



GLOBAL JOURNAL OF MEDICAL RESEARCH: E
GYNECOLOGY AND OBSTETRICS
Volume 14 Issue 5 Version 1.0 Year 2014
Type: Double Blind Peer Reviewed International Research Journal
Publisher: Global Journals Inc. (USA)
Online ISSN: 2249-4618 & Print ISSN: 0975-5888

Comparative Study of Lipid Profile, Oxidant, Antioxidant Levels and Thyroid Hormone Status in Normal and Abnormal Pregnancy

By Parvathi.D, Uma.T & Lakshmi Vasavi.H

Rajiv Gandhi Institute of Medical Sciences, India

Abstract- Aim: The aim of the study is to evaluate thyroid status, Oxidative stress & lipid profile levels in abnormal and normal pregnancies.

Material and Methods: The study includes 25 abnormal pregnant women, 25 normally healthy pregnant women. Ages ranged from 20 to 30 years. Thyroid status was estimated by ELISA method. For determination of oxidative stress Malandialdehyde (MDA) is estimated as oxidants by TBARS, Uric acid is estimated by pap method, cholesterol & HDL-C by CHOD-POD method, triglycerides by GPO-POD method.

Results: In the present study, we observed that TSH is significantly increased compared to normal pregnancies TSH and FT4 are slightly decreased compared to normal pregnancies. In the present study cholesterol levels were increased in abnormal pregnancies compared to normal pregnancies and decrease in HDL cholesterol were observed in preeclamptic and eclamptic pregnant women.

Keywords: lipid profile, oxidant, antioxidant, abnormal pregnancy.

GJMR-E Classification : NLMC Code: WQ 200



Strictly as per the compliance and regulations of:



Comparative Study of Lipid Profile, Oxidant, Antioxidant Levels and Thyroid Hormone Status in Normal and Abnormal Pregnancy

Parvathi.D ^α, Uma.T ^σ & Lakshmi Vasavi. H ^ρ

Abstract- Aim: The aim of the study is to evaluate thyroid status, Oxidative stress & lipid profile levels in abnormal and normal pregnancies.

Material and Methods: The study includes 25 abnormal pregnant women, 25 normally healthy pregnant women. Ages ranged from 20 to 30 years. Thyroid status was estimated by ELISA method. For determination of oxidative stress Malandialdehyde (MDA) is estimated as oxidants by TBARS, Uric acid is estimated by pap method, cholesterol & HDL-C by CHOD-POD method, triglycerides by GPO-POD method.

Results: In the present study, we observed that TSH is significantly increased compared to normal pregnancies TSH and FT4 are slightly decreased compared to normal pregnancies. In the present study cholesterol levels were increased in abnormal pregnancies compared to normal pregnancies and decrease in HDL cholesterol were observed in preeclamptic and eclamptic pregnant women. In the present study Hb levels were decreased in abnormal pregnancies compared to normal pregnancies. The levels of MDA and uric acid were significantly increased in abnormal pregnancies. The glucose (RBS) levels were increased compared to normal pregnancies.

Conclusions: Altered Thyroid status which leads to hypothyroidism may be due to the loss of protein and protein bound hormones in urine in abnormal pregnancies compared with normals. Increased oxidative stress is due to increase in TSH levels which directly induces the pro-inflammatory cytokines. Study suggests that all abnormal pregnancies studied are associated with hypothyroidism.

Keywords: lipid profile, oxidant, antioxidant, abnormal pregnancy.

I. INTRODUCTION

The thyroid gland secretes hormones having important role in embryogenesis and fetal development during normal pregnancy. ⁽¹⁾ Thyroid function tests change during pregnancy due to the influence of two main hormones: human chorionic gonadotropin (hCG), the hormone that is measured in the pregnancy test and estrogen, the main female hormone. HCG can weakly turn on the thyroid and the high circulating hCG levels in the first trimester may result in a slightly low TSH. When this occurs, the TSH

Author α: Department of Gynecology and Obstetrics, Rajiv Gandhi Institute of Medical Sciences, Srikakulam, Andhra Pradesh, India.

