the orifice of the right coronary artery.4,11

We believe that intermittent coronary artery spasm as the cause of angina pectoris in the absence of atherosclerotic coronary artery disease is a definite entity. Holter monitoring and coronary angiography during the attacks of pain may be fruitful in establishing the diagnosis.

References


Pulmonary Sarcoidosis Presenting as Bronchogenic Carcinoma*

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Massive pulmonary fibrosis simulating bronchogenic carcinoma on chest roentgenograms, planigrams and bronchograms is a distinctly unusual manifestation of sarcoidosis. Bronchial stenosis can result from any of three types of bronchial involvement: compression of the major bronchi by enlarged perihilar nodes, sarcoid lesions in the bronchial wall, and diffuse bronchial constriction associated with massive fibrosis. The symptoms produced by sarcoidosis can be quite similar to those commonly associated with bronchogenic carcinoma, and when combined with suspicious findings on chest roentgenograms, planigrams, and bronchograms, can lead to a strong clinical suspicion of carcinoma. Since the two diseases can co-exist in the same patient, diagnostic thoracotomy is usually required even with a prior diagnosis of sarcoidosis.

The pulmonary fibrosis seen in patients with advanced sarcoidosis can roentgenographically simulate pulmonary tuberculosis, idiopathic pulmonary fibrosis, congestive heart failure, pulmonary involvement with scleroderma, rheumatoid lung, pneumoconiosis, or lymphangitic carcinoma.1 The presence of a mass lesion simulating bronchogenic carcinoma is distinctly unusual. We report a case where massive fibrosis suggested bronchogenic carcinoma on chest roentgenogram, at bronchoscopy, and on bronchograms.

Case Report

A 53-year-old white farmer from Texas was referred to the Ochsner Medical Center in December, 1973 for evaluation of a mass lesion in his left upper lobe demonstrated on chest roentgenogram. He had smoked approximately six cigars daily for the ten years between 1946 and 1956. In 1956 a “spot” had been discovered in the lower left lung field on chest roentgenogram, which was no longer present on repeat study one year later. A routine chest roentgenogram in 1969 had revealed a diffuse interstitial infiltrate predominantly in the upper half of both lung fields. He remained asymptomatic, and a repeat study in 1971 revealed no change in the infiltrate. Evaluation at another hospital had included a left scalene node biopsy which had revealed a noncaseating granulomatous adenitis.

Upon admission, the patient reported malaise with a persistent nonproductive cough for the preceding six months, but he denied dyspnea, wheezing, chest pain, hemoptysis, fever, or weight loss. Auscultation of the lungs revealed a prolonged expiratory phase without rales or wheezes. There was no adenopathy or finger clubbing. Skin tests for tuberculosis, histoplasmosis, and mumps were negative. The chest roentgenogram revealed diffuse interstitial changes with a marked prominence of the left upper lobe hilus, suggesting a mass lesion, with radiation of fibrotic strands toward the periphery (Fig 1). Tomograms revealed stenosis of the apicoposterior segment bronchus of the left upper lobe. Pulmonary function studies revealed a pure restrictive impairment, with the vital capacity 76 percent of predicted, the FEV1 83 percent of predicted, and the maximum breathing capacity 111 percent of predicted. The resting alveolar-arterial oxygen gradient was elevated to 28 mm Hg, and the resting arterial oxygen pressure was normal (77 mm Hg), with no change on exercise.

Fiberoptic bronchoscopy revealed stenosis of the apicoposterior and anterior segmental bronchi of the left upper lobe, with normal-appearing mucosa. Brush biopsies of the segmental bronchi were negative for malignancy, and multiple washings were negative for fungi and acid-fast bacilli. A bronchogram showed a persistent filling defect of the anterior segment bronchus, with definite narrowing of the apicoposterior segment bronchus (Fig 2).
FIGURE 1. EPA roentgenogram of chest demonstrating diffuse interstitial changes with a mass lesion at the upper left hilum with stranding toward the periphery.

Because of the suspicion of malignancy, exploratory thoracotomy was performed. Multiple hard, whitish plaques were present throughout the parenchyma of the left lung. A large, hard, fixed hilar mass was present near the left upper lobe bronchus, which had caused collapse of a portion of the anterior segment of the left upper lobe. The mass was very firm and fixed to the pulmonary artery. Multiple biopsies of areas of the mass and of distal parenchymal lesions were all compatible with sarcoidosis. Because of this finding, and because of the risk involved in attempted removal of the mass, resectional surgery was not performed. Microscopic examination of the biopsies revealed extensive granulomatous inflammation of undetermined etiology, consistent with sarcoidosis. Special stains for acid-fast bacilli and fungal organisms were negative, as were cultures for bacteria, fungi, and Mycobacterium tuberculosis.

