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

Antonio De Luca*, Jacopo Giulio Rizzi, Thomas Caiffa[1], Massimo Zecchin, Matteo Cameli[2]*, Eustachio Agricola[3]*, Maurizio Cusmà Piccione[4]*, Roberta Manganaro[4]*, Andrea Barison[5], Gianluca Di Bella[4], Gianfranco Sinagra, Concetta Zito[4]*

Cardiothoracovascular Department, Division of Cardiology, Azienda Sanitaria Universitaria Giuliano Isontina and University of Trieste, [1]Department of Paediatrics, Institute for Maternal and Child Health IRCCS 'Burlo Garofolo', Trieste, [2]Department of Medical Biotechnologies, Division of Cardiology, University of Siena, Siena, [3]Unit of Cardiovascular Imaging, IRCCS San Raffaele Hospital, Milan, [4]Department of Clinical and Experimental Medicine, University of Messina, Messina, [5]Cardiothoracic Department, Fondazione Toscana Gabriele Monasterio and Scuola Superiore Sant'Anna, Pisa, [6]Department of Women's and Children's Health, Pediatric Cardiology Unit, University Hospital of Padua, Padua, [7]Cardiology Unit, Infermi Hospital, Rimini, Italy

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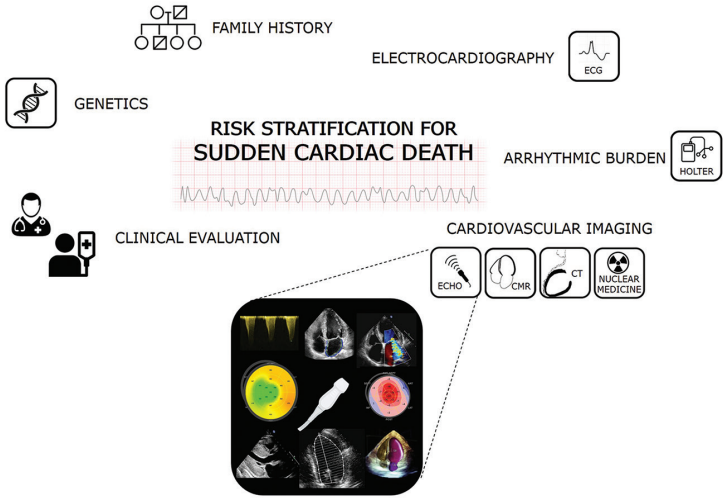
Graphical Abstract



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

Address for correspondence: Dr. Antonio De Luca, Cardiothoracovascular Department, Division of Cardiology, Ospedale di Cattinara - Polo Cardiologico, Via Valdoni 7, Trieste 34149, Italy.
E-mail: deluca.antonio.md@gmail.com

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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

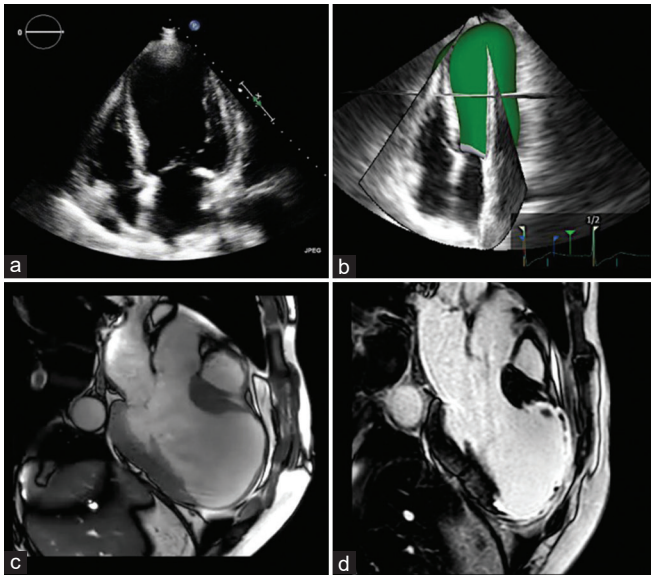


Figure 1: Case of ischemic heart disease in a patient hospitalized for subacute myocardial infarction. (a) Transthoracic echocardiography, apical four-chamber view: dilatation and severe systolic dysfunction of the left ventricle, along with a large apical aneurysm, which is associated with a higher 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

