

Serostatus-Driven Epstein–Barr Virus Management After Kidney Transplantation: Screening, Surveillance, and Pre-emptive Prevention of PTLD

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Abstract

Epstein–Barr virus (EBV) is a principal driver of post-transplant lymphoproliferative disorder (PTLD), one of the most serious complications of kidney transplantation. Because the risk of EBV-related disease is determined primarily by donor–recipient EBV serostatus, post-transplant EBV management is increasingly conceptualised as

serostatus-driven. This narrative review synthesises evidence published predominantly within the last five years on EBV screening, post-transplant surveillance, interpretation of EBV DNA-emia, and pre-emptive prevention of PTLD in kidney transplant recipients, complemented by selected solid organ, paediatric, haematology, and laboratory-methodology studies. We argue that pre-transplant donor–recipient serology should define the baseline risk tier—high-risk donor-positive/recipient-negative (D+/R–), intermediate D–/R–, and lower-risk seropositive recipients—and thereby govern the intensity of monitoring, whereas viral-load kinetics, specimen type, assay calibration, immunosuppression exposure, and clinical context should govern intervention. Practical messages include the superiority of trend-based over single-threshold interpretation of EBV DNA-emia, the need for assay and specimen consistency in longitudinal follow-up, the limited role of antiviral prophylaxis, and the selective, evidence-informed use of immunosuppression reduction and pre-emptive rituximab in high-risk recipients. A six-step serostatus-driven algorithm is proposed to standardise screening, risk-adapted surveillance, interpretation of DNA-emia, pre-emptive intervention, and escalation to PTLD evaluation, while avoiding unnecessary testing or excessive immunosuppression reduction in lower-risk patients. Priorities for future research include international standardisation of EBV PCR, integration of EBV-specific immune monitoring into risk prediction, and prospective trials defining which recipients benefit from pre-emptive immune-directed therapy.

Keywords: Epstein–Barr virus; kidney transplantation; post-transplant lymphoproliferative disorder; serostatus; EBV DNA-emia; viral load monitoring; rituximab; pre-emptive therapy.

Introduction

Kidney transplantation requires long-term immunosuppression, which improves graft survival but increases susceptibility to viral reactivation, opportunistic infections, and virus-driven malignancies. Epstein–Barr virus (EBV) is particularly relevant in this context because it establishes lifelong latency in B lymphocytes and, under impaired T-cell immune surveillance, may progress from asymptomatic DNA-emia to post-transplant lymphoproliferative disorder (PTLD). PTLT remains one of the most severe complications after kidney transplantation, with potential consequences including graft dysfunction, allograft loss, treatment-related morbidity, and death. Contemporary reviews emphasize that PTLT is biologically heterogeneous, ranging from EBV-positive early lesions to EBV-negative late lymphoid malignancies, and that management often requires a stepwise multidisciplinary approach involving nephrology, infectious diseases, hematology, oncology, pathology, and transplant surgery [1–4].

A central principle emerging from recent evidence is that EBV management after kidney transplantation should be serostatus-driven. Pre-transplant donor-recipient EBV serology identifies recipients at the highest risk for primary EBV infection after transplantation, especially donor-positive/recipient-negative mismatch (D+/R-). This group lacks pre-existing EBV-specific immune memory and is therefore particularly vulnerable to donor-derived EBV acquisition, early EBV DNA-emia, and PTLD. Large contemporary kidney transplant cohorts and registry-based analyses consistently show that donor-recipient EBV serostatus is strongly associated with post-transplant EBV-related outcomes, making serostatus the practical foundation for surveillance intensity and pre-emptive decision-making [5,6].

Serostatus alone, however, does not fully define risk. The intensity and type of immunosuppression modify EBV-related outcomes, especially in EBV-mismatched recipients. Lymphocyte-depleting induction therapies such as antithymocyte globulin and alemtuzumab may increase PTLD risk compared with non-depleting induction strategies, likely by weakening early EBV-specific T-cell control. Recipient age, pediatric status, rejection treatment, maintenance immunosuppression intensity, and viral co-infections also influence the transition from transient EBV DNA-emia to clinically significant disease. Thus, EBV serostatus should be viewed as the baseline risk determinant onto which dynamic clinical and immunological variables are added [7–9].

