Steroid treatment of protracted cholestatic hepatitis A in a child with \( \beta \)-thalassemia

To the Editor,

Prolonged cholestasis is a rare atypical form of hepatitis A virus (HAV) infection that is characterized by serum bilirubin levels higher than 10 mg/dL and a prolonged jaundice (usually longer than 12 weeks). Patients with sickle-cell disease, uremia, and glucose-6-phosphate dehydrogenase deficiency may have increased risk of HAV infection (1). Moreover, different HAV genotypes (2) and polymorphisms within the hepatocanalicular transporters (3) are suggested to contribute to a more pronounced course. Recently, we encountered a 6-year-old girl with a prolonged cholestatic form of HAV infection. She developed mild hemolytic anemia as well. She was diagnosed with \( \beta \)-thalassemia trait by hemoglobin electrophoresis. Cholestasis due to HAV infection develops as a consequence of systemic and intrahepatic releases of endotoxin and pro-inflammatory cytokines (4). However, the cause of the prolonged course of the disease is unclear. In the present case, the HAV infection might have induced hemolysis because the patient had more severe hemolysis than expected with \( \beta \)-thalassemia trait. A similar mechanism may presumably be responsible for the protracted disease course in other cases of hemolytic anemias.

Although supportive treatment usually suffices, some cases of HAV infection may have an intractable course.

<table>
<thead>
<tr>
<th>TB (mg/dL)</th>
<th>DB (mg/dL)</th>
<th>ALT (U/L)</th>
<th>AST (U/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial</td>
<td>15.8</td>
<td>9.5</td>
<td>304</td>
</tr>
<tr>
<td>4th day</td>
<td>7.1</td>
<td>3.8</td>
<td>140</td>
</tr>
<tr>
<td>11th day</td>
<td>3.7</td>
<td>2</td>
<td>50</td>
</tr>
<tr>
<td>15th day</td>
<td>1.1</td>
<td>0.5</td>
<td>40</td>
</tr>
<tr>
<td>30th day</td>
<td>0.7</td>
<td>0.3</td>
<td>35</td>
</tr>
</tbody>
</table>

Table 1. Dramatic improvement in cholestasis and normalization of liver transaminase levels after steroid therapy

Pruritis may affect patients’ quality of life, and the prolonged course may potentially lead to significant anxiety in both the parents and the child. Cholestasis associated with this inflammatory process (4) can be improved or alleviated by steroids. Besides inducing a rapid decline in serum bilirubin levels and relief of pruritis, corticosteroids are known to shorten the duration of prolonged cholestasis (5). No consensus has been reached for the appropriate dosage and schedule of corticosteroid treatment for the cholestatic form of HAV infection in children. In the present case, a relatively low dosage of steroid (20 mg/day) led to the prompt resolution of cholestasis (Table 1). The steroid dose was then rapidly tapered and discontinued within 1 month from therapy initiation.

To our knowledge, this is the first report of the association between prolonged cholestatic hepatitis A and thalassemia trait. We considered that the patients who are prone to hemolysis or have hemolytic disease may be more susceptible to cholestatic form of HAV infection. Steroids can be used as a treatment for prolonged jaundice, as in the present case, to shorten the course of cholestasis and hence prevent complications of cholestasis. Steroid administration may have additional benefits on quality of life issues and lessen parental anxiety.

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REFERENCES


