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Utility of Gastric and Duodenal Biopsies During Follow-up Endoscopy in Children With Eosinophilic Esophagitis

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Abstract

Objectives: Eosinophilic esophagitis (EoE) is a chronic antigen-mediated immune disorder of the esophagus. Consensus guidelines recommend obtaining esophageal, gastric, and duodenal biopsies at diagnostic endoscopy when EoE is suspected. The utility of repeated gastric and duodenal biopsies during follow-up endoscopy in patients previously diagnosed with EoE is not established. The aim of the present study was to explore the role of gastric and duodenal biopsies in children with an established diagnosis of EoE undergoing repeat endoscopy to assess histological response to treatment.

Methods: Retrospective chart review of children diagnosed with EoE at a tertiary care center was conducted. A total of 160 patients with EoE with demographic clinical, endoscopic, and histological data at diagnosis and follow-up endoscopy were included. The frequency of gastric and duodenal biopsies at follow-up endoscopy with abnormal histology and their correlation to endoscopic findings was determined.

Results: At follow-up endoscopy, 83% (132/160) of patients had gastric and 74% (118/160) had duodenal biopsies. Histology was normal in 81% of gastric and 92% of duodenal biopsies. The most frequent gastric abnormalities were chemical and inactive chronic gastritis. The most frequent duodenal abnormality was villous blunting with increased intraepithelial lymphocytes. Two patients with normal gastric and duodenal histology progressed to eosinophilic gastroenteritis at follow-up endoscopy.

Conclusions: Gastric and duodenal biopsies obtained in EoE patients during follow-up endoscopy show pathology in a minority of patients, increase costs, and may add potential risk of adverse events. Large multicenter, prospective studies of endoscopic practice during follow-up of EoE are warranted to provide evidence supporting best practices.

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Keywords

endoscopy; esophagus; extraesophageal biopsy

Eosinophilic esophagitis (EoE) is a chronic, dietary antigen-mediated immune disorder with increasing prevalence and significant morbidity in children. Incidence of EoE is 0.7 to 10/100,000 per person-year with a prevalence of up to 43/100,000 children (1). The current economic burden of EoE to the United States was estimated to be as high as 1.4 billion dollars (2). In the absence of other less or noninvasive diagnostic modalities, esophagogastroduodenoscopy (EGD) with esophageal biopsy remains the criterion standard and only proven modality to establish an EoE diagnosis and to subsequently assess disease activity.

A diagnosis of EoE is established in patients with chronic symptoms of esophageal dysfunction and >15 eosinophils/high-powered field (eos/HPF) in an esophageal biopsy in the absence of pathologic gastroesophageal reflux disease. Consensus guidelines recommend obtaining gastric and duodenal biopsies during initial diagnostic EGD to exclude other causes of esophageal eosinophilia and investigate noneosinophil-related sources of symptoms (3–5).

Follow-up EGD is routinely performed to evaluate the esophageal histologic response to treatment of EoE, but current guidelines are not established regarding the utility of repeated gastric and duodenal biopsies practices. Each subsequent EGD presents additional morbidity, including biopsy and anesthesia risks (in relation to the duration of the procedure), and increasing the cost of care for patients. The primary aim of the present study was to determine the frequency gastric and duodenal biopsies are obtained during follow-up endoscopy in children with EoE and whether this leads to additional diagnoses. Our secondary objective was to assess additional costs of extraesophageal biopsies during follow-up EGD in pediatric patients with EoE.

METHODS

Methods are available online as Supplemental Digital Content 1 (<http://links.lww.com/MPG/A891>).

RESULTS

Database review identified 160 patients with EoE that met inclusion criteria. The median age at diagnosis was 7 years (range 10 months to 17 years), and 73% were boys. Most common presenting complaints were abdominal pain, dysphagia, and vomiting (Table 1). Degree of esophageal eosinophilia at diagnosis is shown in Supplemental Digital Content 2, Table, <http://links.lww.com/MPG/A892>. During initial diagnostic endoscopy, 99% (159/160) of patients had gastric biopsies and 98% (157/160) had duodenal biopsies, consistent with consensus recommendations. Gastric and duodenal biopsies could not be obtained in 1 patient with an esophageal stricture. Two additional patients did not have duodenal biopsies for unspecified reasons. No pathology was identified in 82% (130/159) of gastric biopsies.

