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*Article*

# A Retrospective Study of Whether Sacubitril–Valsartan Improves Cardiac Function in Patients with Acute Coronary Syndrome and Hypertension after Percutaneous Coronary Intervention

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## Abstract

**Objectives**: This study aimed to explore the effect of sacubitril–valsartan on cardiac function in patients with acute coronary syndrome (ACS) and hypertension after percutaneous coronary intervention (PCI). **Methods**: The study was conducted on 166 patients with ACS and hypertension who underwent PCI at our hospital from July 2022 to June 2023. The patients were divided into two groups: an observation group and a control group, with 83 cases in each group. The observation group was given sacubitril–valsartan, and the control group was given enalapril. The treatment period was 3 months. The cardiac function indexes, biochemical indexes, adverse reactions, and the occurrence of new cardiovascular and cerebrovascular events were compared between the two groups before and after medication. **Results**: We found no statistically dramatic differences between the two groups in gender, age, body mass index, diabetes, hyperlipidemia, family history of ACS, family history of hypertension, smoking history, drinking history, regular exercise history, low-salt and low-fat diet, New York Heart Association cardiac function classification, number of vessels involved in coronary artery disease, stent length, stent inner diameter, number of stent implants, duration of hypertension, medication time, use of antihypertensive drugs, systolic blood pressure (SBP), and diastolic blood pressure (DBP). The observation group had lower left ventricular end-diastolic internal diameter (LVEDD) levels, higher left ventricular ejection fraction (LVEF) levels, and lower left ventricular end-systolic internal diameter (LVESD) levels compared with the control group. The levels of N-terminal pro-brain B-type natriuretic peptide (NT-proBNP), cardiac troponin I (cTnI), and creatine kinase isoenzyme MB (CK-MB) in the observation group decreased compared with those in the control group. Compared with the pre-medication period, LVEDD levels decreased, LVEF levels increased, and LVESD levels decreased in the observation and control groups, whereas NT-proBNP, cTnI, and CK-MB levels decreased in the observation and control groups. The total incidence rates of adverse reactions and new cardiovascular and cerebrovascular events in the observation group were found to be lower than those in the control group. **Conclusion**: The application of sacubitril–valsartan in patients with ACS complicated with hypertension after PCI can effectively improve patients' cardiac function, reduce biochemical indexes related to myocardial injury, and lower the incidence of adverse cardiovascular events. It features good safety, providing a new treatment strategy and method for patients with ACS complicated with hypertension.

## Keywords

sacubitril–valsartan; acute coronary syndrome; hypertension; percutaneous coronary intervention; cardiac function

## Introduction

Acute coronary syndrome (ACS) is a group of syndromes characterized by acute myocardial ischemia resulting from coronary atherosclerosis, leading to a disruption in blood supply to the heart. It is considered a severe form of coronary heart disease, encompassing acute myocardial infarction and unstable angina pectoris [1,2]. The clinical manifestations of ACS often include a squeezing pain in the chest area that can radiate to the left shoulder, back, neck, and jaw. It occurs intermittently or continuously, has a high mortality rate, and seriously threatens life safety. The incidence rate of ACS has been increasing steadily in the few years. It is more commonly observed among the elderly population, as well as among individuals with diabetes, hypertension, smoking history, and prolonged heavy alcohol consumption [3,4]. Percutaneous coronary intervention (PCI) is a common treatment for ACS. It is a relatively simple and minimally invasive procedure that yields positive treatment outcomes. It causes minimal trauma to the patient's body and effectively restores coronary blood perfusion. It shortens the recovery time of patients and has high safety [5–7]. However, the overall therapeutic effect of PCI needs to be further improved. Relevant studies have found [8,9] that in patients with ACS and pre-existing hypertension, changes in blood pressure may activate and worsen

