only 5.5% documented their responses. From non-documented vaccinees, 9% were susceptible to HBV infection. It seems that a voluntary vaccination policy may not be as effective, as shown in another study where between 25% and 55% of HCW were not vaccinated under a voluntary system.

Therefore, optimal vaccine coverage would be best achieved by mandatory vaccination of all HCW while being trained. In addition all vaccinated HCW should have their response documented.

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References


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Hepatitis C infection correlates with alteration of serum immunoglobulins pattern in chronic liver disease

Hyperglobulinemia affecting the three main immunoglobulin classes was considered a hallmark of chronic active hepatitis, but studies before 1989 probably contained patients with both autoimmune hepatitis and chronic hepatitis C virus (HCV) infection.

We assayed serum levels of total IgG, IgM, IgA and IgG subclasses (IgG 1-4) in 50 patients (40 men; median age 35 years) with chronic hepatitis C and 25 healthy control subjects. All patients had positive serum anti-HCV antibody, positive serum HCV RNA, and histologically-proven chronic hepatitis. IgG, IgA and IgM were assayed by nephelometry (BN-II Analyzer; Behring Diagnostics, Marburg, Germany). IgG subclasses were assayed using human IgG subclasses enzyme immunoassay kit (Binding Site, Birmingham, UK). Median values were compared by using the Mann-Whitney U test.

IGM, total IgG, IgG1, IgG2, IgG3 and IgG4 levels were increased in patients with HCV infection when compared with the control subjects (Table).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Chronic HCV patients</th>
<th>Healthy controls</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgA</td>
<td>400 (375, 421)</td>
<td>387.5 (350, 450)</td>
<td>0.4</td>
</tr>
<tr>
<td>IgM</td>
<td>173 (160, 190)</td>
<td>127 (110, 150)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IgG</td>
<td>1395 (1350,1448.5)</td>
<td>1111 (1065, 1150)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IgG1</td>
<td>906.8 (836.7, 941.5)</td>
<td>722 (692, 747.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IgG2</td>
<td>365.5 (353.7, 379.5)</td>
<td>291 (279, 301)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IgG3</td>
<td>59.99 (58.1, 62.3)</td>
<td>47.8 (45.8, 49.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IgG4</td>
<td>62.8 (60.8, 65.2)</td>
<td>50 (47.9, 51.8)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Values as median (5th, 95th percentiles)

HCV infection is associated with cryoglobulinemia and occasionally with B-cell lymphoproliferative disorders. Andre and McQuilkan reported that the increase in gamma globulin is due to stimulation of numerous plasma cell clones by exogenous or endogenous antigens. A specific cytokine imbalance could prompt B cells to increase IgG production in patients with HCV infection, independently from liver damage.

Gonzalez-Quintela et al reported elevated IgG in these patients. Elevation of IgG1 and IgG2 level may be due to higher humoral response, and the lymphocytotoxic activity has been found mainly in these subclasses. These findings are similar to those in models of generalized enhanced B-cell responses. In many of these models elevated serum IgG subclass levels have been noticed to exist along with elevated specific antibody levels.

Serum IgM levels were higher in patients with chronic hepatitis C than in healthy controls, corroborating earlier findings. The elevated levels of IgM and IgG in HCV infection could be due to dysfunction of T cells, currently taken as responsible for virus persistence.

IgA levels were similar in patients and control subjects. Elevated IgA level has been reported in alcoholic liver disease. Watt et al reported elevated IgA in chronic HCV infection.

In conclusion, serum immunoglobulin pattern is altered in chronic hepatitis C infection. This may be useful in differentiation of these patients.

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