Tuberculous pericardial disease

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THE PERICARDIUM may be involved in a wide spectrum of diseases. A diagnosis of pericarditis is often incomplete until the aetiological cause is established, although this may not be always possible. Some of the important causes of pericarditis are:- rheumatic, bacterial (including tuberculosis), viral, malignant, collagen diseases, metabolic diseases and myocardial infarction.

In Singapore to-day, tuberculosis is still one of the most important causes of pericarditis, although the incidence of tuberculosis is decreasing as a whole.

Tuberculous pericarditis can present in a wide variety of ways. We report here five cases seen at Tan Tock Seng Hospital, Singapore, between the years 1968 to 1969, illustrating the wide clinical spectrum of T.B. pericardial disease and the problems encountered in its diagnosis and management.

CASE REPORTS

Case No. 1.

C.M.C., a 68-year-old Chinese male, was seen at T.T.S.H. in March 1968, for cough and breathlessness on exertion for two weeks. On physical examination, a right pleural effusion was found. There were no signs of heart failure or cardiac tamponade, but a distinct pericardial rub was heard over a wide area of the praecordium. A chest X-ray (Fig. 1) confirmed

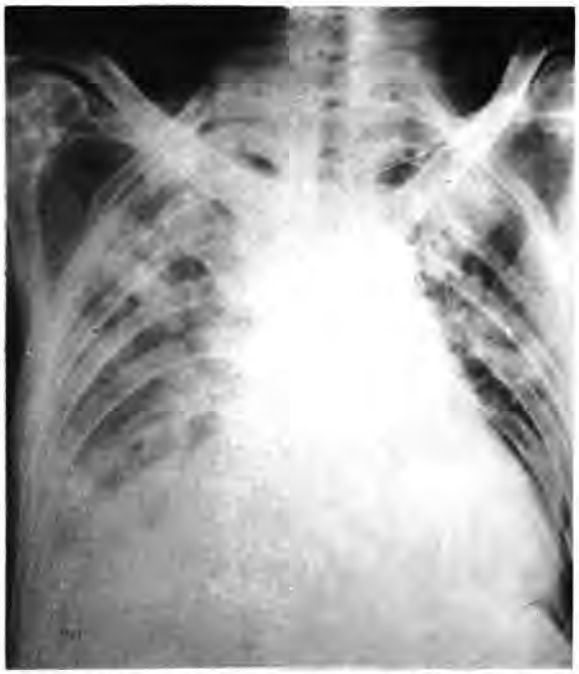

Fig. 1. Right pleural effusion, extensive bilateral pulmonary tuberculosis and pericardial effusion.

the right pleural effusion and showed extensive tuberculous lesions over both lungs; and an enlarged heart probably because of a pericardial effusion. Sputum for Acid Fast Bacilli (A.F.B.) was positive on direct microscopy and cultures. Right pleural aspiration yielded 500 mls. of hemorrhagic fluid.

He was treated with injection Streptomycin and Tabs. Paraaminosalicylic Acid and Isoniazid (Tabs. PAS. & INH.) and made good progress. After four months in hospital, he was discharged, and his chest X-ray then showed marked clearing of the pulmonary lesions, together with a normal-sized heart. When reviewed recently as an outpatient, he had no abnormal physical findings.

Case No. 2.

S.F., a 56-year-old Indian male, was first admitted to a psychiatric hospital in 1963 for schizophrenia. He was found to have minimal pulmonary tuberculosis and received chemotherapy consisting of Streptomycin, PAS. & INH. On completing the course of Streptomycin, PAS. & INH. were continued for two years. He was re-admitted in May 1969, for a relapse of schizophrenia and was found to be in heart failure. On physical examination, the patient was dyspnoeic and ill with a blood pressure of 80/50 mm. Hg. and a pulse rate of 120/min. The jugular venous pressure was grossly elevated. The apex beat was not palpable and the heart sounds on auscultation were normal. Bilateral pleural effusions were found, and the liver was five cm. enlarged with no splenomegaly and no ascites. Moderate oedema of both legs were present. A chest X-ray showed a very large heart consistent with a pericardial effusion, together with bilateral pleural effusions and tuberculosis lesions over the upper zone of both lungs.

