



## CORRELATION OF THYROID AND LIPID PROFILE IN PRE ECLAMPSIA PATIENTS

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### ABSTRACT

**Background:** Preeclampsia is a common medical complication during pregnancy.

**Methods:** The study was conducted on 50 pregnant females, divided into two groups of 25 each: cases of PIH (study group) and normotensive pregnant females (control group) and all these pregnant females were at 20 weeks or more gestation. Blood sample was collected from these patients at the period of gestation when they attended Gynaecology O.P.D at chalmeda anand rao hospital, karimnagar. Thyroid profile and Lipid profile (S. Cholesterol and Triglycerides) was evaluated and compared in these patients.

**Results:** The mean T<sub>3</sub> value in the study group was 1.18 ng/ml while it was 1.16 ng/ml in the control group (p=0.8156) and mean T<sub>4</sub> value in the study group was 8.19 g/dl compared to 7.69 g/dl in the control group (p=0.2681). The difference did not attain statistical significance. The mean TSH value in the study group was 2.73 mIU/L which was significantly higher statistically than the value of 1.27 mIU/L in the control group (p<0.0001). Lipid profile levels (Serum cholesterol and triglycerides) were significantly higher in the study group compared to the control group (p<0.0001). The mean serum cholesterol value in the study group was 254.64 mg/dl compared to 172.64 mg/dl in the control group. The mean triglycerides value in the study group was 235.86 mg/dl compared to 133.20 mg/dl in the control group.

**Conclusions:** It was concluded that patients with PIH had a significant increase in TSH levels, a non significant alteration in T<sub>3</sub> and T<sub>4</sub> levels and a significant increase in lipid profile levels (S.cholesterol and triglycerides) compared to normotensive pregnant females. This suggests role of thyroid hormones and dyslipidemia in the development and pathogenesis of PIH. Therefore, early detection of thyroid abnormalities and dyslipidemia may affect the occurrence and severity of PIH and help in better management of the disease in established pre-eclamptic women.

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## INTRODUCTION

A mother becomes almost a new person during the period of pregnancy. Profound local and systemic changes in maternal physiology are initiated by conception and continued throughout pregnancy [1].

Pre-eclampsia is a multisystem disease unique to human pregnancy characterised by hypertension and organ system derangement. The disease is responsible for considerable morbidity and mortality complicating 5-8% of pregnancies and remains in the top three causes of maternal morbidity and mortality globally. It is the leading cause of fetal growth restriction, intrauterine fetal demise and planned preterm birth [2]. Pre-eclampsia usually occurs after 20 weeks gestation and is classically defined as a triad of hypertension, edema and proteinuria [3].

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Pregnancy is usually associated with mild hyperthyroxinemia, but pre-eclamptic women have a high incidence of hypothyroidism that might correlate with the severity of pre-eclampsia [4,5]. In pre-eclampsia, there is decreased estrogen production due to placental dysfunction resulting in lowering of Thyroid binding globulin, total T<sub>3</sub> and total T<sub>4</sub> along with growth retardation of the fetus [5]. Abnormal lipid metabolism seems important in the pathogenesis of pregnancy induced hypertension (PIH). Endothelial dysfunction is the most important event in the pathogenesis of pre-eclampsia and lipids have a role on this event. Increased TG, found in PIH, is likely to be deposited in predisposed vessels, such as the uterine spiral arteries and contributes to the endothelial dysfunction [6]. Altered lipid synthesis leading to decrease in PGI<sub>2</sub>:TXA<sub>2</sub> ratio is also supposed to be an important way of pathogenesis in PIH [7]. So in the background of altered thyroid and lipid profile being potential risk factors for occurrence of PIH, the present study aims to evaluate and compare the thyroid and lipid (cholesterol and triglycerides) profiles in PIH and normal pregnancy so that early identification of changes in these

## Correlation of Thyroid and Lipid in Pre Eclampsia Patients

parameters might be of help in preventing the occurrence of PIH.

### MATERIAL AND METHODS

The present study was conducted in the Department of obstetrics and gynecology, caims, karimnagar. 50 pregnant females were studied the ethical committee of our institution approved the study protocol. The objectives of the study were explained and written consent was taken from individual subjects. The subjects selected for the present study were attending and admitted were at 20 weeks or more gestation.

#### Study group

25 pregnant females with pregnancy induced hypertension at 20 weeks or more gestation. The diagnosis of Pre-eclampsia was based on the definition of National High Blood Pressure Education Program Working Group, B.P  $\geq$  140/90 mmHg after 20 weeks gestation and proteinuria  $\geq$  300 mg/24 hours or  $\geq$  1+ dipstick.

