P- ISSN 1991-8941 E-ISSN 2706-6703 *Journal of University of Anbar for Pure Science (JUAPS)* Open Access
2025,(19), (01):32 – 38

# Effect of Oxidative Stress and Dyslipidemia in Iraqi Patients with Hypertension

**Amna Adil Mohammed[1]*, Maryam I. Salman[1], Mohammed Ismael Dawood[2]**

[1]Department of Biology, College of Sciences, University of Anbar, Ramadi, Iraq
[2]Head of Department of Medicine, College of Medicine, University of AL Fallujah, Iraq
*E-mail (corresponding author): amn22s1003@uoanbar.edu.iq

**ARTICLE INFO**

Received: 30 / 05 /2024
Accepted: 15/ 08 /2024
Available online: 10/ 06 /2025

**DOI: 10.37652/juaps.2024.150394.1267**

**Keywords:**
***hypertension, lipid profile, oxidative stress, malondialdehyde***



**ABSTRACT**

Hypertension is a dangerous health issue mostly due to its asymptomatic nature. The present study was designed to assess malondialdehyde (MDA) as an indicator for oxidative stress and lipid profiles in patients with hypertension from Anbar, Iraq. It included 90 males and females aged between 30–50 years as participants (50 patients with hypertension and 40 healthy people as controls). The patients were presenting for internal medicine consultations at Al-Ramadi Teaching Hospital. After blood sample collection and serum separation, MDA levels were measured by using sandwich enzyme-linked immunosorbent assay. Lipid variables were measured by employing Chemistry Analyzer Smart-150. Results revealed that patients with hypertension had significantly higher ($P \leq 0.001$) systolic and diastolic blood pressure than controls. MDA levels in patients were significantly higher ($P \leq 0.001$) than in controls. The results also revealed significantly higher ($P \leq 0.022$) triglyceride and VLDL levels, as well as significantly higher ($P \leq 0.003$) cholesterol and low-density lipoprotein cholesterol levels, in patients than in controls. However, high-density lipoprotein cholesterol levels were significantly lower in patients than in controls ($P \leq 0.003$). In conclusion, the results showed an elevated level of oxidative stress in addition to a clear disorder in lipid profiles in patients with hypertension. This study indicates that oxidative stress and dyslipidemia have potential roles in hypertension development.

## Introduction

Hypertension is closely associated with hyperlipidemia and is a threat that combines with a wide range of cardiovascular events [1]. Its prevalence has increased annually. The number of persons with hypertension worldwide was 590 million (equal to 14.5%) in 1975. This number later amplified to 1.13 billion (equal to 15.3%) in 2015. The number of people with hypertension is expected to increase to 1.56 billion by 2025 [2]. In Iraq, hypertension ranks as the sixth most common cause of mortality, with a frequency ranging from 35.6% to 40%. In 2015, 6535 fatalities were attributed to hypertension [3]. The imbalance between reactive oxygen species (ROS) generation and antioxidant defenses may lead to oxidative damage. ROS cause lipid oxidation, which leads to malondialdehyde (MDA) formation [6].

*Corresponding author at: Department of Biology, College of Sciences, University of Anbar, Ramadi, Iraq
**ORCID: https://orcid.org/0009-0007-5235-9372**
**Tel: +964 7825650281**
**Email:** amn22s1003@uoanbar.edu.iq

In living organisms, MDA may cross-link proteins, nucleic acids, and other macromolecules. It can also severely impair the functioning of enzymes involved in the respiratory chain complex and mitochondria. A large body of research has shown that blood MDA is now a reliable biomarker of oxidative damage levels that can be used in vitro and in vivo [4,5].

Blood MDA concentrations are elevated in hypertensive animal models and human bodies due to increased oxidative stress and excessive ROS generation. A symptom of hypertension is an increase in lipid peroxidation products, such as MDA, due to an increase in free radical production; an increase in $O_2^{\bullet-}$ generation or a decrease in nitric oxide (NO) production could facilitate the improvement in functional arterial spasms [6].

