A 31-Year-Old Man with Primary Hyperparathyroidism Due to Parathyroid Adenoma: A Case Report

Md. Mahfuzer Rahman,[1] Zeenat Faruquey,[2] Golam Rabbani,[3] Akter Banu,[4] Md. Abul Kalam Azad,[5] Md. Jahangir Kabir,[6] ASM Shafiujjaman[7]

1. Professor
   Department of Medicine
   Rangpur Medical College, Rangpur, Bangladesh
2. MD Resident, Phase B (Internal Medicine)
   Rangpur Medical College, Rangpur, Bangladesh
3. Assistant Professor
   Department of Rheumatology
   Rangpur Medical College, Rangpur, Bangladesh
4. Assistant Professor
   Department of Pediatrics
   Rangpur Medical College, Rangpur, Bangladesh
5. Associate Professor
   Department of Medicine
   Rangpur Medical College, Rangpur, Bangladesh
6. Assistant Professor
   Department of Medicine
   Rangpur Medical College, Rangpur, Bangladesh
7. Junior Consultant
   Department of Medicine
   Pirganj Upazila Health Complex, Rangpur, Bangladesh

Correspondence to:
Md. Mahfuzer Rahman
Professor
Department of Medicine
Rangpur Medical College, Rangpur, Bangladesh
E-mail: mahfuz66466@yahoo.com

Submission Date : 16 June 2024
Accepted Date : 24 August 2024
Published Date : 30 September 2024
DOI:https://doi.org/10.3329/jrpmc.v9i2.77372

Abstract

Primary hyperparathyroidism (PHPT) is an endocrine disorder characterized by chronic elevation of serum concentrations of calcium and parathyroid hormone (PTH).The most frequent forms of clinical presentation are symptomatic renal or skeletal disease with moderate or severe hypercalcemia. The biochemical profile of PHPT is characterized by hypercalcemia and high or inappropriately normal PTH concentrations. Parathyroidectomy is the only definitive cure. Medical treatment with the calcimimetic cinacalcet has been shown to normalize calcemia in a high percentage of cases. Our patient presented with recurrent vomiting, abdominal pain, electrolyte imbalance, renal impairment, bilateral nephrocalcinosis, hypercalcemia with high intact PTH, osteoporosis and right parathyroid adenoma and we go for surgical intervention.

Keywords: Primary hyperparathyroidism, Hypercalcemia,Parathyroid adenoma, Nephrocalcinosis, PTH

Citation: Rahman MM, Faruquey Z, Rabbani G, Banu A, Azad MAK, Kabir MJ, Shafiujjaman ASM.A 31-Year-Old Man with Primary Hyperparathyroidism Due to Parathyroid Adenoma: A Case Report. J Rang Med Col. 2024 Sep;9(2):94-97.doi: https://doi.org/10.3329/jrpmc.v9i2.77372

Introduction:

Primary hyperparathyroidism (PHPT) is a rare clinical condition caused by an autonomous secretion of parathyroid hormone caused by a single parathyroid adenoma, hyperplasia, or cancer.[1,2] Patients usually present with evidence of primary hyperparathyroidism, elevated serum calcium and parathyroid hormone levels. Primary hyperparathyroidism leads to osteoporosis, ureteric calculi, and hypercalcemia.[3] PHPT due to parathyroid adenomas need surgical resection of the adenomas; therefore, surgical management of PHPT is recommended. All PHPT patients who meet criteria for surgery should undergo parathyroidectomy.[4] The Fourth International Workshop and American Association of Clinical Endocrinologists recommend parathyroidectomy in asymptomatic patients who meet one or more of the following criteria: age <50 years, serum calcium level >10 mg/dL above normal, urinary calcium level >400 mg/24 hours, 30% or more decrease in renal function, or complications of primary hyperparathyroidism such as nephrocalcinosis, osteoporosis, or a severe psycho-neurologic disorder.[5]

In this case report, a 31-year-old man exhibited a clinical presentation characterized by recurrent vomiting, abdominal pain, electrolyte disturbances, renal dysfunction, bilateral nephrocalcinosis, hypercalcemia accompanied by

elevated intact parathyroid hormone levels, osteoporosis, and a right parathyroid adenoma, prompting the decision to pursue surgical intervention.

