Revisiting the Role of Cardiac MRI in Hypertrophic Cardiomyopathy: Current Practice and Future Perspectives

Clerio Azevedo

November 4 2024

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Hypertrophic Cardiomyopathy

Most common genetic cardiomyopathy; as many as 1:500 individuals in the general population

Most common cause of SCD in the young

More than 1400 known mutations in at least 11 genes encoding proteins of the cardiac sarcomere

Very heterogeneous phenotypic expression and diverse natural history

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Hypertrophic Cardiomyopathy Role of CMR

Diagnosis

Phenotypic characterization

Screening of family members

Differential diagnosis

Risk stratification for SCD

Management decisions

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Diagnosis

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Hypertrophic Cardiomyopathy

Unexplained LV hypertrophy (≥15 mm wall thickness) in the absence of another disease capable of producing the magnitude of increased wall thickness.

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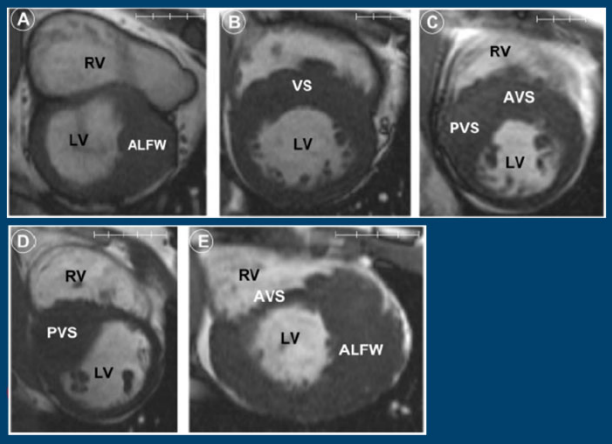
Imaging

Utility of Cardiac Magnetic Resonance Imaging in the Diagnosis of Hypertrophic Cardiomyopathy

Carsten Rickers, MD; Norbert M. Wilke, MD; Michael Jerosch-Herold, PhD; Susan A. Casey, RN; Prasad Panse, MD; Neeta Panse, MD; Jochen Weil, MD; Andrey G. Zenovich, MSc; Barry J. Maron, MD

(*Circulation*. 2005;112:855-861.)



- Echo did not identify LVH in 6% of patients
- Echo underestimated the severity of LVH in 10% of patients

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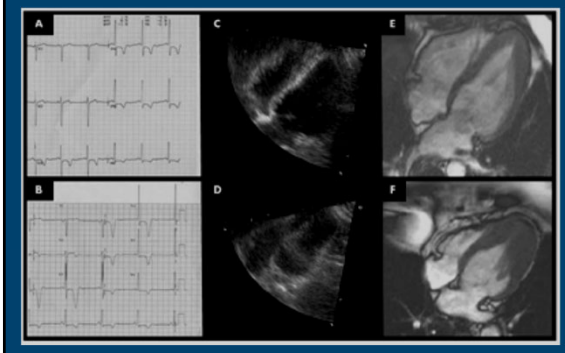
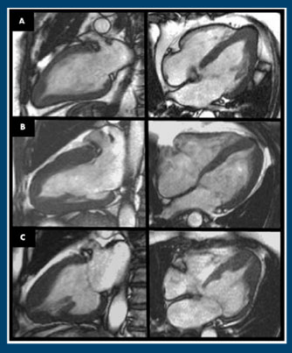
CARDIOVASCULAR MEDICINE

Detection of apical hypertrophic cardiomyopathy by cardiovascular magnetic resonance in patients with non-diagnostic echocardiography

J C C Moon, N G Fisher, W J McKenna, D J Pennell

Heart 2004;**90**:645–649. doi: 10.1136/hrt.2003.014969





- 10 patients with suspected apical HCM and normal echo: all cases confirmed by CMR (apical hypertrophy as severe as 2.8 cm)

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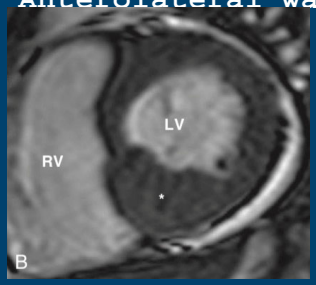
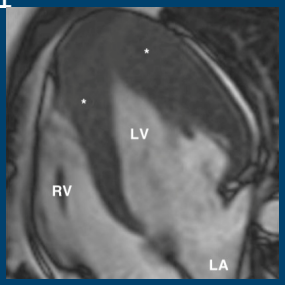
Particularly "Echo Blind" regions

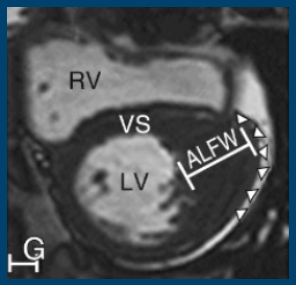
Inferoseptal wall

Anterolateral wall

LV apex







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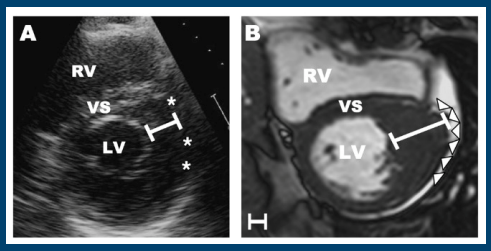
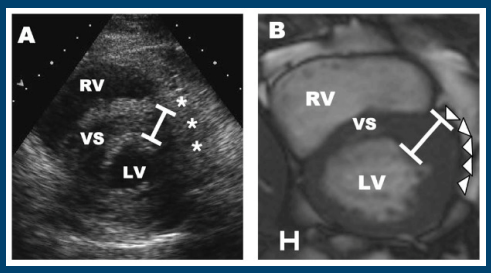
Echo may underestimate wall thickness

(Am J Cardiol 2010;105:1842–1843)

Management Implications of Massive Left Ventricular Hypertrophy in Hypertrophic Cardiomyopathy Significantly Underestimated by Echocardiography but Identified by Cardiovascular Magnetic Resonance

Martin S. Maron, MD[a,*], John R. Lesser, MD[b], and Barry J. Maron, MD[b]





- Echo may underestimate by as much as 14 mm compared to CMR

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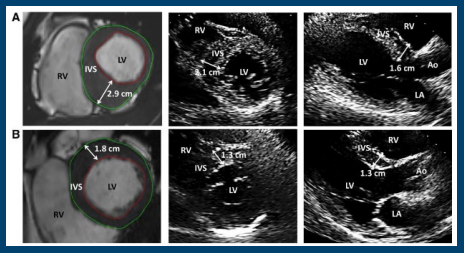
Discrepant Measurements of Maximal Left Ventricular Wall Thickness Between Cardiac Magnetic Resonance Imaging and Echocardiography in Patients With Hypertrophic Cardiomyopathy

Waseem Hindieh, MD*; Adaya Weissler-Snir, MD*; Helene Hammer, MD; Arnon Adler, MD; Harry Rakowski, MD; Raymond H. Chan, MD, MPH

(*Circ Cardiovasc Imaging*. 2017;10:e006309.DOI:10.1161/CIRCIMAGING.117.006309.)



- In 92.8% of patients, TTE underestimated (33.0%) or overestimated (59.8%) maximal LVWT.
- Underestimation was because of focal LVH or poor acoustic windows, while overestimation resulted from inclusion of RV myocardium, LV trabeculations and papillary muscle, as well as imaging plane obliquity.
- In 15.9% of patients, measurement discrepancy occurred at diagnostic or prognostic cut-offs.

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Phenotypic characterization

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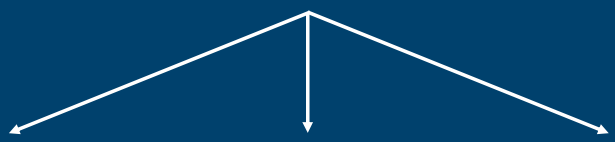
Phenotypic characterization



Patterns of LVH

- Distributions of LVH
- Maximal wall thickness
- Presence of RV hypertrophy
- Presence of apical aneurysm

LVOT obstruction

- SAM of the mitral valve
- Elongated anterior MV leaflet
- Papillary muscle abnormal position/insertion
- Small LV cavity size and basal septal hypertrophy
- MR secondary to SAM
- Quantification of LVOT obstruction severity

Small-vessel ischemia

- Stress perfusion CMR

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Patterns and distributions of LVH

JACC VOL. 74, NO. 19, 2019
NOVEMBER 12, 2019:2333–45

Distinct Subgroups in Hypertrophic Cardiomyopathy in the NHLBI HCM Registry

Stefan Neubauer, MD,[a] Paul Kolm, PhD,[b] Carolyn Y. Ho, MD,[c] Raymond Y. Kwong, MD, MPH,[c] Milind Y. Desai, MD,[d] Sarahfaye F. Dolman, MPH,[b] Evan Appelbaum, MD,[e] Patrice Desvigne-Nickens, MD,[f] John P. DiMarco, MD, PhD,[g] Matthias G. Friedrich, MD,[h] Nancy Geller, PhD,[f] Andrew R. Harper, MBBS,[a] Petr Jarolim, PhD,[c] Michael Jerosch-Herold, PhD,[c] Dong-Yun Kim, PhD,[f] Martin S. Maron, MD,[i] Jeanette Schulz-Menger, MD,[j] Stefan K. Piechnik, PhD,[a] Kate Thomson, PhD,[a] Cheng Zhang, PhD,[b] Hugh Watkins, MD, PhD,[a] William S. Weintraub, MD,[b] Christopher M. Kramer, MD,[g] on behalf of the HCMR Investigators

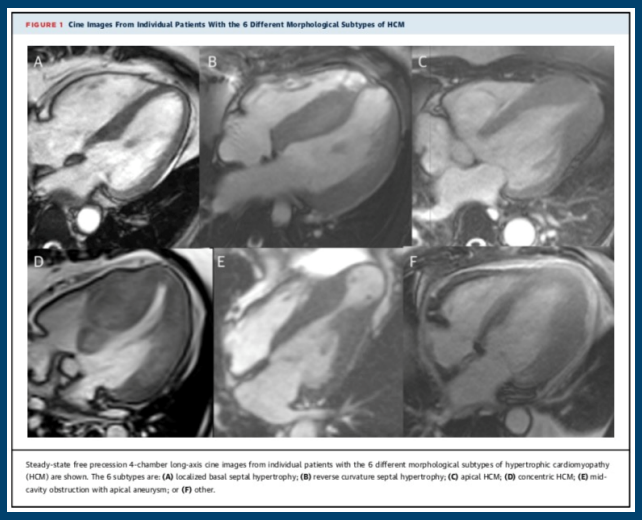

FIGURE 1 Cine Images From Individual Patients With the 6 Different Morphological Subtypes of HCM

Steady-state free precession 4-chamber long-axis cine images from individual patients with the 6 different morphological subtypes of hypertrophic cardiomyopathy (HCM) are shown. The 6 subtypes are: **(A)** localized basal septal hypertrophy; **(B)** reverse curvature septal hypertrophy; **(C)** apical HCM; **(D)** concentric HCM; **(E)** mid-cavity obstruction with apical aneurysm; or **(F)** other.

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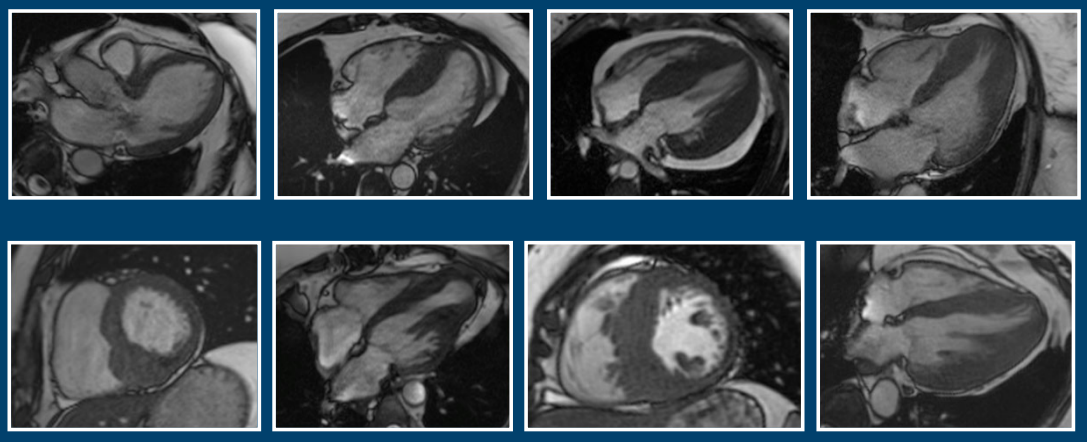
Patterns and distributions of LVH



