

Diagnostic value of radionuclide scanning and ultrasonography in thyroid developmental anomaly imaging

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Abstract

Thyroid is particularly prone to morphogenetic variability. Developmental failure of the thyroid gland is in 85% of cases the underlying cause of congenital hypothyroidism, diagnosed at birth with a frequency of 1:3000–1:4000 newborns. However, the incidence of less severe developmental variants of the thyroid is much higher. Determination of the aetiology of congenital hypothyroidism is crucial for predicting its severity and outcome as well as impacts dose of L-thyroxine during substitution. Thyroid imaging is necessary to establish diagnosis, and it involves mainly thyroid ultrasound examination and scintiscan. Awareness of both the advantages and limitations of sonographic and scintigraphic imaging are central to the successful interpretation of their results and reasonable recommendation of these procedures for patients with thyroid developmental anomalies of different age and clinical picture. Hence, the aim of this review is to provide the most important and up-to-date information

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on the place of radionuclide scanning and ultrasonography in visualization of different thyroid developmental abnormalities.

Key words: thyroid dysgenesis, radionuclide imaging, thyroid ultrasonography

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Introduction

Thyroid gland is particularly prone to morphogenetic variability. The total incidence of thyroid developmental abnormalities in the population is difficult to assess. The occurrence of thyroid gland anomalies leading to congenital hypothyroidism (CH) can be estimated on the basis of data from newborn TSH screening [1]. However, the incidence of asymptomatic developmental variants of thyroid morphogenesis, usually not manifesting clinically, remains unknown. Anatomical variants like thyroid haemiagenesis, accessory thyroid, thyroglossal duct cyst, or pyramidal lobe, if not associated with local symptoms or disturbances in thyroid hormonal function, usually remain undiagnosed.

The aim of this review is to provide the most important and up-to-date information on the place of radionuclide scanning and ultrasonography in visualization of thyroid developmental abnormalities.

Congenital hypothyroidism — definition and diagnosis

Congenital hypothyroidism is defined as thyroid hormone deficiency present at birth. It occurs in Europe with a frequency of 1:3000–1:4000 newborns [2–4]. Nowadays, in most developed countries, CH is diagnosed by neonatal screening [5]. Blood is collected on the third day after birth and abnormal results of TSH concentration tests mean the child is recalled for further evaluation. The subsequent diagnostic tests involve confirmation

of the diagnosis, which comprises repeated TSH measurement as well as additional serum thyroxine and thyroglobulin assessment [6]. The diagnosis of CH is followed by immediate introduction of L-thyroxine substitution. The treatment is continued throughout at least the first three years of life, which are crucial for neuropsychological development. Therapy may be discontinued when diagnosis of transient CH is made after a four-week trial of L-thyroxine withdrawal, or maintained lifelong if temporary suspension of substitution results in confirmation of the permanent form of CH [7]. The most common causes of CH are thyroid developmental anomalies. Thyroid dysgenesis, responsible for inappropriate results of neonatal TSH screening tests in 85% of cases, involves thyroid agenesis, ectopy and hypoplasia, and less frequently thyroid haemiagenesis. A second common cause of CH is dyshormonogenesis, which is responsible for 10–15% of cases diagnosed [1, 8].

Why is it so important to establish the aetiology of congenital hypothyroidism?

Some clinicians believe that, since the management of CH is the same regardless of its origin, establishing the cause of CH is not obligatory. Such an opinion is based on the conviction that imaging, especially with radionuclide isotopes, may be harmful to children. What is more, as the thyroid scintiscan should be performed under conditions of endogenous TSH stimulation, it might result in an unnecessary delay in treatment introduction. Meanwhile, determination of the aetiology of CH seems as important as treatment. However, to establish diagnosis, thyroid imaging is necessary, which mainly involves thyroid ultrasound examination and scintiscan.

