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Case Report

CASE REPORT

Saddle pulmonary embolism: right ventricular strain an indicator for early surgical approach

Vinod Namana[1,*], Sabah Siddiqui[2], Ram Balasubramanian[1], Rajeswer Sarasam[3], and Vijay Shetty[1,2]

[1]Department of Cardiology, Maimonides Medical Center, Brooklyn, NY, USA, [2]Department of Medicine, Maimonides Medical Center, Brooklyn, NY, USA, and [3]Department of Medicine, Baystate Medical Center, Springfield, MA, USA

*Corresponding address. Department of Cardiology, Fellow in Cardiovascular Medicine, 4802 10th Avenue, Brooklyn, NY 11219, USA. Tel: +718-283-6892; Fax: +718-635-7436; E-mail: vnamana@maimonidesmed.org

Abstract

Current mainstay treatment for pulmonary embolism (PE) includes oral anticoagulation, thrombolytic therapy, catheter embolectomy and acute surgical embolectomy. Surgical embolectomy is reserved for hemodynamically unstable patients (cardiogenic shock, cardiac arrest) and contraindication to thrombolytic therapy. We report a case of saddle PE in a young female with echocardiographic signs of right ventricular (RV) dysfunction who underwent early acute surgical embolectomy with a positive outcome. It would be beneficial to use bedside echocardiography even in hemodynamically stable patients to determine RV strain as this could act as an early indicator suggesting the escalation of therapy.

INTRODUCTION

Pulmonary embolism (PE) is the third most frequent cardiovascular disease in United States [1]. More than 100 000 cases are reported annually and 25% present with sudden death, which makes it an important cause of morbidity and mortality [2]. Acute PE therefore warrants a quick diagnosis, risk stratification and should be treated aggressively [1]. We report a case of saddle PE in a young female with echocardiographic signs of right ventricular (RV) strain who underwent early acute surgical embolectomy with a positive outcome and also discuss the role of echocardiography and early surgical approach.

CASE

A 47-year-old woman presented to emergency room after an episode of presyncope at home. The day prior to the presentation, she woke up with cramps in her left leg, which was relieved with Ibuprofen. She has no significant past medical history. She exercises on treadmill every day for 30 min, never used any prescription medications and reported no use of oral contraceptive pills. She is self-employed involving manufacturing of luggage tags. Family history is noncontributory.

On arrival, she was anxious, alert, fully oriented and talking in full sentences. She was normotensive (110/80 mmHg), normothermic, tachycardic (120 beats per min), tachypneic (22 breaths per min) and saturating 98% on 2 l of oxygen via nasal cannula. Her physical examination was significant for raised jugular venous pressure to 15 cmH_2O, holosystolic high-pitched murmur of Grade III/VI in intensity in left lower sternal border that was best heard on inspiration. Lung auscultation revealed minimal diffuse rales bilaterally. Radial and pedal pulses were intact. Right lower extremity had 1+ pitting edema and Homans' sign was positive. Upper and lower extremities were warm to touch, and there was no cyanosis.

