CASE REPORT

Vitamin-D-deficiency rickets even with abundant sunlight - A case to highlight emerging problem

Tzer Hwu Ting, MRCPCH [1], Anand S Devnani, MS (Ortho) [2]

[1] Department of Paediatrics, University Putra Malaysia, [2] Department of Orthopaedics, University Putra Malaysia

SUMMARY

We describe a case of vitamin-D-deficiency rickets in a young child to highlight its existence in Malaysia where sunlight is abundant throughout the year. The child presented with deformity of both legs. He came from an educated urban family but remained indoors most of the time. Radiographs of knees and wrists showed changes of florid rickets. Low serum 25-hydoxyvitamin-D, high parathyroid hormone, normal serum phosphate and calcium levels, and normal renal function clinched the diagnosis of vitamin-D-deficiency rickets. He improved remarkably after treatment with oral Vitamin-D. We emphasise the importance of exposure to sunlight to prevent rickets.

KEY WORDS:
Vitamin-D deficiency, rickets, tropics, Asia

INTRODUCTION

Rickets is a disorder of bony deformity in children due to impaired mineralisation of growing bones. Normal bone mineralisation requires adequate amount of vitamin-D, calcium and phosphate. Nutritional rickets is due to deficiency of vitamin-D or calcium or both.[1] It is prevalent in resource-limited areas of Africa and Asia.[1] It is thought to be rare in Malaysia where resources are adequate and sunlight, which is the main source of vitamin-D, is abundant. There are no recent reports of vitamin-D-deficiency rickets in tropical South East Asia. We report a case of a young Malaysian child with vitamin-D-deficiency rickets coming from an educated urban family to highlight the presence of the problem.

CASE REPORT

A two-and-a-half-year-old Malay boy presented to the Orthopaedic clinic with bowing of both legs. Apart from delayed walking at two years other developmental milestones were normal. He was the third of four children of non-consanguineous parents. There was no family history of skeletal deformity. There was no history of delayed dentition or history of liver, gastrointestinal or renal diseases. He was breastfed till age two-and-a-half years, unlike his older siblings who had mixed breast and formula milk. He had three meals a day consisting of rice, chicken, fish and vegetables. Parents denied feeding problem. Both parents had formal education. Father was a clerk at a government department while mother was a housewife. The family lived in the capital city in an apartment with few windows and limited sunlight. The child was active and playful at home but seldom played outdoors due to social reasons, spending most time indoors with his mother.

Examination revealed a child small-for-age (weight 10.3kg, just below 3rd centile; height 76cm, well below 3rd centile on the National Center for Health Statistics growth chart) with normal head circumference (25-50th centile). His height was equivalent to the 50th centile of a 12-month-old child. There were bowing of both legs and swelling of both wrists and ankles (Figure 1). There was no chest deformity or rachitic rosary. Teeth were normal. Examination of other systems was unremarkable.

Blood investigations showed raised alkaline phosphatase 1158U/L, normal calcium 2.25mmol/L, phosphate 1.73mmol/L; normal liver and renal function. Intact parathyroid hormone was elevated 23.5pmol/L (normal 1.6-6.9pmol/L), 25-hydroxyvitamin-D level was low 26nmol/L. Radiographs of the lower limbs and wrists revealed florid rickets (Figure 2).

He was treated with oral vitamin-D, one-alfacalcidol 0.5 mcg daily (0.05mcg/kg/day) for three months as the preferred vitamin D2 (ergocalciferol) and D3 (cholecalciferol) were not available. Parents were advised on regular sun exposure and diet rich in vitamin-D with introduction of formula milk and increase intake of oily fish and eggs. Alkaline phosphatase dropped to 506U/L three months after treatment, 300U/L after six months and remained normal with vitamin-D containing multivitamin on follow-up. Serum calcium and phosphate remained persistently normal without supplementation. Radiographs six months after treatment showed almost complete resolution (Figure 2). The growth improved with the weight crossing up the third centile and height approaching the third centile six months after treatment. Bowing deformity of the legs improved spontaneously over the four years on follow-up.

