Research Square

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Association between average systolic blood pressure during the acute phase and mortality in very elderly (≥80 years) patients with acute myocardial infarction

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Abstract

Background

The optimal blood pressure (BP) target during acute myocardial infarction (AMI) in patients aged ≥ 80 years is undefined. We aimed to assess the association between average acute-phase BP and mortality in this population.

Methods

This prospective study included consecutive patients aged ≥ 80 years who were admitted for AMI to the coronary care unit of Peking University Third Hospital between 2013 and 2023. Patients with cardiogenic shock or cardiac arrest at admission were excluded. The in-hospital BPs of each patient were collected and the average in-hospital systolic BP (aSBP) and diastolic BP (aDBP) were calculated for analysis. The primary outcome was 1-year all-cause mortality.

Results

A total of 872 patients were analyzed (mean age 84.2 years, 59.3% male) and 208 patients (23.9%) died within 1 year post-discharge. The association between aSBP and the primary outcomes followed an inverse J-shape curve. Multivariate logistic regression models revealed that compared with aSBP < 120 mm Hg, the risk of 1-year all-cause mortality was reduced by 57% (OR, 0.43; 95% CI, (0.24, 0.78), P = 0.006) in the group of aSBP > 130 mm Hg. The group of 120 to 130 mm Hg showed a trend towards lower mortality risk (OR, 0.86; 95% CI, 0.52–1.43;P = 0.566), but it did not reach statistical significance. The benefit of aSBP ≥ 130 mm Hg was more pronounced in men, patients aged > 85 years, those without diabetes or chronic kidney disease, and those with STEMI or hypertension. The relationship between aDBP and primary outcomes followed a U-shape curve. Patients with aDBP at 60 to 70 mm Hg showed a non-significant trend toward a lower risk. Further multi-center RCTs are warranted to validate our findings and provide evidences for the optimized BP management for very elderly patients in the acute phase of AMI.

Conclusion

Among the very elderly with AMI, an in-hospital average SBP higher than130 mm Hg was significantly associated with a lower risk of 1-year all-cause mortality compared with those having lower aSBP levels.

Introduction

China's rapidly aging population presents a growing healthcare challenge, with the number of individuals aged 80 years or older projected to rise from 19.3 million in 2010 to reach 150 million by 2050 [1].

Cardiovascular (CV) death is the leading cause of mortality in this group, particularly after AMI [2], with prognosis being notably worse than in younger patients [3,4]. In a Swedish cohort, the one-year composite endpoint of non-fatal AMI, stroke, or cardiac mortality exceeded 30% in those aged >80 years [5], highlighting the urgent need to identify modifiable risk factors in this vulnerable population.

BP management in elderly patients with coronary vascular disease (CVD) is a subject of ongoing debate. While hypertension is a well-established risk factor, observational data suggest that excessively low SBP may be associated with increased CV risk in older adults with prior CVD[6-8]. Following the physiological stress of AMI, BP can fluctuate significantly, and therapeutic strategies during the acute phase often differ from those applied in the chronic stable setting. Currently, no consensus from randomized control trials defines an optimal BP target during hospitalization for AMI. Observational studies consistently demonstrate a J- or U-shaped relationship between BP and outcomes, suggesting that excessively low BP (e.g., SBP <110/70 mm Hg) is associated with an increased risk of both in-hospital and long-term adverse events, potentially due to compromised coronary perfusion [9,10].

Admission SBP following AMI is a key component of the Global Registry of Acute Coronary Events (GRACE) risk score and a powerful predictor of both in-hospital and 6-month mortality[11]. A large body of evidence confirms that a low admission SBP correlates with higher mortality in the general AMI population [12-15], and this association extends to elderly patients [16]. However, data detailing the impact of the sustained BP burden throughout the acute phase—specifically the average BP (aBP) under pharmacological intervention—on long-term outcomes in the super-elderly (≥80 years)are limited. Elucidating this relationship could provide a scientific basis for optimizing BP targets during this critical period. Accordingly, this study aimed to investigate the association between aBP during the acute phase and mortality risk in AMI patients aged ≥80 years.

Methods

Study design and population

This was a single-center prospective cohort study of consecutive elderly patients (aged≥80 years) admitted to the Coronary Care Unit of Peking University Third Hospital with acute myocardial infarction (AMI) between Jan 2013 and Dec 2023. The diagnosis of AMI was based on the Fourth Universal Definition of Myocardial Infarction[17]. Patients presented with cardiac arrest and cardiogenic shock at admission, or with missing blood pressure data, or those lost to follow-up were excluded. The study protocol was approved by the Ethics Committee of the Peking University Third Hospital , and informed consent was obtained from the patients.

Measures and data

Blood Pressure Measurement

BP and heart rate were measured using a semi-automatic sphygmomanometer (Philips System Med) according to a standardized protocol. After at least 10 minutes of rest in the supine position, two consecutive readings were taken at 1-minute intervals, and the average was recorded. Measurements were routinely performed twice daily (at 6:00 AM and 4:00 PM) throughout hospitalization. To minimize variability due to length of stay, we calculated the mean in-hospital SBP based on all measurements obtained during the first week of admission. Patients with fewer than three measurements during this period were excluded from the primary analysis. Both average systolic pressures(aSBP) and diastolic pressures (aDBP) were derived.

Clinical data collection and Covariates

Baseline demographic, clinical, and laboratory data were extracted from the electronic medical record system. Covariates included age, sex, smoking status, alcohol consumption, cardiovascular risk factors (hypertension, diabetes mellitus), comorbidities (prior MI, prior cerebral infarction, chronic kidney disease), and admission characteristics (Killip class, heart rate, BP). Blood samples were collected at admission and serially every 4 to 6 hours during the first 48 hours. Peak cardiac troponin T (cTnT) and peak N-terminal pro-B-type natriuretic peptide (NT-proBNP)were defined as the highest recorded values during hospitalization. Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI equation[18]. Echocardiography was performed during the index hospitalization. Left ventricular ejection fraction (LVEF) was assessed using the Simpson's method, and the lowest LVEF within 48 hours of admission was recorded for analysis. In-hospital management data, including medications (RAASi/ARNI, beta-blockers), coronary angiography, percutaneous coronary intervention (PCI), complete revascularization, and in-hospital complications (ventricular arrhythmias, mechanical complications, bleeding events), were also collected.

