Am J Transl Res 2025;17(4):2800-2808
www.ajtr.org /ISSN:1943-8141/AJTR0160527

Original Article

Correlation of uric acid and lipid levels with preeclampsia and final pregnancy outcome in late pregnancy

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Received September 13, 2024; Accepted November 29, 2024; Epub April 15, 2025; Published April 30, 2025

Abstract: Objective: To investigate the correlation between uric acid (UA), lipid levels, and preeclampsia (PE), as well as their effect on pregnancy outcome in women in late pregnancy. Methods: A retrospective analysis was conducted on the clinical data from 126 pregnant women with PE who were admitted to the First Affiliated Hospital of Xi'an Medical University from June 2021 to January 2024 (research group). Additionally, clinical data from 130 healthy pregnant women who gave birth during the same period were served as controls. General information, UA levels, blood lipid levels [total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), free fatty acids (FFA), lipoprotein-a (Lp-a), apolipoprotein-a1 (ApoA1), apolipoprotein B (ApoB), LDL-C/HDL-C, and ApoA1/ApoB] and pregnancy outcomes were compared between the two groups. A logistic regression model was used to identify the influencing factors for PE. The predictive value of UA and lipid levels for PE diagnosis and prognosis was evaluated using receiver operating characteristic (ROC) curve analysis. Results: No significant differences were observed between the groups in terms of age, parity, mode of delivery, neonatal gender, gestational cardiac disease, HDL-C, FFA, ApoA1, or ApoA1/ApoB (all $P>0.05$). However, the research group exhibited significantly higher body mass index (BMI), prevalence of gestational diabetes, and gestational hypertension, UA, TC, TG, LDL-C, Lp-a, ApoB, and LDL-C/HDL-C ratio compared to the control group, but lower neonatal weight (all $P<0.05$). Furthermore, the research group had a higher incidence of gestational diabetes, gestational hypertension, postpartum hemorrhage, fetal growth retardation, preterm delivery, and neonatal asphyxia (all $P<0.05$). Multivariate logistic regression analysis identified BMI, neonatal weight, UA, TC, TG, and LDL-C as independent influencing factors for PE. ROC curve analysis demonstrated high diagnostic accuracy for BMI (AUC=0.835), neonatal weight (AUC=0.755), UA (AUC=0.765), TC (AUC=0.706), and LDL-C (AUC=0.792) in predicting PE. Conclusion: Maternal BMI, neonatal weight, serum UA, TC, TG, and LDL-C levels are risk factors for the development of PE. Among these, BMI, neonatal weight, serum UA, TC, and LDL-C levels have a high predictive value for PE and can serve as valuable indicators for its early prediction and management.

Keywords: Blood lipid, uric acid, preeclampsia, final pregnancy outcome, advanced stage of pregnancy

Introduction

Preeclampsia (PE) is a severe hypertensive disorder unique to pregnancy, representing one of the most serious complications of gestation. The global incidence of PE is about 2% to 8%, reaching as high as 9.4% in China, with a rising trend in recent years [1-4]. PE typically manifests after 20 weeks of gestation, featuring hypertension and edema, often accompanied by proteinuria. PE may emerge suddenly and deteriorate rapidly, leading to functional impairment of other organs with a threat to the health of the pregnant woman and the fetus [5]. Therefore, understanding the pathogenesis and developing effective treatments for PE remain a focus of obstetrics research.

