

Abdominal Pain Relieved By A Warm Hot Water Bottle: An Atypical Presentation Of Eosinophilic Gastroenteritis

Ahmad R Shirazi-Nejad, John M Hebden

Department of Gastroenterology, Sheffield Teaching Hospitals NHS Foundation Trust, Sheffield, UK

Doi: 10.12890/2020_001687 - European Journal of Case Reports in Internal Medicine - © EFIM 2020

Received: 24/04/2020

Accepted: 29/04/2020

Published: 12/05/2020

How to cite this article: Shirazi-Nejad AR, Hebden JM. Abdominal pain relieved by a warm hot water bottle: an atypical presentation of eosinophilic gastroenteritis. *EJCRIM* 2020;7: doi:10.12890/2020_001687.

Conflicts of Interests: The Authors declare that there are no competing interests.

This article is licensed under a [Commons Attribution Non-Commercial 4.0 License](https://creativecommons.org/licenses/by-nc/4.0/)

ABSTRACT

A 26-year-old woman presented with a 3-month history of worsening episodic abdominal pain, which was associated with frequent passage of watery stools, nausea and dyspepsia. Her peripheral eosinophil count was markedly elevated. This responded well to a reducing regimen of corticosteroids. Her symptoms completely resolved with a corresponding fall in eosinophil count. The patient was diagnosed with eosinophilic gastroenteritis. We have not considered steroid-sparing agents at this point, but should she have future exacerbations then this will be considered.

LEARNING POINTS

- Keep eosinophilic gastroenteritis in mind when reviewing patients with atypical gastrointestinal symptoms and elevated peripheral eosinophil counts, particularly in patients with a history of atopy.
- The clinical history, histology and cross-sectional imaging is complementary in securing a diagnosis.
- Follow-up imaging and endoscopic evaluation can be useful in monitoring response to treatment.

KEYWORDS

Eosinophilic gastroenteritis, diarrhoea, pain

CASE DESCRIPTION

A 26-year-old woman was referred by her general practitioner (GP) to the gastroenterology outpatient clinic. She had been fit and well until the onset of symptoms and her only regular medication was the combined oral contraceptive pill. She had not had any previous abdominal surgery, and her obstetric history included one previous pregnancy with uncomplicated vaginal delivery 7 years previously. Furthermore, she did not have a history of asthma/eczema/hay fever/atopy. Her family history was unremarkable. She does not smoke and rarely drinks alcohol.

Her symptoms included intractable dull abdominal discomfort with intermittent spasms of more intense pain, lasting for 1–2 minutes. For the pain she had been using over-the-counter analgesia to little avail but found some relief from holding a hot-water bottle against her abdomen. This was associated with an abrupt onset of change in bowel habit, from chronic constipation to passing watery stools up to 5 times per day. She had not experienced any tenesmus, urgency, incomplete emptying, dyschezia or nocturnal symptoms. She also described feeling nauseated but had not vomited. She had frequent episodes of dyspepsia, which was largely relieved by twice-daily PPI, initiated by her GP. There was no objective evidence of any weight loss, but she had a reduced appetite and had been only able to tolerate smaller portion sizes over the past few months.

On examination, she did not display any peripheral stigmata of anaemia or chronic disease. She did not have any rashes, joint effusions or ocular signs. Abdominal examination revealed striking erythema ab igne, but no overlying skin blistering or desquamation. She had left iliac fossa tenderness with a degree of voluntary guarding, but no other features of peritonism. There was no clinically palpable hepatosplenomegaly or supraclavicular lymphadenopathy, and bowel sounds were normal on auscultation.

Full blood count (FBC) performed before clinic in early June by her GP revealed: haemoglobin (Hb) 140, total white cell count (WCC) 19.4, eosinophils 12.94, faecal calprotectin (FCP) 83, erythrocyte sedimentation rate (ESR) 2, C-reactive protein (CRP) 5.4, adjusted calcium 2.25, and albumin 48, while IgA endomysium antibodies (EMA) was negative. On the day of her clinic appointment in late June, further investigations were arranged and these revealed: Hb 149, WCC 15.8, eosinophils 8.16, renal and liver profile all within normal limits, rheumatoid factor (RhF) negative, ESR 2, CRP 4.5, complement C3 1.19, complement C4 0.18, c-ANCA/p-ANCA both negative, ANA negative, and immunoglobulin screen showing IgA slightly low at 0.71, and IgG and IgM both normal, while serum electrophoresis showed no specific electrophoretic abnormalities.

