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Secondary syphilis psoriasiform in HIV-infected patients: A case series

Katia Fitriani, Alessandro Alfieri*, Nurwestu Rusetiyanti, Devi Artami Susetiati, Niken Trisnowati, Satiti Retno Pudjiati

Department of Dermatology and Venereology, Faculty of Medicine, Public Health and Nursing, Universitas Gadjah Mada, Yogyakarta, Indonesia/Dr. Sardjito General Hospital, Yogyakarta, Indonesia

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ABSTRACT

Psoriasiform secondary syphilis is an uncommon and diagnostically challenging variant of secondary syphilis that can closely mimic psoriasis vulgaris, particularly in people with HIV. This case series adds to the limited literature from resource-limited settings by illustrating how psoriasiform secondary syphilis may be misinterpreted as psoriasis both clinically and histopathologically, and how repeated clinicopathologic correlation is essential to avoid inappropriate immunosuppression. We reported three HIV-infected male patients who presented with generalized psoriasiform erythematous scaly plaques, some with palmoplantar involvement, initially diagnosed as psoriasis. One patient had been treated with methotrexate for several months without clinical improvement. Serologic testing in all cases demonstrated active syphilis with reactive nontreponemal and treponemal tests, including a very high venereal disease research laboratory (VDRL) titer in one patient, and all were confirmed HIV-positive. Initial histopathologic examinations variably suggested secondary syphilis or psoriasis; in two patients, repeat biopsy or deeper sectioning was required to reveal plasma cell–rich perivascular infiltrates and vascular changes consistent with secondary syphilis, while one case was ultimately considered to represent coexistence of psoriasis and syphilis. All patients received intramuscular benzathine penicillin G according to syphilis stage, with additional topical or systemic antiinflammatory therapy when indicated, and showed clinical improvement. In conclusion, psoriasiform secondary syphilis should be routinely considered in the differential diagnosis of psoriasiform eruptions in individuals with sexually transmitted infection risk or known HIV infection, and that discrepant clinical, serologic, and histopathologic findings warrant repeat biopsy, deeper sectioning, and multidisciplinary review.

Keywords:
syphilis;
Treponema pallidum;
sexual transmitted infection;
HIV;
psoriasiform

ABSTRAK

Sifilis sekunder psoriasiformis merupakan varian sifilis sekunder yang jarang dan menantang secara diagnostik karena sangat mirip psoriasis vulgaris, terutama pada orang dengan HIV. Salah interpretasi dapat berujung pada pemberian imunosupresan yang tidak tepat. Laporan seri kasus ini bertujuan menggambarkan perangkap klinis dan histopatologis serta menekankan pentingnya korelasi klinikopatologis berulang. Dilaporkan tiga pasien laki-laki dengan HIV yang datang dengan plak eritem psoriasiformis berskuama generalisata, sebagian dengan keterlibatan palmoplantar, yang seluruhnya awalnya didiagnosis sebagai psoriasis. Seorang pasien telah menerima metotreksat selama beberapa bulan tanpa perbaikan. Pemeriksaan serologi pada semua kasus menunjukkan sifilis aktif dengan tes nontreponemal dan treponemal reaktif, termasuk satu pasien dengan titer *venereal disease research laboratory* (VDRL) sangat tinggi. Pemeriksaan histopatologi awal bervariasi, sebagian mengarah ke sifilis sekunder, sebagian menyerupai psoriasis; pada dua pasien diperlukan biopsi ulang atau pemotongan lebih dalam untuk menyingkap infiltrat perivaskular kaya sel plasma dan perubahan vaskular yang konsisten dengan sifilis sekunder, sedangkan satu kasus dinilai sebagai koeksistensi psoriasis dan sifilis. Semua pasien mendapat benzatin penisilin G intramuskular sesuai stadium sifilis, disertai terapi antiinflamasi topikal atau sistemik bila diperlukan, dengan perbaikan klinis bermakna. Simpulan, sifilis sekunder psoriasiformis perlu rutin dipertimbangkan sebagai diagnosis banding erupsi psoriasiform pada individu dengan faktor risiko infeksi menular seksual atau infeksi HIV. Ketidaksesuaian temuan klinis, serologis, dan histopatologis sebaiknya mendorong dilakukannya biopsi ulang, pemotongan jaringan yang lebih dalam, dan diskusi multidisipliner untuk mencegah terapi imunosupresif yang tidak tepat dan keterlambatan penatalaksanaan sifilis.

