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Subclinical Hypothyroidism in Pregnancy: A Review

[1].Dr. Ritimukta Panda, [2]. Dr. Kavya Sudha [3]. Dr. K.Jaya Sri, [4]. Dr. Rachita Sarangi, [5.] Dr. Bhagirathi Kar, [6]. Dr. Gangadhar Sahoo

[1,2,3,] PG student, Department of gynaecology and obstetrics , IMS and SUM Hospital, Siksha "O" Anusandhan University (Deemed to be), K8, Kalinganagar, Bhubaneswar-751003, Odisha, Bhubaneswar

[4] Professor, Department of paediatrics, IMS and SUM Hospital, Siksha "O" Anusandhan University (Deemed to be), K8, Kalinganagar, Bhubaneswar-751003, Odisha, Bhubaneswar

[5] Professor, Department of Medicine, IMS and SUM Hospital, Siksha "O" Anusandhan University (Deemed to be), K8, Kalinganagar, Bhubaneswar-751003, Odisha, Bhubaneswar

[6] (corresponding author), Dean, IMS and SUM Hospital, Siksha "O" Anusandhan University (Deemed to be), K8, Kalinganagar, Bhubaneswar-751003, Odisha,Bhubaneswar

[1]ritimuktapanda@soa.ac.in

[4]rachita Sarangi@soa.ac.in

[5]bhagirathi Kar@soa.ac.in

[6]dean.ims@soa.ac.in

Abstract

Pregnancy-reference levels of Subclinic hypothyroidism (SCH), along with normal level of serum thyroxine, are called high thyroid stimulation hormone level (TSH).

Autoimmune Thyroiditis is also common in patients of subclinical hypothyroidism. Subclinical Hypothyroidism in pregnancy is the cause of some adverse obstetric consequences. Changes in the metabolism of thyroid hormones during pregnancy needs to be kept in mind, while diagnosing thyroid abnormalities. There is a jump in the obstetric and neonatal results, like preterm delivery, miscarriage, fetal growth restriction, preeclampsia, gestational diabetes mellitus, low birth weight, abruptio placentae and poor Apgar scores at birth. Treatment with Cevothyroxine therapy may help reduce some of these adverse effects, however there is restricted evidence to provision it. The behavior of subclinical hypothyroidism should target maternal TSH concentrations of less than 2.5mIU/L. However there is a lack of recommendation for the official starting dose of levothyroxine. So, individualised low doses of levothyroxine can be started and thereafter titred to the maintain the TSH in the target level

Keywords: Subclinical hypothyroidism, pregnancy, screening, levothyroxine, TSH

1. INTRODUCTION:

Subclinical Hypothyroidism determined by a normal level of serum thyroxine (T4) concentration with raised serum thyroid stimulating hormone (TSH) overhead the pregnancy related exact variety[1].

In an epidemiology study done by Unnikrishnan et al. in eight Indian cities, the occurrence of subclinical hypothyroidism (SCH)was 8.02%. it was 11.4% for women and 6.2% for men in India.

Changes in thyroid function in pregnancy

The plasma volume increases by 40% and there is increased production of thyroid binding globulin.[4]The placenta secretes hCG, which has an alpha subunit similar to TSH, therefore reacts as a TSH agonist. Therefore, its higher levels lead to gestational transient hyperthyroxinaemia, which is observed in 0.3% of pregnancies[2][3].

This results in decrease in the level of TSH in early pregnancy. Thyroid gland is below pressure in pregnancy, leading to hypothyroidism among women with reduced thyroid reserve or deficient in iodine.

The renal clearance of iodine has been enhanced. Placenta deiodines T3 and T4 are increased serum thyroxine binding globulines.

The level of thyroxine may be affected by the changes in metabolism. Increased level of thyroxine is noticed in In the 2nd and 3rd trimesters, the first anditdecreases.Thyroxine is necessary for the growth of various fetal organs, mainly the fetal skeleton and brain. Thyroxine in early trimester is provided by the maternal thyroid, till the fetal thyroid functions at 12-14 weeks of gestation[4].

Effects of subclinical hypothyroidism in pregnancy

Increased negative obstetrical risk and neonatal results, such as pre-term delivery, mistake, restriction of fetal development, pre-eclampsia, gestational diabetes mellitus,abruptio placentae and poor Apgar scores at birth have been observed in patients with sub clinical hypothyroidism in several observational studies[5].

