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Azərbaycan
Kardiologiya
Cəmiyyəti

**Ürək çatışmazlığı xəstələrində rutin Kardiak MRT — hər kəsə
lazımdır mı? Skar
yükü-proqnostik və terapevtik əhəmiyyəti**
***Routine Cardiac MRI in Heart Failure Patients — Is It Necessary for
Everyone? The
Prognostic and Therapeutic Significance of Scar Burden***

t.e.d., Dos. Dr. Yasmin Rüstəmovə, FESC, FACC

14/12/2025



No disclosures...

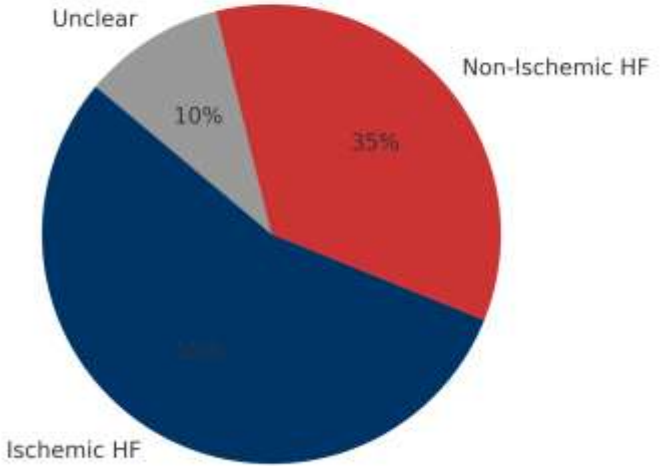




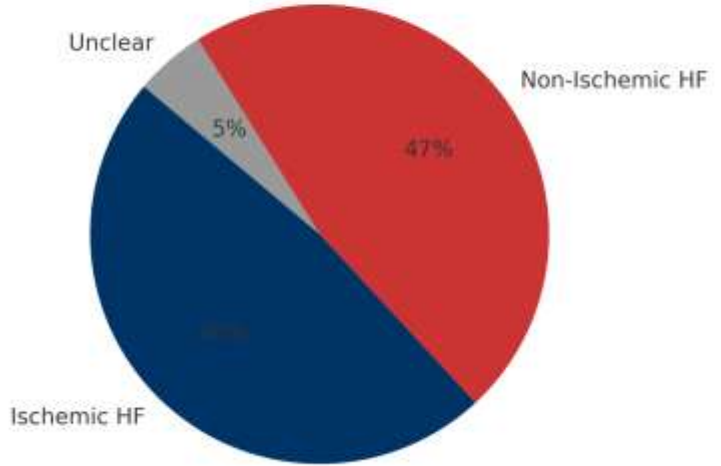
Why this topic matters

Impact of Cardiac MRI on Heart Failure Etiology Classification

Before CMR



After CMR ($\approx 15\%$ Reclassified)



Hypertrophic Cardiomyopathy

Diagnosis

- HCM is defined by an LV wall thickness ≥ 15 mm in any myocardial segment that is not explained solely by loading conditions. Lesser degrees of wall thickening (13–14 mm) require evaluation of other features including family history, genetic findings, and ECG abnormalities.

Prevalence

- 0.2% in Europe
- unexplained asymptomatic hypertrophy in young adults in the United States has been reported in the range of 1:500
- Symptomatic hypertrophy based on medical claims data has been estimated at <1:3000 adults



