

Maternal Hyperthyroidism and Developing Thyroid Cancer: What is the Next?

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COMMENTARY

Optimal actions in the maternal thyroid hormones (THs; 3,5,3'-triiodothyronine (T3) and thyroxine (T4)) during pregnancy are central to a normal fetal and neonatal consequence (Elbakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-v, 2018a-u; Ahmed and Ahmed, 2012; Ahmed et al., 2008; 2010; 2012; 2013a,b, 2014, 2015a,b, 2018a,b; Ahmed and Incerpi, 2013; Van Herckel 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). On the other hand, a controversial association between THs and cancer was observed. Some authors postulating that hyperthyroidism can induce cancer development and progression (Gabriele et al., 2003; Furumoto et al., 2005; Davis et al., 2006; Nishida et al., 2008; Kress et al., 2010), although others have stated a tumor inhibition action of THs (Ruiz-Llorente et al., 2011; Manka et al., 2018). Galdiero et al. (2016) reported that about 90% of the endocrine malignancies are a thyroid cancer. A toxic nodular goiter (TNG) can increase the risk of metastatic follicular thyroid carcinoma (FTC) (Sahin et al., 2005). It appears that the prediction of metastatic follicular carcinoma does not fluctuate with the attendance or lacking hyperthyroidism (Tsuchiya et al., 1987; Paul and Sisson, 1990; Lin et al., 2004; Giovanella et al., 2010). As well, Cakir et al. (2007) suggested that the prevalence of thyroid carcinoma was lower in subjects with Graves' disease than in subjects with toxic adenoma (TA) or toxic multi nodular goiter (TMG). Several investigators reported that Graves' disease (20-25%) is the utmost communal cause of hyperthyroidism and the frequency of thyroid nodules (Pacini et al., 1998; Kraimps et al., 2000; Taneri et al., 2005). Indeed, cancer related

to Graves' disease appears to be more destructive than those linked to multi nodular toxic goiter (MTG) or uni nodular toxic goiter (UTG) (Cappelli et al., 2006). In addition, Wong et al. (2003) observed that thyrotoxicosis in women was associated with Hurthle cell carcinoma of the thyroid (a rare type of thyroid neoplasm).

More importantly, develop thyroid cancer in patients with Graves' and thyroid nodules were more severe than in patients with diffuse goiter (Pazaitou- Panayiotou et al., 2012). The possibility of Graves' disease and development of thyroid cancer, mainly of papillary carcinoma can be attributed to the presence of thyroid auto antibodies (Filetti et al., 1988; Belfiore et al., 1990; Pellegriti et al., 1998; Gagliano et al., 2016).

Finally, it is worth pronouncing that maternal hyperthyroidism or thyrotoxicosis may increase the rate of development and progression of thyroid carcinoma and thyroid nodules. The disorders in the actions of the maternal thyroid gland (thyroid carcinoma or nodules) may disrupt the fetal and neonatal development and increase the possibility of mortality and morbidity. These disturbances may be depending on a family history of thyroid cancer, multiple endocrine neoplasia syndromes, a rapid growth of a nodule, and cervical lymphadenopathy. Thus, the incidence of a nodule in a hyper thyroidism or thyrotoxicosis should be carefully assessed to eliminate the attendance of malignancy. Additional studies are necessary to study the clinical and pathological features of materno fetal thyrotoxicosis and thyroid micro carcinoma (distinguish aggressive lesions from benign adenomas). It is important to study the prediction of non-metastatic hyperthyroidism with the follicular thyroid carcinoma. Additionally, every suspicious nodule related to hyperthyroidism should be appraised importantly.

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