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STUDY OF PARATHYROID DISEASE SPECTRUM AND ANALYSIS

Medicine

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ABSTRACT

Proliferative Parathyroid (PT) lesions are rare and frequently missed due to vague presentation. Many cases present with generalized weakness, musculoskeletal symptoms, renal stones, pancreatitis and neuropsychiatric symptoms due to associated hyperparathyroidism. Aim of this study is to evaluate the spectrum of PT lesions.

KEYWORDS

PT hyperplasia, PT adenoma, PT carcinoma, PT cysts, Hyperparathyroidism, hypercalcemia.

INTRODUCTION

PT glands were identified in animals in 1880s. The first parathyroidectomy was performed by Felix Mandel in Vienna in 1925. [1,2] There were many advances in the study of function and management of PT disease afterwards. PT glands are 4 in number and lesions vary from diffuse hyperplasia, adenomas, and cysts to carcinomas. Normal PT gland is about the size of apple seed and approximately weighs around 0.5 grams. Average weight of PT adenoma is around 1 gram. Micradenomas and Giant adenomas weigh less than 0.1 gram and more than 2 grams respectively. Carcinomas can weigh between 2 to 10 grams. [3,4]

The prevalence of primary hyperparathyroidism in adult population is 1-7 per thousand. Hyperparathyroidism is caused in 85% of cases by a single parathyroid adenoma and in 5% of cases by two adenomas. All four glands are hyperactive in diffuse Parathyroid hyperplasia which contributes to the remaining 10 %. Parathyroid carcinoma is a rare cause (<1% cases) of sporadic primary hyperparathyroidism.[5]

PT hyperplasia can be secondary to chronic kidney disease, vitamin and calcium deficiencies. It can also be seen as a part of MEN I (Pituitary adenoma, PT hyperplasia/ adenomas, pancreatic tumors), MEN IIA (PT hyperplasia/ adenomas, medullary thyroid carcinoma, pheochromocytoma) or isolated familial hyperparathyroidism. Etiology of adenomas is unclear in most of the patients and is associated with genetic mutations like CYCLIN D1/ PRAD 1 genes in sporadic cases.[3] Incidence of parathyroid carcinoma and cysts is rare and present as palpable neck swellings. Hyperparathyroidism is more common in carcinomas than cysts.

Aim

Aim of the study is to evaluate various PT lesions, detected and surgically treated in 3 tertiary care hospitals, over a period of 5 years.

Materials and Methods: Data of operated PT lesions from three different tertiary care centers was taken for a period of five years. Apart from history of presenting illness, Parathormone (PTH) and calcium levels were noted in all the patients and compared.

RESULTS

Twelve cases of PT lesions were identified from three tertiary hospitals in a period of five years, of which three were benign hyperplasia, seven were adenomas, one was carcinoma and one was a cyst. PT Hyperplasia was commonly seen in elderly (Mean age- 60 years). Adenoma were commonly seen in middle aged patients (Mean age 47 years). In our study adenomas were the most common, comprising around 58% of parathyroid lesions, while hyperplasias were 25%. Carcinomas and cysts were rare comprising 8.3% each.

Among the above cases, eight were female and four were male patients. Hyperplasia and adenomas showed characteristic female preponderance.[6] Typical hyperparathyroidism symptoms were found in eight cases, comprising of generalized malaise, weakness, muscle pains, renal stones and psychological disturbances. Neck mass was palpable in two cases of which one was a cyst and one was a carcinoma. Elevated parathormone levels and elevated calcium levels were found in nine patients each. Elevated PTH levels were found in 100% of hyperplasia and carcinoma patients, and 71 % of adenoma patients. Hyperparathyroidism and neck masses were indications for surgery.

