Postural Shortening Due to Primary Hyperparathyroidism Caused by Parathyroid Adenoma

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ABSTRACT

Osteoporosis can be primary or secondary. Secondary osteoporosis is the result of an underlying disease such as an endocrine abnormality, and an example of such is primary hyperparathyroidism. The most common cause of primary hyperparathyroidism is parathyroid gland adenoma. The diagnosis of primary hyperparathyroidism is based on the following biochemical examinations: parathyroid hormone, serum calcium, creatinine clearance, 24 hour urinary calcium, and another examination such as parathyroid gland scan.

This is a rare case of an adult man who presented with a chief complaint of decreasing body height, back pain, difficulty in taking deep breaths and difficulty in his activities. The patient was diagnosed with primary hyperparathyroidism caused by parathyroid gland adenoma. His complaint was reduced after parathyroidectomy. His new complaint was that his tooth can be pulled out easily. We found high levels of parathyroid hormone and low levels of serum calcium caused by secondary hyperparathyroidism.

Key words: osteoporosis, primary hyperparathyroidism, adenoma

INTRODUCTION

Osteoporosis is characterized by scanty bone mass and micro-architectural deterioration of bone tissue, leading to enhanced bone fragility and increasing possibility of the risk of fracture. The consequences of osteoporotic fracture include diminished quality of life, decreased functional independence, and increased mortality and morbidity.^{1,2} Generalized osteoporosis can be primary or secondary. Primary or idiopathic osteoporosis occurs in postmenopausal women and in persons older than 60 years. Secondary osteoporosis is the result of an underlying disease such as an endocrine abnormality, neoplasm or drugs that adversely affect bone. One of such endocrine abnormalities is primary hyperparathyroidism. The most common cause of this abnormality is adenoma of the parathyroid gland.⁴ A report from Mayo clinic has suggested that the incidence of primary hyperparathyroidism is approximately 27.7 cases/100.000 persons/year.^{2,3,4}

The symptoms of primary hyperparathyroidism disappear if the abnormal parathyroid tissue is removed. Since this is the only definitive approach to primary hyperparathyroidisism, surgery is an acceptable approach to this disease.^{5,8,9}

Illustration of this case is an adult man who came to the hospital with a complaint of decreasing body height, back pain, difficulty in taking deep breaths and his activities. The patient was diagnosed to have a primary hyperparathyroidism caused by parathyroid gland adenoma. This case is being important to be reported due to it being rarely found in Indonesia.

CASE ILLUSTRATION

A 33 year-old male came to Cipto Mangunkusumo Hospital with shortening of body height since 6 months ago as the chief complaint.

His height was reduced 30 cm gradually since 6 months, due to back pain, difficulty in sitting, moving, and lying, he spends most of his time in standing position. There was no history of trauma nor of consumption of drugs such as corticosteroid, anticonvulsant, etc. There was no history of gastrointestinal disturbance or malnutrition.

During the last one month, he has had difficulty breathing, especially when trying to take a deep breath.

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There was no history of trauma as well as other complaint.

His general condition was moderate with normal vital signs. His body weight was 30 kg, and his body height was 139 cm. The chest inspection showed pectus carinatus. There was no abnormality found in other examinations (Figure 1).



Figure 1. Shortening Body Height and Disproportional Posture

The laboratory results were as follows: calcium ion 1.81 mmol/l, phosphate alkaline 5.210 U/L, parathyroid hormone (PTH) 1.813 pg/ml, urine calcium 278 mg/24 hrs, urine organic phosphor 0.3 g/24 hrs, creatinine clearance test 136.68 ml/m. Other examinations: thoracolumbal X-ray: osteoporosis with vertebral corpus compression; parathyroid ultrasound: right parathyroid nodule. Parathyroid scan: parathyroid nodule with increased uptake, suspect adenoma (Figure 2). Bone density mass: T score –6.35 (arm), -8.91 (L2), -7.61 (femur neck). Spirometry: severe lung restriction.

