Case Report

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Secukinumab-Induced Inflammatory Bowel Disease: A Case Report

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ABSTRACT

Secukinumab, an interleukin-17A (IL-17A) inhibitor, is a biologic agent widely used for autoimmune conditions such as psoriasis, psoriatic arthritis, and hidradenitis suppurativa. While effective for these disorders, IL-17A inhibition has been increasingly associated with paradoxical gastrointestinal inflammation, including the development or exacerbation of inflammatory bowel disease (IBD).

We describe a 43-year-old woman with hidradenitis suppurativa who developed new-onset ulcerative colitis after approximately eight months of treatment with secukinumab. She presented with bloody diarrhea and abdominal pain. Laboratory evaluation revealed elevated inflammatory markers, including an erythrocyte sedimentation rate of 42 mm/hr, a C-reactive protein level of 18 mg/L, and fecal calprotectin of 306 µg/g. Colonoscopy demonstrated Mayo 2 inflammation confined to the rectosigmoid colon, and biopsy confirmed moderate active chronic colitis with cryptitis and crypt architectural distortion. Secukinumab was discontinued, and the patient was treated with prednisone 40 mg daily, resulting in rapid and complete resolution of symptoms.

This case highlights a paradoxical immune-mediated complication of IL-17A inhibition. Although secukinumab is highly effective for dermatologic and rheumatologic diseases, clinicians should be aware of its potential to induce or unmask IBD. Early recognition of gastrointestinal symptoms and prompt discontinuation of the agent are essential to prevent disease progression.

Keywords

Secukinumab, Cosentyx™, Inflammatory bowel disease, Ulcerative colitis, Hidradenitis suppurativa.

Introduction

Interleukin-17A (IL-17A) is a pro-inflammatory cytokine that plays a central role in autoimmune diseases such as psoriasis, psoriatic arthritis, and hidradenitis suppurativa [1,2]. Secukinumab, a fully human monoclonal antibody that selectively inhibits IL-17A, has shown remarkable therapeutic benefit in these conditions by reducing systemic and tissue-specific inflammation [3]. However, recent reports have described a paradoxical effect of IL-17 inhibition—namely, the induction or exacerbation of inflammatory bowel disease (IBD) [4].

This paradox arises from the dual nature of IL-17A in immune regulation. Although IL-17A contributes to inflammation in the skin and joints, it also serves a protective function in the gastrointestinal tract by maintaining epithelial barrier integrity and coordinating mucosal defense [5]. Its inhibition can therefore disrupt intestinal homeostasis and lead to inflammation in susceptible individuals [6].

We present a case of biopsy-confirmed ulcerative colitis that developed in a patient treated with secukinumab for hidradenitis suppurativa. This report underscores the importance of recognizing this rare but clinically significant adverse effect of IL-17A blockade and contributes to the growing literature on biologic-induced intestinal inflammation.

Case Presentation

A 43-year-old woman with a history of hidradenitis suppurativa,

asthma, bipolar disorder, schizoaffective disorder, and morbid obesity presented for evaluation of rectal bleeding and chronic diarrhea. She was a former smoker, consumed alcohol occasionally, and denied illicit drug use. Her family history was negative for inflammatory bowel disease, colon cancer, or autoimmune conditions.

In 2016, she had undergone a screening colonoscopy for rectal bleeding that revealed benign polyps and internal hemorrhoids. A repeat upper endoscopy and colonoscopy in June 2024 demonstrated mild chronic gastritis and a benign rectal polyp without evidence of inflammatory bowel disease, celiac disease, or Barrett's esophagus.

The patient was started on secukinumab (Cosentyx™) 300 mg subcutaneously monthly for hidradenitis suppurativa in August 2024. Approximately eight months after initiating therapy, she developed bloody, mucous diarrhea accompanied by lower abdominal cramping. Laboratory evaluation in April 2025 revealed elevated inflammatory markers, including an ESR of 42 mm/hr, CRP of 18 mg/L, and fecal calprotectin of 306 µg/g. Hemoglobin was 12.1 g/dL, and renal function was mildly reduced with a creatinine level of 1.30 mg/dL and eGFR of 52 mL/min/1.73 m². Serologic testing for HIV, hepatitis B and C, and tuberculosis was negative.

