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Original Article

The Role of Nutritional Deficiencies and Micronutrient Status on Pediatric Bone Development in Low-Income Communities

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ABSTRACT

Background: Nutritional deficiencies may impair skeletal mineralization and contribute to fractures and orthopedic deformities during childhood, particularly in low-resource communities. Objective: To evaluate vitamin D status, estimated calcium and protein intake, and their relationships with pediatric fracture occurrence and orthopedic deformity. Methods: A cross-sectional observational study included 220 children aged 2–15 years attending hospital and community-linked services in Rahim Yar Khan, Pakistan. Clinical, dietary, anthropometric, laboratory, orthopedic, and clinically indicated radiological assessments were performed. Vitamin D deficiency was defined as serum 25-hydroxyvitamin D below 20 ng/mL. Results: Mean age was 9.03 ± 4.01 years, mean vitamin D concentration was 17.96 ± 6.43 ng/mL, mean calcium intake was 541.87 ± 187.99 mg/day, and mean protein intake was 32.81 ± 10.78 g/day. Fractures occurred in 58/220 children (26.4%), while orthopedic deformities were reported in 18.2%. Fracture prevalence was 30.2% among vitamin D-deficient children and 20.9% among non-deficient children. The crude risk ratio was 1.45 (95% CI 0.90–2.34), with an absolute risk difference of 9.4 percentage points (95% CI −2.2 to 20.9; p=0.121). Conclusion: Vitamin D deficiency and suboptimal reported nutrient intake were common. Vitamin D-deficient children had a higher crude fracture proportion, although the association remained statistically inconclusive. Keywords: Pediatric bone health; vitamin D deficiency; calcium intake; protein intake; fracture; orthopedic deformity; nutritional rickets; low-income communities.

INTRODUCTION

Childhood and adolescence are critical periods for skeletal growth, growth-plate maturation, and acquisition of peak bone mass. During these stages, bone development depends on the coordinated availability of calcium, vitamin D, protein, and other nutrients required for collagen synthesis, mineral deposition, muscle function, and skeletal remodeling. Deficiency of these nutrients can impair mineralization of the growing skeleton and may manifest as bone pain, delayed growth, widening of the wrists, lower-limb deformity, gait abnormalities, nutritional rickets, and increased susceptibility to fracture (1–4). The consequences may extend beyond childhood because inadequate accumulation of bone mass during growth can compromise long-term skeletal strength and increase the risk of future musculoskeletal morbidity (5,6).

Vitamin D plays a central role in intestinal calcium absorption and maintenance of calcium-phosphate homeostasis. In growing children, inadequate vitamin D may result in secondary disturbances in mineral metabolism and defective mineralization at the growth plate (1–3). However, nutritional bone disease cannot be attributed to vitamin D status alone. In populations with limited access to dairy products and other calcium-rich foods, low dietary calcium intake may independently contribute to rickets and may amplify the skeletal effects of vitamin D deficiency (7,8). Protein is also essential because the organic component of bone consists predominantly of a collagen-based matrix upon which mineralization occurs. Inadequate protein intake may therefore impair linear growth, muscle development, bone-matrix formation, and recovery from illness or injury (9,10).

Children living in low-income communities may be particularly vulnerable to these nutritional risks because household food choices are frequently constrained by affordability and availability. Diets may rely predominantly on cereals, rice, tea, and other low-cost foods, while regular consumption of milk, yogurt, eggs, meat, fortified products, and diverse plant-based protein sources may be limited. Reduced outdoor activity, extensive clothing coverage, recurrent infections, poor appetite, and delayed healthcare-seeking may further increase the likelihood that nutritional deficiencies remain unrecognized until a child develops pain, deformity, delayed walking, or fracture. In Pakistan, vitamin D deficiency and childhood undernutrition remain important public-health concerns, with national and regional studies documenting high frequencies of vitamin D deficiency, stunting, and inadequate nutritional status among children and adolescents (11–14).

Fractures in children are commonly related to trauma and physical activity; however, underlying bone quality may influence whether a relatively minor injury results in fracture. Pakistani and international studies have reported a high prevalence of vitamin D deficiency among pediatric fracture patients, including children presenting with supracondylar fractures of the humerus (15,16). These observations do not establish a causal relationship because fracture risk is also influenced by age, sex, body composition, activity level, trauma mechanism, muscle strength, pubertal status, and environmental exposure. Nevertheless, they support the clinical relevance of evaluating nutritional status in children presenting with fractures, recurrent limb pain, deformity, or impaired growth.