Although the exact etiology and mechanisms of PE remain unclear, endothelial dysfunction is widely accepted as a primary contributor. Abnormal lipid metabolism can impair endothelial function, a key factor in the pathogenesis of preeclampsia. This dysfunction may lead to placental ischemia, triggering oxidative stress and an inflammatory response, which further exac-

https://doi.org/10.62347/HBLW4532

erbates maternal dyslipidemia [6, 7]. Serum triglyceride (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) levels are elevated in patients with PE [8, 9]. Serum free fatty acids may play an important role in the pathogenesis of PE by enhancing mitochondrial oxidative stress injury, although relevant studies are limited [10]. Lipoprotein-a (Lp-a) is also involved in the pathogenesis of PE, with levels correlating with disease severity [11]. The kidneys are often the first organ affected in PE. Uric acid (UA), the end product of purine metabolism, is primarily excreted by the kidneys. Impaired renal function in PE can hinder UA elimination, leading to its accumulation and elevated serum levels [12, 13]. Study has shown that elevated UA levels may precede the onset of hypertension or proteinuria during pregnancy [14]. Hypertension-induced renal injury decreases the glomerular filtration rate, increases UA reabsorption, and decreases secretion, further raising serum UA levels [15]. Clinically, serum UA levels are used to monitor the severity of PE and to predict complications [16, 17].

Disorders of lipid metabolism and UA levels have been linked to the development of PE, as well as adverse maternal and neonatal outcomes. This study aimed to identify risk factors associated with PE and retrospectively investigate the correlation between PE progression and maternal serum UA and lipid metabolism levels. By comparing these factors in pregnant women with PE and healthy controls, the study also evaluated the effect of these measurements on maternal and neonatal outcome. The findings may contribute to improving the prevention and management of PE.

Information and methods

General information

A total of 126 pregnant women diagnosed with PE and admitted to the First Affiliated Hospital of Xi'an Medical University from June 2021 to January 2024 were selected as the research group. Additionally, 130 healthy pregnant women who gave birth in the same period without other comorbidities were selected as the control group. This study was approved by the Ethics Committee of the First Affiliated Hospital of Xi'an Medical University.

Inclusion and exclusion methods

Inclusion criteria: (1) Met the diagnostic criteria for PE established by the American Congress of Obstetricians and Gynecologists [18], which defined PE as new-onset hypertension (systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg) occurring after 20 weeks of gestation, accompanied by proteinuria (≥300 mg/24 hr) or other signs of end-organ dysfunction; (2) Singleton pregnancy; (3) Complete clinical diagnostic and treatment data; (4) No recent exposure to radiation therapy.

Exclusion criteria: (1) Pre-existing conditions, including hypertension, diabetes mellitus, systemic lupus erythematosus, chronic liver or kidney diseases, and other chronic diseases; (2) Mental or neurological disorders that impair communication or expressive abilities; (3) History of habitual abortion.

Observation indicators

Baseline characteristics, laboratory findings, and pregnancy outcomes, such as pregnancy complications, perinatal complications, and postpartum hemorrhage were collected from pregnant women in both groups.

Baseline data collection: Electronic medical records of all pregnant women were retrieved from the hospital's pathology management system, including age, gestational age, body mass index (BMI), mode of delivery, neonatal gender, and pregnancy complications.

Laboratory indicators acquisition: For each participant, 5 ml of peripheral venous blood was collected on the day of admission for the detection of serum UA, TC, TG, HDL-C, LDL-C, free fatty acids (FFA), Lp-a, apolipoprotein B (ApoB), and apolipoproteinA1 (ApoA1) levels. The levels of UA, TC, TG, HDL-C, LDL-C, ApoB, ApoA1, and Lp-a were measured by a fully automated biochemical analyzer, and the levels of serum FFA were determined by enzyme colorimetric assay. Additionally, LDL-C/HDL-C and ApoA1/ApoB ratios were calculated.

