# How to Do Echo in a Multimodality Approach to Assess the Risk of Sudden Death: A Consensus Statement of the Italian Society of Echocardiography and Cardiovascular Imaging

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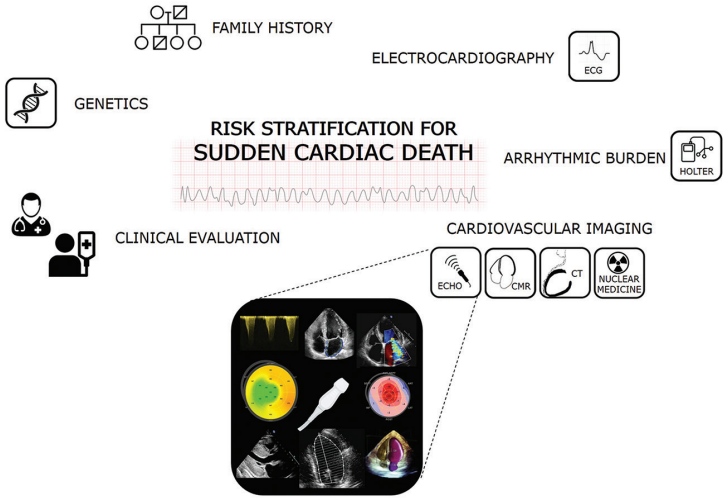
## Graphical Abstract




Risk stratification for sudden cardiac death (SCD) is a multiparametric process that integrates the data from family history, clinical evaluation, electrocardiographic findings, arrhythmic burden, and cardiovascular imaging. Echocardiography is the first-line imaging modality for both diagnosis and risk stratification of cardiovascular diseases associated with SCD. Advances in echocardiography and multimodality imaging have identified a number of parameters with proven prognostic value in SCD risk stratification.

CMR = Cardiac magnetic resonance, CT = Computed tomography, ECG = Electrocardiogram, ECHO = Echocardiography

**Keywords:** Cardiac magnetic resonance, echocardiography, multimodality imaging, risk stratification, sudden death

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## Introduction

Despite significant advances in knowledge, risk stratification, and treatment of cardiovascular diseases in recent years, sudden cardiac death (SCD) remains a major clinical and public health problem. SCD is defined as a sudden, unexpected death resulting from the abrupt cessation of cardiac activity.[1] SCD accounts for approximately 50% of all cardiovascular-related deaths, with up to 50% being the first manifestation of underlying heart disease.[1] The estimated annual incidence of SCD ranges from 30 to 125 cases per 100.000 person-years, markedly increasing with age and occurring more frequently in men.[1] In younger individuals, SCD is most commonly caused by cardiomyopathies and primary electrical disorders. In contrast, for those over 35 years old, coronary artery disease (CAD), particularly acute coronary syndromes, accounts for about half of the cases. In the elderly, the primary causes include acute and chronic CAD, valvular heart disease, and heart failure.[1]

While indications for the secondary prevention of SCD are well established for patients with a history of cardiac arrest or major ventricular arrhythmias, effectively identifying those at high risk for primary prevention remains a relevant issue in clinical practice.[1]

Several clinical and noninvasive risk markers have been proposed for arrhythmic risk stratification across different conditions. Among these, multimodality imaging plays a central role in identifying individuals at increased risk of SCD.[1-3]

Echocardiography, thanks to its noninvasiveness, wide accessibility and repeatability, is the first-line imaging modality for diagnosis and risk stratification of several conditions associated with SCD, allowing the evaluation of cardiac function and the detection of structural cardiovascular disease [Tables 1-3].[1,2]

Assessment of left ventricular ejection fraction (LVEF) remains, to date, one of the main risk stratification parameters and continues to play a crucial role in guiding the primary prevention of SCD, particularly when severely reduced.[1,2]

In addition to LVEF, emerging echocardiographic parameters – such as left ventricular global longitudinal strain (GLS) and mechanical dispersion (MD) assessed through speckle-tracking technique – have been associated with an increased risk of major ventricular arrhythmias and SCD in both ischemic and Non-Ischemic Heart disease.[1,4,7,8]

Cardiac magnetic resonance (CMR) is the gold standard for the evaluation of biventricular size and function, while enabling noninvasive tissue characterization. Its role in risk stratification for major ventricular arrhythmias and SCD is growing.[1,2] Notably, CMR is able to detect myocardial fibrosis, mainly through late gadolinium enhancement technique, disclosing the arrhythmic substrate within

**Table 1: Echocardiographic diagnostic criteria of cardiovascular conditions associated with sudden cardiac death**

| |
|---|
| DCM |
| LVEDD (mm) |
| Males >58 |
| Females >52 |
| LVEDV (mL/m$^2$) |
| Males ≥75 |
| Females ≥62 |
| LVEF <50% |
| ARCV |
| Regional RV akinesia, dyskinesia, or aneurysm and 1 of the following (end-diastole): PLAX RVOT ≥32 mm (or PLAX/BSA ≥19 mm/m$^2$) or PSAX RVOT ≥36 mm (or PSAX/BSA ≥21 mm/m$^2$) or fractional area change ≤33% (major criteria) |
| Regional RV akinesia or dyskinesia and 1 of the following (end-diastole): PLAX RVOT ≥29–<32 mm (PLAX/BSA ≥16–<19 mm/m$^2$) or PSAX RVOT ≥32–<36 mm (PSAX/BSA≥18–<21 mm/m$^2$) or fractional area change >33%–≤40% (minor criteria) |
| HCM |
| LV wall thickness >15 mm (or *z*-score >2 in Children) or ≥13 mm in in adult first-degree relatives; consider the diagnosis if LV wall thickness 13–14 mm in the presence of other abnormalities, such as family history, genetic findings, and ECG abnormalities |
| Obstructive HCM |
| Doppler evaluation of LVOTO at rest, during Valsalva maneuver, in the sitting and semi-supine positions, and then on standing if no gradient is provoked (severe if peak pressure gradient ≥50 mmHg) |
| RCM |
| Restrictive LV and/or RV pathophysiology in the presence of normal or reduced diastolic volumes (of one or both ventricles), normal or reduced systolic volumes, and normal ventricular wall thickness |
| TOF |
| Ventricular septal defect |
| Pulmonary stenosis |
| Overriding aorta |
| RV hypertrophy |
| ccTGA |
| Atrio-ventricular and ventriculo-arterial discordance |
| D-TGA |
| Pulmonary artery arising from the left ventricle |
| Aorta arising from the right ventricle |
| Ebstein anomaly |
| Apical displacement of septal and posterior tricuspid valve leaflets (>8 mm/m$^2$) |
| Atrialized right ventricle |
| Congenital coronary artery anomalies |
| AAOCA |
| ACAPA |
| Mitral valve prolapse |
| Systolic displacement of one or both mitral leaflets ≥2 mm above the plane of the mitral annulus in the sagittal view |
| MAD |
| Separation between the ventricular myocardium and the mitral annulus supporting the posterior mitral leaflet, in both systole and diastole |
| Severe aortic valve stenosis |
| Aortic valve area ≤1 cm$^2$ (or ≤0.6 cm$^2$/m$^2$) with or without mean gradient ≥40 mmHg and peak aortic velocity ≥4.0 m/s |

