

A Case of Psoriasis Secondary to Tinea Corporis Treated with Skuclizumab and Review of Related Literature

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Abstract

The patient is a 69-year-old man with a 16-year history of psoriasis. Dermatologic examination: dry skin all over the body, head, face, trunk, and limbs can be seen widely distributed about soybean to fava bean size edematous erythema, border unclear, the surface can be seen a little flaking and scratch marks. Histopathology of skin lesions: (Abdominal) Epidermal protuberance flattened, a small number of lymphocytes, and eosinophils infiltration was seen around the capillaries in the superficial dermis. Multiple direct immunofluorescence was negative. Combined with the patient's disease course, rash manifestations, and histopathologic findings. The diagnosis was considered: common type psoriasis. The patient was given subcutaneous injection of Skuclizumab 300 mg, and after half a month of treatment, a rash with itching appeared on the head, face, forehead, chest, hands and feet, and the symptoms of rash and itching gradually aggravated with the passage of time, and the report of fungal microscopy was positive for toenail fungus. The patient was given oral itraconazole 200 mg bid and anti-allergic drugs, topical antifungal drugs, and symptomatic support and was discharged after improvement of the condition. Six months later, the patient's fungal microscopy was negative when he was injected with skuclizumab regularly, and the patient is still being followed up at present.

Keywords

Skuclizumab, Psoriasis, Eczema, Fungal Infections

1. Introduction

Psoriasis is a common chronic, immunologic, systemic disease characterized by

manifestations of skin damage. Studies have shown that T-cell subsets Th1 and Th17 and the cytokines they secrete (e.g., TNF- α , IL-12, IL-23, and IL-17) play an important role in the pathogenesis and progression of psoriasis. Under normal conditions, a dynamic balance is maintained between Th1/Th17 and Th2 cells. However, when the balance of the immune system is disturbed, a Th1/Th17 or Th2 bias may occur. Psoriasis is predominantly associated with a Th1/Th17 immune response, with Th1 cells secreting gamma-interferon (IFN- γ) and TNF- α , while Th17 cells secrete IL-17 and IL-22. In contrast, atopic dermatitis (AD) is predominantly dominated by a Th2/Th22 immune response, with Th2 cells secreting IL-4 and IL-5. Therefore, targeting these cytokines with biologics (e.g., skitice-lumab) has emerged as an important therapeutic option for moderately severe and refractory psoriasis [1] [2].

However, the widespread use of biologics may disrupt cytokine homeostasis, and it is not uncommon for a small number of patients to develop adverse skin reactions manifesting as itching or aggravation of pre-existing itching, with lesions that are often characterized by acute eczema-like dermatitis in the clinic practice [3]-[6]. This article provides a case report and a review of the literature on the treatment of psoriasis with strychnicolizumab, leading to eczema-like drug reactions

2. Clinical Information

A 69-year-old male was admitted to the hospital with “recurrent erythema with itching over the whole body for more than 1 year, aggravated for 10 days”. The patient had been injected with “Skucilizumab” 300 mg subcutaneously for psoriasis on November 3, 2021, and complained that after half a month of Skucilizumab treatment, a rash with itching appeared on the head, face, and forehead. The patient has been admitted to the hospital because of “psoriasis” since November 3, 2021, when she received several subcutaneous injections of “Skucilizumab” 300 mg, complaining that after half a month of Skucilizumab treatment, a rash appeared on the head, face and forehead, accompanied by itching, and the erythema and itching were mild at the initial stage, and could be improved after injecting the medication again, but the rash and itching gradually worsened with the passage of time. After the injection again on April 20, 2022, the symptoms continued to worsen, and the itching was so intense that it interfered with sleep at night. During the course of the disease, the patient had no chills and fever, no cough and sputum, no panic and chest tightness, no joint pain and other discomforts.

Past history: history of psoriasis for 16 years. Denied history of eczema, allergic rhinitis, and asthma; denied history of fungal infections such as tinea corporis, tinea pedis, and tinea cruris; denied history of hypertension, coronary heart disease, and diabetes mellitus; denied history of infectious diseases such as hepatitis, tuberculosis, etc.; denied history of surgery, trauma, and blood transfusion; denied family history of the hereditary disease; denied history of food and drug allergy.

Dermatologic condition: dry skin all over the body, head, face, trunk, and limbs

can be seen widely distributed about soybean to fava bean size edematous erythema, the boundary is not clear, which can be seen on the little flaking and scratch marks. PASI: 40.5.

