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Multidisciplinary management of patients with hypertrophic cardiomyopathy

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The article presents a review of literary and scientific sources from the MEDLINE database on PubMed, Web of Science, Scopus, and Google Scholar platforms, focusing on hypertrophic cardiomyopathy and its treatment aspects. In accordance with the recommendations of leading experts, a modern definition of hypertrophic cardiomyopathy is shown with a description of the classification of structural and functional cardiac abnormalities, associated symptoms, and generalized risk factors for sudden cardiac death in patients. A ranking of patients related to the left ventricular outflow tract obstruction and clinical manifestations is submitted. Based on international guidelines, the main principles for treating patients are given, depending on hemodynamic and clinical characteristics, including pharmacotherapy and invasive methods. Pathophysiological reasoning and clinical justification for prescribing drugs to correct complications are provided. The use of septal myectomy and alcohol septal ablation to correct morphological cardiac abnormalities and improve the clinical status of patients is outlined in a historical context. The results of one's own experience in surgical treatment of patients with hypertrophic cardiomyopathy are presented.

Key words: hypertrophic cardiomyopathy, clinical phenotypes, treatment targets, pharmacotherapy, septal myectomy, alcohol septal ablation

Cardiomyopathies (CMP) are a group of diseases characterized by structural and functional cardiac abnormalities that manifest clinically with predominant heart failure syndromes and life-threatening arrhythmias. The 2023 European Society of Cardiology Guidelines on the management of CMP (hereafter, 2023 Guidelines) classify CMP into the following categories: hypertrophic cardiomyopathy (HCM), dilated CMP, restrictive CMP, arrhythmogenic right ventricular CMP; non-dilated left ventricular CMP [1].

Among the various nosological forms, HCM draws particular attention. Before receiving its current designation, HCM underwent a lengthy classification process with numerous nomenclatures, particularly during its early studies. The most significant terms included: aortic subvalvular stenosis (R. Brock), idiopathic hypertrophic subaortic stenosis (E. Braunwald et al.), and hypertrophic obstructive CMP (J. Cohen et al). [2–4]. Currently, HCM classification is based on the localization of myocardial hypertrophy, which results in hemodynamic disturbances. Obstructive HCM is characterized by asymmetric hypertrophy of the interventricular septum protruding into the left ventricular outflow tract (LVOT), causing narrowing of the LVOT and impaired blood ejection into the aorta. Asymmetric

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hypertrophy of the interventricular septum located below the LVOT is classified as non-obstructive HCM. Another possible form is concentric myocardial hypertrophy, which involves the free wall of the left ventricle and the apex of the heart, resulting in apical obstruction.

Another characteristic anatomical feature of hypertrophic cardiomyopathy is changes in the anterior leaflet of the mitral valve. In HCM, it is displaced anteriorly toward the interventricular septum, thickened, and obstructs the LVOT, creating an additional barrier to blood outflow into the aorta. This condition is known as systolic anterior motion of the mitral valve.

The clinical significance of HCM is highlighted by in-depth studies at multiple levels. Advanced high-resolution cardiac imaging has enabled the development of reliable diagnostic criteria for anatomical and functional cardiac changes in HCM patients, allowing for the tracking of their dynamics during disease progression, correlations with symptoms, and responses to various treatment regimens. Significant progress in understanding HCM – specifically in determining etiological origins, myocardial pathophysiology, clinical manifestations, and prognostic markers for life-threatening complications has been achieved through studies of cardiac molecular architecture, elucidating the role of proteins in cardiomyocytes responsible for biomechanical, signaling, and translational functions. Notably, genes encoding contractile proteins in cardiomyocyte components play a critical role. A new stage in understanding HCM pathogenesis emphasizes the study of gene mechanisms in regulating sarcomeric protein stability in cardiomyocytes. Specific gene mutations have been identified as triggers of hypokinetic myocardium and proarrhythmic activity in patients with HCM [5].

Despite the availability of substantial scientific evidence and advanced diagnostic methods providing multifactorial informative criteria, the etiology of certain clinical scenarios with potentially fatal outcomes, such as myocardial dysfunction and sudden cardiac death (SCD) in HCM patients, remains incompletely understood. Further refinement is needed for existing pharmacotherapeutic and invasive treatment strategies, taking into account patients' hemodynamic, clinical, and genetic profiles.

Clinical and instrumental features of hypertrophic cardiomyopathy

The fundamental principles of diagnosis and treatment HCM were outlined in 2011 in a guideline developed by American experts [6], with subsequent additions by specialists from the European Society of Cardiology in 2014 [7]. Extended recommendations for the management of patients with cardiomyopathy were published by leading cardiologists in 2023 [1]. This document aims to provide a targeted update of the 2014 recommendations and to highlight new aspects of diagnostic and therapeutic strategies for HCM in both adults and children.

