

Maternal Lipid Profile and Its Impact on Pregnancy Outcome

Shazia Parveen¹, Iffat Zaman², Nasreen Noor³, Shagufta Moin⁴, Syed Manazir Ali⁵

ABSTRACT

Aim: To evaluate the association of altered lipid levels and development of adverse maternal and fetal outcomes.

Materials and methods: Women attending outpatient department (OPD), antenatal care (ANC) and in patient department (IPD) of the Department of Obstetrics and Gynecology, Jawaharlal Nehru Medical College, Hospital, Aligarh Muslim University, Aligarh, were included in this study after obtaining informed consent and ethical approval from the institute.

Results: Mean age of pregnant women in the study group was 24.89 ± 3.12 years, whereas in the control group, it was 24.72 ± 3.76 years. Mean prepregnancy weight was 51–55 kg in both study and control groups. Mean BMI of the pregnant women in the study group was 21.89 ± 1.89 kg/m², whereas in the control group, it was 21.52 ± 1.47 kg/m². The levels of total cholesterol, triglycerides (TG), low density lipoprotein (LDL) and VLDL were significantly high in women who developed gestational diabetes mellitus (GDM) when compared to those who did not develop GDM and the difference was found to be statistically significant ($p < 0.001$). The levels of total cholesterol, TG, and VLDL were significantly high in women who developed preeclampsia as compared to the women who did not develop preeclampsia ($p < 0.05$). The levels of total cholesterol, TG, LDL and VLDL in women who developed preterm labor were found to be significantly high in women who developed preterm labor ($p < 0.01$) as compared to women who did not have PTL. The levels of total cholesterol, TG, LDL, and VLDL in women who developed intrahepatic cholestasis of pregnancy (IHCP) were statistically significant when the two groups were compared ($p < 0.001$). The levels of total cholesterol, TG, LDL, VLDL, in women who developed macrosomia were not statistically significant when the two groups were compared ($p > 0.05$). Women who developed small for gestational age (SGA) had statistically significant high levels of total cholesterol, TG, LDL, and VLDL ($p < 0.05$).

Conclusions: The present cohort study was undertaken in the north Indian population to explore the association between dyslipidemia in pregnancy and its adverse maternal and fetal outcomes. It was seen that third trimester maternal dyslipidemia is associated with various maternal and fetal complications such as gestational diabetes mellitus, preeclampsia, IHCP, preterm labor, and SGA babies.

Keywords: Gestational diabetes mellitus, Lipid profile, Macrosomia, Preeclampsia, Small for gestational age.

Journal of South Asian Federation of Obstetrics and Gynaecology (2022): 10.5005/jp-journals-10006-2051

INTRODUCTION

Pregnancy is a physiological state in which there is an alteration in lipid levels. There is accumulation of TG-rich remnants in maternal circulation due to reduced lipolysis of TG-rich lipoproteins, reduced uptake by the placental tissue, and concomitant decrease in lipoprotein lipolysis.¹ During pregnancy there is an increased levels of both TG and TC, which are essential for the development of fetus;^{2–5} however, high levels are associated with adverse outcomes like gestational diabetes mellitus, preterm labor,⁶ pregnancy-induced hypertension (PIH),^{7,8} large for gestational age babies.^{9–11} Conversely decreased level of total cholesterol is associated with SGA babies.^{12,13} These may have a long-term impact on the health of the baby and mother. Previous researches have shown that pregnancy-induced hyperlipidemia contributes to increased occurrence of gestational diabetes mellitus and preeclampsia. Despite this, there are still controversies on the relationship between maternal lipid disturbances and pregnancy complications and perinatal outcomes. So the present cohort study was undertaken to explore the association between dyslipidemia in pregnancy and its adverse pregnancy outcome.

In this study, we aim to evaluate the association of lipid profile and development of GDM, preeclampsia, preterm labor, IHCP, and adverse fetal outcome in the form of SGA, macrosomia, NICU admission, and stillbirth.