*corresponding author: andro.alfieri@gmail.com

INTRODUCTION

Syphilis is a chronic, systemic sexually transmitted infection caused by Treponema pallidum, characterized by multistage clinical manifestations and highly diverse cutaneous morphologies, hence the designation "the great imitator".[1,2] Secondary syphilis typically presents with a generalized, non-pruritic papulosquamous exanthem involving the trunk and extremities, often accompanied by palmoplantar lesions, mucous patches, condyloma lata, and systemic symptoms such as fever, malaise, and lymphadenopathy.[3–6] A wide spectrum of atypical variants has also been described, including psoriasiform, lichenoid, nodular, pustular, annular, and corymbiform patterns, which may closely resemble other inflammatory dermatoses and delay appropriate etiologic treatment.[7]

Human immunodeficiency virus (HIV) infection alters both cellular and humoral immune responses through progressive $CD4^+$ T-cell depletion and immune dysregulation, thereby modifying the natural course, severity, and clinical expression of many infections, including syphilis.[2,4,8] Syphilis–HIV co-infection is common due to shared routes of transmission and overlapping risk groups, particularly men who have sex with men and individuals with high-risk sexual behavior. In HIV-infected patients, secondary syphilis may present more extensively and aggressively, with overlapping stages, unusual morphologies, and atypical serologic profiles, and may be associated with an increased risk of neurologic involvement and treatment failure.[7]

Psoriasiform secondary syphilis is an uncommon but clinically important variant in which erythematous scaly plaques closely resemble psoriasis vulgaris in both morphology and distribution, including palmoplantar and typical psoriatic sites.[1,4,9,10] Patients may initially receive topical or systemic antipsoriatic or other immunosuppressive therapies under a presumed diagnosis of psoriasis, which can further delay recognition of the underlying infectious etiology.[11] Histopathologic features may also overlap, particularly when only limited sections are evaluated, because psoriasiform secondary syphilis can show psoriasiform epidermal hyperplasia and parakeratosis, while lymphoplasmacytic perivascular infiltrates and vascular changes may be subtle or focal, making careful clinicopathologic correlation and serologic testing indispensable.[7,12,13]

In Indonesia, where the burden of HIV and other sexually transmitted infections remains substantial and access to dermatopathology and lack of specialized care is uneven,[14] misdiagnosis of atypical syphilis as primary inflammatory dermatoses such as psoriasis may have important consequences for patient outcomes and ongoing transmission. The present three-case series of HIV-infected male patients with psoriasiform eruptions ultimately attributed to secondary syphilis, including one case with probable coexistence of psoriasis, contributes local evidence on this rare but relevant presentation and underscores the need for heightened clinical suspicion, routine syphilis–HIV screening, and close collaboration with pathologists in Indonesian practice. The main aim of this case report is to describe the clinical and histopathologic characteristics and therapeutic outcomes of psoriasiform secondary syphilis in HIV-infected patients, and to highlight its implications for diagnosis and management of psoriasiform dermatoses in the Indonesian health-care setting.

CASE

Case 1

A 52 y.o. man presented with

erythematous patches on several body areas, previously diagnosed as psoriasis vulgaris at a private hospital. The eruption began 3 mo earlier as non-pruritic erythematous patches on the palms and soles. He received methotrexate for 1 mo without improvement, after which therapy was switched to cyclosporine for another month; the lesions then worsened and became pruritic. He also reported diffuse hair loss, facial darkening, jaundice, 30 kg weight loss over 3 mo, persistent oral ulcers, and recurrent upper respiratory infections. He denied high-risk sexual behavior, tattoos, and injection drug use, but reported a blood transfusion 11 yr earlier.

Cutaneous examination of the nape, chest, hands, and lower legs revealed multiple erythematous patches and plaques with irregular borders and fine scaling (FIGURE 1). Auspitz sign was negative. Laboratory tests showed VDRL 1:64, reactive TPHA, and reactive anti-HIV-1/anti-HIV-2/ELISA, with an absolute CD4 count of 115 cells/μL. Skin biopsy demonstrated diffuse epidermal parakeratosis, hypogranulosis, basal cell vacuolization, mild spongiosis, and a diffuse subepidermal infiltrate of lymphocytes, plasma cells, neutrophils, and eosinophils, with vascular proliferation and endothelial cell plumping, without malignancy—findings more consistent with secondary syphilis than psoriasis (FIGURE 2).

