



Case Report

Management of acute pulmonary embolism after acute aortic dissection surgery



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ABSTRACT

Acute aortic dissection (AAD) continues to be associated with high mortality and morbidity. Pulmonary embolism is also a life-threatening disease. The treatment of these life-threatening diseases remains controversial in case complications arise. Thrombolytic therapy and intensive treatment would be needed to manage these fatal diseases. A 49-year-old man with progressive back pain was admitted to our hospital. Computed tomography (CT) scan revealed type A AAD. Emergency operation for hemiarach replacement was performed. Two weeks postoperatively, the patient's oxygenation worsened and his D-dimer levels elevated. CT scan revealed a massive thrombus in the bilateral pulmonary arteries. Intensive anticoagulation therapy was started immediately. On postoperative day 27, the patient was weaned from mechanical ventilation, but the false lumen with thrombus was recanalized again. The patient was discharged on postoperative day 75 without resulting in major complications for aortic dissection. The diagnosis of pulmonary embolism concomitant with AAD is difficult. The treatment of pulmonary embolism after AAD is controversial. Our strategy seems to be suitable for acute pulmonary embolism that occurs during the treatment of AAD.

Learning objective: The diagnosis of pulmonary embolism concomitant with acute aortic dissection (AAD) is difficult. The treatment of pulmonary embolism after AAD is controversial. Investigating factor XIII levels might help in the early detection of pulmonary embolism.>

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Introduction

Although there have been advancements in surgical techniques and perioperative intensive care, acute aortic dissection (AAD) remains associated with high mortality and morbidity. Pulmonary embolism (PE) is also a life-threatening disease. In the present case, the patient's condition was complicated by PE after AAD surgery; treatment of these catastrophic diseases remain controversial. Thrombolytic therapy and intensive treatment would be needed to manage these fatal diseases.

Case report

A 49-year-old man with a history of hypertension suffered progressive back pain and visited our hospital. His height and weight were 170 cm and 90 kg, respectively (body mass index = 31.14). Computed tomography (CT) scan revealed type A AAD with a patent false lumen from the ascending aorta to the terminal aorta (Fig. 1). The plasma D-dimer levels were elevated (330 µg/mL). The emergency operation was performed and an entry tear was found in the anterior wall of ascending aorta. As the patient's condition had severely deteriorated, hemiarach replacement (HAR) was performed to repair the central entry. We ended the operation by assessing for necrosis in the intestine and fortunately the intestine was not necrotic. Postoperatively, in the first week, the patient was weaned from mechanical ventilation. Although the patient's respiratory status stabilized with bilevel positive airway pressure therapy, on the morning of 14th postoperative day (POD) his D-dimer levels suddenly increased from 38 µg/mL at POD 7 to 94 µg/mL (Fig. 2); at first we thought it was a prolonged AAD

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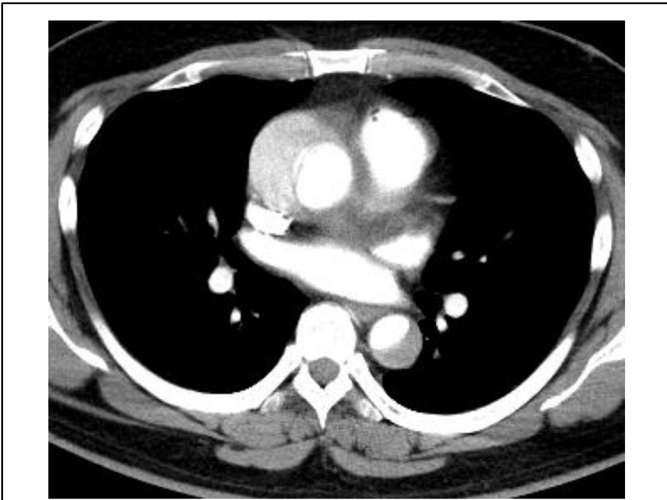


Fig. 1. Type A acute aortic dissection with a patent false lumen.

reaction but his oxygenation was slightly worsened PaO₂/FiO₂(P/F) ratio at 250, making us suspect deep vein thrombus. We checked lower limb veins, and ultrasonography revealed a venous thrombus in the bilateral extremities. Subsequently, his oxygenation gradually worsened with P/F going from 250 to 150 and he required re-intubation. CT scan revealed a massive thrombus filling the bilateral pulmonary artery and it was thought to be the reason for the worsening oxygenation (Fig. 3a and b). Unfortunately the patient had local infectious tendency at the surgical site and

patient state was weakened. According to the cardiovascular team assessment, interventional removal of PE or mechanical inferior vena cava (IVC) filter would not be recommended and the patient state was weak and the P/F ratio was not so bad after re-intubation and thus we decided to treat with thrombolytic therapy.

