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# Kaposi Sarcoma Associated with Human Immunodeficiency Syndrome: A Case Report

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**Abstract:** Kaposi Sarcoma is a neoplasm described in four distinct types, with the epidemic form being the most prevalent among individuals living with HIV. This form is characterized by a more aggressive disease presentation, affecting mucous membranes and visceral organs. We report the case of a 42-year-old male patient who sought medical care due to cutaneous lesions, initially diagnosed as psoriasis. Subsequently, the lesions worsened, and a biopsy was performed, confirming the diagnosis of Kaposi Sarcoma. Additional examinations later revealed HIV and CMV infections, leading to the initiation of antiretroviral therapy (ART). Shortly thereafter, the patient was hospitalized with febrile consumptive syndrome and remained hospitalized for 32 days for symptom control and remission. It is important to recognize the clinical presentation of patients in order to establish an earlier diagnosis of Kaposi Sarcoma, thereby enabling the prompt initiation of specific treatment for the condition.

**Keywords:** Kaposi Sarcoma; HIV; HHV-8; Case Report.

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## 1. Introduction

Kaposi sarcoma (KS) is a heterogeneous angioproliferative tumor that, in most cases, arises in the skin. Four types of the disease are described in the literature: classic, endemic, iatrogenic, epidemic, and non-epidemic [1-4]. Regarding epidemiology, compared with the general population, KS rates are 500 times higher among people living with Human Immunodeficiency Virus (HIV), and in this population it is known as epidemic KS [2,3].

The etiology of the disease remains unknown; however, it is known to have a strong association with human herpesvirus 8 (HHV-8), which persists latently in the body. It was identified in 1994 in lesions from patients with Acquired Immunodeficiency Syndrome (AIDS), confirming the hypothesis that an oncogenic virus was the etiological agent of KS. In patients living with HIV (PLHIV), continuous exposure to HHV-8 associated with immunosuppression favors viral activation and proliferation, contributing to the development of KS [1,5]. In other words, tumor development depends on factors such as host immune function and inflammatory mechanisms; therefore, in most cases, HHV-8 infection alone does not lead to the onset of KS [3].

In its typical presentation, KS is characterized by the proliferation of spindle-shaped endothelial cells, mainly affecting the skin. In its HIV-related form, it is aggressive and may particularly involve mucous membranes and visceral organs [2]. Factors such as advanced age, prolonged HIV infection, chronic infections, and conditions related to chronic inflammation play an important role in the pathogenesis of the disease [2,3]. KS was offi-

cially recognized as an AIDS-defining illness by the Centers for Disease Control and Prevention (CDC) in 1982. In the revised 1993 AIDS classification, KS was listed as one of the three AIDS-defining cancers, together with non-Hodgkin lymphoma and invasive cervical cancer. With the introduction of combined antiretroviral therapy (cART) in 1996 and its broad access in countries with greater resource availability, the risk of developing KS among people living with HIV has significantly decreased over the years [1,5].

The following is a report of a case of Kaposi sarcoma associated with Acquired Human Immunodeficiency Syndrome in a patient living with HIV, highlighting its clinical, diagnostic, and therapeutic aspects.

## 2. Case Report

A 42-year-old male patient, phototype II, sought medical care due to cutaneous lesions, initially receiving a diagnostic hypothesis of psoriasis, which did not improve with topical corticosteroid treatment. Two months later, with worsening of the lesions, a biopsy was performed at a specialized service, confirming the diagnosis of Kaposi sarcoma. Additional complementary examinations were then performed, revealing HIV infection, with a viral load (VL) of 2,160,000 and CD4+ T-cell count of 120, associated with cytomegalovirus (CMV) infection. Shortly thereafter, antiretroviral therapy (ART) was initiated.

At the beginning of treatment, the patient was hospitalized with febrile consumptive syndrome, presenting retroperitoneal adenomegaly that regressed after the use of Ganciclovir, as well as cutaneous lesions suggestive of fungal disease, which were treated with itraconazole. He remained hospitalized for several days and was discharged with partial improvement. Two days later, he required readmission due to generalized edema, predominantly affecting the right hemibody (Figure 1), associated with diffuse ecchymoses on the trunk, genitalia, face, and limbs (Figures 2A–2C), requiring a total of 32 days of hospitalization.