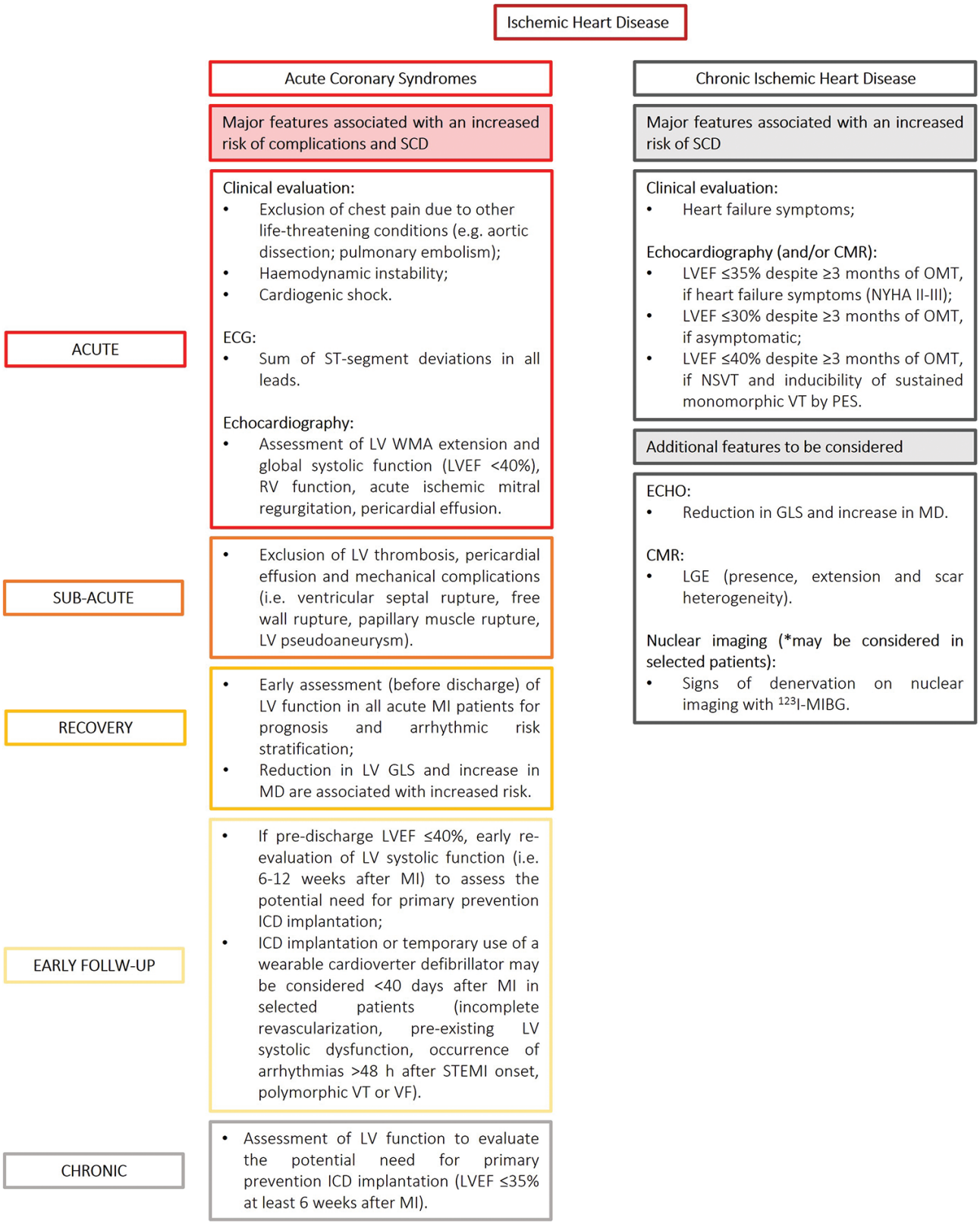
Ischemic Heart Disease

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]