An esophagogastroduodenoscopy (EGD) and colonoscopy performed on May 13, 2025, showed normal duodenal and terminal ileal mucosa, with Mayo 2 inflammation localized to the rectosigmoid region (Figure 1). The inflamed area demonstrated erythema, friability, and superficial ulceration, while the remainder of the colon appeared normal. Histopathologic examination confirmed moderate active chronic colitis with cryptitis and crypt architectural distortion, consistent with ulcerative colitis. No granulomas or dysplasia were observed.

Given the temporal relationship between the onset of symptoms and secukinumab therapy, the absence of prior IBD history, and rapid clinical improvement following drug withdrawal, secukinumab-induced colitis was diagnosed. The patient was treated with prednisone 40 mg daily, resulting in complete resolution of diarrhea and hematochezia within two weeks. She remained on

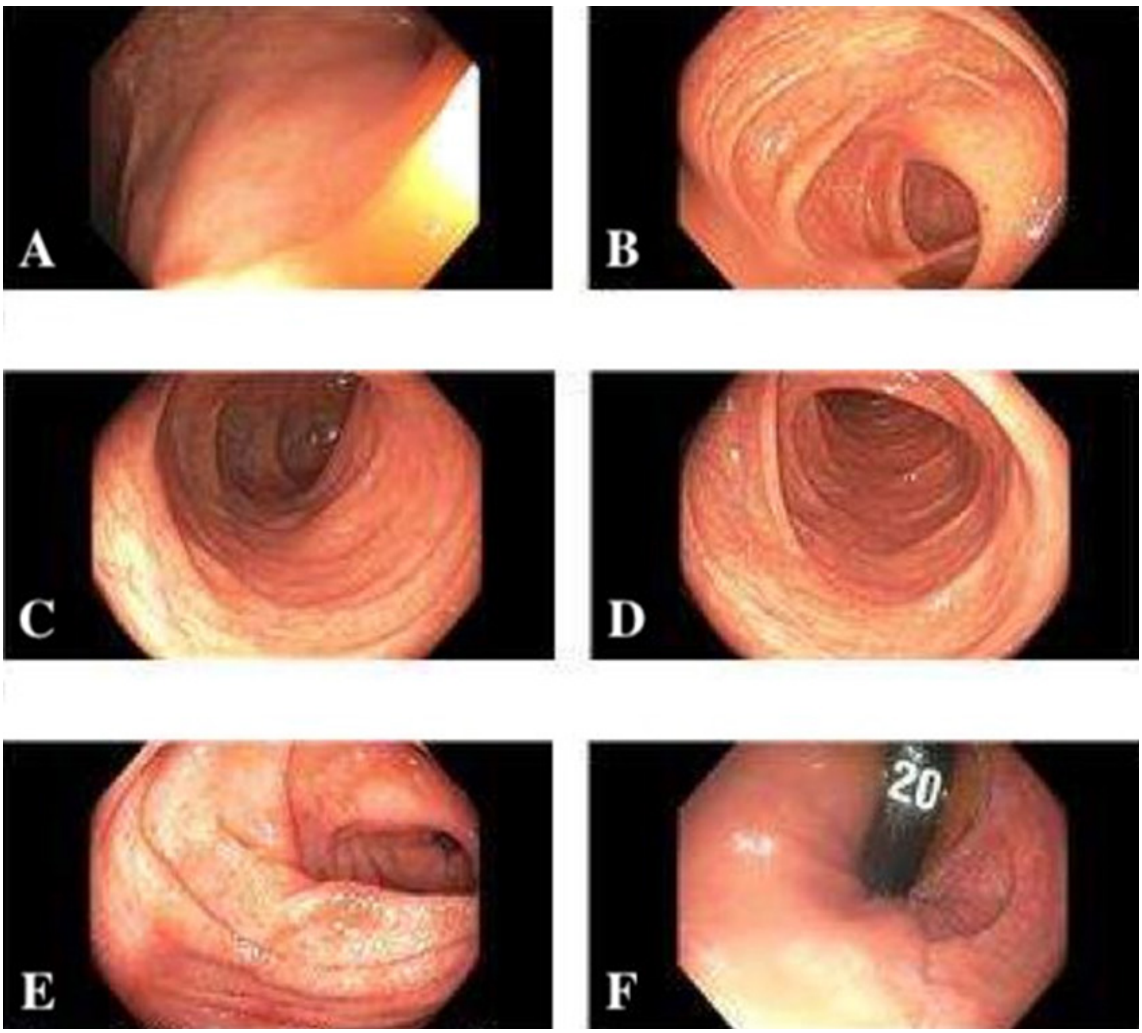

Figure 1: Colonoscopy images showing normal-appearing (A) cecum, (B) ileocecal valve, (C) colon, and (D) colon. (E) Recto-sigmoid region demonstrating Mayo 2 inflammation. (F) Retroflexion in the cecum revealing no additional lesions.

omeprazole 40 mg twice daily for her gastritis. Secukinumab was permanently discontinued, and dermatology planned to transition her to adalimumab (Humira™) for management of hidradenitis suppurativa. At one-month follow-up, the patient remained asymptomatic, and a repeat colonoscopy was scheduled six months after initiating adalimumab.

