Original

Association between High-Sensitivity C –reactive Protein Levels and Kidney Function in a Hypertensive Population: A Case-Control Study in a Government Hospital, Warri, Nigeria

[1]Augustine Onovuakpo Eguvbe, [2]Samuel Olubayo Olubori, [3]Pedro Ejomafuvwe Amrevwodjemu

[1]Department of Chemical Pathology, Delta State University, Abraka, Delta State, Nigeria.
[2]Department of Chemical Pathology, Irrua Specialist Teaching Hospital, Irrua, Edo State, Nigeria.
[3]Department of Pharmacology and Therapeutics, Delta State University, Abraka, Delta State, Nigeria.

Corresponding author: *Augustine Onovuakpo Eguvbe,* Department of Chemical Pathology, Faculty of Basic Clinical Sciences, Delta State University, Abraka, Delta State, Nigeria.augustine.eguvbe@delsu.edu.ng:+2348153685021

Article history: Received 04 October 2025, Reviewed 21 November 2025, Accepted for publication 11 December 2025

ABSTRACT

Background: Low-grade vascular inflammation plays an important role in the pathophysiology of hypertension and is commonly observed in individuals with impaired renal function. Hypertension, a major modifiable risk factor for cardiovascular disease, has been associated with elevated inflammatory markers and declining kidney function. This study compared serum high-sensitivity C-reactive protein (HSCRP) levels and renal function between hypertensive patients and normotensive individuals.

Methods: This hospital-based case–control study included 300 participants recruited at Central Hospital, Warri, Nigeria, between January 2023 and March 2024. Participants comprised 200 adults with hypertension and 100 normotensive controls. Sociodemographic and clinical data were collected using a structured questionnaire. Serum HSCRP levels and estimated glomerular filtration rate (eGFR) were measured. Data were analysed using SPSS version 23, with statistical significance set at $p<0.05$.

Results: Hypertensive participants were significantly older than controls (58.11 ± 13.03 vs 43.71 ± 10.67 years; $p<0.001$) and had a higher proportion of females (73.5% vs 47.0%; $p=0.007$). Impaired renal function (eGFR <60 mL/min/1.73 m²) was more prevalent among hypertensives than controls (26.5% vs 8.0%; $p=0.004$). Mean HSCRP levels were significantly higher in hypertensive patients (4.22 ± 3.30 vs 1.51 ± 1.48 mg/L; $p<0.001$). Among hypertensives, HSCRP showed a weak but significant negative correlation with eGFR ($r=–0.142$; $p=0.044$).

Conclusion: Hypertensive patients exhibit increased inflammatory burden and a higher prevalence of renal impairment. The inverse association between HSCRP and eGFR suggests that inflammation may contribute to declining kidney function, underscoring the need for early detection of subclinical inflammation and renal dysfunction in hypertension.

Keywords: Hypertension, low-grade inflammation, high-sensitivity C-reactive protein, estimated glomerular filtration rate, renal function.

How to cite this article

Eguvbe AO, Olubori SO, Amrevwodjemu PE. Association between High-Sensitivity C –reactive Protein Levels and Kidney Function in a Hypertensive Population: A Case-Control Study in a Government Hospital, Warri, Nigeria. The Nigerian Health Journal 2025; 25(4): 1501 – 1509. https://doi.org/10.71637/tnhj.v25i4.1232

INTRODUCTION

Hypertension, the main modifiable risk factor of cardiovascular disease, is linked with low-grade inflammation and impaired renal function.[1, 2, 3] It is one of the leading risk factors of chronic kidney disease (CKD).[4] Arterial hypertension can lead to CKD via excessive activation of the renin angiotensin system and increased production of angiotensin II. The consequences of release of angiotensin II include high blood pressure and stimulation of proinflammatory and profibrotic effects in the kidneys.[4] Thus, Low-grade inflammation restricted to vascular tissue play a significant role in the pathophysiology of hypertension and is a characteristic finding in patients with impaired renal function.[5, 6]

