RESEARCH PAPER

C-Reactive Protein and Hypertension Grade in Newly Detected, Treatment-Naïve Adults: A Case-Control Study

Dr. Rudra Dutt Kaushik[1], Dr. Aman Ansari[2], Dr. Deepak Sharma[3*], Dr. Rishabh Ahlawat[4], Dr. Nivedita Sharma[5]

[1]*Postgraduate Resident, Department of General Medicine, School of Medical Sciences & Research, Sharda Hospital, Greater Noida, Uttar Pradesh, India*
[2]*Postgraduate Resident, Department of General Medicine, School of Medical Sciences & Research, Sharda Hospital, Greater Noida, Uttar Pradesh, India*
[3*]*Professor & Head, Department of General Medicine, School of Medical Sciences & Research, Sharda Hospital, Greater Noida, Uttar Pradesh, India*
[4]*Postgraduate Resident, Department of General Medicine, School of Medical Sciences & Research, Sharda Hospital, Greater Noida, Uttar Pradesh, India*
[5]*Postgraduate Resident, Department of General Medicine, School of Medical Sciences & Research, Sharda Hospital, Greater Noida, Uttar Pradesh, India*

ABSTRACT

Background: Systemic inflammation is increasingly recognised as a contributor to hypertension pathophysiology. C-reactive protein (CRP), a widely available inflammatory marker, has been associated with elevated blood pressure in various populations, yet data on newly detected, treatment-naïve hypertensive individuals remain limited, particularly in Indian settings.

Aim: To evaluate CRP levels in newly detected hypertensive adults compared with normotensive controls and to assess the relationship between CRP and hypertension grade according to the European Society of Hypertension (ESH) 2023 classification.

Methods: This case- control observational study was conducted at Sharda Hospital, Greater Noida, from April 2024 to November 2025. A total of 180 participants were enrolled: 90 newly detected, treatment-naïve hypertensive cases and 90 age- and sex-matched normotensive controls. Hypertension was defined and graded using ESH 2023 criteria. CRP was measured using immunoturbidimetric assay. Statistical analyses included independent t-tests for group comparisons, Pearson correlation for CRP-blood pressure relationships, one-way ANOVA for CRP across hypertension grades, and Spearman correlation for ordinal trend assessment.

Results: Hypertensive cases demonstrated markedly higher CRP levels than controls (26.98 ± 4.54 vs 5.14 ± 2.37 mg/L; $p < 0.001$). CRP showed strong positive correlations with both systolic blood pressure ($r = 0.921$, $p < 0.001$) and diastolic blood pressure ($r = 0.858$, $p < 0.001$). Among hypertensive cases, CRP increased progressively across ESH grades: Grade 1 (21.83 ± 2.27 mg/L), Grade 2 (28.56 ± 2.78 mg/L), and Grade 3 (35.60 ± 2.86 mg/L), with significant differences confirmed by ANOVA ($F = 89.128$, $p < 0.001$). Spearman correlation demonstrated a strong ordinal association between hypertension grade and CRP ($\rho = 0.772$, $p < 0.001$).

Conclusion: In newly detected, treatment-naïve adults, CRP levels are significantly elevated in hypertensive individuals compared with normotensive controls and increase progressively with hypertension severity. These findings support an inflammatory association with hypertension and suggest CRP may serve as a marker of disease severity in early hypertension.

Keywords: C-reactive protein; hypertension; inflammation; ESH 2023; blood pressure; newly detected hypertension

How to cite this article: Kaushik RD, Ansari A, Sharma D, Ahlawat R, Sharma N. C-Reactive Protein and Hypertension Grade in Newly Detected, Treatment-Naïve Adults: A Case-Control Study. Int J Drug Deliv Technol. 2026;16(21s): 537-548. DOI: 10.25258/ijddt.16.21s.57

Source of support: Nil.

Conflict of interest: Nil.

INTRODUCTION

Systemic arterial hypertension is a highly prevalent chronic condition affecting over 1.28 billion adults worldwide, with approximately two-thirds residing in low- and middle-income countries [1,2]. In India, the burden has escalated dramatically, with urban populations showing higher prevalence rates than rural areas [3]. Beyond its haemodynamic consequences, hypertension is increasingly understood as a systemic disorder involving inflammatory processes that contribute to vascular dysfunction and end-organ damage [4,5].

The role of inflammation in hypertension pathogenesis has gained substantial attention. Chronic low-grade inflammation promotes endothelial dysfunction by

**Author for Correspondence:* Dr Deepak Sharma

reducing nitric oxide bioavailability, increasing oxidative stress, and activating pro-inflammatory pathways [6,7]. These processes contribute to arterial stiffness, vascular remodelling, and sustained elevation of blood pressure. Among the inflammatory markers implicated in cardiovascular disease, C-reactive protein (CRP) has emerged as a clinically accessible and well-validated biomarker [8,9].

CRP is an acute-phase reactant synthesised primarily in the liver in response to interleukin-6 (IL-6) and other pro-inflammatory cytokines [10]. Beyond its role as a passive marker of inflammation, CRP exerts direct effects on vascular biology, including inhibition of endothelial nitric oxide synthase, promotion of endothelin-1 release, and enhancement of monocyte adhesion to endothelial cells [11,12]. Clinical studies have demonstrated associations between elevated CRP and incident hypertension, cardiovascular events, and mortality [13,14].

