Research Article

Al-Iraqia Medical College Journal (AIMCJ)
ISSN (Online): 3104-4565
ISSN (Print): 3104-4557

ARTICLE INFO
Received: 24/1 / 2026
Revised: 6/ 2/ 2026
Accepted: 7/ 2/ 2026
Publish online: 15 /4 / 2026

*Corresponding Author: Yasameen Hussein Abbood
Email: yasmine.h.abbood@aliraqia.edu.iq

CITATION
Yasameen Hussein Abbood. Androgenic Marker as an Indicator of Early Renal Impairment in Hypertensive Patients. *AIMCJ*. 2026;3(1): 77-84.

DOI: https://doi.org/10.58564/AIMCJ3.1.2026.246

Androgenic Markers as Indicators of Early Renal Impairment in Hypertensive patients

Yasameen Hussein Abbood*

Department of Chemistry and Biochemistry/ College of Medicine, Al-Iraqia University, Baghdad, Iraq.

Abstract

Hypertension is a significant factor leading to early renal failure and is frequently linked with metabolic and hormonal irregularities in men.

Increasing evidence suggests that testosterone deficiency may heighten renal vulnerability via mechanisms related to endothelial dysfunction. The purpose of the present study was to determine the association between total testosterone, free testosterone, sex hormone-binding globulin (SHBG), and early renal impairment in hypertensive men.

The study included 100 male patients diagnosed with essential hypertension (cases) and 60 male volunteers (control group). Participants were selected from internal medicine outpatient clinics at AL-Numan hospital; venous blood was centrifuged at 3000 rpm for 10 minutes to obtain serum.

Hypertensive individuals had significantly lower total testosterone (10.81 ± 2.92 nmol/L) and free testosterone levels (175.11 ± 48.24 pool/L), along with high levels of SHBG. These hormonal derangements were associated with a decrease in estimated Glomerular Filtration Rate (eGFR) and significant changes in Albumin-to-Creatinine Ratio (ACR), uric acid, and bicarbonate imbalances.

The current study shows significant differences in serum levels of androgenic markers between patients and controls. Quantitative analysis of serum parameters in hypertensive men found that free testosterone and SHBG showed a high correlation with renal dysfunction.

Keywords: Hypertension; Total testosterone; Free testosterone; SHBG; Renal biomarkers; eGFR; ACR.

AIMCJ. (2026),3; (1):77-84
https://doi.org/10.58564/AIMCJ3.1.2026.246

Introduction

One of the most common chronic ailments worldwide and a significant cause of renal and cardiovascular morbidity is hypertension, which progressively affects vascular and metabolic pathways (1). There is a growing body of evidence that it involves more than merely elevated blood pressure, with endocrine changes that can contribute to clinically insidious kidney dysfunction occurring early (2). Androgen status is an endocrine parameter recognized for its potential to increase men's sensitivity to renal issues. Testosterone, particularly in its free and physiologically active form, plays a crucial role in maintaining endothelial integrity, facilitating nitric oxide signaling, regulating oxidative stress, and overseeing metabolic processes (3). Even though total testosterone levels may remain within the normal range, significant changes in free testosterone can occur due to alterations in sex hormone-binding globulin (SHBG), which are typical in individuals with high blood pressure and metabolic issues (4,5). These disturbances in androgen bioavailability could support microvasculopathy, glomerular inflammation, and low-grade inflammation - pathways that accelerate the progression of renal dysfunction (6). Recent clinical findings suggest that lower androgen levels are linked to early indicators of kidney impairment, such as decreased eGFR and elevated albuminuria (7,8).

This paper examines the link between androgenic hormones and early renal impairment in hypertensive individuals; nonetheless, many studies have largely focused on total testosterone levels, overlooking the significance of free testosterone and SHBG. men who have high blood pressure. Moreover, there has been minimal investigation into hormone levels in association with important kidney health indicators, including the ACR, eGFR, creatinine, and various biochemical markers. This research aims to assess how total testosterone, free testosterone, and SHBG can aid in predicting early kidney dysfunction in hypertensive individuals, improve knowledge of the topic, and elevate clinical assessments.

