GKDA Derg 2020;26(4):254-7
doi: 10.5222/GKDAD.2020.77598

Olgu Sunumu / *Case Report*

Negative Baseline Imaging Does not Exclude Acute Pulmonary Embolism in Patients with Recurrent Syncopal Episodes and Cardiac Biomarker Elevation[§]

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Yineleyen Senkop Atakları ve Kardiyak Biyomarker Yükselmesi Olan Hastalarda Normal Saptanan Bazal Görüntüleme Testleri Akut Pulmoner Emboliyi Ekarte Ettirmez

Çıkar Çatışması: Çıkar çatışması yoktur.
Finansal Destek: Bu çalışma, herhangi bir fon tarafından desteklenmemiştir.
Hasta Onamı: Hastalardan aydınlatılmış onam alınmıştır.

Conflict of Interest: There is no conflict of interest.
Funding: The authors declared that this study has received no financial support.
Informed Consent: Informed consent was obtained from the patients.

Cite as: Kocabas U, Altay H, Özkalaycı F, Pehlivanoğlu S. Negative baseline imaging does not exclude acute pulmonary embolism in patients with recurrent syncopal episodes and cardiac biomarker elevation. GKDA Derg. 2020;26(4):254-7.

ABSTRACT

Acute pulmonary embolism (PE) is an important vascular disease with high mortality and morbidity and syncope is an uncommon presentation sign of acute PE. This report presents two cases illustrating acute PE as a cause of recurrent syncopal episodes with elevated cardiac troponin and N-terminal pro-brain natriuretic peptide levels despite normal initial trans-thoracic echocardiographic examination and negative Doppler ultrasound imaging for detection of deep vein thrombosis.

Keywords: *cardiac biomarker, pulmonary embolism, syncope, troponin*

ÖZ

Akut pulmoner emboli (PE), yüksek mortalite ve morbiditeye sahip önemli bir vasküler hastalıktır ve senkop akut PE'nin ender görülen bir belirtisidir. Bu makalede, yineleyen senkop atakları ile birlikte yüksek kardiyak troponin ve N-terminal pro-B-tipi natriüretik peptid düzeylerine sahip olup, ilk değerlendirmede normal trans-torasik ekokardiyografi ve normal alt ekstremite derin venöz Doppler ultrasonografi bulguları saptanan 2 akut pulmoner emboli olgusu sunulmuştur.

Anahtar kelimeler: *kardiyak biyomarker, pulmoner emboli, senkop, troponin*

Received/Geliş: 23.05.2020
Accepted/Kabul: 21.07.2020
Published Online/Online yayın: 31.12.2020

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[§] This case report was presented as a poster presentation at EuroEcho 2019 Congress, 04-07 December 2019, Vienna, Austria.

INTRODUCTION

In patients who are admitted to emergency department (ED) due to recurrent syncopal episodes, acute pulmonary embolism (PE) is rarely considered as a possible cause as syncope is an atypical presentation of PE and only 10% of patients have a syncope as the initial symptom [1,2]. This report presents two cases illustrating acute PE as a cause of syncope with elevated cardiac troponin (cTn) and N-terminal pro-brain natriuretic peptide (NT-proBNP) levels despite negative findings of baseline imaging modalities including echocardiography and Doppler ultrasound used for the detection of deep vein thrombosis.

CASE 1

An 83-year-old woman with a history of Parkinson's disease presented to ED due to recurrent syncopal episodes. According to her anamnesis, she had four episodes of syncopes during last three days without chest pain, dyspnea, palpitation or hemoptysis. On admission, physical examination was unremarkable and 12-lead electrocardiogram (ECG) revealed a normal sinus rhythm with a heart rate of 70 bpm and first-degree atrioventricular block without ischemic changes. Arterial blood gas analysis in room air showed mild hypoxemia and hypocarbia (pH: 7.43, PaO_2: 58 mmHg, $PaCO_2$: 33 mmHg, and lactate: 1.1 mmol/L). Laboratory tests revealed raised levels of cTn (60 pg/mL, normal range: 0-15.6 pg/ml) and NT-proBNP (7929 pg/mL, normal range: 0-100 pg/mL). Patient's pre-test probability for acute pulmonary embolism was low (Wells' score <2) but D-dimer level was elevated (1908 ng/mL, normal range: 0-198 ng/mL, age-adjusted cut-off value: 830 ng/mL). Transthoracic echocardiography (TTE) demonstrated preserved biventricular systolic functions without any cardiac chamber enlargement and mild tricuspid regurgitation with a pulmonary systolic pressure of 35 mmHg and also no pericardial effusion was detected. Doppler ultrasound imaging for detection of deep vein thrombosis was negative. Computed tomography pulmonary angiography (CTPA) showed filling defects in the bilateral main pulmonary arteries consistent with acute bilateral pulmonary embolism (Figure 1). She was treated with enoxaparin 60 mg two times a day during hospitalization. The patient was discharged with rivaroxaban therapy after four days of hospitalization period without any complication.

