

A rare cause of abdominal pain: eosinophilic gastroenteritis

N.A.F. Verheijden*, S.A.M. Ennecker-Jans

Amphia Hospital, 4818CK Breda, *corresponding author: tel.: +31 (0)76-595 15 55, +31 (0)76-595 11 24,
e-mail: NVerheijden@amphia.nl

ABSTRACT

Eosinophilic gastroenteritis is a disease that is characterised by an eosinophil-driven inflammation of the digestive tract, presenting with non-specific symptoms, including abdominal pain, nausea, and diarrhoea. The diagnosis is established by histopathological analysis revealing eosinophilic infiltration of the lamina propria. The disease is relatively rare but a proper diagnosis is important, since specific treatment may limit the disease severity and progression.

KEYWORDS

Eosinophilic gastroenteritis, abdominal pain, eosinophilic infiltration

INTRODUCTION

Eosinophilic gastroenteritis (EG) is a rare condition, first described in 1937 by Kaijser *et al.* It is defined as a disorder primarily affecting the gastrointestinal tract with eosinophil-rich inflammation, in the absence of known causes of eosinophilia (e.g. drug reactions, parasitic infections or malignancy).¹ Three different forms of EG can be distinguished: mucosal disease, muscle layer disease and subserosal disease.

The symptoms of EG are related to the layer involved. Mucosal disease is the most common form and presents with nonspecific symptoms such as abdominal pain, nausea, vomiting, diarrhoea or malabsorption. The second form, muscle layer disease, is a more serious form that presents with symptoms due to intestinal obstruction. The third form, subserosal disease, is uncommon and presents with ascites. Incidence in the USA is approximately 2.5 per 100,000 adults.^{2,3} EG has been diagnosed with increasing frequency in recent years.⁴⁻⁶ This is most likely due to increased awareness. Nevertheless, we underline the importance of recognising EG, since proper treatment can prevent further

What was known on this topic?

Eosinophilic gastroenteritis is a blood disorder, which damages the mucosa of the gastrointestinal tract. Symptoms are related to the layer of bowel mucosa involved. Peripheral eosinophilic counts and serum IgE should be performed in patients with nonspecific abdominal complaints. When elevated, endoscopic investigation of the upper gastrointestinal tract should be performed and biopsies of both abnormal and normal mucosa should be taken.

What does this add?

Symptoms of EG may mimic irritable bowel disease. Proper treatment can prevent further mucosal damage and progression to severe malabsorption and malnutrition. Therefore, it is important to recognise EG. Several forms of treatment are described, including diet restriction and treatment with oral corticosteroids. There have been no prospective, randomised therapeutic clinical trials. Thus, treatment is empiric and based upon the severity of clinical symptoms.

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CASE REPORT

A 61-year-old man, with a history of myocardial infarction, atrial fibrillation, and asthmatic rhinitis, presented at the Emergency Room with diffuse abdominal pain. He did not complain of nausea, vomiting or diarrhoea. The pain started four days ago. His stools did not change and he did not have fever. A year ago he had an episode with the same symptoms. Gastroscopic investigation did not show any abnormalities at that time. It was performed,

however, a few weeks after spontaneous disappearance of his complaints.

Laboratory investigations showed elevated peripheral eosinophil counts and an elevated serum IgE (table 1). Further imaging did not show any pathology, especially no signs of ischaemia. Faecal examination was negative. The patient was admitted. Since the abdominal pain persisted, we performed gastroscopic investigation. This showed polypoid gastric mucosa with erythema. Furthermore erosions of both gastric and duodenal mucosa were seen (figure 1).

Biopsies were taken from the abnormal appearing mucosa. These biopsies revealed distinct eosinophilic infiltration (figure 2). Therefore the diagnosis of eosinophilic gastroenteritis was confirmed.

Because of the serious symptoms we immediately started treatment with prednisone 20 mg for two weeks, before performing skin prick tests. There is some evidence that avoidance of food allergens can improve disease activity. The symptoms declined rapidly after starting treatment. Moreover, there was a remarkable decline in the peripheral eosinophil counts. Prednisone was tapered during six weeks. Skin prick tests were performed afterwards, and were negative.

Table 1. Laboratory findings in our patient suffering from eosinophilic gastroenteritis

Laboratory findings	Normal	At diagnosis	6 weeks after treatment with Prednisone
Peripheral eosinophil counts	0.5	18.04	0.38
Serum IgE	<100	2238	507

Figure 1. Macroscopic picture of the affected duodenal mucosa, showing erosions and duodenal folds