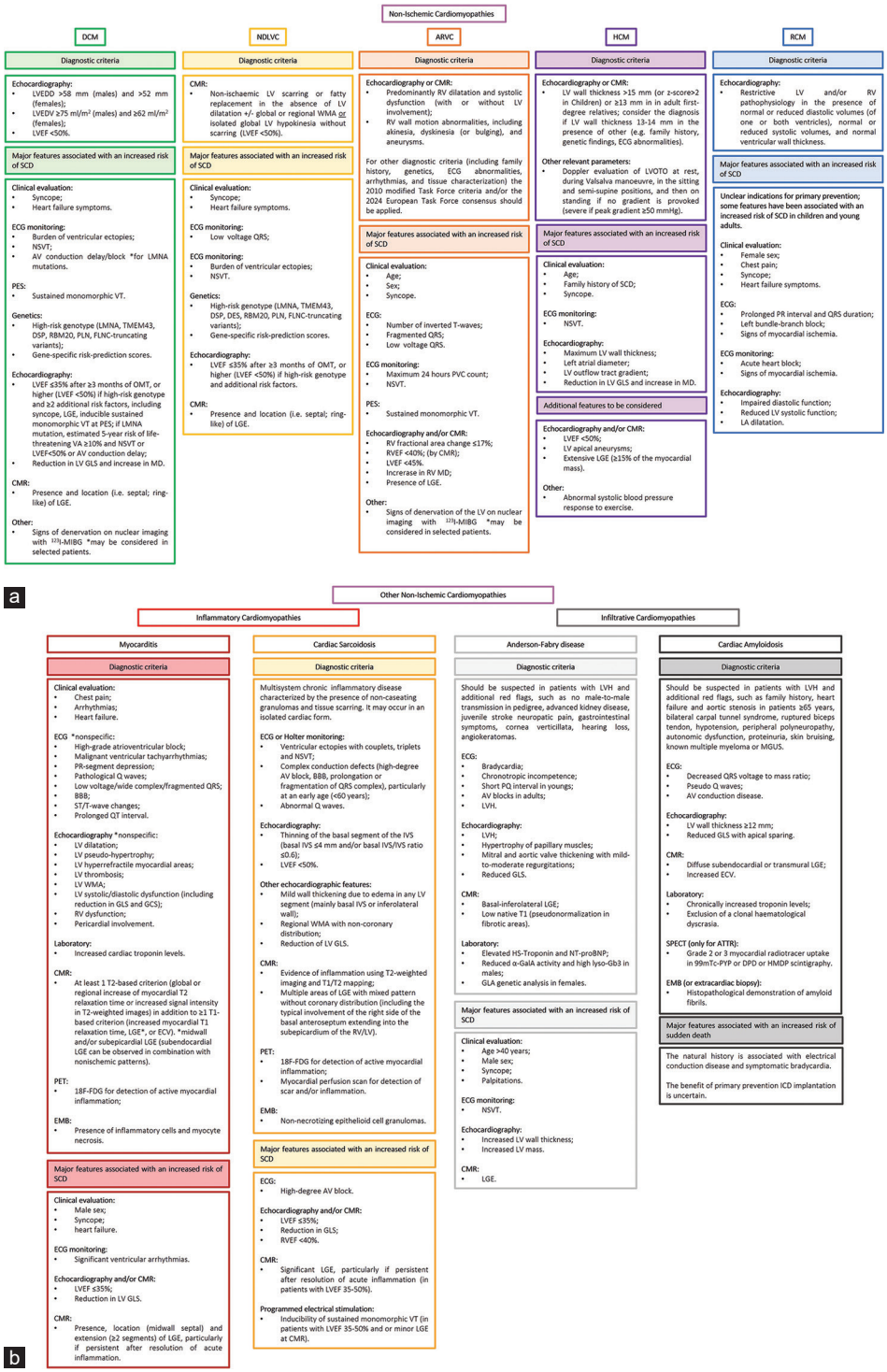
Cardiomyopathies

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

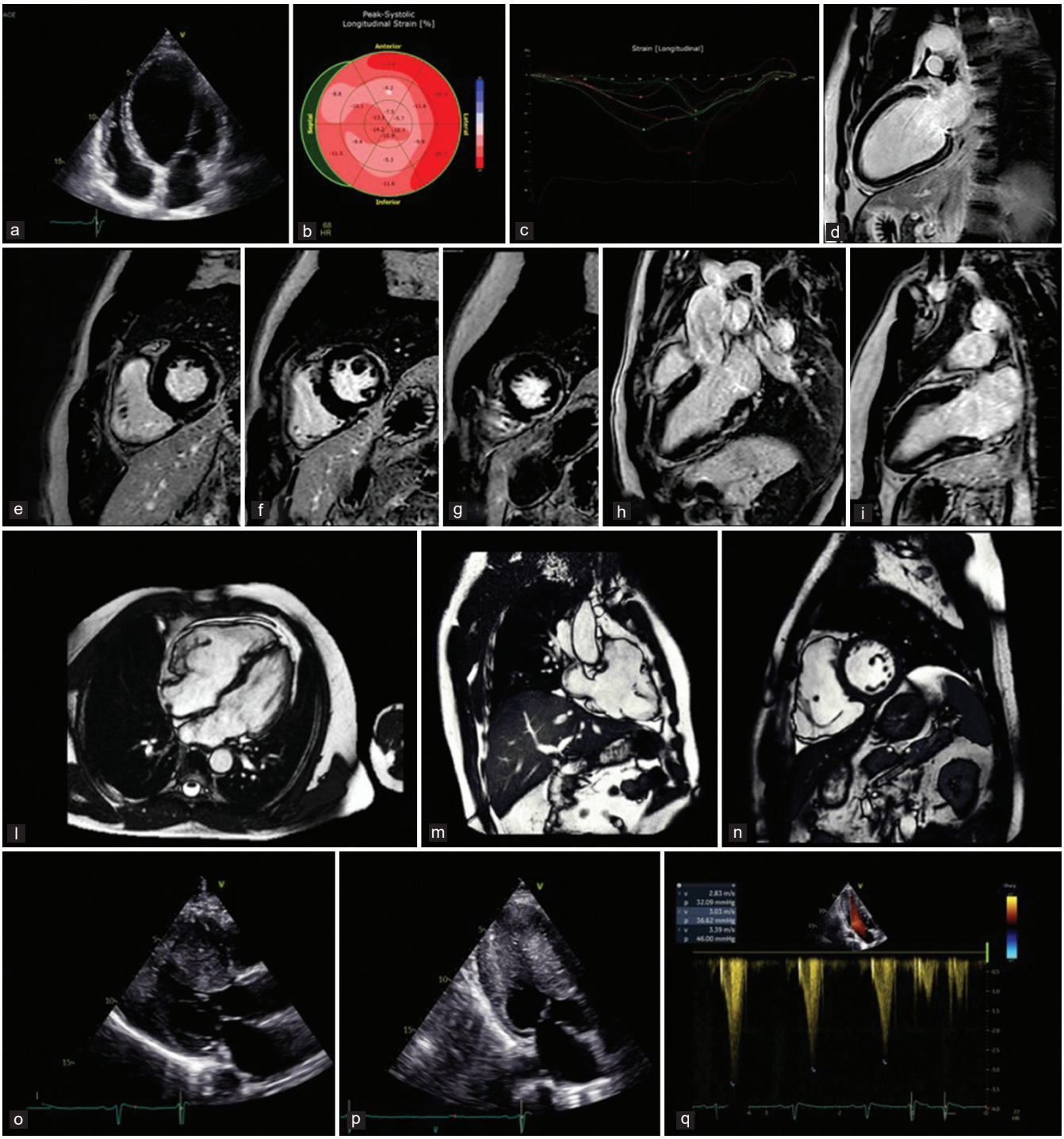


Figure 2: Sudden cardiac death risk stratification in the setting of cardiomyopathies, first section. (a-d) Case of dilated cardiomyopathy in a patient with a pathogenic mutation in the FLNC gene; (a) transthoracic echocardiography, apical four-chamber view: dilatation of the left ventricle with severe 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.

