Clinical Case Report

Medicine®

OPEN

High-risk pulmonary embolism assessed by transthoracic echocardiography

A case report

Jiahong Wu, MD[a], Jing Zhang, MD[a], Fangfang Yang, MD[a], Chuanbao Li, MD, PhD[b,*], Mei Ni, MD, PhD[c,*]

Abstract

Rationale: Acute pulmonary embolism (APE) as a life-threatening illness may present with a wide range of manifestations. APE was diagnosed using computed tomographic pulmonary angiography (CTPA); however, transthoracic echocardiography (TTE) can reveal hemodynamic status. Early thrombolysis is the most effective therapy for the treatment of massive pulmonary embolism.

Patients concerns: Herein, we report a case of high-risk APE with a wide range of manifestations, including chest pain, dyspnea, low-blood pressure, and syncope.

Diagnoses: A 55-year-old, previously healthy woman, complained of dyspnea and pleuritic chest pain for 40 days, along with transitory (10 minutes) episodes of syncope that had occurred 2 days previously.

Interventions: Because of the high-risk APE, the patient received intravenous thrombolytic therapy with low-dose recombinant tissue plasminogen activator (rt-PA, 50 mg over 30 minutes) and an anticoagulant (subcutaneous low-molecular-weight heparin, once every 12 hours for 5 days).

Outcomes: Five days after thrombolysis, bedside TTE revealed RV diastolic dimension decreased to 22 mm. Color ultrasonography revealed a significant decrease in systolic and mean pulmonary artery pressure.

Lessons: TTE may provide initial suspicion of APE and may help identify patients with unstable hemodynamic status before the onset of shock. Moreover, concomitant TTE signs of decreased RV load may predict better prognosis for high-risk APE patients.

Abbreviations: APE = acute pulmonary embolism, CT = computed tomographic, CTPA = computed tomographic pulmonary angiography, RV = right ventricular, TTE = transthoracic echocardiography.

Keywords: CT, pulmonary embolism, thrombolysis, transthoracic echocardiography

1. Introduction

Acute pulmonary embolism (APE) is a potentially life-threatening cardiovascular emergency. The severity of APE is directly related to the early mortality risk. According to the APE-related death, APE is divided into high-risk and non-high-risk.[1] With high-risk APE, the mortality risk is >15%,and thrombolysis and anticoagulation therapy must be administered as early as possible.[2,3] The markers for risk stratification assessment in the clinic include hemodynamic status, signs of right ventricular (RV) dysfunction, and myocardial injury. Here, we report a case of high-risk APE with unstable hemodynamic status. Bedside transthoracic echocardiography (TTE) revealed classical signs of RV overload. The patient's hemodynamic status improved significantly after early thrombolysis and anticoagulant therapy.

2. Case presentation

The patient provided informed consent for the publication of her clinical and radiological data. This case report was approved by Medical Ethical Committee of Qilu Hospital, Shandong University.

A 55-year-old, previously healthy woman, complained of dyspnea and pleuritic chest pain for 40 days, along with transitory (10 minutes) episodes of syncope that had occurred 2 days previously. On admission, her body temperature was 36.0°C; pulse wave was 98 beats/min (bpm); respiratory rate was 18 breaths/min; and blood pressure was 98/61 mm Hg. Slight cyanosis on the lips was observed. Bilateral respiratory movements were identical. Breath sounds were diminished and no moist rales were heard. The heart rhythm was regular, with pulmonary second sound > aortic second sound (P2>A2). No edema was found in the lower extremities.

Arterial blood gas analysis showed the following results: pH, 7.43; PaO_2, 52 mm Hg; $PaCO_2$,31 mm Hg; and HCO_3, −22.5 mmol/L (with 5 L/min flow rate of oxygen inhalation). The leukocyte count was 6.82×10^9 cells/L. The level of cardiac troponin I was 0.011 μg/L; CK-MB, 8.78 ng/m; pro-BNP, 3425 pg/mL; and D-dimer level,2.47 μg/mL. Electrocardiography revealed signs of RV strain, including inversion of T waves in leads V1–V3 and the classical S1Q3T3 type.

Editor: N/A.

Funding: This work was supported by the Science Foundation of Shandong Province, China (grant number ZR2014CM010), and the Postdoctoral Science Foundation of China (grant number 2017M612293).

The authors have no conflicts of interest to disclose.

[a] Department of Radiology, Qilu Hospital of Shandong University, [b] Department of Emergency, Chest Pain Center, [c] The Key Laboratory of Cardiovascular Remodeling and Function Research, Chinese Ministry of Education and Chinese Ministry of Health, Qilu Hospital of Shandong University, Jinan, Shandong, China.

