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Original Article

Impact of catheter fragmentation followed by local intrapulmonary thrombolysis in acute high risk pulmonary embolism as primary therapy



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ABSTRACT

Background: Pulmonary embolism (PE) with more than 50% compromise of pulmonary circulation results significant right ventricular (RV) afterload leading to progressive RV failure, systemic hypotension and shock. Prompt restoration of thrombolysis, surgical embolectomy, or percutaneous mechanical thrombectomy (PMT) prevents progressive hemodynamic decline. We report our single center experience in high risk PE patients treated with standard pigtail catheter mechanical fragmentation followed by intrapulmonary thrombolysis as a primary therapy.

Methods: 50 consecutive patients with diagnosis of high risk PE defined as having shock index >1 with angiographic evidence of >50% pulmonary arterial occlusion are included in the present study. All patients underwent emergent cardiac catheterization. After ensuring flow across pulmonary artery with mechanical breakdown of embolus by rotating 5F pigtail catheter; bolus dose of urokinase (4400 IU/kg) followed by infusion for 24 h was given in the thrombus. Hemodynamic parameters were recorded and follow up pulmonary angiogram was done. Clinical and echo follow up was done for one year.

Results: Pigtail rotational mechanical thrombectomy restored antegrade flow in all patients. The mean pulmonary artery pressure, Miller score, Shock index decreased significantly from 41 ± 8 mmHg, 20 ± 5 , 1.32 ± 0.3 to 24.52 ± 6.89 , 5.35 ± 2.16 , 0.79 ± 0.21 respectively ($p < 0.0001$). In-hospital major complications were seen in 4 patients. There was a statistically significant reduction of PA pressures from 62 ± 11 mmHg to 23 ± 6 mmHg on follow up.

Conclusions: Rapid reperfusion of pulmonary arteries with mechanical fragmentation by pigtail catheter followed by intrapulmonary thrombolysis results in excellent immediate and intermediate term outcomes in patients presenting with high risk pulmonary embolism.

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Acute pulmonary embolism (PE) is common, but has clinically highly variable presentation ranging from asymptomatic to massive PE. Massive PEs are those that cause more than 50% pulmonary arterial compromise, leading to right ventricular failure, circulatory collapse, hypotension, and/or shock. Mortality rate without treatment from massive PE is approximately 30 percent; usually within the first few hours of the initial event.^{1–3}

The goal of therapy in patients presenting with massive PE is rapid recanalization of pulmonary arteries with thrombolysis or embolectomy; to decrease right ventricular afterload and reverse right ventricular failure and shock, decrease the risk of recurrence, and prevent chronic thromboembolic pulmonary hypertension.^{4,5} Intravenous Thrombolytic therapy is the first-line treatment in patients with high risk PE presenting with cardiogenic shock and/or persistent arterial hypotension.⁶ A substantial proportion of patients, however, may not be eligible for intravenous thrombolysis because of major contraindications.⁷ Surgical embolectomy is an alternative therapeutic option in patients in whom thrombolysis is absolutely contraindicated or has failed. However number of experienced tertiary care centers with around-the-clock availability of emergency surgical embolectomy are limited.^{8,9} Percutaneous catheter embolectomy or mechanical fragmentation of proximal pulmonary arterial clots followed by local thrombolytic therapy may be considered as a very attractive alternative to surgical embolectomy and systemic thrombolysis because of their capacity to establish pulmonary blood flow rapidly. Several reports have shown that mechanical fragmentation combined with local thrombolysis is a good therapeutic option for restoring pulmonary flow and decreasing PAP; with comparable short term outcomes to systemic thrombolysis.^{10,11} However information on long term outcomes are limited in literature. We report here our single center experience in high risk PE patients treated with standard pigtail catheter mechanical fragmentation followed by intrapulmonary thrombolysis on immediate and long term hemodynamic and clinical outcomes.

