Case Report

Coexistence of intestinal Kaposi sarcoma and plasmablastic lymphoma in an HIV/AIDS patient: case report and review of the literature

Bing Wang, Bingbing Song, Cyrus Oster, Jeffery Cao, Anwar Raza, Jun Wang

Department of Pathology, Loma Linda University Medical Center, CA 92354, USA

Correspondence to: Jun Wang, MD. Department of Pathology, Loma Linda University Medical Center, 11234 Anderson Street, Room 2516, Loma Linda, CA 92354, USA. Email: jwang@llu.edu.

Abstract: Human immunodeficiency virus (HIV) infection or acquired immunodeficiency disease (AIDS) is associated with increased risk for various malignancies including Kaposi sarcoma (KS) and lymphoma. We report a rare case of coexistence of KS and plasmablastic lymphoma (PBL) in the gastrointestinal (GI) tract in a HIV/AIDS patient. A brief review of literature is also presented.

Keywords: Human immunodeficiency virus (HIV); acquired immunodeficiency disease (AIDS); Kaposi sarcoma (KS); plasmablastic lymphoma (PBL); intestinal neoplasms

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Introduction

Human immunodeficiency virus (HIV) infection or acquired immunodeficiency disease (AIDS) is associated with increased risk for various malignancies including Kaposi sarcoma (KS) and lymphoma (1-3). Although the skin is the most common site of involvement for KS, the gastrointestinal (GI) tract is also often involved in HIV/AIDS patients (4). Lymphoma is another common GI malignancy seen in AIDS patients. Here we report a case of HIV/AIDS with multiple HIV-associated malignancies involving the GI tract.

Case presentation

A 32-year-old Hispanic male patient with a history of HIV and cutaneous KS presented to the ED with bloody diarrhea. Lab tests confirmed HIV with increased HIV viral load in peripheral blood (590K copies/mL) and decreased CD4 count (251/μL, 12%). The patient was receiving highly active antiretroviral therapy (HAART) treatment previously and was suspected to be non-compliant with his medication regimen. Esophagogastroduodenoscopy suggested esophagitis in the middle and distal thirds of the esophagus and multiple erythematous nodules were found in the second and third portions of the duodenum. Colonoscopy also revealed numerous erythematous nodules in the colon. Multiple biopsies from the GI tract were taken for pathological examination.

Histopathology

The esophageal biopsies showed ulcerative esophagitis with prominent lymphoplasmacytic infiltrate positive for herpetic viral and cytomegaloviral inclusions, indicative of immunodeficiency in the patient (*Figure 1*).

Multiple biopsies of the duodenum showed histopathological findings consistent with GI KS (*Figure 2*): spindle cells form slit-like spaces containing red blood cells as well as presence of extravasated red blood cells. The lesion packed the lamina propria accompanied by a variably mixed mononuclear inflammatory cell infiltrate. Usually, the tumor cells display strong immunoreactivity for HHV8 LNA-1 antibody. In the current case, given the history of HIV and presence of multiple skin KSs combined with typical histopathological findings of KS, no HHV8 LNA-1 immunostaining was performed.

