Acute Pulmonary Thromboembolism and Deep Vein Thrombosis during the Medical Treatment of Acute Aortic Dissection was Successfully Treated by the Combination of Inferior Vena Cava Filter Installation and Anti-Coagulant Therapy: A Case Report

Yoshihiko Kagawa, MD,1 Satoshi Ota, MD, PhD,2 Kozo Hoshino, MD, PhD,3 Norikazu Yamada, MD, PhD,1 Mashio Nakamura, MD, PhD,1 and Masaaki Ito, MD, PhD1

A 71-year-old woman was admitted with Stanford type A acute aortic dissection (AAD). Computed tomography (CT) revealed thrombosis of the false lumen, and we planned to treat medically. She developed transient pleural effusion and hypoxemia, which persisted despite her pleural effusion disappeared. We performed CT and found a large thrombus in the pulmonary artery and femoral vein. We administered low dose-unfractionated heparin and installed a retrievable inferior vena cava filter, which caused the thrombus in the pulmonary artery to disappeared without exacerbating AAD. Our strategy seems to be suitable for acute pulmonary thromboembolism that occurs during the treatment of AAD.

Keywords: acute aortic dissection, acute pulmonary thromboembolism, deep vein thrombosis

Case Report

A 71-year-old woman with a history of hypertension suffered a 2-hour episode of sudden severe back pain and visited our hospital. Her height was 160 cm and her weight was 70 kg. Her blood pressure was 170/90 mmHg in both arms, her heart rate was 55 beats per minute, and her oxygen saturation level was 100% in room air. On auscultation, respiratory sounds were clear, and no murmurs were detected. A laboratory examination demonstrated a white blood cell count of 12100/µL, a hemoglobin concentration of 13.0 mg/dL, and a platelet count of 22.9 × 10^3/µL. Blood biochemical tests produced normal results, including an aspartate aminotransferase level of 17 IU/L, an alanine aminotransferase concentration of 15 IU/L, a creatinine kinase concentration of 71 IU/L, and a creatinine level of 0.58 mg/dL. A troponin T test produced negative results. However, a coagulation test showed that the patient’s D-dimer (60.9 µg/mL) levels were markedly elevated. Electrocardiography revealed a normal sinus rhythm, and did not detect any significant ST-T wave changes (Fig. 1). Chest radiography demonstrated mediastinal widening, but no pleural effusion was observed (Fig. 2). Contrast-enhanced computed tomography (CT) depicted an acute aortic dissection (AAD), which extended from the ascending aorta to the common iliac artery (Fig. 3). A transthoracic echocardiographic examination did not detect any aortic regurgitation, pericardial effusion, or dilatation of right heart. So, we diagnosed the patient with a Stanford type A AAD. As complete thrombosis of the aortic false lumen was achieved on admission, the patient was treated with antihypertensive agents.

The clinical course of the patient’s AAD was good, and she underwent rehabilitation according to the program recommended by the appropriate treatment guidelines. However, transient pleural effusion was showed to develop on chest radiography of day 4, and we considered that this would reduce the patient’s oxygen saturation level. Although the pleural effusion subsequently disappeared on chest radiography of day 12, the patient’s oxygen saturation level remained low (about 85%) in room air. Rehabilitation did not progress because of hypoxemia, and sometimes she walked only in the room with oxygen...
inhalation. We wondered the reason of hypoxemia and considered the possibility of other coexisting disease such as pneumonia, atelectasis, pneumothorax, pulmonary thromboembolism and so on.

On day 17, we performed contrast-enhanced CT in order to follow-up the patient’s AAD and investigate the cause of her hypoxemia. As a result, thrombi were detected in the right main pulmonary artery and left femoral vein (Fig. 4). In addition, an echocardiographic examination detected right ventricular overload (transmitral pressure gradient was 34 mmHg). So, we finally diagnosed the patient with a submassive acute pulmonary thromboembolism (APTE) and proximal deep vein thrombosis (DVT) that had developed during treatment for a Stanford type A AAD although she wore an elastic stocking from admission. Fibrinolysis was difficult in the present case as the patient was in the acute phase of AAD, so we installed an inferior vena cava (IVC) filter and started careful anticoagulation therapy to prevent her AAD worsening. After the initiation of anticoagulation therapy, the patient’s oxygenation level improved immediately. We adjusted the amount of unfractionated heparin administered to achieve an activated partial thromboplastin time of about 45 s. The right ventricular overload was shown to disappear by a transthoracic echocardiographic examination on day 26. After confirming that we were able to achieve sufficient anticoagulation without the patient’s AAD worsening, we retrieved the IVC filter on day 34 after admission (17 days after the implantation of the IVC filter). A test for hereditary thrombophilia was negative, and it was considered that hypercoagulability was the only likely cause of subsequent thromboses during the repair of the AAD. After receiving warfarin-based anticoagulation therapy, the patient was discharged on day 39. A CT scan performed at 4 months after discharge did not detect any residual thrombi in the pulmonary artery or femoral vein, and the patient’s AAD had not worsened.

