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A comparative study to assess distribution of serum lipid profile among pregnant Women and its association with pregnancy induced hypertension

Medical Sciences

KEYWORDS : Preeclampsia;Lipid Profile distribution.

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ABSTRACT

Introduction: Association of serum lipid profile with gestational proteinuric hypertension is highly suggested to reflect some new diagnostic tools. Simple measurement of serum lipid perameters may be of good predictive value in toxaemia of pregnancy, avoiding the costly endocrinal investigations

Objectives – To compare distribution of serum lipids among pregnant women.

To find out association of serum lipids with pregnancy induced hypertension.

Methods-study was carried out on pregnant women and non pregnant women attending or admitted in obstetrics and gynaecology department of NSCB Medical college and hospital, Jabalpur between 1st june 2012 to 31st October 2013. Each serum sample from two groups was evaluated for total cholesterol, Triglyceride,HDL-Cholestrol,LDL –Cholestrol and VLDL-Cholestrol . Comparison is drawn and analysed using chi-square or fisher's exact statistic as appropriate.

Result-Dyslipidemia is found in the form of significantly increased total cholesterol, LDL, VLDL & Triglyceride concentration in patients of preeclampsia as compare to normotensive pregnant women.

Conclusion: Dyslipidemia play an important role in pathophysiology of preeclampsia. The prevention and correction of various causative factors for dyslipidemia may significantly prevent development of preeclampsia.

INTRODUCTION

Human pregnancy is associated with pronounced physiological hyperlipidemia. Serum triglycerides and choloestrol level increases steadily as pregnancy progress. These changes are mediated by gestational hormones PIH affect the functions of various organs involved in lipid and lipoprotein metabolism, particularly the liver and kidney.

PIH associated with a typical serum lipoprotein pattern, characterized by higher concentration of Triglyceride,VLDL, LDL, TC.[1]The most important feature of PIH is hypertension via vasospasm in kidney, uterus, placenta and brain. Primary source of prostacyclin and thromboxane are endothelial cells and thrombocyte respectively.[2]In normal pregnant woman endothelial prostacyclines reaches 8-10 times more than a non pregnant woman, but in preeclamptic woman this rising is only 1-2 times more. Oxidized LDL inhibits endothelial prostacyclin synthesis and inactivates endothelial derived relaxing factor and also stimulate synthesis and release of endothelin hormone which has vascular smooth muscle contracting effect. The most important feature in toxaemia 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 hypertension.

Women with prior preeclamptic pregnancies are increased risk for cardiovascular diseases.[3]Preeclamptic women are insulin resistant during and after pregnancy and higher Triglyceride levels. Metabolic changes seen in preeclampsia, such as insulin resistant, hypertriglyceridemia and hypertension similar to metabolic syndrome, consequently a predisposition to metabolic syndrome may induce women to develop preeclampsia. Women with a history of pre-eclampsia have significant differences in lipid parameters and an increased susceptibility to lipoprotein oxidation when compared with women who had normal pregnancy. Disorders of lipoprotein metabolism are reported to be a major cause of hypertension and proteinuria in Pre-eclampsia[4].

Obviously the association of serum lipid profile with gestational proteinuric hypertension is higherly suggested to reflect some new diagnostic tools. The hormonal inbalance is a prime factor for the etiopathogenesis of PIH and this endocrinal inbalance is well reflected in alteration of serum lipid profile. There-fore simple measurement of serum lipid perameters may be of good predictive value in toxaemia of pregnancy, avoiding the costly endocrinal investigations[5].

This study was planned to assess the distribution of serum lipids with following objectives.

To compare distribution of serum lipids among pregnant women.

To find out association of serum lipids with pregnancy induced hypertension.

MATERIAL AND METHODS

Study design Cross sectional study

Study period 1 st June 2012 to 31 st October 2013.

Work place Department of Obstetrics and Gynecology N.S.C.B Medical College Jabalpur.

Study Groups All pregnant women Group A- Normotensive women.

Group B- Hypertensive women.

Inclusion Criteria- All pregnant women age group 20-30yrs with similar sociodemographic characters, parity, gravid, BMI- admitted in the department of Obst. & Gyne.,NSCB Medical College, Jabalpur.

Exclusion criteria- Patients with history of hypertension , known case of Diabetes,renal disease thyroid disease

Sample Size calculation- Sample size was determined using simple random sampling formulae- $N=z2pq/d^2$.

Where,z=1.96 at 5% alpha,95%of confidence and 80 power(beta),p is estimated prevalence of gestational hypertension which was assumed at 12%,q is 1-p and d is marginal error(absolute precision)which was considered 5%.

This yielded 113 numbers required. Further multiplied by 1.5 as design effect to adjust clustering of samples and finally found total required sample size was 169 or 170 in each group.

