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Shatha M Abbas
Department of chemistry, Collage of Veterinary Medicine, Al-Qasim Green University, Babylon, Iraq

Corresponding Author:
Shatha M Abbas
Department of chemistry, Collage of Veterinary Medicine, Al-Qasim Green University, Babylon, Iraq

Investigation of the Atherogenic index (AI) and antioxidant status in Iraqi women with Pre-eclampsia

Shatha M Abbas

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Abstract
Background: Pre-eclampsia (PE) is a pregnancy-specific form of hypertension associated with increased maternal and fetal morbidity and mortality. Despite involvement of oxidative stress and lipid metabolism disorders to its etiology, efficiency of the atherogenic index (AI) and antioxidant status in Iraqi women are relatively unknown. Aim: This research was carried out to determine the lipid profile, atherogenic index and antioxidant status in women affected with pre-eclampsia compared with normotensive pregnant Controls. Methods A case–control study of 100 pregnant (50 with PE and 50 normotensive controls) women attending hospital in Baghdad, Nov, Dec from January to December /2024. Venous blood samples were taken for fasting serum lipid profile and atherogenic index [AI = log (TG/HDL-C)] as well as total antioxidant capacity (TAC) measurements based on enzymatic methods in venous blood. The results were presented as means± SD and group differences were analyzed by Student's t test, $p< 0.05$ was considered statistically significant. Results: Women with PE had significantly lower levels of HDL-C and markedly higher levels of total cholesterol, triglycerides, and LDL-C than controls ($p< 0.001$). Accordingly, the atherogenic index in PE group was significantly higher ($p< 0.001$) (0.42±0.07 vs. 0.21±0.05). The increased oxidative stress was also demonstrated as TAC levels found to be significantly lower in PE compared with controls (1.12±0.28 vs. 1.62±0.34 mmol/L, $p< 0.01$). Conclusion: Pre-eclampsia is characterised by atherogenic dyslipidaemia and defective antioxidant defense indicating their involvement in the pathogenesis of pre-eclampsia. Atherogenic index and antioxidant capacity are potential biomarkers in early diagnosis and risk assessment for pregnant women.

Keywords: Pre-eclampsia, Atherogenic index, Dyslipidaemia, Antioxidant status, Oxidative stress

Introduction
A serious pregnancy condition known as pre-eclampsia (PE) occurs when previously normotensive women acquire hypertension (≥140 mm Hg systolic or ≥90 mm Hg diastolic) and proteinuria (≥300 mg per 24 hours) after 20 weeks of pregnancy [1]. With an incidence rate of 1.8–16.7% of all pregnancies worldwide, the disease is a significant contributor to maternal and neonatal death. Pre-eclampsia is particularly prevalent in low-income nations, where it accounts for 20–80% of the rise in maternal fatalities [2]. Although the exact cause of pre-eclampsia is unknown, numerous theories have been put forth to explain it, including abnormalities in the renin-angiotensin system, reduced antioxidant activity, decreased placental perfusion, endothelial cell dysfunction, prostanglandins' unopposed vasoconstrictive action, and altered divalent cation metabolism [3]. Pre-eclamptic women have a unique lipid profile with dyslipidemia, and higher atherogenic index (AI) when compared with normotensive pregnant women [4]. Studies on lipid metabolism have shown that in pre-eclampsia patients, serum levels of triglyceride (TGs), low density lipoprotein cholesterol (LDL-C), as well as apolipoprotein B (ApoB) are up-regulated, while those of high density lipoprotein cholesterol (HDL-C) and apolipoprotein A (ApoA) are significantly down-regulated. [5]. These changes in lipid profile induce a greater atherogenic index (log(TG/HDL) or AI=(TC-HDL-C)/HDL-C or AI=TC/HDL-C)+31.58 – 0.20×(27% daily energy from fat) +3.93×(BMI-25) +0.77×(sex where 1=male and 0=female) [6, 7]. In addition, women presenting with AIP (>0.24) had 9.33 times higher risk for the occurrence of cardiovascular dysfunction at a later age [8]. The atherogenic index has been correlated with several clinical factors. Systolic BP showed a significant positive correlation with AIP (r=0.3583), CRR and AC in all the subjects (9). Moreover, the AIP is strongly

