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Tetany Causing Rhabdomyolysis in a Patient with Severe Hypovitaminosis D

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Abstract

Vitamin D is an extremely crucial vitamin that serves various functions in the body and the deficiency of which can lead to a wide range of clinical manifestations ranging from slight numbness and tingling in the extremities to serious cancers. We present the case of a 21-year-old woman, who presented with carpopedal spasms resulting in rhabdomyolysis secondary to severe Vitamin D Deficiency. She reported that she lived a sedentary life style and that her diet was very poor. Initial laboratory findings elicited that she had hypovitaminosis D, hypocalcemia, hypokalemia, and an elevated creatine kinase level. She was started on treatment with fluids, calcium supplements, and high dose ergocalciferol. Few days later her symptoms started to resolve along with her laboratory abnormalities. This case demonstrates that something as simple as Vitamin D deficiency can cause an enormous burden on the patient and lead to serious medical complications if no interventions are made in time.

Keywords

vitamin D deficiency; rhabdomyolysis; tetany; hypocalcemia; carpopedal spasms

Introduction

Vitamin D is a fat-soluble vitamin that helps with the absorption of dietary calcium and phosphorus from the intestine [1]. In addition, it also helps to suppress the release of parathyroid hormone (PTH) which is involved in the process of bone resorption [1]. Vitamin D deficiency (VDD) by any cause can lead to decreased absorption of calcium which in turn causes elevated PTH levels and eventually it can lead to osteopenia and osteoporosis [1].

Rhabdomyolysis is defined as breakdown of muscle tissue that leads to the release of muscle fiber contents into the blood which if severe can lead to acute renal failure [2]. Rhabdomyolysis most commonly ensues secondary to prolonged exertion. Currently, there is only one case report associating Vitamin D deficiency-induced myopathy with rhabdomyolysis [3]. To our knowledge, we report the first case of tetany-induced rhabdomyolysis secondary to severe VDD.

Case Presentation

A 21-year old African American lady who presented to the Emergency Department (ED) with complaints of generalized weakness and severe muscle cramps of her hands and feet for several months duration. She denied any strenuous activity or prolonged exertion. She does not have any significant past medical history including no history of loose or frequent bowel movements and she is not taking any

medications regularly. She specifically endorsed no history of ever taking any diuretics or laxatives. Her urine was of clear straw color.

On physical examination she was obese with a total body weight of 86kg (Kilogram) and a BMI of 31kg/m2 (Kilogram per meter squared). Both of her hands were contracted and both hands and feet were painful to movement in addition to experiencing numbness and tingling. She also experienced weakness of the distal muscles of bilateral hands and forearms. Further examination elicited that she had a positive Trousseau's sign but a negative Chvostek's sign otherwise her neurological exam including deep tendon reflexes were intact. Examination of the rest of the systems including cardiovascular and pulmonary were within normal limits. She also had normal peristaltic movements.

Initial laboratory tests in the ED elicited hypocalcaemia of 6.9 mg/dl, elevated PTH 896 pg/ml (normal value 10 to 55 pg/ml), creatine kinase 392 units/L, hypokalemia of 3.0 mEq/L, and Vitamin D level of 12.8 ng/ml. Of note, she had a normal kidney function with a creatinine of 0.54 mg/dL, a normal alkaline phosphate level of 84 u/L (normal value 34 to 104 u/L), low normal phosphate of 3.5 mg/dL, and low normal magnesium of 1.7mg/dL. Moreover, her EKG elicited normal sinus rhythm at 80 beats per minute with non-specific T wave abnormalities and mildly prolonged QTc interval of 482ms.

We started treatment for severe VDD with fluids, calcium supplements, and high dose ergocalciferol (50,000 units weekly for 30 days) while in the hospital. Over the next two days, her cramps in the hands started to resolve. By the third day her creatine kinase trended down to 152 and she was discharged on ergocalciferol 50,000 units once every week for 3 months, calcium carbonate 1,250 three times a day with meals, potassium chloride, and magnesium supplements. She was followed up in an out-patient clinic after 1 week and had a complete resolution of her symptoms and the carpopedal spasms. She was also instructed to follow up in the clinic periodically to have her laboratory abnormalities monitored.

Discussion

Vitamin D deficiency or insufficiency definition depends on the level of 25-hydroxy vitamin D (25(OH) D) in the serum. The normal level of 25(OH) D in blood is 30-50 ng/ml [4]. Vitamin D deficiency is defined as a 25-hydroxy vitamin D level of less than 20 ng/ml, while vitamin D insufficiency is defined as a level between 21 ng and 29 ng/ml [5]. Recently conducted survey in the United States showed that the prevalence of vitamin D insufficiency has doubled in the last 10 years. This most commonly affects the pigmented population such as Asians, Hispanics and African Americans about 90% of the time and ¾ of the entire Caucasian population is affected by vitamin D insufficiency as well [6].

Prevalence of hypovitaminosis D can be influenced by several factors making it a multifactorial etiology. The most common and the most important risk factor in the world that leads to VDD is inadequate sunlight exposure [7]. The wide spread fear of skin cancer in modern cities leads to utilization of sun protective lotions which increases the risk of having VDD. Additional risk factors that predispose patients to VDD are malnutrition, obesity, African American ethnicity, female gender, winter, and keeping most of the body covered [7, 8]. Dark-skinned people are more at risk due to the fact that vitamin D synthesis can decrease by 50% to 90% since melanin also functions as a natural sunscreen [8]. Moreover, VDD has also been associated with increased risk of common cancers such as breast, prostate, and colon,

autoimmune diseases, hypertension, infectious diseases, and even depression [9, 10].

Vitamin D is also related to improving muscle strength and it is conducted through receptors which are very specific to the muscle tissues [11]. Over the years, serum 25 (OH) D has also been related to physical performance in athletes [12]. In subjects not receiving statins, low serum 25 (OH) D levels have been associated with myositis [13] and reduced muscle function [14]. Treatment of VDD depends on the level of vitamin D. One of the most common VDD treatment strategies is giving oral ergocalciferol at 50,000 IU once a week for 8 weeks in total. The goal of the treatment is to reach a minimum level of 30 ng/ml of vitamin D in the blood and optimal time of checking a level after therapy has not been well defined in the literature. Once the vitamin D levels replete with the initial loading dosage, the patient should be started on maintenance dosages of cholecalciferol at 800 to 1,000 IU every day from combine dietary and supplemental regimens [15, 16, 17]. Moreover, in the case above, tetany was most likely caused by hypocalcemia and vitamin D deficiency and not hypokalemia as mild hypokalemia (levels between 3 to 3.5 mEq/L) rarely cause symptoms. Her calcium level was low enough to cause tetany.

Conclusion

In conclusion, hypocalcemia secondary to Vitamin D deficiency can lead to severe spasms in hands and feet (carpopedal spasm). With no treatment, these spasms can persist for a long time leading to rhabdomyolysis. The incidence of rhabdomyolysis secondary to severe Vitamin D deficiency is unknown. Our case aims at increasing the awareness of such complication in people with severe vitamin D deficiency. With some level of suspicion especially in cases with prolonged carpopedal spasms, we can avoid the morbidity and possible mortality of untreated rhabdomyolysis. Lastly, appropriate treatment of Vitamin D Deficiency is also important from the view of the cardiovascular system health. It has been determined that VDD can also lead to increased morbidity and mortality by increasing the risk of developing heart failure, myocardial infarction, and even sudden cardiac death [18].

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