



Letter

Racial disparity in eosinophilic esophagitis from a single, defined population

Eosinophilic esophagitis (EoE) is an immune-mediated disease of the esophagus characterized by esophageal eosinophilia and symptoms of esophageal dysmotility.^{1,2} Studies have attempted to define the epidemiology of the disease, with most reports suggesting EoE has a predominance in white individuals.^{2–4} However, a few recent studies have shed light on this disease in the African American population.^{5,6} These reports have suggested that the disease may have different clinical presentations between racially distinct populations and that the incidence in the African American population may be higher than reported from prior studies. It is difficult to know whether there is truly a racial disparity in the prevalence of the disease.

LeBonheur Children's Hospital (Memphis, Tennessee) has the benefit of serving a defined population that is approximately 50% white and 50% African American. In addition, all local pediatric gastroenterologists' endoscopies are performed through a single system; therefore, this institution has the benefit of being able to catch every endoscopic procedure performed in pediatric cases within a defined catchment area. With this setting in place, we sought to characterize and define any racial disparities in EoE in this population.

A retrospective chart review was performed of all patients 0 to 18 years of age given a diagnosis of EoE (*International Classification of Diseases, Ninth Revision* [ICD-9] code 530.13) seen in all pediatric gastrointestinal clinics in the catchment area from June 2012 through June 2013. These dates were selected based on when the electronic medical record began using this specific ICD-9 code, which was not available before 2011. All charts were reviewed by a single reviewer. Patients were included if they had esophageal eosinophilia (>15 eosinophils per high power field) on at least 1 esophageal biopsy despite acid suppression therapy or a normal esophageal pH probe study. The following data were obtained through chart review: age, sex, race and ethnicity, presenting symptom, associated symptoms, food allergies, other atopic diseases, peripheral eosinophilia, and endoscopic and histopathologic findings. Local population data were obtained from the 2012 US Census.⁷ The study was approved The University of Tennessee institutional review board.

We identified 38 cases of EoE seen in our clinics in the specified period. In this cohort, there were 22 (58%) white patients, 13 (34%) African American patients, and 3 (8%) patients of other races. The catchment area of our hospital is approximately 1.3 million people (49.8% white, 46.3% African American, and 3.9% other). Given this known makeup of our catchment area, we found a nonsignificant, slightly larger proportion of white patients with EoE than would be

expected ($P = .05$). Most patients were male (74.3%), which has been previously reported in most studies.^{2–6} We examined clinical differences between white and African American patients (Table 1). When comparing the white with African American patients, we found African American patients to be younger at time of diagnosis (5 vs 12 years old, $P < .005$), to have a slightly higher peak eosinophil count in their esophageal biopsies at time of diagnosis (50 vs 40.5, $P = .04$), and to be less likely to have food impaction (0% vs 32%, $P = .03$).

With these data, we hope to shed more light on the topic of racial disparity in EoE. Prior large retrospective studies of EoE in pediatric populations in the United States have clearly shown the cohorts to be overwhelmingly white, with percentages ranging from 84% to 94%.^{2–4} There is yet to be any clear explanation for this predominance. It may be related to selection bias and referral bias from the location of the reports. However, at least 1 study has attempted to control for this using comparative samples from their clinics and defining the geographic distribution of their patients to better define their expected population.³ Nevertheless, the investigators still found a stark predominance of white patients affected (84%). In a more recent study, Weiler et al⁵ reported no racial disparity EoE in their cohort. In the present cohort, we did find predominance in white patients; however, it was not nearly as vast a difference as in most prior reports.

Table 1

Sex and clinical characteristics of cohort by race^a

	All patients	White	African American	P value
Patients, n (%)	38 (100)	22 (57.9)	13 (34.2)	.051 ^b
Male patients, %	74.3	72.7	76.9	1.0
Age at diagnosis (y)	12	12	5	.003 ^c
Duration of symptoms (mo)	30	36	24	.26 ^c
Peak eosinophil count per HPF, n	50	40.5	50	.04 ^c
Peripheral eosinophil count, n	305	232	462	.15 ^c
Symptoms, n (%)				
Impaction	7 (18.4)	7 (31.8)	0 (0.0)	.025
Dysphagia	10 (26.3)	5 (22.7)	5 (38.5)	.26
Abdominal pain	22 (57.9)	14 (63.6)	7 (53.8)	.41
Vomiting	24 (63.2)	11 (50.0)	10 (76.9)	.11
Weight loss	5 (13.2)	2 (9.1)	3 (23.1)	.26
Failure to thrive	2 (5.3)	0 (0.0)	2 (15.4)	.13
Endoscopic findings, n (%)				
Normal	9 (23.7)	4 (18.2)	5 (38.5)	.18
Linear furrows	23 (60.5)	16 (72.7)	6 (46.2)	.11
White plaques	9 (23.7)	5 (22.7)	3 (23.1)	.65

Abbreviation: HPF, high-power field.

^aAll values are medians. Analysis was performed with the Fisher exact test between groups unless stated otherwise.

^bBy χ^2 test using expected values based on sample population.

^cBy Mann-Whitney test between groups.

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Defining whether there is a true racial disparity in EoE may be vital to understanding the pathophysiology of the disease. Given that multiple reports have shown the disease to present differently in African American versus white cohorts,^{4–6} it is distinctly possible that there are different underlying mechanisms that lead to a similar phenotype in different populations. This may be akin to different mechanisms of disease in other atopic conditions, such as that seen in atopic dermatitis, in which strong associations have been seen with mutations in filaggrin in European cohorts, but the same has not been found in African American cohorts.⁸

This study has several limitations. It is a small sample; however, given the period and the population studied, the numbers of patients is to be expected. In addition, using the specific ICD-9 code may have caused us to miss some cases that were not coded correctly. However, we believe that there is unlikely to be a racial bias in those missed patients who were coded using another ICD-9 code. There is always a concern that disparities in access to care may skew these data. Because all pediatric gastroenterologists in our catchment see patients through a single, university-based practice, we believe that the bias of insurance should not affect our outcome. However, if there is a racial disparity in uninsured patients unable to be seen by a physician, this could affect outcomes. Unfortunately, to our knowledge, that information is not available. Moreover, some patients with EoE in the catchment area may have gone to other hospitals in the nation for EoE care. However, that would likely mean that they received an initial diagnosis from a local physician and would be included if diagnosed in the specified period.

In conclusion, these data suggest that EoE may in fact have a predominance in white individuals, but that this predominance may not be quite as prominent as previously reported. In addition,

African American individuals do appear to be diagnosed at a younger age as reported previously.

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