Study on the Relationship between Cardiomyocyte Apoptosis and Left Ventricular Function in Spontaneously Hypertensive Rats

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At present, hypertension is a relatively common cardiovascular disease. It not only affects the normal operation of target organs such as the heart and kidneys, but also causes cardiovascular and cerebrovascular diseases, which can lead to death. The apoptosis of cardiomyocytes is widespread in the cardiovascular system and is closely related to vascular diseases. Therefore, the purpose of this article is to explore the relationship between changes in left ventricular function, myocardial multidimensional strain and interstitial fibrosis in spontaneously hypertensive rats (SHR) during cardiomyocyte apoptosis. Whether the research is consistent in terms of order, as the age of hypertensive rats increases, whether there is a close connection between myocardial cell apoptosis and the structure and function of the left heart. The method in this article is to use the method of experimental comparison to randomly group 60 experimental mice and observe the changes of various indicators of rats of different ages, from 12 weeks to 84 weeks. The observations include left ventricular mass index (LVMI), left ventricular ejection fraction (LVEF), left ventricular short axis shortening rate (FS), LVEDP, LV+dp/dtmax, LV-dp/dtmax. At the same time, using the TUNEL labeling method, the left ventricular myocardial tissue was sliced, and the apoptosis index of subendocardial and subepicardial myocardial cells was calculated. Then 3 groups were randomly selected from the experimental group, and Western blotting was used to quantitatively detect apoptosis-related the expression of the proteins Bcl-2, Bax, and Fas were compared between the groups. After analysis and determination, it can be found that the apoptosis index of cardiomyocytes is positively correlated with LVMI, CVF, and PVCA (r is 0.83, 0.89, 0.72, respectively, p=0.00). Corresponding conclusions are drawn from the comparison of data. As hypertensive rats grow older, the apoptotic index of cardiomyocytes will continue to increase, and when the myocardial hypertrophy is severe to heart failure, the apoptotic index of cardiomyocytes will increase significantly. This shows that the increase in cardiomyocyte apoptosis is closely related to left heart remodeling, the development of myocardial fibrosis and overall cardiac dysfunction.

Keywords: Spontaneous Hypertension, Cardiomyocytes, Left Ventricular Function, Preventive Effect
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Left ventricular dysfunction is a relatively common complication of hypertension. If the general hypertension patients are not treated in time, it will lead to left ventricular remodeling. The remodeling of left ventricle is mainly caused by excessive ventricular pressure and increased internal load. The neuroendocrine system will make the myocardium hypertrophy, which will affect the calcium circulation of myocardial cells, which will indirectly lead to heart failure and endanger human life and health. Therefore, it is very important to explore the relationship between hypertension and left ventricular function.

Cardiomyocyte apoptosis refers to the highly differentiated terminal cells of the human cardiomyocytes after growth, differentiation and maturation. Generally speaking, it is a normal physiological phenomenon [1]. In recent years,

biomolecular technology has made great breakthroughs. Cardiomyocyte apoptosis has received widespread attention. Cardiomyocytes also have their own anti-apoptotic ability. Many experts and scholars at home and abroad have been studying this. Without stopping, some scholars believe that normal cardiomyocytes in the human body rarely have apoptosis, and apoptosis will increase only when there are pathological changes and endocrine disorders [2-3]. Existing studies have also found that in patients with hypertensive diabetes, myocardial ischemia, pressure overload, and dilated cardiomyopathy, there is obvious apoptosis in cardiomyocytes [4]. Whether the occurrence of apoptosis will have a greater impact on the number of cells is still under investigation. Some domestic experts believe that when the body is suddenly stimulated by the outside world, there will be increased apoptosis, and when the stimulation is severely damaged, it may cause cell necrosis. Therefore, the direct factor that induces cardiomyocyte apoptosis and necrosis is the effect of hypertension. It is caused by myocardial biopsy in patients with heart failure and hypertension with left heart hypertrophy [5-6]. This shows that the proportion of cardiomyocyte apoptosis in patients with heart failure will be higher. When cardiomyocytes divide, the process of apoptosis is more obvious [7]. In addition, due to high blood pressure, myocardial fibrosis will affect the normal blood supply and delivery, making the left heart function unable to function normally, and the interstitium of myocytes and perivascular reactivity become fibrotic, which affects the whole body. Therefore, in the process of treating hypertension, the role and relationship between muscle cell apoptosis and the left ventricle can be solved in a fundamental way [8].

