



## HORSE SHOE KIDNEY WITH STAGHORN CALCULUS AND PARATHYROID ADENOMA : A CASE REPORT.

### General Surgery

**Dr Lisha Suraj**

Associate Professor Department Of Surgery Mahatma Gandhi Missions Medical College And Hospital, Navi Mumbai.

**Dr Pallavi S. Kadam\***

Senior Resident Department Of Surgery Mahatma Gandhi Mission's Medical College And Hospital, Navi Mumbai. \*Corresponding Author

### ABSTRACT

Parathyroid adenoma is the third most common endocrine disorder with a reported incidence of 0.25%<sup>(1)</sup>. The incidence of primary hyperparathyroidism (PHPT) increases with age, and is more common in females than in males<sup>(2)</sup>. The various presentations include hypercalcemia, multiple recurrent renal calculi, recurrent bone fractures, pancreatitis and pituitary calcification. Here we present a case of 60 year old lady presented with horseshoe kidney and staghorn calculus and later diagnosed to have parathyroid adenoma and underwent parathyroidectomy and nephrolithotomy..

### KEYWORDS

#### INTRODUCTION:

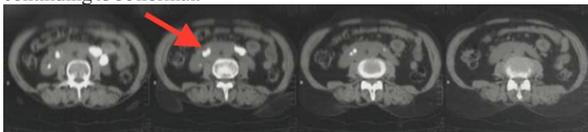
Hyperparathyroidism (HPT) can be primary, secondary and tertiary. 75–85% of cases of Primary HPT are caused by a single parathyroid adenoma<sup>(3)</sup>, the removal of which give relief to patient. It is the 3rd most common endocrine disorder with incidence of 0.25%<sup>(1)</sup>. Symptomatic Primary HPT can present with skeletal involvement (bone pain, fracture and palpable multiple Osteitis Fibrosa Cystica) renal calculi with nephrocalcinosis and renal failure, fatigue with proximal muscle weakness, pancreatitis, gall bladder stone, constipation and depression. Hence the clinical manifestations are described as kidney stones, abdominal goans, painful bones, psychic moans and fatigue.

The increase in PTH (parathyroid hormone) levels acts on kidney to increase production of calcitriol which increase the loss of phosphate and retention of calcium due to reabsorption by the gut. PTH acts on bone to cause demineralization of bone which decreases density of bone and pathological fractures. It also lead to increased excretion of urinary calcium (hypercalciuria) which is a primary risk factor for stone formation in kidney.

#### Case scenario:

60 year old female a diagnosed case of staghorn calculus in a horseshoe kidney and underwent nephrolithotomy on one side presented with a swelling over the operated site since 6 months. On enquiry patient gave history of recurrent renal calculi. On examination she was found to have a swelling on the right lower neck on supraclavicular region which was not moving with deglutition. On investigation her serum calcium level was found to be high with raised parathyroid hormone level. She underwent ultrasonography of the neck which showed a 2\*2 cm swelling on the right lower neck near the lower pole of thyroid gland. A CECT (Contrast enhanced computerized tomography) scan of the neck confirmed the swelling to be of parathyroid origin. She underwent a 99mTc (Technetium) labelled sestamibi scan which showed an increased uptake only in the right lower parathyroid gland.

After due work up & proper pre operative preparation patient was taken for surgery. A Kocher's incision was taken, flaps raised and strap muscles separated to expose the right lobe of the thyroid along with parathyroid swelling. Parathyroid adenoma of 3\*4 cm was removed, hemostasis achieved and wound closed in layers. She had an uneventful recovery. Post operative serum calcium levels and PTH levels were normal in the post operative period and on further follow up for one year. She later underwent surgery for removal of staghorn calculus on the other side and incisional hernia repair. After one year follow up patient does not have any further stone formation and serum calcium & PTH levels are continuing to be normal.



**FIGURE 1:** CT KUB showing horseshoe kidney pointed by arrow.



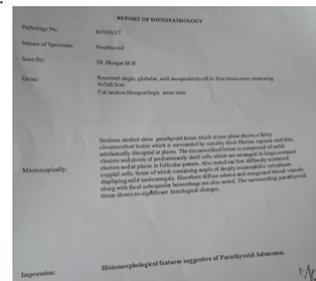
**FIGURE 2:** Tc 99m Sestamibi Scan Showing parathyroid activity at the lower pole of thyroid gland.

| Test Name                                      | Results | Units | Ref. Int.     |
|--|---------|-------|---------------|
| PTH (PARATHYROID HORMONE) INTACT, SERUM (CLIA) | 323.89  | pg/mL | 15.00 - 75.00 |

Notes:

1. Test results should be interpreted in conjunction with serum calcium and phosphorus levels, and clinical findings.
2. Elevated PTH with normal serum calcium levels may be indicative of secondary causes of hyperparathyroidism like vitamin D deficiency. It may not always be indicative of Primary hyperparathyroidism.
3. PTH is secreted in a pulsatile manner with an overall circadian rhythm characterized by a nocturnal rise.

