Adult Post-Transplant Lymphoproliferative Disorder

Subjects: Hematology

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Post-transplant lymphoproliferative disorder (PTLD) is a rare but severe complication of hematopoietic or solid organ transplant recipients, with variable incidence and timing of occurrence depending on different patient-, therapy-, and transplant-related factors. The pathogenesis of PTLD is complex, with most cases of early PLTD having a strong association with Epstein–Barr virus (EBV) infection and the iatrogenic, immunosuppression-related decrease in T-cell immune surveillance. Without appropriate T-cell response, EBV-infected B cells persist and proliferate, resulting in malignant transformation. Classification is based on the histologic subtype and ranges from nondestructive hyperplasias to monoclonal aggressive lymphomas, with the most common subtype being diffuse large B-cell lymphoma-like PTLD. Management focuses on prevention of PTLD development, as well as therapy for active disease. Treatment is largely based on the histologic subtype.

post-transplant lymphoproliferative disorder
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1. Introduction

Post-transplant lymphoproliferative disorder (PTLD) is the most common malignancy in recipients of solid organ transplantation (SOT), excluding nonmelanoma cutaneous and in situ cervical malignancies [1]. In contrast, PTLD occurs in a minority of patients following allogeneic hematopoietic stem-cell transplantation (alloHSCT) [2][3]. Regardless of the setting, PTLD has been associated with significant morbidity and mortality. PTLD mainly arises from B cells but may rarely develop from T or NK cells, which account for 2–15% of all PTLD cases. More than half of B-cell PTLDs are driven by abnormal expansion of Epstein–Barr virus (EBV)-infected B cells. Non-B-cell PTLDs are usually not EBV-related, occur after a long latency period post-transplantation, and are associated with a more aggressive course and poor prognosis, with a median overall survival (OS) of ~6 months [4]. Higher PTLD rates have been observed in multiorgan and intestinal (<20%), lung (3–10%), and heart (2–8%) transplants due to the higher level and prolonged need of immunosuppressive therapy (IST) to prevent allograft rejection [1][5][6].

The primary management of PTLD includes reduction or complete cessation of IST. However, this is not always possible considering the risk for allograft loss or dysfunction, especially in cases of vital organ transplantation, such as heart transplant. In this context, a variety of therapeutic options have been proposed including chemo- and immunotherapy, radiation therapy, and various novel treatments, all with variable results [7]. While there are no guidelines for treatment, the type of lesion and EBV status generally drives the type of therapy based on consensus statements [8][9][10].

2. Pathogenesis

The pathogenesis of PTLD is complex and not fully understood. The dominant theory is that the required post-transplant IST negatively impacts the ability of cytotoxic T cells for immune eradication via multiple mechanisms, including inability to produce vital cytokines for immune destruction, such as interferon gamma (IFN-γ), interleukin 2 (IL-2), and tumor necrosis factor-alpha (TNF-α). This dysfunction allows abnormal clones of B lymphocytes (most commonly infected with EBV) to proliferate, ultimately transforming to PTLD.

In most instances, PLTD has been strongly associated with EBV infection [11][12]. EBV can be transmitted from a seropositive donor to a previously EBV-naïve/seronegative recipient and manifest as a primary infection, but PTLD can also be a result of EBV reactivation in a seropositive recipient who had previously acquired EBV via environmental exposure, in the setting of immunosuppression [13]. The virus remains latent in B lymphocytes or progresses with viral replication and B-cell lysis. Primary EBV infections in immunocompetent adults are usually self-limited and do not result in any significant complications because proliferation of B cells is normally suppressed by a virus-specific cytotoxic T-lymphocyte response, eradicating the majority of phenotypically abnormal B cells infected with EBV [13]. Disruption in T-cell surveillance, in the setting of post-transplant iatrogenic immunosuppression, can lead to unchecked B-cell proliferation and transformation, hence developing into PTLD [13].

Less is known about the pathogenesis of EBV-negative PTLD. It is believed to be similar to that of de novo non-Hodgkin's lymphoma (NHL) in immunocompetent hosts based on available molecular and immunohistochemical data, rather than true PTLD [14][15]. Hit-and-run EBV infections, indicating an EBV infection that initiates the pathogenesis of PTLD and then resolves, infections with other herpesviruses (e.g., cytomegalovirus (CMV), human herpesvirus 6 (HHV-6) or other viruses), persistent antigen stimulation by the graft, and long-term IST, have all been proposed as potential mechanisms [13][16]. From a clinical perspective, EBV-negative PTLD tends to occur in older transplant recipients, has a longer latency after transplant, is typically associated with SOT, and tends to have high-risk cytogenetic features [17].