1. Patients and methods

1.1. Patient population

This was an open labeled, noncomparative, single center experience. Approval for the study was obtained from local Institutional Ethics Committee. Prospectively consecutive patients presenting with clinical diagnosis of pulmonary embolism and shock index¹² (heart rate in beats per minute divided by systolic blood pressure in mm of mercury, HR/SBP) >1 from July 2006–July 2009 were enrolled in the trial. The patients were admitted to ICU and written informed consent was obtained. Bed side transthoracic echocardiography was done to confirm the suspicion of pulmonary embolism, to estimate pulmonary arterial pressure and to exclude right atrial or ventricular thrombi. All patients underwent emergent right heart catheterization and pulmonary angiography. Patients who showed a rapid deterioration of their cardiopulmonary condition were put on oxygen supplementation with noninvasive pressure support or intubation. Positive

inotropic and vasoactive support was initiated according to the hemodynamic conditions. All patients underwent emergent right heart catheterization and pulmonary angiography.

Inclusion criteria for the study were: Patients with angiographically confirmed acute massive pulmonary embolism with shock index >1, pulmonary arterial occlusion with >50% involvement of the central (main and/or lobar) pulmonary arteries (Miller index >0.5),¹³ and pulmonary hypertension (mean pulmonary artery pressure >25 mmHg).

Exclusion criteria included patients with echocardiographically confirmed right sided thrombi, Acute gastrointestinal bleeding, Electrolyte imbalance, Anticoagulation with international normalized ratio >1.8 or severe coagulopathy, Anaphylactoid reaction to contrast media, Acute stroke, Acute renal failure or severe chronic nondialysis dependent kidney disease, Unexplained fever or untreated active infection, Severe anemia, Uncooperative patient.

A total of 50 consecutive patients (9 females, 41 males) with average age of 47 ± 12 years were included in this study.

2. Methodology

All pulmonary angiograms and therapeutic interventional procedures were performed in cardiac catheterization laboratory (Phillips Medical Systems; Netherlands). After giving local anesthesia; 5F sheath was introduced in the femoral vein for procedure. Initially 5F multipurpose catheter was advanced over 0.035-inch Teflon-coated guide wires under fluoroscopic guidance and was used to measure right heart and pulmonary artery pressures. Subsequently, 5F standard pigtail catheter was used to obtain initial pulmonary angiography with an injected volume of 30–40 mL using cine mode with a frame rate of 25/s. Angiographic quantification of degree of pulmonary artery involvement was assessed by the Miller score. Miller score is calculated as the sum of obstruction and perfusion scores, ranging from 0 (best) to 34 (worst). Calculation of the Miller obstruction score ranges from 0 to 16: 9 major segmental branches in the right PA (3 in the upper lobe, 2 in the middle lobe, 4 in the lower lobe) and 7 major branches in the left PA (2 in the upper lobe, 2 in the lingual, 3 in the lower lobe). The presence of filling defect in any of these branches is scored 1 point. The perfusion is scored by dividing each lung into 3 zones (upper, middle, and lower), and the flow into each zone is characterized as absent (3 points), severely reduced (2 points), mildly reduced (1 point), or normal (0 points.) A Miller score of 17 or more indicates a greater than 50% obstruction of pulmonary vascular bed and forms an angiographic definition of a massive PE. The Miller index is Miller score divided by 34 (range 0–1.0).

After confirming the inclusion criteria, mechanical catheter thrombectomy was initiated using a pigtail catheter. The catheter was quickly spun manually so as to fragment the central thrombus and establish initial flow into pulmonary artery (Fig. 1). After ensuring initial flow, the pigtail was left in place inside the same large proximal embolus for subsequent local thrombolytic therapy. Initial bolus dose of Urokinase (4400 IU/kg body weight) was given over 10 min followed by continuous infusion of 4400 IU/kg/hr for 24 h. All patients were monitored continuously for clinical and hemodynamic

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