He was then transferred in July 1969 to Tan Tock Seng Hospital where his sputum was found to be positive for A.F.B. on direct microscopy and culture. A pericardial tap done via the xiphisternal route yielded 375 mls. of hemorrhagic fluid. Air injected into the pericardial cavity after aspiration revealed that the parietal pericardium was thickened (Fig. 2). The pericardial fluid was negative for A.F.B. on direct microscopy but was positive on culture. An E.C.G. done showed non-specific ST segment depression over the praecordial leads. He was treated with digitalis and diuretics, and a second course of anti-tuberculous therapy, consisting of Streptomycin, PAS. & INH. together with prednisolone, was given. While in the ward, pericardial aspiration was repeated twice because of cardiac tamponade.

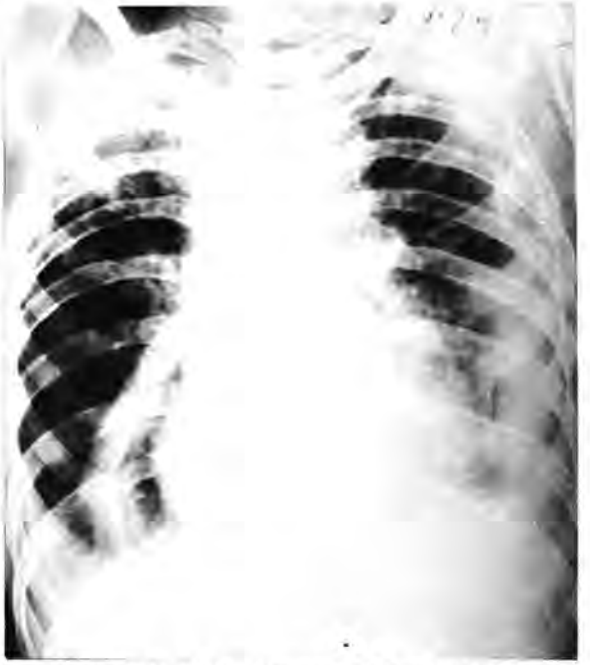

Fig. 2. Injection of air after pericardial aspiration demonstrating thickened parietal pericardium.

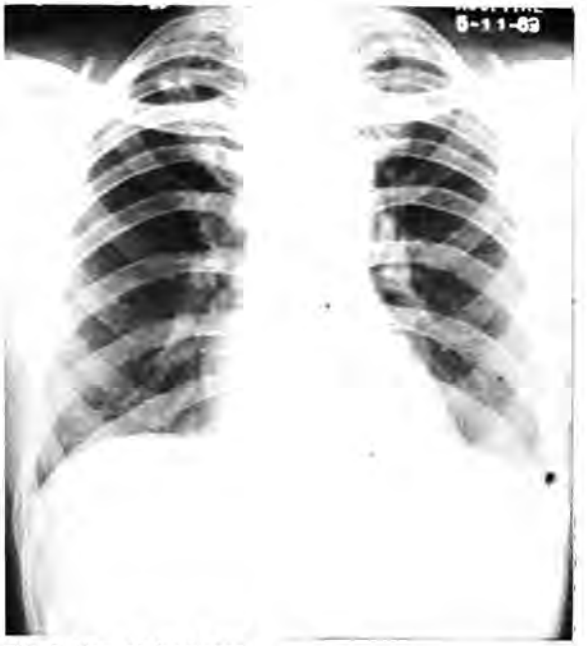

Fig. 3. Normal-sized heart.

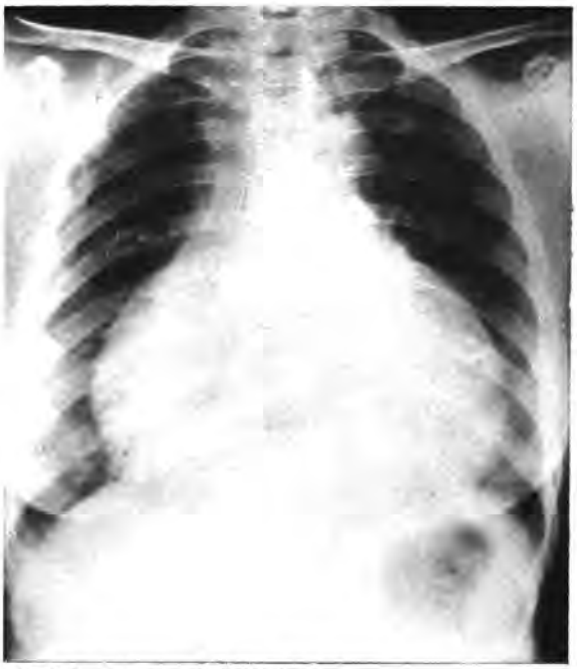

Fig. 4. Large pericardial effusion.

