The Egyptian Journal of Hospital Medicine (January 2024) Vol. 94, Page 285-290

The Additive Value of Cardiac MRI in Cases of Cardiomyopathy

Shaimaa Abdelhamid Hassanein, Ahmed Hussein Shehata Elsobky*,
Elsayed Elmekkawy Elsayed, Yasmin Hosney Hemeda
Department of Radiodiagnosis, Faculty of Medicine, Menoufia University, Egypt
***Corresponding author:** Ahmed Hussein Shehata Elsobky, **Mobile:** (+20) 01000104072, **E-mail:** ahmedelsobky10@yahoo.com

ABSTRACT

Background: Worldwide, myocardial diseases are major contributors to patient morbidity and death. Cardiovascular Magnetic Resonance (CMR) has gained recognition as a useful clinical technique that may evaluate ventricular function and volumes, myocardial fibrosis, iron loading, flow measurement, tissue characterisation, and myocardial perfusion imaging all in a single scan scenario.

Objective: This study aimed to identify the role and diagnostic potential of cardiac magnetic resonance imaging to diagnose cardiomyopathy.

Patients and methods: A cross-sectional study that was conducted through the period from October 2020 to May 2022 at Radio Diagnosis Department, National Heart Institute. The study involved 50 patients who had clinical suspicion of cardiomyopathy. They were referred from Cardiology Department. The mean age of patients was 57.42 ± 6.45 years.

Results: There was a significant difference between Echo and Cardiac MRI regarding ejection fraction and end diastolic volume among the studied patients. Also, EF% was significantly higher with cardiac MRI (45.60 ± 8.12%) compared to Echo (43.00 ± 7.49%), with a mean difference of 2.60 ± 2.42%. Furthermore, EDV/ml was significantly higher with cardiac MRI (161.54 ± 48.14 ml) compared to Echo (158.50 ± 46.38 ml), with a mean difference of 3.04 ± 5.26 ml.

Conclusion: Cardiac magnetic resonance technique was more accurate for diagnosis among patients suspicioned of cardiomyopathy compared to echocardiography.

Keywords: Echocardiography, Cardiomyopathy**,** Hypertrophic cardiomyopathy, Magnetic resonance imaging**.**

INTRODUCTION

With an incidence of 1:500 in the general population, hypertrophic cardiomyopathy (HCM) is the most prevalent hereditary cardiomyopathy [1, 2]. The symptoms of HCM include myocardial fibrosis, coronary microvascular dysfunction, myocardial fibre disarray, and unexplained, typically asymmetric left ventricular (LV) enlargement [3].

Even with an autosomal dominant mode of transmission, penetrance is age-dependent and imperfect, and phenotypic manifestation is very varied [4].

Currently, over 1400 mutations are affecting different sarcomere components have been shown to be the cause of this cardiac condition [5]. Most HCM patients have a lifetime that is almost normal, and the condition's natural history is often benign [6, 7]. However, a number of individuals suffer with limited symptoms and unfavourable outcomes as a result of ventricular and supraventricular arrhythmias, myocardial ischemia, systolic and/or diastolic heart failure, and sudden cardiac death [8].

In HCM, cardiac imaging continues to be the mainstay for clinical diagnosis and treatment recommendations [9].

Due to its broad availability and inexpensive cost, traditional 2-D transthoracic echocardiography (TTE) has been the primary imaging modality for evaluation of patients with HCM for decades [10]. Nonetheless, CMR has demonstrated more penetration in clinical practice in recent years [11]. So, the aim of the current study was to identify the role and diagnostic potential of CMR imaging to diagnose cardiomyopathy.

PATIENTS AND METHODS

A cross-sectional study that was conducted through the period from October 2020 to May 2022 at Radio Diagnosis Department, National Heart Institute. The study involved 50 patients who had clinical suspicion of cardiomyopathy. They were referred from Cardiology Department. The age of patients ranged from 39 to 84 years.

Inclusion criteria: Patients of both sexes who had clinical suspicion of cardiomyopathy on basis of echocardiography, ECG changes, ages of patients ranged from 39 to 84 years.

Exclusion criteria: Congenital heart disease, patients presenting with myocardial masses not related to cardiomyopathy, pericardial diseases and masses**,** patients with allergy to gadolinium contrast media and history of renal disease, individuals with pacemakers or metal implants (cerebral aneurysmal clips), claustrophobia, hemodynamic instability, atrial fibrillation, contraindications for MR imaging, patients who had one or more of absolute or relative MRI contraindication such as electronic, magnetic, mechanically activated implants and cochlear implants and examinations markedly compromised by motion artifacts.

