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Review Article

Review On Thyroid Disease

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ABSTRACT

Thyroid diseases are the most common diseases that occur when conditions affect the thyroid gland. The thyroid gland is an endocrine gland that is a butterfly-shaped and in front of the neck, wrapped around the windpipe (trachea), just below the larynx. The production of hormones thyroxine (T4) and triiodothyronine (T3) is its main purpose. Its function affects nearly every organ of the human body. The thyroid is controlled by the pituitary gland secretes a hormone known as thyroid-stimulating hormone (TSH). TSH then instructed the thyroid gland on how much hormone to make and release. But it also responds to signals from the hypothalamus, secretes thyrotropin-releasing hormone (TRH). TRH then causes the pituitary to release TSH, which signals the thyroid gland. The thyroid creates and releases hormones. When your thyroid makes either too little or too much of these important hormones, it's called a thyroid disease. When the thyroid gland doesn't make enough thyroid hormone to meet your body's needs, this is called hypothyroidism, and when the thyroid gland makes too much

INTRODUCTION

The thyroid gland is made up of two lobes (left and right), each roughly the size of a small hen's egg, that are located just behind the larynx in the horse's throatlatch area. Triiodothyronine (T3) and thyroxine (T4) are the two main hormones produced by the thyroid gland. Although these hormones have many different impacts on the body, their primary function in the adult body is to speed up metabolism. The pituitary gland, a little gland located at the base of the brain, controls the thyroid gland's production of these hormones. Thyrotropin, also known as thyroid stimulating hormone (TSH), is one of the regulating hormones secreted by the pituitary gland, along with a number of other hormones. TSH, as its name suggests, causes the thyroid gland to release T4 and T3. Not only that, but the hypothalamus, a distinct region of the brain situated directly above the pituitary gland, controls the amount of TSH secreted by the gland. Thyrotropin-releasing hormone (TRH), which is secreted by the

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hypothalamus, causes the pituitary gland to release TSH. A sophisticated feedback loop involving the thyroid gland, pituitary gland, and hypothalamus maintains blood levels of T4 and T3 within a relatively limited range. A decrease in T4/T3 causes the thyroid gland to secrete more T4 and T3, which in turn causes the synthesis of TRH, TSH, and TSH. On the other hand, increases in T4 and T3 have the opposite effect, suppressing the release of TSH and TRH and, consequently, T4 and T3 (1). Thyroid hormones are necessary for healthy growth and energy metabolism since they affect practically all nucleated cells (2). Although thyroid dysfunction is common, easily recognized, and treatable, it can have serious negative effects if left undetected or mistreated (3, 4). The persistence of severe thyroid dysfunction is astounding, given the growing awareness of thyroid disease and the accessibility of sensitive laboratory assays for thyroid hormone monitoring (5, 6). In reaction to some of these medications, the thyroid is able to adjust by raising blood TSH, continuing to generate a normal quantity of thyroid hormone despite disturbance. Based on indicators of thyroid hormone action, the effectiveness of this compensation can be evaluated in adults, but it is far more challenging to ascertain in utero, as well as in newborns and young children. The most well-studied thyroid hormone-dependent and thyroid hormone-vulnerable mechanism is brain development. In order for the brain and senses to grow properly, local thyroid hormone activation and the timing of triiodothyronine (T3) availability are essential. During this time, the most challenging agents to identify and measure are those that disrupt thyroid hormone signaling. Finding and assessing thyroid illness in its early stages is a major focus in clinical thyroid disease. The goal of recent studies evaluating the impacts of environmental chemicals that interfere with thyroid function has been to pinpoint the most modest and early effects (7). Serum TSH is the most sensitive test for detecting mild excess or deficiency of thyroid hormone in most outpatient clinical settings. As the most effective screening test for both hypothyroidism and hyperthyroidism, the sensitive thyroid stimulating hormone (TSH) or thyrotropin assay has emerged. Treatment options for people with Graves' disease include radioactive iodine (now the treatment of choice), antithyroid medications (often associated with relapses), and thyroidectomy. Levothyroxine replacement therapy is the usual treatment for clinical hypothyroidism, and it needs to be customized for each patient. The emphasis is placed on raising awareness of subclinical thyroid disease, which frequently goes undiagnosed, and on developing a care plan that includes patient education and involvement in addition to routine follow-up surveillance by a single doctor (8).

