

Aortobronchial Fistula Secondary to Chronic Post-traumatic Thoracic Aneurysm

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A few patients with traumatic aortic laceration remain undiagnosed and survive long enough to develop a chronic aneurysm. Such aneurysms are frequently asymptomatic; alternatively, they may manifest themselves in the form of chest pain, dysphonia, dysphagia, bronchial irritation, or sudden death. A case of aortobronchial fistula secondary to a chronic post-traumatic aneurysm of the aortic isthmus is presented. Hemoptysis was the main sign. The affected segment of the thoracic aorta was replaced with a Dacron graft and a left superior lobectomy was performed. Nevertheless, the patient died during the postoperative period due to adult respiratory distress syndrome. Pathogenesis, diagnosis, and management of aortobronchial fistulae are discussed. (*Tex Heart Inst J* 1996;23:174-7)

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Aortobronchial fistulae are uncommon, although their frequency is most likely underestimated; more than 30% are diagnosed at autopsy.¹ Aortobronchial fistulae are more frequent in the left bronchial tree, and the communication is typically between the aneurysm and the membranous wall of the bronchus.^{2,3} Two recent reviews of the literature revealed fewer than 70 reported cases of fistulous communication between the thoracic aorta and the tracheobronchial tree.^{1,2} Causes of aortobronchial fistula include pulmonary tuberculosis,⁴ staphylococcal pneumonia,⁵ bronchogenic carcinoma,⁶ and aortic dissection without aneurysm.⁷ Nevertheless, thoracic aortic aneurysms (mainly mycotic or atherosclerotic) are 1 of the most common causes of aortobronchial fistula.^{1,2} During the last few years, most reported cases of aortobronchial fistula have occurred after implantation of a prosthetic vascular graft in the thoracic aorta.^{1,2} Aortobronchial fistulae arising from post-traumatic chronic thoracic aneurysms are extremely rare, and only a few reports have been published.^{8,9} We report the case of a fistula between the aorta and the tracheobronchial tree in a patient with a chronic post-traumatic aortic aneurysm.

Case Report

A 44-year-old man was admitted to our hospital in June 1995 because of recurrent hemoptysis. Past history included tobacco use and alcohol abuse. Nine years before, he had suffered multiple facial injuries in a traffic accident. Chest radiography was not performed at that time. During the subsequent 9 years, the patient remained asymptomatic. Three days before admission, the patient had several episodes of hemoptysis. On admission, he was afebrile and denied having experienced weight loss, anorexia, or chest pain. Electrocardiography showed him to be in sinus rhythm at 70 beats/min. His chest radiograph revealed a rounded mass arising from the left upper mediastinum, with an interstitial infiltrate in the left upper lobe (Fig. 1). The results of blood analyses on admission are given in Table I. Viral markers for hepatitis B and C were negative. Coagulation test results were within the normal physiologic range.

A computed tomographic scan of the thorax revealed an aneurysm of the thoracic aorta immediately distal to the origin of the left subclavian artery. Magnetic resonance imaging showed a saccular aneurysm (Fig. 2) with a parietal thrombus; the aneurysm adhered to the left upper bronchus (Fig. 3).

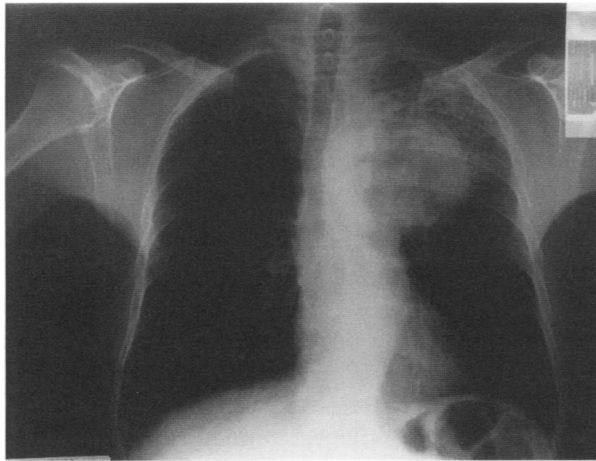


Fig. 1 Chest radiograph showing a rounded mass arising from the left upper mediastinum, with an interstitial infiltrate in the left upper lobe.



