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Dietary Elimination Therapy is an Effective Option for Adults with Eosinophilic Esophagitis

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Abstract

Background & Aims—Eosinophilic esophagitis (EoE) is an immune-mediated disorder. Food elimination is an established treatment for children, but data in adults are limited. We aimed to determine the response of adults with EoE to dietary therapy.

Methods—This was a retrospective cohort study using the University of North Carolina EoE database from 2006–2012. Subjects were ≥18 years, had EoE by consensus guidelines, and had undergone dietary therapy either with targeted or six-food elimination (SFED). Outcomes were symptomatic, endoscopic, and histologic improvement. Demographic, endoscopic, symptomatic, and laboratory predictors of response to dietary therapy were assessed.

Results—Of 31 adults who underwent dietary therapy (mean age 36 years; 48% male; 90% white; mean baseline eosinophil count 78 eos/hpf), 22 had targeted and 9 had SFED. Symptoms improved in 71% (68% in targeted, 78% in SFED) and endoscopic appearance improved in 54% (53% in targeted, 56% in SFED). After dietary therapy, the mean eosinophil count decreased to 43 eos/hpf (p=0.009). Eleven subjects (39%) responded with <15 eos/hpf (32% in targeted and 56% in SFED; p=0.41). No clinical, endoscopic, or histologic factors predicted response to dietary

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therapy. Of the 11 responders, 9 underwent food reintroduction to identify trigger(s), and 4 (44%) reacted to dairy, 4 (44%) to eggs, 2 (22%) to wheat, 1 (11%) to shellfish, 1 (11%) to legumes, and 1 (11%) to nuts.

Conclusions—Dietary elimination is a successful treatment modality for adults with EoE. Further research should emphasize which factors can predict effective dietary therapy.

Keywords

eosinophilic esophagitis; dietary therapy; targeted elimination diet; six food elimination diet

Introduction

Eosinophilic esophagitis (EoE) is a chronic, immune mediated disorder of the esophagus defined symptomatically by esophageal dysfunction and pathologically by eosinophil infiltration into the esophageal mucosa, in the absence of competing causes of esophageal eosinophilia.[1–3] There are data to support roles for both aeroallergens and food allergens in the etiology of EoE.[4] Evidence for aeroallergens as a causative agent comes from animal models as well as seasonal and geographic variation in the diagnosis of EoE.[4–7] The role of food allergens is supported by the success of dietary elimination therapy in the pediatric population.[2, 8–11]

While swallowed corticosteroids act topically to reduce esophageal inflammation and are frequently used for pharmacologic treatment of EoE,[12–14] no corticosteroid is FDA approved, not all patients respond,[14] and when discontinued, EoE almost always recurs.[15, 16] Therefore, dietary elimination therapy is an attractive option as either initial therapy or second-line treatment. The three main dietary modalities are an elemental formula devoid of allergens, the six-food elimination diet (SFED) centered on the removal of dairy, wheat, nuts, eggs, seafood, and soy, and targeted elimination where foods identified by allergy testing or patient report are removed.[2] While these approaches have been used with success in children,[8–11, 14] there are fewer data in adults.[17–20] In this population, the SFED and elemental diet appear to be efficacious,[21–23] but do not always produce reliable symptomatic or histologic improvement.[17, 21]

The aim of this study was to review our center's experience with dietary elimination therapy for treatment of EoE in adults, to determine the clinical and histologic response rates, and to analyze predictors of response to dietary elimination. We hypothesized that dietary elimination was effective in our patient population.

Methods

This was a retrospective cohort analysis of patients at University of North Carolina (UNC) Hospitals from 2006–2012. Cases of EoE, diagnosed as per consensus guidelines,[1, 2] were identified from the UNC EoE Clinicopathologic database. The details of this database have been previously described.[24, 25] For inclusion, patients had to be 18 or older and have undergone dietary elimination therapy for the treatment of EoE. Patients who were treated continuously with the combination of swallowed steroids and dietary therapy were excluded

because the effect of diet could not be isolated, but patients who received a course of dietary elimination as monotherapy were included, even if they had used steroids previously. Our standard practice was to allow four weeks off steroids prior to the initiation of dietary therapy, and given that dietary therapy was maintained for six weeks, at least 10 weeks passed after the discontinuation of steroid therapy prior to evaluating the effect of dietary therapy.