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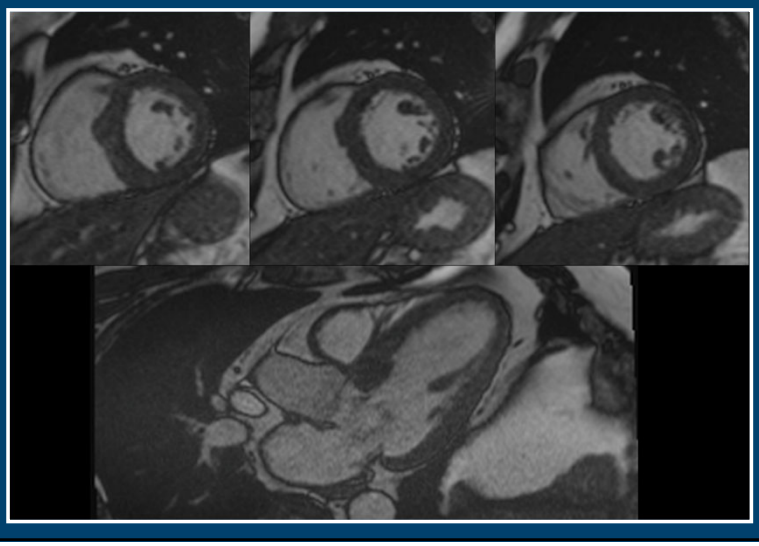
Patterns and distributions of LVH

Maximal wall thickness:

Careful with the crista supraventricularis



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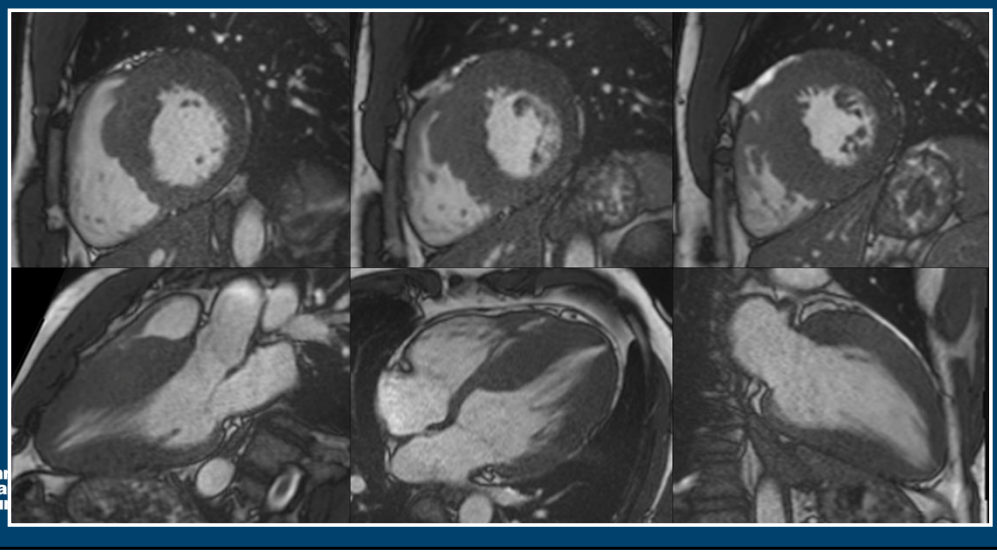
Patterns and distributions of LVH

Maximal wall thickness:

Careful with the crista supraventricularis



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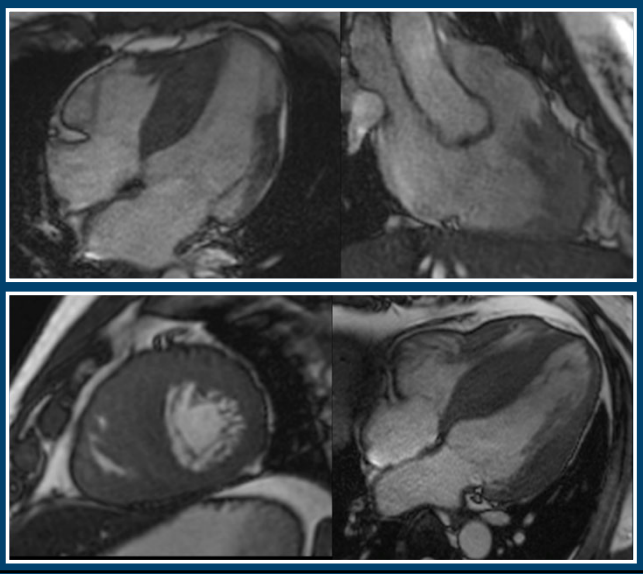
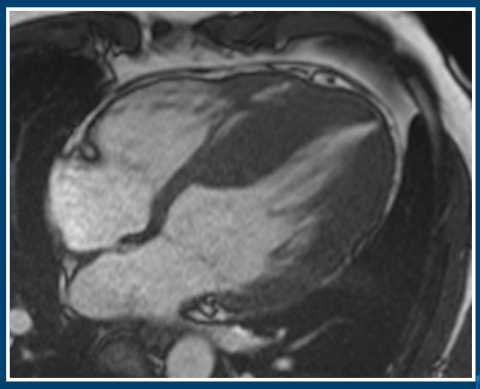
Patterns and distributions of LVH

Right ventricular hypertrophy





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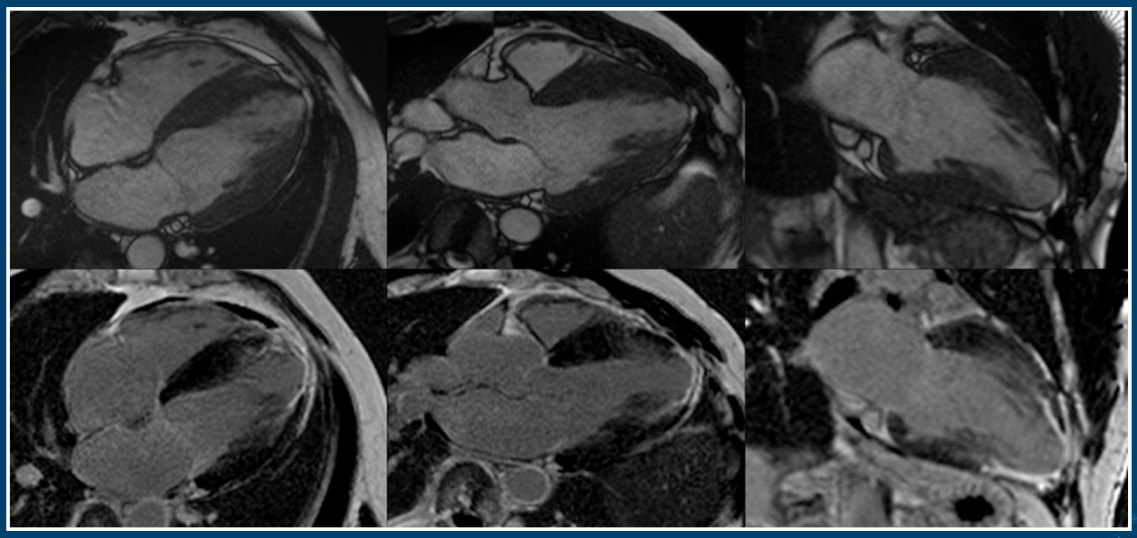
Patterns and distributions of LVH

Apical aneurysm



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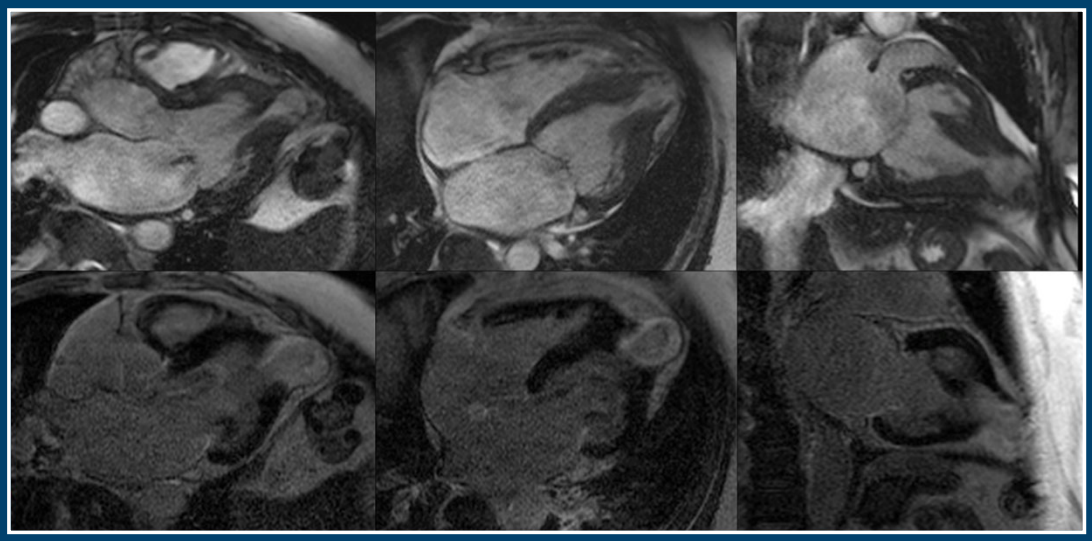
Patterns and distributions of LVH

Apical aneurysm



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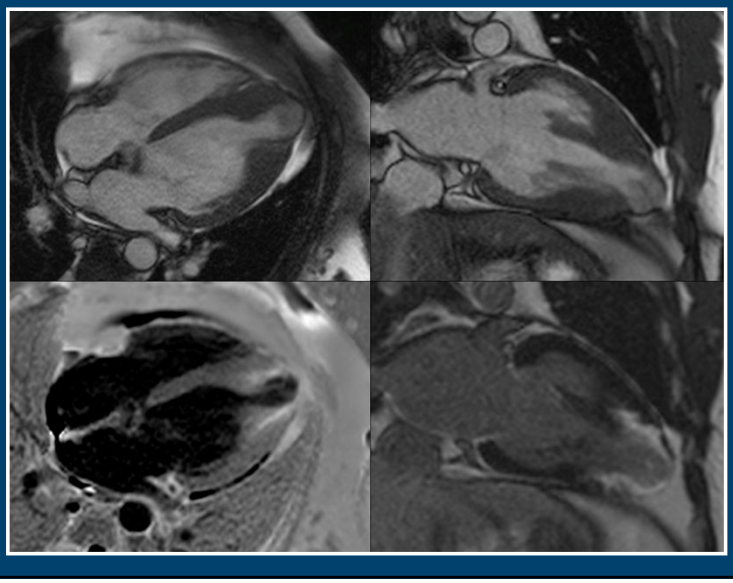
Patterns and distributions of LVH

Apical aneurysm



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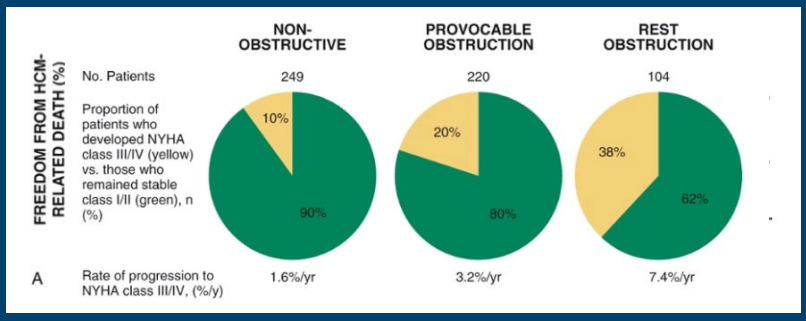
LV outflow tract obstruction

The majority of patients (70%) have the propensity to develop LV outflow obstruction with dynamic gradients of 30 mm Hg or more, either at rest or with physiologic exercise.

Most relevant clinical determinant of HCM-related progressive heart failure symptoms.



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LV outflow tract obstruction Pathophysiology

- LVOT obstruction is due to SAM of the mitral valve contacting the ventricular septum in midsystole.
- Morphologic alterations of the LVOT area that contribute to the development of SAM-septal contact:
  - Narrowed diameter of the LVOT due to increased septal wall thickness;
  - Apically positioned papillary muscles that tether the mitral valve plane toward the ventricular septum;
  - Elongated anterior leaflet of the mitral valve.
- SAM of the mitral valve often produces posteriorly directed mitral regurgitation.

