

Does Maternal Antepartum Hypothyroidism Cause Fetal and Neonatal Hyponatremia?

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HYPOTHESIS FORMATION

The activities of maternal thyroid hormones (THs) are important for the advancement of the prenatal and postnatal development (El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-v & 2018a-j; Ahmed et al., 2008, 2010, 2012, 2013a,b, 2014; 2015a,b& 2018a,b; Ahmed and Ahmed, 2012; 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; Endendijk et al., 2017; Gigena et al., 2017). The homeostasis between the levels of THs (Ahmed and Incerpi, 2013) and the placental development (Valerio et al., 2015) regulates the electrolytes equilibrium between dams and their fetuses/newborns.

On the other hand, hyponatremia, electrolyte abnormality, can cause by the excess free H₂O intake and the impairment in its excretion due to arginine vasopressin (AVP) excess (Agathis et al., 2015). Another probable cause of hyponatremia is hypothyroidism/myxedema (Macaron et al., 1978; Schutt-Aine et al., 1980; Chelimsky et al., 1997; Nakano et al., 2000). More importantly, possible mechanisms of hyponatremia associated with hypothyroidism can be explained as the following (Derubertis et al., 1971; Hanna and Scanlon, 1997; Kreisman and Hennessey, 1999; Adroque and Madias, 2000; Schmitz et al., 2001; Milionis et al., 2002; Chen et al., 2005; Warner et al., 2006; Liamis et al., 2011; Schwarz et al., 2012; Filippatos and Elisaf, 2013; Hammami et al., 2013; Baajafer et al., 2014; Pantalone and Hatipoglu, 2014; Abuzaid and Birch, 2015; Filippatos et al., 2016; Liamis et al., 2017): (1) increased the

syndrome of inappropriate antidiuretic hormone secretion (SIADH; increase urine Na⁺); (2) decreased the cardiac outputs; (3) increased the level of antidiuretic hormone (ADH); (4) salt-losing nephropathy and hypovolemia; (5) decreased the glomerular filtration rate (GFR); (6) low-iodine and solute intake; (7) decreased the water delivery to the kidney diluting segment; (8) decreased the excretion of water content; and (9) water retention. In disagreement with the above results, some investigators did not find association between the infants with congenital hypothyroidism and hyponatremia (Croal et al., 1997; Asami and Uchiyama, 2004; Katoch et al., 2013; Berndt et al., 2015). On the other hand, Liamis et al. (2017) reported that several causes of hyponatremia such as infections, drugs or adrenal insufficiency should be considered with the mild/moderate hypothyroidism.

On the above data, it can be showed that the normal passage of transplacental THs may be essential for the electrolyte equilibrium between the dams and their newborns. In addition, any disruption in the activities of maternal antepartum THs (hypothyroidism) may disturb the electrolyte equilibrium between the dams and their fetuses/neonates. My hypothesis is the maternal antepartum dyselectrolytemia may intensify the risk of pre-delivery and may cause neonatal disorders. Though, the mechanism of maternofetal dyselectrolytemia or metabolic derangement remains in determinate. Thus, I advise to treat the maternal electrolyte imbalance and thyroid functions before the gestation or correct the dyselectrolytemia and thyroid functions in the neonates after the labor. Moreover, a care postpartum for both dams and their newborns may be required to get a good outcome.

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