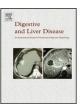
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#### **Alimentary Tract**

# Clinical characteristics, treatment outcomes, and resource utilization in children and adults with eosinophilic gastroenteritis



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#### ABSTRACT

*Background*: Eosinophilic gastroenteritis is a rare condition where eosinophilic inflammation occurs in the gastrointestinal tract in the absence of secondary causes. Little is known regarding aetiology, pathogenesis, or natural history.

Aims: To characterize the clinical, endoscopic, and histopathologic features of eosinophilic gastroenteritis and to summarize treatment outcomes.

Methods: Pathology reports of all patients who had undergone upper endoscopy with biopsy between January 1, 2000 and June 20, 2013 were reviewed. Eosinophilic gastroenteritis was diagnosed if there were  $\geq$ 20 eosinophils/hpf on either gastric of duodenal biopsy, symptoms attributable to the gastrointestinal tract, and no known secondary cause of eosinophilia. Descriptive statistics characterized patients diagnosed with eosinophilic gastroenteritis and bivariate analysis compared adults and children.

Results: There were 44 patients diagnosed with eosinophilic gastrointestinal disease. The most common symptoms were vomiting (71%) and abdominal pain (62%). Of the eosinophilic gastroenteritis cases, 12 (30%) had esophageal involvement, and 11 (28%) had colonic involvement. For treatment, 36 (80%) received corticosteroids. Overall, 27 (60%) had symptom resolution and 23 (51%) had endoscopic resolution. Cases underwent a mean of five endoscopic procedures per year.

*Conclusion:* Eosinophilic gastroenteritis presents with non-specific gastrointestinal symptoms and in almost one-third of cases has concomitant esophageal or colonic involvement. It remains difficult to treat, with high rates of endoscopic utilization.

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#### 1. Introduction

Eosinophilic gastroenteritis (EoG) is a rare condition first described in 1937 [1]. It belongs to the family of eosinophilic gastrointestinal disorders (EGID) where eosinophilic inflammation occurs in the GI tract in the absence of secondary causes. Proposed secondary causes include adrenal insufficiency, medication hypersensitivity reactions, collagen vascular disease, malignancy, hypereosinophilic syndrome, or parasitic infection [2,3].

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Though the aetiology and pathogenesis of the disease have yet to be fully elucidated, the condition is thought to be a polygenic allergic disorder on the spectrum between IgE mediated and delayed Th2 responses, but not fitting completely into either category [2,4]. Case reports suggest that EoG has no singular ethnic or age predilection. It may be more prevalent from the 3rd to 5th decades of life [5,6]. The disease has been associated with atopic conditions including food allergy, asthma, and atopic dermatitis [7]. As described by Klein and Talley, there can be eosinophilic infiltration throughout the different layers of the GI tract (mucosal, muscle layer, subserosal) [8,9]. This and the location of the eosinophilia in the GI tract impact the clinical presentation, and help to explain the diverse symptoms and signs that may be attributable to the condition. Endoscopic or full-thickness surgical biopsy demonstrating eosinophilic infiltration of the GI tract is necessary to make the diagnosis of EoG [10-12].

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Because the current literature is limited to small case series and single case reports, there are limited epidemiologic, clinical, and histopathologic data describing the disease. This makes diagnosis and treatment challenging, as there are no consensus statements to guide the evidence-based management of this condition. The aim of this study was to characterize the clinical, endoscopic, and histopathologic features of a cohort of patients with EoG, and to summarize treatment outcomes and resource utilization.

#### 2. Materials and methods

We conducted a retrospective cohort study at the University of North Carolina at Chapel Hill. Pathology reports of all patients who had undergone upper endoscopy with biopsy between January 1, 2000 and June 20, 2013 were obtained to identify patients with EoG. These reports were reviewed if the term "eosinophil" was mentioned anywhere in the report. Cases of EoG were defined by ≥20 eosinophils/hpf (hpf=0.24 mm²) on either gastric or duodenal biopsy, symptoms attributable to the GI tract (i.e. abdominal pain, nausea, vomiting, weight loss, feeding intolerance, etc.), and no known secondary cause of eosinophilia. While there are no diagnostic guidelines published for EoG, this definition is consistent with what has been used in prior reports [9,13,14]. Colonoscopy reports from the same day as the index endoscopy were obtained to identify patients who also had colonic involvement.

