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CASE REPORT

AIDS-associated plasmablastic lymphoma presenting as a poorly differentiated esophageal tumor: A diagnostic dilemma

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Abstract

Plasmablastic lymphoma (PBL) is a rare form of diffuse large B-cell lymphoma characterized by weak/absent expression of conventional B-cell markers and strong expression of plasma cell markers. It is strongly associated with human immunodeficiency virus (HIV) and Epstein Barr virus infection, and shows an unusual tropism to the oral cavity. Herein we describe a patient with AIDS who presented with weight loss and dysphagia owing to a large gastroesophageal mass. His radiographic and endoscopic findings and long history of cigarette consumption suggested carcinoma. Biopsy demonstrated a poorly differentiated tumor stained negatively to routine lymphoid markers including CD20. However, gene rearrangement studies confirmed a B-cell process and a more detailed immunohistochemical analysis revealed the cells stained positively for CD138 (plasma cell antigen). These findings were diagnostic of PBL. Our report reviews the wide differential diagnosis of PBL and underscores the importance of a broad array of viral and molecular studies needed to establish this diagnosis.

INTRODUCTION

An increasingly large number of HIV-infected patients are developing HIV-associated, but not AIDS-defining neoplasms including esophageal cancer, head and neck malignancies, lung cancer, liver and anal neoplasms and renal cell carcinoma[1]. The pathogenesis of these tumors is complex and is in part related to alcohol and cigarette consumption, the consequences of chronic inflammation including hepatitis C virus (HCV)-associated cirrhotic liver disease and the presence of oncogenic viruses such as human papilloma virus, Epstein Barr virus (EBV) and hepatitis B virus (HBV). Many of these patients are receiving highly active anti-retroviral therapy (HAART) and are no longer destined to die from AIDS-related complications.

Plasmablastic lymphomas (PBLs) were originally described in HIV-infected patients as an aggressive variant of diffuse large B-cell lymphoma (DLBCL), with a peculiar tropism for the oral cavity[2]. PBLs are composed of rapidly growing, large neoplastic cells displaying some degree of plasma cell differentiation. Phenotypically, PBLs display an unusual immunohistochemical profile characterized by weak or absent expression of conventional B-cell markers

coupled with strong expression of plasma cell markers. Recent reports have identified this neoplasm in extra oral sites in both HIV seropositive and seronegative individuals.

Herein, we describe the clinical course of a patient with AIDS who presented with a constrictive and ulcerating esophageal mass which was thought initially to be a poorly differentiated carcinoma, but after a more detailed immunohistochemical evaluation, proved to be PBL. We emphasize the unusual clinical features of this rare form of non-Hodgkin's lymphoma (NHL) and the diagnostic challenges associated with its identification.

CASE REPORT

A 40-year-old Caucasian male with a 25-pack-year history of cigarette consumption but no alcohol or illicit drug use, sought medical attention. He had lost 20 lbs over a period of 2 mo, and complained of loss of appetite and progressive odynophagia (solids > liquids). He also experienced low-grade fevers and drenching night sweat that was not ameliorated by acetaminophen. His past medical history was significant for HIV and chronic active HBV co-infection, diagnosed a year earlier when, while homeless, he presented with muscle weakness and altered mental status. His initial CD4+ count was < 50 cells/μL and his HIV viral load was > 100 000 copies/mL. Neurological evaluation led to a diagnosis of AIDS-associated encephalopathy and myelopathy. He was transferred to a skilled nursing facility where he received rehabilitatory care along with once daily HAART consisting of a fixed dose coformulation of tenofovir 300 mg and emtricitabine 200 mg, ritonavir 100 mg and atazanavir 300 mg. His condition gradually improved and 6 mo later his CD4+ count increased to 180 cells/μL and his HIV viral load fell to < 75 copies/mL.

On physical examination he appeared disheveled and emaciated with dry mucous membranes, proximal muscle wasting but no hairy leukoplakia or oral candidiasis. There was no lymphadenopathy or hepatosplenomegaly, and his myelopathy-associated spastic gait and lower extremity hyperreflexia were stable. Laboratory tests included: white blood count 7600 cells/mm$^{[3]}$; hemoglobin 12.1 mg/dL; platelet count 303 × 10^3/μL; total protein 7.2 mg/dL; albumin 4 mg/dL; lactate dehydrogenase 496 IU/L (normal range, 125-243 IU/L); normal electrolyte and hepatic transaminase levels; positive HBV surface antigen and HBV e antigen; HBV DNA 9360 copies/mL and negative Hepatitis A virus and HCV serologies. His CD4+ count had dipped to 103 cells/μL, but his HIV viral load remained non-detectable. A chest roentgenogram demonstrated a retrocardiac soft tissue density. Chest and abdominal computed tomogram (CT) further showed the density to be a 4.9 cm × 5.1 cm concentric distal esophageal mass associated with extensive gastric wall thickening (Figure 1). The dominant mass corresponded to an area of intensely increased metabolic activity (SUV = 40.3) and was associated with right iliac adenopathy

