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Utility of Delayed Enhancement Cardiac MRI in Evaluation of Restrictive Cardiomyopathy

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Abstract

Background: Restrictive cardiomyopathy (RCM) is a heterogeneous group of myocardial diseases characterized by non-dilated rigid ventricle, leading to severe diastolic dysfunction and restrictive filling. RCM is the least common type of the cardiomyopathies. Itexits in the pediatric and adult age groups and can be either primary or secondary to systematic disorders. The two types show different lines of management and different prognosis.

Aim of Study: This study aimed to evaluate the role of delayed enhancement cardiac MR in differentiating the types of RCM and in reaching the diagnosis of secondary RCM.

Patients and Methods: A total number of 20 cases with echocardiography showing evidence of diastolic dysfunction and elevated filling pressure were enrolled for delayed-enhancement cardiac MRI examination to confirm the diagnosis of RCM and establish the etiological diagnosis in secondary cases.

Results: CMR allowed accurate assessment of the atrial and ventricular volumes, the ventricular function, and the atrio-ventricular valve regurgitation. Fourteen cases showed LGE in different patterns denoting secondary etiology, the specific cause could be reached in most of cases, while the remaining 6 cases were primary.

Conclusion: CMR is an efficient tool confirming the diagnosis of RCM.LGE can differentiate primary from secondary RCM, it can direct the diagnosis to specific subtypes of RCM, depending on the pattern of scar formation and hence determine the prognosis and guide the management.

Key Words: *Cardiac MRI – Delayed enhancement – Restrictive cardiomyopathy – Myocardial infiltration.*

Introduction

ACCORDING to a statement of the European Society of Cardiology (ESC) working group on myocardial and pericardial diseases, cardiomyopathy is defined as a myocardial disorder in which there is abnormality in the structure and function of the myocytes, in the absence of coronary artery disease, hypertension, valvular disease, and congenital heart disease that could justify this abnormality [1].

Restrictive cardiomyopathy (RCM) is a heterogeneous group of myocardial diseases that usually result from increased myocardial stiffness causing impaired ventricular filling by variety of causes. They vary according to pathogenesis, clinical presentations, diagnostic criteria, treatment and prognosis [2]. Biventricular chamber size and systolic function are usually normal or near-normal until later stages of the disease. Affecting either or both ventricles, RCM may cause signs or symptoms of left or right heart failure. Arrhythmias and conduction disturbances are frequently encountered [3].

Studies showed up to 10%-15% of patients in heart failure with preserved ejection fraction were caused by RCM [4]. In a pediatric single center

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List of Abbreviations:

AHA	: American Heart association.
CMR	: Cardiac magnetic resonance.
CP	: Constrictive pericarditis.
DCM	: Dilated cardiomyopathy.
EDV	: End diastolic volume.
EF	: Ejection fraction.
EMF	: Endomyocardial fibrosis.
ESC	: European Society of Cardiology.
ESV	: End systolic volume.
FOV	: Field of view.
GFR	: Glomerular filtration rate.
HCM	: Hypertrophic cardiomyopathy.
IR	: Inversion recovery.
IR-b-TFE	: Inversion recovery balanced turbo field echo.
LGE	: Late gadolinium enhancement.
LV	: Left ventricle.
RCM	: Restrictive cardiomyopathy.
RV	: Right ventricle.
SSFP	: Steady state free precession.
STIR	: Short tau inversion recovery.
SV	: Stroke Volume.
TE	: Time of echo.
TR	: Time of repetition.

study, the morbidity of RCM in children accounted for 4.8% of all cardiomyopathy. High incidence of sudden death was observed; 2-year survival rate was 50%, and the 5-year survival rate dropped to only 25% [5].

Restrictive cardiomyopathy (RCM) can be idiopathic, familial, or secondary to systematic disorders. Marked increase in left and/or right ventricular filling pressures causes symptoms and signs of congestive heart failure. Echocardiography and cardiac magnetic resonance (CMR) play a major role in diagnosis. Echocardiography reveals normal or hypertrophied ventricles, preserved systolic function, marked bi-atrial enlargement, and impaired diastolic function, often with restrictive filling pattern. CMR offers higher spatial resolution than echocardiography, can provide detailed information about anatomic structures, perfusion, ventricular function, and tissue characterization. CMR with late gadolinium enhancement (LGE) can direct the diagnosis to specific subtypes of RCM, depending on the pattern of scar formation [6].

