Guideline for the management of infants born to mothers with thyroid disease

This guideline provides guidance for the postnatal assessment and management of infants born to mothers with thyroid disease. If should be used in conjunction with **appendix one** – pathway for infants born to mothers with thyroid disease and **appendix two** – family information leaflet.

Maternal Hypothyroidism

The prevalence of hypothyroidism during pregnancy is 0.3-0.5% for overt hypothyroidism, with raised Thyroid Stimulating Hormone (TSH) and low T4 levels. Subclinical hypothyroidism with raised TSH and normal T4 levels has a higher incidence of 2-3%.

Autoimmune thyroiditis (Hashimotos disease) is the main cause of hypothyroidism during pregnancy. Determination of thyroid peroxidase antibodies confirms the autoimmune origin. Pregnant women with a history of hypothyroidism should have regular Thyroid Function Tests (TFT) to optimise therapy. TSH receptor antibodies/ Thyroid stimulating immunoglobulin (TRAb/TSI) are no longer routinely tested in hypothyroidism. Babies born to mothers with Hashimoto thyroiditis are at low risk of developing transient hypothyroidism and hyperthyroidism is extremely rare. Neonates with transient congenital hypothyroidism will have a raised TSH which will be picked up by the routine neonatal blood spot screening. Therefore, these babies will not require further investigations or observation after delivery.

Maternal Hyperthyroidism

Maternal hyperthyroidism occurs in 0.1 - 0.4% of all pregnancies. Graves' disease is the most common reason accounting for 85% of cases. Other causes include; single toxic adenoma, multinodular toxic goitre, thyroiditis, and rarely gestational hyperthyroidism and mutations in the TSH receptor.

Women with Graves' disease have thyroid receptor antibodies that can stimulate or inhibit the fetal thyroid. Inhibitory TRAbs may occasionally cause transient neonatal hypothyroidism in neonates of mothers with Graves' disease. Pregnant women with hyperthyroidism require close observation of thyroid activity with fetal vigilance for tachycardia and goitre.

Mothers with hyperthyroidism, especially those with a current or previous history of Graves' should have TSI measured during pregnancy. TSI often remain positive after treatment with radioactive lodine or thyroidectomy. Positive TRAb or TSI is associated with increased risk of neonatal thyrotoxicosis.

Women who have a negative TRAb/TSI have a very low risk of fetal or neonatal thyroid dysfunction and require normal postnatal care ONLY.

Neonatal Thyrotoxicosis

Neonatal thyrotoxicosis is mainly caused by the transplacental transfer of TRAb/TSI in Graves' disease. Rarely, genetic mutations in the TSH receptor can cause neonatal hyperthyroidism. This should be suspected if there is a strong family history of thyrotoxicosis.

Neonatal thyrotoxicosis occurs in 1-10% of offspring of mothers with Graves'. However, the incidence can be as high as 20% if mothers require anti thyroid drugs (ATDs) in the last trimester. Mortality is significant, between 12% and 20%, usually from cardiac failure. Onset is variable, from birth up to 10 days due to the effects of maternal ATDs wearing off quicker than maternal antibodies. Duration of neonatal thyrotoxicosis depends on the persistence of the maternal antibodies and usually remits after 8-20 weeks. Clinically, infants may have goitre, be irritable, tachycardic and have eye signs (see appendix one – Care Pathway, for list of signs and symptoms)

Investigation and Management

See Care Pathway for infants born to Mothers with Thyroid Disease (appendix one).

Abnormal Results

Reference ranges taken from - Microsoft Word - Reference ranges Jan 18.docx (rcpch.ac.uk)

Parameter	Age if applicable	Normal Range
Thyroid Stimulating Hormone (TSH)	0-3 days	5.2 – 14.6mU/L
	4 days – 12 months	0.6 – 8.1mU/L
Free T4	0 – 6 days	11-32pmol/L
	7 days – 3 months	12-18pmol/L
	Over 3 months	12-24pmol/L
Free T3		3-9pmol/L

Thyrotoxicosis is associated with significant morbidity and mortality. The decision of whether to treat is complex. All cases where treatment is considered must be discussed with a local or paediatric endocrinologist.

Contact local or tertiary paediatric endocrinologist for advice.

Dr Chris Bidder <u>Christopher.bidder@wales.nhs.uk</u>

Dr Matthew Ryan matthew.ryan@wales.nhs.uk

Or tertiary advice from the paediatric endocrinology team at UHW - 02920 747747

References

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