Studies have demonstrated that athyreotic children present worse neuropsychological development in comparison to children with thyroid hypoplasia, ectopy, or dyshormonogenesis, and hence require higher doses of L-thyroxine [9–12]. Thus, the determination of CH aetiology is crucial for predicting its severity, outcome as well as impacts L-thyroxine titration. The aetiopathogenesis of thyroid dysgenesis is still unknown [1]. Genetic factors were proven to have a causative role in only 2% of cases. Moreover, thyroid developmental abnormalities are probably of multifactorial origin, and Mendelian transmission almost never happens [13]. Thus, most cases occur sporadically, and the risk for the second child of a couple to be born with thyroid developmental failure is close to that of the general population. On the other hand, dyshormonogenesis is usually inherited in an autosomal recessive manner; hence, the risk for another child is 25%. Therefore, differentiation of the cause of CH is particularly important to ascertain reliable parental genetic counselling [6].

The role of thyroid scintigraphy and ultrasonography in determining the aetiology of congenital hypothyroidism

Ultrasound examination

Recent years have brought about widespread use of ultrasound techniques in the diagnostics of the thyroid gland. The technical development of sonographic imaging has converged together with its increasing accessibility [14]. The low cost of the

examination also plays an important role in its availability. Imaging in CH primarily involves ultrasound examination, which requires no preparation, and can thus be performed and repeated at any time, without interruption of treatment. It allows repeatable, real-time, and non-invasive assessment of the thyroid morphology. This is also a rapid, safe, and comfortable examination. It does not expose patients to radioactive isotopes or Roentgen radiation and so can be performed with no harm in children at any age. With the employment of colour Doppler technique, enabling visualization of tissue vascularisation, the thyroid can easily be discriminated from other surrounding tissues [15]. Ultrasound examination does, however, provide entirely morphological information and no conclusions pertaining to the hormonal function of the observed structures can be derived on the basis of this examination. Sonography is also useful due to its high sensitivity in the detection of thyroid pathologies. It allows the demonstration of pathological conditions accompanying thyroid developmental anomalies, i.e. concomitant focal lesions as well as autoimmune or neoplastic processes.

Despite its unquestionable advantages, this examination also has several limitations. The ultrasound waves do not penetrate through calcified structures; hence, sonographic assessment of ectopic thyroid in retrosternal, intralaryngeal, or intratracheal locations is largely limited. Although some of the features of thyroid tissue detected exclusively on the basis of ultrasound examination augment the suspicion of a malignant transformation, the differentiation between a benign and a malignant lesion cannot be performed entirely on the basis of ultrasonographic assessment. Finally, it must be remembered how crucial the quality of the equipment used for the examination and the experience of the sonographer are for the credibility and reproducibility of this examination [14]. Additionally, the interpretation of sonographic pictures taken from neonates requires broad experience and awareness of specificities of paediatric head and neck anatomy. For the inexperienced sonographer, other anatomical structures as the thymus, oesophagus, or lymph nodes might be misdiagnosed as thyroid gland [16]. Moreover, worse cooperation of a young patient is another factor influencing the quality and reliability of the examination. The minimal technical requirements should also be fulfilled, and a linear probe of 7.5 MHz or higher frequency ought to be used. It is important that the whole region of the neck is scanned in searching for the presence of ectopic thyroid tissue. However, one of the significant limitations of ultrasonography is poor sensitivity in visualization of ectopic thyroid, if compared to radionuclide scanning [17, 18].

Diagnosis of thyroid of normal size or goitre on ultrasound examination is suggestive of dyshormonogenesis or transient CH due to prenatal exposure to excessive iodine, antithyroid drugs, or TSH receptor (TSH-R) blocking autoantibodies. However, discriminating is thyroid scintiscan.

The study of Beltrao et al. revealed close concordance of diagnoses established by scintiscan and ultrasonography [16]. The discordance, at a level of 18%, mainly concerned cases diagnosed as thyroid agenesis on ultrasound examination while the scintiscan revealed the presence of ectopic thyroid.

Scintigraphy

Some authors support the idea of performing the diagnostic scintiscan in the neonatal period before L-thyroxine is administered,

during the period of natural endogenous TSH stimulation. The need for establishing the reason for CH should not result in delay in treatment as TSH concentration in subjects with confirmed CH is markedly elevated (usually above 100 $\mu\text{IU/ml}$) and remains elevated for many days after onset of treatment, during which time scintigraphy can be performed [19]. Whereas sonographic assessment of even new-born children is considered harmless, application of radioisotopes for scanning in neonates still raises many controversies due to unknown implications of exposure to radiation at such a young age. Nevertheless, ^{123}I thyroid imaging has been used for many decades without evidence of risk for thyroid cancer.

