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ORIGINAL ARTICLE

Gastroenterology: Eosinophilic Gastrointestinal Disorders

Manometric findings in children with eosinophilic esophagitis and persistent post-remission dysphagia

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Abstract

Objectives: Dysphagia is a frequent symptom of active eosinophilic esophagitis (EoE), but at times it persists despite attaining histologic healing and lack of fibro-stenotic changes. We aimed to describe the manometric findings in this subset of patients.

Methods: A retrospective review of charts between 2013 and 2023 at a tertiary pediatric gastroenterology center, treating roughly 1500 EoE patients per year. We included children with EoE referred to high-resolution impedance manometry (HRIM) for persistent dysphagia despite histologic healing (i.e., <15 eosinophils/high-power field [Eos/hpf]). Data including initial EoE diagnosis, endoscopy reports, esophageal biopsies, treatment regimens, and HRIM were retrospectively collected.

Results: The estimated prevalence of post-remission dysphagia in our cohort was exceedingly rare (<0.05%). Four patients met the eligibility criteria of histologic remission and absence of fibro-stenotic features on endoscopic evaluation and thus, were included in this case series. Patients achieved remission with steroids, proton-pump inhibitor, or both within a median time of 5 months from diagnosis. Peak Eosinophil count at remission was ≤5 Eos/hpf in three patients and ≤10 Eos/hpf in one. On HRIM, all four patients had a hypomotile esophagus and abnormal bolus clearance. Lower esophageal sphincter integrated relaxation pressure values were normal in three patients and elevated in one. Two patients were diagnosed with ineffective esophageal motility, one with aperistalsis and one with achalasia type 1.

Conclusions: Post-remission dysphagia is rare in EoE. Esophageal dysmotility with a hypomotile pattern may contribute to the persistent dysphagia in children with EoE. HRIM should be considered in patients with EoE in whom symptoms persist despite histologic remission.

KEYWORDS

dysmotility, EoE, esophagus, manometry

1 | INTRODUCTION

Over the past few decades, eosinophilic esophagitis (EoE) has emerged as the leading cause of food impaction and dysphagia in children.[1] Several mechanisms have been implicated in the pathophysiology of esophageal dysmotility in EoE including tissue remodeling, subepithelial fibrosis, and smooth muscle hypertrophy.[2,3] With early diagnosis and adequate treatment, most children can achieve both resolution of symptoms and histologic remission.[1] However, a small subset of patients may experience "post-remission

dysphagia" that is, persistent dysphagia despite histologic remission and absence of fibrotic changes.[4] While dysmotility patterns secondary to active EoE have been described, the manometric findings in post-remission dysphagia have not been previously described in children or adults. Recent studies in adults, incorporating endoluminal functional lumen imaging (Endoflip), have also included patients in histologic remission.[5] We therefore aimed to report high-resolution impedance manometry (HRIM) findings in children with EoE in histological remission, who were evaluated for post-remission dysphagia.

2 | MATERIALS AND METHODS

We retrospectively reviewed the Electronic Medical Record of Cincinnati Children's Hospital Medical Center over a 10-year period (July 2013–June 2023) to retrieve data on children younger than 18 years old who underwent HRIM for persistent dysphagia after histologic remission of EoE and lack of endoscopic features of esophageal stenosis. Data regarding demographics, symptoms, esophagogastroduodenoscopy (EGD) findings, barium studies, and HRIM were manually retrieved.

HRIM studies were performed using Laborie (Medical Measurement System, Enscheda) systems. Solid state 36 channel, 1 cm spaced catheters were used for all patients. No sedation or anesthetic agent was administered before performing the manometry studies. Metrics calculated included: Pharyngeal contractile integral (PhCI), upper esophageal sphincter (UES) relaxation time and integrated relaxation pressure (IRP), proximal contractile integral (PCI), distal contractile integral (DCI), lower esophageal sphincter (LES) IRPs, and LES baseline pressure.

The first EGD with biopsies taken from the distal and proximal esophagus showing esophageal inflammation with a peak eosinophil count (PEC) ≥ 15/high-power field (hpf) was taken as the initial diagnosis of EoE. Histologic findings other than PEC were also noted. We then searched for the first EGD after diagnosis with a PEC < 15/hpf to define the time of histologic remission. Endoscopic appearance of the EGD closest to the HRIM study was reviewed for abnormalities, including edema, furrowing, exudates, rings, and strictures. Patients were excluded if they were asymptomatic or if they were not in histologic remission (PEC < 15/hpf) on both proximal and distal esophageal biopsies taken closest to the manometry study. Barium studies were also reviewed for stenotic features. HRIM was performed following the published minimum standards.[6] Manometry tracings were manually reviewed to gather data on the relevant metrics. A manometric diagnosis was made based on the Chicago classification 4.0.[7]

The study was approved by the local institutional review board (study number 2019-1097).

