Left Pleural Hemorrhagic Effusion

A Presenting Sign of Thoracic Aortic Dissecting Aneurysm

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Left hemorrhagic pleural effusion was the presenting sign of painless aortic dissecting aneurysm in two elderly hypertensive patients. Computed tomography (CT) of the chest revealed the aneurysmal dilatation of the thoracic aorta and an intimal flap connecting its descending part with the left pleural space. The patients were treated conservatively with blood transfusions and drugs directed to control blood pressure. The first reported 71-year-old patient remains in stable condition for 16 months without evidence of recurrent active aortic dissection. The second 85-year-old patient remained in stable condition for 28 days, but finally had a second fatal episode of dissection into the left pleural space. The differential diagnosis of nontraumatic left hemorrhagic pleural effusion in an elderly hypertensive patient should include dissecting aneurysm of the descending thoracic aorta and CT of the chest should be performed as the next preferable diagnostic procedure.

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CT=computed tomography

Acute dissection of the thoracic aorta is one of the medical emergencies which demands prompt medical or surgical therapy. The choice of the particular therapeutic approach depends on the site of the intimal flap. While surgical intervention is the preferred treatment for dissection of the ascending aorta, conservative management is suggested for most cases with distal dissection without aortic rupture.1

Pleural effusion, commonly on the left side, is a rare complication of the rupture of the thoracic aorta.2,3 We report two elderly patients with dissecting aneurysm of the thoracic aorta, presenting with left pleural hemorrhagic effusion.

CASE REPORTS

CASE 1
A 70-year-old man with a history of hypertension treated with nifedipine, mild COPD, and chronic renal failure was admitted to the hospital in December 1991 with a 3-day duration of dyspnea. On examination, the patient was a slightly overweight, pale, elderly man in no apparent distress. The pulse rate was 88 beats per minute; respiration rate, 26 breaths per minute; and blood pressure, 160/90 mm Hg. Increased dullness to percussion and decreased breath sounds were apparent over the base of the left lung. Laboratory studies revealed a hemoglobin level of 89 g/L (8.9 g/dl), a leukocyte count of 14.7X10⁹/L, and a serum creatinine value of 247.5 µmol/L (2.8 mg/dl). An ECG was normal. The chest x-ray film demonstrated a left pleural effusion and lobulated aortic contour (Fig 1), and a diagnostic aspiration of the pleural effusion revealed hemothorax. Computed tomography (CT) of the chest revealed dilatation of the descending aorta (diameter, 6 cm) with a 5-cm intimal flap connecting with the left pleural space (Fig 2).


FIGURE 2. Patient 1. The CT scan shows the intimal flap separating the true lumina and false lumina which communicated with the left pleural space that contains pleural effusion.
pleural space (Fig 2). Therapy with propranolol, 120 mg/d, was started immediately, and the patient received two units of packed RBCs. On the 12th day of admission, the patient was discharged in a stable hemodynamic condition (blood pressure, 110 to 130/70 to 80 mm Hg; heart rate, 58 to 60 beats per minute) with recommendation for treatment with atenolol, 100 mg once daily. During 16 months of follow-up, the patient was in stable clinical condition without evidence of recurrence of pleural effusion or any other symptoms or signs of active aortic dissection.

**Case 2**

An 85-year-old woman with a history of hypertension was admitted to the hospital in February 1991 with shortness of breath. The physical examination revealed a pale tachypneic (30 respi- rations per minute) elderly woman in fairly good general condition. Her blood pressure was 120/80 mm Hg and the pulse was regular, 78 beats per minute. The chest examination revealed dullness and decreased breath sounds over the left lower base. Laboratory tests revealed a hemoglobin level of 83 g/L (8.3 g/dl), a leukocyte count of 20.810⁹/L (20,800×10⁹/mm³), a serum urea level of 33.6 mmol/L (94 mg/dl), and a creatinine value of 221 µmol/L (2.5 mg/dl). An ECG showed normal sinus rhythm and signs of left ventricular hypertrophy. The chest x-ray film demonstrated a left pleural effusion, an enlarged silhouette of the heart, and lobulated aortic contour. A chest tube was inserted, and 200 ml of bloody fluid was evacuated. The CT scan of the chest revealed dilatation of the ascending aorta and of the aortic arch and an intimal flap of about 7 cm in the arch extending to the descending aorta and communicating with the left pleural space. Two units of packed RBCs were administered and the patient was treated with propranolol, 80 mg/d, and nifedipine, 40 mg/d. The systolic blood pressure was maintained within the limit of 110 to 130 mm Hg. Because of a secondary infection in the left pleural space caused by *Pseudomonas aeruginosa* and *Proteus vulgaris*, therapy with piperacillin, 16 g/d, was started on the 12th day; the infection resolved within 2 weeks. Dyspnea and hemoptysis appeared on the 28th day and were accompanied by progressive decline of blood pressure. The patient refused any diagnostic or surgical procedures and died within 16 h of the beginning of that fatal episode. An autopsy was not performed.

**Discussion**

Our two patients had a left pleural hemorrhagic effusion as a rare presentation of dissecting aortic aneurysm. They had no evidence of congestive heart failure or severe COPD, but suffered from dyspnea as a main complaint. Dissection of the descending thoracic aorta communicating with the left pleural space was suspected when a hemorrhagic aspirate was obtained and a CT scan of the chest confirmed that diagnosis. Our two patients were treated conservatively by blood transfusion and antihypertensive drugs. One of them died on the 28th day of admission from progressive dissection. The second patient survived, and 16 months after the acute episode no signs of recurrence appeared.