*Contd...*

**Table 1: Contd...**

| |
|---|
| Aortic root and ascending aorta dilatation |
| Aortic root dilatation: >40 mm in males, >34 mm in females or >22 mm/m$^2$ |
| Ascending aorta dilatation: >40 mm in males, >36 mm in females or >22 mm/m$^2$ |
| Aortic aneurysm: Diameter >1.5 times (>50%) larger than the predicted value |

AAOCA=Anomalous aortic origin of a coronary artery, ACAPA=Anomalous coronary artery from the pulmonary artery, ccTGA=Congenitally corrected transposition of the great arteries, D-TGA=Dextro-transposition of the great arteries, ECG=Electrocardiogram, LV=Left ventricular, LVEDD=Left ventricular end-diastolic diameter, LVEDV=Left ventricular end-diastolic volume, LVEF=Left ventricular ejection fraction, LVOTO=Left ventricular outflow tract obstruction, PLAX=Parasternal long axis, PSAX=Parasternal short axis, RV=Right ventricular, RVOT=Right ventricular outflow tract, RCM=Restrictive cardiomyopathy, MAD=Mitral annular disjunction, HCM=Hypertrophic cardiomyopathy, ARCV=Arrhythmogenic right ventricular cardiomyopathy, AV=Atrio-ventricular, TOF=Tetralogy of Fallot, DCM=Dilated cardiomyopathy, BSA=Body surface area

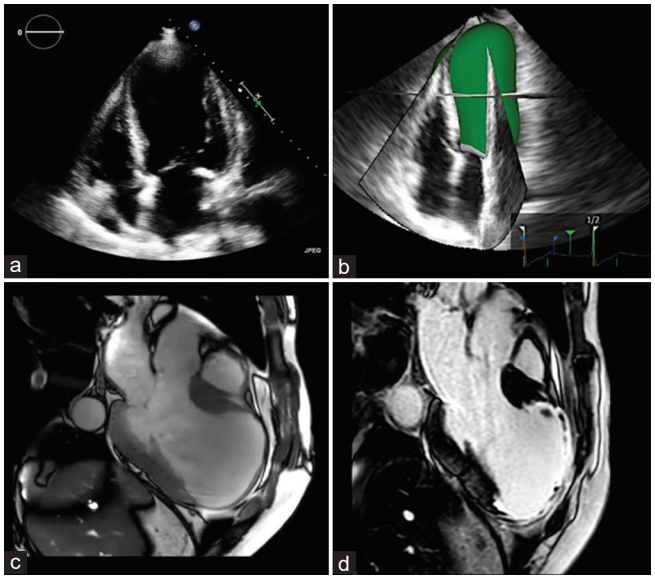



**Figure 1:** Case of ischemic heart disease in a patient hospitalized for subacute myocardial infarction. (a) Transthoracic echocardiography, apical four-chamber view: dilatation and severe systolic dysfunction of the left ventricle, along with a large apical aneurysm, which is associated with a higher risk of sudden cardiac death. (b) Three-dimensional transthoracic echocardiography allows for a more accurate assessment of biventricular volumes and function. (c) Cardiac magnetic resonance, cine steady state free precession imaging, three-chamber view: the presence of a large apical aneurysm is confirmed. (d) Cardiac magnetic resonance, post contrast imaging, three-chamber view, showing the presence of extensive late gadolinium enhancement at the level of the apical aneurysm.

the heart muscle, thus providing relevant prognostic information.[5,6]

This document aims to describe the usefulness of echocardiography and multimodality imaging in the diagnosis and stratification of SCD risk in different clinical scenarios.

**Table 2: Echocardiographic red flags in cardiac amyloidosis and Anderson-Fabry cardiomyopathy**

| |
|---|
| Cardiac amyloidosis |
| Thickening of the septum, posterior wall and RV wall |
| Granular sparkling |
| Valve thickening |
| Interatrial septum thickening |
| Preserved or mildly reduced LVEF |
| Grade 2 or worse diastolic dysfunction |
| Enlarged atria |
| Pericardial effusion (generally small) |
| Pleural effusion |
| Abnormal GLS with apical sparing pattern |
| Anderson-Fabry disease |
| LV hypertrophy with or without RV hypertrophy |
| Papillary muscle hypertrophy |
| Binary sign (echo-bright endocardium with an adjacent hyporeflective subendocardial layer) |
| Enlarged atria |
| Diastolic dysfunction |
| Abnormal GLS, generally in the inferolateral segments |
| Valve thickening |
| Ascending aorta dilatation |

GLS=Global longitudinal strain, LV=Left ventricular, LVEF=Left ventricular ejection fraction, RV=Right ventricular

