

# Pulmonary Embolectomy for Acute Massive Pulmonary Embolism

— 1 case —

— Abstract —

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Pulmonary thromboembolism originated mostly from the venous thrombus, especially deep vein thrombosis in the lower extremities, which migrated upward and lodged the pulmonary vasculatures. Massive pulmonary thromboembolism usually leads to in-hospital mortality if the patient was not treated properly. Under the cardiopulmonary bypass, a 49-year old man was treated successfully by emergent pulmonary embolectomy of pulmonary thromboembolism, which originated from the deep vein thrombosis in the right leg. Ligation or filtering device insertion of the inferior vena cava was not performed. The patient's postoperative course was uneventful and discharged on postoperative 15th day. He continued to receive oral anticoagulation with aspirin and persantin, which had been started on the third postoperative day. And he was well till recent days through the outpatient followup. The clinical courses of this patient are described, and massive pulmonary embolism and its management are discussed.

## Introduction

Despite recent advances in diagnostic tools and surgical skills, pulmonary thromboembolism remains a major clinical challenge. Pulmonary emboli are present in roughly 142,000 patients dying in the United States each year. Furthermore, over one half million people suffer nonfatal pulmonary emboli each year<sup>21)</sup>.

But in Korea, there was a few instances which were reported in literatures<sup>38~42)</sup>.

While heparin therapy represents the mainstay of treatment, a small group of patients do not respond to this therapy and die from massive obstruction of the pulmonary arterial tree<sup>21)</sup>. Because of the simplistic nature of the obstruction, Tren-

delenberg first suggested removal of clots from the pulmonary arteries in 1908, but successful embolectomy was not performed until 1924<sup>21,38)</sup>. In 1961, pulmonary embolectomy utilizing cardiopulmonary bypass, allowing for adequate support of the patient during the procedure, was first reported<sup>4)</sup>. The cardiopulmonary bypass has allowed a more physiologic resuscitation of the patient in profound shock with hypoxemia and acidosis<sup>21,34)</sup>. Since that time, several reports have emerged describing successful use of cardiopulmonary bypass technique<sup>6,10,11,27,31,33,34)</sup> but these cases were confined to chronic pulmonary embolism. As for the acute massive pulmonary embolism, there was no report for surgical experience. This report reviews our recent experience and discusses the role and technique of emergent pulmonary embolectomy in acute massive pulmonary embolism.

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## Patient and Methods

A 49-year old man was admitted to Kyung Hee University Medical Center via emergency room with dyspnea and chest pain of a 5 day duration. Four months before admission, he had undergone incision and drainage for superficial thrombophlebitis of the right leg. At that time, he was diagnosed to have deep vein thrombosis, which was treated by oral anticoagulation. He was well until 5 days before admission, when he began to suffer substernal chest pain and intermittent dyspnea. He visited on local clinics, but his symptoms was not improved and therefore he was referred to our hospital for the further evaluation and management.

Physical examination revealed an anxious and relatively obese in general inspection. Blood pressure was recorded at 60/40mmHg, respiratory rate at 30 per minute, and body temperature at 36C. The examination of the cardiovascular system was normal except tachycardia (144 per minute). The trill was not palpated on the precordium. There were no murmurs, clicks, third or fourth heart sounds, or pericardial friction rub. There was no evidence of jugular venous distension, and peripheral arterial pulses were thread and rapid. Examination of the lungs revealed no abnormality. There was no peripheral edema, and the remainder of the physical examination revealed no major abnormalities.

The white cell count was 13,900, the hematocrit 47.8% and hemoglobin 16.5 gm per 100ml. The arterial blood gas analysis revealed that the partial pressure of oxygen was 59 mmHg, the partial pressure of carbon dioxide 28mmHg, the pH 7.42, the base deficit 4 and oxygen saturation 91% under the room air breathing. An EKG revealed T wave inversion in I, III, aVF, IV - V3 (figure 1). Chest roentgenogram was grossly normal finding except pulmonary oligemia. A pulmonary angiography on

the day of admission revealed a large filling defect in the main pulmonary artery, especially in the right main and left upper pulmonary artery (figure 2). Pulmonary arterial pressure measured 60/20 mmHg.

