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Extracavitary/solid variant of primary effusion lymphoma

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Abstract Primary effusion lymphoma (PEL) is a distinct clinicopathologic entity associated with human herpesvirus 8 (HHV8) infection that mostly affects patients with immunodeficiency. Primary effusion lymphoma usually presents as a malignant effusion involving the pleural, peritoneal, and/or pericardial cavities without a tumor mass. Rare cases of HHV8-positive lymphoma with features similar to PEL can present as tumor masses in the absence of cavity effusions and are considered to represent an extracavitary or solid variant of PEL. Here, we report 3 cases of extracavitary PEL arising in human immunodeficiency virus–infected men. Two patients had lymphadenopathy and underwent lymph node biopsy. One patient had a mass involving the ileum and ascending colon. In lymph nodes, the tumor was predominantly sinusoidal. The tumor involving the ileum and ascending colon presented as 2 masses, 12.5 × 10.6 × 2.6 cm in the colon and 3.6 × 2.7 × 1.9 cm in the ileum. In each case, the neoplasms were composed of large anaplastic cells, and 2 cases had "hallmark cells." Immunohistochemistry showed that all cases were positive for HHV8 and CD138. One case also expressed CD4 and CD30, and 1 case was positive for Epstein-Barr virus–encoded RNA. Evidence of B-cell differentiation was poorly developed in all tumors. These cases highlight the importance of assessing HHV8 in an anaplastic tumor that arises in a human immunodeficiency virus–positive patient and further contributes to the limited literature currently available for extracavitary PEL.

Keywords: Non–Hodgkin lymphoma; Kaposi sarcoma–associated herpesvirus (HHV8); HIV; Primary effusion lymphoma; Plasmablastic lymphoma; Anaplastic large cell lymphoma

1. Introduction

Human herpesvirus 8 (HHV8), also known as *Kaposi sarcoma–associated herpesvirus*, is an oncogenic, herpes double-stranded DNA lymphotropic virus. It is endemic in sub-Saharan Africa and the Mediterranean region, with a seroprevalence of 50% to 70% and 20% to 30%, respectively. In North America, there is a 1% to 3% infection rate among asymptomatic blood donors [1].

Primary effusion lymphoma (PEL), also known as *body cavity–based lymphoma*, is an HHV8-associated large B-cell neoplasm that typically involves the pleural, pericardial, or peritoneal body cavities as an effusion and is not associated with a tumor mass [2]. Primary effusion lymphoma is a rare lymphoma representing approximately 4% of all AIDS-related lymphomas and 0.3% of all aggressive lymphomas in human immunodeficiency virus (HIV)–negative patients in the clinical setting of immunodeficiency [2]. Cases of PEL have been reported in older individuals, in the absence of immunodeficiency, but primarily from HHV8 endemic geographical areas [3,4]. Clinically, patients usually present with B symptoms. The diagnosis of PEL is usually made by examination of cytologic preparations of involved effusion fluids. The tumor cells are large with abundant basophilic cytoplasm, sometimes with vacuoles, and irregular nuclear contours and prominent nucleoli. The cytomorphologic appearance can range from immunoblast-like to anaplastic, and the

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Table 1
Clinicopathologic features of 3 cases of extracavitary PEL

Case	Age (y)	Sex	HIV	Presentation	Biopsy site	Follow-up (mo)
1	55	M	+	Flulike symptoms, fever, night sweats, CT—a large abdominal mass	Ileum, colon (right hemicolectomy)	13, NED
2	42	M	+	Weakness, fever, generalized lymphadenopathy, polyclonal γ-globulinemia, polyneuropathy	LN	25, NED
3	42	M	+	Generalized lymphadenopathy	LN	Lost to follow-up

CT, computed tomography; LN, lymph node; M, male; NED, no evidence of disease.

tumor cells frequently exhibit plasmablastic differentiation. Detecting evidence of infection by HHV8 in the tumor cells is essential for the diagnosis of PEL.