After taking Blood Sample from all patients, on each sample from different groups following Investigations done-

- Lipid Profile - Serum cholesterol
- HDL cholesterol
- Direct LDL cholesterol
- Serum triglyceride
- VLDL Cholesterol
- Cholesterol/HDL ratio

Statistical Analysis : Data analysis and statistical methods selection- All case report forms were checked for completeness and inappropriate or illogical responses. The forms were entered using Microsoft 2007 Excel worksheet. The databases were validated and all inconsistencies and differences were resolved. Statistical analyses were performed using STATA 12 for Windows (StataCorp LP, Texas, USA). Categorical data are presented as frequency counts (percent) and compared using the chi-square or Fisher's exact statistic as appropriate. Continuous data are presented as means (standard deviation) and compared using the t-test.

OBSERVATION

This cross sectional study was carried out in dapartment of Obstetric and Gynecology during a period 1st June 2012 to 31st October 2013.168 normal pregnant women and 172 cases of PIH were studied.In PIH patients ,the relevant finding were correlated with cardinal features of PIH namely hypertension,albuminuria and edema.These findings were compared with those obtained in group of patient with normal pregnancy.

TABLE-1

DISTRIBUTION OF CASES AND CONTROL IN DIFFERENT AGE GROUPS

Age Groups	Case		Control	
	No.	%	No.	%
<=20 yrs	30	17.44	29	17.26
20 - 25 yrs	119	69.19	109	64.88
26 - 30 yrs	18	10.47	20	11.90
31 - 35 yrs	4	2.33	7	4.17
=>36yrs	1	0.58	3	1.79
Total	172		168	

Pearson chi square = 2.3323 P = 0.675

The above table shows 69.19% of cases studied belonged to the age group 21-25yrs whereas 1% of cases above 35yrs and 17.44% were below 20 yrs.Means PIH women were mostly between the age group of 21-25 yrs.Only17.44% of them were comparatively younger and below 20yrs of age.

TABLE-2

DISTRIBUTION OF CASES AND CONTROL BASED ON PERIOD OF GESTATION AT TIME OF TEST

Period of gestation at time of test	Case		Control	
	No.	%	No.	%
32	143	83.14	18	10.71
33	16	9.30	132	78.57
34	12	6.98	10	5.95
35	1	0.58	8	4.76
Total	172		168	

Chi Sq: 193.6, P = < 0.01

In this 83.14% of cases were at 32 weeks and in control 78.57% were at 34 weeks of gestation. Difference in the two groups based on weeks of gestation was found to be highly significant. (P< 0.05)

TABLE-3

DISTRIBUTION OF CASES AND CONTROL ACCORDING TO THEIR GRAVIDITY

Gravida		Case		Control	
		N	%	N	%
Primi		125	72.67	89	52.98
Multi		47	27.33	79	47.02
	2	31	18.02	61	36.31
	3	12	6.98	16	9.52
	4	3	1.74	2	1.19
	7	1	0.58	0	0.00
Total		172		168	

Pearson chi square= 14.1380 P< 0.0001

It shows that 72.67% of cases were primigravida 18.02% cases were 2nd and 12%cases were 3rdgravida.Means most of cases of PIH more frequently in primigravida.

TABLE 4

COMPARISON OF SBP AND DBP IN CASES AND CONTROL

	Value	Mean	SD	[95% Conf. Interval]		t - test	p-value
Case	Systolic Blood Pressure (mm of Hg)	154.50	18.24	151.74	157.27	22.98	<0.0001
Control		117.99	9.63	116.52	119.45		
Case	Diastolic Blood Pressure (mm of Hg)	101.34	13.29	99.34	103.34	20.34	<0.0001
Control		77.98	6.79	76.94	79.01		

Blood pressure measurement was observed 154.50/101.34 (Systolic/Diastolic)mm of Hg in study subjects and 117.99/77.98 (Systolic/Diastolic)mm of Hg among control. The difference in systolic and diastolic blood pressure between study and control groups was found highly significant (P<0.0001). Study subjects were reported comparatively higher blood pressure than control groups.

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TABLE-5
DISTRIBUTION OF ELEVATED CHOLESTEROL LEVEL BETWEEN CASES AND CONTROL

group	Cholesterol(mg/dl)		Total	Odds Ratio (95% CI)
	Normal (150 to 200 mg/dl)	Abnormal (>200mg/dl)		
No of Case	67	102	169	1.42 (0.90 – 2.23)
%	39.64	60.36	100	
No of Control	81	87	168	1(Referent)
%	48.21	51.79	100	
Total	148	189	337	

Pearson chi square = 2.5119 P = 0.113
Fisher's exact = 0.125

Distribution was not found statistically significant (p>0.05), however it was observed that cases of PIH were 1.4 times more likely to elevated cholesterol level,as compared to controls.