Outcomes

The primary outcome was all-cause mortality within one year after discharge. Secondary outcomes included: (1) cardiac mortality at 1 year (defined as death from sudden cardiac death, heart failure, recurrent MI, or fatal arrhythmia); (2) in-hospital all-cause mortality; and (3) 30-day all-cause mortality.

Follow-up information was obtained through hospital records or structured telephone interviews with patients or their families. To ascertain the cause of death, two cardiologists who were blinded to the patients' BP status independently reviewed the available medical records and death certificates. Loss to follow-up was 0.8% at 30 days and 1.9% at 1 year.

Statistical Analysis

Continuous variables were presented as mean ± standard deviation (SD), or median with interquartile range (IQR), as appropriate, and categorical variables were expressed as frequencies (percentages), stratified by the primary endpoint. Normality of continuous variables was assessed using the Kolmogorov-Smirnov test. Comparisons between groups were performed using the independent

samples t-test (for normally distributed data) or the Mann-Whitney U test (for non-normally distributed data). Categorical variables were compared using the Chi-square test or Fisher's exact test, as appropriate.

To explore the association between aSBP and the risk of 1-year all-cause mortality, restricted cubic splines (RCS) were fitted. Based on the inflection points observed in the RCS analysis, aSBP was categorized into three subgroups: <120 mm Hg, 120 to 130 mm Hg, and ≥130 mm Hg. Multivariable logistic regression models were constructed to assess the independent association between aSBP categories and the primary outcome. Covariates were selected based on clinical relevance, literature review, and variables showing a univariate association with the outcome at a significance level of $P < 0.10$. The final model adjusted for the following confounders: age, sex, AMI classification, smoking status, alcohol consumption, history of hypertension, diabetes mellitus, previous myocardial infarction, previous cerebral infarction, previous chronic kidney disease, peripheral artery disease, previous PCI, previous coronary artery bypass grafting, heart rate at admission, maximum cTnT, maximum NT-proBNP, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, glycosylated hemoglobin, hematocrit, PCI after AMI, complete revascularization, and hemorrhagic events. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) and P-values were calculated. Sensitivity analyses were performed using logistic regression models stratified by age, sex, comorbidities, and AMI classification to evaluate the consistency of the findings.

All tests were two-tailed, and a P-value < 0.05 was considered statistically significant. Statistical analyses were performed using R software (version 4.3.1) and Empower® software (www.empowerstats.com; X&Y Solutions, Inc., Boston, MA).

Results

Baseline Characteristics

A total of 940 elderly patients (aged≥80 years) with acute STEMI or NSTEMI were admitted to the Coronary Care Unit of PUTH during the enrollment period. Of these, 6 patients presented with cardiac arrest and 41 with cardiogenic shock at admission, 4 had incomplete blood pressure records, and 17 were lost to follow-up at 1 year post-discharge (primarily due to invalid contact information). After these exclusions, 872 patients constituted the final study cohort (*Supplemental Fig. 1*). At admission, 431 patients (49.43%) were diagnosed with Non-ST segment elevation myocardial infarction（NSTEMI）and 441 (50.57%) with ST segment elevation myocardial infarction (STEMI). Regarding mortality, 105 of 889 patients (11.8%) died during hospitalization, 128 of 881 patients (14.5%) died within one month, and 208 of 872 patients (23.9%) died within one year post-discharge due to all causes. Cardiac mortality accounted for 71 deaths (7.99%) in-hospital and 130 deaths (14.9%) at one year.

Table 1summarizes the baseline characteristics of the study population, stratified by 1-year all-cause mortality status. The mean age of the cohort was 84.2 years, and 59.3% were male. Compared with patients who survived at 1 year, those who died were older and had a higher prevalence of comorbidities,

including diabetes mellitus, chronic kidney disease, and atrial fibrillation. Clinically, the mortality group presented with lower SBP and higher heart rate at admission, higher peak values of cTnT and NT-proBNP during hospitalization, lower LVEF and eGFR, and a more severe Killip class (II/III) on admission. They also experienced higher rates of post-AMI complications. In terms of in-hospital management, patients who died within one year had lower rates of prescription for ACEIs/ARBs/ARNI and beta-blockers. They were also less likely to undergo coronary angiography, achieve complete revascularization via PCI, or attain TIMI grade 3 flow. Additionally, the incidence of hemorrhagic events was higher in this group compared to those who were alive at 1 year.

Average SBP and Clinical Outcomes

Primary Outcome: 1-Year All-Cause Mortality

The mean aSBP was 121.24 mm Hg among patients who survived to 1 year and 117.92 mm Hg among those who died. Restricted cubic spline analysis revealed an inverse J-shaped association between aSBP and the risk of 1-year all-cause mortality (*Fig. 1*). The risk decreased sharply with increasing aSBP up to approximately 120 mm Hg, after which the curve gradually plateaued.

In the multivariable-adjusted model (*Table 2*), compared with the reference group (aSBP <120 mm Hg), patients with an aSBP of 120 to 130 mm Hg had a numerically lower but not statistically significant risk of all-cause mortality at 1 year (OR, 0.86; 95% CI, 0.52–1.43;*P*=0.566). In contrast, patients with an aSBP≥ 130 mm Hg had a significantly lower risk of mortality (OR, 0.43; 95% CI, 0.24–0.78;*P*=0.006), indicating that a higher aSBP was independently associated with improved 1-year survival for elder patients with AMI.

Secondary Outcomes

Similar inverse J-shaped associations were observed between aSBP and the risks of 1-year cardiac mortality, 30-day all-cause mortality, in-hospital all-cause mortality, and in-hospital cardiac mortality (*Fig. 1*).

For 1-year cardiac mortality, compared with the <120 mm Hg group, the risk was significantly reduced by 62% in the≥130 mm Hg group (OR, 0.38; 95% CI, 0.18–0.77;*P*=0.007). Although the 120 to 130 mm Hg group showed a trend towards lower risk (OR, 0.87; 95% CI, 0.43–1.36; *P*=0.359), it did not reach statistical significance. For 30-day all-cause mortality, consistent results were observed: compared with the <120 mm Hg group, the OR was 0.88 (95% CI, 0.49–1.58;*P*=0.671) for the 120–130 mm Hg group and 0.38 (95% CI, 0.18–0.80;*P*=0.011) for the≥130 mm Hg group. In-hospital outcomes, although patients with aSBP≥130 mm Hg showed a trend toward lower all-cause mortality (OR, 0.48; 95% CI, 0.22-1.07;*P*=0.073) and cardiac mortality (OR, 0.38; 95% CI, 0.14-1.04;*P*=0.059) compared with the reference group, these differences did not reach statistical significance(*Table 2*).

