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Original Article

Biochemistry Section

Estimation of Lipid Profile, Hepatic Enzymes, Malondialdehyde, and Uric Acid in Preeclampsia: Implications for Early Intervention

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ABSTRACT

Introduction: Preeclampsia, a serious pregnancy complication, poses significant risks to both maternal and foetal health, potentially leading to morbidity and mortality. This condition is characterised by changes in lipid profiles, hepatic enzymes, Malondialdehyde (MDA), and uric acid levels. Despite significant medical advancements, identifying precise biomarkers for preeclampsia remains complex. Moreover, there is a lack of epidemiological research on preeclampsia within the southern Indian population.

Aim: To estimate the levels of serum lipid profiles, hepatic enzyme levels, MDA, and uric acid levels in pregnant women with preeclampsia. Also, to examine the association between MDA and uric acid levels among women with preeclampsia and those with normal pregnancies.

Materials and Methods: This cross-sectional study included 162 pregnant patients aged between 18 and 35 years, who attended the Outpatient Department (OPD) or were admitted to a tertiary care hospital in Visakhapatnam, Andhra Pradesh, India between February 2021 and October 2021. The participants were divided into three groups: Group A (54 normotensive pregnant women), Group B (54 pregnant women with non severe preeclampsia), and Group C (54 pregnant women with severe preeclampsia). Lipid profiles, hepatic enzymes, MDA, and uric acid were evaluated in all subjects, and their relationship with preeclampsia severity was assessed. The data were statistically analysed using one-way Analysis of Variance (ANOVA) followed by the Tukey post-hoc test.

Results: The study groups (A, B, and C) had comparable age and gestational periods. However, significant variations were observed in lipid profiles, hepatic enzymes, MDA, and uric acid levels among them, which associated with the severity of preeclampsia. Increasing severity was associated with higher cholesterol and triglyceride levels, as well as a decrease in High Density Lipoprotein (HDL) cholesterol. Furthermore, disease progression led to significant elevations in Alkaline Phosphatase (ALP) , uric acid, and MDA levels. In particular, Group A displayed total cholesterol levels of 138.3±20.32 mg/dL, triglycerides of 109.98±15.22 mg/dL, and a negative association with HDL-cholesterol at 30.57±3.65 mg/dL. In contrast, Group C exhibited considerably higher levels of total cholesterol, triglycerides, AST, ALT, uric acid, and MDA compared to Group A.

Conclusion: As preeclampsia worsens, cholesterol and triglyceride levels increase, while HDL-cholesterol decreases, indicating a deteriorating metabolic profile. Additionally, ALP, uric acid, and MDA levels rise, indicating increased oxidative stress and liver function impact with the progression of the ailment.

Keywords: Blood pressure, Hypertension, Oxidative stress, Proteinuria

INTRODUCTION

Preeclampsia is a serious medical complication of pregnancy that affects 7-10% of women in India [1]. It is characterised by hypertension, with systolic blood pressure ≥140 mmHg and diastolic blood pressure ≥90 mmHg, along with proteinuria (≥300 mg/day) after 20 weeks of pregnancy in a previously normotensive and non proteinuric woman [2]. Despite extensive research, the causes and pathogenesis of preeclampsia remains unclear. However, endothelial dysfunction and abnormal placentation are considered critical factors in its development [2]. Multiple factors can provoke endothelial changes, and it is believed that abnormal lipid profiles are not merely a manifestation of preeclampsia but directly affect endothelial dysfunction [3]. Lipid-mediated oxidative stress could be a cause of endothelial dysfunction, and MDA, a product of lipid peroxidation, is used as a marker of oxidative stress. Studies have shown increased MDA levels in preeclampsia [4-8]. Abnormalities in hepatic enzymes such as Aspartate Transaminase (AST), Alanine Transaminase (ALT), Gamma-glutamyltransferase (GGT), and ALP during pregnancy are rare, but they occur in about 20-30% of pregnancies and may be an initial sign of pathological conditions [9]. Liver involvement in preeclampsia is an indicator to prevent complications like eclampsia, hepatic rupture, and necrosis. Liver enzymes have a significant prognostic role in predicting preeclampsia [10]. The pathophysiological mechanisms of liver involvement in preeclampsia include:

1) **Impaired hepatic blood flow:** Preeclampsia can lead to vasoconstriction of blood vessels, including those supplying the liver. This can result in reduced blood flow to the liver, known as hepatic hypoperfusion, leading to liver damage and impaired liver function.
2) **Endothelial dysfunction:** Preeclampsia is characterised by endothelial dysfunction, which affects blood vessel integrity and function. The liver's sinusoidal endothelial cells may be affected, leading to the release of liver enzymes into the bloodstream.
3) **Oxidative stress and inflammation:** Preeclampsia is associated with increased oxidative stress and inflammation throughout the body, including the liver. These processes can cause hepatocellular injury and promote the release of liver enzymes.
4) **Placental factors:** Abnormalities in placental development and function can lead to the release of factors into the maternal circulation, triggering liver dysfunction. These factors may include

antiangiogenic proteins, such as soluble FMS-like tyrosine kinase 1 (sFlt-1), which is elevated in preeclampsia and contributes to liver damage. The elevation of liver enzymes, such as AST and ALT, in preeclampsia indicates liver injury and dysfunction. Higher levels of liver enzymes may be associated with more severe liver involvement and poorer maternal and foetal outcomes [10,11].

Uric acid, an end product of purine metabolism, serves as a marker of oxidative stress. Increased uric acid production can be attributed to reduced renal excretion, tissue ischaemia, oxidative stress, and heightened activity of xanthine oxidase. Hyperuricaemia leads to impaired nitric oxide generation, causing endothelial dysfunction and contributing to hypertension, vascular disease, and renal disease. Elevated uric acid levels are commonly observed in preeclamptic women [12]. Only a limited number of studies have explored the potential of lipid profiles, hepatic enzyme levels, MDA, and uric acid levels as early predictors [5-8,12]. Moreover, none of these studies have comprehensively investigated all of these parameters within a single study conducted specifically on the population of Southern India. Consequently, robust correlations among the designated groups have not been established.

Therefore, this present study was meticulously conducted to examine the biochemical markers associated with lipid profiles, hepatic enzymes, MDA, and uric acid in individuals with preeclampsia. Furthermore, a comparison of these parameters between non severe and severe preeclampsia cases, as well as normotensive pregnant women, was performed. Additionally, the study aimed to analyse and compare oxidative stress parameters (MDA and uric acid) between women with preeclampsia and those experiencing a normative pregnancy. By promptly assessing and evaluating these parameters during the early stages of pregnancy, it is plausible to facilitate early intervention and impede the progression of preeclampsia.

MATERIALS AND METHODS

A cross-sectional study was conducted at a tertiary care hospital in Visakhapatnam, Andhra Pradesh, India, from February 2021 to October 2021. The study received ethical clearance from the Institutional Ethics Committee (IEC) of Andhra Medical College (IEC approval no. 103/IEC AMC/MAR 2021). Informed consent was obtained from all the participants.

Inclusion criteria: The study included pregnant women aged between 18 and 35 years who were either attending OPD or admitted to the Department of Obstetrics. They had a gestational age of ≥20 weeks, whether primi- or multigravida, with singleton pregnancies. Normotensive pregnant women and pregnant women with hypertension (blood pressure ≥140/90 mmHg and ≥160/100 mmHg in non severe and severe preeclamptic women, respectively) were eligible for inclusion [13]. Proteinuria was detected through a urine dipstick test, which indicated albumin levels of "+" or higher based on disease severity [2].

Exclusion criteria: Patients with known cardiac disease, renal disease, diabetes, dyslipidaemia, alcoholism, liver disease, gout, Rh negative blood group (multigravida), pre-existing hypertension, and multiple pregnancies were excluded from the study.

Sample size: The following formula was used for sample size calculation:

$$n=Z_{1-\alpha/2}^{2}\ P(1-P)/d^{2}$$

Where n is the sample size, $Z_{1-\alpha/2}{}^{/2}$ is the standard normal variate, 'P' is the expected prevalence, and 'd' is the absolute error or precision [14,15]. The sample size obtained was 162 based on prevalence, ensuring comprehensive data for analysis.

A total of 162 patients (54 in each group) were categorised into three groups: Group A consisted of 54 normotensive pregnant women, Group B included 54 pregnant women with non severe preeclampsia, and Group C comprised 54 pregnant women with severe preeclampsia.

Data collection: Urine analysis was conducted as part of routine screening to exclude urinary tract infections and proteinuria.

