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Research Article

Contribution of Vitamin D Deficiency to Hypocalcemic Seizures in Infants Aged 1 Month to 2 Years

Dr Meghashree N[1*], Dr Nitish Kumar P Nandini[2], Dr Chandrashekhara[3]
[1*]Assistant Professor, Department of Pediatrics, Kodagu Institute of Medical Sciences, Madikeri, Karnataka-571201, India.
[2]Assistant Professor, Department of Pediatrics, Raichur Institute of Medical Sciences, Raichur, Karnataka-584102, India.
[3]Senior Resident, Department of Pediatrics, Navodaya Medical College Hospital and Research Center, Raichur, Karnataka-584103, India.
Correspondence Author: Dr Meghashree N
Assistant Professor, Department of Pediatrics, Kodagu Institute of Medical Sciences, Madikeri, Karnataka-571201, India.
Email: megha.narahatti@gmail.com
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ABSTRACT
Background: Vitamin D deficiency is a major contributor to disturbed calcium homeostasis, especially during early life when calcium demand is high due to rapid growth.
Objective: To evaluate the contribution of vitamin D deficiency to hypocalcemic seizures in infants aged 1 month to 2 years.
Methods: This prospective observational study was conducted in the Department of Pediatrics, Navodaya Medical College Hospital and Research Center, Raichur. A total of 100 infants aged 1 month to 2 years presenting with seizures were enrolled. Infants with CNS infection, congenital brain malformation, and maternal drug or supplement exposure affecting bone mineral metabolism were excluded. Demographic details, feeding history, seizure characteristics, clinical presentation, and biochemical parameters were recorded.
Results: Among 100 infants presenting with seizures, 27 (27.0%) had hypocalcemia. Vitamin D deficiency was identified as the major contributor to hypocalcemic seizures. Hypomagnesemia was present in 6 (6.0%) infants. The mean serum calcium level was 8.52 ± 1.713 mg/dL, while the mean corrected calcium level was 8.48 ± 1.637 mg/dL. The mean phosphorus level was 6.11 ± 1.957 mg/dL, alkaline phosphatase was 378.34 ± 233.574 U/L, and mean vitamin D level among tested infants was 13.25 ± 9.073 ng/mL. One infant with refractory seizures/status epilepticus had severe vitamin D deficiency with a vitamin D level of 4.6 ng/mL, while the infant's mother also had markedly low vitamin D level of 5.10 ng/mL.
Conclusion: Vitamin D deficiency was a major contributor to hypocalcemic seizures among infants aged 1 month to 2 years. The low mean vitamin D level among tested infants and the documented severe infant-maternal deficiency case highlight the importance of maternal and infant vitamin D status in maintaining calcium balance.

INTRODUCTION

Hypocalcemic seizures are an important and potentially preventable cause of seizures in infancy. Infants aged 1 month to 2 years are particularly vulnerable to disturbances in calcium homeostasis because this period is marked by rapid skeletal growth, high calcium requirement, immature regulatory mechanisms, and dependence on maternal nutritional stores during early life [1]. Calcium plays a central role in neuronal membrane stability, synaptic transmission, muscle contraction, and neuromuscular excitability. When serum calcium levels fall below the normal range, neuronal excitability increases, lowering the seizure threshold and predisposing infants to recurrent or prolonged seizures [2].

Vitamin D is one of the most important regulators of calcium and phosphorus metabolism [3]. It enhances intestinal calcium absorption, supports bone mineralization, and helps maintain normal serum calcium levels. Deficiency of vitamin D can impair calcium absorption from the gut, leading to reduced serum calcium levels and secondary hyperparathyroid responses [4]. In infants, severe vitamin D deficiency may present not only with features of rickets but also with acute hypocalcemic seizures, sometimes before obvious skeletal deformities become clinically apparent. This makes vitamin D deficiency a clinically significant but often under-recognized contributor to seizures in infancy [5].