All study participants were subjected to the following procedures:

Thorough history taking, which included personal history (Age, sex and past history included chest pain and other cardiac related symptoms). Physical and general examinations were carried out by our colleague.

Received: 05/07/2023
Accepted: 06/09/2023

MRI study of the heart as follows (MRI scan, Toshiba Vantage, Japan).

Patient's position:

- All patients included in this study were in the supine position, headfirst.

The Coil:

- Cardiac coil (sensitivity encoding 6 element phased-array coil, receive only) was used.
- The coil was positioned on the chest, with the bottom portion of the coil lying beneath the patient and the top section of the coil's midline lying directly below the sternoclavicular notch. Four straps were used to properly secure it to the patient. It was verified that the magnet was connected.
- Starting with the vertical long axis view and plan the horizontal long axis view.
- Working backwards from the horizontal long axis view to plan the short axis view.

Image acquisition:

- Scout vistas without holding your breath in any of the three orthogonal planes.
- At the LV level, planning the vertical long axis picture from the axial orthogonal image.

Cardiac MRI finding LV:

- As ejection fraction (EF %), end-diastolic volume (EDV ml), end-systolic volumes (ESV ml) and stroke volume (SV ml).

Ethical approval: Menoufia Faculty of Medicine Medical Ethics Committee gave its approval to this study. All participants gave written consents after receiving all information. The Helsinki Declaration was followed throughout the study's conduct.

Statistical analysis

SPSS version 21.0 was used to generate and analyse all of the data. For continuous variables, the presentation format was means ± standard deviation [SD], whereas percentages and relative frequency distributions were used for categorical variables. To compare continuous variables, the Mann-Whitney (t) test was employed. At $p \leq 0.05$ statistical significance was determined.

RESULTS

A total of 50 patients who had suspicion of cardiomyopathy were included in our study, their ages ranged from 43-70 years with a mean of 57.42 ± 6.45 years. Half of them were males, 18 patients were smokers (36%). Additionally, there were 46 patients who had comorbidities, 16 patients (32%) had hypertension, followed by 14 patients had DM (28%), and 12 patients (24%) had DM + HTN, and 4 patients (8%) were obese (Table 1).

Table (1): Demographic data of the studied patients

Variable	Studied patients (N=50)	
Age/year		
Mean ± SD	57.42 ± 6.45	
Range	43.00 -70.00	
Sex	**N**	**%**
Male	25	50.0
Female	25	50.0
Special habits		
No		
Smoker	32	64.0
	18	36.0
Comorbidity		
No	4	8.0
DM	14	28.0
HTN	16	32.0
DM - HTN	12	24.0
Obesity	4	8.0

DM: Diabetes Mellitus, **HTN**: hypertension

Also, according to symptoms distribution, there were 7 patients who had acute chest pain (14%), 13 patients (26%) had dyspnea, and 13 patients (26%) had lower limb edema (Table 2).

Table (2): Symptoms distribution among the studied patients

Symptoms	Studied patients (N=50)	
Acute chest pain	7	14.0
Cardiogenic shock	0	0.0
Dyspnea	13	26.0
Lower limb edema	13	26.0
Viral syndrome, acute epigastric pai	0	0.0
Fever, sore throat	0	0.0

Concerning ECG findings, there were 20 patients hadn't relevant (40%), 12 patients (24%) had Wide QRS, followed by 11 patients had ischemic changes (22%), and 4 patients (8%) had right bundle branch block, and 4 patients (6%) had strain pattern (Table 3).

Table (3): ECG findings among the studied patients

Variable	Studied patients (N=50)	
ECG findings		
- Ischemic changes	11	22.0
- Wide QRS	12	24.0
- Non relevant	20	40.0
- RBBB	4	8.0
- Strain pattern	3	6.0

QRS: Questionnaire on Resources and Stress, **ECG:** electrocardiogram, **RBBB:** right bundle branch block.

Furthermore, echo measurements indicated that ejection fraction was in range from 34 to 60% with a mean of 45.00 ± 7.49%. While, end diastolic volume was in range of 102 to 230 ml with a mean of 158.50 ± 46.38 ml (Table 4).

