

Vitamin D and Calcium: The Synergistic Players!

Shinky Mehta

Dr BSA Medical College and Hospital, New Delhi,
India

Kuldip Singh Khokhar¹, Shinky Mehta^{2*} and Aditi Khokhar³

¹World College of Medical Sciences, Haryana, India

²Dr BSA Medical College and Hospital, New Delhi, India

³Department of Pediatrics, Rutgers New Jersey Medical School, USA

COLUMN ARTICLE

Rickets is defined as poor mineralization of the growth plate [1]. Osteomalacia is abnormal matrix mineralization in established bone. Although present in children with rickets, it is used to describe bone mineralization defects after completion of growth [2]. Young children can develop rickets if their calcium or phosphate requirements are not met during periods of active growth.

Nutritional vitamin D deficiency has been increasingly reported in high- and low-income countries. High-income countries have observed a resurgence of nutritional vitamin D deficiency, mainly among immigrants of African, Asian, or Middle-Eastern origin. The incidence among established Caucasian populations is stable or decreasing. Risk factors for vitamin D deficiency include breast-feeding without vitamin D supplementation, maternal vitamin D deficiency, dark skin pigmentation, and inadequate exposure to sunlight. Individuals with naturally dark skin tone require at least three to five times longer exposure to make the same amount of vitamin D compared to the white skin tone persons. Other risk factors include obesity, fat malabsorption syndromes, nephrotic syndrome, and use of anticonvulsants and antiretroviral therapies [3].

Clinical features of rickets in infants and young children include delayed fontanelle closure, craniotabes, swelling of wrists and ankles, frontal bossing, rachitic rosary and delayed tooth eruption (no incisors by the age 10 months or no molars by 18 months). As children start walking they may develop lower extremity deformities such as genu varum, genu valgum or windswept deformity. Severe deficiency can lead to bone pain, restlessness, and irritability. Other rare and severe manifestations include hypocalcemic seizure and tetany, hypocalcemic dilated cardiomyopathy, raised intracranial pressure, failure to thrive and poor linear growth, delayed gross motor development with muscle weakness. Radiographic features are splaying, fraying, cupping, and coarse trabecular pattern of metaphysis, widening of the growth plates, osteopenia and minimal trauma fracture [4].

The diagnosis of nutritional rickets is made on the basis of history, physical examination, and biochemical testing and is confirmed by radiographs. Biochemical testing alone is not sufficient to diagnose nutritional rickets and may not differentiate whether the primary cause is vitamin D or dietary calcium deficiency because combined deficiencies are common. Most children with vitamin D deficiency are asymptomatic, highlighting the interplay between serum

Citation: Shinky Mehta., et al. "Vitamin D and Calcium: The Synergistic Players!". EC Paediatrics ECO.02 (2019): 19-20.

25 hydroxy vitamin D level and dietary calcium intake in maintaining serum calcium concentrations and bone integrity. In developing countries where calcium intake is characteristically very low, with few or no dairy products, dietary calcium deficiency is the main cause of nutritional rickets among children outside the infant age group. Dietary calcium deficiency is diagnosed by obtaining a calcium intake history. Because the sources of calcium will vary by country and region, it is recommended that clinicians develop a dietary calcium intake questionnaire specific to their country or region [5].

Vitamin D supplementation and adequate calcium intake are vital for prevention of rickets and osteomalacia. From birth to 12 months of age all infants should receive 400 IU/day of vitamin D supplementation orally independent of their mode of feeding [6]. Beyond 12 months of age, all children and adults need to meet their nutritional requirement for vitamin D through diet and/or supplementation, which is at least 600 IU/day. Complementary foods introduced no later than 26 weeks should include sources rich in calcium. Recommendations for dietary calcium intake to prevent rickets are 200 and 260 mg/day for infants 0 - 6 and 6 - 12 months of age respectively and > 500 mg/day for children over 12 months of age [5].

Nutritional rickets, although a treatable entity, should be prevented by regular vitamin D supplementation and adequate calcium intake in diet as it can lead to severe manifestations. Pediatricians should educate families about the importance of vitamin D supplementation at each health visit. Awareness of this entity among general patient population can prevent its related morbidity and mortality.

4. Rauch F. "The rachitic bone". *Endocrine Development* 6 (2003): 69-79.
5. Munns CF, *et al.* "Global Consensus Recommendations on Prevention and Management of Nutritional Rickets". *Hormone Research in Paediatrics* 85.2 (2016): 83-106.
6. Gallo S, *et al.* "Effect of different dosages of oral vitamin D supplementation on vitamin D status in healthy, breastfed infants: a randomized trial". *Journal of the American Medical Association* 309.17 (2013): 1785-1792.

©All rights reserved by Shinky Mehta., et al.

BIBLIOGRAPHY

1. Van Schoor NM, *et al.* "Vitamin D deficiency as a risk factor for osteoporotic fractures". *Bone* 42.2 (2008): 260-266.
2. Pitt MJ. "Rickets and osteomalacia are still around". *Radiologic Clinics of North America* 29.1 (1991): 97-118.
3. Khokhar A, *et al.* "Genetic disorders of vitamin D metabolism". *Clinical Pediatrics* 55.5 (2015): 404-414.