Isolated Syncope As The Sole Symptom Of Pulmonary Thromboembolism: A Case Report

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Anahtar kelimeler: Pulmoner tromboembolizm, Senkop, Transtorasik ekokardiyografi

ABSTRACT

The aetiology of syncope remains unknown in more than a third of cases and syncope is concomitantly presented in 10% of patients with pulmonary thromboembolism which indicates a hemodynamically unstable clinical condition. We described a case of a 59-year-old previously healthy male patient with pulmonary thromboembolism with isolated syncope attack in the absence of dyspnoea, pleuritic chest pain and haemoptysis. Clinical suspicion is the main step in the diagnosis of pulmonary thromboembolism. We emphasize that pulmonary thromboembolism should be considered in an unexplained isolated syncope attack and value of echocardiography to evaluate right ventricular function and exclude other cardiac reasons of syncope.

Key words: Pulmonary thromboembolism; Syncope; Transthoracic echocardiography.
INTRODUCTION

Diagnosis of pulmonary thromboembolism (PTE) is a challenge for clinicians in the absence of dyspnoea, pleuritic chest pain, haemoptysis, tachycardia and tachypnea which are found in 97% of patients (1). Approximately 3% of emergency department visits are due to syncope (2). There should be an increased suspicion of PTE in the differential diagnosis of syncope, especially in the absence of predisposing factors to embolism and hemodynamic instability (3). Tricuspid annular plane systolic excursion (TAPSE) by echocardiography is a valuable index in PTE for evaluating right ventricular dysfunction (4). We aimed to introduce a rare case of PTE with isolated syncope attack.

CASE REPORT

A 59-year-old previously healthy male patient was admitted to the chest disease outpatient clinic in September 2015 with transient syncope episodes twice for the last three days. He didn’t have a known neurological disease or syncope episode previously. At anamnesis of the patient he had no dyspnoea, pleuritic chest pain, cough, and haemoptysis. No seizures were witnessed, and he didn’t experience any incontinence. On physical examination, breath sounds were normal bilaterally. There was no abnormal physical finding of heart, abdominal and central nervous system. Oxygen saturation at pulse was 94%. He had a respiratory rate of 16/minutes, pulse rate of 80/minutes, and supine blood pressure of 100/60 mmHg and hypotension was not observed within 3 min in the orthostatic position. His body weight was 73 kg and height was 168 cm, body mass index was 25.8 kg/m2. He had no history for surgery, trauma, malignancy, immobility, any chronic disease and medication usage. He had no previous deep venous thrombosis (DVT) or PTE.

Blood biochemistry, sedimentation, and complete blood counts were normal. Arterial Blood gas analysis revealed a pH of 7.44, 74.3 mmHg of PaO2, 32.2 mmHg of PaCO2, 21.82 mmol/l of HCO3- and 94.9% of oxygen saturation in the room air. There was an increased alveoloarterial oxygen gradient (A-a O2 gradient). He had a very high D-Dimer level, 3777 ng/ml (0-500). Troponin I was 69.3 pg/ml (0-26.2)

Transthoracic echocardiography (TTE) revealed a deviated interventricular septum having “D-shape” sign, very severe pulmonary hypertension (pulmonary artery pressure=120 mmHg), significantly dilated right ventricle (RV) and atrial spaces, grade 3 tricuspid regurgitation, 13 mm of TAPSE with normal left ventricle (LV) function (LV ejection fraction: 60%) Pulmonary CT angiography (CTA) was performed and thrombus was seen on both major pulmonary arteries (Figure 1A-B).

Figure 1A

Figure 1B

Figure 1: Contrast enhanced (a) axial and (b) coronal pulmonary CTA views, There are filling defects consistent with bilaterally acute thromboembolism at main, lobar, and right segmentary branches of pulmonary arteries.
There was no infarct or effusion at parenchyma of lung (Figure 1C).

Figure 1C: Bilaterally lung parenchymas are normal at figure (1-c)

Also bowing of interventricular septum to the LV was seen. The ratio of the RV to the LV was found to be two (Figure 2). Cranial CT of the patient was normal.

Figure 2: At axial view of contrast enhanced pulmonary CTA, interventricular septum is in abnormal position, bowing to left ventricle. Right ventricle to left ventricle diameter ratio is found 2 and accepted as right ventricular dysfunction.

There was a regular rhythm consistent with sinus rhythm with Q and T waves in lead III and an S wave in lead I, negative T waves at V1-3, lead I and right bundle branch block at electrocardiography. Chest X-ray was normal. A Doppler scan of the legs revealed an acute DVT in the left lower limb on the posterior tibia vein. Thrombolytic therapy with recombinant tissue plasminogen activator (r-tPA) 100 mg over a 120-minutes period was applied to the patient. Following intravenous unfractionated heparin was started with bolus heparin at 80 IU/kg IV and maintenance infusion at 18 IU/kg/hour. Warfarin therapy was added at the second day of infusion with 5 mg. After a 13-day course of hospital treatment, he was discharged on oral warfarin therapy. After thrombolytic and heparin infusion treatment, TTE was repeated on day 7 and it was found that pulmonary artery pressure decreased to 30 mm Hg, interventricular septum was returned to the middle line, TAPSE increased to 18 mm, RV size was in normal size while right atrium was slightly enlarged.