Discussion

This case illustrates the rare but clinically significant occurrence of secukinumab-induced ulcerative colitis, an increasingly recognized paradoxical immune-mediated event. Although IL-17A inhibitors are potent anti-inflammatory agents in diseases such as psoriasis and hidradenitis suppurativa, their use can paradoxically provoke intestinal inflammation [3].

The mechanism underlying this phenomenon is complex and reflects the dual, context-dependent nature of IL-17A in immune regulation. In the gastrointestinal tract, IL-17A serves not only as an inflammatory mediator but also as a crucial defender of mucosal integrity[3]. It promotes the expression of tight-junction proteins such as claudins and occludin, supports the secretion of antimicrobial peptides, and facilitates immunoglobulin A (IgA) transport across the mucosal epithelium. These functions are essential for maintaining epithelial barrier integrity and preventing microbial translocation. When IL-17A is inhibited, these protective processes are disrupted, leading to increased intestinal permeability, altered microbial composition, and activation of mucosal immune pathways that promote inflammation.

Furthermore, IL-17A inhibition disturbs the balance of mucosal cytokine networks and immune cell populations[4]. In the absence of IL-17A signaling, compensatory upregulation of other pro-inflammatory mediators, including interleukin-6, interleukin-22, and tumor necrosis factor-alpha, has been observed. This cytokine imbalance may foster a chronic inflammatory milieu within the intestinal mucosa. Experimental models have shown that IL-17A blockade can expand RORγt-positive innate lymphoid cells and promote a colitogenic environment [4]. These findings collectively suggest that IL-17A, though inflammatory in certain tissues, functions protectively in the gut—a tissue-specific paradox that helps explain why IL-17 inhibitors worsen or unmask IBD rather than ameliorate it.

Clinical trials lend further support to this paradox. In studies evaluating IL-17 inhibitors for Crohn's disease, such as those involving secukinumab and brodalumab, patients experienced worsening of intestinal inflammation, leading to early termination of these trials [5]. Post-marketing surveillance and case reports have since confirmed similar occurrences across multiple IL-17A inhibitors, with the majority attributed to secukinumab use [6].

In the present case, the absence of previous gastrointestinal pathology, the close temporal relationship between secukinumab initiation and the onset of symptoms, and the patient's rapid recovery following discontinuation of the drug strongly suggest a causal relationship. While it remains uncertain whether IL-17A inhibition induces new IBD or unmasks subclinical disease, the pathophysiologic mechanisms likely overlap, involving impaired barrier function and dysregulated mucosal immunity in genetically or immunologically predisposed individuals.

Clinicians prescribing IL-17 inhibitors should be aware of this potential complication and maintain a high index of suspicion for new gastrointestinal symptoms such as diarrhea, abdominal pain, or hematochezia. Baseline assessment of bowel habits and inflammatory markers may be prudent, particularly in patients with a family history of IBD or prior unexplained gastrointestinal symptoms. Prompt discontinuation of the drug and initiation of corticosteroid therapy can lead to resolution of inflammation, as demonstrated in this patient. Alternative biologic therapies, such as anti-TNF or IL-23 inhibitors, may be considered for continued management of the underlying autoimmune disease.

Conclusion

Secukinumab and other IL-17A inhibitors have revolutionized the management of chronic autoimmune diseases but can paradoxically precipitate inflammatory bowel disease by disrupting the gut's mucosal barrier and immune homeostasis. This case reinforces the importance of clinical vigilance, early recognition of gastrointestinal symptoms, and timely intervention. The paradoxical effects of IL-17A inhibition remind clinicians that cytokine networks are context-dependent and that immune pathways protective in one tissue may be pathogenic in another. Early identification and drug discontinuation are critical for preventing long-term complications and ensuring patient safety.

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