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Case Report

AN INTERESTING CASE OF IDIOPATHIC PRIMARY HYPOPARATHYROIDISM

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ABSTRACT

Chronic hypocalcemia has various neurological and neuromuscular manifestations which include muscle spasms, facial grimacing, carpopedal spasm, raised intracranial pressure, changes in finger nails and hair, lenticular cataracts, and in extreme cases laryngeal spasms and convulsions. Hypoparathyroidism is one of the most common causes of chronic hypocalcemia.
We present a case of recurrent convulsions, who was incidentally found to have basal ganglia calcifications, severe hypocalcemia associated with hypomagnesemia and hyperphosphatemia and was eventually diagnosed as primary hypoparathyroidism

INTRODUCTION

Hypoparathyroidism is an uncommon endocrine-deficiency disease characterized by low serum calcium levels, elevated serum phosphorus levels, and absent or inappropriately low levels of parathyroid hormone (PTH) in the circulation(1). Hypoparathyroidism has various causes which include postsurgical, autoimmune, genetic and infiltrative(2). When PTH secretion is insufficient or its action is flawed, hypocalcemia develops(3). Hypocalcemia due to hypoparathyroidism is associated with a range of clinical manifestations, ranging from paresthesias, cramps, tetany to life threatening arrhythmias, laryngospasm and seizures(4). Hypocalcemia associated with hyperphosphatemia, observed in long-standing hypoparathyroidism may lead to intracranial calcification, most commonly seen in the basal ganglia(1,4–7).
Here, we present a twenty one year old woman who was found to have recurrent convulsions who was diagnosed as primary hypoparathyroidism.

CASE REPORT

A 21 year old female diagnosed with Epilepsy 3 years ago, on regular medication of Oxcarbazepine, presented to the emergency room with one episode of generalised-tonic-clonic seizures, lasting for two minutes, resulting in a road traffic accident. The patient suffered broken teeth and multiple abrasions over her body. Patient did not have dysmorphic features, no positive familial history, no signs of other endocrine dysfunction with no previous surgical history. On examination, patient was found to have positive Trousseau's sign and negative Chvostek's sign. Her vitals were stable and her neurological examination revealed no deficit.

A CT scan brain was done as a precautionary measure and the patient was found to have multiple chunky and soft calcifications involving the bilateral capsuloganglionic region, bilateral caudate nucleus involving head & body, bilateral lentiform nucleus & thalamus, subcortical U fibers in bilateral frontal, right parietal lobe and also in dentate nucleus [Figure:1]. Her ECG showed QT interval prolongation. Patient's initial investigations revealed severe hypocalcemia (4.6 mg/dL), hypomagnesemia (1.1 mg/dL) and hyperphosphatemia (5.3 mg/dL).Fundus examination of the patient revealed bilateral early cortical and sub capsular cataract. Patient was initiated on intravenous antiepileptic, intravenous calcium gluconate, intravenous magnesium sulphate, oral vitamin D3 tablets (calcitriol) and other supportive measures. Her vitamin D levels were found to be normal (25.8 ng/mL) while her PTH levels were found to be less than 3 pg/mL. She developed one more episode of generalised tonic clonic seizures in the hospital which was

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managed with intravenous benzodiazepines. Patient's thyroid profile and serum cortisol were found to be normal.

The patient's calcium (8.1 mg/dL) and magnesium levels (1.8 mg/dL) were corrected, after which she developed symptoms and signs of hypercalcemia including confusion, anxiety, muscle weakness and sinus tachycardia. She was treated with intravenous fluids and loop diuretics after which she became asymptomatic.

An ultrasound abdomen was done, which was found to be normal and a 24 hour urinary calcium creatinine ratio. Patient was discharged after she remained asymptomatic for the next four days. She did not have any signs of hypocalcemia at the time of discharge. Her last calcium and phosphorous levels were 6.5 mg/dL and 7.7 mg/dL; with a normal magnesium level.

