Parathyroid glands: so small but yet so big

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Learning objectives

• To describe the actual role of imaging in hyperparathyroidism with emphasis in Four-Dimensional Computed Tomography (4D-CT).
• To review the anatomy and physiology of parathyroid glands.
• To discuss the surgical techniques and the postsurgical hyperparathyroidism.
Background

Hyperparathyroidism is a relatively common disease that can cause multiple symptoms due to the alteration of phosphocalcic metabolism. Parathyroid adenoma is the main cause of primary hyperparathyroidism.

Preoperative location of the lesion is essential for planning a targeted parathyroidectomy. Identifying the glands may be challenging due to their variable location given its embryological development. In the past, ultrasound (US) and Scintigraphy have been the main diagnostic tests but recently, the 4D-CT appears promising for the better diagnosis of parathyroid pathology.
Findings and procedure details

OVERVIEW:

Parathyroid glands regulate calcium homeostasis by secreting parathyroid hormone (PTH) when there is hypocalcaemia. PTH stimulates calcium (Ca) absorption at different levels: kidneys, bones and bowel (# Ca: # PTH # # Ca).

Normal glands are small (6 x 4 x 2 mm) and frequently not detectable on imaging. There are usually four glands although, occasionally, they can be supernumerary (2 - 9 %). Eutopic location is deep to midpole of the thyroid lobes for superior glands and inferior or deep to lower poles for the inferior glands. Despite this, the glands may have an ectopic situation.

Glands are irrigated by branches of thyroid arteries and they have a close relationship with the recurrent laryngeal nerves which may be accidentally injured during parathyroidectomy.

CLINICAL SETTINGS:

Hyperparathyroidism is a relatively common disease predominantly affecting 50 - 70 year-old women. It is generally sporadic but it may be hereditary in the context of multiple endocrine neoplasia (MEN) syndrome.

Clinically, it causes variable symptoms affecting bones, genitourinary system, central nervous system and gastrointestinal system and is potentially lethal due to cardiac risk.

Primary hyperparathyroidism consists of an increased PTH secretion (or inappropriately normal in the setting of an elevated serum calcium level) that causes high serum calcium levels and normal or decreased serum phosphate (P) levels (PTH #, Ca #, P n/#).

It is caused by parathyroid adenoma (near 85 %), hyperplasia (around 15 %) or carcinoma (very rare) with the exception of MEN syndrome in which 90 % of patients have parathyroid hyperplasia.

Secondary hyperparathyroidism is diagnosed when PTH is elevated with hypocalcaemia and high phosphate levels (PTH #, Ca # P #). There is an increased secretion of PTH in response to any cause of hypocalcaemia such as renal failure: phosphate renal retention increases calcium excretion to maintain normal mineral equilibrium. Subsequently, parathyroid glands increase blood PTH levels to compensate the existing hypocalcaemia. As a result, there is not hypercalciuria or nephrolythiasis. Chronic renal
failure is the most common cause of secondary hyperparathyroidism and other causes include malnutrition and vitamin D deficiency.

Tertiary hyperparathyroidism is the result of prolonged gland stimulation in the setting of a secondary hyperparathyroidism that provokes autonomous function of parathyroid tissue by and autoimmune mechanism finally causing hypercalcaemia (# PTH, Ca #, P #). In fact, the main cause of hypercalcaemia in renal failure is a tertiary hyperparathyroidism (given that secondary hyperparathyroidism provokes hypocalcaemia).

Pseudohyperparathyroidism is considered when serum PTH levels are increased due to peripheral hormone resistance and, therefore, serum calcium and phosphate levels are low (# PTH, # P, # Ca).

On the other hand, in hypoparathyroidism, blood PTH levels are low leading to decreased serum calcium levels and increased phosphate levels (PTH #, Ca #, P #). Pseudohypoparathyroidism is considered when serum PTH is increased but there are low serum calcium levels and high phosphate levels (PTH #, Ca #, P #). Typical clinical manifestations of hypoparathyroidism are paresthesias, muscular or laryngeal spasms, tetany and seizures and there may be intracranial calcifications (in basal ganglia, cerebellum, subcortical white matter, corona radiate and thalamus).

**EMBRIOLOGY AND ANATOMY:**

Parathyroid glands originate from the pharyngeal endoderm at the fifth week of gestation: superior glands originate from the fourth branchial pouche (as well as the thyroid) and inferior glands from the third pouche (along with the thymus).