C-reactive protein (CRP) is an acute phase reactant and a non-specific inflammatory marker produced mainly in the liver, under the influence of pro-inflammatory cytokines.[7, 8] HSCRP is derived from HSCRP assays with the ability to detect lower levels of serum CRP.[7] HSCRP results are interpreted as follows: CRP < 1 mg/l imply low cardiovascular risk; 1 to 3 mg/l imply intermediate cardiovascular risk; > 3 mg/l imply a high cardiovascular risk.[9] Inflammation play a significant role in the pathogenesis of atherosclerosis and cardiovascular risk and HSCRP has been shown to be a leading biomarker for predicting cardiovascular risk.[10] The proposed mechanisms linking HSCRP to the development of atherogenesis include vascular cell activation, activation of complement system, accumulation of monocyte, apoptosis, collection of lipids, and thrombosis in coronaries.[11] In addition, low-grade inflammation has been documented to accelerate renal function decline.[12] Hence, high levels of HSCRP have been documented to predict hypertension and declining renal function in individuals without kidney disease.[13] Stuveling et al, reported CRP as a risk factor for impaired kidney function.[14] Thus, CRP levels play a role in the pathogenesis of acute kidney injury (AKI) and chronic kidney disease (CKD) by activating Smad3, which leads to cell death and progressive renal fibrosis.[15] Assessing vascular inflammatory alterations with inflammatory markers in the serum such as high-sensitivity C-reactive protein (HSCRP) is easier than using cardiac imaging methods.[16]

Kidney function is most commonly assessed by the glomerular filtration rate (GFR), which is defined as the rate in mL per minute at which plasma is filtered in the glomerular capillaries. GFR is usually estimated from plasma concentration of creatinine.[17] CKD, characterised by a slow decline in renal function, less than 60 ml/min/1.73 m^2 for at least three months, is becoming a leading cause of death worldwide.[18] Thus, optimising factors, such as inflammation, that play a role in renal function decline, may be necessary to improve treatment outcome in patients with CKD. Research assessing the relationship between HSCRP and renal function in hypertensive patients are scarce in this environment. Thus, this study was aimed at determining and comparing the serum levels of HSCRP and renal function and their relationship in hypertensive patients vs. normotensive individuals.

MATERIALS AND METHODS

Study design

The research was a case-control study.

Study setting

The study was done at the General Outpatient and Cardiology units of Central Hospital, Warri, Delta State. It began in January 2023 and ended in March, 2024. Central Hospital, Warri, is a large secondary health facility with a two hundred and fifty bed capacity and receives patients and referrals from Warri and most parts of Delta State.

Study population

Newly diagnosed hypertensives were recruited as cases while normotensive individuals including patient relatives, staff and colleagues, were recruited as controls. This research enlisted three hundred participants consisting of two hundred hypertensives and one hundred controls. The inclusion criteria were participants aged eighteen years and above, hypertension (blood pressure ≥ 140/90 mmHg) and normal blood pressure (blood pressure below 140/90 mmHg). The exclusion criteria were history of diabetes mellitus, acute infections, inflammatory conditions, females taking hormone replacement drugs, and trauma.

Sample size and sampling procedure

This research enlisted three hundred participants consisting of two hundred hypertensives and one hundred controls. The sample size for this case–control study was estimated using the standard formula for detecting an odds ratio between two proportions in analytical case–control designs. An assumed detectable odds ratio (OR) of 2.0 was used, with a 5% level of

Print ISSN: 0189-9287 Online ISSN: 2992-345X

significance ($Z\alpha/2 = 1.96$) and 80% statistical power ($Z\beta = 0.84$). The expected prevalence of exposure among controls (p_2) was set at 0.15, from which the corresponding prevalence among cases (p_1) was derived using the relationship between odds ratio and exposure probability. This yielded $p_1 = 0.26$. Substituting these values into the case–control sample size equation for equal groups produced an estimated minimum of approximately 200 participants per group (≈400 total) for a 1:1 ratio. For the planned 2:1 case–control ratio, the total required sample size was adjusted to approximately 300 participants. A consecutive sampling approach was employed in the selection of the research population from patients visiting the General outpatient and Cardiology unit of Central Hospital, Warri. A written informed consent was gotten from each study participant.

Demographic and baseline characteristics

A questionnaire was used to collect data such as identification number, age, gender, weight, height, waist circumference, medical history and laboratory results. Body weight, height and waist circumference were measured using universally accepted methods, and body mass index (BMI) was calculated from the weight (kg) divided by the height (meter) squared. Hypertensive cases were determined after the second visit of the participants to the clinic with an elevated blood pressure of 140/90 mmHg and above on two or more occasions. Obesity was defined as BMI that is 30 kg/m² or greater.

