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Review Article

Evaluation of electrocardiographic and echocardiographic findings in patients with cardiomyopathy: a narrative review

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ABSTRACT

Cardiomyopathies represent a heterogeneous group of myocardial diseases characterized by structural and functional alterations of the heart muscle. These conditions are a significant cause of morbidity and mortality worldwide, frequently leading to heart failure, arrhythmias, and sudden cardiac death (SCD). Electrocardiography (ECG) and echocardiography are the cornerstone diagnostic tools in the evaluation of these conditions, providing critical information about cardiac structure, function, and electrical activity. The purpose of this narrative review is to examine the most clinically significant cardiomyopathies-dilated cardiomyopathy (DCM), hypertrophic cardiomyopathy (HCM), and restrictive cardiomyopathy (RCM)-with a focus on their epidemiology, pathophysiology, and the characteristic ECG and echocardiographic findings used in diagnosis and management. Relevant literature was identified through PubMed and major cardiology guidelines up to March 2021. Search terms included: "cardiomyopathy," "dilated cardiomyopathy," "hypertrophic cardiomyopathy," "restrictive cardiomyopathy," "ECG findings cardiomyopathy," and "echocardiography cardiomyopathy." HCM is the most prevalent primary cardiomyopathy with an estimated prevalence of 1 in 500 persons. Dilated cardiomyopathy (DCM) has a prevalence of approximately 1 in 2,500 and is the leading indication for cardiac transplantation. Restrictive cardiomyopathy (RCM) is the rarest of the three, accounting for approximately 2-5% of cardiomyopathy cases. Each type demonstrates distinct and clinically meaningful ECG and echocardiographic patterns. Cardiomyopathies are associated with significant cardiovascular risk, and HCM in particular remains the leading cause of SCD in young athletes in the United States. Early identification using ECG and echocardiography is essential for risk stratification and management. Distinguishing physiological cardiac remodeling from pathological disease requires comprehensive diagnostic evaluation.

Keywords: Cardiomyopathy, Dilated cardiomyopathy, Hypertrophic cardiomyopathy, Restrictive cardiomyopathy, ECG findings, Echocardiography, Sudden cardiac death, Heart failure

INTRODUCTION

Cardiomyopathy is a disease of the heart muscle (myocardium) that is not attributable to coronary artery disease, hypertension, valvular disease, or congenital cardiac defects.[1] It represents a significant and growing global health burden, responsible for a substantial proportion of heart failure hospitalizations and SCD events, particularly among the young. The World Health Organization (WHO) and subsequent expert consensus bodies have classified cardiomyopathies into primary forms-where myocardial disease is the predominant pathology-and secondary forms, where myocardial involvement arises as a result of a systemic disorder by Elliott et al.[1-4]

The three major forms of primary cardiomyopathy are DCM, HCM, and restrictive cardiomyopathy (RCM),

each with distinct pathophysiological mechanisms, clinical presentations, and diagnostic profiles by Arbustini et al.[1] DCM is characterized by ventricular dilation and systolic dysfunction, HCM by pathological myocardial hypertrophy with preserved or hyperdynamic systolic function, and RCM by impaired ventricular filling in the setting of preserved ventricular dimensions and relatively normal systolic function by Muchtar et al.[17]

Advances in cardiac imaging, particularly two-dimensional (2D) and Doppler echocardiography, have transformed the diagnosis and management of cardiomyopathies. These techniques, combined with ECG assessment, allow clinicians to characterize structural abnormalities, quantify functional impairment, and stratify risk for arrhythmic events and SCD by Lang et al.[10] The integration of genetic testing, cardiac MRI, and electrophysiological studies has further enhanced diagnostic precision by Maron et al.[12] This review synthesizes current knowledge on the ECG and echocardiographic findings across the three major cardiomyopathy subtypes.

Aim and objectives

The aim of this study is to evaluate cardiomyopathy and analyze the characteristic ECG and echocardiographic findings in affected patients across the three major subtypes: DCM, HCM, and RCM.

The specific objectives are to examine the epidemiology, etiology, and pathophysiology of each cardiomyopathy subtype; describe the clinical presentation and symptomatology; evaluate characteristic ECG abnormalities associated with each type; describe echocardiographic features that distinguish each subtype; and outline current management strategies.

