Maternal Hypothyroidism and Fetal Hepatic Diseases: Ongoing Debates and Key Issues

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COMMUNICATION

The normal activities of maternal thyroid hormones (THs) are needed for the fetal/ neonatal development (El-baky et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-v & 2018a-i; Ahmed et al., 2008, 2010, 2012, 2013a,b, 2014; 2015a,b& 2018a;b; Ahmed and Ahmed, 2012; Ahmed and Incerci, 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; Gigena et al., 2017), particularly the hepatic function, lipid homeostasis, the basal metabolic rate of hepatocyte, and the bilirubin metabolism (Huang and Liaw, 1995; Malik and Hodgson, 2002; Tindall et al., 2007; Paquette et al., 2011; Yao et al., 2014). In addition, the liver has a significant action in THs metabolism by deiodinases (D1, D2 and D3) (Malik and Hodgson, 2002; Tindall et al., 2007; Paquette et al., 2011; Ahmed, 2012b; Bano et al., 2016). The alterations in hepatic lipid homeostasis due to the abnormality in thyroid states might cause a non-alcoholic fatty liver disease (NAFLD; a very common liver disorder) (Liu et al., 2007; Burra, 2013). These changes were a dose-dependent manner (Chung et al., 2012). Though, the direct relationship between the pathogenesis of NAFLD and thyroid disorders is still unknown. On the other hand, thyroid disorders may disturb the liver function (Malik and Hodgson, 2002), glycogen accumulation and hepatic fatty acid composition (Yao et al., 2014), and elevate the alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase (Huang and Liaw, 1995).

The association between the hypothyroidism and cholestatic jaundice and gallstones can be attributed to the following (Van Steenbergen et al., 1989; Huang and Liaw, 1995; Gaitan and Cooper, 1997; Inkinen et al., 2000; Malik and Hodgson, 2002): (1)diminished the bilirubin UDP-glucuronyltransferase; (2) decreased bilirubin and bile excretion; (3) increased the membrane cholesterol-phospholipid ratio; (4) decreased the membrane fluidity; (5) altered the membrane transporters and the activity of Na⁺, K⁺-ATPase; and (6) hypotonia and hypercholesterolaemia of the gall bladder. These abnormalities can be reversible with a short-term replacement with thyroxin (T4) (Huang and Liaw, 1995; Gaitan and Cooper, 1997; Malik and Hodgson, 2002). Alternatively, the elevations in the levels of T4 and thyroxine-binding globulin (TBG), reductions in the activity of D1 [decrease the conversion of T4 to 3,5,3′-triiodo-L-thyronine (T3)] were associated with the liver diseases such as Hepatitis C virus infection (HCV) (Huang and Liaw, 1995) or acute hepatitis of mild or moderate severity (Hegedus, 1986; Kano et al., 1987; Malik and Hodgson, 2002). On the other hand, therapy by anti thyroid drugs might cause hepatitis, transient subclinical hepatotoxicity or cholestasis, while therapy with interferon (IFN) in liver diseases might cause thyroid dysfunctions (Benelhadj et al., 1997; Deutsch et al., 1997).

On the basis of these data, it can be concluded that there is a significant relationship between the maternofetal thyroid gland and the liver in normal or abnormal states. It is recommended to detect the levels of T4 and thyroid-stimulating hormone (TSH) with liver disorders, to include or exclude the thyroid disorders with unexplained liver abnormalities. Further studies are essential to elucidate the prospective relations with human health.
REFERENCES


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