



Evaluation of a Selection of Lipid Profile and Kidney Function in Hypertensive Patients in Ramadi City, Western Iraq.

O. M. Mohammed ^(*), H. L. Abdulhadi ⁽²⁾

⁽¹⁾Department of Medical Laboratories Techniques, College of Health and Medical Technology, University of Al Maarif, Al Anbar, Iraq.

⁽²⁾ Department of Biology, College of Education for Pure Sciences, University of Anbar, Ramadi, Iraq.

Article information

Article history:

Received: July 31, 2025

Revised: October 15, 2025

Accepted: October 21, 2025

Available online: January 01, 2026

Keywords:

Cardiovascular Risk

Hypertension

Kidney Function

Lipid Profile

Uric Acid

Correspondence: Othman Mueen
Mohammed

othman.mueen@uoa.edu.iq

Abstract

The most common modifiable cardiovascular disease (CVD) risk element is hypertension, and CVD has a wide variety of conditions that include coronary artery disease, heart failure, cerebrovascular accidents, myocardial infarction, atrial fibrillation and peripheral arterial disease, among chronic kidney disease or CKD and cognitive impairment. Hypertension is the leading cause of death and disability worldwide. This cross-sectional research carried out in Ramadi, Iraq, assessed 50 cases of hypertension and 25 healthy individuals to determine the demographic trends, biochemical indicators, and cardiovascular risk factors. The prevalence of hypertension was diagnosed higher in women (68%) than in men (32%), and most common in the age category of 50 60 (56%), as compared to the regional trends of ageing. There was a high hereditary connection whereby 68 per cent of the patients claimed a family history of hypertension. Blood pressure measures showed that systolic pressure (16.72 ± 1.70 mmHg vs. 11.50 ± 0.65 mmHg) and diastolic pressure (9.89 ± 1.15 mmHg vs. 7.68 ± 0.61 mmHg) highly increased in the patients compared with experiences of controls ($P < 0.0001$). Biochemical studies have revealed an increased amount of uric acid in hypertensive patients (5.28 ± 1.35 mg/dL vs. 3.94 ± 0.52 mg/dL) but not of urea. The results highlight how genetic, metabolic, and lifestyle influencing factors are interconnected in causing hypertension in western Iraq and the importance of monitoring uric acid levels, controlling lipids, and the need to have targeted community health promotion programs to aid in reducing cardiovascular risks.

DOI: [10.33899/jes.v35i1.60278](https://doi.org/10.33899/jes.v35i1.60278), ©Authors, 2026, College of Education for Pure Science, University of Mosul.

This is an open access article under the CC BY 4.0 license (<http://creativecommons.org/licenses/by/4.0/>).

1. Introduction

Cardiovascular diseases and premature mortality issues are caused mostly by hypertension as a leading cause across the globe (Mills et al., 2020). Regardless of the use of antihypertensive pharmacological treatment, the average arterial pressure (BP) of people all around the world has remained stationary or reduced by a little bit during the course of the four decades. Ironically, the number of people with high blood pressure has risen, especially in low- and middle-income nations (LMICs) [1]. In 2010, the world projection was that the number of people living with hypertension was about 31.1 per cent of the total adult population, which translates to 1.39 billion people. It was higher in LMICs and reached 31.5 per cent (1.04 billion people) as compared to 28.5 per cent (349 million people) in high-income countries [1]. Differences in the risks of hypertension, including the intake of high sodium and low potassium, obesity, alcohol consumption, sedentary life, and low dietary habits, can likely lead to the differences in the occurrence of the condition within a region [1]. Irrespective of this increased burden, there is a low level of awareness, management, and control of blood pressure, specifically in LMICs.

Moreover, not much comprehensive research has been done concerning the impacts of hypertension on the economy. Thus, researchers have an urgent need to conduct investigations on the cost-effective prevention and management interventions in economically deprived sub-groups [1].

Etiological approach at the etiological level, the majority of hypertension cases (90-95 per cent) can be associated with essential or primary hypertension, which is explained by multifactorial interactions between genes and the environment [2]. A history of family is also a persistent risk factor, as heritability estimates have been between 35% and 50% in most research [2]. Genome-wide association studies (GWAS) have found almost 120 gene locations linked to BP regulation and altogether clarified about 3.5% of the sum of the trait variance [3]. Such discoveries open the door to precision medicine, and it is in relation to hypertension that omics-based diagnostics and patient-specific therapy may become game changers [2]. In extremities, some cases of hypertension are genetically pure monogenic; in that the is completely explained by the mutation of one gene. These can be illustrated by Liddle syndrome, glucocorticoid-remediable aldosterone's and mutation of the Phosphodiesterase 3A gene (cGMP-inhibited phosphodiesterase 3A), coding phosphodiesterase that is inhibited by cGMP [4]. Secondary hypertension, on the contrary, has other disorders as a cause, e.g., primary aldosteronism, pheochromocytoma, or renal artery stenosis, in which case treatment of the primary disorder can restore blood pressure to normal [3].