Table (4): Echo measurements among the studied patients

Echo measurements	Studied patients (N=50)	
	Mean ± SD	Median (IQR)
EF%	45.00 ± 7.49	34-60
EDV/ml	158.50 ± 46.38	102-230

EDV: End diastolic volume, **EF%**: ejection fraction, **IQR:** interquery range.

Also, cardiac MRI indicated that mean ejection fraction was 45.60 ± 8.12%. End diastolic volume was 161.54 ± 48.14ml, end-systolic volumes were 90.30 ± 34.51 ml, and stroke volume was 71.24 ± 16.32. Also, RA thrombus found in 4 patients (8%) (Table 5).

Table (5): Cardiac MRI measurements among the studied patients.

MRI measurements	Studied patients (N=50)	
	Mean ±SD	Median (IQR)
EF%	45.60±8.12	42(36.00-64)
EDV/ml	161.54±48.14	163(98.00-239)
ESV/ml	90.30±34.51	96(40.00-149)
SV/ml	71.24±16.32	76(50.00-90)
Others, N (%)		
RA thrombus	4	8.0%
No	46	92.0%

EF: Ejection fraction, **EDV:** End diastolic volume**, ESV:** end-systolic volumes, **SV:** stroke volume.

There was a significant difference between Echo and cardiac MRI regarding ejection fraction and end diastolic volume among the studied patients ($P < 0.05$). Also, EF% was significantly higher with cardiac MRI was 45.60 ± 8.12% compared to Echo (43.00 ± 7.49%), with a mean difference of 2.60 ± 2.42% (p=0.029).

Furthermore, EDV/ml was significantly higher with cardiac MRI (161.54 ± 48.14ml) compared to Echo (158.50 ± 46.38 ml), with a mean difference of 3.04 ± 5.26 ml (p=0.001), (Table 6 & Figures 1, 2).

Table (6): Comparison between Echo and Cardiac MRI among the studied patients

	Studied patients (N=50)			Sig. test	
	Echo	CMRI	Differences	U	*P value*
EF% Mean ±SD Median (IQR)	43.00± 7.494 45 (34-60)	45.60± 8.12 42 (36.00-64)	2.60± 2.42	3.75	0.029*
EDV/ml Mean ± SD Median (IQR)	158.50± 46.38 160 (102-230)	161.54± 48.14 163 (98.00-239)	3.04± 5.26	4.53	0.001 *

EF: Ejection fraction, **EDV:** End diastolic volume**, U:** Mann-Whitney test**, *Significant**

