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Relationship between β-receptor blocker and atrial fibrillation in patients with heart failure: an observational study based on MIMIC IV database

Ying Wang[1] and Kenan Lou[1*]

Abstract

Background The β-receptor blocker is used to treat heart failure (HF), and its role in the occurrence of atrial fibrillation (AF) is unclear in patients with HF. This study aimed to investigate the relationship between β-receptor blocker use and AF in patients with HF.

Methods All data was collected from the Medical Information Mart for Intensive Care-IV (MIMIC-IV) database. The relationship between β-receptor blocker and AF was analyzed by univariate logistic regression, multivariate logistic regression, and subgroup analysis. The machine learning algorithm including logistic and Random forest was used to analyze the importance of variables in AF. The interaction analysis was conducted to determine whether other factors influence the relationship between β-receptor blocker and AF.

Results A total of 953 participants were involved. We found that the use of beta-blockers increased the risk of AF this result was not affected by confounding factors (OR (95%CI): 2.821[2.014,3.951], $p < 0.01$). The interaction analysis showed that myocardial infarction (MI) and β-receptor blocker had an interaction on AF (p for interaction < 0.001). The results of additive and multiplicative interaction analysis indicated that β-receptor-blocker use and infarction are antagonistic in the development of AF in patients with HF ((S (95% CI): 0.283[0.142, 0.563]; AP (95% CI): -1.187[-2.020, -0.354]; RERI (95% CI): -2.237[-3.722, -0.752], OR (95% CI):0.374[0.184, 0.772]).

Conclusion This study found that β-receptor blocker use was an important risk factor for AF in patients with HF. β-receptor blocker use was antagonistic to MI in AF in patients with HF.

Clinical trial Not applicable.

Keywords β-receptor blocker, Atrial fibrillation, Heart failure, Myocardial infarction, Interaction analysis

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Introduction

Heart failure (HF) occurs due to structural and/or functional abnormalities of the heart, leading to elevated intracardiac pressure and/or inadequate cardiac output at rest and/or during exercise [1, 2]. Literature reports indicate that, the age-adjusted incidence of HF has been declining, likely due to better management of cardiovascular diseases [3]. However, due to the aging population, the overall incidence of HF continues to rise [4–7]. Atrial fibrillation (AF) is the most common arrhythmia in HF patients, with approximately 30–40% of HF patients also having AF. The coexistence of both conditions significantly increases the incidence of cardiovascular events and mortality [8]. HF and AF exhibit bidirectional pathological interplay: HF drives cardiac remodeling that elevates AF susceptibility, whereas AF aggravates HF progression via tachyarrhythmia-induced hemodynamic impairment, atrial pump dysfunction, and thromboembolic risk, collectively worsening clinical outcomes [9]. Therefore, there is a need to emphasize the importance of joint management of both AF and HF.

The β-receptor blocker is one of the most important medications used in the clinical treatment of HF and arrhythmias. By inhibiting the activity of the sympathetic nervous system, β-receptor blocker can slow the heart rate, reduce myocardial oxygen consumption, and improve ventricular function. The use of β-receptor blocker has been shown to significantly reduce mortality in chronic HF by 33–35% [10]. Some studies have found that β-receptor blocker can also treat AF. By inhibiting excessive sympathetic nervous activity, β-receptor blocker slows the conduction speed of both the atria and ventricles, effectively controlling the heart rate in AF. β-receptor blocker also increases the ventricular filling time by slowing the heart rate, which helps improve left ventricular diastolic function and overall cardiac function, thus reducing cardiac load [11–13].

While the β-receptor blocker is used in the treatment of AF and HF, it remains unclear whether they influence the development of AF in HF patients. Therefore, this study aimed to investigate the relationship between β-blocker use and the occurrence of AF in HF patients.

