Annals of Clinical and Medical Case Reports[R] ISSN 2639-8109 | Volume 16

Case Report Open Access

Progressive Terminal Ileitis with Stenotic Symptoms During Adalimumab Therapy for Rheumatoid Arthritis: A Case Report of Suspected Paradoxical Inflammatory Bowel Disease

Raggi Matthias* **and Al Eissa Ahmad**

Department of General and Visceral Surgery, Sana Karl-Olga-Krankenhaus Stuttgart, Germany

***Corresponding author:**

Raggi Matthias,
Department of General and Visceral Surgery,
Sana Karl-Olga-Krankenhaus Stuttgart,
Germany

Received: 01 July 2026
Accepted: 16 July 2026
Published: 18 July 2026
J Short Name: ACMCR

Citation:

Raggi Matthias, Progressive Terminal Ileitis with Stenotic Symptoms During Adalimumab Therapy for Rheumatoid Arthritis: A Case Report of Suspected Paradoxical Inflammatory Bowel Disease. Ann Clin Med Case Rep® 2026; V16(3): 1-2

1. Abstract

Tumour necrosis factor-alpha (TNF-α) inhibitors are widely used in the treatment of rheumatologic disorders and constitute a cornerstone of therapy for inflammatory bowel disease (IBD). Paradoxically, de novo intestinal inflammation has been increasingly recognized as a rare adverse effect of anti-TNF therapy. We report the case of a 79-year-old woman with rheumatoid arthritis receiving adalimumab biosimilar therapy who developed isolated terminal ileitis with progressive stenotic symptoms. Despite systemic corticosteroid treatment, both clinical and sonographic findings worsened, resulting in prestenotic dilatation of the small bowel. This case highlights the diagnostic and therapeutic challenges associated with paradoxical IBD induced by anti-TNF agents.

2. Introduction

Biologic therapies have significantly improved outcomes in patients with rheumatoid arthritis (RA) [1]. Among these agents, TNF-α inhibitors are highly effective in controlling systemic inflammation and are also routinely used to treat Crohn's disease and ulcerative colitis [2-5]. However, paradoxical inflammatory reactions, including the development of de novo IBD, have been increasingly reported in patients receiving anti-TNF therapy for non-gastrointestinal indications. Recognition of this uncommon adverse event is important given the expanding use of biologic therapies.

3. Case Presentation

A 79-year-old woman with a history of rheumatoid arthritis treated with adalimumab biosimilar (Hyrimoz®, 40 mg every three weeks) was admitted because of recurrent abdominal pain and long-standing constipation. She reported several months of obstructive bowel symptoms characterized by bloating, excessive flatulence, frequent unsuccessful attempts at defecation, and minimal stool passage [6-9].

Her medical history was notable for sigmoid diverticulitis treated conservatively in January 2025 and arterial hypertension. She had no known history of inflammatory bowel disease.

Physical examination revealed a hemodynamically stable, afebrile patient in good general condition. Abdominal examination demonstrated distension and diffuse tenderness, most pronounced in the lower abdomen, without signs of peritonitis.

Initial abdominal ultrasonography showed isolated inflammation of the terminal ileum with bowel wall thickening up to 8 mm and marked submucosal edema. The inflammatory segment extended to the ileocecal valve, while the cecum appeared unaffected. No proximal small bowel dilatation or free intraperitoneal fluid was observed [10-12].

Ileocolonoscopy revealed a short segment, approximately 5cm in length, of inflamed terminal ileum with superficial ulcerations covered by whitish exudates. A sharp transition to normal mucosa was noted proximally. The colon was otherwise unremarkable except for a few diverticula. Histopathological examination of ileal biopsies was non-specific and did not establish a definitive diagnosis.

The patient received a single intravenous dose of 100 mg prednisolone, resulting in significant symptomatic improvement. She was subsequently discharged on oral prednisolone 50 mg daily for one week, and further administration of adalimumab was withheld.

At follow-up six days later, the patient reported worsening obstructive symptoms despite corticosteroid therapy. Repeat ultrasonography demonstrated progression of the inflammatory lesion, with bowel wall thickening increasing to 9 mm. In addition, new prestenotic dilatation of the proximal ileum and increased small bowel filling were observed, indicating evolving

luminal narrowing. No free fluid or complete bowel obstruction was present.

Given the progression despite corticosteroid treatment, the absence of alternative etiologies, and the ongoing anti-TNF therapy, a paradoxical inflammatory bowel disease-like reaction was considered the most likely diagnosis. Following multidisciplinary discussion between gastroenterologists and rheumatologists, the patient was referred for further inpatient evaluation, as surgical intervention might become necessary if progression continued.

4. Discussion

Paradoxical inflammatory bowel disease is an uncommon but increasingly recognized complication of anti-TNF therapy. Although TNF inhibitors are effective treatments for Crohn's disease, cases of new-onset IBD occurring during treatment for rheumatologic and dermatologic disorders have been reported.