Blood and urine samples

About five to ten milliliters (5-10 ml) of venous blood and random urine were taken from each participant. The blood was centrifuged at room temperature within 35 – 45 minutes of collection. The serum component was kept at -20℃ until analysis. The urine was stored in the refrigerator at 2 to 8℃ . Serum and urine creatinine concentrations were assayed on a spectrophotometer using the kinetic modification of the Jaffe procedure. Serum level of HSCRP was analysed by Enzyme Linked Immunosorbent Assay (ELISA) according to the manufacturer's protocol. Estimated glomerular filtration rate (eGFR) was estimated from serum creatinine using the Modification of Diet for Renal Disease formula based on age, sex, race and serum creatinine. [19]

Data analysis

The data were analyzed using the Statistical Package for Social Sciences (SPSS) software version 23. Normally distributed continuous variables were presented as mean, standard deviation, and ranges. Categorical variables were presented using frequencies and percentages. The differences in means of continuous variables between the hypertensive group and controls were compared using the student T test. Chi square test was used to analyze categorical variables. The Pearson's correlation coefficient was used to correlate HSCRP with eGFR. Statistical significance was set at <0.05.

RESULTS

Three hundred participants made up of two hundred hypertensive and one hundred normotensive individuals were enrolled for the study. In the hypertensive and control groups, the modal age groups were 40-60. The hypertensives were significantly older than the controls (58.11 ± 13.03 years and 43.71 ± 10.67 years respectively, $p < 0.001$). In the hypertensives, there were 147 (73.5%) females and 53 (26.5%) males while the controls had 47 (47.0%) females and 53 (53.0%) males. The difference in sex distribution was statistically significant ($p = 0.007$). The age and sex characteristics are shown in detail in Table 1 below.

Anthropometrics

No significant difference was seen in the body weight of the hypertensives and controls (76.05 ± 17.96 vs. 76.18 ± 9.19 kg, respectively, $p = 0.946$) (Table 1). Height was also comparable between the two groups (1.61 ± 0.08 vs. 1.63 ± 0.05 m respectively, $p = 0.208$). Also, the BMI did not differ significantly in both groups (28.86 ± 6.44 vs. 28.55 ± 4.63 kg/m² respectively, $p = 0.736$). BMI categorization showed no significant differences either, with similar proportions of overweight and obese individuals across both groups. However, waist circumference differed significantly between the groups. Hypertensive individuals had a higher mean waist circumference of 98.77 ± 13.15 cm, compared to 91.23 ± 8.18 cm in the control group ($p < 0.001$). This suggests that central obesity may be more prevalent among hypertensive patients (Table 1).

Social history

Regarding social habits, alcohol consumption was higher in the hypertensive group (16.5%) compared to the control group (7.0%), though this difference did not

Print ISSN: 0189-9287 Online ISSN: 2992-345X

reach statistical significance (p = 0.063). Smoking was rare in both groups, with only one smoker in the control group and none in the hypertensive group, and this difference was not statistically significant (p = 0.246).

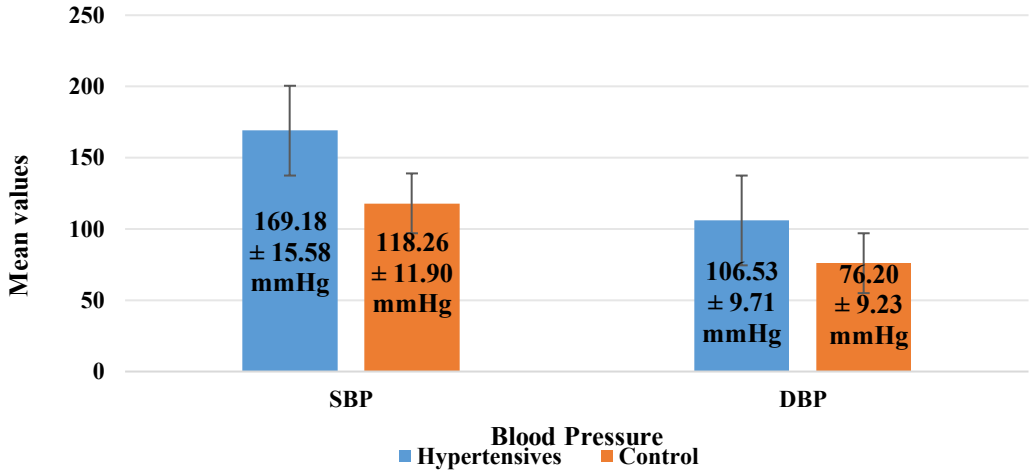
Table 1: Demographic and Social Parameters