This review summarizes recent evidence on EBV in kidney transplant recipients and proposes a practical serostatus-driven framework for screening, surveillance, interpretation of EBV DNA-emia, and pre-emptive prevention of PTLD. The central argument is that EBV donor-recipient serostatus should guide the intensity of monitoring, while EBV viral load kinetics, assay methodology, and clinical context should guide intervention decisions. This approach aims to prevent PTLD while avoiding unnecessary testing or excessive immunosuppression reduction in lower-risk recipients [1–9].

Scope and methodology of the review

This narrative review was prepared in accordance with the quality criteria of the Scale for the Assessment of Narrative Review Articles (SANRA). PubMed/MEDLINE, Scopus, and the Cochrane Library were searched from January 2014 to December 2025, with priority given to literature published within the last five years; older foundational studies were retained when they provided essential context for EBV PCR monitoring, PTLD diagnosis, or long-term kidney transplant outcomes. Search terms combined the concepts “Epstein-Barr virus,” “EBV DNAemia/viraemia,” “PCR/viral load monitoring,” “post-transplant lymphoproliferative disorder,” “serostatus,” and “kidney/renal transplantation,”

complemented by selected solid organ, paediatric transplant, haematology, and laboratory-methodology references identified through citation tracking. English-language original studies, cohort and registry analyses, reviews, and methodological evaluations relevant to kidney transplantation, donor–recipient serostatus, EBV viral load surveillance, and PTLT prevention were eligible; non-English reports and studies without extractable transplant-relevant data were not prioritised. Study selection and interpretation were performed by the authors by consensus [1–4,10].

Because EBV surveillance literature is heterogeneous with respect to study design, population, organ type, specimen type, PCR platform, viral load units, and intervention thresholds, this article does not attempt a formal meta-analysis. Instead, it uses a structured narrative synthesis, emphasizing clinically interpretable patterns across recent cohorts and reviews. Particular attention is given to evidence that can inform practical transplant protocols: who should be monitored, when monitoring should be intensified, how EBV DNA-emia should be interpreted, and when pre-emptive intervention should be considered [5,6,10–12].

Pre-transplant EBV serostatus and risk stratification

Pre-transplant EBV serological screening is the cornerstone of EBV risk stratification in kidney transplantation. Standard assessment generally includes EBV viral capsid antigen IgG and IgM and Epstein–Barr nuclear antigen IgG, allowing identification of prior infection versus EBV-naïve status. In clinical practice, the most important distinction is whether the recipient is EBV-seronegative before transplantation, because this defines susceptibility to primary EBV infection under immunosuppression. When donor and recipient serostatus are combined, recipients may be classified as high risk (D+/R–), intermediate risk (D–/R–), or lower risk when the recipient is EBV-seropositive regardless of donor status [5,6].

The D+/R– recipient represents the highest-risk phenotype. These patients may acquire EBV from the donor organ or early post-transplant exposure at a time when immunosuppression is strongest and EBV-specific immunity is absent. Recent cohort evidence shows that EBV-mismatched recipients have higher rates of EBV DNA-emia and PTLT than EBV-seropositive recipients. In a serostatus-driven management model, this group should therefore be identified before transplantation, flagged in the transplant record, and assigned an intensified post-transplant monitoring pathway [5,6,13].

Risk stratification should also incorporate clinical modifiers. EBV-mismatched adults receiving lymphocyte-depleting induction appear to be at higher PTLT risk than those receiving non-depleting induction, and pediatric kidney recipients are often EBV-seronegative at transplant and therefore disproportionately represented

among high-risk groups. Rejection episodes and intensified immunosuppression may further increase EBV replication risk. These modifiers do not replace serostatus, but they refine how aggressively a patient should be followed after transplantation [7,8,14].

TABLE 1. Serostatus-based EBV risk stratification and surveillance intensity in kidney transplant recipients.

Donor–recipient serostatus (risk tier)	EBV-specific immunity	Surveillance intensity	Pre-emptive considerations
D+/R– (high)	Absent (EBV-naïve)	Intensified, structured viral-load monitoring, especially months 0–6 and the first post-transplant year	Early stepwise immunosuppression reduction; consider pre-emptive rituximab if viral load rises or persists despite reduction
D–/R– (intermediate)	Absent (EBV-naïve)	Structured monitoring, generally less intensive than D+/R–	Immunosuppression reduction guided by viral-load kinetics
R+ (any donor) (lower)	Present (immune memory)	Selective, clinical-trigger-based testing rather than fixed high-frequency monitoring	Intervene only with persistent or high viral load or compatible clinical findings; avoid overreaction to isolated low-level DNA-emia

D, donor; R, recipient; DNA-emia, EBV DNA in blood. Clinical modifiers (lymphocyte-depleting induction, paediatric status, rejection treatment) refine surveillance within each tier [5–8,13,14].