Table 1: Summary of patient details

S.No	Lesion	% of total cases	% of hyper PTH	AGE	SEX	PTH	Ca+2	Presentation / Assoc Conditions	Management
1	Hyperplasia	25	100	58	F	↑	↑	Renal stones	Removal of 3 1/2 glands
2	Hyperplasia			66	F	↑	N	CKD	Removal of 3 1/2 glands
3	Hyperplasia			56	M	↑	↑	Pancreatitis	Removal of 3 1/2 glands
4	Adenoma	58	71	45	F	↑	↑	PTH Crisis	Excision
5	Adenoma			52	M	↑	↑	Renal stones	Excision
6	Adenoma			32	F	↑	↑	Generalized aches	Excision
7	Adenoma			46	F	↑	↑	Weakness	Excision
8	Adenoma			59	F	N	↑	Goitre	Hemithyroidectomy + PT gland excision
9	Adenoma			39	F	N	N	Thyroid nodule/incidentaloma	Hemithyroidectomy + PT gland excision
10	Adenoma			55	M	↑	↑	Renal stones	Excision of two adenomas
11	Carcinoma	8.3	100	72	M	↑	↑	Neck swelling	Excision
12	Cyst	8.3	0	42	F	N	N	Neck swelling	Excision of cyst + parathyroidectomy

Hemithyroidectomy was planned in two cases for suspected thyroid nodule, but biopsy proved one of them as parathyroid adenoma and the other had an incidentaloma. The incidentaloma and cyst cases had normal PTH and calcium levels in their immediate postoperative period. Exploration and excision of 3 and half glands was done for hyperplastic PT in three cases. Adenomas and cysts were managed with excision, one of the former showing malignant changes.

