STUDIES ON HBsAg IN HIV SEROPOSITIVE CASES

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ABSTRACT

Two hundred and fifty five patients with human immunodeficiency virus (HIV) infection were screened for hepatitis B virus surface antigen (HBsAg) over a period of 3 years. These patients were followed up every 6 month. 10.2% of HIV seropositive cases revealed presence of HBsAg. It was also observed that almost half (47.6%) of these cases developed carrier state.

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KEY WORDS : Hepatitis B virus; Human immunodeficiency virus; Acquired immunodeficiency syndrome.

Introduction

Human immunodeficiency virus (HIV) and hepatitis B virus share a common mode of transmission i.e. sexual, parenteral and perinatal. It has been observed that hepatitis B virus surface antigen (HBsAg) positive patients with HIV infection take longer time to achieve HBsAg seronegativity. This prompted us to undertake the present study.

Material and Methods

The material for the present study included all 26 cases found to be HBsAg positive in 255 HIV seropositive patients hospitalised from June 1991 to April 1994. In addition to recording of detailed history, each patient underwent general physical examination and systemic examination of abdomen, chest, cardiovascular and central nervous systems and a set of investigations consisting of haemoglobin estimation, total and differential white cell count, platelet count, ESR, urine analysis, liver function tests, blood for VDRL, Mantoux test, radiogram chest and HBsAg testing (reverse passive haemagglutination test). Competitive ELISA was used for screening and Western blot test was performed for confirmation of HIV infection. All

patients with no positive clinical and laboratory findings were followed up once a year and the remaining every six months. Clinical examination and investigations were repeated during each follow up.

Results

Majority (96.1%) of patients were in third and fourth decades of life. The youngest was 20 years and the oldest patient was 53 years of age. Most (92.2%) of the patients volunteered history of extramarital heterosexual exposure. There was history of receiving blood transfusion in 11.3%. A small number (3.8%) denied history of extramarital sexual exposure, receiving blood transfusion, surgery, intravenous drug abuse or of tattooing. HBsAg was found to be positive in 26 (10.2%) of 255 HIV seropositive cases.

Mode of presentation of HBsAg positive patients was sexually transmitted disease in 12 cases (42.4%), viral hepatitis in 5 (19.3%), blood donation in 3 (11.5%), pulmonary tuberculosis and fever of unknown origin in 2 (7.7%) each; and herpes zoster in one (3.8%). One patient (3.8%) had volunteered for HIV ELISA testing.

HBsAg antigenaemia was observed in 20 patients (77%) at the onset (19.3% having

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viral hepatitis and 57.7% were asymptomatic HBsAg carriers). In 3 patients (11.5%) HBsAg antigenaemia appeared after 6 months and in another 3 patients (11.5%) after one year (asymptomatic HBsAg carrier) (Table 1). No case was diagnosed HBsAg positive after one year of follow up of HIV seropositive cases. Two patients (7.7%) were diagnosed to be suffering from AIDS, and hence invalidated out of service, 2 patients (7.7%) were released from service on superannuation and one (3.8%) died of generalised cryptococcosis. The remaining 21 patient were followed up for HBsAg status. Of these, 11 patients (52.4%) showed disappearance of HBsAg by 6 months

TABLE 1

Time taken for HBs antigenaemia in HIV seropositive patients

Time of first detection of HBsAg positivity	Number	Percentage
At the outset (diagnosed during investigation for concomitant disease in HIV seropositive cases)		
a. Viral hepatitis	5	19.3
b. Asymptomatic HBsAg carrier	15	57.7
After 6 months		
a. Chronic HBsAg carrier	—	—
b. Asymptomatic HBsAg carrier	3	11.5
After 12 Months		
a. Chronic HBsAg carrier	—	—
b. Asymptomatic HBsAg	3	11.5
Total	26	100

TABLE 2

Time taken for HBsAg seronegativity

Duration	Number of HBsAg seronegative patients	Percentage
< 6 months	11	52.4
7 - 12 months	1	4.8
13 - 18 months	2	9.5
19 - 24 months	—	—
25 - 30 months	1	4.8
> 31 months	6*	28.5
Total	21	100

*Remained HBsAg positive

(3 patients had viral hepatitis earlier and 8 were HBsAg carriers), one patient (4.8%) become negative between 7 to 12 months, 2 patients (9.5%) in 13 to 18 months one patients in 25 to 30 month and 6 patients (28.5%) showed persistence of HBs antigenaemia even after 30 months follow up (including 2 patients who had viral hepatitis earlier).

Discussion

Hepatitis is known to be caused by a number of viruses. Of these, hepatitis B virus (HBV) is of special significance since it may lead to either carrier state, chronicity of disease, development of cirrhosis or malignancy. HBsAg is an important marker of HBV infection. It appears in blood about six weeks after infection. Approximately 90% of patients with acute hepatitis B have HBsAg detectable in their serum when they present for medical care. However, it disappears from blood by 3 months. Persistence of HBsAg for more than six months implies a carrier state [1].

Both HIV and HBV are predominantly transmitted by sexual contact and parenterally e.g. transfusion of infected blood and blood products, contaminated needles etc. Hence, there are considerable number of cases having mixed infection. The prevalence of HBsAg in HIV seropositive is recorded as 5 to 10 percent [2]. The prevalence of HBsAg in HIV infection cases was 10.2% in the present study, whereas incidence of HBsAg in healthy population [3] and pregnant women [4] is 5% and 4.8% respectively in Indian population. Higher incidence in the present group appears to result from simultaneous infection through common route of entry by HIV and HBV.

There is increasing evidence that important interaction between HIV and HBV may occur. This could be produced by at least two mechanisms. First, both have a stage of reverse transcription in their replication cycle. Second, destruction of HBV infected hepatocytes with consequent clearance of hepatitis B virus is thought to be mediated by cytotoxic

lymphocytes [5]. Because of these mechanisms in patients with past or present infection with HBV, HIV infection can lead to significant alteration in HBV replication, antigen/antibody display and clinical consequences. A few reports have described reappearance of HBsAg in HIV infected patients previously thought to be immune to HBV indicated by the presence of antiHBs [6]. In our study, definite history of viral hepatitis was present in 19.2% HIV seropositive cases with HBs antigenaemia. There was no history, clinical or laboratory abnormality suggestive of viral hepatitis in the remaining 80.8%. HBsAg appeared after six months in 11.5% and in another 11.5%, it appeared 12 months after detection of HIV infection.

It had been documented that approximately 10% patients contracting hepatitis B do not clear HBsAg from the serum within six months. No data is available about time taken to achieve HBsAg negativity in HBV infection alongwith HIV infection cases. In our study, HBsAg become negative in 52.4% in 6 months where as in 47.6% cases it had taken more than 6 months. Of the HIV seropositive patients who had viral hepatitis, 60% became HBsAg negative by 6 months whereas in 40% it persisted beyond 6 months. It is, therefore, inferred that comparatively much longer time

is required to achieve HBsAg negativity in HIV seropositive patients. This data is not recorded in literature.

To conclude, the study revealed considerable percentage of HIV seropositive cases having HBsAg antigenaemia. HIV infection appeared to prolong the HBsAg positive state thereby increasing the chances of development of chronicity.

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