INTRODUCTION

Cardiomyopathies (CMPs) are myocardial diseases associated with cardiac dysfunction. In 1995, the World Health Organization (WHO) modified the classification of the cardiomyopathies, in agreement with new acquired concepts on the pathogenicity of the muscular heart disease. According to this new classification, primary cardiomyopathies can be: (1)

- 1 Dilated.
- 2 Restrictive.
- 3 Hypertrophic.
- 4 Arrhythmogenic Right Ventricular Dysplasia.
- 5 Non Classified.

Cardiac MRI has become an important imaging technique for the diagnosis and follow- up of CMP. In fact, echocardiography, usually the first step in CMP evaluation, has some pitfalls, mainly its limited acoustic window & its dependence on the operator skills in both carrying out & interpreting the examination. On the contrary, cardiac MRI allows a reproducible and accurate evaluation of myocardial morphology, function, perfusion, and tissue damage in a noninvasive and "one-stop shop" way. (2, 3 & 4)

Fundamental principles of magnetic resonance imaging (MRI) has been widely used to image the brain and other stationary organs within the body. More recently there has been considerable interest in its cardiovascular applications, largely as a result of software and hardware advances that enable high temporal and spatial resolution imaging. The term cardiovascular magnetic resonance (CMR) is used internationally to refer to MRI of the heart and great vessels. (5)

CMR offers a broad range of imaging techniques that allow physicians to determine cardiac physiology, anatomy, tissue characterization, flow patterns and vascular angiography. Image acquisition is multiplanar and no geometrical assumptions are made in contrast to 2D echo, so that measurements are both very accurate and reproducible. (5)

The strength of magnetic resonance imaging (MRI) lies in its intrinsic tissue contrast. It has emerged as the gold standard for measurement of cardiac volumes, mass, and ejection fraction. Tissue characterization is possible with different imaging weighting (T1/T2) and with the application of gadolinium-containing extracellularT1-shortening contrast agents. Myocardial edema, often present in acute inflammation of whatever cause, results in a high signal in T2-weighted imaging. (6)

Extracellular, gadolinium-containing contrast agents distribute in the vascular and interstitial spaces, but are excluded from the cellular space. If the interstitial space is enlarged as in myocardial fibrosis (whether for ischemic or non ischemic reasons), or if the ability of the myocyte to exclude the contrast agent is impaired as in acute myocardial infarction, the concentration of the contrast agent is increased in that region, resulting in a high signal in special T1-weighted imaging late after the application of the contrast agent. This is called "late gadolinium enhancement" (LGE).

Because of the high spatial resolution, the transmural extent or the distribution, or both, of LGE, and therefore a pathological structural process, can be quantified. (7)

Most of the non-invasive imaging techniques are capable of diagnosing cardiomyopathy, but for some years the dominant technique has been echocardiography. The utility of MRI scanning in functional evaluation of ventricular function has increased considerably in recent years and this technique may take over as the "gold standard". (4)

Hypertrophic cardiomyopathy is a primary myocardial disease characterized by focal or diffuse left ventricular wall thickening in the absence of dilatation. Hypertrophic cardiomyopathy is often characterized by impaired regional myocardial function, arrhythmias, and decreased coronary flow reserve. In hypertrophic cardiomyopathy, cardiac MR imaging is used to assess left ventricular wall thickness and mass, regional myocardial function, and degree of left ventricular outflow tract obstruction. (8)

Apart from idiopathic primary restrictive cardiomyopathy, amyloidosis is a common cause of secondary restrictive cardiomyopathy. MR imaging can show functional impairment, biventricular hypertrophy, and nonspecific inhomogeneous gadolinium enhancement. Ventricular enhancement may also occur in certain forms of glycogen storage diseases(9).

Arrhythmogenic right ventricular dysplasia (ARVD) is characterized by structural and functional abnormalities of the right ventricular wall leading to ventricular arrhythmias and progressive right ventricular failure. ARVD is associated with sudden cardiac death. In addition to the morphologic and functional evaluation of the right ventricle with cardiac MR imaging, delayed enhancement MR imaging can demonstrate diffuse or segmental replacement of myocardium in the right ventricular free wall by fibrofatty tissue. (10)

DCM is characterized by enlargement and impaired contraction of left or both ventricles. With an estimated prevalence of 36/100 000 in adults in the United States. (11)

Although no cause is apparent in many cases, dilated cardiomyopathy is probably the end result of damage to the myocardium produced by a variety of toxic, metabolic, or infectious agents. It may be due to fibrous change of the myocardium from a previous myocardial infarction. Or, it may be the late sequel of acute viral myocarditis, possibly mediated through an immunologic mechanism. A reversible form of dilated cardiomyopathy may be found with alcohol abuse, pregnancy, thyroid disease, and chronic uncontrolled tachycardia. Many cases of dilated cardiomyopathy are described as *idiopathic* - meaning that the cause is unknown. (12)

Cardiac MRI study in dilated CMP should always include lateenhancement images, which are an important element in tissue characterization and can help differentiate between dilated CMP secondary to coronary artery disease and other causes of dilated CMP. (13)