A 72-year-old man with sudden chest pain, which had started two hours ago, moderate in intensity and character of the oppression, came to Emergency Department (ED). He was hypotensive (95/60 mmHg), with cyanotic lips, but not breathless, and was having a heavy vertigo. By physical examination, there were normal breathing sounds, normal heart rhythm, mild tachycardia (92/min), and there was a murmur over the Erb’s point. Arterial haemoglobin oxygen saturation was 90.6%. Abdominal and extremities’ physical findings were normal. Routine laboratory examination revealed elevated D-dimers (6993 ug/L), troponin I (0.274 ug/L), slightly reduced red blood cells count (4.11x10^12/L), haematocrit (0.4), haemoglobin (139 g/L), platelets (149x10^6/L) and potassium (3.7 mmol/L). Other laboratory parameters were within normal range. Electrocardiogram showed sinus rhythm, 92/min., and Q in the III lead. 

The man was diagnosed with hemodynamic shock, but the cause of the shock was unclear. Differential diagnosis assumed a cardiogenic, a haemorrhagic or a neurologic pathogenesis. Heart ultrasound was performed for evaluation of potential sources of cardiogenic emboli. The echocardiography showed dilated right ventricles (50 mm), severe tricuspid insufficiency (Vmax. 3.21 m/s, PGmax. 41.3 mmHg), with estimated severe pulmonary hypertension (60 mmHg). Also, there were compressed left ventricles (39 mm), with preserved systolic function, and “kissing phenomena”.

Thus pulmonary embolism (PE) was strongly suspected and CT angiography of pulmonary arteries showed massive PE. A half an hour following alteplase and unfractionated heparin administration, arterial pressure started to normalize, and repeated heart echocardiography showed normalization of the ventricular dimensions, with reduction of the pulmonary hypertension. The patient began feeling well, while the vertigo and the chest pain disappeared. During the next few days his condition completely restituted. By that particular case of not typically presented massive pulmonary embolism, the heart echo facilitated the diagnostic procedure, and justified the necessity of heart ultrasound use in ED once again.1-5.

KEYWORDS: pulmonary embolism, heart echocardiography, cardiogenic shock.

Literature