Autoimmune thyroid disease and insulin-dependent diabetes mellitus during pregnancy and \textit{post partum}

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\textbf{Summary.} - The aim of this study was to assess thyroid dysfunction and autoimmunity in pregnant insulin-dependent diabetes mellitus (IDDM) women during pregnancy and early \textit{post partum}. Fifteen pregnant IDDM women and 77 healthy pregnant women were studied. Free T4, TSH, TPO-Ab and Tg-Ab were assayed during the first and third trimester of pregnancy and 3 months \textit{post partum}. In IDDM women FT4 levels significantly decreased (p < 0.05) during third trimester and 3 months \textit{post partum} and also TPO-Ab during third trimester (p < 0.01). 26% of IDDM and 4% of the controls presented \textit{post partum} thyroid dysfunction. We recommend that pre-pregnant IDDM be screened for TPO-Ab. Those with a positive result would be followed with serial monitoring of free T4 and TSH levels during each trimester as well as during the \textit{post partum} period.

\textit{Key words:} pregnancy, \textit{post partum}, IDDM, thyroid dysfunction, antithyroid peroxidase antibodies.

\textbf{Riassunto} (Malattia autoimmune della tiroide e diabete mellito insulino-dipendente in gravidanza e nel post partum). - Gli scopi dello studio sono stati quelli di valutare le disfunzioni della tiroide e i fenomeni autoimmunitari nelle donne diabetiche con diabete mellito insulino-dipendente (IDDM) durante la gravidanza e nel post partum. Sono state studiate 15 gravidhe IDDM e 77 gravidhe normali. Sono stati valutati l'FT4, TSH, TPO-Ab e Tg-Ab al 1\textsuperscript{a} e 3\textsuperscript{a} trimestre di gravidanza e 3 mesi dopo il parto. Nelle IDDM i livelli di FT4 erano significativamente (p < 0.05) ridotti al 3\textsuperscript{a} trimestre di gravidanza e dopo il parto, così come gli TPO-Ab nel 3\textsuperscript{a} trimestre (p < 0.01). Nel post partum il 26% delle IDDM e il 4% dei controlli mostravano disfunzioni tiroidee. I soggetti con risultati positivi dovrebbero essere seguiti con un monitoraggio serio di FT4 e TSH durante ogni trimestre di gravidanza così come dopo il parto.


\textbf{Introduction}

Insulin-dependent diabetes mellitus (IDDM) is commonly associated with other autoimmune endocrine disorders. Thyroid autoantibodies occur more frequently in IDDM patients than in the normal population [1]. Furthermore, an increased prevalence of subclinical hypothyroidism has been reported in pregnant diabetic women [2]. Several studies have found a wide range (10-25\%) in the prevalence of \textit{post partum} thyroid dysfunction in IDDM patients [3-5]. Thyroid peroxidase (TPO) is now recognized as the specific antigen of the thyroid microsome, and a new sensitive method for detection of antibodies to TPO (TPO-Ab) has been developed [6]. The aim of the present investigation was to assess in a prospective sequential study the autoimmunity and the thyroid function during pregnancy and \textit{post partum} period.

\textbf{Materials and methods}

Fifteen pregnant women selected from the Obstetric-Endocrine Unit at the University Hospital of Granada, with a diagnosis of IDDM and without a history of thyroid disease participated in this prospective study. The mean age (± SD) was 25 ± 6 years and duration of diabetes was 18 ± 5 years. 77 non diabetic pregnant served as control group (mean age: 26 ± 7 years) [7]. All patients completed the study. Informed consent was obtained from all patients. The clinical protocol was approved by the Hospital Ethics Committee. All IDDM patients and controls were evaluated before gestation, during the first and third trimester of pregnancy, and 3 months \textit{post partum}. Tests included serum free T4 (FT4) (normal range for non pregnant: 0.8-2.2 ng/dl), serum thyroid stimulating hormone (TSH) by a sensitive immuno-radiometric assay (normal range: 0.4-4.0
mU/l). Antithyroid peroxidase antibodies (TPO-Ab) were measured by RIA and were considered positive titers at > 150 U/ml. Antithyroglobulin antibodies were measured by RIA and were considered positive titers at > 200 U/ml.

**Statistical analysis**

Data were analyzed using Mann-Whitney’s U-test. SPSS software was used for statistical analyses. The level of significance was set at 0.05.

