Basal Ganglia and Cerebellar Calcification: Rare Finding in Hypoparathyroidism

Nisha Batra1*, Ravi Kant2
Received: 17 March 2020; Accepted: 24 September 2022

Case Description

A 45-year-old female presented with complaints of muscle cramps and generalized body aches. She had a doubtful history of one episode of seizure at home a few days back lasting for seconds to minutes, resolved spontaneously. On examination, her vitals were normal, the trousseau sign was positive but there was no evidence of any tremors or choreiform movements, and systemic examination was unremarkable. The patient had a history of cataract surgery in both eyes at the age of 40 years. Investigations revealed hypocalcemia with serum calcium levels of 5.3 mg/dL with high phosphate levels of 7.6 mg/dL. Her ionized calcium was also low, 0.5 mmol/l (normal range 1.13–1.32 mmol/l) and she had low intact parathyroid hormone (PTH) levels of 5.4 pg/mL. CT Head was done which showed bilateral symmetrical basal ganglia and cerebellar calcifications (Figs 1 and 2). The patient was diagnosed with hypoparathyroidism and was evaluated for the etiology of the same. She had no previous history of goiter or neck surgery. There was no history of jaundice, arthralgia, any coexistent autoimmune disorder, skin or dental abnormalities, tremors, or hyperpigmentation. Biochemical workup did not reveal any etiology and hence, a diagnosis of idiopathic hypoparathyroidism was made. She was started on calcium and calcitriol therapy with urinary calcium monitoring. The patient improved symptomatically within a duration of 2 weeks with no more evident tetany or muscle cramps.

Discussion

Parathyroid hormone (PTH) plays a vital role in the maintenance of calcium homeostasis. Primary Hyponoparathyroidism is defined by an abnormally low level of PTH, leading to hypocalcemia and hyperphosphatemia.

Basal ganglia calcification (BGC) is a nonspecific finding, reported in 1% of computed tomography (CT) head scans done due to any cause.1 It can be physiological or pathological. Physiological BGC is usually an incidental asymptomatic finding in elderly patients while pathological calcifications may lead to tremors, extrapyramidal symptoms, or cognitive decline in affected patients.1 Two most common causes of pathological BGC include Primary hypoparathyroidism and pseudo-hypoparathyroidism.2 Other reported causes are—sarcoidosis, malignancies, certain infections including HIV, tuberculosis, birth asphyxia, carbon monoxide toxicity, lead exposure, radiation therapy, and certain inherited neurodegenerative disorders. The mechanism of intracranial calcification in Primary hypoparathyroidism is not completely understood. Altered calcium phosphate products could be one of the causes, however, it has failed to explain all manifestations. Intracranial calcification is found to be related more to the duration of hypocalcemia and hyperphosphatemia rather than the level of PTH. Basal ganglia is the most commonly affected area, however, certain other areas in the brain are involved too including cerebellum, grey-white junctions, thalamus, and dentate nuclei.3,4 These calcifications have been reported in 0.3–1.5% of patients with hypoparathyroidism, and are often detected incidentally,5,6 but are significantly associated with cognitive decline, cerebellar dysfunction, and parkinsonism in affected patients. An adequate treatment maintaining the calcium and phosphate homeostasis to near normal levels may slow the progression of intracranial calcification and lead to remarkable improvement in neurological manifestations.

References


1Senior Resident; 2Additional Professor, Department of General Medicine, All India Institute of Medical Sciences Rishikesh, Uttarakhand, India; *Corresponding Author

How to cite this article: Batra N, Ravi Kant. Basal Ganglia and Cerebellar Calcification: Rare Finding in Hypoparathyroidism. J Assoc Physicians India 2023;71(2):93–99.