Review Articles

Review of Imaging of the Parathyroid Glance in Health and Disease

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Abstract

Primary hyperparathyroidism is usually caused by a single parathyroid adenoma and it is progressively diagnosed worldwide. The recent advancement of imaging techniques changed the surgical approach of primary hyperparathyroidism patients, from wide traditional bilateral neck exploration to limited neck exploration. Pre-operative imaging is vital in localizing ectopic adenoma to lucid the map before surgical resection. The manuscript at hand is a comprehensive review of the primary hyperparathyroidism covering anatomical, physiological and pathophysiological basics, to the most recent imaging modalities and their respective tasks in patient management.

Keywords: Primary hyperparathyroidism imaging, Nuclear Imaging, Ultrasound, Computed Tomography and M R Imaging.

(J Med J 2007; Vol. 41 (4): 225- 235)

Received	***Accepted***
June 14, 2007	*September 24, 2007*

1- Introduction

Symptomatic or asymptomatic Hyperparathyroidism (HPT) is one of the most frequent endocrine diseases. Adenoma disturbing one or more of the Parathyroid (PT) glands commonly cause profuse secretion of Parathyroid Hormone (PTH).[1] The main cause among patients is single adenoma reaching (85%); followed by PT hyperplasia (10%); multiple adenomas (4%); PT carcinoma (<1%) and PT cyst.[2] The incidence of Primary Hyperparathyroidism (PHPT) in United States has risen from 0.08 per 1000 to 0.5 per 1000; [1] on the other hand, it has reached the value of 2 per 1000 in women and 0.5 per 1000 in men over 40 years. [2]

The estimated prevalence within Europeans is 3 per 1000; reaching 21 per 1000 among women between 55-75 years.[3] The prevalence in Asians appears lower. [4] Furthermore, HPT caused by hyperplasia might be part of familial syndromes, such as the multiple endocrine neoplasia syndromes and familial isolated HPT. [1]

Anatomy and Embryology

The PT glands are usually four small ovoid structures each measure about 6x4x2mm. Although each one is enclosed within its own thin collagenous capsule, all are positioned between the thyroids fibrous capsule and the pretracheal fascia.

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Most adult humans have two pairs, and about 2-5% of them have supranumerary that vary from 2 to 6 glands but less than 4 is very rare. Their position is quite variable; the superior glands are more constant than the inferior ones and they are typically situated at the level of inferior border of cricoid cartilage. They migrate in close association with the thyroid and thymus glands.

Location of the superior PT is 70% posterior to the middle third of the thyroid lobe, 22% posterior to upper third, less than 2% of them are ectopic. Variability is the rule regarding location of the inferior PT glands, possibly consequenced to their complex migration path and its relation to thymus development. The inferior PT glands are commonly (50%) found close to the lower pole of the thyroid lobe. Nevertheless, PT glands could be found anywhere from common carotid bifurcation up to the mediastinum down. 80-85% of PT adenomas are eutopic and 15-20% are ectopic. Ectopic abnormal glands might be within, outside the thymus, or alongside the esophagus in the superior mediastinum, uncommonly within or lateral to carotid sheath, but hardly ever in the inferior mediastinum at the aorto pulmonary window. Extranumerary of PT glands is a misguiding condition where the patient has 4 normal glands but with abnormal fifth or sixth gland concealed within the mediastinum. [5]

Physiology and Pathophysiology

Bone and teeth act as a balancing reservoir for body fluids, by holding about 95% of all body calcium such as calcium phosphate or hydroxyapatite crystals. Parathyroid hormone controls the level of ionized calcium in blood and in the extracellular fluid. The liver (70%) and kidneys (20%) quickly metabolize secreted PTH then it vanishes from circulation within 2-3 minutes half-life. Less than 1% of the secreted hormone lastly reaches PTH receptors of the targeted cells mainly in bone and kidneys, which initiate reactions to raise blood calcium level.[1]

Primary hyperparathyroidism is a condition when the PTH is inappropriately secreted irrespective to the extracellular calcium level. Parathyroid cells show intense multiplication action causing gland enlargement along with lowering their response to the inhibiting effect of increased calcium level on PTH secretion. The intense secretion of PTH can cause osteopenia due to severe persistent resorption of calcium in bone, while in severe conditions, it may result in osteitis fibrosa cystica and renal stones due to the excessive chronic flow of calcium in urine. [1]

2- Imaging Modalities in PHPT

Parathyroid Scintigraphy

No parathyroid specific radiopharmaceutical has been developed; moreover, scintigraphic localization of parathyroid tissue is further complicated by the intrinsic close proximity of parathyroid glands to the metabolically active thyroid gland. Using 2 tracers with different uptake patterns in thyroid and parathyroid glands was adapted to circumvent this problem. [6] ^{75}Se-methionine/^{131}I subtraction scanning was the technique first used in early studies. The imaging technique was later improved by Ferlin et al., replacing ^{75}Se-methionine with ^{201}Tl-chloride, a myocardial perfusion agent, and ^{131}I with $^{99m}TcO_4^-$ in early 1980's. [7] Both ^{75}Se-methionine and Thallium accumulate in both the parathyroid and the thyroid tissues and consequently subtraction of thyroid activity is required to identify parathyroid adenoma.

