

Original article

Biochemical Changes and Plasma Level of Lipoprotein-Associated Phospholipase A₂ in Patients with and without Coronary Artery Disease: A Cross-Sectional Study

Bahaedin Ben-Mahmud^{1*}, Abdulmunam Fellah², Khaloud Essokni¹

¹Department of Life Science, School of Basic Science, Libyan Academy, Tripoli, Libya

²Department of Medicine, Faculty of Medicine, University of Tripoli, Tripoli, Libya

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Corresponding Email. bahaedin.benmahmud@academy.edu.ly

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ABSTRACT

Coronary artery disease (CAD) is the leading cause of death worldwide. The aim of the study is to evaluate the association between plasma lipoprotein-associated phospholipase A₂ (Lp-PLA₂) and the clinical and biochemical risk factors for CAD among Libyan individuals. This cross-sectional study evaluated plasma concentration of Lp-PLA₂ between patients with CAD and those without CAD in both genders based on clinical history, exercise ECG, two-dimensional echocardiography, and coronary angiography. Several biochemical parameters were estimated. Plasma levels of Lp-PLA₂ were measured. A total of 181 individuals were recruited for this study, involving 125 with CAD and 56 without CAD. Plasma Lp-PLA₂ concentration was significantly higher in patients with CAD versus individuals without coronary artery disease. In addition, the plasma concentration of Lp-PLA₂ was higher in patients >60 years of age compared with patients <60 years old. To conclude that Plasma level of Lp-PLA₂ concentration was independently correlated with CAD, inflammatory marker such as high sensitivity C-reactive protein among Libyan patients.

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INTRODUCTION

Low-density lipoprotein (LDL) and high-density lipoprotein (HDL) particles in the blood are bound by the enzyme Lp-PLA₂. It contributes to the vascular endothelium's pro-atherogenic actions and inflammation. The development of plaque accumulation in the arterial walls, which results in CAD and stroke, is significantly influenced by Lp-PLA₂. A higher risk of cardiovascular events has been correlated with elevated Lp-PLA₂ levels. Based on recent research, several inflammatory cells, including mast cells, T-lymphocytes, and macrophages, are crucial for the formation of atherosclerosis [1]. In contrast to macrophages from normal intimal tissue, these cells, that originate from atherosclerotic plaques, reveal a substantially greater production of Lp-PLA₂, leading to plaque instability [2,3].

This article highlights the enzyme's role in breaking down oxidized lipids, suggesting that Lp-PLA₂ may influence the progression of atherosclerotic plaque. Plaque remodeling is an ongoing process that involves multiple factors [4,5]. Therefore, lipoproteins in the plasma have an essential part in the development of atherosclerosis, a condition characterized by the accumulation of plaque on arterial walls.

Certain plaques can become unstable and rupture into the bloodstream, leading to severe heart attacks. Furthermore, lipoproteins can become harmful due to various chemical changes, and if these changes persist, they can lead to negative

consequences. Phospholipids in lipoproteins are particularly susceptible to oxidative modifications, which have been associated with inflammatory responses linked to atherosclerosis.

Consequently, Lp-PLA₂ has been identified as a significant factor in cardiac events [6,7]. Lp-PLA₂ is also recognized as a valuable molecular biomarker for coronary artery disease (CAD) [8,9]. Therefore, elevated Lp-PLA₂ levels can predict future CAD incidents in healthy older adults, regardless of other risk factors such as type 2 diabetes [10,11].

Moreover, Lp-PLA₂ can also aid in predicting the long-term onset of peripheral arterial disease. This study explored the connection between biochemical parameters and cardiovascular risk factors that affect Lp-PLA₂ levels in individuals, both with and without coronary artery disease.

METHODS

Study design and subjects

This study is a cross-sectional analysis that included 181 adults, aged 35 to 83 years, who underwent clinical and biochemical examinations at the National Heart Center in Tajoura from 2017 to 2019. The research received approval from the ethical committee at Tajoura Teaching Hospital. The data collected from the hospital were registered and securely stored in a standardized questionnaire, which included comprehensive information such as medical history, personal history, medication history, and results from physical examinations, including weight, height, and blood pressure. Participants were excluded based on the following criteria: (1) individuals younger than 35 years or older than 85 years; (2) pregnant women; and (3) individuals who had not fasted for 10 to 12 hours before laboratory investigations, as this could influence metabolic test results. The diagnosis of (CAD) was defined by vascular stenosis greater than 50% in the left main artery, left anterior descending artery, left circumflex artery, or right coronary artery. The following clinical indicators were considered in diagnosing CAD: (i) typical angina pectoris symptoms; (ii) ischemic electrocardiogram (ECG) changes, such as ST-segment depressions or new left bundle branch block; (iii) pathological Q waves observed on the ECG; (iv) imaging evidence of new loss of viable myocardium wall movements or new regional wall motion abnormalities, such as hypokinesia or akinesia; and (v) positive stenosis detected through coronary angiography.

