

Research Article

Thyroid Dysfunction in Pregnancy and Preeclampsia

Manjunatha S¹, Basavaraja GN^{2*}, Ramesh S Patil³

¹ Assistant Professor, Department of Obstetrics and Gynaecology, Hassan Medical College, Hassan, Karnataka, India

² Assistant Professor, Department of Surgery, Hassan Medical College, Hassan, Karnataka, India

³ Assistant Professor, Stat, Department of Community Medicine, Ashwini Rural Medical College, Solapur

***Corresponding author**

Dr. Basavaraja G N

Email: dr_basavarajagn2008@rediffmail.com

Abstract: Pre-eclampsia is a leading cause of maternal & foetal morbidity & mortality worldwide. There is a state of hypothyroxinemia in normal pregnancy, it is more pronounced in preeclampsia. Moreover hypothyroidism has been listed as one of the causes of high blood pressure. Few studies have showed the effect on TSH levels exposing pre-eclamptic patients to the risk for low birth weight babies. Our study investigates the functioning of thyroid gland in normal pregnancy and in preeclampsia. Study was conducted on 30 diagnosed patients of preeclampsia and 30 age and parity matched normotensive pregnant subjects. Blood samples from both the groups were assayed by CLIA (chemiluminescence immunoassay) system for Thyroxine (T₄), Tri-iodothyronine (T₃) & Thyroid stimulating hormone (TSH). Comparison between both the groups was done by unpaired 't' test. There was no significant difference in the T₄ & T₃ levels in normal pregnancy and in pre-eclampsia. TSH levels in pre-eclampsia patients were increased significantly. The study shows significant increase in the TSH levels in pre-eclampsia than in normal pregnancy. The thyroid disorder is one of the predisposing causes for pre-eclampsia. Hence thyroid hormonal assay can be considered as a screening test for early diagnosis and treatment of pre-eclampsia to prevent further complications of it.

Keywords: Preeclampsia, Thyroxine (T₄), Tri-iodothyronine (T₃), Thyroid stimulating hormone (TSH)

INTRODUCTION

During normal pregnancy, changes in thyroid function are well-documented, but information about thyroid function in complicated pregnancy is scanty. It has long been recognized that maternal thyroid hormone excess or deficiency can influence maternal & fetal outcome at all stages of pregnancy and can interfere with ovulation and fertility [1, 2].

Although, pregnancy is usually associated with very mild hyperthyroxinemia, woman complicated with preeclampsia have high incidence of hypothyroidism that might correlate with the severity of preeclampsia [3]. Maternal hypothyroidism is the most common disorder of thyroid function in pregnancy, which has been associated with fetal loss, pregnancy-induced hypertension, preterm delivery, placental abruption, and reduced intellectual function in the offspring [4, 5].

During pregnancy, there is an increased thyroid demand and increased iodine uptake and synthesis of thyroid hormones. Even though there is a state of hypothyroxinemia in normal pregnancy, it is more pronounced in preeclampsia. Moreover hypothyroidism has been listed as one of the causes of high blood pressure i.e. the physiological changes in thyroid gland

during pregnancy have been suggested as one of the pathophysiologic cause of pre-eclampsia [6].

A study done to see the influence of pre-eclampsia on thyroid parameters, have suggested that pre-eclampsia has the effect on TSH levels exposing pre-eclamptic patients to the risk for low birth weight babies [7].

There are limited number of studies on the levels of thyroid hormones in preeclampsia and has been suggested that there may be an existence of mutual influences between preeclampsia and thyroid function [8].

The aim of this study is to compare the serum levels of T₄, T₃ & TSH in normal pregnancy and preeclampsia.

MATERIALS AND METHODS

This case-control study was conducted on 30 diagnosed patients of preeclampsia and 30 normal pregnant subjects in the Department of OBG, Hassan Medical College, Hassan.

Inclusion criteria

Inclusion criteria were 30 diagnosed patients of preeclampsia (BP > 140/90mmHg & proteinuria

>300mg/l in 24 hour) after 20 weeks of gestation and controls were 30 matched normotensive pregnant subjects and both group had no history of thyroid disease before pregnancy.

Exclusion criteria

The patients with the history of hypertension, renal disorders, cardiovascular diseases, any metabolic disorders before or during the pregnancy and history of intake of any medication such as levothyroxine that may affect on thyroid function were excluded from the study.

Written informed consent was obtained from all patients participating in the study and they were assured about the privacy of the data. Blood pressure measurements were recorded in sitting position using sphygmomanometer [9].

Blood pressure more than 140/90mmHg on two or more occasions at least 6 hrs apart & proteinuria >300mg/l in 24 hour were considered as preeclampsia patients.

