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Original Article

The effect of sacubitril/valsartan sodium on cardiac function and ventricular remodeling in patients with heart failure after PCI for acute myocardial infarction

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Abstract: Objective: To evaluate the effect of sacubitril/valsartan sodium on patients with heart failure following percutaneous coronary intervention (PCI) for acute myocardial infarction (AMI), with a focus on changes in cardiac function and vascular endothelial function. Methods: This retrospective study analyzed data from 108 patients diagnosed with AMI and heart failure after PCI between June 2023 and June 2024. Patients were allocated into two groups based on the treatment regimen. The control group received standard therapy supplemented with valsartan. The study group received sacubitril/valsartan sodium in place of valsartan, in addition to the same standard regimen. Key outcome measures included markers of myocardial injury [cardiac troponin I (cTnI), high-sensitivity C-reactive protein (hs-CRP), and N-terminal pro-brain natriuretic peptide (NT-proBNP)], echocardiographic parameters, indices of cardiac function and ventricular remodeling, six-minute walk distance (6MWD), Kansas City Cardiomyopathy Questionnaire (KCCQ) scores, major adverse cardiovascular events (MACEs), and incidence of adverse reactions. Results: Baseline characteristics were comparable between the two groups. Post-treatment, the study group demonstrated a significantly higher clinical efficacy ($P < 0.05$). Myocardial injury markers decreased more markedly, and improvements in cardiac function were significantly greater in the study group compared to the control group ($P < 0.05$). Although the study group exhibited lower incidences of MACEs and adverse reactions, the differences were not statistically significant. Conclusion: Sacubitril/valsartan sodium can significantly improve cardiac function and vascular endothelial function and attenuate ventricular remodeling in patients with heart failure after PCI for AMI. The treatment is well tolerated and demonstrates a favorable safety profile.

Keywords: Sacubitril/valsartan sodium, acute myocardial infarction, PCI, heart failure, ventricular remodeling

Introduction

Acute myocardial infarction (AMI) is a common cardiovascular emergency characterized by severe myocardial ischemia and necrosis resulting from acute coronary artery occlusion. Although the widespread adoption of percutaneous coronary intervention (PCI) has markedly reduced acute-phase mortality in AMI over recent years, irreversible myocardial injury and adverse ventricular remodeling remain prevalent [1, 2]. These pathological changes predispose a substantial proportion of patients to heart failure (HF), significantly compromising long-term survival and quality of life [3, 4]. Therefore, effectively preventing or reversing post-PCI ventricular remodeling and mitigating the progressive decline in cardiac function has become a major clinical challenge in the management of AMI.

Sacubitril/valsartan, a first-in-class angiotensin receptor-neprilysin inhibitor (ARNI), exerts dual effects by inhibiting the renin-angiotensin-aldosterone system (RAAS) and enhancing the activity of the natriuretic peptide system [5, 6]. This dual mechanism has been shown to improve cardiac function and reverse ventricular remodeling in patients with chronic heart failure [7, 8]. Currently, research regarding the use of sacubitril/valsartan in the early management of heart failure following acute myocardial

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infarction (AMI) remains relatively sparse. In particular, there is a lack of comprehensive evaluations concerning its impact on myocardial injury repair, ventricular remodeling, and long-term cardiovascular outcomes when compared to conventional therapies, such as valsartan.

This study aims to evaluate the therapeutic efficacy of sacubitril/valsartan sodium versus valsartan in patients with HF following PCI for AMI, focusing on improvements in cardiac function, reversal of ventricular remodeling, and prevention of major adverse cardiovascular events (MACEs). By systematically assessing these outcomes, this research seeks to provide evidence-based insights to support optimized early intervention strategies for post-AMI heart failure and to explore the potential prognostic advantages of sacubitril/valsartan in this setting.

Materials and methods

General data

This retrospective study analyzed clinical data from patients diagnosed with AMI who underwent PCI and subsequently developed heart failure between June 2023 and June 2024. A total of 108 patients who met the inclusion and exclusion criteria were enrolled. Based on their postoperative medication regimen, patients were divided into two groups: the control group (n = 39), which received standard therapy - including antiplatelet agents, diuretics, lipid-lowering drugs, and β-blockers - combined with valsartan; and the study group (n = 69), which received sacubitril/valsartan sodium in place of valsartan, in addition to the same standard treatment. This study was approved by the First People's Hospital of Tianshui Gansu's Ethics Committee.