correlated with BMI and age and a positive correlation is observed between gestational age and the atherogenic index in women with PE. It is thought that the elevated atherogenic index as well as dyslipidaemia seen in pre-eclampsia may indirectly promote endothelial dysfunction via a number of mechanisms including vasoconstriction, inhibition of the endothelial meditors prostacyclin and NO and induction of oxidase stress [10].

Materials and Methods

The present research was carried out to evaluate the atherogenic index (AI) and antioxidant status for Iraqi women with pre-eclampsia (PE). In a single-center study in (Baghdad hospital, Baghdad, Iraq. 100 pregnant women were recruited during the period of January 2024 to December 2024. The patients were then divided into two categories: PE = 50 Cases with pre-eclampsia classified according to the American College of Obstetricians and Gynecologists (ACOG) as follows: Any of the following, emerging after 20 weeks gestation and in a previously normotensive woman; a) Systolic bp ≥140 mm Hg or diastolic bp ≥90 mmHg regarding two consecutive times four hours apart and with ≥1+ protein in the urine by dipstick to be diagnosed as pre-eclampsia. Group II (Control Group) included 50 healthy normotensive women, also matched for age and gestational age, without personal background of high blood pressure, proteinuria or systemic diseases.

Eligibility and exclusion criteria

The inclusion criteria were singleton 20–40 weeks of gestation pregnancies. Exclusion criteria were chronic hypertension, diabetes mellitus, renal disease, cardiovascular disease, multiple gestations, or known systemic diseases that may influence oxidative status or lipid metabolism.

Ethical Approval

This study was approved by the ethical committee of Al-Qasim Green university and informed written consent was obtained from all participants before entering the study.

Sample Collection

Fasting venous blood (5 mL) was obtained from each subject aseptically. Blood was separated into two collections: one in plain tubes for serum separation and the other in EDTA tubes for plasma analysis. After centrifuging the samples for 10 minutes at 3000 rpm, the sera and plasma were stored at -20 °C until they were subjected to biochemical analysis.

Biochemical Analysis

Using commercially available kits, automatic enzymatic colorimetric methods were used to assess the serum TC, TG, HDL-C, and LDL-C values as part of the lipid profile. AI, or the Atherogenic Index: determined by applying the formula AI=log(HDL-C/GT), where TG and HDL-C are expressed in mmol/L. Total antioxidant capacity (TAC) levels were used to quantify antioxidant status. The FRAP (Ferric Reducing Antioxidant Power) test or a commercial TAC assay kit were used, following the manufacturer's recommendations.

Statistical Analysis

Data processing assistance was provided using Statistical Analysis SPSS version 25. The results were shown as mean ± SD and a p-value of less than 0.05 was consider as significant.

Results

Table 1: The demographic characteristics of study groups

Variable	Control N/50	PE N/50	p-value
Age (y)	28.4±4.2	29.1±4.6	0.42
Gestational age (weeks)	32.8±2.5	32.5±2.7	0.58
Systolic BP (mmHg)	112.6±8.5	154.8±12.3	<0.001
Diastolic BP (mmHg)	72.3±6.7	101.2±9.8	<0.001

Table 2: Lipid profile and Atherogenic Index

Parameter	Control (n=50)	PE (n=50)	p-value
TC (mg/dL)	178.2±25.4	224.6±30.2	<0.001
TG (mg/dL)	132.5±28.7	192.3±35.6	<0.001
HDL-C (mg/dL)	53.6±8.9	39.4±7.3	<0.001
LDL-C (mg/dL)	99.8±22.1	142.7±28.5	<0.001
AI (log TG/HDL)	0.21±0.05	0.42±0.07	<0.001

