Case Report

Primary hyperparathyroidism parathyroid adenoma

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Abstract

Hyperparathyroidism is divided into primary, secondary, and tertiary hyperparathyroidism. Most parathyroid hyperplasia is the result of secondary hyperparathyroidism due to renal disease. Tertiary hyperparathyroidism is the autonomous secretion of parathyroid hormone in the setting of long-standing renal disease resulting in hypercalcemia. 80 to 85 percent of primary hyperparathyroidism is caused by parathyroid adenoma followed by primary parathyroid hyperplasia (15%) and parathyroid carcinoma (5%). Parathyroid adenoma is part of a spectrum of parathyroid proliferative disorder that includes parathyroid hyperplasia, parathyroid adenoma, and parathyroid carcinoma. Patients typically present with evidence of primary hyperparathyroidism with elevated serum calcium levels and elevated serum parathyroid hormone levels[1]. A case with features of primary hyperparathyroidism is presented herewith.

Keywords: Parathyroid adenoma, hyperparathyroidism, hypercalcemia.

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CASE REPORT

A 35 year old female presented with complaints of fatigue, dyspepsia and constipation. The patient reported no significant past medical history. Results of physical examination were unremarkable. Subsequent laboratory analysis demonstrated a serum alkaline phosphatase level of 264.0 U/L (normal range 38.0-126.0 U/L) which prompted further investigation. Further investigation revealed hypercalcemia with a serum calcium level of 11.5 mg/dl (normal range 8.4-10.2 mg/dl) and an elevated parathyroid hormone level of 252.6 pg/ml (normal range 7.5-53.5 pg/ml). The serum phosphorus levels were 4.0 mg/dl (normal range 2.5-4.5 mg/dl). T3, T4, TSH were within normal limits.

Investigation	Patient Values	Normal Range
Serum alkaline phosphatise	264 U/L	38.0-126.0 U/L
Serum calcium	11.5 mg/dl	8.4-10.0 mg/dl
PTH	252.6 pg/ml	7.5-53.5 pg/ml
Serum phosphorus	4.0 mg/dl	2.5-4.5 mg/dl

After evaluation of the imaging findings detailed in the next section, the diagnosis of primary hyperparathyroidism resulting from a solitary right parathyroid adenoma was made.

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INVESTIGATIONS

Parathyroid Mibi Scan

Figure 1: 15 mci tc99m mibi injected iv, scan of neck obtained anterior, rao and lao views
Figure 2: 20 min SPECT
Figure 3: 2 hr SPECT Parathyroid MIBI scan suggested a strong possibility of functioning right parathyroid adenoma.

USG

Ultrasonography was done on GE Voluson S6 using 4-12 MHz sector transducer. A well defined oval shaped hypoechoic lesion of size approx. 13 x 7 mm was seen posterior to lower pole of right lobe of thyroid. On Colour Doppler evaluation lesion showed marked vascularity with prominent peripheral vascular arcs and polar feeding vessel. Both lobes of thyroid and isthmus were normal in location, shape, size and echotexture. Internal jugular vein, common carotid artery and isthmus were normal.

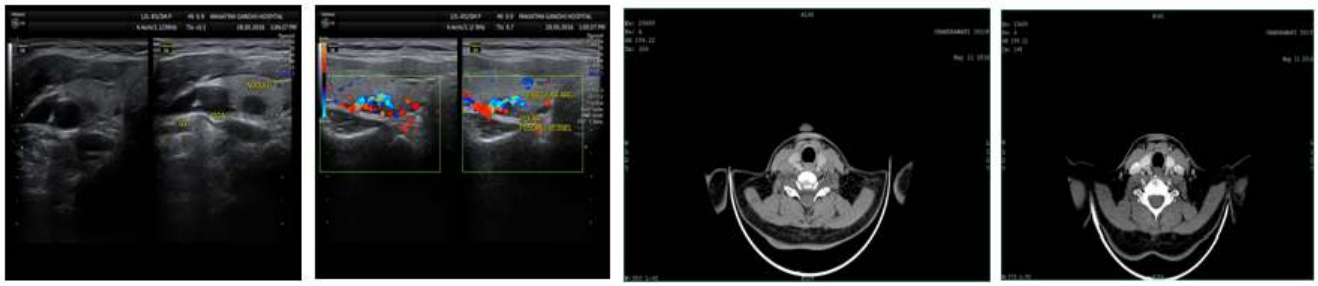

Figure 4: Transverse section at the level of inferior part of right lobe of thyroid showing a hypoechoic nodule posterior to the lobe.
Figure 5: Longitudinal section with colour Doppler showing polar feeding vessel and prominent peripheral vascular arcs.
Figure 6: Precontrast image. A well defined oval hypoattenuating lesion of size approx. 13 x 7 mm is seen posterior to lower pole of right lobe of thyroid.
Figure 7: Postcontrast image. The lesion shows peripheral as well as central enhancement.