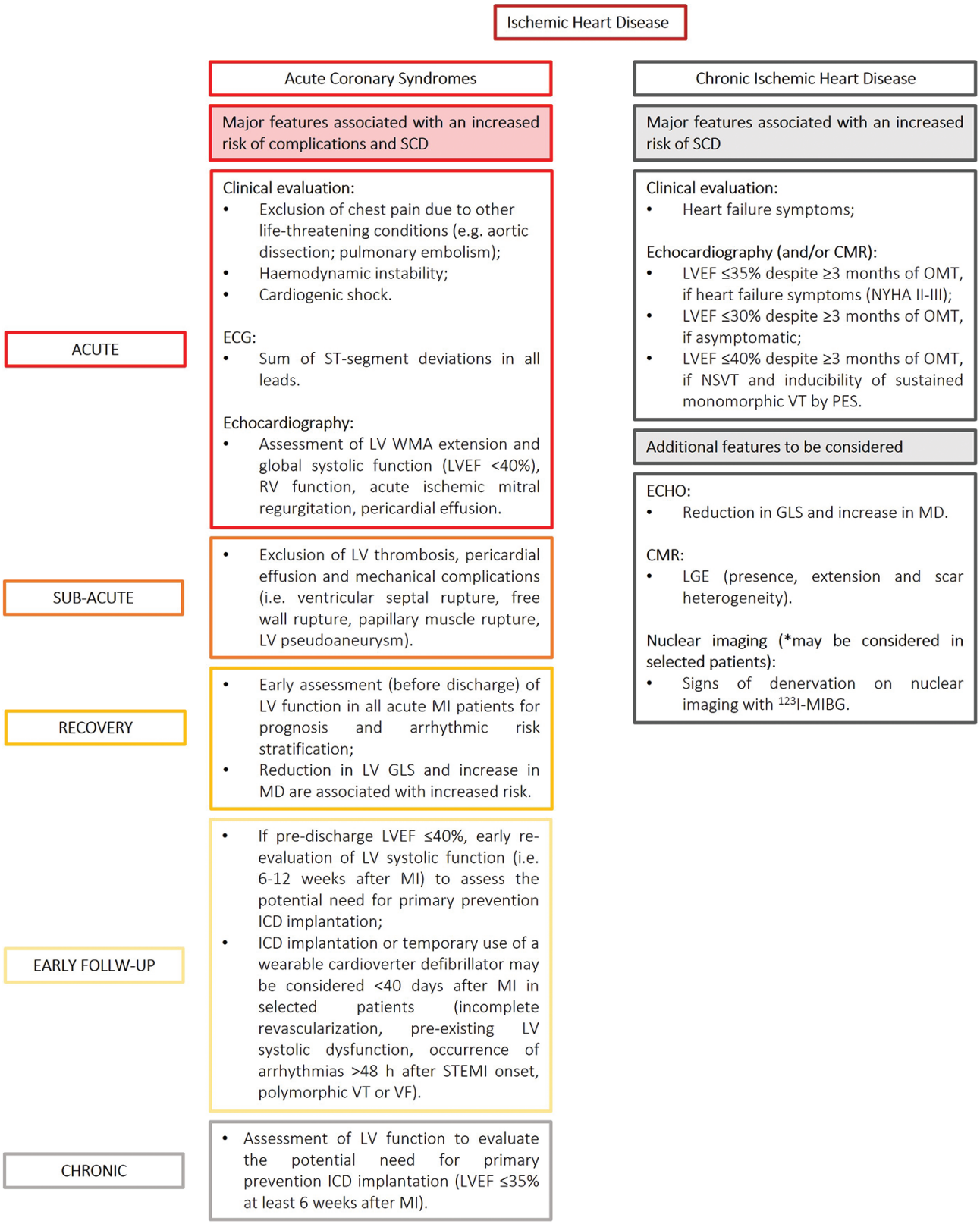
## Ischemic Heart Disease

Ischemic heart disease is the leading cause of sudden cardiac death in the general population, particularly among adults and the elderly.[1,7,8] Echocardiography is an essential diagnostic tool for evaluating patients with ischemic heart disease, especially for assessing left ventricular systolic function, the presence of valvular abnormalities, and mechanical complications of acute coronary syndromes. Left ventricular ejection fraction is the primary metric used to assess the risk of sudden cardiac death [Flow Chart 1 and Figure 1].[1,7,8]

