Exploring the Intricacies and Clinical Perspectives of Chronic Diarrhea: A Case Report

Tanveer Hussain[1], Anum Abbas[2], Sadia Ahmed[3], Muhammad Umar[4]

Abstract

Summary: Crohn's disease (CD) is a chronic inflammatory disorder that may affect any segment of the gastrointestinal tract, with a predilection for the terminal ileum and colon. It commonly presents chronic diarrhea, abdominal pain, and per rectal (PR) bleeding. We report the case of a gentleman who experienced recurrent diarrhea and PR bleeding, initially managed as infective colitis but subsequently diagnosed with Crohn's disease following colonoscopy and histopathological confirmation. He was treated successfully with corticosteroids for induction and azathioprine for maintenance therapy. This case emphasizes the need to consider Crohn's disease as a differential diagnosis in patients with chronic diarrhea and PR bleeding, particularly in regions where infectious causes are more prevalent, and may delay diagnosis.

Keywords: diarrhea, disease, bleeding.

Contributions:
TH - Conception, Design
- Acquisition, Analysis, Interpretation
AA - Drafting
TH SA- Critical Review

All authors approved the final version to be published & agreed to be accountable for all aspects of the work.

Conflicts of Interest: None
Financial Support: None to report
Potential Competing Interests: None to report

Institutional Review Board Approval
1057/RTH-RWP
22-02-2024
Holy Family Hospital, Rawalpindi

Review began 13/10/2025
Review ended 15/01/2026
Published 31/01/2026

How to cite this article: Hussain T, Abbas A, Ahmed S, Umar M. Exploring the Intricacies and Clinical Perspectives of Chronic Diarrhea: A Case Report. JRMC. 2026 Feb. 14;1(1).

https://doi.org/10.37939/jrmc.v1i1.3192

Introduction

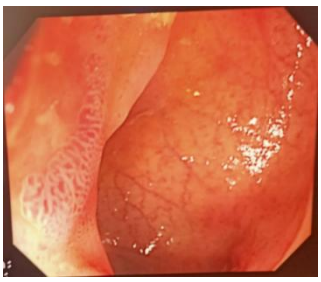
Crohn's disease (CD) is one of the two main forms of inflammatory bowel disease (IBD). It may involve any part of the gastrointestinal tract, most often the terminal ileum and proximal colon, and is characterized by transmural inflammation, skip lesions, strictures, and fistulae.[1]
The global incidence of CD continues to rise, particularly in newly industrialized regions of Asia, the Middle East, and South America.[2] In Western countries, incidence ranges from 0.1 to 16 per 100,000 person-years, but recent studies indicate a growing burden in South Asia.[3]
The pathogenesis is multifactorial, involving genetic susceptibility, mucosal immune dysregulation, environmental triggers, and gut microbiota.[4] Genome-wide association studies have identified key genes including NOD2, ATG16L1, and IL23R, highlighting the role of innate immunity and microbial recognition.[5]
Diagnosis remains challenging in infection-endemic settings where CD may mimic intestinal tuberculosis, infectious colitis, or ulcerative colitis. Colonoscopy typically demonstrates longitudinal ulcers, cobblestoning, skip lesions, and strictures, supported by cross-sectional imaging and histology.[6] Most patients develop progressive disease requiring immunosuppressants or biologics, while only 20–30% follow an indolent course.[8]

Case Presentation

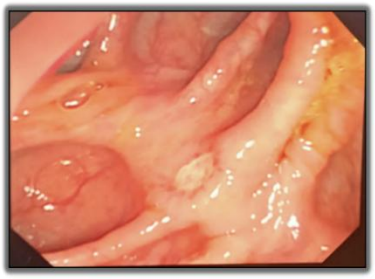
A 44-year-old gentleman from Azad Kashmir presented to the gastroenterology outpatient clinic with a 2-year history of chronic diarrhea. The diarrhea was of small volume, loose in consistency (Bristol stool scale type 6–7), occurring intermittently, and occasionally mixed with blood. Over the past 3 months, he also developed abdominal pain. The pain was dull, continuous throughout the day, and significantly worsened after meals, reaching an intensity of 7–8/10 on the pain scale. The diarrheal episodes were frequently associated with urgency, tenesmus, and abdominal bloating. He denied any history of weight loss, extraintestinal manifestations such as arthralgia, aphthous ulcers, or skin lesions. He had sought medical care at several centers in Saudi Arabia and local hospitals, where he was managed empirically with antibiotics, antispasmodics, and probiotics, but his symptoms persisted without improvement. On examination, he appeared thin and lean, with a BMI of 24 kg/m². There was mild tenderness in the right iliac fossa, but no guarding or signs of peritonism. Perianal inspection revealed a small skin tag, with no evidence of abscess or fistula.

