Nutritional Rickets in Georgia

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ABSTRACT. Opinions expressed in commentaries are those of the authors and not necessarily those of the American Academy of Pediatrics or its Committees. Commentaries are not peer-reviewed. ***Pediatrics*** **2001;107(4). URL: http://www.pediatrics.org/cgi/content/full/107/4/e45;** ***rickets, vitamin D, breastfeeding, undernutrition, weaning practices, vitamin D-deficient rickets.***

Carvalho and colleagues,[1] in their article entitled, "Severe Nutritional Deficiencies in Toddlers Resulting From Health Food Milk Alternatives," describe 1 case of vitamin D-deficient rickets and 1 case of kwashiorkor associated with the misuse of milk alternatives during weaning. (Milk alternatives are commercial beverages, such as unfortified soy, rice, almond, and oatmeal drinks that look like cow's milk but are not nutritionally equivalent because they do not contain comparable levels of vitamins or minerals. A milk substitute is defined as a drink that is nutritionally equivalent to cow's milk.) The American Academy of Pediatrics recommends that "infants weaned before 12 months of age should not receive cow's milk feedings but should receive iron-fortified infant formula"[2] and that after 12 months of age, a child should be weaned to whole cow's milk or a nutritionally equivalent milk substitute.[3] The article by Carvalho et al[1] points to the potential dangers of weaning children onto readily accessible milk alternatives that are not nutritionally equivalent to whole cow's milk; it also highlights the importance of parental and physician awareness of the nutritional needs of young children and the nutrient composition of products offered to children.

In response to the 2 cases reported by Carvalho et al,[1] the Georgia Department of Human Resources, Division of Public Health began an investigation in October 1999, in collaboration with the Centers for Disease Control and Prevention, to determine whether there had been additional hospitalizations in Georgia for rickets or kwashiorkor associated with the use of milk alternatives among children 6 to 59 months of age. We reviewed *Georgia Hospital Discharge Data* obtained from the Georgia Hospital Association for the period January 1997 through June 1999.[4] Data for the latter half of 1999 were not available at the time of the study. Cases reviewed for rickets were hospitalizations with *International Classification of Diseases, Ninth Revision, Clinical Modification* codes 268.0, 268.2, or 268.9, and cases reviewed for protein energy malnutrition including kwashiorkor were hospitalizations with *International Classification of Diseases, Ninth Revision, Clinical Modification* codes 260, 261, or 262 listed anywhere on the discharge record.[5] The medical records of all potential cases were obtained from the respective hospitals and reviewed. Case patients with medical conditions such as chronic renal disease as the underlying cause of rickets or nutritional deficiency were excluded. Telephone interviews with parents were conducted on the remaining primary nutritional cases because the medical records did not completely document the patients' nutritional assessment. Although we found no additional cases of rickets or kwashiorkor associated with use of milk alternatives, we did identify 5 additional cases of vitamin D-deficient rickets and 3 additional cases of protein energy malnutrition. Factors common to the rickets cases raise important issues regarding the identification and prevention of rickets in the United States.

All 5 additional vitamin D-deficient rickets cases were breastfed black male children who did not receive vitamin D supplements while breastfeeding. Several of the children shared other common risk factors for the development of vitamin D-deficient rickets, including inadequate sunlight exposure and breastfeeding >6 months without any dietary or supplemental source of vitamin D (Table 1). Three of the children were diagnosed at 8 months of age; 2 presented with symptoms of hypocalcemia (seizures not responsive to traditional therapy and acute tetany), and the other, who had a history of febrile seizure, presented with neurologic symptoms. Two were diagnosed at 21 months of age; one presented with acute respiratory distress and was diagnosed by chest radiograph and the other presented with fever and failure to bear weight and was diagnosed by a radiograph of the left tibia. Physical examination findings varied among the 5 patients and included genu varum, rachitic rosary, frontal bossing, and

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Received for publication Sep 22, 2000; accepted Sep 22, 2000.

