
With the opening of the hemophilia center in Oct. 1972 an intensive replacement therapy with clotting factor concentrates was started the first time for many patients. Concerning the induced liver damage no following resume could be drawn. From 113 examined patients 17 (15%) acquired an icteric hepatitis (I.H.). Excluding 2 cases the interval between beginning of intensive clotting factor treatment and the onset of icterus was 4-18 months. The icterus lasted 13-117 days (average 100). The elevation of ALT in the range of 1-45 U (100-279 µU) of clotting factor concentrates had been injected until to this moment. (The average consumption by the 113 examined patients from Oct. 1972 to Oct. 1975 was 37 186 µU per patient and year.) In Oct. 1975 the distribution of anti-HBs and HBs-Ag positive patients (radio immuno assay) was: Frequently transfused cases (more than 15000 µU per year): Anti-HBs 86%; HBs-Ag 68%; total 94%. Less frequently transfused cases: Anti-HBs 71%; HBs-Ag 81%; total 81%. In 18 patients (not identical to those with I.H.) liver biopsies could be performed with the following results: chronic hepatitis: 9, subacute hepatitis without necrosis: 4, alcoholic liver damage: 3, acute dystrophy: 1, cholangiolitis: 1.


Abnormal liver function tests (LFTs) in patients with hemophilia A and B who have been treated with commercial factor concentrates have been observed since 1975. One hundred and seventeen of these had had LFTs performed at least twice and at least six months apart. Of those, 15 were chronically HBs-Ag positive and the rest anti-IgB positive. Forty percent of the anti-IgB group had persistently abnormal LFTs for greater than 6 months, the majority for more than two years. Owing to the uncertain relationship of this "transaminosis" with true liver pathology, especially in the anti-body positive patients, seven closed liver biopsies were performed without incident under high coverage with factor VIII replacement. All patients were symptomatic at the time of biopsy. One had a prior episode of illness probably related to subclinical hepatitis. Eight patients underwent an open liver biopsy as part of a staging laparotomy for Hodgkin Disease. Six biopsies showed varying degrees of chronic persistent hepatitis (CPH). One was not evaluated owing to changes related to chemotherapy. Postmortem histologic evaluation of one biopsy revealed chronic persistent hepatitis with significant fatty change. The biopsy results suggest that the "transaminosis" found in transfused hemophiles represents histologic liver disease. In the majority of patients studied, CPH, a relatively benign entity, was present; and this does not justify withdrawal of concentrate therapy at this time.