The main role of cardiac CT in cases with RCM is to excluded the deferential diagnosis of constrictive pericarditis,as it gives valuable information about the pericardial thickness and calcifications [7].Histo-pathological examination can be valuable in setting a definite diagnosis when noninvasive studies have failed, although there are risksrelated to the endo-myocardial biopsy [8].

Aim of the work:

This study aimed to evaluate the role of delayed enhancement CMR in differentiating the types of RCM and in reaching the diagnosis of secondary RCM.

Patients and Methods

The study is observational cross sectional study. A total number of 20 cases, referred from the Cardiology Department of the Cairo University Hospitals between June 2018 and Dec. 2019, nine of them (45%) were males. The patient's ages ranged from 3 years to 53 years with a median age of 23.45 years. Eleven patients (55% of the study population) were in the pediatric age group (below 18 years). Patients presented with overt symptoms and signs of heart failure, multi-organ presentation or do not fit the typical risk profile for heart failure with preserved ejection fraction. Their echocardiography shows evidence of diastolic dysfunction and elevated filling pressure. They were enrolled for cardiac MRI examination (CMR) toconfirm the diagnosis and establish etiological diagnosis. Patients with contra-indications to MR examination (including patients with clipsed cerebral aneurysms or other metallic devices) and those with contra-indication to contrast administration (GFR less than 60ml/min) were excluded.

All procedures performed in this study were in accordance with the ethical standards, approved by The Ethics Committee of our University Hospitals, and complied with the Declaration of Helsinki 1975 as revised in 2000 and its latter amendments. Written informed consent was obtained from all individual participants (or their parents) included in this study.

Patient preparation and set up:

No special instructions were required prior to the examination. Medications are not to be discontinued. Before the examination; the heart rate and rhythm were evaluated. All steps of the study were explained in details for each patient (including the breath-holding instructions).

A Philips Achiva, 1.5 Tesla (Netherland) superconducting magnet was employed. All patients were examined in the supine position, head first. Head phones supplied with the MRI machine are used to reduce repetitive gradient noise and in the same time allow the patient to hear the breath hold instructions.

Four carbon fibers ECG pads were placed on the anterior chest wall. The QRS complex was checked on the MRI monitor, adjustments of the site of the leads was done accordingly.

The SENSE (sensitivity encoding) cardiac coil (6 element phased array coil) was used. The respiratory sensor was placed over the maximum area of respiratory movement under the coil.

In patients younger than five years old, we used either general anesthesia or chloral hydrate.

Cardiac MR Examination:

All patients underwent a standard MR examination that included the following steps:

1- Scout images: Acquired in orthogonal planes for planning of the cardiaclong and short axes.
2- Functional cine images: Acquired using ECG gated, steady state free precession sequences in short axis plane, axial planeand four chambers planes. Slices were obtained during repeated breath-holds with the following parameters:
   - TR/TE: 4.4/2.5.
   - FOV: Ranging between 250-350mm according the patient's age.

- Phases: 25.
- NSA: 1-2 according to the patient's comply with the breath holding.
- Slice thickness: 6-8mm and slice gab: 0mm.
- Matrix: 128x128.

3- Standard delayed gadolinium enhancement imaging:

Performed after using look locker to determine the best time of delay by using inversion recovery balanced turbo field echo (IR-b-TFE), starting 10min after intravenous infusion of gadolinium chelate contrast material (0.28mmol/kg). The images were acquired in the short axis plane and at least one of the long axis planes with the following parameters:

- TR/TE: 3.8/1.86.
- FOV: Ranging between 250-350mm according the patient's age.
- TI: 250-450.
- NSA: 1-2 according to the patient's comply with the breath holding.
- Slice thickness: 6-8mm and slice gab: 0mm
- Matrix: 128x128.

