Research progress on exercise-induced cardiac injury

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Abstract: Integrating recent domestic and international research on exercise-induced cardiac injury and combining it with the author's related work, this paper mainly discusses the definition, clinical manifestations, pathogenesis, early warning, and prevention and treatment of exercise-induced cardiac injury, in the hope of providing reference and insights for the early warning and prevention and treatment of exercise-induced cardiac injury in military training personnel.

Keywords: Military Personnel, Exercise, Cardiac Injury, Early Warning, Prevention and Treatment

1. Introduction

Appropriate exercise loads can promote the remodeling of cardiac structure and function, leading to the formation of "athlete's heart," characterized by increased capillary density in the myocardium, thickening of myocardial fibers, enhanced myocardial contractility, and sufficient cardiac functional reserve. Overload exercise may lead to exercise-induced cardiac injury, which is associated with exercise-induced arrhythmias and sudden death during exercise. In recent years, research on exercise-induced cardiac injury has extended beyond athletes to other populations, such as the military, a special group that frequently undergoes high-intensity training. Training personnel in this group often exhibit abnormalities in cardiac function and arrhythmias, manifesting symptoms such as chest distress, palpitations, chest pain, dizziness, shortness of breath, fatigue, syncope, and abnormal auscultation findings of the heart [1], which severely affect training levels and the improvement of military combat effectiveness. Conducting research on early warning and prevention and treatment of exercise-induced cardiac injury is of positive significance for reducing non-combat-related attrition in the military, alleviating the burden on medical support, and enhancing the combat effectiveness of troops. This article summarizes the recent research progress on exercise-induced cardiac injury as follows.

2. Definition

Intense exercise-induced cardiac damage is quite common among people, especially in groups that regularly engage in high-intensity sports and training, such as athletes and military training personnel. Currently, there is no precise definition for cardiac damage caused by high-intensity exercise. Exercise-induced myocardial micro-injury, ischemic myocardial damage, exhaustive cardiac injury, and cardiac damage caused by high-intensity exercise in high and low-temperature environments all fall within the category of cardiac injuries caused by high-intensity exercise. Exercise-induced myocardial micro-injury refers to the structural and functional metabolic changes in myocardial cells and subcellular components that occur during intense exercise, where the body's metabolic rate accelerates, blood levels of catecholamines increase, the myocardial oxygen demand increases, leading to myocardial ischemia and hypoxia, and some decompensatory changes occur [2]. Ischemic myocardial damage is similar to exercise-induced myocardial micro-injury, and it indicates that after high-intensity exercise, the myocardium may have ischemic damage without typical enzymatic changes, but there will be abnormalities in cardiac troponin T (cTnT). Miniature myocardial injury is a type of ischemic myocardial damage. Domestic scholars, such as Liu Ling and others [3], have confirmed that there is no significant linear relationship between the serum concentration changes of cTnT and creatine kinase (CK), and cTnT, due to

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its sensitive reaction and strong specificity, can provide a basis for diagnosing cardiac damage. Miniature myocardial injury and exercise-induced myocardial micro-injury have similarities to some extent, with relatively small exercise loads and lighter myocardial damage. Exhaustive cardiac injury refers to the cardiac damage caused by exhaustive exercise, which is defined as continuing to exercise on the basis of fatigue until the body is completely unable to move. Exhaustive exercise, due to its large load, can cause a series of changes to the heart, including myocardial morphology and structure, cardiac electrophysiology, cardiac biochemistry, and cardiac function. In addition, high-intensity exercise in special environments is more likely to cause cardiac damage, such as in high and low-temperature environments, where the probability of cardiac damage is significantly increased. This is because special environments can produce strong stress responses in the body and mind, and stress can have many adverse effects on the cardiovascular system, thereby exacerbating exercise-induced cardiac damage. Based on the comprehensive analysis of the different types of cardiac damage, we believe that the content of exercise-induced cardiac damage is more extensive, and it is more accurate and reasonable to use the definition of exercise-induced cardiac damage to describe the adverse effects of high-intensity exercise on the heart.

