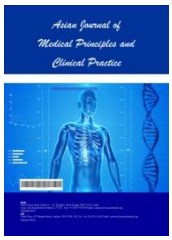

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Hypertrophic Cardiomyopathy: Causes, Investigations, and Treatment

Omar Elsaka [a*]

[a] *Department of Cardiology, Mansoura University, Faculty of Medicine, Mansoura Manchester Medical Program (MMMP), Mansoura, Egypt.*

Author's contribution

The sole author designed, analyzed, interpreted and prepared the manuscript.

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ABSTRACT

Background: Hypertrophic cardiomyopathy (HCM) is the most common hereditary heart disease with a variety of genotypes and phenotypes. In previous terms, hypertrophic obstructive cardiomyopathy and idiopathic hypertrophic subaortic stenosis are no longer used to describe this business. Patients may or may not have a ventricular outflow tract (LVOT). Restlessness or LVOT inhibition occurs in 70% of patients and is the most common cause of heart failure. The pathology of HCM includes left ventricular hypertrophy with or without right ventricular hypertrophy, contractile mitral valve advancement, flexible and flexible LVOT inhibition, mitral valve recurrence, diastolic dysfunction, myocardial ischemia, and Includes fibrosis. A complete understanding of pathology and pathophysiology is essential for neural control and surgery.
Conclusion: Hypertrophic Cardiomyopathy requires invasive treatment. Alcohol Septal ablation and surgery are two common methods of invasive treatment.

Keywords: Heart failure; hypertrophic cardiomyopathy; left ventricular outflow obstruction; mitral regurgitation; systolic anterior motion.

**Corresponding author: E-mail: omarelsaka0808@gmail.com;*

1. INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is a genetic disorder that is usually acquired through autosomal treatment with flexible entry. The disease has complex symptoms and potentially dangerous consequences for patients and their families. The disease has a variable presentation and carries the incidence of sudden death. In fact, HCM is one of the leading causes of sudden cardiac death in newborns and adolescents. A notable feature of this disease is myocardial hypertrophy, which is usually asymmetric and occurs when there is no obvious stimulus for the hypertrophic stimulus. This hypertrophy can occur in any region of the left ventricle, but generally affects the interventricular septum, resulting in obstruction of the left ventricular (LV) outflow tract. Decades ago, HCM was documented and known as idiopathic hypertrophic subaortic stenosis (IHSS) or asymmetric septal hypertrophy (ASH). These terms are replaced with hypertrophic cardiomyopathy, as segmental hypertrophy can occur anywhere in the ventricle, not just the septum. Also, this matter can be quiet without subaortic flow obstruction, but it still carries the same dire risk of arrhythmogenic sudden death and its many clinical symptoms [1].

HCM can be divided into blocking and non-blocking types. The inhibition of HCM is caused by an obstruction to the flow of the LV midsystolic tract due to the movement of the systolic anterior mitral valve caused by Bernoulli. However, the importance of this ban is an important issue. Some researchers and experts believe that the inhibition is less closely related to the hemodynamic and pathophysiological manifestations of this organism than to segmental hypertrophy, which, along with its increased myocardial oxygen uptake and the substrate for fatal ventricular arrhythmias, is of great concern, as well as the treatment and diagnosis of HCM . HCM is a family disease. There are defects in a few genes that code for sarcomere proteins, such as: B. the heavy chain of myosin, actin, tropomyosin and titin. Many mutations have been identified that are associated with the risk of death and hypertrophy associated with the genotype. Interestingly, the genetic basis of ventricular hypertrophy is not directly related to prognostic risk stratification. Patients with certain changes, such as certain tropomyosin mutations, have low levels of ventricular hypertrophy with little or no LV flow, but still have an unparalled risk of sudden death [2].

1.1 Causes and Risk Factors

Abnormal calcium kinetics: Abnormal myocardial calcium kinetics is responsible for some of the features of malignant myocardial hypertrophy and HCM, especially in patients with diastolic functional impairment. Abnormal myocardial calcium kinetics and abnormal calcium flux can lead to an increase in intracellular calcium concentrations, which can lead to cell hypertrophy and proliferation due to an increase in the number of calcium channels [3].

Genetic causes: Familial HCM occurs in about 50% of cases as an acquired disease as a highly autosomal recessive disorder. Some, if not all, rare forms of the disease can be caused by autoimmune mutations. At least 6 different genes on at least 4 chromosomes are associated with HCM, more than 50 different mutations have been found to date. Family HCM is a genetic disorder because it can be caused by a genetic defect in more than one area. In 1989, Seedman and participants first reported the genetic basis of HCM. They reported the presence of a genetic disorder found on the long arm of chromosome 14. They then discovered that it was a type of beta cardiac myosin heavy chain code. There are wide differences in the phenotypic manifestations of this genetic mutation, as well as the variability of clinical symptoms and the degree of severe hypertrophy. Phenotypic variability is associated with differences in genotypes, specific mutations associated with specific symptoms, levels of hypertrophy, and prognosis [4].

