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Dyslipidemia and other parameters in women with pregnancy induced hypertension

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ABSTRACT

Background: Several investigations have confirmed changes in lipid profiles and other markers (interleukin [IL]-6, C-reactive protein [CRP]) in women with pregnancy induced hypertension (PIH). Although, the cause an effect correlations between these lipid profiles and other markers with the PIH are yet unknown.
Aim: To study the relationship of serum lipid profile, CRP, and IL-6 with PIH.
Method: A cross-sectional study was done on 60 pregnant women with PIH treated in the General Salah Alden Hospital in Tikrit city, Iraq, from January 2021 to April 2021. Another group of 60 pregnant women who were normal, at the same period, were selected as the control group. The average age of the participants was (27.1 ± 5.0) years. All the blood samples taken were examined for lipid profile, CRP, and IL-6.
Results: The results statistically showed significant differences in IL-6 level and CRP. Whereas, in the Lipid profile (triglyceride [TG], low density lipoprotein cholesterol [LDL-C], high density lipoprotein [HDL]-C, total cholesterol [TC], and very low density lipoprotein cholesterol [VLDL]) were found. Also, it was found that there were significant differences in PIH patients as compared to the control group.
Conclusion: The findings revealed that the lipid profile was aberrant, and the amount of IL-6 protein was positively linked with blood pressure.

Keywords: *Dyslipidemia, Pregnancy hypertension, Interleukin-6 (IL-6), Inflammatory cytokines, C- reactive protein CRP*

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INTRODUCTION

Pregnancy hypertension is an illness that frequently affects roughly around 10% of pregnant women.[1] Hypertensive illnesses affecting pregnancy are still in serious concern in underdeveloped nations, endangering the health of the mother and fetal viability, which leads to increasing maternal and newborn mortality and morbidity.[2]

Pregnancy induced hypertension (PIH) may be developed by one of the four factors. The first is oxidative stress, which can promote hypertension by causing the production of inflammatory factors. Second is the genetic elements that are linked to heredity. Third, the immunologic abnormalities can cause hypertension during pregnancy. The incidence of hypertension during pregnancy is thought to be linked to placental ischemia, which been verified as the fourth cause.[3-6]

Many researchers have discovered that the IL-6 level in patients with PIH is greater than in normal pregnant women. Furthermore, the mother and newborn prognoses in patients with high IL-6 levels are worse than in pregnant women with normal IL-6 levels, implying that IL-6 has an impact on the prognosis of PIH patients.[7] According to several studies, increase in the inflammatory cytokines IL-6 play a crucial part in the etiology of hypertension in response to a fall in the uterine perfusion pressure, (Hypertension in pregnancy in response to IL-6: involvement of AT1 – receptor activation).[8]

Scholars have known for a long time that C-reactive protein (CRP) is a non-specific inflammatory marker. Recent researches, however, have discovered a strong link between CRP and poor pregnancy outcomes, such as stillbirth and PIH.[9]

MATERIALS AND METHODS

A cross-sectional study was done on 60 pregnant women suffering from PIH. These patients were treated at the General Salah-Alden Hospital in Tikrit city, Iraq, from January 2021 to April 2021. Another 60 normal pregnant women, at the same period, were selected as the control group. The average age of the participants was (27.1 ± 5.0) years. All of them assessed physically examination like blood pressers measurement. The patient was diagnosed with hypertension when the systolic blood pressure was >140 mm Hg and diastolic blood pressure was >90 mm Hg. The patients, all women, were diagnosed with dyslipidemia, when their total cholesterol (TC) level was >220 mg/dL or triglyceride (TG) level was >200 mg/dL, and there was a high density of lipoprotein (HDL-C), at a level of <45 mg/dL. The blood samples were taken after 12–14 h of fasting for measuring the serum lipid profile by a standard clinical laboratory procedure. The serum lipids, including TGs, TC, and high density lipoprotein cholesterol (HDL-C) were measured using the enzymatic methods, whereas low density lipoprotein cholesterol (LDL-C) and very low density lipoprotein cholesterol (VLDL), were calculated according to the Friedewald et al. formula[6], IL-6 was assayed using Quantikine R and D systems, ELISA kit, and for CRP the nephelometry method was used. Statistical analysis was acquired by involving, chi-square, t-test, and one way ANOVA. The numerical values were expressed as mean ± standard deviation. The P-values of 0.05 or less were considered as statistically significant.

RESULTS

In the present study 120 pregnant women were evaluated; they were divided equally into two groups, where 60 were hypertensive (PIH) and grouped together, and the rest of the individuals without hypertension were considered the control group. The results are as follows.

Table 1 shows the comparison between the serum associate inflammatory factors IL-6 and CRP in PIH and the control group. A significant difference was found between the two groups (8.17 ± 2.4 vs., 6.67 ± 1.9, $P < 0.5$) and (18.2 ± 8.3, vs., 7.5 ± 2.5, $P < 0.05$), respectively.

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TABLE 1. The inflammatory markers IL-6 and CRP in the patients group (PIH) and normal pregnancy

Characteristic	Patients group (PIH) n = 60 (Mean ± SD)	Control group (normal pregnancy) n = 60 (Mean ± SD)	P-value
IL-6 (pg/mL)	8.17 ± 2.4	6.67 ± 1.9	<0.05
CRP (mg/mL)	18.2 ± 8.3	7.5 ± 2.5	<0.05

IL-6: interleukin-6; CRP: C-reactive protein, PIH: pregnancy induced hypertension.

