Is Plaque Rupture Always Responsible in Acute Coronary Syndrome?

Putrika Ratna Gharini

Department of Cardiology and Vascular Medicine, Faculty of Medicine, Public Health and Nursing, UniversitasGadjahMada – Dr. Sardjito General Hospital, Yogyakarta, Indonesia

Corresponding author:
Putrika Ratna Gharini, MD, - email: ikagharini@gmail.com
Department of Cardiology and Vascular Medicine, Faculty of Medicine, Public Health and Nursing, Universitas Gadjah Mada – Dr.Sardjito Hospital

Jalan Farmako, Sekip Utara, Yogyakarta, Indonesia 55281

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]

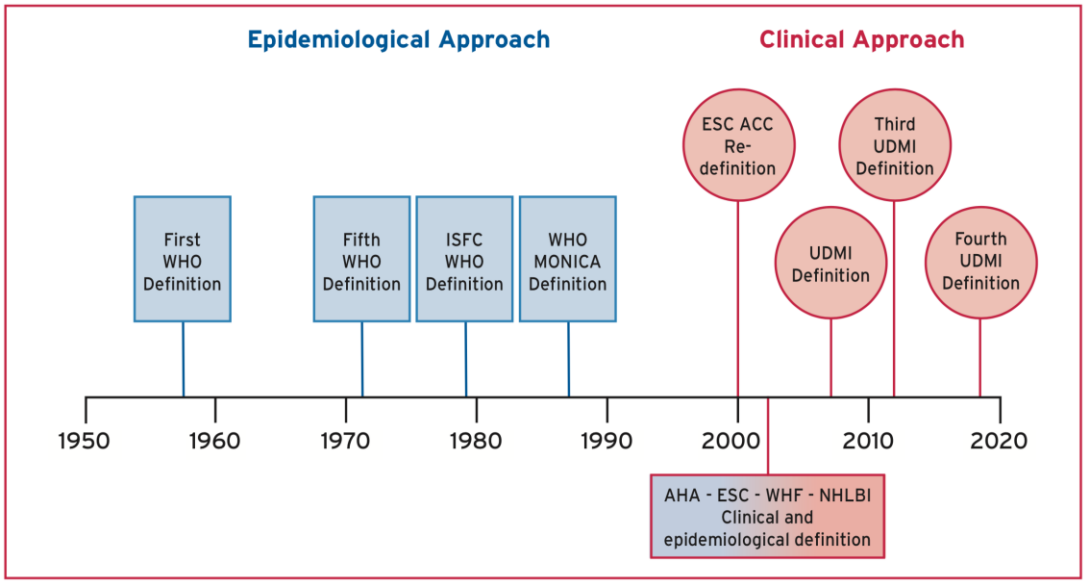

Figure 1.History of documents on the definition of myocardial infarction. ACC = American College of Cardiology; AHA = American HeartAssociation; ESC = European Society of Cardiology; ISFC = International Society and Federation of Cardiology; MONICA = MONItoringof trends and determinants in CArdiovascular 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[6]

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]

