

Basal Ganglia Calcification and Parkinsonism Symptoms Due to Secondary Hypoparathyroidism

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Abstract

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Basal Ganglia Calcification (BGC) is a rare radiological finding often associated with various neurological disorders, including Parkinsonism. Secondary hypoparathyroidism, characterized by insufficient Parathyroid Hormone (PTH) production, can lead to calcium imbalance and subsequent deposition in the basal ganglia, contributing to neurodegenerative symptoms. This condition presents with Parkinsonism features such as bradykinesia, rigidity and tremors. The interplay between low PTH levels and disturbed calcium homeostasis plays a key role in the pathogenesis of BGC and its neurological consequences. Early recognition and management of hypoparathyroidism are crucial to prevent further progression of Parkinsonian symptoms associated with basal ganglia calcification.

Keywords: Fahr's disease; Basal ganglia calcification; Hyperparathyroidism; Parkinsonism

Introduction

Basal ganglia calcification, also known as Fahr's disease, is a rare neurological disorder characterized by the abnormal accumulation of calcium deposits in the basal ganglia, which are a group of nuclei located deep within the brain. The basal ganglia play a crucial role in various motor and non-motor functions, including movement regulation, cognition and emotion. The clinical features are: Parkinsonism, ataxia, headache, seizures, vertigo, stroke-like events orthostatic hypotension, tremor, dysarthria and paresis [1].

Secondary Parkinsonism refers to a set of symptoms similar to those of Parkinson's disease but caused by factors outside the typical degenerative process that leads to Parkinson's. Basal ganglia calcification is a condition that can result in secondary Parkinsonism [2].

Secondary hypoparathyroidism is a medical condition characterized by insufficient secretion or action of Parathyroid Hormone (PTH) due to factors external to the parathyroid glands [3].

Basal ganglia calcifications and Parkinsonian symptoms secondary to secondary hypoparathyroidism are rare but can occur in some cases.

Method

In this study, the relationship between basal ganglia calcification in a 72-year-old patient with secondary Parkinsonism and secondary hypoparathyroid symptoms was investigated. Our research shows that although the combination of these diseases is rare, it is not impossible. The case studied by us had a history of thyroidectomy due to nodular goitre without effective treatment, which was seriously investigated with the onset of Parkinson's symptoms and seizures. The purpose of this report is to increase the awareness of physicians, prompt timely diagnosis and inform appropriate management strategies for such unusual manifestations.

Case Presentation

Our case was a 72-year-old woman with a history of thyroidectomy due to nodular goiter was treated with levothyroxine for 15 years ago. The patient has not had regular medical visits. She has had Parkinson's movements for 5 years ago. She has also had paresthesias around her mouth and hands for 1 year ago, which have not been investigated. The patient went to the emergency room due to decreased consciousness and convulsive movements and was admitted to the hospital because of profound hypocalcemia. The tests

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performed for the patient were as described in Table 1. The patient was diagnosed with iatrogenic hypoparathyroidism and in the brain CT (Figure 1), she had basal ganglia

calcification, which was the cause of Parkinson's disease in this patient.

Table 1: Laboratory parameters of the patient.

Test	Result	Normal range	Unit
Ca	304	8.5-10.5	mg/dL
ph	9.7	2.6-4.5	mg/dL
Ionized-Ca	0.3	1.05-1.3	mmol/dL
PTH	10	-	-

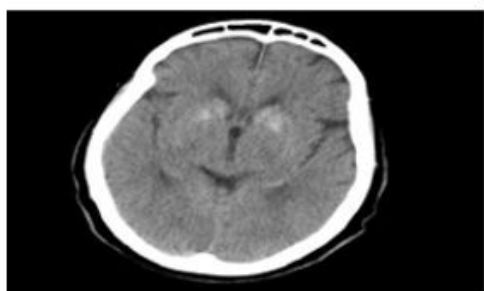


Figure 1: Brain CT.

Results and Discussion

The diagnosis of Fahr's disease is based on the presence of basal ganglia calcification along with normal levels of serum calcium and phosphorus [4]. It is necessary to pay attention to this point that Fahr's disease must be distinguished from Fahr's syndrome, in which there are specific causes for the calcification of meningeal nodules (such as hypoparathyroidism) and also from the radiological classification of nodules without clinical symptoms. The specific cause, which is an accidental finding. It is seen in 0.7 to 0.9 percent of cases and is seen mainly in people over 60 years old [5]. Although it generally seems to be an idiopathic disease, recently, a relationship with the long arm of chromosome 14 has been found in a family with several affected members. Dilip Sapkota, et al. reported a case of 61-year-old female patient who initially presenting with a slow walking pace two years prior, her condition evolved to include right-hand tremors, affecting her ability to perform daily tasks. Six months before seeking medical advice, she noted deteriorating speech quality slow and slurred-compounded by swallowing difficulties with solid foods, leading to choking episodes [6]. They reported case of late-onset Fahr's disease accompanied by calcification of the basal ganglia has highlighted the importance of considering Fahr's disease in the differential diagnosis for elderly patients presenting with cognitive deficits reminiscent of Alzheimer's disease and movement abnormalities akin to Parkinson's disease.

It is possible that tissue damage due to free radicals or abnormal iron transfer played a role in igniting the calcification process of basal ganglia [7]. The onset of the disease at the age of 20-40 years is accompanied by

schizophrenic psychoses and catatonic symptoms and the onset between the ages of 40-60 years is associated with dementia and arthritis. Its most common neurocognitive symptoms are parkinsonism, chorea, dystonia, tremor, walking disorder, dysarthria, convulsions and myoclonus and the pattern of cognitive impairment found in it is Frontal-subcortical type [8]. Depression has also been relatively common, but mania secondary to Fahr's disease has been reported very rarely. This hypothesis that there is damage to the limbic system, menstrual complexes and hypothalamus in mood disorders is also confirmed by the symptoms of this disorder. According to the findings of biological researchers, the limbic apparatus and the menses and hypothalamus glands are closely related. Timely identification of this condition plays a pivotal role in the selection of appropriate therapeutic interventions and in prognosticating the outcomes of related disorders.

Also, individuals with hyperparathyroidism may have various neurological symptoms similar to those of Parkinsonism, such as muscle weakness, reduced mobility and tremors, caused by changes in calcium levels impacting neuromuscular function.

Diagnosing Fahr's disease alongside hyperparathyroidism requires thorough evaluation. Treating hyperparathyroidism to address the calcium imbalance may help slow down the growth of calcification and related neurological symptoms.

Conclusion

Overall, the complicated relationships between hyperparathyroidism, Fahr's disease and Parkinsonism emphasize the challenges in detecting and treating illnesses that include both endocrine and neurology. Hyperparathyroidism's disruption of calcium and phosphate balance may greatly affect the development of neurological disorders including Fahr's disease and Parkinsonism, emphasizing the need of a thorough diagnostic strategy. To effectively treat these diseases, a multidisciplinary approach is needed to address both the underlying endocrine abnormalities and the accompanying neurological symptoms. Hyperparathyroidism may worsen or imitate the symptoms of Fahr's disease and Parkinsonism, highlighting the need for consistent monitoring and prompt action. Understanding the connections between these problems may result in more precise diagnoses, customized therapies and better results for

people dealing with both endocrine and neurological disorders.

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Author Contributions

All authors approved the final manuscript.

Competing Interests

The authors declare that they have no competing interests.

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Availability of Data and Material

Considering that this study is about rare disease availability to patients and data may hard to some extent.

Written informed permission from the patient to publish the facts and photographs was granted.

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