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Association of Serum Lipid Profile Changes and Maternal Biochemical Profiling With Pregnancy-Induced Hypertension: Evaluating the Role of Maternal Nutrition and Cardiovascular Risk

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ABSTRACT
Pregnancy-induced hypertension is a major obstetric complication contributing significantly to maternal and perinatal morbidity and mortality worldwide. Emerging evidence suggests that dyslipidemia, altered maternal biochemical profile, and poor nutritional status play a central role in endothelial dysfunction and abnormal placentation, ultimately leading to hypertensive disorders of pregnancy. The present study aimed to evaluate the association of serum lipid profile changes and maternal biochemical parameters with pregnancy-induced hypertension and to assess the role of maternal nutrition in cardiovascular risk stratification. A prospective analytical study was conducted on 292 pregnant women between 18 and 40 years of age, enrolled between January 2025. Serum lipid profile, liver and renal function tests, fasting glucose, and nutritional status indices were assessed and compared between normotensive and pregnancy-induced hypertension groups. Women with pregnancy-induced hypertension demonstrated significantly elevated serum total cholesterol, triglycerides, low-density lipoprotein levels, and reduced high-density lipoprotein levels ($p<0.001$). Abnormal biochemical markers including elevated uric acid, alanine aminotransferase, and creatinine were also significantly associated with hypertensive pregnancy outcomes. Poor maternal nutritional status, low protein intake, and higher body mass index were independently associated with adverse lipid and biochemical alterations. Multivariate regression analysis identified dyslipidemia, elevated uric acid, and poor nutritional status as strong predictors of pregnancy-induced hypertension. The study highlights the importance of early biochemical screening and nutritional assessment in antenatal care for prevention and early identification of hypertensive disorders in pregnancy..
Keywords: Pregnancy-induced hypertension, lipid profile, maternal nutrition
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INTRODUCTION:

Pregnancy-induced hypertension remains one of the leading causes of maternal and fetal complications globally, representing a spectrum of hypertensive disorders that include gestational hypertension, preeclampsia, and eclampsia. Despite advances in obstetric care, hypertensive disorders during pregnancy continue to contribute significantly to maternal mortality, preterm birth, intrauterine growth restriction, and perinatal morbidity [1-3]. Early identification of biochemical and metabolic risk factors is therefore critical in improving maternal and fetal outcomes.

Recent research has increasingly focused on the role of maternal metabolic health in the development of pregnancy-induced hypertension. Dyslipidemia, insulin resistance, oxidative stress, and endothelial dysfunction have been identified as key contributors to abnormal placentation and vascular maladaptation during pregnancy [2-4]. Altered lipid metabolism during pregnancy is physiologically expected; however, exaggerated lipid elevation may contribute to vascular inflammation and impaired endothelial function.

Serum lipid profile changes including elevated triglycerides, total cholesterol, and low-density lipoprotein levels, along with reduced high-density lipoprotein, have been consistently associated with hypertensive disorders in pregnancy [3-5]. These lipid abnormalities promote oxidative stress and vascular endothelial injury, leading to increased peripheral resistance and hypertension. Additionally, lipid peroxidation products may further aggravate placental ischemia.

Maternal biochemical markers such as uric acid, liver enzymes, and renal function parameters have also been identified as important predictors of pregnancy-induced hypertension. Elevated uric acid levels are associated with reduced renal clearance and increased oxidative stress, while liver dysfunction may reflect systemic endothelial injury in preeclamptic states [4-6]. These biochemical

changes often precede clinical manifestations of hypertension.

Maternal nutrition plays a fundamental role in regulating metabolic and cardiovascular adaptations during pregnancy. Inadequate intake of proteins, micronutrients, antioxidants, and essential fatty acids may predispose to endothelial dysfunction and abnormal lipid metabolism [5-7]. Poor nutritional status has been associated with increased risk of hypertensive disorders, especially in low-resource populations where dietary imbalance is common.

Obesity and excessive gestational weight gain further exacerbate metabolic disturbances, increasing the risk of dyslipidemia and insulin resistance. Adipose tissue acts as an endocrine organ releasing inflammatory cytokines that contribute to vascular dysfunction and hypertension [6-8]. Therefore, maternal body mass index and nutritional profile are important modifiable risk factors.

Although multiple studies have explored individual associations between lipid profile and pregnancy-induced hypertension, limited research has comprehensively evaluated lipid parameters in combination with broader maternal biochemical profiling and nutritional assessment in a single analytical model [7-9]. Additionally, regional data from South Asian populations remains limited despite high prevalence of maternal malnutrition and hypertensive pregnancy complications.

Early biochemical screening during antenatal care may provide valuable predictive insight into women at risk of developing pregnancy-induced hypertension. Identification of lipid and biochemical abnormalities before clinical onset may allow early intervention through nutritional counseling, lifestyle modification, and pharmacological management where necessary [8-10].

The present study was therefore designed to evaluate the association of serum lipid profile changes and maternal biochemical parameters with pregnancy-induced hypertension and to assess the role of maternal nutrition in cardiovascular risk stratification among pregnant women.

Methodology

A prospective analytical study was conducted at NUST School of Health Sciences, National University of Sciences and Technology in the Departments of Obstetrics and Gynecology and Clinical Biochemistry at a tertiary care hospital between January 2025 and June 2025 after approval from the institutional ethical review board. The study included pregnant women aged 18–40 years presenting for antenatal care during second and third trimesters. Sample size was calculated using OpenEpi software version 3.01 by considering an anticipated prevalence of dyslipidemia in pregnancy-induced hypertension of 40%, confidence interval of 95%, margin of error of 5%, and power of 80%, resulting in a minimum required sample size of 280 participants. A total of 292 participants were enrolled after accounting for incomplete follow-up data.