The relationship between CRP and hypertension severity has been explored in various populations. Several studies have reported that CRP levels increase with advancing blood pressure stage [15,16]. However, evidence specifically addressing newly detected, treatment-naïve hypertensive individuals remains limited, particularly in Indian populations where genetic, dietary, and environmental factors may influence inflammatory profiles [17]. Furthermore, few studies have employed contemporary classification systems such as the European Society of Hypertension (ESH) 2023 guidelines for hypertension staging [18].

Understanding the inflammatory burden at the time of initial hypertension detection, before therapeutic intervention, may provide insights into early pathophysiological processes and identify individuals at higher cardiovascular risk. The present study aimed to evaluate CRP levels in newly detected hypertensive adults compared with normotensive controls and to assess the relationship between CRP concentration and hypertension grade according to ESH 2023 criteria.

METHODS

Study Design and Setting: This case- control observational study was conducted in the Department of General Medicine, School of Medical Sciences & Research, Sharda Hospital, Greater Noida, Uttar Pradesh, India, from April 2024 to November 2025. The study protocol was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrolment.

Participants: Consecutive adults attending the Medicine outpatient and inpatient departments who met eligibility criteria were screened and enrolled. The case group comprised individuals with newly detected hypertension who were treatment-naïve (no prior antihypertensive therapy). The control group consisted of age- and sex-matched normotensive individuals. Hypertension was diagnosed and staged according to ESH 2023 guideline thresholds.

Inclusion criteria were: (1) age ≥18 years; (2) newly detected hypertension (cases) or normotension (controls) as defined by ESH 2023 criteria; and (3) no prior antihypertensive medication use.

Exclusion criteria were: (1) current antihypertensive therapy; (2) diabetes mellitus; (3) chronic kidney disease or other renal disorders; (4) active infection or fever within two weeks; (5) known inflammatory, autoimmune, or haematological disease; (6) malignancy; (7) complications of hypertension including heart failure, coronary artery disease, stroke, or peripheral arterial disease; (8) moderate or severe valvular heart disease; and (9) pregnancy or lactation.

Sample Size: Sample size was calculated using the Cochran formula for prevalence studies. With $Z = 1.96$, estimated hypertension prevalence (p) = 0.31, and margin of error (e) = 0.07, the minimum required sample size was 166 participants. To account for potential non-response and to enable balanced case-control comparison, we planned and recruited 180 participants (90 cases and 90 controls).

Clinical Measurements: Blood pressure was measured using a calibrated sphygmomanometer with participants seated and rested for at least five minutes. Two readings were obtained over the right arm, separated by five minutes, and the average was recorded. Hypertension grades were assigned according to ESH 2023 criteria: Grade 1 (systolic 140-159 mmHg or diastolic 90-99 mmHg), Grade 2 (systolic 160-179 mmHg or diastolic 100-109 mmHg), and Grade 3 (systolic ≥180 mmHg or diastolic ≥110 mmHg). Height and weight were measured, and body mass index (BMI) was calculated.

Laboratory Procedures: Venous blood samples were collected from the antecubital vein using atraumatic technique after overnight fasting. Serum was separated by centrifugation and stored appropriately until analysis. CRP was measured using the immunoturbidimetric Tina-quant CRP method according to manufacturer instructions, with results expressed in mg/L. Fasting plasma glucose and standard biochemical parameters were also measured.

Statistical Analysis: Data were analysed using IBM SPSS version 25 and Microsoft Excel. Continuous variables were summarised as mean ± standard deviation (SD). Categorical variables were expressed as frequencies and percentages. Between-group comparisons for continuous variables were performed using independent samples t-test. Chi-square test was used for categorical comparisons. Pearson correlation coefficient was calculated to assess linear relationships between CRP and blood pressure. One-way analysis of variance (ANOVA) was used to compare CRP across hypertension grades. Spearman rank correlation was employed to assess the ordinal association between hypertension grade and CRP. A two-sided p-value < 0.05 was considered statistically significant.

RESULTS

Participant Characteristics: A total of 180 participants were enrolled, comprising 90 hypertensive cases and 90 normotensive controls (Figure 1). Baseline characteristics are presented in Table 1. Cases were significantly older than controls (53.26 ± 5.56 vs 44.78 ± 8.10 years; $p < 0.001$). The proportion of males was higher among cases (53.33%) than controls (77.78%), with a significant sex difference

between groups (χ^2 = 11.91, p < 0.001). Cases had significantly greater body weight (76.54 ± 11.11 vs 70.32 ± 4.50 kg; p < 0.001) and BMI (27.1 ± 3.2 vs 25.1 ± 1.8 kg/m²; p < 0.001) compared with controls.

Mean systolic blood pressure was markedly higher in cases than controls (164.36 ± 10.93 vs 120.71 ± 2.96 mmHg; p < 0.001), as was diastolic blood pressure (96.17 ± 5.52 vs 78.42 ± 1.48 mmHg; p < 0.001), confirming the validity of group classification. Among the 90 hypertensive cases, 27 (30.0%) were classified as Grade 1, 58 (64.4%) as Grade 2, and 5 (5.6%) as Grade 3 hypertension according to ESH 2023 criteria (Table 2).