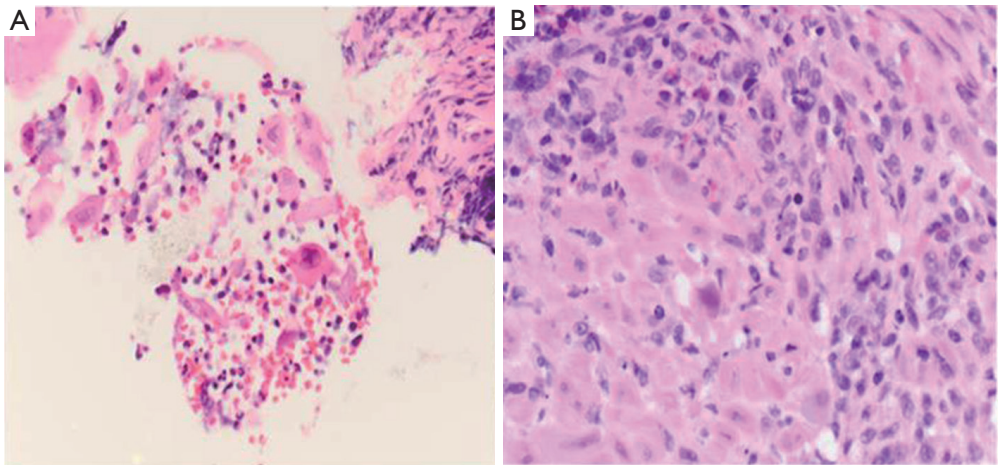

Figure 1 Esophageal biopsies showing esophagitis with herpetic viral inclusions (A) and cytomegaloviral inclusions (B) (HE stain: A, ×200; B, ×400).

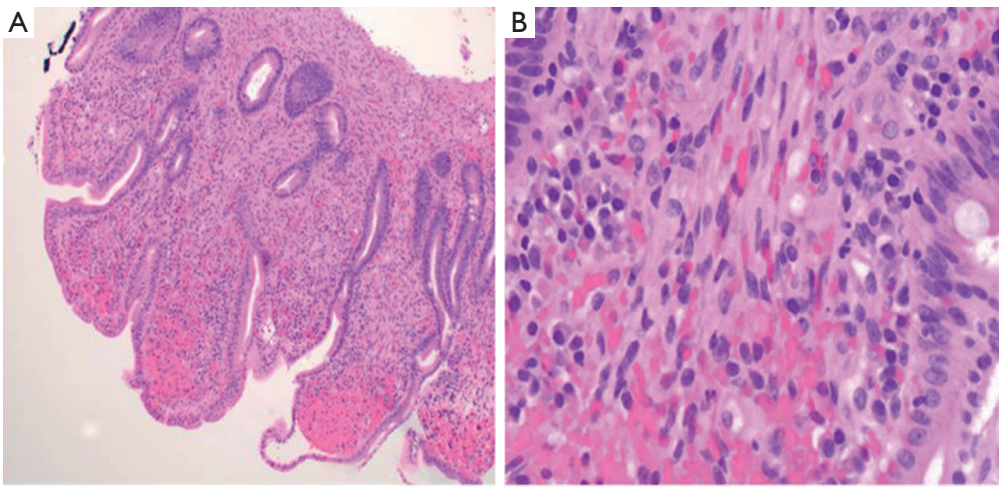

Figure 2 Duodenal biopsies showing typical KS features: spindle cell lesions with extravasated red blood cells that expand lamina propria (A: low power view; B: high power view (HE stain: A, ×40; B, ×400).

Interestingly, in the multiple fragments from colonic mucosa, as in the duodenum, KS was identified and the haphazard vascular network of KS was highlighted by CD34 (*Figure 3*). Furthermore, foci of atypical large cell infiltration were found on the surface of the colonic mucosa separate from the KS lesion. These large cells often appeared plasmacytoid/immunoblastic with abundant cytoplasm, eccentric nuclei and variably prominent nucleoli with disruption of the lamina propria and mucosal surfaces demonstrated. The large cells were CD45 positive, suggestive of hematopoietic cell lesions. Further immunohistochemical studies were carried out to help diagnose the colonic lesions. The atypical large cells were positive for CD138, MUM-1, CD30, and CD4 but negative for CD20. Ki-67 stain showed a very high proliferative index (*Figure 4*). Given the patient's history of HIV infection, the morphological and immunophenotypic features were most consistent with the diagnosis of plasmablastic lymphoma (PBL). Further testing for positive EBV (EBER) and negative anaplastic lymphoma kinase (ALK) confirmed the diagnosis.

