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A STUDY OF GROWTH PARAMETERS IN THE BABIES BORN TO HYPOTHYROID MOTHERS

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Abstract

Background: Normal maternal thyroid function is crucial for fetal growth and neurocognitive development. Intrauterine growth restriction (IUGR) is a multifactorial condition resulting from maternal, placental, or fetal factors. Any imbalance in maternal thyroid function may adversely affect both mother and fetus. **Aim:** To assess the correlation between thyroid-stimulating hormone (TSH) levels and clinically diagnosed cases of IUGR. **Materials and Methods:** A prospective study was conducted over one year at a tertiary care hospital, involving 120 consecutive clinically suspected singleton IUGR pregnancies between 34–40 weeks of gestation. The reference range for TSH in the third trimester was considered to be 0.3–3.0 mIU/L. TSH levels were recorded and subjected to descriptive analysis. **Results:** Of the 90 confirmed IUGR cases, three antenatal mothers from rural backgrounds had TSH levels below 12.6 mIU/L. All were vegetarians. Among these hypothyroid mothers, one baby was stillborn, while the other two were delivered via caesarean section with very low birth weights (<1.5 kg) and required neonatal intensive care unit (NICU) admission. **Conclusion:** As the etiology of IUGR is multifactorial, TSH levels alone may not serve as a reliable biomarker for its prediction.

Keywords: thyroid, hypothyroidism, TSH, Pregnancy

Introduction

Pregnancy exerts a significant influence on the thyroid gland and its function. In iodine-sufficient regions, the thyroid gland enlarges by approximately 10% during pregnancy, whereas in iodine-deficient areas, the increase may range from 20% to 40%. To meet the heightened metabolic demands of pregnancy, the thyroid undergoes adaptive changes in hormone production and regulation via the hypothalamic-pituitary-thyroid axis.[1] As a result, thyroid function test values in healthy pregnant women differ from

those in non-pregnant women, necessitating the use of pregnancy-specific—and ideally trimester-specific—reference intervals for accurate interpretation.[2]

Intrauterine growth restriction (IUGR) is a complex but prevalent fetal growth disorder in modern obstetric practice, resulting from either intrinsic fetal abnormalities or external adverse influences.[3] It presents a significant clinical and public health concern, particularly in developing countries.[4] IUGR is diagnosed when the estimated fetal weight falls below the 10th percentile for the corresponding gestational age, often accompanied by clinical signs of fetal malnutrition.[5] Globally, IUGR affects approximately 24% of newborns, accounting for an estimated 30 million infants annually.[6]

In Asia, up to 75% of infants are affected by IUGR.[7–8] In India, the National Neonatal Perinatal Database reports an IUGR incidence of 9.65% among hospital-born live births, while a recent UNICEF survey estimates the rate at 25–30%.[6–9]

Thyroid disorders are the second most common endocrine dysfunctions encountered during pregnancy.[10] Hypothyroidism may be either overt or subclinical. Overt hypothyroidism is characterized by elevated TSH levels (≥10 mIU/L) and reduced T4/free T4 (FT4), whereas subclinical hypothyroidism shows elevated TSH (≤10 mIU/L) with normal T4/FT4 levels. The estimated prevalence of overt hypothyroidism in pregnancy is 0.3–0.5%, and that of subclinical hypothyroidism is 2–3%.[10] During pregnancy, metabolic demand increases, leading to enhanced basal metabolic rate and increased thyroid hormone secretion, influenced by human chorionic gonadotropin (hCG) and human chorionic thyrotropin, which share molecular similarities and receptor cross-reactivity with TSH.[11]

The prevalence of IUGR is reported to be 25% in cases of overt hypothyroidism and 8% in subclinical hypothyroidism. Currently, there are no reliable predictive interventions for IUGR. Therefore, the evaluation of thyroid-stimulating hormone (TSH) as a potential biomarker for predicting IUGR is relevant. This study aims to explore the correlation between low maternal TSH levels and the occurrence of IUGR.