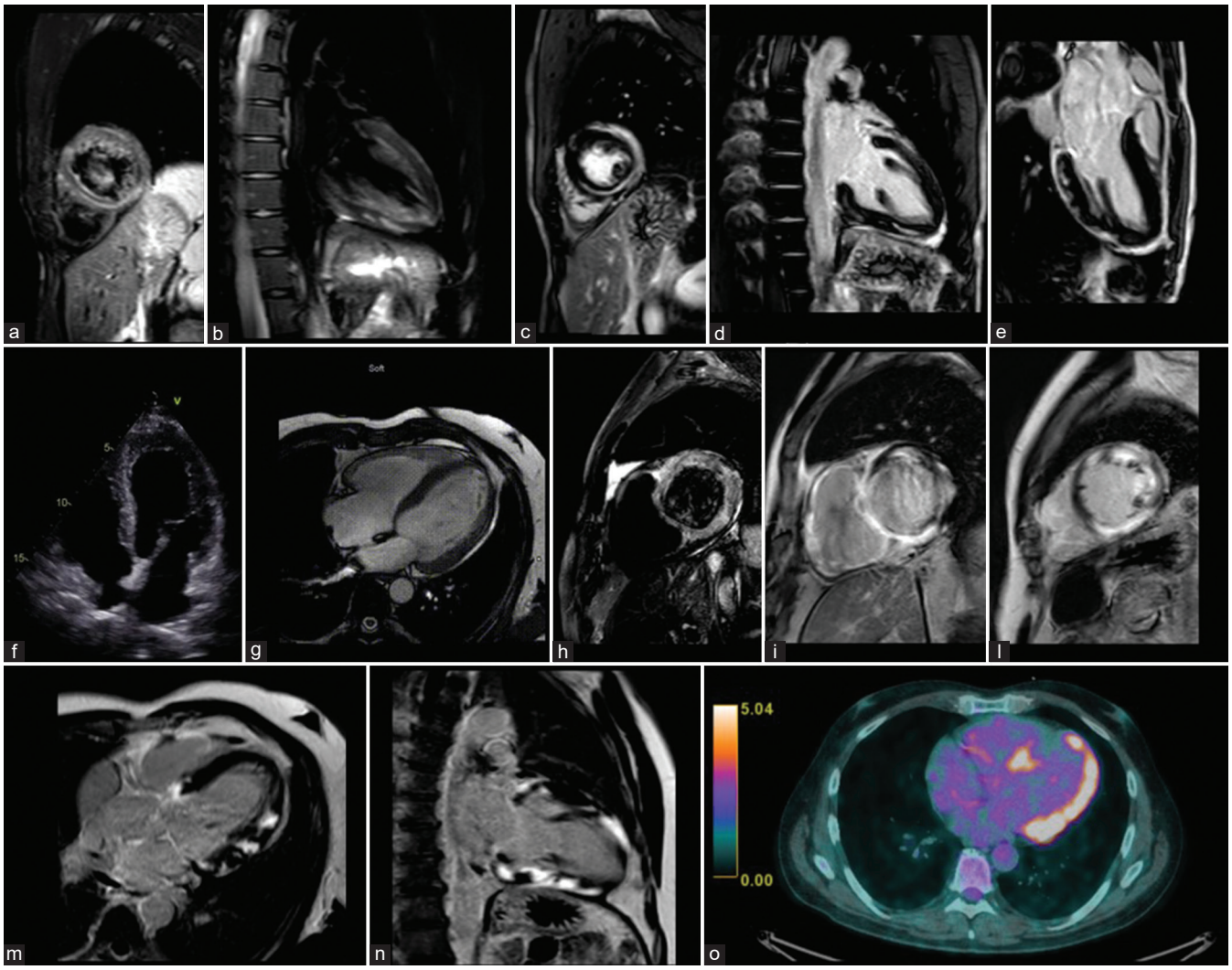


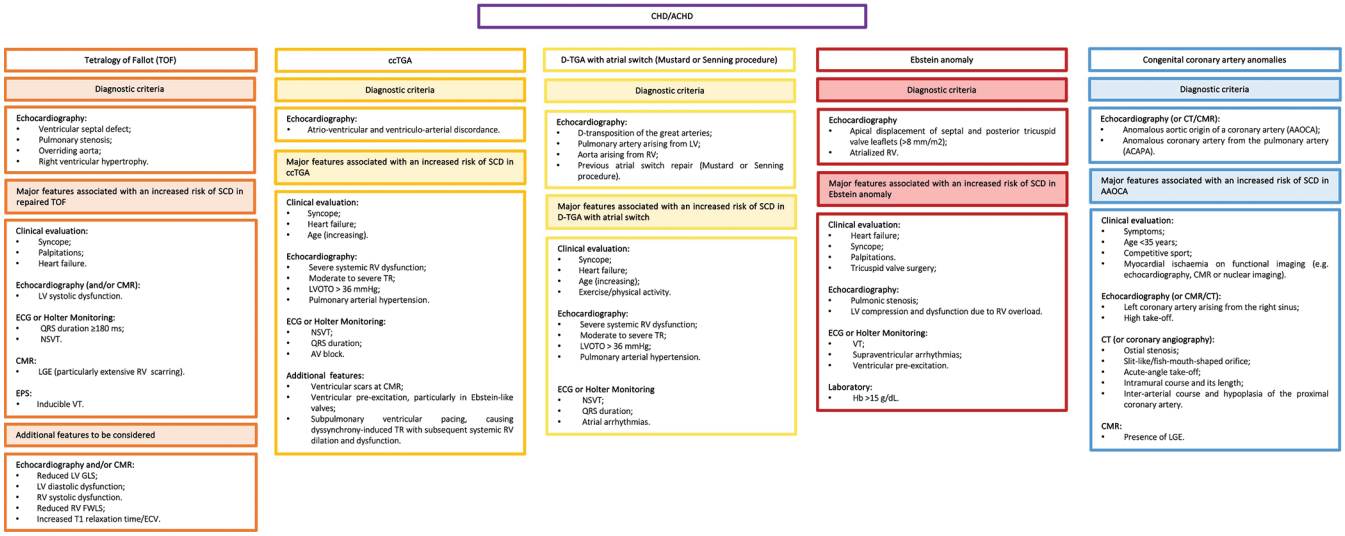
Figure 3: Sudden cardiac death risk stratification in the setting of cardiomyopathies, second section. (a-e) Case of myocarditis in a patient presenting with chest pain; (a-b) cardiac magnetic resonance, T_2-weighted short-tau inversion recovery imaging, showing signal hyperintensity of the interventricular 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

