South Australian Perinatal Practice Guidelines

Thyroid disorders in pregnancy

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Thyroid function during pregnancy

- In pregnancy, the values influenced by the serum thyroid binding hormone level (total thyroxine, total triiodothyronine, and resin triiodothyronine uptake) change significantly
- Plasma iodide levels decrease as a result of fetal iodide use and increased maternal renal clearance. In about 15 % of pregnant women, these lower iodide levels are associated with a noticeable increase in thyroid gland size (Schroeder 2002)

Hyperthyroidism

- Hyperthyroidism is the second most common endocrine problem developing in pregnancy (occurs in 0.2 % of pregnancies) (Major, Nageotte 1999)
- Graves' disease is responsible for 95 % of hyperthyroidism cases in pregnancy
- Many of the signs mimic those of normal pregnancy e.g. palpitations, hyperactivity, sweating, increasing frequency of bowel movements (Major, Nageotte 1999).
- More specific signs include tachycardia at rest, goitre and exophthalmos

Diagnosis

- Diagnosis depends on the measurement of thyrotropin (TSH) which is suppressed in active disease. In addition, both free and total thyroid hormone concentrations are increased
- Other investigations in hyperthyroidism and thyrotoxicosis include:
  - Measurement of Technetium uptake. This is contraindicated in pregnancy, because of the risk of fetal uptake of the isotope and damage to the fetal thyroid
  - Ultrasound may show diffuse enlargement, characteristic of autoimmune disease, or multinodularity, suggestive of autonomous multinodular goitre
  - Serological tests may show positive thyroid antibodies, higher titres reflecting an increased risk of post partum recurrence
  - High titres of thyroid stimulating immunoglobulin (TSIg) carry an increased risk of neonatal Graves' disease (also in women who have had thyroidectomy for Graves' disease)

Management

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- Drug treatment is based on thioamide agents: carbimazole (CBZ) and propylthiouracil (PTU). Of the two agents propylthiouracil is the drug of choice during pregnancy and breastfeeding:
  - Initial treatment is 50-100 mg three times a day, reducing to 50 mg once or twice daily when the hyperthyroid state is controlled
  - Because of the risk of fetal hypothyroidism with all thioamide treatment, the lowest possible maintenance dose should be used to maintain TSH in the normal range. Six-weekly checks of the free thyroid hormone and TSH concentrations are recommended, but note that TSH levels may take longer to return to normal
  - Failure of control may indicate the need for partial thyroidectomy
  - Autoimmune hyperthyroidism often improves during pregnancy so that the dose of drug may be reduced, but a flare up post partum is common, requiring an increase in dosage

Other drug treatments:

- Beta-blockers (e.g. propranolol 40 mg every 8 hours, oxprenolol 80 mg every 8 hours, or atenolol 100 mg daily) may be useful to control symptoms until antithyroid medication can take effect or to control symptoms before surgery
- Potassium iodide, often used in preparation for thyroid surgery, is well recognised to induce fetal goitre even with small daily doses
- Radio-iodine (131I) in therapeutic doses is liable to destroy fetal thyroid function

Complications

- Untreated hyperthyroidism carries a high risk of preterm birth and perinatal mortality
- Fetal and neonatal thyrotoxicosis may result from placental transfer of thyroid-stimulating antibodies. Fetal tachycardia can be used as an indicator of this. Thioamide treatment has been used successfully to control the fetal thyrotoxicosis, monitoring the fetal heart rate as a guide to dosage
- In women with unrecognised hyperthyroidism the stress of an infection, labour or operative delivery may lead to the rare thyroid storm. This is a medical emergency with a high risk of morbidity and mortality to mother and fetus
- Calcium channel blockers (nifedipine) are first line management for preterm labour

Hypothyroidism

- Hypothyroidism is usually diagnosed and treated before pregnancy, as the hypothyroid state is often associated with infertility
- Untreated, it is associated with poor pregnancy outcome, including miscarriage, stillbirth, preterm labour and poor neurological development in the newborn (Nader 2004)
- The most common aetiology is thyroid damage due to surgery or radiotherapy. Other causes include autoimmune disease (Hashimoto's thyroiditis and myxoedema) and iodine deficiency

Diagnosis

- Clinical features include dry skin, alopecia, loss of energy, fatigue, muscle cramps constipation, cold intolerance, mental disturbance, bradycardia and myotonic (slow relaxing) reflexes

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- The diagnosis is based on high thyroid stimulating hormone (TSH) concentrations with low free thyroid hormone values
- The presence of thyroid microsomal antibodies also increases the risk of thyroid dysfunction post partum

Treatment

- Thyroid replacement is with L-thyroxine 100-200 microgram per day as a single dose, monitoring response by the decline in the serum TSH concentration
- In pregnancy, about 30 % of hypothyroid women on replacement treatment will require a higher dose than before pregnancy. The important point is to monitor and treat on the basis of thyroid function tests rather than by clinical judgement
- Assessment of thyroid function tests once in each trimester is usually sufficient
- In the puerperium, any increase in thyroxine dose will need reduction again at about six weeks post partum
- Breastfeeding is recommended

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