Materials and Methods

This prospective, observational study was conducted at a tertiary care hospital over a one-year period from 2024 to 2025, following approval by the institutional ethics committee. A total of 90 consecutive, clinically suspected cases of singleton IUGR pregnancies between 34 and 40 weeks of gestation were enrolled after obtaining informed consent.

Gestational age was determined based on the last menstrual period (LMP) or first-trimester ultrasound findings when available. IUGR was clinically diagnosed based on poor maternal weight gain and fundal height measurements that were inconsistent with the gestational age (i.e., fundal height less than expected for the period of gestation).

In all enrolled cases, serum TSH levels were measured using an automated chemiluminescence method, and the values were documented for analysis

Statistical analysis:

The study used Microsoft Excel to analyze data, representing categorical variables as frequencies and percentages, and continuous variables as mean ± SD. The t test was used to compare diabetic patients with and without nephropathy. Statistical significance was with a p-value of less than 0.05.

Results

A total of 5440 live births were recorded in our tertiary care hospital from 2024 to 2025, and 90 consecutive women with singleton pregnancies having clinical suspicion of intrauterine growth restriction and a gestational age of 34 weeks or beyond were enrolled in the study. Approximately 61.1% (n=55) were from the rural background and 38.9% (n=35) were primigravida. The mean age of the subjects was 26.8 years ± 3.8 SD. 42.2 percent of the antenatal mothers were in the age group of 20-25 years (n=38) followed by 44.4% in the age group of 26- 30 years as shown in Table 1. All the 90 study participants were non-smokers and non- alcoholics while 72.2% (n=65) were non-vegetarians

Table 1: Age distribution in study population

S. No.	Age	Number (n=90)	Percentage (%)
1	<20 years	5	5.6
2	21-25 years	38	42.2
3	26-30 years	40	44.4
4	>31 year	7	7.8

Table 2: Demographic characteristics of the study population

S. No.	Demographic	Number (n=90)	Percentage (%)
1	Rural	55	61.1
2	Urba	35	38.9

Table 3: Body mass index of the study population

S. No.	BMI (Wt /Ht2)	Reference	Number (n=90)	Percentage (%)
1	Underweight	<18.	19	21.1
2	Overweight	>2	1	1.1
3	Normal weight	8.5-2.5	70	77.8

The Body Mass Index (BMI) ranged from 14.2 kg/m2 to 25.5 kg/m2 with the average BMI being 19.24 kg/m2 The BMI of <18.5 kg/m2 was observed in 19 underweight antenatal mothers (21.1%) whereas only one (1.1%) of the subjects evaluated was overweight with a BMI of 25.5 kg/m2 (Table 3). The average height of the subjects was 151.2 cm ± 8.9 SD and the average pre-pregnancy weight as recorded was 50.4kg ±

14.3 SD. Among 90 deliveries, 58 women had normal vaginal delivery while 32 underwent lower segment caesarean section.

Out of 32 caesarean delivery, 21 caesareans were done due to fetal distress out of which 17 (53.1%) were conducted for non-reassuring fetal heart rate. Other causes were failed induction and poor Manning score. 28 neonates required admission to neonatal intensive care unit (NICU). The major cause was respiratory distress syndrome (n=14) 43.8%. Other causes for admission included hypoglycaemia, neonatal jaundice, and meconium aspiration syndrome in decreasing order. The neonates were admitted to NICU for about 2 to 5 days.

The TSH levels below 0.3 mIU/L was recorded in Two antenatal mothers in the age-group of 26-30 years. All two antenatal mothers were from rural background and vegetarian. In these, one was born dead while the other one babies were born alive. The antenatal mother with normal BMI delivered live babies. However, the mode of delivery in the two born alive was LSCS due to fetal distress. Both neonates had a very low birth weight; less than1.5 kgs and required admission to neonatal intensive care unit for 2-5 days.

