



A REVIEW ON CARDIAC HYPERTROPHY & TREATMENT STRATEGY

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ABSTRACT

The heart is capable of answering to stressful situations by extending muscle mass, which is broadly defined as cardiac hypertrophy. This process decreases ventricular wall stress for the heart undergoing a greater than normal workload. In starting stage, cardiac hypertrophy is associated with normal or increased cardiac function and is considered to be adaptive or physiological; while, at later stages, if the stimulus is not removed, it is affiliated with contractile dysfunction and is termed pathological cardiac hypertrophy. The exercise-induced extend in the capacity of pumping blood leads to thickening of the ventricle wall, insinuated as physiological hypertrophy, while the reduced capacity of pumping blood as a result of hypertension and volume overload on the heart indicates pathological hypertrophy. The prevention master plan for cardiac hypertrophy involves both surgically and medicinally like thiazide diuretics, angiotensin-converting enzyme (ACE) inhibitors, angiotensin (Ang) II receptor blockers, beta-blockers, and calcium channel blockers. In this review, we focus on significant knowledge regarding cardiac hypertrophy and current emerging approaches to forbid hypertrophy with the ultimate goal to prevent or delay the onset of heart failure and sudden death in patients.

KEYWORD: Cardiac hypertrophy, Physiological cardiac hypertrophy, Pathological cardiac hypertrophy, Treatment.

INTRODUCTION

Macroscopic gaining mark out cardiac hypertrophy (CH) as a thickening of the interventricular wall and/or septum¹; in the cell, it is characterized by an increment in cardiomyocyte size, with increased protein synthesis and altered in the management of the sarcomeric structure.^[1] It has been known since the 1990s that biochemical signaling events and alterations in gene expression are necessary for hypertrophic response.^[2-4] Cardiac hypertrophy is a disease connected by the heart in which the ventricle wall of the heart becomes thickened, for the most part, the left ventricle, which is more susceptible to hypertrophy. In this disease, the ability to pump the blood achieves decreased that ultimately outcome in cardiac malfunction.^[5,6] Cardiac hypertrophy is respects as the myocardial retort to a difference of extrinsic and intrinsic stimuli that impose elevated biomechanical stress and diagnosis is generally based on the calculated echocardiographic or magnetic resonance imaging evaluate of the left ventricular mass. Additionally, cardiac hypertrophy may be arranged as physiological and pathological hypertrophy.^[7-8] Cardiac hypertrophy is present in various types of heart diseases are ischemic heart disease, hypertension, and heart failure, and valve diseases. On the behalf of pathogenic point of view, these various mechanisms induce

cardiomyocyte growth, due to an increase in mechanical stress or in response to an increase in neurohormonal stimulation.^[9-11] This physiological cardiac hypertrophy is mostly accompanied by elevated mitochondrial energy production.^[12] Pathological cardiac hypertrophy is allied with a deterioration of cardiac contraction and energy metabolism, whereas physiological cardiac hypertrophy is allied with an improvement in normal cardiac contractile function at rest.^[13] Both physiological and pathological cardiac hypertrophy give back changes in wall thickening and ventricular volume, though pathological cardiac hypertrophy is distinguished by severe cardiac remodeling that ultimately leads to heart failure (HF) and dysfunction.^[14] Different types of signaling mechanisms are involved in the pathogenesis of cardiac hypertrophy which are MAPK, PKC, NFAT, PI3K-AKT/PKB, M-TOR, and IGF-1.^[15-18] Signal transduction in retort to mechanical stress is distinguished by the diversity of pathways that are activated at the same time.^[19] According to the discovery so far, some such methods have been discovered for the treatment of cardiac hypertrophy which are proving effective in this include, beta-blockers, thiazide diuretics, angiotensin-converting enzyme (ACE) inhibitors and Ang-II receptor blockers, calcium channel blockers.^[20-24] This review about cardiac hypertrophy and its types as

well as the treatment master plan for cardiac hypertrophy has also been described in this review.

Classification of Cardiac Hypertrophy

Two types of cardiac hypertrophy have been found which pathological and physiological cardiac hypertrophy are. Pathological and physiological hypertrophy can be subclassified as concentric or eccentric based on changes in shape that are dependent on the initiating stimulus.^[25,26] Cardiac hypertrophy is classified as physiological hypertrophy when it is related with normal or increased cardiac function and is generally seen to happen in reaction to the growth of the body, pregnancy, or exercise.^[27] Physiological cardiac hypertrophy is distinguished by mild heart growth, as it is commonly associated with a 10-20% increase in heart weight normalized to body weight.^[28] Cardiac hypertrophy is classified as pathological hypertrophy when it is related with cardiac dysfunction.^[29] Pathological cardiac hypertrophy is generally caused by cardiovascular disease (CVD).^[30] Pathological cardiac hypertrophy can ultimately contribute to the development of heart failure.^[31] A pathological stimulus brings about volume overload produces growth in diastolic wall stress resulting in eccentric hypertrophy which is distinguished by large dilated cavities and relatively thin walls in the myocardium while a pathological stimulus causing pressure overload produces growth in systolic wall stress that results in concentric hypertrophy which is characterized by thick walls and remarkably small cavities in the myocardium.^[8,25,26] In pathological hypertrophy, the enlargement of cardiac myocytes including progression

of new sarcomeres normalizes ventricle wall stress and allows normal cardiovascular function at rest, called compensated growth. Isometric exercise like weight lifting results in muscular tension development in opposition to resistance and the mechanical changes produces pressure load on the heart resulting in concentric hypertrophy.^[32,33] Isotonic exercises are running, walking, cycling and swimming involve the movement of large muscle groups. The severe vasodilatation of the skeletal muscle vasculature results in eccentric hypertrophy by increasing the venous return to the myocardium causing volume overload, distinguished by chamber enlargement and a proportional switch in wall thickness.^[25,34]