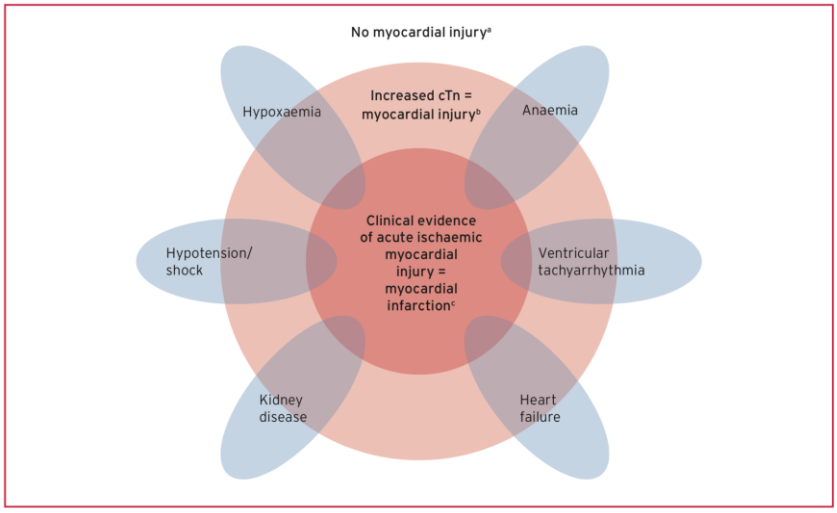

Figure 2.Spectrum of myocardial injury, ranging from no injury to myocardial infarction. Various clinical entities may involve these myocardial categories, e.g. ventricular tachyarrhythmia, heart failure, kidney disease, hypotension/shock, hypoxaemia, and anaemia. cTn = cardiac troponin; URL = upper reference limit. (a) represents no myocardial injury withcTn 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[6]

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).[6] 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. Nevertheles 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.[7]

Further Reading: Fourth universal definition of myocardial infarction (2018), Expert consensus document. European Heart Journal (2019) 40, 237–269, doi:10.1093/eurheartj/ehy462

Table I Reasons for the elevation of cardiac troponin values because of myocardial injury

Myocardial injury related to acute myocardial ischaemia
Atherosclerotic plaque disruption with thrombosis.
Myocardial injury related to acute myocardial ischaemia because of oxygen supply/demand imbalance
Reduced myocardial perfusion, e.g. • Coronary artery spasm, microvascular dysfunction • Coronary embolism • Coronary artery dissection • Sustained bradyarrhythmia • Hypotension or shock • Respiratory failure • Severe anaemia
Increased myocardial oxygen demand, e.g. • Sustained tachyarrhythmia • Severe hypertension with or without left ventricular hypertrophy
Other causes of myocardial injury
Cardiac conditions, e.g. • Heart failure • Myocarditis • Cardiomyopathy (any type) • Takotsubo syndrome • Coronary revascularization procedure • Cardiac procedure other than revascularization • Catheter ablation • Defibrillator shocks • Cardiac contusion
Systemic conditions, e.g. • Sepsis, infectious disease • Chronic kidney disease • Stroke, subarachnoid haemorrhage • Pulmonary embolism, pulmonary hypertension • Infiltrative diseases, e.g. amyloidosis, sarcoidosis • Chemotherapeutic agents • Critically ill patients • Strenuous exercise

©ESC/ACC/AHA/WHF 2018

(Source: Fourth UDMI, 2018[6])

References

1. JB H. 1919. Thrombosis of the coronary arteries. J Am Med Assoc. 1919;
2. Friedberg C.K., Horn H. 1939. Acute myocardial infarction not due to coronary artery occlusion. JAMA [Internet], 112:1675–1679.
3. Miller R.D., Burchell H.B., Edwards J.E. 1951. Myocardial infarction with and without acute coronary occlusion : a pathologic study. JAMA Intern Med [Internet], 88:597–604.
4. World Health Organization. Expert Committee on Cardiovascular Diseases and Hypertension & World Health Organization. 1959. Hypertension and coronary heart disease : classification and criteria for epidemiological studies, first report of the Expert Committee on Cardiovascular Diseases and Hypertension. World Health Organization. https://apps.who.int/iris/handle/10665/40434
5. Antman E., Bassand J.P., Klein W., Ohman M., Lopez Sendon J.L., Rydén L., et al. 2000. Myocardial infarction redefined - A consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee f or the redefinition of myocardial infarction. J Am Coll Cardiol, 36:959-969.
6. Thygesen K., Alpert J.S., Jaffe A.S., Chaitman B.R., Bax J.J., Morrow D.A., et al. 2019. Fourth universal definition of myocardial infarction (2018). Eur Heart J, 40:237-269.
7. Eggers K.M. 2015. Defining acute myocardial infarction. Heart Metab, 67:34-38.