INTRODUCTION

Hypoparathyroidism is one of the disorders in the field of endocrinology characterized by a decrease in parathyroid hormone levels in the body, leading to the conditions of hypocalcemia and hyperphosphatemia. Hypoparathyroidism most commonly occurs in adults after surgery on the thyroid gland. Other causes of hypoparathyroidism include autoimmune diseases, genetic factors, or the influence of other endocrine glands. Rare causes include invasive diseases, tumor metastasis, and ionizing radiation.

Hypoparathyroidism most commonly occurs, especially after surgery in the neck area, involving the thyroid, parathyroid, and laryngeal glands in the presence or absence of malignancy. Approximately 20-30% will experience transient hypoparathyroidism after surgery, and 1-7% of patients will experience permanent hypoparathyroidism following total thyroidectomy.

One clinical manifestation of hypoparathyroidism is the occurrence of hypocalcemia. The presence of persistent hypocalcemia for more than 6 months post-total thyroidectomy is one sign of permanent hypoparathyroidism after surgery. Decreased levels of parathyroid hormone in the blood result in calcium loss through the kidneys and reduced calcium absorption in the gastrointestinal tract. A classic manifestation of hypocalcemia is neuromuscular excitability characterized by twitching or spasms in the muscles, tingling, and numbness in the extremities. Prolonged hypocalcemia can lead to neuropsychiatric disturbances. One frequently occurring neuropsychiatric disorder is the presence of parkinsonism due to calcification in the basal ganglia, known as Fahr's syndrome.

Patients post-total thyroidectomy have a high risk of developing permanent hypoparathyroidism. A clinical approach involving the measurement of parathyroid hormone and calcium levels 4-6 hours after the thyroidectomy procedure for 24 hours can predict the occurrence of permanent hypoparathyroidism. Therefore, the need for calcium and vitamin D supplementation can be initiated to reduce the incidence of acute hypocalcemia.

Acute hypocalcemia is considered a medical emergency, and prompt intravenous calcium replacement is crucial to reduce morbidity and mortality.

CASE PRESENTATION

A 70-year-old female patient was admitted to the hospital with complaints of stiffness in both hands, both right and left, for the past 3 days before admission. The stiffness in the hands extended to the patient's finger joints, making it difficult for her to move her hands and fingers, as well as perform simple actions such as gripping. The stiffness was reported to gradually worsen, especially in the evening before being admitted to the hospital, prompting
the patient to seek medical attention promptly. Stiffness was also felt in both legs and the patient's toe joints, making it difficult for her to move her legs and walk. Stiffness in both hands and legs was not accompanied by pain. There has been a decreased appetite and fluid intake since 3 days before. The patient is reported to be lying in bed, as conveyed by her child. Bowel movements and urination are said to be normal. Complaints of cough, shortness of breath, nausea, and vomiting were denied.

The patient has a history of total thyroidectomy surgery in 2008 at Sanglah General Hospital. A few days after the procedure, the patient experienced stiffness symptoms in both hands. In 2018, the patient had similar symptoms, experiencing stiffness in both hands and legs, leading to a return to the hospital for treatment. The patient has a history of heart disease, specifically coronary heart disease, and regularly seeks treatment at Bangli General Hospital. The patient has also been admitted to Sanglah General Hospital for the mentioned heart condition. The patient's medication history includes: levothyroxine 100 mcg every 24 hours orally since 2008, with a later dose reduction to 50 mcg every 24 hours orally since 2018; spironolactone 25 mg every 24 hours orally; bisoprolol 2.5 mg every 24 hours orally; furosemide 40 mg every 24 hours orally; folic acid 2 mg every 12 hours orally; ramipril 5 mg every 24 hours orally; and aspirin 80 mg every 24 hours orally. The patient denies a history of other diseases, liver disease, or any other chronic illness. The patient is a homemaker, married, and has three children. There is no family history of similar complaints or other chronic diseases. The patient denies a history of smoking or alcohol consumption.

In the physical examination, the patient appears moderately ill with a conscious and comous mentis state. Blood pressure is measured at 90/60 mmHg, pulse rate at 80 beats per minute, respiratory rate at 20 breaths per minute, and axillary temperature at 36.7°C. Examination of the head reveals no signs of anemia or jaundice. In the neck examination, a transverse surgical scar is noted in the middle of the neck, with a jugular venous pressure (JVP) of approximately 0 cm H2O. No enlargement of lymph nodes is found in the neck, axilla, or inguinal areas. Lung examination shows symmetrical static and dynamic chest movements, with no increased tactile vocal fremitus on palpation. Percussion reveals a resonant sound, and auscultation detects vesicular breath sounds in both lung fields without rales or wheezing.

In the heart examination, the point of maximal impulse (PMI) is observed at the 5th intercostal space (ICS) midclavicular line (MCL) sinistra. Percussion identifies the right heart border 1 cm lateral to the right parasternal line and the left heart border 4 cm lateral to the left midclavicular line. Auscultation reveals a regular S1 S2 heart sound without murmurs. Abdominal examination does not reveal any abnormalities. In the upper extremities, warmth is palpable without edema. Motor strength in both upper and lower extremities is assessed as 4/4, with normal physiological reflexes (biceps, triceps, patellar, and Achilles) and the absence of pathological reflexes. Chvokest's and Trousseau's signs are not elicited in the patient.