Rarely, HIV+ patients develop HHV8-positive lymphomas with features similar to PEL involving solid organs, without evidence of effusion. Extranodal sites that can be involved include the skin, lung, gastrointestinal tract, central nervous system, and, rarely, lymph nodes. This extracavitary/solid presentation of PEL may precede [5] or follow a typical case of PEL [6,7], or be the only site of involvement [8]. Extracavitary/solid variants of PEL show similar morphology, immunophenotype, genotype, and HHV8

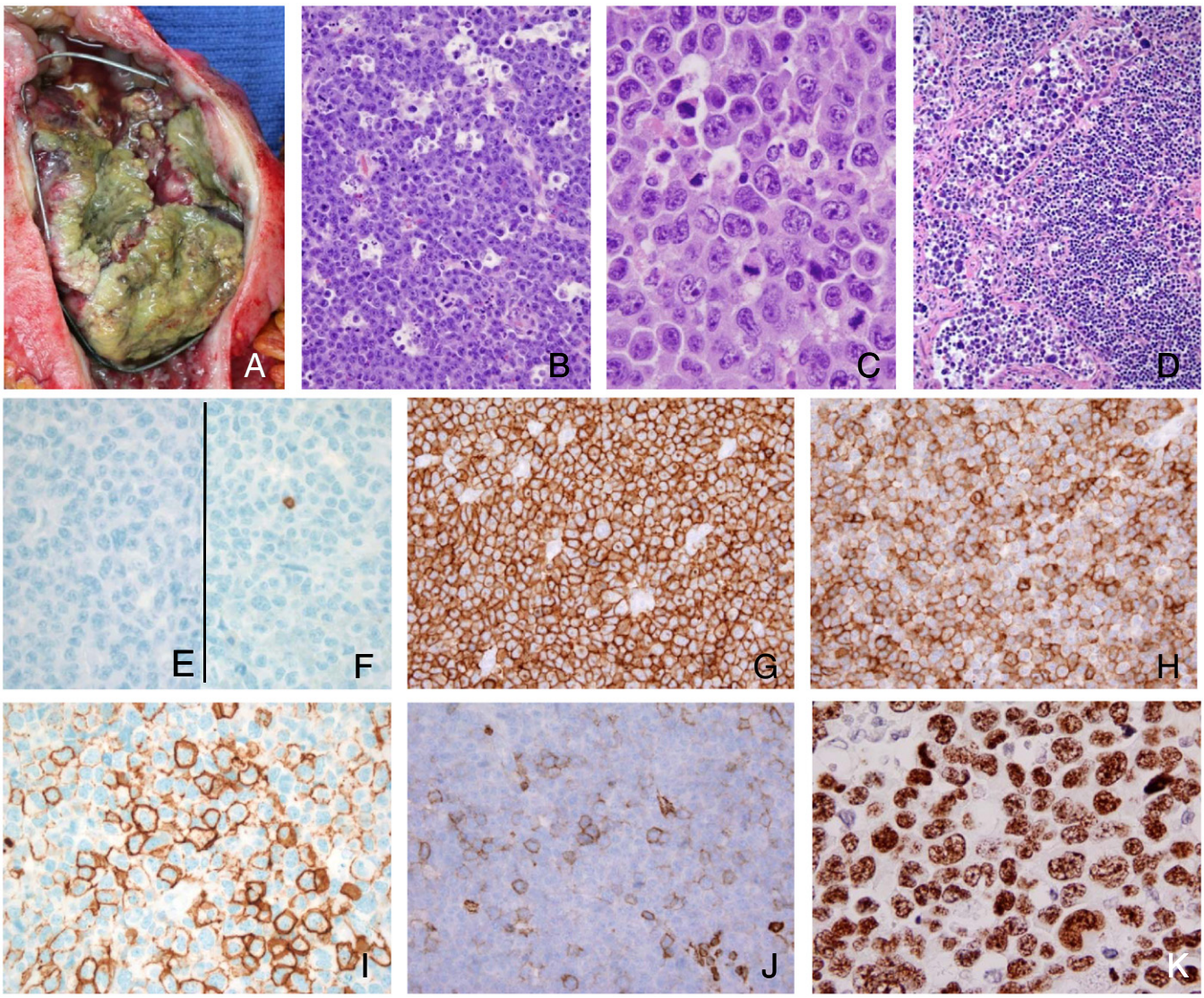

Fig. 1. Case 1. (A) Gross features of solid-variant PEL that presented as a large mass involving the colon. (B) Starry-sky pattern. (C) Anaplastic/plasmablastic cytologic findings of tumor cells including "hallmark" cells. (D) Medium-power view of pericolonic lymph nodes showing intrasinusoidal infiltration by lymphoma cells. (E-K) Immunohistochemical results for case 1. The tumor cells were negative for CD20 in ~99% of cells (E) and CD3 (F). The tumor cells expressed CD4 (G) and CD30 (H), and a subset of tumor cells were positive for CD138 (I). (J) Only a few tumor cells were positive for CD45/LCA. (K) The tumor cells were intensely positive for HHV8.

viral status to classic PEL [9-11] and are referred to in the World Health Organization classification as extracavitary or solid PEL [11].