PATHOLOGIC EVALUATION

Gross pathologic evaluation of the surgically resected mass posterior to the lower pole of the right lobe of the thyroid gland showed a 1.3 x 0.7 x 0.8 cm fragment of tan-red tissue with a smooth external surface. A portion of the specimen submitted for frozen sectioning revealed hypercellular parathyroid tissue. The microscopic diagnosis was parathyroid adenoma.

DISCUSSION

Autopsy series show two superior and two inferior parathyroid glands in most individuals (Figure 8). Supernumerary glands are seen approximately 3–5% of the time, and fewer than four glands are found in up to 3% of patients. The superior glands are derived from the fourth branchial pouch along with the lateral lobes of the thyroid; the inferior glands arise from the third branchial pouch along with the thymus gland. These embryologic relationships help to explain the normal and variable anatomic locations of the superior and inferior parathyroid glands.

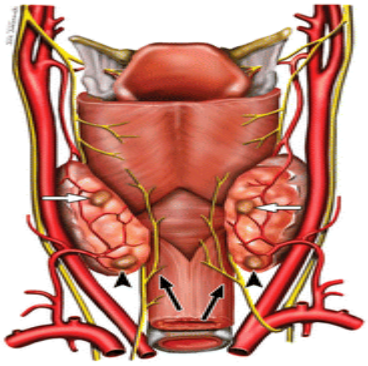

Figure 8:

Diagram shows posterior view of typical locations of paired superior (*white arrows*) and inferior (*arrowheads*) parathyroid glands and their relationship to thyroid gland and surrounding structures. Note close relationship parathyroid glands have with recurrent laryngeal nerves (*black arrows*), illustrating why nerve injury is a significant concern of endocrine surgeons, particularly with four-gland explorations[2]. Parathyroid hormone (PTH) secretion is normally stimulated by a fall in the extracellular calcium concentration. PTH subsequently increases renal calcium reabsorption in the cortical thick ascending limb. It stimulates the hydroxylation of 25-hydroxy-vitamin D at the proximal convoluted tubule in the kidney and increases bone resorption through stimulation of osteoclast activating factors such as interleukin-6 from osteoblasts. Through these actions PTH helps to restore any tendency to hypocalcemia. Current understanding of calcium homeostasis has been advanced by the discovery of the calcium receptor that allows calcium to act with PTH and 1, 25-dihydroxy-vitamin D in maintaining calcium homeostasis. When extracellular calcium binds to the calcium receptor in the parathyroid cell, PTH secretion and parathyroid cell growth are inhibited. At the kidney this interaction between calcium and the calcium receptor inhibits the 1-hydroxylation of 25-hydroxy-vitamin D. Calcium affects the thyroid C cells, stimulating calcitonin release[3]. Hyperparathyroidism is divided into primary, secondary, and tertiary hyperparathyroidism. Most parathyroid hyperplasia is the result of secondary hyperparathyroidism due to renal disease. Tertiary hyperparathyroidism is the autonomous secretion of parathyroid hormone in the setting of long-standing renal disease resulting in hypercalcemia. 80 to 85 percent of primary hyperparathyroidism is caused by parathyroid adenoma followed by primary parathyroid hyperplasia (15%) and parathyroid carcinoma (5%). The patient in this case report is also a case of parathyroid adenoma. Parathyroid adenoma is part of a spectrum of parathyroid proliferative disorder that includes parathyroid hyperplasia, parathyroid adenoma, and parathyroid carcinoma. Patients typically present with evidence of primary hyperparathyroidism with elevated serum calcium levels and elevated serum parathyroid hormone levels[3]. The serum concentration of calcium seems to underestimate the presence of advanced disease in patients in whom the bones are affected[4]. One of the important uses of parathyroid ultrasound is to try to localize parathyroid adenomas in patients with primary hyperparathyroidism to help with surgical planning[5,6]. The examination should be performed with the neck hyperextended and should include longitudinal and transverse images from the carotid arteries to the midline bilaterally and extending from the carotid artery bifurcation superiorly to the thoracic inlet inferiorly. The upper mediastinum may be imaged with an appropriate probe by angling under the sternum from the sternal notch. Although the normal parathyroid glands are usually not visualized with available sonographic technology, enlarged parathyroid glands may be visualized[7]. Primary hyperparathyroidism, whether caused by an adenoma or hyperplasia, can be cured surgically with a high rate of success. When performed by experienced surgeons, traditional surgical therapy—bilateral four-gland exploration—is successful in more than 95% of cases. The development of unilateral and focused surgical approaches over the past decade, however, has made it even more imperative for imaging to accurately locate abnormal parathyroid glands before surgery. With optimized preoperative mapping, the success rate of these less invasive techniques equals that of the traditional bilateral approach[2].

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