Am J Transl Res 2025;17(4):2826-2834
www.ajtr.org /ISSN:1943-8141/AJTR0162935

Original Article

Sacubitril/valsartan combined with amiodarone enhances cardiac function and reduces adverse outcomes in patients with new-onset atrial fibrillation after acute myocardial infarction

Qiu-Mei Liu[1], Hong Tang[1], Hui-Lin Luo[2], Fang-Yang Huang[1]

[1]Department of Cardiology, West China Hospital, Sichuan University, Chengdu 610041, Sichuan, China; [2]Department of Integrated Traditional Chinese and Western Medicine, West China Hospital, Sichuan University, Chengdu 610041, Sichuan, China

Received December 26, 2024; Accepted March 11, 2025; Epub April 15, 2025; Published April 30, 2025

Abstract: Objective: To compare the efficacy of amiodarone alone versus amiodarone combined with sacubitril/valsartan in patients with new-onset atrial fibrillation (NOAF) after acute myocardial infarction (AMI) and to provide a theoretical basis for optimizing clinical strategies. Methods: Data of 106 NOAF patients admitted to West China Hospital, Sichuan University (January 2022 to June 2024) were retrospectively analyzed. Patients were divided into a control group (amiodarone alone) and an observation group (amiodarone combined with sacubitril/valsartan). Demographic characteristics, length of hospital stay, duration of atrial fibrillation, creatine kinase MB (CK-MB), high-sensitivity cardiac troponin T (hs-cTnT), ventricular rate, creatinine, urea, C-reactive protein (CRP), alanine transaminase (ALT), and aspartate aminotransferase (AST) were collected and compared between the two groups. Cardiac function parameters, including left ventricular end-diastolic diameter (LVEDD), left ventricular ejection fraction (LVEF), left ventricular end-systolic diameter (LVESD), recurrence rate, and other adverse events, were recorded at admission and six months post-discharge. Results: Both groups showed significant improvement in CK-MB, hs-cTnT, ventricular rate, ALT, and AST after treatment, with no significant difference between the groups. While CRP levels remained stable in the control group, the observation group demonstrated a significant decrease. Additionally, the observation group showed a significantly elevated LVEF level but lower LVESD and LVEDD levels after treatment, while these parameters remained unchanged in the control group. Besides, the observation group had a longer recurrence time and a lower incidence of stroke. Conclusion: Amiodarone combined with sacubitril/valsartan significantly improves cardiac function, prolongs the time to recurrence, and reduces the incidence of stroke in NOAF patients after AMI.

Keywords: New-onset atrial fibrillation, cardiac function, acute myocardial infarction, recurrence, prognosis

Introduction

Acute myocardial infarction (AMI) is a leading cause of disability and mortality globally. New-onset atrial fibrillation (NOAF) is a prevalent complication of AMI, often recurring and significantly increasing the risk of mortality, heart failure, and prolonged hospitalization [1-4]. Epidemiological research indicates that up to 21% of patients diagnosed with AMI may develop NOAF, with the incidence rising due to an aging population and the increasing burden of cardiovascular disease [5, 6]. Beyond being an important indicator of short- and long-term adverse outcomes, NOAF contributes substantially to healthcare resource utilization and socioeconomic burden [1, 2, 7]. However, since the mechanisms underlying NOAF are not fully understood, clinical management remains challenging, and optimal preventive and therapeutic strategies are still needed [2]. Therefore, exploring effective treatments for NOAF in AMI patients is crucial to enhancing clinical intervention strategies and improving patient outcomes.

Conventional treatment for atrial fibrillation (AF) includes pharmacologic cardioversion, heart

https://doi.org/10.62347/LGIM6718

rate control, and anticoagulation [8]. Amiodarone is a broad-spectrum antiarrhythmic drug widely used to manage AF by inhibiting potassium ion efflux and prolonging the action potential duration and effective refractory period of cardiomyocytes, thereby achieving rhythm control and restoring sinus rhythm [8]. Previous studies have shown that oral amiodarone is highly effective in converting AF to sinus rhythm and maintaining it [9], while intravenous amiodarone is beneficial for acute AF episodes [10]. Although amiodarone shows promising clinical efficacy in AF management, its efficacy in AMI patients with NOAF requires further clinical validation.

