Pulmonary Artery Obstruction Caused by Thoracic Aortic Dissection

— A Case With Unique Pathological Findings —

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Pulmonary artery obstruction is a rare complication of acute thoracic aortic dissection. A 74-year-old woman was admitted to hospital with respiratory distress. Computed tomography scan showed right pulmonary artery occlusion and a thoracic aortic dissection of the Stanford A type. Lung perfusion scan revealed a defect in the entire right lung field. These findings mimicked acute pulmonary thromboembolism accompanying aortic dissection. On the other hand, pulmonary angiography revealed a round smooth defect of the right pulmonary artery, indicating an extrinsic compression. The patient finally died of cardiac tamponade. Autopsy disclosed that the right pulmonary artery was compressed by a hematoma in the adventitial space around the pulmonary artery. Such a mechanism of pulmonary artery obstruction caused by acute aortic dissection is unique, and is distinct from that caused by chronic non-dissecting aortic aneurysms, which themselves compress the pulmonary arteries. This complication inevitably follows aortic rupture; therefore, emergency operation to repair the dissected aorta must be performed to avoid the following catastrophic event. (*Circ J* 2004; 68: 392–395)

Key Words: Adventitia; Dissecting aneurysm; Hematoma; Pulmonary artery obstruction

The most common complication of thoracic aortic dissection is rupture, usually into the pericardial space, pleural cavity or mediastinum. Obstruction of the pulmonary artery is a rare complication of thoracic aortic dissection and is often misdiagnosed^{1,2} or detected at autopsy³ because it mimics acute pulmonary embolism. Buja et al reported that obstruction of the pulmonary artery was caused by hematoma in the adventitial space³ but the clinical aspects and pathophysiology of this rare complication are not yet sufficiently understood.

Case Report

A 74-year-old woman with a history of systemic hypertension presented to hospital complaining of severe dyspnea, which had begun suddenly on the day of admission. On physical examination, she was afebrile with a respiratory rate of 24 breaths/min and the lungs were clear on auscultation. Cardiac examination revealed a grade 2/6 diastolic murmur at the parasternal border. The pulse rate was 103 beats/min and regular, with a blood pressure of 132/72 mmHg in the left arm and 112/62 mmHg in the right arm. The patient's neck veins were distended. The abdomen was unremarkable. Laboratory examinations revealed a white blood cell count of 22,000/ μ L, red blood cell count of 363×10^4 / μ L, hemoglobin of 11.4 g/dl, and platelet count of 20.2×10^4 / μ L. Coagulation tests showed the activated partial thromboplastin time, prothrombin time and fibrin

degradation product were normal, but D-dimer was elevated (8.2 μ g/ml; normal range <1.0 μ g/ml). C-reactive protein was 20.8 mg/dl (normal range <0.4 mg/dl). Blood biochemical tests showed that concentrations of aspartate aminotransferase, alanine aminotransferase, lactic dehydrogenase, blood urea nitrogen, serum creatinine, low density lipoprotein cholesterol, and triglyceride were all within the normal range. Plasma glucose was elevated to 185 mg/dl, but hemoglobin A1c was normal. Arterial gas analysis on 10 L/min oxygen inspiration was pH 7.45, PaCO₂ 29 mmHg, PaO₂ 51 mmHg, SaO₂ 88%, and HCO₃⁻ 20.1 mmol/L. A chest x-ray revealed widening of the mediastinum, a cardiothoracic ratio of 60%, and normal lung fields. Electrocardiography showed sinus tachycardia of 110 beats/min without any specific changes. Echocardiography revealed an intimal flap that pulsed in the ascending aorta, and a tear was observed above the sinus of Valsalva. Moderate aortic regurgitation was shown by color Doppler echocardiography with no pericardial effusion. Right cardiac catheterization disclosed a right ventricular pressure of 60/12 mmHg, a mean right atrial pressure of 14 mmHg, and a superior venous pressure of 12 mmHg. The floating-catheter was not advanced into the pulmonary artery in order to avoid dissemination of the supposed intraluminal thrombus. Computed tomography scanning revealed dissection and dilatation of the ascending aorta and aortic arch with a false lumen enhanced by contrast medium. Dissection of the descending aorta was also observed, but the false lumen was already thrombosed. The left pulmonary artery was normally enhanced with contrast medium, whereas the right pulmonary artery was not enhanced, suggesting an obstruction. An entire right lung field defect in the lung perfusion scan examination also indicated obstruction of the right pulmonary artery.

