STUDIUM PRZYPADKU / *CLINICAL VIGNETTE*

Echocardiography in the catheterisation laboratory

Echokardiografia w pracowni hemodynamiki

Misa Fister[1], Ivan Knezevic[2], Spela Stupnik[3], Marko Noc[1]

[1]Centre for Intensive Internal Medicine, University Medical Centre, Ljubljana, Slovenia
[2]Department of Cardiovascular Surgery, University Medical Centre, Ljubljana, Slovenia
[3]Department of Anaesthesiology, University Medical Centre, Ljubljana, Slovenia

A 69-year-old, previously healthy man complained about mild chest discomfort for the preceding week. The discomfort, mainly in the epigastric area, intensified during the previous 10 h, and it was followed by a sudden collapse. Upon arrival of the local physician, the patient was conscious and hypotensive. The 12-lead electrocardiogram (ECG) was consistent with inferolateral ST-elevation myocardial infarction (STEMI) with the presence of Q waves in the inferior leads (Fig. 1). The alerted primary percutaneous coronary intervention (PCI) centre advised treatment with acetylsalicylic acid only. Because of presumably subacute STEMI and cardiogenic shock of unknown mechanism, our routine pretreatment before primary PCI with unfractioned heparin and P2Y12 inhibitor was withheld. The patient was transported directly to our catheterisation laboratory about 70 km away. On admission to the catheterisation laboratory, the patient was diaphoretic with arterial pressure of 70/40 mm Hg, heart rate of 120 bpm, and pulse oximetry saturation of 95% on Ohio mask. Transthoracic echocardiography (TTE) was immediately performed, revealing a large pericardial thrombus-coagulum causing cardiac tamponade (Fig. 2). A diagnosis of contained free wall rupture was made and a cardiac surgeon alerted. Because of the unstable haemodynamic situation and imminent danger of complete left ventricular (LV) rupture, coronary angiography was withheld, and the patient was transferred directly to the operating room for tamponade resolution and LV wall repair. The femoral artery and vein were cannulated, and peripheral veno-arterial extracorporeal membrane oxygenation (VA ECMO) was initiated. This was followed by removal of a very large thrombus-coagulum and blood from the pericardial sack (Fig. 3). A small defect located in the lateral LV wall was then exposed (Fig. 4) and successfully repaired by polytetrafluoroethylene patch and bio glue. The patient was uneventfully weaned off the VA ECMO and mechanical ventilation. The tracheal tube was removed the following day, and the patient fully recovered. Peak cardiac troponin I increased only to 14 (normal < 0.04) μM/L indicating a relatively small myocardial necrosis. Coronary angiography (CAG) before hospital discharge demonstrated ostial occlusion of the obtuse marginal branch without additional obstructive coronary artery disease (Fig. 5). PCI of the obtuse marginal branch, which was the culprit for the rather small and obviously completed infarction and life-threatening contained rupture, was not performed. Mechanical complications of are nowadays in the era of timely and effective mechanical reperfusion by primary PCI very rare and frequently diagnosed with significant delay. Immediate diagnosis is important because open heart surgery, rather than primary PCI, is the treatment of choice. This was the case in our patient, with contained free rupture that was diagnosed by TTE. We believe this examination is very important in haemodynamically compromised patients transported directly to the catheterisation laboratory by the "STEMI fast track", and a portable echocardiography device should become standard equipment in "24-7" primary PCI centres. We also believe that in such cases aggressive prehospital anti-aggregation and anticoagulation prior to primary PCI should be withheld because of possible mechanical complications and the subsequent need for immediate open heart surgery.

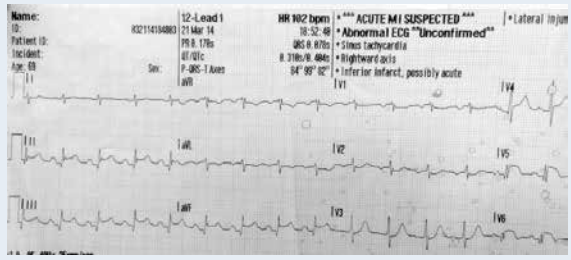

Figure 1. 12-lead ECG recorded upon arrival of local physician to the scene, indicating inferolateral ST-elevation myocardial infarction

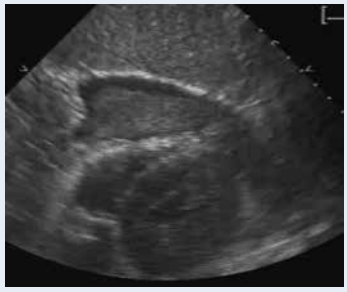

Figure 2. TTE (subcostal view) recorded immediately upon the arrival of the patient to the catheterisation laboratory, using a portable device. Large pericardial thrombus-coagulum causing cardiac tamponade was documented

Figure 3. Large thrombus-coagulum removed from pericardial sack in a surgical bowl (diameter 13 cm)

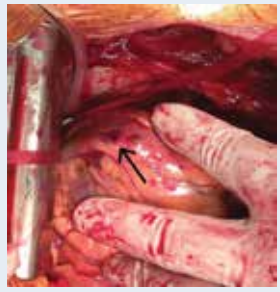

Figure 4. Following removal of large pericardial thrombus-coagulum, a small LV wall rupture was seen (arrow)

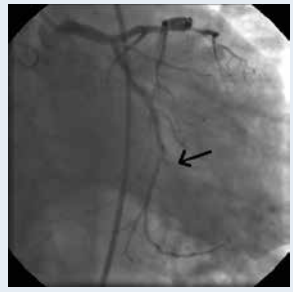

Figure 5. CAG before hospital discharge (RAO view) showing thrombotic ostial occlusion of obtuse marginal branch (arrow), which was the culprit lesion for the infarction associated with contained LV free wall rupture

Address for correspondence:
Marko Noc, MD, PhD, Centre for Intensive Internal Medicine, University Medical Centre, Zaloska 7, 1000 Ljubljana, Slovenia, tel/fax: +386 1 522 22 96, e-mail: marko.noc@mf.uni-lj.si

Conflict of interest: *none declared*