The Egyptian Journal of Hospital Medicine (October 2024) Vol. 97, Page 4559-4567

Patterns of Right Ventricle Affection in Adult Patients with Hypertrophic Cardiomyopathy: Cardiac Magnetic Resonance Study

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ABSTRACT

Background: Hypertrophic cardiomyopathy (HCM) primarily affects the left ventricle (LV), but right ventricular (RV) involvement is increasingly recognized and may have prognostic implications. Advanced imaging modalities like cardiac magnetic resonance (CMR) offer valuable insights into RV changes in HCM but remain underutilized.

Objective: This study aimed to evaluate RV structural and functional changes in HCM using CMR.

Patients and Methods: A retrospective analysis of 64 adult HCM cases who underwent CMR between January 2020 and March 2024. Patients were categorized into HCM with RV hypertrophy (RVH) and without RVH. Key parameters such as RV and LV wall thickness, volumes, ejection fractions, and fibrosis by late gadolinium enhancement (LGE) were analyzed. Correlation and agreement between RV and LV parameters were assessed using Pearson correlation and Intraclass Correlation Coefficient (ICC). **Results:** RVH was present in 15.6% of cases and associated with significantly greater RV wall thickness and fibrosis (90% vs. 9.3%, p = 0.001). RVH cases exhibited higher LV end-diastolic volume (164.45 ± 52.01 vs. 138.75 ± 34.3, p = 0.05) and universal LV fibrosis (100% vs. 59.3%, p = 0.012). Significant correlations between RV and LV end-diastolic volumes (r = 0.559, p = 0.001) and stroke volumes (r = 0.620, p = 0.001) were noted. Agreement analysis showed good concordance for EF (ICC = 0.736) and stroke volume (ICC = 0.758).

Conclusion: RVH in HCM is linked to marked structural and functional changes. CMR provided valuable insights into biventricular dynamics, and future research should integrate clinical and genetic data to refine management strategies.

Keywords: Hypertrophic cardiomyopathy, Right ventricle, Cardiac magnetic resonance.

INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is the most prevalent hereditary heart condition globally, affecting approximately 0.16% to 0.29% of the population. While, European Society of Cardiology (ESC) defines HCM primarily by the thickness of the left ventricular (LV) wall—measuring 15 mm or more in one or more myocardial segments without other causes such as increased loading conditions—it's worth noting that right ventricle (RV) can also be significantly involved. This RV involvement, although less commonly discussed, is not rare and can play an important role in shaping disease's prognosis [1]. In HCM, RV involvement encompasses both structural adaptations consistent with a hypertrophic phenotype and notable functional abnormalities. Unlike well-established diagnostic criteria for left ventricular hypertrophy (LVH), there is no universally accepted definition for right ventricular hypertrophy (RVH). One of the most influential studies on echocardiographic evaluation of RV identified a normal RV wall thickness as being ≤ 7 mm [2]. Right ventricular hypertrophy (RVH) was classified by **McKenna *et al.*** [3] into three severity levels: mild (6–8 mm), moderate (9–12 mm), and severe (greater than 12 mm). Whereas RVH was defined as the anterior, free, or apical wall of RV's end-diastolic thickness of ≥ 5 mm by **Maron *et al.*** [4].Moreover, RVH is categorized as extreme or severe when wall thickness reaches ≥ 10 mm. The prevalence of RV involvement in HCM cases shows considerable variation based on diagnostic methods and criteria used. Early understanding of RV involvement was primarily obtained from postmortem studies and catheterization techniques. More recently, CMR imaging has detected RVH in about 30% of HCM cases [4]. In contrast, echocardiography identifies RVH in 44% of cases with HCM, highlighting differences in detection rates between diagnostic modalities [3]. Severe RVH is relatively uncommon, occurring in only 1.3% of overall HCM population [5]. The pattern of RVH in HCM is highly variable, ranging from concentric thickening to diffuse hypertrophy involving RV apex, mid septum, basal septum, and/or free wall. RVH or increased RV mass can develop either in isolation, though this is rare, or more commonly in conjunction with LVH [6]. Some studies have demonstrated that RVH is independently associated with LVH. **Maron *et al.*** [4] highlighted a significant correlation between maximum RV and LV wall thickness, as well as between RV and LV mass [4]. While, global studies have provided valuable insights into RV involvement in HCM, regional differences in genetic, environmental, and healthcare factors may influence its presentation and prevalence. In our country, data on RV hypertrophy and its patterns in HCM cases are scarce, limiting our ability to fully appreciate its clinical relevance. Therefore, this study aimed to assess CMR value in the assessment of associated right ventricular changes in HCM adult cases.

