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Original Article

Outcome effects of enalapril with or without bisoprolol in patients with acute myocardial infarction

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Abstract: Objective: This study was designed to determine the effects of enalapril combined with bisoprolol on patients with acute myocardial infarction (AMI) and their prognosis. Methods: This study retrospectively analyzed data of 104 patients receiving AMI treatment in the First People's Hospital of Shanghai from May 2019 to October 2021, including 48 patients treated with enalapril alone (control group) and 56 patients treated with enalapril combined with bisoprolol (observation group). The efficacy, adverse reactions, cardiac function [left ventricular ejection fraction (LVEF), left ventricular end-diastolic diameter (LVED), left ventricular end-systolic diameter (LVES) and left ventricular mass (LVM)] of the two groups were measured and analyzed. The patients were followed up for one year to compare their prognosis. Results: The observation group showed a significantly higher total response rate than the control group ($P < 0.05$), but the incidence of adverse reactions was not different significantly between the two groups ($P > 0.05$). After treatment, LVES, LVED and LVEF increased significantly in both groups ($P < 0.05$), and the observation group showed significantly lower LVES and LVM levels and had a higher LVEF level than the control group ($P < 0.05$). The follow-up results revealed no significant difference in prognosis and survival between the two groups ($P > 0.05$). Conclusion: Enalapril combined with bisoprolol is effective and safe in the treatment of AMI, because this regimen can effectively improve patients' cardiac function.

Keywords: Enalapril, bisoprolol, acute myocardial infarction, heart function, prognosis

Introduction

Acute myocardial infarction (AMI), which is triggered by coronary atherosclerosis-induced continuous ischemia and hypoxia, causes myocardiocyte necrosis [1]. That is, blood lipids are deposited in the coronary arteries for a long time and form plaques, and the rupture of plaques leads to secondary thrombosis and platelet adhesion. Platelet adhesion to blood vessels, in turn, causes insufficiency of distal myocardial blood supply and then myocardial injury or death [2]. According to the survey statistics over the past years, the incidence of AMI is about 40%, and the mortality and incidence of this disease are increasing annually [3]. According to statistics, since 2012, rural areas have shown a higher mortality from AMI than urban areas. In 2016, 58.69 people per 100,000 people in urban areas died from AMI, while 74.72 people per 100,000 people in rural areas died from it. Clinically, AMI patients are often experience severe retrosternal pain, arrhythmia, circulatory decline or heart failure. The current treatments for AMI are primarily based on drugs and surgery, and the main purpose is to restore myocardial blood perfusion, save dying myocardium, prevent expansion of infarction and narrow the scope of myocardial ischemia, so as to protect and maintain cardiac function [4]. However, the prognosis of patients after surgical treatment is still not satisfactory. Accordingly, it is necessary to explore new therapeutic drugs or regimens for AMI.

As an angiotensin-converting enzyme inhibitor (ACEI), enalapril can effectively inhibit angiotensin-converting enzymes and lower the content of angiotensin II. Thus, it can lower blood pressure and heart load, with a long-lasting effect [5, 6]. Yokota et al. [7] found that enalapril could reduce the level of serum myocardial injury

index and improve cardiac function in patients with AMI, with obvious therapeutic benefit. It possesses remarkable efficacy in hypertension and heart failure, as well as in AMI. As a β1-adrenoceptor blocker, bisoprolol can be adopted to treat diseases including hypertension, coronary heart disease and heart failure, and it can effectively lower the risk of recurrence of myocardial ischemia and arrhythmia [8, 9]. Jun et al. [10] revealed a favorable effect of bisoprolol on secondary prevention of patients with AMI.

However, the efficacy of the combination of two drugs on myocardial infarction is still in need of investigation. Accordingly, we used both enalapril and bisoprolol for patients with AMI to explore the efficacy and prognosis of the combined regimen, with the purpose of providing a basis for clinical practice.

