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Research Article

**ASSESSMENT AND IMPORTANCE OF LIPID PROFILE IN
PRE-ECLAMPSIA WOMEN****¹Dr. Tooba Hameed, ²Dr. Sana Iqbal, ³Dr. Aasma Nighat Zaidi, ⁴Dr. Nauman Aziz,
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Medicine and Dentistry, Lahore, ^{4,5}Sahiwal Medical College, Sahiwal, ⁶Mayo Hospital, Lahore.**Article Received:** January 2019**Accepted:** February 2019**Published:** March 2019**Abstract:****Objective:** The aim of this study is to know the assessment and importance of lipid profile in preeclampsia women.**Study Design:** A cross-sectional analytical study.**Place and Duration:** In the Physiology Department of Nishtar Medical University, Multan in Collaboration with Gynecology and Obstetrics Department of Nishtar Hospital Multan for One year Duration from September 2017 to September 2018.**Methods:** The study consisted of 90 subjects, each of which consisted of 30 people. 30 were apparently healthy subjects, 30 mild preeclamptic and 30 severe preeclamptic. Clinical details were collected. Fasting blood samples were obtained by aseptic methods and serum lipid profile was analyzed.**Results:** Of the 90 subjects studied, 30 had normal triglycerides (176.76 mg / dl). However, 30 subjects with severe preeclampsia show serum triglycerides 242.mg / dl according to normal. The difference in both groups was statistically more significant than normal.**Conclusion:** It was concluded that high circulating levels increased with preeclampsia in the pathogenesis of preeclampsia associated with preeclampsia, which may be a risk factor during pregnancy.**Key words:** Lipids, Preeclampsia.**Corresponding author:****Dr. Tooba Hameed,**

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INTRODUCTION:

Preeclampsia is defined as proteinuria (24 hours urine proteins > 300 mg) or hypertension after 20 weeks of pregnancy [1]. Hypertension is determined as an increase in blood pressure of 30/15 mmHg from the reference value, which is confirmed by 2 readings at 6 hours difference in which blood pressure would be greater than 140/90 mmHg. Preeclampsia is a specific human pregnancy disease that negatively affects the mother due to the fetus (due to intrauterine growth restriction) and (due to vascular dysfunction) [2]. The preeclampsia incidence is between 3% and 10% of all pregnancies and there is no verification that this changes significantly. Preeclampsia is determined by increased peripheral vascular resistance, decreased organ perfusion and increased vasospasm. It has been suggested that blood pressure falls from the first trimester to the second trimester for healthy pregnant women and increases again in the third trimester [3]. For women who develop pregnancy hypertension or preeclampsia, blood pressure remains constant in the first half of pregnancy and increases continuously after birth [4]. The preeclampsia risk raised in females with a history of antiphospholipid antibodies, preeclampsia, twin pregnancy, pre-existing diabetes mellitus (DM), family history, null parity, women of maternal age under 20 years of age or high body mass index before pregnancy (BMI) [5]. The risk increases with a previous pregnancy, autoimmune disease, kidney disease and chronic hypertension for 10 years or longer [6]. Preeclampsia is divided into severe and mild forms depending on hypertension severity, the degree to which other organ systems are affected and the amount of proteinuria [7]. Vasospasm is the main event in PIH. Vasospasm itself may have a harmful effect on blood vessels. The endothelial damage is caused by Segmental dilatation and arteriolar spasm. The endothelial contraction is due to Angiotensin II [8]. These changes cause leakage of interendothelial cells in which fibrinogen, blood components and platelets are deposited subendothelially [9]. These changes in vascular system lead to bleeding, necrosis and other endogenous changes observed in heavy PPH, along with ischemia of surrounding tissues. Lipid peroxidation is normally a process that occurs in all tissues and cells at low levels. It includes transformation of unsaturated fatty acids to lipid hydroperoxides and primary products known as various secondary metabolites by oxidation. Any