**Results**

The prevalence of positive TPO-Ab in IDDM women in the first and third trimester was 33% and slightly higher at three months post partum (40%). These percentages were significantly different (p < 0.01) if compared with those of non diabetic pregnant (7.6%, 9% and 8.5% respectively).

Table 1 details thyroid function and autoimmunity of IDDM and control group. FT4 was significantly (p < 0.05) decreased in IDDM patients when compared with controls during the third trimester (mean 0.9 vs 1.6) and 3 months post partum (mean 1.0 vs 1.4). There were no significant differences in TSH levels, TPO-Ab and Tg-Ab between groups during pregnancy and post partum. Three IDDM patients (20%) developed TSH levels higher than 10 mU/l and required therapy with thyroid. In the early post partum period, 4/15 (26%) of the IDDM patients and 3/77 (4%) of the control group developed thyroid dysfunction.

**Conclusions**

The present study concerned two groups of pregnant women with IDDM and women without IDDM before gestation. We studied these women sequentially during pregnancy and in the early post partum period to determine whether the presence of IDDM is associated with changes in thyroid function and autoimmunity.

We showed previously that the prevalence of a positive test for TPO-Ab is increased in patients with type 1 diabetes mellitus and we reported a prevalence of 38% in our study population, with a higher prevalence in women than in men [8]. Our study in IDDM women shows a 4-fold increase in thyroid antibody positivity compared to the autoantibody incidence of 8.7% previously found by our group in healthy subjects [9]. Other studies agree. For example, Cardoso et al. [10] in an African diabetic population found that 46% of the IDDM patients had significant levels of serum thyroid autoantibodies. Prina et al. [11] found a much higher prevalence of thyroid disease in IDDM (23.4%) than expected for a normal population (3.5%) and they showed that young age at onset of IDDM seems to be significantly associated with the development of autoimmune thyroid disease.

We found a high prevalence of TPO-Ab positive in IDDM during the first trimester which is maintained throughout gestation. During pregnancy, despite a reduction in TPO-Ab titers, serum FT4 significantly decreased in IDDM patients and three patients needed thyroid therapy. None of the patients in the control group evidenced thyroid dysfunction requiring therapy during the study. In the early post partum the prevalence of thyroid dysfunction was also different when we compared patients with IDDM and nondiabetic women (26% vs 4% respectively).

**Table 1** - Thyroid function and thyroid antibody, first, three trimester of gestation and three months early post partum in IDDM patients and control group

<table>
<thead>
<tr>
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<th>1st trim.</th>
<th>3rd trim.</th>
<th>Post partum</th>
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<tbody>
<tr>
<td></td>
<td>TSH</td>
<td>FT4</td>
<td>TPO-Ab</td>
</tr>
<tr>
<td>IDDM</td>
<td>1.3 ± 0.9</td>
<td>1.1 ± 0.5</td>
<td>1424 ± 2400</td>
</tr>
<tr>
<td>Control</td>
<td>1.5 ± 0.9</td>
<td>1.2 ± 0.5</td>
<td>106 ± 370</td>
</tr>
<tr>
<td>IDDM</td>
<td>1.8 ± 1.2</td>
<td>1.0 ± 0.3</td>
<td>534 ± 603</td>
</tr>
<tr>
<td>Control</td>
<td>1.2 ± 0.7</td>
<td>1.4 ± 0.5</td>
<td></td>
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</table>

(*) p < 0.05 IDDM vs control. Results are expressed as mean ± SD.
IDDM: insulin-dependent diabetes mellitus.
Glioyer et al. [12] recently showed that women with asymptomatic autoimmune thyroid disease who are euthyroid in early pregnancy carry a significant and progressive risk of becoming hypothyroid during gestation, despite a marked reduction in antibody titers as was also seen in our study. Progression to subclinical hypothyroidism was associated with and predicted by serum TSH levels and TPO-Ab titers in the first trimester.

In conclusion, we would recommend that all pre-pregnant IDDM be screened for anti TPO-Ab. Those with a positive result should be followed with serial monitoring of free T4 and TSH levels during each trimester as well as during the post partum period.