[*] Correspondence: Chuanbao Li and Mei Ni, Qilu Hospital of Shandong University, 107 Wen Hua Xi Road, Jinan, Shandong 250012, China (e-mails: nimei999@163.com; bao2460@126.com).

Medicine (2018) 97:18(e0545)

Received: 17 January 2018 / Accepted: 3 April 2018

http://dx.doi.org/10.1097/MD.0000000000010545

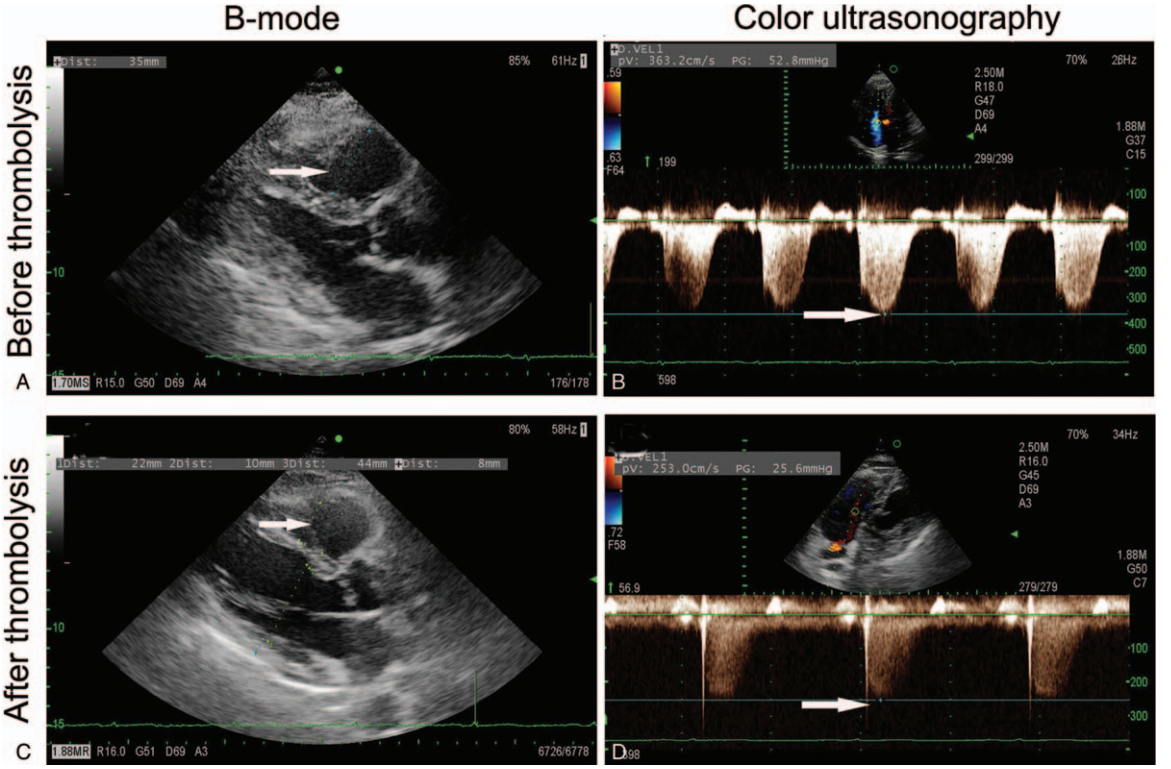

Figure 1. Echocardiography of 55-year-old woman presenting heart pain. (A and B) Before thrombolysis therapy: arrow indicates dilated right ventricle (A), severe tricuspid regurgitation and pulmonary hypertension (67.8 mm Hg, B), arrow indicates PGTI; (C and D) Five days after thrombolysis therapy: arrow indicates decreased right ventricle diastolic dimension (C), decreased systolic pulmonary artery pressure to 35.6 mm Hg, arrow indicates PGTI (D).

Bedside TTE revealed RV overload characterized by a dilated RV (parasternal view, RV diastolic dimension: 35 mm, Fig. 1A) and systolic flattening of the interventricular septum in B-mode. The McConnell sign indicated hyperkinesia of the apical segment of the RV free wall with hypokinesia of the remaining parts of the RV free wall. Color Doppler ultrasound revealed severe tricuspid regurgitation and severe pulmonary hypertension; the systolic pulmonary arterial pressure was 67.8 mm Hg (Fig. 1B). Enhanced chest CT scan revealed filling defects in the right and left main pulmonary arteries (Fig. 2A and B), as well as bilateral pleural effusion. Because most of the published data are related to deep vein thrombosis, we used lower extremity venous ultrasound and detected bilateral muscle vein thrombosis.

