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Research Article

ANALYZING THE CHANGES IN THYROID HORMONES AND THE SERUM LIPID PROFILE IN NON-PREGNANT, PREGNANT, AND PREECLAMPTIC WOMEN

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ABSTRACT

Objective: The objective of the study was to investigate lipids and thyroid profile status among women with (100 pregnant women) or without pregnancy (100 normal) and with preeclampsia (100 pre-eclamptic women).

Methods: The Department of Obstetrics and Gynecology at the Pacific Institute of Medical Sciences in Udaipur treated 300 patients (obstetric cases) for the current case control comparative study. The total lipid and thyroid profile were examined for in a fasting blood sample.

Results: The study showed increased level of triiodothyronine, thyroxine in pre-eclampsia than normal pregnant but non-significant. Lipid profile (TC, TG, LDL, and VLDL), TSH were significantly higher in pre-eclampsia than in normal pregnant and non- pregnant women.

Conclusion: 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: Thyroid profile, Lipid profile, Pre-eclampsia, Pregnant women, Triiodothyronine, Thyroxine, Thyrotropin-stimulating hormone

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INTRODUCTION

A significant obstetric issue today in medical practice is pregnancy-induced hypertension. It creates a significant medical conundrum because it endangers not only the health of the mother but also the development of the fetus. Hypertensive disorders of pregnancy are widespread throughout the world and are to blame for 12% of maternal deaths that occur during pregnancy and the puerperium. Preeclampsia causes a five-fold increase in perinatal death and is the primary cause of maternal mortality in affluent nations. Reduced uteroplacental perfusion is the primary factor in preeclampsia-related fetal impairment [1,2].

A physiological process occurs during pregnancy. During pregnancy, the mother's various organ systems go through physiological changes to provide the growing fetus with enough sustenance. Circulatory, metabolic, and hormonal changes are made. Preeclamptic women frequently have hypothyroidism, which may be related to the severity of preeclampsia. Pregnancy is typically linked with mild hyperthyroxinemia. Pre-eclampsia has additionally been noted in 43.7% of overt hypothyroidism patients and 16.7% of subclinical hypothyroidism instances during pregnancy [3-5].

Preeclampsia frequently has dyslipidemia, which may cause endothelial activation by oxidizing lipids that are susceptible to this reaction. A preponderance of both atherogenic small low-density lipoproteins (LDL) and vascular cell adhesion molecules is linked to hyperlipidemia in preeclampsia [6,7].

In the current study, lipid and thyroid hormone levels in healthy pregnant women, preeclampsia patients, and healthy non-pregnant (control) people were investigated.

METHODS

At the Pacific Institute of Medical Sciences in Udaipur, the study involved 300 cases and was carried out with informed consent. In the case–control comparative study, the subjects were divided into three categories:

- Group 1 (n=100) = Non pregnant women
- Group 2 (n=100) = Pregnant women
- Group 3 (n=100) = Pre-eclamptic women.

Detailed information from the interviews was entered on the printed pro forma after questions were asked, case history was obtained and bedside urine examination for sugar was done. Name, age, husband's name, place of residence (urban or rural), menstruation history, obstetric history, concurrent medical conditions, and obstetric difficulties were all recorded for the patient. The fasting blood sample was collected, and analysis was done in the clinical biochemistry lab of PIMS, Udaipur. Women with past and present study of thyroid disease, diabetes mellitus, or glycosuria were excluded from the study.

The following profiles were checked in blood samples:

Triiodothyronine (T3) 0.6–1.81, thyroxine (T4) range 4.5–12.6, and Thyrotropin-stimulating hormone (TSH) 0.23–5.5 FT4and FT3.

The sample was collected, subjected to a centrifugation process at 3000 rpm for 15 min at 34 degrees Celsius, and the resultant serum was used for the thyroid and lipid assay.

The statistical significance was evaluated by student's t-test using graph pad 7.0 online version.

RESULTS

Age groups were separated into Groups 1, 2, and 3, with 92 cases falling into the 20–40 year age range and 58 cases falling into the 41–60 year age range. There were 97 total urban cases across all groups, compared to 53 rural instances. There were 86 tribal cases and 64 non-tribal cases (Table 1).

