SCIE DOI: 10.14744/scie.2022.67299
South. Clin. Ist. Euras. 2022;33(4):429-434

Original Article

Evaluation of Neurological Prognosis in Children with Hypocalcemic Seizures Due to Vitamin D Deficiency

Gül Demet Kaya Özcora,[1] Elif Söbü,[2] Türkan Uygur Şahin[3]

[1]Department of Physical Therapy and Rehabilitation, Hasan Kalyoncu University Faculty of Health Sciences, Gaziantep, Türkiye
[2]Department of Pediatrics, Kartal Dr. Lütfi Kırdar City Hospital, İstanbul, Türkiye
[3]Department of Pediatrics, Bezmialem University Faculty of Medicine, İstanbul, Türkiye

Submitted: 15.05.2022
Revised: 07.06.2022
Accepted: 08.06.2022

Correspondence:
Gül Demet Kaya Özcora,
Hasan Kalyoncu Üniversitesi,
Sağlık Bilimleri Fakültesi,
Gaziantep, Türkiye

E-mail: guldemetkaya@hotmail.com

Keywords: Convulsion; hypocalcemia; neurological development.

ABSTRACT

Objective: This study aimed to evaluate the neurologic findings of patients who had hypocalcemic seizures due to vitamin D deficiency during a 1 year period.

Methods: Thirty-two patients aged between 6 days and 24 months who were followed up for hypocalcemic seizures between July 2017 and July 2018 were included in the study. Gross motor, fine motor, language, and social developmental levels were evaluated using the Denver developmental test at the time of diagnosis and the 6th month.

Results: Seventy-two percentages of the patients were male (n=23) and 28% (n=9) were female. Corrected serum calcium level averaged 6.03±1.06 mg/dL, and phosphorus was 5.2±1.72 mg/dL, alkaline phosphatase was 575.2±405.8 U/L, parathormone was 231.4±123.8 ng/mL, and 25 OH-vitamin D levels were found as 9.6±8.5 ng/mL. When the vitamin D levels of the mothers were examined, the average was 7.16±2.62 ng/mL. The 6-month difference in both gross and fine motor development was found to be statistically significant ($p<0.001$ and $p<0.001$, respectively). The 6-month difference in the evaluation of social development and language development was not statistically significant ($p=0.083$ and $p=0.180$, respectively). The 6-month difference in fine and gross motor development over the patients was considered to be statistically significant ($p<0.001$).

Conclusion: Psychomotor retardation is also observed in infants with vitamin D deficiency, because vitamin D is a neurohormone that has an important role in calcium metabolism, which plays a role in neurotransmitter production, release, reuptake, and in stages of brain cell proliferation, differentiation, and development.

INTRODUCTION

Vitamin D deficiency develops in mothers and infants due to long-term breastfeeding, low exposure to sunlight, the use of sunscreen creams in developed countries, and malnutrition, frequent pregnancy, and wearing covering clothing year-round are the prevalent causes in developing countries.[1] Vitamin D deficiency is one of the most important causes of childhood hypocalcemia and its frequency in our country has been reported as 1.6–19%.[2] The blood calcium level is approximately 1% of the total body calcium, and a serum calcium level below 8.5 mg/dL in the absence of hypoalbuminemia or acidosis is defined as hypocalcemia.[1]

Calcium plays essential roles in normal cell function, neural conduction, membrane stabilization, skeletal structure, blood coagulation, and the intracellular conduction system. Psychomotor developmental retardation can also be seen in children with vitamin D deficiency and hypocalcemia due to the critical role of calcium in the production, release, and re-uptake of neurotransmitters in neuronal proliferation and differentiation process.[3,4] Calcium takes part in almost every stage of neuromuscular functions. Hypocalcemia increases neuronal excitability and may result in neurologic manifestations in the form of tetany, irritability, seizures, and delirium.[1–5]

Although all newborns have been given vitamin D prophylaxis within the scope of the Ministry of Health preventive health services since 2005,[6,7] we still see hypocalcemic convulsions due to vitamin D deficiency in infants and newborns.

In this study, we wanted to draw attention to maternal vitamin D deficiency, the necessity of maternal prophylaxis

programs, and to evaluate the effect of vitamin D on the neurodevelopmental process.

MATERIALS AND METHODS

Thirty-two patients aged 0–24 months who were followed up for hypocalcemic seizures due to vitamin D deficiency between July 2017 and July 2018 were included in the study. Patients with chronic diseases, hepatic or renal dysfunction, a history of febrile or afebrile convulsions, prematurity, and using medication that could affect vitamin D metabolism were excluded from the study.

