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Congenital vitamin D deficiency: presenting with feeding difficulty in early infancy: a case report

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Abstract

Background Vitamin D deficiency remains a significant public health concern, particularly among exclusively breastfed infants. Infants born to mothers with vitamin D deficiency, often influenced by cultural factors affecting diet, lifestyle, and clothing, are at increased risk of developing early and potentially fatal complications of hypocalcemic vitamin D deficiency. While seizures and tetany are well-recognized manifestations of hypocalcemia in infants, less common symptoms, such as feeding difficulties and recurrent apnea, are rarely documented. .

Case presentation We present the case of a 50-day-old Ethiopian full-term infant, born to a Muslim Ethiopian mother, who experienced feeding difficulties, frequent brief episodes of apnea, and cyanosis since birth. The underlying cause was identified as hypocalcemia-induced laryngospasm due to congenital vitamin D deficiency, which resulted from maternal vitamin D deficiency. The mother, who has worn a niqab since childhood, had minimal sun exposure, contributing to her low vitamin D levels. The infant was successfully treated with intravenous calcium gluconate, followed by oral calcium and vitamin D supplementation, leading to complete resolution of symptoms and normalization of biochemical parameters.

Conclusion Hypocalcemia-induced laryngospasm presenting with feeding difficulties and recurrent apneic episodes is a rare yet serious clinical condition. This report emphasizes the need to consider hypocalcemia as a potential cause of unexplained feeding difficulties or recurrent apnea in newborns and infants. Therefore, clinicians should remain vigilant and maintain a high index of suspicion to ensure timely diagnosis and treatment. It is essential to measure serum calcium and vitamin D levels in both the mother and infant, particularly for newborns of high-risk mothers, such as those who wear conservative religious clothing like a niqab. Furthermore, routine biochemical screening for vitamin D deficiency should be incorporated into antenatal care for all high-risk mothers, with appropriate supplementation to prevent potential complications in both mothers and their infants.

Keywords Congenital rickets, Vitamin D deficiency, Hypocalcemia, Laryngospasm, Feeding difficulty

Introduction

Vitamin D is an essential fat-soluble vitamin crucial for human physiology. It plays a pivotal role in maintaining normal serum levels of calcium and phosphate, which are necessary for bone mineralization, muscle contraction, nerve conduction, and overall cellular function. Despite its importance, vitamin D deficiency (VDD) is a widespread global health issue and remains one of the most underdiagnosed and undertreated nutritional

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deficiencies. While its skeletal complications are well-documented, the broader consequences of VDD are still being uncovered [1, 2].

The prevalence of VDD is particularly high among pregnant and lactating women. Maternal vitamin D status is a key determinant of neonatal and infant vitamin D levels, as the fetus relies entirely on maternal stores during pregnancy and early stages of infancy [3]. Vitamin D, in the form of 25-hydroxyvitamin D [25(OH)D_3], crosses the placenta and has a half-life of approximately 2 months in the fetal circulation. During the second trimester, serum concentrations of 1,25-hydroxyvitamin D increase by 50–100% compared with pre-pregnancy levels, and by 100% during the third trimester, ensuring adequate calcium transfer for fetal skeletal development [4, 5]. However, if the mother is deficient in vitamin D, the newborn will likely be born with VDD, predisposing the infant to symptoms such as hypocalcemia and related complications [6].

Vitamin D deficiency in infants, although often associated with exclusively breastfed infants, is infrequently reported. If left untreated, it can result in severe consequences, including hypocalcemic seizures, growth failure, lethargy, irritability, and increased susceptibility to respiratory infections [7]. While hypocalcemic seizures due to VDD in early infancy are well-documented in the literature, their presentation with feeding difficulties or recurrent apneic episodes has been rarely described [8]. Here, we present a case of an infant successfully treated for severe hypocalcemia caused by congenital VDD, manifesting as feeding difficulty and recurrent apneic episodes. We also outlined evidence-based preventive measures for both mothers and infants who may be at risk for vitamin D deficiency.

