Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Infectious Diseases

Undetectable Virus, Visible Tumour: Kaposi Sarcoma in a Virologically Controlled HIV Patient

Danaoui Khadija[1*], El Wardi Amine[2]

[1]Department of Infectious Diseases, CHR Hôpital Moulay Hassan Ben Mehdi, Laayoune, Morocco
[2]Anatomopathologist, Private Practice, Laayoune, Morocco

DOI: https://doi.org/10.36347/sjmcr.2026.v14i06.061 | **Received:** 05.05.2026 | **Accepted:** 21.06.2026 | **Published:** 24.06.2026

***Corresponding author:** Danaoui Khadija
Department of Infectious Diseases, CHR Hôpital Moulay Hassan Ben Mehdi, Laayoune, Morocco

Abstract **Case Report**

Kaposi sarcoma (KS) is a human herpesvirus 8 (HHV-8)-driven angioproliferative tumour that remains one of the most frequent malignancies associated with human immunodeficiency virus (HIV) infection, even in the era of effective antiretroviral therapy (ART). We report the case of a 52-year-old man with a 7-year history of HIV infection on a tenofovir disoproxil fumarate/lamivudine/dolutegravir (TLD) regimen, with an undetectable plasma viral load but a persistently low CD4+ T-cell count of 250 cells/µL, who presented with violaceous, hyperkeratotic plaques on the dorsum of both feet. Skin biopsy with histopathological and immunohistochemical analysis (HHV-8 nuclear positivity, CD34 positivity of vascular structures) confirmed the diagnosis of Kaposi sarcoma. Staging computed tomography of the thorax, abdomen and pelvis revealed bilateral pulmonary nodular and micronodular lesions of lymphatic distribution, consistent with pulmonary involvement of KS. The patient was referred to oncology and started on systemic chemotherapy, with favourable clinical evolution. This observation illustrates that immune reconstitution under ART is not synonymous with immunocompetence, and that KS staging should systematically include thoracic imaging even in virologically controlled patients with discordant CD4+ recovery.

Keywords: Kaposi sarcoma; HIV; HHV-8; pulmonary involvement; immune discordance; antiretroviral therapy; CD34; chemotherapy.

1. INTRODUCTION

Kaposi sarcoma is a low-grade vascular neoplasm caused by human herpesvirus 8 (HHV-8, also known as Kaposi sarcoma-associated herpesvirus). It remains the most common HIV-associated malignancy worldwide and is classified as an AIDS-defining illness. Four clinico-epidemiological forms are classically described: classic, endemic (African), iatrogenic (transplant-related), and epidemic (HIV-associated). The epidemic form typically arises in the setting of profound cellular immunodeficiency, although cases occurring despite virologic suppression and partial immune recovery on antiretroviral therapy are increasingly recognised, reflecting a phenomenon of immune discordance between virological and immunological responses.

Cutaneous involvement is the most common presentation, manifesting as violaceous-to-brownish macules, plaques or nodules, frequently distributed on the lower limbs. Visceral dissemination, particularly to the lungs, gastrointestinal tract and lymph nodes, defines a more aggressive phenotype and carries a worse prognosis, often requiring systemic chemotherapy in addition to optimisation of antiretroviral therapy. We describe a case of cutaneous KS with pulmonary dissemination in a patient with long-standing, virologically suppressed HIV infection but incomplete CD4+ recovery, emphasising the diagnostic work-up and multidisciplinary management of this condition.

2. CASE PRESENTATION

2.1 Clinical History

A 52-year-old man, known to have HIV-1 infection for seven years and maintained on a fixed-dose combination antiretroviral regimen of tenofovir disoproxil fumarate, lamivudine and dolutegravir (TLD), presented to the outpatient clinic with skin lesions on the dorsum of both feet that had progressively enlarged over several months. The patient's most recent virological assessment showed an undetectable plasma HIV RNA viral load; however, his CD4+ T-cell count remained low at 250 cells/µL despite prolonged virologic control, reflecting incomplete immune reconstitution.

Physical examination revealed multiple confluents, violaceous-to-erythematous, hyperkeratotic plaques and papules on the dorsal aspect of both feet and extending onto the lower legs, with a verrucous, indurated surface and irregular borders (Figures 1 and 2). The lesions were non-tender, non-ulcerated, and there was no associated lymphoedema at presentation. No mucosal, oral, or visceral symptoms were reported at this stage. The clinical appearance was highly suggestive of cutaneous Kaposi sarcoma.