Other possible causes of HCM are as follows: Abnormal sympathetic stimulation: Overproduction of catecholamines or excessive intake of neural noradrenaline can cause HCM. Myocardial ischemia; It progresses to myocardial fibrosis and abnormal compensatory hypertrophy. Subendocardial ischemia: this is associated with abnormal cardiac microcirculation, which eliminates the accumulation of energy important for calcium absorption during diastole; Subendocardial ischemia leads to continuous interaction of contraction factors between diastole and an increase in diastolic rigidity and cardiac instability: it involves obstruction of the catenoid septum leading to myocardial hypertrophy and rupture [5].

1.2 Pathophysiology

One factor that has received much attention since the early definitions of hypertrophic cardiomyopathy (HCM) is the drop in dynamic pressure throughout the LV outlet. The pressure gradient by anterior systolic movement of the mitral valve against the hypertrophied septum appears to be related to a sustained contraction of the already constricted leaflet (already less anomalous septal hypertrophy and possibly abnormal area of the mitral valve). Three explanations have been given for systolic elevation of the mitral valve: the mitral valve is pulled against the septum by papillary muscle contraction, which is caused by abnormal valve position, and septal hypertrophy that changes papillary shape. Muscles; The mitral valve is pressed into the septum due to the irregular shape in the exhaust channel; The low pressure created when blood is pumped out through a narrow outlet at high speed (Venturi effect) causes the mitral valve to be pulled into the septum. Most patients with HCM have abnormal diastolic activity (whether there is a pressure gradient or not) that impairs ventricular filling and increases filling pressure despite a normal or narrow chamber cavity. Abnormal calcium kinetics and subendocardial ischemia in these patients are associated with severe hypertrophy and a myopathic process [6].

1.3 Types of Hypertrophic Cardiomyopathy

The main components of HCM are HCM blocking and non-blocking. In addition, HCM inhibition can be defined as sub-aortic and mid-ventricular based on the site of inhibition. Alternatively, HCM inhibition can be defined as normal systolic activity or systolic dysfunction (Fig. 1) [7].

1.4 Obstructive HCM

Obstruction HCM is a type of HCM characterized by enlarged myocytes of the heart which causes the walls of the LV to grow. Hypertrophy results in obstruction of intraventricular blood flow. Decreased LV flow (LVOT) is a manifestation of the HCM blocking factor and is a common cause of shortness of breath, chest pain, and syncope. Decreased LVOT may be due to anatomical (low cross section LV discharge leaflets) and hemodynamic properties [8].

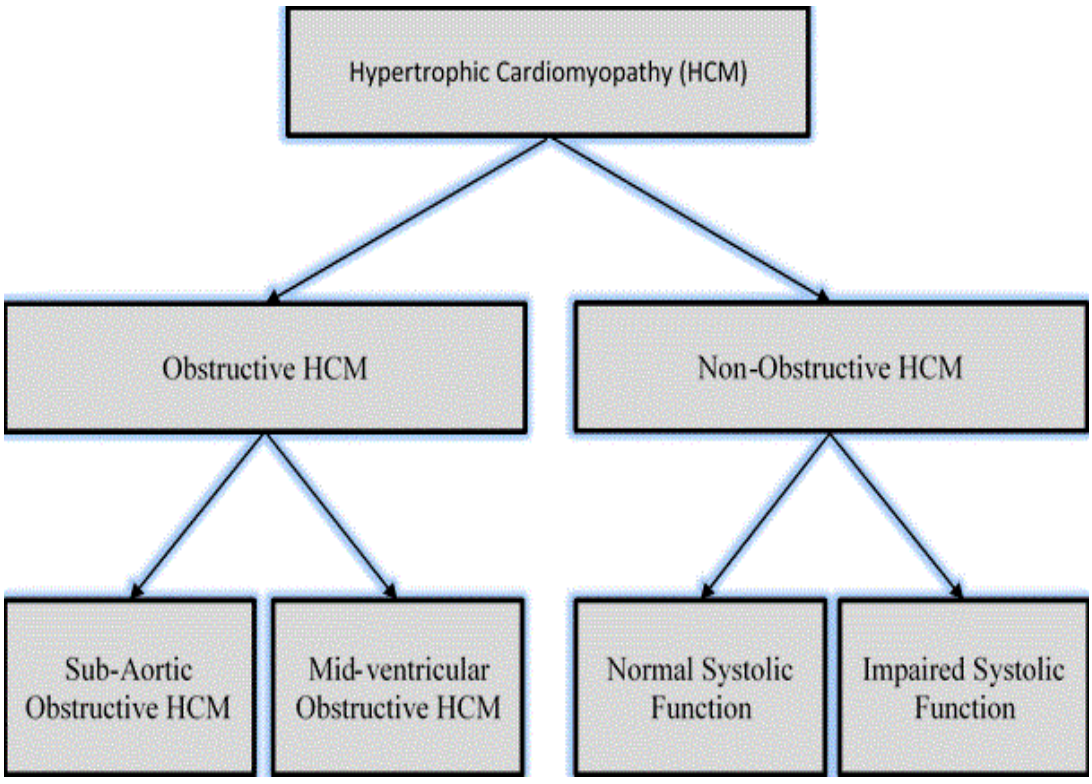

Fig. 1. Types of Hypertrophic Cardiomyopathy [7]

1.4.1 Sub-aortic obstructive HCM

HCM for subaortic occlusion surgery provides a combination of basal septum stiffness, mitral valve pre-extraction, and mitral valve pre-contraction movement, contributing to LV outlet occlusion. Several mechanisms can induce contractile movements within the mitral valve. The first is the force of the Venturi, which reduces the output at the LV outlet, increases the velocity, and then reduces the pressure on the mitral valve, causing the valve to move toward the septum. The second is the flow. The anatomical structure of the mitral valve and pre-excavated papillary muscles is modified to push the mitral valve out of the LV outlet and expose it to traction flow. Redness leads to the gradual development of internal contractile movements of the mitral valve [9].

