IJNRD.ORG ISSN : 2456-4184
INTERNATIONAL JOURNAL OF NOVEL RESEARCH AND DEVELOPMENT (IJNRD) | IJNRD.ORG
An International Open Access, Peer-reviewed, Refereed Journal

A Study Of Children With Rickets In Kyrgyzstan And India In 4th Year Student Of The Faculty Of Medicine

Author - Tajibaeva Feruzakhan (Professor- PhD)

Student - Kanika, Megha kshirsagar, Rishikesh, Saurabh, Aniket

Abstract

Rickets is a childhood bone disease caused by impaired mineralization, leading to deformities, growth failure, and bone pain. The most common cause is vitamin D deficiency, though calcium deficiency, phosphate abnormalities, chronic diseases, and genetic defects also contribute. Risk factors include exclusive breastfeeding without supplementation, maternal vitamin D deficiency, dark skin, limited sunlight exposure, and low dietary calcium. Diagnosis is based on clinical features, radiographs, and biochemical tests. Treatment depends on cause—nutritional rickets responds to vitamin D and calcium, while genetic forms may require active vitamin D metabolites, phosphate, or targeted therapy such as burosumab. Prevention through supplementation, food fortification, and maternal care is key, making eradication of nutritional rickets a realistic global health goal.

Keywords

Rickets; Vitamin D deficiency; Calcium deficiency; Hypophosphatemia; Bone mineralization; Children; Nutritional rickets; Global health; Prevention.

Introduction

Rickets is a pediatric bone disease characterized by defective mineralization of the growing skeleton, leading to skeletal deformities, impaired growth, and increased bone fragility. Although it was once thought to be nearly eradicated in developed countries after the discovery of vitamin D in the early 20th century, rickets remains a significant global health problem. It continues to affect millions of children worldwide, particularly in low- and middle-income countries, and is re-emerging in some well-resourced regions due to changing lifestyles, migration patterns, and nutritional habits. The most common cause of rickets is nutritional deficiency, especially inadequate vitamin D intake or insufficient sunlight exposure needed for cutaneous

