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ORIGINAL ARTICLE

Predictive value of admission biomarkers for mortality and rehospitalization in hypertensive crisis

Emir Bećirović[1], Minela Bećirović[1], Amir Bećirović[1], Amina Džidić Krivić[2], Armin Šljivo[3], Kenana Ljuca[4], Lemana Buljubašić[5], Nadina Ljuca[5], Admir Abdić[6], Emir Begagić[7]

[1]Internal Medicine Clinic, University Clinical Centre Tuzla, Tuzla, [2]Department of Neurology, Cantonal Hospital Zenica, Zenica, [3]Department of Cardiology, University Clinical Centre Sarajevo, Sarajevo; Bosnia and Herzegovina, [4]Department of Gynaecology and Obstetrics, University Clinical Centre Ljubljana, Ljubljana, Slovenia; [5]School of Medicine, University of Tuzla, Tuzla, [6]Department of Surgery, Cantonal Hospital Bihać, Bihać, [7]Department of Neurosurgery, Cantonal Hospital Zenica; Bosnia and Herzegovina

ABSTRACT

Aim To identify predictors of all-cause mortality and 6-month rehospitalisation in patients with hypertensive crisis, focusing on inflammatory indices, metabolic markers measured at admission, and antihypertensive treatment profiles.

Methods This prospective observational study included 210 adult patients with hypertensive crisis. Demographic, clinical, and therapeutic data were collected, including data on comorbidities, antihypertensive drug use, and treatment adherence. Laboratory parameters obtained at admission included neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), pan-immune-inflammation value (PIV), homocysteine, and uric acid. Patients were followed for 12 months. Multivariate logistic regression and receiver operating characteristic (ROC) curve analyses were conducted to identify independent predictors.

Results Mortality occurred in 10.9% of patients, and 27.1% were rehospitalised within 6 months. Deceased patients exhibited significantly higher levels of PLR (p=0.0329), SII (p=0.0355), homocysteine (p=0.0488), and uric acid (p=0.021). In multivariate analysis, homocysteine (OR=3.55; p<0.001), uric acid (OR=1.03; p=0.007), PLR (OR=1.04; p=0.047), and SII (OR=1.01; p=0.030) remained independently associated with mortality. Chronic kidney disease (OR=2.15, p=0.012) and poor treatment adherence (OR=1.92; p=0.017) were also significant predictors. ROC analysis demonstrated moderate discriminative power, with AUC values of 0.68 for PLR, 0.66 for SII, 0.65 for homocysteine, and 0.63 for uric acid.

Conclusion Elevated inflammatory indices and metabolic markers, particularly homocysteine and uric acid, were independently associated with increased mortality risk. Additionally, chronic kidney disease and suboptimal adherence to antihypertensive therapy significantly contributed to adverse outcomes. These findings underscore the importance of comprehensive risk assessment and personalised management in this high-risk population.

Keywords: homocysteine, hypertensive crisis, inflammation, medication adherence, prognosis, uric acid

INTRODUCTION

Hypertensive crisis represents a severe elevation in blood pressure requiring urgent clinical attention (1). It is broadly categorized into two distinct entities: hypertensive urgency, characterized by the absence of acute target organ damage, and hypertensive emergency, marked by evidence of organ injury such as myocardial infarction, stroke, acute pulmonary oedema, or acute kidney injury (AKI) (2). Although less frequent than chronic hypertension, hypertensive crisis carries a disproportionately high risk of morbidity and mortality (3). It is estimated that up to 1–2% of individuals with hypertension will experience a hypertensive crisis during their lifetime, most often in the context of uncontrolled or untreated disease (4).

Recently, increasing attention has been directed towards identifying prognostic factors associated with short- and long-term outcomes in patients presenting with hypertensive crisis (5). Traditional risk markers, such as advanced age, renal impairment, and established cardiovascular comorbidities, have been extensively studied (6). However, these alone often fail to capture the full spectrum of systemic pathophysiological burden. Emerging evidence suggests that inflammatory and metabolic markers, including neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), pan-immune-inflammation value (PIV), homocysteine, and uric acid, may reflect ongoing vascular stress and en-

***Corresponding author:** Emir Bećirović
Internal Medicine Clinic, Intensive Care Unit, University Clinical Centre Tuzla
Prof. dr. Ibre Pašića bb, 75000 Tuzla, Bosnia and Herzegovina
Phone: +387 35 303 304;
E-mail: becirovic.emir@live.com
ORCID ID https://orcid.org/0000-0002-4134-987X

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dothelial dysfunction. They may provide additional prognostic insight beyond conventional parameters (7,8). These markers are particularly attractive in clinical practice because they are widely available, inexpensive, and can be calculated from routine laboratory tests, making them suitable for use in both tertiary and resource-limited healthcare settings (9).