Case report:

A 31-year-old man was referred to our hospital for evaluation of recurrent vomiting, abdominal pain and bilateral nephrocalcinosis. For vomiting he was admitted in local hospital and at that time there were hyponatremia, hypokalemia, renal impairment and bilateral nephrocalcinosis. He also complained of constipation, increase thirst and polyuria. He had no family history of such kind of illness. He had no history of taking any medications including thiazide diuretics.On general physical examination we found him lean and thin with a BMI of 17.3 kg/m^2. Examination of neck revealed normal findings, there was no thyroid enlargement or nodule. Systemic examination including fundoscopy was normal.On investigation, serum calcium level 12.10 mg/dl, (8.1-10.4 mg/dl), inorganic PO4 1.9 mg/dl, (2.5-5.0 mg/dl), intact PTH 1513.40 pg/ml,(9.0-80.00 pg/mL), 24 hour urinary calcium 284.00 mg/24 hour and urine volume 5100 ml/24 hour.His serum creatinine was 4.8 mg/dl. Liver and thyroid function tests were normal. X-ray KUB showed bilateral nephrocalcinosis (Figure-1) and USG of thyroid gland showed right parathyroid enlargement, and which was slight inhomogeneous measuring 41×17×9 mm, volume 3.5 cc. BMD scan showed a T score of -7.3 for anterior posterior iliac spine & L1-4 and -3.2 for left femur. Parathyroid SPECT-CT scan reveals increased tracer uptake in the region of upper pole of right lobe due to parathyroid adenoma (Figure-2). Our patient met the indications of surgery[4] and was gone through surgical intervention. Right hemithyroidectomy with parathyroid adenoma excision was done. During surgery PTH level was 467 pg/ml(15.0-65.0). Five minutes after surgery PTH level was 63.4 pg/ml (15.0-65.0) and fifteen minutes after surgery 34.4 pg/ml. After surgery histopathology was done and report revealed well circumscribed benign neoplasm within fibrous capsule, compressed non neoplastic parathyroid tissue was seen at edge, follicle formation was seen, and mitoses and bizarre nuclei was absent. There was no convulsive event in post operative period and patient was discharged with calcium and active vitamin D. Patient is now in good condition with regular follow up. After 1year of treatment his serum calcium level was 7.50 mg/dl, inorganic PO4 1.77 mg/dl, intact PTH 156.70pg/ml.

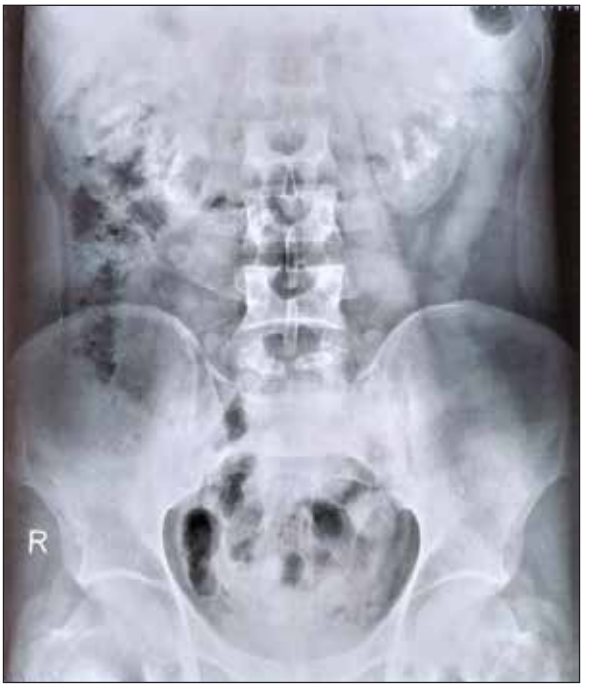

Figure-1: X-ray KUB shows bilateral nephrocalcinosis

Figure-2: SPECT CT image of 99mTc Sestamibi parathyroid scan showing increased tracer uptake in the region of upper pole of right lobe due to parathyroid adenoma.

Discussion:

This case highlighted about the rare presentation of primary hyperparathyroidism due to parathyroid adenoma. The hallmark of primary hyperparathyroidism is hypercalcemia, hypophosphatemia with detectable or elevated intact PTH levels during hypercalcemia. Elevated serum calcium levels along with high intact PTH levels and a positive parathyroid sonogram confirmed the diagnosis of PHPT for this patient. The prevalence of primary hyperparathyroidism is about 1 in 500 and 2-3 times more common in women than men. 90% of patients are over 50 years of age. It also occurs in familial MEN syndrome where parathyroid adenoma most frequently found in which 90% of patients by age 40.[6,7] Primary hyperparathyroidism is usually asymptomatic and occasionally presents with mild hypercalcemia and sometimes diagnosed incidentally on routine investigations for minor nonspecific symptoms and sometimes patients may present with polyuria, abdominal pain, increase thirst, peptic ulcer disease, hypercalcemia, nephrolithiasis, and osteitis fibrosa cystica.[8-10] Although majority of the patients of primary hyperparathyroidism are asymptomatic, some present with features of hypercalcemia and end organ damage such as osteoporosis, renal calculi, nephrocalcinosis, renal impairment. The main manifestations of our patient were polyuria, increase thrust, recurrent abdominal pain, on evaluation we found bilateral nephrocalcinosis, hypercalcemia, increase parathyroid hormone level, osteoporosis, renal impairment. There are few medical therapies at present for primary hyperparathyroidism but they are not alternative to surgery. Surgery should be performed if the criteria of surgical intervention met.[4,5] New therapeutic agents which target the calcium sensing receptors (e.g. cinacalcet) are of proven value in primary hyperparathyroidism where surgical intervention is contraindicated. For patients with PHPT and osteoporosis (or those with low bone mineral density [BMD] that would warrant intervention) who prefer to avoid surgery, bisphosphonates are suggested. In patients who are unable to tolerate bisphosphonate therapy, denosumab may be used as an alternative. However, limited evidence supports denosumab use in patients with PHPT, and risk of accelerated bone loss and vertebral fracture after discontinuation of denosumab. Drugs in development include calcitriol analogs that inhibit PTH secretion directly but do not stimulate gastrointestinal calcium or phosphate absorption and drugs that block the PTH receptor.[11] Similar type of case was reported from Mymensingh Medical College where 32-year-old lady presented with recurrent upper abdominal pain, polyuria, polydipsia and generalized body ache; was published in PubMed in 2014.[12]