Permanent CH can be assumed if ultrasonography or radionuclide imaging shows an absent or ectopic thyroid gland, consistent with athyreosis or thyroid dysgenesis, one demonstrate dyshormonogenesis, or if at any time during the first year of life the serum TSH rises above 20 $\mu\text{IU/l}$ due to undertreatment. If permanent CH has not been established by two to three years of age, the American Academy of Pediatrics (AAP) and the European Society of Paediatric Endocrinology (ESPE) recommend a 30-day trial off L-thyroxine therapy [20, 21]. In most guidelines it is recommended that scintiscan be performed optionally at the age of three years, during temporary L-thyroxine substitution withdrawal, which is also necessary to differentiate subjects with transient and permanent forms of CH. The cessation of L-thyroxine treatment should last for at least four weeks and the child is also recommended to follow a low-iodine diet for at least two weeks preceding the examination. Thus, the procedure is not recommended at the developmental age below three years, due to potential devastating consequences on neuropsychological development. Hence, the establishing of CH diagnosis associated with temporary L-thyroxine withdrawal is advised at the age of three to five. What is also important, cooperation with the patient during the examination at a more advanced age is also much easier. Some authors suggest that radionuclide scan should be performed on TSH stimulation without L-thyroxine withdrawal as human recombinant TSH (hr-TSH) has not yet been registered for this purpose [22, 23]. With the expected availability of rh-TSH, testing for recovery of the gland will be easier and safer. Indeed, without discontinuing replacement therapy, thyroid imaging with ^{123}I performed after rh-TSH injection will indicate if a gland is present orthotopically with acceptable size and uptake, which will allow for safe discontinuation of exogenous L-thyroxine.

In contrast to ultrasonography, the great advantage of scintigraphy lies in its effectiveness in identification of functioning thyroid tissue. Additionally, some general conclusions on thyroid presence, localization, and size may also be derived on the basis of scintiscan. Hence, it allows cases of thyroid agenesis to be differentiated from ectopy or hypoplasia.

The radionuclides used in diagnostics of CH are ^{123}I and technetium ^{99}Tc pertechnetate (TcP). To perform scintigraphy with ^{123}I , 30 to 50 μCi [$(1.11\text{--}1.85) \times 10^6 \text{ Bq}$] of ^{123}I is administered orally and an uptake image is taken in 3 to 6 hours and, if necessary, another image in 24 hours. For technetium imaging, 0.5 to 1 mCi [$(1.85\text{--}3.7) \times 10^7 \text{ Bq}$] of TcP is administered intravenously and an image is taken 20 minutes later; hence it provides almost immediate information in comparison to longer and more complex procedure of ^{123}I scintiscan. The use of ^{131}I is controversial due to the relatively large dose of radioisotope administered during examination [6, 19].

In the study by Schoen et al., among 210 infants with CH, normal thyroid was diagnosed more often by ^{123}I scintigraphy (49%) in comparison to TcP scintiscan (31% of cases) [19]. Hence, the use of TcP might lead to misdiagnosis of thyroid dysgenesis in cases where eutopic thyroid was visualized when ^{123}I had been used. This might have significant implications on parental counselling about the certainty of permanent forms of CH and the possibility of discontinuation of L-thyroxine substitution in the future.

Recommendations suggest preferential use of ^{123}I due to the specificity of this radionuclide to be trapped entirely in thyroid cells, while TcP is taken up by multiple tissues, including salivary glands, which might disturb the interpretation of scintigraphic pictures, especially in the case of thyroid ectopy. Another advantage of ^{123}I is its specific affinity to thyrocytes, meaning that radiation absorbed during ^{123}I scintigraphy is limited to the thyroid tissue. The total body irradiation is three times lower than that of a chest radiographic evaluation and equivalent to the amount of radiation received during a round-trip (10-hour) transcontinental flight on a commercial airline or during one month living at sea level [19]. ^{123}I thyroid scintigraphy may also be helpful in defining underlying mechanisms of dyshormonogenesis. Unfortunately, ^{123}I is more expensive than TcP and must be ordered in advance, while TcP is routinely stocked by nuclear medicine departments and is thus more easily available than ^{123}I . It is believed that the results of TcP scintiscans are valid only for findings of eutopic or absent thyroid glands. However, on scintiscans with TcP thyroid ectopy is more prevalently diagnosed, while less frequently eutopic thyroid is described [24].

