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Pitfalls in the diagnosis of plasmablastic lymphoma: resolving immunophenotypic ambiguity through integrated diagnosis

Abstract

Plasmablastic lymphoma (PBL), an aggressive large B-cell lymphoma with immunoblastic/plasmablastic morphology and plasmacytic immunophenotype, is often associated with human immunodeficiency virus (HIV) infection. In this report, we describe a 32-year-old man with a history of HIV infection who presented with multifocal lymphadenopathy without extranodal disease or serous body cavity effusions. Histologically, the specimen showed a diffuse, pleomorphic proliferation of large, atypical cells with immunoblastic and plasmablastic features. By immunohistochemistry and in situ hybridization, these large, atypical cells were positive for CD45, MUM1 and EBV (EBER) and lacked representative plasma cell markers, CD138 and CD38, as well as B-cell markers, CD20 and CD19. Only focal kappa light chain excess was seen by in situ hybridization, whereas immunohistochemistry and flow cytometry were negative, making the diagnosis particularly challenging. Targeted mutational profiling by next generation sequencing detected pathogenic mutations in *TP53* and *STAT3* and an *IGH/MYC* fusion. Taken together with clinical, morphological, and immunohistochemical findings, a diagnosis of PBL was made. This case illustrates the pitfall in the diagnosis of PBL and underscores the need for an integrated diagnosis including a broad immunophenotypic panel and mutational profiling to establish an accurate diagnosis.

Keywords: plasmablastic lymphoma, plasmacytic immunophenotype, mutation profiling, BCMA, HIV, EBV

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Highlights

1. Plasmablastic lymphoma (PBL) shows overlap with other lymphomas and myeloma.
2. Nodal presentation, retention of CD45 and lack of plasmacytic markers pose challenges in PBL diagnosis.
3. Pathogenic mutations detected by NGS can aid in the diagnosis of PBL.

Introduction

Plasmablastic lymphoma (PBL) is an aggressive large B-cell lymphoma composed of atypical B cells with immunoblastic and/or plasmablastic morphology and an immunophenotype reminiscent of terminal B-cell differentiation.[1,2] It predominantly occurs in extranodal sites in patients with immune deficiency and dysregulation (IDD) and is associated with Epstein-Barr virus (EBV) in up to 60% of cases.[3,4] Given that PBL is associated with a poor prognosis,[4,5] an accurate diagnosis and prompt clinical management of the patient is of utmost importance.

The essential diagnostic criteria for PBL include plasmablastic/immunoblastic morphology, expression of plasma cell antigens (e.g. CD138, MUM1, Blimp1), absence of mature B cell markers (e.g. CD20 and PAX5), and lack of co-expression of ALK and Kaposi's sarcoma herpesvirus/human herpesvirus-8 (KSHV/HHV8). Desirable diagnostic criteria include EBV positivity, detection of MYC rearrangements (found in up to 60% of patients) and detection of monoclonal *IG* rearrangements.[1,6] In addition, genetic alterations are frequently seen in *MYC*, *STAT3*, *RAS* (*NRAS, KRAS, HRAS*), and *TP53*.[1,4,7] Subsets of lymphomas that can show plasmacytic differentiation or plasma cell neoplasms are in the differential diagnosis of PBL and include diffuse large B-cell lymphoma (DLBCL), particularly, EBV+ DLBCL, Burkitt lymphoma, plasma cell myeloma, ALK-positive large B-cell lymphoma (ALK+ LBCL), and extracavitary (solid) primary effusion lymphoma (EC-PEL).[3,7] Separation of these entities from PBL requires the integration of clinical, immunophenotypic and molecular characteristics to render a precise diagnosis.

We encountered a case of PBL where the diagnosis was reached after extensive immunophenotypic workup and targeted mutational profiling by next generation sequencing (NGS). Given the difficulty imposed by a nodal presentation, loss of lineage- and differentiation-specific markers, and retention of CD45, this case illustrates major pitfalls surrounding the diagnosis of PBL, which we describe in this report.