This test however failed to demonstrate a definite clinical advantage over morphological imaging modalities, because of the intrinsic variability and low reproducibility in interpreting results among different centers.[8]

^{99m}Tc-Sestamibi

Using another myocardial perfusion agent, Coakley et al. incidentally observed ^{99m}Tc-sestamibi (hexakis 2-methoxyisobutyl isonitrile) uptake and retention in abnormal parathyroid glands of patients with PHPT. [9] This was subsequently confirmed by numerous reports. [10]

Sestamibi is a lipophilic, monovalent cationic isonitrile compound that passively diffuses across the cell membrane and is primarily sequestered in the mitochondria. [11] Because of the superior image quality, its more favorable dosimetry, and improved accuracy, ^{99m}Tc-sestamibi rapidly replaced ^{201}Tl as the radiopharmaceutical of choice for parathyroid imaging. [12]

The retention of this tracer in parathyroid lesions is presumably related to the presence of oxyphil cells in these lesions. [13] Oxyphil cells are rich in mitochondria, which are the site of intracellular sestamibi sequestration. ^{99m}Tc-sestamibi accumulates both in the thyroid and in the parathyroid tissues within minutes after intravenous administration. However, sestamibi washes out from thyroid tissue at a much faster rate than from the parathyroid tissue and this makes it especially useful for parathyroid imaging. This differential retention or washout might be related to downregulation of the P-glycoprotein outflux carrier in the parathyroid tissue. [14]

^{99m}Tc-sestamibi can accurately localize parathyroid adenomas in 85%–95% of patients with primary hyperparathyroidism. Different ^{99m}Tc-sestamibi imaging protocols to localize parathyroid adenoma have been adopted based on the institutional logistics and experience. [15]

Single-Tracer Dual Phase Scintigraphy

Based solely on the differential washout rate of ^{99m}Tc-sestamibi from the thyroid and the parathyroid tissues, planar imaging of the neck and thorax is recorded in early phase (15 min) and then in delayed phase (2–3 h) after the intravenous injection of 20 mCi ^{99m}Tc-sestamibi. A focal area of increased tracer accumulation in early phase images that persists in the delayed phase is considered positive for parathyroid adenoma. Although this technique is easy to perform, both sensitivity and specificity are limited in patients with nodular goiter. [16] Solid thyroid nodules can concentrate ^{99m}Tc-sestamibi quite avidly, regardless of whether they are benign or malignant and whether they appear as "hot" or "cold" on the $^{99m}TcO_4^-$ scan; and this can result in false positive results. [17] False-negative results are also reported in cases with early focal uptake that washes out in the delayed images as encountered in some parathyroid adenomas. [18]

Dual-Tracer Subtraction Scintigraphy

To overcome the limitation of single tracer dual phase technique, dual-phase ^{99m}Tc-sestamibi imaging with administration of a second thyroid specific radiopharmaceutical was introduced. Images are then subtracted to allow the detection of focal uptake specific for abnormal parathyroid tissue. Various protocols have been described based on the type of thyroid-imaging agent used, whether ^{123}I or $^{99m}TcO4^-$, and the sequence of the administration of tracers.

^{99m}Tc-pertechnetate ($^{99m}TcO_4^-$) is ubiquitously available in all nuclear medicine departments at low cost. After completion of ^{99m}Tc-pertechnetate thyroid imaging, ^{99m}Tc-sestamibi injection and early sestamibi images are obtained without changing the patient's position. This technique has been reported to have 89% sensitivity and 98% specificity. [19] Geatti et al. further modified the technique by reducing the ^{99m}Tc-pertechnetate dose and increasing the ^{99m}Tc-sestamibi dose to overcome the occasional superposition of thyroid activity over a parathyroid adenoma located posterior to thyroid tissue. Using this protocol, he reported a sensitivity of 95% without any false-positive results. [20]

^{99m}Tc-pertechnetate can also be administered after the completion of the late (2–3 h) ^{99m}Tc-sestamibi images when most of the ^{99m}Tc-sestamibi has already washed out from the thyroid. The late ^{99m}Tc-sestamibi image is then subtracted from the last images obtained after ^{99m}Tc-pertechnetate to obtain a "pure" ^{99m}Tc-pertechnetate thyroid image. [21]

Technical Aspects in Parathyroid Scintigraphy

Unfortunately, parathyroid lesions, even when markedly enlarged, are relatively small structures,

and may go unrecognized if parallel hole collimator is used alone. The use of a pinhole collimator in the neck, with a trade-off in image acquisition time, increases imaging resolution and also magnifies structures being imaged. To avoid patient motion artifacts, a dynamic image sequence can be obtained; this will allow for motion correction and selection of motion free frames in image generation. [22] To detect ectopic parathyroid adenomas however, additional imaging of the chest using parallel hole collimator should be performed routinely.

Single Photon Emission Computed Tomography (SPECT) mode adds information on the depth of the lesion and its topographic correlation with other anatomic structures. This information is useful not only for localizing a parathyroid lesion but also for differentiating a thyroid lesion from a parathyroid lesion.

SPECT is most helpful in the evaluation of the mediastinum for possible ectopic parathyroid gland.[23] Although the addition of SPECT only marginally improves sensitivity from 86% to 90.5%, [22] most authors now favor a wider application of this imaging modality, especially in ectopic lesion.