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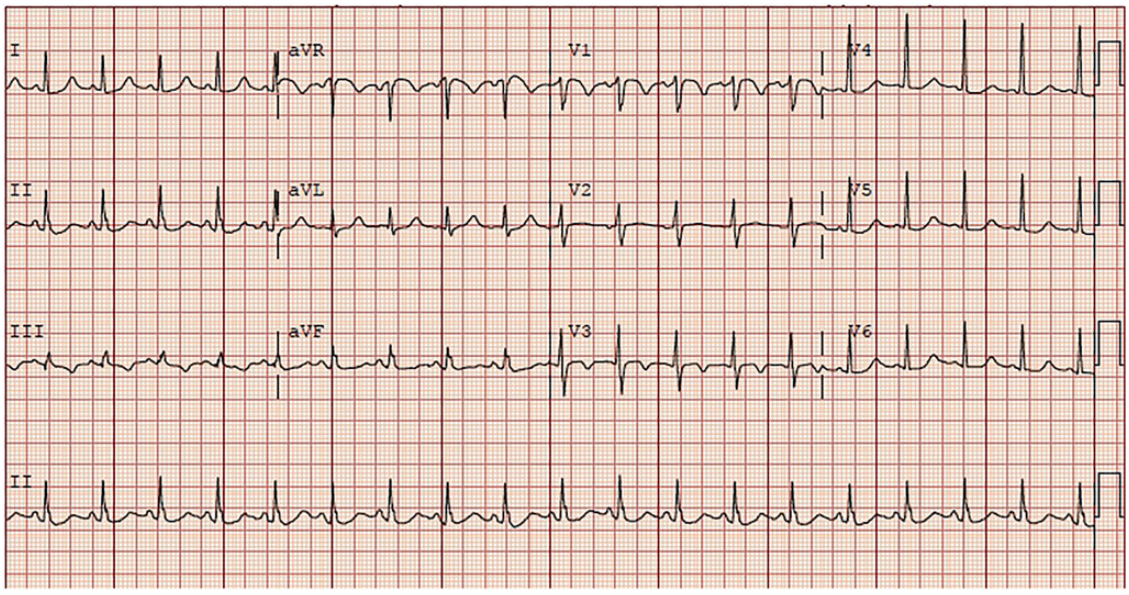

Figure 1: Electrocardiogram on presentation showing sinus tachycardia with a heart rate of 115 beats per minute, T wave inversions in leads III and V3.

Her electrocardiogram (Fig. 1) on presentation showed sinus tachycardia with a heart rate of 115 beats per minute, T wave inversions in leads III and V3 and chest X-ray showed a widened superior mediastinum (Fig. 2). Labs were significant for elevated white blood cell count (15 000), initial troponin I was 0.83 ng/ml (normal 0–0.04), which increased to 1.99 and d-dimer on presentation was >5000 ng/ml DDU. Her initial arterial blood gas on 40% FiO_2 (5 l via nasal cannula) was pH: 7.31, PCO_2: 31 mmHg, PO_2: 231 mmHg, HCO_3: 15.6 mEq/l, SPO_2: 100% and the calculated A-a gradient was 15 mmHg (normal <15 mmHg) and 6 h later the arterial blood gas on 100% FiO_2 (15 l via nonrebreather facemask) was pH: 7.24, PCO_2: 28 mmHg, PO_2: 296 mmHg, HCO_3: 12 mEq/l, SPO_2: 100% and the calculated A-a gradient was 382 mmHg.

Our suspicion for PE was high, and bedside echocardiogram was performed. It showed moderate to severe RV enlargement, moderately hypokinetic right ventricle, abnormal atrial and ventricular and septal motion and mild pulmonary hypertension (Fig. 3). Computed tomography (CT) of the chest with intravenous contrast (Fig. 4) confirmed our diagnosis of acute PE and showed 'saddle embolus' involving the main pulmonary artery extending into both right and left pulmonary arteries and associated secondary and tertiary branches.

Venous duplex scan was positive for an acute infra-popliteal occlusive thrombus in the right calf muscular vein and also positive for an acute occlusive superficial vein thrombosis involving cephalic vein from ante-cubital fossa to the mid arm.

Because of the presence of submassive saddle embolus, RV dysfunction, evidence from the Aklog *et al.* [3], Ahmed *et al.* [4] and Leacche *et al.* [5] studies and the presence of in hospital cardiothoracic surgery team decision was made to proceed with surgical embolectomy. Patient underwent pulmonary embolectomy, and large saddle embolus was removed (Figs 5 and 6). Patient was weaned off inotropes on post-operative day (POD) 2 and extubated on POD 3. Subsequent echocardiogram showed normalization of RV function. Patient was discharged home on POD 6 with oral anticoagulation and a referral to Hematology service.

