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# Coexistence of Psoriasis Vulgaris and Systemic Lupus Erythematosus: A Rare Clinical Case

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**Abstract**

Psoriasis vulgaris is a chronic skin inflammation characterized by the appearance of clearly limited erythematous plaques, along with rough, thick, and silvery-white scales. Systemic Lupus Erythematosus (SLE) is a multifactorial autoimmune disease with diverse clinical manifestations and can involve one or more organs. This case report presents a patient with Psoriasis Vulgaris with SLE. A 47-year-old woman presented with erythematous plaque lesions with thick scales and some hyperpigmentation, numerous, plaque-sized lesions with regional distribution on the scalp, right and left cubital areas, gluteal, left tibia, and yellow unguium dyschromia was seen on the distal lateral aspect, solitary on the right 3rd digit. Histopathological examination found typical Psoriasis Vulgaris. The patient was diagnosed with Psoriasis Vulgaris. In 2022, the patient was diagnosed with SLE and received Hydroxychloroquine sulfate therapy. The patient was treated with a combination therapy including methotrexate (MTX) and topical medications. This case highlights the importance of comprehensive evaluation for Psoriasis Vulgaris therapy with a previous history of SLE and the choice of treatment in patients with the coexistence of PV and SLE.



## 1. Introduction

Psoriasis vulgaris (PV) is a lifelong immune-mediated inflammatory skin disease. Psoriasis affects approximately 3% of the global population, with initial symptoms commonly appearing during the second decade of life. In the case of PV, the condition is driven by an immune system imbalance primarily involving T lymphocyte activity [1]. PV occurs worldwide, but its prevalence varies, and an estimated 60 million people suffer from PV, with an onset age of 30-39 years [2, 3]. The prevalence of PV in East Asia reaches 0.14%. Cases of PV in Indonesia reach 2.5% of the population [1, 3].

Systemic Lupus Erythematosus (SLE) is a multifactorial autoimmune disease with diverse clinical manifestations

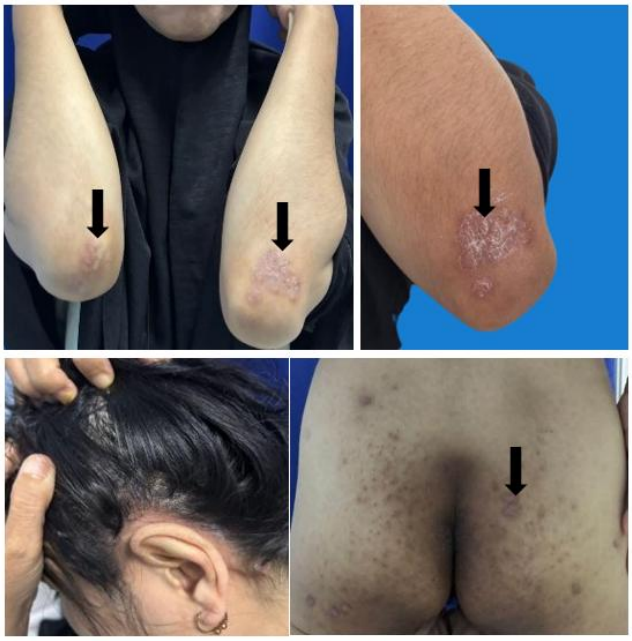

**Figure 1.** Clinical photos of the scalp, right and left cubital and gluteal regions.

**Figure 2.** Clinical photo of the 3rd right digit region.

and can involve one or more organs [4, 5]. The immunology of SLE involves dysregulation of the immune response, including innate and adaptive immunity. B lymphocytes play a key role in the adaptive immune response of SLE, which involves autoantibody production, autoantigen presentation, and autoreactive T cell activation [4].

Psoriasis and SLE are two inflammatory diseases with an autoimmune basis [6]. Although both show different skin manifestations, the risk of their occurrence is higher in individuals with other autoimmune diseases compared to the general population [7]. The incidence of coexistence of PV with SLE is considered very rare. This is due to the differences in pathogenesis. The pathogenesis of these autoimmune diseases involves the activation of different T cell subtypes: activation of Th1 cells in the case of PV, while activation of Th2 cells in the case of SLE. In the course of the disease, B lymphocytes and Th2 lymphocytes play a significant role in SLE, while in PV, Th1 lymphocytes play a dominant role. The only similarities between these two diseases are an increase in Th17 lymphocytes and increased serum levels of IL-7 and IL-23 [8].

