Original Article

Evaluation of Cardiac Magnetic Resonance Imaging in Heart Failure Patients Suspected of Hypertensive Cardiomyopathy in Rajaie Cardiovascular, Medical, and Research Center Between 2015 and 2017

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ABSTRACT

Background: Chronic hypertension is one of the most important threats to public health in that it could cause structural and functional myocardial dysfunction, leading to myocardial hypertrophy and fibrosis. The early detection of cardiac remodeling due to hypertension has curative and preventive advantages; in this regard, cardiac magnetic resonance imaging (CMR) is a helpful modality.

Methods: In this cross-sectional descriptive study. 20 patients with heart failure and no other risk factors, except hypertension, who were referred to the Heart Failure Clinic of Rajaie Cardiovascular, Medical, and research Center between 2015 and 2017 were evaluated with CMR. The left ventricular mass index (LVMI) was normal in 55% and increased in 45% of the patients, and the left ventricular myocardial thickness (LVMT) was normal in 25% and increased in 75%. Twenty-five percent of the patients had normal LVMI and LVMT. Concentric remodeling was observed in 30% of the patients and 45% had concentric hypertrophy. All the patients had an increased LV end-systolic volume index. LV noncompaction without a specific fibrosis pattern was detected in 25% of the patients. Twenty-five percent of the patients had a scattered pattern, possibly due to diffuse interstitial fibrosis.

Conclusions: In this study, most of the patients with hypertensive cardiomyopathy were in the normal LVMI/LVMT or cardiac remodeling group and all of them had an increased LV end-systolic volume index. Both of these findings are suggestive of increased wall stress, which could lead to heart failure with a reduced ejection fraction and its progression. ***(Iranian Heart Journal 2019; 20(2): 40-46)***

KEYWORDS: Hypertension, Cardiomyopathy, CMR (cardiovascular magnetic resonance)

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Hypertension is one of the important problems of public health in that it affects about 50%t of patients who suffer from stroke or ischemic heart disease.[1] Chronically elevated blood pressure could cause structural and functional myocardial disturbances. Left ventricular (LV) hypertrophy, a normal response to increased afterload, is commonly seen in hypertensive patients.[2] Increased hypertension can lead to myocardial interstitial fibrosis, a relevant factor in LV hypertrophy and diastolic dysfunction.[3,4] Hypertensive cardiomyopathy is a structural heart disease and generally has an association with LV concentric hypertrophy as well as with systolic and diastolic dysfunction in patients with chronic hypertension. This disease could appear in the absence of other situations which could cause functional myocardial impairment and hypertrophy. Chronic increased blood pressure could cause myocardial functional and structural disturbances, leading to ischemia, fibrosis, and hypertrophy. Hypertensive cardiomyopathy usually causes relaxation impairment instead of contraction disorders. The patients are usually asymptomatic at rest, but their LV has no compliance for receiving the returned blood; consequently, it cannot eject enough cardiac output, causing exertional intolerance. The important point is that LV hypertrophy itself is a risk factor for morbidity and mortality, so the early detection of ventricular hypertrophy in hypertensive patients is crucial for better treatment. In addition to pathological findings, echocardiography and cardiac magnetic resonance imaging (CMR) are the ideal tools in hypertensive cardiomyopathy.[5] Echocardiographic findings are helpful in the diagnosis of hypertensive cardiomyopathy and in its discrimination from other cardiomyopathies, including asymmetric left septum hypertrophy, which is commonly seen in hypertrophic cardiomyopathy and is highly characteristic of it; however, in LV hypertrophy due to hypertension or hypertensive cardiomyopathy, concentric LV hypertrophy is observed.[6] Other studies have shown that although 13– 31% of patients with hypertensive cardiomyopathy have symmetric hypertrophy,[7,8] 4–47% of these patients have asymmetric septum hypertrophy.[8,9] In this regard, echocardiographic findings themselves are not reliable for a definite diagnosis. Pathological evaluations are useful in the differential diagnosis of hypertensive cardiomyopathy. Invasive biopsy is a helpful tool for the specific diagnosis of hypertensive cardiomyopathy.[10] Pathology is a powerful method; be that as it may, not only is biopsy an invasive procedure with its additional risks but also different studies have reported diverse histological findings.

