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PREVALENCE OF VITAMIN D DEFICIENCY AMONG INFANTS AND TODDLERS: CROSS SECTIONAL STUDY

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Abstract:
Nutritional rickets is obtaining attention of public health professionals and clinicians of over all the world as this disease has been an epidemic problem in many developed and under developed countries, where it was considered that the disease has been eliminated [1]. The highest interest of current debate is what should be accurate measuring levels of 25-hydroxyvitamin D [25(OH0D] to evaluate vitamin D deficiency. To maintain the appropriate level of vitamin D in the body as deficiency leads to certain bones issue whereas increased level of vitamin D in the body can cause serious problems like cancer, neurological disorders and hypertension. A relationship was noted between the risk of vitamin D deficiency and lack of supplementation among breastfed infants and among toddlers with a higher body mass index. Among toddlers, there was a protective effect seen between milk consumption and lower risk of deficiency. These data underscore the fact that all breastfed infants should receive vitamin D supplementation for the duration of breastfeeding. However, further studies are needed to determine whether a 200 IU daily dose will provide adequate supplementation.

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INTRODUCTION:

Nutritional rickets is obtaining attention of public health professionals and clinicians of over all the world as this disease has been an epidemic problem in many developed and under developed countries, where it was considered that the disease has been eliminated [1]. The highest interest of current debate is what should be accurate measuring levels of 25-hydroxyvitamin D [25(OH0D] to evaluate vitamin D deficiency. To maintain the appropriate level of vitamin D in the body as deficiency leads to certain bones issue whereas increased level of vitamin D in the body can cause serious problems like cancer, neurological disorders and hypertension [2] [3]

Over past several years, dietary calcium requirements have highlighted in North America especially in adolescents and adult women. In developed countries they have habitual calcium intakes of 25-33 percent of these recommended intakes. Thus, the question that needs to asked is how relevant are these recommendations for populations in other parts of the world who are likely to have different lifestyles and genetic make-up [5] [6]

In north countries, nutritional rickets was eliminated in 20th century as soon as discovery of vitamin D came into process and the importance of sunlight, dietary supplement and food fortification has major role in prevention of vitamin D deficiency [7]

In the USA, nutritional rickets is almost exclusively confined to breastfed African American infants5 , while in Europe it has been reported extensively in the children of recent immigrants from India, Pakistan and Bangladesh , north Africa7 and the Middle East. A similar pattern is reported from Australia [8]. Most common characteristic of these situations are increased skin pigmentation and limited sun exposure due to clothing coverage in the mother and prolonged breast feeding of the affected infant. Literature shows that vitamin D deficiency is most common among pregnant women which increasing the risk for and severity of vitamin D deficiency in their offspring [9] [10]. Although vitamin D deficiency is probably the final common path in the development of the disease in this group, there is evidence that low dietary calcium and high phytate intakes might play a major role in the pathogenesis. This aspect will be discussed late in the paper [11]. Vitamin D deficiency and rickets contributes to major health problem among infants adolescent females, and pregnant women are at major risk [12] [13]. Social and some religious outfits which prevent to adequate sun exposure of pregnant women and their adolescent which are more prone to their children and young infants to vitamin D deficiency and rickets [14]

Low dietary calcium intakes in a smaller percentage of children are also reported to play a role. Studies provide evidence that the pathogenesis is related to dietary calcium deficiency possibly due to the introduction of irrigation which has been associated with an increase in the size of the annual rice harvest and a reduction in dietary variation [15][16]. Rickets appeared mainly due to the vitamin D deficiency this has linked with high latitude, cold winters, and limited skin exposure [17].

