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CLINICAL CASE REPORT

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A Case of Solitary Kidney Atrophy Due to Primary Hyperparathyroidism

A Case Report

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Abstract: Although primary hyperparathyroidism (PHPT) is asymptomatic in most patients, its main clinical manifestation is nephrolithiasis. In general, hypercalcemia would lead to unilateral renal stones, which may become bilateral over time. We present a rare case of a large unilateral asymptomatic ureteral stone in a patient with hypercalcemia secondary to PHPT, which eventually led to renal atrophy.

The diagnosis of PHPT should be considered in patients with hypercalcemia and renal stones, as asymptomatic PHPT may result in a devastating renal outcome.

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Abbreviations: Ca = calcium, Cr = creatinine, CT = computed tomography, eGFR = estimated glomerular filtration rate, FECa = fractional excretion of calcium, PHPT = primary hyperparathyroidism, PTH = parathyroid hormone.

INTRODUCTION

Primary hyperparathyroidism (PHPT) results from overproduction of parathyroid hormone (PTH) from the parathyroid glands. PHPT occurs mainly from parathyroid dysplasia, parathyroid adenomas, or even malignancy affecting 1 or more of the parathyroid glands. PHPT is more common during middle age, although it can occur among children and the elderly; however, the prevalence is higher among postmenopausal women.[1] Elevated PTH levels cause a disruption in calcium homeostasis promoting bone resorption, which induces hypercalcemia. During routine laboratory tests, PHPT may be diagnosed incidentally as hypercalcemia because most patients usually are asymptomatic.[2]

The common clinical presentation in symptomatic patients with no previous history of renal pathology is nephrolithiasis and symptoms of renal disease. The cardiovascular, neuromuscular, and gastrointestinal systems also may be involved, leading to various symptoms.[3] Psychological disturbances have also been noted. Nephrolithiasis occurs in ~7% to 40% of patients with PHPT.[4,5] Approximately 5% of patients with nephrolithiasis will have hyperparathyroidism.[6] A large staghorn calculus will develop in some cases if early intervention is not undertaken. We present an interesting case of unilateral renal atrophy due to a silent right-side dominant stone in a patient with PHPT.

CASE REPORT

A 54-year-old previously healthy woman was diagnosed with hyperuricemia and right hydronephrosis at a local medical department. She had no remarkable past medical history and no regular calcium or vitamin D consumption. She was referred to our center for further evaluation.

At admission, physical examination showed no significant finding. Kidney sonography disclosed right hydronephrosis with 1 large ureteral stone (~1.5 cm). Biochemistry data revealed an elevated blood ionized calcium (Ca) level (8.38 mg/dL; reference 3.68–5.6 mg/dL), low phosphate level (1.6 mg/dL; reference 2.5–5 mg/dL), and impaired renal function (blood nitrogen, 29 mg/dL [reference 7–25 mg/dL]; creatinine [Cr], 1.4 mg/dL [reference 0.5–1.3 mg/dL]; and estimated glomerular filtration rate [eGFR], 39.9 mL/min/1.73 m^2). Other laboratory data showed sodium 138 mEq/L (reference 133–145 mEq/L), potassium 4.7 mEq/L (reference 3.3–5.1 mEq/L), uric acid 6.8 mg/dL (reference 2.3–6.6 mg/dL), 25-hydroxyvitamin D 23.8 ng/mL (reference 20–100 ng/mL), high urine calcium excretion rate (urine Ca/Cr) 0.39 mg/mg (reference 0.05–0.1), fractional excretion of calcium (*FECa*) 9.58% (reference 1%–2%), and normal tumor markers survey (CEA 2.3 ng/mL; CA125 3.9 U/mL; and CA19 < 0.8 U/mL). Moreover, the urinalysis revealed pH 6.0 (reference 5–8) and no evidence of pyuria. Presumed diagnosis was hyperparathyroidism based on the patient's high serum calcium level and urine calcium excretion rate, and it was confirmed further by an elevated intact parathyroid hormone (iPTH) level (457 pg/mL; reference 12–72 pg/mL).

The patient received isotonic saline hydration at 2500 cc per day and an intravenous loop diuretic of furosemide at 20 mg every 8 h for the immediate management of hypercalcemia. Moreover, 1 subcutaneous injection of 60 mg denosumab (a receptor activator of nuclear factor-kappa B ligand inhibitor) was administered to impede the calcium releasing from the bone.[7] Thus, the serum ionized calcium level decreased to 4.7 mg/dL in 5 days and the renal function (serum Cr level)

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Ethical statement: Ethical approval was not necessary as our study was focused on the retrospective observation of a patient's hospital course, which in no way affected his treatment. Informed consent was obtained from the patient regarding the reporting and publication of this case report.
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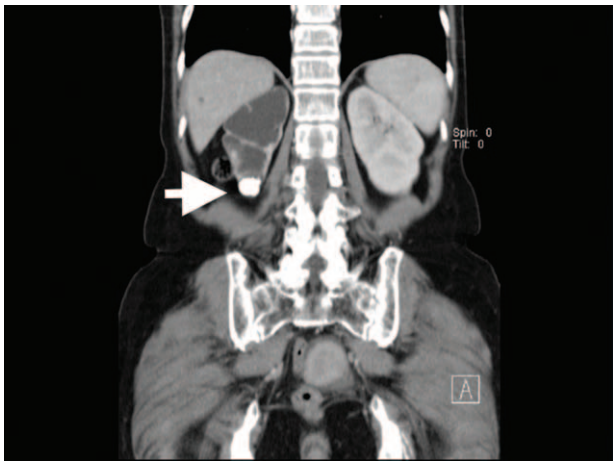

