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Article

Application Evaluation of Esmolol Hydrochloride-Assisted Interventional Therapy on New-Onset Atrial Fibrillation in Patients with Severe Sepsis: A Retrospective Study

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Abstract

Objective: This study aims to explore the application effect of adjuvant therapy with esmolol hydrochloride on new-onset atrial fibrillation (NOAF) in patients with severe sepsis. **Methods**: Retrospective analysis was conducted on 170 patients with NOAF and severe sepsis admitted to our hospital from January 2022 to January 2023. After excluding eight patients who did not meet the inclusion criteria, the remaining 162 patients were included in the study. Based on different treatment methods, the patients were divided into the control group (n = 83, routine treatment) and the observation group (n = 79, esmolol hydrochloride in combination with routine treatment). The cardiac function indexes such as left atrial diameter, left ventricular end-diastolic dimension, left ventricular end-systolic dimension and left ventricular ejection fraction; left atrial wall tension-related indexes, including atrial natriuretic peptide, B-type natriuretic peptide and N-terminal pro-B-type natriuretic peptide; inflammatory factors, including a C-reactive protein, high-sensitivity C-reactive protein, interleukin-6 and procalcitonin and the incidence of adverse reactions were compared between the two groups. Statistical methods were used to process and compare the above-mentioned data and index results. **Results**: No significant difference was observed in cardiac function indexes, left atrial wall tension-related indexes and inflammatory factors in both groups before treatment ($p > 0.05$). After treatment, the observation group had lower cardiac function indexes, left atrial wall tension-related indexes and inflammatory factors than the control group ($p < 0.05$), and the incidence of adverse reactions in the two groups was similar ($p < 0.05$). **Conclusion**: The adjuvant therapy with esmolol hydrochloride has a certain clinical effect on patients with severe sepsis and NOAF, which improves the cardiac function of such patients and reduces their inflammation levels up to a point, showing a clinical application value.

Keywords

esmolol hydrochloride; severe sepsis; new-onset atrial fibrillation

Introduction

Clinically, sepsis is a common critical illness and a fatal organic dysfunction induced by a dysregulated host response to infection [1]. Although its mortality has declined over the past few decades, morbidity remains high [2]. Severe sepsis frequently occurs in cardiovascular complications. New-onset atrial fibrillation (NOAF) is the most common arrhythmia in the course of sepsis and a crucial incentive of complications such as heart failure and thrombosis [3]. Severe sepsis combined with NOAF can lead to death because NOAF leads to the deterioration of cardiac function and hemodynamic disturbance, greatly increasing therapeutic difficulty and mortality hazard. Inflammation, electrolyte disturbance, immunosuppression and iatrogenic factors (including vasopressor) are responsible for the elevated risk of NOAF in patients with sepsis [4,5]. Given the rapid changes in the conditions, high mortality and multiple uncertainties in patients with sepsis and NOAF, clinical treatment must constantly explore and improve the means with rapid onset and comprehensive efficacy. Based on imaging diagnosis, interventional therapy utilises catheters, puncture needles and other interventional devices to treat the disease through the guidance of medical imaging equipment, and it has extensive application potential in the treatment of vascular diseases, which reduces surgical risk and the damage to organs and tissues. However, interventional therapy in patients with severe sepsis and NOAF must be accompanied with drugs to increase the efficacy. At present, anti-arrhythmic drugs such as amiodarone and propafenone are widely used in this disease, which are useful for restoring atrial and ventricular arrhythmias and sinus rhythm, but the effect is not very satisfactory. Anticoagulation therapy is prone to complications, including bleeding and thrombocytopenia. Esmolol hydrochloride is

a fast-acting and highly selective β1-adrenergic receptor blocker [6]. Based on medical and pharmacokinetic analyses, this drug could treat atrial fibrillation and control the ventricular rate, with the advantages of fast action onset, short half-life, eliminated half-time and mild and one-time adverse effects, but its ability to improve patients' conditions remains unclear. The reason is that the utilisation of β-blockers in the treatment of clinical sepsis with NOAF is not yet widespread, and β-blockers are relatively contraindicated for septic shock because of the effect of cardiac suppression. Based on the shortcomings of current therapeutic agents, this study investigates the efficacy of adjuvant therapy with esmolol hydrochloride in patients with NOAF and severe sepsis to explore more effective treatments.