^{1,2}Department of Obstetrics and Gynecology, JNMCH, Aligarh, Uttar Pradesh, India

³Department of Obstetrics and Gynecology, JNMC, AMU, Aligarh, Uttar Pradesh, India

⁴Department of Biochemistry, Jawaharlal Nehru Medical College and Hospital, Aligarh Muslim University, Aligarh, Uttar Pradesh, India

⁵Department of Paediatrics, Jawaharlal Nehru Medical College and Hospital, Aligarh Muslim University, Aligarh, Uttar Pradesh, India

Corresponding Author: Shazia Parveen, Department of Obstetrics and Gynecology, JNMCH, Aligarh, Uttar Pradesh, India, e-mail: shzparveen@gmail.com

How to cite this article: Parveen S, Zaman I, Noor N, et al. Maternal Lipid Profile and Its Impact on Pregnancy Outcome. *J South Asian Feder Obst Gynae* 2022;14(3):302–306.

Source of support: Nil

Conflict of interest: None

MATERIALS AND METHODS

The present study was conducted on women attending OPD, ANC, and IPD of the Department of Obstetrics and Gynecology, Jawaharlal Nehru Medical College, Hospital, Aligarh Muslim University, Aligarh, after obtaining informed consent from the

women and ethical approval from the institute. The study was prospective cohort type. For the present study, a total of 200 pregnant women beyond 28 weeks, naturally conceived with singleton pregnancies, were included and women with multiple pregnancy, diabetes mellitus, inherited metabolic diseases coronary artery disease, and chronic hypertension were excluded. Women were divided into two groups: study group—100 women having deranged lipid profile; control group—100 women having normal lipid profile.

After informed consent, all women underwent routine investigations, GST with 75 gm glucose and lipid profile. Venous blood samples were taken after overnight fasting for the estimation of lipids. Normal lipid profile included total cholesterol <200 mg%, TG <150 mg%, high density lipoprotein (HDL) 30–70 mg%, LDL <100 mg%, and VLDL 2–30 mg%.

To diagnose GDM, we followed Diabetes in Pregnancy Study Group of India (DIPSI) criteria where all pregnant women beyond 28 weeks of gestation were given 75 gm of glucose with 200–300 mL of water irrespective of last meal. Venous sugar level was recorded after 2 hours, a value of >140 mg% was assigned as GDM.

Pregnancy-induced hypertension was diagnosed with a systolic BP of 140 mm Hg or more and diastolic BP of 90 mm Hg or more on two occasions 4 hours apart.

Preterm labor was diagnosed with the onset of painful uterine contractions before 37 weeks of pregnancy with cervical dilatation and effacement.

Intrahepatic cholestasis of pregnancy was diagnosed in third trimester with pruritus and biochemical evidence of deranged liver functions.

Statistics: Chi-square test and student *t*-test.

OBSERVATION AND RESULTS

In our study, the mean age of pregnant women in the study group was 24.89 ± 3.12 years, whereas in the control group, it was 24.72 ± 3.76 years. Both groups were comparable with regard to age distribution.

The maximum number of pregnant women had prepregnancy weight of 51–55 kg in both study and control group. Both groups were comparable with regards to prepregnancy weight.

The mean BMI of the pregnant women in study group was 21.89 ± 1.89 kg/m², whereas in the control group, it was 21.52 ± 1.47 kg/m². Both groups were comparable with regard to prepregnancy BMI. On comparing the data, there was no statistical significant difference among the two groups (*p* > 0).

In the study group, 20% pregnant women developed GDM while in control group 17% pregnant women developed GDM.

No statistically significant difference was observed when the two groups were compared (*p* > 0.05).

Lipid profile of women who developed GDM in the study group was compared with those who did not develop GDM. The mean cholesterol, TG, LDL, and VLDL among women who developed GDM in study group were 313.10 ± 27.77, 245.20 ± 19.77, 128.40 ± 8.38, 59.15 ± 3.28, respectively, while in women who did not develop GDM was 281.11 ± 47.49, 205.37 ± 42.63, 120.55 ± 12.33, 51.07 ± 11.40, respectively. The mean HDL in women who developed GDM was 39.50 ± 5.79 while in women who did not develop GDM was 40.41 ± 5.91. The increase in total cholesterol, TG, LDL, and VLDL was statistically significant (*p* < 0.01) when the groups were compared while there was no statistically significant difference in HDL levels when the groups were compared (*p* > 0.05) (Table 1).