FIGURE 1. Abdominal CT with contrast shows right renal atrophy with hydronephrosis and nephrolithiasis with the largest being 1.5 in size in the lower ureter (arrow). CT = computed tomography.

returned to the normal range. Computed tomography (CT) with contrast was performed to evaluate the right hydronephrosis. The right renal sac appeared as an atrophic area at the lower pole with focal ureteral wall thickening associated with luminal stenosis at the upper segment and a few stones, the largest being ~1.5 cm in size in the lower ureter (Figure 1). Moreover, the abdominal CT showed no right renal artery stenosis. Sestamibi scintigraphy of the parathyroid glands confirmed a parathyroid adenoma in the left upper thyroid bed (Figure 2).

Thus, PHPT was confirmed and a general surgeon and urologist were consulted for further management. Parathyroidectomy with partial thyroidectomy and right nephrectomy were performed sequentially. Pathological evaluation of the resected specimens revealed adenoma of the parathyroid gland and normal thyroid tissue. A postoperative 6-month follow-up revealed normal serum calcium levels, iPTH levels (28.18 pg/mL), and renal function tests, and a low calcium secretion rate (FECa: 0.2%).

DISCUSSION

PHPT and malignancy contributes to >90% of cases of hypercalcemia. Early diagnosis can provide the patient with optimal therapy and accurate prognostic information. Malignancy often implies a poor life expectancy. However, PHPT has a relatively benign course. The etiology of hypercalcemia can be classified into several categories, including hyperparathyroidism, vitamin D-related causes, malignancy, medications, other endocrine disorders, genetic disorders, and miscellaneous causes.[8] Figure 3 illustrates how to evaluate a patient with hypercalcemia.[8–10] The high serum calcium level with a high urine calcium secretion rate in our patient indicated overactivity of PTH. Imaging studies verified the diagnosis of PHPT.