Author σ: Department of Biochemistry, Rajiv Gandhi Institute of Medical Sciences, Srikakulam, Andhra Pradesh, India.

e-mail: t_daffadils@yahoo.co.in

Author ρ: Department of Pathology, Rajiv Gandhi Institute of Medical Sciences, Srikakulam, Andhra Pradesh, India.

will be slightly decreased in the first trimester and then return to normal throughout the duration of pregnancy. Estrogen increases the amount of thyroid hormone binding proteins in the serum which increases the total thyroid hormone levels in the blood. ⁽²⁾

The most common cause of hyperthyroidism in pregnancy is Grave's disease and hypothyroidism is autoimmune thyroid disease (Hashimoto's thyroiditis). The complications associated with hyperthyroidism during pregnancy are first trimester spontaneous abortions, preterm deliveries, low birth weight infants, still births, neonatal deaths, fetal, neonatal hyperthyroidism and intrauterine growth retardation; in hypothyroidism are preeclampsia, preterm labour, placental abruption, threatened abortions, neonatal hypothyroidism, neonatal hyperbilirubinemia, postpartum hemorrhage and increased frequency of low birth weight infants. ⁽²⁾

The aim of the study is to evaluate thyroid status in abnormal and normal pregnancies by the estimation of FT3, FT4, and TSH, Oxidative stress is estimated by estimating MDA as oxidant & uric acid as antioxidant & lipid profile levels include cholesterol, triglyceride, HDL, Hb%. Abnormal pregnancies includes pregnancy induced hypertension (preeclampsia, eclampsia), gestational diabetes, hydroamnios, and abortions.

II. MATERIALS AND METHODS

Study Population: The study populations were investigated consisted of 50 women divided into two groups consisted of 25 abnormal pregnant women, 25 normally healthy pregnant women. Ages ranged from 20 to 30 years. The prospective study was carried out at the RIMS Hospital, Srikakulam, Andhra Pradesh, India, between January 2013 to June 2013. The study was approved by the Institutional Human Ethical Committee (IHEC). Informed verbal consent was obtained from all subjects. The objectives of the study were explained and a written concept was taken from each subject. The data on family history and personal history of diabetes, jaundice, hypertension and miscarriages were collected through standard questionnaire.

Biochemical Investigation: Serum FT3 ⁽³⁾, FT4 ⁽⁴⁾, and TSH ⁽⁵⁾ were assessed quantitatively using ELISA kits. The FT3 and FT4 were expressed as pmol/L, however the TSH was expressed as mIU/L. Total

cholesterol and HDL-C estimated by CHOD-POD method^(6,7) and Tgl by GPO – POD method⁽⁸⁾. For determination of oxidative stress Malandialdehyde (MDA) is estimated as oxidant by thiobarbituric acid reactive substance (TBARS)⁽⁹⁾, Uric acid is estimated by pap method⁽¹⁰⁾, Hb is estimated by Drabkin's method⁽¹¹⁾, Glucose is estimated by glucose oxidase and peroxidase method⁽¹²⁾.

Statistical Analysis: All data were expressed as mean $\pm$ S.D. The 'P' value was used to compare mean values of abnormal pregnant women with normal pregnant women.