Case presentation

A 50-day-old Ethiopian full-term infant, born to a Muslim Ethiopian mother, presented to the emergency department with worsening respiratory symptoms, including rapid breathing, cough, recurrent brief episodes of apnea, hoarseness, and frequent gagging. The infant also had a persistent high-grade fever for 1 week. Since birth, the infant had experienced feeding difficulties, poor weight gain, cyanotic spells, and generalized hypotonia. Despite multiple treatments, the symptoms persisted. Notably, there was no history of seizures or tetany. On physical examination, the infant was in respiratory distress with a fever of 38.7 °C. Severe malnutrition was evident, with an anthropometric evaluation showing a weight-for-length (W/L) *z*-score of less than −3 standard deviations (SD). The head circumference was normal for this age. Muscle tone was weak, and auscultation revealed wheezing in both lungs, while heart sounds were normal with no murmurs. Neurological examination was unremarkable, and there were no dysmorphic features. The infant was born at term following an uncomplicated spontaneous vaginal delivery (SVD) and had a birth weight of 3.4 kg, appropriate for the gestational age. The pregnancy was uneventful. The mother, a 32-year-old Ethiopian Muslim, has worn a niqab covering her entire body except for her eyes since childhood. She did not take vitamin D or calcium supplements during pregnancy, and her diet lacked essential nutrients, relying predominantly on cultural foods. She had three healthy children, and the family history was unremarkable. The infant had been exclusively breastfed since birth.

Upon admission, the infant was diagnosed with severe acute malnutrition (SAM) and late-onset sepsis (LLOS) with a suspected chest focus to rule out congenital heart disease. Initial investigations revealed anemia (hemoglobin 8.5 g/dL, hematocrit 25.6%), a low white blood cell count (4500/mm^3) with a normal differential, and normal platelet counts. Chest X-ray and echocardiogram were normal. Other tests were within normal limits, including C-reactive protein, cerebrospinal fluid analysis, blood culture, and glucose levels. The infant received intranasal oxygen, broad-spectrum antibiotics, and nutritional support with diluted F-100 therapeutic milk for 3 days. Despite these interventions, the clinical condition worsened, with increased respiratory distress, hoarseness, stridor, and frequent apneic episodes with cyanosis.

The patient was subsequently transferred to the pediatric ward for further management. However, the underlying cause of the patient's clinical decline was not addressed appropriately. Then, considering the clinical presentation, hypocalcemia was suspected as the underlying cause of the symptoms, particularly the stridor and hoarseness. Laboratory investigations confirmed severe hypocalcemia, with ionized calcium at *0.65 mmol/L* (*reference range: 1.1–1.2 mmol/L*), total calcium at 1.76 mg/dL (reference range: 8.7–10 mg/dL), and elevated alkaline phosphatase at 694 U/L (*reference range: 150–300 U/L*). The patient was treated with multiple intravenous doses of 10% calcium gluconate. Remarkably, all symptoms, including feeding difficulty, apneic episodes, and stridor, resolved completely within 48 hours of initiating treatment. The observed clinical and biochemical abnormalities raised suspicion of congenital rickets. Further testing revealed low serum 25-hydroxyvitamin D [25(OH)D_3] levels, 10.2 ng/mL (*reference > 30 ng/mL*), confirming the diagnosis of hypocalcemic vitamin D deficiency. Radiological investigations, including wrist and chest X-rays, showed no evidence of rickets. Renal and liver function tests were within normal ranges. However, due to financial constraints, serum phosphorus and parathyroid hormone (PTH) levels could not be assessed (Table 1).

Table 1 Summary of initial laboratory and biochemical parameters of a 50-day-old full-term male infant with congenital vitamin D deficiency, 2023

Parameter	Results	References value
WBC × 10^3 (per µL)	4.5	4.0–12.0
Hemoglobin (g/dL)	8.5	11.5–14.5
Hematocrit (%)	25.6	33–43%
MCV (fL)	82.8	76–90
Platelet count × 10^3 (µl)	364	150–450
Ca^{+2} (mmol/L)	**0.65**	**1.1–1.2**
P (mg/dL)	NA	
Mg (mg/dL)	2.1	1.8–2.5
PTH (ng/L)	NA	
ALP (U/L)	**694**	**< 300**
Creatinine (mg/dL)	0.57	0.22–0.59
BUN (mg/dL)	14	7–18
25(OH)D (ng/mL)	**10.2**	**> 30**
AST (IU/L)	26	15–50
ALT (IU/L)	21	5–45
RBS (mg/dL)	104	60–100
CSF analysis	Normal	—
Blood culture	No growth	—
Wrist X-ray	Normal	—
Chest radiography	Normal	—
Echocardiography	Normal	—

Bold values are used to highlight the diagnostic and affected laboratory findings for emphasis

Ca^{+2} ionized calcium, *P* phosphorus, *Mg* magnesium, *ALP* alkaline phosphatase, *25(OH)D* 25-hydroxyvitamin D; *PTH* parathyroid hormone, *CSF* cerebospinal fluid, *WBC* white blood count, *MCV* mean corpuscular volume, *BUN* blood urea nitrogen, *ALT* alanine transaminase, *AST* aspartate transaminase, *RBS* random blood sugar, *NA* not available

