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Cardiac Fibrosis in Heart Failure: The Need for a Comprehensive Care Strategy

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Description

Heart Failure (HF) is a major health problem and is associated with high resource use and health care costs. Despite significant improvements in HF treatment, morbidity and mortality remain high. In particular, cardiac fibrosis is considered an important cause of the increasing burden of HF. Indeed, it is a key factor in heart failure and its progression and adverse effects in both Coronary Artery Disease (CAD) and non-ischemic heart disease. Cardiac fibrosis is a heterogeneous and dynamic process that depends on the etiopathogenic cause of heart failure and the stage of the disease. Therefore, it has been proposed that the integration of cardiac fibrosis into the management of heart failure is a medical necessity that requires appropriate diagnostic and therapeutic strategies. After reviewing the general aspects of cardiac fibrosis, this review article focuses on the analysis of its accurate and precise non-invasive diagnosis and the evaluation of individual treatment and prevention options.

Aortic stenosis

Cardiac fibrosis is defined as excessive accumulation of collagen fibers in the myocardium of a damaged heart. Characterization of cardiac fibrosis depends not only on the amount of collagen, but also on the quality of collagen [1-3]. Type I collagen makes up about 85% of all collagen proteins and forms thick fibers important for strength, while type III collagen forms flexible thin fibers important for elasticity. An abnormal increase in the ratio of type I to type III collagen has been reported in endomyocardial biopsies in patients with severe Aortic Stenosis (AS) and heart failure with preserved left ventricular ejection fraction. In contrast, type III collagen type I predominance has been described in Eosin Methylene Blue EMBs of patients with end-stage, Heart Failure with reduced Ejection Fraction (HFrEF) due to either CAD or idiopathic dilated cardiomyopathy. Another factor that critically determines the stiffness or elasticity of collagen fibers is the degree of covalent bonds between the micro fibrils of the structural components [4,5]. The degree of myocardial collagen cross-linking in EMBs is related to LV stiffness and filling pressure in hypertensive patients with either HFpEF or HFrEF. Based on the characteristics of the deposits, two main types of cardiac fibrosis are distinguished, repair fibrosis and reactive fibrosis The first appears as focal macroscopic or microscopic scars based on collagen fibers that form during the healing process and replace dying cardiomyocytes after ischemic and non-ischemic injuries. The latter manifests as diffuse collagen fibers and groups that accumulate in the interstitial and perivascular regions and develop in response to chronic exposure of the heart to biomechanical stress that occurs in various cardiac and extracardiac diseases. Both patterns of fibrous deposits can coexist [6-8]. For example, in explanted hearts from advanced CAD and Myocardial Infarction (MI) patients, in addition to a macroscopic repair scar reflecting the loss of large numbers of cardiomyocytes, there is reactive interstitial fibrosis in areas distant from the myocardial scar.

Cardiac fibrosis

On the other hand, in patients with severe, reactive diffuse interstitial and perivascular fibrosis is associated with reparative microscars that reflect the loss of small foci of cardiomyocytes. Cardiac fibrosis is characterized by activated cardiac fibroblasts and myofibroblasts, the secretion of which leads to changes in the extracellular processing of fibrillar collagen, which promotes excessive accumulation of collagen fibers [9,10]. Activation of cardiac fibroblasts involves many changes, including proliferation and increased expression of periostin, extensive endoplasmic reticulum formation and differentiation of myofibroblasts with ultrastructural and phenotypic characteristics of smooth muscle cells derived from the formation of contractile polymerized stress fibers. which contain de novo synthesized α-Smooth Muscle Actin (α-SMA). Although local cardiac fibroblasts are the main source of activated fibroblasts, there is considerable heterogeneity in their development during development, in the adult heart and in disease states.

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