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Rickets:

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June 28, 2026

Introduction:

Hi everyone and welcome back to PedsCases. I am Sarah Clements, a second year medical student at McMaster University. Today we will be discussing Rickets.

This podcast was co-developed with Dr. Ranu Malhi, a general pediatrician at McMaster Children's Hospital.

Why is this topic important?

Rickets poses a significant public health concern during infancy and childhood, requiring early identification and timely intervention to prevent long-term growth, skeletal and systemic complications (1). The prevalence of rickets is higher in low- and middle-income countries (LMIC) in comparison to high-income countries (HIC) due to the introduction of supplemental vitamin D. Despite this availability of supplemental Vitamin D, specific populations in HICs such as Canada are disproportionately impacted by rickets, highlighting the need for focused prevention strategies (2).

Now let's go over the learning objectives. By the end of this podcast you should be able to:

1) Define rickets and explain the roles of vitamin D, calcium and phosphate in bone mineralization.
2) Differentiate the types and common causes of rickets.
3) Identify the risk factors associated with rickets and epidemiological implications in a Canadian context.
4) Recognize common clinical symptoms and diagnostic investigation findings.
5) Develop an approach to the management of rickets.
6) Identify prevention strategies to apply in clinical practice for high-risk populations.

Defining Rickets:
Rickets is a metabolic bone disease caused by a defect in mineralization of the osteoid matrix prior to the closure of the epiphyseal growth plates (3). Vitamin D, calcium and phosphorus are essential factors that have a significant role in the calcification process in growing bones (2). Any disruption to calcium-phosphorus homeostasis or deficiency in the amount of vitamin D, calcium or phosphate can lead to rickets (3). Vitamin D is synthesized in the body when the skin is exposed to ultraviolet B radiation from the sun or through the dietary intake of fatty fish, egg yolk, and fortified foods (2). After Vitamin D is synthesized, it is activated in a two-step process. Firstly, vitamin D is metabolized in the liver to its major circulating form calcidiol (25-hydroxyvitamin D3) and converted to its hormonally active form calcitriol in the kidneys (1,25-dihydroxycholecalciferol) (4). The major regulators of the enzyme renal 1-hydroxylase that produces calcitriol include parathyroid hormone, FGF-23, calcium, and phosphate (4).

Types and Etiology of Rickets:
The causes of rickets can be grossly classified into calcipenic or phosphopenic rickets and further subdivided by acquired or hereditary etiologies (2). Calcipenic rickets is a deficiency in calcium that can be caused by nutritional deficiencies, malabsorption syndromes, secondary acquired disorders or a category of hereditary disorders that cause vitamin D-dependent rickets (VDDR) (2). Phosphopenic rickets is a deficiency in phosphate levels which is caused by vitamin D resistant disorders, specifically FGF23-mediated and non-FGF23-mediated etiologies (2).

Let's break down each of the major etiologies starting with calcipenic rickets. Firstly, nutritional rickets is the most common cause of calcipenic rickets globally and is characterized by a deficiency in vitamin D leading to low calcium levels (2). Inadequate levels of vitamin D in infants and children are often associated with maternal vitamin D deficiency, exclusive breastfeeding without supplementation, or inadequate dietary intake of vitamin D (5). Low vitamin D levels contribute to decreased intestinal absorption of calcium and a compensatory increase in parathyroid hormone (PTH) (3). PTH promotes the internalization of sodium-dependent phosphate co-transporters in the renal tubules leading to renal phosphate loss with progression of the disease course (2). Without sufficient calcium or phosphate, bone mineralization is impaired. In addition to dietary deficiencies, individuals who live at high latitudes have less exposure to UVB radiation from the sun and are at a higher risk of vitamin D deficiency (6). Secondly, malabsorption syndromes such as celiac disease or cystic fibrosis can contribute to poor calcium absorption despite an adequate diet (2). Thirdly, secondary acquired disorders such as kidney or liver disease could disrupt pathways leading to the activation of vitamin D (2). Lastly, hereditary vitamin D-dependent rickets (VDDR) impairs the synthesis of calcitriol or the response to calcitriol at the level of the vitamin D receptor (2). Type I VDDR reflects a mutation in the enzyme that converts vitamin D to its active form and type II VDDR is a mutation in the gene that codes for the vitamin D receptor (3).

