

## About A Case of Eosinophilic Colitis

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| Received: 09.04.2021 | Accepted: 03.06.2021 | Published: 29.08.2021

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### Abstract

### Case Report

Eosinophilic colitis (EC) is caused by the significant accumulation of eosinophilic polynuclear infiltrate in the colonic mucosa. It is a pathology in infants and children under 2 years of age, but is rarer and less studied in adults. The positive diagnosis is based on clinical, endoscopic, histological and biological arguments. Treatment involves the antigen avoidance diet against the responsible antigen and medical treatment. The clinical symptomatology for adults with eosinophilic colitis is variable and nonspecific. Diarrhea and abdominal pain are the most common signs. There is no histological consensus for the diagnosis. The presence of more than 40 eosinophils per field in at least two different colonic segments could be suggested as a diagnostic criterion. Also, there is no consensus on the treatment of eosinophilic colitis, but the potential efficacy of corticosteroids and budesonide has been demonstrated in the vast majority of cases studied.

**Keywords:** Colitis, eosinophil, colon, allergy.

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## INTRODUCTION

Eosinophilic gastrointestinal disorders consist of eosinophilic esophagitis, eosinophilic gastritis, eosinophilic gastroenteritis, eosinophilic enteritis and eosinophilic colitis.

These disorders are either primary, without a known cause responsible for the eosinophilic infiltrate, or secondary to known and identified causes [1, 2]. The lining of the stomach and small intestine is more frequently involved, while colonic involvement is less common.

Eosinophilic colitis is a rare disease characterized by abnormal eosinophilic infiltration of the colonic mucosa. Its real impact is difficult to estimate. The diagnosis is histopathological, based on colonic biopsies which shows an increased number of eosinophils.

Patients present with a variety of digestive symptoms, including abdominal pain, diarrhea, and rectal bleeding, unrelated to any other cause [1,2].

## OBSERVATION

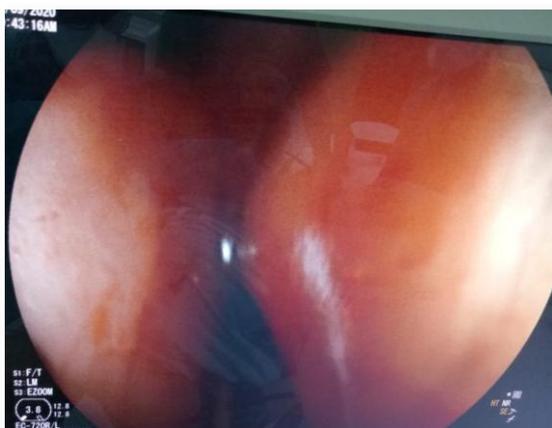
We report the observation of a 20-year-old patient with a history of seasonal allergic rhinitis without current treatment, admitted for etiological

assessment of chronic liquid diarrhea sometimes mucous without blood at a rate of 3 to 4 stools per day, without other associated digestive or extra-digestive manifestations, evolving in a context of apyrexia and deterioration of the general condition that associates asthenia, anorexia and weight loss of 6 kg in 2 months. The clinical examination was without abnormality.

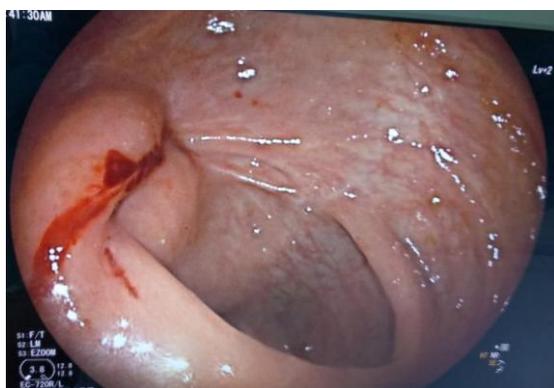
The biological assessment objectified hemoglobin at 12.1 g / dl, GB: 9800 / uL with one of the eosinophils at 610 / uL and platelets at 401000 / uL.

Kidney function and blood ionogram were normal, and the assessment of malabsorption was normal. A tuberculosis assessment including a chest x-ray, sputum testing for Kock's bacilli and a quantiferon was requested in order to rule out colonic intestinal tuberculosis and was found to be normal.

Coprological and parasitological examinations of the stools were negative. A total colonoscopy progressing to the low cecal on a moderately prepared mucosa, objectifying at the level of the last ileal loop, an erythematous mucosa, site of an impassable narrowing with an ileocecal valve edematized, swollen and retracted, without visible ulceration (Figure 1 and 2).

