

Frequency of Nutritional Vitamin D Deficiency in Children 6 Months to 5 Years of Age Presenting with Rickets at Civil Hospital, Karachi

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Abstract

Objective: To determine the frequency of Vitamin D deficiency in children presenting with rickets.

Methods: Descriptive case series was conducted at the department of Paediatrics Unit III, Dow University of Health Sciences and Civil Hospital Karachi in children 6 months to 5 years of age presenting with clinical rickets. Children taking vitamin D were excluded. The subjects fulfilling inclusion criteria were enrolled after informed consent. Blood samples were taken from each patient and were sent to the laboratory for vitamin D (25[OH] D) level. Serum level of vitamin D less than 20ng/ml was labeled as vitamin D (25[OH] D) deficiency rickets. The data was collected on pre-designed performa by the principal researcher. The data was analyzed using SPSS version 10.

Results: The age of enrolled participants was 1.5 ±1 year. Of 159 enrolled participants, 91 (57.2%) were male and 68 (42.8%) were female. Frequency of sun exposure of 2-3 days a week was 97 (61.01%) and using vitamin D fortified foods were 34 (21.38%). The frequency of vitamin D deficiency was 120 (75.47%) cases.

Conclusion: In this study the frequency of vitamin D deficiency in children 6 months to five years with rickets was 75%.

Keywords: Rickets, vitamin D, deficiency, sun exposure, children.

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Introduction

Vitamin D is a fat-soluble vitamin that occurs naturally in two forms: vitamin D3 (cholecalciferol); and vitamin D2 (ergocalciferol)¹. Vitamin D3 (cholecalciferol) can be synthesized in skin epithelial cells through conversion of 7-dehydrocholesterol by ultraviolet B radiation from the sun^{1,2}. Ergocalciferol is made in mushrooms and yeast and the synthetic form is often used to fortify foods and in dietary supplements. After ingestion vitamin D undergoes hydroxylation in two steps. Once in the liver, it gets converted to 25-hydroxy vitamin D (25[OH] D). The second hydroxylation occurs mainly in the kidney

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by the 1- α hydroxylase enzyme, to convert 25[OH] to the biologically active¹, 25 dihydroxyvitamin D (1,25[OH]2D)¹. Vitamin D has hormone-like activity and regulates the functions of over 200 genes and is essential for growth and development of the body especially regulation of calcium absorption from intestine as well as direct actions on bone resorption^{2,3}. Deficiency of vitamin D results in Rickets; a disease of growing bone, that occurs in children due to lack of mineralized matrix at the growth plates¹. Prevalence of rickets in South East Asia ranges from 15-18%⁴. The diagnosis of Rickets is mainly clinical which manifests as skeletal deformities such as widening of the wrists (66.6%), widening of the costochondral junctions (rachitic rosary 36.66%), anterior bowing of the tibia and femur (8.33%), softening of the cranial bones (craniotabes 8.33%)^{2,5}. Nutritional vitamin D deficiency remains the most common cause of rickets globally². Gen-

erally serum level of 25(OH) D less than 20ng/ml is considered as a vitamin D deficient state^{6,7}. Vitamin D deficiency is a worldwide problem. An Indian study showed that up to 82% of children were deficient in vitamin D (25[OH] D)⁸. Another study of infants of Pakistani, Turkish and Somali immigrants to Norway showed that 47% of infants had the deficiency⁹. While a study from Karachi, Pakistan showed that 55% of healthy breast-fed children and 45% of nursing mothers were deficient of Vitamin D (25[OH]D)¹⁰. Poor diet, cultural practices of the region for increased covering of the skin and poverty are some of the important reasons for vitamin D deficiency³. In our clinical practice in Karachi in general and at Civil Hospital in particular, we come across a lot of patients with clinical findings of vitamin D-deficiency i.e. Rickets. In fact 75-80% of patients who were admitted in the hospital due to other illnesses have an associated finding of rickets⁴. Therefore, rickets is a grave medical problem in pediatric community. Although various international studies have been done on this topic^{1,6,8}, however, in Pakistan very few studies have been conducted in this regards including a study done at Hazara⁵ which has addressed vitamin D-deficiency specifically. Therefore there is need of a study which should identify the magnitude of vitamin D-deficiency-a preventable disease; and thus this study will help and may be a reference for other forums to plan fruitful strategies to decrease the morbidity (like repeated infections due to chest deformities, fractures, growth problems) due to vitamin D-deficiency.