The operative technique was as follows: The access was performed by median sternotomy, and the partial cardiopulmonary bypass was established. Central thromboembolic clots were extracted through a longitudinal pulmonary arteriotomy on partial cardiopulmonary bypass (26°C at body temperature). Both pleural spaces were opened, and vigorous manual pulmonary compression forced peripheral clots into the main pulmonary artery for extraction under direct vision. The extracted clots revealed relatively fresh, of which amount was 35gm, color was reddish (figure 3). With the use of gall stone forceps and usual sucker, these were removed as possible. After obtaining good back flow from both distal pulmonary artery the main pulmonary arteriotomy was closed. Ligation or filtering device insertion of the inferior vena cava was not performed because these devices were not prepared and surgeon's will for the continuous anticoagulation was intended. This patient improved symptomatically and was weaned from vasopressors and mechanical ventilation. He was discharged on 15th post-operative day. He continued to receive oral anticoagulation, which had been started on the third postoperative day. And he was well till recent days through the outpatient followup.

## Discussion

In 1819, Laennec<sup>4,6,21)</sup> described pulmonary apoplexy, which has been the first description in literatures. In 1858, Virchow described two types of the thrombus in pulmonary artery: first, the embolus that arose as a thrombus in a systemic vein and, after being dislodged from its site of origin, was swept into the venous return and

