ScientificScholar®
Knowledge is power
Publisher of Scientific Journals

www.clinicalimagingscience.org

Journal of Clinical Imaging Science

Cardiopulmonary Imaging *Original Research*

Patterns of Left Ventricular Hypertrophy and Late Gadolinium Enhancement on Cardiac MRI in Patients with Hypertrophic Cardiomyopathy and their Prognostic Significance – An Experience from a South Asian Country

Fateh Ali Tipoo Sultan[1], Sheema Saadia[1]

[1]Department of Medicine, Aga Khan University Hospital, Karachi, Sindh, Pakistan.

***Corresponding author:**
Fateh Ali Tipoo Sultan,
Department of Medicine, Aga Khan University Hospital, Karachi, Sindh, Pakistan.

tipoo90@hotmail.com

Received : 31 December 2020
Accepted : 17 February 2021
Published : 04 March 2021

DOI
10.25259/JCIS_235_2020

Quick Response Code:

ABSTRACT

Objectives: Cardiac magnetic resonance (CMR) imaging is very pertinent in the diagnosis and risk stratification of patients with hypertrophic cardiomyopathy (HCM). We aimed to assess the patterns of left ventricular (LV) hypertrophy, late gadolinium enhancement (LGE), and their prognostic significance in HCM patients in Pakistani population, as no such data are available from Pakistan.

Material and Methods: This was a retrospective, single center study. All patients who had confirmed diagnosis of HCM on CMR at Aga Khan University Hospital during the period of 2011–2019 were identified and included in the study.

Results: A total of 74 patients were included with the mean age of 45.6 ± 15 years and the majority 71.6 % (n = 53) being male. Maximal LV wall thickness was 21.1 ± 5 mm, asymmetrical septal hypertrophy being the most common pattern (62.2%, n = 46). LGE was present in 75.7% (n = 56) with most common site being septum plus LV free wall (24.3%, n =18). Mean ejection fraction% was found to be lower in patients with LGE (P < 0.001). Major adverse cardiac events (MACE) were observed in 40.5% (n = 30). Presence of LGE and right ventricular involvement was found to have a statistically significant association with MACE (P value 0.018 and 0.046, respectively). In multivariable analysis, only LGE was significantly associated with MACE (odd ratio: 4.65; 95% CI: 1.21–17.88).

Conclusion: Asymmetrical septal hypertrophy was the most common pattern of hypertrophy. LGE was present in three fourth of the study population and it was significantly associated with MACE.

Keywords: Hypertrophic cardiomyopathy, Cardiac magnetic resonance, Late gadolinium enhancement

INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is the leading cause of sudden cardiac death (SCD) in young people worldwide. It is a genetic cardiac disease with a heterogeneous phenotypic expression caused by autosomal dominant mutations in contractile sarcomeric proteins. It has a prevalence of 1:500 in general population.[1,2] Conventionally, it is diagnosed by the presence of left ventricular (LV) hypertrophy on 2D-echocardiography; however, cardiac magnetic resonance (CMR) is increasingly being used as a specific imaging modality.

CMR is more pertinent not only in the diagnosis of HCM but also in predicting the prognosis by detection of late gadolinium enhancement (LGE). CMR has an edge over echocardiography as it provides more accurate assessment of LV function and LV hypertrophy.[3] It can also accurately detect the different patterns of LV hypertrophy, that is, asymmetric, symmetric, apical, and mid ventricular.

Worldwide, much data are available on the patterns of hypertrophy and LGE on CMR in HCM. However, there is paucity of data on this topic in our population. This study was conducted to assess the patterns of LV hypertrophy and LGE on CMR, in HCM patients in Pakistani population. We also determined the prognostic significance of LGE in these patients. This is the first study of its kind in Pakistani population. As Aga Khan University Hospital (AKUH) is the first and the only center offering comprehensive CMR services in the country since 2011, so this was the best place to conduct such study.

MATERIAL AND METHODS

This study was conducted after approval from the ethical review committee. All the patients who had undergone CMR at AKUH with the suspected diagnosis of HCM, during the period 2011 to 2019 were identified using Health Information Management Services. Only those patients were included in the study who had confirmed diagnosis of HCM on CMR.

A pre-designed data entry form was filled for each patient after reviewing their medical records. Information was collected regarding age, gender, clinical features at presentation, Electrocardiogram (EKG) findings, echo data, and CMR findings. Follow-up data regarding death, arrhythmias and hospital admissions for heart failure were also collected by reviewing the medical records.

HCM

The diagnosis of HCM was based on the CMR demonstration of a hypertrophied LV (wall thickness ≥15 mm), with a non-dilated cavity in the absence of another cardiac or systemic disease that could produce the magnitude of hypertrophy evident.

