

Prognostic significance of stress hyperglycemia ratio in patients undergoing percutaneous coronary intervention: a dose-response meta-analysis

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ABSTRACT

Background Stress hyperglycemia is common during percutaneous coronary intervention (PCI) and is associated with worse outcomes. The stress hyperglycemia ratio (SHR), which standardizes acute glucose levels to chronic glycemic status, may provide superior prognostic information compared with admission glucose alone.

Objective To systematically evaluate the association between SHR and clinical outcomes in PCI patients and to quantify the exposure-response relationship.

Data sources PubMed, Embase, Cochrane Library, Web of Science, Scopus, and ClinicalTrials.gov were searched from inception to August 5, 2025.

Study selection Observational studies reporting SHR prior to PCI and subsequent clinical outcomes were included.

Data extraction Two investigators independently extracted study characteristics, SHR definitions, and adjusted effect estimates. Risk of bias was assessed using ROBINS-I (Risk Of Bias In Non-randomized Studies of Interventions), and certainty of evidence was evaluated with the Grading of Recommendations Assessment, Development and Evaluation framework.

Data synthesis Thirteen studies comprising 41 096 PCI patients were included. Elevated SHR was associated with increased risks of all-cause mortality, major adverse cardiac and cerebrovascular events (MACCE), cardiovascular mortality, recurrent myocardial infarction, and stroke, but not with repeat revascularization. Sensitivity analyses yielded consistent results. The dose-response analysis suggested an approximately linear dose-response pattern between SHR and MACCE.

Limitations Most studies were observational, SHR definitions varied, and residual confounding cannot be excluded. The limited number of studies restricted the interpretability of dose-response analyses.

Conclusions Elevated SHR was associated with an increased risk of adverse outcomes after PCI. The dose-response analysis suggested an approximately linear dose-response pattern between SHR and MACCE. However, these findings should be interpreted cautiously given the observational nature of the evidence and the limited number of studies for certain analyses.

PROSPERO registration number CRD420251045853.

WHAT IS ALREADY KNOWN ON THIS TOPIC

- ⇒ Stress hyperglycemia has been associated with adverse outcomes in patients with cardiovascular disease.
- ⇒ However, evidence regarding the prognostic value of the stress hyperglycemia ratio (SHR) in patients undergoing percutaneous coronary intervention (PCI) has been inconsistent, and the dose-response relationship between SHR and clinical outcomes has not been clearly quantified.

WHAT THIS STUDY ADDS

- ⇒ This meta-analysis found that elevated SHR was associated with increased risks of all-cause mortality, major adverse cardiac and cerebrovascular events (MACCE), cardiovascular mortality, recurrent myocardial infarction, and stroke in patients undergoing PCI.
- ⇒ The dose-response analysis suggested an approximately linear association between SHR and MACCE within the observed range of data.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

- ⇒ These findings suggest that SHR may have potential as a marker for risk stratification after PCI, which could help identify patients at higher risk of adverse outcomes related to stress-induced dysglycemia.
- ⇒ Future studies may consider incorporating SHR into prognostic models in PCI populations.
- ⇒ Further prospective research is needed to validate these findings and to clarify the risks associated with extreme low SHR values.

INTRODUCTION

Coronary atherosclerotic heart disease remains one of the most prevalent cardiovascular conditions worldwide, with persistently high incidence and mortality rates. In particular, acute coronary syndrome (ACS) is characterized by complex clinical presentations,

a high burden of complications, and frequent delays in treatment, collectively contributing to substantial mortality and public health burden. As the cornerstone of revascularization therapy, percutaneous coronary intervention (PCI) restores coronary blood flow through balloon angioplasty or stent implantation and has significantly improved clinical outcomes in patients with coronary artery disease.^{1,2}

Inflammation plays a central role in the initiation and progression of coronary artery disease. PCI itself can induce procedure-related vascular injury and inflammatory activation, thereby promoting neointimal hyperplasia, thrombosis, and restenosis.^{3,4} Notably, this PCI-related inflammatory stress is often accompanied by acute disturbances in glucose metabolism, suggesting that procedure-related and disease-related metabolic responses may carry prognostic significance beyond baseline glycemic status.

Hyperglycemia occurring under acute disease-related stress, termed stress hyperglycemia, has been well established as an independent predictor of adverse outcomes in patients with ACS, particularly those with acute myocardial infarction.⁵ Early studies primarily relied on admission blood glucose (ABG) as the exposure of interest; however, its prognostic value is limited because ABG reflects both acute glycemic fluctuations and chronic glycemic exposure. To address this limitation, Roberts and colleagues proposed the stress hyperglycemia ratio (SHR), which standardizes admission glucose to the estimated chronic glycemic level derived from glycated hemoglobin (HbA1c), thereby more accurately capturing the acute stress-related glycemic response.⁶ Recent studies suggest that SHR provides better prognostic information compared with blood glucose alone and is now widely used in ACS and PCI patients.⁷

To date, no meta-analysis has systematically evaluated the association between SHR and clinical outcomes after PCI. Therefore, we conducted this systematic review and meta-analysis to address this gap. Previous studies have predominantly dichotomized SHR into high and low categories, an approach that overlooks its nature as a continuous variable and relies on arbitrarily defined cut-off values. In this study, we treated SHR as a continuous variable and performed dose-response meta-analyses to further explore its potential prognostic implications in patients undergoing PCI.

MATERIALS AND METHODS

Protocol and registration

This systematic review and meta-analysis was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (online supplemental table S1) and was prospectively registered in the PROSPERO database (CRD420251045853). The full registration record is available at: <https://www.crd.york.ac.uk/PROSPERO/view/CRD420251045853>.

Search strategies

We systematically searched PubMed, Embase, the Cochrane Library, Web of Science, Scopus, and ClinicalTrials.gov from inception to August 5, 2025. Reference lists of relevant reviews were also screened manually to identify additional eligible studies. No language restrictions were applied. A comprehensive search strategy combining Medical Subject Headings (MeSH) terms and free-text keywords was used. Key terms included 'stress hyperglycaemia ratio', 'percutaneous coronary intervention', 'major adverse cardiac and cerebrovascular events', 'major adverse cardiovascular events', 'all-cause mortality', 'cardiovascular mortality', 'recurrent myocardial infarction', 'stroke', and 'revascularisation'. The complete search strategy is provided in online supplemental table S2.