Post-transplant EBV surveillance

EBV surveillance after kidney transplantation should be risk-adapted rather than uniform. High-risk D+/R– recipients should undergo structured EBV viral load monitoring, especially during the first post-transplant year and particularly during the early window when primary EBV infection is most likely. Evidence from EBV-mismatched solid organ transplant cohorts, including kidney recipients, suggests that donor-acquired EBV DNA-emia frequently emerges during the first months after transplantation, with a clinically important concentration of events between months 2 and 6. This supports intensified surveillance in this interval, followed by individualized monitoring according to prior viral load behavior and immunosuppression exposure [13].

In contrast, EBV-seropositive kidney transplant recipients may not benefit from the same high-frequency surveillance strategy. In these patients, low-level or intermittent EBV DNA-emia may reflect latent viral shedding rather than clinically meaningful progression toward PTLT. A more selective approach is therefore reasonable, with EBV PCR testing triggered by augmented immunosuppression, rejection treatment, unexplained cytopenias, lymphadenopathy, persistent fever, graft dysfunction, or clinical suspicion for PTLT. This approach avoids

overinterpretation of isolated low-level results while maintaining vigilance when clinical context changes [1,12,15].

Laboratory methodology is central to EBV surveillance. Whole blood and plasma EBV PCR assays may produce substantially different viral load values because whole blood detects both cell-associated and circulating viral DNA, whereas plasma may better reflect active viral replication. Prospective evaluations of WHO-standardized EBV PCR and comparative studies of specimen type show that assay and sample selection influence viral load interpretation, making universal numeric thresholds difficult to apply across centers. For this reason, each transplant program should use consistent specimen type and assay methodology for longitudinal follow-up whenever possible [10,11,16].

The most clinically useful interpretation of EBV monitoring is trend-based rather than threshold-based. A single viral load value is often less informative than the pattern of change over time, especially whether EBV DNA-emia is rising rapidly, persistent, or increasing despite immunosuppression adjustment. This is particularly important in D+/R– recipients, where rising EBV viral load may reflect primary infection and higher PTLD risk. Conversely, stable low-level DNA-emia in a clinically well EBV-seropositive recipient may require observation rather than immediate intervention [10–12,16].

EBV DNA-emia: clinical interpretation and actionable findings

EBV DNA-emia is best understood as a spectrum rather than a diagnosis. It may represent transient viral replication, primary EBV infection, reactivation under immunosuppression, or early lymphoproliferation. Contemporary adult and pediatric solid organ transplant studies show that EBV DNA-emia is relatively common, but only a minority of patients progress to PTLD. This gap between viral detection and clinical disease explains why EBV PCR cannot be interpreted in isolation and must be integrated with serostatus, viral load kinetics, immunosuppression intensity, and symptoms [12,17,18].

There is no universally accepted EBV viral load cutoff for intervention. Differences in whole blood versus plasma testing, assay calibration, viral load units, transplant population, and baseline serostatus all limit comparability between studies. Even when WHO-standardized methods are used, clinically meaningful differences between specimen types remain. As a result, many experts favor center-specific thresholds combined with individualized assessment of viral kinetics, rather than a single universal cutoff applied to all kidney transplant recipients [10,11,16,19].

In D+/R– recipients, EBV DNA-emia is more likely to represent primary infection and carries greater concern when viral load rises, persists, or reaches high levels. Higher peak viral load and EBV viral load at the time of PTLD diagnosis have

been associated with worse outcomes in kidney transplant recipients with PTLD. Therefore, sustained or rapidly increasing DNA-emia in an EBV-mismatched recipient should prompt closer monitoring, evaluation of immunosuppression, and consideration of pre-emptive intervention [6,13,20].

In EBV-seropositive recipients, low-level DNA-emia is often less specific. Longitudinal data in adult renal transplant recipients suggest that EBV DNA-emia may occur beyond the early post-transplant period without necessarily predicting PTLD, especially when levels are low or transient. Overly aggressive responses to isolated low-level DNA-emia may increase rejection risk by prompting unnecessary immunosuppression reduction. In this group, intervention should be guided by persistent viral load increase, high peak viral load, compatible clinical findings, or additional risk modifiers rather than by detection alone [15,21].