Subjects and Methods

This was a descriptive study i.e. a case series, conducted at the Department of Pediatric Unit-III, Dow University of Health Sciences & Civil Hospital Karachi from 1st July to 31st Dec, 2012. The sample size was 159 cases of Vitamin D (25 [OH] D) deficiencies in children was on average 72% [error margin of 7% at 95% confidence interval] the computed sample size was 159).

The inclusion criteria were children 6 months to 5 years of age, of both sex and children presenting with rickets (diagnosed clinically having skeletal deformities such as widening of the wrists; widening of the costochondral junctions-the rachitic rosary; anterior bowing of the tibia and fibula; and softening of the cranial bones-the craniotables). The exclusion criteria included children who received vitamin D supplements either orally or parentally within last three months. Sampling was non-probability purposive sampling. Cases were collected from patients presenting in paediatric outpatient department of Civil Hospital Karachi with rickets and fulfilling the inclusion criteria were enrolled for the study. After taking informed and written consent (on consent form) from parents or guardian, a data collection instrument (questionnaire) was filled for each patient. Blood samples were taken from each patient and were sent to laboratory for vitamin D (25[OH] D) level. If serum level of vitamin D came to be less than 20ng/ml then the child was labeled as having Vitamin D (25[OH] D) deficiency rickets. Children who were found to be deficient were given 6 lac I/U of Vitamin D3 I/M stat as part of Stoss therapy.

The data was analyzed; frequency and percentages were calculated for qualitative variables like sex, frequency of vitamin D (25[OH] D) deficiency. Mean standard deviation were calculated for quantitative variables like age and level of vitamin D (25[OH] D), duration of sun exposure. Stratification was done with regards to age, gender, dietary intake of vitamin D fortified foods and duration of sun exposure to see the effect of these on outcomes through Chi-square test and p-value. All data was analyzed by using SPSS version 10.

Results

A total of 159 children were enrolled during study period. The mean age of enrolled participants was 1.5 ± 1 year. Of 159 enrolled participants, 91 (57.2%) were male and 68 (42.8%) were female, male to female ratio of 1.3:1. Frequency of sun exposure (in the early morning) of 2-3 days a week was 97 (61.01%) and using vitamin D fortified foods

were 34 (21.38%) as shown in Table 1. The frequency of vitamin D deficiency was 120 (75.47%) cases. Stratified analysis of outcome based on age, sex, and duration of sun exposure and use of fortified food is summarized in tables¹⁻⁴. Among children of <2 years of age, 75.9% had vitamin D deficiency compared to 75% in children of ≥ 2 years of age (Table 2) and is statistically not significant ($p=0.52$). Among male children, 68.1% had vitamin D deficiency compared to 85.3% in female and is statistically significant ($p=0.01$). Among children exposed to sunlight daily, 66.1% had vitamin D deficiency compared to 81.4% in children exposed to sunlight 2-3 times a day and is statistically significant ($p=0.02$). Among children using vitamin D fortified food, 58.8% had the deficiency compared to 80% in children not using vitamin D fortified food which is statistically significant ($p=0.012$).

Discussion

Rickets in children has been identified as a persistent global health concern largely through published case series, retrospective chart reviews at local institutions and cross-sectional studies¹¹⁻¹⁴. Recent reports have noted cases not only from regions with more limited sunshine, such as New Zealand¹⁵, the United Kingdom¹⁶ and the United States¹⁴, but also from sunnier regions such as Africa¹⁵, Saudi Arabia¹⁶ and Australia¹⁷. Lack of sunlight exposure (even at low latitudes), breast-feeding, darker skin and recent immigration are the most common risk factors in these reports¹⁸. In our study the risk factors that were identified were male gender, inadequate sunlight exposure and inadequate use of vitamin D fortified food.

