INVESTIGATION OF CHRONIC HEPATITIS C INFECTION IN INDIVIDUALS WITH HAEMOPHILIA

Two recent articles have discussed the role of liver biopsy in the management of patients with coagulation disorders who are infected with hepatitis C virus (HCV) (Hanley *et al*, 1996; Ahmed *et al*, 1996). Both reports referenced a review of 126 biopsies in 115 haemophilic patients in which there was clinically significant haemorrhage in 12·5% procedures and which identified two unreported deaths, representing a fatality rate of over 1% (Aledort *et al*, 1985). One of these deaths occurred at the Royal Free Hospital, London, in the early 1980s when it was the practice to biopsy patients with chronic liver disease (Bamber *et al*, 1981). Since death from liver biopsy contributes so strongly to the argument about the investigation of liver disease in haemophilia, it is important that this death is reported.

The patient, born in 1957, was diagnosed as having haemophilia A at 2 months of age when he presented with bruising. There was a family history of haemophilia with a first cousin known to have haemophilia A. He was first treated with blood products at the age of 2 years in 1959, when he received fresh frozen plasma (FFP) for treatment of a haemorrhage when his front teeth were knocked out. From 1966 he was treated with cryoprecipitate and a small amount of unsterilized lyophilized factor VIII concentrate. His first exposure to unsterilized large-pool factor VIII was on 26 June 1972 to cover an aspiration of the left knee. It is significant that he became jaundiced in September 1972 when the HBsAg was negative; this presumably was non-A non-B hepatitis, or hepatitis C infection. During the following 8 years he continued to have a cholestatic jaundice intermittently. A decision was therefore made to biopsy the liver and this was performed on 3 October 1980. Following the biopsy, he bled into the abdomen and was taken to the operating theatre where a torn capsule was found and hepatic artery ligation was required to stop the haemorrhage. Blood product cover for this surgery included 42 units of whole blood, 21 units of platelets, 11 units of FFP, as well as factor VIII concentrate. During the next 3 d he was in intensive care, where he continued to bleed and developed acute renal failure. 4 d after the biopsy a further laparotomy was performed and a right hemihepatectomy was performed. Following this surgery he continued to bleed and required haemodialysis. He died on GRO-A 1980, 8 d after the liver biopsy, aged 23 years.

Therefore the conclusion that was made by Aledort *et al* (1985) must still be relevant: the risks of liver biopsy for haemophilic patients are not insignificant. The availability of serological testing for chronic HCV, PCR for persisting viraemia, genotypic analysis of HCV, quantitation by PCR of HCV RNA, ultrasound and CT means that most patients may be managed without resorting to liver biopsy. Finally, it must be remembered that the cost of clotting factor concentrate to cover each liver biopsy is approximately £7000 but the annual cost of alpha-interferon (3 mu × 3 weekly) is £3000.

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