

## Recurrent seizures as manifestation of hypoparathyroidism-related hypocalcemia in a patient with post-subtotal thyroidectomy



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### ABSTRACT

**Background:** Hypocalcemia, a condition where the serum calcium concentration or total serum calcium is below normal, could be associated with various manifestations including severe symptoms requiring immediate hospitalization. The condition can cause by several causes including iatrogenic thyroidectomy. Here, we report a patient of hypocalcemia associated with hypoparathyroidism due to post-thyroidectomy.

**Case presentation:** A 32-year-old woman admitted to the hospital complained of stiffness and tingling in the extremities accompanied by recurrent seizures. The serum calcium level was low and this hypocalcemia condition was associated with low parathyroid hormone levels after subtotal thyroidectomy surgery. Laboratory examination indicated calcium 4.8 mg/dL and parathyroid hormone 2.21 pg/ml. The patient had bradycardia with prolonged QT on electrocardiography (ECG) and developed cataracts in both eyes which could be associated with chronic hypocalcemia. The patient's condition improved after correction of hypocalcemia. After discharge, the patient was given calcium and levothyroxine supplementation due to hypothyroid conditions.

**Conclusion:** The hypocalcemia condition in the patient was associated with low parathyroid hormone level post-subtotal thyroidectomy surgery. This case highlights the impotence of detailed analyses of hormonal, radiological and histological investigations of patients with thyroid gland diseases to diagnose such conditions early and to ensure timely management.

**Keywords:** Hypocalcemia, hypoparathyroidism, PTH, hyperthyroidism, thyroidectomy.

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### INTRODUCTION

Hypocalcemia is defined as a condition where the serum calcium ion concentration or total serum calcium is below normal after being corrected by serum albumin values. Calcium plays an important role in maintaining normal cell functions, particularly in the transmission of nerve impulses, cell membrane stability, intracellular signaling and bone tissue structure.<sup>1</sup> Hypocalcemia has a prevalence of 18% in all hospitalized patients and 85% in intensive care units. Acute hypocalcemia can cause severe symptoms that require immediate hospitalization.<sup>2,3</sup>

Thyroidectomy is a surgical procedure that is often performed by general surgeons as well as head and neck surgeons. The complications of thyroid resection occur in less than 5% of cases and one of the common complications is hypocalcemia.<sup>4</sup> Hypocalcemia in post-thyroidectomy

patients because the incidental removal the parathyroid gland(s) or devascularization of the parathyroid glands. The incidence of incidental parathyroidectomy during thyroidectomy ranging between 6.4–31.1%.<sup>4</sup>

This case report highlights the diagnosis and treatment of a patient with recurrent hypocalcemia related to hypoparathyroidism after subtotal thyroidectomy with initial manifestations of recurrent seizures.

### CASE PRESENTATION

A woman, 32-year-old, was admitted to the Emergency Department of Dr. Soetomo Hospital. She had seizures since nine hours before being admitted to the hospital. Before the seizure, the patient was able to feel the initial signs such as a thick feeling and tingling. Seizures occurred for about five minutes, after which the patient

