Eosinophilic gastroenteritis with ascites is an uncommon disorder of unknown etiology; characterized by recurrent episodes of infiltration of the gastrointestinal wall with eosinophils and peripheral eosinophilia. Eosinophilic colitis with ascites is extremely rare, in reviewing the literature (Pubmed and Medline), we learned that there is only one case reported about eosinophilic colitis with ascites.

**Case Report.** A 32-year-old man was admitted with 3-weeks history of abdominal pain, and distention. He described the abdominal pain as being generalized colicky in nature, associated with nausea, vomiting, and diarrhea 4-6 times per day resolved 2 days after admission. No obvious aggravating or relieving factors. The past medical history was unremarkable and he denied any history of smoking or alcohol consumption. Physical examination was unremarkable apart from ascites, no stigmata of chronic liver disease. On the following days, the peripheral eosinophilia, ascites and abdominal pain resolved spontaneously.

**ABSTRACT**

A 32-year-old man presented with 3-weeks history of abdominal pain and distention. Physical examination showed ascites, with no stigmata of chronic liver disease. Cytological preparations from the ascitic fluid showed a heavy population of mature eosinophils. Histological examination of colonic biopsies revealed a heavy expansion of the mucosa by sheaths of eosinophils. On the following days, the peripheral eosinophilia, ascites and abdominal pain resolved spontaneously.
A good number of those eosinophils are observed traversing the muscularis mucosa. There is no evidence of parasitic eggs, granulomas, inflammatory bowel disease, ischemia, viral inclusions, amyloid deposits or malignancy (Figure 3). On the following days, the peripheral eosinophilia, ascites and abdominal pain resolved spontaneously. Accordingly, the patient was discharged.

**Discussion.** Eosinophilic gastroenteritis (EGE) is a rare condition of unknown cause characterized by peripheral eosinophilia, eosinophilic infiltration of the gastrointestinal tract, and gastrointestinal symptomatology.1 Eosinophilic colitis with ascites is extremely rare, in reviewing the literature (Pubmed and Medline), 49 cases of eosinophilic colitis were reported in adults, from March 1953 until December 2004, of these only one case of eosinophilic colitis with ascites was reported.2 Nine cases of eosinophilic colitis with malabsorption were reported mostly related to intestinal worms.3,11 Klein et al.12 suggested a classification, based on the histology of the lesion: mucosal, muscularis, and subserosal disease. The signs and symptoms of eosinophilic gastroenteritis are related to the layer(s), and extent of bowel involved with eosinophilic infiltration: mucosa; muscle; and subserosa.13 The prevalence of each subtype is unknown as reporting and referral biases. Surgical series report a predominance of muscular disease with obstruction,2 while medical series primarily describe patients with mucosal involvement.14,15

Eosinophilic mucosal infiltration produces nonspecific symptoms, which depend upon the organ(s) involved. The entire gastrointestinal tract from the esophagus to the colon, including bile ducts, can be affected.15,17 Serosal eosinophilic infiltration, the rare form of presentation, may result in development of eosinophilic ascites. Our patient had predominantly subserosal type of eosinophilic colitis. This layer involvement usually occurs as a part of a transmural infiltration of the gastrointestinal tract layer as in the case being described here. Erythrocyte sedimentation rate is normal or high in eosinophilic gastro enteritis with peripheral eosinophilia; in our patient ESR was 2 mm/l hour, and the peripheral eosinophilia was obviously elevated (10,000/uL). The evidence of elevations in IgE suggested that atopy might be involved in the pathogenesis of the disease. Immunoglobulin E level was high in our patient. The endoscopic appearance in eosinophilic gastroenteritis is non-specific, including erythematous, friable, nodular, and occasional ulcerative changes. Definitive diagnosis requires histological evidence of eosinophilic infiltration. Eosinophilic infiltrates are usually patchy in distribution and may be present in otherwise, normal, non-inflamed bowel wall.

In our patient, we carried out different examinations, such as gastroduodenoscopy and colonoscopy; the biopsies taken from upper gastrointestinal were normal, while the lower gastrointestinal biopsies showed eosinophilic colitis. Eosinophilic gastroenteritis either will remit spontaneously2 as our patient remitted spontaneously and did not require any treatment, or it probably progress to malabsorption13 in a form of iron deficiency anemia, which we can treated easily with iron supplements, or diarrhea that can managed with antidiarrheal agents. In severe cases of eosinophilic gastroenteritis, we can used steroids in a dose of 20-40 mg/day. Flare-up after tapering the steroids could happen, which requires another dose of steroids. Eosinophilic ascites have a wide differential diagnosis including hyper-eosinophilic syndrome, lymphoma, strongyloids stercoralis infection, ruptured hydatid cyst, and EGE. Thus, we should always considered EGE in the differential diagnosis of eosinophilic ascites.
References