Image analysis:

Images were transferred to a workstation (Brilliance 170 P) equipped with dedicated cardiac software for further analysis:

1- Assessment of the bi-atrial and bi-ventricular size:

Global functional parameters were derived from cine MRI, with the aid of commercially available software. The endocardial borders of both ventricles were traced manually from the short axis images during systole and diastole. LV and RV end-diastolic volume (EDV) and end-systolic volume (ESV) were calculated on the basis of Simpson's rule. Subsequently, stroke volume (SV) and ejection fraction (EF) was calculated using EDV and ESV values. The endocardial borders of both atria were traced manually from the four chamber/axial images at end systole before mitral valve opening. The maximum atrial volume was calculated.

2- Assessment of delayed enhancement pattern:

Visual assessment; the myocardial enhancement was defined as an area of increased signal intensity relative to the normal myocardium in the late-enhancement images.

3- Assessment of pericardial thickness:

Normal pericardial thickness is less than 2mm, pericardial thickness of 4mm or more indicates abnormal thickening and is highly suggestive of constrictive pericarditis.

4- Septal bounce:

Abrupt septal movement ('notch' or 'bounce') in early diastole toward left ventricle, is suggestive of constrictive pericarditis.

5- Valve regurgitation:

By visual assessment of the tricuspid and mitral valves.

Statistical methodology:

We used SPSS version 16 for statistical analysis. Due to small number of included patients, the data is abnormally distributed. Values were described in terms of median (range) for numerical data and number (percentage) for categorical one.

Results

Twenty patients were included in this study, nine of them (45%) were males. The patient's ages ranged from 3 years to 53 years with a median age of 23.45 years. Eleven patients (55% of the study population) were in the pediatric age group (below 18 years).

Only one patient had LV dilatation and two patients had RV dilatation. Nineteen patient has LV EF 40% and Eighteen patients had RV EF >40%. Regarding the atrial volumes; 12 patients (60%) had bi-atrial enlargement while the remaining 8 patients (40%) had isolated right atrial enlargement.

Regarding the late gadolinium enhancement; fourteen patients (70%) showed late enhancement in different patterns as follow; half the patients showed mid wall patchy enhancement, five showed sub-endocardial enhancement (either patchy or diffuse) and two patients showed trans-mural enhancement, not limited to a vascular territory.

Two patients showed intra-cardiac thrombi. Six patients (30%) showed apical obliteration.

Thirteen patients (65%) had valvular regurgitation; two patients had mitral regurgitation, two patients had tricuspid regurgitation and nine patientshad combinedmitral and tricuspid regurgitation.

Two patients (10%) showed septal bounce. All patients showed pericardial thickness 2mm or less.

Discussion

Echocardiography is the first imaging modality for the assessment of patients with dyspnea and/or heart failure. RCM are usually characterized by normal or small LV cavity size (<40mL/m ^{2}) with preserved LV ejection fraction, bi-atrial enlargement, diastolic dysfunction, and elevated filling pressure [9].

Cardiac magnetic resonance (CMR) is a highly specific and non-invasive technique that uses static (black blood) images, cine and contrast enhanced imaging as well as parametric mapping for the assessment of RCM. CMR help to confirm the diagnosis of RCM and establish its etiologic diagnosis [10].

According to our results, CMR had shown excellent capability for assessment of the ventricular function, the atrial and ventricular volumes, associated valvular regurgitation, and detection of intra-cardiac thrombi.

Regarding the ventricular function, in our study 18 patients had RV EF >40% and 19 patients had EF 40%. According to the study done by C heng et al., among RCM patients LVEF was 46.6%±11.8 and RVEF was 45.8%±11.9 [11].

Regarding the atrial and ventricular volumes and valve lesions; our study showed that all patients had atrial enlargement (twelve patients showed bi-atrial enlargement and eight patients showed isolated right atrial enlargement), most patient showed normal ventricular volumes (seventeen patients showed normal left and right ventricular volumes), Thirteen patients showed valvar regurgitation; nine of them showed combined mitral and tricuspid regurgitation.