Variable	Hypertensive N=200 n (%)	Control N=100 n (%)	Statistics	P –values
Age			F= 68.340	<0.001*
< 40 years	9 (4.5)	30 (30.0)		
40–60 years	96 (48.0)	63 (63.0)		
> 60 years	95 (47.5)	7 (7.0)		
Mean ± SD	58.11 ± 13.03	43.71 ± 10.67	T= 54.903	<0.001*
Modal age	57 years	42 years		
Median age	58yrs	45yrs		
Range	92-19= 73yrs	66-22= 44yrs		
Gender			χ^2 = 7.200	0.007*
Male	53 (26.5)	53 (53.0)		
Female	147 (73.5)	47 (47.0)		
Weight (kg) (Mean ± SD)	76.05 ± 17.96	76.18 ± 9.19	T= 0.005	0.946
Height (m) (Mean ± SD)	1.61 ± 0.08	1.63 ± 0.05	T= 1.600	0.208
BMI (Kg/m²)			F= 3.866	0.276
Underweight	3 (1.5)	2 (2.0)		
Normal	57 (28.5)	18 (18.0)		
Overweight	68 (34.0)	48 (48.0)		
Obese	72 (36.0)	32 (32.0)		
Mean ± SD	28.86 ± 6.44	28.55 ± 4.63	T= 0.114	0.736
Waist circumference (cm) (Mean ± SD)	98.77 ± 13.15	91.23 ± 8.18	T= 16.454	<0.001*
Alcohol intake	33 (16.5)	7 (7.0)	χ^2 = 3.462	0.063
Smokers	0 (0.0)	1 (1.0)	χ^2 = 1.345	0.246

F= Fisher's exact, T= Student's *T*-test, χ^2 = Chi-square, BMI= Body mass index, SD= standard deviation, *=statistically significant (p<0.05)

The mean systolic and diastolic blood pressures for both groups are presented in Figure 1. In the hypertensive group, the mean systolic blood pressure was 169.18 ± 15.58 mmHg compared to 118.26 ± 11.90 mmHg in the control group. Similarly, the mean diastolic blood pressure was 106.53 ± 9.71 mmHg in the hypertensive group and 76.20 ± 9.23 mmHg in the control group. These differences in blood pressure between the two groups were statistically significant (P < 0.001).

Print ISSN: 0189-9287 Online ISSN: 2992-345X



SBP= Systolic blood pressure, DBP = Diastolic blood pressure
SBP (Student *T*-test= 495.139, P< 0.001), DBP (Student *T*-test= 438.814, P< 0.001)

Figure 1: Systolic and diastolic Blood pressure between the two groups

Renal Function

There was a higher serum creatinine level in the hypertensives than in the controls, but the difference was not statistically significant (106.3 ± 94.7 vs. 84.7 ± 14.0 µmol/l respectively, p = 0.095) (Table 2). The mean eGFR was significantly lower in the hypertensives than in the controls (0.73 ± 0.26 vs. 0.86 ± 0.20 mL/s/m^2 respectively, p = 0.001) (Table 2). Figure 3 indicates that 147 individuals (73.5%) in the hypertensive group had normal renal function, whereas 53 (26.5%) had impaired renal function (eGFR< 60mls/m^2/min/1.73 m^2). In contrast, among the control group, 92 (92.0%) had normal results, while 8 (8.0%) had impaired renal function. The difference in the two groups was statistically significant (P= 0.004).

HSCRP

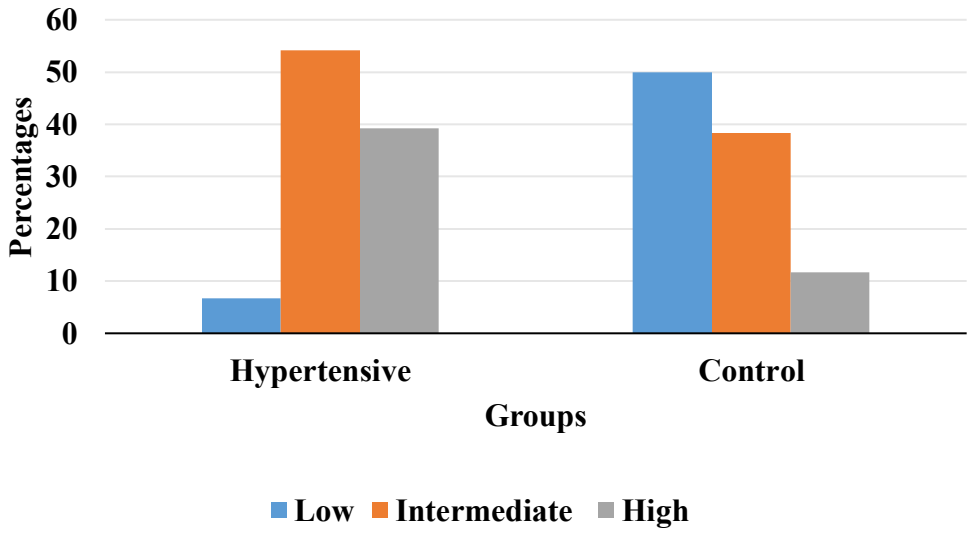
The mean HSCRP in the hypertensives was significantly higher than in the controls (4.22 ± 3.30 vs. 1.51 ± 1.48 mg/L respectively, p < 0.001) (Table 2). In the hypertensive and control groups respectively, 6.7% and 50.0% had low HSCRP, 54.1% and 38.0% had intermediate HSCRP, while 39.2% and 12.0% had high HSCRP. The differences in the two groups were statistically significant (P-value <0.001) (Figure 2).