imbalance between proximal and antioxidant forces dominated by the antioxidants can be defined as oxidative stress, which is an important cause of lipid peroxidation in general. Although peroxidation of lipids affects many primary reaction sites, cellular components include protein thiols and polyunsaturated fatty acids associated with membrane. In the disease process, Oxidative stress plays an important role. In the placenta, the reactive oxygen species synthesis increases and the antioxidant enzymes activities are declined. There is also verification of lipid peroxidation in the placenta and maternal blood. Endothelium seems to be the target organ for the preeclampsia process [11]. The change in the function of the endothelial cells in the preeclampsia involves coagulation cascade activation, increased membrane permeability, better response to suppressors, and increased vasoconstriction, which contributes to the reduction of perfusion affected organs. In various ways, Endothelial cells can be activated including lipoproteins, free fatty acids, lipid peroxides or oxidized lipoproteins, fragments of restricted cytotrophoblastic microvessels and fibronectin degradation products.

MATERIALS AND METHODS:

This cross-sectional analytical study was held in the Physiology Department of Nishtar Medical University, Multan in Collaboration with Gynecology and Obstetrics Department of Nishtar Hospital Multan for One year Duration from September 2017 to September 2018.

This analysis was carried out in 90 pregnant women aged between 16 and 32 years and gestational age between 26 and 34 weeks.

Group A = normal healthy pregnant women as control group.

Group B = mild preeclamptic female Group C = severe preeclamptic women Clinical details were recorded. Blood samples were collected by aseptic methods and analyzed for serum lipid profile.

RESULTS:

Table 1 shows the comparison of mild, severe mean, age and height averages (SEM) among women in the preeclamptic control group, the mean age of the mild preeclamptic group (19.9 ± 0.55) and the severe preeclamptic group (21.63 ± 0.75) was significantly lower than control (23.96 ± 0.82).

Table 1: comparison of maternal age height, weight in normal pregnant women (control) and preeclamptic groups (All the values are expressed in Mean \pm SEM)

Variable	Group A Normal Pregnant Women (Control) n=30	Group B Mild Preeclamptic Women n=30	Group C Severe Preeclamptic Women n=30
Age (years)	23.96 \pm 0.82	19.90 \pm 0.55	21.63 \pm 0.75
Weight (Kg)	58.00 \pm 84	60.60 \pm 1.20	60.90 \pm 1.23
Height (m)	1.54 \pm 0.01	1.55 \pm 0.01	1.54 \pm 0.01

N = Number of subjects

** = $P < 0.05$ when compared to control

*** = $P < 0.001$ when compared to control

There were no significant changes in weight and height in the control and preeclamptic groups.

Table 2: comparison of systolic and diastolic blood pressure in control group and preeclamptic group (All the values are expressed in Mean \pm SEM)

Variable	Group A Normal Pregnant Women (Control) n=30	Group B Mild Preeclamptic Women n=30	Group C Severe Preeclamptic Women n=30
Systolic blood pressure (mmHg)	112.83 \pm 2.41	149.33 \pm 1.43	183.16 \pm 2.49
Diastolic blood pressure (mmHg)	76.00 \pm 48	104.16 \pm 0.93	121.83 \pm 1.40

N = Number of subjects

*** = $P < 0.001$ when compared to control

Table 2 shows that the mean value of systolic and diastolic blood pressure in patients with mild preeclampsia (149.33 \pm 1.43) and severe preeclamptic women (183.16 \pm 0.93) and (121.83 \pm 1.40) as comparison with control group significantly higher (112.83 + 2.41) and (76.00 \pm 1.48).

