Maternal Hypothyroidism and Reproduction

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HYPOTHESIS

The balance between the thyroid hormones (THs), thyroid-stimulating hormone (TSH), growth hormone (GH) and insulin growth factors (IGFs) is crucial for the in utero and postnatal periods (Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-v& 2018a-j; Ahmed et al., 2010, 2013a,b, 2014, 2015a,b &2018a,b; Ahmed and Incerpi, 2013; Van Hercket 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, early clarifications have pronounced the association between the thyroid dysfunction and disorders in the reproduction process and sexual activity (Dousaf and Mastorakos, 2000; Krassas and Perros, 2003; Aitken and Krausz, 2001; Agarwal and Saleh, 2003; Wajner et al., 2009; Rijntjes et al., 2017). Hypothyroidism can alter the male fertility as the following (Donnelly and White, 2000; Carani et al., 2005; Krassas et al., 2008 & 2010): (1) decrease the concentration of total testosterone; (2) diminish the concentration of free testosterone about 60%; (3) reduce the libido; (4) erectile dysfunction; (5) increase the risk of testicular atrophy (Wortsman et al., 1987); and (6) defect the spermatogenesis. These results can be due to thyroid disorders induced the egress of free radicals (Iwasaki and Gagnon, 1992; Aitken and Krausz, 2001; Agarwal and Saleh, 2002; Mancini et al., 2009). Other data were reported by Gallagher (1966), Longcope et al. (1990), Krassas (2000), and Redmond (2004) who demonstrated that hypothyroidism can alter the female fertility as the following: (1) change the metabolism of peripheral estrogen; (2) alter the secretion of gonadotropin-releasing hormone (GnRH) producing some abnormalities in the release of pulsatile luteinizing hormone (LH); (3) hyperprolactinemia; (4) decrease the concentration of total testosterone and estradiol; (5) reduce the metabolism of androgen; (6) augment the excretion of 2-oxygenated estrogens; and (7) luteal phase defect (Daya et al., 1988). In general, the hypothyroid state of THs may cause infertility, and maternal complications (Davis et al., 1988; Stray-Pedersen & Stray-Pedersen, 1984; Wajner et al., 2009; Rijntjes et al., 2017). More interestingly, the deterioration of thyroid disorders on the luteal phase can be attributed to the egress of free radicals and oxidative stress (Ahmed et al., 2008). These data are observed in human, animals and in vitro models. The oxidative stress can reduce the female fertility. In addition, a reduction in the antioxidant system has been assumed in infertility, and polycystic ovary (Ruder et al., 2008).

In the light of the previous thoughts, my data assume several visions. The balance in the maternal HPTA shows a noteworthy role in the reproduction process. In addition, the maternal thyroid dysfunctions (hypothyroidism) may deteriorate the sperm motility, luteal phase deficiency, reproduction process and sexual activity. The prolonged harmful effects of thyroid abnormalities can increase the egress of free radicals and cause male and female infertility. Thus, the management of the thyroid markers before the marriage and the gestation should be crucial for the endocrine, metabolic, and sexual improvement. This can improve the fertility of both male and female. However, additional studies are needed to discover whether the abnormalities in the maternal HPTA/thyroid gland during pregnancy may cause persistent disturbances in the reproduction process and sexual activity. The urologists and endocrinologists are essential to working together to avoid these unusual disorders. The issue remains open.
REFERENCES


Maternal Hypothyroidism and Reproduction


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