Study selection

Studies were included if they met the following criteria: (1) retrospective or prospective cohort studies with full-text availability; (2) $\geq 80\%$ of the study population undergoing PCI; (3) reporting at least two categories of SHR; (4) reporting at least one clinical outcome, including major adverse cardiovascular events (MACEs), major adverse cardiac and cerebrovascular events (MACCEs), all-cause mortality, cardiovascular mortality, recurrent myocardial infarction, stroke, or revascularization. Studies reporting MACE were included if definitions aligned with our MACCE composite endpoint.

Exclusion criteria are as follows: (1) reviews, meta-analyses, animal studies, abstracts, or letters; (2) absence of baseline SHR or irrelevant exposure definition; (3) missing outcome data; (4) duplicate publications.

Outcome definitions

The primary outcomes were all-cause mortality and MACCE. MACCE was defined as a composite of all-cause mortality, cardiovascular mortality, non-fatal myocardial infarction, stroke (ischemic, hemorrhagic, or transient ischemic attack), and coronary revascularization.^{8,9} Studies that originally reported MACE were included if their reported events were consistent with this composite definition. This ensured consistency in outcome definitions across studies. Secondary outcomes included cardiovascular mortality, recurrent myocardial infarction, stroke, and repeat revascularization. Detailed outcome definitions for each study are provided in online supplemental table S3.

Data extraction

Two investigators independently screened studies and extracted data using a standardized form. After duplicates were removed, titles and abstracts were screened, followed by full-text review. Reference lists were also examined to identify additional eligible studies. Extracted information included: (1) author, year of publication, and country/region; (2) study design; (3) patient characteristics, including sample size, age, sex, baseline SHR,

proportion of ACS, proportion of PCI, prevalence of diabetes, outcome events, comorbidities, and medical history; (4) raw outcome event counts and/or effect estimates with 95% CI; (5) follow-up duration; and (6) definitions of SHR and clinical outcomes.

Effect estimates from multivariable models adjusted for cardiovascular risk factors (eg, hypertension, dyslipidemia, diabetes, sex, smoking) were prioritized. If data were missing and could not be obtained from other publications by the same research group, corresponding authors were contacted for additional information. Discrepancies were resolved through discussion or consultation with a third reviewer.

Statistical analysis

Definitions of SHR varied across studies. SHR1 was defined as admission blood glucose (mg/dL or mmol/L) divided by HbA1c-derived estimated chronic glucose, whereas SHR2 used fasting plasma glucose as the numerator with the same denominator. Although admission and fasting glucose reflect acute stress-related hyperglycemia and basal metabolic status, respectively, both capture peri-procedural stress responses during PCI and share similar pathophysiological mechanisms. For the primary analysis, a linear relationship was assumed. To improve statistical power and precision, SHR1 and SHR2 were pooled and analyzed as SHR, with prespecified subgroup and sensitivity analyses conducted to assess the robustness of this approach.

All analyses were performed using Stata V.18.0, with two-sided $p \leq 0.05$ considered statistically significant. Relative risks (RRs) and their 95% CIs were calculated based on the number of outcome events in the highest versus lowest SHR categories in each study. Heterogeneity was assessed using Cochran's Q test and the I^2 statistic, and random-effects models were applied throughout. Potential sources of heterogeneity were explored using subgroup, sensitivity, and leave-one-out analyses.

Subgroup analyses were prespecified for primary outcomes only (MACCE and all-cause mortality) and stratified by SHR definition, clinical presentation (ACS vs non-ACS or mixed populations), and PCI proportion (100% vs <100%). Subgroup interactions were assessed using interaction tests, and outcomes with insufficient studies were not analyzed to avoid unstable estimates.

To explore potential sources of heterogeneity for MACCE and all-cause mortality, study-level meta-regression and subgroup analyses were performed. Random-effects meta-regression models were fitted using restricted maximum likelihood, with the natural logarithm of the risk ratio ($\ln RR$) as the dependent variable.¹⁰ Diabetes mellitus (DM) prevalence was modeled as a continuous moderator per 10% increment. The regression coefficient (β) represents the change in $\ln RR$ per 10% increase in DM prevalence. Categorical variables were analyzed using indicator variables. Given substantial variation in DM prevalence across studies and the limited number of studies within certain strata, subgroup

analyses were conducted descriptively where appropriate. Meta-regression was additionally used to assess potential effect modification at the study level. Sensitivity analyses were performed by excluding studies with extreme DM prevalence (0% or 100%) to evaluate the robustness of the findings. Meta-regression results reflect study-level associations rather than individual-level effects. Given the limited number of included studies, meta-regression analyses were exploratory in nature.

Dose-response meta-analyses were conducted when ≥ 3 studies reported outcomes across ≥ 2 SHR categories, including all-cause mortality and MACCE. Adjusted HRs with 95% CIs were preferentially extracted to ensure comparability. Studies reporting ORs with low event rates were considered approximate HRs^{11 12} (online supplemental table S4). ORs from low event studies were included only in sensitivity analyses to explore consistency of effect direction rather than to represent direct HRs. Dose-response reconstruction was performed using the Greenland and Longnecker approach, assuming log-normal risk distributions.¹³ Restricted cubic spline models with three knots placed at the 10th, 50th, and 90th percentiles were used to explore potential non-linear dose-response relationships. Sensitivity analyses using alternative knot specifications were performed to evaluate robustness.¹⁴ For open-ended exposure categories, midpoints were estimated using standard methods: the lowest category midpoint was set as the upper boundary minus half the adjacent interval width, and the highest category midpoint as the lower boundary plus half the adjacent interval width. When fewer than three studies remained for a given outcome, dose-response analyses were interpreted descriptively rather than as formal spline models. All statistical analyses and interpretation were reviewed by a trained biostatistician.

