

A Study on Thyroid Stimulating Hormone Levels among Pregnant Women

Salma Akhtar Walida,¹ Kamrun Naher,² Shamima Nasrin,³ Salma Parvin,⁴ Shahjada Mohammad Dastegir Khan,⁵

ABSTRACT

Background & objective: The thyroid gland is critical for maintaining metabolism and ensuring normal fetal neurological development. A deficiency of thyroid hormone during pregnancy can lead to irreversible neurological deficits and mental retardation in the child. Maternal hypothyroidism can also cause pregnancy complications such as pre-eclampsia and preterm delivery. The present study was designed to screen for maternal hypothyroidism by assessing serum thyroid-stimulating hormone (TSH) levels in pregnant women during their second and third trimesters.

Methods: This cross-sectional study was conducted at the Antenatal Clinic of Mymensingh Medical College Hospital from September 2004 to June 2005. A total of 100 pregnant women in their second and third trimesters were consecutively included. Serum TSH was measured using an immunoradiometric assay. For participants in the 2nd and 3rd trimesters, TSH levels below 0.2 mIU/L and above 3.0 mIU/L were considered decreased and raised respectively.

Results: The mean age of the participants was 25.4 ± 5.1 years. A majority (82%) of the women were in their third trimester. The mean serum TSH level was 1.90 ± 1.32 mIU/L. The study found that 96% of the pregnant women had normal TSH levels, while 3% had decreased and 1% had raised TSH levels. The distribution of participants by trimester showed a strong skew towards the third trimester, with 82% of women in their third trimester and 18% in their second trimester.

Conclusion: The findings suggest that the prevalence of abnormal TSH levels in pregnant women visiting the Mymensingh Medical College Hospital antenatal clinic is low. However, the high proportion of women attending their first antenatal visit in the third trimester underscores the need for public awareness campaigns to encourage earlier prenatal care. A comprehensive screening program for pregnant mothers is recommended to ensure timely diagnosis and treatment, thereby preventing potential adverse outcomes for both the mother and the fetus.

Key words: Thyroid stimulating hormone (TSH), pregnant women, antenatal clinic etc.

Authors' information:

¹ **Dr. Salma Akhtar Walida**, FCPS (Obstetrics and Gynecology), FCPS (Gynaecological Oncology), Associate Professor, Department of Gynaecological Oncology, National Institute of Cancer Research & Hospital (NICRH), Dhaka, Bangladesh.

² **Prof. Dr. Kamrun Naher**, FCPS (Obstetrics and Gynecology), HOD, Department of Obstetrics and Gynecology, Mymensingh Medical College, Mymensingh, Bangladesh.

³ **DR. Shamima Nasrin**, Assistant Professor, Department of Gynaecological Oncology, FCPS(Obstetrics and Gynecology), FCPS (Gynaecological Oncology), MCPS(Obstetrics and Gynecology), National Institute of Cancer Research & Hospital (NICRH), Dhaka, Bangladesh.

⁴ **Dr. Salma Parvin**, MS (Obstetrics and Gynecology), FCPS(Obstetrics and Gynecology), FCPS(Gynaecological Oncology), Junior Consultant, National Institute of Cancer

⁵ **Dr Shahjada Mohammad Dastegir Khan**, MD(Neurology), Associate Professor, Department of Neurology, Sir Salimullah Medical College & Mitford Hospital, Dhaka, Bangladesh.

Correspondence: Dr. Salma Akhtar Walida, Phone: +8801711736732 E-mail: sa982056@gmail.com

INTRODUCTION:

The thyroid gland maintains optimal metabolism and is essential for normal growth and maturation. Thyroid hormones, like thyroxin (T4), stimulate oxygen consumption, regulate metabolism, and are necessary for healthy development¹. A deficiency in T4 in utero can cause clinical, mental retardation in humans². T4 is vital for neuron development from the second trimester of intrauterine life to the second postnatal year, and its deficiency during this period can lead to defective development of dendrites and dendritic spines^{3,4}.