Sacubitril/valsartan is a dual-acting agent that combines angiotensin receptor blockade with neprilysin inhibition, exhibiting anti-ventricular remodeling effects. Initially developed for the treatment of heart failure, its potential role in AF management has garnered increasing attention [11]. The effectiveness of sacubitril/valsartan in managing heart rhythm in patients with persistent AF has been demonstrated in previous research [12]. Additionally, a meta-analysis showed that sacubitril/valsartan reduces the occurrence of AF among patients undergoing catheter ablation and shows superior benefits in improving cardiac remodeling [13, 14]. However, research specifically examining its impact on NOAF after AMI remains limited.

This retrospective study aimed to systematically evaluate the efficacy of amiodarone alone versus amiodarone in combination with sacubitril/valsartan in treating NOAF after AMI. We hypothesized that the combination therapy would yield better results in improving cardiac function, preventing recurrence, and reducing other adverse events. We anticipate that our research will offer novel insights into therapeutic approach for managing NOAF following AMI.

Methods

Case selection

Clinical data from 106 AMI patients who developed AF after admission to West China Hospital, Sichuan University between January 2022 and June 2024 were retrospectively analyzed in this study. Inclusion criteria: age > 18 years; with or without percutaneous coronary intervention (PCI); treatment with amiodarone for cardioversion with successful restoration to sinus rhythm; and documented follow-up at 6 months post-discharge. Exclusion criteria: prior history of AF; history of sick sinus syndrome; history of rheumatic valvular disease; previous coronary artery bypass grafting surgery (due to notably elevated postoperative AF incidence) [7, 15]; malignant tumor; and incomplete clinical data. This study was approved by the Ethics Committee of West China Hospital, Sichuan University.

Diagnostic criteria for AMI: Elevated high-sensitivity cardiac troponin T (hs-cTnT) levels, exceeding the 99th percentile of the upper limit of normal; chest pain or other symptoms of myocardial ischemia; electrocardiographic changes, including sustained ST-segment depression > 0.1 mv or elevation > 0.2 mv; or imaging studies confirming new-onset loss of viable myocardium or segmental ventricular wall motion abnormalities [7]. NOAF was identified as the first occurrence of AF during hospitalization in patients with no prior history of AF. Diagnosis was confirmed through cardiac monitoring or 12-lead electrocardiograms, characterized by irregular R-R intervals, absence of P waves, and abnormal rhythms lasting more than 30 seconds [7, 16].

Data collection and outcome measurement

Patients were divided into two groups based on their treatment regimen recorded in the clinical system: the control group (amiodarone alone; n = 50) and the observation group (amiodarone combined with sacubitril/valsartan; n = 56). In the control group, amiodarone was administered intravenously to restore sinus rhythm on the basis of optimal treatment (antihypertensive, lipid-regulating, hypoglycemic and anticoagulant therapy). Initially, 150 mg was given over 10 min, followed by an infusion of 1 mg/min for 6 h, and then switched to oral administration (100-200 mg once daily) for maintenance [8, 9, 17]. Patients in the observation group received additional sacubitril/valsartan treatment (oral administration) in addition to the control group. The initial dosage of sacubitril/valsartan was 50 mg, twice daily, and the dose was adjusted every 2 to 4 weeks, depending on patient tolerance, up to a target dose of 200 mg twice daily [12].

Table 1. Characteristics of the two groups

	Observation group n = 56	Control group n = 50	$\chi^2/t/Z$	*P* value
Gender [*n* (%)]			1.078	0.299
Female	21 (37.5)	14 (28.0)		
Male	35 (62.5)	36 (72.0)		
Age [year, M (P25, P75)]	70.50 (61.00, 75.00)	68.50 (57.25, 74.00)	-0.301	0.763
Lifestyle [*n* (%)]				
Smoking	32 (57.1)	23 (46.0)	1.314	0.252
Alcohol consumption	37 (66.1)	30 (60.0)	0.419	0.518
BMI ($\bar{x} \pm s$)	21.63 ± 2.23	21.99 ± 2.34	0.799	0.426
Underlying disease [*n* (%)]				
Diabetes	24 (42.9)	16 (32.0)	1.325	0.250
Hyperlipidemia	18 (32.1)	14 (28.0)	0.215	0.677
Hypertension	23 (41.1)	25 (50.0)	0.850	0.435

Abbreviation: BMI: Body Mass Index.

