

Failure to Thrive in an 18-Month-Old Female Infant with Rickets: A Case Report

Nadeen Aladham^{1,2}, Ahmad Khiami, MD¹, Nitasha Abbas^{1,3}, Jay Prashar⁴, Justine Almeida⁴

¹Department of Pediatrics, Kid Care

²Ross University School of Medicine

³Lincoln Memorial University Debusk College of Osteopathic Medicine

⁴Avalon University School of Medicine

ABSTRACT: Undetected vitamin D deficiency can lead to severe complications such as Rickets in infants. This case report describes an unusual case of an 18-month-old female infant who presented with failure to thrive early in infancy and was later diagnosed with Rickets. The infant initially presented with failure to thrive, labs were ordered, and findings demonstrated low 25-hydroxyvitamin D and elevated alkaline phosphatase. Radiographic imaging of the knee was ordered and showed transverse sclerotic metaphyseal bands involving the distal femoral metaphysis and portion of the proximal tibial metaphysis, findings consistent with untreated rickets. A thorough investigation was conducted to determine the primary cause of the vitamin D deficiency, identifying the primary cause as nutritional deficiency. Per endocrinology, the patient was started on cholecalciferol 800 IU daily with dietary changes. This case emphasizes the importance of checking vitamin D levels in all pediatric patients who present with failure to thrive.

KEY WORDS: Failure to thrive, Nutrition, Rickets, Rural healthcare, Vitamin D deficiency.

1. INTRODUCTION

Vitamin D is a fat-soluble vitamin obtained through diet or dermal synthesis.¹ Synthesis begins when ultraviolet light hits the skin converting 7-dehydrocholesterol to 25-hydroxyvitamin D in the liver.¹ Lastly, 25-hydroxyvitamin is converted to 1,25-dihydroxyvitamin by the enzyme 1-alpha-hydroxylase in the kidney.¹ A disturbance in vitamin D synthesis can alter calcium homeostasis thus, affecting bone development in patients. Monitoring vitamin D levels in infants with failure to thrive (FTT) is essential to their ability to adequately grow.

Rickets is a known bone condition caused by vitamin D deficiency in infants and children.² Classification can be based on calcium or phosphate levels: classifying it as calcipenic associated with insufficient intake of vitamin D or phosphopenic rickets associated with renal phosphate wasting.^{3,4} It is also linked to maternal vitamin D deficiency, prematurity, infants with dark skin pigmentation and those who are exclusively breastfed without addition of any supplement.² In the United States, ~15% of vitamin D deficiencies or insufficiencies occur in the pediatric population, with only ~1-2% of these infants presenting with a 25-hydroxyvitamin D concentration <10 ng/mL.² Mineralization deficiency may occur months before rickets is obvious on physical, laboratory, or radiologic findings and, early signs may include failure to thrive, lethargy, and recurrent respiratory infections.⁴

The following case describes an 18-month-old female infant who presented with failure to thrive at infancy and later diagnosed with Rickets.

2. CASE PRESENTATION

An 18-month-old female infant with a history of neonatal jaundice presented to the clinic with follow-up on FTT. The patient was born vaginally at 36 weeks with a birth weight of 4.15 pounds.

The infant was born without any complications except an elevated bilirubin level of 11.1 at birth. On one-week follow up, the patient weighed 5.6 pounds. The infant was exclusively breastfed for 1 month and switched over to Similac Advance for 12 months, without any additional vitamin D supplementation. The patient was up to date on her vaccinations with no known allergies or family history. Her mother was never tested for vitamin D levels during pregnancy.



At her two-and-a-half-month examination, the infant weighed 9.5 pounds with a height of 54 cm and a head circumference of 37.25 cm. Her weight and height at this visit fell below the fifth percentile with a head circumference above the tenth percentile. The infant was followed every 2 months to closely monitor her weight, height, and head circumference to ensure adequate growth (Chart 1, Chart 2).

