Vitamin-D deficiency rickets in Jamaican children

COLIN GEORGE MILLER and WINSTON CHUTKAN

From the Department of Paediatrics and Orthopaedics, University Hospital, University of the West Indies

The first report of vitamin-D deficiency rickets from Jamaica was made in 1958 (Bower, 1958); 2 7-month-old malnourished nonidentical twins with biochemical and radiological evidence of rickets were presented, and in both there was a rapid response to fairly small doses of calciferol. Vitamin-D deficiency rickets has nevertheless generally been considered a rarity in Jamaica (Ashworth and Waterlow, 1974) and Jelliffe (1971) stated that the disease is virtually nonexistent in the Caribbean. The apparent belief that vitamin-D deficiency rickets is rare in West Indians in general is supported by the fact that though the disease is common among Asian immigrants in the United Kingdom (Dunnigan et al., 1962; Felton and Stone, 1966; Richards, Sweet, and Arneil, 1968; Swan and Cooke, 1971; Ford et al., 1972), there has been only one report of rickets among West Indian children living in Britain (Benson et al., 1963).

In the 15-year period from 1954 to 1969 only 3 cases of vitamin-D deficiency rickets were recognized at University Hospital (including the 2 children referred to above). However, during the 5-year period, 1969–73, the authors have seen and treated 9 children with clinical, radiological, and biochemical findings consistent with vitamin-D deficiency rickets. We present the main clinical and laboratory findings in these patients, and discuss the possible aetiological factors.

Patients and methods

All 9 children were of dark complexion and of predominantly Negro extraction. The diagnosis of vitamin-D deficiency rickets was based on clinical, biochemical, and radiological evidence of rickets, the absence of overt renal disease, and the rapid and sustained response to fairly small doses of oral calciferol (vitamin D$_2$). Standard laboratory methods were used for most determinations. Xylose absorption and excretion was estimated by the method of Chanarin and Bennett (1962).

Results

Table I summarizes the main clinical and laboratory features on presentation. There were 6 boys and 3 girls ranging in age from 3 years 5 months to 8 years 9 months. All but 3 presented with deformity of the legs (Fig. a, b). 2 patients were seen in the casualty department with leg pain and upper respiratory tract infection, respectively, and one child was found to have rickets on routine examination at home. Only 3 cases were familial (2 sibs and a cousin) and these were the only children who had spent the early years of their lives in the city of Kingston. Age of onset was said to have been between 3 and 4 years in 5 cases. 2 children developed signs soon after beginning to walk (at approximately 24 months) while one child was said to have been well until age 6 years. The apparent late onset in most cases may have been misleading since abnormalities were usually gross at the time of presentation.

Serum calcium ranged from 8·6–11·1 mg/100 ml on admission, being within normal limits (9–11 mg/100 ml) in 7 of the 9 patients. Serum phosphorus levels ranged from 1·7 mg–6 mg/100 ml and were below the normal range (4–6 mg/100 ml) in 5 cases. Alkaline phosphatase was raised in all 9
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Clinical and laboratory findings in 9 cases of vitamin-D deficiency rickets

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Sex</th>
<th>Age first seen (yr) (m)</th>
<th>Presenting features</th>
<th>Calcium (mg/100 ml)</th>
<th>Phosphorus (mg/100 ml)</th>
<th>Alkaline phosphatase (K-A units)</th>
<th>Urea (mg/100ml)</th>
<th>Hb</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>8 9</td>
<td>Painful joints, genu valgum</td>
<td>10</td>
<td>4.3</td>
<td>100</td>
<td>22</td>
<td>9.4</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>4</td>
<td>Genu valgum</td>
<td>9</td>
<td>6</td>
<td>80</td>
<td>34</td>
<td>7.5</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>4 6</td>
<td>‘Wind swept legs’</td>
<td>11-1</td>
<td>4.6</td>
<td>70</td>
<td>26</td>
<td>9.9</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>4</td>
<td>Abdominal pain, genu varum</td>
<td>9.2</td>
<td>2.5</td>
<td>91</td>
<td>26</td>
<td>10.4</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>5</td>
<td>Pain in abdomen and legs</td>
<td>9.7</td>
<td>1.7</td>
<td>90</td>
<td>18</td>
<td>7.0</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>5 11</td>
<td>Genu varum</td>
<td>9.5</td>
<td>2.9</td>
<td>81</td>
<td>30</td>
<td>5.8</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>3 5</td>
<td>Slight genu varum on routine examination</td>
<td>10.5</td>
<td>3.5</td>
<td>130</td>
<td>19</td>
<td>9.8</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>4 9</td>
<td>Pain in legs, genu valgum</td>
<td>8.6</td>
<td>3.8</td>
<td>41</td>
<td>26</td>
<td>7.9</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>5 3</td>
<td>Difficulty in walking, genu valgum</td>
<td>9.3</td>
<td>5.1</td>
<td>81</td>
<td>36</td>
<td>8.0</td>
</tr>
</tbody>
</table>

Table I summarizes the treatment given and the response. 3 cases (1, 4, and 5) were given fairly large doses of calciferol daily (20 000 to 50 000 IU) over periods of from 2 to 8 weeks’ duration, in the mistaken belief that they were suffering from vitamin-D resistant rickets. No further vitamin D was added to their diet after it became apparent in each case, from the rapid biochemical and radiological response, that simple vitamin-D deficiency was the likely cause of their disease. All 3 continued to grow well and biochemical and radiological changes reverted to normal, having remained so on follow up. The other 6 cases healed on either no added vitamin D (Case 3) or on relatively small doses (400–500 IU daily). In one increased aminoaciduria was present on admission in 2 of the 3 children investigated.