Thyroid function is controlled by the thyroid-stimulating hormone (TSH) from the anterior pituitary¹. TSH secretion is regulated by thyrotropin-releasing hormone (TRH) and is subject to negative feedback from high circulating thyroid hormone levels. The normal TSH level in adults is 0.5 to 5 T0/L. During pregnancy, TSH levels decrease while T4 increases, which is a normal physiological change⁵. Congenital hypothyroidism is a known cause of mental retardation⁶. Symptoms are often subtle, and irreversible brain damage can occur before a diagnosis is made⁷. Studies have shown that hypothyroid infants treated before three months of age have a 70% chance of an IQ above 85, while 85% of those treated between three and seven months have definite mental deficiency⁸. TSH assays are widely used for screening pregnant women for hypothyroidism. Previous studies suggest that early detection and treatment can reduce serious consequences for both the mother and fetus⁹. When thyroid deficiency occurs in both the pregnant woman and her fetus, the child's neuropsychological development is adversely affected. Thyroid hormone must be supplied by the mother during the first trimester, as the fetus does not secrete its own until the middle trimester. In the middle and last trimesters, both the mother and fetus supply the hormone, but primarily the mother. This is evident in infants with sporadic congenital hypothyroidism, who are often normal at birth because of placental T4 passage, but if left

untreated, will become permanently mentally retarded. This understanding led to neonatal screening programs¹⁰.

The importance of maternal thyroid secretion is also evident in regions with endemic iodine deficiency. When iodine intake is very low, both the mother and fetus have poor thyroid function, leading to not only mental retardation but also neurological deficits like spasticity, ataxia, and deaf-mutism, which are not seen in sporadic congenital hypothyroidism. These abnormalities can be prevented by increasing the mother's iodine intake, but this must be done at the beginning of the second trimester¹¹. Maternal hypothyroidism, even in areas with sufficient iodine intake, can lead to decreased fertility, and in those who become pregnant, there is an increased frequency of pre-eclampsia and preterm delivery. Their infants may be small for gestational age and mildly mentally retarded. Consequences for the mother also include elevated serum TSH, goiter, and hypothyroidism. This risk is particularly high in iodine-deficient women, who may face long-term goiter and other issues. Lack of iodine also carries social and cultural consequences, as infertility and fetal wastage can affect a woman's quality of life and her role in the family^{12,13}. These risks underscore the importance of studying TSH levels during antenatal visits. The present study was therefore designed to assess TSH levels of pregnant women during antenatal care in their second and third trimesters, to screen for hypothyroidism in pregnant mothers. The findings of this study may be helpful for the obstetric-care providers to prevent potential adverse outcomes for both the mother and the fetus that result from clinical and subclinical hypothyroidism.

METHODS:

This was a cross-sectional study conducted at the Antenatal Clinic of Mymensingh Medical College Hospital from September 2004 to June 2005. The study was approved by the Ethical Review Committee of Mymensingh Medical College Hospital (MMCH). A total of 100 pregnant women at their second and third trimesters who visited

the antenatal clinic (ANC) during the study period were consecutively included in the study. Women in their first trimester were excluded because thyroid hormone deficiency is most critical for the pregnant women and their fetus in the later stages of pregnancy.

After selection of participants, 0.5 mL of venous blood was collected from the antecubital vein into a sterile test tube. The sample was sent to the nuclear medicine center at Mymensingh Medical College Hospital for TSH estimation. Serum was separated using a spectrophotometer, preserved at -20°C, and TSH was measured using an immunoradiometric assay. Normal TSH level in the first trimester of pregnancy was considered to range from 0.1–2.5 mIU/L, in the second trimester: 0.2–3.0 mIU/L, and in the third trimester: 0.3–3.0 mIU/L. As our study participants were in their 2nd and 3rd trimesters, TSH levels below 0.2 and above 3.0 mIU/L were considered decreased and raised respectively. Data were processed and analyzed using the SPSS (Statistical Package for Social Sciences) software. Descriptive statistics like frequency with corresponding percentages for categorical variables, and mean and standard deviations from the mean for continuous variables were employed to describe the characteristics of the study participants.

RESULTS:

The mean age of participating women was 25.4 ± 5.1 years. One-third (33%) of the participants were over 30 years old. In terms of socioeconomic status, the majority (58%) belonged to the middle class, followed by the upper class (23%) and lower middle class (19%). Over half (53%) had a primary level of education, while 28% had a secondary education. Regarding obstetric history, 42% were primiparous (first pregnancy), 43% had 1–3 children, 7% had 4–6 children, and 2% had more than six children. Four women reported a history of miscarriage. The distribution of participants by trimester showed a strong skew towards the third trimester, with 82% of women in

their third trimester and 18% in their second trimester.

