Research Paper

Comparison of Efficacy and Safety of Sacubitril/Valsartan and Perindopril in Acute Myocardial Infarction Complicated by Post Surgery Heart Failure

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The purpose of this study is to explore the applicability and efficacy of sacubitril/valsartan in individuals with post-percutaneous coronary intervention heart failure subsequent to acute myocardial infarction. 106 individuals with verified acute myocardial infarction diagnosis who underwent percutaneous coronary intervention surgery were selected and divided into two groups with 53 patients in each group. Along with conventional therapy, control group was treated with perindopril, while observation group was treated with sacubitril/valsartan. Before and after treatment, color Doppler ultrasound was harnessed to measure various cardiac function indicators and multifunctional immunoassay analyzer was used to measure myocardial injury biomarkers. Before and after treatment, hemodynamic parameters, encompassing blood pressure, heart rate and the average hourly urine output over a 24 h period, were subjected to comparative analysis between the two groups. Moreover, the evaluation of patients' quality of life was carried out utilizing the Kansas City cardiomyopathy questionnaire, while clinical effectiveness, occurrence of adverse cardiovascular events and readmission within 3 mo of treatment was documented. Before treatment, there is no difference in cardiac function indicators, myocardial injury biomarkers, blood pressure, heart rate, and urine output between the two groups ($p>0.05$). However, after treatment, the observation group exhibited lower levels of cardiac function indicators, myocardial injury biomarkers, heart rate and higher levels of left ventricular fractional shortening and left ventricular ejection fraction, systolic and diastolic blood pressure and more urine output compared to the control group ($p<0.05$). The observation group exhibited elevated Kansas City cardiomyopathy questionnaire scores, higher clinical effectiveness and reduced readmission rate compared to the control group. No statistically significant variances ($p>0.05$) were detected in the incidence of adverse reactions between the two groups. Compared to treatment with perindopril, the use of sacubitril/valsartan can effectively treat acute myocardial infarction complicated with post-percutaneous coronary intervention heart failure.

Key words: Acute myocardial infarction, percutaneous coronary intervention surgery, heart failure, sacubitril/valsartan

The manifestation of Acute Myocardial Infarction (AMI) arises due to myocardial necrosis triggered by acute/persistent ischemia and hypoxia affecting the coronary arteries. In severe cases, it may be followed by Heart Failure (HF) and even sudden cardiac death[1-3]. AMI features a high incidence, multiple complications, high mortality rate and poor prognosis. Currently, the clinical treatment of AMI mainly focuses on Percutaneous Coronary Intervention (PCI) surgery and oral medication, while severe cases may require Coronary Artery Bypass Graft surgery (CABG)[4,5]. However, due to myocardial reperfusion following the restoration of blood flow, patients may experience abnormal ventricular diastolic and systolic function, with severe cases developing HF[6]. HF is a syndrome characterized by circulatory dysfunction resulting from impaired myocardial diastolic or systolic function leading to insufficient cardiac output[7]. Previous studies have disclosed that when HF occurs, inadequate perfusion of the

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body's tissues and organs occurs due to the heart's inability to effectively pump blood. This will cause organ dysfunction and hemodynamic abnormalities in various organs of the body, as the tissues are unable to meet their normal metabolic demands. Importantly, HF complicating AMI after PCI is a major contributor to postoperative mortality[8,9]. Therefore, actively seeking efficacious drugs for the treatment of post-PCI HF is of great significance for improving patient prognosis and promoting their overall recovery.

According to reports, sacubitril/valsartan is the first dual inhibitor of angiotensin II receptors and neprilysin. It not only possesses the natriuretic peptide system with cardio protective effects but also obstructs the excessive activation of the renin-angiotensin-aldosterone system. It is a class I medication recommended in HF guidelines. It boasts several functions like promoting natriuretic and diuresis, vasodilation, lowering blood pressure, preventing and reversing ventricular remodeling, and ultimately delaying the progression of HF[10-12]. Currently, sacubitril/valsartan has made remarkable progress in clinical application for the treatment of patients with HF. However, there is limited research on the efficacy and prognosis of sacubitril/valsartan in patients with AMI undergoing emergency PCI and complicated by HF. Hence, this study aims to explore the therapeutic effects of sacubitril/valsartan and perindopril in patients with AMI complicated by HF following PCI, hoping to provide some diagnostic and treatment strategies.

