Case Report

Intractable hypocalcemic seizures with neuropsychiatric symptoms- An under-diagnosed case

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Abstract

Idiopathic hypoparathyroidism is one of the important and treatable causes of hypocalcaemia. Patients with hypocalcaemia as a consequence of hypoparathyroidism can have varied neuropsychiatric presentations such as intractable seizures, depressive symptoms, psychosis, generalized parasthesias and extrapyramidal syndrome. Many times these patients are being wrongly treated as a case of depression without addressing the underlying cause. We present a case of 26-year-old female patient who presented in emergency with multiple episodes of complex partial seizures since the last 6 months and under the treatment for depression for 2 years. She was diagnosed as a case of hypocalcemia and hypoparathyroidism was considered as the underlying cause.

Keywords: Hypocalcemia, idiopathic hypoparathyroidism, neuropsychiatric symptoms

Introduction

Hypocalcaemia may present as tetanic spasms, parasthesias, intractable seizures, and neurocognitive manifestations.[1] Idiopathic hypoparathyroidism is an important cause of hypocalcemia. The manifestations of hypoparathyroidism are that of hypocalcaemia.[2] These patients are usually misdiagnosed as having adult epilepsy disorder or depression, thus overlooking the underlying etiological workup.[3,4] Few patients of idiopathic hypoparathyroidism present mainly with symptoms of depression and psychological abnormalities. Existing literature shows that the frequency of mental disorders is more common in patients with secondary hypoparathyroidism. The association of behavioural and psychological disorder with idiopathic hypoparathyroidism has rarely been reported. It poses a diagnostic challenge and is thus of importance to the primary care physician who may come across such patients with neuropsychiatric symptoms with no suggestive history of secondary hypoparathyroidism.[5-7] Hypocalcemia is also an important aetiology of acute symptomatic or new-onset seizures. This case report highlights hypocalcemia due to hypoparathyroidism as one of the under-diagnosed causes of neurocognitive symptoms.

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Case Report

A 22-year-old woman presented to emergency with complaints of sudden-onset generalized numbness and tingling sensation, facial deviation towards left side followed by impaired consciousness for few minutes, without tonic-clonic movements, frothing from mouth, up rolling of eyes, bladder and bowel incontinence, postictal confusion and focal deficits. The patient had an episode of seizure 1 h prior to presentation and another complex partial seizure after 2 h of the first episode. She had such episodes for the last 6 months. She was started on phenytoin (300 mg) but seizures were uncontrolled. She had decreased sleep, excessive crying, suicidal tendencies, and auditory hallucinations for the past 2 years for which she was taking antipsychotic medications (Clonazepam)

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with no history of fever, head injury, tetany, carpopedal spasms in the patient and mental illness in the family.

Psychiatric examination done using the Montreal Cognitive Assessment and the Hamilton Anxiety Scale revealed moderate cognitive impairment and anxiety in the patient. Neurological examination revealed deficits in attention, calculation and simple task execution without focal deficits. Chovstek's and Trousseau's sign were positive. MRI brain revealed bilateral calcifications of cerebellum, caudate nucleus and globus pallidus. Laboratory serum calcium (corrected) was 5.9 mmol/L, with high serum phosphate 1.75 mmol/L, low parathyroid hormone levels 1.3 pmol/L (normal range 1.6–6.8 pmol/L), and vitamin D levels 10 ng/mL (Normal range 20-50 ng/mL). Serum magnesium, creatinine, thyroid-stimulating hormone, free T3, free T4 and serum albumin were normal and ECG suggested prolonged QT interval. The patient was diagnosed with idiopathic hypoparathyroidism leading to hypocalcemia, hypophosphatemia, and parathyroid deficiency. For complex partial seizures, she was started on oxcarbamazepine (300 mg twice daily). Due to uncontrolled seizures, while hospitalized, another antiepileptic drug (Clobazam 10 mg) was added and the dose of oxcarbamazepine was escalated gradually. Calcium supplementation was started, initially given intravenously on the first day (10 ampules of 10% Calcium gluconate in 1 litre of 5% Dextrose given at a rate of 50 ml/hr) followed by oral supplementation (2 gm) daily. Alfa-calciferol supplementation (0.25 microgram) was also added. On the 10th day of admission, her calcium rose to 7.6 mmol/L. She had no seizures for the next 1 week and improved symptomatically by day 14. She was discharged with oral calcium supplementation, alfa-calciferol (0.25 microgram) and antiepileptic medication.

However, she returned to the emergency 10 days after discharge with another episode of complex partial seizures and generalized paresthesias. Again, her serum calcium had fallen to 5.9 mmol/L on admission. She was given 10 ampules of calcium gluconate (10%) in 1 litre of 5% dextrose at a rate of 50 ml/hr intravenously followed by oral supplementation. Injection Teriparatide (20 microgram) was initiated daily which was gradually tapered to 10 microgram over a period of three months. She was monitored using serum calcium and urinary calciuria. Her serum calcium gradually improved over three weeks and she was discharged with follow up consultation with physician and psychiatrist. After six months of follow up, although she continues to have mild depression and anxiety, her seizures are controlled and her serum calcium levels are 9.2 mmol/L and serum parathyroid levels are within normal range.

Discussion

The diagnosis of idiopathic hypoparathyroidism was made by the constellation findings of hypocalcemia, hypophosphatemia and reduced parathyroid hormone levels. The common causes of hypoparathyroidism are post thyroid surgery, autoimmune polyendocrine syndromes, hemochromatosis, magnesium deficiency and kidney diseases. The differential diagnoses include vitamin D deficiency, hypomagnesaemia, malabsorption, kidney diseases and medication like steroids, diuretics, and anti-epileptics.[7-9]

No evidence of hypomagnesemia, vitamin D deficiency, kidney disease, past history of thyroid surgery and intake of medications such as steroids and diuretics led to diagnosis of idiopathic hypoparathyroidism in our patient. Evidence suggesting pseudohypoparathyroidism were also absent in our patient (cataract, shortened metacarpals/metatarsals, facial dysmorphism, goiter). The MRI brain of our patient was suggestive of basal ganglia calcification, the radiological feature seen in Fahr's disease. However, serum levels of calcium, phosphorous and vitamin D are normal in patients with Fahr's disease unlike in our patient.[9]

The clinical manifestations of primary hypoparathyroidism are categorized into acute and chronic hypocalcemia. The patients of acute hypocalcemia can have cardiac manifestations in the form of QT interval prolongation, T wave abnormalities and patterns of anteroseptal injury without evidence of infarction.[10] The patients of chronic hypocalcemia however may manifest as extrapyramidal symptoms, seizures, skin changes and rarely signs and symptoms of raised intracranial pressure. These patients can also present with neuropsychiatric manifestations.[4,6,11,12]

Most of the cases described in the literature are of secondary hypoparathyroidism as a consequence of surgery or drug-induced caused by proton pump inhibitors.[6,12,13] Regis *et al.* described hypocalcemia in a 22-year-old male patient manifesting as depression and seizures due to chronic omeprazole use. Thomas *et al.* and Wodarz *et al.* described parathyroid and thyroid surgery respectively as causes of hypocalcemia manifesting with neuropsychiatric symptoms.[13-15]

Take home messages

- Idiopathic hypoparathyroidism is a crucial differential diagnosis in patients with manifestations of hypocalcaemia
- Hypocalcemia may manifest in the form of psychiatric and neurological symptoms.
- Patients remain misdiagnosed or underdiagnosed due to the atypical neuropsychiatric clinical picture
- Prompt diagnosis and management may improve patient's quality of life.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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