KLİNİKİ MÜŞAHİDƏ AÇIQ GİRİŞ (OPEN ACCESS)

HYPERTROPHY: A COMPARATIVE CASE SERIES

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ABSTRACT

Left ventricular hypertrophy (LVH) represents a common imaging phenotype with diverse etiologies. Differentiating hypertrophic cardiomyopathy, infiltrative disease, metabolic disorders and pressure-overload remodeling remains challenging using echocardiography alone. This comparative case series demonstrates how multiparametric cardiac magnetic resonance (CMR), integrating ventricular morphology, native T1 mapping and late gadolinium enhancement (LGE), improves etiological diagnosis. Four representative patients with unexplained LVH underwent standardized 3.0-T CMR. Distinct imaging signatures enabled differentiation between cardiac amyloidosis, genetically confirmed mitochondrial cardiomyopathy, pressure-overload hypertrophy secondary to aortic valve disease and hypertrophic cardiomyopathy. These cases emphasize that LVH is a phenotype rather than a diagnosis and illustrate the incremental diagnostic value of comprehensive tissue characterization.

Introduction

Left ventricular hypertrophy is encountered in a broad spectrum of cardiovascular diseases. Although increased wall thickness is easily recognized on echocardiography, substantial overlap exists among different disease entities. CMR has emerged as the reference imaging modality because it combines accurate assessment of ventricular morphology and function with myocardial tissue characterization. Native T1 mapping and LGE provide disease-specific biomarkers that frequently establish the correct diagnosis and influence subsequent management. The purpose of this case series is to compare four clinically distinct causes of LVH using a uniform multiparametric CMR approach.

Aim

To demonstrate how multiparametric CMR differentiates various causes of left ventricular hypertrophy using representative comparative clinical cases.

Methods

The present retrospective educational case series included four representative patients referred for evaluation of unexplained LVH. Clinical presentation, electrocardiography, echocardiography and standardized 3.0-T CMR findings were reviewed. Final diagnoses were established through integration of clinical and imaging findings, with genetic confirmation in the mitochondrial cardiomyopathy case. Representative images were selected to highlight distinguishing CMR features.

Results – Representative Cases

Representative Case A – Cardiac Amyloidosis

Age/Sex: 60-year-old male

Presentation: Fatigue, dyspnea, complete AV block, ventricular tachycardia.

Echocardiography: LV wall thickness 20 mm, LVEF 60%; suspected HCM.

CMR Findings:

Concentric LV hypertrophy (maximum wall thickness 25 mm)

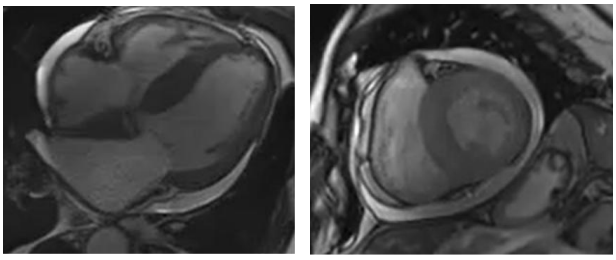
Native T1: 1355–1614 ms

Diffuse circumferential subendocardial LGE

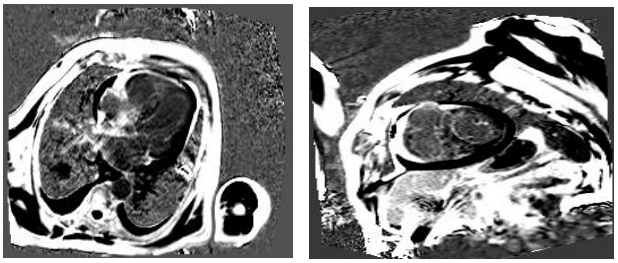
Final Diagnosis: Cardiac amyloidosis.

Azərbaycan Kardiologiya Cəmiyyəti

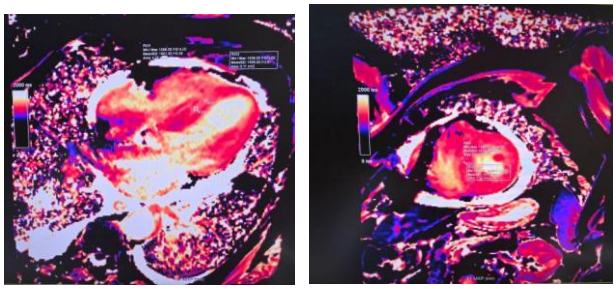
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A. cine 4 ch B.cine SA



C. LGE 4ch D. LGE SA



F.T1map 4CH G.T1map SA

Representative Case B – Genetically Confirmed Mitochondrial Cardiomyopathy

Age/Sex: 20-year-old male

Presentation: Recurrent exertional syncope and progressive exertional dyspnea.