Dyslipidemia refers to abnormal amounts of lipids and lipoproteins in the blood, including high levels of total cholesterol (TC, ≥200 mg/dL), triglycerides (TG, >150 mg/dL), high-density lipoprotein cholesterol

(HDL-C, <40 mg/dL), and low-density lipoprotein cholesterol (LDL-C, >100 mg/dL) [7]. These excessive lipids then build up on the walls of arteries, especially when endothelial dysfunction or injury is present. Dyslipidemia exacerbates endothelial dysfunction, which affects the lining of the arteries or the endothelium; the production of NO, a molecule that aids in regulating vascular tone and safeguards against oxidative stress and inflammation, further reduces [8].

Hypertension is considered a hidden killer due to its silent symptoms and causes the deaths of numerous people all over the world. Therefore, studying some of the physiological changes associated with this disease is necessary. Given that many studies have suggested that dyslipidemia and oxidative stress might play a major role in the pathogenesis and progression of hypertension, the aim of the present study is to evaluate the lipid peroxidation indicator MDA and lipid profiles, which include TC, TG, HDL, LDL, and VLDL, in a sample of individuals with hypertension.

## Materials and Methods

### Study design

A total of 90 males and females participated in this study. They comprised a patient group of 50 individuals with hypertension and a control group of 40 healthy individuals. Individuals participating in the research ranged in age from 30 years to 50 years. The study period was December 2023–April 2024. Hypertension was defined as systolic blood pressure (SBP) ≥ 140 mm Hg and/or diastolic blood pressure (DBP) ≥ 90 mm Hg [9].

### Inclusion criteria

Patients had a clinical diagnosis of hypertension.

### Exclusion criteria

Patients were excluded if they had diabetes mellitus, had heart diseases, had a history of smoking, were pregnant, or had vascular diseases.

### Ethics and approval statement

The ethical approval committee of the University of Anbar, Ramadi, Iraq, approved all study procedures that entailed collecting blood samples.

### Collection of blood samples

Blood samples were obtained through venipuncture after a 10 h overnight fast. A total of 5 mL of blood was drawn by using a disposable syringe. The drawn blood was then placed in a white tube and allowed to stand at room temperature (18 °C–25 °C) to induce blood clotting, then centrifuged for 5 min at 3500 rpm. The resulting serum was dispensed into white tubes and kept in a deep freezer at −18 °C until they were used.

### Biochemical analyses

Serum MDA was determined by using a sandwich enzyme-linked immunosorbent assay technology kit (Melsin Medical Co., Limited, CAT No. EKHU-0372). Serum cholesterol (TC), TG, and HDL were measured by employing Chemistry Analyzer Smart-150 (GenoTEK, Canada) according to the manufacturer's instructions. LDL was calculated by using the formula given by Friedewald:

LDL cholesterol (mg/dL) = TC − HDL cholesterol − (triglycerides/5).

VLDL was calculated by using the formula proposed by Wilson:

VLDL cholesterol = 0.2 × triglycerides [10].

### Statistical analysis

All obtained results were subjected to Student’s t test to identify the differences between the means of the examined variables. $P \leq 0.05$ and $P \leq 0.01$ were considered to indicate statistical significance. Statistical Package for Social Science software version 25 was utilized [11].

## Results and Discussion

### Blood pressure

The results revealed a significant increase ($P \leq 0.001$) in SBP (166.00 ± 11.909 mm Hg) and DBP (102.10 ± 5.810 mm Hg) in patients with hypertension compared with those in controls (119.75 ± 8.161 mm Hg, 79.75 ± 6.299 mm Hg) (Figs 1 A and B).

