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Myocardial Infarction Model in Mice: Left Anterior Descending Coronary Artery Ligation

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Abstract: Background: A stable and feasible animal model for myocardial infarction (MI) is of great significance for the research on MI treatment. This study clarified the establishment process of the mouse MI model, aiming to enhance the efficiency of the modeling procedure. Method: Male C57 mice were randomly divided into a sham group and a model group, with 10 mice in each group. In the sham group, after anesthesia, the chest skin was incised to expose the heart, which was briefly exteriorized and then rapidly returned to the thoracic cavity before suturing. In the model group, MI was induced by ligation of the left anterior descending coronary artery. Subsequently, the 24-hour survival rate was recorded, heart rate, blood pressure, and electrocardiogram were measured, and myocardial infarct size was assessed by 2,3,5-triphenyltetrazolium chloride (TTC) staining. Result: After modeling, the 24-hour survival rate of mice in the model group was 70%. There was no significant difference in heart rate between the model group and the sham group, while both diastolic and systolic blood pressures were higher in the model group compared to the sham group. Compared with the sham group, the model group exhibited ST segment elevation and T wave inversion on the electrocardiogram. TTC staining revealed no infarcted areas in the hearts of the sham group, whereas distinct pale infarcted areas were observed in the model group. Conclusion: The method of inducing myocardial ischemia and necrosis by ligating the left anterior descending coronary artery of mice employed in this study proved to be a reliable approach for successfully establishing the model.

Keywords: Myocardial infarction; Mouse; Left anterior descending coronary artery ligation; Animal model; Disease research

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1. Introduction

Myocardial infarction (MI) is a severe cardiovascular disease characterized by a sudden reduction or cessation of blood flow in the coronary arteries, leading to persistent myocardial ischemia, hypoxia, and subsequent necrosis [1]. This condition impairs cardiac function and may result in severe complications such as arrhythmias, shock, and heart failure, posing a major threat to human health [2]. With the formation of an

aging society in our country, cardiovascular and cerebrovascular diseases have gradually become the main threats that seriously affect people's lives and health [3]. Myocardial infarction remains the leading cause of death and disability among individuals aged 50 and older [4]. Therefore, establishing an appropriate animal model of myocardial infarction is of great significance for studying the pathogenesis and treatment measures of MI. Various methods are available for establishing mouse models of MI, including pharmacological induction, minimally invasive techniques, and electrocoagulation. However, the most commonly employed method is ligation of the left anterior descending coronary artery. This technique is favored due to its straightforward procedure, precise vascular occlusion, and its close recapitulation of the pathological process of MI, thereby offering good potential for clinical translation. In this study, the left anterior descending coronary artery ligation method was used to establish a mouse model of MI. Heart rate, blood pressure, and electrocardiogram were measured, and TTC staining was performed to validate the model, aiming to provide a methodological foundation for future research on MI modeling.

2. Method

2.1. Animal groups

After 7 days of adaptive feeding, SPF-grade male C57 mice (6-7 weeks old) were fasted for 12 hours with free access to water. The mice were then randomly divided into the sham group and the model group using a random number table, with 10 mice in each group.

2.2. Model establishment

All surgical instruments were sterilized prior to the experimental procedures. For the sham-operated group, mice were placed in an induction chamber with 5% isoflurane for anesthesia induction. Upon reaching deep anesthesia, each mouse was positioned supine on a sterile surgical platform equipped with a respiratory mask, and anesthesia was maintained with 1.5% isoflurane. The left thoracic area was shaved and disinfected, and a sterile drape with an opening was applied. An ophthalmic scissor was used to incise the skin between the third and fourth intercostal spaces (**Figure 1A**), and a pre-placed suture was prepared. The muscle layer was bluntly dissected using forceps. With gentle compression applied to the thoracic cavity using the left hand and curved forceps held in the right hand, the intercostal space with the most pronounced cardiac pulsation was identified, and the ribs were rapidly spread to exteriorize the heart. The heart was immediately returned to the thoracic cavity, and air was evacuated from the chest. The skin incision was sutured and disinfected again. Mice were then placed on a heating pad until spontaneous breathing resumed and they recovered from anesthesia.

For the MI model group, anesthesia, skin preparation, disinfection, and heart exteriorization were performed as described above. The heart was gently lifted to visualize the left anterior descending coronary artery, located between the left atrial appendage and the pulmonary artery (**Figure 1B**). Ligation was performed near the apex of the heart. The ribs were then spread with curved forceps to facilitate returning the heart to the thoracic cavity, followed by air evacuation and subsequent procedures as described above (**Figure 1C**), thereby completing model establishment.