Inferior parathyroid glands and thymus migrate caudally until parathyroid glands separate from the thymus and finally rest in the posterior region of the lower pole of the thyroid. Inferior glands have more variable location than superior glands: embryological descent of the thymus extends from the angle of the jaw to the pericardium so any alteration in the parathymic migration may cause ectopic inferior parathyroid glands, usually in an inferior and anterior location. Ectopic inferior glands may be found along the thyrothymic ligament, within the thymus gland or in the anterior mediastinum and, less frequently, cranially near the carotid bifurcation or the superior pole of the thyroid.

Superior parathyroid glands follow thyroid migration; they have a short descent path, hence their location is more stable and they are usually located behind the middle third of the thyroid lobes (80 %). Nevertheless, they may also be ectopic, usually posteriorly along the tracheoesophageal groove or, less frequently, in the posterior mediastinum (retroesophageal or retropharyngeal space). A parathyroid gland located next to the
inferior lobe of the thyroid may be an ectopic superior gland as well as an eutopic inferior gland.

Given their common embryological origin, some superior glands may be located within the thyroid capsule or the thyroid parenchyma. Inferior glands are rarely intrathyroidal because they have separate embryological origin from the thyroid gland.

In conclusion, ectopic parathyroid glands may be located anywhere in their path of migration from the hyoid bone to the carina.

Normal glands are small in size, measuring approximately 6x4x2 mm, frequently oval or elongated but they may be multilobulated (1 %). Usually there are four parathyroid glands but supernumerary glands may be present (2-9 %).

**ROLE OF IMAGING:**

- **US**

US sensibility for parathyroid adenomas reaches 82 %.

Normal parathyroid glands are isoechoic to thyroid parenchyma although they are frequently not seen on US given their small size.

Parathyroid adenomas tend to be homogenously hypoechoic to the thyroid (Fig. 1 on page 16, Fig. 2 on page 16, Fig. 3 on page 17, Fig. 4 on page 18).

Doppler examination reveals a hypervascular lesion and an extrathyroid polar vessel (frequently a branch of a thyroid artery) may be seen surrounding the adenoma before entering it at one of the poles (vascular arc) (Fig. 2 on page 16). Vascular pattern of a parathyroid adenoma consists of internal flow with peripheral branching (in contrast to the central hilium of a lymph node).

Atypical adenomas may have anechoic components due to cystic degeneration (Fig. 5 on page 19, Fig. 6 on page 20 Fig. 7 on page 21). Fine needle aspiration (FNA) of the cyst fluid may be used to measure intracystic PTH levels, which should be elevated in a parathyroid origin lesion.

A hyperechoic band may be seen separating the adenoma from the thyroid tissue (Fig. 6 on page 20).
False-positive results for parathyroid adenomas may be: central compartment lymph nodes (frequent in the context of autoimmune thyroiditis), thyroid nodes or asymmetric thyroid tissue.

False-negative results are mainly due to ectopic locations including intrathyroidal parathyroid adenomas (Fig. 8 on page 22) although maybe due also to microadenomas or atypical adenomas.

A hyperechoic band separating the candidate lesion from the thyroid gland (corresponding to the capsule) is usually seen but this sign is not specific. Another fact to consider for differentiating the candidate lesion from thyroid tissue is that, unlike the thyroid gland, parathyroid adenomas do not usually move when swallowing because most of them are separated from the overlying thyroid capsule.

- **99Tc-MIBI Scintigraphy and Single Photon Emission Computed Tomography (SPECT)/CT**

Parathyroid Scintigraphy sensibility is similar to US (around 88 %) but it is higher for detecting hyperplasia or ectopic adenomas. 99mTc-sestamibi is the radiopharmaceutical of choice for imaging parathyroid pathology.

The test consists of an early stage (10 - 15 minutes) and a late stage (2 - 3 hours). Adenomatous and hyperplasic parathyroid tissue show avid uptake and long retention of radiotracer while activity in the normal thyroid tissue significantly decreases with time. Therefore, asymmetric foci of increased radiotracer uptake on early images that persist on delayed images represent abnormal hyperfunctioning parathyroid tissue (Fig. 2 on page 16, Fig. 3 on page 17, Fig. 4 on page 18).

False-positive results for parathyroid pathology may correspond to thyroid nodes (most frequent), lymph nodes, ectopic thyroid tissue, thymic remnant or tumors. However, they are uncommon in the clinical setting of hyperparathyroidism.

