CASE REPORT

A YOUNG MAN WITH GENERALIZED TONIC-CLONIC SEIZURES AND HYPOCALCAEMIA

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ABSTRACT

A young man presented with history of generalized tonic clonic seizures, second episode in one year. Previous clinical history was significant for episodic carpopedal spasms. Clinical examination was significant for the presence of Trousseau sign. ECG was evident of prolonged QT interval and Corrected QT interval. Laboratory investigations revealed low serum calcium (corrected serum calcium was also low). Neuroimaging showed multiple bilateral hyper dense foci of calcification in cerebral hemispheres, thalami, caudate nucleus and cerebellar lobes. Serum intact parathyroid hormone level was found to be low. Investigations for other endocrine pathologies did not reveal any abnormality. EEG was normal. Crossectional imaging of chest, abdomen and pelvis was also done to rule out other polyendocrine deficiency syndromes and was normal. Thus diagnosis of primary hypo-parathyroidism was established and patient started on life time calcium plus vitamin D supplements.

Keywords: Hypocalcaemia, Hypoparathyroidism, Seizures.

INTRODUCTION

Parathyroid hormone is a major factor in regulation of calcium metabolism along with vitamin D. It exerts direct effects on bone and kidney and indirect effects on the gastro intestinal tract. Most common causes of hypoparathyroidism are post surgery, autoimmune, developmental disorder of thyroid gland and impaired regulation of parathyroid hormone. Hypoparathyroidism can present with a variety of clinical manifestation depending on the severity of hypocalcaemia. Effects of low serum calcium on body are clinically manifested by tetany (symptoms of tetany range from perioral numbness, muscle cramps, carpopedal spasm to laryngospasm) focal or generalized seizures, cardiac dysrythmia, hypotension and heart failure. Basal ganglia calcifications due to hypo-parathyroidism is rare with prevalence of 0.5%[1]. Basal ganglia calcification is a manifestation of long standing hypoparathyroidism[2,3]. Calcification in brain can be easily picked by neuro-imaging[4]. First clinical manifestation is recurrent seizures in some patients with underlying idiopathic hypoparathyroidism[5]. In all such patients, index of suspicion should be kept high to investigate for hypoparathyroidism including other endocrine deficiencies. Treatment includes, seizure control, calcium and vitamin D supplementation which is lifelong.

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Received: 23 Apr 2019; revised received: 01 Aug 2019; accepted: 05 Aug 2019

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A 28 years old young male presented to emergency department with history of episodic loss of consciousness and jerky movements of the limbs, with a total of 2 episodes for last one year. He also complained of intermittent stiffening of the hands. On systemic inquiry he did not give any history of polyuria, headaches, visual problems, lethargy, cold intolerance, weight gain, discoloration of skin or mucous membranes, postural dizziness or altered bowel habits. There was no significant history in the past for any major surgical procedure or prolonged medical treatment. Patient has no known exposure to radiations in the past. His family history was not indicative of any endocrine disorder among near relatives. Travel history is non-contributory. On

examination, patient was a healthy looking young man, fully concious and alert to surrounding. Vitals signs were Pulse 78/min, regular, BP 130/70 mm Hg and respiratory rate 15/min. He was not febrile. There was no goiter,

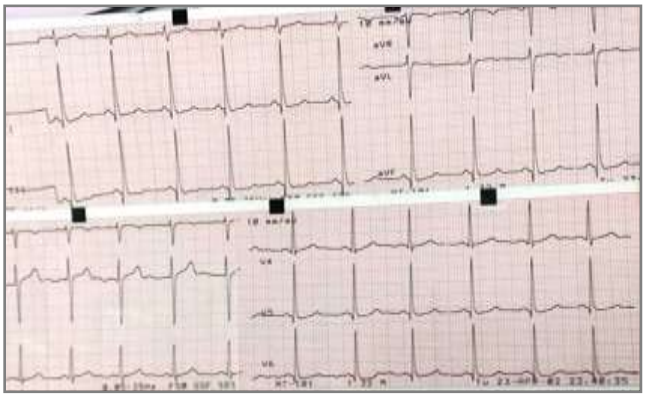

Figure-1: ECG of the patient with QT prolongation (QTc-) 0.550 sec) in this case.

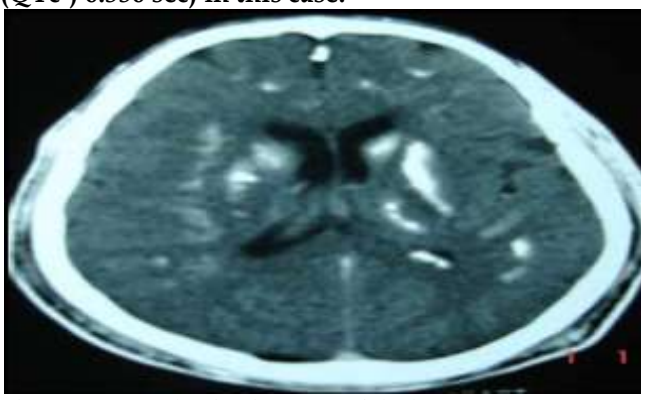

Figure-2: CT brain image with hyper dense areas (calcification) in bilateral caudate nuclei, thalami and cerebellar lobe.

no scar marks on neck, and no lymphadenopathy. There was no abnormal pigmentation on mucous membranes, skin or palmer creases. Keeping in view history of episodic stiffening of hands, testing for Trousseau's sign was performed. Twenty secs into the test, patient experienced clear carpopedal spasm. Detailed examination did not reveal perioral twitching; no changes suggestive of cataract could be appreciated on torch examination. Auscultation of chest and precordium did not reveal any abnormal signs. There was no organomegally in abdominal examination and no mass was palpable on bimanual palpation. Nervous system examination was non-focal.

