case report

Life-saving percutaneous intervention in young patient with massive pulmonary embolism

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Massive pulmonary embolism (PE) is not an uncommon condition, and it usually carries a high risk of mortality. It is one of the fatal conditions that commonly affect young patients. A definitive treatment for patients with massive PE is still lacking, and surgical intervention carries a substantial mortality risk. Thus, percutaneous intervention (clot fragmentation and/or aspiration) remains an option in some patients, specifically in those with a risk of bleeding, contraindicating the use of thrombolysis. There have been no randomized trials to validate percutaneous intervention in massive PE. A sufficient level of evidence is still lacking, and its use depends upon the expert committee's opinion and study of previous case reports. We present a 23-year-old man with first onset massive PE secondary to protein C deficiency, who was treated successfully with the combination of systemic thrombolysis and percutaneous interventions.

Pulmonary embolism (PE) is a common, serious and potentially fatal disease. The most severe form of PE, which is massive PE, accounts for about 5% of cases, and is defined by the presence of a large central PE with hypotension, with a systolic blood pressure of <90 mm Hg. It carries a high mortality rate, estimated between 17.4% and 28%, particularly during the first few hours after admission.[1,2] It is believed that the mortality is caused by acute severe right heart failure resulting in cardiogenic shock. Despite this high mortality rate, the treatment of major PE has not been well standardized and remains controversial and elusive. Several modalities of treatment have been implemented upon treating massive PE in particular. Both the British Thoracic Society and the American College of Chest Physicians have developed guidelines that systemic thrombolysis is the mainstay of treatment in massive PE.[3] However, in the event that thrombolysis is contraindicated, one can consider thrombectomy via surgery or percutaneous intervention, depending on the local settings, as there is no head-to-head study that compares the two methods.

CASE

A 23-year-old nursing student, presented to our emergency department with a 3 day-history of worsening of shortness of breath and hemoptysis. He had been well until 1 month earlier when he developed a mild shortness of breath on regular activities associated with cough and minimal hemoptysis. He did not seek medical help until 2 weeks prior when he was diagnosed with pneumonia and treated with oral antibiotics at an outpatient clinic. On completing the antibiotics, the patient reported worsening of the symptoms. On the morning prior of the presentation, the shortness of breath suddenly became intolerable, and he reported of difficulty in talking, associated with pleuritic chest pain. He also had coughed out more fresh blood along with some blood clots. He was then brought to this center by ambulance.

He gave no history of any previous medical illness or pulmonary tuberculosis. He also claimed to have normal appetite and had maintained his weight quite reasonably. On arrival at the emergency department, his vitals were as follows: temperature was 37.2°C; the pulse rate was 156 beats per minute, regular; the respiratory rate was 50 breaths per minute; and the blood pressure was 82/50 mm Hg. Oxygen saturation was only 85% on air. On examination, his breathing was shallow because of chest pain. The lungs revealed coarse crackles over the left lower zone, with no heart

murmurs. The rest of the extremities appeared normal.

Arterial blood gases, while the patient was breathing on the high flow mask of 15 L oxygen, revealed severe hypoxemia with the pH of 7.39 (normal blood pH, 7.35-7.50); oxygen partial pressure (PaO_2) of 64 mm Hg (normal values PaO_2 80-100 mm Hg); carbon dioxide partial pressure ($PaCO_2$) of 40 mm Hg (normal values $PaCO_2$ 35-45 mm Hg) and a bicarbonate level of 24 mmol/L. Because of severe respiratory failure, he was mechanically ventilated in the emergency department. The electrocardiography (**Figure 1**) revealed sinus tachycardia, right bundle branch block, and an S1Q3T3 pattern. The chest radiograph (**Figure 2**) revealed a wedge-shaped opacity in the left lower lobe, which is a finding of pulmonary infarction (Hampton hump) at the left lung.

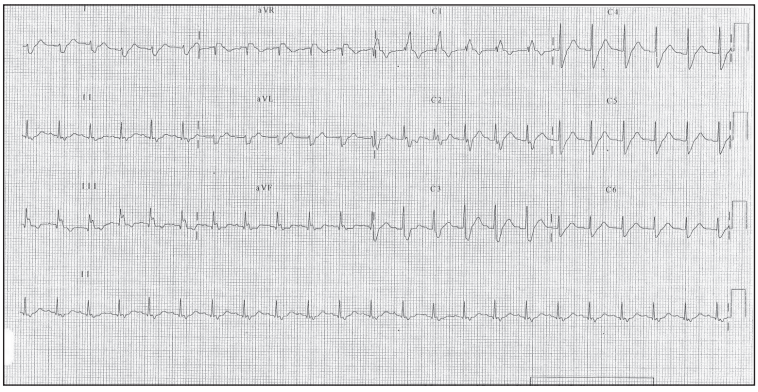

Figure 1. Electrocardiography of the patient in the case report showing sinus tachycardia, right bundle branch block and S1Q3T3 pattern.