Shihua et al., studies 116 patient with RCM and found thatleft and right atrial size, and right ventricular diastolic diameter were significantly larger in patient with RCM than in normal subjects (p<0.05). However, there were no statistical differences between the two groups in left ventricular diastolic diameter. Mitral valve assessment showed mild mitral regurgitation (43%), moderate mitral regurgitation (21 %), mild tricuspid regurgitation (28%) and severe tricuspid regurgitation (40%) in patients with RCM [12].

CMR showed to be a very efficient tool in the differentiating between constrictive pericarditis and restrictive cardiomyopathy. In our study constrictive pericarditis was excluded based on the normal pericardial thickness in all cases. Although 2 cases showed septal bounce (defined as paradoxical bouncing motion of the interventricular septum initially directed towards and then away from the left ventricle during early diastole), this could be attributed to pulmonary hypertension. Both cases showed late gadolinium enhancement.

A study done by Cheng et al., on 23 patients with surgically documented CP, 22 patients with RCM and 25 normal controls. According to the study the maximal pericardial thickness in CP patients (6.9±2.6mm, range 4-12mm) was significantly larger than in normal subjects (1.5±0.4mm, range 0.9-2.7mm, p<0.001) and RCM patients (2.0±0.7mm, range 1.0-3.4mm, p<0.001). The same study found that LGE was present in 7 of 22 RCM patients (31.8%) and absent in all CP patients and normal subjects [11].

Walker et al., stated that septal bounce is most commonly associated with constrictive pericarditis but can also be seen in cardiac tamponade and pulmonary hypertension. In the broadest sense, a septal bounce-like motion may also be seen in the setting of elevated right heart pressures, right ventricular pacing, and left bundle branch block. [13].

Although delayed contrast enhanced cardiac magnetic resonance imaging has traditionally been used to evaluate ischemic disease and myocardial viability, it is increasingly being used in the evaluation of non-ischemic cardiomyopathies. Unlike myocardial infarction, which demonstrates subendocardial or transmural delayed contrast enhancement in a vascular distribution, non-ischemic cardiomyopathies demonstrate enhancement that is not limited to a vascular territory. In combination with other cardiac MR imaging features, the location (subendocardial, transmural, subepicardial, or mesocardial) and pattern (patchy or diffuse) of abnormal delayed myocardial enhancement allow differentiation between ischemic and non-ischemic cardiomyopathies and, in cases of non-ischemic cardiomyopathy, narrowing of the differential diagnosis [14].

The American Heart Association (AHA) classified the RCM into a 'primary' in which the heart is the sole or predominantly involved organ and 'secondary' in which the myocardial dysfunction is part of a systemic disorder [15]. In our study;six patients (30%) showed no late gadolinium myocardial enhancement; denoting primary etiology (Fig. 1) while the remaining fourteen patients (70%) showed different patterns of LGE; keeping with secondary cause.

