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Revisiones

Thyroid dysfunction in pregnancy

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DISFUNCIÓN TIROIDEA EN EL EMBARAZO

El embarazo comporta una serie de cambios hormonales e inmunológicos que dan lugar a modificaciones en la fisiología normal del tiroides. Por tanto, la evaluación de la función tiroidea durante el embarazo debe interpretarse teniendo en cuenta estos cambios. En nuestra opinión, la gran prevalencia de trastornos tiroideos asociados al embarazo y las graves consecuencias que pueden causar a la madre y el feto apoyan la necesidad de realizar pruebas de cribado de disfunción tiroidea de manera sistemática, tanto antes como durante el embarazo. Una vez se ha diagnosticado una disfunción tiroidea será necesario realizar una monitorización frecuente para ajustar el tratamiento de forma precisa. El objetivo del tratamiento del hipertiroidismo con fármacos antitiroideos es lograr que la concentración de tiroxina sérica (T4) se mantenga en el límite alto del rango normal (T4 libre, 2-2,5 ng/dl; T4 total, 12-18 µg/dl) con la mínima dosis posible, mientras que, en el caso del hipotiroidismo, el objetivo es conseguir que la concentración de tirotropina se mantenga entre 0,5 y 2,5 mU/l.

Palabras clave: Embarazo. Hipertiroidismo. Hipotiroidismo. Posparto. Manejo.

Pregnancy induces complex hormonal and immunological changes that modify normal thyroid physiology. Therefore, evaluation of thyroid function during pregnancy should be interpreted according to these changes. In our opinion, the high prevalence of pregnancy-related thyroid disorders and their important consequences for both mother and fetus indicate the need for routine thyroid function screening both before and during pregnancy. Once thyroid dysfunction is diagnosed, the management of the disorder requires frequent monitoring to adjust treatment accurately. The goal of treating hyperthyroidism with thionamide drugs is to maintain serum thyroxin (T4) in the upper normal range (free T4, 2-2.5 ng/dl; total T4, 12.0-18.0 µg/dl) using the lowest possible dose of the drug, while in hypothyroidism the goal is to return serum thyrotropin to the range between 0.5 and 2.5 mU/l.

Key words: Pregnancy. Hyperthyroidism. Hypothyroidism. Postpartum. Management.

INTRODUCTION

Pregnancy is a unique situation in which the physician is faced with at least two interactive patients. Any medical action or inaction may have positive or negative consequences for both the mother and the fetus.

Maternal physiological changes during pregnancy

The physiological changes that occur in normal pregnancy have important repercussions for the thyroid gland[1,2]. Usually thyroid gland volume enlarges and thyroid hormone production increases approximately 50% above the preconception baseline. These changes are secondary to a variety of factors.

Human chorionic gonadotrophin. Rising plasma levels of placental human chorionic gonadotrophin (hCG), which has a weak thyrotropin (TSH) agonist action due to the structural homology

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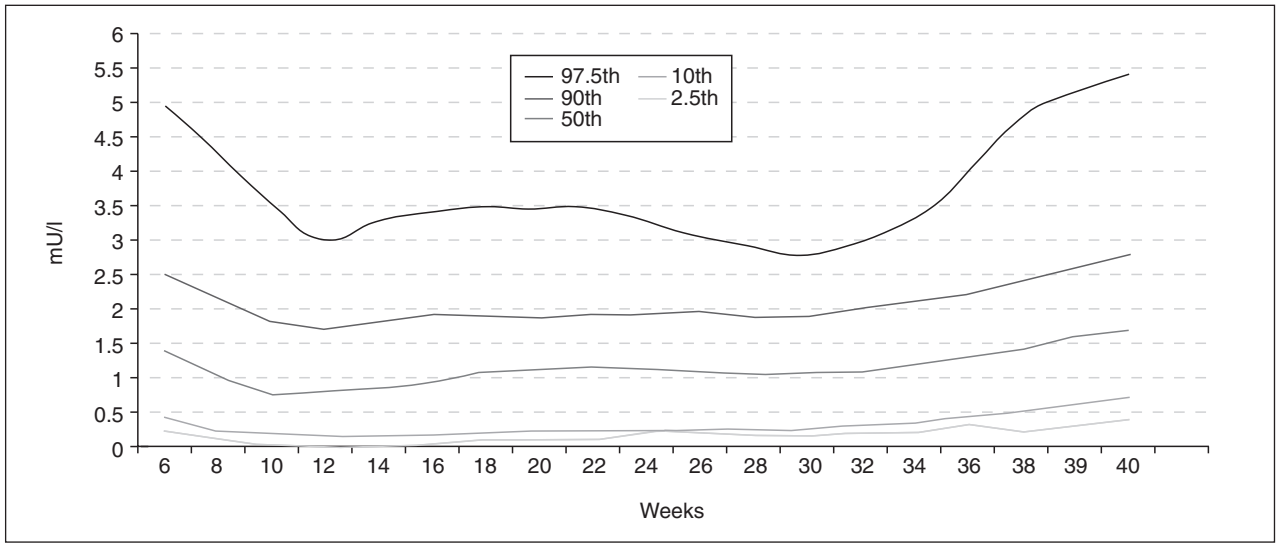

