Pulmonary Embolism and Deep Vein Thrombosis Complicating Acute Aortic Dissection during Medical Treatment

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Abstract

Acute aortic dissection of Stanford type A with intramural hematoma was diagnosed based on computed tomography (CT) findings in a 60-year-old man. During medical treatment, pulmonary embolism and deep vein thrombosis developed. CT revealed thrombosis in the right pulmonary artery, and $^{99m}$Tc pulmonary perfusion scintigraphy showed defects in the right lung field. CT showed thrombus in the common iliac vein. An inferior vena caval filter was placed because anticoagulation therapy was contraindicated. A CT scan before discharge showed no thrombus in the pulmonary artery or common iliac vein, but a newly captured thrombus was found inside the filter.

Key words: acute aortic dissection, pulmonary embolism, deep vein thrombus, medical treatment, inferior vena caval filter

Introduction

A 60-year-old man with a history of hypertension and hyperlipidemia was admitted to our hospital complaining of severe chest and back pain of sudden onset. He was 160 cm tall and weighed 65 kg (body mass index, 25.4 kg/m²). On physical examination, he was afebrile with a respiratory rate of 20 breaths/min. On auscultation, respiratory sounds were clear and no murmur was detected. The pulse rate was 55 beats/min and regular. Systolic blood pressure was 80 mmHg (palpation) in both arms. The abdominal findings were unremarkable.

Laboratory examinations revealed a white blood cell count of 10,000/μl, a red blood cell count of 447×10⁴/μl, a hemoglobin of 15.3 g/dl, and a platelet count of 22.3×10⁴/μl. Coagulation tests showed that D-dimer (6.6 μg/ml) and thrombin-antithrombin III complex (19.1 ng/ml) were elevated. Blood biochemical tests showed elevations of blood urea nitrogen (24 mg/dl), total cholesterol (220 mg/dl), and triglyceride (242 mg/dl). Plasma glucose (148 mg/dl) and hemoglobin A1c (6.1%) were also elevated. Arterial blood gas analysis on 3 L/min oxygen by mask showed pH, 7.360; PaCO₃, 42.0 mmHg; PaO₂, 93.7 mmHg; HCO₃⁻, 23.1 mmol/L; and base excess, -1.7 mmol/L.

A chest X-ray film revealed widening of the mediastinum with a cardiothoracic ratio of 53% and normal lung fields. Electrocardiography showed a sinus rhythm of 58 beats/min with incomplete right bundle-branch block. A contrast-enhanced computed tomography (CT) scan of the chest showed a false lumen with intramural hematoma from the ascending aorta to the common iliac artery (Fig. 1). Echocardiography showed normal left ventricular contraction (ejection fraction, 80%) without left ventricular hypertrophy, chamber dilatation, or aortic regurgitation, although there was a slight pericardial effusion (systole, 8 mm; diastole, 3 mm).

Based on these findings, our initial diagnosis was acute aortic dissection (AAD) of Stanford type A. Because of the accompanying intramural hematoma, we chose medical treatment, which included a β-blocker, mainly for antihypertensive control, and bed rest. On day 3, mechanical ventilation was started because of respiratory failure, probably due to pulmonary effusion and the increase in the plasma cytokines (1). Two hours after the start of mechanical ventilation, PaO₂ suddenly decreased, although the setting of the mechanical ventilator had not been changed. Because oxygenation remained low, we suspected pulmonary embolism (PE).
Figure 1. A contrast-enhanced CT scan of the chest showed a false lumen with intramural hematoma from the ascending aorta to the common iliac artery (arrows).

A contrast-enhanced CT scan of the chest performed on day 8 showed a thrombus in the right main pulmonary artery (Fig. 2).

Although the possibility of deep vein thrombosis (DVT) due to bed rest was considered, the findings of physical examination of the lower limbs were unremarkable. Coagulation tests showed that fibrin degradation products (FDP) (108 μg/ml), D-dimer (144.6 μg/ml), and fibrinogen (640 mg/dl) were markedly elevated. We placed an inferior vena caval (IVC) filter in the infrarenal position on day 9. 99mTc pulmonary perfusion scintigraphy on day 11 showed defects in the right upper and middle lung fields, which indicated thromboembolism of the pulmonary artery (Fig. 3, left). Because venous-contrast CT from the pelvis to the lower limb on day 12 showed thrombus in the left common iliac vein, DVT was diagnosed. As recanalization or re-dissection of the thrombosed false lumen is potentially fatal, anticoagulation therapy and thrombolytic therapy were contraindicated. Instead, we used a permanent IVC filter. On day 48, a CT scan showed that the thrombus in the right pulmonary artery and the DVT had disappeared but that the false lumen in the aorta, although reduced in size, remained. In addition, a newly captured thrombus was seen inside the IVC filter (Fig. 4). 99mTc pulmonary perfusion scintigraphy on day 49 showed no defects in either lung field (Fig. 3, right). Coagulation tests showed that FDP (8 μg/ml) was normal. However, D-dimer (8.3 μg/ml) and fibrinogen (550 mg/dl) remained slightly elevated. Because the patient’s general condition had improved, he was discharged from the hospital on day 54.