3. Clinical manifestations

Our previous studies have suggested that the incidence of exercise-induced cardiac injury due to high-intensity military training is relatively high, with common symptoms including chest distress, palpitations, chest pain, dizziness, shortness of breath, and fatigue; physical signs include abnormal heart auscultation, such as muffled heart sounds, bradycardia, tachycardia, premature beats, etc. Other studies have reported that exercise-induced cardiac injury can manifest as changes in cardiac morphology and structure, abnormalities in myocardial injury markers, exercise-induced arrhythmias, reduced cardiac function, syncope, and even sudden death during exercise [1].

3.1. Cardiac structure and functional changes

Most research results suggest that the changes in cardiac structure and function caused by exercise are physiological. However, the clinical manifestations and electrocardiogram characteristics of some athletes cannot be explained solely by physiological or functional changes, which are related to the exercise load. Appropriate exercise can lead to the development of an "athlete's heart." The "athlete's heart" has the following characteristics: the growth of athletic myocardial fibers is adapted to the corresponding growth of capillaries, the remodeling of athletic myocardial cells and subcellular structures is adapted to their oxidative metabolic function, and the remodeling of myofilament isoforms is adapted to the contraction process. However, excessive exercise, such as exhaustive exercise, can have many adverse effects on cardiac structure and morphological function. Thomas et al. [4-5] used histological methods to observe the ultrastructure of myofibers and sarcotubular membranes in the myocardium of rats at rest, after a single bout of exhaustive swimming exercise, and after 7 days of repeated exhaustive swimming exercise, studying the impact of repeated exhaustive exercise on the ultrastructure of myocardial subcellular membranes. Observations of mitochondrial morphology revealed that neither single nor repeated bouts of exhaustive exercise led to myocardial hypoxia, but they did cause sarcotubular swelling and an increase in spacing. Electron microscopy showed complete collapse of the sarcotubules, and it was found that 7 days of repeated exhaustive exercise significantly increased the extent of myocardial subcellular membrane damage.

3.2. Myocardial injury biomarkers

High-intensity exercise can lead to elevated levels of creatine kinase isoenzyme (CK-MB) and cardiac troponin T (cTnT), which are biomarkers indicative of myocardial injury. The enzymatic assays can be significantly interfered with by damaged skeletal muscle, resulting in poor specificity, thus the use of CK-MB levels as a diagnostic basis for myocardial damage is questioned. Compared with CK-MB, cTnT has stronger specificity and higher sensitivity, and has been established as a new clinical indicator for the diagnosis of myocardial injury. Unlike the regressive diagnosis of CK-MB (>6 hours), cTnT can rapidly detect myocardial injury and real-time reflect the entire process of myocardial damage, making it a truly specific biomarker for myocardial injury, widely used in the diagnosis, monitoring, and prognostic evaluation of myocardial injury [6-7]. Some studies have reported that among 8 cases of myocardial injury

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occurring after exhaustive training such as 5000m armed cross-country and 400m obstacle courses, there were 2 cases of sinus tachycardia, 1 case of sinus arrhythmia, 2 cases of ST-T changes in precordial leads, and 6 cases of elevated myocardial enzymes [8]. Our investigative research also found multiple cases of cardiac sudden death, severe arrhythmias, and abnormally elevated myocardial enzymes after high-intensity exercise, some of which were even misdiagnosed as viral myocarditis, primary cardiomyopathy, etc.

3.3. Exercise induced arrhythmias

Exercise-induced arrhythmias have always been a concern in the field of sports medicine, and their occurrence is associated with pathological cardiac damage caused by repeated high-intensity exercise, which often affects the health of athletes [9-10]. The epidemiological survey results of exercise-induced arrhythmias show that elite athletes have a high incidence of mild arrhythmias, especially sinus arrhythmia and sinus bradycardia. The incidence of severe arrhythmias in young athletes is relatively low, with atrial fibrillation, myocardial ischemia, and frequent ventricular premature beats rarely occurring, and bundle branch block and atrioventricular block are more commonly seen. Athletes who have engaged in high-intensity training and endurance athletes have a higher incidence of atrial fibrillation than athletes in other disciplines [11-12].