In the different forms of heart failure, immoderate cardiac workload lead to an enlargement of the heart in an endeavor to handle the increased hemodynamic demand. This mechanism, known as hypertrophy, is classified as “physiological” hypertrophy when it occurs in healthy individuals following exercise and is not related with cardiac damage, or “pathological” hypertrophy (Fig. 1). In pathological hypertrophy, albeit the increased heart size is initially a balancing mechanism, continued hypertrophy can ultimately lead to a decline in left ventricular function and hereby represents an independent risk factor for heart failure.^[35] Additionally, function in the heart which suffers from cardiac hypertrophy eventually decompensate in the last leading to left ventricle dilation and heart failure while physiological hypertrophy does not decompensate into dilated cardiomyopathy or heart failure.^[8,25,34,36]

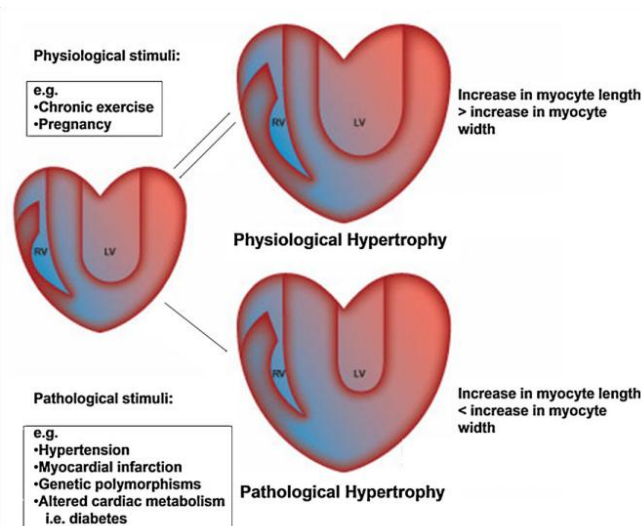


Fig. 1: Pathological and physiological stimulation triggers pathological and physiological hypertrophy.

Treatment of Cardiac Hypertrophy

The treatment of cardiac hypertrophy with exercise, monitoring blood pressure and use of antihypertensive drugs. For the treatment of cardiac hypertrophy different class of drugs have been recommended to have a modulatory role in the treatment of cardiac hypertrophy

and the drugs are thiazide diuretics (chlorthiazide, hydrochlorothiazide); ACE inhibitors (enalapril, lisinopril, and captopril); Ang-II receptor blockers (Losartan, Azilsartan); β -blockers (atenolol, carvedilol, metoprolol, bisoprolol); Calcium channel blockers (amlodipine, diltiazem, nifedipine, and

verapamil).^[22,24,37] Hypertrophic cardiomyopathy can manage effectively both medicinally and surgically and should now be premeditated as a curable genetic disease affiliated with a normal life span and low disease-associated mortality.^[38] Septal reduction therapy is beneficial for patients with medically refractory heart failure symptoms due to obstructive HCM. Extended transaortic septal myectomy and alcohol septal ablation decrease left ventricular outflow tract (LVOT) obstruction and improve functional condition.^[39] Septal myectomy is a surgical treatment for cardiac hypertrophy in which removing of a portion of the septum which was obstructing the stream of blood from the left ventricle to the aorta.^[40,41] A different technique of surgical procedure is Alcohol septal ablation (ASA) is an effective procedure if performed in an experienced institution and may settle symptoms in a subset of patients with obstructive hypertrophic cardiomyopathy.^[42] ASA technique in which reduces the obstruction to the blood being ejected from the heart. The technique makes a mild controlled heart attack during which the area of cardiac muscle responsible for the obstruction is killed that ultimately creates the obstructed area thin.^[43]

CONCLUSION

Cardiac hypertrophy has become a very big and important all over the world. We have presented some important things that are helpful in understanding cardiac hypertrophy and also its treatment. In this review, both types of cardiac hypertrophy physiological and pathological cardiac hypertrophy are explained and the difference between them is also shown. Although both pathological and physiological cardiac hypertrophy are affiliated with an increase in heart mass, pathological hypertrophy is affiliated with a complex arrangement of events, comprising up regulation of fatal genes, histopathology, and cardiac dysfunction while physiological hypertrophy is affiliated with normal cardiac structure and normal or enhanced cardiac function. An important challenge for the future is that we can acquire as much information as possible about cardiac hypertrophy disease and use it in its treatment and this knowledge translates into novel pharmacological and gene therapeutic treatments for pathological cardiac hypertrophy and cardiac failure. Hence there is a need for new studies which can suggest potential drug therapies that could surely reduce cardiac hypertrophy and save the life for long time.

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