Management of Subclinical Hypothyroidism in Pregnancy

Universal screening for thyroid dysfunction is not recommended (except in high-risk women) — it aims to detect rare cases of overt hypothyroidism that require treatment. However, more common mildly elevated TSH levels, especially in early pregnancy, can cause uncertainty.

Normal thyroid function is important in conception, pregnancy progression and foetal neuronal development. There is uncertainty about the management of mild hypothyroidism during pregnancy.

PRACTICE POINTS: TREATMENT OF SCH DETECTED DURING PREGNANCY

• The definition and management of subclinical hypothyroidism in pregnancy are controversial.
• For elevated TSH, treat according to guidelines above (see three bullet points).
• For women treated with thyroxine during pregnancy, aim for TSH between 0.1-2.5 mU/L.
• TSH should be re-checked every six weeks after commencing levothyroxine, until 20 weeks gestation, and once in the 3rd trimester - to guide levothyroxine dose adjustment.
• Levothyroxine can be ceased after delivery in all women except those:
  • attempting to conceive again with a history of unexplained spontaneous abortion
  • who had a TSH > 10 mU/L prior to commencing thyroxine therapy
  • who have strongly positive Anti-TPO antibodies

All women should be advised to have Free T4 and TSH tested six weeks post-partum.

Definition of SCH: What is the appropriate upper limit for TSH in pregnancy?

Subclinical hypothyroidism (SCH) is a biochemical diagnosis - a raised TSH with normal FT4. Changing the definition of a ‘raised TSH’ has altered the prevalence of SCH. In the 2011 American Thyroid Association (ATA) guidelines, 2.5 mU/L was suggested as the upper limit for TSH in the 1st trimester – this was too low resulting in over diagnosis of SCH, unnecessary treatment of pregnant women, and unwarranted anxiety amongst women and health care professionals. Reference range studies have generated inconsistent results, but a TSH cut-off of 4 mU/L is now regarded as more reasonable upper limit in the first trimester (except where a local trimester-specific TSH reference intervals are available). The use of an upper TSH limit of 4 mU/L or trimester specific reference intervals have been endorsed by the 2017 ATA guidelines. Several studies have shown adverse obstetric and foetal outcomes when maternal TSH is above 4 mU/L (but in randomised controlled trials to date, thyroxine treatment has not improved outcomes).

Treatment of subclinical hypothyroidism in pregnancy - guidelines are changing.

Women with previously diagnosed hypothyroidism should have their thyroxine dose adjusted preconception to achieve a TSH in the lower part of the reference range (0.1-2.5 mU/L). When pregnancy is confirmed, women should increase their thyroxine dose by about 30%, which can be done by taking a double dose, 2 days per week.

In women without previous hypothyroidism, a balanced approach to an elevated TSH during pregnancy is as follows:

• If TSH is above 10 mU/L, free T4 and TPO Antibodies (Ab) should be measured on the same sample, and thyroxine treatment started immediately at a dose of approximately 100 ug/day.
• If TSH is mildly elevated (4 to 10 mU/L), TSH should be confirmed on a second sample together with free T4 and TPO Ab, and treatment instituted at a dose of 50 to 75 ug/day, provided TSH remains above 4 mU/L.

• Women with borderline TSH of 2.5 to 4.0 mU/L do not require immediate treatment; measurement of TPO Ab levels may be appropriate, and thyroxine treatment can be considered if TPO Ab are positive (to prevent progression to mild hypothyroidism during pregnancy).

These guidelines apply to low risk women with SCH detected incidentally during pregnancy. They do not necessarily apply to women treated with assisted reproduction technology or with adverse obstetric histories (for example multiple previous miscarriages). For these women, many authorities recommend thyroxine treatment of even borderline high TSH levels (although the evidence supporting this approach is weak).

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References
3. Regulation of Maternal Thyroid during Pregnancy. Glinoer et al JCEM 1990; 71(2) : 276-287
4. Treatment of subclinical hypothyroidism or hypothyroxinemia in pregnancy. NEJM 2017;376(9):815-825

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