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THE IMPACT OF PREGNANCY ON THE THYROID

Several physiologic complex changes take place during pregnancy, which together tend to modify the global economy of the thyroid gland and have variable impact at different time points during gestation. Iodine deficiency (ID) occurs when gestation takes place in areas with even a mild iodine restriction only. Since this occurs when thyroid hormone requirements are increased, ID induces a vicious circle leading to excessive glandular stimulation, relative hypothyroxinemia and gestational goitrogenesis, affecting both mother & fetus. The recommended daily iodine intake level during pregnancy has recently been reviewed and set to ideally be in the order of 250 µg (200-300 µg/d), which can be provided in the form of iodine supplements contained in multivitamin pills specifically designed for the purpose of pregnancy.

Concerning thyroid autoimmunity (TAI), there is presently good evidence to suggest that TAI is associated with a significant increase in the the risk of infertility, miscarriage, and adverse obstetrical events when gestation progresses to term. Because of the high prevalence of TAI in young women, because subclinical hypothyroidism often remains undiagnosed, because potential obstetric repercussions are associated with untreated hypothyroidism, and finally because there are potential consequences of maternal hypothyroxinemia on fetal development, there is a justification to propose a systematic screening for TAI and hypothyroidism in pregnancy.

When women present hypothyroidism during pregnancy, there is an incrased risk for adverse maternal and infant outcomes, including spontaneous abortion, gestational hypertension, placental abruption, breech presentation, fetal distress, preterm birth, low birth wight, and perhaps fetal and neonatal death. Most available information indicates that an early and adequate treatment with l-thyroxine drastically reduces the frequency of these abnormalities.

The fetus requires thyroxine for its normal development, and particularly for its brain architectural build-up. Fetal hormone production/secretion does not begin until approximately 20 weeks of gestation. Thus, the fetus is entirely dependent on thyroid hormone transferred from the mother during the first trimester. Published studies consistently document a relationship between maternal hypothyroxinemia and impairments in the neuro-intellectual development of the offspring. Three recent sets of studies, in the USA (Haddow et al. in 1999), the Netherlands (Pop et al. in 1999 and 2003) and Canada (Rovet et al. in 2004) have suggested adverse neurodevelopmental outcomes in the offspring of mothers presenting hypothyroxinemia during pregnancy (at early and/or late stages). The available data are less conclusive and more difficult to interpret in women with only mild, subclinical or atypical hypothyroidism. The strength of both the fetal problems as well as the neurodevelopmental associations is stronger in women who have overt hypothyroidism than in those with subclinical or atypical hypothyroxinemia. The neuropsychological consequences of maternal hypothyroxinemia in the progeny probably represent a multifactorial condition. Presently available observations may be explained in part by the obstetrical consequences of undiagnosed (or undertreated) hypothyroidism and in part by the direct consequences of an insufficient maternal transfer of thyroid hormones to the developing brain during early gestation. Finally, crucial environmental factors are also important to recognize, such as the role of undiagnosed hypothyroidism during several months (or years) after parturition.

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