Pregnancy outcomes: Data on pregnancy outcomes included pregnancy complications (gestational diabetes mellitus, gestational hypertension syndrome), perinatal complications (fe-

Table 1. Comparison of baseline data in the two groups of pregnant women

		Research group (n=126)	Control group (n=130)	$Z/t/\chi^2$	*P*
Age (year, $\bar{x}\pm s$)		27.00 (26.00, 29.00)	28.00 (26.00, 29.75)	-1.698	0.087
Parity [n (%)]	Primiparous	84	85	0.247	0.884
	Multiparous	28	32		
	Triparous	14	13		
BMI (kg/m^2)		28.86±2.48	25.83±1.98	10.778	<0.001
Mode of delivery [n (%)]	Vaginal	85	82	0.542	0.462
	Cesarean section	41	48		
Neonatal gender [n (%)]	Male	65	70	0.131	0.717
	Female	61	60		
Neonatal weight (kg)		2.46±0.48	2.79±0.33	-6.241	<0.001
Pregnancy complications	Gestational diabetes	37	6	28.044	<0.001
	Gestational hypertension syndrome	15	3	9.015	0.003
	Gestational cardiac disease	9	11	0.154	0.694

Note: BMI: body mass index.

tal distress, fetal developmental delay, preterm delivery, intrauterine fetal death, and neonatal asphyxia), and postpartum hemorrhage.

Logistic regression analysis of the relationship between the indicators and PE

Logistic regression was used to analyze the correlation between serum levels of UA, TC, TG, HDL-C, LDL-C, FFA, Lp-a, ApoB, ApoA1, LDL-C/HDL-C, ApoA1/ApoB, and the occurrence of PE in pregnant women.

Statistical methods

Graphpad Prism 9 software was used for data analysis. The normality of measured data was analyzed by Kolmogorov-Smirnov (K-S) test. Data with normal distribution were expressed as ($\bar{x}\pm s$) and compared using the t-test. Data not following a normal distribution were analyzed using rank-sum test. The independent samples t-test was used for between-group comparisons and the paired t-test for within-group comparisons. Counted data were expressed as the number of cases and percentage [N (%)], and analyzed using the chi-square test. Logistic regression was used to identify independent risk factors for PE in pregnant women. The Receiver Operating Characteristic (ROC) curve was used to evaluate the diagnostic efficacy of independent prognostic factors. A *P*-value <0.05 was considered significant.

Results

Comparison of baseline characteristics between the two groups

There were no significant differences between the two groups in terms of age, parity, mode of delivery, neonatal gender, and gestational cardiac disease (all P>0.05). However, the BMI and prevalence of gestational diabetes and gestational hypertension in the research group were significantly higher than those of the control group, while the neonatal weight was significantly lower (all P<0.05), see **Table 1**.

Comparison of serum UA and lipid levels between the two groups

The research group showed significantly higher levels of UA, TC, TG, LDL-C, Lp-a, ApoB, and LDL-C/HDL-C compared to the control group (all P<0.05). No significant differences were observed between the two groups in HDL-C, FFA, ApoA1, or ApoA1/ApoB ratio (all P>0.05), as shown in **Table 2**.

Comparison of pregnancy outcomes between the two groups

The incidence of postpartum hemorrhage was significantly higher in the research group than that of the control group (P<0.05). Additionally, the incidences of delayed fetal development, preterm birth, and asphyxia neonatorum in the

Table 2. Comparison of serum UA and lipid levels in the two groups of pregnant women ($\bar{x}\pm s$)

	Research group (n=126)	Control group (n=130)	$Z/t/\chi^2$	P
UA (μmol/L)	379.65±51.31	335.57±36.78	7.881	<0.001
TC (mmol/L)	6.63 (5.81, 7.29)	5.48 (4.78, 6.48)	5.690	<0.001
TG (μmol/L)	4.27±1.51	3.65±0.99	3.929	<0.001
HDL-C (μmol/L)	1.78±0.44	1.85±0.39	-1.302	0.194
LDL-C (μmol/L)	4.14±0.96	3.09±0.87	9.122	<0.001
LDL-C/HDL-C	2.27 (1.83, 2.96)	1.63 (1.34, 2.02)	7.040	<0.001
FFA (μmol/L)	0.54±0.15	0.54±0.12	0.336	0.737
Lp-A (μmol/L)	247.40±41.33	219.55±39.74	5.493	<0.001
ApoA1 (μmol/L)	227.60±31.36	223.17±31.28	1.131	0.259
ApoB (μmol/L)	144.90±23.27	136.45±21.36	3.023	0.003
ApoA1/ApoB	1.57 (1.34, 1.82)	1.63 (1.41, 1.93)	-1.530	0.126