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Influences of thyroid diseases in diabetic pregnant women

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Summary. - Thyroid disorders are particularly frequent in women and, second to diabetes mellitus, are the most common endocrine diseases during pregnancy. An association between insulin-dependent diabetes mellitus and thyroid autoimmunity has long been recognized. Management of thyroid diseases in pregnancy is different than in non-pregnant women, due to physiological changes of thyroid hormone economy in the childbearing period. Thyroid dysfunction may affect carbohydrate metabolism and worsen glucose control in diabetic patients. On the other hand, poorly compensated diabetes mellitus may cause alteration in the production and metabolism of thyroid hormones. Pregnant women with insulin-dependent diabetes mellitus have an increased risk of developing post partum thyroiditis. These observations have lead to the recommendation that thyroid function should be checked in diabetic women during pregnancy and in the post partum.

Key words: thyroid disease, diabetes mellitus, euthyroid sick-syndrome, post partum thyroiditis.

Riassunto (Malattie tiroidee e gravidanza nella donna diabetica). - Le tiroepatie sono più frequenti nel sesso femminile e, dopo il diabete mellito, sono le endocrinopatie più comuni durante la gravidanza. La frequente associazione tra tiroepatie autoimmune e diabete mellito insulinodipendente è conosciuta da tempo. La diagnosi ed il trattamento delle malattie tiroidee in gravidanza hanno aspetti particolari a causa delle fisiologiche modificazioni dell'economia ormonale tiroidea che intervengono in questa condizione. Le disfunzioni tiroidee possono influenzare il metabolismo glucidico e peggiorare il controllo della glicemia nella donna diabetica. A sua volta, il diabete mellito scompenso può modificare la produzione ed il metabolismo degli ormoni tiroidei. Le donne affette da diabete mellito insulinodipendente presentano un aumentato rischio di sviluppare una tiroideite post partum. Per questi motivi è opportuno controllare la funzione tiroidea nelle donne diabetiche durante la gravidanza e nei primi mesi dopo il parto.

Parole chiave: malattie tiroidee, diabete mellito, sindrome da bassa T3, tiroideite post partum.

Introduction

Pregnancy leads to complex metabolic changes in the mother, which affect the function of her endocrine system. Altered carbohydrate homeostasis may cause overt diabetes or abnormal glucose tolerance in predisposed individuals. Pregnancy also affects thyroid function and thyroid hormone metabolism. Therefore, the interaction of pregnancy and diabetes mellitus or thyroid disease requires special consideration because of the increased risks to the well-being of the mother and the fetus. Several aspects of diagnosis and treatment of diabetes mellitus and thyroid disease are altered during pregnancy.

Thyroid diseases are common in the general population [1], and an association between diabetes mellitus and thyroid diseases has long been recognized [2]. Thyroid disorders are particularly frequent in women and, second to diabetes mellitus, are the most common endocrine diseases during the childbearing period. Therefore, it is important to understand the interaction of thyroid disease and diabetes mellitus in the care of pregnant women affected by both disorders.

Effects of pregnancy on thyroid function

Several changes in thyroid homeostasis occur during normal pregnancy [3]. The concentration of thyroxine binding globulin (TBG) in maternal serum rises early in pregnancy and is doubled by the end of the first trimester. TBG is the major thyroid hormone transporting protein, and its increase produces an elevation of total serum thyroxine (TT4) and triiodothyronine (TT3) concentrations, while free thyroid hormone levels (FT4 and FT3) remain within the range of non-pregnant women. Elevated serum concentrations of TBG and the increased plasma volume of pregnancy result in an increase in the total T4 pool. In order to maintain serum free T4 levels within the normal range, T4 production rate must increase to allow additional T4 to accumulate. Placenta is rich of the type 3 iodothyronine-monodeiodinase, an enzyme which degrades both T4 and T3. The accelerated degradation of thyroid hormones by placenta is an additional factor requiring an increased production rate of thyroid hormones. Human chorionic gonadotropin (HCG) is a glycoprotein secreted by the placental syncytiotrophoblast. HCG has a weak intrinsic thyroid-
stimulating activity due to its structural similarity to thyroid stimulating hormone (TSH). Because of the extremely high concentrations of HCG reached during the first trimester of pregnancy, minor elevations of FT4 and FT3 can be demonstrated in this stage, and these correlate with the rise in HCG and a fall in TSH levels.

