



Eosinophilic Oesophagitis Presenting as Food Impaction in Adulthood: A Narrative Review of Diagnostic Delay and the Treatment Decision

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Abstract

Food impaction is the moment eosinophilic oesophagitis becomes unmistakable, and for most patients it is not the first sign. It is the point at which years of adapted eating finally fail, and the endoscopy that relieves it is also the opportunity to make the diagnosis that should have been made earlier.

Objectives: To review the published evidence on eosinophilic oesophagitis presenting as food impaction in adults: how often the diagnosis is already known, what delay costs in terms of stricture formation, what the treatment options achieve, and what should happen at the index endoscopy.

Material and Methods: Narrative review of randomised trials, cohort studies, position papers and epidemiological analyses indexed in PubMed and the major gastroenterology and allergy journals. No new patient data were generated; every estimate quoted is one published by the original investigators.

Results: In a cohort of 198 oesophageal food impactions, eosinophilic oesophagitis was already diagnosed in only 17.2 per cent. Reported histologic remission rates are approximately 45 per cent for proton pump inhibitors, 80 to 93 per cent for swallowed topical corticosteroids, around 60 per cent for dietary elimination and around 60 per cent for dupilumab. In the phase 3 trial, histologic remission to 6 or fewer eosinophils per high-power field was achieved by 60 per cent on weekly dupilumab against 5 per cent on placebo at 24 weeks. Untreated disease progresses from inflammatory to fibrostenotic, with stricture risk rising by approximately 5 per cent for each year the diagnosis is not made.

Conclusion: Effective treatments exist and most patients reach remission on one of them, so the determinant of outcome is when the diagnosis is made rather than which drug follows it. Taking oesophageal biopsies at the index impaction endoscopy, irrespective of appearances, is the single intervention the evidence most clearly supports.

Abbreviations: DSQ – Dysphagia Symptom Questionnaire, EoE – Eosinophilic Oesophagitis, eos/hpf – Eosinophils per High-Power Field, EREFS – Endoscopic Reference Score, PPI – Proton Pump Inhibitor, STC – Swallowed Topical Corticosteroid.

Keywords: Eosinophilic Oesophagitis; Food Impaction; Diagnostic Delay; Oesophageal Stricture; Dupilumab

Introduction

Eosinophilic oesophagitis is a chronic, immune-mediated disease of the oesophagus defined by symptoms of oesophageal dysfunction together with eosinophil-predominant inflammation, conventionally 15 or more eosinophils per high-power field [1,2]. Prevalence estimates cluster around 23 per 100,000 and have risen consistently across high-income settings [3,4].

In adults the dominant symptoms are dysphagia and food impaction [1,5]. Endoscopic features include linear furrows, white exudates, concentric rings, reduced vascularity and strictures, graded by the Endoscopic Reference Score [6,7]. Chronic inflammation produces remodelling, and remodelling produces the fixed narrowing that causes the impaction [8,9].

The clinically important feature of the condition is that it is progressive and that the interval before diagnosis determines how much of that progression has occurred [10,11]. This review examines the presentation that most reliably brings these patients to attention, and what should be done with it [34].

Material and Methods

Search strategy: PubMed and the archives of the major gastroenterology, endoscopy and allergy journals were searched for studies of eosinophilic oesophagitis and oesophageal food impaction. Search terms combined eosinophilic oesophagitis with food impaction, food bolus obstruction, diagnostic delay, stricture, dilation, dupilumab, topical corticosteroid and elimination diet. Reference lists of position papers and guidelines [2,12] were screened by hand for primary sources.

Eligibility: Studies were included if they reported the prevalence of the condition among patients presenting with food impaction, quantified diagnostic delay or its consequences, or evaluated a treatment. Paediatric studies are cited where they establish disease behaviour and are identified as such.

Data extraction and handling: Design, setting, sample size, the histologic threshold used and the reported outcome were extracted as published. The remission definition is reported alongside every treatment result, because histologic, endoscopic and symptomatic remission diverge substantially in this condition and are not interchangeable. No pooling or re-analysis was performed. Figures 1 to 4 and Tables 1 and 2 reproduce published values only. This is a narrative synthesis and was not registered as a systematic review.