Moreover, the influence of antihypertensive treatment patterns and patient adherence remains underexplored in this clinical setting (10). Complex medication regimens and suboptimal compliance may contribute not only to the onset of a crisis but also to poorer outcomes during follow-up. Early identification of high-risk individuals may inform the development of targeted follow-up strategies and customised therapeutic approaches (11).

While prior studies have frequently examined individual biomarkers or organ-specific endpoints, few have assessed the combined predictive value of biochemical, inflammatory, and treatment-related factors (12). Furthermore, regional data from Southeast Europe, including Bosnia and Herzegovina, remain limited.

Although guidelines emphasise blood pressure control, there remains limited consensus regarding which clinical or laboratory parameters should be prioritised for early risk stratification in hypertensive crisis (13). Integrating inflammatory, metabolic, and therapeutic dimensions into a unified prognostic framework may improve clinical decision-making and patient outcomes (14). By identifying simple, reproducible predictors of mortality and rehospitalisation, clinicians may be better equipped to implement early interventions and optimise follow-up care (15).

This study aimed to evaluate multimodal predictors of adverse outcomes in patients with hypertensive crisis, with a particular focus on inflammatory indices, homocysteine and uric acid levels, comorbid conditions such as chronic kidney disease, and patterns of antihypertensive treatment. The study further sought to examine the relationship between these parameters and the risk of all-cause mortality and six-month rehospitalisation. We hypothesised that inflammatory and metabolic markers measured at admission, together with chronic kidney disease and poor antihypertensive adherence, independently predict mortality and rehospitalisation in patients with hypertensive crisis.

SUBJECTS AND METHODS

Patients and study design

This prospective observational cohort study was conducted at the Clinic for Internal Medicine, University Clinical Centre Tuzla, over a period of six months in 2024. The study included 210 adult patients admitted with a confirmed diagnosis of hypertensive crisis, further classified as either hypertensive urgency or hypertensive emergency. Patients with hypertensive urgency and hypertensive emergency were analysed separately in all outcome analyses.

Inclusion criteria were age 18 years or older, systolic blood pressure of 180 mmHg or higher, and diastolic blood pressure of 110 mmHg or higher on admission, and clinical features consistent with hypertensive crisis, following the current European Society of Cardiology (ESC) guidelines (16). Patients were excluded if they had secondary hypertension of endocrine origin, pregnancy-related hypertension, active malignancy, or incomplete laboratory data. All patients, including those who experienced in-hospital mortality within the hypertensive urgency group, were included in the final analysis.

Upon admission, data were collected on demographics, comorbidities (including diabetes mellitus and chronic kidney disease), smoking status, family history of cardiovascular disease, current antihypertensive medications, and adherence to prescribed therapy. Antihypertensive regimens were reviewed, and any changes made during hospitalisation were documented. All patients underwent laboratory testing, which included complete blood count and hemogram-derived indices such as NLR, PLR, SII, and PIV. In addition, metabolic and biochemical markers were analysed, including serum homocysteine, uric acid, cystatin C, electrolytes, and renal function parameters.

Patients were prospectively followed for 6 months. The primary outcomes were all-cause mortality and rehospitalisation due to cardiovascular or hypertension-related events. In-hospital complications were also recorded, including acute pulmonary oedema, non-ST elevation myocardial infarction (NSTEMI), ischaemic stroke, haemorrhagic stroke, and AKI.

All patients provided written informed consent prior to inclusion.

The study protocol was approved by the Ethics Committee of the University Clinical Centre Tuzla and was conducted following the ethical standards set forth in the Declaration of Helsinki.

Methods

Demographic, clinical, and therapeutic data were collected for all included patients using a structured evaluation protocol upon hospital admission. This encompassed a detailed medical history, physical examination, review of prior diagnoses, and confirmation of antihypertensive medication use through patient interviews and prescription records. Comorbidities such as diabetes mellitus (DM type 2) and chronic kidney disease (CKD) were identified based on documented medical history and supporting laboratory findings. Tobacco use and family history of cardiovascular disease were also recorded. Blood pressure was measured using a calibrated automated sphygmomanometer, with values confirmed by repeated measurements. Therapeutic adherence was assessed using a standardised questionnaire administered to attending physicians, evaluating both the regularity and consistency of antihypertensive drug use. The assessment was based on patient self-report and prescription verification over the preceding 30 days. Poor adherence was defined as missing ≥20% of prescribed doses or complete therapy interruption prior to admission. Changes to therapy during hospitalisation were documented. Patients were stratified according to the number and type of antihypertensive agents used on admission, including angiotensin-converting enzyme inhibitors, angiotensin receptor blockers (ARBs), beta-blockers (BBs), calcium channel blockers (CCBs), and diuretics.

