cryptogenic BOOP will respond to corticosteroid therapy and often resolve completely. Some patients suffer progressive lung impairment and death.

High-dose corticosteroids and cyclophosphamide failed to prevent progression of this patient's pulmonary or renal disease, suggesting that despite the subacute onset of his condition, the disease process in both organs accelerated rapidly at the end of his clinical course.

The sequence of histologic changes was that of BOOP followed by the development of florid PAN of the classic type, apart from one focus of necrotizing vasculitis in a medium-sized pulmonary artery. Although the lung exhibited one focus of necrotizing vasculitis, further vasculitis was not evident in lung at autopsy despite florid vasculitis elsewhere.

The pathogenetic processes underlying the various forms of bronchiolitis obliterans is unknown. It has been suggested that, when associated with connective tissue diseases, heart-lung transplantation, and perhaps viral infection, the disease represents an autoimmune process whereby the host immune system responds to airway epithelial cell class II antigen expression induced by gamma interferon.\(^{13,14}\) Spontaneous gamma interferon production by cells obtained at bronchoalveolar lavage was measured in this patient as part of another study and was very high (390 units/10\(^6\) cells in 24 h [normal individuals release \(\leq 10\) units/10\(^6\) cells in 24 h]).\(^{14,15}\) This finding is consistent with the above hypothesis.

The failure to respond to treatment is a little unusual for BOOP, although death from progressive disease has been well described.\(^{14}\) This patient clearly had a very aggressive form of the disease.

This report describes an association between BOOP and PAN, which suggests that similar pathogenetic mechanisms may be present in some patients with these disorders. Also, since BOOP can clearly precede the clinical manifestations of PAN, awareness of these facts may lead to careful monitoring for PAN and therefore to earlier therapy.

REFERENCES


Bilateral Proximal Pulmonary Artery Aneurysms Simulating Hilar Adenopathy*  

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Proximal pulmonary artery aneurysms (PAAs) are rare. Most are associated with secondary pulmonary hypertension or a variety of rare systemic disorders. An asymptomatic adult patient presented with bilateral hilar enlargement on a routine chest roentgenogram. Computed tomography of the chest revealed 5 cm bilateral proximal PAAs with a normal pulmonary trunk. The clinician should consider proximal PAA in the differential diagnosis of hilar enlargement.  

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DcOVA = specific diffusing capacity; PAA = pulmonary artery aneurysm; PT = pulmonary arterial trunk

Bilateral hilar enlargement is a relatively frequent roentgenographic pattern. In almost all cases, bilateral hilar enlargement is due to enlarged hilar lymph nodes or hilar vessels. Bilateral hilar adenopathy is typically seen with either benign granulomatous disease (sarcoid and infection) or malignant disease. Vascular causes for bilateral hilar enlargement are far less common. The majority of proximal PAAs, those involving the pulmonary trunk and/or major divisions, are associated with pulmonary hypertension, either primary or secondary to a number of cardiopulmonary disorders.\(^{14}\) Isolated proximal PAAs in the absence of pre-disposing conditions are particularly unusual.\(^{4}\) Also, all

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previously reported cases of idiopathic proximal PAAs have involved the main PT.4-6

We describe the case of a 64-year-old asymptomatic woman with bilateral, symmetric aneurysms of the right and left pulmonary arteries.

CASE REPORT

A 64-year-old black woman, nonsmoker, was referred for evaluation of bilateral hilar enlargement. She denied dyspnea, cough, chest pain, or other cardiopulmonary symptoms. She denied travel outside the United States and denied exposure to silica or beryllium. Her past medical history included pulmonary tuberculosis for which a left upper lobectomy was performed 40 years previously. On physical examination, she appeared well. The blood pressure was 112/58 mm Hg; pulse, 88 beats per minute; and respirations, 18/min. Cardiac examination revealed a normal S1 and S2 with no abnormal pulsations. There was a grade 2/6 systolic flow murmur at the left sternal border which did not change with inspiration. The jugular venous pressure was not elevated, and there was no hepatosplenomegaly, lymphadenopathy, or peripheral edema. The chest revealed changes consistent with the prior lobectomy and auscultation was otherwise normal. There were no stigmata of Marfan's syndrome.

