

A Comparative Study on Prevalence and Severity of Hypothyroidism among Women with Preeclampsia

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Abstract

Introduction: Thyroid dysfunction is one of the most common endocrine disorders encountered during pregnancy after diabetes mellitus. Pregnancy develops significant changes in hypothalamic-pituitary-thyroid axis, iodine metabolism, and the immune function. Thyroid physiology alters to meet increased metabolic demands in pregnancy.

Aim: The aim of this study was to compare the prevalence and severity of hypothyroidism among women with preeclampsia and normotensive patients.

Materials and Methods: This is a hospital-based observational case-control study. A total of 200 women are included, out of them 100 normal pregnant women in the control group and 100 preeclamptic women in the case group are included in this study. Informed consent was obtained from the patients. The results are statistically analyzed and discussed below.

Results: Out of 200 subjects, the patients' mean age in control group and study groups was 28.07 ± 4.89 and 24.42 ± 4.93 years. In study group out of 100 patients, 70 had a euthyroid state, 28 had subclinical thyroid state, and two patients had overt thyroid. In the control group out of 100 patients, 86 had a euthyroid state, 11 had subclinical thyroid, and three patients had overt thyroid. Mean value of TSH in the study group is higher compared to the control group. Mean value of FT4 in the study group is less compared to the control group.

Conclusion: We concluded that the preeclampsia had a higher incidence of hypothyroidism (SCH) in contrast to the normotensive women, and there is a correlation between the severity of preeclampsia and hypothyroidism.

Key words: Hypothyroidism, Preeclampsia, Pregnancy, Thyroid

INTRODUCTION

Thyroid dysfunction is one of the most common endocrine disorders encountered during pregnancy after diabetes mellitus. It has long been recognized that maternal thyroid hormone excess or deficiency can influence the outcome for mother and fetus and interfere with ovulation and fertility.^[1,2] The prevalence of hypothyroidism in pregnancy is around 2.5% according to the western literature. There are few reports of hypothyroidism during pregnancy from India with rates ranging from 4.8% to 11%.^[3,4] Pregnancy

develops significant changes in hypothalamic-pituitary-thyroid axis, iodine metabolism, and the immune function. Thyroid physiology alters to meet increased metabolic demands in pregnancy. There is estrogen stimulation which increases the circulating levels of thyroid-binding globulin. The proposed mechanism is increased excretion of the iodine secondary to fetal intake and placenta metabolism, leading to a decline in iodine availability. Total concentrations of thyroxine (T4) and triiodothyronine (T3) increase in the first trimester of pregnancy achieving the plateau early in the second trimester, followed by reaching concentration 30–100% greater than the pre-pregnancy levels after the rise in thyroid-binding globulin.

Thyroglobulin increases during pregnancy secondary to the enhanced activity of the thyroid gland. Hypothyroidism in pregnancy complicates pregnancy through various mechanisms. It increases the risk of preeclampsia. Studies have found that women with hypothyroidism during

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pregnancy have an increased risk of preterm labor and instrumental deliveries. They were found to have increased abortion, intrauterine death as compared to women with normal functioning thyroid.

Hypothyroidism has been observed as one of the causes of high blood pressure. There is the failure of estrogen production and placental dysfunction in preeclampsia, resulting in low TBG, TT3, and TT4. Experimental studies have indicated that release of NO is altered in hypothyroidism and the resulting endothelial cell dysfunction might be a pathogenetic mechanism for hypothyroidism in preeclampsia.

Thyroid dysfunction during pregnancy is associated with adverse maternal complications such as miscarriages, anemia complicating pregnancy, preeclampsia, abruptio-placentae, postpartum hemorrhage, and fetal complications such as premature birth, low-birth weight, and increased neonatal respiratory distress. Maternal and fetal hypothyroidism can also result in irreversible brain damage with mental retardation and neurologic abnormalities which justifies screening for thyroid dysfunction during early pregnancy with interventional levothyroxine therapy for thyroid hypofunction.^[5-10]

Aim

The aim of this study was to compare the prevalence and severity of hypothyroidism among women with preeclampsia and normotensive patients.

MATERIALS AND METHODS

This is a hospital-based observational case-control study. A total of 200 women are included, out of the 100 normal pregnant women in the control group, and 100 preeclamptics women in the case group. The patients were classified as euthyroid, subclinical hypothyroid, overt hypothyroid, and hyperthyroid based on their TSH levels and T4 testing. Inclusion criteria in the case group include pregnant women >37 weeks' gestation, a diagnosed case of preeclampsia, previously normotensive, and in the control group, including pregnant women >37 weeks and normotensive. Exclusion criteria include previous H/O medical renal and hepatic disease, hyperthyroidism and endocrine disorders, RHD, not on any chronic drugs, multiple gestation, and molar pregnancy.