EBV DNA-emia may also occur in complex clinical contexts involving other viral infections or immune perturbations. Studies of multiplex viral monitoring demonstrate that EBV, CMV, and BK virus replication may coexist in kidney transplant recipients, particularly during suspected rejection or intensified immunosuppression. In such cases, EBV viral load may reflect broader immune dysregulation rather than EBV-specific disease. Concurrent infection, graft dysfunction, and systemic inflammation should therefore be considered when interpreting EBV PCR results [9,22].

EBV DNA-emia and PTLD

PTLD represents a heterogeneous group of lymphoid proliferations occurring under post-transplant immunosuppression. EBV-positive PTLD is classically linked to impaired T-cell control of EBV-infected B cells, especially in EBV-naïve recipients who acquire primary infection after transplantation. Early PTLD is more often EBV-associated and may arise within the first year, while later PTLD can be EBV-negative and may follow different oncogenic mechanisms. This biological heterogeneity explains why EBV DNA-emia is a valuable risk marker but not a definitive diagnostic test [1–4].

Clinically, PTLD in kidney transplant recipients may present with lymphadenopathy, fever, weight loss, cytopenias, graft dysfunction, gastrointestinal symptoms, or extranodal disease. Case series and reports illustrate that gastrointestinal involvement can be severe and may present with bleeding or ischemic complications, underscoring the need for tissue diagnosis when clinical suspicion is high. Imaging, hematology consultation, and biopsy remain essential because EBV PCR alone cannot distinguish benign viral replication from malignant transformation [23,24].

Prognosis in PTLD depends on several factors, including age, disease stage, histological subtype, EBV association, extranodal involvement, and EBV viral

load at diagnosis. Kidney transplant cohorts suggest that higher EBV viral load at PTLT diagnosis and older age are associated with worse survival. Regional experiences also demonstrate variability in presentation and outcomes, reflecting differences in surveillance intensity, access to diagnostic pathways, and treatment strategies. These findings support earlier recognition and standardized algorithms for high-risk kidney transplant recipients [20,24,25].

Prevention and management strategies

The first-line response to clinically significant EBV DNA-emia is usually a reduction of immunosuppression. The goal is to restore EBV-specific immune surveillance while preserving graft function. Common approaches include reducing calcineurin inhibitor exposure and decreasing or temporarily withholding antimetabolite therapy. This strategy is biologically rational and widely recommended, but it requires careful balancing because excessive immunosuppression reduction can precipitate rejection, particularly in patients with recent rejection episodes or high immunological risk [1,4,9].

Rituximab has emerged as a potential pre-emptive or therapeutic option in selected high-risk patients. Retrospective solid organ transplant data suggest that rituximab exposure after EBV DNA-emia may reduce PTLT risk over time, although prospective randomized evidence remains limited. Pediatric kidney transplant series also suggests that pre-emptive rituximab may help clear EBV DNA-emia in EBV-naïve recipients who do not respond to immunosuppression reduction alone. Nevertheless, rituximab should not be applied routinely to all patients with EBV DNA-emia because B-cell depletion carries infection risk and may complicate immune recovery [8,26,27].

Antiviral therapy has a limited role in EBV prevention compared with CMV prevention because EBV often persists in latently infected B cells, where nucleoside analogues have limited effect. Current reviews do not support routine antiviral prophylaxis solely to prevent EBV-driven PTLT. More promising future approaches include immune-based monitoring and EBV-specific adoptive T-cell therapy, which has stronger precedent in hematopoietic stem cell transplantation but remains investigational in kidney and other solid organ transplant populations [2,28,29].