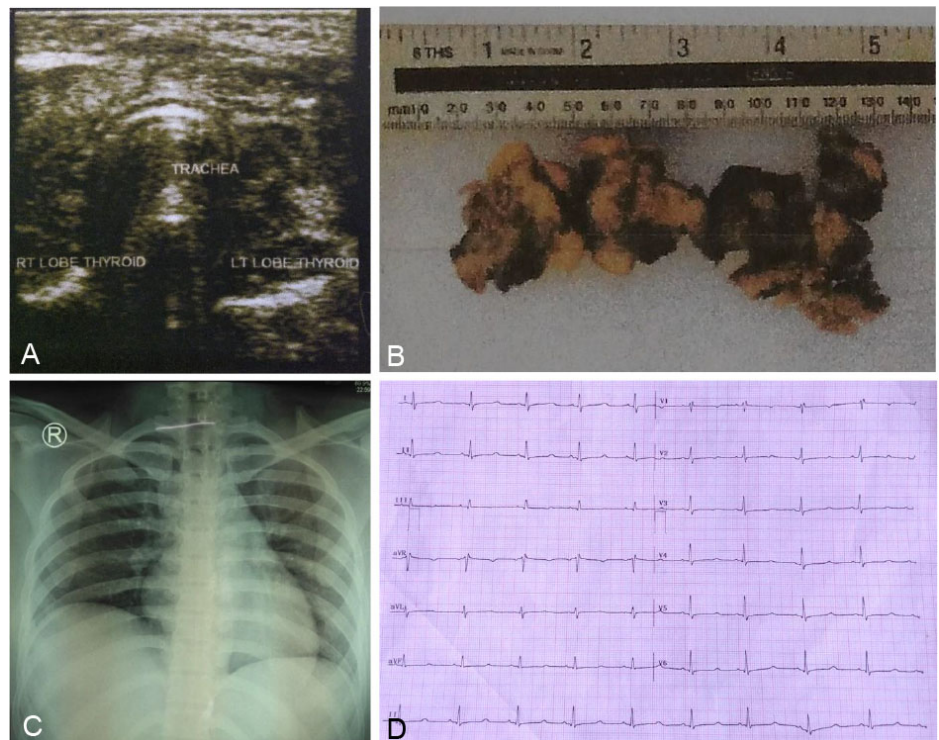
was conscious. The patient had a history of thyroid gland surgery three years before admission. Initially, the patient complained of a lump in the neck that was felt to be getting bigger for three months. Patients also felt easily tired, increased hair loss, increased body weight and defecation occurred once every 2-3 days. Complaints of hand tremors, frequent sweating, chest palpitations were absent. The patient also complained of a hoarse voice. The thyroid ultrasound revealed a hypoechoic lesion with indistinct border, irregular edge with calcification measuring 1.2 cm on right thyroid gland (**Figure 1A**) and during color Doppler ultrasound (CDUS) examination, the peri lesion vascularization was appeared. This suggested of Hashimoto's right lobe thyroiditis. The left lobe of thyroid had no residual mass or nodule. The patient had thyroidectomy and a mass of 6 x 4 x 3 cm, approximately 20 grams with rough outer surface was removed (**Figure**

1B). The histopathological examination showed proliferation of small-sized thyroid follicles (atrophy) containing moderate colloid material, coated with follicular epithelial cells, some cuboid with eosinophilic cytoplasm (Hurthle cell) and a large area of lymphocyte inflammation cells forming lymphfollicles with reactive germinal center with a conclusion of Hashimoto thyroiditis. The level of thyroid hormone was within normal limits.

One week after thyroidectomy the patient complained of having seizure. The patient's blood calcium level was low, the patient was treated with oral levothyroxine 100 mcg every 24 h and oral calcium 500 mg every 12 h. A seizure occurred when the patient was tired up to two times a week. Patients did not routinely come to the hospital for evaluation and stopped taking medicine. Six months after thyroidectomy, the patient complained of white patches on the right and left eyes. The patient was diagnosed with cataracts and suggested for surgery. The surgery on the right eye cataract was carried out five months later. The patient was planned for cataract extraction on the left eye but delayed due to low blood calcium level and the anesthesiologist did not approve the surgery.

On admission, the patient was alert with Glasgow Coma Score (GCS) 15, blood pressure 110/70 mmHg, pulse rate 58 x/min, respiratory rate 20 x/min and axillary temperature 37.2°C. Examination of the head and neck revealed a surgical scar on the front of the neck. Chvostek's sign was positive. Examination of the upper and lower extremities revealed Trousseau's sign and carpopedal sign were positive.

Laboratory examination indicated hemoglobin 10.4 g/dL, hematocrit 30.4%, white blood cells 8,300/ $\mu$ L, platelet 341,000/ $\mu$ L, neutrophils 71.8%, lymphocyte 21.9%, mean corpuscular volume (MCV) 72 fL, mean corpuscular hemoglobin (MCH) 26.7pq, mean corpuscular hemoglobin concentration (MCHC) 24 g/dL, blood urea nitrogen (BUN) 7 mg/dL, serum creatinine 1.31 mg/dL, serum glutamic oxaloacetic transaminase (SGOT) 51 U/L, serum glutamic oxaloacetic transaminase (SGPT) 87 U/L, albumin 3.8 g/dL, glucose 100 mg/



**Figure 1.** Thyroid ultrasound revealed a hypoechoic lesion with indistinct border, irregular edge with calcification on right thyroid (A). Thyroidectomy removed a mass of 6x4x3 cm from right thyroid gland (B). The chest x-ray showed normal heart and lungs (C). Electrocardiography (ECG) suggested sinus rhythm bradycardia with prolonged QT (D).

dL, sodium 143 mmol/L, potassium 4.0 mmol/L, chloride 97 mmol/L, and calcium 4.8 mg/dL. The chest x-ray of the heart and lungs showed no abnormalities (Figure 1C). Electrocardiography (ECG) results suggested sinus rhythm bradycardia, 58x/min, normal axis, prolonged QT (Figure 1D).