Materials &Methods

Subjects and methods:

The research involved 100 male patients diagnosed with hypertensive disease and 60 healthy male controls lacking a history of hypertension, diabetes, or known kidney disease. Participants were collected from internal medicine outpatient clinics at AL-Numan hospital from September 2025 to December 2025.

Inclusion criteria: the study included males aged ≥18 years, diagnosed with hypertension (SBP ≥140 mmHg, DBP ≥90 mmHg).

Exclusion criteria: Individuals with diabetes mellitus, liver or thyroid disorders, other chronic illnesses, or medications that could induce hypertension were not included in this study

Demographic factors, medical history (duration of hypertension), drug history, smoking status, anthropometric measurements (height, weight, BMI, waist circumference), and blood pressure readings (average of 2 seated measurements) were documented.

AIMCJ. (2026),3; (1):77-84
https://doi.org/10.58564/AIMCJ3.1.2026.246

Blood samples were obtained in the morning following a 10–12-hour overnight fast. Five milliliters of venous blood were collected from both healthy individuals and patients. The blood was placed in tubes without anticoagulants and left at room temperature for 15 minutes. The samples were subsequently centrifuged at 3000 RPM for 10 minutes to obtain serum, and then they were kept in Obendorf tubes at -20 degrees for later use.

The tests conducted included Total Testosterone, Free Testosterone, and sex hormone-binding globulin (SHBG), measured via Chemiluminescent Immunoassay Cobas e411. A urine sample was used to assess the albumin/creatinine ratio (ACR). Additional biochemical assays for creatinine, urea, uric acid, and bicarbonate were performed using a colorimetric kit and measured with an automated chemistry analyzer from China.

Statistical Analysis

Statistical analysis of the data was conducted using SPSS version 26; a t-test was applied to assess the means of the variables. A significance level of $P \leq 0.05$ was used to determine statistical significance. The ANOVA test was used to compare groups at a significant level ($P \leq 0.05$), and the variable values were presented as mean ± standard deviation.

Results:

Table 1 shows that individuals with hypertension were slightly older (44.33 ± 9.52 years) than the control group (42.21 ± 7.09 years), and hypertensive men had a slightly higher BMI (25.66 ± 3.72 kg/m²). Central obesity, markedly higher in hypertensive men, measured 103.04 ± 9.44 cm ($p < 0.001$). Systolic and diastolic BP were significantly higher in hypertensive men ($p < 0.001$). The hypertensive group had a mean disease duration of 8.7 ± 4.2 years.

Table 2 shows notable distinctions between the hypertensive cohort and healthy controls across all assessed renal and hormonal metrics ($p < 0.001$). Hypertensive individuals exhibited elevated creatinine (1.37 ± 0.24 vs. 0.86 ± 0.11 mg/dL), urea (43.81 ± 9.54 vs. 28.46 ± 5.63 mg/dL), and uric acid (6.03 ± 1.12 vs. 4.18 ± 0.94 mg/dL), along with lower bicarbonate (21.29 ± 2.44 vs. 24.46 ± 2.51 mmol/L). These changes were associated with a significant decline in eGFR (65.06 ± 14.22 vs. 104.96 ± 11.51 mL/min/1.73 m²) and considerable increases in ACR (89.68 ± 34.70 vs. 11.81 ± 4.16 mg/g). Furthermore, hypertensive individuals had lower total testosterone (10.81 ± 2.92 vs. 18.67 ± 3.31 nmol/L) and free testosterone (175.11 ± 48.24 vs. 320.02 ± 55.45 pool/L), whereas SHBG levels were elevated (48.06 ± 8.94 vs. 34.21 ± 7.31 nmol/L).

Table 3 illustrates the relationship between hormonal markers and kidney function measures. Lower testosterone levels are associated with reduced kidney function and higher renal damage markers, as shown by a modest positive association between total testosterone and eGFR ($r = 0.42$) and a negative correlation with ACR ($r = –0.39$). Free testosterone showed stronger correlations than total testosterone, indicating a more robust positive relationship with eGFR ($r = 0.47$) and a notable negative correlation with ACR ($r = –0.44$). SHBG showed a markedly significant positive correlation with ACR ($r = 0.38$, $p < 0.001$) and a notable negative correlation with eGFR ($r = –0.33$, $p < 0.01$).