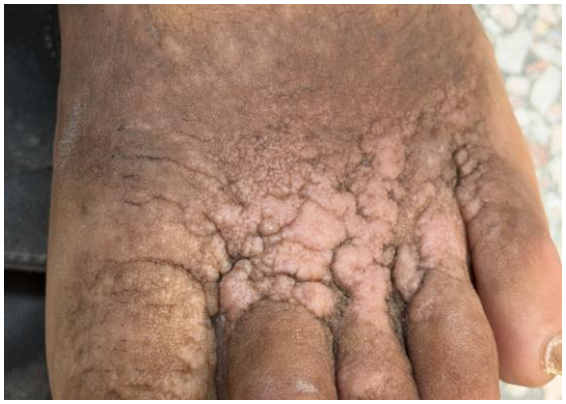

Figure 1: Violaceous, hyperkeratotic, confluent papules and plaques on the dorsum of the hand region, illustrating the typical purplish, verrucous surface of cutaneous Kaposi sarcoma lesions

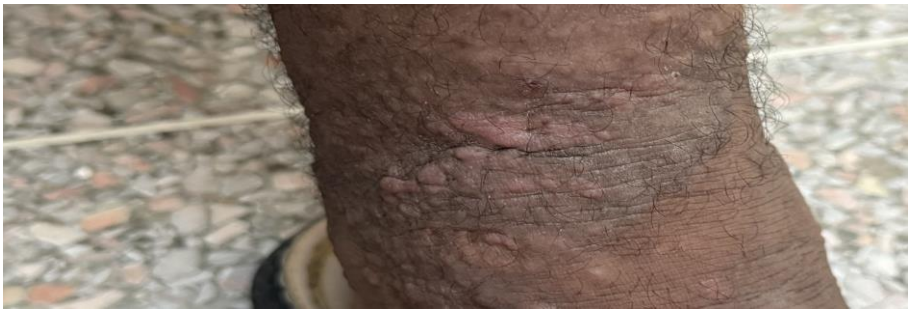

Figure 2: Erythemato-violaceous infiltrated plaque over the ankle/dorsal foot region, with a firm, slightly hyperkeratotic surface, corresponding to the site of initial presentation

2.2 Histopathological and Immunohistochemical Findings

An incisional skin biopsy was performed on one of the foot lesions. Histopathological examination on haematoxylin and eosin (H&E)-stained sections showed an acanthotic epidermis overlying a dermis infiltrated by a nodular and fascicular proliferation of spindle-shaped cells, forming irregular slit-like vascular spaces containing extravasated erythrocytes (Figures 3 and 4). Mild nuclear pleomorphism was observed, with scattered mitotic figures, but no significant cytological atypia or necrosis. These features were consistent with a vascular spindle-cell neoplasm of the Kaposi sarcoma type.

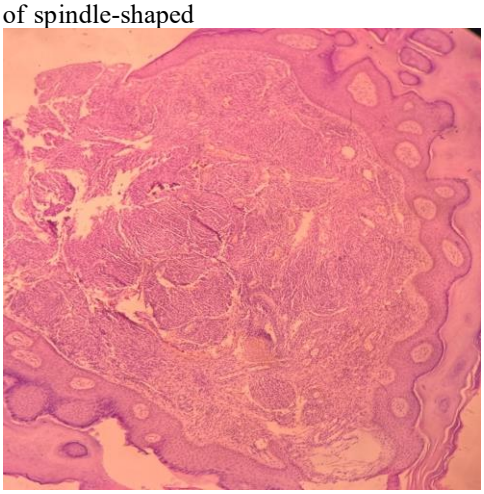

Figure 3: Low-power photomicrograph (H&E) showing a nodular dermal proliferation beneath an acanthotic, hyperkeratotic epidermis, with multiple confluent tumour nodules occupying the dermis

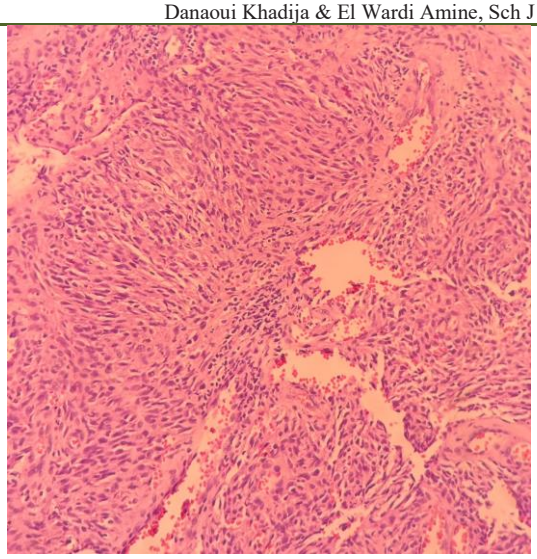

Figure 4: High-power photomicrograph (H&E) demonstrating fascicles of spindle-shaped cells with slit-like vascular spaces containing extravasated red blood cells, a hallmark feature of Kaposi sarcoma

Immunohistochemical analysis was performed to confirm the diagnosis. Tumour cells showed strong and diffuse nuclear positivity for HHV-8 latency-associated nuclear antigen (LANA-1), confirming viral pathogenesis, while the underlying vascular structures demonstrated strong positivity for CD34, an endothelial/vascular marker. This immunohistochemical profile was diagnostic of Kaposi sarcoma (Table 1).