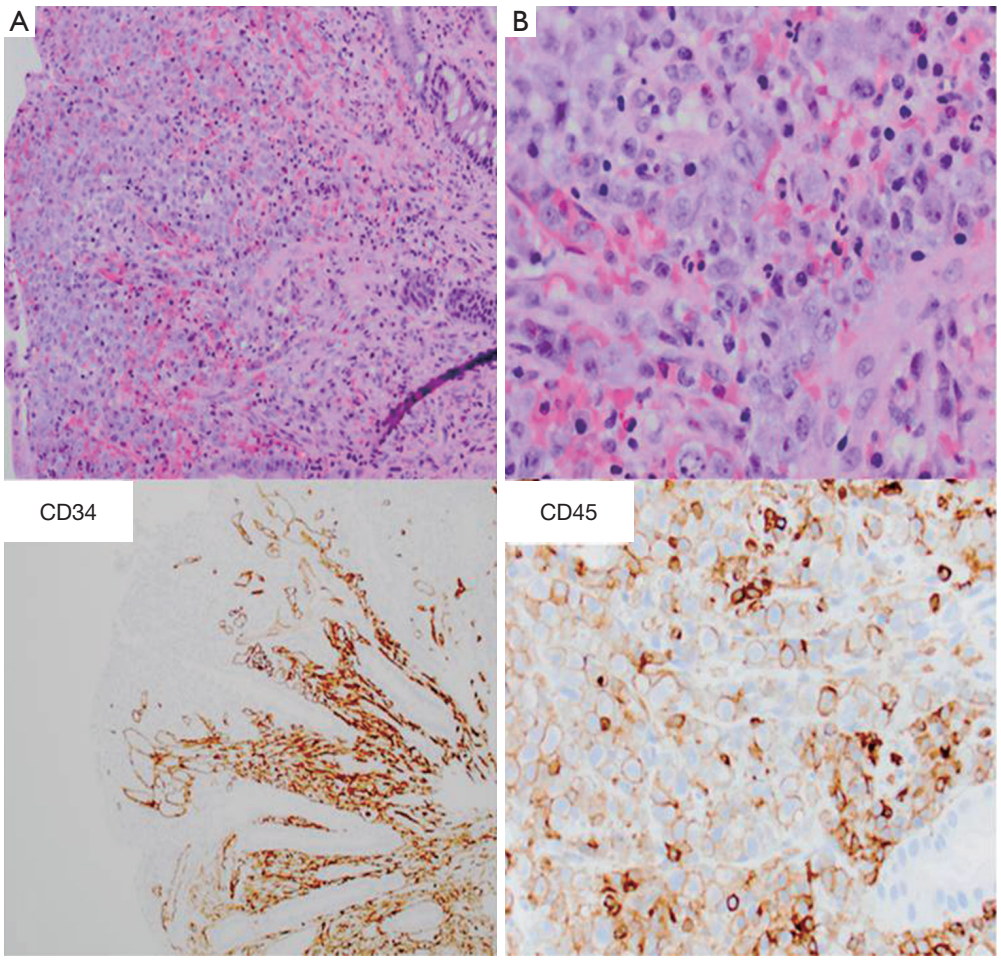

Figure 3 Descending colon biopsies (A: top, HE stain, ×100; bottom, immunoperoxidase stain, ×400. B: top, HE stain, ×400; bottom, immunoperoxidase stain, ×100). The low power view (A) revealed two different types of lesions. Foci of atypical large cell infiltration were found near the mucosa surface, and those large cells often appear plasmacytoid with abundant cytoplasm, eccentric nuclei and variably prominent nucleoli (B), which was CD45 positive. Underneath is the lesion of KS, CD34 staining showing the haphazard vascular network of Kaposi sarcoma in the lamina propria. KS, Kaposi sarcoma.

Discussion

HIV/AIDS is associated with increased risk for multiple malignancies, including KS and lymphoma (1-3). In the current case, the HIV patient presented with multiple KSs and coexistent PBL in the GI tract. The patient also displayed esophagitis associated with multiple viral infections including herpes and cytomegalovirus. This unique case fully exemplified that immunodeficiency in HIV/AIDS patients may lead to multiple viral infections and increased risk for various malignancies in the intestinal tract.