Subgroup and Sensitivity Analyses

The inverse association between aSBP≥130 mm Hg and lower 1-year mortality was consistent across most subgroups (*Fig. 3*). However, significant interactions were observed for sex, age, diabetes mellitus, prior myocardial infarction, chronic kidney disease, and AMI type (all *P* for interaction < 0.05). The protective effect of higher aSBP was more pronounced in male patients, those aged >85 years, patients without diabetes or chronic kidney disease, and those presenting with STEMI. Notably, among patients with pre-existing hypertension, a higher aSBP (≥130 mm Hg) was also associated with a lower risk of 1-year all-cause mortality.

Average DBP and Clinical Outcomes

The aDBP was similar between survivors and non-survivors at 1 year (64.56 mm Hg vs. 64.36 mm Hg; *P*=0.542). Restricted cubic splines revealed a U-shaped association between aDBP and 1-year all-cause mortality, with the nadir of risk observed between 60 and 70 mm Hg (*Fig.2*).

Using aDBP <60 mm Hg as the reference, patients with aDBP between 60 and 70 mm Hg had a borderline lower risk of 1-year mortality (OR, 0.65; 95% CI, 0.41–1.04; *P*=0.071), whereas those with aDBP >70 mm Hg showed no significant difference (OR, 1.14; 95% CI, 0.62–2.10;*P*=0.673) (*Table 3*).

For short-term outcomes, compared with the <60 mm Hg and >70 mm Hg group, patients with aDBP of 60–70 mm Hg had the lowest risk of in-hospital and 30-day all-cause mortality (both *P*<0.05 for pairwise comparisons with the <60 mm Hg group). Similar trends were observed for in-hospital cardiac mortality, but the differences between groups were not statistically significant (*Table 3*).

Discussion

This study provides novel insights into the association between acute-phase blood pressure (BP) and outcomes in the super-elderly (≥80 years) population with AMI—a cohort historically under-represented in cardiovascular trials. Our primary finding demonstrates that an average systolic blood pressure ≥130 mm Hg during the acute phase was independently associated with lower short- and long-term all-cause mortality, compared with aSBP <120 mm Hg. Additionally, we observed a U-shaped relationship between average DBP and 1-year mortality, with a risk nadir between 60–70 mm Hg, although this association did not reach statistical significance. Collectively, these data suggest that in this vulnerable population, maintaining a higher SBP may confer a survival benefit, challenging the conventional “lower is better” paradigm in all AMI patients.

Current guidelines provide limited guidance on BP targets during the acute phase of AMI. The latest hypertension guidelines focus primarily on long-term secondary prevention rather than acute in-hospital targets[19-21]. A position statement from the British and Irish Hypertension Society suggested avoiding DBP below 70 mm Hg during acute coronary syndromes [22], and recommended a discharge target of <140/90 mm Hg. Similarly, the 2023 AHA/ACC chronic coronary disease guideline suggests a target of <130/80 mm Hg for stable CVD [23], but explicitly notes that the optimal BP for post-ACS patients is not well defined. Observational studies have consistently demonstrated a J- or U-shaped relationship

between BP during follow-up and outcomes after AMI, suggesting that excessively low BP is associated with increased risk of adverse events [9,10]. Our study extends this observation by focusing specifically on the acute phase and the super-elderly—a population often excluded from major trials.

Admission SBP is a well-established component of risk scores such as GRACE and Framingham[11,24], and a higher admission SBP (>120 mm Hg) has been associated with lower risk of death and reinfarction in AMI patients—a finding that contrasts with the “lower is better” paradigm in stable CVD [25]. In a study of elderly patients (≥75 years) with AMI, those with an average SBP <125 mm Hg during the first 48 hours had a twofold higher risk of 1-year cardiovascular death compared with those with SBP ≥125 mm Hg [16]. Similarly, we observed a U-shaped relationship between the admission BP and the risk of mortality (Supplemental Table 1). However, admission SBP represents a single time-point measurement and does not capture the dynamic BP burden during the acute phase, particularly in the context of revascularization, medication, and hemodynamic fluctuations. In our cohort, we observed that a higher aSBP (≥130 mm Hg) was independently associated with lower mortality. This finding aligns with the concept that in the super-elderly, who often have impaired coronary auto-regulation, stiffened vascular structure, and reduced myocardial contractile reserve, maintaining adequate perfusion pressure may be critical to prevent ischemic complications and progression to heart failure. Moreover, we collected pre-admission home BP data in patients with a history of hypertension. Interestingly, the reduction in SBP from pre-AMI home levels to acute-phase in-hospital SBP (ΔSBP) was positively associated with 1-month and in-hospital mortality (*supplemental* Table 1), suggesting that a sharp decline in SBP following AMI may be a marker of hemodynamic instability or an independent risk factor for poor short-term outcomes. This observation further supports avoiding aggressive BP reduction during the acute phase.

In this study, we observed a U-shaped relationship between acute-phase average DBP and 1-year all-cause mortality, with a risk nadir between 60–70 mm Hg; however, this association did not reach statistical significance after multivariable adjustment. Our findings align with the JNC 7 guideline [26], which identified increased risk with DBP <60 mm Hg, and with the PROVE IT-TIMI 22trial, in which DBP <70 mm Hg was associated with higher cardiovascular risk in post-ACS patients[9]. The observed pattern is mechanistically plausible, as coronary perfusion occurs predominantly during diastole, and low DBP may compromise myocardial oxygen delivery, particularly in patients with stiffened arteries and coronary artery disease [27-30].To explore potential effect modification by revascularization, we performed subgroup analyses stratified by PCI status. Among patients who underwent PCI, those with DBP 60–70 mm Hg and >70 mm Hg had 36% and 19% lower mortality, respectively, compared with DBP <60 mm Hg. Among those who did not receive PCI, mortality was 19% lower in the DBP 60–70 mm Hg group but 38% higher in the DBP >70 mm Hg group; however, these differences were not statistically significant. Although the interaction was not significant, these findings suggest that the relationship between DBP and outcomes may differ by revascularization status, potentially because PCI alleviates flow-limiting stenosis and reduces dependence on perfusion pressure. Clinically, these data support the avoidance of aggressive DBP lowering below 60 mm Hg in super-elderly AMI patients, particularly in those with known coronary artery disease.