Methods and data

Clinical data

A total of 104 AMI patients treated in the First People's Hospital of Shangqiu from May 2019 to October 2021 were collected and analyzed retrospectively. Among them, 48 patients treated with enalapril alone were assigned to a control group, and 56 patients treated with enalapril combined with bisoprolol were assigned to an observation group. This study was approved by the Medical Ethics Committee of the First People's Hospital of Shangqiu.

Inclusion and exclusion criteria

Inclusion criteria: Patients with detailed clinical data, patients who met the WHO diagnostic criteria of AMI [11], and patients without mental disorder and cognitive dysfunction.

Exclusion criteria: Patients who had taken statins or β-blockers before admission, patients with low compliance, patients with a history of drug allergy or physical organic diseases, patients with coagulation dysfunction, patients with chronic myocardial infarction or cardiac insufficiency, and pregnant women.

Therapeutic regimens

All patients were given routine treatment such as oxygen inhalation and sedation to ensure their absolute bed rest. Also, patients were given aspirin, clopidogrel, atorvastatin calcium tablets, low molecular weight heparin, and nitrates.

The control group was treated with oral enalapril-folic acid tablets (Shenzhen Ausa Pharmaceutical Co., Ltd., State Food and Drug Administration (SFDA) approval number: H20103783), based on routine treatments, with a dose of 5-10 mg, twice a day.

The observation group was treated with oral bisoprolol fumarate tablets (Beijing Huasu Pharmaceutical, SFDA approval number: H10-970082) based on the treatments in the control group, with a dosage of 2.5-5.0 mg, 4 times a day. The adverse drug reactions in the two groups were closely observed.

Detection of cardiac function indexes

Before therapy and after 6 months of therapy, a Philips IE33 ultrasonic diagnostic instrument was adopted to measure the cardiac function indexes of patients, including left ventricular ejection fraction (LVEF), left ventricular end-diastolic diameter (LVED), left ventricular end-systolic diameter (LVES) and left ventricular mass (LVM).

Follow-up

The patients' electronic medical records and outpatient reexamination records within one year were collected, and the patients' survival was analyzed.

Outcome measures

Primary outcome measures: The efficacies (total response rate) in the two groups were compared. The clinical efficacy was evaluated according to the following criteria: Markedly effective: Arrhythmia disappeared, and patients had significant alleviation in myocardial infarction symptoms including hypotension, chest tightness, and chest pain, with normal electrocardiogram (ECG) results. Effective: Preventricular contraction decreased by > 60%, and myocardial infarction symptoms were alleviated. Ineffective: Preventricular contraction decreased by < 60%, and myocardial infarction symptoms were not alleviated or even tended to worsen. Total response rate = [(the number of cases with markedly effective response + the number of cases with effective response)/

Table 1. Comparison of baseline data

Factor	Control group (n=48)	Observation group (n=56)	χ^2	*P* value
Age			0.003	0.951
≤ 60 years old (n=56)	26	30		
> 60 years old (n=48)	22	26		
Sex			0.165	0.684
Male	29	36		
Female	19	20		
BMI			0.015	0.903
≤ 24 kg/m^2	22	25		
> 24 kg/m^2	26	31		
Course of disease			5.246	0.022
≤ 5 h	15	30		
> 5 h	33	26		
Past medical history				
Hypertension	12	12	0.186	0.667
Diabetes mellitus	8	8	0.113	0.737
Hyperlipemia	8	14	1.076	0.299
History of smoking			0.431	0.512
Yes	36	45		
No	12	11		

BMI, body mass index.

total number of cases] * 100%. The cardiac function of patients before and after therapy was compared, and the changes in LVEF, LVED, and LVES before and after therapy were evaluated. The 1-year survival of patients was counted, and the prognostic factors were analyzed through regression.

Secondary outcome measures: The drug treatment of the two groups was compared. The incidence of adverse reactions including cough, hypotension, and sinus bradycardia, was compared between the two groups. The total adverse reaction rate = (total number of cases with adverse reactions/total number of cases) * 100%. The ECG indexes of the two groups were compared, including Q wave time and ST segment offset.