The early onset of symptoms in this infant suggested that inadequate postnatal intake of vitamin D was unlikely to be the cause of the deficiency. Instead, the clinical and biochemical findings strongly indicated congenital vitamin D deficiency. A detailed maternal history and investigation revealed that the mother had limited sun exposure due to prolonged indoor activity and wearing a niqab, which covered her entire body. She also did not receive vitamin D supplementation during pregnancy. Maternal biochemical tests showed severe vitamin D deficiency, with low total calcium of 4.8 mg/dL (*reference range: 8.7–10 mg/dL*), a low 25(OH)D_3 level of 13.8 ng/mL (*normal range: 30-50 ng/mL*), and an elevated level of ALP at 405 U/L (*normal range: 30–120 U/L*), while renal and liver function tests were normal. These findings pointed to maternal nutritional vitamin D deficiency as the likely cause of the infant's congenital deficiency. The infant's symptoms, including feeding difficulties, recurrent apnea, hoarseness, and frequent gagging, were likely attributed to laryngospasm caused by severe hypocalcemia.

By the seventh day of treatment, the infant's ionized calcium levels improved to 0.9 mmol/L (reference range: 1.1–1.2 mmol/L), leading to a complete resolution of symptoms. The treatment was transitioned to oral Calcidenk (containing 1000 mg of elemental calcium and 880 IU of vitamin D_3) at a dosage of half a tablet daily. The infant showed steady weight gain, and the anthropometric abnormalities were corrected. After a 2-week hospital stay, the infant was discharged with a maintenance plan of daily Calcidenk for 3 months. At the 1-month follow-up, the baby was healthy, well-nourished, and symptom-free and demonstrated normal feeding habits and age-appropriate developmental milestones. The mother also received calcium and vitamin D supplementation.

Discussion

Vitamin D deficiency (VDD) is a global health challenge and remains one of the most underdiagnosed and undertreated nutritional deficiencies. Despite Ethiopia's abundant sunlight as a tropical country, VDD is widely prevalent [1, 9]. Infants who are exclusively breastfed are particularly vulnerable, especially when born to mothers with risk factors such as low vitamin D stores, dark skin, conservative clothing such as the niqab, indoor lifestyles, or sunscreen use, all of which limit ultraviolet light exposure, the primary source of vitamin D synthesis [6, 10].

Maternal VDD is a significant risk factor for VDD in newborns and infants. The only source of vitamin D available to the fetus is derived from the mother, and the vitamin freely crosses the placenta, particularly during the second half of pregnancy. Infants of mothers with VDD have reduced concentrations of 25-hydroxyvitamin D in their cord blood samples. Therefore, adequate vitamin D supply during pregnancy is the primary factor influencing a baby's vitamin D status during the neonatal and early infancy stages, rather than obtaining vitamin D solely from milk after birth [3, 11].

Vitamin D is key in fetal growth through its interaction with parathyroid hormone and calcium (Ca^{2+}) homeostasis. Studies have demonstrated that insufficient vitamin D levels during prenatal and postnatal periods can significantly affect bone mineralization, closely associated with low birth weight (LBW) and small-for-gestational-age (SGA) births. Furthermore, several studies have highlighted the influence of micronutrients on birth weight [12, 13]. However, congenital hypocalcemic VDD may not necessarily be associated with SGA or LBW. The absence of SGA or LBW can result from several factors. Maternal compensatory mechanisms during pregnancy, such as enhanced placental nutrient transfer, may mitigate the impact of the deficiency on fetal growth.

Additionally, the specific timing and severity of the deficiency play crucial roles; for instance, deficiencies that manifest later in gestation may not significantly impair overall growth but could affect other developmental processes. Consequently, the interplay of these factors can result in a clinical presentation that deviates from the expected phenotypic features [14].