His CT pulmonary angiogram (**Figure 3**) demonstrated a large thrombus in the right main pulmonary artery (PA). A thrombus was also seen in the proximal left descending artery with a segment of the left lower lobe consistent with pulmonary infarction. The diagnosis of massive PE was made. Surgical services were unavailable at the time of embolectomy, and because of the presence of a contraindication to thrombolysis (hemoptysis), we decided to bring him to the cardiac invasive laboratory. In the cardiac invasive laboratory, a right femoral vein puncture was made and a 14-F myelin sheath was inserted. Using a Cournand catheter, a 0.035 guidewire was inserted into the main PA; the catheter was then changed to a pigtail catheter. The PA pressure was recorded (as shown in **Figure 4**) as 62/35 mm Hg (normal, 8-20 mm Hg) and the systemic pressure as 75/40 mm Hg.

The right and left pulmonary angiograms were performed and shown in **Figures 5 and 6**, respectively. The right pulmonary angiogram showed a huge filling defect, obliterating the distal main right pulmonary trunk. Likewise, the left pulmonary angiogram showed a filling defect in the lower left artery.

Then, a 14-F myelin sheath was inserted into the main PA for support. A Balloon OPTA PRO 7×14 mm (Cordis Corporation, Bridgewater, NJ, United States) (**Figure 7**) was inflated to compress and passed through the thrombus several times (clot fragmentation). The result is shown in **Figure 8**. A significant amount of thrombus was still visible; however, the

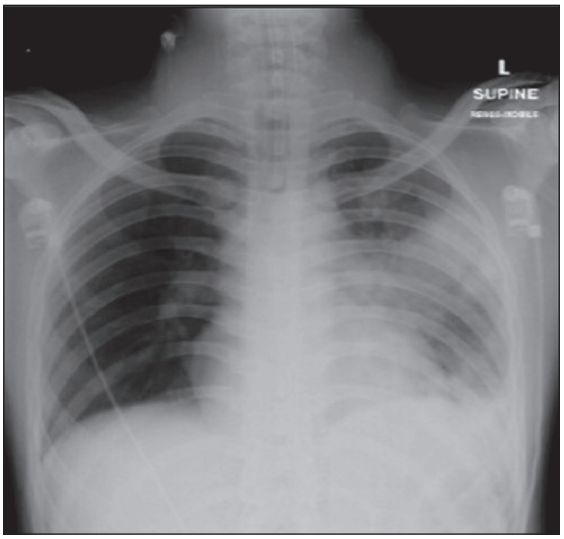

Figure 2. Chest radiograph of the patient in the case report revealing a wedge-shaped opacity in the left lower lobe which is a finding of pulmonary infarction (Hampton hump) at the left lung.

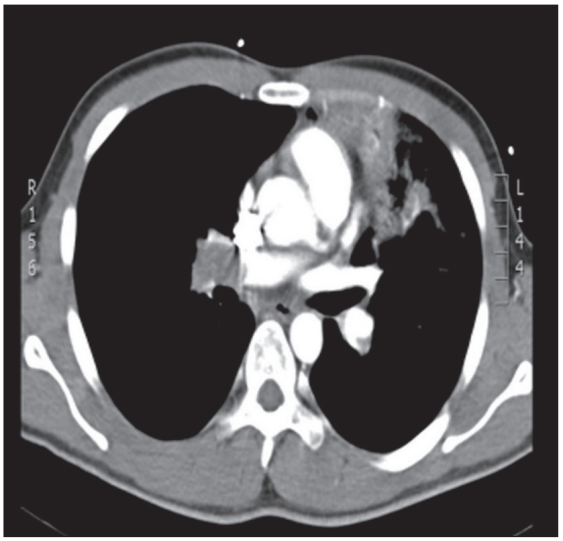

Figure 3. CT pulmonary angiogram of the patient in the case report revealing a large thrombus in the right main pulmonary artery.

