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A Cases Report of 55-Year-Old Lady with Recurrent Seizure with History of Thyroidectomy

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Abstract

Permanent hypoparathyroidism is not uncommon following thyroidectomy. However, hypoparathyroidism can present with neurological features like seizures, paresthesia, depression, psychosis, extrapyramidal manifestations and features of raised intracranial pressure. Here we present a case of recurrent seizure with history of thyroidectomy 10 years back. The patient was clinically and biochemically hypocalcaemic due to hypoparathyroidism and her brain imaging shows diffuse intracranial calcification. Patient was managed with anticonvulsant, calcium gluconate, calcitriol and levothyroxine. Chronic hypocalcaemia due to postoperative hypoparathyroidism can cause diffuse intracranial calcification which in turn triggers several neurological symptoms like seizure. Therefore judicial surgery and regular post-operative follow up is required to prevent such complications. *[Journal of National Institute of Neurosciences Bangladesh, January 2022;8(1): 100-102]*

Keywords: hypocalcaemia; hypoparathyroidism; intracranial calcification

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Introduction

Thyroid surgery has some potential complications, of which hypoparathyroidism is an important one. After thyroidectomy, a person may experience hypoparathyroidism which may be transient or permanent with the incidence of 1.3% and 8.7% respectively[1]. The classical manifestation of hypocalcemia resulting from hypoparathyroidism is carpopedal spasm. However, clinical presentation may be influenced by severity and timing of serum calcium reduction (acute or chronic), patient's age, complications, and comorbidities[2]. Hypoparathyroidism can present with neurological features like seizures, paresthesias, depression, psychosis, extrapyramidal manifestations and features of raised intracranial pressure. A high calcium x phosphate product may cause ectopic calcifications and can have a role in epileptogenesis[3]. Here, a case of recurrent seizure is presented who had history of thyroidectomy.

Case Presentation

A 55-year-old lady presented to emergency department of National Institute of Neurosciences (NINS) & hospital with recurrent generalized tonic clonic seizure during previous 24 hours. Each episode of seizure existed for few minutes and was associated with tongue biting, urinary incontinence and post-ictal confusion. Her seizure was managed with intravenous

anticonvulsants in emergency and sent to in-patient department for further evaluation. In neurology ward, she was drowsy, irritable with normal vital signs. Systemic examination was unremarkable except for a scar in the neck and presence of positive Trousseau sign. Patient had history of nodular goiter and underwent thyroidectomy 10 years back. The exact indication, procedure, histopathology and post-operative complications could not be well evaluated as patient had lost all the documents related to surgery. However, she experienced occasional twitching in face and spasm in hands after surgery and levothyroxine replacement along with calcium and vitamin D supplement was prescribed. Patient was irregular in follow up and compliance to medication was poor. Investigations revealed low serum total calcium (1.57 mmol/L), high phosphorus (6.8 mg/dl) and low intact parathyroid hormone (iPTH 2.3 pg/ml); whereas magnesium (0.75 mmol/L) and albumin (40gm/L) was normal. Thyroid function showed elevated TSH (31.54 uIU/ml) and low fT4 (0.78 ng/dl) (Table 1).

Computed tomography (CT) scan of head revealed extensive bilateral intracranial calcification (Figure I). Patient was managed with antiepileptic, calcium gluconate, calcitriol and levothyroxine in adequate dose.

