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POSTPARTUM THYROIDITIS

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CLINICAL RECOGNITION

Postpartum thyroiditis is the term used for patients who develop painless thyroiditis in the postpartum period. It occurs within 6 months (typically 2 to 4 months) after delivery and runs a clinical course identical to that of painless thyroiditis occurring without relation to pregnancy. The clinical course of thyroid dysfunction is similar to subacute thyroiditis but with no anterior neck pain or tenderness of the thyroid. The prevalence of postpartum thyroiditis ranges from 3 to 8 per cent of all pregnancies. Most women with subclinical autoimmune thyroiditis and antithyroid microsomal antibodies of more than 1:5120 before pregnancy develop postpartum thyroiditis. After delivery, other forms of autoimmune thyroid dysfunction may also occur, including Graves' disease, transient hypothyroidism without preceding destructive thyrotoxicosis, and persistent hypothyroidism (Fig. 1).

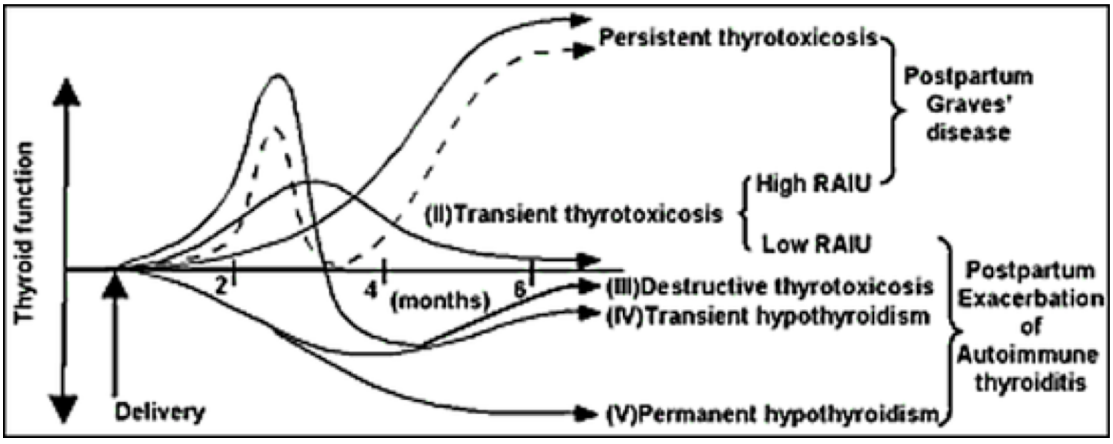

Fig. 1 Various clinical courses of thyroid dysfunction after delivery. A typical form of postpartum thyroiditis is “(III) Destructive thyrotoxicosis”. RAIU, radioactive iodine uptake.

The typical course is characterized by three sequential phases: the thyrotoxic, the hypothyroid and recovery phase. The thyrotoxic phase occurs 1–3 months after parturition and lasts for a few months, followed by hypothyroidism at 3–6 months after delivery. Finally, normal thyroid function is usually achieved within a year. Most patients have a complete remission, but some develop persistent hypothyroidism.

PATHOPHYSIOLOGY

This disorder is believed to be an autoimmune disorder and is characterized by lymphocytic infiltration of the thyroid gland and by transient thyrotoxicosis followed by hypothyroidism or by one or the other occurring in the first year after parturition. An immune rebound mechanism has been established for the induction of postpartum thyroiditis. Thyrotoxicosis is induced by leakage of intrathyroidal hormones into the circulation caused by damage to thyroid epithelial cells from inflammation. The early phase of thyrotoxicosis in postpartum thyroiditis can be classified as destruction-induced thyrotoxicosis or simply as destructive thyrotoxicosis.

DIAGNOSIS AND DIFFERENTIAL

Diagnostic Tests Needed and Suggested

Serum TSH is suppressed, associated with an increase in serum FT3 and FT4 levels. The thyroid radioactive iodine uptake (RAIU) is low. When the measurement of RAIU is difficult due to nursing, the measurement of anti-TSH receptor antibody and/or thyroid blood flow by ultrasonography may be useful to differentiate between destruction-induced thyrotoxicosis and Graves' thyrotoxicosis. Positive TPOAb or TGAb indicate the autoimmune nature of the disease.

THERAPY

If symptoms or signs of thyrotoxicosis are severe, beta-blocker drugs can be administered for the duration of the thyrotoxic phase. Propylthiouracil can be used to inhibit conversion of thyroxine to triiodothyronine. Obviously, RAI treatment is excluded since RAIU is suppressed in the toxic phase. During the hypothyroid phase, L-T4 at replacement doses is recommended.

FOLLOW-UP

Patients who have developed permanent hypothyroidism should be treated with replacement doses of L-T4. All patients should be monitored for thyroid function test at their next pregnancy, delivery and postpartum.

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