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TABLE 1. Potential Risk Factors for Developing Rickets Among the Five Rickets Cases

Rickets Cases	1	2	3	4	5
Age at diagnosis (mo)	21	21	8	8	8
Month of birth	September	June	August	June	September
Month of diagnosis	June	March	April	February	May
Race, complexion	Black, unknown*	Black, unknown*	Black, moderate olive	Black, dark brown	Black, moderate olive
Clothing worn outside 4 wk before diagnosis	Unknown*	Unknown*	All body covered except face and hands	All body covered except head and hands	All body covered except head, arms, and hands
Sun exposure (h/wk)	Minimal	Unknown*	6–12	Minimal	21
Reason for minimal or inadequate sun exposure	2 working professional parents	Unknown*	Cold weather	Sibling <5 y, and no sidewalks	Thought to be adequate
Number of months breast milk was primary milk source	15	20	8	8	8
Pediatric vitamin D supplements	No	No	No	No	No
Maternal vitamin D intake (IU/d)	Unknown*	Unknown*	150	195	300
Maternal dietary restrictions	Unknown*	Unknown*	Dieting, restrict milk intake	None	Lactose intolerant, no red meat
Other maternal dietary issues	Unknown*	Unknown*	Low calcium intake (4 oz milk/d)	Low calcium intake (8 oz milk/d), low volume breast milk produced	Drank 30 oz rice milk/d vitamin D fortification unknown
Maternal age (y)	Unknown*	Unknown*	21	31	34
Total number of children in household (includes case)	3	3	2	2	4
Number of siblings <5 y (excluding patient)	1	Unknown*	1	1	1
Household income	Unknown*	Unknown*	$10 000–$29 999	$10 000–$29 999	$30 000–$49 999
Number of doctors	Unknown*	Unknown*	1	1	1
Immunizations up to date	No, missing 18-mo shots	Yes	No, missing 6-mo shots	Yes	Yes

* Unknown because information not in the medical record and parent not available for interview.

large anterior fontanelle. All 5 children had radiographic findings pathognomonic for rickets, including frayed and cuffed metaphyses and generalized osteopenia. Both children presenting at 21 months of age had heights and weights equal to or less than the fifth percentile; one was no longer walking and the other walked with an abnormally wide gait. None of those presenting at 8 months of age were developmentally delayed, and the 2 whose lengths were recorded had lengths >25th percentile. All 5 children had abnormally low serum 25-hydroxy vitamin D concentrations and elevated alkaline phosphatase levels. Four children had low serum calcium levels at presentation, while the fifth had a low normal level. All 4 of the children tested had elevated parathyroid hormone levels.

Our investigation was limited to cases of rickets among hospitalized patients and probably underestimated the magnitude of the problem in Georgia. Rickets often is diagnosed in outpatient clinics. In a recent North Carolina study (R. Schwartz, personal communication, 2000), ~20% of children diagnosed with vitamin D-deficient rickets were hospitalized. Because, vitamin D-deficient rickets is not a reportable medical condition, the frequency of cases among nonhospitalized patients cannot be readily determined. Likewise, among children in Georgia, the frequency of vitamin D-deficient rickets caused by the misuse of milk alternatives is unknown because no surveillance systems currently exist for monitoring illness associated with the misuse of milk alternatives. In response to legitimate concerns that the 2 cases reported by Carvalho et al[1] represented a more far-reaching problem, the Food and Drug Administration recommended that clinicians and health officials report cases of malnutrition associated with the misuse of milk alternatives through the agency's MedWatch surveillance system (www.fda.gov/medwatch/how.htm). Prevention, once again, lies in the advice of clinicians and the action of parents. The remainder of this commentary addresses: 1) risk factors for vitamin D-deficient rickets, 2) the role of vitamin D supplementation for nursing mothers, 3) the role of vitamin D supplementation in infants, and 4) the role of the clinicians in preventing vitamin D-deficient rickets.

