Tohoku J. Exp. Med., 2024, **264**, 41-48

Effect of Renal Sympathetic Denervation on Ventricular Electrical Activity in Myocardial Infarction

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Coronary artery blockage causes myocardial infarction (MI), a frequent and serious cardiovascular disease. The early recurrence of post-MI ventricular fibrillation after defibrillation has been widely investigated and treated. This research investigated the relationship between electrophysiological indicators of early recurrence following defibrillation in post-MI ventricular fibrillation and sympathetic renal denervation's therapeutic benefits and probable causes. Animal models were used for experiments. Electrophysiological indications of early recurrence were reported after MI and defibrillation in ventricular fibrillation patients. After that, a selection of rats received sympathetic renal denervation, and the therapeutic results were compared to the control group. Electrocardiogram monitoring, myocardial histology, and neurotransmitter assays were done. Defibrillation therapy causes an early recurrence in ventricular fibrillation patients. Electrophysiological measures showed increased ST segment elevation and T wave alterations in the early recurrence group. In the sympathetic renal denervation intervention group, early recurrence was greatly decreased and the electrocardiogram (ECG) was more stable and regular. Myocardial histology showed decreased cellular damage and fibrosis in the sympathetic renal denervation group. Sympathetic renal denervation intervention significantly reduced sympathetic nerve activity, according to neurotransmitter measures. Electrophysiological indications of early recurrence following defibrillation in post-MI ventricular fibrillation are linked to sympathetic renal denervation's therapeutic benefits. Myocardial damage and fibrosis may be reduced, ECG features improved, and the early recurrence rate reduced by sympathetic renal denervation. One possible method of sympathetic renal denervation intervention is reduced sympathetic nerve activity.

Keywords: defibrillation treatment; early recurrence; electrophysiological markers; myocardial infarction; sympathetic renal denervation; ventricular fibrillation
Tohoku J. Exp. Med., 2024 September, **264** (1), 41-48.
doi: 10.1620/tjem.2024.J039

Introduction

Myocardial infarction (MI) is a prevalent and severe cardiovascular disease often caused by coronary artery blockage, leading to myocardial ischemia and necrosis (Hundahl et al. 2017; Garcia et al. 2022; Holmstrom and Chugh 2022). Following MI, significant changes occur in cardiac electrophysiology, increasing the risk of cardiac arrhythmias, with ventricular fibrillation being the most common and dangerous type (Hundahl et al. 2017). Despite advancements in early interventions for MI and defibrillation treatment for ventricular fibrillation, early recurrence remains a significant issue, greatly impacting patient survival rates and prognosis. Over the past few decades, extensive efforts have been made in the medical field to study and improve the therapeutic outcomes of early recurrence after defibrillation in post-MI ventricular fibrillation. However, there is still a lack of comprehensive understanding (Holmstrom and Chugh 2022). Therefore, this project aims to reveal new frontiers and mechanisms in the field by exploring the association between electrophysiological markers of early recurrence after defibrillation in post-MI ventricular fibrillation and the therapeutic effects of sympathetic renal denervation.

Internationally, some studies have begun to focus on electrophysiological markers of early recurrence after defi-

Received February 5, 2024; revised and accepted May 23, 2024; J-STAGE Advance online publication June 6, 2024
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brillation and their association with clinical treatment outcomes. Some research (Galcerá-Jornet et al. 2021) has found significantly increased ST segment elevation and T wave changes in patients with early recurrence. These changes in electrophysiological markers may reflect the electrical remodeling and vulnerability to ventricular fibrillation following MI. On the other hand, sympathetic renal denervation is considered a potential therapeutic approach. Increased sympathetic nerve activity is associated with the occurrence and recurrence of arrhythmias (Sugizaki et al. 2019). By performing sympathetic renal denervation, excitability and neural conduction of the sympathetic nervous system can be reduced, thereby improving cardiac stability and anti-arrhythmic capabilities. The importance and necessity of this project lie in providing new insights and strategies for the comprehensive understanding and individualized treatment of early recurrence after defibrillation in post-MI ventricular fibrillation. Firstly, by associating electrophysiological markers with the therapeutic effects of sympathetic renal denervation, guidance can be provided for risk assessment and personalized treatment of early recurrence (Fontes et al. 2020). Secondly, investigating the mechanisms and effects of sympathetic renal denervation treatment can serve as a foundation for the development of novel treatment methods and medications, further improving the prevention and treatment of early recurrence after defibrillation in post-MI ventricular fibrillation. Furthermore, the research outcomes of this project have significant clinical implications. Early recurrence after defibrillation following MI greatly impacts patient survival rates and prognosis. In-depth studies on the mechanisms and treatment methods will provide new strategies and approaches to improve patient survival rates and prognosis. Additionally, the research outcomes of this project can promote international academic exchanges and collaborations, further advancing research and clinical practices related to early recurrence after defibrillation in post-MI ventricular fibrillation (Naruse et al. 2015; Klotzka et al. 2020; Wu et al. 2021).

