

**COMPARATIVE ASSESSMENT OF SERUM LIPID PROFILE AND URIC ACID
BETWEEN PREGNANT NORMOTENSIVE AND PREGNANCY-INDUCED
HYPERTENSIVE WOMEN IN PORT HARCOURT**

ABSTRACT

Aim: The aim of this study was to compare the serum lipid profile and uric acid between normotensive and hypertensive pregnant women.

Study Design: Cross-sectional study

Study Area and Duration: Port Harcourt, Nigeria. January – July, 2022.

Method: A cross-sectional study was carried out among 50 hypertensive and 50 normotensive pregnant women from RSUTH, PH. Blood pressure was measured. Demographic data such as age, parity gestational age was collected. Fasting blood samples were collected and analyzed for total cholesterol, triglyceride, low-density lipoprotein (LDL), high-density lipoprotein (HDL) and uric acid.

Results: The study showed a significantly higher ($P < 0.05$) level of TC and LDL among the hypertensive group compared to the normotensive group. There was no statistically significant difference in the uric acid levels between the subcategories of the study group.

Conclusion: From the results of the present study, it was concluded that PIH is associated with increased levels of TC and LDL. This association may be important in understanding the pathologic processes of the disease and in developing strategies for its prevention and early diagnosis.

Keywords: *Eclampsia, Preeclampsia, Lipid profile, Uric acid, pregnancy induced hypertension*

INTRODUCTION

Hypertensive disorders of pregnancy, an umbrella term that include preexisting and gestational hypertension, preeclampsia, and eclampsia, complicate up to 10% of pregnancies and are a significant cause of maternal and perinatal morbidity and mortality[1]. Hypertension is common in pregnancy. Approximately 1 in 10 women will have one or more episodes of raised blood pressure before delivery. The hypertensive disorders of pregnancy pose a significant risk to maternal life during pregnancy, labour, and the immediate postpartum period. Preeclampsia affects 2% to 8% of pregnancies worldwide and is responsible for 10% to 15% of maternal deaths. It is also linked to an increase in maternal morbidity. Because of intrauterine growth restriction (IUGR), preterm birth, and oligohydramnios, preeclampsia can cause perinatal morbidity and mortality. Preeclampsia is diagnosed after 20 weeks of gestation and the onset can occur both intrapartum and postpartum. The diagnosis is established when hypertension (defined as systolic BP ≥ 140 and diastolic BP ≥ 90 on two occasions at least 4 hours apart) is associated with proteinuria (≥ 300 mg/24 hr or random urine protein/creatinine ratio ≥ 0.3) [2]. According

to Jena et al., [3], the following factors may contribute to the pathogenesis of pregnancy-induced hypertension: uteroplacental ischemia, endothelial dysfunction, anti-angiogenic factor, renin-angiotensin system and genetics in preeclampsia[1]. The risk factors for preeclampsia are classified based on various factors such as maternal, paternal, or pregnancy state-specific [3]. It has been reported that lipoprotein metabolism is directly related to PIH. Even in normal pregnancy, there is an increase in plasma lipids, but it is not atherogenic and may be physiological due to hormonal control. When this mechanism for adjusting physiologic hyperlipidemia is disrupted, pregnancy complications occur. Normal human pregnancy causes pronounced physiologic hyperlipidemia, including a gestational increase in blood triglyceride and cholesterol levels [4]. Increased lipid synthesis raises the PGI₂:TXA₂ ratio, which plays a role in the pathogenesis of pregnancy-induced hypertension. As a result, hyperlipidemia may be an essential marker of pregnancy toxemia. Elevated lipoprotein levels may influence fibrinolysis and have a negative impact on pregnancy outcomes [5]. In preeclampsia, oxidative stress is thought to be caused by an increase in the formation of lipid peroxides, reactive oxygen species, and superoxide anion radicals, resulting in an imbalance in the production of pro-oxidant and antioxidant defenses. As a result, endothelial dysfunction, platelet and neutrophil activation, and altered lipid synthesis lead to a decrease in the prostaglandin I₂ and thromboxane A₂ ratio [6]. Uric acid, a byproduct of purine degradation, is primarily produced in the liver and excreted primarily by the kidneys [7]. Uric acid is the end product of purine metabolism in humans: it is the result of hypoxanthine and xanthine degradation by xanthine oxidase (XO) and xanthine dehydrogenase (XDH) (XD). [8]. The link between elevated serum uric acid and preeclamptic pregnancies was discovered almost a century ago [9]. Reduced uric acid clearance due to decreased glomerular filtration rate, increased reabsorption, and decreased secretion may be the cause of elevated serum levels in preeclamptic episodes [10]. Hyperuricemia in pregnancy is linked to poor fetal outcomes and preeclampsia. Uric acid inhibits amino acid transfer in the placenta and thus inhibits fetal growth [11]. The current study was carried out to comparatively assess the lipid profile and uric acid between pregnant normotensive and pregnancy-induced hypertensive women in Port Harcourt.