Consultation to the Ophthalmology Department found oculi dextra pseudophakia with visus >2/60 and oculi sinistra complex cataracts DD juvenile with visus 1/300. The patient was suggested for elective cataract surgery on oculi sinistra.

Based on the history, physical examination, laboratory and other assessments and tests, the patient was diagnosed with post-cute symptomatic seizure related to hypocalcemia, suspected iatrogenic hypoparathyroidism (post subtotal thyroidectomy), prolonged QT interval, sinus bradycardia, oculi sinistra complicated cataracts and juvenile.

Initial management includes a high-calorie, high-protein diet of 2100 kcal per day, 1000 mL of Asering (electrolyte

infusion containing calcium chloride, potassium chloride, sodium chloride, sodium acetate, anhydrous dextrose) infusion every 24 h, 1000 mg slow bolus of calcium gluconate injection followed by 2000 mg of calcium gluconate drip in 100 mL of 0.9% NaCl every 8 h, and 500 mg oral calcium every 12 h. Post-correction in Emergency Department showed calcium 5.2 mg/dL and magnesium 1.4 mg/dL. Then the patient was transferred to the High Care Unit. On the third day of treatment, the patient had no complaint or seizure. Laboratory examination indicated calcium 6.7 mg/dL, urine calcium 36 mol/24 h, urine potassium 32 mmol/24 h, urine sodium 158 mmol/24 h, urine chloride 149 mmol/24 h, free thyroxine (FT4) 0.53 ng/dL, thyroid stimulating hormone (TSH) 85.253 IU/mL, and parathyroid hormone (PTH) 2.21 pg/ml. Fifth day of treatment, the calcium was 7.7 mg/dL and magnesium 1.6 mg/dL. After seven days on treatment, the laboratory test indicated calcium 8.7 mg/dL and magnesium 1.9 mg/dL. The patient was discharged from the hospital with

oral calcium 500 mg every 8 h and oral levothyroxine 100 mcg every 24 h.

One week after hospital discharge, the patient visited the hospital with no complaint of seizure, tingling or stiffness in the arms and legs. The laboratory investigation indicated the level of calcium was 7.2 mg/dL, FT4 1.28 ng/dL and TSH 18.5 IU/mL. The treatment of oral calcium and oral levothyroxine was continued.

## DISCUSSION

Hypocalcemia is a common electrolyte disorder whose clinical manifestations vary since low serum calcium level may impair organ function. Based on the onset, hypocalcemia can be divided into acute or chronic hypocalcemia. Hypocalcemia could be life-threatening in acute conditions and requires immediate intervention.<sup>5</sup> Chronic hypocalcemia might be asymptomatic and is generally mild and rarely requires correction.<sup>6</sup> Chronic manifestations of hypocalcemia include subcapsular lentic cataracts, dry skin, exfoliative dermatitis, coarse hair, brittle nails, chronic pruritus, and ossification of the paravertebral ligaments.<sup>2,4</sup>

Our patients presented with generalized seizures and had chronic symptoms of hypocalcemia - cataracts in both eyes. The ECG examination revealed sinus bradycardia with prolonged QT. Severe hypocalcemia could cause prolonged muscular contraction of the airways and larynx leading to respiratory failure, heart failure, refractory hypotension and arrhythmias. Hypocalcemia seizure is categorized as symptom of severe hypocalcemia. Milder symptoms of hypocalcemia include muscle cramps and twitching, tingling feeling and prolongation of QT values on ECG examination.<sup>7</sup> In addition, hypocalcemia could lead to irreversible calcification of the basal ganglia, cerebellum or cerebrum.<sup>7</sup> In some chronic cases, hypocalcemia can cause symptoms of psychosis, psychoneurosis and decreased the level of intelligence.<sup>8</sup>

Management of acute hypocalcemia is by administering 10 mL of 10% calcium gluconate solution in 100 ml of 0.9% sodium chloride or 5% glucose for 10-20 min depending on condition.<sup>3</sup> It can be given by slow intravenous injection

over at least 3 min in an emergency. ECG monitoring is recommended in particular in those at risk of arrhythmias or heart disease. Monitor of serum calcium level 1-2 h after initial dose is needed. If the patient persists with symptoms of hypocalcemia, it is often necessary to administer continuous maintenance doses by infusion to prevent relapse.<sup>3,9</sup>

Symptoms of hypocalcemia can be in the form of calcium deposition in soft tissues resulting in visual disturbances due to cataracts, papillae edema or calcification of the basal ganglia. In our patient, the morphology of the cataract that occurred bilaterally and without other concomitant primary ocular conditions suggested that the cause of the cataract was a long-term low serum calcium level. One of the pathomechanisms of cataract formation in hypocalcemia patient is membrane damage and increased sodium levels in the lens.<sup>10</sup>