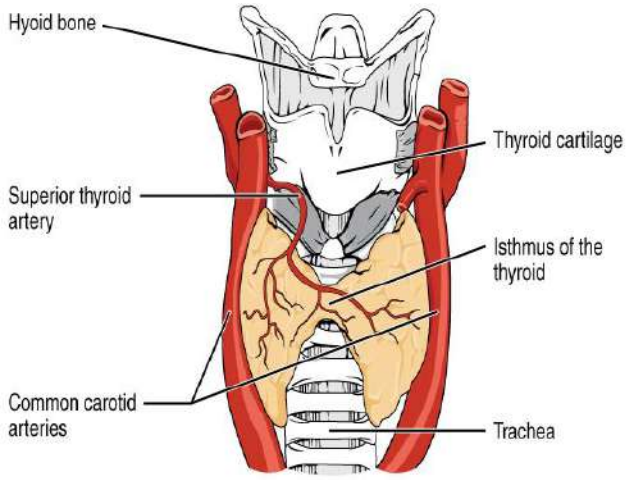

Figure 1: Thyroid Gland

Thyroid gland issues are incredibly common. According to estimates, up to half billion people worldwide may be susceptible to thyroid issues. The most frequent thyroid dysfunction is hypothyroidism, yet overactive thyroid glands are also conceivable. Another significant illness that affects up to 20% of people over 60 is subclinical hypothyroidism. The majority of people with

subclinical hypothyroidism, according to clinical endocrinologists, need treatment (9). Patients with this illness may not exhibit any symptoms at all, but some do show minor findings, such as altered lipid metabolism, cardiac, gastrointestinal, neuropsychiatric, and reproductive disorders, as well as a higher risk of goiter development. In order to detect subclinical hypothyroidism more frequently, patient awareness and physician education are required (10).

Hypothyroidism

Globally, environmental iodine shortages are the most frequent cause of thyroid diseases, including hypothyroidism (11). One of the most prevalent conditions that arises from thyroid gland disorders is primary hypothyroidism. Primary hypothyroidism is the diagnosis made in about 95% of cases of hypothyroidism (12). Reduced thyroid hormone and elevated TSH levels are linked to primary hypothyroidism. Treatment for hyperthyroidism, iodine insufficiency, thyroid cancer, or autoimmune diseases like Hashimoto's thyroiditis (HT) may cause these high levels. Hashimoto's thyroiditis is the most prevalent cause of hypothyroidism in the United States and most of the Western world, where iodine deficiency is uncommon (13). Weight gain, exhaustion, difficulty concentrating, sadness, generalized muscle soreness, irregular menstruation, and constipation are only a few of the non-specific symptoms and diverse clinical presentations of hypothyroidism. The repercussions of left-untreated or insufficiently treated hypothyroidism encompass infertility, cardiovascular disorders, as well as symptoms related to the nervous system and muscles (14, 15, 16). Hypothyroidism is a chronic disorder connected to a thyroid hormone lack of triiodothyronine (T3) and thyroxine (T4). Through a negative feedback process, the pituitary is stimulated to increase the synthesis of thyroid-stimulating hormone (TSH) when the thyroid is unable to produce T4 or T3 (17, 18). The thyroid gland mostly produces T4 and only produces trace amounts of T3. Less than 20% of T3 in peripheral tissue comes from the thyroid gland (19, 20). The most effective method of diagnosing hypothyroidism is to measure the blood levels of TSH and T4. Primary hypothyroidism is indicated by elevated TSH and decreased thyroid hormone concentrations. Serum TSH and T4 readings that are considered normal vary slightly between labs. Serum TSH within the range of 0.4 to 4.0 mU/L indicates a person without any indications of aberrant thyroid function (21). Hormone replacement therapy (HRT) is the best treatment for hypothyroidism. Normal TSH levels are the intended clinical outcome of the treatment. Brand names for the synthetic drug levothyroxine, which is frequently used to treat hypothyroidism, include Levothroid, Levoxyl, Synthroid, and Unithroid (22). For over 60 years, the "gold standard" for treating primary hypothyroidism has been thyroid hormone replacement therapy with levothyroxine, an exogenous form of T4 (23).

