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CASE REPORT

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Submassive pulmonary embolism as a mask of acute coronary syndrome

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Abstract

The paper presents a case of a 77-year-old man, who was admitted to hospital suffering from chest pain. The ECG showed horizontal ST segment depression in the V4–V6 leads. Non-ST segment elevation acute coronary syndrome was diagnosed. However, transthoracic echocardiography revealed signs of pulmonary embolism. The present case indicates the need to consider pulmonary embolism in the differential diagnosis of acute coronary syndromes and underlines the role of echocardiography, which should be performed at the earliest possible stage.
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Key words: submassive pulmonary embolism, acute coronary syndrome

Introduction

Clinical symptoms of pulmonary embolism (PE) are not specific and are often the cause of mistaken diagnosis [1, 2]. The chest pain and ST segment changes in the precordial leads observed in PE may lead to incorrect diagnosis of acute coronary syndrome. It is possible that the treatment used will be unsuitable and the invasive diagnosis of coronary arteries unnecessary [3].

The present paper reports a case of a cardiologically healthy patient who was diagnosed on admission to hospital with non-ST segment elevation acute coronary syndrome. Echocardiography examination performed on the second day of hospitalisation verified the tentative diagnosis and resulted in changes in the treatment strategy.

Case description

A 77-year-old man was admitted to the Department of Internal Diseases at a District Hospital in Pińczów on account of recurrent chest pain at rest and dyspnoea. The anginal pain appeared three days earlier (CCS III). On the day of admission he presented pain at rest (CCS IV), accompanied by moderate dyspnoea. The patient had no previous record of heart trouble and had not been treated cardiologically up to that point. He had been diagnosed with arthrosis and benign prostate hypertrophy but had not been taking any drugs on a regular basis.

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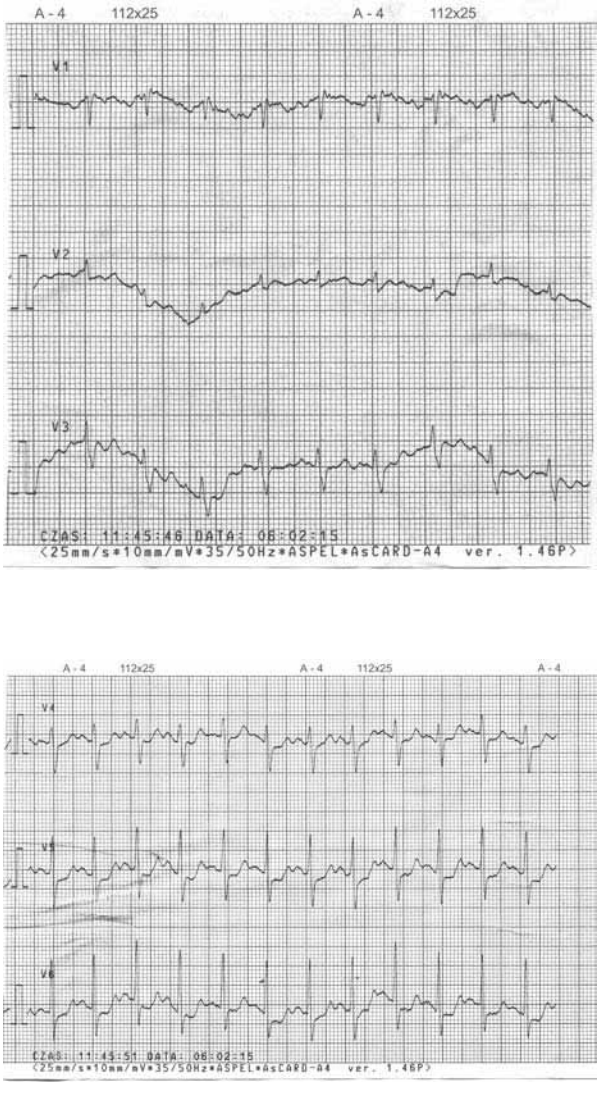

Figure 1. ECG — precordial leads (performed on admission).

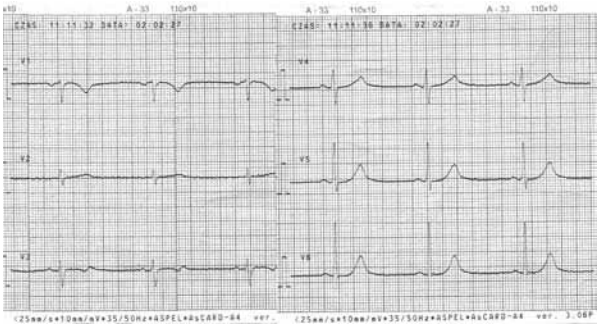

Figure 2. ECG — controlled examination.

