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Role of Cardiac MRI in Diagnosis, Follow Up and Post Management Evaluation of Hypertrophic Cardiomyopathy

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ABSTRACT

Background: Cardiac magnetic resonance imaging (CMR) is a valuable assessment tool for assessment of hypertrophic cardiomyopathy (HCM). Many advanced sequences is rapidly developing.

Objective: To assess the value of Cardiac MRI In diagnosis, Follow Up and Post-management evaluation of hypertrophic cardiomyopathy.

Patients and Methods: A diagnostic test accuracy study with prospective evaluation was performed in Cardiology Department of Kobry Elkobba Military Medical Complex and Radiology and Medical Imaging Department of Magdy Yacoup Heart Foundation. Cardiac MRI was done for 50 patients who were screened for HCM, were suspected to have HCM by clinical and echocardiographic examination and some come for follow up.

Results: 74% of the cases had asymmetrical septal hypertrophy. 40% of the cases had systolic anterior motion of the mitral valve leaflet (SAM). 28% of the patients had left ventricular outflow obstruction (LVOTO), 85.7% of them had asymmetric septal phenotype and all had SAM. 18% of the cases had systolic midventricular obliteration (MVO), and all of them had mid ventricular phenotype. 62.1% of the cardiac segments exhibited extracellular volume (ECV) expansion with no evident LGE with significant P-value (0.009). Follow up of the 6 patients underwent septal myomectomy revealed that left ventricular thickness range decreased from 22 to 26 mm to 18 to 24 after the operation. The SAM, mitral regurge and LVOTO resoluted in most of cases.

Conclusions: MRI is a crucial technique for diagnosis, follow up and risk stratification for patients with suspected or diagnosed to have HCM.

Keywords: Cardiomyopathy, Hypertrophic, Magnetic resonance, Phenotype, Risk assessment.

INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is the most prevalent genetic form of cardiomyopathy, cardiac magnetic resonance imaging (CMR) is advantageous over other imaging modalities in the assessment of individuals with suspected or confirmed HCM [1-2].

CMR capacity to describe cardiac tissue is an additional benefit. The relationship between gadolinium retention by the myocardium in fibrotic regions and clinical consequences has been intensively studied. Recently new T1 mapping methods, which may offer information on diffuse fibrosis, have enhanced this [3]. CMR is also a valuable method for the differential diagnosis of HCM, as it can distinguish this disease from others that present with an increase in ventricular width, such as the physiologic changes associated with high-performance athletes, cardiac deposit diseases, aortic valvular disease and hypertensive cardiomyopathy [2].

Our study aimed to assess the value of cardiac MRI in diagnosis, risk stratification and Follow Up of patients with hypertrophic cardiomyopathy.

PATIENTS AND METHODS

This study was recruited in Cardiology Department of Kobry Elkobba Military Medical Complexes and Radiology and Medical Imaging Department of Magdy Yacoup Heart Foundation over the period from October 2020 to December 2021. 50 patients were enrolled in this study, 33 males and 17 females, their ages ranged from 17 to 69 years with a mean age of 40.5 ± 12.8 years.

Inclusion criteria: The patients included in this study were asymptomatic patients with positive family history of HCM, suspected cases by clinical history, echocardiography examination, known cases of HCM under medical treatment and known cases of HCM underwent surgical myomectomy, alcohol ablation or had MRI safe or conditional implantable cardioverter defibrillators (ICDs).

Exclusion criteria: Patients with MRI non-compatible cardiac devices. Patients with metallic implants (Ex. Mechanical Prosthetic valves), patients with gadolinium hypersensitivity, patients with renal impairment of GFR below 30 ml/min and patient with severe claustrophobia. Patients with left ventricular hypertrophy due to infiltrative cardiomyopathy and hypertension or athletics.

All patients included in this study were subjected to complete history taking, clinical examination by the cardiologist, cardiac MRI examination with the studying of myocardial function and viability.

Prior to the examination, each patient was given a thorough explanation of the procedure. Patients were informed that they would have to be stable during the exam and that they will hear loud noise from the system. Training patients to hold their breath for relatively

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lengthy periods of time by having them undertake a deep inhalation and continuing to hold their breath without pushing.