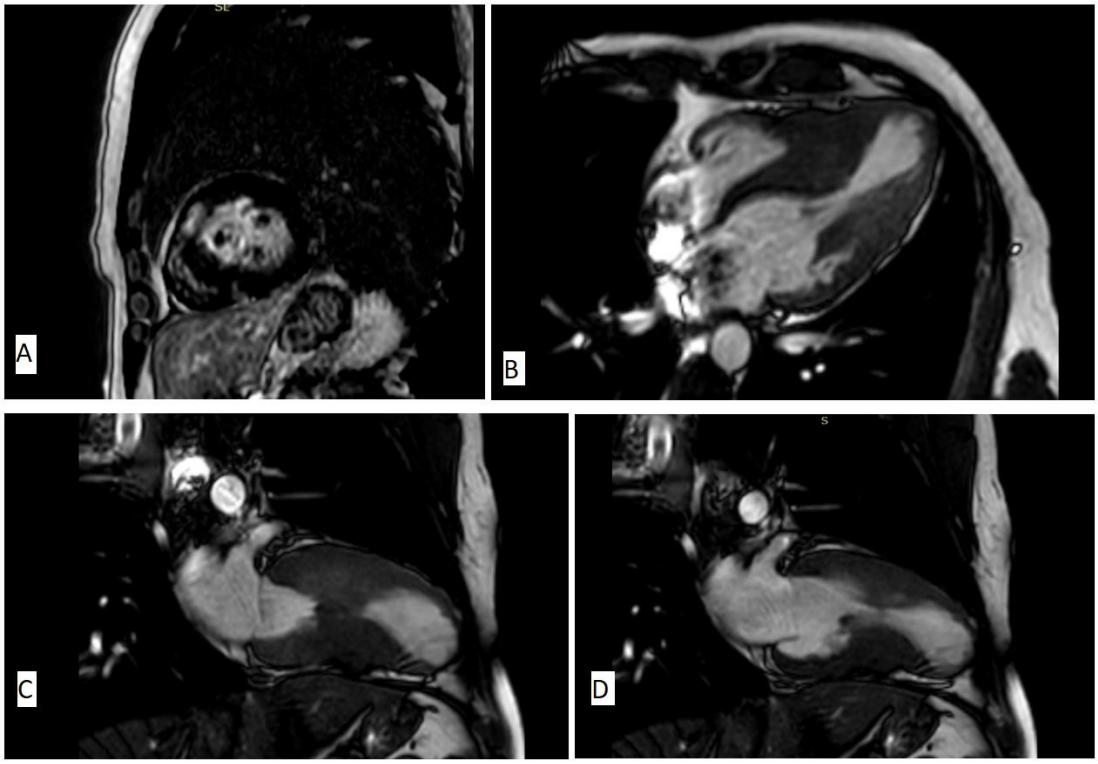

Figure (1): a, b, c, d: Left ventricle: mid ventricular wall hypertrophy (ranging from 20 to 30 mm, maximum thickness was seen in mid AS segment) with obstruction, not dilated LV with impaired systolic function (EF= 31%). Hypokinesia at apical segment with thin wall. **On late gadolinium enhancement (LGE) images:** Focal patchy enhancement is noted in mid anterior segment and transmural enhancement in mid septal segments. EF= 31%, EDV= 164ml, ESV= 114ml, SV= 50ml.

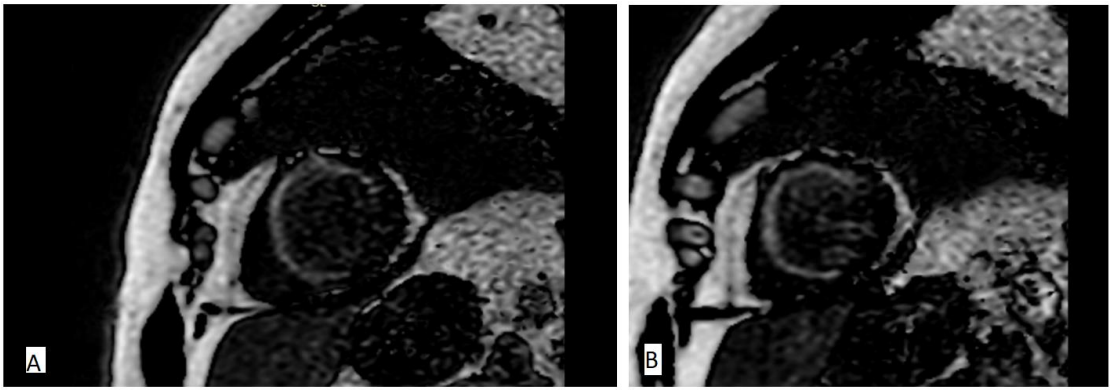

Figure (2): a, b: Left ventricle: not hypertrophied with impaired systolic function (EF=43%) hypokinesia at mid anterior and septal segments and apical segments. **On late gadolinium enhancement (LGE) images:** Transmural enhancement (>50%) at mid antero-septal and (>75%) at apical septal and inferior segments (non-viable). Subendocardial enhancement at mid anterior segment and lateral segments (viable). EF= 43%, EDV= 198ml, ESV= 114ml, SV= 84ml.

DISCUSSION

The most prevalent hereditary cardiomyopathy is HCM, which affects 1 in 500 people in general. The symptoms of HCM include myocardial fibrosis, coronary microvascular dysfunction, myocardial fibre disarray, and unexplained, often asymmetric LV enlargement [(4)]. The cornerstone for clinical diagnosis and treatment recommendations in HCM continues to be cardiac imaging. Due to its affordability and general availability, conventional 2-D TTE has been the go-to imaging modality for assessing HCM patients for many years. But CMR has become more prevalent in clinical practice in recent years [(11)].

This study showed that there were 46 patients who had comorbidities, 16 patients (32%) had hypertension, followed by 14 patients had DM (28%), and 12 patients (24%) had DM + HTN, and 4 patients (8%) were obese. In a study of 118 heart failure (HF) clinical trials by **Khan *et al.*** [(12)]**,** they demonstrated that the most prevalent comorbidities were diabetes, atrial fibrillation, hypertension, ischemic heart disease, hyperlipidemia, and chronic renal disease. Chronic renal disease, atrial fibrillation, and hypertension were the conditions with the most increases over time. Additionally, **Sharma *et al.*** [(13)] discovered that individuals with a higher number of non-cardiovascular comorbidities also had a higher number of cardiovascular comorbidities, such as peripheral vascular disease, coronary artery disease, hypertension, and a history of HF.