The patient was discharged on a regimen of 40 mg of prednisone daily, which was tapered gradually to 10 mg daily, and he remained asymptomatic. A repeat chest roentgenogram after seven months of low dose prednisone revealed little change (Fig 3). Repeat bronchograms revealed an apparent decrease in the stenosis of the apicoposterior segment bronchus, with persistent occlusion of the anterior segment bronchus (Fig 4). Repeat pulmonary function studies at this time were unchanged from the preoperative ones.

FIGURE 2. Left bronchogram demonstrating occlusion of the anterior segment bronchus with narrowing of the apicoposterior segment bronchus.

FIGURE 3. EPA chest roentgenogram 7 months after operation demonstrating little change from preoperative roentgenogram.

FIGURE 4. Repeat left bronchogram after 7 months therapy with prednisone demonstrating apparent decrease in stenosis of the apicoposterior segment bronchus with persistent occlusion of the anterior segment bronchus.
DISCUSSION

Although the roentgenographic changes due to pulmonary sarcoidosis may resolve completely in a large percentage of cases, probably 20 percent will ultimately develop widespread fibrosis with eventual distortion of the lung architecture. This distortion is caused by a gradual upward migration of parenchymal lesions over a period of years, which results in dense fibrosis with upward retraction of the hila. Coarse, irregular strands commonly extend from the hila toward the periphery.

The presence of a mass lesion simulating bronchogenic carcinoma is an unusual finding, and was not mentioned in a series of 1,254 patients with proven sarcoidosis, 94 percent of whom had abnormal chest roentgenograms. On reviewing the literature we could find only four cases similar to ours. Hahn's patient was found to have multiple hard, dense nodules which resulted in a "frozen hilus." Multiple biopsies of the lung, hilar mass, and pleural implants were suggestive of sarcoidosis. Resectional surgery was deemed inadvisable, and the patient remained well for 11 years after operation without further specific therapy.

Sarcoidosis commonly causes symptoms which are also usually associated with bronchogenic carcinoma, including fatigue, malaise, weight loss, cough, dyspnea, chest pain, and occasionally hemoptysis. When these are combined with suspicious chest roentgenograms, planigrams, bronchoscopic findings, and bronchograms, the clinical suspicion of carcinoma is even stronger. Since bronchogenic carcinoma has been reported to co-exist with sarcoidosis, even if the diagnosis of sarcoidosis has been firmly established previously, diagnostic thoracotomy will be required to rule out co-existent carcinoma.

The finding of bronchial stenoses in our patient contributed further to the suspicion of carcinoma. Three different types of bronchial involvement have been described in sarcoidosis. The first is caused by enlarged hilar nodes which compress the major bronchi and which may result in collapse or eventual bronchiectasis. This is quite rare, in spite of frequent massive enlargement of the mediastinal nodes. The second type is the occurrence of sarcoid lesions in the bronchial wall. This type is not unusual and diagnosis can frequently be made by means of bronchoscopic biopsy. The third type is the diffuse bronchial constriction which occurs in the later stages of fibrosis and results in narrowing of the major bronchi. Longcope and Freiman suggest that this is not infrequent.

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REFERENCES

4 Longcope WT, Freiman DG: A study of sarcoidosis based on combined investigation of 160 cases including 30 autopsies from Johns Hopkins Hospital and Massachusetts General Hospital. Medicine 31:1-132, 1952

A Technique for Unknotting an Intracardiac Flow-Directed Balloon Catheter*


Described is an unusual complication occurring during right-sided cardiac catheterization using a 7F flow-directed balloon catheter. During an attempt to direct the catheter from the main pulmonary artery into the pulmonary wedge position, the tip became entangled in a loop of catheter and knotted. Initially, all attempts to unknot or remove the catheter failed. A movable-core guide wire was passed through the major lumen of the catheter, resulting in the immediate unknotting of the catheter, thus allowing its withdrawal.

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UNKNOTTING INTRACARDIAC FLOW-DIRECTED BALLOON CATHETER 731