Exclusion criteria

Patients with the diagnosis of cardiac amyloidosis, Fabry's disease, or cardiac sarcoidosis were excluded from the study. Similarly, patients with LV hypertrophy due to hypertension, aortic stenosis, or athlete's heart were excluded from the study.

CMR data acquisition

CMR was performed using a 1.5T Siemens Avanto scanner. A breath hold steady-state free-precession ECG-triggered sequence was used to evaluate global LV and right ventricular (RV) function. In each patient, two long axis views (one vertical and one horizontal) and two LVOT views were acquired. A set of contiguous short-axis views were also acquired from the mitral plane to the apex with the following parameters: Slice thickness 7 mm, distance factor 25%, field of view 34 cm, matrix 192 × 192, flip angle 80, TR/TE 58.74/1.12, and bandwidth 930 hz/px.

LGE images were obtained 8–10 min after bolus injection of gadolinium. Images were acquired in the same views as used for cine CMR. The inversion time was optimized to null signal from the normal myocardium.

CMR analysis

All the images were analyzed by a reader who was qualified and experienced in cardiovascular imaging. The analysis of CMR images was done on third party software – Medis Q mass. The endocardial and epicardial borders were drawn manually on the series of short axis cine slices of the LV at end-diastole and end-systole to obtain end-diastolic volume and end-systolic volume, respectively. The LV ejection fraction (EF) was calculated from the EDV and ESV, and presented as percentages to EDV.

LGE analysis was done visually; all tomographic short axis LV slices from base to apex were inspected to identify LGE. In addition, two chamber, four chamber, three chamber, and LVOT views were also inspected for identification of LGE visually.

RV involvement was considered when RV free wall thickness was >5 mm. Maximal LV wall thicknesses were defined as the greatest dimension anywhere within the LV myocardium. No definite cutoff for mild, moderate, and severe LV wall thickness/hypertrophy is available for CMR. We just took the echocardiographic cutoff values which are: Mild hypertrophy – male 11–13 mm, female 10–12 mm; moderate hypertrophy – male 14–16 mm, female 13–15 mm; and severe hypertrophy – male >16 mm, female >15 mm.

LVOT and mid cavity obstruction was assessed visually. Similarly, no quantification was done for assessment of severity of mitral regurgitation.

Follow-up

Follow-up clinical events were recorded by review of hospital records of clinic visits, hospital admissions, and telephonic interviews with the patient or a family member, in case the patient was unavailable. The clinical events considered were death, hospital admissions for heart failure, life-threatening arrhythmias, and appropriate Implantable Cardioverter Defibrillator (ICD) discharges. All deaths were presumed to be cardiac deaths unless a clear non-cardiac cause could

be established. Life-threatening arrhythmia was defined as documented ventricular tachycardia (VT) or ventricular fibrillation (VF) by EKG strips or ICD interrogation.

Statistical analysis

Data were entered and analyzed using the Statistical Package for the Social Sciences, version 24.0. Quantitative variables were expressed as mean and standard deviation or median (interquartile ranges) as appropriate. Qualitative variables were expressed as absolute frequencies and percentages. Qualitative data were compared using the two test or Fisher's exact test, as appropriate. Continuous data were compared using an independent samples *t* test or the Mann-Whitney U-test, depending on their distribution. A two-sided $P < 0.05$ was considered statistically significant for all tests.

RESULTS

A total of 74 patients who fulfilled the inclusion criteria were included in the study. Baseline clinical and demographic characteristics are summarized in Table 1. Mean age was 45.6 ± 15 years with the majority 71.6 % (n = 53) being male. Dyspnea (62.2%, n = 46) was the main symptom at presentation, followed by syncope (39.2%, n = 29).

Findings of CMR are shown in Table 2. Maximal LV wall thickness was 21.1 ± 5 mm with more than half of patients 56.8% (n = 42) having severe LV hypertrophy. Asymmetrical septal hypertrophy was the most common pattern of hypertrophy present in 62.2% (n = 46) of the patients, followed by septal and anterior wall hypertrophy (17.6%, n = 13). Various patterns of LV hypertrophy are shown in Figures 1-5.

LGE was present in 75.7% (n = 56) of the patients [Figures 6 and 7] and most common site was septum plus LV free wall (32.1%, n = 18), followed by apical site (17.8%, n = 10). Most common pattern of LGE was patchy which was present in 73.2% (n = 41).

Patients were divided into two groups on the basis of presence or absence of LGE [Table 3]. There was no statistically significant difference between two groups on basis of age and gender. However, patients with LGE were more likely to have systolic dysfunction (P = 0.018) and mean EF was also found to be lower in patients with LGE ($P < 0.001$). Maximal LV wall thickness and mean LV mass was also greater in patients with LGE (P = 0.001 and 0.016) compared to those without LGE.

