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Nutritional Rickets in Ekiti, Southwestern Nigeria: Investigating the Roles of Calcium and Vitamin D

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Abstract

Background: Nutritional rickets remains a common cause of skeletal deformities among children in developing countries. While vitamin D deficiency is traditionally implicated, evidence from tropical regions with abundant sunlight suggests calcium deficiency may be more prominent.

Objectives: This study evaluated the relative roles of calcium and vitamin D in children with rickets in Ekiti, Southwestern Nigeria.

Methods: A descriptive comparative study was conducted involving 32 children with clinical features of rickets and 32 age- and sex-matched controls without rickets at Ekiti State University Teaching Hospital, Ado-Ekiti. Sociodemographic and clinical data were collected, and biochemical analyses included serum calcium, phosphate, alkaline phosphatase (ALP), vitamin D, total protein, albumin, and renal function tests. Radiographs of wrists and knees were obtained for affected children. Independent sample t-tests compared mean biochemical values, while Chi-square tests assessed associations between variables. Statistical significance was set at $p < 0.05$.

Results: The mean age of affected children was 3.06 ± 0.13 years (range 2–5 years). Bilateral genu varum was the most common presentation (31.3%). Hypocalcaemia was observed in 87.5% of cases and elevated ALP in 75.0%, whereas 87.5% had normal serum vitamin D levels. Significant differences between cases and controls were found for serum calcium (p = 0.02) and ALP (p = 0.01), but not for vitamin D or protein levels. Renal parameters were largely normal.

Conclusion: Calcium deficiency appears to be the predominant biochemical abnormality in children with rickets in this tropical setting, despite adequate sunlight exposure and largely normal vitamin D levels. Emphasis on calcium assessment and supplementation is essential for effective management and prevention.

Keywords: Nutritional rickets; calcium deficiency; vitamin D; alkaline phosphatase; tropical region; Nigeria.

Introduction

Rickets is a word that originated from the Greek *rhakhis*, meaning spine, and has been associated with a childhood disorder that also includes osteomalacia in adulthood. Bony lesions indicative of rickets has been documented in *Homo sapiens loquens* from ancient times, with reports from the first to second centuries of the Common Era serving as evidence of its existence, a clear description of the condition emerged in the mid-seventeenth century (1). Anatomy (histopathology) contributed to its understanding through autopsy reports, such as those by Schmorl, who found evidence of rickets in 96% (214 of 221) of the cadavers, demonstrating its widespread occurrence in the in 20th century (2). Experimental animal studies also played a role in establishing the chemotherapeutics of calcium and heliotherapy in the aetiopathogenesis of rickets; subsequently, McCollum discovered vitamin D as a treatment (3,4).

The various aetiologies of rickets are summed up under two categories: congenital and acquired. Primary causes are related to: vitamin D deficiency or dependent (type I or II); hypocalcaemia or chronic renal failure; and hypophosphataemia (congenital vitamin D resistant, autosomal dominant or autosomal recessive) (5,6). Secondary causes are related to disease conditions like tumour-induced osteomalacia, malabsorption, McCune-Albright syndrome, epidermal nevus syndrome and Dent's disease (7,8). Other predisposing factors are: dark skin, too little sunlight exposure, exclusive breastfeeding without vitamin D supplementation, celiac disease, and certain genetic conditions (9–11).

Heliotherapeutically, ultraviolet light from the sun converts the skin ergosterol to an inactive form of vitamin D, which undergoes some enzymatic reactions in the liver and kidneys to the active state. Another source of vitamin D is dietary intake through the gastrointestinal system. The active form of vitamin D helps the absorption of dietary calcium in the alimentary system and the reabsorption of calcium from the kidneys. Failure of this normal process results in hypocalcaemia with attendant skeletal deformities (usually in the head, thorax and limbs) and neuromuscular symptoms (muscle weakness, hyperexcitability). The physiological underlying mechanism of skeletal deformities involves insufficient calcification of the zone of calcification of the physeal plate, and this implies that rickets is a childhood disease, while the equivalent in adults (with fused physes) only results in softening of the bones (osteomalacia) (12). The diagnosis of rickets is based on clinical evaluation; imaging and laboratory investigations to confirm the diagnosis, knowing the severity and type/aetiology and treatment requirement, which could be dietary replacement, limb splintage to relieve progressive painful deformities and or surgical correction of residual deformities after adequate correction of biochemical deficiencies.

