

## Do Pinworms Cause Eosinophilic Esophagitis?

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### Abstract

Eosinophils are found in the gastrointestinal tract with the exception of the esophagus. Eosinophilic esophagitis (EE) clinically results in heartburn, abdominal pain and vomiting, and most children with EE are atopic. Diagnosis is supported by esophageal biopsy findings of >15 eosinophil's/hpf. *Enterobius vermicularis* infection is characteristically mild. However, there are case reports of eosinophilic ileocolitis, gastroenteritis and appendicitis secondary to pinworm infestation, but no case reports of EE associated with pinworm infestation have been reported.

**Keywords:** Eosinophils; Pinworm; Respiratory; Leukotrienes; Helminthic; Perianal; Abdominal; Exacerbated

### Introduction

Eosinophils are found in circulation and in tissues. Their half-life in the blood is up to 18 hrs but up to several weeks in tissue. Eosinophils are found normally in the GI tract excluding the esophagus, respiratory tract, skin and upper genitourinary tract [1]. These locations allow them direct access to invading organisms, particularly parasites. Eosinophils contain preformed granules consisting of four major cationic proteins with cytotoxic properties known as major basic protein, eosinophils peroxidase, eosinophilic cationic protein and eosinophils-derived neurotoxin. In response to parasitic infection, eosinophils become activated and release these granules in an attempt to kill the invading organism. They also generate leukotrienes especially LTC<sub>4</sub> which is further metabolized to LTD<sub>4</sub> and LTE<sub>4</sub>. These leukotrienes are potent bronchoconstrictors and increase airway mucus secretion [1,2]. In the process of killing invading organism, there is damage to the surrounding normal tissue.

Eosinophilic esophagitis (EoE) was first reported in 1977 but has become more notable recognized in the last 10 to 15 years. Eosinophils are not normally present in the esophagus; however, gastrointestinal reflux can cause eosinophilic infiltration. Reports suggest that esophageal reflux can cause up to 10 eosinophils per high power microscopic field. Increased eosinophils above 15 per high power microscopic field would usually not be associated with gastrointestinal reflux [3]. The diagnostic criteria of eosinophilic esophagitis via the 2011 consensus report is symptoms of esophageal dysfunction, >15 eosinophils per high power microscopic field, and little or no response to high-dose proton pump inhibitors (>2mg/kg/day) or normal pH monitoring [4].

Eosinophilic esophagitis (EoE) presents differently depending on age where children most often present with heartburn and abdominal pain. Treatment has focused on elimination of possible offending agents seen with a food elimination diet. Patients can be placed on an elemental formula, blindly eliminate the reported six most likely foods

to cause eosinophilic esophagitis (milk, soy, wheat, egg, peanut, and seafood), or eliminate foods based on results of skin testing [5,6]. To control the inflammation, patients are treated with swallowed inhaled steroids such as fluticasone or budesonide. Patients are also maintained on high dose proton pump inhibitors [4,5].

*Enterobius vermicularis* is the most common helminthic infection in the United States. They have a simple life cycle that only involves a human host. Eggs are ingested orally passing through the gastrointestinal tract to the colon where they develop into larvae and eventually adult worms. After mating in the colon, the female migrates out of the rectum and deposits eggs, usually at night, on the perianal region. The deposited eggs lead to irritation and itching which are the main complaints of persons infected with the parasite. With high parasite burden, abdominal pain, nausea and vomiting can be seen. Autoinoculation occurs with fecal oral transfer of eggs. Transmission to others occurs by the same route when exposed to eggs on contaminated linens, surfaces or food. The mainstay of treatment is with either mebendazole or albendazole. A single dose of either drug is given but should be repeated in 1-2 weeks to increase cure rates [7].

### Case Report

12 year old male presents with intermittent abdominal pain for 2 years. The pain lasted for 10-15 minutes then resolved spontaneously. Pain was worse after eating, and he does complain of occasional food sticking in his throat while eating that is relieved with drinking water. The pain usually recurs every few weeks but can be present several times a day. He occasionally has loose stools that he relates to lactose intolerance. No history of constipation. Stools are without blood or mucus. He denies nausea or vomiting associated with the abdominal pain. No history of weight loss or fever. Recently he was involved in a sledding accident that has exacerbated his abdominal pain. Ibuprofen and Acetaminophen do help relieve the pain.

A CT scan of the abdomen revealed possible thickening of the colon and chronic changes in the appendix. Significant findings of blood work were a normal white blood cell count with elevated eosinophils at 21% (normal 0-6%). CRP, ESR, TSH, free T<sub>4</sub>, and CMP were normal.