AIMCJ. (2026),3; (1):77-84
https://doi.org/10.58564/AIMCJ3.1.2026.246

Table 4 presents the multivariable regression model used to identify independent predictors of eGFR in men with hypertension. Age significantly reduced eGFR (β = –0.41, p < 0.001). BMI showed a notable negative association (β = –0.29, p = 0.020), suggesting that a higher BMI reduces the kidneys' ability to filter blood. Systolic blood pressure was another key predictor (β = –0.26, p = 0.012). Free testosterone was the key hormone influencing eGFR (β = +0.89, p < 0.001). Conversely, SHBG had a significant negative effect on eGFR (β = –0.24, p = 0.031). ACR was a notable predictor (β = –0.20, p = 0.036), suggesting that higher albuminuria and changes in the urinary creatinine ratio are independently associated with decreased kidney function.

Table 1: Demographic and Clinical Characteristics of the Study Population

Variable	Controls (n=60)	Hypertensive (n=100)	P-value
Age (years)	42.21 ± 7.09	44.33 ± 9.52	NS
BMI (kg/m²)	24.61 ± 2.73	25.66 ± 3.72	NS
Waist circumference (cm)	86.04 ± 7.92	103.04 ± 9.44	**<0.001**
Systolic BP (mmHg)	118.20 ± 7.56	154.64 ± 12.89	**<0.001**
Diastolic BP (mmHg)	75.78 ± 6.21	92.09 ± 8.19	**<0.001**
Smokers, n (%)	14 (23.3%)	46 (46.0%)	**0.004**
Family history of hypertension, n (%)	10 (16.7%)	44 (44.0%)	**<0.001**
Physical inactivity, n (%)	12 (20.0%)	57 (57.0%)	**<0.001**
Central obesity, n (%)	15 (25.0%)	69 (69.0%)	**<0.001**
Dyslipidemia, n (%)	13 (21.7%)	61 (61.0%)	**<0.001**
Duration of hypertension (years)	—	8.73 ± 4.26	—

"Values of p<0.05 were considered to be significant."

Table 2 Comparison of Biomarkers Between the Hypertensive Group and the Control Group.

Parameters	Controls (mean ± SD)	Hypertensive (mean ± SD)	P- value
Creatinine (mg/dL)	0.86 ± 0.11	1.37 ± 0.24	**<0.001**
Urea (mg/dL)	28.46 ± 5.63	43.81 ± 9.54	**<0.001**
Uric Acid (mg/dL)	4.18 ± 0.94	6.03 ± 1.12	**<0.001**
Bicarbonate (mmol/L)	24.46 ± 2.51	21.29 ± 2.44	**<0.001**
eGFR (mL/min/1.73m²)	104.96 ± 11.51	65.06 ± 14.22	**<0.001**
ACR (mg/g)	11.81 ± 4.16	89.68 ± 34.70	**<0.001**
Total Testosterone (nmol/L)	18.67 ± 3.31	10.81 ± 2.92	**<0.001**
Free Testosterone (pmol/L)	320.02 ± 55.45	175.11 ± 48.24	**<0.001**
SHBG (nmol/L)	34.21 ± 7.31	48.06 ± 8.94	**<0.001**

"Values of p<0.05 were considered to be significant."

ACR; albumin/creatinine ratio, SHBG; sex hormone-binding protein.

AIMCJ. (2026),3; (1):77-84
https://doi.org/10.58564/AIMCJ3.1.2026.246

Table 3. Correlation Between Hormonal Parameters and Renal Functions Among the Study Group.

Parameter	eGFR (r)	ACR (r)	Bicarbonate (r)	P-value
Total Testosterone	+0.42	–0.39	+0.28	**<0.001**
Free Testosterone	**+0.47**	**–0.44**	**+0.31**	**<0.001**
SHBG	**–0.33**	**+0.38**	**–0.22**	**<0.01**

"Values of p<0.05 were considered to be significant."

SHBG: sex hormone-binding protein.

