Case Report

Ultraviolet B Phototherapy Worsened Psoriasis Vulgaris

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Abstract

Psoriasis vulgaris is a chronic, complex, immune-mediated inflammatory disease with a genetic predisposition and a global prevalence of 2%–4%. One treatment option for psoriasis is narrowband ultraviolet B (NBUVB) phototherapy. Phototherapy can reduce cytokines and chemical mediators that trigger psoriasis. However, there is a clinical subset of photosensitive psoriasis in 5.5%–24% of cases, wherein NBUVB exposure at subminimal doses can worsen psoriasis lesions. Herein, we present a case of a 23-year-old man with a seven-year history of psoriasis and who completed eight sessions of the secukinumab injection protocol with full remission. The patient complained of worsening psoriasis lesions, and his psoriasis area and severity index score increased to 6.5. He was scheduled to undergo two sessions of whole-body NBUVB phototherapy at 500 mJ/cm^2 for, which then further worsened the lesions, progressing into erythrodermic psoriasis. Phototherapy was withheld, and the patient was hospitalized. In 5.5%–24% of psoriasis cases, UV light exposure can worsen psoriasis. This condition is related to the genetic predisposition of HLA-Cw*0602 and CARD14 mutation at locus 2 (PSORS2), which causes a different immune response when the skin is exposed to UV light, including phototherapy. In these cases, there is no physiological suppression of propsoriatic cytokines and mediators. Instead, propsoriatic mediators are increased through various pathways, including tumor necrosis factor-alpha, interferon gamma, interleukin (IL)-1, IL-6, or IL-17, after UV exposure. Thus, understanding photosensitive psoriasis is essential, and further studies are needed to determine the best course of treatment in these patients.

Keywords: Photosensitive psoriasis, phototherapy, psoriasis vulgaris.

How to Cite this Article: Harsono ET, Siswati AS, Anggatama M, Khalidah M, Budiyanto A. Ultraviolet B Phototherapy Worsened Psoriasis Vulgaris. J Pak Assoc Dermatol. 2025;35(4):381-385.

Received: 19-04-2025 **Revision:** 11-07-2025 **Accepted:** 21-10-2025

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Introduction

Psoriasis vulgaris is a chronic inflammatory disease that is mediated by a complex immune response and has a genetic predisposition.[1] Psoriasis has a global prevalence of 2%–4% and can affect men and women.[2] The manifestations of psoriasis are not only limited to the skin but may also involve various organs, including the nails, joints, and tongue (geographic tongue). Skin lesions typically appear as well-defined, oval, erythematous plaques with thick, silver scales that may be accompanied by itching.[1] Erythrodermic psoriasis is a severe psoriasis variant, with an estimated prevalence of 1%–2.25% among patients with psoriasis. It is the most common cause of erythroderma, accounting for approximately 25% of all cases.[3]

Psoriasis vulgaris treatment depends on disease severity and may include topical therapy, phototherapy, systemic therapy, or a combination of these approaches.[4] Exposure to ultraviolet (UV) radiation from natural sunlight or artificial light sources is beneficial for managing psoriasis. However, some individuals with psoriasis may experience worsening because of UV radiation, particularly in cases involving photosensitive skin condi-

tions, including porphyria, chronic actinic dermatitis, systemic lupus erythematosus, or polymerphous light eruption. Ultraviolet A (UVA) and ultraviolet B (UVB) rays can trigger psoriasis lesions in a phenomenon known as photosensitive psoriasis, even at suberythemal doses. These doses can induce new lesions or exacerbate existing psoriasis. The prevalence of photosensitive psoriasis among patients with psoriasis ranges from 5.5% to 24%, with a higher incidence in females and a younger age of onset.[5] This condition may significantly complicate psoriasis management.

In this case report, we present a patient with psoriasis vulgaris aggravated by phototherapy. This report aims to provide insights into a rare psoriasis phenotype, known as photosensitive psoriasis.

Case Report

A 24-year-old male with a history of erythrodermic psoriasis and who completed the eight-session secukinumab injection protocol presented to our Dermatology and Venereology Clinic at Sardjito General Hospital, Yogyakarta. The patient reported multiple new reddish patches on his back and all four extremities. One month prior to his visit, he noted the appearance of new, scaly, itchy reddish patches on his back. He was diagnosed with psoriasis vulgaris with a psoriasis area and severity index (PASI) score of 1.7 and was prescribed with a topical formulation containing 6% salicylic acid, 9% liquor carbonis detergens (LCD), and 15 g of clobetasol in 30 g of petroleum jelly, applied twice daily on the lesions.