The infant was continuously monitored through regular checkups. At her 12-month visit, her weight was at 10th percentile, height at 50th percentile and head circumference at 25th percentile. With insufficient weight and height growth, labs were ordered to further evaluate causes of FTT. The patient's initial lab included a comprehensive metabolic panel (CMP) (Table 1) and complete blood count (CBC) with iron (Table 2). Initial CMP showed a blood urea nitrogen (BUN) of 6, creatinine of 0.3, total protein of 6.3, iron of 42 and alkaline phosphatase of 1609, and the rest of the labs were unremarkable. These lab findings plus the clinical finding of FTT indicated signs of malnutrition-induced rickets and iron deficiency. To further confirm a diagnosis of Rickets, CMP was repeated with Vitamin D 25-hydroxyvitamin levels (Table 1), renal ultrasonography, and radiographic imaging of the knee (Figure 1). The patient's vitamin D 25-hydroxyvitamin was 7.4, showing vitamin D deficiency. Her renal ultrasonography was unremarkable, and the radiographic imaging of the knee showed transverse sclerotic metaphyseal bands involving the distal femoral metaphysis and portion of the proximal tibial metaphysis, findings consistent with untreated rickets.

3. MANAGEMENT AND TREATMENT

The patient was referred to endocrinology for evaluation of FTT, low vitamin D levels, and concerns of the diagnosis of Rickets. Endocrinology repeated labs, showing Vitamin D of 28 indicating insufficiency and an elevated alkaline phosphatase of 229. All other lab results were unremarkable. Endocrinology recommended that the patient start cholecalciferol 800 IU daily and intake calcium rich foods with added butter and fats to increase calorie density in her diet.

4. OUTCOME

Following initiation of cholecalciferol 800 IU daily and implementation of a calorie dense, calcium rich diet, the patient demonstrated gradual improvement in growth and biochemical parameters. Within two months of therapy, serum 25-hydroxyvitamin D increased from 7.4 ng/mL to 28 ng/mL, and alkaline phosphatase levels decreased from 1609 U/L to 229 U/L. Clinically, the child showed improved appetite, energy, and weight gain, with height and weight percentiles trending upward on serial growth charts. Radiographic follow-up revealed resolution of metaphyseal sclerotic changes consistent with healing rickets. The patient tolerated vitamin D supplementation without adverse effects. At the three month follow-up, she remained clinically stable with normalized laboratory values and continued catch-up growth.

5. PROGNOSIS

Malnutrition-induced rickets is an easily treatable and preventable bone disease, and is often completely curable with early identification and adequate nutritional intervention. In our case, we expect the patient to have a good prognosis with little to no long term permanent growth defects.

6. DISCUSSION

Failure to Thrive

FTT is defined as an inadequate growth in weight; diagnosed either by a series of weight measurements that falls below the fifth percentile or a continuous decrease in weight in >2 percentiles.⁵ An initial clinical finding of FTT warrants an extensive workup including a thorough history and physical examination to determine the underlying cause. The most common cause of FTT is divided into inadequate calorie intake, increased metabolic demands, or malabsorption with adequate feeding.^{5,6}

Once FTT is identified in an infant, many aspects of the history must be explored.⁷ Due to its complexity, evaluation can begin with calorie intake since it is considered the primary cause. Details on the type of food, amount, frequency, if the infant is breast-fed or formula-fed, can assist in supporting whether the infant is receiving adequate calorie intake or not.⁷ The patient's mother in this case was requested to keep a nutrition log with information on the infant's diet, including type of food eaten, amount, and how often. The



mother endorsed that the patient was a picky eater without any vegetables in her diet. She drinks water, juice, occasionally almond milk, but does not consume a lot of milk. Based on evaluation of the patient's nutrition, there was high suspicion that nutritional deficiency was the primary cause of the FTT. However, a thorough evaluation still needs to be conducted to eliminate other causes.

Furthermore, an elimination history is explored to identify gastrointestinal or genitourinary conditions that lead to excessive excretion of nutrition/calories, decreasing overall calorie.⁷ Gathering details on birth history can depict if the infant has any medical conditions such as biliary atresia, celiac disease, chronic gastrointestinal conditions, or inborn errors of metabolism which increases the risk of malabsorption.^{7,5} A newborn screening needs to be done to rule out any abnormal congenital disorders.

FTT due to inadequate nutrition intake or malabsorption can lead to various vitamin deficiencies. These vitamin deficiencies might be hinted at with a thorough history such as skin rash or failure to thrive. In our case, the infant initially depicted FTT on each wellness visit, later presenting with low 25-hydroxyvitamin D level and low iron, revealing vitamin D deficiency. After investigating the causes of FTT, it was determined that our patient likely had FTT secondary to nutritional deficiency.

