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Imaging study of thyroid dysfunction in patients (Review Article)

Dr. Maytham Sabeeh Ismail

Al-Muthanna University, College of Medicine, Al-Muthanna, Iraq

Abstract:

The thyroid imaging has evolved from early radionuclide thyroid scanning to the development of the advanced technique of SPECT, PET and fusion imaging. The advancement in cross-sectional techniques such as USG, CT and MRI has further improved the evaluation of intrathyroid pathologies. OCT has supplemented the imaging for better selection of patients for operation.

Ultrasound can be used as the primary imaging modality for guiding treatment of patients with CH, potentially decreasing radiation exposure and cost. Scintigraphy can be reserved for the few patients with equivocal ultrasound findings, such as hypoplastic thyroid gland.

The thyroid is a 2-inch-long, butterfly-shaped gland weighing less than 1 ounce. Located in the front of the neck below the larynx, or voice box, it has two lobes, one on each side of the windpipe. The thyroid gland makes two thyroid hormones, triiodothyronine (T3) and thyroxine (T4). T3 is made from T4 and is the more active hormone, directly affecting the tissues. Thyroid hormones affect metabolism, brain development, breathing, heart and nervous system functions, body temperature, muscle strength, skin dryness, menstrual cycles, weight, and cholesterol levels.

Hypothyroidism is the common clinical condition of thyroid hormone deficiency and, if left untreated, can lead to serious adverse health effects on multiple organ systems, with the cardiovascular system as the most robustly studied target. Overt primary hypothyroidism is defined as elevated thyroid-stimulating hormone (TSH) concentration in combination with free thyroxine (fT_4) concentration below the reference range.

Key words: patients, Hypothyroidism, Thyroid hormone.

Introduction:

The thyroid gland plays a critical role in regulating metabolic functions including heart rate and cardiac output, lipid metabolism, heat regulation, and skeletal growth. Recent advances in thyroid imaging have considerably improved the diagnosis, treatment, follow-up, and prognosis of high prevalence thyroid diseases such as thyroid nodule, goiter, thyroiditis, and thyroid cancer that affect the normal thyroid function. The relative roles of various imaging modalities in the evaluation of various thyroid diseases are discussed here. (1).

congenital hypothyroidism (CH) is defined as thyroid hormone deficiency present at birth. It can be subdivided into permanent and transient types. Permanent CH refers to persistent deficiency of thyroid hormone that requires lifelong treatment [1]. Transient CH refers to a temporary deficiency of thyroid hormone. The deficiency is present at birth, but recovery to normal thyroid hormone production usually occurs within the first few months or years of life.(12).

Thyroiditis is a general term for inflammation of the thyroid gland, and it can be associated with thyroid dysfunction. Thyroiditis is classified according to clinical symptoms (painful or

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painless), onset of symptoms (acute, subacute, chronic), and underlying etiology (autoimmunity, infection, drugs, radiation). Painful types of thyroiditis include subacute, suppurative, and radiation induced. Painless types include drug induced, fibrous (Riedel thyroiditis), Hashimoto thyroiditis (HT), postpartum, and silent.

The main cause of thyroid dysfunction in patient with proteinuria was usually thought as the urinary loss of thyroid hormone for its link to serum albumin. Reversely, it is reported that 10–30% patients with autoimmune thyroid disease could also involve with glomerular injury. And the most common associated glomerular disease was membranous nephropathy, too [3]. Considering that both membranous nephropathy and thyroid dysfunction could be caused by autoimmune disorder, whether there was any special significance of thyroid dysfunction in patients with pMN remains unclear.

Thyroid abnormalities affect a considerable portion of the population (1±3). However, the prevalence and the pattern of thyroid disorders depend on ethnic and geographical factors and especially on iodine intake (4±6). patients is generally considered to have iodine-intake (7, 8). patients (4) reported differences in incidences of hypo- and hyperthyroidism in southern and northern parts of patients. (6) reported prevalences of earlier diagnosed and unrecognized hypothyroidism of 3.6% and 1.8% respectively, in females over 70 in a small community in (attendance rate 99%). In Oslo the prevalence of earlier diagnosed hypothyroidism was 4.5% and unrecognised hypothyroidism was 1% in females over 70 years (attendance rate 71%)

