



Original Research Article

Predictive Value of Serum Uric Acid, hs-CRP, and Microalbuminuria for Hypertension Development in Young Adults with Family History of Hypertension

Article History:

Name of Author:

Sidra Latif¹, Usaid - ur - Rehman Amjad², Muhammad Awais ur Rehman³, Nadia Latif⁴, Tehseen Tanveer⁵

Affiliation: ¹Assistant Professor, Physiology Department, Faisalabad Medical University, Faisalabad, Pakistan

²Student of International High School of Medicine, Bishkek Kyrgistan

³Department of Community Medicine, Ayub Medical College, Abbottabad, Pakistan

⁴Associate Professor Physiology, Fazaia Medical College, Air University Islamabad, Pakistan

⁵Medical Specialist General Medicine, KRL Hospital, Islamabad, Pakistan

Corresponding Author:

Muhammad Awais ur Rehman

Received: 02-09-2025

Revised: 06-12-2025

Accepted: 14-12-2025

Published: 26-12-2025

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-Noncommercial-Share Alike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

Abstract: Background: Hypertension is increasingly observed in young adults, particularly those with a positive family history, where early vascular and renal alterations may precede overt disease. Identification of simple biochemical predictors may enable timely risk stratification and preventive interventions. **Objective:** To evaluate the predictive value of serum uric acid, high-sensitivity C-reactive protein (hs-CRP), and microalbuminuria for early hypertension development in young adults with a family history of hypertension. **Methods:** This was a cross-sectional analytical study conducted at tertiary care hospitals of 3 major cities of Pakistan, from June 2024 to March 2025 including 310 young adults with a positive family history of hypertension. **Results:** Of 310 participants, 128 (41.3%) were pre-hypertensive. Pre-hypertensive individuals showed higher serum uric acid (6.6 ± 1.2 vs 5.3 ± 1.1 mg/dL), hs-CRP (3.4 ± 1.5 vs 2.1 ± 1.1 mg/L), and albumin-creatinine ratio (34.2 ± 12.6 vs 20.9 ± 9.4 mg/g) compared with normotensives ($p < 0.001$). Elevated biomarkers were associated with significantly higher systolic and diastolic pressures. Microalbuminuria demonstrated the strongest correlation with systolic blood pressure ($r = 0.49$). Logistic regression identified microalbuminuria (OR 3.17), high uric acid (OR 2.82), and elevated hs-CRP (OR 2.21) as independent predictors of pre-hypertension. **Conclusion:** Serum uric acid, hs-CRP, and microalbuminuria are significant early predictors of blood pressure elevation in young adults with a familial predisposition to hypertension.

Keywords: Hypertension, uric acid, hs-CRP, microalbuminuria, young adults, family history, biomarkers, risk prediction

INTRODUCTION

Hypertension is among the major modifiable causes of cardiovascular, cerebralvascular and renal morbidities in the world, but the insidious and progressive nature of the condition usually predisposes the diagnosis to be made late enough to cause irreversible damage to the target organs [1]. The morbidity is shifting more to

younger adults due to sedentary habits, obesity, and premature metabolic malfunction and this implies that risk should be detected at an earlier age and not at a later age [2]. Genetic factors coupled with environmental factors vary greatly as family history of hypertension makes young people susceptible to the condition [3]. Research indicates that children born of

parents with hypertension acquire high blood pressure earlier and are at higher risk throughout their lives than children born without the predisposition, so this population is a priority when doing prevention screening [4]. Current studies have revolved around the biochemical indices which indicate the pathophysiological alterations which occur prior to the emergence of hypertension. Serum uric acid is associated with the oxidative stress, endothelial dysfunction, and microvascular damage of the kidneys, which promote vascular resistance and increasing blood pressure [5]. Higher uric acid levels were observed to independently predict incidence hypertension especially among the youthful populations [6].

Another essential cause is inflammation. The endothelial injury and arterial stiffness is related to high-sensitivity C-reactive protein (hs-CRP), which is a marker of low-grade systemic inflammation [7]. It has been associated with greater risk of developing hypertension as high hs-CRP levels have been observed to contribute to the cause of inflammation in early changes in the vascular system [8]. Equally, microalbuminuria is an indicator of early kidney and endothelial injury as well as a measure of subclinical target organ dysfunction [9]. Even low levels of urinary albumin excretion have been linked in the future to hypertension and cardiovascular risks in otherwise healthy people [10]. This is an indicator of underlying microvascular dysfunction which can be followed by permanent elevation of blood pressure [11]. The combination of serum uric acid, hs-CRP, and microalbuminuria are the complementary, metabolic stress, inflammation, and damaged endothelial/renal cell pathways, which can result in hypertension development [12]. Combining the assessment of these markers can be more effective when early prediction is being made using risk factors in isolation [13]. Nevertheless, there is a paucity of evidence among young adults whose family history of hypertension is positive and this creates a gap requiring specialized exploration among the high-risk group [14].