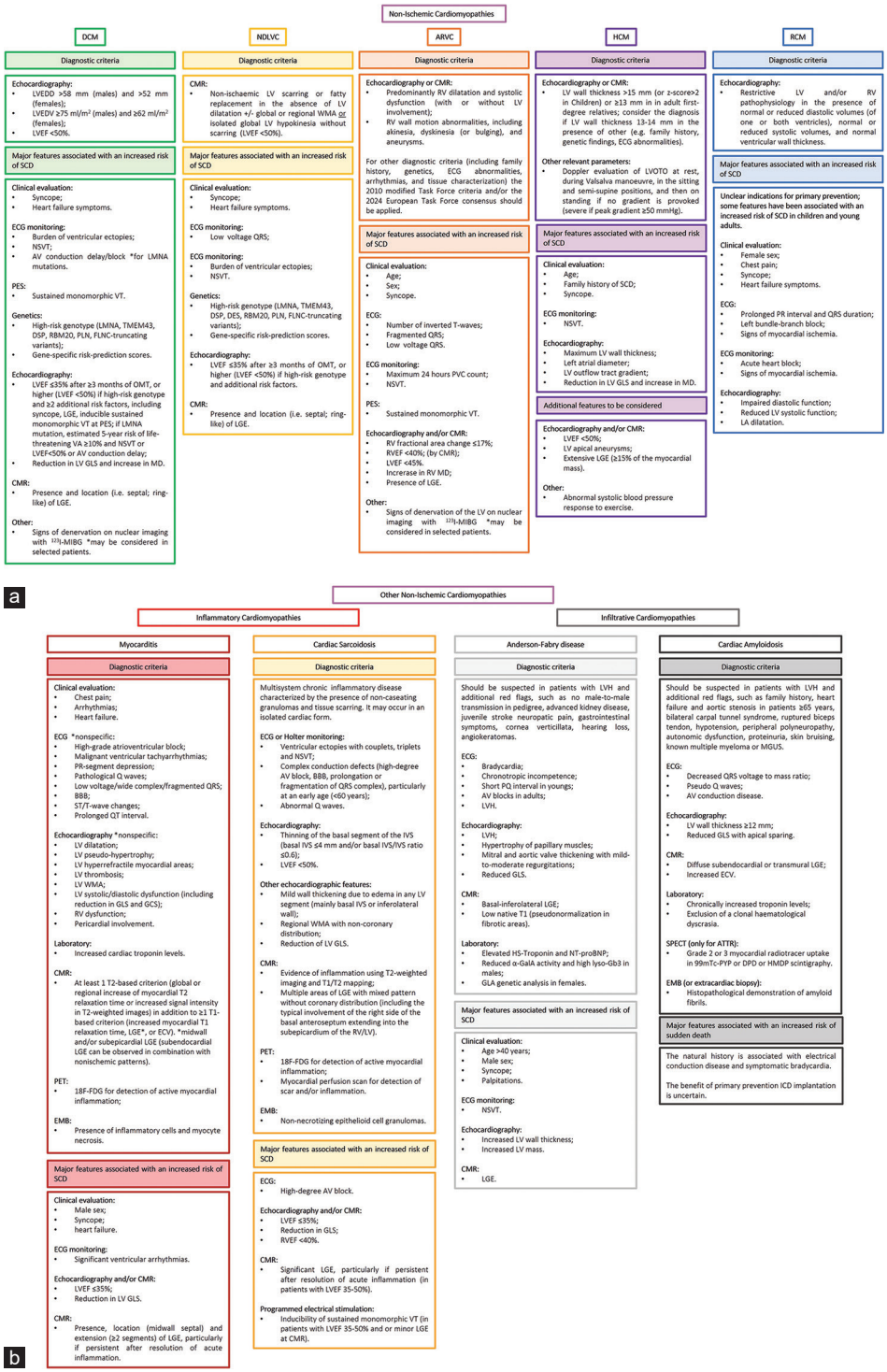
## Cardiomyopathies

In younger individuals, sudden cardiac death is mainly attributed to cardiomyopathies and primary electrical disorders.[2] Echocardiography is the first-line imaging modality in the evaluation of patients with suspected cardiomyopathy, providing valuable information for diagnosis and risk stratification of ventricular arrhythmias and heart failure.[2] Assessment of systolic function through the left ventricular ejection fraction remains the cornerstone of risk stratification for adverse events. Other clinical and imaging characteristics have to be considered in a comprehensive, multiparametric approach [Flow Chart 2a, 2b and Figures 2, 3].[9-20]

The key factors associated with an increased risk of sudden cardiac death in other nonischemic cardiomyopathies [Figure 3 and Flow Chart 2b].




**Flow Chart 1:** Key factors associated with an increased risk of sudden death in both acute and chronic ischemic heart disease. CMR = Cardiac magnetic resonance, ECG = Electrocardiogram, ECHO = Echocardiography, ICD = Implantable cardioverter-defibrillators, GLS = Global longitudinal strain, LGE = Late gadolinium enhancement, LV = Left ventricular, LVEF = Left ventricular ejection fraction, MD = Mechanical dispersion, MI = Myocardial infarction, MIBG = Metaiodobenzylguanidine, NYHA = New York Heart Association, NSVT = Nonsustained ventricular tachycardia, OMT = Optimal medical therapy, PES = Programmed electrical stimulation, RV = Right ventricular, SCD = Sudden cardiac death, SPECT, = Single-photon emission computed tomography, STEMI = ST-elevation myocardial infarction, VF = Ventricular fibrillation, VT = Ventricular tachycardia, WMA = Wall motion abnormalities




**Flow Chart 2:** Key factors associated with an increased risk of sudden cardiac death in non-ischemic cardiomyopathies. ARCV = Arrhythmogenic right ventricular cardiomyopathy, AV = Atrio-ventricular, CMR = Cardiac magnetic resonance, DCM = Dilated cardiomyopathy, ECG = Electrocardiogram, HCM = Hypertrophic cardiomyopathy, LA = Left atrial, LGE = Late gadolinium enhancement, LV = Left ventricular, LVEDD = Left ventricular end-diastolic diameter, LVEDV = Left ventricular end-diastolic volume, LVEF = Left ventricular ejection fraction, LVOTO = Left ventricular outflow tract obstruction, MIBG = Metaiodobenzylguanidine, NDLVC = Non-dilated left ventricular cardiomyopathy, NSVT = Nonsustained ventricular tachycardia, OMT = Optimal medical therapy, PES = Programmed electrical stimulation, PVC = Premature ventricular contractions, RCM = Restrictive cardiomyopathy, RV = Right ventricular, RVEF = Right ventricular ejection fraction, SCD = Sudden cardiac death, VA = Ventricular arrhythmia, VT = Ventricular tachycardia, WMA = Wall motion abnormalities, ATTR = Transthyretin amyloidosis, BBB = Bundle branch block, DPD = 3,3-diphosphono-1,2-propanodicarboxylic acid, ECV = Extracellular volume, EMB = Endomyocardial biopsy, FDG = Fluorodeoxyglucose, GCS = Global circumferential strain, GLS = Global longitudinal strain, HMDP = Hydroxymethylene diphosphonate, HS = High-sensitivity, IVS = Interventricular septum, LVH = Left ventricular hypertrophy, MGUS = Monoclonal gammopathy of undetermined significance, PET = Positron emission tomography, PYP = Pyrophosphate, SCD = Sudden cardiac death, SPECT = Single-photon emission computed tomography