False-Positive Results

The most frequent cause of false-positive results in sestamibi parathyroid imaging is the solid thyroid nodule, either solitary or in a multinodular gland. False positive results have also been reported in enlarged thymic tracts with cystic morphology, thyroid carcinoma, lymphoma and other causes of lymphadenopathy, including metastatic disease, inflammation and even sarcoidosis. Sestamibi uptake in the brown tumors of hyperparathyroidism has also been described. [24]

False-Negative Results

Several factors may be involved in the non-scintigraphic visualization of parathyroid adenoma. Sestamibi is less sensitive for detecting hyperplastic parathyroid glands than for detecting adenomatous ones, in part this might be due to small lesion size. Parathyroid lesions which express P glycoprotein or multidrug resistance protein fail to accumulate sestamibi or demonstrate rapid washout of this tracer. [25]

As mentioned previously, the presence of mitochondria-rich oxyphil cells presumably accounts for sesatmibi uptake in parathyroid lesions, and glands with fewer oxyphil cells, and hence fewer mitochondria may account for both lower uptake and test sensitivity. [26]

There are data that suggest that sestamibi is less sensitive for detecting Multi-Gland Disease (MGD). [27] Exactly why the test is less sensitive for MGD than for the solitary lesion is uncertain, but it might be due to smaller adenomas found in patients with MGD. [28] This fact does indicate that preoperative sesatmibi imaging cannot be the sole basis on which the decision to perform surgery or not is taken. [29]

Ultrasonography

The importance of Ultrasonography (US) in the preoperative evaluation of patients with PHPT has increased with the adoption of the minimally invasive surgical techniques at most medical centers. Typical US appearances of parathyroid adenoma vary from round or oval to elongated homogenous hypoechoic or anechoic nodule relative to thyroid tissue without sound transmission placed close to the thyroid or lower in paratracheal and paraesophageal space. [30, 31] Its capsule separates it from thyroid gland by showing a distinct echogenic line. Hyperechoic component, cystic change and calcification are recognized as morphological variations, especially in large adenomas.[30, 32] Ninety % of PT adenomas demonstrate an intra-parenchymal hypervascular pattern on colour flow imaging, [30] with enlarged feeding artery and peripheral arc of vascularity. [33]

The diagnosis of PT hyperplasia relies on the identification of more than one enlarged PT gland. The hyperplastic PT glands tend to be more spherical than adenomas and may show

presence of calcification. The rare PT carcinoma may have similar appearance to adenoma; however, it is vital to assess any invasion of nearby structures and immobility of the abnormal gland at swallowing. Metastases to adjacent cervical lymph nodes are seen in 21-28% of PT carcinoma.

The accuracy of US in detecting PT abnormalities, in patients who underwent surgery and in those who did not, ranges from 34% to 83%. While in another study, the sensitivity and positive predictive value of ultrasound were 86.9% and 89.1%, respectively, with no significant difference in patients without (88.2% and 90%), and in patients with (80% and 84.2%) prior thyroid or PT surgery. The sensitivity diminishes in patients with SHPT and recurrent or persistent HPT because of the higher frequency of hyperplastic glands (as opposed to adenomas) in the former and the higher prevalence of ectopic adenomas in the latter. [34]

Ultrasound correctly predicted surgical findings are seen in 74% of patients undergoing surgery, including 84% of patients with solitary eutopic adenomas, only 25% of patients with solitary ectopic adenomas, and only 16% of patients with multigland PT disease. The capacity of US and sestamibi scintigraphy in predicting surgical findings correctly is estimated to be (74% vs. 82%, respectively) and positive predictive value is (93% vs. 90%, respectively) with positive results up to 98% when both tests were employed. [35]

CT Scan

Abnormal PT glands are intensely enhanced post-contrast administration (Figure 2). The identification of an enhancing nodule in locations characterized by hyperfunctioning PT glands is required for the CT to diagnose abnormal PT tissue. [31] Sensitivity of CT in detecting abnormal PT tissue ranges from 46 to 87%. In one study, the CT showed its capability in localizing PT disease in (86%) of patients and offering valuable preoperative localizing information. [36]

Four-dimensional CT (4D CT) provides both functional and highly detailed anatomic information about PT tumors. 4D-CT prove to have better sensitivity than sestamibi imaging and ultrasonography in its precise (quadrant) localization of hyperfunctioning parathyroid glands. [37] 4D-CT demonstrated improved sensitivity (88%) over sestamibi imaging (65%) and ultrasonography (57%).

MRI

The most common picture of hyperfunctioing PT glands on MRI is isointense-to-low signal intensity on T1-weighted images and high signal intensity on T2-weighted images with intense-contrast enhancement (Figure 3). Occasionally, high signal intensity may be encountered on T1 and T2-weighted images, reflecting gland hemorrhage or cystic components. However, if low signal intensity is rarely seen with both sequences, this indicates sclerosis or fibrosis, old hemorrhage, and cellular degeneration.[32,38] Signal intensity increase on T1-weighted images, and isointense to the muscle on T2-weighted images was seen in PT carcinoma. [32] The signal pattern of the abnormal PT tissue has been shown to correlate with the histologic characteristics of the abnormal glands. MR imaging accurately detects ectopic hyperfunctioning PT tissue.[31]

The sensitivity of MRI in detecting hyperfunctioning PT tissue in operated patients and in those not operated ranged from 50-88% while reported detection sensitivity in PT adenoma ranged from 65-80%.[30]

