Severe Respiratory Failure Caused by Recurrent Pulmonary Hemorrhage in Takayasu's Arteritis*

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We encountered a 52-year-old man with Takayasu's disease (pulless disease) and severe respiratory failure due to recurrent pulmonary hemorrhage. Angiography revealed occlusion of multiple branches of the pulmonary artery, which were filled via collateral circulation from the coronary, intercostal, and intermainary arteries. This is a rare case that causes massive pulmonary bleeding and respiratory failure in Takayasu's arteritis.

(Pulmonary Disease, Pulmonary Hemorrhage, Takayasu's Arteritis, Angiography.)

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Pulless disease (Takayasu's arteritis) occurs worldwide, although the majority of cases have been reported from Asia and Africa and most large series consist of Asians, with a heavy predilection for women. This is a systemic disease with generalized as well as local symptoms. More than half the patients with this disease have pulmonary artery involvement. Local symptoms in pulmonary artery involvement are subtle such as a hemoptysis. We report a case of severe respiratory failure caused by recurrent pulmonary hemorrhage in Takayasu's arteritis.

CASE REPORT

A 52-year-old man was admitted to the hospital with acute respiratory distress. He had received the diagnosis of pulseless disease 4 years prior to this, due to the absence of the right radial artery pulsation and bruits of bilateral carotid arteries. His condition was stable until severe and progressive dyspnea awoke him from his sleep.

On physical examination, he appeared to be in respiratory distress. The blood pressure was 209/90 mm Hg as measured by arterial transducer applied to the right femoral artery. The pulse rate was 140 beats per minute, and the respiratory rate was 40 breaths per minute. His body temperature was 37.1°C. Bruits were audible at both carotid arteries and the mid-abdomen. Harsh systolic heart murmur was audible in the third left sternal border. His breath sounds were slightly wheezy and crackled in both lungs. The hemoglobin value was 9.8 g/dl; the WBC count was 22,500/mm³, with 70 percent neutrophils; and the platelet count was 43.5 x 10⁴/mm³. Arterial blood gas analysis disclosed that partial pressure of oxygen was 31.0 mm Hg; partial pressure of carbon dioxide, 43.5 mm Hg; and pH, 7.43. A chest x-ray film showed bilateral consolidation, especially at the right upper middle lobe. He was intubated and ventilated by a respirator under positive end-expiratory pressure. The pulmonary arterial pressure was 10 to 43 mm Hg. Massive blood was aspirated from the endotracheal tube, and recurrent bleeding was observed during hospitalization.

Selective angiography showed obstruction of the left common carotid artery, the right subclavian artery, and the bilateral bronchial arteries, and a marked stenosis of the left subclavian artery. The celiac artery and the superior mesenteric artery also were occluded, and both were filled by the meandering artery from the inferior mesenteric artery. Pulmonary arteriography showed complete occlusion of vessels to the right middle and lower lobes and the left lingual lobe, without signs of either cutting off or thrombus (Fig 1). Bronchial arteries were communicated with bilateral coronary arteries and filled the right middle lung. Aortography showed an enlarged intercostal artery and an intermammary artery supplying both lungs (Fig 2). His bleeding gradually improved with antihypertensive drug treatment and supportive therapy.

DISCUSSION

Clinical signs and symptoms of pulmonary artery involvement in Takayasu's arteritis are usually subtle and rarely appreciated in conditions such as minor little hemoptysis or dyspnea. The incidence of pulmonary artery involvement has been reported in 118 out of 210 patients. However, there has been no reported case that has been asymptomatic, presenting with an abrupt onset of respiratory failure. In our patient, acute respiratory distress was caused by massive recurrent pulmonary hemorrhage. It is considered that the abnormality of the pulmonary arteries is chronic, because rich collateral circulation and mild elevation of the pulmonary arterial pressure were present. Pulmonary arteries were obstructed in many vessels, and the bronchial artery was obstructed at the ostium. Lung perfusion may have been filled by an enlarged intercostal artery, intermammary artery, and both coronary arteries with connection to bronchial arteries. So it is less likely that pulmonary hemorrhage was due to occlusion of pulmonary arteries. There are three suspected causes for his bleeding: rupture of collateral vessels, rupture of microaneurysm due to

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vasculitis,' or hyperemic response.

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