Materials and Methods

General Data

As a retrospective analysis, 170 patients with severe sepsis and NOAF admitted to our hospital from January 2022 to January 2023 were included, and the remaining 162 patients were finally included in the study by excluding the eight cases that did not meet the inclusion criteria. The patients were divided into the control group (n = 83) and the observation group (n = 79) in accordance with different treatment methods. This study has been approved by the ethics committee of the Fourth Hospital of Hebei Medical University (approval no.: 2020ky327). As a retrospective analysis, the informed consent of patients was not required.

The inclusion criteria were as follows: (1) the duration of atrial fibrillation exceeded 30 s as recorded by the surface electrocardiogram or single-lead electrocardiogram; (2) the onset time of NOAF was less than 24 h; (3) the age of patients was more than 18 years old; (4) patients had complete clinical data. The exclusion criteria were as follows: (1) patients with permanent pacemaker; (2) those with abnormal thyroid function; (3) those with cardiogenic shock; (4) those with a history of bronchial asthma.

Treatment Methods

The control group underwent routine treatment. For the routine treatment of sepsis, the following methods were taken: (1) In early fluid resuscitation, normal saline, ringer lactate solution (manufacturer: SJZ No. 4 Pharmaceutical Co., Ltd.; specification, 500 mL; NMPA approval no. H20044961; origin: Shijiazhuang, China) and other medicines expanded blood volume, corrected shock and improved the prognosis. (2) Another method is life-sustaining therapy. In this method, mechanical ventilation and nutrition support were used to give patients the necessary life support. (3) During clearance of pathogenic pathogens, levofloxacin (manufacturer: Daiichi Sankyo Pharmaceutical [Beijing] Co., Ltd.; specification: 0.5 g; NMPA approval no.: H20040091; origin: Beijing, China), moxifloxacin hydrochloride and sodium chloride injection (manufacturer: Jiangsu Chia Tai Fenghai Pharmaceutical Co., Ltd.; specification: 250 mL:0.4 g of moxifloxacin and 2.0 g of sodium chloride; NMPA approval no.: H20183412; origin: Nanjing, China) as well as other drugs were used for antibacterial treatment. Surgical treatment such as drainage, debridement, local resection and suture of perforation could remove infected focus and drain the purulence if the treatment effect was not ideal. (4) Another method is the use of vasoactive agents. In this method, norepinephrine (manufacturer: Tianjin King York Pharmaceutical Co., Ltd.; specification: 1 mL:2 mL; NMPA approval no.: H12020621; origin: Tianjin, China) maintained the blood pressure level and blood supply of important organs such as the heart and brain as well as reduced mortality. (5) The last method is the suppression of inflammatory factors. In this method, dexamethasone (manufacturer: Tianjin Lisheng Pharmaceutical Co., Ltd.; origin: Tianjin, China; specification: 0.75 mg; NMPA approval no.: H2020122) inhibited the secretion and release of inflammatory cytokines *in vivo* and reduced the inflammatory response.

The routine treatment of NOAF included medical and interventional treatments. For medical treatment, 150 mg of amiodarone (manufacturer: Shandong Fangming Pharmaceutical Group; specification: 2 mL:150 mg; NMPA approval no.: H20044923; origin: Heze, China) was used through intravenous injection, followed by a lower dose (60 mg/h) for 6 h and finally 30 mg/h for 18 h.

