DEAR EDITOR

I read with great interest the review article recently published in your most valuable journal titled “Reinfection of cytomegalovirus in renal transplantation” by Ishibashi et al.1. It emphasized that the incidence of acute rejection, as a CMV indirect effect, in the mismatched glycoprotein H (gH) antibody group was higher that observed in the matched and D+/R− groups. They noted that acute rejection may be the consequence of strong recipient-derived cytotoxic T lymphocyte responses against ongoing CMV activities that had escaped humoral responses. Lack of CMV specific memory T cells may contribute to the lower rate of acute rejection in D+/R− setting.

I agree that beside direct effects of CMV infection in renal transplant recipients, it has been associated to indirect effects such as the potential role in allograft rejection; however, the underlying pathogenic mechanisms remain largely unknown. It has been proposed that latent CMV infection is linked to immune senescence and vascular disease. In addition, the titers of anti-endothelial cell antibodies (AECAs) against endothelial cell lining the vasculature were significantly higher in recipients with vascular rejection, supporting a humorally mediated pathogenesis2. The occurrence of high levels of AECAs in relation to CMV infection has been also demonstrated in 80% of renal and heart and in more than 40% of liver transplant patients3. Thus, I recommend if the levels of AECAs are measured, the titres of AECAs in the mismatched gH antibody group could be as a predictive marker of subsequent risk of acute renal allograft vascular rejection.

REFERENCES