the chronic inflammatory response of the heart. This phenomenon can accelerate the process of atherosclerosis and promote structural remodeling of the heart, increasing the risk of adverse cardiovascular events. Shakubatroxobin and valsartan, the main components of which are Shakubatroxobin and Zosartan, are the first angiotensin receptor enkephalinase inhibitors in the world. These new drugs belong to angiotensin-converting enzyme inhibitor (ACEI), and they have a dual mechanism of action, which can lower blood pressure, improve cardiac function, and inhibit myocardial hypertrophy and fibrosis [10]. The study demonstrated that sacubitril–valsartan is linked to a notable decrease in the risk of heart failure hospitalization and cardiovascular death among patients with reduced left ventricular ejection fraction (LVEF) [11]. In clinical practice, even if PCI can alleviate coronary artery stenosis and restore coronary blood supply effectively, patients may still develop secondary heart failure. Relevant studies have indicated that patients with ACS who experienced reduced LVEF after undergoing PCI demonstrated significantly improved cardiac function and substantial reversal of cardiac remodeling with the administration of sacubitril–valsartan [12]. This improvement was found to be more pronounced compared with conventional treatments. Therefore, the objective of this study was to investigate the effect of sacubitril–valsartan on cardiac function following PCI in patients with ACS and hypertension. The results aim to offer insights and guidance for the clinical application of sacubitril–valsartan.

## Materials and Methods

### *General Information*

This study involved a total of 166 patients with ACS and hypertension who underwent PCI at our hospital from July 2022 to June 2023. On the basis of the different treatment methods, these patients were separated into two groups: the observation group (n = 83) and the control group (n = 83). The observation group used sacubitril–valsartan after PCI. The control group used enalapril after PCI. This study has undergone a thorough review and approval by the Ethics Committee of The First People's Hospital of Jiashan (Approval No.: 2024041), and an ethics certificate was obtained. Adhering to the principle of confidentiality, we rigorously safeguard the personal and family information of patients with ACS complicated by hypertension and strictly prohibit any unauthorized disclosure.

### *Inclusion Criteria and Exclusion Criteria*

Inclusion criteria: (1) age above 18; (2) primary PCI; (3) heart rate above 60 bpm with(out) $\beta$-blockers; (4) essential hypertension stage I or II, systolic blood pressure (SBP) between 140 and 179 mmHg, diastolic blood pressure (DBP) between 90 and 109 mmHg; and (5) the subject had never taken enalapril or elbow devalsartan before the study.

Exclusion criteria: (1) stenotic of left main coronary artery was over 50% after PCI; (2) surgery only included thrombosuction or balloon angioplasty; (3) second-degree atrioventricular block, atrial block fibrillation, tachycardia, or sick sinus syndrome; (4) New York Heart Association (NYHA) cardiac function class was above III; (5) chronic respiratory disease, severe liver, kidney disease, or hyperthyroidism; and (6) serious cognitive impairment and mental illness.

### *Method*

Observation group: Routine interventions, oxygen, rest, low-sodium diet, control of water intake, correction of electrolyte disturbances, and oral administration of conventional medications (ACEI or angiotensin II receptor blockers (ARB), beta-blockers, calcium channel blockers (CCB), diuretics) + Sacubitril–Valsartan (Novartis Pharma Stein AG, National Pharmaceutical License No. H20170344, Beijing, China), with an initial dose of 25 mg/dose for two times/day. The dose was increased at the patient's discretion until 200 mg/dose for 2 times/day. The treatment cycle was 3 months.

Control group: Routine interventions were carried out, including oxygen, rest, low-sodium diet, control of water intake, correction of electrolyte disorders, and oral administration of conventional drugs (ACEI or ARB, $\beta$-blockers, CCB, and diuretics) + enalapril (Yangzijiang Pharmaceutical Group Jiangsu Pharmaceutical Co., Ltd., China National Pharmaceutical License No. H32026568, Jiangsu, China), with an initial dosage of 5 mg/times for two times/day. The treatment cycle was 3 months. The initial dose was 5 mg/times for two times/day, and the dosage was increased according to the patient's condition until 10 mg/times for two times/day. The treatment cycle was 3 months.

### *Observation Indicators*

#### Comparison of Baseline Characteristics

We collected the basic information of patients through medical records and electronic medical records, including gender, age, body mass index (BMI), diabetes, hyperlipidemia, family history of ACS, family history of hypertension, smoking history, drinking history, regular exercise history, low-salt and low-fat diet, New York Heart Association (NYHA) cardiac function classification, number of vessels involved in coronary artery disease, stent length, stent inner diameter, number of stent implants, duration of hypertension, medication time, use of antihypertensive drugs (ACEI or ARB, beta blockers, CCB, diuretics), SBP, and DBP.