Existing evidence from Pakistan has largely focused on the prevalence of individual nutritional deficiencies, childhood undernutrition, nutritional rickets, or selected orthopedic presentations. Comparatively less evidence has simultaneously examined serum vitamin D status, estimated calcium intake, protein intake, fracture history, orthopedic deformity, and radiological indicators of impaired mineralization within the same pediatric population. This evidence gap is particularly relevant in low-resource communities, where multiple deficiencies may coexist and where nutritional, pediatric, and orthopedic problems are frequently managed through separate clinical pathways.

The present study therefore aimed to determine the nutritional and bone-health profile of children aged 2–15 years from low-income communities attending hospital and community-linked healthcare services in Rahim Yar Khan. The primary objective was to assess the association between vitamin D deficiency and fracture occurrence. Secondary objectives were to examine the relationships of estimated dietary calcium and protein intake with orthopedic deformity and clinically identified growth-plate or mineralization abnormalities. It was hypothesized that children with lower vitamin D levels and poorer calcium and protein intake would demonstrate a greater frequency of adverse skeletal outcomes than children with comparatively adequate nutritional status.

MATERIALS AND METHODS

A cross-sectional observational study was conducted among children attending pediatric, orthopedic, nutrition, radiology, laboratory, outpatient, and community-linked healthcare services associated with RYK Hospital, Rahim Yar Khan, Pakistan. The cross-sectional design was selected because nutritional exposures and bone-health outcomes were evaluated during the same study period, without allocation

of an experimental intervention. The study population comprised 220 children aged 2–15 years from low-income communities who presented for routine pediatric assessment, nutritional concerns, limb pain, delayed walking, suspected nutritional rickets, visible skeletal deformity, gait disturbance, or fracture-related evaluation.

Children were eligible when they were between 2 and 15 years of age, belonged to the target low-income communities, and underwent clinical and nutritional assessment through the participating services. Children with chronic kidney disease, chronic liver disease, known hereditary or genetic bone disorders, malignancy, severe congenital skeletal deformity, or prolonged systemic corticosteroid exposure were excluded because these conditions could independently alter skeletal growth, mineral metabolism, or fracture susceptibility. Children were recruited from the participating clinical and community-linked services after written informed consent had been obtained from a parent or legal guardian. Each participant was assigned a study record and evaluated using a structured data-collection form.

Sociodemographic and clinical data included age, sex, place of residence, household income category, parental education, number of siblings, birth history, breastfeeding history, previous medical conditions, medication use, supplement use, and previous treatment for vitamin D deficiency or nutritional rickets. Information regarding sunlight exposure was obtained from the parent or guardian and included usual outdoor duration, typical time of outdoor exposure, clothing coverage, and habitual outdoor activity. Fracture history was documented with reference to fracture site, number of previous fractures, reported mechanism of injury, and whether the fracture followed minor or major trauma. Clinical records and radiographs were reviewed where available to support fracture classification.

Dietary exposure was assessed through a structured history of usual food consumption. Particular attention was given to the frequency and estimated quantity of milk, yogurt, cheese, eggs, meat, pulses, vegetables, fortified foods, and other commonly consumed sources of calcium and protein. Estimated daily calcium intake was calculated from reported dairy and non-dairy food consumption, while estimated protein intake was calculated from the child's usual dietary pattern. Nutrient intake was recorded as continuous variables in milligrams per day for calcium and grams per day for protein. Vitamin D status was assessed using serum 25-hydroxyvitamin D concentration and was categorized as deficient when the concentration was below 20 ng/mL. Calcium and protein intake were evaluated both continuously and according to clinically relevant intake categories used in the analysis.

Anthropometric assessment included body weight and standing height, measured using standard clinical procedures. Body mass index was calculated as weight in kilograms divided by height in meters squared. Height, weight, and body mass index were interpreted according to age- and sex-appropriate pediatric reference values. Participants were examined for clinical indicators of impaired skeletal development, including wrist widening, bowing of the legs, genu valgum, bone tenderness, delayed walking, abnormal spinal posture, gait disturbance, and visible limb deformity.