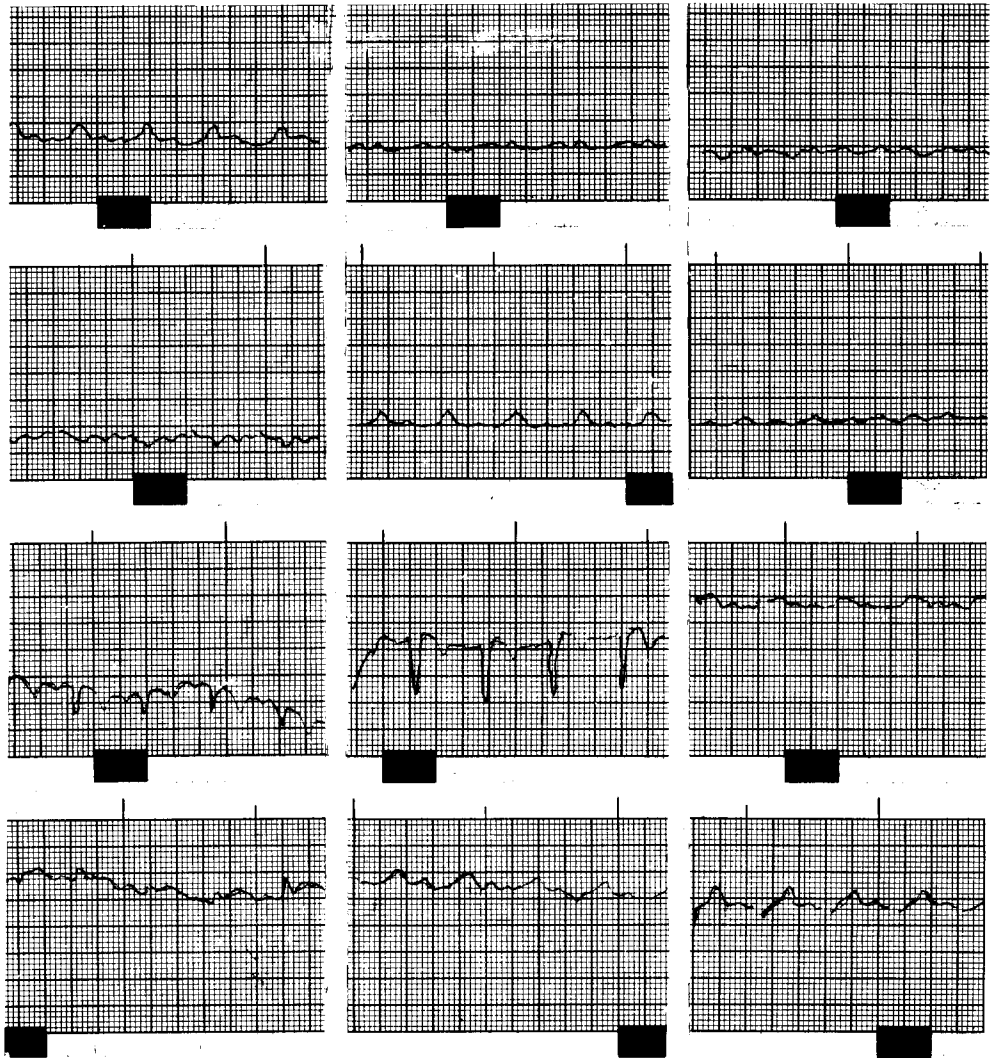


Fig. 1.



Fig. 2.



Fig. 3.

through the heart in the pulmonary arteries. Secondly, the thrombus that occurred in situ in the pulmonary artery distal to the occluding embolus as a result of stagnation in blood flow<sup>14</sup>. Since its inception in 1908 by Trendelenberg<sup>10,21</sup>, pulmonary embolectomy has been surrounded by controversy. Trendelenberg's attempts to perform pulmonary embolectomy with long term survival were unsuccessful. However, Sharp<sup>5</sup> achieved pulmonary embolectomy using cardiopulmonary bypass in 1961, which resulted in a long term survival.

Most statistics<sup>9,14,16,21,23</sup> indicated definite increase in the incidence of pulmonary thrombolism, of which reasons were suggested that the increase in older members of the population, large numbers and greater magnitude of operative procedures, increased recognition and use of hormonal agents for birth control. Despite of other reports in western countries, there was a few instances which were reported in literatures in our country<sup>27-31</sup>. Furthermore, all of these are chronic pulmonary embolism. There is no report for emergent embolectomy for the management of acute massive pulmonary embolism. And, this low prevalence for the surgically treatable pulmonary embolism might be due to physician's ignorance in minority.

Occlusion of pulmonary artery affects the airways, the pulmonary vasculatures, the right and left heart, and the bronchial circulation<sup>20</sup>. The pulmonary artery has a characteristic feature that is its low vascular resistance, a fact that enables flow in the pulmonary vascular bed to be increased manifold with minimal elevation of pulmonary arterial pressure<sup>18</sup>. Therefore, the considerable reduction in diameter of the main pulmonary artery or the primary branches is required to reduce pulmonary blood significantly or to produce pulmonary hypertension proximal to the obstruction<sup>33</sup>. Otherwise, massive pulmonary embolism increases the resistance to the blood flow through the lungs, substantially increases pulmonary ar-

terial pressure and increases pulmonary arterial pressure and increases right ventricular work. In severe cases, right ventricular failure occurs, left ventricular output is decreased and circulatory collapse occurs<sup>18,20</sup>.

In other point of humoral factor, the embolus stimulates the release of vasoactive amines—serotonin from platelets which introduces the pulmonary vasoconstriction and right ventricular failure<sup>18</sup>.

Confusion exists as to the adjectives used to describe pulmonary embolism. Although "massive" pulmonary embolism in this report implies patient who is collapsed and required emergent cardiorespiratory support, some reports use the term "massive" to describe an anatomic obstruction of the pulmonary artery of 50% or greater on angiography, with or without physiologic alteration<sup>7,21,33</sup>. The authors would recommend that the term "massive" be used only in patients who have either cardiac arrest or serious arterial hypotension and hypoxemia. The term "major" pulmonary embolism would then describe a 50% or greater pulmonary arterial anatomic obstruction<sup>7,21</sup>. This case revealed significant occlusion of the main pulmonary artery (nearly 50%) on angiogram, which was suggested the cause of circulatory collapse. But there was no evidence of reflex humoral mechanism through a preoperative evaluation.