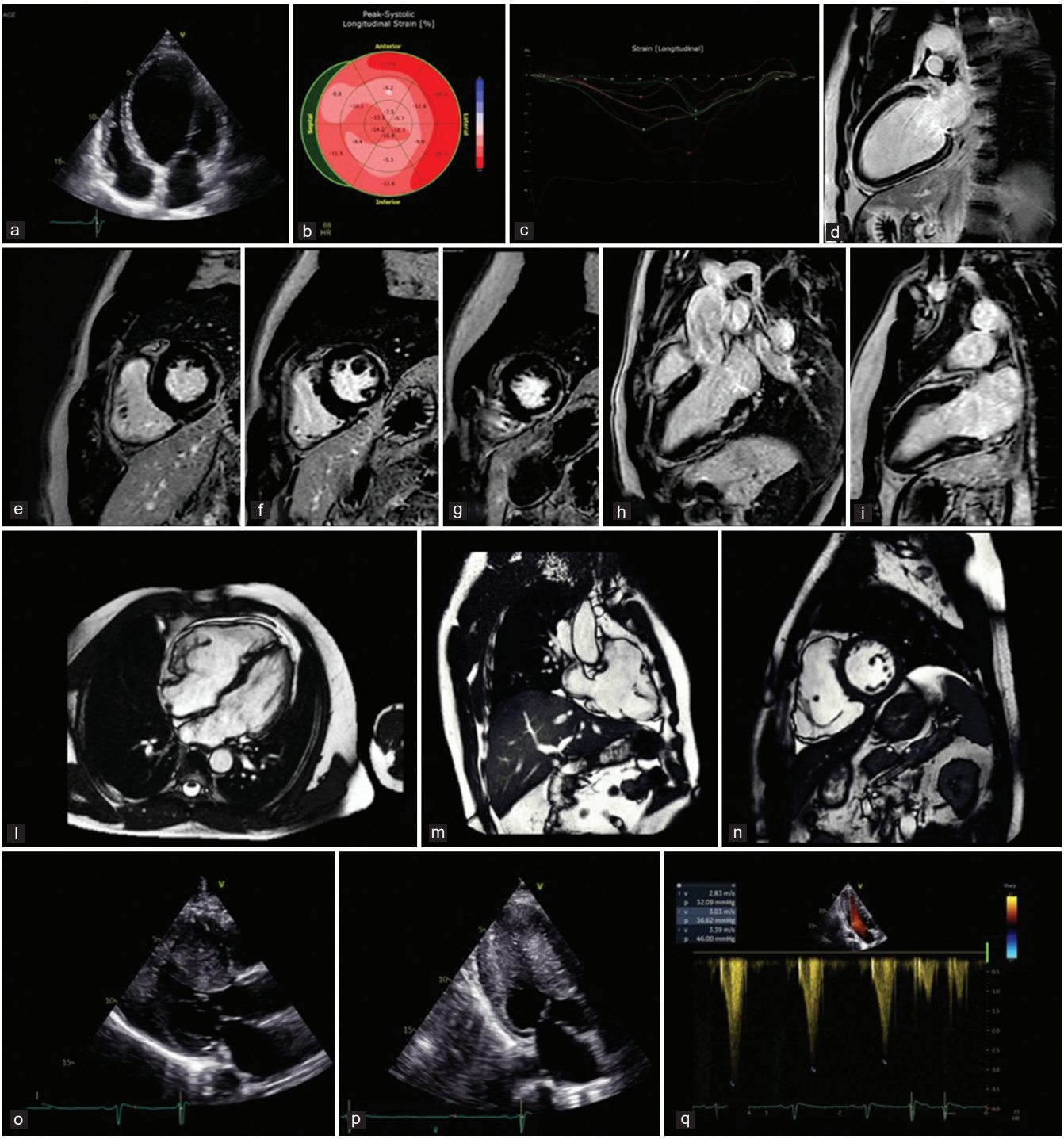



**Figure 2:** Sudden cardiac death risk stratification in the setting of cardiomyopathies, first section. (a-d) Case of dilated cardiomyopathy in a patient with a pathogenic mutation in the FLNC gene; (a) transthoracic echocardiography, apical four-chamber view: dilatation of the left ventricle with severe systolic dysfunction; (b) reduced global longitudinal strain; (c) increased mechanical dispersion; (d) cardiac magnetic resonance, postcontrast imaging, showing presence of late gadolinium enhancement at the level of the inferior wall. (e-i) Case of non-dilated left ventricular cardiomyopathy with preserved left ventricular systolic function in a patient with a pathogenic mutation in DSP gene; cardiac magnetic resonance, postcontrast imaging, shows the presence of extensive late gadolinium enhancement in the inferior, posterior, and lateral walls. (l-n) Case of arrhythmogenic right ventricular cardiomyopathy in a patient with a pathogenic mutation in the PKP2 gene; cardiac magnetic resonance, cine steady state free precession imaging, showing dilatation of the right ventricle with severe systolic dysfunction, along with the presence of large aneurysms in the sub-tricuspidal region and the right ventricular outflow tract. (o-q) Case of obstructive hypertrophic cardiomyopathy; (o-p) transthoracic echocardiography, showing the presence of severe hypertrophy of the interventricular septum and left ventricular outflow tract obstruction due to systolic anterior motion of the mitral valve; (q) transthoracic echocardiography, continuous wave spectral Doppler: evidence of significant left ventricular outflow tract peak systolic gradient.