RISK FACTORS ASSOCIATED WITH THE DEVELOPMENT OF VITAMIN D-DEFICIENT RICKETS

Inadequate sunlight exposure is the most important factor associated with the development of vitamin D-deficient rickets. In humans the main source of vitamin D comes from the ultraviolet light (290–320 nm) photo conversion of 7-dehydrocholesterol in the skin to previtamin D3, which thermally isomer-

ises to cholecalciferol (vitamin D3).[6] Serum vitamin D concentrations vary with the amount of sunlight exposure a person receives,[7] and dietary or supplemental sources of vitamin D are thought to be necessary if sunlight exposure is not adequate.[8,9]

Defining how much sun exposure is needed to prevent rickets is difficult because the amount of ultraviolet light that penetrates the skin is affected by a number of factors, including air pollution, latitude, time of day, amount of body surface covered by clothing, amount of cloud cover, and degree of skin pigmentation.[10–12] In addition, sunscreens that block ultraviolet radiation needed for photosynthesis of vitamin D may impede the photosynthesis of vitamin D in the skin.[13,14] Although white infants need 30 to 120 minutes per week of sunlight exposure, depending on clothing coverage,[15] no accurate estimates of adequate sunlight exposure exist for infants with darker skin complexion; however, their requirement will be greater because melanin competes with 7-dehydrocholesterol for ultraviolet radiation photons.[16–18] Like other recent case series of vitamin D-deficient rickets, all of our cases were black,[19,20] and only 2 of these children (cases 3 and 5) met the estimated sunlight exposure requirement for white children (Table 1).

Infants of vitamin D-deficient mothers also are at increased risk for rickets because the vitamin D stores of the newborn depend entirely on the vitamin D stores of the mother. If the mother is vitamin D-deficient, the infant will be deficient because of decreased maternal–fetal transfer of vitamin D.[21–27] Vitamin D supplementation for vitamin D-deficient pregnant women improves the infant's birth weight and subsequent linear growth.[28,29] Low maternal vitamin D intake may have played a role in the development of rickets in several of the infants who we studied (Table 1).

Infants who have inadequate sunlight exposure or who have poor stores of vitamin D at birth need adequate dietary or supplemental sources of vitamin D to prevent vitamin D-deficient rickets. The recommended daily intake of vitamin D for infants and children is 200 IU vitamin D per day.[30] Few foods naturally contain vitamin D, and those that do, such as egg yolks, fish liver oils (eg, cod liver oil), fatty fish (eg, salmon, herring, mackerel, and sardines), shrimp, and chicken liver, are not typically found in infant or toddler diets.[31] Other food items such as vitamin D-fortified cow's milk (400 IU vitamin D/L) and breakfast cereals (40–100 IU/1 cup), and commercial infant formulas (400 IU/L) contain vitamin D only because they are fortified. Infant cereals, cheese, and most commercial yogurts typically are not fortified with vitamin D. The vitamin D content of breast milk is low (12–60 IU/L) even with adequate maternal vitamin D intake.[32–35] Breastfed infants with no supplemental source of vitamin D and with limited sunlight exposure are at greater risk for developing vitamin D-deficient rickets.[36] Prolonged breastfeeding without sufficient dietary or supplementary sources of vitamin D may have contributed to the development of rickets in our cases (Table 1).

Because of lack of information about the amount of sun exposure needed to prevent rickets in all children in our diverse population and concerns about excessive sun exposure causing skin cancer, all exclusively or predominantly breastfed infants and toddlers without a significant dietary source of vitamin D may need a vitamin D supplement.

PREVENTION OF VITAMIN D-DEFICIENT RICKETS BY SUPPLEMENTATION FOR BREASTFEEDING WOMEN

The vitamin D content of breast milk varies with maternal vitamin D intake.[15,29] Infant serum 25-hydroxyvitamin D concentration does not correlate with breast milk 25-hydroxyvitamin D levels unless the mother receives high doses of vitamin D. Supplementation of nursing mothers' diet with 2000 IU per day of vitamin D may be needed to normalize vitamin D metabolites of breastfed infants who do not get adequate sunlight exposure.[37,38] Although this dose is within the tolerable intake levels outlined by National Academy of Sciences in 1997, studies have not been conducted to determine the safety of this level of supplementation in lactating women over a period >3 months.[30,39] Therefore, giving 2000 IU of vitamin D daily to breastfeeding women cannot be endorsed.