10 mL venous blood to be drawn for thyroid hormone analysis (FT3, FT4, and TSH) using chemiluminescent assay. Particulars of the women are noted, such as name, age, symptoms, menstrual history for menarche, last menstrual period and past menstrual cycles, and history of present pregnancy. Obstetric history to be asked for

marriage duration, infertility, gravida and parity status, recurrent abortions, preeclampsia, growth restriction, low birth weight, preterm delivery, prematurity, late losses, neonatal deaths, and mental retardation in a previous pregnancy.

Medical history was asked for any associated medical disorders such as diabetes, thyroid disorders, exposure to radiation, or autoimmune disorders. Significant surgical history, family history was also asked. A thorough clinical examination, including height, weight, pulse, blood pressure, pedal edema, and thyroid enlargement, was done by systemic examination. In an obstetrical examination, presentation and liquor were noted, and fetal heart sounds were auscultated.

All preliminary and baseline investigations such as complete blood count, blood grouping and typing, urine routine and microscopy, and blood sugar were done. Ultrasonography was done for fetal growth, liquor, and placenta. All investigations about preeclampsia complications such as liver and kidney function tests and serum uric acid were also done.

RESULTS

Out of 200 subjects, the patients' mean age in control and study groups was 28.07 ± 4.89 and 24.42 ± 4.93 years ($P = 0.35$) [Figure 1].

In the study group, out of 100 patients, 70 had a euthyroid state, 28 had subclinical thyroid state, and two patients had overt thyroid. In the control group, out of 100 patients, 86 had a euthyroid state, 11 had subclinical thyroid, and three patients had overt thyroid [Figure 2].

Mean value of TSH in the study group is higher compared to the control group. Mean value of FT4 in the study group is less compared to the control group [Figure 3].

DISCUSSION

The age distribution of patients included in our study ranged from 18 to 40 years. Majority of them belonged to the <21–30 years in both the groups. The patients' mean age in the control and study group was 28.07 ± 4.89 and 24.42 ± 4.93 years, respectively, which was comparable ($P = 0.35$).

In a similar study done by Ashokkumar *et al.*,^[11] comparing preeclampsia with normotensive women, the mean (SD) age of the study group and the control group was 28.4 ± 6.24 years and 27.5 ± 5.91 years, respectively, which is quite similar to our study.

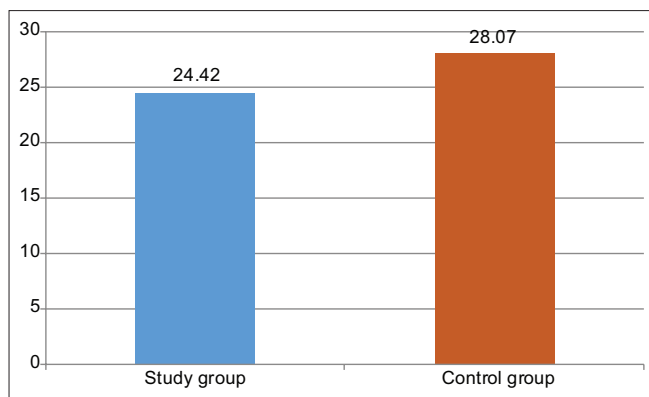


Figure 1: Age distribution

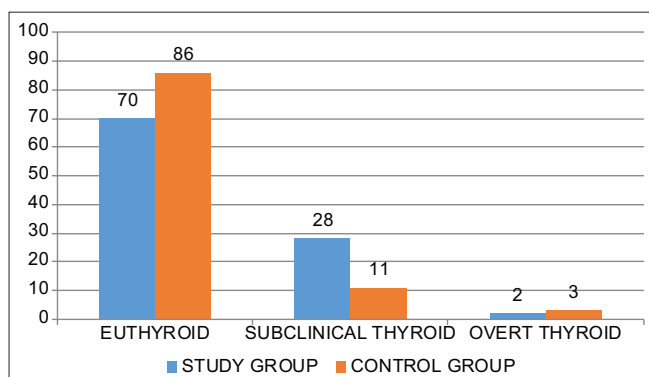


Figure 2: Type of thyroid state

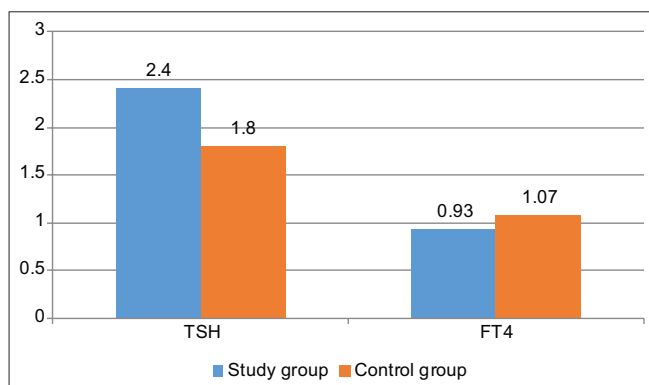


Figure 3: Thyroid function test

TSH, free T4 was done for both the groups and the results were analyzed. The control group in our study had 86 euthyroid subjects (86%), 11 subclinical hypothyroid (11%), and three overt hypothyroid (3%).

