Acute pulmonary embolism mimicking STEMI. Do the potential benefits of pulmonary artery angiography support the decision to perform the procedure?

Ostra zatorowość płucna naśladująca STEMI. Czy potencjalne korzyści angiografii płucnej ułatwiają decyzję o leczeniu zabiegowym?

This case report concerns a 68 year old male with type 2 diabetes, stage 3 hypertension, hypercholesterolemia, myocardial infarction (MI) 20 years ago. He was admitted to the catheterization laboratory with suspected acute inferior wall MI. Angiography of pulmonary arteries revealed massive thrombosis.

Introduction
The incidence of pulmonary embolism (PE) is estimated at 1 in 1000 per year. Most PE cases occur in patients with decreased mobility and with illnesses favoring the occurrence of clotting disorders [1]. PE is one of the most common reasons for the sudden onset of dyspnea. Timely diagnosis of PE and the introduction of proper treatment is crucial for patient survival and prognosis.

Case report
We are presenting a case of a 68 year old male with type 2 diabetes (diagnosed within last 12 months), stage 3 hypertension, hypercholesterolemia, obesity (height – 170 cm, weight – 88 kg, body mass index – BMI – 30.6 kg/m²), benign prostate hypertrophy, and coronary artery disease – after myocardial infarction (MI) 25 years ago – with no medical documents, who has never had an invasive diagnostics of coronary disease. The individual was receiving permanent medication consisting of acetylsalicylic acid 75 mg o.i.d, bisoprolol 5 mg o.i.d., perindopril 5 mg o.i.d, simvastatin 20 mg o.i.d., doxazosin 2 mg o.i.d. and metformin 500 mg t.i.d. The patient interview revealed that the first symptoms of chest pain appeared 4 days prior to the admission. The pain was sporadic, sometimes coinciding with different phases of the breathing cycle. It often appeared suddenly, was not associated with physical effort and often accompanied by dyspnea. It was alleviated partially by non-steroidal anti-inflammatory drugs use. Due to recurrent chest pain episodes the patient decided to seek medical help at an emergency department (ED). At the ED the patient was qualified for invasive angiography because of chest pain described by the admitting ED doctor as stenocardial. ECG performed at the ED showed sinus rhythm with heart rate of 100 bpm, elevated ST segments in leads II, III, and aVF (by about 0.5 to 1 mm) with pathologic Q waves in these leads and ST depression of 0.5 mm in II and aVL (Fig. 1). Laboratory blood analysis showed increased troponin I level of 0.67 µg/l (ULN <0.01 µg/l). Electrolytes concentrations and blood morphology parameters were within normal ranges. With the suspicion of the inferior wall STEMI the patient was qualified for the invasive diagnostics. While in the ED the patient was given 600 mg of clopidogrel p.o. and 5000 units of heparin i.v.

On admission to our Clinic the patient was in severe general condition, with a heart rate of 125 bpm and blood pressure of 100/40 mm Hg. Crepitations were present at the base of lungs fields on auscultation. Both calves were gently oedematous. In the echocardiographic examination (poor image quality) performed on the spot left ventricle was not enlarged (left ventricle diastolic diameter - LVEDd – 52 mm) with slightly compromised systolic function (akinesia of left ventricle inferior wall, left ventricle ejection fraction - LVEF – 48%), left atrium was slightly enlarged (43 mm), minor degree mitral valve and moderate tricuspid insufficiency was noted, right ventricular systolic pressure was estimated at 40 mm Hg, right ventricle was not enlarged (40 mm in the 4 apical 4 chamber presentation) and no features of PE were observed. In the coronography the left coronary artery revealed no significant narrowings -while the right coronary artery was chronically occluded (Fig. 2). Due to falling blood pressure and clinical signs of shock intravenous catecholamines and fluid replacement were introduced. Due to poor effects of the treatment and subsequent cardiac arrest resulting from electromechanical dissociation the decision to perform a pulmonary angiography during cardiopulmonary resuscitation (CPR) was undertaken. Pulmonary angiography sho-
wed the presence of massive thrombi at the bifurcation of the pulmonary trunk as well as in the right and left pulmonary arteries. Occlusion of the left upper lobe artery and massive thrombosis of the left lower lobe artery with significantly impaired blood flow (Fig. 3). The patient was given a bolus of 50 mg of rtPA (alteplase) i.v. with a subsequent continuous i.v. heparin infusion of 1600 IU/kg/h with CPR including mechanical ventilation being simultaneously performed. Pulse and pressure returned after 65 minutes of resuscitation. In severe but stable condition the patient was transported to the intensive care unit. The patient’s condition improved gradually. Ultrasound examination of the lower extremities vessels showed massive deep vein thrombosis in the left popliteal and femoral vein. No significant pathology was observed in abdominal ultrasonography. The venous thromboembolic disease was diagnosed as idiopathic. Warfarin was introduced into therapy. The individual was extubated on the third day, rehabilitated and after 18 days of hospitalization discharged without neurological deficits.