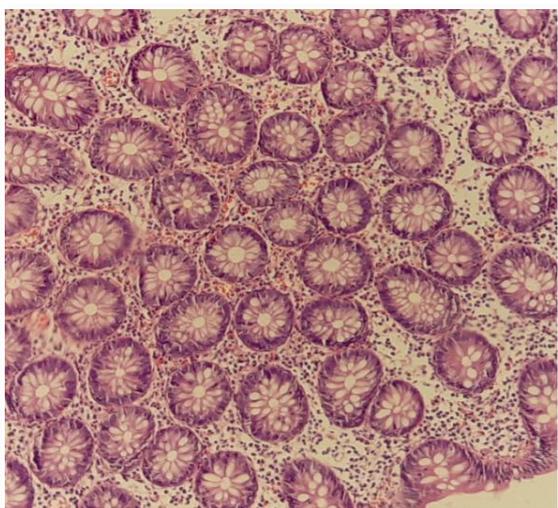


**Fig-1: Endoscopic image showing erythematous colonic mucosa**

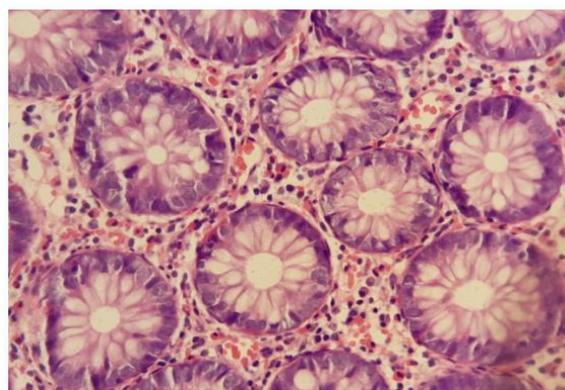


**Fig-2: Endoscopic image of the swollen ileocecal valve without superficial ulcerations**

Colorectal biopsies revealed a simple, mucosecreting columnar epithelium exulcerated in places, the site of a moderate, diffuse and polymorphic inflammatory infiltrate, rich in eosinophils, exceeding 40 to 50 eosinophilic polymers per field at high magnification (Figure 3 and 4).



**Fig-3: Histological image showing an epithelium rich in eosinophilic polymuclear cells (HE x 20).**



**Fig-4: Colonic mucosa with increased number of eosinophils in the lamina propria compatible with colonic eosinophilia [HE x 40].**

These arguments led to the conclusion of the diagnosis of eosinophilic colitis. The treatment consisted of systemic corticosteroid therapy at a dose of 1 mg / kg / day for 4 weeks, followed by a gradual decline associated with the treatment of her allergic rhinitis with an antihistamine. The evolution was marked by a clear clinical improvement with disappearance of the symptoms.

## DISCUSSION

Eosinophilic colitis is defined by the significant accumulation of eosinophilic infiltrate in the colonic mucosa. It is often associated with varieties of disorders such as: eosinophilic gastroenteritis, eosinophilic enteritis, food allergic colitis, drug reactions, parasitic digestive infections and inflammatory diseases of the digestive tract (IBD). The diagnosis is made on a set of clinical, endoscopic, histological and biological arguments [3-8].

EC is classified in two forms, primary and secondary. Primary forms include atopic and non-atopic variants. However, the secondary forms include two forms: one with systemic eosinophilic disorders and the other with non-systemic disorders. The allergic mechanism of EC is usually of the non-Ig E type mediated. Some studies point to a T-type lymphocyte-mediated process, but the exact immunological mechanism responsible for this entity is not clearly understood [9].

The epidemiology of EC is difficult to study, in part due to the lack of well-defined diagnostic criteria [10]. Eosinophilic colitis appears to have a first peak in newborns and a second peak in young adults [11, 12].

The prevalence of eosinophilic gastrointestinal disorders appears to be higher in urban settings than in rural areas [14], among women and the caucasian race [11,13].

Symptoms are variable and nonspecific, and often intermittent and interspersed with periods of remission. Diarrhea is the most frequent symptom, present in more than 60% of cases, while rectal bleeding is only found in 10 to 20% of the cases studied. Abdominal pain is also common, seen in 60 to over 80% of cases. Nausea and vomiting have been noted in about 30% of cases, abdominal bloating, constipation [15], minimal weight loss is also possible, while a real deterioration in general condition is exceptional [11].

The most common allergic diseases associated with eosinophilic colitis are rhinitis, eczema, dermatitis, food allergy, and asthma [16].