Laboratory measurements

The clinical chemistry workup included fasting blood glucose, total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides (TG), and High-sensitivity-CRP were measured by COBAS INTEGRA 400 plus analyser (Roche Diagnostics; Basle, Switzerland). Lp-PLA₂ with quantitative test measured by commercial Human Lp-PLA₂ ELISA kit (provided by Elabscience Biotechnology). This ELISA kit is used as the Sandwich-ELISA principle according to manufacturer instructions.

Body height and weight were measured using scales and calibrated meters, respectively. BMI was estimated using the following formula: body weight (kg)/height (m²), the cut-off point was 25 kg/m², Blood pressure was measured after a 15-minute rest, and the subject was seated using an automated sphygmomanometer which was placed on the right arm. The mean arterial pressure was calculated using the following equation: $(2/3) \times$ diastolic pressure + $(1/3) \times$ systolic pressure. Diabetes mellitus was defined as fasting glucose \geq 126 mg/dl estimated on 2 occasions. Metabolic syndrome was defined as subjects who had three or more of the following criteria: (i) BMI $>$ 25kg/m², (ii) hypertriglyceridemia: (TG) \geq 150 mg/dl (iii) low HDL level $<$ 50 mg/dl, (iv) hypertension in diabetic patients $>$ 130/ 80 mmHg while in healthy controls $>$ 140/ 90 mmHg

Statistical analyses

All variables obtained from the data collected were recorded in the questionnaire. Comparisons were conducted using SPSS version 24 for Windows (IBM Corporation, New York, USA) and GraphPad Prism version 6. The t-test and Fisher's exact test were utilized to analyse the correlation between Lp-PLA₂ and the most relevant risk variables in both patients with and without coronary artery disease across both genders. The cross-sectional relationships among hs-CRP, Lp-PLA₂, and other related metabolic factors associated with the risk of coronary artery disease were examined using logistic regression analysis. A p-value of $<$ 0.05 was considered statistically significant.

RESULTS

A total of 181 participants were included in the study as shown in Table (1), of whom (69.6%) were men and (30.4%) females. The mean age of participants without CAD was 53.7 years (SEM = 1.7 years) and with CAD was 61.9 years (SEM = 0.6), respectively. Patients with and without CAD were older (61.9 ± 0.6 vs 53.7 ± 1.7) and showed a higher prevalence of male patients (82.4%), with diabetes mellitus (65.6%), hypertension, (63.2%) and smoking only in males

(25.2%). In addition, patients with CAD showed higher levels of triglycerides (137.2 ± 7.6 vs 96.3 ± 7.7 mg/dl and hs-CRP (5.3 ± 0.3 vs 3.3 ± 0.3 mg/dl), and showed lower HDL levels (31.7 ± 0.9 vs 45.0 ± 2.3). Plasma Lp-PLA₂ levels were significantly higher in patients with CAD than in individuals without coronary disease (270 ± 9.1 versus 162.2 ± 11.8 ; $P < 0.0001$), as shown in table 1.

Table 1. The biochemical and clinical characteristics of 181 Libyan participants who underwent exercise ECG, echocardiography, and coronary angiography. Participants were either diagnosed with coronary artery disease (CAD) or found to have normal coronary angiography (without CAD)