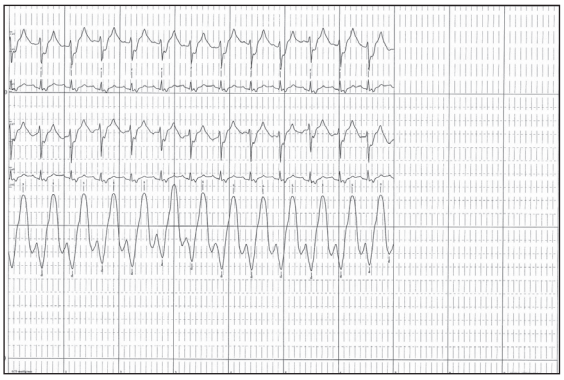

Figure 4. Pressure tracing of pulmonary artery with pressure recorded as 62/35 mm Hg.

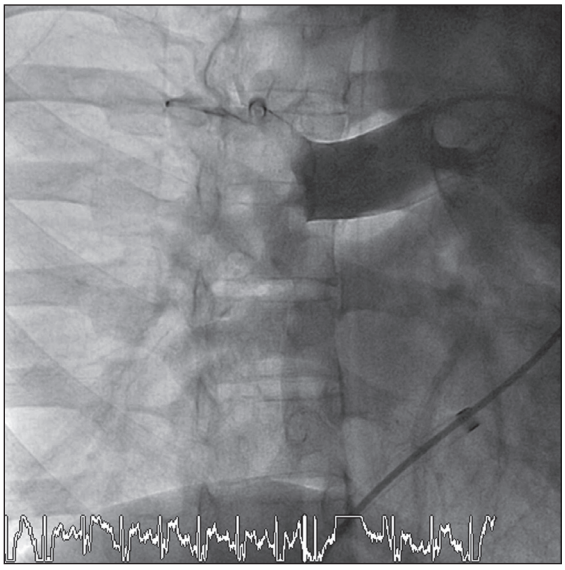

Figure 5. Right pulmonary angiogram showing huge filling defect obliterating the distal main right pulmonary trunk.

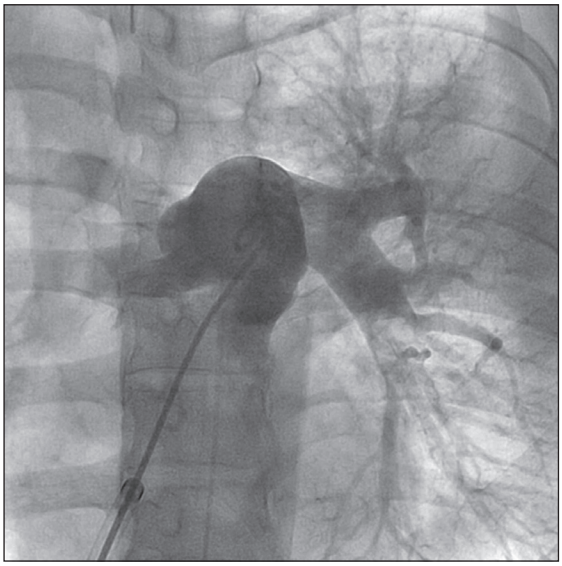

Figure 6. Left pulmonary angiogram showing a filling defect in the lower left artery.

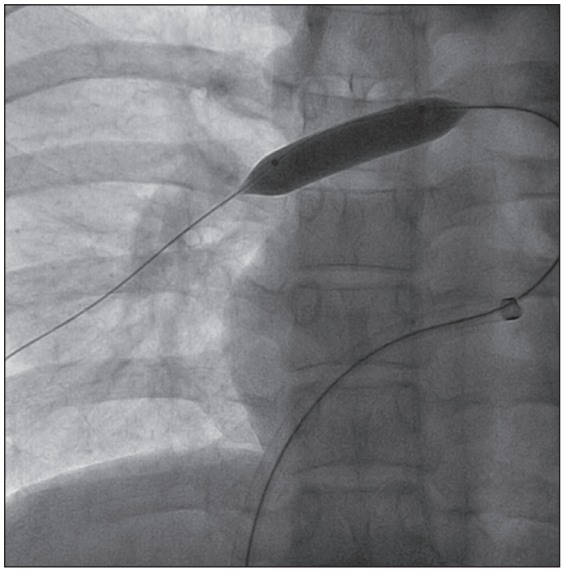

Figure 7. Balloon clot fragmentation done with OPTA PRO 7×14 mm (The balloon was inflated to compress and passed through the thrombus a several times).

acute occlusion was relieved, and a much improved blood flow could be appreciated in the right pulmonary arterial system.

