IRAQI Academic Scientific Journals
Samarra Journal of Pure and Applied Science
SJPAS
www.sjpas.com
p ISSN: 2663-7405
e ISSN: 2789-6838

Measuring the levels of aldosterone, angiotensin II, and some biochemical variables in hypertension patients' blood serum

Khalidah M. Abdulrazzaq*, Firas Tahir Mahir and Thaaer Fadhil Khalil
Department of Chemistry, College of Science, Tikrit University

https://doi.org/10.54153/sjpas.2025.v7i2.957

Article Information
Received: 07/07/2024
Revised: 01/08/2024
Accepted: 29/08/2024
Published: 30/06/2025

Keywords:
Hypertension, Aldosterone, Urea, Blood pressure.

Corresponding Author

E-mail: Khalidah.muwafaq@st.tu.edu.iq
Mobile:

Abstract

In this work, the levels of aldosterone, angiotensin II, and various biochemical variables were measured in the blood serum of people with high blood pressure and compared to healthy controls, aged between (45-60 years). The levels of aldosterone, angiotensin II, urea, creatinine, sodium, and potassium were estimated in high blood pressure patients. Statistical findings exhibited a noteworthy increase in aldosterone hormone and sodium rates in hypertension patients for both genders compared to the control group. Besides, the results displayed a substantial increase in the angiotensin II hormone and urea rate in male patients with hypertension compared to the control group; however, no considerable differences appeared in female patients compared to healthy people. The results exhibited a substantial decrease in the potassium rate. However, the results did not show a considerable difference in the creatinine and uric acid rates in both genders, individuals with high blood pressure, compared to the control group.

Introduction:

hypertension is a chronic medical condition resulting from hypertension in the arteries above its average level [1]. The prevalence increases dramatically with age, affecting approximately 6% of individuals aged 18 to 34 years but over 77% of those aged 75 years or older. High blood pressure is primarily responsible for 35% of all myocardial infarctions and strokes, as well as half of all episodes of congestive heart failure. Most of the causes of high blood pressure are unknown (essential hypertension); other causes are kidney disease, stenosis of the aorta, and myeloma. When high blood pressure causes symptoms in a severe form, the organs most affected are the brain, heart, and kidneys [2]. The renin hormone works to hydrolyze proteins and form angiotensin I (the angiotensin I protein has no biological effect and only appears to be transformed into angiotensin II) by the action of another enzyme secreted from the lung lining, which causes vasoconstriction and a subsequent increase in blood pressure. It is part of the renin-angiotensin-aldosterone system, which is the main target of drugs that lower blood pressure. Angiotensin II also stimulates the secretion of aldosterone, another hormone produced by the adrenal cortex. Aldosterone promotes sodium

retention in the distal nephron in the kidney, which causes fluid retention and, thus, high blood pressure [3, 4]. Renal function tests, including those measuring urea, creatinine, and uric acid, are crucial for diagnosing kidney disease, determining renal failure, tracking the course of the illness, and assessing treatment response [5]. Ammonia, which is released when amino acids are removed, is converted to urea in the liver [6]. The kidneys excrete a majority of the non-protein nitrogen, or urea, which is over 75%. The gastrointestinal system and skin also lose trace amounts of this nitrogen [7]. Measurements of urea are commonly accessible and recognized as a useful indicator of renal function [8]. Plasma creatinine concentration measurements are frequently utilized clinically as an indicator of kidney function. Creatinine is produced when muscle creatine breaks down; spontaneous, non-enzymatic water loss converts about 1-2% of the daily pool of muscle creatine to creatinine [9, 10].

This study aims to study a comparison between patients with hypertension and healthy controls, for both sexes, by estimating the hormones aldosterone and angiotensin II, kidney function and electrolytes.

Methods

Samples were collected at Tikrit Teaching Hospital from (60) persons with hypertension (24 males and 36 females) aged between (45-60 years). Thirty blood samples were also obtained from healthy persons of the same age group as a control group. This work was carried out by taking a sample of blood with a size of (5 ml) and leaving it for (15) minutes, then separating the blood serum from the clotted part using a centrifuge at a speed of (4000) rounds per minute (r.p.m.) for (10) minutes. Clear blood serum was withdrawn and kept in a freezer at 20 degrees Celsius until it was used to perform various analyses according to the requirements of the study.

An examination kit was utilized for aldosterone and angiotensin II, from the Chinese company Sun Long Biotech, based on the Sandwich-ELISA method in blood serum. Calibration curves were obtained to calculate the hormone levels and are shown in Figure (1) Calibration curve for aldosterone and Figure (2) Calibration curve for angiotensin II.

