the absence of ST changes. They represent early appearance of the atrial repolarization (Ta) wave. Normally, Ta occurs invisibly during the QRS, but appears earlier in many pericarditis patients producing PR segment deviation. All our subjects did not have PR deviations before testing, so that exercise appeared to bring out latent PR segment deviation with and without pericarditis. Return of elevated STs to the baseline is harder to explain in view of the intensifying effects of exercise on acute myocarditis, the cause (though presumably subepicardial) of ECG changes in acute pericarditis. Since we would not intentionally exercise patients with acute pericarditis, no series to quantitate this (vs early repolarization) can be designed.

Therefore, acute pericarditis appears to contraindicate exercise challenge, so that responses we report strictly apply to our patient. Yet, they question the proposal that exercise would not change or exacerbate the ST abnormalities of acute pericarditis. Taken with the results in early repolarization, this serendipitous finding reduces or eliminates the reported utility of the exercise ECG in differentiating the two conditions.

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Posterior Mediastinal Sarcoidosis*

We report two cases of enlargement of the posterior mediastinal lymph nodes due to sarcoidosis. Bilateral hilar enlargement, pulmonary parenchymal involvement, or extrathoracic manifestations of sarcoidosis were absent. A diagnostic thoracotomy had to be performed in both instances.

Sarcoidosis is a systemic disease of unknown origin characterized by the presence of noncaseating granulomas in one or more organs. Although intrathoracic lymph nodes are often involved, posterior mediastinal lymph nodes are only rarely affected. To our knowledge, no reports of sarcoidosis of the posterior mediastinal lymph nodes without extramediastinal manifestations of disease have previously been documented in the world literature.

CASE REPORTS

Case 1
A 45-year-old white woman was admitted on May 3, 1982 for investigation of high-grade fever, cough, and shortness of breath at rest. A chest x-ray film demonstrated streaky infiltrates of both the right and the left pulmonary bases. In addition, there was a moderate superior mediastinal widening, without bilateral hilar enlargement. Computerized tomography confirmed the presence of an enlarged right paratracheal lymph gland projecting behind the superior vena cava. Transbronchial lung biopsies (n = 5) were performed and were free of pathologic changes. Analysis of blood showed a mild hypercalcemia (10.7 mg/dl). Cutaneous testing for Mycobacterium tuberculosis and Dermatophagoides pteronyssinus were positive. No mycobacteria were isolated from sputum, urine, or gastric contents. There were no precipitating antibodies against a wide variety of fungi, including the following: Aspergillus fumigatus; Aspergillus niger; Candida albicans; Microsporidium faeni; Penicillium notatum; Penicillium breui compactum; and Thermopelospora polyaspera. Serologic screening revealed a fourfold increase in titer of complement fixation antibodies for Mycoplasma during hospitalization. Consequently, the diagnosis of Mycoplasma-induced pneumonia was made.

After treatment the patient was lost to follow-up during the next two years. On Nov 11, 1984, she came to our outpatient clinic with a history of nonproductive cough. Physical examination revealed no abnormal findings. In particular, there were neither enlarged peripheral lymph nodes nor hepatosplenomegaly. A control chest x-ray film showed an increased widening of the superior mediastinum. Hilar enlargement and the streaky infiltrates, present on the previous chest x-ray film, were absent. Computerized tomography demonstrated a round mass located in the posterior mediastinum (Fig 1A).

On right thoracotomy a firm glandular mass 7.5 by 6 cm was found between the azygos vein and the esophagus. The mass was removed, and histologic examination showed the presence of noncaseating epithelioid granulomas, documenting the diagnosis of sarcoidosis (Fig 1B). Signs of fungal infestation were absent.

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Posterior Mediastinal Sarcoidosis (Rosseel et al)
preparation failed to demonstrate acid-fast rods. Cultures for mycobacteria were negative. The patient was discharged in excellent health, and subsequent examinations revealed no further evolution of sarcoidosis.

CASE 2

A 26-year-old white man was admitted on Feb 10, 1985 with a history of weight loss, low-grade fever, malaise, and nocturnal episodes of perspiration. There was a familial history of sarcoidosis. There had been no occupational exposure to beryllium or silicon. Findings from physical examination were normal. Neither lymphadenopathies nor hepatosplenomegaly was present. The sedimentation rate was 58 mm/hr (Westergren's method), and the fibrinogen level was 438 mg/dl. Cutaneous tests with tuberculin and other common antigens were all negative. Serologic screening was negative. A chest x-ray film showed widening of the superior mediastinum with aortopulmonary window lymph node involvement (Fig 2A).