To ensure baseline comparability, demographic and clinical characteristics - including gender, age, history of hypertension or diabetes, infarct location, time from onset to PCI, and New York Heart Association (NYHA) functional classification - were collected and analyzed. No statistically significant differences were observed between the two groups ($P > 0.05$), confirming that the baseline characteristics were well balanced.

Inclusion and exclusion criteria

Inclusion criteria: Patients were eligible for inclusion if they met all of the following criteria: (1) Diagnosis of AMI in accordance with the latest guidelines issued by the Chinese Society of Cardiology, with confirmation of ST-segment elevation myocardial infarction (STEMI) via coronary angiography and electrocardiography [9]. (2) Fulfillment of the diagnostic criteria for heart failure with mildly reduced ejection fraction (HFmrEF). (3) Left ventricular ejection fraction (LVEF) between 40% and 49%, as determined by echocardiography. (4) First episode of AMI, with PCI performed within 12 hours of symptom onset. (5) B-type natriuretic peptide (BNP) ≥ 150 ng/L or N-terminal pro-BNP (NT-proBNP) ≥ 600 μg/L.

Exclusion criteria: Patients were excluded if they met any of the following conditions: (1) History of chronic heart failure or HFmrEF due to other cardiovascular diseases. (2) Prior implantation of cardiac assist devices or history of heart transplantation. (3) Severe dysfunction of major organs (e.g., severe hepatic or renal insufficiency). (4) Known allergy to sacubitril/valsartan or other study medications. (5) Severe cognitive impairment that could compromise treatment adherence. (6) Presence of autoimmune diseases or malignancies with a life expectancy of less than one year. (7) Pregnancy or lactation.

Treatment methods

Patients in the control group received standard medical therapy, which included antiplatelet agents, lipid-lowering drugs, diuretics, and medications aimed at improving cardiac function and attenuating left ventricular remodeling. The primary medications included aspirin (100 mg once daily) and simvastatin (10 mg once daily), both administered orally with warm water. Additional agents included metoprolol (12.5 mg twice daily), captopril (12.5 mg twice daily), hydrochlorothiazide (25 mg twice daily), and isosorbide dinitrate (10 mg twice daily). Valsartan was initiated at a dose of 20 mg twice daily and titrated to a maintenance dose of 160 mg twice daily.

The study group received the same standard treatment regimen, with the substitution of

sacubitril/valsartan for valsartan. Sacubitril/valsartan was initiated at 50 mg twice daily and titrated to 100 mg twice daily based on patient tolerance. Both treatment regimens were administered in two cycles, with each cycle lasting 14 days.

Observation indicators

Primary outcome measures: 1. Serum biomarker analysis: Blood samples (5 mL) were collected before treatment and 6 months after treatment. Serum levels of NT-proBNP, cardiac troponin I (cTnI), and high-sensitivity C-reactive protein (hs-CRP) were quantified using enzyme-linked immunosorbent assay (ELISA) kits. All reagents were sourced from Wuhan Boet, Shanghai Kanglong, and Shanghai Fuyu Biochemical Technology.

2. Assessment of left ventricular function: Doppler echocardiography was performed before treatment and 6 months post-treatment to assess cardiac function. Key parameters included left ventricular end-diastolic volume (LVEDV), left ventricular end-systolic volume (LVESV), and LVEF.

3. Ventricular remodeling indicators: Ventricular structure and remodeling were assessed using color Doppler ultrasound before treatment and 6 months after treatment. Measurements included interventricular septal thickness (IVST), left ventricular posterior wall thickness (LVPWT), and left ventricular mass index (LVMI).

Secondary outcome measures: 1. MACEs: The incidence of major adverse cardiovascular events was monitored during the 6-month follow-up period after PCI. Events included heart failure, angina, and recurrent myocardial infarction. The incidence rate was calculated using the formula: MACE incidence = (number of heart failure + angina + recurrent myocardial infarction)/total number of cases * 100%.