With regard to interventional therapy, under local anaesthesia, patients were given fentanyl citrate (manufacturer: Jiangsu Nhwa Pharmaceutical Co., Ltd.; specification: 10 mL:0.5 mg; NMPA approval no.: H20113508; origin: Xuzhou, China) for sedation and analgesia, and heparin anticoagulation maintained activated coagulation at 300–500 s. Cold saline perfusion under the guidance of a three-dimensional mapping system was performed. Under local anaesthesia, a 10-level mapping catheter was placed through the right jugular vein (or a four-level mapping catheter through the left femoral vein) in the coronary sinus. The atrial septum was punctured through the right femoral vein, and then a Swartz sheath was placed behind the left atrium. The first dose of intravenous heparin (5000 U) was injected, followed by additional heparin (1000 U) per hour. The left atrial and pulmonary vein models were reconstructed in accordance with the CARTO system (Johnson & Johnson, CART03, NJ, USA), and a four-level ablation catheter of cold saline perfusion (7.5F Navistar, Biosense Webster, power: 30–35 W, temperature: 43°C, flow rate of heparinised saline: 17 mL/min) was sent for circumferential pulmonary vein ablation at 0.5–2.0 cm from the pulmonary vein, with the discharge at each point more than 30 s. After completing bilateral circumferen-

tial pulmonary vein ablation, a 10-level annular electrode catheter (Biosense Webster, EPQ7P012, CA, USA) similar to the size of the pulmonary vein was selected to mark the pulmonary vein potential of the left upper, left lower, right upper and right lower positions until complete pulmonary vein isolation. If there was a basis for typical cavotricuspid isthmus-dependent atrial flutter before and during surgery, then linear ablation was performed at the isthmus connecting the tricuspid annulus and inferior vena cava until bidirectional conduction block occurred.

The observation group was treated with esmolol hydrochloride (manufacturer: Qilu Pharmaceutical [Hainan] Co., Ltd.; specification: 10 mL:0.1 g; NMPA approval no.: H20066758; origin: Haikou, China) based on routine treatment. An optional load dosage (500 μg/kg for 1 min) was given, followed by continuous infusion at a dose of 50 μg/kg/min for 4 min. If necessary, an optional load dosage was given, followed by a continuous infusion of 100 μg/kg/min or 150 μg/kg/min for 4 min until the target heart rate was reached.

The efficacy was compared after 1-week treatment between the two groups.

Measurement

Collection of Basic Information

The researchers interviewed the patients and their families through interviews and questionnaires. They also collected general data such as gender, age and acute physiology and chronic health evaluation (APACHE) II score.

Collection of Observation Indexes

The cardiac function, left atrial wall tension-related indexes and inflammatory factors of patients after admission and 1-week treatment were collected as follows:

(1) Cardiac function. A Philips 7C colour Doppler ultrasound diagnostic instrument (manufacturer: Jiangsu Anmao Medical Technology Co., Ltd.; model: EPIQ7C; NMPA approval [I]: 20193062262; origin: Xuzhou, China) and S5-1 probe were used, with a frequency of 2–3.5 MHz. The patients maintained a left lateral position and breathed calmly for connecting thoracic lead electrocardiogram. Satisfactory left ventricular cross-section in the long axis, papillary muscular horizontal section in the short axis and cardiac cross-section of apical four chambers were obtained. The left atrial diameter (LAD, normal range: <35 mm), left ventricular end-diastolic dimension (LVEDD, normal range: 45–55 mm in males and 35–50 mm in females), left ventricular end-systolic dimension (LVESD, normal range: 25–37 mm in males and 20–35 mm in females) and left ventricular ejection fraction (LVEF, normal range: 50%–70%) were measured by using the Simpson method.

(2) Left atrial wall tension-related indexes. Fasting venous blood (3 mL) was taken for static condition at room temperature for 40 min and centrifuged at 3000 r/min for 15 min to separate serum. The enzyme-linked immunosorbent assay detected atrial natriuretic peptide (ANP, normal range: <100 pg/mL) and B-type natriuretic peptide (BNP, normal range: <100 pg/mL), and the electrochemiluminescence immunoassay tested N-terminal pro-brain natriuretic peptide (NT-proBNP, normal range: <125 pg/mL).

