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CASE REPORT

Being Immobilized Involves A High Risk of Pulmonary Embolism

Hareketsiz Kalmak Pulmoner Emboli İçin Yüksek Risk İçerir

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ABSTRACT

Introduction: The clinical symptoms of pulmonary embolism are diverse and in some patients, pulmonary embolism can be easily overlooked and cause serious clinical consequences.
Case Report: This report describes a case of acute pulmonary embolism whose first symptom was presyncope. A 23-year-old male patient presented with presyncope and dyspnea. Acute coronary syndrome and neurological disorders such as stroke and seizures were excluded by clinical history and dynamic changes in the electrocardiogram. After the diagnosis was completed with computed tomography pulmonary angiogram, the severity of acute pulmonary embolism was evaluated and then the fibrinolytic drug was given to the patient.
Conclusion: This case is of guiding importance for the early diagnosis and treatment of these patients who had pulmonary embolism after long-term immobilization, which poses a potential risk for venous thromboembolism. Cardiopulmonary diseases should be highly suspected in patients with problems with basic vital signs. After evaluating the possibility of pulmonary embolism and performing a D-dimer scan, a computed tomography pulmonary angiogram should be performed as soon as possible. Additionally, the critical degree of pulmonary embolism should be assessed and appropriate reperfusion and anticoagulation therapy should be administered.

Keywords: Emergency department, hospitalization, immobilization, pulmonary embolism

ÖZ

Giriş: Pulmoner embolinin klinik semptomları çeşitlidir ve bazı hastalarda pulmoner emboli kolayca gözden kaçabilir ve ciddi klinik sonuçlara neden olabilir.
Vaka Raporu: Bu raporda ilk semptomu presenkop olan akut pulmoner emboli vakası anlatılmaktadır. 23 yaşında erkek hasta presenkop ve dispne ile başvurdu. Akut koroner sendrom ve inme ve nöbetler gibi nörolojik bozukluklar klinik öykü ve elektrokardiyogramdaki dinamik değişikliklerle dışlandı. Tanı bilgisayarlı tomografi pulmoner anjiyogram ile tamamlandıktan sonra akut pulmoner embolinin şiddeti değerlendirildi ve ardından hastaya fibrinolitik ilaç verildi.
Sonuç: Bu vaka, uzun süreli hareketsiz kalma sonrasında pulmoner emboli geçiren ve venöz tromboembolizm için potansiyel risk oluşturan bu hastaların erken tanısı ve tedavisi için yol gösterici öneme sahiptir. Temel hayati belirtilerinde sorun olan hastalarda kardiyopulmoner hastalıklardan şüphelenilmelidir. Pulmoner emboli olasılığını değerlendirdikten ve D-dimer taraması yaptıktan sonra, mümkün olan en kısa sürede bilgisayarlı tomografi pulmoner anjiyogram yapılmalıdır. Ek olarak, pulmoner embolinin kritik derecesi değerlendirilmeli ve uygun reperfüzyon ve antikoagülasyon tedavisi uygulanmalıdır.

Anahtar kelimeler: Acil servis, hastaneye yatış, immobilizasyon, pulmoner emboli

Introduction

Venous thromboembolism (VTE) includes deep vein thrombosis (DVT), and pulmonary embolism (PE) and represents different clinical symptoms of the same disease process. VTE is one of the most common causes of death in hospitalized patients. Immobilization is a common risk factor for VTE, and prolonged inactivity reduces blood flow and leads to venous stasis. Endothelial damage along with hypercoagulation also plays a role and contributes to the pathophysiology of venous thrombosis(1). PE is a common and life-threatening disease. PE usually results from a thrombus originating from the deep venous system of the lower extremities. However, rarely, it also originates from the pelvic, renal, upper extremity veins or right heart chambers. After reaching the lung, large thrombi can settle in the bifurcation of the main pulmonary artery or lobar branches and cause hemodynamic deterioration. Pulmonary thromboembolism is not a disease in itself. Rather, it is a complication of underlying venous thrombosis (2). The

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causes of PE are multifactorial. Causes include deep vein thrombosis, hypercoagulable, immobilization, surgery and trauma, pregnancy, oral contraceptives and estrogen replacement, malignancy, and hereditary factors. The classical presentation of PE is the sudden onset of pleuritic chest pain, shortness of breath, and hypoxia. However, most patients with PE don't have any obvious symptoms at presentation. On the contrary, symptoms can range from sudden devastating hemodynamic collapse to gradually progressive dyspnea (3). The diagnosis of PE should be suspected with unexplained respiratory symptoms that may be considered with an alternative diagnosis.

Atypical symptoms such as seizures, syncope, abdominal pain, fever, cough, wheezing, decreased level of consciousness, new-onset atrial fibrillation, hemoptysis, flank pain, and delirium in elderly patients may be observed in PE patients. Syncope may be the only symptom of PE; however, syncope or loss of consciousness is the main symptom in less than 1% of PE patients (4). In this article, a case of acute PE detected in a 23-year-old male patient who presented with presyncope and dyspnea is explained.