CMR is capable of revealing data on the heart's 3D anatomy, function, histological characteristics, and valvular diseases, as well as the perfusion of epicardial coronary arteries without ionized radiation. Myocardial fibrosis could be evaluated through gadolinium injection (an extracellular factor that accumulates in the regions of fibrosis and edema). Delayed gadolinium enhancement can detect infarction and fibrosis zones. The pattern and amount of late gadolinium enhancement are effective in the discrimination of hypertrophic cardiomyopathy from hypertensive cardiomyopathy.[11] The evaluation of the LV mass and wall thickness with CMR has higher accuracy than that with echocardiography and is especially useful in detecting minor changes in the ventricular mass over time and after treatment.[12] Additionally, it is useful as an independent prognostic factor in predicting cardiovascular mortality.[13]

In the present study, we aimed to evaluate hypertensive cardiomyopathy in patients via CMR with a view to determining specific fibrosis patterns and assessing findings on the cardiac mass, wall thickness, end-systolic and end-diastolic volumes, and the stroke volume of the LV and the right ventricle (RV).

METHODS

CMR was done in all the patients (Siemens Avanto 1.5 T, Germany). Vertical and horizontal long-axis and sagittal planes of the LV outflow tract were acquired through balanced steady-state free precision cine images (acquired voxel size=1.6×1.3×8.0 mm; 25 phases per cardiac cycle). The short-axis cines were received from the mitral valve annulus to the apex (acquired voxel size=1.6×1.3×8.0 mm; 25 phases per cardiac cycle). The analysis of the images was done by standardized protocols for the measurement of the cardiac function and volumes and the LV mass. The short-axis views were used for the manual measurement of myocardial wall thickness. The assessment of myocardial fibrosis was done via late gadolinium enhancement. The optimization of the inversion time was used for myocardial nulling.

For the analysis of the CMR data, we drew upon the *Normal Values for Cardiovascular Magnetic Resonance in Adults and Children Journal*.

Patients with increased ventricular thickness and normal mass were categorized as concentric remodeling and patients with increased mass and also wall thickness were grouped as concentric hypertrophy.

The LV mass index was defined as the mass indexed to the body surface area, and the LV end-diastolic volume index was defined as the LV end-diastolic volume to the body surface area.

With respect to the left ventricular mass index (LVMI), the patients were divided into 3 groups: 1) normal range (49–85 for men and 41–81 for women), 2) increased range (>85 for men and >81 for women), and 3) decreased range (<49 for men and <41 for women). The results in relation to these definitions are outlined in Table 2.

The left ventricular ejection fraction (LVEF [%]) was categorized as follows: 1) normal value (57–77 for men and women), 2) increased value (>77 for men and women), and 3) decreased value (<57 for men and women).

The left ventricular end-systolic volume index (LVESVI [mL/m^2]) classification was as follows: 1) normal value (14–38 for men and 14–34 for women), 2) increased value (>38 for men and >34 for women), and 3) decreased value (<14 for both sexes).

The left ventricular myocardial thickness (LVMT [mm]) was classified as follows: 1) normal value (5.7–10.9 for men and 4.6–9 for women), 2) increased value (>10.9 for men and >9 for women), and 3) decreased value (<5.7 for men and <4.6 for women).

The left ventricular stroke volume index (LVSVI [mL/m^2]) was classified as follows: 1) normal value (42–66 for men and 38–66 for women), 2) increased value (>66 for both sexes), and 3) decreased value (<42 for men and <38 for women).

The right ventricular ejection fraction (RVEF [%]) classification was as follows: 1) normal value (52–72 for men 51–71 for women), 2) increased value (>72 for men and >71 for women), and 3) decreased value (<52 for men and <51 for women).

The right ventricular end-systolic volume index (RVESVI [mL/m^2]) was classified as follows: 1) normal value (19–59 for men and 12–52 for women), 2) increased value (>59 for men and >52 for women), and 3) decreased value (<19 for men and <12 for women).

