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Review Article

Hypothyroidism in pregnancy current guidelines, prevalence, and implications for maternal and fetal health

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ABSTRACT

Hypothyroidism during pregnancy is a common endocrine disorder that can drastically impact the mother and fetus in the process of development. The two types of hypothyroidism in pregnancy are distinguished as overt and subclinical, with the latter being more prevalent. Subclinical hypothyroidism (SCH) is commonly caused by autoimmune thyroid disorders and iodine deficiency. In iodine-sufficient areas, hypothyroidism is caused by Hashimoto's thyroiditis, whereas, in iodine-deficient areas, hypothyroidism is primarily caused by iodine deficiency. Diagnosing hypothyroidism during pregnancy is often challenging because its symptoms often mimic those of normal pregnancy. During pregnancy, thyroid function undergoes significant alterations. The disruption of the normal functioning of the hypothalamic-pituitary-thyroid axis causes fluctuations in both thyroid stimulating hormone (TSH) and thyroxine (T4) levels. Elevated human chorionic gonadotropin (hCG) and estrogen levels trigger the thyroid gland to increase hormone production. If hypothyroidism is not managed properly throughout pregnancy, it could lead to preeclampsia and premature delivery and neurodevelopmental concerns in the baby. It is recommended to consider hypothyroidism screening for women with autoimmune conditions or an existing history of thyroid disease due to their increased risk. The main treatment for pregnant women with hypothyroidism is levothyroxine, whose dosage is regulated to meet the increased demand for thyroid hormones. Continuous monitoring of free T4 levels and TSH is crucial in maintaining hormonal balance. The present review explores the prevalence of hypothyroidism during pregnancy, physiological changes, maternal and fetal complications, and the evidence-based guidelines for the management. Following current recommendations and proper treatment minimizes the risks for the mother and the baby.

Keywords: Hypothyroidism, Pregnancy, Maternal and fetal health

INTRODUCTION

Hypothyroidism during pregnancy is a common endocrine disorder characterized by elevated thyroid-stimulating hormone (TSH) with low free thyroxine (FT4) levels.[1,2] There are two types of this condition. Overt hypothyroidism is characterized by elevated TSH and reduced free thyroxine (FT4) level. In pregnant women, overt hypothyroidism is less common as per the clinical findings with a reported prevalence rate of 0.3-0.5%.[3] Subclinical hypothyroidism (SCH) is diagnosed with increased TSH and normal FT4. Subclinical hypothyroidism is more common than overt hypothyroidism, with prevalence of 2-3% among pregnant women.[3] Some evidence suggests that some populations, especially those in India, suffer from very high prevalence of SCH at 32.94 %.[4]

Compared to overt hypothyroidism, SCH is more common, and is frequently associated with autoimmune problems such as Hashimoto's thyroiditis and inadequate iodine consumption.[5] Autoimmune thyroiditis, primarily related to Hashimoto's thyroiditis, is the leading cause of hypothyroidism in areas with sufficient iodine intake.[6] Regions that have low levels of iodine consumption contribute a lot to the increased risk of hypothyroidism.[3]

From recent findings in Khyber Pakhtunkhwa, Pakistan, it was reported that about 41.3% of pregnant women had inadequate iodine, and urinary iodine levels below 150 µg/g indicated increased risk of thyroid dysfunction.[7] In pregnancy, hypothyroidism might manifest through non-specific symptoms, which can be easily confused with manifestations of pregnancy. Fatigue, intolerance to cold, constipation, muscle cramps, and weight gain are among the most common symptoms that pregnant women experience.[8] Other symptoms may include visible goiters, hair loss and dry skin. Despite being prevalent, these symptoms can easily be overlooked because they often accompany thyroid conditions.[8]