A closed MRI machine 1.5-T magnet (Siemens MAGNETOM Aera) was used at Kobry Elkobba Military Medical Complexes and (Siemens MAGNETOM Sola) and also at Magdy Yacoup Heart Foundation, Radiology and Medical Imaging Department. The examination required about 30 minutes for imaging and about 20 minutes for interpretation.

Prior to the installation of ECG leads, skin preparation was required for good electrode contact and reliable detection of myocardial potentials. This was achieved by shaving of chest hair, using clean gauze to thoroughly dry the skin area for positioning of the electrodes using commercially prepared gels for this purpose.

The patients were instructed to lay supine on the MRI table. Four ECG electrodes were put closely together on the anterior chest wall of the left parasternal aspect in an L-shaped arrangement. The QRS complex is then examined on the MRI display, and the location of the leads was adjusted appropriately.

The respiratory sensor was positioned across the largest region of respiratory movement (abdomen or thorax). There was a strap used to fasten the sensor. When the respiratory wave emerged on the monitor, the respiratory signal was examined to establish the patient's breathing pattern and to synchronize breath hold instructions with the patient's capabilities. Then, the multi-element phased-array coils (8 channels) necessary for parallel imaging were used with the body coil for imaging.

Table (1) showed the summary of institutional MRI protocol for HCM.

Table (1): The summary of institutional MRI protocol for HCM

Sequence	Planes and coverage	Goal of sequence	Parameters
T1-weighted double inversion rapid spin-echo recovery	Axial between the thoracic inlet and diaphragm.	Anatomical overview	TR; 1.000 msec; TE: 35-40 msec; 6 mm slice thickness; matrix: 256x256
Steady state free precession	Left ventricular short axis (base-apex stack); 2-chamber; 3-chamber; 4-chamber	Evaluation of left ventricular mass and function; evaluation of compromised left ventricular outflow tract, systolic anterior mitral valve motion, and mitral regurgitation	TR: 3 to 5 msec; TE: 1.5 to 2 msec; flip angle: 55o; slice thickness: 8 mm (with 2 mm interslice gap for short axis stack); matrix: 128 x 256.
Delayed improvement (T1-weighted 2D gradient echo at 10-15 min)	2-chamber; 3-chamber;	Evaluation of regions of myocardial fibrosis and/or autoinfarction	TR; 5-6 msec; TE: 2-3 msec; flip angle: 50o; 10 mm slice thickness matrix: 256x256
Phase contrast imaging (PC-MRI)	Left ventricular outflow tract (through plane and/or inplane); mitral valve orifice (through plane)	To quantify any outflow tract peak systolic velocity; to assess diastolic filling velocities	TR; 30 msec; TE: 1.2 msec; flip angle: 30^{o}; 5 mm slice thickness voxel size: 2x1.2x6 mm; velocity encoding value: 150-250 cm/s
TI mapping (pre and post contrast	pre- and 15 min following bolus contrast administration in 3 short-axis images (basal, midventricular, and apical levels)	Tissue characterization and quantitative assessment of fibrosis	echo time/repetition time = .03/413.57ms, field of view = 450 × 450mm,matrix size = 256 × 169, cardiac delay time TD = 500ms, 0.22-s acquisition time for a single image, and The 5(3)3 MOLLI variant was used

The interpretation was performed by three individuals with MD degree and about 3-5 years longevity experience in cardiac imaging. The interpretation was carried out by a collaborative reading crew.

The images were uploaded to a Siemens workstation for post-processing and image analysis. With the help of the short axis pictures, the endocardial margins of both ventricles were manually traced by cine images during peak systole and end diastole giving the following functional parameters: LV end – diastolic volume (in milliliters), LV end – systolic volume (in milliliters), stroke volume (in milliliters), and LV ejection fraction (in percent), RV end – diastolic volume (in milliliters), RV end – systolic volume (in milliliters), stroke volume (in milliliters) and RV ejection fraction (in percent).

The normal LV ejection fraction = > 50%, Supernormal when equal or more than 65%, end stage HCM when the ejection fraction = < 50%.

The effect of HCM on regional function was assessed qualitatively by film analysis using a 17-segment model (defined as normal, akinetic, dyskinetic or hypokinetic).