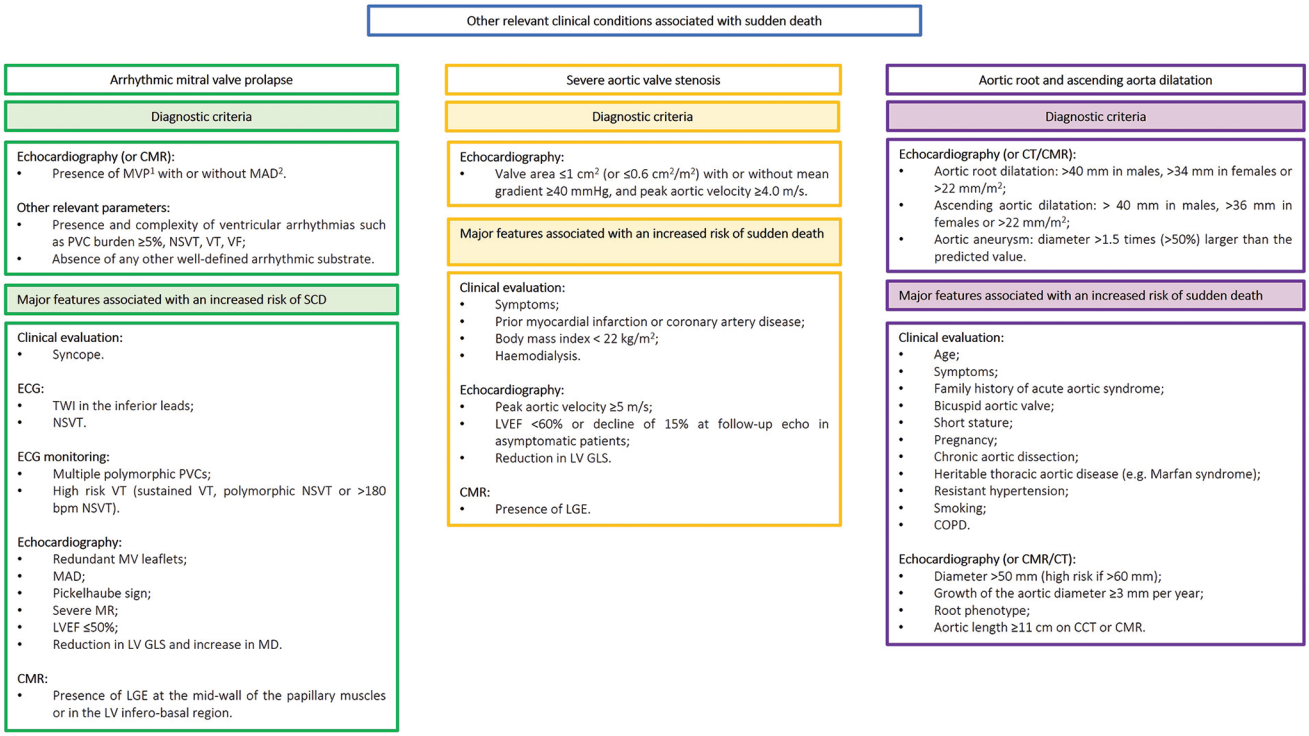
Several studies have consistently demonstrated higher rates of SCD among adults with congenital heart disease compared to the general population.[21] Patients with repaired tetralogy of Fallot are at increased risk of SCD, primarily due to ventricular arrhythmias. In this context, imaging modalities 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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Conflicts of interest

