Hepatitis C Virus in Renal Tissue of Patients with Glomerulonephritis

R.	Rocco	Misiani ^a
O.	Omar	Vicari ^a
P.	Piermario	Bellavita ^a
A.	Aurelio	Sonzogni ^a
M.	Maria Grazia	Marin ^b

^aDivision of Nephrology and Dialysis, Department of Immunohaematology, Institute of Pathology, Ospedali Riuniti, Bergamo, ^bDepartment of Biomedical Sciences, University of Brescia, School of Medicine, Brescia, Italy

Dr. Piermario Bellavita, Department of Immunohaematology, Ospedali Riuniti, Largo Barozzi 1, I-24128 Bergamo (Italy)

Dear Sir,

We have recently found the presence of hepatitis C virus (HCV) markers in sera and cryoprecipitates from most patients with cryoglobulinemic glomerulonephritis [1]. Moreover, the association between renal disease and HCV infection has also been reported in some cases of membranoproliferative glomerulonephritis without detectable serum cryoglobulins [2]. However, the causal role of HCV in producing histological lesions and clinical features of cryoglobulinemic and noncryoglobulinemic glomerulonephritides has not been demonstrated. Very recently, Doutrelepon et al. [3] described a patient with membranoproliferative glomerulonephritis associated with mixed cryoglobulinemia and HCV infection. The detection of HCV RNA by the polymerase chain reaction (PCR) in urine and renal tissue from this patient led the authors to infer that HCV was responsible for the glomerulonephritis. One can argue that several viruses can easily enter the urine during the viraemic stage even in the absence of obvious renal lesions, and that detection of HCV in renal tissue by PCR does not necessarily prove the involvement of this agent in the pathogenesis of the renal disease [4]. Indeed, PCR is a highly sensitive test potentially able to reveal the presence of a single HCV RNA molecule in the infected blood of a normal kidney specimen. In order to assess the possibility of an unspecific finding, we tested the renal tissue of 13 anti-HCV positive patients for HCV RNA; 10 of these patients had various forms of glomerulopathy and 3 had histologically normal kidneys. HCV RNA was extracted from paraffin-embedded kidney specimens obtained by renal biopsy in 7 cases and by autopsy in 6. PCR was performed with primers derived from the 5'-noncoding, highly conserved region of the HCV genome, as previously described [5]. We found HCV RNA in renal tissue from 4 of 5 patients with cryoglobulinemic glomerulonephritis, 3 of 5 with noncryoglobulinemic glomerulopathy, and 2 of 3 with normal kidney. Our findings indicate that the detection of HCV RNA by PCR in renal tissue of patients with glomerular disease cannot be viewed as evidence of a cause and effect relationship between HCV and renal lesions. Nevertheless, these results do by no means exclude that HCV may be the major aetiological agent of cryoglobulinemic glomerulonephritis.

References

Misiani R, Bellavita P, Fenili D, Borelli G, Marchesi D, Massazza M, Vendramin G, Comotti B, Tanzi E, Scudeller G, Zanetti A: Hepatitis C virus infection in patients with essential mixed cryoglobulinemia. *Ann Intern Med* 1992;117: 573-577.

Johnson RJ, Gretch DR, Yamabe H, Hart J, Bacchi CE, Hartwell P, Couser WG, Corey L, Wener MH, Alpers CE, Willson R: Membranoproliferative glomerulonephritis associated with hepatitis C virus infection. *N Engl J Med* 1993; 28:465-470.

Doutrelepon JM, Adler M, Willems M, Durez P, Yap SH: Hepatitis C infection and membranoproliferative glomerulonephritis. *Lancet* 1993; 341:317.

Vas SI: Primary and secondary role of viruses in chronic renal failure. *Kidney Int* 1991;40 (suppl 35):2-4.

Imberti L, Cariani E, Bettinardi A, Zonaro A, Albertini A, Primi D: An immunoassay for specific amplified HCV sequences. *J Virol Methods* 1991;34:233-243.

©1994 S. Karger AG, Basel

0028-2766/94/

0683-0400\$8.00/0