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Diffuse myocardial fibrosis in chronic heart failure: A problem yet to be solved

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Chronic heart failure is one of the leading causes of mortality and hospitalization worldwide, and its incidence is expected to increase dramatically due to its association with ageing and prevalent risk factors (e.g. hypertension, obesity, diabetes...). Alterations in the different histocellular component of the heart (i.e. myocardial remodeling) contribute to the onset and progression of heart failure. In particular, diffuse myocardial fibrosis (DMF) is one the main hallmarks of myocardial remodeling, being associated with the development of cardiac dysfunction and poor outcome. DMF is a heterogeneous and dynamic process, dependent on the triggering insult, the pathophysiological mechanisms involved and the stage of the disease. Moreover, when analyzing the impact of DMF we have to consider not only the quantity of collagen fibers but also their physicochemical properties (e.g. degree of cross-linking). In this context, it is important to accurately phenotype the histomolecular patterns of DFM in heart failure patients, in order to design specific anti-fibrotic therapies aimed at the underlying pathophysiological mechanisms, facilitating the implementation of precision medicine strategies in the management of heart failure. Since cardiac biopsies are not routinely performed for diagnosis, we have been working on the identification of non-invasive circulating (blood) biomarkers of DFM phenotypes with clinical usefulness for diagnosis, risk stratification and therapy monitoring of patients. On the other hand, our group has also been focused on analyzing the molecular mechanisms involved in collagen fibers formation and maturation to identify more specific targets for addressing DMF in heart failure. In this seminar I will present some examples of our recent findings related to these 2 lines of research: the search for biochemical non-invasive biomarkers of heart failure and the identification of novel potential therapeutic targets.