Risk of bias and certainty of evidence

Risk of bias and certainty of evidence were independently assessed by two investigators, with disagreements resolved by a third reviewer. Risk of bias in non-randomized studies was evaluated using the Risk Of Bias In Non-randomized Studies of Interventions (ROBINS-I) tool, assessing seven domains: (1) confounding; (2) participant selection; (3) intervention classification; (4) deviations from intended interventions; (5) missing data; (6) outcome measurement; and (7) selection of the reported result.

Study quality was additionally assessed using the Newcastle-Ottawa Scale (NOS), evaluating three domains: selection of study groups, comparability between groups, and outcome assessment, with a maximum score of 9 points. Studies scoring ≥ 7 were considered high quality. NOS was used as a complementary assessment, while ROBINS-I was prioritized for evidence grading within the Grading of Recommendations Assessment, Development and Evaluation (GRADE) framework. Results from both tools are presented in the online supplemental materials.

Publication bias was assessed using funnel plots when appropriate, with asymmetry interpreted cautiously due

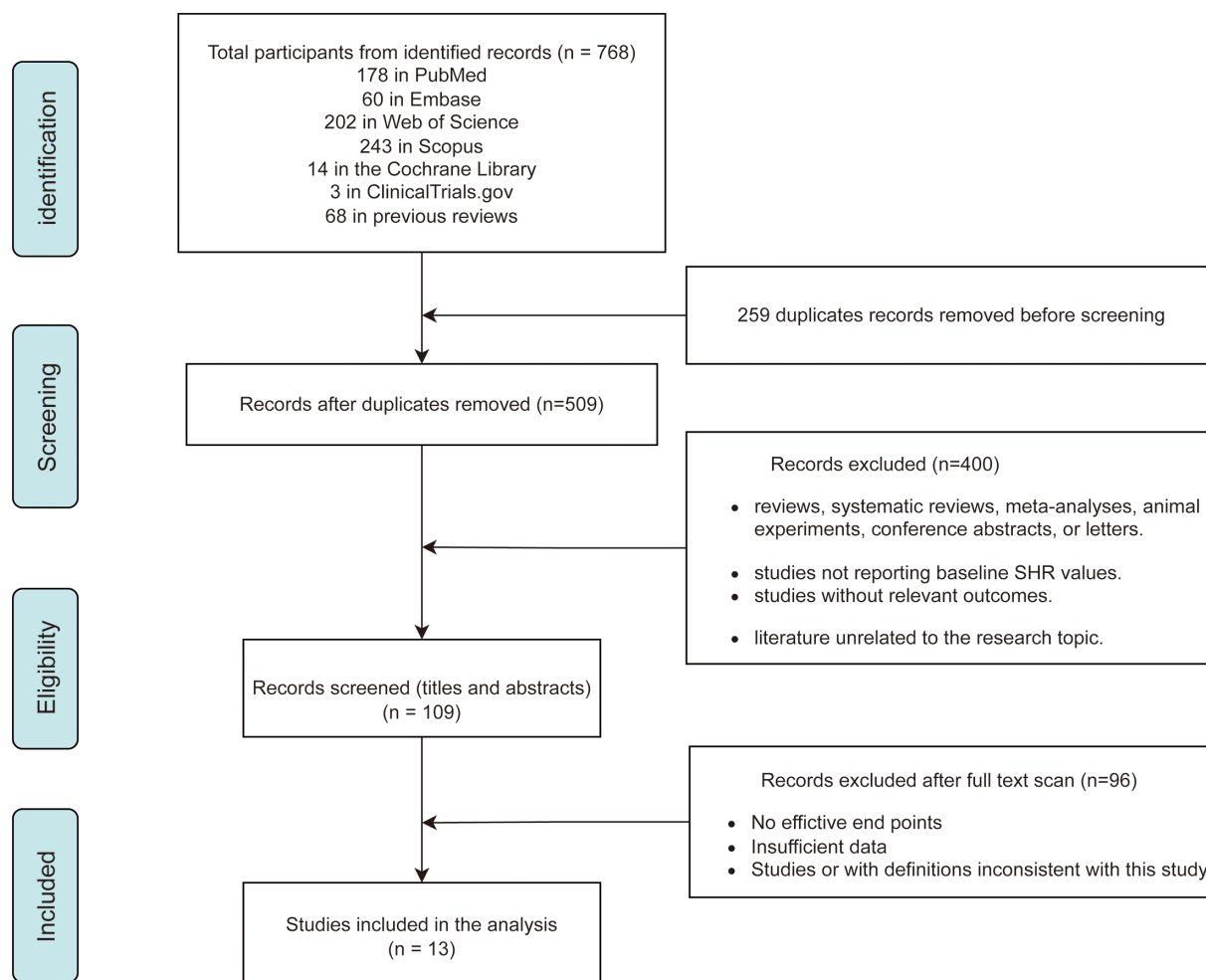


Figure 1 Study selection process.

to the limited number of included studies. Egger's and Begg's tests were additionally performed when applicable.

The certainty of evidence was graded as high, moderate, low, or very low using the GRADE framework. In addition to the I^2 statistic, assessment of inconsistency considered effect size consistency, CI overlap, number of studies, and clinical plausibility, rather than relying solely on I^2 thresholds. Summary of Findings tables were generated using GRADEpro software.

RESULTS

Figure 1 summarizes the study selection process. A total of 768 records were identified through database searches. After removal of duplicates, 259 records remained, of which 57 articles underwent full-text assessment. Two studies were excluded due to unclear SHR definitions¹⁵ or failure to meet prespecified criteria.¹⁶ Ultimately, 13 cohort studies^{17–29} were included in the final analysis.