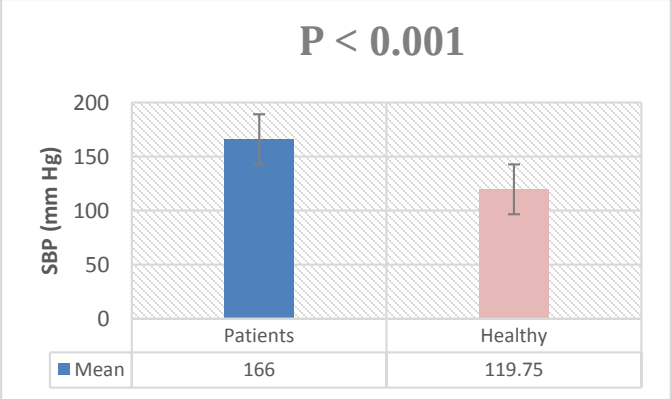


**Figure 1: A. SBP levels in patients and controls**

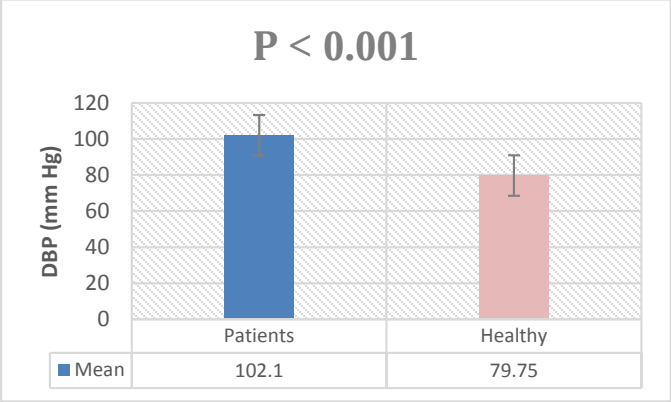


**Figure 1: B. DBP levels in patients and controls**

Hypertension is a medical disorder characterized by increased blood pressure in the arteries, surpassing 140 over 90 mm Hg [12]. It is not only the primary risk factor that may be modified to reduce the risk of cardiovascular disease (CVD), but it also serves as a measure of coronary artery disease severity [13]. The etiology of hypertension is often idiopathic for the majority of people. Hypertension is categorized as primary or essential, which affects more than 90% of hypertensive patients. Primary hypertension is a condition that cannot be cured. However, it can be effectively controlled via appropriate therapy, which may involve lifestyle modifications and medication use [14]. Less than 10% of people with hypertension have secondary hypertension. The main cause of secondary hypertension is often linked to kidney dysfunction, such as chronic kidney disease or renovascular disease. Compared with primary hypertension, secondary hypertension typically manifests abruptly and frequently results in elevated blood pressure levels [15].

**MDA**

The mean value of MDA was significantly higher ($P \leq 0.001$) in the patient group than in the control group. The mean MDA value in the hypertensive group was 10.745 ± 2.5388 nmol/mL and that in the control group was 7.655 ± 0.7119 nmol/mL (Fig 2).

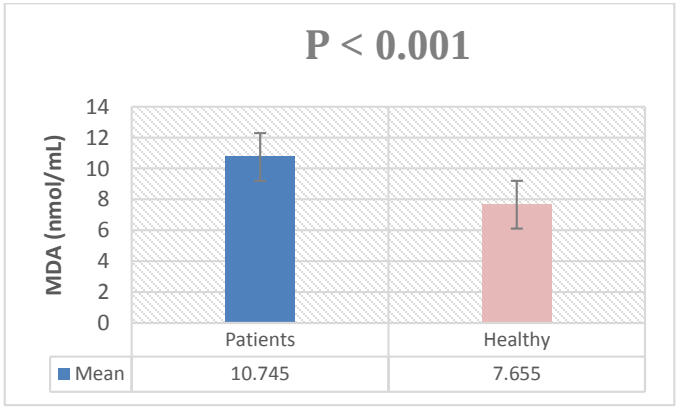


**Figure 2: MDA levels in patients and controls**

Numerous studies have revealed a substantial correlation between MDA levels and hypertension [16,17,18, &19].

Oxidative stress is the damage that occurs when ROS overcome antioxidant defenses. It promotes the thickening and narrowing of the vascular lumen; damages the endothelium; weakens endothelium-dependent vascular relaxation; and increases vascular contractile action. All these effects illustrate the influence of oxidative stress on the development of hypertension [19].

