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New Universal Definition of Myocardial Infarction

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Recently, a joint ESC/ACCF/AHA/WHF Task Force published an expert consensus document on the universal definition of myocardial infarction [1]. The following points are extracts from this document that summarize its main features.

The main reason for agreeing on a new definition of myocardial infarction derives from the development and the wide availability of very sensitive and specific serological biomarkers that are able to detect even minimal myocardial necrosis.

Acute myocardial infarction is diagnosed when there is evidence of myocardial cell necrosis in the clinical setting of myocardial ischemia. In contrast to the historical World Health Organization (WHO) definition where symptoms, ECG and enzymes had equal weight for the diagnosis (the presence of any two would suffice), today biomarkers take precedence with imaging having also a diagnostic role. Consequently, acute myocardial infarction is diagnosed if a rise and fall of cardiac biomarkers (preferably troponin) is detected together with at least one of the following: a) symptoms of ischemia, b) new ST-T changes or new left bundle branch block (LBBB), c) development of pathological Q waves and d) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Prior myocardial infarction requires for its diagnosis any of the following criteria: a) new Q waves, b) imaging of a regional loss of viable myocardium that is thinned and fails to contract and c) pathological findings of a healed or healing myocardium.

CLINICAL CLASSIFICATION OF MYOCARDIAL INFARCTION

The clinical classification of myocardial infarction consists of 5 types:

- a) *Type 1*: Spontaneous myocardial infarction related to ischemia due to a primary coronary event (plaque erosion, rupture, fissuring, dissection)
- b) *Type 2*: Myocardial infarction secondary to ischemia due to either increased oxygen demand or decreased supply (spasm, embolism, anemia, arrhythmia, hypertension, hypotension)
- c) *Type 3*: Sudden unexpected death (biomarkers may have not been obtained or not yet raised)
- d) *Type 4*: Myocardial infarction associated with percutaneous coronary intervention (PCI) (type 4a) and stent thrombosis (type 4b). To diagnose peri-procedural necrosis in patients with normal baseline troponin values, biomarkers should be greater than 3 times the 99th percentile URL (upper reference limit).
- e) *Type 5*: Myocardial infarction associated with coronary artery bypass grafting (CABG). To diagnose peri-operative necrosis biomarkers should be greater than 5 times the 99th percentile URL, together with new Q waves, LBBB, graft or native artery occlusion or imaging of new loss of viable myocardium.

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CARDIAC BIOMARKERS, ECG & IMAGING

Although elevation of troponin is indicative of myocardial cell damage it does not indicate the mechanism. Therefore several conditions with elevated troponin do not imply overt ischemic heart disease (for example cardiac failure, renal failure, drug toxicity, sepsis, pulmonary embolism).

Electrocardiographic manifestation of ischemia that may lead to myocardial infarction are ST segment elevation (with hyperacute symmetrical increased amplitude T-waves being an early finding) or ST segment depression. New ST elevation requires a J point in two contiguous leads ≥ 0.2 mV in men and ≥ 0.15 mV in women for leads V2-V3 and ≥ 0.1 mV for the other leads. New ST depression and T wave changes requires horizontal or down-sloping depression ≥ 0.05 mV in two contiguous leads and/or T wave inversion ≥ 0.1 mV in two contiguous leads with a prominent R wave or R/S ratio >1 .

Imaging techniques are applied in the acute and the healing or healed phase of myocardial infarction. Rest echocardiography is the commonest method used but cannot distinguish ischemia from infarction. Radionuclide imaging, stress echocardiography and magnetic resonance imaging (MRI) can be used to identify viability of myocardial tissue.

IMPLICATIONS

The new definition of myocardial infarction has several epidemiological and clinical trial implications. In epidemiology, since biomarkers are able to detect smaller infarcts the incidence of non STEMI is increased while unstable angina is becoming a rarer diagnosis. Therefore, comparison of temporal trends of myocardial infarction incidence in registries will be affected and historical controls may be difficult to evaluate. In clinical trials with myocardial infarction as an outcome, an attempt to quantify myocardial damage by multiples of the 99th percentile URL of the biomarker is encouraged so that comparisons between various severity categories of infarction can be made possible.

Finally, the new definition, with more patients with limited myocardial injuries being diagnosed as myocardial infarctions may have psychological, legal, insurance and professional consequences.

SUGGESTED READING

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