Clinical and biochemical parameters	Females no CAD	Females + CAD	P value	Males no CAD	Males + CAD	P value
Number	33	22	-	23	103	-
Gender	18.2	12.2	-	12.7	56.9	-
Age	53.2 ± 2.2	64.2 ± 1.3	0.0004	54.4 ± 2.9	61.5 ± 0.7	0.0006
BMI	27.4 ± 0.9	32.0 ± 1.4	<0.0001	25.3 ± 0.9	27.9 ± 0.4	<0.0001
Smokers	0	0	-	17 (73.9%)	26 (25.2%)	-
Hypertension	12	30	-	6	67	-
DM2	10	17	-	6	65	-
On anti-lipid therapy	10	14	-	6	80	-
Duration of diabetes	8.3 ± 2.9	13.5 ± 1.8	<0.0001	10.0 ± 3.9	10.4 ± 0.8	>0.3
Arterial Pressure						
SBP	133.2 ± 4.9	145.9 ± 4.4	0.07	122.6 ± 4.5	134.8 ± 2.1	<0.02
DBP	79.9 ± 1.9	81.8 ± 2.6	<0.003	80.2 ± 2.5	81.6 ± 1.0	<0.0001
MAP	97.6 ± 2.7	103.2 ± 2.9	0.17	94.4 ± 3.0	99.3 ± 1.3	0.12
FBS	201.9 ± 18.8	190.4 ± 19	<0.04	174.2 ± 24	197.5 ± 11.8	<0.0001
HbA _{1c}	5.8 ± 0.3	8.1 ± 0.5	<0.0001	5.7 ± 0.4	7.6 ± 0.2	<0.0001
Lp-PLA ₂	155.6 ± 2.2	250.7 ± 3.5	<0.0001	171.7 ± 4.7	274.2 ± 1.0	<0.0001
hs-CRP	3.2 ± 0.1	5.8 ± 0.2	<0.0001	3.5 ± 0.1	5.3 ± 0.03	<0.0001
Lipoprotein						
Total cholesterol	171.3 ± 8.0	164.1 ± 8.8	>0.5	149.9 ± 7.7	152.3 ± 4.4	>0.8
LDL	83.1 ± 3.7	81.8 ± 5.2	>0.8	82.7 ± 4.5	84 ± 2.5	>0.8
HDL	43.4 ± 2.6	35.6 ± 2.5	<0.05	47.1 ± 4.1	30.9 ± 0.9	<0.0001
LDL/HDL ratio	2.0 ± 0.1	2.4 ± 0.2	>0.05	2.0 ± 0.2	2.8 ± 0.1	<0.0009
Triglyceride	102.6 ± 12.4	140.9 ± 13.7	<0.05	87.4 ± 6.1	137.5 ± 8.8	<0.01
TyG index	$.46 \pm 0.06$	4.8 ± 0.08	<0.05	4.5 ± 0.05	4.8 ± 0.04	0.0009

This study emphasizes the variation in plasma Lp-PLA₂ levels between middle-aged individuals (< 60 years) and those over 60 years old, both with and without CAD. This observation indicates a potential clinical application for Lp-PLA₂ measurements in identifying individuals at risk for CAD who might not be detected through traditional methods. Specifically, plasma Lp-PLA₂ concentrations are higher in the older age group (> 60 years) compared to those younger than 60, with a significance level of $P < 0.003$, as shown in figure 1.

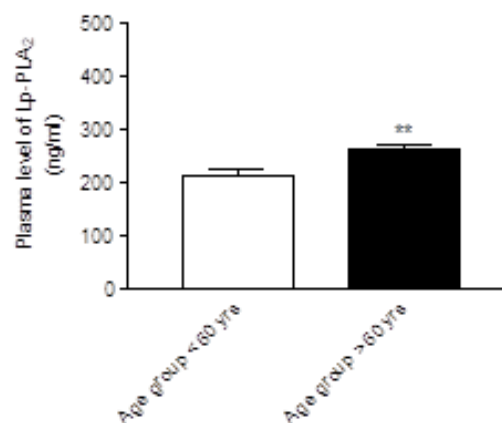


Figure 1. The mean plasma concentration of Lp-PLA₂ in groups below and above 60 years old. There is a statistical difference between age groups below and above 60 years old and plasma LpPLA₂ level with $P < 0.003$.