PATIENTS AND METHODS

This retrospective descriptive study was conducted using a 1.5 T MRI scanner in The Radiology

Received: 24/07/2024
Accepted: 24/09/2024

Department of Mansoura University Hospitals. The study focused on adult cases diagnosed with HCM. The study included 64 cases with HCM who underwent cardiac MRI between January 2020 and March 2024 at Diagnostic and Interventional Radiology Department of Mansoura University Hospitals.

Inclusion criteria: Adult cases with HCM.

Exclusion criteria: Individuals with other causes of left ventricular hypertrophy (e.g., infiltrative disease, Fabry disease, valvular heart disease, myocarditis and coronary artery disease). Additionally, children under 18 years of age, individuals with standard contraindications to CMR (such as metal implants, severe claustrophobia, or inability to hold their breath), and cases unwilling to undergo MR examination.

CMR technique: The study used a 1.5 T MRI scanner (Siemens) with a comprehensive imaging protocol. Cine images of 2-, 3-, and 4-chamber views were captured using a breath-hold, ECG-triggered, balanced steady-state free-precession sequence. The standard scan settings included 25 phases, an echo time of 1.2 ms, a repetition time of 33–54 ms, a flip angle ranging from 64° to 79°, a slice thickness of 8 mm, and a 2 mm gap between slices. Additionally, short-axis cine images were taken to cover both ventricles from base to apex. These images were analyzed to assess RV and LV size and function by tracing endocardial and epicardial boundaries at end-diastole and end-systole using specialized software. A standard intravenous dose of 0.1 mmol/kg of a gadolinium-based contrast agent was administered to all cases.

Late gadolinium enhancement (LGE) images were captured 10 to 15 minutes following contrast agent administration. LGE MRI was performed using a phase- and magnitude-sensitive inversion recovery-prepared steady-state free-precession sequence, with inversion time carefully adjusted to suppress signals from healthy myocardium. These images were acquired in both short-axis and long-axis views, aligned with cine image planes to maintain consistency in localization

CMR post-processing: CMR images were analyzed using Philips software to calculate right and left ventricular end-diastolic volumes (RVEDV and LVEDV), end-systolic volumes (RVESV and LVESV), ejection fractions (RVEF and LVEF), and ventricular masses (RVM and LVM). All volume and mass measurements were indexed to body surface area (BSA) and reported in mL/m² or g/m², respectively. Each image was meticulously reviewed for signs of RV and LV hypertrophy, as well as presence of RV and LV LGE. LGE analysis was performed by an experienced cardiac MRI specialist.

Ethical consideration: The study protocol was submitted for review and approval by The Institutional Research Board (IRB) of Mansoura University (Approval Code: R.24.05. 2615). Participant privacy was strictly maintained throughout the study, and all collected data were utilized solely for purposes of this research. This work has been carried out in accordance with The Code of Ethics of World Medical Association (Declaration of Helsinki) for studies involving humans.

Statistical analysis

Data analysis was conducted using SPSS software, version 26 (SPSS Inc., PASW Statistics for Windows, Version 26, Chicago, SPSS Inc.). Qualitative variables were expressed as frequencies and percentages, while quantitative variables with a normal distribution were reported as mean ± standard deviation. Normality was assessed using Kolmogorov-Smirnov test, and a p-value of 0.05 was considered statistically significant. Chi-Square test was applied to compare qualitative variables between groups when appropriate. Student's t-test was used to compare normally distributed quantitative variables between two independent groups. Pearson correlation assessed linear relationship between two normally distributed continuous variables, while Spearman correlation was used for non-normally distributed continuous or ordinal variables to determine strength and direction of their relationship. Interclass correlation coefficients (ICC) and Kappa (K) values were calculated to evaluate agreement between right and LV parameters, along with their 95% confidence intervals (CI). The agreement levels were interpreted as follows: excellent (ICC or K > 0.75), good (ICC or K between 0.60 and 0.74), moderate (ICC or K between 0.40 and 0.59), and poor (ICC or K < 0.40).

RESULTS

Demographic Data: The study included a total of 64 cases, comprising 18 females (28.1%) and 46 males (71.9%). The mean age of participants was 49.41±13.2 years, with a median age of 52 years (range: 18–79). The demographic comparison between HCM groups with and without RVH revealed that among those with RVH (n=10), 3 were females (30%) and 7 were males (70%), whereas in the group without RVH (n=54), 15 were females (27.8%) and 39 were males (72.2%), with no significant difference in gender distribution (P=0.886). The mean age was 46.6 ± 9.9 years in the RVH group and 49.9 ± 13.8 years in non-RVH group, also showing no statistically significant difference (P=0.468).

