Painful fissured plaque in a girl with Crohn’s disease

Meshal M. Alhameedy, MD‡, *, Areen M. Alhemediy, MS‡

Case
A 16-year-old Saudi girl diagnosed with Crohn’s disease (CD) when she was 10 years old, receiving infliximab (5 mg/kg IV) every 8 weeks for 2 years, presented with a painful cutaneous eruption in the posterior auricular region for the last 6 months. Examination found hyperkeratotic eroded fissured crusted erythematous plaque in the left posterior auricular area extending to the earlobe (Fig. 1A), progressively increasing over the last 6 months, associated with moderate to severe pain and mild pruritus. Dermatoscopic examination revealed uniform dotted vessels and a white-to-yellowish scale. There was no involvement of the scalp, oral and nasal mucosa, genitalia, or nails. Histopathological analyses (Fig. 1B) were performed. Special stains using Periodic acid-Schiff and Grocott methenamine silver revealed no organisms. However, tissue culture and sensitivity grew methicillin-susceptible Staphylococcus aureus. Basic laboratory panels, including complete blood count, liver and renal function tests, hepatitis B and C and HIV serology, purified protein derivative testing, and chest X-ray, were normal.

Question 1
What is your diagnosis?
A. Metastatic Crohn’s disease
B. Lupus vulgaris
C. Blastomycosis
D. TNF-inhibitor–induced psoriasis
E. Granuloma fissuratum

Correct answer: D. TNF-inhibitor–induced psoriasis.

Discussion
Histopathological analysis showed regular psoriasiform acanthosis, bulbous club-shaped rete ridges, parakeratosis, neutrophil accumulation in the stratum corneum, hypogranulosis, mild suprapapillary plates thinning, and sparse perivascular lymphohistiocytic infiltrate (Fig. 1B). Based on these findings, a diagnosis of tumor necrosis factor (TNF)-inhibitor–induced psoriasis is favored.

TNF-α is a proinflammatory cytokine essential for effective immune surveillance. Abnormally elevated TNF-α has been implicated in the pathogenesis of psoriasis and CD, making anti-TNF-α therapies an effective targeted therapeutic option for both. Various adverse cutaneous reactions to TNF-inhibitors (TNFi) have been described, including eczematous, lichenoid eruption, cutaneous vasculitis, lupus-like reaction, and exacerbation of preexisting psoriasis or new-onset psoriasis.

Of inflammatory bowel disease patients receiving anti-TNF-α therapy (the majority with infliximab), approximately 30% report a cutaneous eruption—the most common of which is a psoriasiform presentation. Moreover, a unique presentation of painful plaques over the posterior auricular region was recently reported in young women diagnosed with inflammatory bowel disease, most of whom had CD and were all treated with infliximab before the eruption. Additionally, TNF-induced psoriasis is predominantly seen in female patients.

The exact pathogenesis of TNFi-induced psoriasis is not entirely understood. Emerging evidence suggesting interleukin-23/T helper 17 axis dysregulation may play an essential role in the pathogenesis is supported by genome-wide association studies. These studies...
found that polymorphisms in the interleukin-23 receptor gene (IL-23R) are associated with an increased risk of both CD and psoriasis.

It is uncommon for bacterial superinfections to arise in psoriatic plaques (often demonstrated by erosion, fissuring, or yellow crust). However, our patient was an exception, which we hypothesize may be secondary to immunosuppression from the infliximab, manipulation of the lesion, or colonization of postauricular skin with *S. aureus*.

Histopathologically, the distinction between idiopathic psoriasis and TNFi-induced psoriasis is difficult. Nonetheless, a few features, including the absence of parakeratosis, neutrophilic collection below the stratum corneum, mild papillary plate thinning, and 3 or more eosinophils in the dermis, have been significantly associated with TNFi-induced psoriasis versus idiopathic psoriasis.

**Question 2**

What is the best next step in management?

A. Methotrexate  
B. Cyclosporine  
C. Topical treatment with or without phototherapy  
D. Switch to alternative TNF-inhibitor  
E. Switch to non-TNF-inhibitor biologic

**Correct answer:** C. Topical treatment with or without phototherapy.

A recent management algorithm for TNFi-induced psoriasis is proposed. If the primary disease is under control with mild to moderate psoriasiform eruption, topical treatment with or without phototherapy is suggested. If it is unsuccessful, adding methotrexate or cyclosporine should be attempted if the potential benefits outweigh the risks. For patients whose underlying disease is well-controlled with severe TNFi-induced psoriasis, transition to an alternative TNFi before reaching for an alternative biologic is advised.

As her underlying CD is under control using infliximab, we initiated topical treatment with calcipotriol/betamethasone dipropionate ointment twice daily for 3 months, plus topical antibiotic (sodium fusidate 2% ointment) twice daily for 2 weeks. However, the patient was absent for the follow-up.

**Conflicts of interest**

None.

**Funding**

None

**Author contributions**

MMA: conceptualization, methodology, investigation, writing—original draft, and writing—review & editing.  
AMA: investigation and writing—original draft.

**Study approval**

N/A.

**Patient consent**

Informed, written consent was received from the patient and confirmed to the journal pre-publication, stating that the patients gave consent for their photos and case history to be published.
References
1. Cleynen I, Van Moerkercke W, Billiet T, et al. Characteristics of skin lesions associated with anti-tumor necrosis factor therapy in patients with inflammatory bowel disease: a cohort study. Ann Intern Med 2016;164:10–22.
2. Ko LN, Pinard J, Merola JF, Patel M. Novel posterior auricular cutaneous reaction after anti-TNF-α infusion in young women with Crohn’s disease. JAAD Case Rep 2017;3(6):512–4.
3. Mazloom SE, Yan D, Hu JZ, et al. TNF-α inhibitor-induced psoriasis: a decade of experience at the Cleveland Clinic. J Am Acad Dermatol 2020;83:1390–8.
4. Hu JZ, Billings SD, Yan D, Fernandez AP. Histologic comparison of tumor necrosis factor-α inhibitor-induced psoriasis and psoriasis vulgaris. J Am Acad Dermatol 2020;83:71–77. Epub 2020 Jan 11. Erratum in: J Am Acad Dermatol 2020;83(4):1237.