CT of the abdomen/pelvis was carried out the day after clinic and showed thickened segments of the distal ileum including the terminal ileum, with no small bowel dilatation. Moderate volume ascites was also detected.

Stool microbiology including pathogenic ova/cysts/parasites, rotavirus antigen, *Clostridium difficile* toxigenic antigen, *Cryptosporidium* oocysts, *Campylobacter* culture, *Salmonella*, *Shigella* and *E. coli* O157 was all negative.

Gastroscopy performed in early July was macroscopically normal to the second part of the duodenum (D2). Unfortunately, only one duodenal biopsy was taken due to poor tolerability of the procedure, but the histology was essentially normal, without excess infiltration of eosinophils.

We did not perform a bone marrow biopsy or screen for PDGFRA, BCR-ABL and JAK2 mutations.

The first step in the management of this patient was to exclude parasitic/enteric infection and obtain relevant information to rule out hyper-eosinophilic syndrome or haematological causes of raised peripheral eosinophilia. Usually, eosinophilic gastroenteritis is diagnosed by confirmatory histopathology.

After excluding infection, we decided to cautiously initiate a reducing regime of prednisolone 30 mg once daily for 2 weeks to be reduced by 5 mg weekly thereafter. This was started before endoscopic evaluation was undertaken due to the severity of the patient's symptoms and the low clinical suspicion of other pathology.

The patient was reviewed on a fortnightly basis. Unfortunately, the patient was unable to have sedation for her gastroscopy and the procedure was abandoned due to poor tolerance. The colonoscopy was cancelled for similar reasons. Abdominal ultrasound scan (USS) 3 months after the initial CT revealed complete resolution of the ascites and a normal appearance of the distal ileum. Over a 2-month period, the peripheral eosinophil count fell from 12.94 to 0.01. The patient was appropriately followed up.

DISCUSSION

Kaijser first described eosinophilic gastroenteritis in 1937 as a rare disease characterised by infiltration of eosinophils into the intestinal mucosa^[1]. The literature on this subjects is mainly comprised of case reports and small observational studies regarding diagnostic criteria and treatment strategies, but other data on the disease process remain scarce, probably due to its rarity. Eosinophilic gastroenteritis appears to result from a complex interplay between environmental exposure to antigens and specific genetic susceptibility, thought to be mediated by a Th2-related allergy response^[2]. In the related condition of eosinophilic oesophagitis, there appear to be an elevated number of eosinophils found histologically in the oesophageal mucosa. Furthermore, the frequency seems to be elevated in those with atopic conditions and asthma. Traditionally, eosinophils have been thought to be recruited in response to certain invading pathogens, such as parasites. However, in eosinophilic disorders of the gastrointestinal tract, the recruitment and activation of eosinophils can occur even in the absence of an identifiable pathogen. Eosinophil activation leads to release of cytokines, interleukin-5 (IL5) and eotaxins, which ultimately results in the production of cytotoxic chemicals causing mucosal inflammation and damage^[3,4]. Various different treatments have been tried for such disorders, including dietary modification, acid suppressants and immunosuppressive drugs, including steroids and steroid-sparing agents. Further studies are needed to effectively define the mechanistic pathogenesis of this disorder and which treatment strategies should be employed^[5]. Steroids appear to be the mainstay of treatment, but relapse is common upon treatment tapering or withdrawal. In the paediatric population, exclusion diets have been used, but novel treatments with monoclonal antibodies targeting specific receptor sites have been used. Alternatives include prolonged courses of macrolide antibiotics, but large studies have not yet elucidated the optimum therapeutic choice, particularly in relapsing or refractory disease.

REFERENCES

1. Kaijser R. Zur Kenntnis der allergischen Affektionen des Verdauungskanals vom Standpunkt des Chirurgen aus. *Arch Klin Chir* 1937;**188**:36–64.
2. Powell N, Walker MM, Talley NJ. Gastrointestinal eosinophils in health, disease and functional disorders. *Nat Rev Gastroenterol Hepatol* 2010;**7**(3):146–156.
3. Rothenberg ME. Eosinophilic gastrointestinal disorders (EGID). *J Allergy Clin Immunol* 2004;**113**(1):11–28; quiz 9.
4. Sato H, Honma T, Owaki T, Tominaga K, Yokoyama J, Terai S. Clinical and pathological profile of eosinophilic gastroenteritis. *Eur J Gastroenterol Hepatol* 2019;**31**(2):157–162.
5. Zhang M, Li Y. Eosinophilic gastroenteritis: a state-of-the-art review. *J Gastroenterol Hepatol* 2017;**32**(1):64–72.