CARDIOMYOPATHY: DEFINITION AND CLASSIFICATION

Cardiomyopathies are a group of diseases that primarily affect the myocardium, leading to mechanical and/or electrical dysfunction that typically results in inappropriate hypertrophy or dilation by Elliott et al.[4] These conditions may be inherited or acquired, may present at any age, and can manifest across a broad spectrum of clinical severity, ranging from asymptomatic disease to fulminant heart failure and SCD by Maron et al.[13]

The American Heart Association (AHA) classifies cardiomyopathies as primary (genetic, acquired, or mixed) and secondary, the latter encompassing infiltrative diseases such as amyloidosis and storage disorders such as haemochromatosis by Maron et al.[13] The European Society of Cardiology (ESC) classification organizes cardiomyopathies by morphofunctional phenotype (HCM, DCM, RCM, arrhythmogenic right ventricular cardiomyopathy, and unclassified forms), with further subclassification into familial and non-familial forms Elliott et al.[4] Despite differing nosologies, both classifications agree that HCM, DCM, and RCM constitute the three most clinically significant subtypes.

DCM

Definition and pathophysiology

DCM is defined by the presence of left ventricular (LV) dilatation and impaired LV systolic function in the absence of abnormal loading conditions or coronary artery disease sufficient to explain the degree of myocardial dysfunction by Schultheiss et al.[20] The fundamental pathological process involves progressive dilation and thinning of the LV walls, which adopt a spherical rather than the normal conical shape. This geometric remodeling reduces the efficiency of ventricular contraction, resulting in decreased stroke volume and cardiac output by McNally et al.[16]

At the cellular level, DCM is characterized by myocyte loss, hypertrophy of surviving myocytes, interstitial fibrosis, and disruption of the extracellular matrix. These changes collectively impair myocardial contractility and compliance by Hershberger et al.[6] Functional mitral regurgitation frequently develops secondary to annular dilation and papillary muscle displacement, further exacerbating haemodynamic compromise by Schultheiss et al.[20]

Epidemiology

DCM has an estimated prevalence of approximately 1 in 2,500 in the general population, though this may be an underestimate due to the often-subclinical nature of early disease by Schultheiss et al.[20] It is the leading cause of cardiac transplantation in the developed world and accounts for a significant proportion of new heart failure diagnoses globally. The condition affects males more commonly than females at a ratio of approximately 3:1, and its incidence appears to be increasing, likely reflecting improved diagnostic detection by McNally et al.[16]

Etiology

The etiology of DCM is heterogeneous. Genetic causes account for approximately 25-35% of DCM cases, most commonly through mutations in genes encoding sarcomeric proteins, cytoskeletal proteins, nuclear envelope proteins, and ion channel components by Hershberger et al.[6] Mutations in the TTN gene (encoding titin), LMNA (encoding lamin A/C), MYH7 (beta-myosin heavy chain), and SCN5A (sodium channel) are among the most commonly implicated by Gerull et al and Hershberger et al.[5,6]

Acquired causes include viral myocarditis (particularly Coxsackievirus B and adenovirus), chronic alcohol use,

cardiotoxic chemotherapeutic agents (such as anthracyclines), and peripartum cardiomyopathy by Caforio et al.[2] Autoimmune mechanisms, including the production of anti-myocardial antibodies, are increasingly recognized as contributors to disease pathogenesis by Schultheiss et al.[20] In a substantial proportion of cases (≈30%), no identifiable cause is found, and these are classified as idiopathic DCM.

Clinical presentation

Clinical manifestations of DCM vary widely depending on the degree of ventricular dysfunction. The most common presenting symptoms include exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea, peripheral oedema, and fatigue-all consistent with congestive heart failure by Schultheiss et al.[20] Palpitations and syncope may occur as a result of arrhythmias including atrial fibrillation (AF), ventricular tachycardia, and left bundle branch block (LBBB). Some patients present with embolic events, including stroke, due to thrombus formation in the dilated and hypokinetic LV (Krum and Abraham).

Physical examination may reveal a displaced and diffuse apex beat, a third heart sound (S3) indicating reduced LV compliance, a pansystolic murmur of functional mitral regurgitation, elevated jugular venous pressure, and peripheral oedema.