Discussion

During medical treatment, AAD can be complicated by DVT and PE. However, few cases have been reported. Thirty-nine cases of AAD were treated medically by our department from May 2000 through December 2003, and the present case was the only one complicated by PE (2). According to a recent report, 2 of 129 cases (1.6%) of Stanford type B AAD were complicated by PE during medical treatment (3), a rate consistent with that in our department.

Current guidelines recommend monitoring D-dimer as an indicator for diagnosing PE and DVT (4). Although the value of D-dimer was markedly higher in the present case than in other cases (5), D-dimer was already elevated before DVT and PE developed. D-dimer is not a specific indicator (6) and might be elevated during the medical treatment of most cases of AAD. Therefore, we could not use D-dimer as an indicator of DVT and PE. Because contrast-enhanced CT of the chest and abdomen is normally performed several times during the treatment of AAD in Japan, a large PE can usually be detected. However, contrast-enhanced CT of the lower limb is rarely performed during medical treatment. Therefore, DVT and PE complicating AAD might be difficult to diagnose.

Since this case was AAD of Stanford type A with intramural hematoma, medical treatment was selected on the basis of the guidelines of the Japanese Circulation Society (7), which has reported that medical treatment is more effective than surgical treatment for AAD of Stanford type A with intramural hematoma. PE and DVT should be treated with anticoagulation therapy (4). Mitsube et al (8) have reported a case of PE and DVT during medical treatment of Stanford type B AAD treated with an IVC filter and simultaneous heparin and urokinase, however, PE developed when the patient started walking after bed rest of over 2 weeks. In the present case, PE developed within a week after the onset of AAD. Therefore, anticoagulation therapy and thrombolytic therapy were contraindicated because recanalization or re-dissection of the thrombosed false lumen is potentially fatal. Instead, a permanent IVC filter was used. An IVC filter is indicated to prevent the recurrence of PE due to DVT in the lower limb when anticoagulation therapy or thrombolytic therapy is contraindicated or ineffective. Greenfield and Michna (9) reported that anticoagulation being contraindicated was the most frequent reason for IVC filter placement in their patients.

In the present case, placement of an IVC filter was effective and allowed the patient to be discharged without additional anticoagulation therapy. Although CT showed improvements in the AAD, DVT, and PE before discharge, a newly captured thrombus was identified inside the IVC filter. Decousus et al (10) have demonstrated that while the use of a permanent IVC filter has initial benefits, it may also have long-term complications. There is no consensus about whether or not additional anticoagulation therapy should be performed when a new thrombus from the lower limb is captured by an IVC filter. Ortega et al (11) have reported no significant difference in outcome between patients who received anticoagulation therapy and those who did not after IVC filter placement. In the present case, because the thrombosed false lumen remained even just before discharge, we decided against anticoagulation therapy.
Figure 2. A contrast-enhanced CT scan of the chest showed a thrombus in the right main pulmonary artery (arrows).

Figure 3. Left: $^{99m}$Tc pulmonary perfusion scintigraphy showed defects in the upper and middle right lung fields (day 11). Right: $^{99m}$Tc pulmonary perfusion scintigraphy showed no defects in either lung field (day 49).

Figure 4. Left: CT scans of the chest and abdomen showed that the thrombus in the right pulmonary artery and deep vein thrombus had disappeared, although a false lumen of reduced size remained in the aorta (arrows). Right: A newly captured thrombus was seen inside the IVC filter (arrows).

A foot pump decreases the incidence of DVT by increasing venous return (12). During the medical treatment of AAD, a foot pump can be used to help prevent DVT due to bed rest. We started using this management in our department after our experience with the present case; however, the effects of a foot pump on hemodynamics should be evaluated further.

Here, we reported a case of PE and DVT during medical treatment of AAD in which the placement of an IVC filter was effective. The PE and DVT showed improvement after IVC filter placement, although a thrombus was captured by the IVC filter. Therefore, patients with IVC filters should be
carefully observed to detect long-term complications, such as IVC filter thrombosis. This case demonstrated the difficulty of diagnosing and treating PE and DVT complicating AAD.

References