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A REVIEW ON: HYPERTROPHIC CARDIOMYOPATHY

HCM, ITS PHENOTYPES, DIAGNOSIS AND TREATMENT

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Abstract: The most common congenital heart disease is hypertrophic cardiomyopathy (HCM). According to certain historical research, HCM affects roughly one in every 200–500 people, and is also a common reason behind SCD or arrhythmias in young athletes. HCM is seen in almost every age group of people. The morphological manifestations are highly variable, and range from asymptomatic to failure of the heart, and sudden or unexpected cardiac death. In the majority of people, hypertrophy of the left ventricle, and altered ventricular morphology induce dynamic LVOT obstruction. HCM symptoms are treated with pharmacological treatments, including septal reduction therapies and other pharmacotherapies. Another beneficial therapy for people with HCM is surgical myectomy of the heart. Several pharmaceutical medicines, such as β-blockers, are also discussed in this study. Some phenotypic variants of HCM are explained in this article. cMRI, electrocardiography, and echocardiography are some of the powerful tools used for HCM diagnosis. The main clinical concerns and therapy options for HCM are summarized in this study.

Keywords **- HCM, Idiopathic Hypertrophic Subaortic Stenosis, Apical HCM, Asymmetric HCM, LVOT Obstruction.**

I. INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is a congenitally and practically varying disorder. It is a disease characterized commonly by hypertrophy of left ventricles in a possible range, it possibly outcomes, including sudden or unexpected cardiac death or heart failure but also normal life survival anticipation. Identification of HCM is crucial and important, both for giving prevention strategies and for treatment, and in stimulating the incorporating of clinically as well as genetically surveillance of family members.[1] Hypertrophic cardiomyopathy is a complex, however relatively common form of genetic heart muscle disorder that happens in one out of 500 humans, however often is going undiagnosed withinside the community, and has caused some confusion to each sufferer and physicians periodically over the years. HCM is the maximum and common reason of heart-associated unexpected dying in human beings under 30 years of age, and it may also be chargeable for exercising disability at almost any age. Although HCM is a lifelong disorder without a recognized cure, a number of treatments at the moment are available to alter its course.[2]

Hypertrophic cardiomyopathy is a genetic cardiac disorder resulting from mutations in certainly considered one among at the least 12 sarcomer or nonsarcomer genes and is identified as the very common reason of unexpected or surprising cardiac death (SCD) in the younger and a crucial substrate for disability at any age. Technological traits coupled with superior knowledge of epidemiology, clinical course, and molecular defects responsible for HCM have promoted new diagnostic management strategies.[3] HCM is categorized as obstructive or non-obstructive depending on whether normal coronary heart anatomy of the left ventricle obstructs or does not obstruct the blood outflow. The obstructive type of HCM can be known as hypertrophic obstructive cardiomyopathy. Historically, it is mentioned as "Idiopathic Hypertrophic Subaortic Stenosis." In the past, the HOCM was also known as asymmetric septal hypertrophy. Apical hypertrophic cardiomyopathy, also known as Yamaguchi syndrome, is a non-obstructive type of variant of HCM. It was initially observed in people of Japanese ancestry. It became first described in individuals of Japanese descent.[4]

II. DEFINITION

"Hypertrophic cardiomyopathy (HCM, or HOCM while obstructive) is a situation where in the coronary heart turns into thickened without an apparent cause. The components of the coronary heart most generally affected are the ventricles and the interventricular septum. These outcomes in the heart being much less capable to pump blood efficiently and additionally may also cause electric conduction problems."[5,6,10]

III. HISTORY AND PHYSIOLOGY RELATED TO HCM

An autosomal dominant mendelian sample with a difference in expressivity or age-related penetrance is inherited by HCM. Offspring of an affected character have nearly a 50% chance of acquiring a mutation and having a disease. Alternatively, occasional occurrences in the proband may result in de novo mutations that are not detected in the parents.[7] Although at the beginning taken into consideration a disease in general of younger adults, hypertrophic cardiomyopathy is generally recognized in older patients.

