Maternal Hypothyroidism-Milk Ejections: What is the Link?

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HYPOTHESIS

The normal activity for the maternal hypothalamic-pituitary-thyroid axis (HPTA) is dynamic for the developing fetuses and neonates (Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-v & 2018a-j; Ahmed et al., 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), especially the function and activity of mammary gland (Varas et al. 2001 & 2002; Hapon et al. 2003 & 2007a,b; Ahmed et al., 2008; Campo Verde Arbocco et al. 2015 & 2016; Pennacchio et al., 2017). Thyroid hormones (THs) stimulate the signal transducer activator of transcription 5 (STAT5) proteins and ovarian hormone receptors-induced the differentiation of the mammary epithelial cell by their nuclear receptors (TRs) (Favre-Young et al., 2000; Zhao et al., 2005; Bagamasbad and Denver, 2011; Campo Verde Arbocco et al., 2015 & 2016; Pennacchio et al., 2017). More interestingly, THs, prolactin (PRL), glucocorticoids and ovarian hormones (estrogen and progesterone) can regulate the involution switch process in the mammary epithelial cell by activating several signaling pathways such as mitogen-activated protein kinase (MAPK) and JAK/STAT (Li et al., 1997; Lemay et al., 2007; Watson, 2009; Whyte et al., 2009; Bertucci et al., 2010; Bagamasbad and Denver, 2011; Zhao et al., 2012; Campo Verde Arbocco et al., 2017). Indeed, there is reduction in the expression of deiodinase type I [converts tetraiodothyronine (T4) to triiodothyronine (T3)] in the mammary gland to avoid the deleterious influence of any excessive in THs (Anguiano et al., 2004).

On the other hand, in the previous studies, several authors demonstrated the association between the thyroid dysfunction (hypothyroidism) and hindering the lactation mechanism (Hapon et al. 2003 & 2007a,b; Campo Verde Arbocco et al. 2015, 2016 & 2017). This defect can be illustrated as the following (Hapon et al. 2003 & 2007b): (1) reduced the quality of milk nutrition quality; (2) dysfunction in the milk ejection due to the reduction in the response of oxytocin (OXT); (3) disorders in the accumulation of milk in the alveoli; (4) decreased the growth rate of the litter/pups and increased the litter mortality; (5) milk stasis and mammary involution (Rhoads and Grudzien-Nogalska, 2007; Bertucci et al. 2010; Campo Verde Arbocco et al., 2017); (6) disorders in the levels of all previous hormones and in the expression of their receptors; and (7) disturbance in the signaling of PRL and elevation in the signaling of PRL inhibitors including LIF and STAT3 (Campo Verde Arbocco et al., 2016).

From the previous data, I hypothesized that the maternal THs play active roles in the lactation process. The maternal hypothyroidism may directly or indirectly increase the risk of the premature mammary involution and destruction of the mammary tissue. The maternal hypothyroidism with suckling disorders may suppress the normal development. Thus, we can avoid these abnormalities if we controlling the activities of the maternal PRL, T3, T4, growth hormone (GH), and thyroid-stimulating hormone (TSH) during the gestation and lactation. Additional experiments are needed to identify the associations between the maternal hypothyroidism, lactation disorders and mammary carcinogenesis.
REFERENCES


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


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