A Patient with Eosinophilic Gastroenteritis Presenting with Severe Abdominal Pain

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Summary:

Eosinophilic gastroenteritis is characterized by eosinophilic infiltration of the stomach and intestine. It is a rare disorder with bizarre presentations, making it difficult to diagnose and often leading to misdiagnoses. It can present with abdominal pain, diarrhea, vomiting, obstruction, ascites etc. Here we report a case of a middle aged female who presented with severe abdominal pain and was initially suspected a case of acute pancreatitis. She received conservative management, but symptoms did not improve. This led to further evaluation, which revealed peripheral blood eosinophilia. Although the mucosa appeared normal on esophagogastroduodenoscopy, biopsy from multiple sites revealed eosinophilic infiltration in the gastric and duodenal lamina propria. The patient was then successfully treated with oral steroid. Due to its varied presentations, Eosinophilic gastroenteritis should be considered as the differential diagnosis in different abdominal presentations.

Introduction:

Eosinophilic gastrointestinal disorders (EGID) are a spectrum of conditions involving different parts of the gastrointestinal tract, principally arising from infiltration of eosinophils in different layers, giving rise to a wide variety of presentations (1, 2). It is a rare disorder. The true incidence rate is difficult to establish; however, an estimate of 1-30 / 100000 is reported (3). Eosinophilic gastroenteritis (EGE) falls under the EGID spectrum, involving the stomach and the intestines. It can be divided into three types based on the layers involved – mucosa, muscularis and serosa (4). Data regarding EGE is scarce and there is no published case report in Bangladesh to our knowledge. Here we report a case of a middle aged female with severe abdominal pain for one and a half months and later diagnosed as EGE.

Case presentation:

Normotensive, newly diagnosed diabetic female in her 40s, presented with severe abdominal pain for one and a half months. The pain was sudden in onset, severe, diffuse, colicky, aggravated after meal, partially relieved by intravenous analgesics and not relieved by change in posture. Pain was associated with vomiting, 3-4 times per day, which partially relieved the pain. There was no associated transient visible peristalsis. She had lost 2 kilograms of weight. Her bowel habit was normal. She did not give any history of fever, cough, hemoptysis, contact with smear positive pulmonary tuberculosis patient, swelling in different parts of the body, itching or night sweat.

With these complaints, she was hospitalized and clinically labeled as a case of acute pancreatitis in local hospital based on the severity of the pain. Normal serum levels of amylase and lipase were observed and due to the absence of a definitive diagnosis, conservative management was implemented with nothing per
oral and analgesics. Initially pain and vomiting was controlled to some degree, but it reappeared again and became difficult to control.

She had no history of asthma or any atopic disease or known food allergy. She had appendectomy 20 years back and caesarian section 12 years back. She has 2 children. Her daughter has asthma. There was no family history of GI malignancy.

Examination revealed a BMI of 23 kg/m² with no anemia, jaundice, lymphadenopathy or edema. Abdominal examination revealed no organomegaly or ascites.

Methods:

Complete blood count showed a high WBC count with >50% Eosinophils. Peripheral blood film revealed mature WBCs. CRP was found to be uncharacteristically high. Serum Immunoglobulin E (IgE) was increased. Hepatic and renal functions were normal. BCR-ABL was negative. Abdominal and trans-vaginal ultrasound revealed dilated bowel loops and mild pelvic collection.

Esophagogastroduodenoscopy was arranged. During the procedure, the mucosa and other components appeared to be normal. However, with a high index of suspicion of EGE due to high peripheral eosinophil count, biopsy was taken from multiple sites of stomach and duodenum. Histopathology revealed that some of the villi was short and blunt, with both gastric and duodenal lamina propria being infiltrated by numerous chronic inflammatory cells, including a fair number of eosinophils (>50/HPF)

Figure 1

Figure 2

Intestinal parasitosis is quite prevalent in Bangladesh, and our patient presenting with peripheral eosinophilia underwent a stool microscopic examination, which yielded normal results. ELISA for Echinococcal antibody was also negative. In order to exclude involvement of other organs, the patient also underwent several tests including liver function, renal function, pulmonary function, ECG, and troponin I. Despite the medical recommendation for a bone marrow aspiration to further investigate the condition, the patient declined to undergo another invasive procedure following the upper GI endoscopy. Additionally, a colonoscopy was advised to assess potential involvement of the colon, but the patient also refused to proceed with this suggested examination.

