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Plasmablastic Lymphoma: 2026 Update on Diagnosis, Risk Stratification, and Management

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ABSTRACT

Disease Overview: Plasmablastic lymphoma (PBL) is a rare CD20-negative aggressive lymphoma with poor prognosis when treated with standard treatment options. Although PBL is associated with HIV infection and other immunosuppressed states, it can also affect immunocompetent individuals.

Diagnosis: The diagnosis of PBL requires a high clinical suspicion and pathological confirmation. EBV-encoded RNA (EBER) expression and *MYC* gene rearrangements are frequently detected in the malignant cells. The differential diagnosis includes EBV+ diffuse large B-cell lymphoma (DLBCL), extracavitary primary effusion lymphoma, ALK+ DLBCL, and HHV8+ large B-cell lymphoma, among others.

Risk Stratification: Age ≥ 60 years, advanced clinical stage, and high International Prognostic Index scores are associated with worse survival.

Management: Combination chemotherapy regimens, such as infusional EPOCH, are recommended. The addition of bortezomib or daratumumab might improve outcomes. B-cell maturation antigen-targeted therapies have shown early efficacy. The participation of patients with PBL in prospective clinical trials is warranted.

1 | Disease Overview and Pathogenesis

Plasmablastic lymphoma (PBL) was first reported by Delecluse and colleagues in 1997. In the seminal case series, 16 patients were affected by an aggressive CD20-negative lymphoma involving the oral cavity [1]. The malignant cells resembled plasmablasts and lacked CD20 expression despite their B-cell lineage, demonstrated by immunoglobulin gene rearrangements. PBL was characterized by chemotherapy resistance and poor survival. PBL is recognized as a CD20-negative aggressive non-Hodgkin lymphoma (NHL) by the World Health Organization Classification of Lymphomas [2].

PBL is estimated to account for 2% of all HIV-related lymphomas. A study from the National Cancer Database (NCDB) reported 481 cases of PBL between 2010 and 2013, representing less than 1% of all cases of diffuse large B-cell lymphoma (DLBCL) [3]. Despite the strong association with HIV infection, PBL has been reported in other immunodeficiencies and autoimmune conditions, as well as transformation events from indolent lymphoproliferative disorders, and rarely in otherwise immunocompetent individuals [4, 5].

PBL affects individuals of all ages and sexes, although most patients are adults and there is a male predominance. PBL is

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diagnosed at a younger age than DLBCL, not otherwise specified (NOS) [6], and HIV-positive individuals are diagnosed at a younger age than HIV-negative individuals [6]. A higher proportion of Blacks and White Hispanics has been reported in PBL than in DLBCL, NOS [3].

Clinically, PBL frequently presents with extranodal involvement and could affect any organ [6]. The rate of extranodal disease is higher in PBL than in DLBCL, NOS, although the proportion of patients with advanced clinical stage is similar [3]. The most common extranodal sites involved include the oral cavity, gastrointestinal tract, lymph nodes, paranasal sinuses, and skin [6]. Constitutional symptoms are common at diagnosis.

The cell of origin in PBL is the plasmablast, an activated B-cell that has undergone somatic hypermutation and class-switching recombination [2]. The pathogenesis of PBL is not entirely understood, but chronic Epstein Barr virus (EBV) infection and the acquisition of *MYC* gene rearrangements play a role.

Chronic EBV infection has been associated with an antiapoptotic effect in B-cells via overexpression of the latent membrane protein 1 (LMP-1), which mimics CD40, providing T-cell-like support, and inducing the expression of FLIP signaling through nuclear factor-*kappa* B, protecting B-cells from FAS-mediated apoptosis [7]. Additionally, LMP-2A protects infected B-cells from B-cell receptor-mediated apoptosis [8]. Also, EBV produces BHRF-1, a viral BCL-2 homolog, which binds to BAK, PUMA, BID, and BIM, preventing TGF-beta-mediated apoptosis [9]. Finally, EBNA-3A/3C may prevent *MYC*-induced apoptosis [10].