Laboratory result when first examined showed that white blood cell 10.46 x 10^9/μL, neutrophil 7.70 x 10^9/μL, lymphocyte 1.90 x 10^9/μL, monocytes 0.70 x 10^9/μL, basophil 0.07 x 10^9/μL, red blood cells 4.32 x 10^12/μL, hemoglobin 10.80 gr/dL, hematocrit 34.74%, MCV 80.41 fL, MCH 25.0 g/dL, thrombocytes 403.20 x 10^9/μL, SGOT 19.0 U/L, SGPT 9.20 U/L, random blood sugar 153 mg/dL, BUN 52.60 mg/dL, creatinin 3.55 mg/dL, TSHs 8.51 IU/ml, FT4 1.35 mg/dL, sodium 132 mmol/L, potassium 5.50 mmol/L, chloride 88.2 mmol/L, calcium (Ca) 4.6 mg/dL, inorganic phosphate 10.96 mg/dL, magnesium 2.04 mg/dL, albumin 3.60 g/dL, HDL cholesterol 21 mg/dL, LDL cholesterol 68 mg/dL, total cholesterol 116 mg/dL, and triglyceride 11 mg/dL.

Chest X-ray examination revealed cardiomegaly with aortosclerosis (aortosclerosis heart disease), no apparent abnormalities in the lungs, and thoracic spondylosis. The electrocardiogram (EKG) results for the patient when first admitted to the hospital show a sinus rhythm of 72 beats per minute and a normal corrected QT interval (QTc) of 390 ms. The patient underwent an echocardiogram in 2018, revealing a dilated left ventricle, left ventricle hypertrophy, eccentricity, ejection fraction of 21%, global hypokinesia, severe mitral regurgitation, and mild tricuspid regurgitation.

The initial management for this patient involves hydration with intravenous infusion of NaCl 0.9% at a rate of 20 drops per minute. Correction for hypocalcemia is carried out with intravenous administration of Calcium (Ca) gluconate 1000 mg in 250 ml of 5% Dextrose over 30 minutes, followed by continuous intravenous infusion (drip) of Ca gluconate at a rate of 1 mg/kgBW/hour for 24 hours. The patient is also provided with oral calcium supplementation of CaCO3 500 mg every 8 hours and Vitamin D (Calcitriol) 0.5 mcg every 12 hours orally. Thyroid hormone replacement therapy is initiated with oral levothyroxine 50 mg once daily on an empty stomach in the morning. Additionally, the patient is referred to the cardiology department for their heart condition and is prescribed aspirin 80 mg once daily orally, ramipril 5 mg once daily orally, spironolactone 25 mg once daily orally, simvastatin 20 mg once daily orally, and bisoprolol 2.5 mg once daily orally.

On the second day of treatment, the patient's complaints of stiffness in both hands and feet began to decrease, and there was a gradual increase in serum calcium levels during the administration of Ca gluconate drip. On the fourth day of treatment, the patient complained that both of her hands were shaking again, especially noticeable when at rest. Subsequently, the patient was referred to the Neurology department and advised to undergo a Head CT Scan.

The results of the Head CT Scan revealed no signs of intracranial bleeding but showed multiple calcifications in the right and left cerebellar hemispheres, bilateral basal ganglia, right thalamus, periventricul and centrum semiovale, suggesting Fahr syndrome or metabolic-related changes, as well as age-related brain atrophy. The patient was diagnosed with Parkinsonism associated with Fahr Syndrome and was prescribed...
trihexyphenidyl 2 mg every 12 hours orally and levodopa 100 mg once daily.

During the treatment, the increase in blood calcium levels occurred gradually, and on the sixth day of treatment, the continuous IV administration of Ca gluconate was stopped. The treatment was continued with oral administration of CaCO3 500 mg every 8 hours and Vitamin D (Calcitriol) 0.5 mcg every 12 hours.

During the treatment, a decrease in kidney function was also observed, characterized by an increase in BUN and SC levels. The patient was then diagnosed with Acute Kidney Injury (ACKD) potentially related to prerenal factors on Chronic Kidney Disease (CKD), and there were suspicions of Nephrotic Syndrome (NS) or Posterior Nutcracker (PNC). Kidney function was monitored every 2 days with BUN and SC levels continually decreasing. Additionally, an upper and lower abdominal ultrasound examination (USG) was performed, revealing multiple gallstones, a simple cyst in the upper pole of the left kidney, and normal findings for the liver, spleen, pancreas, right kidney, bladder, and uterus.

The presence of hypocalcemia after post-total thyroidectomy in this patient raises suspicion of a decrease in the patient’s parathyroid hormone levels. Subsequently, the patient underwent an intact parathyroid hormone examination to assess the parathyroid hormone levels in the body, with a result of 2.19 pg/mL (15-65 pg/mL), indicating that the patient is experiencing hypoparathyroidism, leading to the occurrence of hypocalcemia.