Severe pain is a classic presenting symptom of dissecting aortic aneurysm. Painless dissection was described only in a minority of the patients (5 percent). The clinical presentation in dissection of the ascending aorta usually is aortic insufficiency, heart failure, or pulse deficits. Hemorrhagic pleural effusion, which appears usually on the left side, is a rare complication of thoracic dissecting aortic aneurysm and occurs in about 10 percent of patients, the majority of whom have distal dissection.

The radiologic assessment of patients with a suspected aortic dissection is a cornerstone of the diagnostic process. A chest roentgenogram reveals pathologic findings such as abnormal aortic contour, widened mediastinum, "calcium signs," or pleural effusion in 80 to 85 percent of cases. A contrast-enhanced CT scan currently is the method of choice for the diagnosis and management of patients with suspected dissection; however, because of false-negative results of CT scans, echocardiography with Doppler flow studies also should be done in patients with suspected ascending aortic dissection. Angiographic evaluation should be reserved for patients in whom the previously noted studies were inconclusive or for those in whom surgery is planned.

A current opinion states that uncomplicated descending aortic dissection should be treated medically. Surgical approach should be reserved for patients with proximal dissection or for cases of distal dissection complicated by rupture, compromise of a major vessel, or continued or recurrent pain. In spite of the aforementioned, our first patient was treated medically because of the concomitant presence of chronic renal failure; patient 2 and her family refused surgery for her.

Dissecting thoracic aneurysm in elderly patients had, probably, some distinctive features. Glower et al. showed the prevalence of descending aortic dissection in patients who were older than 65 years. An extensive review of the English-language literature did not disclose any case of dissecting aneurysm of the aorta in the manner of our patients: painless dyspnea without signs of congestive heart failure and associated with hemorrhagic left-sided pleural effusion. We suggest that the advanced age of these patients led to atypical manifestation of the aortic dissection. It is well known that older patients often have painless myocardial infarction. Some investigators have shown that the aging process is accompanied by an increased threshold for perception of pain, and this may explain the absence of pain in our patients.

We suggest that in elderly hypertensive patients with a left-sided pleural effusion and abnormalities of aortic contour evidenced in the chest x-ray film, the differential diagnosis should include dissecting aneurysm of the thoracic aorta. Computed tomography of the chest should be the next preferable investigative step.

**References**

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**Childhood Pulmonary Alveolar Proteinosis**

**Extracorporeal Membrane Oxygenation With Total Cardiopulmonary Support During Bronchopulmonary Lavage**

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Partial cardiopulmonary bypass with extracorporeal membrane oxygenation to allow bilateral bronchopulmonary lavage in pulmonary alveolar proteinosis has been described. However, this technique is complicated by a very low arterial P O2 and cardiovascular embarrassment. Total cardiopulmonary support avoids these problems and was successfully used in a 2½-year-old girl.

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| BAL=bronchoalveolar lavage, ECMO=extracorporeal membrane oxygenation; PAP=pulmonary alveolar proteinosis |

Pulmonary alveolar proteinosis (PAP) is a rare condition, particularly so in children, and is of unknown etiology.1-8 Phospholipid material accumulates in alveolar spaces, impairing gas exchange and leading to arterial hypoxemia. The most effective treatment for PAP is bronchopulmonary lavage, which attempts to temporarily clear the alveoli of accumulated material and results in clinical remission, which may be temporary,2,4,7,9 or prolonged.1,5,6,10-13 We report a case of childhood PAP in which lavage was conducted on full venoarterial cardiopulmonary bypass with extracorporeal membrane oxygenation (ECMO). Immediate dramatic clinical and radiologic improvement followed. To our knowledge, this technique has not been reported previously and yet offers significant advantages over techniques currently employed.

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**CASE REPORT**

A second child of healthy unrelated parents was born at 36 weeks’ gestation weighing only 2.2 kg (tenth percentile). Investigation of failure to thrive required several hospital admissions. At 2 years of age, still without a definitive diagnosis, she became tachypneic with a slight cough. The cough worsened and 2 months later, hospital admission was arranged for increased dyspnea.

At the time of hospital admission, she was noted to be extremely thin, weighing only 8 kg (well below the third percentile). Marked pectus excavatum and clubbing were noted, with cyanosis, tachypnea, and marked intercostal and subcostal recession. There were scattered fine crepitations on auscultation, and saturations with 28 percent oxygen by face mask were 80 to 94 percent. Results of cardiovascular examination were normal.

The radiographic appearance was of bilateral reticulonodular shadowing (Fig 1). Electrocardiogram showed P pulmonale and right ventricular hypertrophy. High-resolution lung computed tomography (CT) revealed extensive consolidation within both lungs, most marked posteriorly. Histologic examination of a percutaneous lung biopsy specimen showed almost every alveolar space to be filled with a dense PAS-positive granular eosinophilic material, consistent with a diagnosis of PAP. Silver stains did not reveal pneumocystic organisms and culture of the biopsy specimen was negative. Suction catheter specimens were negative for Pneumocystis immunofluorescence.

Treatment with antibiotics and steroids was commenced which resulted in a slight improvement in saturations. Enteral nutrition via nasogastric tube was commenced to give 1,340 kcal/d, resulting in a slight weight gain. Despite these improvements it was believed that her small size and degree of respiratory embarrassment would make unilateral lavage via double-lumen endotracheal intubation a high-risk procedure. Therefore, 6 weeks following hospital admission, in November 1992, she was transferred to Groby Road Hospital for bronchopulmonary lavage on full venoarterial ECMO.

General anesthesia was induced and the patient was intubated and ventilated. Biomedicus cannulas were used to cannulate the right internal jugular vein (14F) and right common carotid artery (12F). A chest radiograph confirmed the cannula positions and bypass was established uneventfully.1,4 Ventilation was ceased and pulmonary lavage was carried out with pH-normalized saline

**FIGURE 1.** Radiologic appearance at presentation, showing bilateral reticulonodular shadowing.