Prevalence. HCM is a heterogeneous cardiac disorder observed across all age groups, from childhood to old age. Data on HCM prevalence vary across sources due to several factors. First, the condition is often asymptomatic for some time and is detected incidentally during screening, such as in family studies. Clinically, only 10 % of HCM cases are identified, and only 6 % of these cases are symptomatic. Epidemiological studies based on echocardiographic criteria report a prevalence of HCM in the adult population ranging from 0.16 % to 0.23 %, with a mean of 0.20 % (1 in 500) [8]. Echocardiography provides limited information on morphological characteristics compared to more advanced imaging techniques. According to computed tomography (CT) data, the prevalence of HCM is higher than previously reported, approximately 1 in 200 (0.5 %) [9].

Clinical manifestations. HCM may be suspected during outpatient evaluation in the presence of characteristic complaints, although some patients may have an asymptomatic period in the early stages. Symptomatic HCM, due to intramural coronary vessel compression and reduced myocardial perfusion, may present with precordial and retrosternal chest pain at rest, resembling angina. A second group of symptoms is associated with LVOT obstruction, reduced left ventricle (LV) cavity size, and low systolic output, manifesting as dizziness (often accompanied by chest pain), palpitations (especially after exertion due to exacerbation of obstruction), reduced afterload due to peripheral vasodilation, and syncope with transient loss of consciousness. The most common causes of syncope are paroxysms of ventricular tachycardia (e.g., Torsade de pointes) or asystole, leading to decreased cardiac output, hypotension, and impaired cerebral perfusion.

Symptoms of LV heart failure in HCM patients include weakness, dyspnea, exercise-induced tachycardia, orthopnea, paroxysmal nocturnal dyspnea, and resistance to pharmacotherapy. Heart failure occurs in 90 % of obstructive HCM cases, even with preserved LV ejection fraction, and is explained by diastolic dysfunction. In some patients, heart failure progresses to end-stage disease with reduced ejection fraction.

Physical examination. In HCM patients without obstruction, there are no specific cardiovascular findings. In those with obstruction, most patients have palpable precordial thrill and a coarse systolic murmur auscultated at the third or fourth left intercostal space, caused by turbulent flow through the narrowed

outflow tract. Murmur intensity depends on the degree of obstruction. Mitral regurgitation produces a soft pansystolic murmur at the apex, radiating to the axilla.

Electrocardiographic findings. ECG abnormalities are observed in 95 % of HCM patients, including repolarization abnormalities (ST-segment depression and T-wave inversion in lead I and left precordial leads), as well as intraventricular conduction disturbances. Signs of septal hypertrophy include pathological Q waves in leads II–III and V4–V6. Deep «giant» T waves in V3–V5 indicate apical hypertrophy (apical HCM). Later stages may show P-wave changes, such as «P-mitrale» or «P-pulmonale».

Holter monitoring is recommended to detect arrhythmias, which are adverse prognostic indicators in HCM, due to the presence of an arrhythmogenic substrate characterized by myocardial disarray, interstitial collagen deposition, and replacement fibrosis following myocyte death from coronary microvascular dysfunction and ischemia. Atrial fibrillation occurs in 20 % of patients, associated with LVOT obstruction, increased end-diastolic pressure, and atrial hypertrophy, significantly increasing the risk of peripheral thrombosis and ischemic stroke. Ventricular tachycardia is a fatal complication manifesting as SCD defined as death from an underlying cardiac condition within one hour of symptom onset, or unexpected death in an apparently healthy individual within 12–24 hours [10].

A significant proportion of SCD cases are due to cardiac disease, including HCM and genetically determined arrhythmogenic disorders (channelopathies), such as Brugada syndrome, long QT syndrome (Jervell – Lange – Nielsen syndrome), and Romano – Ward syndrome [11]. HCM is a major cause of SCD and ranks among the leading factors of mortality. SCD may be the first clinical manifestation of the disease within a family. There is an established association between anatomical cardiac changes and SCD incidence; patients with LV wall thickness ≥ 30 mm are at particularly high risk [12].

SCD is of particular concern in individuals under 35 years [13]. In children, the risk of SCD increases after age 6, peaking annually between ages 9 and 15 [14]. Literature data indicate that HCM is a primary trigger of SCD in young people due to strenuous physical activity or sports participation. An analysis of 1866 cases of sudden death in athletes in the USA (1980–2006) showed a mean age of 19±6 years for those with cardiac causes [15]. SCD most frequently occurred during or immediately after exercise. Approximately 70 % of SCD cases occurred in athletes participating in competitive team sports, such as basketball and football. The most common structural cardiac pathology identified at autopsy as the cause of death was HCM.