The main finding of the study was the distribution of serum TSH levels. The mean TSH level was 1.90 ± 1.32 mIU/L. A vast majority of the participants (96%) had TSH levels within the normal range for their respective trimesters. Of the remaining participants, 3% had a decreased TSH level and 1% had a raised TSH level, indicating subclinical hypothyroidism.

Table I. Distribution of participants by their demographic characteristics

Demographic variables	Frequency	Percentage
Age (years)		
18 – 21	27	27.0
22 – 25	31	31.0
26 – 29	09	9.0
≥ 30	33	33.0
Socioeconomic status		
Higher class	23	23.0
Middle class	58	58.0
Lower class	19	19.0
Educational status		
Illiterate	19	19.0
Just can sign her name	10	10.0
Primary	53	53.0
Secondary and higher	28	28.0

Table II. Distribution of participants by obstetric characteristics

Obstetric variables	Frequency	Percentage
Parity		
Primipara	42	42.0
1 – 3	43	43.0
4 – 6	7	7.0
> 6	2	2.0
H/o of Miscarriage/Abortion		
	4	4.0
Gestational period		
2 nd trimester	18	18.0
3 rd trimester	82	82.0

Table III. Distribution of participants by serum TSH level

Serum TSH value (mIU/L)	Frequency %	Mean ± SD
Normal (0.2–3.0 mIU/L)	96(96.0)	1.90 ± 1.37
Raised (> 3.0 mIU/L)	1(1.0)	6.5
Decreased (< 0.2 mIU/L)	3(3.0)	0.13 ± 0.01

DISCUSSION

This study aimed to assess the thyroid status of pregnant women in their 2nd and 3rd trimesters visiting the Mymensingh Medical College Hospital antenatal clinic. Our findings underscore the importance of TSH as a key indicator of maternal and fetal health. The mean age of participants (25.4 ± 5.1 years) was consistent with a study by Khandakar and associates,¹⁴ whose mean age was 25.2 ± 5.3 years. However, it was slightly higher than the mean age reported by Kumar et al.¹⁵ (21.1 ± 5.2 years), suggesting a possible demographic difference in the study populations.

Our demographic data revealed a predominantly middle-class and primiparous study group. The notable finding that 82% of participants were in their third trimester suggesting that pregnant women in this region may not seek antenatal care until later in their pregnancy. This contrasts sharply with large-scale international studies by Haddow et al¹⁶ ($n = 9403$) and Klein et al¹⁷ ($n = 25000$), where participants were exclusively in their second trimester. This disparity may be a significant public health issue, as early screening is crucial for preventing adverse outcomes.

The core finding of this study is the high prevalence of normal TSH levels among the participants, with 96% of women showing TSH levels within the established normal range for their trimesters. The mean TSH level (1.90 ± 1.32 mIU/L) was comparable to Kumar et al¹⁵ (mean TSH: 1.03 ± 1.02 mIU/L) and was slightly lower than Khandakar and associates'¹⁴ study report (mean TSH: 2.56 ± 0.92 mIU/L). The low prevalence of abnormal TSH levels—only 3% with decreased TSH and 1% with raised TSH—is particularly encouraging. This finding stands in stark contrast to an earlier epidemiological study by Awwal¹⁸, who found that 39% of pregnant women in Bangladesh during 1991-92 were biochemically hypothyroid. This significant difference suggests that increased awareness and the widespread use of iodized salt have likely improved the overall thyroid health of the population over time. This shift highlights the

success of public health initiatives related to iodine deficiency.

CONCLUSION:

This study demonstrates that the vast majority of pregnant women in their second and third trimesters attending the Mymensingh Medical College Hospital antenatal clinic have normal serum TSH concentrations. The prevalence of decreased or elevated TSH levels is rare, which aligns with recent findings in areas where iodine deficiency is no longer a widespread issue. However, the findings also highlight a critical need for public awareness campaigns to encourage earlier antenatal visits, particularly during the first trimester, to ensure timely screening and intervention. Implementing a comprehensive thyroid screening program for all pregnant women is essential to identify those at risk and prevent the potentially devastating consequences of hypothyroidism for both mother and child. Future studies should be conducted with a larger sample size to further validate these findings and provide more robust data for public health policy decisions.