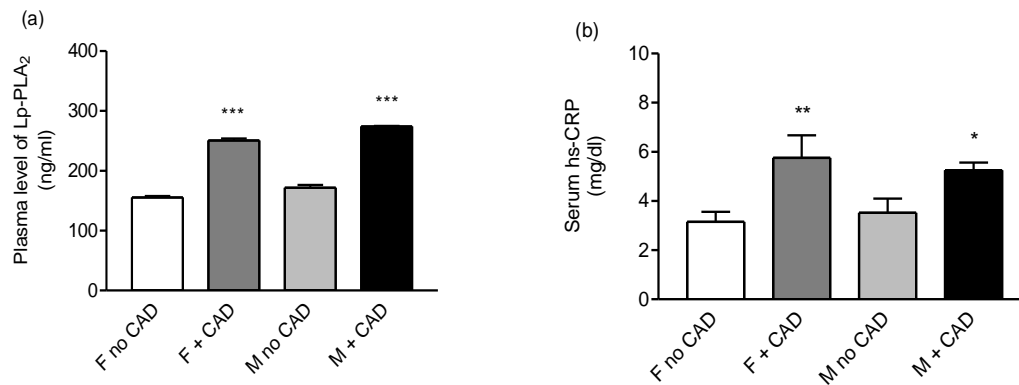


Figure 2. The levels of plasma Lp-PLA₂ and serum hs-CRP are statistically significant in patients with CAD compared to individuals without CAD in both genders, as depicted in figures (a) and (b). The data are presented as mean ± SEM.

The triglyceride glucose (TyG) index acts as an alternative measure for insulin resistance. Insulin resistance is a crucial contributor to the development of metabolic syndrome, which is significantly more prevalent in patients with coronary artery disease (CAD) compared to those without it, as shown in Figure (2).

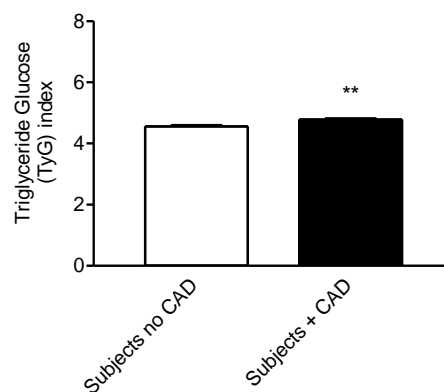


Figure 3. Triglyceride glucose index (TyG) of plasma Lp-PLA₂ and serum hs-CRP are statistically significant in patients with CAD compared to individuals without CAD, as illustrated in figures (a) and (b). The data are presented as mean ± SEM.

DISCUSSION

A total of 181 individuals participated in this study, with 69.6% being men and 30.4% women. This cross-sectional study examined the relationship between Lp-PLA₂, biochemical markers of atherosclerosis, and clinical characteristics. Conducted among the Libyan population, the research evaluated the correlation between Lp-PLA₂ levels and various risk factors, including lipid profiles, diabetes mellitus, age, gender, body mass index (BMI), hypertension, and inflammatory marker such as high-sensitivity C-reactive protein (hs-CRP).

Importantly, Lp-PLA₂ levels, measured using a sandwich ELISA kit, showed stable results and good repeatability [12,13]. The investigation revealed that women were more likely than men to be overweight or obese, regardless of the presence of CAD. Additionally, a study by Seyfarth et al. identified a positive correlation between BMI and Lp-PLA₂ activity [14]. Consequently, in patients with metabolic syndrome, a higher BMI may lead to changes in plasma Lp-PLA₂ concentrations [15].

Elevated plasma levels of Lp-PLA₂ have been found in patients with coronary artery disease (CAD) in both men and women. Conversely, the lower Lp-PLA₂ levels observed in females could be linked to estrogen secretion, as research indicates that premenopausal women generally have lower levels compared to menopausal women. For instance, a study by Yoshimura et al. [16]; reported a 26% decrease in Lp-PLA₂ levels in menopausal women just two weeks after beginning estrogen replacement therapy. Furthermore, Lp-PLA₂ levels were significantly higher in CAD patients than in those without CAD. Both groups can experience insulin resistance, which is especially pronounced in individuals with ischemic heart disease [17]. This insulin resistance can result in elevated insulin levels and a lower glucose disposal rate due to an increased requirement for endogenous or exogenous insulin, potentially leading to greater body weight,

obesity, and type 2 diabetes, particularly in older populations [18]. Additionally, an increase in serum levels of highly sensitive C-reactive protein (hs-CRP) has been observed in both male and female CAD patients, with a strong correlation found between hs-CRP and Lp-PLA₂ levels in individuals with coronary disease, helping to identify those at high risk for CAD [19].