MYC is the most dysregulated oncogene in cancer and regulates DNA replication, cell proliferation, growth, metabolism, and survival [11, 12]. BCL-6 is a repressor of *MYC* in germinal center B-cells, while BLIMP-1 is a repressor of *MYC* in terminally differentiated B-cells. In PBL, *MYC* gene rearrangements dysregulate its function, overcoming the modulatory effects of BCL-6 and BLIMP-1 [13].

Although EBV infection and *MYC* gene rearrangements are prevalent in PBL, they are not universal. Therefore, other mechanisms must play a role in the pathogenesis of PBL. These mechanisms could be related to the RAS–RAF, JAK–STAT, MCL1, IRF4, MAPK–ERK, PI3K-AKT, Wnt, and NOTCH pathways [14–20]. A proposed pathophysiology of PBL is depicted in Figure 1.

2 | Diagnosis and Differential Diagnosis

The best approach to diagnose PBL is to obtain tissue for pathological evaluation. A relatively large sample is desirable, given PBL's unusual morphology and absence of CD20 expression. Therefore, excisional, incisional, or core needle biopsies are preferred over fine-needle aspiration biopsies.

The immunophenotype of PBL is characterized by expression of classic plasma cell markers, such as CD79a, IRF-4/MUM-1, BLIMP-1, CD38, and CD138, lack of expression of B-cell markers, such as CD20 and PAX-5, and variable CD19 and CD45 expression [21–23]. Some cases have an aberrant expression of T-cell markers, such as CD2, CD3, CD4, or CD8. The proliferation marker Ki-67 is expressed by almost all the neoplastic cells. The expression of BCL2 is variable [22]. The expression of CD10 and BCL6 is uncommon and consistent with a non-germinal center origin. EBV involvement by