Venous blood samples were collected within 24 hours of admission and analysed in the hospital's central laboratory. A complete blood count (CBC) was performed, and haematological indices including NLR, PLR, SII, and PIV were calculated. Serum biochemical parameters were measured, including homocysteine, uric acid, electrolytes, liver transaminases, cystatin C, albumin, and creatinine, as part of the comprehensive renal panel. Inflammatory markers such as C-reactive protein (CRP) and ferritin were also assessed. Haematological analyses were conducted using the Sysmex XN-1000 automated haematology analyser (Sysmex Corporation, Japan), with samples collected in K2EDTA tubes. Patients were monitored

throughout hospitalisation for the development of complications, including acute pulmonary oedema, NSTEMI, ischaemic stroke, haemorrhagic stroke, and AKI. These were confirmed through clinical examination, electrocardiography, chest radiography, echocardiography, brain imaging, and laboratory tests, as appropriate.

Following discharge, patients were prospectively followed for six months. Follow-up data were obtained via outpatient clinic visits, structured telephone interviews, and a review of hospital records to document all-cause mortality and cardiovascular-related rehospitalizations. The final follow-up assessment was completed at the end of the six-month period.

Statistical analysis

Categorical variables were presented as absolute frequencies (N) and percentages (%), while continuous variables were expressed as means with standard deviations (SD) or as medians with interquartile ranges (IQR), depending on the distribution. Data normality was assessed using the Kolmogorov–Smirnov test. Group comparisons were performed using Pearson's χ^2 test for categorical variables and either the Student's t-test or the Mann–Whitney U test for continuous variables, as appropriate. Variables with $p<0.10$ in the univariate analysis, along with clinically relevant parameters, were included in a multivariate logistic regression model to identify independent predictors of all-cause mortality and 6-month rehospitalisation. Adjusted odds ratios (ORs) with corresponding 95% confidence intervals (CI) were reported. Model fit was assessed using the Wald χ^2 statistic. The diagnostic performance of selected laboratory markers, including PLR, SII, homocysteine, and uric acid, was evaluated through receiver operating characteristic (ROC) curve analysis. Predictive accuracy was quantified as the area under the receiver operating characteristic (ROC) curve (AUC) for each biomarker. Optimal cut-off values were determined using the Youden index, and corresponding sensitivity and specificity were reported. To account for the potential confounding effect of chronic kidney disease, additional stratified ROC analyses were performed according to the CKD status. A two-tailed $p<0.05$ was considered statistically significant.

RESULTS

This prospective study included 210 patients diagnosed with hypertensive crisis, of whom 52.9% were male. The mean age was 65.7 years, and the average body mass index (BMI) was 28.1 kg/m². Hypertensive emergency was observed in 50.5% of patients, while 49.5% presented with hypertensive urgency. Among comorbidities, DM type 2 was presented in 30.0% of patients, CKD in 25.7%, and 44.8% had a positive family history of cardiovascular disease. Tobacco use was reported in 32.4% of cases. During the 6-month follow-up, 57 patients (27.1%) were rehospitalised, and 23 (10.9%) died.

When outcomes were stratified according to hypertensive crisis type, mortality and rehospitalisation occurred more frequently in patients with hypertensive emergency compared to those with hypertensive urgency. Mortality occurred in 20 (18.9%) patients with hypertensive emergency and three (2.9%) patients with hypertensive urgency ($p<0.001$). Rehospitalisation was observed in 38 (35.8%) patients with hypertensive emergency and 19 (18.3%) with hypertensive urgency ($p=0.006$) (Table 1)

Initial blood pressure values were 198±18 mmHg systolic and 110±10 mmHg diastolic. The most frequently used antihypertensive drug classes at admission were BBs (59.5%), ACE inhibitors (54.8%), diuretics (49.5%), and CCBs (45.2%). ARBs were used in 30.5% of patients. A total of 73.3% were receiving at least two antihypertensive agents on admission. Good therapeutic adherence was documented in 70.0% of cases, and therapy modifications were made during hospitalization in 35.0% of cases.