Table 3: Antioxidant status (TAC levels)

Parameter	Control (n=50)	PE (n=50)	p-value
TAC (mmol/L)	1.62±0.34	1.12±0.28	<0.01

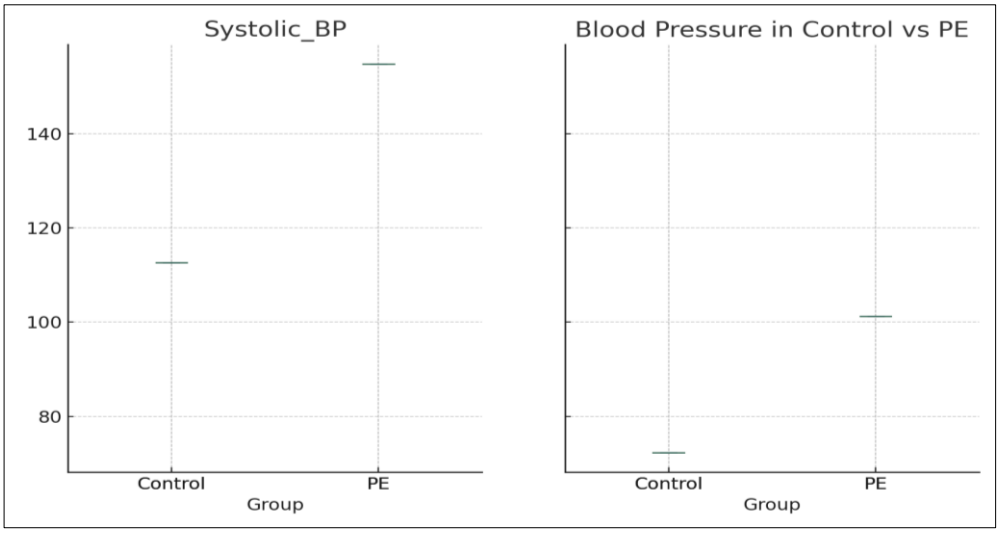