There are no conflicts of interest.

References

1. Zeppenfeld K, Tfelt-Hansen J, de Riva M, Winkel BG, Behr ER, Blom NA, *et al.* 2022 ESC guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. Eur Heart J 2022;43:3997-4126.
2. Arbelo E, Protonotarios A, Gimeno JR, Arbustini E, Barriales-Villa R,

Basso C, *et al.* 2023 ESC guidelines for the management of cardiomyopathies. Eur Heart J 2023;44:3503-626.
3. Barletta V, Fabiani I, Lorenzo C, Nicastro I, Bello VD. Sudden cardiac death: A review focused on cardiovascular imaging. J Cardiovasc Echogr 2014;24:41-51.
4. Smiseth OA, Rider O, Cvijic M, Valkovič L, Remme EW, Voigt JU. Myocardial strain imaging: theory, current practice, and the future. JACC Cardiovasc Imaging 2025;18:340-81.
5. Porcari A, De Luca A, Grigoratos C, Biondi F, Faganello G, Vitrella G, *et al.* Arrhythmic risk stratification by cardiac magnetic resonance tissue characterization: Disclosing the arrhythmic substrate within the heart muscle. Heart Fail Rev 2022;27:49-69.
6. Faganello G, Porcari A, Biondi F, Merlo M, De Luca A, Vitrella G, *et al.* Cardiac magnetic resonance in primary prevention of sudden cardiac death. J Cardiovasc Echogr 2019;29:89-94.
7. Zwart B, Claessen BE, Damman P, Woudstra P, Vink MA, Balder JW, *et al.* 2023 European Society of Cardiology guidelines for the management of acute coronary syndromes: Statement of endorsement by the NVVC. Neth Heart J 2024;32:338-45.
8. Vrints C, Andreotti F, Koskinas KC, Rossello X, Adamo M, Ainslie J, *et al.* 2024 ESC guidelines for the management of chronic coronary syndromes. Eur Heart J 2024;45:3415-537.
9. Gimelli A, Liga R, Agostini D, Bengel FM, Ernst S, Hyafil F, *et al.* The role of myocardial innervation imaging in different clinical scenarios: An expert document of the European Association of Cardiovascular Imaging and Cardiovascular Committee of the European Association of Nuclear Medicine. Eur Heart J Cardiovasc Imaging 2021;22:480-90.
10. McDonagh TA, Metra M, Adamo M, Gardner RS, Baumbach A, Böhm M, *et al.* 2021 ESC guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J 2021;42:3599-726.
11. Corrado D, Anastasakis A, Basso C, Bauce B, Blomström-Lundqvist C, Bucciarelli-Ducci C, *et al.* Proposed diagnostic criteria for arrhythmogenic cardiomyopathy: European Task Force consensus report. Int J Cardiol 2024;395:131447.
12. Aquaro GD, De Luca A, Cappelletto C, Raimondi F, Bianco F, Botto N, *et al.* Prognostic value of magnetic resonance phenotype in patients with arrhythmogenic right ventricular cardiomyopathy. J Am Coll Cardiol 2020;75:2753-65.
13. Castrichini M, De Luca A, De Angelis G, Neves R, Paldino A, Dal Ferro M, *et al.* Magnetic resonance imaging characterization and clinical outcomes of dilated and arrhythmogenic left ventricular cardiomyopathies. J Am Coll Cardiol 2024;83:1841-51.
14. Radmilovic J, D'Andrea A, D'Amato A, Tagliamonte E, Sperlongano S, Riegler L, *et al.* Echocardiography in athletes in primary prevention of sudden death. J Cardiovasc Echogr 2019;29:139-48.
15. Filomena D, Vandenberk B, Dresselaers T, Willems R, Masci PG, Robyns T, *et al.* Cardiac diagnoses and long-term outcomes in ring-like late gadolinium enhancement evaluated by cardiac magnetic resonance. Eur Heart J Cardiovasc Imaging 2025;26:841-52.