Echocardiography: Mild LV hypertrophy (15 mm); suspected HCM.

CMR Findings:

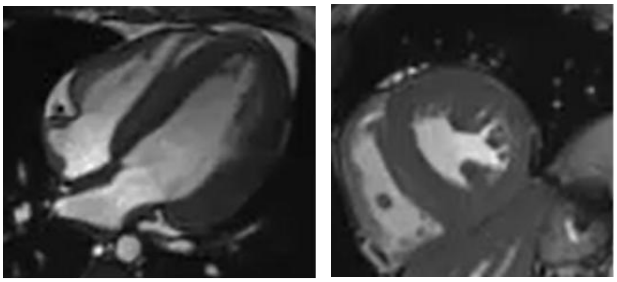
Maximum wall thickness 15–17 mm

Low native T1: 1079–1125 ms

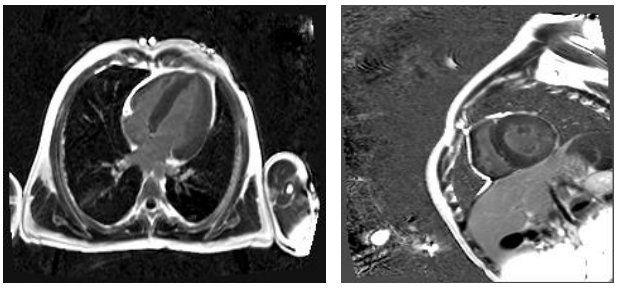
Focal basal inferolateral mid-wall LGE

Diagnostic confirmation: Genetic testing confirmed mitochondrial cardiomyopathy.

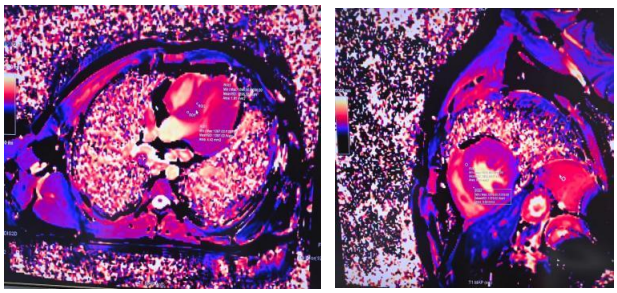
Final Diagnosis: Genetically confirmed mitochondrial cardiomyopathy.



A.cine 4 ch B. cine SA



C. LGE 4ch D.LGE SA



G. T1map 4ch F. T1mapS

Representative Case C – Pressure-Overload LVH Secondary to Aortic Valve Disease

Age/Sex: 55-year-old male

Presentation: Progressive exertional dyspnea.

Echocardiography: Severe aortic valve disease (AS/AR), IVS 19 mm; suspected HCM.

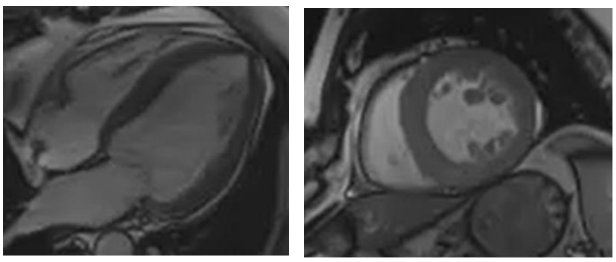
CMR Findings:

LV remodeling with maximum wall thickness 15 mm

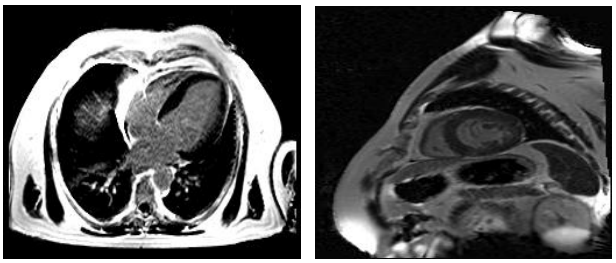
Native T1: 1263–1308 ms

No LGE

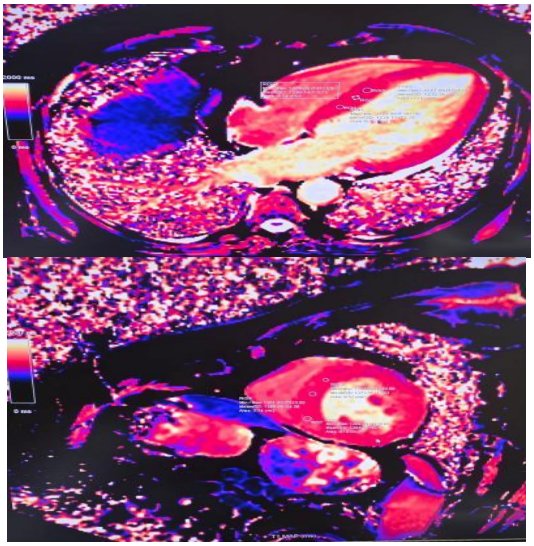
Final Diagnosis: Pressure-overload LV hypertrophy secondary to aortic valve disease



A.cine 4ch B.cine SA



C. LGE 4ch D. LGE SA



E. T1 map 4ch G. T1map SA

Representative Case D – Hypertrophic Cardiomyopathy

Age/Sex: 58-year-old male

Presentation: Exertional dyspnea and chest pain.