Numerous risk factors can increase the chances for a woman to develop hypothyroidism during pregnancy. Studies show that older women who are pregnant (after 30 years) have a greater chance of developing thyroid problems in childbirth.[1-3] According to scientific evidence, pregnant women with a BMI higher than 30 kg/m^2 are at a higher risk of thyroid dysfunction.[9] Autoimmune-related conditions, including type 1 diabetes and autoimmune thyroiditis, also increase the rate of thyroid dysfunction in pregnancy.[10] The presence of anti-thyroid peroxidase (anti-TPO) antibodies is another major risk factor associated with adverse pregnancy outcomes such as preterm birth and miscarriage.[11]

Additionally, the reduced maternal hypothyroxinaemia due to iodine deficiency during pregnancy increases the likelihood of having goitre, stillbirth, and infant cognitive delays.[12] Women are recommended to intake 250 µg of iodine daily during their pregnancy and postpartum phases.[13] To achieve this goal, the women must ensure that they take prenatal vitamins containing 150–200 µg of iodine either from potassium iodide or iodate.[13] This review focuses on current guidelines, effects of gestational hypothyroidism, maternal and fetal health impacts, and management approaches during pregnancy.

THYROID PHYSIOLOGY IN PREGNANCY

The hypothalamic-pituitary-thyroid (HPT) axis during pregnancy undergoes alterations. Elevated human chorionic gonadotropin (hCG) concentrations in the first trimester mimic TSH, thereby reducing serum TSH and enhancing stimulation of thyroid activities. This is known as the "hCG-mediated thyroid stimulation" and peaks at the first trimester.[14] At the same time, the high levels of estrogen stimulate the synthesis of thyroxine-binding globulin (TBG), increasing the total thyroxine (T4), leaving free T4 unchanged.[13]

Pregnancy leads to significant anatomical changes of the thyroid gland, manifested mainly as enlargement of the gland by 10–20%, and more extensively in iodine-deficient regions. It is accompanied with an increase in vascularisation and follicular cells proliferation.[15] Additionally the placenta synthesizes the third iodothyronine deiodinase (D3), which mediates the inactivation of maternal thyroxine, hence increasing demand for maternal production of thyroid hormone.[16]

Thyroid hormone synthesis increases by approximately 50% during the second trimester to meet the metabolic requirements of pregnancy.[15] This increase is due to higher TBG and hCG levels, which enable more production and uptake of thyroid hormones.[15] An important component needed to make thyroid hormone, iodine, travels from the mother to the baby via the placenta. Since the fetal thyroid does not start its functioning until the second trimester, the maternal thyroid hormones are essential during the first trimester for fetal neurodevelopment.[17]

During the third trimester, the placenta sustains the expression of D3, which promotes the inactivation of maternal thyroid hormones. This stimulates the maternal thyroid to make additional adjustments to maintain appropriate hormone levels. Increased metabolic needs of the developing fetus and placenta increase the need for thyroid hormones.[15,16] Increased maternal thyroid hormone levels continue, and the gland may enlarge slightly. While the fetus is producing its own thyroid hormones, maternal hormones still play an important role in fetal development.[16] The thyroid physiological changes in pregnancy are also depicted in Figure 1.

MATERNAL AND FETAL COMPLICATIONS OF UNTREATED HYPOTHYROIDISM

Maternal outcomes

If maternal hypothyroidism is not properly treated, it can lead to several complications. Prior studies by Lucaccioni et al revealed an association between overt hypothyroidism and increased preeclampsia, anemia, and placental abruption.[18] Further investigation in Denmark showed that women affected by hypothyroidism had a higher risk of developing preeclampsia, as evidenced by adjusted odds ratio (aOR) of 1.3 compared to euthyroid women.