There are many researches works across the globe, with panoptic causes of rickets proving the environmental, religious belief, socio-cultural and complexional influence on the disease entity (13–17). Environmental factors are linked to the degree of sunlight exposure in the production of vitamin D necessary for absorption of calcium and its metabolism. The religious sect keeping wives and children in an indoor system, locally called 'Puda', may be depriving young growing children of adequate sunlight exposure. This is akin to rich families living in a glass house. Socio-cultural has to do with dietary habits, being influenced by Poverty, Ignorance and Disease (PID), in nourishing the growing child with the right diet at the right time and allowing compliance with medical treatment appropriately. There is adequate sunlight exposure amongst the blacks and whites in Africa and other continents alike along the equator.

The consequences of what happens to patients who were denied medical treatment, and the outcome of good and prompt treatment, were well illustrated in a Saudi Arabian study (18). The active form of rickets with severe painful deformity might benefit from surgical correction, and, thereafter, medical adjuvant therapy continues (19). The Indolent form of rickets with deformity would require correction of the biochemical deficiencies before intervention; in contrast, the burnt-out rickets with deformity in the older children might be treated surgically with any of the various corrective operations (20).

In light of the various causes of rickets observed globally, this study aimed to identify the predominant cause of rickets specifically in Ekiti, Southwestern Nigeria. It sought to achieve several objectives: to ascertain the main aetiological

factors contributing to rickets in this region; to establish the diagnostic methods employed; to evaluate the renal health of patients diagnosed with rickets; and to assess any related protein deficiencies. This research is notable for being the first of its kind in this locality, based on an extensive review of existing literature and online resources.

Materials and Methods

Study design

This study was a descriptive comparative analysis conducted at Ekiti State University Teaching Hospital, Ado-Ekiti, Nigeria, focusing on 32 paediatric patients exhibiting clinical features of rickets. These were compared with 32 paediatric patients without clinical features of rickets, who presented with non-endocrine-related pathology. Care was taken to ensure that both groups were closely matched in terms of age and sex, serving as a control group for the analysis.

Participants' selection and data collection

Consecutive paediatric patients who presented at the Orthopaedic clinic with clinical features of rickets; swollen wrist, angular knee deformities and rickety rosary and have not started calcium or vitamin D supplements were recruited into the study group after obtaining an informed consent. Closely matched paediatric patients with no clinical features of rickets and no endocrinology-related pathology were recruited into the control group after obtaining informed consent from the parents or guardians. During the first clinic visit, after thorough evaluation, information regarding their sociodemographic profiles and clinical features was obtained and recorded using a structured proforma. Blood samples were collected to analyze serum levels of Vitamin D, Calcium, Phosphate, Alkaline Phosphatase, Total Protein, Albumin, as well as Serum Electrolytes, Urea, and Creatinine from both the study and control groups. The authors bore the cost of analysing the blood samples. Additionally, X-rays of both knees and wrists (anteroposterior and lateral views) were requested for the study group as part of the standard evaluation protocol for rickets. Biochemical laboratory results and X-ray findings were documented within the proforma. Patients who had previously received treatment for rickety conditions, or whose parents or guardians did not provide consent, were excluded from the study. The laboratory reference values for biochemical parameters at the study centre were established as follows: Calcium 2.02-2.60 mmol/L; Phosphate 0.65-1.40 mmol/L; Alkaline Phosphatase 40-125 IU/L; Vitamin D 30-100 nmol/L; Total Protein 58-80 g/L; and Albumin 35-50 g/L. These reference values were consistent with documented standards in the literature (18,21,22).