Sensitivity of MR and sestamibi imaging was higher for adenomas (94% and 89%, respectively) than for hyperplastic glands (74% and 68%, respectively). The specificity of sestamibi imaging (94%) exceeded that of MR imaging (75%) but all ectopic glands were localized correctly by both techniques. When MR imaging and sestamibi were interpreted together, their sensitivities for adenomas and for hyperplastic glands were 94% and 84%, respectively, while their overall sensitivity was 89% and overall specificity was 95%.[38]

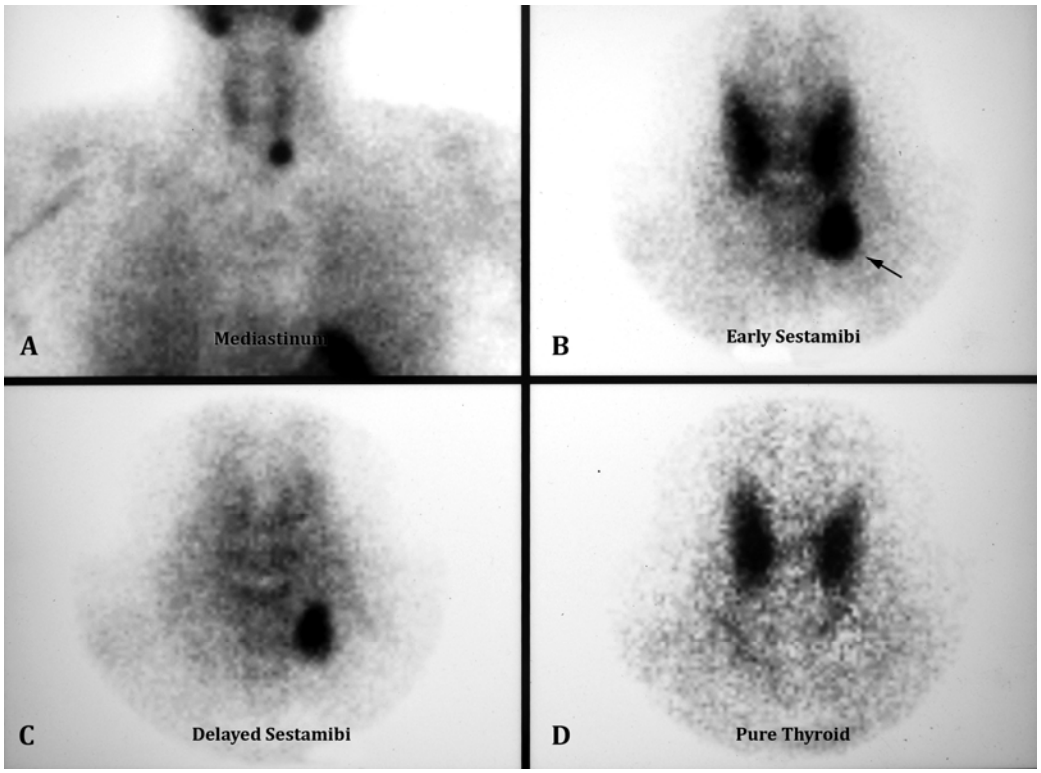

Figure (1): Dual-phase parathyroid scintigraphy of mediastinum (panel A) and neck (panels B to D). A focal area of abnormal activity is seen just inferior to left thyroid lobe in early 99mTc-sestamibi images (panel B). It persists in the delayed images (panel C) and is not present in pure 99mTCO4- thyroid image (panel D).

