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LETTER TO EDITOR (VIEWERS CHOICE)

IS NUTRITIONAL RICKETS A DISEASE FROM THE PAST?

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Rickets is a disorder of bone mineralization caused by altered phosphocalcium metabolism, leading to impaired growth plate development and structural deformities.[1] Nutritional rickets, primarily due to vitamin D and calcium deficiency, remains a significant pediatric health issue worldwide, particularly among high-risk populations.[2] Despite being preventable, cases continue to emerge, emphasizing the need for awareness and early intervention.

We report two cases that highlight the clinical variability of nutritional rickets and emphasize the importance of early recognition and comprehensive management for better outcomes.

A 3-year-old child from Pakistan presented with progressive gait abnormalities and lower limb weakness. Dietary intake of dairy products and vegetables was inadequate. No vitamin D supplementation was provided during the first year of life. Clinical assessment showed a waddling gait, pelvic instability, proximal lower limb weakness, reduced reflexes, bilateral wrist widening, and leg bowing. Biochemical findings revealed hypocalcemia, hypophosphatemia, secondary hyperparathyroidism, and undetectable vitamin D levels. Radiographs demonstrated widened metaphysis, confirming rickets. Another 15-month-old infant of African descent presented with failure to thrive, iron-deficiency anemia, hypocalcemia, hypophosphatemia, and vitamin D deficiency but showed no radiological abnormalities. The patient was exclusively breastfed for 9 months and her diet lacked adequate dairy, green vegetables, and animal protein. Both cases responded favorably to dietary improvements, high-dose cholecalciferol, and calcium supplementation, with progressive recovery and normalization of the above-mentioned biochemical abnormalities.

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The presentation of nutritional rickets depends on the age of onset, duration of deficiency, and severity of metabolic disruption.[2,3] Calcipenic rickets, the most common variant, results from inadequate vitamin D, leading to impaired intestinal calcium and phosphorus absorption.[4] While dietary intake and supplementation play crucial roles, sun exposure remains the predominant source of vitamin D.[5] Beyond skeletal deformities, rickets may cause muscle weakness, hypotonia, motor delays, and seizures, highlighting its systemic impact.[6,7]

The increasing incidence of nutritional rickets among ethnic minorities and migrant populations raises public health concerns.[8] Factors such as darker skin pigmentation, limited sun exposure, maternal vitamin D deficiency, and prolonged exclusive breastfeeding contribute to higher risk.[9] Studies indicate that children from immigrant backgrounds have a greater likelihood of developing rickets compared to non-migrant peers.[10] Additionally, refugees and displaced individuals face multiple nutritional challenges, exacerbating deficiency-related disorders.[11]

Effective preventive strategies, including universal vitamin D supplementation in infancy and food fortification policies, have demonstrated success in reducing rickets incidence.[12] In high-risk populations, extending supplementation beyond infancy may be warranted. Strengthening public health initiatives through improved education on dietary intake, sun exposure, and supplementation can mitigate the burden of nutritional rickets.

The resurgence of this preventable condition in sun-rich regions underscores the importance of proactive measures. Integrating vitamin D supplementation programs, nutritional education, and targeted health surveillance can aid in early detection and prevention. Collaborative efforts between healthcare providers, policymakers, and community stakeholders are essential to reducing rickets prevalence and ensuring optimal pediatric bone health.

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