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Role of Micro Particles in Heart Failure Development

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Description

As a major health concern, Heart Failure (HF) is associated with high prevalence, morbidity, mortality, and financial costs in developing countries, whereas mortality rates have been declining over the past decade. The HF phenotype's presentation has significantly changed over the last decade. In recent studies, the incidence of HF with reduced Ejection Fraction (HFrEF) has decreased, while the incidence of HF with preserved Ejection Fraction (HFpEF) has increased. Interestingly, the clinical outcomes, including mortality and readmission, for patients with HFpEF were no better than for those with HFrEF. However, both HF phenotypes have sufficiently distinct CV risk factors, metabolic comorbidities, and aging. Whether Cardio Vascular (CV) and metabolic risk factors significantly affect HF incidence remains controversial.

It is recommended by current HF clinical guidelines to use limited number of biomarkers (natriuretic peptides, galectin-3, cardiac troponins, ST2) to risk stratify patients with acute, acutely decompensated, and chronic HF, as well as possibly to use biomarker-guided therapy. Yet, biomarker determinations for individuals with both HF phenotypes (HFrEF and HFpEF) require prospective validation due to uncertainty about their usefulness in predicting prognosis. The discovery of new biomarkers, which could have a higher predictive value regardless of limitations recently defined for "old" biomarkers, seems enticing in this context.

Micro Particles (MP's) are defined as small, phospholipid-rich vesicles (less than 100 nm in diameter) formed by several types of cells as a result of apoptosis or activation by several stimuli. MPs exhibit controversial effects, depending on their origin (derived from activated cells or apoptotic cells). As a matter of fact, activated endothelial cells contribute to the repair of vasculature, whereas apoptotic endothelial cells worsen the integrity and function of endothelium. MPs play a role in the pathophysiology of a diverse range of conditions, including inflammation, blood coagulation/thrombosis, cell cooperation, cell differentiation/growth, malignancy/tumor progression, metastasis, and angiogenesis/neovascularization.

In individuals with suspected CV disease or exhibiting CV/metabolic risk factors, as well as in patients with known CV disease, there were high circulating levels of MP's derived mainly from erythrocytes, mononuclear cells, endothelial cells and platelets. Despite the fact that erythrocyte and platelet-derived MP's significantly contribute to the coagulation cascade and inflammation associated with atherothrombosis, myocardial infarction, and ischemia-induced cardiac dysfunction, endothelial cell-derived MP's were found to be indicative of endothelial dysfunction and a predictor of HF progression and development. The recent studies have shown that circulating MP's derived from endothelial cells may play a causal role in atherosclerosis, endothelial dysfunction, HF, myocardial infarction, thromboembolism, diabetes-induced vasculopathy, and renal disease. Some triggers, like neurohormones, active molecules, growth factors, cytokines, and free radicals, have been suggested to affect the repair ability of progenitor cells through epigenetic modifications that lead to MP secretion.

MP's may also be linked to endogenous repair systems and CV risk factors. Recently, it was shown that MP's from apoptotic and activated endothelial cells (described as having "Impaired phenotypes") are imbalanced. Not only may it be a biomarker of endothelial dysfunction in HF patients and predictor of HF phenotype's development, but it may also be a predictor of cardiac disease in individuals.

Conclusion

Apoptotic endothelial cell-derived MP's associated with decreased angiogenic MP's shattering from activated endothelial cells are discussed as a common attribute of cardiac dysfunction beyond etiology and concomitant risk factors. Intriguingly, impaired phenotypes of MP's may be associated with non-CV diseases including metabolic syndrome, obesity, and insulin resistance. It is not clear whether MP's impaired phenotype contributes to CV disease including heart failure or whether underdiagnosed CV disease is a result of previously imbalanced MP's.