

South Australian Perinatal Practice Guidelines

Thyroid disorders in pregnancy

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Note:

This statewide guideline has been prepared to promote and facilitate standardisation and consistency of practice, using a multidisciplinary approach.

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The clinical material offered in this statewide standard/policy provides a minimum standard, but does not replace or remove clinical judgement or the professional care and duty necessary for each specific patient case. Where care deviates from that indicated in the statewide guideline contemporaneous documentation with explanation must be provided.

This statewide guideline does not address all the elements of clinical practice and assumes that the individual clinicians are responsible for:

- > Discussing care with consumers in an environment that is culturally appropriate and which enables respectful confidential discussion. This includes the use of interpreter services where necessary,
- > Advising consumers of their choice and ensuring informed consent is obtained,
- > Providing care within scope of practice, meeting all legislative requirements and maintaining standards of professional conduct, and
- > Documenting all care in accordance with mandatory and local requirements

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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

- > 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

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- > 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
- > 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

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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

Reference

1. Schroeder BM. ACOG Practice Bulletin on Thyroid disease in pregnancy. American Family Physician 2002; 65: 2161 - 2.
2. Major CA, Nageotte MP. Thyroid disease. In: James DK, Steer PJ, Weiner CP, Gonik B editors. High risk pregnancy: management options. 2nd ed. London: Harcourt; 1999. p. 709 - 15.
3. Nader S. Thyroid disease and other endocrine disorders in pregnancy. Obstet Gynecol Clinics 2004; 31: 257 - 85.

Version control and change history

PDS reference: OCE use only

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