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Eosinophilic Colitis and Crohn's Disease: A Rare Overlapping Entity in an Adult

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Abstract | **Case Report**

***Background*:** Eosinophilic colitis (EC) is a rare form of primary eosinophilic gastrointestinal disorder, predominantly affecting neonates and young adults. Unlike eosinophilic esophagitis, EC remains poorly understood, with limited data derived from small case series and reports. It is characterized by eosinophilic infiltration of the intestinal wall, submucosal edema, and peripheral eosinophilia. However, the presence of eosinophils in colonic biopsies necessitates ruling out secondary causes, making EC a diagnosis of exclusion. ***Case Presentation*:** We report the case of a 32-year-old male with no significant medical history who presented with recurrent watery diarrhea over seven months, associated with atypical diffuse abdominal pain, predominantly in the right iliac fossa. Laboratory findings revealed an inflammatory syndrome, mild peripheral eosinophilia, and positive ASCA antibodies, while biochemical tests were unremarkable. An abdominal CT scan showed thickening of the right colon and cecum with adjacent infiltration. Colonoscopy revealed erythematous cecal mucosa with superficial ulcerations, along with edematous and erythematous mucosa in parts of the right colon. Histopathological analysis of colonic biopsies demonstrated overlapping features of eosinophilic colitis and Crohn's colitis. The patient was treated with corticosteroids and azathioprine (2.5 mg/kg/day), leading to significant clinical and biological improvement. ***Discussion*:** A review of the literature reveals considerable variability in the role of eosinophils in both inflammatory bowel disease and eosinophilic colitis. While eosinophilic infiltration is commonly observed in IBD, its exact significance remains unclear. The coexistence of EC and Crohn's disease is rare and raises questions regarding a possible shared immunopathological mechanism. ***Conclusion*:** This case underscores the importance of considering eosinophilic colitis in the differential diagnosis of chronic diarrhea with eosinophilia, particularly in patients with atypical presentations of IBD. A comprehensive diagnostic workup, including histopathological assessment, is essential to distinguish between primary and secondary EC. Further studies are needed to elucidate the role of eosinophils in the pathogenesis of Crohn's disease and their impact on disease progression and treatment response.

Keywords: Eosinophilic Colitis, Crohn's Disease, Inflammatory Bowel Disease, Eosinophilia.

INTRODUCTION

Eosinophilic gastrointestinal disorders (EGIDs) can involve any segment or combination of segments of the gastrointestinal tract, from the esophagus to the rectum, leading to a variety of clinical presentations. These disorders are classified into three main subcategories: eosinophilic esophagitis (EE), eosinophilic gastroenteritis (EG), and eosinophilic colitis (EC) [1]. EC is extremely rare, with only a few cases reported since 1979. It most commonly affects neonates and young adults and is part of the broader spectrum of eosinophilic gastrointestinal disorders [2]. The clinical presentation of EC is nonspecific, with symptoms including abdominal pain, weight loss, diarrhea (either bloody or non-bloody), and malabsorption. When the entire bowel wall is involved, intestinal obstruction and even perforation may occur [3]. The majority of primary EC cases are idiopathic [4, 5]. However, colonic eosinophilia can also be secondary to parasitic infections, inflammatory bowel disease (IBD), autoimmune diseases (e.g., scleroderma, Churg-Strauss syndrome), celiac disease, drug reactions, or in association with hypereosinophilic syndrome [2-7]. Thus, primary EC is a diagnosis of exclusion. The pathogenesis of EC and other EGIDs is not well understood, although studies, particularly those focusing on pediatric populations,

suggest that food allergies, atopy, or gut dysbiosis may contribute to the development of these disorders [8]. Primary eosinophilic gastrointestinal disease (EGID), first described by Kaijser in 1937 [9], is a rare group of gastrointestinal disorders characterized by eosinophilic inflammation without any identifiable cause for the eosinophilia, such as parasitic infection, drug reactions, or malignancy [10]. EC represents the least common manifestation of EGID, whether it occurs alone or in conjunction with disease in other parts of the gastrointestinal tract [11]. EC appears to have a bimodal distribution, affecting neonates at a relatively high prevalence and a separate group of young adults, with no gender preference [10]. Peripheral eosinophilia is inconsistent. The definitive diagnosis is established through biopsy [6].

