

中文題目：急性心肌梗塞併發嚴重僧帽瓣逆流之心因性休克－追縱五年之個案報告

英文題目：Cardiogenic shock caused by acute myocardial infarction complicated with severe mitral regurgitation – a case with five years' follow-up

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Introduction

Acute mitral regurgitation (MR) is one of the major complications of acute coronary syndrome. The mechanism of ischemic MR can be papillary muscle rupture or mitral valve tethering. Multiple therapeutic modalities that include accurate determination of the infarct related artery, timely percutaneous coronary intervention (PCI) and early application of intra-aortic balloon pump (IABP), can restore heart function, decrease myocardium injury, and reduce the severity of MR. On the other hand, ACE inhibitors and beta-blockers are also important during acute phase of treatment. Combined PCI following by adequate medical treatment can successfully rescue the case of cardiogenic shock that is caused by acute myocardial infarction complicated with acute, severe MR.

Case presentation

This 69-year-old man presented with cold sweating and cough with water-like sputum. He denied chest discomfort, left shoulder or jaw pain. The initial electrocardiogram showed ST depression in leads V2 to V5 and T wave inversion in leads V1 to V4. The chest X ray showed borderline cardiomegaly and pulmonary congestion. Biochemistry data revealed elevated cardiac enzymes. Under the impression of non-ST elevation myocardial infarction, dual anti-platelets and anti-coagulant therapy with heparin had been administered. However, progressive dyspnea was noted after a few hours and the chest X ray showed acute pulmonary edema. Therefore, early intervention strategy was chosen and cardiac catheterization was performed. The coronary angiography revealed two-vessel disease in which there was a 90% stenosis in middle segment of left anterior descending artery and total occlusion in proximal circumflex artery.

The PCI was performed upon the IABP support, and concluded with a bare-metal stent deployment in the left anterior descending artery lesion. After the PCI, the patient was admitted to the coronary care unit where echocardiography was performed and showed akinetic posterior segment of left ventricle, hyperkinetic anteroseptal segment, and a significant eccentric jet via the mitral valve, which was implicated as a complication resulting from posterior papillary muscle dysfunction. The cardiovascular surgeon was consulted immediately but the surgical intervention was declined due to the high estimated surgical risk. The infarct-related artery was suggested as the proximal circumflex artery therefore another PCI was performed where another bare-metal stent was successfully deployed.

After the second PCI, cardiac enzymes passed the peak at 51th hour of onset. His chest pain was

improved gradually as well. The EKG revealed gradual resolution of ST-T changes including ST elevation in leads III and aVF, and ST depression in leads V2~V4. Pulmonary edema was gradually improved. The IABP had been used for 5 days while his gradual recovery from cardiogenic shock. ACE inhibitor was initiated at low dose (captopril 6.25 mg tid) on the next day of IABP weaning. The patient was discharged on the 20th day after onset of AMI without any significant sequelae from shock. During the outpatient department follow up, the systolic murmur at apex was noticed milder and regression of MR from severe to mild degree was proved by echocardiography. The patient had been follow-up uneventfully for more than five years.

Discussion

In the primary PCI for this case, the infarct-related artery was initially assumed the non-occlusive left anterior descending artery and the total occlusion of circumflex was assumed as a chronic lesion. In most cases of cardiogenic shock, left anterior descending artery is considered more important than left circumflex coronary artery. Obviously, it is not true in this presented case. ST-elevation AMI of posterior wall can be recognized by ST depression in precordial leads, which was over-looked initially in this case. Posterior wall infarction is more likely complicated with posterior papillary muscle dysfunction that results in eccentric MR. The acute MR in turn causes abrupt reduction of cardiac output, surge of left ventricular filling pressure, left atrial pressure, and the wedge pressure that are manifested as acute pulmonary edema and cardiogenic shock.

The surgical intervention is no longer the first option for an ischemic mitral valve regurgitation in the 2015 American Association for thoracic surgery consensus guideline. The reason is that no strong evidence supports improved survival or elimination of dilated cardiomyopathy from the acute surgical intervention. The highest class of recommendation for the mitral valve surgery is reasonable at the time of coronary artery bypass grafting.

Concerning medical treatment for acute MR, ACE inhibitor is indicated as the first line medication by the AHA heart failure guideline. Even with relatively low dose, the usage of ACE inhibitor still exhibited substantial benefit for this case, in terms of reduction of degree of MR, control of left ventricular remodeling, and preservation of left ventricular function.

In conclusion, early PCI of the infarct-related artery and an early echocardiography examination are essential for successful management of cardiogenic shock caused by AMI with mechanical complications. A favorable long-term outcome requires optimization of post-AMI medications with good drug adherence that can be enhanced with an effective patient education.