Whiting et al. mentioned that 32% of sufferers imparting with HCM were >60 years of age. In a community hospital-primarily based totally series of sufferers with hypertrophic cardiomyopathy mentioned through Petrin and Tavel, 83% of sufferers were >50 years of age. Likewise, in a recent study from the Cleveland Clinic, hypertrophic cardiomyopathy has become more commonly recognized in patients over 65 years of age than in those who are less than 40 years of age.[8]

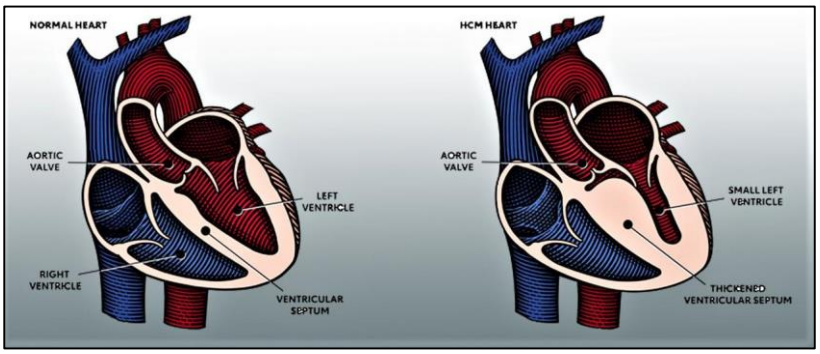

Figure 1: - Normal Heart & Hypertrophic Heart[9]

The maximum common imparting symptom of hypertrophic cardiomyopathy is dyspnea. Patients can also develop syncope, palpitations, angina, paroxysmal nocturnal dyspnea, orthopnea, dizziness, congestive coronary heart failure, and unexpected cardiac death. The latter represents the maximum devastating imparting symptom. Physical findings include a double apical impulse due to forceful left atrial contraction toward the extremely noncompliant left ventricle, normal s1, and split s2 s3 due to decompensated heart failure, and a significant "a wave" of pressure in jugular vein and laterally displaced carotid pulse, and double apical impulse.

A systolic ejection murmur is shows as follows:

- Faded intensity with rising preload (squatting) or after load (handgrip) and increased intensity with a lower in preload [Valsalva maneuver (a breathing method that may slow your heart when it's beating too fast), standing], and with any lower in after load (vasodilator administration).[10]

By 1961, in spite of the fact that it was nevertheless poorly understood, this disorder entity was recognized to be distinct from aortic stenosis and termed IHSS. As dimensional echocardiography was not really possible, the diagnosis was usually made during cardiac catheterization. Until, the aortic (semilunar) valve cannot be visualized, differentiating Hypertrophic cardiomyopathy from aortic stenosis persists challenging, regardless of invasive hemodynamics.[11]

IV. Signs and Symptoms of Hypertrophic Cardiomyopathy

Some sufferers with HCM develop shortness of breath and chest discomfort, in addition to fainting, dizziness, palpitations, light-headness with physical exertions or physical activity. Symptoms might also additionally start at any age, and frequently do now no longer seem until midlife (30s or 40s). Symptoms can expand at distinct rates, with long durations of stability, and frequently range from day-to-day; intense exercise limitation is, however, uncommon. Occasionally, sufferers can be not able to sleep in a flat position or can also additionally awaken due to short of breath.[12] "Triple ripple apical impulse" and "Spike and dome" pulse are other signs or symptoms that could be found in physical examination.[13]

V. Morphologic Patterns of HCM

In hypertrophic cardiomyopathy (HCM), the interventricular septum is usually hypertrophied to various degrees. Hypertrophy can be concentric, as seen at the apex or free wall of the LV, or concentric, can be seen on the mid-ventricular level, or it can even spread into the right ventricle.