It is recommended that in assessment of CH, imaging studies should be compared to thyroglobulin concentration levels, which are a biochemical marker of the amount of thyroid tissue. When the level of thyroglobulin is undetectable, radionuclide scanning may be avoided and complete thyroid agenesis should be diagnosed. Inversely, subjects presenting with normal or enlarged thyroid gland accompanied by low or undetectable thyroglobulin levels raise suspicion of thyroglobulin deficiency [6].

Large thyroid of eutopic localization presenting increased radioisotope uptake suggests dyshormonogenesis, with exclusion of iodide trapping. In determining the type of dyshormonogenesis, perchlorate discharge test and genetic studies might be helpful [6, 19].

Some authors suggest that radionuclide scanning can be replaced with less harmful examinations, including ultrasonography and thyroglobulin assessment, while radioisotope imaging should be restricted to cases in which non-invasive diagnostic techniques failed to establish diagnosis. A serious limitation of the radionuclide scan is that the absence of radioisotope uptake, mimicking thyroid agenesis, can also be observed in TSH-R gene mutations, iodide trapping defects, and transient CH in children born from mothers with anti-TSH-R blocking antibodies (TRAb), mothers on antithyroid drugs, or mothers exposed to excessive iodine. Hence, only thyroid ultrasonography or biochemical studies, including measurement of serum Tg or TRAb, would help in differential diagnosis [6, 15, 21].

One more limitation of scintigraphic imaging is the lack of objective standards for interpretation of scintiscans of patients diagnosed due to CH.

Another problem is that even positive results of L-thyroxine withdrawal trial and visualisation of normal thyroid gland on scin-

tiscan, excluding permanent forms of CH, do not always result in discontinuation of treatment. Some physicians and parents are unwilling to agree to cessation of L-thyroxine, being afraid of devastating implications of even slight thyroid dysfunction at the developmental age [19].

However, the diagnosis of the cause of CH with the use of scintigraphic imaging offers parents and physicians a chance to give the most effective counselling and lifetime prognosis of CH [19]. In such a context, the use of TcP may lead to overdiagnosis of thyroid ectopy; hence, ^{123}I scintigraphy should be preferred.

The scintigraphic and sonographic picture of thyroid developmental anomalies

Thyroid agenesis

Thyroid agenesis is characterised by developmental failure of thyroid primordium or involution of thyroid anlage at the very early stages of embryogenesis. It results in a lack of functional thyroid tissue, named athyreosis. It occurs with similar frequency in men and women. Thyroid agenesis is the cause of CH in 35–40% of cases [25].

On ultrasound examination the thyroid cannot be visualized, neither in its typical nor potential ectopic localizations. In the thyroid bed, only cystic structures of unknown origin and clinical significance can be observed (Figure 1) but not presenting as functioning thyroid tissue on thyroid scintiscans [14]. Diagnosis of athyreosis is confirmed by thyroid scintiscan, where no radio-

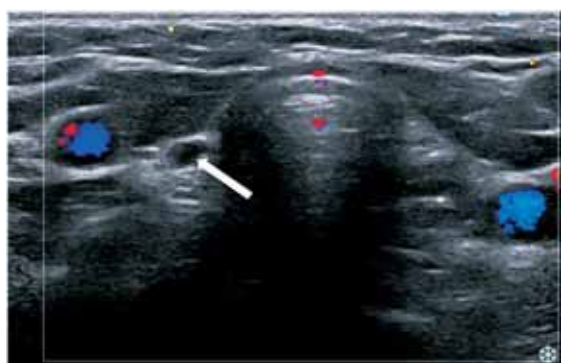


Figure 1. Cystic structure in the right thyroid bed (arrow) in a patient with thyroid agenesis. Note also the characteristic indentation of sternothyroid and sternohyoid muscles into thyroid bed.

isotope uptake is present in the head or neck region (Figure 2). This is consistent with undetectable levels of thyroglobulin.