In several major potential educations in the US and Finland no connotations of SCH and adverse obstetric findings could also be seen.

In 202 singleton pregnancies at weeks 12–14, Ashoor assessed TSH & FT4 levels. Later in the pregnancy these patients had mistaken or foetal death.

On comparing their thyroid function tests with 4318 normal pregnancies, it was observed that patients with raised TSHabove the 97.5th percentile and FT_4 levels below the 2.5th percent had either a miscarriage or fetal loss.

3995 pregnant women with SCH were analysed in a meta-analysis of 17-unit trainings determined that expecting females having SCH were prone to have loss in and a neonatal death when compared to euthyroid women[6].

A recent meta-analysis found that motherly SCH is linked to intellectual handicap indicators in children, as opposed to euthyroid indicators.The association of adverse neurocognitive development in neonates and maternal SCH has not been clearly demonstrated, though its biologically plausible[7].

Diagnosis of subclinical hypothyroidism

The recommended values of references of TSH in In the first quarter, different quarters are 0.2–2.6 mIU / L; in the third quarter are 0.3–3.5 mIU / L; in IIIrd trimester, 0.4–4.0 mIU/L. T4 for dialysis or ultrafiltrate serum, with online extraction, tandem mass / liquid / fluid chromatography is the best way to test FT4 serum during pregnancy.serum FT4. However, Serum TSH is found to be a better indicator of thyroid function status in pregnancy[8][9].

Responsive hypothyroidism (OH) with decreased FT4 concentration is referred to as increased TSH (> 2.5 mIU / L). It is also said that OH is used in patients with TSH > 10.0 mIU / L independent of their FT4 stage. A serum TSH between two and 10 mIU / L with a typical FT4 concentration is considered subclinical hypothyroidism.

In nearly 50 percent of females with SCH and almost 80 percent of those with apparent hypothyroidism, thyroid self-anticbodies are found. Evaluating for the existence of thyroid peroxidase antibodies (TPOAb) is recommended in pregnant women with SCH to Determine the autoimmunity of the woman with thyroid.

Treatment of Subclinical hypothyroidism

There isn't adequate evidence for/against the recommendation of universal levothyroxine treatment in women with pregnancy having SCH, negative for TPOAb, which is due to the lack of randomized control trails.

In 2007, a study conducted by the American congress of gynecologists and obstetricians did not find enough evidence for the treatment of SCH among pregnant women. The American Thyroid Association (ATA) subsequently proposed SCH treatment in 2011 during pregnancy only if women have positive TPOAb[(6)]. The endocrine society recommended universal treatment of all pregnant women with SCH[10].

A retrospective study found that treatment of women having SCH with LT4 was related to lower risk with low birth weight as well as low Apgar score, without statistically significant difference in other outcomes. Treatment with LT4 is suggested for pregnant females with optimistic TPOAb when TSH is more than 4.0mIU/L and might consider for Women who are pregnant with TPOAb positive if TSH is greater than 2.5mIU / L or women with TPOAb positive if TSH is 4.0-10.0mIU / L.

Since there is a lack of official recommendation regarding the initial dose of LT4 for patients with SCH newly diagnosed during pregnancy, initiating treatment with a low dose such as 50mcg of LT4 and titrating according to requirement seems like a good approach[11].

The recommended treatment by ATA is to target maternal TSH concentrations of less than 2.5mIU/L. It is suggested to repeat the thyroid profile once monthly during the first 20 weeks of pregnancy and atleast once at around 30weeks of gestation. It is suggested by the endocrine society to repeat the thyroid profile once in 4 - 6 weeks throughout gravidness and to regulate the amount of LT4 for the maintenance if TSH within the goal ranges of each trimester[12].

In the postpartum period, dosage of LT4 must be decreased to the dose used prior to conception. thyroid profile must be done 6 weeks after delivery. LT4 can be discontinued in the postpartum period for those patients in who it was started during pregnancy, particularly when the dose is less than 50mcg.

2. CONCLUSION:

The correlation between SCH and specific adverse neonatal and pregnancy results has been well established. However, there is a need to conduct randomized trials in patients with SCH to assess the benefit of LT4 as an intervention during early stages of pregnancy or prior to conception. In the current scenario, the doctors and pregnant women with SCH appears to have confusion as to the effects of thyroid hormone therapy on mother and neonate outcomes

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