Dear Sir,

Hepatitis C virus (HCV) related chronic liver diseases are important causes of morbidity and mortality in patients undergoing renal replacement therapy. \( \alpha \)-Interferon therapy is widely used to treat HCV-related chronic liver disease in recent years. Because \( \alpha \)-interferon has immunostimulant properties, it is not well known how pretransplant \( \alpha \)-interferon therapy affects the course after renal transplantation in hemodialysis patients with HCV-related chronic liver disease [1, 2]. Two patients with HCV-related chronic liver disease who were treated with \( \alpha \)-interferon before renal transplantation are presented.

Case 1: A 25-year-old male patient with end-stage renal disease of unknown etiology had begun hemodialysis therapy in April 1990. His serum liver enzyme levels were found to be elevated (alanine aminotransferase 160 U/l, aspartate aminotransferase 62 U/l) in November 1990. Serology revealed positive anti-HBs, positive cytomegalovirus (CMV) IgG, negative CMV IgM, and positive HCV antibodies. A liver biopsy was performed in April 1991 because of continuously high serum aminotransferase levels. The biopsy specimen was consistent with chronic hepatitis due to HCV infection. Following liver biopsy, \( \alpha \)-interferon treatment was started (3 million U s.c, three times weekly for 6 months). His serum aminotransferase levels dropped to normal values during the 2nd month of therapy, and thereafter we did not observe any elevation. After the end of the \( \alpha \)-interferon therapy, a second liver biopsy was performed in February 1992 which showed prominent regression of the inflammatory findings as compared with the first biopsy specimen. The patient received a kidney transplant from his brother with triple immunosuppressive treatment (azathioprine 2 mg/ kg, ciclosporin 5 mg/kg, and prednisolone) in November 1992. We have not observed any liver or graft function abnormalities since kidney transplantation.
Case 2: A 46-year-old male patient with end-stage renal disease of unknown etiology started hemodialysis treatment in March 1991. In June 1991 his serum liver enzyme levels were found to be high (alanine aminotransferase 178 U/l, aspartate aminotransferase 257 U/l). Serology results were positive anti-HBs negative CMV IgM, positive CMV IgG, and positive HCV antibodies. His serum aminotransferase levels continued to be high, and a liver biopsy was performed 6 months later which disclosed chronic HCV hepatitis. α-Interferon therapy (3 million U s.c., three times weekly for 6 months) was instituted. His aminotransferase levels normalized during the 2nd month and remained normal throughout the therapy. After α-interferon therapy had been completed, a second liver biopsy specimen (July 1992) showed improvement as compared with the first biopsy findings. A kidney transplantation from his sister was performed in February 1993 with triple immunosuppressive treatment (azathioprine 2 mg/kg, ciclosporin 5 mg/kg, and prednisolone). We have not observed any hepatic or graft dysfunction at follow-up 12 months after kidney transplantation. The serum aminotransferase levels of both patients were within normal limits during the pre- and posttransplant periods following α-interferon therapy. We also have not observed any liver or graft dysfunction after transplantation. Data concerning the effects of α-interferon therapy on graft function during the perioperative period are limited [1, 3]. Although the presence of HCV RNA in the sera of both patients before and after α-interferon therapy and following kidney transplantation could not be clarified, our results suggest that α-interferon therapy may be effective in patients with HCV-related chronic liver disease undergoing renal replacement therapy, without any adverse effect on graft function. However, because number and follow-up periods of such patients are limited, more studies are necessary.

References


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