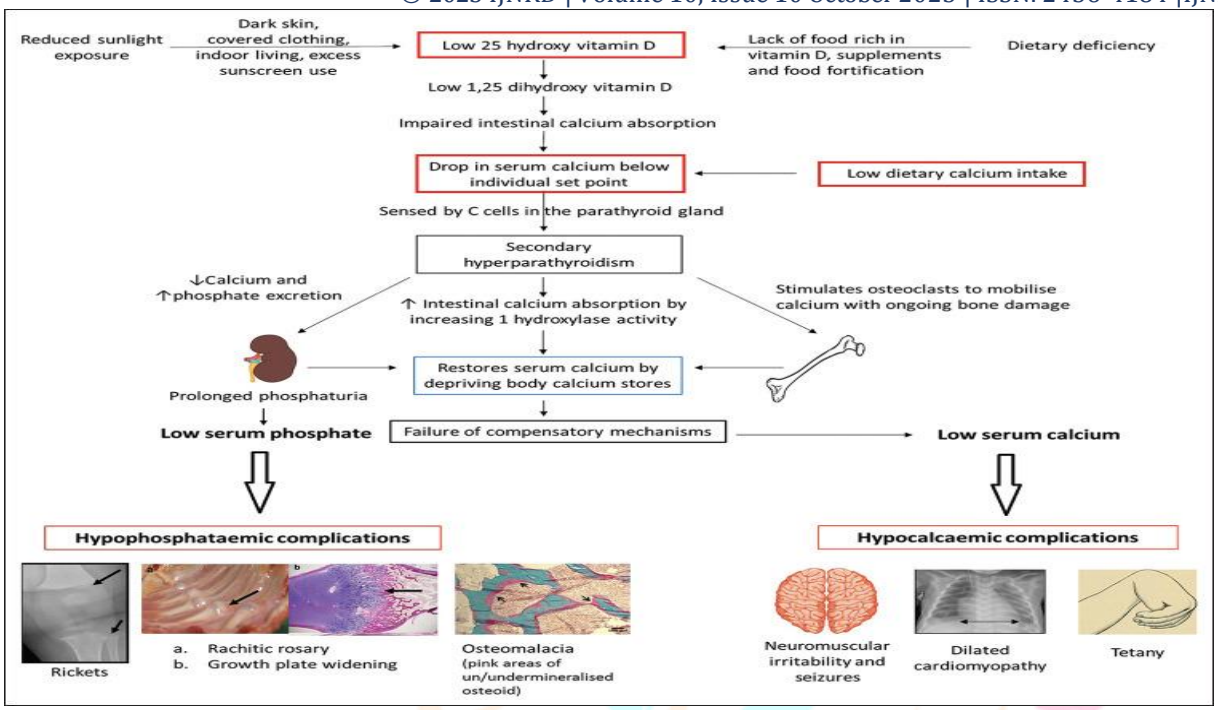
synthesis of the vitamin. Calcium deficiency also plays a major role, especially in parts of Africa and Asia where dairy consumption is low. Beyond nutritional causes, rickets can arise from genetic disorders affecting vitamin D metabolism, receptor function, phosphate regulation, or renal handling of minerals, leading to forms such as vitamin D–dependent rickets and hypophosphatemic rickets. Clinically, rickets manifests with bowing of the legs, delayed growth, widened wrists and ankles, bone pain, muscle weakness, and in severe cases, seizures or respiratory distress due to hypocalcemia. Early recognition is crucial, as untreated rickets can lead to permanent skeletal deformities, stunted growth, and lifelong disability. Diagnosis is based on a combination of history, clinical examination, radiographic findings (such as cupping and fraying of metaphyses), and laboratory investigations including serum calcium, phosphate, alkaline phosphatase, vitamin D metabolites, and parathyroid hormone. Genetic testing may be required in atypical or treatment-resistant cases. The management of rickets depends on its underlying cause. Nutritional rickets is effectively treated with vitamin D supplementation (ergocalciferol or cholecalciferol) and calcium repletion, while heritable forms may require active vitamin D metabolites (calcitriol, alphacalcidiol), phosphate supplementation, or newer targeted therapies such as burosumab for X-linked hypophosphatemic rickets. Prevention is the cornerstone of combating nutritional rickets. Strategies include vitamin D supplementation for infants, pregnant and breastfeeding mothers, food fortification programs, ensuring adequate dietary calcium intake, and promoting safe sun exposure. Global health initiatives emphasize that with coordinated action, nutritional rickets—like iodine deficiency disorders—could be eradicated by 2030.

Types of Rickets:

- Nutritional rickets – vitamin D or calcium deficiency.
- Vitamin D–dependent rickets – defects in activation (Type I) or receptor function (Type II).
- Hypophosphatemic rickets – FGF23-related or renal tubular phosphate loss (includes XLH and hereditary forms).