Table 2: Biochemical variables of study participants

Variable	Hypertensive N=200 n (%)	Control N=100 n (%)	Statistics	P –values
Serum Creatinine (µmol/l)				
Mean ± SD	106.3 ± 94.7	84.7 ± 15.0	T= 2.809	0.095
Minimum	35.4	53.0		
Maximum	831.0	114.9		
eGFR (mL/s/m²)				
Mean ± SD	0.73 ± 0.26	0.86 ± 0.20	T=10.465	0.001*
Minimum	0.05	0.53		
Maximum	1.23	1.20		
HSCRP (mg/L)				
Mean ± SD	4.22 ± 3.30	1.51 ± 1.48	T=38.046	<0.001*
Minimum	0.30	0.50		
Maximum	15.00	7.00		

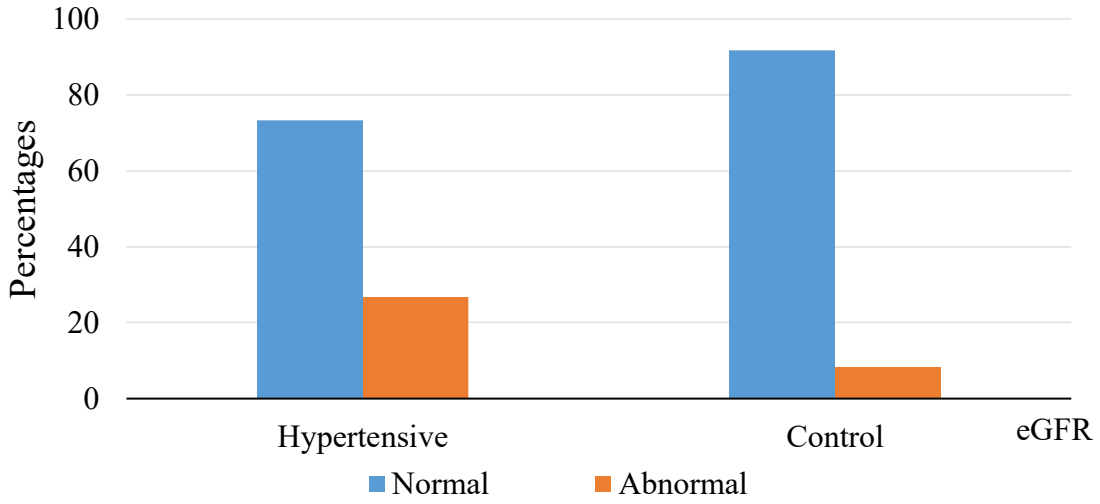
eGFR= Glomerular filtration rate, HSCRP = high-sensitivity C-reactive protein, T= Student's *T*-test, SD= standard deviation, *=statistically significant (p<0.05)

Print ISSN: 0189-9287 Online ISSN: 2992-345X



X^2= 47.713, P< 0.001
Low, intermediate and high Hs-CRP corresponds to < 1 mg/l, 1 to 3 mg/l and > 3 mg/l respectively
Figure 2: Proportion of hypertensive and controls with low, intermediate and high HSCRP

A bar chart comparing the frequency of renal impairment in hypertensive and control groups is shown in Figure 3.



X^2= 8.233, P= 0.004, eGFR= Estimated Glomerular Filtration Rate
Figure 3: Proportion of hypertensive and controls with impaired renal function using eGFR (ml/min/1.73 m^2)

The correlation between high-sensitivity C-reactive protein (HSCRP) and glomerular filtration rate (eGFR) was assessed in both hypertensive patients and controls as shown in Table 3. In the hypertensive group, the Pearson correlation coefficient was -0.142 (p = 0.044), indicating a weak negative correlation that was statistically significant. In the control group, the correlation coefficient was 0.135 (p = 0.178), showing a weak positive but non-significant relationship between HSCRP and eGFR.