There is also an indication, as in another recent study, that uric acid (UA) is an important factor in the pathogenesis of hypertension due to the effects of enhancing renal afferent arteriopathy, activation of RAAS, oxidative stress, systemic inflammation and endothelial dysfunction [5]. Serum urea, creatinine, uric acid, and electrolytes are biochemical tests commonly employed in measuring hypertension; they are better than the urine tests because they are easier to sample in medical facilities [2]. Moreover, hypertension (HTN) and dyslipidemia (DLIP) are frequently comorbid, and the reason may be found in having common pathophysiological pathways, e.g., being obese and having endothelial dysfunction ([6]. Their synergetic interaction sharply increases the chance of cardiovascular events and deaths to a greater extent than each of the above [7]. These co-occurrences require combined clinical interventions in risk assessment and treatment.

1.2: Study goal

1. Weigh up cholesterol (TC) and triglycerides (TG) in patients with hypertension and compare these values with the control population to determine how much they get in the way
2. Measure the levels of serum uric acid and urea to evaluate kidney functions among hypertensive patients.
3. Study the influence of the period of hypertension on parameters of lipid and kidney function and identify possible trends.
4. Identify correlations between high lipid levels and reduced kidney functions toward the comprehension of the organ damage by hypertension.

2. Materials and Methods

2.1 Study Design, Area and Population:

This study was conducted in the city of Ramadi, located in the western part of Iraq, to investigate the prevalence and risk factors associated with high blood pressure among the local population. The research employed a cross-sectional comparative study design, comparing a group of patients diagnosed with high blood pressure to a control group without any history of hypertension.

Ramadi, the capital city of the Anbar Governorate, was selected for its diverse population and varying socioeconomic conditions. The city's healthcare facilities provided a suitable environment for conducting this research.

A total of 75 participants were included in the study, divided into two groups: 50 patients diagnosed with high blood pressure (Patient Group) and 25 healthy individuals without any history of hypertension (Control Group).

2.2 Inclusion and Exclusion Criteria for the Study :

2.2.1 Inclusion Criteria

Patient Group: Individuals aged 18 years and above diagnosed with high blood pressure by a certified healthcare professional

Control Group: Individuals aged 18 years and above with no history of hypertension or other cardiovascular diseases

2.2.2 Exclusion Criteria

Individuals who have secondary hypertension caused by specific conditions, such as renal artery stenosis or endocrine disorders, are excluded.

Individuals with other severe medical conditions that could affect blood pressure readings, such as severe renal disease or heart failure

3.2 Samples collection and preparation

About 5 ml of venous blood was collected from healthy individuals and hypertensive patients (infected and uninfected with Hypertension) and placed into gel tubes containing clotting activators, and then centrifuged at 3000 rpm for 5 minutes to separate serum and stored at -20°C to determine biochemical parameters

4.2 Measured parameters (Determination of biochemical parameters)

The levels of four biochemical parameters, including urea, Uric acid, Triglycerides and cholesterol in all the serum specimens were measured using the Mindray BS-240 Fully Automatic Biochemistry Analyser[8].

5.2 Statistical Analysis:

Data were analyzed using SPSS software version 25. We used descriptive statistics to analyze the demographic and clinical characteristics. Comparisons between groups were carried out using the independent t-test for continuous variables and the chi-square test for categorical variables. A p-value less than 0.05 was considered significant[9].

3. Results And Discussion

1.3. Distribution of patients with hypertension according to sex groups:

The current study recorded the highest number of cases of high blood pressure in females, 34 (68%), and the lowest among males, 16 (32%), as shown in Table 1.

Table 1: Shows the distribution of patients according to sex.