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Now for phosphopenic rickets- vitamin D resistance, also known as familial hypophosphatemia, is the main cause. The most common form is X-linked dominant hypophosphatemia caused by a mutation in the PHEX gene that leads to increased levels of FGF23 (3). FGF-23 decreases phosphate absorption in the kidneys leading to phosphate wasting in addition to suppressing renal enzyme activity that converts vitamin D to its active form, calcitriol (3). The other two forms of familial hypophosphatemia include autosomal dominant hypophosphatemia which is a mutation in the FGF-23 gene and autosomal recessive hypophosphatemia with various gene mutations (3).

Clinical Case:
Before discussing rickets further, let's explore a clinical case. You are a fourth-year medical student on a rural pediatric elective in Inuvialuit working at a community health centre. Nanuq, a 20-month-old male Inuit boy was brought to the health centre for concerns with delayed motor milestones. He is currently not walking independently but is meeting his other developmental milestones appropriately. You learn on history that he was exclusively breastfed for the first 12 months with inconsistent vitamin D supplementation. His parents' express concerns with food insecurity and limited consumption of vitamin D rich foods. During the long winter months, Nanuq spends minimal time outside.

At the visit, Nanuq plots on the 12th percentile for length and 16th percentile for weight. On physical exam, he has mild hypotonia and visible widening of his wrists bilaterally. He appeared hesitant to weight bear on his legs and would only stand briefly with support.

Laboratory investigations indicate low calcidiol levels with mildly decreased calcium and phosphate levels. His alkaline phosphatase and parathyroid hormone levels were markedly elevated. X rays of his wrists showed early metaphyseal changes, including widening of his growth plates and mild cupping.

Keep this case in mind as we go through this podcast episode.

Epidemiology and Risk Factors:
The prevalence of rickets is higher in LMICs, often attributed to malnutrition associated with poor socioeconomic conditions (7). In HICs, there is a lower prevalence of rickets given the introduction of vitamin D supplementation and education on the importance of vitamin D for bone health (2). However, rickets is persistent in HICs despite the introduction of prevention guidelines particularly among specific populations (8).

In Canada, Indigenous infants and children from northern communities and immigrant children are disproportionately impacted by rickets (9,1). Contributory risk factors among Indigenous communities as highlighted by the Canadian Pediatric Society, include lower socioeconomic status, reduced sun exposure during the winter season, being at a

northern high latitude, and food insecurity (9). In addition, maternal vitamin D deficiency was a risk factor identified among Indigenous women, associated with dietary intake (9). As a result of colonialism, which led to residential schools and forced displacement, the increased cost and decreased access and availability of healthy and fortified foods have contributed to food insecurity among Indigenous communities (9). In addition, there has been decreased harvesting and consumption of country foods, which include game meat, birds, and fish (9). Among immigrant and refugee populations, restricted access to nutrient-rich foods high in vitamin D and limited healthcare resources contributes to nutritional vitamin D deficiency and higher risks of rickets (7). Additionally, in comparison to many immigrants' countries of origin, Canada is at a higher latitude and has a colder climate. This change in latitude and sun exposure exacerbates the risk of rickets among this population given insufficient production of vitamin D with less ultraviolet light throughout the year (6).

Generally, risk factors to developing rickets include babies that are exclusively breastfed or have a milk intolerance and do not take additional vitamin D supplementation, darker skin pigmentation, low birth weight, low socioeconomic status, lack of sun exposure, and poor nutrition (7).

Clinical Presentation:
Rickets most commonly presents with abnormalities in the bones of the skull, chest, weight-bearing limbs and spine given poor bone mineralization (2). Findings for the skull include softening of the skull bone, frontal bossing and widened fontanelles (2). On the chest, widening of the costochondral junction known as rachitic rosary, pigeon chest, and Harrison's groove may be observed (2). Given the difference in weightbearing with age, infants tend to have lower limb deformities and children have upper limb deformities (2). Findings include bowed femurs or tibias, knock knees, and widening of growth plates at the wrist and ankles (9).

