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Hypoparathyroidism Presenting as Movement Disorder and Seizure in a Case of Post- Surgical Hypothyroidism

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Abstract **Introduction**: Recurrent laryngeal nerve injury and hypoparathyroidism are common complications of thyroid surgery. Hypoparathyroidism can cause carpopedal spasm, paresthesia, seizures and rarely movement disorders along with extensive intracranial calcification and many patients receive antiepileptic drugs for seizures without proper metabolic evaluation. **Case:** We report a 42 year old Indian female presenting with persistent ataxic gait with moderate truncal ataxia along with dyskinetic movements in both hands and slurred speech. She was operated for multinodular goiter (MNG) 21 years back – developed extensive intracranial calcification (diagnosed as Fahr's Disease) and was put on Phenytoin therapy for repeated seizures about one year but seizures remain uncontrolled. Investigations revealed euthyroid state (with thyroxin replacement) along with features of hypoparathyroidism. She was treated accordingly which resulted in improvement of movement disorders quickly and seizure were also controlled. Phenytoin was successfully withdrawn with no further seizure. **Discussion**: Acquired hypoparathyroidism (Post-thyroid surgery here) is a common cause of intracranial calcifications (including Basal ganglia) which causes seizures and rarely movement disorders. This case was wrongly diagnosed as Fahr's syndrome. All her symptoms improved after proper metabolic correction. Hence the importance of proper metabolic assessment in cases of seizures and movement disorders is emphasized here. **Conclusions**: Metabolic abnormalities should always be evaluated in patients with seizure disorder, especially if there is history of thyroid surgery and metabolic correction is more important than anticonvulsant medication and it improves almost all neurological problems including the movement disturbances and cerebellar ataxia.

Keywords: *thyroidectomy, hypoparathyroidism, hypomagnesemia*

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1. Introduction

Total thyroidectomy is considered the procedure of choice for benign multinodular goiter whenever possible. [1] Continuous developments in surgical techniques and better understanding of thyroid anatomy have increased the safety of thyroid surgery and reduced the incidence of complications. Nevertheless, surgery for goiter embodies a unique challenge as recurrent laryngeal nerve injury and hypoparathyroidism are most common complications after thyroid surgery. [2] Hypoparathyroidism can cause extensive intracranial calcification. [3] Carpopedal spasm, paresthesia and seizures are the common presenting manifestations. [4] Because of high incidence of seizure disorder due to hypocalcemia, many patients receive various antiepileptic drugs including phenytoin. [4] Rarely calcification of basal ganglia and cerebellum is associated with various movement disorders and ataxia. Here, we report a case of 42-year old Indian female who was on phenytoin therapy for uncontrolled seizures and also was having stance and gait ataxia with dyskinetic movements of hands. She was subsequently found to have diffuse intracranial calcifications from secondary hypoparathyroidism due to prior thyroid surgery.

2. Case

A 42-year old *Indian woman presented in the hospital outpatient clinic with persistent gait disturbance and slurred speech for the last 1 month and tingling sensation all over body for last 2 months but without any paresthesias or tingling sensation in and around the mouth and lips. She developed multinodular goiter (MNG) 20 years ago and was operated at the age of 21 years. Though not much old records were available but she distinctly remembered that thyroxin replacement (100ug daily) started since her surgery without any other symptoms. No FNAC or histopathological reports are available. She was stable with thyroxin replacement for a long time without much medical problem. However she started to have repeated episodes of generalized seizures since last 1 year. She was evaluated in a local hospital. Her EEG was nonspecific with few sharp waves in the vertex, and CT

scan of brain showed extensive calcification in cerebellum, both basal ganglia and paraventricular regions. She was diagnosed as Fahr's disease and was put on phenytoin 300mg daily. Her convulsive episodes improved with phenytoin therapy but were not yet controlled since her visit to our centre.