MATERIALS AND METHODS

General information:

A total of 106 patients diagnosed with AMI and undergoing PCI were selected from our hospital's admissions between May 2020 and February 2023. The age range of all patients was 37 y to 90 y, with 75 male and 31 female patients. Among them, 62 patients had anterior myocardial infarction, and 14 patients had inferior wall myocardial infarction. Through the implementation of distinct treatment protocols, the patients were arbitrarily distributed into two groups. The control group (n=53) subjected to perindopril treatment, and the observation group (n=53) subjected tp sacubitril/valsartan therapy. General data of all patients were compared, and no statistically significant variances were observed ($p>0.05$), denoting comparability between the groups, as displayed in Table 1.

Inclusion criteria:

Patients with AMI complicated by post-PCI HF were classified according to the Killip classification criteria[13,14]. This research encompassed patients classified as cardiac function Class II (left HF with <50 % lung field rales) and Class III (acute pulmonary edema with rhonchi, rales, dry rales, and moist rales throughout the lung fields). Plasma N-Terminal pro-B-Type Natriuretic Peptide (NT-proBNP) levels were ≥400 pg/ml and all participants received approval from our hospital's ethics committee.

TABLE 1: COMPARISON OF GENERAL DATA BETWEEN THE TWO GROUPS (n, %) ($\bar{x}\pm s$)

Characteristics	Variables	Observation group (n=53)	Control group (n=53)	t/χ^2	p
Gender	Male	35 (66.04)	40 (75.47)	1.14	0.286
	Female	18 (33.96)	13 (24.53)		
Age (years old)	-	48.45±12.65	47.49±11.84	0.403	0.688
Body mass index (kg/m2)	-	24.33±3.96	24.47±4.28	0.175	0.862
Patients with comorbidities	Comorbid hyperlipidemia	26	21	5.061	0.080
	Comorbid hypertension	30	46		
	Comorbid type 2 diabetes	23	16		
Number of stents	0-1	27	30	0.787	0.675
	2	10	7		
	3-4	4	5		
Smoking history	-	17	9	3.262	0.071

Exclusion criteria:

Patients with allergies to relevant medications such as perindopril and sacubitril/valsartan used in the treatment; patients classified as Killip Class IV (cardiogenic shock) in HF; patients with comorbidities such as liver or kidney disease, bone marrow infection, malignant tumors, immune system disorders, hematological disorders, cardiac amyloidosis, or hyperthyroid heart disease; patients with pregnancy or under lactation; patients with a history of mental illness; patients with HF or cardiogenic shock caused by other factors and patients who had undergone CABG or coronary angioplasty procedures are excluded from the study.

Treatment methods:

All patients received standardized Dual Antiplatelet Therapy (DAPT) with atorvastatin and rosuvastatin, and all patients were treated with low-molecular-weight heparin sodium. Vasoactive drugs such as nitroglycerin and epinephrine, as well as diuretics like hydrochlorothiazide and furosemide, were used as conventional treatments based on hemodynamic performance. DAPT such as clopidogrel and ticagrelor were administered depending on the presence or absence of contraindications to antiplatelet medications.

Control group: Along with the conventional treatment regimen, patients were subjected to perindopril therapy (produced by Haisco Pharmaceutical (Meishan) Co., Ltd., National Drug Approval Number: H20203507, dosage: 4 mg).

Observation group: Apart from conventional treatment, patients were given sacubitril/valsartan (produced by Beijing Novartis Pharma Ltd., National Drug Approval Number: J20171054) as a replacement for enalapril. The initial dosage was 50 mg per dose, orally administered twice daily, and the dosage was doubled every 2 w based on the patient's condition and tolerability. Nevertheless, it is imperative to ensure that the dosage does not surpass 200 mg per administration, taken twice daily.