The 13 studies involved 41 096 PCI patients: 10 studies^{17,21–29} used SHR1 (n=35 611), 2 studies^{23,24} used SHR2 (n=4386), and 1 study²⁰ reported both SHR1 and SHR2 (n=1099), classified as SHR1 for analysis. Eight studies reported all-cause mortality, nine reported MACCE, three reported cardiovascular mortality, four reported recurrent myocardial

infarction, three reported stroke, and three reported repeat revascularization. The brief **table 1** is as follows, and the full table is presented in the supplementary file (online supplemental table S5). Studies that originally reported MACE were included if their outcomes were consistent with the MACCE definition used in this meta-analysis.

Meta-analysis results

All-cause mortality

Eight studies (nine datasets),^{17–24} including 17 694 patients, were analyzed. Seven datasets used SHR1, and two used SHR2. Overall, higher SHR was associated with increased risk of all-cause mortality following PCI (RR=2.085, 95% CI 1.451 to 2.996; $p<0.001$), although substantial heterogeneity was observed ($I^2=92.2\%$) (**figure 2**). Subgroup analyses by SHR definition showed significant associations for both SHR1 (RR=1.984, 95% CI 1.304 to 3.018; $p=0.001$) and SHR2 (RR=2.432, 95% CI 1.648 to 3.587; $p<0.001$; P for interaction=0.492). Similar associations were observed in ACS-only cohorts (RR=2.077, 95% CI 1.302 to 3.313; $p=0.002$) and non-ACS/mixed populations (RR=2.126, 95% CI 1.095 to 4.127; $p=0.026$), with no evidence of significant interaction ($p=0.948$). Studies with 100% PCI populations showed a higher pooled effect estimate (RR=2.734, 95% CI 1.717 to

Table 1 Characteristics for included patients and relevant outcomes

Author (year)	Nation	Participants (n)	Age (years) (mean±SD)	Vascular lesion type	Proportion of ACS (%)	Proportion of index PCI (%)	Proportion of DM (%)	Outcomes
Jie Yang (2022) ²⁵	China	5562	58.0±10.1	ACS	100	100	39.3	②
Haozhang Huang (2024) ²⁶	China	874	64.1±10.8	CAD	37.1	100	39.6	②
Shiqiang Xiong (2017)	China	373	66.5±11.3	ACS	100	100	59.8	①②③ ④⑤⑥
Shogo Okita (2025) ¹⁸	Japan	2386	68.0±12.5	STEMI; NSTEMI	100	100	37.3	①② ④⑤
Ömer Furkan Demir (2024) ²⁷	Turkey	223	69.6±9.7	ACS	100	100	44.8	②
Zehuo Lin (2023) ²⁸	China	2841	62.3±12.2	STEMI	100	100	31.0	②
Yanjun Song (2023) ²³	China	2740	58.5±10.5	Angina patients with CTO	50.9	100	46.7	①②③
Yeoree Yang (2017) ¹⁹	Korea	4362	63.1±10.7	ACS; non-ACS	54	100	45.1	①②④ ⑤⑥
Qu-Cheng Wei (2023) ²⁰	China	1099	62.6±13.6	STEMI	100	100	27.1	①②⑥
Wang Liao (2023) ²⁴	China	1643	62.5±12.6	STEMI	100	100	33.8	①
Jin Liu (2023) ²¹	China	2171	62.9±12.1	AMI	100	89.3	42.1	①
Takayuki Ojima 1 (2018)	Japan	3882	65.9±12.6	STEMI	100	94.4	0	①
Takayuki Ojima 2 (2018)	Japan	2405	65.3±10.8	STEMI	100	93.4	100	①
Zheng Qiao (2024) ²⁹	China	10532	60.6±9.9	CTVD	63.6	100	38.5	③④

Note: ① all-cause mortality; ② major adverse cardiac and cerebrovascular events; ③ cardiovascular mortality; ④ recurrent myocardial infarction; ⑤ stroke; ⑥ repeat revascularization.
NSTEMI, Non-ST-segment elevation myocardial infarction; STEMI, ST-segment elevation myocardial infarction.

4.355; $p<0.001$) compared with studies including $<100\%$ PCI (RR=1.304, 95% CI 0.954 to 1.780; $p=0.096$; P for interaction=0.003). Excluding studies with extreme diabetes prevalence (0% or 100%) did not materially change the pooled estimate (RR=2.365, 95% CI 1.453 to 3.850; $p=0.001$) (figure 3 and online supplemental table S6.1).

Major adverse cardiac and cerebrovascular events

Nine studies,^{17–20 23 25–28} including 14564 patients, reported MACCE. Higher SHR was overall associated with an increased risk of MACCE (RR=1.902, 95% CI 1.406 to 2.574; $p<0.001$), with substantial heterogeneity ($I^2=89.2\%$) (figure 2). Subgroup analyses by clinical presentation showed significant associations in ACS-only (RR=2.073, 95% CI 1.310 to 3.279; $p=0.002$) and non-ACS/mixed populations (RR=1.683, 95% CI 1.145 to 2.474; $p=0.008$; P for interaction=0.552). Restricting to SHR1 studies produced similar

results (RR=1.810, 95% CI 1.314 to 2.494; $p<0.001$) (figure 3 and online supplemental table S6.1).

Cardiovascular mortality

Three studies^{17 23 29} (n=12734) reported cardiovascular mortality. Higher SHR was associated with increased risk of cardiovascular mortality (RR=2.414, 95% CI 1.409 to 4.136; $p=0.001$), with moderate heterogeneity ($I^2=42.9\%$) (figure 2). SHR1-only analyses were consistent (RR=1.904, 95% CI 1.340 to 2.706; $p<0.001$). Given the limited number of studies, these findings should be interpreted cautiously.

Recurrent myocardial infarction

Four studies,^{17–19 29} including 17653 patients, showed higher SHR increased risk of recurrent MI (RR=2.250, 95% CI 1.663 to 3.044; $p<0.001$), with no heterogeneity observed ($I^2=0\%$; $p=0.517$) (figure 2).