Case presentation

A 32-year-old man with a history of HIV controlled on anti-retroviral therapy, presented with cervical lymphadenopathy. Positron Emission Tomography (PET) imaging showed hypermetabolic activity involving bilateral neck, bilateral axilla, and left inguinal lymph node regions without extranodal disease or serous body cavity effusions. A core needle biopsy of the right axillary lymph node was performed and showed effacement of the nodal architecture by a diffuse proliferation of large, atypical cells with irregular nuclear borders, single central prominent nucleoli and moderate to abundant cytoplasm. Mitotic figures were frequently seen; however, necrosis was not observed. By immunohistochemistry (IHC), the large, atypical cells were positive for CD45, CD30, MUM1, OCT2, BOB1, CMYC, MEF2B, BCMA and weakly positive for BCL6 and PAX5. EBER in situ hybridization showed EBV positivity in the majority of the atypical cells. These atypical cells lacked staining for CD138, CD38, CD20, CD19, CD10,

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CD15, BCL2, Cyclin D1, EMA, CD68, Melan-A, KSHV/HHV8, CD56, ALK1, STAT6, GATA3, CD43, and HGAL. Staining for CD2, CD3, CD4, CD5, CD7, and CD8 highlighted background T-cells. PDL1 showed a subset of weakly positive tumor cells and scattered background macrophages. Ki67 was positive in 80% of the atypical cells. There was focal kappa light chain restriction by kappa and lambda in situ hybridization whereas light chain restriction was not detected by immunohistochemistry or flow cytometry. By flow cytometry, no significant abnormality or aberrant expression patterns were detected. The CD4:CD8 ratio was 0.8:1.0, indicative of suboptimal antiviral control. Fresh material for karyotypic analysis was not available. PBL was in the differential diagnosis based on morphological features, expression of MUM1, B-cell maturation antigen (BCMA) and EBV (EBER), and lack of expression of CD20 (without prior exposure to rituximab) and CD19. Nodal presentation, retention of strong CD45, PAX5 (weak), OCT2, BOB1, and BCL6 (weak) together with the lack of plasma cell markers, CD138 and CD38, however, were features not typical of PBL (Figure 1).

