

Symptomatic late onset hypocalcemia in a full term female neonate with vitamin D deficiency due to maternal hypovitaminosis D: A rare case report

Swathi Chacham, Janampally Ravikiran, Uppin Narayan Reddy, Jillalla Narsing Rao, Mahender Reddy, Imeduddin

ABSTRACT

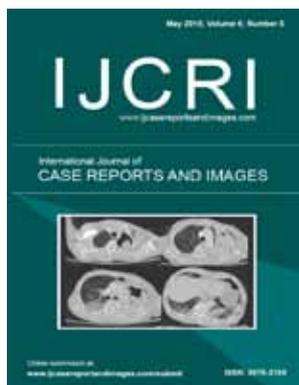
Introduction: Neonatal hypocalcemia (NH) is a common metabolic complication in neonates, more so in premature and high risk neonates. It is classified as early and late neonatal hypocalcemia. Early NH occurs in the first 24–48 hours of life while the late NH is observed at the end of the first week. Vitamin D deficiency is an important cause for hypocalcemic seizures in neonates, in developing countries. High rate of skeletal growth coupled with low vitamin D stores and maternal vitamin D deficiency makes them vulnerable to vitamin D deficiency.

Case Report: A 2600 grams, term female neonate was born to a gravid 3 mother by C-section and had normal extra-uterine transition (APGAR score: 8&9 at 5 and 10 minutes of life). On eighth day of life, the neonate had multifocal clonic seizures with normal sensorium in between. No maternal risk factors were identified. There was no fever, lethargy, poor feeding, and clinical findings were unremarkable in the neonate. There was family history of neonatal seizures. Initial blood sugar and magnesium were normal. However, serum calcium levels were low (total 5.9 mg/dL, ionized 0.9 mg/dL) along with low phosphorous levels (1.7 mg/dL). Sepsis screen was negative, blood culture was sterile and cerebro spinal fluid analysis was normal. Similarly, neurosonogram, electroencephalogram, serum ammonia and lactate were normal, suggesting late onset hypocalcemic seizures. Both the neonatal and maternal vitamin D1 and 25-OH vitamin D were low, confirming maternal vitamin D deficiency causing neonatal vitamin D deficiency. The neonate responded to calcium and vitamin D supplementation with normal serum calcium levels in follow-up.

Conclusion: We report a term, female neonate with late onset hypocalcemic seizures and vitamin D deficiency, due to maternal vitamin D deficiency.

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Keywords: Neonatal hypocalcemia, Vitamin D deficiency, Seizures, Neonate

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INTRODUCTION

Neonatal hypocalcemia (NH), a commonly reported metabolic abnormality. Ionized calcium is vital for various metabolic pathways like blood. Coagulation, neuronal depolarization, integrity of cell membrane and plays a crucial role in enzyme catalysis. Major part of the body calcium exists in bones and muscles (99%) and the rest of the calcium is present in extracellular fluid (1%). Around 40% of the calcium in the extracellular fluid is bound to albumin, 10% is bound to citrate, phosphorus, lactate and sulfate and the rest (50%) exists as free ionized form, aiding in metabolic functions [1, 2]. Hypocalcemia is defined as total serum calcium of <8 mg/dL (2 mmol/L) or ionized calcium of <1.2 mmol/L in term neonates and <7 mg/dL (1.75 mmol/L) of total calcium or <4 mg/dL (1 mmol/L) of ionized calcium in preterm infants [3]. Neonatal hypocalcemia is classified into early and late based on the time of presentation [1]. The early NH usually manifests within 72 h, requiring short term calcium supplementation. While, the late NH occurs after 1st week of life and requires long-term calcium therapy [4–6]. There is a physiological nadir in serum calcium levels at 24–48 hours of life in healthy term neonates, which can reach hypocalcemic levels in neonates with perinatal risk factors like maternal diabetes, prematurity and perinatal asphyxia [1, 4–7], leading to early NH. Late NH usually results from either increased phosphate load (due to cow milk or renal insufficiency), hypomagnesemia, maternal vitamin D deficiency leading to neonatal vitamin D deficiency and hypoparathyroidism [1]. Breastfed infants born to and nursed by vitamin D deficient mothers usually have low serum 25(OH) D levels.