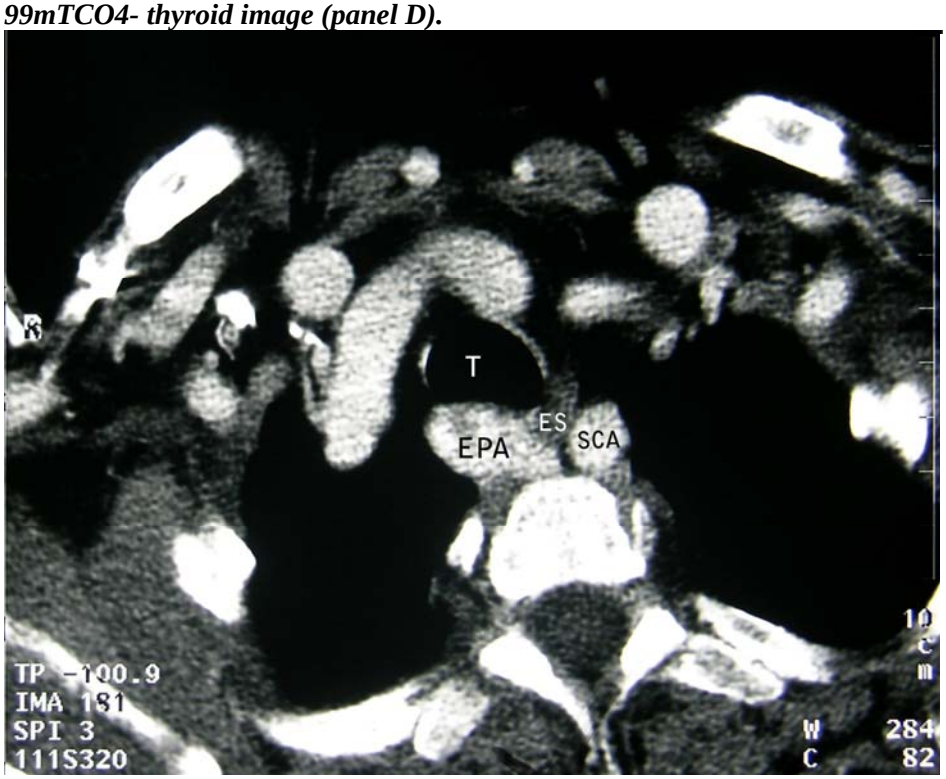

Figure (2): CT scan of the superior mediastinum with intravenous contrast showing the ectopic parathyroid adenoma (EPA) posterior to the trachea(T) and to the right of the esophagus and left subclavian artery(SCA).

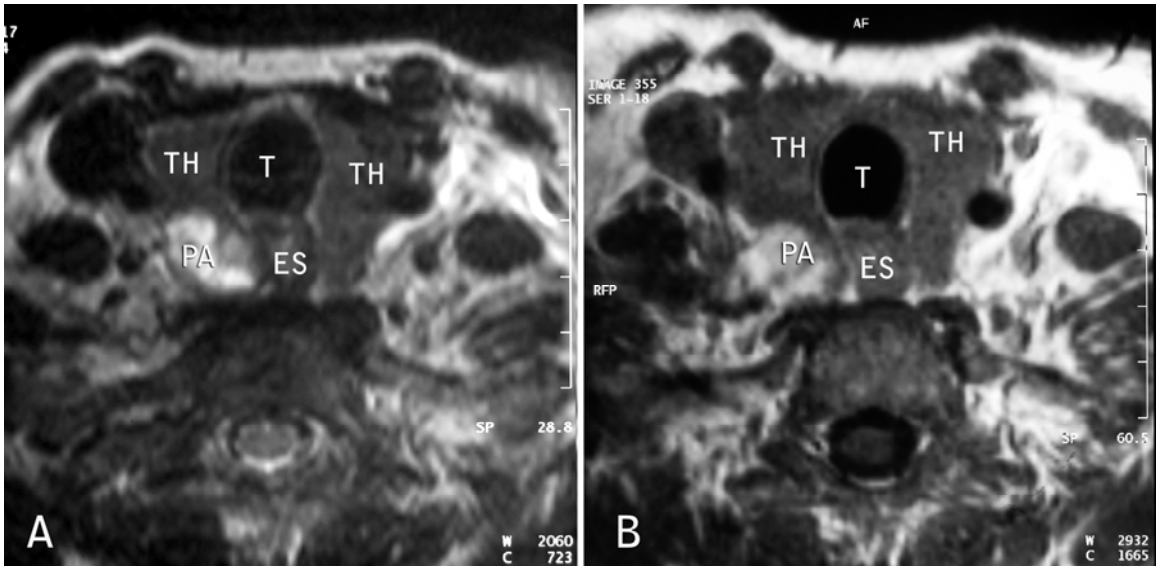

Figure (3): Axial MRI T2 weighted image (A) showing an adenoma (hyperintense), on the right of the inferior parathyroid gland while axial T1 weighted image with contrast (B) showing enhancement of the adenoma.

X-Ray

A- Osteopenia: is the most common radiological finding, which may be generalized and or asymmetric; initially the fine trabeculations are lost resulting in coarse and thick trabeculation, and may progress to ground glass appearance. The Radiographs show change when about 30-50% of bone density is lost, whereas Dual-energy X-ray Absorptiometry (DXA) and Quantitative Computed Tomography (QCT) are more sensitive in the evaluation of osteopenia.

B- Bone resorption: occurs at many different anatomical sites and may be classified as:

1. **Subperiosteal:** unusual lace-like appearance may be seen beneath the periosteum with speculated external cortex and resorption may progress to complete cortical disappearance. The most common sites are the radial aspect of middle phalanges of index and middle fingers, and medial aspects of metaphyses of long bones (tibia, humerus, and femur).
2. **Intracortical:** appears as linear striation in the cortex, produced by resorption of bone in the Haversian Canals and best seen on the cortical surface of the second metacarpal. While in the skull, resulting in salt and pepper appearance, indicates rapid bone turnover, and usually accompanies advanced disease.
3. **Endosteal:** widening of medullary cavity and thinning of the inner cortex appears as scalloped lucencies on the inner aspect of the bony cortex. Typically seen in hands and associated with subperiosteal or cortical resorption.
4. **Subchondral:** The resorbed bone will collapse followed by new bone formation and fibrous replacement. On radiographs, it appears as subchondral lucency, surrounded by sclerosis that may produce irregular articular margin with the appearance of widened joint space. Most commonly, it is seen in joints of the axial skeleton; namely, sacroiliac, acromioclavicular, temporomandibular, discovertebral, sternoclavicular, patella and symphysis pubis, but may affect joints of appendicular skeleton.
5. **Subligamentous and subtendinous:** It occurs at the insertion sites on bone; most often at femoral trochanters, dorsal aspect of patella, ischial tuberosities and calcaneal

insertions of plantar aponeurosis. Moreover, it occurs at Achilles tendon, inferior margin of distal end of clavicle and humeral tuberosities.