As a result, various HCM phenotypes have been identified. We are discussing about three types of HCM listed below.[14]

1. Asymmetric (Septal) HCM
   - Dynamic LVOT Obstruction
   - Mitral Regurgitation
2. Apical HCM
3. Midventricular HCM

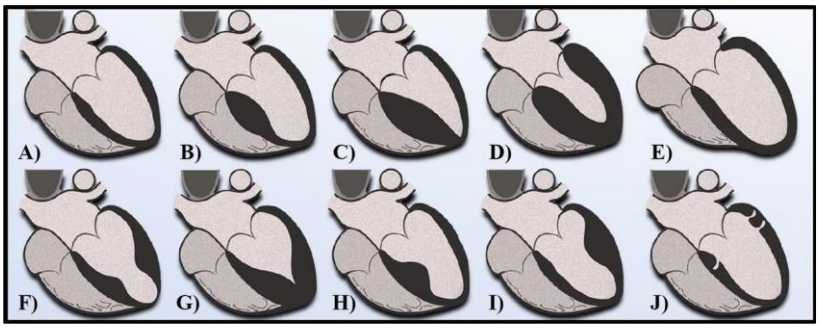

Figure 2: - HCM Phenotypes diagrams Showing: -
A) Normal or HCM-Phenotype-Negative Cardiac Morphology, B) Focal Basal Septum HCM, C) Diffuse Septum HCM,D) Concentric and Diffuse HCM, E) Burned-Out Phase HCM, F) Midventricular HCM, G) Apical HCM, H) Focal Mid-septum HCM,I) Free-Wall HCM, J) Crypts in Genotype-Positive and Phenotype-Negative HCM.

1. Asymmetric (Septal) Hypertrophic Cardiomyopathy

The asymmetric septal form is the most commonly seen morphologic variant of HCM and accounts for as much as 60%–70% of cases.[15] The ventricular septum is asymmetrically thickened in this HCM, and the anteroseptal myocardium is frequently affected. In the midventricular, subaortic, and apical areas, septal hypertrophy can be detected. In some sufferers, the hypertrophy could be focal and can include only one or left ventricular segments, most commonly including the anterolateral wall, posterior septum, basal anterior septum, and apex.[16] Septal hypertrophy of the anteroseptal myocardium causes the septum to take on a sigmoidal form, slendering or narrowing the LV cavity, and encroaching on the LVOT. This pattern is frequently linked to SAM of the mitral valve leaflet. When present, this combination generally outcomes in subaortic obstruction.[17] When present, this combination also results in subaortic obstruction in children. Septal hypertrophy is specified as a septal wall thickness of two standard deviations or more over the mean for sex, age, or body size.[18]

The combination of the structural changes, mitral valve abnormalities, and hyperdynamic systolic flow alongside the LVOT additionally causes posteriorly directed mitral valve regurgitation. However, asymmetric or uneven septal hypertrophy with a reversed S septum does not produce LVOT obstruction.[19] Echocardiography is the preliminary and most frequent imaging modality utilized for examination of LVOT gradients and obstructive anomalies, especially during physiologic provocation, due to its practical usefulness, availability, and high temporal resolution. However, velocity and peak gradients throughout the LVOT may be accurately calculated using phase contrast MR imaging.[20] The peak pressure gradient is derived with a simplified Bernoulli equation, being equal to four times the square of the peak systolic velocity via the LVOT in meters per second (4V2). The gradient is considered significant if more than 30 mm Hg. Electrocardiographically gated cardiac CT is not regularly carried out for this condition, because it does not provide information or data on fibrosis and the flow dynamics of HCM. However, cardiac CT gives excellent spatial resolution constitutes an alternative modality in sufferers in whom MR imaging is contraindicated or in sufferers with implanted pacemakers or defibrillators, in whom MR imaging is usually not executed because of susceptibility to artifacts.[21]

This type of cardiomyopathy might be associated with the following –

- Dynamic left ventricular outflow obstruction (in up to 30%) – gradient ≥30mmhg,
- Systolic anterior motion (SAM) of the anterior mitral leaflet,
- Myocardial fibrosis,
- Mitral leaflet abnormalities or mitral regurgitation (in up to 20%), and
- Left atrial enlargement[14,17]

1.1. Dynamic LVOT Obstruction

LVOT obstruction is found in 70% of HCM cases, however in one or two of them the obstruction may be demonstrated only after physiologic provocation like after the Valsalva maneuver. LVOT obstruction is defined or termed as a gradient greater than 30 mm Hg.[14,22] SAM is taken into consideration to arise predominantly through drag forces, even though a Venturi mechanism has also been invoked.[23]

