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Head Tingling as a Rare Neurological Manifestation of Severe Hypovitaminosis D in an Adolescent: A Case Report

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Abstract

Although Hypovitaminosis D (Vitamin D Deficiency) is commonly known to cause musculoskeletal manifestations, it can also be manifested with unusual symptoms, making the diagnosis challenging, especially in adolescents without the obvious risk factor of obesity. This article presents a case report with an atypical neurological manifestation, head tingling, that illustrates how evasive the diagnosis of hypovitaminosis D can be, especially in an adolescent with low-normal weight, as it is often overlooked in non-obese individuals. This case highlights a potential direct neurologic manifestation of severe hypovitaminosis D in adolescents with normal calcium levels, expanding the clinical spectrum of vitamin D deficiency. The report summarizes the unusual presenting symptoms, the response to vitamin D supplementation, and the recommendations for pediatric clinicians with regard to the current guidelines.

Keywords

Hypovitaminosis D, Vitamin D Deficiency, Adolescents, Head Tingling, Pediatric-Case Report

1. Introduction

Hypovitaminosis D in adolescents has become endemic and often goes unnoticed due to the absence of classic symptoms such as osteomalacia. There is no universally accepted normal vitamin D level; however, some authorities define deficiency as a serum 25-hydroxyvitamin D [25(OH)D] level < 12 ng/mL (<30 nmol/L) and insufficiency as 12 - <20 ng/mL (30 - <50 nmol/L) [1]. Due to lack of consensus,

other authorities and laboratories still consider levels of 20 - 29 ng/mL (50 - 74 nmol/L) as insufficient [2] [3].

Hypovitaminosis D is more prevalent among adolescents with excess weight, darker skin, limited sunlight exposure, restricted diets, or malabsorption disorders, but it is often overlooked in non-obese individuals [1]. Though more common in older adults, its incidence in adolescents is rising, partly due to decreased outdoor activity and increased nutritional demands during growth and puberty [2] [4].

Common symptoms include musculoskeletal complaints; however, atypical neurological symptoms such as acral paresthesia have also been documented [5]. To our knowledge, head tingling has not previously been associated with vitamin D deficiency alone, independent of hypocalcemia. Common causes of head or scalp tingling include neurologic, metabolic, infectious, inflammatory, and psychiatric conditions, as well as deficiencies in vitamins B1, B6, B12, and E [1] [6].

This report presents a rare case of severe vitamin D deficiency manifesting primarily as head tingling, sometimes accompanied by extremity tingling, in an adolescent male.

2. Case Presentation

2.1. Chief Complaint and History of Present Illness

A 14-year-old African American male, previously healthy, presented with a three-week history of daily diffuse head tingling. Episodes were intermittent, lasting several seconds, and sometimes associated with hand and foot tingling. There were no known triggers. He denied muscle cramps, pain, numbness, headaches, dizziness, fatigue, scalp changes, mood or vision disturbances, or other neurological symptoms.

2.2. Medical, Family, and Social History

Past medical history included mild scoliosis and a remote hand fracture. He had no history of hospitalizations or medication use. Family history was non-contributory, with no history of diabetes or malabsorption. Social history revealed recent academic stress due to transitioning to a competitive school. He reported 6 - 7 hours of sleep per night, denied substance use, and consumed a typical American diet.

2.3. Physical Examination

Comprehensive neurological and musculoskeletal examination was unremarkable, aside from mild scoliosis. He had normal cranial nerve function, strength, sensation, reflexes, coordination, and gait. BMI was 16.8 (8th percentile). No scalp abnormalities were noted.

2.4. Differential Diagnosis

The initial diagnosis was considered a stress-related disturbance. However, a broad

differential was explored, including metabolic disorders (e.g., diabetes, hypothyroidism), neurological diseases (e.g., migraines, multiple sclerosis, epilepsy), nutritional deficiencies (B vitamins, vitamin E, folate), autoimmune diseases (e.g., lupus, fibromyalgia), toxins, infections (Lyme disease, shingles), and congenital neuropathies [7] [8]. All these pathologies were ruled out through thorough history, physical examination, and laboratory studies.

