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Rickets on the Rise: A Case Series Highlighting the Importance of Growth Monitoring and Nutritional Assessment in Preventing Rickets

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ABSTRACT: Nutritional rickets, though a preventable condition, continues to show an increased incidence in clinical practice. It is crucial to recognize these cases during early stages in order to prevent the progression from vitamin D deficiency to clinical rickets, with the potential for irreversible complications. This case series describes five pediatric patients in which vitamin D deficiency and rickets was identified by close observation through growth monitoring and nutritional assessment. These workups were conducted due to the initial presentation of failure to thrive in each of these cases, prompting further evaluation to identify the underlying causes of growth faltering. A common theme throughout was normocalcemia, however, it is important to understand that this finding does not rule out the potential for rickets, as both subclinical rickets and stage two compensatory rickets can lead to normal laboratory findings. Therefore, growth faltering should always prompt nutritional assessment because early recognition, growth monitoring, a well-balanced diet with calcium-rich foods, compliance with vitamin D supplementation, and consistent follow-ups are essential to prevent the risk of developing clinical rickets.

KEYWORDS: diagnostic vigilance, rickets, nutritional deficiencies, rural healthcare, vitamin D deficiency.

1. INTRODUCTION

What was once considered to be a historical disease, rickets has re-emerged throughout the past three decades in many high-income countries. Recent surveys have shown an increase in incidence of up to 24 per 100,000 patient-years throughout North America, Australia, and Europe.[1]

Rickets is a childhood bone disorder characterized by defective mineralization and disorganization of the epiphyseal growth plate. Nutritional rickets, due to vitamin D and/or calcium deficiency, remains the most common form worldwide. Diagnosis is based on a triad of clinical, radiographic, and biochemical markers. Clinically, the patient may present with bowing of the legs, poor posture, rachitic rosary, widening of joints and/or craniotabes.[2] Radioimaging classically reveals metaphyseal fraying, cupping, and widening of the growth plate. Elevated alkaline phosphatase (ALP) is one of the key biochemical markers. Additional abnormalities may include elevated parathyroid hormone (PTH), low or normal calcium, low or normal phosphate, and low vitamin D. Most guidelines define vitamin D insufficiency as a serum 25-hydroxyvitamin D level between 20-30 ng/mL, deficiency as less than 20 ng/mL, and severe deficiency as less than 12 ng/mL. Increased ALP levels are typically cited as greater than 461 U/L, however, this can vary depending on age.[3]

In patients with vitamin D deficiency, there is a staged progression to the development of rickets. Early disease, or stage one, may present with silent or symptomatic hypocalcemia, irritability, lethargy, growth failure, or recurrent respiratory infections. As deficiency persists to stage two, secondary hyperparathyroidism results in order to normalize serum calcium, resulting in increased bone turnover, elevated ALP, PTH, and hypophosphatemia. Finally, during stage three, the compensation becomes insufficient and calcium levels are unable to be maintained. At this point, radiographic changes will be able to be seen.[4] In early stages, patients may have subclinical levels in which there may be no discernible clinical changes or radiographic findings, however low vitamin D and raised ALP may be observed.

While many children with low vitamin D levels never progress to rickets, if there is delayed recognition or treatment, permanent skeletal deformities and impaired growth may occur. Vitamin D plays a critical role in growth, bone health, immune modulation, as well as lowers the risk of respiratory tract infections, as such, it is recommended that children and adolescents (1-18 years) be empirically supplemented with vitamin D to prevent the avoidable and negative consequences of deficiency.[1]

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Several of the patients presented throughout our case series were identified during close observation for faltering weight rather than skeletal abnormalities. According to the American Academy of Pediatric guidelines, the criteria for faltering weight include; a weight-for-length or BMI-for-age less than the fifth percentile, a z score for age less than -2 for weight gain velocity in children aged less than 2, or a decline in weight, weight-for-length, or BMI greater than or equal to one z score.[5] Evaluation of these patients should always include careful assessment of growth charts, dietary intake, feeding practices, and socioeconomic factors, including food insecurity, that may contribute to poor nutritional status.