Risk factors for SCD in HCM include family history of SCD, ventricular tachycardia, recurrent syncope, resuscitation after cardiac arrest, LVOT gradient > 30 mm Hg, LV apical aneurysm, limited myocardial perfusion reserve, hypotension during exercise, LV systolic dysfunction, end-stage heart failure, and late gadolinium enhancement on cardiac magnetic resonance imaging (CRM) [1, 16].

The annual mortality rate from HCM in adults is 0.5–1.0 %. Leading causes of death in young individuals are SCD, whereas in older adults, heart failure and stroke secondary to atrial fibrillation predominate [16]. In obstructive HCM, progression of heart failure and mortality risk are fourfold higher than in non-obstructive HCM [17].

Central to the diagnosis, treatment and monitoring of HCM from initial step to follow-up of patients are main instrumental imaging tools such as Echocardiography and cardiovascular magnetic resonance (CMR) with non-invasive nature and widespread availability.

Echocardiography provides relevant information on left ventricular wall thickness, LV anatomy and function, left atrial enlargement, associated abnormalities of the mitral valve and left ventricular outflow tract, of obstruction, the presence of latent and dynamic obstruction, pulmonary hypertension, presence and degree of systolic anterior motion and mitral regurgitation, elevated LV filling pressures, diastolic function [1, 6].

CMR can provide important detailed information on cardiac morphology, ventricular function and myocardial tissue characteristics. CMR is superior in the detection of LV apical and anterolateral hypertrophy, aneurysms, and thrombi [1, 6]. By using the intrinsic magnetic properties of different tissues and the distribution of gadolinium-based contrast agents, CMR can be used to detect expansion of the myocardial interstitium caused by fibrosis. The distribution and severity of interstitial expansion etiological correlates with specific diagnoses and suggest Anderson – Fabry disease, cardiac amyloidosis which characterized by a highly specific pattern of myocardial and blood-pool gadolinium kinetics.

Based on the results of instrumental imaging examination of patients have been determined specific cardiac morphological and functional features of HCM.

The 2023 guidelines define HCM taking into account previous publications [6], according to which HCM is characterized by cardiac hypertrophy, particularly of the LV (wall thickness ≥ 15 mm), in the absence of conditions causing pressure or volume overload (e.g., hypertension, ischemic heart disease, valvular lesions) that could explain the thickening. In the majority of clinically confirmed HCM cases, LV wall

thickness is 15 mm or more (mean 21 mm), although massive hypertrophy (30–50 mm) can also occur. Mild LV wall thickening (13–14 mm) may be observed in individuals engaged in significant physical activity («athlete's heart»). A broader definition of HCM includes an increase in LV mass; symmetric LV wall thickening > 15 mm (or more than two standard deviations above normal, adjusted for age, sex, and body size); and asymmetric hypertrophy (septal/posterior wall thickness ratio > 1.3, or > 1.5 in hypertensive patients).

Other pathological conditions in which cardiac hypertrophy is part of a multisystem phenotype have also been described. These phenocopies include syndromic disorders (Anderson – Fabry disease, primary amyloidosis, etc.), metabolic and multisystemic diseases.

The dominant anatomical feature of HCM – myocardial hypertrophy, most commonly of the interventricular septum – combined with an elongated anterior mitral leaflet, produces a narrowing that impedes LV outflow. This leads to left ventricular outflow tract (LVOT) obstruction, increasing the pressure gradient between the ventricular cavity and the site of obstruction (gradient > 30 mm Hg). The degree of obstruction is individual, may vary in each patient, and can lead to secondary hypertrophy (with the septum always thicker than the posterior wall).

Another manifestation of hypertrophic HCM is diastolic dysfunction, which arises from pathophysiological processes associated with morphological changes, including ischemia, hypoxia, impaired energy metabolism, alterations in intracellular calcium, impaired active relaxation, and increased myocardial stiffness. A significant number of patients with LVOT obstruction develop secondary mitral regurgitation. Diastolic dysfunction leads to increased LV end-diastolic pressure, elevated pulmonary capillary wedge pressure, and pulmonary congestion. In this context, the LV cavity is often small, while the left atrium is frequently hypertrophied and dilated. Cardiac systolic function parameters (end-systolic volume, ejection fraction) are usually normal or even «supernormal» in early disease stages; systolic function is enhanced due to LV hyperdynamic activity, with ejection fraction reaching 80–90 %. However, in the presence of significant myocardial hypertrophy, systolic dysfunction may develop over time, as evidenced by clinical signs and reduced LV ejection fraction on echocardiography.

Thus, HCM represents a significant clinical problem that requires a comprehensive diagnostic process utilizing modern, innovative techniques and genetic analysis to differentiate clinical variants, identify phenocopies, and select optimal therapeutic strategies, including surgical interventions.