The demographic data of the patients; nutritional status; history of vitamin D prophylaxis; maternal vitamin D intake during pregnancy; seasonal distribution of seizures; recurrence of seizures; presence of accompanying infections; serum calcium, phosphorus, parathormone, alkaline phosphatase (ALP), and vitamin D levels of the patients; maternal vitamin D levels; and electrencephalogram (EEG) results were evaluated. Vitamin D status was classified as sufficiency >20 ng/mL, insufficiency 12–20 ng/mL, and deficiency <12 ng/mL according to the Global Consensus Recommendations on the Prevention and Management of Nutritional Rickets.[7]

The Denver II developmental screening (D-II-DST) test, which was adapted for Turkish children in 2009 and aims to evaluate the general development of children aged 0–6 years, was performed at the time of diagnosis and repeated 6 months after the first seizure. Language, personal-social development, and the fine and gross motor development of the patients were evaluated as abnormal, suspicious, or normal. When evaluating the test results, if the results showed an age-appropriate development in four areas or if it was suspicious in one area, the developmental test result was considered "normal," one of four areas as "abnormal," or ≥two suspicious in four areas or 1 is abnormal with one area is suspicious considered "suspicious." If ≥2 of four areas were abnormal, development was considered "abnormal."

The SPSS version 21.0 (SPSS, Inc., Chicago, IL) was used for statistical analysis. The variables were investigated using visual (histogram and probability plots) and analytic methods (Kolmogorov–Smirnov/Shapiro–Wilk's test) to determine whether or not they are normally distributed. Descriptive analyses were presented using proportions, medians, minimum (min), and maximum (max) values where appropriate. Differences in proportions between groups were evaluated by the Chi-square test or Fisher's exact test where appropriate. Mann–Whitney U-test was used to compare the non-normally distributed continuous data between two groups. The homogeneity test was used to compare dependent and multi-category variables. Categorial variables are defined as number (n) and percentage (%) values. P<0.05 was considered as significant.

Permission was obtained from the Medical Faculty Clinical/Human Research Ethics Committee, and Helsinki Declaration rules were followed to conduct this study. The data presented in this study was collected retrospectively from medical records.

RESULTS

Thirty-two patients who were diagnosed as having hypocalcemic convulsions between July 2017 and July 2018 were included in the study. Seventy-two percentages (n=23) of the patients were male and 28% (n=9) were female. The average age was 7.2±4.1 months (range, 6 days to 24 months). The most frequent admissions were in winter with 44% (14/32), followed by spring with 34% (11/32), autumn with 13% (4/32), and summer with 9% (3/32). All patients were hospitalized and treated, and the mean hospitalization duration was 6.25±2.4 days.

When the nutritional status of patients was evaluated, five patients were receiving only breast milk, nine patients were receiving breast milk and formula, and 18 patients were receiving formula and supplementary food.

At the time of hospital admission, 28% (n=9) of the patients had lower and/or upper respiratory tract infection, and 6% (n=2) had acute gastroenteritis.

The kidney and liver functions of the patients were normal. When the laboratory data were examined, the average corrected serum calcium level was 6.03±1.06 mg/dL, the level of phosphorus was 5.2±1.72 mg/dL, ALP 575.2±405.8 IU/L, parathormone 231.4±123.8 ng/mL, and the level of 25-OH vitamin D was 9.6±8.5 ng/mL. Eleven (34%) patients had not received any vitamin D prophylaxis. Although the prophylaxis intake of 21 patients was not regular, their average intake period was 43.8±20.6 days.

Table 1. Gross motor development of patients at the 6th month after the hypocalcemic seizure

Gross motor development	6th month			
	Abnormal	Suspicious	Normal	Total
Abnormal, n (%)	2 (6.3)	6 (18.8)	4 (12.5)	12 (37.5)
Suspicious, n (%)	0 (0.0)	0 (0.0)	8 (25)	8 (25)
Baseline, n (%				
Normal	0 (0.0)	0 (0.0)	12 (37.5)	12 (37.5)
Total	2 (6.3)	6 (18.8)	24 (75.0)	32 (100.0)
p-value		<0.001		

None of the mothers received vitamin D supplements during pregnancy. The vitamin D levels of the mothers were 7.16±2.62 ng/mL.

Fourteen (43.75%) of our patients were breastfed, and the average duration of breast milk intake was 8.2±7.4 months.

There was a family history of seizures in two patients. A sibling was reported to have epilepsy in one of these patients, and in the other patient, a sibling had a history of hypocalcemic seizures due to vitamin D deficiency.

