GRAFT SURVIVAL AND HLA ANTIGEN SHARING BETWEEN CADAVER DONORS AND FIRST GRAFT RECIPIENTS TREATED WITH CYCLOSPORIN

Antigens shared	No	Actuarial graft survival (%)	
		1 yr	3 yr
HLA-B+DR			
4	2	100	100
3	25	92	92
2	84	76	76
1	31	58	58
0	6	67	67
HLA-DR			
2	16	94	94
1	87	80	80
0	45	57	57

For HLA-B+DR sharing log rank p=0·01. For HLA-DR sharing log rank p=0·003.

tised. Such patients with high levels of panel reactivity have become increasingly common and they can expect to wait a long time for a suitable donor. This problem alone is sufficient to justify matching at the time of recipient selection, and the benefit of increased survival rates which we observe is a further justification for avoiding A incompatibilities. It is now possible to achieve such high graft vival rates through HLA matching and the use of cyclosporin, that living related donor transplantation is only justified because of the continued shortage of cadaver kidneys.

We conclude that HLA B and DR antigen matching in cyclosporin treated patients is strongly beneficial, and 4-year graft survival rates of over 90% have been achieved by ensuring that no B or DR antigen is mismatched between donor and recipient.

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1. International Symposium on Relevant Immunological Factors in Clinical Kidney Transplantation (Heidelberg, June 19–21, 1985).

NON-A, NON-B HEPATITIS AND HEAT-TREATED FACTOR VIII CONCENTRATES

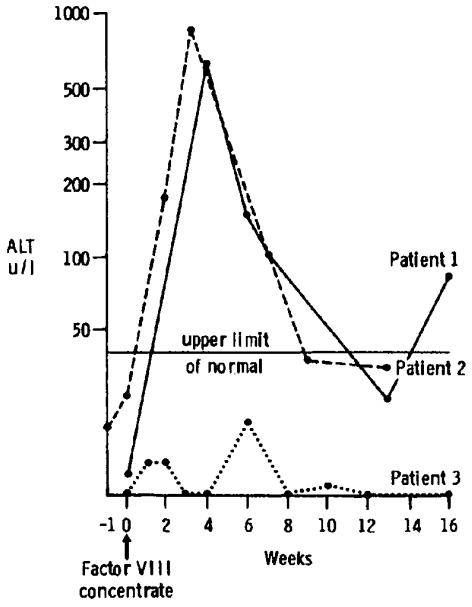
SIR,—Dr Colombo and colleagues (July 6, p 1) describe the development of non-A, non-B (NANB) hepatitis in haemophiliacs receiving batches of heat-treated factor VIII concentrate. We wish to report our experience with dry-heat treated FVIII concentrate in two mild haemophiliacs (patients 1 and 2) and a patient with type IIa von Willebrand's disease (patient 3). None of these individuals had been treated previously with blood or blood products. Before receiving the FVIII concentrate all patients had normal biochemical indices of liver function and were clinically well.

Patient 1 received 16 200 units of Armour intermediate purity, heat-treated FVIII ('Factorate' heat treated) to cover a surgical procedure and patient 2 had 1800 units of the same batch of material to cover dental extractions. Raised plasma transaminase levels (figure) were first observed after 3 and 2 weeks, respectively, reaching a peak 4 weeks after FVIII administration. Both patients became ill with malaise, nausea, and jaundice. Patient 1 required admission to hospital. He had severe jaundice (peak bilirubin 290 μmol/l) which persisted for 16 weeks. There was no serological evidence of hepatitis A or B, cytomegalovirus, or Epstein-Barr virus in either patient; NANB hepatitis was diagnosed.

The development of NANB hepatitis in these two patients contrasts with the uneventful progress of the third patient, given 11 675 units of small donor pool, dry-heat treated, intermediate purity NHS FVIII concentrate, to cover bilateral hip arthroplasty. Reviewed weekly for one month and fortnightly thereafter for 4 months, her hepatic indices have remained persistently normal.

As with the concentrate used by Colombo et al the Armour heat-treated FVIII used in our first two patients had been tested in chimpanzees, without ensuing NANB hepatitis. Our observations thus support Colombo's view that the chimpanzee may be a poor model for NANB hepatitis, and that there is no alternative to clinical trials for virus-depleted blood products.

In two respects the NANB hepatitis in our patients and that described by Colombo et al differ. In our patients the incubation period was very short and similar to that observed in some patients by Kernoff et al[1] and Fletcher et al.[2] Also both our patients became ill and one had to be admitted to hospital. Possible explanations may relate to methods of heat treatment or to the source of the donor pools.



Plasma alanine aminotransferase (ALT) concentrations after use of heat-treated FVIII concentrates.

Although no firm conclusions can be drawn from the uneventful clinical course of the patient who received the NHS product, this was nevertheless unusual since NANB hepatitis almost invariably follows the administration of F VIII concentrate in previously untreated patients.[2] The observed differences in the transmission of NANB between the commercial and the NHS products may relate to the size of the donor pools. In the commercial product the donor pool numbers 1500–3000 compared with the NHS material which was derived from 309 regularly used donors only.

Whilst our three patients remained HTLV-III antibody negative it seems clear that, at least for the commercially obtained product, the elimination of hepatotropic viruses by dry-heat treatment remains disappointing.

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