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Case Report
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Gastrointestinal Kaposi Sarcoma and Multicentric Castleman Disease in an HIV-Positive Patient Following Liver Transplantation

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Abstract

Background: Kaposi Sarcoma (KS) is the most frequent neoplasm associated with Human Herpesvirus 8 (HHV-8) in immunosuppressed individuals, with substantially higher incidence in solid organ transplant recipients. The coexistence of KS and Multicentric Castleman Disease (MCD) is uncommon and associated with greater clinical complexity.

Case Presentation: We report a 21-year-old male liver transplant recipient with HIV co-infection who presented with prolonged fever (80 days), watery diarrhea, and 20 kg weight loss. Post-transplant complications included biliary and hepatic artery stenosis, late graft rejection, and poor adherence to immunosuppressive therapy. HIV was diagnosed in January 2025, with antiretroviral therapy interrupted due to gastrointestinal intolerance. On admission, CD4+ count was 235 cells/mm3. Imaging revealed diffuse lymphadenopathy, splenomegaly, and rectal wall thickening. Histopathological and immunohistochemical evaluation confirmed gastrointestinal KS and HHV-8-associated MCD. Treatment with doxorubicin and rituximab resulted in clinical improvement and symptom resolution.

Conclusion: This case illustrates the diagnostic complexity of HHV-8-related diseases in patients with combined immunosuppression and highlights the importance of early recognition of visceral KS, even in the absence of cutaneous lesions.

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Introduction

Kaposi Sarcoma (KS) is the most frequent neoplasm associated with Human Herpesvirus 8 (HHV-8) in immunosuppressed individuals, [1,2] with a substantially higher incidence in transplant recipients [2,3]. Its pathogenesis results from the interaction between HHV-8 infection and immunosuppression, favoring viral replication and oncogenesis.

The spectrum of HHV-8-related diseases includes KS, Multicentric Castleman Disease (MCD), primary effusion lymphoma, and KSHV-Associated Inflammatory Syndrome (KICS) [1,2]. In the post-transplant setting, KS tends to exhibit more aggressive behavior, driven by viral lytic replication and cytokine production, especially the Viral Homolog of Interleukin-6 (vIL-6), promoting angiogenesis and cell proliferation [4,5]. HHV-8-associated MCD, although less prevalent, is potentially severe and characterized by exuberant systemic inflammation with fever, lymphadenopathy, hepatosplenomegaly, and cytopenia's [4,6]. The coexistence of KS and MCD is uncommon and associated with greater clinical complexity and worse prognosis [3,6].

The risk of these manifestations is strongly modulated by the intensity of immunosuppression, aggravated by HIV co-infection and antiretroviral therapy failure, with consequent CD4+ lymphocyte depletion and increased viral replication [5,9].

We report the case of a young liver transplant recipient with HIV co-infection who developed gastrointestinal KS and HHV-8-associated MCD. This case illustrates the impact of combined immunosuppression on the clinical expression of the KSHV-related spectrum and highlights the

need to consider these entities in immunocompromised patients with prolonged fever, lymphadenopathy, and visceral involvement without a defined etiology.

Case Presentation

A 21-year-old male, who had undergone liver transplantation in 2021 for autoimmune cirrhosis, was admitted on March 16, 2026, with prolonged fever (80 days), watery non-bloody diarrhea, nausea, vomiting, and 20 kg weight loss, associated with asthenia and hyporexia.

Post-transplant course was complicated by biliary and hepatic artery stenosis, requiring reintervention. In 2024, he presented late graft rejection associated with poor adherence to the immunosuppressive regimen, and was treated with corticosteroid pulse therapy. In January 2025, HIV infection was diagnosed, and antiretroviral therapy (tenofovir/lamivudine/dolutegravir) was initiated but subsequently interrupted due to gastrointestinal intolerance. On admission, the CD4+ count was 235 cells/mm^3 and the HIV viral load was 672 copies/mL.