Observation of indicators:

Cardiac function parameters: Before and after treatment, the IU22 color Doppler ultrasound system was employed to gauge the Left Ventricular End-Diastolic Diameter (LVEDD), Left Ventricular End-Systolic Diameter (LVESD), Left Ventricular Fractional Shortening (LVFS), Interventricular Septal Thickness (IVST) at Diastole, Left Ventricular Mass Index (LVMI), and Left Ventricular Ejection Fraction (LVEF) in both groups of patients. These metrics were employed to evaluate the alterations in both the structural and functional attributes of the left ventricle. The measurements were obtained according to established guidelines and standards for echocardiographic assessment. To discern dissimilarities in these metrics between the two groups prior to and following treatment, a statistical analysis was conducted.

Myocardial injury functional indicators: Before and after treatment, a multifunctional immune assay instrument was used to determine the myocardial injury markers including serum NT-proBNP, Cardiac Troponin I (cTnI), and Creatine Kinase-Myocardial Band (CK-MB).

Hemodynamics and urine output: The STAR8000E model general bedside electrocardiographic monitor provided by Comen Medical was harnessed to monitor blood pressure and heart rate. Measurements were taken on the day of admission (prior to receiving systemic treatment) and on the following day after 4 w of treatment (after waking up and before bedtime). Patient systolic and diastolic blood pressures, as well as heart rate, were observed and recorded, with their respective averages being used as the daily statistical data. Additionally, the 24 h average hourly urine output was measured and recorded for both the groups of patients.

Assessment of quality of life and clinical efficacy: The Kansas City Cardiomyopathy Questionnaire (KCCQ) was utilized to evaluate the quality of life of both the groups after receiving different pharmacological treatments[15]. This questionnaire comprises five dimensions, which includes physical limitation, clinical symptoms, social function, self-perception, and psychological status, with a total of 23 items. The comprehensive score spans from 0 to 100, with elevated scores denoting a superior quality of life experienced by the patients. The clinical efficacy was evaluated by the following criteria, which includes marked improvement indicates heart rate decreases to the normal range, <100 beats/min during activity; HF symptoms are essentially eliminated, and cardiac function recovers to ≤Class II. Effective indicates heart rate decreases but remains >100 beats/min during activity, accompanied by persistent latent HF and cardiac function at Class II-III. Ineffective indicates no improvement in HF symptoms or death.

Overall effective rate=((Number of marked improvement patients+Number of effective patients)/ Total number of patients)×100 %.

Cardiovascular adverse events and readmission rate: The occurrence of adverse events, such as angina, dry cough, hypotension, hyperkalemia, and dyspnea, and the status of readmission were observed and recorded during the 3 mo follow-up period after PCI in both patient groups.

Statistical analysis:

For the purpose of data processing and analysis in this study, Corporation Statistical Package for the Social Sciences (SPSS) 20.0 software (SPSS Inc., Chicago, IL, United States of America (USA) was used. Measurement data were represented as mean±standard deviation ($\bar{x}\pm s$) and t-test were adopted for comparisons between two groups. Enumeration data were presented as n, %, with Chi-square (χ^2) employed for comparisons. The variance of repeated measurement data was taken to compare relevant indicators at different time points between the patient groups. A significance level of $p<0.05$ was deemed to be statistically significant.

RESULTS AND DISCUSSION

The outcomes denoted that there were no statistically significant variances ($p>0.05$) in cardiac function parameters such as LVEDD, LVESD, and LVFS between the two patient groups prior to treatment. However, after treatment, the observation group displayed lower levels of LVEDD, LVESD, IVST, and LVMI and higher levels of LVFS and LVEF compared to the control group ($p<0.05$). These findings demonstrated that administering sacubitril/valsartan was beneficial for improving cardiac function in patients with myocardial infarction complicated by post-PCI HF as shown in Table 2.

As the results revealed, no statistically significant variances ($p>0.05$) were observed in the levels of NT-proBNP, cTnI, CK-MB, and other myocardial injury markers between the two groups prior to treatment. However, subsequent to the administration of treatment, the observation group exhibited diminished levels of NT-proBNP, cTnI, CK-MB, and various other markers in contrast to the control group ($p<0.05$). This suggested that administering sacubitril/valsartan was capable of ameliorating myocardial damage in patients with myocardial infarction complicated by post-PCI HF as shown in Table 3.