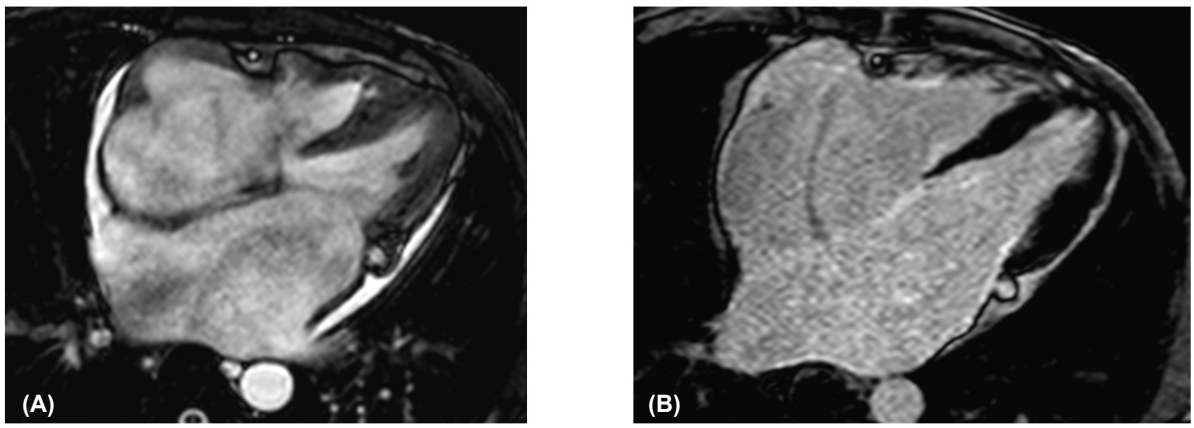


Fig. (1): CMR confirming the diagnosis of RCM with no delayed enhancement suggestive of primary etiology in a 29 year old male patient presented with shortness of breath. (A): Four chamber plane, SSFP showing bi-atrial dilatation. (B): Four chamber plane, IR after Gd-DTPA injection showing no significant late myocardial enhancement. Mild pericardial effusion is also noted.

Two patients showed failed myocardial nulling in TI scout images with diffuse sub-endocardial delayed enhancement, diagnosed as amyloidosis (Fig. 2). In cardiac amyloidosis, CMR shows a characteristic pattern of global subendocardial late enhancement coupled with abnormal myocardial and blood-pool gadolinium kinetics. The findings agree with the transmural histological distribution of amyloid protein and the cardiac amyloid load and may prove to have value in diagnosis and treatment follow-up **[16]**. The sensitivity and specificity of any nulling abnormality for the identification of cardiac amyloidosis was 100% and 60% respectively in a study done by Pandey et al. **[17]** Vogelsberg H et al., reported that patients with biopsy-proven cardiac amyloidosis had a distinct pattern of LGE, which was distributed over the entire subendocardial circumference, extending in various degrees into the neighboring myocardium. They concluded that using this pattern as a diagnostic criterion, the sensitivity of CMR for diagnosing cardiac amyloidosis was 80%, yielding a specificity of 94%. The positive predictive value was 92%, and the negative predictive value was 85% **[18]**.

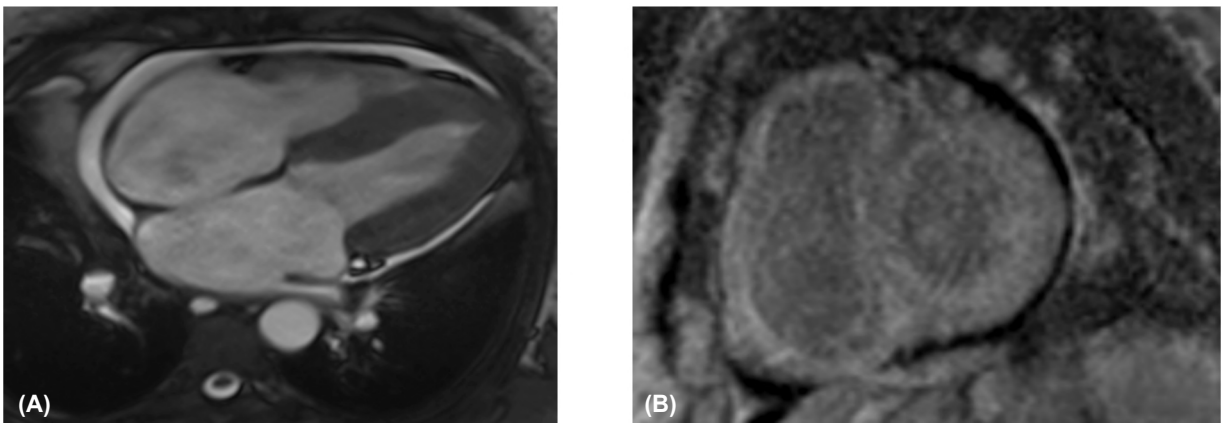


Fig. (2): CMR confirming the diagnosis of RCM with delayed enhancement pattern suggestive of myocardial amyloidosis in a 53 year old female patient presented with shortness of breath. (A): Four chamber plane, SSFP showing bi-atrial dilatation. There was suboptimal nulling of the myocardial signal. (B): Short axis plane, IR after Gd-DTPA injection showing diffuse subendocardial delayed hyper-enactment, evident also in the RV.