According to symptoms distribution, the present study showed that there were 7 patients who had acute chest pain (14%), 13 patients (26%) had dyspnea, and 13 patients (26%) had lower limb edema. According to a recent study by **Ljungman *et al.*** [(14)], the kind of cardiomyopathy had some influence on the first symptoms. The cohort's most prevalent symptom at beginning was dyspnea (63.3%), which was followed by exhaustion (51.9%) and chest discomfort (30.5%). In 81.8% of patients with amyloidosis and 85% of individuals with DCM, dyspnea was evident at initial presentation. The most prevalent first symptom among patients with GCM (80%) and myocarditis (82.7%) was chest discomfort. Ventricular arrhythmias were the most prevalent first symptom in individuals with ARVC, occurring in 70% of cases. Ventricular arrhythmias were seen in 60% of GCM patients at debut. Additionally, **Zaiser *et al.*** [(15)] observed that the most common symptoms of HCM were fatigue (89%), dyspnea (89%), dyspnea with physical exertion (89%), and lightheadedness (89%). Additional symptoms that were often mentioned were palpitations (fluttering or fast heartbeat) (81%), chest discomfort (angina) (70%), and chest pain (angina) after physical exercise (70%).

In the present study, According to ECG findings, there were 20 patients hadn't relevant (40%), 12 patients (24%) had wide QRS, followed by 11 patients had ischemic changes (22%), and 4 patients (8%) had right bundle branch block, then 4 patients (6%) had strain pattern. Echocardiography and ECG continued to be the gold standard for identifying people at risk for HCM. In particular, there may be electrocardiographic anomalies in the absence of echocardiographic hypertrophy or the amount of LV hypertrophy may not approach the diagnostic threshold [(11)]. Although earlier research has revealed variations in tissue Doppler and strain by echocardiogram in individuals who are genotype positive but phenotypically negative, these findings have been contradictory [(16, 17)].

Echo measurements indicated that, ejection fraction was in the range from 34-60% with a mean of 45.00 ± 7.49%. While, end diastolic volume was in range of 102-230 ml with a mean of 158.50 ± 46.38 ml. In this concern and in a study by **Pagourelias *et al.*** [(18)] they demonstrated that traditional echo parameters have poor accuracy in a well-defined population with enlarged myocardium, mostly due to their low sensitivity.

In this study, cardiac MRI indicated that, mean ejection fraction was 45.60 ± 8.12%. End diastolic

volume was 161.54 ± 48.14 ml, end-systolic volume was 90.30 ± 34.51ml, and stroke volume was 71.24 ± 16.32 ml. Also, RA thrombus was found in 4 patients (8%). **Mayala** [19] discovered rather good CMR sensitivity and specificity values in her study. This suggests that CMR is a viable imaging modality that is commonly used to diagnose various cardiomyopathies. Through, left ventricle quantification, **Goebel *et al.*** [20] were able to distinguish between dilated cardiomyopathy, hypertrophic cardiomyopathy, and healthy heart function. Patients with dilated cardiomyopathy had significantly lower ejection fractions and higher left ventricle end-diastolic, end-systolic, and systolic volumes than in patients with healthy hearts. Compared to healthy people, end-diastolic septum thickness was significantly larger in HCM patients [20]. This reaffirms how crucial CMR is for identifying cardiomyopathy. Additionally, CMR can help distinguish between ischemia and non-ischemic cardiomyopathy, which may help with patient care that is as efficient as possible.

In our study, there was a significant difference between Echo and Cardiac MRI regarding ejection fraction and end diastolic volume among the studied patients. EF% was significantly higher with cardiac MRI (45.60 ± 8.12%) compared to Echo (43.00 ± 7.49%), with a mean difference of 2.60 ± 2.42%. EDV/ml was significantly higher with cardiac MRI (161.54 ± 48.14 ml) compared to Echo (158.50 ± 46.38 ml), with a mean difference of 3.04 ± 5.26 ml. Over ten years ago, case reports, case series, and small clinical trials showed the importance of MRI in the diagnosis of first unexplained anomalies on electrocardiograms associated with apical hypertrophy [21], or left ventricular hypertrophy limited to the anterior/anterolateral LV free wall HCM [22]. Echocardiography was unable to identify these anomalies before, and it was only through MRI that LV hypertrophy could be seen and HCM could be diagnosed [23, 24].