1.2. Mitral Regurgitation

Because of a mismatch in posterior and anterior leaflet mobility and also the length, SAM of the leaflets of the bicuspid or mitral valve produces mitral regurgitation as well as subaortic obstruction.[24] Mitral regurgitation because of SAM is normally directed towards the posterior wall of a left atrium, forming a right angle with the turbulent LVOT flow, and occurs in a mid to late systole. This may be readily appreciated using transthoracic or transesophageal echocardiography and MR imaging. The degree or grade of a mitral regurgitation is proportionate to the LVOT gradient, and interventions that enhance the LVOT gradient will therefore reduce mitral regurgitation. Earlier mitral regurgitation or a direction of the jet of regurgitant other than posterior strongly indicates independent mitral valve disease, which can necessitate mitral valve surgery.[25]

2. Apical Hypertrophic Cardiomyopathy

This variant was first reported in patients of Japan in 1976 by Sakamoto and Suzuki[26], Yamaguchi et al.[27], and their associates. This Apical HCM is also expressed as Yamaguchi syndrome. It is characterized by the presence of a giant and negative T-waves on the electrocardiogram and a spade-like structure of the LV cavity, since most of the hypertrophy is confined to the apical region of the LV. In the Japanese community, apical HCM reports for about 15% of cases, where in whites, it reports for up to 2% of cases.[28] Apical hypertrophy cardiomyopathy is characterized as myocardial hypertrophy that predominantly includes the apex of left ventricle. Apical HCM was initially identified among Asians, however it is currently being diagnosed more often in the Western world.[29]

The reported rate of occurrence of apical hypertrophic cardiomyopathy varies in the literature, ranging from 25% of all sufferers with HCM in Japan to fewer than 2% of the sufferers or patients with HCM in Western countries. This type of patients also has a chest pain and stroke. Therefore, one of the aims of imaging is to distinguish apical hypertrophy from unstable apical thrombus and angina. The diagnosis of pure apical HCM can be made while LV thickening is predominantly confined to the apex, measuring 15 mm or more, with a ratio of the apical left ventricular wall thicknesses to basal left ventricular wall thicknesses of 1·3 to 1·5. The apical part of the RV is often concerned as well.[17]

Unlike common HCM, apical HCM –

- It indicates a predilection for middle-elderly or aged men,
- It is rarely associated with sudden or unexpected cardiac death,
- It is often complicated by hypertension, and
- It has a relatively good prognosis.[29]

Cardiac MRI is strongly suggested to diagnose and evaluate apical HCM. The characteristic “ace of spades” configuration of the LV cavity at end diastole, which was initially described on ventricular angiograms, is easily demonstrated at cardiac MR imaging or cardiac multidetector CT. Specific complications of apical HCM includes development of apical infarction with result antaneurysm formation, the do-known burned-out apex.[19] Apical HCM may also show typical electrocardiographic abnormalities in the form of a giant or large negative-T waves.[27] Because of its high spatial resolution, cMRI permits differentiation of apical HCM from LV noncompaction cardiomyopathy, which generally demonstrates deep intertrabecular recesses and prominent

trabeculae. Because of the lower spatial resolution of two-dimensional echocardiography, prominent trabeculae in the LV apex associated with LV noncompaction can potentially be misdiagnosed as the apical HCM.[29]

3. Midventricular Hypertrophic Cardiomyopathy

Isolated midventricular obstruction is a rare form of HCM characterized by LV hypertrophy predominantly localized in the midmyocardial segments. This gives a characteristic hourglass or dumbbell shape to the LV at cardiac CT and MR imaging.[19] Midsystolic muscular apposition of the interventricular septum with the free wall of the left ventricle might result in midcavity blockage. This is referred to as a peak midcavity gradient of greater than 30 mm Hg.[30] Midventricular HCM is frequently linked with an apical aneurysm induced by elevated systolic pressures in the apex due to midventricular obstruction. MR imaging as well as multidetector CT well demonstrate the characteristic "dumbbell" configuration of the left ventricular cavity with marked muscular midcavity systolic constriction.[31]

