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Case Report

Massive Pulmonary Embolism Concomitant With Cardiac Tamponade as Initial Presentation of Lung Adenocarcinoma: A Diagnostic and Therapeutic Dilemma

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Pulmonary embolism and cardiac tamponade are potentially fatal acute conditions that rarely present concomitantly in the emergency department (ED). Both require early diagnosis and urgent intervention, and are usually observed as separate easily identifiable diseases. However, in a patient exhibiting a concomitant presentation of pulmonary embolism with cardiac tamponade, diagnosis, and therapeutic intervention are extremely challenging. A 48-year-old woman presented with cardiac tamponade as an initial symptom of an underlying lung adenocarcinoma and masked massive pulmonary embolism (MPE), which led to the development of sudden cardiac arrest after successful pericardiocentesis. She presented with a high index of suspicion for a diagnosis of MPE using echocardiography after successful pericardiocentesis, and this diagnosis was confirmed using computed tomography. Extracorporeal membrane oxygenation and adjusted-dose unfractionated intravenous heparin administration were performed; unfortunately, they were unsuccessful. This report would help ED physicians because this case demonstrates that lung cancer can initially present as pulmonary embolism with cardiac tamponade and pulmonary embolism can be misdiagnosed in the presence of concomitant cardiac tamponade. Bedside echocardiography may fail to diagnose life-threatening MPE with coexisting cardiac tamponade. MPE can also lead to the development of sudden cardiac arrest after successful pericardiocentesis. Thrombolytic and anticoagulant use in MPE with coexisting hemorrhagic cardiac tamponade is a controversial issue. The risk-benefit ratio of both therapies needs to be considered on a case-by-case basis for improved clinical outcomes.

Key words: *pulmonary embolism, cardiac tamponade, echocardiography, pericardiocentesis*

Introduction

Massive pulmonary embolism (MPE) is a serious life-threatening condition in the emergency department (ED). It requires early diagnosis and urgent intervention. MPE can be very easily misdiagnosed in the absence of typical clinical presentations such as dyspnea, chest pain, and hemoptysis.[1] If diagnosis is delayed or the condition is misdiagnosed, MPE can result in significant morbidity and mortality. Without treatment or with delayed treatment, MPE is generally associated with an overall mortality of up to 60%.[2] Generally, bedside Doppler echocardiography is the most useful diagnostic tool in urgent cases of MPE. Thrombolysis is the foundation of therapy for MPE with hemodynamic instability, and anticoagulant agents are required for submassive or non-MPE with hemodynamic stability.[3] However, both forms of treatment are contraindicated in aortic dissection, intracranial tumor, and hemorrhagic cardiac tamponade.[4]

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Here, we present a challenging case of a 48-year-old woman admitted to the ED with cardiac tamponade and respiratory distress that initially masked MPE and led to the development of sudden cardiac arrest after successful pericardiocentesis.

Case Report

A 48-year-old woman without significant comorbidities presented to the ED with two episodes of syncope and progressive dyspnea approximately two weeks prior to presentation. Initial examination revealed that she was afebrile and had a blood pressure of 185/70 mmHg, pulse rate of 138 beats/min, and oxygen saturation of 95% using a pulse oximeter. Her neck veins were markedly distended, and she showed a positive Kussmaul sign. Chest auscultation revealed coarse basilar breath sounds bilaterally with a normal cardiac examination. Bilateral lower limb edema was noted. Electrocardiography revealed sinus tachycardia with prominent low-voltage QRS complexes. Echocardiography showed a large pericardial effusion with Doppler echocardiographic signs of impending tamponade (Fig. 1). Urgent bedside echocardiography-guided pericardiocentesis was performed to address the patient's hemodynamic instability and determine the etiology of the pericardial effusion. Approximately a minute later, she developed sudden cardiac arrest. Cardiopulmonary resuscitation was immediately initiated with the administration of intravenous epinephrine (1 mg) and intubation with ventilatory support. Urgent echocardiography demonstrated a dilated right ventricle (RV), a small left ventricle cavity without spontaneous contractions, and little residual pericardial fluid (Fig. 2). These Doppler echocardiographic findings, persistently elevated right-sided pressures after successful relief of tamponade, and persistent tachycardia with shortness of breath strongly suggested pulmonary embolism. Because her condition continued to deteriorate, extracorporeal membrane oxygenation was initiated which restored hemodynamic stability. Computed tomography revealed a right upper lung lobe mass (62.77 mm), with pleural effusion and bilateral proximal pulmonary embolism (Figs. 3 and 4). Emergency surgical thrombectomy, catheter-directed embolectomy, and emergency inferior vena cava (IVC) filter placement were not recommended by our cardiac surgeon owing to the patient's poor condition. A loading bolus of unfractionated intravenous heparin (5,000 IU) was

