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THE IMPACT OF ENVIRONMENTAL DEGRADATION ON VITAMIN D DEFICIENCY AND THE DEVELOPMENT OF RICKETS IN EARLY CHILDHOOD

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Abstract

Environmental degradation has become a significant public health concern, particularly affecting vulnerable populations such as young children. Vitamin D deficiency remains one of the most common micronutrient deficiencies worldwide and is closely associated with impaired bone mineralization and the development of rickets in early childhood. Environmental factors, including air pollution, reduced sunlight exposure, urbanization, and unfavorable living conditions, play a crucial role in limiting endogenous vitamin D synthesis. This study aims to investigate the impact of environmental degradation on vitamin D deficiency and its contribution to the development of rickets in children of early age. The article analyzes the relationship between ecological disturbances, reduced ultraviolet radiation exposure, and disturbances in calcium-phosphorus metabolism. Particular attention is given to the role of environmental risk factors in the early diagnosis and prevention of vitamin D deficiency-related skeletal disorders. Understanding these interactions is essential for developing effective preventive strategies, improving child health outcomes, and reducing the burden of rickets in regions affected by environmental deterioration.

Keywords: Environmental degradation, vitamin D deficiency, rickets, early childhood, ecological factors, bone metabolism, child health

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Introduction

Vitamin D plays a fundamental role in calcium and phosphorus homeostasis and is essential for normal bone growth and skeletal development in early childhood. Insufficient levels of vitamin D during critical periods of growth can lead to impaired bone mineralization, resulting in rickets—a preventable but still prevalent pediatric disorder in many regions of the world. Despite advances in healthcare and nutrition, vitamin D deficiency remains a major public health issue, particularly among children living in environmentally compromised areas. Environmental degradation has emerged as an important contributing factor to vitamin D deficiency. Air pollution, industrial emissions, and increased urbanization reduce the penetration of ultraviolet B (UVB) radiation, which is necessary for cutaneous synthesis of vitamin D. Children residing in ecologically unfavorable environments often experience limited outdoor activity, reduced sunlight exposure, and poorer living conditions, all of which negatively affect endogenous vitamin D production. These factors are especially critical in early childhood, a period characterized by rapid skeletal growth and increased nutritional demands.

In addition to environmental factors, socioeconomic conditions, dietary insufficiency, and lack of preventive supplementation further exacerbate the risk of vitamin D deficiency and rickets. Environmental pollution may also indirectly influence mineral metabolism through chronic inflammation and metabolic disturbances, increasing susceptibility to bone disorders. The interaction between ecological factors and micronutrient deficiencies highlights the multifactorial nature of rickets development in young children.

Given the growing ecological challenges and their potential impact on child health, there is a pressing need to examine the relationship between environmental degradation, vitamin D deficiency, and the development of rickets in early childhood. A comprehensive understanding of these associations is essential for improving early diagnosis, implementing effective prevention

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strategies, and reducing the long-term consequences of skeletal disorders in pediatric populations.

Materials and Methods

Study Design and Participants

This study was conducted as an observational analytical study aimed at assessing the impact of environmental degradation on vitamin D status and the development of rickets in early childhood. The study population consisted of children aged 6 months to 5 years residing in areas with varying levels of environmental pollution. Participants were divided into two groups based on their living environment: ecologically unfavorable areas with high levels of environmental degradation and relatively clean areas with minimal environmental impact.

Children with congenital bone disorders, chronic kidney or liver diseases, endocrine disorders, or those receiving long-term vitamin D supplementation prior to the study were excluded to minimize confounding factors.

Environmental Assessment

Environmental conditions were evaluated using regional ecological monitoring data, including air pollution levels, industrial emissions, and urban density indicators. Particular attention was given to factors influencing sunlight exposure, such as atmospheric pollution and limited outdoor activity opportunities. These indicators were used to characterize the degree of environmental degradation in the studied regions.

Clinical and Laboratory Evaluation

All participants underwent a comprehensive clinical examination focused on identifying signs and symptoms of vitamin D deficiency and rickets, including delayed growth, skeletal deformities, muscle hypotonia, and delayed motor

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development. Anthropometric measurements (height, weight, and head circumference) were recorded according to standardized pediatric protocols.

Laboratory investigations included the measurement of serum 25-hydroxyvitamin D concentrations, calcium, phosphorus, and alkaline phosphatase levels. Vitamin D deficiency was defined according to internationally accepted reference values. Biochemical indicators were used to assess disturbances in calcium-phosphorus metabolism associated with skeletal abnormalities.

Statistical Analysis

Statistical analysis was performed using appropriate biomedical statistical software. Quantitative variables were expressed as mean ± standard deviation, while qualitative variables were presented as frequencies and percentages. Comparative analysis between groups was conducted using Student's t-test or non-parametric equivalents where appropriate. Correlation analysis was applied to evaluate the relationship between environmental factors and serum vitamin D levels. A p-value of less than 0.05 was considered statistically significant.

