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HBSAG Positivity in HIV Seropositive Individuals

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Abstract: *HIV and AIDS have brought about a global epidemic, far more extensive than what was predicted. HIV infection appears to influence the natural history of infection with Hepatitis viruses. There is a high degree of epidemiological similarity between the Hepatitis B virus and HIV as regard to high risk groups, route of transmission and presence of virus in the body fluids. The present study aimed at determining the HBsAgseropositivity in HIV positive individuals and to see the correlation between HBsAg positivity and immune status as reflected by absolute CD4 counts. Out of a total of 1056 serum samples tested, 2.7% were positive for HBsAg marker. In HBsAg negative subjects, absolute CD4 count of more than 500 cells/µl was seen in 59.5% cases, while in the HBsAg positive subjects, this count was seen in 10.7% cases. A low countof less than 200 cells/µl was seen in 35.7% cases who were HBsAg positive and in 13.5% subjects who were HBsAg negative. Thus, every individual detected to be HIV positive must be screened for Hepatitis B markers. This will predict prognosis of HBV infection and progression to AIDS.*

Keywords*: AIDS, Hepatitis B, immune status*

I. Introduction

The Acquired Immunodeficiency Syndrome (AIDS) has become a major phenomenon during the last two decades. HIV virus, its causative agent, is a plague that has changed the face of our society and will continue to wreak havoc in the health sector for years to come.

The modes of transmission of HIV are heterosexual or homosexual contact, inoculation of infected blood or blood products, needle stick injury and vertically from mother to fetus.

Like HIV, Hepatitis B Virus (HBV) infection represents a worldwide public health problem, with mortality and morbidity it produces from acute and chronic illness. About 5-10% of HBV infections result in chronic carrier state with persistence of Hepatitis B surface antigen (HBsAg) in the circulation for more than six months. The prevalence of HBsAg carrier rate varies in India, from region to region and is influenced by environmental, behavioural and host factors.[1]

Several properties of HBV and HIV suggest that coinfections are frequent and clinically important.[2] Like HIV, Hepatitis B can be spread parentrally and sexually.

HIV infection alters the course of Hepatitis B. These alterations probably arise from HIV induced impairment of both humoral and cellular immunity. Increased carrier rates, viral replication and milder liver injury are seen in patients with Hepatitis B.

Alternately, HBV infection is thought to be a cofactor, directly or indirectly affecting the rate of progression of AIDS.[3] Further, the HBV infection also increases the risk of successful HIV transmission.[4]

The present study evaluates the prevalence of HBsAg positivity ratein HIV infected individuals and co-relates the CD4 counts with HBV seropositivity. This would indirectly throw some light on the already established synergism between the two viruses.

II. Materials And Methods

This study is a retrospective analysis of HBsAg positivity in HIV positive individuals. It was conducted in the Department of Microbiology, Goa Medical College, Bambolim, Goa, India, over a period of one year, from January to December, 2013.

All sera samples received from HIV positive subjects in the Department of Microbiology, during the study period were tested for HBsAg, using a rapid, qualitative, two site sandwich immunoassay, on a membrane, using the principle of immunochromatography (Orchid Biomedical Systems, Verna, Goa, India). The test procedure was followed as per the instruction manual provided in the kit.

The absolute CD4 lymphocyte count ofeach subject was noted. The CD4 count was done using the BD FACS Count System which is an automated instrument and reagent kit designed specifically for enumerating the absolute cell counts of CD4, CD8 and CD3 T lymphocyte in unlysed whole blood.

III. Results

A total of 1056 serum samples were received from HIV positive subjects for HBsAg evaluation during the study period. Of all these samples tested, 28 i.e. 2.7% were found to be positive for HBsAg marker; while 1028(97.3%) wereHBsAg negative.

An analysis and comparison of the absolute CD4 counts of the subjects who were HBsAg positive and negative is depicted in Table no 1.

In the HBsAg negative subjects, an absolute CD4 count of more than 500 cells/µl was seen in 59.5% cases while low counts of less than 200 cells/µl and between 200 and 350 cells/µl were seen in 13.5% and 8.1% cases respectively.

However, a reverse was seen in HBsAg positive subjects. An absolute CD4 count of less than 200 cells/µl was seen in 35.7% subjects. Another 35.7% of subjects showed CD4 counts between 200 and 350 cells/µl, while a CD4 count of more than 500 cells/µl was seen in only 10.7% of subjects.

IV. Discussion

AIDS is an epidemic, which has become unquestionably, the major public health problem of our era. It is a disease with treatment that is still beyond the reach of the common man and prognosis, very guarded.

AIDS is closely linked to drugs, sex and death. The sexual route, being the commonest mode of transmission of HIV, invites coinfection with other sexually transmitted infections. Among viruses, the Hepatitis B, C and D viruses can spread parentrally and sexually; thus increasing the frequency of coinfection if these viruses with HIV.

HBV infection theoretically acts as a cofactor in HIV, hastening the progression of HIV seropositivity to AIDS case. In the present study 71.4% HBsAg positive and HIV positive cases had a low absolute CD4 count of less than 350 cells/µl. Amongst these cases, 35.7% had counts of less than 200 cells/µl, thus fitting into AIDS.

Indirect effect of HBV on HIV replication via cytokines such as TNFα has been shown to increase expression of HIV infected cells in vitro.[5]This enhanced HIV expression may contribute to a progressively increased viral burden and CD4 cells depletion that accompanies clinical progression to AIDS.[6]

HBV infection also acts as a direct viral cofactor, increasing the risk of successful transmission per contact during sexual exposure between an HBV infected and uninfected person. This is because the lymphocytes of the HIV susceptible individual are activated by residual HBV infection. The effect of immune activation of mononuclear cells in increasing their susceptibility to infection when challenged with HIV was studied and observed by Setoet al.[4]

The resolution of acute infection is mediated by the host's immune response, primarily by T lymphocytes. In the presence of HIV infection, when the cellular immunity is impaired, there is bound to be unfettered replication of HBV and more frequent development of chronic infection.[7] Further, this effect of impaired cellular immunity increases the risk of these patients becoming chronic HBsAg carriers.[8]

Although in the present study, all the HBsAg positive subjects were asymptomatic and not in the acute phase of HBV infection, the HBsAg carrier rate could have probably been influenced by the HIV seropositive status.Similar observation has been noticed in HBV positive persons with Downs syndrome or patients treated with immunosuppressive drugs.[8]

In a study conducted by Lebovics et al,[9] 10% of HIV positive persons with HBV infection became carriers of HBsAg as compared to HBV positive patients without infection (less than 5%).

HBV and HIV coinfection can lead to increased incidence of HBV reactivation and reinfection. In a study in Northern India, the rate of HBV infection in HIV positive patients was 2.25%.[10]Another study from Iran showed a much higher coinfection of HBV and HIV i.e. 14.5%.[11]

V. Tables

Table 1: Absolute Cd4 Counts OfHbsAg Positive And Negative Subjects

CD counts (cells/µl)	HBsAg negative subjects	HBsAg positive subjects
<200	139(13.5)	10(35.7)
200-350	83(8.1)	10(35.7)
350-500	194(18.9)	5(17.9)
>500	612(59.5)	3(10.7)
Total	1028(97.3)	28(2.7)

NB: Figures in parenthesis indicate percentages.

VI. Conclusion

Early and effective screening for HBV is a must in every detected case of HIV. This will help to initiate appropriate measures and as well as therapy which will reduce the morbidity and mortality associated with Hepatitis. It will also help to predict prognosis of HBV infection and progression of HIV infection.

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