EBV-positive PTLD has a different molecular profile and tumor microenvironment changes compared to EBV-negative PTLD [18][19][20][21]. Commonly encountered chromosomal aberrations are gains of 7q, 11q24–q25, and del(4)(q25–q35); combinations of the aberrations tend to occur more frequently in PTLD-negative cases, as they typically harbor more complex cytogenetics compared to EBV-positive PTLD [12]. A distinctive chromosomal abnormality in EBV-positive PTLD is the gain of 9p21, which causes changes in cyclin-dependent kinase inhibitor 2A (*CDKN2A*) expression and thus changes in cell cycle regulation [12]. Gain/amplification of 9p21 has also been found to lead to upregulation of the immune checkpoint receptor ligand, programmed cell death 1 ligand 2 (PD-L2), in EBV-positive cases that is believed to help escape immune surveillance [15]. In contrast, gain of 3/3q encoding the transcription factor Forkhead box p1 (*FOXP1*) is encountered in EBV-negative PTLD. This leads to enhanced expression of *FOXP1*, which appears to be critically related to the pathogenesis of EBV-negative PTLD. *TP53* mutations also appear to be more common in EBV-negative cases [12].

EBV has also been found to cause changes in the expression of *BCL6* and *MYC*, as well as activation of the NF-kB, PI3K/AKT/mTOR, and BCL2 molecular pathways, ultimately resulting in the malignant PTLD phenotype [22]. On an epigenetic level, EBV can alter the microRNA expression [23][24], which variably impacts gene expression in B lymphocytes, leading to their uncontrolled proliferation and transformation [22]. Furthermore, EBV has been reported to cause immune system dysregulation by downregulating the major histocompatibility complex class I and II expression, thus effectively escaping the immune system. Chronic EBV infection can upregulate the checkpoint inhibitors, such as programmed cell death protein 1 (PD-1) inhibitory receptors on the surface of T lymphocytes, leading to poor function of effector T cells, a phenomenon called T-cell exhaustion, which prevents optimal infection control [22]. Upregulation of the ligand of the PD-1 receptor (PD-L1) has also been observed in the setting of EBV infection, further promoting effector T-cell dysfunction and anergy against EBV [22]. Lastly, an association between PTLD and cytomegalovirus (CMV) was recently suggested [16].

It is clear that the biology of EBV-positive versus negative PTLD cases significantly differs from a genetic perspective [12][17]. EBV-positive cases have less frequent and less complex chromosomal molecular abnormalities, whereas EBV-negative cases can be associated with complex karyotypes, typically seen in cases of diffuse large B-cell lymphoma in immunocompetent patients. Genetic studies have also confirmed the resemblance of the latter two entities in a transcriptomic level [12].

Despite the clear differences in disease biology, a clear difference in prognosis or response to available therapies between EBV-positive and EBV-negative cases has not been established [25]. However, there have been reports of EBV negativity serving as an adverse prognostic factor. One study demonstrated that the median overall survival (OS) of EBV-negative PTLD patients was significantly lower compared to EBV-positive patients, 1 vs. 37 months, respectively [26][27]. In contrast, a large retrospective analysis from the CIBMRT showed no impact of EBV status on survival [28]. More studies are needed to better define the complex molecular basis of PTLD and create a more accurate classification system with better prognostic ability, as this will allow a more individualized approach to therapy.

3. Pathologic Classification

PTLD is divided into six pathologic subtypes based on the 2017 World Health Organization (WHO) classification (**Table 1**) [13][29]. Three subtypes are described as nondestructive PTLD and include plasmacytic hyperplasia, infectious mononucleosis-like PTLD, and florid follicular hyperplasia, whereas the three remaining subtypes are mentioned as destructive PTLD and include polymorphic, monomorphic, and classic Hodgkin lymphoma-like PTLD [13].

Table 1. Pathologic World Health Organization classification of PTLD.