Statistical analyses

This study adopted SPSS 20.0 (SPSS Inc., Chicago, IL, USA) for analyses of collected data, and GraphPad Prism 8 for data visualization. The Kolmogorov-Smirnov test was performed to evaluate the normal distribution. Data in a normal distribution were described by mean ± SD, and analyzed using the t test. Inter-group comparisons and intra-group comparisons were conducted using the independent sample t test and paired t test, respectively. Classified variables were compared by the chi-square test. Logistic regression was conducted to analyze risk factors for cardiovascular adverse events. P < 0.05 was considered a significant difference.

Results

Comparison of clinical data

Comparison of clinical data revealed no significant difference between the two groups in age, sex, body mass index, course of disease, cardiac function classification, previous medical history or smoking history (P > 0.05, **Table 1**).

Efficacy analysis

According to a comparison of efficacy between the two groups, the control group showed a significantly lower total response rate than the observation group (P=0.004, **Table 2**).

Changes of cardiac function before and after therapy

The cardiac function indexes (LVEF, LVED, LVES, and LVM) of the two groups were analyzed and compared before and after therapy. According to the results, before therapy, LVEF, LVED, LVES, and LVM levels were not greatly different between the two groups, while after therapy, LVEF, LVED, LVES, and LVM levels in both groups increased greatly. Additionally, after therapy, the observation group showed a significantly higher LVEF level and significantly lower LVES and LVM levels than the control group, but the LVED level was not greatly different between the two groups (**Figure 1**).

Changes in ECG indexes before and after treatment

The ECG indexes (Q wave time and ST segment offset) before and after treatment were compared between the two groups. The results revealed that the two groups were not different in Q-wave time and ST-segment offset before treatment (P > 0.05), but after treatment, the

Table 2. Evaluation of clinical efficacy

Group	Markedly effective	Effective	Ineffective	Total response rate (%)
Control group (n=48)	25 (52.08)	11 (22.91)	11 (22.91)	36 (75.00%)
Observation group (n=56)	33 (58.92)	20 (35.71)	33 (58.92)	53 (94.64%)
χ^2 value				8.080
P value				0.004

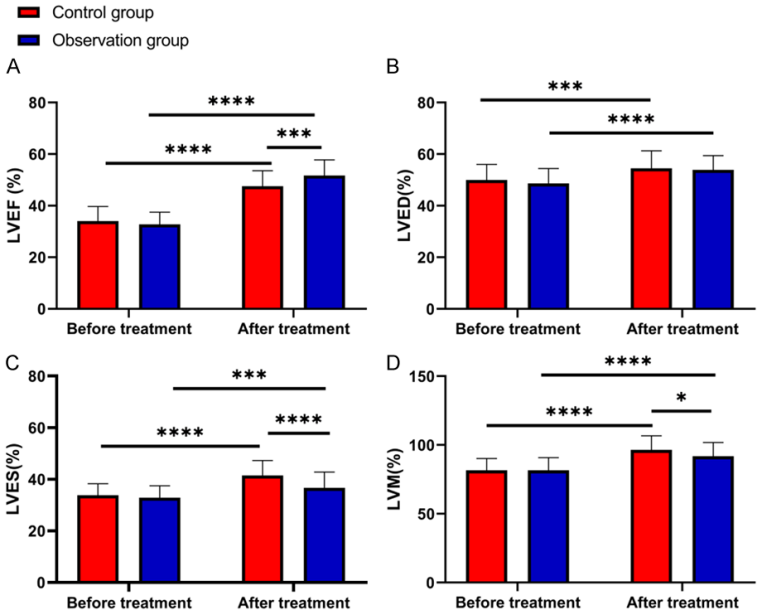

Figure 1. Changes of cardiac function in patients before and after treatment. A. Comparison of left ventricular ejection fraction (LVEF) changes before and after treatment. B. Comparison of left ventricular end-diastolic diameter (LVED) changes before and after treatment. C. Comparison of left ventricular end-systolic diameter (LVES) changes before and after treatment. D. Comparison of left ventricular mass (LVM) changes before and after treatment. Note: *P < 0.05; **P < 0.01; ***P < 0.001; ***P < 0.0001.

