Patch-type kaposi sarcoma as an initial sign of hiv infection with syphilis coinfection: A case report

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SUMMARY

Kaposi sarcoma (KS) is a vascular neoplasm associated with advanced Human Immunodeficiency Virus (HIV)-related immunosuppression. The patch stage of KS is often challenging for clinicians because it can mimic benign dermatoses, thereby delaying diagnosis. We report a 41-year-old male presenting with multiple violaceous rashes whose dermoscopic and histopathological features are consistent with the patch stage of KS. Laboratory tests confirmed HIV and syphilis infections. The coexistence of KS, HIV, and syphilis highlights the relationship between immunosuppression and sexually transmitted infections (STIs). Early detection of subtle KS lesions enabled timely HIV testing, appropriate antimicrobial therapy, and rapid initiation of antiretroviral treatment, thereby preventing potential progression to visceral or nodular KS. This case highlights the importance of clinical vigilance for early signs of KS, routine STI screening in patients with unusual skin lesions, and the use of skin biopsy to distinguish KS from its mimickers. The report provides insights into the diagnostic challenges posed by early KS and highlights the importance of integrating evaluations of dermatologic and infectious diseases in high-risk populations.

INTRODUCTION

Kaposi sarcoma (KS) is a human herpesvirus-8 (HHV-8)-associated vascular neoplasm that may appear as violaceous macules, patches, plaques, or nodules, with potential mucosal and visceral involvement. It is clinically classified into four epidemiological types: classic, endemic, iatrogenic, and epidemic or HIV-associated KS.[1-2] Although KS is relatively uncommon worldwide, a population-based analysis estimated 34,270 new cases in 2020, with an age-standardised incidence rate of 0.39 per 100,000 population.[3] Importantly, the global burden of KS remains closely linked to HIV infection, with more than two-thirds of KS cases estimated to be HIV-attributable.[4]

Indonesia continues to face a concentrated HIV epidemic, with an estimated 570,000 people living with HIV and a disproportionate burden among key populations, including men who have sex with men, sex workers, transgender people, and people who inject drugs.[5-6] This pattern is clinically relevant because HIV and sexually transmitted infections (STIs) often share overlapping transmission routes and biological interactions. Syphilis, in particular, may increase susceptibility to HIV through mucosal ulceration and inflammation, while HIV infection may alter the clinical course of syphilis.[7] In Indonesia, syphilis remains an important public health problem, with 76,923 new cases reported in 2020.[8]

Early patch-stage KS is diagnostically challenging because its subtle violaceous lesions may resemble benign vascular or inflammatory dermatoses. This can delay recognition of underlying immunosuppression, particularly when KS appears before HIV has been diagnosed. Histopathological examination, supported by HHV-8 immunohistochemistry when available, is therefore important in early or atypical lesions.[1-2] This report presents patch-type Kaposi sarcoma as the initial clinical sign of HIV infection with late latent syphilis, highlighting the importance of early biopsy, HIV testing, and comprehensive STI screening in patients with suspicious violaceous skin lesions.

CASE PRESENTATION

A 41-year-old Russian male, married and residing in Bali for the past two years, presented to the Dermatology and Venereology Outpatient Clinic of Ngoerah Hospital, Bali, in June 2025, with a one-month history of multiple non-pruritic, non-painful erythematous-to-violaceous rash on the nose, trunk, and upper and lower extremities. The lesions initially appeared on the left hand and gradually involved the nasal region, right arm, trunk, and left leg. He also reported one month of watery diarrhea and unintentional weight loss, although no objective measurements were available. The patient had self-medicated with metronidazole and hyoscine butylbromide, without recalling doses or temporal relation to the rash. He denied fever, respiratory symptoms, mucosal lesions, genital ulcers, urethral discharge, prior drug allergy, or systemic comorbidities. Social and sexual history revealed more than ten lifetime female partners, inconsistent condom usage, and the last sexual contact was three months prior. He denied a history of transfusion, tattoos, intravenous drug use, alcohol consumption, or smoking.

On physical examination, vital signs were stable, and the general condition was good. Dermatological assessment revealed multiple well-defined erythematous-to-violaceous macules, patches, papules, and plaques, round to oval in shape, measuring 0.5–3 cm, distributed discretely on the nasal region, bilateral arms, trunk, and left leg. Mucosal abnormalities, Koebner phenomenon, or stigmata of atopy were negative.