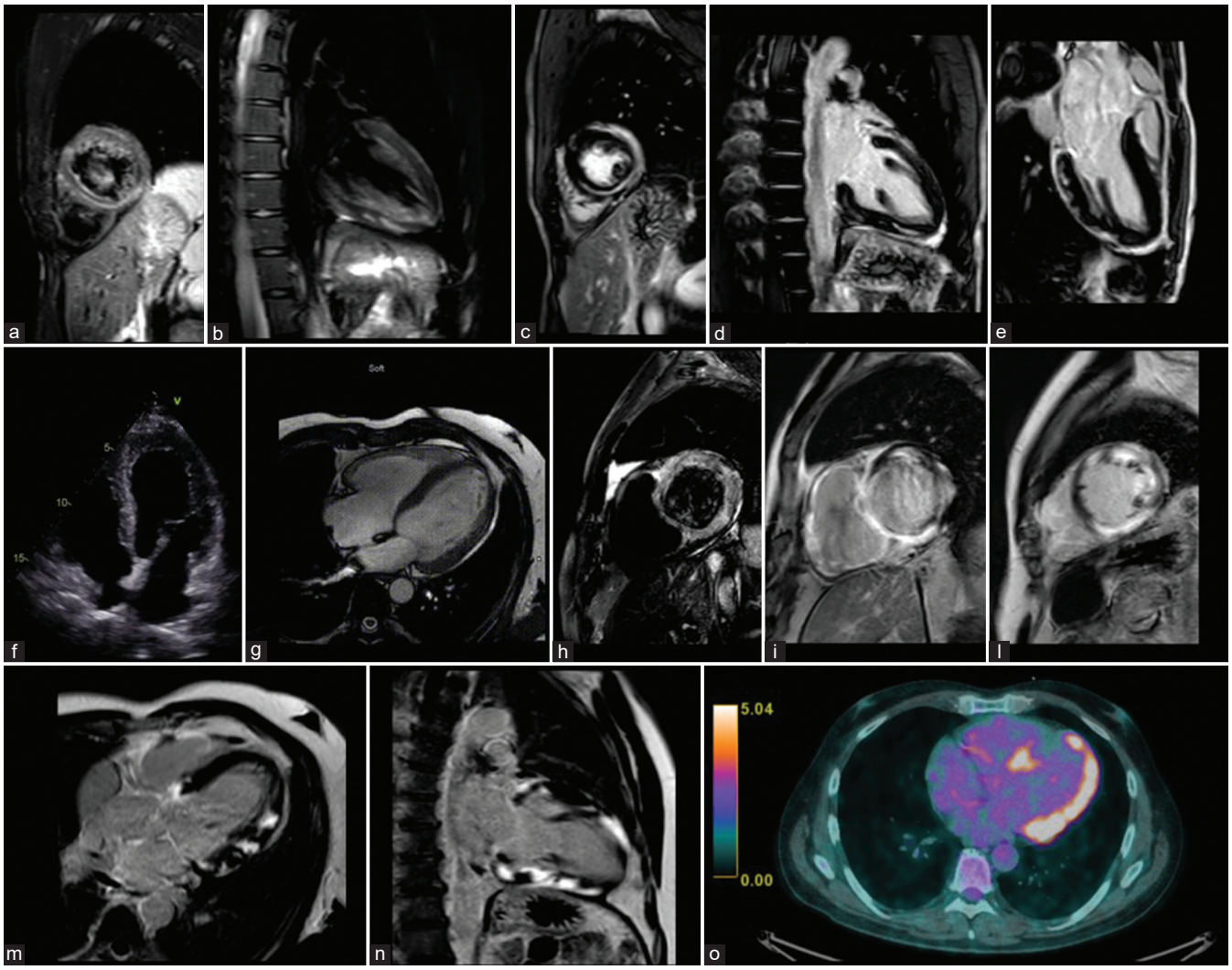


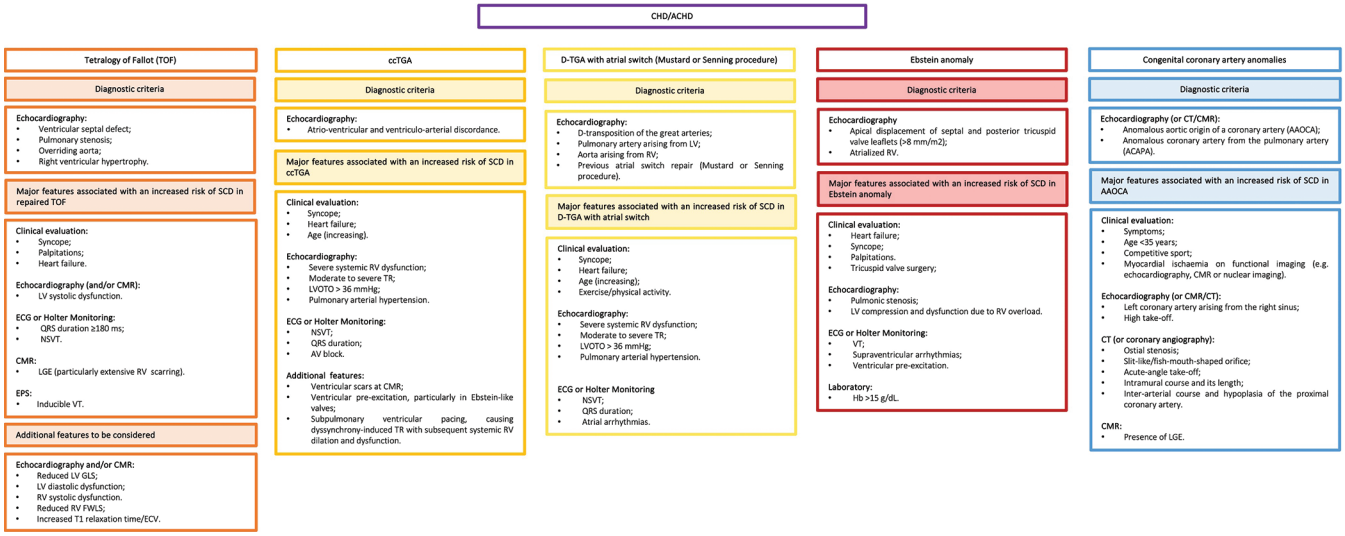

**Figure 3:** Sudden cardiac death risk stratification in the setting of cardiomyopathies, second section. (a-e) Case of myocarditis in a patient presenting with chest pain; (a-b) cardiac magnetic resonance, $T_2$-weighted short-tau inversion recovery imaging, showing signal hyperintensity of the interventricular septum and inferior wall, consistent with the presence of myocardial edema; (c-e) cardiac magnetic resonance, postcontrast imaging, showing extensive areas of intramyocardial and subepicardial late gadolinium enhancement in the same regions.(f-o) Case of cardiac sarcoidosis; (f) transthoracic echocardiography, apical four-chamber view: dilatation and severe systolic dysfunction of the left ventricle; a thinning of the basal interventricular septum can be observed; (g) cardiac magnetic resonance, cine steady state-free precession imaging, confirming the echocardiographic findings; (h) cardiac magnetic resonance, $T_2$-weighted short-tau inversion recovery imaging, showing areas of increased signal intensity due to the presence of myocardial edema; (i-n) cardiac magnetic resonance, postcontrast imaging, showing extensive biventricular late gadolinium enhancement with mixed pattern and the typical involvement of the interventricular septum; (o) positron emission tomography, showing high $^{18}$F-fluorodeoxyglucose uptake of the myocardium.

## Congenital Heart Disease

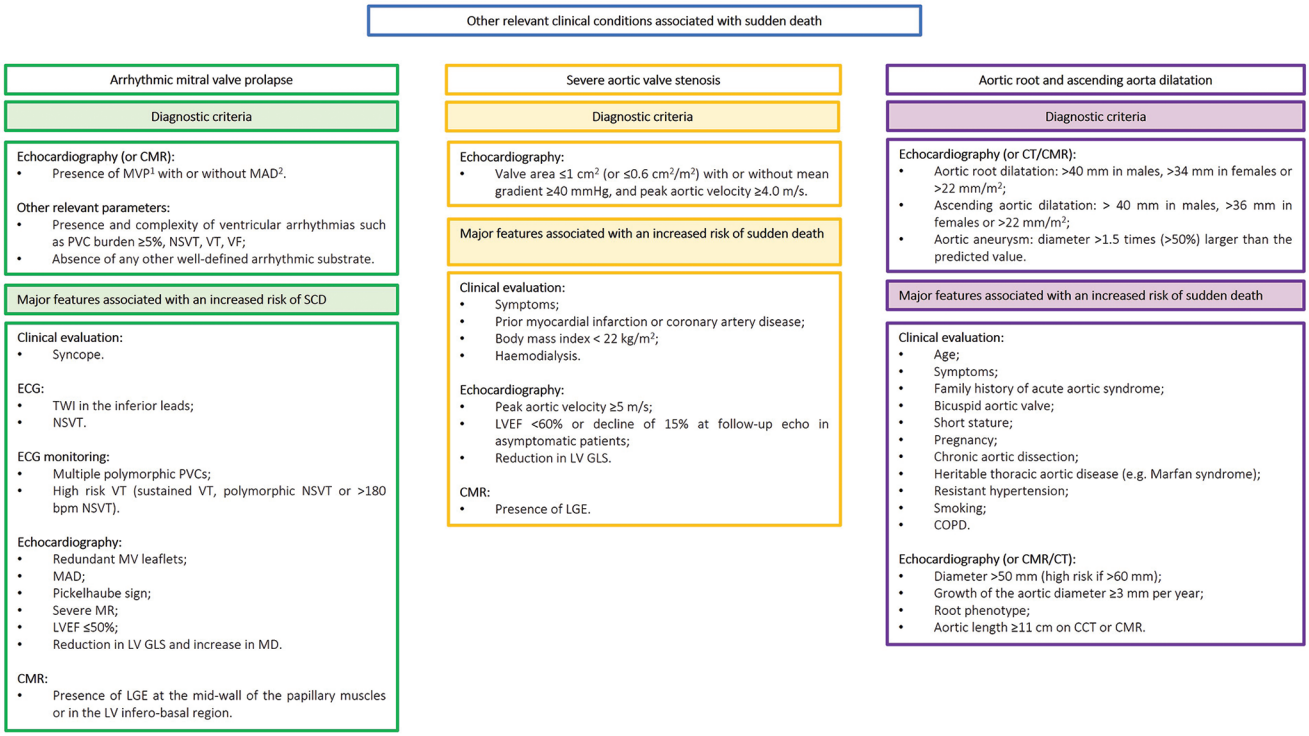
Several studies have consistently demonstrated higher rates of SCD among adults with congenital heart disease compared to the general population.[21] Patients with repaired tetralogy of Fallot are at increased risk of SCD, primarily due to ventricular arrhythmias. In this context, imaging modalities play a crucial role, and particular attention should be given to left ventricular dysfunction and biventricular scarring.