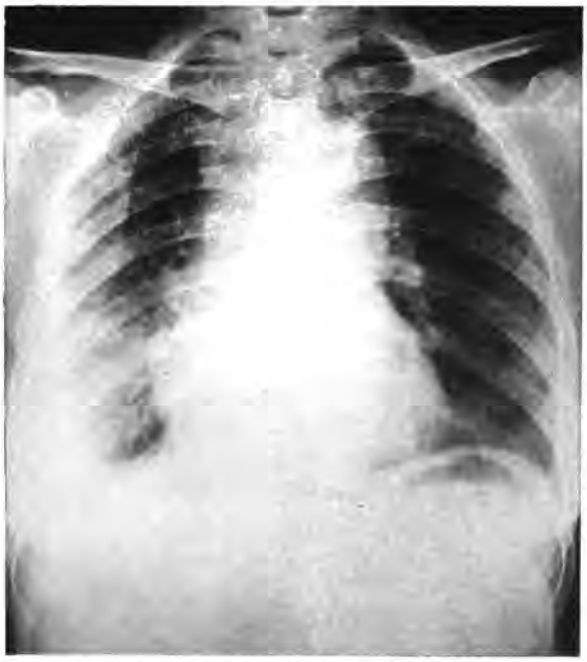

Fig. 5. Normal-sized heart and right pleural effusion.

He improved remarkably and the signs of cardiac tamponade gradually disappeared. In November 1969, there were no abnormal physical findings and his chest X-ray showed a normal-sized heart (Fig. 3).

Case No. 3.

C.L., a 68-year-old Chinese female, was admitted to another hospital in April 1969, for breathlessness on exertion and swelling of legs for three months. She was found to have a large pericardial effusion (Fig. 4) and a diagnostic pericardial tap yielded hemorrhagic fluid which grew A.F.B. on culture. She was treated for heart failure and transferred to Tan Tock Seng Hospital where anti-tuberculous therapy (Streptomycin, PAS. & INH.) together with Prednisolone was started.

Repeated chest X-rays showed her cardiac silhouette growing progressively smaller. In September 1969, a right pleural effusion developed (Fig 5) with signs of cardiac tamponade. The jugular venous pressure was elevated to the angle of the jaw and showed a steep 'Y' descent consistent with constrictive heart disease. Blood pressure and radial pulse were normal; pulsus paradoxus was not present. The apex beat was easily felt in the 5th left intercostal space within the left mid-clavicular line. On auscultation, the heart sounds were normal. The liver was six cm. enlarged with a two cm. enlarged spleen; moderate ascites and ankle oedema were present. Sputum was negative for A.F.B.; and an electrocardiogram showed inverted 'T' waves in leads III & AVF.

A total pericardiectomy was done in November 1969. The lateral portion of the right pleura was found to be thickened and the heart encased by thickened pericardium. Histology revealed a thickened, fibrotic pericardium, but no A.F.B. were seen. She made good progress and when reviewed recently showed no signs of cardiac tamponade.

Case No. 4.

L.S.N., a 34-year-old Chinese female, was first admitted to Tan Tock Seng Hospital in July 1968 for breathlessness. She had been unwell for ten years prior to this, having been admitted to Outram Road General Hospital several times for heart failure. There was no past history of tuberculosis or rheumatic fever. On physical examination, her jugular venous pressure was elevated to the angle of the jaw, with prominent 'V' waves. The heart was in atrial fibrillation at 100/min. with a blood pressure of 100/70. The apex beat was heaving in type and felt at the 6th

