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Case Report

Eosinophilic colitis in 36 years old female: a case presentation with review of literature

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ABSTRACT

Eosinophilic Colitis (EC) is a rare form of primary eosinophilic gastrointestinal disease with prevalence in neonates and young adults. EC condition is less understood as compared to the recognized eosinophilic esophagitis. Clinically EC presents as highly variable disease and it is based according to mucosal, transmural, or serosal predominance of inflammation. EC has a vast range of differential diagnosis because eosinophilia of colon occurs in many conditions like parasitic infection, Inflammatory Bowel Disease (IBD), drug-induced allergic reactions and various connective tissue disorders and so it require to rule out these causes by thorough searching for secondary causes which can be treated with antibiotics or dietary and drug elimination before we start the EC treatment. EC responds very well to steroids that may be spared by using antihistamines, leukotriene inhibitors and biologics. Here we present a case of EC in a 36 years old female with pain in abdomen and bloody diarrhoea.

Keywords: Eosinophilic colitis, Pain in abdomen, Colitis, Eosinophilia, Gastrointestinal disease

INTRODUCTION

EC is exceptionally rare with only a few cases being reported since 1979. The absence of defined histological criteria for a specific eosinophil count in the colonic mucosa makes the diagnosis of this entity challenging. Hence, its true frequency is not clear. The clinical presentation includes abdominal pain, diarrhea (bloody or non-bloody), and/or weight loss. EC in its primary form can be associated with other atopic conditions. Colonic eosinophilia can also occur secondary to helminthic infections (e.g. pinworms, hookworms), inflammatory bowel disease, autoimmune disease (e.g. scleroderma, ChurgStrauss syndrome), celiac disease, drug reactions, and in association with the HES. Primary eosinophilic gastrointestinal disease (EGID), originally described by Kaijser in 1937,[1] is a rare spectrum of gastrointestinal disorders characterized by inflammation rich in eosinophils, without evidence of known causes for eosinophilia, such as parasitic infection, drug reaction, or malignancy.[2] The disease can affect any segment or combination of segments of the gastrointestinal tract from the esophagus to the rectum, giving rise to various clinical presentations including Eosinophilic Esophagitis (EE), eosinophilic gastritis, eosinophilic gastroenteritis, and Eosinophilic Colitis (EC). Since secondary eosinophilic inflammation may occur in numerous gastrointestinal disorders such as IgE-mediated food allergy, gastroesophageal reflux disease, and inflammatory bowel disease, the true incidence and prevalence of primary EGID remains largely unknown. A recently established world-wide-web registry found that EGID mainly affects the pediatric population, although it has been reported in patients up to 68 years of age.[3] In the past few years, EE has been increasingly recognized as a distinct condition that affects about 1% of the population,

and accounts for dysphagia and food impaction that remain nonresponsive to traditional anti-reflux management, both in pediatric and adult gastroenterology.[4] Accordingly, several excellent reviews on EE have recently been published.[4-6] In contrast, EC represents the least frequent manifestation of EGID whether or not it presents with disease in other segments of the gastrointestinal tract.[3] EC appears to have a bimodal distribution that affects neonates with a relatively high prevalence and a separate group of young adults with no gender preference.[2]

CASE REPORT

A 36-year-old woman was admitted with the complaints of abdominal pain and bloody diarrhea. She had pain since 15 days not responding to any medication. There was no fever, weight loss, or rash. She had no history of any disease, food, pollen, or drug allergy in her medical history. There was no remarkable feature in her physical examination. ESR: 41 mm/h, hematocrit: 33.9%, leukocyte: 7800/mm^3 (neutrophil: 23%, eosinophil: 60.3%, lymphocyte: 15.4%), platelet: 181,000/mm^3, immunoglobulin E (IgE): 1163 IU/mL (normal range: 20-100 IU/mL), Absolute eosinophil count: 1038 cells/cu mm was counted in her blood examination. Liver and renal functions were in normal range. Parasitological examination and bacterial culture of stool were normal but stool was positive for mucus and occult blood. There were no abnormal findings in her abdominal ultrasonography, CT scan of abdomen & pelvis and x-ray chest. Endoscopic examination was performed showed erosive antral gastritis; colonoscopy was done and findings were focal area of erythematous and congested mucosa of colon feature suggestive of patchy colitis (Figure 1, 2, 3, 4). Multiple colonic biopsies were taken. The colonic biopsy was reported as EC with 30-35/hpf eosinophils. Methyl-prednisolone 40 mg/ day was started. With this treatment, the patient's symptoms regressed and patient is asymptomatic in successive follow up.

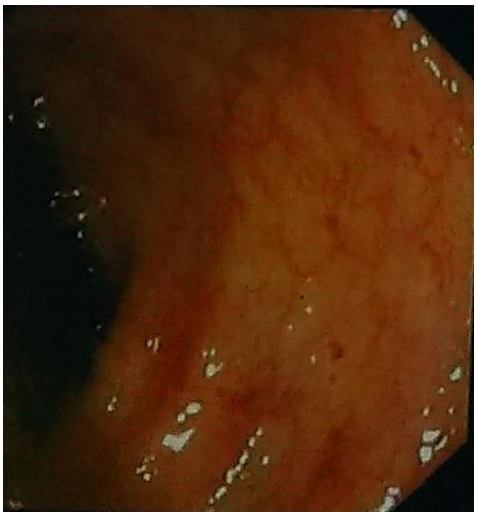

Figure 1: Colonoscopic finding erythematous and congested mucosa.