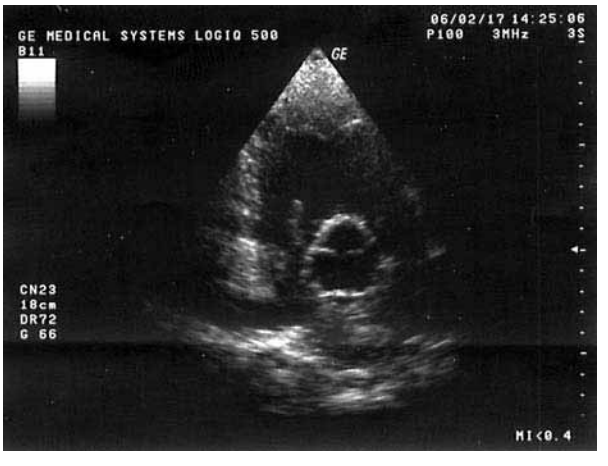

Figure 3. Transthoracic echocardiographic examination. Short axis view. A mobile structure in the right atrium protruding into the right ventricle in diastole.

Physical examination on admission revealed tachycardia of 130 bpm, elevated blood pressure (170/80 mm Hg) and varicose veins in the legs (without clinical signs of thrombosis). ECG revealed sinus tachycardia, incomplete right bundle branch block and horizontal ST segment depression (up to 2 mm) in the V4–V6 leads (Fig. 1). The markers of myocardial necrosis were not elevated (CPK MB 24 U/l, troponin T 0.01 ng/ml). No other biochemical abnormalities were detected. Non-ST segment elevation acute coronary syndrome was diagnosed. Early pharmacological treatment was used (Aspirin 1 × 100 mg, Aclotin 2 × 250 mg, Clexane 2 × 60 mg *s.c.* Metocard 2 × 25 mg, Accuporo 1 × 10 mg, Zocor 1 × 20 mg). No abnormalities were found in the follow-up ECG examination (Fig. 2).

The results of a chest X-ray, which was performed on the second day, showed no abnormalities apart from a slightly enlarged intermediate artery (22 mm). Transthoracic echocardiography revealed a mobile structure in the right atrium about 23 mm long (morphologically corresponding to a thrombus), protruding into the right ventricle in diastole (Figs. 3, 4). Moreover, features of right ventricle pressure overload were detected: right ventricle dilatation (41 mm) with segmental wall motion abnormalities, functional tricuspid regurgitation (grade II/III) and significant pulmonary hypertension of 67 mm Hg + central venous pressure (CVP) (Fig. 5). There was no regional impairment of left ventricular contraction (ejection fraction 60). The echocardiographic image was suggestive of PE. The diagnosis was confirmed by spiral computed tomography (Fig. 6). The scan demonstrated a contrast defect in the lumen of the left pulmonary artery extending into the lower lobe artery as well as a similar smaller contrast defect in the lumen

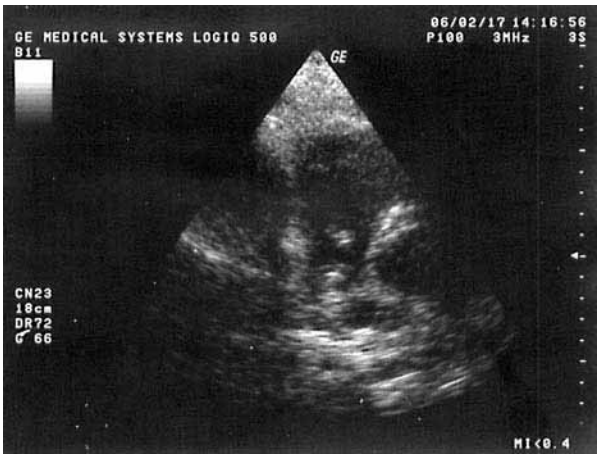

Figure 4. Transthoracic echocardiographic examination. Modified short axis view. A mobile structure in the right atrium.