In the study group, 13% pregnant women developed preeclampsia while in control group 2% women developed preeclampsia. Statistically significant difference was observed when the two groups were compared (*p* < 0.01).

Lipid profile of women who developed preeclampsia in the study group was compared with those who did not develop preeclampsia. The mean cholesterol, TG, and VLDL among women who developed preeclampsia in study group were 320.15 ± 33.95, 243.23 ± 32.34, 59.15 ± 5.41, respectively, while women who did not develop preeclampsia were 282.63 ± 45.68, 208.87 ± 41.83, 51.72 ± 11.07. The mean HDL and LDL among women who developed preeclampsia in study group were 42.30 ± 5.76 and 127.76 ± 7.38, respectively, with women who did not develop preeclampsia women were 39.91 ± 5.86 and 121.27 ± 12.39. The increase in HDL and LDL was statistically not significant when the two groups were compared (*p* > 0.05). The increase in total cholesterol, TG, and VLDL was statistically significant when the two groups were compared (*p* < 0.05) (Table 2).

In the study group, 12% pregnant women developed preterm labor while in control group 18% pregnant women developed preterm labor. No statistical significant difference was observed when the two groups were compared (*p* > 0.05).

Lipid profile of the women who developed preterm labor in the study group was compared with those who did not develop preterm labor. The mean cholesterol, TG, LDL, and VLDL among women who developed preterm labor in study group were 324.33 ± 21.48, 251.25 ± 19.93, 131.08 ± 8.12, 61.75 ± 2.76, respectively, with women who did not develop preterm labor were 282.48 ± 46.19, 208.17 ± 41.85, 120.89 ± 11.99, 51.45 ± 10.89. The mean HDL was 40.75 ± 4.24 in women who developed preterm labor with women who did not develop preterm labor was 40.15 ± 6.08. The increase in total cholesterol, TG, LDL, and VLDL was statistically significant when the two groups were compared (*p* < 0.01) while

Table 1: Comparison of lipid profile of women who developed GDM vs who did not develop GDM in study group

Sl. No.		GDM		<i>t</i> -value	<i>p</i> -value
		Yes	No		
1	Total cholesterol	313.10 ± 27.77	281.11 ± 47.49	2.884	<i>p</i> < 0.01
2	Triglyceride	245.20 ± 19.77	205.37 ± 42.63	4.058	<i>p</i> < 0.001
3	HDL	39.50 ± 5.79	40.41 ± 5.91	-0.619	<i>p</i> > 0.05
4	LDL	128.40 ± 8.38	120.55 ± 12.33	2.689	<i>p</i> < 0.01
5	VLDL	59.15 ± 3.28	51.07 ± 11.40	3.123	<i>p</i> < 0.01

GDM, gestational diabetes mellitus; HDL, high density lipoprotein; LDL, low density lipoprotein



Table 2: Comparison of lipid profile of women who developed preeclampsia vs who did not develop preeclampsia in the study group

Sl. No.		Preeclampsia		t-value	p-value
		Yes	No		
1	Total cholesterol	320.15 ± 33.95	282.63 ± 45.68	2.841	p <0.01
2	Triglyceride	243.23 ± 32.34	208.87 ± 41.83	2.833	p <0.01
3	HDL	42.30 ± 5.76	39.91 ± 5.86	1.373	p >0.05
4	LDL	127.76 ± 7.38	121.27 ± 7.38	1.836	p >0.05
5	VLDL	59.15 ± 5.41	51.72 ± 11.07	2.369	p <0.05

HDL, high density lipoprotein; LDL, low density lipoprotein

Table 3: Comparison of lipid profile of women who developed preterm labor vs who did not develop preterm labor in study group