Therapeutic management of patients with hypertrophic cardiomyopathy

The treatment strategy for patients with HCM is aimed at correcting pathophysiological processes – such as elevated LVOT gradient, impaired myocardial relaxation and stiffness, diastolic dysfunction, and increased pulmonary artery pressure – caused by anatomical abnormalities of the heart (LV hypertrophy, interventricular septal hypertrophy, LVOT obstruction, secondary mitral regurgitation), which manifest clinically as arrhythmias, angina, heart failure, and SCD. Considering multiple pathological markers, various therapeutic strategies targeting specific disease mechanisms have been proposed, summarized in the 2023 guidelines [1]. The main medical interventions for HCM include pharmacotherapy, invasive procedures, implantation of mechanical circulatory support devices, and heart transplantation.

Pharmacotherapy. First-line medications for HCM treatment are negative inotropic agents without vasodilatory effects – primarily beta-blockers (metoprolol, bisoprolol) [18]. Non-dihydropyridine calcium channel blockers (verapamil, diltiazem) also exert negative inotropic effects, alleviate LVOT obstruction, improve LV diastolic filling, and reduce symptoms [19]. Conservative therapy additionally includes medications indicated in specific clinical scenarios requiring restoration of sinus rhythm and heart rate control. According to the 2023 Guidelines these drugs, in addition to their antiarrhythmic properties, also positively influence hemodynamic abnormalities and ventricular remodeling processes specific to HCM. For pharmacological cardioversion, disopyramide – a negative inotropic agent is recommended. It reduces mitral regurgitation, decreases elevated LVOT gradient, limits LV contractility, and prevents QT interval prolongation without significant adverse effects [20]. Disopyramide can be safely administered in outpatients as monotherapy or in combination with beta-blockers or calcium channel blockers [21].

Advances in understanding the pathogenic cascade of HCM from molecular studies have stimulated the development of a novel targeted therapy capable of potentially modifying the disease by addressing a key component – myocardial hypertrophy. In 2022, the U.S. Food and Drug Administration approved mavacamten (MYK-461), the first-in-class cardiac myosin inhibitor, for the treatment of symptomatic obstructive HCM in adults, aimed at improving cardiac function and alleviating symptoms [22]. Mavacamten reduces myocardial contractility by inhibiting adenosine triphosphatase activity of cardiac myosin heavy chains [23]. Animal studies have demonstrated that chronic administration of MYK-461 inhibits the development of ventricular hypertrophy by downregulating profibrotic and hyper-

trophic pathogenic myosin gene variants. In the first randomized, placebo-controlled trial in HCM patients, mavacamten reduced LVOT gradient (< 30 mm Hg) and improved heart failure functional class (from II–III to I) [24]. J. Chase Cole et al. analyzed publications on mavacamten in HCM patients from active clinical trials between January 2015 and March 2023 [25]. The authors concluded that mavacamten is effective for persistent obstructive HCM and may reduce the need for invasive interventions. Its use is associated with improved exercise capacity and decreased frequency of septal reduction procedures [26]. Ongoing studies continue to evaluate mavacamten and a newer cardiac myosin inhibitor, aficamten (CK-27), which has a shorter half-life [27].

Invasive methods. Despite advances in conservative therapy and targeted approaches to specific pathogenic genes, a subgroup of HCM patients remains with persistent severe symptoms, progressive disease, and elevated SCD risk, unresponsive to pharmacotherapy, necessitating alternative therapeutic strategies. Indications for non-pharmacological therapy include significant LVOT obstruction, severe dyspnea, chest pain, presyncope or syncope, and refractoriness to maximal medical treatment.

Implantable cardioverter-defibrillator (ICD). Along with other adverse morphological and clinical characteristics, arrhythmias serve as a prognostic marker for SCD in HCM and are part of the risk factors guiding ICD implantation. According to the 2023 guidelines, ICD implantation may be considered in HCM patients with a sinus rhythm, a resting or provoked LVOT gradient of ≥ 50 mm Hg, and resistance to pharmacotherapy [1]. Updated 2024 recommendations emphasize that patients with maximal LV wall thickness ≥ 30 mm have elevated SCD risk and may benefit from ICD implantation [28].

Septal reduction therapy. Septal reduction in HCM includes surgical septal myectomy and alcohol septal ablation. Surgical septal myectomy, first performed by A.G. Morrow in 1961, involves resection of hypertrophied basal interventricular septal tissue, enlarging the LVOT, relieving obstruction, and improving clinical outcomes [29]. Subsequent refinements have improved anesthesia, myocardial protection, and surgical techniques, with indications including persistent symptoms refractory to pharmacotherapy, resting LVOT gradient ≥ 50 mm Hg, and NYHA class III–IV heart failure [30]. In a 2022 analytical review, B.J. Maron et al. highlighted that, based on 60 years of experience, septal reduction reliably alleviates heart failure by eliminating mechanical LVOT obstruction and, if needed, correcting mitral regurgitation, resulting in normalized LV pressure, restored systolic function, symptom reduction in > 90 % of patients, and return to normal daily activities [31].

