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# THYROID DISORDERS AND PREGNANCY

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**Abstract**

**Importance**. Thyroid hormones from the mother are essential for normal fetal development, like brain growth. There are metabolic needs in pregnancy, producing physiological adjustments inside the thyroid that bring about increased manufacturing of hormones. Placental iodothyronine deiodinases have a careful control of the passage of these hormones to the fetus. Both higher and lower levels of thyroid hormone exert teratogenic effects. For this reason, right detection and management of thyroid issues in pregnancy are vital to save you unfavorable outcomes in the maternal, fetal and new child [1].

**The Objective** is to highlight the importance of thyroid disorders during pregnancy and its management.

**The main goal** of prenatal screening and diagnosis of thyroid related pathophysiology changes that occur during pregnancy.

**Keywords**: Thyroid disorders, Pregnancy, Screening, Diagnosis, Management, Hypothyroidism, Hyperthyroidism, Maternal health, Fetal development, Levothyroxine, Antithyroid drugs (MMI, PTU), Iodine deficiency, Neurodevelopment, Preconception counseling, Pregnancy outcomes, TSH (Thyroid Stimulating Hormone), FT4 (Free T4), FT3 (Free T3), Thyroid function, Autoimmune thyroid disease, Graves' disease, Subclinical hypothyroidism, Pregnancy loss, Gestational diabetes mellitus (GDM), Thyroid hormones, TPO antibodies, Thyroid-binding proteins.

## Introduction

Diseases of the thyroid are the second one maximum universal endocrine disorder in pregnancy. Pregnancy causes anatomical and physiological alterations to the thyroid gland, so it is difficult to distinguish between pregnancy with thyroid disease and normal symptoms of pregnancy. Thyroid hormones are instrumental in the physical and cerebral development of the fetus, and their quantification during pregnancy must be specially interpreted due to altered reference ranges. Thorough tracking and well timed treatment of thyroid disease are required to decrease risks for each mom and infant [2]. Powerful management of thyroid dysfunctions throughout being pregnant calls for early detection and timely treatment of any pre-present conditions [4]. The accurate diagnosis, monitoring, and control of thyroid disorders in pregnancy and postpartum



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intervals are essential in achieving optimum results for both mother and the unborn fetus. Given the sophisticated physiologic alterations that mark pregnancy and the tremendous role thyroid hormones have in fetal improvement, specifically within the neurodevelopmental levels, the prognosis of thyroid disorder and powerful control thereof need to accept attention early and intensively. Health care providers must be aware of the unique presentation and challenges of thyroid disorders in pregnancy to provide personalized and effective management. Continued research and updated clinical guidelines will also enable the safe and effective management of thyroid conditions in maternal care [7].

**Thyroid and its role.** In pregnancy, human chorionic gonadotropin (hCG) mildly stimulates the thyroid gland to produce more thyroid hormones. This stimulation of thyroid hormone production consequently inhibits the secretion of thyroid stimulating hormone (TSH) from the pituitary gland via negative feedback. The outcome is, thus, in the early period of pregnancy when hCG is at its highest level, serum TSH levels are usually decreased in comparison to non-pregnant females [2]. A pregnant woman's body undergoes physiological changes to ensure adequate thyroid hormone supply for the developing fetus, particularly for crucial brain development. While the baby's thyroid gland becomes operational around 12 weeks gestation, it doesn't reach full functionality until the 18-20 week mark. The surge in estrogen during pregnancy leads to an increase in thyroid-binding proteins, resulting in higher overall T4 and T3 levels, although the concentration of free hormones experiences a slight decline before stabilizing around 20 weeks. Additionally, hCG, a hormone produced during pregnancy, modestly activates the thyroid, contributing to a reduction in TSH levels. These alterations require the establishment of hormone reference ranges tailored to each trimester, and iodine demand is significantly elevated during pregnancy, rising by approximately half [3]. Both thyrotoxicosis and hypothyroidism can lead to negative pregnancy outcomes, and there are concerns about how overt maternal thyroid disorders may impact fetal development [6]. Maternal hypothyroidism is identified when thyroid-stimulating hormone (TSH) surpasses established thresholds for each trimester, or when no trimester-specific benchmarks exist, indicating a TSH level greater than 4 milliunits per liter. The significance of a mother's thyroid hormones in shaping fetal growth and development, particularly in light of maternal thyroid disorders, cannot be overstated. Thyroid hormones are essential for early fetal development, particularly in the period preceding the fetal thyroid gland's maturation and its capacity to generate its own thyroid hormones during the later stages of pregnancy. The significance of a mother's thyroid health persists even after the fetus begins its own hormone synthesis, as evidenced by research on newborns with thyroid hormone deficiencies.