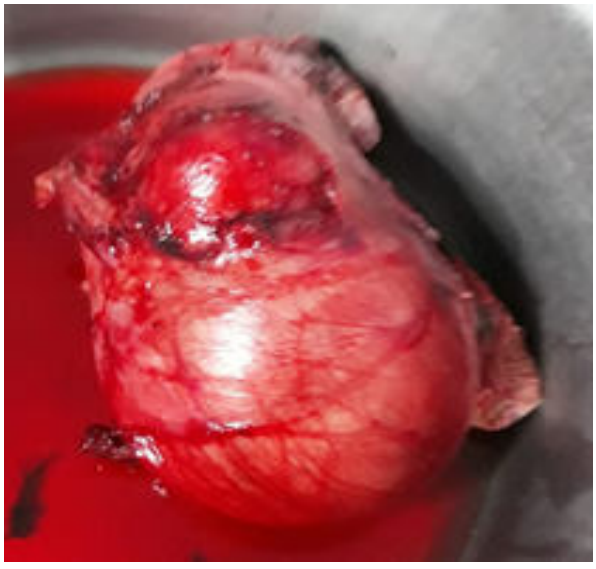

Fig 1: PT adenoma- gross specimen

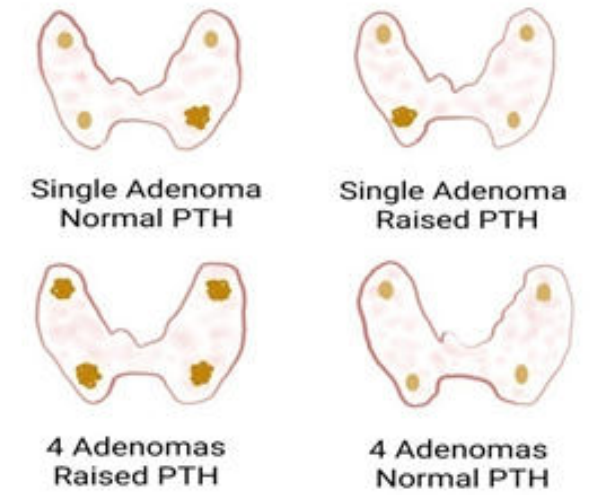

Fig 2: Adenoma- PTH Scenario

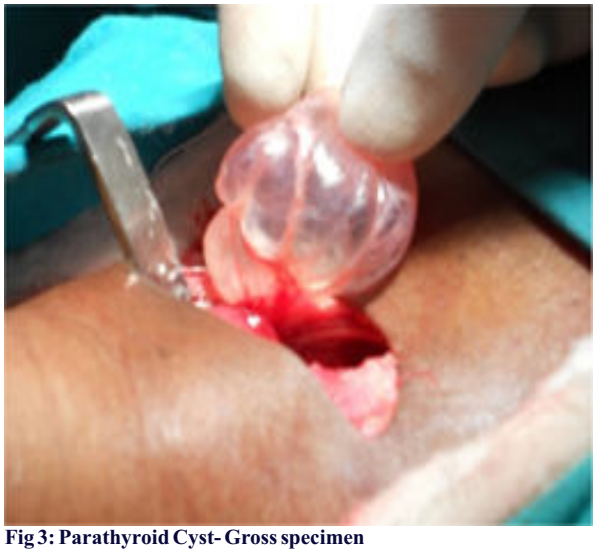

Fig 3: Parathyroid Cyst- Gross specimen

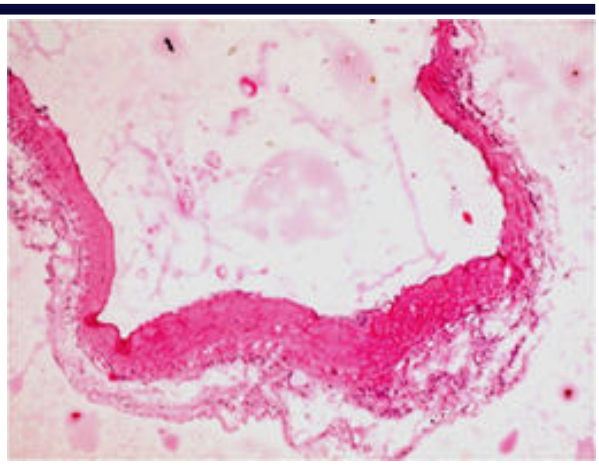

Fig 4: Histopathological picture of PT Cyst

Discussion

PTH acts on bones, intestines and kidneys to play a key role in metabolism of calcium and phosphorus. Low serum calcium stimulates PTH secretion to increase absorption of calcium in distal tubes of nephrons, and to decrease phosphorus resorption. PTH also increases serum calcium by stimulating osteoclasts in the bone, leading to their resorption. It activates vitamin D, thereby increasing absorption of calcium through intestine. Elevated levels of PTH will increase levels of calcium and decrease those of phosphorus. This imbalance is responsible for the symptoms of hyperparathyroidism. [7]

Chronic deficiency of vitamin D and calcium lead to increase in PTH secretion by the PT gland, leading to their hyperplasia. Histopathology of this shows absolute increase in parenchymal cell mass of chief cells and occasionally water cells. Surgical technique involves removal of three and half hyperactive glands and implantation of remnant gland in forearm or neck region.

Adenoma usually develops in one of the PT glands. Rarely it can be seen in more than one gland. It consists of chief cells, oxyphil cells, oncocytes and transitional oncocytes. Rim of the normal PT gland is seen around an adenoma. Management of adenomas consists of excision if single, and if there are four adenomas, removal of three and half glands.[8,9,10] Histopathology of PT carcinoma shows features similar to adenoma, but with mitotic figures, capsular invasion and lobular arrangement of chief cells separated by dense trabeculae. Recurrence of PT adenoma after excision is diagnostic of carcinoma.

PT cysts are rarely functional. These are lined by flattened chief cells, oxyphil and clear cells. Cyst wall has uniform thickness. A high level of PTH in cyst fluid is a characteristic.

Abnormal PT glands are rarely palpable, unless it is a cyst or carcinoma. PT adenomas are associated with hypercalcemia, producing fatigue, bone pains, nephrolithiasis, polyuria and constipation. Very high levels can produce arrhythmias, coma and death. A non-specific rise in urine calcium levels may be seen in some patients. [11,12,13]

Ultrasound, which has 80% sensitivity, does not show small PT glands, unless it is an adenoma or a large PT cyst. MRI and CT scans are also used for imaging. Ideal test for localizing a PT gland is Technetium scan along with SPECT imaging, which is 98% accurate. All symptomatic lesions are managed surgically. Complications include recurrent laryngeal nerve injury and hypocalcemia in the postoperative period.