Hypertrophic phenocopies

Hypertensive
heart

Athlet's
heart

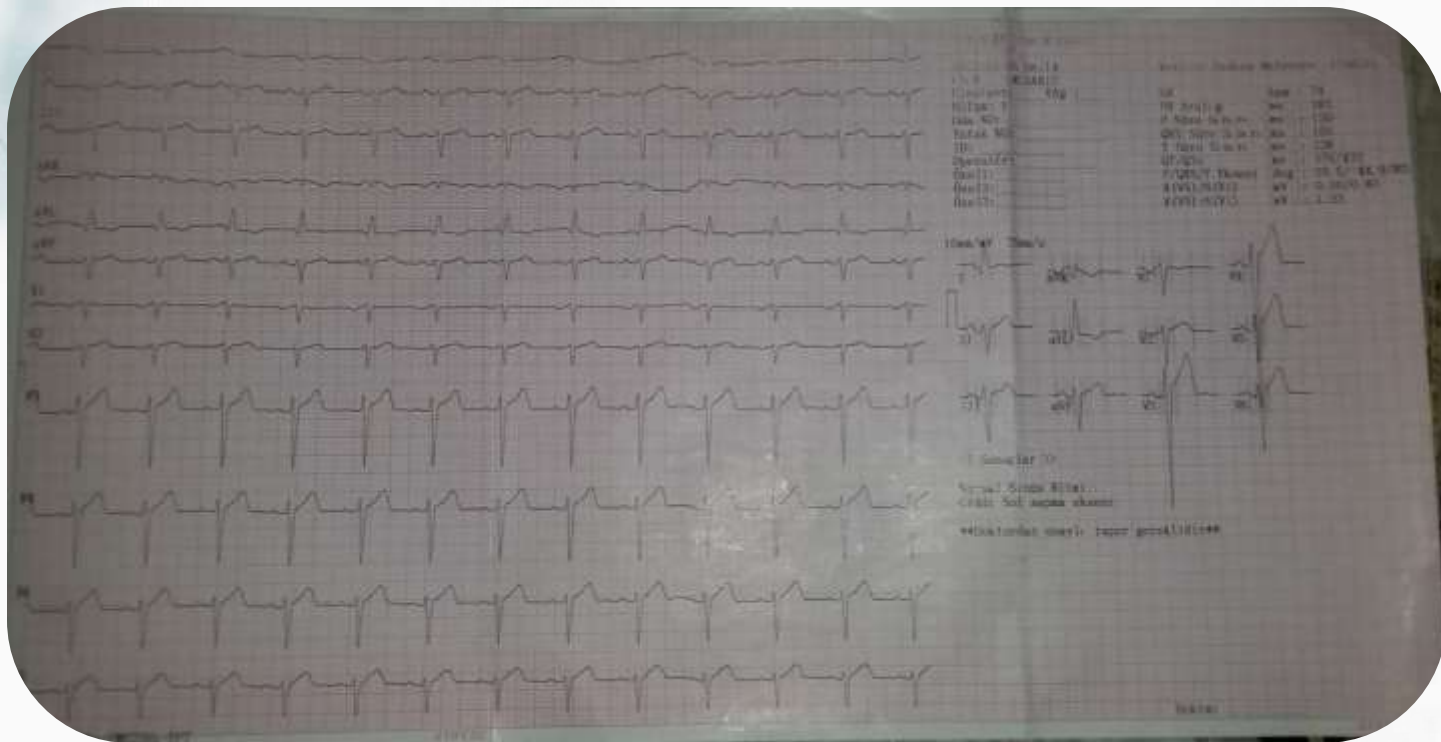
Amyloidosis

Fabry
disease &
others

Clinical Vignette

- 58 years old man, Mr M
- Palpitation, severe shortness of breath → systolic heart murmur → echocardiography → Hypertrophic cardiomyopathy
- No personal medical history, no medication
- Physical examination unremarkable, except systolic murmur (normal blood pressure, etc)
- Family history:
 - Sudden cardiac death in two brothers at 40 years but with history of cardiac disease ??
 - Cousin 43 y. : no cardiac history.
 - Further cardiac examination (ECG, Echo, MRI,)

ECG



EchoCG

LVEF=43%

LV wall thickness max 26 mm

LV PW 20 mm

LV GLS (strain): 15%

LVOT P max 94 mm Hg

P mean 47 mm Hg

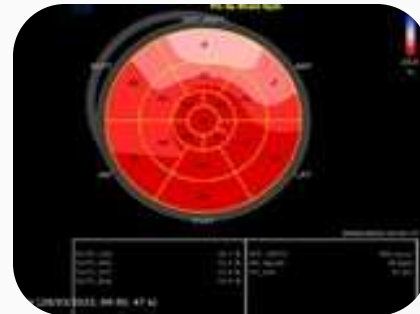
LA = 51mm,

LAVol 94 ml,

LAVI=48 ml/m²

RV thickness 7 mm

SPAP 45 mmHg



5-year risk of SCD (%) based on clinical and imaging variables

- Age
- Max LV wall thickness
- Left atrial diameter
- LVOT gradient
- Family history of SCD
- Unexplained syncope
- Non-sustained VT

Prognostic evaluation (SCD risk)

- **SCD risk score:**

- 58 y
- Echo MWT 26 mm
- Echo LA 51 mm
- Echo LVOT 94 mmHg
- Syncope : no
- nsVT: no
- Family history: yes,

Calculate
All Calculators Become a Contributor Support

Calculator About References

HCM Risk-SCD
Assess risk of sudden cardiac death and need for ICD in hypertrophic cardiomyopathy

Questions

1. Age?	58 Years
2. Maximum LV Wall Thickness?	26 mm
3. Left Atrial Size?	51 mm
4. LVOT Gradient?	94 mmHg
5. Family History of Sudden Cardiac Death?	Yes
6. Non-sustained VT?	No
7. Unexplained Syncope?	No

About

Results

Risk of Sudden Cardiac Death at 5 years
5.34%

Estimated 5-year SCD risk
ICD may be considered

- **Other possible predictors:**

- MRI: LE + and >15% LVmass

O'Mahony C ... Elliott P. Eur Heart J. 2014;35(30):2010

Calibration slope : 0.91 (95% CI: 0.74, 1.08),

C-index : 0.70 (95% CI: 0.68, 0.72)



Role of MRI: Beyond EF

- LVEF is not enough — CMR offers multiparametric insight:
 - Volumes & mass (cine SSFP)
 - Fibrosis (LGE, T1 mapping, ECV)
 - Edema/inflammation (T2/T2 mapping)
 - Perfusion (first-pass stress perfusion)
 - Tissue characterization & differential diagnosis
- **ESC 2023 HCM/DCM Recommendation:**
- *“CMR should be performed in all patients with suspected or confirmed cardiomyopathy for etiological and prognostic assessment (Class I, Level B).”*

CMR in the guidelines

Recommendation Table 5 — Recommendations for cardiac magnetic resonance indication in patients with cardiomyopathy

Recommendations	Class ^a	Level ^b
Contrast-enhanced CMR is recommended in patients with cardiomyopathy at initial evaluation. ^{10,90,116,119–143}	I	B

Continued

Risk stratification

The accuracy of prevalent risk models recommended by American Heart Association/American College of Cardiology(AHA/ACC) and European Society of Cardiology(ESC) for hypertrophic cardiomyopathy(HCM) patients is suboptimal.

ICD for primary prevention in HCMP

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more cases of SCD in HCM patients with LGE >15% of LV mass.¹²⁴ In the most recent report from the registry, there have been 24 deaths from any cause after a mean follow-up of 33.5 ± 12.4 months (median: 36 months and range 1–64 months); the relation with LGE is not reported.⁷⁹⁵ There are very limited data on the role of CMR over and above validated risk algorithms in SCD risk prediction in children with HCM.^{796,797}

On balance, the Task Force maintains the recommendation to first estimate SCD risk using the HCM-SCD Risk calculators. For patients who are in the low to intermediate risk category, the presence of extensive LGE ($\geq 15\%$) may be used in shared decision-making with patients about prophylactic ICD implantation, acknowledging the lack of robust data on the impact of scar quantification on the personalized risk estimates generated by the HCM-SCD Risk calculators.