Pathologic Subtype of PTLD	Location	LN Architecture	Latency from Transplant	Clonality	Further Histologic Subclassification
1. Non-destructive forms i. Plasmacytic hyperplasia ii. Infectious Mononucleosis-like PTLD iii. Florid follicular hyperplasia	Nodal	Preserved	Almost all cases early onset	Polyclonal	
2. Polymorphic PTLD	Nodal and Extranodal	Destructed	Typically early onset	Polyclonal	
3. Monomorphic PTLD	Nodal and Extranodal	Destructed	Both early and late onset	Monoclonal	Per WHO criteria for NHL (most common type is DLBCL)
4. Classic Hodgkin lymphoma like-PTLD	Nodal and Extranodal	Destructed	Both early and late onset	Monoclonal	Per WHO criteria for HL

Abbreviations: LN, lymph node; NHL, non-Hodgkin's lymphoma; DLBCL, diffuse large B-cell lymphoma; HL, Hodgkin's lymphoma.

The monomorphic subtype is the most common, accounting for 75% of all PTLD. Since it cannot be distinguished from sporadic B-cell non-Hodgkin's lymphomas from a pathologic aspect, it follows the sub-classification of non-Hodgkin's B-cell malignancies. Notably, the most common type of monomorphic PTLD is DLBCL-like PTLD [30][31]. Immunohistochemical and gene-expression analyses have shown that, for DLBCL subtype of PTLD, EBV-negative cases are usually of germinal center origin, whereas EBV-positive cases express a post-germinal center profile [23][32][33][34]. Follicular lymphomas and chronic lymphocytic leukemia (CLL) are not considered PTLD. Mantle cell lymphomas are also not considered PTLD in their vast majority; however, EBV-positive mucosa-associated lymphoid tissue (MALT) lymphoma has recently been included as a type of PTLD in the latest WHO classification [29].

Most histologic subtypes have a strong association with EBV, which is observed in almost all cases of nondestructive PTLD. The nondestructive forms are early-onset and less aggressive entities that present with milder symptoms. Poly- or monoclonal subtypes may also be EBV-driven. EBV-unrelated cases typically display monomorphic morphology [15][26][32][35]. The disease subtype appears to have prognostic value. Monomorphic T-cell PTLD is characterized by the lowest overall survival rates in comparison to all other subtypes. The Burkitt subtype of monomorphic B-cell PTLD also appears to be associated with poor OS [36].

4. Clinical Features

The incidence of PTLD is bimodal, with early- and late-onset subtypes. Typically, the incidence of PTLD is high within the first year of transplantation (early-onset) in both alloHSCT and SOT recipients [1]. However, given the recent advances in management leading to prolonged survival, late onset PTLD has also emerged after >5 years post-transplant, sometimes as late as >15–20 years post-transplant, a phenomenon most commonly seen in SOT recipients [13][37].

Early-onset PLTD is mainly associated with EBV (>90% of cases), whereas late onset PLTD can frequently be EBV-negative [11][12]. A recent study on liver transplantation patients, demonstrated 91% and 66% EBV-positivity in early- and late-onset PTLD cases, respectively [38]. Moreover, 88% of very early PTLD cases after SOT vs. 52% of late-onset PTLD cases post kidney transplantation were found to be EBV-positive [38]. Similar frequencies have been reported in other studies [39][40]. In addition, early PTLD usually presents with nondestructive or polymorphic histology, whereas late onset PLTD is usually monomorphic in nature [41].

Post-alloHSCT PTLD derives from the donor’s B lymphocytes is almost exclusively EBV-related, and occurs during the first year after transplantation, particularly within the first 2–6 months [42]. Late-onset PTLD post-alloHSCT is a very rare entity. In contrast, PTLD after SOT mainly arises from the recipient’s B lymphocytes and can also occur years after transplantation, with these cases typically being unrelated to EBV infection. Usually, any early onset PTLD tends to be EBV-driven and presents with non-destructive or polymorphic pathologic subtypes [1][13].

PTLD is characterized by a heterogeneous clinical presentation, ranging from asymptomatic to life-threatening, including spontaneous tumor lysis and organ failure. Symptoms may include fatigue, malaise, and mononucleosis-like symptoms, or even B-symptoms (fever, weight loss, night sweats, and lymphadenopathy). It often develops rapidly, requiring prompt diagnosis and treatment [1]. Some of the most common locations for disease development are the lymph nodes, tonsils, spleen, and bone marrow but, also, solid organs such as liver, lung, and kidney [7]. In comparison to lymphomas in immunocompetent hosts, it is more often associated with extranodal involvement [13].

