

Maternal Lipid Profile as A Risk Factor for Preeclampsia

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ABSTRACT

Background: Preeclampsia occurs in about 3-5% of pregnancies and is an important cause of fetal and maternal morbidity and mortality worldwide. Preeclampsia occurs during second and third trimester of pregnancy, and it is more common in nulliparous women. It is characterized by blood pressure of 140/90 mmHg or rise in systolic blood pressure of more than 30 mmHg or diastolic blood pressure of more than 15 mmHg after 20 weeks of gestation, in conjugation with proteinuria >300 mg/24 hours or greater or equal to 1+ or 100 mg/dl by dipstick response. Hypertriglyceridemia is traditionally defined as a serum or plasma triglyceride (TG) concentration above 1.6 mmol/l. It is found in 13% of woman aged 20-40 years. **Objectives:** Assessing the changes in lipid profile in preeclampsia and as a marker of severity of the condition. **Patients and Methods:** This was a control study conducted at Al-Hussien and Air Force Hospitals during the period from December 2016 to May 2017. Total 100 pregnant women of 18-36 years in the 2nd half of pregnancy (≥ 20 weeks of gestation) were selected and grouped as follows. Group-I (Controls): 40 Normotensive pregnant women; Group-II 30 Mild pre-eclamptic pregnant women and Group-III 30 severe pre-eclamptic pregnant women. **Conclusion:** Preeclampsia has an association with hypertriglyceridemia, and elevated cholesterol level, LDL and VLDL, and decreased HDL level. The more the severity of preeclampsia the higher was the level of serum triglycerides, cholesterol, LDL and VLDL, and the lower the HDL.

Keywords: Lipid Profile, Preeclampsia, Hypercholesterolemia, Hypertriglyceridemia

INTRODUCTION

Preeclampsia occurs in about 3-5% of pregnancies and is an important cause of fetal and maternal morbidity and mortality worldwide ⁽¹⁾.

Preeclampsia occurs during second and third trimester of pregnancy and it is more common in nulliparous women. It is characterized by blood pressure of 140/90 mmHg or rise in systolic blood pressure of more than 30 mmHg or diastolic blood pressure of more than 15 mmHg after 20 weeks of gestation, in conjugation with proteinuria >300 mg/24 hours or greater or equal to 1+ or 100 mg/dl by dipstick response ⁽²⁾.

Hypertriglyceridemia, traditionally defined as a serum or plasma triglyceride (TG) concentration above 1.6 mmol/l, is found in 13% of woman aged 20-40 years ⁽³⁾.

The association of alteration of serum lipid profile in preeclampsia is well documented. Known to be strongly associated with atherosclerotic cardiovascular diseases and has a direct effect on endothelial dysfunction. The most important feature in preeclampsia is hypertension which is supposed to be due to vasospastic phenomenon in kidney, uterus, placenta and brain ⁽⁴⁾.

Altered lipid synthesis leading to decrease in prostacyclin (PGI₂): Thromboxane A₂ (TXA₂) ratio is also supposed to be an important way of pathogenesis in pregnancy induced hypertension ⁽⁵⁾.

Thus, abnormal lipid metabolism seems important in the pathogenesis of preeclampsia. Preeclampsia and related disorders are known to affect function of various organs involved in lipid and lipoprotein metabolism. Several studies have

shown that endothelial dysfunction is related to hyperlipidemia ⁽⁶⁾

Significantly elevated plasma concentration of triglycerides (TG) were found in women with preeclampsia in comparison to normal pregnancy ⁽⁷⁾.

Plasma lipids and lipoproteins increase during pregnancy. The mechanism for pregnancy-induced changes in lipids is not completely understood, but appears to be partly caused by elevated estrogen, progesterone and human placental lactogen ⁽⁸⁾.

Lipid levels in women with preeclampsia are reported to be higher than those in healthy pregnant women ⁽⁹⁾.