LV EDV (End-Diastolic Volume): HCM cases with RVH showed higher mean LV EDV (164.45 ± 52.01) compared to those without RVH (138.75 ± 34.3, p = 0.05), indicating significant dilation in RVH cases. **LV LGE**: LGE was present in all HCM with RVH cases (100%) compared to 59.3% in non-RVH cases (p = 0.012), suggesting more extensive fibrosis in RVH cases. **Myocardial Edema**: Significantly more common in RVH cases (50%) compared to those without RVH (16.7%, p = 0.019) (Table 1).

Table (1): CMR parameters of left heart: LV, LA and left sided valves

	HCM with RVH (10)	HCM without RVH (54)	**P value**
LV parameters			
LV EDV (ml) **Mean± SD.** **Median (minimum-maximum)**	 164.45±52.01 171(103-257)	 138.75±34.3 139(75-234)	0.05*
LV EF (%) **Median (minimum-maximum)** **Mean± SD**	 69(40-86) 67.8±14.2	 74(41-91) 71.8±10.6	0.303
LV ED wall mass (gm) **Median (minimum-maximum)** **Mean± SD**	(number=10) 226(141-306) 222.6±55.84	(number=51) 187(60-395) 192.9±74.35	0.238
Maximum LV wall thickness (mm) **Median (minimum-maximum)** **Mean± SD**	 24.5(19-29) 24.16±3.07	 20(12.7-41) 21.8±6.91	0.291
SAM N (%) **Yes** **No**	 2/10 (20%) 8/10 (80%)	 24/54 (44.4%) 30/54 (55.6%)	0.148
Type of LV hypertrophy N (%) **Concentric** **Apical** **Mid-wall** **Asymmetric**	 2/10 (20%) 0/10 (0.0%) 2/10 (20%) 6/10 (60%)	 10/54 (18.5%) 3/54 (5.6%) 3/54 (5.6%) 38/54 (70.3%)	0.399
LV LGE enhancement N (%) **Yes** **No**	 10/10 (100%) 0/10 (0.0%)	 32/54 (59.3%) 22/54 (40.7%)	0.012*
Myocardial edema N (%) **Yes** **No**	 5/10 (50%) 5/10 (50%)	 9/54 (16.7%) 45/54 (83.3%)	0.019*
LA parameters			
LA N (%) **Dilated** **Not dilated**	 6/10 (60%) 4/10 (40%)	 34/54 (62.9%) 20/54 (37.1)	0.859
Valve regurgitations N (%)			
MR **No** **Mild** **Moderate** **Severe**	 1/10 (10%) 4/10 (40%) 5/10 (50%) 0/10 (0.0%)	 12/54 (22.2%) 26/54 (48.1%) 11/54 (20.4%) 5/54 (9.3%)	0.201
AR **No** **Mild**	 9/10 (90%) 1/10 (10%)	 33/54 (61.1%) 21/54 (38.9%)	0.077

used test: Chi-Square test and Student t test, *statistically significant, AR - Aortic Regurgitation, HCM - Hypertrophic Cardiomyopathy, LA - Left Atrium, LGE - Late Gadolinium Enhancement, LV - Left Ventricle, LV EDV - Left Ventricular End-Diastolic Volume, LV EF - Left Ventricular Ejection Fraction, LV ED wall mass - Left Ventricular End-Diastolic Wall Mass, N- number, MR - Mitral Regurgitation, RVH - Right Ventricular Hypertrophy, and SAM - Systolic Anterior Motion.

The table highlighted key differences in CMR findings between HCM cases with and without RVH. While RV end-diastolic volume (RV EDV) and RV EF showed no significant differences, RV wall thickness and hypertrophy patterns were markedly distinct, with all RVH cases exhibiting thickened walls and hypertrophy (P=0.001). RV LGE was significantly more common in RVH cases (90% vs. 9.3%, P=0.001). In contrast, no significant differences were observed in RA size, tricuspid regurgitation, and pulmonary regurgitation. These results underscore pronounced structural and functional RV abnormalities in cases with RVH (Table 2).