Chemical or inactive chronic gastritis was the most common diagnosis in 14% (22/159) of the gastric biopsies. Duodenal histology was normal in 89% (140/157) of patients who had duodenal biopsies taken. Pathologic duodenal histology was identified in 11% (17/157) of patients with villous blunting and increased intraepithelial lymphocytes (IELs) commonly identified (Table 2); the 7 patients (4%) with moderate villous blunting, crypt elongation, and IELs were all confirmed to have elevated tissue transglutaminase and celiac disease.

Median duration between initial and subsequent endoscopy was 4.8 months (range 6 weeks to 4.5 years). At follow-up endoscopy, 83% (132/160) of patients had gastric and 74% (118/160) had duodenal biopsies. Of patients with gastric biopsies during follow-up endoscopy, 81% (107/132) had normal histology. Abnormal gastric histology was identified in 19% (25/132) of these patients. The most frequent histological abnormalities were inactive chronic gastritis and chemical gastritis (Table 2). Of patients with duodenal biopsies during follow-up endoscopy, 92% (109/118) had normal histology. Pathologic duodenal histology was found in 8% (9/118). The most frequent histological abnormality was villous blunting with increased IELs (Table 2) associated with known celiac disease. Two patients were identified with new gastric and/or duodenal eosinophilia consistent with eosinophilic gastroenteritis (EGE). One of these 2 patients was an 11-year-old girl who presented with abdominal pain and was diagnosed with EoE based on the presence of dense esophageal eosinophilia with up to 80 eos/HPF with normal gastric and duodenal histology. Repeat EGD performed after 8 weeks of empiric 6 food elimination diet with persisting abdominal pain demonstrated a normal appearing esophagus and stomach, but a granular appearance to the duodenal mucosa. Biopsies demonstrated improvement in esophageal eosinophilia with the new finding in gastric biopsies of up to 40 eos/HPF with an increase in lamina propria plasma cells and lymphocytes. Duodenal histology showed mild to moderate non-specific villous thickening and increased IELs and eosinophils. The diagnosis was changed to EoE with EGE. The second patient was a 15-year-old boy with abdominal pain, diagnosed with EoE based on isolated esophageal eosinophilia with peak eosinophil counts of 100 eos/HPF and normal gastric and duodenal histology. Abdominal pain persisted despite treatment with 6 food elimination diet, and the follow-up EGD 2 months later demonstrated pyloric channel and duodenal bulb ulcers. Esophageal histology revealed 150 eos/HPF, and duodenal biopsies were notable for infiltration of the lamina propria with eosinophils and the presence of focal eosinophilic microabscesses. Gastric biopsies were normal. The diagnosis was changed to EoE with EGE.

To determine whether abnormal extraesophageal histology at initial EGD can be used to guide decision making during follow-up endoscopies, follow-up EGD histology was assessed in patients who had abnormal initial gastric or duodenal pathology. Twenty-six of 29 patients with abnormal gastric histology on initial EGD had gastric biopsies obtained at the time of follow-up EGD. Only 9 (35%) had abnormal gastric histology and of these, 7 had inactive chronic gastritis, and 2 had chemical gastritis. Seventeen patients had abnormal duodenal histology during initial EGD and of these, only 4 (23%) had abnormal findings on repeat EGD. These 4 patients were known to have celiac disease with varying degrees of persistent villous blunting, crypt elongation, and increased IELs that had variably persisted during the follow-up EGD. Thus, abnormal histology persisted in a minority of patients at follow-up endoscopy in both the stomach and duodenum.

In an effort to understand whether abnormal visual findings during follow-up endoscopy can guide whether to biopsy the stomach or duodenum, extra-esophageal biopsies in subjects with abnormal endoscopic findings were analyzed. Of the 107 patients in whom the visual gastric endoscopy findings during follow-up EGD were available, only 8 (7%) were visually abnormal with friable mucosa, nodularity, or erythema. Four of these 8 patients also had abnormal gastric histology: 3 with moderate, active chronic gastritis and 1 patient with inactive chronic gastritis. Of the 106 with documented visual duodenal endoscopic findings, only 6 had abnormal findings that included erosions, ulcer, and friable, nodular or granular mucosa. Two of these 6 patients had abnormal histology, and these were the 2 patients with EGE (marked increase in lamina propria eosinophils and focal eosinophil microabscesses).

