Increased Prevalence of Anticardiolipin Antibodies in Renal Transplant Recipients

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Table 1. Prevalence of raised IgG-aCLs and other data of hemodialysis (HD) renal transplantation (tx) and control groups

Dear Sir,

Patients with end-stage renal failure undergoing hemodialysis have been found to be associated with a higher frequency of raised anticardiolipin antibodies (aCLs) than healthy controls [1-5]. The presence of aCLs in hemodialysis patients has been related with clinical events such as recurrent arterial and venous thrombosis; however, the data reported by these studies are controversial. The prevalence of aCLs and their relation with thrombotic events in renal transplant recipients is not well known. We performed a cross-sectional study of IgG-aCLs in hemodialysis patients and renal transplant recipients. The aims of the study were (a) to investigate the prevalence of IgG-aCLs in hemodialysis patients, renal transplant recipients and healthy controls, and (b) to find out whether there is an association of raised IgG-aCLs with thrombotic events in these groups.

There were 45 end-stage renal failure patients (27 M, 18 F; mean age 40) treated by hospital-based hemodialysis in the hemodialysis group, 48 renal transplant recipients (31 M, 17 F; mean age 38) in the transplant group, and 43 healthy subjects in the control group (25 M, 18 F; mean age 39). None of the hemodialysis and renal transplant patients had collagen tissue disease based on clinical history and absence of autoantibodies such as rheumatoid factor and antinuclear antibodies. All patients in both groups were dialyzed using cuprophane dialysis membranes. The minimum transplantation time was 19 months. A triple drug regimen (azathioprine, cyclosporine and prednisolone) was used for maintenance immunosuppression in all renal transplant recipients and all had a serum creatinine level of 2 mg/dl or less.

IgG-aCLs in serum were analyzed with an enzyme immunoassay method (BioHy-Thec, Israel). The mean concentration of IgG-aCLs of the control subjects was 6.9 ± 1.4 GPL units and 95% of values were below 15 GPL units. Therefore, we regarded only values above 15 GPL units as being raised. History of thrombotic events was obtained from the patients and their medical records.
Raised IgG-aCLs were found in 8 (17.7%) of the hemodialysis group, in 9 (18.8%) of the renal transplant group and 1 (2.3%) of the control group. The mean of the raised IgG-aCLs concentration was 26 ± 4 GPL units (range 16-62) in the hemodialysis group and 34 ± 5 GPL units (range 16-61) in the renal transplant group (table 1). The prevalence of the raised IgG-aCLs in hemodialysis and renal transplant groups were significantly higher than that among controls (p < 0.05). However, the prevalence and the mean of the raised IgG-aCLs concentrations were comparable between the hemodialysis and transplant groups (p > 0.05) (table 1).

Only 2 patients receiving hemodialysis had thrombotic event history, thrombosis of native arteriovenous fistula in 1 with raised IgG-aCLs and cerebral infarction in the other without raised IgG-aCLs.

High prevalence of raised aCLs found in our hemodialysis patients is consistent with most of the previous studies [1-5]. Although the number of the thrombotic events were limited in hemodialysis patients, it seems that the presence of raised IgG-aCLs in hemodialysis patients were not associated with enhanced thrombosis risk. In this study, the prevalence and the mean of the raised IgG-aCLs in hemodialysis and renal transplant groups were similar. The presence of raised IgG-aCLs in renal transplant patients was not associated with thrombotic events, so there is no evidence that the increased level of IgG-aCLs constitutes a risk factor for thrombosis in renal transplant patients as in hemodialysis patients. Because high levels of IgG-aCLs are the most predictive for thrombotic events, relatively low levels found in our hemodialysis and renal transplant patients may explain why increased prevalence of raised IgG-aCLs were not associated with enhanced thrombosis risk in these patient groups [6].

High prevalence of the raised IgG-aCLs in renal transplant patients may suggest that, when IgG-aCLs occur in hemodialysis patients, neither discontinuation of the hemodialysis nor immunosuppressive therapy can prevent the production of these antibodies following transplantation. The cause of the increased prevalence of raised aCLs in renal transplant patients may be the continuation of the abnormality that had occurred during the uremic period; however, longitudinal studies are necessary to confirm this possibility.

References