The presence of activated tissue and peripheral blood eosinophils in chronic inflammatory bowel disease (IBD) has been documented for many years. Additionally, eosinophilic granulocytes in rectal mucus and elevated levels of eosinophil cationic protein in fecal or gut lavage fluids have been observed in patients with IBD [12]. Interestingly, allergic and respiratory symptoms, including cases of eosinophilic pneumonia [13], bronchial hyperreactivity, abnormal lung function tests, and positive skin prick tests, were more prevalent in patients with IBD compared to the control group [14].

This article presents a case of a patient with chronic diarrhea diagnosed with overlapping eosinophilic and Crohn's colitis.

CASE REPORT

A 32-year-old male with no significant medical history (such as atopy, medication use, etc.) presented with recurrent episodes of watery diarrhea over the past seven months, associated with atypical diffuse abdominal pain, most pronounced in the right iliac fossa. His physical examination did not reveal any remarkable findings. Examination of the anal margin and the rectal examination were normal.

The biological workup revealed an inflammatory syndrome (C-reactive protein = 65 mg/L, erythrocyte sedimentation rate = 27 mm/h), fecal calprotectin = 1158 mg/kg, slightly elevated eosinophils (3200 cells per mm^3), and ASCA antibodies were positive. Quantiferon, PANCA and antitransglutaminase antibodies were negative. Biochemical tests were unremarkable. Parasitological examination of stool and stool bacterial cultures were negative. Abdominal CT scan showed thickening of the right colon and cecum with adjacent infiltration, where lymph nodes were located.

Gastroscopy showed a congestive gastropathy. Pathology was normal for esophageal and duodenal biopsies and showed chronic gastritis without HP for gastric biopsies. Colonoscopy demonstrated diffuse mucosal edema, erythema, and superficial ulcerations involving the cecum and adjacent segments of the right colon. Multiple colonic biopsies were taken. Histological analysis revealed overlapping features of eosinophilic colitis and Crohn’s colitis. The inflammatory infiltrate was moderate, diffuse, and polymorphous, consisting primarily of lymphocytes, plasma cells, and a substantial number of eosinophils. The eosinophils were notably abundant, with counts exceeding 40 eosinophils per high-power field, and exhibited signs of degranulation. Focal architectural distortion and fibrosis were also observed, suggesting ongoing tissue remodeling. No granulomas were present. Biopsies from the terminal ileum did not show any significant abnormalities.

The patient was started on high-dose corticosteroid therapy with a tapering regimen and azathioprine (2.5 mg/kg/day). With this treatment, the patient's symptoms regressed, and he became asymptomatic during subsequent follow-up visits.

