Rare disease

Eosinophilic gastroenteritis – a diagnostic enigma

Urszula Simoniuk,¹ Chris Mcmanus,² Clement Kiire³¹Surgery Department, Southport General Hospital, Southport, UK;²Respiratory Medicine Department, Southport General Hospital, Southport, UK;³Gastroenterology Department, Southport General Hospital, Southport, UK**Correspondence to** Dr Urszula Simoniuk, ula.daria@gmail.com**Summary**

Eosinophilic gastroenteritis is a heterogeneous and curable disease of the gastrointestinal tract. The diagnosis is rare, with an approximate incidence of 1/100 000. The pathophysiology is based on infiltration of the eosinophils involving various parts of gastrointestinal system, but also different layers of the wall. The authors present a case of the eosinophilic gastroenteritis, which is characterised by the predominant subserosal type associated with asthma, ascites and bilateral pleural effusion. In patients with asthma and abdominal symptomatology, eosinophilic gastroenteritis should be considered as a possible diagnosis. These two atopic conditions may occur more frequently than generally expected, especially in the presence of protein-losing gastroenteropathy.

- ▶ Consider a diagnosis of eosinophilic gastroenteritis in patients with atopy and gastrointestinal symptoms.
- ▶ Histological demonstration of the gastrointestinal tract and/or ascites.
- ▶ Exclude other causes of eosinophilia such as parasites, Churg-Strauss syndrome, hypereosinophilic syndrome.

BACKGROUND

Eosinophilic gastroenteritis is a rare condition of undetermined aetiology and the incidence is difficult to estimate. The aetiology is uncertain; however this disease is well described in the current literature, and responds well to steroid therapy.^{1–4} This illustrative case report will assist clinicians in the differential diagnosis of patients presenting with eosinophilia and gastrointestinal symptoms.

CASE PRESENTATION

In April 2011, a 63-year-old man was admitted to hospital presenting with increasing shortness of breath, wheeze, productive cough and peripheral oedema for 5 months. He had a medical history of bronchial asthma and hay fever. There was no history of any allergies to medications or food. He was a retired plasterer, had kept pigeons for last 50 years and was ex-smoker of 30 years.

Over the last 3 months lost two stones of weight, was anorexic and had chronic watery diarrhoea. In addition, his asthma had significantly deteriorated.

The physical examination revealed severe peripheral oedema, moderate ascites and bilateral pleura effusions. Cardiac examination was normal. Central nervous system examination showed no evidence of neuropathy. The rest of his physical examination was unremarkable.

INVESTIGATIONS

His blood test revealed haemoglobin-12.8, white cell count-6.4, high eosinophil count 2.4, low albumin of 22, erythrocyte sedimentation rate-54 and C reactive protein 22.7. Renal function test including urine analysis was normal. The other liver function tests apart from albumin were normal. The tests for rheumatoid factor, antinuclear antibodies, antineutrophil cytoplasmatic antibodies, antibodies against transglutaminase, endomysium or gliadin were normal. Serum immunoglobulin (Ig) concentration

including pigeon and budgerigar IgG were normal. Hepatitis A, B, C, cytomegalovirus, Epstein-Barr and HIV virology were negative. Tumour marker and thyroid function test's were normal. *E coli* test was negative. Iron studies showed normal iron capacity and saturation.

Marker for intestinal protein malabsorption in faecal α -antitrypsin was 3.6 (2.4). Stool examination showed no evidence of ova parasites, stool cultures were negative.

Chest x-ray showed bilateral pleural effusion. Ultrasound scan of the abdomen showed moderate ascites. CT scan of the chest, abdomen and pelvis confirmed the ultrasound and chest x-ray findings and revealed no evidence of malignancy.

Diagnostic pleural fluid cytology showed large numbers of acute and chronic inflammatory cells including eosinophils but no malignant cells were found. The ascitic fluid tap showed chronic inflammatory cells but no malignant cells. Both pleural and ascitic fluid cultures showed no growth.