Celiac testing was normal. Work-up for infection with *H. pylori* and stool cultures for parasitic infections were negative. An esophagogastroduodenoscopy and colonic endoscopy with biopsies revealed 20 eosinophils/hpf in the esophagus, chronic gastritis with eosinophils and chronic duodenal inflammation with eosinophils. Pinworms were visualized during colonoscopy. Skin testing was positive for milk, eggs, peanut and beef. Skin testing for environmental allergens were positive to grass, mold and dog. Patient was treated with 2 courses of mebendazole, Prevacid 15 mg BID, budesonide 500 mcg swallowed BID, and food elimination diet for milk, egg, peanut and beef. Abdominal pain resolved within 2 weeks. Repeat biopsy 3 months later on therapy was negative for eosinophils in all biopsy specimens, and the lower endoscopy was negative visually for pinworms. Complete blood count was normal with 2% eosinophils. All food had been re-ingested, and the swallowed corticosteroid was discontinued. The patient did not return for subsequent visits.

## Discussion

Pinworm infection leading to eosinophilic disorders of the gastrointestinal tract have been reported in the ileum, appendix and stomach but never in the esophagus [8-10]. A case report of *Gnathostoma spinigerum* infection in a 63 year-old man revealed positive eosinophils on esophageal biopsy [11]. Although this parasite has a much more complicated life cycle including intermediate hosts, infection in humans is still via the oral route with ultimate infection of the stomach and lower gastrointestinal tract [12,13]. This report illustrates that parasitic infection passing through the esophagus may elicit an eosinophilic response.

Another interesting association, although not common, is the exaggeration or development of EoE as related to pollen exposure [14]. Our subject was allergic to pollen, but did not demonstrate oral allergy syndrome symptoms. The entire spectrum of evidence based care for EoE in adults and children has been recently updated [15].

Elimination of pinworms and proper treatment for EoE in our patient resulted in both clinical and pathological improvement. It is logical to suggest that pinworm infestation could have triggered an eosinophilic response in the esophagus and upper gastrointestinal tract leading to this patient's symptoms and pathology. Once the offending agent was removed, the disease resolved. However, the patient was started on both pinworm treatment and conventional treatment for eosinophilic esophagitis, so it is uncertain if the pinworms contributed to the eosinophilic esophagitis or was merely a coincidence.

## References

1. Rich R, Fleisher T, Shearer W, Kotzin B, Schroeder H, et al. (2001) Eosinophils. Clinical Immunology Principles and Practice. (Second Edition) Mosby International Limited pp: 24.1-24.9.
2. Yan BM and Shaffer EA (2009) Primary eosinophilic disorders of the gastrointestinal tract. Gut 58: 721-732.
3. Parfitt J, Gregor J, Suskin N, Jawa H, Driman D, et al. (2006) Eosinophilic esophagitis in adults: distinguishing features from gastroesophageal reflux disease: a study of 41 patients. Mod Pathol 19: 90-96.
4. Liacouras CA, Furuta GT, Hirano I, Atkins D, Attwood SE, et al. (2011) Eosinophilic esophagitis: updated consensus recommendations for children and adults. J Allergy Clin Immunol 128: 3-20.
5. Furuta GT, Liacouras CA, Collins MH, Gupta SK, Justinich C, et al. (2007) Eosinophilic esophagitis in children and adults: A systematic review and consensus recommendations for diagnosis and treatment. Gastroenterology 133: 1342-1363.
6. Hong S and Vogel N (2010) Food allergy and eosinophilic esophagitis: Learning what to avoid. Cleve Clin J Med 77: 51-59.
7. Leder K and Weller P (2012) Enterobiasis and trichuriasis. UpToDate 2009: 1-7.
8. Cacopardo B, Onorante A, Nigro L, Patamia I, Tosto S, et al. (1997) Eosinophilic ileocolitis by *Enterobius vermicularis*: a description of two rare cases. Ital J Gastroenterol Hepatol 29: 51-53.
9. Arca M, Gates R, Groner J, Hammond S, Caniano D, et al. (2004) Clinical manifestations of appendiceal pinworms in children: an institutional experience and review of the literature. Pediatr Surg Int 20: 372-375.
10. Macedo T and MacCarty R (2000) Eosinophilic ileocolitis secondary to *Enterobius vermicularis*: case report. Abdom Imaging 25: 530-532.
11. Tsibouris P, Galeas T, Moussia M, Sotiropoulou M, Michopoulos S, et al. (2005) Two cases of eosinophilic gastroenteritis and malabsorption due to *Enterobius vermicularis*. Dig Dis Sci 50: 2389-2392.
12. Muller-Stöver I, Richter J, Häussinger D (2004) Infection with *Gnathostoma spinigerum* as a cause of eosinophilic oesophagitis. Dtsch Med Wochenschr 129: 1973-1975.
13. Rojekkittikhun W (2002) On the biology of *Gnathostoma spinigerum*. J Trop Med Parasitol 25: 91-98.
14. Mahdavinia M, Bishehsari F, Hayat W, Elhassan A, Tobin MC, et al. (2017) Association of eosinophilic esophagitis and food pollen allergy syndrome. Ann Allergy 118: 116-117.
15. Lucendo AJ et al. Guidelines on eosinophilic esophagitis: evidence-based statements and recommendations for diagnosis and management in children and adults. UEG Journal 0: 1-24.