Discussion

It is necessary for patients to rest during medical treatment for AAD, and they are considered to be at high risk of APTE. Furthermore, hypercoagulability can develop during the acute phase of AAD, resulting in a high risk of venous thromboembolism. On the other hand, anticoagulation therapy can cause AAD to worsen, resulting in the recanalization of false lumens and re-dissection. In a previous study, 2 of 129 cases (1.6%) of Stanford B AAD were complicated by APTE during medical treatment.21 There have been several reports about APTE and DVT occurring during medical treatment for AAD although there were few similarities between the cases (Table 1). Currently, there are no standard treatments for APTE and DVT that occur during medical treatment for AAD. We
considered thrombectomy at this time. But, as she was hemodynamically stable and duration of contraindication to adequate anticoagulant might be temporal, we selected medical therapy with less-invasive IVC filter implantation as first choice. Also, we installed an IVC filter because we could not be certain that anticoagulation therapy would achieve the target therapeutic range in such an acute phase of AAD. After the initiation of anticoagulation therapy, the false lumen in the descending aorta tended to enlarge. However, it did not worsen further after that point. In cases in which the risk of re-dissection increases or a false lumen enlarges during medical therapy, stent grafting might be indicated depending on the anatomical location.\(^6\) We employed a retrievable IVC filter in the present case, and it was considered that this would be sufficient providing that the patient’s AAD and hypercoagulability improved and that she could be released from the requirement to rest. If anticoagulation therapy had not been successful, we planned to replace the retrievable IVC filter with a permanent one. However, permanent IVC filters can cause DVT to relapse, as demonstrated by the Prevention du Risque d’Embolie Pulmonaire par Interruption Cave (PREPIC) study.\(^7\) Fortunately, the patient’s clinical course and CT findings were favorable, so we decided to retrieve the IVC filter.

This does not indicate that anticoagulation therapy is safe for all AAD patients. The dose and timing of anticoagulant therapy are thought to be important. We succeeded this case fortunately, but we cannot suggest how to apply the anticoagulant therapy to those patients from this case alone. Further consideration in more cases is needed.

The treatment for dissection with thrombosed false lumen at the ascending aorta is still controversial.\(^8\)–\(^10\) Like this case, if the frequency of venous thrombosis with ADD is higher with the patient performed medical therapy than surgical, it might be one of the reasons that surgical therapy is recommended for the treatment of ADD.

We need additional evidence about this.

In conclusion, it is important to prevent venous thromboembolisms during medical treatment for AAD. Hypoxemia should be carefully treated whether venous thromboembolisms happen or not, like this case. In AAD patients who subsequently develop APTE, clinicians should consider the state of the AAD and APTE when they decide on the most appropriate treatment. We do not conclude or suggest any efficacy of IVC filter from this case, but installing a retrievable IVC filter and administering anticoagulation therapy might be one of the appropriate treatments for patients that develop APTE during the acute phase of AAD.

**Disclosure Statement**

The authors state that they have no conflicts of interest to declare.

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**Table 1** Previous reports of APTE during medical treatment of AAD

<table>
<thead>
<tr>
<th>Year/Sex</th>
<th>AAD type</th>
<th>Symptoms</th>
<th>Treatment</th>
<th>IVC filter</th>
</tr>
</thead>
<tbody>
<tr>
<td>74-years-old Female(^3)</td>
<td>Stanford B</td>
<td>Dyspnea on day 15</td>
<td>Fibrinolysis</td>
<td>(+)</td>
</tr>
<tr>
<td>76-years-old Male(^4)</td>
<td>Stanford B</td>
<td>Dyspnea on day 21</td>
<td>Thrombolysis aspiration (+)</td>
<td></td>
</tr>
<tr>
<td>60-years-old Male(^5)</td>
<td>Stanford A</td>
<td>Respiratory failure on day 3</td>
<td>Without anticoagulation</td>
<td>Permanent IVC filter</td>
</tr>
</tbody>
</table>

Three reported cases were written about APTE during medical treatment of AAD. (+): not described to be retrieved; APTE: acute pulmonary thromboembolism; AAD: acute aortic dissection; IVC: inferior vena cava
References