	Frequency	Percent(%)	Valid Percent	Cumulative Percent
Male	16	32.0	32.0	32.0
Female	34	68.0	68.0	100.0
Total	50	100.0	100.0	

Based on the findings of this research, the researchers concluded that 68 per cent of patients who were female had hypertension compared to 32 per cent of male patients in the study sample of 50 patients. The trend might be indicative of some latent biological, behavioural, and sociocultural factors which may be involved in the provision of the observed variation in the prevalence of hypertension according to sex. Biologically, men generally may have higher risks of hypertension at an earlier age, but later in women, risks rise sharply with the post-menopausal period. This is likely attributed to the changes in the level of hormonal presence, especially estrogen, which was earlier known to be protective in terms of cardiovascular conditions [10]. The hypertensive females in this study is relatively higher and thus could indicate a postmenopausal population in which women are more prone to high blood pressure[11]. Behavior and lifestyle issues could also be a factor.

The household and socioeconomic factors of women in some areas, including low- and middle-income countries, can lock out access to preventative healthcare, lack physical activity, or eat too many sodium and saturated fat products [1, 12]. Cultural beliefs in certain societies might restrict women from participating in routine exercises or to attend to the doctor early enough before hypertension takes a toll. Also, psychosocial stress, which women tend to have disproportionately because of caregiving roles and expectations in society, may be a factor as well. Prolonged stress has been proven to affect the autonomic neuro system and endocrine output and increase blood pressure in the long run [13]. There is also the need to take into consideration the probability of sampling bias or population dynamics. As an example, the prevalence would be distorted by adding weights to the sample in instances where women were likely to spend time at clinics or attend screening programs.

Thus, it is suggested that more research should be done on larger randomized populations to validate these views and explicate their applicability.

3.2 Distribution of patients with hypertension according to age groups:

The current study recorded the highest infection rate in the age group (50-60) at 56%, followed by the age group (41-50) at 22%, while the lowest rate was in the age group (20-30) at 4%, as shown in Table 2.

Table (2): Distribution of patients with hypertension according to age groups

Age Groups	Frequency	Percent	Valid Percent	Cumulative Percent
20-30	2	4.0	4.0	4.0
31-40	9	18.0	18.0	22.0
41-50	11	22.0	22.0	44.0
51-60	28	56.0	56.0	100.0
Total	50	100.0	100.0	

The findings in this research show that the 51 to 60 age group had the highest prevalence of hypertension (56 per cent) followed by the aged 41 to 50 (22 per cent). This tendency correlates with the vast epidemiological evidence that age becomes a significant non-modifiable risk factor of hypertension development [14]. Several physiological processes have been related to ageing, which in turn lead to increased blood pressure; these are arterial stiffness, endothelial dysfunction, and augmented peripheral vascular resistance [15]. Moreover, the appealing factor in older people is exposure to a great accumulation of lifestyle-related risk factors, as seen in poor diet, physical inactivity, and chronic stress. The fact that the hypertensive patients in the 20-30 age group comprises only 4 per cent is representative of the fact that hypertension prevalence rises gradually with age. Nevertheless, the use of hypertension at a younger age among adults is less prevalent, but it is increasingly being detected in relation to obesity, sedentary and hereditary factors [16]. This needs to highlight early life interventions and screening of BP in the populations, even in younger populations.

3.3. Distribution of patients with hypertension according to the presence of a genetic history.

The results of the current study show that the highest incidence of infection among patients was among people with a hereditary history of high blood pressure, reaching 68%, and the lowest among people with no hereditary history, at 32%. As shown in Table 3.

Table (3): Distribution of patients with hypertension according to hereditary history

Pressure Type	Frequency	Percent(%)	Valid Percent	Cumulative Percent
Hereditary	34	68.0	68.0	68.0
Non-hereditary	16	32.0	32.0	100.0
Total	50	100.0	100.0	

It was also found that a large proportion of hypertensive patients (68%) were positive on the family history of hypertension, and 32 % of the proportion did not. This corroborates the fact that heredity is important in the pathogenesis of high blood pressure. The model that has been proven through numerous studies is that essential hypertension is significantly heritable, i.e., genetic contribution (heritability) constitutes 35-50% [3]. The genetic predisposition is commonly associated with a number of mechanisms where salt sensitivity, maladaptive handling of sodium in the kidney, dysregulation of The renin angiotensin aldosterone system (RAAS) and vascular tone alteration are cited [4]. Moreover, people with a familial history are more susceptible to developing hypertension at a younger age, which sometimes mandates a screening at an early age and lifestyle changes that are more drastic. The influence of environmental risks, like high sodium consumption, obesity or inactivity on physical activity, is usually combined with the effects of hereditary factors since a gene-environment interplay scheme is observed in the development of hypertension [2]. These results support the medical evaluation of family history in the management of individuals, particularly when the primary prevention program involves high-risk patients.

3-4. Distribution of data between the two study groups according to systolic blood pressure.