Methods

Data sources and study population

We designed a cross-sectional study according to Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines. All data in this study were sourced from the Medical Information Mart for Intensive Care-IV (MIMIC-IV) database (https://mimic.mit.edu/iv/). MIMIC-IV compiles clinical data for over 190,000 patients and 450,000 hospital admissions at Beth Israel Deaconess Medical Center (BIDMC) from 2008 to 2019. This database contains both hospital and ICU data, characterized by its large volume, high-quality control, and detailed information, including demographics, vital signs, laboratory test results, imaging data, medications, patient follow-up times, and clinical outcomes. The MIMIC-IV database has been approved by the Institutional Review Boards of Massachusetts Institute of Technology and BIDMC, adheres to the Declaration of Helsinki, and employs anonymization methods to protect patient privacy, thus exempting it from requiring informed consent. Access to the MIMIC-IV database was obtained following the National Institutes of Health (NIH) certification training.

The study population included patients diagnosed with acute or chronic HF within the MIMIC-IV database based on the International Classification of Diseases (ICD) code. (It is worth noting that ICD-9-CM was used during 2008.01-2015.09 and ICD-10-CM was used during 2015.10-2019.12 in MIMIC-IV database) (https://mimic.mit.edu/) [3, 14]. Inclusion criteria were as follows: [1] patients with HF, and [2] age ≥ 18 years. Exclusion criteria included: [1] patients with unspecified HF [2], patients with hypertensive HF [3], patients with rheumatic HF [4], patients with other HF [5], lack of AF information. The flowchart is shown in Fig. 1. Finally, the ICD-codes of acute or chronic HF included in this study included 428.21, 428.22, 428.31, 428.32, 428.41, 428.42, 150.21, 150.22, 150.31, 150.32, 150.41, 150.42, and 150.811.

Data extraction

We used the PostgreSQL tool (version 12) to extract the following data from the MIMIC-IV database: ①Demographic information: sex (male, female), age (years), race (white, non-white), and marital status (single, married, divorced, widowed). ②Physiological data and disease type: body mass index (BMI, kg/m^2) and HF types (chronic, acute); ③Laboratory indicators: anion gap (mEq/L), calcium (mg/dL), creatinine (mg/dL), glucose (mg/dL), hemoglobin (g/dL), platelets (K/uL), potassium (mEq/L), red blood cell (RBC, m/uL), red blood cell distribution width (RDW, %), sodium (mEq/L), white blood cell (WBC, K/uL), urea nitrogen (mg/dL), and estimated glomerular filtration rate (eGFR, ml/min). ④Living habits index: smoking (yes, no), alcohol using (yes, no). ⑤Comorbidities: myocardial infarction (MI, yes, no), diabetes (yes, no), coagulopathy (yes, no), hypertension (yes, no). ⑥Medical records: metoprolol, atenolol, and carvedilol. ⑦Clinical outcomes: AF (yes, no). (Supplementary: non-white included blacks, Asians, Hispanic or Latino, and Black/African American. BMI (kg/m^2) = weight (kg) / height (m). The diagnostic criteria for diseases (including MI, hypertension, diabetes, and coagulopathy) are derived from the ICD coding system).