(3) Serum inflammatory factors. Fasting venous blood (3 mL) was collected for static condition at room temperature for 40 min and centrifuged at 3000 r/min for 15 min to obtain serum. Then, serum C-reactive protein (CRP, normal range: <10 mg/L), high-sensitive C-reactive protein (hs-CRP, normal range: <1.0 mg/L), interleukin-6 (IL-6, normal range: <7 pg/L) and procalcitonin (Pct, normal range: <0.05 ng/mL) levels were measured by enzyme-linked immunosorbent assay.

(4) Adverse reactions. The medical staff recorded the number of adverse reactions such as nausea, vomiting, low heart rate, hypotension, pulmonary oedema and urinary retention in the two groups during the medication period and calculated the corresponding proportion.

Statistical Method

SPSS (version: 26.0; manufacturer: International Business Machines Corporation; origin: Armonk, NY, USA) was used to process the data collected in this study. The enumeration data were expressed as [n (%)] and tested by χ^2. Firstly, the Kolmogorov–Smirnov method was used to test the normality of measurement data, and those conforming to normal distribution were tested by using t test, which is expressed as (mean $\pm$ sd). In addition, those meeting skewed distribution were detected by non-parametric test, which is indicated as M (P_{25}, P_{75}). $p < 0.05$ indicated statistically significant difference.

Results

General Information

No significant difference was observed in general information such as gender and age in the two groups ($p > 0.05$, Table 1).

Comparison of Cardiac Function

Before treatment, no significant difference appeared in LAD, LVEDD, LVESD and LVEF ($p > 0.05$). After treatment, the observation group presented significantly lower LAD, LVEDD, LVESD and LVEF than the control group ($p < 0.05$), and the above-mentioned indices in the observation group were closer to normal levels (Table 2).

Table 1. Comparison of general information [M (P25, P75), n (%)].

Items	Control group (n = 83)	Observation group (n = 79)	χ^2/Z	*p*
Gender			0.083	0.773
Male	46 (55.42)	42 (53.16)		
Female	37 (44.58)	37 (46.84)		
Age [years, M (P_{25}, P_{75})]	58.00 (52.00, 64.00)	59.00 (54.00, 65.00)	−1.298	0.194
BMI [(kg/m^2, M (P_{25}, P_{75})]	21.00 (19.70, 21.90)	20.80 (19.40, 21.70)	−0.449	0.653
Highest body temperature [°C, M (P_{25}, P_{75})]	39.00 (38.60, 39.40)	39.00 (38.60, 39.60)	−0.730	0.465
APACHE II score [points, M (P_{25}, P_{75})]	28.00 (24.00, 33.00)	26.00 (24.00, 30.00)	−1.765	0.078
SOFA score [points, M (P_{25}, P_{75})]	10.00 (9.00, 12.00)	10.00 (8.00, 12.00)	−0.657	0.511
NOAF types			0.326	0.568
Paroxysmal NOAF	53 (63.86)	47 (59.49)		
Continuous NOAF	30 (36.14)	32 (40.51)		
Underlying diseases				
Cardiovascular diseases	21 (25.30)	18 (22.78)	0.140	0.708
Diabetes mellitus	9 (10.84)	8 (10.13)	0.022	0.882
Respiratory diseases	16 (19.28)	14 (17.72)	0.065	0.799
Chronic liver dysfunction	24 (28.92)	21 (26.58)	0.110	0.740
Chronic kidney disease	10 (12.05)	10 (12.66)	0.014	0.906
Place of residence			0.022	0.881
City	43 (51.81)	40 (50.63)		
Town	40 (48.19)	39 (49.37)		
Smoking history			0.321	0.571
Yes	33 (39.76)	28 (35.44)		
No	50 (60.24)	51 (64.56)		
Drinking history			0.202	0.653
Yes	46 (55.42)	41 (51.90)		
No	37 (44.58)	38 (48.10)		
Education level			0.515	0.972
College and above	4 (4.82)	5 (6.33)		
Junior college	8 (9.64)	6 (7.59)		
Senior high school	19 (22.89)	20 (25.32)		
Junior high school	24 (28.92)	23 (29.11)		
Primary school or below	28 (33.73)	25 (31.65)		

NOAF, new-onset atrial fibrillation; BMI, body mass index; SOFA, sequential organ failure assessment score; APACHE, acute physiology and chronic health evaluation.

