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Rapid Communication

Association between Hypothyroidism and Renal Dysfunctions

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The maternal thyroid hormones (THs) (El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-h; Ahmed et al., 2008, 2010, 2012, 2013a,b, 2014; 2015a,b; Ahmed and Ahmed, 2012; 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; Endendijk et al., 2017; Gigena et al., 2017) is necessary for the renal development, renal transport systems, Na/H_2O homeostasis and glomerular filtration rate (GFR) (Iglesias and Diez, 2009; Mariani and Berns, 2012; Dousdampanis et al., 2014; Iglesias et al., 2016).In addition, THs regulate the Na-H exchanger, Na-P co-transporter, and the Na/K AT Pase in proximal convoluted tubule (Basu and Mohapatra, 2012). These regulations are stimulated by their actions on the renin-angiotensin system, the cardiovascular (CV) system, and the renal blood flow (Kobori et al., 1998; Iglesias et al., 2016).

Any alterations in thyroid functions (hypothyroidism and hyperthyroidism) might disrupt the renal function (Iglesias et al., 2016). Hypothyroidism can change the hemodynamic processes (Ichihara et al., 1998; Klein and Danzi, 2007; van Hoek and Daminet, 2009; Stabouli et al., 2010; Vargas et al., 2012; Koch and Chrousos, 2016): **(1)** decrease the sensitivity to β-adrenergic stimulus, the release of renin, the erythropoietin production and atrial natriuretic factor levels; **(2)** increase the mean arterial pressure. Moreover, hypothyroidism can decrease the expression of renal vasodilators and the levels of cystatin C, and increase the matrix Gla protein, the level of serum creatinine and the permeability of glomerular capillary (Singer, 2001; Go et al., 2004; Schmid et al., 2004; van Hoek and Daminet, 2009). It can change the tubular functions (Marcos et al., 1996; Hanna and Scanlon, 1997; Schmitt et al., 2003; Iglesias et al., 2016): **(1)** increase the sensitivity to vasopressin and the levels of Na excretion;**(2)** decrease the activity of Na/K ATPase and Na-H exchanger, defect the urinary acidification, cause hyponatremia and impair the free water excretion.

On the other hand, nephrotic syndrome (NS), plasma protein (albumin, transthyretin or thyroxine binding globulin) in urine, can decrease the levels of serum total thyroxine (TT4) and total triiodothyronine (TT3) (Kaptein et al., 1982; Kaptein et al., 1991; Guo et al., 2014; Liu et al., 2014). These alterations may be depending on the degree of proteinuria and serum albumin levels (Guo et al., 2014). Patients may need exogenous levothyroxine (L-T4) to recover these disorders (Liu et al., 2014). In these cases, the treatment of L-T4 is necessary during the normal neonatal development (Holmberg et al., 1995). Thyroid gland can increase the production to compensate the hormonal urinary losses (Guo et al., 2014). However, patients with low thyroid reserve develop overt hypothyroidism. In addition, primary hypothyroidism has been observed in the congenital NS (CNS) due to intrauterine massive proteinuria and disorders in the hypothalamus-pituitary-thyroid axis (HPTA) (Holmberg et al., 1995; Chadha and Alon, 1999; Vachvanichsanong et al., 2005). On the basis of these data, it can be concluded that the thyroid dysfunction (hypothyroidism) may alter the renal function, and the reverse may be true. Additional studies are needed to interpret the potential associations between the disorders in the functions of thyroid and kidney to avoid unnecessary treatments.

Conflict of Interest

The author declares that no competing financial interests exist.

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