

Case Report

An Unusual Cause of Ascites

Khalid A Al-Mekhaizeem, Maher Kalaoui, Iqbal Siddique
Department of Gastroenterology, Al-Amiri Hospital, Kuwait

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ABSTRACT

Eosinophilic gastroenteritis (EGE) can rarely present with ascites. We present a case of eosinophilic gastroenteritis with serosal involvement.

KEYWORDS: ascites, eosinophilic gastroenteritis, serosal layer

INTRODUCTION

In clinical practice, ascites is usually attributed to liver cirrhosis, cardiac failure and nephrotic syndrome. Other under-recognized causes are via primary serosal involvement. Eosinophilic gastroenteritis (EGE) is a rare gastrointestinal disorder, characterized by gastrointestinal symptoms, peripheral eosinophilia and eosinophilic infiltration of the gastrointestinal tract. The later can involve mucosal, muscular and rarely serosal layers of the gastrointestinal tract. We describe a case of EGE, with mucosal and serosal involvement of the gastrointestinal tract presenting with abdominal pain and ascites. This case will be of clinical interest because it increases the awareness of an under-recognized cause of ascites.

CASE REPORT

A 24-year-old male was referred to our institution in April 2000 for evaluation of abdominal pain and vomiting of two years duration. He described the abdominal pain as recurrent episodes of mid-epigastric discomfort precipitated by food and associated with nausea and vomiting. There was also associated weight loss of more than 15 kg over the preceding two years.

His past medical history was unremarkable and he was not taking any regular medication. He denied smoking or alcohol consumption. There was no family history of inflammatory bowel disease, colon cancer, atopy, allergic rhinitis, or food allergy. The review of the systems was otherwise unremarkable, and there were no risk factors for chronic liver disease.

Physical examination revealed a young man in no apparent distress. There was no pallor, jaundice, or any stigmata of chronic liver disease. Vital signs

revealed a blood pressure of 130/80 mmHg, a regular pulse rate of 88 bpm, respiratory rate of 16/min and temperature of 37 °C. Cardiac examination revealed normal heart sounds with no added sounds, and jugular venous pressure was not elevated. Examination of the chest revealed normal breath sounds. Abdominal examination showed only abdominal distension positive for shifting dullness suggestive of ascites. The remainder of the physical examination was within normal limits.

Blood tests revealed the following: hemoglobin 157 g/l, hematocrit 0.457, mean corpuscular volume 83fl, mean corpuscular hemoglobin 31.4pg, white blood cell count 16.5/mm, neutrophil count 57%, monocyte count 2%, lymphocyte count 24%, eosinophil count 17% (absolute eosinophil count 2,805), platelet count $292 \times 10^9/l$, urea 5.3 mmol/l, creatinine 111 mmol/l, total protein 74 g/l and albumin 38g/l. Liver enzymes demonstrated, an alkaline phosphatase 81 IU/L (normal 30-110), alanine transaminase 12 IU/L (normal 5-40), aspartate transaminase IU/L 40 (normal 5-40) and total Bilirubin was 16 (normal 3-17).

Immunological work-up showed C-reactive protein 44.3 mg/l (normal < 8), C-ANCA, P-ANCA, C3 and C4 were all within normal limit. Stool examination was negative for ova or cyst of parasites. Abdominal ultrasound showed a small amount of ascities, but was otherwise a normal study. An ascitic tap was done under ultrasound guidance, which reveal a total protein 53 g/l, albumin 30 g/l. The serum-ascites albumin gradient (SAAG) was 8. The total white blood cell counts 11,800/mm, 12% neutrophil, 8% lymphocyte, and 80% eosinophil. Bacterial and acid fast stains and cultures were all negative.

Address correspondence to:

Maher Kalaoui MD, MRCP, Amiri Hospital, Gastroenterology Center, P.O. Box: 4077, Safat - 13041, Kuwait. Tel.: 2469628; fax: 2469628.
E-mail: Mkalaoui@hotmail.com

Subsequently, an upper gastrointestinal endoscopy was performed. It showed the esophagus to be normal. There was mild mucosal edema with hyperemia involving gastric body and antrum. The duodenal bulb was normal, however, there was severe edema and hyperemia extending from second duodenal part to proximal jejunum. Histological examination of the biopsy from the second duodenal portion showed mucosal and muscular infiltration with eosinophils. This was compatible with the diagnosis of EGE (Fig. 1).

Colonoscopy showed normal findings up to the cecum, ileoscopy showed severe edema and hyperemia involving the terminal ileum, and biopsy was also consistent with the diagnosis of EGE.

Oral prednisolone at 40/mg was initiated, which was followed by a marked clinical improvement in the abdominal pain and ascites over a 1 week period. The peripheral eosinophilia and abdominal pain resolved. The patient was discharged home in a satisfactory condition.

In outpatient follow-up, the patient's clinical status remained normal and prednisolone was tapered over a 4-month period. One week after stopping the Prednisolone, the patient had a recurrence of his primary symptoms which responded promptly to 5 mg/day of prednisolone. Currently the patient is maintained on this dose and advised for regular outpatient visits.