PBL is a rare form of AIDS-related lymphoma, only accounting for approximately 2.6% of lymphomas. The exact incidence of PBL is not known, but almost all of them are associated with HIV infection or conditions of immunosuppression (5-8). In a comprehensive review of PBL by Castillo *et al.*, most of the PBL occurred in the oral cavity with only approximately 13% of the 112 reported PBL originating in the GI tract (9). Similar to other common lymphomas in the GI tract, clinical symptoms of PBL include abdominal pain, diarrhea, nausea and GI bleeding (9). HIV-associated PBL is aggressive, and 60% of patients present with advanced clinical stage at diagnosis (9-11).

The exact mechanism of lymphomagenesis in HIV patients is not completely understood. HIV is not regarded as a directly transforming virus due to lack of consistent

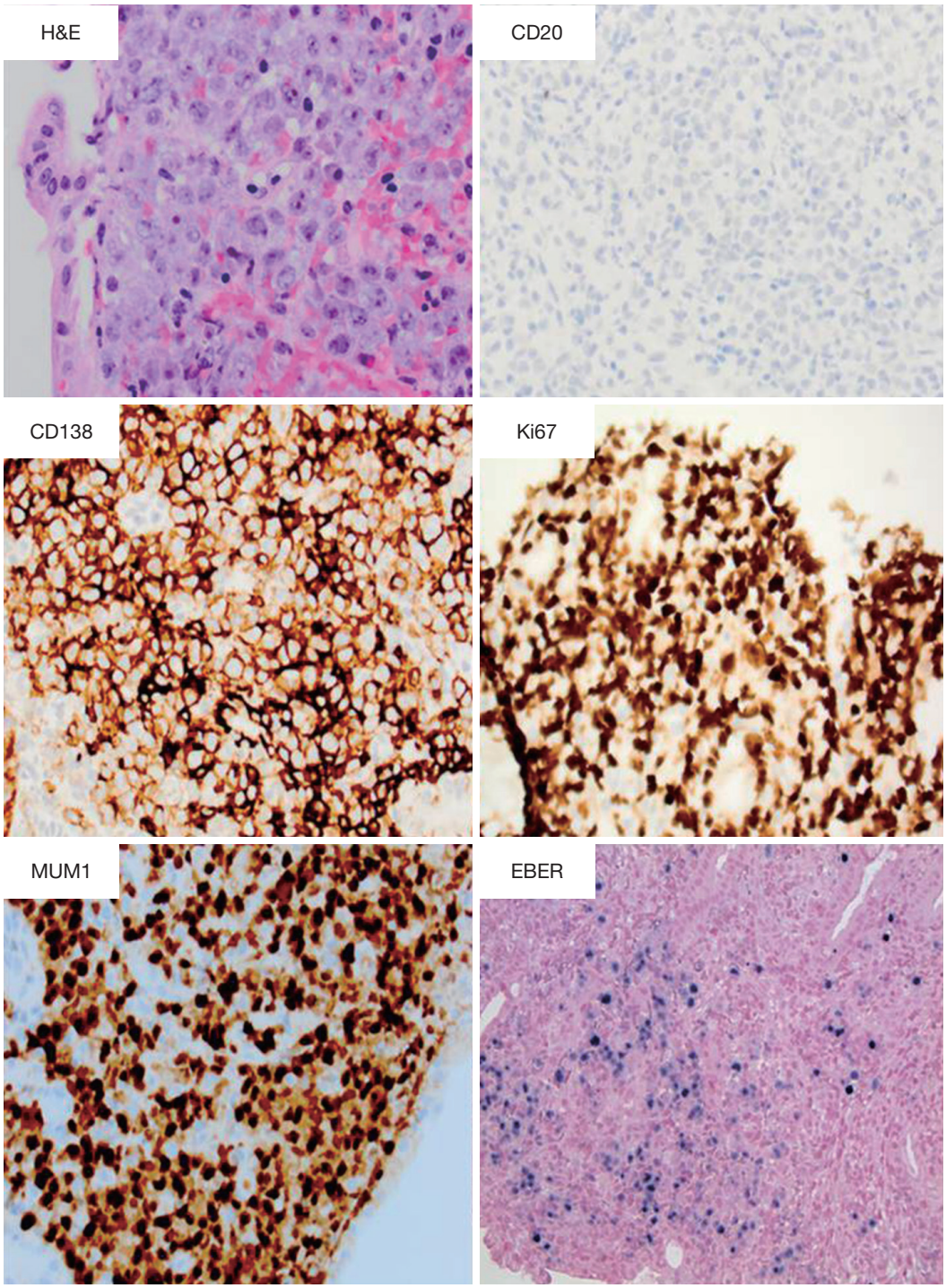

Figure 4 PBL. In H&E stain, a population of large cells with plasmacytoid appearance. PBL cells are CD20 negative, CD138 and MUM1 positive, and show a high Ki67 expression. PBL exhibits positive EBER expression by *in situ* hybridization (original magnification for EBER is ×200 and the rest are all ×400). PBL, plasmablastic lymphoma.

insertion at a transformative site (11). Instead, HIV infection disrupts normal immunomodulatory functions, which allows super-infection by other oncogenic viral pathogens such as Epstein-Bar virus and Kaposi-sarcoma virus, subsequently leading to multiple malignancies such as various lymphomas and KSs (12).

HIV targets human CD4 "Helper" T-cells due to high affinity of viral gp120 proteins for the CD4 molecules. CD4 T-cells function as immune system modulators in both adaptive immune system (i.e., B cells) as well as the innate immune system (e.g., macrophages and monocytes). Once infected by HIV, CD4 cells cannot execute the normal immunomodulatory functions, and eventually are depleted through activation-induced cell death (13,14).

EBV infection has been postulated in the development of many lymphomas. Most lymphomas in HIV-infected patients also show EBV positivity, especially PBL, 60-100% of which are EBV positive (6,8,15). EBV may contribute to lymphomagenesis in variety of ways. The EBV genome encodes a variety of products promoting EBV infection and transformation. For example, the EBV latent membrane protein 1 (LMP1), a constitutively active viral protein, was able to transform lymphocytes in mouse cell models and is required for EBV-mediated B-cell transformation (16,17). LMP2A increases the expression of genes associated with cell cycle induction and inhibition of apoptosis, and