authors have attributed this shift in blood flow to an increased alveolar pressure in the fluid-filled lung. However, in our patient, the shift was noted at a time when the lung was not filled with fluid. It appears likely that the perfusion shift we observed was a response to regional alveolar hypoxia, related to the postlavage decrement in ventilation. Sequential ventilation-perfusion scintigraphic studies also were useful in documenting restoration of gas exchange prior to lavage of an additional area.

The functional impairment in the patient described was initially modest, and the improvement noted could be ascribed to spontaneous improvement as well as to the lavages performed. However, the most important aspect of the lavage sequence described was the ease and safety with which such focal lavage was applied to this patient. This suggests its possible extension to other clinical situations in which lavage would be helpful, but in which a temporary decrease in the gas-exchange function of an entire lung would not be acceptable.

REFERENCES


Melioidosis Complicated by Pericarditis*

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A case of acute and recrudescent melioidosis complicated by pericarditis and pericardial effusion is described. The potential for the appearance of future cases in the United States and the necessity for physicians to remain aware of this potential diagnosis are discussed.

In 1912, Whitmore and Krishnaswami first described the pathologic features of a "disease somewhat resembling but really easily distinguishable from glanders." It was recognized from postmortem findings of "the ill-nourished, neglected, wastrels of the town [Rangoon]." Stanton and Fletcher suggested the name "melioidosis" (Greek, "a resemblance to the distemper of asses") and were the first to make the diagnosis antemortem.

The term, melioidosis, encompasses a wide variety of clinical infections ranging from latent to acute systemic disease. The appearance of pericarditis and pericardial effusion with melioidosis is a distinctly unusual complication.

CASE REPORT

A 19-year-old man developed fever and cough in November 1971, three months following his arrival in South Vietnam. A chest x-ray film revealed a right upper lobe infiltrate. Fever persisted during the next six weeks, and the cough became productive of one cup of yellow sputum per day. The right upper lobe infiltrate became more pronounced, accompanied by formation of a 2-cm thin-walled cavity. On Jan 7, 1972, the patient complained of midsternal chest pain unrelated to respiration, and his electrocardiogram demonstrated first-degree atrioventricular block and ST-segment elevation. It was recognized from postmortem findings of "melioidosis" (Greek, "a resemblance to the distemper of asses") and were the first to make the diagnosis antemortem.

The term, melioidosis, encompasses a wide variety of clinical infections ranging from latent to acute systemic disease. The appearance of pericarditis and pericardial effusion with melioidosis is a distinctly unusual complication.
showed no active disease, and therapy was discontinued.

On April 21, 1972, the patient came to Ireland Army Hospital, Fort Knox, Kentucky, 24 hours following the onset of anterior chest pain radiating dorsally to the left of the spine. Pain was aggravated by inspiration, movement, sitting upright, and lying prone and was relieved when the patient was supine. Mild exertional dyspnea was present, but there was no history of other concurrent symptoms.

On physical examination the patient appeared healthy. His temperature was 37.8°C (100°F), his respiratory rate was 26/min, his pulse was 110 beats per minute, and his blood pressure was 120/80 mm Hg, with an S-10-mm Hg pulse paradoxus (normal). AuScultation revealed diminished breath sounds and bronchial breath sounds over the right base. There was no jugular venous distention. No apical impulse was detected, and the left border of cardiac dullness was in the left anterior axillary line. Heart sounds were muffled, and no friction rubs, murmurs, or extra heart sounds were audible. Peripheral pulses were full, and there was no edema. There was minimal right upper quadrant tenderness, but findings from the remainder of the physical examination were within normal limits.

A chest x-ray film demonstrated marked cardiomegaly without other evidence of congestive heart failure. An ECG revealed ST-segment elevation in leads 1, 2, aVF, and V₅ to V₆. The hematocrit reading was 46.9 percent, and the white cell count (WBC) was 12,000/cu mm. The erythrocyte sedimentation rate was 27 mm/hr. Serum electrolyte levels and prothrombin and partial thromboplastin times were normal. Findings from urinalysis revealed a specific gravity of 1.035 and were otherwise normal. Sputum and throat cultures were unremarkable.

Central venous pressure was 24 cm H₂O. Pericardiocentesis yielded 175 ml of straw-colored fluid with a protein level of 6.1 gm/100 ml; a glucose level of 100 mg/100 ml; a WBC of 10,900/cu mm with 86-percent segmented neutrophils, 12-percent lymphocytes, and 2-percent monocytes; and a red blood cell count of 800/cu mm. Gram and acid-fast stains, and cultures for bacteria, mycobacteria, and fungi were negative. In view of the patient's prior therapy for melioidosis with associated pericarditis and pericardial effusion, he began receiving therapy with tetracycline (750 mg orally every six hours) and sulfisoxazole (1 gm orally every eight hours) rectally, his respiration rate was 20/min, and his pulse was 92 beats per minute. The blood pressure was 120/80 mm Hg, and the central venous pressure was 21 cm H₂O. The WBC was 13,400/cu mm. Pericardiocentesis yielded 350 ml of a serosanguineous exudate. All smears and cultures were negative. Following pericardiocentesis, the central venous pressure decreased to 4 to 5 cm H₂O. Tetracycline dosage was increased to 750 mg orally every six hours, and antituberculosis therapy was discontinued.

