RICKETS IN RURAL KENYAN PRESCHOOL CHILDREN: CASE REPORT

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SUMMARY

Clinical rickets has not been reported previously in Embu district, Kenya. Baseline clinical assessments performed for a nutrition intervention study in preschool children (n=324) identified 28 cases of rickets (8.6% of study sample). Clinical characteristics included: delays of sitting, walking, and teething; bone and chest deformities; widened wrists and ankles; and bowed lower extremities. Risk factors identified were short duration of breastfeeding with feeding of cereal-based supplements with little or no milk, low calcium intake, limited sunlight exposure. Vitamin D and calcium deficiencies likely contributed to these cases. Treatment with Vitamin D3 and milk resulted in clinical improvement.

INTRODUCTION

The first reported cases of rickets in Kenya were documented from 1926–1930 at a hospital in central Kenya (1). With the discovery of the active substance vitamin D, rickets was largely eliminated via preventive fortification of milk and infant formulas, therapeutic vitamin D, and sun exposure (2). However, a resurgence of rickets has been reported in northern latitudes, cold climates, and even in southern latitudes with ample sunshine (3, 4). Multiple factors contribute to rickets including inadequate calcium intake, particularly from milk and milk products; little to no animal source food (ASF) intake; vitamin D deficiency; high-fibre intake; and low sun exposure (5, 6). Classical infantile or nutritional rickets has been attributed to vitamin D deficiency and has been observed in temperate climates during winter months when sunlight exposure is limited (7). In Embu, Kenya, young children are often kept indoors and/or heavily clothed due to concerns about cleanliness, safety, and warmth.

This report documents rickets found among children during baseline assessments for the Child Survival and Nutrition Intervention Study (CSNIS). The study recruited preschool children aged 1-4 years (n=324) from Embu district, Kenya, in the foothills of Mount Kenya with elevations from 1200 to 1460 m. Most children were younger siblings of schoolchildren participating in the Child Nutrition Project (CNP) (8). Anthropometry, morbidity data, and food intake data based on 24 hour maternal recall were collected monthly. Biochemical or radiological investigations were not performed. Clinical assessment performed by a clinician included a health history to evaluate past health status and clinical examination. Approval by the University of California, Los Angeles Human Subjects Protection Committee, the Ethics Committee of the University of Nairobi, School of Medicine, and the Office of the President of Kenya was obtained, with written informed consent or oral witnessed informed consent obtained from mothers. Community permission was also obtained from the District Commissioner and from the community as a whole during meetings convened by the chiefs and sub-chiefs.

CASE REPORT

The baseline clinical assessments revealed clinical rickets in 28 (8.6%) children, including 3 sets of twins. Children ranged from 12 to 38 months old; 49% were boys (Figure 1). Clinical characteristics observed in diagnosed cases included: developmental delays of sitting, walking, and teething; bone deformities such as frontal and parietal bossing of the skull; chest deformities with rachitic rosaries and Harrison groove; widening of wrists and ankles; and bowing of lower extremities (Figures 2, 3, and 4).
Figure 1
*Children with rickets by age and gender*

![Bar chart showing the number of cases of rickets by age and gender.](chart)

**Figure 2**
*Arms and legs of young boy with rickets*

![Image of a young boy with rickets affecting his arms and legs.](image)

**Figure 3**
*Arm of young boy with rickets*

![Image of a young boy with rickets affecting his arm.](image)
Children with rickets shared: short duration of breastfeeding (<4 months), weaning onto unfortified cereal-based complementary foods with negligible cow or goat milk consumption, and kept indoors while mothers cultivated fields. When outdoors children were often heavily clothed and carried wrapped in shawls on their mother’s back. One set of twins was prematurely delivered. Children had low mean baseline calcium intake (234 ± 44 mg/day) compared with the recommended daily intake levels of 500 mg for one to two year-olds and 800 mg for three to eight year-olds (9). They also had high phytate intake (6216 ± 7342 mg/day).

Treatment of affected children consisted of ultra-heat treated (UHT) milk and vitamin D3 tablets (500 mg/day) given crushed and mixed with water. Mothers were advised to increase the child’s home milk intake and to expose them to sunshine daily. Mothers not culturally opposed to eating fish were advised to provide fish soup and/or minced fish. Children were followed weekly by a clinical nurse. Nearly all showed improvement during the first month of treatment, and features of rickets disappeared or diminished by 3–6 months of treatment for most children.

DISCUSSION

This observation of rickets cases among these preschool children is significant since visible characteristics of rickets were not observed in any age group of Embu children during the Nutrition Collaborative Research Support Project (NCRSP) study in Embu district during the 1980s or in Embu school-aged children during the CNP from 1998-2000. In the earlier NCRSP study, children were rarely given ASF and had little vitamin D intake (10). Breastfeeding was rarely exclusive after three months; 90% received supplemental foods by three months (10). Subsequently, the CNP and CSNIS projects documented low calcium intake in children.

In Embu toddlers, milk is the main dietary source of calcium, but CSNIS toddlers consumed less milk than NCRSP children. The prolonged and severe drought during the CSNIS may account for less dairy intake than in the mid-1980s. Also the economic situation was worse, and the price of milk had increased.

Weaning onto cereal porridges, with little to no ASF intake, limits toddler’s vitamin D and calcium intake. Weaning foods were often cereal-based and contained high levels of phytate and oxalates that form complexes with calcium, reducing its bioavailability. High phytate intake was observed in both CNP school children (8) and these preschool children. Phytate and oxalates probably played a role in inhibiting calcium absorption. Lack of sun exposure also may have been a contributing factor, especially since no cases were found in the CNP school children who had sunlight exposure on the way to and from school and during school-play.

Health workers providing health care to children should be aware of the clinical presentation of rickets with bony deformities of frontal bossing, widening of wrists, and bowing of forearms or tibias, chest deformities, and delayed motor development to provide early diagnosis and appropriate treatment. History of breastfeeding, intake of ASF, and exposure to sunlight should be assessed. Management of children with rickets should include Vitamin D3 administration, calcium supplementation, and sunlight exposure. Provision of milk and dairy products is important. Where culturally acceptable and available, fatty fish should be added to the diet. Parents should be advised about preventive measures such as breastfeeding with adequate nutrition for the mother, weaning to animal dairy foods, and daily sunlight exposure.

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