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Review Article

Role of cardiac MRI in nonischemic cardiomyopathies

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ARTICLE INFO

Article history:
Received 17 August 2015
Accepted 29 September 2015
Available online 18 January 2016

Keywords:
Cardiac magnetic resonance
Nonischemic cardiomyopathy
Late gadolinium enhancement

ABSTRACT

Cardiac magnetic resonance (CMR) with its higher spatial resolution is considered the gold standard for evaluating ventricular mass, volumes, and ejection fraction. CMR can be used for accurate diagnosis of several conditions, especially cardiomyopathies. The purpose of this article is to review the utility of CMR in the diagnosis and management of nonischemic cardiomyopathies. We have reviewed both common and rare types of nonischemic cardiomyopathies in detail and elaborated on the specific CMR findings in each. We believe that CMR is an invaluable tool, not only in differentiating nonischemic from ischemic cardiomyopathy, but also in aiding the accurate diagnosis and management of the subtype of nonischemic cardiomyopathy. CMR should routinely be integrated in the diagnostic workup of various cardiomyopathies.

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1. Introduction

Cardiac magnetic resonance (CMR) is considered to be the gold standard for evaluating ventricular mass, volumes, and ejection fraction. CMR has an advantage in that it is not limited by poor acoustic windows, which can often limit echocardiographic studies, thereby enabling diagnosis of pathologies, which are otherwise not readily recognized by echocardiography. CMR can be used for assessing many pathologies, including aortic disease, coronary artery disease (CAD), cardiomyopathies, pericardial disease, and congenital heart disease.[1] The purpose of this article is to review the utility of CMR in diagnosis and management of nonischemic cardiomyopathies.

2. Classification of cardiomyopathies

The current AHA classification of cardiomyopathies divides them into primary, which affect only the heart, and secondary, a much larger group in which myocardial involvement is part of a systemic (multiorgan) generalized disease process. Further subclassification is as follows[2]:

1. Primary
   a. Genetic
      i. Hypertrophic (obstructive) cardiomyopathy (HCM or HOCM)
      ii. Arrhythmogenic right ventricular cardiomyopathy/Dysplasia (ARVC/D)

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http://dx.doi.org/10.1016/j.ihj.2015.09.032
0019-4832/Published by Elsevier B.V. on behalf of Cardiological Society of India.

iii. Isolated ventricular noncompaction
iv. Glycogen storage disorders
v. Conduction defects
vi. Mitochondrial myopathies
vii. Ion channel disorders (e.g. Brugada's, Long QT)

b. Mixed
i. Dilated cardiomyopathy
ii. Restrictive cardiomyopathy

c. Acquired
i. Inflammatory (myocarditis)
ii. Stress provoked (Takotsubo)
iii. Peripartum
iv. Tachycardia induce

2. Secondary
a. Infiltrative, e.g. amyloidosis
b. Storage, e.g. hemochromatosis, Fabry's disease
c. Toxicity, e.g. alcohol, cocaine
d. Endomyocardial, e.g. endomyocardial fibroelastosis, Loeffler's syndrome
e. Inflammatory, e.g. sarcoidosis
f. Endocrine, e.g. DM, thyroid disorders, acromegaly, pheochromocytoma
g. Cardiofacial
h. Neuromuscular/neurological, e.g. Duchenne-Becker, Friedrich's ataxia
i. Nutritional deficiencies
j. Autoimmune/collagen e.g. RA, SLE
k. Electrolyte imbalance
l. Consequence of cancer therapy e.g. anthracyclines, such as doxorubicin, or alkylating agents, such as cisplatin and cyclophosphamide

3. Differentiating nonischemic from ischemic cardiomyopathy

While attempting to diagnose the etiology of cardiomyopathy, it is important to exclude CAD as the etiology, given the differences in management. CMR technique of late gadolinium enhancement (LGE) becomes valuable in establishing the proper diagnosis. Gadolinium chelates are extracellular contrast agents that cannot cross myocyte cell membranes.[3] Normal myocardium is densely packed with viable myocytes that do not permit entrance of gadolinium into the cell; thus there is little gadolinium enhancement of normal myocardium. However, in the setting of an acute myocardial infarction, myocardial cell membrane rupture will allow gadolinium to freely diffuse into the cell[3] resulting in gadolinium hyperenhancement. The necrosis begins in the subendocardium and grows toward the epicardial area near the occluded artery.[3] In chronic myocardial infarction, myocytes get replaced with collagenous scar tissue in the subendocardial region leading to increased gadolinium concentration and hyperenhancement in the subendocardium.[3] Thus, ischemic cardiomyopathy tends to cause LGE in the subendocardium or transmurally and follows a vascular distribution, which lies in stark contrast to nonischemic cardiomyopathy, which generally does not correspond to any particular coronary artery distribution and is often located in the midwall or epicardial regions.[4] Therefore, the pattern of LGE can be used to differentiate between cardiomyopathies of ischemic and nonischemic etiologies.

4. Hypertrophic cardiomyopathy

Transthoracic echocardiography (TTE) is considered the first line imaging modality for patients with hypertrophic cardiomyopathy (HCM). However, CMR can help in diagnosing variant types of HCM, including apical (Yamaguchi's) and lateral wall hypertrophies, otherwise not detected by TTE.[5] CMR also has high accuracy in wall thickness measurements, which has important prognostic value. Cine-CMR with flow velocity encoding can be utilized to evaluate the flow dynamics and dynamic obstruction of the LV outflow tract in such patients. Several patterns of LGE have been described in HCM, which demonstrate areas of fibrosis.[6] LGE has been associated with increased risk of re-entrant tachycardias, ventricular tachycardia, and sudden cardiac death.[6]

5. ARVC/D

ARVC/D is an inherited condition characterized pathologically by fibrofatty replacement of the ventricular wall, primarily RV, and clinically by life-threatening ventricular arrhythmias, heart failure, and sudden cardiac death.[7] The 2010 revised task force criteria is used for diagnosis of ARVC,[8] which includes parameters for regional RV dysfunction, RV volume, and RV global dysfunction.

6. Left ventricular noncompaction (LVNC)

LVNC, also known as left ventricular hypertrabeculation, is a congenital morphological disorder due to an arrest in the normal process of myocardial compaction during development, resulting in persistent prominent ventricular trabeculations and deep intertrabecular recesses. Diagnosis is based on clinical and morphological criteria. Diagnosis is usually established by TTE, but when imaging is suboptimal, CMR can be utilized. CMR criteria for diagnosis of LVNC include noncompacted to compacted myocardial thickness ratio of >2.3 (sensitivity, specificity, and positive and negative predictions of 86%, 99%, 75%, and 99%, respectively[9]), and trabeculated LV mass >20 percent of global LV mass (sensitivity of 94% and specificity of 94%).

7. Myocarditis

Myocarditis is an inflammation of the myocardium that can be caused by a variety of etiologies, commonly viral, but also toxins, drugs, and autoimmune processes.

Findings in CMR include myocardial edema, wall motion abnormalities, and patchy myocardial LGE. Tissue edema is best visualized in T2-weighted spin-echo CMR images.[10] LGE in myocarditis is usually patchy, and involves the subepicardial regions.[10] Since edema, and not fibrosis, is the cause for LGE in