Free radicals have diverse roles in vascular redox systems in patients with hypertension. The present study demonstrated that MDA levels, which are a marker of lipid peroxidation, were higher in the hypertensive group than in the control group. Essential hypertension is characterized by an elevated MDA level or lipid peroxidation and an imbalance in antioxidant status. This finding indicates that oxidative stress plays an important role in the development of essential hypertension and its associated vascular damage [20].

Research has previously demonstrated the importance of oxidative damage in hypertensive individuals. First, ROS can damage the structure and function of endothelial cells. Second, they can break

down NO, which widens blood vessels [4]. Third, they can change how endothelial cells use eicosanoid molecules. Finally, they can change LDL levels and primary hypertension [4].

**Lipid profile**

The mean value of cholesterol in the hypertensive group was 184.4 ± 42.17 mg/dL and that in the control group was 162.62 ± 23.29 mg/dL (Fig 3). The results showed that the mean value of cholesterol was significantly higher (P ≤ 0.003) in the patient group than in the control group.

The TG level in the hypertensive group was 152.18 ± 59.72 mg/dL, whereas that in the control was 127.80 ± 38.94 mg/dL. A significant difference was found between the two groups (P < 0.22) (Fig 4).

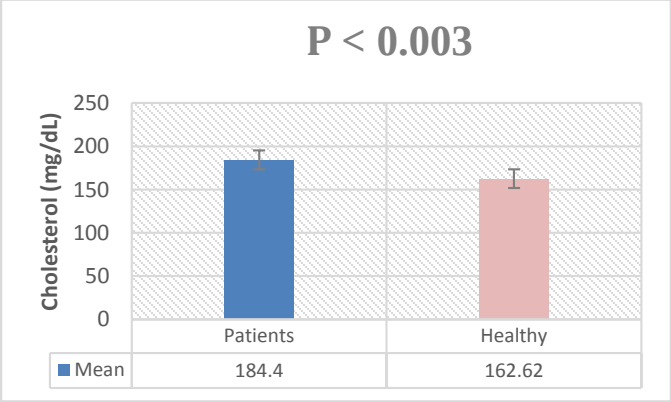


**Figure 3: TC levels in patients and controls**

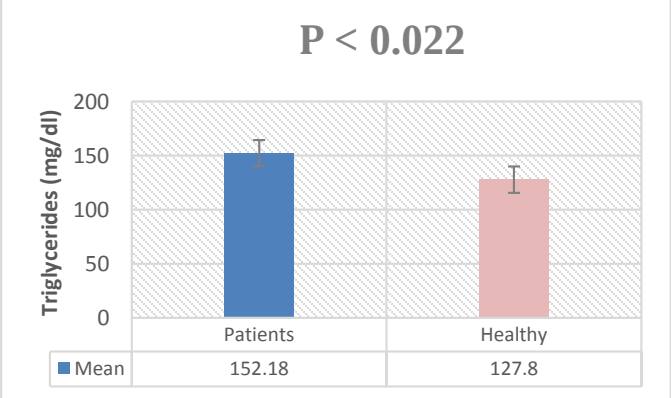


**Figure 4: TG levels in patients and controls**

HDL levels in the hypertensive group (33.58 ± 7.54 mg/dL) were significantly lower (P ≥ 0.003) than those in the control group (41.27 ± 14.01 mg/dL) (Fig 5). The LDL level in hypertensive group was 120.40 ± 33.82 mg/dL, whereas that in the control group had significantly increased to 102.60 ± 22.32 mg/dL (P < 0.004) (Fig 6). The VLDL level in the hypertensive group was 30.43 ± 11.94 mg/dL, whereas that in the control group had significantly increased to 25.56 ± 7.78 mg/dL (P < 0.022) (Fig 7).