1.4.2 Mid-ventricular obstructive HCM

Falicov and Resnekov were the first to describe HCM ventricular obstructive HCM in 1977. It is a rare type of HCM with an hourglass shaped LV cavity and a separate apical chamber. Echocardiographic systolic pressure in the apical chamber is higher but more common than medial ventricular obstruction compared to sub-aortic obstructive HCM when systolic pressure rises throughout the LV cavity. Mid-ventricular HCM obstruction also does not show mitral deficiency but has a high symptom load and indicative of disease progression and HCM end-stage. It is a prediction of sudden HCM-related death and fatal arrhythmia. Apical aneurysm formation between HCM ventricular obstructive is common, indicating progression of end-stage disease and increased risk of stroke, sudden death or fatal arrhythmia [10].

1.5 Non-Obstructive HCM

At non-obstructive HCM, the cardiac pump is still intact. This reduces the amount of blood that the ventricle can absorb and pump out, but blood flow is not blocked [10].

1.5.1 Normal/impaired systolic function

Non-inhibitory HCM is clinically defined as a variant of HCM characterized by the absence of obstruction of the LV tract, with a gradient of <30 mmHg both under exercise conditions and at rest. Its main classification is HCM with normal or impaired systolic function (end stage). Depressed LV systolic activity may be caused by myocardial ischemia and infarction due to the large number of non-invasive HCM patients present with myocardial fibrosis. Patients with non-inhibitory HCM experience severe symptoms due to diastolic dysfunction and microvascular ischemia. These symptoms are difficult to treat and control due to the lack of LV discharge form which is usually the goal of treatment [11].

1.5.2 Apical hypertrophic cardiomyopathy

Apical HCM is another rare form of HCM that does not affect the density of the myocardium in the left ventricular apex. It is very rare for the disease to affect the tip of the right ventricle (RV), or both the tips of LV and RV. The apical HCM was initially thought to be limited to the Japanese, but the apical HCM also affects foreigners, but the prevalence is much lower than the Japanese. Common ventricular angiography findings associate apical HCM with end-diastolic spinal LV space. Most patients with apical HCM have a good prognosis with an annual mortality rate of 0.1%. Some patients with severe symptoms of diastolic dysfunction due to LV chamber stenosis. Approximately 10% of patients develop apical infarction without coronary artery disease (CAD), which can lead to the formation of apical aneurysms. Sudden cardiac death is unlikely to occur in apex HCM, but the formation of apex aneurysms is associated with adverse consequences such as sudden cardiac death, progressive heart failure, and thromboembolic events, and is an annual event. The rate is 10.5% [12].

1.6 Signs and Symptoms

Symptoms of hypertrophic cardiomyopathy (HCM) may include dyspnea, syncope and presyncope, angina, decreased heart rate, orthopnea, paroxysmal nocturnal dyspnea, heart failure, dizziness, and sudden cardiac death. Sudden cardiac death: This is the most devastating HCM manifestation. It has a high incidence of pre-teens and teens and is particularly associated with hard labor. The risk of sudden infant mortality is as high as 6% per year. In more than 80% of cases, the arrhythmia that causes sudden death is ventricular fibrillation. Many of these conditions are aggravated by acute arterial arrhythmias, such as fibrillation, supraventricular tachycardia, or Wolf-Parkinson-White syndrome, while others cause ventricular tachycardia and collapse. Hemodynamic cardiac output: Shortness of breath: This is the most common symptom, seen

in about 90% of patients with symptoms. Dyspnea is primarily the result of increased diastolic filling of the LV (as well as transfer of that high pressure into the pulmonary circulation). High pressure at LV filling is mainly due to diastolic instability due to signs of ventricular hypertrophy [13].

Fainting: Syncope is a very common symptom due to insufficient heart rate or cardiac arrhythmias during exercise. It is most common in children and young adults with small LV chamber sizes and evidence of ventricular tachycardia in ambulatory. Alternatively, fainting may be caused by arrhythmias, tachycardia, or bradycardia. Some patients with HCM have abnormalities in sinus node function, leading to alternating tachyarrhythmias and bradyarrhythmias or chronic sinus syndrome with severe bradyarrhythmias. Syncope and presyncope identify patients at high risk of sudden death and authorize emergency operations and aggressive treatment. Presyncope: Presyncope has "graying-out" spells that occur in an upright position and can be quickly relieved by falling asleep. They are more frequent and identify patients who are at higher risk of sudden death. These symptoms are exacerbated by vaginal stimulation. Presyncope may occur with atrial or chronic ventricular tachyarrhythmias. Angina: The general symptoms of angina are very common in patients with HCM and may occur in the absence of visible coronary atherosclerosis. Disrupted diastolic relaxation and a significant increase in myocardial oxygen consumption are due to ventricular hypertrophy, leading to subendocardial ischemia, especially during exercise. Heart palpitations: Heart palpitations are normal. It is caused by arrhythmias, such as premature and ventricular atrial fibrillation, sinus suspension, atrial fibrillation, atrial flutter, supraventricular tachycardia, and ventricular tachycardia [14].