Table 1. Mortality and rehospitalisation according to hypertensive crisis type

Outcome	No (%) of patients		p
	Hypertensive emergency (N=106)	**Hypertensive urgency** (N=104)	
Mortality	20 (18.9)	3 (2.9)	<0.001
Rehospitalisation	38 (35.8)	19 (18.3)	0.006

Among laboratory parameters, deceased patients had significantly higher values of PLR (120±45 vs. 160±50; p=0.0329), SII (480±130 vs. 610±140; p=0.0355), homocysteine (13.5±4.0 vs. 19.2±5.1 μmol/L; p=0.0488), and uric acid (350±70 vs. 410±85 μmol/L; p=0.021). Although NLR and PIV were also elevated in deceased patients, these differences did not reach statistical significance. Deceased patients were more likely to have CKD (41.3% vs. 25.7%; p=0.027) and lower adherence to therapy (60.9% vs. 74.6%; p=0.022). Parameters such as RDW, MPV, and antihypertensive drug class usage were not significantly associated with mortality. However, patients treated with a combination of ACE inhibitors or ARBs and CCBs showed numerically lower mortality (26.1% vs. 36.1%), although the difference was not statistically significant (p=0.076) (Table 2).

ROC curve analysis demonstrated moderate discriminatory capacity for PLR, SII, homocysteine, and uric acid in predicting mortality, with AUC values of 0.68, 0.66, 0.65, and 0.63, respectively (Figure 1). When ROC analyses were additional-

Table 2. Clinical, laboratory and therapeutic characteristics by mortality status

Variable	Survived	Deceased	p
	Mean±SD		
PLR	120±45	160±50	0.0329
SII	480±130	610±140	0.0355
Homocysteine (μmol/L)	13.5±4.0	19.2±5.1	0.0488
Uric Acid (μmol/L)	350±70	410±85	0.021
NLR	2.4±0.9	3.1±1.2	0.076
PIV	400±100	460±120	0.11
RDW (%)	13.4±0.9	13.6±1.0	0.33
MPV (fL)	10.1±0.6	10.2±0.7	0.48
	No (%) of patients		
CKD	25.7	41.3	0.027
Therapy adherence	74.6	60.9	0.022
Beta-blocker use	58.2	60.9	0.36
CCB use	47.2	39.1	0.31
ACEI/ARBs + CCBs combo	36.1	26.1	0.076

PLR, platelet-to-lymphocyte ratio; SII, systemic immune-inflammation index; NLR, neutrophil-to-lymphocyte ratio; PIV, pan-immune-inflammation value; RDW, red cell distribution width; MPV, mean platelet volume; CKD, chronic kidney disease; CCB, calcium channel blockers; ACEI/ARBs + CCBs combo, combined use of ACE inhibitors or angiotensin receptor blockers with calcium channel blockers

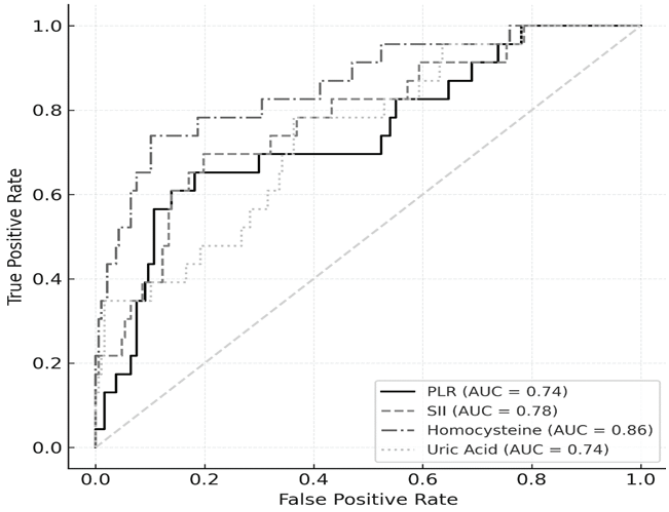

Figure 1. Receiver operating characteristic (ROC) curve analysis demonstrated modest but relevant predictive power of selected biomarkers for all-cause mortality. Area under the curve (AUC) values were: PLR=0.68, SII=0.66, Homocysteine=0.65, and uric acid=0.63. These findings support the prognostic utility of these parameters, especially when interpreted in the context of comorbidities and therapy

ly stratified by chronic kidney disease status, all biomarkers retained comparable discriminatory performance, indicating preserved prognostic value independent of renal impairment.