Table (2): CMR parameters of right heart: RV, RA and right sided valves

	HCM with RVH (10)	HCM without RVH (54)	**P value**
RV parameters			
RV EDV (ml) **Median (minimum-maximum)** **Mean± SD**	 122.15(81-188) 129.23±37.21	 125(63-218) 131.5±35.6	0.857
RV EF (%) **Mean± SD** **Median (minimum-maximum)**	 65.9±9.83 67.5(44-80)	 64.5±10.44 65.5(40-85)	0.704
RV wall thickness (N (%)**)** **Average** **Thickened**	 0/10 (0.0%) 10/10 (100%)	 54/54 (100%) 0/54	0.001*
RV affection pattern N (%) **Normal** **Hypertrophy** **Compressed** **Dilated**	 0/10 (0.0%) 10/10 (100%) 0/10 (0.0%) 0/10 (0.0%)	 51/54 (94.4%) 0/54 (0.0%) 2/54 (3.7%) 1/54 (1.9)	0.001*
RVOT N (%) **Obstructed** **Non-obstructed**	 2/10 (20%) 8/10 (80%)	 2/54 (3.7%) 52/54 (96.3%)	0.112
RV LGE enhancement N (%) **Yes** **No**	 9/10 (90%) 1/10 (10%)	 5/54 (9.3%) 49/54 (90.7%)	0.001*
RA parameters N (%)			
RA **Average** **Dilated** **Compressed**	 1/10 (10%) 9/10 (90%) 0/10 (0.0%)	 3/54 (5.6%) 50/54 (92.5%) 1/54 (1.9%)	0.795
Valve regurgitation N (%)**:**			
TR **No** **Mild** **Moderate** **Severe**	 3/10 (30%) 6/10 (60%) 1/10 (10%) 0/10 (0.0%)	 27/54 (50%) 20/54 (37%) 6/54 (11.1%) 1/54 (.9%)	0.570
PR **No** **Mild**	 10/10 (100%) 0 (0.0%)	 53/54 (98.1%) 1/54 (1.9%)	1.0

used test: Chi-Square test and Student t test, *statistically significant, HCM - Hypertrophic Cardiomyopathy, N- number, PR - Pulmonary Regurgitation, RA - Right Atrium, RV - Right Ventricle, RV EDV - Right Ventricular End-Diastolic Volume, RV EF - Right Ventricular Ejection Fraction, RV LGE - Right Ventricular Late Gadolinium Enhancement, RVOT - Right Ventricular Outflow Tract, and TR - Tricuspid Regurgitation.

The table assessed the agreement between RV and LV parameters using ICC and Kappa (K) values with 95% CI. Stroke Volume (SV) and Ejection Fraction (EF) demonstrated the highest agreement, with ICCs reflecting excellent and good agreement, respectively. End-diastolic volume (EDV), end-systolic volume (ESV), and Enhancement showed moderate to good agreement, indicating partial consistency. In contrast, parameters such as Maximum Wall Thickness and VOT exhibit poor or no agreement, highlighting significant variability between ventricles.

Figure (1) provides a Bland-Altman analysis of EF agreement between right and LVs (Table 3).

Table (3): Agreement between right and left ventricular parameters

	ICC, K (95%CI)
EF	0.736 (0.566-0.840)
EDV	0.678 (0.471-0.805)
EDVI	0.556 (0.231-0.744)
ESV	0.469 (0.126-0.677)
ESVI	0.442 (0.034-0.678)
SV	0.758 (0.60-0.854)
Maximal wall thickness	-0.167 (-7.34, 0.837)
VOT Obstruction	0.023 (-0.701, 0.372)
LG Enhancement	0.462 (0.115-0.673)

EF - Ejection Fraction, EDV - End-Diastolic Volume, EDVI - End-Diastolic Volume Index, Function - Ventricular Function, ESV - End-Systolic Volume, ESVI - End-Systolic Volume Index, SV - Stroke Volume, VOT -Ventricular Outflow Tract, Enhancement - Late Gadolinium Enhancement, ICC - Intraclass Correlation, K - Kappa Agreement. ICC: interclass correlation, k: kappa agreement.

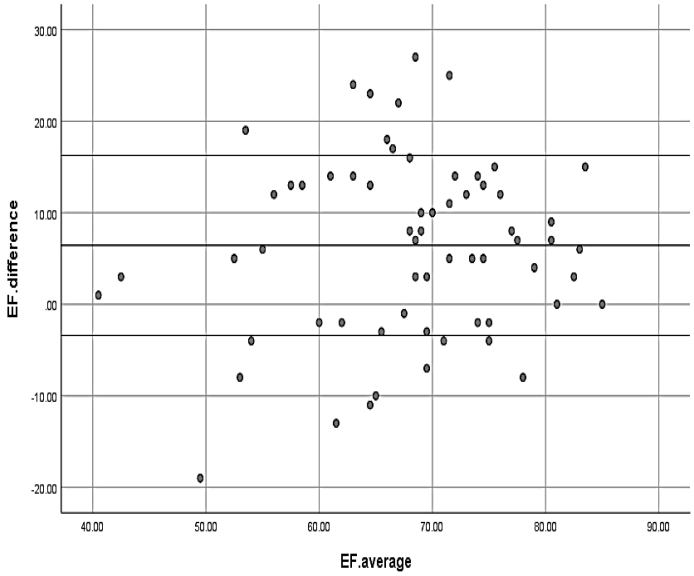

Figure (1): Bland Altman analysis showing agreement between right and left ventricular measurements of EF.