Investigations

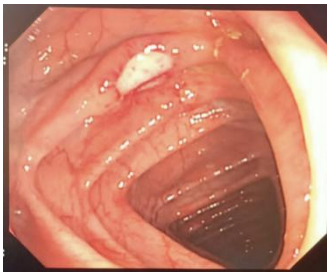
Blood tests: Hemoglobin 13.3 g/dL, MCV 74 fL, CRP 36 mg/L, ESR 52 mm/hr. LFTs and renal functions were normal. His TSH, Anti-Ttg (IgA), ANA c, and p- ANCA were also normal, but ASCA was positive, with Fecal calprotectin 250ug/g and Vitamin B12 150pg/dL and Folic acid 2.1ng/mL.
Stool culture: It was negative for Clostridium difficile toxin.
Colonoscopy: There were multiple deep ulcers with surrounding erythema in the terminal ileum, ascending colon, and hepatic flexure with intervening normal-looking mucosa, but the transverse, descending, sigmoid colon, and rectum were normal.



Terminal Ileum

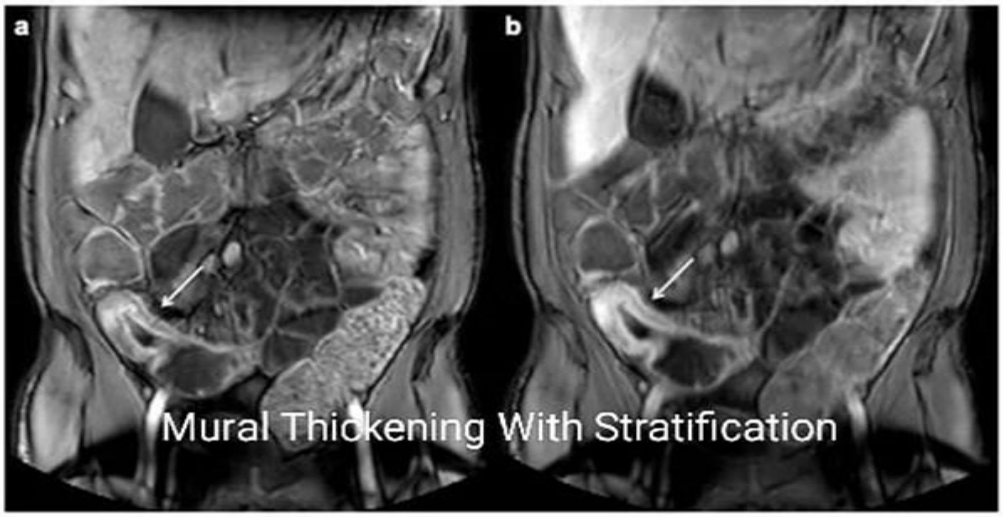


Cecum



Ascending Colon

Histopathology: There was focal chronic inflammation with mild cryptitis and crypt abscesses, ulceration, and no granulomas.
Imaging: MR enterography revealed jejunal wall segmental thickening with delayed mural enhancement, suggestive of IBD.



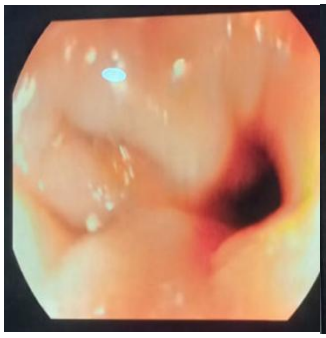
Crohn's disease (Montreal classification A2 L3 B1).