Regarding rehospitalisation, multivariate logistic regression analysis identified chronic kidney disease and poor therapeutic adherence as independent predictors of six-month readmission. Patients with CKD had a twofold increased risk of rehospitalisation (OR=2.01, 95% CI: 1.11–3.64; p=0.015), while poor adherence was associated with an 84% higher risk (OR=1.84, 95% CI: 1.03–3.27; p=0.034). No individual antihypertensive drug class was independently associated with rehospitalisation, although patients treated with a combination of ACE inhibitors and calcium channel blockers showed a non-significant trend toward reduced risk (OR=0.66; p=0.13) (Table 3).

Table 3. Multivariate predictors of rehospitalisation at 6 months

Variable	OR (95% CI)	p
CKD	2.01 (1.11–3.64)	0.015
Poor therapy adherence	1.84 (1.03–3.27)	0.034
ARBs at baseline	0.78 (0.42–1.47)	0.32
Diuretic use	1.22 (0.69–2.14)	0.41
ACEI + CCBs combination	0.66 (0.36–1.21)	0.13

CKD, chronic kidney disease; ARBs at baseline, use of angiotensin receptor blockers at the time of hospital admission; Diuretic use, administration of diuretic medications at baseline; ACEI+CCB combination, combined use of angiotensin-converting enzyme inhibitors and calcium channel blockers.

In a multivariate logistic regression model adjusted for age, homocysteine emerged as the strongest independent predictor of all-cause mortality (OR=3.55, 95% CI: 1.84–6.85; p<0.001), followed by uric acid (OR=1.03, 95% CI: 1.01–1.06; p=0.007), PLR (OR=1.04, 95% CI: 1.00–1.08; p=0.047), and SII (OR=1.01, 95% CI: 1.00–1.02; p=0.030). Chronic kidney disease (OR=2.15, 95% CI: 1.19–3.87; p=0.012) and poor therapeutic adherence (OR=1.92, 95% CI: 1.10–3.36; p=0.017) also remained independently associated with mortality (Table 4).

Among in-hospital complications, acute pulmonary oedema during the index hospitalisation was significantly associated with increased mortality (OR=1.89, 95% CI: 1.02–3.52; p=0.041). NSTEMI showed a non-significant trend toward higher mortality risk (OR=1.77; p=0.081) (Table 4).

Table 4. Multivariate predictors of all-cause mortality and in-hospital complications

Variable	OR (95% CI)	p
Homocysteine	3.55 (1.84–6.85)	<0.001
Uric Acid	1.03 (1.01–1.06)	0.007
PLR	1.04 (1.00–1.08)	0.047
SII	1.01 (1.00–1.02)	0.030
CKD	2.15 (1.19–3.87)	0.012
Poor therapy adherence	1.92 (1.10–3.36)	0.017
Complication: acute pulmonary oedema	1.89 (1.02–3.52)	0.041
Complication: NSTEMI	1.77 (0.91–3.42)	0.081

PLR, platelet-to-lymphocyte ratio; SII, systemic immune-inflammation index; CKD, chronic kidney disease; NSTEMI, non–ST elevation myocardial infarction during index hospitalization

In-hospital complications included acute pulmonary oedema (14.8%), NSTEMI (13.3%), ischaemic stroke (7.6%), haemorrhagic stroke (3.8%), and acute kidney injury (10.0%). The most frequent immediate causes of death were acute pulmonary oedema with respiratory failure, acute coronary syndromes, and severe ischaemic or haemorrhagic stroke. At the same time, a smaller proportion of patients died due to progressive multiorgan failure during hospitalization.

DISCUSSION

The results of this prospective cohort study confirm that inflammatory indices and metabolic markers, particularly PLR, SII, homocysteine, and uric acid, are independently associated with all-cause mortality in patients presenting with hypertensive crisis. Additionally, CKD and poor therapeutic adherence were significant predictors of both mortality and six-month rehospitalisation (17). These findings indicate that adverse outcomes in hypertensive crisis are influenced not only by the severity of blood pressure elevation but also by systemic inflammation, metabolic abnormalities, renal impairment, and long-term management (18). When outcomes were further stratified by hypertensive crisis type, patients with hypertensive emergency exhibited significantly higher mortality and rehospitalisation rates than those with hypertensive urgency. This finding confirms a more malignant clinical course of hypertensive emergency, and supports the need for more intensive in-hospital monitoring and structured post-discharge follow-up in this patient group.