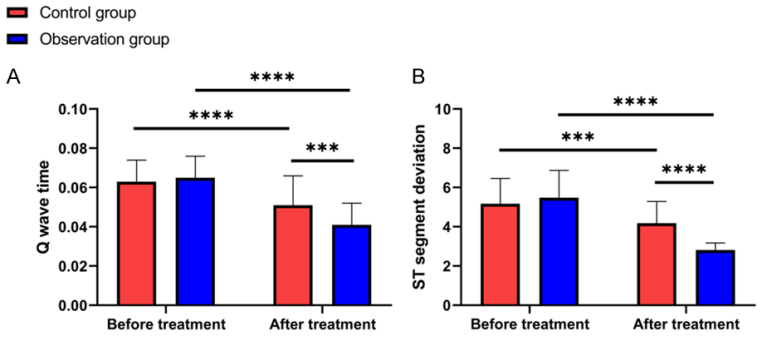

Figure 2. Changes in ECG indexes before and after treatment. A. Changes in Q-wave time before and after treatment. B. Changes in ST segment offset before and after treatment. Note: electrocardiogram (ECG).

Q-wave time and ST-segment offset of the two groups decreased significantly (P < 0.001), with a shorter Q-wave time and lower ST-segment offset in the observation group than in the control group (P < 0.001, **Figure 2**).

Comparison of adverse reactions before and after therapy

According to the comparison of adverse reactions between the groups, the overall incidence of adverse reactions in the control group was not statistically different from that of the observation group (P= 0.740, **Table 3**).

Analysis of factors impacting patients' prognosis

According to the occurrence of adverse cardiovascular events after 6 months of treatment, the patients were grouped into an unfavorable prognosis group (n=26) and a favorable prognosis group (n=78). The clinical data of these two groups were compared. According to the results, age, course of disease, diabetes mellitus (DM), and therapeutic regimen were risk factors for adverse events (**Table 4**). Further Logistic regression analysis revealed that age, course of disease, DM and therapeutic regimen were independent risk factors for cardiovascular adverse events (**Table 5**).

Discussion

Lifestyle changes have accentuated the threat of acute myocardial infarction (AMI) to human

Table 3. Comparison of adverse reactions

Group	Number of cases	Cough	Hypotension	Rash	Sinus bradycardia	Total incidence (%)
Control group	48	2 (4.16)	2 (4.16)	0 (0.00)	1 (2.08)	5 (10.42)
Observation group	56	2 (3.57)	3 (5.35)	1 (1.78)	1 (1.78)	7 (12.50)
χ^2 value						0.110
P value						0.740

Table 4. Univariate analysis

Factor	Unfavorable prognosis group (n=26)	Favorable prognosis group (n=78)	χ^2	*P* value
Age			7.429	0.006
≤ 60 years old (n=56)	20	36		
> 60 years old (n=48)	6	42		
Sex			0.341	0.558
Male (n=65)	15	50		
Female (n=39)	11	28		
BMI			0.634	0.425
≤ 24 kg/m^2 (n=47)	10	37		
> 24 kg/m^2 (n=57)	16	41		
Course of disease			8.161	0.004
≤ 5 h (n=45)	5	40		
> 5 h (n=59)	21	38		
Past medical history				
Hypertension (n=24)	4	20	1.156	0.282
Diabetes mellitus (n=16)	11	5	19.303	< 0.0001
Hyperlipidemia (n=22)	5	17	0.076	0.781
History of smoking			1.507	0.219
Yes (n=81)	18	63		
No (n=23)	8	15		
Therapeutic regimen			13.206	0.0003
Single treatment (n=48)	20	28		
Combined treatment (n=56)	6	50		

BMI, body mass index.