Left Atrial Wall Tension-Related Indexes

Table 3 displays the measurement of left atrial wall tension-related indexes such as ANP, BNP and NT-proBNP in the two groups, showing no difference in both groups before treatment ($p > 0.05$), and the above-mentioned indexes were lower than those in the control group ($p < 0.05$).

Serum Inflammatory Factors

Before treatment, the levels of serum inflammatory factors such as CRP, hs-CRP, IL-6 and Pct were measured in the two groups, and the results showed no difference between the two groups ($p > 0.05$). After completing the treatment, further measurements found lower levels of serum inflammatory factors in the observation group than in the control group ($p < 0.05$, Table 4).

Incidence of Adverse Reactions

After adding esmolol hydrochloride, Table 5 displays no difference in the incidence of adverse reactions of all patients ($p > 0.05$).

Discussion

After treatment, this study found that compared with the control group, the levels of cardiac function indexes, left atrial wall tension-related indexes and inflammatory factors in the observation group were lower ($p < 0.05$), indicating that the patients in the observation group had a better recovery of cardiac function indexes and left atrial wall tension-related indexes. To a certain extent, it can be explained that esmolol hydrochloride seems to be helpful in the adjuvant treatment of NOAF.

Table 2. Comparison of cardiac function.

Indexes	Time	Control group (n = 83)	Observation group (n = 79)	Z	*p*
LAD (mm)	Before treatment	53.00 (48.00, 57.00)	54.00 (49.00, 58.00)	−1.320	0.187
	After treatment	46.00 (43.00, 50.00)	40.00 (36.00, 43.00)	−7.998	<0.001
LVEDD (mm)	Before treatment	67.00 (65.00, 69.00)	66.00 (65.00, 68.00)	−0.962	0.336
	After treatment	63.00 (61.00, 65.00)	56.00 (52.00, 58.00)	−10.436	<0.001
LVESD (mm)	Before treatment	53.00 (49.00, 58.00)	52.00 (48.00, 58.00)	−0.702	0.483
	After treatment	50.00 (47.00, 52.00)	43.00 (40.00, 45.00)	−9.795	<0.001
LVEF (%)	Before treatment	80.00 (76.00, 83.00)	79.00 (77.00, 83.00)	−0.089	0.929
	After treatment	74.00 (71.00, 76.00)	70.00 (68.00, 71.00)	−7.009	<0.001

LAD, left atrial diameter; LVEDD, left ventricular end-diastolic dimension; LVESD, left ventricular end-systolic dimension; LVEF, left ventricular ejection fraction.

Table 3. Left atrial wall tension-related indexes.

Indexes	Time	Control group (n = 83)	Observation group (n = 79)	Z	*p*
ANP (pg/mL)	Before treatment	287.00 (238.00, 352.00)	286.00 (248.00, 333.00)	−0.553	0.580
	After treatment	257.00 (221.00, 279.00)	162.00 (150.00, 182.00)	−10.986	<0.001
BNP (pg/mL)	Before treatment	342.00 (324.00, 367.00)	352.00 (320.00, 374.00)	−0.464	0.643
	After treatment	228.00 (209.00, 246.00)	176.00 (161.00, 195.00)	−9.921	<0.001
NT-proBNP (pg/mL)	Before treatment	353.00 (328.00, 371.00)	350.00 (319.00, 371.00)	−0.794	0.427
	After treatment	256.00 (239.00, 277.00)	204.00 (175.00, 227.00)	−9.562	<0.001

Table 4. Serum inflammatory factors.