Finally, we considered pathology costs/charges related to potentially unnecessary extraesophageal biopsies performed during follow-up EGD. At our institution, esophageal, gastric, and duodenal biopsies are collected in separate containers, and the hospital cost for processing pathology specimens per container is \$673. Medicaid reimbursement per container in Illinois is \$73.83 (6). During the 9 years of the present study (in patients limited to inclusion criteria), 250 extraesophageal biopsies (132 gastric and 118 duodenal biopsies) were performed resulting in pathology charges of \$168,205.

DISCUSSION

Consensus guidelines for the diagnosis of EoE recommend that gastric and duodenal biopsies be obtained during EGD to evaluate for other potential causes of persistent gastrointestinal symptoms. There are, however, no recommendation and limited evidence about the utility and need for repeating gastric and duodenal biopsies during follow-up EGD in those with known EoE undergoing either surveillance endoscopy or assessment following a treatment intervention. A number of non-and minimally invasive modalities have been studied to assess disease activity in EoE, and although some of them have shown promise, none are currently approved to assess esophageal mucosal healing. Histologic assessment of esophageal biopsies remains the criterion standard for assessing disease activity (7–11). The number of postdiagnostic EGDs varies depending on the type of treatment, ranging from a minimum of 1 for assessing histological response to topical steroid treatment or exclusive milk elimination diet to a range between 3 and 9 in children treated with either empiric elimination diets or allergy-directed diets (12,13).

In the present study, gastric and duodenal biopsies were routinely obtained during diagnostic EGD consistent with published guidelines. Within our institution, extraesophageal biopsies were obtained frequently during follow-up endoscopy. The gastric and duodenal biopsy rates at follow-up endoscopy, however, dropped, demonstrating variable practice within our own institution.

Gastric and duodenal biopsies obtained in the majority of patients at the time of follow-up EGD did not result in new pathologic diagnosis. Most of the abnormal gastric histology at follow-up was described as inactive chronic gastritis, an assessment that did not lead to a change in clinical management. Sixty-five percent of patients with abnormal initial gastric histology had normal gastric histology at follow-up EGD, demonstrating that initial

abnormal gastric histology was not helpful in guiding the decision to biopsy the stomach during follow-up EGD. At the time of initial EGD, 4% of patients in our cohort had biopsy findings and an elevated tissue transglutaminase consistent with celiac disease, an association with EoE first reported by Kagalwalla et al (14–16) in 2007, which continues to be described in more recent literature. The most common abnormal duodenal histology at follow-up EGD was persistent histologic changes consistent with known celiac disease. No new cases of celiac disease were identified at follow-up EGD. Thus, abnormal initial duodenal histology was not helpful to guide whether to biopsy, as the only pathology noted was persistent changes of celiac disease. As noted in Table 2, there were 3 patients with nonspecific eosinophilia noted on initial gastric biopsy, which did not reach significance to be diagnosed with EGE. One patient with an increase in gastric eosinophils, occasional IELs and intraepithelial eosinophils on initial EGD had normal gastric histology at follow-up. The second patient with 1 small focus of eosinophilia in the gastric lamina propria and mild eosinophilic cryptitis on initial EGD had inactive chronic gastritis at follow-up, and the third patient with focally increased duodenal eosinophils on initial EGD had normal duodenal histology at follow-up. Taken together, data suggest follow-up endoscopy with initial normal or abnormal gastric or duodenal histology typically did not yield new information that could change the diagnosis or alter management. Although the nonspecific gastric and duodenal eosinophilia in four patients at the time of initial EGD resolved, we still would consider it prudent to rebiopsy these patients at follow-up to assess whether eosinophilia progressed to changes consistent with EGE.