The patient had a history of subtotal thyroidectomy surgery three years before admission due to Hashimoto thyroiditis causing low PTH hormones that have important roles in regulation of thyroid hormones that are critical in homeostasis functions on the respiratory, cardiovascular, gastrointestinal, renal, and other systems.<sup>11-14</sup> In Hashimoto thyroiditis, thyroidectomy is considered after treatment with drugs has failed. Other surgical indications are (1) cytological features of papillary or medullary cancer or follicular lesions, (2) clinical suspicion of malignancy, (3) presence of suppression/urgency symptoms, (4) patient choice regarding cosmetics/anxiety, (5) toxicity, (6) progressive enlargement of the retrosternal goiter, and (7) patient discomfort. Symptoms of suppression/urgency are swallowing disorders, hoarseness, dyspnea, and dilation of blood vessels around the neck/face.<sup>15,16</sup>

Post-thyroidectomy patients are at risk of developing hypoparathyroidism which eventually causes hypocalcemia and calcifications.<sup>17</sup> Most patients who experience parathyroid dysfunction after thyroidectomy will return to normal in less than one month post-surgery. The serum PTH levels recovered to at least 10 pg/mL.<sup>18</sup> In our patient, the FT4 level of 0.53 ng/dL, TSH 85.253 IU/mL, and

PTH 2.21 pg/mL. Calcium homeostasis is maintained by interacting with PTH, vitamin D and calcitonin through a complex feedback loop mechanism in the bones, kidneys and intestines.<sup>19</sup>

The parathyroid gland, which is located in the posterior part of the thyroid gland, is responsible for producing PTH. PTH is the main controller of calcium balance. When the total serum calcium concentration falls below 8.8 mg/dL, the PTH secretion will increase reabsorption and reduce calcium clearance capacity and conversely increase phosphate excretion in the renal tubules. In bone tissue, PTH stimulates osteoclastic activity, thereby mobilizing calcium and phosphate from bone into the blood. PTH increases the absorption capacity of calcium and phosphate in the small intestine by stimulating the production of vitamin D3 (1,25-dihydroxycholecalciferol, calcitriol) from 25-hydroxy-D in the kidney. The calcitriol also reduces calcium reabsorption in the kidney.<sup>20</sup> Conversely, if the serum calcium concentration increases above the normal threshold, calcium is removed from the plasma through the gastrointestinal tract, urine (and the rest is stored back into the bones).<sup>21,22</sup>

Iatrogenic injury to the parathyroid gland during thyroidectomy is unintended consequence. Measurement of PTH levels immediately after surgery is a sensitive and specific method for assessing parathyroid gland function. If postoperative PTH levels are low, administration of activated calcium and vitamin D (calcitriol) may reduce the incidence of symptomatic hypocalcemia. The incidence of low PTH levels after total thyroidectomy varies ranging from 7% to 37%.<sup>18</sup>

The goal of hypoparathyroidism management is to correct acute and chronic hypocalcemia. In acute hypoparathyroidism, intravenous infusion of calcium is required only in symptomatic patients and oral calcium and/or calcitriol is recommended in chronic hypoparathyroidism. PTH hormone replacement such as recombinant human parathyroid hormone (rhPTH) has been shown effective to increase the serum calcium levels.<sup>23</sup> Hypocalcemia conditions generally have a good prognosis if diagnosed properly and received adequate therapy. A study involving 2,631 patients

in 39 hospitals in Italy reported transient hypoparathyroidism complications in 28.8% and long-term hypocalcemia in 0.9%.<sup>24</sup>

## CONCLUSION

Recurrent seizures were occurred due to hypocalcemia condition in a post-thyroidectomy patient. The hypocalcemia condition was associated with low PTH levels after subtotal thyroidectomy surgery. The patient also had cataracts in both eyes which could be associated with chronic hypocalcemia. The patient condition improved after correction of hypocalcemia and the patient received continuous calcium and levothyroxine supplementation due to hypothyroid conditions. This case highlights that in patients with thyroid gland disorders, a detailed analysis based on clinical conditions and hormonal, radiological and histological investigations should be carried out in order to ensure that the appropriate treatments can be delivered.

## PATIENT CONSENT

The patient agreed and signed informed consent regarding publishing this clinical case in an academic journal without exposing the patient's identity.

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## DISCLOSURE OF CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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## AUTHOR CONTRIBUTION

Both authors contributed equally to the study.

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