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Original Research Article

STUDY OF SERUM LIPID PROFILE IN PREGNANCY INDUCED HYPERTENSION

Dr. Monika Jayaswal¹, Dr. Vijay Kumar ², Dr. (Prof.) Sude Kr. Singh ³

1- TUTOR, Dept. of Biochemistry, Darbhanga Medical College & Hospital, Laheriasarai, Darbhanga, Bihar.

2- Assistant Professor, Department of Medicine, Patna Medical College & Hospital, Patna, Bihar

3- Professor & Head, Dept. of Biochemistry, Darbhanga Medical College & Hospital, Laheriasarai, Darbhanga, Bihar.

Corresponding author: Dr. Monika Jayaswal,

Tutor, Department of Biochemistry

Darbhanga Medical College, Laheriasarai, Darbhanga, Bihar

Introduction

Hypertensive disorders of pregnancy encompass a spectrum of disease and constitute a public health problem throughout the world. Furthermore they are a major cause of severe maternal morbidity both acute and long term and adverse perinatal outcomes such as prematurity and Intrauterine growth retardation (IUGR). Hypertension in pregnancy has been classified into the following categories ¹ :-

- (a) Chronic Hypertension.
- (b) Gestational hypertension.
- (c) Preeclampsia-Eclampsia.
- (d) Preeclampsia superimposed on chronic hypertension.

Hypertension is defined as systolic blood pressure more than 140 mm of Hg or diastolic blood pressure more than 90 mm of Hg on at least two occasions at an interval of at least four to six hours². The association of serum lipid profile in essential hypertension is well documented. An abnormal lipid profile is known to be strongly associated with cardiovascular diseases and has a direct effect on endothelial dysfunction. The most important feature in toxemia of pregnancy is hypertension which is supposed to be due to vasospastic phenomenon in kidney, uterus, placenta and brain. Altered lipid synthesis leading to decrease in PGI₂/TXA₂ ratio is also supposed to be an important way of pathogenesis in pregnancy induced hypertension³. Thus abnormal lipid metabolism seems important in the pathogenesis of pregnancy induced hypertension (PIH) too. Obviously the association of serum lipid profile with pregnancy induced hypertension is highly suggested to reflect some new diagnostic tools. Moreover the hormonal imbalance is a prime factor for the etiopathogenesis of PIH and this endocrinal imbalance is well reflected in alteration of serum lipid profile⁴. Therefore simple measurement of serum lipid parameters may be of good predictive value in toxemia of pregnancy avoiding costly endocrinal investigations.

Aims and Objectives

This study was conducted to compare changes in maternal serum lipid profile in primiparous toxemic patients with pregnant normotensive females. Thus the aim of the study is to evaluate triglyceride and lipoprotein concentration in antenatal period and find out a cost effective and sensitive method for early detection and prevention of pregnancy induced hypertension.

Materials And Methods

The study was conducted in the Department of Biochemistry, Darbhanga Medical College & Hospital, Laheriasarai, Darbhanga, Bihar in collaboration with Dept. of obstetrics & Gynaecology, Darbhanga Medical College & Hospital, Darbhanga during the period of April 2017 to March 2018. All the subjects were of 18 to 40 years age group. Lipid profile which included total serum cholesterol, triglycerides, HDL-C, LDL-C, VLDL-C was studied in the following group of individuals.

1. Control Group :- Normal healthy pregnant women of 18 to 40 years age group.
2. Study Group :- Comprising of pregnant women suffering from pregnancy induced hypertension (PIH) of 18 to 40 years age group.

PIH is defined as blood pressure above 140 mm of Hg / 90 mm of Hg occurring in previously normotensive women. Grading of PIH was done as follows:-

Mild PIH – Blood pressure > 140/90 mm of Hg but less than 160/100 mm of Hg.

Severe PIH – Blood pressure > 160/100 mm of Hg.

Cases of twin pregnancy, pregnancy with diabetes mellitus, coexisting hypertensive or Heart diseases, Renal diseases, Thyroid disorders, Hydramnios, Hepatitis, gross obesity or any other complications were excluded from the study. Total number of patients in the study, 40 from normal pregnant women (control group) and 40 cases of pregnancy induced hypertension (25 mild PIH & 15 severe PIH) were included in the study.

Blood samples were drawn from all subjects following a fasting of 12 hours and analyzed for serum Triglycerides (TG) serum total cholesterol (TC), VLDL – cholesterol, LDL – Cholesterol, and HDL-cholesterol.

Serum triglycerides was estimated by GPO – PAP endpoint assay (span diagnostic kits)

serum Total cholesterol was estimated by CHOD-PAP method with ready to use diagnostic kits (Span diagnostic kits).

Serum HDL-C was estimated by PEG-CHOP-PAP endpoint assay with lipid clearing factor.

Serum VLDL was calculated as 1/5 th of Serum Triglycerides.

Serum LDL-C was calculated by Frederickson – Friedwalds formula according to which
 $LDL-C = TC - (HDL-C + VLDL-C)$.

Data was statistically analysed by student T Test and significance was expressed in terms of P value.

The P value < 0.05 was considered statistically significant.

