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Case Report

Primary Hyperparathyroidism: A Case Report

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Abstract

Primary hyperparathyroidism (PHPT) is a common endocrine disorder, with prevalence of one to seven cases per 1000 adults. It is believed to be the most common cause of hypercalcemia, predominantly affecting elderly populations and women two to three times as often as men. Here we report a case of 75 year old male who presented to Medicine OPD with complaints of constipation, abdominal pain and occasional irrelevant talks. Investigation showed hypercalcemia. PTH levels were markedly raised with borderline 1,25 DIHYDROXYVIT D. Patient was dialysed and managed conservatively. Later follow-up reports revealed microoadenoma and underwent surgery.

Keywords: Primary hyperparathyroidism (PHPT), endocrine disorder, Medicine OPD, hypercalcemia, Patient.

INTRODUCTION

Hyperparathyroidism is due to increased activity of the parathyroid glands, either from an intrinsic abnormal change altering excretion of parathyroid hormone (primary or tertiary hyperparathyroidism) or from an extrinsic abnormal change affecting calcium homoeostasis stimulating production of parathyroid hormone (secondary hyperparathyroidism). Primary hyperparathyroidism (PHPT) is the third most common endocrine disorder. It has highest incidence in postmenopausal women. Asymptomatic disease is common, and severe disease with renal stones and metabolic bone disease arises less frequently nowadays [1]. There are striking discrepancies around the world with respect to incidence, symptoms, and complications of PHPT. In developing countries, particularly India, PHPT is still an uncommonly diagnosed, overtly symptomatic disease of "bones, stones, abdominal groans, and psychic moans." This may be because, in India, screening of the healthy population for hypercalcemia is not routinely done and there is limited access to medical treatment. Contemporary series of patients with PHPT from developed nations are largely dominated by elderly females with mild to moderate hypercalcemia and very few with classical symptoms, contrary to the clinical picture seen in developing countries, especially India [2].

Here, we present a case of 75 yr old male who presented to OPD with complaints of constipation, abdominal pain and occasional irrelevant talks.

CASE REPORT

A 75yr old male patient presented to General Medicine OPD with complaints of constipation for 4 days, abdominal pain for 4 days and having history of occasional irrelevant talks. Pt was drowsy and having delayed response. RBS was low and was corrected by dextrose infusion. Attendants gave history of OHA intake 2 days back due to raised RBS by a local practioner. There was no H/O T2DM in past. Xray abdomen erect/ supine were done which were normal. General Examination revealed- HR- 90/min, RR- 22/min, BP- 130/90mmhg. Systemic examination- Per abdomen, Respiratory, Neurological Examination were grossly normal. His investigations revealed-

HB- 15.5gm/dl	TLC- 9400cells/mm^3	PLATELETS- 85000 cells/mm^3
UREA- 92mg/dl	CREATININE- 2.8mg/dl	SODIUM- 136mmol/lt
POTASSIUM- 2.6mmol/lt	CALCIUM 14.70mg/dl	PHOSPHORUS- 2.80mg/dl
BILIRUBIN- 1.1/0.4/0.7	SGOT/SGPT- 37/30 IU/lt	T.PROT- 7.50gm/lt
ALBUMIN- 4.30gm/lt	GLOBULIN- 3.20 gm/lt	

ECG S/O SHORT QT INTERVAL, 2D ECHO- LVEF 55%, Rest WNL. Urgent NEPHROLOGY opinion was taken and patient was advised Hemodialysis. IVF fluids were given and hourly urine output was measured. Patient was dialysed and repeat S.Ca was- 13.9meq/l . Patient was further investigated for the cause of hypercalcemia and investigations were as follows-

iPTH- 425 pg/ml	S.ACE- 21 U/L	1,25, HYDROXYVIT D- 30.3 pg/ml
Myeloma panel- NOT DETECTED		

Patient improved symptomatically and continuous IV fluids were given to prevent dehydration.

A diagnosis of primary hyperparathyroidism was made. Patient stayed with us for a period of 3 days and wanted to be referred to higher centre for further management and was referred accordingly.

Patient was followed up over telephonic conversation and review on OPD consultations. Patient was admitted outside where Sestamibi scan showed a parathyroid adenoma in left inferior parathyroid and he underwent Left inferior parathyroidectomy after which he was symtomatically better. Patient was discharged on calcitriol and calcium.