UNIVERSAL VITAMIN D SUPPLEMENTATION OF BREASTFED CHILDREN

The most recent policy statement of the American Academy of Pediatrics on breastfeeding states that "vitamin D may need to be given before 6 months of age in selected groups of infants (for infants whose mothers are vitamin D-deficient or those infants not exposed to adequate sunlight)".[2] Therefore, the current standard of practice is for clinicians to identify breastfeeding infants at risk of developing vitamin D-deficient rickets and supplement on a case-by-case basis. However, as noted above, defining inadequate sunlight exposure is complex and there are no evidence-based guidelines for sunlight adequacy, with the exception of the recommendation made by Specker et al[15] for white infants in Cincinnati, Ohio. Moreover, the risk of overdose leading to hypervitaminosis D is low, complications of excess vitamin D do not occur unless the daily dose exceeds at least 2400 IU, and there are no known long-term risks from supplementing infants' diets with up to 400 IU vitamin D per day.[30,39] New studies suggest that there even may be some long-term benefits of vitamin D supplementation for infants.[40–42] Still, some oppose universal vitamin D supplementation of breastfed infants because few unsupplemented, exclusively or predominately breastfed infants develop clinically apparent vitamin D-deficient rickets.

THE CLINICIAN'S ROLE IN THE PREVENTION OF VITAMIN D-DEFICIENT RICKETS

1. The diagnosis of vitamin D-deficient rickets should be considered in patients who present with signs and symptoms of hypocalcemia, including muscle cramps, numbness, perioral or digital paresthesias, acute tetany, seizures, hyperreflexia, laryngospasm, or a prolonged QT inter-

val on electrocardiogram.[43] Two of the cases that we reviewed involved infants who presented with signs of hypocalcemia, and the diagnosis of vitamin D-deficient rickets was delayed in case 4 because of a failure to recognize hypocalemia as a cause of seizures. Rickets also should be added to the differential diagnosis of a child with hypotonia, growth failure, and delayed motor development.[44] The diagnosis of vitamin D-deficient rickets was delayed in patients 1 and 2, and other case reports[18,47] despite having routine pediatric care, because their providers failed to recognize the early manifestations of rickets.

2. Clinicians should educate parents about the risk factors for vitamin D-deficient rickets, including inadequate sunlight exposure, and should provide information on dietary sources of vitamin D. Formula-fed infants given at least one half liter (or approximately two 8-oz bottles) of infant formula daily meet the new daily recommended intake of vitamin D (200 IU/day), as do children consuming the same quantity of vitamin D-fortified whole cow's milk. For the breastfed infant not being given any infant formula or the toddler not being given any vitamin D-fortified whole cow's milk or milk substitute, additional dietary or supplemental sources of vitamin D must be provided.

CONCLUSION

Vitamin D is essential for optimal growth and development in infancy and childhood. Unfortunately, sunlight exposure, the major determinant of a child's vitamin D status, cannot be easily assessed and the minimum amount needed to prevent rickets is unknown. Moreover, increased sunlight exposure cannot be recommended because of the risk of skin cancer. The scientific literature suggests the need for universal vitamin D supplementation for all exclusively or predominately breastfed infants. These data also raise concerns about the vitamin D status of toddlers in the United States. Although the magnitude of vitamin D deficiency is unknown, prevalence data could be collected by a national pediatric serosurvey of serum vitamin D and parathyroid hormone levels or by sentinel surveillance at a sample of pediatric and endocrine clinics across the United States. We support further research while discussions continue on universal vitamin D supplementation for exclusively or predominately breastfed infants in the United States.

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Pediatrics 2001;107;e45
DOI: 10.1542/peds.107.4.e45

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