After the procedure, the patient was transferred to the cardiac care unit (CCU). He was then started on intravenous streptokinase 1.5 MU over 1 hour in the CCU for 2 days. We did not adopt the standard regimen of continuous infusion of streptokinase because of the high risk of bleeding. On day 3, hemodynamic parameters were still unchanged as the patient still needed maximal inotropic support. His echocardiogram showed a very dilated and hypokinetic right ventricular chamber with an elevated pulmonary systolic pressure of 50 mm Hg (normal, 8-20 mm Hg); the left ventricle ejection fraction was 70%, and no pericardial effusion was noted. We decided to repeat the pulmonary angiogram (**Figure 9**). It showed some small residual thrombus in the right PA, but much improved blood flow compared to that previously recorded. However, we had given 50 mg recombinant tissue plasminogen activator bolus intrapulmonary during this procedure. The patient then made a steady recovery over the next 7 days. He was extubated on the 10th day of admission. The thrombophilia study confirmed the protein C deficiency, and he was commenced on

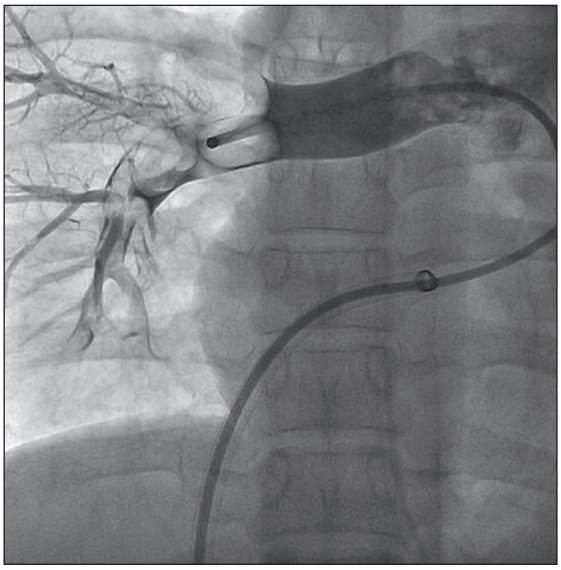

Figure 8. Result after clot fragmentation with Balloon OPTA PRO 7×14 mm, showing a significant amount of thrombus, but much improved blood flow in the right pulmonary arterial system with relief in acute occlusion.

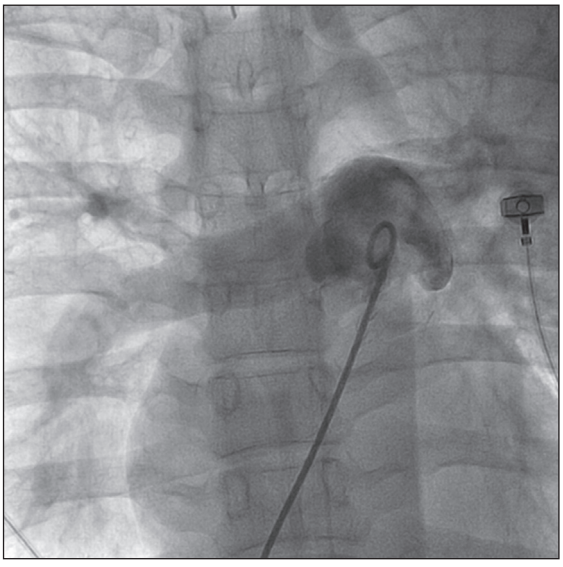

Figure 9. A repeat pulmonary angiogram showing small residual thrombus in the right pulmonary artery

lifelong warfarin. The patient was discharged on the 14th day and last seen in clinic in August 2010. Screening of the family members was arranged in the outpatient clinic.

DISCUSSION

The aim of treatment in massive PE is to relieve the acute right heart failure by lysing or fragmentizing the thrombus, thus restoring cardiac and pulmonary functions. In the presence of contraindication to thrombolysis because of the bleeding risk, surgical or percutaneous interventions have been advocated. To date there is no evidence in the form of randomized controlled trials to compare these two modalities. Arguably, one may advocate thrombolysis with tissue plasminogen activator (tPA) as the best treatment for this patient, as hemoptysis is not an absolute contraindication to thrombolysis. However, tPA is not readily available in our hospital and needs to be ordered from the company, which could certainly result in a significant delay of definitive treatment. Furthermore, transfer to the cathlab was not an issue in terms of delaying the treatment, since the patient presented during working hours and the cathlab was readily available at the time.

The mortality in massive PE is largely contributed by the acute right heart failure. Thus, one can improve survival chances by relieving the pulmonary arterial obstruction. In this patient, during the second angiogram, the burden of the thrombus had already been significantly reduced, and that was before tPA administration. The tPA was given anyway with the intention to lyse whatever residual thrombus that might still be present at the time.

This case illustrated the success of percutaneous thrombus fragmentation using balloon, combined with systemic and intrapulmonary thrombolysis. This technique is potentially life saving when thrombolysis is contraindicated, and especially in peripheral centers where surgical services are not available.

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