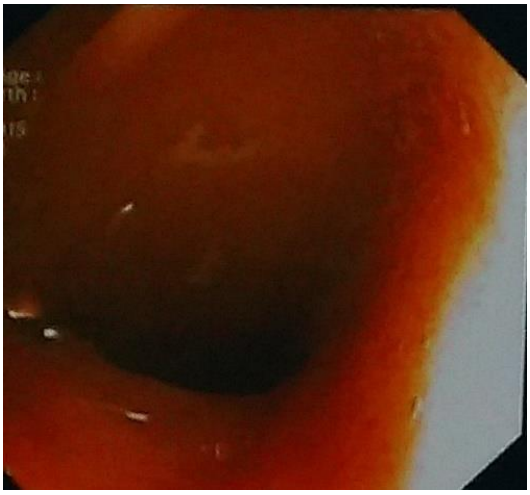

Figure 2: Colonoscopic finding erythematous and congested mucosa.

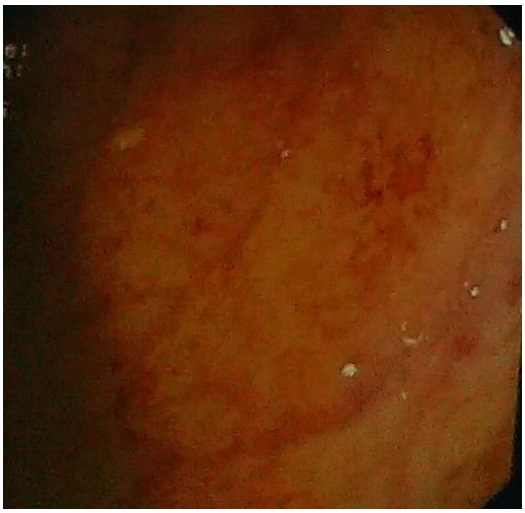

Figure 3: Colonoscopic finding erythematous and congested mucosa.

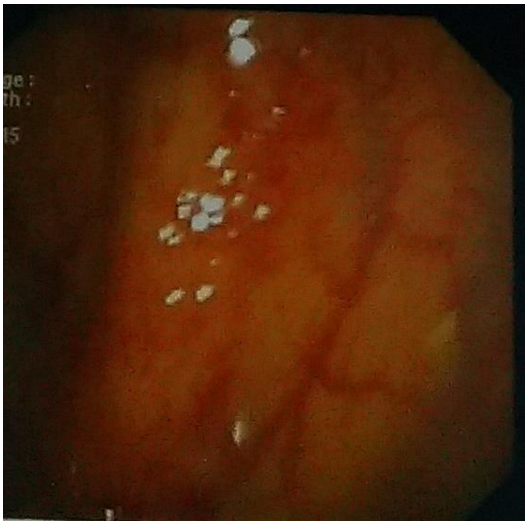

Figure 4: Colonoscopic finding erythematous and congested mucosa.

DISCUSSION

EGID in general has three hallmarks including peripheral eosinophilia (typically in the range of 5% to 35%), segmental eosinophilic infiltration of the gastrointestinal tract, and functional abnormalities.[7,8] Importantly, up to 23% of patients with primary EGID have no peripheral eosinophilia.[7] Symptoms and signs of EGID are usually non-specific and, depending on the affected segment, include abdominal pain, nausea, vomiting, diarrhea, gastrointestinal bleeding, obstruction, malabsorption, weight loss, and ascites. In 1970, Klein et al.[9] subdivided the disease based on the layer of intestinal wall most extensively infiltrated by eosinophils, to distinguish mucosa-predominant, muscularis-propria predominant, and serosa-predominant forms of EGID. The above classification provides good correlation of the physical symptoms and signs with the pathological findings, and it is also applicable to EC. Thus, mucosa-predominant disease shows evidence of mucosal dysfunction, such as protein-losing enteropathy, malabsorption, and diarrhea. Transmural disease is recognized by symptoms of intestinal obstruction and bowel wall thickening on imaging studies. Finally, serosal involvement is distinguished by the presence of eosinophilic ascites, with up to 88% eosinophils seen on fluid analysis.[10] Accordingly, while mucosal EC results in diarrhea,[11] the transmural form has been associated with volvulus,[12] intussusception,[13,14] and even perforation,[15,16] and involvement of the intestinal serosa may manifest with ascites,[17] which was also illustrated by a case that we have encountered recently. The diagnosis of EGID is made from the presence of gastrointestinal symptoms, peripheral eosinophilia, endoscopic and histological findings, and eosinophilic ascites, with no well-defined causes of eosinophilia on thorough evaluation. A multidisciplinary task force has recently reached consensus on the diagnostic criteria of EE, including the presence of more than 15 eosinophils per high-power field in the esophageal squamous mucosa.[6] No such consensus exists for EC, although most authors have used a diagnostic threshold of 20 eosinophils per high-power field. Of note, normal values for tissue eosinophils vary widely between different segments of the colon, ranging from <10 eosinophils per high-power field in the rectum to >30 in the cecum,[5] thus location of the biopsy is critically important for interpretation of findings.