Regarding the biological assessment, blood eosinophilia is a marker to guide the diagnosis in combination with other factors. Blood eosinophilia is not constant, it is only present in 27% to 75% of patients. An increase in the level of eosinophils in the faeces can also be observed [12].

The search for antibodies specific to the allergen involved is almost always negative, as is the search for Ig E specific to certain foods.

The prick-test has a good negative predictive value, it consists of the intra-cutaneous injection of the allergen which, if positive, is indicative of a semi-delayed or delayed allergic reaction, and the patch test which allows the diagnosis of an allergy not mediated by Ig E [15].

The endoscopic exploration during colonoscopy is not pathognomonic of this pathology which shows: a congestive, inflammatory mucosa, with a loss of the appearance of the usual vascularization of the mucosa and especially associated with an aspect of lympho-hyperplasia nodular of the recto-colic mucosa. The involvement is generally segmental, the localization of these lesions is usually at the rectosigmoid level but can extend over the entire colonic mucosa with varying intensity [1-4]. The colonic mucosa is endoscopically normal in approximately 70% of cases [12].

The histological study of the per-endoscopic biopsies revealed a mucosa with an overall well-preserved architecture, focused aggregations of eosinophilic infiltrates at the level of the lamina propria, the epithelial crypt and the muscular mucosa. Sometimes these focused aggregations form eosinophilic microabscesses. Occasionally, the presence of multinucleated giant cells is noted in the submucosa [3]. There is no absolute reference figure to be able to define the necessary number of eosinophilic infiltrates [17]. However, according to literature data, massive infiltration by eosinophils with a density <10 eosinophils / HPF is considered minimal, between 10-

20 eosinophils / HPF is considered moderate and  $\geq 20$  eosinophils / HPF is considered severe for the diagnosis of histological eosinophilic colitis [1-4].

Adult eosinophilic colitis quite often requires adjuvant drug therapy, because IgE-mediated allergic manifestations are rarely identified. Drug treatment with: cromoglycate, montelukast, type 1 antihistamine (anti-H1) are only quite often effective, according to literature studies [2]. However, the use of nonsteroidal anti-inflammatory drugs (NSAIDs), including aminosalicylates and corticosteroids (systemic or topical), appears to be effective in the majority of cases [18].

The effectiveness of corticosteroids in the treatment of eosinophilic colitis is largely related to the inhibition of growth factors, such as IL-3 and IL-5. Several studies suggest that oral corticosteroid therapy at a daily dose of 1 mg / kg / day for a period of 2 to 8 weeks, followed by progressive reduction, would improve the histological symptoms in the vast majority of cases. The main problem with corticosteroids is the high relapse rate [19].

Budesonide is a therapeutic alternative which has also been effective in achieving prolonged remissions [20, 21], with fewer side effects than corticosteroids.

In some severe cases resistant to these usual treatments, use of exclusive parenteral nutrition (NPWT) or treatment with immunosuppressants, such as azathioprine or 6-mercaptopurine, represent alternative treatments [1-4].

The prognosis for eosinophilic colitis is much more guarded, as these diseases progress to chronicity because ECs can be clinical pathological manifestations of other primary diseases. Longitudinal clinical monitoring of the cardiopulmonary system and the upper and lower gastrointestinal tract is required with repeated upper and lower gastrointestinal endoscopies [2-4].

## CONCLUSION

Eosinophilic colitis remains a rare entity. The pathophysiology, diagnostic criteria and treatment are still poorly understood.

Therefore, future studies including a large sample of patients will be necessary to improve the management of eosinophilic colitis in adults, in particular to define and validate specific diagnostic criteria, to clarify the role of anti-IL5, anti- monoclonal antibodies. IgE and anti-TNF alpha in therapeutic strategies and establish the most appropriate treatment for the disease.

