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Treatment of eosinophilic gastroenteritis with montelukast

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To the Editor:

We are reporting what we believe is the first case of a patient successfully treated with a leukotriene modifier for symptoms related to eosinophilic gastroenteritis. One of us (C. F.) initially saw a 13-year-old girl for a 3-year history of progressive nonbilious vomiting. Medical history was remarkable for allergic rhinitis treated with medications and allergen immunotherapy but no history of food allergies. Review of previous allergy skin testing showed absence of reactivity to foods. Her previous allergist had tried an elimination diet with no symptomatic improvement. A complete blood cell count showed 574 eosinophils/mL (8% of total neutrophils). The diagnosis was made by demonstration of marked eosinophilia in biopsy specimens of the esophagus, gastric antrum, and duodenum and elimination of other potential etiologies. Stool cultures and a barium esophagram with small bowel follow through were unremarkable. Symptoms continued despite treatment with oral cromolyn, ranitidine, and hydroxyzine. Montelukast 10 mg given orally daily was started, with complete symptom resolution. Follow-up showed one vomiting episode in 4 months of treatment with montelukast. There was a decrease in peripheral eosinophilia (574 vs 342 eosinophils/mL) compared with baseline 2 months after medications were started. Cromolyn, ranitidine, and hydroxyzine were discontinued with no recurrence of symptoms.

Tissue injury in eosinophilic gastroenteritis has been shown to correlate with the number of

activated degranulated eosinophils present.¹ Cytokines including GM-CSF, IL-3, and IL-5 promote eosinophil proliferation and differentiation in the bone marrow as well as being eosinophil chemotaxins to sites of inflammation.² It has been suggested that the release of these cytokines with autocrine or paracrine activities by eosinophils in the lamina propria of gastrointestinal tissue may lead to the persistent eosinophilic infiltration in eosinophilic gastroenteritis.³ Eosinophil recruitment may also be enhanced by chemotactic factors such as platelet-activating factor, C-C chemokines, and cysteinyl leukotrienes.² Cysteinyl leukotrienes are generated from arachidonic acid through LTC₄ synthase by mast cells, eosinophils, alveolar macrophages, and basophils, leading to end-organ inflammation.² LTD₄ has been shown to be a potent and specific chemotactic factor for human eosinophils in vitro, which has been completely inhibited by a selective leukotriene antagonist.⁴ Many factors may play a role in directing eosinophils to a site of inflammation. Because LTD₄ is both produced by and is a chemotactic factor for eosinophils, this may provide the rationale for treating a patient with eosinophilic gastroenteritis with a leukotriene receptor antagonist. Further research, including double-blind placebo-controlled clinical studies, is needed to clarify the potential role leukotrienes play in the inflammation seen in eosinophilic gastroenteritis and to assess the potential role for leukotriene modifiers in the treatment of this disease.

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Gastroenterology

March 2001 • Volume 120 • Number 4

Hugh A. Sampson, M.D., Scott H. Sicherer, M.D., Audrey H. Birnbaum, M.D.

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