Is Plaque Rupture Always Responsible in Acute Coronary Syndrome?

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ABSTRACT
The majority cause of myocardial infarction is the atherothrombotic event, mainly cause by plaque rupture. Since the 20th century, it was found that the plaque rupture was not the solely condition responsible for the acute coronary syndrome. With the invention of more sensitive myocardial biomarker, a series of guideline was written as guideline for the definition of myocardial infarction. This review discuss about the consensus in the Universal Definition of Myocardial Infarction.

Background
Before the 20th century, it is accepted that the cause of myocardial infarction was the atherothrombotic event. It was proved by the autopsy that confirms the relation between occlusive thrombosis and myocardial infarction.1 At the mid of 20th century, however, the evidences demonstrate that myocardial infarction was not only caused by only atherothrombotic. One third of the cases were proved to have no thrombosis in the coronary arteries.2,3 In 1950, WHO published the clinical criteria for diagnosis of myocardial infarction by a combination of two of three characteristics: typical symptoms (i.e., chest discomfort), enzyme rise and a typical ECG pattern involving the development of Q waves.4 With the advances of the more sensitive myocardial enzymes, namely troponins, the collaboration of European Society of Cardiology (ESC) and the American College of Cardiology published a consensus document to redefine myocardial infarction using a biochemical and clinical approach, and reported that myocardial injury detected by abnormal biomarkers in the setting of acute myocardial ischaemia should be labelled as MI.5 These concepts were revised with the newer consensus documents, namely Universal Definition of Myocardial Infarction Consensus Document in 2007, introducing a novel MI classification system with five subcategories. The development of more sensitive assays for markers of myocardial injury made further revision of the consensus, especially for patient who undergo coronary procedures or cardiac surgery namely the Joint ESC/ACC/AHA/WHF Task Force produced the Third Universal Definition of Myocardial Infarction Consensus Document in 2012.

The history of documents in defining the myocardial infarction was described in Figure 1.6
Figure 1. History of documents on the definition of myocardial infarction. ACC = American College of Cardiology; AHA = American Heart Association; ESC = European Society of Cardiology; ISFC = International Society and Federation of Cardiology; MONICA = MONItoring of trends and determinants in CARDiovascular disease; NHLBI = National Heart, Lung, and Blood Institute; UDMI = Universal Definition of Myocardial Infarction; WHF = World Heart Federation; WHO = World Health Organization. Source: The Fourth UDMI, 2018

Pathological characteristics of myocardial ischemia and infarction

An MI is defined pathologically as myocardial cell death due to prolonged ischaemia. The necrosis progresses from the subendocardium to the subepicardium over several hours. The time course may be prolonged by increased collateral flow, reduced determinants of myocardial oxygen consumption, and intermittent occlusion/reperfusion, which can precondition the heart. Timely implementation of reperfusion therapy, when appropriate, reduces ischaemic injury of the myocardium.6

Myocardial enzymes for the detection of myocardial injury and infarction

Cardiac troponin I (cTnI) and T (cTnT) are components of the contractile apparatus of myocardial cells and are expressed almost exclusively in the heart. Cardiac troponins are the preferred biomarkers for the evaluation of myocardial injury. The assay of high-sensitivity (hs)-cTn assays are preferred for routine clinical use. The myocardial markers, namely creatine kinase MB isoform (CK-MB), are less sensitive and less specific. Myocardial injury is defined as being present when blood levels of cTn are increased above the 99th percentile upper reference limit. The injury may be acute, if there is a rise and/or fall of cTn values.6

Cardiac troponins characterize injury to myocardial cells. Nevertheless they do not indicate the underlying pathophysiological mechanisms, and can arise after mechanical stretch despite normal hearts. Proof of myocardial injury with myocardial cells death can be detected in clinical conditions associated with non-ischemic mechanisms of myocardial injury (Figure 2).6
Figure 2. Spectrum of myocardial injury, ranging from no injury to myocardial infarction. Various clinical entities may involve these myocardial categories, e.g. ventricular tachyarrhythmia, heart failure, kidney disease, hypotension/shock, hypoxaemia, and anaemia. cTn = cardiac troponin; URL = upper reference limit. (a) represents no myocardial injury with cTn values ≤ 99th percentile URL or not detectable. (b) represents myocardial injury with cTn values > 99th percentile URL. (c) represents myocardial infarction = clinical evidence of myocardial ischaemia and a rise and/or fall of cTn values > 99th percentile URL. Source: Fourth UDMI, 2018.

The conditions associated with myocardial ischemic or non-ischemic disease are represented in Table 1. The complexity and comorbidity of the patient's illness could contribute to the myocardial injury, which could even worsen the patient's condition (Table 1). These evidences confirms that not all of myocardial infarction was not caused by the atherothrombosis, but merely correlated to systemic disturbances conditions that affect the heart and myocardial cells.

**Conclusions**

The myocardial injuries resulting from the non-atherothrombotic conditions require more proper diagnostic tools and management strategies rather than those initiated by atherothrombosis. Nevertheless the distinction between type 1 and type 2 MI may be challenging and requires careful judgment. Even the distinction between MI and myocardial injury may cause problems. In general, a diagnosis of MI should not be made if the clinical setting is not of acute ischemia.

References