Two modalities for dietary elimination therapy that were used in standard practice at UNC were assessed retrospectively. The first was targeted elimination therapy. For this, patients underwent evaluation with skin prick testing, and foods that had a positive reaction, as well as any foods identified by patient self-report as being possible triggers regardless of the severity of response, were eliminated. The second was SFED. For this diet, dairy, wheat, eggs, soy, nuts, and seafood were eliminated from the diet regardless of skin-prick test results. The skin-prick testing and dietary elimination were performed using a general approach previously described by Kagawalla and colleagues and Gonsalves and colleagues.[10, 18, 19] In our practice, patients undergo a 6 week period of food elimination followed by an upper endoscopy to assess histologic response. For those subjects who responded to elimination, one food (or food group) was added back for 6 weeks, and endoscopy was repeated. This process was continued until all foods had been added back, allowing specific triggers of esophageal eosinophilia to be identified.

Skin-prick testing for food allergen sensitization was conducted with a standardized panel of 58 items (8 fish, 6 shellfish, 8 nuts, cow's milk, egg white and egg yolk, 5 grains, 5 meats, 8 fruits, 5 vegetables, and 10 herbs) and for aeroallergen sensitization with a standardized panel of 46 items (11 molds, 12 trees, 4 grasses, 10 weeds, 4 animal danders, 2 mites, 2 cockroaches, and a feather mix).

Data were abstracted from the UNC electronic medical record. Using standardized data collection tools, we recorded patient demographics, symptoms, selected laboratory results (erythrocyte sedimentation rate [ESR], C-reactive protein [CRP], routine serum IgE, and peripheral eosinophil counts) endoscopy findings, and eosinophil counts from pathologist review of esophageal biopsy samples. Eosinophil counts had been previously determined for clinical purposes and were recorded as the maximum number of eosinophils per high-power field (eos/hpf; hpf size = $0.24mm^2$). Outcomes included symptom response (dichotomous patient-reported subjective improvement [yes/no] as documented in clinic notes), endoscopic improvement (abstracted from endoscopy reports), and two histologic endpoints: ≥50% decrease in eos/hpf compared to baseline, and <15 eos/hpf. For the purpose of this analysis, <15 eos/hpf was considered a response to therapy. In cases where patients underwent treatment with more than one diet strategy (for example, no response to targeted elimination, leading to a trial of SFED), the most efficacious outcome was used for analysis. Topical steroid use was also recorded, and in cases where patients were treated with multiple agents, the results from the most efficacious regimen were used in this analysis (for example, if non-response to fluticasone therapy was followed by response to swallowed budesonide, we recorded the data associated with the budesonide course).

For analysis, descriptive statistics were calculated and distributions of results were examined. Because all continuous outcome variables in this study were not normally distributed, Wilcoxon rank-sum and signed-rank tests were used to compare medians. For categorical variables, Fisher's exact test was used. To evaluate factors which might predict effective response to dietary therapy, patients who achieved <15 eos/hpf on dietary therapy were compared to those with ≥15 eos/hpf with regard to demographic, endoscopic, and laboratory characteristics as well as presenting symptoms, atopic disease status, and response to steroid therapy. Finally, a sub-analysis was performed examining response rates in the set of patients who had been treated with topical steroids and diet therapy at different times as individual therapies. All data analyses were performed using SAS v9.3. This study was approved by the UNC Institutional Review Board.

