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POSSIBLE EFFECTS AND CHANGES OF MYOCARDIAL INJURY MARKERS IN PERCUTANEOUS CORONARY INTERVENTION

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Objective Percutaneous Coronary Intervention (PCI) is a technique of cardiac intervention treatment in revascularisation of coronary vessel. The use of PCI in patients with coronary heart disease (CHD) had expanded dramatically over the past two decades. With the evolution of operator experience and equipment design, procedural mortality and the need of emergency coronary artery bypass graft surgery (CABG) was occasional, but procedural myocardial injury is still a common complication. The aetiology of post procedural myocardial injury is not clear. It is reported that side branch occlusion, transient coronary spasm, coronary microembolisation or intensity of procedure itself are responsible for increased levels of cardiac injury markers. Today, the cardiac markers of creatine kinase MB (CK-MB), cardiac troponin I (cTnI), cardiac troponin T (cTnT) and myoglobin (Mb) are used to assess the presence of myocardial injury. Many researchers have different opinions on the relationship between the increase of markers and prognosis. The prognosis includes adverse cardiac events such as sudden death, myocardial infarction and repeat revascularisation. Ischaemia modified albumin (IMA) is a new myocardial ischaemia marker in the medical field. However, it is less studied in our country. The authors compared the sensitivity and specificity of three markers, studied the factors associated with elevation of markers and the clinical outcomes after PCI and evaluated the possible effects on myocardium in revascularisation. The authors assessed the changes of plasma

IMA, cTnI and CK-MB that were monitored before and after PCI in patients with CHD.

Methods (1) Sixty patients with CHD who underwent PCI were enrolled, including 34 males and 26 females, with mean age of 58.32±10.33 years. All patients received clopidogrel (75 mg, PO, QD) or ticlopidine (250 mg, PO, twice daily) for more than 3 days before and 6–9 months after the procedure; aspirin (100 mg, PO, QD) and astovastain (10 mg, PO, QN) for more than 3 days before and all the life after the procedure; low- molecular heparin (5000 u, IH, twice daily) for more than 3 days before the procedure. Blood samples were obtained before and 30 min, 6 h, 24 h after the procedure, respectively. And in the same time, 30 patients with CHD undergoing CAG were also enrolled, including 24 males, six females, with mean age of 57.24±13.72 years. The authors obtained blood samples before and 30 min and 12 h after the procedure, respectively. The levels of plasma IMA, cTnI and CK-MB were measured in 90 patients. According to whether the increase of three markers or not, patients were divided into elevated group and normal group. The relative factors were analysed in both groups. (2) The patients with procedure of PCI were reviewed in the hospital and followed for 6 months outside the hospital for the incidence of cardiac events.

Results (1) In PCI groups, the plasma IMA levels elevated significantly from the baseline in 30 min after PCI in 54 of 60 patients. Median IMA levels were higher after PCI compared with the baseline (0.662±0.149 vs 0.338±0.088 ABSU, $p<0.05$), decreased to normal at 6 h and returned to the baseline. The levels of plasma cTnI began to increase 6 h (5.95±3.08 μg/l) after PCI in 19 of the 60 patients, reaching its peak levels in 12–24 h (6.07±2.73 and 6.16±2.70 μg/l, respectively). However, median cTnI levels were not significantly different 30 min after PCI ($p>0.05$). There were not significant difference in cTnI levels between before and after operation besides 30 min after PCI ($p<0.05$). There were no significant difference CK-MB levels between before and after operation ($p>0.05$). None of 60 patients sustained death and urgent target vessel revascularisation. There were also no obvious change in these three plasma markers in CAG groups. (2) The levels of elevated IMA and cTnI were correlated to total inflation times, dilated times and dilated pressure as well as the numbers of stents. The baseline characteristics such as ages, gender, hypercholesterolemia, hypertension were not related to the increase markers. (3) No cardiac events occurred in patients with and without elevated IMA and cTnI levels in the hospital and followed for 6 months outside the hospital.

Conclusions (1) Among the three elevated markers of myocardial damage undergoing PCI, IMA is the more sensitive markers than cTnI and CK-MB. But it is less specific. cTnI is the most specific marker among these three plasma markers. The measure of IMA in combination with cTnI may improve the sensitivity and specificity of plasma markers in identifying the myocardial injury after PCI. (2) When the levels of cTnI and CK-MB increase more three times than their normal levels, they are the predictors of major adverse cardiac events during and outside hospitalisation. But their increase levels are less two times than normal levels, they are not ones. Only the increase of IMA is not the predictor of major adverse cardiac events and is only one of early sensitivity markers of myocardial damage. (3) Before and after PCI the sufficient antiplatelet therapy, anticoagulation therapy and stable atherosclerotic plaque therapy as well as reduction of total inflation times, dilatation times and dilatation pressure during procedure are expected to reduce myocardial injury. And these can reduce cardiac events after PCI. (4) PCI may cause some damage to myocardium, but the damage was minor. The procedure causes no change in function of myocardium and it rarely results in significant adverse cardiac events. Therefore PCI is still one of the safe and effective revascularisation methods to patients with CHD.