TABLE -6

DISTRIBUTION OF ELEVATED HDL LEVEL BETWEEN CASES AND CONTROL

group	HDL(mg/dl)		Total	Odds Ratio (95% CI)
	Normal (35 to 60 mg/dl)	Abnormal (> 60mg/dl)		
No of Case	98	71	169	1.81 (1.12 – 2.93)
%	57.99	42.01	100	
No of Control	120	48	168	1(Referent)
%	71.43	28.57	100	
Total	218	119	337	

Pearson chi square = 6.6627 P < 0.01
Fisher's exact P< 0.0125

Distribution was found statistically highly significant (p<0.01),cases of PIH were1.81 times more likely to elevated HDL level as compared to control.

TABLE-7
DISTRIBUTION OF ELEVATED LDL LEVEL BETWEEN CASES AND CONTROL

group	LDL(mg/dl)		Total	Odds Ratio (95% CI)
	Normal (<130 mg/dl)	Abnormal (>130mg/dl)		
No of Case	98	71	169	2.40 (1.46 – 3.95)
%	57.99	42.01	100	
No of Control	129	39	168	1(Referent)
%	76.79	23.21	100	
Total	227	110	337	

Pearson chi square = 13.5397 P < 0.0001
Fisher's exact P< 0.0001

Distribution was found statistically highly significant (p<0.01),cases of PIH were 2.40 times more likely to elevated LDL level as compared to control.

TABLE -8
DISTRIBUTION OF ELEVATED TRIGLYCERIDE LEVEL BETWEEN CASES AND CONTROL

group	Triglyceride(mg/dl)		Total	Odds Ratio (95% CI)
	Normal (45 to 165 mg/dl)	Abnormal (>165mg/dl)		
No of Case	48	121	169	5.62 (3.43 – 9.23)
%	28.4	71.6	100	
No of Control	116	52	168	1(Referent)
%	69.05	30.95	100	
Total	164	173	337	

Pearson chi square = 55.71 P < 0.000Fisher's exact P< 0.0001

Distribution was found statistically highly significant (p<0.01),cases of PIH were5.62 times more likely to elevated TG level as compared to control.

TABLE– 9
DISTRIBUTION OF ELEVATED VLDL LEVEL BETWEEN CASES AND CONTROL

group	VLDL		Total	Odds Ratio (95% CI)
	Normal (9 to 30 mg/dl)	Abnormal (>30mg/dl)		
No of Case	39	128	167	4.95 (3.00 – 8.18)
%	23.35	76.65	100	
No of Control	101	67	168	1(Referent)
%	60.12	39.88	100	
Total	140	195	335	

Pearson chi square = 46.54 P < 0.0001
Fisher's exact P< 0.0001

DISCUSSION

Pregnancy induced hypertension (PIH) is a syndrome of hypertension in pregnancy, with or without edema and proteinuria. In a number of patients, the clinical appearance is mild, presenting only with small increase in blood pressure or protein in the urine. Though, in other patients severe maternal and fetal complications[6], such as the eclampsia, HELLP syndrome, preterm delivery, abruptio placenta, intra-uterine fetal growth restriction or fetal death may take place.

Different lines of evidence indicate that abnormal lipid metabolism is not a mere manifestation but is also involved in the pathogenesis of disease. The concentration of total cholesterol, LDL, VLDL & triglycerides was found to be significantly increased in preeclamptic female as compare to normal pragnent female. Elevated triglyceride values may compromise vascular function in several ways. for example triglyceride rich litoprotein has a prothrombotic activity. Elevated triglycride might shift the pattern of LDL subclass towords disproportionate increase in smaller danser more allergenic LDL particle. It also stated that early pregnancy dyslidemia is associated with increased risk of preeclampsia[7].

The danger of pregnancy induced hypertension is higher when the age of pregnant women is less than 25 years. In our study, 69.19% of the patients were less than 25 years of age. A study from Andhra Pradesh done by Mohanty S et al in 2006, India reported that primiparous patients with PIH below 20 years of age were 26% while only 15% of the controls were less than 20 years, signifying that younger age of pregnant women was a causative feature to preg-

nancy induced hypertension[8]. Therefore, it can be assumed from the results of this study that younger age of pregnant women are commonly associated with PIH.

In our study group of PIH patients 72.67% of cases were primiparous.