In the preceding months, he had been hospitalized twice for persistent diarrhea. Empirical treatment with ceftriaxone and metronidazole was administered during the first hospitalization. During the second hospitalization, Clostridioides difficile infection was diagnosed and treated with oral vancomycin, with residual symptoms persisting.

On admission, he was hypotensive (blood pressure 80 mmHg × 50 mmHg), tachycardic (110 bpm), febrile (38.8°C), dehydrated, and pale. Hepatosplenomegaly was present (liver palpable 2 cm below the right costal margin; spleen 6 cm below the left costal margin), along with generalized small-volume lymphadenopathy (Table 1).

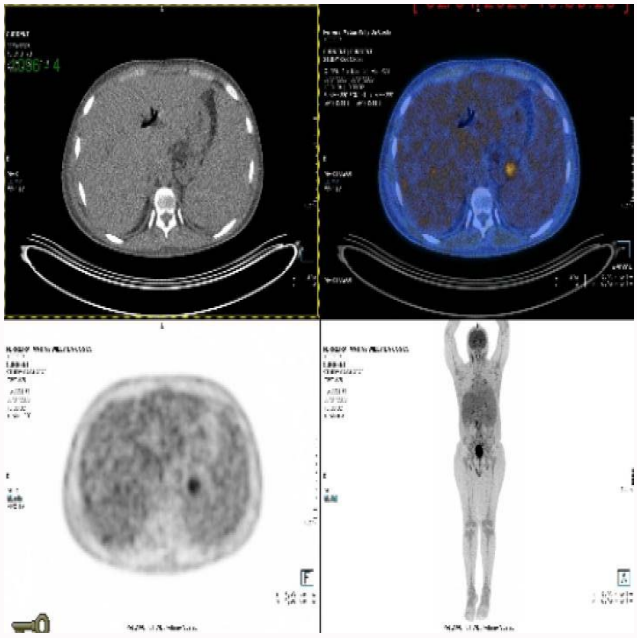

Figure 1: FDG-18 PET/CT demonstrating diffuse hypermetabolic lymphadenopathy involving cervical, thoracic, abdominal, retroperitoneal, and inguinal chains (Figures A and B), with a hypermetabolic focus in the rectum (SUVmax 7.6) showing parietal thickening (Figure B), and mesorectal lymph nodes with SUVmax 7.2. The highest uptake was observed in the pericaval chain (SUVmax 6.1), with the largest lymph nodes measuring up to 2.4 cm in the retroperitoneum.

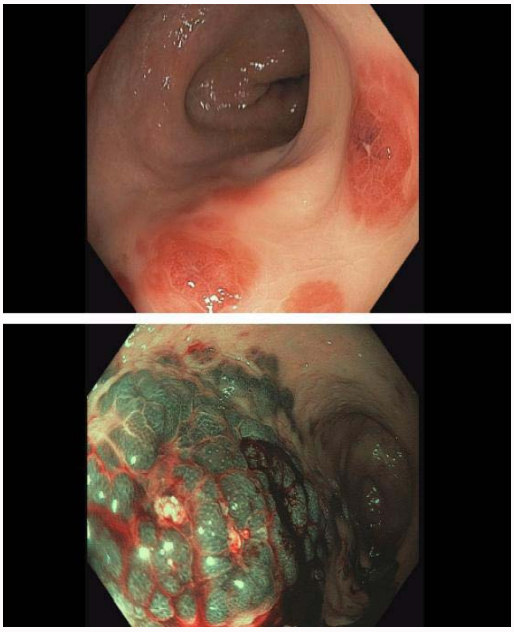

Figure 2: Colonoscopy findings showing nodular and ulcerated lesions throughout the colon (Figures A and B), consistent with gastrointestinal involvement by Kaposi sarcoma in an immunosuppressed transplant recipient.