2. Adverse reaction monitoring: The occurrence of side effects such as dizziness, hypotension, and nausea during treatment was recorded. The incidence rate was calculated and the differences between the two groups were analyzed.

3. Clinical efficacy assessment [10]: Clinical efficacy was evaluated at 1 and 6 months post-treatment using a three-grade scale: Markedly effective: Cardiac function improved by two or more NYHA classes, with varying degrees of improvement. Effective: Cardiac function improved by one NYHA class with different clinical manifestations. Ineffective: No improvement or further deterioration in cardiac function. The overall efficacy was calculated using the following formula: Total efficacy = (number of effective cases + number of markedly effective cases)/total number of cases * 100%.

Statistical analysis

All statistical analyses were performed using SPSS version 21.0. Categorical data were expressed as percentages (%), and continuous data were presented as mean ± standard deviation ($\bar{x} \pm SD$). Chi-square (χ^2) test and t-test were used for comparison and analysis of the results. A *P* value of < 0.05 was considered statistically significant.

Results

Baseline data

There were no statistically significant differences between the control and study groups in terms of sex, age, time from onset to treatment, Killip classification, body mass index (BMI), ST-segment elevation ratio, lesion type, or stent implantation (P > 0.05). These findings indicate that baseline characteristics were well-balanced and comparable between the two groups. Detailed data are presented in **Table 1**.

Clinical efficacy

Clinical efficacy was assessed based on symptomatic improvement, NYHA functional class improvement, and control of MACEs. Outcomes were categorized as markedly effective, effective, or ineffective, with the total clinical efficacy rate defined as the sum of markedly effective and effective cases.

At one month post-treatment, the total clinical efficacy rate was 86.95% in the study group and 79.49% in the control group, with no statistically significant difference (P > 0.05). However, after six months of treatment, the efficacy rate in the study group increased to 95.65%, which was significantly higher than that in the control group (79.49%) (P < 0.05). These results are illustrated in **Figure 1**.

Table 1. Comparison of clinical data between the two groups

Index	Control group (n = 39)	Study group (n = 69)	χ^2/t	*P*
Gender (Male/female)	25/14	40/29	0.391	0.532
Age (years)	56.9 ± 7.2	57.2 ± 8.5	0.186	0.853
Time from onset to treatment (h)	5.92 ± 2.93	6.22 ± 2.55	0.556	0.579
Killip classification (II/III/IV)	21/11/7	31/25/13	0.904	0.636
Body Mass index (kg/m^2)	23.87 ± 0.95	24.09 ± 0.77	1.208	0.230
ST segment elevation [n (%)]	3 (7.69)	9 (13.04)	0.282	0.595
Pathological condition				
Extensive anterior myocardial infarction	20	32	0.266	0.876
Inferior myocardial infarction	14	28		
Multiple vessel disease	5	9		
Stent implantation	20	30	0.610	0.435

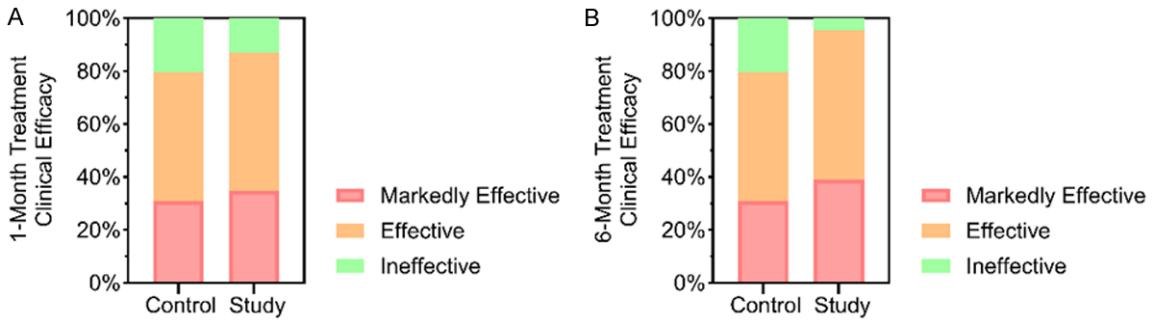

Figure 1. Comparison of clinical efficacy between the two groups. Note: A: The efficacy at 30 days after treatment was compared, and no significant difference was observed between the groups; B: After six months of treatment, the effective rate of the study group was significantly higher than that of the control group. The control group consisted of 39 cases, and the study group included 69 cases.