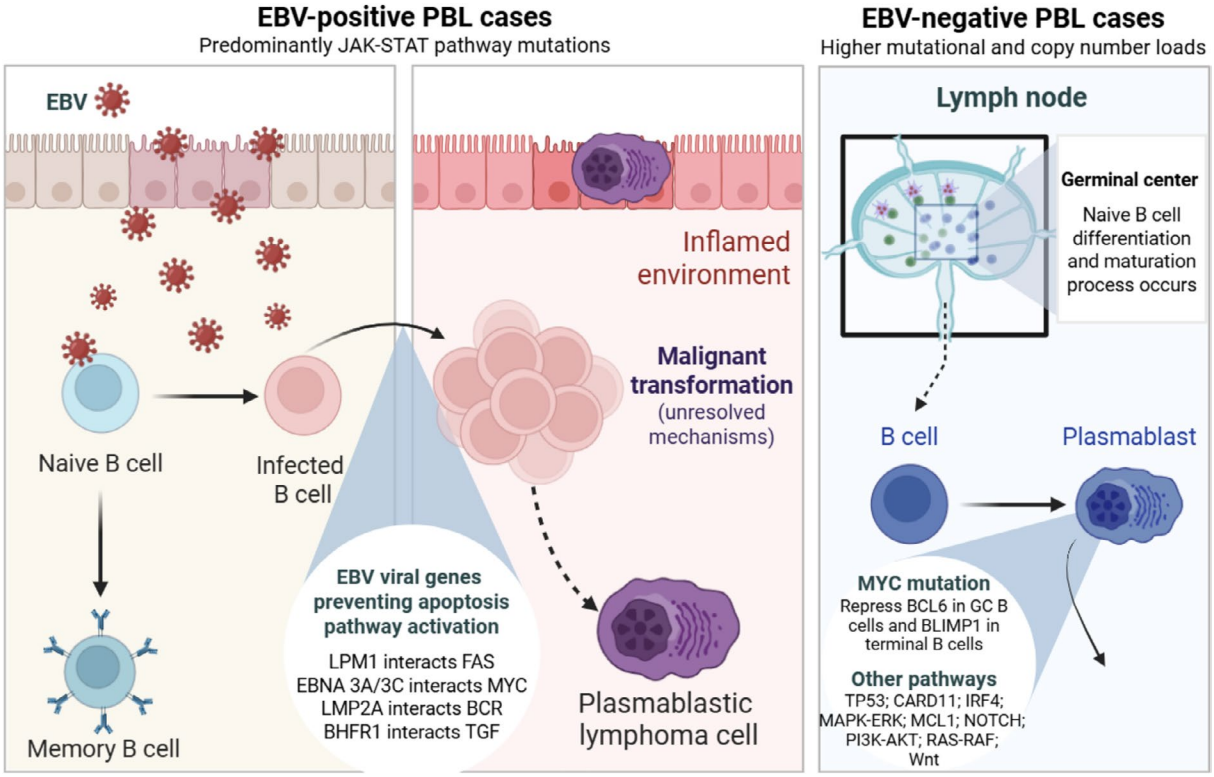


FIGURE 1 | Proposed pathophysiology of plasmablastic lymphoma.

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expression of EBV-encoded RNA (EBER) or EBV LMP-1 is detected in 66% of the cases [6, 24]. Molecular testing revealed that two-thirds of cases have detectable *MYC* rearrangements [21, 25]. Comparative genomic hybridization showed that PBL is closely related to DLCBL [26].

The differential diagnosis of PBL includes plasmablastic myeloma (PBM), EBV+ DLBCL, ALK+ DLBCL, primary effusion lymphoma (PEL), and HHV8+ large B cell lymphoma (LBCL). PBM and PBL are identical in morphology and immunophenotype [23]. Specifically, extramedullary PBM can pose a diagnostic challenge. However, an underlying HIV infection and the detection of EBER in neoplastic cells favor PBL, while the presence of monoclonal paraproteinemia, renal dysfunction, and skeletal lytic lesions favor PBM. EBV+ DLBCL is typically seen in older adults, although the expression of CD20 is universal [27]. PEL is strongly associated with HHV8 infection and clinically manifests as body cavity effusions [28]. Extracavitary variants of PEL are extremely rare and can pose a diagnostic challenge [29]. ALK+ DLBCL is an aggressive CD20-negative lymphoma and can have plasmablastic morphology [30]. However, the expression of ALK in ALK+ DLBCL is universal, and this entity is not typically associated with HIV infection or EBER expression. Also, cytogenetic anomalies involving the *ALK* gene are typical. Finally, HHV8+ LBCL is also an aggressive CD20-negative lymphoma seen in patients with underlying HIV infection, but it can be differentiated by underlying IgA- or IgG-*lambda* light chain restriction and a strong association with HHV8 infection and multicentric Castleman disease [31]. Features of PBL and differential diagnoses are summarized in Table 1.

Like other common aggressive lymphomas, staging should be conducted using positron emission tomography/computed tomography scanning. Clinical staging should follow the Lugano Modification of the Ann Arbor Staging System [32]. Additional diagnostic and staging tests are shown in Table 2.

3 | Risk Stratification and Prognosis

Historically, the prognosis of PBL has been poor with standard therapies. In a literature review of 112 HIV-positive cases, the median overall survival (OS) was 15 months, though this study included treated and untreated patients [24]. Another literature review of 70 HIV-positive PBL patients treated with standard chemotherapy showed a poor median OS of 14 months [33]. The median OS appeared shorter at 9 months in 76 patients with HIV-negative PBL [4].

A multicenter retrospective study of 50 HIV-positive PBL patients diagnosed and treated between 2000 and 2010 revealed a median OS of 11 months [21]. Similarly, a French study of 82 PBL patients diagnosed between 2008 and 2014 reported a 2-year event-free survival rate of 41% [34]. The AIDS Malignancy Consortium presented their experience on 12 patients with HIV-positive PBL and reported a 1-year OS rate of 67% [35]. The French LYSA group reported a large retrospective cohort of 135 patients with PBL diagnosed between 2000 and 2015 in France and observed a median OS of 32 months [36]. A multinational European study reported a 2-year OS rate of 61% in

TABLE 1 | Differential diagnosis of plasmablastic lymphoma.