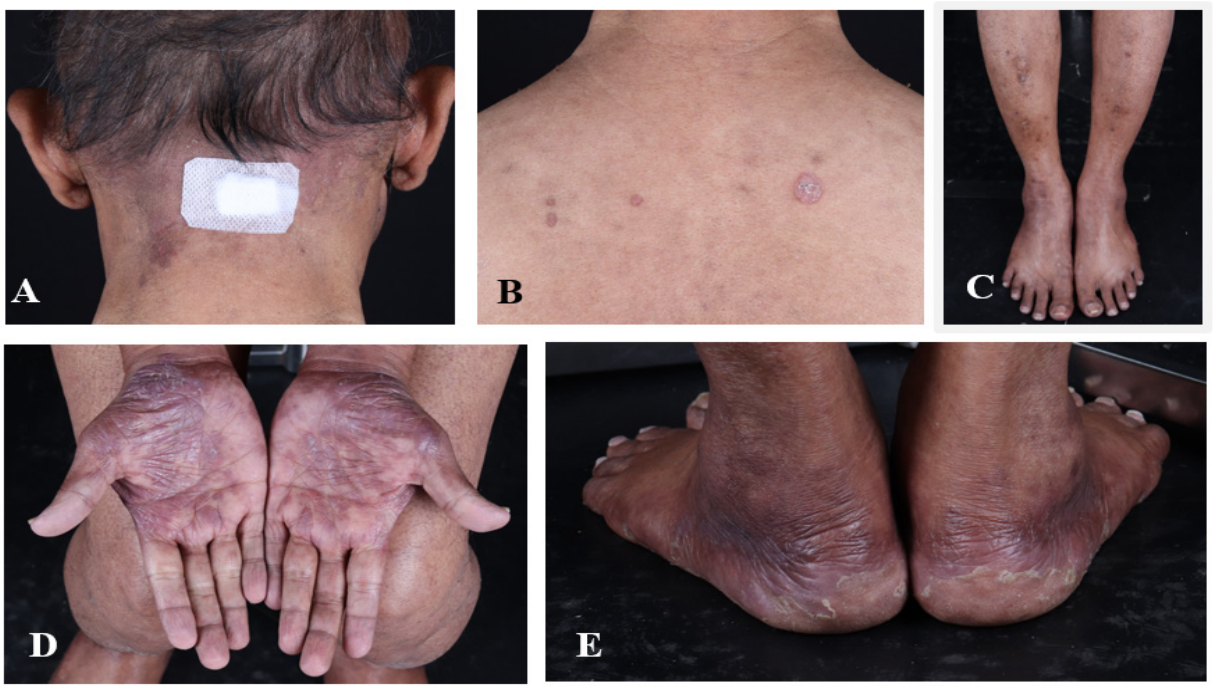

FIGURE 1. Several psoriasiform skin lesions. A. Erythematous patch on the nape; B. Erythematous scaly patch on the back; C–E. Scaly erythematous and hyperpigmented patches on both lower legs and hands.

