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Early Detection Of Rickets In Children Under Two Years And Prevention Of Its Complications: Clinical And Preventive Perspectives

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Abstract

Rickets remains a preventable yet persistent pediatric metabolic disorder characterized by defective mineralization of the growing skeleton. The disease is particularly critical in children under two years of age due to rapid bone development and increased nutritional demands. Despite well-established preventive strategies, delayed diagnosis continues to result in irreversible skeletal deformities and long-term functional impairment. This review critically examines the pathophysiological mechanisms underlying early rickets development, evaluates current diagnostic approaches, and discusses preventive strategies aimed at minimizing disease burden. Evidence indicates that biochemical alterations precede clinical manifestations, highlighting the importance of early screening. Timely intervention through vitamin D supplementation, nutritional optimization, and maternal health management significantly reduces complications. The findings emphasize that effective control of rickets depends more on early detection and prevention than on treatment of advanced disease.

Keywords: Rickets, vitamin D deficiency, early diagnosis, pediatric bone health, prevention, infant nutrition.

Introduction

Rickets is a disorder of impaired bone mineralization that occurs during periods of rapid skeletal growth, primarily affecting infants and young children. Although largely preventable, it continues to pose a significant public health challenge, particularly in regions where nutritional deficiencies and limited sunlight exposure are prevalent [1]. The persistence of rickets in modern healthcare systems reflects not a lack of treatment options, but a failure in early detection and preventive strategies. The disease develops gradually, often beginning with subclinical metabolic disturbances before progressing to overt skeletal abnormalities. In children under two years, this progression is particularly rapid due to accelerated bone growth and high calcium requirements. Clinical recognition at advanced stages when deformities such as genu varum or rachitic rosary appear indicates that the underlying metabolic imbalance has been present for a prolonged period [2]. A critical issue is that early manifestations of rickets are nonspecific. Symptoms such as irritability, excessive sweating, or delayed motor development are frequently overlooked or attributed to other causes. As a result, diagnosis is often delayed until structural bone changes become evident, at which point complete reversal is unlikely.

From a preventive perspective, rickets should be understood as a systemic nutritional and metabolic disorder influenced by maternal health, infant feeding practices, and environmental factors. Addressing these determinants requires integration of clinical screening with public health

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strategies. Therefore, early detection is not merely a diagnostic goal but a central component of disease prevention.

Pathophysiology of Early Rickets Development

The development of rickets is fundamentally linked to disturbances in vitamin D metabolism and calcium–phosphorus homeostasis. Vitamin D facilitates intestinal absorption of calcium and phosphorus, both of which are essential for bone mineralization. In its deficiency, calcium absorption decreases, leading to hypocalcemia and subsequent activation of parathyroid hormone (PTH) [3]. Secondary hyperparathyroidism attempts to maintain serum calcium levels by increasing bone resorption and reducing renal calcium excretion. However, this compensatory mechanism disrupts normal bone formation by impairing mineral deposition at the growth plate. The result is an accumulation of unmineralized osteoid tissue and widening of the epiphyseal plate [4]. Importantly, these biochemical disturbances occur well before clinical symptoms become apparent. Studies demonstrate that serum 25-hydroxyvitamin D levels decline significantly prior to detectable skeletal changes, making biochemical screening a critical tool for early diagnosis [5]. Elevated alkaline phosphatase levels, reflecting increased osteoblastic activity, often serve as an early marker of bone turnover imbalance [6].

Maternal vitamin D status plays a decisive role in early infancy. Neonates depend largely on maternal vitamin D stores, and deficiency during pregnancy predisposes infants to early-onset rickets. This relationship highlights the intergenerational nature of the disease and underscores the need for maternal screening programs [7]. Additionally, exclusive breastfeeding without vitamin D supplementation has been identified as a significant risk factor. While breast milk provides optimal nutrition in most aspects, its vitamin D content is insufficient to meet infant requirements, particularly in regions with limited sunlight exposure [8].

Thus, the pathophysiology of rickets supports the concept that early detection must focus on metabolic indicators rather than structural abnormalities.

Stage	Biological Changes	Clinical Presentation	Detection Strategy
Normal	Adequate mineralization	Healthy	Routine monitoring
Deficiency (subclinical)	Low vitamin D	No symptoms	Biochemical tests
Early rickets	Mineral imbalance	Mild nonspecific signs	Lab screening
Active rickets	Growth plate disruption	Bone deformities begin	Clinical + imaging
Advanced rickets	Severe structural damage	Obvious deformities	Late diagnosis

Table 1. Stages of Rickets Development and Detection Potential

Early Detection Strategies

Early detection of rickets requires a shift from symptom-based diagnosis to proactive screening. Reliance on physical examination alone is inadequate, as visible skeletal deformities represent late-stage disease. Serum 25(OH)D concentration is widely accepted as the most reliable indicator of vitamin D status. Levels below established thresholds are strongly associated with increased risk of rickets and related complications [5]. However, vitamin D measurement should be interpreted

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alongside other biochemical markers, including calcium, phosphorus, and alkaline phosphatase, to provide a comprehensive metabolic profile [6].