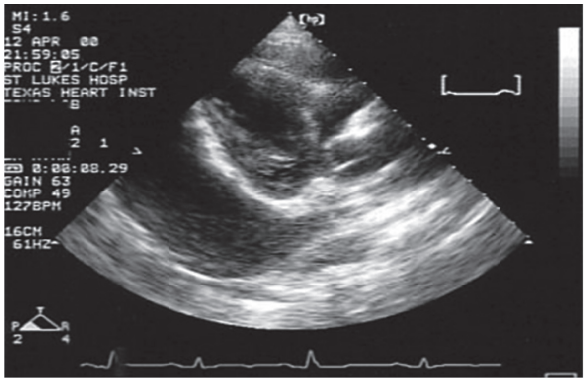

Fig. 1. Echocardiographic image showing a large pericardial effusion and diastolic collapse of the right ventricle.

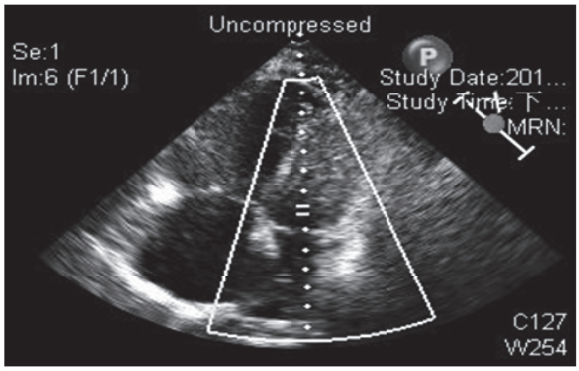

Fig. 2. Echocardiographic image showing a dilated right ventricle (RV) and a small left ventricle cavity without spontaneous contractions.

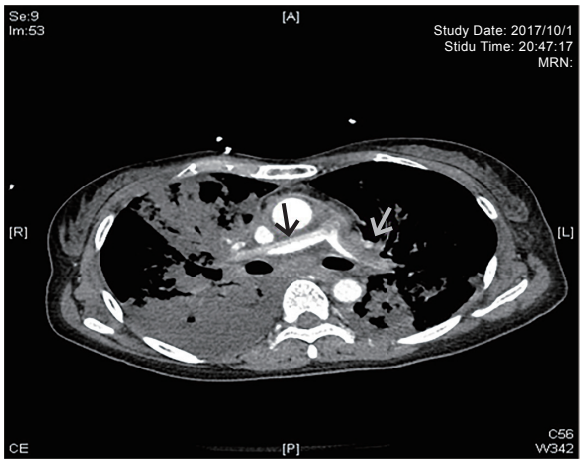

Fig. 3. Contrast-enhanced computed tomographic image (axial view) showing bilateral massive thrombosis (arrows) involving the proximal pulmonary arteries.

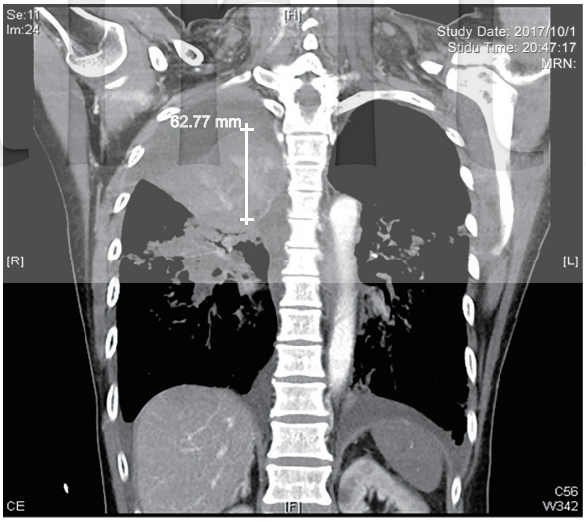

Fig. 4. A chest computed tomography scan (coronal view) showing a mass lesion (62.77 mm) in the right upper lung lobe.

