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Hyperthyroidism in Pregnant Women: Pathophysiology, Risks, Diagnosis, and Management

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Abstract: Hyperthyroidism during pregnancy is a rare but clinically significant endocrine disorder associated with substantial maternal and fetal complications. The condition is commonly caused by Graves' disease or excessive stimulation of the thyroid gland by human chorionic gonadotropin (hCG) in early gestation. Hormonal, immunological, and metabolic alterations in pregnancy influence thyroid hormone regulation, potentially exacerbating pre-existing hyperthyroidism or triggering new-onset disease. Uncontrolled maternal hyperthyroidism increases the risk of preeclampsia, miscarriage, preterm birth, fetal growth restriction, and neonatal thyroid dysfunction. This article reviews the pathophysiology, maternal and fetal risks, diagnostic approaches, and evidence-based management strategies for hyperthyroidism in pregnancy, emphasizing early detection, appropriate pharmacological interventions, and multidisciplinary monitoring to optimize maternal-fetal outcomes.

Keywords: hyperthyroidism, pregnancy, Graves' disease, thyroid hormones, maternal complications, fetal outcomes, antithyroid drugs, diagnosis, endocrine disorders

Introduction

Hyperthyroidism affects approximately 0.1–0.4% of pregnancies worldwide, making it a relatively uncommon yet clinically important condition (Alexander et al., 2017). Pregnancy-induced physiological changes alter thyroid hormone metabolism, sometimes masking or exacerbating hyperthyroid symptoms. The most frequent cause is Graves' disease, an autoimmune disorder involving thyroid-stimulating hormone receptor antibodies (TRAb) (De Groot et al., 2012). Other causes include gestational transient thyrotoxicosis, toxic multinodular goiter, and, rarely, thyroid adenomas (Stagnaro-Green et al., 2011).

Given the potential for serious maternal and fetal complications, timely diagnosis and appropriate management of hyperthyroidism in pregnancy are essential to reduce adverse outcomes.

Pathophysiology of Hyperthyroidism in Pregnancy

Pregnancy introduces unique endocrine and immunological changes affecting thyroid function:

1. **hCG stimulation:** Human chorionic gonadotropin peaks in early pregnancy and can weakly bind thyroid-stimulating hormone (TSH) receptors, transiently increasing thyroid hormone production (Goodwin et al., 2020).
2. **Increased thyroxine-binding globulin (TBG):** Estrogen-induced elevation of TBG raises total T4 and T3 levels, requiring trimester-specific reference ranges for accurate diagnosis (Glinoer, 1997).
3. **Immunological modulation:** Immune tolerance shifts during pregnancy influence autoimmune thyroid disease activity; Graves' disease often improves in late gestation but may relapse postpartum (De Groot et al., 2012).

Maternal and Fetal Risks

Maternal complications:

- Preeclampsia and gestational hypertension (Männistö et al., 2009)
- Heart failure or arrhythmias in severe cases (De Groot et al., 2012)

- Thyroid storm, a life-threatening thyrotoxic crisis (Ross, 2016)
- Increased risk of miscarriage and preterm labor (Stagnaro-Green et al., 2011)

Fetal and neonatal complications:

- Fetal growth restriction and low birth weight (Korevaar et al., 2017)
- Fetal tachycardia and goiter due to transplacental TRAb transfer (De Groot et al., 2012)
- Neonatal hyperthyroidism or hypothyroidism, depending on maternal antibodies and antithyroid drug exposure (Ross, 2016)

Diagnosis and Monitoring

- **Thyroid function tests:** Suppressed TSH with elevated free T4 or total T4 above pregnancy-specific reference ranges confirms hyperthyroidism (Alexander et al., 2017).
- **TRAb measurement:** Recommended in women with Graves' disease to assess fetal risk (De Groot et al., 2012).
- **Ultrasound & fetal surveillance:** Used to evaluate fetal growth, heart rate, and goiter presence in high-risk pregnancies (Stagnaro-Green et al., 2011).

Treatment and Management Strategies

1. **Antithyroid drugs (ATDs):**

- **Propylthiouracil (PTU)** is preferred in the first trimester due to lower teratogenic risk (Ross, 2016).
- **Methimazole (MMI)** is recommended after the first trimester to reduce maternal hepatotoxicity risk from prolonged PTU use (Alexander et al., 2017).
- The lowest effective dose should maintain maternal free T4 at the upper limit of normal to prevent fetal hypothyroidism.

2. **β-blockers:** Short-term use (e.g., propranolol) may control adrenergic symptoms such as tachycardia but should be discontinued as soon as clinically feasible (Ross, 2016).
3. **Surgery:** Thyroidectomy is reserved for women intolerant to ATDs or with large goiters; second trimester is the safest period if surgery is indicated (De Groot et al., 2012).
4. **Multidisciplinary care:** Collaboration between endocrinologists, obstetricians, and pediatricians ensures optimal maternal-fetal outcomes (Stagnaro-Green et al., 2011).

Conclusion

Hyperthyroidism in pregnancy requires prompt recognition, accurate diagnosis using trimester-specific hormone reference ranges, and individualized treatment strategies to minimize risks for both mother and fetus. Antithyroid drugs remain the cornerstone of therapy, with PTU preferred in early pregnancy and MMI in later trimesters. Close fetal surveillance, TRAb monitoring, and postpartum follow-up are critical to detect neonatal thyroid dysfunction and prevent long-term complications. Multidisciplinary care optimizes outcomes, emphasizing the importance of evidence-based clinical guidelines in managing hyperthyroidism during pregnancy.

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