Sl. No.		Preterm labor		t-value	p-value
		Yes	No		
1	Total cholesterol	324.33 ± 21.48	282.48 ± 46.19	3.082	p <0.01
2	Triglyceride	251.25 ± 19.93	208.17 ± 41.85	3.5	p <0.01
3	HDL	40.75 ± 4.24	40.15 ± 6.08	0.325	p >0.05
4	LDL	131.08 ± 8.12	120.89 ± 11.99	2.847	p <0.01
5	VLDL	61.75 ± 2.76	51.45 ± 10.89	3.247	p <0.01

HDL, high density lipoprotein; LDL, low density lipoprotein

Table 4: Comparison of lipid profile of women who developed IHCP vs who did not develop IHCP in the study group

Sl. No.		IHCP		t-value	p-value
		Yes	No		
1	Total cholesterol	321.03 ± 15.11	273.81 ± 47.34	5.248	p <0.001
2	Triglyceride	238.24 ± 19.87	203.16 ± 44.72	4.054	p <0.001
3	HDL	36.93 ± 5.24	41.57 ± 5.61	-3.827	p <0.001
4	LDL	129.34 ± 9.57	119.16 ± 11.73	4.136	p <0.001
5	VLDL	60.20 ± 3.12	49.61 ± 11.30	4.954	p <0.001

HDL, high density lipoprotein; IHCP, intrahepatic cholestasis of pregnancy; LDL, low density lipoprotein

there was no statistically significant difference in HDL levels when the groups were compared ($p <0.05$) (Table 3).

In the study group, 29% pregnant women developed IHCP while in control group 5% pregnant women developed IHCP. Statistically significant difference was observed when the two groups were compared ($p <0.001$) (Table 3).

Lipid profile of the women who developed IHCP in the study group was compared with those who did not develop IHCP. The mean cholesterol, TG, LDL, VLDL, and HDL among women who developed IHCP in study group were 321.03 ± 15.11, 238.24 ± 19.87, 129.34 ± 9.57, 60.20 ± 3.12, 36.93 ± 5.24, respectively, while the women who did not develop IHCP were 273.81 ± 47.34, 203.16 ± 44.72, 119.16 ± 11.73, 49.61 ± 11.30, 41.57 ± 5.61, respectively. The increase in total cholesterol, TG, LDL, VLDL, and HDL, was statistically significant when the two groups were compared ($p <0.001$) (Table 4).

In the study group, 2% pregnant women had macrosomic baby while in control group 8% pregnant had macrosomia. Study group had higher incidence of macrosomia when compared to control group. No statistical significant difference was observed when the two groups were compared ($p >0.05$).

Lipid profile of the women who developed macrosomic baby in the study group was compared with those who did not

develop macrosomia. The mean cholesterol, TG, LDL, VLDL, and HDL among women who developed macrosomia in study group were 321.00 ± 18.38, 230.50 ± 10.60, 43.50 ± 7.77, 43.50 ± 7.77, 60.00 ± 4.24, respectively, while women who did not develop macrosomia were 286.82 ± 46.15, 212.98 ± 42.55, 40.16 ± 5.86, 121.93 ± 12.06, 52.54 ± 10.84, respectively. The increase in total cholesterol, TG, LDL, VLDL, and HDL was not statistically significant when the two groups were compared ($p >0.05$) (Table 5).

In the study group, 8% pregnant women developed SGA while in control group 10% pregnant women developed SGA. Control group had higher incidence of SGA as compared to study group. No statistical significant difference was observed when the two groups were compared ($p >0.05$).

Lipid profile of the women who developed SGA in fetus in the study group was compared with those who did not develop SGA. The mean cholesterol, LDL, VLDL, TG, and HDL in women who developed SGA in study group were 317.75 ± 22.03, 130.37 ± 7.72, and 60.62 ± 1.76, 229.37 ± 30.64, and 43.62 ± 7.65, respectively, while the women who did not develop SGA were 284.88 ± 46.63, 121.40 ± 12.10, 52.00 ± 10.97, 211.94 ± 42.91, 39.93 ± 5.65, respectively. The increase in total cholesterol, LDL and VLDL as compared with women who did not develop SGA and the difference was found to be statistically significant ($p <0.05$) (Table 6).