Human Chorionic Gonadotropin (HCG) promotes the thyroid gland's production of hormones essential for fetal development in early pregnancy, ensuring sufficient levels are available. Yet, the placenta concurrently releases DIO3, an enzyme that breaks down thyroid hormones, leading to elevated maternal TSH levels and a heightened need for levothyroxine in women with pre-existing thyroid conditions. This intricate interplay of hormonal signals highlights the precise coordination required to support healthy fetal development.

In terms of consequences, severe maternal hypothyroidism, particularly overt cases, is associated with significant developmental problems such as cretinism, necessitating treatment during



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pregnancy. However, the impact of milder hypothyroidism, including subclinical hypothyroidism and isolated low T4 levels, remains uncertain. Likewise, while overt hyperthyroidism, often stemming from disorders like Graves' disease, requires treatment, the effects of mild or subclinical hyperthyroidism are not fully understood. Research suggests a U-shaped connection between a mother's thyroid health during pregnancy and the results of the pregnancy, similar to other factors influencing prenatal development, where both insufficient and excessive thyroid hormone levels can have negative consequences [14].

**Clinical Interpretation.** Low TSH (<0.4 mIU/L) may indicate hyperthyroidism. High TSH (>4.5 mIU/L) should prompt FT4 testing. Low FT4 suggests clinical hypothyroidism, often autoimmune if TPO antibodies are present. Normal FT4 with high TSH indicates subclinical hypothyroidism. Elevated FT4 may point to a TSH-secreting tumor, requiring endocrinology referral. Rare causes include adrenal insufficiency, medications, non-thyroidal illness, or thyroid hormone resistance syndrome[18].

**Treatment hypothyroidism.** Thyroid function tests during pregnancy should be interpreted based on gestational age. Overt hypothyroidism must always be treated promptly. Women with pre-existing hypothyroidism typically need increased levothyroxine (LT4) doses during pregnancy and require regular TSH monitoring. While treatment for mild gestational thyroid dysfunction is debated, LT4 is generally recommended for subclinical hypothyroidism, especially if TSH >10 mIU/L or TPO antibodies are present. Untreated women should be closely followed for progression. More research is needed on early LT4 treatment and optimal screening strategies [19]. Initiating levothyroxine replacement therapy early in pregnancy for subclinical hypothyroidism may help reduce the risk of pregnancy loss [23].

**Hyperthyroidism.** Affecting between 0.2% and 1.3% of individuals globally, hyperthyroidism is characterized by low thyroid-stimulating hormone (TSH) levels coupled with elevated free thyroxine (FT4) or free triiodothyronine (FT3) concentrations. Following diagnosis, determining the underlying cause involves utilizing antibody tests, ultrasound imaging, or scintigraphy. Graves' disease and toxic nodular goitre are the most prevalent contributors to this condition, accounting for a combined 86%, while other less common causes include thyroiditis or specific medications. The primary treatment for Graves' disease involves antithyroid medications, though approximately half of patients experience a recurrence within 12 to 18 months. Prolonged treatment can significantly decrease the likelihood of this relapse. Toxic nodular goiter is typically managed through radioiodine therapy or surgical intervention. In cases of mild, self-limiting thyrotoxicosis, the condition often subsides on its own [16]. Women with a previous diagnosis of hyperthyroidism or a previous history of the condition are at high risk for the development of complications during pregnancy and also the perinatal period and should thus be monitored closely during the gestational period [17,28].

**Grave's disease.** Graves' disease (GD), an autoimmune disorder stemming from immune system dysfunction, is characterized by the generation of antibodies that target the thyroid-stimulating



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hormone receptor (TRAb). While GD during pregnancy often manifests as hyperthyroidism, it can be differentiated from gestational transient hyperthyroidism, the most common cause of hyperthyroidism in pregnant women, through careful analysis of patient history and clinical observations. Distinctive hallmarks include a familial or individual background of autoimmune thyroid diseases and certain physical indicators suggestive of Graves' disease, such as a noticeable thyroid enlargement (goitre) accompanied by a whooshing sound (bruit) or eye-related problems (ophthalmopathy).

