Editorial

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Editorial Note on Myocardial Fibrosis

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Editorial

Myocardial fibrosis is a major part of cardiac remodeling which leads to heart failure and death. Myocardial fibrosis results from increased myofibroblast activity and excessive extracellular matrix deposition. Many cells and molecules are involved in this process providing targets for potential drug therapies.

Fibrosis is a common response to injury in most of the cardiomyopathies but the pattern and distribution of fibrosis differ between pathologies with its potential role as a diagnostic marker.

Heart failure is the clinical manifestation of various forms of cardiovascular disease which is a devastating disorder characterized by interstitial fibrosis, reduced ventricular compliance and chamber remodeling.

Myocardial fibrosis is a significant global health problem which is associated with all forms of heart disease. Cardiac fibroblasts comprise an essential cell type in the heart which is responsible for the homeostasis of the extracellular matrix. Upon injury these cells transform to a myofibroblast phenotype and contribute to cardiac fibrosis. This remodeling involves pathological changes that includes chamber dilation, cardiomyocyte hypertrophy and finally leads to the progression to heart failure.

Diffuse myocardial fibrosis resulting from the excessive deposition of collagen fibres through which the entire myocardium is encountered in a number of cardiac diseases. This lesion results from alterations in the regulation of fibrillary collagen turnover by fibroblasts, facilitating the excessive deposition of type I and type III collagen fibres within the myocardial interstitium and around intramyocardial vessels.

The biosynthetic properties of cardiac fibroblasts that result in the alteration of fibrillary collagen turnover are the major determinants in the pathogenesis of diffuse myocardial fibrosis.

Patients with dilated cardiomyopathy (DCM) often presents with progressive congested heart failure, thromboembolic disease and arrhythmia in forms of left ventricular mural thrombus. DCM is seen as a major cause of heart failure apart from hypertension and coronary heart disease.

All heart muscle diseases that cause heart failure finally converge into one dreaded pathological process that is myocardial fibrosis. Myocardial fibrosis predicts major adverse cardiovascular events and death, yet we are still missing the targeted therapies capable of halting and reversing its progression. Fundamentally it is a major problem of disproportionate extracellular collagen accumulation that is part of normal myocardial ageing.

Myocardial fibrosis is the final point of convergence for all heart muscle diseases, ushering in the development of heart failure and death. Effective anti-fibrotic therapies are urgently needed but clinical trials are proving expensive.

Myocardial fibrosis is a common hallmark of many diseases of the heart. Late gadolinium enhanced MRI is a powerful tool to image replacement fibrosis after myocardial infarction (MI). Interstitial fibrosis can be assessed indirectly from an extracellular volume fraction measurement using contrast-enhanced T1 mapping.

Myocardial fibrosis alters the architecture of the myocardium, facilitating the development of cardiac dysfunction, also inducing cardiac arrhythmias, influencing the clinical course and outcome of cardiac failure patients. Focusing on myocardial fibrosis may potentially improve patient care through the targeted diagnosis and treatment of emerging fibrotic pathways.

