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Cytomegalovirus glomerulopathy in renal allografts

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Implication for health policy/practice/research/medical education:

Renal allograft cytomegalovirus (CMV) disease can show discrete features of CMV glomerulopathy, without affectation of the renal tubules and interstitium. This means that absent urine cytology findings and no tubulointerstitial changes in a patient suspected to have CMV renal allograft disease must warrant an exhaustive inspection of the glomeruli for evidence of CMV disease, including the use of immunohistochemical methodologies.

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Sunna et al in a recent issue of this journal described a case of severe nephrotic syndrome caused by isolated renal allograft cytomegalovirus (CMV) glomerulopathy in a 63-year old African American man with end-stage renal disease secondary to hypertension after he received a deceased CMV D+/R- renal allograft (1). Induction therapy was with thymoglobulin, and he was on triple maintenance immunosuppression with mycophenolic acid (Myfortic), prednisone and tacrolimus (1). Several glomeruli showed viral cytopathic effect consistent with CMV infection and immunohistochemical staining for CMV showed occasional glomerular cells with positive label and was negative in tubules and in the interstitium (1).

We are happy to concur with the authors and we note that in 2002, in the *American Journal of Transplantation*, we had reported on two patients with CMV-induced glomerular vasculopathy post-transplantation (2). The first patient was a 46-year old Venezuelan male, following a simultaneous cadaveric kidney and pancreas transplantation, induction was with thymoglobulin, and he was on triple maintenance immunosuppression with mycophenolate mofetil (MMF), prednisone and tacrolimus (2). His allograft biopsy displayed enlarged glomerular endothelial cells in the tufts' capillary loops and immunoperoxidase indirect immunofluorescence

staining showed several infected cells inside the glomerular tufts (2). The second patient in our report was an 18-year old Caucasian man, following a cadaveric CMV D+/R- renal allograft. Induction therapy was with basiliximab, followed by triple maintenance immunosuppression with MMF, prednisone and tacrolimus (2). Transplant biopsy indicated significant cytomegalovirus -related glomerulitis and the glomerular tufts were closed by large cytopathic endothelial cells with inflammation (2). The virally infected glomerular endothelial cells were marked with CMV-specific immunoperoxidase staining (2).

As recalled in the article by Sunna et al, Richardson et al, around 40 years ago, had described a characteristic diffuse CMV glomerulopathy in five of fourteen (36%) kidney allograft recipients without tubulointerstitial changes (3). Nevertheless, five years later, Herrera et al, in a study of seven bone-marrow-transplant individuals who had died with detected herpesvirus infection, argued the reality of a specific limited cytomegalovirus glomerulopathy (4). Herrera et al had concluded that cytomegalovirus glomerulopathy possibly represented anti-endothelial type of rejection or partially resolved acute vascular rejection (4). Counter to the claims by Herrera et al, in 2002, we had argued that CMV renal allograft disease could indeed show discrete features of CMV glomerulopathy, without explicit involvement of the renal allograft tubules and the

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interstitium (2). For this reason, we posit therefore that absent urine cytology findings and no tubulointerstitial changes in a patient suspected to have CMV renal allograft disease must warrant an exhaustive inspection of the glomeruli for evidence of CMV disease, including the use of immunohistochemical methodologies (2).

Author's contribution

MACO is the single author of this paper.

Conflicts of interest

The author declared no competing interests.

Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the author.

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