A Case of Percutaneous Catheter Thrombectomy in a Patient With Massive Pulmonary Thromboembolism

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ABSTRACT

Acute massive pulmonary thromboembolism (PTE) is a life threatening medical condition. Therefore, prompt diagnosis and rapid recovery of the pulmonary flow is essential for decreasing the associated morbidity and mortality. We report here on a case of a 68-year old woman for whom we attempted percutaneous catheter thrombectomy in the treatment of an acute massive PTE. (Korean Circ J 2008;38:227-229)

KEY WORDS: Thrombectomy; Pulmonary embolism.

Introduction

Acute pulmonary thromboembolism (PTE) is a fatal condition with a mortality rate of approximately 30 percent if it is left untreated. Unfortunately, the clinical presentation of a PTE is variable and nonspecific, making an accurate diagnosis difficult. The number of patients treated for PTE has markedly increased recently because of the improved recognition of this condition and the advances in the diagnostic tools. The associated mortality can be reduced by prompt diagnosis and therapy.

Acute massive PTE requires emergent aggressive treatment. Systemic thrombolytic therapy and surgical thrombectomy are the traditional therapeutic options in this situation. Catheter-directed thrombolysis, percutaneous embolectomy and more recently, percutaneous thrombus fragmentation techniques are now available to treat the most severe cases of a massive PTE.

We report here on the case of a 68-year old woman with acute massive PTE that mimicked an acute myocardial infarction, and this was treated with percutaneous catheter thrombectomy.

Case

A 68-year-old woman was admitted to our hospital presenting with back and buttoc k pain due to a traffic accident. She was diagnosed with a sacral bone fracture. Her past medical history was notable for long-standing hypertension and diabetes mellitus. On the fourteenth day of hospitalization, she had a sudden onset of dyspnea and cold sweating followed by an impaired mental status. The physical examination revealed cyanosis, hypoxemia and severe hypotension (60/40 mmHg) despite treatment with intravenous inotropic agents, including dopamine and norepinephrine. The laboratory findings showed a D-dimer level of 4.31 μg/mL, and a CK-MB/Troponin-I level of 2.3/0.66 ng/mL. The chest x-ray showed no abnormality. An electrocardiogram showed sinus tachycardia, ST segment elevation in leads V1-3 and ST segment depression in leads I and aVL (Fig. 1). Thorough echocardiography demonstrated decreased left ventricular systolic function and hypokinesia of the anterior wall, but there was no sign of definitive right ventricular overload like a D-shaped interventricular septum or right ventricular dilatation. Therefore, we diagnosed the patient with an acute myocardial infarction and cardiogenic shock. A coronary angiogram was performed for percutaneous coronary intervention. However, the coronary angiogram revealed no definite flow limitations. The patient developed pulseless electrical activity, and she subsequently required cardiopulmonary resuscitation (CPR). In spite of oral intubation and CPR, the hemodynamic condition remained unstable.

We performed pulmonary artery angiography for a suspected pulmonary thromboembolism. The angiography showed a massive pulmonary thromboembolism at the trunk of the left pulmonary artery (Fig. 2). We attempted to aspirate the thrombi using an 8 French catheter.
guiding catheter and to fragment the clot with a guide wire. We aspirated a large amount of the thrombi and this subsequently improved the blood flow of the pulmonary artery (Fig. 3). However, the cardiogenic shock persisted, although intravenous unfractionated heparin was administered as a bolus (25,000 unit), followed by continuous infusion (1,000 unit/hour), but thrombolytic treatment was not fully achieved. Sadly, the patient died 5 hours later in the intensive care unit.

**Discussion**

Acute PTE is a life-threatening condition with a poor prognosis. However, it is difficult to diagnose a PTE due to its variable, nonspecific clinical presentation. The classic clinical presentation with dyspnea and chest pain might not always be seen. The physical examination and electrocardiogram can also reveal variable signs. An accurate diagnosis is required for the appropriate
treatment and to minimize such patient injury as right ventricular dysfunction, pulmonary hypertension and respiratory insufficiency.34)

In this case, the correct diagnosis was delayed and the patient was misdiagnosed with an acute myocardial infarction based on the electrocardiogram that showed ST segment elevation in the precordial leads. In addition, the transthoracic echocardiography demonstrated regional wall motion abnormality and no signs of definitive pressure overloading in the right ventricle. Therefore, we thought that performing early transthoracic echocardiography would not show signs of right ventricular failure.

An unstable massive PTE is a clinical emergency that requires immediate and effective life-supporting therapeutic measures. In addition to heparin therapy, the accepted treatments for massive PTE include systemic and local fibrinolysis and surgical thrombectomy. In a number of cases, thrombolytic therapy may fail to achieve this goal in time. Percutaneous catheter treatment may represent an additional option for high-risk patients.35)

Minimally invasive procedures such as catheter-directed thrombolysis, percutaneous embolectomy and thrombus fragmentation have recently been introduced. The indications for these procedures are circulatory collapse with the need for cardiopulmonary resuscitation, right ventricular afterload stress and/or pulmonary hypertension and the angiographic findings (Miller score >20/34).6)

Percutaneous catheter thrombectomy has evolved from a very simple concept that has been previously used in many other fields. Sakai et al.35) reported that aspiration thrombectomy removed a massive thrombus from the native coronary artery of a patient with myocardial infarction, and this achieved successful reperfusion. Sato et al.35) reported that a massive pulmonary thromboembolism in a pregnant woman, and she was treated with transcatheter thrombectomy and the patient’s hemodynamics recovered dramatically.

However, percutaneous pulmonary thrombectomy is not a common practice even though the hemodynamics of patients are rapidly improved when it is used.2) The associated complications include perforation or dissection of the cardiovascular structures, pericardial tamponade, pulmonary hemorrhage and distal thrombus embolization. To minimize the risk of these complications, thrombectomy should be performed only in the main and lobar pulmonary arteries, and not in the segmental pulmonary arteries. The procedure should be terminated as soon as hemodynamic improvement is achieved, regardless of the angiographic result.35) After the procedure, the use of a combination of thrombolytic agents allows a larger surface area of the fragmented emboli to be exposed to the thrombolytic agents.35)

In conclusion, we were able to aspirate a large volume of thrombi by performing percutaneous catheter thrombectomy and this subsequently improved the blood flow through the pulmonary artery. Regrettably, the patient in this case died due to the development of cardiogenic shock.

REFERENCES