

## A Case of Massive Pulmonary Thromboembolism in a Young Man Attribute to Computer Gaming

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### 젊은 남자에서 장시간의 컴퓨터게임 후 발생한 대량 폐색전증 1예

김 현, 최강현, 이기만, 신윤미  
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폐색전증은 일반적으로 고령의 환자, 거동이 불가능한 경우, 만성 내과 질환, 외상이나 술 후 또는 혈액응고질환 등의 위험인자를 갖고 있는 환자에서 호발하는 질환으로 알려져 있다. 그러나, 최근 컴퓨터가 보편화되고 특히 젊은 연령층에서 컴퓨터 게임이 대중화 됨에 따라 이 또한 폐색전증의 위험인자가 될 수 있음을 이 증례를 통하여 보고하고자 한다. 36세 남자 환자가 가슴답답함과 호흡곤란증세로 급성심근경색 의증 하에 응급실로 전원되었다. 환자는 청색증과 저산소증을 보였으며, 흉부컴퓨터단층촬영상 양측 주 폐동맥에 혈전증이 관찰되었으며, 혈전용해술을 시행하여 성공적으로 치료하였다. 병력취취상 약 2주 전부터 하루에 12시간 이상 컴퓨터 게임을 하였으며, 이 외에 특별한 위험인자는 없었다. 현재 환자 상태는 양호하여 외래 추적관찰 중이며, 이 환자의 증례를 통하여 장시간의 컴퓨터 게임이 혈전용해술을 시행할 만큼 대량의 폐색전증을 발생할 수 있음을 알 수 있었고, 이에 대한 사회적인 인식 또한 필요할 것으로 사료된다.

**Key Words:** Pulmonary embolism, Deep vein thrombosis, Computer gaming

#### Introduction

Pulmonary thromboembolism usually occurs in older patients or in patients with risk factors, such as, immobilization, cancer, acute and chronic medical illness, trauma, major surgery, pregnancy, during the postpartum period, hematologic disorders, hormone replacement therapy, or a hereditary hypercoagulable state<sup>1-5</sup>. We recently experienced a young patient with a massive pulmonary thromboembolism, and were unable to identify a risk factor other than immobilization due to the play-

ing of computer games.

#### Case Report

A 36-year-old man felt chest discomfort and dyspnea since two weeks ago and then visited a local clinic. He had never before experienced exertional chest pain or discomfort during exercise. Approximately two hours prior to presentation, he felt severe chest discomfort and dyspnea. He was transferred from the local clinic to the emergency room at Chungbuk National University Hospital to rule out acute myocardial infarction. He had no significant medical or family history of a cardiovascular or thromboembolic disease. He was unemployed and a current smoker.

His body weight was 89 kg and his height was 174 cm. He was alert but cyanosis was evident on his face and peripheral extremities. His systolic blood pressure

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was 70 mmHg, pulse rate 100 beats per minute, respiratory rate 22 breaths per minute, and body temperature 36.5°C. Pulse oximetry showed an arterial oxygen saturation of 92% and breathing at O<sub>2</sub> 5 L/min via a facemask. Auscultation of his chest revealed clear breath sounds without wheezing or crackles. He had a regular heartbeat without murmur.