Two weeks later, the patient reported new red, scaly, itchy lesions on his scalp, arms, chest, and back (Figure 1). At this time, his PASI score had increased to 6.5, and he was prescribed with a similar topical mixture containing 6% salicylic acid, 9% LCD, and 30 g of desoximetasone in 60 g of petroleum jelly. He was also sent for phototherapy twice a week, with two sessions of whole-body narrowband UVB (NBUVB) phototherapy at 500 mJ/cm^2. However, after the sessions, we noted worsening of the red patches dispersing over nearly his entire body.

The patient was first diagnosed with psoriasis vulgaris in 2016. In 2022, his condition relapsed, and he was treated with methotrexate at a dose of 17.5 mg/week, folic acid 1mg per day, and 30 g of desoximetasone in 100 mL of coconut oil. He completed three phototherapy sessions at a dose of 500 mJ/cm^2 and two sessions at a dose of 625 mJ/cm^2. However, his lesions worsened, with more redness and peeling, leading to discontinuation of the therapy. Subsequently, he underwent an eight-session secukinumab injection protocol, with significant improvement and full remission. No other relevant associated comorbidity or similar family history was noted.

Figure 1: Clinical presentation of the patient before phototherapy showed red scaly plaques with PASI score of 6,5.

Figure 2: After 2 sessions of whole body NB-UVB phototherapy at a Dose of 500 mJ/cm^2, the patient developed generalized erythroderma.

Our dermatological examination showed diffuse erythematous patches covering nearly his entire body, whereas some areas exhibited erythematous plaques with thick scales on the surface (Figure 2). The patient was finally diagnosed with erythrodermic psoriasis attributed to the exacerbation of psoriasis vulgaris triggered by phototherapy. Phototherapy, which was initially planned for the patient, was soon discontinued. Thereafter, the patient was admitted to the ward to stabilize his con-

dition with a topical steroid and methotrexate injection (50 mg per week). After seven days of treatment, his lesions improved, and he was subsequently discharged. His follow-up was uneventful, with continuous methotrexate injection treatment.

Discussion

Generally, UV exposure is beneficial for patients with psoriasis. However, in 5%–20% of individuals with psoriasis, UV exposure can exacerbate the existing condition, known as photosensitive psoriasis. Clinically, the entity could be distinguished from psoriasis vulgaris based on the lesions that predominantly appear in sun-exposed areas, including the face, neck, and dorsum of the hands. Nevertheless, patients with photosensitive psoriasis may also experience worsening of symptoms with phototherapy.[6]

Phototherapy, including NBUVB and psoralen plus UVA (PUVA) therapy, is the treatment of choice for psoriasis. Phototherapy has proapoptotic and immunosuppressive properties, acting either independently or in combination. UVB or PUVA exposure can upregulate antipsoriatic interleukins (IL) and cytokines, including IL-4 and IL-10, while downregulating propsoriatic cytokines, including IL-8, IL-9, IL-17, IL-22, IL-23, tumor necrosis factor-alpha (TNF-α), and interferon gamma (IFN-γ). Through these effects, UV exposure can induce regulatory T cells, which are considered crucial in the pathogenesis of psoriasis and its response to treatment. Moreover, PUVA phototherapy downregulates helper T cells (Th), particularly Th17 cells, which play a significant role in the pathogenesis of psoriasis.[5]

According to previous studies, photosensitive psoriasis follows a seasonal pattern, becoming severe during summer months and mild or absent during winter, with predominant involvement of sun-exposed areas. In addition, these studies indicate that photosensitive psoriasis is more common among females, associated with an early onset of psoriasis, and triggered by minimal erythema dose (MED) levels of UV radiation. However, this contrasts with earlier findings, where photosensitive psoriasis was more frequently observed in individuals with Fitzpatrick skin type I, hereditary photosensitivity, advanced age, treatment challenges, and increased involvement of the face and hands compared with nonphotosensitive patients.[7]

