

BRIEF ARTICLES

A Case of Kaposi Sarcoma Co-infected with Cytomegalovirus

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ABSTRACT

Background: Kaposi sarcoma (KS) is the most common AIDS-associated neoplasm. It is a vascular neoplasm that occurs as a result of infection with a human herpesvirus (HHV-8). Cytomegalovirus (CMV) and HHV-8 both belong to Herpesviridae, a family of DNA viruses. CMV is highly prevalent in the general population and can cause localized or disseminated disease in AIDS patients.

Case: A 42-year-old male with an HIV infection presented with a painful ulcerated growing white nodule with overlying telangiectatic vessels on the right third toe that he noticed 4 weeks ago. A tangential biopsy revealed a vascular proliferation which was diffusely positive for HHV-8. In addition, scattered inclusion bodies were observed, indicating co-infection with CMV.

Conclusion: This case reinforces the importance of considering KS as a potential diagnosis in all AIDS patients with unusual exophytic growths to avoid potential misdiagnosis and improper management.

INTRODUCTION

Kaposi sarcoma (KS) is an acquired immunodeficiency syndrome (AIDS)-defining illness and is the most common malignancy in AIDS patients.^[1] It is a vascular neoplasm that occurs as a result of infection with a human herpesvirus (HHV-8). KS It is strongly associated with a low CD4 count, which accounts for the significant decline in its incidence after the widespread introduction of highly active antiretroviral therapy (HAART).¹

CASE PRESENTATION

A 32-year-old African American male presented for an evaluation of a painful growing nodule on the right third toe. He noticed the nodule 4 weeks ago. No other skin lesions were reported by the patient that may have proceeded the nodule. The patient was diagnosed with HIV 20 years ago but was not compliant with treatment. He was started on a combination pill of cobicistat, elvitegravir, emtricitabine, and tenofovir, 10 days prior to his current presentation. He had a baseline CD4 count of 222 and a viral load

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of approximately 2 million at that time. His past medical history noted for repeated chlamydia and gonorrhea and recurrent genital herpes. His family history and review of systems was unremarkable. Physical examination revealed an ulcerated white nodule with overlying telangiectactic vessels on the medial aspect of the right third toe (Figure 1).

The initial differential diagnosis included granuloma (lobular pyogenic capillary hemangioma), dermatofibroma, KS, bacillary angiomatosis, and amelanotic melanoma. A superficial shave biopsy was obtained. Histology revealed a vascular proliferation with spindled endothelial cell which was diffusely positive for HHV-8 on immunohistochemistry (Figure 2). Scattered intranuclear inclusions were also observed, which stained positive for CMV, confirming a co-infection with this virus (Figure 3).

DISCUSSION

This case had many features that were not consistent with the typical presentation of AIDS-associated KS. The painful presentation, lack of multifocal involvement, and absence of a characteristic violaceous patch or plaque phase made KS a less likely diagnosis. Therefore, it is important to have KS on differential diagnosis for exophytic growths in HIV patients. Early diagnosis is important for timely screening for visceral involvement by testing the stool for occult blood and obtaining a chest x-ray.

CMV is a member of the herpesvirus family. It is highly prevalent in the general population and is largely asymptomatic.² However, it can cause localized or disseminated disease in AIDS patients. Contrary to studies conducted earlier in the HIV epidemic that found no association between CMV and KS, newer studies found that seropositive CMV patients are at higher risk for developing KS.³ In addition, CMV has demonstrated the ability to induce the lytic cycle in HHV 8 in a vitro model.⁴ CMV's seroprevalence has increased over the past two decades, which may have played a role in KS occurring at higher CD4 counts since the beginning of the HIV epidemic.⁵ The coexistence of these two

Figure 1. white nodule with an overlying telangiectatic vessel on the medial right third toe.



Figure 2. Histology. Proliferation of endothelial cells forming vascular spaces infiltrating through collagen on hematoxylin and eosin (2A; 10X). An HHV-8 immunohistostain is positive within the endothelial cells (2B; 10x).



Figure 3. Histology. Inclusion bodies (3A; 40x). A CMV immunohistostain (3B; 10x)





viruses within the same cutaneous lesion is rare. However, KS co-infected with CMV has also been reported from other sites, including the oral mucosa, thyroid, the ileocecal area, and the thymus, in both HIV-positive and HIV-negative patients⁶. In almost all reported cases, there were relatively few cytomegalic cells, which were detectable only with careful observation and could have been easily overlooked.⁶

CONCLUSION

This case supports the highly variable clinical spectrum of KS and emphasizes the importance of considering KS as a potential diagnosis in all HIV patients with unusual exophytic growths to avoid potential misdiagnosis and improper management. KS co-infected with CMV generally presents with few cytomegalic cells that can be easily missed. Furthermore, CMV was shown to play a role in KS-associated pathogenesis. The increased seroprevalence of CMV may play a role in KS occurrence at higher CD4 counts.

Conflict of Interest Disclosures: None

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