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AN UNUSUAL CASE OF HYPOCALCEMIC SEIZURES

Neurology

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ABSTRACT

*V*itamin D has an important role to play in skeletal and extraskeletal health. It has been estimated that 1 billion people worldwide have Vit D deficiency or insufficiency[1]. Vitamin D deficiency prevails in epidemic proportions all over the Indian subcontinent, with a prevalence of 70%–100% in the general population. Vitamin D deficiency can lead to serious consequences like hypocalcemic seizures and increased risk of respiratory tract infections in neonates and infants and mental retardation[2]. Patients may present with hypocalcemic seizures even in the absence of subtle hypocalcemic signs3. The following is a rare case of first seizure as a manifestation of hypocalcemic secondary to severe vitamin deficiency in a 14yo patient with mental retardation. All treatable metabolic conditions should be excluded at first before commencing treatment with anticonvulsants as this will prevent patients from burdensome polytherapy and related side effects.

KEYWORDS

Introduction

Calcium is essential for both neurotransmitter release and muscle contraction. Given these important physiological processes, it seems reasonable to assume that hypocalcemia may lead to reduced neuromuscular excitability. Counterintuitively, however, clinical observation has frequently documented hypocalcemia's role in induction of seizures .9

Hypovitaminosis D leading to hypocalcemia is an important treatable cause of recurrent seizures. Even though it is not an uncommon condition, primary hypoparathyroidism presenting for the first time as seizures in adulthood is quite infrequent. Patients may present with hypocalcemic seizures even in the absence of subtle hypocalcemic signs inclusive of tetany, Chvostek's sign or carpopedal spasms. As this is an entirely treatable condition, a high index of suspicion for hypovitaminosis D with hypocalcemic seizures should be maintained even in otherwise asymptomatic adults.

clinical scenario

A 14 year old male, was presented to the emergency facility in an unconscious condition. He was a known case of severe mental retardation with IQ of 35 and a known case of seizure disorder on tablet eptoin 5mg/kg and tablet levitericitam since age of 3.He recovered during the hospital stay .The seizure frequency had increased considerably in the last year, and he would have at least 4-5 episodes in a month, thereby creating a considerable toll on his personal life. He had been evaluated with an MRI brain scan and an EEG at the onset of symptoms 6 years earlier and both were reported to be normal. General physical examination was relatively normal. There was no carpopedal spasm or any other signs of tetany like Chvostek's or Trousseau's sign. Investigations revealed normal hemoglobin and glucose level with normal sodium and potassium levels. TLC and DLC levels were also normal. He was found to have a serum calcium level of 3.6 mg% serum 25(OH) vitamin D levels of 13ng/ml(<20ng/ml).his ionized calcium level was 0.8 (1.16-1.32) NCCT head scan was done which waas also normal. A 2D ECHO study was performed, and showed normal results. Then he was started on calcium and vitamin D supplements.. tablet eptoin was stopped and continued on tablet levitericitam.During his hospital stay he did not have any other seizure events. On follow up his calcium level raised to 7.5 after 1 week and ionized calcium became 1.25. after that he has been on regular follow up.. his serum calcium became normal and he seizure free. His levitericitam is being tapered off.

Discussion

In acute and/or severe symptomatic hypocalcemia there is a predominance of neuromuscular, neuropsychiatric, and cardiovascular abnormalities. There is an increase in neuromuscular excitability, latent or evident, with sensory and motor disruption. Perioral or extremity paresthesia, cramps, myalgia, and muscular weakness are mild to moderate symptoms. Neuropsychiatric manifestations include irritability, anxiety, psychosis, hallucinations, dementia, depression, mental confusion, and extrapyramidal abnormalities. Increased intracranial pressure, papilledema, and convulsions can also be present, and must be differentiated from severe tetany muscular spasms4,5. Typical clinical signs of neuromuscular irritability associated with latent tetany include hyperreflexia and Chvostek's and Trousseau's signs, respectively. Severe hypocalcemia may result in bradycardia or ventricular arrhythmias, cardiovascular collapse, and hypotension that is non-responsive to fluids and vasopressors3.

A decrease in myocardial contractility occurs, as well as a typical electrocardiographic abnormality, which is the rate-corrected QT interval (QTc) prolongation. Patients with chronic hypocalcemia may or may not have symptoms of discreet neuromuscular irritation, even with markedly low calcium levels. Asymptomatic cases may be detected by chance, by the dosage of calcium in routine exams, during periods of greater calcium demand (i.e. gestation, lactation, menstrual cycle and states of alkalosis), or during the use of hypocalcemic drugs (i.e. bisphosphonates)6.

Significant cognitive deficits, neuropsychiatric abnormalities, and extrapyramidal symptoms that resemble Parkinson's disease or chorea are associated with the calcification of basal ganglia, which occurs in all forms of chronic hypocalcemia and may be detected with greater sensibility using computerized tomography7. Other findings of chronic hypocalcemia include sub-capsular cataracts, an increase in bone mineral density (BMD), and greater susceptibility to dystonic reactions induced by phenothiazines4.

Differential diagnosis of hypocalcemia will depend largely upon PTH , vitamin D and phosphorus levels, evaluated along with other clinical and laboratory data (Table 1). Cases presenting hypophosphatemia should include differential diagnosis of vitamin D, while cases associated with hyperphosphatemia are determined according to PTH levels.

Laboratory measurements present hypocalcemia, hypophosphatemia. Generally, levels of 1.25(OH) 2D are low and the alkaline phosphatase level is high. In the majority of cases, hypovitaminosis D is sporadic, but there are familial cases in which transmission may be autosomic recessive, dominant, or X-linked.

Management of acute or severe symptomatic hypocalcemia must be made with intravenous calcium, with the goal of interrupting symptoms, preventing laryngeal spasm, and maintain total calcium levels above 7.0–7.5 mg/dL (ionized calcium greater than 0.7 mmol/L). Long-term treatment of patients with chronic hypocalcemia is done with 1 to 3 grams of elementary calcium per day in the various forms of salts available3 All patients who become hypocalcemic must use vitamin D or analogues in addition to calcium. The vast majority of patients obtain control with calcitriol in dosages of 0.25 µg, taken twice daily, up to 0.5 µg four times daily. Hypoparathyroidism causes increased excretion of urinary calcium in relation to serum calcium and predisposes hypercalciuria, nephrolithiasis, and nephrocalcinosis. The product of calcium × phosphate must be kept below 55. Patients must have their kidneys radiologically evaluated regularly in order to rule out nephrocalcinosis4.

Conclusion

1. Ironically, increasing reliance on high end investigations such as a MRI brain scan could lead to certain conditions being missed
2. All treatable metabolic conditions should be excluded at first before commencing with anticonvulsants; this will restrict patients from burdensome polytherapy and related side effects.

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