PREGNANCY PLUS

Hyperthyroidism and pregnancy

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This is one of a series of occasional articles about how to manage a pre-existing medical condition during pregnancy.

Pregnant women with hyperthyroidism need careful management as some may be at increased risk of fetal loss, pre-eclampsia, heart failure, premature labour, and having a low birthweight baby

Various problems may arise in the management of a pregnant patient with hyperthyroidism (see scenario box p 666).[1] This article will explore the problems in relation to the prevalence of hyperthyroidism in pregnancy, therapeutic issues, pregnancy planning, and clinical management. No controlled trials of management have been conducted, but consensus guidelines have recently been published.[2]

How common is hyperthyroidism in pregnancy?

Hyperthyroidism occurs in 2/1000 pregnancies in the United Kingdom.[3] Graves' hyperthyroidism (defined as hyperthyroidism that is the result of stimulation of the thyroid by thyrotrophin receptor stimulating antibodies (TRAb)) is the commonest cause of hyperthyroidism in young women (about 85% of cases) in the United Kingdom.[1] The prevalence of undiagnosed hyperthyroidism in women is about 4.7/1000,[4] and 0.2% of UK women have been previously diagnosed and treated. In areas of mild iodine deficiency the prevalence is higher.[5 6] Box 1 outlines the causes of hyperthyroidism in pregnancy.

In addition to true hyperthyroidism, the more common clinical entity of transient gestational hyperthyroidism may be seen particularly in the first trimester, with a prevalence in Europeans of 2-3% but a much higher prevalence in South Asian populations.

Hyperthyroidism does not often arise for the first time in early pregnancy, but clinicians need to be aware of the symptoms and signs (box 2).

How does hyperthyroidism affect pregnancy?

Pregnancy outcome

Pre-eclampsia, heart failure, fetal loss, premature labour, and having a low birthweight baby are more likely to occur in untreated or poorly controlled thyrotoxic women than in those receiving adequate treatment.[8] A retrospective review of 11 reports documented a 5.6% incidence of fetal death or stillbirth in 249 pregnancies and a further 5% incidence of fetal and neonatal abnormalities.[9] A study of 60 cases of hyperthyroidism in pregnancy over a 12 year period found that metabolic status at delivery correlated with pregnancy outcome.[10] Preterm delivery, perinatal mortality, and maternal heart failure were more common in women who remained thyrotoxic despite treatment or whose hyperthyroidism was first diagnosed during pregnancy.

Women with thyroid hormone resistance (where thyroid hormone and thyrotrophin concentrations are inappropriately high—that is, not due to autoimmunity) also have a high miscarriage rate, indicating a direct toxic effect of thyroid hormones on the fetus.

Fetal and neonatal thyroid dysfunction

Improvement of Graves' hyperthyroidismduring a woman's pregnancy is often associated with a reduction in the titre of maternal serum TRAb concentrations and a change from stimulatory to blocking antibodies. If antibodies do not decline they will cross the placenta and stimulate the fetal thyroid, evidenced by signs of fetal hyperthyroidism such as tachycardia, intrauterine growth retardation, cardiac failure, and the development of fetal goitre.

One to five per cent of neonates of mothers with Graves' disease have hyperthyroidism as a result of the transplacental passage of maternal TRAb concentrations.[11 12] Presentation of neonatal hyperthyroidism may be delayed as antithyroid drugs administered to the mother are cleared more rapidly

Box 1 Causes of hyperthyroidism in pregnancy

- Graves' disease
- Transient gestational hyperthyroidism
- Toxic multinodular goitre
- Single toxic adenoma
- Subacute thyroiditis
- Trophoblastic tumour
- Iodide induced hyperthyroidism
- Struma ovarii
- Thyrotrophin receptor activation

Box 2 Does hyperthyroidism commonly arise de novo in pregnancy?