CONCLUSION

In our study adenomas were the most common, comprising around 58% of parathyroid lesions, while hyperplasias were 25%. Carcinomas and cysts were rare comprising 8.3% each. Elevated PTH levels were found in 100% of hyperplasia and carcinoma patients, and 71 % of adenoma patients. The case of PT cyst was non-functional.

Primary hyper Parathyroidism (PHPT) is seen more frequently than expected i.e., 1-7:1000 adults[5]. Only fraction of the patients who show clear cut symptoms like neck swelling, renal stones, pancreatitis and neuropsychiatric symptoms are generally admitted, investigated and

found to harbor proliferative PT lesions like multiple adenomas (hyperplasia) or adenomas frequently and carcinomas and cysts rarely. Most of the PHPT cases present with vague symptoms, like muscle weakness, fatigue and bone pains and thereby missed, as they take local symptomatic treatment or over the counter medication. We were able to identify only twelve cases because of the above reason. It is suggested to investigate all elderly females who present with general weakness, fatigue or bone pains for PT lesions, so that diagnosis is not missed or delayed.

REFERENCES:

1. Pamathy G, Jayarajah U, Wangmo T, Banagala ASK. Lithium-induced Symptomatic Hypercalcemia and Hyperparathyroidism in a Patient with Bipolar Affective Disorder: A Case Report and Review of Literature. Indian J Psychol Med. 2018 Jul-Aug; 40(4): 378-380
2. Wolfe SA, Fingeret A. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jun 5, 2021. Parathyroid Minimally Invasive Surgery.
3. Gupta M, Singhal L, Kumar A. Hyperparathyroidism Mimicking Metastatic Bone Disease: A Case Report and Review of Literature. J Adolesc Young Adult Oncol. 2018 Jun;7(3):400-403.
4. Allen E, Fingeret A. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jul 26, 2021. Anatomy, Head and Neck, Thyroid.
5. Michael W. Yeh, Philip H. G. Ituarte, Hui Cynthia Zhou, Stacie Nishimoto, In Lu Amy Liu, Avital Harari, Philip I. Haigh, Annette L. Adams; Incidence and Prevalence of Primary Hyperparathyroidism in a Racially Mixed Population , The Journal of Clinical Endocrinology & Metabolism, Volume 98, Issue 3, 1 March 2013, Pages 1122-1129
6. Edafe O, Collins EE, Ubhi CS, Balasubramanian SP. Current predictive models do not accurately differentiate between single and multi gland disease in primary hyperparathyroidism: a retrospective cohort study of two endocrine surgery units. Ann R Coll Surg Engl. 2018 Feb;100(2):140-145.
7. Mizamtsidi M, Nastos C, Mastorakos G, Dina R, Vassiliou I, Gazouli M, Palazzo F. Diagnosis, management, histology and genetics of sporadic primary hyperparathyroidism: old knowledge with new tricks. Endocr Connect. 2018 Feb;7(2):R56-R68
8. Komarowska H, Bromińska B, Luftmann B, Ruchała M. Primary hyperparathyroidism in pregnancy - a review of literature. Ginekol Pol. 2017;88(5):270-275.
9. Leere JS, Karmisholt J, Robaczyk M, Vestergaard P. Contemporary Medical Management of Primary Hyperparathyroidism: A Systematic Review. Front Endocrinol (Lausanne). 2017;8:79.
10. Bilezikian JP, Cusano NE, Khan AA, Liu JM, Marcocci C, Bandeira F. Primary hyperparathyroidism. Nat Rev Dis Primers. 2016 May 19;2:16033.
11. Raruenrom Y, Theerakulpisut D, Wongsurawat N, Somboonporn C. Diagnostic accuracy of planar, SPECT, and SPECT/CT parathyroid scintigraphy protocols in patients with hyperparathyroidism. Nucl Med Rev Cent East Eur. 2018;21(1):20-25.
12. Prabhu M, Damle NA. Fluorocholine PET Imaging of Parathyroid Disease. Indian J Endocrinol Metab. 2018 Jul-Aug;22(4):535-541
13. Parikh AM, Suliburk JW, Morón FE. Imaging Localization and Surgical Approach in the Management of Ectopic Parathyroid Adenomas. Endocr Pract. 2018 Jun;24(6):589-598.