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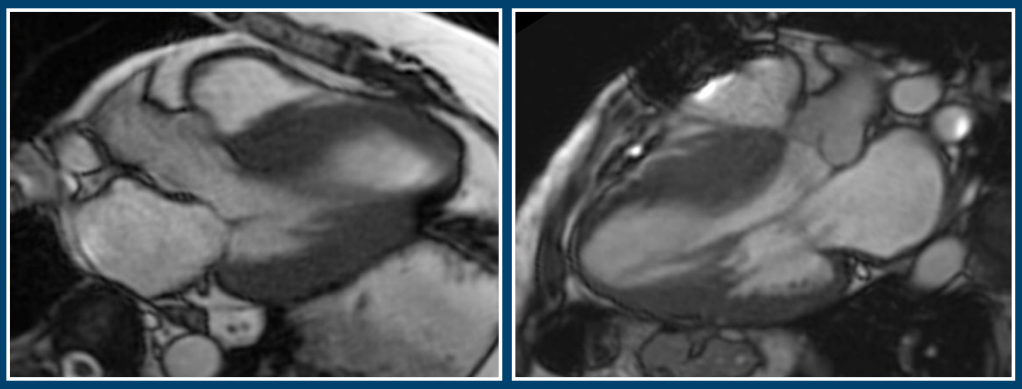
LV outflow tract obstruction

SAM of the mitral valve



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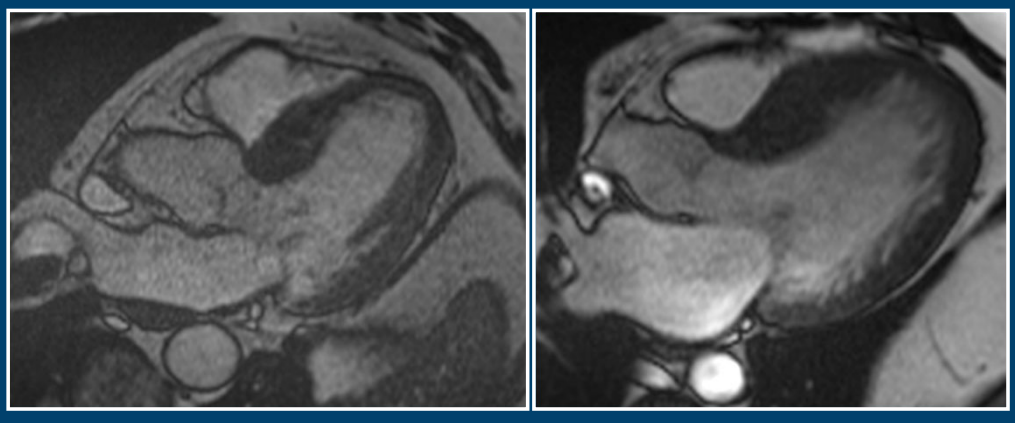
LV outflow tract obstruction

SAM of the mitral valve



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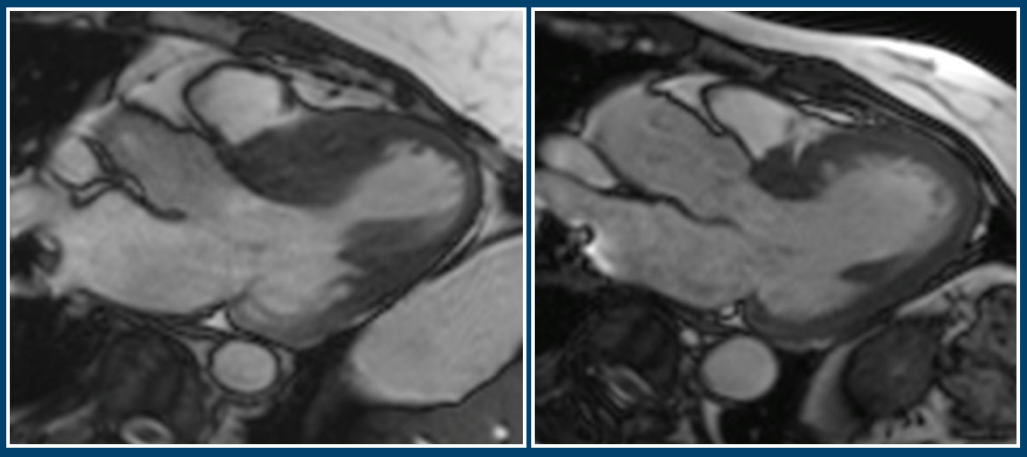
LV outflow tract obstruction

Elongated anterior MV leaflet



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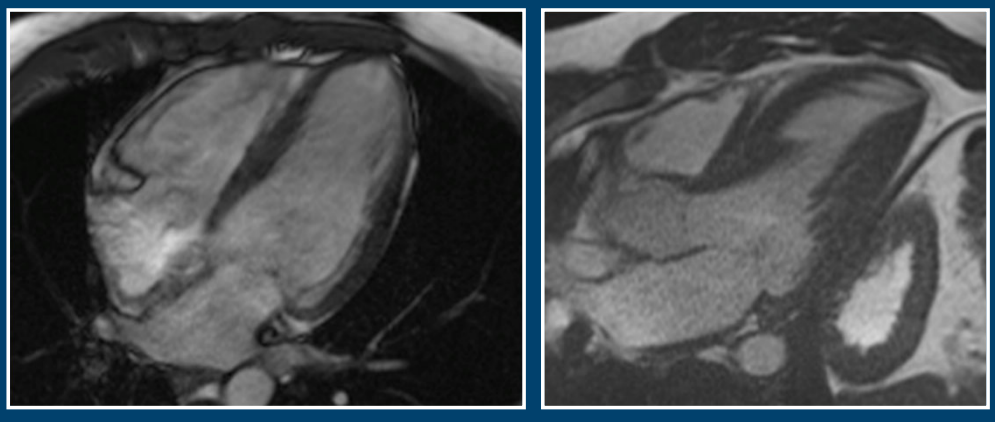
LV outflow tract obstruction

Papillary muscle abnormal position/insertion



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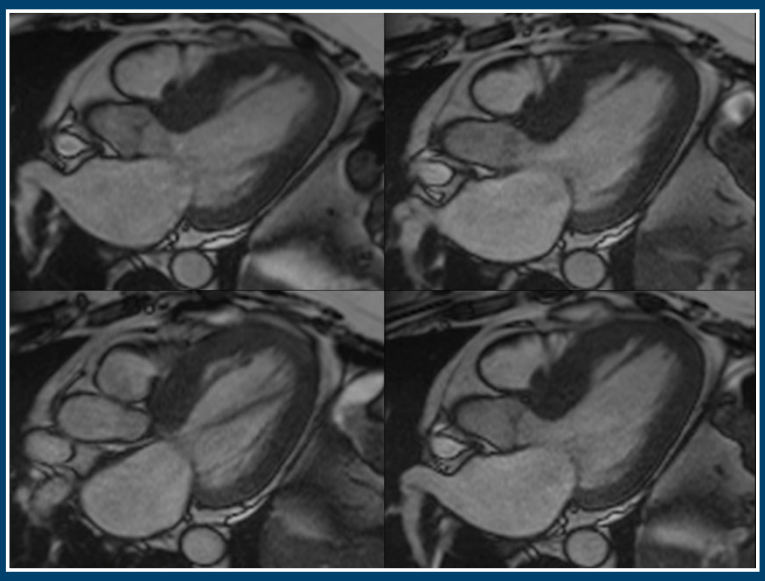
LV outflow tract obstruction

Papillary muscle abnormal position/insertion



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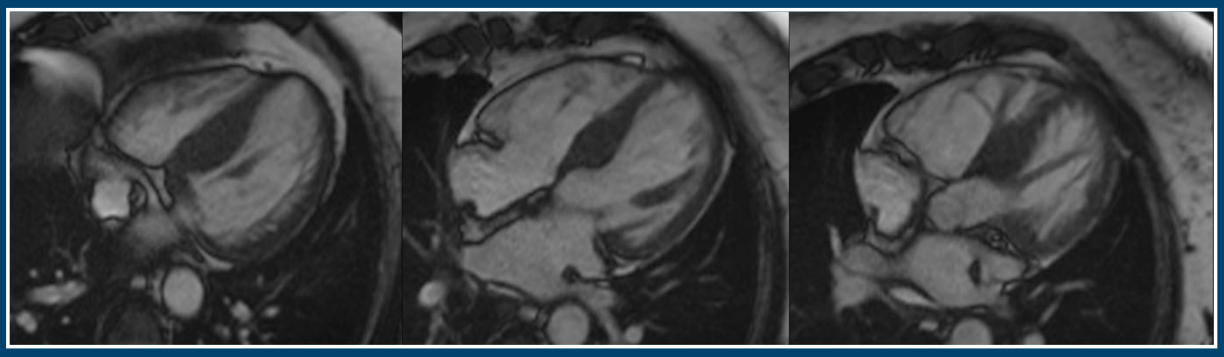
LV outflow tract obstruction

Papillary muscle abnormal position/insertion



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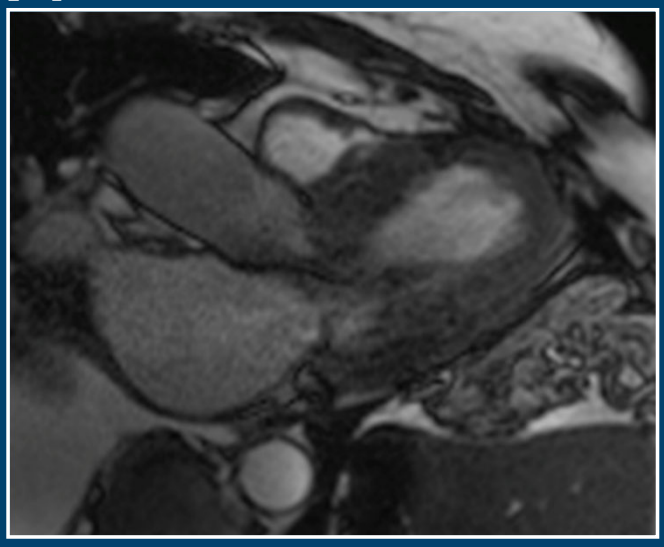
LV outflow tract obstruction

Small LV cavity size and basal septal hypertrophy



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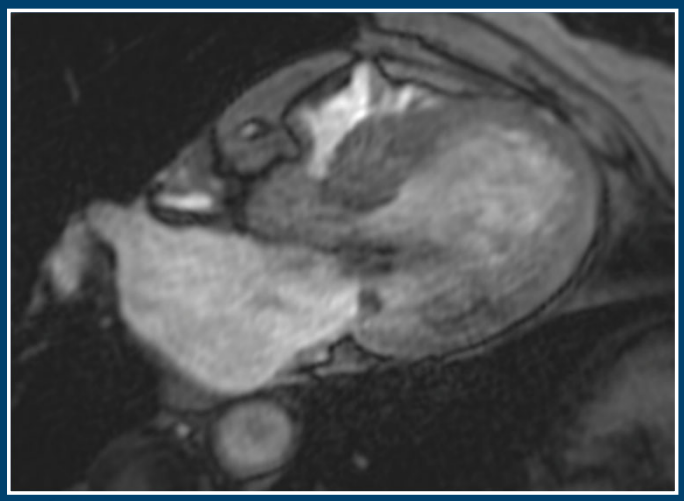
LV outflow tract obstruction

MR secondary to SAM



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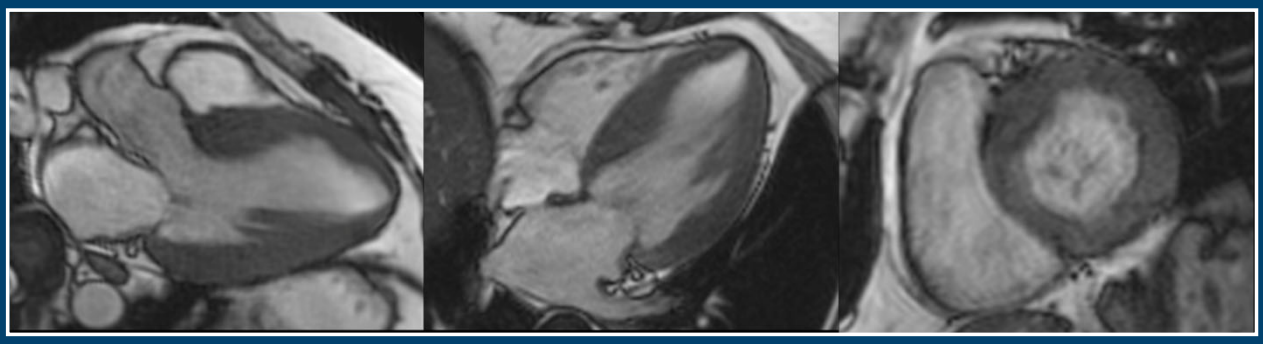
LV outflow tract obstruction

MR secondary to SAM



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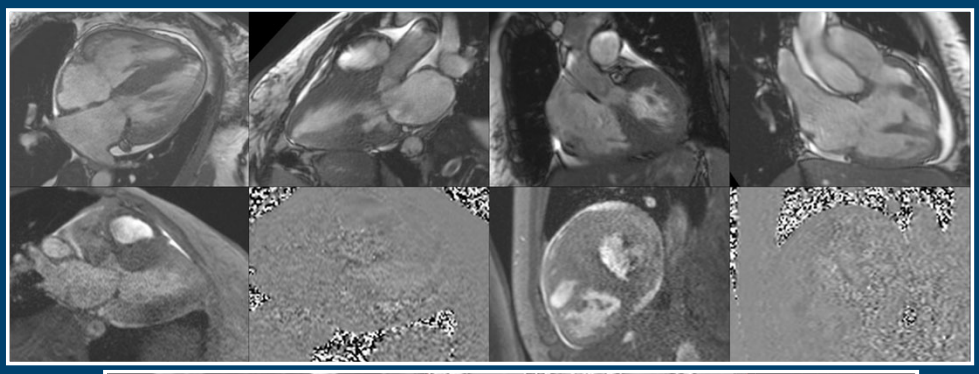
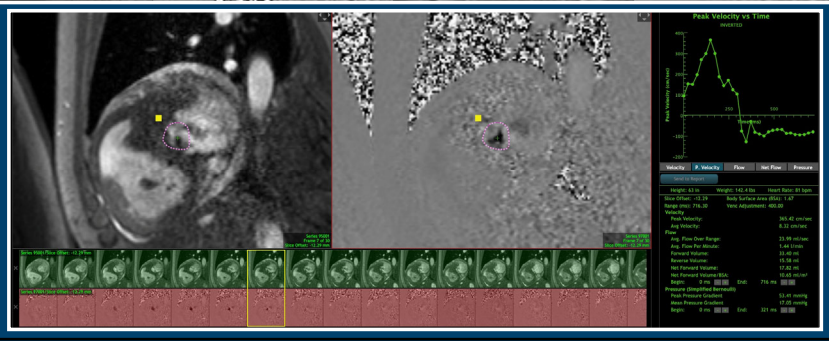
LVOT obstruction severity quantification