What is Known

- Dysphagia is the hallmark of active eosinophilic esophagitis (EoE) and may be caused by inflammation, fibro-stenotic changes, and dysmotility.
- Resolution of dysphagia usually correlates with achieving histologic remission.

What is New

- Some patients with EoE may suffer from post-remission dysphagia, that is, dysphagia despite histologic remission and no evidence of fibrosis.
- Patterns of esophageal hypomotility including ineffective peristalsis, aperistalsis, and frank achalasia are observed in EoE patients who have achieved histologic remission and do not have fibro-stenotic changes.

3 | RESULTS

Over the 10-year period, only four patients were identified that met the inclusion criteria. Taking into account that about 1500 EoE patients are treated in our center per year, the estimated prevalence of post-remission dysphagia in our study was exceedingly rare (<0.05%). Median patient age was 14 years (range 12–17). Treatment regimens and histologic features are described in Table 1.

Patient 1 had a history of feeding difficulties as an infant and findings of eosinophilia in the esophagus at 4 years of age; however, these were considered secondary to reflux as they resolved on follow-up without any medication or food elimination. He had no symptoms over the following years but was eventually diagnosed with symptomatic EoE at the age of 14 years. *Patient 2* suffered from food impaction with a PEC < 8/hpf (without treatment), several months before he was definitively diagnosed with EoE. *Patient 3* had an upper gastrointesttinal (UGI) showing esophageal dilation and narrowing at the gastroesophageal junction, suspicious for achalasia, near the time of his EoE diagnosis, along with retained food in the esophagus on EGD. A repeat EGD 1 year after his original diagnosis still showed active EoE, and histologic remission was evident only at the time of the EGD and concomitant HRIM study in which he was definitively diagnosed with achalasia. *Patient 4* had a distal esophageal stricture at the initial diagnosis of EoE which responded to two dilations and was not observed in the following EGDs.

The median time to histologic remission on topical steroids, proton-pump inhibitors (PPI's) or both was 5 months (range 3–26 months, individual treatment

TABLE 1 Characteristics, findings on esophageal biopsies, and manometry of four patients for which high-resolution manometry was obtained while in histologic remission.

	1	2	3	4
EoE diagnosis				
Gender	F	M	M	M
Age	14	14	12	17
Peak eosinophile count	≤40/hpf	≤20/hpf	≥50/hpf	≥100/hpf
Additional histology	LPF VPE BLH	BLH, VPE	LPF	BLH, VPE, mild LPF
Treatment	Steroids, PPI	PPI	Steroids, PPI	Steroids, PPI
Time to remission	3	5	26	5
EGD closest to HRIM				
Endoscopic appearance	Normal	Mild furrowing	Distal esophagus dilation, thickened mucosa, retained food	Normal
Peak eosinophile count	≤2/hpf	0/hpf	≤5/hpf	≤10/hpf
Additional histology	Expansion of basal layer mild VPE	Normal	40% BLH 60% VPE	Mild BLH mild VPE
EGD to HRIM time	1	6	0	28
HRIM				
Time from diagnosis	21	11	26	55
DCI	508 (260–669)	319 (118–552)	0	0
DL	5.8 (4.6–7.1)	8.5 (7.4–10.2)	0	0
Brake size	5.1 (2.3–9.0)	3.5 (0–6.8)	—	—
normal wet swallows	31%	8%	0%	0%
Median IRP	5	8	35	7
Bolus clearance	Incomplete	Failed	Failed	Incomplete
Diagnosis	IEM (large breaks)	IEM	Achalasia type I	Aperistaltic esophagus

Note: Age in years; time intervals in months. Remission is defined as esophageal biopsies showing a peak eosinophil concentration <15/hpf. DCI is shown as average and range for wet swallows. IRP is shown as 4 s average[4]; diagnosis according to Chicago classification 4.0.