During pregnancy, the increased renal blood flow and glomerular filtration lead to an increase in the urinary clearance of iodine. In addition there is an augmented iodine requirement due to the transplacental transport of iodine which is necessary for fetal thyroid function. In areas where the iodine intake is low or borderline low, the decreased availability of iodine to the mother may result in a state of relative iodine deficiency, and lead to pathological changes. Overall, the increased T4 requirement and the reduced availability of iodine during pregnancy may induce goiter appearance, and precipitate or aggravate maternal hypothyroidism [4]. Therefore, pregnancy justifies the monitoring of thyroid function and thyroid volume in areas with moderate iodine deficiency, including many Mediterranean countries.

**Hypothyroidism and pregnancy**

The main causes of hypothyroidism in pregnancy are Hashimoto’s thyroiditis either goitrous or atrophic (idiopathic myxedema), postabortal hypothyroidism in Graves’ disease, surgical hypothyroidism, and iodine deficiency [5].

Although the presence of hypothyroidism has been associated with successful pregnancies to term, the rates of abortion and stillbirth are doubled in untreated maternal hypothyroidism. Various studies reported up to a 20% incidence of perinatal mortality, and congenital malformations associated with maternal hypothyroidism. Up to 60% of surviving children had impaired mental and somatic development. Overtly hypothyroid women experienced a 20% to 40% incidence of maternal complications, including anemia, preeclampsia, placental abruption, and *post partum* hemorrhage, with low fetal birth weight rating 30% and fetal death 12%. Women with subclinical hypothyroidism had about one third as many of the same problems [5].

Maternal hypothyroidism during early embryogenesis can also impair the neuropsychological development of the fetus. Before the onset of fetal thyroid function, that in human beings occurs at about 12 weeks of gestation, the fetus is dependent on the placental transfer of maternal thyroid hormone for normal development. Therefore, maternal hypothyroidism in the first trimester can result in a decreased availability of thyroid hormone during the initial phases of gross brain development and during the spurt in forebrain neuroblast proliferation. As a consequence, mental retardation, deafness and spasticity, are often found in infants born to hypothyroid mothers, despite apparently normal thyroid function [3]. A much-improved pregnancy outcome is observed in hypothyroid women after thyroid replacement therapy [6]. Evidence was also provided that optimal replacement therapy of hypothyroid mothers reduces the rate of congenital anomaly in the offspring.

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**Hyperthyroidism and pregnancy**

Thyrotoxicosis is diagnosed in between 1 in 500 and 1 in 1500 pregnancies, and is due in 95% of cases to Graves’ disease; toxic nodular goiter is much less common in this age group; in addition there is a spectrum of thyrotoxic disorders during pregnancy related to HCG such as gestational trophoblastic disease and hyperemesis gravidarum [5].

There is a higher incidence (5:1-6:1) of spontaneous abortion, preterm delivery, lower birth-weight infants and neonatal mortality in pregnancies complicated by untreated maternal hyperthyroidism. In addition, hyperthyroidism may precipitate heart failure, and thyroid storm may occur during labor and delivery in a thyrotoxic woman. All these complications are much less frequent when thyrotoxicosis is recognized and treated before pregnancy as opposed to during pregnancy. Nevertheless control of maternal thyrotoxicosis at any time during gestation is critical to ameliorate the outcome of pregnancy. Mothers rendered euthyroid with methimazole have the same low rate of fetal anomalies seen in euthyroid, untreated mothers. In contrast, hyperthyroid, untreated mothers have a higher incidence of fetal anomalies [7]. These findings suggest that uncontrolled maternal hyperthyroidism may cause fetal malformations and that the benefit of methimazole outweighs any possible teratogenic effect.

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**Thyroid dysfunction in diabetic patients**

The prevalence of thyroid dysfunction (hypo- and hyperthyroidism) in diabetic patients ranges between 13 to 33% in various studies, and it is higher than in the normal population (6-6 %) [2]. The likely explanation is the increased frequency of autoimmune thyroid diseases (Graves’ disease and Hashimoto’s thyroiditis) in patients with insulin-dependent diabetes mellitus (IDDM), as a result of the genetic predisposition to both disorders which is described as type 2 polyendocrine autoimmunity syndrome. Many studies have documented an increased frequency of circulating organ specific antibodies in patients with IDDM and their first degree relatives. Genetic evidence also indicate that an association with histocompatibility antigens HL-A8 and HLA-DR3 is common in IDDM, autoimmune thyroid diseases and Addison disease. Serum anti-thyroid antibodies (anti-
thyroglobulin and anti-thyroperoxidase antibodies) are more frequent in IDDM patients than in the general population. The prevalence of anti-thyroid antibodies varies between 6 and 30% in different studies and it is higher in white than in black patients [8, 9]. In a Scottish study of adult diabetics, female patients with IDDM had the highest incidence of thyroid dysfunction (3.2%) [2] while the incidence of thyroid dysfunction in the general female population studied in the Whickham survey was 0.5% [10]. An association between thyroid disease and non insulin-dependent diabetes mellitus (NIDDM) has also been reported [2]. This may be due to the fact that the older age of NIDDM patients is associated to an increased risk of thyroid disease. There is also the possibility that some NIDDM patients actually have IDDM of very slow-onset, and, similarly to juvenile IDDM patients, share a genetic predisposition towards autoimmune diseases. Based on these epidemiological data it has been suggested that thyroid function should be screened annually in diabetic patients to detect asymptomatic thyroid disease.