Causes of false-negative results may be small size of the lesion (most common), MEN context, atypical (cystic) adenomas (Fig. 6 on page 20, Fig. 7 on page 21) or early washout of Tc-99m.

SPECT/CT is increasingly being used specially for locating ectopic adenomas and it has shown to have better sensitivity (68-95 %) than traditional nuclear medicine procedures (Scintigraphy). The fusion of CT with SPECT images has the advantage of combining physiologic information from SPECT and anatomic information from CT and it allows three-dimensional imaging of the parathyroid glands.

SPECT has an early and a delayed phase:
In early-phase SPECT images, parathyroid adenomas are identified as focus of radiotracer accumulation equal to or greater than that in the thyroid (if the adenoma is contiguous with the thyroid gland, it may only be detected if it bulges the thyroid contour or if its radiotracer accumulation is higher than the accumulation in the thyroid). In delayed-phase SPECT images, radiotracer accumulation persists in parathyroid adenomas after washout of radiotracer from thyroid gland.

Finally, multiplanar SPECT images are fused with the CT images for anatomic location (Fig. 3 on page 17, Fig. 4 on page 18).

Potential limitations of SPECT/CT include injection or scanning technique and patient conditions.

- 4D-CT

4D-CT is a relatively recent diagnostic tool helpful to identify, locate and differentiate parathyroid pathology. A 4D-CT is a multiplanar CT (axial acquisition and coronal and sagittal reformations) performed with a basal non-enhanced phase, an arterial and a delayed phase, considering the change of enhancement overtime (phases) as the fourth dimension.

Although 4D-CT is considered a second line tool, it has advantages compared to US and Scintigraphy differentiating adenomas from other mimics and evaluating their anatomical relations. If US or Scintigraphy fail to identify pathology, 4D-CT is more cost-effective than performing another first-line technique or combining both. Furthermore, its sensibility increases when specifically looking for ectopic pathology (79 %).

A correct technique is essential for interpreting 4D-CT results. The protocol consists of:

- A basal non-enhanced study should be performed from the hyoid bone to the superior aspect of the clavicle covering only the area of the thyroid in order to avoid unnecessary radiation. This phase is used to differentiate a candidate parathyroid lesion from iodine-containing thyroid tissue which is already dense on the non-enhanced study.

- An enhanced CT (75ml of contrast at a rate of 4ml/s + 25ml of saline) should cover from the angle of the mandible to the carina to include any possible ectopic locations. Arterial phase should be acquired 25 seconds from the injection and delayed phase acquired 80 seconds after the injection.

All three phases should include thin-section scans.

Patients with contrast allergies or renal failure should only undergo 4D-CT if the benefits outweigh the risks.
Parathyroid adenomas are round or oval hypodense lesions on basal study that have characteristic intense arterial enhancement (138 - 180 UH) and washout of contrast on delayed phase (Fig. 1 on page 16, Fig. 2 on page 16, Fig. 9 on page 23, , Fig. 11 on page 25). This pattern allows differentiating parathyroid pathology from other entities. A polar vessel can be easier identified on 4D-CT.

Atypical adenomas may not have this characteristic contrast enhancement pattern due to less blood flow in the context of internal haemorragic / necrotic degeneration (Fig. 6 on page 20, Fig. 7 on page 21).

A systematic approach to 4D-CT interpretation is essential for identifying parathyroid pathology. The initial search should start by the arterial phase looking for enhancing compatible lesions and be followed by reviewing the non-enhanced and delayed phases for evaluating contrast enhancement pattern (hypodense on the basal study and washout on the delayed phase).

The first step should be looking for parathyroid pathology in the normal (eutopic) locations of the glands. This should be followed by a careful search of possible ectopic lesions anywhere in their path of embryological migration sites from the hyoid bone to the carina. The probable origin of the gland should also be determined taking into account that inferior glands are more variable in location than superior glands:

- In case of a posteriorly located lesion, a superior gland origin should be considered (Fig. 10 on page 24).

- In contrast, if the lesion is located anteriorly, an inferior gland origin should be considered (Fig. 9 on page 23).

The whole study should be revised even once a parathyroid adenoma is identified in order to avoid missing other incidental lesions.

False-negative results may be due to microadenomas, atypical (cystic) (Fig. 6 on page 20, Fig. 7 on page 21) or ectopic adenomas, adjacent thyroid tissue or poor 4D-CT technique (Fig. 12 on page 26).

False-positive results for parathyroid adenomas may be: cervical lymph nodes, thyroid nodes, islands of thyroid tissue, asymmetric thyroid tissue due to previous partial surgery, vessels or other anatomical structures (esophagus, muscles...).