Diagnosing Graves' disease relies heavily on TRAb testing, as the presence of these specific antibodies strongly indicates an autoimmune thyroid disorder. If TRAb levels are undetectable but a physical exam suggests a thyroid nodule, thyroid Doppler ultrasound proves valuable. This imaging method can pinpoint the blood flow patterns within the nodule, aiding in the identification of a hyperactive nodule that might be responsible for hyperthyroidism. However, pregnant individuals should not undergo thyroid scintigraphy, a diagnostic technique utilizing radioactive substances, due to the potential harm radiation could inflict on the developing fetus [22].

**Treatment.** For many years, antithyroid medications like Methimazole and Propylthiouracil have been employed to manage hyperthyroidism during pregnancy. However, worries regarding their potential to induce birth defects have persisted since the 1970s. Newer studies have shed light on this issue, suggesting a possible association between MMI and PTU and congenital abnormalities. Uncertainties remain, particularly concerning PTU, and the review delves into the evidence surrounding these potential dangers, such as their impact during pregnancy and the role maternal thyroid levels may play. Although MMI's link to birth defects is well-established, more investigation is required to comprehensively evaluate PTU's risks. The review emphasizes the need for additional research to determine optimal treatment timing and explore alternative therapies, ultimately aiming to improve safety for both mothers and infants [15].

The use of antithyroid drugs (ATDs) in early pregnancy may be linked to birth defects, particularly with methimazole (MMI), while more research is needed to determine the teratogenic risk of propylthiouracil (PTU). Key factors such as the biological gradient and maternal thyroid function are discussed. The timing and type of treatment play a crucial role in outcomes, and further studies are required to assess the risks in large cohorts and explore alternative or new treatments for managing thyroid issues during pregnancy [24].

**Iodine deficiency.** Iodine deficiency is the most prevalent etiology of hypothyroidism worldwide. During pregnancy, iodine needs are increased by 50% to match the physiological rise in thyroid hormone, the augmented urinary excretion of iodine, and the transfer of iodine to the fetus for thyroid hormone production[10]. Iodine deficiency in pregnancy exacerbates the danger of miscarriage, premature labor, stillbirth, goiter in mother and fetus, neonatal cretinism, impaired intellectual function, deaf mutism, poor school performance, reduced intellectual capacity, and dwarfism [7]. The effect of mild to moderate iodine deficiency on child neurodevelopment remains unclear [11]. ATA, Endocrine Society, US Teratology Society, American Academy of Pediatrics, and ETA now recommend that women contemplating pregnancy, pregnant, or lactating receive a



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daily supplement orally of 150 µg of iodine. The beneficial effects of iodine supplementation are more pronounced in areas with moderate iodine deficiency as opposed to mild deficiency [12].

**Outcome.** Thyroid disorders in pregnant women and their potential effects on the developing fetus are a significant area of study. Due to the fetal thyroid's immaturity during the early stages of pregnancy, the mother's thyroid hormones are crucial for fetal growth and development in the first trimester. Interestingly, a lack of association was found between anti-TPO antibodies and negative pregnancy outcomes. On average, spontaneous abortions occurred at 8 weeks of gestation. Notably, women who experienced miscarriages exhibited elevated average TSH levels (2.55 mU/L), a finding that came close to statistical significance compared to women without miscarriages ($p = 0.054$). These results underscore the crucial role of early TSH testing and prompt treatment in mitigating pregnancy complications and enhancing overall pregnancy success [13,27]. Pregnant women diagnosed with hypothyroidism require pre-pregnancy counseling to discuss treatment objectives and potential effects on both mother and baby. Levothyroxine is the preferred medication, and a dosage adjustment of 20-30% is typically recommended upon pregnancy confirmation to ensure thyroid stimulating hormone (TSH) levels stay within a healthy range, from the lower limit of normal to 2.5 mU/L. Pregnant women should avoid desiccated thyroid and T3 medications as they may harm the developing fetal brain; T4, the key thyroid hormone for brain growth, is the preferred choice. Thyroid function needs to be regularly checked, at least monthly, with more frequent monitoring advised after dosage adjustments and potentially less frequent checks once the second trimester begins. For women experiencing subclinical hypothyroidism, characterized by elevated TSH levels (greater than 4 mU/L) but normal T4, levothyroxine supplementation is recommended when thyroid peroxidase antibodies are detected in the blood. The absence of these antibodies may warrant treatment initiation at higher TSH levels, exceeding 10 mU/L, although individual clinicians might opt for earlier intervention at lower TSH thresholds [8].