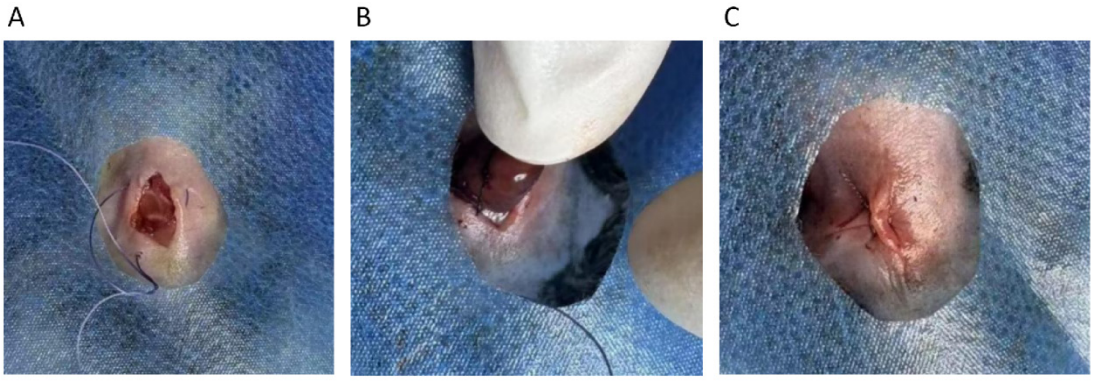

Figure 1. The operation method for the mouse model of MI caused by ligation of the left anterior descending branch of the coronary artery. (A) Expose the third and fourth intercostal spaces. (B) Cut out the heart and ligate the left anterior descending artery. (C) Closure of the cardiac exhaust incision.

2.3. Survival rate analysis

After the modeling process is completed, record the survival rates of the mice in each group:

(Number of surviving mice / Initial number of mice)× 100%

2.4. Heart rate (HR) and blood pressure monitoring analysis

First, power on the non-invasive blood pressure monitor for small animals, connect the sensor and heating device to adjust the temperature, and check whether the instrument is operating normally. Open the KdBpTerm software to verify the connection status and input basic animal information, then set the pressure and sensor signals. Subsequently, place the mouse into a restrainer for thermal insulation, insert its tail into the sensor, wait for the mouse to stabilize, and begin measuring heart rate and blood pressure.

2.5. Measurement and analysis of electrocardiogram

After connecting the equipment, open the Biological Information Medical Signal Acquisition and Processing System. Anesthetize the mouse with isoflurane inhalation and position it supine on a temperature-controlled operating board. Disinfect the limbs with alcohol and insert three-lead needle electrodes subcutaneously into the four limbs: right forelimb electrode (red, negative), right hindlimb electrode (black, ground), and left hindlimb electrode (green, positive). Activate the electrocardiogram measurement function and click Start to display the sampled waveform, thereby completing ECG acquisition.

2.6. 2,3,5-triphenyltetrazolium chloride (TTC) staining analysis

After removal, the hearts were immediately placed in pre-chilled physiological saline. Forceps were used to press the heart until all blood was expelled gently. The hearts were then frozen at -20°C for 30 minutes, subsequently sliced into 5 sections, and immersed in TTC solution. The TTC solution was protected from light and incubated in a 37°C water bath, with the heart sections turned over every 5 minutes. After staining for 15 minutes, the sections were transferred to fixative solution for fixation.

2.7. Statistical analysis

Data statistics and analysis were conducted using GraphPad Prism 8.0 software. The data were expressed

as mean ± standard deviation (SD). The one-way analysis of variance (ANOVA) was used for comparison between the two groups. A $p < 0.05$ indicated a significant difference between the two groups.

3. Result

3.1. Survival rate analysis

After MI modeling, the 24-hour survival rate of mice was 70%. The surviving model mice exhibited symptoms such as listlessness, increased respiratory rate, arched back and huddling posture, decreased food and water intake, ruffled fur, decreased body temperature, and abnormal heart rhythm. In contrast, no abnormalities were observed in the sham group.

3.2. Heart rate and blood pressure monitoring analysis

The results showed that there was no significant difference in heart rate between the model group and the sham group, while the diastolic and systolic blood pressures were higher in the model group than in the sham group (**Figure 2**).