The chest roentgenogram revealed bilateral, symmetric hilar enlargement. The heart and distal pulmonary vasculature were within normal limits. Comparison with old chest roentgenograms showed stable bilateral hilar enlargement for at least five years. Contrast-enhanced computed tomography of the chest demonstrated PAAs, measuring 4 to 5 cm in diameter, involving the distal portion of each main pulmonary artery (Fig 1). The PT was not involved, and peripheral pruning of the pulmonary vasculature was not seen. Routine blood test and VDRL results were normal. An electrocardiogram was normal. Echocardiogram demonstrated normal-sized cardiac chambers with good contractility and mild concentric left ventricular hypertrophy. Right ventricular hypertrophy, right ventricular diastolic overload, and valvular structural abnormalities were not seen. Doppler echocardiography demonstrated moderate tricuspid regurgitation with a peak regurgitant jet velocity of 3 m/s. Utilizing the modified Bernoulli equation, the calculated pulmonary artery systolic pressure was 41 mm Hg. Flow across the pulmonary valve was normal and the PT was not dilated. Pulmonary function testing showed the FVC to be 84 percent predicted; FEV1, 77 percent; FEV1/FVC ratio, 0.68; TLC, 89 percent; and the Dco/VA ratio, 106 percent. Room air arterial blood gases showed a PaO2 of 83 mm Hg, PaCO2 of 44 mm Hg, and pH of 7.46. A ventilation/perfusion lung scan showed a large matched ventilation/perfusion defect in the left upper lung, consistent with the prior lobectomy, and a small subsegmental perfusion defect in the right middle lung, giving a low probability for pulmonary embolism.

**Figure 1.** Contrast enhanced CT demonstrates bilateral 4 to 5 cm pulmonary artery aneurysms. The pulmonary trunk and the proximal right pulmonary artery are normal.

**DISCUSSION**

Aneurysms of the PT and main branches are rare. In a review of 109,571 autopsies in 1947, Deterling and Clagett found eight cases of proximal PAA. Most cases are accompanied by pulmonary hypertension from a variety of causes, most frequently secondary to congenital heart disease with left-to-right shunt.2-3 Other causes include syphilis, Marfan's syndrome, vasculitides such as Behcet's disease, Hughes-Stovin syndrome, giant cell arteritis, and septicemia with endovascular seeding (mycotic aneurysm), and trauma with pseudoaneurysm formation.4 Also, aneurysmal dilatation of the central pulmonary arteries, usually in conjunction with dilatation of the PT, has been described with primary pulmonary hypertension.5 A few cases of proximal PAA have been reported in the absence of pulmonary hypertension or other known causes.5 Most cases of proximal PAA involve the PT, with or without involvement of the major branches.6 The definition of proximal PAA is somewhat obscure. Some authors define aneurysms of the PT as having a diameter of more than 4 cm.2 Older autopsy series have defined proximal PAA as a permanent, more or less circumferential dilatation of the pulmonary artery with some organic degeneration of its walls.4 A more clinically useful definition is a roentgenographically demonstrable sac formed by the dilatation of the proximal arteries. This excludes diffuse dilatation, as can occur with some cases of pulmonary hypertension of any cause,4 and the entity known as idiopathic dilatation of the PT.10

There are rare case reports of proximal PAA in the absence of any secondary diseases or with minimal pulmonary hypertension.4-6,11 The PT is involved in over 80 percent of all cases of proximal PAA, with or without involvement of one or both major divisions. Rarely are both main divisions affected without PT involvement.3

The clinical presentation of proximal PAA largely depends on the underlying condition. Symptoms attributable to the aneurysm itself include chest pain, dyspnea, and cough.2,3 Erosion into a bronchus can lead to fatal hemoptysis.5 Other deadly complications are dissection and rupture, which usually occur in the setting of severe pulmonary hypertension.4 Intraaneurysmal thrombosis leading to distal embolization7 and cor pulmonale8 have been reported with idiopathic cases of proximal PAA.