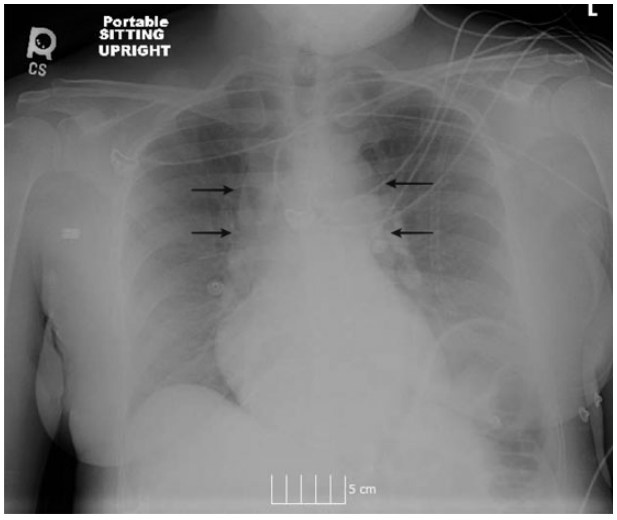

Figure 2: Anteroposterior chest X-ray on presentation showing widened mediastinum (pointing black arrows).

DISCUSSION

Venous thromboembolism (VTE) includes acute PE as well as deep vein thrombosis (DVT) [1]. It is a life-threatening condition that usually results in death if not diagnosed early and treated aggressively [6]. Virchow's triad still forms the best framework for understanding the pathogenesis of VTE. It encompasses modifications in three factors: stasis, hypercoagulability and an injury to the endothelium of the blood vessel wall [7]. The thrombi that then form in the venous system detach from their formation sites and embolize through the right atrium and right ventricle (RV) toward the pulmonary circulation [1].

PE results in elevation of RV afterload, and a subsequent increase in RV wall tension that may lead to dilatation, dysfunction causing decreased right coronary artery flow and increased RV

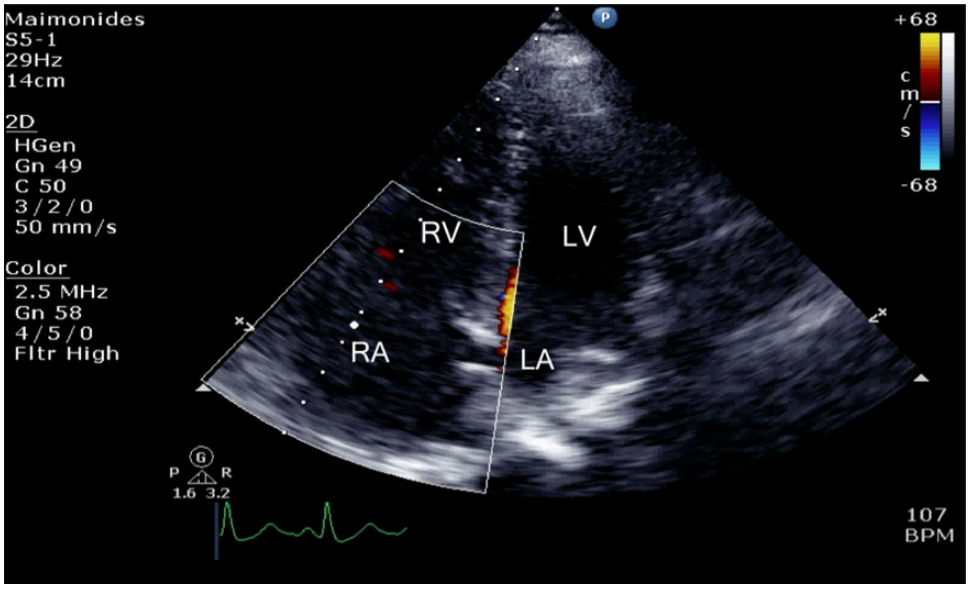

Figure 3: Echocardiogram: apical four-chamber view showing dilated right ventricle (RV). RA, right atrium; LA, left atrium; LV, left ventricle.