Alcohol septal ablation, first performed by U. Sigwart 33 years after the first myectomy and reported in 1995, involves selective destruction of hypertrophied septal tissue via 96 % ethanol delivered through a balloon catheter, inducing a localized iatrogenic myocardial infarction, temporary endovascular occlusion of the septal coronary branch, and consequent reduction of intraventricular pressure [32].

Mechanical circulatory support. Mechanical heart devices («artificial heart») are used in patients with end-stage heart failure to maintain circulation until a suitable donor heart becomes available for transplantation.

Heart transplantation. For patients with HCM, heart transplantation is considered a life-saving option in terminal heart failure when recurrent potentially lethal arrhythmias occur, and pharmacotherapy, ICD implantation, and surgical interventions fail.

In summary, therapeutic practice in HCM has accumulated significant experience. treatment planning should focus on morphological, hemodynamic, and clinical targets, which determine the optimal medical and interventional strategy *(Table)*.

Differential treatment of patients with hypertrophic cardiomyopathy

Pharmacotherapy. The choice of treatment regimen for patients with HCM depends on specific anatomical myocardial changes, which sequentially determine the pathophysiological processes leading to symptom manifestation. In clinical practice, it is advisable to stratify patients with HCM related to the presence or absence of LVOT pressure gradient. In according with ICD-10 HCM is classified as Obstructive HCM (ICD-10 code I42.1) and Non-Obstructive HCM (ICD-10 code I42.2).

Clinical management should also consider the presence or absence of symptoms. Following patient evaluation, individuals with HCM can be classified into the following categories:o asymptomatic non-obstructive HCM, symptomatic non-obstructive HCM, asymptomatic obstructive HCM, and symptomatic obstructive HCM. The goals and strategies of treatment are dynamic and require continuous adjustment during follow-up.

The principles of HCM management are outlined in the 2011 and 2014 guidelines, with additional recommendations included in the 2023 update [1, 6, 7].

Asymptomatic non-obstructive HCM. A proportion of patients with HCM show no signs of LVOT obstruction or symptoms and may have a relatively normal life expectancy. For this group, there is no proven benefit of beta-blockers or calcium channel blockers; thus, pharmacologic or invasive therapy is not recommended. Continuous monitoring is essential to

Table

Morphological, hemodynamic, and clinical targets of therapy in patients with hypertrophic cardiomyopathy

Treatment targets	Treatment goals	Treatment strategy
High LVOT gradient (30–50 mm Hg)	Reduction of left ventricular end-diastolic pressure Improvement of left ventricular filling Reduction or elimination of symptoms	Pharmacotherapy with negative inotropic effect drugs: • Non-vasodilating beta-blockers (metoprolol, bisoprolol) • Non-dihydropyridine calcium channel blockers (verapamil, diltiazem)
High LVOT gradient (≥ 50 mm Hg) Heart failure NYHA functional class III or IV Refractory to maximal pharmacotherapy	Reduction of LVOT pressure gradient Improvement of left ventricular filling Reduction or elimination of symptoms	Invasive therapy: • Septal myectomy • Alcohol septal ablation
Left ventricular hypertrophy with hypercontractility	Reduction of excessive left ventricular contractility	Mavacamten stimulates regression of left ventricular hypertrophy, reduces LVOT obstruction, improves heart failure functional class, and enhances quality of life
Left ventricular hypertrophy with reduced contractility	Regression of left ventricular hypertrophy	Pharmacotherapy: • ACE inhibitors • Angiotensin II receptor antagonists
Angina	Reduction of symptom manifestations	Pharmacotherapy: • Beta-blockers • Non-dihydropyridine calcium channel blockers • In the absence of LVOT obstruction, cautious use of oral nitrates may be considered • Ranolazine reduces symptom manifestations in patients without LVOT obstruction
Arrhythmias (ventricular tachycardia)	Heart rate control	Pharmacotherapy: • Beta-blockers • Non-dihydropyridine calcium channel blockers • In case of intolerance, sodium channel blocker disopyramide is prescribed
Arrhythmias (atrial fibrillation)	Control of rhythm and heart rate Prevention of thromboembolic complications	Pharmacotherapy: • Beta-blockers and non-dihydropyridine calcium channel blockers • Consider amiodarone, disopyramide, sotalol if beta-blockers are insufficient to prevent atrial fibrillation • Anticoagulant therapy
Presence of SCD risk factors	Reduction of mortality	Pharmacotherapy: • Beta-blockers and non-dihydropyridine calcium channel blockers • ICD for patients tolerant to these drugs
Heart failure with preserved LV ejection fraction (diastolic dysfunction)	Reduction of left ventricular end-diastolic pressure Improvement of left ventricular filling Reduction or elimination of symptoms	Pharmacotherapy: • Beta-blockers • Non-dihydropyridine calcium channel blockers • Disopyramide • Cibenzoline • Diuretics if needed
Heart failure with reduced LV ejection fraction (systolic dysfunction)	Reduction of symptoms	Pharmacotherapy: • ACE inhibitors • Angiotensin II receptor blockers • Mineralocorticoid receptor antagonists • Sodium-glucose co-transporter 2 inhibitors • Neprilysin inhibitors