Table 5: Comparison of lipid profile of women who developed macrosomia in fetus vs who did not develop macrosomia in the study group

Sl. No.		Macrosomia		t-value	p-value
		Yes	No		
1	Total cholesterol	321.00 ± 18.38	286.82 ± 46.15	1.041	p > 0.05
2	Triglyceride	230.50 ± 10.60	212.98 ± 42.55	0.579	p > 0.05
3	HDL	43.50 ± 7.77	40.16 ± 5.86	0.793	p > 0.05
4	LDL	43.50 ± 7.77	121.93 ± 12.06	1.054	p > 0.05
5	VLDL	60.00 ± 4.24	52.54 ± 10.84	0.968	p > 0.05

HDL, high density lipoprotein; LDL, low density lipoprotein

Table 6: Comparison of lipid profile of women who developed SGA vs who did not develop SGA in study group

Sl. No.		SGA		t-value	p-value
		Yes	No		
1	Total cholesterol	317.75 ± 22.03	284.88 ± 46.63	1.968	p < 0.05
2	Triglyceride	229.37 ± 30.64	211.94 ± 42.91	1.122	p > 0.05
3	HDL	43.62 ± 7.65	39.93 ± 5.65	1.72	p > 0.05
4	LDL	130.37 ± 7.72	121.40 ± 12.10	2.055	p < 0.05
5	VLDL	60.62 ± 1.76	52.00 ± 10.97	2.211	p < 0.05

HDL, high density lipoprotein; LDL, low density lipoprotein; SGA, small for gestational age

DISCUSSION

Certain physiological changes during pregnancy, including lipid metabolism, support fetal growth and development. The accumulation of adipose cells in the tissues and hepatic lipid synthesis increases and this physiological adaptation is associated with changes in lipid profile during pregnancy. There is increased concentration of TC, TG, LDL-C and decrease in HDL-C during normal pregnancy. Accumulation of lipids in maternal tissues and the development of maternal hyperlipidemia occur in pregnancy. In some cases, a maladaptation occurs and these levels increase over the physiological range leading to dyslipidemia which causes complications like preeclampsia, GDM, and preterm labor.

Our study showed results in consistence with the studies done by Jin et al., Abdu Helmy et al., and Sharami et al.¹⁴⁻¹⁶ where there was significant association between GDM, preeclampsia, preterm labor, and IHCP and deranged lipids. Studies done by Anuradha et al., Singh et al., and Shen et al.,^{17,18} have also shown the positive correlation between dyslipidemia and preeclampsia.

As regard to fetal outcome, studies done by Abdu Helmy et al., Sharami et al., and Jin et al. showed the positive correlation between the deranged lipids and the occurrence of macrosomia and SGA.

We conclude from this study that maternal dyslipidemia is associated with various maternal and fetal complications such as gestational diabetes mellitus, preeclampsia, IHCP, preterm labor, and SGA babies. So evaluation of lipid profile during second and third trimesters can predict these pregnancy-associated complications which helps in counseling the pregnant women to have a modified life style with increased physical activities, dietary modifications, and timely interventions when required as the treatment of hyperlipidemia is a challenging issue because most of the drugs used for the treatment of dyslipidemia belong to category C or X. As the sample size of our study was small with limited time duration, further studies with a large sample size should be done to make a recommendation.

