

# Status Epilepticus in Idiopathic Hypoparathyroidism

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## ABSTRACT

Acute hypocalcaemia which is seen in critically ill patients or which is caused by certain medications usually does not require specific treatment. But chronic hypocalcaemia which is caused by hypoparathyroidism is usually a symptomatic and requires treatment. A 50-years-old female presented in status epilepticus. She had muscle spasms, carpopedal spasms, facial grimacing and papillo-oedema. Her serum calcium and PTH levels were markedly decreased and her serum magnesium levels were normal. The CT scan of her head revealed calcifications in the

brainstem, cerebellum, basal ganglia and the corona radiata and her ECG showed a prolonged QT interval. A diagnosis of chronic hypocalcaemia with hypoparathyroidism was made. The patient gradually responded to calcium infusions, anti-convulsants and supportive treatment. She was discharged in a satisfactory condition after 20 days on oral calcium supplementations and anti-convulsants. This case highlights the importance of the parathyroid hormone and calcium measurements in patients who present in status epilepticus.

**Key Words:** Hypoparathyroidism, Hypocalcaemia, Status epilepticus

## INTRODUCTION

Hypoparathyroidism presenting as status epilepticus is a rare but treatable entity. The parathyroid hormone helps in regulating the calcium and phosphorus levels in the blood. PTH response failure can occur if there is parathyroid gland failure (hereditary or acquired), if PTH is ineffective in the target organs or if the action of PTH is overwhelmed by the loss of calcium [1]. However, irrespective of the cause, the clinical presentation of chronic hypocalcaemia can vary from muscle spasms to carpopedal spasms to a raised intracranial pressure and papillo-oedema. Long standing hypocalcaemia which is associated with hyper phosphataemia (observed with PTH deficiency or resistance ) can lead to calcification of the basal ganglia and occasional extrapyramidal disorders [2]. Acute, rather than chronic hypocalcaemia, is seen in critically ill patients or as a consequence of certain medications and it often does not require specific treatment [1]. Here, a case of chronic hypocalcaemia which was caused by hypoparathyroidism, which presented in status epilepticus, has been presented.

## CASE REPORT

A 50-year-old post-menopausal female was admitted to the emergency department in status epilepticus. There was a history of muscle spasms and tonic movements of the body parts for 10 days, with generalized convulsions for 3 days, with uprolling of the eyeballs and urinary incontinence. The patient's body was in a tense and rigid state for 7 days, with a tonic extension of the neck. There was no h/o fever, cough, bowel problem or trauma. The patient had similar problems in the form of spasms of the hand muscles with extension of the neck and the whole body since 15-18 years, for which she used to take medications, but she was not relieved. There was no h/o any surgery, irradiation, drug intake, especially anti-convulsants, diuretics and iron supplements or any other chronic ailment. On examination, the patient was found to be unconscious, with hypotension. She had muscle spasms, dry

skin and generalized seizures. The rest of the general physical examination was unremarkable. Chvostek's sign was negative. Trousseau' sign was positive. Her systemic examination was within normal limits. There was no focal neurological deficit and no sign of meningeal irritation.