The study population's demographics for non-pregnant, pregnant, and preeclampsia subjects are shown in Table 1. The mean age and mean pregnancy period were comparable across all study groups. Indicators of obesity (weight and BMI) were considerably higher in the pregnancy groups than in the non-pregnant women's control group. Preeclamptic individuals had considerably higher mean blood pressure (both systolic and diastolic) and urine protein levels than pregnant and non-pregnant subjects.

The lipid level variations in non-pregnant, pregnant, and preeclampsia individuals are shown in Table 2. When compared to healthy pregnant and non-pregnant subjects, the lipid profile (total cholesterol, triglyceride, VLDL, and LDL-C levels) in preeclamptic patients was significantly greater. However, compared to the other groups of women, preeclamptic individuals had a significantly lower mean plasma HDL-C content. As a result, preeclampsia women's mean lipid levels were statistically substantially different from those of healthy pregnant women.

The serum thyroid status (T3, T4, FT3, FT4, and TSH) of non-pregnant, pregnant, and preeclampsia participants is shown in Table 3. When compared to non-pregnant women, the mean serum T4 and T3 levels in a normal pregnancy were significantly higher. Both women who were not pregnant and those who were pregnant normally had similar mean FT3 and FT4 levels. The mean serum T4 and T3 levels in preeclampsia patients were noticeably greater than those in non-pregnant women. However, when T3 was much lower than the level of women with normal pregnancies, T4 was obviously greater but not significantly so.

In comparison to non-pregnant women, normal pregnant women had considerably higher mean TSH levels (p=0.001). The mean preeclampsia level was, however, significantly greater than that of normal-weight pregnant women (p=0.001) and the mean TSH level for preeclampsia patients was even higher than in any of the other two groups.

Preeclampsia patients had mean serum FT4 levels that were considerably greater than those of non-pregnant women. However, there was no discernible difference in FT3 levels between preeclampsia and normal pregnancy. In comparison to women who were not pregnant, those with normal pregnancies had a mean serum FT that was considerably greater. Preeclampsia patients exhibited significantly greater FT3 levels than non-pregnant women, but their mean levels were much lower than those of pregnant patients with normal pregnancies.

Table 1: Demographic characteristics of cases

Parameters	Group 1 (n=100)	Group 2 (n=100)	Group 3 (n=100)
Age	38	34	32
Period of gestation (weeks)	---	36	35
Weight (kg)	58	67	69
BMI (kg/m^2)		20	35
Hypertensive	---	---	100(100%)
Systolic blood pressure (mmHg)	120±4	118±2	168±4
Diastolic blood pressure (mmHg)	82±2	80±4	118±9

Table 2: Lipid level changes in three groups

Parameters	Group 1 (n=100)	Group 2 (n=100)	Group 3 (n=100)
Total Cholesterol (mg/dL)	152±13	169±11	221±15
Triglyceride (mg/dL)	91±10	134±16	252±21
HDL-Cholesterol (mg/dL)	43±5	39±6	32±7
LDL-Cholesterol (mg/dL)	76±7	86±12	139±14
VLDL-Cholesterol (mg/dL)	25±6	40±7	52±5

Table 3: Thyroid levels in different groups

Parameters	Group 1 (n=100)	Group 2 (n=100)	Group 3 (n=100)
T3	0.78±0.12	2.28±0.42	1.98±0.38
T4	7.50±3.32	14.13±4.52	14.25±6.02
FT3	2.57±0.54	3.46±1.18	2.87±1.02
FT4	1.17±0.36	2.46±0.82	2.42±0.78
TSH	2.18±1.14	3.79±3.22	5.34±2.42

DISCUSSION

Increases in weight and BMI are linked to increases in body fat percentage levels, which may help to explain why we found that the mean BMI was higher in both groups of pregnant women in our study.

It is well known that hypertriglyceridemia and preeclampsia go hand in hand. Endothelial cell dysfunction during pregnancy may be caused by the interactions indicated above in addition to increased endothelial triglyceride buildup. Predisposed vessels, such as the uterine spiral arteries, are likely to accumulate increased triglycerides observed in pregnancy-induced hypertension (preeclampsia). If true, the production of tiny, dense LDL may both directly and indirectly contribute to endothelial dysfunction [8,9].

Additionally, this hypertriglyceridemia may be linked to an increased tendency to clot. Hyperoestrogenaemia may have caused a considerable decrease in LDL-C content in the control group in this investigation. On the other hand, the participants with pregnancy-induced hypertension had considerably higher LDL-C values. In addition, several investigations have shown that preeclampsia is associated with hyperlipidemia and a predominance of atherogenic small LDL, as well as an increase in vascular cell adhesion molecules [10,11].