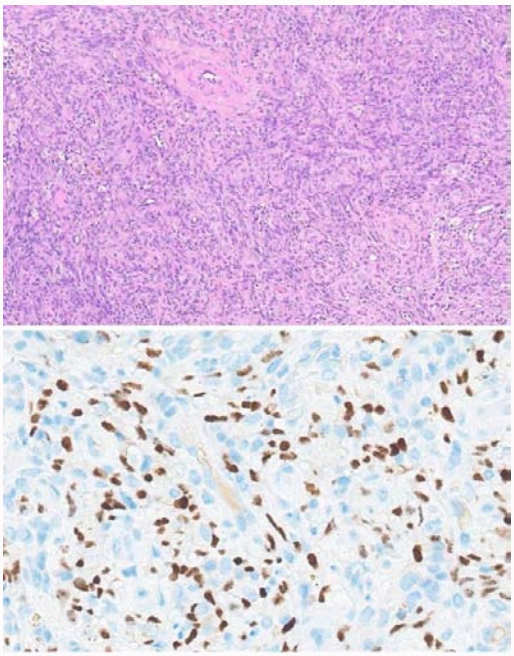

Figure 3: Kaposi sarcoma showing proliferation of atypical spindle cells (Figure A, HE 20×), with positive nuclear immunoexpression for HHV-8 (Figure B, clone 13B10 Cell Marque).

Computed tomography revealed diffuse lymphadenopathy, splenomegaly, and rectal wall thickening. Chest CT showed small pulmonary nodules and mild pleural effusion. FDG-18 PET/CT demonstrated hypermetabolism in cervical, thoracic, abdominal, and inguinal lymph node chains, as well as a rectal hypermetabolic focus (SUVmax 7.6) (Figure 1).

Investigation for opportunistic infections was negative,

including bronchoscopy with bronchoalveolar lavage, serologies, and parasitological examinations. Qualitative HHV-8 viral load was detectable.

Upper gastrointestinal endoscopy and colonoscopy revealed nodular and ulcerated lesions in the gastrointestinal tract, suggestive of Kaposi sarcoma (Figure 2). Inguinal lymph node biopsy confirmed HHV-8-associated multicentric Castleman disease, with immunohistochemical positivity for the virus. Gastric and rectal biopsies confirmed Kaposi sarcoma (Figure 3).

Antiretroviral therapy was reinitiated, and prophylaxis with sulfamethoxazole-trimethoprim was instituted. Treatment with doxorubicin (20 mg/m²) combined with rituximab was chosen, without specific antiviral therapy for HHV-8.

The patient showed good treatment tolerance, without significant adverse events. He was discharged after 23 days, with partial symptom improvement. After four weeks of rituximab therapy, digestive symptoms resolved with progressive clinical recovery.

He returned to his hometown in May 2026, maintaining oncological follow-up and good clinical evolution Table 1.

Table 1: Laboratory findings on admission.

Parameter	Result	Reference Value
Hemoglobin	9.2 g/dL	13.0-17.0 g/dL
Leukocytes	3,200/mm³	4,000-10,000/mm³
Platelets	78,000/mm³	150,000-400,000/mm³
Creatinine	1.8 mg/dL	0.7-1.2 mg/dL
Urea	68 mg/dL	17-49 mg/dL
INR	1.45	0.96-1.30
Alkaline Phosphatase	832 U/L	40-129 U/L
Gamma-GT	205 U/L	8-61 U/L
CRP	124 mg/L	≤5 mg/L
Ferritin	2,648 ng/mL	22-491 ng/mL
Triglycerides	189 mg/dL	<150 mg/dL
Fibrinogen	178 mg/dL	200-400 mg/dL

Note: CRP: C-Reactive Protein; Gamma-GT: Gamma-Glutamyl Transferase; INR: International Normalized Ratio.