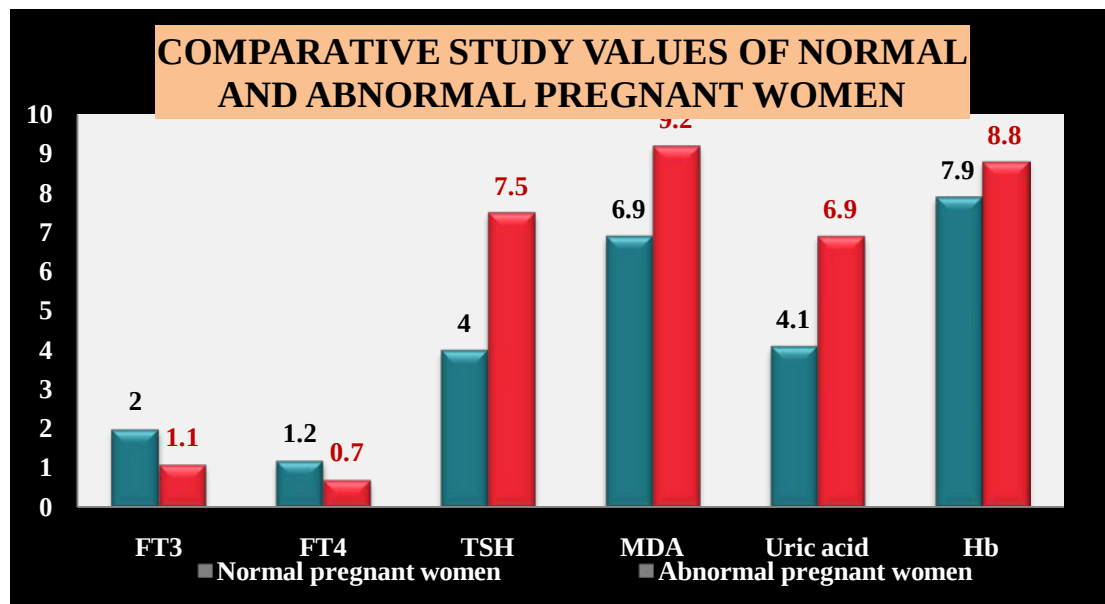
III. RESULTS

In the present study, observed that TSH (7.5 ± 3.93) is significantly increased compared to

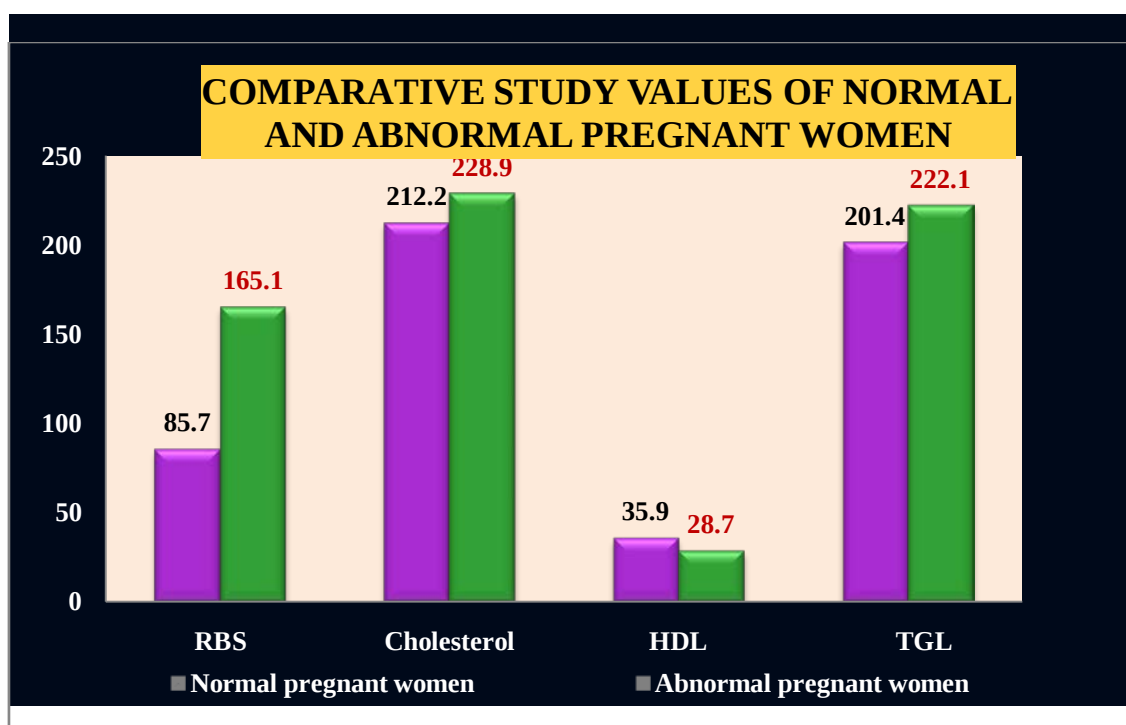
normal pregnancies TSH (4.0 ± 2.82). FT3 (1.1 ± 0.34) and FT4 (0.7 ± 0.24) are slightly decreased compared to normal pregnancies (2.0 ± 0.95) and (1.2 ± 0.26). In the present study cholesterol levels were increased in abnormal pregnancies (228.9 ± 47.12) compared to normal pregnancies (212.2 ± 38.95) and decrease in HDL cholesterol were observed in preeclamptic and eclamptic pregnant women. In the present study Hb levels were decreased in abnormal pregnancies (8.8 ± 1.7) compared to normal pregnancies (7.9 ± 1.9). The levels of MDA and uric acid were significantly increased in abnormal pregnancies. The glucose (RBS) levels were increased compared to normal pregnancies [Table 1].