Next, we assessed whether visual gastric and duodenal endoscopic findings could guide biopsy practice during follow-up endoscopy. Visual endoscopic findings did not always correlate with pathologic histology, which, when present, was diagnosed either as nonspecific active or chronic inflammation without an increase in eosinophils. Although the presence of mild nonspecific gastritis did not change the diagnosis or management, 2 patients who had normal visual and histologic findings during the initial EGD had abnormal visual changes with histologic changes at follow-up EGD consistent with EGE. These 2 were also patients who had persistent abdominal pain. It is possible that these patients had EGE on initial EGD that was missed secondary to sampling bias. The more likely explanation is, however, the presence of new visual abnormal findings in the duodenum is consistent with progression of disease outside the esophagus to involve the duodenum. In a previously reported small EoE cohort, 19% of all stomach biopsies and 21% of all duodenal biopsies were found to have mucosal eosinophilia (17). That study failed to delineate whether abnormalities were present at diagnosis or follow-up EGD and did not differentiate clinically meaningful abnormalities from nonspecific findings that did not alter clinical decision making. Although uncommon, eosinophilic gastrointestinal disorders should be considered in patients with poor response to EoE-directed therapy especially those with persistent symptoms of abdominal pain, diarrhea, or persistent peripheral eosinophilia (18). In the context of these data and our 2 patients diagnosed with eosinophilic gastrointestinal disorders at follow-up EGD, it seems reasonable to perform gastric and duodenal biopsies if initial symptoms persist or abnormal visual findings are noted during subsequent EGD, especially if the visual abnormality is present in the duodenum.

Cost related to performing a procedure is another important consideration given the economic burden of healthcare. Procedural cost varies by year, geography, institution, and payer mix, making estimates of EGD economic burden difficult to determine and generalize from a specific cohort to a larger population. By eliminating unnecessary gastric and duodenal biopsies, hospital costs potentially would decrease by \$1346 (stomach and duodenum) and Medicaid payments decrease by \$147 per patient per follow-up EGD. Although the number of follow-up endoscopies per year varies depending on the type of treatment, many patients with EoE undergo repeated EGDs as part of their treatment and monitoring. The reported incidence of EoE is 0.7 to 10 per 100,000 children per year (1) and the US population of children younger than 18 years, per the US census bureau, was 73,728,088 as of July 2012 (19). Using above incidence numbers, hospital charge estimates, and the presumption of 3 follow-up EGDs in the first year after EoE diagnosis, elimination of gastric and duodenal biopsies would decrease Medicaid reimbursement by up to \$3,250,000 annually. Furthermore, the time spent obtaining additional gastric and duodenal biopsies increases the overall total procedure time and the time the child is under anesthesia. Anesthesia services are computed in 15 minute units (20) and thus eliminating unnecessary extraesophageal biopsies could potentially keep anesthesia charges more consistently within a single 15-minute unit. Although this may have little relevance to the experienced endoscopist, centers in which the majority of procedures are performed by fellows in training may save an anesthesia unit. This extra time under anesthesia is a potential concern since animal studies have shown that factors influencing the extent of brain injury include age at the time of drug exposure and cumulative anesthesia dose (21). Thus, judicious decision making as it pertains to obtaining gastric and duodenal biopsies could decrease the duration and amount of anesthesia, which in turn could reduce anesthesia costs and limit potential anesthesia-related neurotoxicity. The effect of biopsy guidelines on anesthesia time is another factor that would have to be assessed prospectively.

The first limitation of our study is the retrospective nature of data collection and the fact that gastric and duodenal biopsies were not done in all patients during follow-up EGD. Secondly, visual endoscopic assessments were not standardized and data collection was dependent on prior physician reports, and thus subject to bias. Thirdly, the data regarding progression of disease extended to only 1 follow-up EGD. It is quite possible that subsequent EGDs performed over a longer period of time could identify additional abnormal findings in the stomach and duodenum that would modify the diagnosis and possibly alter the treatment. Finally, because proton pump inhibitor usage was not required for diagnosis of EoE before 2011 consensus guidelines, a minority of our patients may have had proton pump inhibitor-responsive esophageal eosinophilia. The question as to the utility of extraesophageal biopsies, however, remains similar and relevant to this population as well.

