Case Report

Journal of Emergency Medicine Case Reports

Concurrent Pulmonary Embolism and Acute Myocard Infarction: A Case Report

Ethem Acar¹, Ahmet Demir¹, Birdal Yildirim¹, Gökhan Kaya¹

Mugla Sıtkı Kocman University, Faculty of Medicine, Department of Emergency Medicine, Mugla, Turkiye

Abstract

Introduction: Pulmonary embolism and ST-elevation MI are both life threatening diseases. Although rare, concomitant pulmonary embolism and myocardial infarction pose even greater risk. Here we aimed to report a case of ST-elevation acute coronary syndrome developing one hour after diagnosis of pulmonary embolism. Case-Report: A 48-year-old male patient with known coronary artery disease had admitted for chest pain and dyspnea. He had got deep vein thrombosis in 10 days prior to this presentation. There wasn't ST elevation on his electrocardiogram. Pulmonary embolism was detected in the pulmonary CT angiography taken to the patient. The ECG was repeated after the patient, whose treatment was started, had new chest pain and sweating. The patient, whose ST elevation was detected in the inferior leads in the new ECG, was taking in to PCI. The patient, who had a stent in RCA, left the hospital voluntarily on the 3rd day of his admission to the coronary intensive care unit from where he was hospitalized with the diagnosis of ACS and PTE. In conclusion, it should be kept in mind that these two conditions may be present in patients presenting with complaints suggesting both AKS and PTE such as chest pain and shortness of breath.

Keywords: chest pain, electrocardiography, ST elevation, acute coranary syndrome, pulmonery thromboembolism

Introduction

Pulmonary Thromboembolism (PTE) is one of the most important clinical conditions of Venous Thromboembolism (VTE) and often develops on the background of underlying deep vein thrombosis (DVT)¹. Since patients have complaints of chest pain and dyspnea during their arrival, Electrocardiography (ECG) is performed as soon as possible at the time of admission. ECG is evaluated in terms of whether there are findings for cardiovascular differential diagnoses. Although there is no specific ECG change for PTE, sinus tachycardia, right bundle branch block and S1Q3T3 pattern can be seen frequently². The co occurrence of PTE and ST elevation ACS together is a rare situation in the literature³.

In this case report, we aimed to draw attention to the subject by handling a case that presented to us with chest pain and dyspnea and was diagnosed with inferior myocardial infarction after PTE.

Case Report

A 48-year-old male patient applied to our emergency department with the complaints of sudden dyspnea and chest pain that started in the morning. At the time of admission, his general condition was moderate, his body surface was sweaty and had tachypnea. Vital findings: blood pressure arterial 110/70 mmHg, pulse 70 / min, fever 36.6° C, finger tip oxygen saturation in room air was 88%. There was no pathological finding in the lung sounds and heart sounds of the patient who has no jugular venous filling. History revealed that patient was hospitalized in another institution 10 days ago due to deep vein thrombosis (DVT) and he taken on low molecular weigh the heparin (LMWH). He was also diagnosed with coronary artery disease (CAD) 1 year ago. On this medical history, the patient's ECG was taken in order to exclude ACS and cardiac events, and no pathology was detected in the ECG except for T negativity in leads D1, AVL, V3,4,5,6 (figure 1). In his blood tests, complete blood count and emergency biochemistry tests were normal. Troponin test was evaluated as 14 pg / mL (normal). Since the patient had DVT, PTE was primarily considered in the etiology of the current presentation table and Pulmonary Computed Tomography angiography (Pulmonary CT Angio) was performed. The patient was diagnosed with PTE after pulmonary CT angiography, " acute embolic filling defects were observed in all lung lobes on the right and lower lobes and pulmonary artery branches leading to the lower lobe segments on the left "(Figure 2). The patient began to describe

Corresponding Author: Ethem Acar e-mail: dr.ethemacar@gmail.com
Received: 04.03.2020 • Accepted: 19.05.2020
DOI:10.33706/jemcr.698491
©Copyright 2020 by Emergency Physicians Association of Turkey - Available online at www.jemcr.com