The current study recorded statistically significant differences ($P < 0.05$) between the two study groups, as shown in Table 4.

Table 4: Distribution of data between the two study groups according to systolic blood pressure.

Parameter	Number of values	Mean \pm SD	t, df	P value
systolic pressure Patients mmHg	50	16.72 \pm 1.700	t=14.79, df=73	<0.0001****
systolic pressure control mmHg	25	11.50 \pm 0.6455		

**** Correlation is highly significant at the <0.0001 level ($p < 0.0001$)

The present experiment indicates the statistically significant difference in systolic blood pressure (SBP) between hypertensive patients and normal controls ($P < 0.0001$). The average SBP of patients was $16.72 \text{ mmHg} \pm 1.70$, as opposed to $11.50 \text{ mmHg} \pm 0.65$ of the control group. This difference is supported by the t-value ($t = 14.79$, $df = 73$), which is quite high. This result corresponds to the clinical meaning of hypertension since systolic blood pressure of 140 mmHg or more is among the main criteria of its diagnosis [17]. Being elevated, SBP is a significant risk factor of the development of the cardiovascular events, especially stroke, cardiac failure, and kidney disease. Additionally, it is noted that the systolic pressure is more significant as a risk factor as compared to the diastolic pressure in middle-aged adults and further in elderly folks because of hardening of the arteries [14]. This acute contrast between the patient and the control group demonstrates high diagnostic reliability of the SBP measures as well as the study design options to successfully establish the distinction between the normotensive and the hypertensive study groups [18].

3.5. Distribution of data between the two study groups according to diastolic blood pressure.

The current study recorded statistically significant differences ($P < 0.05$) between the two study groups, as shown in Table 5.

Table (5):. Distribution of data between the two study groups according to diastolic blood pressure.

Parameter	Number of values	Mean \pm SD	t, df	P value
diastolic pressure Patients mmHg	50	9.890 ± 1.153	$t=8.956$, $df=73$	$<0.0001^{****}$
diastolic pressure control mmHg	25	7.680 ± 0.6103		

**** Correlation is highly significant at the <0.0001 level ($p < 0.0001$)

On the same note, the difference between DBP in both groups was also found to be significant at $P < 0.0001$ as the patients had a mean DBP of $9.89 \pm 1.15 \text{ mmHg}$ with a control group having a mean of $7.68 \pm 0.61 \text{ mmHg}$ ($t = 8.956$, $df = 73$). Although there has been a certain degree of underemphasis on diastolic pressure, it is a very important indicator of vascular resistance and left ventricular performance. Alongside increasing the risks of coronary artery disease, kidney damage, and microvascular complications, elevated DBP ($\geq 90 \text{ mmHg}$) is linked to developing the risks in younger adults [19].

The present findings also prove the presence of hypertension according to the general guidelines and show that SBP and DBP are highly increased in hypertensive patients in comparison with healthy volunteers [20]. The data discussed indicates the significance of having a thorough blood pressure measurement in clinical practice since the high values in each element (and both, as well) might mean higher cardiovascular risk.

3.6. Estimation of biochemical parameters (kidney functions) between patients and control subjects in patients with hypertension.

The current study recorded statistically significant differences between the two study groups for the chemical parameter (uric acid) ($P < 0.05$), while the biochemical parameter (urea) did not record significant differences ($P < 0.05$), as shown in Table 6.

Table (6): Distribution of biochemical parameters between the two study groups with hypertension

parameter	Number of values	Mean \pm SD	t, df	P value
urea patients mg/dL	50	27.98 ± 11.55	$t=0.5602$, $df=73$	0.5770ns
urea control mg/dL	25	29.32 ± 3.966		
uric acid patients mg/dL	50	5.276 ± 1.354	$t=4.761$, $df=73$	$<0.0001^{***}$
uric acid control mg/dL	25	3.936 ± 0.5211		

*** Correlation is significant at the 0.001 level ($p < 0.001$)

3.7. Estimation of the lipid profile (cholesterol, triglycerides) between the two study groups in patients with hypertension

The current study recorded statistically significant differences between the patient and control groups for both variables related to the assessment of the lipid profile, as shown in Table 7.