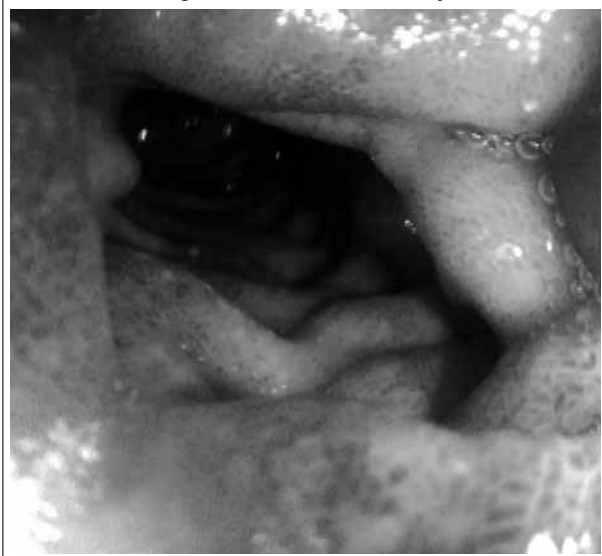
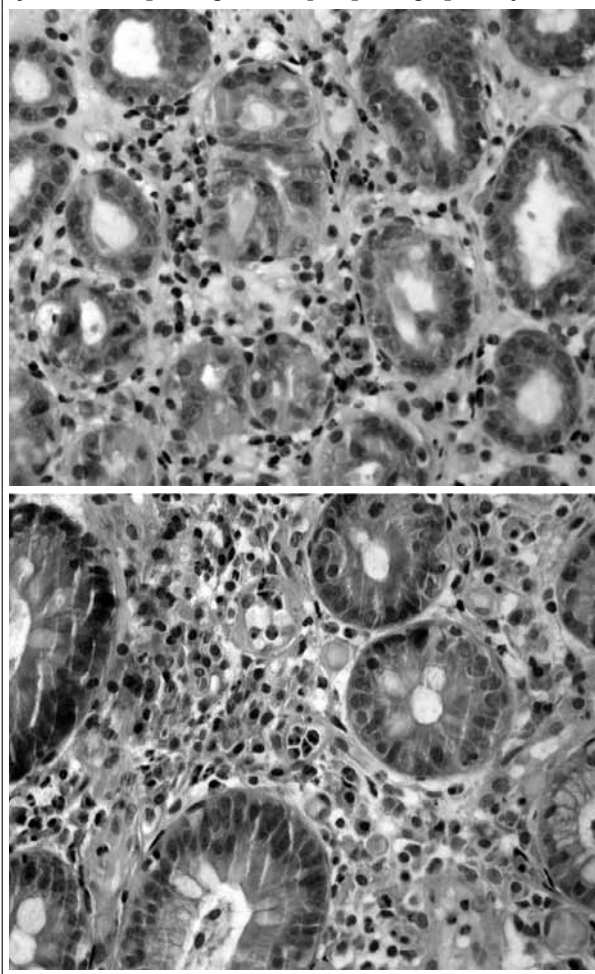


Figure 2. Biopsies of gastric mucosa showing prominent eosinophilic infiltration of the lamina propria, consisting of >20 eosinophilic granulocytes per high power field



DISCUSSION

Diagnosis

Since EG presents with nonspecific symptoms, it is not easy to diagnose and may be confused with irritable bowel disease. Because treatment can prevent further mucosal damage, it is important to recognise EG. Our patient had suffered the same symptoms previously, but investigations at that time did not reveal a diagnosis. History and laboratory evaluation of our patient showed several features of EG. For example, the patient had a history of asthmatic rhinitis and a previous episode of abdominal symptoms. Besides, peripheral eosinophil counts were elevated. The sensitivity of this test is about 80%. In one study 23% of patients lacked peripheral eosinophilia. Furthermore, serum IgE levels were elevated.

Patients with EG may suffer from malabsorption with hypoalbuminaemia or anaemia. Up to 50% of patients had a history of food allergy or intolerance.^{7,8} To confirm

diagnosis, a biopsy of gastric or duodenal mucosa is necessary. Endoscopic appearance in eosinophilic gastroenteritis is nonspecific and includes mucosal folds, hyperaemia or ulceration. Because of mucosal sparing, it is recommended to obtain at least six biopsy specimens from both normal and abnormal areas of the bowel. Histopathology will reveal eosinophilic infiltration of the lamina propria. Diagnostic criteria vary from >20 to >50 eosinophils per high power field.^{8,9}

Treatment

The literature shows scarce data on the proper treatment in EG. According to different studies, the first step in treating EG is diet restriction.¹⁰ It is recommended to perform skin prick testing to identify any food allergies. If present, patients should start a restricted diet. Referral to a dietician will make this approach more successful.

Although there is an evident association between food allergy and EG, results of elimination diets are often poor. When improvement of symptoms with diet restriction is poor or not feasible, the following step in treatment of EG is the use of steroids. An effective relief of symptoms usually occurs within two weeks. Several studies report good results with steroids in dosages from 20 to 40 mg/day, for six to eight weeks.¹¹⁻¹⁴

Successful treatment with budesonide has been described in certain studies as well. It inhibits both eosinophilic activation and survival.^{11,14-16} The main advantage of budesonide is its high metabolism, and therefore a lower risk of side effects such as adrenal suppression.¹⁴ Other less common drugs have also been found to be effective in the treatment of EG. These therapies focus at the assumed allergy component in the pathogenesis of EG. Cromolyn prevents the release of mast cell mediators and eosinophil mediator release.¹⁷ A positive response with oral cromolyn was seen in some^{18,19} but not all case reports.⁸ There was a variation in results with Montelukast, an antagonist of the leukotriene receptor Cys-LT₁. Activation of this receptor results in contraction of smooth muscle, oedema and eosinophil migration. Montelukast was effective and steroid sparing in some reported cases²⁰⁻²² but less successful in others.²³ Recently a review in this journal described promising effects of several new therapies, including imatinib, in hypereosinophilic syndromes.²⁴ A single case report showed a rapid decline of symptoms with imatinib in a patient suffering from chronic eosinophilic leukaemia with gastrointestinal involvement.²⁵ So several forms of treatment are described. There have been no prospective, randomised therapeutic clinical trials. Thus, treatment is empiric and based upon the severity of clinical symptoms. The subsequent course is variable. Patients may experience periodic flares months to years after the first episode.

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