

Thyroid Dysfunction during Pregnancy and Its Effects on Maternal and Fetal Outcomes

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Introduction

Thyroid function plays a vital role in the physiological regulation of pregnancy and fetal development. Pregnancy imposes significant alterations on the maternal thyroid gland due to increased metabolic demands, elevated estrogen levels and placental production of thyroid-stimulating hormones. These changes can unmask or exacerbate underlying thyroid disorders, leading to either hypothyroidism or hyperthyroidism during pregnancy. Both overt and subclinical thyroid dysfunctions have been linked to adverse maternal and fetal outcomes. Hypothyroidism in pregnancy, particularly when untreated, is associated with increased risks of miscarriage, preeclampsia, anemia, placental abruption and preterm delivery. Likewise, maternal hyperthyroidism can lead to complications such as intrauterine growth restriction, fetal tachycardia, preterm labor and maternal heart failure. The thyroid hormone is essential for fetal neurodevelopment, especially in the first trimester, as the fetal thyroid gland becomes functional only after 12 weeks of gestation. Consequently, maternal hypothyroidism during early pregnancy may result in impaired cognitive development in the child. Timely identification and management of thyroid disorders during pregnancy are therefore crucial. Despite clear guidelines, screening practices vary globally and many cases remain undiagnosed, particularly in resource-limited settings. Moreover, the interpretation of thyroid function tests during pregnancy requires gestational age-specific reference ranges, which are not always readily available. This article aims to explore the impact of thyroid dysfunction during pregnancy on maternal and fetal health, emphasizing the importance of early diagnosis, optimal treatment and careful monitoring throughout gestation [1].

Description

Maternal hypothyroidism during pregnancy can be categorized as overt or subclinical, each with varying degrees of impact. Overt hypothyroidism, defined by elevated TSH and low free T4 levels, has been consistently associated with increased risk of obstetric complications such as miscarriage, gestational hypertension, placental abruption and stillbirth. It also negatively affects fetal growth and neurological development, especially when untreated in early pregnancy. Subclinical hypothyroidism, characterized by elevated TSH with normal T4 levels, is more common and its clinical significance remains a topic of debate. However, studies have suggested that it may still contribute to preterm delivery, lower birth weight and impaired intellectual outcomes in children. Thyroid Peroxidase Antibody (TPOAb) positivity in euthyroid women further increases the risk of progression to hypothyroidism and pregnancy loss. The physiological changes of pregnancy, including increased renal iodine clearance and elevated thyroid-binding globulin, make pregnant women particularly vulnerable to iodine deficiency and thyroid

insufficiency. The American Thyroid Association recommends levothyroxine therapy for women with overt hypothyroidism and consideration of treatment for subclinical cases with TPOAb positivity or high TSH levels. Monitoring thyroid function every 4-6 weeks throughout pregnancy is essential to adjust therapy and maintain optimal maternal and fetal health. Failure to adequately treat hypothyroidism can result in neurodevelopmental delays in the offspring, which underscores the need for awareness, especially in areas with limited access to prenatal care. Universal screening remains a controversial issue, though targeted screening of high-risk women is widely practiced. Ultimately, early detection and management of hypothyroidism can dramatically improve maternal and perinatal outcomes [2].

Hyperthyroidism during pregnancy, although less common, poses unique challenges for maternal and fetal health. Most cases are due to Graves' disease, an autoimmune disorder marked by the presence of Thyroid-Stimulating Immunoglobulins (TSIs). These antibodies can cross the placenta and stimulate the fetal thyroid, leading to fetal or neonatal thyrotoxicosis. Clinically, hyperthyroidism in pregnant women can manifest as weight loss, palpitations, heat intolerance, anxiety and goiter. In severe cases, it may precipitate thyroid storm, a life-threatening emergency. Maternal complications include preeclampsia, heart failure, placental abruption and preterm birth. Fetal consequences of uncontrolled maternal hyperthyroidism include low birth weight, fetal growth restriction and even stillbirth. Antithyroid Drugs (ATDs), such as Propylthiouracil (PTU) and methimazole, remain the cornerstone of treatment. PTU is preferred during the first trimester due to the teratogenic risk of methimazole, after which methimazole is often reintroduced to avoid PTU-related hepatotoxicity. However, both drugs cross the placenta and can affect fetal thyroid function, necessitating careful dose titration and close monitoring. The goal is to maintain maternal free T4 in the high-normal range to avoid both maternal symptoms and fetal hypothyroidism. Radioactive iodine therapy is contraindicated in pregnancy due to fetal thyroid ablation risk. In rare cases, surgery may be considered during the second trimester if medical management fails or is contraindicated. In addition to pharmacologic treatment, monitoring fetal heart rate, growth and amniotic fluid levels via ultrasound is crucial to detect early signs of fetal thyrotoxicosis. Multidisciplinary management involving obstetricians, endocrinologists and neonatologists is key to optimizing outcomes [3].