Note: UA: uric acid, TC: total cholesterol, TG: triglycerides, HDL-C: high-density lipoprotein cholesterol, LDL-C: low-density lipoprotein cholesterol, FFA: free fatty acids, Lp-a: lipoprotein-a, ApoA1: apolipoprotein-a1, and ApoB: apolipoprotein B.

Table 3. Comparison of pregnancy outcomes between the two groups of pregnant women [n (%)]

	Research group (n=126)	Control group (n=130)	χ^2	*P*
Postpartum hemorrhage	18	7	5.753	0.017
Fetal distress	10	11	0.023	0.878
Delayed fetal development	9	2	4.887	0.027
Premature birth	20	5	10.501	0.001
Dead fetus in uterus	3	4	0.117	0.733
Asphyxia neonatorum	7	1	4.842	0.028

research group were significantly higher than those of the control group (all P<0.05). However, there was no significant differences between the two groups in terms of the incidences of fetal distress and dead fetus in uterus (both P>0.05), as shown in **Table 3**.

Analysis of risk factors for PE in pregnant women

Using PE as the dependent variable (0= no, 1= Yes), and variables including age, parity, BMI, mode of delivery, neonatal gender, neonatal weight, gestational diabetes, gestational hypertension, gestational cardiac disease, UA, and blood lipid levels as independent variables, univariate logistic regression was conducted (**Table 4**). Significant risk factors associated with the occurrence of PE included BMI (OR=1.886, P<0.001), neonatal weight (OR= 0.088, P<0.001), gestational diabetes (OR= 0.116, P<0.001), gestational hypertension (OR=0.175, P=0.007), UA (OR=1.022, P< 0.001), TC (OR=1.777, P<0.001), TG (OR= 1.521, P<0.001), LDL-C (OR=3.506, P<0.001), LDL-C/HDL-C (OR=3.554, P<0.001), Lp-a (OR= 1.018, P<0.001), and ApoB (OR=1.017, P= 0.003).

Multifactorial logistic regression analysis of PE occurrence in pregnant women

Further multivariate logistic regression analysis revealed that BMI (OR=2.023, P<0.001), neonatal weight (OR=0.143, P=0.006), UA (OR=1.030, P<0.001), TC (OR=1.889, P= 0.005), TG (OR=1.684, P=0.021), and LDL-C (OR=2.800, P=0.015) were all independent risk factors for PE in pregnant women (**Table 5**).

ROC curve analysis results

ROC curve analysis demonstrated high diagnostic efficacy of various influencing factors for predicting the occurrence of PE: BMI (AUC=0.835, cut-off =26.79 kg/m^2), neonatal weight (AUC=0.755, cut-off =2.415 kg), UA (AUC=0.765, cut-off =371.4 μmol/L), TC (AUC= 0.706, cut-off =5.615 mmol/L), and LDL-C (AUC=0.792, cut-off =3.675 mmol/L), as shown in **Table 6** and **Figure 1**.

Table 4. One-way logistic regression analysis of factors associated with PE in pregnant women

