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Aids-Related Disseminated Kaposi Sarcoma with Renal Involvement: A Rare Non-Transplant Case Report

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ABSTRACT

Kaposi's sarcoma (KS) is a vascular malignancy associated with human herpesvirus 8 (HHV-8). In Africa, the commonest type is the AIDS-associated KS seen in immunocompromised individuals, infected with HIV. Although KS primarily affects the skin and mucosal surfaces, visceral involvement is also well-documented, with the lungs and gastrointestinal tract being the most frequently affected sites. Renal involvement in non-transplant patients remains exceptionally rare. We present a case of a 33-year-old HIV-positive woman with a history of non-adherence to highly active antiretroviral therapy (HAART), who developed disseminated KS involving the oral mucosa, lungs, gastrointestinal tract, liver, and the right kidney. She presented with respiratory distress, generalized lymphadenopathy, and a hyperpigmented sublingual mass. Histopathological examination of the oral lesion confirmed KS, and post-mortem findings revealed extensive tumor infiltration of multiple organs, including the right kidney. This case highlights the aggressive nature of disseminated KS in severely immunocompromised patients and underscores the critical importance of HAART adherence in preventing disease progression. Given the exceptional rarity of non-allograft renal KS, this report adds to the limited literature on its occurrence and emphasizes the need for heightened clinical awareness when evaluating advanced KS cases.

Keywords

Kaposi's sarcoma, HIV/AIDS, Non-allograft renal Kaposi sarcoma, HHV-8 disseminated Kaposi sarcoma, Human herpesvirus 8, HAART non-adherence, HIV-related malignancy.

Introduction

Kaposi's sarcoma (KS) is a low-grade, multicentric vascular tumor associated with human herpesvirus 8 (HHV-8), also known as Kaposi sarcoma-associated herpesvirus (KSHV) [1]. Owing to the high incidence of HHV-8 in sub-Saharan Africa (up to 50%), KS is one of the most common cutaneous malignancies, particularly among people living with HIV [2]. With the advent of highly active antiretroviral therapy (HAART), the incidence of KS has significantly decreased in HIV patients who adhere to treatment and have no other immunocompromising conditions [3]. Nonetheless, KS remains an AIDS-defining illness, typically

emerging when CD4 counts are critically low [4].

KS typically presents as painless, hyperpigmented or purplish mucocutaneous lesions. Visceral involvement is possible and when it occurs, it commonly involves the gastrointestinal tract, lungs, lymph nodes, and liver. Non-allograft renal involvement is exceedingly rare [5]. To our knowledge, renal Kaposi sarcoma in an HIV-positive patient without a history of kidney transplantation has not been previously documented. Here, we present a rare case of HIV-associated disseminated KS with renal involvement in a patient with severe immunosuppression and a critically low CD4 count.

Case Report

A 33-year-old gravida 10, para 6 + 3 at 13 weeks and 6 days of amenorrhea, known to be HIV-positive and on HAART for 11 years, who had defaulted treatment for three months prior to presenting to our facility complained of a purplish sublingual mass, cough, generalized chest pain, and worsening difficulty in breathing. She reported intermittent fevers but denied drenching night sweats or significant weight loss. She reported no history of edema, no hematuria, or any other urinary symptoms. She reported no skin lesions. On examination, she was afebrile, she had mild jaundice and enlarged cervical, left pre-auricular, and bilateral post-auricular lymph nodes. She was in respiratory distress (respiratory rate: 36 breaths per minute), with an initial peripheral oxygen saturation of 97% on room air. Her blood pressure was 108/64 mmHg, and she was tachycardic (pulse rate: 133 bpm). Her most recent CD4+ cell count (one month prior to admission) was 47 cells/µL. Her laboratory Investigations were significant for moderate anemia (Hb of 8.2g/dL), mild thrombocytopenia (109 x10^3/µL), and elevated serum GGT (88U/L). Serum urea and creatinine were within normal ranges (Table 1).

Table 1: Laboratory findings upon admission.