[22] Transposition of the great arteries (both congenitally corrected and dextro-transposition of the great arteries following atrial switch repair) is another relevant condition strongly associated with SCD. In these scenario, systemic right ventricular dysfunction and moderate-to-severe tricuspid regurgitation are significantly associated with higher risk of events.[23] Ebstein anomaly and congenital abnormalities of coronary arteries are also linked to increased risk of SCD.[24,25] Finally, in patients with Eisenmenger physiology or incompletely palliated congenital heart disease, arrhythmic risk stratification should be evaluated case by case [Flow Chart 3].

## Other Relevant Clinical Conditions Associated with Increased Risk of Sudden Death

Echocardiography is the primary imaging modality for evaluating valvular heart disease.[26] In recent years, mitral




**Flow Chart 3:** Key factors associated with an increased risk of sudden cardiac death in congenital heart disease. AAOCA = Anomalous aortic origin of a coronary artery, ACAPA = Anomalous coronary artery from the pulmonary artery, AV = Atrio-ventricular, ccTGA = Congenitally corrected transposition of the great arteries, D-TGA = Dextro-transposition of the great arteries, CMR = Cardiac magnetic resonance, CT = Computed tomography, ECG = Electrocardiogram, FWLS = Free wall longitudinal strain, GLS = Global longitudinal strain, HB = Haemoglobin, LV = Left ventricular, LVEF = Left ventricular ejection fraction, LVOTO = Left ventricular outflow tract obstruction, LGE = Late gadolinium enhancement, NSVT = Non-sustained ventricular tachycardia, RV = Right ventricular, SCD = Sudden cardiac death, TOF = Tetralogy of Fallot, TR = Tricuspid regurgitation, VT = Ventricular tachycardia




1. MVP: systolic displacement of one or both mitral leaflets ≥2 mm above the plane of the mitral annulus in the sagittal view.
2. MAD: clear separation between the ventricular myocardium and the mitral annulus supporting the posterior mitral leaflet, in both diastole and systole.