Electrocardiogram was performed which showed QT interval prolongation with QTc of 0.550 sec (fig-1). Baseline hematology investigations were WNL. Biochemistry evaluation revealed low serum calcium and high phosphate

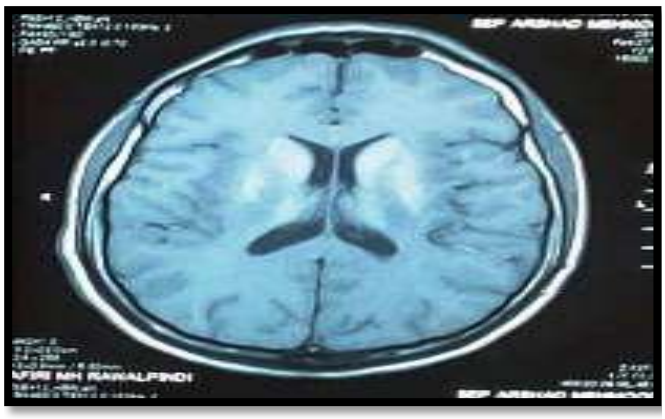

Figure-3: MRI brain (T1-image) with hyper intense signals (calcification) in caudate nucleus and thalami bilaterally.

(table). Findings were reconfirmed on repeat sampling. Corrected calcium was also low. Twenty four hours urinary calcium excretion was

Table: Serum calcium, phosphate, iPTH level and 24 hours urinary calcium of the patient.

Test	Ref range	Value in this case
Serum Calcium	2.15-2.60 mmol/L	1.89 mmol/L
Serum phosphate	0.81-1.68 mmol/L	2.54 mmol/L
Serum Magnesium	0.65-1.05 mmol/L	0.75 mmol/L
24 hours urinary calcium	1.25-6.0 mmol/day	0.9mmol/day
Serum iPTH	0.8-6 pmol/L	0.32 pmol/L

also performed and found below normal range. Comprehensive workup was carried out for other endocrine deficiencies and autoimmune pathologies including thyroid profile, antithyroid microsomal antibodies, gastric parietal cell antibodies, serum ANA, anti-ENA antibodies and no abnormality was detected. Virology screen including HBV, HCV and HIV serology was normal.

No abnormal waveforms were detected on EEG. No masses were visible on Ultrasonography of neck and abdomen. Cross-sectional imaging (contrast enhanced) of the chest, abdomen and pelvis were normal. Neuroimaging including CT scan and CEMRI of brain was performed. Bilateral extensive hyperdense foci of calcification were found on neuroimaging involving frontal lobes, bilateral thalami, caudate nucleus and cerebellar lobes (fig-2 & 3). To clinch the diagnosis patient was shifted to central referral hospital Rawalpindi for intact PTH level which were performed and was found low (table).

Based on clinical, lab and imaging data a diagnosis of primary hypoparathyroidism was established. A formal counseling session was performed. Patient was briefed about the diagnosis, treatment modalities and follow up plan every 3 months. He was commenced on calcium and vitamin D supplements for lifelong along with oral levitiracetam for seizure control. Patient responded to treatment, he remained fit free and his carpopedal spasms improved.

DISCUSSION

The prevalence of seizures in primary hypoparathyroidism is 45% and 29% of them seizure is the sole manifestation of hypocalcaemia[6]. Most commonly tetanic contractions in hands (Carpopedal spasms), perioral spasms are the usual effects of hypocalcaemia on the human body. A neuroimaging study should be performed on all patients presenting with first seizure. Non contrast CT scan brain performed in case of first seizure episode presenting to the emergency department changed the management plan in 9% to 17% adults in a systemic review[7]. The classic presentation of tetanic muscular contractions, Trousseau's sign and Chvostek's sign are due to muscular hyperexcitability when the serum ionized calcium falls below 1.1 mmol/L. In our case patient presented with seizures (GTCs). Trousseau's sign was elicited although Chvostek's sign could not be appreciated in this case. Hypocalcaemia characteristically causes prolongation of QT interval in electrocardiogram, a finding present in our case[8,9]. The two common causes of hypocalcaemia with low PTH are surgery of thyroid (80%) and autoimmune hypoparathyroidism (20%)[10]. Primary hypoparathyroid disorder is rare. In view of the fact that hypoparathyroidism is a hormonal deficiency, replacement of parathyroid hormone is a potentially attractive intervention[11], especially for those who cannot maintain stable serum calcium with calcium and vitamin D supplementation. In our case patient was commenced on oral calcium and Vitamin D supplements as the first line therapy to which he responded.

CONCLUSION

Primary hypoparathyroidism is a rare disease and its presentation with GTCs as a first symptom is even rarer. Clinicians should keep a high index of suspicion for primary hypoparathyroidism in case who present with hypocalcaemia related seizures.

CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

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