Women with preeclampsia present arterial lesions at the uteroplacental implantation site. These morphological lesions are usually observed in cases of acute atherosclerosis, and are characterized by areas with fibrinoid necrosis surrounded by lipid-laden macrophage ⁽⁹⁾. These microscopic lesions are similar to atherosclerosis found outside pregnancy. Lipid deposits are also seen in the glomeruli of pre-eclamptic patients, a finding known as glomerular endotheliosis. Glomerular lesions are associated with proteinuria, a predictive indicator and marker of disease severity ⁽²⁾.

It has also been suggested that triglycerides may be involved in this renal damage ⁽¹³⁾.

The severity of both hypertension and proteinuria seems to reflect the degree of endothelial damage ⁽²¹⁾. The possible correlation between the altered lipid profile and the severity of renal lesions, as reflected by proteinuria, may contribute towards clarify the complex pathophysiology of preeclampsia.

Pregnancy is associated with physiologic hyperlipidemia, and in normal pregnancy, this feature is not atherogenic that is believed to be under hormonal control ⁽¹³⁾. Women who develop preeclampsia, experience more dramatic lipid changes compared with normotensive women.

Also patients with hyperlipidemia, especially hypertriglyceridemia have a higher incidence of and are prone to develop more severe cases of preeclampsia ⁽²¹⁾.

The aim of this study was to assess the changes in lipid profile in preeclampsia and as marker of severity of the condition.

PATIENTS AND METHODS

This was a control study which was conducted at Al-Hussien and Air Force Hospitals during the period from December 2016 to May 2017. **The study was approved by the Ethics Board of Al-Azhar University.**

Total 100 pregnant women of 18-36 years in the 2nd half of pregnancy ≥ 20 weeks of gestation, were selected and grouped as follows. **Group-I (Controls): 40** Normotensive pregnant women; **Group-II: 30 Mild** preeclamptic pregnant women and **Group-III: 30 Severe** preeclamptic pregnant women

Preeclampsia was defined as development of blood pressure $\geq 140/90$ mm Hg and proteinuria ≥ 300 mg as confirmed by 24 h urine collection in women with no known history of hypertension, renal disease, endocrine abnormalities and had single pregnancy and had no family history of lipid or carbohydrate disorders.

Preeclampsia with severe features is defined as the presence of one of the following symptoms or signs in the presence of preeclampsia⁽⁴⁾:

- Systolic blood pressure (SBP) of 160 mm Hg or higher or diastolic blood pressure (DBP) of 110 mm Hg or higher, in two occasions at least 4 hours apart while the patient is on bed rest (unless antihypertensive therapy has previously been initiated)
- Impaired hepatic function as indicated by abnormally elevated blood concentrations of liver enzymes (to double the normal concentration), severe persistent upper quadrant or epigastric pain that does not respond to pharmacotherapy and is not accounted for by alternative diagnoses, or both.
- Progressive renal insufficiency (serum creatinine concentration >1.1 mg/dL or a doubling of the serum creatinine concentration in the absence of other renal disease)
- New onset cerebral or visual disturbances
- Pulmonary edema
- Thrombocytopenia (platelet count $<100,000/\mu\text{L}$)

All patients underwent full history taking, as well as general and local examination to determine whether inclusion and exclusion criteria applied to them.

The aim of the study was explained to them and an informed consent was taken. The study was approved by the hospital ethics committee.

History regarding height and weight of the study group and controls, during the 1st trimester was noted from their antenatal registration cards and information taken from them, as preeclampsia results in excessive weight gain due to water retention ⁽⁶⁾.

BMI was calculated as per formula: Weight (Kg)/Height (meter)² (Quetelet's Index). The subjects and controls were examined for vital signs, pulse, and blood pressure.

Blood pressure was measured in supine position or sitting position first by palpatory method and then by auscultatory method. According to Korotkoff sounds, appearance of sound (phase I) was taken as systolic BP, and disappearance of (phase V) Korotkoff sound was taken as diastolic BP.

In this study serum triglyceride level, cholesterol, HDL, LDL and albumin in urine were measured to all participants after 20 weeks of gestation.

Urine albumin (protein) was estimated using Urine Dipsticks.