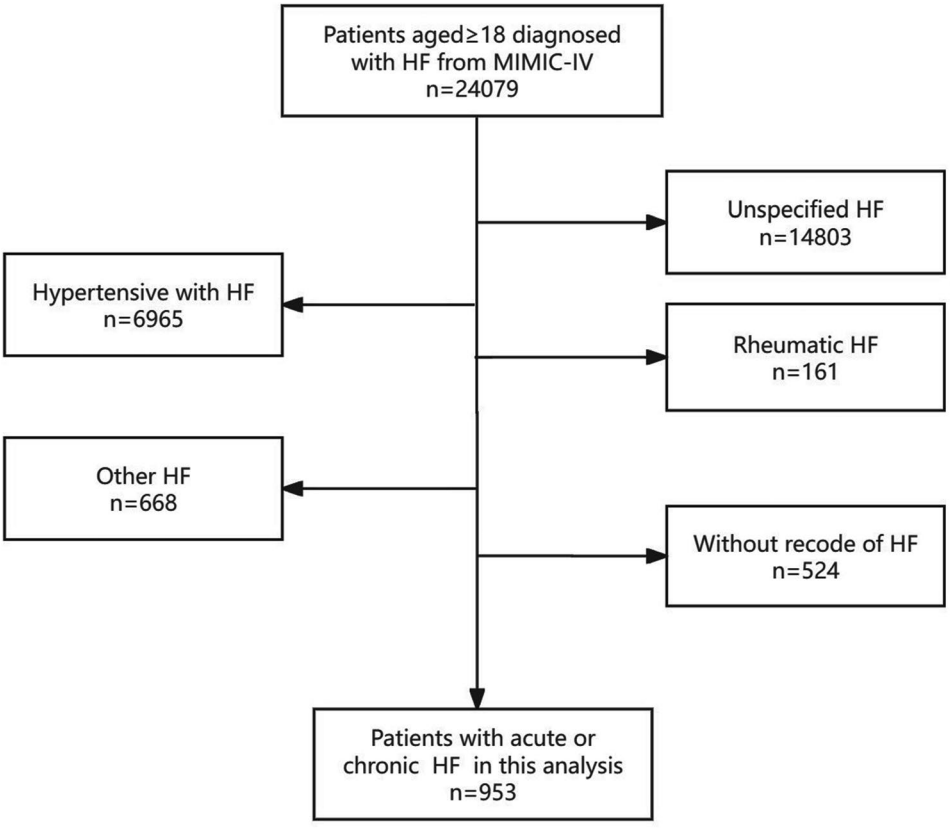

Fig. 1 Flow charts of the inclusion and exclusion of participants

Exposure variable and outcomes

The exposure variable in this study was β-receptor blocker use. One or more of metoprolol, atenolol, and carvedilol were used in patients, who served as patients were treated with β-receptor blocker. Otherwise, it is defined as patients who did not receive it. The outcome indicator is whether AF occurs. "1" in the AF record was defined as occurrence (yes), and "0" was defined as without occurrence (no).

Statistical analysis

In this study, we divided patients into an AF group and a non-AF group, and compared the differences of variables between the two groups. Categorical data were expressed as n (%) and their differences were compared using the χ^2 test. Normally distributed continuous data were expressed as mean ± standard deviation (SD) and their differences were compared using the t-test. Non-normally distributed continuous data were expressed as median (P25, P75) and compared using the Mann-Whitney U test. Machine learning algorithms were used to rank the importance of variables that differed between the groups. Subsequently, univariate logistic regression analysis was conducted to identify factors influencing AF. Multivariate logistic regression analysis was then performed to determine the independent relationship between β-receptor blocker use and AF after establishing multiple models. We corrected the covariates that were different between AF group and a non-AF group because these covariates may affect the occurrence of AF. We first adjusted for covariates with differences within the same category between two groups, followed by systematically correcting all differing variables. Specifically, Model 1 adjusted for demographic characteristics (age, race, and marital status); Model 2 adjusted for variables in categories with smaller datasets, including comorbidities (MI, and coagulopathy), disease types (HF types), and lifestyle habits (smoking, alcohol use); Model 3 accounted for laboratory indicators such as serum potassium and urea nitrogen; finally, Model 4 integrated adjustments for all significant variables between two groups. Subgroup logistic regression analysis was used to explore the relationship stability between β-receptor blocker use and AF. Interaction effect analyses based on additive and multiplicative models were also conducted to preliminarily explore interactions between β-receptor blocker use and other risk factors for AF. The indices for additive interaction effect included synergy index (S), attributable proportion of interaction (AP), relative excess risk of interaction (RERI). The 95% confidence interval

for interaction effect analyses was calculated by Delta method. All statistical analyses in this study were performed using SPSS (version 25.0) and RStudio (version 4.1.2) software. A p less than 0.05 was considered statistically significant.

Results

Characteristics of participants in this study

This study finally included 953 patients with acute or chronic HF (The following abbreviation is HF), including 500 males (52.466%) and 453 females (47.534%), with a median age of 69 [57,78] years. There were 390 (40.923%) patients with AF. Table 1 showed the baseline characteristics of patients grouped by AF status, indicating that there were significant differences between the without AF group and AF group in terms of 13 variables, including age, creatinine, potassium, urea nitrogen, EGFR, race, marital status, HF types, smoking, alcohol use, MI, coagulopathy, and β-receptor blocker using (all $p < 0.05$). The differences in other characteristics including BMI, anion gap, calcium, glucose, hemoglobin, platelets, RBC, RDW, sodium, WBC, sex, diabetes, and hypertension, were not statistically significant between the two groups (Supplementary Table 1).

