# **Eosinophilic Colitis Presenting as Chronic Diarrhea: A Rare Case Report**

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### Abstract

Eosinophilic colitis (EC) is the rarest form of eosinophilic gastrointestinal disorders (EGIDs), presenting diagnostic challenges due to its nonspecific symptoms and overlap with conditions such as hypereosinophilic syndrome (HES). We report a case of a 62-year-old male who presented with chronic diarrhea, abdominal pain, and significant weight loss over a three-month period. Laboratory investigations revealed peripheral eosinophilia (absolute eosinophil count: 2,030/µL) with elevated fecal calprotectin. Endoscopic evaluation demonstrated diffuse mucosal and submucosal nodules from the rectum to the cecum, and histopathology confirmed marked eosinophilic infiltration with focal microabscesses. Systemic involvement was excluded through imaging studies. Given the absence of secondary causes, a diagnosis of EC was established. The patient was initiated on corticosteroid therapy, leading to resolution of symptoms and normalization of eosinophil counts. This case underscores the importance of considering EC in patients with chronic diarrhea and eosinophilia. It also highlights the role of histopathology in establishing the diagnosis. While corticosteroids remain the mainstay of treatment, emerging therapies such as biologics and dietary interventions may offer alternative management strategies. Further research is required to refine diagnostic criteria and optimize long-term treatment outcomes. **Keywords:** Chronic diarrhea; eosinophilic colitis; hypereosinophilic syndrome.

# INTRODUCTION

Eosinophilic gastrointestinal disorders (EGIDs) are a heterogeneous group of inflammatory conditions characterized by excessive eosinophil infiltration in the gastrointestinal (GI) tract, in the absence of secondary causes of eosinophilia, such as infections, autoimmune disorders, or malignancies. These disorders can affect various parts of the GI tract and are classified into distinct subtypes, including eosinophilic esophagitis (EoE), eosinophilic gastritis (EG), eosinophilic gastroenteritis (EGE), eosinophilic enteritis (EE), and eosinophilic colitis (EC). Among these, EC is the rarest and least understood subtype, posing diagnostic and therapeutic challenges due to its nonspecific clinical presentation and the lack of standardized diagnostic criteria. <sup>2,3</sup>

The precise incidence and prevalence of EC remain unclear, as it has been described as an "exceptionally rare" entity, with only a limited number of reported cases since its first description in 1979.<sup>4,5</sup> Due to its rarity, EC is often misdiagnosed as other inflammatory or functional bowel disorders, resulting in delays in appropriate management.

## CASE PRESENTATION

A 62-year-old gentleman presented with intermittent episodes of loose stools, occurring 4–5 times per day, associated with abdominal pain over the past three months. There was no history of hematochezia, fever, or night sweats. However, the patient reported an unintentional weight loss of 7–8 kg during this period, despite an unchanged appetite. He denied recent antibiotic use, a family history of inflammatory bowel disease, or symptoms suggestive of pulmonary or allergic conditions.

On physical examination, signs of malnutrition were evident, but no other significant abnormalities were detected in the gastrointestinal system. Laboratory findings included a hemoglobin level of 13.5 g/dL (normocytic and normochromic) and a total leukocyte count (TLC) of 11,280 cells/ $\mu$ L, with a differential count showing 59% neutrophils, 19% lymphocytes, and 18% eosinophils. The platelet count was 2.64 lakh/ $\mu$ L. The absolute eosinophil count (AEC) was significantly elevated at 2,030/ $\mu$ L. Inflammatory markers, such as C-reactive protein and erythrocyte sedimentation rate, were not elevated.

Further workup included a negative celiac serology. Stool microscopy was unremarkable, and stool cultures did not yield any pathogenic organisms. Fecal calprotectin levels were elevated at  $176 \mu g/g$ , indicating ongoing colonic inflammation.