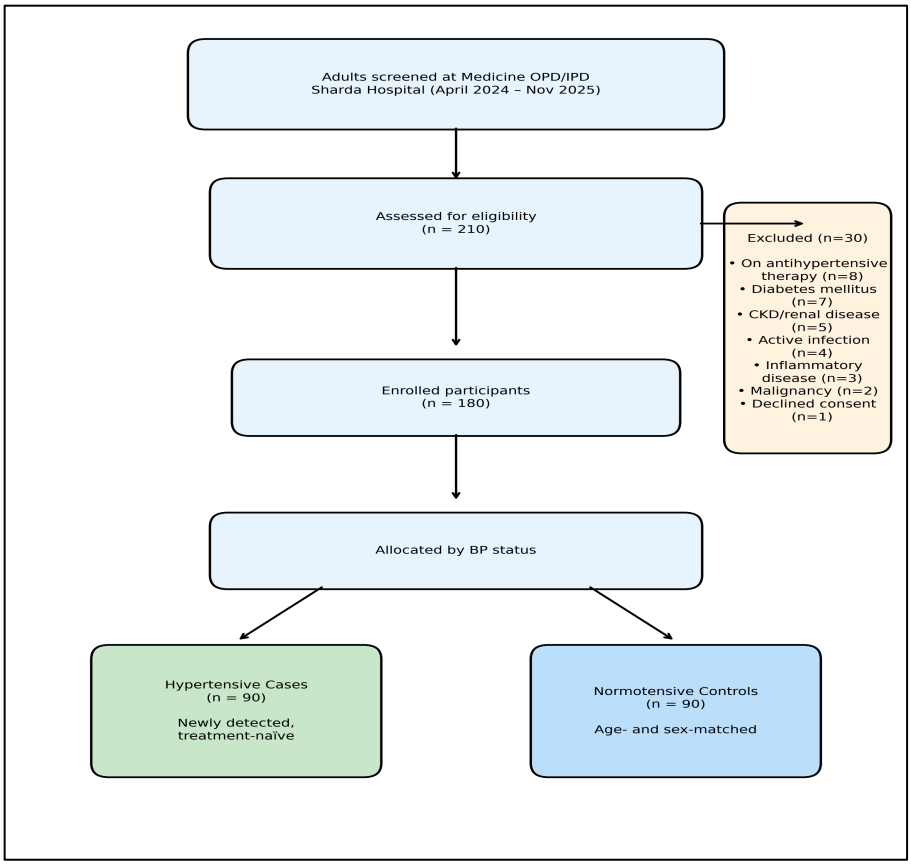

Figure 1. Study flow diagram showing participant screening, exclusions, and final group allocation.

Table 1. Participant characteristics by study group

Characteristic	Cases (n=90)	Controls (n=90)	p-value
Age (years), mean ± SD	53.26 ± 5.56	44.78 ± 8.10	< 0.001
Male sex, n (%)	48 (53.33%)	70 (77.78%)	< 0.001
BMI (kg/m²), mean ± SD	27.1 ± 3.2	25.1 ± 1.8	< 0.001
SBP (mmHg), mean ± SD	164.36 ± 10.93	120.71 ± 2.96	< 0.001
DBP (mmHg), mean ± SD	96.17 ± 5.52	78.42 ± 1.48	< 0.001
FPG (mg/dL), mean ± SD	113.14 ± 16.76	79.09 ± 3.75	< 0.001

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose.

Table 2. Distribution of participants by ESH 2023 blood pressure category

ESH 2023 Category	Cases, n (%)	Controls, n (%)
Normal	0 (0.0%)	90 (100.0%)
Grade 1 Hypertension	27 (30.0%)	0 (0.0%)
Grade 2 Hypertension	58 (64.4%)	0 (0.0%)
Grade 3 Hypertension	5 (5.6%)	0 (0.0%)

CRP Levels: Cases versus Controls: CRP levels were markedly elevated in hypertensive cases compared with normotensive controls (Table 3, Figure 2). The mean CRP concentration in cases was 26.98 ± 4.54 mg/L (range 18-40 mg/L) versus 5.14 ± 2.37 mg/L (range 2-8 mg/L) in controls. This difference was highly statistically significant ($t = 40.461$, $p < 0.001$), indicating a substantially greater inflammatory burden among newly detected hypertensive individuals.

Table 3. CRP distribution by study group

Statistic	Cases (n=90)	Controls (n=90)	Overall (n=180)
Mean ± SD (mg/L)	26.98 ± 4.54	5.14 ± 2.37	16.06 ± 11.53
Minimum	18	2	2
Maximum	40	8	40
t-statistic	40.461	—	—
p-value	< 0.001	—	—

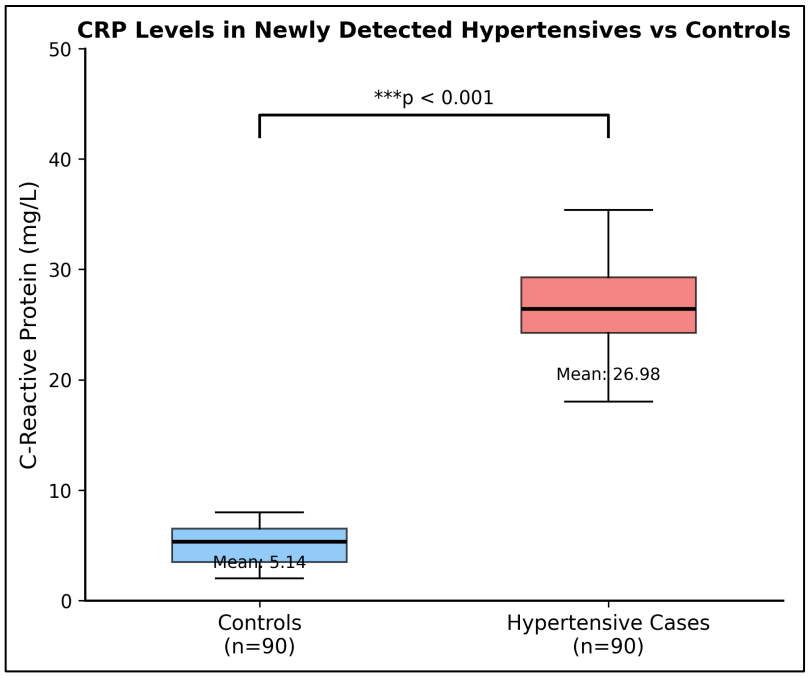

Figure 2. Box plot showing CRP levels in hypertensive cases versus normotensive controls.