#### Control group

25 pregnant females at same gestation with normal blood pressure, no proteinuria and without any other systemic or endocrine disorder.

#### Exclusion criteria

All cases of previous history of essential hypertension or chronic hypertension, known cases of thyroid disorders and associated molar pregnancy and multiple pregnancies were excluded from the study.

#### Specimen collection

Blood sample was collected from these patients at the gestational age of 20 weeks or more, when they attended O.P.D at caims karimnagar. Under all aseptic conditions, 5 ml blood sample was collected by venipuncture and allowed to clot. The serum was then separated by ultracentrifugation of the sample at room temperature and supernatant (serum) was taken in a separate test tube. The serum was then used for Thyroid and lipid profile analysis in the laboratory.

### METHODOLOGY

Thyroid profile was done by ELISA Method using ERBA THYROKIT. T<sub>3</sub>, T<sub>4</sub> by Competitive enzyme immunoassay [8] and TSH by Immuno-enzymometric assay [9] and Lipid profile- Serum cholesterol using Enzymatic colorimetric method (Allain *et al*)-Cholesterol Oxidase Peroxidase method [10] and Triglycerides using Enzymatic colorimetric method (Mc Gowan *et al*) : Glycerol 3 – Phosphate Oxidase Method [11].

#### Statistical analysis

It was done by student's t-test using SPSS version 10.0

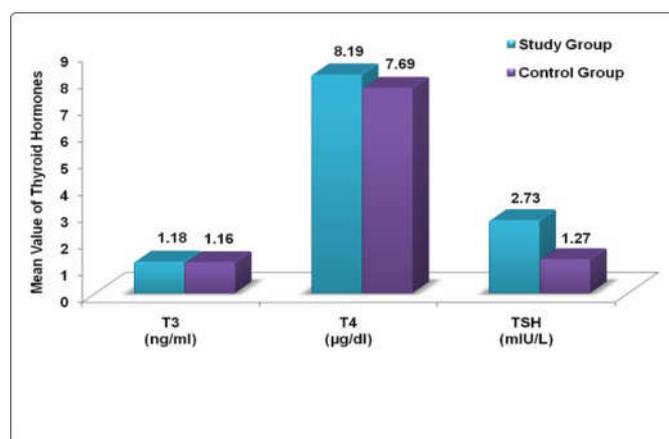
### RESULTS

Table 1 shows demographic characteristics of both the study and control groups. The mean age and period of gestation were comparable in both the groups. The mean BMI was increased in both the groups. The mean SBP and DBP were significantly higher in the study group compared to the control group.

**Table 1** characteristics of study group and control group

Parameter	Study group	Control group
Mean Age (in years)	25.12 $\pm$ 3.95	24.14 $\pm$ 3.80
Mean Period of gestation (in weeks)	32 $\pm$ 3.68	31 $\pm$ 3.70
Body Mass Index (kg/m <sup>2</sup> )	29 $\pm$ 5.7	27 $\pm$ 3.9
Mean S.B.P (mmHg)	155.68 $\pm$ 14.68	115.68 $\pm$ 7.08
Mean D.B.P(mmHg)	105.48 $\pm$ 11.02	73.96 $\pm$ 6.25
Fasting Plasma Glucose (mg/dl)	81.96 $\pm$ 10.59	79.90 $\pm$ 8.24

The mean T<sub>3</sub> value in the study group was 1.18 ng/ml while it was 1.16 ng/ml in the control group (p=0.8156) and mean T<sub>4</sub> value in the study group was 8.19 g/dl compared to 7.69 g/dl in the control group (p=0.2681). The difference did not attain statistical significance. The mean TSH value in the study group was 2.73 mIU/L which was significantly higher statistically than the value of 1.27 mIU/L in the control group (p<0.0001) (Table 2) (Figure 1)



**Figure 1** Comparison of mean thyroid profile in study and control group

**Table 2** Comparison of mean thyroid profile (t<sub>3</sub>, t<sub>4</sub> and TSH) values in study and control group

	Study Group (n=25)	Control Group (n=25)	T	P	S
T <sub>3</sub> (ng/ml) (Mean $\pm$ SD)	1.18 $\pm$ 0.50	1.16 $\pm$ 0.33	0.2338	0.8156	NS
T <sub>4</sub> (µg/dl) (Mean $\pm$ SD)	8.19 $\pm$ 2.89	7.69 $\pm$ 1.30	1.1138	0.2681	NS
TSH (mIU/L) (Mean $\pm$ SD)	2.73 $\pm$ 1.85	1.27 $\pm$ 0.90	5.0062	<0.0001	Highly Significant

Lipid profile levels (Serum cholesterol and triglycerides) were significantly higher in the study group compared to the control group (p<0.0001). The mean serum cholesterol value in the study group was 254.64 mg/dl compared to 172.64 mg/dl in the control group. The mean triglycerides value in the study group was 235.86 mg/dl compared to 133.20 mg/dl in the control group (Table 3).