7.1.5.4. Abnormal exercise blood pressure response

Approximately one-third of adult patients with HCM have an abnormal systolic blood pressure response to exercise characterized by progressive hypotension or a failure to augment the systolic blood pressure that is caused by an inappropriate drop in systemic vascular resistance

urheartj/advance-article/doi/10.1093/eurheartj/ehad194/724660

Limitations

- Selection Bias

Large multicenter registries such as **HCMR** often include patients with **mild symptoms and a relatively favorable prognosis**

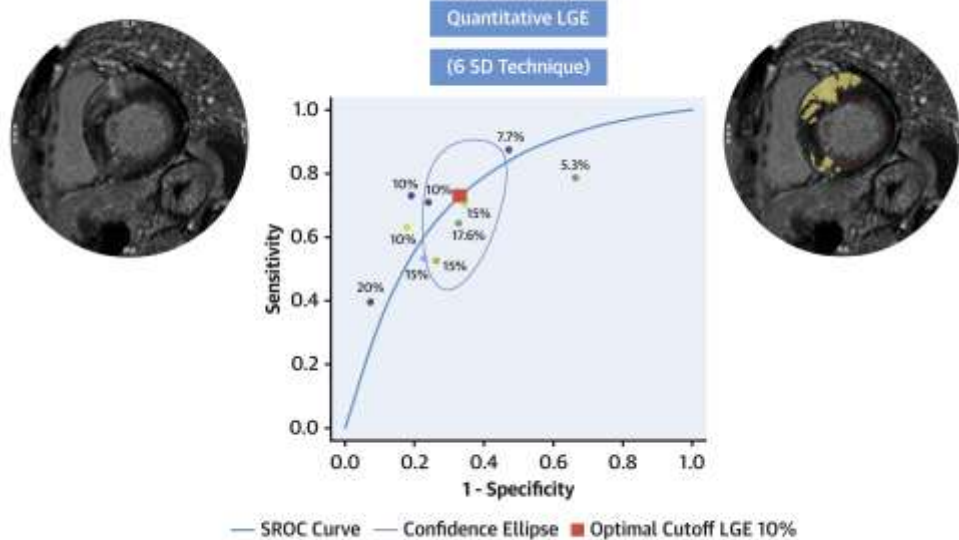
- Referral Bias

In contrast, cohorts from **specialized referral centers** are generally “**sicker**” — showing higher LVOT gradients, greater fibrosis burden, and multiple risk factors. This leads to a **higher observed risk** and different outcome proportions compared to multicenter registry data.

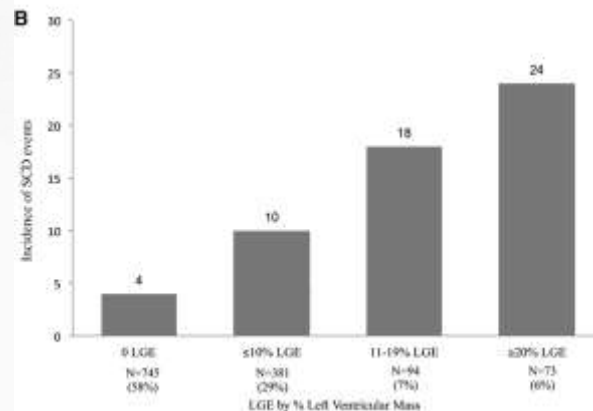
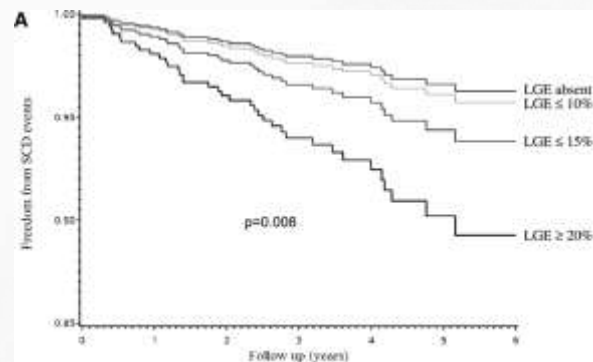
LGE -remains some debate about the methods used to quantify LGE with the 2-standard deviation technique; the only one that is validated against necropsy

LGE predicting SCD

CENTRAL ILLUSTRATION: Prognostic Value of LGE% in Predicting SCD in HCM



Kiaos A, et al. *J Am Coll Cardiol Img.* 2024;17(5):489-497.



Kiaos, A.; Daskalopoulos, G.N.; Kamperidis, V.; Ziakas, A.; Efthimiadis, G.; Karamitsos, T.D. Quantitative Late Gadolinium Enhancement Cardiac Magnetic Resonance and Sudden Death in Hypertrophic Cardiomyopathy: A Meta-Analysis. *JACC Cardiovasc. Imaging* 2024, 17, 489–497.

Chan RH, Maron BJ, Olivetto I, Pencina MJ, Assenza GE, et al Prognostic value of quantitative contrast-enhanced cardiovascular magnetic resonance for the evaluation of sudden death risk in patients with hypertrophic cardiomyopathy. *Circulation.* 2014 Aug 5;130(6):484-95. doi: 10.1161/CIRCULATIONAHA.113.007094. PMID: 25092278.

ICD for primary prevention in HCMP

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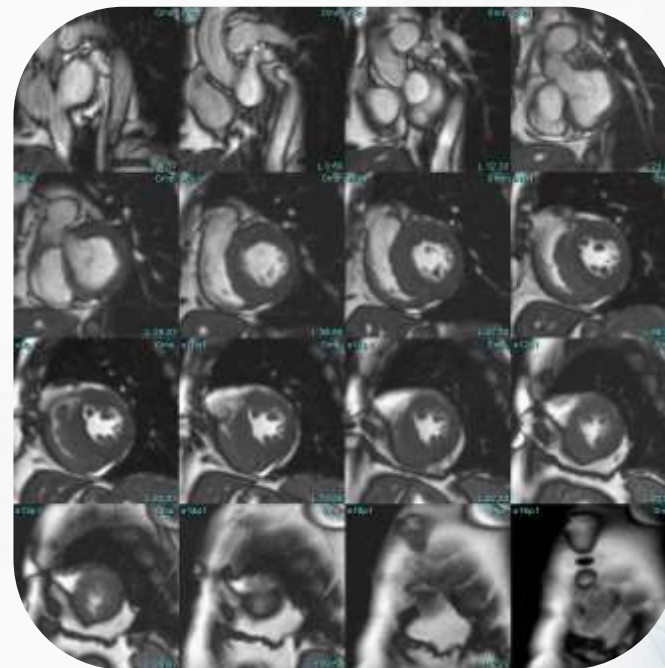
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urheartj/advance-article/doi/10.1093/eurheartj/ehad194/724660