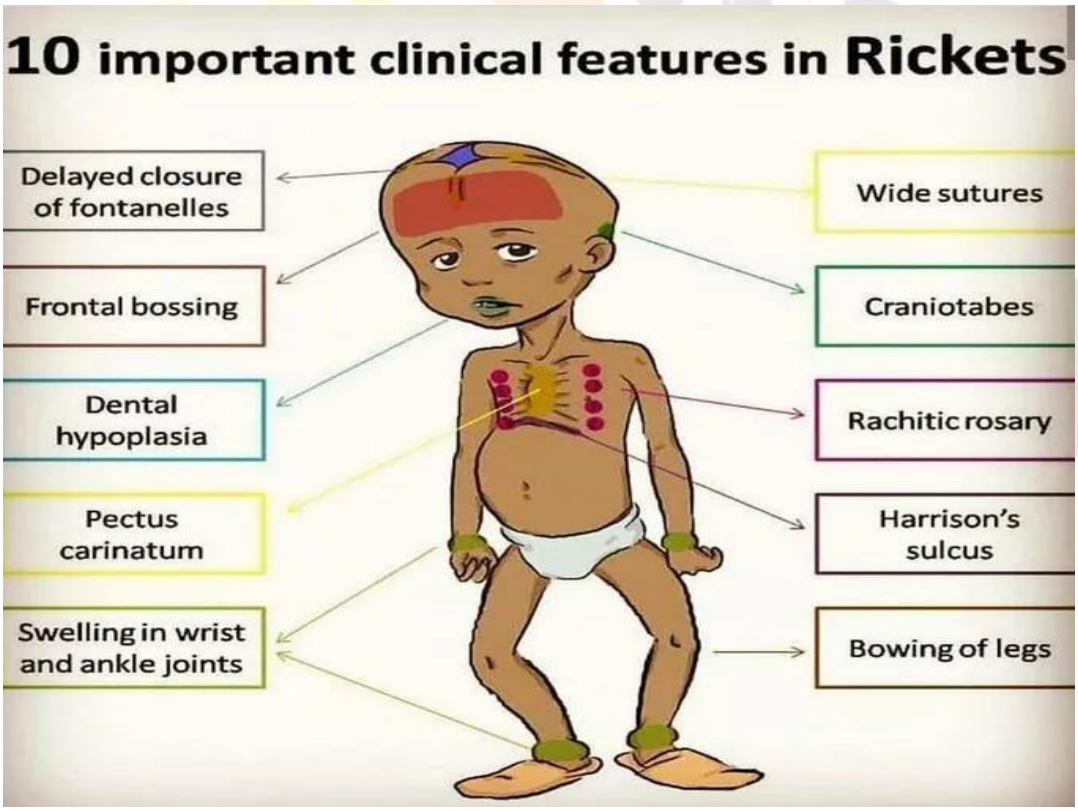
Pathophysiology

Pathophysiology of vitamin D deficiency and dietary calcium deficiency. Both aetiologies lead to calcium deprivation, and secondary hyperparathyroidism ensues in an attempt to optimize serum calcium levels. Prolonged calcium deprivation and phosphate loss ultimately manifest in hypocalcaemic and hypophosphataemic complications seen in nutritional rickets



A Study Of Children Rickets in Kyrgyzstan And India

Rickets, a bone-softening disorder in children primarily due to vitamin D and calcium deficiencies, remains a significant public health concern in both Kyrgyzstan and India. Here's a comparative overview based on recent studies



Rickets in Kyrgyzstan

While specific national data on rickets prevalence in Kyrgyzstan is limited, a 2021 UNICEF-supported national survey assessed the nutrition and micronutrient status among children aged 6 to 59 months and 5–9

years. The study aimed to evaluate deficiencies in nutrients such as iron, vitamin A, and other micronutrients, which are closely linked to bone health and could provide indirect insights into the prevalence of rickets .

