Original Article

Frequency and Clinical Profile of Vitamin D Deficiency Rickets in Children: A Retrospective Study

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Abstract

Objective: This study aimed to determine the frequency and clinical profile of vitamin D deficiency rickets in children.

Methods: This retrospective descriptive study was conducted at King Edward Medical University from March 2024 to June 2025. We included 265 children aged 6 months to 12 years, diagnosed with vitamin D deficiency rickets based on clinical, biochemical, and radiological findings. Data regarding demographics, socioeconomic status, feeding practices, sunlight exposure, clinical presentation, laboratory parameters, and radiological features were retrieved from medical records.

Results: The mean age of affected children was 3.8 ± 2.1 years, with most cases occurring under 5 years of age (75.1%). Males (58.1%) were more frequently affected than females (41.9%). Low socioeconomic status (63%), exclusive breastfeeding without supplementation (55.1%), and inadequate sunlight exposure (69.1%) were significant risk factors. The most common clinical features were bowing of the legs (58.9%), widened wrists (54.3%), growth retardation (50.6%), and delayed motor milestones (42.3%). Hypocalcaemic seizures occurred in 10.9% of patients, predominantly among infants. Biochemical findings showed hypocalcaemia (7.9 ± 1.1 mg/dL), low phosphorus (3.4 ± 0.9 mg/dL), elevated alkaline phosphatase (1082 ± 354 IU/L), and markedly low vitamin D (11.7 ± 4.2 ng/mL).

Conclusion: Vitamin D deficiency rickets remains a prevalent paediatric health issue with significant clinical and developmental consequences. The findings of this study highlight the critical role of nutritional insufficiency, inadequate sunlight exposure, and socioeconomic disparities in perpetuating this disease.

Keywords: Vitamin D Deficiency, Rickets, Epidemiology, Malnutrition.

Introduction

The term" rickets is derived from the English word wrack, which means to twist. It is believed that nutritional rickets existed in earlier times; nevertheless, it has continued to persist even into the 21st century, and its causation is a multifactorial etiologic disease.[1,2] Since the primary source of vitamin D is ultraviolet light B with a wavelength of 290–315 nm, which converts 7-dehydrocholesterol in the skin to pre-vitamin D3, which then undergoes oxidative thermal isomerisation to cholecalciferol (vitamin D3), insufficient sunlight exposure remains the key factor related to the development of vitamin-D-deficient rickets.[3] Rickets is a pathology of growing bones that is manifested by the decalcification of the epiphyseal growth plates, resulting in deformities of the skeletal system and a variety of systemic complications.[4] Vitamin D deficiency rickets occurs due to insufficient vitamin D stores or metabolism.[5] It is still a leading health condition in children in low- and middle-income countries, where nutritional deficiencies persist, and access to fortified foods is limited. Although the use of nutritional science in the field of public health has contributed to the current low adoption of rickets in high-income countries, the disease has not been eliminated worldwide. Rather, it continues to be a major aetiology of morbidity and growth failure in at-risk children.[6]

Vitamin D is prominent in calcium and phosphate homeostasis, which are essential to healthy mineralization and bone development. The lack of vitamin D leads to impaired absorption of calcium in the intestine, secondary hyperparathyroidism, demineralisation of the bones, and typical degenerative alterations in the skeleton.[7] Children with vitamin D deficiency rickets have variable manifestations, including nonspecific signs and symptoms, such as irritability, delayed motor development, poor growth, and recurrent respiratory illness, as well as overt changes in the skeleton, such as genu varum (bowlegs), genu valgum (knock knees), frontal bossing, rachitic rosary, and widening of wrists. In severe cases, it can cause seizures or tetany due to hypocalcaemia, which further contributes to the morbidity of the patient when untreated.[8] prevalence of rickets varies in various geographical areas due to variations in climate, dietary patterns, culture, and interventions in the health system.[9] For example, children in South Asia, the Middle East, and sub-Saharan Africa are disproportionately affected by dietary factors, including low levels of vitamin D and calcium intake, cultural beliefs and restrictions about exposure to sunlight, and the absence of supplementation programs. Exclusively breastfeeding infants with no vitamin D supplementation are at an even higher risk of developing the condition

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than those in communities that are known to have vitamin D-deficient mothers. The problem has also been worsened by urbanisation and more indoor lifestyles, whereby people have less access to sunlight, even in sunny countries.[10]

