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Case Report

HYPOVITAMINOSIS D: RARE CAUSE OF SEIZURE DISORDER IN AN ADULT

Gautam A [1]*, Agrawal PK[2], Bansal MK[3], Pursnani N[4]

1. **Assistant Professor,P. G. Department of Medicine, S. N. Medical College, Agra, Uttar Pradesh.**
2. **Assistant Professor, P. G. Department of Medicine, S. N. Medical College, Agra, Uttar Pradesh.**
3. **Associate Professor,P. G. Department of Medicine,S. N. Medical College, Agra, Uttar Pradesh.**
4. **P.G. Resident,Department of Medicine,Mahatma Gandhi Medical College, Jaipur, Rajasthan.**

Corresponding Author: Dr. Gautam A *Assistant Professor,P. G. Department of Medicine,S. N. Medical College, Agra, Uttar Pradesh.Cell: +919927089665.

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ABSTRACT

Since its recognition as an important constituent of vitamin family, vitamin D has got maximum recognition in last few years. Almost associated with every system of body vitamin D has now found to be associated with several prevalent diseases like cardiovascular diseases[1], diabetes[2], Central Nervous System disorders etc.[3], [4]. High prevalence of vitamin D deficiency in community is also being reported in a last few years. Deficiency of vitamin due to any cause can lead to mild hypocalcaemia, compensatory mild rise in parathyroid hormone levels and fall in blood phosphate levels is a well known pathological situation. This condition is frequently severe in infants and neonate, where serum calcium levels can fall critically and can lead to seizures[5]. "Vitamin D deficiency is also prevalent in India" is a recognized fact that has been proved by several epidemiological studies in past few years. Here we present a case of middle aged women who developed status epilepticus due to severe hypocalcaemia secondary to nutritional vitamin D deficiency. This is a first case reported ever in available literature.

KEY WORDS: Hypovitaminosis D, Seizure.

CASE REPORT:

A 52 year old Muslim female was brought to casualty in a status epilepticus. She had six episodes of seizure in last 1 hour, and till the treatment was started three more episodes developed in casualty. Promptly she was treated with IV lorazepam and intubated as the condition appeared to be life threatening. There was no history of headache, vomiting, head injury, diabetes, hypertension, any cardiovascular disorder, tuberculosis any drug abuse or chronic medication for any other disease. There was no previous or family history of seizure. Her vitals were stable with BP 100/76 mm of Hg, pulse rate 110/min regular and slight increased respiratory rate. She was in altered sensorium. Her planters were extensor, but there were no signs of raised intracranial tension or focal neurological deficit. Her

immediate NCCT head was normal. Her electrolytes were sent with arterial blood gas (ABG) analysis to exclude metabolic cause of seizure. Till then she was loaded with phenytoin and continued with SOS IV lorazepam; seizures still continued. Except with very low serum calcium levels (0.4 mmol/L, normal 1.12–1.32 mmol/L) all other investigations including ABG were normal. Her hemoglobin was 9.0 gm/dl microcytic hypo chromic and random blood sugar was 132 mg%. IV calcium gluconate started to correct the hypocalcaemia resulting in dramatic suppression of seizures. Consciousness regained after 24 hours when she was extubated. Patient denies of any tetanic or carpopaedal spasm, parasthesia or tingling sensation in limbs in past. Further workup for hypocalcaemia reveled severe vitamin D deficiency (13.46 nm/L, deficiency <25 nm/L) with markedly elevated parathyroid hormone level (635.80 pg/ml, normal 15-65 pg/ml). Remaining blood investigations showed normal serum sodium, potassium and magnesium levels but reduced serum phosphates (0.5 mmol/L, normal 0.81–1.4 mmol/L). Serum alkaline phosphatase levels were increased (196 U/L, normal 33-96 U/L). MRI brain and EEG were normal. Post extubation period was uneventful and she was discharged on 5^{th} day without antiepileptic. In follow up her serum calcium levels remains normal with adequate oral calcium and vitamin D supplementation. Further vitamin D levels were not done due to financial constrains. She remains asymptomatic thereafter. On the basis of investigations we concluded the diagnosis of severe hypovitaminosis D leading to hypocalcaemic seizures.