As a physician, choosing treatment in patients with co-occurring PV and SLE is challenging. One of the main treatment methods for PV, which uses UV radiation for therapy, can cause and/or worsen not only skin lesions but also the course of SLE. Conversely, antimalarial drugs, which are the basis of SLE treatment, can cause or worsen PV skin lesions or even be associated with flare conditions in PV [9].

Hopefully, this case report can provide information regarding the coexistence of PV and SLE and appropriate therapy so that both autoimmune diseases can remain controlled and the patient's quality of life can improve.

## 2. Cases

A 47-year-old woman came to the RSUZA Dermatology and Venereology Polyclinic complaining of red spots accompanied by thickened scales on the scalp, elbows, and legs, and blackish spots on the buttocks that felt itchy. The patient admitted that the reddish and scaly rash, like dandruff on the head, had been felt since a young age, namely 30 years ago, but had worsened in the last 1 year. In 2022, the patient was diagnosed with SLE, namely with complaints of joint pain accompanied by weakness, and a drastic weight loss of ± 10 kg in 3 weeks, and was hospitalized for 1 week at the East Aceh Hospital. The patient currently has regular check-ups at the Internal Medicine Polyclinic and regularly takes medication. When not taking medication, the patient complains of bone and joint pain.

The patient also complained of itchy right fingernails, white lines appearing, becoming brittle with a change in color to cloudy yellow, and sometimes getting itchier. The patient currently has regular check-ups and regularly takes Hydroxychloroquine. When not taking medication, the patient complains of bone and joint pain. There are no family members with the same complaints.

Dermatological examination showed erythematous plaques with thick scales and some hyperpigmentation in the scalp region, right and left cubital, left tibial gluteal, multiple numbers, plaque size, and generalized distribution (Figure 1). In the 3rd digit region of the right hand, yellow dyschromia unguium is seen in the distal lateral part, solitary, and regionally distributed (Figure 2).

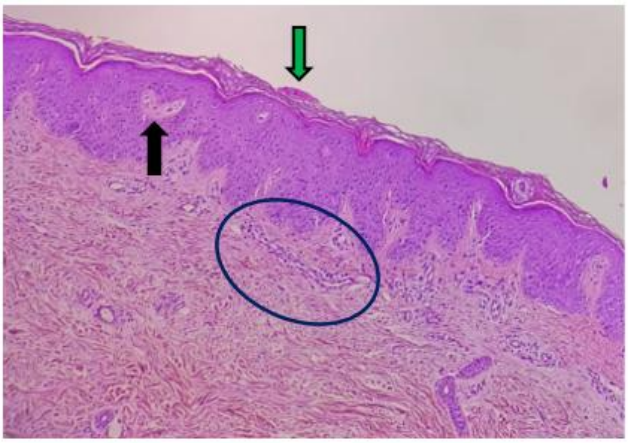

**Figure 3**. The epidermis layer is composed of squamous cells with normal cell nucleus morphology, minimal acanthosis, elongated rete edge, parakeratosis with focal infiltration of neutrophil cells (Munro microabscess) (green arrow), small focus of neutrophil inflammatory cell infiltration is also seen in the spongiosum layer (kogoj microabscess) (black arrow). In the sub epidermis layer, pervascular infiltration (blue circle). The stroma consists of fibrocollagen connective tissue with focal infiltration of neutrophil inflammatory cells and mature lymphocytes and the skin adnexal structure is also seen within normal limits. (H&E x100).