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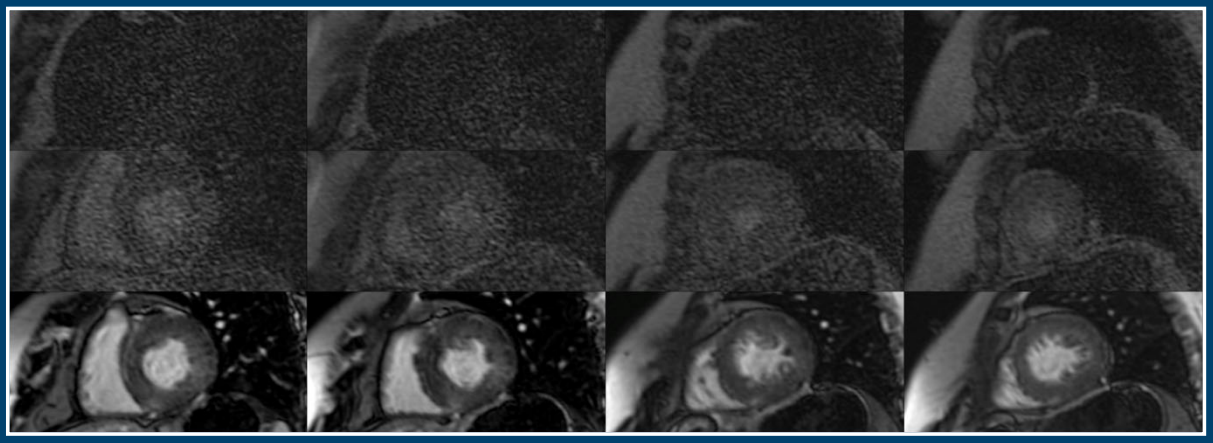
Small-vessel ischemia



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Screening of family members

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Screening of family members

TABLE 7 Screening With Electrocardiography and 2D Echocardiography in Asymptomatic Family Members*

Age of First-Degree Relative	Initiation of Screening	Repeat ECG, Echo
Pediatric		
Children and adolescents from genotype-positive families, and families with early onset disease	At the time HCM is diagnosed in another family member	Every 1-2 y
All other children and adolescents	At any time after HCM is diagnosed in a family member but no later than puberty	Every 2-3 y
Adults	At the time HCM is diagnosed in another family member	Every 3-5 y

Unexplained wall thickness of ≥13 mm is sufficient for diagnosis in relatives of individuals with HCM or those who are genotype positive.

Genotype positive - Phenotype negative (G+P-)
- Myocardial crypts
- Elongated mitral valve leaflets
- Delayed-enhancement

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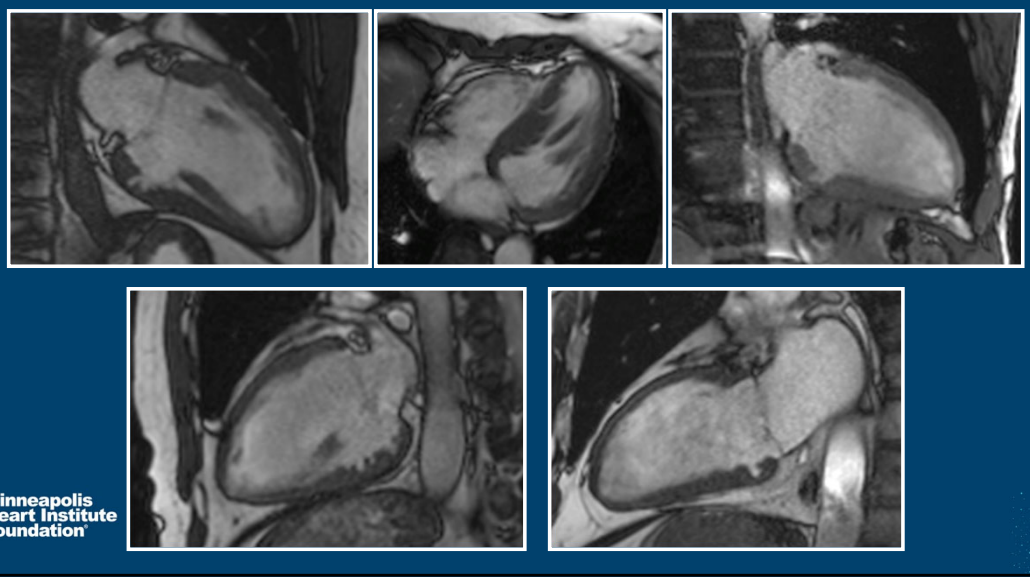
Screening of family members

G+P-: Myocardial crypts



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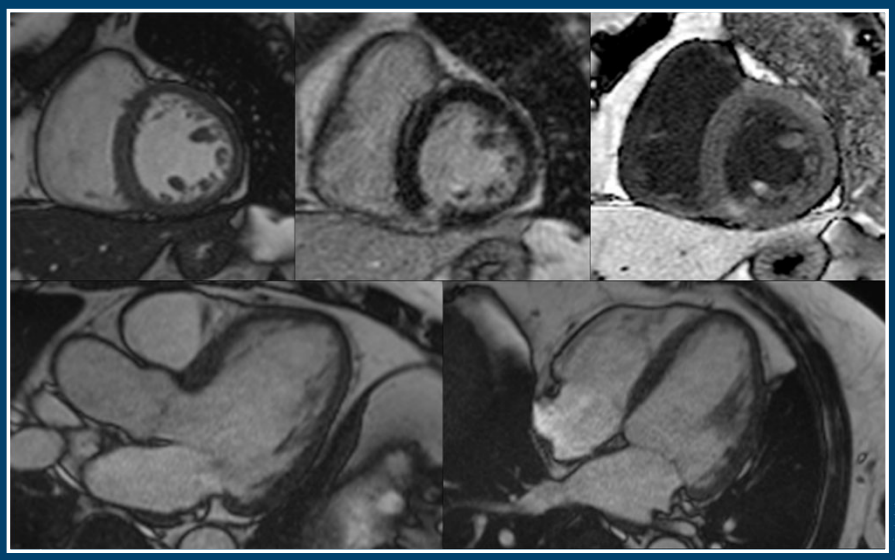
Screening of family members

G+P-: Delayed-enhancement



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Differential diagnosis

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Differential diagnosis: Athlete's Heart

Ambiguous "gray zone" of overlap between the two conditions (when the maximum LV wall thicknesses is 13 to 15 mm.

Regression of LVH with deconditioning: decrease in wall thickness > 2mm after 4-6 weeks period of deconditioning.

	PATHOLOGIC LV HYPERTROPHY (HCM)	PHYSIOLOGIC LV HYPERTROPHY (ATHLETE'S HEART)
Focal pattern of LV hypertrophy	+	0
LV cavity < 45mm	+	0
LV cavity > 55mm	0	+
Left atrium enlargement	+	0
Bizarre ECG patterns	+	+
Abnormal LV filling pattern	+	0
Family history of HCM	+	0
Decreased LV thickness with deconditioning	0	+
VO_2 increase > 110%	0	+
Late gadolinium enhancement	+	0
Pathogenic sarcomere mutation	+	0

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Differential diagnosis: Hypertensive cardiomyopathy

Is the severity hypertension consistent with the degree of LVH?

A concentric pattern of hypertrophy favors a diagnosis of hypertensive cardiomyopathy.

LVOT obstruction with SAM and/or more extensive DE favors hypertrophic cardiomyopathy.

Regression of LVH with aggressive anti-hypertensive treatment strongly suggest hypertensive cardiomyopathy.

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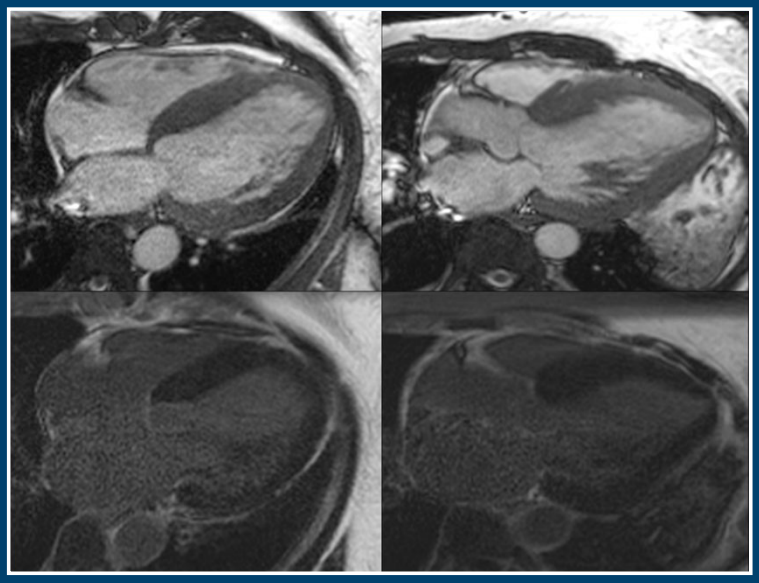
Differential diagnosis: Hypertensive cardiomyopathy



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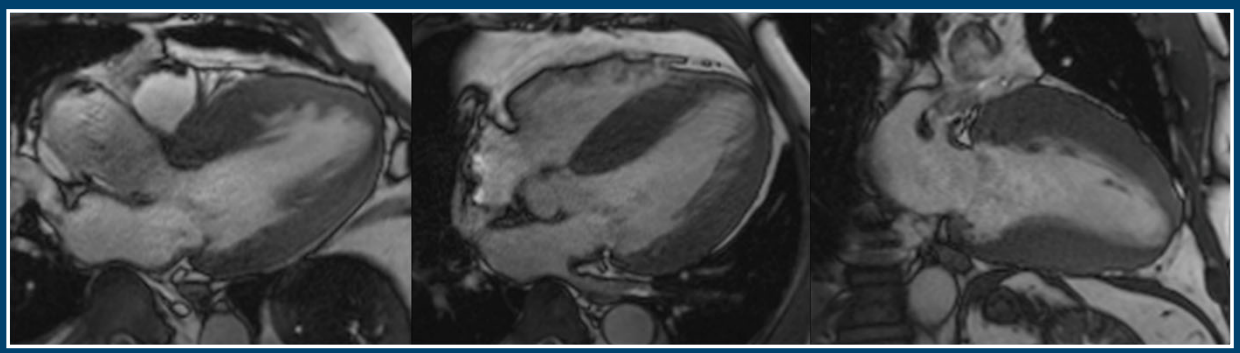
Differential diagnosis: Hypertensive cardiomyopathy



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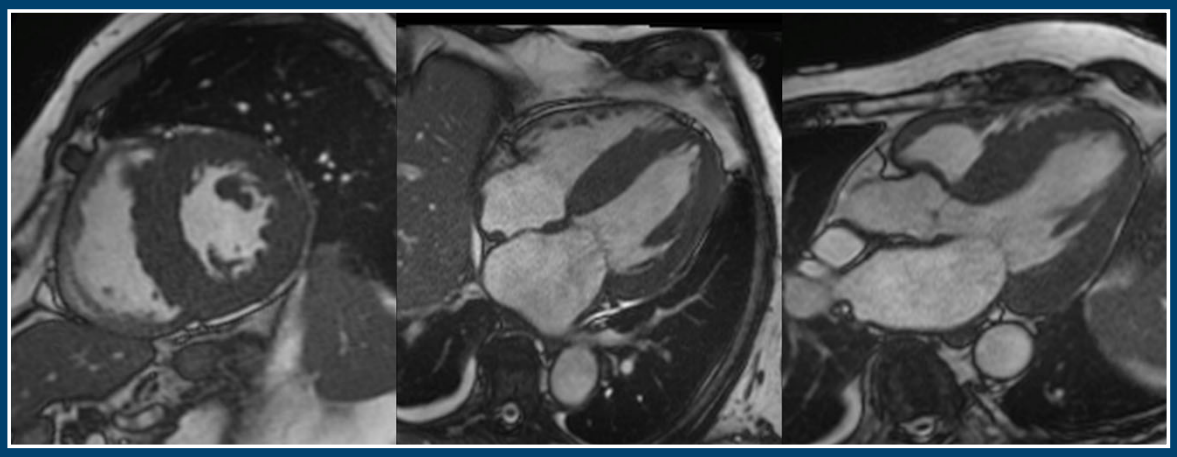
Differential diagnosis