	PBL	PEL	HHV8+ DLBCL	ALK+ DLBCL	EBV+ DLBCL	PBM
Association with immunosuppression	++	+++	+++	–	++	–
Age at diagnosis	< 60	< 60	< 60	< 60	> 60	> 60
Preferential site of involvement	Extranodal > nodal	Pericardial, pleural, peritoneal	Nodal > extranodal	Extranodal or nodal	Extranodal or nodal	Intramedullary
Cytologic features	Plasmablastic	Plasmablastic, immunoblastic or anaplastic	Plasmablastic	Centroblastic, immunoblastic or plasmablastic	Centroblastic, immunoblastic or plasmablastic	Plasmablastic
Immunophenotype	CD138+, MUM-1/IRF4+, CD20—	CD30+/−, LANA-1+, CD138+, CD20—	LANA-1+, cIgM lambda restriction, CD20+/−, CD138—	Non-GCB, CD20+, ALK+	Non-GCB, CD20+, CD30+	CD20-, CD138+, MUM1/IRF4+, CD56+
Association with EBV	++	+++	–	–	+++	–
Association with HHV-8	–	+++	+++	–	–	–
Association with HIV	++	+++	+++	–	+/–	–

Abbreviations: ALK, anaplastic lymphoma kinase; EBV, Epstein Barr virus; GCB, germinal center B-cell; HHV8, human herpesvirus 8; N/A, not available; PBL, plasmablastic lymphoma; PBM, plasmablastic myeloma; PEL, primary effusion lymphoma.

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TABLE 2 | Recommended workup for patients diagnosed with plasmablastic lymphoma.

Clinical test	Clinical implication
CBC with differential and CMP	Evaluate bone marrow, liver and kidney function
Performance status assessment	Prognostic marker
Serum LDH level	Prognostic marker
Uric acid level	Evaluate tumor lysis syndrome
SPEP and immunofixation	Exonerate myeloma
HIV antibody and viral load	Initiate or change antiretroviral therapy
HBV and HCV testing	Evaluate coinfections
Positron emission tomography/computed tomography	Staging and assessment of extranodal involvement
Bone marrow aspiration and biopsy	Staging and to exonerate myeloma
Brain MRI and lumbar puncture	If neurological symptoms, to evaluate CNS involvement
Echocardiogram	Evaluate cardiac function before anthracycline exposure
Fertility preservation	Sperm banking, oocyte cryopreservation, etc.

Abbreviations: CBC, complete blood count; CMP, comprehensive metabolic panel; CNS, central nervous system; HBV, hepatitis B virus; HCV, hepatitis C virus; LDH, lactate dehydrogenase; MRI, magnetic resonance imaging; SPEP, serum protein electrophoresis.

49 PBL patients with available clinical data, though the inclusion period was not reported [14]. A study of 42 patients with PBL diagnosed and treated in British Columbia between 1997 and 2019 reported a median OS of 27 months [37]. A multicenter retrospective Chinese study evaluated 102 PBL patients treated between 2008 and 2019 and reported a 2-year OS rate of 65% [38]. A large retrospective cohort of 281 patients from Australia, Canada, Singapore, and the United Kingdom diagnosed with PBL between 1999 and 2020 reported a median OS of 1.7 years and a 5-year OS rate of 36% [39]. A recent United States-based multicenter study of 344 PBL patients (not yet peer-reviewed), of whom 48% were HIV-positive, reported improved outcomes with median OS of 5 years, with superior OS in patients living with HIV [40]. Use of higher intensity regimens or proteasome inhibitors did not show a survival benefit.