Discussion

Thyroid function undergoes significant physiological changes during pregnancy, yet data on pregnancy outcomes in women with both intrauterine growth restriction (IUGR) and hypothyroidism remain limited. Maternal thyroid dysfunction is associated with increased maternal and fetal morbidity, and IUGR itself is linked to adverse perinatal outcomes.[12] During pregnancy, the maternal thyroid gland is under increased physiological stress, characterized by elevated thyroxine-binding globulin, increased iodine demand, and stimulation by human chorionic gonadotropin (hCG).[13] In the first trimester, the fetus relies entirely on maternal thyroxine, which crosses the placenta and is converted to triiodothyronine. This hormone is essential for fetal growth, as well as brain and lung development.[13]

Borderline hypothyroid women may progress to subclinical or overt hypothyroidism during pregnancy. The prevalence of hypothyroidism in pregnancy is estimated to be 2–3%, with 0.3–0.5% presenting as overt hypothyroidism and 2–2.5% as subclinical hypothyroidism.[14] Studies suggest that thyroid dysfunction is more common in females than males in rural populations, although not always statistically significant.[15–16]

In our study, both hypothyroid antenatal mothers were from rural backgrounds and were vegetarians. Some literature suggests that a vegan diet may be associated with a lower risk of hypothyroidism, although the evidence is mixed.[16] Body mass index (BMI) typically increases during pregnancy due to weight gain. Previous studies have shown a positive correlation between BMI and TSH levels in the first and second trimesters, and a negative correlation with free T4 (FT4) in later trimesters, although no significant association was found with FT3.[17] In our study, one mother had a low BMI, while the others had normal weight.

Early diagnosis and treatment of hypothyroidism during pregnancy are essential. TSH remains the most widely used, reproducible, and reliable test for detecting thyroid

dysfunction, though interpretation requires trimester-specific reference ranges to avoid underdiagnosis of hypothyroidism or overdiagnosis of hyperthyroidism. According to the 2012 American Thyroid Association guidelines, the TSH reference ranges are: First trimester: 0.1–2.5 mIU/L, Second trimester: 0.2–3.0 mIU/L, Third trimester: 0.3–3.0 mIU/L[18–19]

A study by Ruchi Kishore et al. found that among hypothyroid pregnant women, 13% had IUGR and 6.2% experienced intrauterine demise.[18] Other researchers have conducted fetal blood sampling (cordocentesis) in severe IUGR cases and reported significantly reduced levels of free T3 and T4. In our study, three mothers had TSH levels below 12.6 mIU/L. Of these, one delivered a stillborn baby, and the two live births were delivered via lower segment cesarean section (LSCS) due to fetal distress. Both neonates had a birth weight under 1.5 kg and required neonatal intensive care.

The search for reliable biomarkers to predict IUGR continues. IUGR is multifactorial, and while various serum markers—originally used for prenatal screening of aneuploidy and neural tube defects—have been explored, their predictive efficacy for IUGR remains unproven. TSH alone may not be a sufficient standalone biomarker, but it could have potential when combined with other indicators. While overt hypothyroidism (TSH >10 mIU/L with low FT4) is clearly treated during pregnancy, the management of subclinical hypothyroidism remains controversial due to uncertain benefits. This ambiguity has led to ongoing debate over universal screening for thyroid dysfunction in pregnancy.

Conclusion

Several studies suggest a possible association between thyroid dysfunction and IUGR; however, the results remain inconsistent and inconclusive. While overt hypothyroidism is widely acknowledged to increase the risk of adverse pregnancy outcomes, including IUGR, the utility of TSH as a standalone biomarker for predicting IUGR is limited. Given the multifactorial nature of IUGR, relying solely on TSH for prediction is insufficient. Future large-scale, multicenter studies incorporating trimester-specific thyroid reference ranges and other potential biomarkers are needed to better understand the complex interplay between thyroid function and fetal growth.