**FIGURE 3:** Pre-operative Parathyroid Hormone Levels which were severely raised.



**FIGURE 4:** Intra-operative picture showing parathyroid adenoma.



**FIGURE 5:** Histopathology report s/o parathyroid adenoma.

## DISCUSSION

In 80-85% cases primary hyperparathyroidism is due to solitary parathyroid adenoma<sup>(4,5)</sup>, 1% to 4% due to Parathyroid carcinoma and 15% due to parathyroid hyperplasia<sup>(4,6,7)</sup>. Rarely it can be a manifestation of multiple endocrine neoplasia syndromes type I or IIa (MEN I or MEN IIa). Hyperparathyroidism occurs in approximately 95% of patients with MEN I and there can be hyperplasia of all 4 parathyroid glands in this condition. MEN I usually manifests in the second or third decade of life<sup>(8)</sup>. In MEN IIa 5% to 20% of patients are reported to have hyperparathyroidism, and the disease typically presents later in life<sup>(8)</sup>. Similar to MEN I, it is also the result of parathyroid hyperplasia<sup>(4)</sup>. The recurrence rate in such patients is higher than in those with hyperparathyroidism due to a solitary adenoma<sup>(8)</sup>.

In hyperparathyroidism, the serum PTH level is increased which increases the bone resorption, reabsorption of calcium in the nephron, and intestinal absorption leading to an increase in the serum calcium levels<sup>(8)</sup>. Broadus and colleagues<sup>(9)</sup> reported that there is higher urinary excretion of calcium in those patients with stones compared to those who do not have stones. Other risk factors of stone formation also influences the stone formation<sup>(10,11,12)</sup>. The type of stone found in patients with primary hyperparathyroidism are calcium phosphate, calcium oxalate, or mixed calcium stones<sup>(13,14)</sup>.

With improved screening programmes and diagnostic tools, patients with primary hyperparathyroidism are diagnosed early and the incidence of urolithiasis in primary hyperparathyroidism has decreased from nearly 80% in early series to 7% to 20% in recent series<sup>(15,16)</sup>.

Screening is done with estimation of serum calcium levels. If serum calcium is elevated, intact PTH and phosphorous are measured. In hyperparathyroidism, the intact PTH level is usually high-normal or elevated and phosphorous depressed. Ionized calcium is measured when the serum albumin is abnormal because this will enhance detection<sup>(17)</sup>.

(99m)Tc-sestamibi scans have been used successfully to identify the metabolically active tissues, including parathyroid adenomas. It can locate a parathyroid adenoma and facilitate minimally invasive parathyroidectomy<sup>(18,19)</sup>. It may also help in the detection of ectopic parathyroid adenomas and in the evaluation of patients suspected of having normocalcemic hyperparathyroidism<sup>(20,21)</sup>. (99m)Tc-sestamibi scans have a sensitivity of 88.44% for solitary parathyroid adenomas and 33% for carcinoma<sup>(21,22)</sup>.

Once the diagnosis is confirmed, the adenoma can be removed surgically which decreases the further stone formation and renal injury. Mollerup and colleagues examined 107 patients with renal calculi and hyperparathyroidism. After surgical correction of hyperparathyroidism, the stone recurrence rate was 30% at 5 years, significantly lower than the rate before operation and comparable to the rate of recurrence in idiopathic stone formers<sup>(23)</sup>. Deaconson and associates<sup>(24)</sup> reported similar reductions in stone events after parathyroidectomy.

## CONCLUSION

Horseshoe kidney with staghorn calculus and hyperparathyroidism due to parathyroid adenoma is a rare entity. It needs a high amount of suspicion, proper evaluation and treatment to keep the patient off the agony of the stone disease due to hyperparathyroidism which they are already prone due to congenital anomaly of the kidney.