This article starts with the meaning and morphology of cardiomyocytes and spontaneous hypertension, and explores the relationship between the left ventricular function of hypertensive rats based on different apoptosis indexes. By establishing a hypertensive rat model, using different changes in protein expression, cardiac catheterization, and myocardial activity to conduct comparative studies, we can more accurately grasp the disease process, explore the effects of hypertension-induced cardiomyocytes, and propose more optimized treatments Program. This is conducive to the promotion of medical research problems, innovative methods to solve problems, and a good theoretical basis for the treatment of hypertension and heart disease and other diseases caused by it. By comparing the changes and analyzing the specific experimental results, we can find out the basic point of balance between the body's reaction and drug efficacy, and organically combine the two. Provide valuable technical experience for future animal experiments, draw out the similarities and differences in research directions through comparative advantage analysis, and learn advanced experience at the same time, and make suggestions for improvements in future medical technology advancement, in-depth understanding of animal disease, and strengthen animal medicine The specialization and accuracy of the aspects provide theoretical basis for the field of animal medicine.

THEORETICAL BASIS AND METHOD

Core Concepts

Cardiomyocytess

Cardiomyocytes are what we commonly call myocardial fibers. Like other muscles, they also have the ability to contract and expand. Cardiomyocytes are directly innervated by the central nervous system and have stripes. Some muscles are cylindrical and have extensive tissue branches [9-10]. Cardiomyocytes have a wide range of functions. Some of them are rich in myofibrils, which are mainly distributed in atrial and ventricular muscles, which protect the heart while ensuring work. These cells are also called working cells [11]. Generally speaking, cardiomyocytes will produce excitatory conduction under external stimulation, but there is no automatic rhythm. Compared with the corresponding special conduction tissue, the conductance is lower. Cardiomyocytes have only one nucleus, which is located in the center of the entire cell, and the ends of the fibers are connected to each other and interact to form a very effective muscle fiber network [12-13]. In addition, cardiomyocytes also include specially differentiated cardiomyocytes such as atrial muscle, ventricular working cell, sinoatrial

node, intraatrial bundle, and atrioventricular junction. When human body functions are violated and heart failure occurs, this is actually the deterioration of the contractile function of myocardial cells, which leads to a decrease in the number of cells and the inability to maintain normal life activities, which leads to the phenomenon of cell apoptosis. As cells continue to age and degenerate, the number of apoptosis is gradually increasing, and the loss of cardiomyocytes will become more and more serious. Finally, the occurrence of heart failure will be inevitable [14]. Cardiomyocyte apoptosis will affect the normal operation of the heart. The direct result is thickening of the ventricular wall, increased left ventricular mass, structural changes, and a series of pathological manifestations such as interstitial collagen deposition and fibrosis, and hypertrophy of cardiomyocytes. Among them, myocardial fibrosis is a relatively common pathological phenomenon, which is mainly affected by the abnormal metabolism of myocardial collagen. The collagen fibers in the myocardium will become more than usual [15].

Left ventricular hypertrophy

Cardiomyocytes are closely related to the left ventricle. If the cardiomyocytes produce greater pressure, it will lead to left ventricular hypertrophy and a series of pathological stimulus responses [16]. Left ventricular hypertrophy is also an important factor that directly affects left ventricular function. It is clinically manifested as an increase in the weight of the myocardium, the ventricular wall and septum are obviously thickened, the ventricle is also remodeled, and the myocardial cells become fibrosis and hypertrophy [17]. When left ventricular hypertrophy occurs, cardiomyocytes receive additional stimulus signals, which induce induction of transduction signals and gene transcription activation in the cells. This is reflected in the structure of lenient hypertrophy, and myocardial fibrosis will also cause compliance of the left ventricle. Decreased sex, hypertrophic cells will also limit the diastolic filling, which will affect the signal transmission. In severe cases, it will increase the dispersion and cause malignant arrhythmia [18]. In addition, left ventricular hypertrophy can be accompanied by high blood pressure, myocardial infarction and other cardiovascular diseases. It is also an inducing risk factor for arrhythmia, myocardial infarction and sudden death, and increases the mortality of cardiovascular diseases [19]. Left ventricular hypertrophy is also a mechanism of target organ damage in hypertension. Under this abnormal structure, it can also induce hypertension worsening and heart failure. Therefore, we must actively pay attention to the harm caused by left ventricular hypertrophy and understand the molecular mechanisms contained in left ventricular hypertrophy in order to maintain healthy heart operation and cardiovascular health. Hypertension is an important factor leading to left ventricular hypertrophy, and continuous pressure load affects the adaptability of cardiomyocytes [20]. Therefore, such diseases should be treated in time. Once they occur, they are closely related to the occurrence and development of myocardial ischemia, stroke and sudden death.