For morphological assessment, HCM considered as non-dilated left ventricular hypertrophy (LVH) when other cardiac or systemic causes of LVH are absent. The presence of HCM is clinically indicated in adults with a left ventricular (LV) wall thickness more than 15 mm as evaluated by echocardiography or cardiac MRI. A two-standard-deviation or greater increase in left ventricular (LV) wall thickness over the age- or BMI-specific mean is diagnostic of this condition in children.

In the longitudinal plane, the LV short-axis stack was separated into three about equal levels (basal, middle, and apical). Each of these three levels consisted of three to four consecutive short-axis slices, with an average of nine to twelve slices per patient. In the short-axis plane, at the mid and basal LV levels, each slice was automatically separated into 6 equal segments by specialized software, whilst the apical level slices were segmented into 4 segments, with the apex being the last segment.

In accordance with the normal American Heart Association segmentation methodology, a total of 17 LV segments were evaluated for each patient. For each short-axis slice, the junction of the ventricular septum and anterior free wall was determined by the insertion point of the right ventricular wall. The LV free wall was separated into four equal segments: anterior, posterior, inferolateral, and anterolateral. While, the ventricular septum was dissected into two equal halves at the base and the middle of the LV. The apex was subdivided into four equal parts: the septum, the front, the sides, and the bottom.

Using commercial software, the maximum LV wall thickness measurements for each section were automatically computed. In the three LV levels (i.e., basal, mid, and apical), the 17 segments with the largest wall thickness (excluding the apex) were recorded. The percentage of hypertrophied LV chamber was determined by dividing the number of LV segments with thicker walls (15 mm) by the total number of LV segments.

The patient was categorised as having a pattern of noncontiguous LV hypertrophy if there was at least 1 segment of normal LV myocardial wall thickness between 2 or more adjacent segments of hypertrophied myocardium in either the circumferential (short axis) or longitudinal (long axis) cross-sectional plane.

Evaluation of LVOT was done by assessing the degree of hypertrophy and the precise location of the thickening of the myocardium, assessing the valvular and subvalvular mitral apparatus for anomalies including enlarged mitral valve leaflets or abnormal anterior papillary muscle insertion, SAM are evaluated in cine SSFP sequences.

The area of the hole of LVOTO was measured by trans planar flow planimetry using phase contrast sequences. The peak systolic velocity and the pressure gradient is estimated using the phase contrast velocity encoding sequence. Resting left ventricular outflow tract obstruction defined as a peak pressure gradient at rest > 30 mmHg.

During diastole, when the leaflets are fully extended in a direction parallel to the anterior septum and left ventricular (LV) free wall, the AML and PML were measured. The length of each leaflet was measured from its most distal position to where it was inserted into the posterior aortic wall for the anterior leaflet and from its most distal point to where it was inserted into the basal left ventricular posterior free wall for the posterior leaflet. AML was considered elongated when exceed 30 mm and PML was considered elongated when exceeded 17 mm in length.

Analysis of the 3-chamber view cine was performed in real-time, with measurements taken in stop-frame mode utilizing internal calibration to distinguish between the mitral valve leaflet tip and neighboring chordae tendinae.

Tissue characterization and fibrosis analysis was done by qualitative Assessment of fibrosis by LGE, localization was based on the AHA 17- segment model. Threshold > or = 6 SDs exceeding the mean for non-enhanced myocardium was used to define areas of Late gadolinium enhancement.

For the assessment of myocardial ECV, SAX T1 map images were evaluated in which an area of interest (ROI) > 12 pixels was manually created on the pre- and post-contrast pictures in each of the myocardial segments, except the apex, for a total of 16 segments per patient. In the blood pool, a ROI was also drawn in the pre- and post-contrast images. To minimize contamination and partial volume effects at the

endocardial and epicardial borders, these ROIs were rigorously crafted. The ECV percentage was determined to be extended at 29-30% or above. The ECV was then calculated using the formula below:

$$ECV = (1-Hct) \times (\Delta R1_{myocardium}/\Delta R1_{blood}),$$

Where Hct is the hematocrit level, and ΔR1 represents the change in T1 relaxivity (R1=1/T1) before and after gadolinium-based contrast administration.