She had average built, 100/60 mm Hg of blood pressure, 50 kg in weight. She looked anxious and little restless. Her thyroid gland was not palpable and there was a visible thyroidectomy scar. She had positive Trousseau's sign, positive Chvostek's sign, ataxic gait with moderate truncal ataxia and dyskinetic movement in both hands. She had no pyramidal signs and her finger-nose test was normal as also other tests for coordination. So she had mostly features of midline cerebellar dysfunction. The baseline laboratory evaluations are depicted in Table 1. Serum phenytoin level was within the therapeutic range. The EMG NCV study of all four limbs was normal. She was diagnosed as a case of post-surgical Hypothyroidism (on thyroxin replacement), post-surgical hypoparathyroidism and vitamin D deficiency.

Table 1. Baseline Investigations

Test	Results	Normal Range
Haemoglobin, gm/dL	11.10	12-16
Craetinine, mg/dL	0.91	0-1.5
Erythrocyte Sedimentation Rate, mm	34	0-22
Total Leucocyte Count, (leucocytes/cumm)	8400	4000-11,000
Free thyroxine (FT4), ng/dL	1.26	0.9-1.6
Serum albumin, mg.dL	4	3.4-5.4
Thyroid stimulating hormone, µU/mL	3.53	0.4-3.8
Serum Calcium, mg/dL	4.55	8.4-10.5
Phosphatase, U/L	214	134-359
Albumin corrected serum calcium, mg/dL	4.55	1.1-1.35
Intact PTH, pg/mL	4.13	10-70
Serum Vitamin D, ng/mL	14.08	30-100

Her thyroxin replacement (100ug per day) continued as before, calcium and calcitriol was added in addition and was given one shot of intramuscular 6 lakh units of vitamin D. She had no further seizure on follow up after 3 months and phenytoin was successfully withdrawn over next 3 months. The movement disturbances resolved completely after correction of metabolic abnormalities and definitely before any resolution of basal ganglia calcification.

3. Discussion

Benign multinodular goiter is one of the most common endocrine surgical problems and the patient underwent total thyroidectomy 20 years back as the procedure of choice. [5] She received thyroid replacement but was never evaluated for hypocalcemia. When she developed repeated episodes of convulsions about a year back and CT scan of brain showed extensive calcification in cerebellum, both basal ganglia and paraventricular regions, she was diagnosed as Fahr's disease and was put on phenytoin. Fahr's disease or Idiopathic Basal Ganglia Calcification is a rare, genetically dominant, inherited neurological disorder characterized by abnormal deposits of calcium primarily in the basal ganglia and also in the cerebral cortex. [6] However, Fahr's disease is diagnosed in subjects with family history of similar problem and after ruling out all possible causes of intracranial calcification especially hypocalcemia associated with hypoparathyroidism. Long lag time from first reported symptom to diagnosis is well known in hypocalcemia and many patients (more than one-third) get various antiepileptic drugs including phenytoin for the seizure due to delay in diagnosis of hypoparathyroidism. [4] Though the childhood seizure is mostly treated with anticonvulsants therapy, [7] structural and metabolic abnormalities should always be looked in adult. Hypocalcemia from hypoparathyroidism is an important but often unrecognized cause of seizure disorder. [4] The common imaging abnormalities from hypoparathyroidism are the basal ganglia calcification followed by cerebral cortex and cerebellar calcification [4] and almost similar to intracranial calcifications seen in Fahr's disease. [6] Calcification of the basal ganglia may cause movement disturbance of parkinsonian and/or choreoathetoid type. [8] Movement disturbances in the form of choreoathetosis or paroxysmal dystonia in hypoparathyroidism with calcification in the basal ganglia & the thalamus is well known, [9] but cerebellar ataxia is rare. [10] Nevertheless both the conditions resolved completely with correction of metabolic abnormalities before any resolution of basal ganglia calcification.

4. Conclusion

Metabolic abnormalities should always be evaluated in patients with seizure disorder and movement disorders, especially if there is history of thyroid surgery. Anticonvulsant medications should be avoided in patients with hypoparathyroidism as convulsions can occur from the metabolic alterations. Metabolic correction is more important than anticonvulsant medication and improve almost all neurological problems including the movement disturbances and cerebellar ataxia.

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