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Maternal Thyroid Cancer

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LETTER TO EDITOR

The normal cooperation in the maternal hypothalamic-pituitary-thyroid axis (HPTA) during pregnancy is critical for the fetal and neonatal development(El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-u & 2018a-d; 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; Moog et al., 2017). On the other hand, thyroid cancer is the utmost common type of endocrine neoplasia (90% of the endocrine malignancies) (Carling and Udelsman, 2014; Galdiero et al., 2016). There are links between the thyroid cancer and the chronic inflammation (Cunha et al., 2014; Galdiero et al., 2016). In addition, Lumachi et al. (2010) reported that the cytokines, pro inflammatory mediators, can control the response of systemic inflammatory, show a critical action in autoimmune thyroid diseases, and stimulate the development and growth of normal and neoplastic thyroid cells. Carling and Udelsman (2014) observed that the percentage of thyroid cancer can be divided into 5-10% is follicular thyroid carcinoma (FTC) and 80–85% is papillary thyroid carcinoma (PTC). The variations in the expression of mitogen-activated protein kinase (MAPK) and the PI3K-AKT pathway, including RET (encoding proto-oncogene tyrosine-protein kinase receptor ret), BRAF (encoding serine/threonine kinase B-raf), and RAS genes (which encode small GTPases) could cause the PTC (Cancer Genome Atlas Research, 2014; Hsiao et al., 2014). In addition, the alterations in the expression of PIK3CA, AKT1, and peroxisome proliferator-activated receptor gamma (PPAR-γ) could cause the FTC (Hsiao et al., 2014). On the other hand, thyroid cancer can affect tumor angiogenesis in particular the release of the vascular endothelial growth factor (VEGF; A and B), and chemokines (CXCL8 and IL-8), and lymph angiogenesis in particular the VEGF C and D factors (Curiel et al., 2004; Detoraki et al., 2009; Granata et al., 2010; Bruno et al., 2013; Mantovani et al., 2013; Visciano et al., 2015; Galdiero et al., 2016). Also, lymph angiogenesis and angiogenesis can increase the risk of the tumor growth and formation of metastasis (Detoraki et al., 2009; Bruno et al., 2013). More importantly, the micro environments such as the extracellular matrix components (ECM), blood and lymphatic vessels, immune cells, endothelial cell progenitors, and fibro blasts, play significant roles in the tumor initiation and progression (Ryder et al., 2008; Bissell and Hines, 2011;Coussens et al., 2013; Jung et al., 2015).

From the previous data and the current view, it can be inferred that the maternal thyroid cancer may delay the actions of all central biological systems during the prenatal and postnatal periods. Also, the treatment may be vital to inhibit the development of thyroid cancer. This can keep the stability in the HPTA during the perinatal period. All efforts should be accomplished to avoid the exposure to radiation and to find thyroid tumors carefully during pregnancy and lactation periods. Further studies are needed to discover the diagnostic and prognostic markers, and to design novel drugs for the common endocrine malignancy in particular the thyroid cancer. In addition, the molecular actions controlling the immune cytokine networks as well as lymph angiogenesis/ angiogenesis in several types of thyroid cancer should be examined. These problems need more analysis.

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