Preeclampsia's endothelial dysfunction may result from oxidative stress and dyslipidemia. Free radicals can be produced by a wide range of enzyme activities. They combine with polyunsaturated fatty acids to form lipid peroxides with a substantially longer half-life and hypercoagulability since they are very reactive [10,11].

In the current study, we assessed the thyroid status in preeclampsia without thyroid abnormalities and normal pregnancy. An important change in thyroid activity during pregnancy is indicated by elevated serum thyroid hormone levels [12].

In this investigation, whereas free forms of the hormones (FT3 and FT4) were comparable in both groups, serum T3 and T4 levels were considerably greater in pregnant women compared to non-pregnant controls. The considerable rise in the blood levels of thyroid-blinding globulin, the main binding protein, may be the cause of the rise in the serum binding forms of thyroid hormone. Pregnancy-related elevated estrogen levels cause the production of this globulin. In addition, during pregnancy, increased metabolic demand, mental stress, and the stimulatory action of placental-derived hCG in the blood may all contribute to an increase in overall thyroid activity and a rise in thyroid hormone levels.

Increased estrogen levels during pregnancy lead to the liver producing more proteins. Hepatocytes produce more thyroid-binding globulin as a result, which is the protein responsible for transporting T throughout the body. On the other hand, high estrogen inhibits peripheral degradation of thyroid-binding globulin due to oligosaccharide modification. The serum's level of thyroid-binding globulin rises as a result. More hormones bind to the thyroid-binding globulin as the binding capacity of the plasma rises as a result of the raised serum level of the globulin. As a result, there is an increase in the overall plasma level of thyroid hormones. Despite this, the levels of free thyroid hormone are unaffected, and hyperthyroidism does not develop.

Regarding free hormone levels during pregnancy, some findings are debatable. According to several researchers, pregnant women's free

hormone levels are either unaltered, go down, or even go up when compared to non-pregnant controls. The current study adds to the continuing debate because it finds no appreciable difference in free thyroid hormone levels between non-pregnant and pregnant women [13].

In this study, thyroid hormone levels in preeclampsia were compared to those in healthy pregnancies. The mean serum total and free T levels were somewhat higher in preeclampsia patients than in women having healthy pregnancies, but there was no statistically significant difference between the two groups. Women with preeclampsia did, however, exhibit statistically significantly lower total and free T3 levels compared to those with normal pregnancies. We hypothesize that the non-significant higher T levels and substantial lower T3 levels in preeclampsia were caused by impaired extrathyroidal conversion of T to T. Preeclampsia is an autointoxication brought on by pregnancy with multisystem diseases; the brain, liver, and kidneys are the most commonly impacted organs. Preeclampsia manifests functional abnormalities in these organ systems [3].

The maintenance of normal blood levels of T4 and T3, as well as peripheral deiodination — the conversion of T4 to T3 rely mostly on the liver and kidneys. This is why preeclampsia's involvement of the liver and kidneys is likely to alter the levels of serum T4 and T3.

Researchers have noted that preeclamptic women may be impacted by a number of diseases in other investigations. Systemic diseases, malnutrition deficient in protein and energy, starvation, anorexia nervosa, Cushing's syndrome, and excessive steroid therapy are a few of them. The extra thyroidal deiodination of T4 to T3 has decreased when certain systemic illnesses in women have been seen [14]. However, because there is such a large range of acceptable ranges, variations in T and T typically neither go beyond acceptable ranges nor cause appreciable metabolic alterations.

CONCLUSION

The main finding of the current study is that preeclamptic pregnant women with excessively high TSH levels were statistically considerably more common. Diseases of the thyroid gland increase the risk of developing preeclampsia. A person is 4.8 times more likely to develop preeclampsia if their TSH titers are above 5 mIU/mL. The small sample size in this study necessitates more research on this high-risk possible preeclampsia marker. The relationship and mechanism of thyroid anomalies in preeclamptic women in various geographical areas may be revealed by a multicenter study. Such a study might reduce the incidence and severity of morbidity and death associated with preeclampsia by allowing us to detect thyroid problems and take appropriate therapeutic action to address them.

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COMPETING INTERESTS

None.

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