On the following day the patient remained febrile but appeared improved. The central venous pressure was 4.4 cm H₂O, and the erythrocyte sedimentation rate was 48 mm/hr.

On the sixth hospital day the patient became afebrile and remained so. The hematocrit reading was 42.7 percent, and the WBC was 4,500/cu mm. In view of his clinical improvement and the fall in WBC, chloramphenicol therapy was discontinued. A chest x-ray film revealed bilateral pleural effusions, a right lower lobe infiltrate, and a cardiothoracic ratio of 37.5/28.5.

Although a pericardial "sound" had been heard two days previously, it was not until the seventh hospital day that a three-component pericardial friction rub was audible at the lower left sternal border. The blood pressure was 92/50 mm Hg, but no paradoxical pulse was noted. Sinus arrhythmia with a rate varying from 54 to 72 beats per minute was noted while the patient was at rest. With activity, this converted to regular sinus rhythm with a rate of 88 to 100 beats per minute. An ECG revealed less prominent ST-segment elevation with T-wave inversion in leads 2.

Two days later, the patient's vital signs were normal. A chest x-ray film revealed a normal cardiac shadow and clearing of the infiltrate and effusions. The findings from physical examination were normal, except for sinus arrhythmia and a soft pericardial friction rub.

On the 11th hospital day the friction rub was no longer audible. A cardiac scan was normal. Two days later, an ECG demonstrated T-wave inversion in leads 2, 3, aVF, and V₄ to V₆. A chest x-ray film revealed no active disease.

By May 8 the electrocardiographic changes had cleared in the precordial leads. The results of hematologic and chemical tests were completely within normal limits. Serologic tests for cytomegalovirus; herpes simplex; influenza types A, B (Hong Kong), B (Lee), and B (Massachusetts); adenovirus; and mycoplasma were all negative. Convalescent serologic titers for melioidosis were 1:512 by complement fixation and 1:1,280 by hemagglutination. On May 18, an ECG revealed only nonspecific ST-T wave changes. The patient was discharged to continue tetracycline and sulfisoxazole therapy for a total of eight weeks; however, he was lost to follow-up.

**DISCUSSION**

Melioidosis is a disease of protean manifestations caused by *P. pseudomallei*, a natural soil saprophyte endemic to tropical and subtropical areas. The disease was rare in the United States prior to the return of large numbers of armed forces' personnel from southeast Asia. Serologic studies of similar military personnel who have no history of the disease indicate that subclinical infection is common. Melioidosis may recur months to years after initial exposure. Recent returnees from Vietnam are, therefore, a potential reservoir for the disease, and cases may appear in this country during the next few years.
The patient described here represents a typical case of pulmonary melioidosis with the rare association of pericarditis and pericardial effusion. The patient first presented with persistent high fever, productive cough, and an upper lobe infiltrate which progressed to ascites formation. Electrocardiographic signs of pericarditis and roentgenographic signs compatible with pericardial effusion developed during the initial illness. These resolved following therapy. With recrudescence the patient presented with chest pain. The physical examination, x-ray films, and ECG revealed evidence of pericarditis. The diagnosis was confirmed by pericardiocentesis. The transient lower lobe infiltrate and bilateral pleural effusions developed afterwards, and these resolved with therapy.

The spectrum of clinically evident melioidosis ranges from a chronic indolent process to an acute septicemic form with a high mortality. Pulmonary infection in which the pathologic findings are confined to the lungs may clinically be confused with pulmonary tuberculosis. Myocardial melioidosis has been reported as a manifestation of the systemic form of this disease, and one case presented as an acute myocardial infarction. Myocardial involvement cannot be excluded in the present case.

It seems likely from the course of the patient’s illness that the pericarditis and pericardial effusion were secondary to infection with S. pseudomallei, even though the organism was never recovered from the pericardial fluid. These complications must, therefore, be added to the manifestations of melioidosis, and the physician should be alerted to this diagnosis in patients with signs and symptoms of pericarditis who have a history of prior residence in southeast Asia.

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Tumor Emboli Presenting as Pulmonary Hypertension

A Diagnostic Dilemma

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A case of recurrent tumor emboli secondary to choriocarcinoma is described. The patient presented with obvious pulmonary hypertension and was diagnosed and treated as a case of multiple pulmonary embolism. Information which suggested the possibility of tumor emboli was indeed present but recognized only retrospectively.

Pulmonary hypertension accompanies a wide variety of disease processes, including diffuse pulmonary parenchymal disease, congenital heart disease, left-sided congestive cardiac failure, and occlusive pulmonary vascular disease. When it is well developed, it may dominate the clinical picture, and it requires a thorough diagnostic evaluation. Given the setting of a young woman in the postpartum period, the diagnostic possibility of pulmonary emboli is uppermost, and pulmonary angiography is probably the single best procedure to employ in the diagnosis. The following case report illustrates how treacherous even the angiogram can be if the history is not paid meticulous attention.

CASE REPORT

A 29-year-old woman was admitted to the University of Michigan Medical Center, Ann Arbor, on Sept 10, 1973, for the evaluation of chest pain, pulmonary infiltrates, and progressive dyspnea. She had had these symptoms for the preceding four months and has been treated elsewhere for pneumonia with antibiotic therapy without success. Past history revealed that ten months previously, following an

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