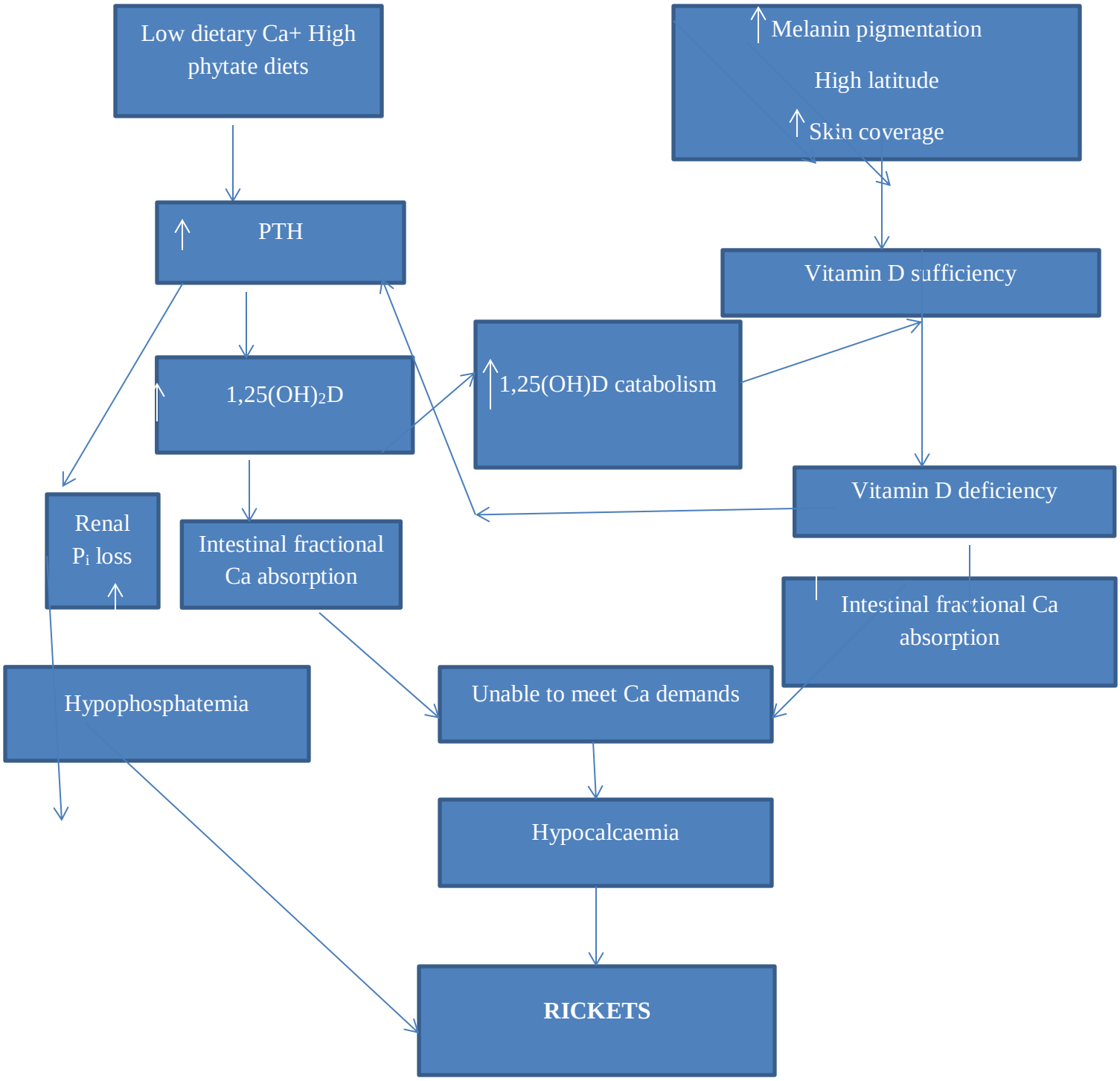

Fig 1. The pathogenesis of nutritional rickets

Pathogenesis of rickets: Vitamin D deficiency is the clear cause of nutritional rickets.

The final common path is an inability to meet the calcium needs of the growing skeleton resulting in hypocalcaemia and elevated parathyroid hormone (PTH) concentrations which in children with low dietary calcium intakes but vitamin D sufficiency increase 1,25(OH)2 D concentrations and vitamin D catabolism. If vitamin D status is poor, this may result in vitamin D deficiency compounding the effects of low dietary calcium intakes.

The pathogenesis of the bone disease is associated to low dietary calcium intakes as it responds rapidly to calcium supplements, which are more effective than vitamin D supplementation [18] Support for the role of calcium deprivation being important in the aetiology comes from the finding of higher concentrations of 1,25(OH)2 D in affected than control children [19] and fractional intestinal absorption of calcium being similar to that of controls at about 60 per cent [20]. Other possibly related factors in affected children include the finding of a higher frequency of a vitamin D receptor gene polymorphism allele (FF) in affected children and lower breast-milk calcium concentrations in mothers of children with rickets than in mothers with unaffected children [21]. Studies that are currently ongoing suggest that vitamin D requirements may be influenced by dietary calcium intakes (Thacher TD unpublished results). This concept is not surprising as low calcium intakes increase vitamin D catabolism thus increasing requirements to maintain a normal circulating concentration of 25(OH)D [22].

METHODS:

Total 380 participants were included in the study. Age ranged between 8-24 months. Exclusion criteria included of a chronic disease and use of medication of vitamin D during the previous 3 months. A written informed consent was signed by parents by informing the purpose of study.

CLINICAL AND ANTHROPOMETRIC DATA:

Demographic data which include Age, sex, height, weight, nutritional intake, daily sun exposure skin color and sensitivity. Skin color and sensitivity were measured by research assistant by established methods. Whereas skin pigmentation scale ranged from 1 (heavily pigmented e.g., black) to 4 (lightly pigmented, e.g., white) the skin sensitivity scale ranged from 1 (burns easily) to 6 (never burns).