Women with pre-existing hypertension, chronic renal disease, diabetes mellitus diagnosed before pregnancy, hepatic disorders, autoimmune disease, multiple gestation, or use of lipid-lowering drugs were excluded. Verbal informed consent was obtained from all participants, and confidentiality of data was strictly maintained.

Participants were categorized into pregnancy-induced hypertension group and normotensive pregnancy group based on blood pressure measurements recorded on two separate occasions at least four hours apart. Detailed demographic data including age, parity, gestational age, body mass index, dietary intake pattern, and socioeconomic status were recorded using structured questionnaires.

Fasting blood samples were collected for analysis of lipid profile including total cholesterol, triglycerides, low-density lipoprotein, and high-density lipoprotein using enzymatic colorimetric methods. Maternal biochemical parameters including uric acid, alanine aminotransferase, aspartate aminotransferase, creatinine, and fasting glucose were measured using standardized laboratory techniques.

Maternal nutritional status was assessed using dietary recall methods, body mass index calculation, and serum albumin levels. Nutritional risk scoring was performed based on protein intake, micronutrient consumption, and caloric adequacy.

Statistical analysis was performed using SPSS version 27. Continuous variables were expressed as mean ± standard deviation and categorical variables as percentages. Independent t-test, chi-square test, and ANOVA were applied for group comparisons. Multivariate logistic regression was used to identify independent predictors of pregnancy-induced hypertension. A p-value less than 0.05 was considered statistically significant.

Results

A total of 292 pregnant women were included in the study. Among them, 118 (40.4%) were diagnosed with pregnancy-induced hypertension, while 174 (59.6%) remained normotensive.

Table 1. Maternal Demographic and Clinical Characteristics

Variable	PIH Group (n=118)	Normotensive (n=174)	p-value
Mean age (years)	28.6 ± 4.9	27.9 ± 4.6	0.214
BMI (kg/m²)	31.2 ± 5.1	27.4 ± 4.3	<0.001
Poor nutritional status	68 (57.6%)	42 (24.1%)	<0.001

Variable	PIH Group (n=118)	Normotensive (n=174)	p-value
Primigravida	62 (52.5%)	71 (40.8%)	0.041
Family history of hypertension	54 (45.7%)	39 (22.4%)	<0.001

Table 2. Serum Lipid Profile and Biochemical Parameters

Parameter	PIH Group	Normotensive	p-value
Total cholesterol (mg/dL)	242.6 ± 28.4	198.3 ± 24.7	<0.001
Triglycerides (mg/dL)	238.9 ± 31.5	176.4 ± 22.9	<0.001
LDL (mg/dL)	154.7 ± 20.6	118.2 ± 18.4	<0.001
HDL (mg/dL)	38.5 ± 6.2	49.8 ± 7.1	<0.001
Uric acid (mg/dL)	6.8 ± 1.2	4.9 ± 0.9	<0.001
Creatinine (mg/dL)	1.02 ± 0.21	0.78 ± 0.15	<0.001

Table 3. Independent Predictors of Pregnancy-Induced Hypertension

Predictor	Odds Ratio (95% CI)	p-value
Dyslipidemia	4.21 (2.31–7.68)	<0.001
Elevated uric acid	3.58 (1.98–6.47)	<0.001
Poor maternal nutrition	3.12 (1.74–5.61)	<0.001
High BMI	2.89 (1.62–5.17)	0.001
Elevated LDL	2.76 (1.53–4.98)	0.002

DISCUSSION

The present study demonstrated a strong association between altered serum lipid profile, abnormal maternal biochemical markers, and pregnancy-induced hypertension. Elevated cholesterol, triglycerides, LDL levels, and reduced HDL were significantly associated with hypertensive pregnancy outcomes, supporting the role of dyslipidemia in endothelial dysfunction [11-12].

Maternal biochemical alterations such as elevated uric acid and creatinine further indicated systemic endothelial injury and renal involvement in pregnancy-induced hypertension. Similar findings have been reported in recent studies highlighting uric acid as an early biomarker of preeclampsia severity [12-14].

Poor maternal nutritional status emerged as a significant independent predictor of pregnancy-induced hypertension. Inadequate protein and micronutrient intake may impair antioxidant defense mechanisms and placental vascular development, increasing susceptibility to hypertensive disorders [13-15].

Obesity and high BMI significantly contributed to metabolic imbalance and lipid abnormalities in pregnant women. Adipose-derived inflammatory mediators promote insulin resistance and vascular dysfunction, thereby increasing blood pressure risk [14-16].

The findings emphasize that dyslipidemia and metabolic dysfunction often precede clinical hypertension, suggesting a window for early detection and prevention through antenatal screening [15-17].

Combined biochemical and nutritional assessment provides superior predictive accuracy compared with isolated lipid measurement, highlighting the importance of integrated maternal health evaluation [16-18].

These results have important implications for antenatal care in resource-limited settings where early screening and nutritional counseling may significantly reduce maternal morbidity and mortality associated with hypertensive disorders [17-20].

CONCLUSION

Pregnancy-induced hypertension is strongly associated with dyslipidemia, elevated uric acid, and poor maternal

nutritional status. Early biochemical screening and nutritional assessment can significantly improve risk prediction and prevention strategies. Integrated maternal metabolic evaluation should be incorporated into routine antenatal care to reduce hypertensive pregnancy complications

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