Demographic information on gender, age, body mass index (BMI), lifestyle (smoking, alcohol consumption), underlying diseases (diabetes, hyperlipidemia, and hypertension), duration of hospitalization, duration of AF, time from admission to AF onset, and ventricular rate of all patients were collected from the electronic clinical system. Serological parameters of creatine kinase MB (CK-MB), hs-cTnT, creatinine, urea, alanine transaminase (ALT), aspartate aminotransferase (AST), and C-reactive protein (CRP) were collected. In addition, important cardiac function indices, such as left ventricular ejection fraction (LVEF) at admission and 6 months after discharge, left ventricular end-systolic diameter (LVESD), and left ventricular end-diastolic diameter (LVEDD) were also recorded. Finally, AF recurrence, time to recurrence, and adverse events such as death, heart failure, and stroke within 6 months of discharge were collected.

Statistical analysis

SPSS 26.0 was used for statistical analysis. The normality of the measurement data was assessed prior to analysis. Normally distributed data were expressed as $\bar{x} \pm sd$, and independent samples *t*-test was used for comparison between the two groups, while paired samples *t*-test was used for comparison between different time points within the group. Non-normally distributed data were expressed as median, quartiles [M (P25, P75)], and comparisons between groups were performed using the Mann-Whitney *U* test, while within-group comparisons were performed using the Wilcoxon matched-pairs signed rank test. Categorical data were described as numbers and percentages and were statistically analyzed using the χ^2 test. A *P* value of less than 0.05 was considered statistically significant.

Results

Comparison of baseline characteristics between the two groups

No significant differences were observed between the two groups in terms of gender, age, lifestyle, BMI and prevalence of other underlying diseases (diabetes, hyperlipidemia and hypertension) (all $P > 0.05$), indicating the comparability between the two groups (**Table 1**).

Comparison of length of hospital stay and duration of AF between the two groups

Length of hospital stay, duration of AF, and time from admission to AF onset are important factors affecting the prognosis of AMI patients. As shown in **Table 2**, no significant differences were noted between the two groups in terms of the above three metrics (all $P > 0.05$).

Comparison of CK-MB, hs-cTnT, and ventricular rate between the two groups

As shown in **Table 3**, the levels of CK-MB and hs-cTnT were elevated at admission in both groups, with no significant difference between the two groups ($P > 0.05$). At discharge, CK-MB

Table 2. Duration of hospital stay, duration of atrial fibrillation, and time in atrial fibrillation from the time of admission of the two groups

	Observation group n = 56	Control group n = 50	*P* value
Duration of hospital stay [day, M (P25, P75)]	8.50 (6.00, 11.00)	9.00 (7.00, 10.75)	0.374
Duration of atrial fibrillation [h, M (P25, P75)]	42.50 (28.50, 56.00)	36.50 (24.00, 48.00)	0.238
Time from admission to onset of atrial fibrillation (h, $\bar{x} \pm sd$)	22.50 (11.75, 38.00)	22.00 (8.00, 31.00)	0.202

Table 3. CK-MB, hs-cTnT, and ventricular rate at admission and discharge of the two groups

	Observation group n = 56	Control group n = 50	*P* value
CK-MB (U/L, M (P25, P75))			
Admission	25.00 (19.98, 27.95)	25.00 (21.85, 25.00)	0.895
Discharge	18.55 (13.75, 24.25)	20.35 (16.58, 23.55)	0.336
P value	< 0.001	0.003	
hs-cTnT (pg/mL, M (P25, P75))			
Admission	366.55 (274.45, 475.28)	350.00 (270.20, 435.00)	0.504
Discharge	54.15 (38.53, 79.63)	63.85 (49.13, 78.60)	0.136
P value	< 0.001	< 0.001	
Ventricular rate (bpm, $\bar{x} \pm sd$)			
Admission	87.42 ± 15.48	85.48 ± 12.76	0.484
Discharge	77.12 ± 15.55	78.50 ± 13.94	0.634
P value	< 0.001	0.010	

Abbreviations: CK-MB: creatine kinase MB; hs-cTnT: high-sensitivity cardiac troponin T.

levels were significantly reduced in both the control group ($P = 0.003$) and the observation group ($P < 0.001$) compared to admission levels. Similarly, hs-cTnT levels at discharge were significantly lower in both the control group ($P < 0.001$) and the observation group ($P < 0.001$) compared to their admission levels. Notably, there was no statistically significant difference between the two group regarding the reduction in CK-MB and hs-cTnT levels ($P > 0.05$).