Data collection occurred in two stages: first, at the time of presentation, demographic and clinical evaluation information was gathered; second, information regarding radiological and laboratory findings was compiled.

Ethical approval

Ethical approval for the study was obtained from the hospital's ethics and research committee with approval number: EKSUTH/A67/2025/10/004. Informed consent was obtained from the parents or guardians before patients were recruited for the study, ensured compliance with the ethical principles of voluntary engagement, non-maleficence and beneficence, privacy and confidentiality.

Data analysis

Data were subsequently collated and analyzed using IBM Statistical Package for Social Sciences (SPSS) version 25. An independent sample t-test was used to compare means of laboratory results, and a Chi-Square test of association was used for the categorical variables. Kendall's Coefficient of Concordance was used as a test of agreement amongst measured items. Statistical significance was determined at $p < 0.05$, with a confidence interval set at 95%.

Results

Thirty-two (32) patients met the inclusion criteria for the study group with a mean age of 3.06 ± 0.13 years, median 3 years, mode 3 years, and age range of 2 – 5 years. Singleton constituted 26(81.2%) and twins 6(18.8%). Gender: male 13(40.6%) and female 19(59.4%) equating to approximate ratio of 2:3. Religion: Christianity 24(75.0%) and Islam 8(25.0%). Clinical presentation: bilateral genu varum 10(31.3%), windswept 9(28.1%), unilateral genu varum 5(15.6%), unilateral genu valgum 4(12.5%), bilateral genu valgum 2(6.3%) and rickety rosary 2(6.3%).

The radiological images confirmed the bony deformities and features of rickets in the study group: widening of the physis, metaphyseal splaying, and fraying. Electrolytes, Urea, and Creatinine were normal in 30(93.8%) and deranged in 2(6.2%). Serum calcium: low 28(87.5%) and normal 4(12.5%). Serum phosphate: low 1(3.1%), normal 27(84.4%) and high 4(12.5%). Serum alkaline phosphatase: normal 8(25.0%) and high 24(75.0%). Serum vitamin D: low 4(12.5%) and normal 28(87.5%). Serum protein: normal 31(96.9%) and high 1(3.1%). Serum albumin: low 1(3.1%) and normal 31(96.9%).

Thirty-two patients were recruited in the control group, 13 males and 19 females, with an age range of 3- 5 years and a mean age of 4.1 ± 0.7 years. Biochemical laboratory parameters were within the laboratory reference range. They were all trauma

cases, and no classical features of rickets were seen on their radiographs as applicable to the region of their injury.

Table 1 shows the comparison of mean serum Calcium, Phosphate, Alkaline phosphatase, Vitamin D and Protein between the control group and the study group. There were statistically significant differences in the mean serum Calcium and Alkaline phosphatase between the control group and the study group (p = 0.02 and p = 0.01, respectively), while no significant difference in other biochemical parameters.

In Table 2A, the following association between variables in the study group were illustrated:

1. Category of birth and serum calcium, p = 0.304, null hypothesis accepted; no statistical-significance association between serum calcium values in patients who were either born as singleton or set of twins with comparison of variable born as a singleton or a set of twins.
2. Gender and serum calcium, p = 0.683, null hypothesis accepted. Serum calcium has no statistically significant association with gender. Gender, in the real sense, had no distinguishing trait as per serum calcium in evaluating patients with rickets.
3. Serum calcium and serum vitamin D, p = 0.001, there was no need to accept the null hypothesis; rather, the alternative hypothesis. The interplay between the two factors (serum calcium and vitamin D), very germane in the management of patients with rickets, was statistically significant: each was independently probatory.

