Ischemic mitral regurgitation – could be different?

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Introduction: Secondary, ischemic mitral regurgitation (MR) is still common complication of acute myocardial infarction (MI), and the definition of ischemic MR is MR that occurs after MI with consequent, segmental wall motion abnormalities with structurally normal valve leaflets and chordae tendineae. Most common reason of ischemic MR is posterior papillary muscle dysfunction due its blood supply from only one artery – posterior descending branch.

Case report: 60-years-old female with previous history of arterial hypertension presented to emergency department (ED) because of chest pain. She complained that she had chest pain for about 2 weeks, precipitated by exercise, lasting up to 10 minutes. The day she came to the ED she had severe chest pain, lasting for 16 hours continuously. Her initial electrocardiogram showed no signs of ischemia or infarction. Due to high levels of troponin, creatine kinase, lactate dehydrogenase and regression of chest pain she was, initially, treated medicamentously. At day first of hospitalization echocardiography showed mild mitral regurgitation, mild left atrial dilatation (43mm), akinetic posterior and inferior wall, hypokinetic lateral wall and ejection fraction (EF) about 45%. Few days later, patient presented with heart failure (HF) (Killip III), soon after and Killip IV. After initial stabilization with vasopressors and inotropes coronary angiography was performed. In subtotal stenosis of proximal segment of circumflex artery and subtotal stenosis of proximal segment of right coronary artery were implanted stents. Echo showed severe mitral regurgitation (**Figure 1**) with elements of elevated pressures in pulmonary circulation (**Figure 2**), and EF about 30%. After 45 days patient left home with medical therapy according to guidelines for heart failure treatment. In following months she became „frequent flyer“ HF patient, with rapid progression of left ventricular dysfunction (**Figure 3**) and consequent right ventricular dysfunction (**Figure 4**) and soon after she died.

Conclusion: „Time is myocardium and myocardium is time“. If our patient came earlier in the hospital, percutaneous coronary intervention at the right time could prevent severe myocardial damage, ischemic MR with rapid progression of heart failure and death.

FIGURE 1. Severe mitral regurgitation, vena contracta 8mm, ERO 0.4 cm².

FIGURE 2. Indirect signs of elevated pressures in pulmonary circulation. Gradient across the tricuspid valve is about 60 mmHg, and right ventricular systolic pressure about 70 mmHg.

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FIGURE 3. Progression of left ventricular dysfunction (PLAX), dilatation of ventricle and EF about 25%.

FIGURE 4. Progression of (indirect) pulmonary hypertension, gradient across the tricuspid valve above 100 mmHg.