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The Evolving Role of Coronary Micro Vascular Dysfunction in Heart Failure with Preserved Ejection Fraction

Daniel Martinez*

Department of Cardiology, University of Barcelona, Barcelona, Spain

Introduction

Heart Failure with Preserved Ejection Fraction (HFpEF) has become an increasingly recognized form of heart failure, accounting for a significant proportion of heart failure cases, particularly in older adults and those with comorbidities such as hypertension, diabetes, and obesity. Unlike Heart Failure with Reduced Ejection Fraction (HFrEF), where systolic dysfunction is the primary mechanism, HFpEF is characterized by a Preserved Left Ventricular Ejection Fraction (LVEF) but impaired diastolic function, resulting in symptoms of heart failure. One of the key pathophysiological features of HFpEF is Coronary Microvascular Dysfunction (CMD), a condition where the small coronary arteries fail to properly regulate blood flow to the heart muscle, leading to inadequate myocardial perfusion despite a normal or near-normal epicardial coronary artery structure. [1]

Coronary microvascular dysfunction has been increasingly recognized as a critical factor in the development and progression of HFpEF. CMD in HFpEF is thought to contribute to myocardial ischemia, increased myocardial stiffness, and impaired left ventricular relaxation, all of which worsen symptoms and clinical outcomes. The mechanisms underlying CMD in HFpEF are complex and multifactorial, involving endothelial dysfunction, increased vascular stiffness, inflammation, and metabolic derangements. As our understanding of CMD's role in HFpEF expands, it is becoming evident that targeting microvascular dysfunction could provide new therapeutic opportunities for improving outcomes in this challenging condition. This paper explores the evolving role of coronary microvascular dysfunction in HFpEF, its pathophysiological mechanisms, diagnostic challenges, and potential therapeutic strategies. [2]

Description

Coronary microvascular dysfunction is a condition in which the coronary microcirculation—comprising arterioles and capillaries—fails to meet the metabolic demands of the heart muscle. In healthy individuals, these small blood vessels regulate blood flow to various regions of the myocardium by responding to changes in metabolic demand and oxygen requirements. In patients with HFpEF, however, this regulatory mechanism becomes impaired, leading to inadequate perfusion despite normal epicardial coronary arteries. Several factors contribute to CMD in HFpEF, including endothelial dysfunction, increased vascular stiffness, and inflammation. Endothelial dysfunction in the microcirculation leads to a reduced ability to dilate blood vessels in response to increased myocardial demand. This, combined with increased vascular stiffness and microvascular rarefaction (a reduction in capillary density), results in an imbalance between oxygen supply and demand, leading to myocardial ischemia, increased myocardial stiffness, and diastolic dysfunction. Studies have shown that patients with HFpEF frequently have elevated levels of markers of endothelial dysfunction, such as soluble Vascular Cell Adhesion Molecule-1 (sVCAM-1), and that these markers correlate with worse clinical outcomes. [3]

The role of CMD in HFpEF is not limited to myocardial ischemia alone but extends to increased myocardial stiffness and Impaired Left Ventricular (LV) relaxation, which are hallmark features of HFpEF. CMD contributes to increased LV filling pressures, as the microvascular dysfunction limits the heart's ability to effectively relax and fill during diastole. Moreover, CMD promotes fibrosis in the myocardium, leading to the stiffening of the heart muscle and worsening diastolic dysfunction. As a result, the heart becomes less able to accommodate blood during the diastolic phase of the cardiac cycle, leading to elevated left atrial pressures and pulmonary congestion. This dysfunction in the microcirculation and the subsequent myocardial changes exacerbate the symptoms of HFpEF, including shortness of breath, fatigue, and exercise intolerance. Furthermore, inflammation and metabolic disturbances, such as insulin resistance and adiposity, play key roles in the pathophysiology of CMD in HFpEF. Adipose tissue-derived cytokines and pro-inflammatory molecules contribute to endothelial dysfunction and microvascular rarefaction, perpetuating the cycle of microvascular impairment and worsening heart failure symptoms. [4]

Despite its critical role, the diagnosis of coronary microvascular dysfunction in HFpEF remains challenging due to the limitations of traditional diagnostic tools. Coronary angiography, which is typically used to evaluate epicardial coronary artery disease, cannot detect microvascular abnormalities and may not be helpful in diagnosing CMD in HFpEF. Instead, advanced techniques such as echocardiography, Cardiac Magnetic Resonance (CMR) imaging, and Possession Emission Tomography (PET) have been employed to assess microvascular function indirectly. For example, CMR with myocardial perfusion imaging can detect areas of subendocardial ischemia that are suggestive of microvascular dysfunction. Invasive coronary function testing, such as the measurement of Coronary Flow Reserve (CFR) using Doppler wire, is considered the gold standard for diagnosing CMD. However, these tests are not routinely used in clinical practice due to their complexity and invasiveness. As a result, CMD in HFpEF remains underdiagnosed and often untreated. Recent advances in non-invasive imaging techniques may improve the ability to detect and monitor microvascular dysfunction, providing new opportunities for early intervention and better management of HFpEF. [5]

Conclusion

The growing recognition of Coronary Microvascular Dysfunction (CMD) as a critical factor in the pathophysiology of heart failure with preserved Ejection Fraction (HFpEF) has profound implications for the diagnosis and treatment of this condition. CMD plays a central role in myocardial ischemia, increased myocardial stiffness, and impaired left ventricular relaxation, all of which contribute to the hallmark symptoms and poor prognosis associated with HFpEF. The pathophysiology of CMD in HFpEF involves complex interactions between endothelial dysfunction, vascular stiffness, inflammation, and metabolic disturbances, making it a multifactorial process that is difficult to address with a single therapeutic approach.Despite the challenges in diagnosing CMD, emerging diagnostic techniques such as cardiac magnetic resonance imaging (CMR), Positron Emission Tomography (PET), and invasive coronary function tests are helping to identify microvascular

***Address for Correspondence:** Daniel Martinez, Department of Cardiology, University of Barcelona, Barcelona, Spain; E-mail: daniel.martinez@ub.edu*

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abnormalities in HFpEF. As these technologies become more widely available and better integrated into clinical practice, they could improve early diagnosis and facilitate targeted therapies. However, the most significant hurdle remains the lack of effective treatments aimed specifically at microvascular dysfunction in HFpEF. Current treatments for HFpEF primarily focus on managing symptoms and comorbidities, but they do not directly address the underlying microvascular dysfunction.

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