Inclusion Criteria:

- 1- Age between 18:36 years.
- 2- Pregnancies beyond 20 weeks of gestation but not in labor.
- 3- With gestational hypertension (Blood pressure $\geq 140/90$ mm Hg) or preeclampsia (blood pressure $\geq 140/90$ mm Hg and proteinuria ≥ 300 mg/dl over 24 hours).
- 4- Age, BMI, gravidity matched in healthy pregnant women beyond 20 weeks of gestation and not in labour.

Exclusion Criteria:

- Chronic hypertension
- Preeclampsia superimposed on chronic hypertension
- Eclampsia
- Any other medical illness
 - Diabetes mellitus
 - Renal disorders
 - Liver disorders
 - Cushing's syndrome
 - Obesity
- Drugs affecting lipid profile
 - Estrogens
 - Lipid lowering drugs
 - Corticosteroids
 - Anti-epileptics
 - Alcohol intake

Statistical Analysis

Data were collected, revised, coded and entered to the Statistical Package for Social Science (IBM SPSS) version 20. Qualitative data were presented as number and percentages while quantitative data were presented as mean, standard deviations and ranges when parametric. The comparison between two groups with qualitative data were done by using *chi-square test* and/or *Fisher exact test* was used instead of Chi-square test when the expected count in any

cell was found less than 5. The comparison between more than two independent groups regarding quantitative data with parametric distribution was done by using *one way analysis of variance (ANOVA)* followed by post hoc analysis using *LSD test*. The confidence interval was set to 95% and the margin of error accepted was set to 5%. So, the p-value was considered significant as the following: P > 0.05: Non-significant, P < 0.05: Significant and P < 0.01: Highly significant.

RESULTS

Table (1): Comparison between the three studied groups regarding demographic data

| | | Normal | Mild | Severe | Chi-square test | | Post Hoc analysis by LSD | | |
|-----------------|---------|--------------|--------------|--------------|--------------------|---------|--------------------------|--------|--------|
| | | No.=40 | No.=30 | No.=30 | X ² /F* | P-value | p1 | P2 | P3 |
| Age | Mean±SD | 30.53 ± 3.91 | 29.6 ± 3.92 | 29.4 ± 3.81 | 0.856 | 0.428 | 0.326 | 0.233 | 0.842 |
| | Range | 19 - 36 | 18 - 36 | 21 - 36 | | | | | |
| Gravidity | Mean±SD | 2.78 ± 1.21 | 2.57 ± 1.22 | 2.83 ± 1.18 | 0.415 | 0.662 | 0.475 | 0.841 | 0.393 |
| | Range | 1 - 6 | 1 - 5 | 1 - 5 | | | | | |
| Gravidity | 1 | 5 (12.5%) | 7 (23.3%) | 4 (13.3%) | 4.000 | 0.947 | 0.771 | 0.936 | 0.941 |
| | 2 | 13 (32.5%) | 8 (26.7%) | 8 (26.7%) | | | | | |
| | 3 | 13 (32.5%) | 8 (26.7%) | 10 (33.3%) | | | | | |
| | 4 | 5 (12.5%) | 5 (16.7%) | 5 (16.7%) | | | | | |
| | 5 | 3 (7.5%) | 2 (6.7%) | 3 (10.0%) | | | | | |
| | 6 | 1 (2.5%) | 0 (0.0%) | 0 (0.0%) | | | | | |
| Parity | Mean±SD | 1.25 ± 0.87 | 1.37 ± 1.00 | 1.43 ± 0.86 | 0.369 | 0.693 | 0.596 | 0.405 | 0.777 |
| | Range | 0 - 3 | 0 - 3 | 0 - 3 | | | | | |
| Parity | 0 | 9 (22.5%) | 7 (23.3%) | 4 (13.3%) | 2.845 | 0.828 | 0.658 | 0.684 | 0.697 |
| | 1 | 14 (35.0%) | 9 (30.0%) | 12 (40.0%) | | | | | |
| | 2 | 15 (37.5%) | 10 (33.3%) | 11 (36.7%) | | | | | |
| | 3 | 2 (5.0%) | 4 (13.3%) | 3 (10.0%) | | | | | |
| Gestational age | Mean±SD | 30.08 ± 4.53 | 35.80 ± 1.71 | 34.63 ± 2.50 | 29.781* | <0.001 | <0.001 | <0.001 | 0.176 |
| | Range | 22 - 38 | 33 - 39 | 29 - 38 | | | | | |
| BMI | Mean±SD | 27.82 ± 1.13 | 27.80 ± 0.97 | 29.36 ± 0.70 | 26.690* | <0.001 | 0.913 | <0.001 | <0.001 |
| | Range | 25.7 - 29.8 | 26.1 - 30.1 | 27.9 - 30.5 | | | | | |