Table 1: Summary of the immunohistochemical profile confirming the diagnosis of Kaposi sarcoma

Marker	Target	Result
HHV-8 (LANA-1)	Viral latent nuclear antigen	Strong, diffuse nuclear positivity
CD34	Endothelial / vascular marker	Positive in underlying vascular structures

2.3 Staging and Pulmonary Involvement

Given the histologically confirmed diagnosis of Kaposi sarcoma, staging contrast-enhanced computed tomography (CT) of the chest, abdomen and pelvis was performed to assess for visceral dissemination. Thoracic CT revealed a nodular thickening of the minor (horizontal) fissure with spiculated margins measuring 31 × 29 mm, an additional subpleural nodular thickening measuring 25 × 16 mm, and multiple bilateral pulmonary nodules and micronodules of predominantly lymphatic (peribronchovascular and subpleural) distribution, the largest measuring 15 × 11 mm on the right and 9 mm on the left (Figures 5 and 6). Bilateral apical emphysematous bullae and a few sub-centimetric latero-tracheal lymph nodes were also noted, without pleural or pericardial effusion.

This CT pattern, in the appropriate clinical and histopathological context, was considered highly suggestive of pulmonary involvement by Kaposi sarcoma, prompting referral to the oncology department for systemic staging completion and treatment.

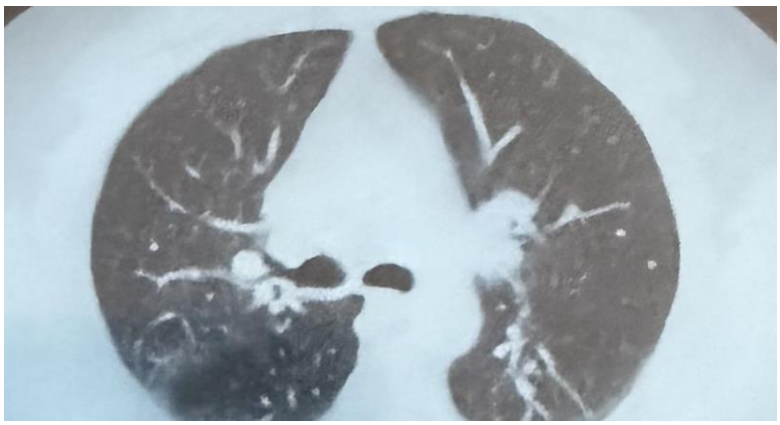

Figure 5: Axial chest CT (lung window) showing bilateral pulmonary micronodules with a predominantly peribronchovascular and subpleural distribution

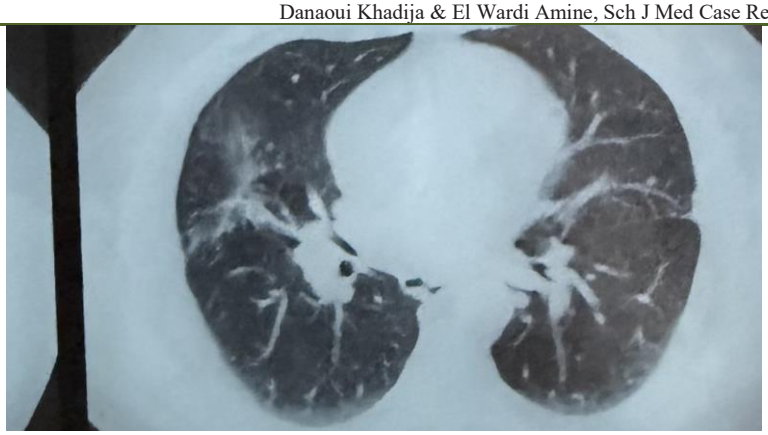

Figure 6: Axial chest CT (lung window) at a more caudal level demonstrating additional bilateral nodular and micronodular pulmonary opacities consistent with lymphatic spread of Kaposi sarcoma

2.4 Management and Outcome

Following confirmation of cutaneous Kaposi sarcoma with pulmonary dissemination, the patient was referred to the oncology department for multidisciplinary management. Antiretroviral therapy was continued unchanged, and systemic chemotherapy was initiated as the primary treatment for visceral (pulmonary) Kaposi sarcoma. Clinical follow-up showed a favourable evolution, with regression of the cutaneous lesions and clinical improvement, supporting continuation of the same therapeutic strategy with regular oncological and infectious disease follow-up.

