Severe bout of cutaneous psoriasis in a patient with psoriatic arthritis undergoing treatment with infliximab

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Infliximab, anti-TNF α chimeric monoclonal antibody, has recently demonstrated its efficacy and safety in treating active (PsA) psoriatic arthritis dermatological and articular manifestations Likewise dermatological lesions in psoriasis vulgaris (1,2) having recently been approved as treatment in both cases. Described below is the “paradoxical” case of a patient who suffered a generalized bout of psoriasis while undergoing treatment with infliximab.

Male, aged 41, diagnosed with psoriatic spondyloarthropathy 20 years earlier with daily axial pain and severely limited mobility. He had presented plaque psoriasis with slight-moderate affection since he was 14, although he had received no specific treatment due to the minor presence of his ailment. He began treatment with infliximab iv 5 mg/kg as per standard dosage and his usual NSAI. The patient’s articular symptoms greatly improved from the first infusion, with his cutaneous lesions almost totally disappearing.

One week after the 3rd infusion, he presented a generalized outbreak of psoriasis, initially affecting his feet and subsequently extending to practically the entire corporeal surface, accompanied by pruritis and oedemas on lower limbs. He presented a good general state without fever and blood pressure 120/70. Laboratory tests showed an ESR 67 mm/hour, CRP 6.8 mg/L, and anemia with slight-moderate affectation since he was 14, although he had received no specific treatment due to the minor presence of his ailment. He began treatment with infliximab iv 5 mg/kg as per standard dosage and his usual NSAI.

The earliest studies on the immunological mechanisms of PsA evidenced the role of T lymphocytes in Ps with an increase in Th1 cytokines. Subsequent studies have demonstrated the central role played by innate immunity in the pathogenesis of psoriasis (11), with an increase of innate immune response humoral components, complement activation, elevation of chemokines like IL-8 and an increase in pro-inflammatory cytokines, among which TNF-α is one of the most important.

Anti-TNF-α treatment is one of the possible strategies for treating this illness, in addition to other biological treatments aimed at other targets. We are unaware of the mechanism whereby infliximab led to the worsening of our patient’s psoriasis. Further studies are required to explain “paradoxical” phenomena of this kind.

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