Generalized tonic-clonic seizures were observed in 31 patients and hypomotor seizures with lip licking and swallowing in one patient. Two patients with status epilepticus were admitted to the emergency department. Resuscitation was performed on one patient with status epilepticus due to cardiopulmonary arrest. An EEG was performed in all patients within the first 48 h, no pathology was detected in the EEG of 31 patients, but generalised delta slowing was observed in the EEG of the patient who needed resuscitation.

Intravenous calcium gluconate treatment was given to all patients until the blood calcium level returned to normal, after which oral treatment was switched to 50–75 mg/kg/day calcium lactate. Vitamin D treatment was initiated 2000 IU/day for patients aged <1 year and 3000 IU/day for patients aged 1–2 years. Antiepileptic treatment was initiated in three patients with status epilepticus and hypomotor seizure. Benzodiazepine infusion and levetiracetam were administered to patients with status epilepticus and iv levetiracetam treatment was initiated for the patient with hypomotor seizures, having had similar seizures twice on the same day. Seizure recurrence was not observed after the treatment. The patient who had hypomotor seizures had a family history of epilepsy and had two seizures on the same day, so the antiepileptic treatment was reduced and stopped in the 1st-month outpatient follow-up, and no seizure recurrence was observed. The treatment of the patient with arrest, who presented with status epilepticus, was extended up to 3 months due to the continuing irregularity of the background activity and the drug was reduced and discontinued at the 3rd month when EEG was found to be normal. The treatment of the other patient was stopped by tapering at the 1st-month follow-up. Neurologic sequelae and epilepsy development were not observed in any patients after the hypocalcemic convulsions.

The D-II-DST test was performed at the first presentation (postictal 24–48 h) and 6 months after the treatment. Results related to language, personal-social development, and fine and gross motor development were classified as normal, suspicious, or abnormal.

The 6-month difference in gross motor development of the patients was statistically significant (p<0.001). Of the 12 patients whose gross motor development was abnormal at baseline, two were abnormal, six were considered suspicious, and four were considered normal by the end of the 6th month. All of the eight patients whose gross motor development was suspicious at baseline were considered normal by the 6th month (Table 1).

The 6-month difference in fine motor development over the patients was considered to be statistically significant (p<0.001). Of the 20 patients, whose fine motor development was abnormal at baseline, six were seen as abnormal, six were suspicious, and eight were evaluated as normal by the 6th month. All five patients whose fine motor develop-

Table 2. Fine motor development of patients at the 6th month after the hypocalcemic seizure

Fine motor development	6th month			
	Abnormal	Suspicious	Normal	Total
Abnormal, n (%)	6 (18.8)	6 (18.8)	8 (25)	20 (62.5)
Suspicious, n (%)	0 (0.0)	0 (0.0)	5 (15.6)	5 (15.6)
Baseline, n (%)				
Normal	0 (0.0)	0 (0.0)	7 (21.9)	7 (21.9)
Total	6 (18.8)	6 (18.8)	20 (62.5)	32 (100.0)
p-value		<0.001		

Table 3. Social development of patients at the 6th month after the hypocalcemic seizure

Social development	6th month			
	Abnormal	Suspicious	Normal	Total
Abnormal, n (%)	10 (31.3)	2 (6.3)	0 (0.0)	12 (37.5)
Suspicious, n (%)	0 (0.0)	1 (3.1)	1 (3.1)	2 (6.3)
Baseline, n (%)				
Normal	0 (0.0)	0 (0.0)	18 (56.3)	18 (56.3)
Total	10 (31.3)	3 (9.4)	19 (59.4)	32 (100.0)
p-value		0.083		

Table 4. Language development of patients at the 6th month after the hypocalcemic seizure

Language development	6th month			
	Abnormal	Suspicious	Normal	Total
Abnormal, n (%)	8 (25.0)	1 (3.1)	0 (0.0)	9 (28.1)
Suspicious, n (%	0 (0.0)	2 (6.3)	0 (0.0)	2 (6.3)
Baseline, n (%				
Normal	0 (0.0)	4 (12.5)	17 (53.1)	21 (65.6)
Total	8 (25.0)	7 (21.9)	17 (53.1)	32 (100.0)
p-value		0.180		

ment was suspicious at baseline were accepted as normal by the 6th month (Table 2).

The 6-month difference in social development assessment of the patients was not statistically significant (p=0.083) (Table 3).

In the assessment of language development, the 6-month difference over the patients was not statistically significant (p=0.180) (Table 4).

