ISSN 1975-4612

www.kse-jcu.org

CASE REPORT J Cardiovasc Ultrasound 2008;16(4):123-125

A Case of Consecutive Right and Left Ventricular Dysfunction

Seong-Mi Park, MD, PhD, Jong-Il Choi, MD, PhD, Soon-Jun Hong, MD, PhD, Do-Sun Lim, MD, PhD and Wan-Joo Shim, MD, PhD

Division of Cardiology, Korea University College of Medicine, Seoul, Korea

An acute pulmonary embolism (PE) and the apical ballooning syndrome (ABS) are both critical and need proper management during the acute stage. We experienced a case of recurrent severe dyspnea because serious right ventricular dysfunction due to PE and left ventricular dysfunction due to ABS occurred consecutively in the short-term and bedside echocardiography has an important role in management in acute settings.

KEY WORDS: Pulmonary embolism · Apical ballooning syndrome · Bleeding · Echocardiography.

Introduction

Both an acute pulmonary embolism (PE) and apical ballooning syndrome (ABS) can be presented with severe dyspnea. Moreover, PE is known as one of the triggering factors for the ABS and it is not easy to differentiate the ABS from acute myocardial infarction in acute clinical setting. Here, we present the case of dyspneic patient with consecutive severe right and left ventricular dysfunction.

Case

A 56-year-old woman was brought to the emergency department for progressive severe dyspnea for of 5 days. She had hypertension, but did not receive medical treatment. The heart rate was 100 beats/min, the blood pressure was 80/50 mmHg, and the respiratory rate was 30 breaths/min. An arterial blood gas analysis (ABGA) showed an arterial oxygen saturation of 89%, a $PaCO_2$ of 30 mmHg, and a PaO_2 of 50 mmHg on room air. She was intubated and received mechanical respiratory support. The electrocardiogram showed sinus tachycardia and T-inversion in multiple leads. A chest radiograph disclosed cardiomegaly with a prominent pulmonary artery trunk. A transthoracic echocardiogram (TTE) demonstrated a significantly enlarged right ventricle (RV) with decreased function, but normal left ventricular (LV) function with no regional wall motion abnormalities (Fig. 1). The pulmonary artery systolic pressure was 74 mmHg and the ventricular septum was flattened. An acute PE was highly suspected as the diagnosis and intravenous heparin was initiated with an 80 U/kg bolus and 18 U/kg/h infusion. Thrombolysis was not chosen as a treatment because she had evidence of active gastric bleeding in the Levin tube. A computed tomography (CT) of the chest obtained a few hours subsequently showed extensive filling defects in both the main pulmonary artery and the peripheral branches and an acute PE was confirmed. On hospital day 5, the dyspnea had improved and she was extubated. The echocardiogram showed that the right overloading was much relieved, with a pulmonary artery systolic pressure of 34 mmHg and normal LV function.

On hospital day 7, she complained of right flank pain and an abdominal CT revealed a subcapsular and perirenal hematoma and contrast leakage suggested active extravasation. Her hemoglobin was 7.4 g/dL and decreased further to 5.8 g/dL in spite of a blood transfusion. She underwent an emergent coil embolization for perirenal bleeding.

The next day, she had sudden severe dyspnea again and her electrocardiogram showed ventricular fibrillation. A bedside TTE showed that right overloading was much improved, but LV function was significantly depressed with an ejection fraction of 25%. The LV showed akinetic mid-to-apical walls and a balloon-like apex (Fig. 2). The cardiac enzymes were mildly increased (CK-MB, 19.1 ng/mL; troponin, 0.31 ng/mL) and an ABGA showed an arterial oxygen saturation of 73.3%, a $PaCO_2$ of 72.2 mmHg, and a

• Received: July 14, 2008 • Accepted: October 16, 2008
• Address for Correspondence: Wan-Joo Shim, Division of Cardiology, Korea University College of Medicine, 126-1 Anam-dong 5-ga, Seongbuk-gu, Seoul 136-705, Korea Tel: +82-2-920-5445, Fax: +82-2-927-1478, E-mail: wjshimmd@unitel.co.kr

