Nutritional rickets and osteomalacia in school children and adolescents

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ABSTRACT

Objectives: To review experiences of nutritional rickets and osteomalacia in school children and adolescents at King Khalid University Hospital, Riyadh, Kingdom of Saudi Arabia.

Methods: Records of children and adolescents aged 6-18 years, seen at King Khalid University Hospital, Riyadh, Kingdom of Saudi Arabia, during the period January 1994 through to December 1999, who were diagnosed to have rickets or osteomalacia were reviewed. The diagnosis was based on clinical, biochemical and radiological data. Data extracted and analyzed included age, sex, presenting symptoms and signs, dietary history and sun exposure, blood count, bone profiles, renal and liver profile, and 25-hydroxy vitamin D3 and 1, 25 dihydroxy vitamin D3. Hand and wrist x-rays were carried out for all patients while bone density of lumbar spine and 3 femoral sites and bone scan were performed on the majority of patients.

Results: Forty-two children and adolescents (25 females and 17 males) were diagnosed. Their age ranged between 6-18 years with a mean of 13.5. Non specific symptoms, such as bone pain and fatigue were the most presenting symptoms, while skeletal deformities and fractures were the presenting symptoms in only 5 and 3 patients. Lack of direct sun exposure and poor calcium intake was evident. Bone profiles at the time of diagnosis revealed mean serum calcium of 2.1 mmol/L, range 1.5–2.3 (Normal=2.2-2.7), phosphorus 1.1 mmol/L, range 0.7–1.9 (Normal=1.4–2.1) and alkaline phosphatase activities of 1,480 U/L, range 834 – 2,590 (N=<600). Serum concentrations of 25-hydroxy Vitamin D were low (<10 mg/L) while that of 1, 25 Dihydroxy Vitamin D varied between low to normal (<10-45 ng/L). Bone density of the lumbar spine and 3 femoral sites were performed in 26 patients and showed markedly reduced values, while bone scan demonstrated a high uptake of tracer throughout the skeleton "super scan". Multiple stress fractures were evident in 8 children.

Conclusion: Although a community-based study to assess the magnitude of the problem is needed, it seems that rickets and osteomalacia of nutritional origin are not that uncommon and deserves special attention from all pediatricians and practicing physicians. They also suggested that further studies are needed to help understand the pathophysiology, and identify the contributing factors for the development of the disorder.

Keywords: Rickets, children.

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Rickets and osteomalacia are anatomically distinct conditions arising from the common event of mineral insufficiency. Rickets is often referred to as failure of mineralization of a growing bone, whereas osteomalacia indicates failure of a mature bone to mineralize.[1-7] In developing countries, nutritional rickets is still being seen in infants and toddlers in such a magnitude as to be considered a community health problem.[5,8–23] In older children and adolescents it is also reported with increasing frequency.[8,19, 24-25] In

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sunny Saudi Arabia, there is no precise clinical data on the magnitude of the disease in older children and adolescents. However, there is an impression fostered by the clinical experience that this is not uncommon. This article reviews our clinical experience with nutritional rickets and osteomalacia in school children and adolescents (6-18 years) at the King Khalid University Hospital (KKUH), Riyadh, Saudi Arabia over a 6 year period January, 1994 through to December 1999.

Methods. Children and adolescents aged 6-18 years who were seen at the KKUH during the period January 1994 through to December 1999, and confirmed to have nutritional rickets or osteomalacia were included. In this study, KKUH is the major teaching hospital of the King Saud University, Riyadh, Kingdom of Saudi Arabia (KSA) and provides primary, secondary and tertiary health care services to the local population and also receives patients referred from all over the country. The diagnosis was based on clinical, biochemical, and radiological data. Rickets and osteomalacia associated with Vitamin D dependency, renal or liver disorders malabsorption and drug therapies such as steroid or anti-convulsant medications were excluded by appropriate clinical and laboratory investigations.

The records of all patients were reviewed and data extracted for analysis included age, sex, presenting symptoms and signs, dietary history, sun exposure and medication intake as well as detailed physical examination. Laboratory investigations included complete blood count, renal, liver and bone profiles. Serum concentrations of 25 hydroxy Vitamin D (25 Hydroxy (OH) D) and, 1, 25 dihydroxy Vitamin D (1,25 (OH) 2 D) were measured commercially by Bio-Scientia Laboratory, Germany. Parathyroid hormone (PTH) level was carried out if indicated. Hand and wrist x-rays were carried out for all patients while other x-rays were carried out when appropriate. Bone density of the lumbar spine and 3 femoral sites and bone scans were performed in 26 patients as described before.[26] All patients were treated with oral Vitamin D preparations, ± calcium supplement with proper sun exposure.