Landmark RCTs such as PARADIGM-HF have established the efficacy of ACEI/ARNI in HFrEF [31], yet patients aged ≥80 years remain underrepresented. Real-world evidence supports benefits extending to the oldest-old, including those ≥90 years [32]. In our cohort, only 39.45% of patients received RAASi/ARNI, likely reflecting the high prevalence of low BP and declining renal function. In subgroup analyses stratified by medication use, the inverse J-shaped association between aSBP and mortality was directionally consistent, with a trend toward a stronger effect in those receiving RAASi/ARNI(*P* for interaction=0.036). These data suggest that the protective effect of higher aSBP is not confounded by differential medication use.

A key methodological challenge in observational BP studies is reverse causality—the possibility that low BP is merely a marker of underlying disease severity rather than a true risk factor. To mitigate this concern, we employed several strategies: (1) exclusion of patients with cardiac arrest or cardiogenic shock; (2) multivariable adjustment for a broad range of confounders, including comorbidities and cardiac function; and (3) sensitivity analyses stratified by revascularization status. The rationale for the latter is that if the observed harm of low BP were entirely attributable to reverse causality, revascularization—which alleviates the severity of underlying coronary disease—would not be expected to modify the association. In our cohort, revascularization did not materially alter the relationship between BP and mortality, arguing against reverse causality as the sole explanation. This interpretation is further supported by Mendelian randomization studies demonstrating that MI itself does not lead to sustained changes in BP [33]. Nevertheless, as with all observational studies, residual confounding cannot be completely excluded.

Study Limitation

This study has several limitations. First, the observational, single-center design with a moderate sample size limits causal inference and generalizability; multi-center RCTs are warranted to validate our findings. Second, despite adjusting for a broad range of potential confounders, we cannot exclude the possibility of residual confounding from unmeasured factors, including frailty[34], cognitive function, and post-discharge medication adherence. Third, and importantly, 24-hour ambulatory blood pressure monitoring (ABPM)—considered the gold standard for BP assessment and recommended by current hypertension guidelines was not performed[19,20]. ABPM would have provided valuable data on BP variability, circadian patterns, and overall BP burden, which may offer stronger prognostic value than in-hospital measurements. Finally, while only 57.68% of patients underwent PCI and the rate was similar with the researches published before—reflecting real-world clinical decision-making that balances bleeding risk, comorbidities, and patient preferences—stratified analyses confirmed that revascularization status did not significantly alter the observed association between acute-phase BP and mortality. This suggests that our primary findings are robust and not substantially confounded by treatment selection.

Conclusion

Our findings suggest that in AMI patients aged ≥ 80 years, maintaining an average SBP ≥ 130 mm Hg during the acute phase is associated with lower mortality, while aggressive BP reduction should be avoided. These results support the concept of age- and context-specific BP targets in this vulnerable population. From a clinical perspective, this implies that for super-elderly patients with AMI, a cautious approach to blood pressure management—prioritizing the avoidance of hypotension over intensive lowering—may be beneficial. Further prospective studies are warranted to validate these findings and to establish optimal blood pressure ranges that balance efficacy and safety in routine geriatric care.

Abbreviations

BP
blood pressure
AMI
acute myocardial infarction
SBP
systolic BP
DBP
diastolic BP
CV
Cardiovascular
CVD
coronary vascular disease
GRACE
the Global Registry of Acute Coronary Events
cTnT
cardiac troponin T
NT-proBNP
N terminal pro-B-type natriuretic peptide
eGFR
Estimated glomerular filtration rate
LVEF
Left ventricular ejection fraction
PCI
percutaneous coronary intervention
RCS
restricted cubic splines
OR
odds ratio
CI
confidence interval

NSTEMI
Non-ST segment elevation myocardial infarction
STEMI
ST segment elevation myocardial infarction
ABPM
ambulatory blood pressure monitoring

Declarations

Author contributions

All authors have read and approved the submission of this manuscript. FYY contributed to the acquisition, analysis, interpretation of data. SZ, LXJ, XPX, XY and LFM contributed to the study concept and interpretation of data. ZLY and XWX were responsible for designing the study, supervision, and drafting the manuscript. All authors contributed to critical revisions of the manuscript for important intellectual content.

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Declarations Ethics Approval and Consent to Participate

The study was carried out in accordance with the Declaration of Helsinki and participants gave written informed consent. The study protocol was approved by the Ethics Committee of the Peking University Third Hospital.

Clinical trial number: not applicable.

Availability of data

Data are available upon reasonable request）

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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Tables

Table 1
Characteristics of the Study Participants.

Characteristics	Total	No All-Cause Mortality in 1 year	All-Cause Mortality in 1 year	P value
	(N = 872)	(N = 664)	(N = 208)	
Age, y	84.20 ± 3.62	83.74 ± 3.33	85.68 ± 4.07	< 0.001
Male, N (%)	517(59.29)	395 (59.49)	122 (58.65)	0.432
Never Smoked, N (%)	585(67.08)	447 (67.32)	138 (66.99)	0.930
Alcohol consumption, N (%)	143(16.40)	105 (16.01)	38 (18.72)	0.364
Hypertension, N (%)	665(76.26)	505 (76.05)	160 (76.92)	0.797
Diabetes mellitus, N (%)	367(42.09)	266 (40.06)	101 (48.56)	0.030
Previous myocardial infarction, N (%)	122(13.99)	85 (12.80)	37 (17.79)	0.070
Previous cerebral infarction, N (%)	185(21.21)	131 (19.73)	54 (25.96)	0.055
Chronic kidney disease, N (%)	171(19.61)	90 (13.55)	81 (38.94)	< 0.001
COPD, N (%)	63 (7.22)	43 (6.48)	20 (9.62)	0.127
Peripheral artery disease, N (%)	74(8.49)	53 (7.98)	21 (10.10)	0.340
Atrial fibrillation,, N (%)	145(16.63)	90 (13.55)	55 (26.44)	< 0.001
Previous PCI, N (%)	143(16.39)	121 (18.22)	22 (10.58)	0.009
Previous CABG, N (%)	24(2.75)	18 (2.71)	6 (2.88)	0.894
Malignant tumor, N (%)	105(12.04)	73 (10.99)	32 (15.38)	0.094
Clinical parameters				
SBP at admission, mmHg	131.17 ± 19.59	132.28 ± 19.43	127.63 ± 20.03	< 0.001
DBP at admission, mmHg	71.10 ± 11.71	70.92 ± 11.72	71.61 ± 11.79	0.392