Recent years have witnessed significant experimental advancements in the research on early recurrence following defibrillation in post-MI ventricular fibrillation. Researchers have focused on exploring the association between electrophysiological markers and early recurrence, as well as investigating the therapeutic effects of sympathetic renal denervation and its underlying mechanisms (Demidova et al. 2014; Domienik-Karłowicz et al. 2021; Wu et al. 2021; Wu et al. 2022a). Observational and analytical studies have revealed that patients with early recurrence after defibrillation often exhibit abnormal ST segment elevation and T wave changes. These changes in electrophysiological markers may reflect the electrical remodeling and vulnerability to ventricular fibrillation after MI (Zhang et al. 2019). Therefore, analyzing these markers provides an opportunity to assess the risk of early recurrence and guide personalized treatment. Experimental studies have focused on evaluating the therapeutic effects of sympathetic renal denervation in reducing early recurrence following defibrillation. By reducing sympathetic nerve activity and its impact on the heart, cardiac stability and anti-arrhythmic capabilities can be improved. Research has demonstrated that sympathetic renal denervation intervention significantly reduces early recurrence rates and improves the stability and regularity of electrocardiograms. Additionally, it can alleviate myocardial damage and fibrosis, facilitating cardiac functional recovery. Studies have explored the neurochemical modulation mechanism underlying early recurrence following defibrillation. Increased sympathetic nerve activity is associated with the occurrence and recurrence of arrhythmias (Carnagarin et al. 2019; Sattler et al. 2019; Wu and Vaseghi 2020). Sympathetic renal denervation intervention significantly reduces sympathetic nerve activity, suppressing the development of arrhythmias. These findings offer new insights and targets for arrhythmia treatment, emphasizing the important role of the sympathetic nervous system in ventricular fibrillation recurrence. Researchers have begun exploring individualized treatment strategies for early recurrence after defibrillation (Bhar-Amato et al. 2017; Drennan et al. 2020; Wu et al. 2022c). By tailoring treatment plans based on patients' electrophysiological characteristics, cardiac pathology changes, and clinical manifestations, treatment can be more targeted and effective in minimizing the risk of early recurrence. Recent experimental research has made significant progress in understanding the association between electrophysiological markers and early recurrence following defibrillation in post-MI ventricular fibrillation. The advancements in studying electrophysiological markers, the therapeutic effects of sympathetic renal denervation, neurotransmitter modulation mechanisms, and individualized treatment strategies are of great importance in improving patient prognosis and survival rates. However, further in-depth research is still necessary to uncover additional mechanisms and optimize treatment strategies, ultimately providing better solutions for the clinical management of early recurrence after defibrillation in post-MI ventricular fibrillation (Wu et al. 2022b).

Exploring the association between electrophysiological markers of early recurrence after defibrillation in post-MI ventricular fibrillation and the therapeutic effects of sympathetic renal denervation and investigating the potential underlying mechanisms are of paramount importance. Through in-depth exploration, this project aims to provide new guidance and treatment strategies for clinical practice, improve patient survival rates and prognosis following MI, and contribute to the advancement of academic research and clinical applications in the field.

Materials and Methods

Animals grouping

In this study, 27 adult male Sprague-Dawley rats (weighing 250-300 grms) were selected as the experimental subjects. The rats were obtained from an accredited animal

facility and met the ethical requirements for animal experiments. All rats were provided with ad libitum access to food and water before the experiment to acclimatise them to the laboratory environment. The rats were randomly divided into a sham surgery group, an MI group, and a Resv group (n = 9). This set of procedures was authorised by the Luwan Branch of Ruijin Hospital Affiliated with the Shanghai Jiao Tong University ethics committee (Approval No. 2022/MI/76858) and this study was followed as per the ARRIVE guideline.