Parameter	Result	Normal range
WBC	8.39*10^3/µL	3.5-9.50*10^3/µL
Hb	8.2g/dL	11.5-25 g/dL
PLT	109*10^3/µL	156-342*10^3/µL
Serum total bilirubin	18 umol/L	Up to 22.0umol/L
Serum GGT	88U/L	Up to 40U/L
Serum protein Total	50g/L	66-87g/L
Serum Albumin	27g/L	38-47g/L
Serum ALT	16U/L	Up to 35U/L
Serum SGOT	26U/L	Up to 32U/L
Serum Alk. Phosphatase	175U/L	65-270U/L
Urea	3.9 mmol/L	2.7-6.4mmol/L
Serum creatinine	45 mmol/L	44-106mmol/L
Serum Chloride	110 mmol/L	90-110mmol/L
Serum sodium(Na)	130 mmol/L	138-150mmol/L
Serum potassium(K)	3.8mm0l/L	3.6-5.3mmol/L
Serum phosphorus	1.0mmol/L	0.9-1.5mmol/L
Serum Calcium	1.8mmol/L	2.1-2.5mmol/L

Histology examination of the oral lesion showed proliferating spindle cells forming blood-filled slit-like spaces and vascular channels, with marked red cell extravasation and hemorrhage; features consistent with Kaposi sarcoma. The overlying squamous epithelium was hyperplastic (reactive). (Figure 1). The patient was managed with supportive treatment, including oxygen therapy and intravenous fluids, and was being optimized for possible chemotherapy initiation. Unfortunately, the disease progression was rapid, with worsening difficulty in breathing and our patient died four days after admission due to respiratory failure and extensive lung involvement by the tumour.

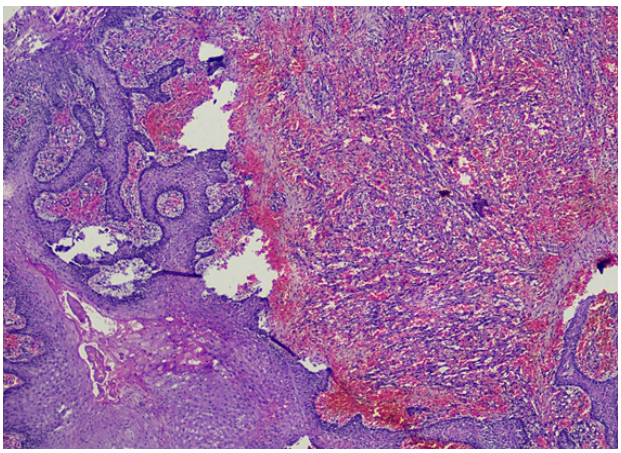

Figure 1: Oral mucosal lesion biopsy. Sections show tumour composed of proliferating spindle cells with blood filled slit-like spaces (H&E X00).

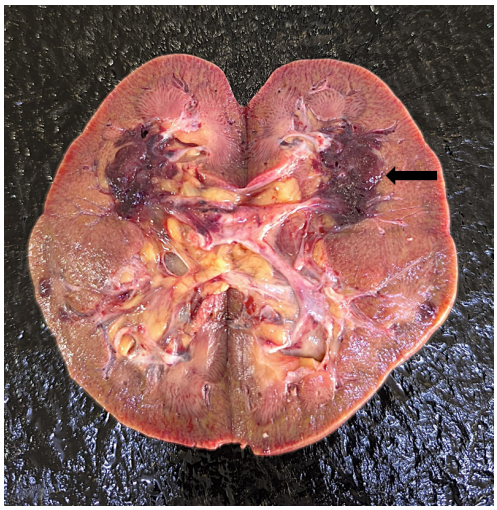

Figure 2: Gross image of the right kidney showing infiltration of the renal parenchyma by tumour.

At autopsy she had central and peripheral cyanosis, severe mucosal pallor, and peripheral lymph nodes were enlarged. There was 700 mL of bilateral hemorrhagic pleural effusion. Both lungs were diffusely infiltrated by a solid, dark red tumor. The esophagus and intestinal mucosa were also involved. The liver, right kidney, para-pancreatic lymph nodes, and multiple lymph node chains (mesenteric, hilar, para-tracheal, and para-esophageal) showed extensive tumor infiltration. The right kidney medulla was infiltrated by a similar dark red solid tumor (as described

above) with irregular borders. (Figure 2). Histology of the lungs, liver, kidney and lymph nodes showed diffuse infiltration of the parenchyma by spindled cells forming slit-like and sieve-like spaces filled with blood (similar to the oral mass histology). There was extravasation of the red blood cells and hemorrhage. Scattered haemosiderophages were seen. (Figure 3, Figure 4, and Figure 5). Immunohistochemical staining for HHV-8 and CD31 on the renal tumor showed strong and diffuse positivity, confirming the diagnosis of disseminated Kaposi sarcoma with renal involvement. (Figure 6 and Figure 7).