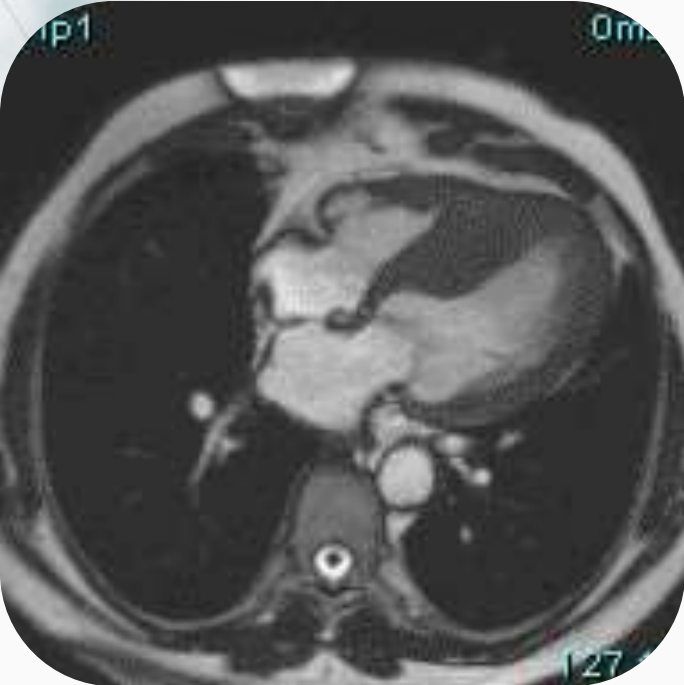
CMR

- LV EDV=179 ml
- LV ESV=63ml
- LV EDVi=91 ml/m²
- LV ESVi= 32 ml/m²
- LV EF 65%
- LV wall thickness max 33 mm (inferoseptal mid segment)
- LV Mass =248 gr
- LV Massi=126 gr/m²
- LA volume 88ml/m²
- LAVI = 45 ml/m²
- RV EF 48%
- RV Mass = 62 gr
- RV Massi= 32 gr/m²



Ventricles	LV	Range	RV	Range
Ejection Fraction (%)	65	51 - 71	48	40 - 60
Stroke Volume (ml)	116		43.1	
End-Diastolic Volume Index (ml/m ²)	90.9	57 - 105	45.8	48 - 112
End-Systolic Volume Index (ml/m ²)	32.0	14 - 38	23.8	41 - 117
End-Diastolic Volume (ml)	179	106 - 214	89.9	77 - 201
End-Systolic Volume (ml)	62.9	29 - 74	46.8	24 - 84
Heart Rate (bpm)	69		69	
Cardiac Output (l/min)	8.0		3.0	
Cardiac Output Index (l/min/m ²)	4.06		1.52	
Stroke Volume Index (ml/m ²)	58.9		22.0	12 - 52
Mass (g)	248(ED)	56 - 140	62(ED)	21 - 49
Mass Index (g/m ²)	126(ED)	41 - 81	32(ED)	12 - 28

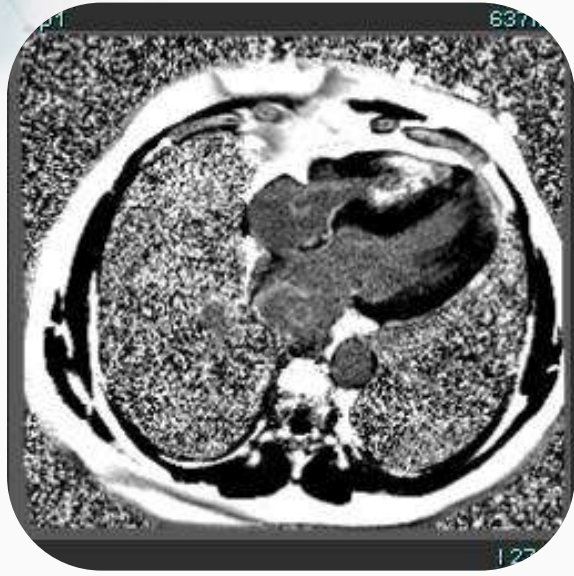
CMR cine-images



CMR T2w images – edema



CMR LGE



CMR LGE

present &
> 15% LV mass

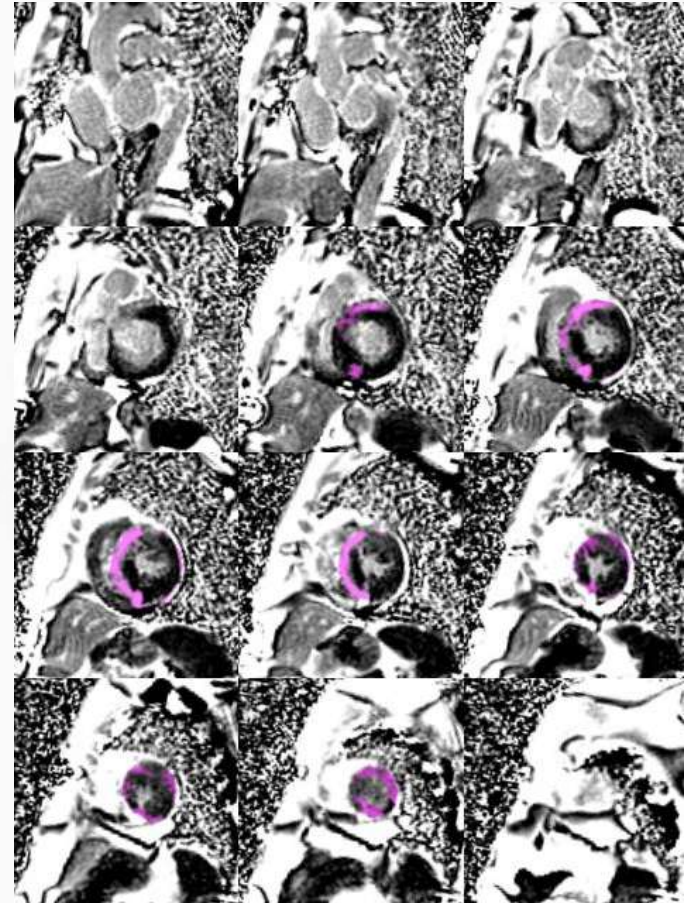
Study Date: Feb 10, 2022			
ID: 16-02-22 13:00 TS urek RY			
Age: 58 years			
Sex: Male			
Weight: 80 kg			
Height: 176 cm			
BSA: 1.96 m ²			
Referred By: Rustonova Y Dr			



