Psoriasis: Targets and Therapy Dovepress

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CASE REPORT

Psoriasis Flare Following Paramyxovirus Infection

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Abstract: Psoriasis is a chronic, immunologically mediated disease of multifactorial origin, with genes playing a key role and environmental factors, such as infections, often triggering its onset or exacerbation. While acute streptococcal infections are commonly linked to guttate psoriasis, viral and fungal infections have also been associated with psoriasis flares. We report a case of severe psoriasis exacerbation during viral parotitis caused by paramyxovirus in a 49-year-old male patient with a long-standing psoriasis diagnosis. Following successful treatment with secukinumab, the patient experienced a flare-up coinciding with symptoms of mumps infection. Serological tests confirmed the presence of mumps virus RNA. Secukinumab was discontinued, and treatment with risankizumab resulted in rapid remission of psoriasis. While paramyxovirus infections are not typically associated with psoriasis flares, emerging evidence suggests that dysregulated antiviral immune responses may induce IL-23 production, possibly contributing to inflammation in psoriasis. This case highlights the need for further research on the role of antiviral immune responses in psoriasis exacerbations and the potential therapeutic implications of targeting the IL-23 pathway.

Keywords: psoriasis, paramyxovirus, infection

Psoriasis is a chronic, immunologically mediated disease that is considered a complex disorder of multifactorial origin. Although genes play a major role in the pathogenesis of psoriasis, environmental factors are often required to trigger the onset and recurrence of the disease.[1,2] Among these, infections have been widely recognized as responsible for the onset and exacerbation of psoriasis through various mechanisms.[1] Acute streptococcal infection has long been identified as a triggering factor of guttate psoriasis and an exacerbating factor of plaque psoriasis. Indeed *Streptococcus pyogenes* can activate innate immune cytokines and Th1/Th17-related genes in keratinocytes.[3] Viral and fungal infections have been linked to the onset or exacerbation of psoriasis through the dysregulation of the host's antiviral immune response.[4] Recently, a relationship between infections and psoriasis involving the interleukin (IL)-23/17 signaling pathway has been discovered, the latter being considered the major immune pathway in psoriasis disease pathogenesis.[2] Here we report a case of severe psoriasis flare occurring during viral parotitis associated with paramyxovirus infection.

A 49-year-old male with an established diagnosis of psoriasis from 7 years before was referred to our clinic because of a worsening of his psoriasis which had been treated with topical corticosteroids, narrow-band ultraviolet B phototherapy, ciclosporin and methotrexate. Psoriasis Area Severity Index (PASI) was 22. To initiate a biological antipsoriatic therapy, the patient was screened for the presence of tuberculosis, hepatitis B, C, and HIV without clinically relevant results. Secukinumab was then introduced in November 2020, at a dose of 300 mg at weeks 0, 1, 2, 3, 4 and then 4-weekly thereafter. Within 12 weeks, he went into complete remission (PASI score 0). Thirty-six months after secukinumab initiation, no relapse of psoriasis was seen. Soon after, the patient experienced an acute flare of his psoriasis. A large number of plaques covered progressively his face, trunk, and limbs (Figure 1). Due to the concomitant development of fatigue, fever, chills, headache, and sore throat, the patient was administered paracetamol and bed rest. General symptoms persisted with reduced appetite followed by the occurrence of bilateral parotid gland pain and swelling. Serologic tests showed a high titer of IgM and IgG against Paramyxovirus. In addition, a saliva sample detected the presence of Mumps virus (MuV) RNA by RT-PCR. Serological tests for influenza and other parainfluenza viruses, coxsackie A virus, cytomegalovirus, echovirus, and enterovirus were negative, as well as throat swab specimens

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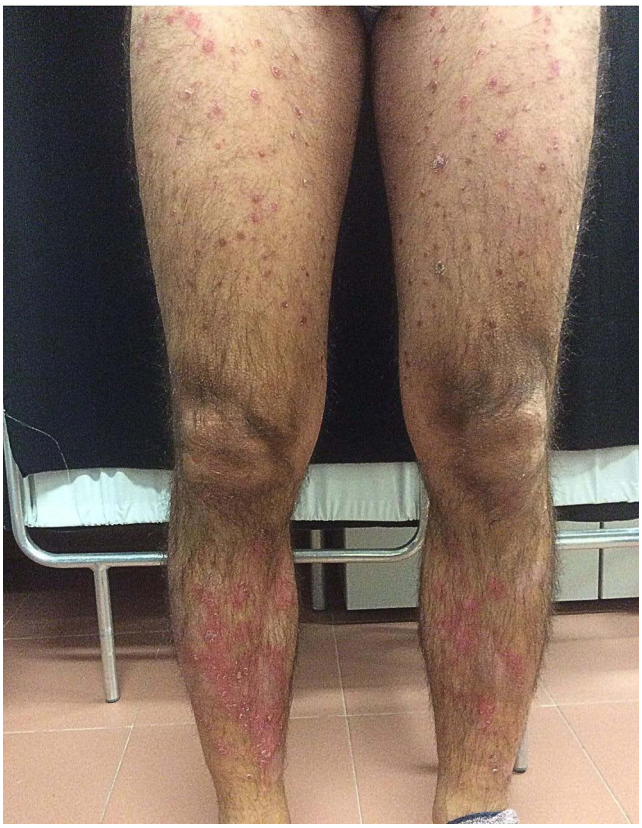

Figure 1 Psoriasis flare with psoriatic plaques involving the limbs.

for bacteria, oral and lingual swabs for Candida (Table 1). Due to the persistence of severe psoriasis after his fever and parotid swelling had disappeared, secukinumab was stopped. The patient was treated with risankizumab subcutaneously, at a dose of 150 mg at week 0, week 4, and then 12-weekly thereafter (Figure 2). Complete remission of psoriasis

Table 1 Test regarding Infections Performed during the Clinical History of the Patient

	Test Methodology	Result
Before starting biologic therapy		
Tuberculosis	Quantiferon (IGRA)	Negative
HBV	Serological test	Negative
HCV	Serological test	Negative
HIV	Serological test	Negative
During the flare up		
Influenza	Serological test	Negative
Parainfluenza v.	Serological test	Negative
Coxsackie A virus	Serological test	Negative

(*Continued*)

Table 1 (Continued).