Correlation of CRP with Blood Pressure: CRP demonstrated strong positive correlations with both systolic and diastolic blood pressure across all participants (Table 4, Figures 3-4). The Pearson correlation coefficient between CRP and systolic blood pressure was r = 0.921 ($p < 0.001$), indicating a very strong linear relationship. Similarly, CRP correlated strongly with diastolic blood pressure (r = 0.858, $p < 0.001$). These findings indicate that higher inflammatory burden, as reflected by CRP, is closely associated with greater blood pressure elevation.

Table 4. Correlation of CRP with blood pressure parameters

Blood Pressure Parameter	Pearson r	p-value
Systolic BP (mmHg)	0.921	< 0.001
Diastolic BP (mmHg)	0.858	< 0.001

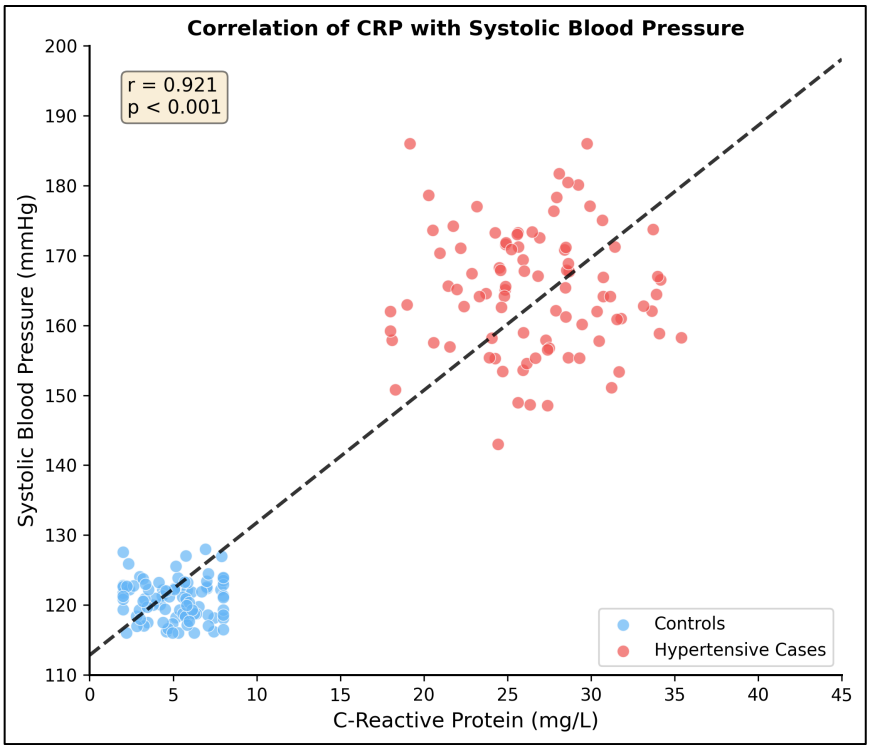

Figure 3. Scatter plot showing the correlation between CRP and systolic blood pressure. Blue circles represent controls; red circles represent hypertensive cases. The dashed line represents the fitted regression line.

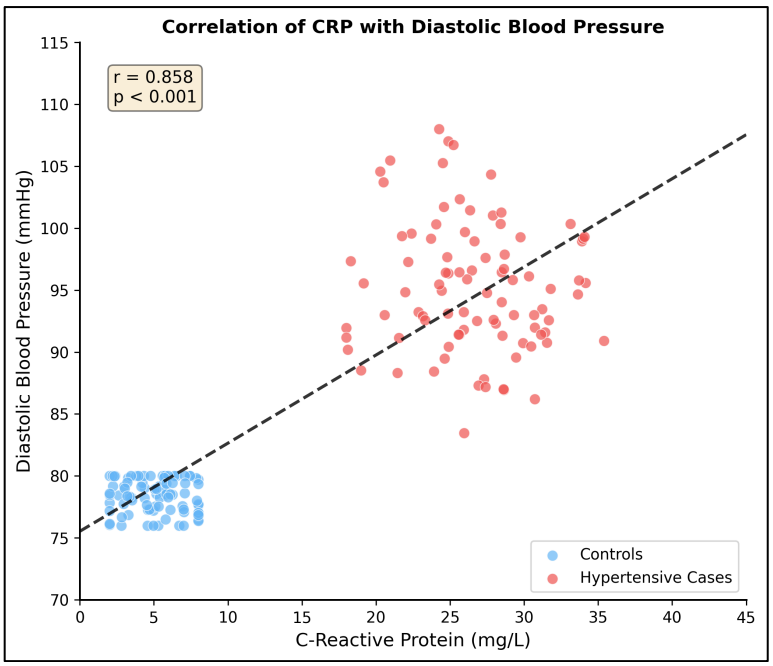

Figure 4. Scatter plot showing the correlation between CRP and diastolic blood pressure. Blue circles represent controls; red circles represent hypertensive cases. The dashed line represents the fitted regression line.

