

Hypocalcaemic Tetany and Seizures Secondary to Vitamin D Deficiency

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Abstract

Presentation

A female adolescent presented with a generalised tonic-clonic seizure preceded by hands stiffness. She presented with a sudden history of being unresponsive with head deviation to right side, followed by up-rolling of eyes, and whole body shaking which self-terminated after five minutes. On examination, she was in postictal state, with inward clenching of both hands and fingers. The rest of her systemic examination was normal.

Diagnosis

Laboratory results showed severe hypocalcaemia, hypomagnesaemia, hyperphosphatemia, elevated parathyroid hormone (PTH), and critically low vitamin D. ECG showed a prolonged QTc interval. Biochemical profile was consistent with secondary hyperparathyroidism. Severe vitamin D deficiency was the cause of her hypocalcaemia.

Treatment

The patient was managed acutely with intravenous calcium gluconate and magnesium, followed by oral calcium and vitamin D supplementation.

Discussion

This case highlights hypocalcaemia-induced seizures due to vitamin D deficiency in an adolescent, emphasising the importance of early recognition, biochemical assessment, and supplementation in at-risk populations.

Introduction

Hypocalcaemia is one the most common biochemical disorders seen in children. Severe hypocalcaemia can cause potentially life-threatening conditions such as cardiac arrhythmias, respiratory arrest, seizures, and tetany.

This case report describes the presentation, investigations, and management of severe symptomatic hypocalcaemia due to vitamin D deficiency in an adolescent female. Paediatric Innovation, Education & Research (PIER) Network guidelines¹ regarding acute management of hypocalcaemia were followed. Patient received intravenous calcium therapy as per guidelines. On stabilizing the serum calcium levels, patient was started on oral calcium and vitamin D supplementation.

Case Report

A female adolescent presented following a history of sudden loss of consciousness with head deviation to the right side, followed by generalised tonic-clonic seizure which self-terminated after five minutes at home. There was no history of tongue bite or urinary incontinence. She had complained of stiffness and pain in her hands one day prior to this event. On examination, she was noted to have inward clenching of hands and fingers (Trousseau's sign). On tapping her facial nerve, there was visible twitching of her facial muscles (Chvostek's sign).

Initial laboratory investigations showed severe hypocalcaemia, hypomagnesemia, and markedly raised PTH as illustrated in Table 1. Her vitamin D level was available after a few hours which was found to be critically low. Her ECG showed prolonged QTc interval of 518ms.

She received one intravenous calcium gluconate bolus as per PIER Network guidelines¹, followed by intravenous calcium infusion as per British National Formulary for Children (BNFC)² dosage. Other guidelines used were NHS Scotland guidelines³ to manage her hypocalcaemia. She received 24 hours of intravenous calcium maintenance. Paediatric endocrinologists recommended transition to oral calcium and high-dose cholecalciferol supplementation on satisfactory improvement in serum calcium to 1.9mmol/L^{3,4}. After 24 hours, she was slowly weaned off from IV calcium maintenance to avoid sudden drop in calcium levels. She had associated hypomagnesemia and received intravenous magnesium sulphate. Genetic testing for pseudohypoparathyroidism was initiated⁵, although the biochemical profile strongly supported secondary hyperparathyroidism. Radiographs of the wrists and knees showed no evidence of rickets. The cause of her hypocalcaemia was found to be severe vitamin D deficiency. She had a history of extremely poor dietary intake, combined with limited sun exposure, leading to vitamin D deficiency.

She was discharged home on oral calcium, magnesium, and cholecalciferol supplementation, with the advice to stop calcium and magnesium supplementation after 2 weeks, and to continue high dose cholecalciferol supplementation for another 3 months⁶. Her ECG normalised, and QTc interval was 420ms. The current plan from endocrine team is to keep her on 6 monthly follow ups with measurement of bone profile and vitamin D level.

Investigation	Patient Result	Reference Ranges	Interpretation
Ionised Calcium	0.51 mmol/L	1.15 – 1.29 mmol/L	Severe hypocalcaemia
Adjusted Calcium	1.09 mmol/L	2.10 – 2.60 mmol/L	Severe hypocalcaemia
Serum Magnesium	0.72mmol/L	0.70 – 1.00 mmol/L	Hypomagnesaemia
Serum Phosphate	Elevated	0.8 – 1.5 mmol/L	Hyperphosphataemia
Parathyroid Hormone	Markedly Elevated	1.6 – 6.9 pmol/L	Secondary hyperparathyroidism
Vitamin D	11.1nmol/L	> 50 nmol/L	Severe vitamin D deficiency
ECG	518ms	460 ms	QT prolongation

Table 1: Laboratory Findings

Discussion

This case illustrates the classical but uncommon presentation of hypocalcaemia with tetany, carpopedal spasms, and seizures in the setting of profound vitamin D deficiency. The vitamin D deficiency alone usually produces gradual, chronic hypocalcaemia that rarely leads to overt neuromuscular irritability. Seeing acute, symptomatic hypocalcaemia severe enough to trigger seizures is clinically striking. The marked elevation of PTH confirmed secondary hyperparathyroidism, reflecting an appropriate compensatory physiological response to hypocalcaemia, to maintain normal serum calcium levels, and excluding primary hypoparathyroidism.

A PTH measurement at the time of hypocalcaemia is key to figuring out the underlying aetiology. Hypocalcaemia associated with low PTH can be caused by parathyroid aplasia, following neck surgery, e.g. thyroidectomy, DiGeorge syndrome, and X Linked hypoparathyroidism⁴. Hypocalcaemia associated with high PTH can be caused by iatrogenic reasons such as excessive phosphate intake, e.g. high phosphate containing milks, TPN, Vitamin D deficiency, impaired vitamin D metabolism, and vitamin D dependent rickets.

Every child with suspected hypocalcaemia should have plasma calcium, adjusted calcium, alkaline phosphatase, urea and electrolytes, serum PTH levels, vitamin D levels, ECG, X-rays of wrists and knees done to diagnose the cause of hypocalcaemia, and to guide management.

A study⁷ done in London previously showed severe hypocalcaemia due to vitamin D deficiency occurred in approximately 6 per 10,000 infants per year, primarily affecting exclusively

breastfed Asian infants under one year of age, often presenting with afebrile or febrile-type seizures. Maternal deficiency strongly predicted infant deficiency, and a small number of children showed clinical or radiological rickets, highlighting the need for targeted prevention.

This case reinforces the need to consider metabolic causes of new-onset seizures in children and adolescents. Vitamin D deficiency is a preventable cause of hypocalcaemia, with timely recognition and supplementation critical in avoiding serious complications.

Declarations of Conflicts of Interest:

None declared.

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