3. DISCUSSION

Epidemic (HIV-associated) Kaposi sarcoma classically occurs in the setting of advanced immunosuppression, with most cases reported at CD4+ counts below 200 cells/µL and detectable viral replication. The present case is noteworthy because it occurred in a patient with durable virologic suppression on a modern integrase-inhibitor-based regimen (TLD), yet with a CD4+ count that remained at 250 cells/µL after seven years of treatment. This pattern, often termed 'immunological non-response' or 'discordant response' to ART, has been associated with an older age at treatment initiation, a low CD4+ nadir, persistent immune activation, and a higher residual risk of AIDS-defining and non-AIDS-defining malignancies, including Kaposi sarcoma, despite undetectable viraemia.

The diagnostic approach followed in this case adheres to recommended practice: a clinically suspicious cutaneous lesion was confirmed histologically, with immunohistochemical demonstration of HHV-8 LANA-1 nuclear positivity, which is considered highly sensitive and specific for Kaposi sarcoma and helps distinguish it from other vascular or spindle-cell mimics such as bacillary angiomatosis, pyogenic granuloma, angiosarcoma or dermatofibroma. CD34 positivity further supports the endothelial/vascular lineage of the spindle-cell proliferation.

Systemic staging is essential once cutaneous KS is confirmed, since visceral involvement—particularly pulmonary and gastrointestinal—significantly worsens prognosis and changes management from purely local or antiretroviral optimisation to systemic chemotherapy. Pulmonary KS may be asymptomatic, as in this patient, or present with cough, dyspnoea, haemoptysis or chest pain. Characteristic CT findings include bilateral nodules and micronodules with a peribronchovascular and subpleural (lymphatic) distribution, irregular or spiculated margins, septal thickening, and occasionally pleural effusion, closely mirroring the pattern observed in our patient. Bronchoscopy with visualisation of typical violaceous endobronchial lesions can further support the diagnosis, although biopsy is often avoided because of the risk of bleeding given the vascular nature of the lesions; in this case, the combination of confirmed cutaneous histology, the clinical context, and characteristic imaging was considered sufficient to support the diagnosis of pulmonary dissemination, in line with usual practice for AIDS-associated KS staging.

Management of HIV-associated KS rests on two complementary pillars: optimisation of antiretroviral therapy to maximise immune reconstitution, and, in cases of advanced, symptomatic, rapidly progressive or visceral disease, systemic chemotherapy (commonly pegylated liposomal doxorubicin or paclitaxel regimens, depending on local protocols and availability). In our patient, ART was continued without modification given persistent virologic suppression, and chemotherapy was added because of confirmed pulmonary involvement, leading to favourable clinical evolution. This case underscores the importance of considering KS, and systematically performing thoracic imaging, in any HIV-positive patient who develops suspicious skin lesions, irrespective of viral load control, particularly when CD4+ recovery remains incomplete.

4. CONCLUSION

This case illustrates that Kaposi sarcoma can develop in HIV-infected patients despite long-term virologic suppression, when CD4+ immune recovery

remains incomplete. A high index of clinical suspicion for cutaneous lesions, prompt histopathological and immunohistochemical confirmation, and systematic staging with thoracic imaging are essential steps in management. Multidisciplinary collaboration between infectious disease specialists and oncologists allowed timely initiation of systemic chemotherapy alongside continued antiretroviral therapy, resulting in favourable clinical evolution. Clinicians should remain vigilant for HIV-associated malignancies even in patients who appear virologically well controlled.

Declarations

Patient consent: Written informed consent was obtained from the patient for the publication of this case report and the accompanying clinical, radiological and histopathological images. All identifying information, including names of treating physicians and institutional identifiers, has been removed to preserve confidentiality.

Conflict of interest: The authors declare no conflict of interest related to this report.

REFERENCES

1. Cesarman E, Damania B, Krown SE, Martin J, Bower M, Whitby D. Kaposi sarcoma. Nat Rev Dis Primers. 2019; 5:9.
2. Schneider JW, Dittmer DP. Diagnosis and treatment of Kaposi sarcoma. Am J Clin Dermatol. 2017;18(4):529-539.
3. Bhutani M, Polizzotto MN, Uldrick TS, Yarchoan R. Kaposi sarcoma-associated herpesvirus-associated malignancies: epidemiology, pathogenesis, and advances in treatment. Semin Oncol. 2015;42(2):223-246.
4. Gbabe OF, Okwundu CI, Dedicoat M, Freeman EE. Treatment of severe or progressive Kaposi's sarcoma in HIV-infected adults. Cochrane Database Syst Rev. 2014;(9):CD003256.
5. Restrepo CS, Martinez S, Lemos JA, *et al.,* Imaging manifestations of Kaposi sarcoma. Radiographics. 2006 ;26(4):1169-1185.
6. Yarchoan R, Uldrick TS. HIV-associated cancers and related diseases. N Engl J Med. 2018 ;378(11):1029-1041.