

Research Article

Comparative Analysis of Serum Lipid Profile between Normotensive and Hypertensive Pakistani Pregnant Women

Rabia Anjum¹, Nureen Zahra¹, Kanwal Rehman², Rabail Alam¹, Asia Parveen¹, Muhammad Tariq³ and Muhammad Sajid Hamid Akash^{2,4*}

¹Institute of Molecular Biology and Biotechnology, The University of Lahore, Lahore, Pakistan ²Institute of Pharmacology, Toxicology and Biochemical Pharmaceutics, College of Pharmace

²Institute of Pharmacology, Toxicology and Biochemical Pharmaceutics, College of Pharmaceutical Sciences, Zhejiang University Hangzhou, China

³Department of Pharmacy, The University of Lahore, Lahore, Pakistan

⁴College of Pharmacy, Government College University Faisalabad, Faisalabad, Pakistan

Abstract

Pregnancy-induced hypertension (PIH) is one of a major cause of maternal mortality. Serum lipid profile plays pivotal role in the regulation of normal blood pressure during pregnancy. The aim of our study was to evaluate the alteration of serum lipid profile during normotensive and hypertensive pregnancy. This case-control study was conducted among the pregnant women visiting Jinnah Hospital Lahore, Pakistan from September, 2012 to March, 2013. Total 200 participants were evaluated out of which 50 were normotensive pregnant women (28 ± 8 years) taken as a normal control group and 150 were enrolled as hypertensive (30 ± 6 years) study group. Average blood pressure for normotensive pregnant women was 115/75 whereas, for hypertensive pregnant women it was 148.45/95.40. The levels of different serum lipids were measured using respective analytical kits. Hypertension was directly associated with increased levels of serum TGs (161.02 \pm 3.58 vs. 105.31 \pm 8.53), TC (188.90 \pm 4.11 vs. 152.45 ± 1.99), LDL (136.50 ± 3.17 vs. 70.48 ± 2.14) and VLDL (117.06 ± 1.05 vs. 41.06 ± 1.70), and fall in HDL (49.41 ± 1.56 vs. 37.16 ± 1.64) as compared to that of normotensive pregnant women. The increased levels of TGs, TC, and LDL in hypertensive pregnant women were also correlated with significantly increased values of TC/HDL, TGs/HDL and LDL/HDL in hypertensive pregnant women. From the results of our study, it can be concluded that lipid profile plays their critical role in regulating blood pressure during pregnancy. Increased levels of TC, TGs, LDL and VLDL induced hypertension, whereas, HDL regulated the blood pressure to normal levels. This association may be significant in understanding the development of hypertension during pregnancy and may help in developing the strategies for prevention and treatment of PIH.

Keywords: Triglycerides; LDL; HDL; Total cholesterol; VLDL; Normotensive pregnancy; Hypertensive pregnancy

Introduction

Pregnancy-induced hypertension (PIH) is one of the major risk factors in present day health care practice because it not only causes maternal mortality but also impairs fetal development during pregnancy [1-3]. Hypertension is directly associated with increased levels of total cholesterol (TC), triglycerides (TGs), low density lipoproteins (LDL) and very low density lipoproteins (VLDL) whereas, at the same time, the levels of high density lipoproteins (HDL) are decreased. TC, TGs, LDL and VLDL are known as bad cholesterols as they play their destructive role in several diseases.

Normally, in early pregnancy, altered levels of serum lipid profile increase the risk of PIH [4,5]. Pregnant women having hypertension usually have high levels of TC, TGs, LDL and VLDL whereas, the levels of HDL are decreased as compared to that in normal pregnant women [3]. PIH may cause several critical problems in pregnancy such as premature delivery, intrauterine growth restriction, fetal death, maternal mortality and morbidity [6]. Several factors responsible for PIH have been identified [7,8] but among them the most important are lipid profiles [2,3,5,9-11]. It has been reported that metabolism of lipoproteins is directly associated with PIH [3,11,12].

Keeping in view of the significant role of lipid profile such as TC, TGs, HDL, LDL and VLDL in PIH, the aim of our study was to compare the altered lipid profile in hypertensive pregnant women with that of normotensive pregnant women. Moreover, we also calculated the ratios of TC/HDL, TGs/HDL and LDL/HDL, and it was found that increased values of these ratios were directly correlated with increased levels of TC, TGs and LDL in hypertensive pregnant women.