Thyroid autoimmunity, even in the absence of overt dysfunction, has been associated with adverse pregnancy outcomes. The presence of thyroid autoantibodies, particularly TPOAb and thyroglobulin antibodies, has been linked to increased rates of miscarriage, preterm labor and postpartum thyroiditis. Women with positive antibodies are also more likely to develop hypothyroidism during pregnancy or after delivery. Although the mechanisms remain under investigation, it is hypothesized that thyroid autoimmunity reflects a broader state of immune dysregulation that may affect placental function and immune tolerance. Some studies have shown that levothyroxine supplementation in euthyroid TPOAb-positive women may reduce miscarriage and preterm birth rates, but conclusive evidence is still lacking. Postpartum thyroiditis, a common consequence of autoimmune thyroid disease, typically presents within the first year after delivery and may manifest as transient hyperthyroidism, hypothyroidism or both. It is essential to identify at-risk women early and monitor them closely postpartum. Maternal mental health may also be impacted by thyroid dysfunction, with increased risk of postpartum depression

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in women with untreated hypothyroidism. Additionally, thyroid autoimmunity is more prevalent in women with other autoimmune conditions such as type 1 diabetes and lupus, warranting comprehensive endocrine screening in such populations. Overall, the presence of thyroid autoantibodies during pregnancy serves as an important risk marker, necessitating individualized monitoring strategies even in the absence of overt hormone derangement. Future studies are needed to better define the role of early intervention in antibody-positive, euthyroid women to improve maternal and fetal outcomes [4].

Management of thyroid dysfunction during pregnancy requires a nuanced, trimester-specific approach to ensure the best outcomes for both mother and child. Interpretation of thyroid function tests is complicated by physiological changes such as increased Human Chorionic Gonadotropin (hCG), which suppresses TSH levels in early pregnancy. Thus, pregnancy-specific reference ranges for TSH and free thyroid hormones are essential for accurate diagnosis. Iodine sufficiency is another critical factor, as inadequate iodine intake can lead to maternal and fetal hypothyroidism. Supplementation with 150-250 µg of iodine daily is recommended during pregnancy and lactation. Close collaboration among healthcare providers is key, especially in managing complex cases involving preexisting thyroid disease. Women with known thyroid disorders should ideally be evaluated preconceptionally to optimize thyroid function before pregnancy. Once pregnant, thyroid function should be monitored every 4-6 weeks and levothyroxine or antithyroid medications should be adjusted accordingly. Education and counseling are important to ensure medication adherence and reduce anxiety associated with thyroid disease in pregnancy. Neonates born to mothers with thyroid dysfunction should undergo thyroid function screening soon after birth, especially if maternal antibodies or medications may have affected fetal thyroid status. Finally, continued follow-up postpartum is necessary, particularly for women with autoimmune thyroid disease or a history of postpartum thyroiditis. With early detection, appropriate therapy and vigilant monitoring, the adverse outcomes associated with thyroid dysfunction during pregnancy can be largely mitigated [5].

Conclusion

In conclusion, thyroid dysfunction during pregnancy represents a significant but manageable threat to maternal and fetal health. Both hypothyroidism and hyperthyroidism are associated with a spectrum of complications, ranging from miscarriage and preterm birth to neurodevelopmental delays and fetal growth restriction. Autoimmune thyroid disease further complicates pregnancy outcomes and may contribute to postpartum thyroid dysfunction. Early diagnosis through appropriate screening, trimester-specific interpretation of thyroid function tests and individualized treatment plans are essential components of effective care. Levothyroxine and antithyroid medications, when

used judiciously, can minimize risk and improve pregnancy outcomes. Multidisciplinary coordination and patient education play vital roles in optimizing treatment adherence and monitoring. As evidence grows, there is increasing support for refining screening protocols and improving access to prenatal endocrine care, particularly in underserved regions. Ultimately, proactive management of thyroid disorders in pregnancy holds the promise of healthier pregnancies, safer deliveries and improved long-term outcomes for mothers and their children.

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Conflict of Interest

None.

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