We have found that gastric and duodenal biopsies obtained during follow-up EGD in children with EoE have limited utility. Furthermore, abnormal extraesophageal histology at initial EGD was a poor guide to direct subsequent gastric and duodenal biopsies because the histologic abnormality resolved in a majority of patients. The presence of persistent gastrointestinal symptoms or abnormal visual findings during follow-up EGD support obtaining gastric and duodenal biopsies to, however, exclude the possibility of progression to EGE in those previously diagnosed with EoE. Routine gastric and duodenal biopsies

obtained in the absence of the above mentioned situations during follow-up EGD potentially introduce unnecessary risk and cost. Prospective multicenter studies of endoscopy practice and utility in patients with EoE are needed to validate these findings and facilitate specific guidelines and recommendations regarding advisability of obtaining additional biopsies from stomach and duodenum in children with EoE.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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What Is Known

- Esophagogastroduodenoscopy is required to diagnose eosinophilic esophagitis.
- Consensus guidelines recommend obtaining gastric and duodenal biopsies during initial diagnostic esophagogastroduodenoscopy when eosinophilic esophagitis is considered to exclude other causes of esophageal eosinophilia and investigate non-eosinophil-related sources.
- Current guidelines are not established regarding the utility of repeated gastric and duodenal biopsies during follow-up esophagogastroduodenoscopy.

What Is New

- Variable practice exists regarding extraesophageal biopsy practice in those with established diagnosis of eosinophilic esophagitis undergoing repeat esophagogastroduodenoscopy.
- Gastric and duodenal biopsies obtained during follow-up esophagogastroduodenoscopy result in new pathologic diagnosis in a minority of patients.
- Persistent symptoms or abnormal visual findings during follow-up esophagogastroduodenoscopy support obtaining extraesophageal biopsies to exclude the possibility of progression from eosinophilic esophagitis to eosinophilic gastroenteritis.

TABLE 1.

Demographics and presenting symptoms at diagnostic esophagogastroduodenoscopy

n = 160	n (%)
Male	117 (73)
Median age, years (range)	7 (1–17)
Presenting symptoms	
Dysphagia	46 (29)
Abdominal pain	41 (25)
Vomiting	36 (22)
Weight loss/poor weight gain	20 (12)
Heartburn/reflux	11 (7)
Food impaction	8 (5)
Dyspepsia	7 (4)
Chest pain	2 (1)
Unspecified	9 (6)

TABLE 2.
Pathologic findings at the time of diagnostic and follow-up esophagogastroduodenoscopy

	Histology	Diagnostic EGD	Follow-up EGD
Stomach		29	25
	Total abnormal		
	Chemical gastritis	12	6
	Inactive chronic gastritis	10	14
	Lymphocytic gastritis	1	0
	Marked increase in lamina propria plasma cells and intraepithelial lymphocytes	1	0
	Active chronic gastritis	1	3
	Helicobacter pylori gastritis	1	0
	Mild eosinophilia in lamina propria and occasional intraepithelial lymphocytes and intraepithelial eosinophils	1 [*]	0
	Increased eosinophils at mucosal base	1 [*]	1 [*]
	Small focus of eosinophilia in lamina propria and mild eosinophilic cryptitis	1 [*]	0
	Increased eosinophils (40 eosinophils/high-powered field) with mild nonspecific increase in lamina propria plasma cells and lymphocytes	0	1
Duodenum		17	9
	Total abnormal		
	Moderate villous blunting, crypt elongation, intraepithelial lymphocytes	7 [‡]	4 [‡]
	Mild-moderate villous blunting, increased intraepithelial lymphocytes	4	0
	Patchy moderate villous blunting, increased intraepithelial lymphocytes	0	1 [‡]
	Epithelial lymphocytosis with no villous blunting	0	1 [‡]
	Peptic duodenitis	4	1
	Focally increased lamina propria eosinophils	1 [*]	0
	Marked increase in lamina propria eosinophils and focal eosinophilic microabscesses	0	1
	Mild to moderate villous thickening and increase in lamina propria lymphocytes/eosinophils	0	1
	Yeast from duodenal ulcer	1	0

EGD = esophagogastroduodenoscopy.

* Patients not diagnosed with eosinophilic gastroenteritis.

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[‡]Diagnosed/known celiac disease.

[‡]Findings resolved on third follow-up endoscopy, not diagnosed with celiac.