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Analysis of the Curative Effect of Sarkubactrovalsartan in the Treatment of Patients with Acute Anterior Myocardial Infarction after PCI

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Abstract: *Objective*: To study the clinical value of sacubatravalsartan in patients with acute anterior myocardial infarction (AAMI) after percutaneous coronary intervention (PCI). *Methods*: 124 patients with AAMI from April 2022 to June 2024 were randomly divided into study group (56 cases) and control group (68 cases). Both groups were given aspirin during the treatment, the control group was given enalapril after operation, and the study group was given sacubatravalsartan after operation, both of which were treated for 2 months. Before and after treatment, CRP, IL-6, TNF - α, Hcy, BNP and other serological indicators were detected, HR, LVEDd, LVEF and other cardiac function indexes were detected, and postoperative complications and adverse drug reactions were compared between the two groups. *Results*: before treatment, there was no significant difference in each index level between the two groups ($P > 0.05$); after treatment, CRP, IL-6, TNF - α, Hcy, BNP, HR and LVEDd in the study group were lower than those in the control group ($P < 0.05$), and LVEF was higher than that in the control group ($P < 0.05$); the incidence of postoperative complications in the study group was lower than that in the control group ($P < 0.05$); there was no significant difference in the total adverse reaction rate between the two groups ($P > 0.05$). Conclusion: sacubatravalsartan can effectively improve the cardiac function of patients with AAMI after PCI, reduce the incidence of postoperative complications, and has high safety.

Keywords: Acute anterior myocardial infarction; PCI; Sarkubatrovalsartan; Cardiac function

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1. Introduction

Acute anterior myocardial infarction (AAMI) is a common critical emergency in cardiovascular medicine, characterized by its sudden onset and high risk. If not treated promptly, it can be life-threatening [1]. The clinical treatment of AAMI mainly focuses on anticoagulation and thrombolysis, with both medications and surgery showing certain efficacy. Among them, percutaneous coronary intervention (PCI) can directly clear and dilate the blocked coronary arteries, thereby alleviating coronary obstruction and reducing patients' clinical symptoms [2]. However, various complications can occur after surgery, so patients need to be treated with medication after PCI

to improve surgical efficacy. Studies have pointed out that the dual-acting angiotensin receptor complex inhibitor, Sacubitril/Valsartan, has a good therapeutic effect on AAMI patients after PCI, which can improve patients' clinical symptoms and reduce postoperative complications [3]. Currently, there is no unified view on the scientific nature and effectiveness of Sacubitril/Valsartan for AAMI patients after surgery. Based on this, this study adopts Sacubitril/Valsartan treatment for AAMI patients after PCI, aiming to provide a reference for clinical medication.

2. Materials and methods

2.1. General information

A total of 124 AAMI patients admitted to the cardiology department of our hospital from April 2022 to April 2024 were included as research subjects. They were divided into a study group (56 cases) and a control group (68 cases) using a random number table method. Control group: 39 males and 29 females; aged 58–74 years old, with an average age of (64.55 ± 3.69) years old; New York Heart Disease Association (NYHA) cardiac function classification [4]: 28 cases in grade II, 40 cases in grade III. Study group: 32 males and 24 females; aged 59–77 years old, with an average age of (65.22 ± 3.80) years old; NYHA cardiac function classification: 25 cases in grade II, 31 cases in grade III. This study was approved by the medical ethics committee of the hospital. There were no statistically significant differences in general information (gender, age, cardiac function classification, etc.) between the two groups ($P > 0.05$), indicating comparability.