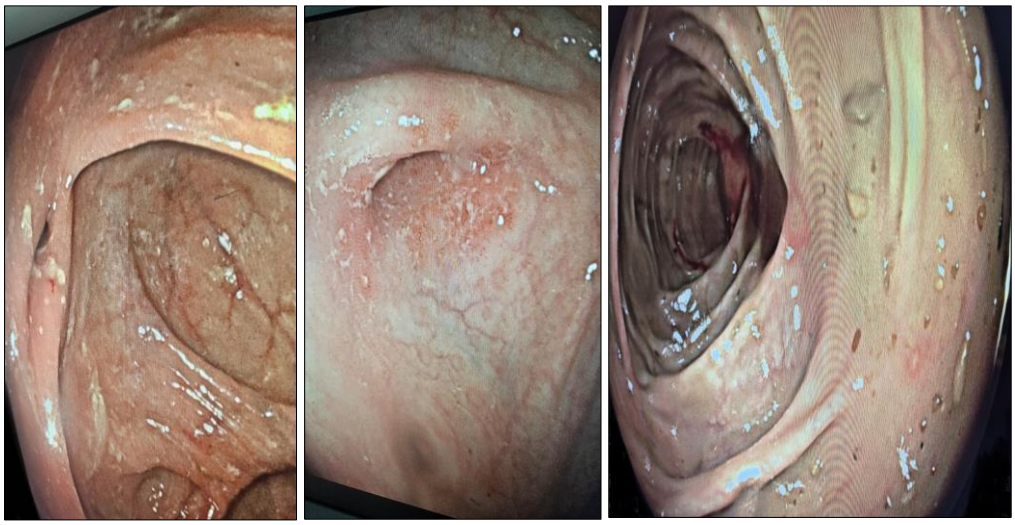

Figure 1: Colonoscopic finding erythematous and edematous mucosa

DISCUSSION

A literature review reveals significant variability in eosinophil involvement in IBD and eosinophilic gastroenteritis, with some rare cases resembling the one presented here. Eosinophilic gastrointestinal disorders (EGIDs) are chronic conditions characterized by eosinophilic infiltration of the digestive tract, primarily affecting the stomach and small intestine, with colonic involvement being rare. Peripheral eosinophilia is inconsistent. EC remains the least common form of EGID, though its incidence has increased over the past decade [15].

EC follows a bimodal age distribution, peaking in neonates and young adults, with no gender preference [16-5]. It can be classified as primary or secondary [17]. Secondary EC results from eosinophilic disorders, such as hypereosinophilic syndrome, or other conditions, including IBD, parasitic infections, autoimmune diseases, vasculitis, medications, malignancies, and IgG4-related disease [2-19]. Drug-induced EC has been linked to various medications, though its exact mechanism remains unclear [2-20].

Primary EC is often associated with allergic reactions, either IgE-mediated (anaphylactic-type food allergies) or non-IgE-mediated (food enteropathy), with milk proteins being the most common trigger in pediatric cases [10]. Allergic conditions such as rhinitis, eczema, and asthma are frequently linked to EC [16]. The pathogenesis of EGIDs remains unclear, though food allergies, atopy, and gut dysbiosis have been implicated in pediatric cases [18]. In adults, potential causes remain poorly understood. While IgE involvement in EGIDs is debated, the dominant hypothesis suggests a Th2-mediated mechanism, with interleukin-5 and eotaxins playing a pathogenic role [19].

The diagnosis of EGID relies on clinical symptoms, peripheral eosinophilia, histological findings, and exclusion of secondary causes. EC presentation varies with the location and extent of bowel involvement. Mucosal involvement leads to abdominal pain, nausea, vomiting, and diarrhea, while muscular layer involvement may cause obstruction. Subserosal infiltration can result in ascites [7]. Laboratory tests, including eosinophil counts and IgE levels, have limited sensitivity and specificity [15]. Endoscopic findings are often normal, making biopsy collection crucial for diagnosis, even when the mucosa appears unremarkable [21].

Eosinophils are normally present in the colonic lamina propria, with regional variations in density. However, there is no consensus on normal eosinophil levels. Some studies suggest that an eosinophil count >40 per high-power field in at least two colonic segments confirms EC [17]. Additional histopathological features, such as eosinophil degranulation, submucosal involvement, and edema, are useful diagnostic markers [5].

The association between eosinophilic disorders and IBD is complex. Reports suggest a potential overlap between eosinophilic esophagitis and Crohn's disease (CD) [22]. Eosinophilic granulomas have also been identified in association with Crohn's disease in the ileum [23]. While EC and CD share histological features, key differences exist. Unlike CD, EC typically lacks granulomas, fibrosis, and crypt distortion. The inflammatory infiltrate in EC is predominantly eosinophilic, whereas CD shows mixed inflammatory cells. Some cases of EC may eventually be diagnosed as CD [24].

Eosinophils play a role in IBD pathogenesis [25]. Studies have shown altered eosinophil and mast cell numbers in IBD patients, correlating with disease activity [26]. Eosinophilic infiltration is more frequent in CD than in UC, making it a useful histological marker [27]. Inflammation-driven eosinophil activation leads to tissue damage via the release of cytotoxic granule proteins and pro-inflammatory cytokines [28]. Eosinophil counts are elevated in both active and remissive phases of CD and UC, with a strong correlation between eosinophil activation and disease severity [29].