ECG findings in DCM

Electrocardiographic abnormalities are present in more than 80% of patients with DCM, though findings are typically non-specific by Caforio et al.[2] The most important ECG finding is left bundle branch block (LBBB), which indicates ventricular conduction delay and is a predictor of adverse outcomes. LBBB is identified by a wide QRS complex (>120 ms) with a broad or notched R wave in leads V5-V6 and a deep S wave in V1, producing a characteristic 'M' pattern in lateral leads and a 'W' pattern in V1.

Additional ECG findings include left axis deviation, non-specific intraventricular conduction delay, and sinus tachycardia reflecting neurohormonal activation. Left atrial enlargement may be evidenced by broad, notched P waves (P mitrale) in lead II or a dominant negative terminal deflection of the P wave in V1. A pseudo-infarction pattern-manifested as poor R-wave progression or QS complexes in precordial leads V1-V4-may mimic anterior myocardial infarction and result from diffuse myocardial fibrosis rather than ischemic injury by Caforio et al.[2]

Low-voltage QRS complexes, particularly in limb leads, can occur secondary to diffuse myocardial fibrosis and may indicate a worse prognosis. Repolarization abnormalities, including T-wave inversions especially in lateral leads and nonspecific ST-T changes, are frequently observed. Atrial arrhythmias, particularly AF, and ventricular arrhythmias including ventricular premature complexes and non-sustained ventricular tachycardia (NSVT) are common and important for risk stratification by Schultheiss et al.[20]

Echocardiographic findings in DCM

Echocardiography is the primary imaging modality for diagnosis and follow-up of DCM. The hallmark finding is LV dilatation, evidenced by an increased LV internal diameter in diastole (LVIDd), typically exceeding 5.5 cm in men and 5.0 cm in women, though indexing to body surface area is preferred by Lang et al.[10] Associated features include thinning of the LV walls (relative to the degree of dilation), a spherical LV cavity geometry (reflected by an LV eccentricity index approaching 1), and reduced wall motion amplitude (global hypokinesia).

The most critical functional parameter is the LV ejection fraction (LVEF), which is typically reduced to below 40% in symptomatic DCM, reflecting severely impaired systolic function by Lang et al.[10] Unlike the regional wall motion abnormalities observed in ischaemic cardiomyopathy, DCM characteristically demonstrates diffuse or global reduction in contractility without discrete segmental involvement, although exceptions exist by Schultheiss et al.[20]

Doppler echocardiography frequently demonstrates functional mitral regurgitation (MR), characterized by a central or posteriorly directed MR jet due to annular dilation and tethering of the mitral leaflets. Diastolic dysfunction is also commonly identified and may progress from an impaired relaxation pattern to a restrictive filling pattern in advanced disease, correlating with elevated LV filling pressures by Lang et al.[10] Left atrial (LA) enlargement, right ventricular (RV) dilation in advanced cases, and intracardiac thrombus-most commonly located at the LV apex-are additional echocardiographic features with important clinical implications by Schultheiss et al.[20]

Management of DCM

Management of DCM is guided by the degree of LV dysfunction and symptom burden. Guideline-directed medical therapy (GDMT) for heart failure with reduced ejection fraction (HFrEF) forms the cornerstone of treatment, including angiotensin-converting enzyme inhibitors (ACE-I) or angiotensin receptor–neprilysin inhibitors (ARNI), beta-blockers, mineralocorticoid receptor antagonists, and sodium-glucose cotransporter-2 (SGLT2) inhibitors by McDonagh et al.[15] Device therapy, including cardiac resynchronization therapy (CRT) in patients with LBBB and QRS duration >150 ms, and implantable cardioverter-defibrillators (ICD) in patients with LVEF ≤35%, are recommended to reduce SCD risk by McDonagh et al.[15] Cardiac transplantation remains the definitive treatment for end-stage DCM refractory to medical and device therapy.

