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Effect of diltiazem sustained-release capsules on cardiorenal composite outcomes in hypertensive patients with coronary heart disease: A real-world propensity score-matched study

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Abstract: **Background**: Hypertension frequently coexists with coronary heart disease (CHD) and integrated management is essential to reduce cardiovascular and renal complications. **Objectives**: To evaluate the efficacy and safety of add-on sustained-release diltiazem capsules in patients with hypertension and CHD. **Methods**: This retrospective cohort analysis included 302 consecutive patients with CHD treated at the Affiliated Hospital of Jiangnan University between May 2021 and May 2024. Patients were divided into a control group (n=158) receiving standard guideline-directed therapy and a diltiazem group (n=144) receiving add-on diltiazem sustained-release capsules. Propensity scores were used to match and balance baseline covariates to reduce selection bias. Following matching, intergroup comparisons for baseline characteristics, occurrence of cardiorenal composite endpoints, blood pressure, heart rate (HR) control during follow-up and adverse drug reactions were conducted. Multivariate regression analysis was used to determine predictors of cardiorenal endpoint events. **Results**: After matching, baseline covariates were well-balanced between groups. During a median follow-up of 18 months, the cumulative incidence of cardiorenal composite endpoint events was lower in the diltiazem group than in controls (16.39% vs. 32.79%, P=0.002). Multivariate Cox analysis showed that diltiazem therapy was independently associated with a lower risk of composite outcomes (hazard ratio = 0.465, 95% CI: 0.284–0.760, P = 0.002). Patients receiving diltiazem also had a lower mean HR and higher achievement rates for HR control and combined blood pressure/HR targets (all P<0.05). The reduction in composite outcomes was primarily driven by fewer heart failure rehospitalizations. Drug-related adverse events were comparable between groups. **Conclusion**: Adding sustained-release diltiazem capsules to standard therapy is associated with a lower cardiorenal composite endpoint risk in patients with hypertension and CHD. The observed benefit was mainly driven by fewer heart failure rehospitalizations, alongwith improved control of blood pressure and heart rate. The renal findings should be considered exploratory because the number of renal events was limited.

Keywords: Coronary heart disease; Cardiorenal composite endpoints; Diltiazem sustained-release capsules; Hypertension; Propensity score matching; Real-world evidence

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INTRODUCTION

Hypertension is a core driving factor in the development and progression of atherosclerosis. Persistently elevated blood pressure (BP) accelerates the process of coronary atherosclerosis through multiple mechanisms, such as hemodynamic stress, endothelial dysfunction, oxidative stress and inflammatory responses, ultimately leading to clinical events, including myocardial ischemia, angina pectoris and myocardial infarction (Zheng *et al*., 2020; Satoh *et al*., 2025). Coronary heart disease (CHD) is a common cause of organ damage caused by hypertension. When combined with hypertension, it significantly increases the risk of cardiac and renal complications and seriously damages the physical health of patients (Wenjie, 2025; Dobosz *et al*., 2025). An ideal antihypertensive drug should stably and effectively reduce BP, improve coronary blood flow, reduce myocardial oxygen consumption, stabilize atherosclerotic plaques and potentially protect important target organs (Ajimu *et al*., 2023; Zou *et al*., 2025). As a non-dihydropyridine calcium channel blocker, diltiazem exerts effects including coronary vasodilation, improvement of myocardial blood supply, lowering of BP and protection of vascular endothelial function and is widely used to treat hypertension and CHD (Xue *et al*., 2020; Martins de Carvalho *et al*., 2022).

Although diltiazem has been widely used for the management of hypertension and angina, the available evidence mainly focuses on BP reduction, symptom relief and short-term anti-anginal efficacy. Data on its long-term impact on integrated cardiorenal outcomes in patients with concomitant hypertension and CHD remain limited, particularly in the real-world setting. Thus, this study conducted this retrospective propensity score-matched cohort study to evaluate whether add-on sustained-release diltiazem capsules provide prognostic benefits beyond those of standard therapy. Currently, systematic real-world research is lacking on the impact of diltiazem sustained-release capsules on cardiorenal endpoint events in patients with hypertension complicated by CHD. Based on real-world data, this study explored the effects of sustained-release diltiazem capsules on cardiorenal endpoints in patients with hypertension complicated by CHD, providing a rationale for optimizing clinical treatment strategies.