Reference

1. Alexander EK, Pearce EN, Brent GA, Brown RS, Chen H, Dosiou C et al. 2017 guidelines of the American thyroid association for the diagnosis and management of thyroid disease during pregnancy and the postpartum. Thyroid. 2017;27(3):315-89.
2. Wiles KS, Jarvis S, Nelson-Piercy C. Are we overtreating subclinical hypothyroidism in pregnancy? BMJ. 2015;351:h4726.
3. Faraci M, Renda E, Monte S, Prima FD, Valenti O, Domenico RD, et al. Fetal growth restriction: current perspectives. *J Prenat Med*. 2011;5(2):31–3.
4. Sharma D, Shastri S, Sharma P. Intrauterine Growth Restriction: Antenatal and Postnatal Aspects. *Clin Med Insights Pediatr*. 2016;10:67–83. doi:10.4137/cmped.s40070.

5. Sharma D, Chandnani K. Clinical study of IUGR cases and correlation of Doppler parameters with perinatal outcome. *Int J Reprod, Contracept Obstet Gynecol*. 2016;5:4290–6.
6. United Nations Childrens Fund and World Health Organization, Low Birth weight: Country, regional and global estimates. New York, USA 2004.
7. Onis MD, Blössner M, Villar J. Levels and patterns of intrauterine growth retardation in developing countries. *Eur J Clin Nutr*. 1998;52:5–15.
8. Singh M. Disorders of weight and gestation. In: Singh M, editor. Care of the Newborn. vol. 1999. New Delhi: Sagar Publications; 1999. p. 224–45.
9. Kady SM, Gardosi J. Perinatal mortality and fetal growth restriction. *Best Pract Res Clin Obstet Gynaecol*. 2004;18(3):397– 410.
10. Ramprasad M, Bhattacharyya SS, Bhattacharyya A. Thyroid disorders in pregnancy. *Indian J Endocrinol Metab*. 2012;16(2):167–70.
11. Khadilkar S. Thyroid-Stimulating Hormone Values in Pregnancy: Cutoff Controversy Continues? *J Obstet Gynecol India*. 2019;69(5):389–94.
12. Sahu MT, Das V, Mittal S, Agarwal A, Sahu M. Overt and subclinical thyroid dysfunction among Indian pregnant women and its effect on maternal and fetal outcome. *Arch Gynecol Obstet*. 2010;281(2):215– 20.
13. Saki F, Dabbaghmanesh MH, Ghaemi SZ, Forouhari S, Omrani GR, Bakhshayeshkaram M. Thyroid Function in Pregnancy and Its Influences on Maternal and Fetal Outcomes. *Int J Endocrinol Metab*. 2014;12(4):19378.
14. Cignini P, Cafà EV, Giorlandino C. Thyroid physiology and common diseases in pregnancy:review of literature. *J Prenat Med*. 2012;6(4):64–71.
15. Padmanabhan P. Study of thyroid profile estimations by immunodiagnosis in rural population. *GJRA*. 2019;8(3):3970–5.
16. Tonstad S, Nathan E, Oda K, Fraser G. Vegan Diets and Hypothyroidism. *Nutrients*. 2013;5(11):4642–52.
17. Kumar S, Chiinngaihlun T, Singh MR, Punyabati O. Correlation of Body Mass Index (BMI) with Thyroid Function in Euthyroid Pregnant Women in Manipur, India. *J Clin Diagn Res*. 2017;11(4):13–5.
18. Stagnaro-Green A, Abalovich M, Alexander E, Azizi F, Mestman J, Negro R, et al. American Thyroid Association Taskforce on Thyroid Disease During Pregnancy and Postpartum. Guidelines of the American Thyroid Association for the diagnosis and management of thyroid disease during pregnancy and postpartum. *Thyroid*. 2011;21(10):1081–1125.
19. Kishore R, Mishra N, Yadav J. Hypothyroidism in pregnancy and its impact on maternal and fetal outcome. *J Evol Med Dent Sci*. 2015;4(79):13849–55.