2ml of venous blood samples were obtained from cases and controls. Blood samples from both the groups were assayed by CLIA (chemiluminescence immunoassay) system for Thyroxine (T₄), Tri-

iodothyronine (T₃) & Thyroid stimulating hormone (TSH).

Comparison between both the groups was done by unpaired 't' test. A p-value less than 0.05 were considered as significant.

RESULTS

This case control study includes 30 diagnosed patients of preeclampsia and normotensive pregnant subjects. The mean age was 24 ± 2 and 24 ± 5 yrs for normal and preeclampsia patients respectively. The mean parity was 1.87 ± 2 and 1.84 ± 1. There was no significant difference was observed in the view of mean age and parity.

Thyroxine (T₄) in normal pregnancy (9.03 ± 1.18 nmo/L) and in pre-eclampsia (10.16 ± 1.13 nmo/L) and Tri-iodothyronine (T₃) levels in normal pregnancy (1.21 ± 0.3) and in pre-eclampsia (1.25 ± 0.11), Thyroid stimulating hormone (TSH) levels in normal pregnancy (2.48 ± 1.05) and in pre-eclampsia (7.22 ± 1.3).

There was no significant difference in the T₄ (p=0.08) & T₃ (p=0.49) levels in normal pregnancy and in pre-eclampsia. TSH levels in pre-eclampsia patients were increased significantly (p=0.0001).

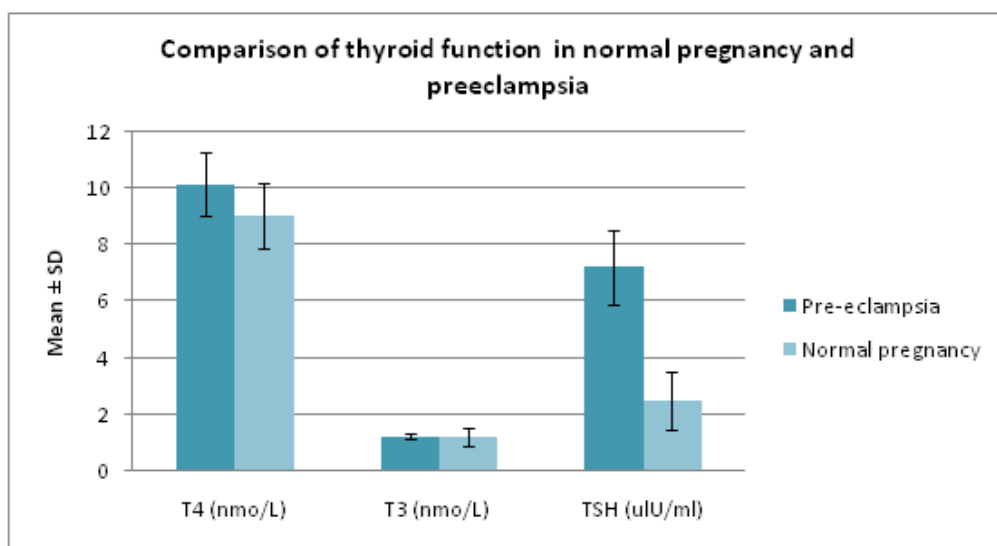


Fig. 1: Comparison of thyroid function in normal pregnancy and preeclampsia

DISCUSSION

Though the effect of preeclampsia and thyroid dysfunction in pregnancy is very well studied, the relationship between the two is poorly established. Therefore this study was undertaken to know the influence of preeclampsia on thyroid function. The thyroid hormones levels were within the normal range and did not show any statistical difference between normal pregnancy and preeclampsia. But TSH levels were higher in preeclampsia subjects which was significant.

Kumar et al observed the similar findings in preeclamptic and eclamptic women with high TSH level and low thyroid hormones their finding suggested that preeclamptic women had higher incidence of biochemical hypothyroidism compared with normotensive pregnant women [10]. It has been suggested that reduced concentration of thyroid hormones in preeclampsia may be due to the loss of protein-bound hormones in the urine [11].

Lao et al reported decreased levels of FT4 and increased levels of TSH in preeclampsia [12]. Abnormalities in placental function can interfere with oestrogen production that lead to decrease levels of TBG, T3 & T4 [13]. Hypothyroidism can cause vascular smooth muscle contraction both in systemic and renal vessels, which leads to increased diastolic hypertension, peripheral vascular resistance, and decreased tissue perfusion [14, 15].

Thyroid dysfunction can be associated with proteinuria, which is known to result in increased excretion of thyroxine and thyroid-binding globulins [16]. Endothelial activation/dysfunction is a central pathogenic feature in women with preeclampsia, which is a multiple system disorder during human pregnancy [17]. Increased circulating VEGF-1 concentration in preeclamptic women were associated with decreased circulating levels of free VEGF and PlGF, leading to an anti-angiogenic state and causing endothelial cell dysfunction [18]. There is strong evidence that TSH can act as a tissue specific angiogenesis in physiological and pathological conditions. Thus increases levels VEGF and TSH protein correlated with each other. TSH up regulates VEGF expression in vivo and vitro [19].

