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Pulmonary embolism resulting in a pleural Dressler-like syndrome

Zatorowość płucna powikłana opłucnowym zespołem podobnym do zespołu Dresslera

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Abstract

Pulmonary embolism was reported as one of possible complications after implantable cardioverter-defibrillator (ICD) lead implanting procedures. Pleural effusion, with fluid bloody in appearance and with predominance of granulocytes, can be a common resolution of lasting pulmonary embolism. In this paper we present a case of ICD lead exchange procedure causing pulmonary embolism and pleural effusion, with bloody lymphocytic fluid as its consequence, with good response to corticosteroids treatment.

Key words: acute pulmonary embolism, ICD, pleural effusion

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Introduction

Pulmonary embolism (PE) is a major clinical problem in clinical practice, though its exact epidemiology is difficult to determine [1]. Among many predisposing factors for venous thrombosis, an implantable cardioverter-defibrillator (ICD) device is not considered [2]. We present a case of a lead replacement procedure complicated by PE, possibly caused by blood clot detached from the removed electrode. PE then caused an infarction of the lung and a pleural Dressler-like syndrome. This case deals with less obvious cause, presentation and course of PE, all in one patient.

Case report

A 32-year-old man with hypertrophic cardiomyopathy diagnosed at the age of 14, with right ventricle outflow obstruction, with an ICD device implanted in primary prevention, underwent a replacement of the defibrillating electrode due to its damage five weeks prior to the admission to our department. Before lead removal a possible blood clot on the defibrillating electrode was found in echocardiography examination. Ten days after the lead replacement procedure the patient started to feel chest pain and dyspnea with cough; his fever spiked to 39 °C. His condition worsened despite ambulatory treatment with oral amoxicillin/clavulanic

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acid. He was admitted to cardiology department with a suspicion of infective endocarditis. He received intravenous ceftriaxon, clarithromycin and vancomycin. All blood cultures returned negative; in three echocardiography examinations, including transesophageal one, there were no signs of bacterial vegetation. Despite the treatment with antibiotics no clinical improvement was noticed after 14 days and radiological progression in chest X-ray was observed — peripheral consolidations in the basal segments of left lung were now accompanied by pleural effusion. A chest computed tomography (CT) scan was performed with conclusion of lung abscess and pleural empyema, so patient was referred to our pulmonology department.

Our radiologist, after reviewing the CT scan, found a filling defect within the left inferior pulmonary artery and its segmental branches with pulmonary infarction and pleural fluid (Figure 1), so pulmonary embolism was diagnosed. In transthoracic echocardiography both right heart chambers were dilated, there were evidences of pulmonary hypertension — tricuspid valve pressure gradient was 40 mmHg, with shortening of acceleration time with a visible notch. Patient's electrocardiogram (ECG) is shown in Figure 2. Patient was started on enoxaparin 1 mg per kg two times a day. More than 4000 mL of pleural fluid was obtained in series of punctures — bloody appearance, with characteristics of exudation, 85% lymphocytes and 15% eosinophils, no neoplasm cells in the fluid, fluid cultures for bacteria and tuberculosis were negative. Because the effusion was constantly increasing, a suspicion of pleural lymphocytic inflammation was made (a pleural Dressler-like syndrome) and patient was commenced with intravenous methylprednisolone 40 mg daily. After two weeks of treatment almost complete regression of pleural abnormalities was observed in a control CT scan and he was switched to oral prednisone 30 mg daily. Pulmonary embolism was treated chronically with oral rivaroxaban.

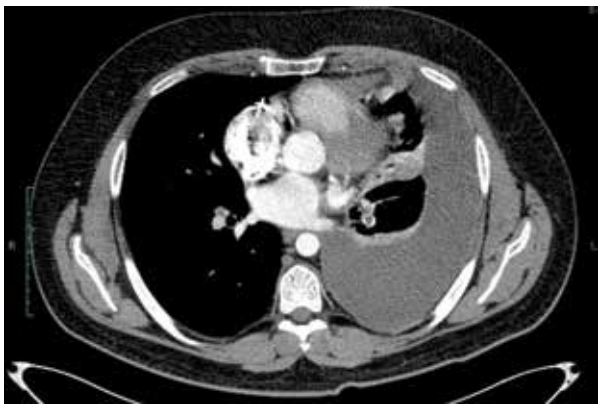

Figure 1. Chest CT. Filling defect in the left pulmonary artery; infarction of left lower lobe; fluid in the left pleural space