The risk of vitamin D deficiency in infancy is influenced by multiple maternal, nutritional, and environmental factors. Infants born to vitamin D-deficient mothers may have low vitamin D stores at birth. This risk may be further increased by exclusive breastfeeding without vitamin D supplementation, poor maternal nutrition, limited sunlight exposure, darker skin pigmentation, cultural clothing practices, prematurity, low birth weight, and delayed or inadequate complementary feeding. Although breastfeeding is the ideal source of infant nutrition, breast milk contains relatively low vitamin D unless maternal vitamin D status is adequate or supplementation is provided [6]. Therefore, breastfed infants may remain at risk of hypovitaminosis D if preventive supplementation is not ensured. Hypocalcemic seizures due to vitamin D deficiency are especially relevant in developing countries, where nutritional deficiencies, limited awareness, low supplement use, and reduced access to preventive pediatric care may contribute to a higher disease burden [7]. In many infants, seizures may be the first alarming manifestation of an underlying metabolic abnormality. These cases may initially be managed as epilepsy, febrile seizures, or central nervous system infection unless serum calcium and vitamin D status are assessed early. Failure to identify vitamin D deficiency may lead to recurrent seizures, repeated hospital visits, unnecessary anticonvulsant use, costly neuroimaging, lumbar puncture, and prolonged parental anxiety [8].

The clinical presentation of vitamin D deficiency-related hypocalcemic seizures can be variable. Infants may present with generalized tonic-clonic seizures, tonic seizures, focal seizures, jitteriness, irritability, poor feeding, or features of neuromuscular irritability [9]. Classical signs of hypocalcemia such as tetany, Chvostek sign, or Trousseau sign may be absent in many infants. Similarly, radiological evidence of rickets may not always be obvious at the time of seizure presentation. Because of this, biochemical assessment remains essential [10]. Measurement of corrected serum calcium, phosphorus, alkaline phosphatase, magnesium, and serum 25-hydroxyvitamin D can help identify the underlying metabolic cause and guide appropriate treatment. Early diagnosis of vitamin D deficiency in infants with hypocalcemic seizures has major therapeutic and preventive value [11]. Treatment with calcium and vitamin D supplementation can rapidly correct biochemical abnormalities and prevent further seizure episodes [12]. In addition, identifying vitamin D deficiency in an infant should prompt evaluation of maternal vitamin D status, feeding practices, sunlight exposure, and supplementation history. This broader approach can help prevent recurrence in the same infant and reduce risk among future siblings or other high-risk infants [13].

Objective

To evaluate the contribution of vitamin D deficiency to hypocalcemic seizures in infants aged 1 month to 2 years.

METHODOLOGY

This was a prospective observational study derived from the infant seizure cohort conducted in the Department of Pediatrics, Navodaya Medical College Hospital and Research Center, Raichur. A total of 100 infants aged 1 month to 2 years presenting with seizures were initially enrolled in the study. Vitamin D assessment was performed in infants where clinically indicated and where laboratory data were available, particularly among infants diagnosed with hypocalcemia. The study population included infants aged 1 month to 2 years who presented with seizures and were evaluated for hypocalcemia and the possible contribution of hypovitaminosis D. The main analytical subgroup consisted of infants with hypocalcemic seizures. Infants aged 1 month to 2 years presenting with seizures and otherwise developmentally normal were included. Infants with central nervous system infection, congenital brain malformation, and infants whose mothers were receiving drugs or supplements known to affect bone mineral metabolism were excluded from the study. After obtaining informed consent from parents, demographic information, feeding history, seizure details, clinical presentation, and biochemical parameters were recorded using a structured proforma. Special emphasis was placed on biochemical markers related to calcium and vitamin D metabolism. Blood samples were collected and analyzed for serum calcium, corrected calcium, phosphorus, alkaline phosphatase, albumin, magnesium where available, and vitamin D levels where available.

Vitamin D estimation was performed using Abbott Architect i1000SR by chemiluminescence immunoassay. Other biochemical investigations were performed using the ROCHE Integra 400 analyzer. Serum calcium was measured by the

cresolphthalein/end-point reaction method, phosphorus by the phosphomolybdate method, alkaline phosphatase by the rate kinetic method, and albumin by the bromocresol purple method. Hypovitaminosis D was categorized as mild deficiency at 10–20 ng/mL, moderate deficiency at 5–10 ng/mL, and severe deficiency at less than 5 ng/mL. The primary outcome variable was the presence of hypovitaminosis D among infants with hypocalcemic seizures. Secondary biochemical outcomes included serum calcium, corrected calcium, phosphorus, alkaline phosphatase, magnesium, and albumin levels. Data were analyzed using frequencies and percentages for categorical variables. Associations between relevant variables were assessed using the chi-square test or Fisher's exact test where appropriate. A p-value of ≤0.05 was considered statistically significant.