Developmental delay was evident in 2 children (Cases 3 and 6) who had not walked until 2 years of age, while 2 others (Cases 2 and 4) walked at 15 and 14 months, respectively (average for Jamaican children 12–13 months). The 3 familial cases, living in Kingston, undoubtedly had inadequate exposure to sunlight (see below) and the parents of one other patient (Case 5) admitted that the child preferred staying indoors. The heights of all the children except Case 5 were below the 10th centile (Ashcroft and Lovell, 1966; Desai, Miall, and Standard, 1969) while 5 children were thin and obviously undernourished.

Anaemia (Hb <10 g/dl) was present in 7 cases. A history of pica (usually of dirt or sand) was common, and worm infestation, particularly with *Trichurus trichiura*, was proven in 5 of the 8 cases from whom stool was examined.

Table II summarizes the treatment given and the response. 3 cases (1, 4, and 5) were given fairly large doses of calciferol daily (20 000 to 50 000 IU) over periods of from 2 to 8 weeks’ duration, in the mistaken belief that they were suffering from vitamin-D resistant rickets. No further vitamin D was added to their diet after it became apparent in each case, from the rapid biochemical and radiological response, that simple vitamin-D deficiency was the likely cause of their disease. All 3 continued to grow well and biochemical and radiological changes reverted to normal, having remained so on follow up. The other 6 cases healed on either no added vitamin D (Case 3) or on relatively small doses (400–500 IU daily). In one
patient (Case 7) definite radiological and biochemical evidence of healing occurred on a ward diet estimated as containing only 200 IU calciferol daily, plus exposure to sunlight for two \( \frac{1}{2} \) hour periods each day. Surgical correction of gross deformities was carried out in 3 cases. Duration of follow-up has varied from 5 months to 3\( \frac{1}{2} \) years. No clinical, biochemical, or radiological evidence of recurrence has been detected, and in particular serum phosphorus has remained within the normal range in all 9 cases.

**Familial cases**

Two patients (Cases 3 and 4) were first seen 9 months apart. No family history of bone disease was admitted by the parents in either case but it was later noted that they had given the same address. A visit to the home was therefore made by one of the authors (C.M.) in May 1972. The children lived on the second storey of a high-rise apartment building in urban Kingston and at 3.00 p.m. their apartment was in semidarkness as the metal louvres were closed to keep out the afternoon sun. The two bedroom apartment was occupied by 4 adults (the mother of Case 3, her half-brother, the father of Case 4, and their mother) and 6 children. During the day the younger children were left in the care of their grandmother who did not allow them outside for fear they might fall from the balcony or stairway. The ‘family’ had moved to their present home in October 1968 when Case 3 was 2 years 3 months of age and his cousin, Case 4, 1 year 3 months. Case 3 had 2 sisters who were aged 3 years and 1 year, respectively, at the time of the removal, and a younger brother (Case 7) who was born shortly afterwards and had lived all his life in the apartment. Also living in the home was a younger female sib of Case 4 who was 2 years of age at the time of the visit.

All members of the family were examined clinically and appropriate laboratory investigations were carried out. Case 3’s eldest sib, aged 7 years at examination, had been attending school for over 2 years. She walked to school daily and no evidence of rickets was detected. His younger female sib aged 5 years, had been admitted to the Tropical Metabolism Research Unit (T.M.R.U.) of the University of the West Indies when she was 10 months of age suffering from severe malnutrition. After discharge she had been seen at regular intervals at the T.M.R.U., and on examination, apart from still being small for her age, she was well with no evidence of rickets. Examination of Case 3’s youngest sib (Case 7), however, showed that he was very short for his age and obviously undernourished. In addition there was slight bowing of his legs, bossing of the skull, and other general signs of rickets. He was therefore admitted to hospital for investigation and treatment as previously summarized. Examination of Case 4’s younger sib and of the adults living in the home showed no evidence of rickets.

**Discussion**

In view of the clinical, biochemical, and radiological features, there can be no doubt that all our 9 patients were suffering from rickets. The response to treatment with a normal ward diet and relatively small doses of vitamin D\(_3\) in most cases, and the continued normal growth and failure to
show evidence of recurrence after stopping therapy in the 3 patients given relatively large doses of calciferol (20 000–50 000 IU daily), indicate that simple vitamin-D deficiency was the cause of their disease.