**Table 1. Comparison of baseline data.**

| Group | Observation group (n = 83) | Control group (n = 83) | $\chi^2/t$ | $p$ |
|---|---|---|---|---|
| Gender (n, %) | | | 0.218 | 0.641 |
| Male | 43 (51.81) | 46 (55.42) | | |
| Female | 40 (48.19) | 37 (44.58) | | |
| Age | 60.18 ± 7.35 | 60.33 ± 7.24 | 0.128 | 0.899 |
| BMI (kg/m$^2$) | 22.15 ± 1.02 | 22.03 ± 1.05 | 0.755 | 0.452 |
| Diabetes (n, %) | | | 0.223 | 0.637 |
| Yes | 36 (43.37) | 33 (39.76) | | |
| No | 47 (56.63) | 50 (60.24) | | |
| Hyperlipidemia (n, %) | | | 0.097 | 0.756 |
| Yes | 38 (45.78) | 40 (48.19) | | |
| No | 45 (54.22) | 43 (51.81) | | |
| Family history of ACS (n, %) | | | 0.103 | 0.748 |
| Yes | 32 (38.55) | 30 (36.15) | | |
| No | 51 (61.45) | 53 (63.86) | | |
| Family history of hypertension (n, %) | | | 0.402 | 0.526 |
| Yes | 35 (42.17) | 31 (37.35) | | |
| No | 48 (57.83) | 52 (62.65) | | |
| Smoking history (n, %) | | | 0.097 | 0.756 |
| Yes | 42 (50.60) | 44 (53.01) | | |
| No | 41 (49.40) | 39 (46.99) | | |
| Drinking history (n, %) | | | 0.000 | 0.986 |
| Yes | 46 (55.42) | 48 (57.83) | | |
| No | 37 (44.58) | 35 (42.17) | | |
| History of regular exercise (n, %) | | | 0.230 | 0.631 |
| Yes | 30 (36.14) | 33 (39.76) | | |
| No | 53 (63.86) | 50 (60.24) | | |
| Low-salt and low-fat diet (n, %) | | | 2.427 | 0.119 |
| Yes | 40 (48.19) | 50 (60.24) | | |
| No | 43 (51.81) | 33 (39.76) | | |
| NYHA classification (n, %) | | | 0.037 | 0.982 |
| I | 24 (21.69) | 23 (24.10) | | |
| II | 28 (26.51) | 28 (30.12) | | |
| III | 31 (31.33) | 32 (34.94) | | |
| Number of blood vessels involved in coronary artery disease (n, %) | | | 0.489 | 0.783 |
| 1 | 29 (34.94) | 25 (30.12) | | |
| 2 | 30 (36.14) | 31 (37.35) | | |
| 3 | 24 (28.92) | 27 (32.53) | | |
| Bracket length (mm) | 13.80 ± 2.17 | 14.04 ± 2.14 | 0.720 | 0.473 |
| Inner diameter of stent (mm) | 2.81 ± 0.63 | 2.78 ± 0.50 | 0.273 | 0.785 |
| Number of stents implanted (pieces) | 1.20 ± 0.54 | 1.22 ± 0.44 | 0.158 | 0.875 |
| Duration of hypertension (years) | 8.06 ± 1.36 | 8.10 ± 1.38 | 0.170 | 0.865 |
| Medication time (years) | 7.94 ± 1.07 | 7.78 ± 1.15 | 0.907 | 0.366 |
| Antihypertensive drug use (n, %) | | | 0.180 | 0.981 |
| ACEI or ARB | 15 (18.07) | 13 (15.66) | | |
| Beta blockers | 25 (30.12) | 26 (31.33) | | |
| CCB | 28 (33.73) | 29 (34.94) | | |
| Diuretics | 15 (18.07) | 15 (18.07) | | |
| SBP (mmHg) | 150.23 ± 10.36 | 151.70 ± 11.50 | 0.865 | 0.388 |
| DBP (mmHg) | 100.24 ± 8.17 | 100.59 ± 7.97 | 0.279 | 0.781 |

BMI, body mass index; ACS, acute coronary syndrome; NYHA, New York Heart Association; SBP, systolic blood pressure; DBP, diastolic blood pressure; ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blockers; CCB, calcium channel blocker.