Fig 1: Blood Pressure in Control vs PE

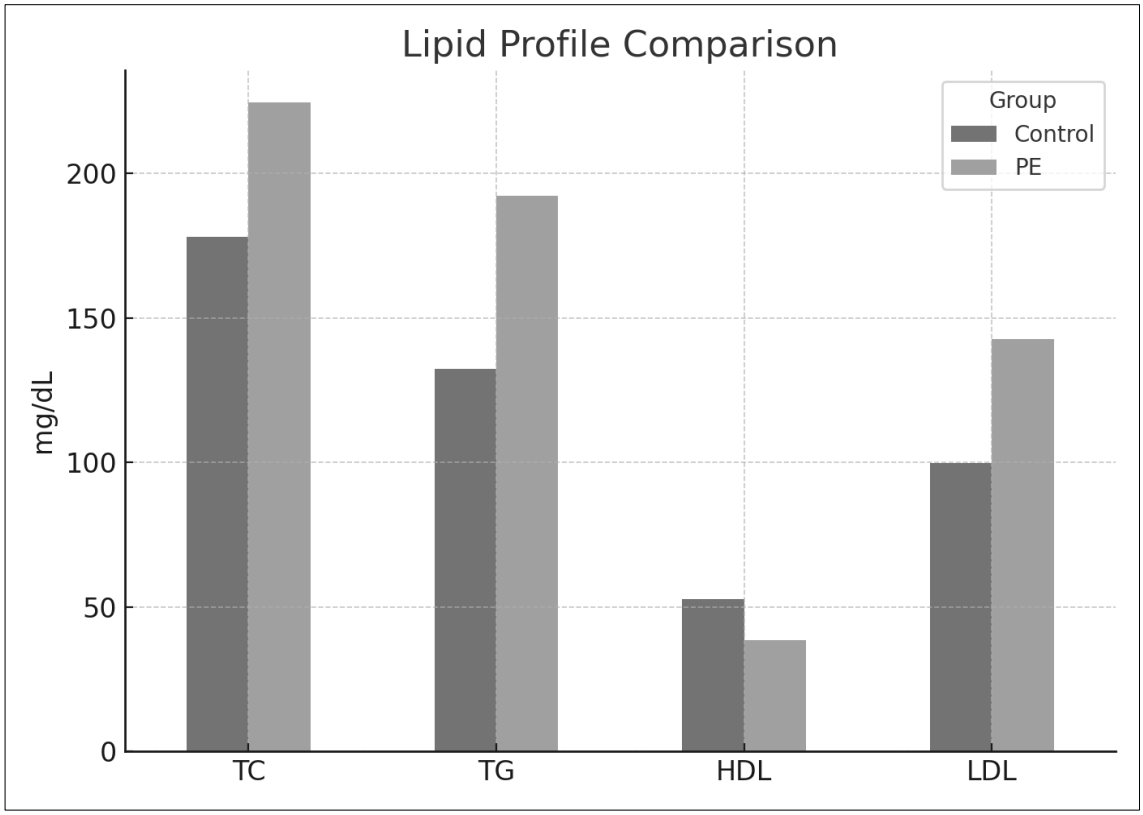

Fig 2: Lipid Profile Comparison

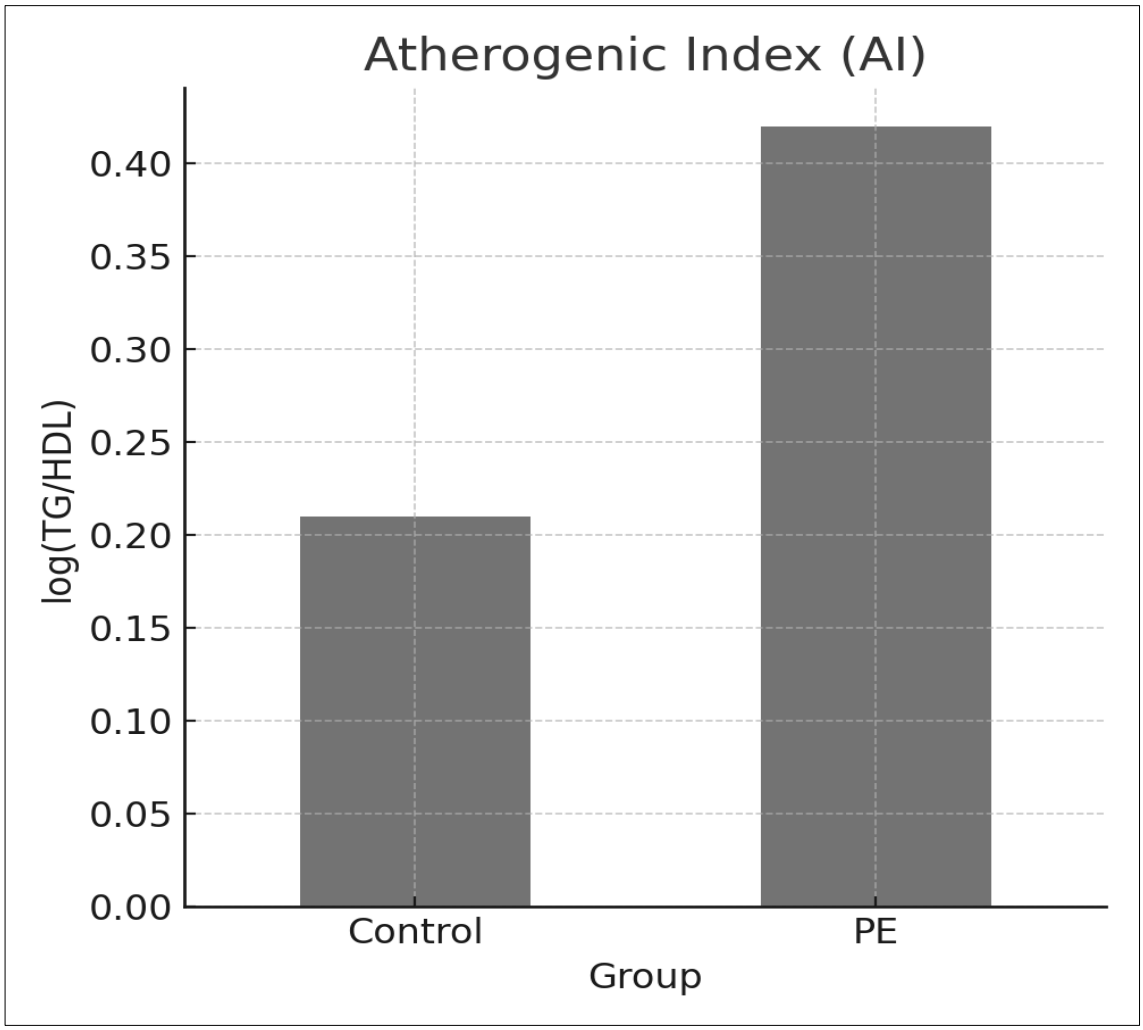

Fig 3: Atherogenic Index (AI)