Thyroid hormone is essential for the normal development of many human tissues and regulates the metabolism of virtually all cells and organs of the human body throughout life. Hypothyroidism, the clinical condition of thyroid hormone deficiency, is a common disorder in the general population. Overt hypothyroidism is defined by thyroid-stimulating hormone (TSH) levels above the upper limit of the reference range while levels of free thyroxine (fT_4) are below the lower limit of the reference range. The reference range is typically statistically defined by the 2.5th and 97.5th percentiles of the measured circulating thyroid hormone values in populations defined as healthy. In subclinical hypothyroidism, TSH levels are elevated but fT_4 levels are still within the reference range. Untreated hypothyroidism, especially overt hypothyroidism, can lead to serious adverse effects on multiple organ systems, both in the short and long term. Most adult patients with hypothyroidism have acquired hypothyroidism, which originates either in the thyroid (primary hypothyroidism) or in the pituitary or hypothalamus (central hypothyroidism). Hypothyroidism can also result from severe iodine deficiency because the synthesis of thyroid hormone requires the trace element iodine. Chronic autoimmune thyroid disease, Hashimoto thyroiditis, is the most common cause of primary hypothyroidism in iodine-replete areas. Because thyroid hormone is also essential for multiple aspects of normal development in childhood, most developed countries have established neonatal screening programmes to detect congenital hypothyroidism (prevalence of 1 in 500–3,000 newborns, depending on ethnicity)(1-3) as well as programmes to prevent severe iodine deficiency (for example, universal salt iodization).

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hypothyroidism is one of the most frequent endocrine diseases. It is usually detected by clinicians and often now looked for by other specialists such as gynecologists and cardiologists, who are more aware of its unwanted effects. Therefore, the purpose of these clinical practice guidelines has been to develop a systematic statement designed to assist health care professionals and patients in making decisions about appropriate health care for the management of hypothyroidism. (4).

We also sought to illustrate traditional concepts regarding overt hypothyroidism and to provide an updated view of the controversies and assertions in the field of subclinical hypothyroidism. The guidelines are divided into four areas, addressing diagnosis, screening, treatment and a special section for hypothyroidism in pregnancy. (4, 5).

In these guidelines, the topic of hypothyroidism in paediatrics was not included. The main questions posed were: How to make the diagnosis of hypothyroidism? Who should be screened for hypothyroidism? How should case finding be done? When should thyroid ultrasonography be performed? Which patients with subclinical hypothyroidism should be considered for treatment with thyroid hormones? How should patients with hypothyroidism be treated and monitored? When and how to screen hypothyroidism in pregnant women? How are hypothyroidism and subclinical hypothyroidism defined in pregnant women? What is the role of thyroid autoimmunity in fertility and pregnancy? When and how to treat hypothyroidism in pregnant women? Each question was answered according to the available literature and was concluded with a series of recommendations.(4, 6).

Hypothyroidism reflects decreased thyroid function and is one of the most common conditions seen in clinical practice. Hypothyroidism most often results from dysfunction of the thyroid itself (primary hypothyroidism) but can also occur as a result of defects along the hypothalamic/pituitary axis (less common) or from intake of lower-than-required doses of exogenous thyroid hormone in patients with primary hypothyroidism (more common). Overt hypothyroidism, defined as a serum thyrotropin level greater than the upper normal limit with a concomitant serum free thyroxine (T4) value less than the lower normal limit is less common than subclinical hypothyroidism, defined as a thyrotropin value greater than the upper normal limit in association with a serum free T4 value within the reference range. (7)

Overall, the presence of overt hypothyroidism and subclinical hypothyroidism increases with age, with both entities more common in women than in men.2 Estimates of the true prevalence of both overt hypothyroidism and subclinical hypothyroidism in the adult population vary by geographic location but in general range from approximately 0.2% to 1.0% for overt hypothyroidism to as high as approximately 10% for subclinical hypothyroidism, with the prevalence of subclinical hypothyroidism increasing with age, as is discussed later herein.(8)

Because thyroid hormone plays a fundamental role in the regulation of normal metabolism, reduced thyroid hormone levels as occur in hypothyroidism are associated with metabolic slowing and may lead to an array of signs and symptoms, including fatigue, reduced exercise capacity, muscle weakness, weight gain, bradycardia, cold intolerance, slowing of the normal reflex relaxation phase, constipation, depression, and menstrual irregularities (Table). In more

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severe cases, hypothyroidism can result in pleural or pericardial effusions, or even rhabdomyolysis.(9).

Etiology

Hypothyroidism is majorly divided into two categories, primary and secondary (central) hypothyroidism. Hypothyroidism is termed primary when the thyroid gland itself is not able to produce adequate amounts of thyroid hormone. The less common, secondary, or central hypothyroidism is labeled when the thyroid gland itself is normal, and the pathology is related to the pituitary gland or hypothalamus.

The most prevalent etiology of primary hypothyroidism is an iodine deficiency in iodine-deficient geographic areas worldwide. Autoimmune thyroid diseases are the leading causes of hypothyroidism in the United States and the iodine-sufficient regions. Hashimoto thyroiditis is the most common etiology in the United States, and it has a strong association with lymphoma. Etiology can be influenced locally by iodine fortification and the emergence of new iodine-deficient areas. (10, 11)

Mechanism (pathophysiology)

The most common cause of hypothyroidism is the inability of the thyroid gland to produce a sufficient amount of thyroid hormone; however, less commonly pituitary and hypothalamus may also result in thyroid dysfunction. The hypothalamus secretes thyrotropin-releasing hormone (TRH) that stimulates the pituitary gland to produce thyroid-stimulating hormone (TSH). Thyroid-stimulating hormone stimulates the thyroid gland to produce and secrete mainly T4 (approximately 100-125 nmol daily) and smaller quantities of T3. The half-life of T4 is 7-10 days, and eventually, T4 is converted to T3 peripherally by 5'-deiodination. Levels of T3 majorly and T4, to some extent, in turn, exert negative feedback on the production of TRH and TSH. Alteration in the structure and function of any of these organs or pathways can result in hypothyroidism.

