of congested intrapapillary capillary loops, and poor visibility of submucosal vessels. However, these findings were not specific for LE. The authors of this study suggested that the presence of all three of the above findings might indicate EoE or LE rather than GERD [7].

Treatment Options

No clear treatment guidelines have been proposed for LE management. As the symptoms of LE are similar to those of EoE, clinicians have attempted to manage LE using similar treatment strategies, including PPI use, swallowed fluticasone, and endoscopic dilation. Several studies of LE patients have demonstrated symptomatic improvement with PPI treatment, which may be due to either the control of acid reflux in cases where LE is a manifestation of GERD or the anti-inflammatory properties of PPIs [5, 10, 17]. As corticosteroids have been found effective in other gastrointestinal lymphocytic diseases, such as lymphocytic colitis, and in the treatment of EoE, their use has been applied to LE as well. Swallowed fluticasone led to symptomatic improvement of LE symptoms in four studies [10, 11, 14, 15]. One study noted improvement after a 1-month trial. Other studies have not recommended a specific treatment duration when using topical corticosteroids. In the four studies that considered esophageal dilation as therapy for LE, the intervention was targeted to strictures and rings rather than use of empiric dilation when no endoscopic abnormalities were present [12, 15, 16, 18].

At this time, treatment of LE is challenging. Some patients have improvement with PPI treatment, underscoring the hypothesis that LE may be related to GERD or EoE. As some patients with LE have a similar clinical presentation to those with EoE, swallowed topical corticosteroids have also been used with variable success. Symptomatic treatment of dysphagia by dilation of rings and strictures can also be considered.

Conclusions

Awareness of lymphocytic esophagitis by gastroenterologists and pathologists has increased in recent years. At this time, LE should be considered when evaluating patients with dysphagia. When LE is identified on esophageal biopsies, patients can be reassured that it has a typically benign course. A trial of PPI could be considered with escalation to swallowed fluticasone if there is no improvement in symptoms. Patients

who have dysphagia as their predominant complaint can be considered for dilation, especially if strictures or rings are present. Additional research is needed to determine whether LE is truly a distinct clinical entity or a manifestation of other GI disorders. Further investigation is also needed to elucidate its pathophysiology and optimal management strategy.

Compliance with Ethical Standards

Conflict of Interest All authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

Support: This material is the result of work supported with resources at the Dallas VA Medical Center.

VA/US Government disclaimer: The contents do not represent the views of the US Department of Veterans Affairs or the United States Government.

References

Papers of Particular Interest, Published Recently, Have Been Highlighted as:

• of Importance

1. Rubio CA, Sjö Dahl K, Lagergren J. Lymphocytic esophagitis: a histologic subset of chronic esophagitis. *Am J Clin Pathol*. 2006;125:432–7. **The first study to describe lymphocytic esophagitis.**
2. Purdy JK, Appelman HD, Golembeski CP, McKenna BJ. Lymphocytic esophagitis: a chronic or recurring pattern of esophagitis resembling allergic contact dermatitis. *Am J Clin Pathol*. 2008;130:508–13.
3. Basseri B, Levy M, Wang HL, Shaye OA, Pimentel M, Soffer EE, et al. Redefining the role of lymphocytes in gastroesophageal reflux disease and eosinophilic esophagitis. *Dis Esophagus*. 2010;23:368–76.
4. Ebach DR, Vanderheyden AD, Ellison JM, Jensen CS. Lymphocytic esophagitis: a possible manifestation of pediatric upper gastrointestinal Crohn's disease. *Inflamm Bowel Dis*. 2011;17:45–9.
5. Cohen S, Saxena A, Waljee AK, Piraka C, Purdy J, Appelman H, et al. Lymphocytic esophagitis: a diagnosis of increasing frequency. *J Clin Gastroenterol*. 2012;46:828–32. **Provided data on natural history and quality of life of lymphocytic esophagitis patients after a 3-year follow-up.**
6. Haque S, Genta RM. Lymphocytic oesophagitis: clinicopathological aspects of an emerging condition. *Gut*. 2012;61:1108–14. **The largest study of patients with lymphocytic esophagitis, identified from a review of a large pathology database. Also proposed criteria for defining lymphocytic esophagitis.**
7. Tanaka K, Rubio CA, Dlugosz A, Truskaite K, Befrits R, Lindberg G, et al. Narrow-band imaging magnifying endoscopy in adult patients with eosinophilic esophagitis/esophageal eosinophilia and lymphocytic esophagitis. *GastrointestEndosc*. 2013;78:659–64.
8. Sutton LM, Heintz DD, Patel AS, Weinberg AG. Lymphocytic esophagitis in children. *Inflamm Bowel Dis*. 2014;20:1324–8.
9. Xue Y, Suriawinata A, Liu X, Li Z, Gabbard S, Rothstein R, et al. Lymphocytic esophagitis with CD4 T-cell-predominant