The importance of β-receptor blocker use on AF

The logistic regression algorithm was used to rank the importance of factors on AF risk and its result showed that the top 10 orders of 13 factors were coagulopathy, β-receptor blocker use, eGFR, MI, race, smoking, potassium, HF types, creatinine, age (Fig. 2A). The results of the Random forest algorithm showed that the importance order of the top 10 factors was age, coagulopathy, β-receptor blocker using, MI, potassium, urea nitrogen, creatinine, race, marital status, and alcohol using (Fig. 2B). The above results indicated that the importance of β-receptor blocker using was located in the top three.

Table 1 Clinical characteristics of HF patients

Variables	Without AF($n=563$)	AF($n=390$)	U/χ^2	p
age, years	64.000[52.000,75.000]	73.000[64.000,82.000]	-8.541	< 0.001
creatinine, mg/dL	0.900[0.700,1.300]	1.000[0.800,1.400]	-2.768	0.005
potassium, mEq/L	4.100[3.700,4.500]	4.200[3.900,4.600]	-3.216	0.001
urea nitrogen, mg/dL	19.000[13.000,27.000]	21.000[16.000,34.000]	-4.450	< 0.001
eGFR, ml/min	0.992[0.805,1.057]	0.997[0.790,1.037]	3.386	< 0.001
race, n (%)				
white	378(75.600)	288(88.073)	19.620	< 0.001
non-white	122(24.400)	39(11.927)		
marital status, n (%)				
single	183(35.603)	77(22.581)	18.508	< 0.001
married	217(42.218)	167(48.974)		
divorced	45(8.755)	30(8.798)		
widowed	69(13.424)	67(19.648)		
HF types, n (%)				
chronic	202(35.879)	181(46.410)	10.630	0.001
acute	361(64.121)	209(53.590)		
smoking				
no	441(78.330)	344(88.205)	15.472	< 0.001
yes	122(21.670)	46(11.795)		
alcohol use, n (%)				
no	483(85.790)	354(90.769)	5.342	0.021
yes	80(14.210)	36(9.231)		
MI, n (%)				
no	387(68.739)	291(74.615)	3.876	0.049
yes	176(31.261)	99(25.385)		
coagulopathy, n (%)				
no	454(80.639)	211(54.103)	76.940	< 0.001
yes	109(19.361)	179(45.897)		
β-receptor blocker using, n (%)				
no	240(42.629)	81(20.769)	49.286	< 0.001
yes	323(57.371)	309(79.231)		

Abbreviation: HF: heart failure, eGFR: estimated glomerular filtration rate, AF: atrial fibrillation, MI: myocardial infarction

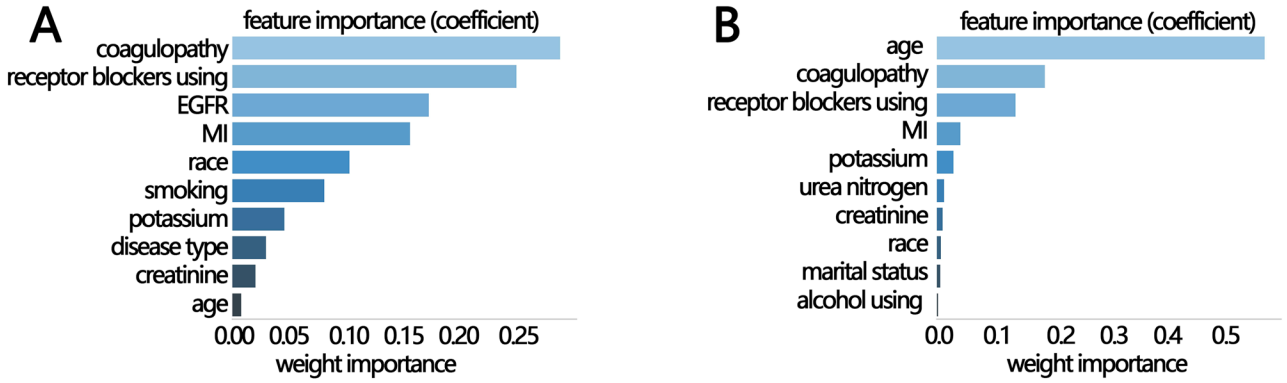

Fig. 2 The importance ranking bar chart of the top 10 variables by two methods. (**A**) logistic. (**B**) Random forest. Note: the longer the bar of the variable indicates that the higher the correlation coefficient with AF, the more advanced the ranking, and the more important it is. Abbreviation: eGFR: glomerular filtration rate, MI: myocardial infarction

