IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 13, Issue 1 Ver I. (Jan. 2014), PP 41-43
www.iosrjournals.org

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

Tanveer H. Banday[1], Sadaf Bashir Banday[2], Irfan Wani[3], Shah Naveed[4], Jagadeesh S. G.[5], Ashwin K.[6]
Assistant professor, Department of medicine, AIMS Bangalore

Abstract: *Eosinophilic gastrointestinal disorders (EGID) are one ofrare causes of chronic diarrhea. The disorders are characterizedby inflammation rich in eosinophilic infiltration in the gastrointestinal(GI) tract without evidence of known causes foreosinophilia such as parasitic infection, drug reaction, or malignancy [1]. It was originally described by Kaijserin 1937.EGID can involve one or multiple segments of the GI tractfrom the esophagus to the rectum (mainly in the antrum of the stomach and small intestine) and can also occupy various sitesthrough 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 diarroheea and ascites presenting as acute pancreatitis, a rare documented presentation of Esonophillic gastroenteritis*.
Keywords: *Eosinophilic gastroenteritis, Esonophilic ascites, Chronic diarrhea*

I. Case Report

A 30-year-old malewas admitted to our hospital with complains of diarrhea, abdominal pain, and weight loss for about 3months . The patient had developed intermittentwatery diarrhea occurring after meals with anaverage of 4 or 7 stool passages each day. Stool was yellowish andwatery in character, and ended withlower abdominal discomfort. During this period, body weight decreasedfrom 55 kg to 47 kg, and was accompanied by generalizedweakness. 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 hospitalsseveral times and had been evaluated for his symptoms. However, no specific cause of the chronic diarrheahad 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 ourcenter. 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 emanciated. Vitalsigns were stable including blood pressure 110/70 mm Hg,heart rate 74beats/minute, respiration rate 20breaths/minute and he was afebrile throught course of diseases. The conjunctiva was anemic and thesclera 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 normallimits other than total protein 5.49 g/dL and albumin 2.74 g/dL. Liver function test serum SGOT 34U/LSGPT 40 U/L and alkaline phosphatase was 112 U/L .Serum creatinine was 1mg/dl serum urea 23mg/dl .Stool was negative for occult blood, ova andparasites. Fecal fat content wan normal.Stool culture showed no growth. On third day of admission he developed severe pain abdomen serum amylase& lipase were done which were high 605IU/l &788 IU/l respectively.

Bone marrow examination showed eosinophills 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(anteropyloric)thickening(fig1)

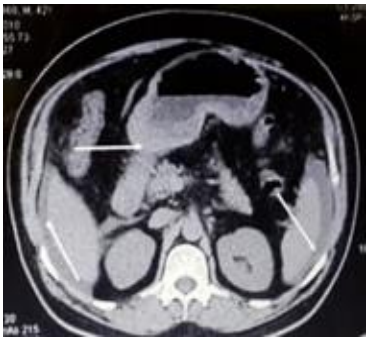

Fig1:Upper GI endoscopy showed hyperemia of antral mucosa, Duodenum (1st part) showed evidence of edema along with multiple whitish nodular lesions

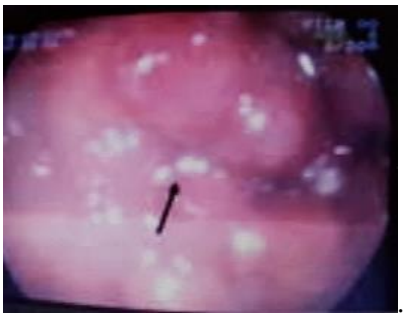

Fig2Duodenum biopsy showed normal villous pattern with mild inflamation, eosinophilis were present

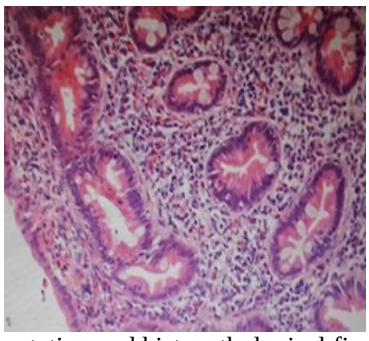

Fig3The constellation of clinical presentation and histopathological findings were suggestive of eosinophilic gastroenteritis. Subsequently, the patient was started on oral steroid 40mg prednisolone daily.