DISCUSSION

Primary hyperparathyroidism is a disorder of the parathyroid glands, four pea-sized glands located near the thyroid gland in the neck. “Primary” means that disorder begins in the parathyroid glands, rather than resulting from another health problem such as kidney failure. In primary hyperparathyroidism, one or more of the parathyroid glands is overactive. As a result, the gland makes too much parathyroid hormone (PTH) [3]. PHPT is defined as hypercalcemia with increased or inappropriately normal plasma parathyroid hormone (PTH). It is most commonly seen after the age of 50 years, with women predominating by three to fourfold [4].

Less than 20% of patients may present with overt symptoms. Occasionally, patients present with pain from a fracture or from renal colic. Obtundation, neuromuscular weakness, or both from severe hypercalcemia are very uncommon and are usually caused by large adenoma. Moderate to severe hypercalcemia can cause constipation and is a risk factor for pancreatitis. Dehydration or immobilization can worsen hypercalcemia. Fatigue, depression, and impaired memory are not infrequent, but a causal link between these conditions and parathyroid disease is uncertain. In health care settings, where there is limited resources, patients present with higher serum calcium levels and are more often symptomatic [5].

Primary hyperparathyroidism, due to autonomous hypersecretion of PTH, usually occurs in the setting of a parathyroid adenoma (80%) but can also be seen with parathyroid gland hyperplasia (15%–20%) or carcinoma (<0.5%). Stimulation of the parathyroid glands as a response to hypocalcemia or apparent insensitivity of the parathyroid glands to elevated serum calcium levels results in secondary hyperparathyroidism and dysregulation of the normal negative feedback loop (pseudohypoparathyroidism). The most common cause of secondary hyperparathyroidism is renal failure, which results in phosphate retention, hypocalcemia, and $1,25(OH)_2D_3$ deficiency, leading to a compensatory increase in the production of PTH. Tertiary hyperparathyroidism is seen in cases of secondary hyperparathyroidism in which the parathyroid glands continue to function autonomously despite correction of the initial cause, resulting in hypersecretion of PTH in the setting of normal calcium levels [6].

DIAGNOSIS: Hypercalcemia in conjunction with abnormally elevated parathyroid hormone levels makes PHPT the most likely diagnosis. Calcium and parathyroid hormone levels should be tested at the same time because individual levels fluctuate quickly. Repeated calcium measurements may be required since hypercalcemic patents may occasionally have normal calcium levels. Measurement of serum calcium should be adjusted for albumin, as 40% of calcium is bound to serum proteins, predominantly albumin. If the adjusted serum calcium is normal but parathyroid hormone is elevated, serum ionized calcium should be measured. PHPT can present with an elevated ionized calcium despite a normal albumin-adjusted serum calcium [7].

TREATMENT: Surgery to remove abnormal parathyroid tissue is the only known cure for PHPT. Symptomatic patients with PHPT should be advised to undergo surgery. Surgery is also recommended for asymptomatic patients with the following indications:

- Age < 50 years
- Serum calcium > 1 mg/dL (> 0.25 mmol/L) above upper limit of normal

- Bone mineral density T-score of ≤ 2.5 (osteoporosis) or a low-energy fracture on imaging study.
- Creatinine clearance reduced to < 60 mL/min, or 24-hour urine for calcium > 400 mg/day and increased stone risk by biochemical stone risk analysis, or nephrolithiasis or nephrocalcinosis on imaging study
- Even when there is no specific indication for surgery, it is an established and appropriate treatment because it is the only known cure.
- Situations that may prompt nonsurgical management include:
- First trimester pregnancy
- Severely limited cervical access
- Prior vocal cord paralysis
- Short expected lifespan

Currently, the only medication shown to lower serum calcium in patients with PHPT is the calcimimetic agent cinacalcet. Cinacalcet normalizes serum calcium in 70% to 80% of patients with PHPT. However, it has not been shown to impact bone mineral density, hypercalcemic symptoms, kidney stones, or quality of life.

Bisphosphonates may be used in combination with cinacalcet in patients with T-scores ≤ 2.5 at the lumbar spine, hip, or one-third radius, or who have fragility fractures. These agents have been shown to be effective in preventing decreases in bone mineral density and lowering bone remodeling [7].

Our patient had left posteroinferior parathyroidectomy, on follow up the patient general condition was well, with no abdominal complaints, with serum calcium levels in decreasing trend.

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