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CASE REPORT

Resolution of Long-Standing Irritable Bowel Syndrome Symptoms After Treatment of Psoriasis With Risankizumab

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Psoriasis is a chronic immune-mediated inflammatory disease associated with systemic comorbidities, including gastrointestinal disorders. Emerging evidence supports a bidirectional gut-skin axis, with shared immunologic pathways involving the interleukin (IL)-23/IL-17 axis. Irritable bowel syndrome (IBS), traditionally considered a functional disorder, has also been linked to low-grade inflammation and cytokine dysregulation. We report a case of a 67-year-old man with moderate plaque and inverse psoriasis and longstanding IBS with diarrhea (IBS-D) who experienced complete resolution of gastrointestinal symptoms following treatment with risankizumab, an IL-23 inhibitor. Improvement in IBS symptoms began within weeks of therapy initiation and was sustained at follow-up, alongside complete skin clearance. No other changes in medications or lifestyle were identified. This case highlights a potential role of IL-23–mediated inflammation in IBS pathophysiology and suggests that targeted inhibition of the IL-23/IL-17 axis may benefit select patients with concurrent dermatologic and gastrointestinal disease. **KEYWORDS:** Psoriasis, IL-23, IBS, irritable bowel syndrome

Psoriasis is a chronic, immune-mediated inflammatory skin disorder characterized by keratinocyte hyperproliferation and dysregulated activation of the interleukin (IL) 23/T helper 17 (T_H17) axis.[1] Growing evidence supports a bidirectional gut-skin connection, whereby dysbiosis of the gut microbiome influences systemic inflammation, and cutaneous inflammation from psoriasis may likewise impact gut function.[1–4]

Although the etiology of irritable bowel syndrome (IBS) is poorly understood and it has traditionally been classified as a functional gastrointestinal disorder, growing evidence supports a multifactorial pathogenesis with gut-brain dysfunction and cytokine-driven mechanisms.[5–7] Studies have shown elevated mucosal levels of IL-6, IL-8, and IL-23 in IBS, suggesting a pathogenic role of low-grade immune activation.[5–9] There is potential overlap between psoriasis and IBS, with IL-23/T_H17 axis dysregulation, alterations in gut microbial composition, and neuro-immune communication via the gut-brain-skin axis.[5–7,10]

Risankizumab, a monoclonal antibody targeting the p19 subunit of IL-23, is approved by the United States Food and Drug Administration for the treatment of moderate-to-severe plaque psoriasis, psoriatic arthritis, Crohn's disease, and ulcerative colitis.[1,10–13] Unlike inflammatory bowel disease (IBD), IBS may involve subtle immune dysregulation and barrier dysfunction, with possible involvement of cytokine pathways such as IL-23.[2] By selectively inhibiting IL-23 signaling, risankizumab modulates T-cell activation and cytokine release, alleviating skin inflammation in psoriasis and restoring intestinal immune balance in IBD.[10,14,15]

This case report describes a 67-year-old man with plaque and inverse psoriasis who experienced complete resolution of longstanding IBS symptoms following initiation of risankizumab therapy. The observed clinical response highlights a potential immunologic link between IL-23 blockade and improvement in IBS gastrointestinal symptoms, suggesting that targeted inhibition of the IL-23/IL-17 axis may benefit a subset of patients presenting with concurrent psoriatic and functional bowel symptoms.[10,14]

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CASE DESCRIPTION

A 67-year-old man with a past medical history of anxiety disorder, atrial fibrillation, hypertension, hearing loss, and IBS with diarrhea (IBS-D) presented to our dermatology clinic with plaque psoriasis involving the face, scalp, right leg, and trunk and inverse psoriasis on bilateral axillae and genitalia. Prior to systemic therapy, the patient had 10% body surface area (BSA) involvement, moderate disease based on physician global assessment (PGA), and an itch numerical rating scale (NRS) of 6. He was a nonsmoker and had previously failed topical therapies including roflumilast 0.3% cream, tapinarof 1% cream, clobetasol 0.05% ointment, and fluocinonide 0.05% solution.

After normal baseline lab tests were confirmed, the patient was initiated on deucravacitinib. At 2-month follow-up, the patient reported initial improvement before flaring again, with 10% BSA, moderate PGA, and NRS 7. He continued deucravacitinib for an additional 2 months, and at 4-month follow up, given suboptimal response, therapy was switched to risankizumab.

