Case Report

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Management of Pulmonary Embolism and Pericardial Effusion

Behshad Pazooki[1], **Alborz Sherafati**[2*]

1. Department of Internal Medicine, Imam Khomeini Hospital, Tehran University of Medical Sciences, Tehran, Iran.
2. Department of Cardiology, Imam Khomeini Hospital, Tehran University of Medical Sciences, Tehran, Iran.

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ABSTRACT

Pulmonary embolism is a serious complication occurring in different cancers. Patients who have cancer may have complications like brain metastasis or pericardial effusion, which may change the therapeutic management of pulmonary embolism. In this article, a patient is presented with lung adenocarcinoma resulting in pericardial effusion. He was admitted with pulmonary embolism and atrial flutter, which made his management difficult.

Introduction

Patients with cancer are at increased risk of thrombosis due to their hypercoagulative state, immobilization, or treatments (surgery or chemotherapy) [1]. The prevalence of venous thromboembolism can reach 20% in patients who have cancer [1]. This prevalence is about 5% in patients with lung cancers [1]. Presence of pulmonary embolism can worse the prognosis and lower the patient`s survival. On the other hand, complications of cancer, including pericardial effusion, can influence the patient`s management. In this report, a patient suffering from lung adenocarcinoma and pericardial effusion is presented who was admitted to the emergency department due to pulmonary embolism.

*** Corresponding Author:**
Alborz Sherafati, MD.
Address: *Department of Cardiology, Imam Khomeini Hospital, Tehran University of Medical Sciences, Tehran, Iran.*
E-mail: *asherafati@sina.tums.ac.ir*

Case Presentation

A 50-year-old man was admitted to the emergency department because of dyspnea. His symptoms started 3 days before admission, with sudden onset, and without stop. He was also complaining of non-massive hemoptysis and episodic palpitation from 3 days before. There was no history of chest discomfort or orthopnea at presentation. In his medical history, he reported a lung adenocarcinoma diagnosed about six months before his present admission and was under treatment with chemotherapy from the time of diagnosis.

His cancer was not controlled. He had a history of cigarette smoking, about 20 pack/year from 30 years ago. His drug history was unremarkable. In physical examination, his blood pressure was 75/40 mmHg, his pulse was regular, and his heart rate was 150 beats per minute. His respiratory rate was 30 per minute, and his O_2 saturation was 85% in room air. He had an elevated jugular venous pressure. Unilateral left leg edema was also detected with tenderness on palpation.

Based on our hospital protocol, modified Geneva score was calculated for the patient, which was 16. Scores more than 10 are considered as high risk for pulmonary embolism. Thus pulmonary CT angiography was performed. In pulmonary CT angiography, bilateral filling defects in favor of thrombosis were detected in left and right pulmonary arteries (Figure 1).

Because of hemodynamic instability, fibrinolytic therapy was considered for treatment, but in the patient`s electrocardiogram, atrial flutter with 2:1 atrioventricular block and the rapid ventricular response was detected (Figure 2). It was a question whether the arrhythmia or the pulmonary thrombosis had caused hemodynamic instability. Also, echocardiography was performed in the next step, which showed mild right ventricular systolic dysfunction. Cardiac troponin was also checked, which was negative.

Based on the above data, massive pulmonary embolism was unlikely due to the lack of evidence in favor of right ventricular strain. Thus, arrhythmia was considered responsible for the patient`s instability, and 300 mg intravenous amiodarone was initiated. The patient`s rhythm was converted successfully to sinus rhythm. After termination of the patient`s arrhythmia, his systolic blood pressure rose to 120 mm Hg, and his hemodynamic profile became stable. Fibrinolytic therapy was deferred based on hemodynamic improvement. However, another challenge happened in the patient`s management. During echocardiography, moderate pericardial effusion was detected with no hemodynamic effect.

It was questioned if the initiation of anticoagulant would worsen the pericardial effusion severity and may result in cardiac tamponade. Based on consensus, anticoagulation therapy with intravenous heparin was started and followed up by daily echocardiography, which showed no progression in the severity of pericardial effusion. After 5 days, heparin was converted