Results

Thirty-one patients who underwent dietary therapy and met inclusion criteria were identified (Table 1). The average age was 36 ± 9 years, the gender distribution was balanced (52% female), and they were predominantly white (90%). The dominant presenting symptom was dysphagia (90%). The rate of any atopic disease was 84%. Thirty (97%) patients underwent food allergen sensitivity testing and 25 (81%) underwent testing for environmental allergen sensitivities. Of those, 80% had at least one food sensitivity (mean 7 ± 6 positive skin prick tests) identified from a panel of 58 potential allergens. On environmental sensitivity testing, 96% had at least one sensitivity (mean 16 ± 12 positive skin prick tests) identified from a panel of 46 potential allergens. Food sensitivities by skin prick testing were more frequent in the targeted diet group than in the SFED group (95% vs 38%, p = 0.002), likely reflecting a patient and provider preference to perform targeted elimination when there was a demonstrated allergen sensitization. While peripheral eosinophil levels for all subjects were slightly higher than the upper limit of normal (ULN) (0.42±0.44 compared to ULN of 0.4), patients in the SFED group had significantly lower peripheral eosinophil counts than the targeted diet group. There were no statistical differences between IgE levels for the targeted and SFED groups.

On baseline endoscopy, rings and furrows were the predominant endoscopic features, seen respectively on 68 and 74% of EGDs, and 29% of patients required dilation. Seven (23%) patients underwent dietary therapy because they did not have a response to topical steroids, 16 (52%) underwent dietary therapy after responding to steroid therapy either because they had an interest in identifying their allergen triggers or because they wished to avoid protracted steroid therapy, and 8 (26%) underwent dietary therapy as first line treatment.

Dietary therapy significantly reduced, but did not frequently normalize, esophageal eosinophil counts. For all patients, the eosinophil count decreased from a baseline of 78 eos/hpf to 43 eos/hpf after dietary therapy (p = 0.004) (Table 2). Esophageal eosinophil counts fell below the threshold for disease diagnosis (15 eos/hpf) for 11 subjects (39%) with a greater rate of response in the SFED group than in the targeted group, but this was not statistically significant (56% vs 32%, p = 0.41). Only 5 patients achieved normalization of esophageal biopsies defined as an eosinophil count < 5 eos/hpf. There were no differences in eosinophil count response between the targeted and SFED subgroups. Symptoms improved

in 22 patients (71%) who underwent any dietary therapy, and endoscopy findings improved in 15 (54%).

To assess characteristics of dietary responders, those with <15 eos/hpf on dietary therapy were compared to those with ≥15 eos/hpf (Table 3). There were no differences in demographic characteristics, presenting symptoms, or endoscopic findings. They also did not differ significantly in their rate of use of SFED versus the targeted diet or in being treated with multiple diets. There was no association between the presence of food or environmental allergen sensitization on skin-prick testing results and response to dietary therapy. The number of food or environmental sensitivities and the severity of the skin prick response were also not associated with response to dietary therapy. Additionally, receiving guidance from a dietitian did not predict response to dietary therapy. Of the 31 patients in this study, nine (29%) had a dietitian consult (6 targeted, 3 SFED, p = 0.74), eight (89%) of these underwent repeat endoscopy, and two (25%) achieved a histologic response < 15 eos/hpf. In contrast, 9 of the 20 patients (45%) without dietitian consult achieved < 15 eos/hpf (p = 0.33). Baseline eosinophil counts were somewhat lower in patients who responded (53 versus 95), but this difference did not meet statistical significance. IgE levels, however, were significantly higher in those who did not respond to dietary therapy (142 vs 468 kU/L; p = 0.049). Among subjects using dietary therapy as first line treatment, 50% (3/6) responded (<15 eos/hpf). In those who pursued dietary therapy after successful steroid therapy, 44% (7/16) responded, and in those who did not respond to steroid therapy, 17% (1/6) responded to dietary therapy (p = 0.20). (Three patients declined to undergo repeat EGD on therapy and were excluded from this portion of the analysis.)