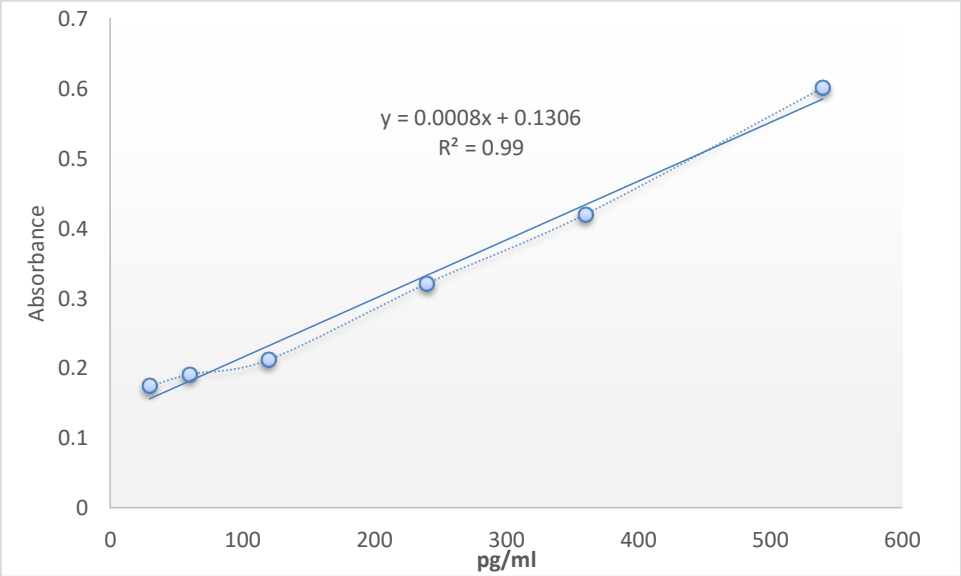

Fig. 1 *Standard curve of ALD*

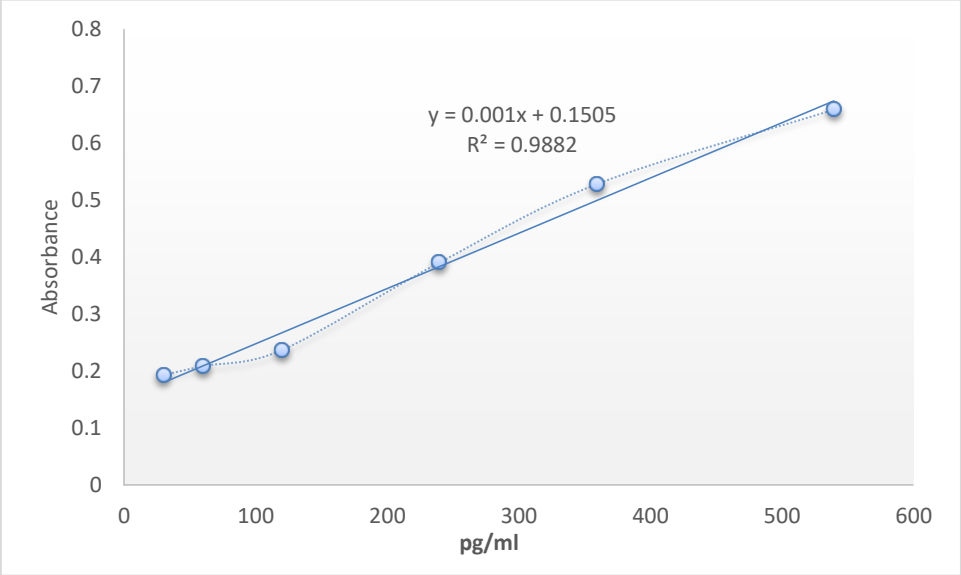

Fig. 2 *Standard curve of ANG-II*

The urea rate in blood serum was measured utilizing a ready-made analysis kit from the French company Biolabo [11]. The creatinine rate in blood serum was measured utilizing a ready-made analysis kit from the French company Biolabo [12]. The uric acid concentration in blood serum was measured utilizing a diagnostic kit from the French company Biolabo [13]. The sodium concentration in blood serum was measured utilizing the diagnostic kit from the Egyptian company Vitro SCINT, utilizing the colorimetric method [14]. The potassium concentration in blood serum was measured utilizing the diagnostic kit from Agappe of Switzerland utilizing the photometric turbidimetric method [15].

Statistical analysis: The results were statistically analysed using SPSS version 20, extracting the arithmetic mean and standard deviation, and the T-Test to compare between two groups. The significant differences for these groups were chosen at a probability level of $p<0.05$.

Result and Discussion

Table (1) shows the mean ± standard deviation of the levels of the hormones aldosterone and angiotensin II and some biochemical indicators according to sex in the blood serum of the samples under study.

