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Guidelines

Third universal definition of myocardial infarction ☆

Jan Vojáček[a,*], Petr Janský[b], Tomáš Janota[c]

[a]Department of Cardiovascular Medicine I, University Hospital Hradec Kralove, Czech Republic
[b]Department of Cardiovascular Surgery, University Hospital Motol, Prague, Czech Republic
[c]Cardio ICU, 3rd Department of Internal Medicine University Hospital, Charles University, Prague, Czech Republic

Authors of the original ESC update document [1]: Kristian Thygesen, Joseph S. Alpert on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Universal Definition of Myocardial Infarction.

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*Corresponding author.
E-mail address: vojacjan@fnhk.cz (J. Vojáček).

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1. Preamble

The ESC guidelines help physicians to make decisions in their practice, but do not override the responsibility of health professionals to make decisions and to follow the rules and regulations applicable to drugs and devices.

2. Introduction

In 2000, the First Global MI Task Force presented a new definition of MI, which implied that any necrosis in the setting of myocardial ischaemia should be labelled as MI. These principles were further refined by the Second Global MI Task Force, leading to the Universal Definition of Myocardial Infarction Consensus Document in 2007, which emphasized the different conditions which might lead to an MI. However, the development of even more sensitive assays for markers of myocardial necrosis mandates further revision, particularly when such necrosis occurs in the setting of the critically ill, after percutaneous coronary procedures or after cardiac surgery. The Third Global MI Task Force has continued the Joint ESC/ACCF/AHA/WHF efforts by integrating these insights and new data into the current document, which now recognizes that very small amounts of myocardial injury or necrosis can be detected by biochemical markers and/or imaging.

2.1. *What is new in the third universal definition of myocardial infarction*

- Criteria for an acute myocardial infarction: new identification of intracoronary thrombus by angiography or autopsy.
- Elevation of cardiac troponin values because of myocardial injury:
  - myocardial injury related to primary myocardial ischaemia (MI type I);
  - myocardial injury related to supply/demand imbalance (MI type II);
  - myocardial injury not related to myocardial ischaemia.
- New multifactorial or undetermined myocardial injury (heart failure, stress Tako-tsubo cardiomyopathy, severe pulmonary embolism or hypertension, sepsis and critically ill patients, renal failure, severe acute neurological diseases e.g. stroke, subarachnoid haemorrhage, infiltrative disease, e.g. amyloidosis, sarcoidosis, strenuous exercise).
- ECG criteria of ST elevation *age and gender specific*.
- Criteria for periprocedural MI.

2.1.1. *Definition of myocardial infarction*

Definition of myocardial infarction is given in Table 1.

2.1.2. *Pathological characteristics of myocardial ischaemia and infarction*

MI is defined in pathology as *myocardial cell death due to prolonged myocardial ischaemia*. After the onset of myocardial ischaemia, histological cell death is not immediate, but takes a finite period of time to develop—as little as 20 min—or less in some animal models. It takes several hours before myocardial necrosis can be identified by macroscopic or microscopic post-mortem examination. Complete necrosis of myocardial cells at risk requires at least 2–4 h, or longer, depending on the presence of collateral circulation to the ischaemic zone, persistent or intermittent coronary arterial occlusion, the sensitivity of the myocytes to ischaemia, preconditioning, and individual demand for oxygen and nutrients. The entire process leading to a healed infarction usually takes at least 5–6 weeks. Reperfusion may alter the macroscopic and microscopic appearance.

2.1.3. *Biomarker detection of myocardial injury with necrosis*

Myocardial injury is detected when blood levels of sensitive and specific biomarkers such as cTn or the MB fraction of creatine kinase (CKMB) are increased. Cardiac troponin I and T are components of the contractile apparatus of myocardial cells and are expressed almost exclusively in the heart. Although elevations of these biomarkers in the blood reflect injury leading to necrosis of myocardial cells, they do not indicate the