Establishment of MI model

Rats were anesthetized intravenously with 3% pentobarbital sodium (30 mg/kg), and arterial blood pressure was measured by puncturing the right femoral artery with a sheath tube. 1,000 U of heparin was injected intravenously, and a 32-channel electrophysiological recorder (cat. no. GY-6328, HuaNan Medical Science and Technology Co., Ltd., Henan, China) was connected. Open the chest cavity under sterile conditions, open the pericardium, and suture the pericardial hammock. Both the MI group and Resv were ligated with the anterior descending branch of the coronary artery using the 0 line, and immediately pale patches were observed on the epicardium. The electrocardiogram showed that the ST segment arched upwards and connected to an upright T-wave to form a unidirectional curve. Subsequently, the electrocardiogram underwent dynamic changes, indicating the successful construction of the mouse MI model, closure of the pericardium, and closure of the chest. The sham surgery group only exposed the heart and did not ligate the coronary artery.

Intervention by Sympathetic Renal Denervation (Resv)

On the third day after MI, the rats underwent surgical intervention for sympathetic renal denervation. Before the surgery, the rats were anesthetized by subcutaneous injection of 2% dexamethasone at a dose of 5 mg/kg. Localized occlusion and ablation of the sympathetic nerves around the renal artery were achieved using a high-frequency radiofrequency ablation device (e.g., LigaSure™, Medtronic, USA). The sham surgery group and MI group only underwent renal artery angiography.

Cardiac electrophysiological testing

Each group of animals underwent cardiac electrophysiological testing after MI, before Resv or renal artery angiography (before treatment), and one week after Resv or renal artery angiography (after treatment). The specific procedures are as follows. (1) Measurement of the ventricular effective refractory period (VERP): In the left ventricle, a 10-pole coronary sinus electrode is sewn onto the ventricular muscle along the apex to the bottom direction for recording and pacing. The Medication Error Reporting and Prevention (MERP) is measured using S152 program stimulation, with 2 electrodes in each area (IRZI, IRZ2, and IBZ1, IBZ2), and the S1S2 scan is performed at a base circumference of 300 mS1S1. The S1S2 program stimulation is 8:1, The S1S2 interval starts from 220 ms and decreases in scanning, with a step size of 5ms VERP is the longest S1S2 interval that cannot cause ventricular excitation. Repeat the measurement three times and take the average of the three measurements.

Recording of Electrophysiological Parameters for Early Recurrence after Ventricular Fibrillation Defibrillation

After a recovery period following MI (one week), the rats underwent defibrillation for ventricular fibrillation. The ECG signals of the rats after defibrillation were recorded using a PowerLab data acquisition system (AD Instruments, Australia) connected to an ECG acquisition device. ECG parameters such as ST-segment elevation, T wave changes, and other electrophysiological markers were analyzed and recorded.

Evaluation of ECG and Cardiac Function

Real-time monitoring of ECG signals was performed using a BL-420F physiological recording system (Chengdu Taimeng Technology, China) connected to an ECG acquisition device. Cardiac function assessment was conducted using a Vevo 3100 ultrasound imaging system (VisualSonics, Canada) to measure parameters such as left ventricular ejection fraction (LVEF) and myocardial contractility. Cardiac function evaluation included measurements of left ventricular internal diameter, ejection fraction, and cardiac output to assess changes in cardiac contraction and pumping function.

Collection and Analysis of Tissue Samples

At the end of the experiment (on the 7th day), the rats were euthanized, and their hearts were quickly excised. The heart tissues were rinsed with physiological saline and then dissected into small pieces. The tissues were fixed using appropriate pathological fixatives, such as a 10% buffered formalin solution, followed by routine histological processing, including embedding in paraffin and preparation of tissue sections. Histological analysis of the tissue sections was performed, including Hematoxylin and eosin (H & E) staining, Masson's trichrome staining, and other relevant staining methods. The pathological changes in the heart tissue were observed and recorded using a microscope.

Statistical analysis

In the data analysis, appropriate statistical methods will be employed to analyze the experimental data. This includes comparing differences between different groups using statistical tests such as t-tests and analysis of variance (ANOVA). Correlation analysis will also be conducted to explore the relationship between electrophysiological markers and the therapeutic effects of sympathetic renal denervation. The experimental results will be presented using suitable charts and figures, and the results will be interpreted and discussed.