Table 2 Univariate logistic regression analysis between variables and AF

Variable	OR	95%CI	*p*
age	1.041	[1.032,1.051]	< 0.001
creatinine	1.098	[0.981,1.228]	0.104
potassium	1.279	[1.058,1.547]	0.011
urea nitrogen	1.012	[1.006,1.019]	< 0.001
eGFR	0.408	[0.156,1.070]	0.068
race			
white	ref	ref	ref
non-white	0.420	[0.284,0.621]	< 0.001
marital status			
single	ref	ref	ref
married	1.829	[1.310,2.554]	< 0.001
divorced	1.584	[0.930,2.700]	0.091
widowed	2.308	[1.503,3.543]	< 0.001
HF types			
chronic	ref	ref	ref
acute	0.646	[0.497,0.841]	0.001
smoking			
no	ref	ref	ref
yes	0.483	[0.335,0.698]	< 0.001
alcohol using			
no	ref	ref	ref
yes	0.614	[0.405,0.931]	0.022
MI			
no	ref	ref	ref
yes	0.748	[0.560,0.999]	0.049
coagulopathy			
no	ref	ref	ref
yes	3.533	[2.647,4.716]	< 0.001
β-receptor blocker using			
no	ref	ref	ref
yes	2.835	[2.108,3.812]	< 0.001

Abbreviation: AF: atrial fibrillation, OR: odd ratio, CI: confidence interval, eGFR: estimated glomerular filtration rate, MI: myocardial infarction

Table 3 Association between β-receptor blocker using and AF by adjusted logistic regression analysis

Model	Without β-receptor blocker	With β-receptor blocker [OR, 95%CI]	*p*
Model 1	ref	2.821 [2.014, 3.951]	< 0.001
Model 2	ref	3.496 [2.527, 4.838]	< 0.001
Model 3	ref	3.049 [2.249, 4.132]	< 0.001
Model 4	ref	4.035 [2.761, 5.898]	< 0.001

Abbreviation: AF: atrial fibrillation, OR: odd ratio, CI: confidence interval, MI: myocardial infarction

Model 1 was adjusted for age, race, and marital status

Model 2 was adjusted for smoking, alcohol use, MI, HF types, and coagulopathy

Model 3 was adjusted for potassium and urea nitrogen

Model 4 was adjusted for potassium, urea nitrogen, age, race, marital status, smoking, alcohol use, MI, HF types, and coagulopathy

Relationship between β-receptor blocker use and AF risk

We then explored the relationship between β-receptor blocker use and AF risk. The results of univariate logistic regression analysis indicated that among the 13 factors analyzed, all indicators were significantly associated with AF (Table 2, all $P<0.05$) except creatinine and eGFR. Therefore, creatinine and eGFR were removed in our subsequent analyses. Especially, compared to patients not using β-receptor blockers, those using β-receptor blockers showed a significantly higher likelihood of developing AF (Table 2, OR 95% CI: 2.835 [2.108, 3.812]).

To further explore the independent effect of β-receptor blocker use on AF, we constructed 4 adjusted logistic regression models for analysis. As shown in Table 3, the results indicated that β-receptor blocker use promoted the occurrence of AF and its influence was not affected by confounding factors in Model 1 (OR 95%CI: 2.821 [2.014, 3.951], $p<0.001$), Model 2 (OR 95%CI: 3.496 [2.527, 4.838], $p<0.001$), Model 3 (OR 95%CI: 3.049 [2.249, 4.132], $p<0.001$), and Model 4 (OR 95%CI: 4.035 [2.761, 5.898], $p<0.001$). Four adjusted models comprehensively indicated their independent association.