OBSERVATIONS :-

Comparison of parameters among study groups

Parameters		Mild PIH (n=25)	Severe PIH (n=15)	Normal Pregnant women (n=40)
Serum Triglycerides(mg/dl)	Mean/SD T-Test(p)	241.71 ± 41.35 p<0.001	254.29 ± 28.98 P<0.001	163.14 ± 20.21
Serum Total Cholesterol (mg/dl)	Mean/SD T-Test(p)	291.02 ± 11.44 P<0.001	293.18 ± 8.54 P<0.001	258.76 ± 15.36
Serum HDL-C	Mean/SD	65.63 ± 5.49	57.88 ± 7.75	45.37 ± 7.24

(mg/dl)		T-Test(p)	P<0.001	P<0.001	
Serum LDL-C (mg/dl)		Mean/SD T-Test(p)	181.41 ± 6.67 p>0.05	187.00 ± 6.43 p>0.05	182.99 ± 13.58
Serum VLDL-C (mg/dl)		Mean/SD T-Test(p)	49.13 ± 8.42 P<0.001	50.11 ± 5.88 P<0.001	32.48 ± 3.99

DISCUSSION:-

In the present study normal pregnant woman were compared with mild PIH and severe PIH cases . Results have shown that serum Total cholesterol, triglycerides and HDL-C rose in mild PIH cases, but fall in HDL-C in severe PIH cases. Serum LDL-C rose in mild PIH cases and Severe PIH cases but were not significant. Serum VLDL-C rose progressively in mild PIH to severe PIH cases. The rise in total cholesterol has been confirmed by Oliver and Boyd⁵ (1934) , Mullick et al⁶(1964),Potter & Nestel⁷ (1979), Adegoke et al⁸ (2003), Hubbel et al⁹(1998).Serum triglycerides rose much more significantly in our study in PIH which goes with the finding of Enquobahrio et al (10) (2004), cekman(11) et al(2003). This may be due to hyperestrogenemia during pregnancy inducing biosynthesis of endogenous TG .Endothelial TG accumulation contributes to endothelial dysfunction (Mikhail(12) et al, 1995). This is explained by progressive decreased activity of Hepatic lipase, Lipoprotein lipase and LCAT. This leads to impairment at multiple sites in the elimination of TG as well as cholesterol during pregnancy and PIH.

The increase in VLDL-C in mild to severe PIH cases is due to hypertriglyceridemia .Thus due to increased VLDL concentration , these TG accumulate over vascular endothelium causing endothelial dysfunction and one important aspect in the pathogenesis is abnormal invasion of trophoblast in uterine vessels.(Sibai,2003).

SUMMARY & CONCLUSIONS:-

The results have shown that :-

1. TC – Serum level of TC rose in Mild & Severe PIH cases which is statistically significant.
2. TG – Serum level of TG rose in Mild & Severe PIH cases which is statistically significant.
3. HDL-C – Serum HDL-C rose in Mild & Severe PIH cases which is statistically significant.
4. VLDL-C – Serum VLDL-C rose in Mild & Severe PIH cases which is statistically significant.
5. LDL-C- Serum LDL-C also rose slightly in mild & severe PIH cases but is not statistically significant.

REFERENCES

1. August & Kohan Textbook of Obstetrics Medicine 6 th edition.
2. Harrisons principles of internal medicine , volume 2, 19 th edition
3. Dutta D.C. Textbook of Obstetrics 9 th edition.
4. Robson S.C.(1999) Hypertension and Renal Disease in pregnancy. In Dewhurst's Text Book Of Obs & Gynaecology For PG's Ed. Edmonds D.K. 6th Edition Blackwell Science Limited , New york Pg 67-69.
5. Oliver M.F. & Boyd G.S. (1934) Clinical SC. 14:15
6. Mullick S. Bagga , O.P. & Mullick V (1964) ; Am J Obs & Gynae 89;766
7. Potter , J.M.& Nestel , P.J.(1979) The hyperlipidemia of pregnancy in normal and complicated pregnancies. Am. J. obs & Gynae. (u.s) 133 (2), 165-70.
8. Adegoke, O.A., Lyare , E.E. and Gbenebitse , S.O.(2003) Postgraduate med. J.2010(1) 32-36.
9. Hubel C.A. Lyall. F , wissfeld. L,Gandley R.E. & Robertson J.M. (1998) Small low density lipoproteins and vascular cell adhesion molecule-1 are increased in association with hyperlipidemia in preeclampsia metabolism 47(10)1281-88.

10. Enquobahrie , D.A, Williams ,M.A, Butter, C.L., Fredrick, I.O., Miller R.S. and Lut Lyay D.A.(2204). Maternal plasma lipoprotein in early pregnancy Am. J. Hypertension.17(7) 574-81.
11. Cekman et al Plasma lipid and lipoprotein conc. In PIH clinical biochemistry .36(7),575-78.
12. Mikhail et al (1995) Lipid profile in women with preeclampsia. Relationship between plasma TG levels and severity of preeclampsia . Relationship between plasma TG levels and severity of preeclampsia J. assoc. Acad. Minor phys. 6(1) 43-45.