Table (4): Correlation between RV and LV parameters in HCM

			LV parameters							
			LV EF %	**Thickness mm**	**EDV ml**	**EDVI ml/m2**	**ESV ml**	**ESVI ml/m2**	**SV ml**	**LVOTO**
Right ventricular parameters	**RV EF %**	r	0.147	0.241	0.141	0.061	**-0.304**	**-0.313**	**0.401**	.013
		p value	0.248	0.055	0.267	0.663	**0.015***	**0.023***	**0.001***	.920
	Thickness mm	r	-0.812	-0.232	0.493	0.700	0.667	0.900	-0.493	-0.664
		p value	0.050	0.658	0.321	0.188	0.148	0.037	0.321	0.150
	EDV ml	r	0.110	0.027	**0.559**	**0.551**	0.199	0.144	**0.609**	0.127
		p value	0.388	0.833	**0.001***	**0.001***	0.115	0.303	**0.001***	0.316
	EDVI ml/m2	r	0.023	-0.074	**0.452**	**0.505**	0.213	**0.272**	**0.470**	-0.006
		p value	0.871	0.600	**0.001***	**0.001***	0.126	**0.049***	**0.001***	0.964
	ESV ml	r	**-0.356**	**-0.246**	**0.246**	0.237	**0.398**	**0.372**	0.045	0.057
		p value	**0.004***	**0.050***	**0.050***	0.088	**0.001***	**0.006***	0.727	0.653
	ESVI ml/m2	r	**-0.409**	-0.211	0.237	**0.280**	**0.398**	**0.461**	-0.023	0.063
		p value	**0.002***	0.129	0.087	**0.043***	**0.003***	**0.001***	0.871	0.654
	SV ml	r	**0.263**	0.115	**0.464**	**0.401**	0.040	-0.041	**0.620**	0.080
		p value	**0.036***	0.365	**0.001***	**0.003***	0.751	0.771	**0.001***	0.532
	RVOTO	r	0.024	0.037	-0.154	-0.119	-0.136	-0.108	-0.098	-0.029
		p value	0.848	0.773	0.225	0.396	0.283	0.443	0.443	0.819

r: correlation coefficient, *statistically significant, LV - Left Ventricle, RV - Right Ventricle, EDV - End-Diastolic Volume, EDVI - End-Diastolic Volume Index, ESV - End-Systolic Volume, ESVI - End-Systolic Volume Index, SV - Stroke Volume, LVOTO - Left Ventricular Outflow Tract Obstruction, RVOTO - Right Ventricular Outflow Tract Obstruction, r - Correlation Coefficient, and * - Statistically Significant.

The table highlights correlations between RV and LV parameters in HCM. Significant positive correlations were found for volume-related parameters: RV EDV strongly correlated with LV EDV (r=0.559, P=0.001) and LV EDVI (r=0.452, P=0.001), while RV stroke volume (SV) correlated significantly with LV EDV (r=0.464, P=0.001) and LV SV (r=0.620, P=0.001). RV EDVI also correlated with LV EDV (r=0.505, P=0.001) and LV EDVI (r=0.470, P=0.001). RV end-systolic volume (ESV) showed weaker but significant correlations with LV EDV (r=0.246, P=0.050) and LV SV (r=0.401, P=0.003). However, RV ejection fraction (EF) and RV outflow tract (RVOT) demonstrated no significant correlations with LV parameters. These findings suggest interdependence between RV and LV volume-related parameters in HCM, whereas functional measures like RV EF and RVOT showed limited association with LV characteristics (Table 4).

Figure (2) represented CMR images in a case of biventricular HCM.

