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Concomitant Streptococcal Endocarditis and Tuberculous Pleuro-Pericardial Effusion

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NOTES AND COMMENTS

Concomitant Streptococcal Endocarditis and Tuberculous Pleuro-Pericardial Effusion

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A young adult heroin addict with *Streptococcus viridans* endocarditis developed the unusual complication of massive pericardial and pleural effusion which was shown to be due to concomitant tuberculosis. In addition, his course was complicated by a hemiplegia secondary to ruptured cerebral mycotic aneurysm treated neurosurgically with complete recovery. Since these multiple problems were resolved by specific diagnosis and application of medical and surgical techniques now available, the patient is made the subject of this report.

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Case Report

A 23-year old Negro male was admitted to the hospital on September 11, 1971 with complaints of cough, dyspnea, yellow sputum, palpitations, night sweats and anorexia. He was a known narcotic addict, who had been using intravenous heroin for two years prior to admission. Physical examination revealed a low grade fever, poor oral hygiene, apical systolic thrill, apical Grade IV/VI holosystolic murmur, hepatomegaly and subungual splinter hemorrhages.

On admission, his hemoglobin was 10.4 grams% and white blood cell count was 13,500 cells/cmm. Urine analysis, serum electrolytes, creatinine, serum enzymes, proteins blood urea nitrogen, blood sugar and test for Australia antigen gave normal results. Reticulocyte count was 2.7%, and hemoglobin electrophoresis revealed 60% hemoglobin A and 40% hemoglobin S. Purified protein derivative (PPD)-S and B skin tests were negative. Admission chest x-ray was unremarkable (Figure 1). Serum IgG was 2005 mg%. Six admission blood cultures grew alpha hemolytic streptococci sensitive to all the antibiotics tested. The patient was given intramuscular Clindamycin 450 mg every 8 hours with good response.

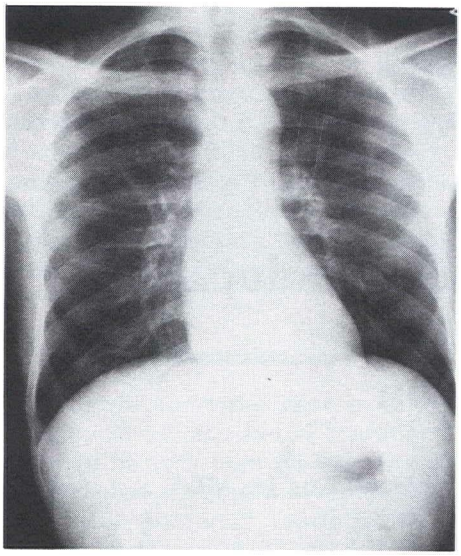

Figure 1
Normal chest film on admission.

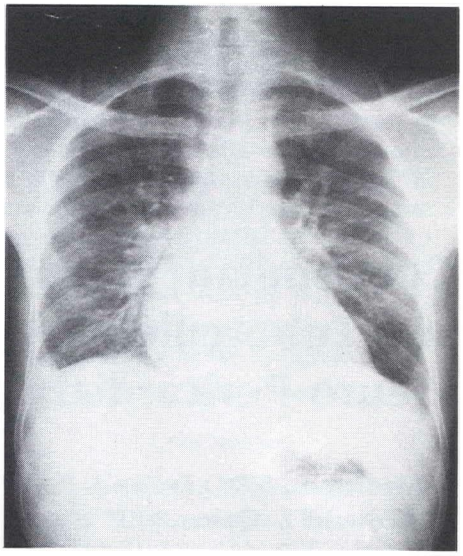

Figure 2
Film of chest demonstrating enlarged heart, plethoric lung fields and right pleural effusion.

On September 19th, patient developed anterior substernal chest pain and progressive dyspnea. Heart was found to be enlarged and a pericardial rub was heard. An electrocardiogram was consistent with pericarditis. On September 21st, a pericardiocentesis was performed and 100 cc of serosanguinous fluid was aspirated with immediate improvement in his clinical condition. The pericardial fluid contained 70 mg of glucose/100 ml, 4.9 gms of proteins/100 ml with 3500 cells, mostly polymorphonuclear leucocytes. A left sided pleural effusion was also detected and tapped. The pleural fluid yielded positive smears and cultures for mycobacterium tuberculosis (human type). Pleural biopsy revealed granuloma with acid fast bacilli. Pericardial fluid was negative for acid fast bacilli on smear and culture. Antituberculous therapy was initiated with isoniazid, streptomycin, and ethambutol. Management also included digitalization and diuretics.

On October 11th, the patient's clinical condition again deteriorated. He was found comatose and convulsing with right hemiplegia and complete heart block. A temporary transvenous pacemaker was inserted and tracheostomy was performed. Based on arteriogram, diagnosis was made of left temporal hematoma presumably secondary to ruptured cerebral mycotic aneurysm. A left temporal craniotomy and evacuation of the clot resulted in gradual recovery. Follow-up brain scan was negative.

On November 5th, chest x-ray was reported normal with normal heart size (Figure 3). His PPD-S and B continued to be negative. The patient was discharged on January 27, 1972 with residual right hemiplegia. Follow-up in the outpatient clinic in the next ten months revealed recovery was complete, except for a residual Grade IV/VI apical systolic murmur, and apical S_3 gallop and right inferior quadrantanopsia.

Discussion

Small pericardial effusions have been reported to occur in some cases of subacute bacterial endocarditis, but massive nonsanguineous pericardial effu-

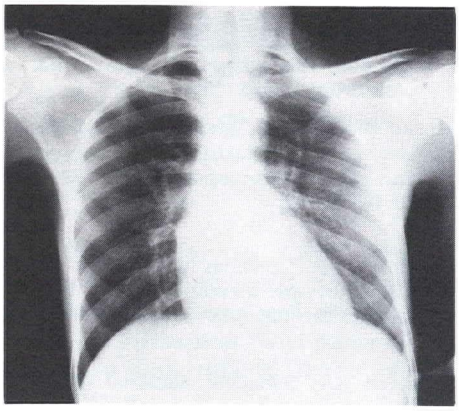

Figure 3
Normal heart size, as shown by chest film.

sion is a rare occurence [1-5]. Tykot and Relkin reported one patient with *Streptococcus viridans* endocarditis who developed massive pericardial effusion of unknown cause with cardiac tamponade.This patient required three pericardiocenteses before he recovered.[6] Large pericardial effusion can also be a complication of a myocardial mycotic aneurysm or mycotic aneurysm involving the coronary cusps.[7-8] In our patient, we demonstrated the probable tuberculous etiology of the pericardial effusion by isolation of tubercle bacilli from the pleural fluid. However, the smears and cultures of the pericardial fluid were negative. This association of *Streptococcus viridans* endocarditis and tuberculous pleuro-pericardial effusion has not been previously reported in English literature.

Cerebral mycotic aneurysms are usually asymptomatic until they rupture and prognosis in the patient with ruptured mycotic aneurysm is usually poor. A high index of suspicion, prompt diagnosis with repeated neurological examination, angiography and early surgical intervention leading to complete recovery have been reported in a number of cases in recent years. [9-19]

Summary

A case is described of Streptococcus viridans endocarditis with an unusual complication of massive tuberculous pleuro-pericardial effusion. The patient's hospital course was also complicated by a hemiplegia secondary to ruptured cerebral mycotic aneurysm. This was treated by left temporal craniotomy and clot evacuation, resulting in excellent recovery.

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