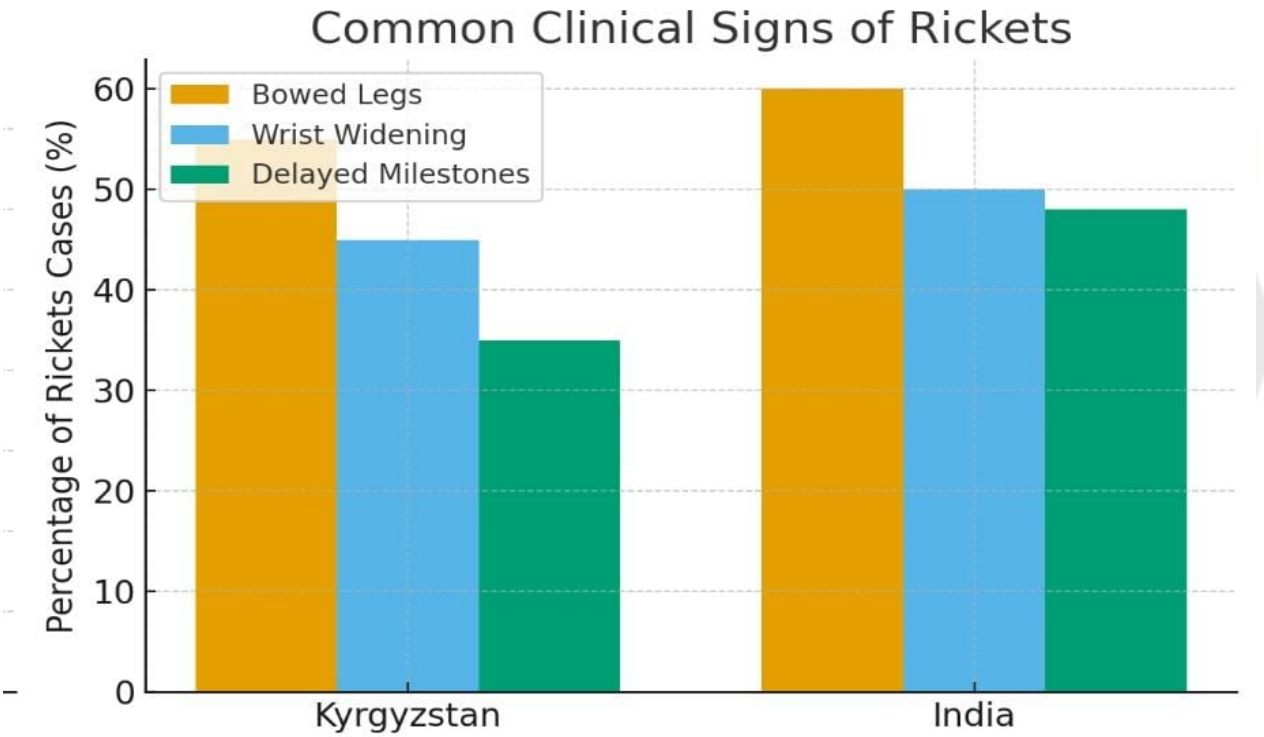
Rickets in India

India exhibits a notable prevalence of rickets, particularly among children aged 0–5 years. A clinical study reported that approximately 46% of children in this age group were affected by rickets. The condition was more prevalent in males (70%) compared to females (30%) .

Comparative Overview

ASPECT	**KYRGYZSTAN**	**INDIA**
Prevalence	Limited national data available	Approximately 46% in children aged 0–5 years
Age Group	Most Affected	Not specified 0–5 years
Gender Distribution	Not specified	Males: 70%, Females: 30%
Contributing Factors	Limited sun exposure, dietary deficiencies	Limited sun exposure, dietary deficiencies, malnutrition