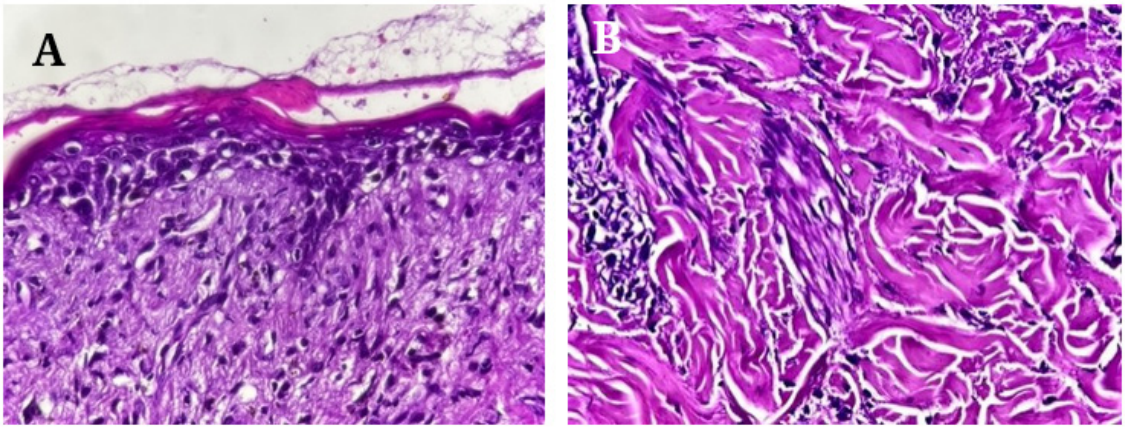

FIGURE 2. Histopathologic findings showed. A. epidermal parakeratosis, basal cell vacuolization, and mild spongiosis; B. dermal infiltrate composed of lymphocytes, plasma cells, neutrophils, and eosinophils.

The patient was diagnosed with psoriasiform secondary syphilis in the setting of HIV infection, and methotrexate was discontinued. He was treated with intramuscular benzathine penicillin G 2.4 million IU and referred for multidisciplinary HIV care and antiretroviral therapy. Three months later, there was marked improvement of the skin lesions and a decline in VDRL titer to 1:16.

Case 2

A 25 y.o. man presented with widespread erythematous scaly patches and plaques involving almost the entire body. Three months earlier, he had been diagnosed at a primary health center with secondary syphilis and HIV infection and treated with intramuscular benzathine penicillin 2.4 million IU weekly for 3 doses and ART (tenofovir–lamivudine–dolutegravir). The skin lesions did not improve, and he discontinued ART for 3 mo, during which the eruption became more extensive and scaly, with tense vesicles that ruptured and formed crusts.

He reported painful, non-pruritic plaques, nail detachment, plantar desquamation, and spinal arthralgia without systemic respiratory symptoms. His sexual history revealed early onset of sexual activity with multiple male partners, inconsistent condom use, and receptive oral and anal intercourse. The last sexual contact, 3 mo earlier, was with a male partner with genital ulcer disease and multiple partners. Vital signs were normal, with bilateral inguinal lymphadenopathy.