2. METHODS

2.1 Study Area

The study was undertaken in the Rivers State University Teaching hospital, Port Harcourt, Rivers State. This research was specifically carried out in the antenatal clinic of the Department of Obstetrics & Gynaecology, which specializes in the care of pregnant women, their unborn children and the management of diseases specific to women and the general health of the female.

2.2 Study Design

This was a cross-sectional study of pregnant women receiving antenatal care at the Rivers State University Teaching Hospital, Port Harcourt, Rivers State.

2.3 Study Sample

The population of this study was defined by participants' pregnancy and attendance at the antenatal clinic of the Rivers State University Teaching Hospital, precisely from November 2021 to February 2022. Simple random sampling was used to select participants who met the inclusion criteria. The study was undertaken on 100 pregnant women, among whom 50 were antenatal mothers with PIH undergoing antenatal care at the Rivers State University Teaching Hospital. They were randomly selected and screened for PIH at their antenatal visits. Fifty (50) healthy antenatal mothers that are normotensive from the same hospital were randomly selected as the control group for comparison.

The sample size was calculated using the Leslie Fischer's formula.

$$\frac{(Z\alpha + Z\beta)^2 (P_0 (1 - P_0) + P_1 (1 - P_1))^2}{(P_1 - P_0)^2}$$

where:

N = required minimum sample size for each group.

Z α = % of normal distribution corresponding to the required significant level of 5% = 1.96

Z β = point of normal distribution corresponding to the statistical power of 80% = 0.842

P₀ = response in the first group (cases) from previous study = 0.90

P₁ = expected response in the second group (control) = 0.80

$$n = \frac{(1.96 + 0.842)^2 (0.80 (1-0.80) + 0.90(1.0-0.90))^2}{(0.9-0.8)^2}$$

≈ 49 (minimum sample size in each group)

This was rounded up to 50 subjects in each group with a total of 100 women for the study.

2.4 Ethical Consideration

Ethical approval for the study was sort and obtained from the Institutional Ethics Committee of the Rivers State University and the Institutional Ethics Committee of the Rivers State University Teaching Hospital. After explaining the nature and purpose of the study, informed consent was taken from each subject for participation in the study.

2.4 Biochemical Assessments

Venous blood samples were collected aseptically from each participants and subjected to biochemical analyses. The serum levels of the lipid profile, such as total cholesterol (TC), triglycerides (TGs), and high-density lipoprotein (HDL), were analyzed using the enzymatic

colorimetric method using Randox kits. Biochemical analysis of uric acid was determined by quantitative estimation on the colourimetric method using enzymatic uricase method using Randox reagent kit according to the manufacturer's instruction.

2.5 Data Analysis

Quantitative data obtained from the laboratory analysis of samples were subjected to descriptive and statistical analysis. The Statistical Package for Social Sciences (SPSS) version 20.00 software was used. Student z-test, Chi-square and Pearson's product-moment correlation tools were used to test for intergroup variation and relationship in the respective variables. P-value of <0.05 was considered statistically significant.