Indexes	Time	Control group (n = 83)	Observation group (n = 79)	Z	*p*
CRP (mg/L)	Before treatment	83.00 (74.00, 96.00)	82.00 (70.00, 97.00)	−0.142	0.887
	After treatment	70.00 (63.00, 80.00)	52.00 (35.00, 68.00)	−6.446	<0.001
hs-CRP (mg/L)	Before treatment	31.00 (20.00, 42.00)	29.00 (18.00, 39.00)	−1.024	0.306
	After treatment	11.00 (7.00, 17.00)	6.00 (4.00, 9.00)	−6.969	<0.001
IL-6 (pg/mL)	Before treatment	374.00 (329.00, 410.00)	383.00 (339.00, 417.00)	−0.757	0.449
	After treatment	74.00 (64.00, 96.00)	42.00 (36.00, 50.00)	−10.624	<0.001
Pct (ng/mL)	Before treatment	4.24 (3.28, 5.13)	3.97 (2.84, 5.11)	−1.086	0.278
	After treatment	2.83 (2.01, 3.98)	2.57 (1.66, 3.21)	−2.878	0.004

Table 5. Incidence of adverse reactions.

Groups	Bradycardia	Low heart rate	Hypotension	Pulmonary oedema	Urinary retention	Total incidence rate
Control group (n = 83)	2 (2.41)	2 (2.41)	1 (1.20)	1 (1.20)	3 (3.61)	9 (10.84)
Observation group (n = 79)	3 (3.80)	3 (3.80)	4 (5.06)	1 (1.27)	3 (3.80)	14 (17.72)
χ^2	–	–	–	–	–	1.572
p	–	–	–	–	–	0.210

Sepsis, a common disease, has high morbidity and mortality [7,8]. The clinical manifestations of sepsis are chills, fever (or hypothermia), polypnea, hypouresis, mental confusion and other symptoms [9]. Based on the severity, sepsis can be divided into sepsis, severe sepsis and septic shock. When developing into severe sepsis and septic shock, signs and symptoms involve multiple organ systems and cause neurological complications, endangering the lives of patients [10–12]. Patients with severe sepsis are in critical conditions, and they often need to be admitted to the intensive care unit (ICU) for comprehensive treatment and management to detect and control the development of the disease in time. Atrial fibrillation is the most frequent arrhythmia in ICU. NOAF can be induced by accelerated atrial remodelling during critical illness, and its main features include acute loss of atrial contraction and attack of rapid ventricular rates [13]. Therefore, NOAF is a sign of disease severity, as well as a potential factor leading to poor prognosis. NOAF during critical illness is associated with acute hemodynamic changes and short-term and long-term increases in the risk of stroke, heart failure and death. Assessing the structure and function of the left atrium in patients with NOAF is helpful for risk stratification, guiding treatment and prognostic evaluation. The cardiac functions of patients were evaluated from imaging and biochemical indicators, manifesting that the changed left