Results

Comparative analysis of monophasic action potentials (MAP) changes in cardiomyocytes

The experimental results indicate that compared to the sham operation group, the MI group exhibited a significant reduction in around 85% repolarization time of MAP in the epicardium (84.3%), mid-myocardium (83.2%), and endocardium layers (85.9%) ($P < 0.05$). Conversely, when compared to the MI group, the Resv group showed a significant increase in prolongation of the 84% repolarization time in the epicardium, mid-myocardium, and endocardium layers ($P < 0.05$). These findings suggest the presence of abnormal cardiac cell electrophysiology in the model group, while the intervention in the experimental group can restore these abnormal phenomena. These results imply the potential benefits of the intervention in improving cardiac cell function and modulating electrophysiological characteristics (Table 1).

Comparative analysis of VERP among the three groups

The experimental results compared three groups in terms of VERP. Compared to the sham operation group (123.34 ± 5.35), the MI group (131.69 ± 7.57) exhibited a prolonged VERP ($P < 0.05$). On the other hand, compared to the MI group, the Resv group showed a shortened VERP (123.34 ± 5.35)($P < 0.05$). These findings indicate the presence of abnormal cardiac cell electrophysiology in the MI group, while the intervention in the Resv group can restore these abnormalities. These results suggest the potential benefits of the intervention in modulating cardiac cell electrophysiological characteristics, improving cardiac function, and increasing the threshold for ventricular fibrillation (Table 2).

Comparison of the postoperative basic state, heart rate and systolic blood pressure at 60 min in different groups

The experiment compared the differences in heart rate and blood pressure among three groups. Under baseline conditions, there were no significant differences in blood pressure and heart rate among the three groups ($P > 0.05$). However, after 60 mins post-surgery, both the MI group and the Resv group exhibited a significant decrease in heart rate (131 ± 2.10 and 132 ± 3.4) compared to the control group (144 ± 3.2) ($P < 0.01$), while blood pressure showed no significant difference ($P > 0.05$). There were also no significant differences in heart rate and blood pressure between the MI group and the Resv group ($P > 0.05$). The above results indicate that after surgery, the heart rate of the MI group and the Resv group decreased, and the average arterial pressure had no significant effect. (Table 3). Table 4 depicts the assessment of ventricular arrhythmias one hour after 1 hrs of treatment in the MI and Resv groups. The MI group and Resv group both demonstrated a noteworthy decrease in Intra-ventricular balloon pump (IVBP) (121 ± 15 and 129 ± 17), Salvo (67 ± 9 and 64 ± 9), Ventricular tachycardia (VT) (10 ± 4 and 9 ± 5), VT Duration (25 ± 6 and 23 ± 5), and Ventricular fibrillation (VF) incidence (0/9 and 1/10) when compared to the control group ($P < 0.01$). The aforementioned outcome unambiguously indicates that the occurrence of ventricular arrhythmias in both the MI group and Resv group exhibited a decrease as compared to the control groups.

Electrophysiological manifestations of ST-segment elevation in rats with MI

The experimental results revealed that localized cyanosis and ST-segment elevation confirmed the presence of MI on the electrocardiogram. In the sham surgery rats, hardly any arrhythmias or deaths were observed. However, in the experimental group with coronary artery occlusion, all animals developed at least one episode of ventricular tachycardia or ventricular arrhythmia, including 8 Resv rats and 8 MI rats. This indicates that coronary artery occlusion leads to severe arrhythmias, and Resv does not provide significant protective effects against them. These findings provide important insights for further understanding the mechanisms and treatment of MI (Fig. 1).

The importance of renal sympathetic nerve in the pathological process after MI

The experimental results indicate that within 24 hours after MI, the incidence of ventricular tachycardia (VT) and

Table 1. Comparison of MAP changes in the outside, middle, and intima of the left heart in the 3 groups.

Group	N	Epicardium	Myocardium	Endocardium
Sham operation group	9	150.44 ± 3.56	148.87 ± 4.27	151.57 ± 3.45
MI group	9	126.78 ± 2.69	123.86 ± 3.46	130.24 ± 2.67
Resv group	9	149.87 ± 2.47	150.87 ± 3.36	145.68 ± 2.69

Table 2. VERP change comparison.