Table 4 Multivariable Linear Regression Analysis for Predictors of eGFR in Hypertensive Patients

Predictor	β Coefficient	P-value
Age	–0.41	**<0.001**
BMI	–0.29	**0.020**
Systolic BP	–0.26	**0.012**
Free Testosterone (pmol/L)	**+0.89**	**<0.001**
SHBG (nmol/L)	**–0.24**	**0.031**
ACR	–0.20	**0.036**

Discussion

Hypertension is a major long-term contributor to early kidney dysfunction, leading to hormonal and metabolic disturbances that may increase the risk of kidney damage. Recent studies indicate that waist circumference in individuals with hypertension may be a more sensitive indicator of renal dysfunction than BMI alone. WC is a body measurement used to assess the distribution of fat in the abdominal area, which is addressed in the present research (9,10). Dyslipidemia, characterized by elevated lipid levels in the bloodstream, is commonly observed in individuals with hypertension, central obesity (especially excess fat around the waist), and ectopic fat accumulation within and outside the kidneys and along blood vessels (11). Genetic factors, lifestyle choices, duration, and environmental elements may have influenced this observation (12,13).

However, regular testing indicates increased levels of creatinine, urea, and uric acid, along with a notable decrease in estimated eGFR (14). Additionally, bicarbonate levels were lowered in these individuals, suggesting they could be facing metabolic acidosis, an initial indicator that their kidneys struggle to maintain the correct acid-base equilibrium, which could exacerbate kidney problems that may develop when the kidneys fail to preserve homeostasis (15, 16). Hypertension limits blood flow by narrowing renal blood vessels. As a result, the kidneys are unable to remove all waste products and fluids from the body. Blood pressure increases fluid overload in the blood vessels (17).

AIMCJ. (2026),3; (1):77-84
https://doi.org/10.58564/AIMCJ3.1.2026.246

The patient group showed early signs of impairment and significant changes in ACR. These findings correspond with recent research that revealed hypertension, disruptions in renal microvascular stability, and heightened early glomerular leakage happening prior to the obvious onset of advanced kidney disease (18). Androgens are essential in controlling blood pressure (BP). Additionally, various studies have demonstrated that hormonal imbalances worsen kidney damage (19). The protein sex hormone-binding globulin (SHBG) binds to testosterone. Free testosterone, an active form of the hormone, is acknowledged as a more dependable marker for metabolic and cardiovascular results and exhibits a stronger connection with kidney function compared to total testosterone (20, 21)

However, previous research shows that discrepancies in androgen levels are common in those with hypertension, possibly influencing vascular tone, oxidative balance, and inflammatory pathways (8). Testosterone may attach to the aldosterone receptor, influencing blood pressure and fluid management. As a result, it may be associated with arterial hypertension by stimulating the RAAS system and increasing sodium retention. These processes could cause kidney harm because of high blood pressure (22, 23). A negative correlation between testosterone levels and hypertension supports the notion that hypertension develops with reduced androgen production, regardless of age or body mass index.

The correlation analysis showed that both total and free testosterone had a positive relationship with eGFR and bicarbonate levels, while being negatively correlated with ACR results. In contrast, SHBG exhibited a reverse correlation, indicating that diminished androgen bioavailability plays a role in the onset of kidney damage in those with hypertension.

These results highlight the clinical importance of assessing both free testosterone and SHBG in hormonal imbalances for hypertensive patients to identify androgen deficiency and its potential impact on kidney function.

LIMITATIONS:

The sample size of the study is restricted. Additionally, serum androgen levels were assessed only once, resulting in the inability to identify temporal fluctuations in hormone levels and to accurately determine an individual's long-term status.

Conclusion

The association between lower androgen levels and kidney function biomarkers reinforces the idea that hormonal deficiency significantly contributes to the advancement of renal impairment in patients with hypertensive disease. The evidence indicates that low androgen levels might play a role in advancing renal impairment instead of just being a symptom, and treating it could result in better clinical outcomes. However, these advantages need to be considered alongside possible risks, like fluid retention or exacerbation of heart failure.

Funding: Self-funded

Conflicts of interest: nil

Ethical statement

Informed verbal consent of participants was obtained, and data were collected via a questionnaire for this study.