administered followed by continuous infusion at a mean dose of 1,000 IU/h, maintaining the activated partial thromboplastin time at twice its baseline value, and bilateral lower extremity venous compression ultrasonography revealed no evidence of deep vein thrombosis. On the third day of hospitalization, the patient's condition was deteriorating. After considering the risk-benefit ratio of surgical success, her underlying disease condition, and the potential economic consequences, the family opted to discontinue aggressive treatment; however, the patient died the same day. Analysis of the aspirated pericardial fluid indicated malignant pericardial effusion secondary to lung cancer.

Discussion

Pulmonary embolism and cardiac tamponade are life-threatening complications of malignant disease and are uncommon as initial manifestations. To our knowledge, lung cancer initially presenting as MPE and concomitant cardiac tamponade with cardiac tamponade masking MPE and causing sudden fatal MPE following successful pericardiocentesis is rare. MPE is defined as pulmonary embolism with RV failure that causes shock and has either a systolic blood pressure of < 90 mmHg or a pressure drop of at least 40 mmHg for ≥ 15 min.[1] Our patient was definitively diagnosed with MPE based on sudden cardiac arrest and echocardiographic evidence of RV failure. MPE commonly manifests as acute dyspnea, tachypnea, chest pain, hemoptysis, and shock and may lead to sudden death.[2] As observed in this case, the clinical presentation of cardiac tamponade and pulmonary embolism is indistinguishable; when MPE and cardiac tamponade present together, MPE poses a diagnostic challenge because both can have the same clinical picture such as dyspnea, tachycardia, hypotension, and signs of right-sided heart failure, making differential diagnosis difficult. If these conditions present concomitantly, MPE may be misdiagnosed. Classically, cardiac tamponade presents with pericardial effusion and diastolic right atrial and ventricular compression.[5] MPE presents with a dilated RV, RV hypokinesis, paradoxical septal motion (flattening or bowing of the interventricular septum toward the left ventricle), McConnell's sign (RV free wall hypokinesis with apical sparing), tricuspid regurgitation, and persistently elevated right-sided pressures.[6,7] In our patient, echocardiography showed a large pericardial effusion without prominent RV collapse—the absence of the latter (which would be expected secondary to a high RV pressure) is attributable to pulmonary hypertension associated with MPE. In patients with MPE concomitant with cardiac tamponade, a few typical Doppler echocardiographic signs of MPE may be absent, as was observed in our patient, leading to a misdiagnosis of MPE. In the absence of concomitant chronic obstructive pulmonary disease or other conditions causing pulmonary hypertension, pulmonary embolism may be suspected when left-sided heart pressures are observed to drop with steady right-sided pressures after successful pericardiocentesis. This case emphasizes that, in patients presenting with cardiac tamponade and concomitant MPE, echocardiographic diagnosis of MPE is difficult because the concomitant cardiac tamponade can mask the typical echocardiographic signs that are usually associated with MPE. Then, pericardiocentesis may be possibly harmful in MPE with concomitant cardiac tamponade as in our case. The mechanism of cardiac arrest after pericardiocentesis in MPE with concomitant cardiac tamponade in our case is not clear. Perhaps, the removal of pericardial fluid normalizes the pericardial pressure and results in a disproportionate increase in the RV end-diastolic volume pressure along with significant changes in intrapericardial and RA pressure. Before pericardiocentesis, this pulmonary hypertension in a setting of tamponade may have paradoxically saved

this patient's life. After losing the balance between intrapericardial and RV pressure, the pulmonary mainstream obstruction leads to increased RV afterload and right-sided heart failure. RV overload results in a leftward shift of the ventricular septum, leading to decreased left ventricle diastolic filling and cardiac arrest, which occurs through a circulatory failure.