With respect to other physical exam findings, there may be evidence of faltering of growth, delayed motor milestones, gait changes, failure to thrive, muscle hypotonia, bone pain, limb fractures, and dental abnormalities such as enamel hypoplasia and caries (9). Signs of significant hypocalcemia include paresthesia, tetany, myopathy, and seizures (2).

Diagnostic Investigations:
Beyond the physical exam, the next step would be diagnostic investigations. Let's discuss a diagnostic approach to suspected rickets. First, look for elevated alkaline phosphatase activity and clinical or radiographic findings of rickets (10). Given abnormal bone mineralization with rickets and increased osteoblastic activity, ALP would be markedly elevated (10).

It is important to measure serum calcium, inorganic phosphorus, and PTH levels (10). For calcipenic rickets, PTH would be high, inorganic phosphate normal or low and calcium normal or low depending on progression of disease and compensatory PTH response

(10). The clinical course of vitamin D deficient rickets has progressive stages of worsening severity (11). Early in the clinical course, there is impaired intestinal absorption of calcium resulting in hypocalcemia but levels of inorganic phosphorus are normal (11). In response to low calcium levels, there is a compensatory increase in PTH secretion, which stimulates calcium release from the bone matrix, normalizing levels of calcium. This release of calcium from bone resorption is associated with increased levels of ALP. (11). Hypophosphatemia becomes evident with disease progression as PTH inhibits phosphate reabsorption in the kidneys, leading to phosphate renal wasting. This is when rickets tends to be clinically identified (11). In later stages of rickets, calcium levels decrease again when hyperparathyroidism is not sufficient for compensation (11). To differentiate rickets caused by a vitamin D deficiency, serum 25-hydroxyvitamin D level, also known as calcidiol and a major circulatory form of vitamin D, should be measured and would likely be low (2). For genetic vitamin D dependent rickets (VDDR), Type I VDDR would have low levels and Type II VDDR have high levels of calcitriol, the biologically active hormonal form of vitamin D produced by the kidneys (2).

For phosphopenic rickets, PTH is normal or mildly elevated, inorganic phosphate low, and calcium normal (10). An assessment of renal excretion of phosphate should be investigated, specifically by calculating the tubular reabsorption of phosphorus (TRP) (10). This is a calculation based on the measurement of serum and urine phosphorous and creatinine. If the TRP is low, this would suggest phosphate wasting (10). High levels of TRP indicate conservation of phosphate and would be consistent with nutritional phosphate deprivation (10).

With regards to imaging, X rays would be first-line to identify bone abnormalities. Common findings include widening of the epiphyseal plate due to the accumulation of non-mineralized osteoid, angular deformities, decreased bone density, rachitic rosary, bowing of the lower extremities, and pathological fractures in advanced stages (2, 3). Specifically at the growth plate, there is a loss of definition of the zone of provisional calcification at the epiphyseal/metaphyseal interface which progresses to disorganization of the growth plate (10). Signs of this disorganization include cupping, splaying, and the formation of cortical spurs (10).

Treatment:

Let's now discuss treatment for rickets. For nutritionally deficient vitamin D rickets, vitamin D supplementation is the first line treatment either with multiple daily doses or a single-dose therapy (2). Daily therapy is the most common treatment and uses vitamin D2 known as ergocalciferol or vitamin D3 known as cholecalciferol (11). For dosing, infants less than 1 month receive 1000 international units (IU) daily and infants 1 to 12 months receive 1000-2000 IU daily for three months followed by 400 IU daily for maintenance (11). Children aged 1 to 12 years old receive 2000-6000 IU daily and children over 12 years old 6000 IU daily for three months followed by maintenance dosing of 600 IU daily (11).

Alternatively, to daily therapy, stoss therapy involves a single high dose of vitamin D, which may be more beneficial for patients with poor medication compliance (11). However, stoss therapy is rarely used and with caution with the increased risk of hypercalcemia (12).

After treatment is initiated, biochemical and radiologic abnormalities should be resolved within three months (11). During treatment, it is important to monitor growth, dental changes and recheck biochemical markers including serum ALP, calcium, phosphorus, PTH and 25-OH Vitamin D levels.