alters gene expression profile similar to those described in Hodgkin/Reed-Sternberg (HRS) cells of Hodgkin lymphoma. LMP2A expression may lead to development of Hodgkin lymphoma (18). Epstein Barr nuclear antigens (EBNAs) can directly interfere with P53 and P16 functions (19,20). Furthermore, recent data suggest that HIV may directly contribute to the development of lymphomas in HIV patients by inducing B-cell activation. HIV gp120 has been shown to activate B cells and induce class switch recombination from IgM to IgA and IgG by up-regulating activation-induced cytidine deaminase (21). Recently, another HIV P17 matrix protein with different variants has been found being able to directly promote B-cell growth and thus possibly contribute to malignant transformation of B cells (22).

Lymphomagenesis is complex, and the mechanisms for the pathogenesis of different types of lymphomas in HIV patients are far from clear. PBL is thought to derive from post germinal-center, terminally differentiated B cells, probably in transition from immunoblast to plasma cells (23,24). A recent study has shown that recurring rearrangements involving MYC, a well-known oncogene with the immunoglobulin gene, in PBL (25).

Diagnosis of GI PBL needs endoscopic biopsy and pathological examination. The 2008 World Health Organization (WHO) classification of Tumors of Hematopoietic and Lymphoid Tissues specifically recognizes PBL as a diffuse immunoblastic lymphoma. Morphologically, the cells resemble immunoblasts with some plasmacytic differentiation. They are large and have round to oval nuclei that may be eccentrically located. A single central prominent nucleolus or several peripherally located nucleoli are present, and the cytoplasm is moderate to abundant with a paranuclear hof. However, a plasmablastic morphology may be seen in other lymphoproliferative disorders, such as multiple myeloma, Burkitt lymphoma, diffuse large B-cell lymphoma (DLBCL) with plasmacytoid differentiation and ALK-positive DLBCL. Immunophenotyping has great value in differentiating PBL from other neoplasms. PBL has the features of plasma cells such as CD138 and IRF4/MUM1 positivity, but are negative or weakly positive for CD20 or PAX5. EBV by EBER *in situ* hybridization is positive in most cases, but LMP1 is variably expressed due to low sensitivity. The cytogenetic changes of PBLs are unclear yet. But deregulation of MYC gene expression through translocation or amplification has been reported in 49% of the cases. The common translocations include immunoglobulin heavy-chain, kappa or lambda light-chain genes, like Burkitt lymphoma (23,24).