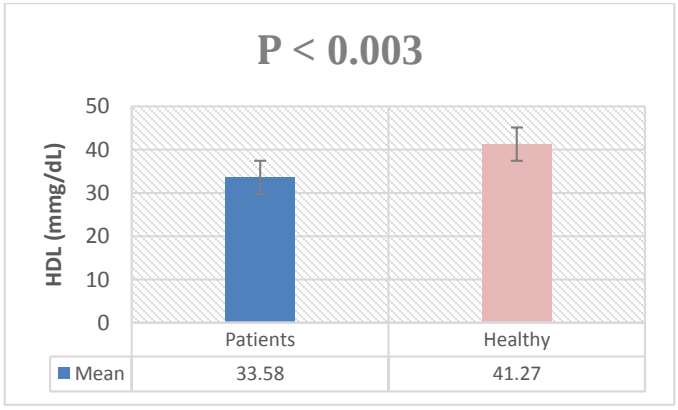


**Figure 5: HDL levels in patients and controls**

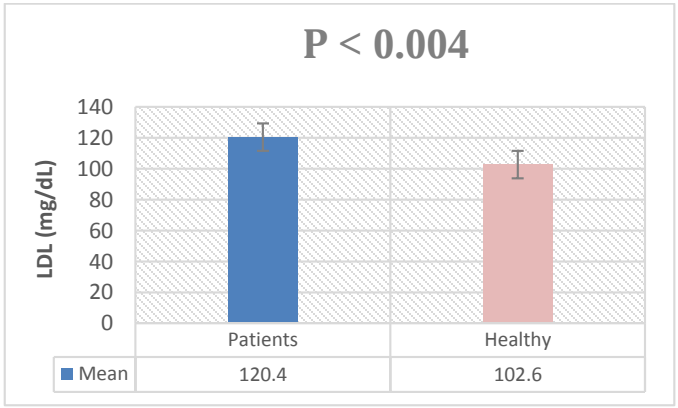


**Figure 6: LDL levels in patients and controls**

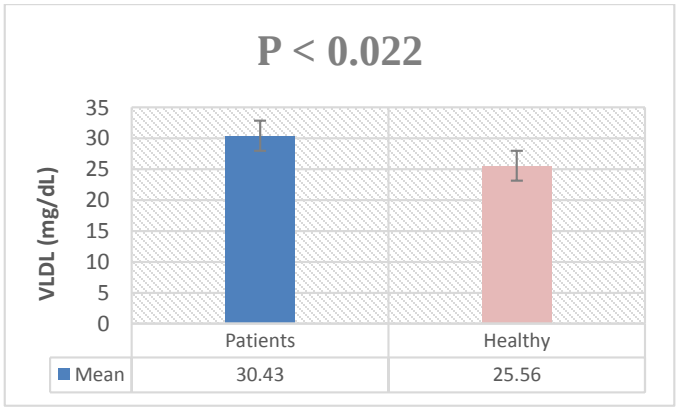


**Figure 7: VLDL levels in patients and controls**

Many previous studies have revealed remarkable occurrences of dyslipidemia and incidences of hypertension [21,22,23,&24]. These conditions share

similar underlying processes in terms of their pathophysiology [25]. Dyslipidemia is a common cardiovascular risk factor that contributes substantially to CVD and mortality [24].

Dyslipidemia may initially hinder the function of the endothelium, change the equilibrium between the relaxing and contracting components produced from the endothelium, and decrease the generation of NO [26]. This change can result in the malfunctioning of endothelial cells and disruptions in the control of blood pressure [13].

The influence of hyperlipidemia on vascular endothelial damage is closely correlated with oxidized LDL, which triggers the occurrence of atherosclerosis mostly by causing the damage and dysfunction of endothelial cells [26].

Furthermore, individuals with dyslipidemia would experience endothelial damage, leading to the impairment of vasomotor activity and disruption of vasoconstriction control. Consequently, this condition would contribute to an additional elevation in blood pressure, establishing a self-perpetuating cycle [27&28]. Within the renin–angiotensin–aldosterone system, angiotensin II induces hypertension and atherosclerosis by activating angiotensin type 1 receptors. This activation leads to enhanced lipid absorption in cells, increased free radical formation, and blood vessel constriction [29]. Dyslipidemia causes irregularities in the fluidity, permeability, and enzyme activity of the cell membrane. All these mechanisms may participate in the pathogenesis of hypertension [28]

## Conclusions

Hypertension is a complex condition with multiple causes. Therefore, studying the risk factors linked to hypertension is crucial for managing it effectively. Individuals with hypertension have markedly elevated concentrations of TG, TC, HDL, LDL, and VLDL compared with healthy individuals. These lipids play an important role in the development of CVD and other related conditions. The present study found that serum MDA levels increased in patients with hypertension, indicating the role of oxidative stress in the pathogenicity of hypertension. People with hypertension are warned about the need to reduce their fat intake and undergo periodic lipid and MDA tests. Further studies on MDA and lipid profiles in hypertension with large sample sizes and long durations are highly recommended.