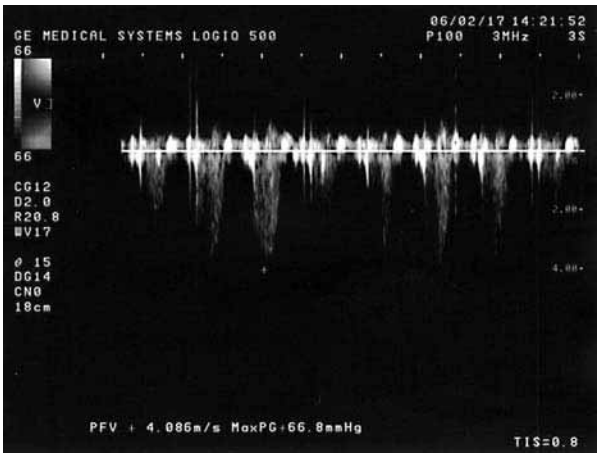

Figure 5. The Doppler ultrasound. Tricuspid regurgitation. Right ventricle systolic pressure — 66.8 mm Hg + CVP.

of the right pulmonary artery extending into the intermediate artery. The diagnosis of submassive PE was made, and the therapy based on a tienopiridin derivative was discontinued; low weight heparin was replaced with heparin infusion (activated partial thromboplastin time controlled). This method of treatment was continued for 7 days, accompanied by anticoagulant therapy for the last 4 days. During his stay in hospital the patient was hemodynamically stable and, apart from the day of admission, did not complain of chest pain. The follow-up echocardiography was performed on the 8th day. The presence of an organised thrombus (12 × 7 mm) attached to the lateral wall of the right atrium (Fig. 7), an essential decrease in right ventricle dimensions (37 mm), a decrease in tricuspid insufficiency (I/II degree) and a reduction in

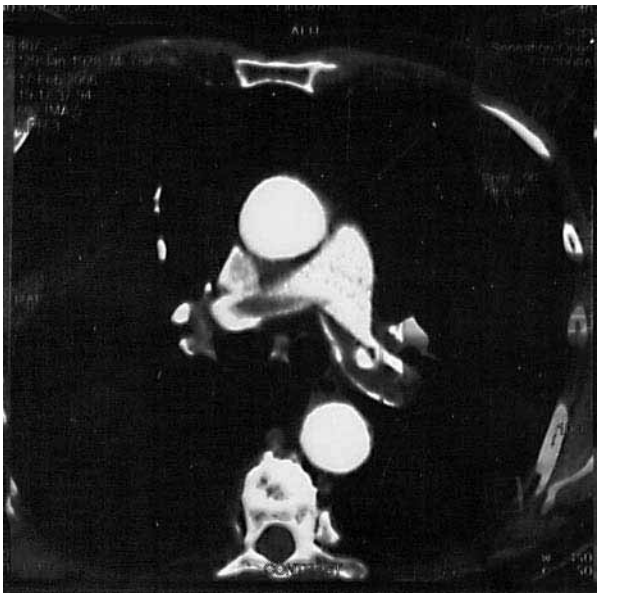

Figure 6. Spiral computed tomography. Thrombi in right and left pulmonary arteries.

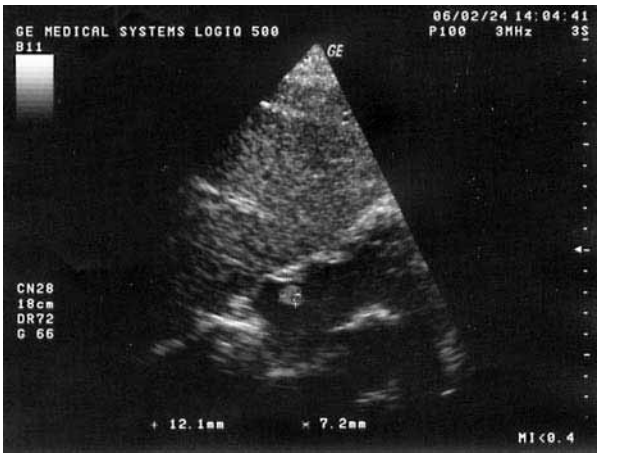

Figure 7. Transthoracic echocardiographic examination — controlled examination; substernal projection. Short axis view. A mobile structure in the right atrium protruding into the right ventricle in diastole. A small organised thrombus in the right atrium.

right ventricle systolic pressure: 37 mm Hg + CVP were observed (Fig. 8).

Ultrasonography revealed organised thrombi in the proximal deep veins, namely the popliteal vein, deep femoral and left common femoral vein. The patient was discharged home in good condition with the recommendation to take oral anticoagulant drugs for a minimum period of 6 months. A follow-up echocardiographic examination was advised after 3 months.