AIMCJ. (2026),3; (1):77-84
https://doi.org/10.58564/AIMCJ3.1.2026.246

Approval for this research was obtained from the Ethics Committee at the College of Medicine, Al-Iraqia University (FM.SA/2-1/2/2026).

REFERENCES

1. Bachlitzanaki M, Aletras G, Papakonstantinopoulou K, Vasilaki N, Bachlitzanaki E, Stratinaki M, Lydakis C. Hypertension: A Proximal Clinical Signature and Promoter of Early Cardiorenal Disease?. *Journal of CardioRenal Medicine*. 2026 Feb 5;2(1):2. https://doi.org/10.3390/jcrm2010002
2. Nikolopoulou L, Dimitriadis K, Pyrpyris N, Tatakis F, Iliakis P, Thomopoulos C, Konstantinidis D, Rallidis L, Tousoulis D, Tsioufis K. The effect of renal denervation on capillary density in patients with uncontrolled hypertension. *Microcirculation.* 2025 Jul;32(5):e70015. https://doi.org/10.1111/micc.70015
3. Rojas-Zambrano JG, Rojas-Zambrano A, Rojas-Zambrano AF, Rojas-Zambrano Sr. AF. Impact of testosterone on male health: A systematic review. *Cureus*. 2025 Apr 24;17(4). https://doi.org/10.7759/cureus.82917
4. Berg WT, Miner M. Hypogonadism and metabolic syndrome: review and update. *Current Opinion in Endocrinology, Diabetes and Obesity*. 2020 Dec 1;27(6):404-10. https://doi.org/10.1097/MED.0000000000000582.
5. Persky V, Abasilim C, Tsintsifas K, Day T, Sargis RM, Daviglus ML, Cai J, Freels S, Unterman T, Chavez N, Kaplan R. Sex hormones and diabetes in 45-to 74-year-old men and postmenopausal women: the hispanic community health study. *The Journal of Clinical Endocrinology & Metabolism*. 2023 Jul;108(7):1709-26. DOI: https://doi.org/10.1210/clinem/dgad018
6. Valdivielso JM, Jacobs-Cacha C, Soler MJ. Sex hormones and their influence on chronic kidney disease. *Current opinion in nephrology and hypertension*. 2019 Jan 1;28(1):1-9. https://doi.org/10.1097/MNH.0000000000000463
7. Yaqoob K, Naderi H, Thomson RJ, Aksentijevic D, Jensen MT, Munroe PB, Petersen SE, Aung N, Yaqoob MM. Prognostic impact of albuminuria in early-stage chronic kidney disease on cardiovascular outcomes: a cohort study. *Heart.* 2025 Jun 1;111(11):506-12. https://doi.org/10.1136/heartjnl-2024-324988
8. Negretto LA, Rassi N, Soares LR, Saraiva AB, Teixeira ME, Santos LD, Souza AL, Jardim PC, Barroso de Souza WK, Jardim TD. Testosterone deficiency in hypertensive men: prevalence and associated factors. *Arquivos Brasileiros de Cardiologia*. 2024;121:e20230138. https://doi.org/10.36660/abc.20230138i
9. Sun JY, Hua Y, Zou HY, Qu Q, Yuan Y, Sun GZ, Sun W, Kong XQ. Association between waist circumference and the prevalence of (Pre) hypertension among 27,894 US adults. *Frontiers in cardiovascular medicine*. 2021 Oct 12;8:717257. https://doi.org/10.3389/fcvm.2021.717257
10. Shaker AM, Nuaman BN. The Metabolic and Hepatic Impact of Central Obesity in MASLD Patients: Evidence from an Iraqi Cross-sectional Cohort. *Al-Iraqia Medical College Journal.* 2025 Dec 15;2(3):54-64. https://doi.org/10.58564/AIMCJ2.3.2025.234
11. Su Y, Sun JY, Su ZY, Sun W. Revisiting waist circumference: a hypertension risk factor that requires a more in-depth understanding. *Current Cardiology Reviews.* 2024 Jul 1;20(4):77-85. DOI: https://doi.org/10.2174/011573403X29057424032204135 6
12. Mohseni P, Khalili D, Djalalinia S, Mohseni H, Farzadfar F, Shafiee A, Izadi N. The synergistic effect of obesity and dyslipidemia on hypertension: results from the STEPS survey. *Diabetology & Metabolic*