HCM

Definition and pathophysiology

HCM is defined as unexplained LV hypertrophy in the absence of another cardiac or systemic disease capable of producing the degree of hypertrophy observed, most commonly identified by LV wall thickness ≥15 mm on echocardiography or cardiac MRI by Ommen et al.[19] The hypertrophy typically involves the interventricular septum to a greater degree than the LV posterior wall-a pattern known as asymmetric septal hypertrophy (ASH)-though it may also be concentric or confined to the LV apex (apical variant) by Maron et al.[13]

The fundamental pathological abnormalities in HCM include myocyte disarray, interstitial fibrosis, and microvascular dysfunction, all of which contribute to the electrical instability that predisposes to arrhythmias and SCD by Ho et al. Diastolic dysfunction is the dominant haemodynamic abnormality, resulting from impaired LV relaxation and reduced compliance of the hypertrophied myocardium. In approximately 70% of patients, dynamic obstruction of the LV outflow tract (LVOT) occurs, precipitated by systolic anterior motion (SAM) of the anterior mitral leaflet toward the hypertrophied septum, generating a pressure gradient across the LVOT by Maron et al and Ommen et al.[13,19]

Epidemiology

HCM is the most common primary cardiomyopathy with a prevalence of approximately 1 in 500 in the general population by Maron et al.[13] It affects males and females equally, though males tend to present with more severe phenotypic expression. HCM is the most frequent identifiable cause of SCD in competitive athletes in the United States, underscoring its clinical importance in the context of pre-participation screening by Maron et al.[13]

Etiology

HCM is predominantly a genetic disorder inherited in an autosomal dominant pattern, with approximately 60% of cases attributable to mutations in genes encoding sarcomere proteins. Mutations in MYH7 (encoding beta-myosin heavy chain) and MYBPC3 (encoding myosin-binding protein C) are the most prevalent, collectively accounting for approximately 70-80% of genotype-positive HCM cases by Maron et al. Other implicated genes include TNNT2 (cardiac troponin T), TNNI3 (cardiac troponin I), and TPM1 (alpha-tropomyosin).

Phenocopies of HCM-conditions producing LV hypertrophy through non-sarcomere mechanisms-must be excluded, particularly in older patients. These include Fabry disease (alpha-galactosidase A deficiency), Pompe disease, Friedreich's ataxia, and cardiac amyloidosis, which may require specific therapies distinct from HCM management by Ommen et al.[19]

Clinical presentation

The clinical spectrum of HCM is broad. A significant proportion of patients remain asymptomatic throughout their lives, while others develop progressive symptoms including exertional dyspnea, chest pain (angina), palpitations, presyncope, and syncope (Maron et al).[13] Syncope or presyncope in HCM may result from LVOT obstruction, diastolic dysfunction with inadequate cardiac output augmentation on exertion, or ventricular arrhythmias by Ommen et al.[19]

On physical examination, patients with obstructive HCM typically manifest a harsh crescendo-decrescendo systolic ejection murmur at the left sternal border, which is characteristically augmented by maneuvers that reduce preload or afterload (e. g., Valsalva maneuver, standing) and diminished by those that increase preload (e.g., squatting) by Maron et al.[13]

ECG findings in HCM

ECG abnormalities are present in approximately 90-95% of patients with HCM and often precede the development of echocardiographic features by Maron et al.[13] However, findings are variable and no single ECG pattern is pathognomonic. The most common finding is LV hypertrophy (LVH), identified by increased QRS voltage using standard criteria such as the Sokolow-Lyon index (S in V1 + R in V5 or V6 ≥35 mm) or Cornell voltage criteria, frequently accompanied by a strain pattern comprising ST-segment depression and T-wave inversion in lateral leads by Surawicz et al.

Characteristic 'dagger-like' deep, narrow Q waves are a hallmark of HCM, seen in 20-50% of cases in the inferior leads (II, III, aVF) and lateral leads (I, aVL, V5-V6). These are caused by abnormal septal depolarization vectors resulting from septal hypertrophy and myocyte disarray, and must be distinguished from pathological Q waves of myocardial infarction by Maron et al.[13] The apical variant of HCM (Yamaguchi syndrome) is characterized by striking giant T-wave inversions in the mid-to-lateral precordial leads (V3-V5), with voltage changes suggesting LVH (Maron et al).[13]

Left atrial enlargement is frequently observed, represented by broad, notched P waves in lead II. Atrial fibrillation is the most common sustained arrhythmia in HCM, occurring in 20-25% of patients, and is a major determinant of stroke risk and clinical deterioration by Ommen et al.[19] Bundle branch blocks, most commonly LBBB, and AV conduction delays may also be encountered.