Table 1: The blood serum's amount of biochemical variables of both male and female people with high blood pressure and their comparison with the control group

Parameter		Male	P value p≤0.05	Female	P value p≤0.05
		Mean ±SD N=24		Mean ±SD N=36	
ALD (pg/ml)	Patients	1189.79± 868	0.003	1208.95± 859	0.01
	Control	581.8±207.96		555.96±181	
ANGII (pg/ml)	Patients	557.68 ±390.7	0.04	542.9±557	NS
	Control	349±212		279.3±81.67	
UREA (mg/dl)	Patients	56±58.2	0.02	39±25.1	NS
	Control	26.5±7.26		30.5±5.9	
Creatinine(mg/dl)	Patients	1.018±1.038	NS	0.76±0.46	NS
	Control	0.72±0.127		0.73±0.15	
UA (mg/dl)	Patients	4.97±1.89	NS	4.9±2.1	NS

	Control	4.59±1.09		4.37±0.79	
Na (mmol/l)	**Patients**	153.7±7.17	0.001	150±9.2	0.004
	Control	141±4.8		144±4.5	
K (mmol/l)	**Patients**	3.3±0.56	0.001	2.9±0.66	0.001
	Control	4.3±0.46		4.2±0.46	

Table (1) and Figure (3) indicate the mean ± standard deviation of the aldosterone level, as it was for the group of patients and healthy individuals' values were (1189.79±868) pg/ml, (581.8±207.96) pg/ml, respectively, with a probability level of P≤0.003 for the male group .
For the female group, the aldosterone level values were (1208.95±859) pg/ml for patients and (555.96±181) pg/ml for healthy individuals at a probability level of P≤0.01. In the present study, both gender groups of patients with high blood pressure showed a significant increase, consistent with the previous study. [16]. Aldosterone is the primary hormone among the mineralocorticoid hormones, as it has a strength of (30-100) times more than the second hormone in sodium ion retrieval and, therefore, has an essential role in raising blood pressure [17].

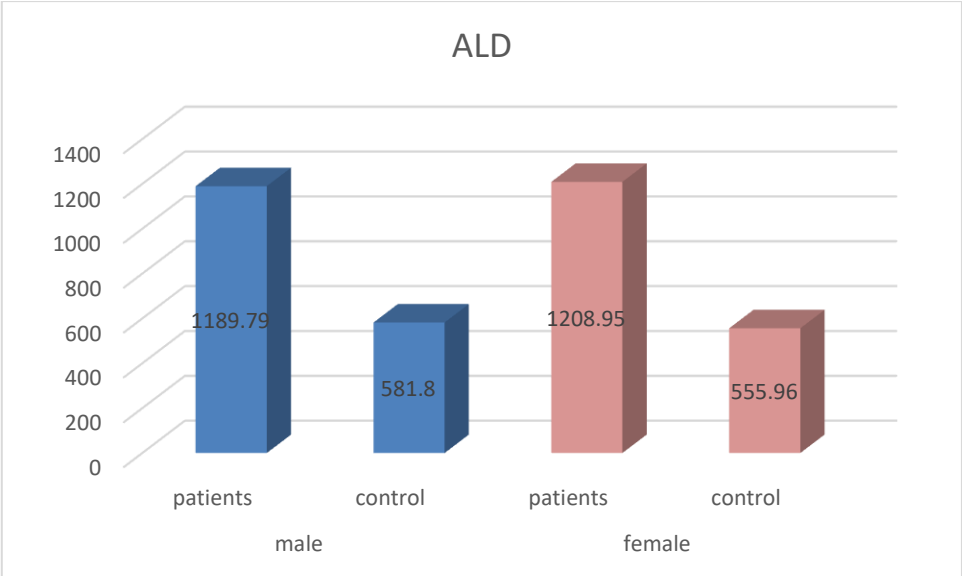

Fig.3 *Comparison of aldosterone levels between genders in hypertension patients and healthy individuals*

As Table (1)indicates, Figure (4) shows the mean ± standard deviation of the angiotensin II hormone level. For the group of male patients and healthy individuals, the level values were (557.68±390.7) pg/ml, (349±212) pg/ml, respectively, with a probability level of P≤0.04. For the group of females, the level values were (542.9±557) pg/ml and (279.3±81.67) pg/ml, respectively, with a probability level of P>0.05; the present work exhibited a considerable increase in the level of angiotensin II hormone in thein the patient group compared to the healthy control group. However, for females, the increase was not significant. The renin-angiotensin system (RAS) is responsible for controlling electrolyte balance and blood pressure. Angiotensin I-converting enzyme (ACE) activity produces angiotensin (ANG) II, the first end product of this system linked to physiological activity [18]. Protection diminishes after menopause; in general, estrogen raises angiotensinogen rates while lowering renin rates, ACE activity, AT1 receptor density, and aldosterone synthesis. Additionally, RAAS analogs like AT2 receptors and natriuretic peptides are activated by estrogen. [19]. In a study

conducted by Yanes et al. [20], They demonstrated that progesterone competes with aldosterone for mineralocorticoid receptors. Angiotensin-converting enzyme activity and renin levels are both elevated by testosterone. At least part of the gender disparities in cardiovascular and kidney illness can be explained by the effects of sex hormones on the RAAS, and these results are consistent with the present work, which displayed that angiotensin levels in females stayed within standard limits and increased in males from the affected groups.