This case series highlights the importance of routine growth surveillance and appropriate nutritional assessment in children with faltering weight. We emphasize how untreated vitamin D deficiency, even in the absence of overt clinical or radiographic rickets, may be accompanied by biochemical evidence of increased bone turnover. Early recognition, close laboratory monitoring, and timely intervention can prevent progression to nutritional rickets and its potential for irreversible growth and skeletal complications.

2. CLINICAL PRESENTATION

Case #1:

Our first, and most severe case, presented with failure to thrive early on. The patient was born vaginally, with no complications, but was premature at 36 weeks. She was born at 4.15 lbs. The infant was breast fed for the first month of life, until she was switched to Similac Advance, but never received any vitamin D supplementation. By her two and a half month check-up, her height and weight were below the fifth percentile. This patient was closely monitored to observe her weight and height to ensure adequate growth. By one year, she was only at the 10th percentile for weight, and further evaluation was carried out to determine an underlying cause for failure to thrive.

Biochemical panels were ordered to assess for metabolic abnormalities, as well as to assess for anemia. Her initial labs revealed an alkaline phosphatase of 1609 U/L, with normal serum calcium at 8.9 mg/dL. Given this significant elevation in ALP, further tests were ordered to assess vitamin D status. Her vitamin D level came back at 7.4 ng/mL (Figure 1).

Initially the patient presented with a normal PTH value, however, as previously noted, during early stages of rickets, patients may have subclinical levels, with elevated ALP or decreased vitamin D being the only significant biochemical values. Further evaluation of this patient with x-ray of the right knee revealed transverse sclerotic metaphyseal bands of the distal right femur and proximal tibia, consistent with clinical rickets (Figure 2).

Case #2:

Our next patient is a 5-year-old girl who initially presented at two years old with failure to thrive. The pediatrician recommended regular follow-ups for weight and diet check, however, there was a significant issue with noncompliance and missed appointments. Due to continued inadequate growth, serial labs were ordered and the patient had continued vitamin D insufficiency & deficiency. ALP was persistently elevated between 244-282 U/L. Calcium in this patient remained normal. This presentation is consistent with an ongoing increase in bone turnover secondary to chronic vitamin D deficiency, potentially leading to subclinical rickets. The patient's guardian provided a two-day diet list (Figure 3), to which the physician encouraged a well-balanced diet with calcium-rich foods, as well as cholecalciferol supplementation; non-compliance with this plan resulted in continued deficiency. Follow-up x-ray did not reveal any acute or concerning osseous abnormalities or radiographic evidence of rickets at this time.

Case #3:

The third patient in our series had mild vitamin D insufficiency (26.7 ng/mL), with normal calcium and a normal ALP. This represents a scenario in which an early insufficiency can be identified and treated before worsening deficiency or metabolic bone disease can progress. On initial evaluation, x-ray currently showed no radiographic evidence of rickets.

Case #4:

This patient also had mild vitamin D insufficiency (26.7 ng/mL), but with persistently elevated ALP ranging between 235-494 U/L, but remained eucalcemic. The rising ALP alongside vitamin D insufficiency raises concern for worsening bone turnover. The ALP continued to trend upwards, indicating the need for continued monitoring and evaluation for progression to or evidence of subclinical rickets. An order was written for this patient to undergo an x-ray of the right knee, however, due to noncompliance, the x-ray was never completed and further follow-up appointments were not attended.

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Case #5:
Our fifth case is representative of a situation in which compliance and follow-up, despite physician persistence, was unable to be achieved. The patient was evaluated for vitamin D deficiency at 10 years old and further followed by pediatric endocrinology. Vitamin D level came back severely deficient at 8.2. Subsequent testing was requested to assess ALP, PTH and calcium levels, however, due to non-compliance, this assessment was not done. The patient was prescribed cholecalciferol 50,000 IU weekly with repeat labs recommended in 1 month. A diet rich in calcium was recommended. The patient never returned to the clinic for continued evaluation.