Echocardiographic findings in HCM

Echocardiography remains the primary diagnostic tool for HCM, and its findings are often pathognomonic. The most characteristic feature is asymmetric septal

hypertrophy (ASH), defined by a ratio of septal-to-posterior wall thickness exceeding 1.3:1 to 1.5:1, with septal thickness typically ≥15 mm in adults by Lang et al and Ommen et al.[10,19] The LV cavity is characteristically non-dilated and often small due to the prominent hypertrophy, contrasting sharply with the dilated cavity seen in DCM.

A critically important echocardiographic finding in HCM is systolic anterior motion (SAM) of the mitral valve, in which the anterior mitral leaflet moves toward the interventricular septum during systole. SAM generates dynamic LVOT obstruction and is identified on Doppler echocardiography as a late-peaking, 'dagger-shaped' systolic jet with a peak velocity typically exceeding 3-4 m/s. A peak instantaneous LVOT gradient of ≥30 mmHg at rest or ≥50 mmHg with provocation (e.g., Valsalva) is considered haemodynamically significant by Ommen et al.[19]

Systolic function, measured by LVEF, is typically normal or hyperdynamic in HCM, except in the small proportion of patients who develop a 'burnt-out' or dilated-phase HCM with systolic dysfunction. Diastolic dysfunction is universal, with echocardiographic evidence of impaired LV relaxation, reduced mitral annular velocities on tissue Doppler imaging (TDI), and elevated LV filling pressures by Lang et al.[10] Left atrial enlargement is secondary to chronic diastolic dysfunction and is associated with increased risk of AF by Ommen et al.[19]

Management of HCM

Management of HCM is individualized and directed toward symptom relief, arrhythmia management, and SCD prevention by Ommen et al.[19] In asymptomatic patients, lifestyle modification is emphasized, including avoidance of competitive sports and strenuous exertion. Beta-blockers (propranolol, metoprolol) are first-line pharmacotherapy for symptom control, reducing heart rate, prolonging diastolic filling time, and ameliorating LVOT obstruction. Calcium channel blockers (verapamil) represent an alternative in beta-blocker-intolerant patients, and disopyramide may be added for refractory LVOT obstruction by Ommen et al.[19]

Mavacamten, a selective cardiac myosin inhibitor recently approved by regulatory authorities, represents a novel targeted therapy that reduces LVOT gradients and improves symptoms in obstructive HCM (Olivotto et al). For patients with drug-refractory obstructive HCM, invasive septal reduction therapies-surgical septal myectomy (the gold standard) or alcohol septal ablation (a percutaneous alternative)-are recommended by Ommen et al.[19] ICD implantation is indicated in high-risk patients for primary SCD prevention, based on a five-year SCD risk score incorporating clinical and imaging parameters.

RESTRICTIVE CARDIOMYOPATHY

Definition and pathophysiology

Restrictive cardiomyopathy (RCM) is the least common of the major cardiomyopathy subtypes, characterized by abnormal diastolic function resulting from impaired ventricular filling in the setting of normal or near-normal LV systolic function and non-dilated ventricles by Muchtar et al.[17] The fundamental pathophysiological mechanism involves increased stiffness of the ventricular walls, which restricts ventricular filling during diastole, reducing end-diastolic volume and cardiac output despite preserved ejection fraction by Kushwaha et al.[9]

As ventricular filling is impaired, diastolic pressures rise, causing pressure to be transmitted retrograde into the pulmonary venous system (left-sided) and systemic venous system (right-sided), resulting in congestive symptoms and biatrial enlargement. In advanced disease, right heart failure with hepatic congestion, ascites, and peripheral oedema predominates by Muchtar et al.[17]

Epidemiology

RCM accounts for approximately 2-5% of all cardiomyopathy cases, making it the rarest major subtype by Muchtar et al.[17] It affects children more commonly than adults in idiopathic forms, with a peak incidence at 5-6 years of age, though secondary forms-particularly cardiac amyloidosis-predominantly affect older adults. Its prognosis is generally poor, with a high rate of progression to end-stage heart failure requiring transplantation by Kushwaha et al.[9]

