THE USE OF SEVERAL MARKERS OF HEPATITIS B INFECTION TO MONITOR RISKS OF INFECTION IN A HAEMODIALYSIS UNIT AND IN LABORATORIES

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INTRODUCTION

The test for HB_sAg, anti-HB_sAg, anti-HB_cAg and DNA polymerase activity may be used not only as markers for hepatitis B infection but they may also be used as indicators for the stage of the infective process. HB_sAg is first detected in the serum during the incubation period over 6 - 26 weeks (1½ - 6½ months) or less and during the early phase (first 2 - 3 weeks) of the acute infection (Hoofnagle, 1979). In many cases of acute infection HB_sAg may only be detected in the serum for a few days. In some patients, HB_sAg may persist despite clinical improvement indicating that a chronic form of hepatitis or a chronic carrier state may be developing (Hoofnagle, 1979). Anti-HB_s is usually detected during the mid - to late period of convalescence form of infection. Usually there is a variable interval between the disappearance of HB_sAg and the appearance of anti-HB_s. In general the presence of antibody is presumptive evidence of a previous infection and immunity to hepatitis B virus (Hoofnagle, 1979). Anti-HB_c is usually detected in the serum while the acute infection is still in progress. Thus during the acute stage, HB_sAg and anti-HB_c may be present at the same time. Anti-HB_c is often the only hepatitis B virus marker in the blood during early convalescence when HB_sAg has disappeared and before anti-HB_s has appeared. In mid-to-late convalescence, anti-HB_c may be present together with anti-HB_s.

DNA polymerase is usually detected in the incubation period and disappears before the acute stage arises. However, it can persist for months or years in chronic carriers (Krugman *et al*, 1974).

This study attempts to determine the prevalence of infection among patients and staff in a haemodialysis unit and laboratory staff and to monitor the stage of the infective process.

MATERIALS AND METHODS

Blood samples were collected as described by Ton *et al* (1979).

Serum samples were tested for the presence of hepatitis B surface antigen (HB_sAg) and antibody to hepatitis B surface antigen (anti-HB_s) by a solid-phase radioimmunoassay (Abbot, Austria-125; Ausab, respectively) while the test for anti-HB_c, antibody to the core antigen was based on the principle of competitive binding (Abbot, Corab^{TM}).

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TABLE I
SIGNIFICANCE OF HBV MARKERS

Category	HBV Markers	Intepretation
I	Positive for DNA polymerase alone	early incubation period
II	Positive for DNA polymerase, HB_sAg and/or anti-HBc	incubation period/Acute hepatitis B
III	Positive for HB_sAg	late incubation/early acute stage
IV	Positive for HBsAg, anti-HBc	'acute' stage, chronic hepatitis or carrier state
V	Positive for anti-HBc	early convalescence 'silent' carrier
VI	Positive for anti-HBc and anti-HBs	mid to late convalescence
VII	Anti-HBs alone	Immunization without infection

Counting of the samples was done on the Packard Autogamma Scintillation Spectrometer, Model 5110.

The method for detecting the enzyme activity was as described by Ton *et al* (1979). Counting of the samples was done on the Packard Tricarb Model 3255 Liquid Scintillation Spectrometer.

RESULTS

The results were divided into seven categories:- positive for DNA polymerase (1), positive for DNA polymerase, HB_sAg and/or anti-HB_c (2), positive for HB_sAg (3), positive for HB_sAg and anti-HB_c (4), positive for anti-HB_c (5), positive for anti-HB_c and anti-HB_s (6), and positive for anti-HB_s alone (7). The interpretation of the various markers as listed is based on current concepts as reported by Hoofnagle (1979) and for ease of reference is tabulated as shown in Table I.

As seen in Table II 88 percent of the haemodialysis patients and 76 percent of the staff from the haemodialysis unit have one or more hepatitis B markers in their serum compared to 21 percent of the Blood Bank staff and 57 percent of the Biochemistry Laboratory staff.

DISCUSSION

Studies carried out on staff and patients of HDU indicate a high exposure rate to HBV infection. Approximately all the patients have markers and can develop into chronic carrier states. From the evidence presented, staff members of HDU also run the risk of becoming chronic carriers or developing hepatitis. The exposure rate of the staff of both the

TABLE II
PATTERN OF HEPATITIS B MARKERS IN HAEMODIALYSIS PATIENTS AND IN DIFFERENT CATEGORIES OF HIGH RISK PERSONNEL

			CATEGORY							
				Incubation	Acute	Acute/Carrier		Early Convalescence	Late Convalescence	Immunization without infection
	No.	No. Expressed (%)	0	I	II	III	IV	V	VI	VII
Haemodialysis Patients	25	22 (88%)	3(12%)	1	1	2	8	3	7	-
Haemodialysis staff	33	25 (76%)	8 (24%)	10	3*	0	4	3	5	-
Blood Bank staff	14	3 (21%)	11 (78%)	0	1	0	0	0	2	-
Biochemistry staff	7	4 (57%)	3 (43%)	2	0	0	0	2	-	
Normal Blood Donors	189	117 (61.9%)	72 (38%)	8	6	4	6	55	38	-

* 3 negative for HB_sAg positive for DNA polymerase, anti-HB_c and anti-HB_s.

biochemistry laboratory and the blood bank is even lower than the normal blood donors which serve as a control. Reports (Maynard, 1978) have shown that laboratory staff especially those dealing with blood and blood products are more at risk to the HBV infection. Our work on such personnel does not seem to bear this out. In fact the blood bank staff appeared to have lower exposure rate than the normal blood donors. The low values might be due to the extra care taken by the blood bank staff in their daily handling of the blood or that the number is too small for comparison. In view of the relatively high prevalence of HBV in our population regular monitoring of blood donors and of those in specialised high risk hospital units like the HDU and laboratory should be carried out as routine procedures in our hospitals. Not only is the prevention of spread of HBV within hospital units of extreme importance but also the prevention of carrier states, chronic liver diseases and liver cancer in the population. It has been reported that there is a high correlation between hepatocellular carcinoma (HCC) and hepatitis B virus (HBV) infections (Szumness, 1978) and HBV in conjunction with a yet unidentified contributing factor or factors might institute the following events:- exposure to HBV -> induction of HB_sAg carrier state -> chronic hepatitis -> cirrhosis -> HCC. In some instances the chain may be shortended and asymtomatic antigenemia may progress directly into HCC without chronic hepatitis or cirrhosis (Szmuness, 1978).

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