DISCUSSION

PTE diagnosis is challenging because there is no specific sign or symptom hence classic triad of dyspnoea, chest pain and haemoptysis are present in minority of patients. It was reported that 20–25% of all PTE cases are presented as sudden death and 81% of PTE discovered at autopsy are unsuspected before death (2,5). Approximately 80-95% of PTE emerge as a result of migration from most commonly deep veins of calf. DVT was found 75% to 88% in patients with PTE (6,7).

Isolated syncope is transient loss of consciousness in the absence of prior or concurrent neurologic, coronary, or other cardiovascular disease signs and symptoms. There are three mechanisms in the setting of PTE. First, obstruction of pulmonary vascular system cause RV failure which affects LV filling with diminished cardiac output and systemic hypotension, tachycardia, and cerebral hypoperfusion. Secondly, pre-existing left bundle branch block or newly developed right bundle branch block cause complete atrioventricular block. Finally, Bezold-Jarisch reflex arc causes bradycardia with vasodilation and hypotension which is triggered by occlusion of main pulmonary artery by thrombus (2,3). Prolonged immobilization, history of thrombophlebitis...
and bone fractures of the lower extremities is the most important risk factors for PTE. While 78% of patients had one known risk factor, 50% of patients have any risk factor identified (7,8). Our case didn’t have any acquired risk factor. We considered possible risk factors as age and male gender.

Syncope is seen almost always with other symptoms in of 10% patients with PTE, but may emerge regardless of hemodynamic instability (2). Our case was presented with only syncope in the absence of dyspnoea, chest pain, haemoptysis, tachypnea and hypotension. We attribute this condition to patient’s cardiopulmonary well-being which may explain this extremely rare presentation. The Framingham study supports us with their study. They conducted 26 year follow-up of 5209 patient and found that there were no statistically significant difference in stroke, myocardial infarction, sudden death, and cardiovascular mortality in respect to presence of isolated syncope (9). Kapoor et al reported that syncope secondary to non-neurologic causes does not increase sudden death risk if patient do not have chronic diseases (3).

Mild hypoxemia and elevated D-Dimer level led us to elucidate cause of syncope. PTE is usually accompanied by arterial hypoxemia and hypocapnia. PaO2 level was lower than 82 mm Hg and PaCO2 lower than 37 mm Hg in 90% of patients with PTE. Also increased A-a O2 gradient (>20 mm Hg) is more sensitive than hypoxemia alone (1,10).

The primary cause of mortality in PTE is RV failure (4). TTE demonstrates secondary changes in cardiac size and function caused by the hemodynamic effects of PTE which are dilatation and reduced function of RV cavity, abnormal septal motion or flattening, reduced LV cavity size, dilated pulmonary arteries, significant tricuspid regurgitation, and dilatation of the inferior vena cava. Also TTE enables direct visualization of free-floating thrombus in the right-sided chambers or the pulmonary artery observed in 7% to 18% of patients (11).

RV fractional area change (RVFAC) and ejection fraction are commonly used parameters to define RV function however, they have limited value due to complex 3-dimensional geometry and non-concentric contraction pattern of RV. TAPSE is an easily measurable parameter which reflects RV systolic function along the long axis and it has been shown to be closely related to the RV ejection fraction. RV function and RVFAC is found to be significantly better when TAPSE is ≥18 mm. Also TAPSE correlates significantly with troponin I which is one of the serum markers of RV dysfunction (4,11).

The main indications for thrombolytic therapy include hemodynamic instability, respiratory distress, pulmonary hypertension and echocardiographic evidence of RV dysfunction in patients with PTE (7,12). Our patient was not in shock clinic but he had a very high pulmonary artery pressure and very low TAPSE level which indicated severe right heart failure that probably would lead to left heart failure in a short time. Clinical outcome in PTE is directly related to RV dysfunction (4,13).

Final diagnosis of our case was established with pulmonary CTA which not only shows size and location of thrombi also gives information about RV dysfunction by determining the ratio of the RV to LV diameters, and shape of the interventricular septum (2,14). Shape of interventricular septum may be flattened or bowing to the LV according to the severity of right heart pressure overload. RV to LV ratio becomes more than one due to increased size of right heart. Our case had increased RV to LV ratio (>2) and leftward bowing of interventricular septum.

Finally, PTE is a potentially fatal disorder. Cardiorespiratory status and thrombus size estimate mortality. Diagnosis depends on clinical symptoms and signs in combination with imaging modalities. TTE is a sensitive method for detection of right ventricular overload in PTE. Syncope may occur regardless of the presence of haemodynamic instability as in our case so PTE should be considered in the differential diagnosis of all patients presenting with syncope.

REFERENCES