3.4. Exercise related syncope and sudden death

Exercise-related syncope and sudden death during exercise have become hot topics of discussion in recent years. Exercise-related syncope refers to the temporary loss of consciousness during or after exercise, without trauma, due to significant changes in blood chemicals or transient cerebral blood supply insufficiency. Patients fall due to the disappearance of muscle tone but can recover quickly [13-14]. The basic cause of syncope is temporary cerebral ischemia and hypoxia, which can mainly be divided into types such as cardiogenic syncope, vasovagal syncope, post-exercise hypotension, orthostatic hypotension, carotid sinus sensitivity syndrome, and micturition syncope, among which cardiogenic syncope is an absolute risk factor for sudden death during exercise [15]. Exercise-related sudden death refers to the sudden and unexpected death that occurs in individuals engaging in physical exercise or athletes with or without symptoms within 24 hours after exercise [16]. Studies have found a close relationship between exercise-related syncope and sudden death during exercise, with frequent cardiogenic syncope being a warning signal for sudden death during exercise [14]. Scholars at home and abroad have found through surveys and research on exercise-related sudden death that factors including brain origin, heart origin, and the athlete's own physique can all cause sudden death during exercise. Sudden cardiac death accounts for most of the sudden deaths during exercise, and diseases causing sudden cardiac death mainly include hypertrophic cardiomyopathy, dilated cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, catecholaminergic polymorphic ventricular tachycardia, long QT syndrome, coronary artery anomalies, commotio cordis, aortic stenosis, etc.

3.5. Classification of exercise induced cardiac injury

Based on clinical manifestations and examination results, exercise-induced cardiac injury is preliminarily classified into common type, arrhythmic type, heart failure type, and sudden death type [1]. The common type refers to those with cardiac-related discomfort, accompanied by changes in cardiac enzymes and echocardiography, with no abnormalities seen on electrocardiogram; the arrhythmic type refers to those with cardiac-related discomfort, with electrocardiogram indicating arrhythmias, which may or may not be accompanied by changes in cardiac enzymes, echocardiography, etc.; the heart failure type is manifested by pulmonary edema or cardiogenic shock, with ultrasound examination suggesting cardiac enlargement and reduced left ventricular ejection fraction; the sudden death type refers to the sudden cessation of heartbeat and breathing occurring immediately or within 1 hour after the onset of acute symptoms. The classification of exercise-induced cardiac injury is of great significance for daily clinical work, allowing for more rational and standardized clinical management. Different clinical types can be treated with corresponding, more targeted measures, thereby providing more scientific and effective treatment for military personnel suffering from exercise-induced cardiac injury.

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4. Pathogenesis

The pathogenesis of exercise-induced cardiac injury is not fully understood at present and may involve changes in hormone secretion, imbalance of nitric oxide (NO) and endothelin, increased free radical content and calcium overload, changes in hemodynamics, alterations and regulation of myocardial energy metabolism, occurrence and regulation of myocardial cell apoptosis, and changes and regulation of myocardial inflammatory responses.

4.1. Changes in hormone secretion

After high-intensity exercise, the main changes are reflected in two aspects: ① Imbalance of NO and endothelin. NO is a novel cellular second messenger with functions such as vasodilation and regulation of vascular tone. Endothelin is a peptide with a strong and persistent vasoconstrictive effect, which is negatively regulated by NO. Research by Wang Fu wen et al. [17] has shown that high-intensity exercise can cause an imbalance of NO and endothelin in the body, inducing myocardial ischemia and leading to myocardial injury. ② Increase in cortisol levels and decrease in testosterone levels. A certain concentration of testosterone has an important impact on the physiology and pathology of the heart and is very necessary for the normal functioning of human cardiac function. It can improve myocardial blood supply and reduce myocardial cell fibrosis and apoptosis. The decrease in testosterone levels after high-intensity exercise will increase the probability of cardiac injury to a certain extent.