Here, we report the clinicopathologic features of 3 cases of an extracavitary/solid variant of PEL involving lymph nodes and the gastrointestinal tract. One of these cases closely resembled anaplastic large cell lymphoma (ALCL). These cases exemplify the importance of assessing for HHV8 infection in the diagnosis of lymphomas arising in HIV+ patients.

2. Materials and methods

Three cases of solid PEL were identified: all were sent in consultation to the Department of Hematopathology at The University of Texas, MD Anderson Cancer Center, Houston, Texas. Two cases were excisional lymph node biopsy specimens, and 1 case was a right hemicolectomy specimen. All specimens were fixed in formalin, and 4-μm-thick tissue sections were prepared and stained with hematoxylin and eosin.

Most immunohistochemical stains were performed at other institutions and were reviewed at our hospital at the time of diagnosis. The specific antibody dilutions for these immunostains are unknown. CD30 (Ber-H2; 1:20; Signet Laboratories Inc, Dedham, Massachusetts), Ki-67 and CD4 (1:300, 4B12; Novocastra, Newcastle, UK) were performed retrospectively for this study using formalin-fixed, paraffin-embedded tissue sections, heat-induced antigen retrieval, and an automated immunostainer. Results of in situ hybridization analysis for Epstein-Barr virus (EBV)–encoded RNA (EBER), also performed at other institutions, were available for all 3 cases.