No statistically significant differences ($p>0.05$) were discovered in blood pressure, heart rate, and urine output between the two groups prior to treatment. When comparing the post-treatment measurements with the pre-treatment measurements within each group, it was confirmed that the patients showed an increase in systolic and diastolic blood pressure, a decrease in heart rate, and an increase in urine output ($p<0.05$). Furthermore, the observation group exhibited higher levels of systolic and diastolic blood pressure, lower heart rate, and greater urine output compared to the control group following treatment ($p<0.05$). These discoveries demonstrated that the use of sacubitril/valsartan stabilized hemodynamic parameters in patients with myocardial infarction complicated by post-PCI HF as shown in Table 4.

TABLE 2: COMPARISON OF CARDIAC FUNCTION PARAMETERS BEFORE AND AFTER TREATMENT IN THE TWO GROUPS ($\bar{x}\pm s$)

Parameters	Treatment	Observation group (n=53)	Control group (n=53)	t	p
LVEDD (mm)	Before	66.49±6.89	66.56±6.87	0.052	0.958
	After	56.47±5.86	61.45±6.57	4.118	<0.001
LVESD (mm)	Before	44.54±4.88	43.78±4.77	0.811	0.419
	After	35.78±3.79	40.13±4.38	5.468	<0.001
LVFS (%)	Before	18.11±1.91	17.44±1.94	1.792	0.076
	After	26.47±2.83	20.44±2.26	12.121	<0.001
IVST (mm)	Before	9.61±0.99	9.67±0.94	0.32	0.750
	After	8.56±0.88	9.13±0.96	3.186	0.002
LVMI (g/m^2)	Before	125.37±13.33	124.45±13.11	0.358	0.721
	After	113.76±12.44	121.18±13.45	2.948	0.004
LVEF (%)	Before	30.45±4.36	30.85±4.15	0.484	0.630
	After	38.56±4.28	33.19±4.06	6.627	<0.001

TABLE 3: COMPARISON OF MYOCARDIAL INJURY MARKERS BETWEEN THE TWO GROUPS (x̄±s)

Parameters	Treatment	Observation group (n=53)	Control group (n=53)	t	p
NT-proBNP (pg/l)	Before	3342.56±336.68	3336.37±339.43	0.094	0.925
	After	586.45±60.34	1684.45±17.55	127.204	<0.001
cTnI (ng/ml)	Before	5.17±1.16	5.14±1.13	0.135	0.893
	After	0.86±0.18	1.41±0.22	14.086	<0.001
CK-MB (U/l)	Before	91.54±10.22	91.78±10.29	0.12	0.904
	After	31.34±3.68	39.13±4.16	10.211	<0.001

TABLE 4: COMPARISON OF HEMODYNAMICS AND URINE OUTPUT BEFORE AND AFTER TREATMENT BETWEEN THE TWO GROUPS (x̄±s)

Parameters	Treatment	Observation group (n=53)	Control group (n=53)	t	p
Systolic pressure (mmHg)	Before	58.67±6.12	59.34±5.77	0.58	0.563
	After	89.45±9.14	80.23±8.35	5.422	<0.001
Diastolic pressure (mmHg)	Before	30.49±3.45	31.56±3.55	1.574	0.119
	After	53.42±5.95	62.81±6.72	7.616	<0.001
Heart rate (beats/min)	Before	114.23±12.26	115.78±12.59	0.642	0.522
	After	82.47±8.44	92.16±9.47	5.561	<0.001
Urine output (ml/h)	Before	18.25±1.95	18.42±1.98	0.445	0.657
	After	42.35±4.67	31.42±3.69	13.369	<0.001

The data obtained from our analysis revealed no statistically substantial deviations ($p>0.05$) in KCCQ scores between the two groups prior to the commencement of pharmacological intervention. This outcome indicated that the baseline quality of life was comparably equivalent between the two cohorts. However, subsequent to treatment with different medications, it was revealed that the observation group had higher KCCQ scores compared to the control group($p<0.05$). Additionally, upon evaluating the clinical effectiveness between the two groups, the observation group showcased a total effective rate of 96.23 %, surpassing the 84.90 % observed in the control group ($p<0.05$). The above findings confirmed that administering sacubitril/valsartan enhanced the quality of life in myocardial infarction patients with post-PCI HF, demonstrating better therapeutic effects as shown in Table 5.