CONCLUSION

In the present study TSH levels were higher in preeclampsia subjects compared to normal pregnant women, which could indicate the possible aetiology for preeclampsia. Elevated TSH levels could be used as predictor of preeclampsia. Women who develop preeclampsia are more like to have decreased thyroid function. Thyroid function screening should be done in first trimester of pregnancy for early diagnosis and treatment of preeclampsia to prevent further complication of it.

REFERENCES

1. Glinoe D; The regulation of thyroid function in pregnancy: pathways of endocrine adaptation from physiology to pathology. *Endocr Rev.*, 1997; 18: 404-433.
2. Casey BM, Leveno KJ; Thyroid diseases in pregnancy. *Obstet Gynecol.*, 2006; 108: 1283-1292.
3. Hwang HS, Maeng YS, Park YW, Koos BJ, Kwon YG, Kim YH; Increased senescence and reduced functional ability of fetal endothelial progenitor cells in pregnancies complicated by preeclampsia. *Gynecol Obstet Invest.*, 1994; 37: 30-33.
4. Casey BM, Dashe JS, Wells CE, McIntire DD, Byrd EW, Leveno KJ et al.; Subclinical hypothyroidism and pregnancy outcomes. *Obstet Gynecol.*, 2005; 105: 239-245.
5. Abalovich M, Gutierrez S, Alcaraz G, Maccallini G, Garcia A, Levalle O; Overt and subclinical hypothyroidism complicating pregnancy. *Thyroid*, 2002; 12: 63-68.
6. Endo T, Komiya I, Tsukkui T; Reevaluation of a possible high incidence of hypertensive in hypothyroid patients *Am Heart J.*, 1979; 98: 684-688.
7. Dhananjaya BS, Kumaran DS, Venkatesh G, Murthy N, Shashiraj H; Thyroid Stimulating Hormone (TSH) Level as a Possible Indicator of Pre-eclampsia. *Journal of Clinical and Diagnostic Research*, 2011; 5(8): 1542-1543.
8. Vojdodic LJ, Sulovic V, Pervulov M, Milacic D, Terzic M; The effect of preeclampsia on thyroid gland function. *Srp Arh Celok Lek.*, 1993; 121(1-2): 4-7.
9. Abalovich M, Gutierrez S, Alcaraz G, Maccallini G, Garcia A, Levalle O; Overt and subclinical hypothyroidism complicating pregnancy. *Thyroid*, 2002; 12: 63-68.
10. Kumar Ashok, Ghosh BK, Murthy NS; Maternal thyroid hormonal status in preeclampsia. *Indian Journal of Medical Sciences*, 2005; 59(2): 57-63.
11. Kaya E, Sahin Y, Ozkececi Z, Pasaoglu H; Relation between birth weight and thyroid function in preeclampsia eclampsia. *Gynecol Obstet Invest.*, 1994; 37: 30-33.
12. Lao TT, Chin RKH, Swaminath R, Lam YM; Maternal thyroid hormones and outcome preeclamptic pregnancies. *Br J Obstet Gynecol.*, 1990; 97: 71-74.
13. Skjoldbrand L, Brundin J, Carlstrom A, Petterson T; Thyroxin binding globulin in spontaneous abortion. *Gynecol Obstet Invest.*, 1986; 21: 187-192.
14. Alfadda A, Tamilia M; Preeclampsia-like syndrome that is associated with severe hypothyroidism in a 20-week pregnant women. *American Journal of Obstetrics and Gynecology*, 2004; 191(5): 1723-1724.
15. Negro R, Mestman JH; Thyroid disease in pregnancy. *Best Practice & Research: Clinical Endocrinology & Metabolism*, 2011; 25(6): 927-943.
16. Patel S, Robinson S, Jbidgood R, Edmonds CJ; A preeclampsia-like syndrome associated with hypothyroidism during pregnancy. *Quarterly Journal of Medicine*, 1991; 79(289): 435-441.
17. Chelbi ST, Vaiman D; Genetic and epigenetic factors contribute to the onset of preeclampsia. *Molecular and Cellular Endocrinology*, 2008; 282(1-2): 120-129.
18. Tsatsaris V1, Goffin F, Munaut C, Brichant JF, Pignon MR, Noel A *et al.*; Over expression of the soluble vascular endothelial growth factor receptor in preeclamptic patients: pathophysiological consequences. *J Clin Endocrinol Metab.*, 2003; 88(11): 5555-5563.
19. Reisinger K, Baal N, McKinnon T; The gonadotropins: tissue-specific angiogenic factors. *Mol Cell Endocrinol.*, 2007; 269(1-2): 65-80.