DISCUSSION

Hypocalcemia constitutes a major cause of infantile seizures in developing countries. The incidence of hypocalcemic seizures due to vitamin D deficiency is highest in children aged under 2 years. Infants are susceptible to vitamin D deficiency due to the rapid skeletal growth rate. It has been reported that 12–25% of symptomatic patients with vitamin D deficiency present with convulsions, and this rate can reach up to 78.6% in infants aged under 3 months.[2] It is thought that the presence of seizures in patients with hypocalcemia may be due to low ionized calcium concentrations in the cerebrospinal fluid.[8] Hypocalcemic convulsions should be kept in mind, especially in infants aged <6 months, who present with a first afebrile convulsion due to its preventable etiology and recognizability to prevent unnecessary investigations.[1] The average age of our patients was 7.2±4.1 months. There were 23 boys (72%) and 9 girls (28%). It was male-dominated, as in the previous studies.[1–5]

The nutritional status of mothers is important, because the vitamin D storage of the baby in the early infantile period depends on transplacental transition in the last trimester and breast milk during breastfeeding. In early infantile D hypovitaminosis, the main factor is maternal vitamin D deficiency, and a study conducted in our country found 46–80% vitamin D hypovitaminosis in pregnant and breastfeeding mothers.[9] Prenatal or postnatal vitamin D levels were not measured or checked in any of the mothers. Despite the recommendations issued by the Turkish Ministry of Health in 2011 recommending that all pregnant women should be given 1200 IU of vitamin D, none of the mothers used vitamin D during pregnancy in our study group. The average vitamin D levels of the mothers were 7.16±2.62 ng/mL. We thought that their low vitamin level was due to malnutrition, frequent and high birth rate, local clothing styles (only with their faces, hands, and feet exposed), and dietary factors (eating eggplant, hot peppers, and meat frequently).

It has been reported that, if the mother does not receive additional nutritional support, long-term breastfeeding may lead to vitamin D deficiency due to the low vitamin D content of breast milk.[9–11] With the "Free Vitamin D for 1 million children up to the age of 1" campaign initiated by the Ministry of Health in 2005, 400 IU of vitamin D per day is recommended to all newborns from the first day of life, regardless of their diet.[7]

The number of our patients who were breastfed was 14 (43.75%) and the average duration of breast milk intake was 8.2±7.4 months. Eleven (34%) patients had not received any vitamin D prophylaxis. Although the prophylaxis intake of 21 patients was not regular, the average intake period was 43.8±20.6 days. In our study, the rate and duration of breastfeeding were low and there was an early switch to supplementary food and malnutrition practices (giving bread soaked in tea/instant biscuits/giving pot liquor). All of the families had a low socioeconomic level.

Unlike rickets seen in older ages, in vitamin D deficiency in neonates and early infants, phosphorus levels may often be normal or high, parathyroid hormone (PTH), and ALP may be normal, and radiologic clinical findings of rickets may not be observed due to inadequate PTH release and often presents with hypocalcemic seizures. The phosphorus levels in our study were 5.2±1.72 mg/dL, ALP 575.2±405.8 U/L, and PTH 231.4±123.8 ng/mL. The phosphorus level was normal, PTH and ALP levels were elevated in our patients, and radiologic findings of rickets were observed accordingly.

Seizures due to hypocalcemia are often generalized tonic seizures followed by generalized tonic-clonic seizures. Status epilepticus is rare. Absence seizures, focal seizures, and epilepsia partialis continua have also been described.[12–14]

In our study, except for one patient, generalized tonic or tonic-clonic seizures were observed (97%), two patients had status epilepticus, and one had cardiorespiratory arrest while being referred for having status epilepticus. In our study, we attribute the high rate of status epilepticus[8,11–13] to the failure to intervene in the seizure swiftly

and effectively, or to not being hospitalized despite the occurrence of short-term seizures at home.

It has been found that vitamin D deficiency predisposes to respiratory infections and lowers the seizure threshold during the infection period.[1–5] In our study, 34.3% of the patients had lower or upper respiratory tract infection, acute gastroenteritis, and accompanying hypocalcemic convulsion.