The pathophysiological mechanisms remain incompletely understood. Proposed explanations include cytokine imbalance, dysregulation of interferon pathways, and altered mucosal immune responses. Clinical manifestations may closely resemble Crohn's disease and range from mild inflammatory lesions to severe complications such as strictures, fistulas, and bowel obstruction.

Several findings in this case support a drug-induced etiology. The patient had no previous diagnosis of IBD, the inflammatory changes were localized to a short segment of the terminal ileum, histology was non-specific, and symptoms developed during long-term anti-TNF treatment. Particularly noteworthy was the rapid progression toward a stenotic phenotype despite systemic corticosteroid therapy.

Differential diagnoses included Crohn's disease, infectious ileitis, ischemic enteritis, and nonsteroidal anti-inflammatory drug-induced enteropathy. However, the clinical presentation and temporal association with anti-TNF therapy favoured a paradoxical inflammatory reaction.

5. Conclusion

This case illustrates a probable paradoxical anti-TNF-induced ileitis presenting as progressive terminal ileal inflammation with evolving stenosis in a patient treated for rheumatoid arthritis. Clinicians should be aware that new gastrointestinal symptoms arising during anti-TNF therapy may represent paradoxical intestinal inflammation. Early recognition and interdisciplinary management are essential, particularly when progressive obstructive symptoms raise the possibility of surgical intervention.

Learning Points

• Anti-TNF agents may rarely induce paradoxical inflammatory bowel disease.

• New-onset terminal ileitis in patients receiving anti-TNF therapy should prompt consideration of a drug-related adverse event.

• Clinical improvement after corticosteroid administration does not necessarily predict favorable disease progression.

• Progressive bowel wall thickening with prestenotic dilatation suggests developing luminal narrowing and requires close monitoring.

• Multidisciplinary collaboration between gastroenterologists, rheumatologists, and surgeons is crucial for optimal management.

References

1. Fiorino G, Danese S, Pariente B, Allez M. Paradoxical immune-mediated inflammation in inflammatory bowel disease patients receiving anti-TNF-α agents. Autoimmun Rev. 2014; 13(1): 15-19.
2. Toussirot É, Houvenagel E, Goëb V, Fouache D, Martin A. Development of inflammatory bowel disease during anti-TNF-α therapy for inflammatory rheumatic disease: a nationwide series. Joint Bone Spine. 2012; 79(5): 457-463.
3. Toussirot É, Wiegering V. Paradoxical adverse events under TNF-α blocking agents and other biological agents given for chronic immune-mediated diseases: an analytical and comprehensive overview. RMD Open. 2016; 2: e000239.
4. O'Toole A, Lucci M, Korzenik J. Inflammatory bowel disease provoked by etanercept: report of 443 possible cases combined from an IBD referral center and the FDA. Dig Dis Sci. 2016; 61(6): 1772-1774.
5. Rahier JF, Magro F, Abreu C, Armuzzi A, Ben-Horin S. Second European evidence-based consensus on the prevention, diagnosis and management of opportunistic infections in inflammatory bowel disease. J Crohns Colitis. 2014; 8(6): 443-468.
6. Cleynen I, Vermeire S. Paradoxical inflammation induced by anti-TNF agents in patients with IBD. Nat Rev Gastroenterol Hepatol. 2012; 9(9): 496-503.
7. Kochar B, Herfarth N, Mamula P, Navaneethan U. Systematic review: paradoxical adverse events associated with anti-TNF therapy in inflammatory bowel disease. Aliment Pharmacol Ther. 2018; 48(7): 696-709.
8. Danese S, Fiorino G, Peyrin-Biroulet L. Positioning therapies in ulcerative colitis. Clin Gastroenterol Hepatol. 2020; 18(6): 1280-1290.
9. Ramos-Casals M, Brito-Zerón P, Muñoz S, Soto MJ. A systematic review of the autoimmune diseases induced by biological agents. Expert Opin Drug Saf. 2008; 7(6): 663-693.
10. Toussirot É, Aubin F. Paradoxical reactions under TNF-α blocking agents and other biological agents given for chronic immune-mediated diseases: an update. Expert Rev Clin Immunol. 2016; 12(5): 577-594.
11. Vavricka SR, Rogler G, Pittet V, Michetti P, Felley C. Systematic evaluation of risk factors for diagnostic delay in inflammatory bowel disease. Inflamm Bowel Dis. 2012; 18(3): 496-505.
12. Harbord M, Eliakim R, Bettenworth D, Karmiris K, Katsanos K. Third European evidence-based consensus on diagnosis and management of Crohn's disease. Part 2: current management. J Crohns Colitis. 2017; 11(1): 3-25.