The Relationship between Cardiomyocytes and Ventricular Function

Cardiomyocyte apoptosis and ventricular remodeling

Under normal circumstances, apoptosis is the normal metabolic process of cells, and it is also a form of cell death. Because in order to maintain a healthy physiological state, it must meet the needs of internal environment stability and the construction of organs and tissues, so it is also the body's internal genes. Regulation to make the declining cells die autonomously [21]. In recent years, under the joint efforts of medical theoretical research and clinical practice, it has been found that apoptosis is widespread in the cardiovascular system, which is one of its pathological changes. In the body diseases caused by hypertension, we explore the vital organs During internal operation, a large number of cell apoptosis were found, and cell apoptosis is closely related to the severity of myocardial hypertrophy, so it can be inferred that cardiomyocyte apoptosis plays an irreplaceable role in the process of left ventricular remodeling [22]. In the process of myocardial thickening, a large number of cardiomyocyte apoptosis has been produced, but when the hypertrophic

characteristics are displayed, the number of apoptosis begins to decline. The two did not occur simultaneously, which shows that cardiomyocyte apoptosis is a dynamic process of change. It can also indicate that the reduction of cardiomyocyte apoptosis contributes to ventricular remodeling. Before the occurrence of left ventricular hypertrophy and after the onset of heart failure, cardiomyocyte apoptosis was significantly increased, and with significant left ventricular hypertrophy, cardiomyocyte apoptosis was significantly reduced [23]. Using this relationship, in the treatment of hypertensive heart remodeling, drugs that promote apoptosis can be used to reverse ventricular hypertrophy, which can promote the apoptosis of vascular smooth muscle cells and improve the effect of left ventricular remodeling.

Protein and ventricular remodeling

In the field of traditional medicine, it has always been believed that cardiomyocytes will not proliferate, and that myocardial hypertrophy is also caused by cardiomyocyte hypertrophy or other cells. With the development of science and technology and the advancement of medicine, in the process of heart development, there are two processes of apoptosis and proliferation of cardiomyocytes, which can maintain the normal work of heart tissue [24-25]. After stimulating the cardiomyocytes, it will be found that in the process of myocardial hypertrophy, both myocardial cell hypertrophy and myocardial cell proliferation. In the analysis of clinical cases, the myocardial hypertrophy caused by aortic valve stenosis produces cardiomyocyte hypertrophy. And proliferation. In the treatment of myocardial remodeling, it is very effective to use the negative regulation of protein to guide cell proliferation. Low-expressed protein can promote the proliferation of vascular smooth muscle cells, while high-level protein can effectively inhibit this proliferation effect, thereby improving vascular remodeling. Therefore, protein plays an important role in the treatment and regulation of cardiovascular disease and hypertension and left ventricular hypertrophy. Moreover, protein is also related to the occurrence and deterioration of tumors. With the help of protein expression levels, the induction of exacerbation genes can make left ventricular hypertrophy improve under pressure load. In the detection of protein expression in cardiomyocytes, it was found that the positive expression rate of cardiomyocyte protein in patients with left ventricular hypertrophy significantly lower, which undoubtedly shows that the low-expressed protein mediates the hypertrophy of cardiomyocytes on the one hand, and promotes the proliferation of cardiomyocytes on the other hand.