left intercostal space at the anterior axillary line. A left parasternal heave denoting right ventricular hypertrophy was present. On auscultation, a pansystolic murmur, grade III, was heard at the mitral area radiating to the axilla, together with a short mid-diastolic flow murmur. A pansystolic murmur was also present at the tricuspid area, and the liver was seven cm. enlarged and pulsatile. The spleen was two cm. enlarged and moderate ascites was present. A chest X-ray (Fig. 6) showed an enormous cardiac shadow occupying almost the entire chest and an electrocardiogram showed atrial fibrillation with tall 'R' waves in V5 and V6, suggesting left ventricular hypertrophy. A diagnosis of mitral and tricuspid incompetence with a possible pericardial effusion was made. She was put on a heart failure regime and discharged in August, 1968, but subsequently had two further admissions for heart failure. On her fourth admission in July 1969, a diagnostic pericardial aspiration was attempted via the xiphisternal route and 800 mls. of straw-coloured fluid were obtained. Air injected after aspiration revealed an enlarged hair-line thin pericardial sac (Fig. 7). The fluid was positive for A.F.B. on direct microscopy, but negative on culture. Pericardial aspiration was later repeated twice and a total of 2,000 ml. of fluid withdrawn. Repeat chest X-rays still showed a very large heart due to the mitral and tricuspid incompetence. She was started on Streptomycin, PAS. & INH, and was relatively well on discharge in December 1969.

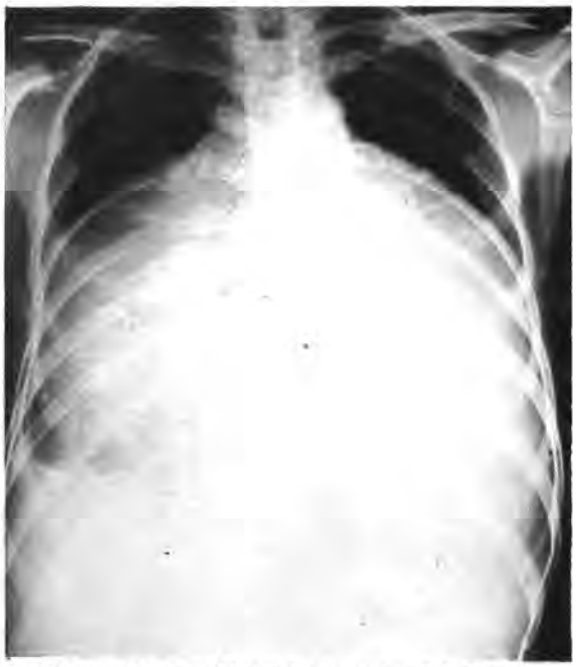

Fig. 6. Grossly enlarged heart due to pericardial effusion.

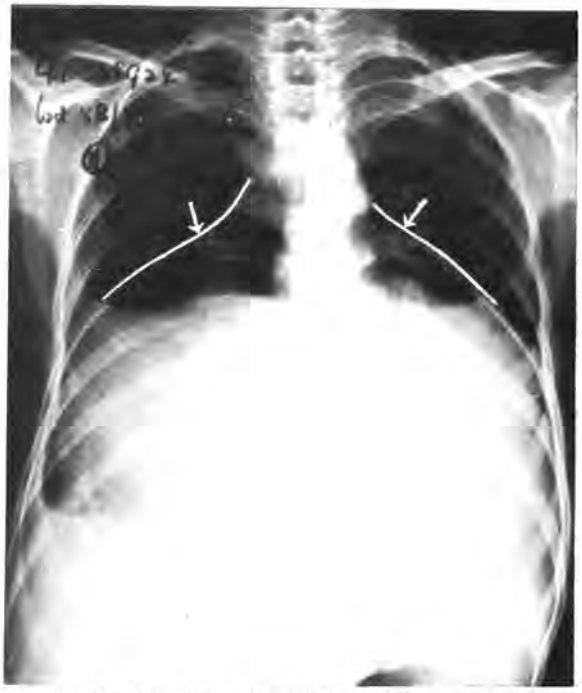

Fig. 7. Hairline thin, pericardial sac (inked and arrowed) demonstrated after pericardiocentesis and injection of air.

Case No. 5.