6. **Terminal tuft resorption (acroosteolysis)**: also the digits may appear spatulous or clubbed.

C- Sclerosis: generalized or Rugger-Jersey spine which is more common in SHPT.

D- Brown tumors (osteoclastomas): A well-circumscribed expansile eccentric lytic lesion of bone that represents osteoclastic resorption with subsequent fibrous replacement. Usually occurs in cortical bone but the common sites include the mandible, clavicle, ribs, pelvis, and femur. Lesions may become sclerotic after resection of the adenoma. Previously, it was considered a characteristic of PHPT, but now it is more commonly seen in SHPT because of the increasing population and life expectancy of dialysis patients.

E- Chondrocalcinosis: The common site is the symphysis pubis and may affect menisci of the knee, triangular cartilage of the wrist. Chondrocalcinosis is a nonspecific finding that has a higher prevalence in PHPT rather than in SHPT.

F- Soft tissue and vascular calcification: more common in SHPT, especially kidneys and pancreas. [39]

3- Interventional Procedures

A-Selective venous sampling (SVS): Bilateral internal jugular veins sampling is used to localize ectopic PT adenomas. This technique must be reserved for centers with specialists and for selected patients. SVS demonstrated an overall 83.3% correct localizing sensitivity of PT adenoma and (91.6%) of hyperplastic PT glands. False positive or indeterminate results of SVS were found in 6% and 2% of patients, respectively. Compared with non-invasive localization studies, SVS for PTH yielded the best results for recurrent or persistent HPT and for patients with previous neck explorations. [40]

B-Percutaneous needle biopsy: may be performed if malignancy is suspected and sometimes fine needle aspiration could be sufficient and both are done usually under ultrasound guidance.

4- Conclusion

This article discusses the commonly used techniques for imaging the PT glands and their role in the preoperative evaluation of patients with PHPT.

Imaging studies are not used to make the diagnosis of primary hyperparathyroidism or clear the road map for surgical therapy since both should be based on clinical data. Imaging is used to guide the surgeon once surgical therapy has been decided. Radiographs have limited diagnostic value, especially in the early stages of the disease, and radiograph screening is not recommended, [41] while bone mineral density test is a useful tool to document bone loss. [42]

Ultrasonography of the neck is a sensitive test and may be equivalent or superior to sestamibi scanning when performed by experienced operators. It is readily available, without radiation exposure and therefore should be the first investigation to patients with PHPT, and if malignancy is suspected, CT or ultrasound guided percutaneous needle biopsy may be performed. The detection of thyroid nodules on ultrasound helps to better interpret the scintigraphic images as well as to increase the ability to predict surgical findings when both tests combined reaches 98%. Whereas, Single-photon emission computed tomography provides tomographic data and is recommended in patients with ectopic adenomas. Also, MRI, 4D CT and fused SPECT CT scan can be useful, particularly in cases of recurrent or persistent disease and in ectopic locations to better guide the surgeon in preoperative planning.

