

Bony manifestation of rickets in a sunny city - a case report from Yazd, IranMohammadhosain Afrand^{1*}, Vajiheh Modaresi²

¹. Medical Scientific Association, Student of Public health, Islamic Azad University, Yazd branch, Yazd, Iran.
hosain.afrand@yahoo.com

². Assistant professor of Pediatric Gastroenterology, Pediatric Department, Islamic Azad University, Yazd branch, Yazd, Iran. drmodarressi@yahoo.com

Corresponding Author:

Mohammadhosain Afrand, Medical Scientific Association, Islamic Azad University, Yazd branch, Yazd, Iran. Tel: +98.3517237280. Fax: +98.3515231421. Postal code: 8917145438, Email: hosain.afrand@yahoo.com

Abstract:

Rickets is disease that occurs in growing bones in which defective mineralization occurs in both the bone and the cartilage of the epiphyseal growth plate, resulting in the retardation of growth and skeletal deformities. Rickets is more common in areas with less sunlight. However, this case report presents a case of the bony manifestation of rickets with the intake of vitamin D supplements in Yazd, a city in central Iran that has sunshine almost every day. A patient was referred to an out-patient general pediatric clinic for deformities of the legs and growth disturbance, with his height far below the normal range. The changes that were most evident in his X-rays were the bowing of the long bones of the legs and forearms and the cupping of the wrist metaphyseal region. In summary, we present a patient with bony manifestation of rickets despite living in a sunny area and taking vitamin D supplements. Thus, it is important to remember that rickets is still a common disease among children in Iran. More studies of this issue should be conducted, including the identification of abnormal cases and rescheduling vitamin D supplementation programs.

Keywords: rickets, vitamin D deficiency, dietary Supplement

Additional Information for citing this article:

Title of Journal: Electronic physician; Abbreviated title of journal: Electron. Physician
doi: 10.14661/2014.728-733

Editorial information:

Type of article: Case report

Received: November.04.2013

Revised: 1st revision: November.17.2013; 2nd revision: November.24.2013; 3rd revision: December.20.2013

Accepted: December.22.2013

Published: February.01. 2014

© 2014 The Authors. This is an open access article under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs License, which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made.

1. Introduction

Rickets is the bony manifestation of altered vitamin D, calcium, and phosphorus metabolism in a child. The causes of rickets are varied and include nutritional deficiencies, especially poor dietary intake of vitamin D and calcium. There is another type of rickets known as non-nutritional rickets (1). The earliest reports describing the syndrome of rickets appeared in the English literature around 1650, suggesting the causes of rickets, osteomalacia, and the various metabolic defects in rickets and osteomalacia (2). By definition, rickets occurs only in children whose growth plates have not closed. Children with rickets are described as apathetic and irritable, often with a short attention span, and they are seemingly indifferent. They tend to sit for long periods of time due to the deformity associated with the bowing of the lower extremities (2, 3). Permanent skeletal deformities may appear in patients who have rickets. The relevant clinical findings are rachitic rosary (enlarged ends of the ribs), scoliosis, skull-craneotabes, bowing of the long bones (caused by loading), thickening of the wrists and ankles, greenstick fractures, genu varum, genu valgum, and laxity of the ligaments (2, 4).

Plain radiographic findings include physeal cupping and widening, fraying of the metaphysis, flattening of the skull and dorsal kyphosis (2, 5, and 6). Vitamin D deficiency is one of most common causes of rickets, and this deficiency

may be due to a lack of sun exposure and inadequate intake of the vitamin in food. In some cultures, whole body clothing prevents adequate sun exposure, e.g., the purdah system in Muslim communities. Socioeconomic status also may predispose people to vitamin D deficiency, and, thus, to having rickets due to many contributing factors, including the frequency and number of labor hours, malnourished infants, the economic situation of the family, living in cities where there is air pollution, living in high altitude areas, and the nutritional status of the mother. Vitamin D deficiency also is common at the end of the winter due to less sun exposure (7). Dietary vitamin D deficiencies can occur in children to different extents depending on ethnicity, skin pigmentation, and variations in the intake of supplements (8).