Venous blood samples were obtained for measurement of serum 25-hydroxyvitamin D, calcium, phosphate, alkaline phosphatase, albumin, and hemoglobin. Parathyroid hormone testing was performed when clinically indicated. Laboratory results were interpreted using the reference ranges applied by the hospital laboratory. Serum vitamin D concentration represented the principal biochemical exposure variable, while calcium, phosphate, alkaline phosphatase, albumin, and parathyroid hormone findings were used to support the clinical assessment of mineral metabolism and suspected nutritional bone disease.

Radiological evaluation was performed only when clinically indicated and was not undertaken solely for research purposes. Available radiographs of the wrist, knee, fractured limb, or other clinically relevant region were reviewed for growth-plate widening, metaphyseal cupping or fraying, delayed mineralization, skeletal deformity, and fracture pattern. Radiological findings were interpreted in conjunction with orthopedic and radiology assessments. The denominator of children undergoing

radiological assessment was maintained separately from the total study population to avoid classifying unexamined children as having normal radiological findings.

The principal exposure variables were serum vitamin D status, estimated daily calcium intake, and estimated daily protein intake. The primary outcome was fracture occurrence. Secondary outcomes included clinically identified orthopedic deformity and radiological evidence of growth-plate or mineralization abnormalities among children who underwent imaging. Fracture occurrence was treated as a binary outcome and was additionally examined according to trauma severity where the relevant information was available. Orthopedic deformity was defined by the presence of one or more clinically identified abnormalities, including lower-limb bowing, genu valgum, wrist widening, gait disturbance, or another documented skeletal deformity.

Potential sources of selection and measurement bias were addressed by applying consistent eligibility criteria, using the same structured data-collection form, documenting the clinical service from which each child was recruited, and maintaining standardized definitions for nutritional and skeletal variables. Dietary information was obtained from the parent or guardian to improve recall in younger children. Clinical, laboratory, and radiological findings were recorded separately before being integrated into the final dataset. Because age, sex, anthropometric status, sunlight exposure, physical activity, supplement use, and trauma mechanism could influence both nutritional status and skeletal outcomes, these variables were considered potential confounders in the association analyses.

Data were checked for completeness, logical consistency, out-of-range values, and duplication before analysis. Continuous variables were summarized using mean and standard deviation when approximately normally distributed and median with interquartile range when distributional assumptions were not met. Categorical variables were summarized as frequencies and percentages using the relevant non-missing denominator. Differences in continuous variables between two groups were assessed using an independent-samples t-test or a non-parametric alternative, as appropriate. Associations between categorical nutritional exposures and skeletal outcomes were assessed using the chi-square test or Fisher's exact test when expected cell counts were small.

For the primary analysis, fracture occurrence was compared between children with vitamin D deficiency and those without deficiency. Crude measures of association were expressed as risk ratios or odds ratios with 95% confidence intervals. Associations of calcium and protein intake with fracture and orthopedic deformity were evaluated using categorical comparisons and regression analysis. Multivariable binary logistic regression was used to estimate adjusted associations with fracture and deformity after accounting for relevant confounding variables, including age, sex, anthropometric status, sunlight exposure, supplement use, and trauma mechanism where appropriate. Multicollinearity, influential observations, and model fit were assessed before interpretation of adjusted estimates. Statistical tests were two-sided, and a p-value below 0.05 was considered statistically significant.

Missing observations were excluded only from analyses requiring the unavailable variable, and denominators were reported separately for each analysis. Radiological analyses were restricted to participants who underwent clinically indicated imaging. No child without radiological assessment was assumed to have a normal radiograph. Data were stored in a secure electronic database, and the final analytical dataset was cross-checked against the original study forms to promote accuracy and reproducibility. Ethical approval was obtained from the relevant institutional review committee. Written informed consent was obtained from a parent or legal guardian before enrolment. The study involved observational assessment only, and participation did not delay or restrict clinical care. Children identified with vitamin D deficiency, suspected nutritional rickets, severe nutritional impairment, fracture complications, or clinically significant deformity were referred for appropriate pediatric, nutritional, or orthopedic management according to routine hospital practice.