Table 4 Subgroup logistic regression and interaction analysis between β-receptor blocker use and AF

Variable	Before adjustment			After adjustment		
	OR [95%CI]	*p*	*p* for interaction	OR [95%CI]	adjusted *p*	adjusted *p* for interaction
age			0.173			0.290
< 60	1.901[1.073,3.372]	0.029		2.22[1.212,4.073]	0.010	
≥ 60	3.032[2.131,4.322]	< 0.001		3.17[2.213,4.545]	0.001	
race			0.917			0.865
white	2.883[2.033,4.092]	< 0.001		3.03[2.125,4.342]	< 0.001	
non-white	3.024[1.322,6.901]	0.009		3.37[1.441,7.932]	0.005	
marital status			0.739			0.791
single	2.420[1.352,4.354]	0.003		2.72[1.483,5.031]	0.001	
married	2.832[1.780,4.491]	< 0.001		2.92[1.824,4.682]	< 0.001	
divorced	4.181[1.440,12.170]	0.009		5.25[1.611,17.101]	0.006	
widowed	2.082[0.950,4.560]	0.068		2.21[0.991,4.952]	0.054	
HF types			0.129			0.112
chronic	2.290[1.490,3.530]	< 0.001		2.37[1.537,3.685]	< 0.001	
acute	3.661[2.400,5.580]	< 0.001		4.13[2.652,6.423]	< 0.001	
smoking			0.306			0.346
no	3.021[2.180,4.170]	< 0.001		3.22[2.312,4.491]	< 0.001	
yes	1.970[0.930,4.170]	0.077		2.25[1.023,4.974]	0.044	
alcohol use			0.841			0.713
no	2.841[2.070,3.890]	< 0.001		3.08[2.232,4.260]	< 0.001	
yes	2.580[1.080,6.180]	0.033		2.56[1.054,6.220]	0.039	
MI			0.007			0.015
no	3.791[2.700,5.340]	< 0.001		4.02[2.849,5.701]	< 0.001	
yes	1.422[0.760,2.660]	0.277		1.72[0.874,3.370]	0.117	
Coagulopathy			0.782			0.760
no	3.031[2.043,4.501]	< 0.001		3.32[2.211,5.002]	< 0.001	
yes	3.322[1.992,5.530]	< 0.001		3.38[2.023,5.671]	< 0.001	

Abbreviation: AF: atrial fibrillation, OR: odd ratio, CI: confidence interval, MI: myocardial infarction. Note: adjusted for potassium and urea nitrogen

In addition, subgroup analysis and interaction effect analysis were used to assess their association stability. The results showed that their association was significantly observed in almost all the subgroups before adjustment (all p for interaction > 0.05, Table 4) except stratifying patients with MI status (p for interaction = 0.007,). We further explored their association stability after adjusting other variables. Because the stratified analysis was also a kind of method for controlling the confounding factors, hence we only adjusted the laboratory indicators related AF (potassium and urea nitrogen) and did not adjust these stratified variables. After adjustment, we found similar results with that before adjustment. The result of interaction analysis before and after adjustment indicated that MI and β-receptor blocker use had an interaction effect on the occurrence of AF (before: p for interaction = 0.007; after: p for interaction = 0.015).

Interaction effect exploration between MI and β-receptor blocker in AF

The above results suggested that there was an interaction effect between MI and the use of β-receptor blocker for AF. We further conducted both additive and multiplicative interaction effect analyses. The results from the interaction analysis based on the additive model showed that the values of S (95% CI), AP (95% CI), and RERI (95% CI) between the two factors were 0.283[0.142, 0.563], -1.187[-2.020, -0.354], and – 2.237[-3.722, -0.752], respectively. The multiplicative interaction analysis indicated that the OR (95% CI) between these two factors was 0.374[0.184, 0.772] (Table 5). The size and significance of the additive interaction between MI and β-receptor blocker use on AF were visualized in Figs. 3A, and 3B. The above results indicated a significant interaction effect between them and their effect was antagonistic.