For biochemical analysis, fasting samples were collected after an 8-12 hour fasting period. Using strict aseptic techniques, 5 mL of venous blood was drawn and analysed using the Beckman Coulter AU 480 instrument to measure various parameters. These included serum total cholesterol, serum triglycerides, serum HDL-cholesterol, calculated serum Low Density Lipoprotein (LDL)-cholesterol, calculated serum VLDL, Serum ALT/SGPT, Serum AST/SGOT, Serum ALP, Serum Uric Acid, and Serum MDA (measured using the Thiobarbituric acid assay method of Buege JA and Aust SD [16]. The cut-off range and method of estimation for all the parameters were considered as provided in [Table/Fig-1].

STATISTICAL ANALYSIS

The results of the statistical analysis, using one-way ANOVA followed by the Tukey post-hoc test, revealed significant differences in the serum lipid profile, liver enzymes, and oxidative stress markers among the three study groups. The obtained p-values, which were less than 0.01, indicate that these differences were highly significant. The aforementioned analyses were performed using IBM Statistical Package for the Social Sciences (SPSS) Statistics for Windows, Version 25.0, released in 2017, developed by IBM Corp. The software package was used to conduct the statistical examinations.

RESULTS

In this current cross-sectional study, out of 162 participants, Group B (n=54) had a mean age of 24.48±3.37 years, while Group C (n=54) had a mean age of 25.13±2.79 years. The gestation period for Group B was 32.48±2.42 weeks, and for Group C, it was 35.96±1.64 weeks (p-value <0.001). Group C patients had higher systolic and diastolic blood pressure compared to Groups A and B [Table/Fig-2].

Group C patients showed notable increases in total cholesterol, triglycerides, LDL-cholesterol, and VLDL-cholesterol levels, along with elevated AST, ALT, ALP, uric acid, and MDA compared to Group B and A patients. Statistically significant differences were observed when compared to Group A [Table/Fig-3].

Parameters	Name of the method for estimation	Cut-off range	
Serum total cholesterol (mg/dL)	Cholesterol oxidase- phenol 4-aminoantipyrine peroxidase method	**Total cholesterol**	**Risk classification**
		<200	Desirable
		200-239	Borderline high
		>240	High
Serum triglycerides (mg/dL)	Glycerol phosphate oxidase- peroxidase method	**Triglycerides**	**Risk classification**
		<150	Normal
		150-199	Borderline high
		200-499	High
		≥500	Very high
Serum HDL-Cholesterol (mg/dL)	Direct enzymatic method	35-75	

Calculated serum LDL-Cholesterol (mg/dL)	Friedwald formula	<100	Good control
		130-159	Moderate control
		>160	High
Serum Alanine Transaminase (ALT/SGPT) (U/L)	UV kinetic method	7-52 U/L	
Serum Aspartate Transaminase (AST/SGOT) (U/L)	UV kinetic method	13-39 U/L	
Serum Alkaline Phosphatase (ALP) (U/L)	pNPP AMP kinetic method	34-104 U/L	
Serum uric acid (mg/dL)	Uricase method	2.3 to 6.6 mg/dL	
Serum Malondialdehyde (MDA) (nmol/mL)	Thiobarbituric acid assay method	2.6 to 3.8 nmol/mL	

[Table/Fig-1]: Parameters method name for the estimation and cut-off range.

Parameters	Group A: Normotensive pregnant (Mean±SD)	Group B: Non severe preeclampsia (Mean±SD)	Group C: Severe preeclampsia (Mean±SD)	p-value
Mean age (years)	23.52±2.76	24.48±3.37	25.13±2.79	0.02
Mean gestational age (weeks)	31.15±2.41	32.48±2.42	35.96±1.64	<0.001
Systolic BP (mmHg)	110±8.69	144.63±5.4	167.59±11.32	<0.001
Diastolic BP (mmHg)	73.89±5.96	92.41±5.12	112.78±4.52	<0.001

[Table/Fig-2]: Comparison of systolic and diastolic blood pressure between the study groups.
p-value <0.05 significant, p-value <0.001 highly significant, One-way ANOVA was used to compare among the groups; followed by Turkey's post-hoc test