The data from Cai et al. (2015) [20], suggest that there is no significant relationship between the plasma concentration and activity of Lp-PLA₂ and serum total cholesterol and LDL levels in patients with coronary artery disease (CAD). This lack of association may be influenced by confounding factors related to atherosclerosis risk, such as genetics, particularly since most patients had been on long-term anti-lipid treatment. In this study, the correlation between Lp-PLA₂ and serum total cholesterol and LDL was not [21,22].

Accumulating clinical evidence indicates a strong link between oxidized LDL and Lp-PLA₂ levels. Oxidized LDL promotes inflammation, damages the endothelium, and facilitates foam cell formation. Therefore, measuring oxidized LDL in the blood is often considered a more critical factor in the development of atherosclerosis than merely having high LDL levels.

A negative correlation was found between HDL and Lp-PLA₂ in women without CAD [14]. Additionally, increased serum triglyceride levels and the Triglyceride Glucose (TyG) index in CAD patients suggest the presence of insulin resistance and obesity related to metabolic syndrome in this study [23].

Plasma levels of Lp-PLA₂ are elevated in older individuals compared to younger ones, indicating that the elderly are more prone to insulin resistance and subacute inflammatory process. This increase in Lp-PLA₂ levels with age is a natural aspect of the aging process and tends to occur in certain individuals as they grow older. These findings are consistent with our results focused on adults over 60 [10,22,24,25].

CONCLUSION

In summary, Lp-PLA₂ has become a significant biomarker for evaluating cardiovascular risk, especially in older adults. Elevated Lp-PLA₂ levels are often linked to subacute inflammation, as evidenced by high hs-CRP levels. These elevated hs-CRP levels reflect the underlying inflammatory processes associated with ischemic heart disease. This relationship suggests that both Lp-PLA₂ and hs-CRP may be useful indicators of the severity of coronary artery disease (CAD), aiding in early detection and potentially informing treatment strategies. As the population ages, understanding how these biomarkers relate to cardiovascular health is essential for improving patient outcomes and tailoring prevention efforts. Further research is needed to elucidate the mechanisms behind these associations and to evaluate their implications for clinical practice in managing CAD among older Libyan patients.

Conflicts of Interest

The authors declare no conflicts of interest.

REFERENCES

1. McMurray HF, Parthasarathy S, Steinberg D. Oxidatively modified low density lipoprotein is a chemoattractant for human T lymphocytes. *J Clin Invest.* 1993 Aug 1;92(2):1004–8.
2. Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD, et al. Third universal definition of myocardial infarction. *Eur Heart J.* 2012 Oct 1;33(20):2551–67.
3. Häkkinen T, Luoma JS, Hiltunen MO, Macphee CH, Milliner KJ, Patel L, et al. Lipoprotein-Associated Phospholipase A₂, Platelet-Activating Factor Acetylhydrolase, Is Expressed by Macrophages in Human and Rabbit Atherosclerotic Lesions. *Arterioscler Thromb Vasc Biol.* 1999 Dec;19(12):2909–17.
4. Silvestre-Roig C, De Winther MP, Weber C, Daemen MJ, Lutgens E, Soehnlein O. Atherosclerotic plaque destabilization: Mechanisms, models, and therapeutic strategies. Vol. 114, *Circulation Research.* 2014. p. 214–26.
5. Dalager MG, Böttcher M, Thygesen J, Andersen G, Bøtker HE. Different Plaque Composition and Progression in Patients with Stable and Unstable Coronary Syndromes Evaluated by Cardiac CT. *Biomed Res Int.* 2015;2015:1–9.
6. Greig FH, Kennedy S, Spickett CM. Physiological effects of oxidized phospholipids and their cellular signaling mechanisms in inflammation. *Free Radic Biol Med.* 2012 Jan;52(2):266–80.
7. Khuseynova N, Imhof A, Rothenbacher D, Trischler G, Kuelb S, Scharnagl H, et al. Association between Lp-PLA₂ and coronary artery disease: Focus on its relationship with lipoproteins and markers of inflammation and hemostasis. *Atherosclerosis.* 2005 Sep;182(1):181–8.
8. Cojocaru M, Cojocaru IM, Silosi I. Lipoprotein-associated phospholipase A₂ as a predictive biomarker of sub-clinical inflammation in cardiovascular diseases. *Maedica (Buchar).* 2010 Jan;5(1):51–5.
9. De Stefano A, Mannucci L, Tamburi F, Cardillo C, Schinzari F, Rovella V, et al. Lp-PLA₂, a new biomarker of vascular disorders in metabolic diseases. *Int J Immunopathol Pharmacol.* 2019 Jan 1;33.
10. Daniels LB, Laughlin GA, Sarno MJ, Bettencourt R, Wolfert RL, Barrett-Connor E. Lipoprotein-Associated Phospholipase