Table 5. Analysis of risk factors

Factors	B	S.E.	Wald	Sig.	Exp (B)	95% C.I. of EXP (B)	
						Lower limit	Upper limit
Age	1.238	0.609	4.14	0.042	3.45	1.046	11.375
Course of disease	-1.485	0.642	5.352	0.021	0.226	0.064	0.797
Diabetes mellitus	2.691	0.764	12.401	0	14.75	3.298	65.968
Therapeutic regimen	1.574	0.647	5.922	0.015	4.824	1.358	17.13

health [12]. People with hypertension, hyperglycemia or obesity face a higher risk of AMI, which often occurs without warning and may be accompanied by pain, chest tightness or a near-death feeling [13]. In recent years, great progress has been achieved in the surgical treatment of AMI. Percutaneous coronary intervention (PCI) is an important method to treat AMI. It can quickly unblock the obstructed blood vessels caused by thrombosis, restore blood circulation and oxygen supply, thus effectively reduce the mortality [14-16]. American

College of Cardiology points out that an good effect can be achieved by PCI within one and a half hours from the time of admission to balloon dilatation [17, 18]. However, prior research has revealed that patients may still suffer heart failure and other adverse reactions after PCI, and may not achieve an optimal therapeutic effect or prognostic improvement.

Hypertension is an important factor giving rise to the onset of AMI, so preventing the occurrence of hypertension is also a primary method to prevent AMI [19]. Enalapril, one of antihypertensive drugs, is an ACEI that can dilate blood vessels and lower blood pressure [20]. According to previous studies [21, 22], enalapril can effectively reduce vasoconstriction and cardiac load, and inhibit myocardial remodeling in rats with AMI and left ventricular function reconstruction, thus effectively alleviating the development of the disease and reducing mortality. As a new β-blocker, bisoprolol can not only protect cardiovascular function, but also reduce blood pressure by inhibiting an overactivated sympathetic nerve. Prior research [23, 24] has revealed that bisoprolol can promote the decrease of CRP expression in patients with AMI.

According to previous studies [25, 26], the combination of β-blockers and ACEI drugs can be beneficial in the treatment of AMI. Additionally, research about CIBISI has revealed that β-blockers combined with ACEI drugs can lower the mortality of patients with myocardial infarction, and effectively alleviate their heart failure and improve prognosis. In the present study, the observation group treated with enalapril combined with bisoprolol showed a higher LVEF level and LVM levels than the control group, but the LVED levels of the two groups were similar, indicating that the combined treatments greatly improved the patients' cardiac function, and delivered higher efficacy. According to analysis of the mechanism, bisoprolol can inhibit an inflammatory reaction and activate the expression of receptors, thus reducing the expression of CRP. β-blockers can be effective in relieving arrhythmia to some extent, increasing myocardial responsiveness and ventricular contractile function, reducing peripheral vascular resistance, alleviating the symptoms of angina pectoris, and improving the prognosis.

In this study, the observation group showed significantly higher treatment efficacy than the control group, suggesting that enalapril combined with bisoprolol strengthened heart function and improved coronary artery perfusion capacity, delivering a good therapeutic effect, but this did not lower the incidence of adverse reactions in the course of treatment. Enalapril, an ACEI drug, can effectively inhibit the release of angiotensin II, dilate blood vessels, increase blood flow in infarcted areas, restore blood supply and strengthen ventricular systolic function. In addition, bisoprolol, as a highly selective β-blocker, has a long-term effect on cardiac function, and can prolong ventricular diastolic period and improve the blood supply capacity, thus effectively recovering myocardial function and improving prognosis. Research by Huang et al. [27] showed that enalapril combined with bisoprolol delivered a higher efficacy and improved the cardiac function and oxidative stress, similar to findings in this study.

Finally, this study analyzed risk factors for adverse cardiovascular events after therapy. Accordingly, logistic regression analysis showed that age, course of disease, DM, and therapeutic regimen were independent risk factors for cardiovascular adverse events. Similar to the results of the present study, a prior study [28] showed that diabetic patients with unsatisfactory blood glucose control faced a greatly higher risk of coronary artery disease, and DM would affect arterioles to cause diseases, increasing factors of AMI and affecting the prognosis. Therefore, the above indexes before therapy can serve as reference indexes for evaluating the occurrence of adverse cardiovascular events in patients.