Evaluation of myocardial injury markers in both groups

Baseline levels of myocardial injury markers - including cTnI, hs-CRP and NT-proBNP - did not differ significantly between the two groups prior to treatment (P > 0.05). After treatment, however, all three markers were significantly lower in the study group compared to the control group, with statistically significant differences (P < 0.05) (**Figure 2**).

Evaluation of cardiac function before and after treatment

Echocardiographic assessments were performed to evaluate changes in cardiac function, including measurements of LVEF, LVEDd, and LVESd. Prior to treatment, there were no significant differences in these parameters between the two groups (P > 0.05). After six months of treatment, both groups demonstrated significant improvements: LVEF increased, while LVEDd and LVESd decreased compared to baseline (P < 0.05). Notably, the study group exhibited a more pronounced increase in LVEF and greater reductions in LVEDd and LVESd relative to the control group, with all differences reaching statistical significance (P < 0.05). These results are illustrated in **Figure 3**.

Ventricular remodeling indicators

To assess the degree of ventricular remodeling, interventricular septal thickness (IVST), left ventricular posterior wall thickness (LVPWT), and left ventricular mass index (LVMI) were measured. No significant differences were observed between the two groups before treatment (P > 0.05). After treatment, both groups showed increases in IVST, LVPWT, and LVMI compared to pre-treatment levels. However, the post-treatment values were significantly lower in the study group than in the control group (P < 0.05) (**Figure 4**).

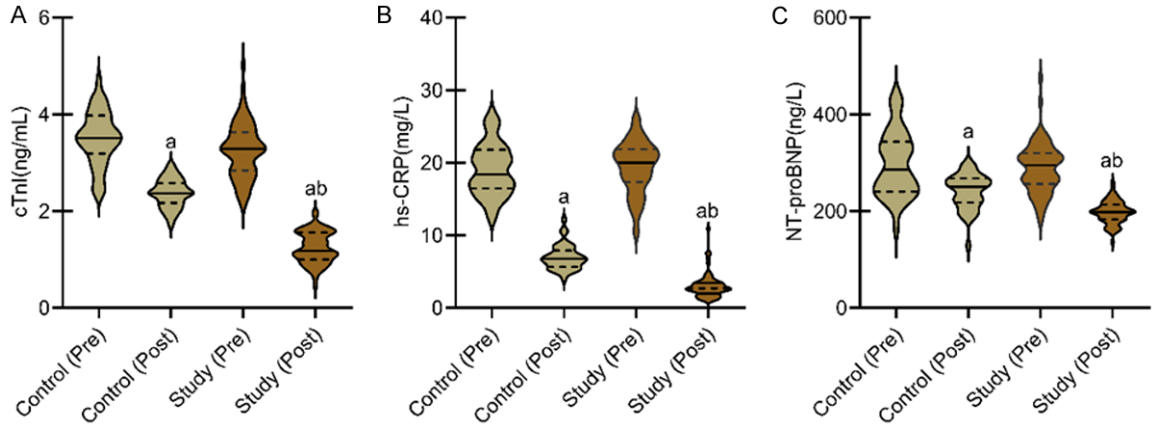

Figure 2. Comparison of myocardial injury markers between the two groups. Note: After treatment, the levels of (A) cTnI, (B) hs-CRP and (C) NT-proBNP in the study group were better than those in the control group. With-in group comparison before and after treatment was performed. When $P < 0.05$, a was used to represent the comparison result of the study group, and b was used to represent the comparison result of the control group. The control group consisted of 39 cases, and the study group included 69 cases. cTnI: Cardiac Troponin I; hs-CRP: High-sensitivity C-reactive protein; NT-proBNP: N-terminal pro-brain natriuretic peptide.