According to a research by **Maron *et al.*** [6], echocardiography may overlook or undervalue LVH in the anterolateral wall, posterior septum, or apical area in 12% of HCM patients. As a result, only MRI can identify the HCM phenotype in a small but significant subset of patients, helping to establish a novel HCM diagnosis [22]. Also, in a sample of ten individuals **Moon *et al.*** [25] showed that MRI allowed the identification of apical HCM in all patients with suspected HCM (based on ECG repolarization abnormalities) but normal echocardiograms. In addition, in a systematic comparison **Valente *et al.*** [26] found that when comparing the clinical diagnostic agreement between CMR and echo in G+/LVH− HCM, it was 90%. In a limited percentage of bearers of the mutation, CMR identified focused and moderately elevated LV wall thickness that was not fully comprehended by echo. This percentage of inconsistent echo and CMR categorization of LVH is similar to findings from individuals with a clinical diagnosis of HCM (no genotyping available) [22], and from earlier research that included a limited number of G+/LVH− people as part of a larger cohort [27].

CONCLUSION

Echocardiography is still the first-line imaging modality for individuals suspected of having cardiomyopathy. While, CMR has established as the gold standard for noninvasive cardiac structural assessment and tissue characterisation. Each imaging modality has advantages and disadvantages. However, CMR technique was more accurate for diagnosis among patients suspicioned of cardiomyopathy compared to echocardiography.

Financial support and sponsorship: Nil
Conflict of Interest: Nil.

REFERENCES

1. **Arbustini E, Behr E, Carrier L *et al.* (2022):** Interpretation and actionability of genetic variants in cardiomyopathies: a position statement from the European Society of Cardiology Council on cardiovascular genomics. European Heart Journal, 43 (20): 1901-1916.
2. **Gartzonikas I, Naka K, Anastasakis A (2023):** Current and emerging perspectives on pathophysiology, diagnosis, and management of hypertrophic cardiomyopathy. Hellenic Journal of Cardiology, 70: 65-74.
3. **Baxi A, Restrepo C, Vargas D *et al.* (2016):** Hypertrophic cardiomyopathy from A to Z: genetics, pathophysiology, imaging, and management. Radiographics, 36 (2): 335-54.
4. **Moravsky G, Ofek E, Rakowski H *et al.* (2013):** Myocardial fibrosis in hypertrophic cardiomyopathy: accurate reflection of histopathological findings by CMR. JACC Cardiovascular Imaging, 6 (5): 587-96.
5. **Maron B, Desai M, Nishimura R *et al.* (2022):** Diagnosis and evaluation of hypertrophic cardiomyopathy: JACC state-of-the-art review. Journal of the American College of Cardiology, 79 (4): 372-89.
6. **Maron M, Maron B, Harrigan C *et al.* (2009):** Hypertrophic cardiomyopathy phenotype revisited after 50 years with cardiovascular magnetic resonance. Journal of the American College of Cardiology, 54 (3): 220-8.
7. **Moura B, Aimo A, Al-Mohammad A *et al.* (2023):** Diagnosis and management of patients with left ventricular hypertrophy: Role of multimodality cardiac imaging. A scientific statement of the Heart Failure Association of the European Society of Cardiology. European Journal of Heart Failure, 25 (9): 1493-506.
8. **Maron B, Maron M (2016):** A discussion of contemporary nomenclature, diagnosis, imaging, and management of patients with hypertrophic cardiomyopathy. American Journal of Cardiology, 118 (12): 1897-907.
9. **Casas G, Rodríguez-Palomares J (2022):** Multimodality cardiac imaging in cardiomyopathies:

from diagnosis to prognosis. Journal of Clinical Medicine, 11 (3): 578-83.

