Successful Surgical Pulmonary Embolectomy in Patient Who Has Massive Pulmonary Embolism with Multi-Thrombogenic Risk Factor – A Case Report

Çoklu Trombojenik Risk Faktörüne Sahip Masif Pulmoner Emboli Vakasının Başarılı Bir Şekilde Cerrahi Pulmoner Emboletkomi ile Tedavisi) – Olgu Sunumu

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Özet


Anahtar Kelimeler: Masif Pulmoner Embolizm, Çoklu Trombojenik Risk Faktörü, Cerrahi Pulmoner Emboletkomi
Abstract

We report a 36-year old woman who suffered from an massive pulmonary embolism with multi-thrombogenic risk factor. Patient was treated successfully with pulmonary embolectomy. Our report reinforces the value of early diagnosis in the presence of a high clinical suspicion of pulmonary embolism. Furthermore, surgical pulmonary embolectomy remains one of the most effective treatment methods.

Key Words: Massive Pulmonary Embolism, Multi-Thrombogenic Risk Factor, Surgical Pulmonary Embolectomy

Introduction

Acute pulmonary embolism (PE) is one of the major challenging diseases in the emergency setting. On average, 90% of all mortalities occur within 2 h of the onset of the symptoms (1). Therefore, the rapid treatment of massive PE is a high priority. The optimization of emergency structures has been demonstrated to significantly reduce the mortality rate from unstable PE (2). The reliable exclusion of PE in hemodynamically stable patients remains an additional problem, since in a number of these patients, the symptoms of PE are barely evident or manifest in an atypical manner. Previous studies have shown that PE has been frequently overlooked as a result, and that the mortality rate in such cases is significantly increased (3,4). The one of the most important diagnostic methods in suspected cases of PE is computed tomography (CT) scans of the pulmonary artery(5,6).

Case Report

A 36-year-old woman was admitted our hospital because of dyspnea. She has been smoking 20 cigarettes per day for 20 years and using oral contraceptive for 5 years. Physical
examination on presentation to the emergency department showed blood pressure of 90/50 mmHg and pulse rate of 110 beats/min. The patient was tachypneic throughout her hospitalization, with a respiratory rate ranging from 20 to 24 breaths/min. The crepitant rales were heard in the right basal pulmonary area on the auscultation. Cardiac findings included prominent pulmonic component of the second heart sound. The electrocardiogram showed sinus tachycardia and incomplete right bundle branch block. Laboratory tests showed mildly anemia (11.2 g/dL), increased white blood cells (12.4 K/μL), an elevated d-Dimer (941 ng/ml), slightly increased C-reactive protein (14.04 mg/dl), PaO2 of 35 mmHg, PaCO2 of 42 mmHg, SO2 62.3% and pH 7.465. Chest radiography revealed an infiltration view on the right basal pulmonary area. She immediately underwent transthoracic echocardiography which revealed a normal sized left ventricle, normal systolic function, and severe dilatation on the right ventricle. Contrast enhanced CT angiography of the chest demonstrated a large, contiguous filling defect reaching from main pulmonary artery to right and left pulmonary artery(Figure 1,2). She was referred for an emergency operation for pulmonary embolectomy since patient hemodynamic condition was unstable.

On the operation, after performing a median sternotomy, cardiopulmonary bypass was established by cannulation of the ascending aorta and by two caval cannulations. We detected dilatation of right ventricle. In the setting of partial cardiopulmonary bypass and beating heart, embolectomy was performed through a right and a left pulmonary incision(Figure 3). The operation was completed in the usual manner. The patient was weaned off cardiopulmonary bypass and maintained stable hemodynamics without inotrope support.

On postoperative day 2, she was discharged from the intensive care unit. All parameters improved markedly. Postoperative transthoracic echocardiography showed marked improvement on right ventricle functions. Lower-extremity Doppler ultrasound was negative for deep venous thrombosis. Results of the hypercoagulability workup including anti-nuclear
antigen, high levels of factor VIII, protein C and protein S activity, protein C and protein S antigen, antithrombin III activity, antithrombin antigen, plasminogen activity, phospholipid IgG/IgM and hyperhomocysteinaemia were negative. Our patient had heterozygous factor V Leiden mutation and multiple risks for thromboembolism (smoking and use of oral contraceptive). We planned the prothrombin time/international normalized ratio (PT/INR) to range from 2 to 3 to control warfarin postoperatively. Patient was discharged without morbidity three weeks later.

**Discussion**

Factor V Leiden deficiency is the most common hereditary hypercoagulable disease in the United States and involves 5% of the Caucasian population(7). Factor V Leiden leads to activated protein C resistance, and it has been demonstrated as a risk factor for venous thrombosis development(8). Approximately one out of 1000 patients will develop deep venous thrombosis (DVT) or pulmonary thromboembolism each year. Heterozygous Factor V Leiden increases the risk of developing DVT by 5 to 7 fold, while homozygous Factor V Leiden increases the risk of developing clots by 25 to 50 fold(9). The association between oral contraceptives and PE is established, although only a limited number of studies address the issue (10,13). Of these, the study by Lauque et al comprised only 11 case reports and is not recent (20). Significantly, more data exist with regard to the association between DVT and contraceptive use. A meta-analysis by Manzoli et al included a considerable number of studies and confirmed a significantly increased risk of thrombosis with oral contraceptive use (13). Despite the wealth of data, the results concerning DVT cannot be converted directly to PE. Nevertheless, the association between oral contraceptives and PE remains undisputed. With regard to the association between thrombophilia and PE, the data of the Lauque et al study is similarly weak. Data from a large retrospective study by Wu et al indicated a significantly increased risk of PE associated with different thrombophilia subgroups; the risk
increased further upon concomitant intake of contraceptives (14). Thus, the risk factors of contraceptive use and thrombophilia, in addition to a history of DVT/PE, are of tremendous importance in the context of PE(15). Our patient predisposing factors were convenient of literature. Furthermore, there was an additional risk factor as smoking.

The first successful surgical pulmonary embolectomy was performed in 1924, several decades before the introduction of medical treatment for PE. Pulmonary embolectomy is technically a simple operation(16). Following induction of anaesthesia and median sternotomy, normothermic cardiopulmonary bypass should be instituted. Aortic cross-clamping and cardioplegic cardiac arrest should be avoided(17). With bilateral PA incisions, clots can be extracted from both pulmonary arteries down to the segmental level under direct vision. Prolonged periods of post-operative cardiopulmonary by-pass and weaning may be necessary for recovery of right ventricle function. With a rapid multidisciplinary approach and individualized indications for embolectomy before haemodynamic collapse, perioperative mortality rates of 6% or less have been reported(18). Preoperative thrombolysis increases the risk of bleeding, but it is not an absolute contraindication to surgical embolectomy(19).

This case report emphasizes the value of early diagnosis in the presence of a high clinical suspicion of pulmonary embolism. An extended workup, including transthoracic echocardiography and CT scan of the pulmonary arteries are mandatory in such a patient especially when there are clinical findings suggestive of pulmonary embolism. Furthermore, surgical pulmonary embolectomy is one of the most effective treatment methods beside of thrombolytic and percutaneous catheter-directed treatment, especially when patient hemodynamic condition is unstable.
References


Figure-1: Arrow shows contiguous filling defect reaching from main pulmonary artery to right pulmonary artery.
Figure-2: Arrow shows filling defect in the branch of left pulmonary artery.
Figure-3: Arrows show thrombogenic materials which were extracted from pulmonary arteries.