



Available online at www.sciencedirect.com

SciVerse ScienceDirect

journal homepage: www.elsevier.com/locate/itjm



CASE REPORT

Eosinophilic ascites: a case report

Un caso clinico di ascite eosinofila

Paolo Pozzato ^{a,*}, Susanna Vicari ^a, Angela Salerno ^b, Alessandra Cancellieri ^b,
Addolorata Gallone ^a, Clarissa Belloli ^a, Nicola Vianelli ^c, Maurizio Ventrucci ^a

^a Department of Internal Medicine, Bentivoglio Hospital, Italy

^b Department of Histopathology and Cytopathology, Maggiore Hospital, Bologna, Italy

^c Seragnoli Institute of Hematology and Medical Oncology, University of Bologna, Italy

Received 29 August 2012; accepted 21 September 2012

Available online 9 November 2012

KEYWORDS

Eosinophilic
gastroenteritis;
Eosinophilia;
Ascites.

Summary

Introduction: Eosinophilic gastroenteritis is a heterogeneous disorder affecting both children and adults, and is characterised by the presence of an intense eosinophilic infiltrate on histopathology of one or multiple segments from the oesophagus to the rectum. Involvement of the serosal layer occurs in 10% of cases of eosinophilic gastroenteritis and typically presents as ascites.

Case report: We report the case of a 25-year-old woman who was admitted to our Hospital complaining of abdominal pain and distension, ascites and diarrhoea of four weeks duration. Laboratory investigations showed a white cell count of 9.2 thousand/mm³ without eosinophilia, low albumin (3 g/dL) and an increased IgE level (279 U/mL). Abdominal ultrasonography demonstrated peritoneal effusion, and cytological analysis revealed a prevalence of eosinophils in the ascites. Upper and lower multiple endoscopic biopsies revealed an increased amount of mucosal eosinophils. The patient was treated with prednisolone 40 mg/day for two weeks with rapid resolution of her symptoms and the ascites.

Discussion: Patients with serosal eosinophilic gastroenteritis present with isolated ascites or ascites in combination with symptoms characteristic of mucosal or muscular involvement, such as abdominal pain, nausea, vomiting, diarrhoea, and weight loss. The diagnostic feature is marked eosinophilia in the ascitic fluid. Data on the natural history and therapy are limited to case reports. There have been no prospective, randomized therapeutic clinical trials. Thus, treatment was empiric and based upon the severity of the clinical manifestations. Patients who are symptomatic or have evidence of malabsorption may be treated with systemic glucocorticoids.
© 2012 Elsevier Srl. All rights reserved.

* Corresponding author: Department of Internal Medicine, Bentivoglio Hospital, via Marconi 35, 40010 Bentivoglio (BO), Italy.
E-mail: paolopozzato@hotmail.com (P. Pozzato).

Introduction

Eosinophilic gastroenteritis (EG) is a rare disorder characterised by gastrointestinal symptoms, blood eosinophilia, and eosinophilic infiltration of the gastrointestinal wall [1]. The disease can affect patients of any age, but typical presentations are in the third through fifth decades of life with a male predominance [2]. Although allergies to food or milk contribute to this disease in some children, allergic etiologies are not common in adults.

Despite its rarity, EG needs to be recognised by the clinician because it can masquerade as irritable bowel syndrome. The diagnosis of EG is confirmed by a characteristic biopsy and/or eosinophilic ascitic fluid in the absence of infection by intestinal parasites or other causes of intestinal eosinophilia.

The clinical features of EG are related to the layer and extent of the bowel involved with eosinophilic infiltration: mucosa, muscle and/or serosa [3]. Mucosal involvement alone may result in abdominal pain, nausea, vomiting, diarrhoea, weight loss, anaemia, protein-losing enteropathy, and intestinal perforation. Patients with muscular layer involvement have symptoms of pyloric or intestinal obstruction and early satiety while serosal eosinophilic infiltration may result in the development of eosinophilic ascites.

Case report

A previously healthy 25-year-old woman was admitted to our Hospital complaining of abdominal pain and distension, and diarrhoea of several weeks duration. There was no history of transfusions, recent travel, respiratory symptoms or rash. The patient was used to eating raw fish at Japanese restaurants. She did not consume alcohol or any illicit drug, and was taking neither medications nor supplements. On physical examination, the abdomen was distended and there was a diffuse tenderness; no rebound or guarding were observed. No lymphadenopathy or hepatosplenomegaly were found.

The results of the laboratory investigations were as follows: Hgb 12.8 g/dL, WBC 24.12 k/mL, differential: segmento-nuclear neutrophils 16.3%, lymphocytes 11.6%, monocytes 2.9%, eosinophils 67.7%, low albumin (3 g/dL) and increased IgE level (405 U/mL). VES, protein C reactive, serum electrolytes, coagulation studies, thyroid and liver tests were normal. Coeliac disease antibody tests were negative, as were HIV and viral hepatitis B and C markers. Parasitic infestation was excluded by negative stool studies and serology for *Strongyloides* and *Toxocara*. Tests for anti-nuclear factor, rheumatoid factor and antineutrophilic cytoplasmic autoantibody were all negative. Ca125 was 102 ng/mL, while other neoplastic markers were negative. Allergen-specific IgE in serum (RAST) showed positivity for *Pleum pratense* (3.04 kUa/L) and cat hair (3.88 kUa/L).