Upper GI endoscopy did not reveal any macroscopic abnormalities, and duodenal biopsies were not obtained. Subsequent ileocolonoscopy showed

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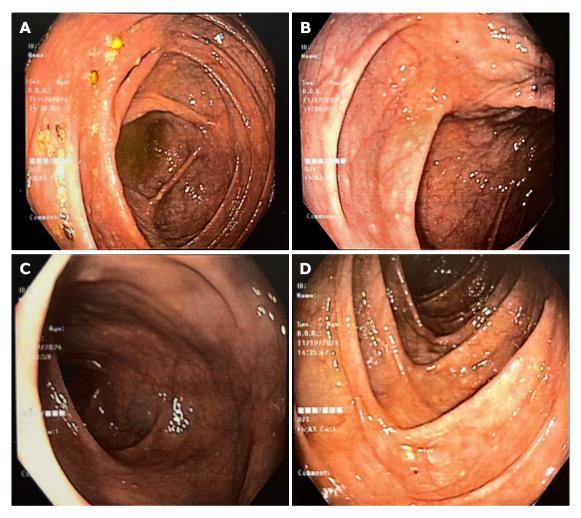


Figure 1 (A-D). Diffuse mucosal and submucosal seed-like whitish nodules observed throughout the colon.

diffusely scattered tiny, seed-like mucosal and submucosal nodules extending from the rectum to the cecum (Figure 1). Multiple biopsies were taken from different colonic segments. Histopathological examination revealed an expanded lamina propria with sheets and aggregates of eosinophils. Crypts exhibited marked reactive changes, with thickening of the muscularis mucosa and focal eosinophilic microabscesses in the submucosa. Notably, there were no features of neutrophilic infiltration, inflammatory bowel disease (IBD), or parasitic or microbial infections (Figures 2).

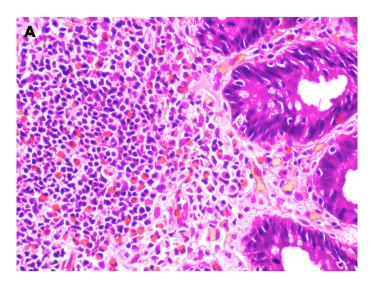
Given the presence of chronic inflammatory changes with significant eosinophilic infiltration in the colonic mucosa, coupled with symptoms of chronic diarrhea and peripheral eosinophilia, a diagnosis of eosinophilic colitis was considered. The differential diagnosis included hypereosinophilic syndrome (HES) with colonic involvement, as there is considerable overlap between these two conditions.

A multidisciplinary team discussion was held to further evaluate possible systemic involvement. Chest computed tomography (CT) and echocardiography were performed, ruling out cardiac or pulmonary involvement. With no evidence of dysfunction in other organ systems, the diagnosis of primary EC was confirmed. A trial of corticosteroid therapy with prednisolone was initiated. The patient showed rapid improve-

# **MAIN POINTS**

- Eosinophilic colitis is a rare and often underdiagnosed subtype of eosinophilic gastrointestinal disorders, presenting with nonspecific symptoms such as chronic diarrhea, abdominal pain, and weight loss.
- Histopathological confirmation of dense eosinophilic infiltration in the colonic mucosa and submucosa remains essential for diagnosis, especially in the presence of peripheral eosinophilia and unremarkable imaging findings.
- The case highlights characteristic endoscopic findings of EC, including diffusely scattered seed-like nodules throughout the colon, and histological features such as eosinophilic microabscesses and lamina propria expansion.
- Systemic corticosteroid therapy leads to rapid clinical and hematologic improvement, but long-term management may require alternative treatments such as biologics or dietary interventions due to the potential for relapse.

ment in diarrheal symptoms and normalization of eosinophil counts with a tapering steroid regimen. Clinical remission was confirmed at outpatient follow-up.



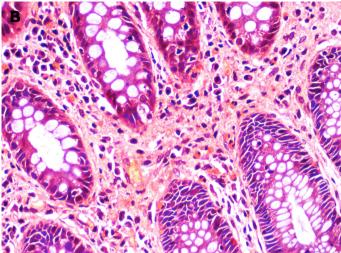


Figure 2. (A) A dense cluster of eosinophils with bright cytoplasm and bilobed nuclei is present in the lamina propria, disrupting colonic crypt architecture with reactive epithelial changes—indicative of significant mucosal and submucosal involvement, a hallmark of eosinophilic colitis. (B) Higher magnification reveals intact goblet cells surrounded by eosinophilic infiltrate, lamina propria expansion, and collagen deposition, consistent with chronic eosinophilic colitis.

## DISCUSSION

Eosinophilic colitis is a rare inflammatory disorder characterized by abnormal eosinophilic infiltration in the colon in the absence of secondary causes such as infections, malignancies, or autoimmune diseases. Due to its rarity and nonspecific clinical presentation, EC is often underdiagnosed or misclassified as other gastrointestinal disorders, such as IBD or irritable bowel syndrome (IBS).