	Test Methodology	Result
Cytomegalovirus	Serological test	Negative
Echovirus	Serological test	Negative
Enterovirus	Serological test	Negative
Bacteria	Throat swab specimens	Negative
Candida	Oral and lingual swabs	Negative
Paramyxovirus	Serological test	IgM, IgG elevated
Paramyxovirus	RNA by RT-PCR on saliva sample	Present

Abbreviations: IGRA, interferon-gamma release assay; HBV, Hepatitis B virus; HCV, Hepatitis C virus; HIV, Human immunodeficiency virus.

(PASI 0) was achieved 2 months after the start of the treatment. At his most recent follow-up visit, 6 months after the initiation of risankizumab therapy, no relapse of psoriasis was seen.

The MuV, a member of the Paramyxoviridae family, is an enveloped, non-segmented, negative-sense RNA virus. It is a significant human pathogen and the most common causative agent of acute parotitis. A decline in mumps infection has been seen following the global, combined measles-mumps-rubella (MMR) vaccination. However, due to recent mumps outbreaks among vaccinated young adults, concerns have been raised about primary and secondary vaccine failures.[5] Hypotheses have considered differences between original and current strains and reduced mumps immunity over time compared with that of measles and rubella.[5] To address efforts to control mumps resurgence, Centers for Disease Control and Prevention (CDC) guidelines recommended administering a third dose to high-risk groups during mumps outbreaks.[6]

In our patient there was a strict temporal relationship between MuV infection and a psoriasis flare after a long-term disease remission. Additional environmental triggers of psoriasis were ruled out.

To date, there is no reported increased risk of developing viral infections during anti-IL-17 therapy, unlike what has been observed for fungal infections. This makes anti-IL-17 agents a viable option also in patients with chronic infections such as HCV or inactive HIV.[7] Moreover, compared with TNF inhibitors, IL-23 inhibitors and IL-17 inhibitors are associated with a reduced risk of several infectious diseases. These agents might be preferred in patients who are considered at risk for infections.[8]

Viruses associated with the onset or flare of psoriasis can act directly as triggers, either as superantigens or as costimulatory factors.[1] Paramyxovirus infections have not been associated with worsening or new onset of psoriasis.

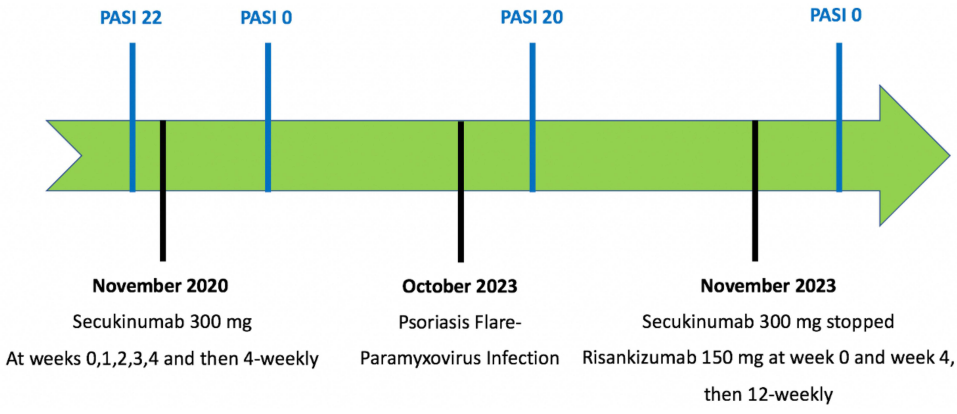

Figure 2 Timeline of patient's psoriasis biologic treatment and PASI index.

Recently, one of the pattern recognition receptor systems and major sensors of RNA viruses, namely retinoic acid inducible-gene I (RIG-I), was shown to be able to induce IL-23 production in the dendritic keratinocytes in mice, suggesting that dysregulated antiviral immune responses may initiate the vicious cycle of inflammation in psoriasis.[9] MuV is recognized by RIG-I and activated IFN regulatory factor 3 (IRF3) via mitochondrial antiviral signaling (MAVS).[10] Therefore, it is conceivable that MuV, like the other RNA viruses, can promote IL-23 production in the dendritic keratinocytes. In light of this, an anti-IL-23 therapy should be considered, at least theoretically, the most appropriate choice to control psoriasis flares associated with viral infections.

Future research should confirm how dysregulated antiviral immune responses of hosts may drive pathogenic immune responses through an increase in IL-23 production. Consequently, the inhibition of such signaling pathways could begin a relevant strategy in psoriasis triggered or exacerbated by RNA virus infections. Moreover, this finding could pave the way for future studies in this area since new viruses, as SARS-COV2, have emerged, and older ones, like paramyxovirus, may undergo to resurgence due to vaccination challenges. Therefore, it is crucial to improve more effective knowledge and treatment strategies in response to virus-related psoriasis flares.

Ethics Statements

The patient has given consent to publish his case details and picture.

No institutional approval was required to publish this case.

Disclosure

The authors declare no conflict of interest.

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