Etiology

The causes of RCM are diverse. Idiopathic RCM, in which no underlying cause is identified, may be sporadic or familial (with mutations in TNNI3, MYH7, and desmin gene reported) (Arbustini et al).[1] Secondary causes are more common and include infiltrative diseases-most importantly cardiac amyloidosis (caused by deposition of misfolded amyloid fibrils within the myocardium), haemochromatosis (iron overload leading to myocardial iron deposition), and sarcoidosis. Endomyocardial fibrosis (EMF), hypereosinophilic syndrome, and radiation-induced cardiomyopathy following chest radiotherapy are additional recognized causes by Muchtar et al and Kushwaha et al.[9,17]

Clinical presentation

The clinical presentation of RCM is dominated by symptoms and signs of biventricular heart failure. Dyspnea, orthopnea, exercise intolerance, peripheral oedema, ascites, and hepatomegaly are common by Muchtar et al.[17] Atrial arrhythmias, particularly AF, are frequent due to biatrial enlargement and contribute to thromboembolic risk. Heart block and conduction

abnormalities may occur in infiltrative forms, especially cardiac sarcoidosis and amyloidosis. Systemic features of the underlying cause (e.g., periorbital bruising and macroglossia in AL amyloidosis, or hepatomegaly and skin pigmentation in haemochromatosis) may provide diagnostic clues by Kushwaha et al.[9]

ECG findings in RCM

ECG findings in RCM are non-specific but characteristic of biatrial involvement and myocardial infiltration. The most common finding is biatrial enlargement, manifesting as broad, notched P waves (P mitrale, indicating left atrial enlargement) and tall, peaked P waves (P pulmonale, indicating right atrial enlargement) in multiple leads by Muchtar et al.[17] In infiltrative RCM, particularly amyloidosis, low-voltage QRS complexes-defined as QRS amplitude <5 mm in all limb leads and <10 mm in all precordial leads-are a classic finding, resulting from replacement of electrically active myocardium by amyloid deposits by Connors et al.[3]

Atrial arrhythmias (AF, atrial flutter, and supraventricular tachycardia) are frequently encountered. Conduction abnormalities including AV block and bundle branch blocks may occur due to infiltration or fibrosis of the conduction system, and are particularly characteristic of cardiac sarcoidosis and ATTR amyloidosis by Connors et al and Muchtar et al.[3,17] Non-specific ST-T changes, including ST-segment depression and T-wave inversion, are commonly present without a definitive pattern.

Echocardiographic findings in RCM

Echocardiography is central to the evaluation of RCM and typically reveals a constellation of findings. The hallmark is biatrial enlargement, often disproportionate to the degree of ventricular enlargement, reflecting chronically elevated filling pressures (Muchtar et al).[17] Unlike DCM, LV and RV dimensions are characteristically normal or even reduced. LV systolic function, measured by LVEF, is typically preserved (≥50%), though it may decline in advanced disease (Lang et al).[10]

Diastolic function assessment by Doppler is critical and typically reveals a restrictive filling pattern: elevated E-wave velocity, reduced A-wave velocity (E/A ratio >2), and shortened deceleration time (<160 ms), reflecting markedly elevated LV filling pressures and poor ventricular compliance (Nishimura and Tajik).[18] Tissue Doppler imaging (TDI) demonstrates markedly reduced mitral annular velocities (E'<8 cm/s), and a high E/E' ratio (>14) confirms elevated filling pressures.

In infiltrative causes-particularly amyloidosis-echocardiography may reveal increased LV wall thickness with a characteristic 'granular sparkling' appearance of the myocardium on 2D imaging, thickened cardiac valves, and thickened interatrial septum by Connors et al.[3] Dilated inferior vena cava (IVC) with reduced respiratory variation indicates elevated right atrial pressure. Pulmonary hypertension features, including elevated tricuspid regurgitant jet velocity, may also be present by Muchtar et al.[17]

Management of RCM

RCM has no disease-specific cure, and management is primarily directed at symptomatic control and prevention of complications (Muchtar et al).[17] Diuretics are used cautiously to manage congestion, though excessive preload reduction is poorly tolerated due to dependence on elevated filling pressures to maintain cardiac output. Beta-blockers and calcium channel blockers may help reduce heart rate and improve diastolic filling time. Anticoagulation is warranted in the presence of AF or intracardiac thrombus.

