Rickets in a white adolescent

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Summary
A case is presented to illustrate that vitamin D-deficiency rickets, although extremely rare, can still occur in white adolescents, provided the diet is sufficiently abnormal.

Case report
A 15-year-old Caucasian male presented with a 2-year history of pain in both knees aggravated by exercise. His diet consisted of chips, bread, sweets and fizzy drinks, while he refused to eat meat, fish, fruit or fresh vegetables because they made him ‘feel sick’. He was drinking milk and taking margarine on bread, however, and dietary assessment showed a normal vitamin D and calcium intake. Ultraviolet exposure had been adequate but since developing bone pains he had tended to remain indoors. He was tall for his age (1-73 m) having had a rapid growth spurt when 12 years old although he had not grown over the previous 2-3 years. At the age of 12 years his height would have been outside the 97th centile and when first seen he was the tallest boy in his class. There was no significant past medical history, and clinical examination was normal.

Investigations
April 1977
Serum calcium 1-6 mmol/l (normal 2-2-2-6 mmol/l).
Serum phosphorus 1-35 mmol/l (normal 0-8-1-4 mmol/l in adult).
Serum alkaline phosphatase 2218 u./l (normal <280 u./l in adult).
Albumin 45 g/l, globulin 22 g/l.
Plasma parathyroid hormone 1150 ng/l (normal <550 ng/l).
Plasma 25-hydroxy-cholecalciferol (25-HCC) 6-2 ng/ml (normal for West of Scotland 4-20 ng/ml).
Faecal fat, xylose absorption, jejunal biopsy normal.
Radiological skeletal survey normal.

Dietary assessment:
Calcium, 900 mg/day; vitamin D, 4 μg (160 i.u.)/day; protein 42 g (animal 16 g, vegetable 26 g)/day; recommended values – Ca 600 mg, vitamin D 2-5 μg (100 i.u.), protein 75 g/day (Department of Health and Social Security, 1973).

July 1977
Readmitted for further investigation. He had received a ward diet for 3 weeks during his previous admission and had been taking vitamin supplements (containing 10 μg (400 i.u.) vitamin D) for the 2 weeks before reassessment.

Serum calcium 2-3 mmol/l; serum phosphorus 1-7 mmol/l; serum alkaline phosphatase 1307 u./l; plasma 25-HCC 18-1 ng/ml.

A transilial bone biopsy was performed and the results of quantitative histology are shown in Table 1.

Discussion
The occurrence of vitamin D-deficiency rickets among the Asian population is well recognized (Dunningan et al., 1962; Holmes et al., 1973) and there have also been several recent surveys of white children which have reported biochemical abnormalities suggesting vitamin D deficiency (Dunnigan and Gardner, 1965; Cooke et al., 1973). However, symptomatic nutritional rickets in adolescent Caucasian children must now be rare.

Unfortunately a bone biopsy was not obtained in this patient at initial presentation but only after 3 months during which time he had received a hospital diet for 3 weeks, had been given dietary advice and had taken a vitamin D supplement for several weeks. However, quantitative histology of this bone biopsy (Table 1) demonstrated greatly increased osteoid volume suggesting rickets. The presence of a normal calcification front and a normal osteoid index, i.e. the ratio of osteoid volume to osteoid surface, are
Case reports

Table 1. Quantitative histology of bone biopsy (July 1977)

<table>
<thead>
<tr>
<th></th>
<th>Trabecular bone volume</th>
<th>Relative osteoid volume</th>
<th>Osteoid surface</th>
<th>Osteoid index</th>
<th>Resorption surface</th>
<th>Calcification front</th>
</tr>
</thead>
<tbody>
<tr>
<td>Value</td>
<td>(%)</td>
<td>(%)</td>
<td>(%)</td>
<td>(%)</td>
<td>(%)</td>
<td>(%)</td>
</tr>
<tr>
<td>15.5</td>
<td>11.6</td>
<td>51.8</td>
<td>22.4</td>
<td>8.9</td>
<td>81.6</td>
<td></td>
</tr>
<tr>
<td>Normal* (±1 s.d.)</td>
<td>21.9 (2.8)</td>
<td>2.3 (0.6)</td>
<td>22.9 (11.0)</td>
<td>20 (4.2)</td>
<td>4.0 (1.3)</td>
<td>83 (10)</td>
</tr>
</tbody>
</table>

* Meunier et al., 1976.

not in keeping with this diagnosis, but are consistent with recent rickets which is responding to treatment. The values for trabecular bone volume and resorption surfaces are consistent with his secondary hyperparathyroidism. The plasma 25-HCC on admission was low normal (6.2 ng/ml) but was considerably below the mean expected for adolescent Caucasians in this area (12 ng/ml) and is in keeping with rickets. Following a vitamin D supplement and a balanced diet there was a rise in 25-HCC (18.1 ng/ml) which corresponded with improvement in biochemical indices and disappearance of bone pain. There seems little doubt, therefore, that this boy had mild vitamin D deficiency rickets. While he ate a bizarre diet, the only significant abnormality was the low protein content. This may provide further evidence that non-mineral factors and in particular a low intake of animal protein are important in the control of 25-HCC and presumably 1,25-HCC production (Hunt et al., 1977; Robertson, Kelman and Dunnigan, 1977). A further factor which may have precipitated rickets in this case was the rapid growth spurt which had occurred 2 years previously.

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References


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