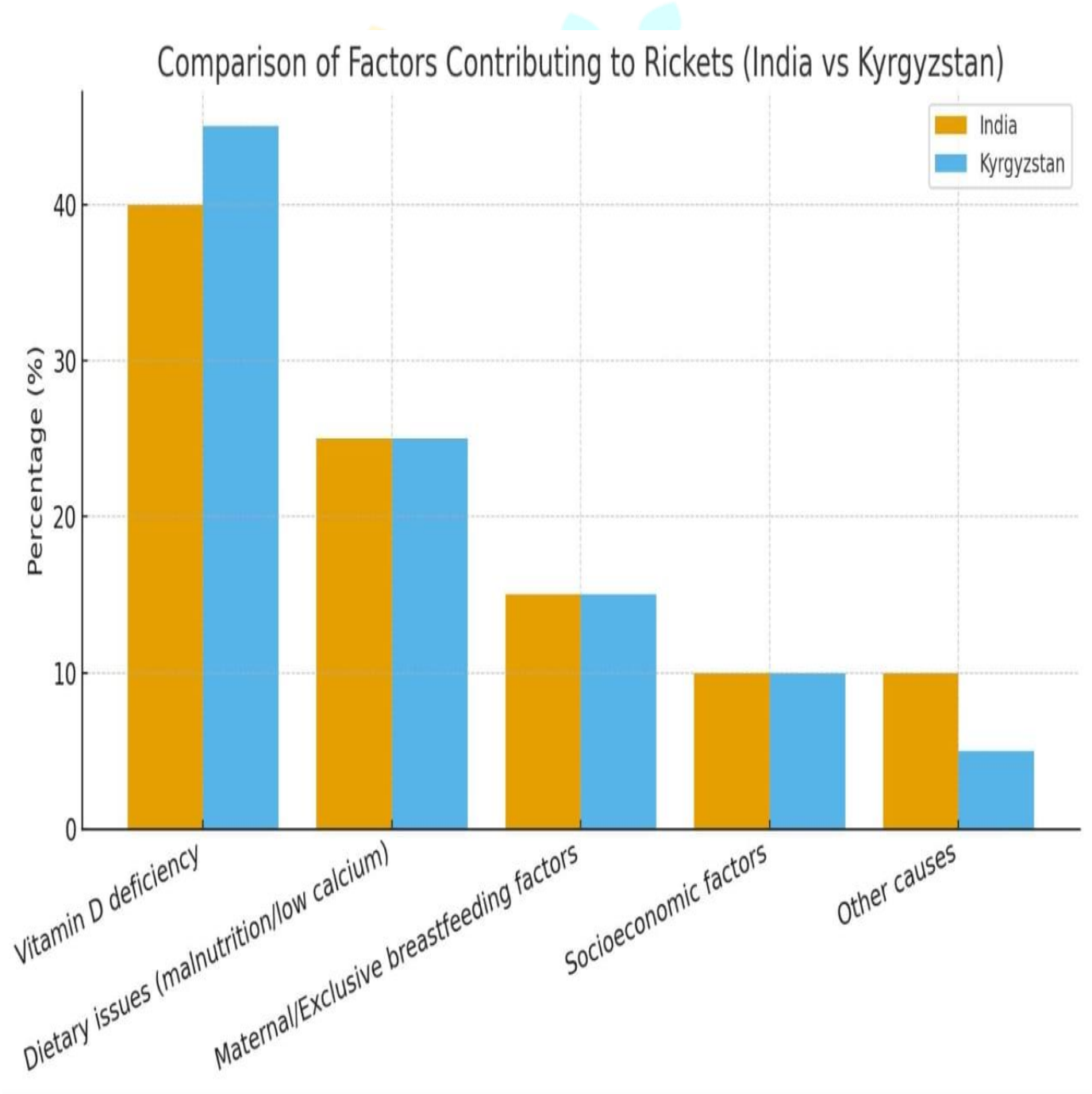
Graphical Representation



This bar chart shows the common clinical signs of rickets in children from Kyrgyzstan and India.

- Bowed legs are the most frequent sign in both countries (≈55% in Kyrgyzstan, 60% in India).
- Wrist widening is also common (≈45% in Kyrgyzstan, 50% in India).
- Delayed milestones are less common in Kyrgyzstan (≈35%) but more frequent in India (≈48%).

FACTORS CONTRIBUTING TO RICKETS IN INDIA AND KYRGYZSTAN

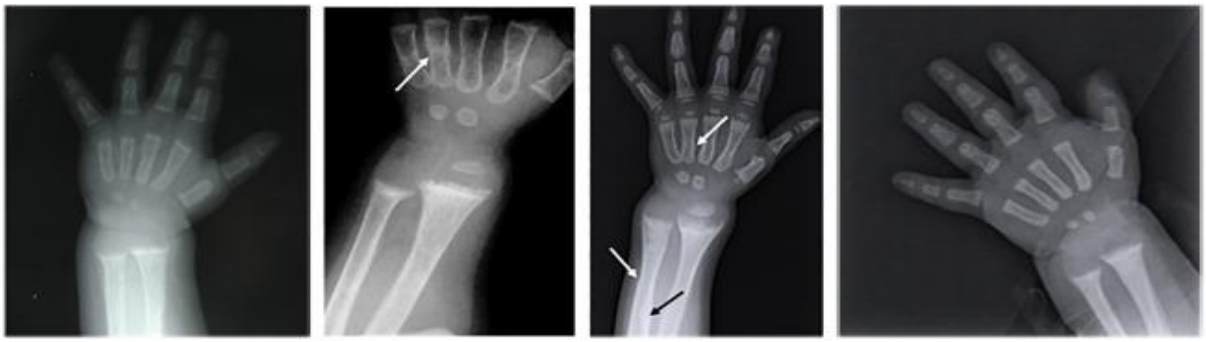


Diagnosis

● Clinical signs → Bowed legs, wrist widening, rachitic rosary, delayed milestones.

● Lab tests → ↓ Calcium/phosphate, ↑ Alkaline phosphatase, ↓ Vitamin D, ↑ PTH.

● X-ray → Cupping, fraying, and widening of metaphyses, osteopenia.



Nutritional vitamin D deficiency rickets Male 3.5 mo

Vitamin D-dependent rickets type 1A Female 16 mo

Vitamin D-dependent rickets type 2A with alopecia Female 2.3 yr

X-linked hypophosphatemic rickets Male 4 mo

Treatment

1. Nutritional Rickets

Vitamin D supplementation: Ergocalciferol (D2) or cholecalciferol (D3) to restore vitamin D levels.