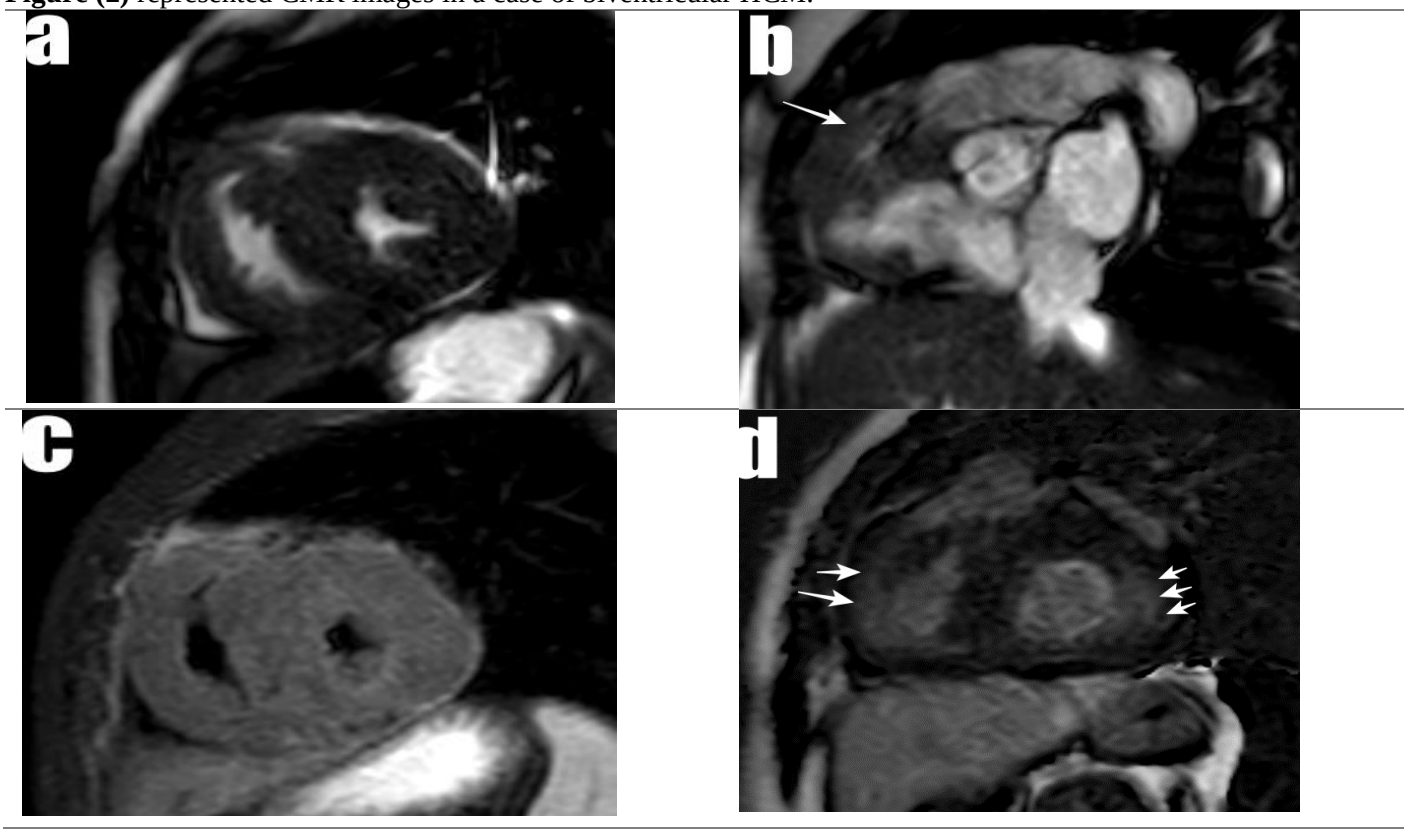

Figure (2): CMR images showing biventricular HOCM: Image (a) Short axis (SA) steady state free precession (SSFP) image with hypertrophied LV wall and RV wall reaching up to 20, 10 mm respectively. Image (b) RVOT SSFP image with hypertrophied RV wall with marked RVOT obstruction and stenotic jet (arrow). Image (c) SA T2 Black blood image showing absence of edema. Image (d) SA LGE showing patchy areas of mid lateral LV and RV free wall and inferior wall enhancement (arrows).

DISCUSSION

HCM has long been recognized as a condition primarily affecting LV, with its clinical management and prognosis heavily focused on LV morphology and function rather than RV [7]. However, emerging evidence highlights the significant role of RV assessment in this complex disease. While, right ventricle has historically received less attention due to technical challenges in imaging and perception of its lesser involvement, recent advancements in CMR have revealed critical insights into RV morphology, function, and their association with adverse outcomes.

We aimed to assess CMR value in assessment of associated right ventricular changes in adult cases with HCM. Sixty-four HCM cases were included retrospectively in this study. Patients were divided into 2 groups HCM with right ventricular hypertrophy (RVH) and without RVH. All CMR LV and RV parameters were compared in both groups.