Additional insights on the prognosis of PBL come from population-based studies. In an NCDB-based study that included 481 PBL and 68 402 DLBCL patients, a diagnosis of PBL was associated with a 1.7-fold higher risk of dying from any cause than patients with DLBCL, NOS [3]. The median OS of PBL patients was 15 months, compared with 56 months in DLBCL patients, and 3-year OS rates of 40% and 59%, respectively. In a separate study using the Surveillance, Epidemiology, and End Results (SEER) database, 248 PBL diagnosed between 2010 and 2016 were identified and analyzed, revealing a 3-year OS rate of 54% [41].

3.1 | Prognostic Factors

Given the rarity of the disease, most of the data on prognostic factors arise from retrospective case series. For this review, we pooled data from published studies evaluating clinical and pathological variables. This analysis is purely descriptive and aims to be hypothesis-generating.

Seven studies evaluated age ≥ 60 versus < 60 years [36–39, 41, 42]. The pooled effect showed that age ≥ 60 was associated with worse OS (hazard ratio [HR] 1.44; 95% confidence interval [CI] 1.12–1.86). One study used a cutoff of 40 years in patients with HIV-positive PBL and found no association with survival [21].

Nine studies evaluated advanced versus early stage [21, 36–39, 41–44]. The pooled effect showed worse OS for patients with advanced stage (HR 2.66; 95% CI 2.20–3.23). Two additional studies reported a significant association between advanced stage and worse survival, but no effect sizes were reported [3, 45].

Four studies reported the impact of the IPI score on survival in PBL [21, 37, 39, 44]. The pooled effect size showed worse OS in patients with high/high-intermediate IPI scores (HR 3.23; 95% CI 1.88–5.57). A multicenter retrospective study of 49 European patients with PBL reported a 2-year OS rate of 11% versus 80% in patients with high versus low IPI scores [14]. A German study of 78 PBL patients confirmed the prognostic value of the IPI score and reported that the NCCN-IPI score was a better fit than the IPI score [42]. These two studies did not report effect sizes; however.

Four studies reported effect sizes of HIV-positive versus HIV-negative status [21, 36, 37, 39]. The pooled effect size was HR 0.81 (95% CI 0.55–1.17). A SEER-based study reported 2-year OS rates of 50% versus 35% for patients with HIV-positive versus HIV-negative status ($p = 0.39$) [41]. Three studies reported no association between HIV status and survival, but no effect sizes were provided [3, 44, 45].

Five studies reported effect sizes on EBER expression on OS in PBL [36–39, 43]. The pooled effect size was HR 0.66 (95% CI 0.41–1.06). Two additional studies reported no impact of EBER expression on OS [44, 45]. Of note, the EBER positivity cutoff was not uniform between studies.

Four studies reported effect sizes of *MYC* gene rearrangements on OS in PBL [21, 37, 39, 44]. The pooled effect size was HR 1.93 (95% CI 0.87–4.30).

Based on the published literature, age ≥ 60, advanced stage, and high/high-intermediate IPI scores are associated with worse outcomes in PBL, while the prognostic role of HIV status, EBER positivity, and *MYC* gene rearrangements is unclear.

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4 | Current Management

The treatment approach in PBL depends on several factors, including the association with HIV infection, the location and extension of the tumor, and the goals of therapy. There is no standard approach for this type of lymphoma. However, combination chemotherapy is the most frequently used [6].

The treatments routinely offered are combination chemotherapy regimens used to treat aggressive subtypes [39]. The use of infusional EPOCH (etoposide, prednisone, vincristine, cyclophosphamide, and doxorubicin) is supported by a pooled analysis of 1546 patients with HIV-associated lymphomas, of whom 6% had PBL, from 19 prospective clinical trials to evaluate patients' outcomes [46]. Treatment with infusional EPOCH was associated with superior OS to CHOP in univariate and multivariate analyzes. A study by the AIDS Malignancy Consortium (AMC-075) prospectively evaluated the addition of the histone deacetylase inhibitor vorinostat to infusional EPOCH with or without rituximab in 90 patients with HIV-associated lymphomas, including 15 patients with PBL [47]. Although the addition of vorinostat was not associated with improved outcomes, the median OS of PBL patients was not yet reached 3 years after treatment initiation, further supporting using EPOCH in this context. The National Comprehensive Cancer Network (NCCN) recommends treating PBL with more intensive regimens than CHOP [48].