Results. During the period January 1994 through to December 1999, 42 children and adolescents (25 females and 17 males) were diagnosed to have nutritional rickets or osteomalacia. Their ages ranged between 6 and 18 years with a mean of 13.5 years. Non-specific symptoms, such as bone pain and fatigue were the most presenting symptoms in 27 (64.3%) patients. Short stature in 6 (14.3%), while skeletal deformities and pathological fractures were the presenting symptoms in 5 (11.9%), and 3 (7.1%) patients. Only one patient presented with hypocalcemic tetany (2.4%). The dietary calcium intake was estimated to be as low as 100-300 mg/day. Milk consumption was generally low, with increased consumption of fast food and soft drinks. Sun exposure was negligible and the majority of activities were indoors. Bone profiles at the time of diagnosis revealed mean serum calcium of 2.1 mmol/L, range 1.5–2.3 (N=2.2-2.7), phosphorous 1.1 mmol/L, range 0.7–1.9 (N=1.4-2.1), and alkaline phosphatase activities of 1480 U/L, range 834-2590 (N=<600). Serum concentrations of 25-hydroxy Vitamin D (25 [OH)D] were <10mg/L (N, 10-40) and 1,25 dihydroxy vitamin D [1,25 (OH)2D] varied between <10-45 ng/L (N=15-50). Bone scan showed the feature of "superscan" in all patients and demonstrated multiple stress fractures in 8 **(Figure 1).** The mean and standard deviation (SD) of bone mineral density (BMD) for the lumbar spine were 0.53 ± 0.23 g/cm^2 (N, 0.91±0.11) with a z-score of –3.1, and for the femoral neck 0.55 ± 0.13 g/cm^2 (N, 0.86 ± 0.11) with a z-score of –2.8.

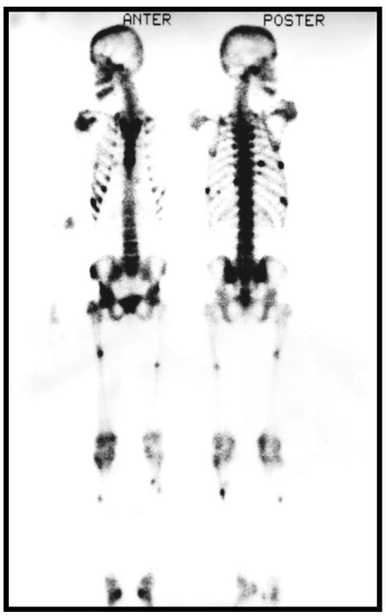

Figure 1 - Bone scan of a patient with osteomalacia demonstrating high uptake of tracer throughout the skeleton "superscan" with multiple focal lesion caused by pseudofractures.

Discussion. Nutritional rickets causes considerable disability among children. Though virtually eliminated from Europe and North America by the fortification of foods with Vitamin D, nutritional rickets remains prevalent in many parts of the world, including Africa and Asia.[5,9–23,27-28] Rickets has been ranked among the 5 most prevalent diseases among infants and toddlers.[5] During periods of rapid growth as in late childhood and adolescents, rickets or osteomalacia might appear as a problem in

association with a relative deficiency of Vitamin D or other nutrients necessary for bone mineralization.[1-2,5-8,22-24] This was supported by Shih[25] who reported a high incidence of osteomalacia in adolescent Chinese approximating 5%-15%, particularly in the later part of the cold season.[5]

In KSA, the overall prevalence of the disease is not known, however, the relatively high number of patients in this series readily supports the contention that this is not an uncommon disease. Furthermore, the majority of our patients were presented with non specific symptoms, which indicates the difficulty in making the clinical diagnosis in most of the less severe conditions. Although, low 25 hydroxy vitamin D [25(OH)D] levels do not necessary reflect a physiological deficiency in a particular patient.[29] Sedrani,[30] in a survey of the Vitamin D status of the Saudi population has shown that a very substantial proportion of school children and adolescents have inadequate plasma concentrations of 25 hydroxy vitamin D [25(OH)D].

Our patients had the classical biochemical and radiological characteristics of rickets or osteomalacia with abnormal bone density and low levels of 25 (OH)D and low to normal, 1 25(OH)2D indicating that they were Vitamin D deficient. Rapid change in lifestyle and nutritional habits in the young where fast food and soft drinks consumption is increasing can not solely explain this. The contribution of dietary Vitamin D to the total circulating pool of 25 (OH)D has been found to be almost negligible in relatively sunny countries.[31] Nevertheless, avoiding sun exposure, and spending more time in indoor activities could be considered as a major factor. Traditional dressing of women was found not to affect Vitamin D status as shown by Sedrani et al[32] who showed that 25(OH)D levels of males and females students were similar. Also, school girls from the age of 6-19 years did not show a drop in 25 (OH) D levels coinciding with the change to traditional dress taking place at puberty.[33] Therefore, we are uncertain as to whether Vitamin D deficiency alone or some other factors such as low calcium intake contribute to this. Calcium deficiency has been suggested as a cause of rickets in children with apparently good exposure to sunlight in Nigeria and Bangladesh.[22-23,34]

The majority of patient's diet is lacking in diary products and it is estimated that the average daily calcium consumption to be 100-300 mg which was well below the daily allowance of 800 mg recommended by the National Institute of Health (NIH).[35,36] Decreased bone mineral density and osteopenia might be a feature of decreased calcium intake in our children. Bone density of the lumbar spine and femoral neck were markedly reduced in our patient. El Desouki[37] has shown that bone mineral density in Saudi children and adolescents is lower compared to caucasian American. Furthermore, some of our patients had low serum levels of calcium and high normal serum phosphorous with markedly increased parathyroid hormone levels (PTH) which might indicate chronic calcium depletion.[38] Clements et al[39] has shown in animal studies that a low calcium diet promoted Vitamin D deficiency through increase in 1, 25 (OH)2D production in response to secondary hyperparathyroidism which caused hepatic conversion of Vitamin D to polar inactivation products that are excreted in the bile.

Finally, more detailed research needs to be considered to confirm our findings. Special attention should be given to preventive measure through education and appropriate supplements of Vitamin D and minerals. Outdoor activities with direct or indirect exposure to sunlight are to be encouraged and supervised.

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