Conclusion:

This case illustrates that hypercalcemia and an elevated intact PTH level in PHPT caused by single parathyroid adenoma. We should think of PHPT when someone presents with recurrent vomiting, abdominal pain and polyuria when usual causes are excluded.

Consent:

For publishing this case report and any related photos, the patient's written informed consent was acquired.

Conflict of interest:

The authors have no conflicts of interest to declare.

References:

1. Marx SJ. Hyperplasia in glands with hormone excess. EndocrRelat Cancer. 2016 Jan;23(1): R1-14. doi: 10.1530/ERC-15-0171.
2. Lee F, Lee JJ, Jan WC, Wu CJ, Chen HH, Cheng SP. Molecular pathways associated with transcriptional alterations in hyperparathyroidism. Oncol Lett. 2016 Jul;12(1):621-626. doi: 10.3892/ol.2016.4623
3. Silverberg SJ, Shane E, Jacobs TP, Siris E, Bilezikian JP. A 10-year prospective study of primary hyperparathyroidism with or without parathyroid surgery. N Engl J Med. 1999 Oct 21;341(17):1249-1255. doi: 10.1056/ NEJM 199910213411701.
4. Bilezikian JP, Brandi ML, Eastell R, Silverberg SJ, Udelsman R, Marcocci C, et al. Guidelines for the management of asymptomatic primary hyperparathyroidism: summary statement from the Fourth International Workshop. J Clin Endocrinol Metab. 2014 Oct;99(10):3561-3569. doi: 10.1210/jc.2014-1413.
5. AACE/AAES Task Force on Primary Hyperparathyroidism. The American Association of Clinical Endocrinologists and the American Association of Endocrine Surgeons position statement on the diagnosis and management of primary

hyperparathyroidism. Endocr Pract. 2005 Jan-Feb;11(1):49-54. doi: 10.4158/EP.11.1.49.
6. Almeida MQ, Stratakis CA. Solid tumors associated with multiple endocrine neoplasias. Cancer Genet Cytogenet. 2010 Nov;203(1):30-6. doi: 10.1016/j.cancergency to. 2010. 09. 006.
7. Ito T, Igarashi H, Uehara H, Berna MJ, Jensen RT. Causes of death and prognostic factors in multiple endocrine neoplasia type-1: a prospective study: comparison of 106 MEN1/Zollinger-Ellison syndrome patients with 1613 literature MEN1 patients with or without pancreatic endocrine tumors. Medicine (Baltimore). 2013 May;92(3): 135-181. doi: 10.1097/MD. 0b013e 3182954af1.
8. Thakker RV, Newey PJ, Walls GV, Bilezikian J, Dralle H, Ebeling PR, Melmed S, Sakurai A, Tonelli F, Brandi ML; Endocrine Society. Clinical practice guidelines for multiple endocrine neoplasia type-1 (MEN1). J Clin Endocrinol Metab. 2012 Sep;97(9): 2990-3011. doi: 10.1210/jc.2012-1230.
9. Donegan D, Singh Ospina N, Rodriguez-Gutierrez R, Al-Hilli Z, Thompson GB, Clarke BL, Young WF Jr. Long-term outcomes in patients with multiple endocrine neoplasia type-1 and pancreaticoduodenal neuroendocrine tumours. Clin Endocrinol (Oxf). 2017 Feb; 86(2):199-206. doi: 10.1111/ cen.13264.
10. Thakker RV. Multiple endocrine neoplasia type-1 (MEN1) and type-4 (MEN4). Mol Cell Endocrinol. 2014 Apr 5;386(1-2):2-15. doi: 10.1016/j.mce.2013.08.002.
11. Primary hyperparathyroidism: Management. Uptodate.2024 Aug. ? topicRef= 2050& source=related_link [Accessed on 30th May 2024]
12. Siddiqui NI, Miah AG, Khan NK, Bosri R, Ghani MH, Uddin S, Tanzeem S, Razib KH. Primary hyperparathyroidism: a case report. Mymensingh Med J. 2014 Apr;23(2):375-9.