More or less prominent tissue eosinophilia in the colon may result from a number of conditions and EC remains therefore a diagnosis of exclusion. Colonoscopic biopsies obtained from patients with inflammatory bowel disease, in particular with Crohn's colitis, often show severe tissue eosinophilia.[18] Parasitic infection of the colon with pinworms, roundworms, or whipworms may lead to marked eosinophilic infiltration, and repeated stool or serological testing may be needed to reveal this specific etiology.[19-23] Drug-induced EC has been described in response to clozapine,[24] carbamazepine,[25] rifampicin,[26] non-steroidal anti-inflammatory agents,[27,28] tacrolimus,[29] and gold.[30] EC has also been associated with autoimmune connective tissue disease including scleroderma, dermatomyositis and polymyositis,[11,31,32] as well as with allogeneic bone marrow transplantation[33] and the rare Tolosa-Hunt syndrome that features inflammatory ophthalmoparesis.[34] The idiopathic hypereosinophilic syndrome (HES) may also affect the colon, but this rare condition presents with sustained and marked peripheral eosinophilia with end-organ damage that extends beyond the gastrointestinal tract (e.g. heart and skin).[35]

The etiology of primary EGID remains largely unknown. Several studies have suggested a relationship with specific food allergies; indeed, about 75% of affected patients have a history of allergy or atopy. Cow's milk and soy proteins are the foods most frequently implicated in the infantile form of EC, although the condition has been described in infants exclusively breast-fed or given protein hydrolysate formulas. Even less is known about the potential causes of the adult form of primary EC. A case report by Inamura et al.[36] has demonstrated accumulation of mast cells in the colon interstitium after immunohistochemical staining for mast cell tryptase, which suggests the pathogenic role of IgE, while other observations suggest that EC may not be an IgE-mediated disease. Thus, colonic T cells in an animal model have been shown to transfer oral antigen-induced diarrhea to naive mice through a STAT6-dependent mechanism.[37] Specific eosinophil chemoattractants, such as interleukin-5 and eotaxins, may also have a pathogenic role in EC.[38] While EE may develop without other gastrointestinal involvement when experimental animals are sensitized and challenged in the lung, direct exposure of the gastrointestinal mucosa seems to result in multisegmental disease.[39] No prospective randomized controlled trials exist to date on specific therapy for EC or any other forms of primary or idiopathic EGID. Therapeutic efforts have been based on case reports and small case series. Corticosteroid therapy has formed the backbone for initial management, and it has proven to be the most effective instrument for symptom control in EC.[2,40,41] Up to 90% of cases will respond within 2 week of treatment, when a slow taper is initiated. However, relapse is frequent and requires recurrent courses or leads to steroid dependence. A role for budesonide has been demonstrated, particularly in disease of the right colon and ileum.[42] It must be emphasized that efforts to rule out parasitic or drug-induced EC are important since empiric treatment with corticosteroids may aggravate the patient's condition, or at least, it may be avoidable. The beneficial effect of elimination and elemental diets has been limited to cases with specific food allergies, especially in treating neonatal disease.[43] Approaches to avoid steroids by using alternative medications have been directed mostly to more prevalent forms of EGID, and expertise about their need and efficacy in EC has been limited. Antihistamine therapy in EGID appears to be gaining prominence. Ketotifen, an H1 antihistamine, has been shown to decrease symptoms as well as tissue eosinophilia.[44,45] The leukotriene inhibitor montelukast, an agent that blocks

the action of potent eosinophil chemoattractant leukotriene D4, by competitively antagonizing its receptor expressed on eosinophils, has also been found to be helpful in EGID.[46,47] Mast cell stabilizers, such as cromolyn, are effective by inhibiting release of mast cell mediators such as histamine H1, platelet activating factor, and leukotoxin.[48] More recently, the role of biologics in EGID has also been studied, with favorable outcomes reported by using monoclonal antibodies targeting interleukin 5 (mepolizumab) and IgE (omalizumab).[49-52]

CONCLUSION

Primary EC is a rare manifestation of the EGID spectrum, an emerging disorder within the primary EGIDs, although distinctly uncommon, has become better defined over the past decade. Its pathophysiology, clinical features, and natural history differ according to the age of presentation: being rather mild, self-limited, and more food-related in infants but chronic in young adults. Adult form may relapse and require short-term steroid therapy. The mode of presentation, especially in adults, depends on the colonic layer being predominantly infiltrated with eosinophils. Diagnosing primary EC is based on colonic biopsies, a situation that is especially challenging in the absence of diagnostic criteria and require careful elimination of secondary causes. Therapeutic approaches towards EC are based on case reports and small case series but randomized controlled trials are needed to establish the best therapeutic approach.

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