Follow-up was available in all patients and mean follow-up duration was 39.6 + 27.3 months. Major adverse cardiac events (MACE) (Death, heart failure, VT/VF) were observed in 40.5% (30/74) of the study population [Table 4].

Table 1: Clinical and demographic characteristics.

Characteristics	Number (n=74)	Percentage
Age mean±SD	45.6±15	-
Male	53	71.6
Hypertension	28	37.8
Diabetes	8	10.8
Dyslipidemia	1	1.4
Family history of sudden cardiac death	23	31.1
Family history of hypertrophic cardiomyopathy	12	16.2
Symptoms at presentation		
Dyspnea	46	62.2
Syncope	29	39.2
Palpitations	25	33.8
Chest pain	16	21.6

Table 2: CMR findings in patients with hypertrophic cardiomyopathy.

CMR findings	Number (n=74)	Percentage
Mean EF	67.8±2	
>60%	62	83.8
51–59%	5	6.8
<50%	7	9.5
Maximal LV thickness (mm)	21.1±5	
Mild	6	8.1
Moderate	26	35.1
Severe	42	56.8
Pattern of hypertrophy		
Asymmetrical septal hypertrophy	46	62.2
Septum and anterior wall	13	17.6
Symmetric	5	6.8
Mid Ventricular	5	6.8
Apical	5	6.8
RV involvement	12	16.2
Increased LV Mass	57	77
LVOT obstruction	21	28.4
Mid-cavity obstruction	12	16.2
Systolic anterior motion	23	31.1
Mitral regurgitation	18	24.3
LGE	56	75.7
Site of LGE	(n=56)	
Septum+LV free wall	18	32.1
Apex	10	17.85
Septum	8	14.28
Multifocal	8	14.28
LV free wall	7	12.5
RV insertion site	5	8.92
Pattern of LGE		
Patchy	41	73.21
Focal	8	14.28
Transmural	4	7.14
Diffuse	2	3.57
Sub-endocardial	1	1.78

CMR: Cardiac magnetic resonance, LV: Left ventricle, RV: Right ventricle, LGE: Late gadolinium enhancement

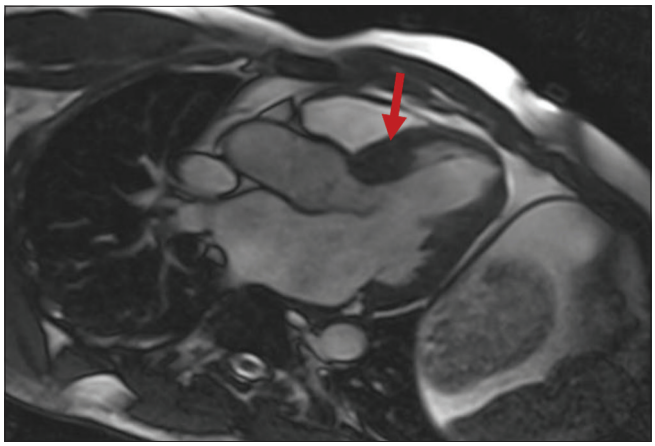

Figure 1: A 70-year old male with hypertension presented with history of syncope. Cardiac magnetic resonance steady-state free precession still frame 3chamber view showing asymmetrical septal hypertrophy (arrow).

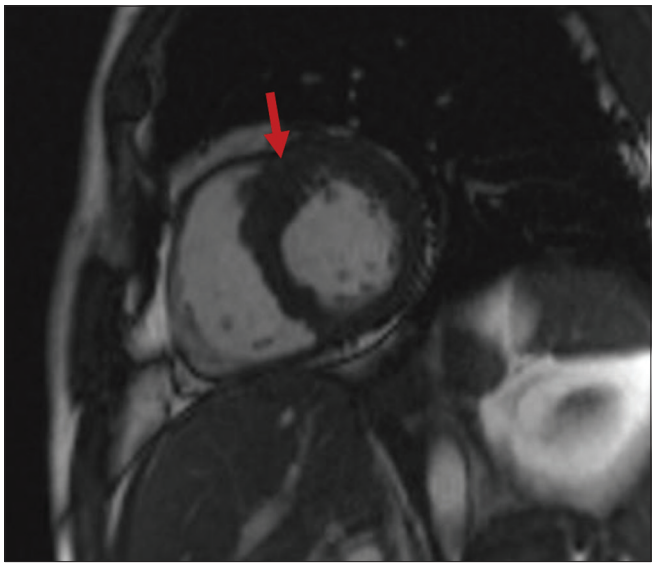

Figure 2: A 55-year old male with history of palpitation and left ventricular hypertrophy on echo. Cardiac magnetic resonance steady-state free precession still frame short axis view showing septal and anterior wall hypertrophy (arrow).