Although vitamin-D deficiency rickets was apparently rarely recognized in Jamaica before 1969, nutritional rickets and osteomalacia have been reported from other parts of the world enjoying adequate sunshine for most if not all the year. Thus vitamin-D deficiency rickets and osteomalacia have been reported from Nigeria, India, Iraq, and Tehran (Antia, 1970; Vaishnava and Rizvi, 1971; Nagi, 1972; Salimpour, 1975). These authors all concluded that the likeliest cause of rickets and osteomalacia in their patients was a combination of dietary deficiency of vitamin D and lack of exposure to sufficient sunlight, the latter often due to the traditional habit of 'living in Purdah'.

The high incidence of rickets and osteomalacia among Asian immigrants in the United Kingdom has already been referred to. However, rickets and osteomalacia are seemingly rare in the large West Indian community living in Britain in spite of the fact that these people have on the average, much darker skins than Asian immigrants (Dent et al., 1973). Although it is not clear whether there has been a true increase in frequency of vitamin-D deficiency rickets in Jamaican children in recent years or whether the increase is merely apparent from better diagnostic awareness, vitamin-D deficiency rickets is certainly not now as uncommon as previously believed. This hospital receives patients referred from all parts of the island of Jamaica, though the majority of patients, live in the urban areas of Kingston and St. Andrew. It is therefore probably significant that only 4 of our cases had spent most of their early lives in the city. 3 of these patients, as previously shown, were close relatives and were living together under the same environmental conditions. 2 of them were obviously thin and underweight for their age and the third (Case 3) had a history suggestive of malnutrition in early life. In addition, one of his sibs had been treated at this hospital in the first year of life with severe malnutrition. There is therefore good circumstantial evidence that all of these 3 cases probably had an inadequate dietary intake of vitamin-D and as previously shown they had been exposed to inadequate sunlight for at least 2 years before being seen. An attempt was made to see whether overt rickets was common among other children living in high-rise apartment buildings in Kingston. Many adults living in these buildings were questioned and approximately 50 children were examined in the basic and primary schools serving the area, by a trained nurse attached to the Department of Paediatrics. Although many of the residents had noticed the deformities in the 2 older patients they knew of no other children with a similar disorder and no child with overt rickets was detected in school.

Malnutrition is common in Kingston yet overt rickets is rare, no doubt because the severely malnourished infant is not growing and thus, rarely develops overt rickets. Although older children who may be receiving a better but still inadequate calorie intake may show signs of rickets in association with overt malnutrition (Nagi, 1972), rickets seems to be rare among children in urban Kingston and St. Andrew. A possible explanation is that prolonged breast feeding is not practised in the urban community (Grantham-McGregor and Back, 1970) and most of the commercial milk preparations and liquid cow’s milk available in the city are fortified with vitamin D. In addition, the majority of the homes of the lower socioeconomic groups have little available space for children to play in and they are encouraged to get outside at an early age. Breast feeding on the other hand is still commonly practised in rural communities; weaning usually taking between 6 and 12 months (H. Fox, personal communication, 1974). Most children are then weaned onto sweetened condensed milk, or non-fortified fresh cow’s milk, both of which are relatively inexpensive and more readily available in the rural parishes. It is not surprising, therefore, that rickets may occur in these children even when apparently well nourished, if for some reason, they are not exposed to adequate amounts of sunlight. The parents of one of our patients admitted that the child for some unknown reason preferred to stay indoors rather than play outside. We were not, however, able to get a similar history in the remaining 5 cases and the exact cause of their condition particularly in the case of one, who was said to have been active and well until aged 6 years, remains obscure. The high incidence of undernutrition, resulting in slow growth and slow appearance of the deformities, could possibly explain the relatively late onset of the disease in these patients. The fact that none of these children had a positive family history of bone disease or deformity, and that the parents and several sibs were seen by us and found to be normal on clinical, biochemical, and radiological grounds would seem to exclude a genetic cause for their rickets. This is further supported by the fact that they remained well after stopping
therapy and showed no evidence of recurrence after returning to their home environment. Unleavened bread is not a staple part of the diet of Jamaican children and it seems unlikely that a high-phytate content in the diet could have played a part in the aetiology of their disease, especially as considerable doubt has been recently cast on the significance of phytate and calcium binding in the aetiology of rickets (Dent et al., 1973). The high incidence of pica and worm infestation may possibly play a part in the aetiology of rickets, but the incidence of both conditions is high in the general population, and in our experience clinical trichuriasis is not associated with signs of rickets.

We conclude that vitamin-D deficiency rickets can no longer be considered a rarity in Jamaican children. Children from rural parts of the country appear to be more susceptible than those living in the city. While a combination of inadequate dietary intake of vitamin D with lack of sufficient exposure to sunlight seems to be the cause in most cases, this fails to explain some cases. Preventive measures, such as giving cod liver oil to young children, should be routinely carried out, particularly in rural areas.

REFERENCES

Correspondence to Prof. C. Miller, Department of Paediatrics, University of the West Indies, Mona, Kingston 7, Jamaica.