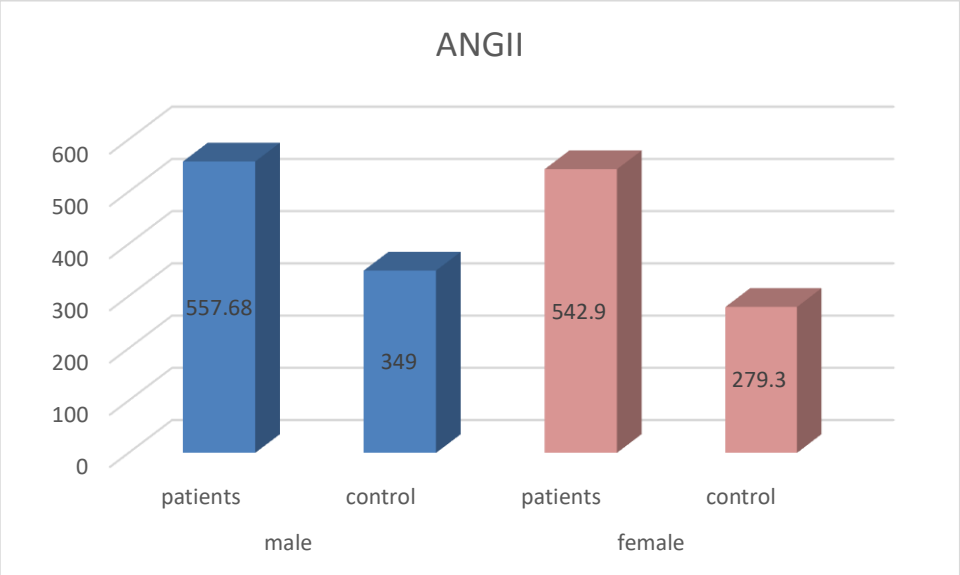

Fig. 4 *Comparison of the angiotensin hormone level between genders in hypertension patients and healthy individuals*

Table (1), Figure (5) indicates the mean ± standard deviation of urea level for the group of patients and healthy individuals as follows: for males' values, (56±58.2) mg/dl and (26.5±7.26) mg/dl, respectively, with a probability level of $P<0.02$; for females' values, (39±25.1) mg/dl and (30.5±5.9) mg/dl, respectively it with a probability level of $P>0.05$. The findings of the present work showed a significant increase in the urea levels in the blood serum of males compared to the control group. At the same time, it was found that there was a non-significant increase in the urea levels in the blood serum of females compared to the control group. These results are analogized with previous works [21]. High blood urea concentration may be caused by decreased distal tubular flow rate, which increases urea reabsorption and decreases excretion. This elevation may be related to decreased GFR as a result of the impact of hypertension on the renal role (reduction in renal blood flow as a cascade of increased renal vascular resistance) [22, 23]. High blood pressure is a dangerous factor for the development of chronic kidney disease and is also a significant cause of end-stage kidney disease. [24, 25].

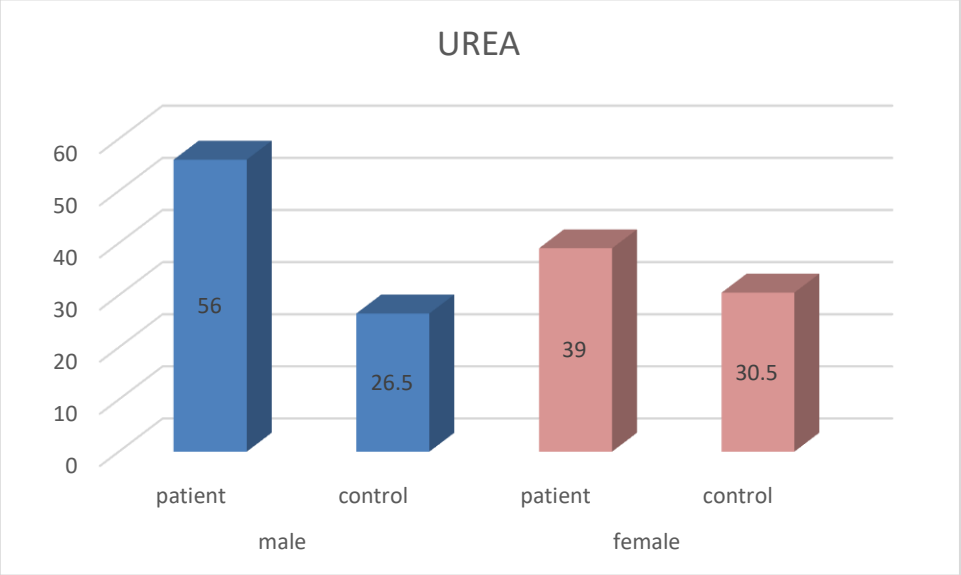

Fig. 5 *comparison of urea levels between genders in hypertension patients and healthy individuals*

Table (1) and Figure (6) display the mean ± standard deviation of creatinine rates for both patients and healthy individuals. For the male group, the creatinine rate values were (1.018±1.038) mg/dl for patients and (0.72±0.127) mg/dl for healthy individuals, with a probability level of P>0.05. For the female group, the creatinine rate values were (0.76±0.46) mg/dl for patients and (0.73±0.15) mg/dl for healthy individuals, with a probability level of P≤0.01. Based on the present work results, there is no significant change in the creatinine rates in the male patient's blood serum compared to the control group. Furthermore, there is a tiny, no significant change in the creatinine rates in the female patient's blood serum compared to the control group.