KS is a rare mesenchymal tumor involving blood and lymphatic vessels, and was first described by Dr. Kaposi in 1872 (26). However, the incidence increases significantly in the HIV/AIDS population, and the rate of epidemical KS was 100,000-fold greater than that of the general population (27). KS is classified into four forms: classic KS, endemic African KS, iatrogenic/immunosuppressive KS, and AIDS-related KS. AIDS-related KS is the most aggressive form. KS is primarily a skin lesion, but sometimes involves visceral organs. Visceral involvement, such as GI tract involvement, is especially common in AIDS-KS with nearly a quarter of patients having GI tract involvement (28-31).

Human herpesvirus-8 (HHV8), also known as KS-associated herpesvirus (KSHV), is etiologically associated with KS (32,33). Following the acute infection, the virus establishes latent infection inside cells. KSHV codes for a functional bcl-2 homologue, which disrupts the antiapoptotic effects of bcl-2 protein and may contribute to neoplastic cell expansion (34). KSHV-encoded G-protein couple receptor (GPCR) may also act as a vial oncogene.

KSHV also codes for proteins mimicking human cytokines such as IL-6, IL-6, which can activate VEGF and induce angiogenesis (35). Expression of those viral genes disrupts immune response and signaling pathways, leading to increased cell proliferation, angiogenesis and local inflammation and eventually the initiation and progression of KS (29). KS growth is further enhanced by HIV coinfection. HIV infection significantly decreases the host immune surveillance, which plays a key role in eliminating the virus during the acute infection and suppressing viral reactivation. HIV-1 virus also induces various inflammatory cytokines and growth factors that enhance tumor growth (2,36,37).

Clinical presentations of GI tract involvement by KS vary from asymptomatic to diarrhea and intestinal bleeding. As the lesions grow, symptoms of intestinal obstruction and intussusception may occur (38-40). The gross appearances of GI tract KS under endoscopy vary from erythematous macules or papules to polypoid lesions (41-43).

The histological features of GI KS are similar to cutaneous KS, which show spindle cells forming slit-like spaces containing red blood cells and pack the intestinal lamina propria. The lesions can sometimes infiltrate the overlying mucosa (29,44,45). KS cells are usually positive with the endothelial markers such as CD31, CD34, and factor VIII-related antigen. The diagnosis of KS has been greatly simplified using immunohistochemical stain for HHV-8 latent nuclear antigen (LNA-1) in regards to differentiation from other vascular lesions (46-48).

Several lesions in the GI tract may mimic KS including GI angiosarcoma, bacillary angiomatosis and pyogenic granuloma. Both angiosarcomas and KS are positive for vascular endothelial markers, however, clinically angiosarcomas are not associated with HIV/AIDS or immunosuppression (49-51). Bacillary angiomatosis also often occurs in immunocompromised patients such as those with HIV infection. It often presents as a cutaneous lesion but occasionally occurs in GI tract. However, Warthin-Starry staining will reveal the existence of extracellular bacilli (52-54). Pyogenic granuloma is a lobular hemangioma which commonly occurs in skin but occasionally occurs in the GI tract. Histologically the features of slit-like vessel channels, endothelial nuclear atypia, and red blood cell extravasation often present in KS will not be present in pyogenic granuloma (55-57). In difficult cases, HHV-8 latent nuclear antigen (LNA-1) staining can help, and it is positive in KS and negative in other lesions.

In conclusion, this case shows concurrence of two HIV-associated malignancies in the GI tract: a relatively common malignancy of KS, and an uncommon malignancy of PBL. The coexistence of these two malignancies exemplified the increased risk for various malignancies in immunocompromised HIV/AIDS patients.

Acknowledgements

None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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