Ventricles	LV	Range	RV	Range
Ejection Fraction (%)	65	51 - 71	48	40 - 60
Stroke Volume (ml)	118		43.1	
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Stroke Volume Index (ml/m ²)	58.9		22.0	12 - 52
Mass (g)	249(ED)	56 - 140	62(ED)	21 - 49
Mass Index (g/m ²)	129(ED)	41 - 81	32(ED)	12 - 28

Ventricles (Long Axis)	LV	Range	RV	Range
Ejection Fraction (%)	68	56 - 75	69	
Stroke Volume (ml)	95.9	50 - 119	20.8	
End-Diastolic Volume Index (ml/m ²)	71.8	59 - 99	15.3	
End-Systolic Volume Index (ml/m ²)	22.9	25 - 37	4.7	
End-Diastolic Volume (ml)	141	90 - 179	30.1	
End-Systolic Volume (ml)	45.0	25 - 66	9.3	
Heart Rate (bpm)	67		67	
Peak Filling Rate (ml/s)	369		96.0	
Peak Ejection Rate (ml/s)	465		115	
Cardiac Output (l/min)	6.4		1.4	
Cardiac Output Index (l/min/m ²)	3.27		0.71	
Stroke Volume Index (ml/m ²)	48.8		10.6	
Mass (g)	249(ED)		28(p20)	
Mass Index (g/m ²)	127(ED)		14(p20)	

Atria (Fast)	LA	Range	RA	Range
End-Diastolic Volume Index (ml/m ²)	44.9		19.8	
End-Diastolic Volume (ml)			38.9	

Lesion Enhancement	
Infarct Mass (g)	62.2
Left Ventricular Mass (g)	240
Infarct (%)	25.9



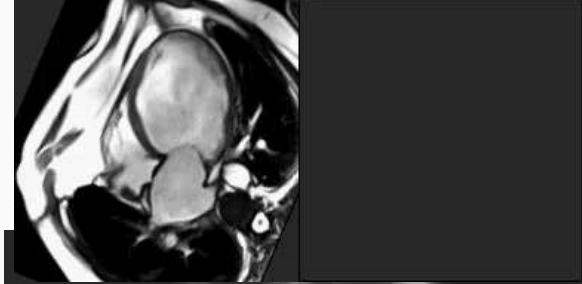
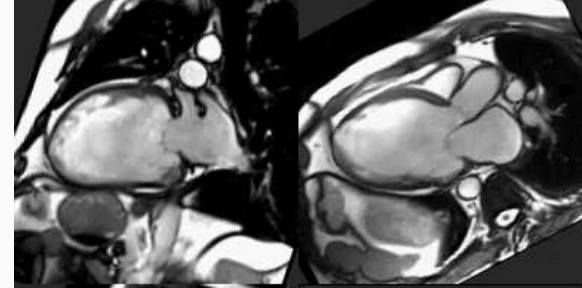
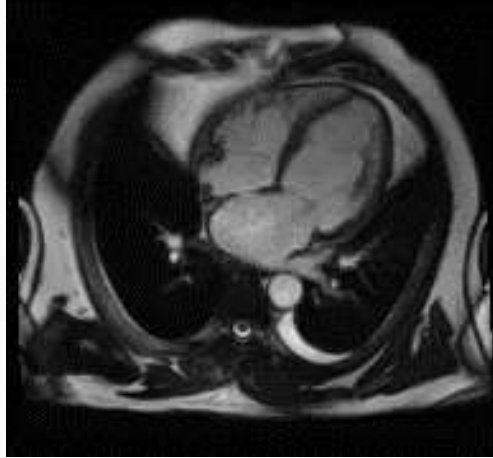
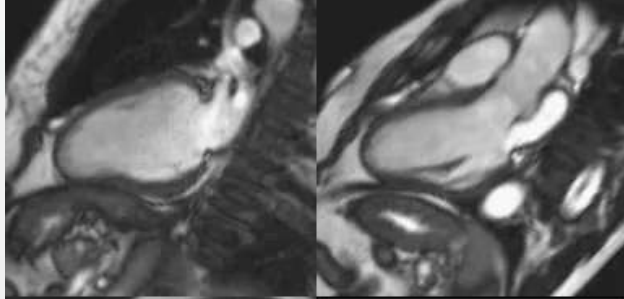


3 years FU – 2 shocks



Dilated Cardiomyopathy

CMR



Dilated Cardiomyopathy

Diagnosis

- For adults this represents an LV enddiastolic diameter >58 mm in males and >52 in females and an LVEDV index of ≥ 75 mL/m² in males and ≥ 62 mL/m² in females by ECHO. Left ventricular global systolic dysfunction is defined by LVEF $<50\%$.

Non-Dilated LV Cardiomyopathy (NDLVC)

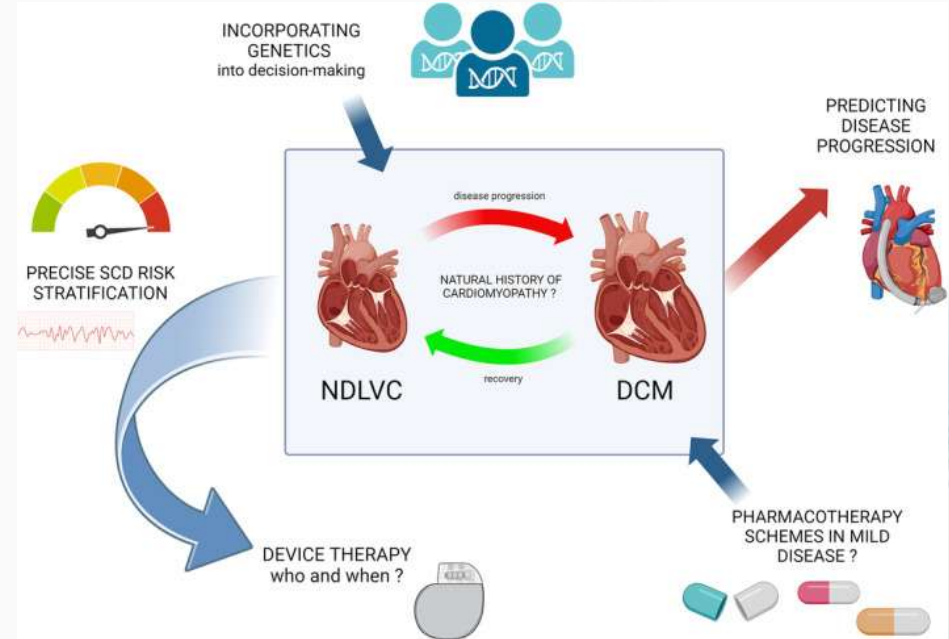
NDLVC is characterized by left ventricular systolic dysfunction (LVEF ↓), with normal or mildly increased LV volumes (i.e., non-dilated), often with myocardial fibrosis or arrhythmogenic substrate on CMR.