atrial structure and function (i.e., remodelling) of patients with NOAF involved diversiform molecular and pathological mechanisms, including cardiac function, left ventricular wall tension-related indexes and inflammatory factors. In the observation group, cardiac function indexes (LAD, LVEDD, LVESD and LVEF), left atrial wall tension-related indexes (ANP, BNP and NT-proBNP) and serum inflammatory factors (CRP, hs-CRP and IL-6, Pct) all decreased significantly than the control group (all $p < 0.05$), confirming the certain improvement effect of esmolol hydrochloride on cardiac function and inflammatory factors in patients with NOAF. The reasons for the above-mentioned research results are as follows: (1) Improvement of cardiac function in patients with NOAF. Esmolol hydrochloride, a short-acting β1 receptor blocker with selectivity, diminishes myocardial metabolic needs before and during cardiac arrest and alleviates ischemia–reperfusion injury. Meanwhile, esmolol utilises the natural cardiac pump function to attain coronary microcirculation and protect the heart immediately before cardiac arrest [14]. Esmolol hydrochloride mainly and competitively antagonises β1 receptor in myocardial cells and reduces the atrial fibrillation threshold via blocking sympathetic nerve excitation, thereby exerting pharmacological effects. Electrophysiological studies suggest that esmolol hydrochloride has a typical β1 receptor blocker effect, that is, it reduces heart rate; prolongs sinus cycle, sinoatrial node recovery time, AH interval and Wenckebach cycle; slows down the conduction of atrial muscle, ventricular muscle, cardiac conduction system and other parts; extends the atrioventricular refractory period and reduces myocardial oxygen demand. A clinical study by Wang *et al*. [15] has also shown that esmolol exerts multifarious potential therapeutic effects on patients with sepsis, including improving cardiac function and coagulation disorders, inhibiting the production of inflammatory factors and correcting tachytrophism. (2) Improvement of serum inflammatory factors in patients with NOAF. Esmolol hydrochloride has a significant inhibitory effect on myocardial injury caused by oxygen free radicals in patients, so it can reduce the levels of inflammatory factors in patients, thereby strengthening the heart, inducing diuresis, regulating neuroendocrine and humoral factors by dilating renal blood vessels and increasing renal blood flow. Furthermore, during the occurrence and development of sepsis, the excitement of sympathetic nerves evokes abundant catecholamines, which induce the expression of downstream inflammatory factors via β1 receptor, resulting in increased levels of inflammatory factors such as CRP and IL-6 in plasma. As an effective β1 receptor blocker, esmolol hydrochloride can block this pathway and produce anti-inflammatory effects. The clinical study of Cocchi *et al*. [16] showed that esmolol hydrochloride may have an effect on inflammation and immune response. Compared with the control group, the patients treated with esmolol had lower CRP levels after 12 h and 24 h, and these results are similar to those obtained in this study. (3) Less side effects in patients with NOAF. The side effects in the observation group did not increase significantly after adding esmolol hydrochloride because esmolol could be hydrolysed and metabolised by esterases in the erythrocyte matrix, and the metabolites had no β-receptor blocking effect in normal people.

The development of this study seems to confirm the efficacy of esmolol hydrochloride in the adjuvant treatment of NOAF and severe sepsis to a certain extent, which might be applied in the treatment of this disease. Meanwhile, this study has certain guiding value for expanding new ideas for clinical treatment, and clinically, the treatment methods of non-common diseases should be actively studied. The limitations of this study included that due to the low incidence of NOAF and severe sepsis, the number of participants selected in this study was limited, which might affect the promotion of research results. Therefore, future research should increase the selection of research participants. Limited by time, financial resources, manpower and other aspects, this study lacked long-term follow-up, so subsequent studies should extend the study time and carry out long-term follow-up to evaluate the therapeutic effect more comprehensively. Additionally, as a single-center study, the results may be disturbed by regional factors, and multi-center research could be carried out.

Conclusion

Esmolol hydrochloride can be used as an important drug for the treatment of arrhythmia in patients with sepsis and NOAF. However, the application of this drug should attach importance to monitoring heart rate, blood pressure and cardiac function, adjusting the maintenance dose in accordance with the hemodynamic status of patients, grasping the indications and contraindications and standardizing rational drug use.

Availability of Data and Materials

Data to support the findings of this study are available on reasonable request from the corresponding author.

Author Contributions

LS and JS performed the research. JS provided help and advice on the experiments. LS contributed to the analysis and interpretation of the data. Both authors contributed to editorial changes in the manuscript. Both authors read and approved the final manuscript. Both authors have participated sufficiently in the work to take public responsibility for appropriate portions of the content and agreed to be accountable for all aspects of the work in ensuring that questions related to its accuracy or integrity.

Ethics Approval and Consent to Participate

This study has been approved by the ethics committee of the Fourth Hospital of Hebei Medical University (approval no.: 2020ky327). As a retrospective analysis, the informed consent of patients was not required.

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Conflict of Interest

The authors declare no conflict of interest.

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