Calcium supplementation: Oral calcium for children with low dietary intake.

Monitor serum calcium, phosphate, alkaline phosphatase, and vitamin D levels.

2. Vitamin D–Dependent Rickets (Genetic)

Type I (VDDR-I): Active vitamin D metabolites (calcitriol or alphacalcidiol).

Type II (VDDR-II): High-dose calcitriol and calcium; more challenging due to receptor resistance.

3. Hypophosphatemic Rickets

Oral phosphate supplementation.

Active vitamin D metabolites to improve bone mineralization.

Targeted therapy: Burosumab for X-linked hypophosphatemia (FGF23-mediated).

4. Supportive Measures

Orthopedic correction for severe deformities after metabolic stabilization.

Adequate nutrition with calcium- and phosphate-rich foods.

Safe sunlight exposure to support vitamin D synthesis.

Conclusion

Rickets is a preventable disease and prevention should start in pregnancy. The simplest measure for prevention is adequate sunlight exposure, however in populations where this is impracticable or implausible vitamin D supplementation should be instituted. There is no global consensus on the amount of vitamin D offered in supplementation. The guidance in the UK from the Department of Health is fragmentary and confusing. Vitamin D 400IU/d is sufficient to maintain vitamin D status in the range where adverse skeletal consequences are very unlikely; suggesting a daily supplement ensures that irrespective of skin colour, latitude, sunlight exposure , pollution, and societal or cultural pressures to cover up, the growing skeleton will get what it needs. It is the view of the authors that supplementation until growth ceases with 10 mcg/day (400 IU/day) in all except those with a known contraindication (e.g. hypercalcaemia, sarcoidosis) should be recommended and that, without such a programme of supplementation and concurrent public health campaign, it is likely the incidence of rickets will continue to rise.

REFERENCE

1. Narasimhan S, Lavik A, Auron M. Rickets. Pediatr Rev. 2025 Sep 1;46(9):494-509. doi: 10.1542/pir.2024-006494. PMID: 40875260.
2. Wharton B, Bishop N. Rickets. Lancet. 2003 Oct 25;362(9393):1389-400. doi: 10.1016/S0140-6736(03)14636-3. PMID: 14585642.
3. Creo AL, Thacher TD, Pettifor JM, Strand MA, Fischer PR. Nutritional rickets around the world: an update. Paediatr Int Child Health. 2017 May;37(2):84-98. doi: 10.1080/20469047.2016.1248170. Epub 2016 Dec 6. PMID: 27922335.
4. Biasucci G, Donini V, Cannalire G. Rickets Types and Treatment with Vitamin D and Analogues. Nutrients. 2024 Jan 31;16(3):416. doi: 10.3390/nu16030416. PMID: 38337700; PMCID: PMC10857029
5. Carpenter TO, Shaw NJ, Portale AA, Ward LM, Abrams SA, Pettifor JM. Rickets. Nat Rev Dis Primers. 2017 Dec 21;3:17101. doi: 10.1038/nrdp.2017.101. PMID: 29265106.
6. Nield LS, Mahajan P, Joshi A, Kamat D. Rickets: not a disease of the past. Am Fam Physician. 2006 Aug 15;74(4):619-26. PMID: 16939184.
7. Takeuchi Y. [Pathophysiology in rickets/osteomalacia]. Clin Calcium. 2007 Oct;17(10):1508-13. Japanese. PMID: 17906401.
8. Michałus I, Rusińska A. Rare, genetically conditioned forms of rickets: Differential diagnosis and advances in diagnostics and treatment. Clin Genet. 2018 Jul;94(1):103-114. doi: 10.1111/cge.13229. Epub 2018 Mar 25. PMID: 29417983.
9. Rennie LM, Beattie TF, Wilkinson AG, Crofton P, Bath LE. Incidental radiological diagnosis of rickets. Emerg Med J. 2005 Aug;22(8):534-7. doi: 10.1136/emj.2003.011668. PMID: 16046749; PMCID: PMC1726861
10. Bouillon R, Antonio L. Nutritional rickets: Historic overview and plan for worldwide eradication. J Steroid Biochem Mol Biol. 2020 Apr;198:105563. doi: 10.1016/j.jsbmb.2019.105563. Epub 2019 Dec 3. PMID: 31809867.