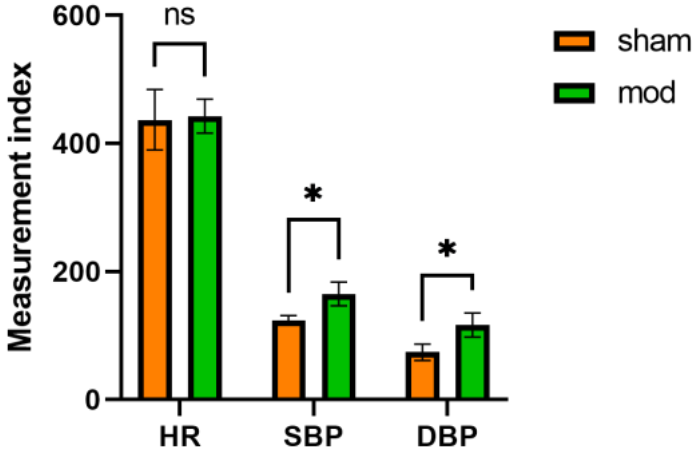

Figure 2. Comparison and analysis of the measurement results of HR, SBP and DBP in the sham group and the model group. n = 10. ns indicates that there is no significant difference between the two comparison groups; *$p < 0.05$.

3.3. Electrocardiogram analysis

Compared with the sham group, the model group exhibited ST segment elevation and T wave inversion on the electrocardiogram (**Figure 3**).

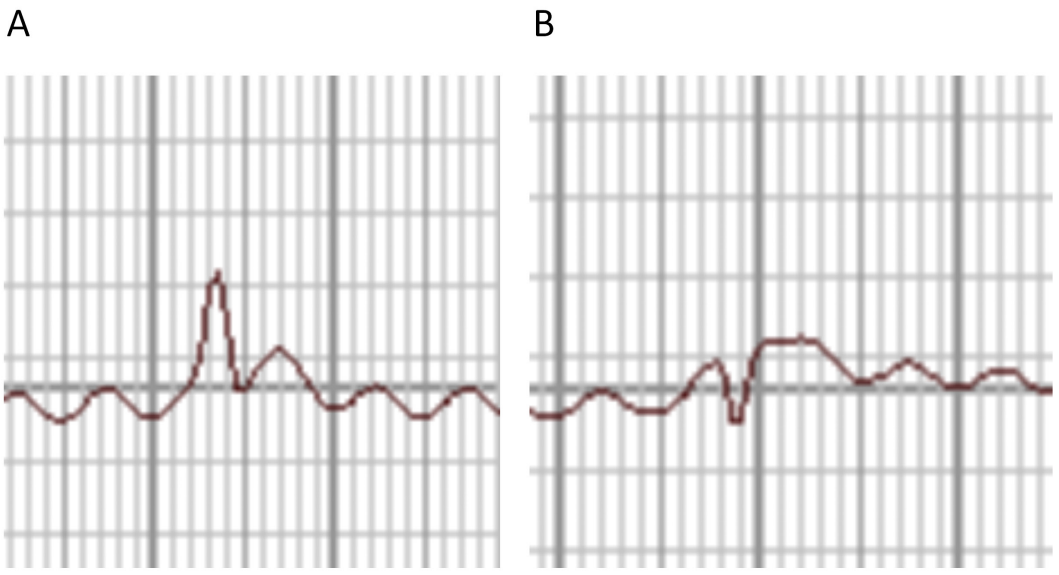

Figure 3. Electrocardiogram test results. (A) sham group, (B) model group.

3.4. TTC staining analysis

TTC staining is regarded as the gold standard method for assessing myocardial infarct size. In this technique, normal myocardium stains brick red, while infarcted myocardium appears pale. TTC staining results of heart sections from the two groups showed no infarcted areas in the sham group, whereas distinct pale infarcted regions were clearly observed in the model group (**Figure 4**).