In Table 2B, the following association between variables in the study group were illustrated:

1. Serum phosphate and serum vitamin D, p = 0.022, null hypothesis is rejected and alternate hypothesis accepted. There was a statistically significant association between serum phosphate and vitamin D values amongst patients with rickets.
2. Serum calcium and serum protein. p = 0.701, meaning that no statistical significance association between the two factors. The difference in this study might be relative.
3. Serum vitamin D and serum protein. p = 0.701, meaning that no statistical significance association between the two factors. The difference in this study might be relative.

In Table 2C, the following association between variables in the study group were illustrated:

1. Serum calcium and serum alkaline phosphatase. p = 0.014, there was statistical significance association in utilizing these factors for the evaluation of patients with rickets according to this study.
2. Serum vitamin D and serum alkaline phosphatase. p = 0.001, the values were independent in assessing patients with rickets for treatment: each was significant in its own right.
3. Serum protein versus alkaline phosphatase. p = 0.557; no statistical significance association existed between the two items: the difference might be relative.

In Table 3, Kendall's Coefficient of Concordance (KCC 0.742) was greater than 0.05; there was no statistical significance amongst the factors, whatever existed might be by chance. They have one thing or the other similar in evaluation of patients with rickets: biochemical parameters cannot be evaluated in isolation without recourse to one another.

Table 1: Comparison of mean values of biochemical parameters (n=32) between the study and control groups

Biochemical parameters	Control	Study group	*p* value
Serum Calcium (mmol/L)	2.29 ± 0.06	1.41 ± 0.44	0.02*
Serum Phosphate (mmol/L)	1.01 ± 0.02	1.13 ± 0.25	0.23
Serum Alkaline Phosphatase (IU/L)	119.17 ± 24.35	153.37 ± 16.58	0.01*
Serum Vitamin D (nmol/L)	44.39 ± 6.12	40.27 ± 8.83	1.02
Serum Protein: Total (g/L)	65.09 ± 4.18	67.95 ± 5.38	0.90
Albumin (g/L)	41.13 ± 4.12	42.20 ± 3.98	0.83

*** Statistically significant**

Table 2A: Association between variables

Birth and serum calcium (p = 0.304)				
		Serum calcium		
		Low (%)	Normal (%)	Total (%)
Category of birth	Single	22 (84.6)	4 (15.4%)	26 (100.0)
	Twins	6 (100.0)	0 (0.0%)	6 (100.0)
	Total	28 (87.5)	4 (12.5)	32 (100.0)
Gender and serum calcium (p = 0.683)				
		Serum calcium		
		Low (%)	Normal (%)	Total (%)
Gender	Male	11(84.6)	2(15.4)	13 (100.0)
	Female	17(89.5)	2(10.5)	19 (100.0)
	Total	28 (87.5)	4 (12.5)	32 (100.0)
Serum calcium and vitamin D (p = 0.001)				
		Serum Vitamin D		
		Low (%)	Normal (%)	Total (%)
Serum calcium	Low	1 (3.6)	27 (96.4)	28 (100.0)
	Normal	3 (75.0)	1 (25.0)	4 (100.0)
	Total	4 (12.5)	28 87.5)	32 (100.0)

Table 2B: Association between variables

Serum phosphate and vitamin D (p = 0.022)				
		Vitamin D		
		Low (%)	Normal (%)	Total (%)
Phosphate	Low	1 (100.0)	0 (0.0)	1 (100.0)
	Normal	3 (11.1)	24 (88.9)	27 (100.0)
	High	0 (0.0)	4 (100.0)	4 (100.0)
	Total	4 (12.5)	28 (87.5)	32 (100.0)
Serum calcium and protein (p = 0.701)				
		Serum protein		
		Normal (%)	High (%)	Total (%)
Serum calcium	Low	27 (96.4)	1 (3.6)	28 (100.0)
	Normal	4 (100.0)	0 (0.0%	4 (100.0)
	Total	31 (96.9)	1 (3.1)	32 (100.0)
Serum vitamin D and protein (p = 0.701)				
		Serum protein		
		Normal (%)	High (%)	Total (%)
Serum vitamin D	Low	4 (100.0)	0 (0.0)	4 (100.0)
	Normal	27 (96.4)	1 (3.6)	28 (100.0)
	Total	31 (96.9)	1 (3.1)	32 (100.0)