This study has confirmed through analysis that enalapril combined with bisoprolol can improve the cardiac function, therapeutic effect and prognosis of patients with AMI. However, this study still has some limitations. First, this study is a retrospective study, with a limited sample size, so the samples are not as uniform as those in randomized controlled experiments. Secondly, the patients cannot be further followed up, so the long-term prognosis of the patients remains unclear. Therefore, we hope to carry out more experiments and follow-up in the future, so as to improve the research conclusions.

In summary, enalapril combined with bisoprolol is effective and safe in the treatment of AMI, because the combined regimen can substantially improve patients' cardiac function and has a high clinical application value.

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Effect of blood lipid gene polymorphism on blood lipid level in east henan (No. LHGJ20-210988).

Disclosure of conflict of interest

None.

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References

[1] Reed GW, Rossi JE and Cannon CP. Acute myocardial infarction. Lancet 2017; 389: 197-210.

[2] Katayama Y, Tanaka A, Taruya A, Kashiwagi M, Nishiguchi T, Ozaki Y, Shiono Y, Shimamura K, Kitabata H, Kubo T, Hozumi T, Ishida Y, Kondo T and Akasaka T. Increased plaque rupture forms peak incidence of acute myocardial infarction in winter. Int J Cardiol 2020; 320: 18-22.

[3] Johansson S, Rosengren A, Young K and Jennings E. Mortality and morbidity trends after the first year in survivors of acute myocardial infarction: a systematic review. BMC Cardiovasc Disord 2017; 17: 53.

[4] DeFilippis AP, Chapman AR, Mills NL, de Lemos JA, Arbab-Zadeh A, Newby LK and Morrow DA. Assessment and treatment of patients with type 2 myocardial infarction and acute noninschemic myocardial injury. Circulation 2019; 140: 1661-1678.

[5] Velazquez EJ, Morrow DA, DeVore AD, Duffy CI, Ambrosy AP, McCague K, Rocha R and Braunwald E; PIONEER-HF Investigators. Angiotensin-neprilysin inhibition in acute decompensated heart failure. N Engl J Med 2019; 380: 539-548.

[6] Badae NM, El Naggar AS and El Sayed SM. Is the cardioprotective effect of the ACE2 activator diminazene aceturate more potent than the ACE inhibitor enalapril on acute myocardial infarction in rats? Can J Physiol Pharmacol 2019; 97: 638-646.

[7] Yokota T, Osanai T, Hanada K, Kushibiki M, Abe N, Oikawa K, Tomita H, Higuma T, Yokoyama J, Hanada H and Okumura K. Effects of telmisartan on markers of ventricular remodeling in patients with acute myocardial infarction: comparison with enalapril. Heart Vessels 2010; 25: 460-468.

[8] Kim HK, Lim KS, Kim SS and Na JY. Impact of bisoprolol on ventricular arrhythmias in experimental myocardial infarction. Chonnam Med J 2021; 57: 132-138.

[9] Chen C, Wu X, Li Y and Peng Y. Study on the application effect of bisoprolol combined with sacubitril valsartan sodium tablets in the cardiac rehabilitation of patients with acute myocardial infarction combined with left heart failure after percutaneous coronary intervention (PCI). Ann Palliat Med 2021; 10: 5455-5461.

[10] Jun SJ, Kim KH, Jeong MH, Kim MC, Sim DS, Hong YJ, Kim JH, Cho MC, Chae JK, Park HS, Park JS and Ahn YK. Effects of bisoprolol are comparable with carvedilol in secondary prevention of acute myocardial infarction in patients undergoing percutaneous coronary intervention. Chonnam Med J 2018; 54: 121-128.