Data concerning the natural course of proximal PAA are limited. One case report describes a stable course over eight years in a patient with moderate pulmonary hypertension (65/32 mm Hg) of unknown etiology.10 Another report describes marked enlargement over a three-year period in a patient with mild pulmonary hypertension (45/18 mm Hg).11 Most authors recommend surgical intervention once the diagnosis is established because of the risk of rupture.2 However, most cases of rupture have occurred in patients with severe pulmonary hypertension.4 It is unclear whether the idiopathic variety poses the same risk. The presence of hemoptysis is a marker of instability and is a strong indicator for intervention regardless of etiology.4

The pathogenesis of proximal PAA is unclear. Most cases are associated with pulmonary arterial hypertension or an underlying disorder causing arterial wall weakness. Virtually all cases coming to autopsy or surgery have some degree of medial degeneration and decrease in elastic fibers on
microscopy. This appears to be true whether pulmonary hypertension is present or absent. Cystic medial necrosis of the pulmonary arteries occurs in association with Marfan's syndrome and long-standing pulmonary hypertension. Atherosclerotic degeneration of the pulmonary arteries has also been described with congenital heart disease and associated pulmonary hypertension. Since only a small percentage of patients with pulmonary hypertension develop proximal PAA, it is speculated that a congenital defect in the connective tissue of the arterial wall is a contributing factor.

The patient presented in this report is asymptomatic and has had roentgenographically stable proximal PAA for at least a five-year period. The etiology of her aneurysms remains unknown. The patient did not have underlying causes of secondary pulmonary hypertension such as heart disease, chronic obstructive pulmonary disease, or thromboembolic disease. Although Doppler echocardiography revealed mild pulmonary hypertension, it is unlikely that this factor should have caused such extensive aneurysm formation. Pulmonary angiography was not performed since computed tomography was diagnostic.

In summary, we have described an adult patient with only mild pulmonary hypertension who presented with bilateral proximal PAA which spared the main trunk. This is a rare presentation of a rare disease. Proximal PAA is an extremely rare disease which belongs in the differential diagnosis of hilar enlargement. Contrast-enhanced computed tomography can be a valuable noninvasive diagnostic tool as it can help to separate lymph nodes from vascular dilatation.

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Implantation Metastasis of Carcinoma after Percutaneous Fine-Needle Aspiration Biopsy*

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Implantation of malignant cells along the needle tract is an extremely rare but potential complication following percutaneous needle aspiration biopsy of malignant lesions. Percutaneous fine-needle aspiration biopsy (FNAB) has recently received more attention for cytologic diagnosis of bronchogenic carcinoma because of its high diagnostic yield, simplicity, and low morbidity. On the other hand, dissemination of cancer cells by needle aspiration biopsy can change a potentially resectable localized lung cancer to an unresectable one. We report two cases: one patient underwent FNAB of a metastatic left adrenal mass that seeded a paraspinal muscle implantation of malignant cells that subsequently developed a tumor mass, and the second patient had tumor cell implantation in the chest wall after FNAB of a pleural-based adenocarcinoma of the lung. The theoretical and practical importance of tumor cell spread along the needle tract is discussed. Because of its rare incidence, however, this complication should not affect the use of needle aspiration biopsy in bronchogenic carcinoma, although care should be undertaken during the procedure.

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FNAB = fine-needle aspiration biopsy

Percutaneous needle aspiration has been widely used for diagnosis of malignant neoplasms. The common complications that might be encountered in performing needle aspiration biopsy of the lung are pneumothorax, hemorrhage, infection, and air emboli. Implantation of tumor cells along the needle tract is an extremely uncommon complication of this technique. Among thousands of fine-needle aspiration biopsies (FNAB) performed in lung cancer patients, there are only a few reports of tumor implantation after this procedure. Most of these complications have followed the use of cutting needles or relatively large-bore needles. However, chest wall implantation of bronchogenic carcinoma after FNAB has occurred, although it is even more rare. The role of immediate radiotherapy after biopsy to prevent tumor implants in the needle track was raised in one report. Because of this rare but significant complication, we present herein two cases: one patient who had an adenocarcinoma of the lung and developed paraspinal muscle implantation after FNAB of his left adrenal mass; the other patient developed chest wall implantation after pleural needle biopsy was performed.

CASE REPORTS

CASE I

A 49-year-old man was examined for obstructive pneumonia of the left upper lobe. Bronchoscopy was done by an outside hospital

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