16. Writing Committee, Drazner MH, Bozkurt B, Cooper LT, Aggarwal NR, Basso C, *et al.* 2024 ACC expert consensus decision pathway on strategies and criteria for the diagnosis and management of myocarditis: A report of the American College of Cardiology solution set oversight committee. J Am Coll Cardiol 2025;85:391-431.
17. Sharma R, Kouranos V, Cooper LT, Metra M, Ristic A, Heidecker B, *et al.* Management of cardiac sarcoidosis. Eur Heart J 2024;45:2697-726.
18. Aquaro GD, Perfetti M, Camastra G, Monti L, Dellegrottaglie S, Moro C, *et al.* Cardiac MR with late gadolinium enhancement in acute myocarditis with preserved systolic function: ITAMY study. J Am Coll Cardiol 2017;70:1977-87.
19. Aquaro GD, Ghebru Habtemicael Y, Camastra G, Monti L, Dellegrottaglie S, Moro C, *et al.* Prognostic value of repeating cardiac magnetic resonance in patients with acute myocarditis. J Am Coll Cardiol 2019;74:2439-48.
20. Gentile P, Merlo M, Peretto G, Ammirati E, Sala S, Della Bella P, *et al.* Post-discharge arrhythmic risk stratification of patients with acute myocarditis and life-threatening ventricular tachyarrhythmias. Eur J Heart Fail 2021;23:2045-54.
21. Baumgartner H, De Backer J, Babu-Narayan SV, Budts W, Chessa M, Diller GP, *et al.* 2020 ESC guidelines for the management of adult congenital heart disease. Eur Heart J 2021;42:563-645.
22. Krieger EV, Zeppenfeld K, DeWitt ES, Duarte VE, Egbe AC, Haeffele C, *et al.* Arrhythmias in repaired tetralogy of fallot: A scientific statement from the American Heart Association. Circ Arrhythm Electrophysiol 2022;15:e000084.
23. Ladouceur M, Van De Bruaene A, Kauling R, Budts W, Roos-Hesselink J, Albert SV, *et al*. A new score for life-threatening ventricular arrhythmias and sudden cardiac death in adults with transposition of the great arteries and a systemic right ventricle. Eur Heart J 2022;43:2685-94.
24. Attenhofer Jost CH, Tan NY, Hassan A, Vargas ER, Hodge DO, Dearani JA, *et al.* Sudden death in patients with Ebstein anomaly. Eur Heart J 2018;39:1970-7a.
25. Bigler MR, Kadner A, Räber L, Ashraf A, Windecker S, Siepe M, *et al.* Therapeutic management of anomalous coronary arteries originating from the opposite sinus of valsalva: Current evidence, proposed approach, and the unknowing. J Am Heart Assoc 2022;11:e027098.
26. Vahanian A, Beyersdorf F, Praz F, Milojevic M, Baldus S, Bauersachs J, *et al.* 2021 ESC/EACTS guidelines for the management of valvular heart disease. Eur Heart J 2022;43:561-632.
27. Sabbag A, Essayagh B, Barrera JD, Basso C, Berni A, Cosyns B, *et al.* EHRA expert consensus statement on arrhythmic mitral valve prolapse and mitral annular disjunction complex in collaboration with the ESC council on valvular heart disease and the European Association of Cardiovascular Imaging endorsed c by the Heart Rhythm Society, by the Asia Pacific Heart Rhythm Society, and by the Latin American Heart Rhythm Society. Europace 2022;24:1981-2003.
28. Mazzolai L, Teixido-Tura G, Lanzi S, Boc V, Bossone E, Brodmann M, *et al.* 2024 ESC guidelines for the management of peripheral arterial and aortic diseases. Eur Heart J 2024;45:3538-700.