Materials and Methods

Study protocol

The present study was conducted at Jinnah Hospital in Lahore, Pakistan between September 2012 and March 2013. The protocol of this study was dually approved by the Institutional Ethics Committee, Jinnah Hospital Lahore, Pakistan for sample collection and Institute of Molecular Biology and Biotechnology, The University of Lahore, Lahore, Pakistan for sample analysis. The informed written consent was obtained from all the participants. A total 200 participants were enrolled in this study selected from the Gynecology ward of Jinnah Hospital Lahore, Pakistan. After recruitment, blood pressure (BP) was measured and on the basis of BP, all the participants were divided into two groups i.e. normotensive pregnant women (28 \pm 8 years) and hypertensive pregnant women (30 \pm 6 years). Normotensive pregnant women were represented as a control group whereas; hypertensive pregnant women were taken as a test group for evaluation of serum lipid profile.

*Corresponding author: Muhammad Sajid Hamid Akash, Institute of Pharmacology, Toxicology and Biochemical Pharmaceutics, College of Pharmaceutical Sciences, Zhejiang University Hangzhou, China and College of Pharmacy, Government College University Faisalabad, Faisalabad, Pakistan, E-mail: sajidakash@gmail.com

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Exclusion criteria

Patients with all maternal and/or fetal abnormalities, known renal disease, diabetes, hepatic dysfunction, dyslipidaemia and pre-existing hypertension before pregnancy expect PIH were excluded from this study in both control and test groups.

Inclusion criteria

All the participants had similar low socio-economic status and dietary habit. The hypertension was diagnosed by the presence of persistent hypertension (more than 140/90 mm of Hg) measured 6 hours apart [13]. The participants having persistent hypertension were on a salt restricted diet.

Blood pressure measurements

Blood pressure was recorded by qualified obstetrics nurses in the Gynecology ward of Jinnah Hospital, Pakistan using mercury sphygmomanometer and stethoscope from the left upper arm after the subjects had been sitting for more than 5 minutes according to the guidelines of American Heart Association [14]. Triplicate readings were recorded after 5 minutes rest interval between the measurements and the average value was recorded.

Sample collection and preparation

Peripheral fasting blood samples were collected from all participants in Gynecology ward of Jinnah Hospital, Pakistan and dispensed into vacunaiter^{*} plain tubes and then were taken to Institute of Molecular Biology and Biotechnology, The University of Lahore, Lahore, Pakistan for centrifugation. The serum obtained after centrifugation was used for estimation of serum lipid profile.

Biochemical analysis of serum lipid profiles

The serum levels of the lipid profile such as TC, TGs, HDL, LDL and VLDL were measured by enzymatic methods with the help of Glaxo kits on ERBA Chem-5 semi auto analyzer at Institute of Molecular Biology and Biotechnology, The University of Lahore, Lahore, Pakistan.

Statistical analysis

Values for both normotensive and hypertensive pregnant women were expressed as mean \pm SD. Level of significance between hypertensive pregnant women and normotensive pregnant women were performed using the Student *t*-test. *P* value < 0.05 was considered statistically significant. GraphPad Prism (version 5.0) was used for statistical analysis. Comparison of BP and serum lipid profile between normotensive and hypertensive pregnant women was calculated using Pearson Correlation Coefficient.

Results

Clinical data were collected from normotensive and hypertensive pregnant women to characterize the lipid profile and BP within study groups (Table 1). Body mass index was significantly increased in all the participants as was expected from inclusion criteria (data not shown). Mean BP (SBP/DBP) was significantly increased in hypertensive pregnant women as compared to that in normotensive pregnant women (Table 1). In hypertensive pregnant women, the serum levels of TC, TGs, LDL and VLDL were significantly high (P<0.05) whereas, the serum level of HDL was significantly low (P<0.05) when directly compared with that of normotensive pregnant women (Table 1). We also calculated the ratios of various lipid profiles such as TC/HDL, TGs/ HDL and LDL/HDL. The values of these ratios (TC/HDL, TGs/HDL and LDL/HDL) for hypertensive pregnant women were significantly higher as compared to that in normotensive pregnant women (Table 1). The significant difference between the ratios of various lipid profile in hypertensive and normotensive pregnant women was significantly correlated with that of serum levels of these lipid profile in hypertensive pregnant women.

