10. THE ROLE OF CARDIAC FIBROSIS IN DIASTOLIC HEART DYSFUNCTION

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Introduction. Cardiac diastolic dysfunction (DD) represents globally one of the main causes for congestive cardiac failure, occurring in 1/3 of reported cases. DD is most commonly a consequence of systemic arterial hypertension and ischemic cardiomyopathy. Histopathologic studies of hearts affected by DD have determined excessive deposits of extracellular collagen and the reconstruction of the heart architecture, known as cardiac fibrosis. The remodeling of the extracellular matrix decreases the filling rate, compliance, and diastolic suction of the heart, therefore altering cardiac function. Current discoveries suggest therapeutic potential for reversing cardiac damage.

Aim of study. Emphasizing the pathophysiological and functional alterations of the fibrotic myocardium in the cardiac cycle and identifying therapeutic methods for countermanding cardiac damage.

Methods and materials. Systemic analysis of up to date articles about cardiac fibrosis and diastolic dysfunction via PubMed, Google Scholar, Medscape. 14 articles (2004-2020) were chosen as the main sources.

Results. The particular attribute in which cardiac fibrosis exerts modifications is the negative pressure created by the ventricle during diastole, leading to a rapid ventricular filling time, in which the collagen fibers act as an elastic system that stores energy with each contraction, consequently releasing it along with elongating the ventricle wall. From a pathophysiological outlook, the key point is the loss of energy during the distortion of the perimysial collagen, caused by the friction of internal molecules, a phenomenon called viscoelasticity. Therefore, the energy loss yields in passive stiffness and noncompliance of the ventricle wall, decreasing the diastolic suction and thus the preload of the heart. The aggregate of energy loss is directly relative to the rate between type I collagen fibers, and type III, with the latter decreasing as the fibrosis progresses. An additional factor that contributes to passive stiffness is the capacity of contraction by the fibrous tissue, independent of that of the muscle fibers, a process accomplished by the myofibroblasts. Excessive collagen synthesis has been demonstrated to be stimulated by the renin-angiotensin-aldosterone system, therefore angiotensin converting enzyme inhibitors, along with the activation of metalloproteinases may allow for reversing cardiac damage.

Conclusion. Cardiac fibrosis and associated diseases can manifest in various pathogenetic ways, therefore understanding the mechanisms of diastolic dysfunction allow for a better management of affected patients.