COPD indicates Chronic obstructive pulmonary disease; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; SBP, systolic blood pressure; DBP, diastolic systolic blood pressure; HR, heart rate; aSBP, average SBP during hospitalization; aDBP, average DBP during hospitalization; AMI, acute myocardial infarction; cTnT max: the maximum value of cardiac troponin T; CK-MB max, the maximum value of the creatine kinase isoenzyme type B; Hct Hematocrit; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVEF, left ventricle ejection fraction;SGLT-2I, sodium-dependent glucose transporters 2 inhibitor. *For continuous variables, values are presented as mean ± SD.

Characteristics	Total	No All-Cause Mortality in 1 year	All-Cause Mortality in 1 year	P value
	(N = 872)	(N = 664)	(N = 208)	
HR at admission, beat per min	79.45 ± 16.61	77.22 ± 15.57	86.83 ± 17.97	< 0.001
average SBP, mmHg	120.41 ± 11.81	121.24 ± 11.40	117.92 ± 12.80	< 0.001
average DBP, mmHg	64.52 ± 6.48	64.56 ± 6.14	64.36 ± 7.52	0.542
AMI classification, N (%)				0.279
NSTEMI	431(49.43)	335 (50.45)	96 (46.15)	
STEMI	441(50.57)	329 (49.55)	112 (53.85)	
Anterior	214(24.54)	154 (23.19)	60 (28.85)	
Inferior or posterior or right ventricle	108(12.39)	161 (24.25)	47 (22.60)	
Laterior	19(2.18)	14 (2.11)	5 (2.40)	
Mechanical complication, N (%)	24(2.75)	12 (1.81)	12 (5.77)	0.002
Killip grading, N (%)				< 0.001
I	434(49.77)	378 (56.92)	56 (26.92)	
II	282(32.34)	209 (31.48)	73 (35.10)	
III	156(17.89)	77 (11.60)	79 (37.98)	
Laboratory results				
NT-proBNP max, pg/ml	8930.39 ± 10797.48	6019.22 ± 7565.51	18440.61 ± 13890.68	< 0.001
cTnT max, ng/ml	4.00 ± 4.86	3.46 ± 4.46	5.71 ± 5.69	< 0.001
CK-MB max, IU/L	181.06 ± 301.39	172.62 ± 318.33	212.60 ± 244.59	< 0.001
HbA1C,%	6.73 ± 1.47	6.75 ± 1.52	6.70 ± 1.32	0.574

COPD indicates Chronic obstructive pulmonary disease; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; SBP, systolic blood pressure; DBP, diastolic systolic blood pressure; HR, heart rate; aSBP, average SBP during hospitalization; aDBP, average DBP during hospitalization; AMI, acute myocardial infarction; cTnT max: the maximum value of cardiac troponin T; CK-MB max, the maximum value of the creatine kinase isoenzyme type B; Hct Hematocrit; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVEF, left ventricle ejection fraction;SGLT-2I, sodium-dependent glucose transporters 2 inhibitor. *For continuous variables, values are presented as mean ± SD.

Characteristics	Total	No All-Cause Mortality in 1 year	All-Cause Mortality in 1 year	P value
	(N = 872)	(N = 664)	(N = 208)	
HCT, %	0.36 ± 0.06	0.37 ± 0.06	0.34 ± 0.06	< 0.001
Total cholesterol, mmol/L	3.96 ± 1.06	3.97 ± 1.05	3.95 ± 1.10	0.433
HDL cholesterol, mmol/L	1.01 ± 0.27	1.02 ± 0.27	1.01 ± 0.28	0.801
Triglycerides, mmol/L	1.31 ± 0.63	1.30 ± 0.62	1.34 ± 0.69	0.721
LDL cholesterol, mmol/L	2.39 ± 0.87	2.41 ± 0.86	2.34 ± 0.87	0.152
eGFR, mL/min per 1.73 m^2	57.42 ± 21.63	61.47 ± 20.04	44.42 ± 21.55	< 0.001
LVEF, %	50.58 ± 12.58	52.62 ± 11.89	43.75 ± 12.43	< 0.001
Treatment during hospitalization, N (%)				
ACE inhibitors or ARBs or ARNIs	344(39.45)	311 (46.84)	33 (15.94)	< 0.001
B receptor blockers	409 (46.90)	353 (53.16)	56 (27.05)	< 0.001
Aldosterone antagonists	99 (11.35)	74 (11.16)	25 (12.08)	0.717
SGLT-2I	40(4.59)	34 (5.13)	6 (2.90)	0.181
Coronary angiograph, N (%)	551(63.19)	464 (69.88)	87 (41.83)	< 0.001
PCI, N (%)	503 (57.68)	429 (64.61)	74 (35.58)	< 0.001
Number of stenosis coronary artery				0.017
0	8 (1.45)	7 (1.51)	1 (1.15)	
1	111 (20.14)	103 (22.15)	8 (9.20)	
2	182 (33.03)	155 (33.33)	27 (31.03)	
3	251 (45.55)	200 (43.01)	51 (58.62)	

COPD indicates Chronic obstructive pulmonary disease; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; SBP, systolic blood pressure; DBP, diastolic systolic blood pressure; HR, heart rate; aSBP, average SBP during hospitalization; aDBP, average DBP during hospitalization; AMI, acute myocardial infarction; cTnT max: the maximum value of cardiac troponin T; CK-MB max, the maximum value of the creatine kinase isoenzyme type B; Hct Hematocrit; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVEF, left ventricle ejection fraction;SGLT-2I, sodium-dependent glucose transporters 2 inhibitor. *For continuous variables, values are presented as mean ± SD.