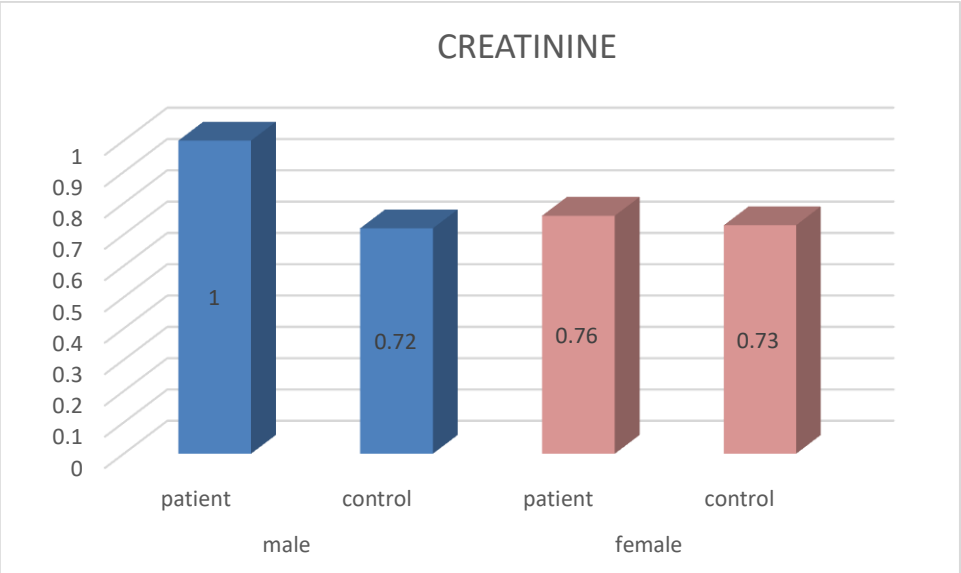

Fig. 6 *comparison of creatinine levels between genders in hypertension patients and healthy individuals*

In Table (1), Figure (7) displays the mean ± standard deviation of uric acid rates for both patients and healthy individuals. The values were (4.97±1.89) mg/dl and (4.59±1.09) mg/dl, respectively, for males, with a probability level of P≤0.03. For females, the values were (4.9±2.1) mg/dl and (4.37±0.79) mg/dl for patients and healthy individuals, respectively, at a probability level of P>0.05.

Our present work agrees with [24] as it showed that there is no significant difference in the average value of uric acid concentration in the patient's blood serum with high blood pressure compared to the control group. This work does not support the hypothesis that hyperuricemia may predict the development of hypertension as in previous studies [26-28].

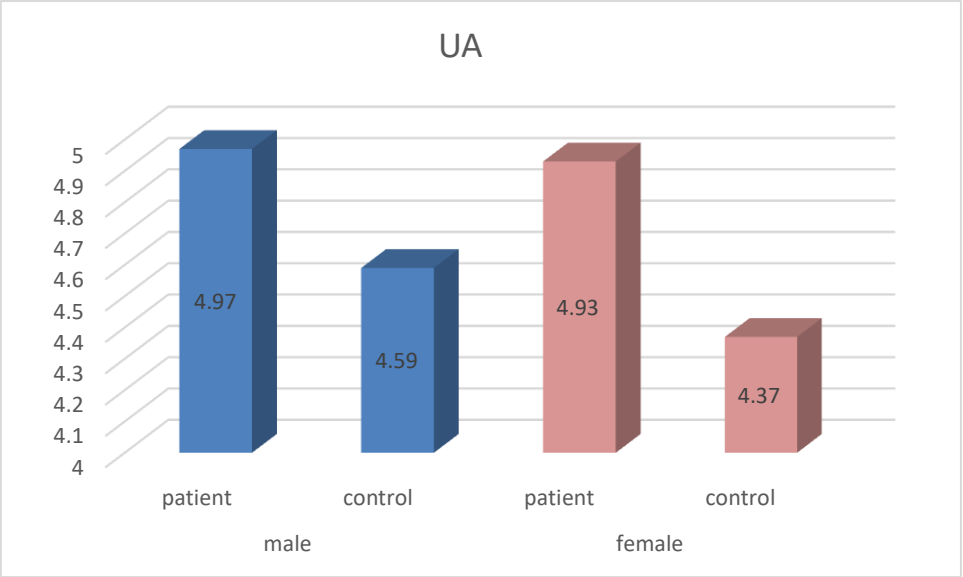

Fig. 7 *comparison of uric acid levels between genders in hypertension patients and healthy individuals*

It can be seen from Table (1) and Figure (8) that the mean ± standard deviation of the sodium level for both the patients and healthy individuals were (153.7±7.17) mmol/l and (141±4.8) mmol/l, respectively. The probability level was P≤0.03 for the male group and P≤0.01 for the female group. For the female group, the sodium levels for both the patients and healthy individuals were (150±9.2) mmol/l and (144±4.5)mmol/l, respectively.