4.2. Imbalance of free radicals and calcium overload

The body contains a free radical clearance system, and the production and clearance of free radicals are in a state of dynamic equilibrium. Once the balance is disrupted, it can cause damage to the body. Studies have shown that under high-intensity exercise loads, the content of free radicals in myocardial cells increases, and mitochondrial calcium overload occurs, leading to myocardial ischemia and hypoxia, and causing myocardial injury. The increase in free radical content is related to both excessive production of free radicals and changes in the activity of enzymes that clear free radicals [17].

4.3. Changes in hemorheology

The rheological properties of blood and their changes under various physiological and pathological conditions are one of the determinants of cardiac dynamic function and tissue oxygenation. The assessment of hemorheology can serve as a sensitive marker for exercise training and an important risk factor highly sensitive to exercise intensity [18]. Xu Feng et al. [19] reported that high-intensity naval training can lead to an increased incidence of abnormal red blood cell deformability and abnormal electrocardiographic activity. The experimental results of Wang Fu wen et al. [17] showed that immediately after rats underwent high-intensity swimming exercise, red blood cell aggregation increased significantly, and blood viscosity increased markedly. This indicates that high-intensity exercise can increase the probability of thrombosis by increasing the red blood cell aggregation index and blood viscosity, trigger microcirculatory disturbances, reduce oxygen supply and blood volume to the heart, leading to the occurrence of myocardial injury.

4.4. Regulation of myocardial energy metabolism

Myocardial energy metabolism is primarily aerobic, with the functional sites of oxidative metabolism and energy production located in the mitochondria of myocardial cells. The number and functional structure of mitochondria in myocardial cells are crucial for the heart's energy metabolism and function. Myocardial cell hypoxia caused by high-intensity exercise is a key factor leading to exercise-induced cardiac injury. Under hypoxic conditions, mitochondrial energy metabolism is inhibited, which in turn affects the expression of proteins within the mitochondria, causing myocardial cell dysfunction. In recent years, it has been found that nuclear respiratory factor-1 (NRF-1) and mitochondrial transcription factor A (mt TFA) regulate mitochondrial energy metabolism by controlling the expression of protein subunits in the mitochondrial respiratory chain [20]. Studies have shown that the occurrence of exercise-induced cardiac injury is closely related to the reduced expression of NRF-1; both single and repeated high-intensity

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exercises can lead to a significant decrease in mt TFA expression, directly affecting the oxidative capacity of myocardial fiber mitochondria, and the energy produced is also reduced.

4.5. Regulation of myocardial cell apoptosis

The body, under the stimulation of endogenous or exogenous apoptotic signals, initiates the program of apoptosis through the regulation of a series of specific genes. Studies have confirmed that after high-intensity exercise, myocardial pro-apoptotic genes Bok, Bnip3, Casp2, and transcription factor Nfkbia, as well as apoptotic regulatory genes ATF3 and Sphk1, may be involved in the regulation of apoptosis during the occurrence of exercise-induced cardiac injury; during exercise, certain factors induce apoptosis, and the signals that induce apoptosis include glucocorticoids, downregulated growth factors, reactive oxygen species (ROS), high intracellular Ca^{2+}, and tumor necrosis factor (TNF). During high-intensity exercise, the concentrations of glucocorticoids, intracellular Ca^{2+}, and ROS all increase, which can induce apoptosis through the action of both extracellular and intracellular proteins.

4.6. Changes in myocardial inflammatory responses

The enhancement of myocardial inflammatory responses plays a significant role in the occurrence of exercise-induced cardiac injury. Studies have confirmed that after a single high-intensity exercise, there is a significant upregulation in the expression of a large number of chemokines and their receptors, mediating inflammatory cell responses. Early growth response genes, intercellular adhesion molecules, and nuclear factor kappa B (NF-κ B) may all be involved in the process of enhanced inflammatory responses during the occurrence of exercise-induced cardiac injury.

