Adalimumab-associated psoriasiform rash in an African-American patient

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DESCRIPTION

A 72-year-old African-American man with a history of biopsy-proven pulmonary and hepatic sarcoidosis presented to primary care clinic with a pruritic rash. The lesions first appeared over his right lateral trunk 3 months ago before spreading to both arms and his groin. Of note, he had been started on adalimumab, a tumour necrosis factor (TNF) inhibitor, about a year ago for treatment of sarcoidosis. Physical examination revealed multiple scaly 0.5 to 2 cm ovoid, violaceous papules and plaques (figures 1 and 2) over the trunk, forearms and anterior hips extending to the groin. Skin punch biopsies of the right trunk and right groin were performed, and the final dermatopathology report confirmed psoriasiform dermatitis with overlying parakeratosis and crust with occasional eosinophils, consistent with a psoriasiform drug eruption. Although the patient's medications included atenolol and adalimumab, both of which have been implicated in druginduced psoriasis, in this scenario, adalimumab was felt to be the more likely culprit given the time course of his rash. Despite treatment with topical triamcinolone 0.1% and topical desonide 0.05%, the psoriasis persisted, and he was ultimately instructed to discontinue adalimumab and switch to



Figure 2 Clinical image depicting rash across the left lower abdomen and trunk.



Figure 1 Clinical image depicting rash on forearm.

mycophenolate mofetil for sarcoidosis treatment. He follows with dermatology and rheumatology.

Psoriasiform eruptions secondary to TNF inhibitors, also known as paradoxical psoriasis, have not been well described in African-Americans. Compared with psoriasis among Caucasians, psoriasis among African-Americans can have morphological differences, including prominent dyspigmentation, greater body surface area involvement, and a predominance of violaceous or brown hues rather than erythema.¹ Psoriasiform eruptions induced by TNF inhibitors are a well-documented drug class effect.² Adalimumab can cause paradoxical psoriasis in 2%-5% of patients receiving the agent for indications other than psoriasis and accounts for 14.4%-34% of all instances of TNF inhibitor-associated psoriasis.3 4 While both classical psoriasis and paradoxical psoriasis involve the increased production of type I interferons by plasmacytoid dendritic cells, paradoxical psoriasis in the setting of TNF blockade appears to be solely an innate inflammatory response without subsequent development of T-cell autoimmunity.⁵

Paradoxical psoriasis can present as plaques (44.8%), palmoplantar pustular psoriasis (36.3%) and psoriasiform dermatitis (19.9%), frequently

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involving the soles, extremities, palms, scalp and trunk.⁶ The average time between initiation of adalimumab and rash onset is 14.4 months, consistent with our patient's presentation.⁶ The decision to continue the same anti-TNF agent, replace with an alternative anti-TNF or switch to a different therapy class depends on the severity of the eruption and control of underlying disease.⁴ Among patients continued on original therapy, 26%–41% had resolution of their psoriasis, compared with up to 64% of patients who were switched to a non-anti-TNF drug.⁴ Topical steroids, vitamin D analogues, phototherapy and methotrexate are commonly used for concomitant treatment of psoriatic lesions.⁶

Aside from scaling, the skin changes in our patient did not resemble typical plaque psoriasis. Given a paucity of published

Learning points

- TNF inhibitor—associated psoriasis, or paradoxical psoriasis, should be suspected in patients who develop a new rash while on TNF inhibitors like adalimumab, especially around 1 year after therapy initiation.
- Psoriasiform rashes due to TNF inhibitor therapy can occur in African-American patients without the classic appearance of plaque-like eruptions.
- Switching to a non-anti-TNF agent often leads to resolution of psoriasis in these cases.

images in the literature depicting paradoxical psoriasis in the African-American population, this case may prompt clinicians to recognise and appreciate this type of skin eruption in African-American patients on TNF inhibitors.

Contributors YC, the corresponding author, obtained patient consent, performed literature review and drafted the initial manuscript. JH was a consultant on the case and revised the manuscript. NK acquired the clinical images and revised the manuscript. UEM was the senior clinician and consultant and revised the manuscript.

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