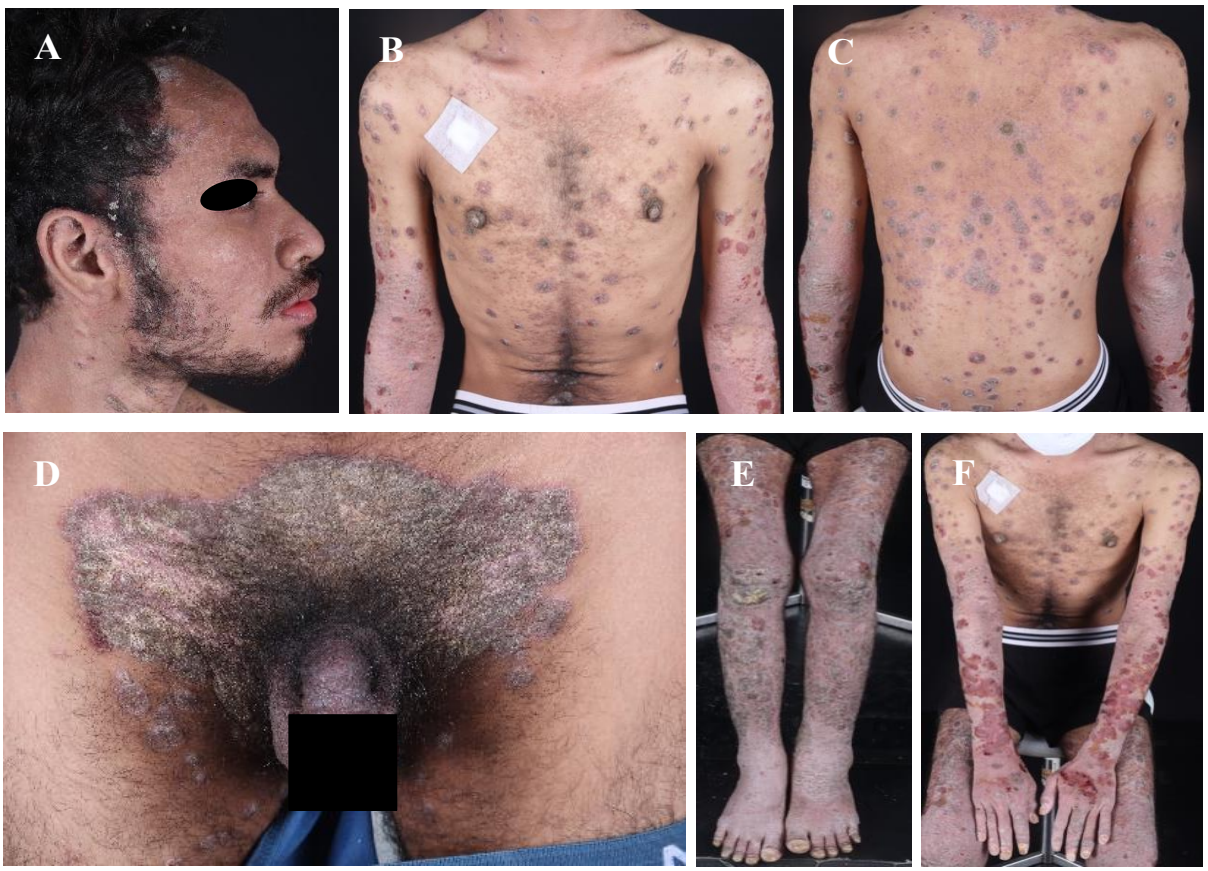

FIGURE 3. Well-demarcated erythematous patches and plaques, some covered with yellow–brown scale and dark brown crusts on A. the face; B &C. trunk; D. inguinal region; and E & F. extremities.

Cutaneous examination showed sharply demarcated erythematous plaques with yellow-brown scale and crusts on the scalp and retroauricular areas; erythematous papules and plaques with scale or central crusted erosions on the chest and back; hyperkeratotic crusts on the areolae; erythematous macules with desquamation on the palms and soles; periungual erythematous plaques with scale, desquamation, and onychodystrophy; and sharply demarcated polycyclic erythematous plaques with thick white–brown confluent scale on the pubic region, scrotum, penis, and inguinal folds (FIGURE 3). Auspitz sign and Koebner phenomenon were negative. Differential diagnoses included secondary syphilis with psoriasiform features, psoriasis, seborrheic dermatitis, talaromycosis, and histoplasmosis.

Laboratory investigations revealed severe anemia and elevated liver function. Peripheral blood morphology suggested anemia most likely due to iron deficiency in the setting of inflammation or infection (serum iron 7 µg/dL, TIBC 137 µg/dL). Syphilis serology showed reactive TPHA and a VDRL titer of 1:16; baseline CD4 count was 47 cells/µL. A repeat course of benzathine penicillin 2.4 million IU weekly for 3 doses led to a reduction in VDRL titer to 1:8 at 1 mo.

Skin biopsy demonstrated epidermal hyperkeratosis, hypergranulosis, focal erosion and ulceration, spongiosis, lymphocytic exocytosis, and intraepidermal microabscesses composed of lymphocytes, histiocytes, and plasma cells. The upper to deep dermis was edematous with patchy

interstitial and mild periadnexal infiltrates of lymphocytes, histiocytes, plasma cells, and neutrophils, with melanin dropping. No fungal elements were seen on PAS staining, and tissue culture was negative for fungi. These findings were consistent with secondary syphilis.

Based on the clinical features, serology, and histopathology, a diagnosis of secondary syphilis in a patient with advanced HIV infection was established. The patient was hospitalized for stabilization, re-initiation of TLD-based ART, and co-management with internal medicine for correction of anemia and liver dysfunction; he received four units of packed red cells, and ART was temporarily withheld until liver enzymes improved. Cutaneous disease was controlled with oral methylprednisolone 16 mg/day (tapered over 2 wk) and topical coconut oil plus clobetasol propionate twice daily. At three-month follow-up, the eruption had largely resolved, leaving hypo- and hyperpigmented macules with a few residual erythematous patches bearing fine scale.

Case 3

A 24 y.o. man presented with erythematous scaly lesions on the palms and soles of 2 mo duration. The eruption began as thick, white, callus-like scaly erythematous patches on both palms and soles, rapidly extending but remaining asymptomatic. He had been seen by a dermatologist and treated with topical corticosteroid without clear diagnosis. The lesions progressed with loss of eyebrows and eyelashes, erythematous scaly patches around the mouth, and subsequent involvement of the elbows, knees, and lower legs. He denied fever or loss of appetite but reported generalized myalgia.

