

# Extensive Intracranial Calcification in a Case of Hypoparathyroidism: Case Report

Mohamad Reza Ehsaei <sup>1\*</sup>, Mohamad Ali Abouei Mehrizi <sup>1</sup>, Farimah Firouz <sup>2</sup>

<sup>1</sup> MD, Department of Neurosurgery, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran

<sup>2</sup> MD, Department of Neurology, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran

\* Corresponding Author Address: Shahid Kamyab Hospital, 17 Shahrivar Street, Mashhad, Iran. Telefax: +989151155803. Email: ehsaeemr@mums.ac.ir

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## Abstract

**Background and Importance:** Hypoparathyroidism is due to parathyroid hormone deficiency and categorized as an endocrine disorder. Acute clinical presentations of hypoparathyroidism are muscle cramps and spasms, tetany, weakness, paresthesia and seizure. Hypoparathyroidism may be accompanied with psychosis, depression, seizures and extrapyramidal manifestations in chronic condition.

**Case Presentation:** The present case reported about a 37-year-old man who presented with the history of several episodes of seizures, slurred speech, progressive limbs and trunk stiffness and increased muscle tone and rigidity.

**Conclusion:** Paraclinical investigations revealed hypocalcemia and very low parathyroid hormone levels with extensive intracranial calcification involving bilateral basal ganglia, cerebellum and subcortical white matters on brain CT scan.

**Keywords:** Hypoparathyroidism; Parathyroid Hormone; Intracranial Calcification

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## Background and Importance

Intracranial calcification has physiological or pathological underlying causes. Common age-related and physiologic patterns are calcifications of the pineal gland, habenula, choroid plexus, falx, dura mater, intracranial artery atherosclerosis and giant arachnoid granulation. Congenital disorders such as tuberous sclerosis, neurofibromatosis type 2, vascular calcifications due to aneurysm and capillary telangiectasia, congenital infections, tumors, idiopathic, metabolic and endocrine abnormalities are considered as a differential diagnosis for pathological calcification [1].

The basal ganglia is a collection of caudate nucleus, putamen, and globus pallidus anatomically situated at the base of the forebrain, which is mostly involved in extensive intracranial calcification [1]. Functionally, these subcortical nuclei affect daily activities including motor control and coordination of movement, cognitive and affective control. Therefore, the most expected symptoms of basal ganglia dysfunctions due to calcification are tremors and movement abnormalities, and psychiatric symptoms, including mania and psychosis [2-8].

## Case Presentation

A 37-year-old man was referred to the neurology clinic for evaluation of progressive limb and trunk stiffness and slowing movement for 3-4 years. He has experienced several episodes of seizure since seven years of age. He is currently on anticonvulsant medication, and his last episode of seizure happened two years ago. The patient was prescribed phenytoin, phenobarbital, tetrabenazine, and Tegretol tablets by a neurologist. For four years to now, he has been gradually experiencing the progressive slight trunk stiffness and slowing movement. Additionally, he developed slurred speech and disarticulation. He finally lost his job due to his disability. His medical history was normal, and no medical problem with his parents or near relatives was reported. Testing the patient's orientation and insight, memory and intellectual function showed normal high cortical function. The comprehensive cranial nerve assessment revealed no abnormal sign. All senses including touch, vibration, pressure, temperature and pain had normal function. No sensory level was detected. While testing his motor system, he had increased muscle tone and rigidity. He had also experienced hypokinesia and bradykinesia. Despite of no tremor, his hand movement had mild dystonia.

The strength of muscles was normal. His deep tendon reflexes showed generally hyporeflexia. There was no Babinski reflex. Moreover, on head and neck physical examination, positive Chvostek's sign was detected. The pupils were equally mid-sized and reactive to light. Eyes had normal movement in all directions, and no papilledema was found on fundoscopic examination.

On admission, he gave informed written consent to participate in the study, and all routine chemical and hematological investigations were performed. Reviewing the results of the CBC, the hemoglobin was 12.2 g/dl, and total leukocyte count 6300/ $\mu$ L. The blood sugar was 90 mg/dl, blood urea 36 mg/dl and serum creatinine 1.3 mg/dl (NI range: 0.5-1.5). There was no abnormality in serum electrolytes. His serum albumin was 4.4 g/dl. Serum electrolyte analysis showed sodium 139 mEq/l (NI range: 135-145 mEq/l), potassium 3 mEq/l (NI range: 3.5-5.3 mEq/l), calcium 6.6 mg/dl (NI range: 8.0-10.4 mg/dl), serum phosphate 4.2 mg/dl (NI range: 2.5-4.5 mg/dl), and serum magnesium was 1.8 mg/dl (NI range: 1.3-2.5 mg/dl). Serum trace elements levels of iron and copper were 131 mcg/dl and 129 mcg/dl, respectively. The patient had a serum parathyroid hormone (PTH) level less than 5pg/ml (NI range: 9-55 pg/ml). All amounts of thyroid hormones and TSH were normal. Other laboratory investigations on the measurement of serum 25-OH- Vit D, anti TTG and urine calcium levels demonstrated 44 ng/dl (NI range: 20-40ng/dl), 3.9 U/ml (NI range < 6.0U/ml) and 10.8 mg/dl (NI range: 20-300 mg/dl), respectively.