Table. Continued

Heart failure refractory to pharmacotherapy NYHA functional class III or IV Recurrent life-threatening arrhythmias	Preservation of life	Mechanical support devices implantation Heart transplantation

The table contains data that have been adapted by the authors in accordance with the recommendations published in international guidelines related to patients management with HCM [1, 6, 7, 12, 28], with ventricular arrhythmias and SCD [10], acute and chronic heart failure [40], results of a randomised, double-blind, placebo-controlled, EXPLORER-HCM study [41]. LVOT – left ventricular outflow tract; ACE – angiotensin-converting enzyme; SCD – sudden cardiac death; ICD – implantable cardioverter-defibrillator; LV – left ventricle.

detecte symptom onset or progression. Clinical course is known to worsen in the presence of hypertension, obesity, type 2 diabetes mellitus, and other comorbidities. Therefore, primary management goals include treatment of comorbid conditions [6]. Patients with obesity are advised to maintain a healthy lifestyle with a focus on weight reduction. In hypertensive patients, beta-blockers and calcium channel blockers are preferred for blood pressure control.

Symptomatic non-obstructive HCM. The overall treatment goal is to reduce end-diastolic pressure, alleviate symptoms, and improve cardiac functional capacity. All patients with HCM should undergo SCD risk stratification, regardless of hemodynamic or clinical characteristics, to allow timely implementation of preventive measures. Symptomatic treatment aims to reduce angina and dyspnea. For prolonged angina episodes, beta-blockers or non-dihydropyridine calcium channel blockers are prescribed [1, 6]. In the absence of LVOT obstruction, cautious use of oral nitrates may be considered [1]. Additionally, ranolazine may be used in non-obstructive HCM to alleviate symptoms.

Management of symptomatic patients without LVOT obstruction focuses on arrhythmia treatment. For atrial fibrillation, pharmacologic cardioversion with beta-blockers or verapamil/diltiazem is recommended, depending on symptom severity and comorbidities. In patients with dyspnea and a preserved LV ejection fraction, oral diuretics are an appropriate treatment.

Asymptomatic obstructive HCM. This form represents a small proportion of patients in the early stages of disease. Pharmacotherapy is generally unnecessary; however, in selected clinical situations, beta-blockers and non-dihydropyridine calcium channel blockers may be administered to reduce LV pressure and prevent complications [7, 19].

N.B. In hypertensive patients with diagnosed HCM, vasodilators and high-dose diuretics should be avoided, as they may exacerbate LVOT obstruction [33].

Symptomatic obstructive HCM. This form is associated with significant hemodynamic disturbances and disease progression, manifesting as SCD risk, diastolic and systolic dysfunction, ultimately leading to refractory chronic heart failure. Treatment aims are comprehensive, targeting pathophysiological mechanisms (increased end-diastolic pressure, reduced LV filling, hypercontractility, arrhythmogenic substrate, diastolic dysfunction, impaired functional capacity) caused by anatomical abnormalities (LV and interventricular septal hypertrophy, LVOT obstruction, mitral valve anomalies with regurgitation) [34]. Clinicians aim to relieve or eliminate symptoms, including angina, dyspnea, arrhythmias, syncope, and heart failure, to improve quality of life, slow disease progression, and delay mortality. Therapy includes both pharmacologic and invasive interventions.

For symptomatic obstructive and non-obstructive HCM, low-dose non-vasodilating beta-blockers (e.g., metoprolol, bisoprolol) are the first-line treatment for angina and dyspnea, titrated slowly to achieve symptomatic relief and maximal tolerated doses (heart rate 55–60 bpm at rest) [1, 18, 35]. Beta-blockers are preferred due to their negative inotropic effect and ability to reduce adrenergic tachycardia, prolonging diastolic filling and enhancing ventricular relaxation.