Figure 1. The first ECG of the patient

chest pain in the pressure style, which was started newly and spread to the left arm while the consultation procedures were going on in the emergency room. ECG was repeated after emerging chest pain occured (Figure 3). In the newly performed ECG, ST segment elevation in DII-III, AVF leads, ST segment depression and T negativity in DI, AVL, V3-6 leads were detected. Emergency cardiology consultation was performed with the diagnosis of ST elevation ACS. The control troponin test was found as 1559 pg / mL.The patient, whose emergency trans thoracic echocardiography (Echo) was planned by cardiology and chest pain and ACS with Echo findings, was taken into percutaneous coronary intervention (PCI) by cardiology. In addition, although a patent foramen ovale evaluation was performed in Echo, it could neither be clearly diagnosed nor said anything.Stent was placed in the patient with stenosis in the right coronary artery on angiography (Figure 4). After wards, the patient, who had 100% full openness, was hospitalized in the coronary intensive care unit. The patient, who was also examined by chest diseases in intensive care unit, was followed up with good condition on the 3rd day of hospitalization and left the hospital with his own consent.

Discussion

In our case, it was presented as an remarkable case since both PTE and ST elevated MI were seen together in the same time frame. It is very rare to beseen these two diseases together in the literature. Both of these diseases are serious diseases even in terms of mortality and morbidity, more over, the concurrency of both makes follow-up and treatment more difficult⁴.

One of the most important parameters used in the differential diagnosis of patients presenting with chest pain is ECG. Infact, it is used in ACS diagnosis. However, there can



Figure 2. PTE compatible filling defects in the patient's pulmonary CT angiography.



Figure 3. The 2nd ECG taken in our emergency department

alsobe various ECG changes in PTE. The most common of these are sinus tachycardia, right bundle branch block and S1Q3T3 pattern². Depending on right ventricular tension and dilation, ST segment and T segment changes are reported in anterior leads in ECG^{5,6}. With this change, we often encounter ST segment depression, and rarely, PTE cases accompanied by ST elevation have also been reported⁷⁻¹¹. Therefore, in addition to ECG, history and physical examination are also important. Because similar ECG findings can be seen in ACS as well. ACS guidelines state that rapid differential diagnosis should be made in order to transfer cases with ECG with ST segment elevation to PCI without wasting time. However, if there is no ST elevation on the ECG, the patient's history, physical examination findings,



Figure 4. PCI image of the patient

ECG, troponin values and risk scores are evaluated together. Our patient also applied to the emergency department with the complaint of chest pain. Medical history had CAD. For this reason, in his ECG, T negativity was detected in the anterior leads. Although these ECG changes were seen in ACS, the patient's DVT history and troponin negativity led us to PTE rather than ACS and directed us to take a pulmonary CT angio. In pulmonary CT angio, images confirming the diagnosis of PTE were detected.

What may be causing the concurrency of PTE and ST segment elevation ACS? When the literature is examined, it is seen that the underlying cause is right-to-left shunt for a reason such as patent foramen ovale, although not in all cases where this concurrency is reported^{4,12,13}. In the review of the literature, in an article presenting a 35-year-old male patient, the researchers stated that the factor 5 leidan mutation caused PTE-ACS association¹⁴. Seguban et al.³ reported that they detected ST elevation in the anterior leads of ECG in a 67-year-old patient with chest pain and dyspnea after an operation due to cancer. However, due to the patient's history and Echo findings, they stated that both pulmonary and coronary angiography were performed considering PTE and both conditions were detected. But the reason for this paradoxical situation in the case reports is not discussed. In a case where a 69-year-old male patient was presented, Wasek et al. stated that their patients had been followed-up for hip fracture, but that he did not use DMAH regularly, and applied to their hospitals with complaints of chest pain and dyspnea. They explained that when they detected ST segment elevation in leads DII-III-AVF and V3-6 in their ECG, they received an emergency PCI and detected a large thrombus in RCA. They also expressed that they detected RV enlargement in the Echo performed for the patient whose dyspnea did not regress even two days after PCI and that they detected the embolism in pulmonary CT Angio for PTE. In this case, they pointed out that they detected patent foramen ovale as the cause of paradoxical thrombus¹².Similarly, in two different case reports, there are similar ST elevation MI and PTE concurrency. In these two case reports, it was stated that patent foramen ovale was detected in cases^{4,13}.In our case, the patent foramen ovale could not be excluded in the transthoracic Echo performed in the emergency department, but the patient had to be postponed to a later date since the patient was taken to the emergency angiogram. No data were found from the patient file regarding the examination of this condition while in hospital. However, in case presentations detected in foramen ovale, although embolism is observed in patients' coronary angiograms, the situation is different in our case because stenosis is in the foreground. For this reason, in our case, the absence of patent foramen ovale seems more reasonable. However, it seems difficult to make a definite judgment as the diagnosis cannot be clarified or the patent foramen oval is not ruled out.