Three weeks prior, he was diagnosed with HIV infection (baseline $CD4^+$ 41 cells/μL) and started on tenofovir–lamivudine–efavirenz, and had been diagnosed with cytomegalovirus chorioretinitis by ophthalmology. There was no personal or family history of similar skin disease and no recalled painless genital or anal ulcer. He reported intermittent paracetamol use, no other significant comorbidities, and no new topical exposures (long-term use of the same bath soap and detergent). He identified as homosexual with 2 male partners, as a receptive ano-genital and oro-genital sex, with inconsistent condom use.

Examination showed alopecia of the eyebrows and eyelashes; erythematous plaques with overlying white scale on the cheeks, perioral region, and chin; and white desquamation on the lips. Bilateral symmetric diffuse erythematous patches with desquamation were present on the palms and soles, with additional thick, white, scaly erythematous plaques on the lower back and dorsal hands and feet and annular plaques with layered white scale on both elbows and knees. Auspitz and Karsvlek signs were negative and no Koebner phenomenon was observed. Penile and scrotal skin appeared xerotic with scattered white scales and a solitary scrotal erosion (FIGURE 4). Initial differentials included secondary syphilis, psoriasis vulgaris, seborrheic dermatitis, and palmoplantar keratoderma in a patient with AIDS. KOH examination was negative for Malassezia, while TPHA was reactive and VDRL was 1:512.

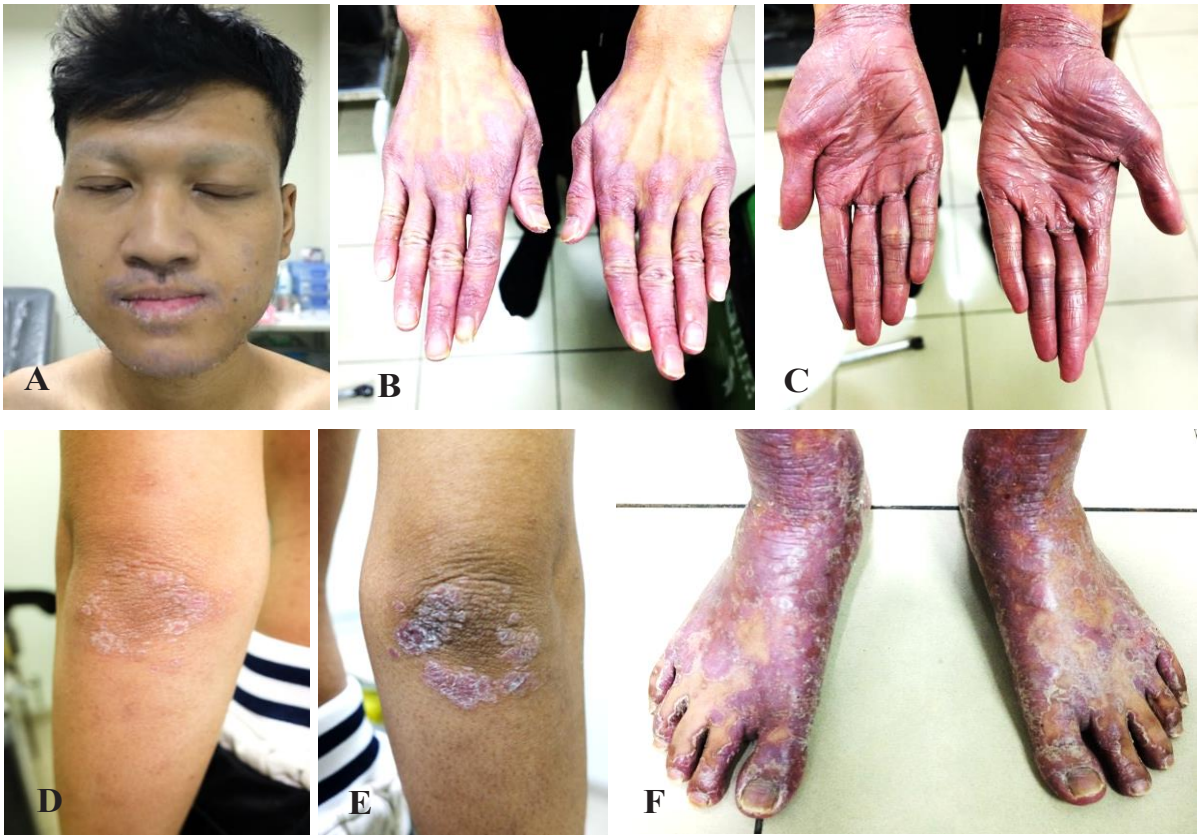

FIGURE 4. A. alopecia of the eyebrows and eyelashes; well-demarcated erythematous plaques are seen, some covered by overlying white scale B. on the dorsal hands, C. palms, D-E. elbows and F. feet.