At his most recent visit, 4 months after initiation of risankizumab, the patient demonstrated substantial improvement in psoriasis, with 0% BSA and clear PGA, NRS 0, along with complete resolution of IBS symptoms. He described a progressive improvement in gastrointestinal symptoms beginning approximately 2 to 3 weeks after starting riskanikuzmab, with no recurrence of diarrhea episodes since starting risankizumab. He reported firmer stool consistency, and by 4 weeks, normalization of bowel movements to 1 to 2 formed stools per day.

The patient first began experiencing gastrointestinal symptoms as an adolescent. Prior to treatment with risankizumab, he had experienced 4 to 5 watery or loose bowel movements per day, accompanied by pain, bloating, and cramping. The patient had no family history IBD and had 3 colonoscopies between 2007 and 2022, without evidence of inflammation. No other new medications or lifestyle changes were noted within the timeframe of starting risankizumab through the time of follow up.

Overall, the patient reported high satisfaction with risankizumab, citing not only near clearance of psoriasis but also marked improvement in his long-standing gastrointestinal symptoms particularly impacted his quality of life.

DISCUSSION

The coexistence of psoriasis and gastrointestinal dysfunction highlights the complex relationship between cutaneous and intestinal immune responses, with the gut-brain-skin axis serving as a central mechanistic link.[1] Psoriasis is now recognized as a systemic inflammatory disease, with increased prevalence of rheumatic, metabolic, cardiovascular, psychiatric, and gastrointestinal comorbidities, including gut dysbiosis, IBD, and IBS.[1,2,16] Evidence supporting the gut-skin axis in IBD includes shared genetic risk factors, overlapping cytokine profiles, and the role of gut dysbiosis in systemic immune activation.[1,2] The immunologic overlap between psoriasis and IBD is largely mediated by the IL-23/T_H17 axis, a pathway that is well established in psoriasis pathogenesis, with activation leading to increased production of proinflammatory cytokines such as IL-17, IL-22, and tumor necrosis factor α.[10] The role of the gut-skin axis in IBS has been less explored; however, a common mechanistic link may be gut dysbiosis, which is increasingly recognized in both psoriasis and IBS.[3,4,17]

The T_H17 pathway plays an integral and protective role in maintaining gut mucosal barrier function and gut homeostasis.[10,15,18] In psoriasis, gut dysbiosis promotes "leaky gut syndrome" through disruption of the intestinal barrier and activation of inflammatory responses, which is thought to occur through translocation of bacteria, microbial products, and pro-inflammatory signaling molecules into the systemic circulation and ultimately the skin.[4,16] This "leaky gut" phenomenon has also been reported in IBS, potentially due to intestinal epithelial and vascular compromise.[19]

The interplay between T_H17 and IL-23 in gut inflammation is complex and incompletely understood. While excessive IL-23–driven T_H17 activation contributes to intestinal inflammation, IL-17 is essential for maintaining intestinal barrier function by stimulating production of antimicrobial peptides, supporting epithelial repair, recruiting and facilitating activation of neutrophils, and restraining overgrowth of commensal bacteria.[16,20] In a systematic review and meta-analysis, Zhu et al[21] identified that the IL-23 receptor polymorphism rs11465804 was associated with an increased risk of constipation-predominant IBS. The potential role of IL-23 inhibition in IBS may warrant further investigation.[22]

Risankizumab binds the p19 subunit of IL-23, blocking its interaction with the IL-23 receptor and downstream signaling in the skin and gastrointestinal tract.[23,24] Risankizumab is a highly safe and effective medication for the induction and maintenance of remission in psoriasis, Crohn's disease, and ulcerative colitis; however, its effects on IBS have not been studied.[12,13,21–24]

To our knowledge, this is the first human report describing improvement of IBS symptoms with IL-23 blockade. The observed improvement of gastrointestinal symptoms in this patient with psoriasis treated with risankizumab raises the possibility that IL-23–mediated inflammation may have played a role in his IBS-D symptoms. Although spontaneous fluctuation of IBS symptoms is possible, the chronicity and stability of his prior symptoms, combined with sustained improvement temporally linked to IL-23 inhibition, make coincidental resolution less likely.

CONCLUSION

IL-23 inhibition with risankizumab may modulate systemic inflammation associated with the gut-skin axis.[3,7,10] Further investigation is warranted to clarify the role of IL-23 signaling in functional gastrointestinal disorders and its potential as a therapeutic target.[5,6,12,13,24–26] Future research should explore whether IL-23 inhibition confers benefits across both inflammatory and functional gastrointestinal conditions, particularly in patients with overlapping dermatologic and gastrointestinal disease.

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