The main significant finding of this study was the noticeable structural and functional abnormalities observed in HCM cases with right ventricular hypertrophy (RVH), as evidenced by marked fibrosis, hypertrophy, and interdependence between LV and RV parameters.

Our study highlighted significant structural and functional differences in left and right ventricular parameters between cases with and without RVH. Patients with RVH demonstrated a notably higher LV EDV (164.45 ± 52.01) compared to non-RVH cases (138.75 ± 34.3, $p = 0.05$), indicating LV dilation. Additionally, LV fibrosis, assessed by LGE, was observed in 100% of RVH cases, compared to only 59.3% in non-RVH cases ($p = 0.012$), suggesting more extensive fibrosis in RVH cases. Myocardial edema was also significantly more prevalent among RVH cases (50%) than in non-RVH cases (16.7%, $p = 0.019$), indicating increased myocardial stress, inflammation, or remodeling. However, no significant differences were observed between groups regarding LV EF, LV mass, maximal end-diastolic wall thickness, or left atrial dilation ($p > 0.05$). When compared to other studies, our findings align with those of **Zhang *et al.*** [8], who also reported a statistically significant difference in LV fibrosis assessed by LGE, observed in 95.6% of RVH cases. Similar to our study, **Zhang *et al.*** [8] found no significant differences in LV ejection fraction or left atrial diameter between RVH and non-RVH cases. Their findings further emphasize the association of LV fibrosis with more severe disease progression in RVH cases and

an increased risk of sudden cardiac death ($p < 0.001$), corroborating clinical relevance of our observations. In contrast to our findings, **Seo *et al.*** [9] reported a significant reduction in LV ejection fraction ($p < 0.001$) in RVH cases but observed no significant differences in LV EDV or LGE. This divergence highlighted variability in manifestation of LV functional and structural parameters across studies. Furthermore, the absence of findings on LV EDV and myocardial edema in **Seo *et al.***'s [9] study underscores unique contributions of our research in identifying these parameters as significant markers in RVH cases. **Nagata *et al.*** [10] reported non-significant differences in LV EDV and LGE between RVH and non-RVH groups, differing from our findings. However, they observed a significant increase in LV mass and wall thickness, along with a reduction in LV ejection fraction in RVH cases. Despite these variations, **Nagata *et al.*** [10] concluded that RVH is associated with an increased incidence of cardiac events, reinforcing clinical importance of RVH as a marker of disease severity and adverse outcomes.

For LV parameters assessment, our study underscored that RVH in HCM is linked to greater myocardial stress, increased fibrosis, and edema, which may suggest worse disease progression. These findings align with the idea that RVH may be a significant prognostic factor for adverse outcomes in HCM cases.

In terms of RV parameters, RV wall thickness was exclusively observed in RVH cases (100%, $p = 0.001$), and RV LGE occurred significantly more often in RVH cases (90% vs. 9.3%, $p = 0.001$), highlighting greater fibrotic burden in these individuals. All RVH cases exhibited hypertrophy, while those without RVH mostly displayed normal RV patterns ($p = 0.001$). However, no significant differences were found in valvular or atrial dilation between groups. These findings align with a study by **Nagata *et al.*** [10], which demonstrated that cases with HCM and RVH show significantly greater RV wall thickness and higher rates of RV fibrosis (RV-LGE) compared to those without RVH ($p < 0.0001$). This suggests that RVH was linked to distinct structural and tissue abnormalities in RV, though overall systolic right ventricular ejection fraction (RVEF) remains relatively preserved.

Our results are also consistent with those of **Seo *et al.*** [9] who reported that RV LGE occurs significantly more frequently in RV involvement group (56.8% vs. 2.3%, $p < 0.001$), emphasizing association between RV involvement and more pronounced ventricular remodeling and fibrosis in HCM. Their study also found that RV involvement in HCM cases is associated with more advanced LV structural changes and biventricular dysfunction, serving as a potential marker of severe disease.

Moreover, **Zhang *et al.*** [8] showed that HCM cases with RV involvement face a higher risk of cardiovascular death, all-cause mortality, and heart failure-related death. These findings underscore clinical importance of abnormal RV conditions and their impact on patient outcomes. This has been further corroborated by multiple studies that highlighted distinct role of RV involvement in HCM prognosis, confirming that RVH and dysfunction are linked to poorer outcomes in these cases [11-15]. Several recent studies using advanced CMR techniques have underscored the importance of assessing RV function in HCM cases. CMR feature tracking has identified impaired RV deformation, such as reduced global longitudinal and radial strain, particularly in cases with LV outflow tract obstruction (LVOTO) [16, 17]. RV dysfunction has been found to precede reductions in LV ejection fraction and is associated with adverse clinical outcomes, even in those with preserved LV function [18]. Additionally, RV function correlates with symptom severity, as determined by NYHA classification, and predicts severe symptomatic cases with high sensitivity and specificity [19]. **Seo *et al.*** [9] also found that RV involvement and impaired RV strain have prognostic value, being linked to an increased risk of adverse clinical events in HCM cases. These findings emphasize clinical relevance of RV evaluation using CMR in comprehensive management of HCM. Unfortunately, in our study, we only assessed morphological and functional parameters using CMR without correlating these findings with clinical data or clinical outcomes related to this complex genetic disease.