Patients were divided into two groups on the basis of presence or absence of MACE [Table 5]. The presence of LGE and RV involvement was found to have a statistically significant association with MACE. In multivariable analysis, only LGE was significantly associated with MACE (odd ratio: 4.65; 95% CI: 1.21–17.88).

DISCUSSION

This is the first study of its kind from this part of the world, describing the patterns of LV hypertrophy and LGE in patients with HCM on CMR, and looking at their prognostic

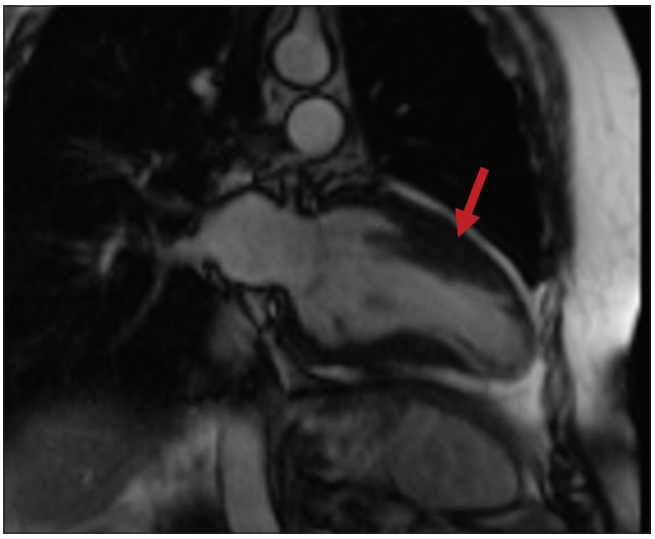

Figure 3: A 71-year old male with history of syncope. Cardiac magnetic resonance steady-state free precession still frame 2chamber view showing mid ventricular hypertrophy (arrow).

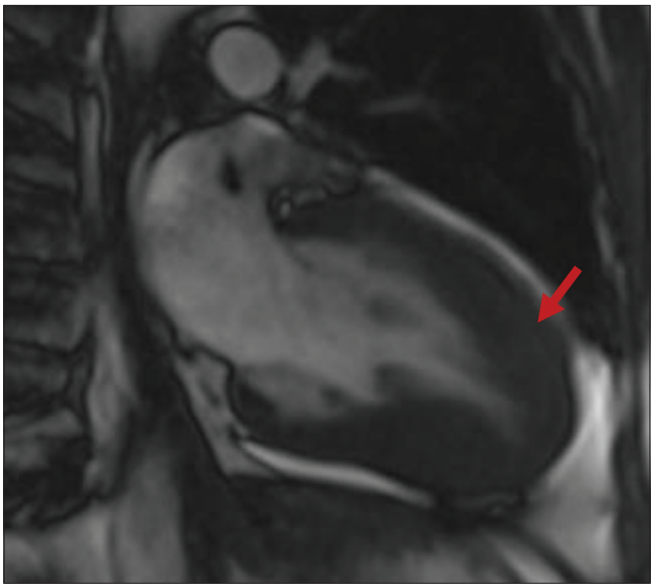

Figure 4: A 38-year old male with history of chest pain, diffuse T wave changes on ECG and normal coronary angiogram. Cardiac magnetic resonance steady-state free precession still frame 2chamber view showing apical hypertrophy (arrow).

significance. This will highlight the importance of CMR in the diagnosis and management of HCM in our population.

CMR plays a pivotal role in the diagnosis and risk stratification of patients with HCM. Due to high resolution, CMR determines the LV wall thickness accurately, compared to echocardiography. There are multiple morphological variants of HCM which are easily recognized by CMR. Asymmetrical septal hypertrophy was the most common morphologic presentation of HCM in our series. In this variant

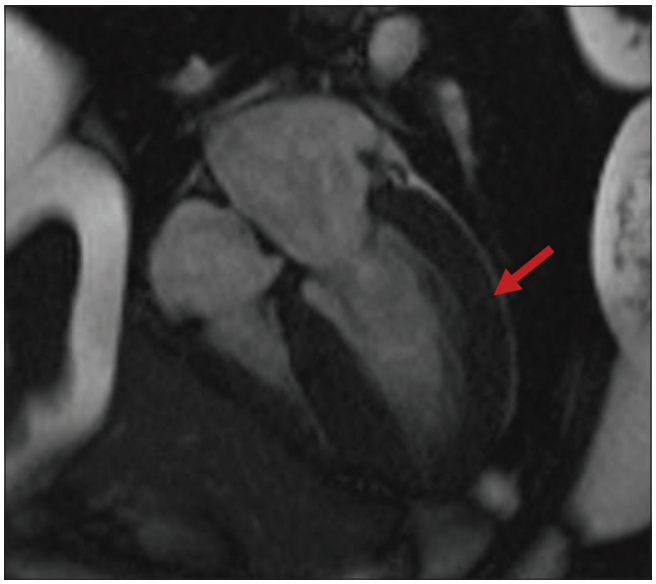

Figure 5: A 49-year old female with history of shortness of breath and left ventricular hypertrophy but no hypertension or aortic stenosis. Cardiac magnetic resonance steady-state free precession still frame 4chamber view showing symmetrical hypertrophy (arrow).