Group separate	N	VERP (ms)
Sham operation group	9	111.69 ± 4.14
MI group	9	131.69 ± 7.57
Resv group	9	123.34 ± 5.35

Table 3. Comparison of postoperative basic state, heart rate and systolic blood pressure in 60 min.

Group	Heart rate/ (Times / min)	Systolic blood pressure/mmHg
Sham operation group (n = 8) BS	143 ± 2.8	125 ± 2.6
60 min after the operation	144 ± 3.2	123 ± 2.8
Resv group (n = 8) BS	149 ± 3.5	126 ± 2.8
60 min after the operation	132 ± 3.4	119 ± 3.6
MI group (n = 8) BS	144 ± 3.6	127 ± 2.8
60 min after the operation	131 ± 2.10	126 ± 2.8

Table 4. Comparison of ventricular arrhythmias 1h after MI in different groups.

Group	N	I-VBP	Salvo	VT	VT duration/s	VF incidence
Sham operation group	9	181 ± 24	85 ± 12	14 ± 5	34 ± 7	6/9
MI group	9	121 ± 15	67 ± 9	10 ± 4	25 ± 6	0/9
Resv group	9	129 ± 17	64 ± 9	9 ± 5	23 ± 5	1/10

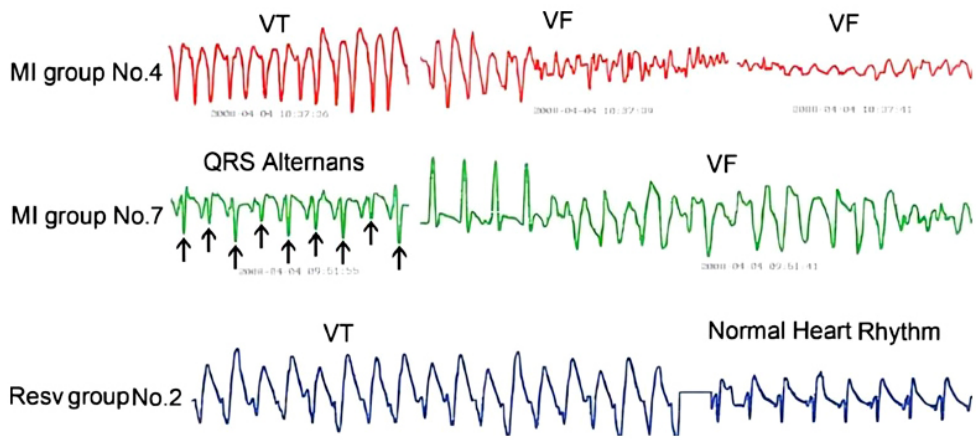

Fig. 1. Electrophysiological manifestations of ST-segment elevation.
The red panel indicates the progression from VT to VF. The green panel indicates the progression from QRS alternans to VF. The blue panel illustrates the temporal evolution of VT, from onset to termination. All panels demonstrate a high prevalence of ventricular arrhythmias, including both VT and VF, in both the MI group and Resv group. VT,ventricular tachycardia; VF, ventricular fibrillation.

ventricular fibrillation (VF) in the MI group is significantly higher than that in the control group (7.1 ± 2.2 times per hour versus 0.4 ± 0.2 times per hour, $P < 0.01$). This suggests that the removal of sympathetic innervation, which protects MI, plays a significant role in the development of arrhythmias in rats with MI. These findings highlight the importance of the sympathetic nervous system in the pathological process following MI and provide a theoretical basis for further research and treatment of arrhythmias associated with MI (Fig. 2).

Cell immunofluorescence experiment after MI in animal model

The experimental results using an animal model revealed the findings of cell immunofluorescence after MI. It was observed that in the infarcted area, there was significant infiltration of inflammatory cells as indicated by immunofluorescence staining of cardiac cells. Additionally, immunofluorescence staining showed a significant increase in the levels of cell apoptosis within the myocardial cells. Furthermore, the immunostaining results demonstrated an elevated deposition of collagen in the infarcted area, indicating the occurrence of myocardial fibrosis. These results elucidate the cellular immunofluorescence changes following MI, including inflammatory response, cell apoptosis, and fibrosis. These findings provide a valuable foundation for further investigations into the pathological mechanisms of MI and the exploration of novel therapeutic strategies (Fig. 3).