In addition to its short-term skeletal effects, vitamin D deficiency rickets has more general effects on child health and growth.[11] Laboratory results indicate that the disease is linked with poor growth and stunting, leading to possible long-term physical disability and poor quality of life. In addition, the lack of vitamin D has been associated with vulnerability to infections, poor immune reactions, and a possible connection to chronic diseases at later stages of life.[12] These complications are costly to healthcare resources that are already at the limit in terms of their capabilities.[13]

Rickets still presents millions of children with problems globally, even though it is a condition that can be easily prevented. Some of the reasons behind its prevalence include the following. Some of the reasons behind its prevalence include the following: insufficient awareness by caregivers, poor screening and preventive measures, socioeconomic inequalities, and improper food fortification or supplementation agendas.[14] Furthermore, late diagnosis of the condition is not an exception, since the onset of the disease demonstrates slight symptoms that can be missed by parents and caregivers. The children develop classical skeletal deformities, and when the children present with these deformities, it may be too late because of the damage that has been done. The clinical profiling of affected children is crucial in determining the nature of these symptoms and risk factors that have been found to accompany vitamin D deficiency rickets.[15]

This study aimed to determine the frequency and clinical profile of vitamin D deficiency rickets in children.

Materials And Methods

A retrospective descriptive study was conducted at King Edward Medical University from March 2024 to June 2025. The study included 265 paediatric patients diagnosed with vitamin D deficiency rickets based on clinical, biochemical, and radiological features. The study included children aged 6 months to 12 years with a documented diagnosis of vitamin D deficiency rickets. Patients with complete medical records, including demographic details, biochemical reports, and radiological findings, were included. The diagnosis was confirmed by serum 25-hydroxyvitamin D levels < 20 ng/mL in conjunction with clinical or radiological evidence of rickets. The study excluded patients with rickets of non-nutritional origin, such as renal tubular acidosis, chronic kidney disease, or genetic/metabolic disorders (e.g., hypophosphataemic rickets), children with incomplete or missing records that prevented confirmation of the diagnosis, or patients who had already been treated with vitamin D supplementation before their first presentation to the hospital.

Data were collected from medical files and electronic health records using a structured data extraction sheet. The information gathered included age, sex, socioeconomic status, nutritional history, and sunlight exposure. Clinical details documented at presentation, such as bowing of the legs, frontal bossing, widening of the wrists, rachitic rosary, delayed motor milestones, and presence of hypocalcaemic seizures, were recorded. Laboratory parameters, such as serum calcium, phosphorus, alkaline phosphatase, and vitamin D levels, were extracted. Radiological findings, including metaphyseal cupping, fraying, and widening of the growth plates, were noted.

Data were entered and analysed using the Statistical Package for the Social Sciences (SPSS) version 26.0. Descriptive statistics were employed to summarise the findings. Categorical variables, such as sex, socioeconomic status, and presenting features, are expressed as frequencies and percentages. Continuous variables, such as age and biochemical parameters, are summarised as means with standard deviations. Associations between categorical variables were assessed using the chi-square test, whereas continuous variables were compared using an independent sample t-test. A p-value of <0.05 was considered statistically significant.

Results

Data were collected from 265 patients. The mean age of the children was 3.8 ± 2.1 years, with most cases occurring in the 6 months–2 years (38.5%) and 3–5 years (36.6%) groups. Males accounted for 154 cases (58.1%) compared with 111 females (41.9%). A majority came from low socioeconomic families (63.0%), while only 6.4% were from high SES. Exclusive breastfeeding without supplementation was reported in 146 children (55.1%), and 183 children (69.1%) had sunlight exposure of < 30 min daily. The most frequent clinical features were bowing of the legs (58.9%), widened wrists (54.3%), and growth retardation (50.6%), with hypocalcaemic seizures noted in 29 cases (10.9%). Radiographs showed classical signs of rickets. Metaphyseal cupping was present in 173 children (65.3%), while metaphyseal fraying was observed in 149 (56.2%). Widening of the growth plates was seen in 138 cases (52.1%), and osteopenia in 88 cases (33.2%). Pathological fractures were relatively uncommon, affecting only 14 children (5.3%), indicating that although deformities were widespread, structural bone failure occurred less frequently (Table 1).