3. RESULT

The data presented in Table 1 represent some anthropometric characteristics of Normotensive and Hypertensive pregnant women attending Antenatal Clinic in the Rivers State University Teaching Hospital in Port Harcourt. Half of the population of the normotensive subjects fell within the age range of 23 and 33 years and the other half fell within the range of 34 and 44. The hypertensive subjects were made of 44% who are within the age bracket of 23 and 33 years and the rest (56%) within their 34 and 44 years of age. Interestingly, most of them (70% of the normotensive and 66.67% for the hypertensive) subjects have attained tertiary education.

The marital statuses of the subjects indicated that 90% and 100% of the normotensive and hypertensive subjects were married, respectively.

Table 1: Demographic Characteristics of Participants

| | Normotensive n =50 (%) | Hypertensive n =50 (%) |
|---------------------------|-----------------------------------|-----------------------------------|
| Age (years) | | |
| 23-33 | 25(50) | 22(44) |
| 34-44 | 25(50) | 28(56) |
| Level of Education | | |
| Secondary | 15(30) | 17(33.33) |
| Tertiary | 35(70) | 33(66.67) |
| Marital Status | | |
| Single | 5(10) | 0(0) |
| Married | 45(90) | 50(100) |

The data in Table 2 shows the outcome on the lipid profile changes in normotensive and hypertensive pregnant women in Port Harcourt. The result indicated significant ($p < 0.05$) increases in the levels of total cholesterol (TC) and Low Density Lipoprotein Cholesterol (LDL-

C) of the hypertensive subjects when compared to that of the normotensive subjects. However, no significant ($p>0.05$) changes were recorded in the levels of triglycerides (TG) and High-Density Lipoprotein Cholesterol (HDL-C) when the values of the hypertensive subjects were compared to their normotensive counterparts.

Table 2: Lipid Profile Changes in Normotensive and Hypertensive Pregnant Women

| Lipid Profile | Study Groups | |
|---|-----------------------------|-----------------------------|
| | Normotensive Pregnant Women | Hypertensive Pregnant Women |
| Total Cholesterol (TC) (mmol/L) | 4.29 ± 0.68 | 5.52 ± 0.63* |
| Triglyceride (TG) (mmol/L) | 1.59 ± 0.58 | 1.66 ± 0.57 |
| High-Density Lipoprotein Cholesterol (HDL-C) (mmol/L) | 1.12 ± 0.22 | 1.05 ± 0.13 |
| Low-Density Lipoprotein Cholesterol (LDL-C) (mmol/L) | 2.44 ± 0.69 | 3.71 ± 0.56* |

Values are expressed as Mean ± SD; n=50; *Significant at $P<0.05$ when compared to Normotensive values.

The data represented in Figure 1 shows the comparison of the uric acid level in Normotensive and Hypertensive Pregnant Women in Port Harcourt. In addition, it was found that the uric acid level of the hypertensive subjects did not significantly ($p>0.05$) vary from that of the normotensive subjects.

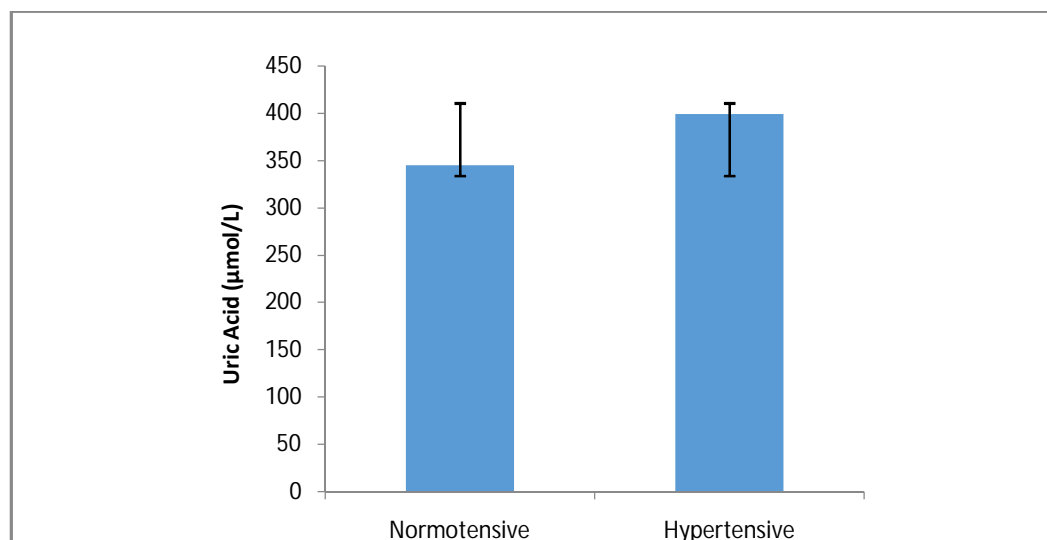


Figure 1: Comparison of Uric Acid ($\mu\text{mol/L}$) Level in subjects.