RESULTS

Among the 27 infants with hypocalcemia, vitamin D deficiency was identified as the major contributor to hypocalcemic seizures. The thesis reported that hypocalcemia was mostly attributed to hypovitaminosis D. One infant with refractory seizures/status epilepticus had severe vitamin D deficiency with a vitamin D level of 4.6 ng/mL, while the infant's mother also had markedly low vitamin D level of 5.10 ng/mL.

Table 1. Contribution of vitamin D deficiency among infants with hypocalcemic seizures

Variable	Finding
Total Infants With Seizures	100
Infants With Hypocalcemia	27 (27.0%)
Hypocalcemia Attributed To Hypovitaminosis D	Most Common Cause
Hypomagnesemia	6 (6.0%)
Severe Vitamin D Deficiency Case	Vitamin D = 4.6 Ng/Ml
Maternal Vitamin D In Severe Case	5.10 Ng/Ml

The biochemical profile showed a mean serum calcium level of 8.52 ± 1.713 mg/dL and mean corrected calcium of 8.48 ± 1.637 mg/dL. The mean phosphorus level was 6.11 ± 1.957 mg/dL, alkaline phosphatase was 378.34 ± 233.574 U/L, and vitamin D level among tested infants was 13.25 ± 9.073 ng/mL. The low mean vitamin D level supports the role of hypovitaminosis D as an important underlying factor in hypocalcemic seizures.

Table 2. Biochemical profile related to calcium and vitamin D status

Parameter	N	Minimum	Maximum	Mean ± SD
Calcium (Mg/Dl)	100	3.80	12.10	8.52 ± 1.713
Albumin (G/Dl)	100	1.90	4.60	3.67 ± 0.486
Corrected Calcium	65	3.96	11.20	8.48 ± 1.637
Phosphorus (Mg/Dl)	100	3.10	14.20	6.11 ± 1.957
Alkaline Phosphatase (U/L)	100	104.00	1004.00	378.34 ± 233.574
Magnesium (Mg/Dl)	25	1.00	4.00	2.21 ± 0.570
Vitamin D (Ng/Ml)	29	3.00	45.00	13.25 ± 9.073

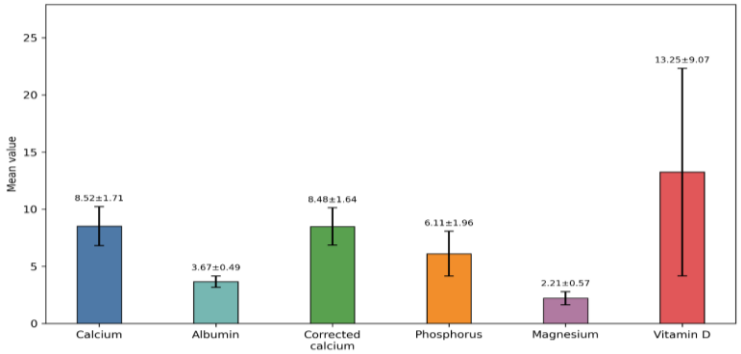

Figure 1: Biochemical Profile Related to Calcium and Vitamin D Status

DISCUSSION

The present study evaluated the contribution of vitamin D deficiency to hypocalcemic seizures among infants aged 1 month to 2 years presenting with seizures. Among 100 infants included in the cohort, 27 (27.0%) were found to have hypocalcemia, and vitamin D deficiency was identified as the major contributor to hypocalcemic seizures. This finding highlights the important role of hypovitaminosis D as a correctable metabolic factor in infantile seizures. Since vitamin D deficiency and hypocalcemia are both treatable, early recognition can prevent recurrent seizures and reduce unnecessary diagnostic and therapeutic interventions. Vitamin D plays a central role in maintaining calcium homeostasis by promoting intestinal calcium absorption and supporting bone mineralization. In infancy, the demand for calcium is high because of rapid skeletal growth. When vitamin D levels are low, intestinal calcium absorption decreases, leading to hypocalcemia and increased neuromuscular excitability. This increased excitability lowers the seizure threshold and may result in acute symptomatic seizures. In the present study, the mean vitamin D level among tested infants was 13.25 ± 9.073 ng/mL, which falls within the deficient or insufficient range, supporting the role of hypovitaminosis D in seizure pathogenesis [14].