Discussion

An ICD device is recommended for primary prevention in patients with hypertrophic cardiomyopathy with moderate to high 5-year risk of sudden cardiac death [3]. Among many complication of this procedure, PE has been reported as highly undermined — a study showed that in 4-month observation, among 185 patients, 25% had a lead-related thrombi, one had a clinically significant pulmonary embolism [4]; these observations are supported by many case reports [5, 6]. No exact data indicate the need of routine transesophageal echocardiography to detect lead-related

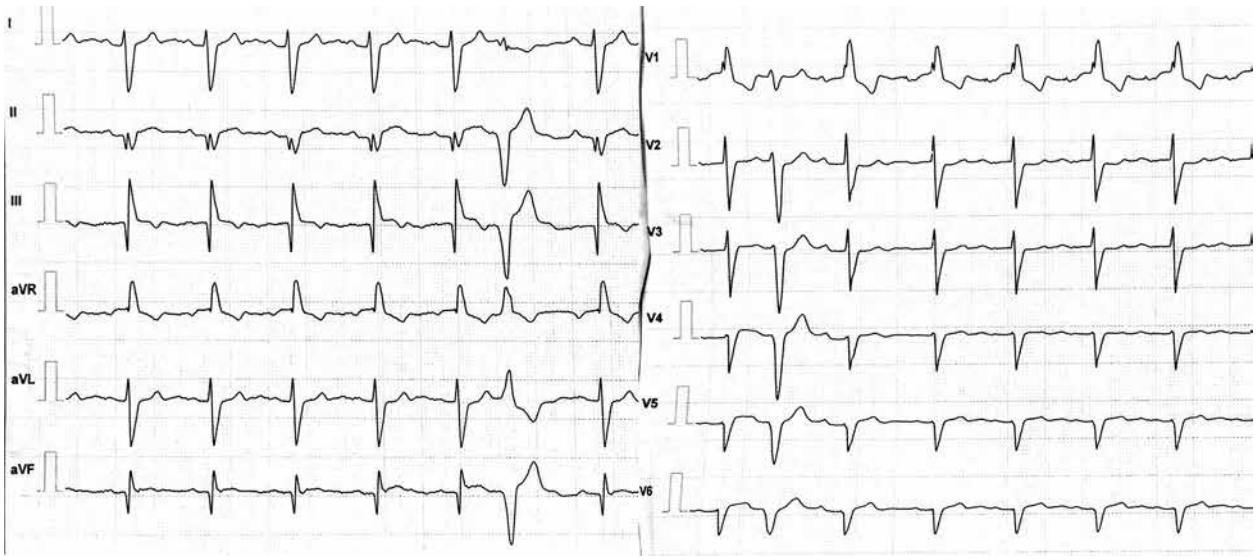

Figure 2. ECG. Sinus rhythm 70 bpm, single ventricular extrasystole. Dextrocardia. First-degree atrioventricular block. Right bundle branch block. Pathologic Q-waves in leads II, III, aVF and V4–V6

thrombi and of anticoagulation treatment in this group of patients [5]. It seems most cases remain asymptomatic and self-resolving [7].

When dealing with pleural effusion, many etiological factors are considered. PE in a large study was the cause in only 1.6% of all cases [8], although pleural effusion is present in 30 to 50% of patients with PE. The cytological characteristics of pleural fluid in PE is mostly bloody in appearance, with dominance of granulocytes, but in about 40% of lymphocytes; also fluid eosinophilia should be considered as PE-suggesting [9]. In each case neoplasm and infections need to be ruled out. What we believe is that the infarction of lung tissue was the promoting factor for lymphocytic fluid and therefore inflammation, which led to recurring pleural effusion despite many pleural punctures.

Eventually corticosteroids were used — we were basing on the same idea that stands for treating Dressler syndrome. Although impressively effective in this case, we do not recommend it as routine management; further investigation in the field is needed.

Conclusion

Pulmonary embolism should be suspected in patients after all sorts of lead-related procedures, even when symptom presentation is poor or unspecific.

Conflict of interest(s)

Authors report no conflict of interest.

Streszczenie

Zatorowość płucna była opisywana jako możliwe powikłanie zabiegów wszczepiania elektrod implantowalnego kardiowertera-defibrylatora (ICD). Wysiękowy krwisty płyn to dość częste następstwo długo trwającej zatorowości, a charakter wysięku jest najczęściej granulocytarny. W pracy omówiono przypadek powikłanego zatorowością zabiegu wymiany elektrody ICD oraz limfocytarnego wysięku opłucnowego jako następstwa ostrej zatorowości leczonego glikokortykosteroidami.

Słowa kluczowe: ostra zatorowość płucna, ICD, wysięk opłucnowy

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