Symptoms of EC can vary widely, but the most common include chronic diarrhea, abdominal pain, and occasional hematochezia. Some patients may present with nausea, vomiting, or weight loss, while others remain asymptomatic, with the disease incidentally detected during endoscopic evaluation. Peripheral eosinophilia, though commonly observed, is not a definitive diagnostic marker, as it may be absent in some cases. Therefore, histopathological confirmation of eosinophilic infiltration in the colonic mucosa remains the gold standard for diagnosis. However, a significant challenge in diagnosing EC is the lack of standardized cutoff values for eosinophil counts in colonic biopsies.

Endoscopic findings in EC are variable, ranging from normal mucosa to diffuse erythema, friability, loss of vascular patterns, or small mucosal nodules, as seen in our patient. While imaging studies such as CT or MRI can help identify wall thickening and inflammation, they are not diagnostic. Therefore, multiple biopsies from different colonic segments are essential for confirming the presence of eosinophilic infiltration and ruling out alternative causes.<sup>7</sup>

# **Treatment**

Treatment strategies for EC primarily focus on reducing eosinophilia and alleviating symptoms. Corticosteroids remain the mainstay of therapy, demonstrating efficacy in reducing eosinophilic infiltration and controlling clinical symptoms. Systemic corticosteroids, such as prednisolone, have shown significant improvement in patients with severe symptoms, particularly those experiencing persistent diarrhea and hematochezia. However, EC is often a relapsing condition, necessitating maintenance therapy to prevent recurrence.

Beyond corticosteroids, emerging therapeutic options are being ex-

plored. These include biologics such as anti-IL-5 monoclonal antibodies (e.g., mepolizumab and reslizumab) and leukotriene receptor antagonists like montelukast, which have shown promise in controlling inflammation while minimizing steroid use. Additionally, dietary interventions, including elimination diets, may benefit patients with suspected food-related triggers.<sup>8</sup>

Despite advances in understanding EC, many challenges remain. The considerable overlap between EC and HES complicates diagnosis, as both conditions share similar histological and clinical features. Furthermore, the absence of standardized diagnostic thresholds makes it difficult to differentiate EC from other eosinophilic gastrointestinal disorders. Multidisciplinary collaboration involving gastroenterologists, hematologists, and pathologists is often required to establish a definitive diagnosis and tailor treatment strategies.

# CONCLUSION

Eosinophilic colitis is a rare yet significant disorder that requires a high index of clinical suspicion for timely diagnosis. Given its nonspecific presentation and the potential for diagnostic overlap with other eosinophilic and inflammatory conditions, a thorough workup, including detailed histopathological examination, is essential. While corticosteroids remain the first-line treatment, ongoing research into novel therapies, including biologics and dietary interventions, may offer better long-term management options. Future studies should focus on standardizing diagnostic criteria and exploring targeted therapies to improve patient outcomes.

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### REFERENCES

 Rothenberg ME. Eosinophilic gastrointestinal disorders (EGID). J Allergy Clin Immunol. 2004;113(1):11-28; quiz 29. [CrossRef]

- Yan BM, Shaffer EA. Primary eosinophilic disorders of the gastrointestinal tract. Gut. 2009;58(5):721-732. [CrossRef]
- Impellizzeri G, Marasco G, Eusebi LH, Salfi N, Bazzoli F, Zagari RM. Eosinophilic colitis: A clinical review. Dig Liver Dis. 2019;51(6):769-773. [CrossRef]
- Alfadda AA, Storr MA, Shaffer EA. Eosinophilic colitis: epidemiology, clinical features, and current management. Therap Adv Gastroenterol. 2011;4(5):301-309. [CrossRef]
- Fernández Salazar LI, Borrego Pintado H, Velayos Jiménez B, González Hernández JM. Differential diagnosis and management of histologic eosinophilic colitis. J Crohns Colitis. 2013;7(1):e20-e21. [CrossRef]
- Macaigne G. Eosinophilic colitis in adults. Clin Res Hepatol Gastroenterol. 2020;44(5):630-637. [CrossRef]
- 7. Jeon YW, Hong SJ, Kim HJ, et al. A hypereosinophilic syndrome presenting as eosinophilic colitis. Clin Endosc. 2012;45(4):444-447. [CrossRef]
- 8. Marasco G, Visaggi P, Vassallo M, et al. Current and Novel Therapies for Eosinophilic Gastrointestinal Diseases. Int J Mol Sci. 2023;24(20):15165. [CrossRef]