The differential diagnosis can be narrowed taking into account these tips:
Lymph nodes have different contrast enhancement pattern than parathyroid adenomas (Fig. 13 on page 27). They show progressively increasing contrast enhancement with peak at the delayed phase.

Thyroid tissue has intense enhancement in the arterial phase but it will have high attenuation on the non-enhanced images (because of iodine-containing) and will also have increasing enhancement between the arterial and the delayed phase. A fat plane separating the candidate lesion from the thyroid gland is usually seen but the absence of this plane does not exclude a parathyroid adenoma and, if present, sequestered thyroid tissue should still be considered.

Once the lesion/s is/are identified and described, a complete radiologic report should include anatomical relations of the lesion (especially with the thyroid gland, the carotid, the inferior thyroid artery, the trachea and the tracheoesophageal groove).

CASE-BASED SCENARIOS:

- Adenoma, hyperplasia and carcinoma: Location. Typical and atypical findings. Preoperative evaluation, differential diagnosis and potential pitfalls.

PARATHYROID ADENOMA

Parathyroid adenoma is the main cause of hyperparathyroidism.

They are typically solid and hypervascular ovoid lesions being frequently the largest diameter the cranio-caudal. Up to 1 % of adenomas may be multilobulated (especially bigger ones). Calcifications are rare.

The average size of adenomas is around 0,8 - 1,5 cm; when smaller, it is considered a microadenoma; when bigger, a carcinoma should be ruled out.

A polar-vessel sign may be identified on imaging (Fig. 2 on page 16): the feeding artery (usually a branch of the inferior thyroid artery) can be enlarged and tortuous because of its increased blood flow.

Cystic adenomas are not frequent (1-4 % of parathyroid adenomas) and they may be completely cystic or have a solid pole irrigated by a polar vessel. Due to their cystic component, they may have atypical pattern on imaging (including Scintigraphy) and, therefore, it is important to keep them in mind when interpreting diagnostic tests (Fig. 5 on page 19, Fig. 6 on page 20, Fig. 7 on page 21).
Cystic adenomas may be functional or nonfunctional: both types have increased intracystic PTH levels but only functional adenomas provoke increased serum PTH levels.

Functional adenomas reach 85% of resected adenomas although this percentage may be biased because functional adenomas are more symptomatic and mostly removed. They are provoked by cystic degeneration of an adenoma in the context of a hypercalcaemia crisis and are seen on imaging as complex cysts with turbid liquid due to intracystic necrosis and haemorrhage. Hyperparathyroidism is the clinical manifestation of functional adenomas.

Nonfunctional adenomas are unilocular simplex cysts with clear liquid that may occur by abnormal retention of PTH, microcysts coalescence or an embryological remnant of the brachial apparatus. Mass effect symptoms (dysphagia, dysphonia) may be present or they may be incidentally diagnosed.

Between 5-25% of parathyroid adenomas are ectopic. As said before, parathyroid glands can be located anywhere in their path of migration from the hyoid bone to the carina. A parathyroid adenoma located next to the inferior lobe of the thyroid is not always an inferior gland adenoma but it may also be an ectopic superior gland adenoma (Fig. 8 on page 22).

Due to their embryological development, a posterior mediastinal parathyroid adenoma is more characteristic of a superior parathyroid gland (Fig. 10 on page 24) and, in contrast, an anterior mediastinal parathyroid adenoma will be more likely from the inferior parathyroid gland (Fig. 9 on page 23).

Surgery is the treatment of choice for parathyroid adenomas (Fig. 4 on page 18, Fig. 7 on page 21, Fig. 11 on page 25, Fig. 12 on page 26). Recurrent laryngeal nerves must be preserved during surgery (Fig. 4 on page 18).

PARATHYROID HYPERPLASIA

Parathyroid hyperplasia should be considered when there is more than one enlarged gland (the term "multiple adenoma" is controversial) (Fig. 14 on page 28, Fig. 15 on page 29). It is anatomopathologically indistinguishable from adenomas.

Asymmetric hyperplastic glands may be mistaken for an adenoma but, in such cases, intraoperative serum PTH levels will remain elevated after gland removal.

PARATHYROID CARCINOMA
Parathyroid carcinoma is rare (less than 1% of cases of hyperparathyroidism). It should be suspected when an ill-defined heterogeneous mass, usually larger than an adenoma is identified, although diagnosis is frequently performed intraoperatively.