Hypertension and ventricular remodeling

Hypertension is not an independent cardiovascular disease, but will cause a series of basic diseases, of which the compression of the heart is extremely important. The increased mechanical tension caused by hypertension is one of the important causes of ventricular hypertrophy. Excessive blood pressure may increase the peripheral resistance of the blood vessels, leading to changes in the structure of the heart and blood vessels. In this way, in order to ensure normal cardiac output, the cardiovascular blood vessels will face special changes. The specific manifestation is thickening. Blood vessel walls, myocardial cells enlarge and interstitial collagen fibers proliferate. If it is allowed to develop without timely treatment, leading to cardiovascular remodeling, myocardial cells will gradually become necrotic, there is no way to stretch and contract, and they can no longer maintain their compensatory function, leading to heart failure. When the patient's blood pressure rises, the heart load increases, the pressure load in the heart cavity rises, and the local tension of the ventricular wall increases. After biofeedback, the protein level in the myocardial cells increases and the synthesis accelerates, and the accumulation of metabolites leads to myocardial hypertrophy. Myocardial tissue renin uses aldosterone to increase system activity and accelerate protein synthesis in myocardial cells, which just provides a material basis for myocardial hypertrophy. In addition, pressure load can also cause vascular endothelial damage and imbalance in the secretion of vasoactive substances, which can also promote ventricular remodeling. Therefore, in order to treat ventricular remodeling, it is necessary to lower blood pressure, improve vascular endothelial function, and reduce the level of receptors in the circulation.

HYPERTENSIVE MYOCARDIAL REMODELING EXPERIMENT

Experimental Materials

Through the application, I selected 60 experimental rats provided by the animal experiment center of the affiliated hospital of the medical university where the author is located. They are SHR male rats and WKY male rats. In order to create research variables, SHR are hypertensive rats, and WKY It is a normal blood pressure rat. All rats were randomly divided into 6 groups, 10 in each group, 3 groups each for SHR and WKY, to ensure that the weight of the experimental rats was 200-240g. In the early stage of the experiment, select a special breeding center, maintain a suitable and well-ventilated environment, ensure that the room temperature is about 22 degrees, the relative humidity is maintained at about 55%, and the light time is maintained at more than 10 hours. Keep adequate food and drinking water. Conventional commercial rat feed is provided exclusively by the breeding center, and the MCT required for the establishment of the entire model is selected from the US sigma company. When each group of rats is raised to a suitable age, the myocardial tissue of the rats should be selected for observation of related indicators. The main reagents include: rabbit anti-mouse Bax monoclonal antibody (Santa Cruz, USA, sc-526), ECL chemiluminescence liquid (Genview, USA), the main instruments include: GE Vivid E9 color Doppler ultrasound diagnostic apparatus, optical Microscope (German LEICA, DM3000 type).

Experimental Process

First, under the normal state of the experimental rats, the BP-2010Series rat tail pressure measurement method is used to accurately measure the blood pressure and heart rate of the rats, and take the average value to record. Subsequently, intraperitoneal injection was used for anesthesia, and disposable electrodes were attached to the exposed skin of the left forelimb and right hindlimb of the rat to facilitate the simultaneous recording of the electrocardiogram. Then use the ultrasonic diagnostic apparatus to scan and collect the contours of the left ventricle and outer membrane of the rat. After the electrocardiogram measurement, each group of rats was examined by cardiac catheterization, and the right common carotid artery was searched deep near the trachea. The left ventricular systolic pressure (LVSP), left ventricular end diastolic pressure (LVEDP), and mean arterial pressure were recorded. (MAP) and other data.

Subsequently, the preparation of myocardial specimens was started. After anesthesia, the heart was taken out after the rat's heart stopped at the end of diastole, washed with a lower temperature normal saline, and sliced and cryopreserved. After 3 days, it was taken out for dehydration-soaked wax treatment, made into wax blocks, and then stained with Masson and TUNEL. The whole rat heart tissue was fixed with 10% formaldehyde for 24 hours. The molten embedded paraffin was poured into a copper embedding frame, and then Place the wax-impregnated tissue block with warmed tweezers.

