Eosinophilic gastroenteritis, complicated with Eosinophilic ascites, acute pancreatitis and chronic diarrhea: A rare presentation of hyper eosinophillic syndrome.

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Abstract: Eosinophilic gastrointestinal disorders (EGID) are one of rare causes of chronic diarrhea. The disorders are characterized by inflammation rich in eosinophilic infiltration in the gastrointestinal(GI) tract without evidence of known causes for eosinophilia such as parasitic infection, drug reaction, or malignancy [1]. It was originally described by Kajserin 1937. EGID can involve one or multiple segments of the GI tract from the esophagus to the rectum (mainly in the antrum of the stomach and small intestine) and can also occupy various sites through the depth of the wall [2]. Clinical manifestations range from non-specific gastrointestinal complaints to more specific symptoms such as protein-losing enteropathy, malabsorption, luminal obstruction and eosinophilic ascites. Thus it is an easily missed condition that needs more awareness from the gastroenterologists and general internists. We report the case of a 30-year-old woman with chronic diarrhea and ascites presenting as acute pancreatitis, a rare documented presentation of Eosinophilic gastroenteritis.

Keywords: Eosinophilic gastroenteritis, Eosinophilic ascites, Chronic diarrhea

I. Case Report

A 30-year-old male was admitted to our hospital with complaints of diarrhea, abdominal pain, and weight loss for about 3 months. The patient had developed intermittent watery diarrhea occurring after meals with an average of 4 or 7 stool passages each day. Stool was yellowish and watery in character, and ended with lower abdominal discomfort. During this period, body weight decreased from 55 kg to 47 kg, and was accompanied by generalized weakness. His abdominal pain was dull and diffuse and epigastric in location, mainly post prandial, associated with occasional vomiting, which was non projectile, non bilious in nature. There was no history of any fever. The patient had been admitted to other hospitals several times and had been evaluated for his symptoms. However, no specific cause of the chronic diarrhea had been identified. Two weeks previous to the current checkup, the symptoms had recurred especially the pain which was now associated with abdominal distention, which prompted the visit to our center. Pain was more in intensity to what he used to have during the course of his illness and distention was progressive. The patient had no personal and family history of allergic disorders such as asthma, atopy, allergic rhinitis, and other hypersensitivities, and denied any exposure to tobacco smoke, alcohol, drugs, herbal medications.

Examination: On admission, the patient appeared chronically ill and emancipated. Vital signs were stable including blood pressure 110/70 mm Hg, heart rate 74 beats/minute, respiration rate 20 breaths/minute and he was afebrile through course of diseases. The conjunctiva was anemic and the sclera was anicteric. There was no cervical lymphadenopathy. He was dehydrated and there was no edema or skin rash. Thyroid examination was normal. On auscultation, the lung fields were clear. Cardiac examination revealed no murmur or gallop. Abdominal examination: On inspection revealed distended abdomen with everted umbilicus. On palpation there was mild tenderness but no rebound tenderness present on deep palpation there was no hepatomegaly or abdominal mass. On percussion there was fluid thrill present and percussion note was stony dull. On auscultation there was hyper active bowel sounds heard. Neurological examination revealed no deficits or muscle weakness.

Investigation: Laboratory findings were as follows: leukocyte count 8,800/mm³ (neutrophil, 50.5%; lymphocyte, 39.7%; eosinophil, 25.4%), total eosinophil count 1,180/mm³ (normal range, 0 to 500/mm³), hemoglobin 9.7 g/dL, erythrocyte sedimentation rate 21 mm/hour, and C-reactive protein 0.233 mg/dL. Biochemical tests were within normal limits other than total protein 5.49 g/dL and albumin 2.74 g/dL. Liver function test serum SGOT 34U/L, SGPT 40 U/L and alkaline phosphatase was 112 U/L. Serum creatinine was 1.6 mg/dL, serum urea 23 mg/dL. Stool was negative for occult blood, ova and parasites. Fecal fat content was normal. Stool culture showed no growth. On third day of admission he developed severe pain abdomen serum amylase & lipase were done which were high 605 IU/l & 788 IU/l respectively.
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Bone marrow examination showed eosinophils in the marrow. Ultrasound-guided abdominal paracentesis showed WBC count of 2400/mL, 90% of which were eosinophils. DLC: P2 L8 E90, ADA 24. Gram stain and AFB were negative, and there was no evidence of malignant cells. Peripheral smear for microfilaria, ANA, ANCA were negative. Serum IgE level was elevated at 548 IU/mL (normal < 180). Abdominal and pelvis computer tomography (CT) showed moderate ascites with mesenteric and gut wall (anterior pyloric) thickening (Fig1).