A Caucasian infant's vitamin D requirements are met by exposure to sunlight for 30 minutes per week, clothed only in a diaper, or for two hours per week when fully clothed with no hat. Asians require approximately threefold longer periods of exposure to sunlight because of the protective pigmentation in their skin, and Africans need six times the exposure of Caucasians (9). Although there are many osseous diseases that can occur in children, the goal of this research was to demonstrate that rickets continues to be a formidable, yet treatable, disease that should be considered in the differential diagnosis of children even though they are taking vitamin D supplements and getting adequate sun exposure.

2. Case Presentation

2.1. Clinical presentation

A young boy, 18 months old, was referred to an outpatient clinic because his parents had noticed that the child, at the age of 10 months, had knock knees and growth abnormalities.



Figure 1. Genus varum

2.2. Past history

The child was healthy at birth, and his birth weight was 2900 g. His occipitofrontal circumference (OFC) was 47 cm (25th percentile for his age). He was short in comparison to the norm, measuring just 73 cm in height (less than the 5th percentile for his age), and he weighed 12 kg. He was breastfed for the first six months, after which formula was added to his diet. He had received vitamin D supplements since he was born. The parents were of normal stature and build, and there was no consanguinity of rickets in the family history. The patient had no history of or features of malabsorption, chronic renal disease, or hepatic disease.



Figure 2. Widened wrists, bowing of arms

2.3. Physical examination

Upon examination, the patient had a bilateral genu varum (Figure 1), widened wrists, bowing of the arms (Figure 2), and short stature; however, his face, skull, spinal column, diaphragmatic insertion, dentition, and costochondral junctions were normal. His hearing, vision, mental acumen, and motor development were normal.

2.4. Laboratory findings

The results of biochemical investigations are summarized in (Tables 1, 2, and 3). His alkaline phosphatase, parathyroid hormone (PTH) was elevated. Calcium, phosphorus, serum BUN, vitamin D₃, and urine calcium, phosphorus and creatinine results were normal.

Table 1. Hematologic index

Lab test	Patient value	Normal value for age
WBC	7× 10(6) / lit	4.1-10.9×10(6) / lit
RBC	4.48 mil/UL	3.6-6.1 mil/UL
Neutrophils	36%	35-80%
Lymphocytes	22%	20-50%
Hgb	11.7 g/dl	10.5-14 g/dl
MCV	82 fl	78-101 fl (Female)
Platelet	305 ×103 / μL	140-450×103/μL

WBC: white blood cell; RBC: red blood cell; Hgb: hemoglobin; MCV: mean corpuscular volume

Table 2. Biochemical study

Lab Tests	Patient	Normal range
AST	37 IU/L	9-80 IU/L
ALT	23 IU/L	5-45 IU/L
ALPK	2260IU/L	150-420 IU/L
GGT	13.9 IU/L	5-32 IU/L
Ca (Total)	9.4	9-11 mg/dL
P	4.9	4-6.5mg/dL
PTH	75 pg/mL	10-65pg/mL
Vitamin D ₃	37.8 ng/mL	4.8-52.8 ng/mL
Na	137 mEq/L	135-147 mEq/L
K	4.4 mEq/L	4.1-5.3 mEq/L
INR	1.1	

AST: aspartate aminotransferase; ALT: alanine aminotransferase; ALPK: alkaline phosphatase; TG: triglyceride; GGT: gamma-glutamyl transferase; Ca: calcium; P: phosphorus; PTH: parathyroid hormone; Na: sodium; K: potassium INR: international normalized ratio

Table 3. Urine analysis

Lab test	Patient value	Normal value for age
Ca	2 mg/dl	Up to 4 mg/dl/24h
Cr	77.25 mg/dl	20-300mg/dl

2.5. Imaging

X-rays showed widening, cupping, and severe fraying of the metaphysical regions, knock knees with the patient in the standing position, and the cupping of the metaphysis were calcified to a greater extent than the metaphysis (osteopenia). These findings were suggestive of the bony manifestations of rickets.