**Figure 1.** Edema in the right lower limb.

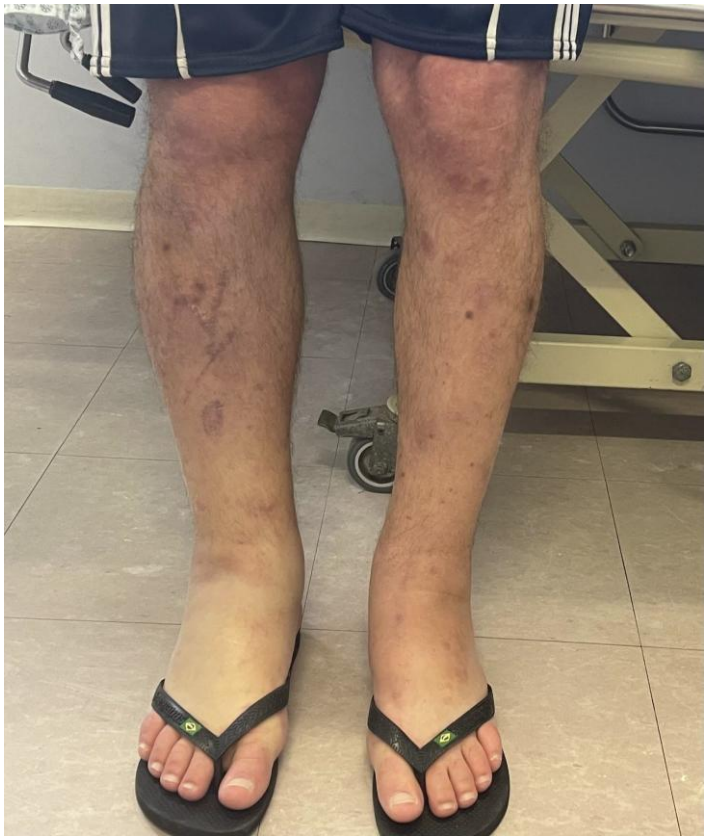
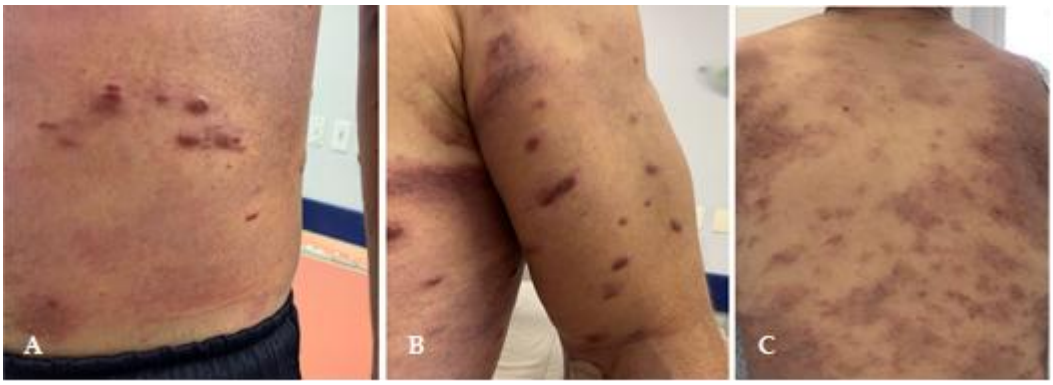


**Figure 2.** Diffuse ecchymoses in the abdominal region (A), left upper limb (B), and trunk (C).




Complementary examinations were performed. Laboratory findings revealed microcytic and hypochromic anemia, characterized by reduced hemoglobin levels (10.5 g/dL), hematocrit (18.3%), and erythrocyte count (3.19 million/mm³), in addition to decreased mean corpuscular volume (57.4 fL) and increased red cell distribution width (RDW: 18.3%), with anisocytosis and mild poikilocytosis with macrocytes on morphological examination. Mild leukopenia (4,050/mm³) and mild thrombocytopenia (130,000/mm³) were also observed. The patient presented hyponatremia (Na: 130.8 mEq/L), blood glucose of 130 mg/dL, total protein of 4.6 g/dL, and albumin of 3.0 g/dL. These laboratory findings were compatible with microcytic and hypochromic anemia associated with metabolic and electrolyte disturbances.