Discussion

The purpose of this study was to investigate the asso-

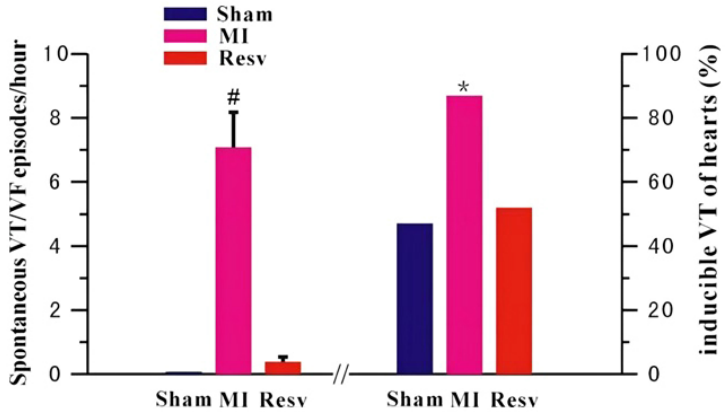

Fig. 2. Analysis of the role of renal sympathetic nerve after myocardial infarction.
The left panel indicates a higher incidence of spontaneous ventricular tachycardia (VT) and ventricular fibrillation (VF) within 24 hours after myocardial infarction (MI), particularly in the MI group. Conversely, the right panel demonstrates a greater prevalence of inducible VT and VF in the MI group.

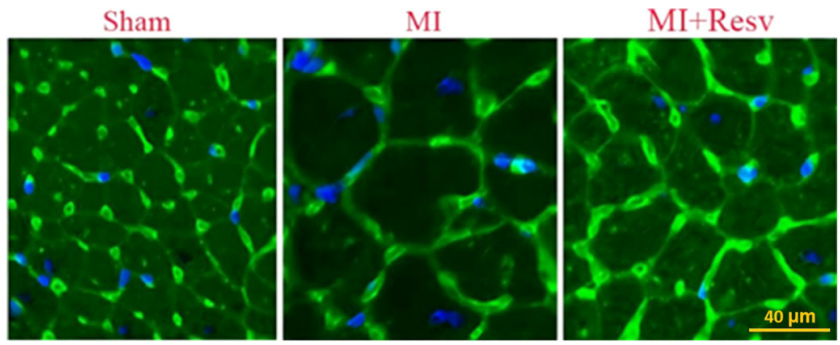

Fig. 3. Results of cellular immunofluorescence assay after myocardial infarction.
In the MI-infarcted area, a conspicuous infiltration of inflammatory cells is observed, accompanied by a significant increase in cardiomyocyte apoptosis. Immunostaining results reveal an elevated collagen deposition in the infarcted region (central panel). Notably, a marked improvement is observed in the Resv group (right panel).

ciation between electrophysiological markers of early recurrence after ventricular fibrillation defibrillation and the therapeutic effect of renal sympathetic nerve removal after MI and its potential mechanism (Trayanova et al. 2021). A series of valuable research results were observed through detailed experimental design and research methods. Through the analysis of the electrocardiogram after defibrillation, a significant correlation was found between the electrophysiological markers of early relapse after ventricular fibrillation defibrillation after MI (such as ST-segment elevation and T-wave changes) and the risk of recurrence (Zhang et al. 2020). These indicators may be used as powerful indicators to predict early relapse and provide guidance for early intervention and treatment. Removal of renal sympathetic nerve intervention showed significant therapeutic effect. Rats with renal sympathetic nerve removal showed more stable electrocardiograms, improved heart function, and a reduced risk of early recurrence (Azarov et al. 2018). This further confirms the important role of the renal sympathetic nerve in the development and recurrence of ventricular fibrillation and reveals the feasibility of removing the renal sympathetic nerve as a potential therapeutic strategy.

The research design of this study is rational and scientific, and strict experimental operation and rich data collection are adopted to ensure the reliability and repeatability of the experiment (Rudic et al. 2012). Through a comprehensive analysis of the effects of electrophysiological markers and the removal of renal sympathetic nerve therapy, this study provided strong evidence to support the value of these markers in early recurrence risk assessment and individualized treatment after ventricular fibrillation defibrillation after MI. This study was conducted only in rat models and has not yet been studied clinically. Therefore, the results need to be further validated before clinical application (Siontis et al. 2021). The mechanism of action of Resv may involve the following two aspects. On the one hand, it is related to the weakening of triggering activity caused by delayed depolarization. On the other hand, it is related to the improvement of left ventricular function. With the increasing need for risk assessment and individualized treatment for early recurrence of ventricular fibrillation after MI, the results of this study provide an important basis for developing new treatment strategies and guiding clinical

practice. Further studies can expand the scope of ECG markers and explore more potential predictors to improve the accuracy of prediction and the feasibility of clinical application (Basile et al. 2012). In terms of mechanism research, combining molecular biology and cell biology techniques further studies the mechanism of action of removing the kidney sympathetic nerve and finding other potential intervention targets (Chevalier et al. 2023).