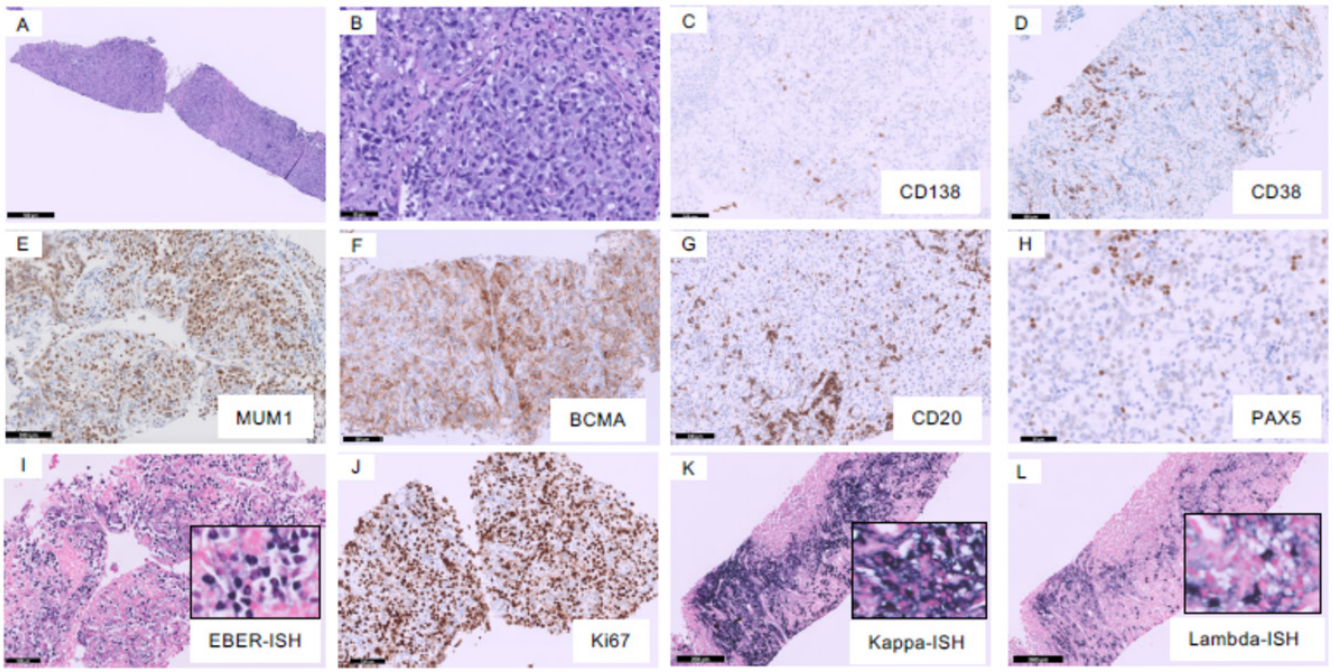


Figure 1 Histology and Immunophenotype of right axillary lymph node

Sections of the biopsy show a diffuse proliferation of large, atypical lymphoma cells with irregular nuclear contours, vesicular chromatin, and single central prominent nucleoli. Hematoxylin and eosin (H&E) (A and B). Lymphoma cells predominantly lack CD138 (C) and CD38 (D), and show positive staining for MUM1 (E) and BCMA (F). These cells lack CD20 (G) with weak positivity for PAX5 (H). EBER in situ hybridization shows positivity in the majority of the lymphoma cells (I). Ki67 highlights 80% of the cells (J). Focal Kappa light chain excess was detected by kappa- (K) and lambda (L) in situ hybridization. Insets in panels I, K and L show high magnification (x300 original magnification) images.

Given the lack of all B-cell surface markers tested, a diagnosis of Burkitt lymphoma was not considered although loss of CD20 and CD19 did not exclude DLBCL. In addition, the morphology and immunophenotype did not support the diagnoses of classic Hodgkin lymphoma or a lymphoma of T- or NK-cell lineage. ALK and KSHV/HHV8 were negative, providing no support for ALK+ LBCL or EC-PEL. Due to the lack of lineage and differentiation-specific markers for a definitive diagnosis, additional ancillary studies were sought. Given the limitation of tissue since only a needle core biopsy was performed, targeted mutational profiling by NGS of 200 gene targets (Stanford Actionable Mutation Panel for Hematopoietic and Lymphoid Malignancies or Heme-STAMP), was selected as the most likely method to provide informative results. There was not enough additional tissue material available for fluorescence in situ hybridization or B-cell receptor gene rearrangement studies. The tumor purity was estimated at 60%. The HemeSTAMP assay typically detects single nucleotide variants, short insertion-deletions, and selected gene fusions in genes recurrently altered in hematopoietic and lymphoid malignancies. In the current case, Heme-STAMP detected the following pathogenic variants: *TP53 R342P* (Variant Allele Frequency [VAF] 14%), *STAT3 D661Y* (VAF 45%), and an *IGHJ6::MYC* fusion. Given the frequency of this constellation of variants in PBL, a final diagnosis of PBL, EBV+, HIV-setting was rendered. No clinical follow-up was available as the patient was lost to follow-up.

Discussion

PBL is an aggressive B cell lymphoma with a poor prognosis, characterized by immunoblastic/plasmablastic morphology and plasmacytic immunophenotype.[1] PBL frequently occurs in patients with immune dysfunction such as HIV infection.[3] A strong association between HIV infection and EBV-positive lymphomas are well recognized although the exact mechanisms underlying EBV's role in lymphomagenesis is still evolving.[3,5,8] We report a case where accurate diagnosis was possible through the integration of a broad immunophenotypic panel together with molecular NGS studies.

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The patient reported in this study presented with multifocal lymphadenopathy and although PBL can rarely involve lymph nodes, it most frequently involves the nasal/oral cavity (~50%), gastrointestinal tract (~20%), bone and soft tissue (~15%) and skin (~5%).[1] In this lymph node core biopsy with sheets of large, atypical cells, plasma cell markers CD38 and CD138, which are typically positive in PBL, were negative. There was no kappa or lambda light chain restriction by flow cytometry or immunohistochemistry, and only focal kappa light chain excess was detected by in situ hybridization. MUM1 was diffusely positive; however, MUM1 expression is seen in activated B cells, T cells, Hodgkin cells, and plasma cells among hematopoietic cells. Its expression is seen in a wide range of lymphomas including DLBCL, plasma cell myeloma, anaplastic large cell lymphoma, and primary effusion lymphoma,[9] which did not lend particular support for PBL in the absence of a plasmacytic immunophenotype and retention of CD45 and at least partial retention of B-cell transcription factors. PRDM1/BLIMP and XBP1 were not available in our immunohistochemistry laboratory and were not performed in this case. BCMA expression, however, did add further support for plasmacytic differentiation and consideration of PBL. The separation of PBL from its main differential diagnostic considerations including EBV+ DLBCL, plasma cell myeloma, ALK+LBCL and EC-PEL, can be challenging (Table 1). This separation relies on the presence of markers that separate mature B-cells from terminally differentiated plasma cells; however, the loss of expected markers in this case (CD138, CD38), retention of CD45 and some B-cell transcription factors, failed to provide sufficient evidence to establish the diagnosis. Recently, anti-CD19 chimeric antigen receptor (CAR) T-cell therapy was reported to induce the evolution of PBL from a patient with chronic lymphocytic leukemia with Richter transformation, underscoring the plasticity of lymphomas under selective pressure.[10]