Finally, the TUNEL method is used to measure the apoptotic index of cardiomyocytes, and the number of apoptotic cells can be quickly detected by observing the changes and breakage of DNA during the process of apoptosis. With the help of an electron microscope, apoptotic cells can be counted. As shown in Table 1. The APOI of cardiomyocytes increased with age, and the SHR subendocardial APOI (APOI-endo) increased significantly at 66w and was higher than that of the WKY group at the same age ($p<0.05$).

Table 1.
Comparison of Apoptosis Index of SHR and WKY Cardiomyocytes

Week Age	Subendocardial Apoi (%)		Epicardial Apoi (%)	
	WKY	SHR	WKY	SHR
12W	1.34±0.40	1.34±0.60	1.23±0.48	1.43±0.53
28W	1.97±1.03	3.04±1.87	1.54±0.56	2.54±1.19
45W	3.42±0.87	4.70±1.56	2.61±0.79	3.67±2.04
66W	4.03±2.06	9.83±3.97	4.89±1.41	4.76±2.21
84W	4.43±2.07	17.90±3.75	5.25±1.69	8.98±2.32

Normal cells and apoptotic cells present different structures in appearance. The normal cell nucleus is blue, while the apoptotic myocardial cell nucleus is brown. When treating specimens, select the myocardium in two parts of the lateral wall subendocardium and subepicardium. Which can calculate the number of apoptotic nuclei and the

total number of nuclei. At the same time, the protein expression of related genes was also extracted and analyzed. The SPSS19.0 statistical software package was used for data statistical processing. Pearson was used for correlation analysis between variables, and $p<0.05$ was considered statistically significant.

Experimental Results

WKY rats are bred from Wistar rats and are a control strain of hypertensive rats. In this experiment, genetically stable congenital spontaneous SHR rats are used to better understand the characteristics of blood pressure changes in spontaneous hypertension. Through experiments, it can be seen that after 12 weeks, the diastolic and systolic blood pressures of SHR rats are relatively high. In comparison, the values of WKY group are lower. Moreover, as time increases, rats of SHR group obviously, it was affected by the abnormality of myocardial cells, with brown hair, lack of energy, etc., and after 84 weeks, the weight also decreased to varying degrees. It can be seen from the electrocardiogram surveyed that the SHR group is significantly higher than the WKY group after 45 weeks, and there is no significant difference between the same type of groups. As shown in Table 2, it is the comparison of the internal structure and function of the left ventricle including body weight at different stages of the experiment. Regarding the comparison of cardiac catheterization, SHR was higher than SKY after 12 weeks, and began to decline after 28 weeks. The LV+dp/dtmax and LV-dp/dtmax of SHR only increased significantly after 28 weeks. In comparison with myocardial collagen fibers, in the SHR group, myocardial CVF and PVCA had no special changes in the early stage of the experiment, but continued to increase as the experiment progressed. At the 66th week, it was already higher than the same week. Age WKY group ($p<0.05$). Hypertension also affects abnormal protein expression. There is no significant difference in Bax, Fas, Bax/Bcl-2 between the SHR group and the WKY group at 12 weeks, and the increase after 66 weeks is significantly higher than that of other groups of the same age ($p<0.05$).

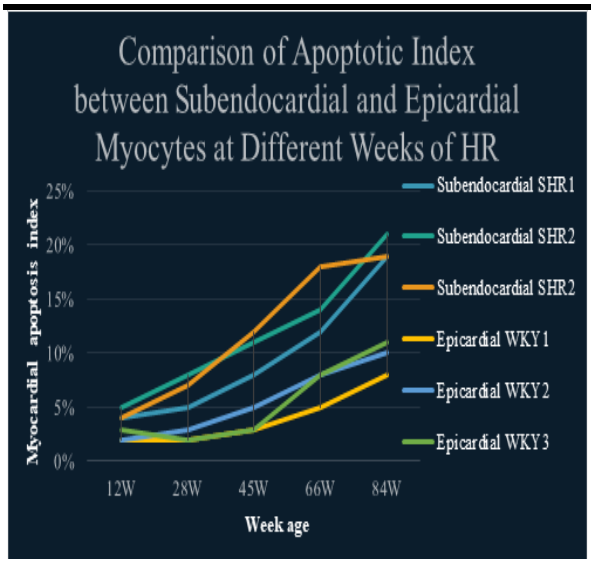
Table 2.
Comparison of SHR and WKY Left Ventricular Structure and Function ($\bar{x}\pm s$)

