

A Secondary Outbreak of Hepatitis B Among Contacts of Drug-Abusers in Dublin

D. A. KELLY[†]
M.D., M.R.C.P.I.

D. G. WEIR
M.D., F.R.C.P.I.

D. CARROLL*
B.Soc.Sc., M.Sc.Econ:

Department of Clinical Medicine
Trinity College, St. James's,
Sir Patrick Dun's and Cherry
Orchard Hospitals

A. G. SHATTOCK
Ph.D.

E. O'CONNOR
L.R.C.P.&S.I.

Virus Reference Laboratory
Department of Medical Microbiology
University College
Dublin

Acute type B hepatitis has been relatively uncommon among drug-abusers in Ireland until 1980 when there was a marked increase in heroin addiction associated with the increased availability of this drug. Subsequently, there has been a continuing epidemic of acute hepatitis B in parenteral drug-abusers¹ (Fig. 1).

The aim of this study was to investigate the extent of the spread of this epidemic to non-drug-abuser contacts of the parenteral drug-abusers; the mode of transmission of the infection to these patients; and to compare the hepatitis B serology in both parenteral and non-drug abusers.

Methods and Patients

Twenty-seven patients who were attending Cherry Orchard and the Federated Dublin Voluntary Hospitals who presented in the six months from July, 1981, with hepatitis B surface antigen (HBsAg) positive hepatitis and who denied parenteral drug abuse, were studied. Twenty-two male and six female HBsAg positive parenteral drug abusers from the same period of the epidemic were used for serological comparison.

Serological Methods: Serological tests for all hepatitis B virus (HBV) markers were carried out at the Virus Reference Laboratory, Department of Medical Microbiology, U.C.D. using radio-immunoassay (RIA, Abbott Ausria II) for the detection of HBsAg with overnight incubation at 45°C to increase sensitivity and immunodiffusion (ID) for the detection of hepatitis B e antigen (HBeAg) and antibody (anti-HBe) as previously described. HBsAg subtypes were determined by ID using in-house reagents prepared from haemophilic serum as previously described.²

Follow-up Patients: Patients' interviews were designed to determine the potential modes of non-parenteral transmission of hepatitis B. Interviews, with the exception of three in hospital, were conducted in the patients' homes by a sociologist. Where possible, a blood sample was taken from patients and their close contacts. In one case, the interviewee was the patient's spouse.

Seventeen of the twenty-seven patients initially included were finally selected. The reasons for exclusion were: two were no longer resident in Ireland, three proved impossible to

* Funded by the Health Education Bureau.

contact, despite numerous attempts, and five admitted to parenteral drug abuse at the interview despite previous denials.

All patients contacted were cooperative at interview with the exception of one patient who refused to name contacts but was otherwise helpful. Fifteen of the 17 patients contacted were resident in Dublin at the time of interview.

Results

Following the epidemic in drug-abusers there has been a steady increase in the numbers of hepatitis B positive patients who were not drug abusers, a peak being attained 4 months later than the start of the epidemic in drug-abusers (Fig. 2). This epidemic has been mainly confined to Dublin where the number of cases among non-drug abusers has doubled in this time (Table 1).

Patients: There were 11 male and 6 female patients. The mean age was 33 years (range 13-53 years). Nine patients

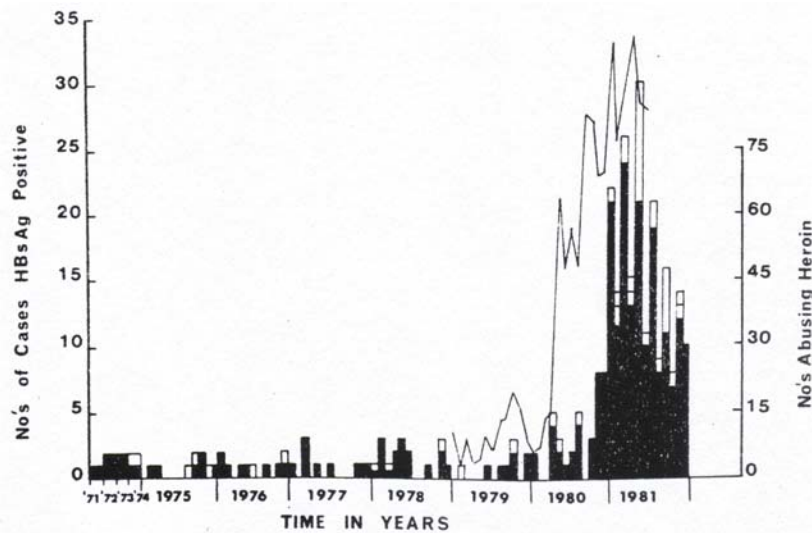


Fig. 1: The extent of parenteral heroin abuse in Dublin 1980-1 and the subsequent epidemic of hepatitis B in parenteral drug abusers (by kind permission Dr. J. Murphy, *Irish Journal of Medical Science*).
 numbers of patients abusing heroin
 HBsAg positive drug abusers
 Anti-HBs positive drug abusers