Unlike vitamin D, calcium is readily available in many different food sources, and it is important that children receive enough daily calcium. For children aged 1-3 years, 700mg of daily calcium is needed, 1000mg for 4-8 years old, and 1300mg for 9-18 years old (13). Calcium supplementation may be required with expert input. For patients with symptomatic, severe hypocalcemia such as seizures, cardiomyopathy or tetany, initial management is intravenous calcium gluconate until serum calcium levels normalize, after which oral calcium supplementation can be given (3).

Orthopedic intervention is often not indicated for rickets given skeletal deformities can resolve completely with medical management alone, but correction of deformities with surgery may be necessary if there is no observable improvement (11).

For genetic causes of rickets, treatment is not curative but rather targets improvement in quality of life (2). Vitamin D dependent rickets is treated with calcitriol and familial hypophosphatemic rickets is treated with oral phosphate and calcitriol (3).

Let's return to Nanuq, for management he was started on 3000 IU vitamin D supplementation daily for three months followed by a maintenance daily dose of 600 IU. His parents were counselled on dietary changes to increase calcium and vitamin D intake through fortified foods. Educational counselling for future pregnancies on risk factors for rickets including living at higher latitudes, limited sun exposure, exclusive breastfeeding, and low vitamin D diet was discussed.

Strategies for Prevention:
When considering Nanuq's clinical case and his multiple risk factors for vitamin D deficiency rickets, there are multiple strategies healthcare providers can apply to help prevent nutritional rickets among infants and children.

During prenatal and well child visits, it is important to gather an understanding of the maternal, infant or child's dietary intake of vitamin D, supplementation of vitamin D, sunlight exposure, and developmental history. To determine the risk of genetic causes of rickets, obtain a family history of skeletal abnormalities, stunted growth, alopecia, dental concerns, and parental consanguinity (2).

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June 28, 2026

Prevention suggestions for prenatal and lactating women include increasing dietary vitamin D and adding a daily prenatal vitamin containing 400 IU vitamin D, folic acid, and iron (9). For women at a higher risk of vitamin D deficiency, recommendations include taking an additional 1000 IU per day of vitamin D (9). For infants less than 12 months of age, low risk breastfed infants should receive 400 IU per day in the first year of life and high-risk infants 800 IU per day (9). Formula fed infants don't require any supplementation if they are drinking more than 800-1000 mL per day of formula (9). High risk formula fed infants require 400 IU per day in the first year of life and if they remain at high risk after one year, should continue supplementation into childhood (9).

When counselling on vitamin D supplementation, take into consideration the socioeconomic barriers that may limit a patient's access to vitamin D rich foods or their ability to afford supplements (9). In addition, when starting supplementation with vitamin D for infants, it would be helpful to show parents and caregivers how to administer supplements to ensure accurate dosing (9). For example, adding vitamin D to a baby's bottle may not be effective if the infant does not consume the whole volume. When feasible and if appropriate for high-risk individuals, consider measuring levels of calcidiol (9).

Lastly, as health care providers, advocacy is essential. Nutritional rickets is an entirely preventable disease (6) For providers working with high-risk populations, support and advocate for initiatives that improve food insecurity. This could include collaborating with local Indigenous communities to strengthen programs that encourage the harvesting and consumption of country foods and/or advocating for Vitamin D supplements to be provided free of cost (9).

You see Nanuq at a follow up appointment 3 months later. He has demonstrated improvement in his muscle tone and is independently walking. His laboratory levels have normalized, and repeat X ray imaging of his wrists show resolution of his previous metaphyseal changes.

Before we end the podcast, let's finish with some key takeaways.

1) Rickets is a metabolic bone disease caused by a defect in mineralization of the osteoid prior to the closure of the epiphyseal growth plates.
2) Nutritional vitamin D deficiency is the most common cause of rickets and largely preventable.
3) Rickets is most prevalent in LMICs, but disproportionately impacts Indigenous and immigrant populations in HICs.
4) Clinical signs of rickets most commonly include skeletal deformities and growth delays that may be reversible with early identification and treatment.
5) Management and prevention of rickets involves vitamin D supplementation, nutritional support, and routine preventative supplementation among high-risk populations.

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That brings us to the end of today's podcast on rickets. I am Sarah Clements and this is PedsCases. Thanks for listening.

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