As indicated by our data, the observation group went through adverse reactions including hypotension, hyperkalemia, dizziness, headache, angina, and dyspnea, with an adverse reaction rate of 15.09 %. In the control group, adverse reactions such as hypotension, hyperkalemia, dizziness, headache, angina, and dyspnea were observed, with an adverse reaction rate of 20.75 %. The comparison of adverse reaction rates between the two groups following treatment showed no statistically significant variance ($p>0.05$). Moreover, the readmission rate in the observation group was 5.66 %, whereas in the control group it was 18.87 % ($p<0.05$). These outcomes demonstrated that within 3 mo subsequent to treatment, the incidence of cardiovascular adverse events was similar between patients treated with perindopril and those treated with sacubitril/valsartan. Nonetheless, the readmission rate was lower in patients treated with sacubitril/valsartan, indicating a better prognosis as shown in Table 6.

AMI, occurring on the basis of coronary arterial atherosclerosis, involves the occurrence of erosion or plaque rupture in the patient's coronary arteries, leading to increased platelet aggregation, which further triggers the formation of a large amount of thrombus within the coronary arteries, thereby damaging the patient's myocardial structure and metabolic capacity, and reducing cardiac function. This poses a grave threat to the patient's life safety[16-18]. At present, in clinical treatment, PCI has shown relatively favorable effects. Postoperatively, a combination of cardiotonic and diuretic agents, as well as measures to control ventricular rate and other anti-HF strategies, can efficaciously control HF symptoms in most patients. Nevertheless, routine interventions fail to effectively block the excessive activation of neuroendocrine caused by HF. Therefore, there is still a gap between the treatment

effect and clinical expectations[19,20]. Research has exhibited that this is mainly related to the inability of angiotensin-converting enzyme inhibitors to block the production of angiotensin II through non-angiotensin-converting enzyme pathways[21]. Thus, targeting this issue specifically is of paramount significance in improving the disease progression of patients with AMI plus post-surgery HF.

Reportedly, sacubitril/valsartan is composed of two drugs, sacubitril and valsartan. The latter selectively acts on angiotensin receptors, blocking angiotensin II, thereby effectively blocking the renin-angiotensin-aldosterone system. Simultaneously, sacubitril suppresses neprilysin, reducing myocardial load and further improving myocardial remodeling[22,23]. To dig deeper into the specific value of sacubitril/valsartan in patients with AMI and post-surgery HF, this research retrospectively analyzed the clinical data of 106 patients who received either perindopril or sacubitril/valsartan for the treatment of AMI, with relevant indicators recorded. Studies have unveiled that cardiac color ultrasound can dynamically observe the internal structure, ejection function and blood flow in the heart. Parameters such as LVEDD, LVESD, LVFS, IVST, and LVEF can well reflect cardiac function[24,25]. Our study discovered that these indicators are improved in the observation group subsequent to treatment, and the degree of improvement was greater in the observation group *vs.* the control group. This suggested that sacubitril/valsartan exerted a good effect on myocardial structure and ejection function, and its remodeling effect was superior to perindopril. This may be due to the inhibitory effect of sacubitril on neprilysin, which cooperates with valsartan to boost myocardial remodeling[26]. Additionally, Zhang *et al.*[27] research has uncovered that sacubitril/valsartan can limit myocardial cell hypertrophy (including the border zone) and interstitial fibrosis, while increasing the profile of Vascular Endothelial Growth Factor (VEGF), thereby ameliorating myocardial perfusion and perfusion reserve in the infarcted area and improving heart function, which was aligned with our study.

As reported, NT-proBNP, cTnI, and CK-MB are important indicators for evaluating heart function. Elevated levels of these indicators reflect myocardial damage and are positively correlated with cardiac insufficiency[28,29]. Fan *et al.* has revealed that sacubitril/valsartan can attenuate myocardial injury, reduce the size of myocardial infarction, and dampen myocardial fibrosis, further supporting the protective effect of sacubitril/valsartan against myocardial infarction[30,31]. Encouragingly, our study also discovered that following treatment, both groups presented a decrease in the levels of serological indicators NT-proBNP, cTnI, and CK-MB, with the observation group exhibiting even lower levels compared to the control group. This hinted that sacubitril/valsartan could more efficaciously restore damaged myocardium. The reason for this may be that valsartan selectively acts on angiotensin receptors, blocking the renin-angiotensin-aldosterone system more thoroughly, impeding the release of inflammatory factors, and avoiding secondary myocardial damage from reperfusion[32-34].