Two weeks later with noticeable symptomaticimprovement, the prednisone was tapered over aperiod of next three weeks . After completion of steroids, the patient'sabdominal pain and physical finding of ascites completelyresolved and a peripheral blood count revealed an absolute eosinophil count of 300/µl (nL< 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 toestimate their true frequency. EGID act all races and ages, frominfancy through adulthood[3].There arethree 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, isaccompanied by abdominal distention and eosinophilic ascites**[6]**.Our patient had chronicdiarrhea and with weight loss and presented to us with acute pancreatitis and abdominal ascites and peripheral

esonophilia.Asciticfluid analysis showed transudative nature with Esonophilia. 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 secondarycauses for eosinophilia must be ruled out which include stoolexamination for ova and parasitic cyst, skin allergy testing and connectivetissue profile[5]. In our cases work up for secondarycauses was negativ**e**. 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 hasbeen observed that in upto**10%** of the cases biopsy may notbe helpful to reach to a diagnosis and diagnosis can be missed in up**to 25%** of cases [9].The recommended doseis 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 does 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 Esoninophils 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 diarrheaand lower abdominal pain and distention and EGID was documented by eosinophilic infiltration on endoscopicbiopsy and exclusion of secondary causes. This case report reviews some of the characteristic clinical, laboratory, and histopathological findings of a rare, readilytreatable, and easily missed disease. Due to the relativelynonspecific symptoms, this diagnosis should be consideredin 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

[1] Yan BM, Shaffer EA. Primary eosinophilic disorders of the gastrointestinal tract. Gut 2009;58:721-32.
[2] Klein NC, Hargrove RL, Sleisenger MH, Jeffries GH. Eosinophilic gastroenteritis. Medicine (Baltimore) 1970;49: 299-319.
[3] Khan S, Orenstein SR. Eosinophilic gastroenteritis: epidemiology, diagnosis and management. Paediatr Drugs 2002;4: 563-70.
[4] M. J. Chen, C. H. Chu, S. C. Lin, S. C. Shih, and T. E.Wang, "Eosinophilic gastroenteritis: clinical experience with15 patients," World Journal of Gastroenterology, vol. 9, no. 12,pp. 2813–2816, 2003.
[5] Dong Ryul Lee: A Case of Eosinophilic Gastrointestinal Disorders Presenting with Chronic Diarrhea and Abdominal Pain Korean J Fam Med. 2011;32:257-262
[6] M. P. S´anchez-Fayos, R. Miranda, L. Renedo, J. C. Porres, andC. H. Gu´ıo, "Eosinophilia and ascites as an expression of a subserous form of eosinophilic gastroenteritis," Revista Clinica Espanola, vol. 191, no. 1, pp. 30–34, 1992.
[7] Straumann A, Simon HU. physiological and pathophysiological roles of eosinophils in the gastrointestinal tract. Allergy 2004; 59:15-25.
[8] Feldman M, Scharschmidt B, Sleisenger M. Sleisenger and Fordtran's gastrointestinal and liver disease. 6th ed. Philadelphia: Saunders; 2006
[9] N. J. Talley, R. G. Shorter, S. F. Phillips, and A. R. Zinsmeister,"Eosinophilic gastroenteritis: a clinicopathological study ofpatients with disease of the mucosa, muscle layer, andsubserosal tissues," Gut, vol. 31, no. 1, pp. 54–58, 1990.
[10] Fleischer DM, Atkins D. Evaluation of the patient withsuspected eosinophilic gastrointestinal disease. ImmunolAllergy Clin North Am 2009;29:53-63.
[11] Mohandas KM, SanthiSwaroop V, Desai DC, Jagannath P, Krishnamurthi S, DeSouza LJ. Pancreatic and biliary obstruction due to eosinophilicgastroenteritis. Am J Gastroenterol 1990; 85:15401.