## REFERENCE

1. Rothenberg, M. E. (2004). Eosinophilic gastrointestinal disorders (EGID). *Journal of Allergy and Clinical Immunology*, 113(1), 11-28.
2. Furuta, G. T., Forbes, D., Boey, C., Dupont, C., Putnam, P., Roy, S. K., ... & Husby, S. (2008). Eosinophilic gastrointestinal diseases (EGIDs). *Journal of pediatric gastroenterology and nutrition*, 47(2), 234-238.
3. Guajardo, J. R., & Rothenberg, M. E. (2003). Eosinophilic esophagitis, gastroenteritis, gastroenterocolitis, and colitis. *Food allergy: adverse reactions to foods and additives*, 217-226.
4. Behjati, S., Zilbauer, M., Heuschkel, R., Phillips, A., Salvestrini, C., Torrente, F., & Bates, A. W. (2009). Defining eosinophilic colitis in children: insights from a retrospective case series. *Journal of pediatric gastroenterology and nutrition*, 49(2), 208-215.
5. Odze, R. D., Wershil, B. K., Leichtner, A. M., & Antonioli, D. A. (1995). Allergic colitis in infants. *The Journal of pediatrics*, 126(2), 163-170.
6. Gonsalves, N. (2007). Food allergies and eosinophilic gastrointestinal illness. *Gastroenterology Clinics of North America*, 36(1), 75-91.
7. Walsh, R. E., & Gaginella, T. S. (1991). The eosinophil in inflammatory bowel disease. *Scandinavian journal of gastroenterology*, 26(12), 1217-1224.
8. Liu, L. X., Chi, J., Upton, M. P., & Ash, L. R. (1995). Eosinophilic colitis associated with larvae of the pinworm *Enterobius vermicularis*. *The Lancet*, 346(8972), 410-412.
9. Van Sickle, G. J., Powell, G. K., McDonald, P. J., & Goldblum, R. M. (1985). Milk-and soy protein-induced enterocolitis: evidence for lymphocyte sensitization to specific food proteins. *Gastroenterology*, 88(6), 1915-1921.
10. Collins, M. H., Capocelli, K., & Yang, G. Y. (2018). Eosinophilic gastrointestinal disorders pathology. *Frontiers in medicine*, 4, 261.
11. Jensen, E. T., Martin, C. F., Kappelman, M. D., & Dellon, E. S. (2016). Prevalence of eosinophilic gastritis, gastroenteritis, and colitis: estimates from a national administrative database. *Journal of pediatric gastroenterology and nutrition*, 62(1), 36.
12. Del Arco, C. D., Taxonera, C., Olivares, D., & Aceñero, M. J. F. (2018). Eosinophilic colitis: case series and literature review. *Pathology-Research and Practice*, 214(1), 100-104.
13. Mansoor, E., Abou Saleh, M., & Cooper, G. S. (2017). Prevalence of eosinophilic gastroenteritis and colitis in a population-based study, from 2012 to 2017. *Clinical Gastroenterology and Hepatology*, 15(11), 1733-1741.
14. Spergel, J. M., Book, W. M., Mays, E., Song, L., Shah, S. S., Talley, N. J., & Bonis, P. A. (2011). Variation in prevalence, diagnostic criteria, and initial management options for eosinophilic gastrointestinal diseases in the United States. *Journal of pediatric gastroenterology and nutrition*, 52(3), 300.
15. Alfadda, A. A., Storr, M. A., & Shaffer, E. A. (2011). Eosinophilic colitis: epidemiology, clinical features, and current management. *Therapeutic advances in gastroenterology*, 4(5), 301-309.
16. Zammit, S. C., Cachia, M., Sapiano, K., Gauci, J., Montefort, S., & Ellul, P. (2018). Eosinophilic gastrointestinal disorder: is it what it seems to be?. *Annals of gastroenterology*, 31(4), 475.
17. DeBrosse, C. W., Case, J. W., Putnam, P. E., Collins, M. H., & Rothenberg, M. E. (2006). Quantity and distribution of eosinophils in the gastrointestinal tract of children. *Pediatric and developmental pathology*, 9(3), 210-218.
18. Elawad, M. A., Hill, S. M., Smith, V., Lindley, K. J., & Milla, P. J. (2004). P0491 EOSINOPHILIC COLITIS AFTER INFANCY. *Journal of Pediatric Gastroenterology and Nutrition*, 39, S243.
19. Chen, M. J., Chu, C. H., Lin, S. C., Shih, S. C., & Wang, T. E. (2003). Eosinophilic gastroenteritis: clinical experience with 15 patients. *World journal of gastroenterology: WJG*, 9(12), 2813.
20. Tan, A. C., Kruiemel, J. W., & Naber, T. H. (2001). Eosinophilic gastroenteritis treated with non-enteric-coated budesonide tablets. *European journal of gastroenterology & hepatology*, 13(4), 425-427.
21. Siewert, E., Lammert, F., Koppitz, P., Schmidt, T., & Matern, S. (2006). Eosinophilic gastroenteritis with severe protein-losing enteropathy: successful treatment with budesonide. *Digestive and liver disease*, 38(1), 55-59.