Massive Pulmonary Embolism: Percutaneous Mechanical Thrombectomy during Cardiopulmonary Resuscitation

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Seven patients with massive pulmonary embolism (PE) causing cardiac arrest underwent percutaneous mechanical thrombectomy (PMT) with Hydrolyser and Oasis catheters during cardiopulmonary resuscitation (CPR). Three received adjunctive recombinant tissue plasminogen activator. Thrombectomy was successful in restoring pulmonary perfusion in six patients (85.7%). One patient died of cardiac arrest. Systolic pulmonary pressure decreased after thrombectomy from a median of 73 mm Hg (range, 63–90 mm Hg) to 42 mm Hg (range, 32–81 mm Hg; $P < .05$). There was one groin hematoma that required blood transfusion. In conclusion, massive PE causing cardiac arrest can be treated with PMT simultaneously with CPR maneuvers to rapidly revert circulatory collapse, with restoration of pulmonary circulation. Larger series are needed to validate this method.

J Vasc Interv Radiol 2005; 16:119–123

Abbreviations: CPR = cardiopulmonary resuscitation, PE = pulmonary embolism, PMT = percutaneous mechanical thrombectomy, rt-PA = recombinant tissue plasminogen activator

PULMONARY embolism (PE) causes or contributes to 50,000–200,000 deaths per year in the United States (1,2). The mortality rate for massive PE producing shock is approximately 30% (3). It is one of the most frequent noncardiac causes of cardiac arrest (4), with mortality rates ranging from 65% to 95% in reported series (2,4). In fatal cases, the majority of deaths occur within 1 hour of presentation (2). This clinical situation is similar to the "golden hour" of myocardial infarction, stroke, and trauma, during which a timely approach to diagnosis and treatment of massive PE with cardiac arrest can change the outcome (2). Percutaneous mechanical thrombectomy (PMT) and fragmentation are therapeutic options for patients with massive PE that have been used especially when there are contraindications to thrombolytic drugs. These mechanical therapies can help to immediately reverse cardiac arrest (5–9). The purpose of this article is to present our clinical experience in the treatment of massive PE causing cardiac arrest. We used PMT simultaneously with cardiopulmonary resuscitation (CPR).

MATERIALS AND METHODS

Patient Group

Seven patients (four women, three men) with a mean age of 56 years (range, 30–79 years) with sudden cardiac arrest secondary to massive PE were treated with PMT during CPR. All patients had pulseless electrical activity on initial assessment. Risk factors for thromboembolic disease in these patients were deep venous thrombosis in four patients, recent major surgery in two patients (cardiac transplantation and hemicolectomy), prolonged immobilization in two, major trauma (femoral neck fracture) in one, and hypercoagulability in one. Four patients had contraindications to thrombolytic agents (two with recent major surgeries, one with major trauma, and one because of recent postpartum status). The other three patients received recombinant tissue plasminogen activator (rt-PA) after thrombectomy. The diagnosis of massive PE was established in three patients with computed tomographic (CT) angiography before the occurrence of cardiac arrest. Two cases studied with echocardiography before cardiac arrest demonstrated right ventricle failure. In the other four cases, clinical diagnosis was confirmed angiographically. All patients were transferred to the angiography suite during CPR to rapidly initiate therapy. CPR maneuvers were performed synchronously with PMT. The intensive care unit team and interventionalists worked coordinated on both sides of the table and there were instances in which PMT and cardiac compressions were performed simultaneously with fluoroscopy.

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None of the authors have identified a conflict of interest.

DOI: 10.1097/01.RVI.0000146173.85401.BA

Devices

At random, the Hydrolyser thrombectomy catheter (6,8) (Cordis, Miami, FL) was used in four cases, and the Oasis thrombectomy catheter (8) (Boston Scientific, Galway, Ireland) was used in three cases. These devices are hydraulic recirculation PMT devices that use the Venturi effect. Heparinized saline solution is injected with an angiographic mechanical injector at 2.5–3.0 mL/sec at 750 psi through a narrow injection lumen that ends in a hairpin loop at the distal end of the catheters, which produce a Venturi effect that fragments adjacent thrombus and directs the debris to a evacuation lumen.