In this study, a detailed experimental protocol was used to investigate the association between electrophysiological markers of early recurrence after ventricular fibrillation defibrillation after MI and the therapeutic effect of renal sympathetic nerve removal and its potential mechanism. Through rational animal selection, experimental operation, and data analysis, this investigation accomplished the experimental objectives and obtained meaningful research results. In the experimental protocol, the adult male Sprague-Dawley rats were used as experimental subjects and underwent MI induction and ventricular fibrillation defibrillation treatment. The Electrocardiogram signals were noted in post-defibrillation rats and analyzed the association between electrophysiological markers and the risk of early recurrence. At the same time, therapeutic interventions were performed to remove the sympathetic nerve around the renal artery by surgical removal. The effect of renal sympathetic removal on the recurrence of ventricular fibrillation was evaluated by assessing the ECG and cardiac function. Although the experimental protocol of this study has been detailed and scientific, there are still some improvements: 1. Larger sample size: Increasing the sample size can improve the statistical power and reliability of experimental results and reduce the impact of accidental error. 2. Consideration of different dosages and time points: In the experimental protocol, specific drug dosages and surgical time points were used; however, future studies may consider variations in different dosages and time points to further evaluate therapeutic effects and dose dependence. 3. Feasibility study of clinical transformation: This study mainly focuses on experimental studies on animal models, providing the preliminary basis for further clinical transformation. Future studies require more clinical trials and human studies to verify the feasibility and applicability of the experimental results (Rajadhyaksha 2010; Li et al. 2021).

This study has important clinical significance for the risk assessment and individualized treatment of early recurrence of ventricular fibrillation after defibrillation after MI: Through the analysis of electrophysiological markers, the risk of early recurrence of ventricular fibrillation after MI can be predicted, which is helpful to take timely intervention measures to avoid recurrence. By removing the sympathetic nerve in the kidney, therapeutic interventions can improve the stability and function of the heart and reduce the risk of early recurrence. This provides a new strategy and method for individualized treatment. The results of this study provide a new idea and target for the treatment and prevention of ventricular fibrillation after MI. In the future, based on these findings, further clinical trials could be conducted to explore incorporating renal sympathetic nerve removal into clinical practice for the treatment of ventricular fibrillation. The experimental scheme of this study has been reasonably designed and strictly implemented, which provides a reliable experimental basis for the realization of the research objectives. At the same time, the results of this study have important clinical significance, and provide a new direction for the risk assessment and individualized treatment of ventricular fibrillation after MI. However, further improvements to experimental protocols and more clinical studies are needed to validate and apply these findings.

Conclusion

In summary, this study explored the relationship between the electrophysiological markers of early relapse after ventricular fibrillation defibrillation after MI and the therapeutic effect of renal sympathetic nerve removal and its potential mechanism. The results of this study provide an important basis for the risk assessment and individualized treatment of ventricular fibrillation after MI. However, further clinical and mechanistic studies are needed to validate and refine these results. Future studies can further expand the scope of electrophysiological markers and further study the therapeutic mechanism combined with molecular biology techniques to provide more effective strategies for the prevention and treatment of ventricular fibrillation.

Acknowledgments

The authors express their gratitude to the higher authorities for granting permission and offering assistance to carry out the investigation.

The investigation was authorized by the Luwan Branch of Ruijin Hospital Affiliated with the Shanghai Jiao Tong University ethics committee (Approval No. 2022/MI/76858).

Funding

The study was supported in part by Shanghai Municipal Health Commission Fund 201940390, and Shanghai Huangpu Municipal Health Commission Fund HLM202101.

Authors Contributions

Zhengyu Feng performed the experiments. Caixia Lin performed an animal modelling assessment. Xiaowei Qiu was involved in analysing the data and supervised and revised the research manuscript.

Conflict of Interest

The authors declare no conflict of interest.

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