Treatment

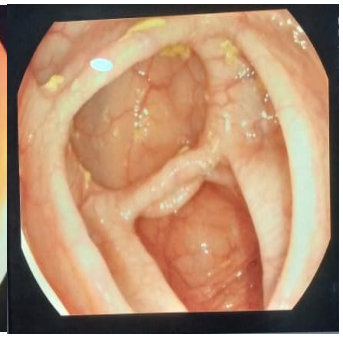
The patient was put on budesonide 9mg once daily orally for 8 weeks; the patient's symptoms were completely resolved within two months. He started azathioprine 2 mg/kg for maintenance therapy. Cobalamin, folic acid, and vitamin D deficiency were also replaced.

Outcome And Follow-Up

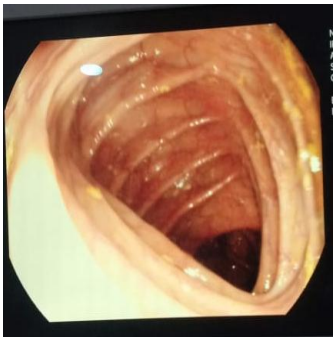
After 8 weeks, the patient had complete resolution of diarrhea, bleeding, and abdominal pain, normalization of CRP (6mg/L) and fecal calprotectin (50 µg/g), and repeat colonoscopy at 12 weeks showed mucosal healing. He remained in clinical remission at 12 months on azathioprine monotherapy.



Terminal Ileum



Cecum



Ascending Colon

Discussion

This case illustrates the diagnostic challenges in regions endemic for infectious diseases. The absence of granulomas on histology initially raised suspicion for an infective etiology. However, the chronic nature of symptoms, persistently elevated fecal calprotectin, positive ASCA serology, and colonoscopic evidence of skip lesions were more typical of Crohn's disease (CD). Granulomas are identified in only 15–65% of CD biopsies and are not essential for diagnosis.[5]

Endoscopic findings such as aphthous ulcers, longitudinal fissures, skip lesions, and ileocecal involvement are highly characteristic of CD.[6,7] Radiological features, including segmental mural thickening and delayed mural enhancement on MR enterography, provide further diagnostic support.[9,10]

Management of CD has shifted considerably in recent decades. Corticosteroids were once the cornerstone of induction therapy, but the introduction of biologics has transformed treatment strategies.[12] Current guidelines advocate an individualized approach: budesonide is recommended for mild-to-moderate ileocecal disease, while systemic corticosteroids, immunomodulators, and biologics (anti-TNF, anti-integrin, and anti-IL-12/23 agents) are reserved for more extensive or refractory cases.[11]

Therapeutic objectives now follow a treat-to-target model, moving beyond symptom relief. This includes short-term clinical remission, intermediate biomarker reduction, and long-term mucosal healing demonstrated by colonoscopy.[13,14] Mucosal healing is associated with reduced hospitalization, need for surgery, and colorectal cancer risk. In our patient with mild ileocolonic disease, budesonide monotherapy with dietary support achieved both clinical and endoscopic remission. Early recognition and timely therapy in such cases are vital to prevent disease progression and long-term complications.

Learning Points:

- Crohn's disease should be suspected in chronic diarrhoea even in infection-endemic regions.
- Endoscopic and radiological features are more reliable than granulomas for diagnosis.
- Budesonide is effective in mild ileocolonic disease.
- Treat-to-target strategies improve long-term outcomes.

Author Information

1. HOD/Associate Professor, Holy Family Hospital, Rawalpindi 2. Senior Registrar, Holy Family Hospital, Rawalpindi 3. Assistant Professor, Holy Family Hospital, Rawalpindi 4. Vice Chancellor, Rawalpindi Medical University, Rawalpindi.
Corresponding author: Dr. Tanveer Hussain, drtanveer_@hotmail.com