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MATERIALS AND METHODS

Study participants

The clinical medical records of 302 inpatients and outpatients with hypertension complicated by CHD who received treatment at the Department of Cardiology of the Affiliated Hospital of Jiangnan University from May 2021 to May 2024 were consecutively retrieved.

The inclusion criteria were as follows: (1) age 18–85 years; (2) met the diagnostic criteria for essential hypertension: systolic BP (SBP) ≥140 mmHg and/or diastolic BP (DBP) ≥90 mmHg, or receiving antihypertensive treatment; (3) confirmed diagnosis of CHD by coronary angiography or computed tomography angiography (CTA), with stenosis ≥50% in at least one major coronary artery; (4) estimated survival time >1 year; (5) complete clinical medical records; and (6) informed consent of patients and their family members for the treatment regimen.

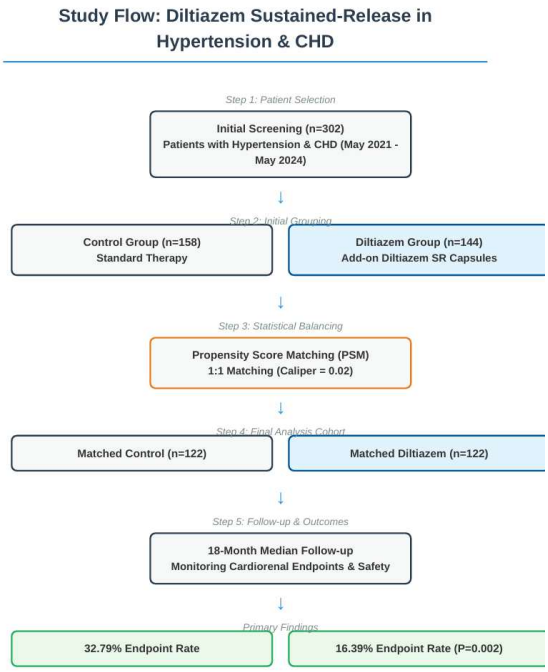

Fig. 1: Flow diagram of patients.

The following patients were excluded: (1) patients with secondary hypertension; (2) patients within 4 weeks of onset of acute myocardial infarction (AMI); (3) patients with hypersensitivity to diltiazem; (4) patients with NYHA cardiac function grade IV or left ventricular ejection fraction (LVEF) <35%; (5) patients with sick sinus syndrome, atrioventricular block of grade II or higher without pacemaker implantation, or baseline heart rate (HR) <50 beats per minute (bpm); (6) patients with severe hepatic insufficiency or active liver disease; (7) patients with estimated glomerular filtration rate (eGFR) <15 mL/min/1.73 m² or on renal replacement therapy; and (8) pregnant or lactating women. This study was approved by the hospital's ethics committee (approval no. LS2024260).

Study methods

All patients were categorized into two groups: a control group receiving standard guideline-directed therapy (n=158) and a diltiazem group receiving standard guideline-directed therapy combined with diltiazem sustained-release capsules. Treatment regimens were determined independently by attending physicians based on individual patient conditions, relevant clinical guidelines and clinical judgment; no treatment decisions were influenced by the study protocol.