Table 2C: Association between variables

Serum calcium and Alkaline phosphatase (p = 0.014)				
		Serum alkaline phosphatase		
		Normal (%)	Low (%)	Total (%)
Serum calcium	Low	23 (82.1)	5 (17.9)	28 (100.0)
	Normal	1 (25.0)	3 (75.0)	4 (100.0)
	Total	24 (75.0)	8 (25.0)	32 (100.0)
Serum vitamin D and alkaline phosphatase (p = 0.001)				
		Serum alkaline phosphatase		
		Normal (%)	High (%)	Total (%)
Serum vitamin D	Low	0 (0.0)	4 (100.0)	4 (100.0)
	Normal	24 (85.7)	4 (14.3)	28 (100.0)
	Total	24 (75.0)	8 (25.0)	32 (100.0)
Serum protein and alkaline phosphatase (p = 0.557)				
		Serum alkaline phosphatase		
		Normal	High	Total
Serum protein	Normal	23 (74.2)	8 (25.8)	31 (100.0)
	High	1 (100.0)	0 (0.0)	1 (100.0)
	Total	24	8	32 (100.0)

Table 3: Test of agreement amongst measured items (unit) in the study group

	E/U/C	Sr Ca^{2+}	Sr PO_4^{2-}	Sr ALP	Sr Vit D	Sr Pr	Sr Alb
Mean Rank	1.72	1.89	5.02	5.39	4.42	4.88	4.69
KCC	0.742						

E/U/C Serum electrolyte, urea and creatinine,
Sr Ca^{2+} Serum calcium
Sr PO_4^{2-} Serum phosphate
Sr ALP Serum alkaline phosphatase
Sr Vit D Serum vitamin D
Sr Pr Serum protein
Sr Alb Serum albumin
KCC Kendall's Coefficient of Concordance

Discussion

As our contribution to the body of knowledge regarding biochemical deficiencies in rickets within our locality, we investigated 32 pediatric patients, building upon the earlier analysis conducted by Oginni et al (13), which included 26 patients. This increased sample size allowed for a more comprehensive understanding of the nutritional factors contributing to rickets in our population. Our findings aimed to reinforce the importance of identifying the underlying causes of this condition to inform better interventions and management strategies in the community. Some patients who would have qualified for the study had received various forms of pharmacotherapeutic measures for rickets before their presentation in the clinics. Practices of quackery and self-medication are still prevalent in this environment, indicating that the actual prevalence of nutritional rickets could be higher than what has been documented for the general population (23). This highlights the potential underreporting of cases and suggests that further investigation into the condition's prevalence is necessary. The control group, which exhibited no clinical features of rickets, had biochemical parameters that fell within the normal laboratory reference range; however, their alkaline phosphatase levels were at the higher end of that range. This may be attributed to the fact that these individuals were in an active growth phase of life.

Unlike many earlier studies that did not thoroughly address clinical presentations, this study identified genu varum as the most common clinical manifestation. Additionally, radiological imaging confirmed the presence of deformities in most cases, including instances of rickety rosary. The biochemical derangement of electrolyte levels, urea, and creatinine as indicators of renal function was noted in 2 patients (6.2%), which could suggest a potential link to absolute or relative Vitamin D production. Interestingly, a notable correlation was observed between serum calcium and vitamin D levels: while serum calcium was low in 28 patients (87.5%), serum vitamin D remained consistently within normal ranges. This finding strongly supports the notion that calcium deficiency is the primary cause of rickets in this region, with other factors potentially serving as contributing elements. This is in support of findings from areas with sufficient sunlight exposure, such as Egypt and India (13,15,17).