Fig. 1. Thyroid-stimulating hormone (TSH) plasma concentration expressed in percentiles according to gestational age. TSH was measured in 13,599 singleton pregnancies. Gestational age-specific. Modified from Dashe et al[33].

between both hormones, have a major thyroid stimulatory influence. hCG increases during the first trimester and plateaus from midgestation to shortly after delivery[3]. The result of this hCG activity is an elevation in serum thyroxine (T4) and triiodothyronine (T3) concentrations and suppression of serum TSH, providing new normal ranges unique to pregnancy (fig. 1).

Thyroid binding globulin. Plasma oestrogen levels rise in pregnancy and induce an elevation of up to 100% in serum thyroid binding globulin (TBG). This occurs mainly during the first 20 weeks and is secondary to an extended half-life because of changes in TBG glycosylation[4]. As a result, by approximately week 10 of pregnancy the total serum T4 (TT4) is elevated by up to 50%, remaining constant at this level until delivery[5]. This large increase in TBG opens many T4 binding sites, which have to be filled to maintain free T4 equilibrium and, therefore, constitute another cause of increased thyroid hormone secretion. These ongoing changes in TBG have made assessment of free thyroid hormone levels in pregnancy a technical challenge, resulting in a polluted literature in which cross-sectional studies have suggested that free T4 (FT4) levels during the first trimester may be higher, lower or the same as those before conception (fig. 2).

Other important factors influencing thyroid function. During pregnancy, other physiological adjustments take place in maternal thyroid homeostasis which, together, may lead to incremental increases in thyroid hormone synthesis. The maternal glomerular filtration rate is also elevated secondary to increased cardiac output, resulting in high renal clearance and iodide excretion[6]. Therefore, iodine intake needs to be increased to accommodate the continuing thyroid hormone synthesis. In addition, transplacental passage of T4 may also stimulate the maternal thyroid by depleting maternal circulating T4[7].

Immune changes. Pregnancy is a time of placenta-induced immune suppression secondary to placental cytokine and hormone secretion resulting in enhanced regulatory T cell function[8,9]. This particular situation can be extremely important to autoimmune reactions and most autoimmune diseases, including thyroid disorders, tend to improve during gestation[10].

Maternal thyroid function is totally reset to normal activity by 6 months after delivery unless thyroid dysfunction develops[11].

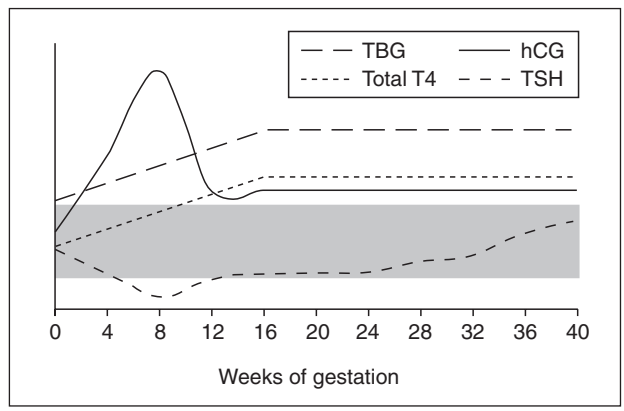

Fig. 2. Changes in plasma concentrations of thyroid function test and hCG according to the evolution of pregnancy. The shaded area corresponds to the normal range in non-pregnant women. hCG: human chorionic gonadotrophin; TBG: thyroid-binding globulin; T4: thyroxine; TSH: thyroid-stimulating hormone. Modified from Brent GA. Maternal thyroid function: interpretation of thyroid function tests in pregnancy. Clin Obstet Gynecol. 1997;40:3-15.