Acute Pulmonary Thromboembolism in the Context of Outing Restrictions during COVID-19 Pandemic

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COVID-19 has spread around the world rapidly. Outing restrictions are performed globally to prevent the further spread of infection. A healthy 49-year-old man was taken in the ambulance due to severe dyspnea. For the previous 10 days, he had been performing remote work at home mainly in a sitting position due to the outing restrictions for the prevention of COVID-19 spread. On admission, his blood pressure was low and could not be measured by automated sphygmomanometer, heart rate was 127 bpm, and oxygen saturation was 98% with oxygenation of 10L/min by a mask with a reservoir. Admission blood tests demonstrated markedly elevated D-dimer of 4.13 μg/mL and NT-proBNP of 6973 pg/mL. Echocardiography demonstrated dilatation of the right ventricle and D-shaped deformity of the left ventricle without wall motion abnormality. We started continuous intravenous norepinephrine and dobutamine due to the pre-shock status, and chest computed tomography revealed pulmonary thromboembolism (PTE) from the bilateral main pulmonary arteries to the subsegmental arteries. We introduced extracorporeal membrane oxygenation and performed surgical embolectomy. After the surgery, his vital signs and circulation stabilized. Twenty days after the surgery, he was discharged from our hospital without any physical impediment. Since he had no apparent genetic factors for thrombotic disorder, long-term sitting at home during remote work could have contributed to the thrombus formation leading to PTE. Regardless of whether individuals are infected by COVID-19, we must be alert for thromboembolism in the context of outing restrictions during the pandemic.

Key words: COVID-19, pulmonary thromboembolism, outing restriction

Introduction

COVID-19 has spread around the world rapidly. Outing restrictions such as lockdown are performed globally to prevent the further spread of infection. We experienced a case of a healthy middle-aged man who developed acute pulmonary thromboembolism (PTE) after a 10-day period of performing remote work at home during the COVID-19 pandemic.

Case Presentation

A healthy 49-year-old man was taken in the ambulance due to severe dyspnea. His body mass index was 26.1 kg/m² and he had no previous history of serious illness. He worked as an electrical system designer. He had come home from the official trip 16 days before. He had gone to work as usual from 11 days to 13 days before. For the previous 10 days, however, he had been performing remote work at home due to the outing restrictions for the prevention of COVID-19 spread. During that period, he had stayed in his room and operated a personal computer mainly in a sitting position. He had secluded himself in his room except to eat meals, use the bathroom and bathe. He had lived in a sitting position not on the chair but on the floor. The day before hospitalization he went outside to ride a bicycle after a long time. However, he lost consciousness and fell from his bike, injuring his head. He was taken to a local hospital and received sutures. Twenty four hours after returning home, he experienced severe dyspnea, called for an ambulance, and was brought to our hospital.

On admission, his vital signs were as follows: consciousness was slightly unclear (Glasgow Coma Scale E3/V4/M5); blood pressure was low and could not be measured by automated sphygmomanometer; heart rate was 127 bpm; oxygen saturation was 98% with oxygenation of 10 L/min by a mask with a reservoir; body temperature was 35.0°C; and respiratory rate was 30 /min. The physical examination showed no swelling in the lower limbs. Admission blood tests demonstrated markedly elevated D-dimer of 4.13 μg/mL and NT-proBNP of 6973 pg/mL. Other laboratory data are shown in Table 1. Electrocardiogram showed negative T wave in II/III/aVF and V1-V4 leads. Echocardiography...
Table 1  Admission blood test

| Test          | Value     |
|---------------|-----------|
| WBC           | 13.8 × 10⁹/μL |
| ALT           | 107 U/L   |
| Lactate       | 17.7 mmol/L |
| RBC           | 4.54 × 10⁹/μL |
| ALP           | 232 U/L   |
| NH₃           | 13.9 μg/dL |
| Hemoglobin    | 14.7 g/dL |
| ChE           | 275 U/L   |
| BS            | 381 mg/dL |
| Hematocrit    | 46.6 %    |
| Na            | 140 mmol/L |
| CRP           | 0.5 mg/dL |
| Platelet      | 166 × 10⁹/μL |
| Cl            | 101 mmol/L |
| K             | 4.0 mmol/L |
| Protein C     | 92 %      |
| PT-INR        | 0.97      |
| UN            | 20.7 mg/dL |
| Protein S     | 80 %      |
| APTT          | 25.4 second |
| Creatine      | 1.54 mg/dL |
| aCL IgM       | < 5 U/mL  |
| Fibrinogen    | 339 mg/dL |
| UA            | 9.7 mg/dL |
| aCL IgG       | < 8 U/mL  |
| D-dimer       | 4.13 μg/mL |
| Ca            | 9.0 mg/dL |
| ANA           | < 40      |
| Mg            | 2.5 mg/dL |
| dsDNA-IgG     | < 10 IU/mL |
| Troponin T    | 0.11 ng/mL |
| Amylase       | 27 U/L    |
| LA            | 1.21      |
| NT-proBNP     | 6973 pg/mL |
| CK            | 90 U/L    |
| CK-MB         | 11 U/L    |
| T-Bilirubin   | 0.5 mg/dL |
| Albumin       | 3.6 g/dL  |
| AST           | 59 U/L    |
| TP            | 6.4 g/dL  |