In conclusion, it should be kept in mind that these two conditions may coexist in patients presenting with complaints such as chest pain and dyspnea, suggesting both ACS and PTE. Although the underlying cause is often a patent foramen ovale, this is uncertain. However, it should be remembered that PTE should also be considered in patients with DVT and PCI with coronary embolism.

Referances

- American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Thromboembolic Disease:, Wolf SJ, Hahn SA, Nentwich LM, Raja AS, Silvers SM, Brown MD. Clinical Policy: Critical Issues in the Evaluation and Management of Adult Patients Presenting to the Emergency Department With Suspected Acute Venous Thromboembolic Disease. Ann Emerg Med. 2018 May;71(5):e59-e109.
- Yeh KH, Chang HC. Massive pulmonary embolism with anterolateral ST-segment elevation: electrocardiogram limitations and the role of echocardiogram. The American journal of emergency medicine. 2008;26(5):632 e1-3. Epub 2008/06/07.
- 3. Joseph Marc S. Seguban, Elaine Alajar, Jessore Isidro, Noemi Pestaño, Felix Eduardo et al. Concurrent Massive Pulmonary

Embolism With Emboli in Transit and Anterior ST Elevation Myocardial Infarction with Increased Bleeding Risk Successfully Treated with Low-Dose Systemic Alteplase: A Case Report. Cardiology and Cardiovascular Medicine 3 (2019): 352-359.

- **4.** Alkhalil M, Cahill TJ, Boardman H, Choudhury RP. Concomitant pulmonary embolism and myocardial infarction due to paradoxical embolism across a patent foramen ovale: a case report. Eur Heart J Case Rep. 2017 Nov 23;1(2):ytx010.
- Cheng TO. Mechanism of ST-elevation in acute pulmonary embolism. International journal of cardiology. 2005;103(2):221-3. Epub 2005/08/06.
- Van Mieghem C, Sabbe M, Knockaert D. The clinical value of the ECG in noncardiac conditions. Chest. 2004;125(4):1561-76. Epub 2004/04/14.
- Wang K, Asinger, R. W., Marriott, H. J. L. ST-Segment Elevation in Conditions Other Than Acute Myocardial Infarction. . N Engl J Med. 2003;349(22):2128-35.
- Wilson GT, Schaller FA. Pulmonary embolism mimicking anteroseptal acute myocardial infarction. The Journal of the American Osteopathic Association. 2008;108(7):344-9. Epub 2008/07/24.
- Lin JF, Li YC, Yang PL. A case of massive pulmonary embolism with ST elevation in leads V1-4. Circulation journal : official journal of the Japanese Circulation Society. 2009;73(6):1157-9. Epub 2008/12/20.
- **10.** Livaditis IG, Paraschos M, Dimopoulos K. Massive pulmonary embolism with ST elevation in leads V1-V3 and successful thrombolysis with tenecteplase. Heart. 2004;90(7):e41. Epub 2004/06/18.
- Falterman TJ MJ, Daberkow D, Weiss LD. Pulmonary embolism with ST segment elevation in leads V1 to V4: case report and review of the literature regarding electrocardiographic changes in acute pulmonary embolism. J Emerg Med. 2001;21(3):255-61.
- 12. Wąsek WC, Samul W, Ryczek R, Skrobowski A. Unique case of ST-segment-elevation myocardial infarction related to paradoxical embolization and simultaneous pulmonary embolization: clinical considerations on indications for patent foramen ovale closure in no-guidelines land. Circulation. 2015 Mar 31;131(13):1214-23.
- **13.** Budavari AI, Glenn TJ, Will KK, Askew JW, Fortuin FD. A case of simultaneous pulmonary embolism and acute myocardial infarction secondary to a previously undiagnosed patent foramen ovale. J Hosp Med. 2009 May;4(5): E5-9.
- 14. Burkhardt C, Eeckhout E, Qanadli SD, Aubert JD, Meier JM. Simultaneous Acute Pulmonary Embolism and Isolated Septal Myocardial Infarction in a Young Patient. Eur J Case Rep Intern Med. 2016 Aug 24;3(6):000436