Vitamin D Deficiency

Severe vitamin D deficiencies in infants can lead to Rickets, a failure in bone mineralization.² Vitamin D is responsible for calcium and phosphorus reabsorption. Thus, low levels will result in elevated alkaline phosphatase, hypocalcemia and hypophosphatemia.² Nutritional vitamin D deficiency can lead to inadequate calcium, impairing mineralization at the growth plate in infants.⁸ To prevent nutritional vitamin D deficiency, the child needs to consume enough vitamin D and calcium.

Labs need to be ordered in an infant with FTT to assist in identifying the cause since there can be various vitamin deficiencies. Initial labs should include a CMP to assess for electrolyte, kidney, and liver function, which can all affect growth. Additionally, thyroid stimulating hormone (TSH), free T4 and growth hormones need to be ordered, because both thyroid hormones and growth hormones can impair growth, hindering the ability to gain weight or increase in height. The patient's initial lab showed a BUN of 6, Cr of 0.3, and total protein of 6.3; potentially signs of malnutrition. Her thyroid hormone and free T4 levels were within range, iron level was 42, and alkaline phosphatase (ALKP) was 1609. Clinical findings and labs reveal signs of malnutrition-induced Rickets. These abnormal labs warrant further testing such as repeat CMP with vitamin D 25-hydroxyvitamin, renal ultrasonography, and radiographic imaging of the knee. The patient's vitamin D 25-hydroxyvitamin was 7.4, showing vitamin D deficiency. Her renal ultrasonography was unremarkable, and the radiographic imaging of the knee showed transverse sclerotic metaphyseal bands involving the distal femoral metaphysis and portion of the proximal tibial metaphysis, findings show treated rickets.

Due to these radiological findings, it was determined that the patient's biological and chronological age did not match, confirming a diagnosis of rickets. A diagnosis of rickets requires extensive treatment to prevent worsening of the developmental delay in an infant. The patient was referred to endocrinology and they noted that she had slightly bowed knees bilaterally. Repeated 25-hydroxyvitamin D was 28, suggesting vitamin D insufficiency. Early detection of nutritional-induced rickets is essential in curing the disease and preventing any further complications. Therefore, changing the patient's diet and adding vitamin supplements is extremely important in treatment. In our case, the patient was recommended to start cholecalciferol 800 IU daily and increase intake of calcium rich foods.

6. CONCLUSION

Our patient was extensively followed in the clinic for FTT, and a thorough workup with a complete history, physical examination, laboratory, and radiological findings led to the diagnosis of rickets. Finding the primary cause of FTT is essential in the treatment and prevention of developmental delay or serious complications, like rickets. This case emphasizes the importance of checking vitamin D levels in all pediatric patients who present with failure to thrive. Although the diagnosis of rickets is unusual and rare, it should always be kept in mind with a clinical presentation of failure to thrive.

7. ACKNOWLEDGMENTS

The authors would like to express their sincere appreciation to the Department of Pediatrics at Kid Care, Beckley, West Virginia, for their continuous support and collaboration throughout the preparation of this case report. We extend our gratitude to the endocrinology team for their valuable input in the diagnosis and management of this patient. We are also grateful to the patient's family for their cooperation and consent, which made this report possible.



8. DATA AVAILABILITY

The datasets generated during and/or analyzed during the current study are available in the National Institutes of Health repository.

Ethical Approval and Accordance

The protocol was approved by Dr. Lori McGrew, Chair of Institutional Review Board (IRB) at Lincoln Memorial University- Debusk College of Osteopathic Medicine in accordance with the Department of Health and Human Services (HHS). This project was determined to be outside of the scope of the IRB since it does not meet the U.S Department of Health and Human Services (HHS) definition of human subjects research.