Table 1 : Showing the comparative study values of Normal and Abnormal pregnant women with statistical analysis

Sl. No.	Parameters	Normal pregnant women		Abnormal pregnant women		Z-value	P-value
		mean	S.D	mean	S.D		
1	FT3	2.0	0.95	1.1	0.34	0.3409	0.3669
2	FT4	1.2	0.26	0.7	0.24	0.223	0.4129
3	TSH	4.0	2.82	7.5	3.93	3.6231	0.001
4	MDA	6.9	3.27	9.2	8.28	1.292	0.001
5	Uric acid	4.1	0.69	6.9	1.59	8.092	0.001
6	RBS	85.7	12.27	165.1	68.82	5.68	0.001
7	Cholesterol	212.2	38.95	228.9	47.12	1.366	0.0869
8	HDL	35.9	11.62	28.7	6.68	2.680	0.0037
9	TGL	201.4	60.47	222.1	45.80	1.364	0.0869
10	Hb%	7.9	1.98	8.8	1.70	0.344	0.3669



Graph-1



Graph-2

IV. DISCUSSION

The present study was conducted to find out the thyroid status in abnormal pregnancies. Pregnancy is associated with substantial but reversible changes in thyroid function. The metabolic demands and hormonal changes which occur as a result of pregnancy induce a complex alteration in thyroid function. Many authors earlier reported the hypothyroidism in abnormal pregnancy^(13, 14, 15, 16). In the present study which included a group of 25 abnormal pregnant women, it is observed that TSH (7.5 ± 3.93) is significantly increased compared to normal pregnancies, TSH (4.0 ± 2.82). FT3 (1.1 ± 0.34) and FT4 (0.7 ± 0.24) are slightly decreased compared to normal pregnancies (FT3 2.0 ± 0.95 and FT4 1.2 ± 0.26).

Preeclampsia women have high incidence of hypothyroidism that might correlate with the severity of preeclampsia. It has been suggested that reduced serum concentrations of thyroid hormones in toxemia may be due to the loss of protein and protein bound hormones in urine. Modest decrease in thyroid hormones with concomitant increase in TSH levels in maternal serum correlated with severity of preeclampsia (or) eclampsia and high levels of endothelia. The endothelial cell dysfunction plays an important role in the pathogenesis of preeclampsia. Nitric oxide, a vasodilator released from the endothelial cells, regulates secretion of thyroid hormones by modulating regional blood flow. The decrease in thyroid hormones with significant increase in TSH has been found to be correlated with the severity of preeclampsia.

In the present study cholesterol levels were increased in abnormal pregnancies compared to normal pregnancies and decrease in HDL cholesterol were observed in preeclamptic and eclamptic pregnant women. Oestrogen is responsible for induction of TG and HDL and suppression of serum LDL and oestrogen levels falls in preeclampsia. The low level of HDL in preeclampsia is however not only because of hypoestrogenaemia but also due to insulin resistance. In the present study Hb levels were decreased in subjects compared to normal pregnancies. Pregnant women are often iron deficient, and iron deficiency had adverse effects on thyroid metabolism. Poor maternal iron status predicts both higher TSH and lower TT4 concentration during pregnancy. Many authors reported increased MDA levels in abnormal pregnancies⁽¹⁷⁾. In the present study also MDA levels is significantly increased (9.2 ± 8.28). The elevated levels of free radicals as evidence by elevated TBRS (thiobarbutric acid reactive substance) they are products of lipid per oxidation effecting cell membrane. Uncontrolled lipid peroxidation is a key contributing factor to pathophysiological condition of preeclampsia.

