

Update on ACC/ESC criteria for acute ST-elevation myocardial infarction

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ABSTRACT

Disruption of vulnerable or high-risk plaques is the common pathophysiological mechanism of acute coronary syndromes with or without ST elevation. The reflection of the same pathophysiological mechanism differs in non-ST-elevation acute coronary syndromes and ST-elevation myocardial infarction (STEMI) in terms of clinical presentation, prognosis and therapeutic approach. Diagnostic and therapeutic evolution had come along together from the beginning of the acute myocardial infarction (MI) concept. Pathological appearance of acute MI is classified as acute, healing and healed phases as a time related phenomenon. Clinical presentation of STEMI, is different than the other ischaemic cardiac events with the sudden onset, the duration and the severity of chest pain or discomfort. Although the old markers creatine kinase and the MB fraction, lactate dehydrogenase are also used for the diagnosis of acute MI, cardiac troponins are very sensitive and specific, and myoglobin is an early marker for acute MI. In electrocardiogram; new or presumed new ST segment elevation at the J point in two or more contiguous leads or Q wave in established MI are typical changes. Echocardiographic or nuclear techniques have been used widely to rule out or confirm STEMI. In conclusion; all clinical, pathological, biochemical, electrocardiographic analysis methods and new imaging techniques have their own unique contribution for evaluating STEMI. (*Anadolu Kardiyol Derg 2007; 7 Suppl 1; 14-5*)

Key words: ST-elevation myocardial infarction, pathological appearance, biochemical analysis, electrocardiography, imaging

As the diagnostic criteria had important changes from the beginning of the time period we first discerned acute myocardial infarction (MI), therapeutic options had also major changes during the years. Although major conceptual changes took place during the years in understanding and classifying acute MI, few changes occurred in pathophysiological considerations (1, 2). Rupture of an atherosclerotic vulnerable plaque; resulting with the platelet activation, adhesion, and aggregation, thrombin generation, and ultimately thrombus formation leading to the occlusion of a coronary artery and necrosis is still the major determinant, effecting the life expectancy of the patient (1). So, most of the efforts focused on the therapeutic strategies to establish an open artery as soon as possible.

The criteria for the definition of acute ST-elevation myocardial infarction (STEMI), must be evaluated in different aspects. Whereas the clinical presentation, the pathophysiological and the biochemical changes are the essential features of STEMI, the reflection of these changes to electrocardiography (ECG) and imaging techniques have great importance to improve the therapeutic strategies and health policies (2-7).

Clinical presentation of STEMI, is different than the other ischaemic cardiac events with the sudden onset, the duration and the severity of chest pain or discomfort. Preceding symptoms had no longer changed from the past definition of Braunwald. The reflection of the same pathophysiological mechanism differs in non-ST-elevation acute coronary syndromes (ACS) and STEMI,

mainly due to incomplete occlusion or the complete occlusion in the presence of good collateral flow of the related artery in non-ST-elevation ACSs (8).

Pathological appearance of acute MI is classified as acute, healing and healed phases. Acute phase refers to the period between 6 hours to 7 days, healing phase is between 7 to 28 days, healed MI is after 29 days. The clinical and ECG timing of an acute ischemic event may not be the same as the pathologic timing of an acute MI (2).

Biochemical markers are valuable especially in the presence of symptoms and ECG changes. Recent advances in this era helped us to detect STEMI more specifically and to estimate the time period of the necrosis. Although the old markers creatine kinase and the MB fraction (CK-MB), lactate dehydrogenase are also used for the diagnosis of acute MI, cardiac troponins are very sensitive and specific and myoglobin is also an early marker for acute MI. The recent data recommend cardiac troponins T and I or if not available creatine kinase-MB must be the preferred markers for the diagnosis of STEMI (2, 6-8).

Electrocardiography is one of the most important diagnostic tools during ST elevation (STEMI). It remains to be at the same level of evidence, especially in comparing non-ST-elevation acute coronary syndromes and STEMI. As it is very important to diagnose STEMI to compare the correct treatment strategy, ECG is also a cornerstone to direct the treatment options. Prognostic implications due to the location and the extension of MI, is easy to demonstrate in most of the cases with ECG (9, 10).

The most prominent changes in ECG are in ST, T and Q waves. According to "The Consensus Document of The Joint European Society of Cardiology/American College of Cardiology Committee for the Redefinition of Myocardial Infarction" ECG changes indicating myocardial ischemia that may progress to MI are: new or presumed new ST segment elevation at the J point in two or more contiguous leads with the cut-off points ≥ 0.2 mV in leads V1, V2, or V3 and ≥ 0.1 mV in other leads, new or presumed new ST segment depression or T wave abnormalities or both, should be observed in two or more contiguous leads. Also, new or presumed new symmetric inversion of T waves ≥ 1 mm should be present in at least two contiguous leads. Electrocardiographic changes in established MI are: any Q wave in leads V1 through V3, Q wave ≥ 30 ms (0.03 s) in leads I, II, aVL, aVF, V4, V5, or V6. (The Q wave changes must be present in any two contiguous leads, and be ≥ 1 mm in depth) (2, 9-12).

Imaging modalities as echocardiographic or nuclear techniques and cardiac magnetic resonance imaging have been used widely to rule out or confirm the presence of acute infarction or ischemia, to identify nonischemic conditions causing chest pain, short- and long-term prognoses and mechanical complications of acute infarction (1, 2, 13).

In conclusion: In certain conditions as acute ischemia, acute or evolving MI and established MI; all clinical, pathological, biochemical, electrocardiographic analysis methods and new imaging techniques have their own unique contribution for evaluating STEMI. It is possible to detect very small amount of myocardium faced to infarction, with these diagnostic approaches.

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