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Maternal and Foetal Outcomes in Overt Hypothyroidism Occuring During Pregnancy

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Abstract

Thyroid disorders are the most common endocrine disorders occurring during pregnancy in women. Development of overt or subclinical hypothyroidism in mother can lead to adverse and detrimental outcomes during pregnancy and abnormal development outcomes for the foetus. Uncontrolled maternal hypothyroidism leads to a plethora of adverse outcomes during pregnancy which include miscarriage, pregnancy induced hypertension, post partum hemorrage, abruption placentae and increased mortality of the foetus. Adverse foetal outcomes resulting from maternal thyroid dysfunction include preterm birth, low birth weight (LBW), neonatal respiratory distress syndrome, perinatal morbidity and mortality and cognitive impairment in the new-born.

Keywords: *Overt hypothyroidism; Pregnancy; Complications*

Introduction

Maternal hypothyroidism is the most common endocrine disorders occurring in pregnant women around the world. The prevalence of overt hypothyroidism during pregnancy varies from 0.4% to 11% world over. [1] In a cross sectional, multi centric study which was conducted in 11 cities from nine different states of India, the prevalence of hypothyroidism in pregnant women in India was found to be 14.3% [2]. Untreated hypothyroidism or poorly treated hypothyroidism in pregnancy can have detrimental effects both on the mother and her developing foetus. The mother is at increased risk of developing pregnancy induced hypertension, premature birth, low birth weight baby (LBW), foetal distress during labour, foetal death, neuro-cognitive deficits in children and lower IQ of the baby. [3]

Changes in Thyroid Gland Physiology in Pregnancy

Pregnancy has a huge impact on the physiology of the thyroid gland. Thyroid hormones are bound to three proteins in serum for their transport. These three proteins are thyroxine-binding globulin (TBG), transthyretin, and albumin. Pregnancy increases the hepatic synthesis of Thyroxin Binding Globulin (TBG). This marked increase in TBG leads to an elevation in total T3 and T4 levels.

Secondly, human chorionic gonadotrophin shares structural homology with TSH. A very high level of circulating chorionic gonadotrophin in the first trimester of pregnancy leads to CG cross-reactivity with the TSH receptor, leading on to a temporary increase in free T [4] and partial suppression of TSH.

The glomerular filtration rate increases in pregnancy leading to increased excretion of iodine in urine and thereby raising the daily iodine needs of pregnant women to 250 micrograms compared to 150 micrograms in non-pregnant women. Women who are staying in iodine sufficient areas and also have adequate iodine intake during pregnancy tend to have adequate iodine stores in the body and they easily adapt themselves to the increased demand for thyroid hormone during pregnancy. However, in areas of iodine deficiency along with poor oral intake of iodine, total-body iodine stores decrease gradually and these women are at risk of developing overt or sub clinical hypothyroidism [4].

Pregnancy Outcome in Overt Hypothyoidism

One of the very first clinical studies looking at maternal and foetal pregnancy outcomes in overt hypothyroid women was performed in 1981 by Montoro et al [5]. It was a small study included just nine women with extremely severe hypothyroidism in pregnancy. The mean serum TSH level was more than 100.0 mIU/l). In this landmark study, the authors concluded that even women with severe hypothyroidism may conceive normally and are able to sustain pregnancy. In this small study, there was one case of fetal loss in 29 weeks of gestational. In 1993, the same authors published a more extensive study of 68 women with hypothyroidism which was overt in 23 cases and subclinical in 45 women [6]. They found that women with persistent hypothyroidism at delivery developed gestational hypertension and, consequently, gave birth to premature infants with low birth weight. The authors concluded that the normalisation of thyroid function during pregnancy may prevent such complications.

Goel et al. showed a higher risk of foetal distress in mothers with clinical hypothyroidism [7]. It seems that hypothyroidism exerts irreversible in¬fluences on the placenta and foetus during pregnancy and decreases the foetal ability to tolerate stress and therefore, neonates present with low Apgar scores at birth [8]. In a study done by Allan et al in 9403 women with single pregnancy, (including 37 women with a TSH level above 10.0 mIU/l), they found a positive correlation between serum TSH level ≥10.0 mIU/l and increased rate of foetal death [9]. However, the limitation of this study was that their thyroid function test analysis was based on a single estimation of serum TSH which was obtained during the second trimester as part of routine antenatal care.

Regional and Trimester Specific Reference Ranges for Pregnant Women

During pregnancy a lot of variations occur in thyroid functions tests and therefore for interpretation of results in pregnancy, method specific and gestation specific reference ranges must be used.

Ideally, population-based trimester-specific reference ranges for serum TSH should be defined through assessment of local population data. To form such reference ranges, pregnant women with no known thyroid disease, with optimal iodine intake (with normal urinary iodine levels), and negative Thyroid peroxidase antibody status only should be included [10].

Where ever local or regional assessments are not available, the American Thyroid Association recommends that the treating physicians should follow trimester-specific reference ranges and cutoffs. In the first trimester, the lower reference range of TSH can be reduced by approximately 0.4 mU/L, while the upper reference range is reduced by approximately 0.5 mU/L. For the typical patient in early pregnancy, this corresponds to a TSH upper reference limit of 4.0 mU/L. [11]

Beneficial effects of thyroid hormone replacement on pregnancy outcomes of hypothyroid mothers.

In order to prevent adverse maternal and fetal outcomes, women with overt hypothyroidism must be treated with levothyroxine replacement therapy with the dose titrated in such a manner so as to achieve a TSH concentration within the trimester specific reference range. According to American Thyroid Association-2017 guidelines, overt hypothyroidism should be treated as soon as detected during pregnancy, because adequate thyroid hormone is needed for pregnancy and normal foetal brain development11. If hypothyroidism has been diagnosed before pregnancy and is being treated with levothyroxine, dose adjustments will be needed during pregnancy. TSH should be measured prior to conception if that is not possible it should be measured early in pregnancy to rectify and treat maternal hypothyroidism at the earliest.

Conclusion

Overt hypothyroidism in pregnancy has several adverse effects on pregnancy outcomes. The long-term effects of untreated hypothyroidism on neuro cognitive function of the child have been well documented. Meticulous treatment and follow-up is important for mothers suffering from hypothyroid to improve pregnancy outcomes.

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