* Pseudo-MAD: apparent separation between the posterior mitral valve leaflet and the left ventricular myocardium during systole.

**Flow Chart 4:** Key factors associated with an increased risk of sudden death in other clinical conditions. CMR = Cardiac magnetic resonance, CT = Computed tomography, COPD = Chronic obstructive pulmonary disease, ECG = Electrocardiogram, GLS = Global longitudinal strain, LV = Left ventricular, LVEF = Left ventricular ejection fraction, LGE = Late gadolinium enhancement, MAD = Mitral annular disjunction, MD = Mechanical dispersion, MVP = Mitral valve prolapse, MR = Mitral regurgitation, MV = Mitral valve, NSVT = Non-sustained ventricular tachycardia, PVC = Premature ventricular contractions, SCD = Sudden cardiac death, TWI = T wave inversion, VF = Ventricular fibrillation, VT = Ventricular tachycardia

**Table 3: Major echocardiographic features associated with an increased risk of sudden cardiac death in different clinical conditions**

| |
|---|
| Acute coronary syndromes |
| LVEF ≤40% |
| RV dysfunction |
| Ischemic MR |
| Pericardial effusion |
| Chronic ischemic heart disease |
| LVEF ≤35% despite ≥3 months of OMT in NYHA II-III class |
| LVEF ≤30% despite ≥3 months of OMT |
| LVEF ≤40% despite ≥3 months of OMT, if NSVT and inducibility of sustained monomorphic VT by PES |
| DCM |
| LVEF ≤35% after ≥3 months of OMT |
| LVEF <50% if high-risk genotype and ≥2 additional risk factors (i.e. syncope, LGE, and inducible sustained monomorphic VT at PES) |
| LVEF <50% if LMNA mutation |
| NDLVC |
| LVEF ≤35% after ≥3 months of OMT |
| LVEF <50% if high-risk genotype and additional risk factors (i.e. syncope, LGE, and inducible sustained monomorphic VT at PES) |
| ARCV |
| RV fractional area change ≤17% |
| LVEF <45% |
| HCM |
| Maximum LV wall thickness |
| Left atrial diameter |
| LV outflow tract gradient |
| RCM |
| Impaired diastolic function |
| Reduced LV systolic function |
| LA dilatation |
| Anderson-fabry disease |
| Increased LV wall thickness |
| Increased LV mass |
| TOF |
| LV and RV systolic dysfunction |
| LV diastolic dysfunction |
| Reduced LV GLS and RV FWLS |
| ccTGA and D-TGA |
| Severe systemic RV dysfunction |
| Moderate to severe TR |
| LVOTO >36 mmHg |
| PAH |
| Ebstein anomaly |
| Pulmonic stenosis |
| LV dysfunction |
| Congenital anomalies of the origin of a coronary |
| Left coronary artery arising from the right sinus |
| High take-off |
| Arrhythmic mitral valve prolapse |
| Redundant MV leaflets |
| MAD |
| Pickelhaube sign |
| Severe MR |
| LVEF ≤50% |
| Severe aortic valve stenosis |
| Peak aortic velocity ≥5 m/s |
| LVEF <60% or decline of 15% at follow-up ECHO in asymptomatic patients |
| Aortic root and ascending aorta dilatation |
| Diameter >50 mm (high risk if >60 mm) |
| Growth of the aortic diameter ≥3 mm per year |
| Root phenotype |

ccTGA=Congenitally corrected transposition of the great arteries, D-TGA=Dextro-transposition of the great arteries, FWLS=Free wall longitudinal strain, GLS=Global longitudinal strain, LA=Left atrial, LGE=Late gadolinium enhancement, LMNA=Lamin A/C, LV=Left ventricular, LVEF=Left ventricular ejection fraction, LVOTO=Left ventricular outflow tract obstruction, MAD=Mitral annular disjunction, MR=Mitral regurgitation, MV=Mitral valve, NSVT=Nonsustained ventricular tachycardia, NYHA=New York Heart Association, OMT=Optimal medical therapy, PAH=Pulmonary arterial hypertension, PES=Programmed electrical stimulation, RV=Right ventricular, TR=Tricuspid regurgitation, VT=ventricular tachycardia, HCM=Hypertrophic cardiomyopathy, NDLVC=Nondilated left ventricular cardiomyopathy, TOF=Tetralogy of Fallot, ARCV=Arrhythmogenic right ventricular cardiomyopathy, DCM=Dilated cardiomyopathy, RCM=Restrictive cardiomyopathy, ECHO=Echocardiography

valve prolapse has garnered significant attention due to its potential association with an increased risk of SCD, primarily linked to ventricular arrhythmias, both in patients with and without severe mitral regurgitation.[27] Aortic stenosis is an increasingly prevalent valvular disease, largely driven by population aging. Echocardiography remains the cornerstone for both diagnosis and risk stratification.[26] Dilatation of the aortic root and ascending aorta is another condition that may be associated with sudden death. Therefore, echocardiographic evaluation of the ascending aorta is essential for early detection and risk stratification [Flow Chart 4].[28]

## Conclusions

SCD remains a major clinical and public health concern. Identifying at-risk patients is challenging and requires a multiparametric approach. Multimodality imaging plays a central role in risk stratification, providing comprehensive insights into biventricular function and detecting tissue abnormalities. Echocardiography serves as the first-line imaging modality for patient assessment, offering precise diagnostic and risk stratification insights.

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Nil.

### Conflicts of interest

There are no conflicts of interest.

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