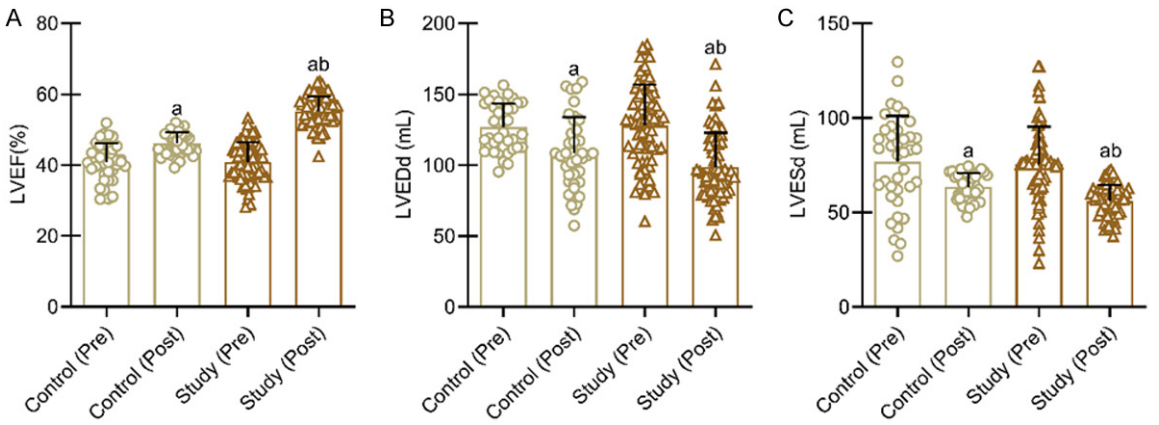

Figure 3. Comparison of cardiac function indexes between the two groups. Note: After treatment, the values of LVEF (A) in the study group were significantly higher than those in the control group, while the values of LVEDd (B) and LVESd (C) in the study group were significantly lower than those in the control group. When $P < 0.05$, a was used to represent the comparative results of study group, and b was used to represent the comparative results of control group. The control group consisted of 39 cases, and the study group included 69 cases. LVEF: Left ventricular ejection fraction; LVEDd: Left ventricular end-diastolic diameter; LVESd: Left ventricular end-systolic diameter.

Evaluation of vital signs

Vital signs - including systolic blood pressure (SBP), diastolic blood pressure (DBP), and resting heart rate (HR) - were monitored throughout the study. No significant differences were observed between the two groups before treatment ($P > 0.05$). After 6 months of treatment, all parameters showed statistically significant reductions within both groups ($P < 0.05$). However, there were no significant differences between the two groups after treatment ($P > 0.05$) (**Table 2**).

Comparison of KCCQ scores and 6MWT values

The Kansas City Cardiomyopathy Questionnaire (KCCQ) score was used to assess the symptoms, functional status, and quality of life in patients with heart failure, where a higher score indicates better quality of life. The 6-Minute Walk Test (6MWT) was used to evaluate exercise tolerance, with longer walking distances indicating better cardiac function. Before treatment, no significant differences were observed in KCCQ scores or 6MWT values between the two groups ($P > 0.05$). After 6 months of treat-