Results

The diagnosis is usually not known: In a cohort of 198 episodes of oesophageal food impaction, eosinophilic oesophagitis had been diagnosed beforehand in only 34 patients, 17.2 per cent (Figure 1) [13]. Median age was 51 years, 70.2 per cent were men, and half had atopic disease. Meat was the causative bolus in 75.3 per cent, and for three quarters of patients this was their first impaction; the remainder had already had one or more [13]. An impaction is therefore usually the presentation of an undiagnosed condition rather than a complication of a known one.

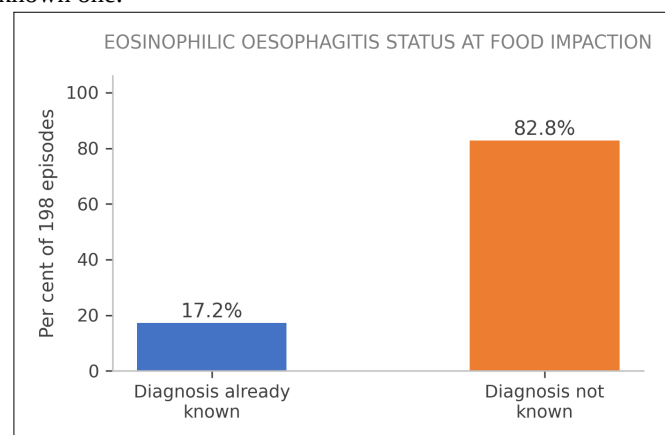


Figure 1: Proportion of oesophageal food impaction episodes occurring in patients with an established diagnosis [13]

Why the delay matters: Untreated disease progresses from an inflammatory to a fibrostenotic phenotype, and the risk of stricture rises by approximately 5 percentage points for each year the disease remains undiagnosed (Figure 4) [10,14]. Reduced oesophageal distensibility follows, and reduced distensibility is what makes an impaction possible [9,15]. Effective treatment can reverse tissue fibrosis in some patients and reduces the rate of impaction, but established stricture usually requires dilation [8,16], and the fibrostenotic phenotype persists after it [35].

What the treatments achieve: Reported histologic remission rates differ substantially by class: approximately 45 per cent for proton pump inhibitors, 80 to 93 per cent for swallowed topical corticosteroids, around 60 per cent for dietary elimination and around 60 per cent for dupilumab (Figure 2) [17,18,19,20]. Proton pump inhibitors are no longer used as a diagnostic exclusion but as a treatment in their own right [21,22]. Budesonide in an orodispersible formulation has randomised support and is licensed in Europe [18,23].

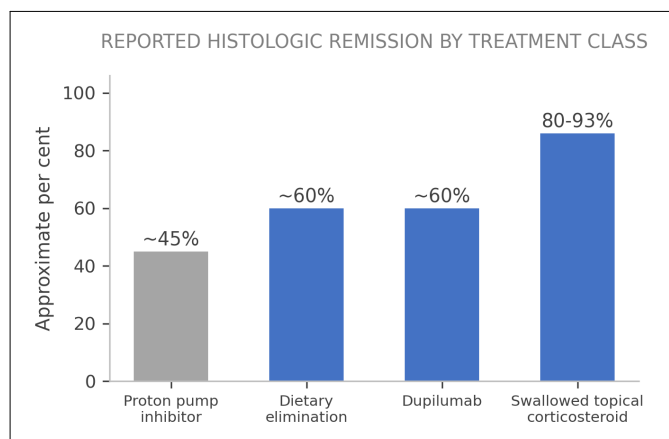


Figure 2: Reported histologic remission rates by treatment class [17,18,19,20]

The biologic: In the phase 3 randomised trial, histologic remission defined as 6 or fewer eosinophils per high-power field was achieved by 60 per cent of patients on weekly dupilumab against 5 per cent on placebo at 24 weeks, with a significant reduction in dysphagia on the Dysphagia Symptom Questionnaire (Figure 3) [19,24]. In the dosing comparison, only weekly administration produced significant symptomatic benefit against placebo [24]. Post-hoc analysis reported more days free of dysphagia at 24 weeks with dupilumab than placebo, 8.7 against 5.5 in a 14-day period, and a greater proportion free of chest pain and heartburn, 48.6 against 28.4 per cent, with effects maintained to 52 weeks [25,26].