Chest computed tomography showed a small bilateral pleural effusion, more pronounced on the right side, right basal atelectasis, and a peripheral nodular opacity in the left lower lobe measuring 1.3 cm, probably inflammatory in nature. The trachea and main bronchi had a usual configuration. Multiple small mediastinal and axillary lymph nodes were observed, with one enlarged node in the left axilla (2.8 × 2.0 cm). The thoracic aorta had normal caliber with parietal atheromatous calcifications. Cardiac volume was preserved. Transthoracic echocardiography demonstrated preserved cardiac chamber dimensions; preserved global and segmental systolic ventricular performance; preserved left ventricular diastolic function; and mitral valve prolapse.

Lymphoscintigraphy of the lower limbs demonstrated dermal reflux of small extension located in the middle third of the right leg, characterizing collateral circulation to the dermis (an alternative lymphatic drainage pathway), possibly related to local/regional lymphatic dysfunction. Additional complementary examinations for tuberculosis were performed, all with negative results.

The patient continued treatment with ART: TDF + 3TC + DTV (tenofovir 150 mg + lamivudine 300 mg + dolutegravir 50 mg), primary prophylaxis with Bactrim, and interdisciplinary follow-up in oncology, hematology, and infectious diseases. Chemotherapy with Paclitaxel 180 mg was also initiated, administered in cycles every 14 days, totaling seven cycles. After completion of treatment, the patient showed complete improvement of the cutaneous-mucosal lesions and generalized edema previously described, demonstrating an excellent clinical response. He remains on ART and under oncological follow-up, with undetectable viral load and a CD4+ T-cell count of 280.

## 3. Discussion

HHV-8 affects endothelial and/or mesenchymal cells, which then acquire endothelial differentiation. It usually affects the integumentary system, but may also involve other organs, such as lymph nodes, the respiratory tract, gastrointestinal (GI) tract, and, in some cases, bone tissue. Early lesions may appear as a granulation-like reaction with immune cell infiltration, intense angiogenesis, and proliferation of spindle-shaped cells, which are

the tumor cells of KS [2,3]. Cutaneous lesions are generally characterized by macules, nodules, and purplish, bluish-red, or hyperchromic plaques that may become verrucous and hyperkeratotic. Lesions may be painful and/or pruritic, and patients commonly present with limb edema. Some lesions may also develop fungal infection and become secondarily infected [2,4].

It is more prevalent among individuals with uncontrolled HIV infection, especially those with low CD4+ cell counts. Severe immunosuppression, marked by reduced CD4+ counts, considerably increases the risk of developing KS, with the average CD4+ count at diagnosis being approximately 175 cells/mm$^3$. It occurs more frequently in patients with detectable viral loads, even among those receiving ART [2,3]. The clinical presentation raises suspicion for KS; however, histopathological confirmation remains the gold standard for diagnosis, although it is usually more difficult in the early stages [6].

Spindle cells are present in all forms of disease. They represent a unifying characteristic, form the basis of diagnosis, and constitute most of the proliferative cell fraction, as determined by Ki-67 staining or other molecular proliferation markers. However, the broad morphological variability of KS may mimic numerous unrelated neoplastic and non-neoplastic conditions, making it a challenging diagnosis [7]. Kaposi sarcoma with pulmonary involvement may be identified through chest radiography or computed tomography. In patients with the disease, diagnosis is confirmed by bronchoscopy, allowing visualization of typical lesions and exclusion of other infectious etiologies. When the disease affects the gastrointestinal tract, it is usually initially detected through evaluation of occult blood loss and/or microcytic anemia. Examinations such as upper gastrointestinal endoscopy and colonoscopy should be indicated for patients presenting symptoms or blood loss in the stool [8].

The clinical management of Kaposi sarcoma takes into consideration several factors, such as comorbidity, patient preferences, extent of disease, and immunological and virological status. Although there is no cure, long-term remission is possible, especially when treatment of the underlying immunosuppression is performed in patients living with HIV, which represents the first-line therapy [2]. In PLHIV receiving optimized ART, when surveillance and local treatment options for Kaposi sarcoma have been exhausted, treatment progresses to chemotherapy.