C.N.N., a 15-year-old Chinese girl, was first admitted to T.T.S.H. in December 1967 for exertional dyspnoea with swelling of the legs for one year. There was no previous history of pulmonary tuberculosis. Physical examination & chest X-ray revealed a pleural effusion in the right chest (Fig. 8). The pulse and blood pressure were normal, and the apex beat was in the 5th left intercostal space within the mid-clavicular line. A third heart sound was heard at the mitral area. The liver was seven cm. and the spleen two cm. enlarged, and there was ascites and leg oedema. A right pleural aspiration showed that the effusion was a transudate, and sputum and pleural fluid were both negative for A.F.B. on direct microscopy and culture. A tuberculin test of one T.U. was two m.m. The hemoglobin, total white and sedimentation rate were normal. Blood for L.E. cells and rheumatoid factor were repeatedly negative; and viral studies of the blood and stools were also negative. Liver function

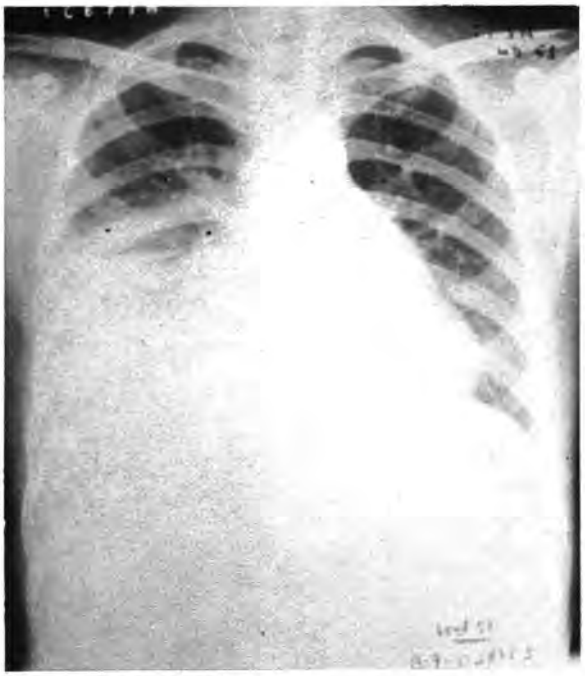

Fig. 8. Right pleural effusion.

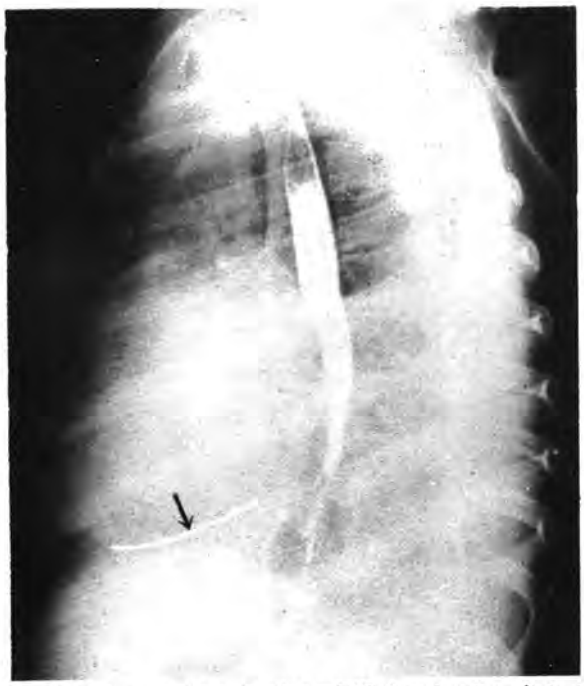

Fig. 9. Barium swallow showing calcified pericardium (inked and arrowed).

tests were all normal, and a liver biopsy revealed fatty change only.

She was discharged in February 1968, with little improvement, and was re-admitted in March 1968, with more severe breathlessness and leg oedema. This time, the jugular veins were markedly distended and elevated to the angle of the jaw. Pulsus paradoxus was not present. Further chest X-ray and a barium swallow showed a distinct calcified pericardium (Fig. 9), and an electrocardiogram showed widespread 'T' wave inversion with bifid 'P' waves in leads II & III.