Of the 11 patients who responded to dietary elimination with < 15 eos/hpf on post-elimination esophageal biopsy, 9 (82%) underwent serial reintroduction of foods to identify their allergic trigger(s); the other 2 opted to maintain their diet restrictions without trigger identification. Three patients (33%) had multiple triggers. Overall, 4 (44%) reacted to dairy, 4 (44%) to eggs, 2 (22%) to wheat, 1 (11%) to shellfish, 1 (11%) to legumes, and 1 (11%) to nuts. This reintroduction process required a mean of 4.8 endoscopies per patient (range: 2–9) over a mean of 9.6 months (range 2–22).

Within our study cohort, we identified 26 patients who received topical corticosteroid treatment at a different time point in their therapeutic course from their dietary therapy. On steroid therapy alone, 69% had symptom improvement, 56% had endoscopic improvement, and 56% had <15 eos/hpf. These response rates did not differ statistically from the response to dietary therapy. Of those who were treated at separate times with steroid and dietary therapy, 7 (30%) responded to both treatments, 8 (35%) did not respond to either modality, 6 (26%) responded to steroid therapy only, and 2 (9%) responded to dietary therapy only (3 patients did not undergo post-therapy endoscopy, and were not included in this portion of the analysis).

Discussion

EoE is an increasingly recognized cause of dysphagia and food impaction in adults.[2, 3] Because EoE is an allergen/immune mediated condition, dietary elimination therapy has

been extensively studied in children and is now a well-established modality in pediatric patients with EoE.[9, 11, 26, 27] Until recently, the utility of dietary elimination was unknown in adults. However, emerging data suggest that dietary therapy is also effective in this population.[18, 22] Our study aimed to determine the clinical and histologic response rates to dietary therapy in adults with EoE at our center and identify predictors of response to dietary elimination. The results demonstrate that dietary therapy provided symptom relief to the majority of patients and resulted in substantial, but not complete, decreases in esophageal eosinophilia, thus supporting dietary elimination therapy as an effective modality in adults with EoE. Two areas of our results bear particular emphasis. First, dietary therapy was successful as first line treatment, and could be offered to all patients diagnosed with EoE as indicated in the most recent consensus guidelines.[2, 3] Second, because this study included patients who failed steroid therapy, the overall study population likely represents harder to treat EoE patients. The success of dietary elimination in this population indicates its utility for therapy in steroid-refractory patients.

The first study of dietary therapy in EoE used elemental formula in a series of pediatric patients, showed a universal response, and provided evidence for the allergic basis of EoE.[28] While this dietary approach was confirmed to be effective,[11, 26, 27, 29] because of its restrictive nature, other strategies for dietary therapy have been subsequently developed. These strategies were first studied in children and include targeted elimination based on results of allergy testing,[9, 30] and the empiric six-food elimination diet.[10, 11, 27] Data to support dietary approaches in adults were initially sparse and discouraging,[17] but more recently several studies have shown that dietary elimination can be effective outside of the pediatric population (Table 4). Gonsalves and colleagues conducted a prospective study of the SFED and found a 94% symptomatic response rate as well as a 73% histologic response (defined by < 15 eos/hpf).[18] Lucendo and colleagues performed a similar study, but subjects undergoing SFED eliminated all cereals (corn and rice in addition to wheat) and legumes in addition to peanuts. They reported essentially identical rates of histologic response (74% with < 15 eos/hpf).[22] In the only other study of targeted elimination in adults, Molina-Infante and colleagues reported a 33% rate for both symptoms and histologic (<15 eos/hpf) response.[20]

Our response rates (71% symptomatic response; 39% histologic response < 15 eos/hpf) are somewhat lower than other studies. There are several possible causes for this. First, in contrast to the other studies, ours included patients who pursued dietary therapy after failing steroid therapy. This population may have harder-to-treat disease that is less likely to respond to any treatment modality. Second, less than one-third of our patients saw a dietitian as part of their course of care. Surprisingly, we found no statistical association between dietitian referral and dietary response, but we would still suspect that patients undergoing elimination diet therapy would benefit from the participation of a dietitian not only to maximize compliance but to ensure adequate nutrition is maintained. Our results, however, may be more reflective of the response rate in a setting where expert dietitians would not be routinely available. Finally, the sample sizes of studies of dietary therapy including our own are small enough that chance alone could account for the difference between our results and those of previous researchers. Interestingly, we did not note a difference between those treated with SFED and those treated with targeted elimination.