A novel approach to treat PBL was the addition of bortezomib to infusional EPOCH (V-EPOCH). In a retrospective case series, 16 patients received V-EPOCH with and without radiotherapy. Complete response (CR) was attained in 15 patients (94%) and partial response in one (6%). The median follow-up was 48 months (range 20–61), and the median OS was 62 months (95% CI 17-not reached) [49]. Several reports have shown encouraging responses and survival outcomes with bortezomib plus chemotherapy in HIV-positive and HIV-negative PBL patients in the frontline setting [50–55].

A study focused on 80 early-stage PBL patients from 13 US academic centers reported a 3-year OS rate of approximately 80%, with a trend towards better outcomes in patients treated with chemotherapy followed by consolidation radiation therapy [56]. There was no detectable difference in PFS and OS between Stage I and Stage II disease, or between HIV-positive and HIV-negative PBL patients.

Autologous stem cell transplantation (SCT) may be considered an option for first line treatment as well as for relapsed cases. The advent of highly antiretroviral therapy (ART) has made autologous SCT a viable option for HIV-positive patients. A review by Al-Malki and colleagues on 20 patients who underwent autologous SCT for PBL in first CR (CR1) or CR2 between 2001 and 2012 reported 1- and 3-year OS rates of 69% and 45%, respectively [57, 58]. A European registry study analyzing the outcomes of 24 PBL patients who underwent autologous SCT between 2001 and 2011 found that 67% received autologous SCT post-frontline therapy, with 50% achieving CR and 17% PR. The study noted a 2-year relapse rate of 30% and observed no relapses beyond 4 months from SCT. However, the follow-up period was relatively short at 30 months [58]. Meanwhile, the experience on allogeneic SCT in PBL is limited to case reports [59].

Although some case reports have reported spontaneous regression of PBL with ART in HIV-positive individuals or reducing immunosuppression in patients with autoimmune conditions [60, 61], most patients will require systemic antineoplastic therapy. ART alone or reduction of immunosuppression is not currently recommended as a standard treatment for PBL.

Based on the limited available data, we recommend using 6 cycles of infusional EPOCH with or without bortezomib for patients with advanced stages [62]. For patients with early-stage disease, we recommend four courses of CHOP or EPOCH with or without bortezomib followed by 30 Gy of consolidative radiation therapy. If feasible, autologous SCT can be considered in patients with high-risk disease in CR1. The high-risk disease includes HIV-negative, high IPI score, and the presence of *MYC* gene rearrangements. Response assessment should follow current recommendations for aggressive lymphomas [32]. ART should be added to all ART-naïve HIV-positive patients or switched to more effective combinations in HIV-positive patients with low CD4+ counts or high HIV viral loads. In the relapsed setting, we recommend regimens designed for aggressive lymphomas and consider incorporating bortezomib, daratumumab, or lenalidomide, followed by autologous SCT for chemosensitive disease with curative intent. A proposed management algorithm is shown in Figure 2.

Current data are limited to providing specific recommendations for frail patients, those with CNS involvement, and those with cardiac, renal, or hepatic dysfunction. These patients should be managed according to current NCCN guidelines for DLBCL, NOS, and the treating physician's clinical judgment.