In the present work, we notice an increase in the sodium level in the hypertensive patient's blood serum of both genders compared to the control group. Also, we observed an increase in the hormone aldosterone level, leading to the retention of sodium in the kidneys. This result aligns with a previous work [29] indicating an increase in sodium intake. Excessive retention of water contributes to salt-sensitive hypertension [30].

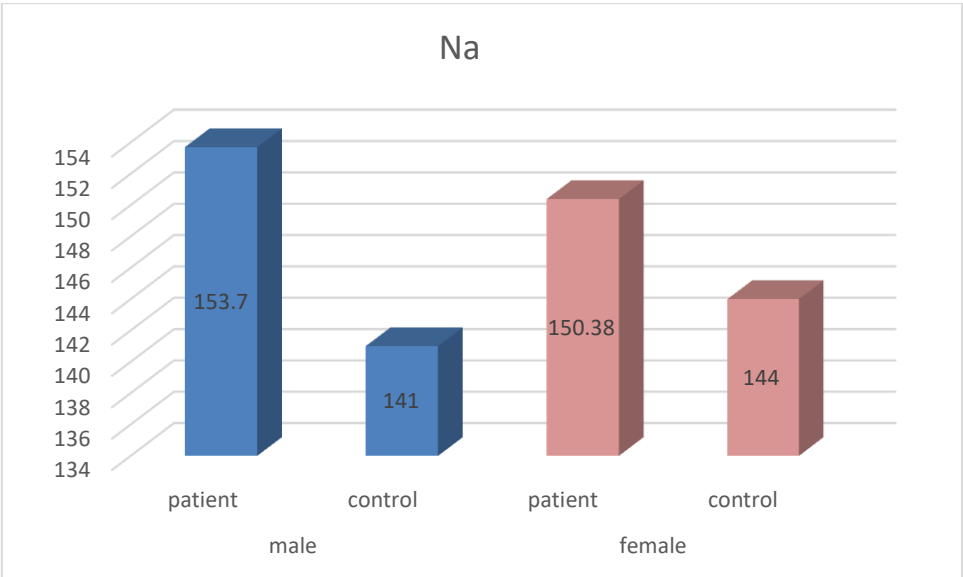

Fig. 8 *comparison of sodium levels between genders in hypertension patients and healthy individuals*

In Table (1)and Figure (9), the mean potassium level values were (3.3±0.56) mmol/l for patients and (4.3±0.46) mmol/l for healthy individuals. The probability level was P≤0.03 for males and P≤0.01 for females. For female patients, the mean potassium level values were (2.9±0.66) mmol/l and (4.2±0.46) mmol/l for healthy females.

The present work results exhibit a significant increase in potassium levels for both genders with high blood pressure compared to healthy individuals. Conversely, there was a substantial decrease in potassium levels in patients with high blood pressure compared to healthy individuals. (Lever et al. Furthermore, previous work has displayed that the total potassium levels in the body are inversely related to blood pressure. This was demonstrated in a work involving 91 patients with high blood pressure and 121 healthy individuals [31]. These findings are consistent with previous work [32-36]."

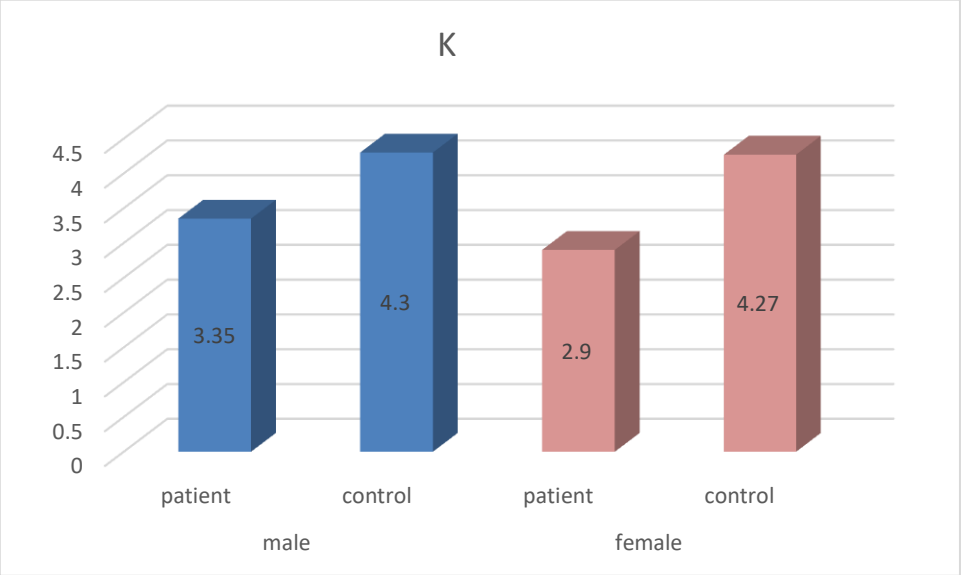

Fig. 9 *comparison of potassium levels between genders in hypertension patients and healthy individuals*

Conclusions

The study's findings indicate that both male and female blood serum levels of the hormones aldosterone and sodium increased significantly, while male blood serum levels of the hormones angiotensin II and urea increased significantly and female blood serum levels did not significantly increase. Additionally, both male and female blood serum levels of creatinine increased slightly but not significantly, and both male and female blood serum levels of potassium significantly decreased. The amount of uric acid in the blood serum of males and females did not differ from one another.