Although the underlying mechanisms of photosensitive psoriasis remain poorly understood, several hypotheses have been proposed. These include koebnerization following sunburn on fair skin or after polymorphic light eruption (PLE), coexisting photosensitivity disorders, and the de novo initiation or exacerbation of psoriasis by UV radiation. Psoriasis induced de novo by photoprovocation is believed to take several days to appear after UV exposure. By contrast, a rapid photosensitive reaction in patients with psoriasis may suggest the presence of an underlying photosensitivity disorder.[8] In some individuals with a genetic predisposition for photosensitive psoriasis, phototherapy may induce a slightly different immune response compared with that in patients without such predisposition. Patients with genetic predispositions, including those possessing the HLA-Cw*0602 allele or a CARD14 mutation at locus 2 (PSORS2), may experience innate immune response activation during phototherapy. This response is associated with the production of antimicrobial peptides (AMPs) triggered by various pathogen-associated molecular patterns (PAMPs), damage-associated molecular patterns (DAMPs), Toll-like receptor (TLR) agonists, or endogenous inflammatory mediators (e.g., TNF-α, IFN-γ, IL-1, IL-6, or IL-17).[5]

Inflammatory mediators in psoriasis, including IL-1β, can directly promote keratinocyte proliferation and leukocyte chemotaxis.[9] Moreover, the IL-1α signaling pathway is crucial for the formation of neutrophil clusters within the epidermis, known as Munro microabscesses, a histopathological feature observed in patients with psoriasis.[10] IL-1 receptor antagonist (IL-1ra) is an immune system regulator that helps keep inflammation under control. The balance between IL-1α and IL-1ra levels in the skin may be the key to understanding why UV radiation should be avoided if skin inflammation exceeds a certain threshold.[11]

IL-17 is now recognized as a central cytokine in the pathophysiology of psoriasis. Besides Th17 cells,

IL-17 is also produced by innate immune cells, including neutrophils, mast cells, gamma-delta T cells, and innate lymphoid cells. IL-17 stimulates keratinocyte growth and contributes to epidermal hyperplasia. It also improves the migration of cellular adhesion molecules, thereby facilitating the chemotaxis of inflammatory cells (e.g., neutrophils). Furthermore, IL-17 influences dendritic cells to produce more IL-23, which, in turn, drives the differentiation of CD4+ T cells into Th17 cells, thereby increasing IL-17 production. Alongside IL-23, IL-17 induces IL-6, IL-8, and TNF-α, all of which play a role in the pathogenesis of psori-asis.[12]

TLRs, which are crucial components of innate immunity, have a cytoplasmic domain similar to that of IL-1 receptors and can recognize PAMPs and DAMPs. Following UVB exposure, DNA damage in exposed cells can trigger the release of DAMPs, which are recognized by TLR4.[5] This process stimulates inflammatory signaling pathways and the innate immune response. The activation of TLRs by their respective ligands leads to the production of inflammatory cytokines and upregulation of costimulatory molecules and major histocompatibility complex molecules on dendritic cells, thereby exacerbating psoriasis lesions.[13]

Besides DAMPs, TLRs are also stimulated by various factors induced by UV light, such as oxidized lipid and heat shock proteins, including HSP60, HSP70, and HSP90.[5] Polymorphisms in TLRs, including TLR4, are also correlated with immunosuppression induced by UV light. This may explain why some individuals are more prone to some dermatological diseases, including lupus erythematosus, PLE, and psoriatic arthritis.[5]

According to recent studies, UV exposure can also damage RNA, which is subsequently detected by TLR3, stimulating the production of inflammatory cytokines (e.g., TNF-α and IL-6) from nonirradiated keratinocytes and peripheral blood mononuclear cells. TLRs can also regulate the expression of AMPs.[14] Laboratory research has demonstrated that UV exposure induces AMPs, including human beta-defensin (HBD) 2, HBD3, RNase7, and S100A7 (psoriasin), in keratinocytes, both in vitro and in vivo, which are implicated in the pathogenesis of psoriasis.[5]

Phototesting or UVB/UVA provocation testing may be conducted for diagnostic purposes, with the MED being assessed 24 h postexposure. After 24 h, the provocation test may reveal macular erythema, which occasionally accompanied by scaly plaques consistent with psoriasis. A skin biopsy is performed seven days after provocation testing, with histopathology showing acanthosis and inflammatory neutrophil infiltrates in the epidermis, indicating early psoriatic changes.[7,8] In the present case, a 24-year-old male experienced worsening of lesions after phototherapy initiated at a dose of 500 mJ/cm^2, which is within the MED range for Fitzpatrick skin type IV (450–600 mJ/cm^2).[15] However, provocation testing and biopsy post-UV exposure were not conducted for diagnostic confirmation.

Given that approximately 5.5%–24% of individuals with psoriasis have a genetic predisposition for photosensitive psoriasis,[5] awareness must be raised regarding the condition. Moreover, a thorough history taking, including any exacerbation of symptoms with sun exposure, and a clinical examination of sun-exposed areas are advised. In addition, findings from epidemiological studies suggest that photosensitive psoriasis is more prevalent in females, younger patients, and those with a family history of photosensitive psoriasis. With this understanding, dermatologists may consider substituting phototherapy with alternative systemic therapies for such individuals.