**Risk.** Elevated levels of free T3 and a higher ratio of free T3 to free T4 during the later stages of pregnancy are strongly connected to a greater likelihood of developing gestational diabetes and experiencing negative pregnancy results. Even when thyroid function is within a healthy range, these indicators are linked to a less desirable metabolic state both during pregnancy and in the initial postpartum period. On the other hand, lower free T4 levels detected in the early stages of pregnancy seem to be associated with a decreased chance of developing gestational diabetes. These findings highlight the significance of thyroid hormones in managing blood sugar levels and maintaining maternal well-being during pregnancy, stressing the importance of closely observing thyroid function throughout this period [21,26].

**Awareness.** An underactive thyroid can manifest in various ways, including fatigue, sensitivity to cold, changes in hair texture, digestive issues, and cardiovascular complications such as shortness of breath, fatigue, and ankle swelling. Untreated hypothyroidism can elevate blood pressure and cholesterol, raising the risk of heart disease. Individuals prescribed thyroid hormone, commonly levothyroxine, should administer it daily on an empty stomach, an hour before their first meal, and



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undergo blood tests every 4 to 6 weeks to adjust the dosage accordingly. Most individuals diagnosed with permanent hypothyroidism require continuous medical treatment throughout their lives.

Overactive thyroid function manifests through symptoms such as exhaustion, unintended weight reduction coupled with heightened appetite, rapid heartbeat, excessive sweating, nervousness, and trembling. Physical examination may reveal an enlarged or lumpy thyroid gland. For expectant mothers diagnosed with hyperthyroidism, pre-pregnancy counseling is crucial to achieve a normal thyroid state before conception, thus reducing potential harm to the fetus [25,28].

**Conclusion:**

Older maternal age is linked to more pregnancy risk factors, increasing the need for hospital monitoring. This may explain why fewer TSH evaluations are seen in pregnant women with morbid obesity or diabetes, as they are likely being monitored in hospitals[5].

Hyperthyroidism due to Grave's disease is seen in approximately 0.2% to 0.4% of pregnant women, the most common cause, then gestational hyperthyroidism and toxic adenomas. Detection of postpartum thyroid illness is critical due to their high frequency and profound effect on maternal and neonatal health [9]. Maternal pre-eclampsia (PE) and gestational diabetes mellitus (GDM) are 2 major disorders associated with maternal and fetal mortality and morbidity. The relationship of thyroid function during pregnancy with the subsequent risk of PE and GDM remains controversial and varies according to iodine intake in the regions studied and thyroid autoantibody status [20,25].

The well-being of both mother and baby during pregnancy hinges on effectively managing thyroid disorders. Accurate identification and treatment of thyroid conditions are crucial for ensuring a healthy pregnancy. Both overt and hidden thyroid dysfunction, whether hypothyroidism or hyperthyroidism, can lead to serious consequences such as fetal growth retardation, pregnancy loss, and childbirth complications. The intricate relationship between thyroid hormones and fetal development demands careful monitoring, particularly in the early stages of pregnancy. Early identification and appropriate medical interventions, such as levothyroxine for hypothyroidism and antithyroid medications for hyperthyroidism, are crucial for safeguarding both the mother and the developing fetus. Additionally, iodine supplementation is essential to prevent deficiencies that can lead to severe developmental and cognitive impairments. Ongoing research is necessary to continually improve treatment strategies and enhance the well-being of pregnant women with thyroid disorders. Healthcare providers need to take a proactive approach, tailor treatment to each patient's unique needs, and keep up-to-date on the newest medical recommendations to effectively manage thyroid conditions in pregnant women.

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