Thyroid hypoplasia

Thyroid hypoplasia is a variant of incomplete morphogenesis of the thyroid gland, in which eutopic thyroid of proper shape presents decreased volume [13]. It is usually accompanied by hormonal dysfunction [26]. Five per cent of patients diagnosed with CH present thyroid hypoplasia [25]. It is usually caused by disturbances in the response of thyroid tissue to trophic action of TSH [26], which might be caused by mutations in TSH-R.

Thyroid presents on ultrasound examination as small, hypotrophic glands often of decreased echogenicity. The scintiscan demonstrates thyroid of proper localization, but the radionuclide uptake might be decreased. The thyroglobulin concentrations may vary, but most typically are lowered in comparison to subjects with thyroids of normal size.

One of the variants of this anomaly is hypoplasia of one thyroid lobe. It usually concerns the left side. One lobe hypoplasia is usually detected accidentally and is associated with less severe clinical consequences as it rarely results in hypothyroidism.

Thyroid ectopy

Ectopy refers to the presence of thyroid tissue in a localization different from that which is typical. It is the result of a disturbed process of descent of the thyroid primordium, which occurs during embryogenesis. As a consequence, the thyroid might be located along the pathway of descent of the thyroid anlage [27]. Autopsy studies reveal that thyroid cells of ectopic location might be histopathologically visualized even in 10% of the population [28]. Most frequently the process of migration of the thyroid primordium is arrested at the very beginning, which results in localization of the gland at the base of the tongue. Lingual thyroid comprises close to 90% of all cases of thyroid ectopy [29]. The incidence of lingual thyroid is estimated at 1:100,000–1:300,000 in the population; however, it is probably far underestimated [30, 31]. Another study suggests that the occurrence of thyroid ectopy is 1:2500–1:4000 patients with thyroid disease [32]. Ectopy is more prevalent in the female sex, with 4–7 fold predominance over males in cases of lingual thyroid [33, 34]. More rarely, sublingual thyroid may occur, when the gland is located between the geniohyoid and mylohyoid muscles. Sporadic cases of ectopic gland are found in submandibular, prelaryngeal, supra-, or inf-

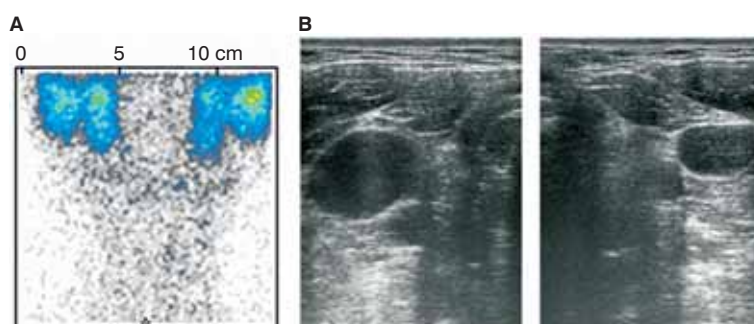


Figure 2. Thyroid agenesis. **A.** Tc-99m thyroid scintiscan. The radioisotope uptake entirely in salivary glands. No thyroid tissue is visible in cervical or any potential ectopic localization. **B.** Ultrasound picture (transverse section).

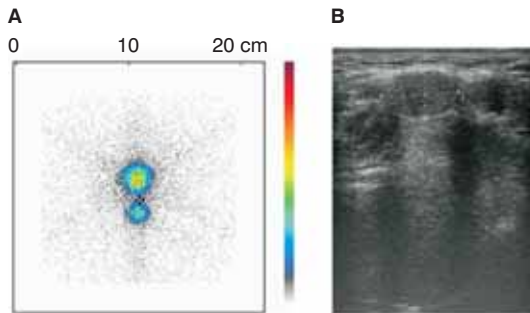


Figure 3. Lingual thyroid. **A.** ^{131}I scintiscan. **B.** Sonographic visualization of a lingual thyroid (transverse section).

rahyoid location. Some cases of ectopy cannot be explained by disturbed descent of the thyroid anlage as thyroid cells can be found in a retrosternal localization inside the trachea, oesophagus, or pericardial sack. Incidentally, ectopic thyroid was found in the retroperitoneum, sella turcica, urinary bladder, or ovary [27]. Due to the lack of the possibility of lateral expansion of the gland, the hormonal function of the ectopic thyroid is usually insufficient to provide adequate hormonal supply. Over 60% of patients with ectopy present with hypothyroidism of varying severity, from incidentally detected subclinical thyroid dysfunction to severe cases of CH [27, 35]. Thyroid ectopy is quoted as the cause of CH in 30–45% of cases [25]. Meanwhile, due to the preserved basic hormonal secretion, patients with thyroid ectopy are most likely to be missed in neonatal screening due to false-negative results of TSH assessment [36]. Figure 3 presents a scintigraphic and sonographic picture of lingual thyroid.