**Table 2. Comparison of LVEDD, LVEF, and LVESD levels ($\bar{x} \pm s$, n = 83).**

| Group | LVEDD (mm) | | *t* | *p* | LVEF (%) | | *t* | *p* | LVESD (mm) | | *t* | *p* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Before medication | After medication | | | Before medication | After medication | | | Before medication | After medication | | |
| Observation group | 65.16 ± 5.32 | 50.18 ± 3.17 | 23.466 | <0.001 | 52.27 ± 4.80 | 65.31 ± 3.19 | 21.243 | <0.001 | 45.27 ± 3.12 | 35.25 ± 3.03 | 21.249 | <0.001 |
| Control group | 64.94 ± 5.31 | 56.22 ± 2.91 | 13.352 | <0.001 | 52.75 ± 4.84 | 60.02 ± 3.01 | 10.848 | <0.001 | 45.43 ± 3.22 | 38.24 ± 2.09 | 16.278 | <0.001 |
| *t* | 0.263 | 12.780 | | | 0.644 | 10.978 | | | 0.343 | 7.403 | | |
| *p* | 0.793 | <0.001 | | | 0.521 | <0.001 | | | 0.732 | <0.001 | | |

LVEDD, left ventricular end-diastolic internal diameter; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic internal diameter.

**Table 3. Comparison of NT-proBNP, cTnI, and CK-MB levels ($\bar{x} \pm s$, n = 83).**

| Group | NT-proBNP (pg/mL) | | *t* | *p* | cTnI (μg/L) | | *t* | *p* | CK-MB (U/L) | | *t* | *p* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Before medication | After medication | | | Before medication | After medication | | | Before medication | After medication | | |
| Observation group | 1425.25 ± 133.61 | 362.58 ± 40.25 | 67.049 | <0.001 | 1.36 ± 0.25 | 0.39 ± 0.08 | 32.638 | <0.001 | 88.46 ± 10.25 | 44.38 ± 7.96 | 30.448 | <0.001 |
| Control group | 1396.46 ± 140.76 | 400.23 ± 50.13 | 60.592 | <0.001 | 1.29 ± 0.27 | 0.72 ± 0.14 | 16.739 | <0.001 | 85.47 ± 9.88 | 60.25 ± 8.02 | 20.624 | <0.001 |
| *t* | 1.351 | 5.335 | | | 1.761 | 19.352 | | | 1.916 | 12.795 | | |
| *p* | 0.178 | <0.001 | | | 0.080 | <0.001 | | | 0.057 | <0.001 | | |

NT-proBNP, N-terminal pro-brain B-type natriuretic peptide; cTnI, cardiac troponin I; CK-MB, creatine kinase isoenzyme MB.

### Comparison of Cardiac Function Indicators before and after Medication

The GE Vivid E9 echocardiography instrument (General Electric Healthcare, Milwaukee, WI, USA) was employed to measure the left ventricular end-diastolic internal diameter (LVEDD), LVEF, and left ventricular systole before and after medication. The left ventricular end-systolic internal diameter (LVESD) was measured over three cardiac cycles, and the average value was calculated.

### Comparison of Biochemical Indicators before and after Treatment

The level of N-terminal pro-brain B-type natriuretic peptide (NT-proBNP) was determined by using the fluorescein-enhanced immunochemiluminescence method. The plasma levels of cardiac troponin I (cTnI) and creatine kinase isoenzyme MB (CK-MB) were detected using the chemiluminescence method.

### Adverse Reactions and New Cardiovascular and Cerebrovascular Events in Two Groups

The data included statistics on the occurrence of adverse reactions such as bradycardia, hypotension, renal damage, hyperkalemia, and other adverse reactions that occurred after taking the medication and before the end of follow-up. Results also included statistics on cardiovascular death, myocardial infarction, apoplexy, hospitalization for heart failure, and other cardiovascular and cerebrovascular diseases that occurred during the follow-up period. The data focused on the occurrence of new incidents.

### *Statistical Methods*

The continuous variables that followed a normal distribution, such as age and BMI, were recorded as mean $\pm$ standard deviation by using SPSS 21.0 (SPSS Inc., Armonk, NI, USA). Independent sample *t*-tests were employed to compare the differences between groups. Measurement indicators were recorded as (number of cases [percentage]) and compared by $\chi^2$ test. The basic formula of the chi-square test was employed when the sample size was bigger than 40, and the theoretical frequency T was above 5. The chi-square test correction formula was employed when the sample size was bigger than 40, and the theoretical frequency T was between 1 and 5. The Fisher's exact probability method was employed when the size of sample was smaller than 40 or the theoretical frequency T was under 1. $p < 0.05$ indicated a statistically significant difference.