Regarding prognosis, EBV status has not been correlated with survival to date. On the other hand, the histologic subtype seems to have an association with nondestructive early-onset forms and polymorphic PTLD having better survival. Early-onset PTLD also appears to have a better prognosis and response to reduction in immune suppressive treatment. Historically, higher mortality rates were reported in post-alloHSCT PTLD [43], compared to post-SOT PTLD [30][35][44]. However, most recently, this paradigm has shifted and outcomes have overall improved with the introduction of rituximab into clinical practice [45][46][47]. Notably, in PTLD where reduction in IST is not feasible, commonly in cases of cardiac transplant, prognosis remains poor [46][48].

5. Diagnosis and Staging

The diagnosis of PTLD is established through histopathological examination of tissue specimen obtained with surgical excisional (preferably), incisional or core-needle biopsy [49]. Immunohistochemical staining should be the standard for sporadic lymphomas, including testing for T and B-cell markers to establish the pathologic subtype of the PTLD based on the WHO criteria (**Table 1**). Fluorescence in situ hybridization (FISH) analysis of lymphoma should be performed to help with further subclassification once a subtype has been confirmed. Although not prospectively validated and not mandatory for diagnosis, an EBV-encoded RNA (EBER) in situ hybridization assay is recommended in all cases of PTLD, due to its association with EBV [50].

After histopathological confirmation, accurate staging is necessary. The same staging guidelines already established for lymphomas in immunocompetent hosts should also be used for PTLD [13]. 18F-fluorodeoxyglucose positron-emission tomography (FDG-PET) in combination with computed tomography (CT) have been suggested as highly sensitive for staging [51]. PET-CT is usually recommended as the initial diagnostic imaging modality. If not available, then CT of the chest, abdomen, and pelvis should be performed instead [49]. Based on disease burden shown in the initial imaging and clinical presentation, further diagnostic studies should be considered, such as magnetic resonance imaging (MRI) or CT of the brain/orbits/sinuses, lumbar puncture in case of neurological symptoms, and/or bone marrow biopsy [10].

6. Risk Factors

A significant risk factor for PTLD development, specific to SOT, is the use of multivisceral or intestinal grafts, since they contain an increased load of donor lymphoid tissue, which is at risk for expansion upon EBV infection during an immunocompromised state. On the other hand, kidney grafts have the lowest risk for PTLD [22]. The risk of PTLD is significantly higher among EBV-naïve SOT recipients who acquire primary EBV infection after transplant in the context of IST, which impairs their initial T-cell response and allows unrestricted viral replication. Given that >90% of organ donors have been infected with EBV, most naïve recipients will receive an EBV-infected allograft and will subsequently develop primary EBV infection [52]. Approximately 10% of these patients will eventually develop PTLD, which poses a 10-fold increased risk compared to patients who were seropositive prior to SOT [53][54][55]. Additional risk factors are described in **Table 2** [48][56][57][58][59][60][61][62][63][64][65].

Table 2. Risk factors for PTLD.

Post-SOT	Post-alloHSCT
Strong Evidence:	**Strong Evidence:**
1. Type of Graft:	1. High degree of HLA mismatch
Intestinal > Lung > Heart > others	HLA-mismatched or unrelated donor
Multivisceral grafts or graft from deceased donors	Haploidentical donor
2. EBV Seronegative/naive EBV recipient pre-SOT	Umbilical cord blood graft use
3. High intensity IST	2. Type of conditioning regimen
4. Anti-thymocyte globulin use as part of induction IST	T-cell-depleting strategies (in vivo and ex vivo)
Weak Evidence:	Anti-thymocyte globulin use
a. Non-white ethnicity	Non-myeloablative conditioning regimens
b. Young recipient and old donor age	3. Recipient old age > 50 years
c. Non-EBV infection	**Weak Evidence:**
d. Recipient HLA-A26 and B38 status	a. Acute GVHD
	b. History of splenectomy

Post-SOT	Post-alloHSCT
	c. Diagnosis of Aplastic Anemia
	d. Non-EBV infection

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threefold that of patients with concordantly positive serologies [66]. Future studies are needed to further investigate those results.