Characteristics	Total	No All-Cause Mortality in 1 year	All-Cause Mortality in 1 year	P value
	(N = 872)	(N = 664)	(N = 208)	
Complete revascularization, N (%)	152 (30.21)	140 (21.12)	12 (5.77)	< 0.001
Reperfusion of culprit artery, N (%)	465 (84.39)	400 (86.21)	65 (74.71)	< 0.001
Final TIMI Grade III, N (%)	492(89.29)	425 (91.79)	67 (77.01)	< 0.001
Hemorrhage events, N (%)	43(4.93)	20 (3.02)	23 (11.11)	< 0.001
Hospital stay, d	10.91 ± 7.58	10.51 ± 6.23	12.32 ± 10.80	0.002

COPD indicates Chronic obstructive pulmonary disease; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; SBP, systolic blood pressure; DBP, diastolic systolic blood pressure; HR, heart rate; aSBP, average SBP during hospitalization; aDBP, average DBP during hospitalization; AMI, acute myocardial infarction; cTnT max: the maximum value of cardiac troponin T; CK-MB max, the maximum value of the creatine kinase isoenzyme type B; Hct Hematocrit; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVEF, left ventricle ejection fraction;SGLT-2I, sodium-dependent glucose transporters 2 inhibitor. *For continuous variables, values are presented as mean ± SD.

Table 2

Comparisons of Primary and Secondary Outcomes by Averaged Systolic Blood Pressure Categories.

Average SBP Categories (mmHg)	Outcomes N (%)	OR (95CI)	P value	OR (95CI)	P value
		Unadjusted		Adjusted[#]	
All-cause mortality in 1 year (N = 208/872)					
< 120	120 (26.32)	1.0 (ref)		1.0 (ref)	
120–130	53 (23.66)	0.87 (0.59, 1.26)	0.455	0.86 (0.52, 1.43)	0.566
> 130	35 (18.23)	0.62 (0.41, 0.95)	0.028	0.43 (0.24, 0.78)	0.006
Cardiac mortality in 1 year (N = 130/872)					
< 120	79 (17.32)	1.0 (ref)		1.0 (ref)	
120–130	31 (13.96)	0.78 (0.49, 1.21)	0.266	0.76 (0.43, 1.36)	0.359
> 130	20 (10.42)	0.56 (0.33, 0.94)	0.027	0.38 (0.18, 0.77)	0.007
All-cause mortality in 1 month (N = 128/881)					
< 120	79 (17.06)	1.0 (ref)		1.0 (ref)	
120–130	31 (13.78)	0.78 (0.49, 1.22)	0.271	0.88 (0.49, 1.58)	0.671
> 130	18 (9.33)	0.50 (0.29, 0.86)	0.012	0.38 (0.18, 0.80)	0.011
All cause mortality in hospital (N = 105/889)					
< 120	67 (14.32)	1.0 (ref)		1.0 (ref)	
120–130	22 (9.69)	0.64 (0.39, 1.07)	0.089	0.75 (0.38, 1.47)	0.398
> 130	16 (8.21)	0.54 (0.30, 0.95)	0.032	0.48 (0.22, 1.07)	0.073
Cardiac mortality in hospital (N = 71/889)					

Adjusted for gender,age, AMI classification, smoking condition, alcohol consumption, history of hypertension, diabetes mellitus, previous myocardial infarction, previous cerebral infarction, chronic kidney disease, peripheral artery disease, previous PCI, previous CABG, HR at admission, cTnT max, NT-proBNP max, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, HbA1c, Hct, PCI after AMI, complete coronary vascularization and hemorrhage events. CI indicates confidence interval; OR, odd ratio.

Average SBP Categories (mmHg)	Outcomes N (%)	OR (95CI)	P value	OR (95CI)	P value
		Unadjusted		Adjusted[#]	
All-cause mortality in 1 year (N = 208/872)					
< 120	46 (9.83)	1.0 (ref)		1.0 (ref)	
120–130	15 (6.61)	0.65 (0.35, 1.19)	0.162	0.90 (0.41, 1.99)	0.806
> 130	10 (5.15)	0.50 (0.25, 1.01)	0.053	0.38 (0.14, 1.04)	0.059

Adjusted for gender,age, AMI classification, smoking condition, alcohol consumption, history of hypertension, diabetes mellitus, previous myocardial infarction, previous cerebral infarction, chronic kidney disease, peripheral artery disease, previous PCI, previous CABG, HR at admission, cTnT max, NT-proBNP max, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, HbA1c, Hct, PCI after AMI, complete coronary vascularization and hemorrhage events. CI indicates confidence interval; OR, odd ratio.

Table 3

Comparisons of Primary and Secondary Outcomes by Averaged Diastolic Blood Pressure Categories.

Average SBP Categories (mmHg)	Outcomes N (%)	OR (95CI)	P value	OR (95CI)	P value
		Unadjusted		Adjusted[#]	
All-cause mortality in 1 year (N = 208/872)					
< 60	63 (30.29)	1.0 (ref)		1.0 (ref)	
60–70	103 (20.04)	0.58 (0.40, 0.83)	0.003	0.65 (0.41, 1.04)	0.071
> 70	42 (27.52)	0.87 (0.55, 1.39)	0.570	1.14 (0.62, 2.10)	0.673
Cardiac mortality in 1 year (N = 130/872)					
< 60	42 (20.19)	1.0 (ref)		1.0 (ref)	
60–70	65 (12.67)	0.57 (0.37, 0.88)	0.011	0.66 (0.39, 1.12)	0.121
> 70	23 (15.07)	0.69 (0.39, 1.21)	0.569	0.73 (0.36, 1.49)	0.388
All-cause mortality in 1 month (N = 128/881)					
< 60	39 (18.57)	1.0 (ref)		1.0 (ref)	
60–70	59 (11.37)	0.56 (0.36, 0.87)	0.011	0.55 (0.31, 0.97)	0.039
> 70	30 (19.74)	1.09 (0.64, 1.85)	0.757	1.22 (0.59, 2.53)	0.597
All cause mortality in hospital (N = 105/889)					
< 60	33 (15.42)	1.0 (ref)		1.0 (ref)	
60–70	46 (8.79)	0.52 (0.32, 0.84)	0.008	0.52 (0.27, 0.97)	0.041
> 70	26 (17.10)	1.12 (0.64, 1.96)	0.703	1.49 (0.68, 3.28)	0.320
Cardiac mortality in hospital (N = 71/889)					

Adjusted for gender,age, AMI classification, smoking condition, alcohol consumption, history of hypertension, diabetes mellitus, previous myocardial infarction, previous cerebral infarction, chronic kidney disease, peripheral artery disease, previous PCI, previous CABG, HR at admission, cTnT max, NT-proBNP max, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, HbA1c, Hct, PCI after AMI, complete coronary vascularization and hemorrhage events. CI indicates confidence interval; OR, odd ratio.