The risk was especially high in women who were first diagnosed with hypothyroidism during pregnancy, and had an aOR of 1.6.[19] Moreover, evidence has revealed that untreated hypothyroidism can elevate the risk of postpartum hemorrhage in women. The studies further suggest that the subclinical hypothyroidism and thyroid autoimmunity may cause these negative consequences, but the evidence is conflicting.[20]

Fetal outcomes

If left untreated, hypothyroidism during pregnancy can lead to preterm delivery, low birth weight, problems with breathing, developmental delays, and increased risk of miscarriage.[21,22] Toloza et al, conducted a meta-analysis and systematic review and revealed that maternal subclinical hypothyroidism is associated with increased risk of preeclampsia and adverse outcomes for both the

mother and the fetus, including intrauterine growth restriction and preterm delivery.[23] Untreated hypothyroidism during pregnancy is linked with increased risk for neurodevelopmental issues in children, such as reduced IQ and cognitive impairment.[24] Research has confirmed that despite treatment with levothyroxine, the occurrence of maternal hypothyroidism can reflect in lower IQ scores in children.[25]

Diagnosis of gestational hypothyroidism

Untreated hypothyroidism in pregnant women may have significant negative impacts on mother's and baby's health. The American Thyroid Association (ATA) recommends focusing on screening at-risk groups instead of universally screening all pregnant women. High-risk groups include those with a previous history of thyroid disease, autoimmune disorders, common pregnancy complications, and those who exhibit thyroid-related symptoms.[26] Table 1 presents trimester specific TSH reference values that form the basis for diagnosing hypothyroidism in pregnant women.[21] High levels of TSH, in combination with FT4, indicate a diagnosis of hypothyroidism. Subclinical hypothyroidism may be detected if the FT4 is normal but TSH is higher than the normal rang.[26]

Table 1: Trimester-specific reference ranges for TSH in pregnancy.[13,21]

Trimester	TSH reference range (mIU/l)
1st	0.1–2.5
2nd	0.2–3.0
3rd	0.3–3.0

Measurement of TPO antibody level may have a role in diagnosing autoimmune thyroiditis, the common cause of hypothyroidism in pregnancy.[27] Individuals with anti-TPO antibodies have an increased risk of miscarriage, preterm birth, as well as postpartum thyroiditis.[11] For this reason, screening for TPO antibodies, especially in high-risk patient groups plays an important role in providing prognostic information and appropriate management.

Guidelines and variations

The 2017 ATA guidelines recommend measuring TSH levels as soon as a pregnancy in high-risk women for thyroid disease is confirmed.[26] The ATA recommends increasing levothyroxine therapy upon confirmation of pregnancy in women diagnosed with hypothyroidism because the thyroid hormones are required in larger amounts during pregnancy.[26] TSH testing should be done every 4 weeks until mid-pregnancy when the thyroid function becomes stable, and at least once at around 30 weeks' of gestation.[21,26] On the contrary, although the British guidelines also use the same TSH targets as the ATA, they differ in their monitoring method. The British recommendations advocate for frequent TSH measurements every four weeks during pregnancy, with an extra checkup around week 30 for confirmation of adequate thyroid function.[28]

Based on guidelines issued by the European thyroid association (ETA) in 2021, all patients with hypothyroidism and levothyroxine therapy who want to have assisted reproductive technology (ART) must ensure that their serum TSH levels are <2.5 mIU/l before starting the treatment.[29] The Royal College of Obstetricians and Gynaecologists (RCOG) in the UK has recommended guidelines for treating thyroid disorders during pregnancy. RCOG does not recommend regular monitoring for thyroid dysfunction and TPO antibodies during pregnancy.

According to RCOG guidelines, women at increased risk, such as those with a history of thyroid disease or autoimmune disorders, may require thyroid screening and TPO antibody testing.[30] In order to effectively treat thyroid disorder during pregnancy and the postpartum period, it is crucial to adhere to trimester-specific TSH reference levels, as specified in the recommendations of the endocrine society. A TSH level under 2.5 mIU/l in the first trimester and below 3.0 mIU/l in the second and third trimesters.[31]