CRP Across Hypertension Grades: Among hypertensive cases, CRP levels increased progressively with advancing ESH 2023 grade (Table 5, Figure 5). Mean CRP was 21.83 ± 2.27 mg/L in Grade 1, 28.56 ± 2.78 mg/L in Grade 2, and 35.60 ± 2.86 mg/L in Grade 3 hypertension. One-way ANOVA confirmed statistically significant differences across grades ($F = 89.128$, $p < 0.001$). The Spearman correlation coefficient between hypertension grade (ordinal) and CRP was $\rho = 0.772$ ($p < 0.001$), demonstrating a strong monotonic trend of increasing CRP with more severe hypertension (Table 6).

Table 5. CRP levels by ESH 2023 hypertension grade among cases

ESH Grade	n	Mean ± SD	Min	Max
Grade 1	27	21.83 ± 2.27	18	24
Grade 2	58	28.56 ± 2.78	24	32
Grade 3	5	35.60 ± 2.86	32	40

ANOVA: F = 89.128, p < 0.001. CRP values in mg/L.

Table 6. Ordinal association between hypertension grade and CRP

Analysis	Spearman ρ	p-value
CRP vs Hypertension Grade (Grades 1-3)	0.772	< 0.001

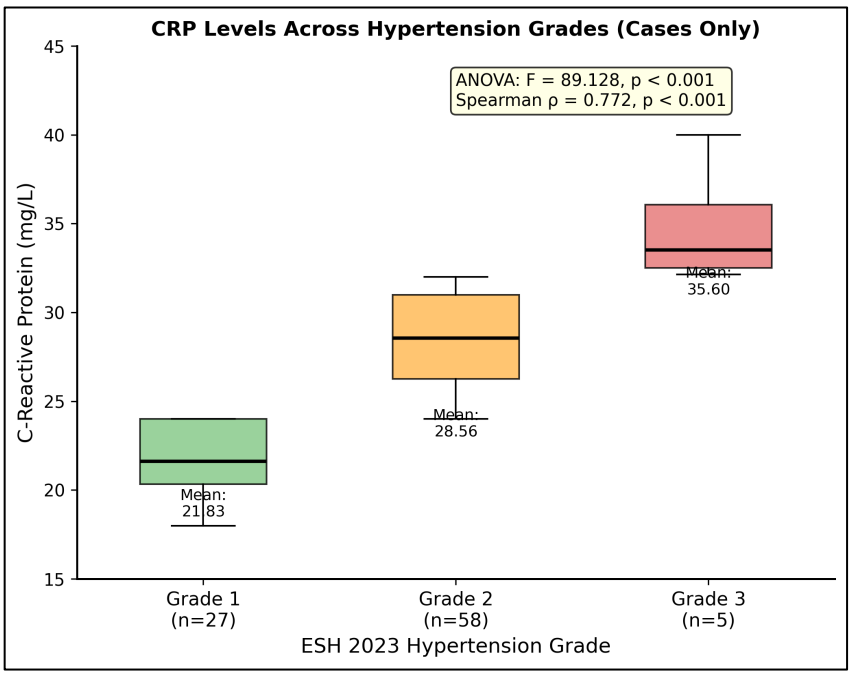

Figure 5. Box plot showing CRP levels across ESH 2023 hypertension grades among cases. CRP increases progressively from Grade 1 to Grade 3.

Sensitivity Analysis: When normotensive controls were included as a 'Normal' category, the analysis across all four blood pressure categories (Normal, Grade 1, Grade 2, Grade 3) revealed a continuous inflammatory gradient (Table 7, Figure 6). Mean CRP values were 5.14 ± 2.37 mg/L (Normal), 21.83 ± 2.27 mg/L (Grade 1), 28.56 ± 2.78 mg/L (Grade 2), and 35.60 ± 2.86 mg/L (Grade 3). ANOVA confirmed highly significant differences (F = 543.186, p = 0.002), and the Spearman correlation across all four categories was exceptionally strong (ρ = 0.907, p < 0.001).

Table 7. Sensitivity analysis: CRP across Normal and Grades 1-3

Category	n	Mean ± SD	Min	Max
Normal	90	5.14 ± 2.37	2	8
Grade 1	27	21.83 ± 2.27	18	24

Grade 2	58	28.56 ± 2.78	24	32
Grade 3	5	35.60 ± 2.86	32	40

ANOVA: F = 543.186, p = 0.002. Spearman ρ = 0.907, p < 0.001. CRP values in mg/L.

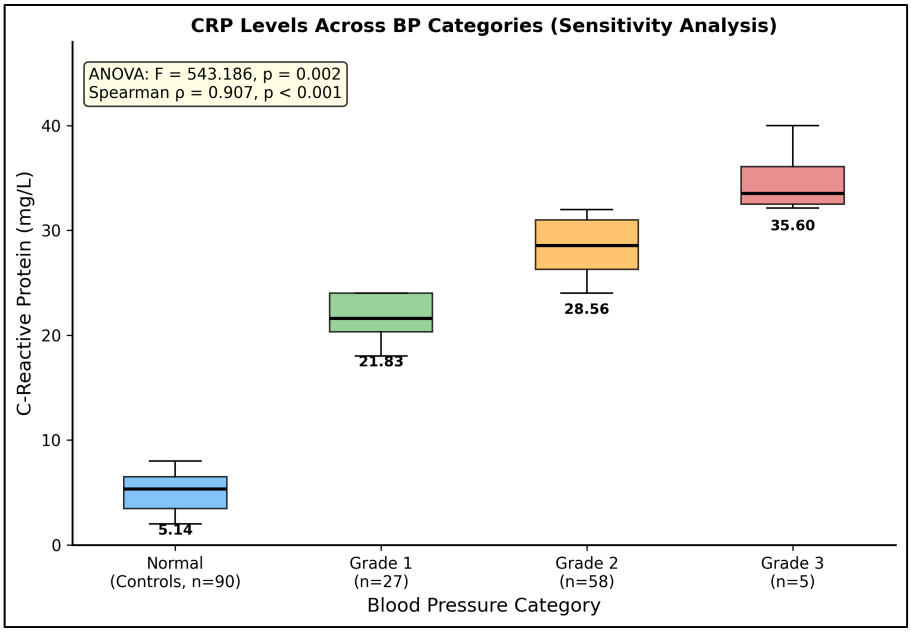

Figure 6. Sensitivity analysis showing CRP levels across Normal (controls) and hypertension Grades 1-3. A continuous inflammatory gradient is evident from normotension through severe hypertension.