The consistency of high SBP/DBP was maintained throughout the whole pregnancy period in pregnant women when directly compared to SBP/DBP of normotensive pregnant women (Table 2). We also measured the levels of serum lipid profile of all participants in all trimester periods (1st, 2nd and 3rd) and then compared these serum levels between normotensive and hypertensive pregnant women (Table 2). The serum levels of TC, TGs, LDL and VLDL were consistently increased (P<0.01) whereas, the serum level of HDL was consistently decreased (P<0.01) from 1st trimester towards 3rd trimester in hypertensive pregnant women. Moreover, we also compared these two groups. The serum levels of TC, TGs, LDL and VLDL in hypertensive pregnant women were significantly very high (P<0.05) as compared to that of normotensive pregnant women. Contrarily, the serum level of HDL was consistently remained very low (P<0.05) in hypertensive

Parameters	Participants					
	Normotensive (n = 50)	Hypertensive (n = 150)	P-value			
BP (mm Hg)	115/75	148.45/95.40	0.001 [*]			
TC (mg/dL)	152.45 ± 1.99	183.90 ± 4.11	0.162			
TGs (mg/dL)	105.31 ± 8.53	161.02 ± 3.58	0.010 [*]			
HDL (mg/dL)	49.41 ± 1.56	37.16 ± 1.64	0.012			
LDL (mg/dL)	70.48 ± 2.14	136.50 ± 3.17	0.000*			
VLDL (mg/dL)	41.06 ± 1.70	117.06 ± 1.05	0.034*			
TC/HDL	3.08 ± 0.00	5.08 ± 0.00	0.021 [*]			
TGs/HDL	2.13 ± 0.00	4.49 ± 0.00	0.013*			
LDL/HDL	1.42 ± 0.00	3.67 ± 0.00	0.036 [*]			

Abbreviations: BP: Blood pressure; TC: Total cholesterol; TGs: Triglycerides; HDL: High density lipoproteins; LDL: Low density lipoproteins; VLDL: Very low density lipoproteins; 'Significant at *P* < 0.05

Table 1: Comparison of lipid profiles between normotensive and hypertensive pregnant women.

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Parameters	Participants						
	Normotensive Pregnant Women			Hypertensive Pregnant Women			
	1 st Trimester	2 nd Trimester	3 rd Trimester	1 st Trimester	2 nd Trimester	3 rd Trimester	
BP	115/75	118/75	120/77	148.18/95 ª	157.52/98.69 ^b	159.82/100 °	
тс	152.45 ± 0.99	156.56 ± 2.05	147.36 ± 1.26	183.16 ± 9.61 ª	189.57 ± 7.64 ^b	198.49 ± 6.12 °	
TGs	105.31 ± 3.53	99.68 ± 2.56	107.52 ± 1.89	161.91 ± 8.24 ª	173.70 ± 7.11 ^b	177.75 ± 6.45 °	
HDL	49.41 ± 1.56	46.34 ± 0.89	52.08 ± 2.05	37.181 ± 2.58 ª	35.17 ± 3.22 ^b	33.39 ± 2.31 °	
LDL	70.48 ± 0.14	74.48 ± 1.26	69.43 ± 1.09	136.2 ± 5.78 ª	145.30 ± 5.50 b	152.50 ± 4.59 °	
VLDL	41.06 ± 0.70	39.18 ± 2.82	43.28 ± 1.15	117.66 ± 2.19 ª	124.13 ± 1.05 ^b	129.35 ± 0.87 °	
TC/HDL	3.08 ± 0.00	3.37 ± 0.00	2.82 ± 0.00	5.08 ± 0.00 ª	5.30 ± 0.00 b	5.85 ± 0.00 °	
FGs/HDL	2.13 ± 0.00	2.06 ± 0.00	2.64 ± 0.00	4.49 ± 0.00 ª	4.93 ± 0.00 b	5.32 ± 0.00 °	
LDL/HDL	1.42 ± 0.00	1.59 ± 0.00	1.33 ± 0.00	3.67 ± 0.00 ª	4.13 ± 0.00 b	4.56 ± 0.00 °	

Abbreviations: BP: Blood Pressure; TC: Total Cholesterol; TGs: Triglycerides; HDL: High Density Lipoproteins; LDL: Low Density Lipoproteins; VLDL: Very Low Density Lipoproteins; a: P<0.05 versus 1st trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; c: P<0.05 versus 3rd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normotensive pregnant women; b: P<0.05 versus 2nd trimester of normote

Table 2: Comparison of lipid profiles in different trimesters of normotensive and hypertensive pregnant women.

pregnant women in all trimesters when directly compared with that of normotensive pregnant women (Table 2).

pregnant women (Table 2). Similar types of findings were also observed in previous studies [19,20,38-40].