Discussion

Kaposi Sarcoma (KS) is the most frequent manifestation associated with HHV-8 in solid organ transplant recipients, with an incidence up to 500 times higher than in the general population [10,11]. In contrast, HHV-8-associated Multicentric Castleman Disease (MCD) remains rare in this setting, with few cases described in the literature. KSHV-related complications in transplant recipients may result from viral reactivation, primary infection, or donor-derived transmission, although systematic screening for HHV-8 is not routinely performed [2,12]. Clinical evolution is influenced by the type of transplanted organ, intensity of immunosuppression, and the serological status of the donor and recipient, with more aggressive forms frequently associated with primary infection [2,10].

The pathogenesis involves a complex interaction between immunosuppression, chronic inflammation, and cell proliferation. HHV-8 establishes latency in B cells and expresses proteins such as LANA, which is essential for maintaining the viral genome and serves as a diagnostic marker. During the lytic phase, viral cytokines are produced, especially vIL-6, which promotes inflammatory activation, angiogenesis, and cell proliferation [5,7,13,14].

Diagnosis is challenging, particularly in HIV-positive and transplanted patients, due to overlap with opportunistic infections and other lymphoproliferative disorders. In the present case, constitutional symptoms and persistent diarrhea initially raised common infectious hypotheses in this context. The exuberant gastrointestinal involvement, in the absence of cutaneous lesions, also hindered early recognition of KS a pattern already described in immunosuppressed patients [2,10]. Definitive diagnosis depends on histopathological and immunohistochemical evaluation, with identification of LANA-positive spindle cells in KS and plasmablastic proliferation in MCD. The concomitant presence of diffuse lymphadenopathy and HHV-8 positivity was fundamental for the diagnosis of MCD in this case.

The coexistence of KS and MCD is uncommon but may occur in settings of combined immunosuppression, such as the association between solid organ transplantation and HIV infection. In these patients, higher viral load and more intense HHV-8 reactivation are described, even with relatively preserved CD4+ counts [2,11].

Management is based on reduction of immunosuppression, optimization of antiretroviral therapy, and disease-specific treatment. KS with visceral involvement usually requires systemic chemotherapy, while HHV-8-associated MCD responds primarily to rituximab. mTOR inhibitors may play an additional role due to their antiproliferative and antiangiogenic effects, possibly related to inhibition of the PI3K/Akt/mTOR pathway [1,4,14]. Despite in vitro antiviral activity against HHV-8, agents such as ganciclovir and valganciclovir have not demonstrated consistent clinical benefit in treating these malignancies. Studies suggest partial responses in MCD, but without sustained curative effect [14].

This case illustrates the diagnostic and therapeutic complexity of HHV-8-associated diseases in patients with combined immunosuppression. The rarity of the coexistence between visceral KS and MCD, associated with the absence of systematic screening and clinical overlap with other conditions, reinforces the need for high clinical suspicion. Early recognition is essential to institute adequate treatment and potentially modify the prognosis of these patients.

Conclusion

This case underscores the diagnostic and therapeutic challenges of HHV-8-related diseases in patients with combined immunosuppression from solid organ transplantation and HIV infection. The coexistence of gastrointestinal Kaposi sarcoma and multicentric Castleman disease, although rare, should be considered in immunocompromised patients presenting with prolonged fever, lymphadenopathy, and visceral involvement without an established etiology. The absence of cutaneous lesions does not exclude KS, particularly in profoundly immunosuppressed individuals, and this atypical presentation may delay diagnosis. Early histopathological and immunohistochemical evaluation is essential for definitive diagnosis and should be pursued proactively when clinical suspicion arises. Management requires a multifaceted approach, including reduction of immunosuppression, optimization of antiretroviral therapy, and disease-specific treatment with systemic chemotherapy and rituximab. Heightened clinical suspicion, multidisciplinary coordination, and awareness of the overlapping spectrum of HHV-8-related diseases are crucial to improving outcomes in this complex patient population.

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