To reduce potential bias from baseline imbalances, the nearest neighbor matching algorithm was used to perform 1:1 propensity score matching and the caliper value was 0.02. The matching model included the following covariates: age, sex, body mass index (BMI), baseline SBP, baseline HR, history of diabetes, chronic kidney disease (CKD) stage, number of vessels with coronary artery stenosis, baseline LVEF and baseline eGFR. Patients in the control group received standard pharmacotherapy according to current guideline recommendations, consisting of (1) antiplatelet therapy with aspirin (75–100 mg/day) and/or a P2Y12 inhibitor (clopidogrel 75 mg/day or ticagrelor 90 mg twice daily); (2) lipid-lowering and plaque-stabilizing therapy with high-intensity statins (atorvastatin 20–40 mg/day or rosuvastatin 10–20 mg/day); and (3) antihypertensive and anti-ischemic therapy with one or more agents. The diltiazem group received sustained-release diltiazem capsules in addition to the same standard therapy as the control group. The dosage was determined by the attending physician based on individual clinical conditions, typically initiated at 90 mg once or twice daily and titrated according to BP, HR response and tolerability, with a total daily dose ranging from 90 to 360 mg. Clinical considerations for adding diltiazem: (1) persistent sinus tachycardia (resting HR >80 bpm) with intolerance, contraindication to β-blockers, or failure to achieve HR target despite adequate β-blocker therapy; (2) clinically suspected or confirmed vasospastic angina component; (3) suboptimal BP control with other antihypertensive regimens, especially the need for stable control of morning BP surge or nocturnal BP; (4) history of supraventricular tachycardia requiring long-term prophylaxis.

For patients receiving concomitant β-blockers, diltiazem was generally initiated at a low dose with close monitoring of HR, BP and electrocardiographic conduction. Dose reduction of β-blockers or diltiazem was considered if resting HR was <55 beats/min, symptomatic bradycardia occurred, or atrioventricular conduction abnormalities were detected.

Baseline characteristics, treatment regimens and follow-up data were collected from hospital electronic records, outpatient medical records and telephone interviews, with the last follow-up conducted on May 31, 2025. The collected data included: (1) demographic information such as age and sex; (2) anthropometric indicators such as height, weight and BMI; (3) behavioral factors: smoking and alcohol use history; (4) comorbidities: diabetes mellitus, hyperlipidemia, stroke or transient ischemic attack and CKD stage; (5) medications: drug name, dosage and frequency; (6) vital signs: sitting BP and resting HR measured in the office at the time of first registration; (7) laboratory parameters: serum creatinine (for eGFR calculation), lipid profile, fasting glucose, glycated hemoglobin and N-terminal pro-B-type natriuretic peptide levels; (8) imaging indicators: LVEF and left ventricular mass index measured by echocardiography, the number of diseased vessels reported by coronary angiography/CTA and the Gensini score; (9) follow-up data: records of patients during regular follow-up or rehospitalization for any reason.

The composite cardiorenal endpoint was prespecified to capture major clinically meaningful cardiovascular and renal events that share common pathophysiological pathways, including vascular dysfunction, hemodynamic stress and progressive end-organ damage. Cardiorenal endpoint events included (1) cardiac death; (2) nonfatal stroke; (3) nonfatal myocardial infarction; (4) rehospitalization for unstable angina or exacerbation of acute heart failure; (5) ≥40% decrease in eGFR from baseline; (6) initiation of long-term renal replacement therapy (dialysis or kidney transplantation).

Observation indicators

Differences in post-propensity score matching (PSM) baseline characteristics, incidence of cardiorenal endpoint events, BP and HR control, target achievement during follow-up and drug-related adverse events were compared between the two groups. The factors influencing cardiac and renal endpoint events were analyzed.

The following criteria were assessed: (1) office BP <140/90 mmHg; (2) achieved HR control: resting HR 60–70 bpm; (3) severe bradycardia: HR <45 bpm.