Diagnosis of thyroid disease in diabetic pregnant women

Thyroid dysfunction in pregnancy requires prompt diagnosis and specific therapy. From a clinical point of view, several important aspects should be considered in a diabetic pregnant woman suspected to have thyroid disease.

Pregnancy and thyroid function

The clinical and laboratory diagnosis of thyroid disease in pregnancy is complicated by several physiologic events. Pregnancy produces an increased thyroid hormone requirement in the mother while the availability of iodine is reduced. As a consequence, thyroid volume may increase, particularly in women with marginally low iodine intake. Furthermore, during the first trimester of gestation there is a marked rise in circulating levels of TBG, in response to hyperestrogenism. This change of the circulating transport protein produces a rise of serum levels of TT4 and TT3 while the concentrations of free thyroid hormones remain in the normal range. Finally, pregnancy is characterized by an increased basal metabolic rate with raised cardiac output, tachycardia, heat intolerance and skin warmth. These manifestations can mimick thyrotoxicosis.

Thyroid function and glucose metabolism

Thyroid dysfunction may affect carbohydrate metabolism and worsen glucose control, precipitating severe ketoacidosis or hypoglycemia. The effect of thyroid hormones at various levels of glucose metabolism has long been recognized [11]. Thyroid hormones increase glucose absorption from the gastrointestinal tract, enhance the utilization of carbohydrates in extrahepatic tissues, enhance hepatic gluconeogenesis, deplete glycogen stores, and increase insulin degradation. As a result, thyroid hormones have a diabetogenic effect, and thyrotoxicosis may induce insulin resistance and abnormal glucose tolerance. Underlying diabetes may be unmasked by thyrotoxicosis, and preexisting diabetes aggravated. Insulin requirement is increased and the tendency for ketoacidosis is higher.

Diabetes and thyroid function

Poorly compensated diabetes mellitus, similarly to other systemic nonthyroidal illnesses, may cause alteration in the production, distribution and metabolism of thyroid hormones [12]. These complex changes, in the absence of a primary disorder of the hypothalamic-pituitary-thyroid axis, result in the so-called “Euthyroid-sick syndrome”. The main feature of this disorders is a reduction of serum concentration of T3, the metabolically active hormone. In spite of this low-T3 state, patients with non-thyroidal illnesses appear clinically euthyroid, and have normal indexes of thyroid hormone action (e.g. basal metabolic rate, systolic time intervals and tendon reflex relaxation time). Reduced serum levels of T3 in the “Euthyroid-sick syndrome” seem to represent an adaptive mechanism to minimize the generalized catabolic effects of T3. Whether the low T3 state develops during systemic illness or metabolic disorders in pregnant women is unknown. However, the hormonal variations associated with this syndrome should be taken into account when evaluating a diabetic pregnant woman, and changes in serum thyroid hormone levels associated with decompensated diabetes mellitus should be differentiated from primary disorders of thyroid function.

Laboratory diagnosis

Aside an accurate clinical examination, there are several tools which are commonly used in diagnostic protocols for thyroid disease. A scheme for the evaluation of thyroid function in pregnant women with diabetes mellitus is shown in Table 1. It includes the measurement of serum levels of FT4, FT3 and sensitive TSH. Normal FT4 and TSH will exclude thyroid dysfunction while normal or low TSH, normal FT4 and low FT3 may be indicative of euthyroid sick syndrome, compatible with poorly controlled diabetes. Undetectable TSH with high FT4 will point to primary hyperthyroidism, while elevated TSH with normal or low FT4 will suggest primary thyroid failure either subclinical or clinical.
Table 1. A scheme for the evaluation of thyroid function in pregnant women with diabetes mellitus