	B	*S.E.*	*P*	*OR*	*95% CI*
Age	-0.079	0.051	0.119	0.924	0.835-1.020
Parity	-0.004	0.185	0.984	0.996	0.692-1.433
BMI	0.634	0.085	<0.001	1.886	1.614-2.253
Mode of delivery	0.014	0.243	0.953	1.014	0.629-1.636
Neonatal gender	-0.091	0.250	0.717	0.913	0.558-1.493
Neonatal weight	-2.430	0.369	<0.001	0.088	0.041-0.176
Gestational diabetes	-2.151	0.462	<0.001	0.116	0.043-0.269
Gestational hypertension syndrome	-1.744	0.646	0.007	0.175	0.040-0.547
Gestational cardiac disease	0.184	0.468	0.695	1.202	0.480-3.083
UA	0.022	0.003	<0.001	1.022	1.016-1.029
TC	0.575	0.112	<0.001	1.777	1.436-2.233
TG	0.419	0.110	<0.001	1.521	1.234-1.903
HDL-C	-0.394	0.303	0.193	0.674	0.370-1.216
LDL-C	1.254	0.179	<0.001	3.506	2.514-5.080
LDL-C/HDL-C	1.268	0.213	<0.001	3.554	2.394-5.534
FFA	0.316	0.933	0.735	1.372	0.220-8.643
Lp-a	0.018	0.004	<0.001	1.018	1.012-1.026
ApoA1	0.005	0.004	0.259	1.005	0.997-1.013
ApoB	0.017	0.006	0.003	1.017	1.006-1.029
ApoA1/ApoB	-0.445	0.340	0.191	0.641	0.326-1.243

Note: BMI: body mass index, UA: uric acid, TC: total cholesterol, TG: triglycerides, HDL-C: high-density lipoprotein cholesterol, LDL-C: low-density lipoprotein cholesterol, FFA: free fatty acids, Lp-a: lipoprotein-a, ApoA1: apolipoprotein-a1, and ApoB: apolipoprotein B, PE: preeclampsia.

Table 5. Multifactorial logistic regression analysis of risk factors for PE in pregnant women

	B	*S.E.*	*P*	*OR*	*95% CI*
BMI	0.705	0.147	<0.001	2.023	1.563-2.792
Neonatal weight	-1.946	0.703	0.006	0.143	0.033-0.536
Gestational diabetes	-0.725	0.771	0.347	0.484	0.099-2.080
Gestational hypertension syndrome	-1.362	1.199	0.256	0.256	0.014-2.096
UA	0.029	0.006	<0.001	1.030	1.018-1.043
TC	0.636	0.224	0.005	1.889	1.239-3.008
TG	0.521	0.225	0.021	1.684	1.101-2.675
LDL-C	1.030	0.425	0.015	2.800	1.227-6.430
LDL-C/HDL-C	0.326	0.462	0.480	1.386	0.662-3.693
Lp-a	0.009	0.006	0.164	1.009	0.997-1.022
ApoB	0.012	0.012	0.323	1.012	0.989-1.038

Note: BMI: body mass index, UA: uric acid, TC: total cholesterol, TG: triglycerides, HDL-C: high-density lipoprotein cholesterol, LDL-C: low-density lipoprotein cholesterol, Lp-a: lipoprotein-a, and ApoB: apolipoprotein B, PE: preeclampsia.

Discussion

Preeclampsia (PE) is a pregnancy-specific hypertensive disorder that, when severe, can progress to significant complications. PE is characterized by hypertension and proteinuria, which may cause varying degrees of multi-organ damage, making it a major contributor to adverse maternal and neonatal outcomes [19]. This disease usually shows a dynamic development, with its continuous deterioration being a critical focus of clinical attention. PE can lead to serious adverse outcomes such as HELLP syndrome, eclampsia, pulmonary edema, pla-

Table 6. ROC curve analysis of various factors predicting PE in pregnant women

	AUC	Cut off	Specificity	Sensitivity	95% CI	Youden index
BMI	0.835	26.79	72.31%	80.16%	0.786-0.883	52.47%
Neonatal weight	0.755	2.415	86.92%	56.35%	0.696-0.815	43.27%
UA	0.765	371.4	86.15%	59.52%	0.705-0.824	45.68%
TC	0.706	5.615	56.15%	80.16%	0.642-0.770	36.31%
TG	0.622	5.015	94.62%	30.95%	0.553-0.691	25.57%
LDL-C	0.792	3.675	76.92%	68.25%	0.737-0.846	45.18%

Note: BMI: body mass index, UA: uric acid, TC: total cholesterol, TG: triglycerides, LDL-C: low density lipoprotein cholesterol, PE: preeclampsia.