Conclusion

We report a case of psoriasis vulgaris that was exacerbated by phototherapy in a 24-year-old male, which presented as photosensitive psoriasis. Understanding the phenotype of photosensitive psoriasis is crucial for dermatologists to enable better management of this condition.

Conflict of Interest: There was no conflict of interest to be declared by any authors.

Funding Source: None.

Authors' Contribution

ETH, MA, MK: Diagnosis and management of the case, manuscript writing, has given final approval of the version to be published.

AB, ASS: Identification, diagnosis and management of the case, critical review of the manuscript, has given final approval of the version to be published.

References

1. Lim WL, How CH, Tan KW. Management of psoriasis in primary care. Singapore Med J. 2021;62: 109–12.
2. Papp KA, Gooderham MJ, Lynde CW, Poulin Y, Beecker J, Dutz JP, et al. Practical and Relevant Guidelines for the Management of Psoriasis: An Inference-Based Methodology. Dermatol Ther. 2022;12: 253–65.
3. Liao W, Singh R, Lee K, Ucmak D, Brodsky M, Atanelov Z, et al. Erythrodermic psoriasis: pathophysiology and current treatment perspectives. Psoriasis Targets Ther. 2016;Volume 6:93–104.
4. Kim WB, Jerome D, Yeung J. Diagnosis and management of psoriasis. Can Fam Physician Med Fam Can. 2017;63:278–85.
5. Wolf P, Weger W, Patra V, Gruber-Wackernagel A, Byrne SN. Desired response to phototherapy vs photoaggravation in psoriasis: what makes the difference? Exp Dermatol. 2016;25:937–44.
6. El-Komy MHM, Mashaly H, Sayed KS, Hafez V, El-Mesidy MS, Said ER, et al. Clinical and epidemiologic features of psoriasis patients in an Egyptian medical center. JAAD Int. 2020;1:81–90.
7. Buhl T, Krüger U, Emmert S, Peter Bertsch H, Mössner R. Photosensitive Psoriasis Vulgaris Inducible by a Single Suberythematous Dose of Ultraviolet B Irradiation. Acta Derm Venereol. 2008;88:414–6.
8. Rutter KJ, Watson REB, Cotterell LF, Brenn T, Griffiths CEM, Rhodes LE. Severely Photosensitive Psoriasis: A Phenotypically Defined Patient Subset. J Invest Dermatol. 2009;129:2861–7.
9. Wang F, Gao Y, Yuan Y, Du R, Li P, Liu F, et al. MicroRNA-31 Can Positively Regulate the Proliferation, Differentiation and Migration of Keratinocytes. Biomed Hub. 2020;5:1–12.
10. Xu X, Zhang Y, Pan Z, Zhang X, Liu X, Tang L, et al. Genome-wide DNA methylation of Munro's microabscess reveals the epigenetic regulation in the pathogenesis of psoriasis. Front Immunol [Internet]. 2022 [cited 2023 Oct 21];13. Available from: https://www.frontiersin.org/articles/10.3389/fimmu.2022.1057839
11. Nguyen TV, Wu JJ, Lim HW, Koo JYM. Acute Exacerbation of Erythrodermic Psoriasis with Phototherapy: Pathophysiology and Results of a National Psoriasis Foundation Survey regarding Photo-Management of Erythrodermic Skin. J Psoriasis Psoriatic Arthritis. 2016;1:142–6.
12. Yamanaka K, Yamamoto O, Honda T. Pathophysiology of psoriasis: A review. J Dermatol. 2021;48: 722–31.
13. Yamamoto T. Psoriasis and Connective Tissue Diseases. Int J Mol Sci. 2020;21:5803.
14. Hong AY, Lee SJ, Lee KB, Shin JW, Jeong EM, Kim IG. Double-Stranded RNA Enhances Matrix Metalloproteinase-1 and -13 Expressions through TLR3-Dependent Activation of Transglutaminase 2 in Dermal Fibroblasts. Int J Mol Sci. 2022;23:2709.
15. Czerwińska, Agniesszka E., Krzyscin, Janusz W. Analysis of Measurements and Modelling of the Biologically Active UV Solar Radiation for Selected Sites in Poland – Assessment of Photo-medical Effects. Publ Inst Geophys Pol Acad Sci Geophys Data Bases Process Instrum.