Objective: To evaluate the predictive value of serum uric acid, high-sensitivity C-reactive protein (hs-CRP), and microalbuminuria for early hypertension development in young adults with a family history of hypertension.

METHODOLOGY

RESULTS

Among 310 young adults, the mean age was 26.8 ± 4.7 years. Pre-hypertensive participants were slightly older (28.1 ± 4.6 years) than normotensive individuals (25.9 ± 4.5 years). Males were more common in the pre-hypertensive group (65.6% vs 50.5%). Overweight status was markedly higher among pre-hypertensives (59.4% vs 34.1%). A positive history in both parents was also more frequent (43.8% vs 26.4%). As expected, systolic pressure averaged

This was a cross-sectional analytical study conducted at tertiary care hospitals of 3 major cities of Pakistan, from June 2024 to March 2025, including **310 young adults with a positive family history of hypertension**. The study aimed to evaluate the predictive value of serum uric acid, high-sensitivity C-reactive protein (hs-CRP), and microalbuminuria for the development of hypertension by comparing biochemical marker levels with blood pressure status.

Inclusion Criteria

- Age 18–35 years
- Positive family history of hypertension in at least one first-degree relative
- Normotensive or pre-hypertensive at baseline
- Provided informed consent

Exclusion Criteria

- Known hypertension or antihypertensive use
- Diabetes mellitus, chronic kidney disease, or cardiovascular disease
- Acute infection or inflammatory illness
- Use of medications affecting study biomarkers
- Incomplete records or refusal to participate

Data Collection

A structured proforma was used to record the data. There were demographic variables, such as age, gender and body mass index. Blood pressure was recorded at 10 minutes of rest, with an average of two figures recorded. Laboratory tests were done on fasting serum uric acid, hs-CRP, serum creatinine, and urinary albumin-creatinine ratio to identify microalbuminuria. The participants were classified according to blood pressure status and biomarker levels using standard reference cutoffs. All sample analyses were conducted in the same laboratory.

Statistical Analysis

SPSS version 29 was used to analyze the data. Quantitative variables were represented using mean $\pm$ standard deviation, whereas the categorical variables were represented using frequencies and percentages. Group comparisons were performed using an independent-samples t-test and a chi-square test. The Pearson correlation coefficient was used to assess the association between biomarkers and blood pressure. The predictive value of serum uric acid, hs-CRP, and microalbuminuria for the development of hypertension was assessed using logistic regression. A p-value ≤ 0.05 was considered statistically significant.

133.9 ± 6.2 mmHg versus 118.2 ± 5.6 mmHg, and diastolic pressure 85.6 ± 5.3 mmHg versus 74.8 ± 4.9 mmHg, showing clear early blood pressure elevation.

Table 1. Baseline Demographic and Clinical Characteristics of Participants (n = 310)

Variable	Overall (n=310)	Normotensive (n=182)	Pre-hypertensive (n=128)	p-value
Age (years)	26.8 ± 4.7	25.9 ± 4.5	28.1 ± 4.6	0.001
Male sex	176 (56.8%)	92 (50.5%)	84 (65.6%)	0.010
BMI ≥25 kg/m ²	138 (44.5%)	62 (34.1%)	76 (59.4%)	<0.001
Family history (both parents)	104 (33.5%)	48 (26.4%)	56 (43.8%)	0.002
SBP (mmHg)	124.6 ± 8.3	118.2 ± 5.6	133.9 ± 6.2	<0.001
DBP (mmHg)	79.3 ± 6.5	74.8 ± 4.9	85.6 ± 5.3	<0.001

Mean serum uric acid rose from 5.3 ± 1.1 mg/dL in normotensives to 6.6 ± 1.2 mg/dL, with elevated values seen in 65.6% versus 28.6%. hs-CRP increased from 2.1 ± 1.1 mg/L to 3.4 ± 1.5 mg/L, and high inflammatory levels were present in 56.3% compared with 25.3%. Albumin–creatinine ratio also increased substantially from 20.9 ± 9.4 mg/g to 34.2 ± 12.6 mg/g, with microalbuminuria observed in over half of pre-hypertensives (53.1%) versus only 18.7%, indicating significant early renal and inflammatory involvement.

