Hypocalcemic Convulsion in a Six-Year-Old Child with Vitamin D Deficiency

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Abstract

Nutritional rickets occurs more commonly in breastfed infants with rapid growth and limited exposure to sunlight. Hypocalcemic convulsions secondary to vitamin D deficiency occur largely in patients with rapid growth rates, such as children younger than 1 year and adolescents. Vitamin D deficiency seems to be an unrecognized and prevalent problem in school childhood. Whereas infants generally exhibit bony deformities, most school-aged children are asymptomatic. In this case, we present hypocalcemic convulsion in a 6-year-old boy with nutritional vitamin D deficiency in order to emphasize that hypocalcemia secondary to vitamin D deficiency can lead to convulsion in other children, as well as infants and adolescents. *(JAEM 2014; 13: 206-8)*

Key words: Hypocalcemic convulsion, school childhood, vitamin D deficiency

Introduction

In addition to being the major symptom of epilepsy, seizures can be the result of numerous transient conditions that cause neuronal excitation, such as fever, electrolyte disturbances, infections of the central nervous system, bleeding, ischemia, and head trauma (1). Hypocalcemia is one of the electrolyte disturbances that can cause seizures. Having a stable extracellular ionized calcium concentration is critical for normal brain cell function, and vitamin D and parathyroid hormone (PTH) play a central role in maintaining a stable extracellular ionized calcium concentration (2).

Vitamin D deficiency continues to be a public health problem that is prevalent throughout the world, especially in developing countries. Having a deficiency in vitamin D decreases the absorption of calcium from the intestine and can lead to hypocalcemia. Hypocalcemia that results from vitamin D deficiency is most prevalent during periods of rapid growth, such as in infancy and adolescence (3). We report this case in order to emphasize that hypocalcemia that is due to nutritional vitamin D deficiency can cause seizures in children who are not in the rapid-growth periods of infancy and adolescence.

Case Presentation

A boy, aged 6 years and 7 months, was brought to the emergency department after having had symptoms of a seizure at home. The seizure was described as a fixed-point gaze and rhythmic contractions in both his arms and his legs. His mother reported that the seizure had lasted about 5 minutes and that he had recovered spontaneously. On initial evaluation in the emergency department, the patient was found to be conscious, and his pupils were isochoric, but his skin and mucous membranes were pale. He had no neck stiffness, but there were contractions in both of his hands, similar to midwife hands. His mother also reported that he had an ongoing low-grade fever and malaise for a few days but had not received any treatment for this. He had no significant medical history of seizures, trauma, drug allergy, or any other illness. The patient had been a full-term baby, had been born by normal vaginal delivery, and had been breastfed for about 1.5 years. He was able to keep his head up when he was 1 month old; he sat, acquired his first tooth at 6 months, and walked at 1 year. We understood from details about his diet that he did not consume milk or milk products and had been drinking too much cola. In general, the patient did not move a lot and did not like to spend time outdoors. Although he lived in sunny Adiyaman, he had not been exposed to adequate amounts of sunlight. Also, there were no individuals in his immediate family who had seizures.

A physical examination showed that there was redness in his throat. However, his tympanic membranes and the sounds of his breathing and his heart were normal. The results of an abdominal examination, as well as an examination of his head, wrists, and legs, were all normal, and there was no organomegaly. There was no ra-

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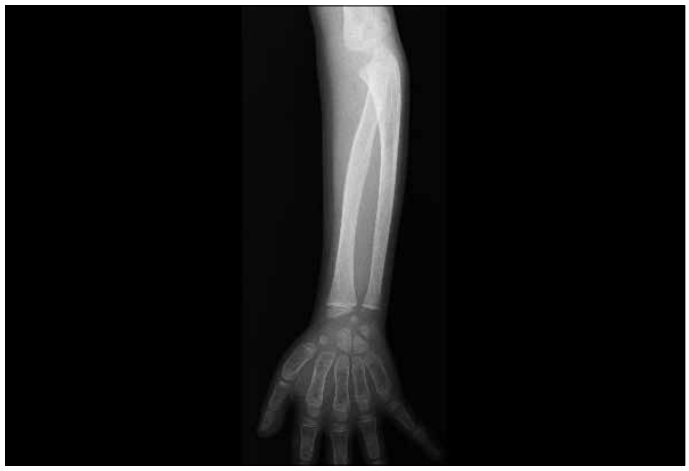