Pulmonary embolism is notorious for the similarity of its clinical manifestations to those of other cardiorespiratory disorders, and this make difficult the establishment of an accurate clinical diagnosis<sup>2,3,7,8,14,33</sup>. There was a classification of pulmonary embolism<sup>25</sup>. This case was appointed into class III—IV by that classification.

There were many methods for the evaluation of pulmonary embolism, as follows: simple chest films, blood chemistry, pulmonary function test, arterial blood gases analysis, ventilation and perfusion scanning, and pulmonary angiography<sup>7,9,14,25</sup>. Of which methods, it was said that the pulmonary angiography afforded the most absolute diagnosis

7,9,20). On admission of this patient, he shock state (Bp 60 /40mmHg, thin and thread pulse), had a arterial hypoxemia, and had a history of deep vein thrombosis. And, therefore, the pulmonary angiography was taken emergently with the great suspicion of pulmonary embolism. With the confirmation of acute massive pulmonary embolus, medical treatments (heparinization or other anticoagulation and thrombolysis) were suggested not further beneficial to this patient – in reestablishing and maintaining an adequate circulation (BP >90 mmHg in systolic, arterial PO<sub>2</sub> >60 mmHg), then the decision for the pulmonary embolectomy was made<sup>9,20,33</sup>.

The mortality from pulmonary embolectomy differs widely in reported series (between 40% and 100%), primarily because of small numbers, different experiences, and lack of criteria for operation<sup>4,11,21,30,33,37</sup>. In especially acute massive pulmonary embolism, Kenneth et al<sup>21</sup>. reported 57% in mortality. Among patients with massive pulmonary embolism who were already moribund, Clarke<sup>5</sup> reported a mortality rate of 94% when pulmonary embolectomy under inflow occlusion was used, as compared with a 31% mortality rate for pulmonary embolectomy when the patient had not had a cardiac arrest. Until these deficits are corrected, no valid comparison with other modes of therapy can be made<sup>9,16,17,20</sup>.

Otherwise, the pulmonary embolectomy is a dramatic and heroic procedure in fewer patients with pulmonary embolism. If the patient who do not respond to vigorous medical therapy and who, without mechanical removal of emboli, would probably die, emergent pulmonary embolectomy under the cardiopulmonary bypass should be recommended<sup>6,10,11,14,27,31,33,34</sup>. As for the vena caval filtering devices, these were indicated in immediately following pulmonary embolectomy in order to prevent further thrombo-embolism<sup>12,13,15,19,23,24,26,28,29,36</sup>. But, in this case, we have not prepared these devices owing to the emergent state and surgeon's will for

the long term anticoagulation. The question of duration of orally administered anticoagulants has long been debated<sup>2,26,32,35,40,41</sup>. It has been assumed that anticoagulation is necessary as long as clots are present and are undergoing change in the pulmonary circulation and deepveins of the legs. In the absence of the contrast phlebography and impedance phlebography, it is recommended that patients should be treated with orally administered anticoagulant for at least three to six months<sup>22,29</sup>. With the willingness and ability of this patient to submit to frequent checks of prothrombin time, this patient has been treated with warfarin for 1 year. And then, he was discontinued this therapy and he is good in health for 2 years.

This procedure has proven a viable clinical tool in those patients in whom the pulmonary arterial obstruction causes severe cardiorespiratory disturbance. Rapid resuscitation and diagnosis, and an expeditious establishment of cardiopulmonary bypass, are associated with marked clinical improvement and the patient may survive. But the indication for embolectomy for massive pulmonary embolism could be better defined with the help of a well planned prospective trials.

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