NDLVC and DCM represent a continuum

- Early stages → *NDLVC*
- Later stages → *DCM*

In **LMNA** or **TTN** mutations, the disease often starts as **NDLVC** and then **progresses to classic DCM** with chamber enlargement.

In **DSP**, **FLNC**, and **PLN** genotypes, the heart often **remains non-dilated** but develops **extensive fibrosis and arrhythmias**, representing the **arrhythmogenic left ventricular cardiomyopathy (ALVC)** phenotype.





Clinical significance

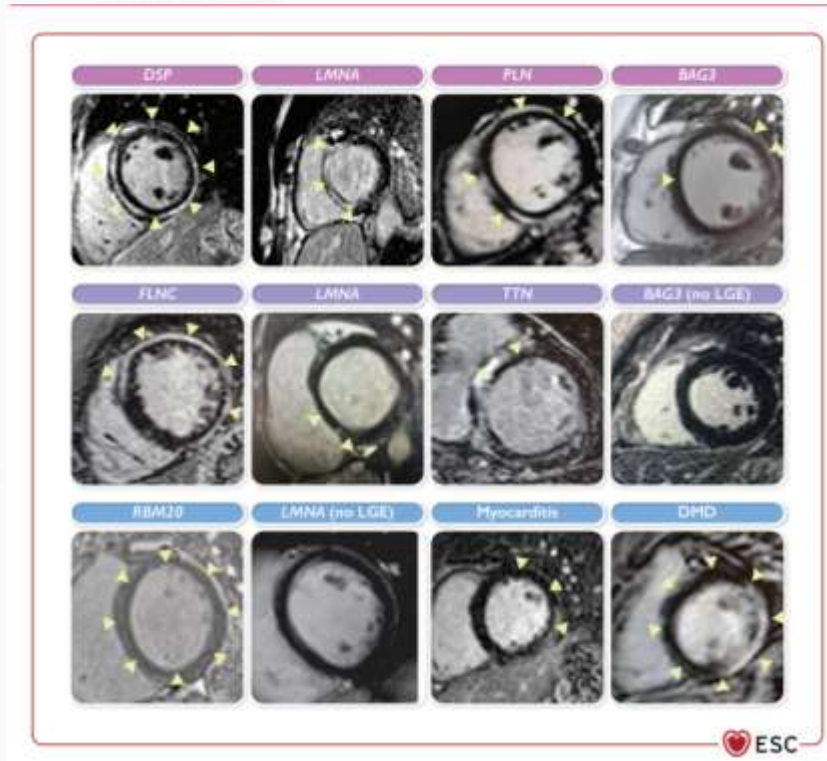
- Patients with **NDLVC** may develop **severe ventricular arrhythmias** even when left ventricular size is normal.
- In **DCM**, there is usually **marked structural remodeling**, but the degree of arrhythmogenic risk depends primarily on the **extent of myocardial fibrosis** rather than chamber size alone.
- Therefore, the **ESC 2023 Guidelines** now group these entities under a unified umbrella term:
- **“Left ventricular cardiomyopathies — either dilated or non-dilated, ischemic or non-ischemic.”**
- This reflects the concept that these phenotypes share overlapping mechanisms and represent **different expressions of the same pathological spectrum**.



Role of MRI: Beyond EF

- LVEF is not enough — CMR offers multiparametric insight:
 - Volumes & mass (cine SSFP)
 - Fibrosis (LGE, T1 mapping, ECV)
 - Edema/inflammation (T2/T2 mapping)
 - Perfusion (first-pass stress perfusion)
 - Tissue characterization & differential diagnosis
- **ESC 2023 HCM/DCM Recommendation:**
- *“CMR should be performed in all patients with suspected or confirmed cardiomyopathy for etiological and prognostic assessment (Class I, Level B).”*

Examples of non-dilated left ventricular cardiomyopathy phenotypes





T1 mapping and ECV: The new frontier

- Detect diffuse fibrosis beyond LGE.
- **Native T1 >1050 ms, ECV >30%** → worse outcomes, higher HF hospitalization.
- Guides timing for ICD / CRT / transplant.

ESC 2022–2023 Guideline Shift

EF alone is no longer enough — decisions must integrate etiology, fibrosis, age, and genotype.

Etiology

ESC Class

Key Modifiers

Ischemic cardiomyopathy

Class I

LVEF \leq 35%, NYHA II–III on OMT \geq 3 mo

Non-ischemic cardiomyopathy

Class IIa

LVEF \leq 35%, < 70 yrs, fibrosis on CMR, good prognosis > 1 yr

New Evidence Prompting Change

- **DANISH trial (2016, NEJM):**
 - In non-ischemic DCM, ICD ↓ SCD but **did not reduce all-cause mortality**
 - Benefit mainly in **patients < 70 years**
- **Modern HF therapy revolution (2016-2024):** ARNI, SGLT2i, MRA → ≈ 40 % lower SCD risk
- **CMR and genetics** identify arrhythmic substrate more precisely than EF alone

From EF to Fibrosis: A New Paradigm for SCD Prevention

- EF reflects *pump failure*, not *electrical vulnerability*
- Many patients with EF < 35 % die from **HF progression**, not SCD
- Some with EF > 35 % and **fibrosis on CMR** remain at high risk