Table 1 Typical features of entities in the differential diagnosis of plasmablastic lymphoma

Diagnosis	Histologic features	Immunophenotypic features	Molecular features
Plasmablastic lymphoma	Destructive infiltration of immunoblastic and plasmablastic cells	CD138+, CD38+, MUM1+, EBER+(60%), CD20-, ALK-, KSHV/HHV8-	MYC rearrangements (~70%), mutation in JAK/STAT pathway genes (~40%), MAPK/ERK pathway genes (~30%), and TP53 aberrations (~30%).
Diffuse large B-cell lymphoma	Diffuse infiltration of medium- to large cells; a subset may show plasmacytoid differentiation	CD45+, CD19+, CD20+, CD22+, CD79a+, PAX5+, Ki67 proliferation index (high), CD3-	Germinal-center B cell (GCB) -DLBCL: Mutations in EZH2, GNA13, MEF2B, KMT2D, TNFRSF14, B2M, and CREBBP. Rearrangements of MYC and BCL2. activated B cell (ABC) - DLBCL: Mutations in MYD88, CD79B, PIM1, and PRDM1. Rearrangements of MYC and BCL6.
EBV+ Diffuse large B-cell lymphoma	Diffuse infiltration of medium- to large cells; a subset may show plasmacytoid differentiation	CD45+, CD19+, CD20+, CD22+, CD79a+, PAX5+, MUM1+, Ki67 (high), EBV+, CD3-	Lower mutational burden than DLBCL unassociated with EBV, constitutive NF-kB activation, JAK/STAT, P13K/AKT, WNT and NOTCH pathways genes
Burkitt lymphoma	Diffuse infiltration of monomorphic, medium-sized cells with basophilic cytoplasm, multiple small nucleoli	CD10+, BCL6+, Ki-67 (high), BCL2−, MYC+	MYC rearrangements (> 95%)
Plasma cell myeloma	Proliferation of mature plasma cells and plasmablastic cells	CD138+, MUM1+, Light chain restriction is detectable	Recurrent cytogenetic aberrations: 1q21 gain/amplification involving CKS1B, ADAR (ADAR1) (40%); t(11;14)(q13;q32), which involves CCND1/IGH (16%), t(4;14)(p16;q32), involving NSD2/IGH (15%)
ALK-positive large B-cell lymphoma	Diffuse growth of monomorphic large cells with a plasmablastic immunophenotype	ALK+, CD138+, MUM1+, CD20−	ALK rearrangements
Extracavitary Primary effusion lymphoma	Proliferation of large cells with immunoblastic, plasmablastic, or anaplastic features	CD19-, CD20-, CD22-, PAX5-, OCT2-, BOB1-, CD79a-, MUM1+, CD38+/-, CD138+/-, CD30+/-	KSHV/HHV8 and EBV driven pathogenesis, LANA-1 directly binds and suppresses TP53 and RB; v-FLIP constitutively activates NF-kB pathway

In addition to clinical features including history of HIV infection and morphological features, several additional characteristics are useful in the separation of PBL from its diagnostic mimics: while DLBCL is usually positive for B-cell markers CD20 and PAX5, PBL is typically negative or only weakly positive, although the lack of B-cell markers does not entirely eliminate the consideration of DLBCL; further support for plasmacytic differentiation is provided by positivity for PRDM1/BLIMP, XBP1, CD38 and CD138.[11] In our immunohistochemistry laboratory, PRDM1/BLIMP and XBP1 are not performed and the case was negative for CD138 and CD38, providing no further support for plasmacytic differentiation. EBV+ DLBCL, in particular, may show several features in common with PBL and include diffuse positivity for MUM1, EBV/EBER and BCMA. The retention of strong CD45 favors a DLBCL; however, the expression of B-cell transcription factors OCT2 and BOB1, does not differentiate PBL from DLBCL. The most compelling reason to eliminate the consideration of DLBCL, particularly, EBV+ DLBCL, is that this lymphoma arose in an immune deficiency background due to HIV infection.