REFERENCES

1. Ganong WF. *Review of Medical Physiology*. 21st ed. London: Appleton & Lange; 2003:320-35.
2. Smith DW, Blizzard RM, Wilkins L. The mental prognosis in hypothyroidism of infancy and childhood: a review of 128 cases. *Pediatrics* 1957;19:1011.
3. Morreale de Escobar G, Escobar del Rey F. Neonatal screening-Brain damage and thyroid hormone. In: Burrow GN, ed. *Neonatal Screening*. New York: Raven Press; 1980:25.
4. Ruiz-Marcos A, Sánchez-Toscano F, Escobar del Rey F, Morreale de Escobar G. Severe hypothyroidism and maturation of rat cerebral cortex. *Brain Res* 1979;162:315.
5. Gardner LI. Historical notes on cretinism. In: Gardner LI, ed. *Endocrine and Genetic Diseases of Childhood and Adolescence*. Philadelphia: WB Saunders Co. 1973:234-8.
6. Smith BW, Klein AH, Henderson JR, Myrianthopoulos NC. Congenital hypothyroidism: signs and symptoms in the newborn period. *J Pediatr* 1975;87(6 Pt 1):958-62.

7. Klein AH, Meltzer S, Kenny FM. Improved prognosis in congenital hypothyroidism treated before age three months. *J Pediatr* 1972;81(5):912-5.
8. Vulsmma T, Gons MH, de Vijlder JJM. Maternal-fetal transfer of thyroxine in congenital hypothyroidism due to a total organification defect or thyroid agenesis. *N Engl J Med* 1989;321:13-6.
9. Glinoer D. The regulation of thyroid function in pregnancy: pathways of endocrine adaptation from physiology to pathology. *Endocr Rev* 1997;18:404-33.
10. Delange F. The disorders induced by iodine deficiency. *Thyroid* 1994;4:107-28.
11. Cao XY, Jiang XM, Dou ZH, Rakeman MA, Zhang ML, O'Donnell K, et al. Timing of vulnerability of the brain to iodine deficiency in endemic cretinism. *N Engl J Med* 1994;331(26):1739-1744.doi:10.1056/NEJM199412293312603
12. Glinoer D, Nayer PD, Delange F, Lemone M, Toppet V, Spehl M et al. A randomized trial for the treatment of excessive thyroid stimulation in pregnancy: maternal and neonatal effects. *J Clin Endocrinol Metab* 1995;80: 258-69. DOI: 10.1210/jcem.80.1.7829623
13. Berghout A, Wiersinga WM. Thyroid size and thyroid function during pregnancy. In: Stanbury JB, Delange F, Dunn JT, Pandav CS, eds. *Iodine in Pregnancy*. Delhi: Oxford University Press; 1998:35-53.
14. Khandakar AMR, Ali MS, Khatun M. Thyroid status of normal pregnant women in Dhaka City. *Mymensingh Med J* 2002;11(1):1-5.
15. Kumar A, Gupta N, Nath T, Sharma JB, Sharma S. Thyroid function test in pregnancy. *Indian J Med Sci* 2003;57(6):252-8.
16. Haddow JE, Palomaki GE, Allan WC, Williams JR, Knight GJ, Gagnon J, et al. Maternal thyroid deficiency during pregnancy and subsequent neuropsychological development of the child. *N Engl J Med* 1999;341(8): 549-555.doi:10.1056/NEJM199908193410801.
17. Klein RZ, Haddow JE, Faix JD, Brown RS, Hermos RJ, Pulkkinen A, et al. Prevalence of thyroid deficiency in pregnant women. *Clin Endocrinol (Oxf)* 1991;35(1):41-46. doi:10.1111/j.1365-2265.1991.tb03494.x.
18. Awwal AMMA. An Epidemiological Investigation of Maternal and Neonatal Indicators of Iodine Status in Bangladesh [PhD Thesis]. Brisbane: The University of Queensland; 1995. <https://doi.org/10.14264/afef194>.