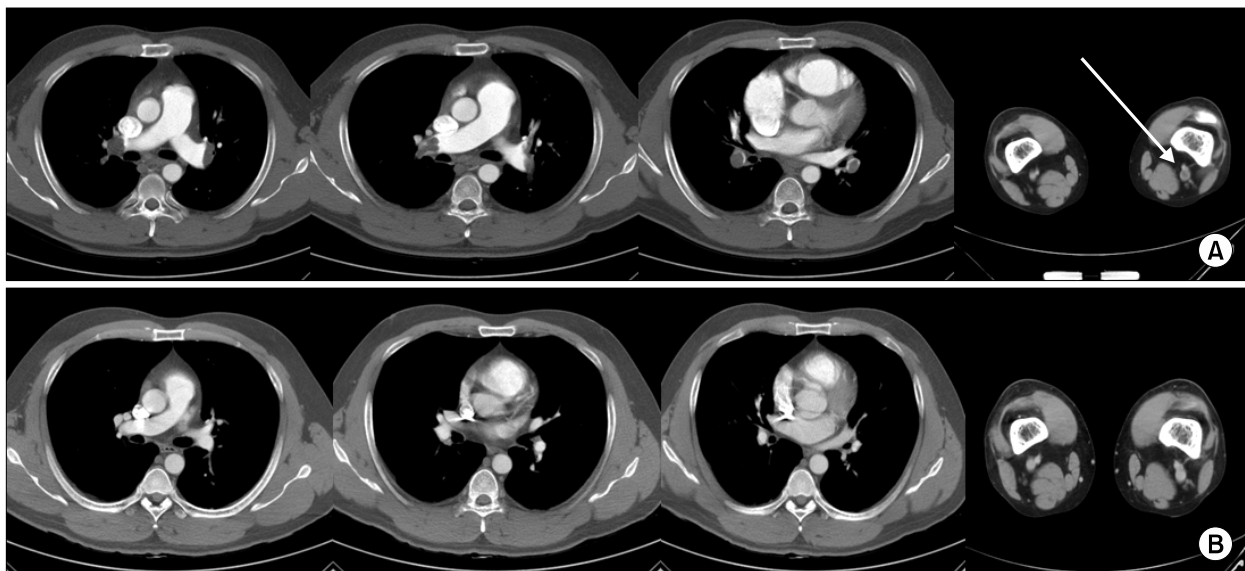


**Figure 1.** The chest AP radiograph (portable) showing no active lesions in the lungs.

Arterial blood gas analysis at O<sub>2</sub> 5 L/min revealed; pH 7.359, Pa<sub>CO<sub>2</sub></sub> 19.4 mmHg, Pa<sub>O<sub>2</sub></sub> 89.5 mmHg, and serum bicarbonate 11.1 mmol/L. His white-cell count was 18,200/ $\mu$ L, hemoglobin level 17.4 g/dL, hematocrit level 49.8%, and platelet count 135,000/ $\mu$ L. Serum urea nitrogen was 32 mg/dL, serum creatinine 1.4 mg/dL, serum total protein 6.1 g/dL, serum albumin 3.9 g/dL, aspartate aminotransferase 2,289 U/L, alanine aminotransferase 1,661 U/L, and C-reactive protein 2.66 mg/dL (normal < 0.3 mg/dL). His CPK level was 126 IU/L (normal < 190 IU/L), CK-MB 5.69 ng/mL (normal < 4.94 ng/mL), troponin T 0.077  $\mu$ g/mL (normal < 0.1  $\mu$ g/mL), and pro-BNP 10,775 pg/mL (normal < 115 pg/mL).

12-lead electrocardiographic findings were T wave inversion in leads II, III, aVF, and V<sub>1</sub> to V<sub>3</sub>. Ejection fraction of the left ventricle was 60% by portable echocardiography, and the left atrium and right ventricle were enlarged and the interventricular septum was hypokinetic. His chest X-ray findings were normal (Figure 1).

At the emergency department, we administered inotropics, normal saline, and oxygen. The patient also received aspirin and heparin. His systolic blood pressure was maintained at 110/70 mmHg. Coronary an-



**Figure 2.** Initial chest and lower leg CT images (A) showing intraluminal filling defects in both main pulmonary artery and left popliteal vein. Follow-up chest and lower leg CT images (B) showing no evidence of pulmonary thromboembolism or deep vein thrombosis.

giography (CAG) was performed immediately to rule out acute coronary syndrome due to; continued chest discomfort, T-wave inversion by 12-lead ECG, and abnormal echocardiographic findings. The CAG showed a myocardial bridge in the mid to distal left anterior descending coronary artery, but no significant stenotic lesion or spasm was observed that could explain his clinical findings. To investigate the cause of hypoxemia, we carried out chest and lower leg computer tomography (CT). The CT scan showed intraluminal filling defects in both the distal main pulmonary artery and the left popliteal vein (Figure 2A). He was diagnosed to have massive a pulmonary thromboembolism and deep vein thrombosis. Because of the massive pulmonary thromboembolism with hypotension, we performed thrombolytic therapy using intravenous recombinant tissue plasminogen activator (t-PA, alteplase) after confirming the absence of contraindications. Alteplase 100 mg was administered intravenously over two hours, and after measuring his activated partial thromboplastin time, we administered conventional heparin and transferred him to the intensive care unit (ICU). During his one day stay in the ICU, his blood pressure stabilized and his O<sub>2</sub> demand decreased. No complications, such as, gastrointestinal bleeding or intracranial bleeding occurred, and the patient was transferred to the general ward. On admission day 2, we changed the treatment from conventional heparin to low molecular weight heparin (enoxaparin, 1 mg per kg, twice a day). Subsequently, because a hematoma had developed at the angiographic puncture site, we treated him with warfarin starting on admission day 5. Because he had no risk factors of pulmonary thromboembolism or deep vein thrombosis, and there were no abnormal physical examination findings and his past medical history was unremarkable, we measured tumor markers (CEA, AFP, CA 19-9) to rule out an occult malignancy, but results were all negative. When we enquired more about his lifestyle, we found that he had been playing computer games in a seated position for up to five hour at a time, and for an average of 12 hours per day for two weeks before presentation. The therapeutic international normalized ratio (INR,