- **Association with familiar syndromes**

Parathyroid pathology may be part of genetically determined disorders comprised in Multiple Endocrine Neoplasia (MEN).

Parathyroid (95%), pituitary and pancreatic tumours are the major disorders of MEN type 1. Primary hyperparathyroidism is the usual presenting feature, frequently affecting multiple glands and requiring reoperations due to recurrent hypercalcaemia after surgery (Fig. 12 on page 26).

Parathyroid hyperplasia or tumour, medullary thyroid carcinoma and pheochromocitomas are characteristic of MEN type 2 (MEN 2A). Parathyroid pathology in MEN type 2 has later onset and lower morbidity and it is also multiglandular.

- **Beyond the glands: Central nervous system, skeletal and genitourinary manifestations of hyperparathyroidism**

Bone disease is frequently present in hyperparathyroidism (32%) (Fig. 16 on page 30). Increased levels of PTH triggers osteoclastic activity leading to bone resorption with cortical thinning and osteopaenia. Bone weakening can provoke pseudofractures or real fractures.

Bone resorption may involve variable skeletal sites although the earliest changes are frequently seen in hands. Subperiosteal bone resorption is more prominent at the radial aspect of the phalanges, especially the proximal and middle phalanges of the second and third fingers, being a pathognomonic sign of primary hyperparathyroidism.

Loss of lamina durae dentium resultant in absence of teeth may also be present.

Subcchondral, subligamentous or intracortical resorption may also be present.

Other radiographic features may be rugger-jersey spine (alternating dense-lucent-dense appearance due to prominent subendplate densities at multiple contiguous levels), salt and pepper sign in skull (multiple millimetric hyperlucent areas caused by resorption of trabecular bone causing a ground-glass appearance), chondrocalcinosis and other calcium deposits.

Brown tumors are a reparative cellular process that represents the terminal stage of bone pathology in hyperparathyroidism rather than a neoplastic process. They are well
defined radiolucent lesions that may expand the cortex but will not penetrate it, distributed anywhere in the skeleton but characteristically in pelvis, ribs and femur.

Genitourinary symptoms are due to recurrent renal stones (35%) being the kidneys the most common site of calcium deposition in hypercalcaemia.

The gastrointestinal system may also be affected by hypercalcaemia presenting with peptic ulcers (20%), pancreatitis (1.5-7%) and irritable bowel syndrome.

Other abnormalities associated to hypercalcaemia may be hypertension with left ventricular hypertrophy, myocardial calcification and, rarely, valvular calcification.

Calcium can also deposit in basal ganglia and cerebral white matter.

**SURGERY: Parathyroidectomy and glandular reimplantation. Complications. Postsurgical hyperparathyroidism: How to locate the site of recurrence (Casanova Test)**

Obtaining normal mineral metabolism by reducing parathyroid tissue is the aim of surgery. Monitoring intraoperative serum PTH is an indicator of adequate resection.

Surgical removal of the adenoma / hyperplasia is the treatment of choice in primary hyperparathyroidism. Targeted parathyroidectomy is essential to minimize surgical risk and the other glands should only be explored if intraoperative PTH serum levels do not descend after adenoma extirpation.

In secondary hyperparathyroidism, recurrence is frequent after subtotal parathyroidectomy and patients may require neck reoperation which increases the risk of complications. Total parathyroidectomy and forearm autotransplantation of parathyroid tissue (to prevent postsurgical hypoparathyroidism) is an alternative treatment for patients with vitamin D resistant secondary hyperparathyroidism.

After total parathyroidectomy, persistent or recurrent hyperparathyroidism may be due to residual tissue left in the neck, supernumerary gland not found at surgery or hyperplasia of grafted tissue. Precise location of the origin of hormone excess may be a challenge but it is crucial to perform a targeted surgery that avoids unnecessary neck reoperation.

Casanova's test is a valuable non-invasive method for a correct assessment of graft function. It assesses parathyroid graft tissue function by a transitory ischemic blockade of the arm with consequent rapid reduction of PTH serum levels in patients with graft-
dependent hyperparathyroidism. In these cases, graft excision is required and neck reoperation is unnecessary.

On the other hand, a negative response to the ischemic blockade (persistent elevated serum PTH levels during the maneuver) indicates that the hormone source is not in the forearm implant.

TAKE HOME MESSAGES:

• Parathyroid adenoma is the main cause of primary hyperparathyroidism, which is a multisystemic disease.

