Simple mechanical thrombectomy with intrapulmonary arterial thrombolysis in pulmonary thromboembolism: a small case series

Khurshid Ahmed¹, Muhammad Munawar¹,², Dian Andina Munawar¹, Beny Hartono¹

¹Binawaluya Cardiac Center, Jakarta 13750, Indonesia
²Department of Cardiology and Vascular Medicine, Faculty of Medicine, University of Indonesia, Jakarta 10430, Indonesia

Abstract

Pulmonary thromboembolism (PTE) is a life-threatening condition with a high early mortality rate caused by acute right ventricular failure and cardiogenic shock. We report a series of three patients who presented with acute and subacute submassive PTE. They were successfully treated by simple catheter-based mechanical thrombectomy and intrapulmonary arterial thrombolysis. Mechanical fragmentation and aspiration of thrombus was performed by commonly used J-wire, multi-purpose and Judkin Right guiding catheters and this obviated the need of specific thrombectomy devices.

Keywords: Thrombectomy; Pulmonary thromboembolism; Thrombolysis

1 Introduction

Pulmonary thromboembolism (PTE) is a relatively common cardiovascular emergency and massive PTE has always been a major source of morbidity and mortality.¹⁻³ The standard medical management for massive PE is systemic thrombolysis and surgical thrombectomy. The percutaneous catheter-based mechanical thrombectomy and intrapulmonary arterial thrombolysis is a promising alternative for the management of massive and sub-massive PTE, particularly when patients have contraindication with systemic thrombolysis are not suitable for surgery or when urgent recanalization of PTE is warranted. The traditional window period for thrombolysis in patients presenting with acute PTE is two weeks.⁴

We present a series of three patients with sub-massive PTE, out of which two patients had acute and one patient subacute presentation. They had undergone catheter-based pulmonary embolectomy and intrapulmonary thrombolysis. All the patients made full recovery after the procedure and they were successfully discharged from the hospital in haemodynamically stable condition. In our cases, we utilized commonly used J-wire and guiding catheters (GCs) for mechanical fragmentation of thrombus and its aspiration and this obviated the need of specific thrombectomy devices.

The procedures were performed in this case series of three patients according to the declaration of Helsinki. Informed written consent for the procedures was obtained from all patients and permission to carry out the procedures had been sought from the hospital authorities (institutional review board number 002/DK/RSBW/III/2011).

2 Case Report

2.1 Case 1

A 49-year-old Indonesian male, hypertensive and diabetic, presented with a 3-week history of progressive dyspnea and chest pain specially during breathing and one week history of haemoptysis prior to admission. There was no history of leg swelling and/or hyperemia.

On presentation, he was haemodynamically stable with blood pressure 130/86 mmHg, oxygen saturation > 95% on room air, normal jugular venous pulsation (JVP) and had no clinical evidence of deep vein thrombosis (DVT). The electrocardiogram (ECG) showed normal sinus rhythm. Laboratory analysis revealed a fibrin degradation test (D-dimer) was 4.08 μg/mL.
Computed tomography of pulmonary angiogram (CTPA) showed a large filling defect in the right pulmonary artery (PA) and a minimal filling defect in the left PA (Figure 1, Pre procedure, Case 1). Right ventricle (RV) was dilated. Cardiac catheterization was performed via right femoral vein access and an 8Fr vascular sheath was inserted. A GC 6Fr Multipurpose A-2 (MPA-2) (Medtronic Inc., Minneapolis, USA) was advanced to main PA and PA pressure 105/48 mmHg was recorded. Initial diagnostic runs were performed using a 6Fr pigtail catheter (Cordis, Johnson & Johnson Company, USA) in the main PA with the help of injector, contrast volume 30 mL/s, in AP projection that demonstrated large thrombi in right PA. Then, catheter-directed intervention was proceeded. A selective right pulmonary arteriography was performed using guiding catheter (GC) MPA-1/8Fr (Cordis, Johnson & Johnson Company, USA) that also revealed the presence of extensive thrombus. Aspiration and fragmentation of the thrombus was performed first with GC MPA-1/8Fr and then with JR3.5/8 Fr (Terumo Co., Tokyo, Japan). The reason why we used both GCs MPA-1 and JR3.5 was because each of these two GCs has preference for reaching to certain branches of PA. With this technique, most of the major branches of PA could be reached and therefore more thrombus could be fragmented and aspirated. This maneuver was repeated in the left PA. Following aspiration, a significant amount of thrombus material was collected and immediately PA pressure reduced to 45/20 mmHg. After ensuring flow across PA, a bolus of 250,000 units of Streptokinase was given in each right and left PAs in the occlusion site over 10 min and then followed by a maintenance dose of 100,000 units per hour for the next 10 h through pigtail catheter kept in the main PA. Repeat CTPA 48 h post-procedure showed resolution of thrombus in left PA, but there was still small thrombus in right PA (Figure 1, Post procedure 48 hours, Case 1).