Thyroid haemiagenesis

Thyroid haemiagenesis (TH) is characterised by a congenital absence of one of the thyroid lobes. According to different reports, in 68–88% of cases the left lobe is absent, and an isolated form of left lobe agenesis is most prevalently observed [37, 38]. Less frequently, left lobe and isthmus agenesis, or right lobe and isthmus absence can be found. The incidence of this anomaly is estimated to be 0.20–0.025% [39–42].

On ultrasound examination, only one thyroid lobe can be visualised. It might be enlarged as a result of prolonged exposition to increased TSH concentrations. The compensative enlargement might present as diffuse enlargement or nodular goitre. Decreased heterogeneous echogenicity is less unusual

[38]. On the agenesis side, the indentation of the sternothyroid and sternohyoid muscles into the thyroid bed is observed, while the sternocleidomastoid muscle is located closer to the trachea instead of being separated by the thyroid lobe, as is visible on the contralateral side [43, 44].

The diagnosis of TH is established on the basis of sonographic assessment combined with scintiscan, which is performed to exclude the presence of functional thyroid tissue on the contralateral side to the lobe demonstrated in ultrasonography, and to visualise potential accessory thyroid tissue located ectopically. Figure 4 demonstrates a Tc-99m scintiscan and ultrasound picture of TH. In differential scintigraphic diagnosis of TH other thyroid pathologies causing unilateral isotope uptake should be considered, sometimes known as “functional haemiagenesis”, i.e. autonomous nodule, primary, and metastatic neoplasms, amyloidosis, unilateral thyroiditis, postinflammatory atrophy, or status post hemithyroidectomy [45] (Figure 5).

Besides sonographic assessment, which is the method of choice in discriminating TH from the listed pathologies, other nuclear imaging techniques might be helpful, including ^{131}I scintiscan followed by rh-TSH stimulation, as well as imaging with ^{201}Tl , Tc-99m-sestamibi or Tc-99m-tetrophosmin [46, 47].

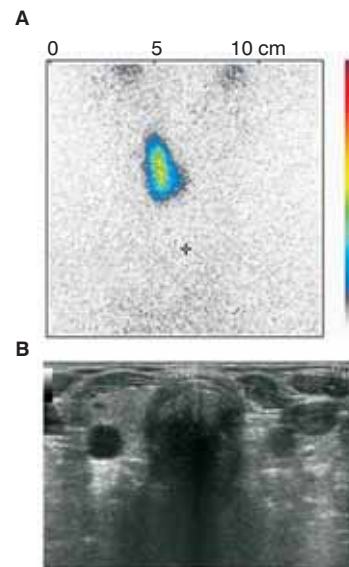


Figure 4. Left-sided thyroid haemiagenesis. **A.** Tc-99m scintiscan. **B.** Sonographic picture (transverse section).

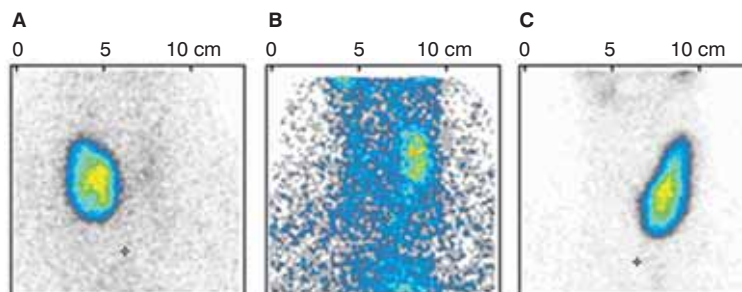


Figure 5. Pathological conditions that, beside thyroid haemiagenesis, should be included in differential diagnosis of unilateral isotope uptake (Tc-99m scintiscans). **A.** Autonomous nodule. **B.** Unilateral subacute thyroiditis. **C.** Status post hemithyroidectomy.