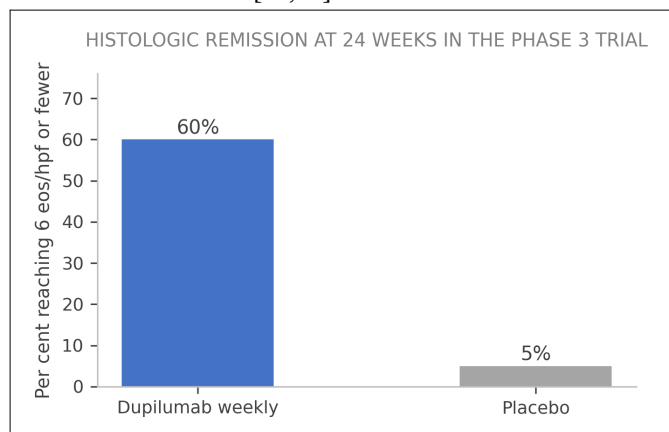


Figure 3: Histologic remission at 24 weeks in the phase 3 randomised trial [19,24]

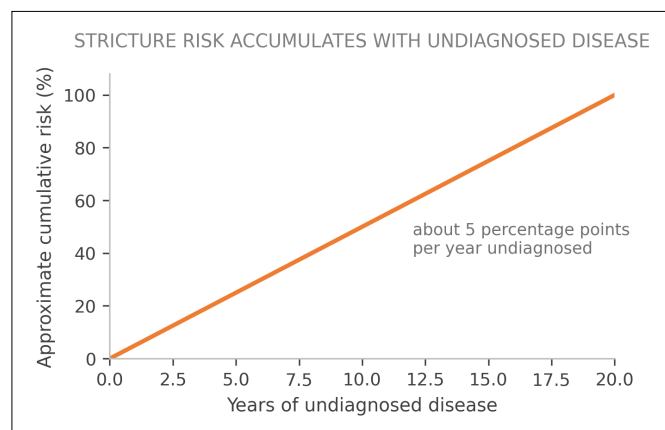


Figure 4: Accumulating stricture risk with each year of undiagnosed disease [10,14]

Study	Design and cohort	Principal reported finding
Impaction cohort [13]	198 oesophageal food impactions	Diagnosis already known in only 17.2%; meat in 75.3%
Dellon et al. 2022 [19]	Phase 3 RCT, dupilumab	Histologic remission 60% vs 5% at 24 weeks
Lucendo et al. 2019 [18]	RCT, budesonide orodispersible tablet	High histologic remission with topical corticosteroid
Lucendo et al. 2016 [17]	Systematic review, PPI response	Histologic remission in roughly half
Kliwer et al. 2023 [20]	RCT, one-food vs six-food elimination	Step-up dietary strategy compared directly
Schoepfer et al. 2013 [10]	Cohort, delay and phenotype	Stricture prevalence rises with duration of undiagnosed disease
Austrian position paper [2]	Society guidance on impaction	Sets out the endoscopic and biopsy pathway

Table 1: Principal studies reviewed, with the finding as reported by the original investigators

Dietary approaches: Elimination of dietary triggers achieves remission in roughly 60 per cent, and randomised comparison of a step-up strategy beginning with fewer foods against broader elimination showed that starting narrow avoids unnecessary restriction in a substantial proportion [20,27]. Allergy testing does not identify the responsible food reliably and is not recommended for that purpose [2,28]. Dietary treatment therefore requires sequential reintroduction with repeat endoscopy, which is demanding for the patient and requires dietetic support to avoid unnecessarily restricted intake [27,29].

What happens at the impaction: Endoscopy for a prolonged or painful impaction is an emergency procedure performed for symptomatic relief. It carries a risk of oesophageal injury and does nothing to the underlying disease [5,30]. The critical point is that biopsies taken at that endoscopy establish the diagnosis, and guidance recommends them irrespective of mucosal appearance, since the oesophagus can look normal [2,12,31]. Real-life audit shows this is frequently not done, which is how a patient can have several impactions without ever acquiring a diagnosis [13,32]. Management of impaction varies between gastroenterologists and that variation determines whether the condition is identified [33].

Step	What is recommended	What the evidence shows
Index impaction endoscopy	Take oesophageal biopsies regardless of appearance	Diagnosis known in only 17.2% beforehand [2,13]
Histologic threshold	15 or more eos/hpf with dysfunction	Standard diagnostic criterion [1,12]
Allergy testing	Not recommended to identify the food	Low sensitivity and specificity [2,28]
Induction therapy	PPI, topical steroid, diet or biologic	Remission 45% to 93% depending on class [17,18,19,20]
Dilation	For established fixed narrowing	Relieves obstruction; does not treat inflammation [16,30]
Long-term therapy	Required to prevent recurrence	Disease relapses on treatment cessation [8,23]

Table 2: The pathway from food impaction to long-term management and its evidential basis

Discussion

The therapeutic position in this condition is unusually comfortable. Four treatment classes induce histologic remission, one of them in more than four fifths of patients, and a biologic with randomised support is now licensed [17,18,19]. A patient who receives the diagnosis has a good chance of reaching remission on the first or second thing tried.

