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Asymptomatic Hypercalcemia Due to Bilateral Parathyroid Adenoma: A Case Report

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ABSTRACT

Hyperparathyroidism caused by parathyroid adenoma is one of causes of hypercalcemia. Majority of parathyroid adenoma cases are found incidentally. We report a male patient aged 40 years, asymptomatic, who is undergoing routine laboratory examinations to extend his work contract. On examination we found an increase in calcium levels and parathyroid hormone levels. Then MSCT examination showed bilateral parathyroid adenoma. The diagnosis pathway and proper treatment for parathyroid adenoma can be challenging and problematic owing to inability of standard imaging to detect small enlargement of parathyroid glands. MSCT has the advantage of being able to detect small parathyroid lesions that cannot be detected by simple imaging modalities. Parathyroidectomy is the definitive treatment for adenoma parathyroid.

Keywords: Hypercalcemia, parathyroid, asymptomatic.

INTRODUCTION

Hypercalcemia can occur when too much calcium enters the extracellular fluid or when calcium excretion from the kidneys is insufficient. Serum calcium normal ranges from 8.8 mg/dL-10.8 mg/dL.[1] Symptoms that arise due to hypercalcemia vary, such as kidney disorders, disorders of the skeletal bones, and often no symptoms at all.[2] Approximately 90% of cases of hypercalcemia are caused by hyperparathyroidism or malignancy.[1] Disorders of the parathyroid glands are most often found with abnormalities of serum calcium. Patients with primary hyperparathyroidism are the most common cause form hypercalcemia in outpatients, often discovered incidentally during the evaluation of serum electrolyte levels. Hyperparathyroidism is most commonly caused by parathyroid adenomas (80-85%). Parathyroid adenomas cause excessive autonomic formation and release of parathyroid hormone (PTH), which is called primary hyperparathyroidism.[3]

Here we report a patient with asymptomatic hypercalcemic adenoma due to parathyroid adenoma. This case report is expected to increase clinicians' knowledge in establishing diagnosing and managing parathyroid adenoma.

CASE REPORT

A man, 40 years old, married, Javanese, working as a cruise ship employee, came to the Endocrine Polyclinic for a general check-up to extend the work contract. The patient came without complaints and denied having a history of complaints such as weakness, joint muscle pain, nausea, vomiting, palpitations, and impaired consciousness. History of kidney stones, radiation to the neck, and previous malignancy were denied. There was no family history of malignancy. History of the patient's habit of smoking one pack/day and denies drinking alcohol regularly.

The vital signs are within normal limits. On examination of the head and neck within

normal limits, there was no enlargement of the thyroid gland and lymph nodes from the neck to the axilla and inguinal. Thoraco-abdominal and extremity examinations were within normal limits.

Laboratory examination showed an increase in serum calcium 11.14 mg/dL (8.82-10.62 mg/dL) and serum parathyroid hormone 14.68 pg/mL (1.01-7.23 pg/mL); TSH, FT4, FT3 within normal limits. Complete blood count, kidney function, liver function, electrolyte blood sugar, and albumin were within normal limits. TSH receptor antibody negative; Thyroid peroxidase negative; Thyroglobulin antibody negative; 25-(OH)D within normal limits.

An ultrasound examination of the thyroid did not reveal a mass. However, the SPECT/CT; Parathyroid study + TOMO (OS605) found a moderate MIBI uptake focus in the left lower neck, suggesting a parathyroid adenoma in the left lower lobe. On examination of the Bone Survey (Figure 1), no abnormalities were found in the bones. We performed the neck Multislice Computerized Tomography (MSCT) with contrast, showing an enlarged bilateral inferior parathyroid gland (detected inferior parathyroid gland right side size 10.9x10.4x10mm, inferior parathyroid gland left side extension to upper anterior mediastinal size 31.0x7.5x5.5mm, normal parathyroid gland size 6x4x2mm) with wash out pattern in the delayed phase of the post-contrast study, suggesting Parathyroid Adenoma (Figure 2).