Figure 1. A common demineralization in the osteoid matrix

chitic rosary on his chest. His neurological examination was negative for Chvostek and Trousseau signs. The patient's temperature was 36.8°C, his blood pressure was 96/62 mm Hg, and his blood glucose was 96 mg/dL. He measured 114 cm in height (10^{th}-25^{th} percentile), and his body weight was 18 kg (3^{rd} percentile). Laboratory evaluation revealed the following: leukocytes 14,500/mm^3, hemoglobin 10.7 mg/dL, hematocrit 31.3%, platelets 430,000/mm^3, C-reactive protein 2.3 mg/dL, calcium 5.7 mg/dL (8.7-10.6), phosphorus 4.1 mg/dL (2.5 to 4.3), albumin 4.0 mg/dL, magnesium 1.5 mg/dL (1.5-2.3), alkaline phosphatase 1120 U/L (100-390), parathormone 329 pg/dL (15-65), and 25-OH vitamin D level 18.4 pg/mL (21-40). The tissue transglutaminase IgA and anti-endomysium IgA antibodies were negative. Other biochemical parameters were within normal limits. There was sinus rhythm on his electrocardiogram, and the QTc length was calculated as 0.41 seconds. Left hand-wrist radiographs revealed a common demineralization in the osteoid matrix (Figure 1). Based on these findings, nutritional vitamin D deficiency was diagnosed. An electroencephalogram (EEG) for differentiation from epileptic seizures was normal.

An intravenous infusion of calcium gluconate was started, and intravenous calcium therapy was continued until the patient's serum calcium levels had returned to normal limits (48 hours). He also received a single dose of 300,000 units of vitamin D, administered intramuscularly, and was treated intravenously with ampicillin-sulbactam for upper respiratory tract infections. After his symptoms had improved and his calcium levels had returned to normal, the patient was discharged with oral 400 units/day of vitamin D and 50 mg/kg/day of calcium lactate. One month later, the patient's vitamin D level was 28.1 pg/mL, PTH was 60 pg/dL, alkaline phosphatase was 580 U/L, and calcium was 9.1 mg/dL. His serum alkaline phosphatase level (274 U/L) returned to normal after 2 months of continued vitamin D supplementation.

Discussion

Vitamin D deficiency decreases calcium absorption in the intestine. In conditions of vitamin D deficiency, low ionized calcium levels stimulate PTH secretion, which increases calcium and phosphorus release from the bone to maintain normal levels of serum calcium. Higher PTH levels increase calcium reabsorption in the renal tubules and also cause a loss of phosphorus in the urine. Therefore, reduced levels of phosphorus and calcium result in decreased bone mineralization (3). The most common causes of nutritional vitamin D deficiency are prolonged breastfeeding, inadequate consumption of milk products, and avoidance of sun exposure (2). Our patient did not consume milk or dairy products, and he had not been exposed to enough sunlight. Furthermore, he had developed a habit of consuming a large number of soft drinks, which disrupts the absorption of calcium.

Vitamin D deficiency continues to be a common problem in school-aged childhood (52%), even in children in Middle Eastern countries, where they have good exposure to sunlight (4). School-based studies suggest that a significant number of girls may have subclinical vitamin D deficiency (5). The clinical presentation of rickets and osteomalacia in this age group differs from the presentation in infants who are under 2 years of age. While infants generally exhibit bony deformities, most school-aged children are asymptomatic. Radiographs in children of this age group may not show the classic radiological changes that are seen with rickets or osteomalacia (6). Ladhani et al. have reported that hypocalcemic symptoms that are due to vitamin D deficiency occur exclusively during periods of rapid growth, such as in children younger than 3 years or older than 10 years (7). In our patient, although the cause of the hypocalcemic seizure was vitamin D deficiency, there were no classic radiological or clinical findings of rickets, with the exception of decreased bone density.

Hypocalcemic convulsions, tetany, and laryngospasm are the most severe complications of nutritional rickets. Schnadower et al. reported that secondary bilateral femoral fractures occurred after hypocalcemic seizures in an adolescent with nutritional vitamin D deficiency (8). Hoecker et al. also reported that severe hypocalcemia in an infant with vitamin D deficiency probably resulted in a reduced seizure threshold and predisposed him to multiple recurrences during febrile illnesses (9). Electrolyte abnormalities, such as hypocalcemia, should be considered when investigating the first seizures in non-epileptic children, especially in patients with prolonged irritability and increased muscle tone. The differential diagnosis of hypocalcemia includes vitamin D deficiency, hypomagnesemia, hypophosphatemic rickets, vitamin D-dependent rickets, malabsorption, and renal and hepatic failure (10). The diagnosis of nutritional vitamin D deficiency is made when low calcium levels are accompanied by increased alkaline phosphatase, increased PTH, and decreased vitamin D levels. In our case, nutritional rickets was confirmed with the very low serum calcium, increased PTH, and decreased vitamin D levels.

Hypocalcemic seizures should be treated with intravenous calcium. Calcium gluconate is preferred over calcium chloride, because calcium gluconate is less irritating and less likely to cause tissue necrosis if extravasation occurs. In the treatment of vitamin D deficiency, high-dose vitamin D (150,000-300,000 units) should first be initiated intramuscularly and then continued with calcium and vitamin D taken orally (2).

To conclude, hypocalcemia with vitamin D deficiency should be considered a reason for first seizures in school-aged children. We have emphasized that symptomatic vitamin D deficiency can be seen, even in children living in the sunny southeast region of our country. Although symptomatic rickets is rarely found in school-aged children, pediatricians and emergency specialists should be aware of this type of vitamin D deficiency, especially in children of this age group.

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