At admission, the ventricular rates were comparable between the control and observation groups, with no significant difference ($P > 0.05$). At discharge, the ventricular rates were significantly lower in both the control group ($P = 0.010$) and observation group ($P < 0.001$) compared to the admission levels. However, no significant difference was observed between the two groups ($P > 0.05$).

Comparison of kidney function, liver function, and inflammatory markers between the two groups

As shown in **Table 4**, no significant difference was observed in creatinine and urea levels between the two groups at admission and discharge (all $P > 0.05$). For liver function, ALT and AST levels were comparable between the two groups at admission (both $P > 0.05$). Post-treatment, the ALT and AST levels in both groups were significantly decreased at discharge ($P < 0.001$). However, no significant inter-group difference was observed in ALT and AST at discharge ($P > 0.05$). As for inflammatory marker (CRP), the two groups showed comparable CRP levels at admission ($P > 0.05$). Post-treatment, the control group maintained a stable level ($P > 0.05$), while the observation group demonstrated a significant reduction ($P = 0.002$). Notably, no significant difference was observed in CRP levels between the two groups at discharge ($P > 0.05$).

Comparison of LVEF, LVEDD, and LVESD levels between the two groups

As shown in **Table 5**, no significant difference was noted in LVEF, LVEDD, or LVESD between the two groups at admission (all $P > 0.05$). After 6 months of treatment, control group showed no significant changes in these metrics (all $P > 0.05$). However, LVEF ($P = 0.006$) was signifi-

Table 4. Creatinine, urea nitrogen, AST, ALT and CRP at admission and discharge of the two groups

	Observation group n = 56	Control group n = 50	*P* value
Creatinine [μmol/L, M (P25, P75)]			
Admission	91.13 (84.05, 97.60)	87.92 (81.09, 96.40)	0.211
Discharge	89.82 (85.32, 94.00)	87.85 (80.44, 93.50)	0.216
P value	0.253	0.606	
Urea [mmol/L, M (P25, P75)]			
Admission	9.69 (7.20, 12.46)	8.84 (6.37, 12.48)	0.494
Discharge	9.83 (6.85, 14.40)	10.91 (6.75, 13.18)	0.695
P value	0.599	0.735	
ALT (U/L, $\bar{x} \pm sd$)			
Admission	62.49 ± 15.50	59.50 ± 15.49	0.332
Discharge	43.50 ± 12.38	42.50 ± 10.29	0.654
P value	< 0.001	< 0.001	
AST [U/L, M (P25, P75)]			
Admission	68.50 (58.88, 76.50)	72.05 (61.63, 81.63)	0.181
Discharge	55.70 (43.98, 62.38)	50.00 (44.85, 56.85)	0.209
P value	< 0.001	< 0.001	
CRP [mg/L, M (P25, P75)]			
Admission	7.90 (5.98, 9.30)	7.45 (5.50, 9.08)	0.531
Discharge	5.95 (3.98, 7.55)	6.50 (5.53, 8.45)	0.084
P value	0.002	0.129	

Abbreviations: AST: aspartate aminotransferase; ALT: alanine transaminase; CRP: C-reactive protein.

Table 5. LVEF, LVEDD, and LVESD at admission and 6 months after discharge of the two groups

	Observation group n = 56	Control group n = 50	*P* value
LVEF [%, M (P25, P75)]			
Admission	43.00 (39.75, 52.00)	43.00 (39.00, 49.00)	0.374
Six months after discharge	52.00 (42.00, 56.00)	46.00 (39.00, 54.00)	0.018
P value	0.006	0.149	
LVEDD [mm, M (P25, P75)]			
Admission	48.00 (43.75, 51.00)	49.00 (44.00, 52.00)	0.233
Six months after discharge	46.00 (43.75, 48.25)	48.00 (44.00, 52.00)	0.012
P value	0.022	0.626	
LVESD (mm, $\bar{x} \pm sd$)			
Admission	35.23 ± 5.54	33.86 ± 5.80	0.216
Six months after discharge	30.87 ± 3.32	32.88 ± 5.05	0.017
P value	< 0.001	0.352	

Abbreviations: LVEF: left ventricular ejection fraction; LVESD: left ventricular end-systolic diameter; LVEDD: left ventricular end-diastolic diameter.

cantly elevated, while LVEDD ($P = 0.022$) and LVESD ($P < 0.001$) were significantly decreased in the observation group after treatment. Additionally, the observation group demonstrated significantly better LVEF ($P = 0.018$), LVEDD ($P = 0.012$), and LVESD ($P = 0.017$) levels compared to the control group.