Uric acid levels were increased in abnormal pregnancies compared to normal, uric acid is one of the important redox systems in the body. Uric acid acts as an extracellular antioxidant because of its ability to remove singlet oxygen hydroxyl and peroxy radical. The current study assessed the effects of TSH on endothelial function, and its possible involvement of inflammation and oxidative stress. Raised TSH levels directly induces TNF α secretion by bone marrow cells and IL 6 by

adipocytes TNF α is a pivotal NO-controlling cytokine, and elevated TNF α levels may promote the expression of inducible NO synthase, leading to increased oxidative stress. So raised serum TSH levels leads to impairment of endothelium dependent vasodilatation, possibly through the induction of low grade inflammation and reduced NO availability by oxidative stress.

In some pregnant women oxidative stress may lead to dysmorphogenesis, abortions and intrauterine growth restriction. The study summarized the role of free radicals (or) reactive oxygen species mediate their action through many of the pro-inflammatory cytokines and this mechanism has been proposed as a common underlying factor for endometriosis, ovarian cancer, polycystic ovary disease, and various other pathologies affecting the female reproductive process.

V. CONCLUSION

TSH is significantly elevated and FT3, FT4 were decreased slightly in abnormal pregnancies compared with normals. TBRS (malondialdehyde) as marker of lipid peroxidation was elevated significantly. Antioxidant uric acid is elevated significantly. The rise in antioxidant is probably to compensate the increased peroxide load in abnormal pregnancies. Hb levels are decreased and RBS levels are significantly increased in abnormal pregnancies. Cholesterol, TGL levels were increased and HDL levels were significantly decreased in abnormal pregnancies. Study suggests that all abnormal pregnancies studied are associated with hypothyroidism.

REFERENCES RÉFÉRENCES REFERENCIAS

1. Khandakar MA¹, Ali MS, Kahtun M Thyroid status of normal pregnant women in Dhaka City. Mymensingh Med J. 2002 Jan; 11(1):1-5.
2. Thyroid Disease and Pregnancy. The American Thyroid Association website at www.thyroid.org
3. Wild, D., Immunoassay Handbook (2 nd edition), Stockton Press, 2001; 551.
4. Wosilait, W.D., Res. Com. Chem. Pathol. Pharmacol. 1977; 16,541.
5. Banker, S.B., Determination of Protein Bound Iodine, Journal Biological Chemistry. 1948; 173, 175.
6. Schettler, G. and Nussel, E., Arb. Med, soz. Med. 1975; 10, 25.
7. Gordon, T. et al., Am. J. Med. 1977; 62, 707.
8. Jacobs, N. J., Vandemark, P. J., Arch. Biochem. Biophys. 1960; 88, 250-255.
9. Kunyaji, Estimation of lipid peroxide, Methods in enzymology, 1984; vol. 105, 328-331.
10. Barham, D., Trinder P., Analyst. 1972; 97, 142.
11. Drabkin, DL. J. boils. Chem. 1932; 98, 719.
12. Trinder, P. Annals. Clin. Bio Chem, 1969; 6-24.

13. Mestman J, Goodwin TM, Montoro MM. Thyroid disorders of pregnancy. Endocrinol Metab Clin N Am 1995; 24:41-71.
14. Bishnoi A, Sachmechi I. Thyroid disease in pregnancy. American Family Physician 1996; 53:215-20.
15. Weetman AP, McGregor AM. Autoimmune thyroid disease: developments in our understanding. Endocr Rev 1984; 5:309-55.
16. Amino N, Tada H, Hidaka Y. Autoimmune thyroid disease in pregnancy. J Endocrinol Investig 1996; 19:59-69.
17. Hubel CA, Roberts JM, Taylor RN, Musci TJ, Rogers GM, McLaughlin MK. Lipid peroxidation in pregnancy: new perspectives on preeclampsia. American J Obstet Gynecol 1989; 161:1025-34.