Most pregnant women with hyperthyroidism are known to have had thyroid disease before the onset of gestation and will already be receiving treatment. A new diagnosis of hyperthyroidism is uncommon in early pregnancy, as untreated disease is associated with reduced fertility. However, in a series of 14 970 first trimester blood samples, undiagnosed Graves' hyperthyroidism was present in about 0.15%.[7] Features such as tachycardia, palpitations, systolic murmur, bowel disturbance, emotional upset, and heat intolerance may be seen in normal pregnancy but should alert the clinician to the possibility of hyperthyroidism, particularly if a goitre or more specific feature of thyroid disease (weight loss, eye signs, tremor or pre-tibial myxoedema) is observed. Newly diagnosed hyperthyroidism should be aggressively treated.

from the fetal circulation than maternal stimulating antibodies.

Maternal euthyroidism is particularly important in the later stages of pregnancy, as poorly controlled hyperthyroidism can lead to suppression of the fetal pituitary thyroid axis resulting from placental transfer of thyroxine. A case-control study noted a low thyrotrophin concentration, a blunted result (that is, suppressed compared with the normal response) with a thyrotrophin releasing hormone test, and low serum thyroxine concentration in a group of neonates whose mothers had had poorly controlled hyperthyroidism in the third trimester of pregnancy. The condition may last up to six months, as described in two case series.[13] Subclinical hyperthyroidism has no known associated adverse pregnancy outcomes.[5]

How does pregnancy affect hyperthyroidism?

A deterioration in previously diagnosed thyroid disease is not uncommon during the first trimester of pregnancy and may be due to an increase in the titre of TRAb concentrations or high levels of human chorionic gonadotrophin acting as a thyroid stimulator. Relapse may also be caused by impaired absorption of antithyroid medication secondary to vomiting that is associated with pregnancy or by reluctance to continue medication in the first trimester.[14]

Human immune regulation involves homoeostasis between T helper 1 (Th1) and T helper 2 (Th2) activity, with Th1 cells driving cellular immunity and Th2 cells humoral immunity. The immune status of pregnancy is a Th2 state, which allows tolerance of the fetus during pregnancy, and this is thought to be the reason why the severity of Graves' hyperthyroidism (and other autoimmune diseases) usually lessens after the first trimester.[1]

Hyperthyroidism before pregnancy may remit during pregnancy but will recur in the postpartum period as the immune status reverts to a Th1 state.

On rare occasions, labour, caesarean section, and infections may aggravate hyperthyroidism to the extent that cases of thyroid storm (a life threatening form of hyperthyroidism) have been observed.[15 16]

Drugs used in hyperthyroidism

Drug	Mode of action	Dose	Adverse effects
Propylthiouracil	Inhibits thyroxine synthesis; inhibits peripheral conversion of thyroxine to triiodothyronine	Starting: 300-450 mg/day; maintenance: 50-100 mg/day	Rash, fever, agranulocytosis
Carbimazole	Inhibits thyroxine synthesis	Starting: 15-40 mg/day; maintenance: 5-15 mg/day	As above, plus aplasia cutis and methimazole embryopathy
Propranolol	Reduces adrenergic symptoms	10-40 mg, 3-4 times/day (short term use only)	Bronchospasm, intrauterine growth restriction, neonatal hypoglycaemia

How is hyperthyroidism treated in pregnancy?

Prepregnancy planning and counselling

Ideally, a woman who knows she has hyperthyroidism should seek prepregnancy advice, although no evidence exists yet for the benefit of this.

Patients already treated for hyperthyroidism caused by Graves' disease

Although patients who have already been treated for hyperthyroidism may have received antithyroid drugs, had surgery, or had radioiodine therapy and be euthyroid (whether receiving thyroxine or not), neonatal hyperthyroidism may still occur. TRAb concentration should be measured early in pregnancy in a euthyroid pregnant women who has previously had surgery or radioiodine therapy.[17] If the concentration is high at this time, the fetus should be evaluated carefully during gestation (with serial ultrasonography) and the antibodies measured again in the third trimester. If the TRAb concentration is high at 36 weeks, the neonate needs to be checked for hyperthyroidism after delivery.

Treatment of hyperthyroidism in pregnancy

Box 3 outlines the main elements in managing hyperthyroidism in a pregnant woman. At all stages of pregnancy antithyroid drugs are the preferred treatment (table).[2] Radioiodine is contraindicated (box 4) and surgery requires pretreatment with antithyroid drugs to render the patient euthyroid.