Additionally, this research unveiled that comparison to the control group, the observation group displayed elevated levels of systolic and diastolic blood pressure, reduced heart rate, and augmented urine output prior to the treatment. These variances were regarded as statistically significant. Furthermore, there was no remarkable difference in the occurrence of cardiovascular adverse events within 3 mo between the two groups, but the readmission rate was lower in the sacubitril/valsartan group.

TABLE 5: COMPARISON OF QUALITY OF LIFE AND CLINICAL EFFICACY BEFORE AND AFTER TREATMENT IN THE TWO GROUPS OF PATIENTS (x̄±s)

Parameters	Treatment	Observation group (n=53)	Control group (n=53)	t	p
KCCQ scores	Pre	61.45±6.59	62.58±6.87	0.864	0.39
	Post	72.56±7.66	82.45±8.55	6.997	<0.001
Clinical efficacy	Marked improvement	23 (43.40)	20 (37.74)	-	-
	Effective	28 (52.83)	25 (47.17)	-	-
	Ineffective	2 (3.77)	8 (15.09)	-	-
	Total effective rate	51 (96.23)	45 (84.90)	3.975	0.046

TABLE 6: COMPARISON OF CARDIOVASCULAR ADVERSE EVENTS AND READMISSION WITHIN 3 MO OF TREATMENT BETWEEN THE TWO GROUPS (n, %)

Parameters	Various reactions	Observation group (n=53)	Control group (n=53)	χ^2	p
Adverse reactions (cases)	Dry cough	2 (3.77)	2 (3.77)	-	-
	Hypotension	3 (5.66)	1 (1.89)	-	-
	Hyperkalemia	1 (1.89)	2 (3.77)	-	-
	Dizziness and headache	1 (1.89)	2 (3.77)	-	-
	Angina	1 (1.89)	2 (3.77)	-	-
	Dyspnea	0 (0.00)	2 (3.77)	-	-
Overall incidence of adverse reactions	-	8 (15.09)	11 (20.75)	0.577	0.447
Readmission (cases)	-	3 (5.66)	10 (18.87)	4.296	0.038

The observation group also displayed higher KCCQ scores and better clinical efficacy in contrast to the control group. This denoted that sacubitril/valsartan was beneficial for stabilizing hemodynamic parameters in patients with post-PCI HF following AMI, and it was safer and more effective than perindopril in improving prognosis, enhancing patients' quality of life, and lowering the risk of readmission[35,36]. Wang *et al.* study has also revealed that sacubitril/valsartan has a dual-target regulatory mechanism. In addition to its similar mechanism of action to angiotensin-converting enzyme inhibitors or angiotensin, it can also repress neprilysin, heighten the level of cyclic guanosine monophosphate in the body, and have good diuretic and vasodilatory effects, thereby alleviating the patient's condition[37-39], which is consistent with the conclusion of our work.

To summarize, sacubitril/valsartan as an adjunctive therapy for post-AMI HF can efficaciously improve patient's hemodynamics and cardiac function, abate myocardial damage, substantially augment urine output, improve therapeutic efficacy and quality of life, and lower the rate of readmission in the short term, without serious adverse reactions. These findings have confirmed that sacubitril/valsartan can be capitalized as a treatment for patients with HF following AMI. Nevertheless, it should be noted that this study is based on a small sample size, and there may be varying degrees of selection and information bias in the process of patient collection and follow-up. Further studies will be conducted to address these limitations.

In contrast with the use of perindopril, sacubitril/valsartan in treating AMI patients with HF subsequent to PCI can effectively improve cardiac function, mitigate myocardial damage, ameliorate hemodynamics, enhance living quality and clinical efficacy, and lower the readmission rate. Therefore, it is worthwhile to promote and apply it in clinical settings.

Conflict of interests:

The authors declared no conflict of interests.