Orthopnea and paroxysmal nocturnal dyspnea: These are the first symptoms of congestive heart failure and, although rare, are seen in patients with severe HCM. They are caused by diastolic dysfunction and high pressure to fill the LV. Orthopnea and paroxysmal nocturnal dyspnea are caused by venous pulmonary congestion. Severe heart failure: This is rare but is seen in patients with severe HCM. It may occur due to a combination of diastolic dysfunction and subendocardial ischemia. Systolic activity in these patients is almost always well maintained. Dizziness: Dizziness is common in patients with HCM with a high pressure gradient throughout the LV outflow tract. Excessive exercise is worse and may be exacerbated by hypovolemia following high levels of exertion or an increase in unexplained fluid loss (e.g., during extreme heat). Dizziness can also occur as a result of actions, such as stopping or Valsalva at the time of delivery, or certain medications, such as diuretics, nitroglycerin, and vasodilating antihypertensive agents, reduce preload and subsequent loading and increase pressure throughout the discharge sheet. LV. Dizziness may also be secondary to arrhythmia-related hypotension and decreased cerebral perfusion. Chronic arrhythmia usually causes symptoms of dizziness, lightheadedness, and presyncope, while persistent arrhythmias are more likely to lead to syncope, collapse, and or sudden cardiac death [15].

1.7 Complications

Most people with hypertrophic cardiomyopathy (HCM) do not have major health problems. But complications of hypertrophic cardiomyopathy can include: Atrial fibrillation: Abnormal formation of heart cells along with strong heart muscle can lead to changes in the cardiovascular system, causing a rapid or abnormal heartbeat. Atrial fibrillation can also increase the risk of blood clots, which can travel to the brain and cause a stroke. Stopped blood flow: In many people, heart failure blocks blood flow to and from the heart, resulting in fatigue, chest pain, dizziness, and fainting. Mitral valve problems: If a rigid heart muscle blocks blood flow to and from the heart, the valve between the left atrium and the left ventricle (mitral valve) may not close properly. As a result, blood can leak back into the left atrium (mitral valve regurgitation), making symptoms worse [16].

Dilated cardiomyopathy: In a very small number of people with HCM, a strong heart muscle may be weak and inactive. The ventricle expands and its pumping capacity decreases. Heart Failure: An overworked heart muscle can be too strong to effectively fill the heart with blood. As a result, your heart cannot pump enough blood to meet your body's needs. Sudden cardiac death: In rare cases, hypertrophic cardiomyopathy can cause sudden cardiac death in people of any age. Since most people with hypertrophic cardiomyopathy don't realize they have it, sudden cardiac death can be the first sign of a problem. This can happen to healthy-looking

youth, including high school athletes and other active young adults [17].

1.8 Prevention

There is no prevention of hypertrophic cardiomyopathy. But it is important to assess the condition as soon as possible to guide treatment and prevent complications. If you have parents, siblings or relatives of a child with hypertrophic cardiomyopathy, a doctor may recommend genetic testing to diagnose the condition. However, there are currently no significant changes in all people with HCM. Also, some insurance companies do not cover genetic testing. If the genetic test is not done or the results are not helpful, your doctor may recommend an echocardiogram every time you have a family member with hypertrophic cardiomyopathy. Young people and athletic competitors should be tested once a year. Adults who do not participate in athletics should be screened every five years [18].

1.9 Investigations

Your doctor will probably order a test to diagnose hypertrophic cardiomyopathy (HCM) or rule out other conditions that may cause similar symptoms [18].

1.10 Echocardiogram

Echocardiogram is often used to diagnose hypertrophic cardiomyopathy. This test uses sound waves (ultrasound) to see if your heart muscles are abnormally tight. It also shows how well your heart's valves and valves pump blood. Sometimes, an echocardiogram is done during exercise, usually on a treadmill. This is called an exercise stress test. Treadmill stress tests are often used to diagnose people with hypertrophic cardiomyopathy (Fig. 2) [19].

1.11 Electrocardiogram (ECG or EKG)

The electrodes are attached to adhesive pads that are placed on the chest and sometimes on the legs. Measure your heart's electrical signals. EKG can show symptoms of abnormal heartbeat and heart failure. In some cases, a portable EKG called a halter monitor is required. This device records your heart rate continuously for one or two days (Fig. 3) [20].