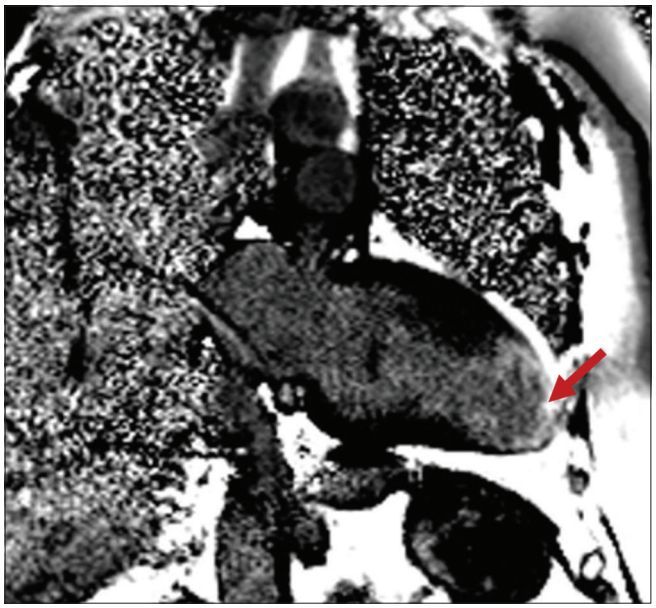

Figure 6: A 71-year old male with history of syncope. Cardiac magnetic resonance late gadolinium image showing apical hyper-enhancement in mid ventricular hypertrophy variant (arrow).

hypertrophy predominantly involves the septum.[4] Septal and anterior wall hypertrophy was the next common pattern of hypertrophy. In this variant hypertrophy predominantly involves the basal anterior wall and contiguous portion of the anterior inter-ventricular septum.[4]

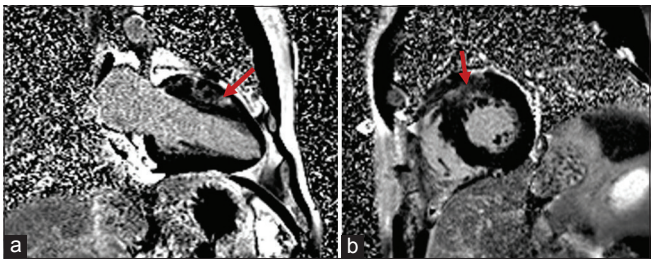

Figure 7: A 50-year old male with history of palpitation and syncope. (a) Cardiac magnetic resonance late gadolinium image 2chamber view showing hyper-enhancement in the anterior wall (arrow). (b) Cardiac magnetic resonance late gadolinium image short axis view showing hyper-enhancement in the anterior wall and adjacent septum (arrow).

Table 3: Comparison of patients with and without LGE.

	LGE+	LGE−	*P*-value
Age	(44±16)	(48.3±14.7)	0.43
Gender			
Male	43 (81.1%)	10 (18.9%)	0.08
Female	13 (61.9%)	1 (38.1%)	
Mean LV mass (g)	(231±99)	(175±40)	0.016
LV wall thickness	(22.3±5.4)	(17.5±2)	0.001
EF Mean	62.7±14.1	74.05±5.3	<0.001
EF			
>60%	43 (71%)	18 (29%)	0.018*
51–59%	5 (100%)	0 (0%)	
<50%	8 (100%)	0 (0%)	
LVOT obstruction	10 (47.6%)	11 (52.4%)	0.001

*Fischer's exact, LGE: Late gadolinium enhancement, LV: Left ventricle, EF: Ejection fraction

Table 4: Outcome of patients on follow-up.

Outcomes (Mean F/U 39.6±27.3)	F/U (*n*=74)	Percentage
Mortality	8	10.8
ICD insertion	33	44.5
Hospitalization	36	48.6
Causes of hospitalization		
Heart Failure	16	21.6
Ventricular Tachycardia/ Ventricular Fibrillation	12	16.2
Syncope	10	13.5
Angina	5	6.7
ICD discharge	11	14.8
Sudden cardiac death	1	1.3

ICD: Implantable cardioverter defibrillator

These findings are consistent with the findings in the literature, asymmetric HCM or septal HCM is the most

Table 5: Association of MACE with demographic and CMR findings.