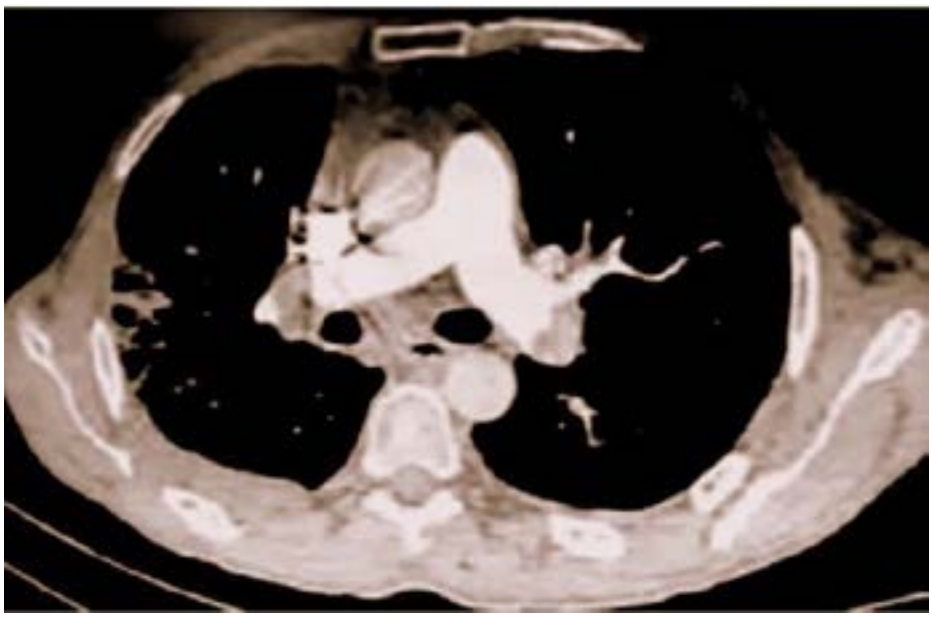
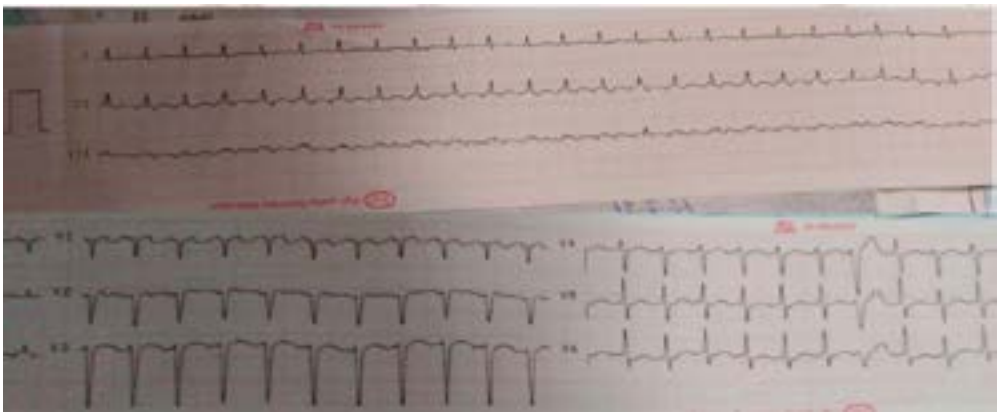


Figure 1. Filling defects in right and left pulmonary arteries in CT scan



2. Twelve-lead electrocardiogram showed atrial flutter with 2:1 atrioventricular block

to subcutaneous enoxaparin, and the patient`s daily follow-up continued. After another 5 other days, the patient was discharged with enoxaparin regarding his malignancy with no complication or progression of pericardial effusion.

Discussion

Arrhythmia can be a manifestation of pulmonary embolism. Atrial fibrillation, atrial flutter, or atrioventricular reentrant tachycardia may occur as the first manifestation. Presence of atrial fibrillation can predict hemodynamic deterioration and 30-day mortality following pulmonary embolism [2]. This arrhythmia is associated with right ventricular pressure overload due to embolism [2]. The atrial arrhythmia is more common in preexisting cardiac disease [3]. Atrial fibrillation can lead to a decrease in the left ventricular filling, shortening of diastolic time resulting in a reduction in coronary perfusion time, and compromising atrial contraction [3]. Thus, it can lead to hemodynamic compromise.

In this patient, finding the cause of hemodynamic instability had an important impact on therapeutic decision making. Hypotension due to pulmonary embolism is considered as massive pulmonary embolism and an indication for fibrinolytic therapy. On the other hand, hypotension due to atrial flutter or fibrillation is an indication for cardioversion.

We investigated signs of right ventricular pressure overload to study the proper treatment. Elevation of cardiac troponin is a sign of myocardial injury due to right ventricular overload [4]. Because of normal level of cardiac troponin, and absence of significant right ventricular dysfunction in echocardiography, atrial flutter was considered as the cause of hemodynamic deterioration. Elevation of blood pressure following the restoration of sinus rhythm confirmed this hypothesis.