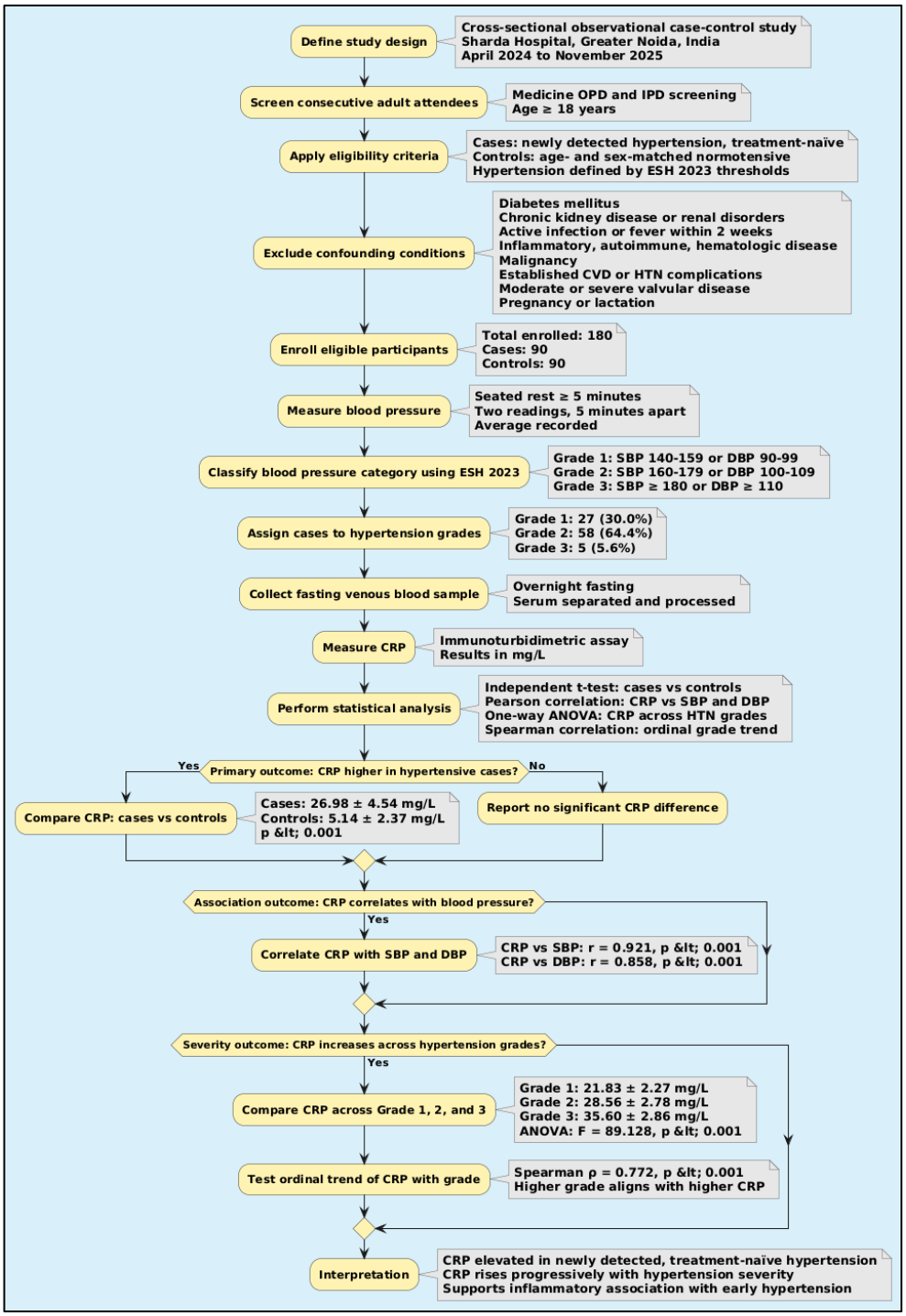

Figure 6 – Study Flow and Outcomes for C-Reactive Protein and Hypertension Grade in Newly Detected, Treatment-Naive Adults

DISCUSSION

This study demonstrates that CRP levels are markedly elevated in newly detected, treatment-naïve hypertensive adults compared with normotensive controls, and that CRP concentration increases progressively with hypertension severity as defined by ESH 2023 grades. The strong correlations between CRP and both systolic and diastolic blood pressure, coupled with the clear graded relationship across hypertension stages, support an association between systemic inflammation and blood pressure elevation at the earliest clinical presentation of hypertension.

Principal Findings: The primary finding of substantially elevated CRP in hypertensive cases (26.98 ± 4.54 mg/L) compared with controls (5.14 ± 2.37 mg/L) indicates a pronounced inflammatory burden at the time of initial hypertension detection. This five-fold elevation suggests that systemic inflammation is not merely a consequence of longstanding hypertension or antihypertensive medication effects, but rather accompanies the early stages of

hypertensive disease. The very strong correlations with systolic (r = 0.921) and diastolic (r = 0.858) blood pressure further support a close relationship between inflammatory activity and blood pressure magnitude.

