Maternal Thyroid Function and Placental Hemodynamics

Ahmed R.G.

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

*Corresponding Author: Ahmed R.G, Division of Anatomy and Embryology, Zoology Department, Faculty of Science, Beni-Suef University, Beni-Suef, Egypt. Email: ahmedragab08@gmail.com

LETTER TO EDITOR

The concentrations of thyroid hormones (THs) are necessary for the normal development (Elbakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-u & 2018a-c; Ahmed et al., 2010, 2013a,b, 2014, 2015a,b & 2018a,b; Ahmed and Incerpi, 2013; Van Herck et al., 2013; Ahmed and El-Gareib, 2014, Incerpi et al., 2014; Candelotti et al., 2015; De Vito et al., 2015; El-Ghareeb et al., 2016; Ahmed and El-Gareib, 2017) in particular the placentation (Barber et al., 2005; Krassas et al., 2010; Loubiere et al., 2010; Aghajanova et al., 2011; Patel et al., 2011; Barjaktarovic et al., 2017). On account of the normal placentation (interstitial invasion of fetal trophoblast cells into maternal decidua and endovascular trophoblast (EVT) invasion into maternal spiral arteries, Cartwright et al., 2010) is vital for maintaining the gestation and for the optimal fetal development; supply nutrients, exchange gases and eliminate the metabolic waste products, any disorders in the placental functions can cause several pregnancy complications such as the preeclampsia, premature delivery, and fetal growth restriction. These processes can be mediated by the normal expression of thyroid receptors (TRs; α and β) and the normal activities of thyroid transporters (THTs) in the trophoblast cells (Barber et al., 2005; Loubiere et al., 2010; Aghajanova et al., 2011; Patel et al., 2011). On the other hand, impairment of the normal placentation in early pregnancy (Cartwright et al., 2010) due to hyperthyroidism could cause fetal growth restriction (Medici et al., 2013; Haddow et al., 2014) and preeclampsia (Aggarawal et al., 2014; Medici et al., 2014). In addition, abnormal placentalation in early pregnancy (Cartwright et al., 2010) due to hypothyroidism could cause premature delivery (Korevaar et al., 2013; Sheehan et al., 2015). From the clinical data, there are association between the thyroid disorders and placental dysfunction, and pregnancy complications (Korevaar et al., 2013; Medici et al., 2013, 2014; Odibo et al., 2014).

From the aforementioned clarifications and the current opinion, it can be inferred that the early normal activities of the maternal thyroid gland may be a regulator of the normal placentation. In addition, maternal thyroid dysfunctions (hyperthyroidism or hypothyroidism) can impair the placental growth factors, cytokines, and blood supply. The placental dysfunction may increase the risk of preeclampsia, fetal growth restriction and mortality. Thus, treatment of thyroid disorders before or during the gestation may decrease the risk of pregnancy complications. Additional investigations are necessary not only to examine the biological and molecular mechanisms between the thyroid dysfunction and placental disorders, preeclampsia and premature delivery but also to compare the data with the clinical studies.
REFERENCES


[22] Ahmed, R.G., 2017c. Anti-thyroid drugs may be at higher risk for perinatal thyroid disease. EC Pharmacology and Toxicology 4.4, 140-142.


[27] Ahmed, R.G., 2017h. Gestational prooxidant-antioxidant imbalance may be at higher risk for...
Maternal Thyroid Function and Placental Hemodynamics

postpartum thyroid disease. Endocrinol MetabSyndr 6, 279. doi:10.4172/2161-1017.1000279.


[33] Ahmed, R.G., 2017n. Letter: Gestational dexamethasone may be at higher risk for thyroid disease developing peripartum. Open Journal Of Biomedical & Life Sciences (Ojbili) 3(2), 01-06.