Laboratory tests: percentage of monocytes: 14.3%, absolute value of monocytes: $1.11 \times 10^9/L$, stool routine: Occult Blood OB (+), coagulation: fibrinogen: 4.38/L, immunofluorescence antinuclear antibody test: positive (1:100), cytoplasmic granular type; urine routine, renal function, electrolyte analysis, cardiac enzyme profile, fasting blood glucose, blood sedimentation, quantification of Hepatitis A and C, antibody to Hepatitis D and E, cytokine test, syphilis antibody, HIV test did not show any significant abnormalities. Electrocardiogram showed sinus rhythm, no obvious abnormality; chest CT scan showed: 1) fibrous foci or chronic inflammatory foci in both lungs; 2) small nodular shadows in both lungs and subpleural, partial calcification; 3) slight thickening of pleura bilaterally, localized calcification; 4) small cardiac shadows, calcification of the aorta; 5) low-density shadows in the liver, possibly cystic lesions. 28 October 2021 Mycobacterium tuberculosis gamma interferon *in vitro* release assay (IGRA): positive.

Dermatopathologic examination (**Figure 1**): (Abdomen) Epidermal protuberances were flat, and a small infiltration of lymphocytes and eosinophils was seen around the capillaries in the superficial dermis. Direct immunofluorescence: IgG (-), C3 (-), IgA (-), IgM (-). Diagnosis: eczema, skin infection of the trunk, psoriasis vulgaris. Dermatologic fungal microscopy report: toenail fungus Positive.

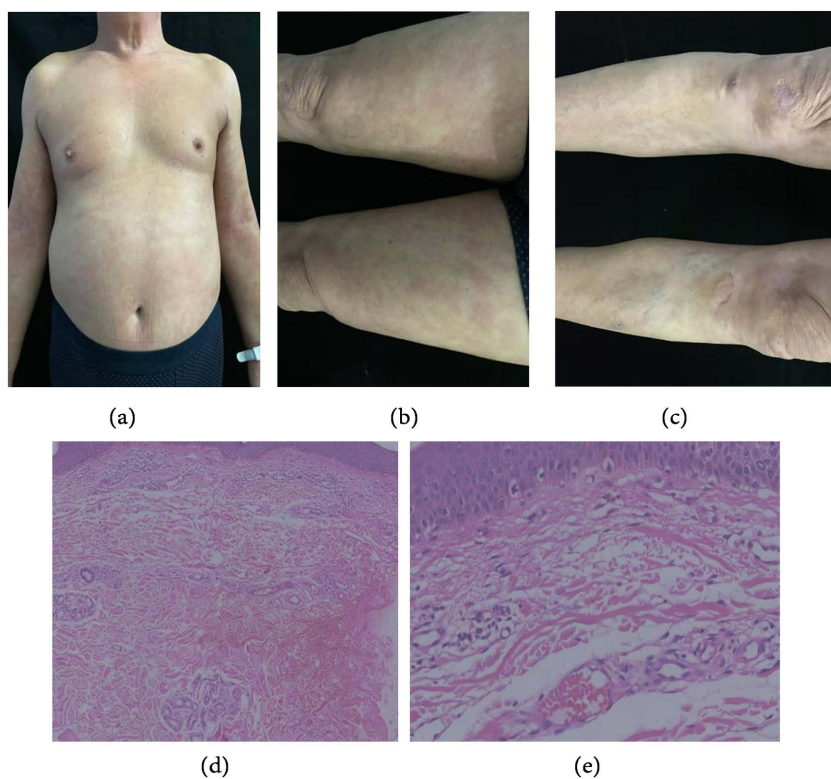


Figure 1. (a) popliteal fossa lesions; (b) back skin lesions; (c) foot skin lesions; (d) HP $\times 40$; (e) HP $\times 100$.

During hospitalization, the patient was given oral itraconazole 200 mg bid and anti-allergy drugs (loratadine tablets, levocetirizine hydrochloride tablets), topical glucocorticoids, antifungal drugs and symptomatic support, and was discharged from the hospital after improvement of the condition. In July 2022, the patient complained that the erythema of the torso and extremities subsided when injecting stavudine, but the erythema and flaking of both hands between the fingers still existed, and fungal microscopic examination: the finger slits were positive. (**Figure 2**), after reviewing liver function, the patient continued to be treated with oral itraconazole 0.2 g bid; in November 2022, when the patient was regularly injected with biologics, the fungal microscopy was negative, and the patient is still being followed up.