Week Age	Experimental Group	Weight	HR LAD(mm)	SBP (mm Hg)	DBP (mm Hg)
12W	WKY	0.276±0.018	361±14	112±7	69±8
28W	SHR	0.335±0.021	333±70	116±14	128±20
45W	WKY	0.389±0.005	333±26	116±11	70±10
66W	SHR	0.351±0.011	399±58	201±17	143±15
84W	**WKY**	**0.423±0.023**	**388±58**	**206±13**	**137±16**

DATA ANALYSIS

As shown in Figure 1, in the experiment, the two experimental groups analyzed the cardiomyocyte apoptosis index in their respective environments. In the three groups of SHR, with the increase of time, the percentage of apoptosis is also increasing. In the SHR1 group, the apoptosis index was 4% at 12 weeks and 5% at 28 weeks. At 45 weeks, the apoptosis index was 8%, at 66 weeks, the apoptosis index was 12%, and at 84 weeks, the apoptosis index was 19%. In the SHR2 group, the apoptosis index was 5% at 12 weeks, 8% at 28 weeks, 11% at 45 weeks, 14% at 66 weeks, and 84 weeks At this time, the apoptosis index was 21%. In the SHR3 group, the apoptosis index was 4% at 12 weeks, 7% at 28 weeks, 12% at 45 weeks, and 18% at 66 weeks, 84 weeks at this time, the

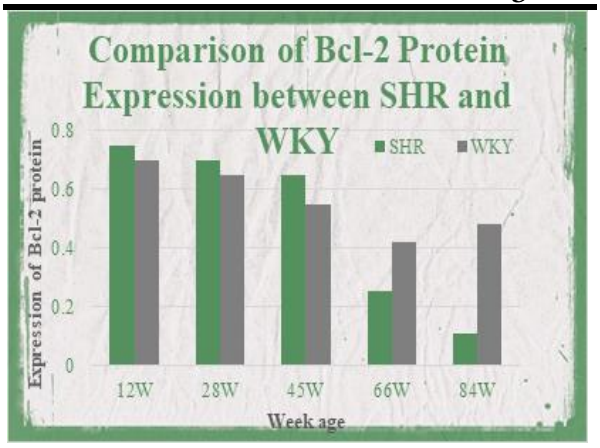
Figure 1.
Comparison of the apoptosis index of subendocardial and subepicardial myocardial cells in each week of SHR



apoptosis index was 19%. Hypertensive rats all began to have a significant increase after 66 weeks.

In the three groups of WKY, the cardiomyocyte apoptosis index is generally lower than that of the SHR group, which shows that hypertension has a significant impact on cardiomyocyte apoptosis. In the WKY1 group, the apoptosis index was 2% at 12 weeks, 2% at 28 weeks, 3% at 45 weeks, 5% at 66 weeks, and 84 weeks when, the apoptosis index was 8%. In the WKY2 group, the apoptosis index was 2% at 12 weeks, 3% at 28 weeks, 5% at 45 weeks, 8% at 66 weeks, and 84 weeks when, the apoptosis index is 10%. In the WKY3 group, the apoptotic index was 3% at 12 weeks, 2% at 28 weeks, 3% at 45 weeks, 8% at 66 weeks, and 84 weeks when, the apoptosis index was 11%.

Figure 2.
Comparison of Bcl-2 Protein Expression between SHR and WKY at Each Age



Protein expression has a greater impact on ventricular remodeling. As shown in Figure 2, the protein expression comparison of two types of rats is listed and compared. Throughout the experiment, the influence of time on protein expression is still very important. At the 12th week, the Bcl-2 protein expression of the two groups was relatively high, at 0.75 and 0.7, respectively. At 28 weeks, the protein expression of the SHR group was 0.7, the protein expression of the WKY group was 0.65, and at the 45th week, the SHR The protein expression of the SHR group was 0.65, and the protein expression of the WKY group was 0.55. At the 45th week, the protein expression of the SHR group decreased significantly to 0.25. The protein expression of the WKY group was 0.42. At the 45th week, the protein expression of the SHR group was 0.11, the protein expression of the WKY group is 0.48, and the difference between the two groups is getting bigger and bigger.