The problem is entirely upstream of that. In the impaction cohort, fewer than one in five patients arriving with a bolus stuck in the oesophagus already knew they had the disease [13]. Three quarters were having their first impaction, which means the remaining quarter had been through it before without the diagnosis being made. Since stricture risk accumulates at roughly 5 percentage points per undiagnosed year, the interval before diagnosis is not neutral waiting time; it is the period during which reversible inflammation becomes irreversible narrowing [10,14].

That places unusual weight on a single, cheap action. Biopsies taken at the index impaction endoscopy make the diagnosis, and guidance recommends them irrespective of how the mucosa looks, because a macroscopically normal oesophagus does not exclude the condition [2,12,31]. The procedure is already being performed for another reason, the patient is already sedated, and the marginal cost is a few seconds. Audit nonetheless shows the step is often omitted [13,32]. There are few situations in medicine where the gap between recommended and actual practice is this wide and this easily closed.

On choosing between treatments, the honest position is that the comparison is less settled than the individual remission rates suggest. The figures come from different populations, different histologic thresholds and different eras, and head-to-head randomised comparison between classes is limited [17,18,19,20]. Topical corticosteroids report the highest histologic remission but require indefinite use and carry local fungal infection risk; dietary elimination avoids drugs but demands repeated endoscopy and dietetic support; dupilumab has the cleanest randomised evidence and the highest cost. That choice

reasonably depends on the patient rather than on a ranking.

Study limitations

The limitations belong to the underlying literature. Remission rates are drawn from separate studies with differing histologic thresholds and populations, so the comparison between classes is indirect [17,18,19,20]. The impaction cohort is a single retrospective series with a third of screened patients excluded for incomplete records [13]. The estimate of stricture risk per undiagnosed year derives from cross-sectional association between symptom duration and phenotype, and symptom duration is patient-recalled [10,14]. Histologic remission is a surrogate: it correlates imperfectly with symptoms, since patients adapt their eating in ways that mask dysphagia [5,29]. Trial follow-up is typically 24 to 52 weeks against a lifelong condition [19,24]. Dietary studies cannot be blinded. And prevalence and access data come almost entirely from high-income settings [3,4].

Conclusion

An adult presenting with a food bolus stuck in the oesophagus has, on the available evidence, about a four in five chance of having eosinophilic oesophagitis that nobody has yet diagnosed. The treatments that follow diagnosis work: most patients achieve histologic remission on a proton pump inhibitor, a topical corticosteroid, dietary elimination or a biologic. What determines their oesophagus in ten years is not which of those is chosen but how many years pass before one of them is started, because stricture accumulates while the diagnosis is missed. The action that matters is therefore the least sophisticated one in this review: take biopsies at the endoscopy you are already performing, whatever the mucosa looks like.

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Conflict of Interest

The authors declare no competing interests.

Ethics Statement

This article reviews previously published studies only. No new patient data were collected and no participants were recruited, so ethical approval was not required.

