Vox Sanguinis
Editor-in-Chief: C. P. Engelfriet, Amsterdam

Reprint
Publisher: S. Karger AG, Basel
Printed in Switzerland

40

Short Communication

Vox Sang. *45:* 87–88 (1983)

0042–9007/83/0451–0087 $2.75/0

A Donor Implicated in Two Cases of Post-Transfusion Non-A, Non-B Hepatitis

J. A. J. Barbara[a], *Marcela Contreras*[a], *Moya Briggs*[b, 1]

[a] North London Blood Transfusion Centre, Edgware, Mddx., UK; [b] Department of Microbiology, Middlesex Hospital Medical School, London, UK

In the absence of specific tests for non-A, non-B hepatitis viruses, evidence for their involvement in post-transfusion hepatitis (PTH) can only be circumstantial. This report describes an example where two successive blood donations, spaced by 7 months, from the same donor were both implicated in cases of PTH.

A UK-born male had donated one of only two units given as whole blood to a patient who became jaundiced 6 weeks after transfusion. Serum taken from this patient at the time of jaundice was negative by RIA [1] for HBsAg, anti-HBc and anti-HAV, but weakly positive for anti-HBs. After notification of this case of PTH, the two donors involved were resampled. One donor had normal liver enzyme levels and no serum markers of hepatitis B virus (HBV) infection. Serum from the other donor was negative by RIA for HBsAg but positive for anti-HBc (IgM class negative) and anti-HBs. This donor also had mildly elevated liver enzyme levels (ALT of 41 and AST of 35; upper limit of normal for both 30 IU/l).

[1] We are grateful to Miss *Marina Mobed* for clerical assistance.

Although the implication of the second donor was only presumptive, he was asked to refrain from blood donation until further notice and his records were withdrawn from our routine donor file. Despite these recommendations, he returned as a new donor 7 months later and his donation was one of four units given as whole blood to a patient. After we notified the hospital, a sample taken from this patient 3 months after transfusion showed elevated transaminases. These were still elevated 1 week later when the patient's serum was checked for HAV and HBV markers. Although anti-HAV was found in the serum, the antibody was not of the IgM class. The serum was negative for HBsAg and anti-HBc though a trace of anti-HBs was present; as with the earlier case, this level was consistent with passive transfer from the implicated donor. Transaminase levels had returned to normal when the patient, who had not suffered jaundice in the interim, was resampled after a further 10 weeks. The previously implicated donor was again sampled and, 18 months after the first PTH case, his GOT was 40 and GPT was 31 IU/l. The levels of his anti-HBV antibodies remained unchanged. The 3 other donors

involved were resampled and their sera showed normal liver enzyme levels and no HBV markers.

An association of elevated ALT levels in donors and increased risk of transmission of non-A, non-B hepatitis after transfusion has been widely reported [2]; the continuing elevated levels of liver enzymes in our implicated donor are consistent with this association. However, although his serum was anti-HBc positive, it contained anti-HBs as well. Therefore, he was not in the category of the 'anti-HBc only' donors that has been associated with increased risk of transmitting non-A, non-B hepatitis [3–5]. The prevalence of anti-HBs in our region is 2% [6]. Therefore, 3,200 of our donations per year will be from anti-HBs positive donors who are not associated with a proportional number of PTH cases. The presence of anti-HBc together with anti-HBs does not in itself indicate a high risk of subsequent non-A, non-B PTH. Nevertheless, the situation described (a donor who is both anti-HBs and anti-HBc positive and is suspected of a possible link with PTH) justifies, in our opinion, the permanent exclusion of this donor from donating blood. This procedure is reminiscent of measures taken for the prevention of PTH B before HBsAg tests became available.

References

1 Barbara, J. A. J.; Briggs, M.: Hepatitis of the non-A, non-B type following blood transfusion in the North London region. Med. Lab. Sci. *38:* 423–426 (1981).

2 International Forum: Based on your analysis of the benefits and costs of routine donor screening for ALT-GPT to reduce the incidence of post-transfusion non-A, non-B hepatitis in your blood services region, what action would you recommend on this matter? Vox Sang. *44:* 48–64 (1983).

3 Stevens, C. E.; T.T.V. Study Group, New York Blood Center: Antibody to hepatitis B core antigen in donor blood and the risk of non-A, non-B hepatitis in recipients. Transfusion *21:* 607 (1981).

4 Cossart, Y. E.; Kirsch, S.; Ismay, S. L.: Post-transfusion hepatitis in Australia. Lancet *i:* 208–213 (1982).

5 Katchaki, M. S.; Siem, T. H.; Brouwer, R.: Non-B post-transfusion hepatitis with hepatitis B core antibodies in donor blood. New Engl. J. Med. *307:* 628 (1982).

6 Tedder, R. S., Cameron, C. H.; Wilson-Croome, R.; Howell, D. R.; Colgrove, A.; Barbara, J. A. J.: Contrasting patterns and frequency of antibodies to the surface, core and e antigens of hepatitis B virus in blood donors and homosexual patients. J. med. Virol. *6:* 323–332 (1980).

Received: February 15, 1983
Accepted: March 17, 1983

Dr. John A. J. Barbara,
North London Blood Transfusion Centre,
Deansbrook Road,
Edgware, Mddx. HA8 9BD (UK)