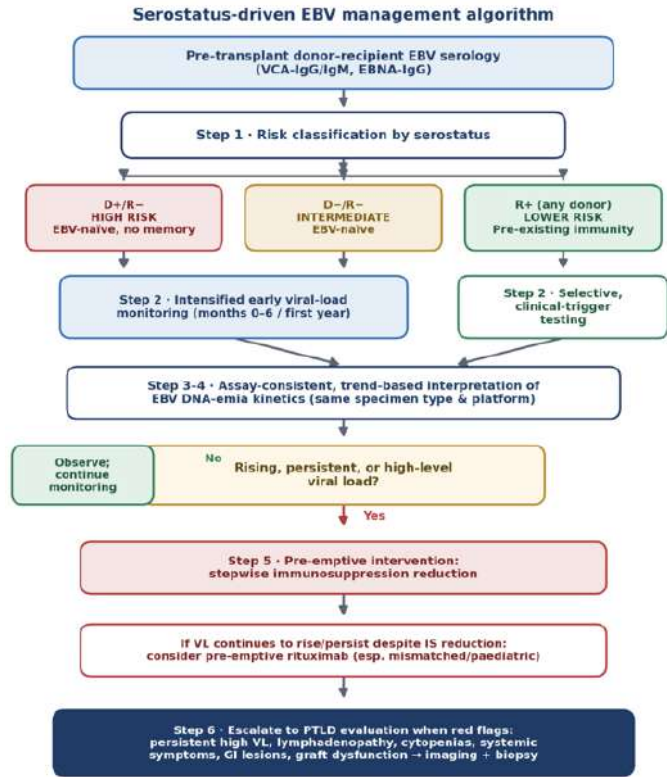
A practical serostatus-driven algorithm

A practical EBV management algorithm should begin before transplantation with donor–recipient EBV serology. Step 1 is classification into high-risk D+/R–, intermediate D–/R–, or lower-risk R+ status. Step 2 is assignment of surveillance intensity: D+/R– recipients should undergo intensified early EBV viral load

monitoring, while R+ recipients may be monitored selectively according to clinical triggers. Step 3 and 4 is assay-consistent interpretation of EBV DNA-emia kinetics, with emphasis on rising, persistent, or high-level viral load rather than isolated values [5,10,13].

Step 5 is pre-emptive intervention when EBV DNA-emia becomes clinically actionable. In high-risk recipients, this generally begins with stepwise immunosuppression reduction and close reassessment of viral load and graft function. Rituximab may be considered when viral load continues to rise or persists despite immunosuppression adjustment, especially in EBV-mismatched pediatric recipients or other high-risk scenarios. Step 6 is escalation to PTLD evaluation when red flags appear, including persistent high viral load, lymphadenopathy, unexplained cytopenias, systemic symptoms, gastrointestinal lesions, or graft dysfunction [8,20,26,27].

FIGURE 1. Serostatus-driven EBV management algorithm in kidney transplant recipients. Schematic overview of a risk-adapted surveillance and intervention pathway integrating donor–recipient EBV serostatus, EBV DNA-emia kinetics, and assay-consistent viral load monitoring. The algorithm emphasizes trend-based interpretation, stepwise immunosuppression modulation, and escalation to PTLD evaluation when clinically indicated [5,10,13].



VL, viral load; IS, immunosuppression; PTLD, post-transplant lymphoproliferative disorder; GI, gastrointestinal

Future directions

Future research should address three major gaps. First, EBV PCR monitoring requires better standardization across laboratories, including specimen type, reporting units, calibration, and clinically meaningful viral load kinetics. Second, risk prediction should move beyond viral load alone by integrating EBV-specific immune monitoring, recipient risk factors, immunosuppression exposure, and possibly viral or host biomarkers. Third, prospective studies are needed to define which patients benefit from pre-emptive rituximab or other immune-directed therapies, particularly among EBV-mismatched kidney transplant recipients [10,16,19,25].

Emerging contexts such as SARS-CoV-2 infection also highlight the dynamic nature of EBV immune control in transplant recipients. Reports of EBV reactivation after COVID-19 in kidney transplant recipients suggest that systemic immune perturbations may alter viral latency and surveillance needs. Regional studies from different healthcare settings further emphasize the need to adapt EBV protocols to local resources, laboratory capacity, and transplant population characteristics [25,30,31].

Conclusions

EBV remains a major determinant of PTLTD risk after kidney transplantation, but its management should not rely solely on viral load detection. The most practical framework is serostatus-driven: donor–recipient EBV serology defines baseline risk, while viral load kinetics, specimen type, immunosuppression exposure, and clinical context determine whether monitoring, observation, immunosuppression reduction, rituximab, or PTLTD evaluation is appropriate. This approach is particularly important for D+/R– recipients, who require intensified early surveillance and proactive prevention strategies [5–8,13].

A structured serostatus-driven algorithm can help standardize EBV management across transplant programs while reducing unnecessary intervention in lower-risk recipients. Until better biomarkers and prospective trial data become available, clinicians should prioritize risk-adapted surveillance, assay consistency, multidisciplinary interpretation, and timely escalation when clinical red flags emerge. In this way, EBV monitoring can be transformed from a purely laboratory-based practice into a clinically integrated strategy for PTLTD prevention and graft preservation [1–4,10,26].

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