DISCUSSION

Normal serum phosphate and calcium levels, reduced 25-hydroxyvitamin-D and raised parathyroid hormone levels clinched the diagnosis as vitamin-D-deficiency rickets in this child. Other forms of rickets were considered initially. Hypophosphataemic rickets was ruled out by the normal serum phosphate and raised parathyroid hormone levels. Vitamin-D-dependent rickets due to either 1α-hydroxylase deficiency or vitamin-D receptor defect were unlikely as these

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Corresponding Author: Tzer Hwu Ting, Department of Paediatrics, Faculty of Medicine & Health Sciences, University Putra Malaysia, 43400 UPM Serdang, Selangor, Malaysia
Email: tingth@upm.edu.my

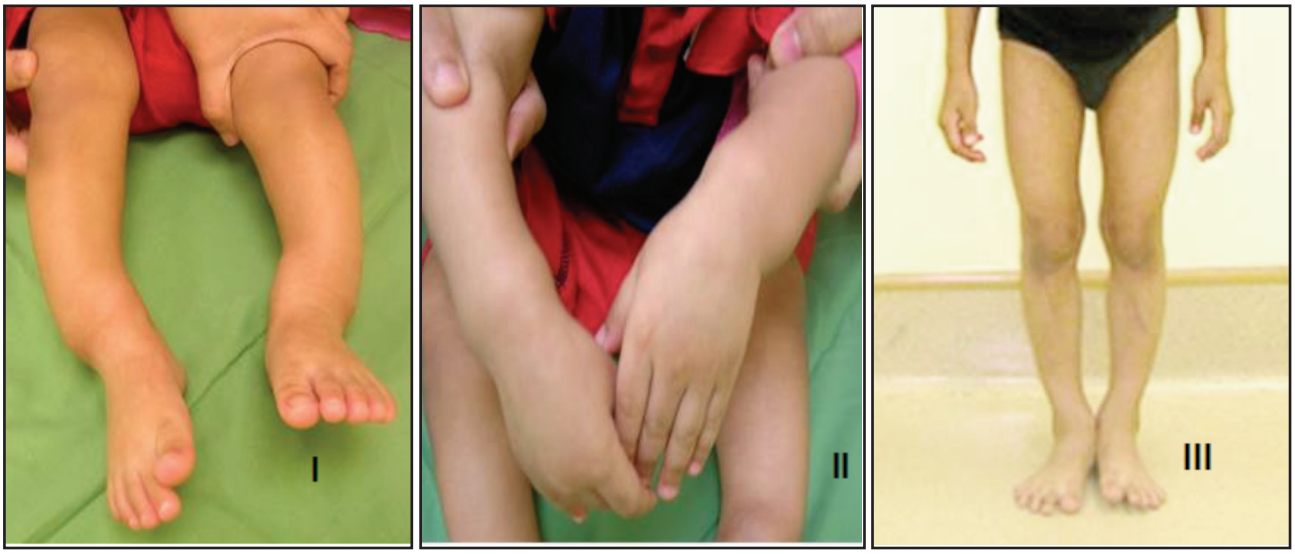

Fig. 1: Clinical photographs of legs (I) and wrists (II) showing bilateral varus deformity of distal tibiae and enlargement of both distal radii respectively and clinical photograph of legs four years later shows spontaneous correction of tibial deformity (III).