range 2 to 3) was reached after overlapping enoxaparin and warfarin therapy and the patient was discharged from hospital on 5 mg of warfarin daily. Three months later, follow up chest and lower leg CT revealed no evidence of pulmonary thromboembolism or deep vein thrombosis (Figure 2B). We have followed up him at our outpatient clinic and he showed no anticoagulation related complication. After 6 months of anticoagulation treatment, warfarin was stopped, and 1 month later we then checked for, but failed to find any evidence of, a hereditary hypercoagulable state (complete blood count, coagulation battery, protein C, protein S, antithrombin III, and antiphospholipid antibody).

### Discussion

Massive pulmonary embolism has been defined to be associated with a systolic blood pressure of <90 mmHg or a drop in systolic blood pressure of  $\geq 40$  mmHg from baseline for a period of >15 minutes, not otherwise explained by hypovolemia, sepsis, or new arrhythmia<sup>1,6</sup>. It is a catastrophic entity that often results in acute right ventricular failure and death. In cases of potentially fatal massive pulmonary embolism, patients are at serious risk of death due to right ventricular failure within the first hour of the event<sup>7</sup>. Mortality can be reduced by prompt diagnosis and therapy.

Certain risk factors increase the likelihood of acute deep venous thrombosis and pulmonary embolism. Acquired risk factors include reduced mobility, total hip and knee replacement, surgery for hip fracture and trauma and spinal cord injury, stroke, malignancy, acute and chronic medical illness, antiphospholipid antibody syndrome, pregnancy, the postpartum period, hormone replacement therapy, oral contraceptives, chemotherapy, and central venous catheterization<sup>1-3,5</sup>. Genetic disorders also increase the risk of thrombosis and events, such as, protein C, protein S, and antithrombin III deficiencies. Furthermore, factor V Leiden, which causes activated protein C resistance, is the most common genetic risk factor of thrombophilia<sup>4</sup>.

Prolonged air or ground travel also increases the risk

of thromboembolism, as do occupations that require long periods of sitting<sup>3</sup>. Deep vein thrombosis as a result of prolonged sitting was first recognized during the Blitzkrieg in World War II, when cases of fatal embolism were noted among Londoners who sat for long periods in deckchairs in air-raid shelters<sup>8</sup>. Thereafter, Homans<sup>9</sup> reported that thromboembolism may occur after prolonged sitting in various situations, such as, in airplanes and cars, and even at the movies. Thromboembolism after prolonged travel achieved notoriety in 1988 when Cruickshank et al.<sup>10</sup> dubbed it “economy class syndrome”, although this term had already been used by others to describe travel in cramped conditions<sup>11</sup>. Immobility, aggravated by limited space in economy class, is presumed to increase the risk of pulmonary embolism<sup>10</sup>.

Recently, computer gaming has become a widespread pastime among young people in Korea. Many young men play computer games for hours per day - some even become professional computer gamers. Beasley et al.<sup>3</sup> reported a new variant thromboembolism associated with computer gaming or work for 12 hours a day, and suggested that the condition be called “eThrombosis”. In Korea, only one previous case report has been issued concerning thromboembolism associated with prolonged sitting at a computer<sup>12</sup>, and this patient expired due to a massive thromboembolism and deep vein thrombosis, according to the autopsy report. Here, we describe the case of a young man who recovered from a life-threatening pulmonary thromboembolism that required thrombolysis, and which was attributed to computer gaming. It appears that steps are required to educate the public concerning the risks posed by computer gaming and occupations that require sitting for long periods, particular, in terms of the risk of acute pulmonary embolism.

### Summary

Pulmonary thromboembolism occurs in old patients with risk factors, such as, immobilization, chronic medical disease, trauma, a hereditary hypercoagulable state,

and others. However, we experienced a young man with a massive pulmonary thromboembolism attributed to immobilization due to computer gaming. The patient had been playing computer games in a seated position for at least five hour continually, and for twelve hours per day over a two-week period. The 36-year-old patient was transferred to our institute rule out the possibility of an acute myocardial infarction. Computer tomography revealed intraluminal filling defects in the distal main pulmonary artery and the left popliteal vein. He received thrombolytic therapy and subsequently recovered without complications. This case raises the possibility that prolonged computer gaming is a risk factor of thromboembolism in young adults.

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