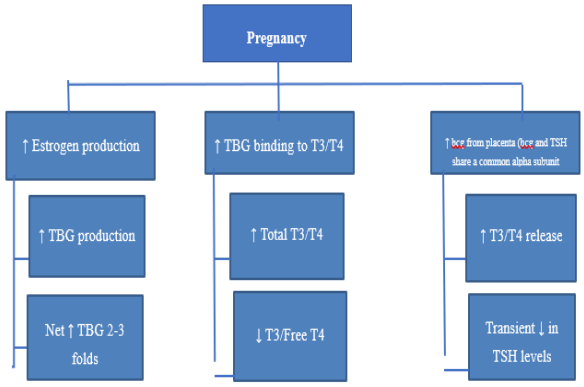

Figure 1: Changes in thyroid hormones during pregnancy.

Treatment protocols

Levothyroxine (LT4) therapy remains the cornerstone for managing hypothyroidism during pregnancy, encompassing both overt and subclinical forms.[32] It is recommended that levothyroxine be titrated to reach a target TSH in the lower half of the trimester-specific reference range. If these are not available locally, maternal TSH concentrations should be kept at ≤2.5 mIU/l during the first trimester and ≤3.0 mIU/l during the second and third terms.[21]

In comparison to the pre-pregnancy dosage, levothyroxine is usually raised by around 30% to 50%.[13] In cases of SCH, characterized by elevated TSH levels (>10 mIU/l) with normal free T4 concentrations, treatment with LT4 is generally recommended.[33] For women with SCH and TSH

levels between 5.22 and 10 mIU/l, especially those who are TPO antibody-positive, initiating LT4 therapy has been shown to reduce the risk of miscarriage and preterm delivery.[34]

To guarantee proper dosage, thyroid function must be closely monitored. Throughout pregnancy, TSH levels should be checked every four to six weeks until euthyroidism is attained. Once stable, monitoring can be reduced but should continue throughout pregnancy to ensure optimal maternal and fetal health. It is important to note that overtreatment with LT4, resulting in TSH levels below 0.10 mIU/l, has been associated with an increased risk of preterm delivery.[35]

Moreover, thyroid function test (TFT) monitoring is necessary every 3-6 weeks for euthyroid women who test positive for thyroid peroxidase antibody (TPOAb) in order to identify TSH increases beyond the typical pregnancy-specific levels.[13] When the TSH is more than 2.5 mU/l but less than the maximum limit of the reference range related to pregnancy, levothyroxine replacement should be taken into consideration.[13] Most women return to their pre-pregnancy levothyroxine dosage after giving birth. It is recommended that thyroid function tests be performed six weeks after giving birth.[13,21]

DISCUSSION

This review highlights the significance of identifying hypothyroidism in pregnancy by detailing the thyroid's physiological adjustments, the potential maternal and fetal risks if untreated, and the vital role of timely detection and proper management. The key finding of this study is the high prevalence of subclinical hypothyroidism (SCH) in pregnant women compared to overt hypothyroidism. Overt hypothyroidism happens in only 0.3–0.5% of pregnancies, but SCH impacts 2–3% worldwide. In some areas like India, it has been reported higher rates, up to 32%. 94%.[36] This variation may be due to differences in iodine nutrition, hereditary inclination, and autoimmune rates.

These findings matched with studies from areas of iodine deficiency, like Khyber Pakhtunkhwa, Pakistan, where iodine deficiency was found in 41.3% of pregnant women.[37] These findings are on the same line with studies exploring that iodine deficiency and autoimmune thyroiditis related to hypothyroidism in pregnancy. Autoimmune conditions are more common in areas with sufficient iodine intake.[38] The maternal symptoms that are related to hypothyroidism, for example fatigue, constipation, and sensitivity to cold, are non-specific but serve as significant markers of possible thyroid dysfunction.