Average SBP Categories (mmHg)	Outcomes N (%)	OR (95CI)	P value	OR (95CI)	P value
		Unadjusted		Adjusted#	
All-cause mortality in 1 year (N = 208/872)					
< 60	23 (10.74)	1.0 (ref)		1.0 (ref)	
60–70	32 (6.12)	0.53 (0.30, 0.94)	0.029	0.57 (0.28, 1.15)	0.114
> 70	16 (10.53)	0.97 (0.49, 1.91)	0.932	1.06 (0.44, 2.56)	0.904

Adjusted for gender,age, AMI classification, smoking condition, alcohol consumption, history of hypertension, diabetes mellitus, previous myocardial infarction, previous cerebral infarction, chronic kidney disease, peripheral artery disease, previous PCI, previous CABG, HR at admission, cTnT max, NT-proBNP max, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, HbA1c, Hct, PCI after AMI, complete coronary vascularization and hemorrhage events. CI indicates confidence interval; OR, odd ratio.

Figures

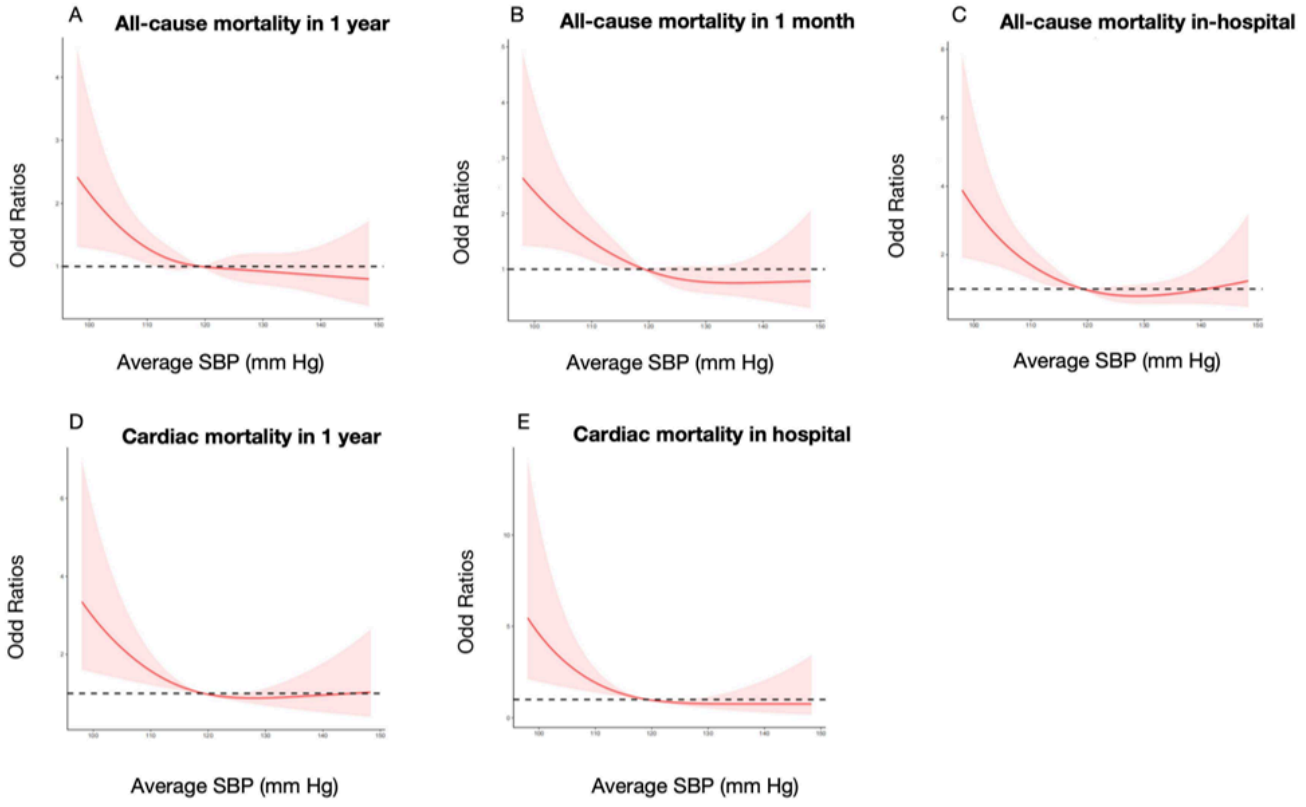


Figure 1

The association between the average in-hospital systolic blood pressure (Average SBP) following acute myocardial infarction with the risk for all-cause mortality in 1 year (A), all-cause mortality in 1 month (B), all-cause mortality in-hospital (C), cardiac mortality in 1 year (D) and cardiac mortality in hospital (E).

