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Parametric techniques for characterizing myocardial tissue through magnetic resonance imaging (Part 1): T1 mapping☆

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Abstract The development of myocardial fibrosis is a common process in the appearance of ventricular dysfunction in many heart diseases. Magnetic resonance imaging makes it possible to accurately evaluate the structure and function of the heart, and its role in the macroscopic characterization of myocardial fibrosis by late enhancement techniques has been widely validated clinically. Recent studies have demonstrated that T1-mapping techniques can quantify diffuse myocardial fibrosis and the expansion of the myocardial extracellular space in absolute terms. However, further studies are necessary to validate the usefulness of this technique in the early detection of tissue remodeling at a time when implementing early treatment would improve a patient's prognosis. This article reviews the state of the art for T1 mapping of the myocardium, its clinical applications, and its limitations.

PALABRAS CLAVE
Miocardio;
Patología;
Fracción de volumen extracelular;

Técnicas paramétricas de caracterización tisular del miocardio mediante resonancia magnética (parte 1): mapas de T1

Resumen El desarrollo de fibrosis miocárdica es un proceso común en la aparición de disfunción ventricular en muchas enfermedades cardiacas. La resonancia magnética permite valorar la anatomía y la función cardiaca con precisión, y su papel en la caracterización macroscópica

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Fibrosis miocárdica difusa;
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Mapas de T1

de la fibrosis miocárdica mediante las técnicas de realce tardío ha sido ampliamente validado en clínica. En fechas recientes se ha demostrado que las técnicas de mapeo T1 miocárdico permiten la cuantificación en términos absolutos de la fibrosis difusa y de la expansión del espacio extracelular miocárdico. Sin embargo, se necesitan estudios adicionales que consigan validar la utilidad de esta técnica en la detección temprana del remodelado tisular en un momento en que la instauración de un tratamiento precoz permita mejorar el pronóstico de los pacientes. Este artículo revisa el estado actual de las técnicas de mapeo T1 del miocardio, sus aplicaciones clínicas y sus limitaciones.

Introduction

The myocardium is a specialized muscle with very particular, well-differentiated characteristics that allow it to perform its main function, which is adapting cardiac output to the physiological needs of the body at every moment. In order to perform that function, the heart must have, in addition to proper contractility, the correct capacity for relaxation and elasticity that will allow it to adapt to the changes of the filling volume (preload) and peripheral resistance (afterload). The composition and characteristics of the myocardial interstice play a key role in maintaining the myocardial function. In many primary diseases of the myocardium there occur changes in the relative composition of the interstice, especially an increase in production and a reduction of collagen replacement.[1]

Diffuse myocardial fibrosis (DMF) is characterized by the collagen deposit in the cardiac interstice and it is a feature of adverse remodeling.[2] DMF is present in the initial stages of many heart diseases, conditioning diastolic dysfunction due to a reduction of myocardial elasticity.[3] In advanced stages, DMF is very evident[4] and it can compromise contractile function and generate systolic dysfunction,[5] and even trigger arrhythmias and sudden death.[6] What is really interesting about DMF is that it is not always an irreversible process particularly when it is detected in early stages. Thus it has been observed that the early implementation of some treatments, such as blockers of the renin–angiotensin–aldosterone system, can inhibit and limit fibrosis.[7] Consequently the early non-invasive detection of myocardial fibrosis would be of a great clinical interest.

Cardiac Magnetic Resonance Imaging (MRI) through late enhancement sequence with gadolinium allows us to visualize the infarction or focal fibrosis by using contrast media that spread throughout the extracellular space. Both the infarction and the focal fibrosis appear bright in this sequence compared to a normal myocardium, whose signal has been canceled and appears hypointense.[8] This is why the processes affecting the myocardium in a diffused manner, in which there are no areas of healthy myocardium, can be difficult to identify using this modality of late enhancement with gadolinium.[9] The endomyocardial biopsy continues to be the reference diagnostic modality for diffuse myocardial processes despite the fact that it is an invasive procedure and therefore subject to sampling errors.[4] This is why the evaluation of diffuse interstitial disease through T1 mapping modalities has been of great clinical interest, due to its potential in the early diagnosis and evaluation of diffuse diseases of the myocardium, both interstitial fibrosis and other infiltrating conditions or deposit-associated diseases. These T1 mapping sequences allow us to quantify the T1 tissue relaxation time by using an analytical expression of the image based on the signal intensity of pixels. Quantifying certain parameters using this modality has been suggested as a prognostic marker in some diseases, and as a surrogate goal when assessing the results of clinical trial.[10]

This article will be the first of two updates describing the new modalities for measuring T1 and T2 myocardial tissue relaxation time and their representation on parametric maps. In this first part, the current status of T1 mapping modalities is reviewed, as well as its clinical applications and limitations.

Beyond myocytes: the extracellular matrix

The cardiac extracellular matrix is a complex network of fibers made out of structural and non-structural proteins, among which myocytes, fibroblasts, immune cells and vascular cells can be found. In addition to the key scaffolding function, participation in the remodeling and in the synthesis of collagen, the cardiac extracellular matrix also has important non-structural functions, interacting with several cellular components.[2] The matrix remodeling is an active process[11] that varies based on the disease that is regulated on a cellular level by mechanical and hemodynamic factors, and also by the action of different neurohormonal systems. Collagen is the main structural protein, predominantly type I (80%) and type III (11%) collagen[2] and it provides support and plasticity. In normal conditions, the collagen fiber network makes up only 2–4% of the cardiac structure.[12] This percentage, known as collagen volume fraction, increases with age,[13] can be sped up in different heart diseases[13–15] and it is a common histological characteristic in chronic heart failure.[4] The increase in the collagen volume fraction results primarily in a diastolic dysfunction,[16] and in later phases, the interstice remodeling is also associated with matrix degradation leading to the development of ventricular dilation and systolic dysfunction.[17] Fibrosis is also the substrate that promotes arrhythmogenesis by altering the conduction and genesis of reentry circuits.