Statistical methods

The data were analyzed using SPSS software (v.26.0). Continuous variables with normal distribution are expressed by mean ± standard deviation ($\bar{x} \pm s$) and were compared by independent sample *t* test. Non-normally distributed data are expressed as median (interquartile range) [M(IQR)] and were analyzed using the Mann–Whitney U test. Categorical variables are represented as numbers (percentages) [n (%)] and were compared using the chi-square (χ^2) test or Fisher's exact test, depending on the situation. Multivariate Cox proportional hazard regression was performed to evaluate the correlation with the clinical results and the results were reported as hazard ratios (HRs) with 95% confidence intervals (CIs). Variables entered into multivariable Cox regression were selected based on clinical relevance, prior literature and univariable association (P <0.10), including age, diabetes, baseline eGFR, SBP, β-blocker use and coronary disease severity. Two-tailed P value <0.05 indicates statistical significance. Covariate balance after matching was assessed using standardized mean differences (SMD), with SMD <0.10 considered acceptable balance. The composite endpoint was prespecified to capture the shared cardiovascular/renal risk continuum commonly observed in patients with hypertension and CHD.

RESULTS

Baseline data after PSM

After PSM, 122 matched pairs were identified. The baseline characteristics were well-balanced between the two groups. All post-matching SMD were <0.10, indicating an adequate covariate balance (Table 1 and Fig. 1).

Comparison of the incidence of cardiorenal endpoint events between the two groups after PSM

The median follow-up duration was 18 months (interquartile range: 12–26 months). During the follow-up, 60 patients (24.59%) in the matched cohort experienced at least one cardiorenal composite endpoint event. However, the cumulative incidence was significantly lower in the diltiazem group than in the control group (16.39% vs. 32.79%, P=0.002). Kaplan–Meier analysis also demonstrated a significantly lower time-to-first composite event rate in the diltiazem group (Fig. 2 and Table 2). Among the individual endpoint components, the greatest absolute reduction was observed in the heart failure rehospitalization rate (14 vs. 4 events), followed by unstable angina (10 vs. 6 events), suggesting that the overall composite benefit was driven primarily by reduced recurrent hospitalizations.

Cox multivariate regression analysis of cardiorenal endpoint events

Diabetes history, baseline eGFR and diltiazem therapy emerged as independent protective factors against composite outcomes after adjusting for age (hazard ratio = 0.465, 95% CI: 0.284–0.760, P=0.002) (Table 3).

Blood pressure and HR control and target achievement between the two groups during follow-up after PSM

Patients in the diltiazem group maintained a low average HR and demonstrated significantly higher rates of target HR achievement and combined BP and HR control during follow-up (P<0.05) (Table 4).

Safety analysis

The incidence of drug-related adverse reactions was compared between the two groups after matching (P>0.05) (Table 5).

Table 1: Baseline data after PSM.

Variable	Control group (n = 122)	Diltiazem group (n = 122)	t/χ^2	P value
Age (case)	65.3 ± 8.9	64.8 ± 8.7	0.462	0.644
Male	82 (67.21)	84 (68.85)	0.079	0.778
BMI	25.2 ± 3.0	25.3 ± 3.2	−0.258	0.797
Smoking, n (%)	43 (35.25)	41 (33.61)	0.077	0.781
Comorbidities, n (%)				
Diabetes mellitus	51 (41.80)	48 (39.34)	0.159	0.690
Hyperlipidemia	103 (84.43)	105 (86.07)	0.141	0.707
History of stroke	16 (13.11)	15 (12.30)	0.042	0.838
CKD stage (1–3a / 3b–4), n (%)	114/8 (93.44/6.56)	116/6 (95.08/4.92)	0.309	0.578
Vital signs				
SBP(mmHg)	150.2 ± 16.0	150.8 ± 15.5	−0.312	0.755
DBP(mmHg)	89.1 ± 10.9	89.6 ± 10.5	−0.369	0.713
HR (beats/min)	80.1 ± 11.9	80.8 ± 11.5	−0.483	0.630
Coronary artery disease, n (%)			0.022	0.882
Single-vessel disease	37 (30.33)	36 (29.51)		
Two-vessel disease	49 (40.16)	51 (41.80)		
Multivessel disease	36 (29.51)	35 (28.69)		
LDL-C (mmol/L)	2.40 ± 0.83	2.39 ± 0.80	0.098	0.922
eGFR (mL/min/1.73 m²)	78.2 ± 17.8	79.6 ± 17.1	−0.658	0.511
NT-proBNP (pg/mL), median (IQR)	300 (165–590)	315 (172–605)	Z = −0.453	0.651
LVEF (%)	58.0 ± 7.9	57.9 ± 8.0	0.102	0.919
Baseline medications, n (%)				
ACEI/ARB	112 (91.80)	112 (91.80)	—	1.000
β-blockers	92 (75.41)	90 (73.77)	0.091	0.763
Dihydropyridine CCBs	45 (36.89)	38 (31.15)	0.942	0.332
Diuretics	32 (26.23)	31 (25.41)	0.022	0.882
Statins	122 (100.00)	122 (100.00)	—	1.000
Antiplatelet agents	122 (100.00)	122 (100.00)	—	1.000

