Reduced Coronary Flow Reserve and Arterial Stiffness: Two Faces of the Same Coin

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The heart and the arterial tree are strongly associated, not only anatomically but also functionally. The cardiovascular (CV) system has the ability to regulate cardiac output and arterial blood pressure, and to respond to changes in heart rate and preload appropriately. This CV link depends largely on the properties of both the heart and of the vasculature into which blood is pumped and becomes particularly evident under pathological conditions. CV remodeling is a process induced by arterial hypertension and combined risk factors.1,2 It includes a preclinical stage during which symptoms are not evident, and a clinical stage, which corresponds to the progression of preclinical disease and to the action of additional triggering mechanisms representing overt organ damage. Advances in noninvasive diagnostic imaging now allow for accurate clinical detection of preclinical disease.

Cardiac remodeling (i.e., left ventricular (LV) concentric remodeling and hypertrophy) affects negatively not only diastolic function and midwall systolic mechanics but also coronary microcirculation, whose functional alterations can be noninvasively detected by transthoracic Doppler echocardiographic derived coronary flow reserve (CFR). On the contrary, the changes occurring in the arterial vessels involve the walls of both small and large arteries. The increased arterial stiffness (or decreased arterial compliance) is one of the mechanisms underlying vascular remodeling. Arterial stiffening can be assessed noninvasively by estimation of the elastic modulus, high-resolution echo-tracking, and pulse-wave velocity (PWV) analysis.3 The various indices of arterial compliance predict CV outcomes, and even a simple surrogate of arterial stiffness such as clinic pulse pressure has a prognostic value.

Ikonomidis et al.4 find an independent association between PWV and CFR in never-treated hypertensive patients. They suggest that the increased PWV induces a mismatch between myocardial oxygen demand (as a result of increase in LV afterload, wall stress, and cardiac workload) and myocardial perfusion which results also in LV diastolic dysfunction.

These findings open a new track about the relationships existing between peripheral and coronary vasculature. However, the exclusion of patients with clear-cut LV hypertrophy appears as an important limitation of this study, in relation to the possible confounding effect of this variable which might have blunted the observed association between PWV and CFR. LV hypertrophy is a recognized hallmark of CV risk and its presence is associated with abnormalities of CFR as well as with LV diastolic dysfunction and PWV increase. Coronary microvessel impairment is directly attributable to changes in LV structure corresponding to LV concentric geometry, of which LV concentric hypertrophy is the maximal expression. Extravascular compressive forces and concomitant hypertrophy of the coronary microvascular walls might be mechanisms underlying the abnormalities of CFR in the hypertensive as in the diabetic heart.5 In this view, it can be hypothesized that the same mechanisms, which at the heart level induce dysfunction of coronary microvasculature and reduction in CFR, can be also responsible for the arterial stiffness of the vascular tree. Abnormalities of CFR and PWV can be considered, therefore, as two faces of the same coin: the CV remodeling which involves both the heart and the vessels and can be diagnosed by the current ultrasound technology.

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