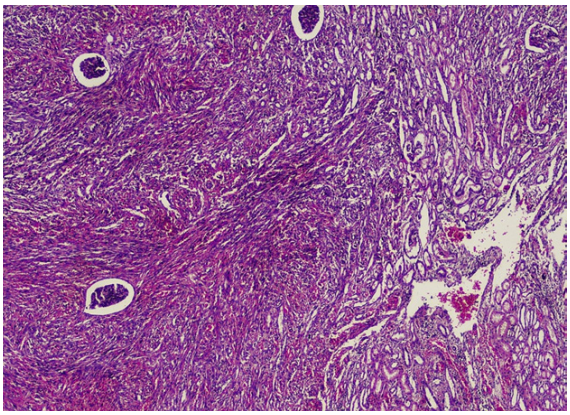

Figure 3: Tumour infiltrating the renal parenchyma (H&E X100).

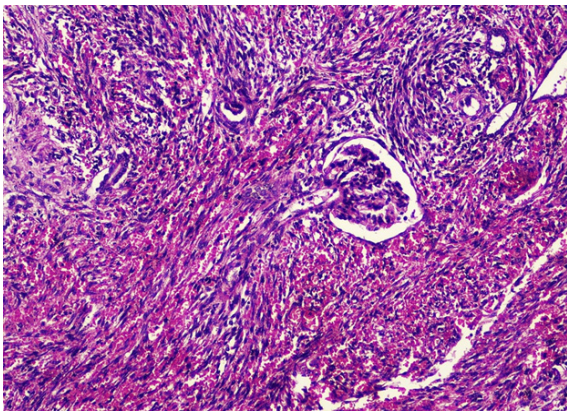

Figure 4: Tumour infiltrating the renal parenchyma (H&E X200).

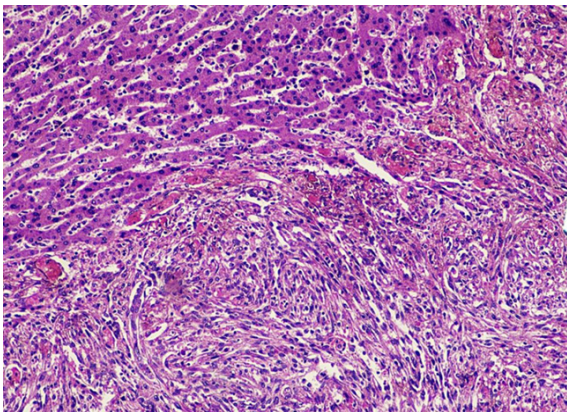

Figure 5: Tumour cells infiltrating the liver parenchyma (H&E X200).

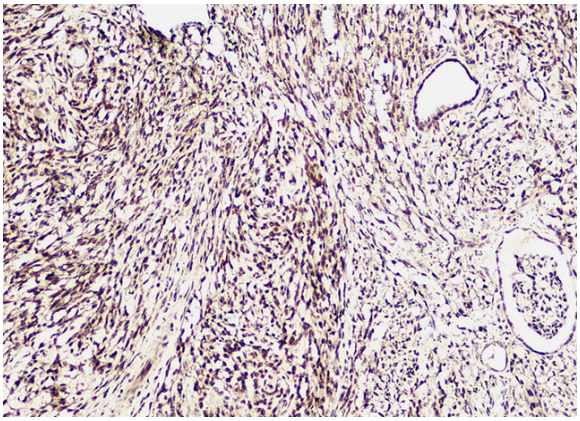

Figure 6: Tumour cells diffusely positive for HHV8 (immunohistochemistry x200).