2.6. Treatment

Treatment consisted of 10,000 IU of vitamin D₃ (cholecalciferol) daily and 600 mg of elemental calcium per day in three equal doses.

2.7. Patient follow-up

The treatment resulted in resolution of the biochemical and radiological abnormalities within three months. Skeletal deformities regressed completely after medical therapy.

2.8. Ethical consideration

Informed consent was obtained directly and in writing from the patient's parents for publication of this manuscript and any accompanying images. Consent for the recommended clinical course of action and research participation was freely given based on full prior knowledge of potential benefits and risks. This personal 'story' does not belong to the caregiver. It is the individual subject who possesses the original 'copyright.' This premise is reflected throughout our medical care system by carefully established privacy controls that protect against dissemination of

patients' information to third parties without the consent of the affected individuals (10). The subject of a case report must be aware of both the premise and the undertaking whenever possible and feasible.

A corollary to the 'copyright' was obtained to preserve confidentiality. Wherever possible, the case report could be reviewed by the patient's parents to permit editing or removal of any confidential or compromising material, thus overtly respecting the 'copyright.' The caregiver and/or author were constantly aware of the need for informed consent and the protection of confidentiality, and this awareness provided an additional level of protection. The Medical Ethics Committee of Shohadaye Kargar Hospital approved the study protocol.

3. Discussion

Tests for rickets should continue to be important elements of the differential diagnosis in children who present for evaluation with a bowed extremity, gait disturbances, diminished height, and failure to thrive. Radiography, radio nucleotide imaging, and measurements of blood urea nitrogen (BUN), creatinine, calcium, phosphorus, alkaline phosphatase, vitamin D₃, PTH, and a variety of urinary measurements, including calcium, are extremely useful aids in establishing the diagnosis of rickets and in elucidating the etiology of the disorder (11, 12).

Children with rickets as a result of vitamin D deficiency typically present with bowing of the lower extremities related to weight bearing when the child begins to walk. They have genu varum, genu valgum, coxavara, and short stature. Our patient presented with similar features. Cupping, flaying, fraying of metaphysis, and generalized osteopenia are characteristic features that are observed in a roentgenographic study. X-rays of the wrists of our patient had all of these features, along with delay in bone age. Vitamin D deficiency can occur even with adequate sun exposure (13). It can occur in people who consume foods that are not fortified with vitamin D or if there is intestinal malabsorption of vitamin D. There are very few foods that naturally contain vitamin D, and most of these are meat or fish, so they may not be acceptable in some cultures. Even though vitamin D is essential for bone health, especially in children, few foods are fortified with vitamin D. The fortification of milk with vitamin D is highly recommended in lactating and pregnant women, and, in developed countries where this process is performed, it has been successful in controlling this condition. Also, such countries have conducted many investigative studies of this issue, which we have not done throughout our country to educate people about preventive public health strategies and to document the use of vitamin D supplementation in conjunction with breast milk. So, routine fortification of milk and other food products with vitamin D should be considered. Also, we should ensure that healthcare professionals are aware of the risk of vitamin D deficiency and its consequences in children.

4. Conclusions

We presented a patient with bony manifestation of rickets despite vitamin D supplementation and living in a sunny area. We must remember that rickets is still a common disease in children in our country. Even though vitamin D supplementation has been recommended for all infants in Iran, especially those who are breastfed, this is not done in all parts of the country. Given that there are special genetic predispositions to have rickets, we recommend more studies of patients who have overt symptoms of vitamin D deficiency. We also recommend a new policy that includes improved fortification of foods with vitamin D and requires screenings of children in order to prevent the adverse consequences of low vitamin D, such bone deformities.

Acknowledgements:

The authors thank the parents of the patient for their valuable contribution to this study. The authors also acknowledge the Ali-EbneAbitaleb Faculty of Medicine at Islamic Azad University, Yazd Branch, Yazd, Iran, for their support of and contributions to this study.

Conflict of Interest:

There is no conflict of interest to be declared.

Authors' contributions:

Both of authors contributed to this project and article equally. Both authors read and approved the final manuscript.