Biochemical analysis revealed hypocalcaemia with a mean serum calcium level of 7.9 ± 1.1 mg/dL (reference, 8.5–10.5) and low phosphorus at 3.4 ± 0.9 mg/dL (reference, 3.5–5.5). Alkaline phosphatase was markedly elevated, averaging 1082 ± 354 IU/L compared to a normal level below 400 IU/L. Vitamin D levels were severely deficient, with a mean of 11.7 ± 4.2 ng/mL, far below the recommended cutoff of >20 ng/mL (Table 2).

Leg bowing was highly prevalent among children aged 3–5 years, affecting 76 of 97 patients (78.3%) compared with 32 of 102 infants (31.4%) and 48 of 66 older children (72.7%), with $p < 0.001$. Widened wrists were most frequent in infants (64.7%), followed by 53.6% in the 3–5-year-old group and 39.4% in children aged 6–12 years ($p = 0.012$). Hypocalcaemic seizures were significantly more common in infants, occurring in 19 of 102 (18.6%) compared with 7 of 97 (7.2%) and only 3 of 66 (4.5%) older children ($p = 0.004$) (Table 3).

Growth impairment was evident in the cohort. The mean height-for-age Z-score was -2.1 ± 0.7, with 141 children (53.2%) meeting the criteria for stunting. The weight-for-age Z-score averaged -1.9 ± 0.8, and 126 children (47.5%) were underweight. Among infants under two years of age, the mean head circumference was 44.2 ± 2.9 cm compared to the normal range of 46–49 cm, with 68 children (25.7%) below the expected range (Table 4).

Table 1. Demographic Characteristics of the study population (n = 265)

Variable	Mean ± SD/Frequency (%)
Age (years)	3.8 ± 2.1
Age group	
6 months–2 years	102 (38.5)
3–5 years	97 (36.6)
6–12 years	66 (24.9)
Gender	
Male	154 (58.1)
Female	111 (41.9)
Socioeconomic status	
Low	167 (63.0)
Middle	81 (30.6)
High	17 (6.4)
Feeding practices	
Exclusive breastfeeding (without supplementation)	146 (55.1)
Mixed feeding	92 (34.7)
Adequate complementary diet	27 (10.2)
Sunlight exposure	
<30 minutes/day	183 (69.1)
≥30 minutes/day	82 (30.9)
Clinical Feature	
Bowing of legs (genu varum/valgum)	156 (58.9)
Frontal bossing	98 (37.0)
Widened wrists/ankles	144 (54.3)
Rachitic rosary	87 (32.8)
Delayed motor milestones	112 (42.3)
Irritability/weakness	76 (28.7)
Hypocalcemic seizures	29 (10.9)
Growth retardation/stunting	134 (50.6)
Radiological Feature	
Metaphyseal cupping	173 (65.3)
Metaphyseal fraying	149 (56.2)
Widening of growth plates	138 (52.1)
Osteopenia	88 (33.2)
Pathological fractures	14 (5.3)

Table 2. Biochemical Profile and Radiological Findings in the study population (n = 265)

Parameter	Mean ± SD	Reference Range
Serum calcium (mg/dL)	7.9 ± 1.1	8.5–10.5
Serum phosphorus (mg/dL)	3.4 ± 0.9	3.5–5.5
Alkaline phosphatase (IU/L)	1082 ± 354	<400
25-hydroxyvitamin D (ng/mL)	11.7 ± 4.2	>20

Table 3. Association of Clinical Features with Age Groups (n = 265)

Clinical Feature	6m–2y (n=102)	3–5y (n=97)	6–12y (n=66)	p-value
Bowing of legs	32 (31.4%)	76 (78.3%)	48 (72.7%)	<0.001
Widened wrists/ankles	66 (64.7%)	52 (53.6%)	26 (39.4%)	0.012
Hypocalcemic seizures	19 (18.6%)	7 (7.2%)	3 (4.5%)	0.004

Table 4. Growth Parameters in Children with Rickets (n = 265)

Parameter	Mean ± SD	Normal Range	Frequency of Abnormal (%)
Height-for-age Z score	-2.1 ± 0.7	> -2 SD	141 (53.2)
Weight-for-age Z score	-1.9 ± 0.8	> -2 SD	126 (47.5)
Head circumference (cm, <2y)	44.2 ± 2.9	46–49	68 (25.7)