Ethical consent: Our institution's Ethical Committee supervised and authorized all research methods (Kobry Elkobba Military Medical Complexes and Radiology and Medical Imaging Department of Magdy Yacoup Heart Foundation). Before enrolling in this research, all participants got a thorough description of its purpose, aims, and methods. All participants in the present study provided written informed consent for publication of the study's data. The Declaration of Helsinki for human beings, which is the international medical association's code of ethics, was followed during the conduct of this study.

Statistical analysis

SPSS (statistical programme for the social sciences software) statistics package version 20 was used to tabulate and analyze the acquired data on an IBM compatible computer. The mean and standard deviation were provided for numerical data, whereas the number and percentage were recorded for qualitative data. Chi-square test by Pearson (X^2) examining the link between two qualitative variables requires the significance test. P-values of 0.05 were used to evaluate statistical significance, with values 0.05 being considered statistically significant. Highly statistically significant is defined as P-value 0.001.

RESULTS

The Phenotypic classification of the studied 50 cases showed that 74% (37/50) of our patients had asymmetric septal phenotype, 18% (9/50) of our patients had mid ventricular phenotype, 4% (2/50) of our patients had focal phenotype and 2 % (1 /50) had apical and symmetric phenotype (Table 2).

Table (2): Phenotypic distribution of the studied 50 patients.

Phenotype	N	%
Asymmetric septal	37	74
Symmetric concentric	1	2
Apical	1	2
Mid ventricular	9	18
Focal (mass like)	2	4
Total	50	100

Left ventricular outflow tract obstruction (LVOTO) was found in 28% (14/50) of the study group, 85.71% (12/14) of the patient with LVOTO had asymmetric septal phenotype with basal segments involvement and one patient had focal phenotype affected the basal septal segment and the other had symmetric concentric basal hypertrophy. All the 14 patients had SAM (Table 3).

Table (3): The relation between the phenotype, LVOTO and SAM in the study group

		LVOTO						Chi-Square	
		No		Yes		Total			
		N	%	N	%	N	%	X^2	P-value
Phenotype	**Asymmetric septal (with basal segments Involvement)**	25	69.4	12	85.7	37	74	7.301	0.121
	Symmetric concetric	0	0	1	7.1	1	2		
	Apical	1	2.8	0	0	1	2		
	Midventriculer	9	25	0	0	9	18		
	Focal (mass like)	1	2.8	1	7.1	2	4		
SAM	**No**	10	27.8	0	0	10	20	5.504	0.138
	Chordal	4	11.1	1	7.1	5	10		
	AML	7	19.5	4	28.6	11	22		
	Mixed	15	41.7	9	64.3	24	48		

The ECV was evaluated in only 15 patients of the study group and the relation of expanded ECV and LGE in the examined 15 patients showed that 37.9 of cardiac segments had expanded ECV and LGE, 62.1% of cardiac segments showed expanded ECV with no LGE and 22.2 % of the cardiac segments showed LGE with no expanded ECV (Table 4).

Table (4): The relation of ECV and LGE in the examined 15 patients.

LGE	Expanded ECV						Chi-Square	
	Yes		No		Total			
	N	%	N	%	N	%	X^2	P-value
Yes	50	37.9	24	22.2	74	30.8	6.828	0.009*
No	82	62.1	84	77.8	166	69.2		
Total	132	100.00	108	100.00	240	100.00		

Follow up the 6 patients underwent myomectomy revealed that the left ventricular thickness ranged from 22 to 26 mm before the operation and from 18 to 24 after the operation with significant p-value of 0.020. 4 patients (4/6) had LVOTO before the myomectomy and after the operation the whole 4 patients showed resolution of the LVOTO. The whole 6 patients had SAM before the surgery and after surgery only 4 had resolution of the previous SAM. The whole 6 patients had MR before the myomectomy and 3 of them had reduction in the severity of the MR after myomectomy (Table 5).

