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Case Report

Hypocalcemia: An Unusual Presentation: Case Report

Oyiyechukwu Onwudiwe[1*], Shadi Barakat[2]

[1]Department of Medicine, St. Agnes Hospital, Baltimore, Maryland; 21229

[2]Diabetes and Endocrinology Center, St. Agnes Hospital, Baltimore, Maryland; 21229

***Corresponding Author:** Oyiyechukwu Onwudiwe. Department of Medicine, St. Agnes Hospital, Baltimore, Maryland; 21229.

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Abstract

Calcium is among the top three ions in the body and plays an important role in maintaining normal cellular function, intracellular signaling, blood coagulation, neural transmission, and integrity of the bone structure. Endocrine disorders have long been known to present with various neuropsychiatric manifestations. We aim to show how hypocalcemia can present acutely as delirium without any other symptoms.

We focus our attention on hypocalcemia as a rare cause of delirium and psychosis as this is more commonly seen in hypercalcemia. We present a case of a 71-year-old patient with psychosis and delirium following an acute drop in calcium levels and review the existing literature.

The relationship between calcium metabolism and psychiatric disturbances is poorly understood. There are few published reports of neuropsychiatric manifestations in hypocalcemia and most are due to bisphosphonates use which was not the case in our patient. She also had no other symptoms of hypocalcemia.

Hypocalcemia may present acutely with only a change in mental status and without any of the classic symptoms of numbness, tingling, or paresthesia. This is a rare presentation of this abnormality and a high index of suspicion is needed for prompt management.

Key Words: hypocalcemia; delirium; psychosis; mental status; acute change

Introduction

Calcium is among the top three ions in the body and plays an important role in maintaining normal cellular function, intracellular signaling, blood coagulation, neural transmission, and integrity of the bone structure.[1] Generally, normal serum calcium ranges between 8.5-10.3mg/dL (2.12-2.57mmol/L) with 8.5mg/dL regarded as low normal. Hypocalcemia is a total serum calcium concentration less than 8.5 mg/dL (< 2.12 mmol/L) in the presence of normal albumin concentrations (3.5-5.5g/dl) or a serum ionized calcium concentration < 4.7 mg/dL (< 1.17 mmol/L).[2]

Hypocalcemia is most commonly caused by hypoparathyroidism. Other causes include Vitamin D deficiency, renal disease, sepsis/severe illness, and medications such as phenytoin, rifampin, and bisphosphonates (used to treat hypercalcemia)(3, 4). Magnesium depletion has also been shown to cause hypocalcemia. [5,6]

Common symptoms of hypocalcemia include neuromuscular irritability, muscle cramps, spasms and tetany, hyperreflexia, generalized seizures, circumoral numbness, and paresthesia of fingers and feet [7,8]. It could also present with prolonged QTc on EKG.

Less commonly hypocalcemia could present as cognitive impairment and personality changes but these are usually seen in chronic insidious cases of hypocalcemia.[7,8]

Our patient presented with psychosis, delirium, and aggression following acute hypocalcemia.

Case Presentation

A 71-year-old African-American female with a past medical history of CKD stage 5, primary and tertiary hyperparathyroidism underwent surgery in June 2021 with resection of all her parathyroid glands. The postoperative course was complicated by hypoparathyroidism. She was discharged on a stable oral calcium regimen. She was re-admitted 3 months later to the ED for hyperactive delirium and psychosis of one-day duration. Before this admission, she had been recently admitted 2 weeks earlier for hypercalcemia, likely due to taking too many calcium/calcitriol supplements. She was discharged only on oral calcium 1g twice a day. She then presented with a few hours' history of agitation. She was found in her room screaming for help. It was unclear what she was talking about. She was also noticed to be very restless and not having appropriate

answers to questions. Physical examination was unremarkable with negative Chvostek and Trousseau's signs. Laboratory tests were significant for serum calcium of 6.6mg/dl (range 8.5-10.5), albumin 3.5g/dl (range 3.4-4.8), ionized calcium 0.81mmmol/l (range 1.0-1.3) and normal serum magnesium. QTc was slightly prolonged at 480ms. She was admitted and treated with IV calcium gluconate, calcium carbonate, and calcitriol.

Altered mental status workup was negative including unremarkable brain imaging. She also had no previous history of any psychiatric diagnosis and since this was an acute change in her mental status, psychiatry was not consulted. There was no other explanation for the sudden change in her mental status except hypocalcemia. She was initially treated with intravenous calcium gluconate with improvement in her mental status as her serum calcium level increased. She was then switched to oral calcium which was followed by a drop in her calcium levels and a worsening of her mental state. Intravenous calcium gluconate was resumed together with oral calcium carbonate and calcitriol. Calcium gluconate was slowly tapered off with close monitoring of her ionized calcium. The patient's mental status also improved slowly with the correction of the calcium and she was back to baseline within a week. Her calcium regimen was strictly followed for 3 more days after improvement to ensure we were not over-correcting and pushing her to the other end of the spectrum. Her ionized calcium levels remained stable at 1.12 and she was discharged on calcium carbonate 2500mg and calcitriol at 0.75mcg three times a day with close follow-up.

Discussion

The relationship between calcium metabolism and psychiatric disturbances is not well understood. It is believed that calcium has some role in monoamine metabolism in the CNS thereby modifying neurotransmission and resulting in alterations in mood and cognition.[9] Neuropsychiatric manifestations have long been described in hypercalcemia [10,11] and are less commonly seen in hypocalcemia. There are few published reports of neuropsychiatric manifestations in hypocalcemia [3,4] and only one report not due to bisphosphonates. This case report found these manifestations in a patient with prolonged and chronic hypoparathyroidism with basal ganglia calcifications. [12]

Our case was noteworthy because our patient's presentation was more acute and she had no imaging findings on head CT and brain MRI. She also had no other symptoms of hypocalcemia except for the mildly prolonged QTc and she improved within a week of correcting her calcium deficit.

Conclusion

Patients with hypocalcemia may have none of the classic symptoms of numbness, tingling, or paresthesia and may present with only a change in mental status. Clinicians have to be made aware of this as a change in mental status from baseline is a very common presentation to the hospital and hypocalcemia is usually not on the differential for its etiology.

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