The most common options include anthracyclines and taxanes, which are effective in disease control, with doxorubicin being the main drug used. Chemotherapy is indicated to control tumor progression, especially in cases with visceral involvement or disease unresponsive to local therapies. More recently, some patients on ART have benefited from immunotherapy [1]. The duration of chemotherapy is determined by treatment response; if there is a small number of lesions, six cycles or fewer may be sufficient. For patients with extensive lesions or visceral disease involvement, the number of cycles depends on clinical response and treatment may be prolonged. Patients may require intermittent therapy for years [9].

## 4. Conclusion

In this context, we report a case of Kaposi sarcoma associated with Human Immunodeficiency Syndrome. As previously mentioned, this condition is currently rare, and diagnosis is often delayed due to multiple previously discussed factors. The clinical presentation observed in this patient resembles those described in the literature. This emphasizes the importance of recognizing clinical characteristics to facilitate earlier diagnosis and, consequently, earlier treatment.

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**Conflicts of Interest:** All other authors declare no conflicts of interest.

## References

1. Rasmussen SA, Mazurek MF, Rosebush PI. Catatonia: Our current understanding of its diagnosis, treatment and pathophysiology. World J Psychiatry. 2016 Dec 22;6(4):391-398. doi: 10.5498/wjp.v6.i4.391.
2. Palich R, Makinson A, Veyri M, Guihot A, Valantin MA, Brégigeon-Ronot S, Poizot-Martin I, Solas C, Grabar S, Martin-Blondel G, Spano JP. Kaposi's Sarcoma in Virally Suppressed People Living with HIV: An Emerging Condition. Cancers (Basel). 2021 Nov 15;13(22):5702. doi: 10.3390/cancers13225702. PMID: 34830857; PMCID: PMC8616070. https://pmc.ncbi.nlm.nih.gov/articles/PMC8616070/.
3. Karabajakian A, Ray-Coquard I, Blay JY. Molecular Mechanisms of Kaposi Sarcoma Development. Cancers (Basel). 2022 Apr 7;14(8):1869. doi: 10.3390/cancers14081869. PMID: 35454776; PMCID: PMC9030761. https://pmc.ncbi.nlm.nih.gov/articles/PMC9030761/.
4. Mangusan RF, Ekwede I, Widell A. CE: HIV-Associated Kaposi Sarcoma in the Combination Antiretroviral Therapy Era. Am J Nurs. 2022 Dec 1;122(12):32-40. doi: 10.1097/01.NAJ.0000901848.07128.92. PMID: 36321823; PMCID: PMC9671845. https://pmc.ncbi.nlm.nih.gov/articles/PMC9671845/.
5. Dittmer DP, Damania B. Kaposi's Sarcoma-Associated Herpesvirus (KSHV)-Associated Disease in the AIDS Patient: An Update. Cancer Treatment and Research. 2019;177:63–80. doi:10.1007/978-3-030-03502-0_3. https://pubmed.ncbi.nlm.nih.gov/30523621/
6. Grabar S, Costagliola D. Epidemiology of Kaposi's Sarcoma. Cancers (Basel). 2021 Nov 14;13(22):5692. doi: 10.3390/cancers13225692. PMID: 34830846; PMCID: PMC8616388. https://pmc.ncbi.nlm.nih.gov/articles/PMC8616388/.
7. Conselho Nacional de Saúde (BR). Resolução n° 466, de 12 de dezembro de 2012. Aprova as diretrizes e normas regulamentadoras de pesquisas envolvendo seres humanos. Diário Oficial da União, Brasília, DF. 2013 jun 13; Seção I:59.
8. Schneider JW, Dittmer DP. Diagnosis and Treatment of Kaposi Sarcoma. American Journal of Clinical Dermatology. 2017;18(4):529-539. doi:10.1007/s40257-017-0270-4. https://pmc.ncbi.nlm.nih.gov/articles/PMC5509489/.
9. Gonçalves PH, Uldrick TS, Yarchoan R. HIV-associated Kaposi sarcoma and related diseases. AIDS (London, England). 2017;31(14):1903–1916. doi:10.1097/QAD.0000000000001567. https://pubmed.ncbi.nlm.nih.gov/28609402/.
10. Ramaswami R et al. Oncologic Treatment of HIV-Associated Kaposi Sarcoma 40 Years on. Journal of Clinical Oncology. 2022;40(3):294-306. doi:10.1200/JCO.21.02040. https://pmc.ncbi.nlm.nih.gov/articles/PMC8769148/.