N.B. Beta-blockers are contraindicated in patients with sinus bradycardia or conduction disturbances. Caution is required with vasodilating beta-blockers, for patients intolerant to or contraindicated for beta-blockers, non-dihydropyridine calcium channel blockers are recommended, including verapamil (titrated from 40 mg three times daily up to 480 mg/day) and diltiazem (titrated from 60 mg three times daily up to 360 mg/day), which improve diastolic parameters and prevent myocardial ischemia.

N.B. Potential adverse effects of verapamil include hemodynamic compromise, increased LVOT obstruction, and pulmonary hypertension leading to pulmonary edema. Verapamil and diltiazem are contraindicated in patients with severe LVOT obstruction (≥ 80 mm Hg), sick sinus syndrome, heart failure, cardiogenic shock, or second- to third-degree AV block, even with a pacemaker. Dihydropyridines (e.g.,

nifedipine) are not recommended in obstructive HCM.

In refractory obstructive HCM, disopyramide is recommended, exerting negative inotropic effects via sodium channel modulation and intracellular calcium reduction, thereby lowering pressure gradients, improving functional capacity, and carrying a low arrhythmogenic risk [20, 21].

N.B. When combining disopyramide with beta-blockers or calcium channel blockers, low doses should be used to minimize adverse effects. Disopyramide is contraindicated in glaucoma and should not be combined with QT-prolonging drugs (sotalol, amiodarone) [36]. Long-term therapy with cibenzoline (a class Ia antiarrhythmic) also reduces the LVOT gradient, improves diastolic function, and modulates remodeling [37].

Arrhythmias are a hallmark of HCM. Atrial fibrillation is common, causing palpitations, skipped beats, and worsening heart failure symptoms. Management follows standard AF protocols but requires consideration of HCM-specific arrhythmia aggressiveness and drug tolerance. Preferred agents include beta-blockers or non-dihydropyridine calcium channel blockers. Amiodarone, disopyramide, or sotalol may be used if first-line agents are insufficient. Anticoagulation for stroke prevention is recommended regardless of CHA_2DS_2-VASc score, even after a single documented atrial fibrillation episode.

For ventricular tachycardia, beta-blockers are the first-line treatment; if refractory or recurrent, an ICD is indicated. Updated 2024 guidelines recommend ICD for such patients: family history of SCD, unexplained syncope, massive LV hypertrophy (≥ 30 mm), significant myocardial fibrosis (> 15 % LV mass), apical aneurysm, and end-stage heart failure (LV ejection fraction < 50 %) [38]. Catheter ablation may be considered in symptomatic atrial fibrillation patients unresponsive to therapy or in recurrent monomorphic ventricular tachycardia with a defined focus.

Heart failure management in symptomatic obstructive HCM requires attention to hemodynamic status. In patients with preserved LV ejection fraction, there is insufficient evidence for ACE inhibitors or angiotensin receptor blockers [39]. Beta-blockers and non-dihydropyridine calcium channel blockers remain the first-line treatment. Patients with systolic dysfunction should receive guideline-directed therapy including ACE inhibitors, ARBs, mineralocorticoid receptor antagonists, sodium-glucose co-transporter 2 (SGLT2) inhibitors, and neprilysin inhibitors [1, 40].

N.B. Positive inotropes (digoxin, pressors) should be avoided due to adverse hemodynamic effects and worsening LVOT obstruction. Loop and thiazide diuretics should be used cautiously in low doses.

Recently, mavacamten, a first-in-class myosin inhibitor, has been introduced for symptomatic obstructive HCM (gradient ≥ 50 mm Hg), reducing contractility and LVOT obstruction, improving functional class, and optimizing myocardial energy utilization [23, 24, 25, 26, 41]. Mavacamten can be combined with beta-blockers (or verapamil/diltiazem if beta-blockers are contraindicated) and titrated to a maximum dose of 15 mg/day.

N.B. Mavacamten is contraindicated in patients with LV ejection fraction < 55 % and requires echocardiographic monitoring; therapy should be discontinued if LV ejection fraction falls below 50 % or clinical status worsens. Combination with disopyramide is not recommended.

Despite advances in pharmacotherapy, drugs alone cannot fundamentally reverse myocardial anatomical changes, slow remodeling, or prevent pathophysiological consequences of symptomatic obstructive HCM. Invasive approaches have therefore been proposed. **ICD** is indicated in patients with life-threatening arrhythmias, particularly non-sustained VT (≥ 3 episodes with HR > 130 bpm) or persistent/resistant atrial fibrillation [1, 28].