Comparison with Previous Studies: Our findings align with and extend previous research on CRP and hypertension. He et al. (2022) [19], studying 120 elderly hypertensive patients and 120 controls, reported mean high-sensitivity CRP of 4.62 ± 1.78 mg/L in hypertensives versus 2.31 ± 1.12 mg/L in normotensives ($p < 0.01$), with moderate correlations between CRP and systolic blood pressure (r = 0.47) and diastolic blood pressure (r = 0.42). While our study demonstrated higher absolute CRP values and stronger correlations, both investigations confirm elevated CRP in hypertension. The higher values in our cohort may reflect differences in assay methodology, population characteristics, or the specific focus on newly detected cases.

Dzieża-Grudnik et al. (2023) [20] examined 101 newly diagnosed hypertensive subjects and 49 normotensive controls, reporting median CRP of 3.2 mg/L (IQR 2.1-4.9) in hypertensives versus 1.4 mg/L (IQR 0.9-2.3) in controls ($p < 0.001$). They observed moderate correlations of CRP with systolic pressure (r = 0.38) and diastolic pressure (r = 0.28). Importantly, their study also documented a progressive increase in CRP across blood pressure categories, with median values rising from 2.9 mg/L in prehypertension to 4.7 mg/L in Stage 2 hypertension. Our findings of a strong ordinal trend (Spearman $\rho = 0.772$) are consistent with this graded pattern.

Kansui et al. (2019) [21] provided longitudinal evidence linking baseline CRP to incident hypertension in a cohort of 1,012 middle-aged Japanese men. They demonstrated that elevated CRP at baseline predicted the development of hypertension over follow-up, suggesting a potential role for inflammation in hypertension pathogenesis rather than merely as a consequence. CRP elevation at initial hypertension detection is consistent with inflammation being present early in the disease process.

Zhang et al. (2019) [22] evaluated CRP among 80 hypertensive patients with cerebral infarction, reporting very strong correlations between CRP and vascular injury parameters. Their findings that hs-CRP correlated with both systolic pressure (r = 0.89) and infarct size (r = 0.97) in certain subgroups suggest that inflammatory burden may relate to end-organ damage beyond blood pressure elevation alone. Our strong correlations (r = 0.858-0.921) are comparable and support the close relationship between CRP and haemodynamic parameters.

Shukla et al. (2025) [23] examined inflammatory indices in 100 newly detected hypertensive patients and 100 controls in an Indian setting. Although they focused on the neutrophil-to-lymphocyte ratio (NLR) rather than CRP specifically, they observed progressive increases in NLR across hypertension stages (mean NLR of 2.13 ± 0.78 in Stage 1 versus 3.42 ± 1.02 in Stage 2; $p < 0.001$), with a significant ordinal trend (Spearman $\rho = 0.542$). This parallel finding using a different inflammatory marker supports the concept of stage-dependent inflammatory activation in hypertension.

Pathophysiological Considerations: The association between CRP and hypertension may be explained by several mechanisms. CRP is synthesised in the liver primarily in response to IL-6, a key mediator of systemic inflammation [10]. Beyond serving as a marker, CRP has direct vascular effects including inhibition of endothelial nitric oxide synthase, reduction of prostacyclin release, and promotion of endothelin-1 production [11,12]. These actions promote vasoconstriction and reduce vasodilatory capacity, potentially contributing to elevated blood pressure.

Furthermore, CRP promotes adhesion molecule expression on endothelial cells, facilitating monocyte recruitment and vascular inflammation [24]. This chronic inflammatory state contributes to arterial stiffness and vascular remodelling, both of which are implicated in hypertension pathophysiology [6,7]. The progressive increase in CRP across hypertension grades observed in our study suggests that inflammatory burden escalates with disease severity, potentially reflecting a bidirectional relationship where inflammation promotes hypertension and hypertension further enhances inflammation.

Clinical Implications: The marked CRP elevation in newly detected hypertensives has potential clinical implications. CRP measurement is widely available, inexpensive, and standardised across laboratories. Our findings suggest that CRP may serve as a readily accessible marker of inflammatory burden at hypertension diagnosis. While this study demonstrates association rather than causation, the graded relationship with hypertension severity raises the possibility that CRP could complement blood pressure measurement in risk stratification. It is important to note that CRP is a non-specific marker elevated in various inflammatory conditions. Our exclusion criteria attempted to minimise confounding by excluding individuals with active infection, known inflammatory diseases, and other conditions affecting CRP. However, residual confounding cannot be entirely excluded in observational studies.

Strengths and Limitations: Strengths of this study include the focus on newly detected, treatment-naïve hypertensive individuals, which eliminates confounding by antihypertensive medications that may affect inflammatory markers. The use of ESH 2023 criteria for hypertension staging provides contemporary classification. Rigorous exclusion criteria minimised confounding from known inflammatory conditions, diabetes, and renal disease.

Several limitations warrant consideration such as : (1) Whether elevated CRP precedes, accompanies, or follows blood pressure elevation cannot be determined. (2) the study was conducted at a single tertiary care centre, which may limit generalisability. (3) despite exclusion criteria, significant differences in age, sex, and BMI between groups represent potential confounders. Cases were older, had more females, and higher BMI, all of which may independently influence CRP levels. While we did not perform adjusted analyses in the primary results, the magnitude of CRP differences (five-fold elevation) suggests that group differences are unlikely to be fully explained by these covariates. (4) the small number of

Grade 3 hypertensives (n=5) limits conclusions about this subgroup. (5) CRP was measured at a single time point, and intra-individual variability was not assessed.