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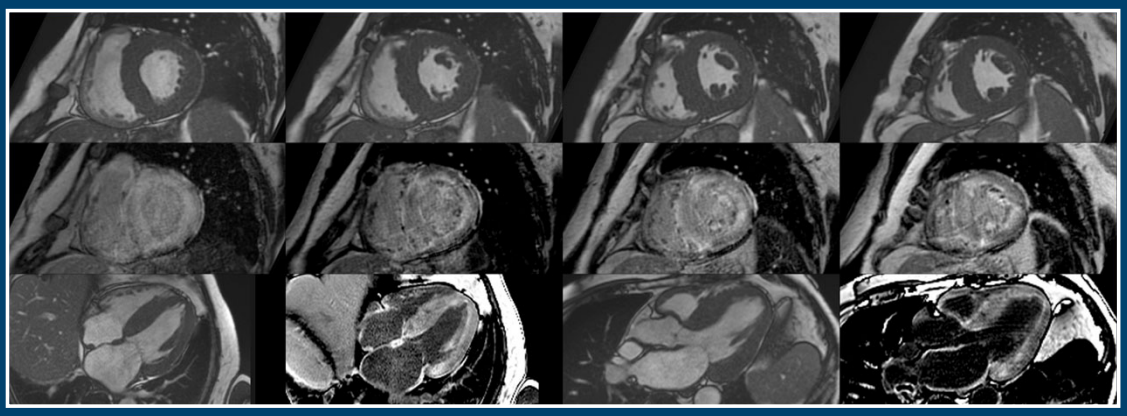
Differential diagnosis: Cardiac amyloidosis



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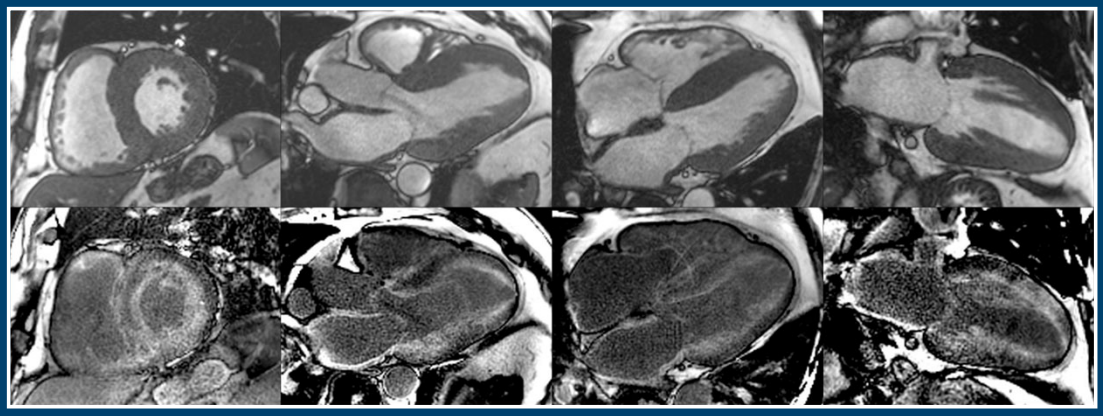
Differential diagnosis: Cardiac amyloidosis



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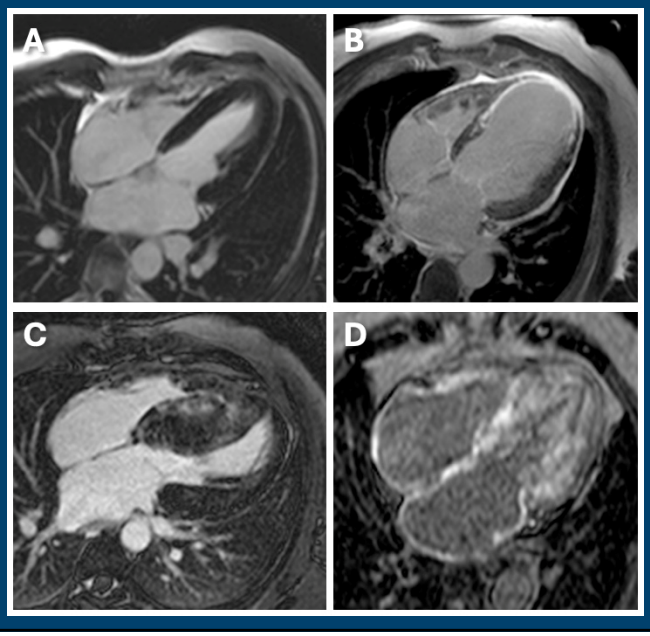
Differential diagnosis: Delayed-enhancement



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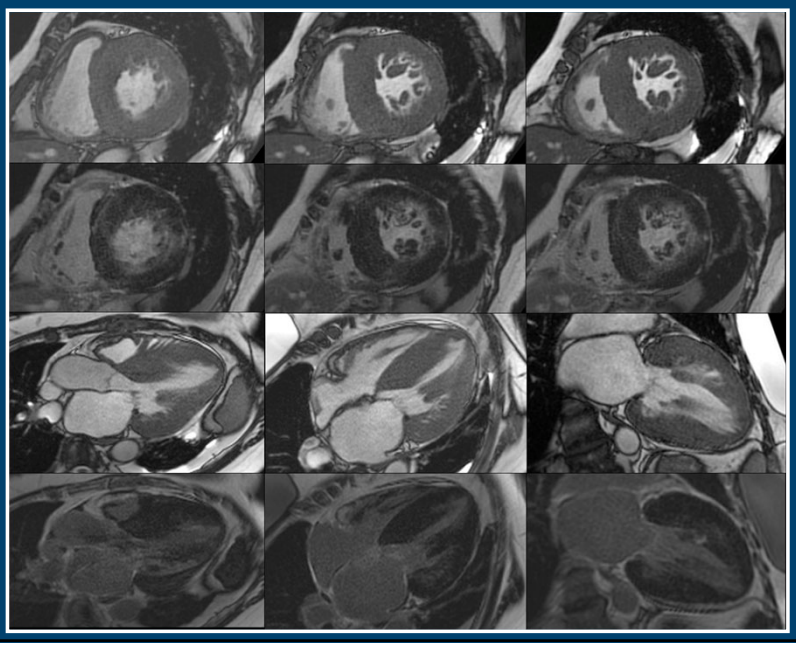
Differential diagnosis: Anderson-Fabry disease



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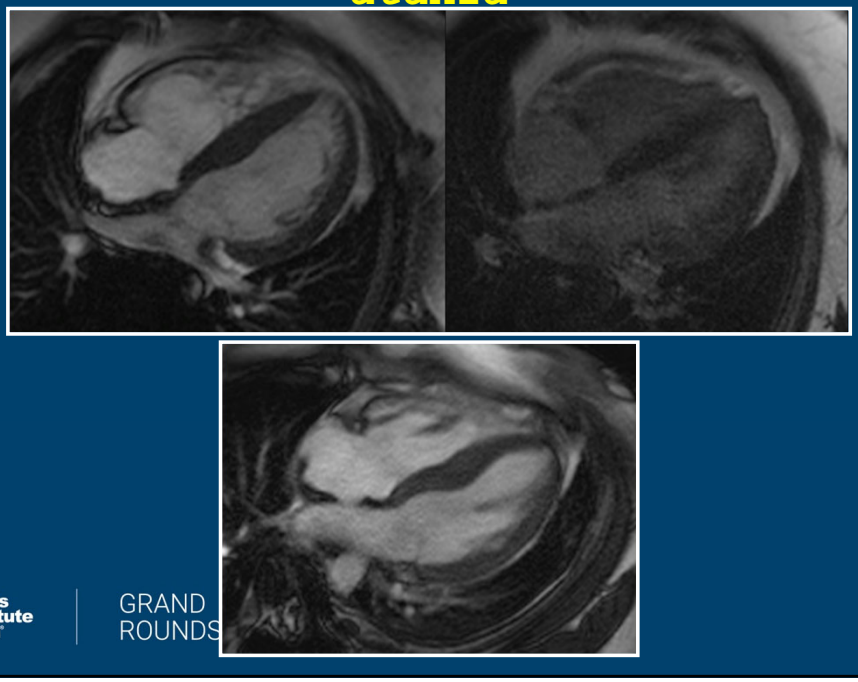
Differential diagnosis: Friedreich's ataxia



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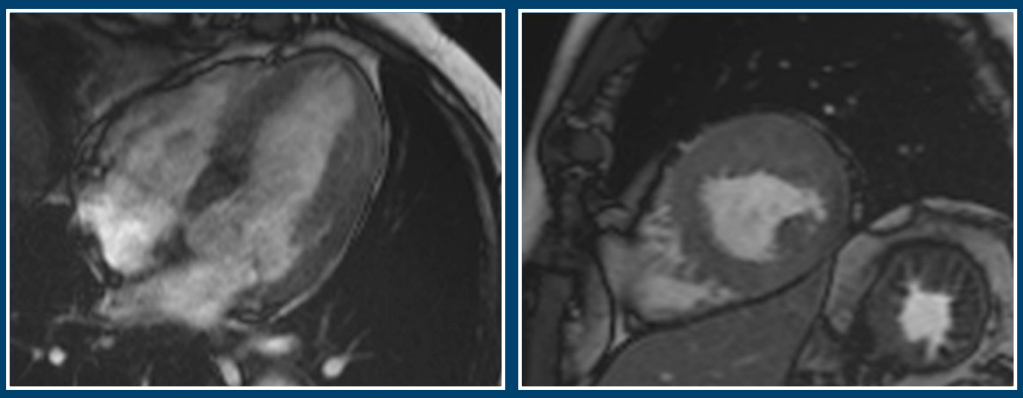
Differential diagnosis: Hypereosinophilic Syndrome



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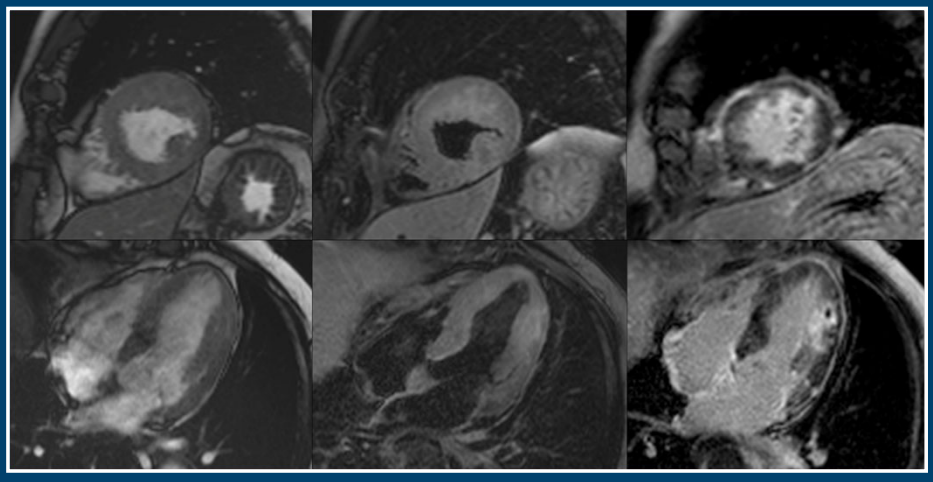
Differential diagnosis: Hypereosinophilic Syndrome



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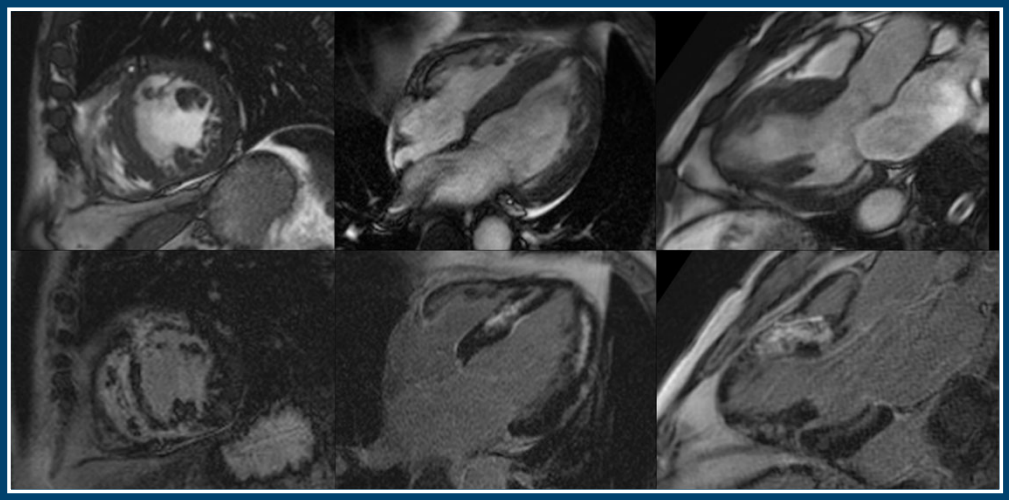
Differential diagnosis: Danon disease