References

1. Dellon ES, Liacouras CA, Molina-Infante J, Furuta GT, Spergel JM, Zevit N, et al. "Updated international consensus diagnostic criteria for eosinophilic esophagitis: proceedings of the AGREE conference". *Gastroenterology* 155(2018):1022-1033.
2. Riedl D, Kiesslich R, Madisch A, Miehke S, Schoppmann SF, Tilg H, et al. "Diagnosis and management of eosinophilic esophagitis and esophageal food impaction in adults: a position paper issued by the Austrian Society of Gastroenterology and Hepatology". *Wien Klin Wochenschr* (2024).
3. Arias A, Perez-Martinez I, Tenias JM, Lucendo AJ. "Systematic review with meta-analysis: the incidence and prevalence of eosinophilic oesophagitis in children and adults in population-based studies". *Aliment Pharmacol Ther* 43(2016):3-15.
4. Dellon ES, Hirano I. "Epidemiology and natural history of eosinophilic esophagitis". *Gastroenterology* 154(2018):319-332.
5. Lucendo AJ, Molina-Infante J, Arias A, von Arnim U, Bredenoord AJ, Bussmann C, et al. "Guidelines on eosinophilic esophagitis: evidence-based statements and recommendations for diagnosis and management in children and adults". *United European Gastroenterol J* 5(2017):335-358.
6. Hirano I, Moy N, Heckman MG, Thomas CS, Gonsalves N, Achem SR. "Endoscopic assessment of the oesophageal features of eosinophilic oesophagitis: validation of a novel classification and grading system". *Gut* 62(2013):489-495.
7. Dellon ES, Cotton CC, Gebhart JH, Higgins LL, Beitia R, Woosley JT, et al. "Accuracy of the eosinophilic esophagitis endoscopic reference score in diagnosis and determining response to treatment". *Clin Gastroenterol Hepatol* 14(2016):31-39.
8. Straumann A, Conus S, Degen L, Frei C, Bussmann C, Beglinger C, et al. "Long-term budesonide maintenance treatment is partially effective for patients with eosinophilic esophagitis". *Clin Gastroenterol Hepatol* 9(2011):400-409.
9. Nicodeme F, Hirano I, Chen J, Robinson K, Lin Z, Xiao Y, et al. "Esophageal distensibility as a measure of disease severity in patients with eosinophilic esophagitis". *Clin Gastroenterol Hepatol* 11(2013):1101-1107.
10. Schoepfer AM, Safroneeva E, Bussmann C, Kuchen T, Portmann S, Simon HU, et al. "Delay in diagnosis of eosinophilic esophagitis increases risk for stricture formation in a time-dependent manner". *Gastroenterology* 145(2013):1230-1236.
11. Dellon ES, Kim HP, Sperry SLW, Rybnicek DA, Woosley JT, Shaheen NJ. "A phenotypic analysis shows that eosinophilic esophagitis is a progressive fibrostenotic disease". *Gastrointest Endosc* 79(2014):577-585.
12. Hirano I, Chan ES, Rank MA, Sharaf RN, Stollman NH, Stukus DR, et al. "AGA institute and the joint task force on allergy-immunology practice parameters clinical guidelines for the management of eosinophilic esophagitis". *Gastroenterology* 158(2020):1776-1786.
13. Impaction Cohort Study Group. "Wide discrepancy between best practice recommendations and real-life management of suspected eosinophilic esophagitis-associated food bolus impaction". *Endosc Int Open* (2025).
14. Bek MK, Straumann A, Schoepfer AM. "Eosinophilic esophagitis: diagnosis, monitoring and treatment, a contemporary review". *Aliment Pharmacol Ther* (2025).
15. Carlson DA, Hirano I, Zalewski A, Gonsalves N, Lin Z, Pandolfino JE. "Improvement in esophageal distensibility in response to medical and diet therapy in eosinophilic esophagitis". *Clin Transl Gastroenterol* 8(2017):e119.
16. Dougherty M, Runge TM, Eluri S, Dellon ES. "Esophageal dilation with either bougie or balloon technique as a treatment for eosinophilic esophagitis: a systematic review and meta-analysis". *Gastrointest Endosc* 86(2017):581-591.
17. Lucendo AJ, Arias A, Molina-Infante J. "Efficacy of proton pump inhibitor drugs for inducing clinical and histologic remission in patients with symptomatic esophageal eosinophilia: a systematic review and meta-analysis". *Clin Gastroenterol Hepatol* 14(2016):13-22.
18. Lucendo AJ, Miehke S, Schlag C, Vieth M, von Arnim U, Molina-Infante J, et al. "Efficacy of budesonide orodispersible tablets as induction therapy for eosinophilic esophagitis in a randomized placebo-controlled trial". *Gastroenterology* 157(2019):74-86.
19. Dellon ES, Rothenberg ME, Collins MH, Hirano I, Chehade M, Bredenoord AJ, et al. "Dupilumab in adults and adolescents with eosinophilic esophagitis". *N Engl J Med* 387(2022):2317-2330.
20. Kliever KL, Gonsalves N, Dellon ES, Katzka DA, Abonia JP, Aceves SS, et al. "One-food versus six-food elimination diet therapy for the treatment of eosinophilic oesophagitis: a multicentre, randomised, open-label trial". *Lancet Gastroenterol Hepatol* 8(2023):408-421.
21. Molina-Infante J, Bredenoord AJ, Cheng E, Dellon ES, Furuta GT, Gupta SK, et al. "Proton pump inhibitor-responsive oesophageal eosinophilia: an entity challenging current diagnostic criteria for eosinophilic oesophagitis". *Gut* 65(2016):524-531.

