Use of risankizumab in two HIV-positive patients with psoriasis



Khalad Maliyar, MD, BA, Perla Lansang, MD, FRCPC, Derm, and Philip Doiron, MD, FRCPC, Derm

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uman immunodeficiency virus (HIV)-associated psoriasis is characterized by greater disease severity, frequent exacerbations, atypical presentations, and treatment resistance. In HIV-associated psoriasis that is not responding to therapeutic options including topical medications, phototherapy, or systemic retinoids, the use of biologic therapy can be considered. The potential additional risk of a serious infection through the use of biologics in immunocompromised patients with HIV is why these patients have been excluded from controlled trials regardless of their level of HIV control.² The purpose of this case series is to shed light on the efficacy and tolerability of biologics in this population, by providing the details of 2 HIVpositive patients with psoriasis successfully managed with risankizumab.

CASE SERIES

Case 1

Patient 1 is a 36-year-old man with a long-standing history of severe guttate psoriasis. His medical history included depression and COVID-19. He was stable on highly active antiretroviral therapy, as his HIV viral load was undetectable and his CD4 count was 1461 cells/ μ L. He had tried numerous topical therapies, phototherapy, methotrexate, acitretin, apremilast, and adalimumab. Appropriate trials of these agents did not achieve adequate control of his psoriasis. He was started on risankizumab therapy, standard dosing with a Psoriasis Area Severity Index (PASI) of 5.4. After 5 months of therapy, his psoriasis completely cleared, PASI of 0,

Abbreviation used:

PASI: Psoriasis Area Severity Index

no adverse events, a CD4 count of 1225 cells/ μ L, and undetectable viral load.

Case 2

Patient 2 is a 58-year-old man with a long-standing history of plaque psoriasis. His medical history includes osteoarthritis, type 2 diabetes mellitus, depression, and prior human papillomavirus infection. He was stable on highly active antiretroviral therapy, as his HIV viral load was not detectable and his CD4 count was 831 cells/ μ L. He had tried several topical therapies, narrowband ultraviolet B phototherapy, psoralen and ultraviolet A, apremilast, and biologic therapies including both ustekinumab and guselkumab. Despite some improvement on biologics, his psoriasis persisted, with a PASI of 4.0, necessitating a switch to risankizumab therapy. After 3 doses, his plaques nearly completely resolved, with no adverse events, a CD4 count of 926 cells/μL, and undetectable viral load. His PASI improved to 1.6.

DISCUSSION

HIV-positive patients may have moderate -to-severe, refractory psoriasis that requires systemic therapy. One recent review of 52 HIV-positive patients with psoriasis treated with biologics (including adalimumab, etanercept, infliximab, ustekinumab, and guselkumab) demonstrated that these patients had

From the Division of Dermatology, University of Toronto, Toronto, Canada.

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Correspondence to: Philip Doiron, MD, FRCPC, Derm, Division of Dermatology, Women's College Hospital, 76 Greenville St, 5th floor, Toronto, ON, Canada M5S 1B2. E-mail: philip.doiron@wchospital.ca.

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neither frequent infections nor worsening HIV.³ To date, there has only been one case report published of a psoriasis patient with HIV infection, treated with the interleukin 23 (IL-23) inhibitor risankizumab. This patient had complete clearance of their psoriasis and no worsening of their HIV immunological parameters. 4 Risankizumab is a humanized IgG1 monoclonal antibody targeting the p19 subunit of IL-23 approved for the treatment of psoriasis and psoriatic arthritis. Our report of 2 patients being successfully treated with risankizumab, adds to the literature that selective inhibition of IL-23 appears to be a suitable therapeutic option for this patient population. Although the data are limited, IL-23 inhibitors appear to be a potential effective treatment option that can be used safely for patients with psoriasis with concomitant HIV infection. Patients should be carefully co-managed by a dermatologist and an HIV specialist.

Conflicts of interest

Drs Doiron, Lansang, and Maliyar has no conflicts of interest.

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