


Eosinophilic Gastroenteritis in Tropical Area: about 6 Cases from Senegal

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Abstract

The aetiologies of hypereosinophilia are dominated by digestive parasitosis and drug intake. In the tropics, because of the frequency of parasitosis, eosinophilic digestive pathologies of primary origin, which are rare, may be overlooked. We report 6 cases of eosinophilic gastroenteritis with polymorphic digestive manifestations testifying to the different locations of eosinophilic infiltration in the digestive parietal layer. Three patients had ascites rich in eosinophils, indicative of serous involvement, while the other two had a muscular form, and the last a mucosal involvement. The evolution was favourable in all cases with corticosteroid therapy, but a recurrence was noted in 4 cases when treatment was stopped.

Keywords

Eosinophilic Gastroenteritis, Ascites, Digestive Eosinophilic Infiltration, Senegal

1. Introduction

Eosinophilic gastroenteritis (EGE) is a rare pathology characterized by an inappropriate infiltration of one or more components of the digestive wall by polymorphonuclear eosinophils. Its etiology is poorly known even if a food allergy is incriminated. It is probably underdiagnosed because of its nonspecific manifestations, the eosinophilic infiltration being able to interest the mucosa, the muscular or the serosa of the digestive wall. EGE is rarely reported in sub-Saharan Africa where parasitosis remains the first cause of hypereosinophilia. We report 6 cases of eosinophilic gastroenteritis reflecting these different impairments.

2. Patients and Methods

This is a retrospective study, including patients hospitalized in the internal medicine and gastroenterology departments of the Principal Hospital of Dakar, between January 1, 2015 and December 31, 2021. We consulted hospitalization registers of the two departments during this period, and we included all the patients with a complete data, and discharged from hospital with the diagnosis of EGE. The diagnosis of EGE was retained after excluding other causes of tissue hypereosinophilia, when there was:

- clinical signs of gastroenteritis (vomiting, diarrhoea, abdominal pain) associated with endoscopic digestive parietal lesions with mucosal eosinophilic infiltration, or blood hypereosinophilia.
- abdominal pain associated with thickening of the colonic wall and blood hypereosinophilia.
- ascites rich in eosinophils.

The data studied were epidemiological (age, sex, terrain, food allergy), clinical (abdominal pain, diarrhea, vomiting, ascites), biological (complete blood count, CRP, liver function tests, parasitological stool examination), endoscopic (upper digestive endoscopy, colonoscopy), histological (biopsies performed during endoscopy), radiological (abdominal scan), therapeutic, and outcome.

Response to treatment was defined by an improvement in clinical signs and disappearance of peripheral eosinophilia. Recurrence was defined by a resumption of symptoms when corticosteroid treatment was reduced or stopped. A patient who remained more than 6 months without attending the follow-up consultation was considered lost to follow-up.

3. Results

During this period, 6 patients were hospitalized with the discharge diagnosis of eosinophilic gastroenteritis. They were 2 men and 4 women with an average age of 39.5 years [21 - 56 years]. None of the patients had a particular medical history, nor atopy or food allergies.

The revealing clinical signs were diarrhoea and vomiting associated with ascites in 2 cases, chronic diarrhoea isolated in 1 case, or associated with abdominal pain in 1 patient, isolated ascites in one patient and an occlusive syndrome without obstruction in one patient. At examination, ascites was found in three patients.

Blood hypereosinophilia was present in 5 patients with an average of 12,640 eosinophils per mm^3 [3530 - 20,470]. Blood eosinophils count was normal in one patient. There was no anemia, nor thrombocytopenia or thrombocytosis. The other biological parameters (hepatic functional explorations, renal function, blood ionogram, calcemia, serum protein electrophoresis and thyroid hormones) were normal in all patients.

There was no inflammatory syndrome. The parasitology stool examination was negative in all cases, as was the retroviral serology. Abdominal paracentesis

in 3 patients brought back a citrine yellow liquid, rich in proteins (respectively 47, 60.5 and 54 g/l) and eosinophils (350, 7520 and more than 7000 eosinophils per mm³, respectively).

Upper digestive endoscopy was normal in 5 cases, and found duodenal ulceration in one case. Colonoscopy showed erythematous, ulcerated or granite rectocolitis in 3 patients, and was normal in 2 cases. All patients had colonic biopsies except one who had a normal colonoscopy. Biopsies were performed both on pathological mucosa and on normal mucosa, and showed an aspect of interstitial or oedematous colitis without eosinophilic infiltrate in 3 patients, and one case of ulcerative colitis of moderate intensity, with a diffuse polymorphic infiltrate, rich in eosinophils (more than 20 per field). Abdominal CT scan showed colonic wall thickening in 3 cases, ascites in 3 patients, and was normal in one case.