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Risk stratification for SCD

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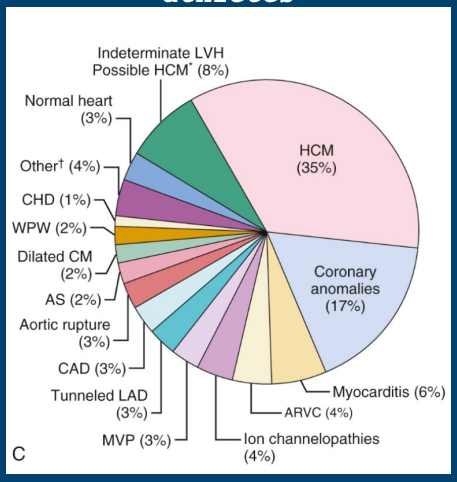
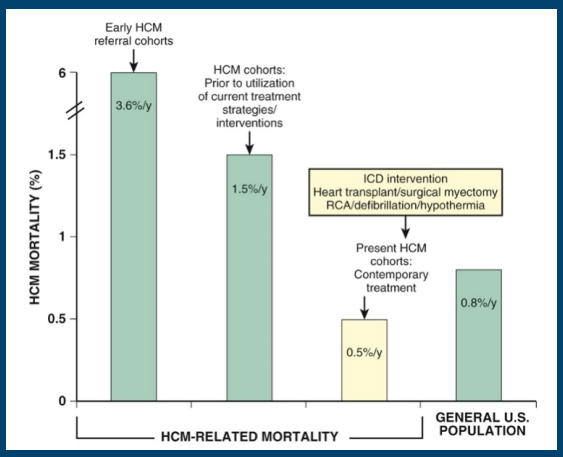
Risk stratification for SCD

SCD in young competitive athletes





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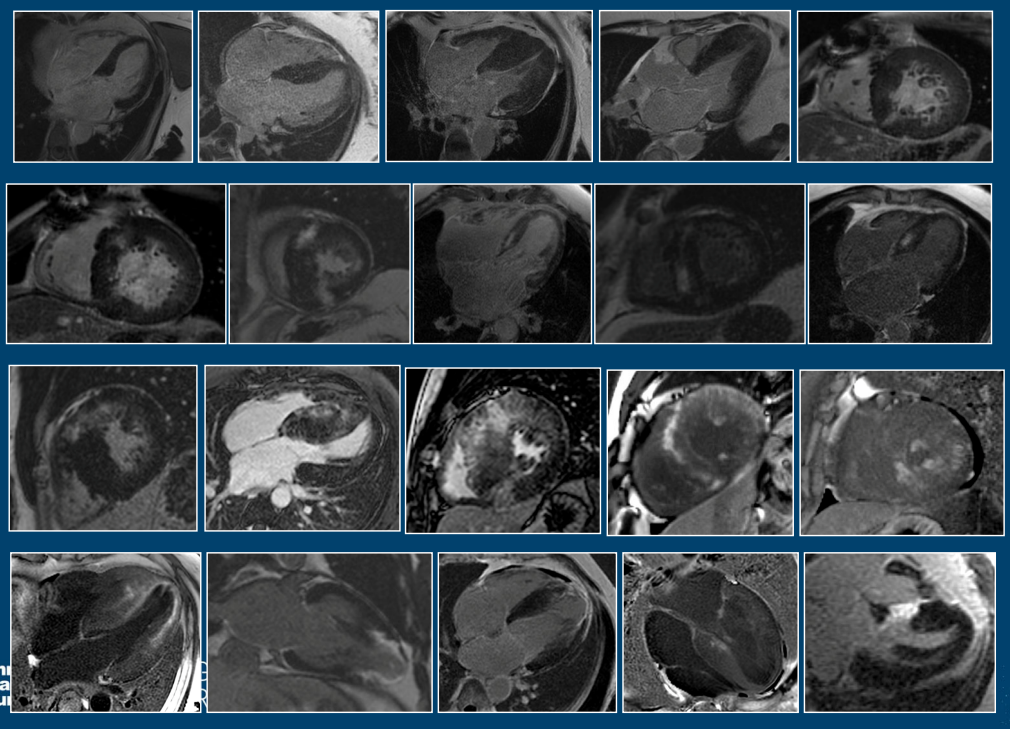

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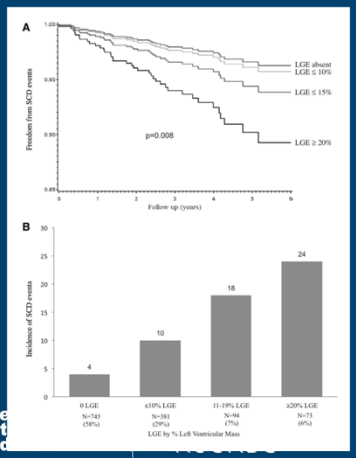
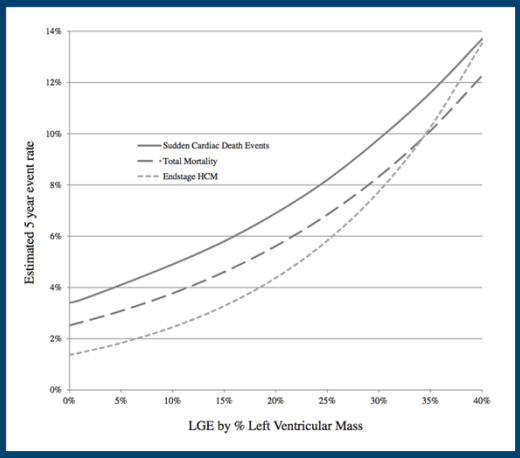
Prognostic Value of Quantitative Contrast-Enhanced Cardiovascular Magnetic Resonance for the Evaluation of Sudden Death Risk in Patients With Hypertrophic Cardiomyopathy

Raymond H. Chan, MD, MPH; Barry J. Maron, MD; Iacopo Olivotto, MD; Michael J. Pencina, PhD; Gabriele Egidy Assenza, MD; Tammy Haas, RN; John R. Lesser, MD; Christiane Gruner, MD; Andrew M. Crean, MD; Harry Rakowski, MD; James E. Udelson, MD; Ethan Rowin, MD; Massimo Lombardi, MD; Franco Cecchi, MD; Benedetta Tomberli, MD; Paolo Spirito, MD; Francesco Formisano, MD; Elena Biagini, MD; Claudio Rapezzi, MD; Carlo Nicola De Cecco, MD; Camillo Autore, MD; E. Francis Cook, PhD; Susie N. Hong, MD; C. Michael Gibson, MD, MS; Warren J. Manning, MD; Evan Appelbaum, MD; Martin S. Maron, MD

1293 patients
Follow-up of 3.3 years
SCD: 37 patients (3%)
DE > 15%: risk 2x higher for SCD





Circulation. 2014;130:484-495

55

Prognostic Value of LGE-CMR in HCM

A Meta-Analysis

Zhen Weng, PhD,[a] Jialu Yao, MD,[b] Raymond H. Chan, MD, MPH,[c] Jun He, MD,[a] Xiangjun Yang, MD, PhD,[b] Yafeng Zhou, MD, PhD,[b] Yang He, MD[a]

JACC: CARDIOVASCULAR IMAGING, VOL. 9, NO. 12, 2016

TABLE 1 Characteristics of Studies Complying With PRISMA Guidelines

First Author (Ref. #)	Patients Enrolled	Mean Follow-Up Time, Months	Design	Field Strength	Scar Assessment by LGE	Population Included
Bruder et al. (19)	220	36.3	Prospective, single center	1.5-T	Visual assessment of LGE by 2 reviewers	Patients with known or suspected HCM who underwent CMR
Rubinshtein et al. (20)	424	43.0	Retrospective, single center	1.5-T	Visual assessment of LGE by 2 reviewers	Patients with HCM who underwent ce-MRI
Chan et al. (11)	1,293	40.2	Prospective, multiple center	1.5-T	Visual assessment of LGE by 2 reviewers	Patients with HCM who underwent CMR
Hen et al. (12)	345	21.8	Retrospective, single center	1.5-T	Visual assessment of LGE by 3 reviewers	Patients with HCM who underwent CMR
Ismail et al. (13)	711	42.6	Prospective, single center	1.5-T	NR	Consecutive patients with HCM referred for CMR

ce-MRI = contrast-enhanced magnetic resonance imaging; CMR = cardiac magnetic resonance; HCM = hypertrophic cardiomyopathy; LGE = late gadolinium enhancement; NR = not reported; PRISMA = Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

TABLE 36.2 Adjusted Hazard Ratios for Sudden Cardiac Death Using Data From Chan et al.[37] and Ismail et al.[41]

LGE %	Pooled $HR_{adjusted}$ Point Estimate	95% CI
0	1.0	—
1	1.03	1.01–1.05
10	1.36	1.10–1.69
15	1.59	1.15–2.20
20	1.86	1.21–2.86
30	2.5	1.33–4.83
40	3.4	1.46–8.16

TABLE 36.1 Metaanalysis for Late Gadolinium Enhancement and Sudden Death

Study	Unadjusted HR, per 10% LV Mass	95% CI	P	Adjusted HR, per 10% LV Mass	95% CI	P
SCD						
Bruder et al.[39]	1.7	1.2–2.5	< .01	NA	NA	NA
Ismail et al.[41]	1.5	1.1–2.1	.007	1.2	0.8–1.7	.2
Chan et al.[37]	1.5	1.2–1.8	< .0001	1.4	1.1–1.9	.002
Pooled	1.5	1.3–1.8	< .0001	1.3	1.1–1.6	.005
HF Death						
Ismail et al.[41]	1.5	1.0–2.1	.028	NA	NA	NA
Chan et al.[37]	1.7	1.1–2.7	.01	NA	NA	NA

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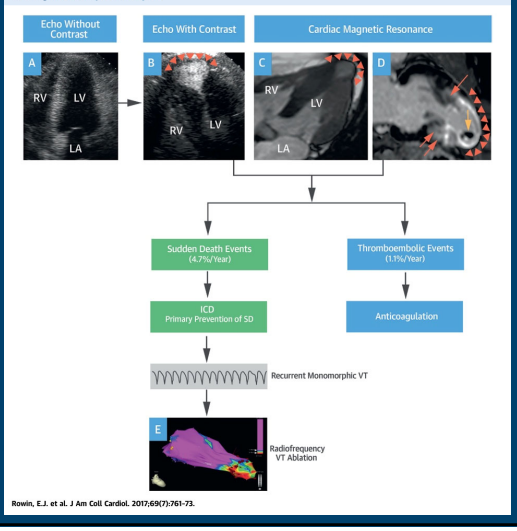
JACC VOL. 69, NO. 7, 2017
FEBRUARY 21, 2017:761–73

Hypertrophic Cardiomyopathy With Left Ventricular Apical Aneurysm

Implications for Risk Stratification and Management

Ethan J. Rowin, MD,[a] Barry J. Maron, MD,[a] Tammy S. Haas, RN,[b] Ross F. Garberich, MS,[b] Weijia Wang, MD,[a] Mark S. Link, MD,[a] Martin S. Maron, MD[a]

CENTRAL ILLUSTRATION Diagnosis, Expanded Risk Stratification, and Management Implications in HCM Patients With High-Risk LV Apical Aneurysms



Rowin, E.J. et al. J Am Coll Cardiol. 2017;69(7):761-73.

FIGURE 5 Survival Free of HCM-Related Adverse Events

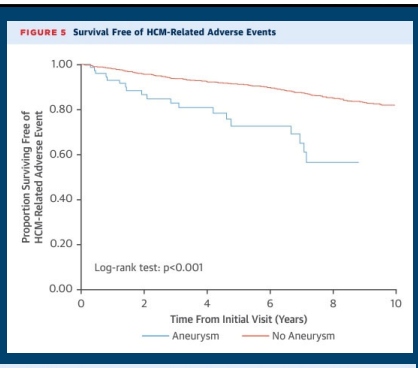
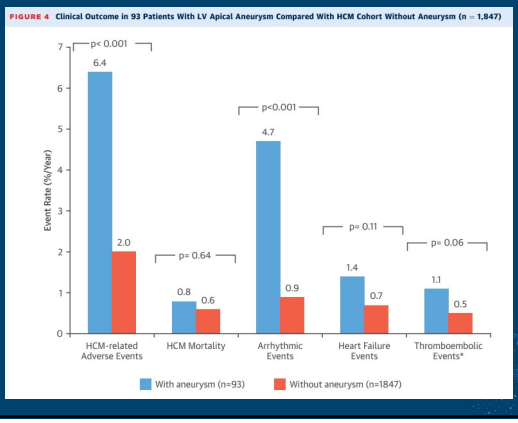


FIGURE 4 Clinical Outcome in 93 Patients With LV Apical Aneurysm Compared With HCM Cohort Without Aneurysm (n = 1,847)



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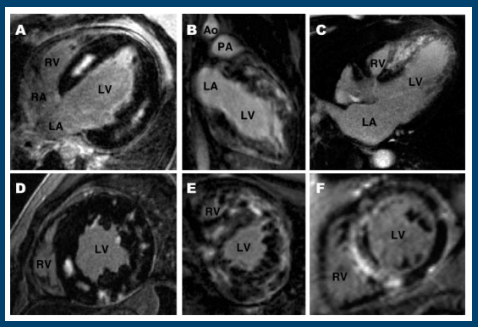
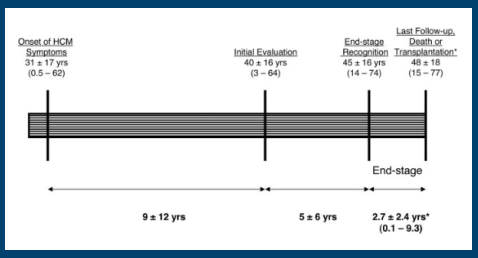
Prevalence, Clinical Profile, and Significance of Left Ventricular Remodeling in the End-Stage Phase of Hypertrophic Cardiomyopathy

Kevin M. Harris, MD; Paolo Spirito, MD; Martin S. Maron, MD; Andrey G. Zenovich, MSc; Francesco Formisano, MD; John R. Lesser, MD; Shannon Mackey-Bojack, MD; Warren J. Manning, MD; James E. Udelson, MD; Barry J. Maron, MD

(*Circulation*. 2006;114:216-225.)