Previous studies exploring reintroduction of foods to identify dietary triggers of EoE have had varying results. In the Lucendo study,[22] multiple food triggers were identified in 64% of their patients, and the most common triggers were milk (62%), followed by wheat (29%), eggs (26%), and legumes (24%). In the Gonsalves study,[18] 3 patients (15%) had multiple allergies, and wheat (60%) and milk (50%) were the most common causative agents, followed by soy (10%). While our results share the finding that dairy is a predominant trigger, our high rate of egg as a trigger had not been observed previously, though this may be an artifact of our small sample size. Further research could examine whether there is geographic variation in EoE allergic triggers.

When we analyzed potential predictors of dietary response, we found few differences in the baseline characteristics of dietary responders and non-responders. However, the IgE level was significantly elevated in non-responders to dietary therapy, and the esophageal eosinophil count was also higher (though this was not statistically significant). This suggests that those who responded to dietary therapy may have had less severe disease than those who did not. It might also suggest that they had many allergens, both food and environmental, playing a role in their disease. Predictors of response to dietary therapy have not been well studied in adults. In the Gonsalves study, predictors of response included symptoms of heartburn and entering the study during its later portion,[18] but similar to this study, there were few clinical predictors. Identification of the determinants of successful dietary therapy would be important so that the patients most likely to respond to this modality can be targeted.

There are potential limitations of this study. Because it was retrospective, data extraction was from chart review and outcomes were not assessed prospectively. Two of our outcomes, symptomatic improvement and endoscopic improvement, could not be assessed with standardized instruments as these were not available during the study period, so symptom and endoscopic findings should be interpreted cautiously. Second, patients were not randomized to dietary therapy or steroid therapy, or to SFED versus targeted elimination. Because factors such as patient preference, prior treatment response, and allergen skin-prick testing results impacted their selection of dietary treatment, there is the possibility that the results may not be representative of the broader population of EoE patients. However, this self-selection has the benefit of reflecting actual clinic practices, where therapeutic decisions are shared between clinicians and patients, with patient interest and motivation playing a major role. For example, if no food sensitizations identified on skin testing, then SFED was typically chosen, whereas if allergy testing identified food sensitivities, patients and providers were likely more inclined to undertake targeted dietary elimination. In addition, the study period overlapped with the publication of data on dietary elimination therapy in adults where it appeared that skin prick testing did not have good utility for identifying food triggers,[18] so practice patterns may have changed over the study period. Finally, this study was conducted at a tertiary care referral center, so results may not be generalizable.

There are also several strengths to the study. This is a relatively large group of adult EoE patients treated with two modalities of dietary therapy, and predictors of response were assessed. Though data were collected retrospectively, the patients were treated by a clinical team with consistent treatment algorithms. Because these results were generated during

routine care of these patients, the results should be reflective of those that would be expected from patients who undergo dietary therapy in a routine clinical setting. We included patients who underwent dietary therapy because of personal preference as a first line therapy or to find allergen triggers after a steroid response, as well as subjects who needed to undergo dietary therapy because of steroid non-response. This latter group might typically be considered a harder to treat subpopulation, and may be one reason that our steroid response and SFED rates are somewhat lower than what has been previously reported. This study is also unique in demonstrating similar rates of symptomatic and pathologic response between targeted diets and SFED.

In conclusion, this retrospective study of adults with EoE undergoing dietary therapy found that both targeted elimination and the SFED were successful treatment modalities for improving symptoms and decreasing esophageal eosinophilia. The rates of response were generally similar to steroid response rates in this self-selected group which included a number of steroid non-responders. In subjects who responded and had food groups added back, dairy and eggs were the most common triggers. While most clinical factors did not differentiate dietary responders from non-responders, there was a suggestion those with more severe disease (higher IgE levels, higher eosinophil counts, lack of prior steroid response) might be harder to treat. Further research should emphasize which factors can predict effective dietary therapy, in order to target therapy to patients most likely to respond.