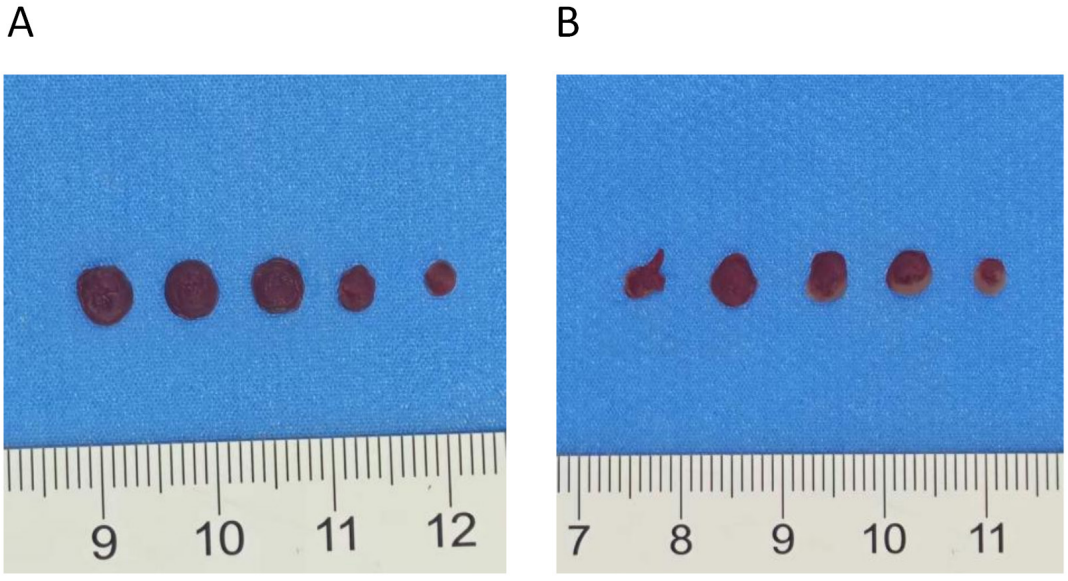

Figure 4. TTC staining result. (A) sham group, (B) model group.

4. Discussion and conclusion

In recent years, the incidence of MI has been increasing annually in China, making it a research hotspot for many scientists. Consequently, the establishment of MI models has become particularly crucial in scientific research, as it plays a significant role in elucidating and exploring disease mechanisms. In this study, an MI model was established by ligating the left anterior descending coronary artery in mice. The results showed a survival rate of 70% in the model group. Blood pressure and heart rate measurements revealed higher diastolic and systolic blood pressures in the model group, which represents an acute compensatory response. Electrocardiogram findings demonstrated ST segment elevation and T wave inversion in the model group, indicative of post-ischemic changes. TTC staining revealed distinct pale infarcted areas in the hearts of the model group, indicating myocardial necrosis, loss of mitochondrial function, and inactivation of dehydrogenases, which prevented reaction with the TTC dye. These findings collectively demonstrate that this modeling method successfully induces myocardial ischemia and necrosis in mice and can be reliably used for studying MI in mouse models.

The left anterior descending coronary artery ligation method is the most widely used experimental approach for establishing mouse models of myocardial infarction. This technique closely recapitulates the pathophysiological process of acute myocardial infarction in humans, avoiding the potential metabolic variability associated with pharmacological induction methods while more accurately reflecting the clinical reality of coronary occlusion, and preserving the option for either ischemia-reperfusion or permanent occlusion studies [5–7]. At the cellular mechanism level, following LAD ligation, local myocardial tissue experiences a sharp decline in oxygen supply, leading to impaired mitochondrial oxidative phosphorylation and ATP synthesis failure, which subsequently triggers a cascade of events including reactive oxygen species burst and opening of the mitochondrial permeability transition pore [8,9]. These processes ultimately result in irreversible myocardial cell damage, with the pale areas revealed by TTC staining corresponding precisely to cellular changes such as dehydrogenase inactivation and mitochondrial dysfunction. At the functional systemic level, the elevated blood pressure observed in the model group indicates activation of the sympathetic-adrenal system and the renin-angiotensin-aldosterone system [10,11]; the ST segment elevation and T wave inversion on electrocardiograms reflect electrophysiological remodeling following transmural ischemia [12,13]. Both aspects collectively validate the successful establishment of the model in this study.

This study has certain limitations. First, the experimental evaluation was conducted only at the acute phase (24 hours post-surgery), without tracking the progression of cardiac function or long-term survival during the chronic phase following myocardial infarction. Second, although the infarct area was validated by TTC staining, histopathological and molecular analyses were not performed to investigate mechanisms such as the inflammatory response.

Nevertheless, the mouse model of myocardial infarction established by left anterior descending coronary artery ligation in this study demonstrates promising potential for future applications. This model can serve as a reliable tool for drug screening and efficacy evaluation, enabling the assessment of therapeutic effects on cardiac function protection and infarct size reduction following treatment [14-16]. Moreover, when combined with techniques such as gene knockout, this model can be employed for functional validation of specific molecules involved in the pathological progression of myocardial infarction [17-18]. Building on this foundation, integrating dynamic monitoring using echocardiography with molecular biology approaches will facilitate systematic investigation of the mechanisms underlying post-infarction myocardial injury, as well as

the temporal dynamics of key events including inflammatory signaling pathways, oxidative stress, pyroptosis, and autophagy, thereby providing a theoretical basis for the identification of novel therapeutic targets [19–21].

Ethical approval

Experimental Animal Ethics Committee of Tianjin Xinrui Biotechnology Co., Ltd. (Project No.: 2026001)

Disclosure statement

The authors declare no conflict of interest.

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