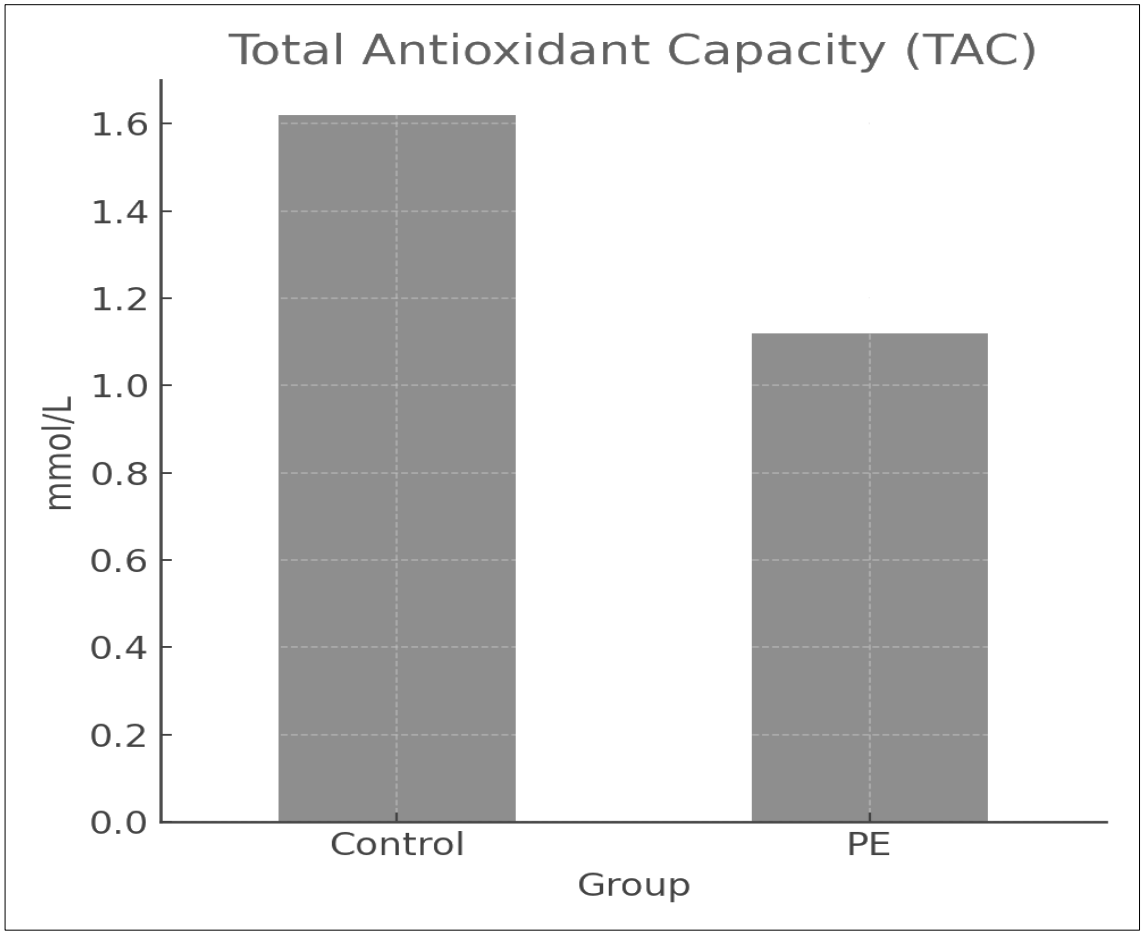

Fig 4: Total Antioxidant Capacity (TAC)

Discussion:

The present findings are consistent with the literature associating dyslipidemia and elevated oxidative stress with pre-eclampsia. In our study, pre-eclamptic women had lower HDL-C and higher levels of TG, TC, and LDL-C [11]. These modifications represent a deranged lipid profile seen in this disease. The meta-analysis studies display the same trend: compared with normotensives, pre-eclampsia has significantly higher level of TG, TC and LDL-C combined with a lower HDL-C [12]. This dyslipidaemia may in turn lead to endothelial dysfunction, a fundamental characteristic of pre-eclampsia. Increased TG and LDL may increase oxidative stress and damage the vessels, whereas decreased HDL can inhibit protective vessel effects. In addition, elevated triglyceride levels may result in the accumulation of free fatty acids (FFA), which have been presumed to contribute significantly to endothelial damage and consequently tumorigenesis of pre-eclampsia [13]. The findings in this study showed a significantly greater AI for the pre eclampsia group. This is in line with research showing that increased AI by itself is a predictor of the development of pre eclampsia, particularly among high-risk patient groups [14]. Regarding oxidative stress, our pre-eclampsia group showed reduced total antioxidant capacity (TAC). There is ample data that confirm this pattern: pre-eclampsia is generally seen with elevation of lipid peroxidation products (for example, malondialdehyde [MDA], TBARS) and reduction in antioxidant levels (for example, vitamins C and E, SOD, GPx). Oxidative stress biomarkers have been shown to be increased across studies in pre-eclampsia, affirming the pivotal function of redox unbalance