*:One way ANOVA test; P > 0.05: Non significant; P < 0.05: Significant; P < 0.01: Highly significant

Table (2): Comparison between the three studied groups regarding blood pressure

| | | Normal | Mild | Severe | One Way ANOVA test | | Post Hoc analysis by LSD | | |
|--------------|---------|---------------|---------------|---------------|--------------------|---------|--------------------------|--------|--------|
| | | No.=40 | No.=30 | No.=30 | F | P-value | p1 | P2 | P3 |
| Systolic BP | Mean±SD | 116.00 ± 7.18 | 148.67 ± 3.92 | 178.33 ± 6.34 | 901.533 | <0.001 | <0.001 | <0.001 | <0.001 |
| | Range | 100 - 130 | 140 - 155 | 170 - 190 | | | | | |
| Diastolic BP | Mean±SD | 76.50 ± 6.91 | 102.17 ± 4.68 | 115.50 ± 4.02 | 454.552 | <0.001 | <0.001 | <0.001 | <0.001 |
| | Range | 70 - 90 | 90 - 110 | 110 - 120 | | | | | |

Table (3): Comparison between the three studied groups regarding albuminuria

| Albuminuria | Normal | | Mild | | Severe | | Chi-square test | |
|-------------|--------|-------|------|-------|--------|-------|-----------------|---------|
| | No. | % | No. | % | No. | % | X ² | P-value |
| Nil | 37 | 92.5% | 0 | 0.0% | 0 | 0.0% | 172.043 | <0.001 |
| Albumin+1 | 3 | 7.5% | 0 | 0.0% | 0 | 0.0% | | |
| Albumin+2 | 0 | 0.0% | 25 | 83.3% | 0 | 0.0% | | |
| Albumin+3 | 0 | 0.0% | 5 | 16.7% | 26 | 86.7% | | |
| Albumin+4 | 0 | 0.0% | 0 | 0.0% | 4 | 13.3% | | |

Table (4): Comparison between the three studied groups regarding lipid profile

| | | Normal | Mild | Severe | One Way ANOVA test | | Post Hoc analysis by LSD | | |
|-------------|---------|---------------|----------------|----------------|--------------------|---------|--------------------------|--------|--------|
| | | No.=40 | No.=30 | No.=30 | F | P-value | p1 | P2 | P3 |
| TGL | Mean±SD | 152.30 ± 9.22 | 195.33 ± 14.38 | 210.57 ± 14.09 | 209.321 | <0.001 | <0.001 | <0.001 | <0.001 |
| | Range | 134 - 173 | 168 - 221 | 188 - 245 | | | | | |
| Cholesterol | Mean±SD | 176.13 ± 8.09 | 217.00 ± 10.49 | 229.47 ± 12.61 | 260.306 | <0.001 | <0.001 | <0.001 | <0.001 |
| | Range | 156 - 188 | 198 - 240 | 209 - 256 | | | | | |
| HDL | Mean±SD | 43.28 ± 4.03 | 40.10 ± 5.39 | 36.33 ± 6.21 | 15.453 | <0.001 | 0.013 | <0.001 | 0.006 |
| | Range | 36 - 53 | 24 - 52 | 25 - 51 | | | | | |
| LDL | Mean±SD | 102.13 ± 9.78 | 138.93 ± 12.23 | 152.13 ± 11.03 | 200.149 | <0.001 | <0.001 | <0.001 | <0.001 |
| | Range | 69 - 115 | 117 - 172 | 131 - 180 | | | | | |
| VLDL | Mean±SD | 30.53 ± 1.91 | 39.60 ± 3.07 | 41.83 ± 2.87 | 190.610 | <0.001 | <0.001 | <0.001 | 0.001 |
| | Range | 27 - 35 | 34 - 48 | 37 - 49 | | | | | |

DISCUSSION

Hypertensive disorders during pregnancies, especially preeclampsia, are a pregnancy-specific disorder that affects 3-5% of pregnant women worldwide ⁽¹⁸⁾. The development of atherosclerosis in the placental spiral arteries of preeclamptic women indicates that elevated levels of triglycerides are involved in this disorder ⁽³⁾.

