Thyroid Dysfunction in Pregnancy

Thyroid dysfunction is a common occurrence during pregnancy and is associated with various adverse outcomes. Both overt and subclinical thyroid diseases can significantly impact maternal and fetal health. Fetal development relies heavily on maternal thyroid hormones, which are delivered via the transplacental route until about 18 weeks of gestation, and continue to be crucial up to delivery. These hormones are vital for fetal size, tissue maturation, and uteroplacental development. The fetal brain starts responding to thyroid hormones from around 5 weeks of gestation, making euthyroidism essential throughout pregnancy to minimize risks to the fetus.

Changes in Thyroid Function During Pregnancy
During pregnancy, physiological changes occur in the thyroid axis. Maternal thyroid-stimulating hormone (TSH) levels decline during the first trimester but return to pre-pregnancy levels by the end of gestation. Free thyroxine (fT4) concentrations also decline throughout pregnancy. Therefore, clinicians should use gestation-specific reference ranges, which are both population- and age-specific, when caring for pregnant patients with thyroid disease.

Hyperthyroidism in Pregnancy

General Impact. Pregnancy and delivery-related immunological changes can affect thyroid status in various ways, potentially aggravating or ameliorating pre-existing thyroid conditions. Approximately 5-10% of women develop thyroid dysfunction postpartum, with higher risks in those with positive thyroid antibodies, Type 1 diabetes mellitus, a family history of thyroid disease, or other autoimmune conditions. Postpartum thyroid dysfunction often presents as postpartum thyroiditis, Graves' disease, or Hashimoto's thyroiditis.

Postpartum Thyroiditis. Postpartum thyroiditis typically presents with symptoms of thyrotoxicosis around 12 weeks post-delivery, followed by hypothyroidism at around 3-6 months. Histologically, it is characterized by destructive thyroiditis with a lymphocytic infiltrate. Management during the thyrotoxic phase may involve symptomatic treatment with propranolol, and thyroxine (T4) may be needed during the hypothyroid phase. Long-term, these patients are at risk of recurrent postpartum thyroiditis and permanent hypothyroidism in subsequent pregnancies, necessitating specialist and annual TFT monitoring.

Hyperemesis Gravidarum and Thyrotoxicosis. Transient thyrotoxicosis may occur in hyperemesis gravidarum, likely due to high levels of human chorionic gonadotropin (HCG) stimulating TSH receptors. Management is supportive, avoiding antithyroid medications. This condition can be distinguished from Graves' disease by a negative family history, lack of autoimmune illness, and negative thyroid antibodies.

Graves' Disease During Pregnancy. Graves' disease is the leading cause of thyrotoxicosis in pregnancy, affecting about 1 in 500 women. It may contribute to menstrual irregularities, conception difficulties, miscarriage, premature labor, low birth weight, and pre-eclampsia. Proper management involves achieving biochemical euthyroidism with antithyroid medications and beta-adrenergic blocking agents. Radioiodine is contraindicated in pregnancy. Propylthiouracil is preferred in the first trimester due to fewer teratogenic effects, while carbimazole is recommended in the second and third trimesters due to concerns about liver function abnormalities. Monitoring anti-TSH-receptor antibodies is crucial to manage potential fetal thyrotoxicosis.

Hypothyroidism in Pregnancy

Overt hypothyroidism. Overt hypothyroidism can cause anovulation and first-trimester abortion, with untreated cases increasing the risk of spontaneous miscarriage, pregnancy-induced hypertension, pre-eclampsia, low birth weight, and perinatal mortality. Maternal hypothyroidism is also linked to neurodevelopmental defects. Thyroid hormone replacement is necessary pre-conception and throughout pregnancy, with frequent TFT monitoring and dose adjustments of levothyroxine based on serum TSH levels. Obstetricians recommend managing hypothyroid pregnancies as high-risk, with prophylactic aspirin for pre-eclampsia and uterine artery Doppler studies for malplacentation screening.

Subclinical Hypothyroidism. Subclinical hypothyroidism rates during pregnancy depend on the iodine status of the population. It is associated with increased risks of pregnancy loss, pre-term delivery, placental abruption, breech presentation, and neurodevelopmental defects. Levothyroxine therapy is often recommended, particularly in TPO antibody-positive women, to enhance live birth rates in assisted conception. However, guidelines on treating TPO antibody-negative women are less clear, with varying recommendations from different professional bodies.

This comprehensive overview underscores the importance of careful monitoring and management of thyroid dysfunction during pregnancy to ensure optimal maternal and fetal outcomes.

End of the chapter

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Thyroid Dysfunction in Pregnancy

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