SAM and an LVOT gradient are generally absent in this form of HCM. The pressure overload in the apical LV chamber increases myocardial stress and decreases perfusion, leading to fibrosis and development of an apical aneurysm.[32] Apical aneurysm formation is rare in HCM as a whole (2%) but has a particular predilection for the apical and midventricular variants, with an occurrence of 18% and 28%, respectively.[33,34] In either case, an apical left ventricular aneurysm poses an elevated or increased in risk of adverse events inclusive of thrombosis, ventricular tachycardia, myocardial ischemia, and necrosis. Minami et al.[30] in their analysis of 490 sufferers of with HCM, concluded that midventricular obstruction is an independent factor of HCM-associated death because of combined lethal arrhythmias and sudden death events. At cardiac MR imaging, apical aneurysm is seen as a focal, dyskinetic, thin-walled bulge often showing delayed transmural enhancement because of scar formation. Thrombus, if present, appears as a non-enhancing filling defect inside this aneurysm cavity which can or cannot be mobile.[33]

VI. Diagnosis of Hypertrophic Cardiomyopathy

There are some diagnostic measures of the Hypertrophic Cardiomyopathy which listed below.

1. ECG (Electrocardiography)

25% of sufferers exhibit a LBBB (It is a condition in which there is a blockage or delay along the pathway that electrical impulses travel to make your heart beat.) or a left anterior hemiblock. The configuration of the hyper voltage and giant or massive negative T waves is distinctive and for HNCM, and pseudo infarct Q waves are distinctive for HOCM. Hyperacute negative T waves are feature for the apical form of HCM.[27] It is likewise recommended when there may be a progressive change (worsening) of signs or symptoms in already recognized HCM patients. It has been recognized that carriers of the genetic mutation of HCM have ECG lesions prior to the occurrence of LV hypertrophy.[35] Peripheral low voltage indicates a cardiac amyloidosis or storage disorder and need to initiate or proceed with a myocardial biopsy.[36] ECG/EKG can assist to determine if there are abnormalities in electric conduction or thickening, or harm or damage to the coronary heart muscle. The ECG pattern is abnormal in 90% of patients or sufferers with HCM. Of note, a small minority of HCM sufferers may have a regular ECG.[37]

2. Echocardiography

A large spectrum of morphological findings, ranging from the isolated thickness of an individual's myocardial segment that differs by only a few mm from the typical LV wall thickness (<12 mm) to significant hypertrophy and wall thickness up to 60 mm. Sudden cardiac death is associated to a maximum wall thickness of more than 30 mm.[38]

Echo (Echocardiography) is the imaging modality generally used to measure the LV wall thickness. It is also used for measuring the obstruction of LV outflow tract which can be defined by LV outflow tracts pressure gradient which is greater or equal to the 50 mm Hg.[39] The mechanism and degree of the LVOTO are measured using Doppler and 2D echocardiography. If non-invasive images do not show the clarity, the transthoracic echo (TTE) or the transesophageal echo (TTE) can be considered for the selected patients. Echo is suggested in the initial evaluation of all sufferers suspected with HCM, as an aspect of family screening, periodic (12–18 months) screening of offspring of HCM sufferers, and repeat screening for modifications in clinical status of HCM sufferers. Contrast TTE is used as suggestion for evaluation of LVOT obstruction while the intensity of wall thickness of left ventricle or apical HCM is not known, also while the other cardiac imaging modalities like MRI is unavailable.[40]

3. Cardiac Magnetic Resonance Imaging

Several imaging modalities of cMRI offers precise diagnostic records about cardiac morphology and ventricular characteristic in addition to characterize myocardial tissue in HCM patients.[41] Imaging takes a vital position in establishing both prognosis and diagnosis in HCM. While family history, physical examination, symptoms, and ECG can all be suggestive factors, none are essential or enough to diagnose an affected person with HCM. With the appearance of LVH (LV wall thickness >15 mm in diastole) in the non-presence of any other cause, there is a need for diagnosis.[42] Cardiac MRI is beneficial in the differential analysis of HCM to signify particular diagnoses based on the magnetic properties, degree and distribution of interstitial expansion. Reduced posterolateral LGE and non-contrast T1 signals indicates Anderson-Fabry disease.[43] cMRI images enhance the evaluation of left ventricular hypertrophy, specifically in the anterolateral LV free wall.[44] Although late gadolinium enhancement MRI has been connected to all-cause mortality and death from failure of the heart, the evidence for its risk value in predicting sudden death in patients with HCM is inconclusive.[45]

VII. Treatment of Hypertrophic Cardiomyopathy

Following are some of the treatments in Hypertrophic Cardiomyopathy.