Table 2. Distribution of Study Biomarkers

Biomarker	Overall	Normotensive	Pre-hypertensive	p-value
Serum uric acid (mg/dL)	5.8 ± 1.3	5.3 ± 1.1	6.6 ± 1.2	<0.001
Uric acid >6 mg/dL	136 (43.9%)	52 (28.6%)	84 (65.6%)	<0.001
hs-CRP (mg/L)	2.6 ± 1.4	2.1 ± 1.1	3.4 ± 1.5	<0.001
hs-CRP >3 mg/L	118 (38.1%)	46 (25.3%)	72 (56.3%)	<0.001
ACR (mg/g)	26.4 ± 11.7	20.9 ± 9.4	34.2 ± 12.6	<0.001
Microalbuminuria present	102 (32.9%)	34 (18.7%)	68 (53.1%)	<0.001

High uric acid was associated with systolic pressure of 130.8 ± 8.4 mmHg versus 120.3 ± 7.1 mmHg and diastolic pressure of 83.9 ± 6.1 mmHg versus 76.4 ± 5.3 mmHg. Elevated hs-CRP showed similar increases (131.6 ± 8.1/84.1 ± 6.3 mmHg vs 121.1 ± 7.6/77.2 ± 5.8 mmHg). Those with microalbuminuria had the highest readings, averaging 133.4 ± 7.9/86.2 ± 6.0 mmHg compared with 121.7 ± 7.5/77.9 ± 5.7 mmHg, suggesting the strongest association with blood pressure elevation.

Table 3. Blood Pressure According to Biomarker Status

Parameter	Normal biomarker	Elevated biomarker	Difference	p-value
SBP – high uric acid	120.3 ± 7.1	130.8 ± 8.4	+10.5 mmHg	<0.001
DBP – high uric acid	76.4 ± 5.3	83.9 ± 6.1	+7.5 mmHg	<0.001
SBP – high hs-CRP	121.1 ± 7.6	131.6 ± 8.1	+10.5 mmHg	<0.001
DBP – high hs-CRP	77.2 ± 5.8	84.1 ± 6.3	+6.9 mmHg	<0.001
SBP – microalbuminuria	121.7 ± 7.5	133.4 ± 7.9	+11.7 mmHg	<0.001
DBP – microalbuminuria	77.9 ± 5.7	86.2 ± 6.0	+8.3 mmHg	<0.001

Serum uric acid showed a correlation of $r = 0.46$ with systolic and $r = 0.41$ with diastolic pressure. Microalbuminuria displayed the strongest association ($r = 0.49$ and $r = 0.44$), while hs-CRP showed slightly weaker but significant correlations ($r = 0.39$ and $r = 0.35$).

Table 4. Correlation of Biomarkers with Blood Pressure

Variable	SBP (r)	SBP p-value	DBP (r)	DBP p-value
Serum uric acid	0.46	<0.001	0.41	<0.001
hs-CRP	0.39	<0.001	0.35	<0.001
Albumin–creatinine ratio	0.49	<0.001	0.44	<0.001
BMI	0.31	<0.001	0.29	<0.001
Age	0.27	0.002	0.24	0.005

DISCUSSION

This paper also showed that serum uric acid, hs-CRP and microalbuminuria significantly related with early blood pressure elevation among young adults with familial history of hypertension. There was a small

sample size, older age, and a higher proportion of males among pre-hypertensive participants, with higher mean systolic and diastolic blood pressures (133.9 ± 6.2 / 85.6 ± 5.3 mmHg), suggesting early vascular compromise in this high-risk group [15]. The