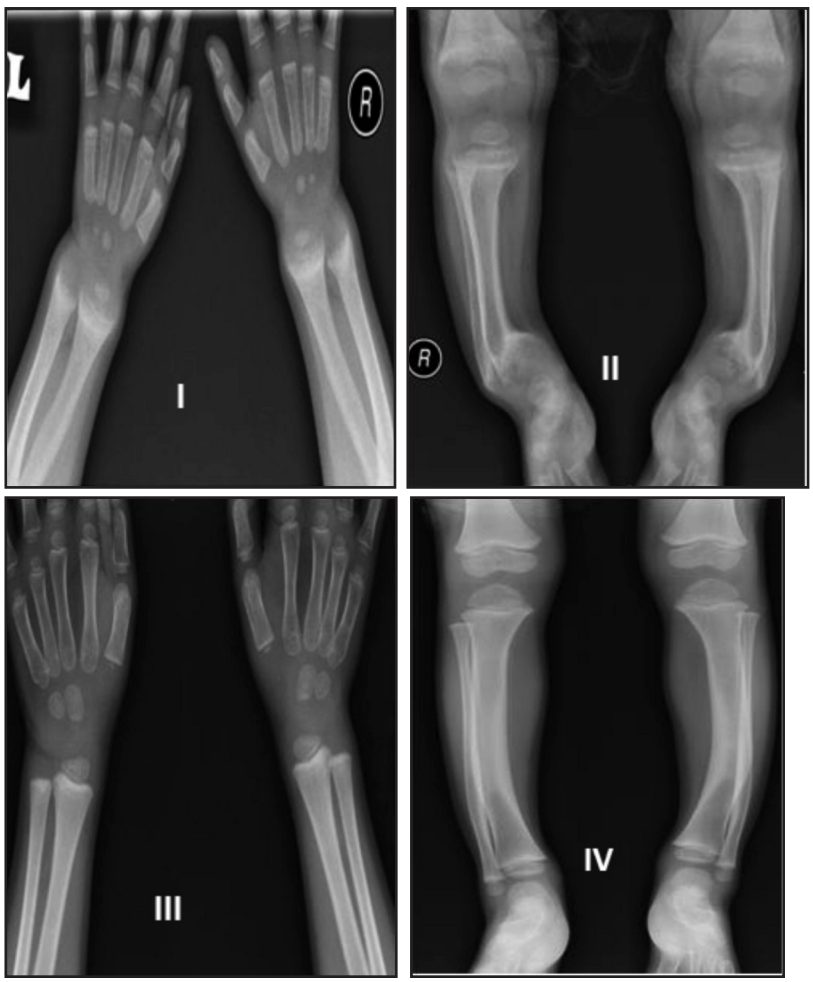

Fig. 2: Radiographs of wrists (I) and lower limbs (II) showing features of rickets with fraying, splaying and cupping of the metaphyses of long bones before vitamin D treatment and 6 months after treatment (III & IV).

conditions require long-term high doses of one-alfacalcidol or calcitriol for treatment. The remarkable biochemical & radiological responses with only three months of therapeutic vitamin-D dose and subsequent normal blood alkaline phosphatase, calcium and phosphate levels without vitamin-D treatment further confirm the diagnosis of vitamin-D-deficiency rickets.

With improved nutrition and public health measures, nutritional rickets is rare except in poorer countries in Africa and Asia.[1] Nutritional rickets is a disease not only of poverty but also due to cultural habits, social customs and attitudes. Vitamin-D-deficiency rickets has been reported in sunny countries in Middle-East due to cultural habits limiting sun exposure.[2] In richer North American and European countries, vitamin-D-deficiency rickets has been reported in immigrants with darker skin complexion.[2]

Rickets has been linked to vitamin-D deficiency for over 100 years.[1] The main source of vitamin-D is from the exposure to sunlight, only a small proportion is derived from dietary sources.[3] Serum level of 25-hydroxyvitamin-D is a marker of total body vitamin-D status. For children, vitamin-D deficiency is defined as serum 25-hydroxyvitamin-D level of less than 37.5 nmol/L, serum level above 50 nmol/L indicates vitamin-D sufficiency.[4] In contrast to reports of vitamin-D-deficiency rickets being generally associated with serum 25-hydroxyvitamin-D level below 12.5 nmol/L, this boy has florid rickets with 25-hydroxyvitamin-D level of 26 nmol/L.

Factors contributing to vitamin-D-deficiency rickets in this child are lack of sunlight exposure, skin pigmentation, extended breastfeeding for three years, vitamin-D-poor diet and possible maternal vitamin-D deficiency. Mother's vitamin-D status was unknown. The two older siblings did not have rickets as they attended school and played outdoors thus had adequate sunlight exposure. Moreover, they were not exclusively breastfed during infancy. Human breast milk contains little vitamin-D to meet the demands of a rapidly growing infant.[4] Breastfed infants need to obtain additional vitamin-D through sunlight exposure or vitamin-D supplementation to prevent rickets.[4]

This case highlights the existence of vitamin-D-deficiency rickets in sunny Malaysia. Vitamin-D deficiency is an emerging health problem. A study of 402 primary school children in Kuala Lumpur showed high prevalence of sub-optimal vitamin-D status with 35.3% having vitamin-D deficiency (≤37.5 nmol/L).[5] Urbanisation, air pollution, modern lifestyle of staying indoor, cultural habits and clothing practices cause inadequate sunlight exposure for vitamin-D synthesis on human skin although sunlight is abundant. Darker skin requires higher amount of sunlight to synthesize the same amount of vitamin-D. Diet alone is unable to provide sufficient vitamin-D for health. Vitamin-D deficiency has been associated with cancers, autoimmune diseases, infectious diseases, and cardiovascular disease in adults.[3]

Sunny climate does not assure vitamin-D sufficiency, exposure to sunlight is critical to prevent vitamin-D-deficiency rickets. Children should be encouraged to play outdoors.

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