- ES-HCM prevalence 3.5%.
- 33 patients who developed ES were diagnosed at an earlier age, had more severe symptoms, larger LV cavity, thicker septum, and less frequent out-flow gradients 30 mm Hg at rest.
- ES is an unfavorable complication (mortality rate 11% per year) and a sudden death risk factor.



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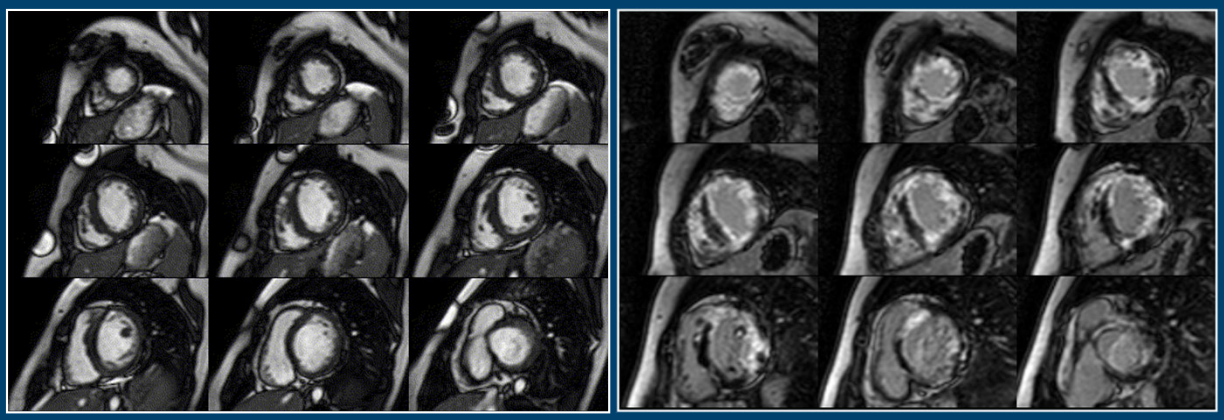
End-stage Hypertrophic Cardiomyopathy



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CLINICAL PRACTICE GUIDELINE

VOL. 83, NO. 23, 2024

2024 AHA/ACC/AMSSM/HRS/PACES/SCMR Guideline for the Management of Hypertrophic Cardiomyopathy

A Report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines

TABLE 8 Clinical Sudden Death Risk Factors for Adults and Children With HCM

Family history of sudden death from HCM	Sudden death judged definitively or likely attributable to HCM in ≥1 first-degree or close relatives who are ≤50 y of age. Close relatives would generally be second-degree relatives; however, multiple SCDs in tertiary relatives should also be considered relevant.[30,31]
Massive LVH	Wall thickness ≥30 mm in any segment within the chamber by echocardiography or CMR imaging; consideration for this morphologic marker is also given to borderline values of ≥28 mm in individual patients at the discretion of the treating cardiologist. For pediatric patients with HCM, an absolute or z-score threshold for wall thickness has not been established; however, a maximal wall thickness that corresponds to a z-score ≥20 (and >10 in conjunction with other risk factors) appears reasonable.[32,33]
Unexplained syncope	≥1 unexplained episodes involving acute transient loss of consciousness, judged by history unlikely to be of neurocardiogenic (vasovagal) etiology, not attributable to LVOTO, and especially when occurring within 6 mo of evaluation (events beyond 5 y in the past do not appear to have relevance).[34]
HCM with LV systolic dysfunction	Systolic dysfunction with EF <50% by echocardiography or CMR imaging.[24,27]
LV apical aneurysm	Apical aneurysm defined as a discrete thin-walled dyskinetic or akinetic segment with transmural scar or LGE of the most distal portion of the LV chamber, independent of size. (In children, apical aneurysm is uncommon, and the risk has not been studied.)[15,16]
Extensive LGE on CMR imaging	Extensive LGE, representing replacement fibrosis, either quantified or estimated by visual inspection, comprising ≥15% of LV mass (extent of LGE conferring risk has not been defined in children).[9-11,20-22,25]
NSVT on ambulatory monitor	≥3 beats at ≥120 bpm has generally been used in studies. It would seem most appropriate to place greater weight on NSVT as a risk marker when runs are frequent (eg, ≥3), longer (eg, ≥10 beats), or faster (eg, ≥200 bpm) occurring usually over 24 to 48 h of monitoring. For pediatric patients, a VT rate that exceeds the baseline sinus rate by >20% is considered significant.[35-37]
Genotype status	Genotype-positive status (ie, harboring a putatively disease-causing pathogenic/likely pathogenic variant) is associated with higher SCD risk in pediatric patients with HCM.[12,14]

Recommendations for CMR Imaging

Referenced studies that support the recommendations are summarized in Online Data Supplement 4.

COR	LOE	Recommendations
1	B-NR	1. For patients suspected to have HCM in whom echocardiography is inconclusive, CMR imaging is indicated for diagnostic clarification.[1-7]
1	B-NR	2. For patients with LVH in whom there is a suspicion of alternative diagnoses, including infiltrative or storage disease as well as athlete's heart, CMR imaging is useful[1-7] (Figure 1).
1	B-NR	3. For patients with HCM who are not otherwise identified as high risk for SCD, or in whom a decision to proceed with ICD remains uncertain after clinical assessment that includes personal/family history, echocardiography, and ambulatory electrocardiographic monitoring, CMR imaging is beneficial to assess for maximum LV wall thickness, ejection fraction (EF), LV apical aneurysm, and extent of myocardial fibrosis with LGE.[1-15]
1	B-NR	4. For patients with obstructive HCM in whom the anatomic mechanism of obstruction is inconclusive on echocardiography, CMR imaging is indicated to inform the selection and planning of SRT.[16-20]
2b	C-EO	5. For patients with HCM, repeat contrast-enhanced CMR imaging on a periodic basis (every 3 to 5 years) for the purpose of SCD risk stratification may be considered to evaluate changes in LGE and other morphologic changes, including EF, development of apical aneurysm, or LV wall thickness (Figure 1, Table 7).

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Future Perspectives

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ORIGINAL RESEARCH

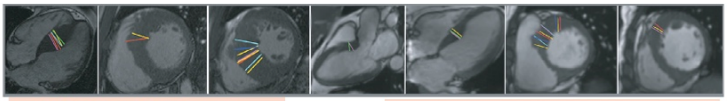
JACC: CARDIOVASCULAR IMAGING, VOL. 14, NO. 11, 2021
NOVEMBER 2021:2123–2134

Maximal Wall Thickness Measurement in Hypertrophic Cardiomyopathy

Biomarker Variability and its Impact on Clinical Care

Gabriella Captur, MD, PhD, MSc,[a,b,c] Charlotte H. Manisty, MBBS, PhD,[c,d] Betty Raman, MBBS, PhD,[e] Alberto Marchi, MD,[f] Timothy C. Wong, MD,[g,h] Rina Ariga, MBBS, PhD,[g] Anish Bhuva, MBBS, PhD,[c,d] Elizabeth Ormondroyd, MSc,[i] Ilaria Lobascio, MD, PhD,[d] Claudia Camaioni, MD,[d] Savvas Loizos, MD,[d] Jenade Bonsu-Ofori, MSc,[j] Aslan Turer, PhD,[k] Vlad G. Zaha, MD, PhD,[k] João B. Augutsto, MD,[c,d] Rhodri H. Davies, MBBS, PhD,[c,d] Andrew J. Taylor, PhD,[l,m,n] Arthur Nasis, MD,[o] Mouaz H. Al-Mallah, MD,[p] Sinitsyn Valentin, MD,[q] Diego Perez de Arenaza, MD,[r] Vimal Patel, MD, PhD,[r] Mark Westwood, MBBS,[d] Steffen E. Petersen, MBBS, PhD,[d,s] Chunming Li, PhD,[t,u] Lijun Tang, MD,[v] Shiro Nakamori, PhD,[w] Reza Nezafat, PhD,[w] Raymond Y. Kwong, PhD,[x] Carolyn Y. Ho, PhD,[x] Alan G. Fraser, PhD,[y] Hugh Watkins, MD, PhD,[i] Perry M. Elliott, MBBS, MD,[c,d] Stefan Neubauer,[y] Guy Lloyd, DPhil,[c,d] Iacopo Olivotto, MD, PhD,[f] Petros Nihoyannopoulos, MD,[e] James C. Moon, MBBS, MD[c,d]

CENTRAL ILLUSTRATION High Variability of the Maximal Wall Thickness Biomarker in Hypertrophic Cardiomyopathy Affects Clinical Care



70 Readers at 6 Conferences

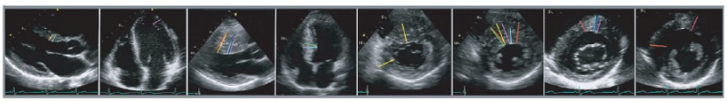
MWT measurements in paired Echo/CMR HCM datasets

No standardized approach

High measurement variability

Simulations assess impact of MWT variability on ESC risk algorithm

ICD decisions in 1 in 7 HCM patients potentially impacted



Captur, G. et al. J Am Coll Cardiol Img. 2021;14(11):2123-2134.

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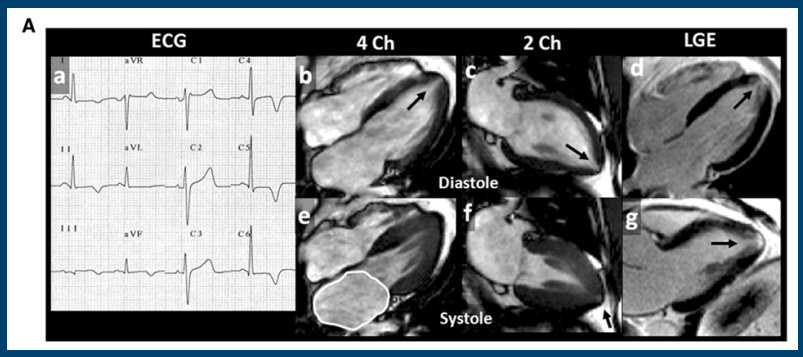
Diagnosis of apical hypertrophic cardiomyopathy: T-wave inversion and relative but not absolute apical left ventricular hypertrophy ☆

Andrew S. Flett [a], Viviana Maestrini [b], Don Milliken [b], Mariana Fontana [b], Thomas A. Treibel [b], Rami Harb [b], Daniel M. Sado [b,c], Giovanni Quarta [d,f], Anna Herrey [b], James Sneddon [e], Perry Elliott [b,c], William McKenna [b,c], James C. Moon [b,c,*]

[a] Department of Cardiology, University Hospital Southampton, Tremona Road, Southampton SO166YD, United Kingdom
[b] The Heart Hospital, part of University College London Hospitals NHS Trust, 16-18 Westmoreland Street, London W1G 8P, United Kingdom
[c] Institute of Cardiovascular Science, University College London, United Kingdom
[d] Department of Cardiology, S Andrea Hospital, Universtiy Sapienza Rome, Italy
[e] East Surrey Hospital, Canada Avenue, Redhill RH1 5RH, United Kingdom
[f] Cardiovascular Department, AO Papa Giovanni XXIII, Bergamo, Italy

International Journal of Cardiology 183 (2015) 143–148



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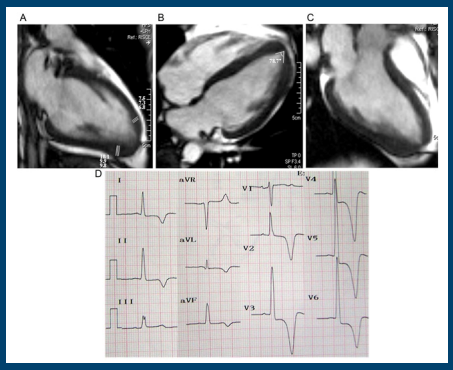
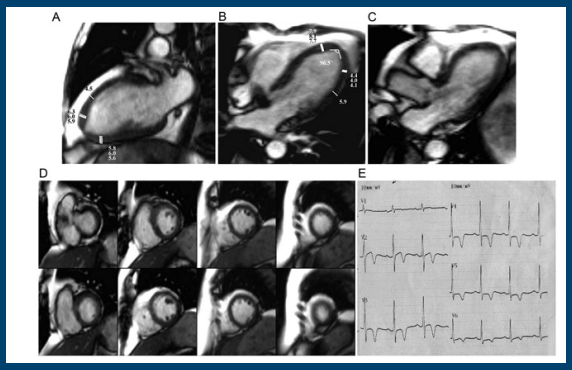
CMR assessment of the left ventricle apical morphology in subjects with unexplainable giant T-wave inversion and without apical wall thickness ≥15 mm

