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Eosinophilic Gastroenteritis

Eosinophilic gastroenteritis (EGE) is a disease characterized by patchy eosinophilic infiltration of the gastrointestinal tract without an identifiable secondary cause. It has a prevalence of approximately 8.4/100,000 in the United States [1].

Presentation

The symptoms of EGE are nonspecific, but 50-75% of patients will have existing allergic disease like asthma, food allergy, or rhinitis [2]. The symptoms of EGE depend on the location and depth of eosinophil infiltration, and include nausea, vomiting, abdominal pain, acute pancreatitis, dyspepsia, malabsorption, ascites, and weight loss when the mucosal layer is infiltrated. Additionally, recurrent acute pancreatitis is a rare, but known, manifestation of the disease [3].

If infiltration extends through to the muscular layer of the GI tract, gut motility may be impaired. This can result in symptoms of intestinal obstruction, strictures, or perforation.

Subserosal infiltration can cause ascites, in addition to the symptoms found in mucosal and muscular layer disease.

Laboratory Values

Peripheral eosinophilia may be seen, *but it is not universally present*. In one study of 52 patients with mucosal EGE, the median peripheral eosinophil count was 0.9 kU/L with an interquartile range of 0.3 – 1.9 [4].

If malabsorption is present, patients may have an abnormal D-xylose test due to carbohydrate malabsorption. Increased fecal fat content and prolonged PT may occasionally be seen in these cases as well.

Serum IgE can also be elevated, especially in children.

Anemia may be present if iron absorption is impaired or there is occult bleeding.

Diagnosis

Diagnosis is made by biopsy showing eosinophilic infiltration of the GI tract, in the presence of appropriate GI symptoms. Additionally, there cannot be any evidence of parasitic infection or other process which explains the findings [5].

Management [2]

Elemental diet or elimination diet without milk, soy, wheat, egg, milk, peanuts/tree nuts, fish/shellfish for at least 6 weeks. However, compliance is a major issue, and even strict compliance is not guaranteed to resolve symptoms.

Glucocorticoids are a commonly recommended therapy (suggested 20-40 mg/day). Initial improvement should occur within two weeks and be followed by a taper. However, recurrence of symptoms is common, and long term low-dose therapy of 5-10 mg/day may be indicated.

Immunomodulators such as 6-mercaptopurine and azathioprine have seen limited use in trials, to good effect. IVIG has also been used in a limited number of patients with steroid-resistant eosinophilic enteritis.

Leukotriene Inhibitors (e.g. montelukast) have shown conflicting results in multiple trials.

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Patients should be tested for H. pylori, because infection has been associated with EGE in case reports, with subsequent resolution of EGE after eradication. Antimicrobial therapy with clarithromycin or erythromycin has also been shown to decrease IL-2 production by T-cells, and very limited data indicates these drugs may have a role in the management of EGE.

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