## Reference

[1] Wu H, Yu Z, Huang Q (2022). Characteristics of serum lipid levels in patients with hypertension: a hospital-based retrospective descriptive study. BMJ Open. 3;12(6):e054682. doi: 10.1136/bmjopen-2021-054682. PMID: 36547874; PMCID: PMC9171285.

[2] Forouzanfar MH, Liu P, Roth GA, Ng M, Biryukov S, Marczak L, Alexander L, Estep K, Hassen Abate K, Akinyemiju TF, Ali R, Alvis-Guzman N, Azzopardi P, Banerjee A, Bärnighausen T, Basu A, Bekele T, Bennett DA, Biadgilign S, Catalá-López F, Feigin VL, Fernandes JC, Fischer F, Gebru AA, Gona P, Gupta R, Hankey GJ etal. (2017). Global Burden of Hypertension and Systolic Blood Pressure of at Least 110 to 115 mm Hg, 1990-2015. JAMA. 2017 Jan 10;317(2):165-182. doi: 10.1001/jama.2016.19043. Erratum in: JAMA. 2017 Feb 14;317(6):648. PMID: 28097354.

[3] Yehya Abdul Hasan, M., Alfarttoosi, A., Abdul Hasan, M., Tawfeeq, W. F., & Alfarttoosi, A. J. (2021). Screening for Hypertension among Adults Attending Primary Health Care Centers. Iraqi Med J, 67(1), 20–25. https://www.researchgate.net/publication/357839177.

[4] Huang, Y., Chen, H., Liu, Q., Hu, J., Hu, D., Huang, Z., Xu, Z., & Wan, R. (2023). Obesity difference on association blood malondialdehyde level and diastolic hypertension in the elderly population: a cross-sectional analysis. European Journal of Medical Research, 28(1), 1–8. https://doi.org/10.1186/s40001-022-00983-7.

[5] Ana M, Briones, Rhian M, Touyz.Oxidative Stress and Hypertension: Current Concepts Current Hypertension Reports. 2010;12(2):135-142.

[6] Alam, R., Ahsan, H., & Khan, S. (2023). The Role of Malondialdehyde (MDA) and Ferric Reducing Antioxidant Power (FRAP) in Patients with Hypertension. Molecular and Cellular Biomedical Sciences, 7(2), 58. https://doi.org/10.21705/mcbs.v7i2.288

[7] Karadimas, T., & Meier, H. C. S. (2024). Association Between Coexisting Hypertension, Dyslipidemia, and Elevated C-Reactive Protein with Cardiovascular Disease in the Health and Retirement Study. University of Michigan Undergraduate Research Journal, 17(0), 159–174. https://doi.org/10.3998/umurj.5512.

[8] Diaba-Nuhoho, P., & Amponsah-Offeh, M. (2023). Metabolic Dysregulation in Cardiovascular Diseases: A Beat on Cardiometabolic Diseases. Current Trends in the Diagnosis and Management of Metabolic Disorders, October, 219–234. https://doi.org/10.1201/9781003384823-13.

[9] Whelton PK, Carey RM, Aronow WS. ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American heart association Task force on clinical practice guidelines. *J Am Coll Cardiol* 20172018;71:e127–248.

[10] Khawaja TM, Shah SHA, Subhan Z, Mehmood I, M.& Shah M (2023). Effect of Losartan on Oxidative Stress and Lipid Profile in Patients with Essential Hypertension. Int.j.pathol.2023;21(1): 27 – 32.

[11] Daniel,W.W.; and C.L. Cross. (2018). Biostatistics: a foundation for analysis in the health sciences.