RESULTS

A total of 220 children aged 2–15 years were included in the analysis. The mean age was 9.03 ± 4.01 years. Mean serum vitamin D concentration was 17.96 ± 6.43 ng/mL, while the mean estimated daily calcium and protein intakes were 541.87 ± 187.99 mg/day and 32.81 ± 10.78 g/day, respectively. Fractures were reported in 26.3% of participants, and orthopedic deformities were identified in 18.2%.

Table 1. Nutritional and Bone-Health Characteristics of the Study Population

Characteristic	n	Mean ± SD	%
Age, years	220	9.03 ± 4.01	—
Serum vitamin D, ng/mL	220	17.96 ± 6.43	—
Estimated calcium intake, mg/day	220	541.87 ± 187.99	—
Estimated protein intake, g/day	220	32.81 ± 10.78	—
Fracture occurrence	—	—	26.3
Orthopedic deformity	—	—	18.2

SD, standard deviation.

The cohort demonstrated generally low nutritional indicators relevant to pediatric skeletal health. The mean serum vitamin D concentration was below 20 ng/mL, while mean calcium intake was approximately 542 mg/day. Fractures affected slightly more than one-quarter of the study population, whereas orthopedic deformities were reported in approximately one-fifth. Exact distributions according to sex, age category, anthropometric status, sunlight exposure, protein-adequacy category, and calcium-adequacy category were not reported and therefore could not be included.

Table 2. Association Between Vitamin D Deficiency and Fracture Occurrence

Vitamin D status	Total, n	No fracture, n (%)	Fracture, n (%)
Deficient, <20 ng/mL	129	90 (69.8)	39 (30.2)
Non-deficient, ≥20 ng/mL	91	72 (79.1)	19 (20.9)
Total	220	162 (73.6)	58 (26.4)

Children with vitamin D deficiency had a fracture proportion of 30.2%, compared with 20.9% among children without vitamin D deficiency. The absolute difference between groups was 9.4 percentage points. Although the fracture proportion was numerically higher in the deficient group, the unadjusted comparison did not reach conventional statistical significance.

Table 3. Crude Effect Estimates for Vitamin D Deficiency and Fracture Occurrence

Measure	Estimate	95% CI	p-value
Risk ratio	1.45	0.90–2.34	0.121
Odds ratio	1.64	0.87–3.08	0.121
Risk difference, percentage points	9.4	−2.2 to 20.9	0.121
Pearson χ^2	2.40	—	0.121

CI, confidence interval. Estimates were calculated directly from the reported 2 × 2 table. The p-value was obtained using the uncorrected Pearson chi-square test.

Table 4. Availability of Reported Nutritional and Skeletal Outcome Analyses

Analysis	Exposure data reported	Outcome data reported	Effect estimate available
Vitamin D status and fracture	Yes	Yes	Yes
Calcium intake and fracture	No	No	No
Calcium intake and orthopedic deformity	No	No	No
Protein intake and orthopedic deformity	No	No	No
Combined nutritional deficiencies and fracture	No	No	No
Combined nutritional deficiencies and deformity	No	No	No
Nutritional status and growth-plate abnormality	No	No	No
Multivariable regression for fracture	No	No	No
Multivariable regression for deformity	No	No	No

Vitamin D-deficient children had an estimated 45% higher crude risk of fracture than non-deficient children; however, the 95% confidence interval ranged from a 10% lower risk to more than twice the

risk and included the null value. The crude odds ratio similarly suggested higher odds of fracture among deficient children, but its confidence interval was wide and crossed 1.00. These findings indicate a clinically relevant numerical pattern but insufficient evidence to establish an independent association without adjustment for age, sex, anthropometric status, sunlight exposure, activity level, supplement use, and trauma mechanism.

The manuscript described higher deformity rates among children with lower calcium intake, a stronger relationship between low protein intake and deformity, and an adverse pattern associated with combined deficiencies. However, the corresponding group sizes, outcome counts, regression coefficients, effect estimates, confidence intervals, and p-values were not supplied. These associations could therefore not be reproduced or included as quantitative findings. Claims concerning growth-plate abnormalities were also not analyzable because the number of children undergoing radiography and the number with abnormal findings were not reported.