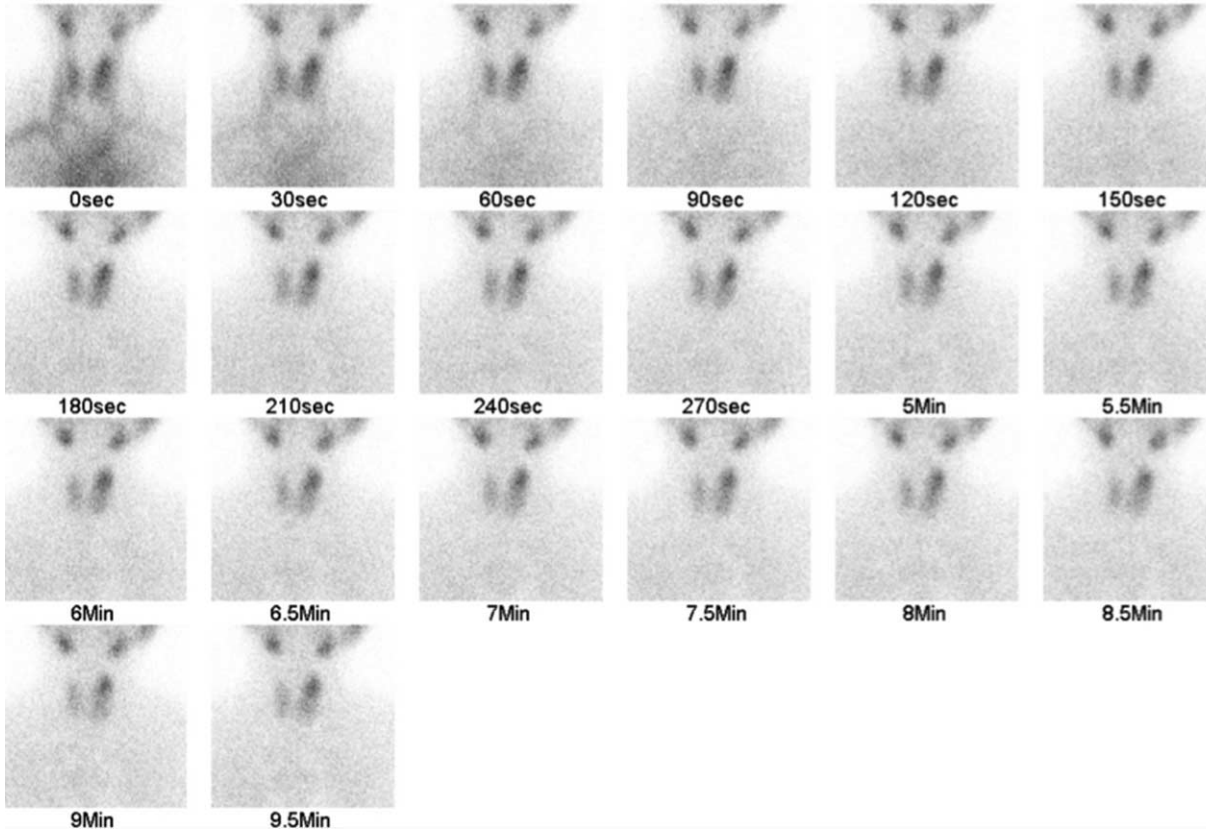

FIGURE 2. Radioactive parathyroid scan reveals increased radioactivity in the left upper thyroid bed. The washout phase shows abnormal local uptake in the left upper thyroid bed. The scintigraphic findings could be compatible with parathyroid adenoma detected in the left upper thyroid bed.

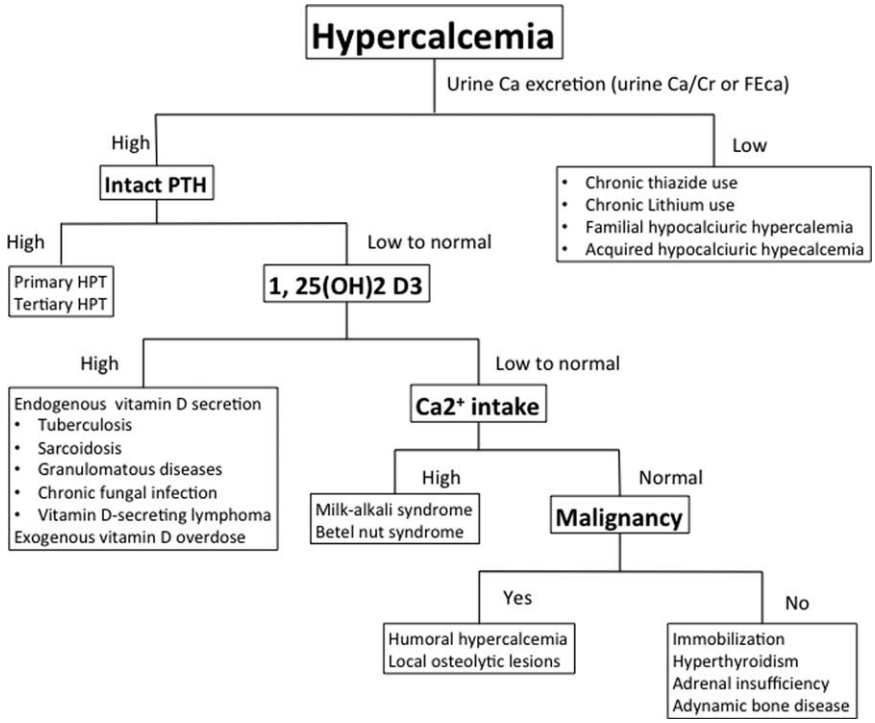

FIGURE 3. Common causes of hypercalcemia.[7–9] Ca = calcium, FEca = fraction excretion of calcium, HPT = hyperparathyroidism, PTH = parathyroid hormone.

Nephrolithiasis is a common presentation in patients with PHPT. If the stone is large enough to cause blockage of ureter, pain may be the first clinical symptom of PHPT. Nephrolithiasis also is associated with urinary tract infection, obstruction, and loss of renal function. Because of the recent improvements in imaging techniques, such as sonography and CT, renal stones may be detected at an early stage. Suh et al[11] reported that renal stones in patients with PHPT were unilateral, without associated hydronephrosis, and asymptomatic, and the stones ranged in size from 3 to 20 mm. This suggests that at an early stage, renal stones are unilateral and small. Renal stones tend to start within 1 kidney and over time involve both kidneys. However, only large renal stones will produce symptoms. A single large renal stone due to PHPT is uncommon.[11,12] To the best of our knowledge, this is the first report of a patient with right kidney atrophy due to silent nephrolithiasis induced by PHPT. We hypothesized that the right renal atrophy observed in our case would lead to an increase in the urine flow of the left kidney, decreasing the risk of renal stone formation on that side. Therefore, we assumed that our case presented with only a large unilateral ureteral stone in the setting of hypercalciuria.

The revised guidelines for the management of asymptomatic PHPT[13] recommend parathyroid surgical intervention in patients presenting with an increased serum calcium level >1 mg/dL above the upper limit of normal, a kidney stone, and a decrease in Cr clearance to <60 mL/min. In our patient, the parathyroid gland was excised due to impaired renal function, renal stone, elevated serum calcium, and the presence of a parathyroid adenoma. Moreover, right nephrectomy was performed in our case for the prevention of further urinary tract infection and squamous cell carcinoma, which may be induced by the renal stone.[14–17]

CONCLUSIONS

We report an unusual case of right nephrolithiasis and renal atrophy. As a single clinical symptom, it is less likely connected with PHPT. In addition, in the clinical practice, we probably would miss the diagnosis of PHPT. Thus, we stress that the diagnosis of PHPT should be considered even in patients with only unilateral nephrolithiasis.

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