Viral hepatitis in drug addicts

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Summary

Viral hepatitis is an important and frequent complication of drug addiction. The discovery of the Australia antigen and its association with serum hepatitis has provided a number of specific serological tests which may be used for the diagnosis of at least a proportion of patients with hepatitis and also for the detection of carriers of the hepatitis virus. These tests might now be used to study in greater detail than has hitherto been possible the epidemiology of serum hepatitis among drug dependents, their contacts and the community at large.

During the last 20 years there has been a steady increase in the use of and addiction to drugs in large cities in the United States and more recently a similar increase has been noted in many of the capital cities of the world. The increase in the number of known and suspected narcotic addicts, especially amongst young males, presents an alarming picture. An apparent rise in the incidence of hepatitis in this group of young adults has also been noted and indeed de Alarcón (1969) has applied successfully the technique of epidemiology used in the study of infectious diseases on the premise that drug addiction is spread from one person to another as a contagious disease. Indirect evidence of the frequency of viral hepatitis amongst narcotic addicts was provided by Cohen & Douglas (1968) who found that the risk of hepatitis in recipients of blood was increased seventy fold when the blood had been obtained from known or suspected drug addicts. Potter, Cohen & Norris (1960) also found retrospectively that five donors implicated in post-transfusion hepatitis were all heroin addicts.

Aetiological factors

The known stability of the aetiological agents of viral hepatitis and the transmission of hepatitis by incompletely sterilized syringes and needles, which had become contaminated with the blood of a carrier or a patient incubating the disease, an anicteric patient or a convalescent patient, have recently been reviewed (Zuckerman, 1970a). Steigmann, Hyman & Goldbloom (1950) were the first to describe an outbreak of hepatitis amongst narcotic addicts and since then many similar reports have been published suggesting that the infection was acquired through the communal use of syringes and needles for the administration of heroin, methylamphetamine and other drugs (Alter & Michael, 1958; Levine & Payne, 1960; Bewley, Ben-Arie & Marks, 1968; Sapira, Jasinski & Gorodetzky, 1968). Whilst epidemiologically outbreaks of hepatitis among narcotic addicts strongly implicate an infectious process a direct hepatotoxic action has been suggested as the cause of liver damage. Marks & Chapple (1967), for example, did not consider that abnormal liver function tests in addicts were primarily due to viral hepatitis. Experiments in morphine-addicted Macacas rhesus monkeys (Brooks et al., 1963) and experiments in human volunteers who were made dependent on a daily morphine dose of 240 mg for 6–8 months (Gorodetzky et al., 1968) established that long-term morphine administration per se is not associated with the development of abnormal liver function tests. The possibility still remains, of course, that other ingredients used for diluting heroin may be hepatotoxic although this does not appear to be an important common factor. It should be noted, however, that it is extremely difficult to distinguish clinically, biochemically and histologically between toxic or drug hepatitis and viral hepatitis. Malnutrition is also unlikely to be of great significance although a number of addicts are underweight. Chronic alcoholism as a cause of liver damage, especially in the very young addicts, seems improbable and the studies of Norris & Potter (1965) would seem to support the contention that alcohol does not play a major role in the induction of inflammatory lesions in the liver.

The Australia antigen

The discovery of the association between the
Australia antigen (Blumberg et al., 1967) and hepatitis has at long last provided laboratory tests for an antigen which is specifically related to serum hepatitis or type B viral hepatitis (Zuckerman, 1970b). The general descriptive term hepatitis-associated antigen has been proposed for the Australia antigen, Au (1), the SH antigen and the Au/SH antigen, which are antigenically very similar, if not identical. The presence of this antigen in blood serves as a specific indication of potential infectivity or evidence of actual infection with the serum hepatitis virus, although it must be stressed that the sensitivity of the currently available techniques does not provide an absolute index of infection (Bulletin of the World Health Organization, 1970). Furthermore, it now seems likely that the Australia (hepatitis-associated) antigen is not the actual infectious agent but that it represents either incomplete virus particles, aggregates of protein subunits or excess production of unstable virus-like particles or protein by infected cells as a result of infection with the serum hepatitis agent (Zuckerman, Taylor & Bird, 1970; Zuckerman, 1970c). Nevertheless, the availability of tests for this antigen has provided the means for studying and re-evaluating the epidemiology of serum hepatitis.

A number of studies in narcotic addicts employing tests for the Australia (hepatitis-associated) antigen have recently been reported. These studies have provided supporting evidence for the infective nature of the hepatic lesions in this population group (Jersild et al., 1970; Farrow et al., 1970; Cherubin, Hargrove & Prince, 1970; Hunter et al., 1970). The problem of hepatitis among drug addicts may well have been underestimated since there is some evidence that the agent of serum hepatitis may be transmitted not only by the parenteral route but also by the oral route (Krugman, Giles & Hammond, 1967) and by close and intimate personal contact. Cherubin et al., (1970) examined by the immunodiffusion test serial serum specimens for the Australia (hepatitis-associated) antigen from patients with viral hepatitis admitted to two hospitals in New York. The antigen was found in the acute sample of blood in 54% of patients with viral hepatitis who were known heroin or methamphetamine users. The antigen was also detected in 2-3% of drug addicts in whom there was no evidence of hepatitis and who were admitted to an institution for the treatment of drug withdrawal. In the group of adolescents and young adults with hepatitis who denied the use of drugs the antigen was found in 50%. Yet the frequency of detectable antigen among volunteer blood donors in New York is only 0.1%. It is suggested that either the incidence of serum hepatitis in New York city in this ‘high-risk’ age-group might be due to a greater addiction problem than had hitherto been considered or that other modes of transmission of hepatitis associated with this antigen are involved.

Knowledge of the epidemiology of serum hepatitis among drug addicts remains incomplete at present and the spread of infection to family contacts, to medical and nursing personnel and indeed to the community at large presents a real problem which should be investigated further.

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References


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