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Causes of elevated cardiac troponin I in patients with normal coronary angiogram

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Background : Myocardial infarction(MI) was redefined by the Joint ESC/ACC Committee in 2000. The redefinition of MI emphasizes the importance of biochemical markers of myocardial necrosis such as cardiac troponin and MB fraction of creatine kinase(CK-MB). Cardiac troponin I(cTnI) is most recently described and has nearly absolute myocardial tissue specificity, as well as high sensitivity. But an increased value for cTnI that indicates myocardial injury is not always synonym of myocardial infarction or ischemia due to coronary artery occlusive disease. Objective : To review the precipitating cause for elevated cTnI in patients with angiographically normal or near normal coronary arteries. Methods : Consecutive patients who underwent coronary angiography for suspected coronary artery disease were included if they had an elevated cTnI value and angiographically normal or minimal disease. Precipitating cause for elevated cTnI was determined by clinical evidences with regard to conditions known to be associated with troponin release. Result : 30 patients qualified, with an average age of 53 years (range 36-72). 47% of the patients were female. Cut-off value for elevated cTnI was 0.06 ng/ml and average of peak cTnI was 34.24 ng/ml. Peak CK-MB value was elevated in 10 patients. Increased cTnI was attributed to myocarditis in 5 patients, pericarditis in 1 patient, severe congestive heart failure in 5 patients, variant angina in 2 patients, severe myocardial bridge in 1 patient, rhabdomyolysis in 1 case and cerebral infarction in 1 patient. Tachycardia was the precipitating cause in 3 patients (sustained ventricular tachycardia, paroxysmal atrial fibrillation and scopolamin induced tachycardia for each), none of whom had hemodynamic compromise. 11 of 30 patients had no identifiable cause for a rise in cTnI value. 5 of 30 patients had regional wall motion abnormality of left ventricle. There were no MI at 24±12 weeks follow up. Conclusion : Although cTnI is a specific and sensitive marker of myocardial injury, an elevation in cTnI value may have a cause other than myocardial infarction or ischemia and may occur without significant angiographic coronary artery disease.

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Serological kinetics of adenylate kinase 3 in rat acute myocardial infarction

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Background and Object: Currently used serological markers for diagnosis of acute myocardial infarction are different in appearance time and specificity for myocardial infarction. So there is no ideal single serolocical marker for myocardial infarction. Adenylate kinase(AK) is a ubiquitous enzyme which contributes to the homeostasis of cellular adenine nucleotide pool. AK is abundant in myocardium, and we postulated that AK3 could be used as biochemical marker for the diagnosis of acute myocardial infarction(AMI).

Material and Methods: We constructed AMI rat model with ligation of anterior descending artery. We measured the concentration of serum AK3 in the AMI rat model by enhanced chemiluminescence(ECL) sandwich ELISA using monoclonal antibodies against recombinant AK3.

Result: The serum AK3 started to increase in 3 hours and reached peak at 6hours after ligation of coronary artery of rat. The significant elevation of AK3 was retained for 2 days ($p<0.05$).

Conclusion: AK3 is useful serological marker for AMI in rat.