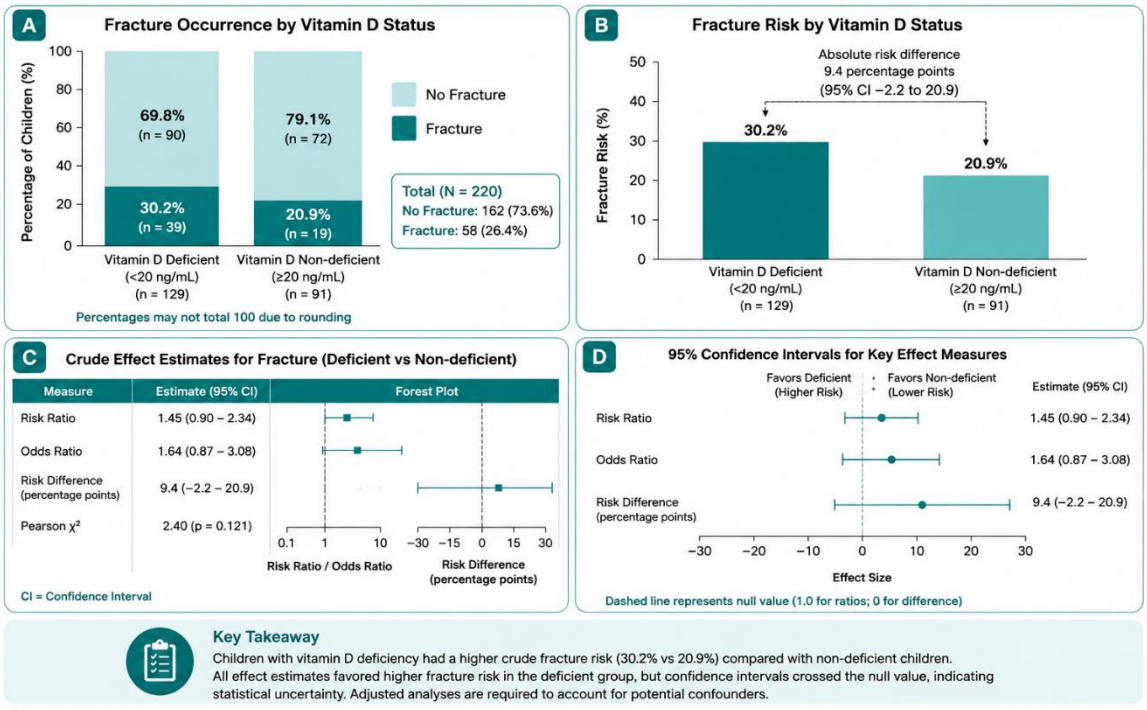

Figure 1 The figure integrates the complete vitamin D–fracture dataset for 220 children. Among 129 vitamin D-deficient children, 39 experienced fractures, corresponding to 30.2%, compared with 19 fractures among 91 non-deficient children, corresponding to 20.9%. This produced an absolute risk difference of 9.4 percentage points (95% CI −2.2 to 20.9), a crude risk ratio of 1.45 (95% CI 0.90–2.34), and a crude odds ratio of 1.64 (95% CI 0.87–3.08). Although all point estimates indicated a higher fracture burden among vitamin D-deficient children, the confidence intervals crossed the null value, indicating substantial statistical uncertainty and the need for adjusted analysis accounting for age, sex, anthropometric status, sunlight exposure, activity level, supplementation, and trauma mechanism.

DISCUSSION

This cross-sectional observational study examined nutritional status and skeletal outcomes among 220 children aged 2–15 years from low-income communities in Rahim Yar Khan. The cohort had a mean serum 25-hydroxyvitamin D concentration of 17.96 ± 6.43 ng/mL, indicating that low vitamin D status was common at the population level. Mean estimated calcium intake was 541.87 ± 187.99 mg/day, while mean protein intake was 32.81 ± 10.78 g/day. Fractures were reported in 58 of 220 children, corresponding to 26.4%, and orthopedic deformities were identified in approximately 18.2% of participants. These findings highlight the coexistence of nutritional vulnerability and clinically relevant skeletal problems among children receiving care in a low-resource setting, consistent with broader evidence linking childhood nutrition, physical activity, and skeletal development (4–6).

