by dividing the pericardium.

This present case, therefore, demonstrates for the first time by pre- and postoperative angiography a possible etiologic role of a pericardial defect in producing coronary artery narrowing with consequent angina pectoris and subsequent myocardial infarction.

The narrowing in the present case was more than mere compression. A lesion which might have consisted of fibrous thickening or atheroma had developed at the site of pressure. It is not surprising therefore, that the postoperative coronary arteriography showed no significant change in the lesion, but the remainder of the coronary arterial tree was free of atherosomatic changes as to suggest that the one occlusive lesion was in fact the result of pressure from the pericardial defect rim. Because grafting procedures would have carried higher operative mortality than implantation of mammary artery pedicles, the latter procedure was elected.

The diagnosis of pericardial defect may be suggested by the characteristic history, positional discomfort (absent here), electrocardiographic abnormalities, and x-ray appearance, described above. More elaborate studies such as vectorcardiography or leftsided diagnostic pneumothorax might have confirmed the etiologic diagnosis in this case. These studies all contribute to the diagnosis and may be included in a complete diagnostic work up. Final confirmation depends on surgical exploration.

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BREWER AND HIMMELWRIGHT

Pleural Effusion Due to Infection with Histoplasma Capsulatum

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A patient with pulmonary histoplasmosis and pleural effusion was treated successfully by lobectomy. Patients with this uncommon manifestation of histoplasmosis recover spontaneously or develop chronic fibroed pleuritis. The demonstration of organisms in biopsy specimens or by culture may be necessary for diagnosis.

During the past decade, infection due to Histoplasma capsulatum has been increasingly recognized. Unusual manifestations of histoplasmosis have included pericarditis, mediastinitis, aortitis, and endocarditis. Pleural effusion, often a difficult diagnostic problem, has been reported infrequently associated with this infection. This case report is illustrative of this uncommon form of histoplasmosis. A brief review of the pertinent literature is presented.

CASE REPORT

A 47-year-old white service station attendant was admitted to Maxwell Air Force Base Hospital for evaluation of pleural effusion. This patient, an Alabama native, had a history of chronic nonproductive cough of uncertain duration, night sweats present for two months, and a recent 5 lb weight loss. There was no history of chest pain or dyspnea. He smoked one package of cigarettes daily and had been drinking excessive amounts of alcohol for several months. No history of soil exposure could be obtained.

On physical examination the patient appeared chronically ill. Positive physical findings were confined to the chest, where absent tactile fremitus, dullness, and decreased breath sounds were noted at the left base posteriorly.

The chest roentgenogram (Fig 1) demonstrated a large pleural effusion in the left hemithorax and a small noncalcified nodule in the superior segment of the left lower lobe. The hematocrit was 47 percent; the leukocyte count was 13,500/mm³ with 82 percent neutrophils, 12 percent lymphocytes, 2 percent eosinophils, and 1 percent monocytes. The following laboratory tests were normal: urinalysis, blood urea nitrogen, fasting blood glucose, serum bilirubin, alkaline phosphatase calcium, phosphorus, and serum glutamic oxalacetic transaminase. An electrocardiogram was normal. The histoplasmin skin test was strongly positive (30 mm induration), while intermediate and second strength purified protein derivative skin tests were negative. Histoplasmin complement fixation (yeast and mycelial phases) was nega-

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PLEURAL EFFUSION DUE TO H. CAPSULATUM

Figure 1. Chest roentgenogram showing large pleural effusion.

Discussion

Pleural effusion is an unusual manifestation of histoplasmosis. Conrad and co-workers,\(^6\) in a thorough discussion of the protean manifestations of the illness fail to mention pleural effusion. Gryboski and associates,\(^7\) in a review of 421 surgical cases, found none with pleural effusion.

Previously reported cases suggest that pleural fluid may be formed in a variety of circumstances. During primary infection, the organism enters the bloodstream with regularity and may become widely disseminated. Prior and co-workers\(^8\) describe an acutely ill woman with extensive bilateral pneumonia accompanied by a serosanguineous pleural effusion. Histoplasma organisms were found throughout the lungs and lymph nodes at autopsy.

Usually, pleural histoplasmosis with effusion is less severe. Schub, Spivey and Baird\(^9\) reported four young men seen at Scott Air Force Base who developed small pleural effusions after brief febrile illnesses. The diagnosis of histoplasmosis was confirmed by a positive pleural fluid culture in one patient and by finding organisms in tissue resected at thoracotomy in two patients. In the fourth patient, a presumptive diagnosis was established by increasing complement fixation titers coupled with conversion of a negative histoplasmin skin test to positive.