[11] Ge Z, Baber U, Claessen BE, Farhan S, Chandrasekhar J, Li SX, Sartori S, Kini AS, Rao SV, Weiss S, Henry TD, Vogel B, Sorrentino S, Faggioni M, Kapadia S, Muhlestein B, Strauss C, Toma C, DeFranco A, Effron MB, Keller S, Baker BA, Pocock S, Dangas G and Mehran R. The prevalence, predictors and outcomes of guideline-directed medical therapy in patients with acute myocardial infarction undergoing PCI, an analysis from the PROMETHEUS registry. Catheter Cardiovasc Interv 2019; 93: E112-E119.

[12] Gulati R, Behfar A, Narula J, Kanwar A, Lerman A, Cooper L and Singh M. Acute myocardial infarction in young individuals. Mayo Clin Proc 2020; 95: 136-156.

[13] Yan J, Zhou J, Huang J, Zhang H, Deng Z and Du Y. The outcomes of acute myocardial infarction patients comorbidity with hypertension and hyperhomocysteinemia. Sci Rep 2021; 11: 22936.

[14] Tibaut M, Mekis D and Petrovic D. Pathophysiology of myocardial infarction and acute management strategies. Cardiovasc Hematol Agents Med Chem 2017; 14: 150-159.

[15] Ozaki Y, Hara H, Onuma Y, Katagiri Y, Amano T, Kobayashi Y, Muramatsu T, Ishii H, Kozuma K, Tanaka N, Matsuo H, Uemura S, Kadota K, Hikichi Y, Tsujita K, Ako J, Nakagawa Y, Morino Y, Hamanaka I, Shiode N, Shite J, Honye J, Matsubara T, Kawai K, Igarashi Y, Okamura A, Ogawa T, Shibata Y, Tsuji T, Yajima J, Iwabuchi K, Komatsu N, Sugano T, Yamaki M, Yamada S, Hirase H, Miyashita Y, Yoshimachi F, Kobayashi M, Aoki J, Oda H, Katahira Y, Ueda K, Nishino

M, Nakao K, Michishita I, Ueno T, Inohara T, Kohsaka S, Ismail TF, Serruys PW, Nakamura M, Yokoi H and Ikari Y; Task Force on Primary Percutaneous Coronary Intervention (PCI) of the Japanese Cardiovascular Interventional Therapeutics (CVIT). CVIT expert consensus document on primary percutaneous coronary intervention (PCI) for acute myocardial infarction (AMI) update 2022. Cardiovasc Interv Ther 2022; 37: 1-34.

[16] Marquis-Gravel G, Zeitouni M, Kochar A, Jones WS, Sketch MH Jr, Rao SV, Patel MR and Ohman EM. Technical consideration in acute myocardial infarction with cardiogenic shock: a review of antithrombotic and PCI therapies. Catheter Cardiovasc Interv 2020; 95: 924-931.

[17] Damluji AA, van Diepen S, Katz JN, Menon V, Tamis-Holland JE, Bakitas M, Cohen MG, Balsam LB and Chikwe J; American Heart Association Council on Clinical Cardiology; Council on Arteriosclerosis, Thrombosis and Vascular Biology; Council on Cardiovascular Surgery and Anesthesia; and Council on Cardiovascular and Stroke Nursing. Mechanical complications of acute myocardial infarction: a scientific statement from the American Heart Association. Circulation 2021; 144: e16-e35.

[18] Mehta LS, Beckie TM, DeVon HA, Grines CL, Krumholz HM, Johnson MN, Lindley KJ, Vaccarino V, Wang TY, Watson KE and Wenger NK; American Heart Association Cardiovascular Disease in Women and Special Populations Committee of the Council on Clinical Cardiology, Council on Epidemiology and Prevention, Council on Cardiovascular and Stroke Nursing, and Council on Quality of Care and Outcomes Research. Acute myocardial infarction in women: a scientific statement from the American Heart Association. Circulation 2016; 133: 916-947.