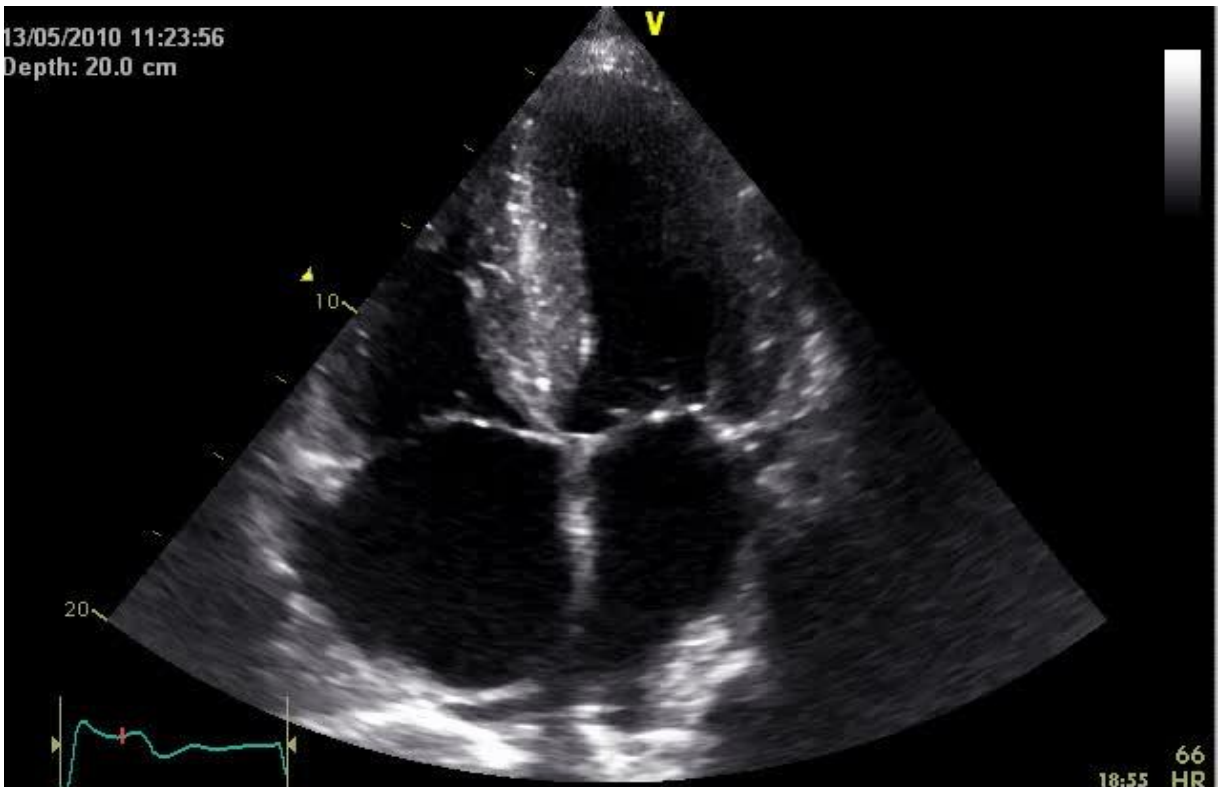

Fig. 2. Echocardiogram for Hypertrophic Cardiomyopathy [19]

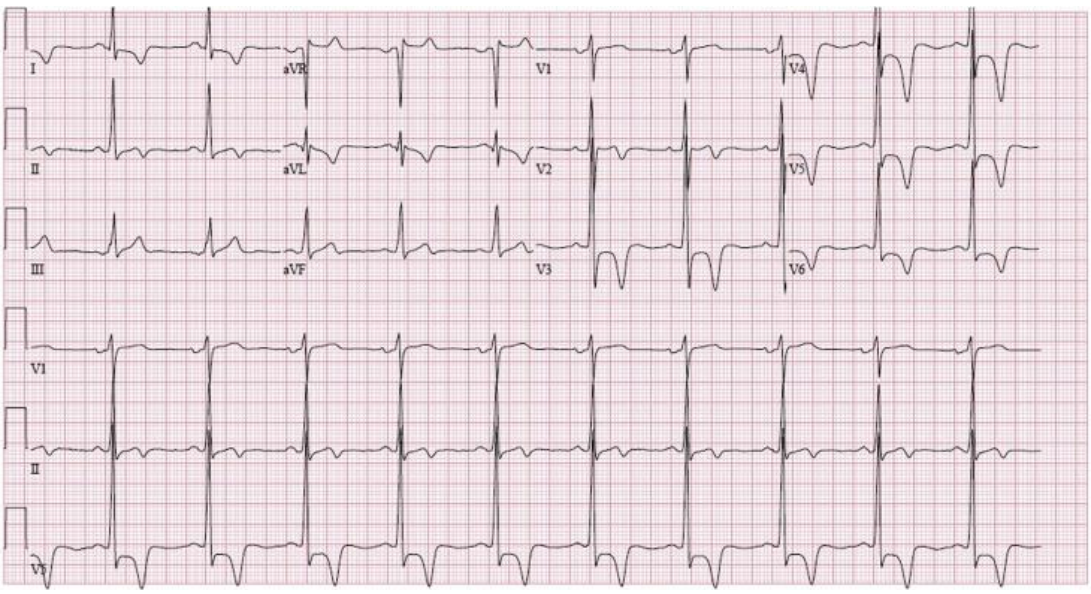

Fig. 3. Apical Hypertrophic Cardiomyopathy [20]

1.12 Cardiac MRI

Cardiac MRI uses powerful magnets and radio waves to create images of your heart. It gives your doctor information about your heart muscle and shows how your heart and heart valves work. These tests are usually done with an echocardiogram (Fig. 4) [21].