Surgical management. Septal myectomy has been validated in leading centers. A study of 250 symptomatic obstructive HCM patients who underwent surgery between 2016 and 2019 assessed surgical efficacy [42]. The inclusion criteria were an LVOT gradient of ≥ 50 mm Hg at rest or after provocation (Valsalva maneuver) and NYHA class III–IV heart failure symptoms resistant to medical therapy (93 patients, 37.2 %). Procedures included extended septal myectomy, resection of abnormal chordal structures, papillary muscle mobilization, and anterior mitral leaflet plication. Pre- and post-operative assessment included LVOT gradient, mitral regurgitation, and functional class. Surgery resulted in significant LVOT gradient reduction (pre-op 92.8±30.7 mm Hg vs. post-op 19.3±8.7 mm Hg, $p<0.001$) and improvement in heart failure class (61.6 % of patients shifted from NYHA III–IV to I–II, $p<0.001$).

Postoperative complications included AV block and life-threatening ventricular arrhythmias: 4 patients (1.6 %) required ICD and 10 patients (4 %) required pacemakers due to complete AV block [42]. In 41 patients undergoing alcohol septal ablation, complete AV block occurred in 16.7 %, requiring pacemaker implantation in 4.8 % [43].

Comparative analyses of septal reduction techniques evaluated safety and efficacy in different timeframes. Y. Yokoyama et al. analyzed 27 observational studies comprising 15,968 patients [44]. The primary endpoint was 1-year all-cause mortality; secondary endpoints included reduction in LVOT gradient and re-intervention rates. Long-term mortality (≥ 5 years)

was higher in the alcohol ablation subgroup compared to myectomy. Alcohol ablation was associated with lower gradient reduction and higher re-intervention rates. Between October 2017 and March 2019, 16 patients underwent extended surgical myectomy after unsuccessful alcohol ablation, resulting in peak systolic gradient reduction from 86 mm Hg (IQR 75–104.7) to 20 mm Hg (IQR 16–22, p<0.001) [45]. No iatrogenic ventricular septal defects or significant postoperative mitral regurgitation were observed. No in-hospital or overall mortality occurred. Extended myectomy post-failed alcohol ablation is therefore safe and effective, but the final decision rests with the cardiac surgeon.

Conclusions

The diagnosis of hypertrophic cardiomyopathy can be suspected through a comprehensive clinical examination and determining in patients the symptoms associated with left ventricular outflow tract obstruction – recurrent syncope with transient loss of consciousness, resuscitation after cardiac arrest, paroxysms of ventricular tachycardia, left ventricle heart failure, family history of sudden cardiac death. The final diagnostic emphasis is proven by specific abnormality of cardiac precise anatomical, functional, and tissue characteristics with prognostic implications resulted from using imaging techniques particularly cardiac magnetic resonance and echocardiography. To reveal the etiological origin and refine the differential diagnosis of hypertrophic cardiomyopathy, particularly in the context of multisystem disorders, the implementation of genetic analysis to identify pathogenic gene variants is essential, serving as a critical component of diagnostic, prognostic, and therapeutic processes.

The heterogeneity of hypertrophic cardiomyopathy manifestations, along with the dynamic evolution of hemodynamic parameters and clinical complications, necessitates a balanced, multidisciplinary approach involving primary care physicians, specialists in cardiac imaging and functional diagnostics, cardiologists, geneticists, and cardiac surgeons. A key preventive measure against disease destabilization is the development of innovative gene therapy approaches aimed at targeted correction of pathogenic gene variants.

Thus, effective management of hypertrophic cardiomyopathy requires a multidisciplinary approach, with personification the optimal treatment strategy for each patient.

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Мультидисциплінарний менеджмент хворих із гіпертрофічною кардіоміопатією

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Стаття містить огляд літературних наукових джерел даних MEDLINE на платформі PubMed, Web of Science, Scopus стосовно гіпертрофічної кардіоміопатії з акцентом на лікувальні аспекти. Відповідно до рекомендацій провідних експертів подано сучасне визначення гіпертрофічної кардіоміопатії з описом класифікаційних структурних та функціональних серцевих аномалій, асоційованих симптомів і узагальнених факторів ризику раптової серцевої смерті хворих. Представлено ранжування пацієнтів з огляду на обструкцію виносного тракту лівого шлуночка та клінічну маніфестацію. Згідно з міжнародними настановами наведено основні заходи лікування хворих залежно від гемодинамічних та клінічних характеристик із застосуванням фармакотерапії та інвазивних методів. Надано патофізіологічну аргументацію та клінічне обґрунтування показань до призначення препаратів з метою корекції ускладнень. В історичному контексті окреслено застосування септальної міектомії й алкогольної септальної абляції для усунення морфологічних аномалій серця та покращання клінічного статусу пацієнтів. Наведено результати власного досвіду хірургічного лікування хворих із гіпертрофічною кардіоміопатією.

Ключові слова: гіпертрофічна кардіоміопатія, клінічні фенотипи, мішені лікування, фармакотерапія, септальна міектомія, алкогольна септальна абляція