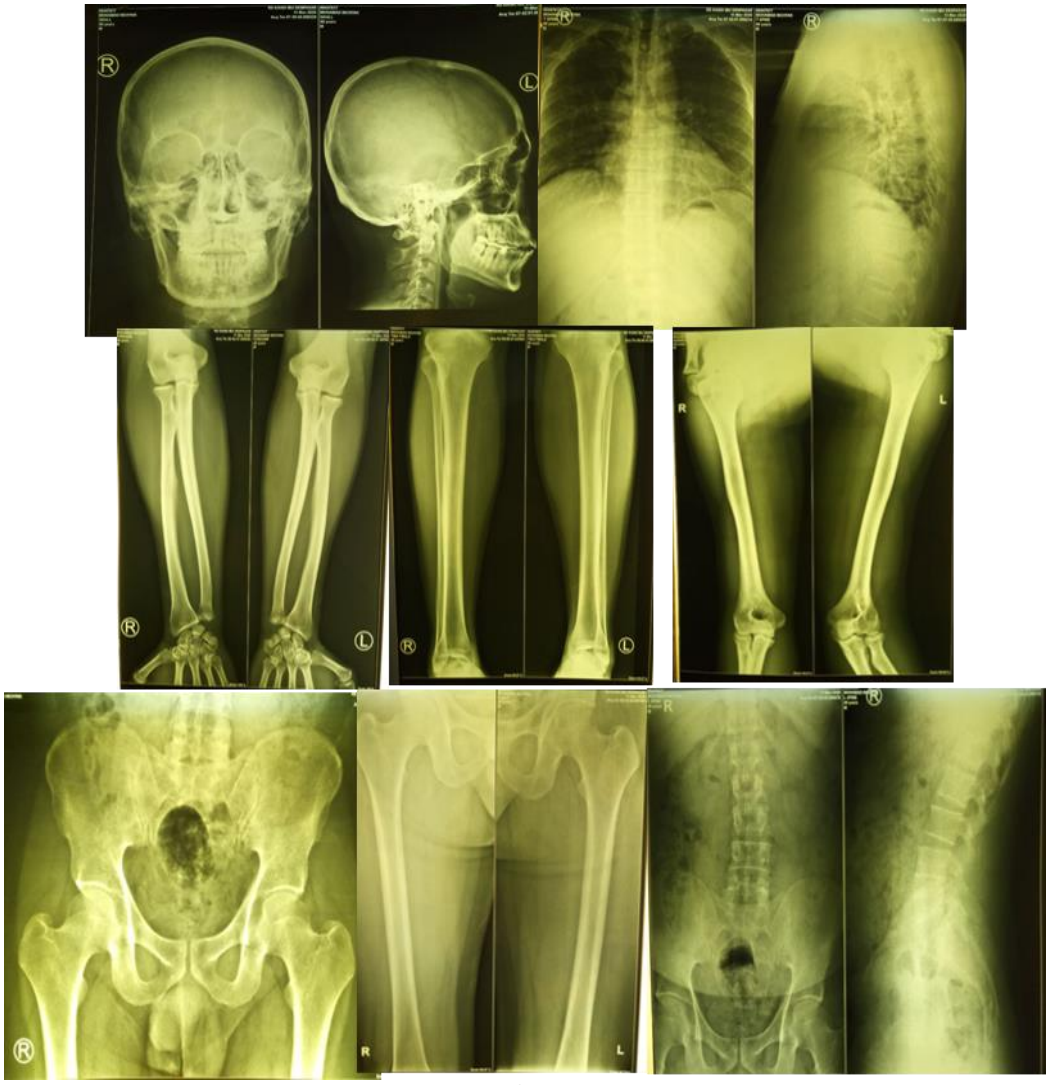

Figure 1. Bone survey was performed showing a normal result.