Figure 1. Computed tomography of pulmonary angiogram relates to cases 1, 2 and 3. (A): Pre-procedure. Computed tomography of pulmonary angiogram showing large filling defect in right pulmonary artery (big arrow) and minimal filling defect in left pulmonary artery (small arrow) (Case 1); Computed tomography of pulmonary angiogram showing thrombus in right pulmonary artery (big arrow) and left pulmonary artery (small arrow) (Case 2); computed tomography of pulmonary angiogram showing large filling defect in right pulmonary artery (big arrow) and minimal filling defect in left pulmonary artery (small arrow) (Case 3). (B): Post-procedure 48 h. Computed tomography of pulmonary angiogram showing filling defect in right pulmonary artery (arrow) (Case 1); computed tomography of pulmonary angiogram showing thrombus in right pulmonary artery (big arrow) and left pulmonary artery (small arrow) (Case 2); computed tomography of pulmonary angiogram showing filling defect in right pulmonary artery (arrow) (Case 3).
Patient was successfully discharged with dual antiplatelet therapy for 12 weeks. In three months and six months clinical follow up patient was in good condition, asymptomatic and in NYHA functional class I.

2.2 Case 2

A 37-year-old man, non-hypertensive and non-diabetic, presented with chest pain and dyspnea at rest for the last one week prior to admission. His hemodynamic status was stable. Blood pressure was 126/80 mmHg. Clinically, there were no signs of overt heart failure and DVT. ECG showed sinus rhythm with T-wave inversion in V1-4. Lab investigation showed D-dimer 5 μg/mL. CTPA showed extensive thrombi in both left and right PAs (Figure 1, Pre-procedure, Case 2) and dilatation of RV. Percutaneous catheter-based intervention was performed via right femoral vein access with 8Fr vascular sheath. GC 6Fr MPA-2 was advanced to main PA and showed PA pressure of 121/50 mmHg. Initial diagnostic runs were performed using a 6Fr pigtail catheter in the main PA with the help of injector, contrast volume 30 mL/s, in anteroposterior projections and left anterior oblique projections, which demonstrated large thrombi in both left and right PAs (Figure 2A and 2B). GC MPA-1/8Fr and JR 3.5/8Fr were used for thrombus fragmentation and aspiration in left (Figure 2C) and right PAs (Figure 2D), respectively.

Following aspiration, a significant amount of thrombus material was collected and patient’s PA pressure significantly reduced to 66/10 mmHg. Subsequently, a bolus of 250,000 units of Streptokinase was given in each right and left PAs over 10 min, and then followed by a maintenance dose of 100,000 units per hour for the next 10 h through pigtail catheter kept in main PA. Repeat CTPA was performed 48 h after the procedure that still showed thrombus in both PAs but much less compared with pre procedure (Figure 1, post-procedure 48 h, Case 2). Hospital stay was uneventful and the patient was successfully discharged with dual antiplatelet therapy for eight weeks. Follow up CTPA at three months showed complete resolution of thrombus in both PAs (Figure 3A). There was also T-wave resolution in precordial leads in ECG at 3 months (Figure 3B).