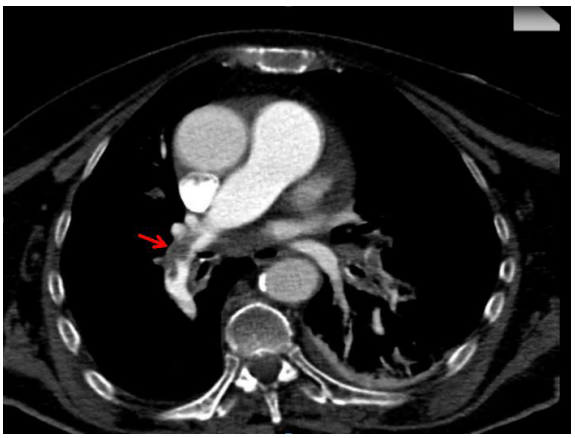

Figure 1. Computed tomography pulmonary angiography showing filling defects in the pulmonary artery consistent with acute pulmonary embolism (red arrow).

CASE 2

A 69-year-old woman presented to ED with symptoms of chest pain and recurrent syncopal episodes for the last 2 days. Her medical history revealed hypertension and hyperlipidemia. On admission, physical examination was unremarkable and 12-lead ECG showed a normal sinus rhythm with a heart rate of 105 bpm and T-wave inversions in inferior leads. Laboratory tests showed elevated levels of cTn (143 pg/mL, normal range: 0-15.6 pg/mL) and NT-proBNP (6115 pg/ml, normal range: 0-100 pg/mL). Patient's pre-test probability for acute pulmonary embolism was low (Wells' score <2) but D-dimer level was elevated (920 ng/mL, normal range: 0-198 ng/mL, age-adjusted cut-off value: 690 ng/mL). Transthoracic echocardiography demonstrated normal left ventricular systolic function with an ejection fraction of 55% and normal right ventricular function and chamber size with a tricuspid annular plane systolic excursion of 22 mm and also pericardial effusion was not detected. Firstly, coronary angiography (CAG) was performed due to elevated cTn and T-wave inversions in inferior leads to reveal possible diagnosis of non-ST-segment elevation myocardial infarction. Diagnostic CAG revealed non-significant coronary artery stenosis. Doppler ultrasound imaging for detection of deep vein thrombosis was negative. Computed tomography pulmonary angiography showed filling defects in the bilateral main pulmonary arteries consistent with acute bilateral pulmonary embolism (Figures 2, and 3). She was treated with injectable enoxaparin 60 mg two times a day during hospitalization.

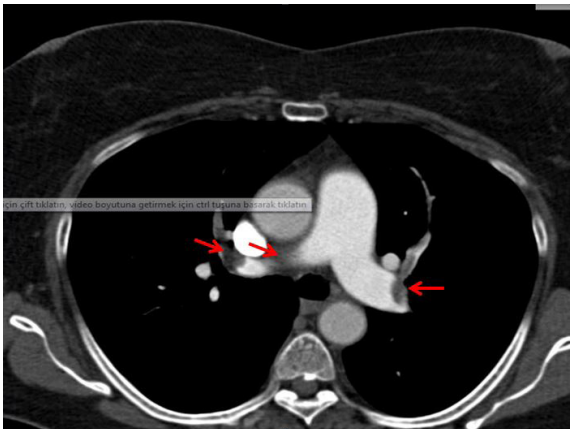

Figure 2. Computed tomography pulmonary angiography showing filling defects in the bilateral main pulmonary arteries consistent with acute bilateral pulmonary embolism (red arrows).

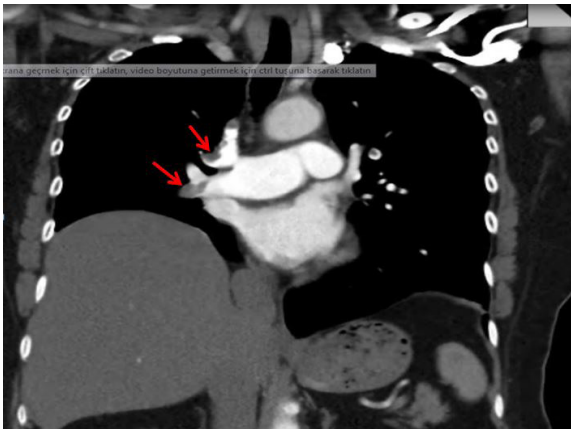

Figure 3. Computed tomography pulmonary angiography showing filling defects in the bilateral main pulmonary arteries consistent with acute bilateral pulmonary embolism (red arrows).

The patient was discharged with rivaroxaban therapy after four days of hospitalization period without any complication.