References

1. Clark OH, Sliperstein AE. The hypercalcemic syndrome. In: Friesen SR, Thompson NW, (eds.) Surgical Endocrinology Clinical Syndromes. Philadelphia, PA: Lippincott; 1990: 311-339.
2. Melton LJ 3RD. The epidemiology of primary hyperparathyroidism in North America. J Bone Miner Res. 2002; 17(suppl 2):n12-n17.
3. Adami S, Marcocci C, Gatti D. Epidemiology of primary hyperparathyroidism in Europe. J Bone Miner Res. 2002; 17(suppl 2):n18-n23.
4. Belezikian JP, Meng X, Shi Y et al. Primary hyperparathyroidism in women: a tale of two cities- New York and Beijing. Int J Fertil Women's Med. 2000; 45:158-165.
5. Marcheix B, Brouchet L, Berjaud J et al. Eur J Cardiothorac Surgery. 2006; 30(5): 808- 810.
6. Bondeson AG, Bondeson L, Ljungberg O et al. Fat staining in parathyroid disease-diagnostic value and impact on surgical strategy: clinicopathologic analysis of 191 cases. Hum Pathol 1985; 16: 1255- 1263.
7. Palestro CJ, Tomas MB, Tronco GG. Radionuclide imaging of the parathyroid glands. Semin Nucl Med. 2005; (4):266-276.
8. Samanta A, Wilson B, Iqbal J, Burden AC, Walls J, Cosgriff P. A clinical audit of thallium-technetium subtraction parathyroid scans. Postgrad Med J. 1990; 66:441- 445.
9. Coakley AJ, Kettle AG, Wells CP, O'Doherty MJ, Collins REC. ^{99m}Tc-Sestamibi: a new agent for parathyroid imaging. Nucl Med Commun. 1989; 10: 791- 794.
10. Casas AT, Burke GJ, Mansberger AR, Wei JP. Impact of technetium-99m sestamibi localization on operative time and success of operations for primary hyperparathyroidism. Am Surg. 1994; 60: 12- 17.
11. Hetrakul N, Civelek AC and Udelsman R. CA. In-vitro accumulation of technetium -99m-sestamibi in human parathyroid mitochondria. Surgery 2001; 130: 1011- 1018.
12. Giordano A, Rubello D, Casara D. New trends in parathyroid scintigraphy. Eur J Nucl Med. 2001; 28: 1409- 1420.
13. Melloul M, Paz A and Koren R, Cytron S, Feinmesser R, Gal R. 99m Tc-MIBI scintigraphy of parathyroid adenomas and its relation to tumour size and oxyphil cell abundance, Eur J Nucl Med 2001; 28: 209- 213.
14. Yamaguchi S, Yachiku S, Hashimoto H et al. Relation between technetium 99m methoxyisobutylisonitrile accumulation and multidrug resistance protein in the parathyroid glands. World J Surg. 2002; 26: 29- 34.
15. Chen H, Sokoll LJ, Udelsman R. Outpatient minimally invasive parathyroidectomy: a combination of sestamibi-SPECT localization, cervical block anesthesia, and intraoperative parathyroid hormone assay. Surgery. 1999; 126: 1016- 1021.
16. Taillefer R. 99mTc sestamibi parathyroid scintigraphy. In: Freeman L, (ed.) Nuclear Medicine Annual 1995. New York, NY: Raven Press; 1995: 51- 79.
17. Versaci A, Pecorella GR, Macri A et al. Retrospective analysis of the association of nodular goiter with primary and secondary hyperparathyroidism. Eur J Endocrinol. 2001; 145: 429- 434.
18. Bénard F, Lefebvre B, Beuvon F, Langlois MF, Bisson G. Rapid wash-out of technetium-99m-MIBI from a large parathyroid adenoma. J Nucl Med. 1995; 36: 241- 243.
19. Casara D, Rubello D, Saladini G, Piotto A, Toniato A, Pelizzo MR. Imaging procedures in the evaluation of hyperparathyroidism: the role of scintigraphy with 99mTc-MIBI [in Italian] In: Rovelli E, Samori G, eds. Primary and Secondary Hyperparathyroidism [in Italian]. Milan, Italy: Wichtig Editore; 1992: 133- 136.
20. Geatti O, Proto G, Mazzolini A, Shapiro B, Orsolon PG, Guerra UP. Localization of parathyroid enlargement: experience with technetium 99m methoxyisobutylisonitrile and thallium-201 scintigraphy, ultrasound and computed tomography. Eur J Nucl Med. 1994; 21:17- 23.
21. Giuliano Mariani, MD; Seza A. Gulec, MD; Domenico Rubello, MD et al. Preoperative Localization and Radioguided Parathyroid Surgery. J Nucl Med 2003; 44: 1443- 1458.
22. Rubello D, Saladini G, Casara D et al. Parathyroid imaging with pertechnetate plus perchlorate/MIBI subtraction scintigraphy: a fast and effective technique. Clin Nucl Med. 2000; 25(7): 527- 531.

23. Billotey C, Sarfati E, Aurengo A, et al. Advantages of SPECT in technetium-99m-sestamibi parathyroid scintigraphy. J Nucl Med. 1996; 37: 1773- 1778.
24. Moka D, Eschner W, Voth E, Dietlein M, Larena-Avellaneda A, Schicha H. Iterative reconstruction: an improvement of technetium-99m-MIBI SPECT for the detection of parathyroid adenoma? Eur J Nucl Med. 2000; 27: 485- 489.
25. Garvie NW. Imaging the parathyroids. In: A.M. Peters, (Editor), Nuclear Medicine in Radiologic Diagnosis, Martin Dunitz: London 2003; 681-694.
26. Sharma J, Mazzaglia P, Milas M et al. Radionuclide imaging for hyperparathyroidism (HPT): which is the best technetium-99m sestamibi modality? Surgery. 2006; 140(6): 856-863.
27. Chung-Yau Lo, Brian H. Lang, W.F. Chan, Annie W.C. Kung and Karen S.L. Lam. A prospective evaluation of preoperative localization by technetium-99m sestamibi scintigraphy and ultrasonography in primary hyperparathyroidism. Am J Surg 2007; 193 (2): 155- 159.
28. Gavin T. Slitt, Hugh Lavery, Anthony Morgan, Bruce Bernstein, James Slavin, Mozaferiddin K. Karimeddini and Robert A. Kozol Hyperparathyroidism but a negative sestamibi scan: a clinical dilemma. Am J surg. 2005; 190 (5):708-712.
29. Gallowitsch HJ, Mikosch P, Kresnik E, Unterweger O, Lind P. Comparison between 99mTc-tetrofosmin/pertechnetate subtraction scintigraphy and 99mTc-tetrofosmin SPECT for preoperative localization of parathyroid adenoma in an endemic goiter area. Invest Radiol. 2000; 35: 453- 459.
30. Ahuja AT, Wong KT, Ching ASC et al. Imaging for primary hyperparathyroidism– what beginners should know. Clinical Radiology 2004; 59: 967-976.
31. Michael B. Gotway, Jessica WT. Leung, Gretchen A et al. Hyperfunctioning Parathyroid Tissue: Spectrum of Appearances on Noninvasive Imaging. AJR. 2002; 179.
32. Michael B. Gotway, MD; Gautham P. Reddy, MD; W. Richard Webb, MD et al. Comparison between MR Imaging and 99mTc MIBI Scintigraphy in the evaluation of Recurrent or Persistent Hyperparathyroidism. Radiology 2001; 218: 783- 790.
33. Kamaya A, QUON A, Jeffrey RB. Sonography of abnormal parathyroid gland. Ultrasound Q. 2006; 22(4): 253- 262.
34. Ghaheri BA, Koslin DB, Wood AH et al. Preoperative ultrasound is worthwhile for reoperative parathyroid surgery. Laryngoscope. 2004; 114(12): 2168- 2171.
35. Haber RS, Kim CK, Inabnet WB. Ultrasonography for preoperative localization of enlarged parathyroid glands in primary hyperparathyroidism: comparison with (99m) technetium sestamibi scintigraphy. Clin Endocrinol (Oxf). 2002; 57(2): 241- 249.
36. Gross ND, Weissman JL, Veenker E et al. The diagnostic utility of computed tomography for preoperative localization in surgery for hyperparathyroidism. Laryngoscope. 2004; 114(2) 227- 231.
37. Rodgers SE, Hunter GJ, Hamberg LM et al. Improved preoperative planning for directed parathyroidectomy with 4-dimensional computed tomography. Surgery 2006; 140(6): 932- 940.
38. Lee VS, Spritzer CE, Coleman RE et al. The complementary roles of fast spin-echo MR imaging anddouble-phase 99m Tc-sestamibi scintigraphy for localization of hyperfunctioning parathyroid glands. AJR Am J Roentgenol. 1996; 167(6): 1555- 1562.
39. E-medicine, Bonnie Freitas, Hyper-parathyroidism, Primary 2002.
40. Reidel MA, Schilling T, Graf S et al. Localization of hyperfunctioning parathyroid glands by selective venous sampling in reoperation for primary or secondary hyperparathyroidism. Surgery 2006; 140(6): 907- 913.
41. E- medicine, Philip N Salen, Hyper-parathyroidism, 2006.
42. E- medicine, Laurence Kim, Hyper-parathyroidism, 2005.