2.3 Case 3

A 70-year-old male presented with sudden onset of shortness of breath for the last 24 h prior to admission. His physical examination was unremarkable except for raised JVP. Blood pressure was 124/80 mmHg. There were no clinical signs of DVT. ECG showed sinus rhythm with T-wave inversion in V1-3. Lab investigation showed D-dimer 0.62 μg/mL. CTPA showed extensive thrombus in right PA and small thrombus in left PA (Figure 1, Pre-procedure Case 3) and also RV dilatation.

An 8Fr vascular sheath was inserted into the right femoral vein. Cardiac catheterization with GC MPA-1/8Fr showed PA pressure of 48/12 mmHg. A selective right pulmonary arteriography showed large thrombus (Figure 4A). Subsequently, thrombus aspiration of right PA was performed by GCs MPA-1/8Fr (Figure 4B) followed by JR3.5/8Fr as mentioned above. Initially to fragment the thrombus mechanically a knuckle J-wire technique was used (Figure 4C) and then a 6Fr pigtail catheter was advanced through the MPA –1/8Fr into the occlusion site and its tip was gently rotated to fragment the clot during aspiration (Figure 4D). Following aspiration, a significant amount of thrombus material was collected and PA pressure immediately reduced to 25/8 mmHg. Subsequently, a bolus of 250,000 units of Streptokinase was given in each right and left PAs in the occlusion site over 10 min and then followed by maintenance dose of 100,000 units per hour for the next 10 h through pigtail catheter kept in main PA. Repeat CTPA 48 h after the procedure still showed some thrombus in right PA (Figure 1, Post procedure 48 h, Case 3) and also RV dilatation.

Hospital stay was uneventful and the patient was successfully discharged with dual antiplatelet therapy for 12 weeks. Follow up CTPA at three months showed complete resolution of thrombus in right PA and also T-wave resolution in precordial leads in ECG.
Ahmed K, et al. Catheter based approach for pulmonary thromboembolism

Discussion

PTE can present in many ways ranging from mild pleuritic pain to sudden fatal collapse. Thrombolytic therapy is the first-line of treatment in patients with high-risk PTE presenting with cardiogenic shock and/or persistent arterial hypotension, routine use of thrombolysis in non-high-risk patients is not recommended, but may be considered in selected patients with intermediate-risk PTE and after thorough consideration of conditions increasing the risk of bleeding. Thrombolytic therapy should not be used in patients with low-risk PTE. Percutaneous catheter embolectomy and clot fragmentation can be performed as an alternative to thrombolysis when there are absolute contraindications, as adjunctive therapy when thrombolysis has failed to improve haemodynamics, or as an alternative to surgery if immediate access to cardiopulmonary bypass is unavailable.[1] Moreover, this indication can extend to the patients without contraindication of thrombolysis and those of late presentation (> 96 h after the onset).[5] Catheter-based interventions should be made an option in the presence of local expertise and availability of devices and facilities.[6]

Two of our patients had acute and one patient subacute presentation. Compared to acute phase, older clots in subacute phase may be less amenable to systemic thrombolysis and expected to lyse less easily with increased likelihood of recurrence and thromboembolic pulmonary hypertension.