The principle modulator of this hypertriglyceridemia is estrogen as pregnancy is associated with hyperestrogenemia. Estrogen induces hepatic biosynthesis of endogenous triglycerides, which is carried by VLDL. This process may be modulated by hyperinsulinism found in pregnancy. Moreover, this hypertriglyceridemia maybe associated with hypercoagulability ⁽¹⁴⁾.

Hypertriglyceridemia could also be involved in the pathogenesis of hypertensive disorders during pregnancy ⁽¹⁷⁾. Women with elevated triglycerides had twice the risk of preeclampsia, studies that adjusted for confounders (age, BMI and parity) indicated that the risk was four times higher, compared with women with normal triglycerides ⁽¹⁸⁾.

It was also suggested that triglyceride assessment between 28 and 32 weeks could be predictive of preeclampsia ⁽²³⁾.

This current study is a case control study designed to evaluate blood serum levels triglycerides and cholesterol of 40 normotensive pregnant women compared to 30 mild and 30 severe preeclamptic women after 20 weeks of gestation.

Triglycerides

In current study, as shown in table (4): the serum level of TG in severe preeclamptic women was higher (210.57±14.09 mg/dl) compared to mild preeclamptic women (195.33 ±14.38 mg/dl) and compared to normotensive control group (152.30±9.22 mg/dl). The rise in serum TG was significantly higher (p< 0.01) in preeclamptic patients.

These findings are similar to some previous studies ^(1,16,17,21).

Cholesterol

In current study, as shown in table (4): the serum level of total cholesterol in severe preeclamptic women was lower (229.47±12.61 mg/dl) compared to mild preeclamptic women (217.00 ±10.49 mg/dl) and compared to normotensive control group (176.13±8.09 mg/dl). The serum total cholesterol was significantly lower (p< 0.01) in preeclamptic patients.

These results were in agreement with ^(1,16,21).

HDL Cholesterol

In current study, as shown in table (4): the serum level of HDL in severe preeclamptic women was higher (36.33±6.21 mg/dl) compared to mild preeclamptic women (40.10 ±5.39 mg/dl) and compared to normotensive control group (43.28±4.03 mg/dl). The rise in serum HDL was significantly higher (p< 0.01) in preeclamptic patients.

These results were in agreement with ^(13,21).

LDL Cholesterol

In current study, as shown in table (4): the serum level of LDL in severe preeclamptic women was higher (152.13±11.03mg/dl) compared to mild preeclamptic women (138.93 ±12.23mg/dl) compared to normotensive control group (102.13±9.78 mg/dl). The rise in serum LDL was significantly higher in severe preeclamptic patients than mild preeclamptic patients than normotensive control group.

VLDL Cholesterol

In current study, as shown in table (4): the serum level of VLDL in severe preeclamptic women was higher (41.83±2.87 mg/dl) compared to mild preeclamptic women (39.60 ±3.07mg/dl) and compared to normotensive control group (30.53±1.91 mg/dl). The rise in serum VLDL was significantly higher in severe preeclamptic patients than mild preeclamptic patients than normotensive control group.

CONCLUSION

In conclusion, the findings of our study suggest that preeclampsia has an association with hypertriglyceridemia and elevated cholesterol level LDL and VLDL and decreased HDL level. The more the severity of preeclampsia the higher is the level of serum triglycerides cholesterol LDL and VLDL and the lower is the HDL. These observations support a role for hypercholesterolemia and hypertriglyceridemia in the development of preeclampsia. Abnormal levels of TGs and hypercholesterolemia 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 monitored throughout the pregnancy period as it would be helpful in the early detection of obstetric-associated complication (pregnancy-induced hypertension; PIH).

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