Echocardiography: Asymmetric septal hypertrophy (24–26 mm), systolic anterior motion (SAM), LVOT gradient 75 mmHg.

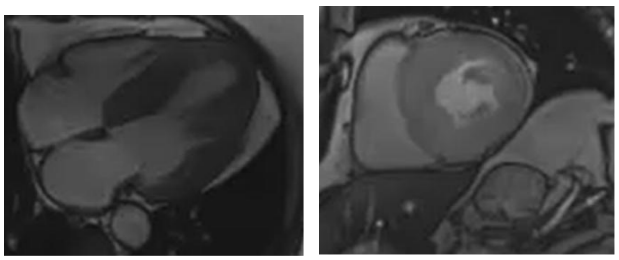
CMR Findings:

Asymmetric hypertrophy

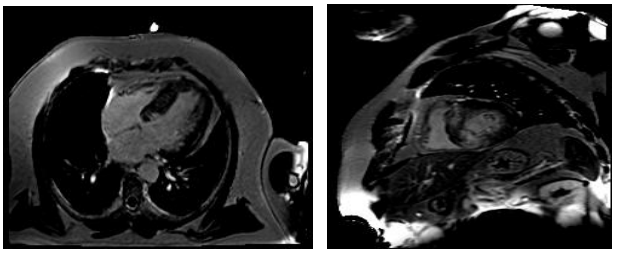
Native T1: 1357–1451 ms

Patchy non-ischemic LGE at the RV insertion points with additional ischemic enhancement related to coexisting coronary artery disease

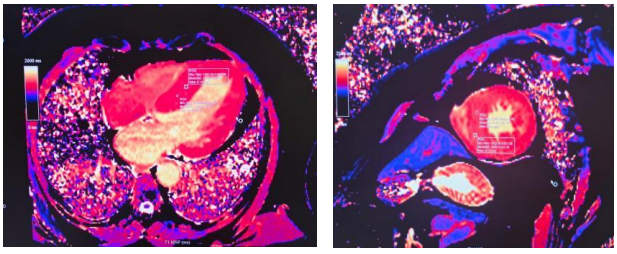
Final Diagnosis: Hypertrophic cardiomyopathy with coexisting obstructive coronary artery disease.



A. Cine 4ch B. Cine SA



C. LGE 4ch D. LGE SA



E. T1map4ch G.T1mapSA

Comparative summary

Disease	Maximum LV Wall Thickness	LVEF (%)	Native T1 (ms)	Characteristic LGE Pattern	Key CMR Features
Cardiac amyloidosis	25 mm	60	1355–1614	Diffuse circumferential subendocardial LGE	Markedly elevated native T1 with diffuse infiltrative enhancement

Mitochondrial cardiomyopathy	15–17 mm	59	1079–1125	Basal inferolateral midwall LGE	Low native T1 with focal non-ischemic fibrosis; genetically confirmed
Pressure-overload LVH (aortic valve disease)	15 mm	60	1263–1308	No LGE	Normal native T1 without replacement fibrosis
Hypertrophic cardiomyopathy	24–26 mm	54	1357–1451	Patchy non-ischemic LGE at the RV insertion points with additional ischemic scar	Elevated native T1 with characteristic non-ischemic fibrosis pattern

Discussion

Although all patients initially presented with LVH, multiparametric CMR demonstrated unique disease-specific imaging signatures. Diffuse subendocardial LGE with markedly elevated native T1 supported cardiac amyloidosis, whereas low native T1 and focal inferolateral fibrosis suggested mitochondrial cardiomyopathy. Absence of LGE and near-normal tissue characterization favored pressure-overload remodeling, while asymmetric hypertrophy with RV insertion-point fibrosis was characteristic of hypertrophic cardiomyopathy. These findings illustrate the importance of integrating morphology with tissue characterization rather than relying solely on wall thickness.

Conclusion

Multiparametric CMR is a comprehensive non-invasive imaging technique for the etiological evaluation of left ventricular hypertrophy. By combining ventricular morphology with myocardial tissue characterization, it enables accurate differentiation of diverse LVH phenotypes and facilitates