**Source of support : None.**

**Conflict Of Interest : None.**

## REFERENCES

1. Bilezikian JP and Silverberg SJ: Clinical practice. Asymptomatic primary hyperparathyroidism. *N Engl J Med.* 350:1746–1751. 2004.
2. Udén P, Chan A, Duh QY, Siperstein A and Clark OH: Primary hyperparathyroidism in younger and older patients: Symptoms and outcome of surgery. *World J Surg.* 16:791–797; discussion 798. 1992.
3. Fraser WD: Hyperparathyroidism. *Lancet.* 374:145–158. 2009.
4. Johnson SJ, Sheffield EA, McNicol AM. Best practice no 183. Examination of parathyroid gland specimens. *J Clin Pathol.* 2005;58:338–342.
5. Siperstein AE, Shen W, Chan AK, et al. Normocalcemic hyperparathyroidism. Biochemical and symptom profiles before and after surgery. *Arch Surg.* 1992;127:1156–1157. discussion 1161–1163.
6. Rodgers SE, Perrier ND. Parathyroid carcinoma. *Curr Opin Oncol.* 2006;18:16–22. [PubMed]
7. Shane E. Clinical review 122: parathyroid carcinoma. *J Clin Endocrinol Metab.* 2001;86:485–493.
8. Larsen PR. *Williams Textbook of Endocrinology.* 10th ed. Philadelphia: Saunders; 2003.

9. AE Broadus, RL Horst, R Lang, et al. The importance of circulating 1,25-dihydroxyvitamin D in the pathogenesis of hypercalciuria and renal stone formation in primary hyperparathyroidism. *N Engl J Med.* 1980;302:421–426.
10. Khanam A, Rahman MA. Parathyroid hormone in urinary stone patients. *Mol Cell Biochem.* 1993;121:1–4. [PubMed]
11. D'Angelo A, Lodetti MG, Giannini S, et al. Hyperparathyroidism: cause or consequence of recurrent calcium nephrolithiasis? *Miner Electrolyte Metab.* 1992;18:359–364. [PubMed]
12. Odvina CV, Sakhaee K, Heller HJ, et al. Biochemical characterization of primary hyperparathyroidism with and without kidney stones. *Urol Res.* 2007;35:123–128.
13. Campbell MF, Wein AJ, Kavoussi LR, editors. *Campbell-Walsh Urology.* Philadelphia: Saunders Elsevier; 2007.
14. Thomas WC, Jr. Urinary calculi in hypercalcemic states. *Endocrinol Metab Clin North Am.* 1990;19:839–849.
15. Klugman V, Favus MJ. Diagnosis and treatment of calcium kidney stones. *Adv Endocrinol Metab.* 1995;6:117–142. [PubMed]
16. Silverberg SJ, Shane E, Jacobs TP, et al. Nephrolithiasis and bone involvement in primary hyperparathyroidism. *Am J Med.* 1990;89:327–334.
17. Monchik JM. Normocalcemic hyperparathyroidism. *Surgery.* 1995;118:917–923.
18. AP Dackiw, JJ Sussman, HA Fritsche, Jr, et al. Relative contributions of technetium Tc 99m sestamibi scintigraphy, intraoperative gamma probe detection, and the rapid parathyroid hormone assay to the surgical management of hyperparathyroidism. *Arch Surg.* 2000;135:550–555. discussion 555–557. [PubMed]
19. Rubello D, Mariani G, Al-Nahhas A, et al. Minimally invasive (99m)Tc-sestamibi radioguided surgery of parathyroid adenomas. *Panminerva Med.* 2005;47:99–107.
20. Lowe H, McMahon DJ, Rubin MR, et al. Normocalcemic primary hyperparathyroidism: further characterization of a new clinical phenotype. *J Clin Endocrinol Metab.* 2007;92:3001–3005. [PubMed]
21. Kettle AG, O'Doherty MJ. Parathyroid imaging: how good is it and how should it be done? *Semin Nucl Med.* 2006;36:206–211. [PubMed]
22. Palestro CJ, Tomas MB, Tronco GG. Radionuclide imaging of the parathyroid glands. *Semin Nucl Med.* 2005;35:266–276.
23. Mollerup CL, Vestergaard P, Frøkjær VG, et al. Risk of renal stone events in primary hyperparathyroidism before and after parathyroid surgery: controlled retrospective follow up study. *BMJ.* 2002;325:807.
24. Deaconson TF, Wilson SD, Lemann J, Jr. The effect of parathyroidectomy on the recurrence of nephrolithiasis. *Surgery.* 1987;102:910–913.