References

1. Torres J, Mehandru S, Colombel JF, Peyrin-Biroulet L. Crohn's disease. Lancet. 2017;389:1741–55.https://doi.org/10.1016/S0140-6736(16)31711-1
2. Ng SC, Shi HY, Hamidi N, Underwood FE, Tang W, Benchimol EI, et al. Worldwide incidence and prevalence of inflammatory bowel disease in the 21st century: a systematic review of population-based studies. The Lancet. 2017 Dec 23;390(10114):2769-78.https://doi.org/10.1016/S0140-6736(17)32448-0
3. Alatab S, Sepanlou SG, Ikuta K, Vahedi H, Bisignano C, Safiri S, et al. The global, regional, and national burden of inflammatory bowel disease in 195 countries and territories, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. The Lancet gastroenterology & hepatology. 2020 Jan 1;5(1):17-30. https://doi.org/10.1016/S2468-1253(19)30333-4
4. Khor B, Gardet A, Xavier RJ. Genetics and pathogenesis of inflammatory bowel disease. Nature. 2011 Jun 16;474(7351):307-17. https://doi.org/10.1038/nature10209
5. Gomollón F, Dignass A, Annese V, Tilg H, Van Assche G, Lindsay JO, et al. 3rd European evidence-based consensus on the diagnosis and management of Crohn's disease 2016: part 1: diagnosis and medical management. Journal of Crohn's and Colitis. 2017 Jan 1;11(1):3-25.https://doi.org/10.1093/ecco-jcc/jjw168
6. Yeshi K, Ruscher R, Hunter L, Daly NL, Loukas A, Wangchuk P. Revisiting inflammatory bowel disease: pathology, treatments, challenges and emerging therapeutics including drug leads from natural products. Journal of clinical medicine. 2020 Apr 28;9(5):1273.https://doi.org/10.3390/jcm9051273
7. Ricciuto A, Griffiths AM. Clinical value of fecal calprotectin. Crit Rev Clin Lab Sci. 2019 Aug;56(5):307-320. https://doi.org/10.1080/10408363.2019.1619159.
8. Alyami A, Hoad CL, Tench C, Bannur U, Clarke C, Latief K, et al. Quantitative magnetic resonance imaging in perianal Crohn's disease at 1.5 and 3.0 T: a feasibility study. Diagnostics. 2021 Nov 17;11(11):2135. https://doi.org/10.3390/diagnostics11112135
9. Torres J, Mehandru S, Colombel JF, Peyrin-Biroulet L. Crohn's disease. The Lancet. 2017 Apr 29;389(10080):1741-55. https://doi.org/10.1016/S0140-6736(16)31711-1
10. Lichtenstein GR, Loftus EV, Isaacs KL, Regueiro MD, Gerson LB, Sands BE. ACG clinical guideline: management of Crohn's disease in adults. Official journal of the American College of Gastroenterology| ACG. 2018 Apr 1;113(4):481-517.https://doi.org/10.1038/ajg.2018.27
11. Colombel JF, Sandborn WJ, Reinisch W, Mantzaris GJ, Kornbluth A, Rachmilewitz D, et al. Infliximab, azathioprine, or combination therapy for Crohn's disease. New England journal of medicine. 2010 Apr 15;362(15):1383-95. https://doi.org/**10.1056/NEJMoa0904492**
12. Cucchiara S, D'Arcangelo G, Isoldi S, Aloi M, Stronati L. Mucosal healing in Crohn's disease: new insights. Expert Review of Gastroenterology & Hepatology. 2020 May 3;14(5):335-45. https://doi.org/10.1080/17474124.2020.1759416
13. Peyrin-Biroulet L, Sandborn W, Sands BE, Reinisch W, Bemelman W, Bryant RV, et al. Selecting therapeutic targets in inflammatory bowel disease (STRIDE): determining therapeutic goals for treat-to-target. Official journal of the American College of Gastroenterology| ACG. 2015 Sep 1;110(9):1324-38.https://di.org/10.1038/ajg.2015.233
14. Torres J, Bonovas S, Doherty G, Kucharzik T, Gisbert JP, Raine T, et al. ECCO guidelines on therapeutics in Crohn's disease: medical treatment. Journal of Crohn's and Colitis. 2020 Jan 1;14(1):4-22.https://doi.org/10.1093/ecco-jcc/jjz180