

## Bronchial Asthma and Eosinophilic Gastroenteritis: A Case Report

Mohamed AA Bassiony\*

Faculty of Human Medicine, Zagazig University, Egypt

### Abstract

Eosinophilic gastroenteritis is a rare but highly recurrent condition of unknown aetiology. It is characterised by peripheral eosinophilia with extensive infiltration of gastrointestinal (GI) wall layers by eosinophils causing a variety of clinical features & complications. Corticosteroids & food restriction are the main treatment options and are effective in inducing remission of most patients.

**Keywords:** Eosinophils; Bronchial asthma; Eosinophilic gastroenteritis; GIT; Esophagus; Corticosteroids; Ascites; Peritonitis

\*Corresponding author: Mohamed AA Bassiony

✉ dr\_mbh13303@yahoo.com

Department of Internal Medicine, Faculty of Human Medicine, Zagazig University, Egypt.

Tel: +20 55 2364612

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### Case Presentation

Mr. Moataz is a 38-year-old Egyptian teacher. He is married & had two children. He is not smoking & had no other special habits of medical importance. He had a history of bronchial asthma for 12 years with occasional use of inhaled B2-agonists for relieve of bronchospasm. He had no known drug or food allergy. He visited the gastrointestinal clinic of our hospital 8 months ago by a complaint of heart burn, odynophagia, repeated vomiting for 25 days earlier. The patient was initially diagnosed as GERD, received proton pump inhibitors (PPI) & prokinetic for one month with only partial improvement. The patient came to the clinic again with severe odynophagia & food impaction along with generalised abdominal pain & enlargement. No diarrhea, bleeding, fever, rash or significant weight loss. He also stated that he had a poorer control of his asthma symptoms through the past 2 months with more frequent use of his reliever inhalers. Chest examination showed bilateral hyper-resonance with prolonged expiration & scattered rhonchi. Abdominal examination revealed generalized tenderness & bilateral shifting dullness with no organomegaly or lower limb edema. Pelvi-abdominal ultrasound & CT confirmed the presence of moderate ascites & mild right-side pleural effusion with normal liver, spleen, portal vessels & mesenteric lymph nodes. Upper GI endoscopy was done showing lower 1/3 esophagitis with white exudate, mild antral & fundal gastritis with four gastric polyps. Colonoscopy also was done with normal findings. Complete blood count (CBC) showed TLC:  $14.7 \times 10^3/\text{dl}$ , hemoglobin: 12.4 g/dl, platelets:  $312 \times 10^3/\text{dl}$ . Differential count: neutrophils 48%, lymphocytes 13%, monocytes 1%, eosinophils 43%, no blast cells. Serum K: 3.2mEq/dl & serum Na: 139mEq/dl. Liver & kidney functions, coagulation

profile, stool & urine analyses, ESR, LDH & ANA results were normal. Bone marrow biopsy & cytogenetics were normal. Food & drug allergy skin test was normal. Diagnostic paracentesis showed turbid ascitic fluid with protein level 5.2 g/dl, serum ascites-albumin gradient (SAAG): 0.6, TLC: 2710/dl with marked eosinophilia (75%). The ascitic glucose level, LDH and adenosine deaminase (ADA) were all normal. Ascitic fluid bacteriological culture, cytology for malignant cells & PCR for tuberculosis came also normal. Tumor markers (carcinoembryonic antigen (CEA) and CA 19-9) were negative. Biopsy results from esophagus showed non-specific inflammation with marked eosinophilic infiltrate. Gastric biopsy revealed chronic inflammatory changes with excessive eosinophilic infiltrates in mucosa & submucosa ( $>35/\text{hpf}$ ). The patient was diagnosed as eosinophilic esophagitis with eosinophilic ascites. He received initially prednisolone 30 mg orally/day along with empirical 6-food elimination diets. The patient showed only partial improvement of his esophageal symptoms and the amount of ascites after two weeks of treatment. The prednisolone dose was increased to 60 mg/day orally with addition of PPI, Kitotifen & clarithromycin for other two weeks with more effective improvement followed by rapid steroid tapering over 6 weeks. Resolution of ascites & pleural effusion occurred after 13 days while resolution of esophageal symptoms occurred after 23 days of the combined treatment. Follow up endoscopic biopsies & ultrasonography along with laboratory results after 6 months came normal. The asthma symptoms also showed dramatic improvement after resolution of the esophageal symptoms. The patient was scheduled for another follow up evaluation after one year.