In neonates and young infants, VDD can present differently than the classic signs of rickets. The most common initial symptoms may include incidental hypocalcemia or seizures. Often, skeletal deformities are minimal or absent, and symptoms caused by hypocalcemia can occur without any radiological evidence of rickets [15, 16]. Similarly, in our case, the infant had hypocalcemia caused by VDD, but there were no radiological signs of rickets, and the patient did not experience any seizures or tetany. Hypocalcemia in children can present with a wide range of symptoms, ranging from subtle to severe. Common manifestations include lethargy, focal clonic seizures, jitteriness, and carpopedal spasms. Rarely, hypocalcemia can lead to stridor due to laryngospasm. The association between stridor and nutritional rickets has been documented in the medical literature, emphasizing the potential respiratory complications of severe vitamin D deficiency [17, 18]. Laryngospasm due to hypocalcemia has been reported in various contexts. For instance, a case involving a child with renal dysplasia described hypocalcemic laryngospasm causing stridor and tetany. Hypocalcemia causes laryngospasm mainly due to its effects on neuromuscular excitability [19]. Another report detailed a fatal case of acute stridor and biphasic wheezing in an infant, attributed to severe hypocalcemia [20]. Additionally, laryngeal spasms leading to stridor and cyanosis of the limbs have been documented in a case report [21], underscoring the potential severity of this rare presentation. However, hypocalcemia-induced laryngospasm, which primarily manifests as feeding difficulties and recurrent apneic episodes, is infrequently reported and can lead to misleading diagnoses.

Our case presented all the known risk factors for VDD. The maternal history and biochemical profiles indicated nutritional VDD. The mother's low vitamin D status was likely due to inadequate sunlight exposure as a result of wearing a niqab, which is a dark-colored conservative Muslim garment that covers the entire body except for the eyes. Studies have shown that women who wear veils or niqabs are at a higher risk of VDD compared with those who do not wear such coverings. These clothing styles limit the amount of sunlight that reaches the skin and hinder the body's ability to synthesize vitamin D from sunlight [22, 23]. Additionally, she did not receive vitamin D supplements during her pregnancy and worked for long periods in an indoor environment. Even with the improved prenatal care available today, some babies are still born with low levels of vitamin D. Physicians should be aware of the risk factors that may lead to this condition and stay vigilant for signs of congenital VDD to initiate appropriate treatment and prevent complications that can occur shortly after birth [24].

To effectively prevent both maternal and perinatal VDD, it is essential to ensure that maternal vitamin D levels are adequate before and during pregnancy. This is particularly important for high-risk women, including those who wear fully concealing religious garments, lead predominantly indoor lifestyles, belong to dark-skinned ethnic groups, or do not consume dietary products that are sufficiently fortified with vitamin D. Therefore, screening and proper supplementation are crucial for all pregnant women, especially around the time of conception [25]. While there is insufficient evidence to recommend universal screening for vitamin D deficiency in all pregnant women, those high-risk groups should undergo serum vitamin D evaluation, and supplementation should be provided as needed [26]. A recent editorial by Hollis and Wagner highlights the importance of vitamin D supplementation for women planning to become pregnant. This recommendation is similar to that for the general adult population and takes into account their serum vitamin D levels before pregnancy [27]. In our case, despite the high risk for VDD, the mother was not screened for vitamin D deficiency, nor provided with vitamin D supplements. This underscores the need for effective prevention strategies, including ensuring that maternal vitamin D levels are adequate before and during pregnancy, particularly for women with limited sun exposure or other risk factors. Additionally, screening for VDD should be conducted in high-risk women even before pregnancy. Proper antenatal care and postnatal monitoring can mitigate the adverse effects of VDD and promote better health outcomes for both mothers and infants.

Conclusion

Hypocalcemia-induced laryngospasm, presenting with feeding difficulties or recurrent apnea episodes, is a rare but serious clinical manifestation. While seizures and tetany are well-known signs of hypocalcemia, these atypical symptoms are less recognized, often leading to diagnostic challenges and potential misdiagnoses. This report underscores the need to consider hypocalcemia as a possible cause of unexplained feeding difficulties or recurrent apnea in infants. Therefore, clinicians should maintain a high index of suspicion to ensure prompt diagnosis and treatment. Determining serum calcium and vitamin D levels in both the mother and infant is crucial, particularly for newborns of high-risk mothers, such as those wearing conservative religious clothing like the

niqab. Routine biochemical screening for vitamin D deficiency should be an integral part of antenatal care for all high-risk mothers. Furthermore, high-risk women should undergo vitamin D deficiency screening even before pregnancy, with appropriate supplementation as needed. Routine biochemical screening for vitamin D deficiency should be an integral part of antenatal care for all high-risk mothers. Furthermore, all high-risk women should undergo vitamin D deficiency screening even before pregnancy, with appropriate supplementation as needed.

Abbreviations

$25(OH)D_3$	25-hydroxyvitamin D
LLOS	Late-late-onset sepsis
SAM	Severe acute malnutrition
PTH	Parathyroid hormone
VDD	Vitamin D deficiency

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Availability of data and materials
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Declarations

Ethics approval and consent to participate
The institution does not require ethical approval for the publication of a single case report.

Consent for publication
Written informed consent was obtained from the patient's legal guardian for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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