Table 3: comparison of fasting triglycerides and total cholesterol in control group and preeclamptic group (All the values are expressed in Mean \pm SEM)

Variable	Group A Normal Pregnant Women (Control) n=30	Group B Mild Preeclamptic Women n=30	Group C Severe Preeclamptic Women n=30
Triglycerides (md/dl)	176.76 \pm 8.05	235.53 \pm 11.54	242.00 \pm 13.56
Total Cholesterol (mg/dl)	198.53 \pm 7.21	212.73 \pm 7.12	229.30 \pm 8.13

N = Number of subjects

** = $P < 0.01$ when compared to control

*** = $P < 0.001$ when compared to control

Table 3 shows that the mean value of serum triglycerides in the mild preeclamptic group (235.53 \pm 11.54) and in the severe preeclamptic group (242.00 \pm 13.56) was significant compared to the control group (176.76 \pm 8.05). There was a

statistically significant difference in serum cholesterol levels in severe preeclampsia (229.3 \pm 8.13) compared to control group (198.53 \pm 7.21) (no significant difference was found between mild preeclamptic values compared to control group).

Table 4: comparison of fasting high density lipoprotein cholesterol, low density lipoprotein cholesterol level in control group and preeclamptic group (All the values are expressed in Mean \pm SEM)

Variable	Group A Normal Pregnant Women (Control) n=30	Group B Mild Preeclamptic Women n=30	Group C Severe Preeclamptic Women n=30
High density lipoprotein cholesterol (mg/dl)	31.30 \pm 1.50	28.13 \pm 1.37	27.33 \pm 0.97
Low density lipoprotein cholesterol (mg/dl)	131.86 \pm 7.18	137.50 \pm 6.59	163.07 \pm 6.10
Very low density lipoprotein cholesterol (mg/dl)	35.28 \pm 1.61	47.10 \pm 2.30	49.71 \pm 3.05

N = Number of subjects

** = P<0.01 when compared to control

*** = P<0.001 when compared to control

Table 4 shows that the difference between HDL (28.13 \pm 1.37) and LDL-C (137.50 \pm 6.59) cholesterol values was not significant compared to the control group among the mild preeclampsia groups. A significant difference was observed in HDL-C (27.33 \pm .97) and LDL-C (153.67 \pm 6.10) compared to control among severe preeclamptic groups. We found a significant difference in VLDL cholesterol between mild preeclamptic group (47.10 \pm 2.3) and severe preeclamptic group (49.71 \pm 3.05) compared to control group (35.28 \pm 1.6).

DISCUSSION:

Preeclampsia remains one of the main causes of fetal and maternal mortality and morbidity. Despite for many years of research work, these diseases etiology is not known. Recent verifications suggests that there may be various underlying pathology or predisposing factors leading to dysfunction of endothelium and leading to symptoms of proteinuria, edema and hypertension¹². Many hypotheses have been proposed for the pathogenesis of the disease and include endothelial dysfunction, prostacyclin - thromboxane imbalance, absolute or relative placental ischemia and immunogenetic. This study was conducted to know the lipid profile in preeclampsia and to investigate the possibility that lipids are a sign of the severity of preeclampsia¹³. There was a significant difference between normal pregnant women and mild and severe preeclampsia in terms of maternal age, but there was no difference in weight and height in normal pregnant women and preeclamptic groups. Similar findings of weight and height were observed by hannele etal¹⁴. Normal human pregnancy leads to a significant physiological hyperlipidemia, including blood triglycerides and increased cholesterol. Women with preeclampsia show an additional change in blood lipids that reflect a disorder in the metabolism of lipids and lipoproteins. In this study, patients in the

mild preeclampsia group showed a significant difference in total cholesterol compared to the control group. Although the average level in mild preeclampsia was slightly higher, a significant difference was observed in cholesterol levels in severe preeclamptic women compared to normal pregnant women¹⁵. In this study, significant differences were found in triglyceride levels in preeclamptic groups compared to control group. These findings are suitable for the study of power et al. These results are suitable for the operation of the satar18. Hubel et al. reported that very low density lipoprotein increased cholesterol in preeclamptic patients. Significant elevated VLDL cholesterol levels were found in preeclamptic groups compared to normal pregnant.

CONCLUSION:

We have observed that lipid profile levels are strongly associated with preeclampsia. This suggests that high lipids may play a role in pathogenesis with preeclampsia and are a risk marker for women.

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