Correspondence

transdermal loss of water and in our cases absorption of urea.

We hope that our cases and those of Dr Garty will
remind our colleagues that skin is not an impervious barrier.

References

1 Beverly DW, Wheeler D. High plasma urea concentration in
2 Garty BZ, Wiseman Y, Metzker A, Reiner SH, Nitzan M. Hypernatremic dehydrat
3 Garty KA, Bettley FR. Skin water loss and accidental hyperthermia in psoriasis, ichthyosis and

Vitamin D metabolites in idiopathic infantile hypercalcaemia

Sir,

We have further investigated two of the infants with idiopathic infantile hypercalcaemia described by Martin et
al1 and confirmed the presence of appropriate suppression of 1α hydroxylase activity in the presence of hypercalcaemia,
suggesting intact feedback mechanisms.

As vitamin D supplements (400 IU/day) were withdrawn and a low calcium diet begun in case 1 the serum calci
content fell. Concentrations of 1,25-dihydroxycholecalciferol (1,25(OH)2D) rose and 25-hydroxycholecalciferol (25(OH)D) fell due to the withdrawal of supplements (Table 1).

Table 1 Reaction of serum calcium and vitamin D concentrations to withdrawal of vitamin D supplements in case 1

<table>
<thead>
<tr>
<th>At diagnosis</th>
<th>2 weeks</th>
<th>4 weeks</th>
<th>7 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>25-OHD (ng/ml)</td>
<td>48.6</td>
<td>53.5</td>
<td>40.6</td>
</tr>
<tr>
<td>1,25(OH)2D (pg/ml)</td>
<td>31.0</td>
<td>36.0</td>
<td>47.0</td>
</tr>
<tr>
<td>Calcium (μmol/l)</td>
<td>3.16</td>
<td>2.38</td>
<td>2.47</td>
</tr>
</tbody>
</table>

25-OHD=25-hydroxycholecalciferol; 1,25(OH)2D=1,25-dihydroxycholecalciferol.

Calcium load tests, modified from the protocol of Barr
and Forfar,2 showed an abnormal response when per-
formed at 7, 13, 18, and 24 months in case 1 and at 22
months in case 2. Furthermore, 1,25(OH)2D concentra-
tions performed during the most recent load tests showed
appropriate suppression in the presence of hypercalcaemia (Table 2), suggesting intact homeostatic mechanisms.

Our assay is able to distinguish between 1,25(OH)2D2
and 1,25(OH)2D3 and it would seem that 1,25(OH)2D3
is more tightly controlled than 1,25(OH)2D2. The signifi-
ance of this observation is unknown as these metabolites
have not previously been measured in children with this condition. Further studies will be necessary to confirm our
findings.

Dr Martin suggested that the low concentrations of
1,25(OH)2D seen in the patients with hypercalcaemia
could be due either to appropriate suppression of 1α hydroxylase activity or to a reduced growth velocity. The results of the loading studies and the fact that the growth
velocity of our first child did not alter with correction of the hypercalcaemia suggests that the former is a more likely explanation. Whether there is poor control of production of 1,25(OH)2D2 and whether this is important in the aetiolo-
gy of the hypercalcaemia is an area for future study.

I thank Dr Barbara Mawer, Manchester University, for performing the assays.

References


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Table 2 Vitamin D metabolites during calcium loading in the two cases

<table>
<thead>
<tr>
<th>Fasting</th>
<th>Calcium loading (hours)</th>
<th>0.5</th>
<th>1</th>
<th>1.5</th>
<th>2</th>
<th>3</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1</td>
<td>Calcium (μmol/l)</td>
<td>2.44</td>
<td>2.77</td>
<td>2.66</td>
<td>2.79</td>
<td>2.79</td>
<td>2.79</td>
</tr>
<tr>
<td></td>
<td>1,25(OH)2D3 (pg/ml)</td>
<td>6</td>
<td>7</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>1,25(OH)2D2 (pg/ml)</td>
<td>63</td>
<td>55</td>
<td>53</td>
<td>53</td>
<td>53</td>
<td>53</td>
</tr>
<tr>
<td>Case 2</td>
<td>Calcium (μmol/l)</td>
<td>2.53</td>
<td></td>
<td>3.37</td>
<td></td>
<td></td>
<td>2.79</td>
</tr>
<tr>
<td></td>
<td>1,25(OH)2D3 (pg/ml)</td>
<td>21</td>
<td>21</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>1,25(OH)2D2 (pg/ml)</td>
<td>60</td>
<td>57</td>
<td>56</td>
<td>47</td>
<td>34</td>
<td>34</td>
</tr>
</tbody>
</table>

1,25(OH)2D3=1,25-dihydroxycholecalciferol.