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Commentary

Heart Failure and Pathogenesis of Coronary Blood Flow

Joshua Reynolds*

Division of Cardiothoracic Surgery, Harvard University, Massachusetts, USA

DESCRIPTION

Heart failure is a clinical entity in which cardiac output is insufficient to maintain appropriate perfusion and normal body functioning, for during exercise and then, in more severe cases, during rest. The two most common types of heart failure are ischemia and non-ischemic heart failure. Reduced coronary blood flow causes cardiac contractile dysfunction in ischemic heart failure, and this is true for shocked and hibernating myocardium, coronary micro-embolization, myocardial infarction, and post-infarct remodelling, as well as perhaps the takotsubo cardiomyopathy. Dilated cardiomyopathy is the most common kind of non-ischemic heart failure, which is caused by genetic mutations, myocarditis, toxic substances, or prolonged tachy-arrhythmias, and involves changes in coronary blood flow and contributing to cardiac contractile dysfunction.

Genetic abnormalities induce hypertrophic cardio-myopathy, however it can also be caused by increasing pressure and volume overload (hypertension, valve disease). Heart failure with a maintained ejection fraction is associated with significant coronary microvascular dysfunction, the cause of which is uncertain. The current study defines the changes in coronary blood flow that are causes or consequences of heart failure in its many forms. Apart from any potential coronary atherosclerosis, all heart failure entities have impaired coronary blood flow to varying degrees: Increased extravascular compression, impaired nitric oxide-mediated, endothelium-dependent vasodilation, and increased vasoconstriction to neuro-humoral activation mediators. Impaired coronary blood flow plays a role in the evolution of heart failure and is thus a viable target for both existing and new therapies.

Heart failure and atherosclerosis are common and frequently co-occur, as they result not only from more or less specific genetic predispositions, but also from lifestyle-related risk factors and comorbidities such as physical inactivity, obesity and metabolic syndrome, diabetes, hypertension, and pollution. In patients with heart failure and retained ejection fraction, the coexistence of predisposing risk factors and comorbidities, coronary atherosclerosis, and coronary microvascular dysfunction is particularly evident. The relationship between coronary artery disease and heart failure is complicated. On the one hand, coronary artery disease can cause myocardial ischemia and infarction, which can lead to heart failure.

Genetic abnormalities, on the other hand, can induce heart failure, and heart failure can decrease coronary blood flow even in the absence of coronary atherosclerosis. Then, heart failure and impaired coronary blood flow interact: Any form of heart failure predisposes to myocardial ischemia by increasing extravascular compression and coronary vasoconstriction in response to neurohumoral activation, and any form of myocardial ischemia impairs Left Ventricular (LV) function further. In heart failure, morphological changes (arteriolar hypertrophy, capillary rarefication) and functional abnormalities, such as impaired endothelium-dependent and metabolic vasodilation, increased vasoconstriction to neurohumoral activation mediators and increased extravascular compression, characterize the coronary circulation.

Generally, Circulation and the coronary microcirculation in particular, and the coronary circulation in specific kinds of heart failure, such as hypertrophy, hypertensive heart failure, or heart failure with intact ejection fraction.

They are previous studies addressed the general causes of reduced coronary dilator reserve in all types of heart failure, as well as the more particular abnormalities of the coronary circulation in the various heart failure entities.

Heart failure is virtually always linked with coronary vascular dysfunction, not only in the presence of coronary atherosclerosis but also in the absence of it. Coronary heart failure and reduced coronary blood flow have complicated cause-and-effect interactions. Coronary Heart failure is definitely a result of myocardial ischemia without or with reperfusion in stunning and hibernating, coronary micro-embolization, myocardial infarction, and post-infarct remodeling-these are ischemic heart failure syndromes. There is a vicious cycle between the impairment of myocardial contractile function and the impairment of coronary circulation in any form of heart failure, in which myocardial ischemia worsens heart failure and vice versa and it is reflected by the reduction of coronary dilator reserve as a predictor of poor clinical outcome from heart failure.

Correspondence to: Joshua Reynolds, Division of Cardiothoracic Surgery, Harvard University, Massachusetts, USA, E-mail: Joshua_Reynolds@harvard.edu

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