Note: Data are presented as mean ± SD, median (IQR), or n (%). BMI, body mass index; CKD, chronic kidney disease; LDL-C, low-density lipoprotein cholesterol; eGFR, estimated glomerular filtration rate; NT-proBNP, N-terminal pro-B-type natriuretic peptide; LVEF, left ventricular ejection fraction; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CCB, calcium channel blocker.

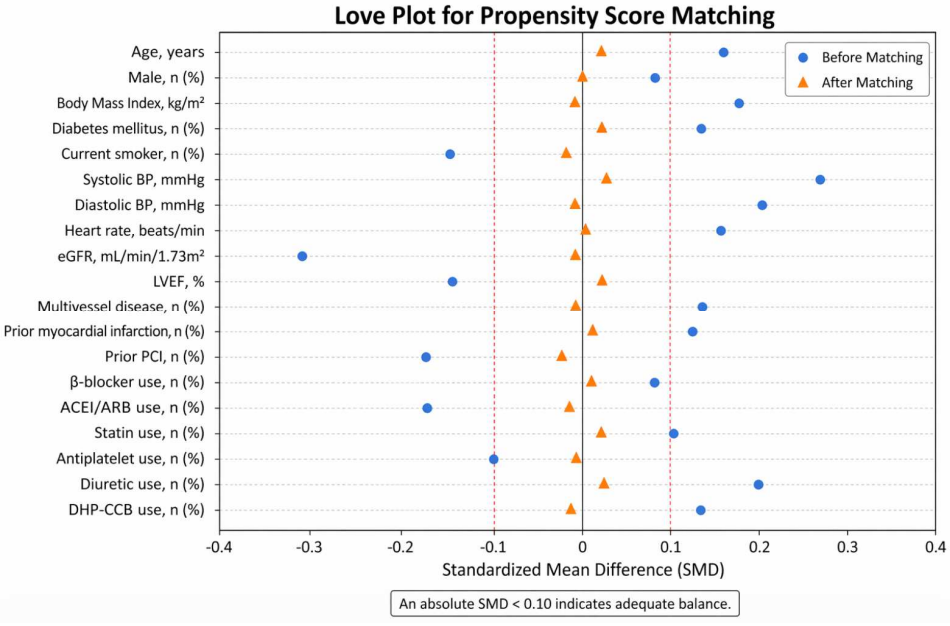

Fig. 2: Love plot showing standardized mean differences before and after propensity score matching.

Table 2: Cardiovascular and renal endpoint events between the two patient groups after PSM (case,n).