DEFINITIONS:

The patients were divided into 3 diagnostic categories according to serum 25OHD levels, as rounded to the nearest integer: vitamin D deficiency (≤20 ng/mL), severe vitamin D deficiency (≤8 ng/mL), and suboptimal vitamin D status (≤30 ng/mL). The definition of vitamin D deficiency was based on growing consensus among experts that a 25OHD level of 20 ng/mL or less is the appropriate diagnostic threshold for deficiency [23] [24] [25]. This threshold also represents the low end of the reference range provided by the manufacturer of the assay. There is agreement among many skeletal health experts that 30 ng/mL represents an optimal level for 25OHD22–24 or a threshold of adequate concentration. This is supported by the fact that serum 25OHD level has been shown to be inversely correlated with PTH level at concentrations of 30 to 40 ng/mL.20,25,26 We decided a priori that a 5% prevalence of vitamin D deficiency would be clinically significant. The definition of severe vitamin D deficiency was based on the sensitivity of the 25OHD assay (7 ng /mL).

RADIOGRAPHIC EVALUATION:

Participants found to have vitamin D deficiency underwent frontal bilateral wrist and knee computed radiography. Soft copies of radiographs were reviewed for rachitic changes on a single high-resolution monitor by 2 pediatric radiologists (P.K.K. and J.P.-R.), each working from a unique randomized list of participants. The inter-rater correlation coefficient was calculated for the Thacher et al27 and demineralization scales. Each radiologist interpreted all images independently. Evidence of rickets on wrist and knee radiographs (eg, metaphyseal fraying) was determined using a validated 10-point scoring system.27 The score progressed in half-point increments from 0 (normal) to 10 (severe) and included both wrists and knees. Scores from the 2 radiologists were averaged, and data on inter-rater reliability was recorded. A scale (0–5) was also used by each radiologist to assess degree of demineralization, and the scores were averaged.

STATISTICAL ANALYSIS:

We used the Fisher exact test to compare the prevalence of vitamin D deficiency between infants and toddlers and to identify binary or polytomous variables associated with vitamin D deficiency within each patient group. To identify continuous variables associated with vitamin D deficiency, we used simple logistic regression with deficiency as the dichotomous

dependent variable. To identify binary or polytomous variables associated with 25OHD level, we used the t test or 1-way analysis of variance (Fisher F test). To identify continuous variables associated with 25OHD level, we used simple linear regression.

We constructed a multiple logistic regression model for vitamin D deficiency and a multiple linear regression model for 25OHD concentration using all predictors of interest, whether or not the simple association was significant. We tested for confounding and masking relationships by adding or removing variables and observing the effect on statistical significance of the remaining variables in the model. The results were corroborated by forward and backward stepwise procedures for automatic variable selection.

We used Pearson r to determine the correlation between 25OHD and PTH levels and to characterize other relationships among variables. We used the intraclass correlation coefficient [(patient variance)/(patient+rater+residual variance)] to determine interrater reliability for radiographic scales. Statistical analyses were conducted with SAS statistical software, version 9.

- Fisher exact test used to compare the prevalence of vitamin D deficiency between infants and toddlers.
- Simple logistic regression was used to identify continuous variables associated with vitamin d deficiency.
- Simple linear regression was used to identify continuous variables associated with 25OHD.

RESULTS:

The prevalence of vitamin D deficiency (≤20 ng/mL) was 44 of 365 children (12.1%) for the total sample, with 7 (1.9%) having severe deficiency (≤8 ng/mL); 146 (40.0%) had levels below an accepted optimal threshold (≤30 ng/mL). The prevalence did not differ between infants and toddlers. There was no significant difference in hormone levels or the strength of the correlation between the variable in infants vs. toddlers. The prevalence of deficiency did not vary by sex, season, time spent outdoors, sunscreen use, sun sensitivity, or skin pigmentation. Breast-feeding without supplementation was strongly associated with vitamin D deficiency among infants, with a more than 10-fold increase in risk relative to infants who were exclusively bottle-fed. A study suggest that there was decreased level of vitamin D in obese children because of deposition of fat soluble vitamin in adipose tissue Vitamin D concentration was not linked with variation of skin pigmentation or sun exposure whereas another study has reported that there is direct link of skin pigmentation with vitamin D deficiency. Infants are often swaddled in blankets or dressed in more layers of clothing than older children or teenagers, cutaneous vitamin D synthesis may have been decreased or prevented in our participants. There is strong significant association between breastfeeding and vitamin D deficiency especially in the absence of vitamin D supplementation that deserves attention. There was no association of sun exposure with $(OH)_2D$.

CONCLUSION:

A relationship was noted between the risk of vitamin D deficiency and lack of supplementation among breastfed infants and among toddlers with a higher body mass index. Among toddlers, there was a protective effect seen between milk consumption and lower risk of deficiency. These data underscore the fact that all breastfed infants should receive vitamin D supplementation for the duration of breastfeeding. However, further studies are needed to determine whether a 200 IU daily dose will provide adequate supplementation.

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