The diagnosis of eosinophilic gastroenteritis was retained in all patients, in its serous form in 3 cases, muscular in 2 cases and mucous in 1 case. All patients benefited from systematic deworming with albendazole. Corticosteroid therapy with prednisone 40 mg per day was initiated in all patients, with a favourable evolution marked by a rapid regression of symptoms. For the three patients with ascites, this had disappeared between 7 and 15 days of treatment. In the case of the occlusive syndrome, transit was normal after 48 hours of treatment, with disappearance of abdominal pain and cessation of vomiting. In the other two cases, the diarrhoea had improved after 3 to 5 days, as had the abdominal pain. Blood eosinophilic count was normal after one month except in the patient who had a very high rate, in whom it went from 17140/mm³ to 1420/mm³ in 30 days.

Treatment with corticosteroids was gradually reduced after one month of treatment to reach minimum doses between 7.5 and 5 mg of prednisone per day, without recurrence of symptoms. One patient was lost to follow-up after 6 months without recurrence. In two cases, after one year of follow-up without treatment, there was no resumption of symptoms. In the 3 other patients, the evolution was marked by a relapse after stopping treatment at 1, 4 and 6 months respectively (two patients with a serous form, and one with a muscular form). However, as soon as the corticosteroid therapy was reintroduced, the symptoms improved.

The 6 observations are summarized in **Table 1**.

4. Discussion

Eosinophilic gastroenteritis (EGE) is defined by histological eosinophilic infiltration of more than 20 eosinophils per field of one or more components of the digestive wall, in one or more portions of the digestive tract without extraintestinal involvement, with the exclusion of another cause of gastrointestinal eosinophilia [1]. It is a rare pathology, first described by Kaijser in 1937 [2]. The prevalence is estimated between 5.1 and 8.4/100,000 inhabitants in large population cohorts in the United States, with a slight female predominance, and a more frequent involvement in Caucasians [3] [4]. This prevalence is difficult to assess

Table 1. Characteristics of patients.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6
Age (years)	38	56	21	29	50	43
Sex	Male	Male	Female	Female	Female	Female
Evolution duration before diagnosis	1 month	1 week	Several years, accentuation since one week	3 months	6 years	2 weeks
Clinical presentation	Diarrhea Vomiting Ascites	Diarrhea Vomiting Ascites	Abdominal pain Occlusive syndrome Vomiting, Constipation	Diarrhea Abdominal pain	Chronic diarrhea	Ascites
Blood eosinophils count (/mm ³)	8500	13,560	3530	17,140	180	20,470
Ascites eosinophils count (/mm ³)	7520	+7000	-	-	-	350
Digestive endoscopy	Upper digestive endoscopy: duodenal ulcerations Colonoscopy: erythematous and ulcerated proctitis	Upper digestive endoscopy: normal Colonoscopy: normal	Upper digestive endoscopy: normal Colonoscopy: normal	Upper digestive endoscopy: normal Colonoscopy: erythematous and granitic rectosigmoiditis	Upper digestive endoscopy: normal Colonoscopy: erosive rectocolitis	Normal
Histology	Nonspecific interstitial colitis	Nonspecific edematous colitis	-	Exulcerated sigmoiditis without eosinophilic infiltrate	Ulcerative colitis with a polymorphic infiltrate rich in eosinophils	
Abdominal CT scan	Antro-pyloric wall thickening Ascites	Thickening of the left colonic wall Ascites	Diffuse thickening of the colonic wall	Thickening of the left colon and transverse colon	Normal	Ascites
Eosinophilic gastroenteritis type	Serous	Serous	Muscular	Muscular	Mucosa	Serous
Treatment	Prednisone	Prednisone	Prednisone	Prednisone	Prednisone	Prednisone
Outcome	Favourable without relapse	Favourable, but relapse 6 months after stopping treatment	Favourable, but relapse 4 months after stopping treatment	Favourable, but lost of follow-up after 6 months without relapse	Favourable without relapse	Favourable, but relapse 1 month after stopping treatment

in Africa where the disease is rarely reported in isolated cases [5]. We report 6 cases of EGE followed in our hospital over a period of 5 years, *i.e.* 0.24% of patients hospitalized in the hepato-gastroenterology and internal medicine departments during this period.

EGE is a pathology whose etiopathogenesis is not completely elucidated. However, atopy (rhinitis, eczema, asthma, food allergy) is found in more than half of the cases [6]. This had not been found in our patients, but allergy tests had not been carried out.