Print ISSN: 0189-9287 Online ISSN: 2992-345X

Table 3: Correlation between high sensitivity C-reactive protein with Glomerular filtration rate in hypertensive patients and controls.

Variable	Statistics	Hs-CRP	eGFR
Hypertensive group			
HSCRP	Pearson Correlation	1	-0.142
	Sig. (2-tailed)		0.044*
	N	200	200
eGFR	Pearson Correlation	-0.142	1
	Sig. (2-tailed)	0.044*	
	N	200	200
Control group			
HSCRP	Pearson Correlation	1	0.135
	Sig. (2-tailed)		0.178
	N	100	100
eGFR	Pearson Correlation	0.135	1
	Sig. (2-tailed)	0.178	
	N	100	100

eGFR= Glomerular filtration rate, HSCRP = high-sensitivity C-reactive protein, Correlation is significant at < 0.05 (*)

DISCUSSION

This present study assessed the serum levels of HSCRP and renal function and their relationship in hypertensive patients. Key findings in this study include a significantly lower eGFR and a higher mean HSCRP in hypertensives than in controls. Also, there was a significantly weak negative correlation between HSCRP and eGFR. These findings imply a higher risk of renal disease, cardiovascular disease and chronic low-grade inflammation in hypertensive patients.

Renal dysfunction was present in 26.5% of hypertensive patients in this study and this was significantly higher than in the controls. Prevalence of renal dysfunction in hypertensive patients in other studies reveal a wide range of values. A hospital-based study done in western Nigeria among adult hypertensives revealed a high prevalence of 50%. [20] The prevalence was 35.5% in a population of diabetic hypertensives while a lower prevalence of 18% was recorded in hypertensive patients. [21, 22] The wide disparity in the prevalence of renal impairment noted above can be explained by the many factors that are linked to reduced renal function. They include diabetes, dyslipidaemia, hypertension, age, hyperuricaemia, nephrotoxic drug usage, coronary heart disease and history of chronic kidney disease. [22] These findings can be explained by the role of hypertension in causing deterioration of renal function. [23]

Significant elevation of HSCRP in hypertensive patients as seen in this study, has been reported in several other studies. A Nigerian study among hypertensive patients reported a significantly higher levels of HSCRP in hypertensives than in controls. [24] In a study of 80 participants, HSCRP levels were significantly higher in hypertensive patients. [25] In another study aimed at assessing inflammatory and antioxidant status in hypertensive patients, HSCRP levels were significantly higher than controls. [26] These findings suggest a higher risk of cardiovascular disease in hypertensive patients.

Chronic low-grade inflammation is a characteristic finding in patients with impaired renal function. [6] Thus, in this study, HSCRP had a significant negative correlation with renal function. Similar findings have been reported by several studies. Shu et al, reported a significant correlation between HSCRP and GFR and impaired GFR (GFR < 60 mls/min/1.73 m^2) in a study conducted among 572 hypertensive patients. [27] HSCRP was reported to have a significant indirect relationship with renal function in a population of hypertensive patients. [28] Also, there was a negative correlation between CRP and eGFR in a case-control study involving chronic kidney disease patients in Nigeria. [29]

Research in a large non-diabetic population reported that elevated CRP levels were positively associated with renal impairment. [14] In myocardial infarction (MI) patients, elevated HSCRP was documented as an independent predictor for acute kidney injury (AKI). [30] Similarly, in a study of post-MI patients, elevated HSCRP was associated with risk of AKI and CKD progression. [31]

Print ISSN: 0189-9287 Online ISSN: 2992-345X

The limitations in this study include the significant disparity in the age and sex distribution between the hypertensives and controls, which can act as potential confounders. Also, being a case-control study, it is limited in its ability to establish a causal relationship between HSCRP and impaired renal function.

CONCLUSION

Hypertensive patients demonstrated significantly higher HSCRP levels and a greater prevalence of impaired renal function compared with normotensive controls. Furthermore, a weak but statistically significant negative correlation between HSCRP and eGFR was observed among the hypertensive group, suggesting that increasing inflammatory burden may be associated with declining kidney function. These findings underscore the need for early detection of subclinical renal impairment and inflammation in individuals with hypertension. Further research focused on the effect of therapies that control chronic low-grade inflammation on renal function is recommended.

DECLARATIONS

Authors' contribution: AOE, SOO and PEA made contributions to conception, design, experimentation, acquisition and interpretation of data and writing of manuscript. AOE and SOO made substantial contribution in interpretation of data and revising the manuscript. All authors read and approved the final manuscript.