The right ventricular end-diastolic volume index (RVEDVI [mL/m^2]) was classified as follows: 1) normal value (61–121 for men and 48–112 for women), 2) increased value (>121 for men and >112 for women), and 3) decreased value (<61 for men and <48 for women).

The right ventricular stroke volume index (RVSVI [mL/m^2]) classification was as follows: 1) normal value (41–73 for men 35–71 for women), 2) increased value (>73 for men and >71 for women), and 3 decreased value (<41 for men and <35 for women).

RESULTS

The demographic characteristics of the study population are summarized in Table 1. The LVMI, LVEDVI, LVEF, LVESVI, LVMT, and LVSVI findings of the patients are outlined in Table 2. Additionally, the RVEF, RVESVI, RVEDVI, and RVSVI data are shown in Table 3.

The CMR evaluation of our patients with hypertensive cardiomyopathy showed LV noncompaction features in 25%. Furthermore, 25% of the patients had nonspecific scattered patterns during the fibrosis assessment.

Twenty-five percent of the patients had normal LVMI and LVMT. Concentric remodeling was observed in 30% of the patients, and 45% had concentric hypertrophy. All the patients had an increased LVESVI.

Table 1. Demographic characteristics of the patients

	Number / median
Age	50
Weight	80
Height	174
Male	n=16(80%)
Female	n=4(20%)

Table 2. Frequency of the normal and abnormal findings in the left side

	LVMI	LVEDVI	LVEF	LVESVI	LVMT	LVSVI
Normal	n=11(55%)	n=7(45%)			n=5(25%)	n=4(20%)
Increased	n=9 (45%)	n=13(65%)		n=20(100%)	n=15(75%)	
Decreased			n=20(100%)			n=16(80%)

Table 3. Frequency of the normal and abnormal findings in the right side

	RVEF	RVESVI	RVEDVI	RVSVI
Normal	n=3(15%)	n=16(80%)	n=12(60%)	n=4(20%)
Increased		n=4(20%)	n=2(10%)	
decreased	n=17(85%)		n=6(30%)	n=16(80%)

DISCUSSION

Chronically elevated blood pressure is one of the most important agents of public health problems that could cause myocardial structural and functional disorders and lead to the fibrosis and hypertrophy of the myocardium. Hypertensive cardiomyopathy is a structural heart disease that occurs in patients with longstanding increased blood pressure in the absence of diseases which could cause cardiac dysfunction and myocardial hypertrophy.

LV hypertrophy and remodeling occurs commonly in hypertensive patients in response to increased afterload.[14] Increased blood pressure leads to myocardial interstitial fibrosis, which is associated with LV hypertrophy and diastolic dysfunction.[15]

LV remodeling and hypertrophy in hypertensive cardiomyopathy patients is an accommodative response to increased afterload in the background of elevated blood pressure which is interpretable by the Laplace Law. Chronically elevated blood pressure leads to increased LV wall stress, an important determinant of myocardial oxygen consumption. In response to increased LV wall stress, LV thickness and mass increase, which results in the normalization of wall stress and eventually concentric hypertrophy. On the other hand, an increased blood volume leads to an increased radius in the cardiac chambers and finally causes eccentric hypertrophy.[14]

Velagaleti[16] on the basis of data from Framingham study suggested that eccentric hypertrophy as well as concentric hypertrophy eventually leads to heart failure with a reduced or preserved EF.

The prevalence of heart failure is increasing worldwide and attempts are underway for its

detection in early stages to prevent the exacerbation of the disease by treatment in early stages.[22]

CMR has a pivotal role in the detection and follow-up of these patients. It is a gold-standard method for measuring the EF, mass, and volume of the LV or the RV. CMR using Gadolinium could be effective in describing the myocardial texture, the existence of fibrosis, viability assessment, and also the diagnosis of diverse cardiomyopathies.[22]

Elevated blood pressure can be a substrate for cardiac remodeling, the early detection of which is of utmost importance for the commencement of treatment and the prevention of heart failure progression, and CMR in this regard is helpful.[22]

In this cross-sectional descriptive study, 20 patients with EF<40% who had no other risk factors for heart failure except hypertension were evaluated by CMR. Sixteen (80%) patients were male and 4 (20%) were female, and the median age of the study population was 50 years.