5 | Future Directions

Daratumumab, an anti-CD38 monoclonal antibody, emerges as an innovative approach in patients with PBL. A retrospective study involving seven patients treated with daratumumab plus EPOCH reported a CR rate of 83% and a 2-year OS rate of 57% [63]. The combination of daratumumab and ICE (ifosfamide, carboplatin, and etoposide) was effective in four patients with relapsed PBL [64]. Three patients were infected with HIV, and all were previously exposed to V-EPOCH. Two underwent ASCT after responding to daratumumab-ICE. Three patients were alive at the time of the report. Daratumumab is likely to be used more frequently in the frontline and relapsed settings.

Other emerging treatment options are immunomodulators such as lenalidomide or pomalidomide. Several case reports supported using lenalidomide alone or in combination in patients with relapsed or refractory PBL. In the frontline setting, lenalidomide induced a CR when combined with CHOP or cyclophosphamide and dexamethasone [65, 66]. In the relapse setting, lenalidomide induced a response when combined with bortezomib [67, 68], daratumumab [69], or tislelizumab [70]. Lenalidomide monotherapy induced short-lived responses [71–73]. Only one case has been documented where pomalidomide was unsuccessfully used following the failure of pembrolizumab in treating PBL [74].

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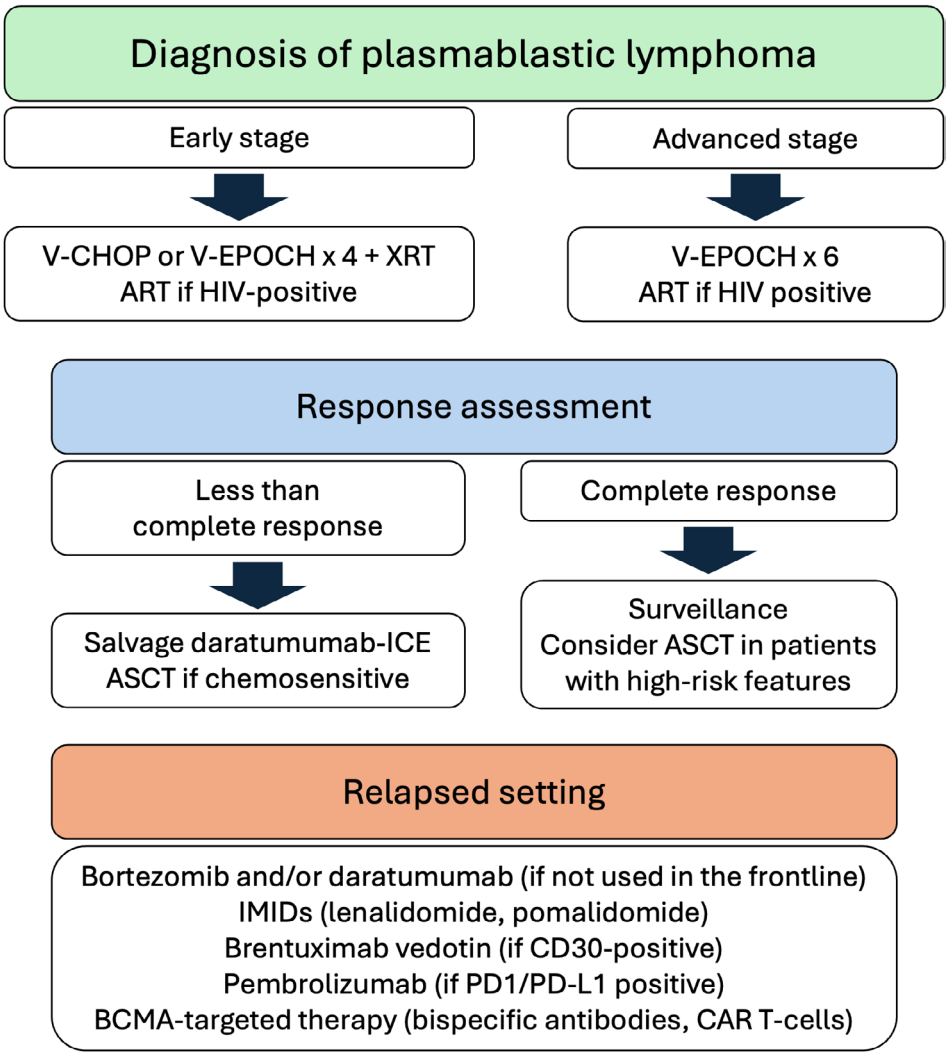


