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COMMENTARY

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# Malperfusion in dilated cardiomyopathy: One more link in the pathologic chain?

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Dilated cardiomyopathy (DCM) is considered the primary non-ischemic heart disease, but there are controversial data regarding the microcirculatory disturbance in these patients. While the major epicardial vessels are always intact in these patients, the malperfusion of the myocardium of the left ventricle (LV) may be present, and the role of this malperfusion in the progression of the disease is not clear. The non-invasive echocardiographic assessment of the coronary blood flow reveals that it may be increased at rest and decreased during pharmacological stress, leading to a decreased coronary flow reserve.[1]

Beyond echocardiographic studies, microvascular dysfunction in DCM has been previously investigated with various imaging modalities. Bietenbeck M. et al. performed the quantitative myocardial first-pass perfusion cardiovascular magnetic resonance (CMR) imaging and coronary sinus flow measurements at rest and during maximal vasodilatation which is a well-validated approach for the quantification of global myocardial blood flow in CMR. The blood flow parameters were reduced at rest and during stress in both groups of patients with DCM and hypertrophic cardiomyopathy.[2] Interestingly, in another study, it was shown that myocardial blood flow assessed by the CMR in DCM could be elevated at rest with a substantial decrease during the stress-test with adenosine. This corresponds well with the previous echocardiographic findings and also can reflect the possible stress-induced stunning instead of chronic hypoperfusion in DCM.[3]

Another promising in microcirculation evaluation, though not a fully available modality, is PET. In a study by Yamaguchi et al., the authors observed the regional 18-fluorodesoxiglucose uptake in all patients with ischemic cardiomyopathy in the fasting state, while patients with DCM showed variable patterns of uptake. Nevertheless, when going to glucose loading state, the DCM patients demonstrated mostly non-reversing patterns of uptake, which allows PET to distinguish DCM from ischemic cardiomyopathy.[4]

(99 m)Tc-methoxy-isobutyl-isonitrile and (123)I-15-(p-iodophenyl)-3(R,S)-methylpentadecanoic acid dual single photon emission computed tomography also provides valuable data on perfusion-metabolism mismatches, which carry the prognostic role in DCM.[5]

Myocardial contrast echocardiography (MCE) being a more simple, bedside method has been previously found to be helpful in differentiating the DCM and ischemic heart disease in patients with acute onset of heart failure.[6] However, another group of authors has the confronting results: the contrast replenishment rate was similar in patients with coronary artery disease and without it.[7]

In this issue of the Journal of Clinical Ultrasound Sha Tang et al, for the first time the MCE was combined with speckle tracking strain analysis in a layer-specific manner in patients with DCM and compared with the same parameters in healthy volunteers.[8] While the poor global and regional LV systolic function assessed by speckle tracking strain in DCM patients in comparison to healthy subjects is awaited, the authors performed the comprehensive analysis of the interaction between segmental parameters by dividing the myocardium into standard segments plus three layers in each segment. Therefore, the data on gradients in strain and perfusion parameters becomes the mainstay of this study. The idea of layer-specific strain analysis is not new, but still can be regarded as a pure experimental method with no clinical implication. For example, in the work by

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Nagata Y et al. the authors offer Endo/Epi strain ratio for gradient assessment.[9] Therefore, the authors demonstrated an interesting correlation between MCE perfusion and longitudinal function at rest in DCM patients with the diffuse pattern of impairment in all segments and layers.

Considering the aforementioned CMR-based and PET-based studies, which showed a substantial decrease in coronary flow reserve between rest and stress, the MCE with pharmacological stress could be the rational development of basic ideas, presented in this study. Moreover, the dynamic nature of MCE can give the opportunity for dynamic malperfusion assessment in patients with DCM during stress.

In addition, new insights into the pathophysiology of DCM may provide the basis for new clinical trials of various agents, intended to restore the damaged microcirculatory link in the chain of global LV deterioration.

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The authors have nothing to disclosure.

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Not applicable.

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