3. MANAGEMENT AND TREATMENT

For each of these patients, supplementation for the vitamin D deficiency was recommended, with a repletion phase of 50,000 IU/week for six weeks, followed by a daily maintenance dose. Dietary recommendations included ensuring a well-balanced diet, with emphasis on calcium-rich foods. Each family was counselled on the importance of ensuring adequate nutrition, as well as the need for routine follow-ups for weight and diet checks, as well as serial labs to ensure that levels stabilize to normal values.

4. OUTCOME AND PROGNOSIS

Rickets induced by vitamin D deficiency is a largely preventable and treatable condition, and is often completely curable with early intervention and adherence with vitamin D supplementation. For our patients, if compliance can be achieved, follow-ups should reveal normalization of biochemical parameters, as well as no increase in the risk of skeletal abnormalities. If the vitamin D deficiencies become chronic, these patients are at risk for progression to clinical rickets with the potential for irreversible damage.

5. DISCUSSION

Each patient in our case series presented with vitamin D levels below the acceptable threshold of 30 ng/mL. Two of our patients remained severely deficient below 12 ng/mL (case one and five), one was moderately deficient between 10-20 ng/mL (case 2), and two were insufficient between 20-30 ng/mL (cases three and four). Many of these patients on follow-up evaluation still remained deficient months later, suggesting inadequate monitoring, incomplete treatment, poor adherence, or insufficient supplementation.

Four of five patients on evaluation had elevated ALP, a crucial marker of osteoblastic activity, and increases substantially when poorly mineralized bone is attempting to mineralize. In cases of nutritional rickets specifically, ALP is often one of the earliest and most sensitive indicators. Due to the elevated presence in many of our cases, follow-up evaluation becomes essential.

Interestingly, the calcium values remained normal for most of our patients. It is important to note that despite this normocalcemic finding, children can have significant vitamin D deficiency, and even early stages of rickets without hypocalcemia due to homeostatic mechanisms such as increased intestinal absorption, increased bone resorption, and secondary hyperparathyroidism. As such, a normal serum calcium finding should not indicate clinically insignificant vitamin D deficiency, and should continue to be monitored.

Another key finding throughout the care of these patients that raises concern is compliance. On multiple instances, although appropriate dosing was prescribed, follow-ups continued to reveal vitamin D deficiency, due to familial noncompliance in efficiently providing the patient with supplementation. Given that for most of these patients, their deficient vitamin D levels had not yet progressed to the level of causing skeletal abnormalities, rickets can effectively be prevented simply by ensuring adequate nutrition and adherence to treatment. As such, it is crucial to ensure that the parents/guardians of these children understand the importance of follow-up and monitoring of the vitamin D, calcium, and ALP levels, and ensuring that levels remain in the normal range. It is of the utmost importance that providers make these families aware of the severe and permanent complications that can arise once rickets progresses to the later stages, at which point the damage may be irreversible.

6. CONCLUSION

Given the rise in malnutrition-induced rickets, especially within developed countries, it is more significant than ever that awareness is brought to the importance of a well-balanced, calcium-rich diet. Rickets caused by vitamin D deficiency is preventable, and once identified, is easily curable with compliance to treatment. It is crucial to ensure that all patients' growth and development is routinely monitored, and adequate follow-up and assessment is carried out when failure to thrive is identified. We can see increased prevalence

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in western society due to low economic status, low educational attainment, and a high percentage of individuals with substance use disorder, which can lead to child neglect and feeding issues. It is important to always keep in mind that when completing a workup for failure to thrive, check vitamin D levels. If there is a high level of suspicion, order an x-ray to confirm severity and diagnosis. Through this analysis and the methods provided, it is clear that early detection, treatment, and monitoring of vitamin D deficiency is important and we always want to avoid the progression to Rickets.

7. ACKNOWLEDGMENTS

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8. DATA AVAILABILITY

This article is freely available and may be used by all members of the scientific and medical community.

Consent to participate

The patients in this case series are under the age of 18, therefore, verbal informed consent was obtained by the patient's parents/guardians to write and publish this case series. The informed consent was obtained by Kid Care, Department of Pediatrics.