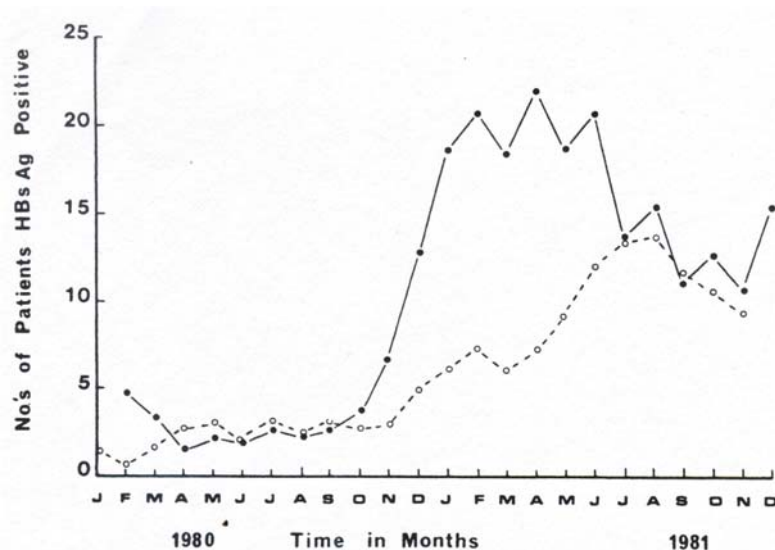


Fig. 2: Running three monthly averages of numbers of cases of hepatitis B in Dublin 1980-81.
 Drug Abusers
 Non-Drug Abusers

Table 1
Number of HBsAg positive patients 1980-1981

	Numbers of HbsAg positive patients	
	1980	1981
Dublin:		
Drug Abusers	34	170
Non-Drug Abusers	45	109
Elsewhere*	<u>07</u>	<u>15</u>
	86	294

*Drug-abuse not determined

Table 2
Source of Hepatitis B

SOURCE	NO. OF PATIENTS	
Heterosexual	5+	(29%)
Homosexual	2	(12%)
*Occupational	4	(23 %)
*Therapy	1	(6%)
‘Shared utensils’	2 ⁺	(12%)
Carrier	1	(6%)
Foreign travel	1	(6%)
Unknown	1	(6%)
	<u>17</u>	

⁺6/7 confirmed contact with drug abusers

*contact with blood products

Table 3
Sub-Type of HBsAg compared to source of Hepatitis

CONTACT	NO. OF PATIENTS	
	SUBTYPE	
	<i>ad</i>	<i>ay</i>
Heterosexual	–	5*
Homosexual	2	–
*Occupational	2	2
Therapy	–	2
‘Shared utensils’	–	1
Carrier	1	–
Foreign travel	1	–
Unknown	–	1

*6/7 confirmed contact with drug-abuser

were single, 6 were married and 2 were separated from their spouses. Seven patients had families ranging in size from 1 to 7 children; two patients had illegitimate children. Occupations ranged from higher professional to unskilled worker and five were unemployed. Eleven patients were hospitalised and five were treated at home. One patient was a 13-year-old haemophiliac who was asymptomatic and was diagnosed following serological testing at an annual check-up. Another patient was a carrier with normal liver function.

The course of the hepatitis was mild in most of the patients, with classic features and no complications; the mean hospital stay was three weeks. Four patients had a prolonged hospital course with persistent cholestasis and transaminases greater than 600 i.u. All made a complete recovery.

Modes of Transmission: The main mode of transmission of hepatitis B to these patients was either contact with blood or blood products or sexual contact with persons already infected (Table 2).

Blood or Blood Products: Five (29%) patients had contact with blood or blood products within the six months before their illness. One, the haemophiliac, had received a blood transfusion, while two laboratory technicians, one microbiologist and one nurse had had occupational contact.

Sexual Contact: Seven patients are believed to have acquired their hepatitis by sexual contact within the six months prior to onset of hepatitis. Of these, four had sexual intercourse with their spouse or regular partner and all of these contacts were HBV infected parenteral drug-abusers. One had sexual intercourse with his spouse who was a psychiatric nurse who had had hepatitis during 1981. Two had homosexual contact with unidentified persons, therefore the source of their hepatitis is less certain. However, hepatitis B has been shown to be endemic in homosexual males.^{3,4}

Other Contact: Two patients claimed that the only form of contact with HBV infected persons was sharing cooking and eating utensils.- Of the remaining three patients, one was a carrier, one could not recall any contact and in the third case the patient had been in the Far East where the HBsAG carrier rate is among the highest in the world.⁵

Follow-up of Patients Contacts: Blood samples were taken from twenty close contacts of the patients in the study group. None of these contacts, including 13 sexual partners were either HBsAG or anti-HBs positive.

Sub-Type of Virus: The sub-type of the HBsAG in the study group was *ad* in 6 (35%) and *ay* in 11 (65%). In the drug-abusers, the sub-type was *ay* in all 28 patients. When the sub-type of the virus was compared to the source of the hepatitis it was observed that the 7 patients whose contacts were drug abusers were all *ay* while those in whom the cause was either not established or occupational were either *ay* or *ad* (Table 3).

