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WHEN THE BRAIN TURNS TO BONE: SECONDARY FAHR'S SYNDROME AS A LATE COMPLICATION OF HYPOPARATHYROIDISM

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INTRODUCTION

Fahr's syndrome is a rare neurological disorder that affects fewer than 1 in 100,000 people, characterized by bilateral intracranial calcifications. While it is often idiopathic or genetic in origin, secondary forms, particularly due to chronic hypoparathyroidism, are well-recognized but frequently underdiagnosed.

CASE DESCRIPTION

A 50-year-old lady with history of total thyroidectomy presented with a 2-day history of persistent throbbing headache. She denied any blurriness of vision, body weakness, and seizure. Her vitals were normal, and the systemic examination revealed no neurological deficit. The headache, however, was not fully resolved with analgesic. A non-contrast CT brain showed the presence of bilateral calcifications of the basal ganglia comprising the corona radiata, heads of caudate nuclei, and bilateral lentiform nucleus that raised the diagnosis of secondary Fahr's Syndrome. This patient responded well with oral calcium supplements and was scheduled for long-term follow-up to monitor his calcium and parathyroid hormone levels.

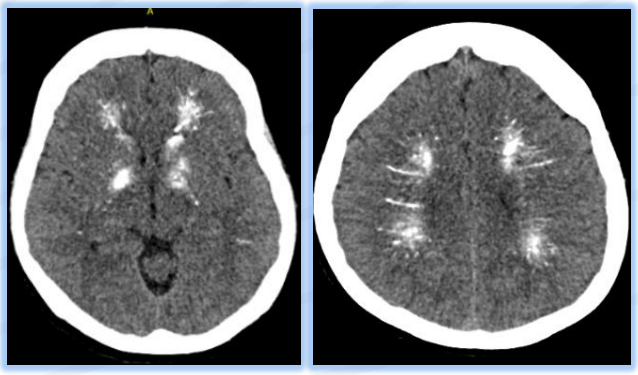
DISCUSSION

Secondary Fahr syndrome is diagnosed when bilateral intracranial calcifications, especially in the basal ganglia and dentate nuclei, occur in association with a known cause. The most common causes are metabolic disorders like in hypoparathyroidism as reported in this case, alongside infection and autoimmune. Presentation may vary from asymptomatic to simple headache, movement disorder, seizure, and even psychiatric symptoms have been reported in extensive calcification. The gold standard for diagnosis is a non-contrast CT brain. Treatment will focus on underlying causes and alleviating the symptoms.

From this case, we emphasize long-term calcium, phosphate, and parathyroid hormone monitoring post-thyroid surgery. The absence of regular follow-up will lead to prolonged metabolic imbalance, eventually contributing to the gradual deposition of calcium in the basal ganglia and other brain structures, causing Fahr's syndrome.

CONCLUSION

Secondary Fahr's Syndrome should become the differential diagnosis for post-total thyroidectomy patients presenting with central and neurological symptoms. Routine long-term endocrine follow-up post-surgery is the key to prevention of this syndrome.



Keywords: Fahr's Syndrome, Hypoparathyroidism

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