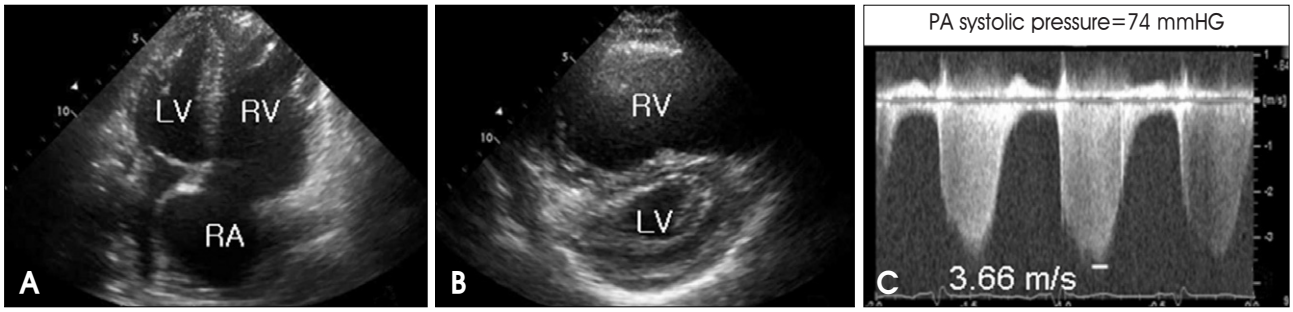

Fig. 1. Initial transthoracic echocardiography for severe dyspnea. Significantly enlarged RV and RA and D-shaped LV were observed (A, B). Calculated PA systolic pressure is 74 mmHg (C). She was diagnosed with acute pulmonary embolism. RV: right ventricle, RA: right atrium, LV: left ventricle, PA: pulmonary artery.

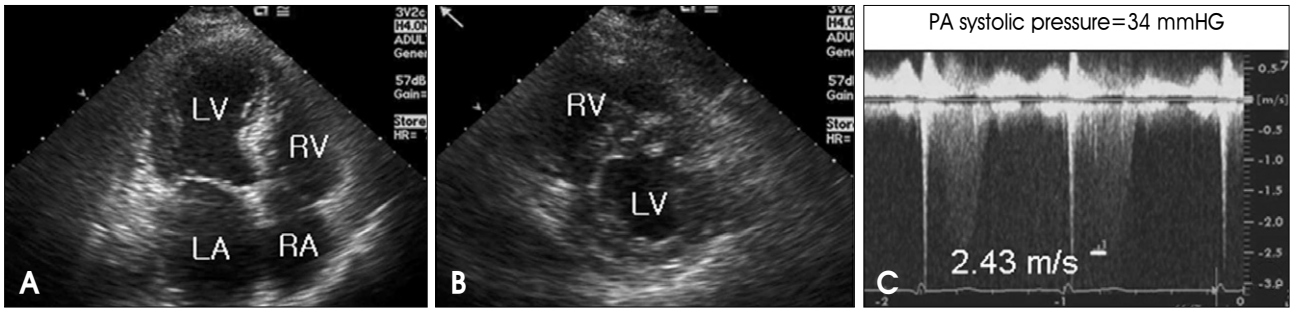

Fig. 2. Follow-up transthoracic echocardiography for second episode of severe dyspnea. On hospital day 8, RV overloading is much improved with a decreased PA systolic pressure of 34 mmHg (C). In contrast, LV and LA are enlarged. LV has a balloon-like akinetic apex and hyperkinetic basal walls (A, B). She was diagnosed with apical ballooning syndrome. RV: right ventricle, RA: right atrium, LV: left ventricle, LA: left atrium, PA: pulmonary artery.

PaO_2 of 28.1 mmHg on room air. She was treated for the ABS and heart failure with an ACE inhibitor and diuretics. Coronary angiography showed no significant coronary artery stenosis to cause severe LV dysfunction and she completely recovered from the ABS by her follow-up TTE 2 weeks later. A follow-up abdominopelvic CT demonstrated a persistent intra-abdominal hematoma and thrombi in both iliac veins and the inferior vena cava. An inferior vena caval filter was placed to prevent further PE.