| Test          | Value     |
|---------------|-----------|
| AST           | 59 U/L    |
| TP            | 6.4 g/dL  |

aCL IgM: anti-cardiolipin antibody immunoglobulin M; aCL IgG: anti-cardiolipin antibody immunoglobulin G; ALT: alanine aminotransferase; ALP: alkaline phosphatase; ANA: anti-nuclear antibody; APTT: activated partial thromboplastin time; AST: aspartate aminotransferase; BS: blood sugar; ChE: cholinesterase; CK: creatine kinase; CK-MB: creatine kinase myocardial band; CRP: C reactive protein; dsDNA-IgG: anti-double stranded DNA immunoglobulin G; LA: lupus anticoagulant; NT-proBNP: N-terminal fragment of pro-B-type natriuretic peptide; PT-INR: prothrombin time-international normalized ratio; RBC: red blood cell; TP: total protein; UA: uric acid; UN: urea nitrogen; WBC: white blood cell.

demonstrated dilatation of the right ventricle and D-shaped deformity of the left ventricle without wall motion abnormality. He was taking no medication before the admission. We started continuous intravenous norepinephrine and dobutamine due to the pre-shock status, and chest computed tomography revealed PTE from the bilateral main pulmonary arteries to the subsegmental arteries without ground-glass opacities (GGO) or mixed GGO and consolidation (Fig. 1).

We introduced extracorporeal membrane oxygenation due to the unstable hemodynamics. The heart team in our hospital concluded that surgical embolectomy was the best choice judging from the following reasons: (1) chest CT showed the acute widespread type PTE; (2) we are more familiar with surgical treatment than catheter intervention in this field; and (3) since this patient was relatively young, it is important to prevent irreversible damage of the right ventricular function as much as possible. Then, we performed the surgical embolectomy using the forceps for minimum invasive surgery under the 5 mm flexible scope guidance (Fig. 2). Using the flexible scope, we could confirm the thrombus which could not be detected under direct vision due to the anatomical running condition of the pulmonary arteries. Finally, we could remove a large number of thrombi (Fig. 3). After the surgery, his vital signs and circulation stabilized. Pulmonary hypertension was improved and deep vein thrombosis (DVT) was detected in the leg veins by ultrasonography. Anti-thrombotic therapy was continued. Twenty days after the surgery, he was discharged from our hospital without any physical impediment.

**Discussion**

We experienced a case of acute PTE in the context of outing restriction during COVID-19 pandemic. To the best of our knowledge, this would be the first report that mentioned surgical embolectomy under endoscopic guidance for PTE. Virchow’s triad—namely, blood flow stagnation, vascular endothelial disorder, and enhancement of blood coagulation ability are classically considered the main factors of thrombus formation. Of these, blood flow stagnation due to remaining in a sitting position for a long time is considered to be the main cause of PTE and DVT during travel, sheltering from natural disasters, or time at home. In particular, there have been reports of PTE and DVT in individuals living in their automobiles as refugees following an earthquake. Due to the outing restrictions enacted to prevent the spread of COVID-19, many people have worked remotely at home, which presumably has led to decrease in active muscle mass and an increase in time spent in a sitting position at home. In our present case of a middle-aged man with no apparent genetic factors for thrombotic disorder, long-term sitting at home during remote work could have contributed to the thrombus formation leading to PTE.

Along with the rapid increase in COVID-19-infected patients, many more people are being forced to shelter at home. The time spent in a sitting position is presumably increasing among these individuals, who would then run a higher risk of PTE as in the current case. However, there have been neither reports of PTE nor proposals for the prevention of PTE in the context of outing restrictions. Regardless of whether people are infected by COVID-19, it is necessary to maintain water intake and aggressive exercise...
of the inferior limbs during periods of remote work and outing restrictions. In addition, we must promote social awareness of the issue worldwide.

**Conclusion**

We experienced a case of acute PTE following the remote work due to outing restrictions under the COVID-19 pandemic.

**Disclosure Statement**

The authors declare that there is no conflict of interest.

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