مراجعة التشخيص الشعاعي للغدة الدريقية في حالاتها الطبيعية والمرضية

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الملخص

أثبتت الدراسات العالمية أن الغدوم الدريقي المفرد هو المسبب المعتاد لفرط الدريقية الأولي، ويعتبر فرط الدريقية الأولي المشخص سريرياً وما قبل، واحداً من امراض الغدد الصماء الشائعة، ويسببه الإفراز الزائد لأنزيم الدريقية نتيجة ورم حميد في إحدى الغدد الدريقية أو تضخم جميع الغدد الدريقية في معظم الحالات، وإن المستوى المتوقع لفرط الدريقية يكون أكثر في النساء وخاصة بعد عمر الأربعين وخاصة في الولايات المتحدة وأوروبا. وبمراجعة العلوم الأساسية يتضح لدينا أن عدد الغدد الدريقية يكون في العادة أربعاً، وتكون خلف الدرقية (80-85%) بينما في بعض الأحيان تكون متعددة ومهاجرة وتقع ما بين الرقبة والمنصف، وتكون الغدد الدريقية السفلى أكثر عرضة لذلك لتعقد مسارها الجنيني.

يتحكم أنزيم الدريقية بمستوى الكالسيوم الأيوني في الدم والسائل خارج الخلايا، بينما يتم استقلابه في الكبد(70%) والكلى(20%)، ويكون عمر النصف البيولوجي 2-3 دقائق، لذلك يسبب زيادة إفراز أنزيم الدريقية تحرر الكالسيوم من العظام وينتج عن ذلك هشاشة في العظام، وفي الحالات المتقدمة يؤدي ذلك إلى التكيس الطبقي للعظم وحصى كلوي.

في صور الطب النووي يتم حقن المواد المشعة وريدياً وتقوم الغدد الدريقية بتركيزها ويؤدي ذلك إلى تحديد مكانها بدقة زائدة أما التصوير التلفزيوني فيعتبر واحداً من اكثر الطرق شيوعاً كإجراء أولي للكشف عن الغدد الدريقية التي بها ورم، وهو يظهر الغدة بشكل دائري مع وجود حويصلة لها، بينما يظهر تضخم الغدد الدريقية على شكل بيضوي، وفي كلتا الحالتين تكون كثافتها أقل من الغدة الدرقية وبحساسية 24-83% أما بالنسبة للتصوير الطبقي المحوري فيتم عمل معظم الحالات مع حقن المادة الظليلية لأن الغدد الدريقية المريضة تظهر كبيرة الحجم وتظهر استقطاباً شديداً للمادة الظليلية وبحساسية من 46-87% ويظهر تصوير الرنين المغناطيسي الغدد الدريقية المريضة بكثافة مساوية أو أقل من الأنسجة الرخوة في T1، بينما تكون ذات كثافة عالية في T2 وتظهر استقطاباً شديداً للمادة الظليلية وبحساسية 50-88%.

وتم عرض جميع التغيرات التي تحدث في العظام في أفلام الأشعة العادية التي لها حساسية 30-50% وأيضا تمَّ التطرق للمداخلات الشعاعية التي يتم عملها في فرط الغدد الدريقية التي تصل حساسيها إلى 83%.

وفي الختام تم استخلاص النتائج والتي من أهمها أن التصوير التلفزيوني أو فحص الطب النووي هو الفحص الذي يجب عمله أولاً، وبعد ذلك يتم تحديد الخطوة التالية حسب نتائج الفحص.

الكلمات الدالة: فرط الدريقية الأولي، التصوير النووي، التصوير التلفزيوني، التصوير الطبقي المحوري، تصوير الرنين المغناطيسي.