Discussion

In our present study, we investigated the role of lipid profile in the regulation of blood pressure throughout the pregnancy period. We used normotensive pregnant women as a control group, whereas, hypertensive pregnant women were test group. It is common that with the increase in pregnancy age, lipid profiles increased gradually [15-18]. In our present study, serum levels of TC, TGs, LDL and VLDL were significantly increased whereas, the level of HDL was significantly decreased in hypertensive pregnant women (Table 1). PIH is characteristically associated with hypertriglyceridemia. The principal modulator of hypertriglyceridemia is oestrogen which is also associated with hyperoestrogenaemia during pregnancy [19]. Oestrogen induces hepatic production of TGs that causes PIH and endothelial dysfunction through the generation of LDL and VLDL [3,20-22]. Endothelial dysfunction is the most important event in the pathogenesis of hypertension during pregnancy and abnormal levels of lipid profile play their critical role in the induction of endothelial dysfunction [23,24]. The findings of TGs, LDL and VLDL in our study are inconsistent with already published reports in which increased levels of these lipid profile were observed in hypertensive pregnant women [3,5,11,12,17,22,25-28]. Other chronic inflammatory diseases like diabetes [29-33] and cancerous conditions like leukemia [34] are also known to affect the serum lipid profile [35-37]. We have observed that the serum lipid profile of normotensive pregnant women showed insignificant differences among all three pregnancy trimesters (Table 2). Contrarily, in hypertensive pregnant women, the serum levels of TC, TGs, LDL and VLDL were significantly increased (P<0.01) during the 3rd trimester of pregnancy as shown in table 2.

In our present study, no significant (P<0.05) alteration of TC could be found in the 3rd trimester of normotensive pregnant women when compared with the serum levels of TC in 1st trimester of normotensive pregnant women. The serum levels of TC in the 3rd trimester of hypertensive pregnant women were significantly higher (P<0.01) when compared with the serum levels of TC in 1st trimester of hypertensive

HDL is good cholesterol for our health as it regulates the BP towards normal levels. In our present study, the serum levels of HDL were not significantly changed (P<0.05) in the 3rd trimester of normotensive pregnant women (Table 2). In hypertensive pregnant women, the serum levels of HDL were significantly decreased in the 3rd trimester of pregnancy. The decreased levels of HDL in the 3rd trimester of hypertensive pregnant women in our study are in agreement with already published reports [3,17,22,41]. We also calculated the ratios of lipid profiles (TC/HDL, TGs/HDL and LDL/HDL) in our present study. The values of these lipid profile ratios followed the same pattern as TGs, TC, HDL and LDL observed in normotensive and hypertensive pregnant women (Table 2). Although, the ratios of these lipid profiles have not been established yet in PIH but the increase in lipid profile during pregnancy cannot be overlooked as it may indicate the risk of PIH. From the results of our study, it can be found that serum levels of TC, TGs, HDL, LDL and VLDL are more profound in the 3rd trimester of pregnancy in hypertensive pregnant women as compared to that of normotensive pregnant women.

Investigational studies to evaluate the therapeutic outcomes of different therapeutic modalities are also important in different diseases [42-50]. Therefore, it is important to conduct such studies in which management of PIH is evaluated with different treatment modalities.

Conclusion

To conclude, the findings of our study suggest that abnormal levels of lipid profile especially TGs, TC, LDL and VLDL may contribute in the promotion of hypertension in pregnant women. This association may help to investigate the underlying pathological process of hypertension in pregnancy. It is therefore imperative that serum lipid profiles should be continuously monitored throughout the whole pregnancy period as it would be helpful in the early detection and/or developing the strategies to prevent any obstetric-associated complication during PIH and/or at the time of delivery.

Conflict of Interest

The authors declare that they do not have any conflict of interest for this article.

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