- **Subclinical hypothyroidism**

Subclinical hypothyroidism is generally accepted to be an early form of mild thyroid dysfunction. Subclinical hypothyroidism can range from mild (serum TSH values of 4·5–9 mU/L) to severe (TSH ≥10 mU/L), depending on the magnitude of the increase in serum TSH. Differences on the appropriate upper limit of the reference range for blood TSH obscure the definition and clinical importance of subclinical hypothyroidism. According to some researchers, the hormonal pattern linked to mild subclinical illness is a compensated state where normal circulating thyroid hormone concentrations are maintained by elevated blood TSH levels (24, 25). However, the

majority of researchers believe that elevated TSH levels indicate a genuine thyroid hormone deficit, even if it is moderate (26). Patients with subclinical illness (TSH ≤10 mU/L) account for at least 75% of cases. Hypothyroidism causes non-specific, non-sensitive symptoms. Because symptoms vary depending on the severity, length, and individual sensitivity to thyroid hormone shortage of the condition, it can be challenging to differentiate between people in a euthyroid state and those with subclinical hypothyroidism. The most common cause of thyroid hormone insufficiency in patients is multiple or recently developed symptoms (27). While subclinical hypothyroidism frequently has no symptoms, it can cause non-specific problems or symptoms that are comparable to overt hypothyroidism, such as weakness, constipation, lethargy, weight gain, and cold sensitivity (28). Subclinical hypothyroidism is often progressing, however reversible cases outnumber those than were previously believed, particularly when serum TSH levels are 10 mU/L or below (29). For most nonpregnant individuals, minimal (e.g., 25-75 μg) doses of levothyroxine are sufficient to restore normal serum thyrotropin levels since the degree of thyroid disease is mild. Serum thyrotropin levels should be measured six weeks after starting the medication and again every six weeks following any dose adjustments. It is advised to have annual thyroid function testing to confirm that serum thyrotropin remains within the target range once the thyrotropin target has been reached. Notably, it was discovered that an unacceptable percentage of patients receiving levothyroxine (15–38%) had thyrotropin levels below the reference range, indicating over replacement and underscoring the necessity of ongoing monitoring of serum thyrotropin levels (30, 31).

- **Hypothyroidism in pregnancy**

Sustaining a pregnancy and promoting the best possible development of the fetus depend on adequate thyroid hormone (32). The thyroid hormone produced by the mother is the only hormone that the fetus needs throughout the first half of pregnancy. Thyroid conditions are widespread in women of childbearing age and are often seen in prenatal clinics, with decreased thyroid function being one such condition. All endocrine and obstetric societies advise treating overt hypothyroidism because it is widely known to increase the risk of unfavorable pregnancy and child outcomes. Adequate iodine intake is crucial for the generation of thyroid hormone in women who are pregnant or who are of reproductive age. There is growing evidence that thyroid autoimmunity and even borderline thyroid function are linked to unfavorable outcomes, which suggests that a universal thyroid screening program for pregnant women should be explored (33). Untreated overt hypothyroidism during pregnancy may raise the risk of low birth weight, spontaneous abortion, preeclampsia, anemia, postpartum hemorrhage, cardiac ventricular dysfunction, and, perhaps, aberrant brain development in mothers. Thyroid hormone replacement medication appears to be able to counteract the negative impact of mild, asymptomatic, untreated maternal hypothyroidism during pregnancy on the cognitive function of the fetus. This finding comes from a population-based investigation. It's unclear if therapy can prevent this issue, but mildly elevated serum TSH levels during pregnancy may also raise the chance of fetal death. Thyroid antibodies appear to be a risk factor for spontaneous abortion in the majority of these women, regardless of thyroid hormone and TSH levels (34).

- **Congenital hypothyroidism**

Congenital hypothyroidism (CH) is the most prevalent congenital endocrine condition in childhood and one of the most common preventable causes of mental impairment (35). Upon initial examination, the most prevalent signs are chilly or mottled skin, macroglossia, and umbilical hernia (36). Most developed nations regularly conduct universal newborn screening, which is the most important method for identifying congenital hypothyroidism. Regional variations exist in specific screening procedures, but generally speaking, the process starts in the first few days of birth with the collection of a heel-prick blood sample on a filter paper card, which is then sent to a central laboratory for examination. TSH is detected in the blood in the majority of screening programs worldwide; in some, T4 is also taken on a regular basis or as a reflex if the TSH is elevated (37). As soon as the diagnosis is made, both term and preterm neonates with low T4 and excessive TSH should begin taking L-thyroxine. The goal of the initial L-thyroxine dose should be to normalize the T4 level as soon as possible, which is 10-15µg/kg/day. The maximal dose of 15µg/kg/day should be begun in newborns with severe hypothyroidism (extremely low T4, very high TSH, and lack of distal femoral and proximal tibial epiphyses on knee radiographs) (38).