CMR-detected myocardial fibrosis

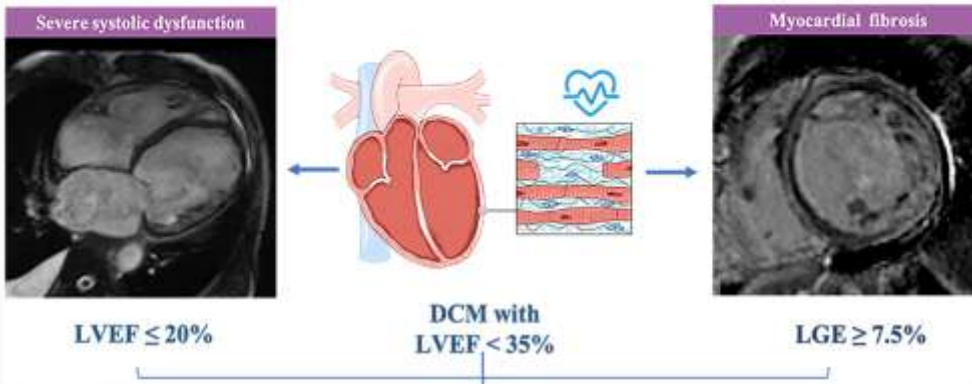


Development cohort (70%, n = 890)



Internal validation cohort (30%, n = 382)

Follow-up: 86.3 months (IQR: 72.5-106.6)



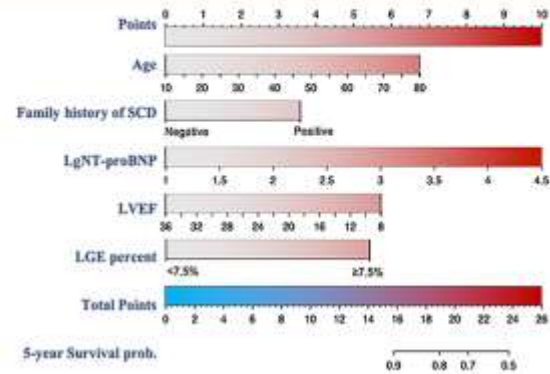
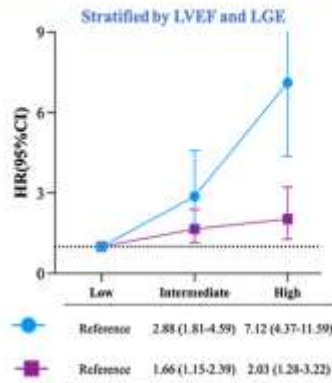
LVEF ≤ 20%

DCM with
LVEF < 35%

LGE ≥ 7.5%



Cardiac MRI-based risk stratification for clinical decision making

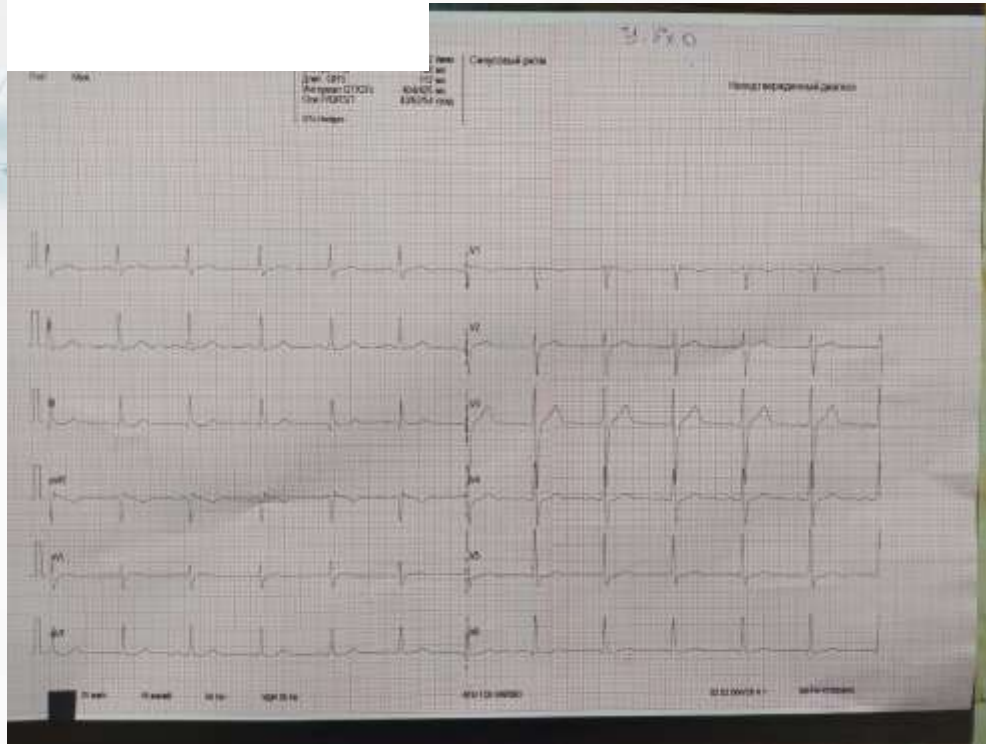


Clinical Vignette

- 45 y\o male pt
- Shortness of breath during prolonged physical activity
- Episodes of nocturnal dyspnea (shortness of breath mainly at night)

- **BP:** 125/78 mmHg
- **HR:** 94 bpm
- Non-remarkable physical examination
- **NT-pro-BNP:** 1200 pg/mL

ECG



Echo

LVEF=33%
LV EDV=188 ml
LV ESV= 120 ml

GDMT

- ARNI 24/26 mg 1 x 2
- Eprelenon 25 mg 1 x 1
- Karvedilol 12,5 mg 1 x 2
- Empagliflozin 10 mg 1 x 1

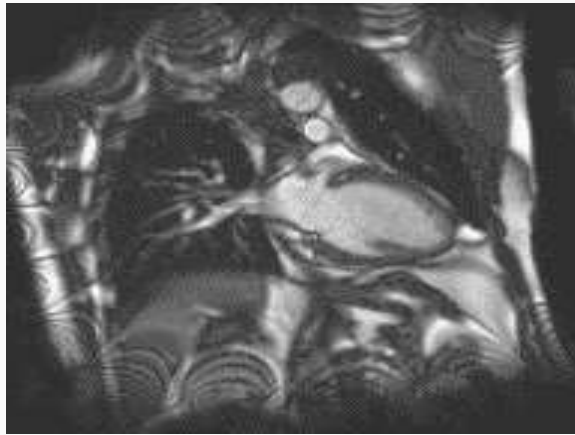
ICD?