In secondary RCM, targeted therapy for the underlying cause is paramount. In transthyretin (ATTR) amyloidosis, tafamidis-a transthyretin stabilizer-has demonstrated mortality benefit by Maurer et al.[14] Systemic immunosuppression is used in sarcoidosis-associated RCM. Cardiac transplantation remains an option for eligible patients with end-stage RCM, though amyloidosis-related RCM requires careful patient selection due to the risk of systemic disease recurrence by Muchtar et al.[17]

DISCUSSION

This narrative review highlights the distinct and complementary roles of ECG and echocardiography in the diagnosis and phenotypic characterization of the three major cardiomyopathy subtypes. While each subtype has a unique pathophysiological basis and clinical phenotype, considerable overlap in clinical presentation-most notably dyspnea, palpitations, and reduced exercise tolerance-underscores the essential role of systematic cardiac imaging in achieving accurate diagnosis.

In DCM, the ECG frequently demonstrates LBBB and pseudo-infarction patterns that may mislead clinicians toward an ischaemic diagnosis. Echocardiography unambiguously demonstrates global LV dysfunction and dilation, clearly distinguishing DCM from ischaemic cardiomyopathy, which typically shows regional wall motion abnormalities in a coronary artery distribution by Schultheiss et al and McNally et al.[16,20] The echocardiographic finding of diffuse global hypokinesia without regional pattern should therefore prompt clinicians to consider non-ischaemic DCM even in the presence of ECG changes resembling prior infarction.

HCM presents a particularly challenging diagnostic scenario given its frequent asymptomatic course and the risk of SCD as a first manifestation. The combination of deep dagger-like Q waves, LVH on ECG, and ASH with SAM on echocardiography represents a highly specific

diagnostic profile (Maron et al and Ommen et al).[13,19] The distinction between physiological LV hypertrophy in athletes and pathological HCM-the so-called 'grey zone' of LV wall thickness between 13-15 mm-is a clinically significant challenge that may require cardiac MRI, genetic testing, and exercise testing for resolution (Maron et al and Maron et al).[12,13]

RCM remains the most diagnostically challenging cardiomyopathy, in part due to the need to distinguish it from constrictive pericarditis-a condition with overlapping haemodynamic features but requiring entirely different management. The presence of biatrial enlargement, granular myocardial texture in amyloidosis, and restrictive Doppler filling pattern on echocardiography, combined with the absence of pericardial thickening and the ECG pattern of low voltage with LV hypertrophy (a 'discordant' ECG-echo pattern highly suggestive of amyloidosis), provides the basis for differentiation by Connors et al and Muchtar et al.[3,17] Cardiac MRI with late gadolinium enhancement has emerged as an increasingly important adjunct in this distinction.

CONCLUSION

Cardiomyopathies constitute an important and heterogeneous group of myocardial diseases with significant implications for cardiovascular morbidity and mortality. ECG and echocardiography remain the foundational diagnostic tools for evaluating these conditions, providing complementary and often synergistic information. DCM is characterized echocardiographically by LV dilation with reduced ejection fraction and globally reduced wall motion, accompanied on ECG by LBBB, left axis deviation, and repolarization changes. HCM is distinguished by asymmetric septal hypertrophy and SAM on echocardiography, with ECG evidence of LVH, characteristic deep Q waves, and giant T-wave inversions in the apical variant. RCM is identified by biatrial enlargement with non-dilated ventricles and a restrictive Doppler filling pattern, with ECG commonly showing low QRS voltage in infiltrative forms and atrial arrhythmias.

Accurate and timely diagnosis through integrated use of ECG and echocardiography enables risk stratification, guides selection of pharmacological and device-based therapies, and informs decisions regarding genetic testing and family screening. Future advances in cardiac imaging, including three-dimensional echocardiography, strain imaging, and cardiac MRI, are expected to further refine diagnosis and prognostic assessment in cardiomyopathy. Clinicians caring for patients with cardiomyopathy must maintain a high index of suspicion, apply available diagnostic tools comprehensively, and adhere to evidence-based management guidelines to optimize patient outcomes.

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