REFERENCES

1. Singh A, Kujur A, Jain P. Feta-maternal impact of altered lipid profile in pregnancy. *Int J Reprod Contracept Obstet Gynecol* 2017;7(1):132. DOI: 10.18203/2320-1770.ijcog20175547.
2. Vrijkotte TG, Krukziener N, Hutten BA, et al. Maternal lipid profile during early pregnancy and pregnancy complications and outcomes: the ABCD study. *J Clin Endocrinol Metab* 2012;97(11):3917-3925. DOI: 10.1210/jc.2012-1295.
3. Maurkiewicz JC, Watts GF, Warburton FG, et al. Serum lipids, lipoproteins and apolipoproteins in pregnant non-diabetic patients. *J Clin Pathol* 1994;47(8):728-731. DOI: 10.1136/jcp.47.8.728.
4. Sattar N, Greer IA, Loudon J, et al. Lipoprotein subfraction changes in normal pregnancy: threshold effect of plasma triglyceride on appearance of small, dense low density lipoprotein. *J Clin Endocrinol Metab* 1997;82(4):2483-2491. DOI: 10.1210/jcem.82.8.4126.
5. Oguru K, Miyatake T, Fakui O, et al. Low density lipoprotein particle diameter in normal pregnancy and preeclampsia. *J Atheroscler Thromb* 2002;9(5):42-47. DOI: 10.5551/jat.9.42.
6. Catov JM, Ness RB, Wellons MF, et al. Prepregnancy lipid related to preterm birth risk: the coronary artery risk development in young adults study. *J Clin Endocrinol Metab* 2010;95(6):3711-3718. DOI: 10.1210/jc.2009-2028.
7. Jan MR, Nazli R, Shah J, et al. A study of lipoprotein in normal and pregnancy induced hypertensive women in tertiary care hospitals of the North West Frontier Province-Pakistan. *Hypertensive Pregnancy* 2012;31(7):292-299 [Bibliography 93]. DOI: 10.3109/10641955.2010.507843.
8. Ziaei S, Bonab KM, Kazemnejad A. Serum lipid levels at 28-32 weeks gestation and hypertensive disorders. *Hypertens Pregnancy* 2006;25(8):3-10. DOI: 10.1080/10641950500543756.
9. Di Cianni G, Miccoli R, Volpe L, et al. Maternal triglyceride levels and newborn weight in prepregnant women with normal glucose tolerance. *Diabet Med* 2005;22(9):21-25. DOI: 10.1111/j.1464-5491.2004.01336.x.
10. Kitajima, Oka S, Yasuhi I, et al. Maternal serum triglycerides 24-36 weeks gestation and newborn weight in nondiabetic women with positive diabetic screens. *Obstet Gynecol* 2001;97(10):776-780. DOI: 10.1016/s0029-7844(01)01328-x.

11. Kushtagi P, Arvapally S. Maternal mid pregnancy serum TAG and neonatal birth weight. *Int J Gynaecol Obstet* 2009;106(11):258–259. DOI: 10.1016/j.ijgo.2009.03.004.
12. Edison RJ, Berg K, Remaley A, et al. Adverse birth outcome among mothers with low serum cholesterol. *Pediatrics* 2007;120(12):723–733. DOI: 10.1542/peds.2006-1939.
13. Sattar N, Greer IA, Galloway PJ, et al. Lipid and lipoprotein concentrations in pregnancies complicated by intra uterine growth restriction. *J Clin Endocrinol Metab* 1999;84(13):128–130. DOI: 10.1210/jcem.84.1.5419.
14. Jin WY, Lin SL, Hou RL, et al. Associations between maternal lipid profile and pregnancy complications and perinatal outcomes: a population-based study from China. *BMC Pregnancy Childbirth* 2016;16:60. DOI: 10.1186/s12884-016-0852.
15. Helmy MA, El-Latif EMA, Mohamed MF, et al. Relation between maternal lipid profile and pregnancy complications and perinatal outcomes. *AIMJ* 2020;1(11):179. DOI: 10.21608/aimj.2021.46596.1337.
16. Sharami SH, Abbasi Ranjbar Z, Alizadeh F, et al. The relationship of hyperlipidemia with maternal and neonatal outcomes in pregnancy: a cross sectional study. *Int J Reprod BioMed* 2019;17:739–748. DOI: 10.18502/ijrm.v17i10.529.
17. Anuradha R, Durga T. Estimation of lipid profile among preeclampsia woman by comparing with normal pregnancy. *Int J Contemporary Med Res* 2016;3(7):1958–1961. <https://dx.doi.org/10.18203/2320-1770.ijrcog20195348>.
18. Shen H, Liu X, Chen Y, et al. Associations of lipid levels during gestation with hypertensive disorders of pregnancy and gestational diabetes mellitus: a prospective longitudinal cohort study. *BMJ Open* 2016;6:e013509. DOI: 10.1136/bmjopen-2016-013509.