[19] Carrick D, Haig C, Maznyczka AM, Carberry J, Mangion K, Ahmed N, Yue May VT, McEntegart M, Petrie MC, Eteiba H, Lindsay M, Hood S, Watkins S, Davie A, Mahrous A, Mordi I, Ford I, Radjenovic A, Welsh P, Sattar N, Wetherall K, Oldroyd KG and Berry C. Hypertension, microvascular pathology, and prognosis after an acute myocardial infarction. Hypertension 2018; 72: 720-730.

[20] Velazquez EJ, Morrow DA, DeVore AD, Ambrosy AP, Duffy CI, McCague K, Hernandez AF, Rocha RA and Braunwald E. Rationale and design of the comParIson Of sacubitril/valsartaN versus Enalapril on Effect on nt-pRo-bnp in patients stabilized from an acute Heart Failure episode (PIONEER-HF) trial. Am Heart J 2018; 198: 145-151.

[21] Yokota T, Tomita H, Mori Y, Kudo T, Hiraga H, Suto N, Higuma T, Abe N, Hanada H, Osanai T and Okumura K. Imidapril and enalapril similarly inhibit plasma matrix metalloproteinase activities and attenuate left ventricular remodeling in patients with acute myocardial infarction. J Cardiovasc Pharmacol 2014; 63: 528-532.

[22] Diao K, Wang D, Chen Z, Wu X, Ma M, Zhu Y, Zhang L, Wang H, Wang M, He S, Li C, Deng Q, Yan T, Wu T, Tang L, Huang B, Sun J and He Y. Rationale and design of a multi-center, prospective randomized controlled trial on the effects of sacubitril-valsartan versus enalapril on left ventricular remodeling in ST-elevation myocardial infarction: the PERI-STEMI study. Clin Cardiol 2021; 44: 1709-1717.

[23] Chen RZ, Liu C, Zhou P, Tan Y, Sheng ZX, Li JN, Zhou JY, Wu Y, Yang YM, Song L, Zhao HJ and Yan HB. Associations between postprocedural D-dimer, hs-CRP, LDL-C levels and prognosis of acute myocardial infarction patients treated by percutaneous coronary intervention. Zhonghua Xin Xue Guan Bing Za Zhi 2020; 48: 359-366.

[24] Wang Y. The efficacy and safety of bisoprolol in the treatment of myocardial infarction with cardiac insufficiency. Comput Math Methods Med 2022; 2022: 3098726.

[25] Nordenskjold AM, Agewall S, Atar D, Baron T, Beltrame J, Bergstrom O, Erlinge D, Gale CP, Lopez-Pais J, Jernberg T, Johansson P, Ravn-Fisher A, Reynolds HR, Somaratne JB, Tornvall P and Lindahl B. Randomized evaluation of beta blocker and ACE-inhibitor/angiotensin receptor blocker treatment in patients with myocardial infarction with non-obstructive coronary arteries (MINOCA-BAT): rationale and design. Am Heart J 2021; 231: 96-104.

[26] Sim HW, Zheng H, Richards AM, Chen RW, Sahlen A, Yeo KK, Tan JW, Chua T, Tan HC, Yeo TC, Ho HH, Liew BW, Foo LL, Lee CH, Hausenloy DJ and Chan MY. Beta-blockers and renin-angiotensin system inhibitors in acute myocardial infarction managed with inhospital coronary revascularization. Sci Rep 2020; 10: 15184.

[27] Huang K, Zhang Y, Yang F, Luo X, Long W and Hou X. Effect of enalapril combined with bisoprolol on cardiac function and inflammatory indexes in patients with acute myocardial infarction. Evid Based Complement Alternat Med 2022; 2022: 6062450.

[28] Kristensen SL, Rorth R, Jhund PS, Docherty KF, Sattar N, Preiss D, Kober L, Petrie MC and McMurray JJV. Cardiovascular, mortality, and kidney outcomes with GLP-1 receptor agonists in patients with type 2 diabetes: a systematic review and meta-analysis of cardiovascular outcome trials. Lancet Diabetes Endocrinol 2019; 7: 776-785.