1.13 PET/CT Assessment

Positron emission tomography (PET) scans show how your tissues and organs function. Radiated tracers called radionuclides are used during the test to show cardiac function. PET scans are useful for detecting chemical reactions in different parts of your body to detect cancer, heart disease, and mental disorders. Because PET scans measure the effectiveness of chemicals, they can help detect infections before other imaging tests detect them (Fig. 5) [22].

1.14 Treatment

The goal of treatment for hypertrophic cardiomyopathy is to relieve symptoms and prevent sudden cardiac death in high-risk individuals. Your specific treatment depends on the severity of your symptoms. Together, you and your doctor will discuss the most appropriate treatment for your condition (Fig. 6) [23].

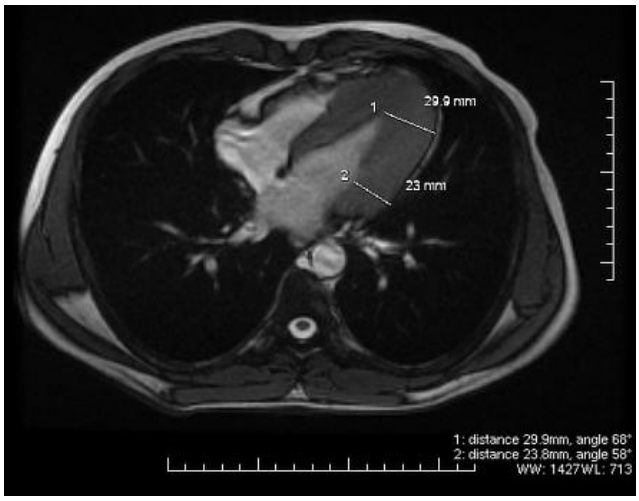

Fig. 4. MRI for Hypertrophic Cardiomyopathy [21]

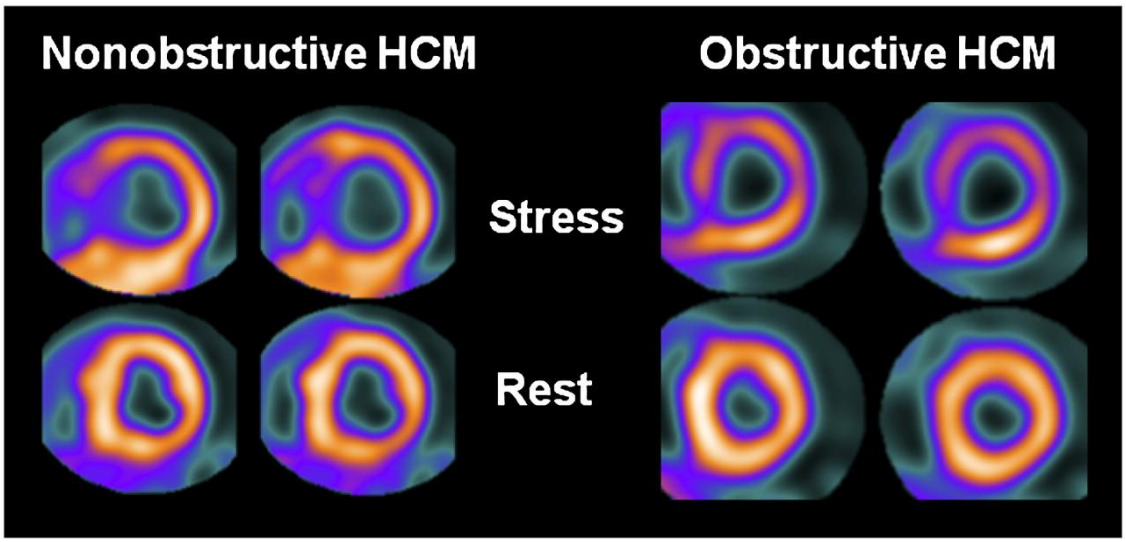

Fig. 5. PET/CT Assessments for Obstructive and Non-Obstructive Hypertrophic Cardiomyopathy [22]