Unfortunately, these symptoms frequently resemble typical alterations occurring during pregnancy, leading to a lack of diagnosis. This highlights the necessity of regular testing for high-risk women, as advised by the American Thyroid Association.[39] Notably, during pregnancy, the thyroid undergoes several physiological changes such as a rise in thyroxine-binding globulin (TBG), elevated production of thyroid hormones, and the placenta's expression of deiodinase type III (D3), all of which increase the demand for thyroid hormones in the mother. As a result, women who are on the verge of thyroid dysfunction might develop hypothyroidism during pregnancy if they do not receive adequate oversight and supplementation.[40,41]

The clinical ramifications of untreated hypothyroidism were underscored as well. Various research works including those conducted by Lucaccioni et al in 2020 and Andersen et al in 2014 identified links between maternal hypothyroidism and elevated risks for preeclampsia, anemia, placental detachment, and postpartum hemorrhage. These observations correspond with findings from a Danish study, which indicated that women who were diagnosed with hypothyroidism during their pregnancy faced a considerably higher likelihood of preeclampsia (adjusted odds ratio of 1.6).[42,43]

The possible role of autoimmune thyroid conditions in these results is still debated but has been proposed in several research efforts.[44] Fetal outcomes were greatly influenced by maternal hypothyroidism. In line with the meta-analysis conducted by Toloza and colleagues in 2022 our review validates the links between maternal subclinical hypothyroidism and preterm births, intrauterine growth restriction, low birth weights, and compromised neurodevelopment.[45] Pioneering studies by Haddow and his team in 1999 showed that offspring of mothers with untreated hypothyroidism exhibited notably lower IQ levels.[46] While treatment with levothyroxine reduces many associated risks, some research suggests ongoing developmental issues may persist even in patients receiving treatment, highlighting the importance of initiating thyroid hormone therapy at an early stage.[47]

Regarding to diagnosis, the guidelines by the American Thyroid Association (ATA), European Thyroid Association (ETA), and Royal College of Obstetricians and Gynaecologists (RCOG recommend using TSH levels that fit each trimester for a clear diagnosis. The ATA and ETA both support a TSH level of 2.5 mIU/l in the first trimester and 3.0 mIU/l in later trimesters, showing the natural drop in TSH levels caused by thyroid boost from hCG.39,47 Also, checking for anti-thyroid peroxidase (anti-TPO) antibodies is key for finding autoimmune hypothyroidism and looking at risk for miscarriage postpartum thyroiditis and preterm labor.[48]

Treatment with levothyroxine (LT4) continues to be the primary approach for managing both overt cases and specific instances of subclinical hypothyroidism. Present research endorses the prompt commencement of treatment, especially for women who are TPOAb-positive and have TSH levels exceeding 10 mIU/l. Studies indicate that even among women with TSH values ranging from 5 to 10 mIU/l, levothyroxine can mitigate negative health

impacts, specifically for those who are TPOAb-positive.[49] Nevertheless, it is important to exercise caution to prevent excessive dosing, as elevated LT4 levels can lead to preterm delivery and fetal tachycardia.[50]

It is important to recognize that there are differences in guidelines. While the ATA and ETA support rigorous observation and treatment, the RCOG recommends a more cautious strategy that focuses on symptoms and risk when it comes to screening and monitoring.[51] These distinctions might illustrate various healthcare priorities, the availability of resources, and different understandings of the available evidence.

CONCLUSION

Hypothyroidism during pregnancy, whether overt or subclinical, presents significant challenges to maternal and fetal health. The condition requires careful management through levothyroxine therapy, with dosing adjustments tailored to trimester-specific thyroid hormone needs. To maintain euthyroidism, FT4 and TSH levels must be regularly monitored. This is especially important since untreated hypothyroidism is linked to a higher risk of complications, including premature birth, preeclampsia, and neurodevelopmental impairments. Regional differences exist in the prevalence of thyroid dysfunction, with autoimmune thyroiditis and iodine deficiency being major contributors. Adhering to updated guidelines, including those from the ATA, Endocrine Society, and others, provides a framework for optimal care, reducing the risks for both mother and child and improving pregnancy outcomes.

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