- **Hyperthyroidism**

Thyrotoxicosis, another name for clinical hyperthyroidism, is a condition brought on by an excess of thyroid hormone and can be brought on by a variety of illnesses. Prognosis and treatment are influenced by the etiologic diagnosis. According to estimates from community-based studies, the prevalence of hyperthyroidism is 0.2 percent in males and 2 percent in females (39). The result of increased thyroid hormone activation is hyperthyroidism. There are some of the causes of hyperthyroidism Graves' disease (toxic diffuse goiter), toxic multinodular goiter (Plummer's disease), toxic adenoma, painful subacute thyroiditis, silent thyroiditis, including lymphocytic and postpartum variations, iodine-induced hyperthyroidism (for example, related to amiodarone therapy), excessive pituitary TSH, or trophoblastic disease (40). Up to 15% of occurrences of hyperthyroidism involve individuals who are older than 60 (41). Approximately 60 to 80 percent of instances of hyperthyroidism are caused by Graves' disease. (42). This autoimmune condition is brought on by an antibody that is active against the receptor for thyroid-stimulating hormone (TSH), which causes the gland to produce and secrete too much thyroid hormone. It may run in families and be linked to other autoimmune conditions. Approximately 50% of patients with Graves' disease also have infiltrative ophthalmopathy (43). Possible signs and symptoms associated with the various causes of hyperthyroidism are weight loss or gain, heat intolerance or increased sweating, menstrual disturbance (decreased flow), Impaired fertility, Alterations in appetite, Frequent bowel movements or diarrhea, Fatigue and muscle weakness Thyroid enlargement (depending on the cause), Nervousness and irritability Mental disturbances, Sleep disturbances (including insomnia), Palpitations and tachycardia Changes in vision, photophobia, eye irritation, diplopia, or exophthalmos, Tremor, dependent lower-extremity edema, Sudden paralysis, Exertional intolerance and dyspnea Pretibial myxedema (in patients with Graves' disease) Not all of these symptoms have to be present in a patient with hyperthyroidism (44). The most sensitive and specific test for thyroid problems is serum TSH, which should be obtained first (45). Antithyroid medications primarily function by obstructing

iodine's regasification, which lowers thyroid hormone levels. In the US, the two agents that are accessible are propylthiouracil (PTU) and methimazole (Tapazole). The length of treatment affects the rate of remission; nonetheless, after two years of therapy, rates of 60% have been seen. For the majority of individuals in the US with toxic nodular goiter and Graves' disease, radioactive iodine is the preferred course of treatment. It is safe, affordable, very efficient, and simple to use. Because of the possible risk of thyroid cancer, leukemia, or genetic harm in future offspring, using radioactive iodine in women who are fertile has been met with resistance. These worries have not been confirmed by long-term patient monitoring (46, 47). Although the use of radioactive iodine to treat childhood hyperthyroidism is still debatable, this population is starting to accept it more (48). Radioactive iodine has gradually replaced surgery in the treatment of hyperthyroidism, while other experts believe it is still underutilized and may still be warranted in certain cases. Most frequently, a subtotal thyroidectomy is carried out. With this operation, the frequency of hypothyroidism is reduced to 25% while some thyroid tissue is preserved; however, 8% of patients experience permanent or recurrent hyperthyroidism (49).