The diagnosis was APE. Because of the high-risk APE, the patient received intravenous thrombolic therapy with low-dose recombinant tissue plasminogen activator (rt-PA, 50 mg over 30 minutes) and an anticoagulant (subcutaneous low-molecular-weight heparin, once every 12 hour for 5 days). The vitamin K antagonist warfarin was administered as soon as possible and preferably on the same day as the initial anticoagulant. Warfarin (3 mg per dose) was administered orally once a day, and the dose was adjusted to maintain the international normalized ratio at a target of 2.5 (range: 2.0–3.0). Other treatments included anti-infective, oxygen, and nutrition support therapies.

Arterial blood gas analysis, 12 hours after thrombolic therapy, revealed a pH of 7.43, PaO_2 of 76 mm Hg, $PaCO_2$ of 38 mm Hg, and HCO_3 of −25.2 mmol/L (under 5 L/min oxygen inhalation). Three days later, the hypoxemia remarkably improved, and arterial blood gas analysis revealed a pH of 7.40, PaO_2 of 87 mm Hg, $PaCO_2$ of 38 mm Hg, and HCO_3 of −24.1 mmol/L (under 3 L/min oxygen inhalation). Five days after thrombolysis, bedside TTE revealed RV diastolic dimension decreased to 22 mm (parasternal view, Fig. 1C). Color ultrasonography revealed a significant decrease in pulmonary artery pressure and systolic pulmonary arterial pressure, to 35.6 mm Hg (Fig. 1D).

Fifteen days after thrombolysis, enhanced chest CT scan revealed no thrombosis in the right and left main pulmonary artery branch (Fig. 2C and D). Three months after hospital discharge, color ultrasonography revealed normal values for both RV heart load and pulmonary artery pressure. The McConnell sign had disappeared.

3. Discussion

Early diagnosis of high-risk APE is vital because immediate treatment is very effective and potentially lifesaving. With advances in studying RV function, echocardiography is a valuable tool for risk-stratification patients with APE.[4] The diagnosis of high-risk APE may be based on the indirect echocardiographic findings of RV dysfunction accompanied by other noninvasive diagnostic approaches including investigating myocardial injury markers. RV dysfunction was found to be an independent predictor of adverse in-hospital outcomes, and in the overall population.[5] Three different sets of echocardiographic criteria are potentially useful for diagnosing APE: RV overload, disturbed RV ejection pattern (the 60–60 sign), and depressed contractility of the RV free wall as compared with its apex (the McConnell sign).[6] Among them, RV overload criteria showed high sensitivity and the 60–60 and McConnell signs showed high specificities for APE diagnosis. The combination of these signs may indicate a hemodynamically compromised patient with suspected PE if bedside TTE is available. Kurzyna et al[6] evaluated 100 consecutive patients with clinical suspicion of APE, including those with previous cardiovascular