	MACE+ (30/74)	MACE– (44/74)	*P*-value
LVEF	63.9±14.6	66.50±12.6	0.42
LV Mass	220.9±95.7	215.7±90.1	0.81
Age	42.8±17.5	47.5±14.2	0.21
LV Thickness	22.4±5.5	20.3±4.9	0.09
LGE+	27 (48.2%)	29 (51.8%)	0.018
LGE–	3 (16.7%)	15 (83.3%)	
RV Involvement (+)	8 (66.7%)	4 (33.3%)	0.046*
RV Involvement (–)	22 (35.5%)	40 (64.5%)	
LVOT+	10 (47.6%)	11 (52.4%)	0.43
LVOT–	20 (37.7%)	33 (62.3%)	

MACE: Major adverse cardiac events, CMR: Cardiac magnetic resonance, LVEF: Left ventricle ejection fraction, LV: Left ventricle, RV: Right ventricle, LGE: Late gadolinium enhancement

common morphologic presentation accounting for about two thirds of the spectrum.[4,5]

In mid ventricular hypertrophy variant, the hypertrophy predominantly involves the mid segments of the left ventricle and may result in mid ventricular obstruction.[6,7] It may be associated with the formation of an apical aneurysm. This variant was seen in small number of patients in our study.

In apical variant, the hypertrophy predominantly involves the apical segments. Diagnostic criteria for apical HCM include an absolute apical wall thickness of >15 mm or a ratio comparing apical LV and basal LV wall thicknesses of ≥1.3–1.5.[8,9] Apical HCM also called "Yamaguchi syndrome" is a relatively uncommon form of HCM. This variant is more frequent in the Japanese population. In this condition, there is obliteration of LV cavity at the apex, giving a characteristic spade-like configuration. In our study, apical variant was found in 6.8% ($n = 5$), which is a little higher than the figures reported from western countries (~2%) but significantly lower than that of Japan (~25%).[4,8]

In the variant with symmetrical hypertrophy, the hypertrophy involves the ventricular wall symmetrically with no regional preferences. LV cavity dimensions are reduced in a concentric fashion.[7,10] This entity should be evaluated closely to differentiate it from other causes of symmetric LV thickening.

On contrast enhanced CMR, areas of LGE can also be detected, which are considered to represent areas of fibrosis. Identification of fibrosis by LGE technique has prognostic significance in predicting adverse clinical outcomes. In our study, LGE was present in 75.7% cases, which is little higher than the figures reported in the literature. LGE was reported in 55% by Maron *et al.*,[11] 42% by Chan *et al.*,[12] 63% by O'Hanlon *et al.*,[13] 68% by Lyon *et al.*,[14] 66.2% by Ismail *et al.*,[15] and 68.9% by Klopotowski *et al.*[16] There are only few studies on the Asian population and, to the best of our knowledge, no data are available on LGE in sub-continental South Asian HCM population. LGE was observed in 73% of study population in Japan by Hen *et al.*[17] Choi *et al.*[18] reported a significantly higher detection of LGE in 91.5% of study population in South Korea. The pooled prevalence of LGE was found to be 60% in a meta-analysis of four studies which evaluated 1063 patients.[19] The variable prevalence can be explained by the different study population, study designs, inter-observer variability, and LGE detection methods.

Most common location for LGE in our study was septum and LV free wall, which was also observed in studies by Maron *et al.* and Olivotto *et al.*[11,20] However, apex was the most common site in a study by Hen *et al.* in Japanese cohort.[17]

Severity of hypertrophy showed statistically significant association with the presence of LGE in our study, which is in agreement with the findings of studies by Ismail *et al.* and others.[11,15,20] Our study found a statistically significant association between LGE and LVOT obstruction, in contrast no such association was found by Maron *et al.*[11] Association of LGE with systolic dysfunction and lower EF was reported in the studies,[11,14,15] and similar findings were observed in our study.

A high rate of MACE was observed in our study population, which is similar to what Songsirisuk *et al.*[21] observed. Cardiovascular complications were reported in 47% of Chinese HOCM population by Ho *et al.*[22] and in 35.6% by Lee *et al.*[23] in Taiwan. However, in contrast lower rate was reported by studies in Europe and USA.[13,24,25] These differences are probably due to the heterogeneous population, variable definition of MACE, rate of ICD insertion, and health-care resources. MACE were more common in patients with LGE, presence of LGE has been found to increase the risk of SCD, cardiovascular complications, and increase in all-cause mortality.[26,27]

The prevalence of RV involvement is 16.2% in our study which is similar to 18% reported in the literature.[4,7] This typically involves the mid-to-apical portion of the RV. RV involvement was also found to be associated with cardiovascular complications in our study and this was in agreement with various other studies,[28,29] in which RV involvement was associated with increased risk of sudden death and ventricular arrhythmias.