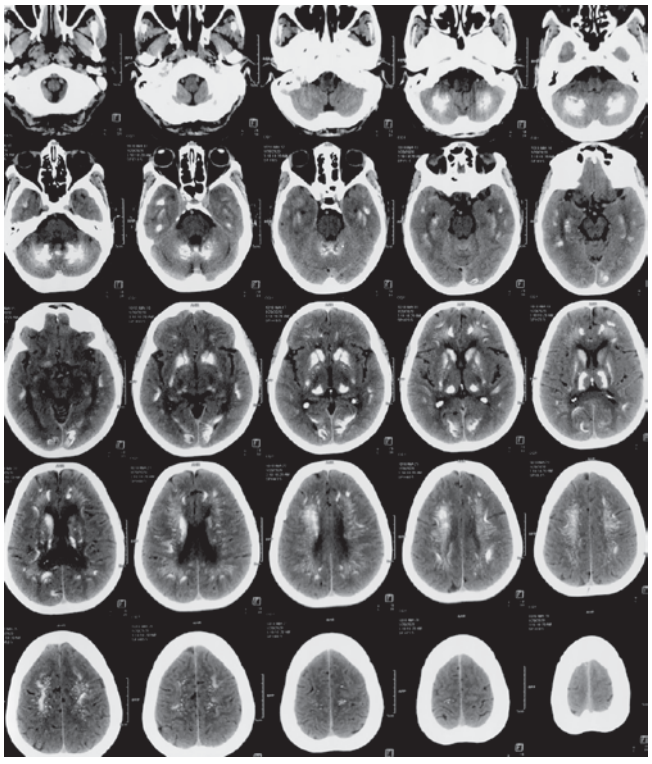

Figure I: Computed tomography (CT) scan of head (axial view) demonstrating extensive bilateral calcification

Table 1: Coagulation Profiles of Patients

Biochemical Test	Results	Normal value
Serum total calcium	1.57 m mol/L	2.10 – 2.55 m mol/L
Serum albumin	40 m mol/L	35 – 52 m mol/L
Serum magnesium	0.75 m mol/L	0.66 – 1.07 m mol/L
Serum phosphorus	6.8 mg/dL	2.3 – 4.7 mg/dL
Serum TSH	31.54 µiu/mL	0.35 – 4.94 µiu/mL
Serum FT3	1.56 pg/mL	1.88 – 3.18 pg/mL
Serum FT4	0.78 ng/dL	0.78 – 1.48 ng/dL
Serum iPTH	2.3 pg/mL	15 – 68.3 pg/mL
Serum 25-OH vit - D	11 ng/mL	10 – 65 ng/mL

Discussion

Postsurgical hypoparathyroidism often causes the most troubling long-term consequence of aggressive thyroid surgery and the reason is injury to the parathyroid glands or their blood supply or inadvertent resection of parathyroid tissue. The risk factors include bilateral central neck surgery, surgery for thyroid malignancy with or without central neck dissection, surgery for parathyroid hyperplasia, female sex, vitamin D deficiency, pregnancy, lactation, autoimmune thyroid disease and prior gastric bypass surgery[4]. In this particular patient, actual cause could not be determined as the patient was unable to show the document related to surgery and she was not in regular follow up.

Clinical manifestations in hypoparathyroidism result from hypocalcaemia, while biochemical abnormalities reveal hyperphosphatemia in addition to decreased detectable levels of serum parathyroid hormone and calcium. Most of the symptom and sign of hypocalcaemia occur because of increased neuromascular excitability like tetany, paresthesias, muscle cramping, seizures, organic brain syndrome and many of the complications of treatment of hypocalcemia result from the deposition of calcium in the soft tissues, cataract, calcification of basal ganglia, nephrolithiasis, nephrocalcinosis, reduced renal function). These patients demonstrate intracranial calcification, particularly extensive calcification bilaterally in the cerebral cortex in plain CT. Intracranial calcifications refer to calcifications within the brain parenchyma or vasculature; it may be due to physiological or age related, genetic/developmental disorders, congenital/acquired Infections, vascular, neoplastic, inflammatory or metabolic. Among the metabolic cause, any disorder affecting calcium homeostasis may lead to brain calcifications including hypoparathyroidism and hyperparathyroidism. Brain calcifications affect most notably the basal ganglia and most commonly the globus pallidus.

Calcifications may occur in the dentate nucleus, corona radiata, subcortical white matter and thalamus. Calcifications were thought to arise from a long-standing hyperphosphatemia; yet, the exact etiology remains unclear[5]. In our patient, calcinosis exceeds the common brain locations and involves the subcortical white matter of the frontal and parietal lobes which is demonstrated in only few case reports of excessive calcification of subcortical white matter in relation to postoperative hypoparathyroidism[6-9].

Conclusion

In conclusion, chronic hypocalcaemia due to postoperative hypoparathyroidism along with hyperphosphatemia can cause diffuse intracranial calcification which in turn triggers several neurological symptoms like seizure. Therefore, judicial surgery and regular post-operative follow up is required to prevent such complications.

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