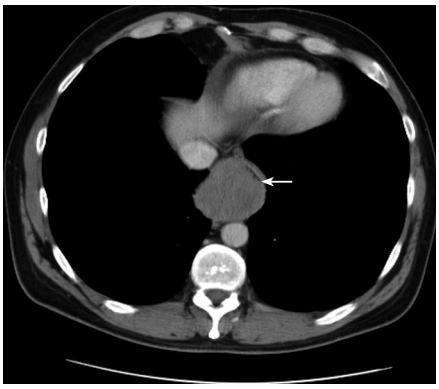

Figure 1 CT scan of chest highlighting the large distal esophageal mass. Note the impressive constriction of the esophageal lumen (arrow).

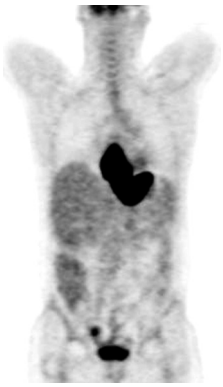

Figure 2 Whole body PET scan demonstrates intensely increased metabolic activity corresponding to the large esophagogastric mass. There is also focal increased activity in a right iliac node.

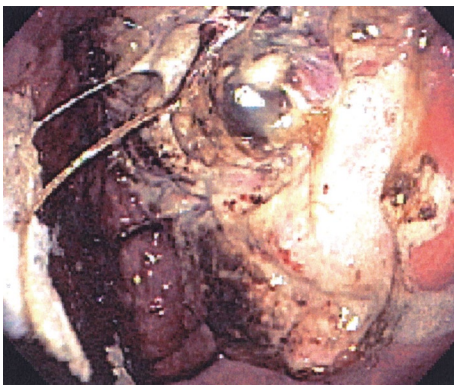

Figure 3 Upper Endoscopic evaluation shows esophageal mass with ulcerative features which extended into the gastric fundus.

(SUV = 40.6) on a whole body F-18 Fluorodeoxyglucose positron emission tomography (PET) scan (Figure 2).

Upper endoscopic evaluation revealed that the mass constricted 40% of the esophageal lumen and extended into the proximal stomach where a large ulcerative lesion was identified (Figure 3). Biopsies of the tumor showed a poorly differentiated, malignant neoplasm composed of irregular sheets of cells, which in many areas were largely necrotic. Cells were cytologically atypical with somewhat eccentric vesicular nuclei and prominent nucleoli (Figure 4A). On initial review of the biopsy, a poorly differentiated carcinoma of gastroesophageal origin was favored. However, immunohistochemical evaluation showed that the tumor cells stained negatively

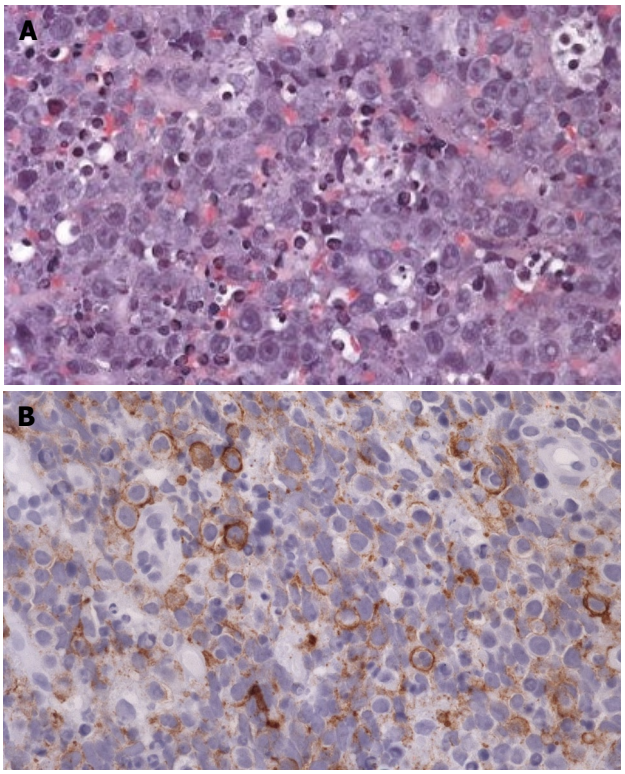

Figure 4 **A**: HE image shows a poorly differentiated, malignant neoplasm composed of irregular sheets of cells. Cells are cytologically atypical with somewhat eccentric vesicular nuclei and prominent nucleoli (x 40); **B**: Immunohistochemistry shows patchy but strong staining of tumor cells for CD138 (x 40).