As shown in Figure 3, hypertensive rats can be relieved to some extent after drug treatment and protein regulation, and the pressure in the left ventricle will also be released. Through the comparison of blood pressure before and after treatment, the effect can be understood more clearly. In the SHR1 group, it was 15.21 kPa before treatment and 15.25 kPa after treatment. In SHR2 group, it was 23.65 kPa before treatment and 26.69 kPa after treatment. In SHR3 group, it was 23.59 kPa before treatment and 22.36 kPa after treatment. In general, in the process of hypertension treatment, due to individual differences and different drugs, the results will be different. In the WKY1 group, it was 21.58kPa before treatment and 20.14kPa after treatment. In the WKY2 group, it was 21.69kPa before treatment and 19.58kPa after treatment. In the WKY3 group, it was 21.69kPa before treatment and 22.21kPa after treatment.

As shown in Figure 4, myocardial apoptosis is also closely related to the blood lipid level of rats. In general, the levels of serum TC, TG, LDL-C, HDL-C in SHR-DW group were significantly lower than those in WKY group (P<0.01). After 10 weeks of treatment, serum TC, TG, LDL in

Figure 3.
Comparison of Blood Pressure of Rats in Each Group before and after Treatment ($\bar{x}\pm s$)

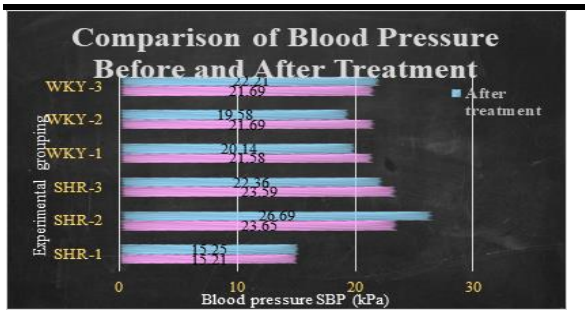
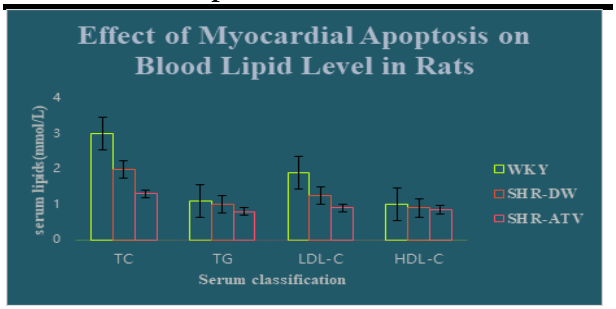


Figure 4.
The effect of myocardial apoptosis on blood lipid levels in rats



SHR-ATV group Compared with the SHR-DW group and the WKY group, the level of -C decreased significantly (P<0.01, P<0.05), while the level of HDL-C was not significantly different from that of the SHR-DW group (P> 0.05).

CONCLUSION

Hypertensive left ventricular remodeling is an important cause of other cardiovascular complications, and it is also closely related to acute myocardial infarction and malignant arrhythmia. Investigating the relationship between the induction of cardiomyocyte apoptosis and left ventricular function is because cardiomyocyte apoptosis is the cytological basis of left ventricular remodeling, by blocking the angiotensin II (AngII) can reduce cardiomyocyte apoptosis and ensure the normal operation of left ventricular structure. Recent studies have also developed many drugs, mainly focusing on the connection between the two. Using special growth factors, combined with receptors, can inhibit cell apoptosis and prevent the formation of fibrosis. It can improve and reverse the ventricular remodeling caused by hypertension to some extent [26].

Apoptosis actually has a full positive effect. When the human body cannot function normally, it will cause apoptosis. Including the production of free radicals, hypoxia, insufficient blood, etc. Among them, Caspase-3 is an important factor for performing cell apoptosis. The stimulating factor is used to activate cell apoptosis to the irreversible stage. At the same time, with the help of protein expression and blood lipid level display, the arteries and blood vessels are analyzed in the environment of high temperature and humidity. The apoptosis of tissue cells clarifies the changes in myocardial organization and the treatment plan of the left ventricle.

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