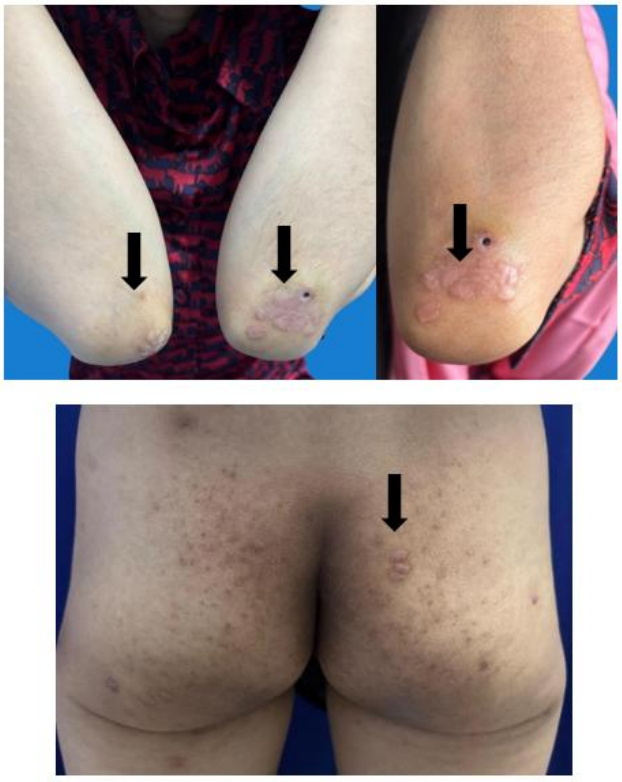

**Figure 4.** After 2 weeks of therapy, there was visible improvement in the lesion.

On physical examination, positive results were found on the kaarsvlek phenomenon and the austpitz sign. On histopathological examination, the epidermis layer is composed of squamous cells with normal cell nucleus morphology, minimal acanthosis, elongated rete edge, parakeratosis with focal infiltration of neutrophil cells (Munro microabscess) (green arrow), small focus of neutrophil inflammatory cell infiltration is also seen in the spongiosum layer (kogoj microabscess). In the subepidermis layer, pervascular infiltration. The stroma consists of fibrocollagen connective tissue with focal infiltration of neutrophil inflammatory cells and mature lymphocytes, and the skin adnexal structure is also seen within normal limits. (H&E x100) (Figure 3). The patient was diagnosed with psoriasis vulgaris. The Psoriasis Area Severity Index (PASI) score was 4.1, indicating mild severity.

The therapy given included a combination of systemic immunosuppressants for SLE, namely Hydroxychloroquine sulfate 200 mg tab once a day, mycophenolate mofetil 360 mg tab twice a day, and Rheu-trex tablet 2.5 mg 4 tablets per week. Clobetasol propionate 0.05% lotion was given on the scalp, and Momethason furoate 0.1% night cream was on the elbows, feet, and buttocks to control psoriasis. After 2 weeks of therapy, there was visible improvement in the lesion. Redness and itching decreased, and the scales also became thinner. The Psoriasis Area Severity Index (PASI) score was 2.6 (Figure 4).

### 3. Discussions

Based on the anamnesis and physical examination that has been done on a 47-year-old female patient in the skin and genital polyclinic of RSUDZA with chief complaints of red spots accompanied by thickened scales on the scalp, both elbows, and legs that do not disappear, as well as blackish spots on the buttocks that are itchy. Psoriasis vulgaris is characterized by chronic skin inflammation, characterized by the appearance of erythematous plaques that are limited, along with rough, thick, and silvery-white scales. These plaques generally occur in areas such as the elbows, knees, scalp, back, navel, and waist [6].

About 90% of PV cases are chronic plaque type. Skin symptoms in psoriasis can vary; one of them is psoriasis vulgaris, which is also known as plaque-type psoriasis, which is the most common type [10]. Psoriasis can occur at any age, but it is most common between 15 and 30 years. Human Leucocyte Antigen (HLA), especially HLA-Cw6, is associated with the onset of psoriasis at an early age and a family history of psoriasis [10, 11].

No family members have the same complaint in this patient. If there is no history of psoriasis in the parents, the risk of developing psoriasis is about 12% [11]. The diagnosis of psoriasis is usually based on clinical

examination. Physical examination includes examination of the primary lesion and common areas that are usually affected by psoriasis. Nails and joints should also be examined for changes that are consistent with psoriasis, and family history needs to be reviewed to clarify the diagnosis [12].

Psoriasis vulgaris is a long-term, hyperproliferative skin condition influenced by a combination of genetic predispositions and environmental triggers, affecting around 0.1–3% of the worldwide population. The disease is fueled by dysregulated Th1 and Th17 cell responses, which produce proinflammatory cytokines such as IFN-γ, IL-17, IL-22, and TNF-α, thereby intensifying skin inflammation. Th1 cells, in particular, contribute to psoriasis pathogenesis by increasing levels of IL-2, TNF-α, and IFN-γ. This heightened Th1 activity stimulates a cascade of inflammatory cytokines—IL-1, IL-6, IL-8, IL-12, IL-15, IP-10, and iNOS—further promoting keratinocyte overgrowth. IL-17, a central mediator, acts on keratinocytes, innate immune cells, and endothelial cells, playing a key role in driving the disease process [11, 12].