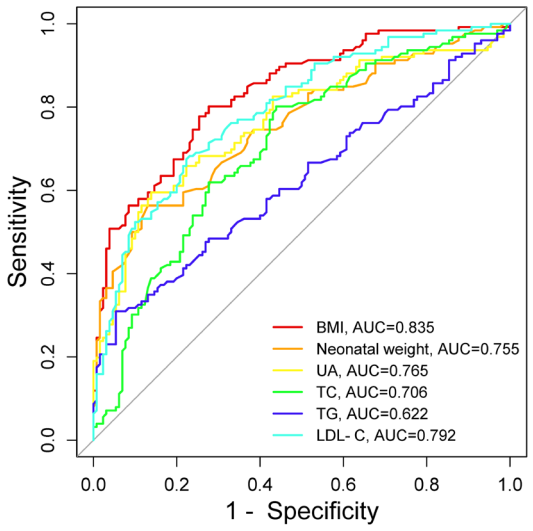

Figure 1. ROC curves of independent factors for predicting PE. Note: BMI: body mass index, UA: uric acid, TC: total cholesterol, TG: triglycerides, LDL-C: low-density lipoprotein cholesterol, PE: preeclampsia.

cental abruption, fetal growth restriction, and even stillbirth, contributing to high maternal and neonatal mortality rates [20-22]. Therefore, early prediction and timely treatment for PE are crucial for improving maternal and perinatal outcomes.

Although the specific role of blood lipids in PE pathogenesis is not fully understood, evidence suggests that elevated blood lipid levels may participate in its pathogenesis by interfering with endothelial function and triggering oxidative stress reactions [23]. Meanwhile, high UA level may trigger the activation of the renin-angiotensin-aldosterone system, promoting vasoconstriction [24]. In addition, elevated adenosine deaminase activity in PE patients may also increase UA levels and inflammatory immune responses, aggravating oxidative stress and endothelial dysfunction [25]. Together, blood lipids and UA exhibit synergistic effects, jointly driving the development of PE.

During pregnancy, metabolic adaptations occur to meet the physiologic needs of the growing fetus. Increased maternal insulin resistance and estrogen levels regulate the metabolic activities of the liver and adipose tissue, influencing blood concentrations of TC, TG, FFA, and phospholipids [26-28]. ApoA1 and ApoB, as the key components of high-density lipoprotein (HDL) and low-density lipoprotein (LDL), respectively, play critical roles in the transport and metabolism of cholesterol. However, this compensatory metabolism may lead to abnormal elevations of blood lipid levels, particularly TG and FFA, possibly leading to hyperlipidemia [29]. Elevated serum UA is now recognized as an early marker of PE, greatly improving diagnostic accuracy for PE. Thabat Al Maiahy [30] reported that serum levels of TC, TG, and LDL were significantly higher in PE patients compared to healthy pregnant women, while serum HDL levels were lower. Similarly, Yusuf Dal found that the serum UA concentration was significantly higher in PE patients compared with healthy pregnant women (6.2±1.7 U/L vs. 4.5±1.2 U/L, P=0.001), with a sensitivity of 82.7% for PE diagnosis when serum UA exceeded 4.7 U/L [31]. Additionally, other studies corroborate that TG, UA, and TC levels are markedly elevated in PE patients [32], aligning with our findings.