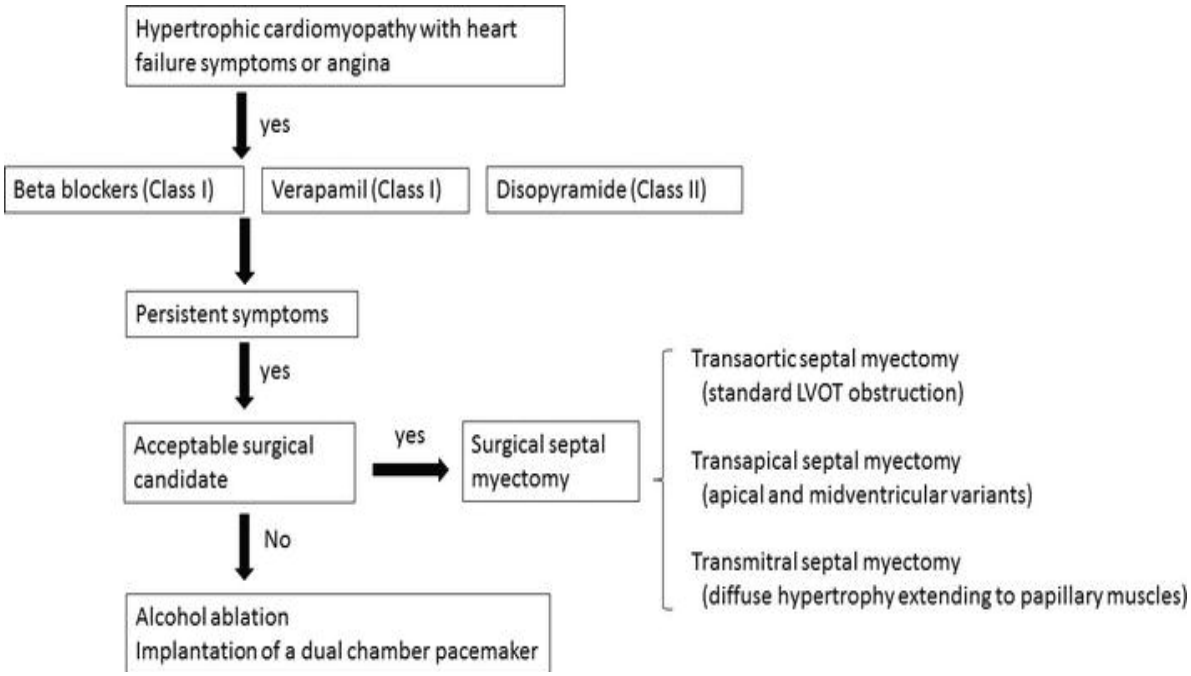

Fig. 6. Decisions in Management of Hypertrophic Cardiomyopathy [23]

Medications can help reduce the risk of coronary heart disease and slow down the heartbeat so that the heart can pump blood better. Remedies for treating hypertrophic cardiomyopathy and its symptoms may include: Beta blockers such as metoprolol (Lopressor, Toprol-XL), propranolol (Inderal, Innopran XL) or atenolol (Tenormin), Calcium blockers such as -verapamil (Verelan, Calan SR,) or diltiazem (Cardizem, Tiazac), cardiac drugs such as amiodarone (Pacerone) or disopyramide (Norpace) and blood thinners such as warfarin (Coumadin, Jantoven), dabigatran (Pradaxa), rivaroxaban (Xarelto) or apixaban to prevent (Eliquis) blood clots if you have atrial fibrillation [24].

1.14.1 Surgeries or other procedures

There are many different surgeries or procedures available to treat cardiomyopathy or its symptoms. These range from open heart surgery to implanting heart rates [24].

1.14.2 Septal myectomy

If the drug does not improve symptoms, this open heart surgery may be recommended. It involves removing part of the thick, thick wall (septum) between the heart chambers. Septal resection helps improve blood flow outside the heart and reduce regurgitation of blood flow through the mitral valve (mitral valve regurgitation). Surgery can be performed using a variety of methods, depending on the location of the congestive myocardium. In one case, with apical myectomy, the surgeon removed the thickened myocardium near the tip of the heart. The mitral valve may be adjusted at the same time (Fig. 7) [25].

1.14.3 Septal ablation

This process destroys strong heart muscles with alcohol. The alcohol is injected into a long catheter into a blood vessel in that area. Potential complications include disruption of the cardiovascular system (heart block), which requires the installation of a pacemaker (Fig. 8) [26].

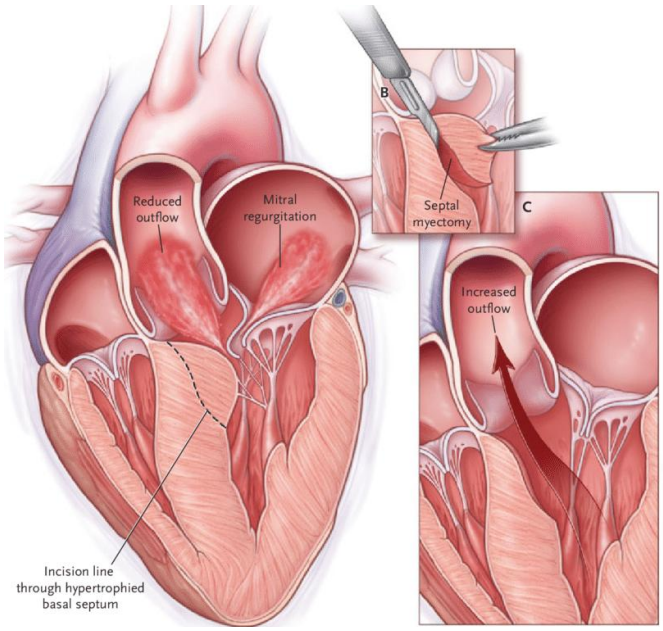

Fig. 7. Septal myectomy [25]