Discussion

This retrospective study analysed the frequency and clinical profile of vitamin D deficiency rickets in 265 children and provided a comprehensive overview of its demographic, nutritional, clinical, biochemical, and radiological features. The results indicate the persistence of the burden of this preventable disorder among paediatric patients, especially in low-income neighbourhoods. The average age of presentation within this cohort

was 3.8 years, and most of the instances fell within the 6-month to 5-year age category. In line with this study, previous research has indicated that the most susceptible stage is infancy and early childhood because the skeleton develops at a high pace and requires increased amounts of vitamin D and calcium. The minor male tendency observed in this study has also been reported by others; however, some literature reveals that sexes are equally susceptible, suggesting that sociocultural differences, such as child-rearing styles, may be a possible underlying factor to this phenomenon.[16]

In this study, socioeconomic status and feeding practices were identified as critical determinants of rickets. Over 60% of the affected children were from low-socioeconomic families, and more than half of them were exclusively breastfed with no vitamin D supplementation. Lack of adequate sunlight exposure was also reported in almost 70% of the cases. These results are in line with those of previous studies that have specifically highlighted that low levels of outdoor activity, low maternal vitamin D levels, and the absence of supplementation policies result in a significant increase in the risk of rickets.[17] These findings in this cohort, with an association between low socioeconomic status and lack of sunlight exposure, point to the underlying relationship between poverty and malnutrition and the environment in perpetuating the disease.[18] The most common clinical findings were bowing of the legs and widening of the wrists and ankles, followed by growth retardation and frontal bossing.[19] This spectrum reflects that of past studies, which defined skeletal deformities as markers of liquidity-deficient vitamin D rickets. Nonetheless, the fact that a relatively high percentage of children had delayed motor milestones (42.3%) and irritability/weakness (28.7%) suggests that, even though spinal deformities were not prominent, nonskeletal manifestations were frequent and may precede the manifestation of overt deformities. Notably, almost 11 percent of children were reported to have hypocalcemic seizures, with a higher incidence rate among infants.[20] The vitamin D level concentration of 11.7 ng/mL in this research study is very low, indicating a severe deficiency. These results agree with those of previous investigations, in which hypocalcaemia and elevated alkaline phosphatase are valid indicators of active rickets. The abnormalities mostly included metaphyseal cups and fraying, as supported by radiological findings. The low rate of pathological fractures (5.3%) in this group implies that several cases of advanced rickets did not show structural failure of bone to a considerable extent.[21] This confirms the findings of existing studies, which have shown the role of chronic vitamin D deficiency in restricting linear growth. The fact that infants with rickets also have a diminished head circumference proves that it may cause more than skeletal deformation in terms of compromising overall development. Moreover, similar conditions, including repetitive respiratory infections and developmental delays, were present in nearly one-third of the patients.[22] In past research, such a relationship has been identified with vitamin D deficiency being connected to poor immunity and delayed neurodevelopment. In the age-based analysis of clinical features, bowing of the legs was common in the age category of 3–5 years, whereas hypocalcaemic seizures were more common in the infant age categories. Past studies have promoted the development of rickets with age and have focused on early diagnosis before the changes to the skeleton become irreversible.[23] In this research, some limitations should be mentioned. It was retrospective in nature and was based on the medically documented previous records; thus, missing or incomplete data in some variables were bound to be present, and this could also influence the results. The research was carried out in a single centre, which could reduce the publication of the findings to populations with varied demographic or cultural backgrounds. In addition, possible confounding elements, such as maternal vitamin D status, amount of calcium intake in the diet, seasonal differences, pain exposure to sunlight, and genetic dispositions, were not measured; thus, they might have affected the clinical picture of rickets.

Conclusions

Vitamin D deficiency rickets remains a significant health problem among children, particularly in the early years of life when growth demands are highest. This study demonstrated that most cases occurred in children under five years of age, with a predominance in males, and were strongly associated with low socioeconomic status, exclusive breastfeeding without supplementation, and poor sunlight exposure. Clinical manifestations ranged from classical skeletal deformities, such as bowing of the legs and widened wrists, to systemic features, such as delayed motor milestones, growth retardation, and hypocalcaemic seizures. Biochemical abnormalities, including low calcium and phosphorus levels, high alkaline phosphatase levels, and markedly reduced vitamin D levels, along with radiological changes, confirmed the diagnosis in all cases.

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