As the function of one thyroid lobe is usually sufficient to provide adequate hormonal production, TH is considered harmless thyroid developmental variant, rarely leading to CH. In the study by Al Taji et al., TH was the cause of CH in 1 out of 170 patients; in the study by Ramos et al. it was the cause in 1% of patients; and in the study by Devos et al. it was the cause in 1 out of 230 newborns with permanent CH [26, 48, 48]. More often, it is responsible for transient hyperthyrotropinaemia or compensated hypothyroidism, which during neonatal screening is more often detected at birth [50]⁵⁰. Most cases of TH, however, are detected in adulthood. Due to its subclinical course, TH is usually diagnosed incidentally during diagnostics of concomitant thyroid diseases or imaging procedures performed due to other indications or as screening examinations.

Scintigraphic and sonographic visualisation of other thyroid developmental variants

Accessory thyroid gland

This term signifies the presence of additional thyroid tissue in an ectopic location, apart from properly formed and located thyroid gland. This anomaly is predominantly clinically silent and is detected accidentally during thyroid imaging performed due to concomitant thyroid pathology. Figure 6 demonstrates accessory thyroid gland in a patient referred for diagnostics of nodular goitre.

Sometimes, when an accessory thyroid enlarges, it might present as a lingual or neck mass and only biopsy or scintigraphic examination might confirm the presence of thyroid tissue. Sometimes, patients might experience local compressive symptoms; however, it rarely leads to hormonal imbalance. Hyperthyroidism might be observed only in the case of autonomisation of thyroid tissue [51]. It should be remembered that the accessory gland may also undergo nodular changes (Figure 7) and even be the site of cancer origin, hence requiring careful sonographic assessment because small lesions may not alter the radioisotope uptake in thyroid scintiscans.

Thyroglossal duct cyst

Thyroglossal duct cysts comprise close to 7% of congenital neck masses [52]. They occur with a similar prevalence in males and females [53]. Thyroglossal duct is a structure that joins the tongue with descending thyroid anlage, and it undergoes involution after the thyroid has completed its descent [54]. However, in some cases it does not disappear and its lumen fills with fluid. In the wall of the cyst nests of thyroid cells might

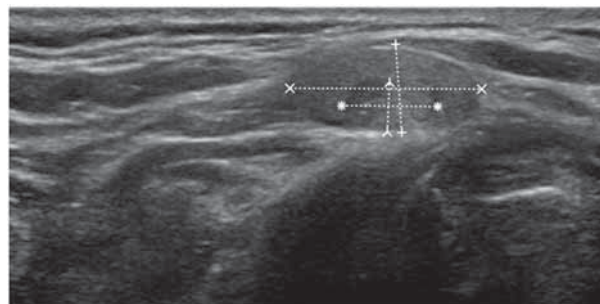


Figure 7. Accessory thyroid gland, with an isoechogenic lesion inside — ultrasound picture (transverse section).

be located, which in 1–2% of cases might undergo neoplastic transformation (Figure 8). The most common localization is midline in the infrahyoid region of the neck (60%). Less frequently, locations involve suprahyoid region as well as lingual, intralaryngeal, and retrosternal localizations [53, 55].

Clinically, thyroglossal duct cyst presents as painless, movable neck mass. Motility of the cyst along with the tongue movements is pathognomonic. The cyst is filled with serous fluid, and sonographically it presents as an anechogenic lesion with regular borders and posterior enhancement. In cases of infection, the cyst

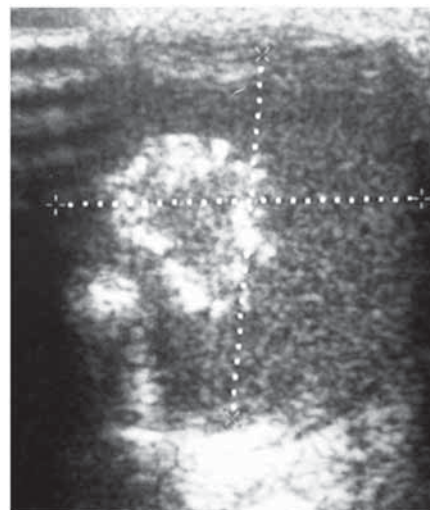


Figure 8. Ultrasound picture of papillary cancer, presenting as cluster of calcifications in a thyroglossal duct cyst.