Thrombolytic therapy with 20,000 units of unfractionated heparin and 480,000 units per day of urokinase was immediately started and administered daily via continuous intravenous infusion for 5 days only, following which the medication was switched to warfarin (Fig. 2). The patient was weaned from mechanical ventilation again on the 27th postoperative day. Totally we needed ventilation therapy for 20 days. CT scan revealed that the major thrombus in the pulmonary artery had disappeared, but the distal arch and descending aorta, which were changed to thrombosed type after central repair operation, had re-changed to a patent false lumen type again (Fig. 3).

While under treatment with warfarin, the patient was discharged on the 75th postoperative day.

Discussion

AAD is a catastrophic disease; its associated mortality rate increases by 1% every hour and by 40%–50% within 48 h of occurrence if the patient is not treated surgically [1]. The all-cause in-hospital death rate associated with AAD after conservative and surgical treatments remains high at 31.6% and 16.7%, respectively [2]. PE is also a life-threatening disease [3] and may prove to be fatal if it gets complicated. Estrera et al. have reported that the condition of 2 (1.6%) of 129 patients with Stanford type B AAD was complicated by acute PE [4].

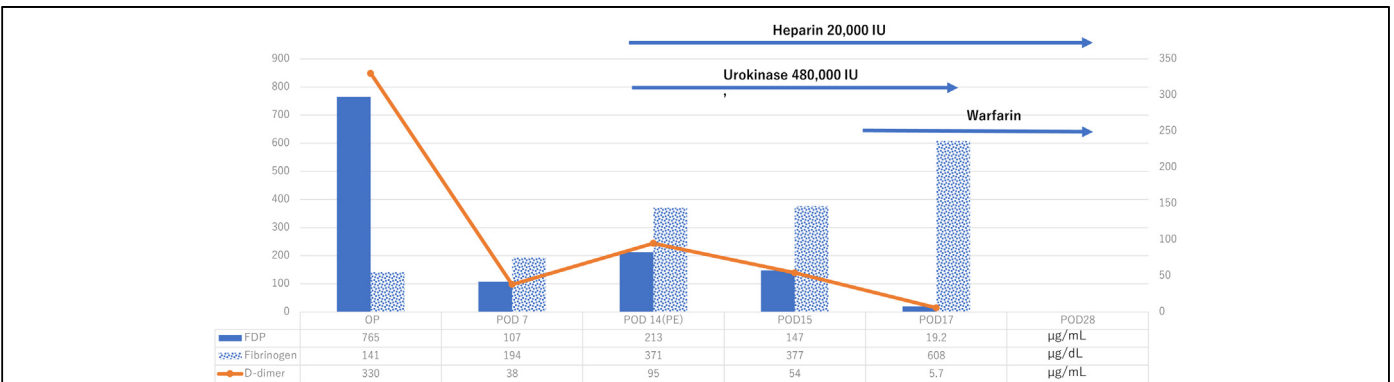


Fig. 2. Graph showing fluctuations in D-dimer level and other factors. On the 14th postoperative day, the patient's D-dimer levels increased again to 94 μg/mL. Anticoagulation therapy is presented toward the top of the graph. FDP, fibrin degradation products; PE, pulmonary embolism; POD, postoperative day.