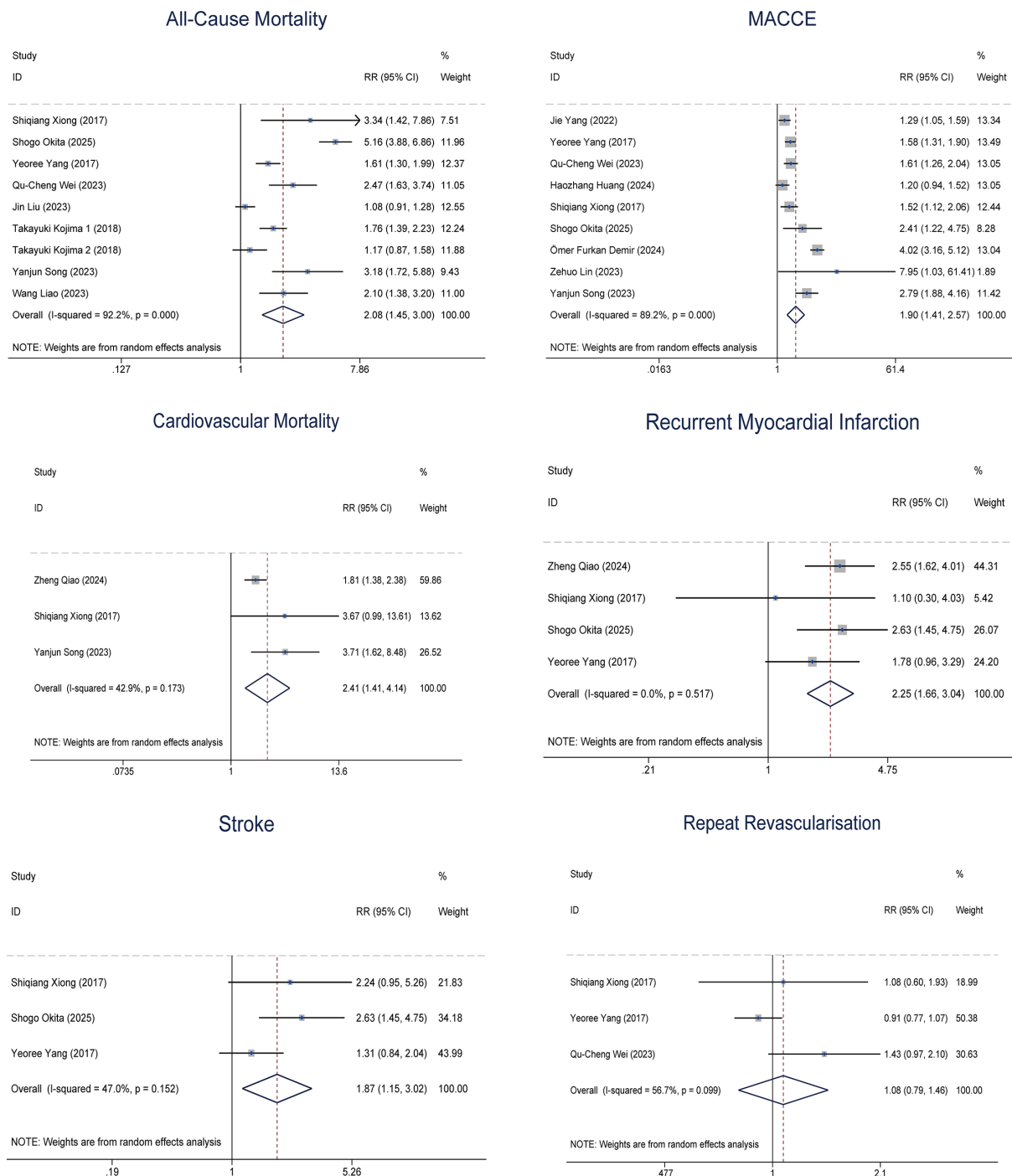


Figure 2 This figure presents the overall meta-analysis results of 13 included studies, evaluating the association between stress hyperglycemia ratio (SHR) and major adverse cardiac and cerebrovascular events (MACCEs), all-cause mortality, cardiovascular mortality, recurrent myocardial infarction, stroke, and repeat revascularization. Higher SHR was significantly associated with increased risks of MACCE and all-cause mortality, but not with repeat revascularization. The figure shows the weighted effect sizes and 95% CIs for each study. RR, relative risk.

Stroke

Three studies^{17–19} (n=7121) demonstrated increased stroke risk with higher SHR (RR=1.866, 95% CI 1.154 to 3.019; p=0.011), with moderate heterogeneity ($I^2=47.0\%$) (figure 2).

Repeat revascularization

Three studies^{17 19 20} (n=5284) showed no significant association (RR=1.077, 95% CI 0.791 to 1.465; p=0.639), with

moderate heterogeneity ($I^2=56.7\%$) (figure 2). These results should be interpreted cautiously due to the limited number of studies.

Dose-response relationship between SHR and clinical outcomes SHR and MACCE

Five studies^{24–28} evaluated the dose-response relationship between SHR and MACCE. In the primary analysis, which