Case Report

A 23-year-old male patient, who was previously fit and healthy and a non-smoker, was brought to the emergency department of our hospital with dyspnea and presyncope. He was a person who had no medical history, no allergies and not used medication regularly. The patient described dyspnea, pleuritic chest pain, pain, and swelling in the right leg that had been going on for 4 days. He stated that he had taken a 2-hour train ride 5 days ago and had been studying intensively for approximately 12 hours for the last 4-5 days. In the primary evaluation, the patient was awake, isochoric pupils and light reflexes were positive and his Glasgow Coma Scale (GCS) was calculated as 15/15. Breath sounds in both lungs and heart rhythm were regular, and no pathological murmur was heard in the auscultation area of both valves. However, there was painful swelling in the right extremity without any temperature increase. The physical examination results at admission were as follows: Fever, 36.5°C; pulse, 100 times/minute; breathing 24 times/minute; and blood pressure 100/60 mmHg. The hemoglobin level of the case was 16.3 g/dl, white blood cell (WBC) count was 7,22/µL, and platelet count was 205.000/µL. His prothrombin time and activated partial thromboplastin time were 1.07 (INR) and 30.5 sec, respectively. However, the D-dimer level was elevated to 4188 ng/mL. Electrocardiogram (ECG) results were the following: sinus rhythm, typical S1Q3T3 manifestation on electrocardiogram (Figure 1), and no dynamic changes in the recheck. Acute

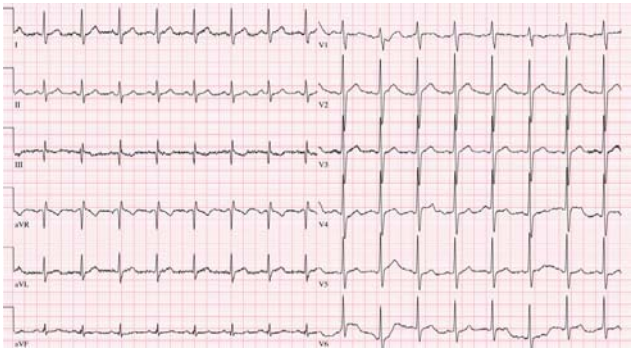

Figure 1. The patient's electrocardiogram on admission

coronary syndrome and neurological disorders such as stroke and seizures were excluded by clinical history and dynamic changes in the electrocardiogram. Deep vein thrombosis was not detected in bilateral lower extremity venous Doppler ultrasonography. Because he spent most of his time working at a desk, a computed tomography pulmonary angiogram (CTPA) was performed to diagnose PE associated with reduced mobility, although there was no evidence of deep vein thrombosis. In CTPA, a filling defect in favor of massive embolism was observed in both pulmonary arteries and all lobar branches (in Figure 2,3). In addition,

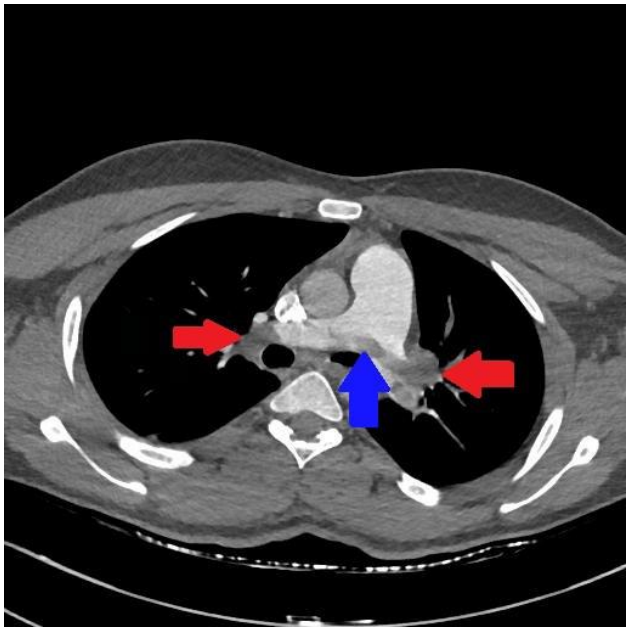

Figure 2. Axial section CTPA image shows the appearance of saddle pulmonary embolism (blue arrow) and pulmonary embolism extending to the lobar branches distal to both pulmonary arteries (red arrows).

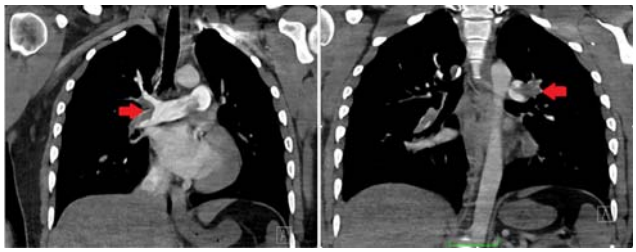

Figure 3. Coronal section CTPA images show pulmonary embolism (red arrows) extending to the lobar branches distal to both pulmonary arteries.