Abbreviations: BLH, basal layer hyperplasia; DCI, distal contractile integral; DL, distal latency; EGD, esophagogastroduodenoscopy; EOE, eosinophilic esophagitis; F, female; HRIM, high-resolution impedance manometry; IEM, ineffective esophageal motility; IRP, integrated relaxation pressure; LPF, lamina propria fibrosis; M, male; PPI, proton pump inhibitor; VPE, vascular papilla elongation.

data in Table 1). The interval between the last normal EGD and HRIM was a median of 3.5 months (range 0–28 months). Three patients had some degree of lamina propria fibrosis on their initial biopsy, but none had endoscopic or microscopic signs of fibrosis on the EGD closest to HRIM. Mucosal eosinophils on proximal and distal esophageal biopsies were completely absent in one of four patients on premanometry biopsies and were at or below 10/hpf for the other three patients.

All patients were evaluated by esophagram or upper GI contrast studies:

Patient 1 showed nonspecific delayed esophageal contractions and the presence of tertiary contractions suggestive of dysmotility; Patient 2 had normal esophagram except for gastroesophageal reflex; esophageal dilation with delayed clearance and residual contrast were noted in Patient 3; Patient 4 had dilatation of the upper and mid esophagus without narrowing and disorganized esophageal motility with delayed clearing of contrast from the esophagus. In all patients, there was no evidence of esophageal stenosis.

All four patients received a manometric diagnosis of hypomotile esophageal dysmotility (Figure 1). Two with ineffective esophageal motility (IEM) and two with aperistalsis (one with achalasia type 1, Patient 3). Abnormal bolus clearance was noted in all patients with incomplete bolus clearance in two patients (Patient 1 and Patient 4). Patient 2 had prolonged swallows (>6 s length), which is consistent with nonspecific esophageal motility disorder.[8] LES IRP values were normal in