**Table 3** Comparison of lipid profile in study group and control group

	Study group (n=50) (Mean $\pm$ SD)	Control group(n=50) (Mean $\pm$ SD)	T	P	S
S.Cholesterol (mg/dl)	254.64 $\pm$ 50.56	172.64 $\pm$ 39.15	9.0661	<0.0001	Highly Significant
S.Triglycerides (mg/dl)	235.86 $\pm$ 64.90	133.20 $\pm$ 48.58	8.9537	<0.0001	Highly Significant

## DISCUSSION

Between 5% and 15% of pregnant women experience thyroid abnormalities, a fact which justifies screening by means of clinical laboratory testing. Maternal thyroid dysfunction during pregnancy has been shown to be associated with a number of adverse outcomes. It has been suggested that there may be an existence of mutual influences between pre-eclampsia and thyroid function [12,13].

The two groups were comparable in terms of fasting plasma glucose and this rule out the influence of systemic illness like Diabetes Mellitus on thyroid gland and on the lipid profile of the patients. In the present study, though T<sub>3</sub> and T<sub>4</sub> level were higher in the study group, the values did not attain statistical significance while the mean TSH level was significantly higher than the control group. Many researchers have shown that women with pre-eclampsia had high level of TSH [14]

The finding of increased TSH levels in pre-eclamptic cases can be explained by excessive release of antiangiogenic proteins- most notably sFlt-1 (soluble Fms-like tyrosine kinase-1) from the placenta into maternal blood, resulting in an antiangiogenic state with low levels of free placental growth factor and free vascular endothelial growth factor which leads to endothelial dysfunction [15].

The explanation of non-significantly increased T<sub>3</sub> and T<sub>4</sub> levels in the present study in PIH cases may be attributed to pathological changes in pre-eclampsia which leads to endocrinological disturbances and consequently to thyroid gland dysfunction that may affect synthesis and secretion of T<sub>3</sub> and T<sub>4</sub>. Other studies have shown decreased T<sub>3</sub> associated with higher T<sub>4</sub> in pre-eclampsia attributed to reduced conversion of T<sub>4</sub> to T<sub>3</sub> in the liver and kidneys [16].

Disorders of lipoprotein metabolism are a major cause of endothelial dysfunction that may result in hypertension and proteinuria, the clinical hall-marks of pre-eclampsia [17].

In the present study, the mean serum cholesterol level was significantly higher in the study group compared to the control group. Physiological insulin resistance is exaggerated in pre-eclampsia. Gestational insulin resistance may accentuate the suppression of lipoprotein lipase activity and increase mobilization of free fatty acids from visceral adipocytes. This may explain the hypercholesterolemia in pre-eclampsia [18,19].

The mean triglycerides level was significantly higher in the study group compared to the control group. Hypertriglyceridemia may be modulated by hyperinsulinism found in pregnancy [20]. In pregnancies complicated by hypertension, there appears to be an exaggeration of insulin resistance and associated metabolic changes. Pre-eclamptic women are more insulin resistant than normotensive controls, so in pre-eclampsia triglyceride levels further increase due to the exaggeration of insulin resistance [21]. Also our findings of greater increase in mean BMI in pregnant females with PIH could explain the significant increase in triglycerides.

## CONCLUSION

In the present study, an effort was made to explore the role of thyroid hormones and lipid profile in the pathogenesis of PIH and it was found that patients with PIH had altered levels of thyroid hormones and dyslipidemia. This suggests role of

thyroid hormones and dyslipidemia in the development and pathogenesis of PIH. Therefore, early detection of thyroid abnormalities and dyslipidemia may affect the occurrence and severity of PIH and help in better management of the disease in established pre-eclamptic women.

Keeping in view the target for the Fifth Millennium Development Goal (MDG 5) which aims to improve maternal health and reduce maternal mortality ratio, by three - quarters by 2015, and PIH being one of the top three leading causes of maternal mortality, it is recommended that each and every pregnant women should have thyroid profile and lipid profile done as soon as she reports to the antenatal clinic and then again preferably in the second trimester to detect any abnormality that might put her at risk for PIH.

