Editorial

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A Novel Approach for Identifying Ischemic Cardiomyopathy

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Since Burch et al.[1] first introduced the terminology "ischemic cardiomyopathy (ICM)," it has been widely used in clinical practice. The condition may originate from prolonged ischemia, a single large infarct, or repeated micro-infarcts inducing left ventricular dysfunction and remodeling through apoptosis and fibrosis as a result of activation of the endothelin or renin-angiotensin-aldosterone system.[2] It can be reversed after successful revascularization in the presence of a viable myocardium. However, there have been several debates about the actual existence of that kind of cardiomyopathy. Because traditionally cardiomyopathy is a result of a genetically or acquired disease-driven myopathic processes, current WHO criteria do not include ICM in the classification of cardiomyopathy.[3] But in many clinical practices and studies, an initial classification as ICM or non-ICM is widely used in patients with severely depressed left ventricular function. Use of the terminology "ICM or non-ICM" persists in spite of a lack of official guidelines because it can provide a simple and easily communicated understanding of the underlying pathogenesis and treatment options. A myocardium that is malfunctioning from prolonged ischemia can be improved after successful revascularization treatment, such as coronary artery bypass surgery. If a patient is classified as non-ICM, then a search for other causes must be begun. However, the current definition of ICM is vague and cannot fully explain the cause of severely depressed left ventricular dysfunction.[4] In addition, as a previous study showed, ICM with post-myocardial infarction has a far different prognosis from ICM without post-myocardial infarction[5]; the underlying causes of non-post myocardial infarction ICM are very complex and cannot be easily explained by the concept of a hibernating myocardium. This complexity may explain the unexpected dearth of positive outcomes in the "Surgical Treatment for Ischemic Heart Failure" trial, such as improved survival, according to viability tests, following bypass surgery in patients with ICM.[6]

In this regard, a study done by Kim et al.[7] provides important insight about possible misclassification as ICM or non-ICM based on the traditional definition using coronary angiography and a history of previous myocardial infarction. According to their observations with delayed hyper-enhancement (DHE)-cardiac magnetic resonance (CMR), some cases classified as ICM had non-ischemic DHE patterns, such as midwall striae, patch, or subepicardial DHE. Also, a small portion of non-ICM patients had ischemic DHE patterns, such as subendocardial DHE. They suggested that the classification of ICM should be individualized after considering DHE-CMR findings. According to their observations, some patients with ICM showed midwall linear DHE, which is a unique finding related to prognosis in non-ICM cases.[8] It provides new insight into the pathology of left ventricular dysfunction in these patients and also the pathophysiology of midwall DHE, which is believed to be from prior myocarditis or an increase in gadolinium diffusion distance due to high wall stress.[9] However, no satisfactory explanation has been proposed because usually the amount of interstitial fibrosis is highest in the subepicardium, not the midwall, in non-ICM patients.[10]

The study done by Kim et al.[7] has a limitation in that identification of ICM without prior myocardial infarction requires evaluation for the presence of myocardial perfusion defects because DHE only represents replacement fibrosis. Therefore, patients without DHE and with non-DHE regions also need to be characterized to explain

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severely depressed left ventricular systolic function. To overcome this limitation, inclusion of new parameters such as myocardial flow reserve or extracellular space volume fraction would be helpful. In addition, a large-scale study can provide clarity on the clinical characteristics of this discordant population.

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