In their study, Ganau and colleagues[17] reported that the LVMI and LV thickness were normal in about 52% of their hypertensive patients. In addition, 13 patients had increased wall thickness with normal mass (concentric remodeling), 27 had increased mass with normal wall thickness (eccentric hypertrophy), and 8% had increased mass and wall thickness (concentric hypertrophy).

Cuspidi and his colleagues[18] in their study found a low prevalence of concentric hypertrophy in comparison with the eccentric type in hypertensive patients, but the concentric type could predispose to adverse complications as well as eccentric hypertrophy.

CMR findings in our study showed normal LVMI in 55% of the patients and increased values in 45%. The LVMI and LV thickness were normal in 25% of the patients, 13% had increased wall thickness with normal mass (concentric remodeling), and 45% had increased mass in addition to wall thickness (concentric hypertrophy). According to these findings, the majority of the patients were in the cardiac remodeling and normal group and all the patients had increased LVESVI. Probably, one of the causes of cardiomyopathy in these patients is increased wall stress, leading to cardiomyopathy and its progression.

The prevalence of LV hypertrophy is different according to the various stages of hypertension and varies between 20% in mild hypertension and 100% in severe hypertension.[19] In a study conducted by Cuspidi and his colleagues[17] based on echocardiographic data on 37 700 individuals, the prevalence of LV hypertrophy was 19–48% in patients with untreated hypertension and 58–77% in high-risk hypertensive ones.

The LVMT was normal in 25% and increased in 75% in our study. Wall thickness in the majority of the patients was in the normal or mildly increased range; therefore, this mild hypertrophy may have been associated with the increased LVESVI in all the patients, leading to elevated wall stress and predisposition to a reduction in the EF.

The late gadolinium enhancement pattern could be a new method for the risk stratification of patients with hypertensive cardiomyopathy.

Given the relationship between fibrosis and diastolic dysfunction, it could be useful in the diagnosis of patients with hypertensive cardiomyopathy who are at risk of diastolic heart failure.[19]

Fifty percent of patients with elevated blood pressure had late gadolinium enhancement without specific patterns and most of them had focal non-subendocardial distributions in a study by Andersen, Rudolph and their colleagues.[20,21] The assessment of fibrosis in our study showed a scattered pattern in 25% of the patients unlike specific patterns in other cardiomyopathies. LV noncompaction was seen in 25% of the patients: they may have had this disorder from the outset and its association with hypertension caused the progression of heart failure or it might have been the longstanding

hypertension which predisposed them to LV noncompaction.

Precise control of hypertension is recommended, especially in patients with increased wall stress based on myocardial thickness, diameter, and volume in initial imaging and also in the presence of systemic hypertension. These patients should be visited at shorter intervals with tight control of their blood pressure.

It is recommended that evaluations of hypertensive patients be in a prospective manner to define which mechanical characteristics of their heart lead to the reduced EF with a view to defining a better approach to diagnosis and treatment in these patients.

CONCLUSIONS

In our hypertensive patients with a reduced EF, increased ventricular wall thickness and mass led to a concentric hypertrophy pattern in the majority of the patients. Increased LVESVI caused elevated wall stress, injury to myocytes, and finally a reduced EF.

Abbreviations

CMR: Cardiovascular Magnetic Resonance; LV: Left Ventricle; RV: Right Ventricle; BSA: Body Surface Area; SV: Stroke Volume; EF: Ejection Fraction; CI: Cardiac Index; LVMT: Left Ventricular Myocardial Thickness; LVMI: Left ventricular Mass Index; LVESVI: Left ventricular End Systolic Volume Index; LVEDVI: Left Ventricular End Diastolic Volume Index; LVSVI: Left Ventricular Stroke Volume Index; RVEDVI: Right Ventricular End Diastolic Volume Index; RVESVI: Right Ventricular End Systolic Volume Index; RVSVI: Right Ventricular Stroke Volume Index.

Limitations

This case-series evaluation had a small sample volume. In addition, ethical issues regarding CMR performance precluded a comparison of the results with those in normal subjects.

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