Group	Cardiovascular death (n, %)	Nonfatal myocardial infarction (n, %)	Nonfatal stroke (n, %)	Rehospitalization for unstable angina (n, %)	Rehospitalization for heart failure (n, %)	Renal events (n,%)	Total events, n (%)
Control group (n = 122)	4 (3.28)	7 (5.74)	6 (4.92)	10 (8.20)	14 (11.48)	5 (4.10)	40 (32.79)
Diltiazem group (n = 122)	2 (1.64)	5 (4.10)	4 (3.28)	6 (4.92)	4 (3.28)	3 (2.46)	20 (16.39)
χ^2 value							9.452
P value							0.002

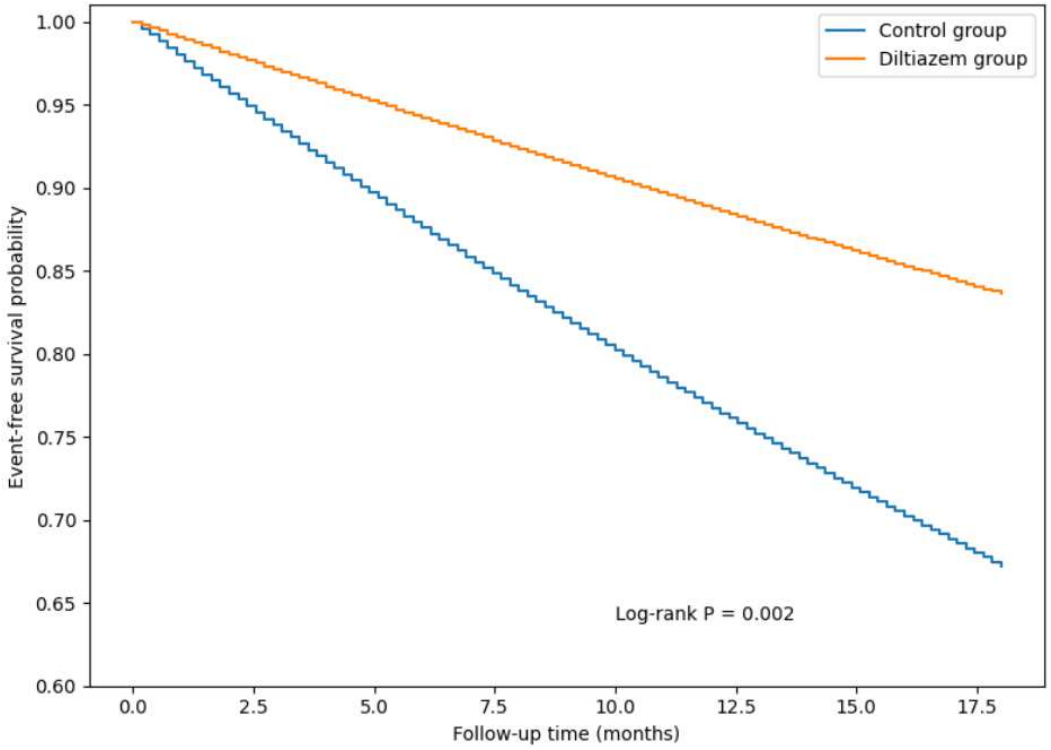

Fig. 3: Kaplan–Meier curve.

Table 3: Cox multivariate regression analysis of cardiovascular and renal endpoint events (PSM cohort).

Variable	B	SE	Wald χ^2	P	hazard ratio	95% CI
Age (per year of increase)	0.038	0.016	5.625	0.018	1.039	1.007–1.072
History of diabetes mellitus (present vs absent)	0.612	0.260	5.537	0.019	1.844	1.107–3.070
Baseline eGFR (per 1 unit increase)	−0.021	0.008	6.890	0.009	0.979	0.964–0.994
Treatment grouping (diltiazem vs. control group)	−0.764	0.246	9.639	0.002	0.465	0.284–0.760

Table 4: Blood Pressure and HR Control and target achievement between the two patient groups during follow-up after PSM

Variable	Control group (n = 122)	Diltiazem group (n = 122)	Statistic	P value
SBP (mmHg)	132.5 ± 10.8	130.8 ± 9.6	1.330	0.185
DBP (mmHg)	79.2 ± 7.5	78.6 ± 6.9	0.668	0.505
HR (beats/min)	72.4 ± 9.3	67.8 ± 8.1	4.222	<0.001
Blood pressure control rate, n (%)	98 (80.33)	105 (86.07)	1.521	0.217
HR control rate, n (%)	51 (41.80)	78 (63.93)	12.956	<0.001
Combined blood pressure and HR control rate, n (%)	42 (34.43)	68 (55.74)	11.754	0.001

Table 5: Drug-related adverse reactions between the two patient groups after PSM (case, n).