- A2 Is an Independent Predictor of Incident Coronary Heart Disease in an Apparently Healthy Older Population. *J Am Coll Cardiol.* 2008 Mar;51(9):913–9.
11. Fortunato J, Bláha V, Bis J, Štásek J, Andrys C, Vojáček J, et al. Lipoprotein-Associated Phospholipase A 2 Mass Level Is Increased in Elderly Subjects with Type 2 Diabetes Mellitus. *J Diabetes Res.* 2014;2014:1–6.
 12. Garg PK, McClelland RL, Jenny NS, Criqui M, Liu K, Polak JF, et al. Association of lipoprotein-associated phospholipase A 2 and endothelial function in the Multi-Ethnic Study of Atherosclerosis (MESA). *Vasc Med.* 2011 Aug 27;16(4):247–52.
 13. S. Ahmed M, Zhan Ji J, H. Meng Q. Lipoprotein-Associated Phospholipase A2: How Effective as a Risk Marker of Cardiovascular Disease and as a Therapeutic Target? *Inflamm Allergy - Drug Targets.* 2011 Aug 1;10(4):236–46.
 14. Seyfarth J, Reinehr T, Hoyer A, Reinauer C, Bächle C, Karges B, et al. Lipoprotein-associated phospholipase A2 activity in obese adolescents with and without type 2 diabetes. *J Inherit Metab Dis.* 2018 Jan 13;41(1):73–9.
 15. Gong H ping, Du Y meng, Zhong L na, Dong Z qiang, Wang X, Mao Y jun, et al. Plasma Lipoprotein-associated Phospholipase A2 in Patients with Metabolic Syndrome and Carotid Atherosclerosis. *Lipids Health Dis.* 2011 Dec 19;10(1):13.
 16. Yoshimura T, Ohshige A, Maeda T, Ito M, Okamura H. Estrogen replacement therapy decreases platelet-activating factor-acetylhydrolase activity in post-menopausal women. *Maturitas.* 1999 Mar;31(3):249–53.
 17. Kendall DM, Sobel BE, Coulston AM, Harmel ALP, McLean BK, Peragallo-Dittko V, et al. The insulin resistance syndrome and coronary artery disease. *Coron Artery Dis.* 2003 Jun;14(4):335–48.
 18. Leite MM, Dutra MT, da Costa MVG, Funghetto SS, Silva A de O, de Lima LR, et al. Comparative evaluation of inflammatory parameters and substitute insulin resistance indices in elderly women with and without type 2 diabetes mellitus. *Exp Gerontol.* 2021 Jul;150:111389.
 19. García-Estévez DA, Araújo-Vilar D, Fiestras-Janeiro G, Saavedra-González Á, Cabezas-Cerrato J. Comparison of Several Insulin Sensitivity Indices Derived from Basal Plasma Insulin and Glucose Levels with Minimal Model Indices. *Horm Metab Res.* 2003 Jan;35(1):13–7.
 20. Cai A, Li G, Chen J, Li X, Li L, Zhou Y. Increased serum level of Lp-PLA2 is independently associated with the severity of coronary artery diseases: a cross-sectional study of Chinese population. *BMC Cardiovasc Disord.* 2015 Dec 26;15(1):14.
 21. Caslake MJ, Packard CJ, Robertson M, Cooney J, Nelson JJ, Ford I, et al. Lipoprotein-associated phospholipase A2, inflammatory biomarkers, and risk of cardiovascular disease in the Prospective Study of Pravastatin in the Elderly at Risk (PROSPER). *Atherosclerosis.* 2010 May;210(1):28–34.
 22. Huang Y, Wu Y, Yang Y, Li W, Lu J, Hu Y. Lipoprotein-associated phospholipase A2 and oxidized low-density lipoprotein in young patients with acute coronary syndrome in China. *Sci Rep.* 2017 Nov 23;7(1):16092.
 23. Ballantyne CM, Hoogeveen RC, Bang H, Coresh J, Folsom AR, Heiss G, et al. Lipoprotein-Associated Phospholipase A 2, High-Sensitivity C-Reactive Protein, and Risk for Incident Coronary Heart Disease in Middle-Aged Men and Women in the Atherosclerosis Risk in Communities (ARIC) Study. *Circulation.* 2004 Feb 24;109(7):837–42.
 24. Kolodgie FD, Burke AP, Skorija KS, Ladich E, Kutys R, Makuria AT, et al. Lipoprotein-Associated Phospholipase A 2 Protein Expression in the Natural Progression of Human Coronary Atherosclerosis. *Arterioscler Thromb Vasc Biol.* 2006 Nov;26(11):2523–9.
 25. Dong L, Qu X, Hu ZG, Peng X, Wang Y, Miao Q, et al. Lipoprotein-associated Phospholipase A2 is Associated with Angiographic Coronary Artery Disease and Coronary Artery Risk Factors in the Elderly. *Int J Gerontol.* 2015 Jun;9(2):82–6.