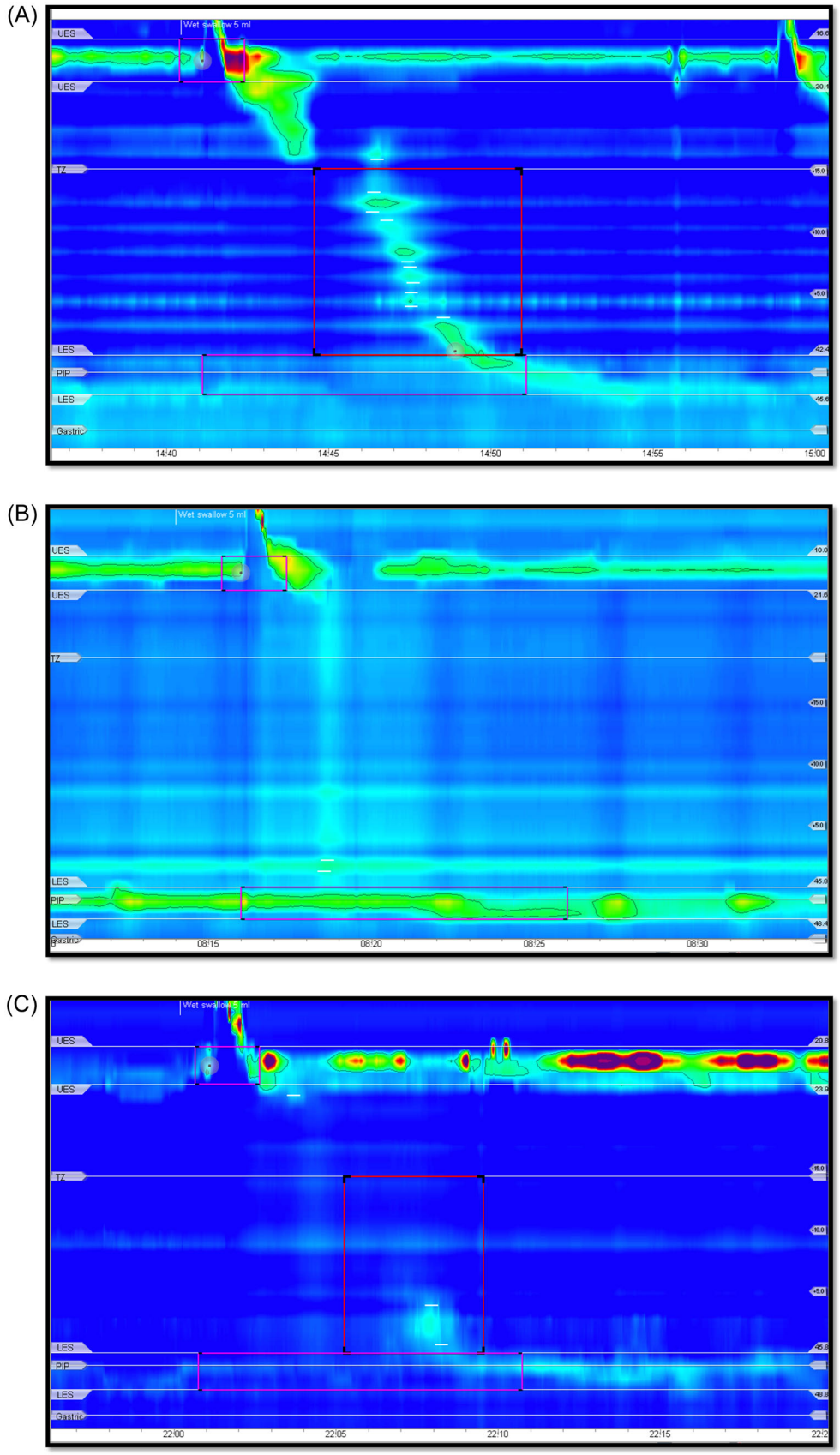

FIGURE 1 High-resolution manometry findings while in histologic remission. (A) Fourteen-year-old male with ineffective esophageal motility (Patient 2). (B) Twelve-year-old male with achalasia type 1 (Patient 3). (C) Seventeen-year-old male with aperistaltic esophagus (Patient 4). LES, lower esophageal sphincter; UES, upper esophageal sphincter.

three patients and elevated in Patient 3, who was diagnosed with Achalasia.

IEM was noted in Patient 1 with DCI within normal values but large esophageal breaks, while in Patient 2, IEM was diagnosed based on >70% of swallows with DCI values below 450 mmHg.

4 | DISCUSSION

We describe four patients with an established diagnosis of EoE who presented with post-remission dysphagia, despite achieving histological remission and absence of fibrotic changes. HRIM revealed a hypomotile pattern of esophageal dysmotility in all four, with IEM in two and aperistalsis in the other two. One of the two with an aperistaltic esophagus was diagnosed later with achalasia.

Measuring distensibility of the esophageal wall, as a surrogate marker for fibrosis, using Endoflip, would have provided critical information that could potentially explain the noted dysmotility. The lack of fibrosis or eosinophilic infiltration on superficial biopsies is intriguing and it may imply that other cellular mechanisms may contribute to the esophageal dysmotility. Indeed, at the level of the muscularis propria, eosinophilic degranulation products and activated mast cells have a causative role in smooth muscle hypertrophy and neuronal dysfunction.[3,9] Smooth muscle hypertrophy and mast cell infiltration have been associated with persistent symptoms in patients with resolution of eosinophilia.[2,10]

Studies applying endoscopic ultrasound (EUS) provide additional evidence of the inflammatory changes of the deeper muscular layers of the esophagus in EoE.[11,12] Fox et al. have shown increased thickness not just of the mucosa, but also of the submucosa and muscularis propria in children with EoE.[13] Selective longitudinal muscle dysfuntion has also been shown to contribute to dysphagia in patients with EoE.[14] In a case study, increased esophageal wall thickness observed by EUS persisted even after mucosal remission in response to topical steroids,[15] implying that even while mucosal biopsies may normalize, underlying muscular alterations may persist. In our study, all four patients achieved mucosal histologic remission within a relatively short period of time from diagnosis. However, diagnostic delay is a common problem in EoE.[16,17] It is therefore not unlikely that our patients suffered from prolonged asymptomatic esophageal inflammation before their initial diagnosis of EoE, allowing for submucosal tissue remodeling to slowly develop, eventually contributing to their esophageal dysmotility, despite resolution of mucosal eosinophilic infiltration.

As has been reported, endoscopic evaluation of fibrosis is somewhat limited as less than 50% of biopsies contain an adequate sample of the lamina propria for evaluation, even when using adult size biopsy forceps.[18] Additionally, as EoE is a patchy disease with intervening normal mucosa, sampling error can occur as well. In our study, three of our patients had evidence of lamina propria fibrosis on their initial biopsies but none on their final biopsies, but we cannot completely exclude that some underlying fibrosis could possibly persist. Additionally, our patients did not undergo endoluminal functional lumen imaging (Endoflip) which has been recently shown to correlate with fibrotic changes of the esophagus in pediatric patients.[19] Nonetheless, this case series highlights the fact that esophageal dysmotility can persist, even in the absence of endoscopic or histologic findings.