The thionamides carbimazole, methimazole (the metabolite of carbimazole), and propylthiouracil are all effective in inhibiting thyroidal biosynthesis of thyroxine during pregnancy. Propylthiouracil is the preferred drug in pregnancy as carbimazole and methimazole are (albeit rarely) associated with teratogenic effects.

An early study also reported less placental transfer of propylthiouracil than of methimazole, but results of a more recent study measuring propylthiouracil and methimazole concentrations and examining placental perfusion in vitro have not shown any advantage for propylthiouracil in relation to placental transport. Furthermore, the two drugs seem to have no difference in effect on fetal and neonatal thyroid function.

This use of propylthiouracil as the initial preferred drug for maternal hyperthyroidism is an expert consensus recommendation of the Endocrine Society.[2] In countries where propylthiouracil is not available, carbimazole and methimazole are

acceptable as the potential fetal and maternal dangers of not treating active hyperthyroidism far outweigh the small risk of rare congenital abnormalities.

Aside from potential induction of hypothyroidism—and the noted possible teratogenic effects—several studies have shown that no long term adverse effects result from exposure to antithyroid drugs in utero, in particular on IQ scores or psychomotor development in individuals exposed to methimazole and propylthiouracil who were evaluated up to the age of 23 years.[19]

The starting dose of propylthiouracil is relatively high, 300-450 mg a day, up to 600 mg daily if necessary, given in two to three divided doses.[20] Some improvement is usually seen after one week of treatment with antithyroid drugs, but four to six weeks may be needed for a full effect. Once the hyperthyroidism has been controlled, the dose needs to be gradually reduced by a quarter to a third every three to four weeks, typically to 50-100 mg twice daily. The main principle of treatment is to administer the lowest dose of antithyroid drugs needed for controlling clinical symptoms, with the aim of restoring normal maternal thyroid function but ensuring that fetal thyroid function is minimally affected.

Of seven published studies examining whether a relation exists between dose of antithyroid drug and neonatal thyroid function, the dose of the drug correlated with neonatal thyroid function in three studies but did not correlate in a further four. Furthermore, even doses as low as propylthiouracil 100 mg a day have been reported to cause mild transient fetal hypothyroidism. Current maternal thyroid status rather than dose of antithyroid drug has therefore been suggested as the most reliable marker for titration of antithyroid drug treatment to avoid fetal hypothyroidism. In practice, to avoid fetal hypothyroidism, maternal free thyroxine concentrations should be kept in the upper third of the normal reference range for non-pregnant women, as with this management serum free thyroxine concentrations are normal in more than 90% of neonates.

Box 3 Management of hyperthyroidism in pregnancy

- Confirm diagnosis
- Discuss treatment with patient (effect on patient, effect on fetus, breast feeding)
- Start propylthiouracil
- Render patient euthyroid—continue with low dose of an antithyroid drug up to and during labour
- Monitor thyroid function regularly during gestation (every four to six weeks) and adjust the dose of antithyroid drug if necessary
- Do serial ultrasonography of the fetus
- Check TRAb at 30-36 weeks' gestation if hyperthyroidism is caused by Graves'disease
- Inform the paediatrician that the woman has hyperthyroidism and that the neonate may therefore be at risk of hyperthyroidism
- Review management postpartum—check for exacerbation
- Check infant for thyroid dysfunction if indicated

Box 4 Hyperthyroidism inadvertently treated with radioiodine in early gestation

Administration of radioactive iodine (iodine-131), either for diagnostic tests or treatment, is contraindicated during pregnancy, and all women who could potentially become pregnant should have a pregnancy test before being given ^{131}I.[18]

In many clinics, however, routine pregnancy testing is not done before administration of ^{131}I. Despite denial of pregnancy, several reports of inappropriate administration of radioiodine have highlighted the concern about the fetal radiation risk.

Because fetal thyroid uptake of ^{131}I starts after 12 weeks' gestation, exposure before 12 weeks is not associated with fetal thyroid dysfunction. Administration of up to 555 MBq ^{131}I for hyperthyroidism during the first trimester therefore does not compromise fetal thyroid function, and the low fetal whole body irradiation is not considered sufficient to justify termination of pregnancy.