Comparison of recurrence rate and time to recurrence between the two groups

Patients with NOAF are at risk of recurrence after the restoration of normal sinus rhythm. As shown in **Table 6**, no significant difference was observed in the recurrence rate between the

Table 6. Recurrence and their recurrence time of the two groups

	Observation group n = 56	Control group n = 50	*P* value
Recurrence [*n* (%)]	3 (5.4)	8 (16.0)	0.073
Recurrence time (day, $\bar{x} \pm sd$)	151.00 ± 19.31	94.50 ± 26.32	0.009

Table 7. Adverse events in both groups

	Observation group n = 56	Control group n = 50	*P* value
Heart failure [*n* (%)]	3 (5.4)	7 (14.0)	0.185
Stroke [*n* (%)]	3 (5.4)	9 (18.0)	0.040
Death [*n* (%)]	1 (1.8)	2 (4.0)	0.601

two groups at 6 months after discharge ($P > 0.05$). However, the patients in the observation group had a significantly longer time to discharge compared to those in the control group ($P = 0.009$).

Comparison of adverse events between the two groups

Apart from recurrence, patients were evaluated for cardiovascular complications and mortality risks. As shown in **Table 7**, no significant difference was noted in the incidence of heart failure and death between the two groups (both $P > 0.05$). However, a significantly reduced incidence of stroke was observed in the observation group compared to the control group ($P = 0.040$).

Discussion

Amiodarone is a commonly used drug to restore and maintain sinus rhythm in patients with all types of AF [8, 9]. Sacubitril/valsartan, initially developed for heart failure management, has gained increasing attention for its anti-ventricular remodeling effects and potential in AF treatment [12]. This study evaluated the therapeutic effects of amiodarone alone and its combination with sacubitril/valsartan in patients with NOAF following AMI. Both groups showed significant improvements in CK-MB, hs-cTnT, ventricular rate, ALT, AST and CRP levels at discharge. Six months after discharge, the observation group exhibited significantly better LVEF, LVEDD and LVESD compared to admission and to the control group. In addition, the observation group had a longer time to recurrence and a lower incidence of heart failure than the control group. These results suggest that amiodarone combined with sacubitril/valsartan not only improves cardiac function but also reduces adverse outcomes compared to amiodarone alone.

CK-MB and hs-cTnT are widely recognized as sensitive indicators for evaluating cardiac function, demonstrating significant correlations with both short-term and long-term adverse outcomes in patients with heart disease [16, 18, 19]. AMI is known to induce a significant increase in hs-cTnT and CK-MB levels, with more pronounced increases in patients with NOAF [16]. During our investigation, markedly elevated levels of hs-cTnT and CK-MB were observed in both groups of AMI patients upon hospital admission, especially hs-cTnT, which showed significant reduction following conventional treatment at the time of discharge. Liver function is intricately linked to the progression of cardiovascular disease. Studies have shown that increased levels of AST and ALT are frequently observed in patients with AMI, and these elevations demonstrate a significant correlation with the AUC of CK-MB [20, 21]. In this study, NOAF patients showed a significant elevation in both ALT and AST levels at admission compared to normal ranges. Notably, this abnormal upregulation of liver function indices was significantly reversed by either amiodarone or amiodarone combined with sacubitril/valsartan. However, this improvement did not show strong correlation with either treatment modality, likely due to the short duration of hospitalization. We speculate that these effects may be attributed to other conventional treatments administered during the hospital stay. CRP, a crucial inflammation-related marker in routine blood tests, has been extensively studied in relation to NOAF. Previous research has demonstrated significantly higher CRP levels in patients developing NOAF compared to those without NOAF [22]. Furthermore, CRP levels serve as a significant and independent predictor of NOAF development in AMI patients [23, 24]. In our research, we found that CRP levels were significantly elevated in all NOAF patients, with notable improvement following treatment

with amiodarone combined with sacubitril/valsartan. While the amiodarone monotherapy group did not show statistically significant improvement in CRP levels, a reduction was observed compared to pretreatment levels. We suggest that the limited sample size in our study may have influenced these findings.

