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Eosinophilic gastroenteritis: Atypical cause for chronic diarrhea in human immunodeficiency virus-associated immunosuppression

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Abstract

Eosinophilic gastroenteritis (EGE) is an uncommon disease in both immunocompetent and immunocompromised. We describe a 57-year-old male with human immunodeficiency virus who presented to us with chronic diarrhea. He had no history of allergies and had significant weight loss, normal systemic examination, and a complete blood count showing no eosinophilia. After an esophagogastroduodenoscopy, the diagnosis of EGE was made by histopathological findings. The symptoms started improving with the initiation of treatment with oral prednisolone.

Key words: Cachexia, esophagogastroduodenoscopy, prednisolone

INTRODUCTION

Primary eosinophilic gastroenteritis (EGE) has not been reported before as an etiology for chronic diarrhea in patients infected with human immunodeficiency virus (HIV). After ruling out common etiologies such as infections and drugs, EGE should be considered as a differential diagnosis despite the patient being immunosuppressed.

CASE REPORT

A 57-year-old Indian male with HIV-positive status was admitted to our hospital with chief complaints of large-volume watery stools without blood or mucus, 3–4 times a day for 4 months with recent exacerbation for 1 week. This was also associated with nausea, loss of appetite, significant weight loss, generalized weakness, and dysphagia to solids. He denied personal or family history of allergies. He was diagnosed with HIV infection 17 years ago and was on treatment with antiretroviral drugs, namely raltegravir, lamivudine, and efavirenz. He was also diagnosed with pulmonary tuberculosis 5 months back for which he was on treatment with isoniazid, rifampicin, pyrazinamide, and ethambutol. He unintentionally lost 20 kg of weight over the past 4 months. He further denied the use of laxatives or any complementary or alternative medications. He had stable vital signs, was cachectic, and had a normal systemic examination. His workup for chronic diarrhea included a normal thyroid function test and no opportunistic infections or helminthic infestation. Stool analysis including culture was negative for *Clostridium difficile* toxin, *Cryptosporidium*, *Vibrio cholerae*, acid-fast bacteria, or any other parasites. Serum IgE concentration was

also within the normal range. Other investigations revealed hemoglobin 11.4 g/dL (13–18 g/dL), platelets 244,000/uL (140,000–440,000/uL), and total leukocyte count 4880/mm³ (4000–10000/mm³) with neutrophils 78.5% (40%–75%), lymphocytes 16.2% (20%–45%), and eosinophils 0.8% (1%–6%). His total protein was 5.4 g/dL (6.60–8.70 g/dL), albumin 1.8 g/dL (3.97–4.94 g/dL), absolute CD4 count 89.91/mm³ of blood (400–1600/mm³ of blood), absolute CD8 count 427.68/mm³ of blood (280–1400/mm³ of blood), CD4 (helper T-cells)% 11.1% (28–58%), CD8 (suppressor T-cells)% 52.8% (19–49%), CD4/CD8 0.21 (1.2–3.3), and HIV-1 viral load by TaqMan plasma (quantitative) 12,59,309 copies/mL.

On further investigations, esophagogastro duodenoscopy revealed white mucosal plaques in the esophagus consistent with esophageal candidiasis. The mucosa of the duodenum showed focal erosions with blunting of the villi as seen in [Figure 1](#). Urease test was negative for *Helicobacter pylori*. Mucosal biopsies from the stomach and duodenum were taken. Histopathology study shown in [Figure 2](#) revealed active inflammation in the gastric and duodenal mucosa with excess eosinophils (20 eosinophils/high-power field) and no microorganisms. This was consistent with EGE. He also had a colonoscopy which was normal. Rifampicin was stopped during this admission for a period of 2 weeks. However, the persistence of diarrhea ruled out drug-induced EGE. He was then started on oral prednisolone 40 mg once daily. The frequency of stools decreased and consistency improved. Prednisolone was gradually tapered over a period of 2 months. This was associated with an increase in the frequency of watery stools like before resulting in a readmission with dehydration and weight loss.

The patient was restarted on oral prednisolone 60 mg once daily. Along with cessation of diarrhea, his appetite and weight improved during follow-up after 30 days. On 6-month follow-up, he has regained 18 kg and has normal stools. He is currently on oral prednisolone 40 mg once daily.

DISCUSSION

Primary eosinophilic gastrointestinal disorders are disorders of the gastrointestinal tract that include eosinophilic esophagitis, eosinophilic gastritis, EGE, eosinophilic enteritis, and eosinophilic colitis, characterized by eosinophilic-rich inflammation in the absence of any known causes of eosinophilia.[1] Eosinophils are present in the mucosal, muscular, or subserosal layers in excess of 20 per high-power field[2] and more commonly involve the stomach and duodenum[3] as seen in our patient. Although serum eosinophil counts are frequently elevated, up to 20% of patients have been reported to have normal counts.[2] Large-scale population studies in the US have estimated the prevalence of EGE to be 28/100,000 persons.[4] Eosinophilic gastrointestinal disorders affect all ethnic groups between 20 and 50 years of age but more commonly around the third decade of life.[5] Unlike in the esophagus, baseline eosinophils are detected in the stomach and intestine under healthy conditions as a part of the host-defense mechanism.[1,5] However, their infiltration to the deeper layers is considered abnormal. Eosinophilia in the intestinal mucosa is not seen with infections caused by common bacteria, viruses, and fungus. Eosinophils play an important role against infections caused by helminths, *Dientamoeba fragilis*, *Mycobacterium* spp., *Isospora belli*, *Sarcocystis*, *Coccidioidomycosis*, and HIV.[5]

In our patient, diarrhea persisted even after stopping antituberculosis therapy, thus ruling out secondary causes of EGE.[2] Primary EGE has not been reported before in HIV-associated immunosuppression and should be kept as a differential diagnosis in patients with HIV presenting with chronic diarrhea.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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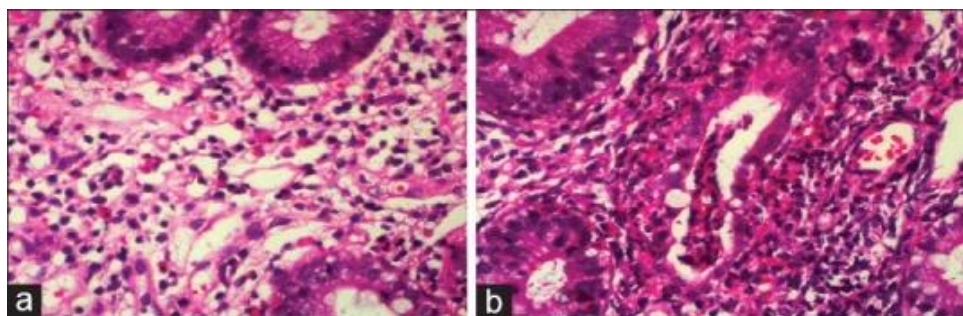
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Figures and Tables

Figure 1

On esophagogastroduodenoscopy, mucosa of the duodenum shows (a) focal erosions and (b) blunting of the villi

Figure 2

Histopathology (magnification: $\times 40$). (a) More than 20 eosinophils seen around the mucous glands and (b) the eosinophils are focally forming crypt abscesses

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