- **Subclinical hyperthyroidism**

Regardless of the presence or absence of symptoms, subclinical hyperthyroidism, or SCHyper, is a biochemical diagnosis defined by normal serum concentrations of triiodothyronine (T3) and thyroxine (T4) in the context of decreasing serum TSH levels. On the other hand, overt hyperthyroidism is distinguished by a reduction in TSH levels and an increase in serum T3 and/or T4. According to the degree of TSH drop, a proposed grading system separates moderate from severe SCHyper (mild SCHyper, TSH 0.1-0.4 mIU/L; severe SCHyper, TSH <0.1 mIU/L) (50). The clinical manifestation of SCHyper can range from minor hyperthyroidism symptoms such arrhythmias, heat intolerance, sleeplessness, increased hunger, diarrhea, weight loss, hair loss, diaphoresis, irregular menses, and hand tremors to the complete lack of symptoms. When SCHyper is found, the first serologic workup include doing the thyroid function tests again. Serum TSH and the concentrations of free and/or total T4 and T3 should be measured as part of this. An autoimmune thyroid etiology may be considered if the biochemical abnormalities are confirmed. Serum thyroid antibody titers, including TRAb, TSH-binding inhibitory immunoglobulin (TBII), thyroid-stimulating immunoglobulin, and thyroglobulin antibody, may be collected. Test positive results for serum thyroid stimulating immunoglobulin, TBII, and TRAb would indicate a diagnosis of Graves' disease; these tests are widely accessible, highly specific, and economical (51, 52).

- **Hyperthyroidism in pregnancy**

Pregnant women with hyperthyroidism should work closely with a clinical endocrinologist to manage this condition since it poses unique challenges. Because radioactive iodine enters the placenta, using it while pregnant is not advised. Propylthiouracil is unquestionably recommended over methimazole as the antithyroid medication of choice for treating hyperthyroidism during pregnancy. Antithyroid medications can have a negative impact on the fetus if taken excessively because they also cross the placenta. To keep the mother's thyroid function in the upper range of normal, the lowest dose of an antithyroid medication should be utilized. Due to the fact that pregnancy itself improves Graves' disease the amount of antithyroid medication needed typically goes down as the pregnancy goes on. Antithyroid

medications can frequently be stopped prior to delivery. If surgery is required, it is best performed in the second trimester of pregnancy. When Graves' illness is present, the patient's active involvement in therapy is essential to the pregnancy's healthy result. The patient's comprehension of the disease's risk, its pathophysiologic components, and the therapeutic methods is crucial. Patient education will improve awareness of changes that might require treatment adjustments as well as adherence to prescribed therapy. Given this context, the patient ought to be more aware of potential issues and notify her endocrinologist of them. Additionally, during the postpartum phase, the patient should be made aware of any changes that might occur in her health or the health of her unborn child (53). She should be encouraged to let the pediatrician know about her thyroid condition and the chance that the child may develop neonatal hyperthyroidism or hypothyroidism. Testing of the baby's thyroid function is required at birth. Additionally, the patient should be informed that there is a chance of a postpartum hyperthyroidism recurrence. This discovery may be connected to postpartum thyroiditis or Graves' disease. Should the patient experience overt hyperthyroidism following delivery as a result of Grave's disease, they may be presented with the option to either restart antithyroid medication therapy or receive radioactive iodine. Maternal TSI (TRAb) measurement may be helpful for evaluating possible fetal danger; the endocrinologist can order this investigation based on clinical discretion (54).

- **Congenital hyperthyroidism**

The more prevalent kind of neonatal hyperthyroidism, known as transient neonatal hyperthyroidism (w1:25,000–50,000), affects 1% to 2% of children born to women who have either active or dormant thyroid disease (55).A favorable prognosis depends on an early diagnosis and the start of treatment. A number of conditions could be present, such as tachycardia (heart rate > 160/min), arrhythmias, cardiac failure, sweating, goiter, prominent eyes, narrow sutures, craniosynostosis, advanced bone age, vomiting, hyperphagia, icterus, diarrhea, poor weight gain, hepatosplenomegaly, lymphadenopathy, and thrombocytopenia. In comparison to the normal range for the same gestational age, serum levels of T4/FT4, T3/FT3, and thyrotropin are greater and lower, respectively (56)

CONCLUSION

Thyroid disease occurs when the thyroid fails to function properly, There are two main types of thyroid disease hypothyroidism and hyperthyroidism. Hypothyroidism a condition when high TSH level indicates that the thyroid gland is not making enough thyroid hormone. Hyperthyroidism is a low TSH level usually indicates that the thyroid is producing too much thyroid hormone. Thyroid dysfunction can affects on metabolism, body weight, heart rate, menstruation, nervous system, fertility, growth and development. Thyroid diseases in pregnancy can affect the health of the mother as well as the child before and after delivery.

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