Table (5): LVOTO, SAM and MR before and after myomectomy in the study group

		Before		After		Chi-Square	
		N	%	N	%	X^2	P-value
MR	Yes	6	100	3	50	1.778	0.182
	Decreased	0	0	3	50		
SAM	No	0	0	4	66	3.375	0.066
	Yes	6	100	2	33		
LVOTO	No	2	33	6	100	3.375	0.066

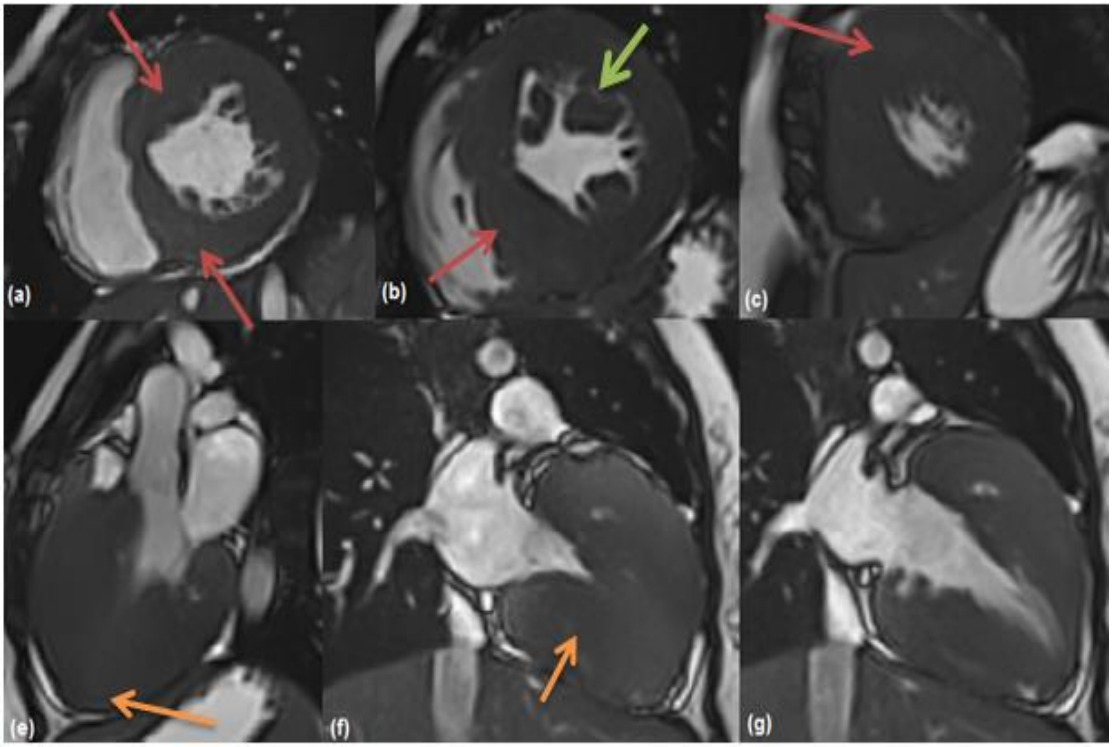

Figure (1): Non uniform hypertrophied myocardial segments with midventricular and apical obliteration. Steady state free precession (SSFP) short axis (a, b, c), 3- chamber view (e) and 2-chamber views in systole (f) and in diastole (g) show non-uniform hypertrophied myocardial segments, most pronounced at the mid ventricular segment of the infero-septal wall (30 mm thickness) (b, red arrow) involving the basal segments of anterior, septal and inferior walls (a, red arrows). All mid ventricular segments of LV (b, red arrow). All apical segments particularly anterior wall (c, red arrow), Hypertrophied both papillary muscles (b, green arrow) and Apical (e, orange arrow) and mid cavitary (f, orange arrow) systolic obliteration is noted.

