Editorial

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Coronary Circulation; Macro or Micro, That It the Question

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The coronary arterial circulation consists of large epicardial arteries and microvascular system.[1] Epicardial coronary arteries can be visualized by coronary angiography and their diameters range from a few millimeters to 400–500 μm. Microvascular system cannot be clearly delineated by coronary angiography, and it is responsible for most of the coronary vascular resistance. The vascular tone and resistance of these vessels can be modulated under various physiological and pharmacological conditions in order to control the myocardial blood flow. Because the microvessels are too small to be revascularized by either percutaneous or surgical intervention and are difficult to assess, the main focus of clinical cardiology has been the large epicardial arteries. However, it is well-known that the patients with microvascular dysfunction have worse outcomes than those without microvascular dysfunction.[2][3] Furthermore, recent technical advances have enabled easy and reliable assessment of the microvascular system in a catheterization laboratory.[4-6] Coronary flow reserve (CFR) has been used to assess the microvascular function in patients without significant epicardial lesions. However, the index of microcirculatory resistance (IMR), which can be measured by using a 0.014 inch coronary temperature-sensing guidewire, has become a more popular tool to assess the microvascular function in a catheterization laboratory. Compared to CFR, IMR is less influenced by the presence of epicardial disease and hemodynamic variables such as blood pressure and heart rate.[6]

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Although there have been studies assessing microvascular function in patients with non-ST segment elevation myocardial infarction, most of those patients had significant epicardial coronary artery disease.[7] Park et al.[8] investigated the association between the microvascular dysfunction and the level of cardiac troponin in patients without significant epicardial coronary artery disease, and they presented their findings in the issue of Korean Circulation Journal. In 19 patients with elevated cardiac troponin I, angiographically normal coronary arteries, and preserved left ventricular function, IMR and CFR were simultaneously measured in all of the 3 vessels. Presence of epicardial coronary artery disease was ruled out by both angiography and fractional flow reserve measurement, which was the strength of this study. In this study, no correlation was observed between the level of troponin I and CFR. However, the highest IMR value was significantly greater in patients with definite elevation of troponin I than in those with only detectable elevation of troponin I. There was a positive correlation between the highest IMR and troponin I level (r=0.459, p=0.048). This study demonstrated that IMR is a better index than CFR for the assessment of microvascular function in a catheterization laboratory. This important finding was noted only because the authors performed a thorough assessment of the whole coronary system in this study. With respect to the mechanism for troponin I elevation in these patients, acute microvascular injury is a more probable mechanism than underlying microvascular dysfunction as only the highest IMR had a correlation with the level of troponin I.

Although the authors comprehensively assessed the macro- and microvascular coronary circulation in these patients, the study results should be interpreted with caution and the following points should be considered:

1) As pointed out by the authors, the number of patients was too small to draw a clinically relevant conclusion from this study. Especially, the thermodilution technique for measuring the CFR is technically demanding, CFR is more vulnerable to confounding variables and its inter- and intra-individual variability is higher than that of the other physiological parameters.

2) The time intervals between the clinical event and laboratory

tests and between measurement of troponin I and CFR/IMR were not reported in this manuscript. It is therefore difficult to interpret the study results as both elevation of troponin I and recovery from microvascular injury are time-dependent variables.

3) The level of troponin I was low in most of the patients, therefore it is difficult to elucidate the relationship between the level of troponin I and microvascular injury. It will be interesting to evaluate the effect of one patient with a very high level of troponin (>20 ng/mL) on the study results.

Considering all these limitations, further studies in a larger population are needed to assess the relationship between microvascular injury (or dysfunction) and myocardial injury in patients without significant epicardial coronary artery disease. Although it is beyond the scope of this manuscript, it will be interesting to perform an imaging study to investigate the cause of myocardial injury in these patients.

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