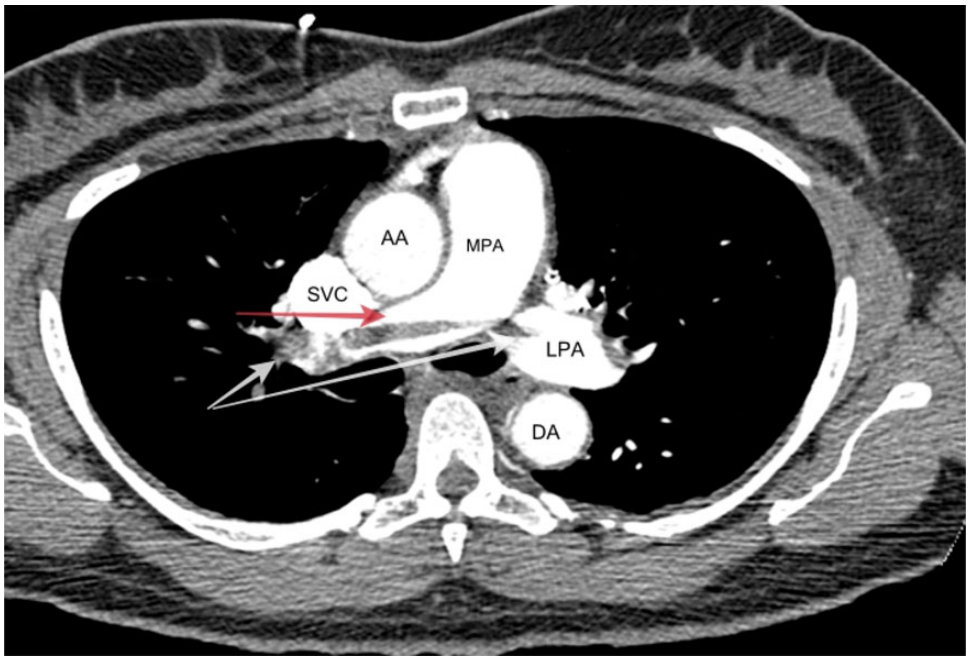

Figure 4: CT of the chest showing saddle emboli (white arrows) in main pulmonary artery (MPA), right pulmonary artery (red arrow) and left pulmonary artery (LPA). AA, ascending aorta; SVC, superior vena cava; DA, descending aorta.

myocardial oxygen demand. Ischemia of RV also occurs as left ventricle compression leads to decreased cardiac output and coronary perfusion. Death eventually results from RV failure [1].

CT and echocardiography can be used to aid in the diagnosis of acute PE. Use of echocardiography in diagnosis and management of hemodynamically unstable patients has been recommended by the European Society of Cardiology (ESC) [8]. However, there were no guidelines on the use of echocardiography in hemodynamically stable patients [8]. Patients with RV strain have more than 2-fold increase in risk of early mortality compared with patients with no signs of RV strain [9].

Current mainstay treatment of PE includes anticoagulation, thrombolytic therapy, catheter embolectomy and acute surgical embolectomy [6, 10]. Limitations of medical therapy include inability to significantly reduce mortality in patients with massive PE [10]. Acute surgical embolectomy is reserved for hemodynamically unstable patients (cardiogenic shock, cardiac arrest) and contraindication to thrombolytic therapy [6, 8].

In our case, patient was hemodynamically stable with echocardiogram and CT scan showing signs of RV strain and saddle embolus, respectively. However, we have taken early surgical approach, based on location, size and extent of emboli and impending hemodynamic collapse (signs of RV dysfunction).

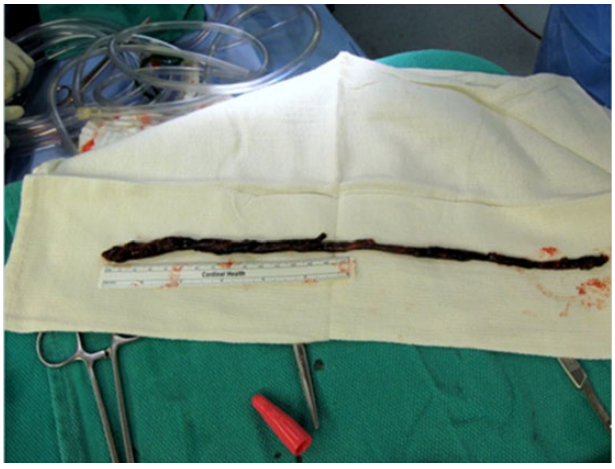

Figure 5: Gross specimen of saddle pulmonary embolus.

