Best Practice & Research Clinical Obstetrics and Gynaecology 29 (2015) 700–706

Contents lists available at ScienceDirect

Best Practice & Research Clinical Obstetrics and Gynaecology

journal homepage: www.elsevier.com/locate/bpobgyn

9

Thyroid disorders during pregnancy and postpartum

Elizabeth N. Pearce, MD, MSc *

Section of Endocrinology, Diabetes, and Nutrition, Boston University School of Medicine, Boston, MA, USA

Keywords:
hypothyroidism
Graves' disease
thyrotoxicosis
postpartum thyroid disease
pregnancy

An awareness of the gestational changes to thyroid physiology and the impact of uncontrolled thyroid disease on pregnancy and infant outcome is essential for the successful management of hypothyroidism and hyperthyroidism. This review summarizes strategies for the management of thyroid disease in pregnancy and post partum, and it highlights areas where there is still a lack of consensus.
© 2015 Elsevier Ltd. All rights reserved.

Physiological changes to thyroid function in pregnancy

Maternal thyroid function changes throughout gestation. When evaluating measurements of thyroid function in pregnant women, it is important to keep in mind that nonpregnancy laboratory reference ranges do not apply. In the first trimester, serum human chorionic gonadotropin (hCG) acts as a stimulator of thyroidal thyrotropin receptors. Therefore, serum thyroid-stimulating hormone (TSH) levels are typically low when hCG levels are high, and they start to increase after 10–12 weeks of gestation, when the hCG levels fall [1]. Conversely, serum free T4 levels are highest when hCG levels are elevated and tend to fall later in pregnancy. High serum estrogen levels in pregnant women increase levels of thyroxine-binding globulin (TBG) and thus increase circulating total (not free) triiodothyronine (T3) and thyroxine (T4) levels, starting early in gestation [2].

The use of trimester-specific, assay-specific normal ranges for thyroid function is recommended. Where such ranges are not available, the following ranges for TSH may be used: 0.1–2.5 mIU/L in the first trimester, 0.2–3.0 mIU/L in the second trimester, and 0.3–3.0 mIU/L in the third trimester [3]. High serum TBG levels interfere with immunoassays for free T4 levels, making these assays unreliable in pregnant women [4]. The free thyroxine index, calculated from measurements of total T4 and T3 resin binding, may be more accurate in pregnancy than free T4 immunoassays [4,5], although this remains controversial. In general, TSH is the most sensitive indicator of thyroid status in pregnant women.

* Boston University School of Medicine, Section of Endocrinology, Diabetes, and Nutrition, 88 East Newton Street, H3600, Boston, MA 02118, USA. Tel.: +1 617 414 1348.
E-mail address: elizabeth.pearce@bmc.org.

http://dx.doi.org/10.1016/j.bpobgyn.2015.04.007
1521-6934/© 2015 Elsevier Ltd. All rights reserved.

Hyperthyroidism in pregnancy

Overt hyperthyroidism, which occurs in 0.1–0.4% of pregnant women, is defined as a serum TSH level below the trimester-specific reference range with elevated levels of T3 and/or free T4. Subclinical hyperthyroidism is defined as a serum TSH level below the trimester-specific reference range with normal peripheral thyroid hormone levels. Subclinical maternal hyperthyroidism has not been associated with adverse maternal or fetal outcomes [6], and treatment for this condition is not recommended.

Gestational thyrotoxicosis

Gestational thyrotoxicosis is the most frequent cause of hyperthyroidism in the first trimester. It is a transient form of thyrotoxicosis due to elevated serum hCG levels [7]. It often occurs in women with hyperemesis gravidarum (defined as severe nausea and vomiting with dehydration, the loss of 5% of body weight, and ketonuria). HCG concentrations correlate with the severity of nausea, and gestational thyrotoxicosis is unusual in women without clinically significant nausea and vomiting [8]. Gestational thyrotoxicosis is also frequent in twin or other multiple pregnancies, where serum hCG levels are especially elevated. Gestational thyrotoxicosis does not require antithyroid drug treatment, and it resolves spontaneously as hCG levels fall after week 10–12 of gestation [3,5,9]. Care is supportive, with hydration and antiemetics.

Graves' disease

Graves' disease is the most common cause of autoimmune hyperthyroidism in pregnancy; it may cause overt or subclinical hyperthyroidism. Symptoms such as fatigue, heat intolerance, and tachycardia are common to both pregnancy and to all forms of hyperthyroidism. Graves' disease can be distinguished from gestational thyrotoxicosis by the presence of a diffuse goiter, a history of hyperthyroid symptoms prior to pregnancy, or the presence of ophthalmopathy. Measurement of serum thyroperoxidase (TPO) antibodies and/or thyroid hormone receptor antibodies may help to confirm the diagnosis of Graves' disease.

Uncontrolled overt Graves' hyperthyroidism is associated with increased risks of miscarriage, stillbirth, preterm delivery, preeclampsia, low birth weight, intrauterine growth restriction, thyroid storm, and maternal congestive heart failure [10]. The antithyroid drugs propylthiouracil (PTU) and methimazole (MMI) block thyroid hormone synthesis, and they are the mainstay of Graves' disease therapy. In the UK and other regions, carbimazole (CBZ), a metabolite of MMI, is also available. Small amounts of PTU, CBZ, and MMI cross the placenta and may decrease fetal thyroid function [10]. Methimazole and carbimazole are associated with cutis aplasia and with a rare embryopathy consisting of choanal or esophageal atresia and dysmorphic facies [11,12]. First-trimester use of PTU has also been associated with birth defects such as urinary tract and face and neck malformations [13]. In addition, PTU has been associated with fulminant hepatotoxicity, including in pregnant women and their fetuses [14]. Although current guidelines recommend changing women from MMI/CBZ to PTU as soon as pregnancy is confirmed, and then changing back to MMI/CBZ after the first trimester [3,5], this approach has not been studied, and some consider that the balance of risks between the two classes of antithyroid drug does not warrant the potential loss of control of hyperthyroidism in the first trimester. Treatment with the lowest possible doses of antithyroid drugs should be employed to keep the free T4 of pregnant women in the high-normal to slightly thyrotoxic range [3,5].

In women initiated on antithyroid drugs during pregnancy, serum TSH and free T4 should be assessed every 2–4 weeks until euthyroidism is achieved, and every 4–8 weeks thereafter. In up to a third of women, Graves' disease remits spontaneously in the last trimester of pregnancy, and antithyroid drugs can frequently be discontinued [15]. Short-term use of propranolol will improve thyrotoxic symptoms until hyperthyroidism is controlled [3,5,10]. In women who are unable to tolerate antithyroid drugs, thyroidectomy may be required for control of Graves' hyperthyroidism. Thyroidectomy is safest in the second trimester.

Download English Version:

https://daneshyari.com/en/article/3907204

Download Persian Version:

https://daneshyari.com/article/3907204

Daneshyari.com