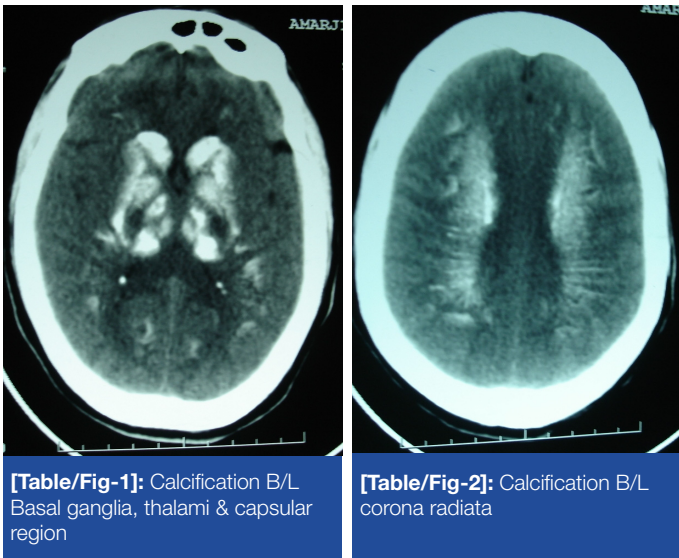
## INVESTIGATIONS

Her complete blood count, renal functions, electrolytes, blood sugar and urine examination were normal. Her serum calcium level was markedly decreased -2.5mg/dl. Her serum magnesium level was just below the normal -1.80mg/dl. Her serum phosphorus level was raised -9.10 mg/dl. Her PTH level was decreased -4.54 pg/ml. Her serum albumin, serum iron , serum ferritin and serum transferrin saturation levels were within normal limits. Her ECG showed a prolonged QT interval. USG of her abdomen was normal.

CT scan of her head showed calcifications in the corona radiata, basal ganglia, brainstem and the cerebellar hemispheres [Table/ Fig-1, 2].

## DISCUSSION

Status epilepticus is a rare presentation of hypoparathyroidism. The parathyroid hormone controls the minute to minute levels of ionized calcium in the blood and the extracellular fluids. Calcium is the primary element that helps in muscle contraction, nerve conduction, bone rigidity and tooth development. Various congenital or acquired disorders can lead to developmental failure of the parathyroid glands, failure of functional hormone production, or destruction of the glands [3]. Sometimes, the action of PTH is overwhelmed by calcium loss like tumour lysis, acute renal failure and rhabdomyolysis. Severe hypomagnesaemia is also associated with hypocalcaemia. The effects of magnesium on the PTH secretion are similar to those of calcium, but the calcium effects dominate [1]. Surprisingly, severe hypomagnesaemia is associated



**[Table/Fig-1]:** Calcification B/L Basal ganglia, thalami & capsular region

**[Table/Fig-2]:** Calcification B/L corona radiata

with blunted PTH secretion because of the intracellular magnesium deficiency which interferes with the secretion and the peripheral response to PTH. Moreover, the serum phosphate levels are often not elevated in hypomagnesaemia, in contrast to the acquired or idiopathic hypoparathyroidism. The predominant clinical features of hypocalcaemia are neuromuscular irritability, tingling of the fingers and toes and spontaneous or latent tetany. In profound hypocalcaemia, grandmal seizures or laryngospasms may be observed. Long standing hypocalcaemia which is associated with hyperphosphataemia (PTH deficiency or resistance) can lead to calcification of the basal ganglia and occasional extrapyramidal disorders [2]. The complications of hypoparathyroidism may include cataracts [4], intra-cranial calcifications, growth stunting, mental retardation [5] and cardiomyopathy [6]. Hypocalcaemia which is associated with parathyroid dysfunction can be differentiated from other causes of hypocalcaemia by routine lab tests [2]. The serum calcium levels are low, owing to the lack of PTH mediated bone resorption and urinary calcium reabsorption. The serum phosphate

levels are increased, owing to the impaired renal clearance. The abnormal calcium, phosphorus and PTH levels differentiate this condition from Fahr's syndrome, which is an idiopathic basal ganglion calcification [7]. Severe symptomatic hypocalcaemia constitutes an emergency that requires immediate attention to prevent seizures and death from laryngospasms or cardiac causes. Death due to hypoparathyroidism is rare, but it has been reported and it is caused by complications rather than directly because of the disease.

## CONCLUSIONS

Hypoparathyroidism leading to hypocalcaemia, although it is an uncommon cause of status epilepticus, it is an important treatable and reversible condition, as it was in this case. Therefore, the routine measurement of the serum calcium and the PTH levels in these patients can be life saving.

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