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Abbreviations

CRP	C-reactive protein
EGD	esophagogastroduodenoscopy
EoE	eosinophilic esophagitis
eos/hpf	eosinophils per high-powered field
ESR	erythrocyte sedimentation rate
PPI	proton-pump inhibitor
SFED	six food elimination diet
ULN	upper limit of normal

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Table 1

Demographic Data and Baseline Characteristics by treatment modality

	All Dietary Therapy (n = 31)	Targeted (n = 22)	SFED (n = 9)
Age (mean years ±SD)	36±9	38±9	33±8
Female (n, %)	16 (52)	12 (55)	4 (44)
White race (n, %)	28 (90)	19 (86)	9 (100)
Atopic Disease (any) (n, %)	26 (84)	19 (86)	7 (78)
Skin Prick Testing			
Positive for Food (n, %)[*, †]	24 (80)	21 (95)	3 (38)
Positive for Environmental (n, %)[†]	24 (96)	18 (95)	6 (100)
Laboratory Values			
Elevated ESR or CRP (n, %)	2 (6)	2 (9)	0 (0)
IgE (mean kU/L ±SD)	342 ±340	389 ±377	188 ±100
Peripheral Eosinophils (cells × 10^9/L ±SD)[‡]	0.42 ±0.44	0.46 ±0.12	0.13 ±0.13
Presenting Symptoms (n, %)			
Dysphagia	28 (90)	20 (91)	8 (89)
Food Impaction	11 (35)	9 (41)	2 (22)
Heartburn	15 (48)	11 (50)	4 (44)
Chest Pain	12 (39)	9 (41)	3 (33)
Abdominal Pain	7 (23)	5 (23)	2 (22)
Nausea	3 (10)	2 (9)	1 (11)
Vomiting	3 (10)	3 (14)	0 (0)
Baseline Endoscopic Findings (n, %)			
Normal	2 (6)	1 (5)	1 (11)
Rings	21 (68)	16 (73)	5 (56)
Stricture	7 (23)	3 (14)	4 (44)
Narrowing	14 (45)	9 (41)	5 (55)
Furrows	23 (74)	16 (73)	7 (78)
Crêpe Paper	1 (3)	0 (0)	1 (11)
White Plaques	14 (45)	10 (45)	4 (44)
Decreased Vascularity	10 (32)	7 (32)	3 (33)
Erosive esophagitis	2 (6)	2 (9)	0 (0)
Hiatal Hernia	2 (6)	1 (5)	1 (11)
Dilation Performed	9 (29)	6 (27)	3 (33)

[*] Fisher's exact p = 0.002 for the comparison between the targeted and SFED groups

[†] Proportions are based on the 30 patients who underwent food allergen sensitivity testing (22 in the targeted group and 8 in the SFED group) and 25 who underwent testing for environmental allergen sensitivities (19 in the targeted group and 6 in the SFED group)

[‡] Wilcoxon exact p = 0.06 for the comparison between the targeted and SFED groups

Table 2

Treatment Response by Dietary Subgroup

	All diets (n = 31)	Targeted (n = 22)	SFED (n = 9)
Baseline maximum eosinophil count (mean eos/hpf ±SD)	78±71	77±59	81±101
Post-diet therapy max eosinophil count (mean eos/hpf ± SD)*	43±49†	39±41†	50±64†
≥50% Reduction in Eos n (%)‡	15 (58)	9 (50)	6 (75)
<15 eos/hpf n (%)	11 (39)	6 (32)	5 (56)
Symptomatic Improvement (n, %)	22 (71)	15 (68)	7 (78)
Endoscopic Improvement (n, %)	15 (54)	10 (53)	5 (56)

*Three patients who did not undergo repeat upper endoscopy are not included

†Sign rank test comparing eosinophil counts on dietary therapy to baseline eosinophil counts; p = 0.004 for all diets, p = 0.03 for targeted, and p = 0.15 for SFED.