EEG showed normal pattern without any abnormality. Computed tomography of the brain revealed extensive calcifications in the bilateral basal ganglia components involving caudate nucleus, putamen, globus pallidus and dentate nuclei. Bilaterally symmetrical calcifications in white matter of cerebellar hemispheres and cerebral subcortical areas in frontal and parietal lobes were also observed (Figures 1-3). In this case, based on clinical signs and symptoms and paraclinical laboratories data and imaging, parkinsonism due to untreated hypoparathyroidism was presumed as an appropriate explanation. His episodes of seizure and positive Chvostek sign were also clinical signs of existing nerve hyperexcitability as a result of hypocalcemia. Calcium and vitamin D supplementation are the essential components of parathyroid insufficiency treatment.

## Conclusion

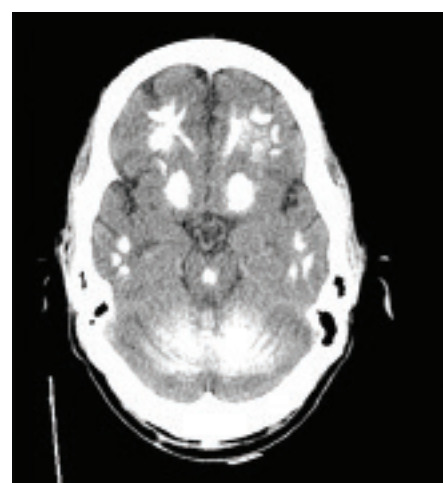
Hypoparathyroidism is an endocrine disorder characterized as low level of PTH leading to hypocalcemia. It can result from surgical damage to the parathyroid glands during thyroidectomy, kind of hereditary or familial hypoparathyroidism, autoimmune invasion as a part of autoimmune polyglandular syndrome (APS), as a complication due to radiation treatment for cancer in head or neck region, abnormal level of magnesium and iron, and among others. Hypoparathyroidism is mostly presented with muscle cramps and spasms, tetany, weakness, paresthesia, and seizure due to low ionized calcium concentration. Other clinical symptoms include patchy hair loss, dry skin, headaches, depression and mood swings, cataract and painful menstruation. According to the literature, patients with hypoparathyroidism may develop neurological signs and symptoms including psychosis,



**Figure 1. CT Scan Axial View: Extensive Bilateral Calcifications in the Periventricular White Matter Involving the Caudate Nucleus, Putamen, Globus Pallidus, Frontal and Parietal Lobes**



**Figure 2. CT Scan Axial View: Extensive Bilateral Calcifications in the Periventricular White Matter Involving the Thalamus, Putamen, Globus Pallidus and Peripheral Cerebral Subcortical Areas**



**Figure 3. CT Scan Axial View: Calcifications in Frontal and Temporal Lobes and Both Cerebellar Hemispheres**

depression, seizures and extrapyramidal manifestations [7, 9-13].

Basal ganglia calcification is usually a silent accidental finding in 1% of all CT scans [14]. It was initially described in 1855 [15], and further studies approved the possible association between basal ganglia calcification and abnormal calcium-phosphate metabolism disorders including hypoparathyroidism or pseudohypoparathyroidism [16-20] which are currently known as the most common cause of pathological basal ganglia calcification [3,8]. Interestingly, hypoparathyroidism, regardless of any cause, with extensive intracranial calcification is a rare presentation [11].

Neuronal excitotoxicity due to intracellular calcium precipitation in neurons' changes and following vascular insufficiency and inflammation accounted for idiopathic basal ganglia calcification [21]. Besides, increased calcium-phosphorus ratio diminished the development of basal ganglia calcification implicating the importance of treatment [3].

To conclude, though basal ganglia calcifications are usually nonspecific accidental findings, hormonal disturbance such as hypoparathyroidism, and pseudohypoparathyroidism should be investigated by clinicians as a main pathological cause of basal ganglia calcifications particularly in symptomatic patients.

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## Conflicts of Interest

The authors declare that they have no conflicts of interest.

## Author's Contribution

**Conception and Design:** All authors

**Data Collection:** All authors

**Drafting the Article:** All authors

**Critically Revising the Article:** All authors

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## Comments

This article is a case report of intracranial calcification due to hypoparathyroidism. Idiopathic hypoparathyroidism is uncommon condition. A percentage of these patients reveals basal ganglia calcification. However, intracranial calcifications outside the basal ganglia are rare. As in this case, physical signs of hypocalcemia is an important clue for diagnosis. The authors did not mention about the ophthalmologic examination, but slit lamp examination may reveal posterior subcapsular lenticular opacities in eyes.

The mechanism of this finding in hypoparathyroidism has not been elucidated completely, but it is related to the duration of hypocalcaemia and hyperphosphatemia.

The differential diagnoses should be considered in each case of intracranial calcification such as Fahr's Syndrome, anoxia, lead poisoning, carbon monoxide intoxication, radiation therapy, methotrexate therapy, etc.

The authors have not stated anything about the outcome of the patient but generally this disease has a good prognosis if detected early and treated.

*Amir Azarhomayoun, MD*

*Department of Neurosurgery, Sina Hospital, Tehran University of Medical Sciences, Tehran, Iran*