Computerized tomography confirmed the presence of posterior mediastinal lymph node enlargement extending from below the carina (Fig 2B). Slight enlargement of the right hilar region was present. Bone marrow biopsy indicated normal cell types. Mediastinoscopy failed to provide tissue for histologic examination. On thoracotomy an enlarged glandular structure in the posterior mediastinum was disclosed. Upon inspection and palpation, pulmonary parenchyma was normal. The mass was resected, and histologic examination yielded the presence of noncaseating epithelioid granulomas, documenting sarcoidosis. Again, signs of fungal infestation were absent. A Ziehl-Neelsen preparation failed to demonstrate acid-fast rods. Cultures for mycobacteria were negative. After this procedure the patient remained free of symptoms.

DISCUSSION

Almost all lymph nodes of the body may become affected in sarcoidosis. Intrathoracic lymph node involvement is the most frequent presentation. It occurs in 60 to 90 percent of all patients with sarcoidosis and affects typically the right paratracheal, bilateral hilar, and aortopulmonary window lymph nodes. Subcarinal and anterior mediastinal lymphadenopathy in sarcoidosis has been reported with increasing frequency since the use of lateral tomographic studies and computerized tomographic scans. Nevertheless, sarcoidosis of the posterior mediastinal lymph nodes is rarely observed, according to the literature; Bein et al reported one case of posterior mediastinal lymph node enlargement in a patient with massive mediastinal and bilateral hilar lymph node involvement by sarcoidosis, and Kutty and Varkey

FIGURE 1. A (top), computerized tomogram showing round nonvascular mass in posterior mediastinum. B (bottom), Lymph node architecture is totally replaced by multiple, well-demarcated epithelioid granulomas. Caseation is absent. Some giant cells containing Schaumann's and asteroid bodies are seen (hematoxylin-eosin, original magnification X300).

FIGURE 2. A (top), Chest roentgenogram showing widening of superior mediastinum and aortopulmonary window involvement. B (bottom), Computerized tomogram showing posterior mediastinal lymph node enlargement.
reported another case of posterior mediastinal lymph node enlargement by sarcoidosis in a patient with generalized lymphadenopathy, hepatosplenomegaly, and parenchymal pulmonary infiltrates. Recently, Schabel et al reported a rather high incidence (6/30 patients) presenting with posterior mediastinal lymph node enlargement together with bilateral hilar lymph node enlargement and various extrathoracic manifestations of sarcoidosis.

In contrast to these cases, our two patients presented had posterior mediastinal lymph node enlargement in the absence of bilateral hilar enlargement, pulmonary parenchymal involvement, or extrathoracic manifestations of sarcoidosis. The findings of an enlarging posterior mediastinal density and of a posterior mediastinal lymphadenopathy together with weight loss and fever in our patients were highly suggestive of underlying malignant disease. Since a precise histologic diagnosis was necessary and no other manifestations of sarcoidosis were found, a diagnostic thoracotomy had to be performed in both instances. In addition, direct examination and cultures of the surgical histologic specimens allowed exclusion of mycobacterial or fungal infection.

Sarcoidosis should not be confused with sarcoid-like tissue reactions in carcinoma and other diseases, however, the absence of neoplastic disease in the resected surgical specimens and the uneventful clinical course during follow-up failed to reveal any malignant disease in our patients.

Hodgkin’s and non-Hodgkin’s lymphomas, primary and secondary neoplastic pulmonary disease, neurogenic tumors, esophageal lesions, or cysts of various origin may, in addition to sarcoidosis, also present as a posterior mediastinal density. Measurements of serum angiotensin-converting enzyme levels and the use of the gallium scan and bronchoalveolar lavage may be of additional value in the differential diagnosis of sarcoidosis from carcinoma or sarcoid-like reactions.

In conclusion, we may say that sarcoidosis of the posterior mediastinal lymph nodes without bilateral hilar lymphadenopathy, pulmonary parenchymal involvement, or extrathoracic manifestations remains a difficult diagnosis. In these exceptional cases, no adequate tissue is available for histologic proof of sarcoidosis by conventional methods such as peripheral lymph node biopsy or mediastinoscopy. Therefore, a diagnostic thoracotomy is warranted in order to rule out the presence of malignant disease and to confirm the diagnosis of sarcoidosis.

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