The decline in the production of T4 results in an increase in the secretion of TSH by the pituitary gland, causing hypertrophy and hyperplasia of the thyroid parenchyma, thereby leading to increased T3 production. (10).

Diagnosis

Clinical Examination:

The most important determinant of the clinical picture is the severity of the underlying pathology, and the degree it alters biochemical and physiological reactions. Usually, symptoms start as mild manifestations, and patients are usually similar to euthyroid individuals. As time passes, symptoms will increase and patients will start to complain of the disease. A previous study has found that less than one third of hypothyroid patients were symptomatic. Increased weight gain is a classical presenting symptom that may suggest a diagnosis of hypothyroidism. However, some hypothyroid patients may lose body weight. When hypothyroidism is severe, several other manifestations develop including pericarditis, intestinal obstruction or pseudo-obstruction, congestive heart failure, pleural effusion, coagulopathies, depression, ataxia, apathy,

ISSN: 0975-3583, 0976-2833 VOL 11, ISSUE04, 2020

psychosis, seizures, and even coma. Dementia and other neurocognitive disorders can also develop in severe cases [12-14] .

Table (1) showed the Signs and Symptoms of Hypothyroidism (Follow to Matthew T. Drake)

TABLE. Signs and Symptoms of Hypothyroidism	
Signs	**Symptoms**
Eyebrow and hair thinning (C) Fatigue	
Skin coarsening (C)	**Weight gain**
Tendon relaxation phase slowing (C)	**Cold intolerance**
Facial/periorbital edema (O)	**Mental slowing**
Macroglossia (O)	**Muscle weakness**
Bradycardia (O)	**Reduced exercise capacity**
Pericardial effusion (R)	**Constipation**
Pleural effusion (R)	**Xeroderma**
Rhabdomyolysis (R)	**Depression** **Menstrual irregularities**
C ¼ common; O ¼ occasional; R ¼ rare.	

Ultrasonography

Ultrasonography is generally the first choice and the most sensitive imaging modality for diagnosing intrathyroid lesions. Because of its superficial location, the thyroid gland is ideally suited for high-frequency sonography (using 7-13 MHz transducer) which facilitates the detection of clinically non-palpable nodules of 2-3 mm size and allows a more accurate morphological characterization of the lesion. It is also used to determine the size and number of thyroid nodules, to assess the volume of thyroid tissue in cases of thyromegaly, and to differentiate thyroid masses from adjacent non-thyroid masses.

Addition of color and spectral Doppler imaging that determines the vascular pattern of thyroid diseases has been found to be very useful tool in screening the thyroid nodule for malignancy. A nodule with exclusively central vascular pattern is characterized as malignant, while a nodule

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with a predominantly perinodular pattern is generally benign. The gray scale ultrasonographic pattern associated with thyroid carcinoma includes a solid, hypoechoic mass that is taller than wide, has an irregular margin and microcalcifications but absent halo sign.

Pregnancy

Thyroid hormone requirements increase during pregnancy. In one prospective study, 45% of pregnant patients required a median increase of 27% in their thyroid hormone requirements 15 . These increases in levothyroxine dosing were required as early as the fifth week of pregnancy in some patients, which is before the first scheduled prenatal care visit. It is recommended that women on fixed doses of levothyroxine take nine doses each week (one extra dose on two days of the week), instead of the usual seven, as soon as pregnancy is confirmed 15 . Repeat thyroid function tests should be obtained five weeks after the increase in dosage. The increase in thyroid hormone requirement lasts throughout pregnancy (21).

Complication

Hypothyroidism can contribute to high cholesterol. If you have high cholesterol, you should get tested for hypothyroidism. Rarely, severe untreated hypothyroidism may lead to myxedema coma, an extreme form of hypothyroidism in which the body's functions slow to a life-threatening point. Myxedema coma requires immediate medical treatment.

Hypothyroidism is treated by replacing the hormones that own thyroid can no longer make. You will take levothyroxine , a thyroid hormone medicine identical to a hormone a healthy thyroid makes. Usually prescribed in pill form, this medicine is also available as a liquid and as a soft gel capsule. These newer formulas may help people with digestive problems to absorb the thyroid hormone. may recommend taking the medicine in the morning before eating.

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ISSN: 0975-3583, 0976-2833 VOL 11, ISSUE04, 2020

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