- intraepithelial lymphocytes and primary esophageal motility abnormalities: a potential novel clinicopathologic entity. *Am J SurgPathol*. 2015;39:1558–67.
10. Pasricha S, Gupta A, Reed CC, Speck O, Woosley JT, Dellon ES. Lymphocytic esophagitis: an emerging Clinicopathologic disease associated with dysphagia. *Dig Dis Sci*. 2016;61:2935–41.
 11. Kasirye Y, John A, Rall C, Resnick J. Lymphocytic esophagitis presenting as chronic dysphagia. *Clin Med Res*. 2012;10:83–4.
 12. Mandaliya R, Dimarino AJ, Cohen S. Lymphocytic esophagitis mimicking eosinophilic esophagitis. *Ann Gastroenterol*. 2012;25:355–7.
 13. Basseri B, Vasiliauskas EA, Chan O, Wang HL, Basseri RJ, Pimentel M, et al. Evaluation of peripapillary lymphocytosis and lymphocytic esophagitis in adult inflammatory bowel disease. *GastroenterolHepatol*. 2013;9:505–11.
 14. Hendy PJ, Wong DS, Florin TH. Spontaneous oesophageal perforation: an unreported complication of lymphocytic oesophagitis. *Gut*. 2013;62:1668–9.
 15. Figueiredo PC, Pinto-Marques P, Borralho P, Freitas J. Unusual cause for smoldering dysphagia. *Lymphocytic esophagitis*. *Dysphagia*. 2014;29:283–5.
 16. Maejima R, Uno K, Iijima K, Fujishima F, Noguchi T, Ara N, et al. A Japanese case of lymphocytic esophagitis. *Dig Endosc*. 2016;28:476–80.
 17. Zhang Z, Jain D, Brand M. Ringed esophagus secondary to lymphocytic esophagitis. *Gastroenterol Hepatol*. 2016;12:237–9.
 18. Niewiarowski TJ, Stoll LM. Recurrent dysphagia in a patient with chronic lymphocytic esophagitis. *GastrointestEndosc*. 2016; doi:10.1016/j.gie.2016.02.016.
 19. Vangimalla S, Gordon I, Thota PN. Image of the month: lymphocytic esophagitis in common variable immune deficiency. *Am J Gastroenterol*. 2016;111:170.
 20. Genta RM. Lymphocytic esophagitis. *Gastroenterol Hepatol*. 2015;11:559–61.
 21. Dunbar KB, Agoston AT, Odze RD, Huo X, Pham TH, Cipher DJ. Association of acute gastroesophageal reflux disease with esophageal histologic changes. *JAMA*. 2016;315:2104–12.
 22. Resnick MB, Finkelstein Y, Weissler A, Levy J, Yakirevich E. Assessment and diagnostic utility of the cytotoxic T-lymphocyte phenotype using the specific markers granzyme-B and TIA-1 in esophageal mucosal biopsies. *Hum Pathol*. 1999;30:397–402.
 23. Wang HH, Mangano MM, Antonioli DA. Evaluation of T-lymphocytes in esophageal mucosal biopsies. *Mod Pathol*. 1994;7:55–8.
 24. Rubio CA, Villnow E, Schmidt PT. Lymphocytic oesophagitis preliminary ultrastructural observations. *Anticancer Res*. 2016;36:2315–22.
 25. Truskaite K, Dlugosz A. Prevalence of eosinophilic esophagitis and lymphocytic esophagitis in adults with esophageal food bolus impaction. *Gastroenterol Res Pract*. 2016; doi:10.1155/2016/9303858.