Another issue about this patient was the presence of pericardial effusion. Pericardial effusion is common in metastatic cancers [5]. Breast cancer, lymphoma, and lung cancers can cause malignant pericardial effusion [5]. Initiation of anticoagulation is a challenge in such patients due to the risk of pericardial bleeding and converting to cardiac tamponade. Thomas et al. reported a case of pulmonary embolism with large hemorrhagic pericardial effusion [6].

Due to a significant rise in International Normalized Ratio (INR) because of liver involvement, and hemorrhagic nature of pericardial effusion, pericardial drainage was performed, and inferior vena cava filter was placed in that case [6]. In another case, Akhbour et al. reported a patient with pulmonary embolism, pericardial effusion, and lung adenocarcinoma. Due to echocardiographic evidence of cardiac tamponade, pericardiocentesis was also performed in that patient [5]. On the other hand, Han et al. reported a case of pulmonary embolism and pericardial effusion following trauma and spinal cord injury [7]. Conservative approach was selected with the initiation of intravenous heparin and follow up echocardiography. No complication, including tamponade, occurred in that case.

In the first two cases, there was another complication other than pulmonary embolism and pericardial effusion that led to pericardial drainage. In Thomas et al. case report, the patient`s coagulopathy and elevated INR was another limitation of the initiation of the anticoagulant. This coagulopathy placed that patient at higher risk of tamponade. In Akhbour et al. case report, echocardiographic signs of cardiac tamponade were present, which was the indication for pericardiocente-

sis. On the other hand, in Han et al. case report, no sign of tamponade or coagulation disorder was present. In our case, there was neither echocardiographic sign of tamponade nor evidence of coagulopathy in laboratory data. Thus, we chose a conservative approach rather than pericardial drainage or placement of inferior vena cava filter. The patient had no complication in his course, which showed the safety of this approach.

Ethical Considerations

Compliance with ethical guidelines

All ethical principles were considered in this article.

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Conflict of interest

The authors declared no conflict of interest.

References

[1] Van Herk-Sukel MP, Shantakumar S, Penning-van Beest FJ, Kamphuisen PW, Majoor CJ, Overbeek LI, et al. Pulmonary embolism, myocardial infarction, and ischemic stroke in lung cancer patients: Results from a longitudinal study. Lung. 2013; 191(5):501-9. [DOI:10.1007/s00408-013-9485-1] [PMID]

[2] Shopp JD, Stewart LK, Emmett TW, Kline JA. Findings from 12-lead electrocardiography that predict circulatory shock from pulmonary embolism: Systematic review and meta-analysis. Academic Emergency Medicine. 2015; 22(10):1127-37. [DOI:10.1111/acem.12769] [PMID] [PMCID]

[3] Barra SN, Paiva LV, Providencia R, Fernandes A, Leitao Marques A. Atrial fibrillation in acute pulmonary embolism: Prognostic considerations. Emergency Medicine Journal. 2014; 31(4):308-12. [DOI:10.1136/emermed-2012-202089] [PMID]

[4] Patane S, Marte F, Curro A, Cimino C. Recurrent acute pulmonary embolism and paroxysmal atrial fibrillation associated with subclinical hyperthyroidism. International Journal of Cardiology. 2010; 142(2):e25-6. [DOI:10.1016/j.ijcard.2008.11.179] [PMID]

[5] Akhbour S, Khennine BA, Oukerraj L, Zarzur J, cherti M. Pericardial tamponade and coexisting pulmonary embolism as first manifestation of non-advanced lung adenocarcinoma. Pan African Medical Journal. 2014; 18:15. [DOI:10.11604/pamj.2014.18.15.2469] [PMID] [PMCID]

[6] Thomas C, Lane K, Cecconi M. Pulmonary embolism with haemorrhagic pericardial effusion and tamponade: A clinical dilemma. BMJ Case Reports. 2014; 2014:bcr2013202285. [DOI:10.1136/bcr-2013-202285] [PMID] [PMCID]

[7] Han JY, Seon HJ, Choi IS, Ahn Y, Jeong MH, Lee SG. Simultaneously diagnosed pulmonary thromboembolism and hemopericardium in a man with thoracic spinal cord injury. Journal of Spinal Cord Medicine. 2012; 35(3):178-81. [DOI:10.1179/2045772312Y.0000000010] [PMID] [PMCID]