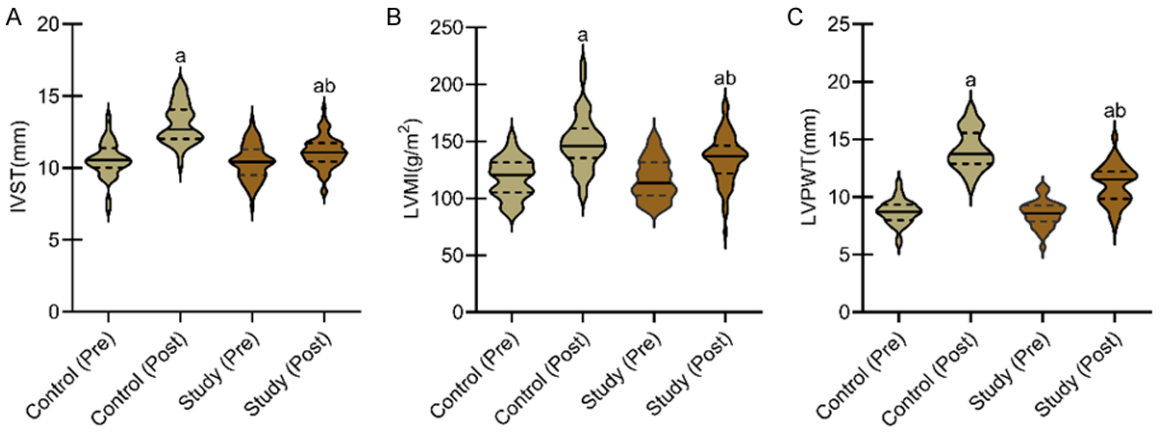

Figure 4. Comparison of ventricular remodeling indexes between the two groups. Note: The elevations of (A) IVST, (B) LVMI and (C) LVPWT in the study group after treatment were lower than those in the control group. With-in group comparison before and after treatment was performed. When P < 0.05, a was used to represent the comparison result of the study group, and b was used to represent the comparison result of the control group. The control group consisted of 39 cases, and the study group included 69 cases. IVST: Interventricular Septal Thickness; LVMI: Left ventricular mass index; LVPWT: Left ventricular posterior wall thickness.

Table 2. Comparison of blood pressure and heart rate between the two groups

Group		Control group (n = 39)	Study group (n = 69)	*t*	*P*
Systolic blood pressure (mmHg)	Pre-treatment	127.06 ± 18.85	126.55 ± 20.02	0.130	0.897
	Post-treatment	119.87 ± 19.72[a]	115.49 ± 18.72[a]	1.146	0.255
Diastolic blood pressure (mmHg)	Pre-treatment	79.52 ± 17.65	80.16 ± 18.17	0.178	0.859
	Post-treatment	68.22 ± 13.52[a]	65.09 ± 15.57[a]	1.051	0.296
Heart rate (times/min)	Pre-treatment	69.84 ± 12.74	68.22 ± 13.31	0.617	0.539
	Post-treatment	68.25 ± 10.42	66.57 ± 12.33	0.718	0.474

Note: Compared with this group before treatment, [a]P < 0.05.

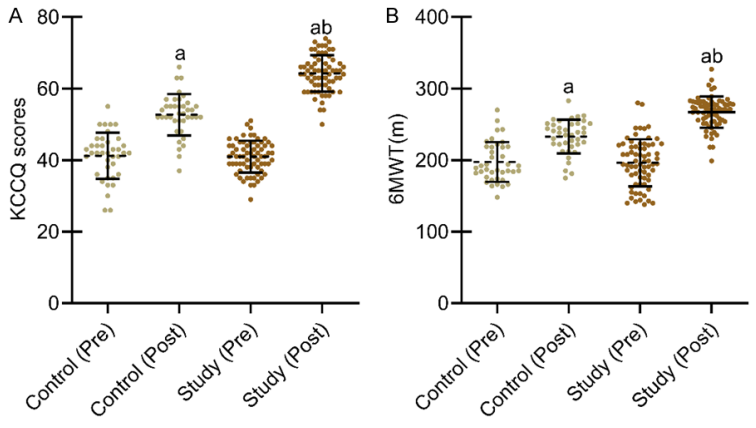

Figure 5. Comparison of KCCQ scores and 6MWT levels between the two groups. Note: After treatment, the (A) KCCQ score and (B) 6MWD in the study group were significantly higher than those in the control group. For comparison before and after treatment between the two groups, when P < 0.05, a is used to represent the comparison result of the study group, and b is used to represent the comparison result of the control group. The control group consisted of 39 cases, and the study group included 69 cases. KCCQ: Kansas City Cardiomyopathy Questionnaire; 6MWD: 6-Minute walk distance.

ment, the study group showed significant improvements in both KCCQ scores and 6MWD results compared to the control group, with statistically significant differences (P < 0.05) (**Figure 5**).

Major adverse cardiovascular events (MACE)

MACEs were defined as recurrent myocardial infarction, angina, arrhythmia, or cardiac death. Over the 6-months follow-up period, the incidence of MACE was 17.95% in the control group and 4.35% in the study group. The difference between the two groups was statistically significant (P < 0.05) (**Table 3**).