<table>
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<tr>
<th></th>
<th>FT4</th>
<th>FT3</th>
<th>TSH</th>
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<tbody>
<tr>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Euthyroid</td>
<td>normal</td>
<td>low</td>
<td>normal</td>
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<tr>
<td>sick syndrome</td>
<td>or low</td>
<td>or low</td>
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<tr>
<td>Primary</td>
<td>high</td>
<td>high</td>
<td>undetectable</td>
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<tr>
<td>hyperthyroidism</td>
<td>or low</td>
<td>or low</td>
<td></td>
</tr>
<tr>
<td>Primary</td>
<td>normal</td>
<td>normal</td>
<td>high</td>
</tr>
<tr>
<td>hypothyroidism</td>
<td>or low</td>
<td>or low</td>
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Some additional tests for the evaluation of thyroid function are particularly helpful in pregnant women since thyroid scintiscan and measurement of radioiodine uptake are not advised during pregnancy. Measurement of serum thyroxinase antibody, thyroglobulin antibody and TSH-receptor antibody are useful in the diagnosis of Hashimoto's thyroiditis and Graves' disease. Serum thyroglobulin (Tg) may help to differentiate hyperthyroidism, due to overproduction of thyroid hormones, and thyrotoxicosis, due to destruction of thyroid tissue, from an excess of exogenous thyroid hormones. In the former cases serum Tg is elevated whereas in the latter condition is usually suppressed. However, Tg may be undetectable also in the presence of circulating anti-Tg antibodies [13]. Measurement of urinary iodine can reveal an iodine overload which may be responsible for both thyrotoxicosis and hypothyroidism.

In pregnant women useful information are provided by thyroid ultrasonography. Indeed it helps to confirm the presence of a nodule in a goitre felt by palpation. Small nodules, less than 0.5 cm, can be reliably detected by ultrasonography and the precise size of a nodule as well as of the gland can be defined. Moreover, this technique allows the distinction between solid and cystic lesions and may confirm the thyroid origin of a neck lump. Finally, sonography can be used to perform a more precise needle aspiration within the nodular lesion to be sampled. Recently, evidence has been provided that the pattern of thyroid hypoeoegeticity by ultrasound helps to differentiate among patients with goiter and circulating thyroid autoantibodies those who have Hashimoto's thyroiditis and are prone to develop hypothyroidism from those with nodular goiter and focal lymphocytic infiltration [14].

Fine needle aspiration biopsy is not contraindicated in pregnancy and can be used for the differentiation of benign from malignant thyroid nodules.

Insulin-dependent diabetes mellitus and post partum thyroiditis

Post partum thyroiditis (PPT) is a clinically heterogeneous disorder which is due to an autoimmune inflammation of the thyroid gland occurring in the first year after delivery [15]. Transient thyrotoxicosis, transient hypothyroidism or both thyroid dysfunctions in a sequence may occur in PPT. Thyrotoxicosis usually develops 2 to 6 months post partum as a result of thyroid follicle destruction produced by cytotoxic effector mechanisms, which are likely to include both cell-mediated and antibody-dependent tissue injury. Thyrotoxicosis lasts 2 to 6 weeks and in about half of patients is followed by transient hypothyroidism. Thyrotoxic symptoms are usually mild and self-limiting, with no or little thyroid enlargement.

The pathogenesis of post partum thyroiditis is related to autoimmune mechanisms. Pregnancy is associated with alterations in the maternal immune system which prevent rejection of the fetus bearing paternal alloantigens. This is demonstrated by the amelioration of several autoimmune diseases during pregnancy. After delivery there is a rebound in immune responsiveness and a worsening of coincident autoimmune disorders. The immune rebound of post partum also involves thyroid autoimmunity, and post partum thyroiditis is a common expression of it. The estimated incidence of post partum thyroiditis is about 5% [16]. According to the autoimmune nature of the disease, pregnant women with circulating thyroxinase antibody are at increased risk for PPT. Pregnant women with IDDM have an increased risk of developing PPT. The incidence of PPT in women with IDDM ranges between 10.5% and 25% in different studies [17]. Therefore, pregnant women with IDDM have a 2 to 3 fold greater chance of developing post partum thyroiditis. These epidemiological data have lead to the recommendation that thyroid antibodies and thyroid function should be checked at 3 months in the post partum.

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