5. Early warning and prevention of exercise induced cardiac injury

Effectively conducting preventive measures for exercise-induced cardiac injury, providing psychological counseling, non-pharmacological and pharmacological interventions for soldiers with high-risk factors before training, is of positive significance for reducing the burden on medical support and enhancing the combat effectiveness of troops. If soldiers experience discomfort such as chest distress, palpitations, or dizziness during or after high-intensity training, and if the electrocardiogram, myocardial enzyme indicators, and cardiac ultrasound-related indicators meet the preliminary early warning criteria established by our research group, training should be immediately terminated and timely treatment should be administered.

5.1. Early warning

The early detection, identification, and early warning of exercise-induced cardiac injury are crucial, as they directly determine whether training personnel can receive timely treatment in the event of such an injury. Seeking safe, effective, and easily disseminated methods for the early warning of exercise-induced cardiac injury is of great significance. There has been little research on early warning methods for exercise-induced myocardial injury and sudden death, so we explored the application of dynamic electrocardiography, cardiac ultrasound, and myocardial injury biomarker indicators in the early warning of exercise-induced cardiac injury.

Dynamic electrocardiography technology has been developing for more than 50 years and its application has become increasingly widespread. The analysis system has added new indicators for detecting the autonomic nervous system, such as deceleration capacity of rate (DC), heart rate variability (HRV), T wave alternans (TWA), and QT dispersion (QTd). By observing the 24-hour dynamic electrocardiogram changes before and after high-intensity load training in newly recruited soldiers, we explored the possible effects of intense exercise on cardiac electrophysiology, providing a basis for the early warning of cardiac injury caused by excessive load exercise and exercise-induced sudden death. Research suggests that after high-intensity military training, the sympathetic nervous activity in the body is significantly enhanced, and the vagal nerve activity is reduced, leading to instability in the electrical activity of myocardial cells, increasing myocardial vulnerability, and inducing various arrhythmias. Comprehensive analysis of multiple indicators of dynamic electrocardiography (mainly including HRV, TWA, QTd, heart rate turbulence, and

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DC) can improve detection sensitivity and accuracy, and can provide a reference basis for the early warning of cardiac injury and exercise-induced sudden death caused by excessive training in the military [21].

The diagnostic criteria for exercise-induced cardiac injury are also difficult to determine, and one of the important reasons is the lack of reliable biomarkers for myocardial injury. In China, the detection of exercise-induced myocardial damage still relies on serum CK-MB, but its specificity is poor because it is strongly interfered with by damaged skeletal muscles in enzymatic assays, and therefore the use of CK-MB levels as a diagnostic basis for myocardial damage is questioned. Some scholars have proposed using the CK-MB/CK ratio to increase the specificity of myocardial injury diagnosis [22]. The activity of CK-MB in normal human serum accounts for about 5% of CK activity; if the CK-MB in serum is significantly increased, it indicates significant myocardial involvement. Other studies have found that when there is ischemic injury to the myocardium, there may not be typical enzymatic changes, but there will be abnormalities in cTnT, and the concept of ischemic myocardial injury has been proposed based on this. Minimal myocardial injury is one of them. Therefore, finding a biomarker that can reflect early myocardial injury and has myocardial specificity has become the key to the early diagnosis of exercise-induced myocardial injury. We studied the changes in myocardial markers before and after high-intensity training in newly enlisted soldiers, providing an early warning method for exercise-induced cardiac injury caused by military training in troops. The results show that the increase in CK-MB/CK and cardiac troponin I (cTnI) reflects early myocardial injury and has good specificity for diagnosis. After high-intensity training, the myocardial injury biomarker indicators in newly enlisted soldiers can increase, and when cTnI > 0.0025 ng/ml, CK-MB/CK > 6.5%, they can play an early warning role for myocardial injury of varying degrees and should be given high attention [23].