Discussion

This patient had consecutive, not concomitant, serious RV and LV dysfunction, and a PE and the ABS were diagnosed. These two diseases are both critical and need proper management in the acute stage. A PE is known as one of the triggering factors for the ABS.[1)2)] However, this patient presented with the ABS in a resolving state of a PE and the two clinical entitites did not seem to be directly related to each other. When she had severe dyspnea again, a worsening PE was suspected first because the PE was not treated with thrombolysis. However, the echocardiogram showed that the RV dysfunction due to the PE was much improved, but the LV function was significantly depressed. The findings on ABGA were quite different from that of a PE. Acute massive intra-abdominal bleeding with an emergent procedure was thought to be the cause of the ABS, which is also known as one of the triggering factors.[1)3)]

Comprehensive and repeated TTE can provide information to determine appropriate treatment and to assess the progress of the acute PE. A massive PE is associated with RV enlargement, RV free-wall hypokinesis with preservation of apical contractility, dilation of the pulmonary arteries, and an elevated RV systolic pressure (a surrogate for pulmonary artery systolic pressure).[4-7)] The clinical presentations of the ABS mimic acute coronary syndrome with typical wall motion abnormalities of the LV apical walls like a balloon, which can be demonstrated by TTE and should be confirmed by coronary angiography.[1)3)]

Both an acute PE and the ABS may be lethal if appropriate treatment is delayed. Bedside TTE of this patient indicated which ventricular dysfunction led to the recurrent dyspnea and differentiated the ABS from acute coronary syndrome in the critical stage. There is a report regarding the appropriate criteria of TTE and it is considered as an 'Appropriate test with highest score 9' for performing TTE in both acute PE and the ABS, especially in the acute setting.[8)] This case shows that serious RV and LV dysfunction can occur consecutively in the short-term and TTE has an important role in management in acute settings.

References

1. Tsuchihashi K, Ueshima K, Uchida T, Oh-mura N, Kimura K, Owa M, Yushiyama M, Miyazaki M, Haze M, Kai R, Morii I: Angina pectoris-Myocardial infarction Investigations in Japan. *Transient left ventricular apical ballooning without coronary artery stenosis: a novel heart syndrome mimicking acute myocardial infarction. J Am Coll Cardiol 2001;38:11-8.*
2. Kai R, Yasu T, Fujii M, Kubo N, Ohta M, Yutani C, Saito M. *Apical ballooning by transient left ventricular dysfunction (so-called "ampulla" cardiomyopathy) associated with therapy for acute pulmonary thromboembolism: a case report. J Cardiol 2001;38:41-6.*
3. Abe Y, Kondo M, Matsuoka R, Araki M, Kohyama K, Tanio H. *Assessment of clinical features in transient left ventricular apical ballooning. J Am Coll Cardiol 2003;41:737-42.*
4. Ten Wolde M, Sohne M, Quak E, Mac Gillavry MR, Buller HR. *Prognostic value of echocardiographically assessed right ventricular dysfunction in patients with pulmonary embolism. Arch Intern Med 2004; 164:1685-9.*
5. Lee JH, Park JH. *Role of echocardiography in acute pulmonary embolism. J Cardiovasc Ultrasound 2008;16:9-16.*
6. Miniati M, Monti S, Pratali L, Di Ricco, Marini C, Formichi B, Prediletto R, Michelassi C, Di Lorenzo M, Tonelli L, Pistolesi M. *Value of transthoracic echocardiography in the diagnosis of pulmonary embolism: Results of a prospective study in unselected patients. Am J Med 2001; 110:528-35.*
7. Pavan D, Nicolosi GL, Antonini-Canterin F, Zanuttini D. *Echocardiography in pulmonary embolism disease. Int J Cardiol 1998;65 Suppl 1: S87-90.*
8. *ACCF/ASE/ACEP/ASNC/SCAI/SCCT/SCMR 2007 appropriateness criteria for transthoracic and transesophageal echocardiography: a report of the American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group, American Society of Echocardiography, American College of Emergency Physicians, American Society of Nuclear Cardiology, Society for Cardiovascular Angiography and Interventions, Society of Cardiovascular Computed Tomography, and the Society for Cardiovascular Magnetic Resonance. Endorsed by the American College of Chest Physicians and the Society of Critical Care Medicine. J Am Soc Echocardiogr 2007;20:787-805.*