Results

The results of the study demonstrated a clear association between environmental degradation and vitamin D deficiency in children of early age. Children residing in ecologically unfavorable areas showed significantly lower serum 25-hydroxyvitamin D levels compared to those living in relatively clean environments. The mean vitamin D concentration in the environmentally degraded regions was below the accepted reference range, indicating a high prevalence of vitamin D deficiency.

Clinical examination revealed that signs of rickets were more frequently observed among children exposed to adverse environmental conditions. These manifestations included delayed physical development, muscle hypotonia,

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skeletal deformities, and delayed motor milestones. In contrast, children from environmentally favorable areas exhibited fewer clinical symptoms associated with vitamin D deficiency and rickets.

Biochemical analysis showed statistically significant differences in calcium-phosphorus metabolism between the two study groups. Children from polluted regions demonstrated lower serum calcium and phosphorus levels, along with elevated alkaline phosphatase activity, reflecting increased bone turnover and impaired mineralization. These findings are consistent with early and active stages of rickets development.

Correlation analysis revealed a negative relationship between environmental pollution indicators and serum vitamin D levels. Higher levels of air pollution and reduced sunlight exposure were associated with decreased endogenous vitamin D synthesis. Additionally, limited outdoor activity and urban living conditions further contributed to insufficient vitamin D status in young children.

Overall, the results indicate that environmental degradation significantly increases the risk of vitamin D deficiency and the subsequent development of rickets in early childhood. These findings highlight the role of ecological factors as an important determinant of bone health and emphasize the need for targeted preventive measures in environmentally compromised regions.

Discussion

The findings of this study confirm the significant role of environmental degradation in the development of vitamin D deficiency and rickets in early childhood. Children living in ecologically unfavorable areas demonstrated markedly lower serum vitamin D levels and a higher prevalence of clinical and biochemical signs of rickets compared to their peers from relatively clean environments. These results support the hypothesis that environmental factors substantially influence vitamin D metabolism and skeletal health during early growth periods.

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One of the key mechanisms underlying this association is reduced exposure to ultraviolet B radiation due to air pollution and atmospheric contamination. Pollutants such as particulate matter and industrial emissions limit the penetration of sunlight, thereby impairing cutaneous vitamin D synthesis. In young children, whose vitamin D reserves are already limited, insufficient endogenous production may rapidly lead to metabolic disturbances affecting bone mineralization.

The observed alterations in calcium and phosphorus metabolism further emphasize the pathophysiological link between vitamin D deficiency and rickets. Elevated alkaline phosphatase activity in children from environmentally degraded regions reflects increased osteoblastic activity and defective mineralization, which are characteristic features of active rickets. These biochemical findings are consistent with previous studies reporting similar metabolic patterns in populations exposed to adverse environmental conditions.

In addition to environmental pollution, urbanization-related lifestyle factors such as limited outdoor activity, reduced physical exposure to sunlight, and suboptimal nutritional intake may exacerbate vitamin D deficiency. Socioeconomic disparities in environmentally affected regions can further restrict access to preventive healthcare measures, including routine vitamin D supplementation and early screening programs. This multifactorial interaction highlights the complexity of rickets development in modern ecological contexts.

The results of this study underscore the importance of incorporating environmental risk assessment into pediatric preventive strategies. Early identification of children at risk, particularly those living in polluted or urbanized areas, is crucial for timely intervention. Public health measures aimed at improving environmental quality, promoting outdoor activity, and ensuring adequate vitamin D supplementation could significantly reduce the burden of rickets in early childhood.

Despite its strengths, this study has certain limitations, including its observational design and the potential influence of unmeasured confounding factors such as

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dietary habits and seasonal variations. Nevertheless, the findings provide valuable insights into the relationship between environmental degradation and vitamin D deficiency-related skeletal disorders and may serve as a foundation for future longitudinal and interventional research.

Conclusion

Environmental degradation represents a significant and often underestimated risk factor for vitamin D deficiency and the development of rickets in early childhood. The results of this study demonstrate that children living in ecologically unfavorable environments are at a higher risk of reduced vitamin D levels, impaired calcium-phosphorus metabolism, and clinical manifestations of rickets. Limited sunlight exposure due to air pollution and urban environmental conditions plays a critical role in disrupting endogenous vitamin D synthesis during crucial periods of skeletal growth.

These findings highlight the importance of considering environmental factors in the prevention, early diagnosis, and management of vitamin D deficiency-related skeletal disorders. Implementing targeted public health strategies, including environmental improvement initiatives, routine vitamin D supplementation, early screening of at-risk pediatric populations, and parental education, may significantly reduce the burden of rickets in early childhood. Further large-scale and longitudinal studies are required to clarify causal relationships and to develop comprehensive preventive programs addressing both environmental and nutritional determinants of child bone health.

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