Exercise-induced cardiac injury among newly enlisted soldiers in the military is not uncommon. Detecting and predicting changes in cardiac structure and function under exercise load in new recruits is of significant importance for studying the occurrence and progression of related diseases. Systematic dynamic testing of cardiac structure and function is difficult to control, and there has been little previous research on this topic. By applying echocardiography to measure cardiac structure and functional indicators before and after military training in newly enlisted soldiers, we explored the changes in cardiac structure and function before and after high-intensity military training in new recruits. This provides a basis for the prevention and treatment of cardiac injury caused by excessive load exercise, thereby better guiding the training of new recruits. The study results indicate that high-intensity training leads to an increase in compensatory ejection fraction and enlargement of the left atrium in new recruits. The reduction in the ratio of early diastolic peak mitral flow velocity to late diastolic peak mitral flow velocity and the ratio of early diastolic mitral annular motion velocity to late diastolic mitral annular motion velocity of the left ventricular lateral wall suggest a decrease in diastolic function and mild impairment of cardiac function [24]. With the extension of exercise time, the cardiac structure and function have undergone adaptive changes in response to the exercise load.

5.2. Prevention and treatment

Among the many methods for the prevention and treatment of exercise-induced cardiac injury, scientifically organizing training (including pre-conditioning exercise training, intermittent low-pressure hypoxic training, etc.) and timely psychological counseling and education are the most basic and effective approaches. They are also relatively easy to implement at the grassroots military level. In addition to these, pharmacological prevention can also play a significant role in the prevention and treatment of exercise-induced cardiac injury.

5.2.1. Exercise pre-conditioning

Exercise pre-conditioning is a special form of ischemic pre-conditioning. It involves a short period of high-intensity exercise, either continuous or intermittent, that causes relative or absolute myocardial ischemia and hypoxia. This process increases the expression of protective effector molecules, enhances the myocardial tissue's tolerance to subsequent prolonged ischemia, mitigates ischemia-reperfusion injury, and induces myocardial protection [25]. Exercise pre-conditioning is a scientific training method that is easy to implement in grassroots military units and should be further promoted.

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The protective effects of exercise pre-conditioning on the heart are increasingly becoming a focus of interest in the field of sports medicine. Exercise pre-conditioning has two phases of protective effects: the early phase and the delayed phase. Its protective role for the heart is mainly reflected in enhancing the stress resistance of myocardial tissue, improving vascular activity, reducing the size of infarction, and decreasing the incidence of arrhythmias and myocardial stunning. The mechanisms by which it induces myocardial protection may involve the signaling pathways of trigger substances - intermediate substances - effector substances. Trigger substances include adenosine, adrenergic agonists, opioids, NO, ROS, etc.; intermediate substances include protein kinase C, etc.; and effector substances include ATP-sensitive potassium channels, heat shock proteins, glutathione, superoxide dismutase, etc. [26].

5.2.2. Intermittent hypobaric hypoxia training

Oxygen is an essential foundation for the survival of humans and many organisms, and it is the most critical factor for life activities. Hypoxia can have both damaging and protective effects on the body. The cardioprotective effects of chronic intermittent hypobaric hypoxia may be achieved by increasing myocardial capillary density and coronary blood flow, enhancing myocardial antioxidant capacity, increasing the expression of heat shock proteins, reducing apoptosis of cardiac cells during ischemia-reperfusion, and weakening the activity of β-adrenergic receptors on myocardial cell membranes [27].

The mechanism of the cardioprotective effects of chronic intermittent hypobaric hypoxia is not yet fully understood. Enhancing the heart's tolerance to ischemia and hypoxia to achieve cardioprotection has always been a hot topic in basic and clinical medical research. Intermittent hypoxia treatment has significant cardioprotective and antiarrhythmic effects, and it also has the advantages of being simple, easy to apply, without obvious adverse reactions, and having a longer duration of action. Intermittent hypoxia training has the potential to be used as a non-pharmacological cardioprotective measure in clinical practice and gradually promoted as a scientific training method in rehabilitation, fitness, disease prevention, and enhancement of athletic performance [28]. It can be extended to the daily training of military training personnel.

5.2.3. Medications

Research on the pharmacological prevention and treatment of exercise-induced cardiac injury began relatively late, and its application in practice is even rarer. Salidroside (SAL), Xin fu kang oral liquid, allicin, and melatonin have been relatively more studied in the prevention and treatment of exercise-induced cardiac injury.