In all of our three cases, we did not use any specific thrombectomy devices like Rotarex, Aspirex, Greenfield, Amplatz etc., which had been previously utilised in the treatment of PTE.[7–9] The mechanical fragmentation of clots and its aspiration worth mentioning was performed with conventional and commonly used J-wire and GCs in our
in rheolytic embolectomy. With the help of MP and JR catheters we could easily access different parts and main branches of PAs and therefore more thrombus could be fragmented and aspirated. The approach utilized in our cases is simple, safe and cost effective in terms of immediate mortality benefits when performed by an experienced interventionist. Thrombectomy with intrapulmonary thrombolysis resulted in significant reduction of mean PA pressures from 91/37 mmHg to 45/13 mmHg immediately after the procedures. The proposed mechanisms of rapid reduction in PA pressure in our patients could be due to increased exposure of fibrin on clot surfaces caused by fragmentation accelerating the thrombolytic action because of intra-lesional streptokinase. Moreover, mechanical fragmentation of clot by J-wire and tip of pigtail catheter could have played an added advantage. Catheter fragmentation facilitates at least partial recanalization of a central embolic occlusion. According to pathophysiological considerations, fragmentation of central emboli and dislocation of the fragments to the periphery result in a relative gain of nonobstructed crosssectional area. Moreover, the increased total surface area of the fragments may accelerate the efficacy of an accompanying thrombolysis or of spontaneous intrinsic lytic activity. There were no periprocedural bleeding complications. The technique required, in all three cases, positioning of an infusion catheter (pigtail) within the embolus, with injection of a bolus of thrombolytic drug, followed by continuous infusion. As all the three patients had undergone catheter directed embolectomy, therefore they were advised to continue with dual antiplatelet therapy for three months after the discharge. Complete resolution of thrombus was achieved an all the patients in three months. Anticoagulation prevents further clot formation, but does not lyse existing thromboemboli or decrease thrombus size. Rare catheter thromboembolism complications include pericardial tamponade and pulmonary hemorrhage. Catheter techniques should only be used in the main arteries since fragmentation within the smaller branches is unlikely to be of benefit and may damage the more delicate structures, with risk of perforation or dissection of a pulmonary arterial branch that may cause massive pulmonary hemorrhage and immediate death. Device-related complications also include blood loss and mechanical hemolysis, transient catheter-induced arrhythmia or catheter-related infection. Other complications include bleeding from heparin anticoagulation, contrast-induced nephropathy, anaphylactic reaction to iodine contrast, and vascular access complications, such as hematoma, pseudoaneurysm, or atrioventricular AV fistula.

In conclusion, we do hereby report the efficacy of percutaneous thrombectomy for the patients with acute and sub-acute submassive PTE. This subset of patients can be successfully treated with simple catheter embolectomy followed by intra-PA injection of streptokinase. Catheter directed thrombectomy can be safely and effectively performed with the help of commonly used J wire and MP and JR GCs obviating the need of specific thrombectomy devices.

Acknowledgement
The authors sincerely thank Dr. Sumera Ahmed for her help in the preparation of this manuscript.

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: 2276–2315.
2. Brevetti GR, O’Brien B, Coomer CL, et al. Emergent surgery for massive pulmonary embolism on the basis of clinical diagnosis. Tex Heart Inst J 2003; 30: 149–151.
3. Funakoshi Y, Kato M, Kuratani T, et al. Successful treatment of massive pulmonary embolism in the 38th week of pregnancy. Ann Thorac Surg 2004; 77: 694–695.
4. Daniels LB, Parker JA, Patel SR, et al. Relation of duration of symptoms with response to thrombolytic therapy in pulmonary embolism. Am J Cardiol 1997; 80: 184–188.
5. Arzamendi D, Bilodeau L, Ibrahim R, et al. Role of rheolytic thrombectomy in massive pulmonary embolism with contraindication to systemic thrombolytic therapy. EuroIntervention 2010; 5: 716–721.
6. Jaff MR, McMurtry MS, Archer SL, et al. Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension: a scientific statement from the American Heart Association. Circulation 2011; 123: 1788–1830.
7. Liu S, Shi HB, Gu JP, et al. Massive pulmonary embolism: treatment with the rotarex thrombectomy system. Cardiovasc Intervent Radiol 2011; 34: 106–113.
8. Eid-Lidt G, Gaspar J, Sandoval J, et al. Combined clot fragmentation and aspiration in patients with acute pulmonary embolism. Chest 2008; 134: 54–60.
9. Greenfield LJ, Proctor MC, Williams DM, et al. Long-term experience with transvenous catheter pulmonary embolec- tomy. J Vasc Surg 1993; 18: 450–457.

http://www.jgc301.com; jgc@mail.sciencep.com | Journal of Geriatric Cardiology