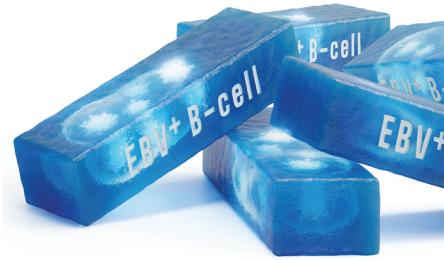
DON'T WAIT FOR EBV+ PTLD TO BECOME UNCONTROLLABLE[1,2]



Delayed intervention can lead to devastating outcomes[1,2]

EBV+ PTLD is rare and rapidly progressive, with patient prognosis bleak after first-line treatment failure[1,2]

In patients with R/R EBV+ PTLD (n=81)

POST-HCT Median OS **3** **weeks**

(95% CI: 1.3-4.3)[1]

In patients with R/R EBV+ PTLD (n=86)

POST-SOT Median OS **4.1** **months**

(95% CI: 1.9-8.5)[2]

Post-transplantation, there is an increased risk of EBV+ PTLD in SOT and HCT patients from immunosuppression[3]

- PTLD occurs in up to **9.1% of patients post-SOT** and **1.1-1.7% of patients post-allogeneic HCT**[4-11*]
- **~50% and ~100% of PTLD cases are EBV-associated** in SOT and HCT, respectively[1,12,13]
- Onset occurs **within 1 year in ~50% of patients post-SOT**; and **<1 year post-HCT**[14,15]

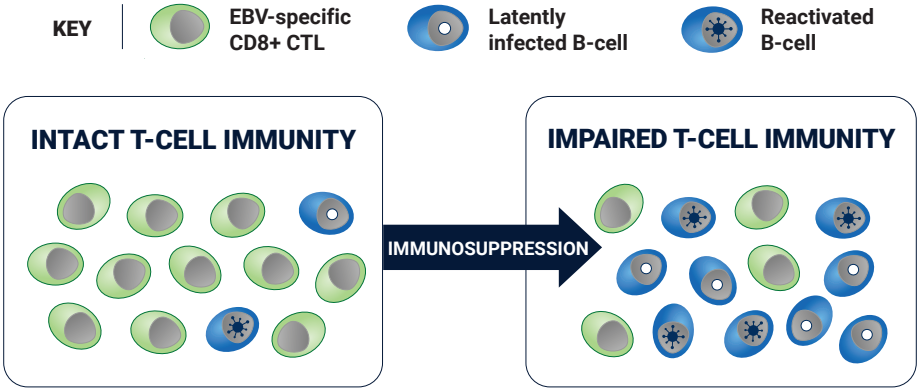
Ongoing assessment for risk of EBV+ PTLD in EVERY transplant patient is critical[1,2,12]

EBV+ PTLD = Epstein-Barr virus positive post-transplant lymphoproliferative disease; R/R = relapsed/refractory; OS = overall survival; SOT = solid organ transplant; HCT = hematopoietic cell transplantation.

*PTLD 5-year cumulative incidence by organ: kidney 0.6%, liver 1%, heart 0.9%, lung 1.7%, pancreas 2.5%, intestine 9.1%.[4-9]

Impaired T-cell immunity plays a significant role in disease development[16,17]

Immunosuppression post-transplantation alters T-cell immunity that keeps EBV infection in check[16,17]



With intact T-cell immunity, EBV-specific cytotoxic T lymphocytes **(CD8+ CTLs) keep EBV infection under control** by killing infected B-cells[16,17]

With impaired T-cell immunity, CD8+ CTLs are impeded, leaving **EBV free to reactivate and rapidly proliferate**, causing a range of malignancies, such as PTLD[16,17]

In EBV+ PTLD, impaired T-cell immunity turns control into chaos[16,17]

Learn more at EBVPTLD.com

EBV+ PTLD = Epstein-Barr virus positive post-transplant lymphoproliferative disease.

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