## Discussion

Eosinophilic gastroenteritis is a rare inflammatory GIT disorder that was first described in 1937 by Kajjer. It affects both sexes mostly between 20 to 50 years of age. Few cases of eosinophilic gastroenteritis were described in asthmatic patients although eosinophils and IgE antibodies are incriminated in the pathogenesis of both conditions. Eosinophilic gastroenteritis is characterized by peripheral eosinophilia with eosinophilic infiltration any part of GIT from esophagus to rectum (>15/hpf) [1,2].

Eosinophils mostly infiltrate mucosa but submucosa, muscosa & serosa may be also involved. The exact aetiology of the disease is unknown although about 50% of eosinophilic gastroenteritis patients have an atopic disorder (allergic rhinitis, eczema or atopic asthma). Food allergy is another proposed mechanism for eosinophilic gastroenteritis especially in children & removal of six food groups (cow's milk, wheat, egg, soy, nuts and fish/seafood) from diet for 6 weeks may be an effective initial treatment for some patients. The most common presenting symptoms are non-specific involving abdominal pain, nausea, vomiting and diarrhea. Food impaction, ascites, GI bleeding & iron-deficiency anemia, pyloric stenosis, malabsorption, intestinal perforation & intestinal obstruction are serious sequelae of the disease [3].

Diagnosis depends on presence of peripheral eosinophilia with proved GI infiltration by eosinophils in biopsy results (>15/hpf) &/or predominant eosinophilic count in ascitic fluid. No radiographic findings are specific or diagnostic. It is also essential to exclude other causes of eosinophilia especially drug allergy (e.g: acetylsalicylic acid, sulfonamides, penicillin, azathioprine & gold salts), food allergy (e.g: cow milk enteropathy), intestinal parasitism, hyper-eosinophilic syndrome, eosinophilic leukemia and Churg-Strauss syndrome (CSS) (vasculitis, severe asthma, allergic rhinitis and tissue eosinophilia) [4].

Treatment should include avoidance of allergens particularly in atopic patients. High-dose corticosteroid therapy, usually for a short duration of time with rapid tapering, is the mainstay of treatment with a dramatic response even in ascitic patients & a rate of remission exceeding 90%. Relapse rate is up to 50% [5].

We presented a known asthmatic patient that had esophageal symptoms & unexplained ascites for months. His esophageal manifestations were initially misdiagnosed & treated as reflux esophagitis. The development of food impaction & unexplained ascites in this asthmatic patient led to the clinical consideration

of eosinophilic gastroenteritis that was confirmed by presence of peripheral eosinophilia in CBC, predominant eosinophils with SAAG <1.1 in diagnostic paracentesis & high eosinophilic infiltrate (>15/hpf) in esophageal & gastric biopsy results after upper GI endoscopy. Our patient showed only partial initial response to high-dose steroid therapy. So, we increased steroid dose & added PPI, clarithromycin & the oral leukotriene inhibitor Kitotifen with a better & long-lasting resolution of clinical (esophageal symptoms & ascites) and histological features.

PPI with empirical 6 food-elimination diet is considered now as the first line therapy for symptomatic & histological remission of eosinophilic esophagitis. Local steroids as budesonide are also effective in inducing clinical & histological remission [5]. Macrolides impair T-lymphocytes proliferation & induce eosinophilic apoptosis. So, they have both antimicrobial & immunomodulatory effects. Additionally, macrolides have a steroid-sparing effect through their influence on steroid metabolism [6].

Mast cell stabilizers (as Na Cromoglycate) & leukotriene inhibitors (as Montelukast & Kitotifen) can be taken orally & act significantly locally in GIT to inhibit inflammatory mediators involved in eosinophilic gastroenteritis. They are also effective steroid-sparing agents. Due to the high recurrence rate, multiple occasions of follow up (every 3-6 months) are required to evaluate the prognosis of the condition [7].

## Conclusion

Eosinophilic gastroenteritis is an uncommon GI disorder of unknown etiology. It is associated with peripheral eosinophilia, high eosinophilic infiltration of GIT with GI dysfunction. When bronchial asthma is co-exist with peripheral eosinophilia & GI symptoms, eosinophilic gastroenteritis is one of the important differential diagnoses that should be suspected. Confirmation of diagnosis mandates presence of high eosinophilic infiltration of GIT particularly in the mucosa & submucosa. Eosinophilic ascites is a common sequelae of the condition & confirmed by high eosinophilic count in ascitic fluid aspirate with SAAG <1.1. Exclusion of other causes of peripheral eosinophilia (food & drug allergy, parasitism, CSS, leukemia etc) is also required before establishing the diagnosis of eosinophilic gastroenteritis. Esophageal involvement responds to PPI, local steroids & food restriction. However, more extensive GI involvement requires systemic steroids. Addition of oral leukotriene inhibitors & macrolides to the previous lines of treatment is effective in resolution of clinical features & histological inflammation.

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