Plasma biotin levels in children with burns and scalds

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SYNOPSIS The plasma concentrations of biotin were measured for up to 49 days after injury in nine children with burns and scalds which involved from 12% to 50% of the surface area of the body. Biotin levels below the minimum of the control range were observed in eight of the nine injured children at some stage during the episode.

In children with burns and scalds the blood concentration of folic acid may be depressed and there are indications that this has an effect on the biosynthesis of the nucleic acids (Barlow, 1970; Barlow and Wilkinson, 1970). Derangements in tryptophan metabolism in injured children suggest that there may be a deficiency of pyridoxine (vitamin B6). These coenzymes are important because they are essential to the growth of tissue. Biotin is a coenzyme involved in the metabolism of carbon dioxide and is essential for the activity of several carboxylases; it is also required for certain transcarboxylation and transcarbamylation reactions and is believed to have a more general effect in protein synthesis (Dakshinamurti and Litvak, 1970). Clinically, signs of biotin deficiency are mainly related to the skin, and because of its many activities as a coenzyme it was decided to see if normal blood levels of biotin were maintained in children suffering from burns and scalds.

Method

Biotin was measured in plasma by a microbiological assay using L. plantarum. The plasma was subjected to an overnight enzymic hydrolysis using purified papain (Boehringer) to free the biotin from protein (Baker et al, 1962), the hydrolysis being terminated by autoclaving at 1·05 kg/cm² for 20 minutes. The organism (NCIB 6376, Torrey Research Station, Aberdeen) was propagated in Bacto Microinoculum broth (Difco) and assayed in Bacto Biotin Assay Medium (Difco). Control plasmas were obtained from a group of 19 children (20 samples) whose blood had been collected for preoperative haematological investigation. These control children were of a similar age range to the nine children with burns and scalds.

Results

The control plasma biotin level was found to be 1·26 ± 0·38 nM/l (mean ± SD) (20 samples). The plasma levels in the injured children are shown in the table together with the details of the injury and the time of sampling.

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age</th>
<th>Area %</th>
<th>Biotin Level</th>
<th>Days after Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>9y</td>
<td>12 S</td>
<td>0·32</td>
<td>6</td>
</tr>
<tr>
<td>2</td>
<td>1y 10m</td>
<td>15 B</td>
<td>0·59</td>
<td>15</td>
</tr>
<tr>
<td>3</td>
<td>1y 1m</td>
<td>18 S</td>
<td>0·34</td>
<td>11</td>
</tr>
<tr>
<td>4</td>
<td>2y 2m</td>
<td>30 S</td>
<td>0·28</td>
<td>37</td>
</tr>
<tr>
<td>5</td>
<td>3y 7m</td>
<td>30 S</td>
<td>0·097</td>
<td>1</td>
</tr>
<tr>
<td>6</td>
<td>6y 6m</td>
<td>35 B</td>
<td>0·49</td>
<td>1</td>
</tr>
<tr>
<td>7</td>
<td>1y 6m</td>
<td>40 S</td>
<td>0·56</td>
<td>1</td>
</tr>
<tr>
<td>8</td>
<td>4y 3m</td>
<td>40 S</td>
<td>0·18</td>
<td>12</td>
</tr>
<tr>
<td>9</td>
<td>4y</td>
<td>50 B</td>
<td>0·34</td>
<td>1</td>
</tr>
</tbody>
</table>

Table Plasma biotin levels in nM/l

B = burn; S = scald

Control level 1·26 ± 0·38 nM/l (mean ± SD)

Range = 0·61–2·05 nM/l

Discussion

The biotin levels found in this group of controls had a range of 0·61–2·05 nM/l, somewhat wider...
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than that reported by Bhagavan and Coursin (1967) (0.86–1.79 nM/l).

In examining the plasma from the injured children, a check was made to ensure that the assay was not affected by the presence of antibiotics, some children being in receipt of antibiotics when the blood samples were taken. The mean biotin level of all the results shown in the table was significantly below the control value (P < 0.01). There did not appear to be any correlation between the extent of the injury and the blood level although this might be affected, in part, by the diet eaten during recovery and by the nutritional state of the patients before injury. Purified papain must be used for the hydrolysis because with a cruder preparation the assay is less reproducible.

It is conceivable that some dietary biotin is normally obtained from the gut flora and that this is suppressed in antibiotic therapy. While this could be an explanation for the falling levels in cases 4, 5, and 9, it certainly would not account for the low levels 24 hours after injury in cases 6 and 7, or for the low levels in children not on antibiotics (cases 1 and 3).

The evidence suggests that the low plasma biotin levels found in children with burns and scalds are due to the injury either through loss of the vitamin or through increased requirements for tissue repair.

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References