AIMCJ. (2026),3; (1):77-84
https://doi.org/10.58564/AIMCJ3.1.2026.246

Syndrome. 2024 Apr 3;16(1):81. https://doi.org/10.1186/s13098-024-01315-x

13. Sun J, Huang B, Zhao W, Li Q, Yin S, Zhang Y, Huang R. Prevalence of dyslipidemia and its associated risk factors in hypertensive population: A single-center cross-sectional study in southern China. *Journal of the Renin-Angiotensin-Aldosterone System.* 2025 Jun;26:14703203251353658. https://doi.org/10.1177/14703203251353658.

14. Ukpeh IE, Akpan MI, Ugbe UM, Enyievi PB, Osuchukwu NC, Bassey EE, Yolo SB. Predictors of blood pressure control among patients with hypertension: a cross-sectional study in a Nigerian tertiary health facility. *BMJ Open.* 2025 Mar 1;15(3):e091010. https://doi.org/10.1136/bmjopen-2024091010

15. Kim GH. The primary role of the kidney in the pathogenesis of hypertension. *Life*. 2024 Jan 14;14(1):119. https://doi.org/10.3390/life14010119

16. Chanda AK, Ghosh BC, Hoq ME, Rimi SA, Paul T, Bhattacharjee T, Jui Y. Impact of Hypertension on Serum Creatinine, eGFR, Albumin and Albumin Creatinine Ratio on Kidney Function among the Employees of Sylhet MAG Osmani Medical College and Hospital. *Journal of Comilla Medical College Teachers' Association.* 2025 May 20;29(1):59-64. 5

17. Jabor ZR, Abbass IS, Mhaimeed AJ. Evaluation and Correlation of Urinary Microalbumin in Early Diagnosis of Patients with Hypertension-Related Chronic Kidney Disease. *Karbala Journal of Pharmaceutical Sciences*. 2023;14(23):44-53. DOI: https://doi.org/10.62472/kjps.v14.i23.44-53

18. Beernink JM, van Mil D, Laverman GD, Heerspink HJ, Gansevoort RT. Developments in albuminuria testing: A key biomarker for detection, prognosis, and surveillance of kidney and cardiovascular disease—A practical update for clinicians. *Diabetes, Obesity, and Metabolism.* 2025 Sep;27:15-33. https://doi.org/10.1111/dom.16359

19. Aryal D, Roy T, Chamcheu JC, Jackson KE. Chronic metabolic acidosis elicits hypertension via upregulation of intrarenal angiotensin II and induction of oxidative stress. *Antioxidants.* 2020 Dec 22;10(1):2. DOI: https://doi.org/10.1007/s00424-024-02912-5

20. Drury ER, Wu J, Gigliotti JC, Le TH. Sex differences in blood pressure regulation and hypertension: renal, hemodynamic, and hormonal mechanisms. *Physiological Reviews.* 2023 Oct 16. DO: https://doi.org/10.1152/physrev.00041.2022

21. Lau LH, Nano J, Prehn C, Cecil A, Rathmann W, Zeller T, Lechner A, Adamski J, Peters A, Thorand B. Associations of endogenous androgens and sex hormone-binding globulin with kidney function and chronic kidney disease. *Frontiers in endocrinology*. 2022 Dec 19;13:1000650. DOI: https://doi.org/10.3389/fendo.2022.1000650.

22. Zitzmann M. Testosterone deficiency and chronic kidney disease. *Journal of clinical & translational endocrinology.* 2024 Sep 1;37:100365. https://doi.org/10.1016/j.jcte.2024.100365

23. Liu J, Liu Z, Sun W, Luo L, An X, Yu D, Wang W. Role of sex hormones in diabetic nephropathy. *Frontiers in Endocrinology.* 2023 Apr 18;14:1135530. DOI: https://doi.org/10.3389/fendo.2023.1135530