In the preeclampsia group, 70 were euthyroid (70%), 28 are subclinical hypothyroid (28%), and two are overt hypothyroid (2%). These findings follow the previous literature stating that preeclampsia women have a higher incidence and prevalence of biochemical hypothyroidism than the normotensive population.^[12-15]

The mean TSH value in the preeclampsia group is more than the controls in our study (2.4 ± 1.3 vs. 1.8 ± 0.9). It is significant. Our study's mean free T4 values in preeclampsia versus controls is 0.93 ± 0.28 versus 1.07 ± 0.33 , which remains within the normal trimester-specific range of FT4. However, the PE group had a mean FT4 level lower than the controls and the difference was statistically significant ($P < 0.0001$).

Thus, subclinical hypothyroidism is more common in the preeclampsia group in the present study and is similar to the study done by Kumar *et al.*,^[11] the mean FT4 is not significantly different in the two groups and the mean TSH value was significantly higher in the preeclampsia women than that of controls ($P < 0.001$). This is partly comparable to our study where the mean TSH and FT4 are significantly different between the groups with the PE group having a high mean TSH and a low mean FT4.

In another Indian study, the mean TSH titers in the preeclampsia pregnancies have been 3.8 ± 0.53 mIU/ml. In the normal pregnancies, it was 2.3 ± 0.24 mIU/ml (Kaliq fetal) which again is comparable to the present study.^[16]

A study by Wilson *et al.*^[17] women with subclinical hyperthyroidism had an incidence of hypertensive disorders of 6.2% compared with 8.5% of euthyroid women and 10.9% subclinical hypothyroid women. After adjusting only women with subclinical hypothyroidism were at increased risk for severe preeclampsia (adjusted odds ratio, 1.6; 95% confidence interval, 1.1–2.4; $P = 0.031$) pointing toward a causal role.

In the calcium for preeclampsia prevention cohort, the mean TSH values were increased 2.42 times above baseline in the PE group compared with a 1.48 times increase in controls (Levin *et al.*, 2009). Thus, this study suggests PE as a possible risk factor for hypothyroidism, and the mechanisms could be mediated through s-linked like tyrosine kinase.^[18]

According to Ashoor *et al.*,^[19] measurements of maternal serum TSH can improve late-PE prediction provided by a combination of factors in the maternal history and mean arterial pressure and uterine artery measurements pulsatility index.

Hypothyroidism may also play a direct role in causing pregnancy hypertension because thyroid hormones act directly on peripheral arterioles to cause dilation (Dernellis and Panaretou, 2002). On the other hand, there a few studies arguing against any relationship between hypothyroidism and preeclampsia. In the present study, the mean TSH is significantly higher in the preeclampsia group and FT4 being significantly lower.

The prevalence of subclinical hypothyroidism in our entire study group is 19.5% (28% in preeclampsia women and 11% in normotensive women) and overt hypothyroidism contributing to 2% preeclampsia group while 3% in normotensive group. The mean age of termination in the study group was 37–38 weeks (72%), while that in the control group was 39 weeks (79%) which allows 15% of postdated. This study also analyzed the relationship between the severity of preeclampsia and hypothyroidism. Out of the 100 preeclampsia patients, 26 belonged to the severe and 74 belonged to the mild preeclampsia group. The TSH was significantly more in the severe preeclampsia group as compared to mild preeclampsia (2.8 ± 1.67 vs. 2.4 ± 1.33); $P < 0.0001$).

The values of free T4 are (1.08 vs. 1.25) numerically less in severe preeclampsia than mild preeclampsia, and they were statistically significant ($P < 0.0001$). These findings strongly suggest an association between the severity of preeclampsia and hypothyroidism.

CONCLUSION

From this study, we concluded that the preeclamptics have a higher incidence of hypothyroidism (SCH) than the normotensive women. There is a correlation between the severity of preeclampsia and hypothyroidism. The treatment of OH and SCH is mandatory. In future, there should be a changing trend toward routine screening of hypothyroidism in contrast to targeted screening, but further, more extensive studies are needed to support this fact.

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