

# SGOT, SGPT and Hepatitis B Antigen in Leprosy

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Serum glutamic pyruvic transaminase (SGPT) and serum glutamic oxaloacetic transaminase (SGOT) levels were determined in seven cases of tuberculoid leprosy carrying hepatitis B antigen (HB-Ag) and compared with the levels in 50 HB Ag-negative cases of tuberculoid leprosy. The mean value for SGPT and SGOT in the former group was 7.86 and 8.86 IU/L respectively, while in the latter it was 9.08 and 11.48 respectively. Similarly the mean values for SGPT and SGOT in eight HB Ag-positive cases of lepromatous leprosy were 8.88 and 13.75 IU/L respectively while the mean figures for 50 HB Ag-negative cases of lepromatous leprosy were 7.96 and 12.48 IU/L respectively. The differences obtained in lepromatous leprosy were statistically insignificant. There was no difference in the serum enzyme levels of HB Ag-positive and negative cases and the range of values obtained were comparable. The absence of liver damage as indicated by enzyme studies suggests the host-parasite relationship of the hepatitis B virus in leprosy to be one of symptomless carriage.

## Introduction

Hepatitis B antigen (HB-Ag), a surface antigen of the Hepatitis B virus, occurs more frequently in sera of patients with leprosy than in the normal populations (Blumberg *et al.*, 1967; Blumberg and Melartin, 1970a). The interaction between man and this virus shows an intriguing variety of host-parasitic relationships which

range from symptomless carriage, through mild chronic liver injury which can lead to cirrhosis of the liver to the explosive attack of acute viral hepatitis (Sherlock *et al.*, 1970). Carriage of HB antigen in leprosy is possibly of two types (1) harmless carriage and/or (2) associated with mild hepatic damage. Blumberg and Melartin (1970*b*) studied liver function in 28 cases of lepromatous leprosy carrying HB antigen and found that only the SGPT levels, though often in the normal range, were significantly higher than those in a similar number of leprosy patients not carrying the antigen. The inference was that the hepatitis B virus caused mild liver injury. Post-mortem studies in the literature, however, do not record cirrhosis of the liver to be a common occurrence in patients with leprosy (Kean and Childress, 1942). This rarity of cirrhosis suggests that HB antigen carriage is not frequently associated with chronic liver injury.

We have had occasion to study HB antigen carriage in a large series of leprosy patients (Kelkar *et al.*, 1973*b*). During these studies, SGPT and SGOT levels of some of the patients with and without HB antigen were estimated, with a view to investigate the nature of the host-parasitic relationship of the hepatitis B virus carriage in patients with leprosy.

### Material and Methods

The patients consisted of seven with tuberculoid and eight with lepromatous leprosy all of whom had HB antigen in their sera. For a comparison, 50 patients with tuberculoid and 50 with lepromatous leprosy and not carrying the HB antigen, were studied. Sera from these patients were tested for SGOT and SGPT levels by the method of Reitman and Frankel (1957). Detection of HB-Ag was done by both agar gel double diffusion and a counter immunoelectrophoretic technique (Kelkar *et al.*, 1974; Kelkar *et al.*, 1972).

### Results

Figures 1 and 2 are scatter diagrams illustrating the values for SGPT and SGOT obtained in the patients studied. Table 1 gives the mean figures for the different groups as well as the numbers of cases showing raised enzyme levels. The accepted upper normal limits for SGPT and SGOT are 15 and 20 IU/L respectively

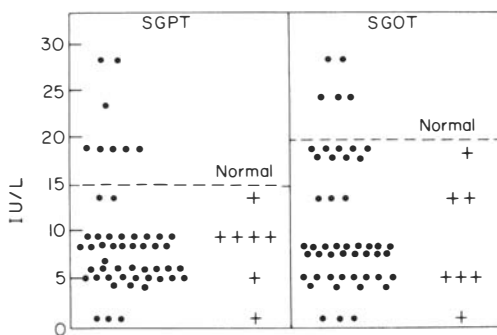


Fig.1. Scatter diagram showing SGPT and SGOT levels of cases of tuberculoid leprosy. + = HB-Ag positive case; ● = HB-Ag negative case.

