WE appreciate the comment regarding our work from Prof. Einollahi. We agree with his points about possibility of antibody-mediated rejection.

It is of interest that CMV can trigger the formation of a variety of auto-antibodies. Especially, anti-endothelial cell antibodies (AECAs) have been proposed to play a role in antibody-mediated rejections. Vascular rejection in renal transplant patients appeared to be related to CMV infection and AECAs positivity. Varani et al. investigated the time of appearance of auto-antibodies with respect to CMV antigenemia in liver transplant recipients. In this study, AECAs were detected in coincidence with or immediately after the CMV antigenemia peak in most cases and were observed for at least 15 days. It is speculated that the CMV-induced endothelial damage and the presence of circulating cytomegalic endothelial cells may be a potent antigenic stimulus that leads to the production of AECAs. CMV antigenemia is considered as a possible indirect marker of endothelial cell infection and can reflect the state of the endothelial barrier.

We reported the differences in the clinical outcomes between the recipients with mismatched and matched combinations of CMV glycoprotein H (gH) antibodies. In our study, most acute rejection was diagnosed within one month after transplantation (mean 25±32 days of rejection after transplantation), and initial positive antigenemia was detected thereafter. On the other hand, the mean weeks of the initial antigenemia detection was 7 weeks after transplantation and its range was varied from 1 to 20 weeks after transplantation. Although this might indicate that most acute rejection was diagnosed prior to AECAs production, association between auto-antibodies and acute rejection could not be ruled out. In addition to measurement of the AECAs titers and their comparison between gH match and mismatch recipients, longer follow up data would be interesting to evaluate the AECAs as a predictor of acute renal allograft vascular rejection.

REFERENCES

