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Relationship between Maternal Thyroid autoantibodies, Preterm Delivery and Neonatal Disorders: Potential Challenges

Ahmed RG*

Division of Anatomy and Embryology, Department of Zoology, Beni Suef University, Egypt

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***Corresponding author:** Ahmed RG, Division of Anatomy and Embryology, Department of Zoology, Beni Suef University, Egypt, Tel/Fax: 002-010-91471828; Email: ahmedragab08@gmail.com

Commentary

The regular changes in the maternal thyroid hormones (THs; 3,5,3'-triiodothyronine (T3) and thyroxin (T4)) economy during the gestation necessitate the establishment of normal delivery and fetal-neonatal development [1-72]. It is recognized that gestational immune modulation can decline the levels of thyroperoxidase antibody (TPOAb) with the progress of gestation and occasionally TPOAb became negligence during the later pregnancy [73]. On the other hand, an autoimmune disease (AID) is accompanying with circulating auto antibodies (self-reactivity of a different protein), mainly mediated by autoimmune T lymphocytes, and caused several tissue damages and inflammation [74-76].In addition, these circulating auto antibodies are established for many years before the beginning of a disease. The presence of TPOAb during pregnancy can increase the risk of maternal thyroid dysfunctions, and cause a hypothyroidism [77-81] and premature delivery [82-85,80].

This autoimmunity is reinforced by the higher level of median thyroid-stimulating hormone (TSH) and the higher risk of gestational hypothyroidism [86-87]. As well, there is a relationship between the maternal TPOAb and thyroglobulin antibodies (TgAbs) and irregular obstetric consequence [82,84,88] such as placental abruption, fetal growth retardation, miscarriage, and neonatal neuropsychological (motor, cognitive, and attention) disorders [89-92]. Also, thyroid autoantibodies can increase the risk of miscarriage [82], neonatal respiratory distress syndrome [93], and cardiovascular, metabolic, and renal disorders [94-97]. More interestingly, [98,92] reported that TPOAb positive during the gestation can cause polyhydramnios and postpartum thyroiditis (PPT). The presence of thyroid antibodies before the gestation can increase the prevalence of gestational complications [99-109]. Finally, thyroid auto antibodies may induce these disorders as the following [102-104,82,106,101].

a) Alter the levels of TSH and the outline of endometrial T cells.

b) Decrease the secretion of interleukins (IL-4 and -10).

c) Increase the secretion of interferon γ.

d) Elevate the migration process of cytotoxic natural killer cells.

e) Disrupt the uterine hormonal and immune responses.

f) Perturb the levels of cytokine networks (inflammatory processes) during the placental–decidual (fetoplacental) development.

g) Maternofetal immune dysregulations.

Collectively, the present commentary suggested that the occurrence of thyroid auto antibodies (TPOAb and TgAbs) during the gestation may cause several pregnancy complications, thyroid disorders (hypothyroidism and PPT), teratogenic outcomes, preterm birth, and fetal and neonatal neuro developmental dysfunctions. These disorders may be mediated by the hormonal-immune dysregulations and may impact the development and childhood life. However, the possible effects of TPOAb and TgAbs on fetal-neonatal neurodevelopment warrant additional examination. Also, screening of maternal thyroid function before and at early pregnancy should be performed specifically in TPOAb- or TgAbs positive pregnant. Such monitoring may be improved the maternofetal, neonatal and child health consequences.

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