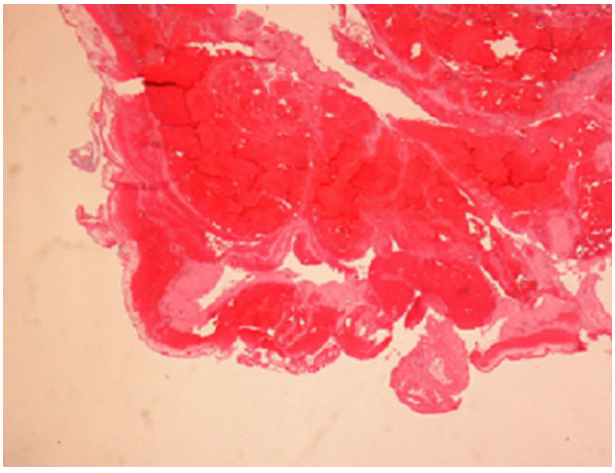

Figure 6: Histopathology of pulmonary thromboemboli.

Surgical embolectomy has been reported to be associated with high mortality rates, but this can be attributed to the fact that it is reserved for a very high-risk population [6, 11]. Studies have shown that rescue surgical embolectomy led to a better in-hospital course when compared with repeat thrombolysis in patients with massive PE who have not responded to thrombolysis [8, 12].

In addition, a study done by Aklog *et al.* [3] noted a high number of intracranial hemorrhages among patients with PE who were treated with thrombolytic therapy in the International Cooperative PE Registry. This study broadened criteria for surgical embolectomy to include patients with anatomically extensive PE and moderate to severe RV dysfunction despite preserved hemodynamics. It reported the largest single-center contemporary experience with emergency pulmonary embolectomy in a 2-year period, with a high survival rate of 89% that they attributed to aspects of the surgical technique, rapid diagnosis, treatment and careful patient selection [3]. The study found that RV strain is associated with an increased rate of mortality and recurrent PE [3]. In the studies of Ahmed *et al.* [4] and Leacche *et al.* [5], patients with impending hemodynamic instability with moderate or severe RV dysfunction underwent early surgical embolectomy with good outcomes. The benefits of surgical intervention diminish sharply 24 h following acute PE diagnosis, and so they proposed strong consideration of early surgical approach in patients with early hemodynamic changes and RV strain [4, 5].

The European Society of Cardiology, 2014, guidelines on the diagnosis and management of acute PE recommend risk stratification after the diagnosis of PE without hemodynamic compromise [8]. This includes assessment of RV function by echocardiography or CT angiography and cardiac troponin. Patients with RV dysfunction and positive troponin are classified as intermediate-high-risk group and recommended considering full-dose systemic thrombolytic therapy to prevent hemodynamic decompensation or collapse (IIa recommendation) [8, 13, 14]. Surgical pulmonary embolectomy or percutaneous catheter-directed treatment may be considered as alternative, 'rescue' procedures for patients with intermediate-high-risk PE, in whom hemodynamic decompensation appears imminent and the anticipated bleeding risk under systemic thrombolysis is high (IIb recommendation) [8].

There has been a trend toward more expedient surgery based on the recognition that RV strain is an indication of impending circulatory collapse and increased risk of mortality [3–5]. Therefore, early echocardiography even in hemodynamically stable patients would be useful as identification of RV strain can alert clinicians and allow more aggressive therapy, including surgical embolectomy as in our case, to be considered with potentially improved mortality.

CONFLICT OF INTEREST STATEMENT

None declared.

FUNDING

None.

ETHICS APPROVAL

Maimonides Medical Center gave permission.

CONSENT

Informed consent was taken from patient.

GUARANTOR

V.N. and S.S. are guarantors of this work.

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