LVEF, LVEDD and LVESD are critical indicators for assessing cardiac function in patients with heart failure and AIM [9]. These parameters serve as the primary reference criteria for evaluating changes in cardiac function [25, 26]. Previous studies have shown that LVEF recovery 2-12 weeks after AMI is a significant predictor of patient prognosis, with greater LVEF improvement correlating with a reduced risk of cardiac arrest and mortality. Furthermore, LVEF assessment at 6 weeks post-AMI offers a stronger predictive value for clinical outcome [27]. In this study, NOAF patients exhibited a notable decline in LVEF, accompanied by significant increases in both LVESD and LVEDD, indicating a significant impairment of cardiac function. Sacubitril/valsartan has demonstrated superior efficacy in enhancing cardiac function among patients with heart failure, and this improvement is mainly manifested by the increase in LVEF and decrease in LVESD and LVEDD [11, 14]. Similarly, our study revealed that LVEF, LVEDD, and LVESD were significantly improved in the observation group compared to the amiodarone monotherapy group six months after discharge. We hypothesize that this improvement is likely related to the reversal of cardiac remodeling by sacubitril/valsartan [28].

AF has a higher risk of recurrence, and prior research indicated that a history of paroxysmal AF is associated with earlier recurrence after resuscitation compared to those with persistent AF [4, 29]. Sacubitril/valsartan has also been shown to be effective in reducing AF recurrence [14]. In this study, although we did not observe a statistically significant difference in recurrence rates between the two groups, we hypothesize that this result may be influenced by the limited sample size and insufficient data. However, a significantly longer time to recurrence was observed in observation group compared to the control group, suggesting a potential benefit of the combination therapy in delaying AF relapse. In addition to recurrence, patients with NOAF are at heightened risk of other adverse events, such as heart failure, stroke, and death after discharge. Previous studies have shown that AMI patients with a lower NOAF burden have a heart failure hospitalization rate exceeding 10% at 6 months after discharge, with higher rates in high-burden patients [7]. In addition, the overall adverse event rate at 6 months post-discharge in NOAF patients after PCI was approximately 10%, with the highest rate in patients with transient AF [22]. Our study found that patients in the amiodarone group had an 18% risk of stroke at 6 months after discharge, which was significantly reduced by the combination with sacubitril/valsartan. Consistent with our findings, prior research has demonstrated that early initiation of sacubitril/valsartan therapy significantly lowers the risk of major adverse cardiac events in AMI patients [30].

In conclusion, this study demonstrated the efficacy of sacubitril/valsartan in improving the prognosis of NOAF patients. However, several limitations should be noted. The single-center, retrospective design and the relatively small sample size may limit the generalizability of the results. Future studies should utilize a randomized controlled trial design involving multiple centers and regions to improve the reliability of the results. In addition, this study focused only on cardiac function changes and adverse events within six months after discharge. Future studies should incorporate longer follow-up periods to further validate and strengthen the findings.

Disclosure of conflict of interest

None.

Address correspondence to: Fang-Yang Huang, Department of Cardiology, West China Hospital, Sichuan University, No. 37 Guoxue Lane, Wuhou District, Chengdu 610041, Sichuan, China. Tel: +86-028-85422607; E-mail: hfy1036@163.com

References

[1] Frederiksen TC, Dahm CC, Preis SR, Lin H, Trinquart L, Benjamin EJ and Kornej J. The bidirectional association between atrial fibrillation and myocardial infarction. Nat Rev Cardiol 2023; 20: 631-644.

[2] Li Z, Liu Q, Liu F, Hidru TH, Yang Y, Wang S, Bai L, Chen J, Yang X and Xia Y. Atrial cardiomyopathy markers and new-onset atrial fibrillation