Due to the eosinophilic infiltration of the digestive wall that can affect all layers, EGE leads to polymorphic clinical presentations, depending on the parietal involvement. In 1970, Klein proposed an anatomical classification distinguishing three types of EGE according to the parietal layer affected: mucous membrane, muscularis and serous [7], responsible for diarrhoea, abdominal pain, nausea or vomiting, or ascites. In our 6 patients, the clinical symptomatology was various (**Table 1**).

Blood eosinophilia, present in 70% of cases [2] [8], is found in 5 of our patients. It has a strong orientation, but its absence should not prevent the realization of a digestive endoscopy when the diagnosis is suspected [9]. The level of blood eosinophils count also makes it possible to classify the disease as minimal, moderate or severe [2]. On the other hand, the severity of peripheral eosinophilia does not reflect the importance of the infiltration of the digestive layer by eosinophils [10], or the severity of the disease. Among our patients, 5 had very high levels of blood eosinophils count, suggesting a hypereosinophilic syndrome. However there were no symptoms suggestive of involvement of another organ, and the presentation was acute in all cases. Another suggestive biological element in this context is the increase of serum IgE levels above 100 IU/ml which is reported in 2/3 of patients [11]. In front of this symptomatology, it is essential to perform an upper digestive endoscopy, as well as an ileo-colonoscopy with staged biopsies. However, these examinations may be normal or show nonspecific lesions.

In our patients, digestive endoscopy examinations were normal or showed lesions such as erythema or ulcerations. The anatomo-pathological examination of the staged biopsies helps in the diagnosis in the case of mucosal involvement by highlighting more than 20 eosinophils per field. Only one patient presented an eosinophilic infiltrate in the colonic mucosa. In the other patients, the depth of the parietal involvement, as well as the discontinuous distribution of eosinophilic infiltration could explain the absence of eosinophils on the biopsies. On the other hand, the presence of a high number of eosinophils in the ascites fluid confirms the diagnosis of serous involvement, as is the case in our three patients with ascites. On CT scan, thickening of the colonic wall was noted in three patients, probably testifying the infiltration of the muscle layer.

The diagnosis of EGE can be difficult, the clinical presentation not being specific. Talley suggests 3 diagnostic criteria that are currently widely used: presence of gastrointestinal symptoms, histological signs of eosinophilic infiltration of the

digestive tract or an elevated level of eosinophils in the ascites fluid, and the exclusion of other causes of tissue hypereosinophilia (digestive parasites, frequent in tropical area, drugs, neoplasia and essential hypereosinophilic syndrome) [8].

Parasitological stool examinations were done in all our patients and were negative in all cases. However, given the tropical environment, they all benefited from systematic deworming with albendazole and ivermectin prior to treatment. An improvement in clinical symptoms or hypereosinophilia was not noted after this antiparasitic treatment. Thus, among our six patients, four met Talley's criteria, and the diagnosis of EEG was retained in the other two based on digestive signs, blood eosinophilia, and the aspect of colonic parietal thickening on CT scan, without any other cause found.

From a therapeutic point of view, we did not prescribe a diet to our patients because none of them reported food allergies. However, skin tests looking for an allergy have not been carried out. Indeed, diet can play an important role in the remission of symptoms, with clinical improvement reported in 75% of patients who had well respected it especially those with mucosal involvement [12]. Furthermore, EGE responds very well to corticosteroid therapy, with disappearance of symptoms, ascites, hypereosinophilia and eosinophilic tissue infiltration [13]. Prednisone is the molecule of choice with remission reported in over 90% of cases [2]. All our patients initially evolved well under prednisone-based treatment. A rapid disappearance of ascites was noted, as was the amendment of all digestive signs. However, recurrence is feared and causes low doses of corticosteroids to be maintained over time in these cases [9] [14].

This study has some limitations. Indeed, we performed a retrospective study on a small number of patients with a diagnosis of EEG. It is a disease less reported in sub-Saharan Africa. There is a significant number of patients with incomplete data or having undergone colonoscopy for chronic diarrhea of undetermined aetiology in whom the anatomopathological examinations of colonic biopsies were not contributory. The search for eosinophils on the samples and especially their count under the microscope are often lacking in the anatomopathological reports. Thus, a complementary prospective study would make it possible to better evaluate the incidence of the GEE in our countries.

5. Conclusion

In front of digestive symptomatology associated with eosinophilia in a tropical environment, parasitoses are evoked in the first place. However, after eliminating them, we must think of eosinophilic gastroenteritis, which is rare. The diagnosis is based on the demonstration of eosinophilic infiltration of the digestive wall on biopsies or on ascites fluid. Eosinophilic gastroenteritis responds very well to corticosteroid treatment, but relapse is frequent.

Data Availability Statement

All the files of the patients are at principal hospital of Dakar.

Conflicts of Interest

No conflict of interest for all authors.

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