CASE REPORT

A 2600 grams full term female neonate presented with multifocal clonic seizures on eighth day of life. The neonate had normal sensorium in between. Seizures were not associated with fever, lethargy or feeding abnormalities and the clinical examination did not reveal any dysmorphic facies. Also, other clinical findings were unremarkable. This was born of a non-consanguineous marriage, to a gravid 3 mother by C-section and had normal extra uterine transition. There is no history of maternal diabetes, pregnancy induced hypertension, epilepsy and drug intake. Maternal hypothyroidism was present, which was controlled with thyroid replacement therapy. Family history of neonatal seizures, developmental delay and renal malformation was present in the elder male sibling, who died in infancy. Hence,

in this neonate seizures were attributed to metabolic causes and the neonate was investigated. Initial blood sugar and magnesium were normal. However, the serum calcium levels were low (total 5.9 mg/dL, ionized 0.9 mg/dL) along with low phosphorous levels (1.7 mg/dL). Sepsis screen was negative, blood culture was sterile and the cerebrospinal fluid (CSF) analysis was normal, ruling out meningitis as the cause for seizures. Similarly, the neurosonogram, electroencephalogram (EEG), serum ammonia and lactate levels were normal, ruling out inborn errors of metabolism. There was no metabolic acidosis in the arterial blood gas analysis and the abdominal ultrasonography showed normal kidneys. Hence the seizures in this case were attributed to late onset hypocalcemia and the neonate was further investigated for hypoparathyroidism and vitamin D deficiency. Chest X-ray showed normal thymic shadow, ruling out the possibility of congenital hypoplasia of parathyroid glands. Serum parathyroid hormone and thyroid profile was normal. However, vitamin D levels were low (7.9 ng/mL) and 25 OH vitamin D levels were also low 23.30 ng/mL (normal range 30 to 74 ng/mL). The neonate's thyroid profile was normal. The cause for vitamin D deficiency was attributed to maternal vitamin D deficiency and the level of mother's vitamin D were done, which were found to be low. Thus, the diagnosis of maternal vitamin D deficiency, resulting in neonatal hypovitaminosis D and symptomatic late onset neonatal hypocalcemia was confirmed. The neonate was treated symptomatically and responded to calcium supplementation (100 mg/kg/day) along with vitamin D supplementation (1000 IU/day) with normalization of serum calcium and vitamin D levels in follow-up at four weeks. The supplements were continued till six months of age and the infant was, neurodevelopmentally, normal at sixth month.

DISCUSSION

Neonatal hypocalcemia (NH) is a common metabolic event in the neonatal period, while early onset NH is a frequent manifestations in high risk neonates, late onset NH is rare. Incidence of late onset hypocalcemia in breastfed neonates is 1/10000 while, that in formula fed infants is 30/10000 [8]. Resistant or prolonged hypocalcemia is defined as symptomatic hypocalcemia not responding to appropriate doses of calcium supplementation, calcium requirement beyond 72 h of age in neonates and hypocalcemia manifesting beyond 1st week of life [1]. As the index infant had hypocalcemia beyond first week of life, it qualifies for resistant or prolonged hypocalcemia. Mostly prevalent causes of late onset NH include phosphate overload, hypoparathyroidism (transient or permanent), hypomagnesemia and vitamin D deficiency [1, 2]. Late onset NH is usually symptomatic and presents with tetany or seizures. The index neonate had severe hypocalcemia (total 5.9 mg/dL, ionized 0.9 mg/dL) manifesting with

seizures after first week of life and was investigated for neonatal seizures including the causes for late onset NH like hypomagnesemia, hypoparathyroidism and vitamin D deficiency. Evaluation for seizures revealed normal blood sugar levels, negative sepsis screen, sterile blood culture along with normal CSF analysis, ruling out hypoglycemia and meningitis. Accordingly, neurosonogram, electroencephalogram (EEG), serum ammonia (39 µg/dL) and lactate (4.5 mg/dL) were normal, ruling out structural malformations and inborn errors of metabolism respectively. Further evaluation for late onset NH showed normal serum magnesium and parathormone (PTH) levels, ruling out the possibility of hypomagnesemia and hypoparathyroidism. Then the neonate was evaluated for vitamin D deficiency and both the serum vitamin D1 (7.9 ng/mL) and 25 OH vitamin D 23.30 ng/mL (normal range 30–74 ng/mL) were low, confirming hypovitaminosis D. The most important causes of neonatal vitamin D deficiency include maternal vitamin D deficiency, renal insufficiency, malabsorption, and hepatobiliary disease. As the index neonate had normal renal function, liver function tests along with normal renal architecture and hepatobiliary tree in abdominal ultra sound, renal and hepatobiliary causes for hypovitaminosis D were ruled out. There were no features of malabsorption in the neonate. As mother's vitamin D levels were low, maternal vitamin D deficiency was attributed to the neonatal hypovitaminosis D and symptomatic late onset neonatal hypocalcemia.

CONCLUSION

We report a term female neonate with symptomatic late onset neonatal hypocalcemia due to vitamin D deficiency and maternal hypovitaminosis D. There was clinical and biochemical response to calcium and vitamin D supplementation.

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Author Contributions

Swathi Chacham – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Janampally Ravikiran – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Uppin Narayan Reddy – Analysis and interpretation of data, Revising it critically for important intellectual

content, Final approval of the version to be published
Jillalla Narsing Rao – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published
Mahender Reddy – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published
Imeduddin – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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