With respect to alloHSCT, the degree of matching between the donor and the recipient, as well as the type of conditioning regiment play a significant role in the development of PTLD. The higher the mismatch (for example, in cases of haploidentical or mismatched unrelated transplants), the higher the need for selective T-cell depletion protocols and IST, hence the higher the risk of PTLD development [67]. The use of reduced-intensity conditioning or anti-thymocyte globulin (ATG) as part of the conditioning regimen are strong contributors to the development of PTLD, the latter in a dose-dependent manner [42][68]. Other risk factors are mentioned in **Table 2** [67][69][70][71][72][73].

7. Surveillance and Prevention

A high or rapidly increasing viral load is associated with an increased risk of PTLD [74][75]. Despite the lack of guidelines recommending pre-emptive serial monitoring of EBV viral load early post-transplant, many centers have adopted this strategy in an effort to promptly identify patients at high risk for PTLD requiring early intervention. Monitoring of EBV DNA viral load is usually performed by quantitative polymerase-chain reaction (PCR).

At present, surveillance is strongly encouraged for the patients with pre- and peri-transplant high-risk factors and/or those who have undergone alloHSCT. The Sixth European Conference on Infections in Leukemia (ECIL-6) consensus recommends starting monitoring EBV levels within the first month after alloHSCT and continue for at least 4 months after, with a weekly frequency at least up until reconstitution of cellular immunity [76]. There is no data to support a preference for whole blood, plasma, or serum; all are acceptable specimens. Despite these recommendations, there are no official guidelines regarding when surveillance should be initiated, who are defined as high-risk patients, the frequency of surveillance, or the threshold to start pre-emptive therapy [22].

Appropriate prevention can help avoid development of PTLD, especially in individuals with high-risk features. In these patients, necessary measures should be carefully implemented to minimize all the potential modifiable risk factors. A major approach is limiting the exposure to aggressive post-transplant IST and tapering of IST post-transplant to the lowest possible level to avoid organ rejection. This is particularly important in patients with increasing levels in circulating EBV DNA.

In patients undergoing alloHSCT, selection of the best-matched donor and optimal conditioning regimen is important. Incorporation of rituximab in the conditioning regimen or induction immunosuppression for alloHSCT or

SOT, respectively, appears to be protective. Avoidance of ATG or specific T-cell depletion protocols can also decrease the risk [77]. When possible, EBV-naïve recipients should avoid EBV-seropositive allografts, as they pose a high-risk situation for development of PTLD [78].

Most recently, vaccine therapy has been considered for patients at risk for PTLD before organ transplantation. A phase I trial (NCT00278200) is currently studying the administration of a photochemically-treated autologous EBV-transformed B lymphoblastoid cell vaccine in EBV-positive or -negative patients who are being considered for SOT and are at high risk for PTLD. The primary objectives are to determine its efficacy in achieving an EBV-specific T-cell and antibody response and whether this is able to prevent primary EBV infection in EBV-negative patients [22].

8. Pre-Emptive Therapy

With the widespread incorporation of serial EBV-DNA monitoring, the initiation of pre-emptive therapy at the time of viral reactivation has become a common practice [79][80]. Most institutions reduce IST as a first step, when feasible and safe from an organ rejection perspective; however, there is not an official consensus. In cases where IST reduction is inadequate or not feasible as an initial approach, therapy with rituximab is pursued. There are no guidelines regarding the EBV threshold that should trigger pre-emptive treatment. This is partially due to variability and lack of standardization in source of samples (whole blood, plasma, and serum) and available assays.

Use of rituximab in high-risk patients is highly effective in preventing PTLD in post-alloHSCT patients with rising EBV load [81][82][83][84]. In post-SOT setting, findings are similar; decreased PTLD rates were reported with the use of rituximab post-heart or kidney transplants associated with uncontrolled EBV viremia that did not respond to IST reduction. These findings are attributed to the CD20+ B-cell depletion caused by rituximab, since these cells are the largest reservoir for latent EBV. The reduction in these cells lowers the risk for their malignant transformation and, thus, PTLD [22].

Rituximab is currently recommended on a weekly basis for 1–4 doses, and this approach is estimated to decrease the incidence of PLTD in >90% of cases [76]. Less popular approaches include the administration of EBV-specific T lymphocytes from the donor or another human; however, this is a time-consuming process and difficult to apply in daily clinical practice. Other approaches, such as the use of antivirals, have not been proven to be effective [22]. Importantly, all the aforementioned approaches should only be considered for high-risk patients; however, there is no consensus defining “high risk”.