REFERENCES

1. Pollard TJ. The acute myocardial infarction. Prim Care 2000;27(3):631-49.
2. Kapur NK, Thayer KL, Zweck E. Cardiogenic shock in the setting of acute myocardial infarction. Methodist DeBakey Cardiovasc J 2020;16(1):16-21.
3. Zeymer U. Diagnosis and initial management of acute myocardial infarction. MMW Fortschr Med 2019;161(4):34-6.
4. Hoole SP, Bambrough P. Recent advances in percutaneous coronary intervention. Heart 2020;106(18):1380-6.
5. Xiao M, Li Y, Guan X. Community-based physical rehabilitation after percutaneous coronary intervention for acute myocardial infarction. Texas Heart Inst J 2021;48(2):1-4.
6. Berezin AE, Berezin AA. Adverse cardiac remodelling after acute myocardial infarction: Old and new biomarkers. Dis Markers 2020;2020:1-21.
7. Thiele H, Ohman EM, de Waha-Thiele S, Zeymer U, Desch S. Management of cardiogenic shock complicating myocardial infarction: An update 2019. Eur Heart J 2019;40(32):2671-83.
8. Orrem HL, Nilsson PH, Pischke SE, Grindheim G, Garred P, Seljeflot I, *et al.* Acute heart failure following myocardial infarction: Complement activation correlates with the severity of heart failure in patients developing cardiogenic shock. ESC Heart Fail 2018;5(3):292-301.
9. Zhu Y, Zhang JL, Yan XJ, Sun L, Ji Y, Wang FF. Effect of dapagliflozin on the prognosis of patients with acute myocardial infarction undergoing percutaneous coronary intervention. Cardiovasc Diabetol 2022;21(1):1-10.
10. Docherty KF, Vaduganathan M, Solomon SD, McMurray JJ. Sacubitril/valsartan: Neprilysin inhibition 5 years after