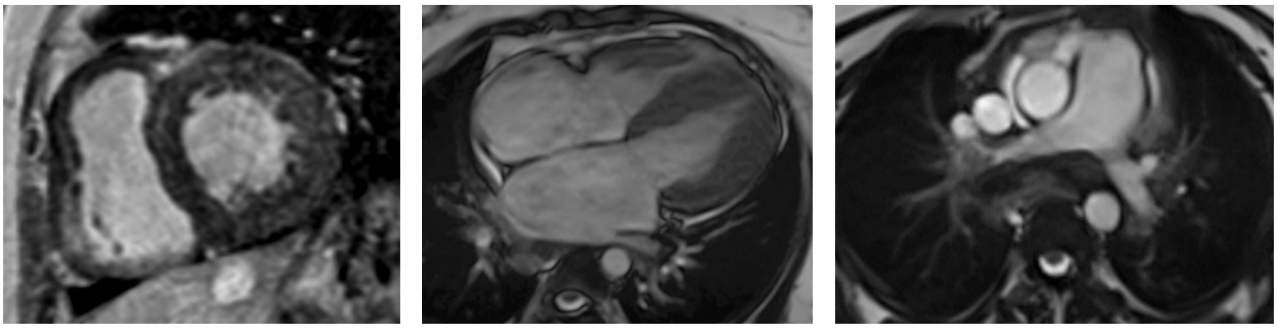
Cardiac sarcoidosis can presents byrestrictive cardiomyopathy, it is usually seen in the presence of other findings, such as mediastinal lymphadenopathy or pulmonary parenchymal disease. Delayed contrast enhancement can be seen in areas of granuloma and scar formation and is frequently patchy and nodular **[19]**.

In our study, onepatient showed patchy midwall late gadolinium enhancement at the mid and basal segments of the anterior wall of the LVas well as superior and inferior insertion points of RV. Multiple mediastinal and bilateral hilar lymph nodes were noted. Complementary CT was done showing beaded appearance of the pulmonary

fissures. The case was diagnosed as sarcoidosis (Fig. 3).

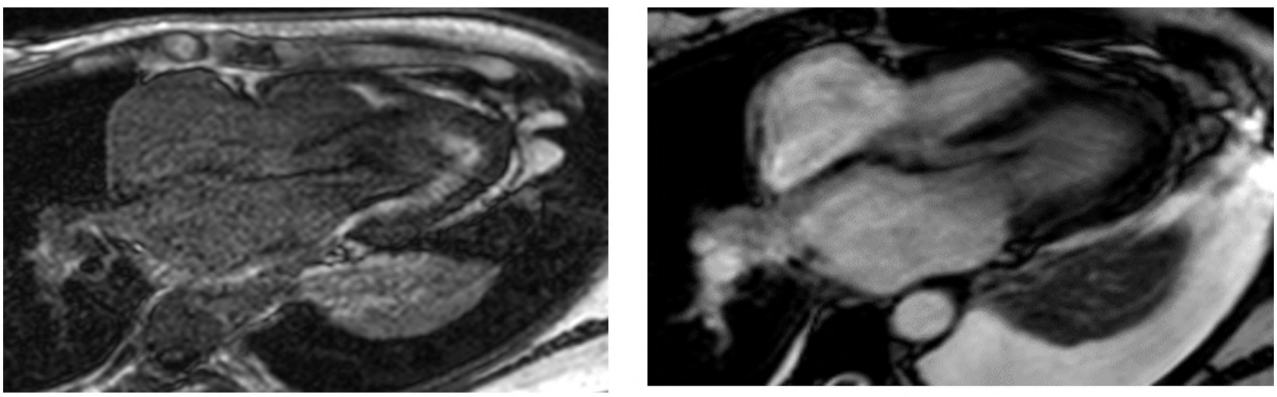
In our study sixpatients showed apical obliteration together with different patterns of delayed myocardial enhancement (patchy/diffuse subendocardial or transmural enhancement), two of them had clinical history of hyper-eosinophilia. In these two cases; the diagnostic possibility of Loffler's endocarditis was suggested (Fig. 4).

According to Kleinfeldt et al., a definite diagnosis of endomyocardial fibrosis is made in the presence of two major criteria or one major criterion associated with two minor criteria (Table 1). Apical obliteration is one of the major criteria for diagnosis of endomyocardial fibrosis **[20]** (Fig. 5).