‡Two patients underwent baseline endoscopic assessment at other institutions and their eosinophil counts are not included.

Table 3

Characteristics of Dietary Responders and Non-Responders*

	<15 eos/hpf (n = 11)	≥15 eos/hpf (n = 17)	p-value
Age (mean years ± SD)	38±7	34±10	0.35
Female (n, %)	7 (64)	7 (41)	0.44
White (n, %)	10 (91)	16 (100)	0.41
Atopic Disease (n, %)	10 (91)	13 (76)	0.62
Skin Prick Testing			
Positive for Food (n, %)	7 (70)	14 (82)	0.64
Positive for Environmental (n, %)	9 (100)	13 (93)	1.00
Laboratory Values			
Elevated ESR or CRP (n, %)	0 (0)	2 (12)	0.68
Peripheral Eos (mean cells × 10^9/L ± SD)	0.46±0.59	0.40±0.37	0.64
IgE (mean kU/L ± SD)	142±94	468±383	0.03
Presenting Symptoms (n, %)			
Dysphagia	9 (82)	16 (94)	0.54
Food Impaction	5 (45)	6 (35)	0.70
Heartburn	5 (45)	9 (53)	1.00
Chest pain	4 (36)	7 (41)	1.00
Abdominal Pain	2 (18)	4 (24)	1.00
Vomiting	0 (0)	2 (12)	0.51
Nausea	1 (9)	2 (12)	1.00
Endoscopic findings (n, %)			
Normal	1 (9)	0 (0)	0.39
Rings	9 (82)	10 (59)	0.25
Stricture	1 (9)	6 (35)	0.19
Narrowing	4 (36)	10 (59)	0.44
Furrows	9 (82)	12 (71)	0.67
Crêpe Paper	0 (0)	1 (6)	1.00
White Plaques	5 (45)	8 (47)	1.00
Decreased Vascularity	3 (27)	7 (41)	0.69
Erosive esophagitis	0 (0)	2 (12)	0.51
Hiatal Hernia	0 (0)	2 (12)	0.51
Dilation Performed	4 (36)	5 (29)	1.00
Baseline Max Eosinophil Count (mean eos/hpf ± SD)	53±23	95±94	0.20
Dietary Therapy Characteristics (n, %)			
Targeted Diet	6 (55)	13 (76)	0.41
Tried multiple diets	3 (30)	3 (19)	1.00
Indication for Dietary Therapy			0.58
Dietary therapy and no prior steroid therapy	3 (27)	3 (18)	
Dietary therapy after steroid response‡	7 (64)	9 (53)	

	<15 eos/hpf (n = 11)	≥15 eos/hpf (n = 17)	p-value
Dietary therapy after steroid failure‡	1 (9)	5 (29)	

*3 patients who did not undergo repeat upper endoscopy to assess response are not included

‡Response defined as < 15 eos/hpf on swallowed steroid; failure defined as ≥15 eos/hpf on swallowed steroids

Table 4

Selected Results from Studies of Dietary Elimination Therapy in Adults with EoE

Lead Author, Year	Diet Type(s)	N	Symptom Response (% improved)	Endoscopic Response (% improved)	Histologic Response
Lucendo, 2013[22]	SFED	67	---	---	73% with <15 eos/hpf
Gonsalves, 2012[18]	SFED	50	94	---	74% with <15 eos/hpf; 78% with >50% reduction in eos
Gonzales-Cervera, 2012[21]	SFED, Targeted	3	100	100	100% improved
Molina-Infante, 2012[20]	Targeted	22	33	---	33% with <15 eos/hpf
Simon, 2006[17]	Targeted	6	17	0	No change*
Gonsalves, 2013[19]	4 food elimination diet	13†	81	70	46% with <10 eos/hpf; 85% with ≥50% reduction in eos
Peterson, 2013[23]	Elemental	18	0	94	72% with <10 eos/hpf

*EGD performed in the 1 patient with symptom improvement, results reported as “almost identical” to previous study.

†Study of adults and children, only adult data included here.