

INTRAFAMILIAL SPREAD OF HEPATITIS Bs ANTIGEN

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Abstract

A survey of 238 household contacts of 93 patients admitted with acute viral hepatitis was carried out. HBs antigenaemia was observed in 7% families of HBs Ag negative hepatitis and 19.4% of HBs Ag positive hepatitis. Abnormalities of transaminases were more frequent in contacts of HBs Ag positive hepatitis. The highest prevalence of antigenaemia was observed among spouses (23.5%) of index cases of HBs Ag positive hepatitis. The evidence indicates a sexual spread of HBs Ag in this series.

Introduction

Acute viral hepatitis, its sequelae and asymptomatic cases of these clinical entities are frequently seen in Pakistan (Ahmad and Quraishi, 1975; Zuberi, 1977). Thirty-three per cent cases of hepatitis and 20.4% cases of cirrhosis have hepatitis B antigenaemia (Zuberi et al., 1978). Variations in the routes of transmission of HBsAg have been observed by various workers (Blumberg et al., 1969; Mosley, 1975; Hersh et al., 1971). The purpose of this study was to determine the pattern of transmission of HBsAg in the household contacts of patients with acute viral hepatitis.

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Material and Methods

The series included 93 cases of acute viral hepatitis admitted to the medical wards of Jinnah Postgraduate Medical Centre, Karachi from January 1st to December 31st 1976 and 238 of their family contacts.

The clinical history, physical findings, eating habits and living conditions were carefully recorded. An attempt was also made to determine the other sources of exposure to hepatitis virus like injections, blood transfusion, surgical and dental procedures and the exposure to hepatitis in relatives other than the index cases. Screening for hepatitis B antigen was carried out by counter-current immunoelectrophoresis (Zuberi and Lodi, 1974) and the cases were divided into antigen positive and negative groups. The transaminase activity was determined by the spectrophotometric method in both the groups. Diagnosis of acute viral hepatitis suspected on clinical history and biochemical findings was confirmed by needle biopsy of the liver.

Results

Clustering of HBsAg was observed in 4(7.02%) of 57 families of HBsAg negative

Hepatitis and 7(19.44%) of 36 families of HBsAg positive hepatitis patients. Transmission of HBsAg occurred more frequently to young male members of the family (Table I), seven of the 13 HBsAg positive contacts were either housewives or school going children.

Table I: Intrafamilial Spread of Hepatitis B Antigen-Sex Distribution

<i>HBs Ag Negative Hepatitis</i>		<i>HBs Ag Positive Hepatitis</i>	
No of patients	— 57	No of patients	—36
No of contacts	—151	No of contacts	—87
HBs/Ag+ve Male	— 5	HBs/Ag+ve Male	— 6
HBs/Ag+ve Female	— 0	HBs/Ag+ve Female	— 2
HBs/Ag—ve Male	— 79	HBs/Ag—ve Male	—51
HBs/Ag—ve Female	— 67	HBs/Ag—ve Female	—28

Eating in wayside hotels and temporary accommodation without toilet facilities appeared to facilitate the spread of Hepatitis Bs antigen. No differences were observed in the clinical history and physical findings of HBsAg positive contacts of two groups of hepatitis.

Abnormalities in serum transaminases were more frequent in contacts of HBsAg positive Hepatitis. Four out of 8 HBsAg positive contacts were spouses of index patients with HBsAg positive Hepatitis and the remaining were parents, siblings or offsprings (Table II).

The frequency of HBs antigenaemia in the relatives of HBsAg positive hepatitis was high but no differences were observed in that of apparently healthy carriers, blood donors and relatives of patients with HBsAg negative hepatitis (Table III).

Table II: Intrafamilial Spread of Hepatitis B Antigen-Relationship to the Patient

<i>Relationship</i>	<i>Contacts of HBs Ag Negative Hepatitis</i>		<i>Contacts of HBs Ag Positive Hepatitis</i>	
	<i>No. tested</i>	<i>HBs/Ag+ve No (%)</i>	<i>No. tested</i>	<i>HBs/Ag+ve No (%)</i>
Spouses	14	—	17	4(23.53)
Offsprings ..	39	2(5.12)	27	1(3.70)
Parents	34	1(2.94)	12	1(8.33)
Siblings	43	1(2.33)	15	2(13.33)
Others	21	1(4.76)	16	—

Table III: Prevalence of Hepatitis B Antigen in Apparently Healthy Groups

<i>Groups</i>	<i>Total No.</i>	<i>HBs/Ag positive No(%)</i>
Controls	1517	48(3.16)
Blood donors ..	1149	42(3.65)
Hospital staff ..	300	1(3.0)
Contacts of HBs Ag negative Hepatitis	151	5(3.31)
Contacts of HBs Ag positive Hepatitis	87	8(9.20)

Discussion

Certain patterns of transmission of HBsAg have emerged from the family studies of blood donors (Szmunness et al., 1975) and patients with acute and chronic liver diseases (Bruguera et al., 1974) with hepatitis B antigenaemia. They are based either on genetic inheritance or on parenteral or non parenteral exposure to hepatitis B antigen.

A recessive inheritance for the susceptibility of infection by hepatitis B virus has been observed by several workers (Blumberg et al., 1969; Bruguera et al., 1974). Szmunness et al (1975) found the highest prevalence of antigenaemia among sibs and parents and lowest among spouses. The evidence is contrary to these findings in the present series.

In the absence of any history of parenteral exposure the non-parenteral modes of spread are most likely in this study. Doubts in the infectivity of stools in the presence of an inhibitor of HBsAg in the faecal suspension and homogenate of intestinal mucosa (Piazza et al., 1973) make a faecal-oral route of transmission unlikely in those living in poor conditions with no toilet facilities.

HBsAg has been detected in the semen, saliva (Heathcote et al., 1974) and in menstrual blood (Mazzur 1973) in both homosexual and heterosexual partners of individuals with hepatitis B antigenaemia. The predominance of spouses

among the HBsAg positive contacts of patients with hepatitis B favours the sexual spread.

The findings in this study suggest that the relatives of patients with HBsAg positive Hepatitis are at a higher risk of acquiring HBsAg than those of HBsAg negative hepatitis. Therefore, the screening of families of hepatitis patients is essential to identify cases of asymptomatic acute and chronic liver diseases and healthy carriers of HBs antigen who are likely to spread the disease.

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