Conflict of interest: The authors declare that they have no conflict of interest concerning this article.

Funding: Self-funded

Acknowledgement: The authors are grateful to the staff of the General Outpatient Clinic and Cardiology unit of the Delta State Central Hospital, Warri.

REFERENCES

1. Oparil S, Acelajado MC, Bakris GL, Berlowitz DR, Cifkova R, Dominiczak AF, et al. Hypertension. Nat Rev Dis Primers. 2018;4, 18014. Doi: 10.1038/nrdp.2018.14
2. Cachofeiro V, Miana M, Heras N, Martin Fernandez B, Ballesteros S, Balfagon G, et al. Inflammation: a link between hypertension and atherosclerosis. Curr Hypertens Rev. 2009; 5(1):40-48. Doi: https://doi.org/10.2174/157340209787314333.
3. Delrue C, Speeckaert MM. Beyond blood pressure: emerging pathways and precision approaches in hypertension-induced kidney damage. Int J Mol Sci. 2025; 26(15):7606. Doi: 10.3390/ijms26157606.
4. Maranduca MA, Clim A, Pinzariu AC, Statescu C, Sascau RA, Tanase DM, et al. Role of arterial hypertension and angiotensin II in chronic kidney disease. Exp Ther Med. 2023; 25(4): 153. Doi: 10.3892/etm.2023.11852.
5. Patidar OP, Bhargava AK, Gupta D. Association of C-reactive protein and arterial hypertension. Int J Adv Med. 2015; 2(2):133-137. Doi: 10.5455/2349-3933.ijam20150511
6. Mihai S, Codrici E, Popescu LD, Enciu A-M, Albulescu L, Georgiana L, et al. Inflammation related mechanisms in chronic kidney disease prediction, progression, and outcome. J Immunol Res. 2018; 2018:2180373. Doi: 10.1155/2018/2180373.
7. Rahali FZ, Mimouni N, Boukhira A, Chellak S. The clinical utility of standard and high-sensitivity C - reactive protein: a narrative review. SN Compr Clin Med. 2024; 6(1). Doi: 10.1007/s42399-024-01693-3
8. Yousuf O, Mohanty BD, Martin SS, Joshi PH, Blaha MJ, Nasir K, et al. High-sensitivity C reactive protein and cardiovascular disease: a resolute belief or an elusive link? J Am Coll Cardiol. 2013; 62(5):397-408. Doi: 10.1016/j.jacc.2013.05.016.
9. Sudjaroen Y. High sensitivity C - reactive protein level and lipid profiles of healthy volunteers with prehypertension. Sci Res Essays. 2015; 10(4):127-131. Doi: 10.5897/SRE2015.6147.
10. Liu HH, Cao YX, Sun D, Jin JL, Zhang HW, Guo YL, et al. High-sensitivity C-reactive protein and hypertension: combined effects of coronary severity and cardiovascular outcomes. Hypertens Res. 2019; 42(11): 1783-1793. Doi: 10.1038/s41440-019-0293-8.
11. Banait T, Wanjari A, Danade V, Banait S, Jain J. Role of high-sensitivity C-reactive protein in non-communicable diseases: a review. Cureus. 2022; 14(10):e30225. Doi: 10.7759/cureus.30225
12. Kadatane SP, Satariano M, Massey M, Mongan K, Raina R. The role of inflammation in CKD. Cells. 2023; 12(12): 1581. Doi: 10.3390/cells12121581.
13. Vidt DG. Inflammation in renal disease. Am J Cardiol. 2006; 97(2A):20A-27A. Doi: 10.1016/j.amjcard.2005.11.012.
14. Stuveling EM, Hillege HL, Bakker SJL, Gans ROB, de Jong PE, de Zeeuw D. C-reactive protein is associated with renal function abnormalities in a non-diabetic population.