Psoriasis vulgaris is often measured by the severity of the disease using the Psoriasis Area Severity Index (PASI) score. The PASI score is obtained by visually examining the skin in four body areas: the head, trunk, arms, and legs. The PASI score is used to evaluate the effectiveness of treatment in clinical practice and clinical trials and to assist in decision-making regarding treatment [13]. PASI-75 indicates a 75% decrease in score from baseline. Meanwhile, PASI-90 and PASI-100 indicate a 90% and 100% decrease in scores from baseline, respectively. PASI-75 is the most important standard for assessing effectiveness in controlled clinical trials. Therapy aims to ensure that the skin is free of lesions or almost clear (PASI-90 or PASI-100) [11]. In this case, the patient's PASI results when coming to the clinic for a check-up were 4.1.

Systemic Lupus Erythematosus is a complex, multifactorial autoimmune disease, and the molecular mechanisms are still largely unknown. An ROS production and degradation imbalance leads to DNA oxidative modification, inducing DNA damage. Dendritic cells recognize self-antigens and present them to B cells, triggering an autoimmune response and excessive production of autoantibodies. Tfh cells help B cells enter and induce an autoimmune response, playing a critical role in the pathogenesis of SLE [11].

PV and SLE are both chronic autoimmune disorders known for their relapsing nature. According to published research, SLE occurs in approximately 0.69% of individuals with PV, whereas PV is identified in about 1.1% of patients who have a prior diagnosis of SLE. Interestingly, some studies indicate that PV is typically diagnosed before the onset of SLE. A notable clinical distinction between the two lies in the frequent involvement of the nail plate and genital region in PV, which is generally not observed in subacute cutaneous lupus erythematosus (SCLE). Beyond clinical presentation, histological features serve as crucial tools for differentiating the conditions. SLE is marked by interface dermatitis, characterized by lymphocytic infiltration and necroptosis of keratinocytes at the dermoepidermal junction. In contrast, PV lesions often exhibit parakeratosis, Munro microabscesses, caused by neutrophils migrating from the dermis into the epidermis, as well as elongation and slight edema of the dermal papillae [8].

Various approaches are used in the treatment of PV and SLE. The choice of treatment in patients with coexisting SLE and Ps is challenging. Phototherapy in PV can cause exacerbation in SLE. Conversely, administration of HCQ in SLE can cause worsening of PV lesions. In this patient, since 2022, he has been receiving HCQ. This is what is thought to worsen the patient's condition. An effective systemic therapeutic agent for PV and SLE is folic acid analog methotrexate (MTX). MTX inhibits dihydrofolate reductase, reduces the synthesis of pyrimidines and purines, and ultimately inhibits the division of rapidly dividing cells, such as keratinocytes and bone marrow cells. In addition to its antiproliferative effects, MTX has anti-inflammatory and immunosuppressive activities. This patient received MTX therapy at a dose of 10 mg per week. After 2 weeks of therapy, the patient's condition improved. Psoriatic lesions thinned, itching decreased, and the patient's SLE was controlled [11].

It is important to coordinate with the internal medicine department regarding administering HCQ to patients so that PV lesions improve and SLE remains under control. The researcher's suggestion for the future is that further research can be carried out on other therapies that can be used as alternative treatments for SLE in PV patients so as not to worsen the patient's condition.

## 4. Conclusions

Psoriasis vulgaris and SLE are two autoimmune diseases that can occur simultaneously, although they have different pathophysiological mechanisms. The incidence of PV coexisting with SLE is considered very rare. This case highlights the challenges in managing patients with the coexistence of psoriasis vulgaris and SLE, where therapy used in one condition can worsen the other.

The choice of treatment in patients with coexisting SLE and Ps is challenging. Folic acid analog MTX is an effective systemic therapeutic agent for PV and SLE. MTX has anti-

inflammatory and immunosuppressive activities, without worsening the conditions of PV and SLE. It is important to coordinate with the internal medicine department regarding the issue of administering HCQ to patients so that PV lesions improve and SLE remains under control.

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