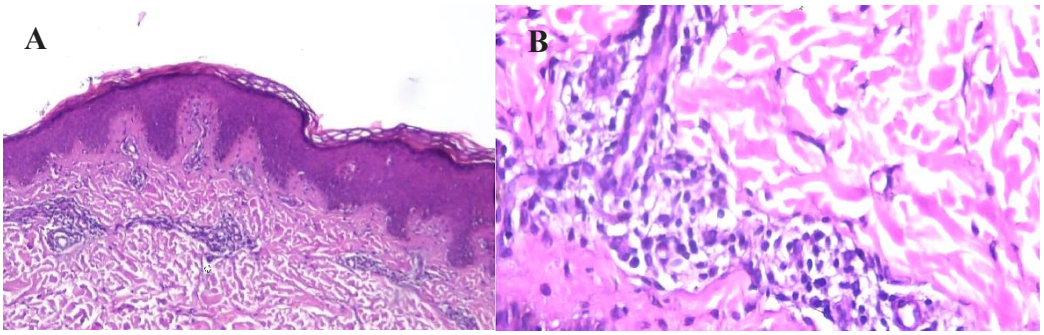

FIGURE 5. A. orthokeratosis with mild lymphocytic exocytosis in the epidermis; B. vascular proliferation with a moderate lymphocytic infiltrate and a few plasma cells and neutrophils.

Initial histopathology from a right elbow biopsy showed parakeratosis and orthokeratosis, hypogranulosis, spongiosis with lymphocytic exocytosis and a few neutrophils, thinning of the suprapapillary plates with dilated papillary vessels, and a moderate predominantly perivascular lymphohistiocytic infiltrate with few neutrophils in edematous stroma, interpreted as psoriasiform dermatitis consistent with psoriasis vulgaris. Because of discordance with strongly positive syphilis serology, deeper sectioning and re-evaluation were performed, revealing orthokeratosis with minimal lymphocytic exocytosis and, in the dermis, vascular proliferation with a perivascular infiltrate of lymphocytes with a few plasma cells and neutrophils. Hypogranulosis, suprapapillary plate thinning, and Munro microabscesses were absent (FIGURE 5), and the revised impression was compatible with secondary syphilis.

Integrating the clinical, serologic, and repeat histopathologic findings, a working diagnosis of secondary syphilis in a patient with AIDS was made. The patient received a single intramuscular dose of benzathine penicillin 2.4 million IU and topical *Aloe vera*–containing petrolatum twice daily. At 2-wk follow-up, the cutaneous lesions had improved. A 1-mo post-treatment serologic reassessment was scheduled, but the patient had not returned for further follow-up at the time of this report.

DISCUSSION

This case series underscores the diagnostic complexity of psoriasiform secondary syphilis in HIV-infected patients and highlights the importance of integrating clinical, histopathologic, and serologic data. All three patients presented with psoriasiform plaques clinically similar to psoriasis vulgaris, and in two cases, initial or subsequent histopathologic interpretations favored psoriasis, illustrating substantial overlap.

Secondary syphilis is classically characterized by a generalized papulosquamous exanthem with palmoplantar involvement, mucous patches, and systemic symptoms.[2–4,15] In HIV-infected patients, cutaneous manifestations may be more extensive, more severe, or display atypical morphologies. Psoriasiform variants have been increasingly reported, in which lesions may mimic plaque psoriasis both in distribution and morphology.[9,16]

Histopathologically, secondary syphilis typically shows a superficial and deep perivascular and periadnexal inflammatory infiltrate enriched with plasma cells, endothelial swelling, and variable interface dermatitis.[7,17,18] Psoriasiform secondary syphilis may additionally demonstrate psoriasiform epidermal hyperplasia and parakeratosis, leading to confusion with psoriasis.[18,19] Conversely, HIV-associated psoriasis may show non-specific inflammatory changes and may occasionally lack classic psoriatic features, further complicating differentiation.[7,12]