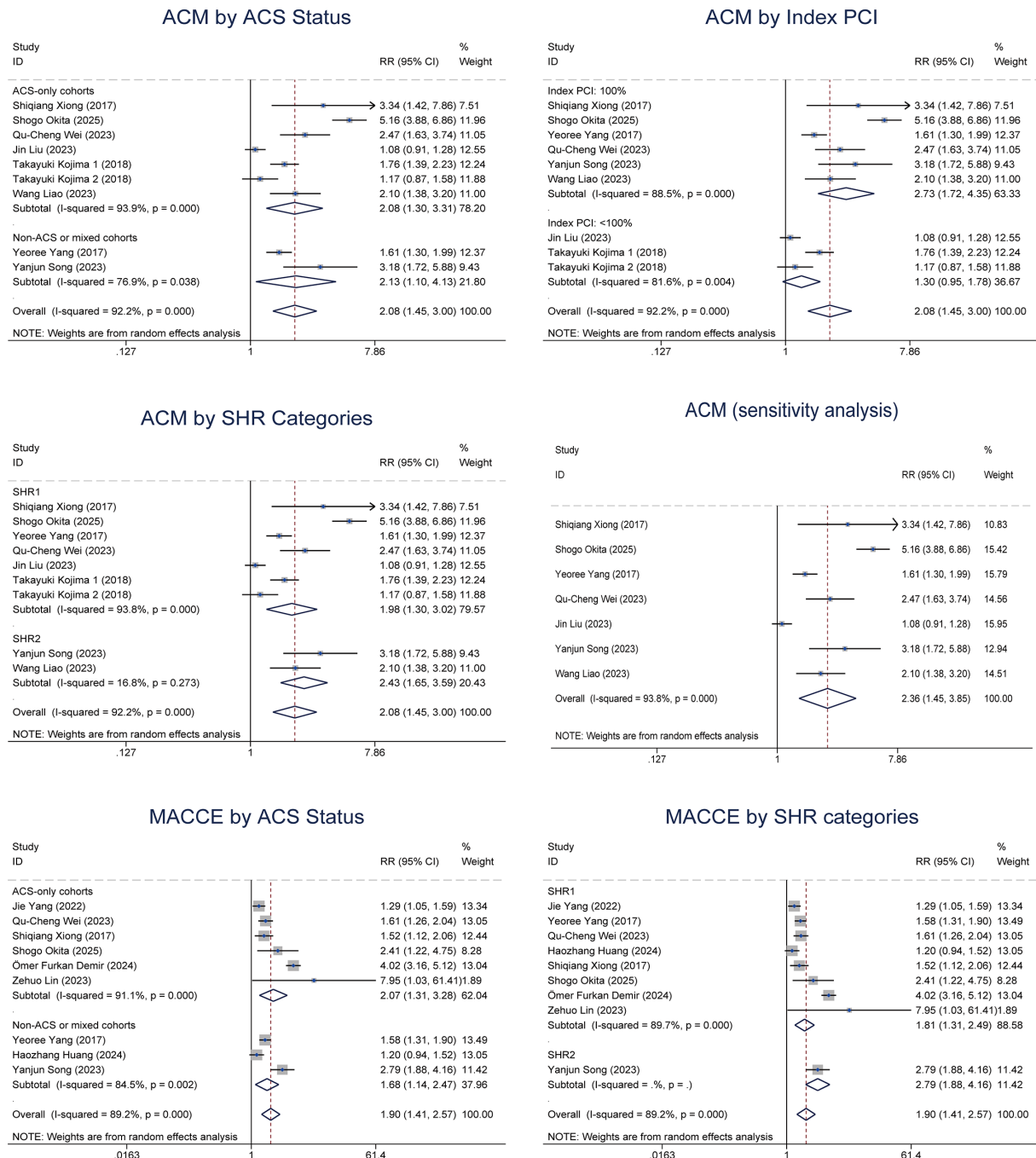


Figure 3 The subgroup analysis results for all-cause mortality (ACM) and major adverse cardiac and cerebrovascular events (MACCEs) across different clinical characteristics. The analysis includes the following: Subgroup analysis for all-cause mortality and MACCE, stratified by the proportion of patients with acute coronary syndrome (ACS) and the proportion of index percutaneous coronary intervention (PCI) patients. Index PCI refers to PCI performed during the index hospitalization/ presentation as reported in each study; sensitivity analysis of the association between stress hyperglycemia ratio (SHR) and all-cause mortality after excluding studies with extreme diabetes prevalence (0% or 100%); subgroup analysis based on SHR definition categories (SHR1 and SHR2).

included only three HR studies, no statistically significant evidence of non-linearity was observed (nonlinear test p value=0.586) (figure 4). A 0.1-unit increase in SHR was associated with a 17.4% increase in MACCE risk (HR=1.174; 95% CI 0.984 to 1.400; p=0.074), suggesting a potential approximately linear dose-response pattern. A sensitivity analysis incorporating all five studies (three

HR studies and two OR studies) yielded similar findings (non-linear test p value=0.602; HR=1.173; 95% CI 1.037 to 1.328; p=0.011). Alternative knot specifications showed similar patterns. Descriptive analyses of OR-based studies demonstrated a consistent directional association (online supplemental table S6.2 and online supplemental figures S1 and S2).

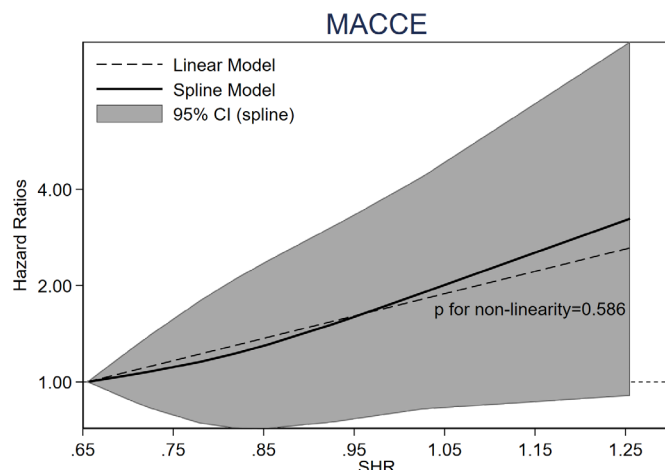


Figure 4 The figure shows the linear and spline models of the HR for major adverse cardiac and cerebrovascular events (MACCEs) across stress hyperglycemia ratio (SHR) values, based on three studies reporting HRs. The dashed line represents the linear model, which was used as the primary analysis. The solid line represents the spline model, and the shaded area indicates the 95% CI of the spline model. No significant evidence of non-linearity was observed (p for non-linearity=0.586), indicating a possible approximately linear association between SHR and risk of MACCE within the observed range of SHR values. HRs are plotted on the y-axis, and SHR values on the x-axis.

SHR and all-cause mortality

Only two HR studies^{21 23} were available for formal spline modeling; therefore, the dose-response analysis was primarily exploratory. An additional analysis including one OR study²⁴ suggested a potential approximately linear trend between SHR and all-cause mortality (p for non-linearity=0.142). Given the limited number of studies and approximate effect estimates, these findings should be interpreted cautiously (online supplemental table S6.2 and online supplemental figures S1 and S3).