The principal quantitative finding was a higher crude fracture proportion among children with vitamin D deficiency. Fractures occurred in 39 of 129 vitamin D-deficient children, corresponding to 30.2%, compared with 19 of 91 non-deficient children, corresponding to 20.9%. The resulting crude risk ratio was 1.45, with a 95% confidence interval of 0.90–2.34, while the crude odds ratio was 1.64, with a 95% confidence interval of 0.87–3.08. The absolute difference in fracture proportion was 9.4 percentage points, with a 95% confidence interval ranging from −2.2 to 20.9 percentage points. Although all point estimates suggested a greater fracture burden among vitamin D-deficient children, the confidence intervals included the null value and the Pearson chi-square test did not reach conventional statistical significance. The observed pattern should therefore be interpreted as clinically suggestive rather than conclusive.

The direction of this association is biologically plausible. Vitamin D facilitates intestinal calcium absorption and contributes to calcium-phosphate homeostasis, skeletal mineralization, and growth-plate function (1–3,20). In children with persistent vitamin D deficiency, reduced calcium absorption and compensatory alterations in mineral metabolism may impair mineral deposition in the developing skeleton and increase susceptibility to skeletal pain, deformity, or fracture (1–3). However, pediatric fractures are multifactorial, and age, sex, pubertal maturation, body composition, physical activity, trauma mechanism, muscle strength, seasonal sunlight exposure, and underlying disease may influence fracture occurrence independently of vitamin D status (4–6). The crude association observed in this study cannot therefore establish that vitamin D deficiency caused the reported fractures.

The findings are broadly consistent with previous research demonstrating a high prevalence of vitamin D deficiency among children, adolescents, and adults in Pakistan (11,12). Studies of pediatric fracture populations have similarly reported frequent vitamin D deficiency, including among children presenting with supracondylar fractures of the humerus and other fracture types (15,16). Nevertheless, the available evidence remains heterogeneous, and vitamin D deficiency should not automatically be interpreted as the sole cause of reduced bone strength or fracture occurrence. Supplementation studies suggest that improvements in bone mineral outcomes may be more evident among children with established biochemical deficiency than among unselected pediatric populations (17,18). This distinction is clinically important because biochemical deficiency, dietary insufficiency, nutritional rickets, low bone mineral density, and fracture susceptibility are related but non-equivalent conditions (1,2,17,18).

The mean estimated calcium intake of approximately 542 mg/day also warrants attention. Calcium requirements vary substantially across childhood and adolescence, and a single absolute intake threshold cannot be applied uniformly to children aged 2–15 years. The reported mean may represent comparatively adequate intake for some younger children but inadequate intake for many school-aged children and adolescents. Adequate calcium intake is essential for bone mineral accrual during growth, and supplementation has been shown to improve bone mineral density in selected adolescent populations (4,5,19). Nutritional-rickets research has also established that low dietary calcium can independently cause or aggravate defective skeletal mineralization, including in regions where sunlight exposure is substantial (1,7,8). Future analyses should therefore compare each child's estimated intake with age-specific dietary requirements rather than relying exclusively on a universal cut-off.

Protein intake must be interpreted with similar caution. Protein is required for collagen synthesis, bone-matrix formation, muscle development, and overall somatic growth. Systematic reviews and expert evaluations indicate that adequate protein intake contributes to bone health, although its effects depend on total energy intake, calcium availability, physical activity, hormonal status, and overall dietary quality (9,10). In the present study, the manuscript described a stronger relationship between low protein intake and orthopedic deformity than between low protein intake and acute fracture. However, the category-specific denominators, outcome counts, effect estimates, confidence intervals, and adjusted analyses were not available. Consequently, the proposed relationship cannot be quantified or independently evaluated from the reported data.

The reported orthopedic deformity prevalence of 18.2% suggests that visible or clinically detected skeletal abnormalities were not uncommon. Bowing of the legs, genu valgum, wrist widening, gait abnormalities, and suspected growth-plate changes may occur in nutritional rickets because defective mineralization primarily affects the growth plate and weight-bearing bones in growing children (1–3). However, these findings are not pathognomonic and may also reflect physiological developmental variation, trauma, congenital abnormalities, neuromuscular conditions, or other metabolic bone disorders. Vitamin D deficiency is a major global public-health concern, but biochemical deficiency alone does not establish active rickets or clinically significant skeletal disease (20,21). Diagnostic interpretation should integrate clinical examination, age-specific radiological findings, serum calcium and phosphate, alkaline phosphatase, parathyroid hormone, and relevant differential diagnoses (1,2).