According to three cohort studies [9,10,11] primiparity almost triples the risk for pregnancy induced hypertension (OR 2.91, CI 1.28 - 6.61); this is supported by adjusted odds ratios for primiparity from two other cohort studies12,13. Different case-control studies[9,13-16] propose that women with pregnancy induced hypertension are two times expected to be primiparous as women without PIH (OR 2.35, CI 1.80 - 3.06). A study from Canada reported, that women with hypertensive disorders were more expected to be nulliparous (range 42.2% -78,2%) when matched with normotensive (4 1.9%) pregnant women. In our study group of PIH patients 72.67% of cases were primiparous.

Increase of serum lipids through pregnancy in general and during pregnancy induced hypertension in particular is described in a number of studies. Worldwide diverse studies have reported elevated[17,18] lipid levels in pregnancy induced hypertension patients.

In the present study,elevated level of cholesterol was observed 60.36% &51.79% in cases and controls respectively. The difference of % in elevated cholesterol in study cases subject to compared of control was observed about 16.54% . cholesterol concentration increased in the patients of pregnancy induced hypertension, but no significant changes in total cholesterol could be observed. These results are consistent with the findings reported in studies conducted in other populations[19,20].However some researchers Franz H et al in1992, Mohanty et al in 2006 have found considerable rise in serum cholesterol in toxemia of pregnancy[1,8].

Triglyceride(TG) level was significantly raise in patients than control(p<.0001). Elevated level of TG was observed 71.6 %&30.95% in cases and controls respectively.The difference of % in elevated level of TG in study cases subject to compared control was observed about 131.34%. In the Finnish and Peruvian population20,21 Kaaja Rel et al in 1995 and Ware js et al in 1999 that patients with pregnancy induced hypertension had higher mean triglyceride and lower mean serum HDL-C concentrations than the control group[21,22].

Low level of HDL-C in PIH is however not only because of hypoestrogenemia but also due to insulin resistance. In our study, elevated level of HDL was observed 42.01% &28.57%, in cases and controls respectively .The difference of % in elevated HDL in study cases compared to control was observed about 47.04%. the mean value HDL was about 51.34+_39.33mg/dl in the pregnancy induced hypertensive patients over the pregnant women with normal pregnancy(49.39+_14.65mg/dl.). Mean value of HDL-C level were higher in cases than control, but that was not statistically significant(p>.05) It means in our population HDL level not significantly changed in PIH patient. May be because of this they had less complication as compared to population of other geographic area. PIH is multifactorial disorder so racial and genetic differences in population of different geographic area also affects the lipid profile,which require further study. Belo et al in their study concluded that preeclamptic women exhibited,in third trimester higher mean serum triglyceride concerntration and lower high density lipoprotein cholesterol. They imphasized that this atherogenic lipid profile In preeclemptic females may be a potential contributor endothelial cell dysfunction.[23]

In present study VLDL-C level rose significantly(p<.0001) in patients, Elevated level of VLDL was observed 76.65% & 39.88% in cases and controls respectively.The difference of % in elevated VLDL in study cases compared to control was observed about 92.201%.Increased VLDL may be due to hypertriglyceridemia leading to increased entry of VLDL-C that carries endogenous triglyceride into circulation. According, to the reports of some Teichman AT et al in 1988,Kokia E et al in 1990 and Satter N et al in 1997. The VLDL-C level might raise up to 2.5 folds at term over the pre-pregnancy level.[24,25,19] VLDL-C level further elevates in pregnancy induced hypertension as found in the present study in validation with those of other researchers[19,26] perhaps due to increased VLDL-C which accumulate over the maternal vascular endothelium, mainly those of uterine and renal vessels.

The change in LDL –C was significant between the groups of patients and controls. LDL-C was significantly raise in cases than control(p<.025) Elevated level of LDL was observed 42.01% &23.21% ,in cases and controls respectively. The difference of % in elevated LDL in study cases compared to control was observed about 80.99%

A study conducted on Ghanaian women by Ahenkorahl et al in 2008 with pregnancy induced hypertension reported that there was a significant increase in triglycerides and LDL-C in the patient groups compared to the control. The lipid peroxidation marker Malondialdehyde (MDA), among the pregnancy induced hypertension subjects was significantly increased as compared to the normotensive pregnant women. A significant positive correlation between MDA and systolic and diastolic blood pressure was also observed. The study obviously indicated that Ghanaian women presenting with pregnancy induced hypertension are very prone to dyslipidemia as well as lipid peroxidation.[27]

We currently rely on secondary prevention of PIH.But derangement of lipid profile would have been started from earlier gestation.So by measuring lipid profile in first trimester we can predict chances of Preeclampsia and by taking precautions prevent this. We concluded that derangement of lipid profile significantly happen in preeclampsia and its play an important pathological role.It should be used as a diagnostic and prognostic indicator of preeclampsia.

These can studies further be extrapolated to not just prediction but also primary prevention of PIH by administration of appropriate antioxidants like vit c, vitE and vit D.

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