However, the fetal thyroid concentrates iodine after 13-15 weeks' gestation and is relatively more avid for iodine than the maternal thyroid; in addition, the fetal tissues are more radiosensitive. ^{131}I given after this gestational age therefore potentially leads to substantial radiation to the fetal thyroid, resulting in biochemical hypothyroidism and even cretinism in the neonate. However, the likelihood of these effects is not certain, and in these circumstances dosimetry studies should be done to enable more accurate patient counselling with regard to, for example, termination of pregnancy.

If the pregnancy continues to term, intrauterine hypothyroidism may be diagnosed by umbilical cord sampling. Management should maintain high normal maternal circulating thyroxine levels. The neonate should be evaluated at birth specifically for hypothyroidism and for malformations that are more common with higher doses of radiation. The neonate should be treated promptly with thyroxine as appropriate; treatment may need to be lifelong.[2]

The administration of levothyroxine together with propylthiouracil as a "block and replace" regimen is not advisable in pregnancy as the amount of antithyroid drug may be excessive in proportion to the amount of thyroxine that crosses the placenta, resulting in fetal goitre and hypothyroidism.

No consensus has been reached on the duration of antithyroid drug treatment during pregnancy as no good level of evidence exists. Some authorities suggest stopping the drug in the third trimester or after four to 12 weeks of treatment with subsequent close monitoring, but relapse of disease may occur, which is most undesirable during labour. Therefore we believe that the drug should be continued in a low dose up to and during labour.

β-adrenergic blocking agents such as propranolol may be used for a few weeks to ameliorate the peripheral sympathomimetic actions of excess thyroid hormone, but prolonged use can result in restricted

SCENARIO

A 35 year old woman develops Graves' hyperthyroidism (the commonest cause of hyperthyroidism) four months after the birth of her second child. She receives treatment with antithyroid drugs for six months. In her third pregnancy she complains of palpitations, excessive sweating, and heat intolerance at 16 weeks' gestation. Although she experienced these symptoms in previous pregnancies, the current symptoms are much worse.

She is found to be severely hyperthyroid, with raised concentrations of serum free thyroxine (51.7 pmol/l (normal range 9.8-23.1 pmol/l) and free triiodothyronine (19.9 pmol/l (3.5-6.5 pmol/l)) and with suppressed concentrations of thyrotrophin (thyroid stimulating hormone) (<0.02 mU/l (0.35-5.5 mU/l)). She is treated with propylthiouracil, initially 150 mg three times daily, which is reduced eventually to 50 mg twice daily as she becomes euthyroid. Thyrotrophin receptor antibodies are measured at 30 weeks' gestation and are negative. Propylthiouracil is continued throughout pregnancy and she breast feeds while taking the drug. The drug is stopped two months postpartum; thyroid function is normal three weeks later.

fetal growth, impaired response to hypoxic stress, postnatal bradycardia, and hypoglycaemia.[21]

Fetal surveillance

Because of the risk of fetal thyroid dysfunction in women with raised TRAb concentration or those taking antithyroid drugs, serial ultrasound scans of the fetus should be performed. Ultrasound evidence of fetal thyroid disease includes intrauterine growth restriction, tachycardia, cardiac failure, hydrops, advanced bone age, and goitre.

If fetal hyperthyroidism is diagnosed, treatment involves modulation of maternal antithyroid drugs. If fetal hypothyroidism has resulted from administration of antithyroid drugs to the mother, this treatment should be decreased or stopped and administration of intra-amniotic thyroxine considered.[22] Early delivery may need to be considered in the case of fetal thyroid dysfunction, depending on the gestation at diagnosis and the severity of fetal symptoms.[2]

Postpartum period

Breast feeding

Because propylthiouracil and methimazole are secreted in human milk (the former less so because of its more extensive binding to albumin), concerns have been expressed in the past about the safety of breast feeding in women taking antithyroid drugs. However, only limited quantities of propylthiouracil and carbimazole are now known to be concentrated into milk. As long as the doses of methimazole or propylthiouracil can be kept moderate (propylthiouracil <250-300 mg a day, methimazole <20 mg a day), the risk for the infant is negligible, and no evidence based argument exists to advise mothers against breast feeding when they take an antithyroid drug.[23] It is prudent to monitor periodically the infant's thyroid function while the mother is taking antithyroid drugs, although a recent reassuring study showed that thyroid function in breastfed infants was not affected, even when antithyroid drugs induced maternal hypothyroidism. The mother should also be advised to take her medication after a feed.[2]