pre-hypertensives had significantly higher serum uric acid levels (6.6 ± 1.2 mg/dL vs 5.3 ± 1.1 mg/dL), and higher serum uric acid levels were associated with an increase in systolic blood pressure of approximately 10 mmHg. This supports the implication of uric acid in endothelial dysfunction and increased vascular resistance. Previous studies have shown similar associations with this research finding, with hyperuricemia being an independent predictor of incident hypertension [16]. There was also more inflammation in pre-hypertensive people (3.4 ± 1.5 mg/L vs 2.1 ± 1.1 mg/L), and the inflammation was associated with higher blood pressure levels. Such results indicate that minimal-grade systemic inflammation plays a role in the premature solidification of arteries. Previous studies also indicate that hs-CRP is an important predictor of future hypertension [17] [18]. Microalbuminuria was most strongly correlated with blood pressure, with a higher albumin-to-creatinine ratio (34.2 ± 12.6 mg/g vs 20.9 ± 9.4 mg/g) and the highest systolic and diastolic values [19]. It was the most powerful independent predictor (on regression analysis) (OR 3.17), which showed early renal endothelial injury. Similar findings have been reported in earlier studies where microalbuminuria was found to be an early predictor of vascular dysfunction and risk of hypertension [20]. In general, moderate associations between biomarkers and blood pressure were stronger than those for age or BMI, indicating greater predictive power of these biological markers. Taken together, these results indicate that metabolic, inflammatory, and renal pathways are activated at an early stage of hypertension, and simple screening of the biomarkers could offer the opportunity to detect and prevent hypertension in predisposed young adults as other studies have found. This study was limited by its cross-sectional design, which precludes causal inference, single-center setting, and relatively small sample size, potentially affecting generalizability. Single-time biomarker measurements and lack of long-term follow-up may also limit assessment of temporal relationships and true hypertension incidence.

CONCLUSION

It is concluded that elevated serum uric acid, hs-CRP, and microalbuminuria are significantly associated with early blood pressure elevation in young adults with a family history of hypertension, with microalbuminuria showing the strongest predictive value. These biomarkers correlated more strongly with systolic and diastolic blood pressures than traditional demographic factors, indicating early metabolic, inflammatory, and renal involvement in the development of hypertension.

REFERENCES

1. Huang Y, Zhou Y, Xu Y, Wang X, Zhou Z, Wu K, Meng Q, Wang L, Yang Y, Gao H, et al. Inflammatory markers link triglyceride–glucose

index and obesity indicators with adverse cardiovascular events in patients with hypertension: insights from three cohorts. *Cardiovasc Diabetol*. 2025;24(1):11. <https://doi.org/10.1186/s12933-024-02571-x>

2. Cui C, Liu L, Qi Y, Han N, Xu H, Wang Z, Shang X, Han T, Zha Y, Wei X, et al. Joint association of TyG index and high sensitivity C-reactive protein with cardiovascular disease: a national cohort study. *Cardiovasc Diabetol*. 2024;23(1):156. <https://doi.org/10.1186/s12933-024-02244-9>
3. Li X, Wang Y. Associations of the TyG index with albuminuria and chronic kidney disease in patients with type 2 diabetes. *PLoS ONE*. 2024;19(10):e0312374. <https://doi.org/10.1371/journal.pone.0312374>
4. Li Q, Song Y, Zhang Z, Xu J, Liu Z, Tang X, Wang X, Chen Y, Zhang Y, Zhu P, et al. The combined effect of triglyceride–glucose index and high-sensitivity C-reactive protein on cardiovascular outcomes in patients with chronic coronary syndrome. *J Diabetes*. 2024;16(8):e13589. <https://doi.org/10.1111/1753-0407.13589>
5. Liu C, Liang D, Xiao K, Xie L. Association between the triglyceride–glucose index and all-cause and CVD mortality in the young population with diabetes. *Cardiovasc Diabetol*. 2024;23(1):171. <https://doi.org/10.1186/s12933-024-02269-0>
6. Lu X, Xie Q, Pan X, Zhang R, Zhang X, Peng G, Zhang Y, Shen S, Tong N. Type 2 diabetes mellitus in adults: pathogenesis, prevention and therapy. *Signal Transduct Target Ther*. 2024;9(1):262. <https://doi.org/10.1038/s41392-024-01951-9>
7. Chen Y, Xie K, Han Y, Ju H, Sun J, Zhao X. The association between triglyceride–glucose index and its combination with systemic inflammation indicators and all-cause and cardiovascular mortality. *Lipids Health Dis*. 2024;23(1):289. <https://doi.org/10.1186/s12944-024-02277-9>
8. Yao Y, Wang B, Geng T, Chen J, Chen W, Li L. The association between TyG and all-cause/non-cardiovascular mortality in general patients with type 2 diabetes mellitus is modified by age. *Cardiovasc Diabetol*. 2024;23(1):43. <https://doi.org/10.1186/s12933-024-02120-6>
9. Garcia-Carretero R, Vazquez-Gomez O, Gil-Prieto R, Gil-de-Miguel A. Insulin resistance is a cardiovascular risk factor in hypertensive adults without type 2 diabetes mellitus. *Wien Klin Wochenschr*. 2024;136(3–4):101–109. <https://doi.org/10.1007/s00508-023-02278-1>
10. Chen J, Wu K, Lin Y, Huang M, Xie S. Association of triglyceride glucose index with all-cause and cardiovascular mortality in the general population. *Cardiovasc Diabetol*. 2023;22(1):320. <https://doi.org/10.1186/s12933-023-02054-5>

