Maternal Hyperthyroidism and Pregnancy Complications

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Commentary

The normal variations in the levels of maternal Thyroid Hormones (THs) throughout gestation are required for the normal development [1-60], in particular the metabolic homeostasis [60-62]. On the other hand, [63-75] classified the hyperthyroidism into three types as the following:

a. Thyrotoxicosis rising from the thyroid.

b. Overt hyperthyroidism is found when the concentration of the Thyroid-Stimulating Hormone (TSH) is in the normal range and the concentrations of the 3,5,3'-triiodothyronine (T3) and free or total Thyroxine (T4) are above the normal range.

c. Subclinical hyperthyroidism is found when the concentration of the TSH is under the normal range and the concentrations of the T3 and free or total T4 are in the normal range.

As well, Glinoer [76] and [77-83] revealed that the Human Chorionic Gonadotropin (HCG) can stimulate the TSH receptor and cause Gestational Transient Thyrotoxicosis (GTT). Other etiologies of thyrotoxicosis during gestation including the following:

a. Hyperthyroidism of trophoblastic disease or symptomatic hyperthyroidism [84-91];

b. Disturbances in the genomic duplication of monospermic or dispermic fertilization and damage in the maternal nuclear genome [92];

c. Exogenous TH, thyroiditis, toxic adenoma, toxic multinodular goiter, and Graves’ disease [93,94]. Hyperreflexia, anxiety, tachycardia, heat intolerance, goiter, lack of weight gain, and palpitations are the most symptoms and signs of gestational thyrotoxicosis [95,96]. The signs of Graves’ disease are ophthalmopathy or the presence of a goiter [97,98]. In dissimilarity, the symptoms of GTT are related to the persistent vomiting of hyperemesis gravidarum [99,100].

On the other hand, number of examinations has revealed a relationship between the hyperthyroidism and an increase in the risk of pregnancy complications [101]. These complications were including the congestive heart failure, pre-eclampsia, miscarriage, pre-term delivery, hypertension or postpartum thyroiditis (thyroid inflammation). Also, intrauterine growth restriction and failure in neonatal growth are also associated with the goiter formation and maternal hyperthyroidism. More importantly, the elevation in the transportation of maternal TSH Receptor Anti Bodies (TRAb) through placenta and lactation to fetuses and neonates can cause fetal and neonatal Graves’ disease [102]. These disorders may delay the maternal and fetal outcomes. Thus, the prenatal care, anti-thyroid medications, and assessing TRAb levels at the late pregnancy may decrease these complications. Also, detection the nature and duration of the GTT symptoms may aid in the diagnostic decisions. Overall, the variations in the signs and symptoms of gestational hyperthyroidism make experimental studies essential.

Conflict of Interest

The author declares that no competing financial interests exist.

References


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