References:

1. Pitt MJ. Rickets and osteomalacia are still around. *Radiol Clin North Am.* 1991; 29(1):97-118. PubMed PMID: 1985332. Epub 1991/01/01. eng. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/?term=1.+Pitt+MJ.+Rickets>

2. Behrman RE, Kliegman RM, Jensen HB, et al. Nelson Text book of Pediatrics, 17th ed. Philadelphia: WB Saunders; 2004.
3. Pettifor JM. Vitamin D &/or calcium deficiency rickets in infants & children: a global perspective. *Indian J Med Res.* 2008; 127(3):245-9. PubMed PMID: 18497438. Epub 2008/05/24. eng. Available from: <http://search.proquest.com/docview/195978999/fulltextPDF?accountid=45229>
4. Welch TR, Bergstrom WH, Tsang RC. Vitamin D-deficient rickets: the reemergence of a once-conquered disease. *J Pediatr.* 2000; 137(2):143-5. PubMed PMID: 10931400. Epub 2000/08/10. eng. Available from: <http://download.journals.elsevierhealth.com/pdfs/journals/0022-3476/PIIS0022347600729969.pdf>
5. Kottamasu SR. Metabolic Bone Diseases. In: Kuhn JP, Slovis TL, Haller JO, eds. Caffey's Pediatric Diagnostic Imaging. 10th ed. Philadelphia: Mosby ; 2004: 2242-53.
6. Do TT. Clinical and radiographic evaluation of bowlegs. *Curr Opin Pediatr.* 2001; 13(1):42-6. PubMed PMID: 11176242. Epub 2001/02/15. eng. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/?term=PMID%3A+11176242>
7. Chatfield SM, Brand C, Ebeling PR, Russell DM. Vitamin D deficiency in general medical inpatients in summer and winter. *Intern Med J.* 2007; 37(6):377-82. PubMed PMID: 17535381. Epub 2007/05/31. eng. Available from: <http://onlinelibrary.wiley.com/doi/10.1111/j.1445-5994.2007.01339.x/pdf>
8. Haddad JG. Vitamin D--solar rays, the Milky Way, or both? *N Engl J Med.* 1992; 30; 326(18):1213-5. PubMed PMID: 1557095. Epub 1992/04/30. eng. Available from: <http://www.nejm.org/doi/pdf/10.1056/NEJM199204303261808>
9. Binkley N, Novotny R, Krueger D, Kawahara T, Daida YG, Lensmeyer G, et al. Low vitamin D status despite abundant sun exposure. *J Clin Endocrinol Metab.* 2007; 92(6):2130-5. PubMed PMID: 17426097. Epub 2007/04/12. eng. Available from: <http://jcem.endojournals.org/content/92/6/2130.full.pdf>
10. Shevell MI. The ethics of case reports. *Paediatrics & child health.* 2004 Feb;9(2):83-4. PubMed PMID: 19654984. Pubmed Central PMCID: 2720465. Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2720465/pdf/pch09083.pdf>
11. Gartner LM, Greer FR. Prevention of rickets and vitamin D deficiency: new guidelines for vitamin D intake. *Pediatrics.* 2003; 111(4 Pt 1):908-10. PubMed PMID: 12671133. Epub 2003/04/03. eng. Available from: <http://pediatrics.aappublications.org/content/111/4/908.full.pdf>
12. Pettifor JM. Rickets and vitamin D deficiency in children and adolescents. *Endocrinol Metab Clin North Am.* 2005; 34(3):537-53. PubMed PMID: 16085158. Epub 2005/08/09. eng. Available from: <http://www.sciencedirect.com/science/article/pii/S0889852905000368>
13. Carvalho NF, Kenney RD, Carrington PH, Hall DE. Severe nutritional deficiencies in toddlers resulting from health food milk alternatives. *Pediatrics.* 2001; 107(4):E46. PubMed PMID: 11335767. Epub 2001/05/23. eng. Available from: <http://pediatrics.aappublications.org/content/107/4/e46.full.pdf>