10. **Rammos A, Meladinis V, Vovas G *et al.* (2017):** Restrictive cardiomyopathies: the importance of noninvasive cardiac imaging modalities in diagnosis and treatment—a systematic review. Radiology Research and Practice, 17: 2874902. doi: 10.1155/2017/2874902.
11. **Hindieh W, Chan R, Rakowski H (2017):** Complementary role of echocardiography and cardiac magnetic resonance in hypertrophic cardiomyopathy. Current Cardiology Reports, 19: 1-3.
12. **Khan M, Samman Tahhan A, Vaduganathan M *et al.* (2020):** Trends in prevalence of comorbidities in heart failure clinical trials. European Journal of Heart Failure, 22 (6): 1032-42.
13. **Sharma A, Zhao X, Hammill B *et al.* (2018):** Trends in noncardiovascular comorbidities among patients hospitalized for heart failure: insights from the Get with the Guidelines–Heart Failure Registry. Circulation: Heart Failure, 11 (6): e004646. doi: 10.1161/ CIRCHEARTFAILURE.117.004646.
14. **Ljungman C, Bollano E, Rawshani A *et al.* (2023):** Differences in phenotypes, symptoms, and survival in patients with cardiomyopathy—a prospective observational study from the Sahlgrenska CardioMyoPathy Centre. Frontiers in Cardiovascular Medicine, 10: 1160089. doi: 10.3389/fcvm.2023.1160089.
15. **Zaiser E, Sehnert A, Duenas A *et al.* (2020):** Patient experiences with hypertrophic cardiomyopathy: a conceptual model of symptoms and impacts on quality of life. Journal of Patient-Reported Outcomes, 4 (1): 102. doi: 10.1186/s41687-020-00269-8.
16. **Ho C, Sweitzer N, McDonough B *et al.* (2002):** Assessment of diastolic function with Doppler tissue imaging to predict genotype in preclinical hypertrophic cardiomyopathy. Circulation, 105 (25): 2992-97.
17. **Aly M, Brouwer W, Kleijn S *et al.* (2014):** Three-dimensional speckle tracking echocardiography for the preclinical diagnosis of hypertrophic cardiomyopathy. The International Journal of Cardiovascular Imaging, 30: 523-33.
18. **Pagourelias E, Mirea O, Duchenne J *et al.* (2017):** Echo parameters for differential diagnosis in cardiac amyloidosis: a head-to-head comparison of deformation and nondeformation parameters. Circulation: Cardiovascular Imaging, 10 (3): e005588. doi: 10.1161/CIRCIMAGING.116.005588.
19. **Mayala H (2018):** The role of cardiac magnetic resonance (CMR) in the diagnosis of cardiomyopathy: A systematic review. Malawi Medical Journal, 30 (4): 291-5.
20. **Goebel J, Seifert I, Nensa F *et al.* (2016):** Can native T1 mapping differentiate between healthy and diffuse diseased myocardium in clinical routine cardiac MR imaging?. PLoS One, 11 (5): e0155591. doi: 10.1371/journal.pone.0155591
21. **Ibrahim T, Schwaiger M (2000):** Diagnosis of apical hypertrophic cardiomyopathy using magnetic resonance imaging. Heart, 83 (1): 1. doi: 10.1136/heart.83.1.e1.
22. **Rickers C, Wilke N, Jerosch-Herold M *et al.* (2005):** Utility of cardiac magnetic resonance imaging in the diagnosis of hypertrophic cardiomyopathy. Circulation, 112 (6): 855-61.
23. **Ismail T, Jabbour A, Gulati A *et al.* (2014):** Role of late gadolinium enhancement cardiovascular magnetic resonance in the risk stratification of hypertrophic cardiomyopathy. Heart, 100 (23): 1851-58.
24. **Chan R, Maron B, Olivotto I *et al.* (2015):** Significance of late gadolinium enhancement at right ventricular attachment to ventricular septum in patients with hypertrophic cardiomyopathy. The American Journal of Cardiology, 116 (3): 436-41.
25. **Moon J, Fisher N, McKenna W *et al.* (2004):** Detection of apical hypertrophic cardiomyopathy by cardiovascular magnetic resonance in patients with non-diagnostic echocardiography. Heart, 90 (6): 645-49.
26. **Valente A, Lakdawala N, Powell A *et al.* (2013):** Comparison of echocardiographic and cardiac magnetic resonance imaging in hypertrophic cardiomyopathy sarcomere mutation carriers without left ventricular hypertrophy. Circulation: Cardiovascular Genetics, 6 (3): 230-37.
27. **Germans T, Wilde A, Dijkmans P *et al.* (2006):** Structural abnormalities of the inferoseptal left ventricular wall detected by cardiac magnetic resonance imaging in carriers of hypertrophic cardiomyopathy mutations. Journal of the American College of Cardiology, 48 (12): 2518-23.