According to these findings, our initial diagnosis was

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Fig 1. Pulmonary angiogram. The right pulmonary artery is interrupted proximally by a smooth round defect from below (arrow), indicating extrinsic compression. Dilatation of the interruption using a PTA balloon of 5.0mm in diameter could not maintain blood flow when the balloon was deflated. The left pulmonary artery and pulmonary trunk are intact.

thoracic aortic dissection of Stanford A type complicated with pulmonary thromboembolism of the right pulmonary artery. Emergency operation for the thoracic aortic dissection and the supposed right pulmonary thromboembolism was discussed; however, the surgeon wanted to improve the patient's respiratory condition before operation. Mechanical ventilatory support was thus started, and pulmonary angiography was performed. It showed total occlusion of the right pulmonary artery. However, the outline of the occluded pulmonary artery was smooth and round inside (Fig 1), suggesting an extrinsic compression. Angioplasty of the pulmonary artery was attempted with a PTA balloon of 5.0mm in diameter. Although balloon inflation to a pressure of 10atm could alleviate the compression, blood flow in the right pulmonary artery could not be maintained because of immediate reocclusion when the balloon was deflated. This result also indicated compression of the pulmonary artery from outside the vessel wall. The patient's respiratory condition worsened rapidly because of new onset of acute respiratory distress syndrome, and the operation was cancelled. On the fifth hospital day, cardiac arrest occurred abruptly because of cardiac tamponade, and the patient died.

Autopsy Findings

(1) The pericardial space was filled with blood and thrombus, indicating cardiac tamponade because of rupture of the ascending aorta.

(2) The dissection of the ascending aorta extended above the sinus of Valsalva to the descending aorta at the level of the diaphragm. The false lumen was thrombosed in the descending aorta and arch, but only partially in the ascending aorta (Fig 2). The maximum outer diameter of the dilated aorta was 6cm in the ascending section.

(3) There was a massive hematoma in the adventitial space of the pulmonary trunk and right pulmonary artery, but less hematoma around the left pulmonary artery. The hematoma compressed the right pulmonary artery from

Fig 2. Overview of the large vessels at autopsy. The intimal lumen of the externally compressed right pulmonary artery is observed in cross-section at the distal site. Part of the hematoma in the adventitial space around the right pulmonary artery is present in the section of pulmonary artery, which extends from the massive hematoma in the adventitial space of the pulmonary trunk. The hematoma compresses the right pulmonary artery from below (*). The entrance tear of the aortic dissection was located 4cm cephalad to the aortic valve. The ascending aorta has been separated from the heart, and the superior vena cava is opened in this photograph. a-Ao, ascending aorta; d-Ao, descending aorta; Hem, hematoma; r-PA, right pulmonary artery; SVC, superior vena cava; Tr, trachea.

below (Fig 2). The right pulmonary artery was pinched by the dilated ascending aorta and the hematoma. The inner lumen of the right pulmonary artery was interrupted near its origin (Figs 2,3A).

(4) Slice sections of the ascending aorta and the pulmonary artery showed that the penetration of the intima and the inner layer of media was at a posterior site of the ascending aorta. The outer layer of media was also perforated in the posterior direction. The hematoma was enveloped by a layer of the adventitia, which successively covered the ascending aorta and the pulmonary trunk (Fig 3B).

(5) The proximal portion of the ascending aorta and pulmonary trunk was surrounded by the hematoma in the common adventitial space (Fig 3C). Extravasated blood through the adventitia was also observed (Fig 3C). No other communication between the ascending aorta and the pericardial space was detected, indicating that cardiac tamponade was induced by the extravasated blood from the rupture of the adventitia.