2.5. Diagnostic Evaluation

Laboratory studies are summarized in Tables 1-3. Initial workup demonstrated a normal complete blood count and comprehensive metabolic panel, including normal calcium, vitamin B12, and glucose levels (Table 1 and Table 2). Hemoglobin A1c (5.4%) and thyroid-stimulating hormone levels were also within the normal range (Table 3). Serum 25(OH)D was markedly low at 5.6 ng/mL (reference range: 30 - 100 ng/mL), leading to a diagnosis of hypovitaminosis D. Linking head tingling to hypovitaminosis D itself in the absence of hypocalcemia was quite debatable; nevertheless, decision was made to attempt a trial of vitamin D supplementation to investigate any direct causal relation.

Table 1. Complete blood count with differential.

Parameter/UOM	Result	Reference Range
WBC ($\times 10^3/\mu L$)	7.1	3.4 - 10.8
RBC ($\times 10^6/\mu L$)	4.74	4.14 - 5.80
Hemoglobin (g/dL)	14.3	12.6 - 17.7
Hematocrit (%)	42.9	37.5 - 51.0
MCV (fL)	91	79 - 97
MCH (pg)	30.2	26.6 - 33.0
MCHC (g/dL)	33.3	31.5 - 35.7
RDW (%)	12.3	11.6 - 15.4
Platelets ($\times 10^3/\mu L$)	311	150 - 450
Neutrophils (%)	44	Not established
Lymphocytes (%)	43	Not established
Monocytes (%)	9	Not established
Eosinophils (%)	3	Not established
Basophils (%)	1	Not established
Neutrophils, absolute ($\times 10^3/\mu L$)	3.1	1.4 - 7.0
Lymphocytes, absolute ($\times 10^3/\mu L$)	3.1	0.7 - 3.1
Monocytes, absolute ($\times 10^3/\mu L$)	0.6	0.1 - 0.9
Eosinophils, absolute ($\times 10^3/\mu L$)	0.2	0.0 - 0.4
Basophils, absolute ($\times 10^3/\mu L$)	0.1	0.0 - 0.3
Immature granulocytes (%)	0	Not established

Abbreviations: UOM = unit of measure WBC = white blood cell count; RBC = red blood cell count; MCV = mean corpuscular volume; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; RDW = red cell distribution width.

Table 2. Metabolic and nutritional laboratory evaluation.

Test	Result	Units	Reference Range
Glucose	71	mg/dL	70 - 99
BUN	8	mg/dL	5 - 18
Creatinine	0.49	mg/dL	0.49 - 0.90
Sodium	139	mmol/L	134 - 144
Potassium	4.0	mmol/L	3.5 - 5.2
Chloride	102	mmol/L	96 - 106
Carbon dioxide (total)	17 ↓	mmol/L	20 - 29
Calcium	9.7	mg/dL	8.9 - 10.4
Total protein	7.6	g/dL	6.0 - 8.5
Albumin	5.1	g/dL	4.3 - 5.2
Total bilirubin	1.0	mg/dL	0.0 - 1.2
Alkaline phosphatase	269	IU/L	114 - 375
AST	22	IU/L	0 - 40
ALT	14	IU/L	0 - 30
Vitamin B12	289	pg/mL	232 - 1245
Folate (serum)	12.8	ng/mL	>3.0

Table 3. Endocrine and glycemic markers.

Test	Result	Reference Range
Hemoglobin A1c (%)	5.4	4.8 - 5.6
TSH (μIU/mL)	2.810	0.450 - 4.500
Vitamin D, 25-hydroxy (ng/mL)	5.6 ↓	30.0 - 100.0

↓ indicates value below reference range.