Psychomotor retardation is also observed in infants with vitamin D deficiency, because vitamin D is a neurohormone with an important role in calcium metabolism and functions in neurotransmitter production, release, and reuptake, and in stages of brain cell proliferation, differentiation, and development.[3,4]

The D-II-DST was performed on our patients at the time of diagnosis and the 6th-month follow-ups; during this period, no additional nutritional support was given except for vitamin D. Of the 12 patients whose gross motor development was abnormal at baseline, six were considered suspicious and four were found normal at the 6th month. All eight patients whose gross motor development was suspicious at baseline were evaluated as normal at the 6th month. The 6-month difference in gross motor development was statistically significant ($p<0.001$). Of the 20 patients, whose fine motor development was abnormal at baseline, six were suspicious and eight were normal at the 6th month. All five patients whose fine motor development was suspicious at baseline were seen to be normal at the 6th month. The 6-month difference in fine motor development was statistically significant ($p<0.001$). The 6-month difference in the evaluation of social development and language development was not statistically significant ($p=0.083$, $p=0.180$). Interhemispheric connections increase and sensory-motor skills develop between the ages of 1 and 2 years, and significant dendritic branches emerge in the speech field after the age of 2 years.[3,4] Because our patients were aged under 2 years and the rapidly developing motor area was affected during this period, gross and fine motor development was affected, and social and language development was observed not to have been affected.

Although there are many studies highlighting vitamin D deficiency in women during breastfeeding and pregnancy[2–5,10,11] and it is known that maternal vitamin D deficiency is the main factor in neonatal and infantile vitamin D deficiency, it was observed that none of the mothers in our study took vitamin D during pregnancy.

The importance of vitamin D supplementation of all infants beginning during the 1st days of life and the significance of prophylaxis should be explained to families. Obstetricians and pediatricians have a great responsibility to ensure that all pregnant women and newborns receive regular vitamin D prophylaxis.

Acknowledgement

No specific funding was received from any funding bodies in the public, commercial, or not for-profit sectors to carry out the work described in this manuscript. The authors have declared no conflicts of interest.

Ethics Committee Approval

This study approved by the Harran University Clinical Research Ethics Committee (Date: 18.11.2019, Decision No: HRU.19.05.23).

Informed Consent

Externally peer-reviewed.

Peer-review

Internally peer-reviewed.

Authorship Contributions

Concept: E.S., G.D.K.Ö.; Design: E.S., G.D.K.Ö.; Supervision: T.U.Ş. G.D.K.Ö.; Fundings: E.S., G.D.K.Ö.; Materials: T.U.Ş., E.S., G.D.K.Ö.; Data: E.S., G.D.K.Ö.; Analysis: E.S., G.D.K.Ö.; Literature search: E.S., G.D.K.Ö.; Writing: E.S., G.D.K.Ö.; Critical revision: T.U.Ş., E.S.

Conflict of Interest

None declared.

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D Vitamini Eksikliğine Bağlı Hipokalsemik Nöbetleri Olan Çocuklarda Nörolojik Prognozun Değerlendirilmesi

Amaç: Çalışmamızda hipokalsemik nöbet tanısı alan süt çocuklarının bir yıllık izlemdeki nörolojik bulgularının değerlendirilmesi amaçlandı.

Gereç ve Yöntem: Çalışmaya Temmuz 2017–Temmuz 2018 tarihleri arasında hipokalsemik nöbet tanısıyla izlenen, yaşları 6 gün ile 24 ay arasında 32 olgu dahil edildi. Kaba motor, ince motor, dil ve sosyal gelişim düzeyleri tanı anında ve altıncı ayda Denver gelişim testi ile değerlendirildi.

Bulgular: Olguların %72'si erkek (n=23) ve %28'i (n=9) kızdı. Düzeltilmiş serum kalsiyum seviyesi ortalama 6.03±1.06 mg/dL, fosfor 5.2±1.72 mg/dL, alkalen fosfataz 575.2±405.8 U/L; parathormon 231.4±123.8 ng/ml ve 25-OH vitamin D düzeyi 9.6±8.5 ng/ml olarak bulundu. Annelerin D vitamini düzeyleri incelendiğinde ortalama 7.16±2.62 ng/ml saptandı. Kaba motor gelişimdeki altı aylık fark istatistiksel olarak anlamlı bulundu ($p<0.001$), ince motor gelişimdeki altı aylık fark istatistiksel olarak anlamlıydı ($p <0.001$). Sosyal gelişim ve dil gelişiminin değerlendirilmesindeki altı aylık fark istatistiksel olarak anlamlı değildi (sırasıyla $p=0.083$ ve $p=0.180$). Altı aylık süreçte hastaların ince ve kaba motor gelişimdeki fark istatistiksel olarak anlamlı kabul edildi ($p<0.001$).

Sonuç: D vitamini bir nörohormondur, nörotransmiter üretimi, salımı ve geri alımında rol oynayan ve beyin hücresi çoğalması, farklılaşması ve gelişimi aşamalarında önemli rol alan kalsiyum metabolizmasının önemli bir düzenleyicisi olduğu için eksikliğinde psikomotor gerilikte görülebilmektedir.

Anahtar Sözcükler: Konvülziyon; hipokalsemi; nörolojik gelişim.