Eosinophils may also contribute to fibrosis and disease progression in IBD. Increased eosinophil activity and interleukin-5 expression have been linked to higher recurrence rates after surgery in CD [30]. Experimental models suggest that blocking eosinophil recruitment reduces intestinal inflammation and remodeling [31]. In UC, eosinophilia correlates with severe disease, surgical intervention, and primary sclerosing cholangitis [32]. While eosinophils are mainly pro-inflammatory, emerging evidence suggests they may also have protective roles. Some studies report increased eosinophil activation during UC remission, possibly due to the release of anti-inflammatory mediators like protectin D1 [33-36].

The overlap between eosinophilic and idiopathic colonic diseases may indicate shared immunopathogenic mechanisms. The gastrointestinal tract is constantly exposed to environmental antigens, triggering diverse inflammatory responses. Individual variations in immune regulation may explain the heterogeneity of these disorders, highlighting the need for further research to clarify their pathophysiology and treatment implications [37].

REFERENCES

1. J.L. Brandon, S. Schroeder, G.T. Furuta, K. Capocelli, J.C. Masterson, L.Z. Fenton, CT imaging features of eosinophilic colitis in children, Pediatr. Radiol. 43 (2013) 697–702
2. Okpara N, Aswad B, Baffy G. Eosinophilic colitis. World J Gastroenterol 2009; 15: 2975–2979