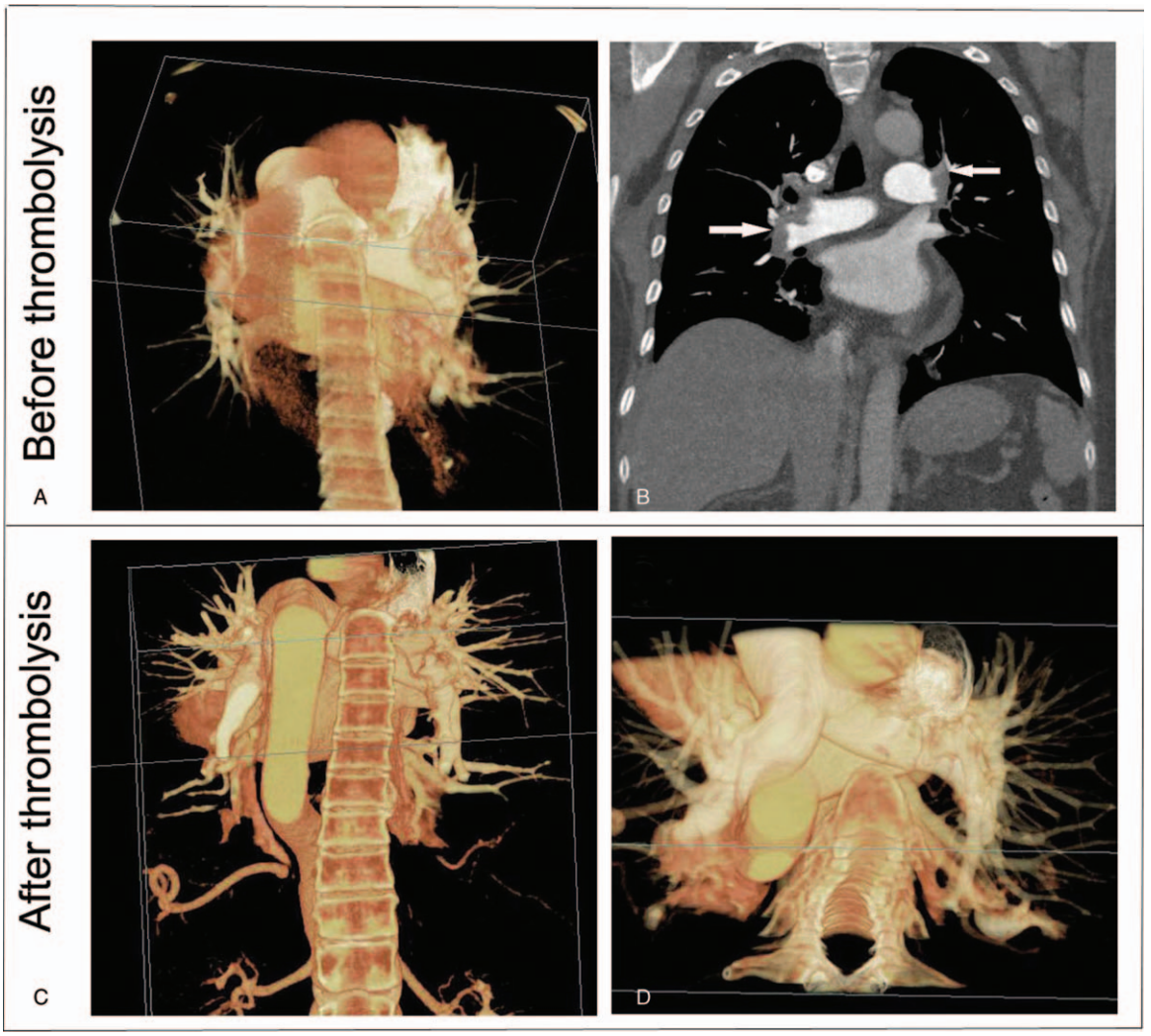

Figure 2. Enhanced chest CT scan. (A and B) Before thrombolysis therapy: arrows indicate filling defects in the right and left main pulmonary arteries; (C and D): Fifteen days after thrombolysis therapy: no thrombosis in the right and left main pulmonary artery branch.

and respiratory disease, and found that combinations of the 60–60 and McConnell signs were 94% specific and 36% sensitive in diagnosing APE (6). The 60–60 and McConnell signs are reliable and helpful in bedside diagnosis of APE when direct visualization of the pulmonary arteries is not possible. RV overload on echocardiography is not specific for APE.

4. Conclusions

TTE may offer initial suspicion of APE and may help identify patients with unstable hemodynamic status before the onset of shock. Moreover, concomitant TTE signs of decreased RV load may predict better outcomes for high-risk APE patients.

Acknowledgments

We would like to thank the native English speaking scientists of Elixigen Company (Huntington Beach, California) for editing our manuscript.

Author contributions

Conceptualization: Jiahong Wu, Chuanbao Li.
Data curation: Jing Zhang.
Investigation: Fangfang Yang.
Resources: Jiahong Wu.
Writing – review & editing: Chuanbao Li, Mei Ni.

References

[1] Torbicki A, Perrier A, Konstantinides S, et al. Guidelines on the diagnosis and management of acute pulmonary embolism: the Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC). Eur Heart J 2008;29: 2276–315.
[2] Serra R, de Franciscis S. The role of thrombolysis for patients with hemodynamically stable acute pulmonary embolism. Thromb Res 2014; 134:7–8.
[3] Niwa A, Nakamura M, Harada N, et al. Observational investigation of thrombolysis with the tissue-type plasminogen activator monteplase for acute pulmonary embolism in Japan. Circ J 2012;76:2471–80.
[4] Pavlidis AN, Kallistratos MS, Karamasis GV, et al. Diagnosis and risk stratification in acute pulmonary embolism: the role of echocardiography. Rev Cardiovasc Med 2013;14:56–65.
[5] Becattini C, Agnelli G, Germini F, et al. Computed tomography to assess risk of death in acute pulmonary embolism: a meta-analysis. Eur Respir J 2014;43:1678–90.
[6] Kurzyna M, Torbicki A, Pruszczyk P, et al. Disturbed right ventricular ejection pattern as a new Doppler echocardiographic sign of acute pulmonary embolism. Am J Cardiol 2002;90:507–11.