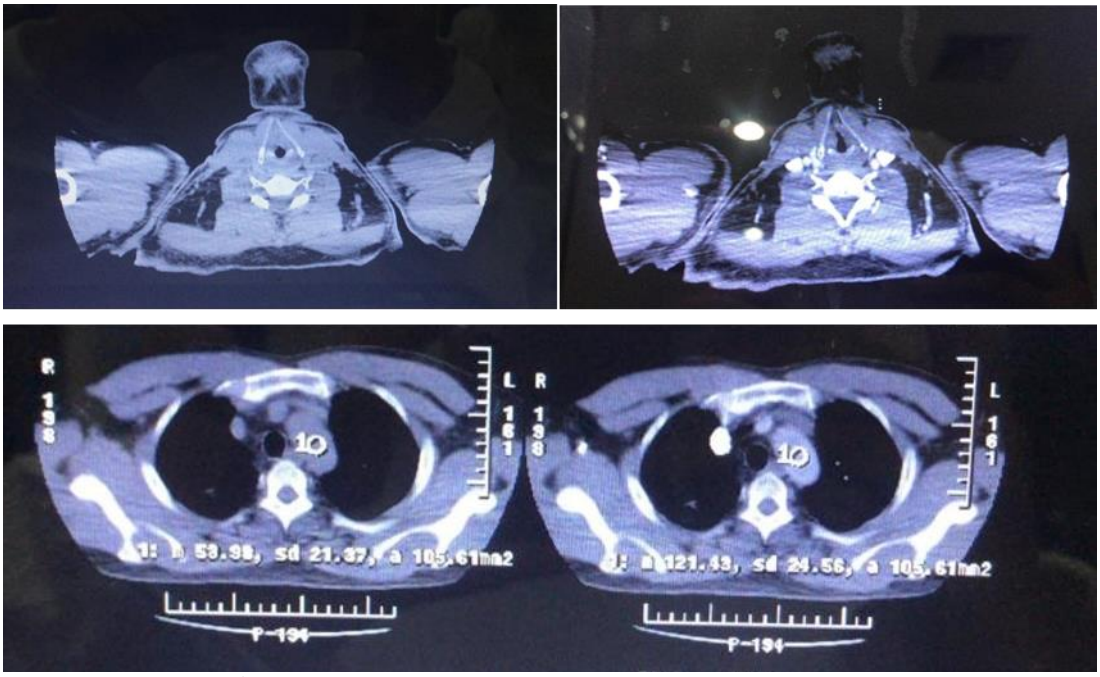

Figure 2. Regional neck MSCT focusing on parathyroid glands without and with contrast on wash out pattern in delayed phase post contrast study, showed a suspicious Parathyroid Adenoma.

The patient was diagnosed with parathyroid adenoma and then scheduled for parathyroidectomy (Figure 3). According to a histological study was performed with a microscopic picture shows solid growth pattern, composed of proliferative oxyphilic cells having cuboid shaped, rounded nuclei with fine chromatin, dense and eosinophilic cytoplasm, no adipose cell are found within the tumor, the conclusion is benign parathyroid adenoma. In this case, the patient did not receive medical therapy before and after the procedure. The patient has no complaints after parathyroidectomy.

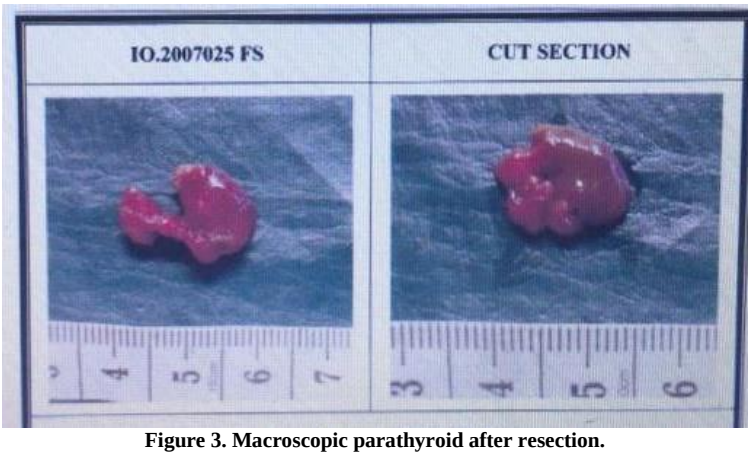

Figure 3. Macroscopic parathyroid after resection.

DISCUSSION

Excessive PTH secretion in hyperparathyroidism predisposes to hypercalcemia. This condition directly affects the bone, intestinal tract receptors, and kidneys. Physiologically, PTH secretion is inhibited by high serum calcium ion levels. This mechanism is not active in adenomas because hypersecretion of PTH co-occurs with hypercalcemia.[6]

Primary hyperparathyroidism is diagnosed when serum calcium levels are elevated, with elevated serum PTH levels.[2] In this case, we report a 40-year-old male patient diagnosed with parathyroid adenoma with an accidental increase in serum calcium and

parathyroid hormone during a general check-up, not accompanied by clinical symptoms.