Based on these findings, we made a diagnosis of Eosinophilic gastroenteritis and started the patient on oral Prednisolone 40 mg/day for 2 weeks, with rapid taper over 2 weeks. Patient responded dramatically.

Conclusion:

Within a week, symptoms subsided, WBC count came down to normal and eosinophil count came down to 1%. Treatment was continued and she was discharged after proper counseling regarding the nature of the disease and the possibility of relapse.

Discussion:

Eosinophils are part of the GI mucosal immune system. They are principally associated with parasitic infections and allergic conditions. The pathophysiology of EGE is not well determined. The process starts with recruitment and activation of a large number of eosinophils in the GIT with consequent release of cytokines, giving rise to the wide spectrum of problems (5).

The presentation of EGE depends on the layers involved. Mucosal involvement, which is the commonest form, usually presents with abdominal pain, diarrhea, vomiting, protein losing enteropathy etc. If the muscle layer is involved, presentation is often obstruction, even perforation. Serosal involvement presents with ascites. Involvement of all three layers has also been reported in rare cases (2). In our case, based on the symptoms and eosinophilic infiltration of the lamina propria, it was of the mucosal variety.
Due to its non-specific presentation, it is often difficult to diagnose EGE, especially at an early stage. However, with peripheral eosinophilia present in over two third cases, a possibility of EGE should be kept in mind when dealing with a patient with bizarre GI symptoms (6). In our case due to the severity of abdominal pain, the local hospital clinically suspected acute pancreatitis. However, with normal enzyme levels, no definitive diagnosis was reached. Initial symptom relief was achieved with painkillers, leading to her discharge. This led to a delay in diagnosis and undue suffering for the patient. This is a difficult issue. There have been several reported cases where EGE have both caused and mimicked acute pancreatitis (7-11). There has been one where it has mimicked acute cholecystitis, leading to emergency cholecystectomy (12). This potential for varied presentation and frequent misdiagnosis has led to EGE being referred to the great chameleon.

Forty five to sixty three percent of EGE patients has a history of allergies, like asthma, allergic rhinitis, atopy etc. (13). Our patient did not give any such history, although her daughter was an asthma patient. In rare cases late cow’s milk allergy and even egg allergy has been reported, where avoidance of milk and dairy products led to significant improvement in symptoms (8, 14).

In order to exclude other organ involvement, various tests were conducted, including liver function tests, renal function tests, pulmonary function tests, and cardiac enzyme assessments. Intestinal parasitosis was also excluded, owing to its high prevalence in Bangladesh (15). However, it is essential to note that the bone marrow test, which is crucial for ruling out hypereosinophilic syndrome, was not performed due to the patient’s refusal. Neither was colonoscopy. We recognize these as limitations.

Although an established treatment protocol does not exist, steroid is widely accepted as the mainstay of treatment. Other treatment options include leukotriene inhibitors, mast cell stabilizers, antihistamines and biological agents (16). Our patient responded dramatically to oral prednisolone and did not have any relapse in our short follow up period.

EGE is a rare disorder with a non-specific presentation. This often leads to diagnostic dilemma, misdiagnosis and delayed diagnosis, resulting in significant suffering for the patient. Diagnosis mainly depends on endoscopic biopsy and histopathology. Treatment is fairly simple, usually with good results. If a higher index of suspicion is kept in atypical GI presentations, more cases will be diagnosed earlier in the future.

Figure legends:

**Figure 1:** Histopathology from duodenal tissue (4 times magnification)

**Figure 2:** Histopathology from duodenal tissue (40 times magnification)

**Patient’s perspective:**

“Four months ago, I suddenly developed severe abdominal pain and vomiting and got admitted into a nearby hospital. They put me on saline and pain medication and restricted all food. I did not eat a single piece of food for 10 days. With these measures, my pain initially subsided but it reappeared and I got admitted in the hospital. They did endoscopy of upper GIT and took a biopsy. After the biopsy report, doctors told I have eosinophilic gastroenteritis and prescribed prednisolone. After taking prednisolone my pain subsided within 2 days. My doctor told me that this disease may recur. But thanks to almighty, I am doing much better now.”

Reference