## Results

### *Comparison of Baseline Characteristics*

No statistically significant difference was found in baseline characteristics between the two groups ($p > 0.05$; Table 1).

### *Comparison of LVEDD, LVEF, and LVESD Levels*

Before medication, no statistical difference was found in LVEDD, LVEF, and LVESD levels between the two sets ($p > 0.05$). However, after medication, the LVEDD and the LVESD levels of the observation set decreased, and the LVEF levels of the observation set increased compared with the control set (t = 12.780, 10.978, 7.403, $p < 0.001$). LVEDD levels were lower, LVEF levels were higher, and LVESD levels were lower in the observation and control groups after the administration of the drug compared with those in the pre-drug period (Table 2).

### *Comparison of NT-proBNP, cTnI, and CK-MB Levels*

Before medication, no statistical difference was found in NT-proBNP, cTnI, and CK-MB between the two sets ($p > 0.05$). However, after medication, these indexes in the observation set were significantly reduced compared with those in the control set (t = 5.335, 19.352, 12.795, $p < 0.001$). The levels of NT-proBNP, cTnI, and CK-MB were reduced in the observation and control groups after the administration of the drug compared with those in the pre-drug period (Table 3).

### *Adverse Reactions and New Cardiovascular Events in the Two Groups*

The incidence of untoward reactions and new cardiovascular events in the observation group significantly decreased compared with that in the other group ($\chi^2$ = 5.240, 4.814, $p$ = 0.022, 0.028; Tables 4,5, Figs. 1,2).

## Discussion

ACS is a condition that occurs when there is a rupture or erosion of coronary atherosclerotic plaques in the arteries, leading to the release of highly thrombogenic substances into the bloodstream. These substances adhere to the damaged surface of plaques. Over time, they aggregate and form a blood clot, also known as a thrombus. This thrombus can then partially or completely block the coronary artery, resulting in a restriction of blood flow. This restriction can lead to severe stenosis or narrowing of the artery, which can have serious consequences such as myocardial ischemia and myocardial infarction [13,14]. PCI

**Table 4. Occurrence of adverse reactions (n, %).**

| Group | n | Bradycardia | Hypotension | Renal impairment | Hyperkalemia | Overall incidence |
|---|---|---|---|---|---|---|
| Observation group | 83 | 2 (2.41) | 2 (2.41) | 1 (1.20) | 1 (1.20) | 6 (7.23) |
| Control group | 83 | 4 (4.82) | 6 (7.23) | 2 (2.41) | 4 (4.82) | 16 (19.28) |
| $\chi^2$ | / | / | / | / | / | 5.240 |
| $p$ | / | / | / | / | / | 0.022 |

**Table 5. Emergency of new cardiovascular events (n, %).**

| Group | n | Cardiovascular death | Myocardial infarction | Stroke | Heart failure hospitalization | Overall incidence |
|---|---|---|---|---|---|---|
| Observation group | 83 | 1 (1.20) | 1 (1.20) | 1 (1.20) | 2 (2.41) | 5 (6.02) |
| Control group | 83 | 4 (4.82) | 3 (3.61) | 2 (2.41) | 5 (6.02) | 14 (16.87) |
| $\chi^2$ | | | | | | 4.814 |
| $p$ | | | | | | 0.028 |