Dysmotility in active EoE has been previously described, and adult studies demonstrate variable prevalence and types of dysmotility findings. Martin et. al demonstrated pan-esophageal pressurization in 10/21 patients, peristaltic dysfunction in six patients, and normal peristalsis in five.[20] Findings of a hypercontractile esophagus (pan-esophageal pressurization, jackhammer esophagus) have been described in several studies of active EoE patients, and the rate of normal findings on manometry varies but may be as high as 75%.[3] In pediatric literature, Cheung et al. described 11 active EoE patients with normal manometry.[21] Contrary to this study, a more recent study showed that 7/17 active EoE pediatric patients had abnormal peristalsis including high amplitude contractions in the distal esophagus and ineffective peristalsis.[22] In a prospective adult study evaluating response to topical steroids, manometry detected motility disorders in 7/20 patients before treatment, with pan-esophageal pressurization being the most frequent finding.[23] These findings resolved after therapy in all but one patient, who had persistent dysmotility (frequent failed peristalsis) despite achieving mucosal remission and had had a 25-year history of symptoms, as well as endoscopic fibrostenotic changes. Contrary to these studies, our study evaluated dysphagia in patients who had achieved histologic remission and showed hypo-contractile esophageal dysmotility patterns which developed after a relatively short duration of active disease. We do not have long-term follow-up to determine if the dysphagia and dysmotility eventually resolved on continued therapy or persisted.

The co-occurrence of EoE and achalasia, as is the case for *Patient 3* in our series, is not without precedent. In an Italian study of newly diagnosed EoE patients undergoing HRIM, 8/109 were diagnosed with achalasia, and almost 15% suffered from obstructive motor disorders.[24] Our patient was given a formal manometric diagnosis of achalasia only after achieving histologic remission but had UGI and EGD findings suspicious of achalasia as early as his first EGD, and therefore it is likely that he initially suffered from both EoE and early achalasia. In a case report, Savarino et al. describe a patient with co-occurrence of achalasia

and esophageal eosinophilia (PEC > 50/hpf), that responded to oral steroids not just in terms of EoE, but also including complete recovery of peristalsis on manometry.[25] Contrary to this, our patient was given a definitive diagnosis of achalasia while the mucosal inflammation was in remission. While the eosinophilic infiltrate in achalasia may be secondary to food stasis, there is evidence that eosinophils play a role in axonal necrosis and motor damage, and this may account for the coexistence of the two disease processes.[3,26] Indeed a recent publication has coined the term "Mixed esophageal disease" implying that different esophageal processes may actually be parts of the same disease.[27]

Our study is limited by its retrospective nature and small number of patients. The fact that we did not find more patients, despite the large volume of EoE patients seen at our center (about 1500 pediatric patients with EoE each year) may indicate that dysphagia in resolved EoE is an uncommon phenomenon in pediatric patients, or perhaps underdiagnosed. We did not study all EoE patients treated in our facility who had persistent dysphagia despite histologic remission of EoE, but rather explored just those who were referred for manometry. This methodology creates a risk of referral bias, and it is possible that we have missed additional EoE patients with post-remission dysphagia referred to other centers or lost to follow-up. We also did not document the initial prevalence of dysphagia among EoE patients in our institute but as EoE is a clinicopathologic diagnosis we may safely assume that almost all patients were symptomatic to begin with. Additionally, our study was not built to show causality, and the hypomotility patterns observed could be secondary to a coexisting condition (like achalasia).

5 | CONCLUSION

Our study should encourage physicians to consider esophageal manometry in EoE patients in histologic remission without fibro-stenotic changes, who continue to have dysphagia. Our findings highlight a possible role for deeper neuromuscular changes as the underlying cause for these symptoms, even in the absence of apparent fibro-stenotic changes. Further studies are needed to investigate and understand the pathophysiological mechanisms underlying esophageal dysmotility in patients with EoE with persistent dysphagia despite histological remission.

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CONFLICT OF INTEREST STATEMENT

V. M. is consulting for Reneron, Shire/Takeda, and Sanofi. Adjudication board for Alladapt. The remaining authors declare no conflict of interest.

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