Table 5 Analysis of interaction between MI and β-receptor blocker use in cardiac fibrillation

	Additive interaction			Multiplication interaction
index	S [95%CI]	AP [95%CI]	RERI [95%CI]	OR [95%CI]
value	0.283[0.142, 0.563]	-1.187[-2.020, -0.354]	-2.237[-3.722, -0.752]	0.374[0.184, 0.772]

Abbreviation: S: synergy index, AP: attributable proportion of interaction, RERI: relative excess risk of interaction, CI: confidence interval, OR: odd ratio, MI: myocardial infarction

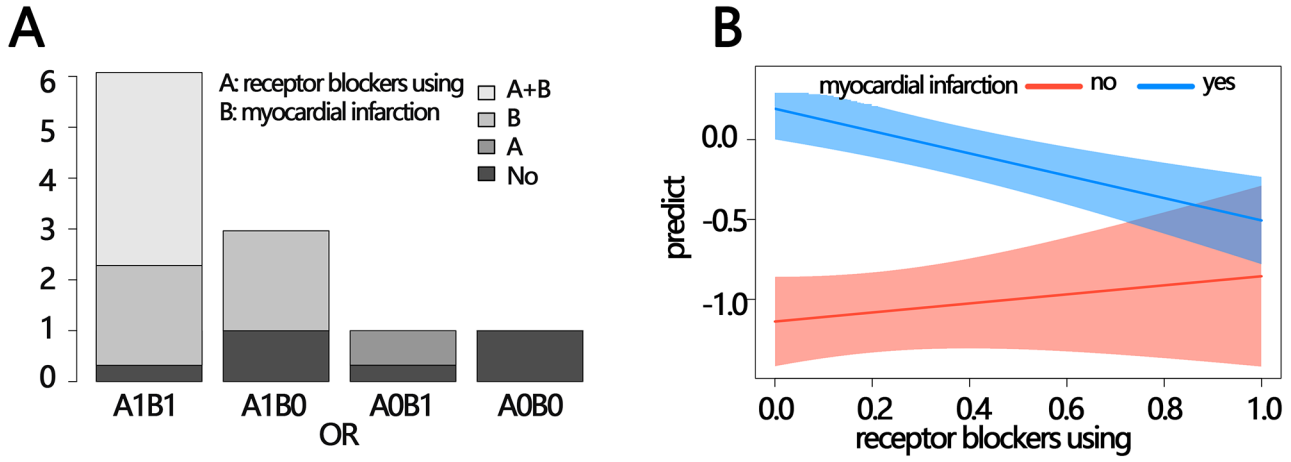

Fig. 3 Visualization of additive interaction analysis. (**A**) Strip plot of interaction size: the X-axis represents different combinations of factors, while the Y-axis shows the distribution of the OR for AF occurrence, visually illustrating the distribution of the dependent variable or effect size under different conditions. (**B**) Interaction significance plot: the X-axis represents the factor of β-receptor blocker use, while the Y-axis indicates the OR for AF occurrence. The different colored lines represent MI factors. The intersection of the two lines visually demonstrates the interaction effect between the two factors

Discussion

The β-receptor blocker is commonly used in the treatment of HF to improve cardiac function, reduce symptoms, and delay the course of the disease. It can also treat AF, however, its role in the development of AF is unclear in patients with HF. The study explored the effect of β-receptor blocker on AF in patients with HF based on the MIMIC-IV database. We found that β-receptor blocker use significantly contributed to the development of AF in patients with HF. In addition, the study revealed that there was an antagonistic effect between β-receptor blocker use and MI on the occurrence of AF.

β-blockers treat HF primarily by inhibiting adrenergic receptors, thereby reducing heart rate, myocardial contractility, blood pressure, and oxygen consumption while protecting the heart from catecholamine-induced damage [15]. Studies suggest that β-blockers may reduce adverse outcomes, including AF, in HF patients. This effect may be attributed to their ability to counteract potassium loss induced by diuretics, which is a known risk factor for AF [16, 17]. By inhibiting the renin-angiotensin-aldosterone system, β-blockers may help retain potassium and thus lower AF risk [18, 19].

