Images in Rheumatology

A Case of Disseminated Cutaneous *Mycobacterium chelonae* Infection During Treatment With Tofacitinib

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Janus kinase (JAK) inhibitors, like other immunomodulators, are known to be associated with an increased risk of infections.¹ An analysis of long-term clinical trial data has shown tuberculosis to be the most common opportunistic infection associated with tofacitinib use.² However, treatment-related nontuberculous mycobacterial (NTM) infections are exceedingly rare, and a thorough review of literature did not reveal any cases.

We present the case of a 55-year-old male who was seen in the rheumatology clinic on follow-up for seronegative rheumatoid arthritis. He had failed several prior therapies, including methotrexate, etanercept, and most recently, adalimumab, before starting tofacitinib 5 mg twice daily, approximately 2 weeks earlier.

While having reported significant improvement in his joint symptoms, he had recently noted a new painful skin rash over his left forearm. The rash was nodular, erythematous, tender, and localized to the left forearm (Figure 1). A full-thickness skin biopsy revealed acid-fast bacilli. Of note, the patient's initial QuantiFERON-TB Gold was negative.

Over the next 2 weeks, the rash spread to involve the whole arm, with new lesions on the face. Tofacitinib was discontinued, and

he was placed on a slow steroid taper instead. Final speciation by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry confirmed the presence of *Mycobacterium chelonae*. He was subsequently started on intravenous antibiotics (tobramycin and azithromycin), which resulted in the resolution of his skin lesions.

M. chelonae is a rapidly growing mycobacterium that commonly presents with cutaneous disease.³ Our case highlights the need to consider NTM as a possible opportunistic infection in the setting of JAK inhibitor therapy.

REFERENCES

1. Winthrop KL. The emerging safety profile of JAK inhibitors in rheumatic disease. *Nat Rev Rheumatol* 2017;13:234-43.
2. Winthrop K, Park S, Gul A, Cardiel M, Gomez-Reino J, Tanaka Y, et al. Tuberculosis and other opportunistic infections in tofacitinib-treated patients with rheumatoid arthritis. *Ann Rheum Dis* 2016;75:1133-8.
3. Wallace Jr RJ, Brown BA, Onyi GO. Skin, soft tissue, and bone infections due to mycobacterium chelonae chelonae: importance of prior corticosteroid therapy, frequency of disseminated infections, and resistance to oral antimicrobials other than clarithromycin. *J Infect Dis* 1992;166:405-12.

Figure 1. *Mycobacterium chelonae* presenting as a nodular, erythematous, tender rash, localized to the left forearm, after 2 weeks of tofacitinib treatment.