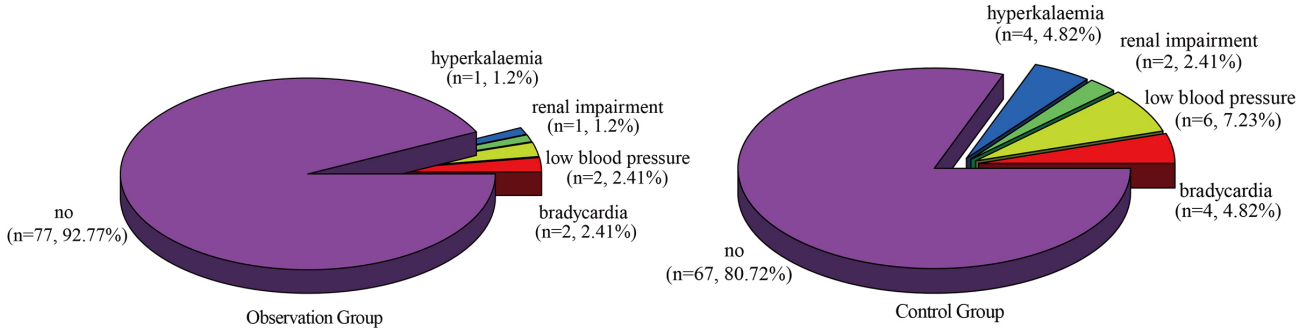


**Fig. 1. Three-dimensional pie chart of adverse reactions.**

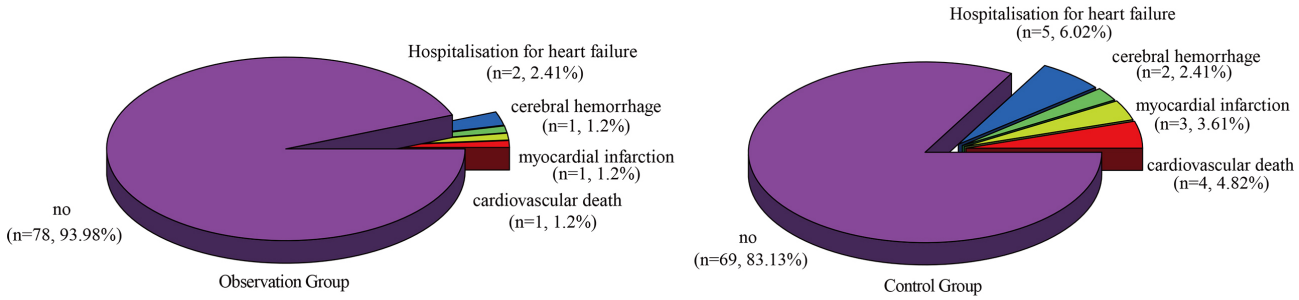


**Fig. 2. Three-dimensional pie chart of the emergency of new cardiovascular events.**

surgery is one of the primary treatments for ACS. After percutaneous puncture, arteriography technology is used to place a stent at the stenotic part of the coronary artery to quickly open the infarction-related arteries and restore blood supply to the heart. The effect is remarkable, but some patients still have heart failure, which is one of the most important causes of postoperative readmission and death [15]. In patients with ACS combined with hypertension, high blood pressure can further increase the burden on blood vessels and affect various aspects of cardiovascular health. It can increase vascular pressure and strain, promote the formation of blood clots, and impair myocardial cell metabolism. These factors ultimately contribute to an increased risk of cardiovascular events. Sacubitril–valsartan has the effects of angiotensin receptor blockers (ARBS) and neprilysin inhibitors. ARBS is a commonly used antihypertensive drug, mainly by inhibiting the activity of angiotensin T receptors to dilate blood vessels and decrease blood pressure. Neprilysin inhibitor is a new type of drug that can inhibit the activity of neprilysin and increase the level of natriuretic peptide to exert natriuretic, diuretic, and vasodilation effects; reduce blood pressure; and improve cardiac function [16–18]. This study highlighted the effectiveness and safety of using sacubitril–valsartan in patients with ACS and hypertension after PCI. The application was found to effectively improve the cardiac function, reduce myocardial damage-related biochemical indicators, and decrease the incidence of adverse cardiovascular events.

A study by Liu *et al.* [12] on the improvement of cardiac function and remodeling after sacubitril–valsartan treatment of ACS heart failure observed a notable decrease

in LVEF after PCI, which indicated that sacubitril–valsartan treatment of patients results in significant improvements. Changes in LVEF were significantly negatively correlated with changes in other indexes. The increase in LVEF and the decrease in left ventricular end-diastolic volume index (LVEDVI) and left ventricular end-systolic volume index (LVESVI) are considered protective factors for improving prognosis. Patients with myocardial infarction and reduced LVEF may derive remarkable benefits from initiating sacubitril–valsartan as first-line treatment for heart failure after PCI. LVEDD, LVEF, and LVESD are indicators related to cardiac function. LVEDD is mainly used to evaluate the diastolic function of the myocardium. LVEF is the indicator of the diastolic function and prognosis of the myocardium. LVESD mainly reflects the contractility of the myocardium. The above indicators are often used to measure the structure of the heart. Changes can reflect ventricular remodeling [19,20]. In this study, sacubitril–valsartan was used for cardiac function rehabilitation after PCI in patients with ACS and hypertension. The results of the study indicated that sacubitril–valsartan was more effective to patients than those who were treated with enalapril. The level of LVEDD was found to be reduced, the level of LVEF increased, and the level of LVESD was found to be reduced in patients with troponin T (TNT). Sacubitril–valsartan previously showed an improvement of cardiac remodeling and heart failure [21]. Previous studies have pointed out that the use of sacubitril–valsartan in patients with heart failure with reduced ejection fraction results in improving NT-proBNP, LVEDD, LVEF, and LVESD [22]. Existing studies also pointed out that compared with enalapril, the application of sacubitril–valsartan significantly improves LVEDD, LVEF, and LVESD; moreover, sacubitril–valsartan can improve cardiac remodeling and has good clinical efficacy [23,24].

