

CASE REPORT

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# Maternal hypovitaminosis D presenting as late-onset hypocalcemic seizure in a term neonate: a case report

Achinta Kumar Mallick<sup>1\*</sup> , Raj Kumar Yadav<sup>2</sup>, Kiran Sannalli<sup>3</sup> and Nitul Mohan Bewal<sup>4</sup>

## Abstract

**Background** Vitamin D deficiency is a known morbidity among pregnant women. Maternal hypovitaminosis D results in poor fetal transplacental vitamin D accretion and decreased stores in the neonate. This in turn may lead to hypocalcemia in neonates, having presentations from being asymptomatic and tremors to hypocalcemic seizures. There are fewer case reports of hypocalcemic neonatal seizures as a result of maternal vitamin D deficiency.

**Case presentation** A term male neonate developed multiple episodes of multifocal seizures on day six of life associated with hypocalcemia. He was also found to have hyperphosphatemia, raised alkaline phosphatase, and elevated parathormone levels with normal serum magnesium and renal function tests. Neonate and maternal serum vitamin D3 levels were also low. A diagnosis of late-onset hypocalcemic seizure secondary to maternal hypovitaminosis D was made and the baby made an uneventful recovery with parenteral calcium and oral vitamin D supplementation. On follow-up with therapy, the baby remained asymptomatic with normal serum calcium, phosphate, and vitamin D3 levels.

**Conclusion** Hypovitaminosis D must also be considered as a contributing factor for late-onset hypocalcemic seizure in neonates.

**Keywords** Hypocalcemia, Late-onset, Seizure, Hypovitaminosis D, Case report

## Background

Vitamin D deficiency is a global morbidity among pregnant women. [1]. Maternal hypovitaminosis D results in poor fetal transplacental vitamin D accretion and decreased stores in the neonate [2, 3]. Hypocalcemia as a result of hypovitaminosis D in neonates may have varying presentations from asymptomatic, tremors to hypocalcemic seizure manifestations [4]. As per the literature, there are fewer case reports of neonatal hypocalcemia

with symptomatic manifestation as a result of maternal vitamin D deficiency. We hereby report a neonate with late-onset hypocalcemic seizure secondary to maternal vitamin D deficiency and made an uneventful recovery. This article will enlighten the readers and will definitely change the approach to the management of such cases in the near future.

## Case presentation

A 6-day-old term male neonate developed multiple episodes of multifocal tonic-clonic convulsions, lasting for 30–50 s. He was the firstborn of non-consanguineous marriage to apparently healthy parents of normal stature. The antenatal period was uneventful without any significant similar family history. The baby was born vigorously by elective cesarean section, weighing 3.84 kg, and was immediately roomed in with the mother. The baby

\*Correspondence:

Achinta Kumar Mallick  
achinta.mallick@gmail.com

<sup>1</sup> Dept of Pediatrics, 174 Military Hospital, Bathinda, Punjab, India

<sup>2</sup> Dept of Pediatrics, 159 General Hospital, Ferozpur, Punjab, India

<sup>3</sup> Dept of Pediatrics, 7 Air Force Hospital, Kanpur, Uttar Pradesh, India

<sup>4</sup> Dept of Internal Medicine, 174 Military Hospital, Bathinda, Punjab, India