Screening strategies should prioritize high-risk populations. These include infants with limited sunlight exposure, those born to vitamin D–deficient mothers, premature infants, and exclusively breastfed infants without supplementation [8,9]. Early identification in these groups allows timely intervention before structural damage occurs.

Radiological imaging, while useful for confirming diagnosis, has limited value in early detection because skeletal changes appear only after prolonged deficiency. Therefore, its role should be considered supplementary rather than primary.

Emerging research suggests that integrating nutritional, environmental, and biochemical data into predictive models may enhance early detection. Although still under development, such approaches reflect a broader trend toward preventive and personalized medicine.

Method	Role	Strength	Limitation
Serum 25(OH)D	Primary marker	High sensitivity	Cost
Alkaline phosphatase	Early bone turnover	Detects early change	Non-specific
Calcium/phosphorus	Mineral balance	Widely available	Less sensitive early
X-ray	Structural assessment	Confirms disease	Late-stage

Table 2. Diagnostic Tools for Early Rickets Detection

Prevention of Complications

The prevention of rickets-related complications depends primarily on early intervention. Once skeletal deformities develop, treatment becomes more complex and outcomes less predictable. Vitamin D supplementation remains the most effective preventive strategy. Clinical guidelines recommend routine supplementation during infancy, particularly for breastfed infants, to ensure adequate levels for bone mineralization [9]. Adequate calcium intake must accompany vitamin D to achieve optimal results, as deficiency in either component can impair bone development [3].

Sunlight exposure contributes significantly to endogenous vitamin D synthesis. However, reliance on sunlight alone is insufficient due to variability in geographic location, seasonal changes, and cultural practices limiting exposure [1]. Untreated rickets can lead to multiple complications, including growth retardation, skeletal deformities, delayed motor development, and increased fracture risk. Early correction of metabolic imbalance prevents these outcomes and supports normal growth patterns [10].

Complication	Mechanism	Prevention
Bone deformities	Poor mineralization	Early supplementation
Growth delay	Chronic deficiency	Nutrition + monitoring
Muscle weakness	Calcium imbalance	Vitamin D therapy
Fractures	Reduced bone strength	Early diagnosis

Table 3. Complications and Preventive Measures

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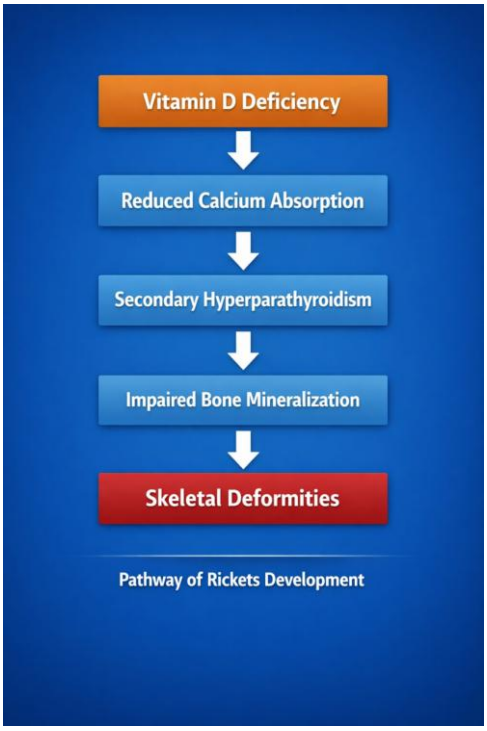

Figure 1. Pathway of Rickets Development

Public Health and Future Directions

The persistence of rickets reflects broader systemic issues in healthcare delivery and nutrition. Prevention requires coordinated efforts at both clinical and population levels. Public health strategies should include maternal supplementation programs, routine pediatric screening, and caregiver education regarding infant nutrition. Evidence indicates that such interventions significantly reduce disease prevalence in high-risk populations [7,9]. Future approaches may incorporate predictive health models capable of identifying at-risk individuals before biochemical deficiency occurs. These strategies align with global trends toward preventive healthcare and personalized medicine.

Conclusion

Early detection of rickets in children under two years is essential for preventing irreversible skeletal damage and long-term health consequences. The disease begins as a metabolic disturbance long before clinical symptoms appear, making biochemical screening the most effective diagnostic approach. Preventive strategies including vitamin D supplementation, adequate nutrition, and maternal health management offer a highly effective means of reducing disease burden. Shifting focus from late-stage treatment to early identification and prevention remains the most critical step in controlling rickets globally.

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