Inclusion criteria: (1) Meet the diagnostic criteria for AAMI in "Emergency Treatment of Acute Myocardial Infarction" [5], and satisfy any two of the following criteria: (1) Asymptomatic or presence of sudden severe chest pain, fever, nausea, arrhythmia, abdominal distension, and other symptoms of varying degrees; (2) Dynamic evolution of ST and ST-T visible on electrocardiogram; (3) Creatine kinase isoenzyme (CK-MB) ≥ 6.3 ng/mL and troponin I (cTnl) ≥ 0.5μg/L; (4) History of ischemic chest pain; (5) Stable basic vital signs, able to walk and move limbs without impediment within 5 meters; (6) All patients underwent PCI surgery in our hospital; (7) Agreed to sign the informed consent form for this study.

Exclusion criteria: (1) Patients with severe liver, kidney, or other organ dysfunction; (2) Patients with hypotension, electrolyte imbalance, and severe endocrine system diseases; (3) Patients with severe consciousness disorders, affective disorders, expression disorders, and history of mental illness; (4) Patients allergic to contrast agents or medications; (5) NYHA cardiac function class ≥ IV.

2.2. Methods

Both groups were given aspirin (Bayer Healthcare, J20130078, 100mg/tablet) 4 days before PCI surgery, at a dose of 300 mg/day preoperatively and 100mg/day postoperatively as a maintenance dose. The control group was given an angiotensin-converting enzyme inhibitor (ACEI) - enalapril (Yangzijiang Pharmaceutical, H32026567, 10 mg/tablet) postoperatively, at a dose of 5 mg per time, twice a day with warm water. The study group was given sacubitril/valsartan (Novartis Pharma, J20171054, 100 mg/tablet) postoperatively, starting at a dose of 100 mg per time, twice a day. After 2 weeks of medication, the dose was adjusted to 150 mg per time, twice a day and maintained for another 2 weeks. Then, the dose was increased to 200mg per time, twice a day until the end of treatment. One month was considered as one course of treatment, and both groups were treated continuously for two courses.

2.3. Observation indicators

(1) Biological indicators

Fasting elbow venous blood (5 mL) was collected from the subjects before and after treatment. C-reactive

protein (CRP) was detected using immunoturbidimetry, and interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), homocysteine (Hcy), and brain natriuretic peptide (BNP) were detected using enzyme-linked immunosorbent assay. The analysis was performed using a SIEMENS ASVIA1800 fully automated biochemical analyzer. CRP and TNF-α reagents were produced by Thermo Fisher Scientific, while IL-6, Hcy, and BNP reagents were produced by Ningbo Meikang Biological Co., Ltd.

(2) Cardiac function indicators

Before and after treatment, the subjects' cardiac function indicators, including left ventricular ejection fraction (LVEF) and left ventricular end-diastolic dimension (LVEDD), were measured using a Mindray DC-N3S Doppler color ultrasound diagnostic instrument. Heart rate (HR) was detected using a Zoncare iE90 electrocardiograph.

(3) Postoperative complications

The occurrence of postoperative complications after PCI was compared between the two groups, which may include arrhythmia, heart failure, vascular bleeding, vascular hematoma, thrombus formation, contrast-induced nephropathy, slow coronary flow, coronary occlusion, coronary perforation, etc. The total complication rate was calculated as the sum of all complications divided by the total number of patients × 100%.

(4) Adverse reactions

Adverse reactions during treatment were recorded in both groups, which may include cough, nausea, abdominal pain, fever, dizziness, gastrointestinal bleeding, hyperkalemia, hypotension, renal failure, etc. The total adverse event rate was calculated as the sum of all adverse events divided by the total number of patients × 100%.

2.4. Statistical methods

The statistical data in this study were processed using SPSS 22.0 software. Count data and measurement data were represented by *n* (%) and mean ± standard deviation (SD), respectively, and were tested using chi-square (χ^2) and *t*-test, respectively. Statistical significance was determined at $P < 0.05$.

3. Results

3.1. Serological indicators

There were no statistically significant differences in the levels of various indicators between the two groups before treatment ($P > 0.05$). After treatment, the levels of CRP, IL-6, TNF-α, Hcy, and BNP in the study group were lower than those in the control group ($P < 0.05$). See **Table 1** for details.