1. Asymptomatic Patients

A number of individuals with hypertrophic cardiomyopathy do not show any signs or symptoms and could live a normal life, but they need to keep away from mainly strenuous activities and competitive athletics. Asymptomatic patients must be screened for risk and life-threatening factors to prevent from the sudden or unexpected cardiac death. In some individuals with resting or inducible outflow obstructions, conditions that will possess dehydration or vasodilation (which include the usage of vasodilators or diuretic blood pressure medications) must be avoided. Septal reduction therapy not suggested in asymptomatic patients.[18]

2. Medications

The main purpose of medications in symptomatic HCM patients is to relieve symptoms of dyspnea, chest discomfort and palpitations, which may also show the pathophysiological mechanism with LVOTO, myocardial oxygen with decreased supply, impaired relaxation of LV diastole, and compliance. β-blockers can be the mainstay of medication as well as agents in first-line due to their effects like negative inotropic effect. Because of the decreased heart rate, the diastolic filling period can be extended, allowing for greater functional inactivation of the myocardial contractile proteins and hence an improved diastolic filling. The patients in which symptoms non-responsive and patients which cannot tolerate the β-blocker, in those the calcium channel blocker can provide effective symptomatic relief. Verapamil is also the mostly studied agent for the medication.

To prevent the myocardial ischemia and to improve the measures of diastolic performance, the Diltiazem can be used. Both diltiazem and verapamil must be used carefully in patients with severe outflow tract obstruction, low systemic blood pressure, and increased pulmonary artery wedge pressure, because a decreased blood pressure with treatment may also precipitate pulmonary edema and cause an increase in outflow obstruction. Administration of some β-blockers with either diltiazem or verapamil needs to be used with additional caution because of the potential for atrioventricular block at high grade. In the patients having physiology of obstruction, the use of dihydropyridine class calcium channel blocker like nifedipine must be avoided, because they have a vasodilatory effect which may also aggravate outflow obstruction. For the symptomatic relief to the patients having pulmonary congestion, diuretics can be effective. Diuretics should be used very carefully in the patients having outflow tract obstruction.[18]

3. Surgical Septal Myectomy

For the septal reduction in cardiomyopathy, the surgical procedure of septal myectomy has the gold standard. To decrease the LVOT gradient, a small section of the thickened muscle is removed from the heart's basal ventricular septum. This procedure of surgery is also called as "Morrow's surgery."[46] Long-time period research with >50 years of follow-up have proven that surgical myectomy reliably reverses coronary heart failure symptoms, permanently abolishing obstruction (90% of cases), restoring normal LV filling pressure and decreasing or abolishing mitral regurgitation.[47,48] For the septal myectomy the operative mortality ranges from the less than 1% at the practiced centers to nearly 3%–4% when related to mitral valve repair. The actual complications before the operation are atrioventricular block, interventricular communication and also the aortic valve insufficiency.[48]

The echocardiography of intraoperative transesophageal is assisted in concomitant structural abnormality correction and septal resection, with a decrease in complication rates and improving surgical results.[18,22] The patients having left atrium <46 mm and not having atrial fibrillation and under the age of 50 years old shows the better surgical results.[48] It is observed that the female patients having HCM are identified at their old age, shows more obstruction than in men, shows the more symptoms and also have the less survival. Also, the female which undergoing a septal myectomy also shows systolic pressure at right ventricle and average to severe mitral regurgitation more than the male patients. (Described by Meghji at el.) Female patients who do not react to clinical treatments and have been identified with obstructive HCM should have surgical treatment as soon as possible.[49]