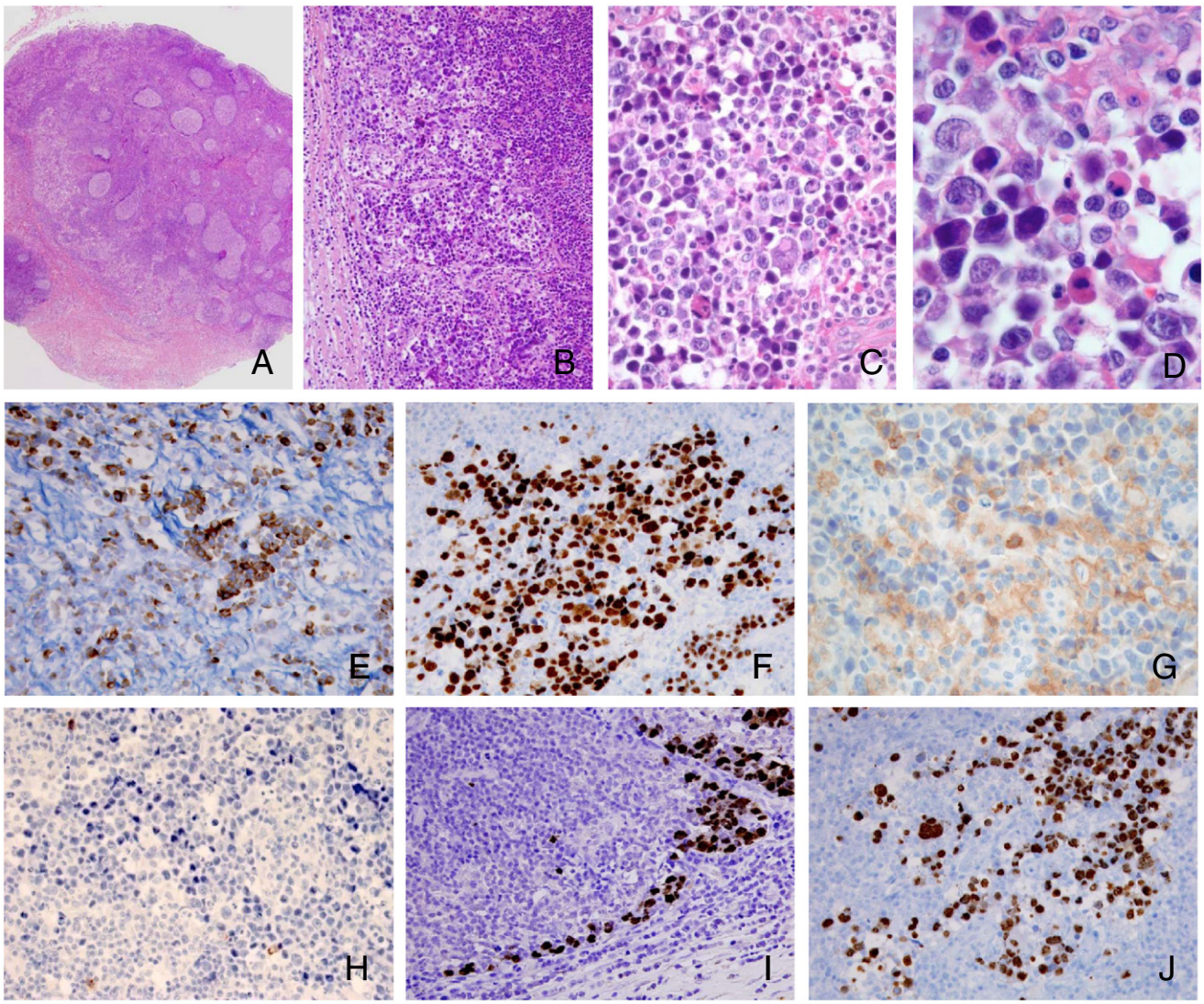

Fig. 2. Case 2. (A, B) Low- and medium-power view of the lymph node involved by solid PEL showing an intrasinusoidal growth pattern. (C, D) The tumor cells showed anaplastic features. (E-J) Immunohistochemical studies revealed that a small subset of the tumor was positive for CD79a (E). The tumor cells were positive for MUM1 (F) and showed focal and weak reactivity for CD4 (G). The tumor cells were negative for CD30 (H) but positive for EBER (I) and HHV8 (J).

3. Results

3.1. Histologic findings

A summary of the clinicopathologic features of these 3 cases is provided in Table 1. Case 1 was a right hemicolectomy specimen that showed 2 ulcerated masses, 1 in the ascending colon 12.5 × 10.6 × 2.6 cm and the other 1 in the terminal ileum 3.6 × 2.7 × 1.9 cm (Fig. 1A). Histologic sections revealed a malignant neoplasm with a diffuse, starry sky pattern (Fig. 1B), mainly involving the submucosa with focal invasion of the muscularis propia. The tumor cells were large, with a plasmablastic appearance characterized by eccentric nuclei with abundant cytoplasm, sometimes with a perinuclear hof. Neoplastic cells with large horseshoe-shaped nuclei, similar to those known as "hallmark cells" and multinucleated cells, were also seen (Fig. 1C). Mitotic figures were numerous. Lymphovascular invasion was prominent. Some pericolonic lymph nodes displayed large atypical lymphoid cells located almost exclusively within sinusoids (Fig. 1D). Focal hyaline-vascular Castleman-like follicles were also seen.

Cases 2 and 3 were lymph node biopsy specimens. In both specimens, the tumor was present as cell clusters and scattered large atypical lymphoid cells located almost exclusively within sinusoids (Fig. 2A, B). The lymphoma cells included cells with plasmablastic and anaplastic morphology and occasional multinucleated Reed-Sternberg–like cells (Fig. 2C, D). Mitoses were easily identified. One case had hallmark cells. The uninvolved lymph node parenchyma showed lymphoid follicles with reactive germinal centers with relatively thin mantle zones that were well delineated from the germinal centers without blurring. Sheets of mature plasma cells in the interfollicular and subcapsular regions were present. No hyaline-vascular changes or other features of Castleman disease were evident.

3.2. Immunohistochemical findings

A summary of the immunophenotypic findings of these 3 cases is provided in Table 2. In case 1, the tumor cells were positive for CD45/LCA (weak), CD138 (subset), CD30 (strong), HHV8 (all cells with dot-like staining pattern), CD4, and λ light chain and were negative for CD3, CD5, CD79a, anaplastic lymphoma kinase 1(ALK1), latent membrane protein 1 (LMP1), and κ light chain. Ki-67 was positive in ~100% of cells. Rare (~1%) tumor cells were positive for CD20 (Fig. 1E-K). Immunohistochemical studies also performed on the pericolonic lymph nodes showed that the tumor cells were positive for HHV8. Cells positive for HHV8 were not identified in mantle zones, and thus, there was no support for HHV8-associated multicentric Castleman disease or associated microlymphomas. The tumor cells were negative for EBER.

In case 2, the tumor cells were positive for CD138 (subset), MUM1/IRF4, CD79a (small subset), CD4 (focal and weak), and HHV8 and were negative for CD3, CD5, CD10, CD20, BCL6, ALK1, and κ and λ light chains (Fig. 2E-J). CD30 was negative in most tumor cells. The proliferation index as measured by Ki-67 (MIB1) was approximately 70% to 80%. The tumor cells were EBER positive but LMP1 negative.