Discussion
Several factors made the proper diagnosis of PE in the patient come late but hopefully right on time. The history was taken by physicians at ED cursorily, the chest pain features and dyspnea were underestimated, crucial tests for the dyspnea diagnosis including serum D-dimer and natriuretic peptides (brain natriuretic peptide - BNP or N-terminal prohormone of brain natriuretic peptide - NT-proBNP) concentration measurements were omitted. Moreover, troponin increase is tended to be strictly related to MI by many physicians, especially when ischemic ECG changes are identified. In the presented patient permanent ST segments elevation in inferior wall leads resulted from previous MI resulting from the right coronary artery occlusion that occurred 20 years ago which blurred the clinical presentation. Misleadingly, massive PE discovered in pulmonary artery angiography caused neither right ventricular dilation nor its severe dysfunction in echocardiography. Such situations happen when PE is evolving gradually over long time like in our patient with idiopathic deep vein thrombosis.

PE may have atypical clinical manifestations what changes the diagnostic path adversely affecting the prognosis of the patient and delaying the appropriate treatment introduction including thrombolysis implementation [2,3]. According to the Polish National Register of Pulmonary Embolism - ZATPOL the highest mortality (27.3%) occurs in the group of patients with so-called “unbelievable diagnosis” where underlying disease progressed nonspecifically with the mask of other disease like in the case we have described [4].

The immediate decision of a pulmonary artery angiography in the presented case eventually saved the patient’s life. What should be noted, pulmonary artery angiography is not performed routinely in cases of shocks of unknown reasons even in the catheterization laboratories and is often done unwillingly by experienced operators.

The procedure was shown to be associated with an increased risk of mortality in unstable patients [5]. In our case patient’s condition excluded the possibility of performing a multislice spiral computed tomography while echocardiography did not support the diagnosis of PE inclining to the thrombolytic therapy use. Despite this, the clinical presentation of shock and subsequent cardiac arrest due to electromechanical dissociation, the history of chest pain and dyspnea were suggestive for the experienced physician performing coronarography to exclude PE. Such a strategy of more common use of pulmonary artery angiography might improve the prognosis and decrease mortality.

Moreover, thrombolytic treatment increases the risk of bleeding what concerns physicians and delays the therapy introduction [6,7]. Nevertheless, there are a few other methods that can be used in hemodynamic instability also improving the outcome. Kuo et al. confirmed that catheter-directed therapy (CDT) is relatively safe and highly effective tool in the treatment of massive PE. There are no randomized studies but available data indicate that the number of complications of CDT is lower than in systemic thrombolsy [8]. European guidelines for the treatment of PE recommend embolectomy and thrombus
fragmentation by CDT in the proximal pulmonary arteries as an alternative to surgery in high-risk patients when thrombolysis has proven to be ineffective or contraindicated (class II b, level B). U.S. guidelines take into account the possibility of using CDT for thrombus fragmentation and embolectomy on pair with surgery in the course of massive PE (class of recommendation II a, level C) in the case of contraindications to thrombolysis or failure of its action [9].

Summing up, despite the dynamic development of medicine the problem of appropriate diagnosis of PE and efficient treatment is still very important. The available measures to diagnose and cure PE should be used more eagerly because despite the possible complications the risk to benefit ratio is positive [10].

Bibliography
4. ZATPOL – Ogólnopolski Rejestr Zatorowości Płucnej: www.zatpol.pl