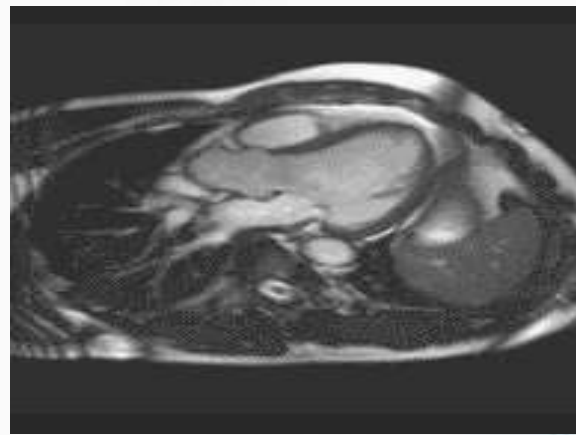
CMR



4CH

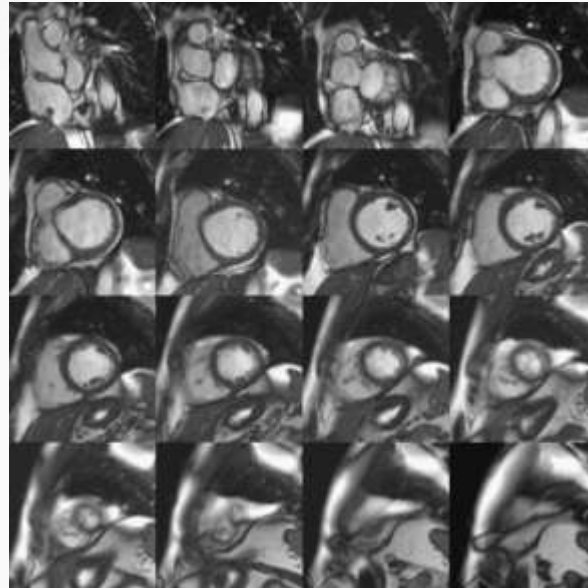


2CH



3CH

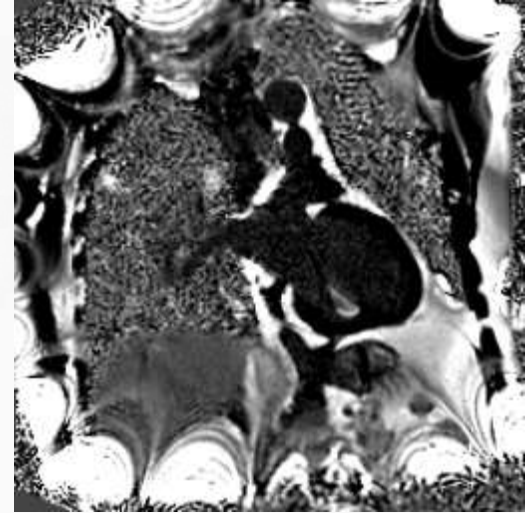
CMR -SA



CMR LGE

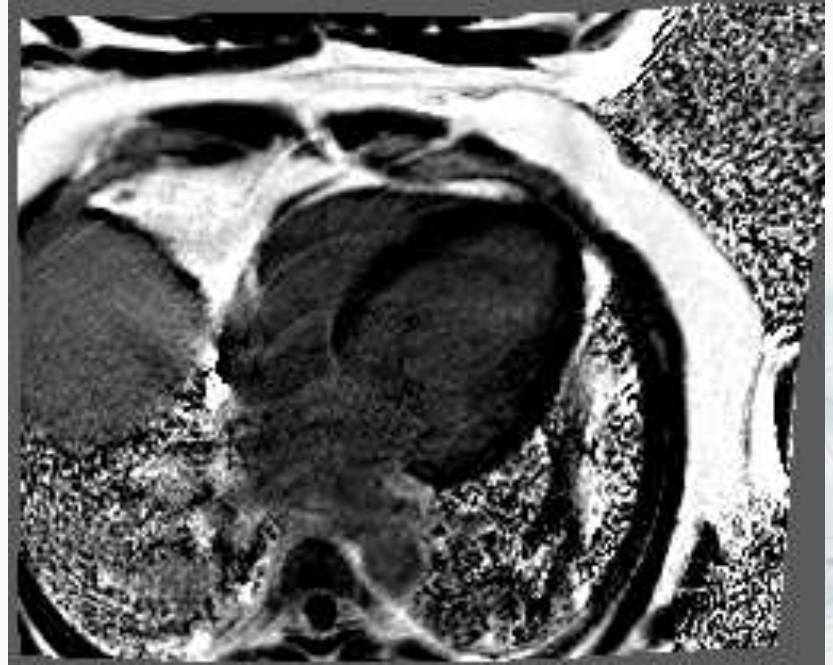


4CH



2CH

DCM vs DCM



Echo in dynamics

Parameter	11.10.2021	07.12.2021	06.04.2022
LVEF (%)	33	45%	50%
LVEDV (mL)	180	180	176
LVESV(mL)	120	98	78

5 years FU

CMR: No mid-wall fibrosis, ECV 34 %

Decision: GDMT with ARNI + SGLT2i

Follow-up: LVEF ↑ to 50 %, no VT episodes

Future Directions

- Parametric mapping integration in guidelines
- AI models for fibrosis quantification
- CMR + PET hybrid for sarcoidosis / inflammation
- Outcome-based CMR endpoints (LGE reduction, ECV changes)

Conclusions

- **CMR = molecular microscope** for non-ischemic HF
- **Fibrosis > EF** for risk stratification
- **Personalized decisions → better outcomes**
- **From EF to Fibrosis → New ESC Paradigm (2023–2025)**



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