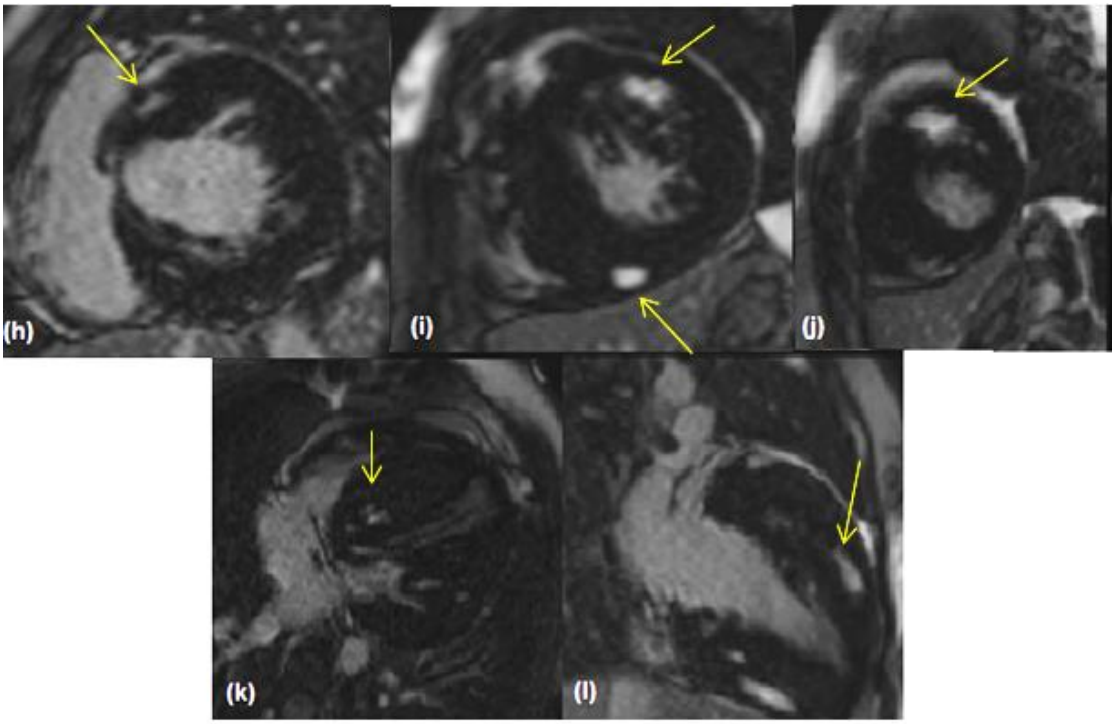

Figure (2): LGE non-territorial patchy mid-myocardial LGE involving most of the hypertrophied segments. LGE short axis views basal (h), midventricular (i) and apical (j) cuts,4-chamber view (k) and 2-chamber view (l) showed non-territorial patchy mid-myocardial enhancement involving most of the hypertrophied segments (basal to mid anterior, anteroseptal & inferoseptal walls, mid to apical lateral walls (yellow arrows).

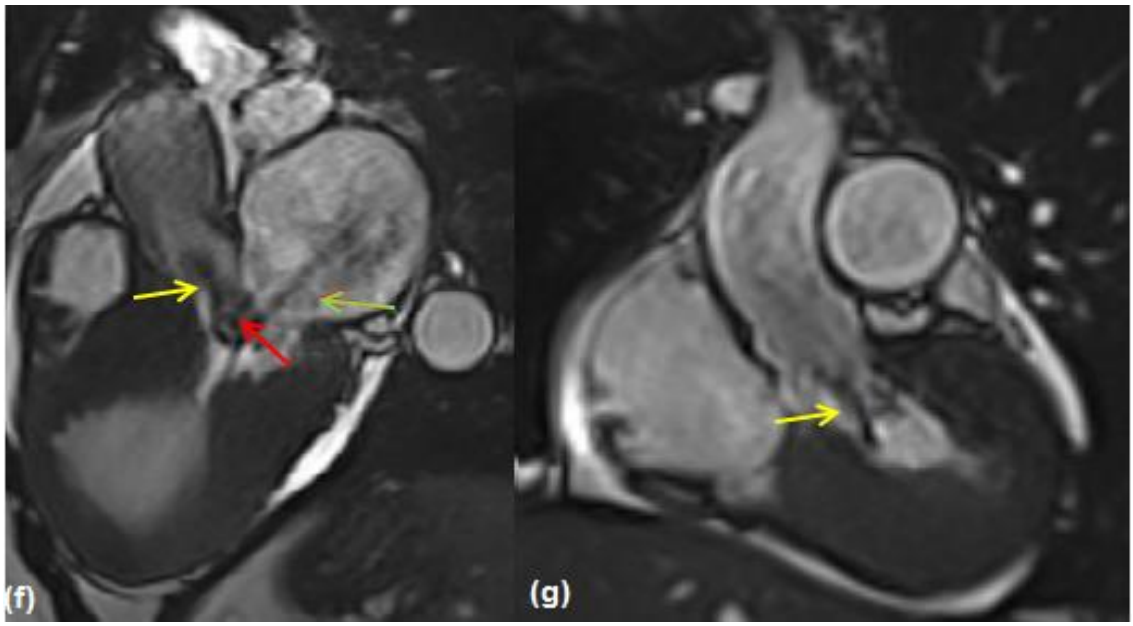

Figure (3): SSFP 3-chamber view (f) and LVOT view (g) showed SAM of the anterior mitral valve leaflet (f, red arrow) with LVOT obstruction showing black jet through the LVOT (candle flame artifact) (f and g, yellow arrows) and mitral regurge , black jet (f, green arrow).