Discussion

The spread of acute hepatitis B among parenteral drug abusers is well recognised.^{6,7,8} There is now considerable evidence of the importance of non-parenteral spread although the exact mechanism is not understood.^{9,10}

This study has shown that the recent epidemic of hepatitis B was not confined to parenteral drug abusers in Dublin, but was followed 4 months later by an increase in hepatitis B among non-drug-abusers, doubling the expected number of cases. As the second outbreak occurred within the incubation period of hepatitis B, this suggests that the increase is due to spread from the HBV infected drug-abusers.

The results of the follow-up study illustrate the difficulties in carrying out this kind of survey and explain the relatively small number of patients eventually included. The main difficulty was in tracing and making contact with patients who had moved or did not wish to co-operate. Five patients from the original study group admitted to drug-abuse at the interview confirming previous reports that many drug-abusers will not admit to abuse in hospital for fear of hospital authorities or police, but are more likely to do so in an informal situation.⁸

Despite the small numbers, the results clearly show that the main source of infection was spread from parenteral drug-abusers to their sexual partners or other close contacts. Transmission of the virus by heterosexual contact varies from 25% to 50% and is associated with promiscuity in the infective phase,^{9,3} which may be the case here. It may also explain the absence of sexual transmission to the contacts of patients who had no connection with drug-abusers, all of whom were monogamous.

The predominant sub-type of HBsAg in affected blood donors in Ireland is *ad*, although, with the increase in drug abusers, the ratio of *ad/ay* is now changing as the latter are all *ay*.¹¹ The finding that all the contacts of the drug abusers in our study were the 'ay' sub-type, suggests that the same virus was transmitted.

Only 2 of our study group were homosexuals and both had the *ad* sub-type.

An alarming observation was the number of medical and technical personnel who had been infected in the course of their work, demonstrating the increased risk to these workers as a result of this epidemic. The need for adequate protective regimes to prevent the spread of this virus among such personnel is highlighted. Ultimately this epidemic has increased the number of HBsAg positive patients in Ireland. Inevitably this will lead to an increase in the number of carriers and of patients with chronic liver disease, cirrhosis and eventually hepatocellular carcinoma.¹²

This study indicates that at present the groups most at risk of acquiring HBsAg positive hepatitis apart from drug abusers themselves, are their sexual partners and the medical and technical staff who care for them. The recent increase in the abuse of drugs and the lack of adequate control of their distribution has led to a far wider dissemination of the virus within our society than before. The implications for the overall health of this society are serious and should not be underrated.

Summary

A recent increase in hepatitis B among non-drug-abusers has been investigated. The evidence suggests that this increase is a direct result of spread from an earlier epidemic among drug-abusers since the timing of the increase was within the incubation period of hepatitis B and the subtype of the virus in these contacts was *ay* which is the predominant subtype in drug-abusers. A detailed follow-up on non-drug-abusers showed that the main mode of transmission was by sexual contact with intravenous drug-abusers.

This outbreak may have serious consequences for Ireland because as the number of HBsAg positive patients increases so will the incidence of chronic liver disease and possibly hepatoma. This is particularly important for the groups most at risk, namely, parenteral drug-abusers, their contacts and the medical and technical staff who care for them.

Acknowledgement

We wish to thank the Health Education Bureau for financial support.

1. Shattock A.G., Kelly M.G., Fielding J. and Arthurs Y. (1982). *Irish J. Med. Sci.*, .In Press.
2. Shattock A.G. and Gorry H. (1974). *Irish J. Med. Sci.*, 143. 41: 214-219.
3. Szmunes W., Much I., Prince A.M. et al. (1975). *Ann. Int. Med.*, 83, 4: 489-495.
4. Szmunes W., Stevens C.E., Sang E.A., et al. (1982). In "Viral Hepatitis". (1981) International Symposium Franklin Institute Press. 467-486.
5. Cossart Y.E. (1977). In "Virus Hepatitis," Bailliere Tindall.
6. Steigman F., Hyman S. and Goldbloom R. (1950). *Gastroenterology*. 15: 612-646.
7. Cherubin C.E., Hargrove R.L., Prince A.M., et al. (1970). *Am. J. Epidemiol*, 91: 510-517.
8. Blanck R.R., Ream N., Conrad M. et al. (1979). *Am. J. Gastroenterol*, 71: 164-167.
9. Heathcote J., Sherlock S. and Gateau Ph. (1974). *Lancet*, ii: 370-373.
10. Francis D.P., Favero M.C., and Maynard J.E. (1981). In *Seminars in Liver Disease* 1. (I): 27-32.
11. Shattock A.G. and Smith A. (1977). *Hep. Sci.*, Memo H-1212/1.
12. Beasley R.P., Lu-Yu J., Lin C.C. and Chien C.S. (1981). *Lancet*, ii: 1129-1133.