DEATH OF ONE CASE FROM POSTOPERATIVE CARDIAC RUPTURE AFTER EMERGENCY PCI IN ACUTE INFERIOR MYOCARDIAL INFARCTION

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Objectives (1) Clinical Data: Patient, male, 67, was admitted to hospital due to the severe chest pain with vomiting and profuse sweating on 3 February 2011. He had history of hypertension and smoking. The blood pressure was 150/90 mm Hg. The ECG showed that the ST segments in II, III, and AVF leads had arched upward elevation for 0.3–0.5 mv. He was diagnosed as acute inferior myocardial infarction. The emergency coronary angiography showed that left main artery, anterior descending branch, and circumflex branch were normal, and the middle of right coronary artery had acute occlusions. The patient was given PCI operation successfully and antiplatelet therapy. The ECG and the echocardiogram were improved after PCI. In the fourth day after operation, he suddenly felt the chest distress, dizziness, and then faint and convulsion of the limbs when the patient passed a stool (the stool was not dry). The blood pressure could not be detected. So we gave cardiopulmonary resuscitation, but the patient died finally. The B ultrasound displayed large number of pericardial effusions. The cause of death of this patient was considered as the cardiac rupture.

Methods (2) Discussion: For acute myocardial infarction, the mortality in the hospital of emergency PCI condition has been dropped to about 10%. The main death causes are malignant arrhythmia, pump dysfunction after massive myocardial infarction or cardiac rupture. Compared with anterior myocardial infarction, cardiac rupture in inferior myocardial infarction is rarely possible due to the protective action of the diaphragm. This patient was given emergency PCI operation successfully, and had normal treatment after the operation. The patient died from the cardiac rupture in the fifth day of onset of illness, which was gone beyond our expectation.

Results The author analyses the possible reasons as follows: Firstly, although the patient was performed emergency PCI successfully, we considered that most of cardiac muscles in the infarcted area were still necrotic according to QS wave in ECG. The patient had not been the chest pain symptom in the past, which could infer that the culprit vessel of the patient before the myocardial infarction had not the high-grade stenosis and the ischaemic preconditioning process.

Conclusions The collateral circulation has not yet been open. After the unstable plaque rupture, the thrombosis blocked acutely. The far-end cardiac muscle had not protected and acute serious ischaemia existed. Most of cardiac muscles might be necrosis in the short period. The reperfusion therapy was made, but the effect was limited. Secondly, the patient was inferior myocardial infarction, and had smaller influences on the pump function. The patient with a comparatively good pump function had a higher pressure in the cardiac chamber during contraction period, and the risk of the cardiac rupture might increase. Thirdly, after the reperfusion therapy of the patient, the reperfusion injury would change the pathological process, and also the blood perfusion with the preferable infarct might influence the mechanism of the inflammatory cells and inflammatory factors which could accelerate the oedema and liquefaction of the necrosis, and make the peak time of the cardiac rupture ahead of time.