Table 3. Comparison of MACE incidence between the two groups n (%)

Group	Recurrent myocardial infarction	Angina pectoris	Arrhythmia	Cardiac death	Incidence of MACE
Control group (n = 39)	2 (5.13)	2 (5.13)	3 (7.69)	0	7 (17.95)
Study group (n = 69)	2 (8.7)	1 (0.00)	0 (0.00)	0	3 (4.35)
χ^2					3.987
P					0.046

Table 4. Evaluation of adverse reactions after treatment [n (%)]

Group	Symptomatic hypotension	Dizziness	Nausea and vomiting	Hyperkalemia	Total incidence
Control group (n = 39)	2 (5.13)	3 (7.69)	1 (2.56)	1 (2.56)	7 (17.95)
Study group (n = 69)	3 (7.69)	4 (5.80)	2 (2.90)	2 (2.90)	11 (15.94)
χ^2					0.360
P					0.549

Comparison of adverse reaction incidence

Adverse reactions mainly include hypotension, abnormal renal function and hyperkalemia. The incidence of adverse reactions after treatment was lower in the study group compared to the control group. However, the difference did not reach statistical significance (P > 0.05) (**Table 4**).

Discussion

Patients with AMI who undergo PCI can achieve rapid reperfusion of the infarct-related artery (IRA). However, factors such as myocardial cell death, ventricular remodeling, and reperfusion injury prevent the complete mitigation of myocardial damage. Studies have shown that approximately 13% of AMI patients develop heart failure within 30 days of discharge, and 20-30% are diagnosed with heart failure within one year, significantly increasing the risk of MACEs [11, 12]. Therefore, preventing and treating heart failure after AMI has become a clinical focus. Moreover, the progression of ventricular remodeling and cardiac function deterioration is influenced not only by the infarct size but also by oxidative stress, endothelial dysfunction, and chronic low-grade inflammation during reperfusion. These factors exacerbate the development of heart failure. Addressing these pathological processes is crucial for optimizing post-AMI treatment strategies. This study explored the efficacy of sacubitril/valsartan in patients with heart failure after AMI PCI, and the results showed that sacubitril/valsartan significantly improved heart function, attenuated ventricular remodeling, and reduced myocardial injury biomarkers, and lowered the incidence of MACE.

Compared to the traditional therapy with valsartan, sacubitril/valsartan provides a more comprehensive inhibition of detrimental cardiac remodeling. This dual mechanism blocking the renin-angiotensin-aldosterone system (RAAS) and enhancing natriuretic peptide system (NPS) activity-promotes a more favorable cardiac adaptive response. As a result, sacubitril/valsartan offers clear clinical advantages in improving cardiac function. The findings of this study align with previous research by Liu Zhengfeng et al., who demonstrated that early use of sacubitril/valsartan sodium effectively improves heart function and heart rate variability markers in AMI patients [13]. The mechanism underlying these benefits may involve the dual action of sacubitril/valsartan: valsartan antagonizes the angiotensin II receptor, inhibiting the overactive RAAS, which reduces myocardial cell apoptosis and fibrosis [14]; concurrently, sacubitril inhibits neprilysin, enhancing the activity of the endogenous natriuretic peptide system (NPS), thereby promoting vasodilation, diuresis, and anti-fibrotic effects, all of which contribute to improved cardiac remodeling and function [15].

Experimental studies have confirmed that sacubitril/valsartan can inhibit myocardial fibrosis gene expression by downregulating the TGF-β1/Smad signaling pathway, thus preventing