[10]Edition. John Wiley & Sons, Inc.. Library of Congress Cataloging-in-Publication Data. ISBN 978-1-118-30279-8.

[12] Forouzanfar MH, Liu P, Roth GA, et al.. Global burden of hypertension and systolic blood pressure of at least 110 to 115 MM Hg, 1990-2015. JAMA 2017;317:165–82. 10.1001/jama.2016.19043

[13] Cheng, W., Zhuang, J., & Chen, S. (2022). Dyslipidemia and the Prevalence of Hypertension: A Cross-Sectional Study Based on Chinese Adults Without Type 2 Diabetes Mellitus. Frontiers in Cardiovascular Medicine, 9(July), 1–12. https://doi.org/10.3389/fcvm.2022.938363.

[14] Olin, B. R., & Pharm, D. (2018). Hypertension : The Silent Killer : Updated JNC-8 Guideline Recommendations.

[15] Saseen J.J., & MacLaughlin E.J. (2017). Hypertension. DiPiro J.T., & Talbert R.L., & Yee G.C., & Matzke G.R., & Wells B.G., & Posey L(Eds.), Pharmacotherapy: A Pathophysiologic Approach, 10e. McGraw-Hill Education. https://accesspharmacy.mhmedical.com/content.aspx?bookid=1861&sectionid=146233698.

[16] Hassan, A. A., & Sayyah, S. G. (2023). Oxidative Stress Marker Malondialdehyde and Glutathione Antioxidant in Hypertensive Patients. 2(1), 31–36.

[17] Verma, M. K., Jaiswal, A., Sharma, P., Kumar, P., & Singh, A. N. (2019). Oxidative stress and biomarker of TNF-α, MDA and FRAP in hypertension. Journal of Medicine and Life, 2019(3), 253–259. https://doi.org/10.25122/jml-2019-0031.

[18] Armas-Padilla, M. C., Armas-Hernández, M. J., Sosa-Canache, B., Cammarata, R., Pacheco, B., Guerrero, J., Carvajal, A. R., Hernández-Hernández, R., Israili, Z. H., & Valasco, M. (2007). Nitric oxide and malondialdehyde in human hypertension. American Journal of Therapeutics, 14(2), 172–176. https://doi.org/10.1097/01.pap.0000249914.75895.48 . [19] Kumar M, Agrawal BK, Mishra A, Meena BS. (2020). A COMPARATIVE STUDY OF MDA LEVEL AS A MARKER OF OXIDATIVE STRESS IN HYPERTENSIVE PATIENTS AND CONTROLS. *International Journal of Medical Science and Diagnosis Research (IJMSDR)*. 4(9):3-5. DOI: https://doi.org/10.32553/ijmsdr.v4i9.669.

[20] Yadav, D. R., Yembarwar, N. K., Guddetwar, S. G., & Geel, V. R. (2022). To Study the Biochemical Changes For Prediction of Essential Hypertension. International Journal of Medical and Biomedical Studies, 6(4), 1–7. https://doi.org/10.32553/ijmbs.v6i4.2496.

[21] Thomas F, Bean K, Guize L, et al. (2002). Combined effects of systolic blood pressure and serum cholesterol on cardiovascular mortality in young (<55 years) men and women. Eur Heart J.23:528–35. 10.1053/euhj.2001.2888.

[22] McDonald M, Hertz RP, Unger AN, et al. (2009) Prevalence, awareness, and management of hypertension, dyslipidemia, and diabetes among United States adults aged 65 and older. J Gerontol A Biol Sci Med Sci.64:256–63. 10.1093/gerona/gln016.

[23] Bachir Cherif A, Bennouar S, Bouamra A, et al.( 2018) Prevalence of diabetes and dyslipidemia in

hypertensive patients in the area of Blida (Algeria). Annales de Cardiologie et d'Angéiologie 2018;67:198–203. 10.1016/j.ancard. 04.015

[24] Yu S, Yang H, Guo X, et al.. Prevalence of dyslipidemia and associated factors among the hypertensive population from rural northeast China. BMC Public Health 2015;15:1152. 10.1186/s12889-015-2486-7.