Discussion

Chronic, non-dissecting, thoracic aortic aneurysms are considered a common causes of acquired pulmonary artery obstruction, which is not surprising in view of the close anatomical relationship between the ascending thoracic aorta and the pulmonary artery. The etiology of such aneurysms includes syphilitic aortitis,⁴ atherosclerosis, and cystic medial necrosis.⁵ In particular, syphilitic aortitis is known to commonly affect the ascending aorta. The chronic inflammation damages the aortic wall and forms a huge aneurysm that compresses or penetrates the pulmonary artery or other adjacent structures.⁶

Conversely, acute aortic dissection infrequently causes pulmonary artery obstruction,⁷ partly because of the nature of this acute condition. A dissected thoracic aorta usually

Fig 3. Successive horizontal sections of the ascending aorta, pulmonary artery, and hematoma. Each slice is approximately 15 mm in thickness. (A) Section at the level below the bifurcation of the pulmonary trunk. The right pulmonary artery is pinched between the hematoma and the dilated ascending aorta, which is 6 cm in diameter. The hematoma extends only slightly into the adventitial space around the left pulmonary artery. Note that the wall of the pulmonary trunk and left pulmonary artery have been lost in the preparation process of the slice section. (B) Section at the level of the entrance tear in the ascending aorta. The perforation of the intima and the inner layer of media can be seen at the posterior site of the ascending aorta (arrowhead). The outer layer of media is also penetrated in the posterior direction forming a hematoma. The hematoma is enveloped by a layer of the adventitia that successively covers the ascending aorta and the pulmonary trunk. (C) Section of the proximal portion of the ascending aorta and pulmonary trunk. The outer layer of media is perforated. The ascending aorta and pulmonary trunk are surrounded by the hematoma in the common adventitial space. Extravasated blood through the adventitia can be seen. No other communication between the ascending aorta and the pericardial space was detected in a detailed exploration, indicating that cardiac tamponade was induced by the extravasated blood from the rupture of the adventitia. Ad, adventitia; Ao, ascending aorta; FL, false lumen; IL, inner layer of media; Hem, hematoma; l-PA, left pulmonary artery; OL, outer layer of media; PT, pulmonary trunk; r-PA, right pulmonary artery; Ex, extravasated blood pool.

ruptures into the pericardial space, pleural cavity or mediastinum instead of undergoing enormous expansion, because the outer wall of the false channel is much thinner and is penetrated more easily than is the inner flap of the dissected media⁸

Interestingly, most cases of pulmonary artery obstruction caused by thoracic aortic dissection have been reported to be associated with hematoma around the pulmonary artery when autopsy or operation was performed^{3,9,11,13} which is consistent with the findings of the present case. Anatomically, the ascending aorta and pulmonary trunk have a common adventitia and therefore, extravasation of blood into the adventitia of the ascending aorta commonly extends into the adventitial space of the pulmonary trunk, and from there into the adventitial space around the left and

right pulmonary arteries. As extravasated blood in the adventitia of the aorta and pulmonary arteries is driven by aortic pressure, it can compress the lumen of the pulmonary artery, which has a relatively low pressure⁹. The resultant hematoma in the adventitia around the pulmonary arteries plays an essential role in obstruction of the pulmonary arteries.

Moreover, the dilated ascending aorta itself also participates in obstruction of the right pulmonary artery, as shown in the slice section of the present case at autopsy. Because the right pulmonary artery runs just behind the ascending aorta, obstruction preferentially occurs in the right pulmonary artery!^{1-3,10-14}

Finally, the patient developed cardiac tamponade because of penetration of the intra-adventitial hematoma into the

parietal pericardial space⁹ Therefore, in this case, aortic rupture proceeded in three steps. First, rupture of the intima and the inner layer of media causing aortic dissection; second, rupture of the outer layer of the media into the adventitial space around the ascending aorta; and third, rupture of the adventitia into the parietal pericardial space.

Obstruction of the pulmonary artery caused by acute thoracic aortic dissection is a unique complication that develops in a manner distinct from obstruction caused by chronic, non-dissecting, thoracic aneurysms. We must be aware that this complication inevitably follows an acute aortic rupture and therefore, emergency operation to repair the dissected aorta should be considered in order to avoid the subsequent catastrophic event.

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