References

.1 Demikhov, O., et al., *Arterial hypertension prevention as an actual medical and social problem.* Bangladesh Journal of Medical Science, 2020. **19**(4): p. 722-729.

.2 Mohammed, M.M., S.M.S. Hassan, and E.M. Mustafa, *Epidemiological study of hypertensive cases among teaching staffs in kirkuk university.* Journal of Kirkuk University–Scientific Studies, 2010. **5**.(1)

.3 Martyniak, A. and P.J. Tomasik, *A new perspective on the renin-angiotensin system.* Diagnostics, 2022. **13**(1): p. 16.

.4 Kesireddy, V., et al., *The association of Life's Simple 7 with aldosterone among African Americans in the Jackson Heart Study.* Nutrients, 2019. **11**(5): p. 955.

.5 Gounden, V., H. Bhatt, and I. Jialal, *Renal function tests.* 2018.

.6 Mohiuddin, S.S. and D. Khattar, *Biochemistry, ammonia.* 2019.

.7 Faye, B., et al., *Nitrogen and Protein Parameters.* Camel Clinical Biochemistry and Hematology, 2018: p. 81-122.

.8 Ebert, N., et al., *Assessment of kidney function: clinical indications for measured GFR.* Clinical kidney journal, 2021. **14**(8 :(p. 1861-1870.

.9 Clarke, H.E., *Impact of Creatine Supplementation on Vascular Endothelial Function, Blood Flow and Functional Capacity in Older Adults*. 2021: The Florida State University.

.10 Bonilla, D.A., et al., *Metabolic basis of creatine in health and disease: a bioinformatics-assisted review.* Nutrients, 2021. **13**(4): p. 1238.

.11 Gutmann, I. and H.U. Bergmeyer, *Urea*, in *Methods of enzymatic analysis*. 1974, Elsevier. p. 1791-1801.

.12 Kawyn, M., *Pyrethroid and Chlorpyrifos Exposure and IQ of Children from the Salud Ambiental Montevideo (Sam) Study in Uruguay*. 2020, State University of New York at Buffalo.

.13 Fossati, P., L. Prencipe, and G. Berti, *Use of 3, 5-dichloro-2-hydroxybenzenesulfonic acid/4-aminophenazone chromogenic system in direct enzymic assay of uric acid in serum and urine.* Clinical chemistry, 1980. **26**(2): p. 227-231.

.14 Henry, R.J., *Clinical chemistry, principles and technics.* 1964.

.15 Nalbandian, R.M., et al., *Dithionite tube test—a rapid, inexpensive technique for the detection of hemoglobin S and non-S sickling hemoglobin.* Clinical chemistry, 1971. **17**(10): p. 1028-1032.

.16 Vasan, R.S., et al., *Serum aldosterone and the incidence of hypertension in nonhypertensive persons.* New England Journal of Medicine, 2004. **351**(1): p. 33-4.1

.17 Gideon, A., *Aldosterone—an Intermediate Factor Linking Psychosocial Stress to Essential Hypertension.* 2024.

.18 Velez, J.C.Q., et al., *Lack of renoprotective effect of chronic intravenous angiotensin-(1-7) or angiotensin-(2-10) in a rat model of focal segmental glomerulosclerosis.* PLoS One, 2014. **9**(10): p. e110083.

.19 Harvey, P.J., et al., *Estradiol induces discordant angiotensin and blood pressure responses to orthostasis in healthy postmenopausal women.* Hypertension, 2005. **45**(3): p. 399-405.

