Incidence of Australia Antigen in Regular Dialysis Patients and Renal Transplant Recipients

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At the Rigshospitalet in Copenhagen maintenance haemodialysis has been performed since May 1964 and renal transplantation since January 1968.

Haemodialysis patients are tested weekly with determination of serum glutamate pyruvate transaminase. Since April 1968 several patients developed moderately increased transaminase values persisting for long periods. Between August 1968 and June 1969 three patients died from hepatitis, one after renal transplantation, and two dialysis nurses and two doctors developed icteric hepatitis. There is thus clinical evidence that the ward was contaminated with hepatitis virus from mid 1968 to mid 1969. In the absence of jaundice, we have defined hepatitis as an otherwise unexplained increase of the transaminase values to more than double of normal for at least one week.

From November 1969 to March 1970 serum samples from all patients in the ward were tested weekly for Australia (Au) antigen. Discharged renal transplant recipients were tested at their ordinary control visits. The dialysis personnel were tested monthly.

Au antigen was detected by immune electrophoresis which is at least four times as sensitive as the Ouchterlony immuno-diffusion method. Positive sera were tested semi-quantitatively by means of electrophoresis in antibody containing agar-gel by the method of Laurell.

Our haemodialysis series comprised 17 patients. Nine of these had received treatment for more than six months. Seven had contracted hepatitis and five of these had evidence of active hepatitis during our study. However, all patients were Au negative. According to reports from Lund, Liverpool and Philadelphia this should be a strong evidence against viral hepatitis in any of these patients. Serum from one patient contained Au antibody.

Our transplant series comprised 42 of 45 patients. One died from hepatitis before our study. Sixteen had contracted hepatitis and 13 of these had elevated transaminase values during our study; two were icteric. Seven of the 16 patients were Au positive. Three patients who had never presented
evidence of hepatitis were positive. The reactions were all stronger than in any other group of patients studied and remained positive.

The personnel group and the other patients were all negative and had normal serum transaminases.

Most of the renal transplant recipients had been subjected to maintenance haemodialysis in our own or in other dialysis units. No correlation between the presence of Au antigen and the duration of the dialysis period or the number of blood transfusions received could be demonstrated. However, only one of the Au positive patients had not undergone haemodialysis, the others had all been dialysed at the Rigshospital during the term with clinical evidence of contamination with hepatitis virus, and all the Au positive patients had received their graft before September 1, 1969. After transplantation the patients had not received blood transfusions or been exposed to hepatitis infection. Accordingly it is most likely that they had been infected with Au antigen during their haemodialysis period. We consequently investigated our haemodialysis unit to see how the Au antigen had been eliminated.

The general preventive measures had been unaltered during the period under review. From Autumn 1968 the consoles were sterilised with 2% formalin. However, two renewals were introduced. By March 1969 Kiil dialysers were replaced by disposable kidneys. Nevertheless, three patients taken under treatment since then were Au positive. From August 1969 only heat sterilised consoles have been used. The consoles represent a potential source of infection as recirculation is employed during cleaning and sterilisation.

CONCLUSIONS

Persistent and very strong Au antigen reactions have been demonstrated in renal transplant recipients. Introduction of disposable dialysers and heat sterilisation of the consoles have apparently prevented further spreading of Au antigen.