22. Gomez-Torrijos E, Garcia-Rodriguez R, Castro-Jimenez A, Rodriguez-Sanchez J, Mendez Diaz Y, Molina-Infante J. "The efficacy of step-down therapy in adult patients with proton pump inhibitor-responsive oesophageal eosinophilia". *Aliment Pharmacol Ther* 43(2016):534-540.
23. Straumann A, Lucendo AJ, Miehlke S, Vieth M, Schlag C, Biedermann L, et al. "Budesonide orodispersible tablets maintain remission in a randomized, placebo-controlled trial of patients with eosinophilic esophagitis". *Gastroenterology* 159(2020):1672-1685.
24. Rothenberg ME, Dellon ES, Collins MH, Bredenoord AJ, Hirano I, Peterson KA, et al. "Efficacy and safety of dupilumab up to 52 weeks in adults and adolescents with eosinophilic oesophagitis (LIBERTY EoE TREET study): a multicentre, double-blind, randomised, placebo-controlled, phase 3 trial". *Lancet Gastroenterol Hepatol* 8(2023):990-1004.
25. Chehade M, Dellon ES, Spergel JM, Collins MH, Rothenberg ME, Pesek RD, et al. "Dupilumab improves dysphagia, odynophagia, chest pain, and heartburn in adult and adolescent patients with eosinophilic esophagitis up to 52 weeks: post hoc analysis of the LIBERTY EoE TREET study". *J Can Assoc Gastroenterol* (2025).
26. Dellon ES, Collins MH, Rothenberg ME, Hirano I, Chehade M, Bredenoord AJ, et al. "Dupilumab improves histopathologic features in eosinophilic esophagitis patients: LIBERTY EoE TREET study results". *Clin Gastroenterol Hepatol* (2025).
27. Molina-Infante J, Arias A, Alcedo J, Garcia-Romero R, Casabona-Frances S, Prieto-Garcia A, et al. "Step-up empiric elimination diet for pediatric and adult eosinophilic esophagitis: the 2-4-6 study". *J Allergy Clin Immunol* 141(2018):1365-1372.
28. Philpott H, Nandurkar S, Royce SG, Thien F, Gibson PR. "Allergy tests do not predict food triggers in adult patients with eosinophilic oesophagitis: a comprehensive prospective study using five modalities". *Aliment Pharmacol Ther* 44(2016):223-233.
29. Safroneeva E, Straumann A, Coslovsky M, Zwahlen M, Kuehni CE, Panczak R, et al. "Symptoms have modest accuracy in detecting endoscopic and histologic remission in adults with eosinophilic esophagitis". *Gastroenterology* 150(2016):581-590.
30. Chang JW, Rubenstein JH, Mellinger JL, Rubenstein JH, Dellon ES. "Esophageal food impaction and eosinophilic esophagitis: a systematic review of management and outcomes". *Dis Esophagus* 32(2019):doz002.
31. Nielsen JA, Lager DJ, Lewin M, Rendon G, Roberts CA. "The optimal number of biopsy fragments to establish a morphologic diagnosis of eosinophilic esophagitis". *Am J Gastroenterol* 109(2014):515-520.
32. Sperry SLW, Crockett SD, Miller CB, Shaheen NJ, Dellon ES. "Esophageal foreign-body impactions: epidemiology, time trends, and the impact of the increasing prevalence of eosinophilic esophagitis". *Gastrointest Endosc* 74(2011):985-991.
33. Hiremath G, Vaezi MF, Gupta SK, Acra S, Dellon ES. "Management of esophageal food impaction varies among gastroenterologists and affects identification of eosinophilic esophagitis". *Dig Dis Sci* 63(2018):1428-1437.
34. Straumann A, Katzka DA. "Diagnosis and treatment of eosinophilic esophagitis". *Gastroenterology* 154(2018):346-359.
35. Runge TM, Eluri S, Cotton CC, Burk CM, Woosley JT, Shaheen NJ, et al. "Outcomes of esophageal dilation in eosinophilic esophagitis: safety, efficacy, and persistence of the fibrostenotic phenotype". *Am J Gastroenterol* 111(2016):206-213.

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