LVOT obstruction was present in 28.4% of the patients in our study, which is comparable to that found in the studies done by Songsirisuk *et al.* (24%) and Maron *et al.* (25%).[21,30] However, in a study done by Lee *et al.* on Taiwanese population, LVOT obstruction was demonstrated in a much higher percentage (48.1%) of HCM patients.[23]

LVOT obstruction was also associated with MACE[22] and was a strong, independent predictor of progression to severe

symptoms of heart failure and death[30] in studies done by Ho *et al*. and Maron *et al*., respectively, which was not the case in our study group.

Limitations

Our study has several limitations; it is a retrospective, single-center study, resulting in a smaller sample size. LGE was assessed qualitatively and no quantification was done in terms of percentage of myocardium involved. The major limitation of this study is that the HCM has already been widely studied and the information is not especially novel with regard to the underlying condition. However, this is the first study of its kind from this region, which has evaluated the findings on CMR and cardiovascular complications in HCM patients.

Our hospital serves as the only center offering CMR facility to a large area of population and hence has been a referral center for CMR in patients with HCM. Therefore, the results of this study may be representative of the Pakistani population with HCM.

CONCLUSION

Asymmetrical septal hypertrophy was the most common pattern of hypertrophy, followed by septal and anterior wall hypertrophy. LGE was present in three fourth of the study population, with patchy enhancement being the most common pattern. Patients with LGE were more likely to have systolic dysfunction and mean EF was found to be lower in patients with LGE. MACE (Death, heart Failure, and VT/VF) were observed in less than half of the patients. The presence of LGE and RV involvement was found to have statistically significant association with MACE.

Despite the limitations of this study, the presence of LGE seems to be a promising additional risk marker in predicting outcome in HCM patients, in this region too. After the availability of this local data, there should be no hesitancy in early use of CMR for diagnosis and risk stratification of patients with HCM in our population.

Acknowledgments

None.

Declaration of patient consent

Institutional Review Board (IRB) permission obtained for the study.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Elliott P, McKenna WJ. Hypertrophic cardiomyopathy. Lancet 2004;363:1881-91.
2. Klues HG, Schiffers A, Maron BJ. Phenotypic spectrum and patterns of left ventricular hypertrophy in hypertrophic cardiomyopathy: Morphologic observations and significance as assessed by two-dimensional echocardiography in 600 patients. J Am Coll Cardiol 1995;26:1699-708.
3. Olivotto I, Maron MS, Autore C, Lesser JR, Rega L, Casolo G, *et al*. Assessment and significance of left ventricular mass by cardiovascular magnetic resonance in hypertrophic cardiomyopathy. J Am Coll Cardiol 2008;52:559-66.
4. Maron MS, Maron BJ, Harrigan C, Buros J, Gibson CM, Olivotto I, *et al*. Hypertrophic cardiomyopathy phenotype revisited after 50 years with cardiovascular magnetic resonance. J Am Coll Cardiol 2009;54:220-8.
5. Rickers C, Wilke NM, Jerosch-Herold M, Casey SA, Panse P, Panse N, *et al*. Utility of cardiac magnetic resonance imaging in the diagnosis of hypertrophic cardiomyopathy. Circulation 2005;112:855-61.
6. Wigle ED. Cardiomyopathy: The diagnosis of hypertrophic cardiomyopathy. Heart 2001;86:709-14.
7. Hansen MW, Merchant N. MRI of hypertrophic cardiomyopathy: Part I, MRI appearances. AJR Am J Roentgenol 2007;189:1335-43.
8. Moon JC, Fisher NG, McKenna WJ, Pennell DJ. Detection of apical hypertrophic cardiomyopathy by cardiovascular magnetic resonance in patients with non-diagnostic echocardiography. Heart 2004;90:645-9.
9. Eriksson MJ, Sonnenberg B, Woo A, Rakowski P, Parker TG, Wigle ED, *et al*. Long term outcome in patients with apical hypertrophic cardiomyopathy. J Am Coll Cardiol 2002;39:638-45.
10. Cannavale A, Ordovas KG, Higgins CB. Magnetic resonance imaging of hypertrophic cardiomyopathy. J Thorac Imaging 2010;28:W12-8.
11. Maron MS, Appelbaum E, Harrigan CJ, Buros CJ, Gibson J, Hanna CM, *et al*. Clinical profile and significance of delayed enhancement in hypertrophic cardiomyopathy. Circ Heart Fail 2008;1:184-91.
12. Chan RH, Maron BJ, Olivotto I, Pencina MJ, Assenza GE, Haas T, *et al*. Prognostic value of quantitative contrast-enhanced cardiovascular magnetic resonance for the evaluation of sudden death risk in patients with hypertrophic cardiomyopathy. Circulation 2014;130:484-95.
13. O'Hanlon R, Grasso A, Roughton M, Moon JC, Clark S, Wage R, *et al*. Prognostic significance of myocardial fibrosis in hypertrophic cardiomyopathy. J Am Coll Cardiol 2010;56:867-74.
14. Lyons KS, Dixon LJ, Johnston N, Noad R, Hamilton A, McKeag N, *et al*. Late gadolinium enhancement is common in patients with hypertrophic cardiomyopathy and no clinical risk factors for sudden cardiac death: A single center experience. Cardiol J 2014;21:29-32.
15. Ismail TF, Jabbour A, Gulati A, Mallorie A, Raza S, Cowling TE,