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	25-OH Vitamin D (ng/mL)	Calcium (mg/dL)	ALP
Case 1: 12/04/23	N/A	8.9	1609
Case 1: 01/03/24	7.4	9.7	262
Case 1: 05/07/24	26	10.8	229
Case 1: 01/02/25	5.1	10.1	248
Case 2: 04/14/25	18.5	9.7	244
Case 2: 12/18/25	21.0	9.5	282
Case 3: 08/05/25	26.7	9.3	138
Case 4: 09/06/24	N/A	10.2	235
Case 4: 08/13/25	26.7	9.4	494
Case 5: 12/23/21	8.2	N/A	N/A

Figure 1: 25-hydroxyvitamin D (25-OH Vitamin D), calcium, and alkaline phosphatase (ALP) levels from the CMP/BMP for patients one through five.

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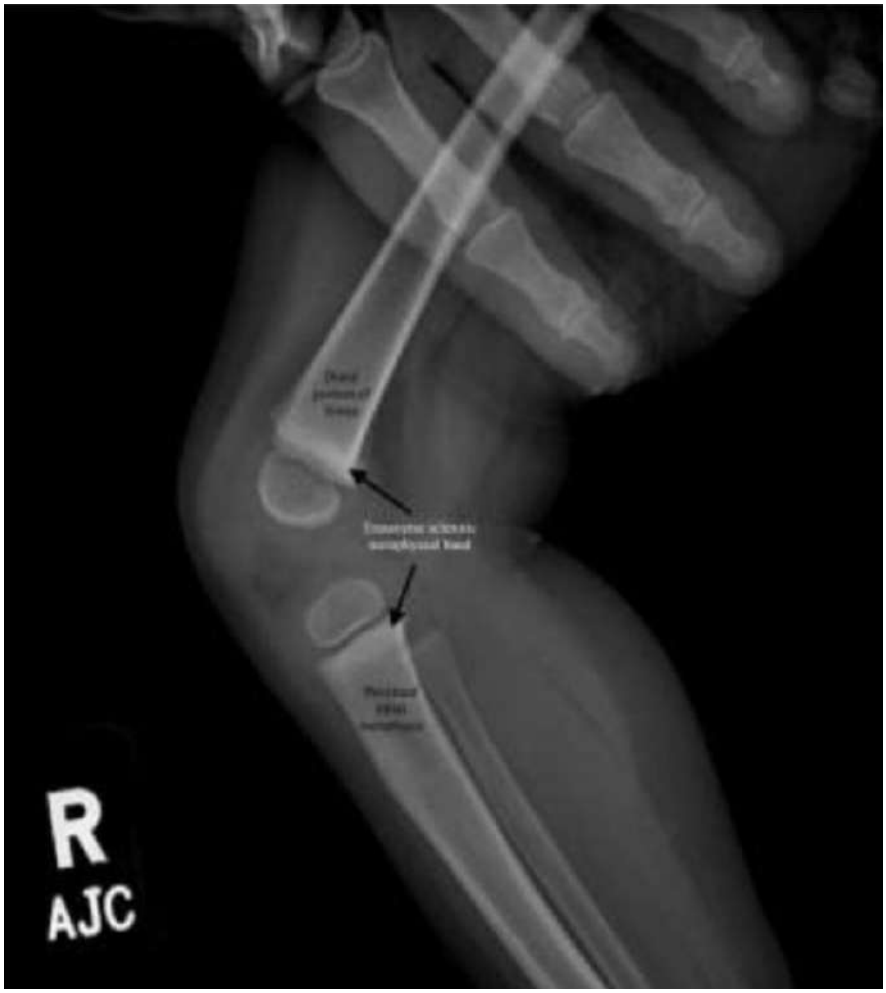

Figure 2: Radiographic imaging of the right knee revealing transverse sclerotic metaphyseal bands of the distal right femur and proximal tibia.

Monday	Tuesday
Mac & Cheese Ramen Noodles Scrambled Eggs Jello Cup Potato Chips	Eggs Strawberry Ice Cream Chicken Nuggets & Fries Sliced Apples BBQ Chicken

Figure 3: Two-day diet list for case #2.

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