for epithelial markers (cytokeratin), lymphoid markers (CD20, CD3, CD30 and PAX-5) and melanocytic markers (HMB-45, melan-A and S100). Based on these inconclusive results, additional immunohistochemical stains for markers of lymphoid and plasmacytoid differentiation was performed. The tumor cells were stained strongly positive for CD-45 (leukocyte common antigen) and CD-138 (plasma cell marker) and weakly positive for CD-79a (pan B-cell marker) (Figure 4B). Tumor cells were also positive for the B-cell transcription factors Bob-1 (focal) and Oct-2 (focal weak). In situ hybridization studies were positive for EBV but negative for human herpes virus type-8 (HHV-8, Table 1). A clonal rearrangement of the immunoglobulin heavy chain gene was identified on polymerase chain reaction analysis.

Following a diagnosis of PBL, the patient was treated with combined chemotherapy consisting of liposomal doxorubicin, cyclophosphamide and etoposide (LACE regimen) in addition to HAART[3]. He tolerated the therapy well, with mild nausea with each treatment cycle, alopecia and a single episode of culture negative neutropenic fever. A follow-up CT scan taken after the second treatment cycle showed dramatic improvement with just mild residual wall thickening of the distal esophagus. After completing a total of six cycles of chemotherapy, his dysphagia and weight loss resolved and a CT-PET scan showed complete resolution of the abnormal activity in esophagus and right iliac region.

Table 1 Immunohistochemical findings of esophagogastric mass

Type	Antigen	Immunoreactivity
Epithelial markers	Cytokeratin 35BH11	Negative
	EMA	Diffusely positive
	BER-EP4	Negative
Lymphoid markers	CD-45	Positive
	CD-3	Negative
	CD20	Negative
	CD79a	Equivocally positive in some cells
	PAX-5	Negative
	CD30	Negative
	CD138	Positive (patchy, strong)
B-cell transcription factors	Bob-1	Positive
	Oct-2	Positive
Melanocyte markers	Melan-A	Negative
	HMB-45	Negative
	S100	Negative
Viral markers	HHV-8	Negative
	EBV (*in situ* hybridization)	Positive

EMA: Epithelial membrane antigen; PAX-5: Paired box gene-5; HHV-8: Human Herpes Virus type-8; EBV: Epstein Barr virus.

His post-chemotherapy upper endoscopic evaluation did not reveal persistent NHL and 6 mo later, he remains in remission.

DISCUSSION

In 1997, Delecluse and colleagues were the first to describe in HIV-infected patients, the occurrence of a high-grade malignant lymphoma subtype named PBL which exclusively involved the oral cavity[2]. These tumors possessed a unique immunohistochemical phenotype characterized by their failure to express common lymphoid markers while stained positively for plasma cell markers[4]. Over the past decade, the clinical spectrum of this NHL has expanded to include extra oral involvement in patients with and without HIV infection. Unusual sites of PBL involvement have included the skin, nasal and paranasal sinuses, long bones, lungs, stomach, anorectum, omentum, testes, spermatic cord, bone marrow, sacrococcygeal cysts and central nervous system[5-10].

PBL accounts for 2.6 % of all AIDS-related NHLs, and rarely represents the sentinel manifestation of AIDS[11-13]. In HIV-negative individuals, PBL is often associated with iatrogenic immunosuppression such as seen with organ transplantation[14]. It has also been reported in association with Azathioprine and Infliximab therapy for management of inflammatory bowel disease[15,16]. PBL has been diagnosed in children as young as age 7, but the majority of the reported cases involve middle-aged adults.