In Table 2, the statistical insignificance of the p-value when evaluating the category of birth and serum calcium suggests that whether a child is a singleton or part of a multiple birth does not significantly affect serum calcium metabolism. This indicates that the calcium derangement observed in these cases was likely acquired during development rather than being congenital. Similarly, the analysis shows that gender also had no significant impact on serum calcium levels among patients with rickets.

The relationship between serum calcium and vitamin D is pivotal to understanding rickets in this study. Notably, the analysis highlighted a significant statistical difference between these two factors: while serum calcium levels were found to be low, vitamin D levels remained within normal ranges. This finding points towards a metabolic issue or a deficiency in serum calcium as the underlying cause of rickets among patients in this region, rather than a problem with vitamin D production. Given that this is a tropical area with ample sunlight, the conversion of vitamin D in the skin is unlikely to be the issue; instead, the real concern seems to be an absolute or relative deficiency of calcium. Additionally, the observation of normal vitamin D levels alongside variations in serum phosphate levels necessitates further investigation into the true etiological factor behind rickets. Calcium and phosphate play crucial roles in bone formation, with over 98% of the body's calcium and 85% of its phosphate incorporated into hydroxyapatite crystals within the bone matrix (24). While both minerals are essential for maintaining bone structure and strength, calcium regulation is generally considered more critical than phosphate regulation. This is due to calcium's primary role in various physiological processes, including muscle contraction, nerve function, and blood clotting, which necessitate strict control of calcium levels in the body. Proper balance and availability of both minerals are vital for healthy bone development and growth.

In our analysis, no significant differences were observed when comparing serum calcium and protein levels, as well as serum vitamin D and protein levels, with both comparisons yielding a *p*-value of 0.701, indicating statistical non-significance. This suggests that the patients might have been consuming adequate protein, which is essential for the transport of calcium and vitamin D. The presence of serum proteinoid substances, particularly albumin, may have helped maintain sufficient levels of these vitamins and minerals. Conversely, when we compared serum calcium to alkaline phosphatase (ALP) and serum vitamin D to ALP, we found significant differences. Specifically, there was a statistically significant decrease in serum calcium alongside an increase in ALP levels, indicating heightened activity from osteoblasts. Notably, serum vitamin D remained nearly normal, reinforcing the notion that calcium deficiency is the primary cause of rickets in this region. In summary, our findings suggest that there was an adequate amount of serum protein present to support enzymatic processes during bone formation. This might explain the lack of significant differences observed in the earlier comparisons involving protein levels, further highlighting calcium deficiency as the predominant aetiology of rickets in this locality.

A non-parametric test of agreement, known as Kendall's Coefficient of Concordance (KCC), was employed to analyze the relationships among the measured values presented in Table 3. The KCC value of 0.742 indicated that there were no statistically significant differences among the factors, suggesting that each parameter shared a common quality in the evaluation of patients with rickets and osteomalacia. The observed similarities imply that relying on a single factor for diagnosing rickets may not be sufficient. Instead, a more effective approach would involve a concise and comprehensive combination of two or more factors, thereby ensuring a broader quality assurance framework for patient care. This approach not only guarantees adherence to global best practices but also enhances the efficacy of service delivery, training, and research in the field.

Conclusion

This study explored the causes of nutritional rickets in Ekiti, Southwestern Nigeria, and more broadly in the nation, focusing on whether calcium or vitamin D deficiency is the primary contributor to the condition. Through comprehensive laboratory investigations and clinical evaluations, the research identified the predominant deficiency and provided insights into effective treatment approaches. The findings underscore the importance of assessing both calcium and vitamin D levels, as well as the renal status of patients, to understand the underlying

factors contributing to rickets. The implications of this research are significant for developing targeted interventions that can improve outcomes for affected children in this region, emphasizing the necessity for calcium supplementation as a crucial part of management in cases where deficiency is identified. This study contributed to the broader understanding of nutritional rickets and highlights the need for awareness and proactive measures in tropical regions where natural sunlight is abundant, yet deficiencies persist.

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