In this study, significant positive correlations were found between LV and RV parameters, such as end-diastolic volume ($r = 0.559$, $p = 0.001$) and stroke volume ($r = 0.620$, $p = 0.001$), highlighting interdependence of both ventricles in HCM. These findings emphasized crucial role of biventricular involvement in RVH pathophysiology in HCM. Our results align with the findings of **Śpiewak *et al.*** [20] who demonstrated that LV end-diastolic volume index (LVEDVI) was positively correlated with the RV end-diastolic volume index (RVEDVI), further supporting the concept of ventricular interdependence. Additionally, the study by **Nagata *et al.*** [10] found significant correlations between RV maximal wall thickness and left ventricular mass index (LVMI) ($R = 0.22$, $P < 0.0001$), as well as with RV end-diastolic volume index ($R = 0.05$, $P = 0.02$), further reinforcing relationship between the two ventricles in HCM.

We tested the agreement between right and LV parameters in HCM, through ICC and Kappa (K) values, which demonstrated varying degrees of concordance reflecting complex inter-ventricular relationships in this condition. There was good agreement for ejection fraction (EF, ICC = 0.736) and stroke volume (SV, ICC = 0.758) indicating synchronized functional performance of

ventricles in maintaining cardiac output. Whereas, end-diastolic volume (EDV, ICC = 0.678) showed moderate agreement suggesting some alignment in filling capacities. End-diastolic volume index (EDVI, ICC = 0.556), end-systolic volume (ESV, ICC = 0.469), and end-systolic volume index (ESVI, ICC = 0.442) exhibited lower levels of agreement, reflecting moderate to poor concordance and highlighted variability in ventricular systolic mechanics and contractile patterns. Parameters such as maximum wall thickness (ICC = -0.167) and ventricular outflow tract obstruction (VOT, ICC = 0.023) showed no meaningful agreement, indicating significant variability between ventricles and underscored distinct structural and pathophysiological differences between ventricles, reflecting heterogeneity of HCM.

Moderate agreement for LGE, ICC = 0.462 further indicated significant variability in fibrosis distribution between ventricles, with LV fibrosis being a hallmark feature and right ventricular fibrosis occurring less frequently. These findings emphasized importance of comprehensive assessment of both ventricles using advanced imaging modalities like CMR to detect structural, functional, and tissue-level differences. The variability in inter-ventricular concordance also underscored need to monitor parameters such as wall thickness, fibrosis, and systolic volumes as these may provide critical insights into disease progression, prognosis, and individualized management in HCM cases.

RECOMMENDATIONS

Given the significant structural and functional differences observed in HCM cases with and without RVH, it is essential to refine diagnostic and management strategies to include comprehensive biventricular evaluation. Routine assessments should prioritize RV parameters, such as wall thickness and fibrosis (LGE), as these are strongly associated with disease progression and adverse outcomes. Advanced imaging modalities, particularly CMR, should be utilized for their ability to provide detailed insights into structural, functional, and tissue-level abnormalities in both ventricles. Furthermore, future research should aim to integrate morphological and functional findings with clinical, genetic, and prognostic data, offering a more holistic understanding of RV involvement and its implications in management of HCM cases.

LIMITATIONS

While this study provided valuable insights into biventricular involvement in HCM, it had several limitations that warrant consideration. The lack of correlation between morphological and functional parameters with clinical outcomes, symptom severity, or genetic profiles limits study's prognostic relevance. Additionally, the relatively small sample size, particularly within RVH group, may restrict generalizability of the findings. The retrospective design introduces potential biases and limits the ability to draw causal relationships. Furthermore, advanced RV metrics such as strain and deformation were not evaluated, which could have provided a more comprehensive understanding of RV dysfunction in HCM cases.

CONCLUSION

This study emphasized the critical role of RVH in HCM, highlighting its association with severe disease and adverse outcomes. CMR is a valuable tool for biventricular evaluation, but future research should integrate clinical and genetic data to better understand the prognostic significance of RV abnormalities and refine personalized management strategies.

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

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