Technique

The access to pulmonary arteries was obtained by unilateral ($n = 4$) or bilateral ($n = 3$) common femoral vein puncture (Seldinger technique) with use of 6-F pigtail catheter(s). Nonionic contrast medium (Radiomiron 370; Schering, Berlin, Germany) was administered. Pulmonary arterial pressures were measured before and after PMT. The procedure was performed through an 80-cm, 8-F or 10-F sheath (Super Arrow-Flex Percutaneous Sheath Introducer Set; Arrow International, Reading, PA) selectively positioned to reach the thrombi. The thrombectomy catheters were positioned in the thrombus and moved in a forward/backward movement in the main and lobar arteries. Eight to 12 passes in a back-and-forth motion were performed with the Hydrolyser and Oasis catheters. Each pass takes 30–40 seconds.

In the three cases with no contraindication to thrombolytic therapy, rt-PA (Actilyse; Boehringer Ingelheim, Ingelheim, Germany) was infused directly into the thrombus after PMT (10-mg bolus and infusion of 10 mg/h during 4 hours; total, 50 mg).

The Urokinase Pulmonary Embolism Trial angiographic severity index was calculated (10). This index has a maximum value of 18, which corresponds to complete occlusion of both pulmonary arteries. An occlusion of two or more lobar arteries (index >5) or equivalent smaller branches is angiographically considered a massive PE.

Semiquantitative estimation of removed thrombi was performed with use of a computer program developed for this purpose. The program calculates the thrombi-occluded area in the pulmonary arterial tree as a percentage with use of a standard diagram that was "shadowed" in thrombi-occluded areas (6).

Institutional review board approval in our institution is not required for retrospective reports such as this.

Study Endpoints

The study endpoint was mortality equal or superior to that expected for cardiac arrest secondary to massive PE without endovascular recanalization.

Statistical Analysis

Data were analyzed with the use of the Wilcoxon test for paired values. A P value $<.05$ was considered statistically significant. Results are presented as median values with ranges in parentheses.

RESULTS

PMT was successful in restoring of pulmonary perfusion in six patients (85.7%). Duration of CPR was 10–35 minutes, with a median of 25 minutes. The PMT devices' activation time was 6 minutes (4–8 minutes). Injected saline solution volume during PMT was 300–400 mL. Recovered volume in the bag was 200–300 mL. Systolic pulmonary artery pressure decreased from a median of 73.0 mm Hg (90–63 mm Hg) to 42 mm Hg (32–81 mm Hg) immediately after thrombectomy ($P < .05$), and in three patients, pressure decreased to 34 mm Hg (33–36 mm Hg) after rt-PA infusion (P = NS). The Urokinase Pulmonary Embolism Trial Angiographic Severity Indexes (10) before and after PMT were 15 (14–16) and 7 (5–10), respectively ($P = .022$). A median of 60% of thrombus (40%–70%) was removed, calculated by semiquantitative computer analysis. No difference was observed in quantity of thrombus removed between the Hydrolyser and Oasis thrombectomy devices.

Complications

There were no major complications directly related to PMT. Two patients treated with rt-PA developed a hematoma at the puncture site; one required transfusion of 2 U of blood. One patient did not recover from cardiac arrest caused by massive PE and died during the CPR and PMT procedure. Another patient had a brain infarction secondary to cardiac arrest but survived. Six patients were discharged within 30 days. A representative case is illustrated in **Figure 1**.

DISCUSSION

Although its frequency is not well established, massive PE is one of the most frequent noncardiac causes of cardiac arrest (4,11). In a study by Wood (3) that included PE with pulmonary hypertension or right heart failure caused by massive PE, the incidence of cardiac arrest was 18%. In the series by Miller et al (12), cardiac arrest occurred in 29% of patients. Kurkciyan et al (4) established that massive PE causes 14.6% of cases of cardiac arrest of extracardiac origin. In the majority of cases of massive PE with cardiac arrest, there is pulseless electric activity/electromechanical dissociation and asystole (4,11). Comes et al (11) found that massive PE caused 36% of cases of unexplained sudden cardiac arrest among patients who had pulseless electric activity as the initial rhythm (11). In our series, all seven patients presented with pulseless electric activity and electromechanical dissociation.

Mortality rates in massive PE when causing cardiac arrest are extremely high, as high as 70% in some series (3,11). Cardiac arrest will occur within 1–2 hours after the onset of clinical presentation in two thirds of fatal massive PE cases (1). A prompt clinical diagnosis of massive PE and treatment is necessary for optimal treatment in this "golden hour" (2). CPR maneuvers in these cases should be accompanied by causative treatment.

When a patient's condition is hemodynamically compromised, diagnostic methods as CT angiography, magnetic resonance angiography, and ventilation/perfusion lung scan are time-consuming. Echocardiography in patients in hemodynamically unstable