DISCUSSION

Our research aimed to study the valuable role of CMR in evaluation of patients with HCM starting from the screening ending to risk stratification using advanced sequences. There are different phenotypic type of HCM. In our study the most detected type was asymmetrical hypertrophy, with frequent involvement of the basal segments of the anterior wall and anterior ventricular septum. This agrees with **Bogart and Olivotto** [4] who showed that most of cases to have asymmetric and segmental involvement with 70% of patients the hypertrophied regions involve the basal segments of the anterior ventricular septum and anterior wall and 5% of patient had diffuse concentric hypertrophy and **Florian *et al.*** [5] discovered asymmetric septal hypertrophy in 132 out of 150 patients.

In our research, LVOTO that was produced by mechanical and dynamic causes (basal septal hypertrophy, SAM, and aberrant chrodal insertion) was present in about 28% (14/50) of our patients, with SAM being present in all 14 patients. However, **Patel *et al.*** [6] detected that 87 (72%) patients with maximum LVOT gradient greater than 30 mmHg. 73 (60%) and 87 (72%) patients, respectively, had resting and provoking SAMs.

According to **Noureldin *et al.*** [7], asymmetric septal hypertrophy with sigmoid septal shape is the most prevalent phenotype and is associated with obstruction in about two thirds of HCM patients.

Patients with midventricular obliteration (MVO) due to midventricular concentric hypertrophy were about 18% of the patients in our study group that nearly agree with **Varma & Neema** [8] who said that about 10% of the cases had MVO.

Mid wall myocardial late gadolinium enhancement (LGE) as a predictor of fibrosis was found in about 84% of our patients, however **Axelsson *et al.*** [9] detected LGE in about 46% of patients. their study group included 195 patients, their ages less than 21 years and **Todiere *et al.*** [10] detected LGE in 81.8% of the 55 patients had LGE.

Correlating the LGE to the ECV, our study revealed that studying ECV was more accurate in detecting the diffuse myocardial fibrosis as we detected expanded ECV in about 55% of the cardiac segments and only 30.8% of the cardiac segment showed LGE. This agrees with **Ali *et al.*** [11] study that found LGE in 20.9% of cardiac segments and ECV expansion was seen in 45.1% of cardiac segments. **Kellman *et al.*** [12] Using LGE imaging in the detection of diffuse myocardial disease stated that ECV mapping has been hailed as a potential method. Furthermore, **Taylor *et al.*** [13] and **Lu *et al.*** [14] revealed that the non-invasive T1 mapping approach gave a superior assessment of the degree and severity of myocardial fibrosis than the standard LGE technique, at a level previously obtained with invasive techniques such as cardiac biopsy.

Our study showed the benefit of septal myomectomy in resolution of the SAM, MR and LVOTO. This agrees with **Schaff and Said** [16] who detected improvement of the previously noted SAM, MR and LVOTO after myomectomy in about 90% of the cases.

In our study, we had several limitations like expensive imaging modality (equipment, running and personnel costs), time consuming technique (30 minutes for imaging and about 20 minutes for interpretation). MRI was challenging for senior patients who find it difficult to lie flat or motionless, inappropriate for patients with ferromagnetic devices/implants such as heart valves, and not well tolerated by certain patients who are uncomfortable and claustrophobic (some may require sedation). In some circumstances, the injection of IV contrast is necessary, and there is a chance for motion artefact.

Finally, we recommend that bigger MRI studies to be conducted to expose more HCM patients to the study population. Parametric mapping techniques for tissue characterization and advanced sequences like feature tracking should be performed on larger groups.

CONCLUSION

In conclusion, MRI is an essential technique in diagnosis, risk stratification and follow up in patients with HCM. Advanced techniques, like T1 mapping increased the value of cardiac MRI as a non-invasive technique for tissue characterization.

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