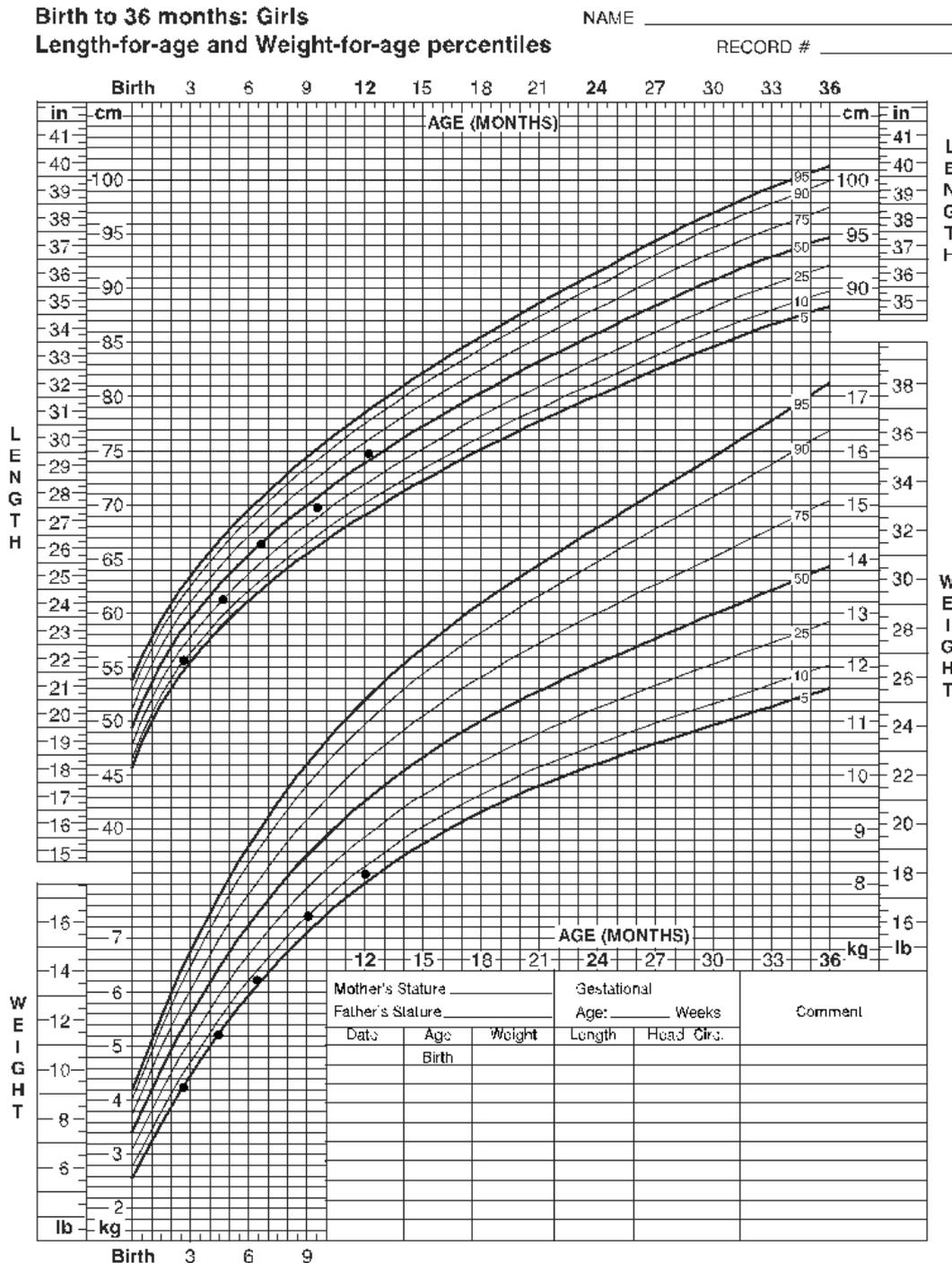
Consent to participate

Patient is under the age of 18 therefore, verbal informed consent was obtained by the patient's mother to write and publish this case report. The informed consent was waived by Dr. Lori McGrew, Chair of Institutional Review Board (IRB) at Lincoln Memorial University- Debusk College of Osteopathic Medicine.