Our results are consistent with other studies, which demonstrated that elevated SII and PLR were independent predictors of in-hospital mortality among patients with hypertensive emergencies (19). A large NHANES-based study also confirmed that high SII levels were associated with increased risk of stroke, regardless of traditional risk factors (20). These findings support our observation that platelet-leukocyte interactions and systemic inflammatory activity are essential contributors to vascular instability in acute hypertension (20).

Although NLR and PIV values were elevated among deceased

patients, these differences did not reach statistical significance. Similar findings were reported in cross-sectional studies involving individuals with elevated blood pressure, in which temporal fluctuations in inflammatory markers, such as NLR, were shown to reduce their reliability as stable prognostic indicators in acute clinical settings (21).

Homocysteine emerged as the strongest independent predictor of mortality in our cohort, with an odds ratio of 3.55. A recent cohort study using data from the NHANES database also identified elevated homocysteine as a significant predictor of cardiovascular mortality, reinforcing its relevance as a high-risk biomarker in hypertensive populations (22). Elevated uric acid levels also remained significantly associated with adverse outcomes. This observation agrees with results from recent meta-analyses that identified hyperuricemia as an independent risk factor for cardiovascular mortality among patients with hypertension (23).

CKD was another consistent predictor of mortality and rehospitalisation. These findings are supported by recent NHANES data showing that renal dysfunction significantly worsens prognosis in hypertensive populations (20). They highlight the importance of including renal function in initial patient assessments and follow-up planning (24). Importantly, additional stratified ROC analyses demonstrated that the prognostic performance of inflammatory and metabolic biomarkers remained preserved regardless of CKD status, indicating that their discriminatory value is not driven solely by renal impairment.

Poor adherence to antihypertensive therapy also showed a clear association with adverse outcomes. This observation is supported by previous analyses indicating that non-adherence significantly increases cardiovascular morbidity and mortality in patients with hypertension, highlighting its critical impact even during acute hypertensive presentations (25). Strategies aimed at improving medication compliance should be prioritised for high-risk groups, particularly those with comorbid CKD (26).

Although no specific antihypertensive drug class was independently associated with outcomes, patients treated with a combination of ACE inhibitors or ARBs and CCBs showed a trend toward lower mortality and rehospitalisation (27). Earlier randomized trials, including ASCOT-BPLA, support this therapeutic strategy. However, the lack of statistical significance in our cohort may reflect variability in dosing, therapy duration, or patient compliance (28).

Receiver operating characteristic curve analysis confirmed moderate predictive accuracy for PLR, SII, homocysteine, and uric acid, with area under the curve values ranging from 0.63 to 0.68. Although these markers are not sufficient on their own for diagnostic use, their combined interpretation with clinical parameters may improve early risk stratification (21). Their accessibility and affordability make them especially useful in routine emergency care.

In-hospital complications such as acute pulmonary oedema, NSTEMI, ischaemic stroke, haemorrhagic stroke, and acute kidney injury further underscore the systemic burden of hypertensive crisis. Among these, acute pulmonary oedema showed an independent association with increased mortality in our cohort, corroborating recent reports that highlight pulmonary complications as strong contributors to early adverse outcomes in hypertensive emergencies (29, 30). In line with this observation, most fatal outcomes in our cohort were attributable to acute pulmonary oedema with respiratory failure, acute coronary syndromes, and severe cerebrovascular events, reflecting the dominant cardiorespiratory and neurovascular mechanisms of early death in hypertensive crisis.

This study has several implications for clinical practice. It emphasises the value of multimodal risk assessments using basic inflammatory and metabolic markers, which are often overlooked in acute care (31). From a guideline perspective, our findings support consideration of simple inflammatory and metabolic biomarkers at hospital admission as adjunct tools for early risk stratification in patients with hypertensive crisis. It also underlines the importance of renal function and treatment adherence, both of which are modifiable risk factors.

However, some limitations must be acknowledged. This was a single-centred study, which may affect the generalisability of the findings. The sample size was sufficient for the primary analyses but insufficient for detailed subgroup analyses. Inflammatory biomarkers were measured only once at admission, so trends over time could not be assessed. Residual confounding is also possible, despite statistical adjustments.

In conclusion, our findings suggest that a combination of inflammatory and metabolic markers, renal function, and therapy adherence influences short-term outcomes in hypertensive crisis. Incorporating parameters such as PLR, SII, homocysteine, and uric acid in risk stratification models could improve the identification of high-risk patients and guide personalised treatment strategies (32).

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Conflicts of interest: None to declare.

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