Fig. 2 Magnetic resonance image in coronal projection, showing a pediculate aneurysm (arrows) with parietal thrombus.

TABLE I. Laboratory Data at Admission

Hemoglobin (g/L)	134
Hematocrit (%)	0.41
Platelet count ($\times 10^9/L$)	345
WBC count ($\times 10^9/L$)	13.3
Neutrophils (%)	78
Lymphocytes (%)	9
Monocytes (%)	11
Eosinophils (%)	2
Basophils (%)	0.4
Creatinine (mg/dL)	0.7
Sodium (mEq/L)	140
Potassium (mEq/L)	4
Total bilirubin (mg/dL)	0.68
Direct bilirubin (mg/dL)	0.16
Alkaline phosphatase (IU/L)	187
GGT (IU/L)	64
SGOT (IU/L)	40
SGPT (IU/L)	21
Albumin (g/dL)	3
α_1 -globulin (g/dL)	0.6
α_2 -globulin (g/dL)	1.1
β -globulin (g/dL)	1
γ -globulin (g/dL)	1.4

GGT = γ -glutamyltransferase; SGOT = serum glutamic-oxaloacetic transaminase (aspartate transaminase); SGPT = serum glutamate pyruvate transaminase (alanine transaminase); WBC = white blood cell

Surgery was performed 4 days after admission. Selective endotracheal intubation was used in the anesthetic management. A left posterolateral thoracotomy was performed through the 5th intercostal space. The aneurysm adhered firmly to the posterior



Fig. 3 Magnetic resonance image in coronal projection, showing the adherence between the aneurysm and the left upper bronchus (arrow).

surface of the left upper lobe, which was partly atelectatic. We cannulated the left atrium and the femoral artery and performed a left atrial-to-left femoral bypass using a centrifugal pump system (BioMedicus, Medtronic Blood Supply Systems; Anaheim, CA) and a heat exchanger (Avecor, Omnitherm; Plymouth, MN). Proximal and distal control of the aneurysm was achieved. The bypass was started, the aorta was clamped, and the aneurysm was opened, its attachment to the left upper lobe still intact. The thrombus was removed and a Dacron graft was anastomosed with continuous suture. The aortic cross-

clamp was removed and the bypass was discontinued. Subsequently, a left upper lobectomy was performed. The patient was extubated 16 hours after surgery. The course was uneventful during the 1st and 2nd postoperative days, and neurologic complications were not observed. Nevertheless, the patient developed adult respiratory distress syndrome; he needed mechanical ventilation on the 3rd postoperative day and died 2 days later of severe hypoxia. A culture of the thrombus was negative. Microscopic examination of the aneurysmal wall showed fibrous connective tissue lined by thrombus. Histologic examination of the upper left lobe revealed chronic bronchitis, but no lung cancer.

Discussion

Pathogenesis. Deceleration injuries of the thoracic aorta arising from motor vehicle accidents are associated with high mortality at the scene.¹⁰ The most frequent site of lacerations is 2 to 3 cm distal to the origin of the left subclavian artery.⁹ More than 90% of survivors who reach the hospital are diagnosed and treated.¹¹ Nevertheless, 2% to 5% of patients with traumatic aortic laceration remain undiagnosed and survive long enough to develop a chronic post-traumatic aneurysm.¹² After the inner layers of the aortic wall have been torn, the adventitia may remain intact, or at least strong enough to avoid massive hemorrhage during the acute episode. After several days, a fibrous tissue surrounds the hematoma and a new endothelium forms. Progressive dilation of the aorta may lead to the development of a false aneurysm. The natural history of chronic post-traumatic aneurysms is that of progressive enlargement and rupture.¹¹ The time between the accident and diagnosis ranges from several months to more than 40 years.^{9,13}

