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 HBsAg, anti-HBsAg, anti-HBcAg 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. HBsAg 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 HBsAg may only be detected in the serum for a few days. In some patients, HBsAg may persist despite clinical improvement indicating that a chronic form of hepatitis or a chronic carrier state may be developing (Hoofnagle, 1979). Anti-HBs is usually detected during the mid-to late period of convalescence form of infection. Usually there is a variable interval between the disappearance of HBsAg and the appearance of anti-HBs. In general the presence of antibody is presumptive evidence of a previous infection and immunity to hepatitis B virus (Hoofnagle, 1979). Anti-HBc is usually detected in the serum while the acute infection is still in progress. Thus during the acute stage, HBsAg and anti-HBc may be present at the same time. Anti-HBc is often the only hepatitis B virus marker in the blood during early convalescence when HBsAg has disappeared and before anti-HBs has appeared. In mid-to late convalescence, anti-HBc may be present together with anti-HBs.

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 (HBsAg) and antibody to hepatitis B surface antigen (anti-HBs) by a solid-phase radioimmunoassay (Abbot, Austria-125; Ausab, respectively) while the test for anti-HBc, antibody to the core antigen was based on the principle of competitive binding (Abbot, CorabTM).
TABLE I
SIGNIFICANCE OF HBV MARKERS

<table>
<thead>
<tr>
<th>Category</th>
<th>HBV Markers</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Positive for DNA polymerase alone</td>
<td>early incubation period</td>
</tr>
<tr>
<td>II</td>
<td>Positive for DNA polymerase, HBsAg and/or anti-HBc</td>
<td>incubation period/acute stage</td>
</tr>
<tr>
<td>III</td>
<td>Positive for HBsAg</td>
<td>late incubation/early acute stage</td>
</tr>
<tr>
<td>IV</td>
<td>Positive for HBsAg alone</td>
<td>'acute' stage, chronic hepatitis or carrier state</td>
</tr>
<tr>
<td>V</td>
<td>Positive for anti-HBc and anti-HBs</td>
<td>mid to late convalescence 'silent' carrier</td>
</tr>
<tr>
<td>VI</td>
<td>Anti-HBs alone</td>
<td>Immunization without infection</td>
</tr>
</tbody>
</table>

RESULTS

The results were divided into seven categories: positive for DNA polymerase (1), positive for DNA polymerase, HBsAg and/or anti-HBc (2), positive for HBsAg (3), positive for HBsAg and anti-HBc (4), positive for anti-HBc (5), positive for anti-HBc and anti-HBs (6), and positive for anti-HBs 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

<table>
<thead>
<tr>
<th>CATEGORY</th>
<th>Number of Samples</th>
<th>Incubation</th>
<th>Acute</th>
<th>Acute/Carrier</th>
<th>Early Convalescence</th>
<th>Late Convalescence</th>
<th>Immunization without infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemodialysis Patients</td>
<td>25</td>
<td>5 (12%)</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>Haemodialysis staff</td>
<td>33</td>
<td>8 (24%)</td>
<td>10</td>
<td>3*</td>
<td>0</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Blood Bank staff</td>
<td>14</td>
<td>11 (78%)</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Biochemistry staff</td>
<td>7</td>
<td>5 (43%)</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Normal Blood Donors</td>
<td>189</td>
<td>72 (58%)</td>
<td>8</td>
<td>6</td>
<td>4</td>
<td>6</td>
<td>55</td>
</tr>
</tbody>
</table>

* 3 negative for HBsAg positive for DNA polymerase, anti-HBc and anti-HBs.
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 \( \Rightarrow \) induction of HBsAg carrier state \( \Rightarrow \) chronic hepatitis \( \Rightarrow \) cirrhosis \( \Rightarrow \) HCC. In some instances the chain may be shortened and asymptomatic antigenemia may progress directly into HCC without chronic hepatitis or cirrhosis (Szumness, 1978).

REFERENCES