2.6. Management and Outcome

The patient was started on vitamin D supplementation at 50,000 IU weekly for 8 weeks. The level of serum 25(OH)D increased to 20 ng/mL, with marked symptoms' improvement (tingling was very rare). After 4 more weeks of the same dosage, due to occasional symptoms, his vitamin D level increased to 39 and symptoms completely resolved. Education regarding modifiable risk factors, dietary intake (recommended dietary allowance for vitamin D: 400 - 600 IU), and sunlight exposure was provided for optimal level maintenance [1].

3. Pathophysiology

Vitamin D, particularly in its active form calcitriol [1,25$(OH)_2$D], is now recognized as a neurosteroid hormone involved in neurodevelopment, neurotransmission, and neuroprotection [7] [8]. Vitamin D deficiency may disrupt neuronal ex-

citability through altered calcium and chloride channel regulation, leading to peripheral neuropathy and central sensitization [5] [7] [8].

Vitamin D receptors are widely expressed in peripheral and central neural tissue, where vitamin D regulates neurotrophic factors such as nerve growth factor [9]. Deficiency may impair neuronal survival, growth, and repair, contributing to neuropathic symptoms, including tingling, via direct neuronal excitability or altered sensory processing [9] [10]. Vitamin D also modulates inflammatory cytokines, and deficiency may promote neuroinflammation and worsen neuropathic symptoms [10] [11]. Recent clinical guidelines recognize vitamin D deficiency's wider implications, including neuromuscular symptoms like muscle weakness and pain that may indirectly contribute to neurologic complaints such as tingling [11].

4. Discussion

This case highlights a rare presentation of severe hypovitaminosis D with normal serum calcium manifesting as head tingling in an adolescent, with complete resolution following vitamin D supplementation. While acral paresthesia and other neurological symptoms have been reported in vitamin D deficiency, head tingling in the absence of hypocalcemia has not been previously described [5] [12]-[14]. Other potential tingling causes were thoroughly excluded through clinical evaluation and laboratory testing [1] [6].

Unlike prior reports linking paresthesia to hypocalcemia in vitamin D deficiency [4] [15], this patient had normal calcium levels, suggesting a direct neurologic effect of vitamin D deficiency. Previous clinical guidelines and trials have reported resolution of paresthesia after correcting vitamin D deficiency with calcium repletion, suggesting a causal role for calcium in those cases [11]. The absence of electrolyte abnormalities, including normal serum calcium, strengthens the hypothesis that vitamin D deficiency itself may contribute to neurologic symptoms rather than acting solely through secondary hypocalcemia. This observation broadens the clinical understanding of hypovitaminosis D and suggests that neurologic symptoms may be an underrecognized manifestation in adolescents.

Additionally, this case supports evidence that serum vitamin D levels below 30 ng/mL may not be optimal, as complete symptom resolution occurred only after levels exceeded this threshold [2] [3].

Limitations include inherent constraints of a single-case report, which preclude definitive causal inference. While the temporal relationship between supplementation and symptom resolution is strong, it does not definitively prove causation, and a coincidental recovery cannot be entirely excluded. This case study did not extend beyond the maintenance phase to ensure that tingling did not recur. Further prolonged studies are needed to reinforce the direct correlation between vitamin D deficiency without hypocalcemia and head tingling.

5. Conclusion

Severe hypovitaminosis D may present with atypical neurological symptoms, in-

cluding head tingling. The case highlights a direct neurologic effect of vitamin D deficiency rather than an effect of secondary hypocalcemia. It supports consideration of targeted vitamin D evaluation in adolescents presenting with unexplained sensory symptoms or known risk factors. Pediatric clinicians should consider vitamin D deficiency in the differential for unexplained paresthesia and aim at optimal levels above 30 ng/dL when supplementing vitamin D. Screening symptomatic or high-risk adolescents should be emphasized in pediatric practice, regardless of obesity or overweight status.

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Ethical Statement

Written informed consent was obtained from the patient's parent for publication of this case report and any accompanying results.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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