Parameters	Group A: Normotensive pregnant (Mean±SD)	Group B: Non severe Preeclampsia (Mean±SD)	Group C: Severe Preeclampsia (Mean±SD)	p-value
Total cholesterol (mg/dL)	138.3±20.32	172.06±11.80	205.44±11.18	<0.001
Triglycerides (mg/dL)	109.98±15.22	148.83±13.13	208.63±20.52	<0.001
HDL-Cholesterol (mg/dL)	43.22±6.67	35.39±2.9	30.57±3.65	<0.001
LDL-Cholesterol (mg/dL)	73.02±14.21	106.74±11.81	133.06±12.46	<0.001
VLDL-Cholesterol (mg/dL)	22.06±2.99	29.81±2.71	41.72±4.08	<0.001
Aspartate Transaminase (AST) (U/L)	16.72±6.27	19±6.16	23.02±13.06	0.002
Alanine Transaminase (ALT) (U/L)	20.18±6.93	22.7±4.97	25.05±12.98	0.02
Alkaline phosphatase (U/L)	22.89±7.83	178.63±52.35	320.07±96.71	<0.001
Uric acid (mg/dL)	4.04±0.74	6.24±0.65	7.67±1.13	<0.001
Malondialdehyde (nmol/mL)	2.52±0.56	4.21±0.60	5.37±0.57	<0.001

[Table/Fig-3]: Comparison of serum lipid profile, liver enzymes, uric acid and MDA between the study groups.
p-value <0.05 significant, p-value <0.001 highly significant, One-way ANOVA was used to compare among the groups; followed by Turkey's post-hoc test

The study found a strong significant positive relationship between serum uric acid and MDA levels in all three groups [Table/Fig-4], indicating oxidative stress.

	MDA					
	Group A: Normotensive pregnant		Group B: Non severe preeclampsia		Group C: Severe preeclampsia	
Variable	r	p-value	r	p-value	r	p-value
Uric acid (mg/dL)	0.61	<0.001	0.77	<0.001	0.84	<0.001

[Table/Fig-4]: Correlation coefficient between serum uric acid and MDA levels between the study groups.
p-value <0.001 highly significant, One-way ANOVA was used to compare among the groups; followed by Turkey's post-hoc test

DISCUSSION

The present study assessed the serum lipid profile, AST, ALT, ALP, serum uric acid, and MDA levels in women already diagnosed with preeclampsia and healthy pregnant women. The results were compared among the three study groups. Preeclampsia, a common complication specific to pregnancy, has an unclear exact pathophysiology [9]. It is believed that endothelial dysfunction and abnormal placentation may be possible causes. Oxidative stress resulting from lipid peroxidation has been associated with endothelial dysfunction [17-19]. Hypertensive disorders during pregnancy, particularly preeclampsia, affect around 3-5% of women worldwide [20]. The presence of atherosclerosis in the placental spiral arteries of preeclamptic women suggests a connection with elevated triglyceride levels. High oestrogen levels during pregnancy lead to hypertriglyceridaemia by stimulating liver triglyceride production transported by VLDL. Hyperinsulinism, which is common in pregnancy, may regulate this process and is also linked to hypercoagulability [21].

Triglyceride levels between the 28th and 32nd weeks of pregnancy have been found to be predictive of preeclampsia [22]. Age did not show a significant association with preeclampsia severity in present study, which aligns with previous research [18,19,21-25]. However, Hazari NR et al., reported a significant decrease in age associated with preeclampsia severity [20]. Blood pressure, both systolic and diastolic, increased with the progression of preeclampsia, consistent with findings by Ahmed AAM et al., and Hazari NR et al., [18,20].

Cholesterol and triglyceride levels are elevated in preeclampsia, and the degree of increase is linked to the severity of the condition. Some studies reported similar results, showing an increase in lipid parameters with increasing preeclampsia severity [23,26], while others found no significant increase [27]. In a previous study, HDL-cholesterol showed a negative correlation with preeclampsia severity, whereas LDL cholesterol and VLDL cholesterol showed a positive correlation [18]. Additionally, severe preeclamptic women had notably higher serum HDL cholesterol levels compared to mild preeclamptic women and normotensive controls [18]. This suggests a potential role for HDL cholesterol in the development and progression of preeclampsia, given its function in removing excess cholesterol from the bloodstream and preventing arterial accumulation.

Severe preeclamptic women showed higher serum LDL cholesterol levels compared to mild preeclamptic women and normotensive controls [18]. Elevated LDL cholesterol is linked to an increased risk of cardiovascular disease due to arterial plaque formation

[18]. Similarly, severe preeclamptic patients had higher serum VLDL cholesterol levels compared to mild preeclamptic patients and normotensive controls [18]. Dysregulated lipid metabolism and altered VLDL cholesterol levels may contribute to endothelial dysfunction and inflammation in preeclampsia.