Sources of financial support: N/A

REFERENCES

1. Pazirandeh S, Burns D. Overview of vitamin D [Internet]. UpToDate. Wolters Kluwer Health; 2025. Available from: <https://www.uptodate.com/contents/overview-of-vitamin-d>
2. Misra M. Vitamin D insufficiency and deficiency in children and adolescents [Internet]. UpToDate. Wolters Kluwer Health; 2024. Available from: <https://www.uptodate.com/contents/vitamin-d-insufficiency-and-deficiency-in-children-and-adolescents>
3. Chanchlani R, Nemer P, Sinha R, Nemer L, Krishnappa V, Sochett E, et al. An Overview of Rickets in Children. *Kidney International Reports*. 2020 Apr 11;5(7):980–90.
4. Carpenter T. Overview of rickets in children [Internet]. UpToDate. Wolters Kluwer Health; 2023. Available from: <https://www.uptodate.com/contents/overview-of-rickets-in-children>
5. Homan G. Failure to Thrive: A Practical Guide. *American Family Physician*. 2016 Aug 15;94(4):295–9.
6. Caglar D. Evaluation of weight loss in infants six months of age and younger [Internet]. UpToDate.
7. Wolters Kluwer Health; 2024. Available from: <https://www.uptodate.com/contents/evaluation-of-weight-loss-in-infants-six-months-of-age-and-younger>
8. Smith A, Shah M, Badireddy M. Failure to Thrive. *StatPearls*. 2023 Nov 12; Sahay M, Sahay R. Rickets-vitamin D deficiency and dependency. *Indian Journal of Endocrinology and Metabolism* [Internet]. 2012;16(2):164. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3313732/>



Published May 30, 2000 (modified 4/20/01).
 SOURCE: Developed by the National Center for Health Statistics in collaboration with
 the National Center for Chronic Disease Prevention and Health Promotion (2000).
<http://www.cdc.gov/growthcharts>



Figure 1: Birth to month 12 growth chart of weight and height.



| Comprehensive Metabolic Panel (CMP)/ Basic Metabolic Panel (BMP) | | | |
|--|--------------|---------------|------------|
| | Initial labs | Repeated labs | Range |
| Na ⁺ | 142 | 137 | 137-144 |
| K | 3.8 | 4.4 | 3.5-5.0 |
| Cl | 112 | 110 | 98-108 |
| CO ² | 23 | 23 | 21-32 |
| ANION GAP | 11 | 8 | 0-40 |
| | | | |
| GLU | 77 | 70 | 55-115 |
| BUN | 6 | 7 | 7-18 |
| CREATININE | 0.3 | 0.3 | 0.6-1.3 |
| T PROTEIN | 6.3 | 6.7 | 6.4-8.2 |
| ALBUMIN | 3.6 | 3.8 | 3.4-5.0 |
| GLOBULIN | 2.7 | 2.9 | 2.1-3.4 |
| A/G RATIO | 1.3 | 1.3 | 1.0-2.6 |
| CALCIUM | 8.9 | 9.7 | 8.5-10.1 |
| | | | |
| BILT | <0.1 | 0.4 | 0-5.0 |
| SGOT/AST | 33 | 31 | 15-37 |
| SGPT/ALT | 17 | 17 | 12-78 |
| ALKP TOTAL | 1609 | 262 | 108-345 |
| | | | |
| T4 Free | 1.06 | | 0.76-1.46 |
| TSH | 2.02 | | 0.358-3.74 |
| | | | |
| VIT D-25 HYDROX | | 7.4 | 32-100 |

Figure 3: Comprehensive Metabolic Panel (CMP)/ Basic Metabolic Panel (BMP).

| Complete Blood Count (CBC) | | |
|----------------------------|------|-----------|
| WBC | 6.1 | 6.2-19.9 |
| RBC | 4.32 | 3.10-5.00 |
| HGB | 12.4 | 9.0-13.4 |
| HCT | 36.6 | 27-40% |
| MCV | 85 | 82-88.7 |

| | | |
|------|------|-----------|
| MCH | 29 | 28-30 |
| MCHC | 34 | 33.5-34.5 |
| RDW | 12.3 | 11.5-14.5 |
| PLT | 365 | 140-450 |
| | | |
| IRON | 42 | 50-175 |

Figure 4: Complete Blood Count (CBC) and iron.



Figure 5: Radiographic imaging of the knee shows transverse sclerotic metaphyseal band involving the distal femoral metaphysis and portion of the proximal tibial metaphysis.

Cite this Article: Aladham, N., Khiami, A., Abbas, N., Prashar, J., Almeida, J. (2025). Failure to Thrive in an 18-Month-Old Female Infant with Rickets: A Case Report. *International Journal of Current Science Research and Review*, 8(11), pp. 5556-5563. DOI: <https://doi.org/10.47191/ijcsrr/V8-i11-13>