THE LANCET

Screening of Blood Donors for Non-A Non-B Hepatitis

DESPITE 40 years' efforts to find ways of preventing it, hepatitis still arises after transfusion of blood and blood products. The discovery of the hepatitis B virus and the development of increasingly sensitive tests for markers of hepatitis B infection was a major step forward, but a bigger contribution came from the recognition that paid blood donors, probably because of their lower socioeconomic status, were much more likely to transmit hepatitis than unpaid donors. In the United Kingdom, since the introduction of hepatitis B screening, transfusionists seem to have been mesmerised by this one virus and the thrust of hepatitis prevention has been towards introducing ever more sensitive tests for it, even though the evidence is that little additional protection is gained from tests more sensitive than the widely used haemagglutination assay. When non-A non-B hepatitis was first recognised, many British workers seemed to regard it as a purely American problem. Lately, non-A non-B hepatitis has been accepted in the U.K. as a serious hazard of treatment with factor VIII and factor IX concentrates, which are prepared from very large pools of donor plasma, but no-one has paid much attention to this type of hepatitis in the patient who receives a few units of blood or platelets. In a U.K. prospective study of post-transfusion hepatitis,[1] frank hepatitis developed in 1%, there were sustained increases of alanine aminotransferase (ALT) in 4·5%, and the ALT was raised at some time after transfusion in 20%. Although only a small proportion of these cases of hepatitis and "transaminitis" seemed to be due to hepatitis B virus, nothing has been done to assess the value of preventive methods other than hepatitis B screening.

American workers have been less complacent. At a conference in San Francisco in 1978 three papers[2-4] showed that, if transfusion recipients were followed prospectively, transaminases rose in about 10%. In a sizeable proportion of these, the evidence of liver dysfunction persisted for a long time. Most had no clinical evidence of hepatitis and no laboratory evidence of infection by hepatitis viruses A or B. There are still no reliable serological tests for non-A non-B hepatitis agents, but the Transfusion Transmitted Virus Study Group[5] now report that up to 40% of cases of post-transfusion non-A non-B hepatitis are preventable by rejection of all blood with a serum ALT above 45 IU. These results, though encouraging, need to be looked at very carefully before any decision is made to introduce ALT testing as part of routine screening elsewhere. For a start, the T.T.V. study showed a large geographical difference in the attack rate of post-transfusion hepatitis, ranging from 4% in St Louis to 18% in Houston. The source of blood donors also influenced the chance of post-transfusion hepatitis: irrespective of the ALT level, blood from paid donors was much more likely to transmit hepatitis than that from unpaid community donors. Although there was a strong association between raised donor ALT and post-transfusion hepatitis, it was far from absolute: about 60% of patients who received blood with an ALT above 45 IU did not get hepatitis, while 5% receiving blood with a normal ALT did. There are some other questions. How important in clinical terms is silent transaminitis after transfusion? Although regular users of blood products do get chronic liver disease which is probably due to non-A non-B agents, there is not much information about the long-term consequences of subclinical hepatitis after a single transfusion episode. In the U.K. there is no report about long-term follow-up of transaminitis patients from the earlier study. Furthermore, the value of ALT or other non-specific tests would have to be tested prospectively in various circumstances; after all, there are many reasons why the ALT may be raised, and in some communities a high proportion of blood donors might have to be rejected when the real reason for the abnormal result was alcohol.

If a new donor screening programme was set up, the high cost might be the least of the problems. Today, all transfusion services are aware of the plight of would-be donors who prove to be symptomless carriers of hepatitis B virus. Once these people are labelled as carriers, they may face difficulties in securing medical or dental care. We should be very much aware of the risks of creating a new and much larger group of donors who are rejected because of a new "hepatitis" test which does not necessarily signify infectivity, and which may be detecting a form of infection whose natural history we know very little about.

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