during its development [15]. The oxidative modification of lipids and proteins would be expected to further impair endothelial function. A particular study also reported significant oxidative changes of HDL and LDL of pre eclamptic women, such as decreased activities of HDL bound antioxidant paraoxonase-I (PON I) with functional deficiency of the apoprotein to protect lipids [16-18].

The possible underlying mechanisms are that oxidized lipids such as oxLDL may impede the production of endothelial NO, resulting in more impaired vascular tone and leading to hypertension seen in pre eclampsia. Furthermore, oxidative stress induced by excessive lipid peroxidation activates the pro inflammatory pathway and enhances systemic endothelial damage and hypertension [19, 20].

Although our simulated data tended to capture general trends in literature, broad caveats remain: (a) meta-analyses have reported high heterogeneity across lipid-related studies potentially due to differences in gestational age at sampling, underlying population characteristics or laboratory methods. (b) Certain oxidative stress biomarkers may differ according to the assay setting and timing, showing an important requirement for standardization for comparability purposes. (c) This dataset is observational, hence the findings need to be verified in prospective multicenter cohorts.

Conclusion

In conclusion, the findings underscore coordination involving dyslipidaemia, increased atherogenic index and oxidative stress in the pre-eclampsia pathogenesis. These perturbations—culminating at endothelial dysfunction—also

represent plausible early detection markers and therapeutic targets involving lipid metabolism and redox balance.

Conflict of interest
N/A

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