Bailin Wu[1,2,3,4], Minjie Lu[1,2,3†*], Yan Zhang[1,2,3], Bo Song[5], Jian Ling[1,2,3], Jinghan Huang[6], Gang Yin[1,2,3], Tian Lan[1,2,3], Linlin Dai[1,2,3], Lei Song[7], Yong Jiang[8], Hao Wang[8], Zuoxiang He[9], Jongmin Lee[10], Hwan Seok Yong[11], Mehul B. Patel[12], and Shihua Zhao[1,2,3†*]

European Heart Journal – Cardiovascular Imaging (2017) **18**, 186–194





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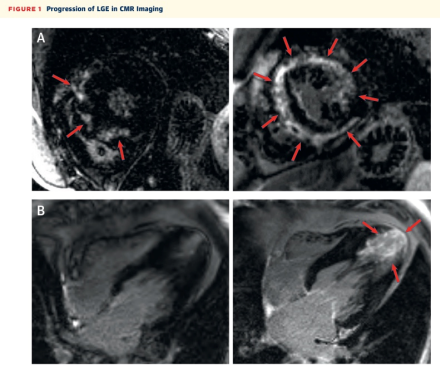
ORIGINAL RESEARCH

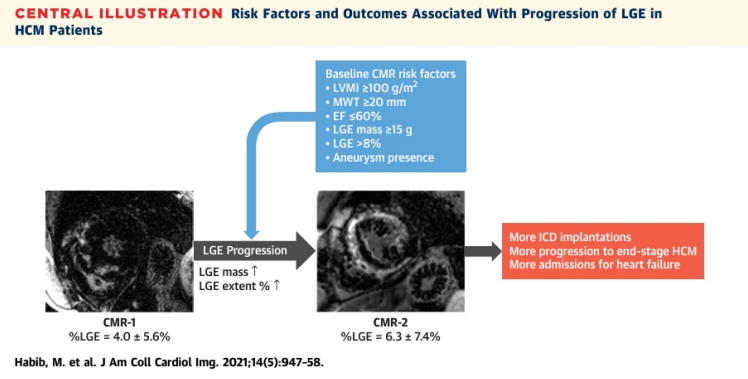
JACC: CARDIOVASCULAR IMAGING, VOL. 14, NO. 5, 2021
MAY 2021:947–58

Progression of Myocardial Fibrosis in Hypertrophic Cardiomyopathy

A Cardiac Magnetic Resonance Study

Manhal Habib, MD, PhD,[a] Arnon Adler, MD,[a] Kimia Fardfini, BSc,[a] Sara Hoss, MD,[a] Kate Hanneman, MD,[a] Ethan J. Rowin, MD,[b] Martin S. Maron, MD,[b] Barry J. Maron, MD,[b] Harry Rakowski, MD,[a] Raymond H. Chan, MD, MPH[a]





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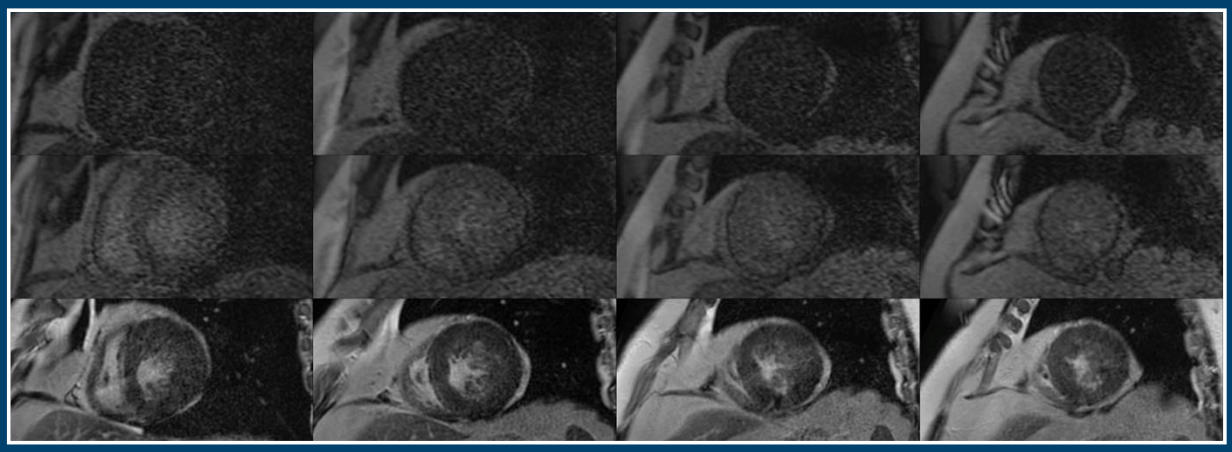
Small-vessel ischemia



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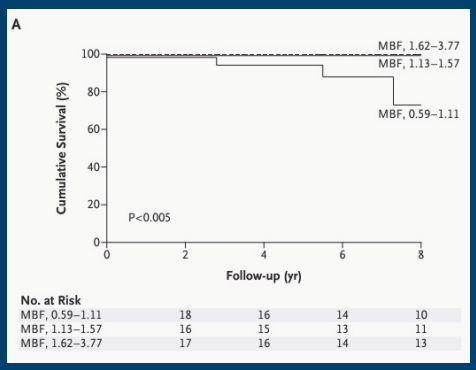
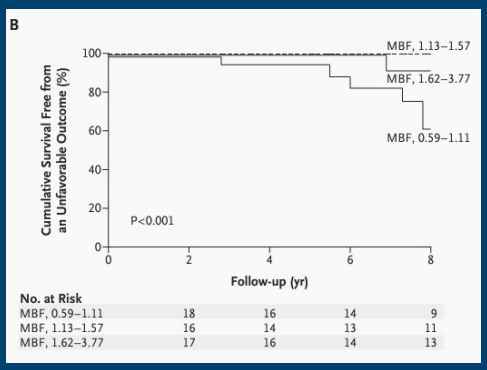
ORIGINAL ARTICLE

Coronary Microvascular Dysfunction and Prognosis in Hypertrophic Cardiomyopathy

Franco Cecchi, M.D., Iacopo Olivotto, M.D., Roberto Gistri, M.D., Roberto Lorenzoni, M.D., Giampaolo Chiriatti, M.D., and Paolo G. Camici, M.D.

N Engl J Med 2003;349:1027-35.





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RESEARCH Open Access

Prevalence and clinical significance of cardiovascular magnetic resonance adenosine stress-induced myocardial perfusion defect in hypertrophic cardiomyopathy

Eun Kyoung Kim[1], Sang-Chol Lee[1*], Sung-A Chang[1], Shin-Yi Jang[1], Sung Mok Kim[2], Sung-Ji Park[1], Jin-Oh Choi[1], Seung Woo Park[1], Eun-Seok Jeon[1] and Yeon Hyeon Choe[2*]

Kim et al. Journal of Cardiovascular Magnetic Resonance (2020) 22:30

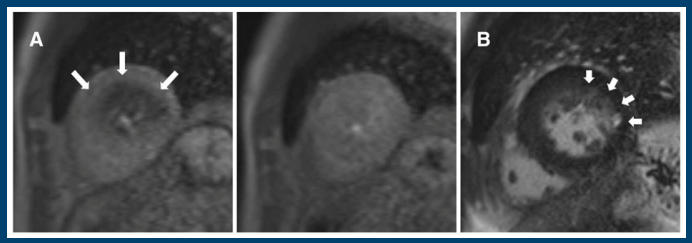

Table 5 Multivariate analysis of clinical and CMR findings between the stress-positive and negative groups

Variables	OR (95% Confidence Interval)	*P* value
Age	0.98 (0.95–1.01)	0.233
Gender, male vs. female	0.12 (0.03–0.58)	0.008
History of diabetes	1.90 (0.15–20.6)	0.598
History of smoking	0.21	
Ex-smoker	1.23 (0.36–4.22)	0.740
Current smoker	0.75 (0.21–2.71)	0.657
Atypical chest pain	0.30 (0.06–1.53)	0.148
History of syncope	1.95 (0.29–12.9)	0.490
NSVT on 24-h holter	6.38 (1.03–39.6)	0.047
Inadequate blood pressure response to exercise	0.66 (0.18–2.36)	0.523
LV outflow tract obstruction	0.32 (0.08–1.26)	0.102
Presence of apical aneurysm	5.58 (1.12–27.7)	0.036
LGE volume	0.99 (0.97–1.03)	0.946
LV mass index	1.03 (1.01–1.06)	0.022

OR Odds ratio. *P* value were calculated with multiple logistic regression
LV Left ventricular, *NSVT* Non-sustained ventricular tachycardia, *LGE* Late gadolinium enhancement

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Circulation

Circulation. 2021;143:606–608.

RESEARCH LETTER

Mavacamten Favorably Impacts Cardiac Structure in Obstructive Hypertrophic Cardiomyopathy

EXPLORER-HCM Cardiac Magnetic Resonance Substudy Analysis

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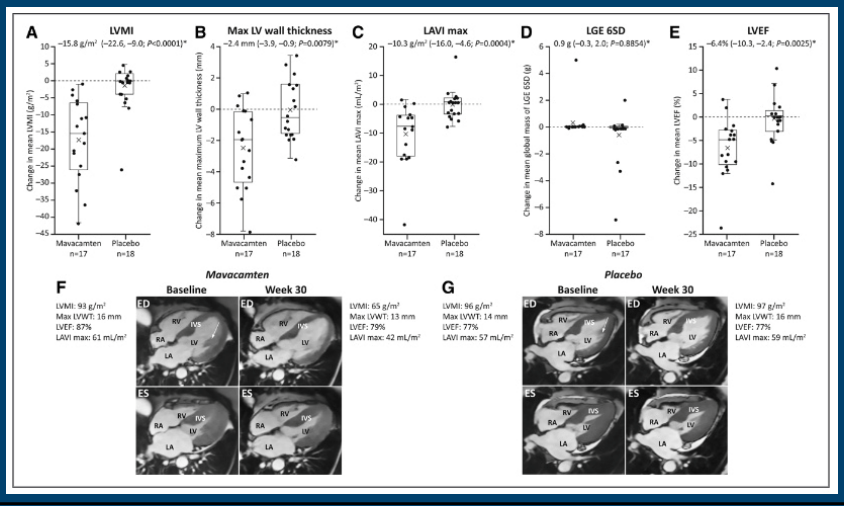

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JACC VOL. 84, NO. 19, 2024
NOVEMBER 5, 2024:1806–1817

Effect of Aficamten on Cardiac Structure and Function in Obstructive Hypertrophic Cardiomyopathy

SEQUOIA-HCM CMR Substudy

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EDITORIAL COMMENT

Unveiling the Unseen

Myosin Inhibitors Cause Reverse Remodeling of Left Ventricular Macro and Microstructure by CMR

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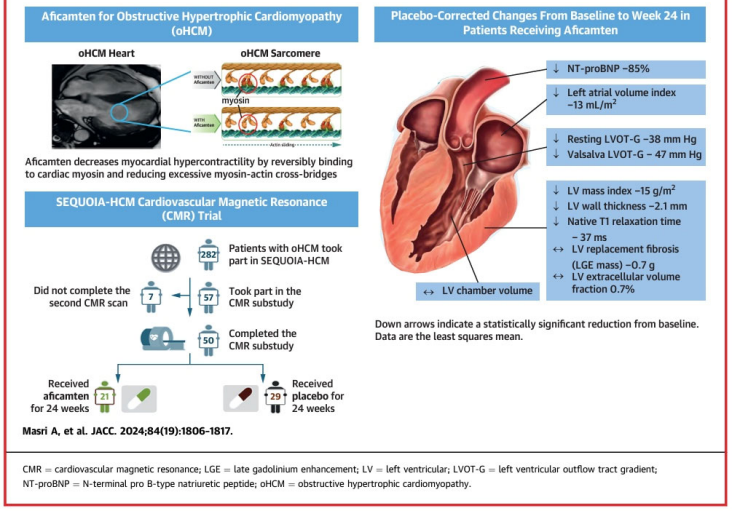
CENTRAL ILLUSTRATION Effect of Aficamten on Cardiac Structure and Function in Patients With Obstructive Hypertrophic Cardiomyopathy: The SEQUOIA-HCM CMR Trial



Masri A, et al. JACC. 2024;84(19):1806–1817.

CMR = cardiovascular magnetic resonance; LGE = late gadolinium enhancement; LV = left ventricular; LVOT-G = left ventricular outflow tract gradient; NT-proBNP = N-terminal pro B-type natriuretic peptide; oHCM = obstructive hypertrophic cardiomyopathy.

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Final considerations

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Thank you

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