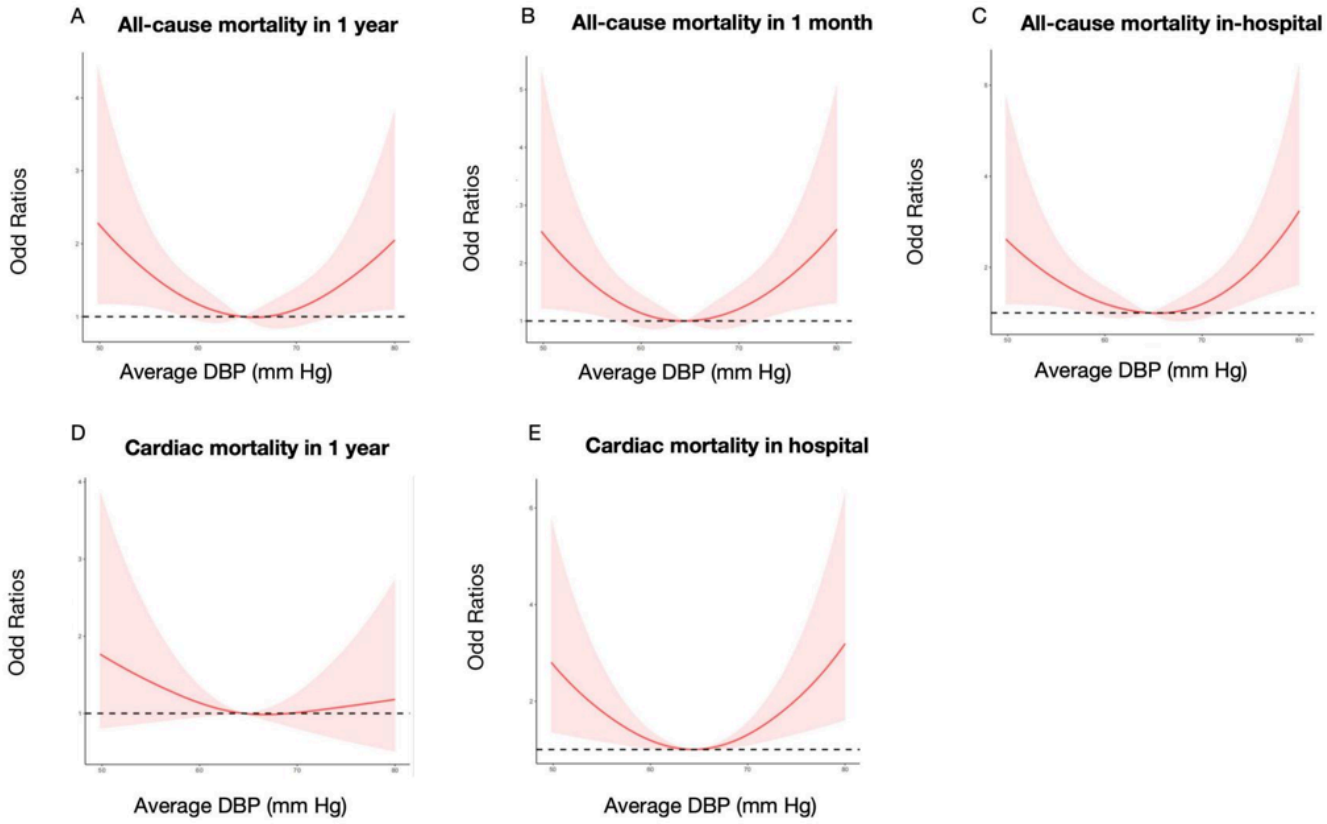


Figure 2

The association between the average in-hospital diastolic blood pressure (Average DBP) following acute myocardial infarction with the risk for all-cause mortality in 1 year (A), all-cause mortality in 1 month (B), all-cause mortality in-hospital (C), cardiac mortality in 1 year (D) and cardiac mortality in hospital (E).

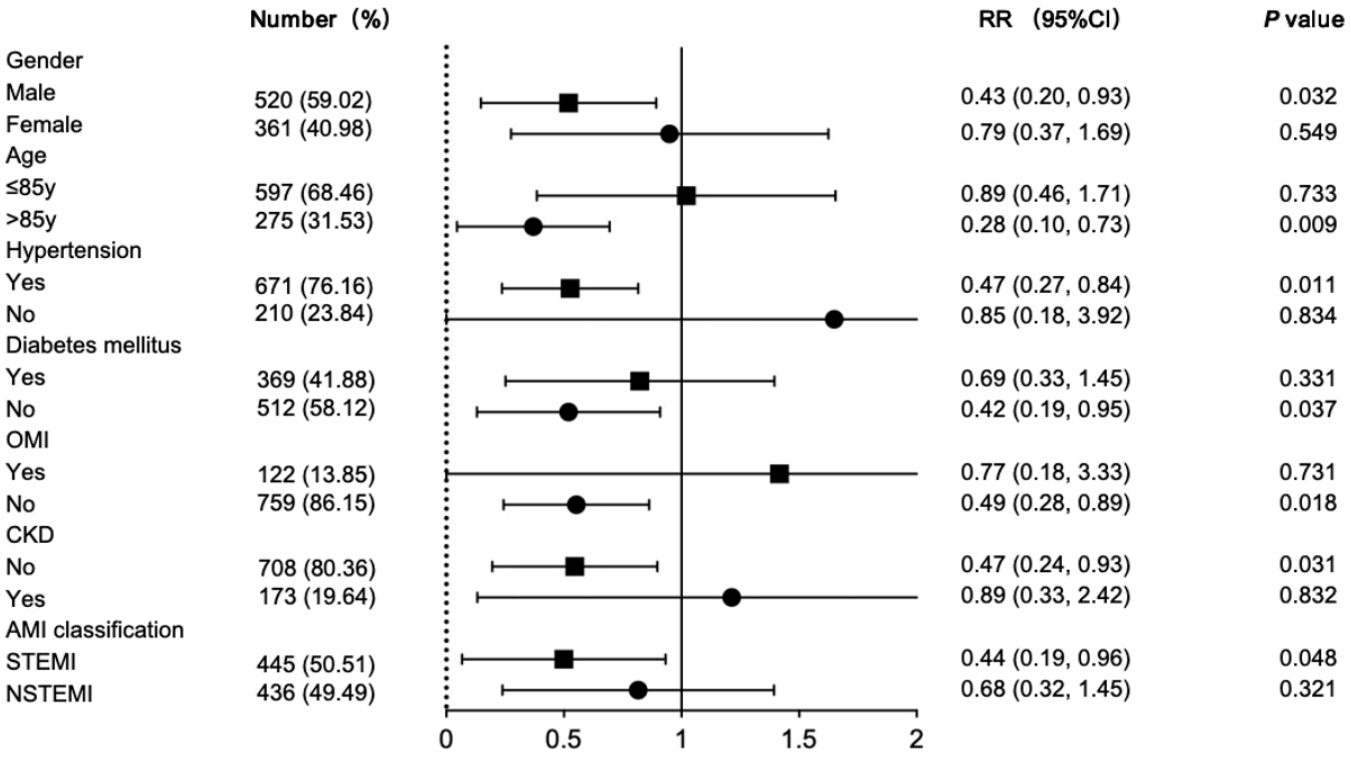


Figure 3

Stratified analysis of average SBP >130 mmHg versus ≤130 mmHg for 1-year all-cause mortality across various subgroups.

OMI: old myocardial Infarction; CKD: chronic kidney disease; STEMI: ST-Elevated Myocardial Infarction; NSTEMI: non ST-Elevated Myocardial Infarction.

Supplementary Files

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