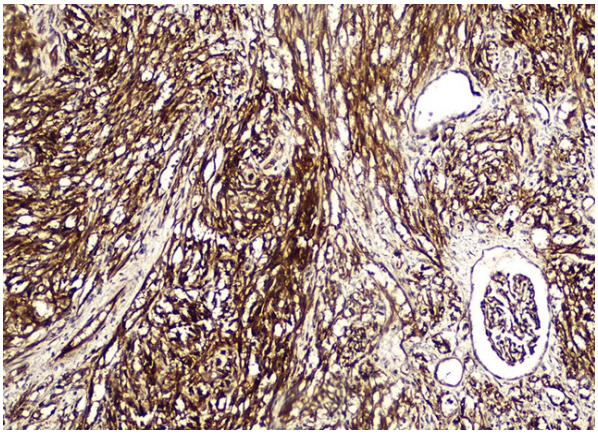

Figure 7: Tumour cells are diffusely positive for CD31 (immunohistochemistry x200).

Discussion

Disseminated Kaposi sarcoma (KS) is a well-documented opportunistic malignancy in HIV/AIDS patients, but non-allograft renal involvement remains a rare finding. In this case, the patient presented with advanced disease, which is consistent with previous studies indicating that KS frequently occurs in patients with profound immunosuppression [5,6]. KS has a high affinity for vascular and lymphatic endothelial cells, which may explain its rapid dissemination in patients with severe immunosuppression [7,8].

Our findings align with research by Semeere et al. [6], who demonstrated that HAART significantly reduces KS progression; however, its efficacy diminishes once KS advances to visceral involvement. Similar to other reports, our case illustrates how non-adherence to HAART accelerates KS progression, ultimately leading to multi-organ failure [9]. Unlike the more common presentations of KS affecting the skin, lungs, and gastrointestinal tract, our case additionally documents renal involvement, a seldom reported phenomenon. Additionally, our patient had no skin involvement.

Story et al. [5] described KS affecting renal allografts in kidney transplant recipients, which were attributed to viral reactivation

and the seroconversion of HHV-8 seronegative transplant recipients following transplant from a seropositive donor. However, our patient had no history of transplantation, making this case unique. This suggests a potential underdiagnosis of renal KS in non-transplant patients with advanced AIDS, especially considering the high seropositivity of HHV-8 (up to 50%) in sub-Saharan Africa. Renal involvement may present with haematuria, proteinuria, or renal dysfunction; however, in this case, renal function remained relatively preserved before death, possibly due to the rapid progression of pulmonary disease leading to early mortality [11]. Furthermore, studies have indicated that delayed diagnosis of visceral KS is associated with poor prognosis [7].

The aggressive nature of KS in this patient posed significant management challenges. While HAART is the primary treatment for AIDS-related KS, chemotherapy (e.g., liposomal doxorubicin, paclitaxel) is indicated in advanced cases with visceral involvement. Unfortunately, this patient succumbed to respiratory failure before chemotherapy initiation. Late presentation, severe immunosuppression, and widespread organ involvement limited therapeutic options. Research suggests that initiating HAART alone can lead to regression of KS lesions, particularly in patients with higher CD4 counts and lower HIV viral loads [9]. However, in patients with extensive visceral involvement, combination chemotherapy remains essential. Our case adds to the literature emphasizing the need for clinicians to maintain a high index of suspicion for renal involvement in advanced KS cases. Emerging evidence suggests that high HHV-8 viral loads correlate with more aggressive KS phenotypes and increased visceral dissemination [10]. This further emphasizes the importance of early HAART initiation and regular monitoring to prevent disease progression.

Conclusion

This case underscores the importance of strict HAART adherence in preventing KS progression. It also highlights a rare instance of renal involvement in disseminated KS, suggesting that renal KS may be under-recognized in non-transplant settings and in areas with high HHV-8 endemicity. Early diagnosis, re-initiation of HAART, and prompt chemotherapy initiation remain key strategies for improving outcomes in advanced KS.

Ethical Considerations

In accordance with local and national regulations, this case report was exempt from formal ethical approval.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest related to this study.

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Data Availability Statement

All data generated or analyzed during this study are included in this article. Additional information is available from the corresponding author upon reasonable request.

Author Contributions

Omega Phillip- conceptualization, data curation, writing – original draft, writing – review and editing. Musoke Sharrif- initial pathological diagnosis. Alele David- reviewed postmortem report, Tonny Okecha-review & editing, Mawanda Anatoli-review & editing, Boaz Mwesigwa- literature view & wrote the final manuscript. Gladys Adokorach-review and editing, ,Lukande Robert-review and editing , Kalungi Sam- project administration, supervision, review and editing.

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