4. Implantable Pacemaker or Implantable Cardioverter-Defibrillator (ICD)

The devices that are commonly implanted and used for primary prevention as well as secondary prevention like for patient sudden death, are single-chamber or dual-chamber ICD's. The guideline-based recommendations in the current situation are single-chamber cardioverter-defibrillators for patients having hypertrophic cardiomyopathy who are at high risk like SCD. Dual-chamber devices are generally suggested for patients with sinus bradycardia or pre-existing paroxysmal atrial fibrillation with fast rates. Dual chamber pacing devices may benefit patients with an increase in resting outflow gradient (greater than 50 mmHg) who are older and have symptoms of heart failure. These devices can have a beneficial effect on HCM patients and can potentially decrease gradient and heart failure symptoms.[50]

5. Alcohol Septal Ablation

This ablation was first carried out effectively in 1994 by Sigwart on three patients suffering from HOCM, but they had not shown any good progress with clinical treatment. The improvement was seen in symptoms of these patients after the alcoholic septal ablation and LVOT gradient was also decreased.[51] An absolute alcohol injection of 1·5–2·5 mL is administered via cardiac catheterization, with the main septal branch at the anterior and descending arteries occluded. This causes the septal infarction with septal reduction and scarring formation.[52] In the current situation, some questions arise like whether the scar formed on the septum can make a contribution as an arrhythmogenic focus at risk for sudden cardiac death.[53] This technique is the alternative for the surgical myectomy in the patients who have advanced age, co-occurring conditions, or the patients who oppose a thoracotomy or open chest surgery. This treatment is avoided if patient is having septal thickness >30 mm and <16 mm.[18,22] An alcohol septal ablation shows a controlled heart attack while the procedure is conducted properly, in which the section of the interventricular septum that comprises the LVOT becomes infarcted and eventually contracts into the scar.[54]

6. Cardiac Transplantation

This is the last and only treatment available for end-stage or last-stage heart failure patients that can show a better result in patient recovery. But there are some complications with the transplantation. To have a successful transplant without any complications, the heart transplant should be performed in a manner before the occurrence of any symptoms like kidney malfunction, pulmonary vessel hypertension, or thromboembolism. It is seen from recent studies that approximately 94 percent of patients with HCM after heart transplantation have a nearly seven-year rate of survival.[55]

VIII. Conclusion

HCM is a condition in which the left ventricular hypertrophy produces either obstruction or inadequate blood flow. Cardiac MRI helps get useful clinical information for determination, screening, and accurate diagnosis of HCM. The differentiation in phenotypes can be seen by using cMRI. When using echocardiography, it shows unsatisfactory or suboptimal results. In the evaluation of abnormal papillary muscles, right ventricle involvement, and a variant like the apical variant, it does not show any satisfactory results in diagnosis. The detection and quantification of scar and fibrosis are unique capabilities of cMRI.

Treatments like alcohol septal ablation can show very useful results in patients with HOCM without undergoing any surgical procedure. Surgical myectomy is one of the other better treatments useful to get relief from the obstruction and to prevent SCD.

Also, the mitral clip and ICD can show useful results in the management of HCM. However, depending on the stage of HCM, the treatments vary. At the end-stage of HCM, the last way to save the life of a patient is through heart transplantation. A successful transplant can increase a patient's life expectancy by nearly seven years.

Table 1: - Clinical Finding in Phenotypes of HCM

HCM Phenotypes	Classic Description	LV Ejection Fraction	LVOT Obstruction	Symptoms
Asymmetric HCM	Most common HCM (60%–70%), SAM, mitral regurgitation, increased gradient across LVOT	Normal to supranormal	20%–30% of cases	Variable from mild to moderate
Apical HCM	Spade-like configuration of LV, burned out apex with apical aneurysm formation	Normal to supranormal	Not seen	Variable; may mimic acute coronary syndrome
Mid-ventricular HCM	Rare, hourglass or dumbbell shape of LV, apical aneurysm formation, apical thrombus can be seen	Normal to supranormal	Obstruction and increased gradient at Mid-ventricular level	Variable from mild to severe
Burned-out HCM	LV thinning and dilatation with hypokinesia and depressed ejection fraction	Decreased	Decreased	Progressive heart failure

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