DISCUSSION

AAcute PE is a frequent vascular disease with high mortality and morbidity rate [1]. The most common symptoms of acute PE are dyspnea, chest pain and hemoptysis. Syncope is an uncommon presentation sign and occurs in %9-13 of patients with acute PE [2-4]. In those patients, presence of syncope indicates poor prognosis [5].

Although diagnostic tools including electrocardiography, TTE or Doppler ultrasound imaging can exclude many possible causes of cardiac syncope such as acute coronary syndrome, high-degree atrioventricular block, cardiac tamponade, severe aortic stenosis, and hypertrophic cardiomyopathy; in some conditions that cause ''transient'' hemodynamic disturbances, these diagnostic tools may be detected normally at the time of patient admission, as in our two cases. Reduced pulmonary blood flow due to total occlusion of central pulmonary arteries is associated with development of syncope awhc can progress to cardiopulmonary collapse and death [3]. On the other hand, in some cases, acute PE in central pulmonary arteries spontaneously undergoes partial resolution. This clinical condition results in transient hemodynamic instability and hypotension associated with syncope [4,6]. After spontaneous partial resolution of thrombus in pulmonary artery, blood pressure may have normalised and in this scenario, it may be difficult to diagnose acute PE. We think that, in our case, the main reason of obtaining normal results with initial imaging studies is spontaneous partial resolution of thrombus in pulmonary artery. In patients with recurrent syncopal episodes, despite presence of normal hemodynamic status at the time of first contact with the patient, elevated cardiac biomarker (cTn and NT-proBNP) levels on admission may suggest transient hemodynamic instability and cardiac injury before hospital admission.

Recently published European Society of Cardiology Acute Pulmonary Embolism Guideline recommends firstly a bedside transthoracic echocardiography in patients with suspected acute PE with hemodynamic instability and if there is no right ventricular dysfunction in bedside TTE, it suggests searching for other causes of shock or instability [1]. Guideline defines hemodynamic instability as follows: i) cardiac arrest (need for cardiopulmonary resuscitation), ii) obs-

tructive shock (systolic blood pressure <90 mmHg or vasopressors required to achieve a blood pressure >90 mmHg despite adequate filling status and end-organ hypoperfusion), and iii) persistent hypotension not caused by new-onset arrhythmia, hypovolaemia, or sepsis. In this definition, it is not mentioned if the presence of syncope before hospital admission may be related to transient hemodynamic instability. We think that, presence of recurrent syncopal episodes and cardiac biomarker elevation reflect transient hemodynamic instability before hospital admission and those patients should be considered as having hemodynamic instability independent of physical examination findings and blood pressure values at initial evaluation. Additionally, we think that, after excluding possible etiologies of cardiac syncope with initial electrocardiography and TTE, presence of recurrent syncopal episodes and elevation of cardiac biomarkers indicates high clinical probability for acute PE. Thus, in these patients with recurrent syncopal episodes and elevated cardiac biomarkers, acute PE should be kept in mind independent of Wells score and initial clinical evaluation and baseline imaging studies and they should be directly evaluated with CTPA to exclude acute PE.

To conclude, these two cases with recurrent syncopal episodes demonstrate that normal initial echocardiographic examination and negative Doppler ultrasound imaging for the detection of deep vein thrombosis do not exclude major acute PE in patients with elevated cTn and NT-proBNP and D-Dimer levels; hence those patients should be firstly evaluated with CTPA.

REFERENCES

1. Konstantinides SV, Meyer G, Becattini C, Bueno H, Geersing GJ, Harjola VP, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). Eur Heart J 2019 pii: ehz405.
https://doi.org/10.1183/13993003.01647-2019
2. Calvo-Romero JM, Pérez-Miranda M, Bureo-Dacal P. Syncope in acute pulmonary embolism. Eur J Emerg Med 2004;11:208-9.
https://doi.org/10.1097/01.mej.0000136696.49343.8f
3. Thames MD, Alpert JS, Dalen JE. Syncope in patients with pulmonary embolism. JAMA. 1977;238(23):2509-11.
https://doi.org/10.1001/jama.1977.03280240055020
4. Altınsoy B, Erboy F, Tanrıverdi H, Uygur F, Örnek T, Atalay F, et al. Syncope as a presentation of acute pulmonary embolism. Ther Clin Risk Manag 2016;12:1023-8.
https://doi.org/10.2147/TCRM.S105722
5. Dellas C, Tschepe M, Seeber V, Zwiener I, Kuhnert K, Schäfer K, et al. A novel H-FABP assay and a fast prognostic score for risk assessment of normotensive pulmonary embolism. Thromb Haemost 2014;111:996-1003.
https://doi.org/10.1160/TH13-08-0663
6. Koutkia P, Wachtel TJ. Pulmonary embolism presenting as syncope: case report and review of the literature. Heart Lung 1999;28:342-7.
https://doi.org/10.1053/hl.1999.v28.a99733