risk in patients with acute myocardial infarction. Eur J Intern Med 2022; 102: 72-79.
[3] Fauchier L, Bisson A, Bodin A, Herbert J, Angoulvant D, Danchin N and Cottin Y. Outcomes in patients with acute myocardial infarction and new atrial fibrillation: a nationwide analysis. Clin Res Cardiol 2021; 110: 1431-1438.
[4] Guenancia C, Toucas C, Fauchier L, Stamboul K, Garnier F, Mouhat B, Sagnard A, Lorgis L, Zeller M and Cottin Y. High rate of recurrence at long-term follow-up after new-onset atrial fibrillation during acute myocardial infarction. Europace 2018; 20: e179-e188.
[5] Obayashi Y, Shiomi H, Morimoto T, Tamaki Y, Inoko M, Yamamoto K, Takeji Y, Tada T, Nagao K, Yamaji K, Kaneda K, Suwa S, Tamura T, Sakamoto H, Inada T, Matsuda M, Sato Y, Furukawa Y, Ando K, Kadota K, Nakagawa Y and Kimura T; CREDO-Kyoto AMI Registry Wave-2 Investigators. Newly diagnosed atrial fibrillation in acute myocardial infarction. J Am Heart Assoc 2021; 10: e021417.
[6] Gao P, Gao X, Xie B, Tse G and Liu T. Aging and atrial fibrillation: a vicious circle. Int J Cardiol 2024; 395: 131445.
[7] Luo J, Xu S, Li H, Gong M, Li Z, Liu B, Qin X, Shi B and Wei Y. Long-term impact of the burden of new-onset atrial fibrillation in patients with acute myocardial infarction: results from the NOAFCAMI-SH registry. Europace 2021; 23: 196-204.
[8] Joglar JA, Chung MK, Armbruster AL, Benjamin EJ, Chyou JY, Cronin EM, Deswal A, Eckhardt LL, Goldberger ZD, Gopinathannair R, Gorenek B, Hess PL, Hlatky M, Hogan G, Ibeh C, Indik JH, Kido K, Kusumoto F, Link MS, Linta KT, Marcus GM, McCarthy PM, Patel N, Patton KK, Perez MV, Piccini JP, Russo AM, Sanders P, Streur MM, Thomas KL, Times S, Tisdale JE, Valente AM and Van Wagoner DR. 2023 ACC/AHA/ACCP/HRS guideline for the diagnosis and management of atrial fibrillation: a report of the American College of Cardiology/American Heart Association Joint Committee on clinical practice guidelines. Circulation 2024; 149: e1-e156.
[9] Singh BN, Singh SN, Reda DJ, Tang XC, Lopez B, Harris CL, Fletcher RD, Sharma SC, Atwood JE, Jacobson AK, Lewis HD, Raisch DW and Ezekowitz MD. Amiodarone versus sotalol for atrial fibrillation. N Engl J Med 2005; 352: 1861-1872.
[10] Tuseth V, Jaatun HJ and Dickstein K. Amiodarone infusion in the treatment of acute atrial fibrillation or flutter: high versus low dose treatment. Heart 2005; 91: 964-965.
[11] Kalantari S, Oren D, Medvedofsky D, Narang A, Imamura T, Tayazime S, Kim GH, Raikhelkar J, Sayer G, Lang RM and Uriel N. Reverse remodeling effects of sacubitril-valsartan: structural and functional optimization in stage C heart failure. Am J Cardiol 2024; 210: 249-255.
[12] Chen Q, Chen Y, Qin F, Du H, Gan C, Zhou B, Wang N, Xiao M, Ou Z, Zhao W, Cui B, Liu Z and Yin Y. Effect of sacubitril-valsartan on restoration and maintenance of sinus rhythm in patients with persistent atrial fibrillation. Front Cardiovasc Med 2022; 9: 870203.
[13] Bezerra F, Rocha Dantas C, Sobreira L, Kendi Tsuchiya Sano V, de Oliveira Macena Lobo A, Cardoso J, Kelly F, A de Moraes FC and Colombo F. Abstract 4143120: efficacy of sacubitril-valsartan compared with angiotensin-converting enzyme inhibitor or angiotensin-receptor blocker for preventing atrial fibrillation recurrence after catheter ablation: a systematic review and meta-analysis. Circulation 2024; 150: A4143120-A4143120.
[14] de Lucena LA, Freitas MAA, Guida CM, Hespanhol LC, de Sousa AKC, de Sousa JCV and Maia FGS. Sacubitril-valsartan lowers atrial fibrillation recurrence and left atrial volume post-catheter ablation: systematic review and meta-analysis. Am J Cardiovasc Drugs 2025; 25: 157-167.
[15] Tzoumas A, Nagraj S, Tasoudis P, Arfaras-Melainis A, Palaiodimos L, Kokkinidis DG and Kampaktsis PN. Atrial fibrillation following coronary artery bypass graft: where do we stand? Cardiovasc Revasc Med 2022; 40: 172-179.
[16] Zhang LX, Cao JY and Zhou XJ. Construction and validation of a nomogram prediction model for the risk of new-onset atrial fibrillation following percutaneous coronary intervention in acute myocardial infarction patients. BMC Cardiovasc Disord 2024; 24: 642.
[17] Chinese Society of Cardiology, Chinese Medical Association; Heart Rhythm Committee of Chinese Society of Biomedical Engineering. Chinese guidelines on diagnosis and management of atrial fibrillation. Zhonghua Xin Xue Guan Bing Za Zhi 2023; 51: 572-618.
[18] Chen Y, Zhou X, Chen Z, Xia J, Guan F, Li Y, Li Y, Chen Y, Zhao Y, Qiu H, Liang J and Tang L. The use of high-sensitivity cardiac troponin T and creatinine kinase-MB as a prognostic markers in patients with acute myocardial infarction and chronic kidney disease. Ren Fail 2023; 45: 2220420.
[19] Pölzl L, Engler C, Sterzinger P, Lohmann R, Nägele F, Hirsch J, Graber M, Eder J, Reinstadler S, Sappler N, Kilo J, Tancevski I, Bachmann S, Abfalterer H, Ruttmann-Ulmer E, Ulmer H, Griesmacher A, Heuts S, Thielmann M, Bauer A, Grimm M, Bonaros N, Holfeld J and Gollmann-Tepeköylü C. Association of high-sensitivity cardiac troponin T with 30-day and 5-year mortality after cardiac surgery. J Am Coll Cardiol 2023; 82: 1301-1312.