However, our study found that β-blocker use was associated with a higher incidence of AF in HF patients. A possible explanation is the severity of HF in our cohort, as patients from the MIMIC-IV database were often critically ill and required ICU admission. Higher β-blocker doses in this population may have excessively reduced ventricular rate, leading to increased atrial pressure, atrial stretch, and electrical remodeling, thereby triggering AF [20]. Moreover, severe HF induces cellular stress and systemic inflammation, activating pathways such as the unfolded protein response and mitogen-activated protein kinase signaling, which may further contribute to AF development [21, 22].

In addition, the β-receptor blocker slows the heart rate, reduces the heart's oxygen consumption, and improves myocardial contractility by depressing the sympathetic nervous system. However, this slowing of the heart rate may lead to delayed atrioventricular conduction and may increase the risk of AF, especially in patients with HF who already have atrioventricular block or structural changes in the atria [11]. Although β-receptor blocker slow atrial excitability, their prolonged use may lead to structural and electrophysiologic changes in the atria, particularly dilatation of the left atrium and delayed electrical conduction. Such changes sometimes contribute to the development of AF [12]. In some patients with HF, especially those with hypertension or chronic coronary artery disease, β-receptor blocker may trigger the development of AF to some extent by reducing sympathetic action and inhibiting normal cardiac responses [13].

In addition, we have found that β-receptor blocker use was antagonistic to MI in the development of AF in patients with HF. MI, is usually defined as a clinical syndrome of myocardial ischemia and necrosis due to interruption of coronary blood flow supply [23]. The pathogenesis of MI is complex and involves multiple physiologic and biochemical processes, including rupture of coronary atherosclerotic plaques, thrombosis, coronary artery spasm, and obstruction of coronary blood flow [24]. β-receptor blocker is widely used after acute MI by slowing heart rate, reducing myocardial oxygen consumption, and improving ventricular function. The use of this class of drugs helps to reduce the occurrence of post-infarction arrhythmias, including AF [11].

In patients with HF, β-receptor blocker reduces the incidence of post-infarction AF by improving cardiac

stability and reducing ventricular remodeling. Post-infarction, especially the development of ventricular dilatation and myocardial fibrosis, may lead to electrophysiologic changes in the atria and increase the risk of AF. However, β-receptor blocker can reduce atrial hyperexcitability by slowing down the electrical activity of the atria, thus reducing the incidence of post-infarction AF to a certain extent [13]. this effect of β-receptor blocker may be particularly important in patients with HF combined with infarction, especially in those with a history of previous infarctions. The use of β-receptor blocker in patients with HF helps to restore the sympathetic-parasympathetic balance and reduce excessive sympathetic activation of the heart [25]. This balance restoration helps reduce the risk of AF triggered by sympathetic overexcitation, especially in patients with post-infarction [26, 27].

Strengths and limitations

The main strength of this study was that the interaction analysis revealed an antagonistic effect on AF between MI and β-receptor blocker use in HF patients. In addition, the study explored the importance of the use of β-receptor blocker in AF with machine learning methods. However, the study still has three limitations. The study did not take into account the specific drug and duration of the blocker, and the dosage of the drug, due to missing data. Second, the research objects of this study are all Europeans, and it is uncertain whether the conclusion can be promoted to the world. Third, the New York Heart Association classification of HF and ejection fraction were not considered in the study because data were not available.

Conclusion

In conclusion, it is recommended to highlight the relationship between beta-blocker use and the occurrence of atrial fibrillation, as well as the antagonistic effect with myocardial infarction. This complex interrelationship reveals that the use of β-receptor blocker not only affects the symptoms and prognosis of HF, but may also modulate the occurrence of AF through its effects on cardiac electrophysiology. Therefore, the use of β-receptor blocker should be individualized according to the patient's specific situation, taking into account the risk of HF, infarction, and AF.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12872-025-04697-6.

Supplementary Material 1

Acknowledgements
Not applicable.

Author contributions
YW contributed to the conception and design. YW and KNL contributed to the collection and assembly of data. YW and KNL analyzed and interpreted the data. All authors wrote and approved the final manuscript.

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Data availability
The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
The Ethics Committee of Ningbo Medical Center LiHuiLi Hospital deemed that this research is based on open-source data, so the need for ethics approval was waived. The study was performed in accordance with the Declaration of Helsinki.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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