TABLE 2. The lipid profiles of the case group with PIH and the control group

Lipid profile	Case group (PIH) n = 60 (Mean ± SD)	Control group n = 60 (Mean ± SD)	P-value
LDL-C (mg/dL)	134 ± 19.6	114.1 ± 17.4	<0.05
HDL-C (mg/dL)	24.2 ± 6.8	30.4 ± 8.4	<0.05
TG (mg/dL)	190.1 ± 18.2	174.2 ± 48.6	<0.05
TC(mg/dL)	188.3 ± 46	176 ± 57	<0.05
VLDL (mg/dL)	38.02 ± 4.3	34.8 ± 2.1	<0.05

LDL-C: low density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; TG: triglycerides; TC: total cholesterol; VLDL: very low density lipoprotein cholesterol.

Table 2 clarifies the mean ± SD of lipid profile (LDL-C, HDL-C, TG, TC and VLDL) in PIH cases and the control groups. The lipid profile was significantly higher in PIH patients compared to the healthy control participants, when $P < 0.05$ (134 ± 19.6 vs. 114.1 ± 17.4), (190.1 ± 18.2 vs. 174.2 ± 48.6) (188.3 ± 46 vs. 176 ± 57), (38.02 ± 4.3 vs. 34.8 ± 2.1) respectively. On the other side, HDL was significantly decreased in PIH group (24.2 ± 6.8 vs. 30.4 ± 8.4).

DISCUSSION

Pregnancy induced hypertension is a common problem during pregnancy leading to maternal and fetal death, as well as neonatal death.[10] According to this research, there might be multiple implicit causes or predisposing factors in PIH patients which may contribute to endothelial dysfunction. Despite decades of intense investigation, the cause of many illnesses remain unknown. However, several explanations for the disease etiology have been proposed, including prostacyclin – thromboxane imbalance, endothelial dysfunction, immuno-genetics, and absolute or relative etiology.[11] Pre-eclampsia and gestational hypertension are hypothesized to be caused by "failures" in the normal hemochorial placental system.[12] Gestational hypertension has hereditary underpinnings, since women with a family history of this illness are three times more likely to develop it during pregnancy.[13] In recent years, it has been widely assumed that PIH is linked to immunological dysregulation, and the cytokines, particularly the inflammatory ones, play a key role in immune control.[14] In the current study, the result in Table 1 revealed that the levels of IL-6 in patients with PIH were substantially greater than those in normal pregnant women ($P < 0.05$). This data supports the study by Li et al.[15] Autoantibodies that activate the angiotensin II type I receptor are secreted by monocytes, neutrophils, and T and B lymphocytes. The chain of events that lead to cytokines, notably interleukin-6 (IL-6), triggering angiotensin type I receptor auto antibody, results in a rise in angiotensin II,

a powerful vasoconstrictor.[16] In a similar research, El-Bassyouni et al., found that the direct percentage of IL-6 in individuals with PIH was higher.[17]

The result in the present cross-sectional research identified a positive link between CRP and PIH. CRP is a non-specific inflammatory protein produced by the liver and controlled by plasma interleukin-6 (IL-6).[18] Serum CRP was considerably raised in individuals with PIH according to Qiu et al.[19] Furthermore, another study showed that postpartum PIH patients exhibited greater levels of higher sensitivity CRP than non-PIH patients.[20] Endothelial dysfunction and the renin-angiotensin system have both been linked to inflammation.[20] As a result, it has been suggested that hypertension is partly an inflammatory condition. CRP levels beyond a certain threshold may raise blood pressure by lowering nitric oxide synthesis in endothelial cells, resulting in vasoconstriction and a rise in endothelin–1.[21] A similar published research in Italian was in high agreement with our findings.[14,20] In contrast to prior studies conducted in China, the findings revealed no significant differences between PIH patients and normal pregnant women.[22]

It is known that hyperlipidemia alters lipid metabolism during pregnancy, and it becomes more pronounced as the pregnancy progresses.[23,24] The particular processes that cause these changes are unknown. Lipid metabolism changes during early pregnancy as a result of hormonal regulation.[25] There may be difficulties with lipid metabolism with hyperlipidemia in difficult pregnancies.[26] In addition the activity of hepatic lipase increases, whereas that of lipoprotein lipase decreases.[27] There is also an increase in TG production at the hepatic level as well as a build-up of TGs in the plasma.

Hypertriglyceridemia and other lipid abnormalities are linked to hypertensive disorders of pregnancy in a beneficial way.[28] The role of lipids in the pathophysiology of hypertensive disorders of pregnancy lipid abnormalities, and their impact on the mother and infant are unknown. The studies on this topic are ongoing.

However, in the current study we have found that TC, VLDL, and LDL were considerably greater in women who developed PIH than in normotensive patients, which is in higher or good agreement with previous result according to Vidyabati et al.[29]

According to the aforementioned findings, lipid profile levels in the early second trimester can aid in the prediction of PIH. As a result, it is feasible to diagnose PIH and associated consequences earlier and manage them, which would enhance the mother and fetal prognosis.[30]

CONCLUSION

The findings revealed that the lipid profile was aberrant, and the amount of IL-6 protein was positively linked with blood pressure.

RECOMMENDATION

The findings of these types of investigations will help to further of our understanding of the pathophysiological ramifications of HIP, as well as therapy development for this condition. More research should be done to determine the causal cause for dyslipidemia and high IL6 production, which leads to PIH, in addition to viable therapeutic interventions in the management of PIH.

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