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Editorial

Fourth Universal Definition of Myocardial Infarction

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Editorial

The fourth Universal Definition of Myocardial Infarction (UDMI), published by the European Society of Cardiology/American College of Cardiology/American Heart Association/World Heart Federation on 25 August 2018 [1], updates the third UDMI of 2012. Much new knowledge has accrued since as evidenced by the bulkier document (33 pages and 201 references) compared to the 2012 version (17 pages, 102 references).

The new UDMI continues the tradition of clinical criteria for MI (encompassing Myocardial Injury) reflected by elevated cardiac biomarkers (preferably cardiac troponin - cTn) in the setting of myocardial ischemia (symptoms, ECG changes, or imaging evidence); the event is acute if there is a rise and/or fall in biomarkers and chronic if biomarkers are persistently elevated. The distinction between Myocardial Infarction (MI) and Myocardial Injury (MIn) is timely in the fourth UDMI. On the wards, junior doctors have been referring to what should now be called MIn as troponinitis. MIn refers to a rise in cTn with one value above the 99th percentile Upper Reference Limit (URL) in the absence of Myocardial Ischemia. MI is MIn with clinical evidence of myocardial ischemia.

The clinical classification of MI into 5 categories is retained. However, the section on Type 2 MI is expanded to better clarify underlying mechanisms including acute and chronic myocardial injury. It also emphasizes that both type 2 MIn/type 2 MI are common and connotes a poor prognosis. Pre-procedural MIn following cardiac/non-cardiac procedures is frequent enough to merit a section on its own. It is defined by increases of cTn values (>99th percentile URL) in patients with normal baseline values or a rise of cTn value >20% from baseline when above the 99th percentile URL but is stable or falling. The sections on types 4 and 5 MI are also considerably strengthened. In fact a new category type 4c MI (restenosis associated with percutaneous coronary intervention - PCI) has been added. The definitions of Type 4 and 5 MI remain unchanged as a rise in cTn >5x and >10x above the 99th percentile URL respectively [2]. However, the expert consensus document also raised other definitions of periprocedural MI with higher cutpoints (35x and 70x). Unique causes of MI or MIn (recurrent, re-infarction, associated with non-revascularization cardiac procedures, associated with non-cardiac procedures, heart failure, Takotsubo syndrome, non-obstructive coronary arteries, kidney disease, critically ill patients) are also included and considered separately.

The biochemical approach for diagnosing MIn or MI is updated. Troponins (cTnI and cTnT) are the preferred biomarkers for MI rule-out/rule-in [3] and their kinetic changes are emphasized. There is variability in time to peak value for cTn. For troponin values <99th percentile URL, changes >3 SD (standard deviations) around the measurement of the individual conventional troponin assay at relevant values are significant [3]. For high-sensitivity troponins (hs-cTn) the global variation (analytical and biological) of these assays is 50-60%. When the initial cTn is >99th percentile URL, a serial change >20% is significant [3]. The section on operationalizing rule-out and rule-in strategies for MIn and MI is succinctly summarized and complements recent expert guidelines on this matter [4,5].

The section on Electro Cardio Graphic (ECG) detection of MI has been considerably strengthened with tips on ECG confounders and application of supplemental ECG leads. Tachycardia, e.g. Atrial Fibrillation (AF), can cause insufficient coronary blood flow for myocardial demand, resulting in cellular hypoxia and abnormal repolarization [6,7]. Patients with new AF, rising cTn and new ST segment depression should not be classified as type 2 MI without further investigation.

Imaging techniques in MI management has been given more prominence. Cardiovascular Magnetic Resonance (CMR) can assess cardiac structure, function and perfusion with the aid of paramagnetic contrast agents [8,9]. In acute MI CMR detects even small sub endocardial MIs [10]. Computed Tomographic Coronary Angiography (CTCA) can differentiate between MI, pulmonary embolism and aortic dissection while also assessing perfusion, possibly useful in low to intermediate-risk patients with normal cTn at presentation [11,12].

The 4th UDMI concludes with useful discussions on regulatory perspectives on MI in trials, implications of the new definition for the individual and public, global perspectives, and its use in the healthcare system. The herculean effort undertaken by the expert committee to update us with newer concepts in the classification and evaluation of MI is laudable and should serve as compulsory reading for all Physicians, laboratorians and radiologists.

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