9. Treatment

Treatment of PTLD can be heterogeneous, based on the disease subtype, transplant type, and patient characteristics [79]. Notably, common therapeutic strategies for PTLD differ from those used in lymphoproliferative disorders of immunocompetent patients. Most frequently used modalities include IST reduction, rituximab monotherapy or in combination with chemotherapy, and, less frequently, use of other novel immunotherapies, or autologous stem-cell transplantation. Surgery or local radiation can be options for localized disease [13] (**Figure 1**).

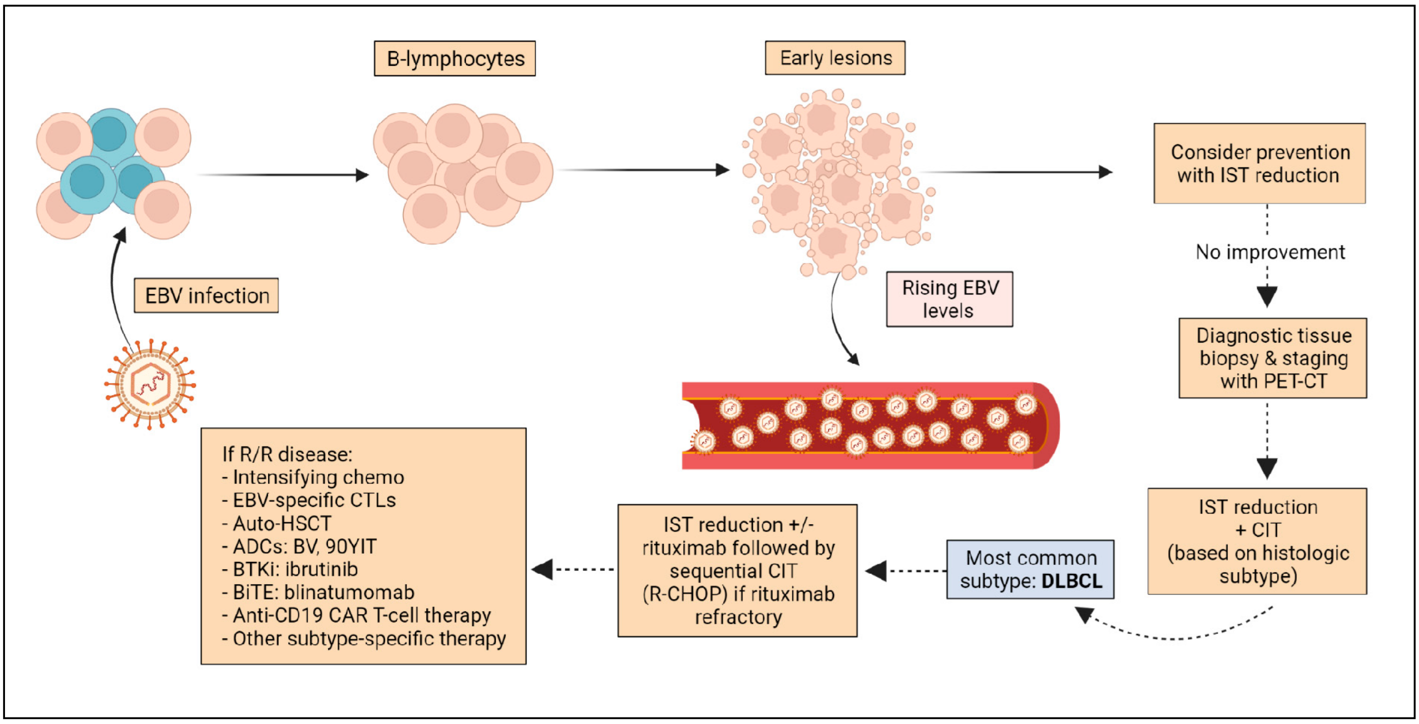

Figure 1. Pathogenesis and treatment algorithm of PLTD. Abbreviations: IST, immunosuppressive therapy; CIT, chemoimmunotherapy; DLBCL, diffuse large B-cell lymphoma; R/R, relapsed/refractory, CTLs, cytotoxic T-lymphocytes; HSCT, hematopoietic stem cell transplant; ADCs, Antibody Drug Conjugates; BV, Brentuximab Vedotin; 90YIT, 90 Yttrium ibritumomab tiuxetan; BTKi, Bruton Tyrosine Kinase Inhibitors; BiTE, Bispecific T cell Engagers; CAR, Chimeric Antigen Receptor.

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