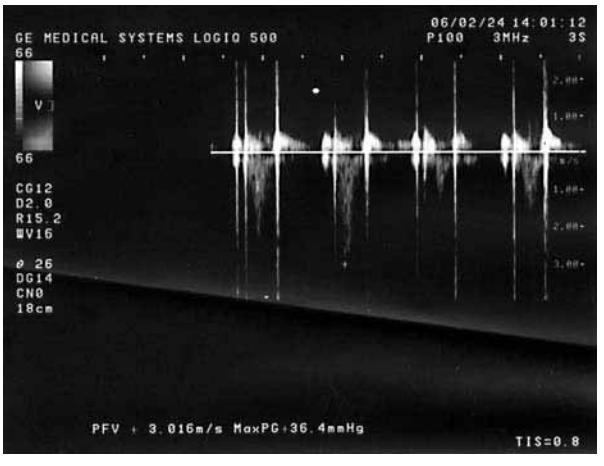

Figure 8. The Doppler ultrasound examination (controlled). Tricuspidal regurgitation. Right ventricle systolic pressure — 36.4 mm Hg + CVP.

Summary

The incidence of a right heart thrombus in patients suffering from PE is about 4% [1, 4]. Long right atrium thrombi of considerable mobility are almost always associated with massive pulmonary artery embolism and may significantly increase the risk of death [5]. The available data suggest that finding a movable mass of thrombus morphology in the right atrium during echocardiography is linked to a high early mortality rate up to 48% during the first 8 days from diagnosis [1]. It is therefore of essential importance to make a proper diagnosis at an early stage.

There were two risk factors in the case of this particular patient, these being the varicose veins in the legs and the patient's advanced age, as risk increases with age. ECG changes, including sinus tachycardia and ST segment depression in the precordial leads, are frequently found in PE, but their specificity is limited. They may wrongly suggest an acute coronary syndrome [1].

Echocardiographic examination makes it possible to carry out bedside diagnostics, establish the hemodynamic consequences and quickly stratify the risk [6]. The following were the echocardiographic features of PE in the case reported: the presence of a thrombus in the right atrium, functional tricuspid insufficiency, an increased rate of tricuspid regurgitant flow (4 m/s) and the very specific sign of regional impairment of right ventricle contraction (the McConnell symptom).

Thrombi were not detected in the pulmonary arteries because of the technical difficulties in obtaining a proper acoustic window in the short axis view. In spite of the fairly unambiguous echocardiographic findings the decision was made to perform spiral computer tomography scanning, which confirmed the presence of large thrombi in the right and left pulmonary arteries.

Patients suffering from PE, with normal blood pressure or without symptoms of cardiac shock but with segmental wall motion abnormalities of the right ventricle, have a worse prognosis than others. This is the basis of a separate submassive PE [1, 6].

The diagnosis of submassive PE made the choice of treatment difficult [6]. This stems from the controversy over the proper treatment strategy of submassive PE [7–9]. In one of a control group of tests on PE urgent intervention was rarely required for patients treated with both heparin and fibrynolysis, but the survival rate did not go up [10]. It should be noted that the risk of serious hemorrhage associated with fibrynolysis is twice as high as in the case of treatment based only on the use of heparin [11, 12].

It is therefore commonly acknowledged that fibrynolysis should be administered to patients with a clinical massive PE. As far as our patient was concerned, fibrynolytic therapy could not be excluded in the event of his clinical condition deteriorating. For this reason it was decided to replace the low molecular weight heparin with unfractionated heparin infusion, despite the similarities in effectiveness and safety profile, because the latter is a short-acting drug and provides the possibility of an easy elimination of the anticoagulant effect.

The role of troponin and brain natriuretic peptide in the risk stratification of patients with PE has been demonstrated. The lack of elevation of myocardial necrosis markers is a factor in the good prognosis in this case.

The case presented shows the importance of echocardiographic examination, which verified the tentative diagnosis, made the originally planned coronary angiography unnecessary, altered the strategy of treatment and, in all likelihood, positively influenced the patient's health.

The case indicates that in clinical practice it is essential to bear in mind the fact that not every chest pain accompanied by electrocardiographic ST-T segment alterations is an acute cardiac syndrome, and not every pulmonary embolism with synchronic thrombi in the large pulmonary arteries leads to significant hemodynamic symptoms.

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