Possible reactivation of hepatitis D with chronic δ antigenaemia by human immunodeficiency virus

We describe a case of reactivation of infection with hepatitis D virus (δ) in an intravenous drug abuser who was a carrier of hepatitis B surface antigen (HBsAg). This condition was manifested by chronic & antigenaemia of at least two years’ standing and was probably induced by infection with human immunodeficiency virus.

Seroconversion of antihuman immunodeficiency virus antibody

This article is a reproduction of that published in: British Medical Journal, 294(658), June 1987, pp.1656-1657. Pagination may not match that of the original.
Temporal changes in results of detailed serological tests for hepatitis B and D viruses in 21 year old drug abuser.

**Case report**

Detailed serological tests for hepatitis B and D viruses were carried out on a series of specimens from a 21-year-old male drug abuser in whom acute hepatitis B had been diagnosed in February 1983. At that time HBsAg was detected by commercial radioimmunoassay and enzyme immunoassay kits (Abbott); hepatitis B e antigen (HBeAg) and antihepatitis B core IgM were detected by in house enzyme immunoassays. There was no jaundice and his liver enzyme activities were slightly raised. Jaundice of three weeks’ duration occurred six months later, however, in August 1983.

Antihepatitis D IgG was detected in October 1983 by enzyme immunoassay (Deltassay B, Noctech). Antihepatitis D IgM was also detected by an in house enzyme linked immunosorbent assay (ELISA) using hepatitis D antigen (HDAg) derived from serum, which indicated that his hepatitis in August had been caused by a superinfection with hepatitis D virus. Antihepatitis D IgG persisted throughout 1984.

Reactivation of infection with hepatitis D virus was first detected in July 1984, when transient HDAg appeared resulting in seroconversion to antihepatitis D IgM. Reactivation was again detected in January 1985, after which he showed increasing amounts of serum HDAg (detected by enzyme immunoassay Deltassay, Noctech) with loss of antihepatitis D IgG and fluctuating amounts of antihepatitis D IgM.

Tests for antibody to human immunodeficiency virus were carried out retrospectively. Enzyme immunoassay gave an equivocal result at first, but a Western blot analysis (using DuPont preprepared nitrocellulose strips) produced a positive result in his specimen of October 1983; all subsequent tests (Wellcome enzyme immunoassay) remained positive. The activities of his liver enzymes were persistently raised and variable throughout, and a liver biopsy in October 1984 indicated the presence of chronic active hepatitis. In October 1986 he had lymphadenopathy but had not returned for follow up at the time of writing.

**Comment**

The serological results strongly suggest that our patient acquired superinfection with hepatitis D virus and human immunodeficiency virus about six months after acute infection with hepatitis B virus. The unusual serological pattern and the rising concentration of HDAg in serum strongly suggest reactivation of replication of hepatitis D virus. Furthermore, his most recent specimens showed reversion from antihepatitis B e antibody to HBeAg, which indicates reactivation of hepatitis B virus as well. Unfortunately the patient has been lost to follow up and the importance of this reactivation of hepatitis D virus is therefore unknown. Although this is the first reported occurrence of reactivation of infection with hepatitis D virus by human immunodeficiency virus, the large numbers of drug abusers in Dublin who are infected with hepatitis D virus and the increase of such infections in proportion with infections with human immunodeficiency virus (A G Shattock et al, international symposium on AIDS, Paris 1986)

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make it likely that other reactivations of infection with hepatitis D virus will occur in this susceptible population.


(Accepted 6 March 1981)

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