Print ISSN: 0189-9287 Online ISSN: 2992-345X

Kidney Int. 2003; 63(2): 654-661. Doi: 10.1046/j.1523-1755.2003.00762-x.
15. Li J, Chen J, Lan H-Y, Tang Y. Role of C-reactive protein in kidney diseases. Kidney Dis (Basel). 2022; 9(2):73-81. Doi: 10.1159/000528693.
16. Adukauskiene D, Ciginskiene A, Adukauskaite A, Pentiokiniene D, Slapikas R, Ceponiene I. Clinical relevance of high sensitivity C-reactive protein in cardiology. Medicina (Kaunas). 2016; 52(1):1-10. Doi: 10.1016/j.medici.2015.12.001.
17. Warwick J, Holness J. Measurement of glomerular filtration rate. Semin Nucl Med. 2022; 52(4):453-466. Doi: 10.1053/j.semnuclmed.2021.12.005.
18. Rispoli RM, Popolo A, De Fabrizio V, d'Emmanuele di Villa Bianca R, Autore G, Dalli J, et al. Targeting inflammatory imbalance in chronic kidney disease: focus on anti-inflammatory and resolution medication. Int J Mol Sci. 2025, 26(7): 3072. Doi: 10.3390/ijms26073072.
19. Levey AS, Greene T, Kusek J, Beck G. A simplified equation to predict glomerular filtration rate from serum creatinine. J Am Soc Nephrol. 2000; 11:155A.
20. Oluwademilade OJ, Ajani GO, Kolawole FT, Obajolowo OO, Olabinri EO, Agboola SM. Burden of chronic kidney disease in hypertensive patients in medical outpatient clinic of a rural tertiary hospital. J Hypertens Manag. 2020; 6:049. Doi.org/10.23937/2474-3690/1510049
21. Sweileh WB, Sawalha AF, Zyoud SH, Al-Jabil SW, Shraim NY. Prevalence of reduced renal function among diabetic hypertensive patients. Int J Physiol Pathophysiol Pharmacol. 2009; 1:41-47. PMID: 21383877; PMCID: PMC3040939.
22. Liu Q, Li Z, Wang H, Chen X, Dong X, Mao H, et al. High prevalence and associated risk factors for impaired renal function and urinary abnormalities in a rural population from southern China. PLoS One. 2012; 7(10):e47100. PMID: 23056593; PMCID: PMC3467213.
23. Burnier M, Damianaki A. Hypertension as cardiovascular risk factor in chronic kidney disease. Circ Res. 2023; 132(8): 1050-1063. Doi: 10.1161/CIRCRESAHA.122-321762.
24. Idemudia JO, Idogun ES. High sensitivity C reactive protein as a cardiovascular risk factor in hypertensive Nigerians. Niger Postgrad Med J. 2012; 19(3):163-166. PMID: 23064173.
25. Abd El Aziz WF, Abou Elnour ES, Mena MB, EL-Brol RM. A comparative analysis of the level of high-sensitivity C-reactive protein in individuals with and without hypertension. Menoufia Med J. 2019; 32: 187-193. Doi: 10.4103/mmj.mmj_210_15.
26. Kuklinska AM, Mroczko B, Musial WJ, Sawicki R, Kozieradzka A, Waszkiewicz E, Szmitkowski M. High-sensitivity C-reactive protein and total antioxidant status in patients with essential arterial hypertension and dyslipidemia. Adv Med Sci. 2009; 54(2): 225-232. Doi: 10.2478/v10039-009-0052-1.
27. Shu H-S, Tai Y-Y, Chang K-T, Chu C-Y, Hsu PC, Su H-M, et al. Plasma high-sensitivity C reactive protein level is associated with impaired glomerular filtration rate in hypertensives. Acta Cardiol Sin. 2015; 31(2): 91-97. Doi: 10.6515/acs20140630c.
28. Hommels MJ, van der Ven AJAM, Kroon AA, Kessels AGH, van Dieijen-Visser MP, van Engelshoven JAM, et al. C-reactive protein, atherosclerosis and kidney function in hypertensive patients. J Hum Hypertens. 2005; 19(7): 521-526. Doi: 10.1038/sj.jhh.1001878.
29. Adejumor OA, Okaka EI, Okwuonu CG, Iyawe IO, Odujoko OO. Serum C-reactive protein levels in pre-dialysis chronic kidney disease patients in southern Nigeria. Ghana Med J. 2016; 50(1):31-38. Doi: 10.4314/gmj.v50i1.5.
30. Shacham Y, Leshem-Rubinow E, Steinvil A, Keren G, Roth A, Arbel Y. High sensitivity C reactive protein and the risk of acute kidney injury among ST elevation myocardial infarction patients undergoing primary percutaneous intervention. Clin Exp Nephrol. 2014; 19(5):838-843. Doi: 10.1007/s10157-014-1071-1.
31. Fu EL, Franko MA, Obergfell A, Dekker FW, Gabrielsen A, Jernberg T, et al. High-sensitivity C-reactive protein and the risk of chronic kidney disease progression or acute kidney injury in post myocardial infarction patients. Am Heart J. 2019; 216:20-29. Doi: 10.1016/j.ahj.2019.06.019.

Print ISSN: 0189-9287 Online ISSN: 2992-345X