The manifestations of hypercalcemia are very diverse. The classic signs of hypercalcemia are kidney stones, pain in the bone, abdominal pain, psychological disturbances, and fatigue. Most of the time, early symptoms of hypercalcemia are undiagnosed, especially in developing countries with limited laboratory facilities.[7,8] Decreased bone mineral density causes osteopenia, osteoporosis, and fractures are frequent complications of delayed diagnosis of hypercalcemia.[9]

The diagnosis of parathyroid adenoma can be established by history, physical examination, and investigations. Clinically, the manifestations of this disease are associated with signs and symptoms of hypercalcemia.[10,11] Asymptomatic hypercalcemia is most often found incidentally. If symptoms are present, a parathyroid adenoma may manifest as an enlarged gland in the neck that can be painful. Physical examination may reveal a lump in the neck, neurological disorders, and multiple fractures.[11]

Investigations showed increased calcium levels and parathyroid hormone, which led to the suspicion of primary hyperparathyroidism. Radiologic examinations that can evaluate the parathyroid glands are ultrasound, SPECT/CT, and MSCT.[12] The role of ultrasound in detecting parathyroid adenomas has a low positive predictive value.[13] Fused single-photon emission computed tomography/computed tomography (SPECT/CT) imaging can be used to make a differential diagnosis between benign and malignant tumors in various body organs, stage the lesion, confirm metastasis or recurrence, and select a treatment strategy.[14] In this case, SPECT/CT examination successfully demonstrated a parathyroid adenoma in the left lower lobe.

Multi-slice CT-Scan (MSCT) with contrast has a high effectiveness for the diagnosis of parathyroid adenoma with a sensitivity and specificity of 95.2%.[15] MSCT has the advantage of being able to detect small parathyroid lesions that cannot be detected by simple imaging modalities.[16] In the case of MSCT it can detect bilateral inferior parathyroid adenomas that cannot be found by SPECT/CT or ultrasound.

Parathyroidectomy is the only definitive treatment for primary hyperparathyroidism in symptomatic and asymptomatic cases. One study reported the correlation of parathyroidectomy with improved quality of life index.[14] Based on the American Association of Endocrine Surgeons Guidelines for Definitive Management of Primary Hyperparathyroidism in 2016, indications for parathyroidectomy surgery include: (1) Age < 50 years, (2) All symptomatic patients, (3) Asymptomatic patients with one or more of: (a) serum calcium level > 1.0 mg/dL above normal, (b) Dual-Energy X-Ray Absorptiometry T-score -2.5 in the lumbar spine, neck of the femur, total hip or 1/3 of the distal radius, (c) vertebral fracture, (d) Glomerular filtration rate/Glomerular filtration rate < 60 ml/min/1.73 m2, (e) urinary Ca excretion > 400 mg/24 hours, (f) nephrolithiasis and nephrocalcinosis, (4) Asymptomatic patients who are able to carry out medical surveillance, (5) Asymptomatic patients who want surgical therapy.[17]

In the case of patients undergoing surgery with an indication of age <50 years, increase in serum calcium level that exceeds 1.0 mg/dL from the upper limit of the reference interval, asymptomatic patients who are able to carry out medical surveillance and asymptomatic patients who want surgical therapy.

Complications of parathyroid adenoma can be divided into complications due to untreated parathyroid adenoma and complications due to resection of parathyroid adenoma. Complications of untreated parathyroid adenoma are hypercalcemia.[14] Complications due to parathyroid adenoma resection can include laryngeal nerve injury, which can have

consequent abnormal voice changes/hoarseness or airway occlusion, postoperative hypocalcemia and hypoparathyroidism.[18] Hungry bone syndrome is a relatively uncommon complication of parathyroidectomy for primary hyperparathyroidism associated with preoperative high bone turnover. It is characterised by a rapid, profound and persistent hypocalcaemia associated with hypophosphatemia, hypomagnesaemia and is exacerbated by suppressed PTH levels.[19]

CONCLUSION

Patients with asymptomatic associated elevated serum calcium levels and parathyroid hormone may have a parathyroid adenoma. A comprehensive and accurate approach to hypercalcemia is crucial in tracing the condition of hyperparathyroidism. There is an urgency to exclude other differential diagnoses associated with elevated calcium and PTH levels. Adenomas of the parathyroid glands are the leading cause of primary hyperparathyroidism, which can be confirmed by further investigation. MSCT is one of the best modalities for diagnosing parathyroid adenoma and localization of the adenoma. Localization of the adenoma site is necessary before performing parathyroidectomy, which is the only definitive treatment in this case.

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