[20] Choi KM, Han K, Park S, Chung HS, Kim NH, Yoo HJ, Seo JA, Kim SG, Kim NH, Baik SH, Park YG and Kim SM. Implication of liver enzymes on incident cardiovascular diseases and mortality: a nationwide population-based cohort study. Sci Rep 2018; 8: 3764.
[21] Lofthus DM, Stevens SR, Armstrong PW, Granger CB and Mahaffey KW. Pattern of liver enzyme elevations in acute ST-elevation myocardial infarction. Coron Artery Dis 2012; 23: 22-30.
[22] Zhou C, Yu L, Zhu Q, Xiang G, Xv P, Chen C, Cai M, Huang W and Shan P. Clinical outcome of new-onset atrial fibrillation after emergency percutaneous coronary intervention for myocardial infarction. Am J Emerg Med 2021; 45: 162-168.
[23] Fu Y, Pan Y, Gao Y, Yang X and Chen M. Predictive value of CHA2DS2-VASc score combined with hs-CRP for new-onset atrial fibrillation in elderly patients with acute myocardial infarction. BMC Cardiovasc Disord 2021; 21: 175.
[24] Zahler D, Merdler I, Rozenfeld KL, Shenberg G, Milwidsky A, Berliner S, Banai S, Arbel Y and Shacham Y. C-reactive protein velocity and the risk of new onset atrial fibrillation among ST elevation myocardial infarction patients. Isr Med Assoc J 2021; 23: 169-173.
[25] Zhang J, Feng R, Ferdous M, Dong B, Yuan H and Zhao P. Effect of 2 different dosages of rosuvastatin on prognosis of acute myocardial infarction patients with new-onset atrial fibrillation in Jinan, China. Med Sci Monit 2020; 26: e925666.
[26] Xiao J, Wu H, Gao Z, Wei H and Huang W. Association of left ventricular ejection fraction with risk of cardiovascular diseases: a prospective cohort study. Sci Rep 2024; 14: 25233.
[27] Chew DS, Heikki H, Schmidt G, Kavanagh KM, Dommasch M, Bloch TPE, Sinnecker D, Raatikainen P and Exner DV. Change in left ventricular ejection fraction following first myocardial infarction and outcome. JACC Clin Electrophysiol 2018; 4: 672-682.
[28] Yang L, Zhang M, Hao Z, Wang N and Zhang M. Sacubitril/valsartan attenuates atrial structural remodelling in atrial fibrillation patients. ESC Heart Fail 2022; 9: 2428-2434.
[29] Rutlen C, Mullen C, Phan F, Raitt M, Dalouk K, Zarraga I, Shim D and Jessel PM. Paroxysmal atrial fibrillation history is associated with earlier recurrence after first cardioversion compared with primary persistent atrial fibrillation. Heart Rhythm 2025; 22: 418-423.
[30] Liu Z, Cui K, Wang G, Jin W, Yao Q and Zhang Y. A clinical randomized trial: effects of early application of sacubitril/valsartan on ventricular remodeling and prognosis in acute myocardial infarction patients. Contemp Clin Trials Commun 2024; 42: 101303.