**DIABETES AND HEART FAILURE: AN INFLAMMATORY PERSPECTIVE**

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Diabetes is associated with an increased risk for developing heart failure (HF). Similarly HF is associated with an increase prevalence of diabetes which intensifies with the disease severity. HF is characterized by a broad increase in pro-inflammatory markers. In fact markers for neuro-humoral activation, extra-cellular matrix turnover, pro-inflammation such as hs CRP, interleukins, osteopontin, ST2, galactine-3, and pro-oxidative markers such as TBARS are markedly elevated even in ambulatory patients with reduced as well as in those with preserved ejection fraction. Scientific evidences have shown that a pro-inflammatory milieu at the vascular, myocardial and systemic levels contributes to the physiopathology of HF and diabetes. At the vascular level, inflammation contributes to coronary atherosclerosis, endothelial and micro-vascular dysfunction. Local myocardial inflammation likely play a significant role on cardiac remodeling and fibrosis, early pathological processes in heart failure with preserved EF. Systemically, inflammation impaired oxygenation at the pulmonary and peripheral levels and as such contribute to symptoms and disease progression in HF. In addition to the increase in various markers it appears that the neutrophil, neutrophil-derived microparticles, and neutrophils extra-cellular traps (NETs) are likely to play a significant role in this disease condition. Further investigations are ongoing in order to better understand the behavior and role of these markers in HF and diabetes