Sensitivity analyses, publication bias, and meta-regression

Leave-one-out analyses (online supplemental figure S4) indicated that exclusion of any single study did not materially change the pooled effect estimates for MACCE, all-cause mortality, cardiovascular mortality, recurrent myocardial infarction, stroke, or repeat revascularization, suggesting that the overall results were stable.

Potential publication bias was assessed using funnel plots (online supplemental figure S5). Due to the limited number of studies for several outcomes, formal assessment of funnel plot asymmetry was limited; however, visual inspection did not suggest substantial asymmetry. Statistical tests, including Begg's and Egger's tests, did not indicate statistically significant publication bias for any of the analyzed outcomes (online supplemental table S7), although these findings should be interpreted cautiously given the small number of studies.

Study-level meta-regression was conducted to explore whether the proportion of patients with diabetes was associated with effect estimates. For MACCE, no evidence of

a significant association was observed (β =0.010 per 10% increase; 95% CI -0.355 to 0.376; p =0.955). Similarly, for all-cause mortality, no evidence of an association was identified (β =-0.047; 95% CI -0.185 to 0.091; p =0.505). Sensitivity analyses excluding studies with extreme diabetes prevalence yielded consistent results. Overall, no statistically significant association was observed between study-level diabetes prevalence and effect estimates for MACCE or all-cause mortality. However, given the limited number of included studies and the substantial between-study heterogeneity, these analyses were considered underpowered and exploratory. Therefore, the absence of statistical significance does not exclude potential effect modification at the individual patient level.

Certainty of evidence

According to the GRADE classification criteria and primarily based on the ROBINS-I assessment results, the certainty of evidence for the primary outcomes (all-cause mortality and MACCE) was rated as very low. For cardiovascular mortality, recurrent myocardial infarction, and stroke, the certainty of evidence was rated as low, whereas for repeat revascularization it was rated as very low (online supplemental table S8). Downgrading was mainly driven by residual confounding inherent in observational studies, heterogeneity in outcome definitions, and inconsistency due to substantial between-study variability. While the Newcastle-Ottawa Scale (NOS) was applied as a supplementary tool, most outcomes were rated as low to moderate quality, reflecting its less conservative nature. In contrast, ROBINS-I-based GRADE assessments were generally more stringent, particularly for MACCE and all-cause mortality, due to greater sensitivity to residual confounding and outcome heterogeneity (online supplemental tables S8-S12). Although a dose-response pattern was observed for all-cause mortality and MACCE, this should be interpreted cautiously given the limited number of studies and residual methodological limitations. Overall, the combined use of ROBINS-I, NOS, and GRADE provides a transparent and comprehensive assessment of risk of bias and certainty of evidence.

DISCUSSION

In this systematic review and meta-analysis, we evaluated the association between SHR and clinical outcomes in patients undergoing PCI. Elevated SHR was associated with increased risks of all-cause mortality and MACCE, as well as higher risks of cardiovascular mortality, recurrent myocardial infarction, and stroke, while no significant association was observed for repeat revascularization. Dose-response analyses using restricted cubic spline models suggested a potential linear relationship between SHR and adverse clinical outcomes, with no evidence of statistically significant non-linearity (P for non-linearity >0.05). In sensitivity analyses, a linear trend was observed in the overall pooled dataset. These findings suggest that SHR may have prognostic value as a continuous

biomarker for risk stratification in PCI patients, although the strength of evidence is limited by the observational nature of the included studies and the relatively small number of datasets.

We explored the potential mechanisms linking stress hyperglycemia to adverse cardiovascular outcomes. Stress hyperglycemia is commonly observed in coronary artery disease, particularly in ACS, and reflects neuroendocrine activation, systemic inflammation, and insulin resistance. Acute hyperglycemia activates nuclear factor kappa-light-chain-enhancer of activated B cells, increases reactive oxygen species production, and promotes inflammatory mediators, exacerbating vascular inflammation and accelerating atherosclerosis.^{30–34} High glucose levels also enhance oxidative stress, reduce nitric oxide bioavailability, and impair endothelial function, leading to compromised coronary perfusion and exacerbated reperfusion injury.³⁵ Additionally, hyperglycemia disrupts the coagulation-fibrinolysis balance, increasing thrombin generation and promoting a prothrombotic state.^{36–39} Collectively, these mechanisms may underpin the adverse prognostic implications of elevated SHR in PCI patients. PCI itself causes endothelial injury and amplifies inflammatory responses, promoting neointimal hyperplasia and thrombosis.^{3–4} This procedure-related inflammatory stress may contribute to adverse outcomes, particularly in patients with pre-existing vascular inflammation. Gach *et al* reported that elevated high-sensitivity C-reactive protein within 24 hours before and after PCI independently predicted short- and long-term MACE.⁴⁰ Moreover, heightened peri-procedural inflammation has been associated with increased risk of subsequent heart failure or heart failure-related hospitalization.⁴¹ These mechanisms provide biological plausibility for the prognostic utility of SHR, which integrates acute and chronic glycemic states, potentially reflecting relative risk more accurately than absolute glucose values.

Coronary artery disease and DM frequently coexist and are associated with more diffuse coronary lesions and poorer clinical outcomes. Although oxidative stress is more closely related to acute glucose fluctuations than sustained chronic hyperglycemia,³² whether the prognostic impact of stress hyperglycemia differs by diabetes status remains controversial. Previous evidence indicates that stress hyperglycemia predicts higher in-hospital mortality after myocardial infarction regardless of diabetes status, with additional risks of congestive heart failure or cardiogenic shock in patients without diabetes.⁴² Among patients with ST-segment elevation myocardial infarction undergoing PCI, admission hyperglycemia independently predicts early and late mortality in both patients with and without known diabetes.⁴³ Similarly, stress hyperglycemia has been identified as an independent marker of adverse outcomes irrespective of diabetes status.⁴⁴ These findings underscore the value of SHR as a refined metric for evaluating stress-related glycemic dysregulation.