Although thrombolysis is the gold standard treatment for MPE with cardiac arrest, thrombolytics are contraindicated in patients with concomitant cardiac tamponade.[8] Therapeutic recommendations in such patients are unclear, and determining an optimal treatment strategy remains clinically challenging. Surgical or catheter-directed embolectomy and emergency IVC filter placement may be alternatives to thrombolytic therapy;[9] however, the optimal therapeutic approach is dictated by the indications of the procedure and the surgeon's expertise. We chose anticoagulant therapy using adjusted-dose unfractionated intravenous heparin, albeit unsuccessful. Recent reports describe the efficacy of systemic thrombolysis in patients with life-threatening MPE in whom thrombolytics are absolutely contraindicated such as those with recent abdominal aortic aneurysm repair, brain tumors, and/or recent nontraumatic brain hemorrhage.[10,11] However, this is a controversial topic, and no clear guidelines are currently available to treat MPE, with or without cardiac arrest, in patients with absolute contraindications to thrombolytic use. Further investigations and controlled trials are warranted to establish disease-specific guidelines and contraindications for thrombolytic use in patients presenting with MPE, with or without cardiac arrest.

Conflicts of Interest Statement

None.

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Ethics and Consent

The patient, who was not identified in this paper, gave us consent to deal with her case.

References

1. Konstantinides SV, Torbicki A, Agnelli G, et al. Corrigendum to: 2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism. *Eur Heart J* 2015;36:2642. doi:10.1093/eurheartj/ehu479
2. Bailén MR, Cuadra JA, Aguayo de Hoyos E. Thrombolysis during cardiopulmonary resuscitation in fulminant pulmonary embolism: a review. *Crit Care Med* 2001;29:2211-2219. doi:10.1097/00003246-200111000-00027
3. Wan S, Quinlan DJ, Agnelli G, Eikelboom JW. Thrombolysis compared with heparin for the initial treatment of pulmonary embolism: a meta-analysis of the randomized controlled trials. *Circulation* 2004;110:744-749. doi:10.1161/01.CIR.0000137826.09715.9C
4. Long B, Koyfman A. Current controversies in thrombolytic use in acute pulmonary embolism. *J Emerg Med* 2016;51:37-44. doi:10.1016/j.jemermed.2016.02.024
5. Armstrong WF, Feigenbaum H, Dillon JC. Acute right ventricular dilation and echocardiographic volume overload following pericardiocentesis for relief of cardiac tamponade. *Am Heart J* 1984;107:1266-1270. doi:10.1016/0002-8703(84)90289-8
6. Goldhaber SZ. Echocardiography in the management of pulmonary embolism. *Ann Intern Med* 2002;136:691-700. doi:10.7326/0003-4819-136-9-200205070-00012
7. McConnell MV, Solomon SD, Rayan ME, Come PC, Goldhaber SZ, Lee RT. Regional right ventricular dysfunction detected by echocardiography in acute pulmonary embolism. *Am J Cardiol* 1996;78:469-473. doi:10.1016/S0002-9149(96)00339-6
8. Konstantinides S, Geibel A, Olschewski M, et al. Association between thrombolytic treatment and the prognosis of hemodynamically stable patients with major pulmonary embolism. Results of a multicenter registry. *Circulation* 1997;96:882-888. doi:10.1161/01.CIR.96.3.882
9. Fava M, Loyola S, Bertoni H, Dougnac A. Massive pulmonary embolism: percutaneous mechanical thrombectomy during cardiopulmonary resuscitation. *J Vasc Interv Radiol* 2005;16:119-123. doi:10.1097/01.RVI.0000146173.85401.BA
10. Koroneos A, Koutsoukou A, Zervakis D, et al. Successful resuscitation with thrombolysis of a patient suffering fulminant pulmonary embolism after recent intracerebral haemorrhage. *Resuscitation* 2007;72:154-157. doi:10.1016/j.resuscitation.2006.06.019
11. Keir SL, Wardlaw JM, Sandercock PA, Chen Z. Anti-

thrombotic therapy in patients with any form of intracranial haemorrhage: a systematic review of the available controlled studies. *Cerebrovasc Dis* 2002;14:197-206. doi:10.1159/000065661