



THYROID AND PREGNANCY

Postpartum psychosis is more prevalent in women with autoimmune thyroid disease

BACKGROUND

After delivering a baby, some women may develop psychiatric issues including postpartum depression. The most extreme of these psychiatric problems is postpartum psychosis and is a psychiatric emergency. Postpartum psychosis is characterized by mood changes with delusions and hallucinations, agitation, insomnia and cognitive impairment, which may be accompanied by thoughts of suicide and infanticide. It occurs in 0.1% of new mothers, often without prior warning signs or prior history of a psychiatric disorder. The cause of postpartum psychosis is unknown. Because they can also cause marked changes in mood and are relatively common in the postpartum period, thyroid disorders have been suggested as either causing or contributing to postpartum psychosis. Most thyroid disorders in the postpartum period are caused by patients developing antibodies that attack the thyroid and either turn it on (hyperthyroidism) or turn it off (hypothyroidism). This is called autoimmune thyroid disease and is characterized by positive TPO antibodies. The aim of this study was to see if autoimmune thyroid disease is associated with postpartum psychosis.

THE FULL ARTICLE TITLE

Bergink V et al. Prevalence of autoimmune thyroid dysfunction in postpartum psychosis. *Br J Psychiatry* 2011;198:264-8. Epub February 22, 2011.

SUMMARY OF THE STUDY

All patients attending the mother and inpatient unit of the Department of Psychiatry at the Erasmus Medical Centre in the Netherlands between August 2005 and November 2008 were evaluated for postpartum psychosis. Overall, 31 patients were identified as having psychosis following the delivery of their first child. None of the women had a prior psychiatric history. A total of 23 patients presented with manic psychosis, 5 with a mixed episode of mania and depression and 3 with psychotic depression. The thyroid status in these patients was compared to 117 women who had delivered their first baby and were followed for the first year after delivery. Blood levels of TPO antibodies, TSH and free T₄ were measured at various times in the patients

with postpartum psychosis and at 4 weeks and 9 months postpartum in the control women.

None of the patients or controls had a prior history of thyroid or autoimmune disease. At 4 weeks postpartum, 5% of the control group had positive autoimmune thyroid disease, while 19% of the patients with postpartum psychosis had autoimmune thyroid disease on admission to the hospital and half exhibited clinical thyroid dysfunction. At 9 months, 13% of the controls and 29% of the women with postpartum psychosis had autoimmune thyroid disease. Patients with postpartum psychosis and autoimmune thyroid disease had a higher rate of progression to clinical thyroid dysfunction (67% in the postpartum psychosis group versus 20% in the control group). Lithium treatment may have played a role in the worsening of thyroid disease in the women with postpartum psychosis and autoimmune thyroid disease because lithium is known to alter thyroid function in patients with autoimmune thyroid disease.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

Women with postpartum psychosis are at higher risk for autoimmune thyroid disease and clinical thyroid disorders, and autoimmune thyroid disease may play a role in the development of postpartum psychosis. These results indicate that patients presenting with postpartum psychosis should be screened by measurement of blood levels of TPO antibodies. Also, patients known to have autoimmune thyroid disease either before pregnancy or developing postpartum may be at higher risk for developing postpartum psychosis. The connection between autoimmune thyroid disease and postpartum psychosis clearly requires further study.

— Glenn Braunstein, MD

ATA THYROID BROCHURE LINKS

Postpartum Thyroiditis: http://thyroid.org/patients/patient_brochures/postpartum.html

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THYROID AND PREGNANCY, continued

ABBREVIATIONS & DEFINITIONS

Antibodies: proteins that are produced by the body's immune cells that attack and destroy bacteria and viruses that cause infections. Occasionally the antibodies get confused and attack the body's own tissues, causing autoimmune disease.

Autoimmune thyroid disease (AITD): a group of disorders that are caused by antibodies that get confused and attack the thyroid. These antibodies can either turn on the thyroid (Graves' disease, hyperthyroidism) or turn it off (Hashimoto's thyroiditis, hypothyroidism).

Thyroxine (T₄): the major hormone secreted by the thyroid gland. Thyroxine is broken down to produce Triiodothyronine which causes most of the effects of the thyroid hormones. Free T₄ refers to the biologically active form that is not bound to proteins in the blood, and, therefore, can enter the cells throughout the body.

Postpartum thyroiditis: an inflammation of the thyroid in women who have just delivered a baby. The inflammation first causes mild hyperthyroidism for 1-3 months after delivery. This is followed by hypothyroidism starting 4-6 months after delivery. The hypothyroidism resolves and normal thyroid function returns 12-18 months after delivery in most women. While many women have both the hyperthyroid and the hypothyroid phase, some women may only have one or the other.

TSH: thyroid stimulating hormone – produced by the pituitary gland that regulates thyroid function; also the best screening test to determine if the thyroid is functioning normally.

TPO antibodies: these are antibodies that attack the thyroid instead of bacteria and viruses, they are a marker for autoimmune thyroid disease, which is the main underlying cause for hypothyroidism and hyperthyroidism in the United States.