DISCUSSION

Hypocalcemia is usually defined as a total serum calcium concentration, corrected for protein, of less than 8.4 mg/dL and/ or an ionized calcium level less than 1.16 mmol/L(2). Hypoparathyroidism is one of the most common causes of chronic hypocalcemia(3). Hypocalcemia manifests as muscle spasms, carpopedal spasm, facial grimacing and in extreme cases laryngeal spasm and convulsions. Patients with hypocalcemia may also present with irritability, depression and psychosis. Respiratory arrest, heart failure, intestinal cramps, malabsorption and papilledema are rare findings. Chvostek's or Trousseau's sign are clues to diagnose hypocalcemia, so is prolongation of QT interval in the electrocardiogram (8).

Our case was evaluated for an episode of generalised tonic-clonic seizure followed by a road traffic accident in the Emergency Room. A CT scan brain was done to rule out trauma to the brain and patient was incidentally found to have multiple calcifications in the brain. Since patient showed features of hypocalcemia (QTc and Trousseau's positive), her calcium, phosphorous and magnesium with her routine investigations. While her renal parameters were found to be normal, the patient was found to have severe hypocalcemia, hypomagnesemia and hyperphosphatemia. This triad gave us a clue towards hypoparathyroidism while the calcifications in the brain itself gave us a clue towards pathology of the parathyroid gland. Patient's PTH was found to be very low and her Vitamin D levels were normal. Thus, pseudohypoparathyroidism and Vitamin D deficiency were ruled out.

The patient did not have dysmorphic features, no other endocrine dysfunction (Cortisol and TFT were normal), no familial history, adolescent onset and no cardiac dysfunction. These findings ruled out syndromes associated with hypoparathyrodism like DiGeorge's, APECED, HDR, MEN, Kenny-Caffey and others.

Thus, we were left idiopathic hypoparathyroidism that are the most likely diagnosis.

Patients with long standing hypoparathyroidism are symptomatic and thus need to be treated. Severe hypocalcemia is a medical emergency and needs to be treated with intravenous calcium therapy, which was initiated for our patient(9). Chronic hypoparathyroidism is treated with oral calcitriol and calcium supplements which was prescribed to our patient at discharge(1,9).

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Conflict of Interest: None

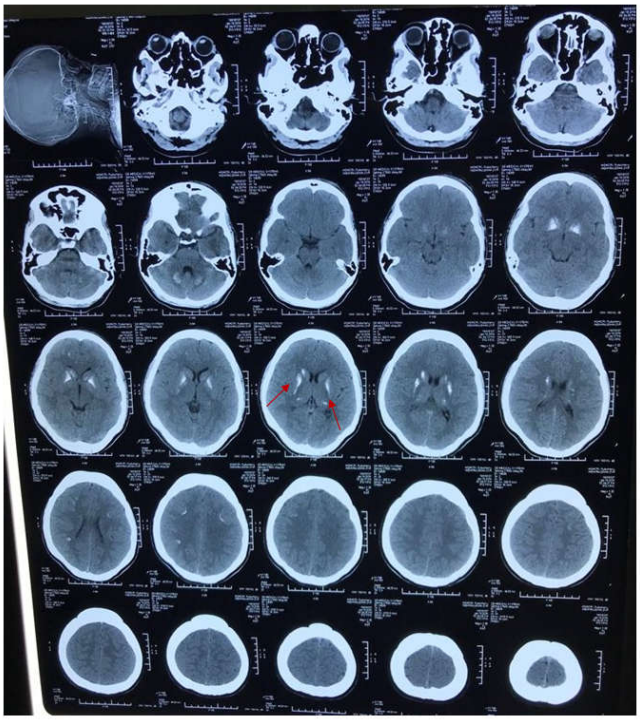

Figure 1 CT scan Brain. Arrows show bilateral basal ganglia calcification

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