Table (7): Distribution of data between the two study groups according to lipid profile

parameter	Number of values	Mean \pm SD	t, df	P value
cholesterol patients mg/dL	50	162.7 \pm 43.95	t=3.628, df=73	0.0005***
cholesterol control mg/dL	25	129.4 \pm 18.12		
triglycerides patients mg/dL	50	215.3 \pm 120.2	t=4.574, df=73	<0.0001***
triglycerides control mg/dL	25	104.6 \pm 13.48		

*** Correlation is significant at the 0.001 level ($p < 0.001$)

3.8 The relationship between weight, lipid profile, and kidney function, and what is between them.

According to the Spearman test, there is a positive relationship between weight and total cholesterol and a negative relationship with triglycerides. As for kidney function, there was a negative relationship between weight and variables, as shown in Table 8.

Table (8): Correlation between weight and variables

	Weight	T.C	T.G	UA	Urea
Weight	1.000	-.288*	.141	.017	.029
Sig. (2-tailed)	–	.042	.330	.909	.840
N	50	50	50	50	50
T.C	-.288*	1.000	.172	.127	.330*
Sig. (2-tailed)	.042	–	.231	.379	.019
N	50	50	50	50	50
T.G	.141	.172	1.000	.243	-.045
Sig. (2-tailed)	.330	.231	–	.089	.758
N	50	50	50	50	50
UA	.017	.127	.243	1.000	-.082
Sig. (2-tailed)	.909	.379	.089	–	.570
N	50	50	50	50	50
Urea	.029	.330*	-.045	-.082	1.000
Sig. (2-tailed)	.840	.019	.758	.570	–
N	50	50	50	50	50

* Correlation is significant at the 0.05 level (2-tailed).

Serum levels of urea and uric acid were used to estimate the state of kidney functioning in hypertensive individuals in the current research study. Although there was no statistically significant difference in the mean level of urea between patients and controls ($P = 0.5770$), a highly significant result in the mean level of uric acid in serum was recorded among hypertensive people ($P < 0.0001$). This observation depicts that the high levels of uric acid could be more related to hypertension compared to the urea levels. Hyperuricemia has been linked to the pathogenesis of primary hypertension, which may involve renal vasoconstriction, oxidative stress, inflammation and renin-angiotensin angiotensin aldosterone system (RAAS) [5, 21]. This renders uric acid as not only a hallmark but also a possible mediator of hypertension[21].

The insignificant change in the urea level can be related to maintained glomerular filtration in the patients in the first stages of hypertension or may be affected by protein intake in the diet and the state of hydration[22]. It is also indicative of the possibility of renal damage as a result of hypertension, having not reached the stage of obvious uremia among such a patient population[23]. Analysis of lipid profile showed that there was a significant increase in the level of total cholesterol and triglyceride (TG) in hypertensive patients as compared to controls ($P = 0.0005$ and $P < 0.0001$ respectively). The results

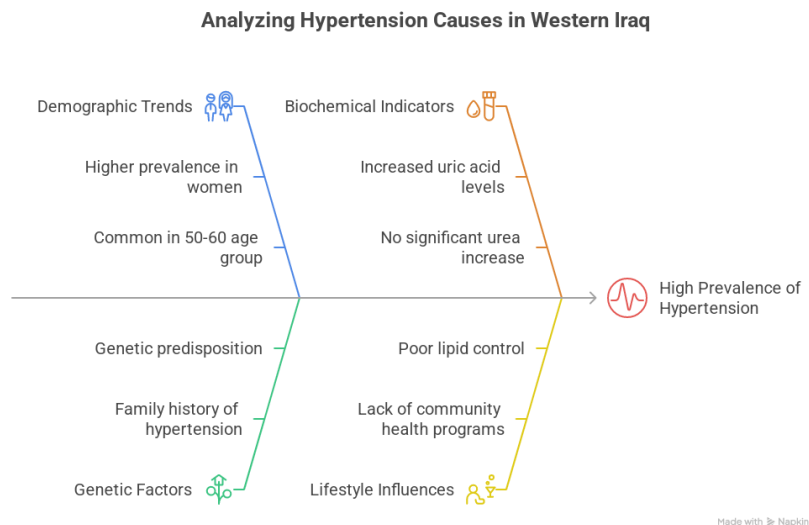
correspond to earlier studies that revealed a high connection between dyslipidemia and hypertension [6]. This comorbidity can be explained by comorbid pathophysiological processes such as:

- The dysfunction of the endothelium, Insulin resistance
- Long-lasting inflammation
- Obesity, which is an accumulation of fat in the stomach

The interaction of hypertension and dyslipidemia in a synergistic effect increases the risk of cardiovascular diseases and causes atherosclerosis, coronary artery disease, and stroke [7, 24]. In such a way, these findings underline the significance of thorough cardiovascular risk assessment in hypertensive patients, not only blood pressure but also lipid control[25].