• Eutopic parathyroid glands are located immediately posterior or inferior to the thyroid gland. However, parathyroid glands may be ectopic and located anywhere in their path of migration from the hyoid bone to the carina.

• Inferior glands have a more variable location than superior glands:
  - Inferior gland adenomas may fall anteriorly in the region of the thyrothymic ligament.
  - Superior gland adenomas may fall posteriorly in the tracheoesophageal groove.

• The majority of parathyroid adenomas are eutopic but they may also be ectopic (up to 5%). Common locations for ectopic parathyroid adenomas include:
  - Posterior mediastinum (always check out the tracheoesophageal groove): think of a superior gland origin.
  - Anterior mediastinum: think of an inferior gland origin.
  - If you do not find the lesion: think about an intrathyroidal adenoma.
  - If you still do not find it: look at the carotid sheath.

• On US, parathyroid adenomas tend to be homogenously hypoechoic to the thyroid. Look for the extrathyroidal polar feeding vessels and remember the peripheral pattern of internal vascularity.

• 4D-CT is a useful tool for identifying parathyroid pathology especially for locating ectopic adenomas. On 4D-CT, parathyroid adenomas are round or oval hypodense lesions on basal study that have characteristic intense arterial enhancement and washout of contrast on delayed phase.
• Cystic adenomas are not frequent and may have atypical enhancement pattern on imaging. Although not frequent, do not forget about them since they are a common cause of false-negative nuclear medicine tests. Remember also that, in selected cases, a FNA may be performed to determine the intracystic levels of PTH if necessary.

• Remember that an accurate diagnosis is essential to minimize surgical risk by performing a targeted parathyroidectomy. Surgeons at our Hospital refer that the duration of surgery in our institution has been radically reduced since the use of 4D-CT, saving them hours of surgery and the uneasiness of not knowing beforehand where to look for the adenoma.
Fig. 1: 68-year old woman with primary hyperparathyroidism.

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Fig. 2: 52-year-old woman with history of renal stones and hypercalcemia.

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Fig. 3: 70-year old man with primary hyperparathyroidism.

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**Fig. 4:** 75-year old woman with increased serum calcium and PTH levels.

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Fig. 5: 25-year-old woman with history of renalureteral crisis and elevated calcium and PTH serum levels. Scintigraphy and 4D-CT were unsuccessful (not shown) due to the cystic component.

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Fig. 6: 72-year old woman with suspected primary hyperparathyroidism.

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Fig. 7: 70-year-old woman with primary hyperparathyroidism.

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Fig. 8: 77-year old woman with primary hyperparathyroidism.

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Fig. 9: 59-year old woman with primary hyperparathyroidism. US and Scintigraphy were unsuccessful (not shown).

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Fig. 10: 76-year-old woman with hypercalcemia. Primary hyperparathyroidism was suspected. US and Scintigraphy were unsuccessful (not shown).

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**Ectopic parathyroid adenoma:** despite its location adjacent to the right inferior lobe of the thyroid, the origin of the adenoma is an ectopic superior gland instead of an eutopic inferior gland.

**A** Axial US image shows an ovoid hypoechoic lesion deep to the inferior aspect of the right thyroid lobe (arrow).  
**B** Sagittal Doppler-US image demonstrates internal blood flow (arrow).  
Non-enhanced **C**, arterial **D** and delayed **E** phase 4D-CT axial images reveal early enhancement of the lesion and delayed contrast washout (arrows).  
**F-G** Surgical confirmation of an ectopic right superior parathyroid adenoma (arrows).

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**Fig. 11:** 69-year old woman with primary hyperparathyroidism.

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Fig. 12: 25-year old man with recurrent hyperparathyroidism four years after total parathyroidectomy in the setting of MEN syndrome type 1. US and Scintigraphy were unsuccessful (not shown).

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Fig. 13: 58-year old woman with no history of hypercalcaemia.

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Fig. 14: 26-year old man undergone renal transplantation, with renal failure and subsequent secondary hyperparathyroidism.

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**Fig. 15:** (Same patient as in previous figure) 26-year old man undergone renal transplantation, with renal failure and subsequent secondary hyperparathyroidism.

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Fig. 16: 46-year old man with secondary hyperparathyroidism.

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Conclusion

Parathyroid pathology is a prevalent and clinically relevant disease. Therefore, it is important for radiologists to be updated with the available radiological techniques and the imaging features to make an accurate diagnosis that minimizes the surgical risk.

4D-CT is a useful tool to locate ectopic parathyroid pathology.
References