Future Directions: Future research should address the temporal relationship between CRP and hypertension through prospective longitudinal studies. Investigation of whether CRP predicts disease progression, treatment response, or cardiovascular outcomes in newly detected hypertensives would have clinical relevance. Studies examining whether anti-inflammatory interventions affect both CRP and blood pressure in this population would help establish whether the relationship is causal. Larger, multicentre studies with adjustment for potential confounders would strengthen the evidence base.

CONCLUSION

In newly detected, treatment-naïve adults, CRP levels are markedly elevated in hypertensive individuals compared with normotensive controls and increase progressively with blood pressure level and hypertension grade. These findings support an inflammatory association with hypertension severity at the earliest clinical presentation. CRP, as a widely available inflammatory marker, may complement blood pressure assessment in characterising inflammatory burden in early hypertension. Longitudinal studies are needed to determine whether CRP predicts disease progression or cardiovascular outcomes and to establish the temporal and causal nature of this relationship.

REFERENCES

1. World Health Organization. Hypertension. Geneva: WHO; 2023.
2. NCD Risk Factor Collaboration. Worldwide trends in hypertension prevalence and progress in treatment and control from 1990 to 2019. Lancet. 2021;398(10304):957-980.
3. Gupta R, Xavier D. Hypertension: The most important non communicable disease risk factor in India. Indian Heart J. 2018;70(4):565-572.
4. Drummond GR, Vinh A, Guzik TJ, Sobey CG. Immune mechanisms of hypertension. Nat Rev Immunol. 2019;19(8):517-532.
5. McMaster WG, Kirabo A, Madhur MS, Harrison DG. Inflammation, immunity, and hypertensive end-organ damage. Circ Res. 2015;116(6):1022-1033.
6. Guzik TJ, Touyz RM. Oxidative stress, inflammation, and vascular aging in hypertension. Hypertension. 2017;70(2):212-221.
7. Dinh QN, Drummond GR, Sobey CG, Chrissobolis S. Roles of inflammation, oxidative stress, and vascular dysfunction in hypertension. Biomed Res Int. 2014;2014:406960.
8. Pepys MB, Hirschfield GM. C-reactive protein: a critical update. J Clin Invest. 2003;111(12):1805-1812.
9. Ridker PM. C-reactive protein and the prediction of cardiovascular events among those at intermediate risk: moving an inflammatory hypothesis toward consensus. J Am Coll Cardiol. 2007;49(21):2129-2138.
10. Gabay C, Kushner I. Acute-phase proteins and other systemic responses to inflammation. N Engl J Med. 1999;340(6):448-454.
11. Venugopal SK, Devaraj S, Yuhanna I, Shaul P, Jialal I. Demonstration that C-reactive protein decreases eNOS expression and bioactivity in human aortic endothelial cells. Circulation. 2002;106(12):1439-1441.
12. Verma S, Wang CH, Li SH, et al. A self-fulfilling prophecy: C-reactive protein attenuates nitric oxide production and inhibits angiogenesis. Circulation. 2002;106(8):913-919.
13. Sesso HD, Buring JE, Rifai N, Blake GJ, Gaziano JM, Ridker PM. C-reactive protein and the risk of developing hypertension. JAMA. 2003;290(22):2945-2951.
14. Ridker PM, Buring JE, Shih J, et al. Prospective study of C-reactive protein and the risk of future cardiovascular events among apparently healthy women. Circulation. 1998;98(8):731-733.
15. Bautista LE, Vera LM, Arenas IA, Gamarra G. Independent association between inflammatory markers (C-reactive protein, interleukin-6, and TNF-alpha) and essential hypertension. J Hum Hypertens. 2005;19(2):149-154.
16. Schillaci G, Pirro M. C-reactive protein in hypertension: clinical significance and predictive value. Nutr Metab Cardiovasc Dis. 2006;16(7):500-508.
17. Joshi P, Islam S, Pais P, et al. Risk factors for early myocardial infarction in South Asians compared with individuals in other countries. JAMA. 2007;297(3):286-294.
18. Mancia G, Kreutz R, Brunström M, et al. 2023 ESH Guidelines for the management of arterial hypertension. J Hypertens. 2023;41(12):1874-2071.
19. He J, Wang L, Zhang Y, et al. Association between serum C-reactive protein levels and hypertension in elderly individuals: a cross-sectional study. Aging Clin Exp Res. 2022;34(5):1123-1130.
20. Dzieża-Grudnik A, Czekaj A, Surdacki A, et al. Inflammatory cytokine profile in newly diagnosed hypertension. Blood Press. 2023;32(1):15-24.
21. Kansui Y, Matsumura K, Morinaga Y, et al. C-reactive protein and incident hypertension in a worksite population of Japanese men. J Clin Hypertens. 2019;21(4):497-503.
22. Zhang W, Chen X, Huang L, et al. Association between C-reactive protein, interleukin-6 and insulin resistance in hypertensive patients with cerebral infarction. J Int Med Res. 2019;47(7):3153-3161.
23. Shukla V, Kumar A, Singh R, et al. Neutrophil-to-lymphocyte ratio in newly diagnosed hypertensive patients and its correlation with hypertension staging. Indian J Med Res. 2025;161(2):142-149.

24. Pasceri V, Willerson JT, Yeh ET. Direct proinflammatory effect of C-reactive protein on human endothelial cells. Circulation. 2000;102(18):2165-2168.