(A) (B) (C)

Fig. (3): CMR confirming the diagnosis of RCM with delayed enhancement pattern suggestive of sarcoidosis in an 18 year old female patient presented with shortness of breath and systemic congestive symptoms. (A): Four chamber plane, SSFP showing bi-atrial dilatation and biventricular myocardial hypertrophy. (B): Short axis plane, IR after Gd-DTPA injection showing left ventricular mid wall anterior, antero-septal and antero-lateral spotty enhancement. (C): Axial images, SSFP, showing bilateral hilar lymph node enlargement.



(A) (B)

Fig. (4): CMR confirming the diagnosis of RCM in a 39 year old female patient presented with shortness of breath and her Labs shows hyperesenophilia. (A): Four chamber plane, SSFP showing bi-atrial dilatation. (B) IR after Gd-DTPA injection showing patchy subendocardial enhancement involving apical and subvalvular segments of the LV and patchy subendocardial enhancement involving mainly the apical segment of the RV. Bilateral pleural effusion was also noted, minimal on the right side and moderate on the left side with underlying partial relaxation collapse of the left lung. The diagnostic possibility of Loffler's endocarditis was suggested.

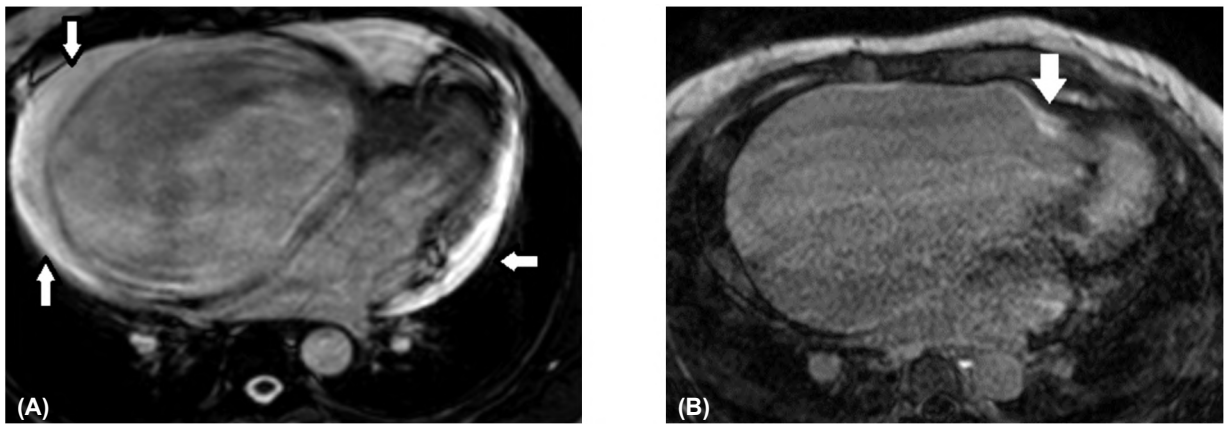


Fig. (5): CMR confirming the diagnosis of RCM in a 49 year old female patient presented with shortness of breath and symptoms of congestion. (A): Four chamber plane, SSFP showing marked right atrial dilatationand RV apical obliteration. (B): Four chambers IR after Gd-DTPA showingsubendocardial enhancement on the RV free wall. Moderate pericardial effusion was also noted. Secondary cause of the RCM is confirmed, endo-myocardial fibrosis is suggested.

Table (1): Criteria for diagnosis and assessment of the severity of endomyocardial fibrosis.