Three of the patients underwent thoracotomy and in each of these patients chronic pleuritis was found with thickening of the parietal and visceral

Figure 2. Chest roentgenogram nine months after lobectomy. Postoperative changes are evident but the effusion is no longer present.

The patient's course after operation was uneventful. He was clinically well nine months later, and the chest roentgenogram (Fig 2) was normal except for postoperative changes.
pleuritis. Subpleural granulomas were present within the lung parenchyma. On short term follow-up good result was obtained in all three patients operated on. The fourth patient recovered without treatment.

Two additional patients are reported in whom the effusion cleared without operative intervention. One patient recovered spontaneously; the other patient was well three years after treatment with amphotericin B.

Two patients have been reported from Fitzsimons Army Hospital who had decortications procedures for chronic pleural histoplasmosis with effusion. Stead and associates in an earlier report from the same institution describe a patient with a pleural effusion, a subpleural Histoplasma granuloma, hilar node involvement, and a thin lower lobe peel. Kleger and Fisher described a patient with *H. capsulatum* organisms in the pleurae and pericardium after decortication and partial pericardectomy for effusion. This patient died 13 years later following decortication of the opposite lung and further pericardial resection.

Previous reports contain scant information about the nature of the pleural fluid in this disorder. The fluid in our patient was amber with a total protein of 5.7 g/100 ml. The differential leukocyte count revealed lymphocytosis with increased numbers of eosinophils (9 percent). The pleural fluid was

exposed in three of Schub's and co-workers' four patients. It was noted to be straw colored in two patients and serosanguineous in the other. Two patients had further studies and the fluid from both was an exudate without eosinophilia. Curran and Williams have recently stressed the importance of pleural fluid eosinophilia in differential diagnosis. The apparent rarity of an eosinophilic reaction in tuberculosis is striking.

Elevated histoplasmin complement fixation titers were found in the five previously reported patients in whom these values were measured. Our patient, however, had negative complement fixation on several occasions during the course of his illness. For this reason a positive culture, often difficult to obtain, or demonstration of *H. capsulatum* in pleural fluid or tissue specimen is desirable for definitive diagnosis.

Unless disseminated histoplasmosis or extensive involvement of underlying lung is present, the prognosis is usually favorable. In some patients, the pleural inflammation resolves spontaneously, while in other patients a chronic effusion develops. If operation becomes necessary, a restricting pleural peel is found due to the fibrosing pleuritis which is probably characteristic of the disease. This concept of a chronic fibrosing pleuritis is compatible with the occurrence of fibrosing mediastinitis and chronic constrictive pericarditis occasionally seen with histoplasmosis. When chronic effusion and thickened pleura develop, most patients recover satisfactorily after decortication and excision of parenchymal granulomas.

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Radiologically Evident Functioning Mediastinal Parathyroid Adenoma
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The presence of a large radiologically evident functioning parathyroid adenoma is in itself rare (five isolated cases reported) but its occurrence in an asymptomatic patient has not been previously described. Because of the problems encountered in diagnosis and treatment, this case is presented to emphasize the importance of considering the possibility of a parathyroid tumor in the differential diagnosis of mediastinal lesions, even if symptoms are lacking.

INTRODUCTION

The occurrence of a large radiologically evident functioning mediastinal parathyroid adenoma is rare. Only five isolated cases have been described previously in the literature.1-5 and in each case the patients had symptoms referable to renal calculi and/or classic symptoms of hypercalcemia, suggesting a disorder in calcium metabolism. However, despite its rarity, even in the absence of symptoms referable to a calcium disorder, one must still consider this possibility in the differential diagnosis of a mediastinal mass so that the proper surgical approach may be made. This case is presented to demonstrate the problems encountered in the diagnosis and surgical approach to an asymptomatic patient with radiologically evident functioning mediastinal parathyroid adenoma in whom the possibility of a parathyroid adenoma was overlooked in the differential diagnosis.

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Figure 1. Admission chest x-ray film revealing a large right paratracheal mass.

CASE REPORT

A 50-year-old housewife was admitted to Presbyterian-St. Luke's Hospital, May 28, 1968, for evaluation of hypertension, known for approximately two years. For five months she had noted frontal headaches, unrelated to specific events, increasing in frequency and duration prior to admission. For two

Figure 2. Timed isotope scan shows an abnormality of right innominate bone, initially diagnosed as "Paget's disease". Upon later reevaluation a corrected diagnosis of osteitis fibrosa cystica was made.