The study applied Spearman's rho to investigate the correlation between body weight and cholesterol, triglycerides, and markers of kidney function (uric acid and urea). A strong negative correlation was obtained between weight and total cholesterol ($r = -0.288$, $P = 0.042$), which is not expected, as it is assumed that weight is a factor that influences high levels of cholesterol in the body. This might relate either to population-specific effects or drug effects and needs further investigation.

A correlation of positive, but non-significant correlation was found between weight and triglycerides ($r = 0.141$, $P = 0.330$) as described in the literature between hypertriglyceridemia and obesity. As far as functions of kidneys, there were no significant correlations between weight and levels of uric acid or urea levels indicating that till now, there is no association of body weight increase with renal function deterioration in this population. Interestingly, there was a strong positive correlation observed between total Cholesterol and urea ($r = 0.330$ / $P = 0.019$). It may indicate a common metabolic load or subclinical kidney failure, but a more specific kidney functional test (e.g., eGFR, creatinine) should be useful to explain such a relationship. Based on these results, we should remember the complicated interrelation between hypertension, lipid metabolism, renal biomarkers and body weight. A striking uptrend in uric acid, cholesterol and triglycerides in the patients with hypertension implies the multi-system effect of high blood pressure and the necessity of combining clinical intervention. The correlation analysis would be more elaborate because it indicates and demonstrates that not all variables always follow predictable patterns in various populations, i.e., a risk profiling of individuals will be necessary. Future works should be conducted on a bigger sample and other markers (e.g. HDL, LDL, creatinine)



4. Conclusions:

1. Demographics: Hypertension prevalence in Ramadi, Iraq, was higher in females (68%) and peaked in the 50–60 age group (56%).
2. Genetic Influence: 68% of hypertensive patients had a hereditary predisposition, aligning with global genetic risk studies.
3. Blood Pressure: Significant differences in systolic (16.72 vs. 11.50 mmHg) and diastolic (9.89 vs. 7.68 mmHg) pressures between patients and controls ($P < 0.0001$).
4. Biochemical Markers: Elevated uric acid in patients (5.28 vs. 3.94 mg/dL, $P < 0.0001$) but no significant urea difference.
5. Lipid Profile: Higher cholesterol (162.7 vs. 129.4 mg/dL) and triglycerides (215.3 vs. 104.6 mg/dL) in patients ($P < 0.001$).

6. Weight Correlations: Negative association between weight and total cholesterol ($\rho = -0.288$, $P = 0.042$), positive with triglycerides ($\rho = 0.141$, $P = 0.330$).

5. Acknowledgements

The authors would like to express their sincere gratitude to the staff of the College of Health and Medical Technology, University of Al Maarif, and the College of Education for Pure Sciences, University of Anbar, for their continuous support and encouragement throughout the course of this study. Special thanks are extended to all participants who contributed to the research and to the laboratories and health centers in Ramadi City for facilitating sample collection and analysis.

6. Declarations

6.1 Ethics approval and consent to participate

Not applicable.

6.2 Consent for publication

Not applicable.

6.3 Availability of Data and Materials

Data will be provided upon receiving a valid request.