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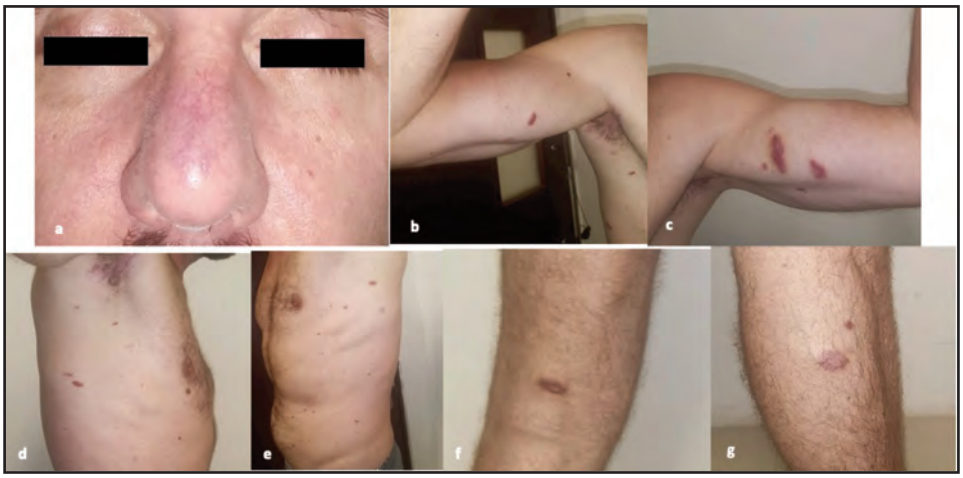

Fig. 1: a-g Clinical photographs on the nasal, right and left brachial, trunk, and left crural regions