PARADIGM-HF. Heart Fail 2020;8(10):800-10.
11. Heyse A, Manhaeghe L, Mahieu E, Vanfraechem C, van Durme F. Sacubitril/valsartan in heart failure and end-stage renal insufficiency. ESC Heart Fail 2019;6(6):1331-3.
12. Singh JS, Burrell LM, Cherif M, Squire IB, Clark AL, Lang CC. Sacubitril/valsartan: Beyond natriuretic peptides. Heart 2017;103(20):1569-77.
13. Milwidsky A, Greidinger D, Frydman S, Hochstadt A, Ifrach-Kashtan N, Mizrachi M, *et al.* Echocardiographic Killip classification. J Am Soc Echocardiogr 2022;35(3):287-94.
14. Ramonfaur D, Hinojosa-González DE, Paredes-Vázquez JG. Killip-Kimball classification in octogenarians with acute coronary syndrome: An 11-year experience. Arch Cardiol Mex 2022;92(4):425-30.
15. Spertus JA, Jones PG, Sandhu AT, Arnold SV. Interpreting the Kansas City Cardiomyopathy Questionnaire in clinical trials and clinical care: JACC state-of-the-art review. J Am Coll Cardiol 2020;76(20):2379-90.
16. Mizutani Y, Ishikawa T, Nakahara S, Taguchi I. Treatment of young women with acute myocardial infarction. Intern Med 2021;60(2):159-60.
17. Sandoval Y, Jaffe AS. Type 2 myocardial infarction: JACC review topic of the week. J Am Coll Cardiol 2019;73(14):1846-60.
18. Bajaj A, Sethi A, Rathor P, Suppogu N, Sethi A. Acute complications of myocardial infarction in the current era: Diagnosis and management. J Investig Med 2015;63(7):844-55.
19. Saito Y, Tateishi K, Kanda M, Shiko Y, Kawasaki Y, Kobayashi Y, *et al.* Volume-outcome relationships for percutaneous coronary intervention in acute myocardial infarction. J Am Heart Assoc 2022;11(6):1-13.
20. Lee MS, Dahodwala MQ. Percutaneous coronary intervention for acute myocardial infarction due to unprotected left main coronary artery occlusion: Status update 2014. Catheter Cardiovasc Interv 2015;85(3):416-20.
21. Schafer A, Konig T, Bauersachs J, Akin M. Novel therapeutic strategies to reduce reperfusion injury after acute myocardial infarction. Curr Problems Cardiol 2022;47(12):1-19.
22. Xiong B, Nie D, Qian J, Yao Y, Yang G, Rong S, *et al.* The benefits of sacubitril-valsartan in patients with acute myocardial infarction: A systematic review and meta-analysis. ESC Heart Fail 2021;8(6):4852-62.
23. Shah AM, Claggett B, Prasad N, Li G, Volquez M, Jering K, *et al.* Impact of sacubitril/valsartan compared with ramipril on cardiac structure and function after acute myocardial infarction: The PARADISE-MI echocardiographic sub-study. Circulation 2022;146(14):1067-81.
24. Gatto L. Does sacubitril/valsartan work in acute myocardial infarction? The PARADISE-AMI study. Eur Heart J Suppl 2021;23(Suppl E):E87-90.
25. An J, Du Y, Li X, Bao Q, Guo Y, Song Y, *et al.* Myocardial protective effect of sacubitril-valsartan on rats with acute myocardial infarction. Perfusion 2022;37(2):208-15.
26. Liu L, Ding X, Han Y, Lv J. Effects and safety of sacubitril/valsartan for patients with myocardial infarction: A systematic review and meta-analysis. J Healthcare Eng 2022;2022:1-8.
27. Zhang J, Ding L, Zhao Y, Sun W, Chen B, Lin H, *et al.* Collagen-targeting vascular endothelial growth factor improves cardiac performance after myocardial infarction. Circulation 2009;119(13):1776-84.
28. Fan J, Ma J, Xia N, Sun L, Li B, Liu H. Clinical value of combined detection of CK-MB, MYO, cTnI and plasma NT-proBNP in diagnosis of acute myocardial infarction. Clin Lab 2017;63(3):427-33.
29. Shen T, Yang X, Zhang Z. Positive relationship of RDW with NT-proBNP and cTnI in acute myocardial infarction patients. Clin Lab 2022;68(6).
30. Wang Y, Dai L, Wang N, Zhu Y, Chen M, Wang H. Rapid rule out of acute myocardial infarction in the observe zone using a combination of presentation N-terminal pro-B-type natriuretic peptide and high-sensitivity cardiac troponin I. Clin Biochem 2019;70:34-8.
31. Fan G, Zhou C, Hou T, Li X, Wang L, Wang C. Effects of sacubitril/valsartan on cardiac function, blood biochemistry and clinical efficacy in early ventricular remodeling after acute myocardial infarction. Biotechnol Genetic Eng Rev 2023;39(1):1-6.
32. Zhao C, Guo Y, Wang Y, Wang L, Yu L, Liang Y, *et al.* The efficacy and safety of Sacubitril/Valsartan on pulmonary hypertension in hemodialysis patients. Front Med 2022;9:1-8.
33. Kario K. The sacubitril/valsartan, a first-in-class, angiotensin receptor neprilysin inhibitor (ARNI): Potential uses in hypertension, heart failure, and beyond. Curr Cardiol Rep 2018;20(1):1-8.
34. Mann DL, Greene SJ, Givertz MM, Vader JM, Starling RC, Ambrosy AP, *et al.* Sacubitril/valsartan in advanced heart failure with reduced ejection fraction: Rationale and design of the LIFE trial. Heart Fail 2020;8(10):789-99.
35. Solomon SD, Vaduganathan ML, Claggett B, Packer M, Zile M, Swedberg K, *et al.* Sacubitril/valsartan across the spectrum of ejection fraction in heart failure. Circulation 2020;141(5):352-61.
36. Kido K, Colvin BM, Szymanski TW, Guglin M. Sacubitril/valsartan off-label uses for heart failure. J Cardiac Fail 2022;28(7):1185-201.
37. Williams B, Cockcroft JR, Kario K, Zappe DH, Brunel PC, Wang Q, *et al.* Effects of sacubitril/valsartan *vs.* olmesartan on central hemodynamics in the elderly with systolic hypertension: The PARAMETER study. Hypertension 2017;69(3):411-20.
38. Kuang H, Huang X, Zhou Z, Cheng X, Xu G. Sacubitril/valsartan in chronic kidney disease: From pharmacological mechanism to clinical application. Eur J Pharmacol 2021;907:1-6.
39. Wang Y, Zhou R, Lu C, Chen Q, Xu T, Li D. Effects of the angiotensin-receptor neprilysin inhibitor on cardiac reverse remodeling: Meta-analysis. J Am Heart Assoc 2019;8(13):1-40.

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