6.4 Conflicts of interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

6.5 Funding

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

7. References

- [1] K. T. Mills et al., "Global Disparities of Hypertension Prevalence and Control: A Systematic Analysis of Population-Based Studies From 90 Countries," *Circulation*, vol. 134, no. 6, pp. 441–450, Aug. 2016, doi: 10.1161/CIRCULATIONAHA.115.018912.
- [2] S. Oparil et al., "Hypertension," *Nature Reviews Disease Primers*, vol. 4, art. no. 18014, Mar. 22 2018, doi: 10.1038/nrdp.2018.14.
- [3] G. B. Ehret et al., "The genetics of blood pressure regulation and its target organs from association studies in 342,415 individuals," *Nature Genetics*, vol. 48, no. 10, pp. 1171–1184, Oct. 2016, doi: 10.1038/ng.3667.
- [4] R. P. Lifton, A. G. Gharavi, and D. S. Geller, "Molecular mechanisms of human hypertension," *Cell*, vol. 104, no. 4, pp. 545–556, Feb. 23 2001, doi: 10.1016/S0092-8674(01)00241-0.
- [5] D. I. Feig, D. H. Kang, and R. J. Johnson, "Uric acid and cardiovascular risk," *New England Journal of Medicine*, vol. 359, no. 17, pp. 1811–1821, Oct. 23 2008, doi: 10.1056/NEJMr0800885.
- [6] S. M. Grundy, "Metabolic syndrome update," *Trends Cardiovasc Med*, vol. 26, no. 4, pp. 364–373, May 2016, doi: 10.1016/j.tcm.2015.10.004.
- [7] S. P. Pinney, A. Anyanwu, A. Lala, J. Teuteberg, N. Uriel, and M. J. J. O. T. A. C. O. C. Mehra, "Left Ventricular Assist Devices for Lifelong Support (vol 69, pg 2845, 2017)," *J. Am. Coll. Cardiol.*, vol. 70, no. 10, p. 1308, 2017, doi: 10.1016/j.jacc.2017.04.031.
- [8] C. Okulmus, N. I. Icil, O. Turkyilmaz, and Z. T. J. C. C. P. Yildirim, "Clinical chemistry reference intervals for swiss albino strain mice commonly used in scientific studies," *Compar. Clin. Pathol.*, vol. 2025, pp. 1–8, 2025.
- [9] A. Hazra and N. Gogtay, "Biostatistics Series Module 4: Comparing Groups - Categorical Variables," *Indian J. Dermatol.*, vol. 61, no. 4, pp. 385–392, Jul.–Aug. 2016, doi: 10.4103/0019-5154.185700.
- [10] J. F. Reckelhoff, "Gender differences in the regulation of blood pressure," *Hypertension*, vol. 37, no. 5, pp. 1199–1208, May 2001, doi: 10.1161/01.hyp.37.5.1199.
- [11] R. Lima, M. Wofford, and J. F. Reckelhoff, "Hypertension in postmenopausal women," *Curr Hypertens Rep.*, vol. 14, no. 3, pp. 254–260, Jun. 2012, doi: 10.1007/s11906-012-0252-8.

- [12] L. Trtica Majnarić, I. Martinović, Š. Šabanović, S. Rudan, F. Babič, and T. Wittlinger, "The effect of hypertension duration and the age of onset on CV risk factors expression in perimenopausal women," *Int. J. Hypertens.*, vol. 2019, p. 9848125, 2019, doi: 10.1155/2019/9848125.
- [13] T. M. Spruill, "Chronic psychosocial stress and hypertension," *Curr Hypertens Rep.*, vol. 12, no. 1, pp. 10–16, Feb. 2010, doi: 10.1007/s11906-009-0084-8.
- [14] S. S. Franklin et al., "Hemodynamic patterns of age-related changes in blood pressure. The Framingham Heart Study," *Circulation*, vol. 96, no. 1, pp. 308–315, Jul. 1 1997, doi: 10.1161/01.cir.96.1.308.
- [15] E. G. Lakatta and D. J. C. Levy, "Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises: Part I: aging arteries: a 'set up' for vascular disease," *Circulation*, vol. 107, no. 1, pp. 139–146, 2003, doi: 10.1161/01.CIR.0000048892.83500.58.
- [16] A. V. Chobanian et al., "Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure," *Hypertension*, vol. 42, no. 6, pp. 1206–1252, Dec. 2003, doi: 10.1161/01.HYP.0000107251.49515.c2.
- [17] P. K. Whelton et al., "2017 ACC/AHA Guideline for High Blood Pressure in Adults: Executive Summary," *Hypertension*, vol. 71, no. 6, pp. 1269–1324, Jun. 2018, doi: 10.1161/HYP.0000000000000066.
- [18] T. H. Jafar et al., "Integration of a multicomponent intervention for hypertension into primary healthcare services in Singapore: A cluster randomized controlled trial," *PLoS Medicine*, vol. 19, no. 6, p. e1004026, Jun. 2022. doi: 10.1371/journal.pmed.1004026
- [19] W. B. Kannel, T. Gordon, and M. J. Schwartz, "Systolic versus diastolic blood pressure and risk of coronary heart disease: the Framingham study," *Am. J. Cardiol.*, vol. 27, no. 4, pp. 335–346, Apr. 1971. doi: 10.1016/0002-9149(71)90428-0
- [20] G. Parati et al., "Home blood pressure monitoring: methodology, clinical relevance and practical application: a 2021 position paper by the Working Group on Blood Pressure Monitoring and Cardiovascular Variability of the European Society of Hypertension," *J. Hypertens.*, vol. 39, no. 9, pp. 1742–1767, Sep. 2021. doi: 10.1097/HJH.0000000000002922
- [21] L. Du et al., "Hyperuricemia and its related diseases: mechanisms and advances in therapy," *Signal Transduct. Target. Ther.*, vol. 9, no. 1, p. 212, Aug. 2024. doi: 10.1038/s41392-024-02002-9
- [22] A. Mihalache, L. Garneata, C. A. Mocanu, T.-P. Simionescu, and G. Mircescu, "Low-salt low-protein diet and blood pressure control in patients with advanced diabetic kidney disease and heavy proteinuria," *Int. Urol. Nephrol.*, vol. 53, no. 6, pp. 1197–1207, Jun. 2021. doi: 10.1007/s11255-020-02696-6
- [23] S. Udani, I. Lazich, and G. L. Bakris, "Epidemiology of hypertensive kidney disease," *Nat. Rev. Nephrol.*, vol. 7, no. 1, pp. 11–21, Jan. 2011. doi: 10.1038/nrneph.2010.154
- [24] C. Borghi, F. Fogacci, D. Agnoletti, and A. F. G. Cicero, "Hypertension and dyslipidemia combined therapeutic approaches," *High Blood Press. Cardiovasc. Prev.*, vol. 29, no. 3, pp. 221–230, May 2022. doi: 10.1007/s40292-022-00519-x
- [25] X. Hou et al., "Joint modifiable risk factor control and incident stroke in hypertensive patients," *J. Clin. Hypertens. (Greenwich)*, vol. 26, no. 11, pp. 1274–1283, Nov. 2024. doi: 10.1111/jch.14836