Patients with PBL can be divided into three distinct categories[17-20]. The first and the more common PBL variant is localized to the oral mucosa, although the tumor may also involve nodal or extranodal sites. Histologically, this variant is characterized by a

Table 2 Differential diagnosis of PBL

Tumor subtype	Carcinoma	PBL	Common DLBCL	BL	Plasmacytoma
Tumor cell size	Large	Large	Large	Intermediate	Intermediate
CD20	-	- to ±	+	+	-
CD45	-	Variable	+	+	-
CD138	-	+	-	-	+
VS38c	-	+	- to ±	-	+
MUM1	-	+	-	-	+
EBV	-	+	-	+	-
HHV-8	-	Variable	-	-	-

PBL: Plasmablastic lymphoma; DLBCL: Diffuse large B cell lymphoma; BL: Burkitt's lymphoma; CD: Cluster of differentiation; MUM-1: Multiple myeloma oncogene-1-protein; EBV: Epstein Barr virus; HHV-8, human Herpes Virus type-8; +: Expression of the antigen in the majority of cells; -: Absence of antigen expression; ±: Weak antigen expression.

monomorphic population of immunoblasts with no or minimal plasmacytic differentiation. The second PBL category is distinguished by its plasmacytic differentiation and extra oral presentation. The tumor cells are composed predominantly of immunoblasts, plasmablasts and mature plasma cells. The third variant of PBL has also been reported in association with HHV-8 and multicentric Castleman's disease. Patients typically present with lymphadenopathy and splenomegaly, often with plasmablasts circulating in the peripheral blood.

Despite morphologically resembling B-cell immunoblasts, PBL is associated with plasma cell immunophenotype, with loss of B cell markers (CD20) and surface immunoglobulin, and acquisition of plasma cell surface markers [VS38c, CD38 (syndecan-1), MUM-1, CD-138]. The plasmablasts are variably immunoreactive for CD45 and CD-79a[6,11,15]. They usually lack Bcl-6, the germinal center-associated B-cell antigen and PAX-5/BSAP, a nuclear factor that is present from the precursor B-cell stage and in all mature B cells but lost in terminal differentiation to plasma cells[11,17]. Newer B-lineage markers like the transcription factors OCT.2 and BOB.1 may be helpful to confirm the B-cell origin of these tumors[20].

EBV infection is strongly associated with PBL. *In situ* hybridization for EBV Encoded RNA in tumor specimens has reportedly ranged from 60% to 100%, suggesting that EBV plays an important role in PBL pathogenesis[9,15,17,21]. In contrast, HHV-8 is not consistently associated with PBL, although rare cases have identified HHV-8 in conjunction with HIV infection, PBL and multicentric Castleman's disease[9,15,17,22,23]. Patients with chronic HBV infection are more likely to develop NHLs, but specific cases of PBL have not been documented in this population[24].

The differential diagnosis of PBL may overlap with a variety of other clinicopathologic entities (Table 2). When PBL presents as an oral lesion, it may be confused with periodontal disease like odontogenic cellulitis, KS or melanoma[12]. Carcinomas can be distinguished from PBLs based on presence of immunoreactivity for epithelial markers such as cytokeratin. Primary effusion lymphoma, as opposed to PBL, usually presents with serous effusions without detectable tumor masses, and is strongly associated with both HHV-8 and HIV infection. The presence of serum monoclonal proteins, and/or bone involvement with radiographically evident lytic lesions, favors the diagnosis of plasma cell myeloma rather than PBL[25].

In the pre-HAART era, HIV-infected patients with PBL were destined to die from their disease shortly after their NHL diagnosis. In the initial report by Delecluse and colleagues, 9 of 11 patients with long-term follow-up had a median survival of 6 mo[2]. In the HAART era, patient prognosis appears better and prolonged survival is the goal of treatment. Among six patients treated with anthracycline-based multiagent chemotherapy in conjunction with HAART, five were alive and disease free with a median follow-up of 22 mo[15]. The importance of an intact immune system in preventing or controlling PBL is underscored by reports of tumor regression with HAART alone and in the absence of chemotherapy[26-28]. Ironically, the early phase of immune reconstitution may be a fertile ground for NHL development. Our patient's diagnosis of lymphoma within 1 year of HAART initiation, and in the context of a rapidly improving CD4+ cell count and non-detectable HIV viral load may be another example[29].

Finally, our patient's complaint of dysphagia in the setting of a large esophagogastric mass, together with his long-standing history of smoking was disconcerting for carcinoma. Though the history of fever, night sweat and an elevated LDH raised the possibility of a lymphoma, the finding of poorly differentiated tumor cells stained negatively for routine lymphoid markers of B and T-cell differentiation did not support this diagnosis. But the subsequent demonstration of the plasma cell antigens, which are not typically part of the routine immunohistochemical panel, in conjunction with a broader array of viral and molecular studies helped us to establish the diagnosis of PBL.

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