Table 1. Comparison of serological indicators before and after treatment between the two groups (mean ± SD)

Group	*n*	Time	CRP (mg/L)	IL-6 (pg/mL)	TNF-α (pg/mL)	Hcy (μmol/L)	BNP (pg/L)
Study group	56	Before treatment	8.13 ± 0.95	36.47 ± 10.97	36.75 ± 11.58	12.25 ± 1.82	1232.21 ± 61.85
		After treatment	4.27 ± 0.23*#	24.85 ± 4.69*#	22.88 ± 6.61*#	6.36 ± 0.86*#	523.59 ± 49.32*#
Control group	68	Before treatment	8.55 ± 1.04	35.05 ± 11.12	35.43 ± 11.32	11.96 ± 1.90	1229.36 ± 61.67
		After treatment	6.27 ± 0.95*#	30.52 ± 6.23*#	28.52 ± 8.33*#	8.59 ± 0.95*#	682.46 ± 52.58*#

Note: *$P < 0.05$ compared to the same group before and after treatment; #$P < 0.05$ compared to the other group at the same time point.

3.2. Cardiac function indicators

There were no statistically significant differences in the levels of various indicators between the two groups before treatment ($P > 0.05$). After treatment, the HR and LVEDD in the study group were lower than those in the control group ($P < 0.05$), while the LVEF was higher than that in the control group ($P < 0.05$). See **Table 2** for details.

Table 2. Comparison of cardiac function indicators before and after treatment between the two groups (mean ± SD)

Group	*n*	LVEF (%)		HR (beats/min)		LVEDD (mm)	
		Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Study group	56	33.21 ± 5.97	43.51 ± 4.96*	114.22 ± 11.11	73.32 ± 8.65*	54.09 ± 4.72	45.20 ± 3.80*
Control group	68	33.45 ± 6.11	38.23 ± 4.63*	113.88 ± 10.98	79.66 ± 10.23*	54.76 ± 4.32	50.23 ± 3.60*
t		0.194	5.430	0.152	3.353	0.536	5.901
P		0.846	0.000	0.879	0.001	0.593	0.000

Note: *$P < 0.05$ compared to the same group before and after treatment.

3.3. Postoperative complications

The total incidence of postoperative complications in the study group was lower than that in the control group ($P < 0.05$). See **Table 3** for details.

Table 3. Comparison of postoperative complications after PCI between the two groups [*n*, (%)]

Group	*n*	Arrhythmia, *n* (%)	Heart failure, *n* (%)	Vascular hematoma, *n* (%)	Thrombosis, *n* (%)	Total complications, *n* (%)
Study group	56	2 (3.57)	1 (1.79)	1 (1.79)	1 (1.79)	5 (8.93)
Control group	68	6 (8.82)	3 (4.41)	3 (4.41)	4 (5.88)	16 (23.53)
χ^2		1.153[a]	0.312[a]	0.312[a]	1.149[a]	6.375
P		0.283	0.577	0.577	0.284	0.012

Note: a represents the continuity correction χ^2 value.

3.4. Adverse drug reactions

There was no statistically significant difference in the total incidence of adverse reactions between the two groups during treatment ($P > 0.05$). See **Table 4** for details.

Table 4. Comparison of adverse reactions during treatment between the two groups [*n*, (%)]

Group	*n*	Nausea	Diarrhea	Cough	Dizziness	Hypotension	Total adverse reactions
Study Group	56	2 (5.00)	1 (2.50)	2 (5.00)	2 (5.00)	1 (2.50)	8 (20.00)
Control Group	68	3 (5.00)	2 (3.33)	2 (3.33)	4 (6.67)	2 (3.33)	13 (21.67)
χ^2		0.105[a]	0.005[a]	0.056[a]	0.041[a]	0.005[a]	0.085
P		0.746	0.943	0.813	0.840	0.943	0.771

Note: [a] represents the continuity correction χ^2 value.