Criterion	Score
Major criteria:	
- Endomyocardial plaques [2 mm in thickness]	2
- Thin (B 1 mm) endomyocardial patches affecting more than one ventricular wall	3
- Obliteration of the right or left ventricular apex	4
- Thrombi or spontaneous contrast without severe ventricular dysfunction	4
- Retraction of the right ventricular apex	4
- Atrioventricular valve dysfunction due to adhesion of the valvular apparatus to the ventricular wall	1-4**a**
Minor criteria:	
- Thin endomyocardial patches localized to one ventricular wall	1
- Restrictive flow pattern across mitral or tricuspid valves	2
- Pulmonary valve diastolic opening	2
- Diffuse thickening of the anterior mitral leaflet	1
- Enlarged atrium with normal-sized ventricle	2
- M-movement of septum and flat posterior wall**b**	1
- Enhanced density of the moderator or other bands	1

- A definite diagnosis of endomyocardial fibrosis is made in the presence of two major criteria or one major criterion associated with two minor criteria. A total score less than 8 indicates mild endomyocardial fibrosis, 8-15 moderate disease and more than 15 severe disease.

a The score is assigned according to the severity of at rioventricular regurgitation.

b M-movement of the interventricular septum refers to a pattern of movement observed on M-mode echocardiography that is thought to be due to obliteration or restriction of the left ventricular apex combined with mitral regurgitation **[18]**.

Conclusion:

CMR is an efficient tool to confirm the diagnosis of RCM, assessing the ventricular function and volume, the atrial volumes, the associated valvular regurgitation and can differentiate it from constrictive pericarditis.

LGE can differentiate primary from secondary RCM, it also can direct the diagnosis to a specific subtype of RCM, depending on the pattern of scar formation and hence determine the prognosis and guide the management.

Limitations:

1- The study is limited by the small number of patients. This could be attributed to the relative rarity of the RCM, as it is the least common type of cardiomyopathy.

2- Lack of histo-pathological correlation in the cases with secondary etiology.

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دور الرنين المغناطيسى باستخدام الاصطباغ المتأخر فى تقييم اعتلال عضلة القلب التقييدى

اعتلال عضلة القلب التقييدى هو اضطراب ينتج عن زيادة تصلب عضلة القلب بما يؤدى إلى اختلال ملء البطينين. عادة ما يكون حجم غرفة البطينين ووظيفة الانقباض طبيعياً أو شبه طبيعى حتى المراحل المتأخرة من المرض. وبالتأثير على إحدى البطينين أو كليهما، قد تظهر علامات أو أعراض فشل القلب الأيسر أو الأيمن. وفى حالة وجود مؤشر عالٍ من الشك يصبح من الضرورى تشخيص هذا المرض فى مرحلة مبكرة حتى يمكن بدء العلاج الفعال.

يعتبر الايكو طريقة أساسية للتشخيص. فى حالة اعتلال عضلة القلب التقييدى الغير الارتشاحى، يظهر الايكو البطين الأيسر غير متوسع وغير متضخم. كما يكون هناك عادة توسع ملحوظ من الأذينين. فى اعتلال عضلة القلب التقييدى الارتشاحى، مثل داء النشوانى وأمراض تخزين الجليكوجين، فإن النتيجة الأولية هى زيادة سماكة جدار البطين الأيسر مع حجم تجويف عادى أو منخفض.

إن الايكو غير قادر على تشخيص أنواع فرعية محددة من هذا المرض بسبب توصيفه النسيجى الضعيف وتقييمه المحدود للبطين الأيمن. يمكن للرنين المغناطيسى القلبى مع دقة أعلى والتصوير بعد الصبغة توفير معلومات تفصيلية عن التراكيب التشريحية، ووظيفة البطين، وتوصيف الأنسجة. يمكن أن يؤدى التصوير بعد الصبغة اعتماداً على نمط تشكيل الندبة إلى توجيه التشخيص إلى أنواع فرعية محددة من المرض. أخيراً يمكن أن قياس سمك التامور بد قة وتصوير التهاب التامور، مما يساعد فى تشخيص التهاب التامور التضيقى.

الرنين المغناطيسى على القلب هو أداة فعالة جداً فى التمييز بين التهاب التامور التضيقى واعتلال عضلة القلب التقييدى، وتقييم وظيفة البطين، وحجم البطينين والأذينن، ووجود جلطة داخل القلب. بالإضافة إلى ذلك يساعد تصوير ما بعد الصبغة لتحديد مسببات اعتلال عضلة القلب المقيدة الثانوية.

لذلك، قد يساعد الرنين المغناطيسى على القلب فى توجيه العلاج للمرضى الذين يعانون من اعتلال عضلة القلب التقييدى.