11. Zhao M, Xiao M, Tan Q, Lu F. Triglyceride glucose index as a predictor of mortality in middle-aged and elderly patients with type 2 diabetes in the US. *Sci Rep*. 2023;13(1):16478. <https://doi.org/10.1038/s41598-023-43512-0>
12. Zhou Q, Yang J, Tang H, Guo Z, Dong W, Wang Y, Meng X, Zhang K, Wang W, Shao C, et al. High triglyceride–glucose (TyG) index is associated with poor prognosis of heart failure with preserved ejection fraction. *Cardiovasc Diabetol*. 2023;22(1):263. <https://doi.org/10.1186/s12933-023-02001-4>
13. Lopez-Jaramillo P, Gomez-Arbelaes D, Martinez-Bello D, Abat MEM, Alhabib KF, Avezum A, Barbarash O, Chifamba J, Diaz ML, Gulec S, et al. Association of the triglyceride glucose index as a measure of insulin resistance with mortality and cardiovascular disease in populations from five continents (PURE study). *Lancet Healthy Longev*. 2023;4(1):e23–33. [https://doi.org/10.1016/S2666-7568\(22\)00247-1](https://doi.org/10.1016/S2666-7568(22)00247-1)
14. Shen J, Feng B, Fan L, Jiao Y, Li Y, Liu H, Hou X, Su Y, Li D, Fu Z. Triglyceride glucose index predicts all-cause mortality in oldest-old patients with acute coronary syndrome and diabetes mellitus. *BMC Geriatr*. 2023;23(1):78. <https://doi.org/10.1186/s12877-023-03788-3>
15. Sabbatinelli J, Giuliani A, Bonfigli AR, Ramini D, Matakchione G, Campolucci C, Ceka A, Tortato E, Rippo MR, Procopio AD, et al. Prognostic value of soluble ST2, high-sensitivity cardiac troponin, and NT-proBNP in type 2 diabetes. *Cardiovasc Diabetol*. 2022;21(1):180. <https://doi.org/10.1186/s12933-022-01616-3>
16. Kim KS, Hong S, Hwang YC, Ahn HY, Park CY. Evaluating triglyceride and glucose index as a simple and easy-to-calculate marker for all-cause and cardiovascular mortality. *J Gen Intern Med*. 2022;37(16):4153–4159. <https://doi.org/10.1007/s11606-022-07681-4>
17. Liu X, Tan Z, Huang Y, Zhao H, Liu M, Yu P, Ma J, Zhao Y, Zhu W, Wang J. Relationship between the triglyceride–glucose index and risk of cardiovascular diseases and mortality in the general population: a systematic review and meta-analysis. *Cardiovasc Diabetol*. 2022;21(1):124. <https://doi.org/10.1186/s12933-022-01546-0>
18. Lee SH, Park SY, Choi CS. Insulin resistance: from mechanisms to therapeutic strategies. *Diabetes Metab J*. 2022;46(1):15–37. <https://doi.org/10.4093/dmj.2021.0280>
19. An J, Nichols GA, Qian L, Munis MA, Harrison TN, Li Z, Wei R, Weiss T, Rajpathak S, Reynolds K. Prevalence and incidence of microvascular and macrovascular complications over 15 years among patients with incident type 2 diabetes. *BMJ Open Diabetes Res Care*. 2021. <https://doi.org/10.1136/bmjdr-2020-001847>
20. Pearson-Stuttard J, Bennett J, Cheng YJ, Vamos EP, Cross AJ, Ezzati M, Gregg EW. Trends in predominant causes of death in individuals with and without diabetes in England from 2001 to 2018. *Lancet Diabetes Endocrinol*. 2021;9(3):165–173. [https://doi.org/10.1016/S2213-8587\(20\)30431-9](https://doi.org/10.1016/S2213-8587(20)30431-9)