SAL is one of the main active components of Rhodiola rosea. Experiments have confirmed that SAL can improve the oxidative metabolism capacity of muscle cells and enhance their fatigue resistance by affecting the expression of proteins such as peroxisome proliferator-activated receptor-γ coactivator-1α (PGC-1α), peroxisome proliferator-activated receptor α (PPARα), and mitochondrial transcription factor A (mt TFA). SAL has effects such as anti-stress, improvement of ischemia-reperfusion injury of the heart, antioxidant, anti-ischemia, inhibition of calcium overload, and scavenging of oxygen free radicals [29-30]. Studies have shown that SAL can effectively inhibit the hemodynamic changes and cardiac function damage caused by myocardial ischemia or reperfusion in rats, possibly by promoting the phosphorylation levels of ERK and p38 in the rat myocardium through the mitogen-activated protein kinase (MAPKs) pathway, thus playing a protective role for myocardial cells [31-32]; SAL has a protective effect on exercise-induced cardiac injury, and its molecular mechanism may be to induce mitochondrial biogenesis by upregulating the expression of PGC-1α, NRF-1, and NRF-2 mRNA, as well as PGC-1α and NRF-2 proteins [33]; SAL can play a protective role on the heart by improving the respiratory function of myocardial mitochondria [34].

The role of Xin fu kang oral liquid in the prevention and treatment of exercise-induced cardiac injury has also received widespread attention. Studies have shown that Xin fu kang oral liquid can significantly improve myocardial energy metabolism in rats with chronic heart failure [35], has a significant protective effect on exercise-induced cardiac injury in rats, and can significantly improve the exercise capacity of rats. It may exert its cardioprotective effect by reducing cortisol and increasing testosterone; Xin fu kang oral liquid can also protect the heart by slowing the heart rate and improving heart rate variability (HRV), and its mechanism is to weaken cardiac vagal excitability [36].

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Sports medicine research has shown that allicin has effects such as scavenging free radicals, anti-tumor, vasodilation, anti-inflammatory, improving blood perfusion, and preventing atherosclerosis; melatonin is mainly a hormone secreted by the pineal gland, which has antioxidant effects, anti-stress effects, anti-platelet, adjusting blood lipids, anti-atherosclerosis, and cardiomyocyte protective effects.

6. Conclusion

Efforts should be made to enhance the awareness of exercise-induced cardiac injury among healthcare workers and training personnel, and to develop scientifically sound and reasonable training programs. The preliminary clinical classification of exercise-induced cardiac injury that we have established needs further standardization, and it is necessary to expand the scale to verify the early warning criteria formulated for exercise-induced cardiac injury. For some special cases of exercise-induced cardiac injury, such as sudden death during exercise, research is very difficult, most of which are retrospective studies, and there are very few cases of successful resuscitation, so it is not easy to clarify the causes of sudden death during exercise, which requires further research and the conduct of large-scale epidemiological surveys. In addition, the damage caused to the heart by repeated high-intensity exercise is different from that caused by a single high-intensity exercise, and the long-term prognosis of exercise-induced cardiac injury also needs further study. Most of the previous research on the prevention and treatment of exercise-induced cardiac injury has focused on animal experiments, and there should be an increase in clinical trial research in the future, such as the preventive and therapeutic effects of drugs like SAL on exercise-induced cardiac injury in military training personnel, which need further research. In terms of non-pharmacological protective measures, such as the prevention and treatment of exercise-induced cardiac injury by exercise pre-conditioning, a feasible quantitative standard should be further established, and what kind of exercise mode, intensity, and duration can induce the best pre-conditioning effect needs further exploration.

It is believed that with the gradual deepening of research on early warning and prevention of exercise-induced cardiac injury and the gradual popularization of related knowledge, both training personnel and healthcare workers will have a deeper understanding of it, thereby reducing the probability of its occurrence. Once it occurs, it can be identified and sent to the hospital as soon as possible, and healthcare workers can provide faster, standardized, and reasonable treatment.

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