When differentiating between PBL and plasma cell myeloma, clinical findings of serum monoclonal proteins, hypercalcemia, renal insufficiency, anemia, and lytic bone lesions on radiographs support the diagnosis of plasma cell myeloma. In addition, aberrant co-expression of CD56, cyclin D1 and CD117 are useful adjuncts in supporting plasma cell myeloma, none of which were seen in our patient. Moreover, EBV infection is much more prevalent in PBL (60% overall, and 82% in HIV setting) than in plasma cell myeloma.[1,3,6,12,13,14] Among recurrent chromosomal translocations seen in plasma cell

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myeloma, including t(4;14)(p16;q32), t(11;14)(q13;q32), and t(14;16)(q32;q23), only *MYC* rearrangement have been described in de novo PBL, suggesting the usefulness of molecular testing to separate the entities in the differential diagnosis.[1,14] In ALK+ LBCL, ALK expression by IHC and *ALK* genetic alterations are seen in addition to which EBV is absent.[3,6,15,16] An essential diagnostic criterion of EC-PEL, is the tumor cell positivity for KSHV/HHV8 latency-associated nuclear antigen (LANA),[6,17] while PBL is not associated with KSHV/HHV8.[1,18,19]

In this case, although the definitive diagnosis could not be reached based on the clinical, morphological, and immunophenotypic findings alone, the final diagnosis of PBL was confirmed through an integrated diagnosis that included molecular testing. Mutation profiling detected *TP53* and *STAT3* mutations and an *IGHJ6::MYC* fusion. In PBL, frequent genetic alterations include *MYC,* JAK/STAT pathway genes **(***STAT3, JAK1, SOCS1***),** MAPK/ERK pathway genes **(***RAS***,** *MAP2K1, BRAF***),** NOTCH pathway genes (*NOTCH1, SPEN, NOTCH4, NCOR2*), *IRF4* (6p25.3), and TP53.[1,4] *MYC* rearrangement is detected in over 50% of PBL with *IG* genes as the most common partner gene.[1,4] *STAT3* mutation is detected in as many as 40% of PBL and reported to be more common in patients with HIV and EBV infections, suggesting the efficacy of STAT or JAK inhibitors as a treatment option for HIV-associated PBL.[1,4,7,20,21] *TP53* mutation is seen in approximately 30% of PBL.[1,4,7] Mutational profile of the current case was consistent with the above findings. The pathogenic variants identified by mutational profiling also provide potential therapeutic targets for treatment of PBL in this patient.[4,21]

Overall, our case met the essential diagnostic criteria for PBL including plasmablastic/ immunoblastic morphology, MUM1/IRF4 positivity, and lack of CD20, PAX5, ALK, and KSHV/HHV8. BLIMP1 and XBP1 immunostaining was not performed because the corresponding antibodies were unavailable in our immunohistochemistry laboratory, which is a limitation of this study. This case also met two of three desirable diagnostic criteria which includes EBV/EBER positivity and the presence of *MYC* rearrangement. The presence of a *IGHJ6* rearrangement also supports a B-lineage origin for this lymphoma. An integrated approach was greatly beneficial in this case where a rare nodal presentation together with an aberrant immunophenotype lacking plasma cell differentiation and lack of specificity of markers such as MUM1 made the distinction among the differential diagnostic considerations challenging.

Another important consideration in this case was the expression of B-cell maturation antigen (BCMA), a member of tumor necrosis family receptors that regulates B-cell proliferation and survival. Expression of BCMA is seen in mature B-cells and terminally differentiated plasma cells and neoplasms that arise from these cell types.[22,23] Therefore, BCMA is expected to be an important marker for diagnosis, disease status, and prognosis, and also a potential therapeutic target for hematological malignancies, especially plasma cell myeloma (PCM) and its precursor neoplasms.[23–26] In this case, CD38 and CD138 were negative, but MUM1 and BCMA were positive, providing support for plasmacytic differentiation. Although the number of reports on immunohistochemistry for BCMA is limited, it may be useful in differentiating tumors with a plasmacytic immunophenotype, including PBL, and facilitate improved outcomes for patients through targeted therapy.

Conclusion

In summary, we report a challenging case of PBL that was difficult to diagnose based on clinical, morphological, and immunohistochemical findings. The final diagnosis of PBL was confirmed with the help of an integrated approach to diagnosis that included molecular testing. The keys to this diagnosis were the patient's history of HIV infection, immunoblastic/ plasmablastic morphology, immunohistochemical findings of MUM1, KSHV/HHV8 and EBER positivity, and mutational profile with *TP53* and *STAT3* mutations and *IGH/MYC* fusion. This case emphasizes the need for additional testing including broader panels of immunohistochemistry and NGS, when typical diagnostic features are lacking.

Ethics statement including patient consent statement

Stanford University Institutional Review Board (IRB) approval was obtained for this study. Patient consent was obtained for this study.

Declaration of interest statement

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Conflicts of interest

The authors declare that there are no conflicts of interest.

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