Abdominal ultrasonography (US) and abdominal computer tomography did not demonstrate any alteration of internal organs, but only peritoneal effusion which was withdrawn during the US study. Cytological analysis revealed a prevalence of eosinophils in the ascites, up to 80%. Bacterial culture and tests for tuberculosis were negative.

Positron emission tomography (PET) was not performed to minimise exposure to ionising radiation in our young fertile patient.

A bone marrow biopsy showed normal haematopoiesis with hypereosinophilia and normal cytogenetics. A gastroscopy showed oedema of the gastric antrum and oedema of the duodenal mucosa with erythema whereas a colonoscopy was normal. Upper and lower multiple endoscopic biopsies revealed an increased amount of mucosal eosinophils (> 15 per high power field). The patient was treated with prednisolone 40 mg/day with rapid resolution of the ascites in about two weeks. Prednisolone was therefore tapered off with a transition to budesonide 9 mg/day, which that was progressively reduced.

After four months, the patient is still asymptomatic and another abdominal ultrasonography demonstrated resolution of the ascites.

Discussion

Eosinophilic gastroenteritis represents one member of a family of diseases which includes eosinophilic esophagitis, gastritis, enteritis and colitis, collectively referred to as eosinophilic gastrointestinal disorders [1]. These eosinophilic infiltrates may not only involve various sites along the length of the gastrointestinal tract, but may also occupy various sites across the depth of the wall. Physicians rarely make the diagnosis of EG. Although the incidence is not high, the diagnosis is also more difficult because of the inaccessibility of much of the length of the small bowel and of the deeper layers of the luminal wall. The prevalence of each subtype is unknown due to biases in reporting and referrals. Surgical series report a predominance of muscular disease with obstruction while medical series primarily describe patients with mucosal involvement [3].

Patients with mucosal inflammation usually present with common, albeit non-specific, complaints of abdominal pain, nausea, vomiting, diarrhoea, anaemia, or protein losing enteropathy. Due to the non-specific nature, these clinical presentations may be confused with irritable bowel syndrome, dyspepsia, peptic ulcer disease or inflammatory bowel diseases. Frequently, atopy and high IgE levels coexist.

Involvement of the serosal layer occurs in 10% of cases. Patients with serosal EG present with isolated ascites or ascites in combination with symptoms characteristic of mucosal or muscular EG. The serosal form of EG, as compared to other types, is reported to be associated with significant bloating, and a higher level of peripheral eosinophilia [4,5]. Eosinophilic pleural effusion may also be present [6].

Evaluation of patients suspected of having EG is undertaken to exclude differential diagnostic possibilities, establish a definitive diagnosis and assess the potential complications associated with the diagnosis. When EG is suspected on the basis of clinical presentation or the results of a tissue biopsy, other causes of hypereosinophilia, such as drug reaction, malignancy, parasites, infection or systemic disease should be excluded first.

In our case, the diagnosis of eosinophilic gastroenteritis with involvement of the serosal layer was made on the basis of clinical presentation, hypereosinophilia in the ascitic fluid and an increased amount of mucosal eosinophils in

endoscopic biopsies. The rapid clinical remission obtained with systemic steroids subsequently confirmed the diagnosis, avoiding additional investigation, such as CT-PET, potentially harmful for a young patient.

The presence of total IgE elevation in serum as in the case herein reported should alert the physician to an IgE mediated phenomenon, such as the consumption of the raw fish.

Conflict of interest

The authors have no conflicts of interest to disclose.

Acknowledgments

The authors would like to thank Gerald Goldsmith for his revision of the English language.

References

- [1] Rothenberg ME. Eosinophilic gastrointestinal disorders (EGID). *J Allergy Clin Immunol* 2004;113(1):11–28.
- [2] Chang JY, Choung RS, Lee RM, Locke 3rd GR, Schleck CD, Zinsmeister AR, et al. A shift in the clinical spectrum of eosinophilic gastroenteritis toward the mucosal disease type. *Clin Gastroenterol Hepatol* 2010;8(8):669–75.
- [3] Talley NJ, Shorter RG, Phillips SF, Zinsmeister AR. Eosinophilic gastroenteritis: a clinicopathological study of patients with disease of the mucosa, muscle layer, and subserosal tissues. *Gut* 1990;31(1):54–8.
- [4] Kuri K, Lee M. Eosinophilic gastroenteritis manifesting with ascites. *South Med J* 1994;87(9):956–7.
- [5] Zhou HB, Chen JM, Du Q. Eosinophilic gastroenteritis with ascites and hepatic dysfunction. *World J Gastroenterol* 2007;13(8):1303–5.
- [6] Khan S. Eosinophilic gastroenteritis. *Best Pract Res Clin Gastroenterol* 2005;19(2):177–98.