Once cases were identified, additional data were extracted from the electronic medical record. These included patient demographics, symptoms, co-morbidities, habits (tobacco and alcohol abuse), medications, endoscopic findings, age specific BMI, and treatments. Symptoms were from patient or caregiver self-report. Comorbid conditions or disease complications required a diagnosis by a provider. Protein-losing enteropathy was defined by an albumin level less then 2.6 without hepatic dysfunction or proteinuria; ascites required a radiographic diagnosis. For patients with followup data available in our system, we assessed treatment outcomes including symptomatic, endoscopic, and histologic response. Interval changes in patient symptoms were also from self-report. All follow-up evaluations were done at the discretion of the physician to detail treatment response or to clarify recurrent symptoms. Endoscopic and histopathologic findings after treatment were compared with pre-treatment findings, but endoscopic response was only assessed for those patients with abnormal endoscopic findings at baseline. The total number of endoscopic procedures performed on patients during the follow-up period was also recorded.

For analysis, patients were characterized with descriptive statistics. Bivariate analysis was performed to compare adults ( $\geq$ 18 years) and children using t-tests for means and chi-square for proportions. We also calculated the mean number of endoscopic procedures performed per patient during the follow-up time frame. This study was approved by the UNC Institutional Review Board.

#### 3. Results

There were 44 patients diagnosed with EGID over the study time frame. Four of these patients were diagnosed with isolated eosinophilic colitis (EoC) without involvement of the stomach or bowel. The mean age was 16 years (range 0.4–83), 58% were male, and 58% were white. The most common presenting symptoms were vomiting (71%) and abdominal pain (62%) (Table 1). While the mean BMI at diagnosis was normal (20 $\pm$ 7), the median BMI was in the underweight category (17, IQR: 16–23). Food allergies were noted in 42%, 64% had a family history of atopic disease, and both the serum total IgE levels and peripheral eosinophil counts were elevated (Table 2).

**Table 1**Characteristics of subjects with eosinophilic gastrointestinal disorders.

Number of patients studied	44
Age at diagnosis, Mean $\pm$ SD; range	$16.0 \pm 19.1; 0.42 - 82.8$
Length of symptoms before biopsy,	$4.9 \pm 9.4$ ; $0.08 - 55.7$
Mean $\pm$ SD; range	
Adult, $\geq$ 18; $N(\%)$	11 (24)
Male, <i>N</i> (%)	26 (58)
Race, N (%)	
White	31 (58)
Black	7 (16)
Asian	0 (0)
Hispanic	2 (4)
Unknown	5 (11)
Symptoms, N (%)	
Dysphagia	12 (27)
Heartburn	8 (18)
Abdominal pain	28 (62)
Nausea	17 (38)
Vomiting	32 (71)
Chest pain	3 (7)
Bloating	8 (18)
Diarrhoea	14 (31)
Constipation	15 (33)
Comorbid conditions, N (%)	
Food allergy	19 (42)
Asthma	12 (27)
Allergic rhinitis	17 (38)
Drug allergy	14 (31)
Eczema	1 (16)
Complications, N (%)	
Family history of atopic disease	29 (64)
Anaemia at diagnosis	4(9)
Failure to thrive	14 (31)
Ascites	1 (2)
Small bowel obstruction	1 (2)
Food impaction	5 (11)
Weight loss >4 pounds	12 (27)
Protein losing enteropathy	3 (7)
Steatorrhea	1 (2)

Eosinophilic infiltration of the gastrointestinal tract varied for this population. Of the EoG cases, 12 (30%) had both gastric and duodenal involvement, 18 (45%) had gastric involvement only, and 10 had duodenal involvement only. In addition, we found that 12

**Table 2**Laboratory and histologic findings of eosinophilic gastrointestinal disorder patients.

Lab results	$Mean \pm SD$	Median	IQR
Serum IgE level	$418\pm722$	188	24-467
Absolute eosinophil count	$1.53\pm2.64$	0.55	0.2 - 1.8
ESR	$9.9 \pm 11.3$		
CRP	$2.9\pm4.0$	0.5	15.6-23.1
BMI	$20.1\pm6.9$	16.7	15.6-23.1
Cases of EGID		N = 44	
EoG predominant, N (%)	40 (91)		
Gastric & Duodenal eosinophilia		12 (30)	
Gastric eosinophilia		18 (45)	
Duodenal eosinophilia		10 (25)	
EoE also present	12 (30)		
EoC also present	11 (28)		
EoC predominant, N (%)			4(9)
Eosinophil count (#/HPF)	Mean ± SD	Median	IQR
Gastric	61 ± 70	37	25-60
Duodenal	$55\pm23$	50	40-75
Esophageal	$56\pm62$	42	9-100
Colonic eosinophil count	$84\pm73$	68	50-80
Gastric Duodenal Esophageal	61 ± 70 55 ± 23 56 ± 62	37 50 42	25-60 40-75 9-100

EoG, eosinophilic gastroenteritis; EoE, eosinophilic esophagitis; EoC, eosinophilic colitis.

Serum IgE level (IU/L); Absolute eosinophil count ( $n*10^9$ /l); ESR (mm/h); CRP (mg/dl); BMI (kg/m<sup>2</sup>).

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