FIGURE 2 | Proposed treatment approach to plasmablastic lymphoma. ART: Antiretroviral therapy; ASCT: Autologous stem cell transplantation; BCMA: B-cell maturation antigen; CAR: Chimeric antigen receptor; ICE: Ifosfamide, carboplatin, and etoposide; IMIDs: Immunomodulators; V-EPOCH: Bortezomib, etoposide, prednisone, vincristine, cyclophosphamide, and doxorubicin.

TABLE 3 | Selected clinical trials actively enrolling patients with plasmablastic lymphoma.

Regimen	Phase	Line of therapy	Sponsor/location	Identifier
Daratumumab, EPOCH	I	First Line	AIDS Malignancy Consortium/USA	NCT04139304
Belantamab mafodotin	II	Relapsed or Refractory	Massachusetts General Hospital/USA	NCT04676360
Daratumumab, bortezomib, dexamethasone	II	Relapsed or Refractory	Fondazione Italiana Linfomi/Italy	NCT04915248
Pomalidomide, EPOCH, rituximab	I	First Line	National Cancer Institute/USA	NCT05389423
Daratumumab	I	Relapsed or Refractory	National Cancer Institute/USA	NCT05907759
Teclistamab	I	Relapsed or Refractory	National Cancer Institute/USA	NCT07332507

Source: ClinicalTrials.Gov (as of March 19, 2026).

Up to 30% of PBL cases express CD30. Brentuximab vedotin (BV) is an antibody-drug conjugate consisting of a chimeric anti-CD30 monoclonal antibody linked to MMAE. It is currently approved for treating classical Hodgkin lymphoma and CD30-expressing T-cell lymphoma. Only a few case reports reported BV being efficacious in relapsed PBL [75–77].

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PD-1 and PD-L1 are also overexpressed and biologically active in PBL [78, 79]. A single case reported a durable, complete response to pembrolizumab plus radiation therapy in an elderly patient with early-stage relapsed HIV-negative PBL [80]. Other cases using anti-PD-1 monoclonal antibodies in patients with relapsed PBL have been less successful [70, 74].

B-cell maturation antigen (BCMA) is highly expressed in normal and malignant plasma cells. In PBL, BCMA was expressed by 70% and 90% of the malignant cells, and the expression of BCMA was intermediate to strong in 60% of the cases [81]. Three patients with relapsed PBL were treated with the BCMAxCD3 antibody teclistamab [82]. One patient attained a durable CR after 4 cycles, one patient relapsed after 5 cycles, and one patient experienced disease progression on therapy. Two patients have been treated with anti-BCMA CAR T-cell therapy in the relapsed setting. One patient received ciltacabtagene autoleucel, which induced a sustained CR [83]. Another patient received an in-house anti-BCMA CAR T-cell product, which induced a CR within 30 days of exposure [84].

Patients with PBL should be considered for clinical trials at any stage of the disease. Table 3 shows a list of selected ongoing clinical trials.

6 | Conclusion

PBL remains a diagnostic and therapeutic challenge, with most patients experiencing short survival times with standard approaches. Adding novel agents, such as bortezomib or daratumumab, might be associated with improved outcomes. BCMA-targeted bispecific antibodies and CAR T-cells might provide an important treatment option in the relapsed setting. However, multicenter prospective studies are needed to confirm these observations. The inclusion of PBL patients in clinical trials is of utmost importance.

Author Contributions

Jorge J. Castillo wrote the initial draft. All the authors critically reviewed and approved the final version of the manuscript.

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Ethics Statement

The authors have nothing to report.

Consent

The authors have nothing to report.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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