Group	Severe bradycardia (n, %)	Second–third degree atrioventricular block (n, %)	Peripheral edema (n, %)	Headache (n, %)	Constipation (n, %)	Drug discontinuation due to adverse events (n, %)	Total events, n (%)
Control group (n = 122)	1 (0.82)	0 (0.00)	8 (6.56)	5 (4.10)	15 (12.30)	4 (3.28)	33 (27.05)
Diltiazem group (n = 122)	3 (2.46)	2 (1.64)	11 (9.02)	3 (2.46)	11 (9.02)	6 (4.92)	36 (29.51)
χ^2 value	0.245	1.333	0.485	0.125	0.689	0.106	0.174
P value	0.622	0.248	0.486	0.723	0.408	0.745	0.677

DISCUSSION

Long-term clinical practice has shown that hypertension and CHD often coexist and exacerbate each other, significantly increasing the risk of cardiovascular and cerebrovascular events and target organ damage. For such patients, comprehensive management of BP and HR is needed to improve patient prognosis to the greatest extent. At present, although the comprehensive management strategies recommended by clinical guidelines cover multiple interventions, such as antihypertensive, antiplatelet and lipid-lowering therapies, some patients still have suboptimal BP and HR control or recurrent myocardial ischemia, necessitating more individualized and multi-targeted pharmacotherapeutic strategies (Chen *et al*., 2022; Zafrir *et al*., 2022).

This study used real-world data and PSM to effectively balance baseline characteristics, including age, sex, comorbidities, severity of coronary artery lesions, baseline cardiac and renal function indicators and baseline medication status between the control and diltiazem groups. This ensured good comparability of key clinical variables between the two groups, thus providing a more reliable basis for evaluating the efficacy and safety of diltiazem.

After PSM, the incidence of composite endpoint events in the diltiazem group (16.39%) was significantly lower than that in the control group (32.79%; $P<0.05$). Diabetes history, baseline eGFR and diltiazem therapy emerged as independent protective factors against composite outcomes after adjusting for age. These findings suggest that the addition of sustained-release diltiazem capsules to standard therapies, including routine antihypertensive, antiplatelet and lipid-lowering treatments, can further lower the risk of cardiovascular and renal events in patients with hypertension complicated by CHD. As a non-dihydropyridine calcium channel blocker (CCB), diltiazem selectively acts on L-type calcium channels in the vascular smooth muscle and myocardial cells. By inhibiting calcium ion influx, it dilates peripheral blood vessels to lower BP and dilates coronary arteries to increase myocardial blood supply and relieve myocardial ischemia, which is crucial for stabilizing plaques and reducing ischemic events such as myocardial infarction in patients with CHD (Chang and Ding, 2021; Xueying *et al*., 2024). Diltiazem can also inhibit calcium ion influx in the sinoatrial and atrioventricular nodes, reduce sinoatrial node automaticity and slow atrioventricular conduction, thereby decreasing the HR, reducing myocardial oxygen consumption and improving the balance between the supply and demand of myocardial oxygen (Tao *et al*., 2023; Zhu *et al*., 2023). In particular, the sustained-release formulation achieves a relatively stable blood drug concentration within 24 h through optimized release kinetics, which helps to reduce fluctuations in BP and HR and improves treatment compliance and persistence (Pedersen *et al*., 2022; Xin *et al*., 2023), thus exerting a unique therapeutic effect in patients with hypertension complicated by CHD.