Patient was from a Muslim family and lives in small congested shady house. She use to follow the Muslim traditions strictly and was regularly wearing traditional clothes (***burkah***). This background substantiates the diagnosis of hypovitaminosis D due to inadequate sun exposure.

DISCUSSION :

Two bioequivalent forms of vitamin D are recognised; vitamin D_2 (D_2) called ergocalciferol and vitamin D_3 (D_3) called cholecalceferol. Source of D_2 are dietary vegetables only where as D_3 is primarily produced in skin on exposure to ultraviolet B radiation of sunlight. Dietary source of D_3 are fish, milk, yogurt, soya and juices.

Vitamin D, calcium and parathyroid hormone are three interrelated components of calcium homeostasis in body[6]. Maintenance of serum calcium require sufficient serum vitamin D levels and effective parathyroid hormone. Vitamin D aids in increase of calcium absorption from gut, decreases renal calcium excretion, and to and fro movement between bones and blood. Decrease serum calcium level stimulates parathyroid secretion of PTH. Thus low serum vitamin D levels due to either dietary deficiency or inadequate sun exposure decreases serum calcium levels and secondary hyperparathyroidism. Low vitamin D levels can also be found in patients with Primary hyperparathyroidism but this is associated with high serum calcium levels. Serum vitamin D levels remains normal in hypocalcemia due to dietary calcium deficiency.

Maximum gut calcium absorption occurs when vitamin D levels ranges between 30-40 ng/ml but this requires adequate dietary calcium supply[7]. So a daily recommended dose of elemental calcium in diet is 1.0 gm for less than 50 years and 1.2 gm for more than 50 years. Simultaneously dietary vitamin D recommendation is 800-1000 IU/day or 50,000 IU/month which ensures adequate serum vitamin D levels. Vitamin D deficiency was supposed to be common among people with low socioeconomic status due to inadequate dietary intake[8], [9], [10]. But in recent years several studies proved its deficiency globally among both high and low socioeconomic groups of peoples. Besides dietary intake the latitude in which the person lives, cultural dressing, level of sun exposure, use of sunscreens, atmospheric pollution, melanin pigmentation is also very important factors for development of vitamin D

deficiency; cause being decreased Ultraviolet B radiation exposure[11]. Chronic gastrointestinal, hepatic or renal disease also leads to development of vitamin D deficiency. Deficiency of vitamin D due to decrease sun exposure cannot be compensated with routine dietary supplement.

Hypocalcemic seizures are predicted when serum calcium level falls below critical level of 8.5 mg/dl (ionized calcium <4.0 mg/dl). A rapid rate of decrease of serum calcium and very low serum calcium levels induces seizures[12], [13], [14]. Vitamin D deficiency is most common cause of hypocalcemia. Causes of vitamin D Deficiency are chronic kidney disease, hepatic failure, decrease intake, intestinal malabsorption, antiepileptic drugs etc. Critical hypocalcaemia leading to seizures due to nutritional vitamin D deficiency is common in infants and neonates but not reported in adults yet. A case of hypocalcaemic epilepsy is reported in a somalian teenage suffering from vitamin D deficiency rickets[15]. Another case of hypocalcaemic seizure with bilateral frontal bone fracture in an adolescent with primary vitamin D deficiency is reported by Schnadower D et al[16]. Few literatures suggest the antiepileptic property of vitamin D is presumed to be one of the causes behind sudden unexpected death in epilepsy (SUDEP)[17]. Normalization of serum vitamin D is also considered to have anticonvulsant effect in pharmaco-resistant epilepsy[18]. Vitamin D deficiency is also one of the proposed reasons behind refractory seizures in infants and neonates. Our case is first ever reported case of hypocalcaemic seizures in adults due to nutritional deficiency of vitamin D in an adult.

CONCLUSION:

Seizures an extra skeletal manifestation of vitamin D deficiency is common in infants and neonates but rare in adults. Vitamin D deficiency could be considered as cause behind pharmaco- resistant and refractory seizures.

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