3. Walker MM, Potter MD, Talley NJ. Eosinophilic colitis and colonic eosinophilia. Curr Opin Gastroenterol. 2019;35(1):42–50.
4. V. Uppal, P. Kreiger, E. Kutsch, Eosinophilic gastroenteritis and colitis: a comprehensive review, Clin. Rev. Allergy Immunol. 50 (2016) 175-188.
5. M.S. Villanueva, Y. Alimi, Microscopic colitis (lymphocytic and collagenous), eosinophilic colitis, and celiac disease, Clin. Colon Rectal Surg. 28 (2015) 118–126.
6. Uppal V, Kreiger P, Kutsch E. Eosinophilic gastroenteritis and colitis: a comprehensive review. Clin Rev Allergy Immunol 2016; 50: 175–188.
7. Dionísio de Sousa IJ, Bonito N, Pais A et al. Eosinophilic colitis. BMJ Case Rep 2016. doi:10.1136/bcr-2016-214496.
8. Walker MM, Potter M, Talley NJ. Eosinophilic gastroenteritis and other eosinophilic gut diseases distal to the oesophagus. Lancet Gastroenterol Hepatol. 2018;3(4):271–80.
9. Kaijser R. Zur Kenntnis der allergischen affektionen des verdauungskanals vom standput des chirurgen aus. Arch Klin Chir. 1937;188:36-64.
10. Rothenberg ME. Eosinophilic gastrointestinal disorders (EGID) J Allergy Clin Immunol. 2004;113:11-29.
11. Guajardo JR, Plotnick LM, Fende JM, Collins MH, Putnam PE, Rothenberg ME. Eosinophil-associated gastrointestinal disorders: a world-wide-web based registry. J Pediatr. 2002;141:576-81.
12. Konstantinos H. Katsanos, Eirini Zinovieva, Evangelia Lambri, Epameinondas V. Tsianos. Eosinophilic-Crohn overlap colitis and review of the literature. Journal of Crohn's and Colitis (2011) 5, 256–261
13. B, Enayati P, Marchevsky A, Papadakis KA. Pulmonary manifestations of inflammatory bowel disease: case presentations and review. J Crohns Colitis 2010;4:390–7.
14. Ceyhan BB, Karakurt S, Cevik H, Sungur M. Bronchial hyperreactivity and allergic status in inflammatory bowel disease. Respiration 2003;70:60–6.
15. Smaoui *et al.* Eosinophilic enterocolitis: a case report. *Journal of Medical Case Reports (2024)* https://doi.org/10.1186/s13256-023-04319-9
16. E.T. Jensen, C.F. Martin, M.D. Kappelman, E.S. Dellon, Prevalence of eosinophilic gastritis, gastroenteritis, and colitis, J. Pediatr. Gastroenterol. Nutr.2016;62(1):36–42
17. [Macaigne G. Eosinophilic colitis in adults. Clin Res Hepatol Gastroenterol. 2020;44(5):630–7.].
18. Shaikh TP, Ansari S, Deolekar S, Mandhane N, Karandikar S. Eosinophilic colitis in 36 years old female: a case presentation with review of literature. Int J Sci Rep 2015;1(3):172-6.
19. Díaz del Arco, C., Pathology -Research and Practice (2017), http://dx.doi.org/10.1016/j.prp.2017.09.029
20. Fragkos KC et al. Severe eosinophilic colitis caused by neuropathic agents in a patient with chronic fatigue syndrome and functional abdominal pain: case report and review of the literature. Z Gastroenterol 2018; 56: 573–577)
21. Reed C, Woosley JT, Dellon ES. Clinical characteristics, treatment outcomes, and resource utilization in children and adults with eosinophilic gastroenteritis. Dig Liver Dis 2015;47:197–201.
22. Suttor VP, Chow C, Turner I. Eosinophilic esophagitis with Crohn's disease: a new association or overlapping immune-mediated enteropathy? Am J Gastroenterol 2009;104:794–5.
23. Catalá M, Sánchez A, Hinojosa J, Primo J, Flors C, Calvete J, et al. An inflammatory fibroid polyp (eosinophilic granuloma) and Crohn's disease located in the ileum. Rev Esp Enferm Dig 1992;82:193–5.
24. Fenoglio-Preiser Cecilia M. Gastrointestinal Pathology: an Atlas and a TextThird Edition. . Lippincott Williams & Wilkins Eds; 2008.
25. Wedemeyer J, Vosskuhl K. Role of gastrointestinal eosinophils in inflammatory bowel disease and intestinal tumours. Best Pract Res Clin Gastroenterol 2008;22:537–49.
26. Bischoff SC, Mayer J, Nguyen QT, Stolte M, Manns MP. Immunnohistological assessment of intestinal eosinophil activation in patients with eosinophilic gastroenteritis and inflammatory bowel disease. Am J Gastroenterol 1999;94:3521–9.
27. Rubio CA. A method for the detection of eosinophilic granulocytes in colonoscopic biopsies from IBD patients. Pathol Res Pract 2003;199: 145–50.
28. Alhmoud T, *et al*. Outcomes of inflammatory bowel disease in patients with eosinophil predominant colonic inflammation. *BMJ Open Gastro* 2020;7:e000373. doi:10.1136/bmjgast-2020-000373
29. Smyth CM, Akasheh N, Woods S, *et al*. Activated eosinophils in association with enteric nerves in inflammatory bowel disease. *PLoS One* 2013;8:e64216.
30. Dubucquoi S, Janin A, Klein O, *et al*. Activated eosinophils and interleukin 5 expression in early recurrence of Crohn's disease. *Gut* 1995;37:242–6
31. Masterson JC, McNamee EN, Jedlicka P, *et al*. Ccr3 blockade attenuates eosinophilic ileitis and associated remodeling. *Am J Pathol* 2011;179:2302–14
32. Barrie A, Mourabet ME, Weyant K, *et al*. Recurrent blood eosinophilia in ulcerative colitis is associated with severe disease and primary sclerosing cholangitis. *Dig Dis Sci* 2013;58:222–8.
33. Xu X, Rivkind A, Pikarsky A, *et al*. Mast cells and eosinophils have a potential profibrogenic role in Crohn disease. *Scand J Gastroenterol* 2004;39:440–7.
34. Heatley RV, James PD. Eosinophils in the rectal mucosa. A simple method of predicting the outcome of ulcerative proctocolitis? *Gut* 1979;20:787–91

35. Lampinen M, Rönnblom A, Amin K, *et al*. Eosinophil granulocytes are activated during the remission phase of ulcerative colitis. *Gut* 2005;54:1714–20
36. Isobe Y, Kato T, Arita M. Emerging roles of eosinophils and eosinophil-derived lipid mediators in the resolution of inflammation. *Front Immunol* 2012;3:270
37. Geboes K. From inflammation to lesion. Acta Gastroenterol Belg 1994;57:273–84.