et al. Role of late gadolinium enhancement cardiovascular magnetic resonance in the risk stratification of hypertrophic cardiomyopathy. Heart 2014;100:1851-8.
16. Klopotowski M, Kukula K, Malek LA, Spiewak M, Polanska-Skrzypczyk M, Jamiolkowski J, *et al*. The value of cardiac magnetic resonance and distribution of late gadolinium enhancement for risk stratification of sudden cardiac death in patients with hypertrophic cardiomyopathy. J Cardiol 2016;68:49-56.
17. Hen Y, Iguchi N, Utanohara Y, Takada K, Machida H, Takayama M, *et al*. Prognostic value of late gadolinium enhancement on cardiac magnetic resonance imaging in Japanese hypertrophic cardiomyopathy patients. Circ J 2014;78:929-37.
18. Choi HM, Kim KH, Lee JM, Yoon YE, Lee SP, Park EA, *et al*. Myocardial fibrosis progression on cardiac magnetic resonance in hypertrophic cardiomyopathy. Heart 2015;101:870-6.
19. Green JJ, Berger JS, Kramer CM, Salerno M. Prognostic value of late gadolinium enhancement in clinical outcomes for hypertrophic cardiomyopathy. JACC Cardiovasc Imaging 2012;5:370-7.
20. Olivotto I, Maron BJ, Appelbaum E, Harrigan CJ, Salton C, Gibson G, *et al*. Spectrum and clinical significance of systolic function and myocardial fibrosis assessed by cardiovascular magnetic resonance in hypertrophic cardiomyopathy. Am J Cardiol 2010;106:261-7.
21. Songsirisuk N, Kittipibul V, Methachittiphan N, Charoenattasil V, Zungsontiporn N, Spanuchart I, *et al*. Modes of death and clinical outcomes in adult patients with hypertrophic cardiomyopathy in Thailand. BMC Cardiovasc Disord 2019;19:1.
22. Ho H, Lee KL., Lau C, Tse H. Clinical characteristics of and long-term outcome in chinese patients with hypertrophic cardiomyopathy. Am J Med 2004;116:19-23.
23. Lee C, Liu P, Lin L, Chen J, Tsai L. Clinical characteristics and outcomes of hypertrophic cardiomyopathy in Taiwan-a tertiary center experience. Clin Cardiol 2007;30:177-82.
24. Maron BJ, Rowin EJ, Casey SA, Haas TS, Chan RH, Udelson JE, *et al*. Risk stratification and outcome of patients with hypertrophic cardiomyopathy >=60 years of age. Circulation 2013;127:585-93.
25. Olivotto I, Cecchi F, Poggesi C, Yacoub MH. Patterns of disease progression in hypertrophic cardiomyopathy. Circ Heart Fail 2012;5:535-46.
26. Weng Z, Yao J, Chan RH, He J, Yang X, Zhou Y, *et al*. Prognostic value of LGE-CMR in HCM: A meta-analysis. JACC Cardiovasc Imaging 2016;9:1392-402.
27. He D, Ye M, Zhang L, Jiang B. Prognostic significance of late gadolinium enhancement on cardiac magnetic resonance in patients with hypertrophic cardiomyopathy. Heart Lung 2018;47:122-6.
28. Roşca M, Călin A, Beladan CC, Enache R, Mateescu AD, Gurzun MM, *et al*. Right ventricular remodeling, its correlates, and its clinical impact in hypertrophic cardiomyopathy. J Am Soc Echocardiogr 2015;28:1329-38.
29. Keramida K, Lazaros G, Nihoyannopoulos P. Right ventricular involvement in hypertrophic cardiomyopathy: Patterns and implications. Hellenic J Cardiol 2020;61:3-8.
30. Maron MS, Olivotto I, Betocchi S, Casey SA, Lesser JR, Losi MA, *et al*. Effect of left ventricular outflow tract obstruction on clinical outcome in hypertrophic cardiomyopathy. N Engl J Med 2003;348:295-303.

How to cite this article: Sultan FAT, Saadia S. Patterns of left ventricular hypertrophy and late gadolinium enhancement on cardiac MRI in patients with hypertrophic cardiomyopathy and their prognostic significance – An experience from a South Asian country. J Clin Imaging Sci 2021;11:14.