[25] Tang, N., Ma, J., Tao, R., Chen, Z., Yang, Y., He, Q., Lv, Y., Lan, Z., & Zhou, J. (2022). The effects of the interaction between BMI and dyslipidemia on hypertension in adults. Scientific Reports, 12(1), 1–7. https://doi.org/10.1038/s41598-022-04968-8.

[26] Zhang Y-Z, Wang L, Zhang J-J, et al.. Vascular peroxide 1 promotes ox-LDL-induced programmed necrosis in endothelial cells through a mechanism involving β-catenin signaling. Atherosclerosis 2018;274:128–38.

[27] ChanCahill, P. A., & Redmond, E. M. (2016). Vascular endothelium - Gatekeeper of vessel health. Atherosclerosis, 248, 97–109.https://doi.org/10.1016/j.atherosclerosis.2016.03.007.

[28] Hurtubise, J., McLellan, K., Durr, K., Onasanya, O., Nwabuko, D., & Ndisang, J. F. (2016). The different facets of dyslipidemia and hypertension in atherosclerosis. Current Atherosclerosis Reports, 18(12). https://doi.org/10.1007/s11883-016-0632-z.

[29] Yue YL, Guo Y, Wang JH. Association of non-enzymatic glycosylation and oxidative stress with angiosclerosis in elderly hypertensive patients. Practical Geriatrics 2014.

[30] Dobrzyńska I, Szachowicz-Petelska B, Weresa J, et al.. Changes in physicochemical properties of kidney cells membrane as a consequence of hypertension and treatment of hypertensive rats with FAAH inhibitor. Chem Biol Interact 2019;299:52–8. 10.1016/j.cbi.2018.11.024.

# الإجهاد التأكسدي واضطراب الدهون لدى مرضى ارتفاع ضغط الدم العراقيين

**آمنة عادل محمد، مريم ابراهيم سلمان، محمد اسماعيل داود**

[1]قسم علوم الحياة، كلية العلوم، جامعة الانبار، الرمادي، العراق. [2]كلية الطب، جامعة الانبار، الفلوجة العراق.

**الخلاصة:**

يعتبر ارتفاع ضغط الدم مشكلة صحية كبيرة، ويرجع ذلك في الغالب إلى عدم ظهور اعراض للمرض اذ ان غالبية الأفراد يصابون بارتفاع ضغط الدم دون أن يدركوا وجوده. شملت الدراسة 90 مشاركا (50 مريضا مصابا بارتفاع ضغط الدم و40 شخصا سليما كتجربة ضابطة)، ذكورا وإناثا تتراوح أعمارهم بين (30–50) سنة. حضر المرضى الى استشارية الطب الباطني في مستشفى الرمادي التعليمي. أظهرت النتائج زيادة معنوية ($P \leq 0.001$) لضغط الدم الانقباضي والانبساطي عند مرضى ارتفاع ضغط الدم مقارنة بالمجموعة الضابطة. كان متوسط قيمة MDA أعلى معنويا ($P \leq 0.001$) في مجموعة المرضى مقارنة بالمجموعة الضابطة. كما أظهرت النتائج وجود ارتفاع معنوي ($P \leq 0.022$) في مستوى الدهون الثلاثية والبروتينات الدهنية واطئة الكثافة جدا عند المرضى مقارنة بالاصحاء كما لوحظ وجود ارتفاع معنوي في مستوى الكولستيرول والبروتينات الدهنية واطئة الكثافة ($P \leq 0.003$)، في حين أظهرت النتائج حدوث انخفاض معنوي في مستوى البروتين الدهني عالي الكثافة ($P \leq 0.003$) عند مرضى الضغط. اوضحت النتائج ارتفاع مستوى الاجهاد التأكسدي اضافة الى الاضطراب الواضح في صورة الدهون عند مرضى ارتفاع ضغط الدم، النتائج تشير بوضوح الى الدور المحتمل للإجهاد التأكسدي واضطراب الدهون في الإصابة بمرض ارتفاع ضغط الدم.