How should we manage the patients with type 2 myocardial infarction?

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Myocardial infarction (MI) referring to irreversible necrosis of cardiac myocytes is caused by prolonged ischemia conventionally due to the reduced blood flow by the presence of coronary stenosis. In The Universal Definition of Myocardial Infarction, published in 2007,[1] five different clinical types of acute MI were introduced; the definitions of the five types have recently been updated in The Fourth Universal Definition of Myocardial Infarction (Table 1).[2] Type 1 MI (T1MI) is characterized by plaque rupture, ulceration, fissuring, erosion, or dissection in the setting of atherosclerotic coronary artery disease (CAD), with resultant intraluminal thrombus, cessation of myocardial blood flow, and acute myocyte necrosis. Type 2 MI (T2MI) is myocardial necrosis resulting from an increase in myocardial oxygen demand and/or a decrease in myocardial blood flow, occurring in the absence of acute plaque rupture or coronary thrombosis. Diagnostic criteria for T2MI include the following: (1) detection of markers of cardiac myonecrosis, for example, elevated troponin concentrations; (2) clinical context lacking signs or symptoms suggestive of acute coronary syndrome or non-ischemic contributors to myocardial injury, for example, myocarditis; and (3) identification of physiological stressors resulting in myocardial supply and demand imbalance. The most frequent mechanisms causing T2MI are anemia, respiratory failure, and tachyarrhythmias.[3]

Mortality rates for acute MI have declined in this 3 decades significantly by the improvement of revascularization techniques and the benefits of antiplatelet agents, beta-blockers, and statins have been demonstrated in patients with MI. As a result, the prognosis for patients with T1MI was improved. In contrast, T2MI is a myocardial disorder that occurs even in patients with, by definition insignificant coronary artery involvement and assumes a discrepancy between CAG findings and prognosis. In clinical practice, it is often experienced that there is a discrepancy between the results of CAG tests and the presence of myocardial damage. It has been thought that the presence of thrombus and coronary spasm are all affected by an imbalance between myocardial oxygen supply and demand. In the case of adenosine stress myocardial perfusion imaging (MPI) test rather than exercise stress MPI which is characteristically diagnosed by evaluating reactive hyperemia of coronary arteries, it is considered difficult to diagnose T2MI which is due to vasospasm or imbalance of myocardial oxygen imbalance. In daily clinical patient management, we determine treatment policy based on many test results and aim to improve the prognosis of patients. In patients with T1MI caused by arteriosclerosis, it can be understood that it is useful to manage patients by diagnosing myocardial ischemia by MPI, whereas in patients with T2MI unrelated to the presence of acute coronary arteriosclerosis, the usefulness of MPI in deciding treatment strategy remains questionable.

In this issue of the Journal of Nuclear Cardiology, Colon et al reported that the significance of adenosine stress MPI in the management of patients with T2MI.[4] They studied 234 cases with T2MI of whom 58 patients showed abnormal MPI results and found that abnormal MPI was associated with increased risk of cardiac events in patients with larger perfusion abnormalities, larger extent of myocardial ischemia and less LVEF, those

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Table 1. Universal definition of MI

Type	Classification	Clinical and diagnostic criteria
1	Spontaneous MI	Plaque rupture/erosion with occlusive or non-occlusive coronary thrombus
2	Supply/demand mismatch	Mismatch between myocardial oxygen supply and demand unrelated to acute coronary atherothrombosis
3	Suspected MI-related death	Cardiac death in a setting suggestive of ischemic process without definitive cardiac biomarker evidence of MI
4a	PCI-related MI	Rise in cardiac biomarkers accompanied by symptoms, electrocardiographic, angiographic, or imaging evidence of ischemia after PCI
4b	Stent/scaffold thrombosis	Confirmed stent thrombosis in context of ischemia and dynamic cardiac biomarker changes
4c	Restenosis with PCI	Focal or diffuse restenosis, or a complex lesion with a rise and/or fall of troponin
5	CABG-related MI	Rise in cardiac biomarkers accompanied by electrocardiographic, angiographic, or imaging evidence of ischemia after CABG

CABG, coronary artery bypass graft; *MI*, myocardial infarction; *PCI*, percutaneous coronary intervention

findings are similar features of T1MI. Of great interest in this paper is the ability to estimate the prognosis of T2MI patients due to an imbalance between myocardial oxygen supply and demand without actually using the results of exercise tests that increase myocardial oxygen demand.

The prognosis in patients with T2MI is poor since T2MI typically occurs among older patients with greater comorbidities.[5] Major adverse cardiovascular event rates are also high in this cohort and approximately 30% of patients will have a cardiovascular event over 5 years.[5] Physicians should consider risk stratification in patients with T2MI for the likelihood of coronary artery disease. Prospective clinical trials are needed to define the efficacy and safety of secondary prevention therapies in patients with type 2 myocardial infarction or myocardial injury, which have the potential to modify future outcomes.

Disclosure

Kazuya Takehana declares that he has no conflict of interest.

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