The upper gastrointestinal endoscopy showed gastritis and duodenitis up to the second part of duodenum. Biopsy revealed eosinophilic infiltration of the lamina propria and submucosa with perivascular infiltration but no vascular necrosis. The appearance was compatible with the clinical diagnosis of eosinophilic gastroenteritis (figure 1).

DIFFERENTIAL DIAGNOSIS

The main differential diagnoses include intestinal parasites, eosinophilic granuloma, myeloid malignancies, hypereosinophilic syndrome and Churg-Strauss Syndrome. All these conditions were considered and excluded on the basis of above mentioned investigation. We could not identify any renal or peripheral nerve involvement. In addition, the immunological screen was unremarkable. There are major differences in the

Figure 1 Histopathology specimen – from gastric atrium showed inflammation with eosinophilic infiltration.

treatment of these various conditions and it is important to establish the correct diagnosis.

TREATMENT

The patient was treated with 40 mg daily prednisolone followed by decreasing doses. He is currently on 5 mg of prednisolone with osteoporosis prophylaxis and he remains well.

OUTCOME AND FOLLOW-UP

On follow-up in outpatient clinic 6 weeks later, he had no symptoms and examination did not reveal any ascites, peripheral oedema or bilateral pleural effusion. His blood test including eosinophil count and serum albumin were normal. The patient reported that his symptoms resolved within 2 weeks of starting steroid therapy.

DISCUSSION

Our case involved a patient with eosinophilic gastroenteritis, protein-losing gastroenteropathy, ascites, pleural effusion as well as asthma. This disease maybe much more common than previously recognised.⁵ For example, 42 such patients with eosinophilic gastroenteritis were identified in a Scandinavian study.⁶ Recent research in the USA estimated a higher prevalence of 28/100 000 in 2011.⁷ There is much speculation regarding aetiology but the most common view is that it is caused by an intestinal allergy. Various allergens stimulate a chain of reactions involving the activation of Th2 cells, mast cells and eotaxin.⁸ There is now substantial evidence verifying the role of eotaxin with early eosinophils recruitment.⁹ Peripheral eosinophilia may be seen in up to 80% of cases but is not a precondition for diagnosis.⁸

Eosinophilic infiltration usually involves more than one layer of the gastrointestinal tract. Based on the maximal depth of eosinophilic infiltration, three types of eosinophilic gastroenteritis have been defined.^{10–11} The three types include the mucosal type, muscularis type and serosal, constituting 70%, 20% and 10% of the diagnosis, respectively.^{12–13} The clinical features depend on the layers and location affected^{10–14–15} as well as the presence of atopic disease. This results in protean manifestation and

symptoms which vary from case to case. The majority of patients with eosinophilic gastroenteritis respond very well to steroid therapy (prednisolone) but this response is not universal.⁶ The important feature in this case is the simultaneous involvement of the serosal (characterised by ascites, bilateral pleural effusion) and mucosal layer (hypoalbuminemia, weight loss, anorexia and clinical symptoms of protein-losing gastroenteropathy). This type of presentation is a result of the involvement of both the mucosal and serosal layer.

According to Talley,¹¹ there are three diagnostic criteria of eosinophilic gastroenteritis: gastrointestinal symptoms, histological demonstration of eosinophilic infiltration in one or more areas of gastrointestinal tract or high eosinophil count in ascitic fluid and no evidence of parasitic or extra intestinal disease. In some cases, eosinophilic gastroenteritis might be an exclusion diagnosis. Histopathology results, clinical features and the exclusion process combine to enable a conclusion of eosinophilic gastroenteritis. Our patient met these criteria. It should be highlighted that a diagnosis of eosinophilic gastroenteritis can be difficult to make and prolonged. The purpose of this case is to alert doctors about this condition so that it can be included in their differential diagnosis of patients presenting in this manner.

Learning points

- Eosinophilic gastroenteritis might be more common than previously believed.
- In patients with unexplained peripheral blood eosinophilia, ascites and pleural effusion consider eosinophilic gastroenteritis.
- Higher index of suspicion of eosinophilic gastroenteritis is required to diagnose correctly.

Competing interests None.

Patient consent Obtained.

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