The biochemical profile of the study population further supports this association. The mean serum calcium level was 8.52 ± 1.713 mg/dL, while the mean corrected calcium level was 8.48 ± 1.637 mg/dL. Although the mean corrected calcium was slightly above the cutoff for hypocalcemia, a considerable subgroup had values below 8 mg/dL and presented with seizures. The wide range of calcium values, from 3.80 to 12.10 mg/dL, indicates significant biochemical variability among infants with seizures. This reinforces the importance of measuring corrected calcium rather than relying only on clinical features, because hypocalcemia may not be obvious on examination. A notable finding was the documented case of refractory seizures/status epilepticus in an infant with severe vitamin D deficiency, with a vitamin D level of 4.6 ng/mL [15]. The infant's mother also had markedly low vitamin D level of 5.10 ng/mL. This is clinically important because it supports the concept of maternal-infant vitamin D linkage. Infants are born with vitamin D stores that depend heavily on maternal vitamin D status during pregnancy. If the mother is deficient, the infant may begin life with inadequate vitamin D reserves, making the baby more vulnerable to hypocalcemia during early infancy [16].

The role of breastfeeding in this context should be interpreted carefully. Breastfeeding itself is not harmful and remains the best source of nutrition for infants. However, breast milk contains relatively low vitamin D unless the mother has adequate vitamin D status or supplementation is provided. Therefore, exclusively breastfed infants born to vitamin D-deficient mothers may be at increased risk of hypovitaminosis D and hypocalcemic seizures [17]. This finding supports the need for maternal vitamin D screening, antenatal supplementation, and infant vitamin D supplementation according to pediatric recommendations. The findings of this study are clinically meaningful because hypocalcemic seizures due to vitamin D deficiency are preventable. In many infants, seizures may be the first dramatic presentation of an underlying nutritional deficiency [18]. Without calcium and vitamin D testing, such cases may be misdiagnosed as epilepsy, febrile seizures, or CNS infection. This may lead to unnecessary anticonvulsant therapy, neuroimaging, lumbar puncture, longer hospital stay and increased parental anxiety. A simple biochemical workup can identify a reversible cause and guide targeted treatment [19].

From a public health perspective, the study emphasizes the importance of preventive strategies. Maternal vitamin D deficiency should be addressed during pregnancy and lactation. Infants, particularly those who are exclusively breastfed, should receive appropriate vitamin D supplementation [20]. Counseling regarding safe sunlight exposure, maternal nutrition, and regular pediatric follow-up may reduce the risk of vitamin D deficiency-related hypocalcemia. Prevention is especially important in regions where vitamin D deficiency is common due to limited sun exposure, cultural clothing practices, indoor lifestyle, poor dietary intake, or lack of supplementation. There are some limitations. Vitamin D levels were available only in 29 infants, and magnesium was measured in 25 infants, which limits complete biochemical interpretation for the entire cohort. Maternal vitamin D levels were not systematically measured in all cases, so the maternal-infant vitamin D relationship could not be fully analyzed. Dietary intake, sunlight exposure, supplementation history, and radiological evidence of rickets were not fully detailed in the results provided. Additionally, the study was

conducted at a single center with 100 infants, so findings may not be generalizable to all populations.

CONCLUSION

Vitamin D deficiency was identified as a major contributor to hypocalcemic seizures among infants aged 1 month to 2 years. In this cohort, 27% of infants presenting with seizures had hypocalcemia, and the low mean vitamin D level among tested infants supports the role of hypovitaminosis D in disturbed calcium homeostasis. The documented case of severe vitamin D deficiency in both infant and mother further highlights the importance of maternal vitamin D status in infant calcium balance. These findings suggest that infants presenting with seizures should be evaluated for corrected serum calcium and, where indicated, vitamin D status, especially in settings where vitamin D deficiency is common.

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