NT-proBNP is an objective biological indicator of cardiac function and holds significant value in the diagnosis, treatment, and prognostic assessment of heart failure [25]. cTnI is a serum marker of myocardial injury. It only exists in myocardial cells and is the component of myofibrils. In normal circumstances, the concentration of cTnl in the bloodstream is extremely low and nearly undetectable. However, when the myocardium is damaged and myocardial cells are necrotic, cTnl is released into the bloodstream, and the concentration of cTnl increases significantly [26]. CK-MB mainly exists in cardiomyocytes. When myocardium is damaged, intracellular CK-MB is released into the bloodstream, leading to an increase in its levels. In patients with ACS, the obstruction or spasm of coronary arteries can cause myocardial cells to be damaged by ischemia, hypoxia, or necrosis, resulting in elevated levels of CK-MB. Therefore, monitoring the level of CK-MB can serve as a major index to assess the extent of myocardial injury and prognosis [27]. After medication intervention in this study, the NT-proBNP, cTnI, and CK-MB levels in the observation group decreased compared with those in the control group. This result suggested that sacubitril–valsartan may be strongly effective in aiding the restoration of damaged myocardium. This enhanced efficacy might be attributed to the selective action of valsartan on angioetnsin II type1 (ATI) receptors and its thorough blockade of the reninan giotensin aldosterone system (RAAS) system, which inhibits the release of inflammatory factors and prevents secondary myocardium damage caused by reperfusion [28–30].

This study investigated the occurrence of adverse cardiovascular events in patients, and results revealed that patients who underwent sacubitril–valsartan treatment had a lower probability of occurrence of such events and demonstrated higher safety than their counterparts. Sacubitril–valsartan improves the cardiac function of patients through multiple mechanisms. It not only has short-term effects on blood circulation but also has long-term effects on local tissues, helping reduce the workload of the heart and keeping patients beneficial in the cardiovascular event chain. Moreover, it can inhibit vascular factors and norepinephrine, enhance renal blood flow, and decrease aldosterone secretion. This mechanism ultimately leads to a reduction in blood pressure and a decrease in the workload on the patient's heart. It has a long-term and steady therapeutic effect and can be used extensively. However, this study still had some limitations. The size of sample size was too small, and the study period was too short. The study results still need large-scale clinical trials for further confirmation.

## Conclusion

This study demonstrated a significant therapeutic effect and high safety in the application of sacubitril–valsartan after PCI in patients with ACS and hypertension. The use of sacubitril–valsartan remarkably improved cardiac function and reduced myocardial damage-related indicators. Additionally, it decreased the occurrence of adverse cardiovascular events. These findings have implications of great importance for the widespread adoption and application of sacubitril–valsartan, as it can improve the efficacy of treatment and enhance the life quality of patients with ACS and hypertension.

## Availability of Data and Materials

The original contributions presented in the study are included in the article/supplementary material. Further inquiries can be directed to the corresponding authors.

## Author Contributions

FL: Conception, Design, Materials, Analysis, Literature Review, Writing. JL: Conception, Design, Materials, Analysis, Literature Review, Writing. YD: Data

Collection, Analysis, Literature Review, Writing. YY: Supervision, Data Collection, Analysis, Writing, Critical Review. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work to take public responsibility for appropriate portions of the content and agreed to be accountable for all aspects of the work in ensuring that questions related to its accuracy or integrity.

## Ethics Approval and Consent to Participate

This study has been approved by the Ethics Committee of The First People's Hospital of Jiashan (Approval No.:2024041). All participants included in this study gave informed consent.


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## Conflict of Interest

The authors declare no conflict of interest.

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