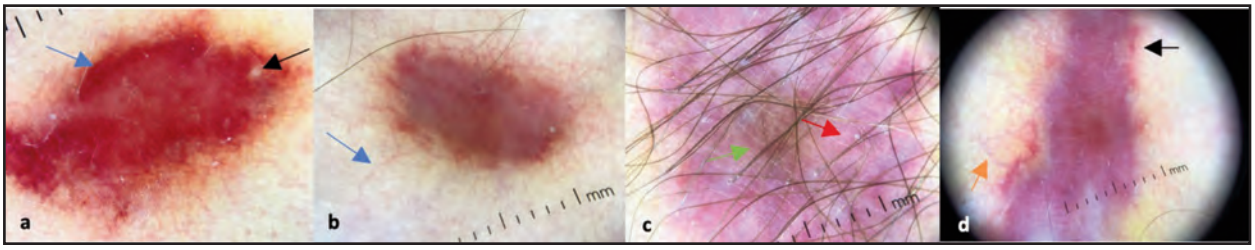

Fig. 2: a-d Dermoscopy examination

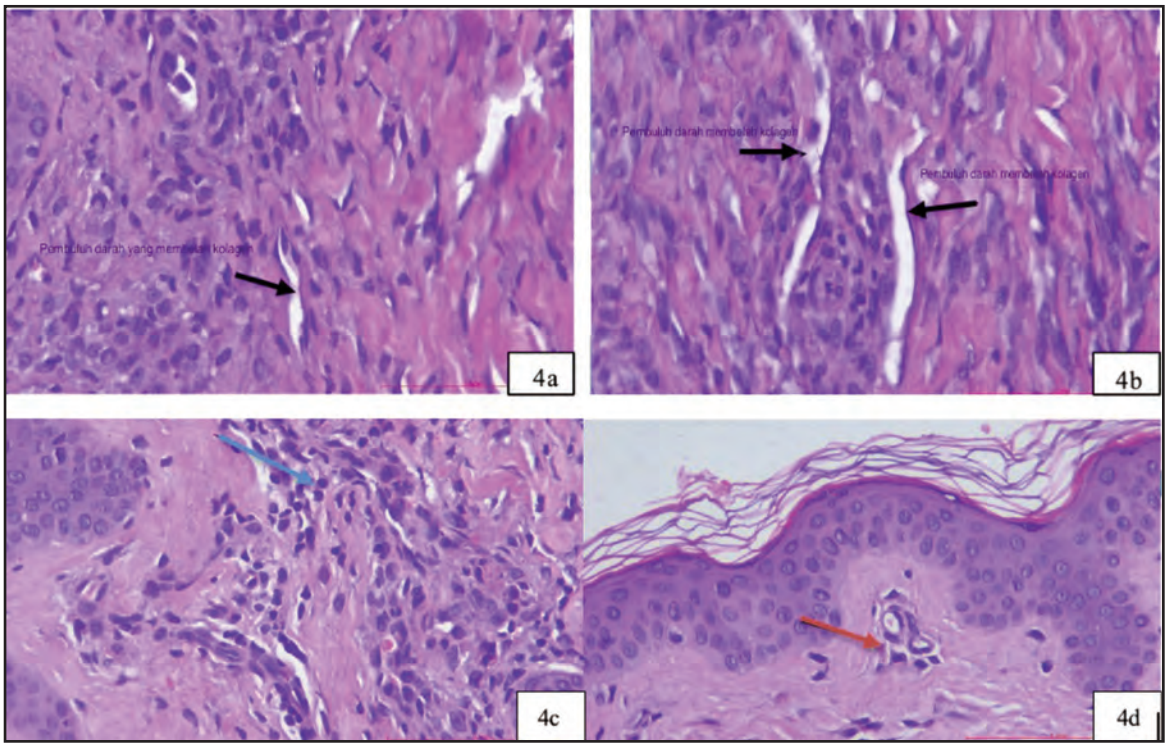

Fig. 2: a-d Histopathological findings

Dermoscopic examination showed polychromatic "rainbow pattern," serpentine, dotted, and coiled vessels, and white lines and clods, features suggestive of Kaposi sarcoma. In the macular–patch lesions, structureless reddish-violaceous areas with fine arborizing vessels at the lesion margins were observed (blue arrows). In the papular–plaque lesions, white scaling and brownish blotches on a reddish-violaceous background (green arrows), along with fragmented vessels at the lesion margins (orange arrows), were noted. A homogeneous polychromatic discoloration forming a rainbow pattern, consisting of light pink, dark red, and violaceous hues, was evident. Serpentine vessels (blue arrows) and dotted and coiled vessels (black arrows) were visible at the lesion margins. White lines and white clods were also observed (indicated by red arrows).

Histopathology of a macular lesion in the left brachial region showed dermal proliferation and dilated vascular channels dissecting through the dermal collagen bundles, some demonstrating a promontory sign (black arrows). Mild perivascular lymphoplasmacytic inflammation is noted (blue arrows), along with scattered hemosiderin pigment (red arrows). Numerous vascular channels surround eccrine structures. The differential diagnosis is consistent with the macular stage of Kaposi sarcoma.

Provider-initiated HIV testing was reactive. Laboratory investigations revealed anemia (Hb 10.5 g/dL), mild thrombocytopenia, elevated liver enzymes, CD4 192 cells/µL, CD4/CD8 ratio 0.11, reactive anti-Cytomegalovirus IgG, Venereal Disease Research Laboratory (VDRL) 1:16, positive Treponema Pallidum Hemagglutination Assay (TPHA), and non-reactive Hepatitis B, Hepatitis C, and Toxoplasma serology. Chest radiography suggested possible pleuritis, and sputum PCR for Mycobacterium tuberculosis was negative. The patient was diagnosed with Kaposi sarcoma, late latent syphilis, and stage IV HIV infection (WHO). He received benzathine penicillin G 2.4 million IU weekly for three doses. From the Internal Medicine Infectious Disease Division, he was started on cotrimoxazole prophylaxis and scheduled to initiate antiretroviral therapy (ART) with tenofovir, lamivudine, and dolutegravir. Screening for AIDS-related opportunistic infections and serial CD4 and viral load monitoring were planned.

After one month follow up, the patient reported no development of new lesions, the pre-existing lesions appeared darker and flatter, without a size in size. VDRL titer decreased to 1:2 one month after benzathine penicillin. The patient was continued on ART and scheduled for routine follow up and monitoring for treatment evaluation and skin lesions progression.

DISCUSSION

KS remains one of the most clinically important AIDS-defining malignancies, particularly in patients with advanced or previously undiagnosed HIV infection. Although KS is uncommon compared with many other malignancies, its epidemiological burden remains closely linked to HIV, with a substantial proportion of global KS cases being HIV-attributable.[3-4] This association is most evident in regions with high HIV and HHV-8 prevalence, particularly sub-Saharan Africa. However, HIV-associated KS continues to be reported in Asia and other non-endemic regions. In Indonesia and Southeast Asia, published data on KS remain limited compared with global reports, making individual case reports important for illustrating regional clinical presentations, diagnostic challenges, and opportunities for earlier recognition.

The present case is noteworthy because patch-type KS was the first visible clue to previously untreated HIV infection. Early KS lesions are often subtle, flat, and faintly violaceous, which makes them easy to misinterpret as purpura, dermatitis, vascular malformations, bacillary angiomatosis, or other benign vascular lesions.[1-2,9] This diagnostic challenge becomes more clinically relevant when HIV infection has not yet been diagnosed, as cutaneous lesions may be the only external sign of significant underlying immunosuppression. In this patient, recognition of suspicious violaceous lesions prompted HIV testing and revealed advanced disease, with a CD4 count of 192 cells/µL and WHO stage IV HIV infection. This finding supports the need to consider HIV-associated KS even when the skin lesions appear mild, asymptomatic, or nonspecific.