In case 3, the tumor cells were positive for CD138 and HHV8 and were negative for CD3, CD4 CD5, CD20, CD30, ALK1, and κ and λ light chains. The tumor cells were also negative for EBER.

Table 2
Immunophenotypic characteristics of 3 cases of extracavitary PEL

Antibody/Marker	Case 1	Case 2	Case 3
HHV8 (LANA-1)	+	+	+
EBER	−	+	−
LMP1	−	−	ND
CD45	+, dim	ND	+ focal and weak
CD138	+, subset	+ subset	+
MUM1/IRF4	ND	+	ND
CD30	+	−	−
CD4	+, diffuse	+, focal and weak	−
ALK (ALK1)	−	−	−
CD20	+, subset	−	−
CD79a	−	+, small subset	ND
Bcl-6	ND	−	ND
CD10	ND	−	ND
κ	−	−	−
λ	+	−	−
CD3	−	−	−
CD5	−	−	−
Ki-67/MIB1	100%	70%-80%	ND
Keratin (AE1/AE3)	−	−	−

ND, not done.

4. Discussion

Although others had described lymphomas involving body cavities, it was not until 1995 that Cesarman et al [12] identified HHV8 DNA sequences within a distinct subgroup of HIV-related lymphomas localized in body cavities. Subsequently, Nador et al [2] advocated that this lymphoma was a clinicopathologic entity associated with HHV8.

Classic PEL is a unique form of B-cell lymphoma that presents most frequently in body cavities as lymphomatous effusions without an associated tumor mass. The tumor cells in PELs can have a range of morphologic appearances, especially in cytospin preparations, including large immunoblasts or plasmablasts or cells with marked pleomorphism and anaplastic morphology. Binucleated or multinucleated cells resembling Reed-Sternberg cells can be found.

Primary effusion lymphomas are B-cell neoplasms [11], but they usually lack pan–B-cell markers including CD19, CD20, and CD79a as well as surface and cytoplasmic immunoglobulins (Igs). The B-cell origin of PELs can be demonstrated by the presence of monoclonal Ig gene rearrangements. Evidence points toward a postgerminal center B-cell derivation because most PELs contain somatic

hypermutation of Ig variable region genes as well as frequent somatic hypermutation of the noncoding region of the *BCL6* gene, although expression of BCL6 is generally absent [11]. Consistent with this notion of postgerminal center B-cell derivation is the expression of plasma cell markers such as CD138/Syndecan-1. Recently, gene expression profiling analysis of PEL from HIV+ patients showed features most similar to multiple myeloma as well as EBV-transformed lymphoblastic lymphoma cell lines, indicating a pre–plasma cell or "plasmablastic" profile [13]. Primary effusion lymphomas usually lack T-cell/natural killer cell antigens, although aberrant expression of T-cell markers, as also seen in plasma cell myeloma and plasmablastic lymphoma, can occur.

The spectrum of PEL has been expanded by the identification of cases of extracavitary or solid lymphomas without malignant effusions [8,9,13]. To the best of our knowledge, about 50 cases of a solid variant of PEL have been reported [5-10,14-29]. The patients described in the literature most often presented with extranodal tumors involving the gastrointestinal tract, lung, central nervous system, or skin. Rarely, PEL can involve lymph nodes [22]. These extracavitary/solid variants of PEL show similar morphology, immunophenotype, genotype, and HHV8 viral status to classic PEL. Clinically, however, HIV+ patients who develop a solid-variant PEL appear to have a slightly better survival when compared with patients with classic PEL, with a median survival of 11 months vs 3 months, in one study [9]. Two patients we report were alive at 13 and 25 months of follow-up. This apparent better survival may be explained, at least in part, by the introduction of more recent and effective antiretroviral therapy and novel treatment modalities.

Solid-variant PEL can be difficult to diagnose. Reasons that explain this difficulty include the extracavitary presentation, the presence of morphologic features in common with other more frequent malignant lymphomas, and the immunophenotype that can be null cell or show only limited evidence of B-cell differentiation. The differential diagnosis is broad and can include ALCL, plasmablastic lymphoma, large B-cell lymphoma arising in HHV8-associated multicentric Castleman disease, immunoblastic variant of diffuse large B-cell lymphoma (DLBCL), ALK-positive large B-cell lymphoma, and plasmablastic/anaplastic plasma cell myeloma.