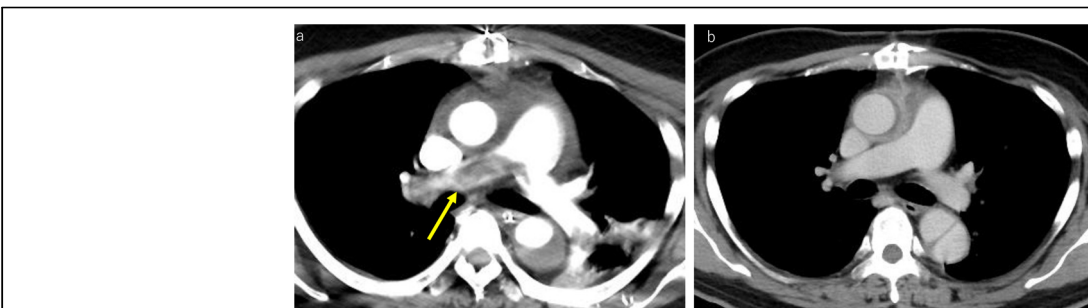


Fig. 3. (a) Massive thrombus filling the bilateral pulmonary artery. (b) Disappearance of the major thrombus in the pulmonary artery. The descending aorta was changed to a patent false lumen type again.

AAD itself or perioperative bleeding and transfusion of blood products may promote the development of a venous thrombus [5]. In our patient, the preoperative D-dimer levels markedly increased to 330 $\mu\text{g}/\text{mL}$ and then decreased sooner. However, they rapidly increased again to 95 $\mu\text{g}/\text{mL}$ 2 weeks postoperatively. In general, we should consider the possibility of a systemic thrombus with such D-dimer levels, but we thought it to be a prolonged reaction of AAD as a recanalization of false lumens or redissection although worsening oxygenation suggested PE existence. In patients who are more likely to develop thrombosis at lower limb venous level, rehabilitation therapy must be provided and thromboembolism should be prevented when long-term rest is required. However, our patient received prophylaxis therapy by wearing compression stockings or sequential calf compression of the lower limbs. Consequently, he developed PE 2 weeks postoperatively (Fig. 2). Liu et al. have reported that disseminated intravascular coagulation-like coagulopathy in patients with AAD is caused by blood flow in the nonendothelialized false lumen, which results in the release of large amounts of cytokines [6]. Thus, AAD activates a procoagulant state. Furthermore, cardiopulmonary bypass amplifies the reaction; thus, systemic activation of coagulation and fibrinolysis as well as inhibition of anticoagulation was observed [7]. In patients requiring a long rest period such as our patient, the possibility for more intensive prophylactic therapy should be considered.

In general, during the acute phase of AAD, hypercoagulability can develop, and it may cause the recanalization of false lumens and re-dissection. However, retrospectively fluctuation in D-dimer levels was not a normal finding after AAD surgery in patients with thrombosis (Fig. 2). If we could have been able to identify PE occurrence earlier, the patient's condition would not have worsened. Tang et al. have reported that the combined measurement of D-dimer and factor XIII levels can help identify PE. Patients with PE exhibit high D-dimer and low factor XIII levels, whereas those with aortic dissection exhibit high factor XIII levels [8]. Thus, investigating factor XIII levels might help early detection of PE. Unfortunately, we did not collect factor XIII so these comparisons could not be made, but we would like to use them in future cases.

It remains controversial how to treat PE patients concomitant with AAD whether to choose fibrinolytic therapy, surgical embolectomy, percutaneous catheter directed treatment, pharmacomechanical approach, and IVC filter. We had no choice to start anticoagulation therapy to our patient because of massive PE even though it was 2 weeks postoperatively. If the remaining dissecting aortic wall were not stable, which occurs in conditions such as the dilatation of aortic diameter and periaortic hematoma, therapeutic anticoagulation might have caused devastating effects. Consequently we believe that the treatment provided was

effective, as the PE clot disappeared and the patient was weaned from mechanical ventilation, even though a thrombus in the false lumen at the residual dissected aorta had re-changed to a patent false lumen type again (Fig. 3d).

The duration of anticoagulant therapy for PE is decided on case-to-case basis. However, in our patient, warfarin therapy was administered for half a year for the disappearance of the PE clot in addition to preventing the recurrence of PE.

In conclusion, it is worth reporting that intensive anticoagulation therapy can save lives in patients with potentially fatal PE concomitant with AAD without resulting in major complications.

Disclosure statement

The authors have no funding, no financial relationship, and no conflicts of interests.

Ethical statement

The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. Written informed consent was obtained from the patient for use of data for academic publication purposes.

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