DISCUSSION

Ascites, which denotes an accumulation of fluid within the peritoneal cavity, has multiple etiologies. The two main pathophysiological defects are increased hydrostatic pressure (portal pressure) or decreased oncotic pressure. The former is usually caused by chronic liver disease with cirrhosis and cardiac failure. The later is attributed to nephrotic syndrome hypoalbuminemia secondary to malnutrition or protein losing enteropathy.

Other less defined mechanisms are lymphatic obstruction and/or leak, and direct peritoneal inflammation and/or infiltration. This direct peritoneal involvement will result in weeping of the fluid inside the peritoneal cavity. Causes of this include peritoneal carcinomatosis, tuberculous peritonitis, pancreatic ascites, biliary ascites, serositis in connective tissue disease and EGE.

The serum minus ascitic fluid albumin is a useful tool in distinguishing various causes of ascites. Conditions associated with portal hypertension such as liver cirrhosis or congestive heart failure are associated with a high gradient (> 11 g/L). In contrast, conditions which cause ascites without raising portal pressure such as tuberculous peritonitis and peritoneal carcinomatosis are characterized by a low gradient (< 11 g/L)^[1].

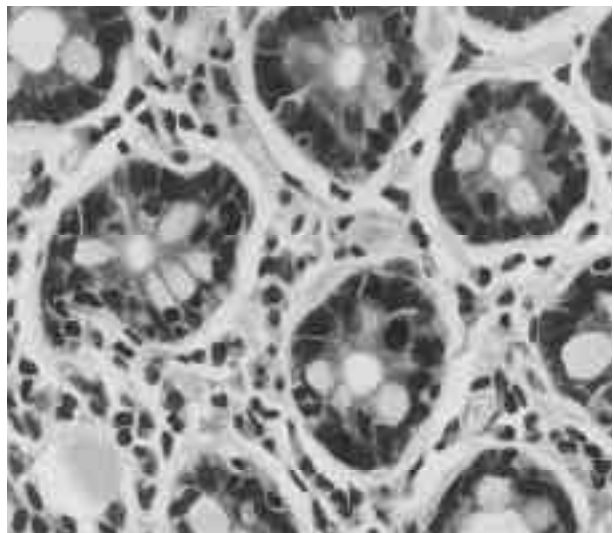


Fig. 1: Duodenal biopsy showing an increased number of eosinophils in the lamina propria. The arrow points to one with prominent granules. (Magnification 400x, H&E Stain)

Eosinophilic gastroenteritis is a disease of unknown etiology, which can affect a wide age group that extends from infancy to adulthood^[2]. Although both sexes can be affected, it has a male gender predominance^[3].

Eosinophilic gastroenteritis is characterized by eosinophilic infiltration of the mucosal, muscular, or the serosal layer of the gastrointestinal tract. The clinical spectrum of this entity correlates with the layer being involved. Mucosal and muscular involvement will result in diarrhea, weight loss, abdominal pain and vomiting. Serosal infiltration by eosinophils leads to serosal inflammation with subsequent weeping of the fluid. This mechanism is responsible for the formation of exudative ascities with low SAAG in our patient. The subserosal type is the rarest form, since it accounts for only 10% of the reported cases in the literature^[4].

This layer involvement usually occurs as a part of a transmural infiltration of the gastrointestinal tract layer as in the case being described here, although rare cases of isolated sub-serosal type have been described^[5].

Our patient's clinical presentation, which consists of high peripheral eosinophil count, eosinophilic ascites, abdominal bloating and the dramatic response to steroids, denotes a predominant subserosal infiltration. This is an infrequent presentation of this already uncommon disease^[6-8]. This clinically interesting condition may present as an isolated entity^[9] or could accompany other disorders such as Crohn's disease, allergic granulomatosis, polyarteritis nodosa, idiopathic hypereosinophilic syndrome^[9]. The diagnosis of this disorder needs a high index of suspicion and is confirmed by tissue biopsy demonstrating eosinophilic infiltration of the gastrointestinal tract.

The presenting symptoms in our patient were diverse, but the clinical detection of eosinophilic ascites, followed by confirmatory jejunal biopsy demonstrating eosinophilic infiltration resulted in the correct diagnosis.

Our case underscores several important points. First, ascites have a wide differential diagnosis besides chronic liver disease, cardiac failure and nephrotic syndrome. Second, the low serum to ascites albumin gradient is instrumental in ruling out portal hypertension and is consistent with peritoneal inflammation. Third, in the serosal type which is the rarest form of EGE, ascites is a usual presenting feature. Fourth, although ascitic fluid eosinophilia has a wide differential diagnosis including hypereosinophilic syndrome, lymphoma, strongyloids stercoralis infection and ruptured hydated cyst, EGE should always be considered in the right clinical context. Finally, early recognition and treatment of this condition will guard against unnecessary procedures and possible complications.

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