In Case 1, misclassification of the eruption as psoriasis resulted in prolonged methotrexate therapy without clinical benefit and delayed etiologic treatment. The absence of elicited high-risk sexual behavior at initial presentation and anchoring bias toward psoriasis contributed to the diagnostic delay. Only after therapeutic failure and re-evaluation, including biopsy and serology, was the correct diagnosis of secondary syphilis with newly identified HIV infection established.

Case 2 illustrated the potential coexistence of psoriasis and syphilis in an HIV-infected individual. Syphilis is known to modulate immune responses, and HIV infection further alters the Th1/Th2/Th17 balance, which is relevant in psoriasis pathogenesis. It is plausible that infection and immune dysregulation

unmasked or exacerbated underlying psoriasis. Sequential biopsies in this patient showed a shift from syphilis-consistent features to more typical psoriatic changes, emphasizing that a single biopsy may not fully represent the disease spectrum and that lesions may evolve over time.

Case 3 emphasized the critical importance of reconciling histopathology with clinical and serologic findings. The very high VDRL titer and strong epidemiologic context strongly suggested active secondary syphilis, in conflict with the initial diagnosis of psoriasis. Deeper sectioning and careful search for plasma cell–rich infiltrates led to reinterpretation compatible with secondary syphilis. This case highlights the value of deeper levels and repeated review by dermatopathologists whenever clinicopathologic discordance arises.

Across these 3 cases, several recurring themes emerge: 1). High index of suspicion: Psoriasiform eruptions, especially with palmoplantar involvement, poor response to antipsoriatic therapy, or systemic symptoms, should prompt consideration of secondary syphilis, particularly in patients with STI risk factors or known HIV infection.[2,10,20] 2). Comprehensive sexual history and serologic testing: Thorough sexual history-taking and routine syphilis and HIV serology are essential in atypical psoriasiform dermatoses.[2,4,20] 3). Need for clinicopathologic correlation: Histopathology alone may be insufficient to differentiate psoriasis from psoriasiform secondary syphilis. Plasma cell–rich infiltrates, endothelial swelling, and a superficial and deep perivascular pattern favor syphilis but may be subtle or focal.[4,18,19] 4). Repeat biopsy and deeper sectioning: When initial histopathologic findings are inconclusive or discordant with the clinical picture and serology, repeat biopsy from a different lesion and deeper sectioning of existing blocks should be considered. 5). Multidisciplinary management: Close collaboration between dermatologists, infectious disease physicians or HIV clinicians, and dermatopathologists is essential for timely and accurate diagnosis, appropriate antimicrobial therapy, and optimization of ART and immunomodulatory treatment.

Early recognition and appropriate treatment of secondary syphilis in HIV-infected patients are crucial not only to prevent progression and systemic complications but also to avoid unnecessary or potentially harmful immunosuppressive therapy given under the assumption of primary inflammatory dermatoses such as psoriasis.

CONCLUSION

This three-case series illustrates that psoriasiform secondary syphilis in people living with HIV can closely mimic psoriasis both clinically and histopathologically, leading to misdiagnosis and potentially inappropriate immunosuppressive treatment, particularly in resource-limited settings such as Indonesia. Our findings underscore the need for a high index of suspicion for secondary syphilis in any psoriasiform eruption with palmoplantar involvement, atypical features, poor response to standard antipsoriatic therapy, or the presence of HIV and other sexually transmitted infection risk factors. We recommend that clinicians systematically obtain a detailed sexual history, perform syphilis and HIV serology in atypical or treatment-resistant psoriasiform dermatoses, and request skin biopsy with willingness to repeat or deepen sectioning when clinicopathologic discordance arises. Multidisciplinary collaboration between dermatologists, infectious disease/HIV specialists, and dermatopathologists is essential to

optimize diagnosis, guide appropriate benzathine penicillin therapy and antiretroviral management, and avoid unnecessary immunosuppression. Strengthening awareness and diagnostic pathways for atypical syphilis presentations at Indonesian referral centers may improve patient outcomes and contribute to better control of syphilis–HIV co-infection nationally.

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