In this study, we confirmed that the serum levels of UA, TC, TG, LDL-C, FFA, Lp-a, ApoB, and LDL-C/HDL-C in PE patients were significantly higher than those of the control group. Furthermore, we observed that the BMIs of PE patients (28.86±2.48 kg/m^2) was significantly higher than in the control group (25.83±1.98

kg/m^2). Overweight and obesity before pregnancy, as well as rapid weight gain during pregnancy, are often accompanied by insulin resistance and dyslipidemia, which collectively contribute to endothelial dysfunction [33]. For example, abnormally elevated TG and LDL-C may alter the expression of endothelin receptors and endothelin-1, exacerbating endothelial dysfunction [34]. Paré et al. also demonstrated that being overweight or obese was independently associated with an increased risk of PE, with higher BMI observed in pregnant women with PE [35].

Further analysis identified BMI, neonatal weight, UA, TC, TG, and LDL-C as independent risk factors for the occurrence of PE in pregnant women. ROC curve analysis revealed moderate predictive power for these factors: BMI (AUC=0.835, cut-off =26.79), neonatal weight (AUC=0.755, cut-off =2.415), UA (AUC=0.765, cut-off =371.4), TC (AUC=0.706, cut-off =5.615), and LDL-C (AUC=0.792, cut-off =3.675) (moderate predictive power: 0.7≤AUC≤0.9). Research by Xiaobo Zhao et al. [36] similarly found that elevated serum UA levels increased the likelihood of gestational hypertension developing into pulmonary embolism. Their ROC analysis demonstrated that serum UA had a sensitivity of 90.7% and specificity of 69.4% for predicting pulmonary embolism development at the critical value of 303 μmol/L. The critical value of UA in this study is 371.4 μmol/L, which is higher, likely due to regional, dietary, cultural, and individual differences. Wangxiang Chen et al. [37] reported that the AUC values of serum TC, TG, HDL-C, and LDL-C levels for predicting hypertension were 0.759, 0.854, 0.770, and 0.785, respectively, indicating that these indicators have high predictive power for the occurrence of PE in pregnant women, which is consistent with the results of the current study.

This study also evaluated the adverse pregnancy outcomes in women with PE and found significantly higher rates of gestational diabetes, pregnancy-induced hypertension syndrome, and postpartum hemorrhage compared to healthy pregnant women. In addition, the incidences of fetal growth retardation, premature birth, and neonatal asphyxia in the PE group were significantly higher than those in the control group. Leonoor van Eerden et al. [38] observed high rates of adverse outcome among 79 women who terminated their pregnancies due to PE. Among these, 13 and 16 pregnant women developed chronic hypertension and thrombosis respectively, while 7 experienced miscarriage. At an average gestational age of 32 weeks, the overall recurrence rate of PE was 29% and 52% of these women experienced various pregnancy complications, including miscarriage. Recent research [39] further highlights that patients with early-onset severe PE are at higher risk of adverse pregnancy outcomes, such as fetal distress, neonatal asphyxia, postpartum hemorrhage, and premature birth. However, treatment with labetalol and magnesium sulfate combined with low molecular weight heparin calcium have been shown to significantly reduce these adverse events.

This study delves into the correlation between late-pregnancy UA and blood lipid levels with the occurrence of PE and its associated pregnancy outcomes, providing new insights for early identification and intervention. However, there are some limitations to the research. First, the sample size of this study was relatively small, which may result in selection bias. Second, as a single-center study, the findings may be influenced by specific population characteristics, limiting their generalizability. In addition, factors such as genetic background, lifestyle habits, and environmental influences that may affect PE development, were not analyzed. Given these limitations, future research should expand the sample size, adopt a multicenter prospective design, control for confounding factors, validate biomarkers, and develop prevention strategies based on the results to optimize the management of PE.

Conclusion

This study highlights a significant relationship between late pregnancy blood lipids and UA levels with PE and its pregnancy outcomes. BMI, UA, TC, TG, LDL-C, and Lp-a are independent influencing factors for the occurrence of PE in pregnant women, with BMI, UA, TC, and LDL-C showing high predictive power for the onset of PE in pregnant women. These findings are of great significance for the early identification of high-risk pregnant women with PE and for the prevention of adverse pregnancy outcomes.

Disclosure of conflict of interest

None.

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