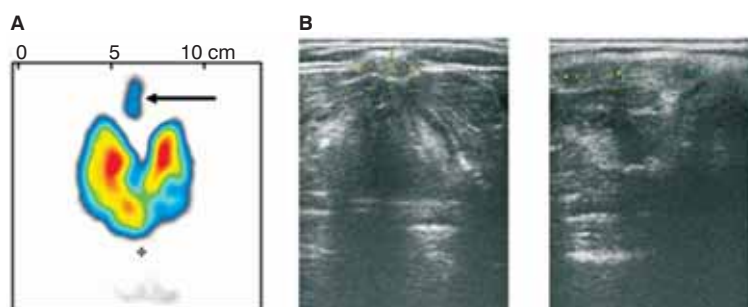


Figure 6. Accessory thyroid gland. **A.** Tc-99m scintiscan. **B.** Sonographic picture.

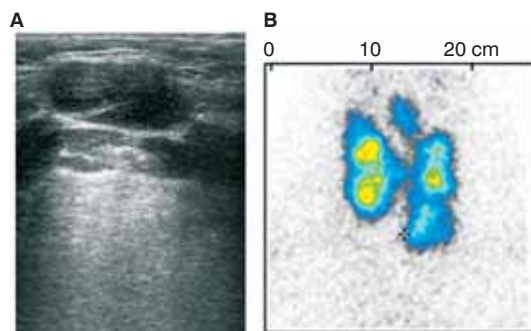


Figure 9. Thyroglossal duct cyst. **A.** Ultrasonographic picture. **B.** Tc-99m scintiscan, revealing the presence of functional thyroid tissue in the wall of the cyst.

fills with pus and the fluid inside might contain dense hyperechoic structures [56]. However, it is important to check the cyst for the presence of thyroid cells in the wall of the cyst, which is only possible by scintigraphic examination [57] (Figure 9).

Pyramidal lobe

Adequate classification of pyramidal lobe as an anatomical or pathological structure is still unclear. Due to the commonness of its incidence, it is regarded as one of the morphological variants of normal thyroid [58, 59]. In fact, the pyramidal lobe is a remnant of the caudal part of the thyroglossal duct and signifies the path of descent of thyroid primordium during embryogenesis [54]. Remnants of thyroglossal duct are reported to be found in 29 to 80% of cases [60]. Pyramidal lobe occurs with similar frequency in men and women, but in females it tends to be longer, with an average size of 29 mm in comparison to males, with pyramidal lobe 14 mm long on average [61]. In some cases it might arise above the lobes or even up to the hyoid bone [62]. It is connected with the thyroid at the left part of the isthmus; rarely it arises from the midline or left thyroid lobe [61]. Pyramidal lobe can sometimes be visualized by imaging. On thyroid scintiscan, it is detected in over half of the cases of toxic parenchymal goitre or in patients after thyroidectomy, but in only 10% of healthy subjects [63]. It is formed from normal thyroid tissue and hence might undergo nodular or neoplastic transformation [60]. Figure 10 represents a pyramidal lobe of significant size in a patient with right-sided TH, visible in both ultrasonographic and scintigraphic imaging.

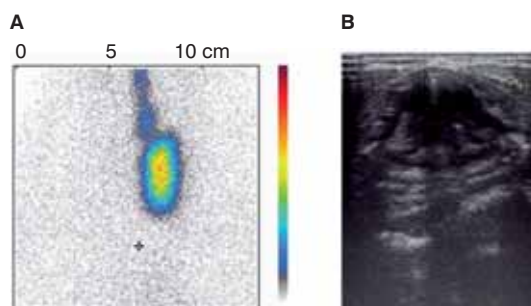


Figure 10. Pyramidal lobe in a patient with right lobe and isthmus agenesis. **A.** Tc-99m scintiscan. **B.** Sonographic picture (transverse section).

Conclusions

Awareness of both the advantages and limitations of sonographic and scintigraphic imaging is the key to successful interpretation of their results and reasonable recommendation of these procedures for patients with thyroid developmental anomalies of different age and clinical picture.

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