Present study highlights the complex interplay between cholesterol fractions and the development of preeclampsia, necessitating further research to understand the underlying mechanisms and clinical implications of these lipid abnormalities. In contrast, Mittal M et al., found no significant changes in TC, HDL-C, and LDL-C levels in normal and preeclamptic/eclamptic pregnancies but observed significantly elevated serum triglyceride levels in preeclamptic and eclamptic women compared to normal pregnant women [28]. Specifically, the mild preeclampsia group had a mean serum triglyceride level of 156.22±66.5 mg/dL, which increased to 168.30±68.1 mg/dL in severe preeclampsia and reached 224.89±84.40 mg/dL in eclampsia. In contrast, normal pregnant women had a lower mean serum triglyceride level of 130.95±44.64 mg/dL, indicating a clear association between elevated triglyceride levels and the presence of preeclampsia and eclampsia [28].

In present study, AST, ALT, and ALP increased with the severity of preeclampsia, consistent with findings by Hazari NR et al., Patil S and Das S et al., [20,29,30]. Abnormal levels of hepatic enzymes were prevalent in preeclampsia, with notable proportions: AST (40%), ALT (45%), GGT (87.5%), LDH (90%), and ALP (55%). These results indicate possible liver dysfunction or injury associated with the condition, highlighting the widespread occurrence of hepatic involvement in preeclamptic pregnancies [20,29,30]. Understanding the impact of preeclampsia on liver enzymes is crucial for improved monitoring and management strategies. However, the study by Kasraeian M et al., reported contradictory results [21]. Mean serum uric acid levels increased with preeclampsia severity, suggesting its potential as an early biomarker [20,29,31]. Banu F et al., reported a significant distinction in serum uric acid levels between mild and severe preeclampsia groups (p-value=0.001) [31].

Similarly, mean serum MDA levels increased progressively with preeclampsia severity, as observed in other studies [32,33]. Priyamvada RP et al., found significantly elevated MDA levels in preeclamptic patients compared to normal and non pregnant individuals, with the highest increase in severe preeclampsia [32]. Oxidative stress markers such as uric acid and MDA increased significantly as the disease advanced. Elevated uric acid levels in preeclampsia are not merely a sign of kidney damage but suggest an antioxidative response. Increased oxidative stress and ROS generation contribute to hyperuricaemia independent of renal dysfunction. Excessive lipid peroxidation from uric acid affects placental tissue and reduces nitric oxide release. Renal endothelial cell injury decreases renal urate clearance, and foetal tissue under hypoxia triggers xanthine oxidase activity, increasing uric acid release. The significant association (p-value <0.001) in present study highlights the role of oxidative stress in the development of preeclampsia. Elevated lipid levels in the study may lead to oxidative stress, emphasising the need to manage oxidative stress and regulate lipid levels during pregnancy to prevent preeclampsia [32]. These findings indicate inadequate compensatory mechanisms to counteract oxidative stress in preeclampsia, resulting in lipid membrane damage. The study highlights the relationship between the extent of lipid damage and preeclampsia severity, providing valuable insights into its pathophysiology and potential diagnostic and management strategies.

Limitation(s)

1. The cross-sectional design of this study limits the ability to determine causality; longitudinal studies are needed to provide more comprehensive insights into the relationship between the variables.
2. Conducting research in a single hospital may introduce biases; conducting multicentre studies would enhance the generalisability and relevance of the findings.
3. Focusing solely on specific markers limits the assessment of preeclampsia; including other markers, such as inflammatory markers, would provide a more comprehensive understanding of the condition.

CONCLUSION(S)

A significant and remarkable increase was noted in several physiological indicators, including systolic and diastolic blood pressure, total cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol, VLDL-cholesterol, alkaline phosphatase, uric acid, and MDA levels, when examined in relation to the severity of preeclampsia. These findings strongly imply that alterations in the lipid profile could potentially contribute to the underlying mechanisms of preeclampsia's development. By promptly assessing and evaluating these parameters during the early stages of pregnancy, it becomes possible to initiate timely interventions and mitigate the progression of preeclampsia. Such early interventions play a crucial role in delaying the advancement of this condition and ensuring the well-being of both the mother and the developing foetus.

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