One of the least frequent complications of chronic post-traumatic aneurysms is the formation of a fistula between the aorta and the bronchial tree, with subsequent cough, pain, dyspnea, and either recurrent or massive hemoptysis.^{8,9} Compression of the tracheobronchial tree by the systolic expansion of the aneurysm may induce pressure necrosis, pulmonary collapse, infection and subsequent inflammation, and adherence between the aorta and the lung;^{1,2} this last can lead to erosion of the aorta into the pulmonary parenchyma or bronchial wall, precipitating the fistulous communication and the appearance of hemoptysis.^{1,2}

Hemoptysis is usually recurrent, and more than 50% of patients with these aortobronchial communications have an episode of massive hemoptysis.^{1,2} When the fistula is 1st established, a small amount of blood may be expectorated. Due to the thrombogenicity of the pulmonary parenchyma, a clot soon

forms in the fistula and the hemoptysis stops. When the clot lyses or becomes dislodged, a new episode of hemoptysis occurs. If the fistulous communication is large enough, massive hemoptysis and death may result. The time elapsed between the 1st episode of hemoptysis and death ranges between several days and 4 years.²

Diagnosis. Chronic post-traumatic aneurysms are frequently asymptomatic and may be diagnosed during routine chest radiographic examinations.⁹ Apart from rupture and sudden death, such aneurysms may present in association with chest pain, dysphonia due to involvement of the recurrent laryngeal nerve, dysphagia, bronchial irritation, and pulmonary infection.^{1,2,9,11} Aortobronchial fistula should be suspected in all patients with a mediastinal mass and recurrent hemoptysis.

Although chest radiography is useful, it has some limitations because the paramediastinal mass may be interpreted as being a tumor.^{2,14} Bronchoscopy is hazardous: massive hemorrhage has occurred when the bronchoscope has dislodged the blood clot sealing the communication.^{1,2} Computed tomography of the thorax, which reveals both the aortic lesion and secondary changes of the adjacent lung, is the diagnostic procedure of choice.¹⁴ Although the fistulous communication itself is rarely detected, it sometimes manifests as a consolidation of the lung adjacent to the aneurysm.³ Aortography usually does not show the clotted fistula, although several cases of aortographic documentation have been reported.¹⁵ Transesophageal ultrasonography and magnetic resonance imaging are useful techniques, but may be difficult to perform in an emergency.^{1,2}

Management. Management of aortobronchial fistulae associated with chronic post-traumatic thoracic aneurysms includes the repair of the aortic and pulmonary defects.^{1,2} Circulation distal to the fistula may be maintained by left atrial-to-left femoral bypass, although vascular repair can be carried out with aortic cross-clamping alone, if necessary. The thoracic aneurysm is managed with resection of the aneurysm and placement of a prosthetic graft. The vascular graft and suture lines should be protected by interposing viable tissue such as pericardium, pleura, skeletal muscle, or omentum.² An alternative approach, which decreases the risk of graft infection, is to resect the false aneurysm, suture the aorta, and place an extra-anatomic bypass graft.¹ Before the operation, selective intubation is important in avoiding blood dissemination to both lungs; but it must be carried out carefully, due to the risk of massive hemorrhage during endobronchial manipulation.

Treatment of the bronchial lesion depends on its location: direct suture of the bronchial fistula, wedge resection, lobectomy, or pneumonectomy. In any event, long-term antibiotic treatment is mandatory.

Morbidity is high, because the fistula is usually infected.¹ Without surgical intervention, aortobronchial fistulae are uniformly fatal. Early mortality of patients who undergo surgery is around 30%.^{1,2} The causes of death include hemorrhage, pulmonary edema, cardiac failure, pneumonia, and sepsis.^{1,2} Prolonged mechanical ventilatory support and neurologic sequelae are relatively frequent.

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