This patient underwent lower right parathyroidectomy based on the clinical diagnosis of primary hyperparathyroidism suspected to be caused by parathyroid adenoma. Before the surgery, the patient was given biphosphonate orally. The histological report was that the lesion was consistent with parathyroid adenoma. There was no sign of malignancy and no tumor mass was left. During the follow up the former complaint was reduced but new complaint arose. His tooth can be pulled off easily. The laboratory result: calcium ion 0.88 mmol/l, phosphate alkaline 361 U/l, anorganic phosphor 4.6 mg/dl, PTH 210 pg/ml. Based on the diagnosis of secondary parathyroidism, this patient received calcium supplementation.

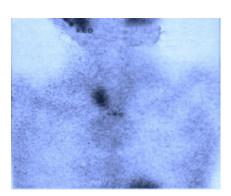


Figure 2. Parathyroid Nodule with Increased Uptake, Suspected Adenoma

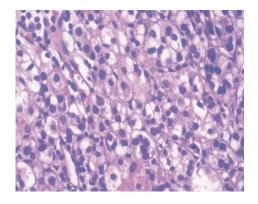


Figure 3. Parathyroid Adenoma. The cells are arranged in a compact mass and some in acinar arrangement. All cells looked similar without mitosis and clear cytoplasm consistent with hormone-producing cells.

DISCUSSION

The patient presented with decreasing body height since 6 months prior to admission. The most common cause of postural shortening in young patients is secondary osteoporosis caused by endocrine disorders, neoplasia, gastrointestinal disturbance, drugs, and other miscellaneous causes. In this patient, the postural shortening was thought to be caused by vertebral compression due to generalized osteoporosis. From the laboratory result, we found hypercalcemia. In the differential diagnosis of hypercalcemia, primary hyperparathyroidism is the most common cause. It is important to distinguish with other causes of hypercalcemia such as malignancy, by measuring PTH levels. Parathyroid adenoma was found in this patient, likely to have caused primary hyperparathyroidism, with generalized osteoporosis as its main clinical feature.^{3,5}

The clinical features of this patient that contributed to the diagnosis of primary hyperparathyroidism were musculosceletal impairment, generalized osteoporosis, confirmed by thoracolumbal x-ray and bone mass density examination, increased parathyroid hormone, and hypercalcemia. From the ultrasound examination, there was nodule in the right parathyroid gland. The parathyroid gland scan also concluded that there was a nodule with increase uptake in the right parathyroid gland that was suspected as adenoma. From the literature, it is said that the most common lesion found in patients with primary hyperparathyroidism is solitary parathyroid adenoma, occurring in 80% of patients.^{1,2,5,6}

Another examination that was important to be examined in such patients is 1,25-OH2 vitamin D levels, because it is correlated to the dietary calcium intake. If the level is increased, daily calcium intake should be restricted.⁵

The urine calcium level in this patient was normal. This could have been caused by the inappropriate procedure of urine collection, since the patient collected his 24 urine sample by himself, so there was no supervision to the procedure. On the other hand, the cohort study about biochemical values in primary hyperparathyroidism performed by Silverberg indicated that in several patients, normal 24-hour urine calcium level was found.^{5,67,9}

The patient has undergone parathyroidectomy, as his conditions matched the NIH criteria. This therapeutic procedure is the only definitive therapy for parathyroid adenoma. Before parathyroidectomy, he was given biphosphonate orally. In patients with primary hyperparathyroidsm, biphosphonate does not affect parathyroid hormone level directly, but it could reduce serum and urinary calcium levels.⁵ Post-parathyroidectomy, the patient has a new complaint, that his tooth can be pulled out easily, and from laboratory tests we found high levels of PTH and low level of calcium. This complaint was caused by secondary hyperparathyroidism, and he received calcium supplementation.^{5,8,9}

For patients who refuse surgery, there is drug called calcimimetics that can decrease parathyroid hormone and calcium levels by acting as agonist to the parathyroid cell calcium sensing receptor. The drug still needs further studies to confirm the efficacy of this drug in inducing sustained reduction in parathyroid hormone and the serum calcium without parathyroidectomy.⁵

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