METHODS

We searched Medline and the Cochrane database of systematic reviews for studies evaluating the diagnosis and treatment of hyperthyroidism in pregnancy using the key words "hyperthyroidism", "Graves' disease", "pregnancy", "management", and "antithyroid drugs".

Maternal and neonatal follow-up

The mother has a significant risk of exacerbation of hyperthyroidism postpartum, and thyroid function should be checked at 6 weeks and 3 months post delivery.

If a high TRAb concentration has been noted at 30 weeks,[17] the neonate should be tested for hyperthyroidism after six hours and, if the test is positive, carbimazole should be started. If the mother has been taking an antithyroid drug up until delivery, the baby should be screened again several days later as he or she may well be euthyroid at birth but develop hyperthyroidism as the antithyroid drug is metabolised. The mother can be reassured that the disease in her baby will be self limiting to about three months because of the disappearance of the thyrotrophin receptor stimulating antibodies in the baby during this time.[11]

Conclusion

Although untreated hyperthyroidism has potentially serious adverse effects on the mother and fetus, when treated promptly and monitored appropriately, the outcome for mother and fetus can be excellent.

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THE COMPETENT NOVICE

Lifelong learning at work

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This series aims to help junior doctors in their daily tasks and is based on selected topics from the UK core curriculum for foundation years 1 and 2, the first two years after graduation from medical school.

The importance of lifelong learning in medicine is well recognised. This article explores how junior doctors can develop learning strategies for use throughout their working life

Most doctors are aware of the aphorism that learning should continue from the cradle to the grave. However, medical school does not prepare anyone fully for the responsibilities, long hours, and challenging tasks that qualification brings.[1 2] The fittest survive by learning from their practice, whereas those who do not continue to learn become dissatisfied and burn out.[3] Current restrictions in working hours, changes in the organisation of health care, shorter hospital stays, and the breakdown of the medical firm make it ever more necessary to learn efficiently from practice.[4]

Numerous editorials and descriptive articles have stated the importance of lifelong learning, but research has not yet provided a definitive answer to the question "How can trainees develop lifelong learning strategies?" We have drawn on medical education theory and empirical studies of both facilitating practice change and the effectiveness of feedback for best evidence on effective lifelong learning. Those sources show that one size does not fit all. Our article does not therefore offer quick fixes such as organising group learning sessions or purchasing a personal digital assistant, but rather it describes the continuous personal endeavour that lifelong learners face.

Lifelong learning: how best to do it

Learn in the workplace

Ideas about work based learning have moved from thinking of learners as solitary beings to thinking of them as members of "communities of practice"[5] in which interaction with other doctors, nurses, and allied professionals of various levels of seniority shapes their personal and professional development.[6] Learners, however, are sometimes more aware of lectures and courses as learning opportunities than they are of the "on the job" learning opportunities that they face all day. As a learner, you therefore need to capitalise on such opportunities by soliciting feedback, seeking out evidence of clinical effectiveness at the point of care, and participating fully in the educational activities of whichever community of practice you are currently working in.

Be in charge

To be an effective lifelong learner in the workplace, you have to make a conscious decision to direct your own development.[7] Research in continuing medical education shows that doctors learn most when they are motivated enough to identify their own learning needs and meet those needs at their own pace.[8 9] For trainees, that means turning workplace experiences into realistic and achievable learning objectives. When you finish rounds on the internal medicine ward, list the topics you need to learn more about; prioritise them, and write down one or two higher level learning objectives for the weeks to come; and set a date to finish them by. For example:

- You just saw four patients with diabetic complications; do you really know everything you need to know about regulating blood glucose? If not, focus on diabetes
- Do you feel defeated by that one patient who just didn't seem to understand your explanations? Focus on communicating with patients
- Are your rounds running late for the fourth time this week? Focus on time management