Since stratified analyses by individual diabetes status were not feasible in most included studies, we performed study-level meta-regression using diabetes prevalence and sensitivity analyses excluding cohorts with extreme diabetes prevalence (0% or 100%). These analyses showed no evidence of effect modification by diabetes prevalence on the associations between SHR and MACCE or all-cause mortality (β close to 0, $p > 0.5$). While these findings should be interpreted cautiously due to limited statistical power, the SHR–outcome association appeared broadly consistent across populations with different diabetes prevalence levels. Importantly, all analyses were conducted at the study level and reflect ecological rather than individual-level associations; thus, the absence of significant moderation does not exclude potential effect modification at the patient level. Meta-regression results should not be interpreted as individual-level effects. Furthermore, the limited number of included studies and substantial residual heterogeneity may reduce statistical power to detect modest moderating effects. Further individual participant-level analyses are warranted.

Experimental and clinical evidence suggests that mild-to-moderate stress hyperglycemia under acute ischemic conditions may exert adaptive or protective effects, such as enhancing cellular energy supply or limiting apoptosis. In contrast, excessive hyperglycemia promotes oxidative stress, endothelial dysfunction, and a procoagulant state, exacerbating myocardial injury and impairing microvascular perfusion, particularly in patients with acute myocardial infarction.¹⁶ It is noteworthy that within the SHR framework adopted in this study, the observed linear relationship is limited to the range of SHR values included, and relatively lower SHR values may not necessarily indicate lower clinical risk. Low SHR could result from chronically elevated glycated hemoglobin (HbA1c) or relatively low blood glucose levels at admission; in the context of acute cardiovascular disease, such situations may reflect severe illness, hemodynamic instability, or multiorgan dysfunction rather than effective glycemic control.^{25–45–46} SHR may therefore reflect underlying disease burden or limited metabolic reserve, which could be associated with higher risk. These findings highlight the importance of careful glycemic management in PCI patients, although any suggestion of an optimal SHR range should be interpreted with caution. Based on prior literature reporting J-shaped or U-shaped relationships, extremely low SHR may be associated with higher risk under certain conditions^{25–47}; however, this was not directly supported by the present analysis and remains hypothesis-generating.

Previous studies primarily used admission or fasting glucose as indicators of stress hyperglycemia, with varying thresholds influenced by baseline glycemic status.^{42–43} Capes *et al* noted that admission hyperglycemia reflects disease severity and counter-regulatory responses rather than true stress-related glycemic deviation.⁴² SHR standardizes acute glucose levels to chronic glycemic exposure estimated by HbA1c, offering a more consistent

framework for assessing stress-related dysglycemia.⁶ Accumulating evidence indicates that SHR offers superior prognostic performance compared with absolute glucose levels for predicting adverse events, although potential modification by diabetes status remains debated.^{25 47 48}

Although diabetes is a well-recognized cardiometabolic risk factor, the interaction between acute stress-induced hyperglycemia and chronic glycemic status is complex. SHR reflects relative hyperglycemia by combining acute glucose levels with background glycemic control, and its predictive value may vary according to individual diabetes status.⁴⁹ For patients without diabetes, elevated SHR more clearly reflects an acute stress response, whereas in patients with diabetes, chronic hyperglycemia may attenuate relative changes in SHR and reduce its discriminative ability. Importantly, the present analyses were conducted at the study level and should be interpreted as ecological associations rather than individual-level effects. Thus, the absence of significant moderation by diabetes prevalence does not exclude potential differences at the patient level. Finally, the limited number of included studies and substantial residual heterogeneity may reduce statistical power to detect modest moderating effects. Further individual participant-level analyses are warranted to better characterize the interaction between SHR and diabetes status.

The substantial heterogeneity observed likely reflects differences in ACS versus non-ACS populations, PCI proportions, SHR thresholds, covariate adjustments, and follow-up durations. Subgroup analyses for ACS versus non-ACS populations showed consistently elevated risk associated with higher SHR, with no significant interaction between groups (P for interaction=0.948). In patients with ACS, elevated SHR may reflect heightened neurohormonal activation and inflammatory burden during acute ischemic events, which could exacerbate myocardial injury, impair microvascular perfusion, and increase susceptibility to thrombotic complications.⁵⁰ Subgroup analyses by PCI proportion indicated a stronger association in studies with 100% PCI compared with <100% PCI (P for interaction=0.003), which may partly explain heterogeneity across studies. Funnel plot asymmetry was limited by the small number of studies, but visual inspection did not suggest substantial publication bias, and statistical tests did not indicate significant bias for primary outcomes.

Strengths and limitations

All included studies were observational, and residual confounding cannot be fully excluded despite the use of ROBINS-I. Substantial heterogeneity was observed in MACCE and all-cause mortality analyses. Although subgroup, sensitivity, and dose-response analyses showed generally consistent patterns, the sources of heterogeneity were not fully explained. The limited number of studies precluded robust reconstruction of dose-response relationships for certain outcomes. In addition, analyses were restricted to the observed range of SHR

values; extrapolation beyond this range is not supported. Overall, the dose-response findings should be considered exploratory and hypothesis-generating.

Strengths of the study include the integration of data from multiple cohorts and the use of dose-response meta-analysis, providing a comprehensive overview of the association between SHR and adverse outcomes in PCI patients. Consistency of results across SHR definitions was observed. Subgroup, sensitivity, and meta-regression analyses across clinically relevant categories further supported the stability of the findings. Additionally, the use of ROBINS-I and GRADE frameworks enhanced transparency in assessing risk of bias and certainty of evidence.

CONCLUSION

In patients undergoing PCI, elevated preoperative SHR was associated with an increased risk of adverse outcomes. Dose-response analysis suggested a possible approximately linear association within the observed range of SHR values. SHR may serve as a practical marker for post-PCI risk stratification; however, prospective studies are required to determine clinically relevant thresholds and validate its clinical utility.

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