Beyond its diagnostic value, this case also reflects the continuing epidemiological overlap between HIV and STIs. Indonesia continues to face a concentrated HIV epidemic, particularly among key populations such as men who have sex with men, sex workers, transgender people, and people who inject drugs.[5-6] Syphilis remains clinically important because it shares overlapping routes of transmission with HIV and may facilitate HIV acquisition through mucosal ulceration, local inflammation, and disruption of epithelial barriers.[7-8] Conversely, HIV infection may modify the clinical course of syphilis and complicate the interpretation of serological response. The coexistence of HIV, HHV-8-associated KS, and late latent syphilis in this patient therefore highlights the importance of comprehensive STI screening when atypical mucocutaneous lesions are encountered, especially in patients with sexual risk factors.

Dermoscopy can provide useful supportive clues when KS is suspected, particularly in early lesions that are clinically subtle. In this case, dermoscopy demonstrated polychromatic discoloration forming a rainbow pattern, accompanied by serpentine, dotted, and coiled vessels, as well as white lines and clods. These findings are consistent with previously reported dermoscopic features of KS and may help differentiate KS from inflammatory or other vascular dermatoses.[10] However, dermoscopy should not be used as a stand-alone diagnostic tool. Its findings must be interpreted together with clinical morphology, distribution of lesions, risk factors, and histopathological examination.

Histopathological confirmation remains central to the diagnosis of early KS. Characteristic features include irregular and slit-like vascular spaces dissecting collagen bundles, spindle-cell proliferation, extravasated erythrocytes, hemosiderin deposition, lymphoplasmacytic infiltrate, and, in some cases, a promontory sign.[1,10] HHV-8 latent nuclear antigen immunohistochemistry is an important

confirmatory tool, particularly in early, subtle, or atypical lesions.[1-2] In this patient, biopsy of a macular lesion enabled diagnosis at the patch stage, before progression into more advanced plaque, nodular, or clinically evident visceral disease. This reinforces the role of early skin biopsy in persistent, multifocal, or unexplained violaceous lesions.

The management of HIV-associated KS depends on the extent of disease, immune status, symptoms, and evidence of visceral involvement. Effective antiretroviral therapy (ART) is the cornerstone of treatment and may lead to stabilisation or regression of early-stage KS by restoring immune function and reducing HHV-8-driven proliferation.[1-2] Local therapy may be considered for limited symptomatic lesions, whereas systemic chemotherapy is generally reserved for extensive, rapidly progressive, symptomatic, or visceral disease.[2] In this case, the patient was started on ART with tenofovir, lamivudine, and dolutegravir, together with cotrimoxazole prophylaxis. Late latent syphilis was treated with benzathine penicillin G 2.4 million IU weekly for three doses, followed by serological monitoring. After one month, the VDRL titre declined from 1:16 to 1:2, suggesting an early favourable serological response, while the absence of new KS lesions indicated early clinical stabilisation.

When compared with reports from highly endemic regions, where KS may present with extensive cutaneous disease, nodular lesions, lymphoedema, or visceral involvement, this case represents an earlier and more subtle cutaneous presentation.[1,3-4] In Southeast Asia and Indonesia, where KS is less frequently reported than in highly endemic regions and may not be the first diagnostic consideration, patch-stage lesions can easily be overlooked.[3-4,9] Therefore, this case adds clinical evidence from Indonesia that early KS may serve as an important entry point for diagnosing advanced HIV infection and concurrent STI coinfection. It also emphasises the value of close collaboration between dermatology, pathology, and infectious disease services in evaluating suspicious skin lesions.[1-2]

This report has several limitations. It describes a single patient, which limits generalisability. Long-term follow-up after ART initiation was not available, restricting assessment of KS regression, immune recovery, and long-term syphilis serological response. The extended evaluation for visceral KS was limited, although the patient had no obvious mucosal involvement and no new lesions during early follow-up. HHV-8 immunohistochemistry was not performed; therefore, the diagnosis was based on clinicopathological correlation supported by dermoscopic and histopathological findings. Despite these limitations, the case provides an important clinical message: persistent violaceous patch lesions, even when asymptomatic, should prompt early biopsy, HIV testing, and comprehensive STI screening, particularly in patients with sexual risk factors or features suggestive of immunosuppression.

CONCLUSION

Prompt biopsy and HIV testing enabled early diagnosis and timely initiation of antiretroviral therapy, which is essential for preventing KS progression. This report reinforces the importance of maintaining clinical vigilance for early KS presentations and contributes additional insight into the diagnostic challenges and epidemiologic overlap of HIV, HHV-8, and syphilis coinfection.

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DECLARATIONS

The authors thank the patient for his cooperation and consent to this case report.

CONFLICT OF INTEREST

The authors declared no conflicts of interest.

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