4. Discussion

AAMI is a severe cardiovascular disease with high morbidity and mortality rates. The direct cause of this disease is abnormal heart function resulting from the death of myocardial cells due to ischemia and hypoxia caused by changes in coronary blood flow [6]. The pathogenesis of AAMI is complex and can be caused by abnormalities in blood, blood vessels, and hemodynamics, leading to narrowing and blockage of cerebral arteries. Modern medical research has confirmed that various factors such as systemic diseases, overexertion, hypertension, hyperlipidemia, high cholesterol, and imbalance in dietary structure can all cause coronary artery blockage or rupture, thereby triggering AAMI [7]. Currently, clinical treatment for AAMI mainly focuses on thrombolysis, improving blood circulation in the coronary arteries, and reducing the risk of embolism. Commonly used drugs include tirofiban, aspirin, beta blockers, etc., and surgical treatment is primarily PCI. Although these treatments can provide good efficacy and rapidly relieve patients' conditions, PCI surgery can easily affect the blood circulation and microenvironment balance of the coronary arteries, leading to various complications such as coronary spasms, perforations, occlusions, contrast-induced nephropathy, and bleeding. Therefore, clinical treatment often requires medication after PCI surgery to reduce the impact of complications on surgical efficacy [8].

Qian *et al.* [9] pointed out that CRP is a protein that regulates the body's immune function. It can enhance the body's immune response by strengthening the phagocytosis of phagocytic cells. Its expression level in the blood rises sharply during the initial stage of immune enhancement, making it a useful indicator for the early diagnosis of various diseases. IL-6 is a cytokine that acts between immune cells. Its level increases rapidly when the body experiences an inflammatory response, and an increase in inflammatory symptoms can burden the heart and elevate the risk of microcirculatory disorders [10]. TNF-α is a proinflammatory cytokine produced by macrophages in the human body. Excessive amounts of TNF-α can be detected in the blood of AAMI patients due to the activation of inflammatory states [11]. Hcy is an intermediate metabolite of sulfur-containing amino acids in the body. It is often used as an independent risk factor for cardiovascular and cerebrovascular diseases, and its expression level in the blood is closely related to the body's microcirculation [12]. BNP is mainly produced by ventricular myocytes and has effects such as promoting urination and sodium excretion, dilating blood vessels, and resisting vasoconstriction. Its expression level increases with the severity of cardiac function damage [13]. The results of this study showed that after treatment, the levels of CRP, IL-6, TNF-α, Hcy, and BNP in the study group were lower than those in the control group, which is similar to the results reported by Xiong *et al.* [14]. This suggests that sacubitril/valsartan can effectively reduce the risk of coronary thrombosis and embolism in AAMI patients after PCI surgery. This may be due to sacubitril/valsartan's dual action as an angiotensin receptor complex inhibitor, which has a better blocking effect on the renin-angiotensin-aldosterone system (RAAS) than ACEI drugs. This further inhibits the process of myocardial fibrosis and thickening while reducing the expression levels of related cytokines, thereby lowering the expression of various indicators in the patients' blood. The results also showed that after treatment, the HR and LVEDD in the study group were lower than those in the control group, while the LVEF was higher. The total incidence of postoperative complications was also lower in the study group compared to the control group, which is consistent with the findings of Chen *et al.* [15], further supporting the effectiveness of sacubitril/valsartan. Additionally, there was no statistically significant difference in the incidence of adverse reactions between the two groups during treatment, indicating that sacubitril/valsartan not only provides better treatment effects than ACEI drugs but also has high safety.

5. Conclusion

In summary, sacubitril/valsartan can effectively improve cardiac function levels in AAMI patients after PCI surgery, reduce the incidence of postoperative complications, and has high safety, making it clinically valuable for promotion.

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Disclosure statement

The author declares no conflict of interest.

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