تقييم مجموعة مختارة من ملف الدهون ووظائف الكلى لدى مرضى ارتفاع ضغط الدم في مدينة الرمادي، غرب العراق.

عثمان معين محمد^(1*)، هيثم لطيف عبدالهادي⁽²⁾

⁽¹⁾ قسم تقنيات المختبرات الطبية، كلية التقنيات الصحية والطبية، جامعة المعارف، الأنبار، العراق
⁽²⁾ قسم علوم الحياة، كلية التربية للعلوم الصرفة، جامعة الأنبار، الرمادي، العراق

المستخلص:

يُعد ارتفاع ضغط الدم أكثر عوامل الخطر شيوعاً لأمراض القلب والأوعية الدموية القابلة للتعديل، وله مجموعة واسعة من الحالات، تشمل مرض الشريان التاجي، وقصور القلب، والسكتات الدماغية الوعائية، واحتشاء عضلة القلب، والرجفان الأذيني، وأمراض الشرايين الطرفية، بالإضافة إلى أمراض الكلى المزمنة (CKD) والضعف الإدراكي. ويُعتبر ارتفاع ضغط الدم السبب الرئيسي للوفاة والإعاقة في العالم. أجريت هذه الدراسة المقطعية في مدينة الرمادي بالعراق، حيث قُيِّمت 50 حالة من ارتفاع ضغط الدم و25 شخصاً سليماً لتحديد الاتجاهات الديموغرافية، والمؤشرات البيوكيميائية، وعوامل خطر الإصابة بأمراض القلب والأوعية الدموية. شُخِّص ارتفاع ضغط الدم لدى النساء (68%) أكثر من الرجال (32%)، وكان أكثر شيوعاً في الفئة العمرية 50-60 عاماً (56%)، مقارنةً بالاتجاهات الإقليمية للشيوخوخة. وُجد ارتباط وراثي قوي، حيث ادعى 68% من المرضى وجود تاريخ عائلي لارتفاع ضغط الدم. وأظهرت قياسات ضغط الدم ارتفاعاً كبيراً في ضغط الدم الانقباضي (16.72 ± 1.70 مم زئبق مقابل 11.50 ± 0.65 مم زئبق) وضغط الدم الانبساطي (9.89 ± 1.15 مم زئبق مقابل 7.68 ± 0.61 مم زئبق) لدى المرضى مقارنةً بتجارب المجموعة السيطرة (قيمة الاحتمال > 0.0001). وكشفت التحاليل الكيموحيوية عن زيادة في كمية حمض اليوريك لدى مرضى ارتفاع ضغط الدم (5.28 ± 1.35 ملجم/ديسيلتر مقابل 3.94 ± 0.52 ملجم/ديسيلتر)، ولكن ليس في اليوريا. تُسلط النتائج الضوء على كيفية ترابط العوامل الوراثية والأبضية ونمط الحياة في التسبب بارتفاع ضغط الدم في غرب العراق، وأهمية مراقبة مستويات حمض اليوريك، والتحكم في الدهون، والحاجة إلى برامج مُوجَّهة لتعزيز الصحة المجتمعية للمساعدة في الحد من مخاطر أمراض القلب والأوعية الدموية.