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Severe ureteric colic and a palpable neck node

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DESCRIPTION

A 49-year-old male, hypertensive for 2 years presented with recurrent renal colic of 6 months duration. x-Ray abdomen and intravenous pyelogram showed multiple stones in both kidneys and ureters resembling beads in a necklace (figure 1A and B) with hydronephrotic changes. His renal function tests reflected mild renal insufficiency (serum urea 55.5 mg/dl, serum creatinine 2.5 mg/dl) serum uric acid was 8.9 mg/dl. Serum calcium was 13.0 mg/dl and serum phosphate 2.9 mg/dl. Intact plasma parathyroid hormone (PTH) was ordered which came 375.1 pg/ml against normal of 14–72 pg/ml. There was a small, firm palpable nodule in the left lobe of thyroid measuring 1.5 cm in diameter. USG neck showed a well circumscribed nodule at the left lower pole of thyroid which was neither lobulated nor had any irregular margins. There was no invasion of surrounding structures. Since sestamibi has high sensitivity and specificity for localising parathyroids, a sestamibi scan was done which showed hyperfunctioning left lower parathyroid gland (figure 2).

There was no history suggestive of multiple endocrine neoplasia in the family.

Patient underwent selective parathyroidectomy of left lower parathyroid along with adenoma. Histopathology of the dissected nodular tissue was consistent with benign parathyroid adenoma (figure 3). Signs of parathyroid carcinoma, like fibrous trabecule, nuclear pleomorphism with capsular and vascular invasion, and increased mitotic figures were absent.

Postoperatively, serum calcium dropped to 7.1 mg (which subsequently improved to normal), plasma intact PTH dropped to 3.6 pg/ml. Patient remained normotensive without antihypertensive drugs. Patient underwent subsequent surgery for nephrolithiasis.

Figure 1  Multiple calculi like beads in a necklace on x-ray KUB (A) and IVP (B).

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DISCUSSION

In a patient with kidney stones, an elevated serum Ca level usually means hyperparathyroidism or less frequently sarcoidosis.1 There is a fourfold increase prevalence of renal stones in patients with primary hyperparathyroidism.2 Seventy-five per cent patients undergoing surgical treatment of primary hyperparathyroidism present with nephrolithiasis.3 Diagnosis of hyperparathyroidism must be considered in patients with nephrolithiasis with hypercalcaemia.

In more than 80% patients of primary hyperparathyroidism the cause is diagnosed as benign parathyroid adenoma. Parathyroid carcinomas account for only 1–3% cases. Parathyroid malignancy is one of the least common among endocrine malignancies. Thirty to seventy-six of malignant adenomas are palpable. Palpable adenoma in this case did raise the suspicion of parathyroid malignancy. The clinical pointers to malignant adenoma are serum calcium more than 14–15 mg/dl, serum PTH more than 10 times upper limit of normal, presence of osteitis fibrosa cystica and local invasion. All these were not seen in our case. Histopathology conclusively ruled out the possibility of malignancy.

There could be a theoretical possibility of humoral hypercalcaemia of other malignancies which is usually due to secretion of PTHrP. This was ruled out by absence of symptoms and signs of any malignancy and paraneoplastic symptoms. Also, in such situations plasma intact PTH is undetectable.

Final impression: Benign parathyroid adenoma of left inferior parathyroid gland.

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Competing interests

None.

Patient consent

Obtained.

REFERENCES


Figure 2 Hyerfunctioning left lower lobe nodule on sestamibi scan.
Figure 3  Histopathology of parathyroid showing parenchymal chief cells arranged in sheets cords nests within delicate capillary network.