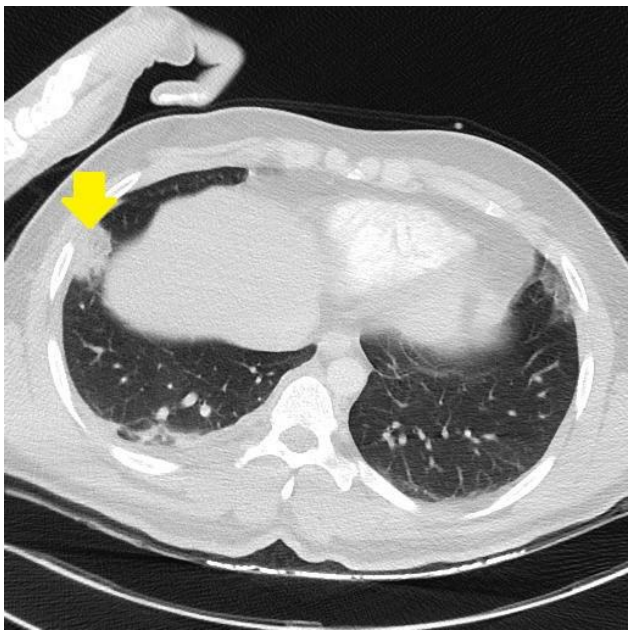

Figure 4. Axial section CTPA image shows consolidation (yellow arrow) consistent with pulmonary infarction in the anterobasal segment of the lower lobe of the right lung.

consolidation compatible with pulmonary infarction was observed in the right lung in the CTPA (Figure 4). Echocardiography (ECHO) revealed an increase in pulmonary artery systolic pressure (58 mmHg), right ventricle dilatation, and mild-moderate tricuspid valve regurgitation were observed. The severity of acute PE was assessed by vital signs, right heart strain findings on bedside ECHO, and massive pulmonary embolism on imaging, and the young patient was given a fibrinolytic drug. Following this, the patient's vital signs were stable and this patient was discharged without any complications 8 days later. Genetic tests were performed 4 months after the patient was diagnosed and were evaluated as negative. In the thrombophilia panel, homocysteine, protein C, and antithrombin III were detected within normal limits, while the level of protein S was below the normal limit.

Discussion

PE is a clinical condition characterized by obstruction of blood flow in the pulmonary artery, typically caused by a thrombus advancing from a vein in the lower extremity. PE is a recurrent cardiovascular disease frequently encountered in emergency departments. The diagnosis of PE can be difficult to determine and can be easily overlooked due to nonspecific symptoms or clinical findings such as acute dyspnea, chest pain, cough, hemoptysis, and syncope, which are also present in other cardiopulmonary diseases. However, early diagnosis and treatment of PE, especially when massive PE is present or complicated by shock and cardiopulmonary arrest, can significantly reduce morbidity and mortality. PE is a common and life-threatening condition that requires immediate evaluation and treatment to improve the results. Acute PE is the most common cause of acute right ventricular (RV) pressure overload.

The primary cause of death after acute PE is acute RV dysfunction (5,6). The incidence of PE is approximately 60 to 120 cases per 100.000 people per year. Approximately 60.000 to 100.000 patients die from PE each year in the United States. PE remains a diagnostic challenge because symptoms are nonspecific, and less than 10% of patients evaluated for PE are diagnosed with PE (4). Our patient was a young man whose first symptom of PE was presyncope, followed by shortness of breath and chest pain. ECG, ECHO, D-dimer, and other examinations made us think about the possibility of PE. After the diagnosis of PE was confirmed by CTPA, fibrinolytic therapy was applied to the patient.

Immobilization is defined as being bedridden for at least three days or having spent most of the time lying or sitting. Immobilization, for VTE, is a common risk factor and prolonged inactivity reduces blood flow and leads to the development of venous stasis. Venous stasis, along with endothelial damage and hypercoagulability, also play a role and contribute to the pathophysiology of venous thrombosis. The risk of PE increases with prolonged bed rest, prolonged sedentary work at a desk, or immobilization of an extremity in a cast. In this case, venous thrombosis may have occurred after our patient had recently studied intensively for a long time and had lower extremity immobility, and this situation may have led to PE.

As an initial symptom of PE, presyncope or syncope can be difficult to diagnose. But these symptoms may be the ''forgotten sign'' of life-threatening PE disease. The need for rapid diagnosis is clear because, with

appropriate treatment, the majority of patients can survive. The possibility of PE should be kept in mind, especially in patients with presyncope or syncope accompanied by shortness of breath. The vital signs, heart rate, ECG, respiratory rate, and oxygen saturation of these patients should be determined immediately and high-risk patients should be given appropriate respiratory support and blood pressure support as soon as possible. When the clinical assessment of the probability and D-dimer screening for PE is completed, CTPA should be performed on a case-by-case basis to help clarify or exclude the diagnosis of PE. Meanwhile, the severity of PE should also be evaluated and reperfusion or anticoagulation treatment should be performed accordingly.

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