العلاقة بين التغيرات الكيميائية الحيوية ومستوى البلازما للفوسفوليبيز A₂ المرتبط بالبروتين الدهني لدى المرضى المصابين وغير المصابين بأمراض الشرايين التاجية: دراسة مقطعية

بهاء الدين بن محمود^{1*}، عبد المنعم الفلاح²، خلود السوكني¹

¹ قسم علوم الحياة، مدرسة العلوم الأساسية، الأكاديمية الليبية، طرابلس، ليبيا

² قسم الطب، كلية الطب البشري، جامعة طرابلس، طرابلس، ليبيا

المستخلص

مرض الشريان التاجي هو السبب الرئيسي للوفاة في جميع أنحاء العالم والهدف من الدراسة هو تقييم العلاقة بين فسفوليبيز A₂ المرتبط بالبروتين الدهني في البلازما وعوامل الخطر السريرية والكيميائية الحيوية لمرض الشريان التاجي لدى الأفراد الليبيين. ميز هذا التحقيق المقطعي بين المرضى الذين يعانون من مرض الشريان التاجي وأولئك الذين لا يعانون من مرض الشريان التاجي في كلا الجنسين على أساس التاريخ السريري، تخطيط القلب القياسي، تخطيط القلب بالإجهاد، تخطيط صدى القلب ثنائي الأبعاد، وتصوير الأوعية التاجية. تم جمع العديد من بيانات المؤشرات البيوكيميائية. تم تحديد مستويات فسفوليبيز A₂ المرتبط بالبروتين الدهني في البلازما بواسطة مقايصة الامتصاص المناعي المرتبط بالإنزيم. تم جمع 181 فرداً في هذه الدراسة، منهم 125 مصاباً بمرض الشريان التاجي و56 شخصاً بدون مرض الشريان التاجي. كان تركيز فسفوليبيز A₂ المرتبط بالبروتين الدهني في البلازما أعلى بشكل ملحوظ في المرضى الذين يعانون من مرض الشريان التاجي مقابل الأفراد الذين لا يعانون من مرض الشريان التاجي (9.2 ± 270 مقابل 11.8 ± 162.2 نانو غرام / مل؛ P < 0.0001 على التوالي)، وخاصة بين المرضى الذين يخضعون لتطعيم مجازة الشريان التاجي مقابل المرضى الذين يعانون من مرض الشريان التاجي. كانوا على العلاج الطبي والتدخل التاجي عن طريق الجلد (P < 0.0001)، على التوالي. بالإضافة إلى ذلك، كان تركيز البلازما لفسفوليبيز A₂ المرتبط بالبروتين الدهني أعلى في المرضى الذين تزيد أعمارهم عن 60 عاماً مقارنةً بالمرضى الذين تقل أعمارهم عن 60 عاماً (12.6 ± 207.9 مقابل 10.4 ± 260.1؛ P < 0.002). ارتبط مستوى تركيز فسفوليبيز A₂ المرتبط بالبروتين الدهني في البلازما بشكل مستقل بمرض الشريان التاجي، وهو علامة التهابية مرتبطة بشكل كبير ببروتينات C التفاعلية، والفئة العمرية الأكبر من 60 عاماً لدى المرضى الليبيين.

الكلمات الدالة: مرض الشريان التاجي، متلازمة التمثيل الغذائي، بروتين سي التفاعلي عالي الحساسية، داء السكري، البروتين الدهني المرتبط بالفوسفوليبيز A₂