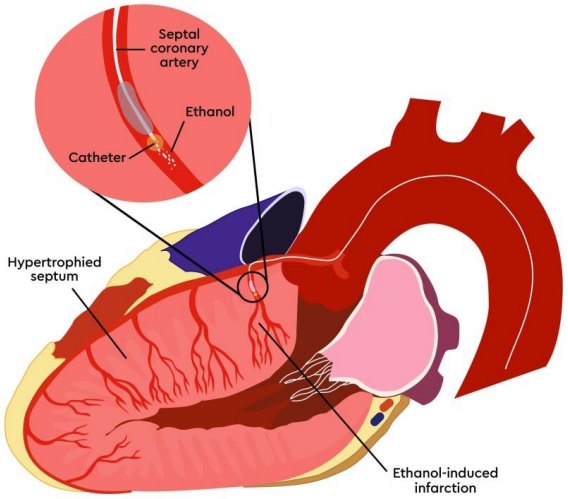

Fig. 8. Alcohol septal ablation [26]

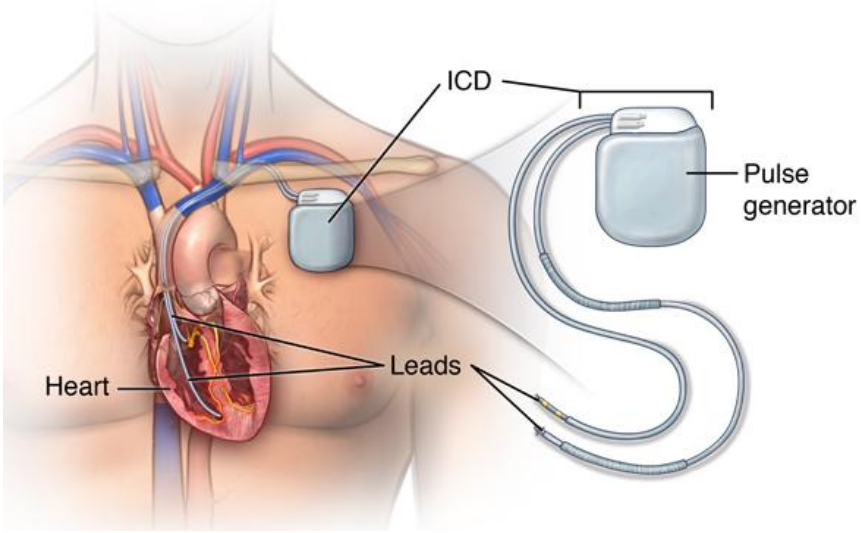

Fig. 9. Implantable Cardioverter Defibrillator (ICD) [27]

1.15 Implantable Cardioverter-defibrillator (ICD)

An ICD is a small device that continuously monitors your heartbeat. Stuck in chest like a pacemaker. In the event of a life-threatening arrhythmia, the ICD delivers a precise electric shock to restore a normal heart rhythm. ICDs have been shown to help prevent sudden cardiac death that occurs in a small number of people with hypertrophic cardiomyopathy (Fig. 9) [27].

2. DISCUSSION

Hypertrophic cardiomyopathy (HCM) is a genetic disorder characterized by left ventricular hypertrophy that can be explained by secondary left ventricle with secondary causes and preserved or elevated output. Usually severe asymmetric hypertrophy involving the basal ventricular septum. Left ventricular tract obstruction relaxes in approximately one third of patients and can cause inflammation in another third. Histologic symptoms of HCM include myositis hypertrophy and dislocation, as well as interstitial fibrosis. Hypertrophy is often associated with left ventricular diastolic dysfunction. For most patients, HCM has a relatively good course. However, HCM is one of the leading causes of sudden cardiac death, especially in adolescents and young adults [28].

Nonsustained ventricular tachycardia, syncope, family history of sudden cardiac death, and severe cardiac hypertrophy are important risk factors for sudden cardiac death. Introducing a cardioverter defibrillator in appropriate, high-risk patients can usually prevent this problem. Atrial fibrillation is a common problem and is not well tolerated. Genetic modification of twelve sarcomere-related proteins leads to HCM. MYH7 and MYBPC3, the complex chains of β-myosin-coding and protein C-binding myosin, respectively, are the two most involved genes and together form about 50% of the HCM families. Underlying genes are still being identified in ≈40% of HCM patients. Genetic mutations in the pathogen also produce the HCM-like phenotype (genotype or phenocopy). The joint use of genetic testing and early detection of family members represents significant advances. Genetic knowledge has improved the understanding of the pathogenesis of HCM cells and has promoted efforts to identify new therapeutics [29].

3. CONCLUSION

HCM is the most common genetic disease of the heart and blood vessels. The phenotypic expression results in various patterns of LV hypertrophy and abnormal mitral valve function. Up to 70% of affected people show remission or inhibition of irritated LVOT. Inhibition of LVOT, MR, diastolic dysfunction, myocardial ischemia and scar formation at risk of dangerous ventricular arrhythmias are major pathophysiologic processes undergoing symptomatology. Patients are initially treated medically; however, significant symptoms of heart failure or syncope despite appropriate drug treatment and significant LVOT prevention

require aggressive treatment. Alcohol removal and ablation surgery are two common methods of invasive treatment.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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