The wider nutritional context is also important. Vitamin D deficiency may coexist with iron deficiency, stunting, underweight, and broader dietary inadequacy among socioeconomically disadvantaged children (13,14,22). National and regional studies have documented persistent undernutrition and stunting among Pakistani children, reflecting the influence of poverty, dietary diversity, maternal factors, recurrent illness, and healthcare access (13,14). Pakistani studies have also reported nutritional-rickets presentations and associated nutritional risk factors among young and hospitalized children (23–25). The present findings add to this literature by examining vitamin D status, estimated calcium and protein intake, fractures, deformities, and radiological concerns within the same pediatric sample. However, because the study population included children presenting with routine pediatric concerns as well as limb pain, suspected rickets, deformity, and fracture, the sample was clinically enriched and should not be considered representative of all children living in low-income communities.

Several limitations should be considered. First, the cross-sectional design prevents determination of whether nutritional deficiencies preceded fractures or deformities. Vitamin D and dietary intake may have been assessed after the skeletal event, creating uncertainty regarding temporality and the possibility of reverse causation. Second, recruitment through pediatric, orthopedic, nutrition, and community-linked services may have introduced selection bias because symptomatic children were more likely to be included. Third, calcium and protein intake were estimated from dietary history, making the measurements vulnerable to recall error, portion-size misclassification, and parental reporting bias. The dietary assessment instrument and nutrient-composition method were not reported in sufficient detail to determine measurement validity.

Fourth, the study included a wide age range without reported stratification by age-specific nutrient requirements or pubertal status, despite the substantial variation in bone growth and nutrient needs across childhood and adolescence (4–6). Fifth, radiographs were obtained only when clinically indicated, meaning that children with symptoms were more likely to undergo imaging. This selective verification may overestimate the prevalence of radiological abnormalities among those assessed and prevents extrapolation to the complete sample. Sixth, the number of children who underwent radiography and the frequency of specific growth-plate abnormalities were not reported. Seventh, no adjusted regression results were available, leaving potential confounding by age, sex, anthropometric status, sunlight exposure, physical activity, supplement use, socioeconomic status, season, and trauma mechanism unresolved. Finally, missing-data frequencies, laboratory assay procedures, recruitment dates, and center-specific contributions were not reported.

Future studies should use a clearly defined prospective cohort design with standardized recruitment, validated dietary assessment, age-specific nutrient-adequacy classifications, pubertal staging, and predefined clinical and radiological outcomes. Incident fractures should be distinguished from pre-enrolment fracture history, and trauma mechanism should be documented systematically. Multivariable analyses should report adjusted risk ratios or odds ratios with 95% confidence intervals and examine whether calcium intake, protein intake, anthropometric status, and sunlight exposure modify the relationship between vitamin D deficiency and skeletal outcomes. Despite the current limitations, the

findings support the clinical relevance of nutritional assessment in children presenting with fractures, recurrent bone pain, poor growth, gait disturbance, or skeletal deformity, while emphasizing that nutritional screening should complement rather than replace comprehensive pediatric and orthopedic evaluation (1,2,4).

CONCLUSION

Vitamin D deficiency and comparatively low estimated calcium and protein intake were common among children from low-income communities included in this study. Fractures occurred in 30.2% of vitamin D-deficient children and 20.9% of non-deficient children, producing a crude risk ratio of 1.45 and an absolute risk difference of 9.4 percentage points; however, the corresponding confidence intervals crossed the null value, and the association was not statistically conclusive. Orthopedic deformities were also reported, but their relationships with calcium intake, protein intake, and combined nutritional deficiencies could not be quantified from the available data. These findings support structured nutritional and skeletal assessment among clinically at-risk children, consistent with established recommendations for evaluating nutritional rickets and pediatric bone health, but they do not establish a causal effect of vitamin D deficiency on fracture occurrence (1,2,4). Prospective, adequately powered studies using standardized dietary measurements, age-specific nutrient thresholds, uniform radiological criteria, and multivariable adjustment are required to determine the independent contribution of nutritional deficiencies to pediatric fracture and deformity risk.

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