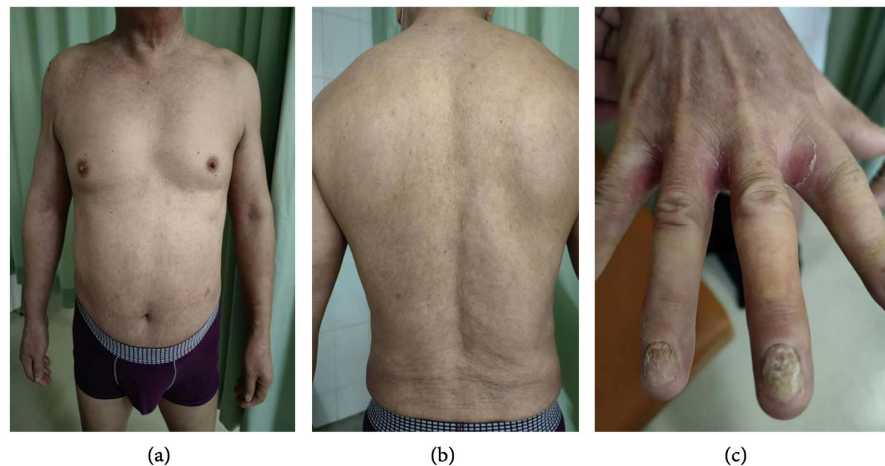


Figure 2. (a) frontal trunk lesions; (b) dorsal trunk lesions; (c) finger lesions.

3. Discussion

Biological agents causing eczema-like lesions on the trunk extremities of patients may be associated with immune drift. The widespread use of biological agents may lead to an imbalance between the Th1/Th17 and Th2 immune systems. Changes in cytokine levels may cause shifts in disease states. For example, targeted inhibition of Th17 and Th1 cell-associated factors may lead to a shift in the immune response toward the Th2 type, which can trigger skin lesions similar to atopic dermatitis (AD). Conversely, targeted inhibition of Th2 cell-associated factors such as IL-4 and IL-13 may cause the immune response to drift toward the Th17/Th1 type, resulting in psoriasis-like skin lesions. This shift in the state of the immune system is known as immune drift [2] [7] [8].

The incidence of AD-like reactions triggered by secukinumab treatment of psoriasis has been reported in the literature to range from 2.6% to 7.6%, with a mean time of onset of 20 weeks post-treatment [9]. However, when patients discontinued skticitlumab and switched to oral cyclosporine, antihistamines, JAK1 inhibitors, and topical glucocorticoids, the AD-like lesions improved significantly and the associated levels of immune and inflammatory factors returned to the normal

range, which further supports the possibility of a drift from Th17-type immunity to Th2-type immunity [10].

The mechanism by which IL-17a inhibitors trigger immune drift is not fully understood, with some suggesting that it may be that the biologics suppress the Th1/Th17 phenotype in psoriasis, leading to a shift in the immune balance toward the Th2 phenotype in atopic dermatitis (AD) [11]. In addition, some have suggested that inhibition of IL-17a may induce upregulation of IL-17c, which promotes the secretion of more IL-17a/f and IL-22 by Th cells, which may contribute to the development of eczema-like dermatitis [12].

Treatment of eczema-like lesions during the course of psoriasis treatment includes the use of moisturizers and topical glucocorticoids for limited mild damage, the discontinuation of biologics, and the search for alternative treatment options for lesions of moderate and greater severity [13]. Depending on the severity of the lesions, choose the appropriate intensity of treatment, such as methotrexate, cyclosporine, or Janus kinase (JAK) inhibitors, which are effective in both psoriasis and atopic dermatitis (AD) [14]. And have been reported that JAK inhibitors such as abatacept, tofacitinib, upadacitinib, and deucravatinib may also be a drug of choice for the treatment of psoriasis [15]. Our patient showed improvement after topical glucocorticoids and symptomatic treatment during hospitalization.

In addition, it is now suggested that biologics may affect skin barrier repair by decreasing the production of antimicrobial peptides by keratin-forming cells, thereby increasing colonization and value-added by *S. aureus* or fungi, which in turn initiates type 2 inflammation [10]. In the skin of healthy people, IL-17 is involved in the regulation of the microbiota and in the protection against fungal infections [16]. In the case of fungal infections, damaged epithelial cells increase the production of pro-inflammatory cytokines, activate and increase the production of IL-17 by Th17 cells, which activates the immune response and the production of chemokines, which ultimately leads to an increase in the number of neutrophils and antimicrobial peptides used to fight the infection [16] [17]. Therefore, adverse reactions leading to increased risk of fungal infections after treatment with biologics are worthy of our attention, which may be related to the development of tinea versicolor in our patient.

The patient denied a history of atopic diseases such as eczema, allergic rhinitis, asthma, etc., and it is not clear whether adverse reactions to the use of biologics are related to the patient's allergic constitution; the number of cases reported in this study is relatively small, and because of the limited conditions of the hospital laboratories, a larger sample size, more relevant examinations in laboratories (changes in the relevant cytokines), and a more systematic study are needed to reach a more definitive conclusion, which will help to apply the biologics. More detailed evaluation and careful selection of biologics are needed to cope with varying degrees of eczema-like drug reactions and chances of co-infection with fungal infections.

Consent

Informed consent was obtained from the patient for this case report.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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