Table 1. Stratification of vitamin D deficiency by sex, duration of sun exposure and vitamin D fortified food

	Vitamin D Deficiency				Total	P-value
	Yes		No			
	Number	%	Number	%		
Sex						
Male	62	68.1	29	31.9	91	0.01
Female	58	85.3	10	14.7	68	
Duration of Sun Exposure						
Daily	41	66.1	21	33.9	62	0.02
2-3 days a week	79	81.	18	18.6	97	
Vitamin D fortified Food						
Yes	20	58.8	14	41.2	34	0.012
No	100	80	2	20	125	

Table 2. Stratification of vitamin D Deficiency by age

Age groups	Vitamin D deficiency				Total
	Yes		No		
	Number	%	Number	%	
<2 years	63	75.9	20	27.1	83
≥ 2 years*	57	75	19	25	76

* $p=0.52$

Holick⁶ and Lips⁷ considered cut off serum level of 25(OH) D less than 20ng/ml as a vitamin D deficient state. In a study conducted in India reported that up to 82% of children had 25 (OH) D deficiency⁸. In a study of infants of Pakistan¹⁹, Turkish and Somali immigrants to Norway showed that 47% of infants had 25(OH)D deficiency⁹. While a study from Karachi reported that 55% of healthy breast-fed children and 45% of nursing mothers were deficient of 25(OH)D¹⁰. Poor diet, cultural practices of the region for increased covering of the skin and poverty are some of the important reasons for vitamin D deficiency³.

In this study total 159 children with rickets were enrolled among which 120 (75.47%) were found to be vitamin D deficient. This is comparable to results of a study done at Peshawar in which 80% of rachitic children were vitamin D deficient²⁰.

The mean age of our study patients was 1.5 ± 1 years, which is nearly, the same age group seen in a study from Peshawar where 72.4% of children were below 24 months with females being predominantly affected²⁰. In our study males were slightly more frequently affected (57.2%) than females (42.8%), which is consistent with an Australian study in which 55% of vitamin D deficiency rickets patients were male²¹. Although no precise reason could be identified for this gender difference but can be due to under reporting in case of girls especially in our part of the world due to social norms.

Frequency of sun exposure of 2-3 days a week was 97 (61.01%) while other study showed that 44.7% children had the history of inadequate exposure to sunlight²⁰.

In our study lack of use of vitamin D fortified foods seemed to be major contributing factor in vitamin D deficiency. In this study 100 (80%) patients were found to be not taking vitamin D fortified foods whereas other study showed 60% children taking inadequate vitamin D²².

Munns CF, et al.²³ studied that duration of exclusive breastfeeding was inversely related to serum vitamin D levels in children <3 years of age while other Pakistani study documented that 60% of children had history of delayed weaning²⁰. Studies relating to vitamin deficiency in pregnant women and their newborns have been done, which indicate significant vitamin D deficiency²³. Risk factors for nutritional rickets in children less than 36 months has been studied²⁴. Hence these studies, along with our study indicate widespread vitamin deficiency in our population. Therefore, attention to routine screening and supplementation to prevent vitamin D deficiency are required. Public health campaigns are also needed to increase awareness of vitamin D deficiency in at-risk groups.

Conclusion

Vitamin D deficiency and associated rickets is emerging as a major public health issue worldwide, including in Pakistan. It is a significant problem which can cause long term morbidity thus creating a burden on the health sector. This is preventable by taking measures such as improving diet, adequate sun exposure and use of vitamin D fortified food. Therefore, public health campaigns to prevent identify and treat vitamin D deficiency, especially in high-risk groups, is essential.

Conflict of Interest

Authors have no conflict of interests and no grant/ funding from any organization.

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