Hemoptysis is occasionally encountered in patients with a chronic thoracic aortic aneurysm usually as a result of rupture, but it is rare in the setting of acute aortic dissection (AAD). Although the mechanism of this rare complication has not yet been sufficiently elucidated, a fatal consequence is likely to follow\(^1,2\) and emergency aortic surgery is mandatory.

**Case Report**

A 77-year-old woman with a history of percutaneous coronary intervention (PCI) to the right coronary artery 3 weeks previously presented to a nearby clinic complaining of the sudden onset of epigastric pain and hematemesis. Gastroduodenal endoscopy failed to reveal the origin of the bleeding, and a Stanford type A AAD was suggested by enhanced computed tomography (CT). The patient was transferred to our hospital for surgery.

There were no significantly abnormal findings on physical examination. Her blood pressure was well controlled under administration of nicardipine. Laboratory tests revealed a white blood cell (WBC) count of 9,300/µL, hemoglobin of 8.3 g/dL, fibrin degradation product of 6.40 µg/mL (normal range < 5.00 µg/mL), and D-dimer of 4.04 µg/mL (normal range < 0.5 µg/mL). A chest x-ray revealed widening of the mediastinum and consolidation in the right hilar region (Figure 1A). Electrocardiography showed sinus rhythm of 58 beats/minute with an abnormal Q wave in leads III and aVF, which was observed after the recent PCI (Figure 1B). Enhanced CT revealed dissection and dilatation of the ascending aorta with a small intimal tear and partially enhanced false lumen. The left and right pulmonary arteries were normal, but there was a massive posterior mediastinal hematoma and it extended along the right pulmonary artery. Hemoptysis is a lethal sign of aortic dissection, therefore, emergency ascending aortic replacement was performed with a good clinical outcome.

**Discussion**

In patients with a Stanford type A AAD, acute aortic valve regurgitation, acute myocardial infarction, rupture, and cardiac tamponade are common, but hemoptysis is rare.\(^3\) Anatomically, the ascending aorta and pulmonary trunk have a common adventitia. Therefore, bleeding into the posterior aspect of the ascending aorta can extend around the pulmonary trunk, and from there along the left and right pulmonary arteries.\(^4,5\) Because the right pulmonary artery is just behind the ascending aorta, it may preferentially be affected as shown in the CT images of the present case (Figure 1A). Sueyoshi, *et al.* reported a case of ruptured intramural hematoma extending along the pul-
In our case, hematoma in the peribronchovascular sheath is considered to be the main mechanism of hemoptysis.

DAPT may have played some role. In the present case, relatively stable vital signs and low WBC and D-dimer values were observed despite the massive hematoma. Physicians should be aware of this type of hemoptysis occurring even with these blood test results in patients under the influence of DAPT.

If a diagnosis of AAD is delayed, huge hematoma may obstruct a pulmonary artery and the superior vena cava, and right heart failure and hemoptysis may ensue. Without prompt surgical intervention, this might also have occurred in our case.

A review of the literature revealed the prognosis of hemoptysis accompanying type A AAD is poor. Only 4 out of 15 patients survived the condition. Four patients died in an acute phase without any surgical intervention, while the others passed away during or after surgery. Physicians should be aware of this rare complication because its early diagnosis and operation is warranted.

Disclosures

Conflicts of interest: The authors have no conflicts of interest to declare.

References