Fig1: Upper GI endoscopy showed hyperemia of antral mucosa. Duodenum (1st part) showed evidence of edema along with multiple whitish nodular lesions.

Fig2: Duodenum biopsy showed normal villous pattern with mild inflammation, eosinophils were present.

Fig3: The constellation of clinical presentation and histopathological findings were suggestive of eosinophilic gastroenteritis. Subsequently, the patient was started on oral steroid 40 mg prednisolone daily.

Two weeks later, with noticeable symptomatic improvement, the prednisone was tapered over a period of next three weeks. After completion of steroids, the patient’s abdominal pain and physical finding of ascites completely resolved and a peripheral blood count revealed an absolute eosinophil count of 300/μl (N < 450).

**II. Discussion:**

EGID consist of heterogeneous subtypes including eosinophilic esophagitis, eosinophilic gastritis, eosinophilic gastroenteritis, and eosinophilic colitis. EGID are exceedingly rare, lacking epidemiological data to estimate their true frequency. EGID act on all races and ages, from infancy through adulthood[3]. There are three subtypes of EGE (mucosal, muscular, and subserosal). Mucosal involvement is by far the most common,[4] and is accompanied by one or more of the following symptoms: decreased appetite, nausea, vomiting, abdominal pain, diarrhea, GI bleeding, protein-losing enteropathy.[5] Serosal involvement, the least common, is accompanied by abdominal distention and eosinophilic ascites.[6] Our patient had chronic diarrhea and with weight loss and presented to us with acute pancreatitis and abdominal ascites and peripheral...
Eosinophilic gastroenteritis, complicated with Eosinophilic ascites, acute pancreatitis and Chronic eosinophilia. Ascitic fluid showed transudative nature with Eosinophilia. Pancreatitis seemed to be of unclear etiology. The diagnosis of EGE is established on high clinical suspicion in conjunction with suggestive histopathologic findings. Although peripheral eosinophilia is very common in all subtypes of EGE, it can be absent in as high as 23% of cases. Before we make a diagnosis of EGED other secondary causes for eosinophilia must be ruled out which include stool examination for ova and parasitic cyst, skin allergy testing and connectivetissue profile[5]. In our cases work up for secondary causes was negative. Peripheral blood eosinophilia is suggestive of EGID, but is noted in only 60-80% of the patient[7]. Endoscopic findings may be nonspecific and can include erythema, friability, ulcerations, erosions, nodules, and loss of vascularity.[8] Biopsy is highly suggestive of EGE it has been observed that in up to 10% of the cases biopsy may not be helpful to reach to a diagnosis and diagnosis can be missed in upto 25% of cases [9]. The recommended dose is prednisolone 20-40 mg/day for 1-2 weeks. The dose isthen tapered off over several weeks.(1) Up to 90% of cases will respond dramatically within 2 weeks of treatment. However, a maintenance dose of prednisolone (10 mg/day) may be continued in many cases with recurrence of symptoms. (10) The most interesting feature in our case involved the episode of acute pancreatitis. A pattern of epigastric pain and elevation of serum amylase 4-5 times the normal value was seen. The patient had no history of gallstones or overconsumption of alcohol. As Eosinophils contain several cytotoxic/antihelminthic factors and proinflammatory mediators, the possibility that eosinophils may elicit pancreatitis due to a direct toxic effect has been considered. Other examples where pancreatic damage by invading eosinophils has been discussed include the hypereosinophilic syndrome. The eosinophilic infiltration of the gastroduodenal wall may have led to the obstruction of the biliary and pancreatic ducts as described in some previous reports [11]. Our patient responded to steroid therapy and was managed conservatively for acute pancreatitis.

III. Conclusion

In the present case, the patient presented with chronic diarrheaea and lower abdominal pain and distention and EGID was documented by eosinophilic infiltration on endoscopic biopsy and exclusion of secondary causes. This case report reviews some of the characteristic clinical, laboratory, and histopathological findings of a rare, readily treatable, and easily missed disease. Due to the relatively nonspecific symptoms, this diagnosis should be considered in patients with pancreatitis of unclear etiology, nonspecific bowel thickening by imaging studies and, otherwise, negativeworkup for parasitic infection and malignancy. Additionally, while peripheral blood or ascitic fluid eosinophilia is suggestive, its absence does not exclude the possibility of this diagnosis.

Bibliography