DISCUSSION

Pregnancy-induced hypertension has been a significant cause of global maternal and infant morbidity and mortality [12]. Thus, bearing in mind the established relation between lipid profile and predisposition to pregnancy-induced hypertension (PIH) and the quest for reliable and fast diagnosis and enhanced management of PIH, the present study was undertaken. As the outcome is expected to help better understand and explore how the relationship between serum lipid level and uric level varies in normotensive and PIH subjects could be helpful to clinicians and pregnant women in this locality. The outcome of the present study is thus discussed in the following paragraphs. The anthropometric characteristics of Normotensive and Hypertensive pregnant women attending Antenatal Clinic in the Rivers State University Teaching Hospital in Port Harcourt indicated a higher population of the PIH subjects were within their 34th and 44th years of age than the normotensive subjects, who had half of its population within the age range of 23 and 33 years. According to Attali & Yogev[13], advanced maternal age could lead to a decrease in fertility and an increase in early fetal loss because of the possible impact on chromosomal and genetic disorders. The age distribution of the present study has thus revealed or validated the fact that increasing maternal age can raise the predisposition to PIH in women. Thus, need for promoting more medical attention on women of advanced maternal age. The result indicated markedly elevated total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) levels in hypertensive pregnant women compared to normotensive pregnant women. Surprisingly, none of the lipid profile parameters showed any significant relationship between blood pressure parameters such as mean arterial pressure and pulse pressure. Considering literature, so much has been insinuated on the predisposition to PIH in the incidence of dyslipidemic conditions in pregnancies[14]. For instance, lipoprotein lipid physiology in pregnancy is said to have important implications for the developing fetus and newborn and the mother. Cholesterol is essential for normal fetal development. It is vital in the formation of cell

membranes[1]. In pregnancy, multiple physiological changes occur that contribute to the alterations in lipid profiles of healthy, gestating women. Initially, there is an anabolic phase with increased lipid synthesis and fat storage in preparation for the increases in fetal energy needs in late pregnancy. Interestingly, during the third trimester, lipid physiology transitions to a net catabolic phase with a breakdown of fat deposits. The catabolism increases substrates for the growing fetus. Overall, the changes in lipid physiology throughout pregnancy allow for proper nutrients for the fetus, and they reflect increasing insulin resistance in the mother[1, 12, 15]. Thus, the above result of the present study has revealed that dyslipidaemic conditions may characterize PIH. Thus, this finding of the present study is in line with the earlier report of Parikh *et al.* [14], which submitted that adverse pregnancy outcomes (APOs) such as hypertensive disorders of pregnancy like dyslipidaemia, amongst others, can increase a woman's risk of developing cardiovascular disease (CVD) risk factors. Furthermore, it can also lead to developing subsequent CVD (including fatal and nonfatal coronary heart disease, stroke, peripheral vascular disease, and heart failure). Endothelial dysfunction is the most critical event in the pathogenesis of hypertension during pregnancy, and abnormal lipid profile levels play a critical role in the induction of endothelial dysfunction [16]. From the present study's result on the changes of the uric acid level showed that uric acid levels were not significantly different between the normotensive and hypertensive women.

CONCLUSION

In conclusion, the results of this study show abnormal lipid metabolism predominantly high total cholesterol (TC) and low density lipoprotein (LDL-C) concentration, which may promote vascular dysfunction and oxidative stress seen in pregnancy-induced hypertension. This association could help develop strategies for prevention and early diagnosis of the disorder and ultimately help to improve maternal and foetal outcomes in patients with pregnancy-induced hypertension.

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