myocardial interstitial remodeling and fibrosis. This provides a molecular basis for improving both systolic and diastolic cardiac function. Despite early PPCI reperfusion therapy, AMI patients may still experience left ventricular (LV) remodeling due to factors such as cardiac stretching, neuroendocrine activation, paracrine and/or autocrine factors, and RAAS activation [16]. Studies have shown that pathological left ventricular remodeling after myocardial infarction is closely associated with the risk of heart failure (HF), with myocardial injury and ventricular remodeling often interacting synergistically [17]. Research has indicated that sacubitril/valsartan can reverse cardiac remodeling, improve cardiac structure and systolic function, and increase LVEF, thereby enhancing the prognosis of HF patients [18]. These findings are consistent with the results of this study. Our study demonstrates that sacubitril/valsartan effectively reduces myocardial remodeling indicators, such as IVST, LVPWT, and LVMI. Previous studies have shown that these pathological processes may be related to overactivation of the RAAS, which leads to myocardial hypertrophy, interstitial fibrosis, and subsequent cardiac remodeling. Through its dual mechanism of regulating both RAAS and NPS, sacubitril/valsartan effectively inhibits myocardial remodeling, improves cardiac compliance, and delays the progression of HF [19]. Notably, activation of the natriuretic peptide system also increases cyclic guanosine monophosphate (cGMP) levels in myocardial cells, promoting recovery of myocardial diastolic function, reducing myocardial cell proliferation, and further intervening in the molecular-level changes in left ventricular geometry.

Myocardial injury markers such as cTnI, hs-CRP, and NT-proBNP are key indicators for evaluating myocardial injury and the efficacy of heart failure treatment [20, 21]. In this study, sacubitril/valsartan demonstrated a superior effect in alleviating myocardial injury compared to conventional treatments. The potential mechanism is likely related to the drug's ability to reduce myocardial stress, mitigate myocardial cell apoptosis, and attenuate fibrosis, thereby lowering the degree of myocardial injury. Furthermore, NT-proBNP, a key biomarker for diagnosing and assessing the prognosis of heart failure, showed a decrease in its levels, suggesting that sacubitril/valsartan not only improves cardiac function but also reduces the risk of heart failure. It is hypothesized that the reduction in NT-proBNP levels is primarily due to the drug's effect on relieving ventricular wall stress, along with its role in decreasing left atrial pressure and alleviating pulmonary congestion, which is crucial for preventing the advancement of heart failure.

Additionally, this study utilized the KCCQ score and 6MWD to assess the patient's quality of life and exercise capacity. The results indicated that sacubitril/valsartan not only improved cardiac function and also enhanced patients' exercise tolerance, thereby improving their quality of life. This effect may be related to the drug's ability to improve myocardial energy metabolism, reduce ventricular load, and enhance cardiac compliance [22, 23]. Some studies have suggested that sacubitril/valsartan also improves skeletal muscle microcirculation and oxygen utilization efficiency, which could further explain the observed improvements in exercise endurance and fatigue reduction. MACEs are critical indicators of the long-term prognosis of heart failure patients after AMI [24, 25]. The findings of this study suggest that sacubitril/valsartan can effectively reduce the risk of such events. This protective effect may result from the drug's improvement of cardiac function, reduction in myocardial fibrosis, enhancement of endothelial function, and attenuation of inflammatory responses, all of which help prevent complications such as recurrent myocardial infarction and arrhythmias [26, 27]. Through its modulation of NPS, sacubitril/valsartan also inhibits platelet activation and improves hemodynamics, potentially offering additional protection against recurrent thrombotic events and sudden death following AMI. Moreover, this study found that the incidence of adverse reactions was 15.94% in the study group, slightly lower than the 17.95% observed in the control group, though the difference was not significantly significant. This suggests that sacubitril/valsartan has a favorable safety profile in patients with heart failure after AMI.

This study has certain limitations. First, being a retrospective, single-center study with a relatively small sample size, the generalizability of the findings may be limited. Second, the follow-up duration was restricted to six months, and

long-term outcomes remain unassessed. Additionally, the study did not perform a detailed stratified analysis of patients' concomitant medications, which could introduce potential confounding factors. Therefore, future multi-center, large-sample, long-term follow-up prospective studies are needed to further validate these findings and optimize individualized treatment strategies for heart failure after AMI.

In conclusion, sacubitril/valsartan can effectively improve heart function in patients with heart failure following AMI PCI, inhibit ventricular remodeling, reduce myocardial injury markers, enhance exercise tolerance, and improve overall quality of life. Moreover, it significantly reduces the risk of MACEs and has a favorable safety profile. These findings position sacubitril/valsartan as an important therapeutic option for managing heart failure after AMI. This study provides valuable clinical evidence for optimizing the management of heart failure following AMI.

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Disclosure of conflict of interest

None.

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