Which Biomarker is the Best Indicator of Acute Myocardial Infarction: A Critical Review in a Medical College of Western Himalayan Region

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Abstract: Acute myocardial infarction (AMI) is one of the major causes of mortality and morbidity worldwide. About 10% of patients who are admitted to emergency departments with chest pain every year are diagnosed with heart attack. AMI is a condition that can be due to ischemic heart disease or coronary artery disease in conjunction, and it becomes manifest when an atherosclerotic plate ruptures and a developing thrombus occludes the coronary artery totally or partially, restricting blood access to the heart. In this case, the opening of the occluded coronary artery is usually provided by inserting a stent. However, when stents are insufficient, coronary bypass is performed by cardiac pulmonary bypass surgery using the left internal mammary artery or saphenous vein to maintain regular nourishment of the heart.

Keywords: Cardiac Biomarkers, Acute Myocardial Infarction, Homocysteine.

INTRODUCTION

AMI has found its place in the concept of acute coronary syndrome (ACS). ACS includes a group of clinical syndromes ranging from unstable angina pectoris, AMI with non-S (downward deflection immediately after ventricular contraction)-segment elevation and T (recovery of ventricles) segment elevation to AMI, with ST-segment and sudden death. The sensitivity and specificity of electrocardiography (ECG) are low in ACS. In the majority of cases with ST-segment elevation in ECG and typical ischemic chest pain, AMI with a Q wave (downward deflection immediately preceding ventricular contraction) develops in most cases and AMI without a Q wave develops in a few. However, the majority of cases without ST-segment elevation develop unstable angina pectoris or AMI without a Q wave, with a few developing AMI with a Q wave. ST-segment elevation changes into ST-segment depression when an oxygen-free environment persists.

As the sensitivity and specificity of ECG are low in diagnosing AMI, the criteria for AMI were decided by the European Society of Cardiology (ESC) and the American College of Cardiology (ACC). Accordingly, a patient has to have at least two of the following: typical symptoms, a characteristic elevation or decrease pattern in cardiac markers (eg, CK-MB izoenzymes), preferably serum troponins (cTnI or cTnT), or a typical ECG trace with Q waves that indicate a diagnosis of AMI.
Ischemia due to decreased coronary artery flow causes deterioration of ventricular function and myocardial necrosis.\textsuperscript{10} Therefore, such enzymes as ALT, AST, LDH, CK, and troponins have been indicators for years as a diagnosis of AMI \textsuperscript{10,14}. 

There are no “ideal” biochemical markers for the diagnosis of almost all diseases in medicine. This fact increases the health costs of countries and morbidity and mortality in its inadequacy. Despite recent advances in medicine, mortality and morbidity due to cardiovascular diseases remain the foremost problem in world health. The presence of an ideal biochemical marker of the cardiac system would reduce the morbidity and mortality rate associated with AMI. An ideal cardiac marker: 1) must be sensitive enough to detect a small degree of damage to the heart, 2) should be specific to the heart muscle (it must exclude damage to other [skeletal] muscles), 3) should give information regarding the severity of the infarct and the prognosis of the disease, 4) should also show the result of reperfusion therapy in AMI, 5) needs to distinguish between reversible and irreversible damage, 6) ought not to be detected in patients showing nonmyocardial damage, 7) should help in early and late diagnosis, 8) should be easy to measure, fast, cheap, and quantitative, and finally, 9) should have long-term storage conditions and be stable under them.\textsuperscript{5,11}

**DISCUSSION**

AST currently has no place in the diagnosis of AMI. Peptide-structured molecules have not yet found any place in the diagnosis of AMI. If CK-MB is used in the diagnosis of AMI, serial increase and decrease should not be seen in the level of CK-MB. CK-MB should be at least 10–14 U/L. In monitoring at 4-hour intervals, CK-MB should be increased by 50%. If viewed in a single time period, it should be twice the normal level. If it is analyzed after 72 hours, it is important that CK-MB is seen to be higher than troponins and LDHs. Cardiac enzymes are superior to ECG in the diagnosis of AMI. Myoglobin, FABP, and GPBB are early biomarkers in the diagnosis of AMI. TnT and TnI are late markers. CK-MB is a remarkable AMI biomarker in the first 10–12 hours. An increase in Tnl is an indicator of myocardial injury if CK-MB is within normal limits. For the diagnosis of AMI, Tnl is more specific. While CK-MB levels return to normal within 72 hours after MI, and as cardiac troponins are released in the troponin complex, their level can be high in the blood even 7–14 days later. In other words, the analysis of troponins can be used to diagnose an individual’s past AMI within 7–14 days. TnT is not specific to the heart. TnT shows biphasic release during AMI. The first peak occurs within 24 hours of symptoms, and the second one is on the fourth day. TnT levels are high in the blood for a few days and return to normal values after 10–14 days. Tnl is specific to the heart. After 9–12 hours, the sensitivity for the diagnosis of AMI is 100% and has monophasic release kinetics. In patients with chronic renal failure, TnT may increase without myocardial damage. Therefore, TnI is a more reliable biomarker in the diagnosis of AMI in patients with chronic renal failure. Multiple cardiac biomarkers are recommended, as they increase specificity and sensitivity in the diagnosis of AMI.

**CONCLUSION**

AMI has a high mortality rate worldwide, but fast and reliable diagnosis can reduce mortality. Biomarkers are elevated because of cell death in the myocardium. Therefore, many bio-chemical parameters of heart-tissue origin have been used in the diagnosis of AMI from past to present. A biomarker that meets the definition of an ideal cardiac biomarker, as we previously described, has yet to be discovered. In other words, there is no consensus on the best cardiac biomarker. There is no doubt that a better one will always exist. Analysis of a single biomarker is not recommended, since there is no ideal and specific single biomarker. It should also be noted that cardiac biomarkers do not make for a diagnosis, but do help in reaching one.

**REFERENCES**