DISCUSSION

Parathyroid hormone is a polypeptide hormone responsible for maintaining calcium and phosphate homeostasis in the body. It is secreted by the parathyroid glands, four small glands located at the posterior aspect of the thyroid gland. The synthesis and secretion of parathyroid hormone are regulated by blood calcium levels through the modulation of the activity of the Calcium-Sensing Receptor located in the parathyroid glands. In conditions of hypercalcemia, the synthesis of parathyroid hormone decreases, affecting the release of parathyroid hormone fragments. Conversely, hypocalcemia stimulates the synthesis of parathyroid hormone. In bone tissue, parathyroid hormone inhibits the activity of osteoblasts and stimulates the activity of osteoclasts, leading to bone breakdown and calcium release. In the kidneys, parathyroid hormone increases calcium reabsorption and inhibits phosphate reabsorption from the renal tubules. Additionally, it stimulates the formation of vitamin D in the kidneys. Vitamin D (1,25-dihydroxyvitamin D) plays an essential role in calcium and phosphate homeostasis. It works to increase blood calcium levels by enhancing calcium release from bones, increasing calcium reabsorption in the kidneys, and improving calcium reabsorption through the gastrointestinal tract. The result of this regulation is the normalization of calcium levels without retaining phosphate.7

Hypoparathyroidism is a clinical syndrome characterized by a decrease in parathyroid hormone levels, with the main manifestations being hypocalcemia and hyperphosphatemia. This condition is a common complication in patients following surgery in the neck area, particularly after thyroid surgery. Hypoparathyroidism can occur due to the inadvertent removal of the parathyroid glands during thyroidectomy or due to vascular disruption of blood vessels during surgery. The majority of clinical symptoms of hypoparathyroidism arise from hypocalcemia, where decreased

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Figure 1. Head CT-Scan of the patient

Table 1. The results of the serum calcium examination during the administration of Ca Gluconate drip

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Hyperparathyroidism is a disease characterized by abnormal calcium deposits in the brain, typically forming calcifications that disrupt blood flow and nerve function. 

This case describes a patient with symptoms of hypocalcemia and hyperphosphatemia. To treat these symptoms, the patient was prescribed anticholinergics and levodopa.

The primary management of hypoparathyroidism is directed towards treating hypocalcemia. The therapy involves intravenous (IV) calcium, aiming to control symptoms and normalize serum calcium levels. Initial therapy includes IV Calcium (1–2 grams of calcium gluconate in 50 ml of 5% Dextrose) administered over 10-20 minutes. The treatment should be followed by an infusion of maintenance dose calcium, specifically calcium gluconate 1 mg/mL mixed with normal saline or 5% Dextrose at a rate of 0.5-1.5 mg/kg elemental calcium per hour. This IV calcium correction protocol is expected to increase serum calcium by 2 mg/dL over 8-10 hours. Calcium gluconate is preferred over calcium chloride due to its lower risk of tissue necrosis in case of extravasation. Active vitamin D metabolites should be administered because parathyroid hormones facilitate the conversion of 25-hydroxyvitamin D to 1,25-dihydroxyvitamin D (calcitriol). The active form of vitamin D is a therapeutic choice in managing hypoparathyroidism to aid calcium absorption in the gastrointestinal tract. The initial dose of calcitriol is typically 0.25–0.5 μg every 12 hours. Oral calcium and vitamin D supplementation should be initiated concurrently with intravenous calcium administration.

In summary, the patient was diagnosed with hypoparathyroidism, and the management involved intravenous calcium, calcitriol, and oral calcium and vitamin D supplementation. The patient responded well to treatment, with symptoms improving and serum calcium levels returning to normal.

**CONCLUSION**

A case has been reported of a woman with secondary hypoparathyroidism and manifestations of Fahr's syndrome following total thyroidectomy. The patient was found to have hypoparathyroidism with characteristics of clinical manifestations caused by hypocalcemia and hyperphosphatemia. According to the management of hypocalcemia, the patient received continuous intravenous calcium therapy to restore the patient's calcium levels to normal. In addition to intravenous calcium, oral calcium supplementation and vitamin D, as well as thyroid hormone replacement therapy, were also given to the patient. Hypoparathyroidism is the most common cause of Fahr's syndrome. Fahr's syndrome is characterized by multiple calcifications in the bilateral basal ganglia accompanied by neurological symptoms such as parkinsonism-like symptoms, including spasticity and involuntary movements. The treatment for Fahr's syndrome is primarily aimed at controlling parkinsonism symptoms with anticholinergics and levodopa.

**CONFLICT OF INTEREST**

There was no conflict of interest in writing this study report.

**ETHICAL CONSIDERATIONS**

The patient has given consent to participate and publish the data.

**FUNDING**

The author was responsible for funding this study without involving other parties.

**AUTHORS' CONTRIBUTION**

The authors contributed to writing this study report, from the first step of proposal preparation, data collection and analysis until the preparation of the report in the form of publication.
REFERENCES


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