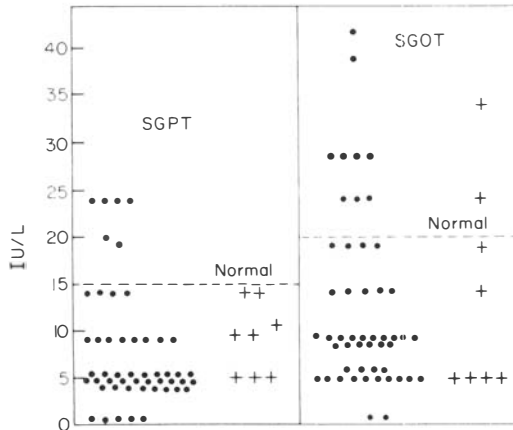


Fig.2. Scatter diagram showing SGPT and SGOT levels of cases of lepromatous leprosy. + = HB-Ag positive case; • = HB-Ag negative case.

(Wooton, 1964). A reference to the table shows that the mean enzyme values of the 50 HB-Ag negative cases of tuberculoid leprosy are actually higher than the means for the seven cases which showed presence of antigen in the serum. The scatter diagram (Fig.1) shows that the range and values obtained are not essentially different. Moreover none of the HB-Ag positive cases had abnormal (raised) enzyme levels.

With lepromatous leprosy the results obtained were slightly different. The mean value of enzymes obtained in 50 cases of lepromatous leprosy not carrying HB-Ag were lower than those obtained with HB-Ag carriers having lepromatous leprosy. These slight differences, however, when analyzed by statistical methods proved to be of no significance ( $P=0.54$  and  $0.68$ ). A glance at the scatter diagram (Fig.2) again brings out the essential similarity of the results in the two groups.

TABLE 1

*SGPT and SGOT (International Units/L) in HB-Ag positive and negative patients with leprosy*

GROUP	No. of cases	SGPT Mean value	No. of cases with SGPT over 15 IU/L	SGOT Mean value	No. of cases with SGOT over 20 IU/L
(I) Tuberculoid	57				
HB-Ag negative	50	9.08	8	11.48	5
HB-Ag positive	7	7.86	0	8.86	0
(II) Lepromatous	58				
HB-Ag negative	50	7.96	6	12.48	9
HB-Ag positive	8	8.88	0	13.75	2

### Discussion

The results for SGPT and SGOT, in general, are in keeping with the well documented observations that there are no serious derangements of liver function in leprosy (Shivde and Junnarkar, 1967). Progressive liver damage due to *Mycobacterium leprae* is not known. Further, the results indicate that there is no difference in the enzyme levels in cases of leprosy with or without carriage of the hepatitis B virus. The inference that can be validly drawn is that the hepatitis B virus does not damage the hepatocyte to cause a rise in enzyme levels. Of the available tests these enzyme estimations are the most sensitive indicators of hepatic parenchymal damage. The interaction between the hepatitis B virus and the leprosy patient, therefore, appears to be that of symptomless carriage.

We still do not know for certain the outcome of symptomless HB-Ag carriage either in the leprosy patient or in normal individuals. The very fact that the hepatitis B virus is present in the serum in such large amounts as to be detected by relatively crude immunological methods indicates a considerable growth of the virus. The accepted site for this is the hepatocyte (Millman *et al.*, 1969). Therefore disruption of some hepatocytes and hepatic damage is unquestionable. The point of interest is whether there is sufficient damage to disorganize normal liver regeneration so that a terminal condition like cirrhosis of the liver develops with the passage of years. A sequential follow-up study would finally decide this issue.

A lot of data is available in the literature regarding the condition of normal individuals carrying HB-Ag (Lous *et al.*, 1970; Singleton *et al.*, 1971; Lebacqz, 1971; Ricci *et al.*, 1972). These studies prove that a variable number of HB-Ag carriers have hepatic injury, with the likelihood of development of cirrhosis of the liver.

Cirrhosis of the liver is not a common finding in patients with leprosy. Thus Kean and Childress (1942) noted cirrhosis in only seven of 103 patients studied at autopsy in Panama. Similarly Shivde and Junnarkar (1967), studying liver biopsies, found only five of 43 cases to have cirrhosis of the liver. In contrast, in a study of portal cirrhosis in patients without leprosy we have found as many as 34% to be associated with HB-Ag (Kelkar *et al.*, 1973a). This raises the question of the relationship of HB-Ag with cirrhosis of the liver in patients with leprosy. Because of the rarity of the combination, a study of a large series of such cases is not feasible. The indirect evidence of enzyme levels presented in the present study suggests that this is not a frequent happening.

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