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Response to Comment on: “Janus kinase inhibitors as an evolving treatment for lymphomas and immune-mediated complications”

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Contributions: AM and SV wrote this response.

Thank you for your thoughtful comments regarding our review, “Janus kinase inhibitors as an evolving treatment for lymphomas and immune-mediated complications.”¹ We agree that Epstein-Barr virus (EBV) reactivation and EBV-associated post-transplant lymphoproliferative disorder (PTLD) are important infectious complications to consider in patients receiving ruxolitinib for graft-versus-host disease (GVHD). The studies highlighted in your letter provide additional evidence that ruxolitinib may contribute to impaired antiviral immune surveillance in this highly immunocompromised population and support ongoing vigilance for EBV reactivation in clinical practice.

However, we believe these observations should be interpreted within their clinical context. The available data linking ruxolitinib to EBV reactivation and PTLD arise almost exclusively from patients with GVHD following allogeneic transplantation, a population already at substantial baseline risk for EBV reactivation due to multiple concurrent risk factors, including transplant-related immune dysfunction, T-cell depletion, ongoing immunosuppressive therapy, and prior corticosteroid exposure. In this setting, routine monitoring for EBV viremia is already incorporated into standard post-transplant care at many centers.

Our review discussed infectious toxicities associated with JAK inhibition as representative examples of impaired host defense rather than as an exhaustive catalog of opportunistic infections. Cytomegalovirus reactivation, *Pneumocystis jirovecii* pneumonia, and invasive fungal infections were cited as clinically relevant manifestations of this risk, but our intent was not to imply that these were the only infectious complications that merit surveillance. We therefore agree that EBV reactivation represents an additional consideration in the transplant/GVHD setting, while noting that the current evidence does not suggest a similar magnitude of risk among patients receiving JAK inhibitors for lymphoma or other non-transplant indications.

We do not believe the data presented necessitate a correction to the review, as the article did not intend to provide an exhaustive list of viral pathogens associated with JAK inhibition. Nevertheless, we appreciate the opportunity to emphasize that EBV reactivation and PTLD are important considerations in ruxolitinib-treated patients following allogeneic transplantation and should be monitored according to established transplant-specific surveillance practices.

References:

1. Jaing T-H, Wang Y-L. Epstein-Barr virus reactivation and post-transplant lymphoproliferative disease risk deserve a place in the JAK inhibitor toxicity profile. Comment on: "Janus kinase inhibitors as an evolving treatment for lymphomas and immune-mediated complications". Haematologica. [xxx](#)