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## References

1. Kortenoever M. Physiological changes in pregnancy. 1960; 21:443-5.
2. Khan KS, Wojdyla D, Say L, Gülmezoglu AM, Van Look PF (2006) WHO analysis of causes of maternal death: a systematic review. *Lancet* 367: 1066-1074.
3. Pankaj D, Narendra M, Duru S: Principles and Practice of Obstetrics on Gyneco. 3rd ed. New Delhi Publication. 2008, P.P. 100-103
4. Qublan HS, Al-Kaisi IJ, Hindawi IM, Hiasat MS, Awamleh I, *et al.* (2003) Severe pre-eclampsia and maternal thyroid function. *J Obstet Gynaecol* 23: 244-246.
5. Kaya E, Sahin Y, Ozkeleşeci Z, Paşaoğlu H (1994) Relation between birth weight and thyroid function in preeclampsia-eclampsia. *Gynecol Obstet Invest* 30:33.
6. Sattar N, Greer IA, Loudon J, Lindsay G, McConnell M, *et al.* (1997) Lipoprotein subfraction changes in normal pregnancy: threshold effect of plasma triglyceride on appearance of small, dense low density lipoprotein. *J Clin Endocrinol Metab* 2483-2491.
7. Robson SC. Hypertension and Renal Disease in Pregnancy. Edmonds DK. editor. In: Dewhurst's Textbook of Obstetrics and Gynaecology for postgraduates. Blackwell Science, New York 1999; 6:167-9
8. Sterling L. Diagnosis and Treatment of Thyroid Disease. Cleveland CRC Press. 1975:9-51.
9. Spencer CA, Takeuchi M, Kazarosyan M, Mackenzie F, Beckett GJ, Wilkinson E (1995) Interlaboratory/ Intermethod differences in Functional Sensitivity of Immunometric Assays of Thyrotropin (TSH) and Impact on Reliability of Measurement of Subnormal Concentrations of TSH. *Clinical Chemistry* 41:367-374.
10. Allain CC, Poon LS, Chan CS, Richmond W, Fu PC (1974) Enzymatic determination of total serum cholesterol. *Clin Chem* 20: 470-475.
11. McGowan MW, Artiss JD, Strandbergh DR, Zak B (1983) A peroxidase-coupled method for the colorimetric determination of serum triglycerides. *Clin Chem* 538-542.

12. Casey BM, Dashe JS, Wells CE, McIntire DD, Byrd W, *et al.* (2005) Subclinical hypothyroidism and pregnancy outcomes. *Obstet Gynecol* 105: 239-245.
13. Allan WC, Haddow JE, Palomaki GE, Williams JR, Mitchell ML, *et al.* (2000) Maternal thyroid deficiency and pregnancy complications: implications for population screening. *J Med Screen* 7: 127-130.
14. Levine RJ, Vatten LJ, Horowitz GL, Qian C, Romundstad PR, *et al.* (2009) Pre-eclampsia, soluble fms-like tyrosine kinase 1, and the risk of reduced thyroid function: nested case-control and population based study. *BMJ* 339: b4336.
15. Maynard S, Epstein FH, Karumanchi SA (2008) Preeclampsia and angiogenic imbalance. *Annu Rev Med* 59: 61-78.
16. Al-NaqeebAsmehan. Correlation between Thyroid-related Hormones and Preeclampsia. *Iraqi Sci J Nursing* 2010; 23:76-80.
17. Sattar N, Bendomir A, Berry C, Shepherd J, Greer IA, Packard CJ. Lipoprotein subfraction concentrations in pre-eclampsia: pathogenic parallels to atherosclerosis. *ObstetGynecol* 1997; 89: 403-408.
18. Ramsay JE, Jamieson N, Greer IA, Sattar N (2003) Paradoxical elevation in adiponectin concentrations in women with preeclampsia. *Hypertension* 42: 891-894.
19. Lorentzen B, Henriksen T (1998) Plasma lipids and vascular dysfunction in preeclampsia. *Semin Reprod Endocrinol* 16: 33-39.
20. Adegoke OA, Iyare EE, Gbeneditso SO (2003) Fasting plasma glucose and cholesterol levels in pregnant Nigerian women. *Niger Postgrad Med J* 10: 32-36.
21. Kaaja R, Laivuori H, Laakso M, Tikkanen MJ, Ylikorkala O (1999) Evidence of a state of increased insulin resistance in preeclampsia. *Metabolism* 48: 892-896.

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