.20 Yanes, L.L., et al., *Testosterone-dependent hypertension and upregulation of intrarenal angiotensinogen in Dahl salt-sensitive rats.* American Journal of Physiology-Renal Physiology, 2009. **296**(4): p. F771-F779.
.21 Yang, Q., et al., *Homocysteine level is positively and independently associated with serum creatinine and urea nitrogen levels in old male patients with hypertension.* Scientific Reports, 2020. **10**(1): p. 18050.
.22 Ljungman, S., et al., *Blood pressure and renal function.* Acta Medica Scandinavica .1980 , :(6-1)208p. 17-25.
.23 Burits, C. and E. Ashwood, *Renal Function and Nitrogen Metabolism.* Tietz Fundamental Clinical Chemistry. 5th ed. McGraw Hill, 2001: p. 414-426.
.24 Duni, A., et al., *Hypertension in chronic kidney disease: novel insights.* Current Hypertension Reviews, 2020. **16**(1): p. 45-54.
.25 Alqudsi, M., S. Hiremath, and J.C.Q. Velez, *Review–current opinion in cardiology hypertension in chronic kidney disease.* Current Opinion in Cardiology, 2020. **35**(4): p. 360-367.
.26 Li, L., et al., *Is hyperuricemia an independent risk factor for new-onset chronic kidney disease?: a systematic review and meta-analysis based on observational cohort studies.* BMC nephrology, 2014. **15**: p. 1-12.
.27 Lv, Q., et al., *High serum uric acid and increased risk of type 2 diabetes: a systemic review and meta-analysis of prospective cohort studies.* PloS one, 2013. **8**(2): p. e56864.
.28 Jensen, T., et al., *Fructose and sugar: A major mediator of non-alcoholic fatty liver disease.* Journal of hepatology, 2018. **68**(5): p.1075-1063 .
.29 Stewart, P.M., *Mineralocorticoid hypertension.* The Lancet, 1999. **353**(9161): p. 1341-1347.
.30 Bailey, M.A. and N. Dhaun, *Salt sensitivity: causes, consequences, and recent advances.* Hypertension, 2024. **81**(3): p. 476-489.
.31 Lever, A., et al., *Sodium and potassium in essential hypertension.* Br Med J (Clin Res Ed), 1981. **283**(6289): p. 463-468.
.32 Rinner, M.D., L. Spliet-van Laar, and D. Kromhout, *Serum sodium, potassium, calcium and magnesium and blood pressure in a Dutch population.* Journal of hypertension, 1989. **7**(12): p. 977-981.
.33 Bulpitt, C., M. Shipley, and A. Semmence, *Blood pressure and plasma sodium and potassium.* Clinical Science, 1981. **61**(s7): p. 85s-87s.
.34 Gupta, R., et al., *Prevalence and determinants of hypertension in the urban population of Jaipur in western India.* Journal of hypertension, 1995. **13**(10): p. 1193-1200.
.35 Malhotra, P., et al., *Prevalence and determinants of hypertension in an un-industrialised rural population of North India.* Journal of human hypertension, 1999. **13**(7): p. 467-472.
.36 Pikilidou, M.I., et al., *Blood pressure and serum potassium levels in hypertensive patients receiving or not receiving antihypertensive treatment.* Clinical and Experimental Hypertension, 2007. **29**(8): p. 563-573.

IRAQI Academic Scientific Journals
Samarra Journal of Pure and Applied Science
SJPAS
مجلة سامراء للعلوم البحتة والتطبيقية
www.sjpas.com
p ISSN: 2663-7405
e ISSN: 2789-6838

قياس مستويات الألدوستيرون والأنجيوتنسين II وبعض المتغيرات الكيموحيوية في مصل الدم لدى مرضى ارتفاع ضغط الدم

خالده موفق عبد الرزاق *، فراس طاهر ماهر، ثائر فاضل خليل

قسم الكيمياء، كلية العلوم، جامعة تكريت، العراق

البحث مستل من رسالة ماجستير الباحث الاول

معلومات البحث:

تاريخ الاستلام: 2024/07/07
تاريخ التعديل: 2024/08/01
تاريخ القبــول: 2024/08/29
تاريخ الـنشر: 2025/06/30

الكلمات المفتاحية:

ارتفاع ضغط الدم، الالدوستيرون، اليوريا، ضغط الدم.

معلومات المؤلف

الايميل:

الموبايل:

الخلاصة:

في هذا العمل تم قياس مستويات الألدوستيرون والأنجيوتنسين II والمتغيرات البيوكيميائية المختلفة في مصل الدم لدى الأشخاص المصابين بارتفاع ضغط الدم ومقارنتها مع الأصحاء الذين تتراوح أعمارهم بين (45-60 سنة). تم تقدير مستويات الألدوستيرون والأنجيوتنسين II واليوريا والكرياتنين والصوديوم والبوتاسيوم لدى مرضى ارتفاع ضغط الدم. أظهرت النتائج الإحصائية زيادة ملحوظة في معدلات هرمون الألدوستيرون والصوديوم لدى مرضى ارتفاع ضغط الدم لكلا الجنسين مقارنة بمجموعة السيطرة. علاوة على ذلك، أظهرت النتائج زيادة كبيرة في هرمون الأنجيوتنسين II ومعدل اليوريا لدى المرضى الذكور المصابين بارتفاع ضغط الدم مقارنة بمجموعة السيطرة؛ ومع ذلك، لم تظهر فروق ذات دلالة إحصائية في المرضى الإناث مقارنة بالأشخاص الأصحاء. وأظهرت النتائج انخفاضا كبيرا في معدل البوتاسيوم. ومع ذلك، لم تظهر النتائج اختلافا كبيرا في معدلات الكرياتنين وحمض البوليك لدى كلا الجنسين، الأفراد الذين يعانون من ارتفاع ضغط الدم، مقارنة بمجموعة السيطرة.