

Maternal Thyroid Dysfunction and Risk of Neonatal Stroke

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

During the gestational period, steady increase in the levels of maternal thyroid hormones (THs; thyroxine (T4) and 3,5,3'-triiodothyronine (T3)) is essential for the regular development during the gestational and suckling periods (El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-v& 2018a-i; Ahmed et al., 2010, 2013a,b, 2014, 2015a,b & 2018a,b; Ahmed and Incerpi, 2013; Van Herk 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 neuro-vascular system (Ahmed, 2017d-e & 2018h,i). THs can bind their receptor on integrin α V β 3 to regulate the extracellular matrix proteins, brain connectivity, and synaptic plasticity (Cheng et al., 2010; Ambrosius et al. 2017a,b). In addition, THs can regulate the systolic and diastolic cardiac function, increase the left ventricular (LV) contractile function, and decrease the systemic vascular resistance (SVR) (Klein and Ojamaa, 2001a,b).

On the other hand, the reduction in the levels of free T3 (FT3) might increase the risk of stroke and decrease the outcome (Ambrosius et al., 2011; Neidert et al., 2011; Filimonov et al., 2017). Also, Sullivan and Sage (2004) and Ambrosius et al. (2017a, b) reported that the elevation in the levels of free T4 (FT4) and the reduction in the levels of thyroid-stimulating hormone (TSH) can increase the ischemic stroke patho-physiology. In addition, the reduction in the levels of TSH in an elderly population can cause heart failure, atrial fibrillation (AF), coronary artery disease (CAD), and coronary heart disease (CHD) (Bauer et al., 1998; Biondi and Cooper, 2008; Rodondi et al., 2010; Collet et al., 2012; Taylor et al., 2013; Chaker et al.,

2016). This is a main cause of morbidity and mortality. On the contrary, Taylor et al. (2013) observed that the elevation in the levels of TSH and the reduction in the levels of FT4 can cause several cardiovascular problems. As well, the disturbances in the functions of thyroid glands can increase the possibility of numerous cardiovascular disorders such as atherosclerosis (Cappola and Ladenson, 2003), dyslipidemia (Duntas, 2002), and hypertension (Nagasaki et al., 2006). These abnormal disorders have also been noticed in patients of subclinical thyroid disorders (Hak et al., 2000; Iqbal et al., 2006; Asvold et al., 2007a,b). These symptoms with the primary haemorrhage, closed vessel ischaemia (venous and/or arterial), global forebrain ischaemia can cause the fetal or neonatal stroke (Raju et al., 2007; Govaert, 2009), fracture risk and cognitive disorders (Cappola et al., 2014; Blum et al., 2015). On the other hand, the administration of THs has been established to confer neuroprotection in stroke (Bettendorf et al., 2000; Genovese et al., 2013; Sadana et al., 2015; Suda et al., 2016) on account of THs may have neurotropic effects during stroke.

From the aforementioned results, it can be concluded that the variations in gestational THs (hypothyroidism or hyperthyroidism) may lead to long-lasting impairment in the neonatal neuro-vascular and cardiovascular systems. These conditions may interrupt the neonatal cognitive functions. Hence, brain imaging with the management of maternal thyroid hormones may diminish the risk of neonatal stroke. Additional studies are needed to investigate whether the regulation of maternal thyroid autoimmunity, hormonal supplementation or antagonism may enhance the functional outcome of neonatal stroke.

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