was on exclusive breastfeeds since birth, with normal urine output. The baby was discharged on day four and was feeding well without any such events during home stay till presentation to the hospital. On examination, the baby was irritable and the anterior fontanel was at level. Capillary refill time was normal and there were no facial dysmorphisms or skeletal malformations. Other systemic examinations were normal. Investigations revealed normal blood glucose and low serum calcium 6.7 mg/dL, ionized calcium 0.70 mmol/L (normal levels 1.20–1.38 mmol/L). Blood urea was 13 mg/dL and serum creatinine was 0.4 mg/dL. Cerebrospinal fluid (CSF) analysis, serum protein, and albumin levels were normal with a negative septic screen. Arterial blood gas showed normal pH and bicarbonate, normal serum ammonia level, and negative urine ketones. The baby was started on a parenteral bolus of 10% calcium gluconate at a dose of 2 mL/kg followed by daily maintenance of 8 mL/kg for 72 h. Further investigations revealed elevated serum phosphate of 9.1 mg/dL (normal: 3.8–6.5 mg/dL) and alkaline phosphatase (ALP) of 536 IU/L (normal: 145–420 IU/L). Serum magnesium was 1.8 mg/dL (normal: 1.5–2.5 mg/dL). The thyroid function test was normal. The serum vitamin D3 level was low, (12 ng/mL, normal 30–100 ng/mL) with a mildly elevated parathormone (PTH) level of 78.3 pg/mL (normal: 12–72 pg/mL), at a serum calcium level of 6.7 mg/dL. Maternal serum vitamin D3 level was 6.9 ng/mL. Neonate cranial and abdominal ultrasonography did not reveal any abnormality. With evidence of hypocalcemia, hyperphosphatemia, normal renal function, elevated serum PTH, and low vitamin D3 levels, a diagnosis of late-onset hypocalcemic seizure secondary to maternal hypovitaminosis D was made. The baby was started thereafter on daily oral calcium supplementation at a dose of 50 mg/kg/day and cholecalciferol 2000 IU/day. Baby responded with the above medications. The baby had a good prognostic outcome. Serum calcium normalized and phosphorus level started decreasing after 1 week of therapy. On follow-up at 6 months, the baby was asymptomatic with normal serum calcium, phosphate, and alkaline phosphatase levels, with normal neurodevelopmental outcomes. The baby was continued on oral cholecalciferol (400 IU/day) and calcium supplements thereafter.

## Discussion

Maternal vitamin D deficiency during pregnancy and lactation is a significant risk factor for rickets in infancy [1–3]. However, neonatal hypocalcemic seizure as a consequence of maternal vitamin D deficiency is not reported much in the literature. Vitamin D insufficiency in healthy pregnant women is not uncommon [5]. Newborn babies of such mothers have reduced vitamin D stores [6]. In

addition, the breast milk of such mothers is also vitamin D deficient [7]. Neonates born to mothers with vitamin D deficiency or insufficiency may also develop vitamin D deficiency and late-onset hypocalcemia manifestation [8, 9]. Exclusive breastfeeding without vitamin D supplementation is another risk factor for vitamin D deficiency in neonates and infants. Late-onset hypocalcemia may also be caused by high phosphate intake like cow's milk. However, there was no such association in our case [10]. Late-onset hypocalcemia caused by excessive phosphate load is usually accompanied by raised PTH and ALP levels [9]. Our case illustrates the importance of looking for vitamin D status in neonates presenting with seizures and hypocalcemia, as this is easily treatable. It is also debatable whether maternal vitamin D supplementation can prevent such a condition, however, there is a paucity of data regarding this. In our case, we ruled out the common causes of late-onset neonatal hypocalcemia (increased oral phosphate load, hypomagnesemia, renal insufficiency, hypoparathyroidism, metabolic alkalosis, and phototherapy) [11]. Our baby had no history of prematurity, maternal diabetes, perinatal asphyxia and sepsis, neonatal jaundice requiring phototherapy or artificial formula, or cow's milk intake. The baby also did not have facial dysmorphism. Transient hypoparathyroidism may also present with hypocalcemia and hyperphosphatemia, but our baby had an elevated PTH level.

## Conclusions

In a case of a neonate presenting with late-onset hypocalcemic seizure, maternal as well as neonatal hypovitaminosis D should also be considered apart from other common risk factors.

## Abbreviations

CSF	Cerebro spinal fluid
ALP	Alkaline phosphatase
PTH	Parathormone

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## Authors' contributions

AKM and RKY conceptualized and drafted the initial manuscript and reviewed and revised the manuscript. KS revised and finalized the editing of the manuscript. NMB critically reviewed the manuscript for important intellectual content. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

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## Availability of data and materials

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## Declarations

### Ethics approval and consent to participate

Not applicable.

### Consent for publication

Verbal and written informed consents were given by the patient's parents for the publication of this case.

### Competing interests

The authors declare that they have no competing interests.

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