The anaplastic large cell cytologic features including hallmark cells and the intrasinusoidal involvement observed in the cases we report may initially suggest the diagnosis of ALCL. In addition, some of the immunophenotypic findings of these lymphomas including strong positivity for CD30 and CD4, as seen in case 1, may further suggest ALCL. Anaplastic morphology has been previously reported in cases of solid-variant PEL [17,22-24]. The intrasinusoidal involvement of tumor cells also has been described in some nodal cases of solid-variant PEL [17,20,22]. Awareness of the existence of cases of solid-variant PEL that morphologically and immunophenotypically can resemble ALCL is important. We recommend performing HHV8 immunostain in cases of malignant lymphomas resembling ALCL in HIV+ patients to avoid missing the diagnosis of solid PEL.

Plasmablastic lymphoma is the prototype of a large B-cell lymphoma with plasmablastic differentiation. Plasmablastic lymphoma has its highest frequency in HIV+ individuals and most often involves extranodal sites, particularly mucosal sites and the oral cavity [11,30]. The tumor cells are positive for plasma cell markers and, usually, negative or weakly positive for B-cell markers such as CD79a [11]. Unlike PELs, 50% to 70% of PBLs show cytoplasmic IgG. Association with EBV infection is found in most PBLs (60%-75%); however, HHV8 is consistently absent [11].

Large B-cell lymphoma associated with multicentric Castleman disease occurs in patients with or without HIV infection and mainly involves lymph nodes and spleen [11]. It is distinctive from PEL in many ways, although both are associated with HHV8 infection. In large B-cell lymphoma associated with multicentric Castleman disease, HHV8-positive plasmablasts express high levels of cytoplasmic IgM, whereas most cases of PEL lack cytoplasmic Ig. Large B-cell lymphoma associated with multicentric Castleman disease harbors unmutated Ig variable region genes and is derived from $CD27^-$ and $CD138^-$ naive B cells. In contrast, in PEL, the tumor cells usually express CD138 and harbor hypermutated rearranged Ig genes, suggesting their origin from germinal center or postgerminal center B cells [11]. Large B-cell lymphoma associated with multicentric Castleman disease is not associated with EBV, whereas PEL is commonly coinfected with the virus [11].

The immunoblastic variant of DLBCL is characterized by greater than 90% of immunoblasts. Immunoblasts have prominent central and large nucleoli and moderate to abundant cytoplasm, often with plasmacytoid differentiation. Immunophenotypic features are most helpful to establish the diagnosis. The immunoblastic variant of DLBCL is usually strongly positive for B-cell markers such as CD20, PAX-5, and CD79a and is negative for ALK and HHV8.

ALK-positive large B-cell lymphoma is a rare lymphoma type, and morphologically, the tumor cells can exhibit a sinusoidal growth pattern and may show plasmablastic differentiation. However, in contrast with PEL and ALCL, ALK-positive large B-cell lymphoma is negative for CD30, except for a focal and weak staining in few cases [11,31]. The tumor cells are strongly positive for ALK protein, mostly with a restricted granular cytoplasmic staining pattern highly indicative of t(2;17)(p23;q23) or CLTC-ALK fusion protein. Few cases may show cytoplasmic, nuclear, and nucleolar ALK staining associated with the NPM-ALK fusion protein. HHV8 and EBV (EBER and LMP1) are negative [11,31].

Unlike well-differentiated plasma cell neoplasms, the highly aggressive plasmablastic/anaplastic plasma cell myeloma can closely resemble the tumor cells of other high-grade B-cell lymphomas with plasmacytoid features. In practice, the distinction between PEL and plasmablastic plasma cell myeloma frequently depends on clinical

correlation. The presence of a serum monoclonal protein and/or bone involvement with radiographically evident lytic lesions favors the diagnosis of plasmablastic plasma cell myeloma [32]. Plasma cell neoplasms are negative for CD45/LCA, CD30 [32], and ALK; usually express cytoplasmic IgG; and may be positive for CD79a, CD56, and cyclin D1 [11]. Plasma cell myeloma rarely occurs in the setting of HIV infection and is rarely positive for EBV, unlike the case for classic and solid-variant PEL [32].

In conclusion, we report 3 cases of extracavitary/solid-variant PEL that arose in HIV+ men. We report these cases to illustrate the morphologic and immunophenotypic variability of these tumors, including 1 case that very closely mimicked ALK-negative ALCL. Awareness of the existence of solid-variant PEL and assessment for HHV8 infection are essential for correct diagnosis.

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