Development of massive pulmonary embolism during echocardiographic imaging

A case report

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Abstract
Rationale: Pulmonary embolism (PE) is a common diagnostic consideration for patients who present to the emergency department (ED) with chest pain, dyspnea, or both. In addition, PE has a very high mortality in patients who are hemodynamically unstable. An electrocardiography, bedside transthoracic echocardiogram, and computed tomography pulmonary angiogram are usually performed to confirm the diagnosis.

Patient concerns: A 53-year-old man was admitted to the cardiology clinic with complaints of dyspnea, chest pain, and general weakness after walking. He had a history of hypertension and smoking.

Diagnosis: During synchronous recording of echocardiographic images, a large mobile thrombus detached from the right atrium, and first embozilized to the right ventricle and then to the main pulmonary artery from the right heart chambers. Soon after, shortness of breath developed which clinically worsened the patient. Transthoracic echocardiogram which demonstrated the thrombus in the pulmonary artery or right heart chambers was suspected of causing acute massive PE.

Interventions: The patient was transferred to Critical Care Unit for monitoring; 100 mg of alteplase was initiated immediately and alleviated the hemodynamic instability within 2 hours of intravenous administration.

Outcomes: To the best of our knowledge, this is the first synchronous echocardiographic recording showing the embolization of a thrombus from the right atrium, first to the right ventricle and then to the main pulmonary artery.

Lessons: Transthoracic echocardiography provides a safe, rapid, and noninvasive diagnostic tool for evaluation of suspected massive PE. Thrombolytic therapy is useful for treating acute massive PE that leads to hemodynamic instability.

Abbreviations: BP = blood pressure, ECG = electrocardiography, ED = emergency department, LV = left ventricle, PE = pulmonary embolism, r-tPA = recombinant tissue plasminogen activator, RV = right ventricle, TTE = transthoracic echocardiogram.

Keywords: acute pulmonary embolism, fibrinolytic therapy, transthoracic echocardiography

1. Introduction
Pulmonary embolism (PE) is a common diagnostic consideration for patients who are admitted to EDs with chest pain, dyspnea, or both. In addition, PE has a very high mortality in patients who are hemodynamically unstable.

An electrocardiography (ECG), bedside transthoracic echocardiogram (TTE), and computed tomography pulmonary angiogram are performed to confirm the diagnosis of PE. Transthoracic echocardiography rarely confirms the diagnosis of pulmonary embolism visualizing a thrombus in the pulmonary artery or right heart chambers.

Prompt diagnosis and appropriate therapy for unstable patients may reduce the mortality of massive PE. Thrombolytic therapy is very useful in treating acute massive PE. This case report is about synchronous recording of embolization of thrombus.

2. Case presentation
A 53-year-old man with a history of hypertension and smoking was admitted to cardiology clinic with complaints of dyspnea, chest pain, and general weakness after walking. At admission, his blood pressure (BP) was 100/60 mm Hg, the pulse rate was 90/min, and the respiratory rate was 20/min. ECG showed sinus tachycardia with heart rate of 100 beats/min.

The TTE demonstrated a normal left ventricle (LV) function, whereas the right ventricle (RV) appeared markedly dilated and hypokinetic. There was leftward displacement of the interventricular septum, increased systolic pulmonary arterial pressure (85 mm Hg) and a big mass was floating in
con deep vein thrombosis. Transthoracic echocardiography rarely obtained and con hospitalization period, chest computed tomography was systolic pressure was estimated to be 48mm Hg. During the function with an LV ejection fraction of 65%. Pulmonary artery moderately depressed RV systolic function, and normal LV transthoracic echocardiography revealed mild RV dilatation with heparin (5000 unit bolus; 1000unit/h) was started. Formal 90% on 3 to 4L/min of oxygen. An intravenous unfractionated Hg, heart rate 90beats/min, and oxygen saturation remained at hours. Upon completion of the infusion, the BP was 110/70mm 100mg alteplase was administered via intravenous infusion in 2 patient was transferred to Critical Care Unit for monitoring, and lungs were clean. Due to severe hemodynamic instability, the patient was tachycardic, the heart rate was rhythmic, and the 464eca.png

Figure 1. Chest computed tomography showing massive bilateral pulmonary embolism.

the right atrium, protruding through the tricuspid valve into the RV in diastole.

During echocardiographic imaging, a large mobile thrombus which was attached to the free wall of the right atrium detached and was embolized to the pulmonary (Video 1, http://links.lww.com/MD/C192). Thereafter, shortness of breath developed and the patient’s clinic was worsened. Presenting vital signs were as follows: BP 85/60mm Hg, heart rate 110beats/min, respiratory rate 24breaths/min, and SpO2 85%. On cardiac examination, the patient was tachycardic, the heart rate was rhythmic, and the lungs were clean. Due to severe hemodynamic instability, the patient was transferred to Critical Care Unit for monitoring, and 100mg alteplase was administered via intravenous infusion in 2 hours. Upon completion of the infusion, the BP was 110/70 mm Hg, heart rate 90beats/min, and oxygen saturation remained at 90% on 3 to 4 L/min of oxygen. An intravenous unfractionated heparin (3000 unit bolus; 1000unit/h) was started. Formal transthoracic echocardiography revealed mild RV dilatation with moderately depressed RV systolic function, and normal LV function with an LV ejection fraction of 65%. Pulmonary artery systolic pressure was estimated to be 48 mm Hg. During the hospitalization period, chest computed tomography was obtained and confirmed diagnosis of massive bilateral pulmonary embolism (Fig. 1). Venous duplex ultrasound of the lower extremities scans was found to be normal. The patient was discharged after 5 days of hospitalization. Ethics committee approval was not necessary because the case presentation was a retrospective study. The patient was informed of his situation and a signed consent form was obtained.

3. Discussion

The patient had risk factors for venous thromboembolism including hypertension and smoking,[4] and no physical signs of deep vein thrombosis. Transthoracic echocardiography rarely confirms a diagnosis of pulmonary embolism by visualizing a thrombus in the pulmonary artery or right heart chambers.[13,6] In the presence of hemodynamic instability, thrombus in the pulmonary artery or right heart chambers should evoke suspicion of massive PE enabling a prompt diagnosis and appropriate treatment.

Our patient had markedly dilated and hypokinetic RV with leftward displacement of the interventricular septum, and increased systolic pulmonary arterial pressure (85 mm Hg). During echocardiographic imaging, a big mass floating in the right atrium and protruding through the tricuspid valve into the right ventricle in diastole was embolized to the pulmonary artery, and the event was recorded in a synchronous manner.

Thrombolytic therapy is very useful for treating the acute massive PE which is a cause of hemodynamic instability.[5] Thrombolytic therapy with 100 mg of r-tPA administered in 2 hours via intravenous administration is recommended in patients with persistent hypotension due to massive PE or recurrent PE, despite anticoagulation (class I, level B).[7] In this case alteplase, which was initiated immediately and administered intravenously in 2 hours, alleviated the hemodynamic instability.

4. Conclusions

To the best of our knowledge, this is the first synchronous echocardiographic recording showing the embolization of a thrombus from right atrium, first to the RV and then to the main pulmonary artery. Transthoracic echocardiography provides a safe, rapid, and noninvasive diagnostic tool for evaluation of suspected massive PE in many clinical scenarios. Thrombolytic therapy is useful for treating acute massive PE that causes patient’s hemodynamic instability.

Author contributions

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