In addition, patients in the diltiazem group maintained a low average HR and demonstrated significantly higher rates of target HR achievement and combined BP–HR control during follow-up ($P<0.05$). Effective HR control is an important factor for improving the prognosis of patients with CHD. An excessively fast HR increases myocardial oxygen consumption and exacerbates vascular endothelial damage and the progression of atherosclerosis (Peng *et al*., 2021; Li *et al*., 2025). By inhibiting sinoatrial node automaticity, diltiazem can effectively reduce resting and exercise HR and maintain it within an ideal range, thereby reducing myocardial oxygen consumption and mitigating myocardial ischemia, thereby directly or indirectly reducing triggers of cardiovascular and cerebrovascular events. Especially for patients who cannot tolerate β-blockers because of airway hyperreactivity, the risk of severe bradyarrhythmia, peripheral vascular disease or other conditions, diltiazem is an effective alternative or supplementary treatment option, providing more flexible choices for clinical treatment (Hou *et al*., 2024). The advantage of the diltiazem group in HR control further supports its value in the comprehensive treatment of hypertension complicated by CHD, particularly in improving HR and the rates of achieving combined BP and HR targets, thereby laying an

important foundation for reducing the risk of cardiorenal endpoint events. This is consistent with the conclusions of previous studies (Chen *et al*., 2020; Kook *et al*., 2020) and others on the importance of HR control for the prognosis of patients with CHD complicated by hypertension.

The study also found no significant between-group differences in drug-related adverse events after matching. Although diltiazem may theoretically increase the risk of bradyarrhythmia or atrioventricular conduction delay, the incidence of severe bradycardia and advanced atrioventricular block remains low. Furthermore, treatment discontinuation owing to adverse events was uncommon and comparable between the groups. These findings suggest that add-on diltiazem sustained-release therapy is generally well tolerated when dose titration and routine monitoring are used in clinical practice. Thus, the combination of a sustained-release diltiazem formulation with standard therapy does not significantly increase the risk of adverse events, such as severe bradycardia, grade II–III atrioventricular block, peripheral edema, headache and constipation and the discontinuation rate because of adverse reactions was comparable with that of the control group. Therefore, in clinical practice, physicians can achieve a good balance between effective treatment and safety by individually adjusting the dose based on the HR, tolerability and other factors of the patient.

The limitations of this study include its retrospective single-center design, moderate sample size and nonrandomized treatment allocation. Although PSM was used to reduce baseline imbalance, residual confounding from unmeasured variables could not be fully ruled out. In addition, treatment selection and dose adjustment were physician-directed, which may have introduced a practice-pattern bias. Nonetheless, further multicenter prospective randomized studies are warranted to validate these findings.

CONCLUSION

In this real-world propensity score-matched cohort, add-on diltiazem sustained-release capsules were associated with a lower incidence of cardiorenal composite outcomes in patients with hypertension and CHD. The observed benefits appeared to be mainly driven by fewer rehospitalizations for heart failure, together with improved HR control. Because the number of renal events was limited and the study was retrospective, these findings should be considered hypothesis-generating and require confirmation in prospective multicenter studies.

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None.

Authors' contributions
Guang Yan Wang: Responsible for research design, data collection and statistical analysis, paper writing and revision; Jing Jing Shi, Jie Yin Liu and Yu Ting Zhai: Participated in data organization, literature review and proofreading; Heng Su: Provided overall research guidance, supervised the entire research process, conducted critical review and final revision of the manuscript. All authors read and approved the final manuscript.

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Data availability statement
The datasets generated and/or analyzed in the current study are available from the corresponding author upon reasonable request.

Ethical approval
All procedures involving human participants were performed in accordance with the standards of the Ethics Committee of the Affiliated Hospital of Jiangnan University (Approval no. LS2024260). Written informed consent was obtained from all participants prior to enrollment. This study was performed in adherence with the STROBE guidelines. See supplementary file for the STROBE checklist.

Conflict of interest
All authors declare no conflict of interest.

Supplementary data
https://www.pjps.pk/uploads/2026/06/SUP1781607230.pdf

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