Subtotal pericardiectomy was done in June 1968. At operation, adhesions between the pericardium and heart with calcifications at the atrio-ventricular grooves and between the interventricular grooves were found. Histology revealed collagenous fibrous connective tissue consistent with a much thickened fibrotic pericardium. Post-operatively, she did very well, as the signs of cardiac tamponade gradually diminished and a chest X-ray (Fig. 10) in March 1969, showed a normal-sized heart with no pleural effusion. When last seen in October 1969, she was perfectly well with no abnormal findings.

DISCUSSION

Schrire, (1967) in one of the largest series of pericardial diseases, reported on a total of 382 patients seen in the Groote Schuur Hospital in Cape Town, South Africa. Forty per cent of these were definitely tuberculous, 40 per cent were most likely

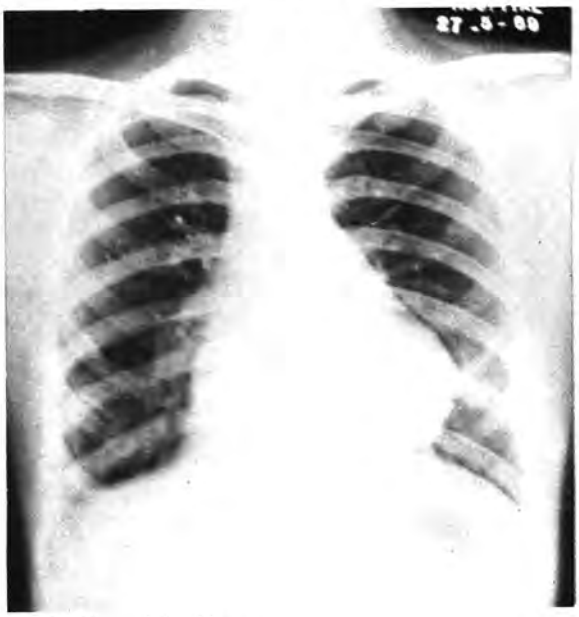

Fig. 10. Normal-sized heart.

tuberculous, whilst 12 per cent were idiopathic, and six per cent pyogenic, with two per cent miscellaneous.

The criteria for making the diagnosis of tuberculous pericarditis in Schrire's series were based on the presence of any of the three following conditions, and their respective incidence in his series were:

(i) A.F.B. detected in the pericardial fluid ... 18%
(ii) Histological finding of T.B. in the pericardium ... 44%
(iii) Organ involvement (Lungs, glands etc.) ... 38%

In three of our cases (Cases Nos. 2, 3, 4), A.F.B. were found in the pericardial fluid. In Case No. 1, A.F.B. were found in the sputum. Case No. 5 presented some problem in diagnosis, as histology of her pericardium showed fibrous connective tissue only, with no definite evidence of tuberculosis. This patient presented with constrictive pericarditis, and as all other causes of this disease were excluded, it was most likely tuberculosis in origin.

A histological finding of tuberculosis was found in only 44% of the definite tuberculosis pericarditis in Schrire's series, and he commented that antituberculous treatment can prevent histological evidence of T.B. from developing. This fact is further supported by Case No. 3 where the pericardium obtained at operation after seven months of antituberculous treatment showed only fibrous connective tissue.

T.B. pericarditis can present as three distinct syndromes – dry (fibrinous) pericarditis, pericardial effusion and constrictive pericarditis. Dry (fibrinous) T.B. pericarditis presenting with chest pain, pericardial rub, and a normal-sized heart is uncommon, and was seen in only 12 of the 382 cases in Schrire's series.

Four of our five patients presented with pericardial effusion. Case No. 4 was a great diagnostic problem because, in addition to the pericardial effusion, she also had mitral and tricuspid incompetence, most likely of rheumatic origin; hence pericardial effusion was confirmed only after a diagnostic pericardial aspiration. Sometimes, the differentiation between pericardial effusion and cardiomyopathy may be impossible to resolve and in such cases a right heart catheterization, with or without the injection of radio-opaque dye or carbon dioxide, could be of immense value. Where the diagnosis is in doubt, pericardial aspiration could be dangerous because of the possibility of piercing the cardiac muscle, but the development of the pericardial electrode needle (Bishop et alia, 1965) has reduced considerably the danger of aspiration in such cases.

The patient who presents with T.B. constrictive pericarditis de novo sometimes poses a special diagnostic problem. Although the classical signs of constrictive heart disease are well known to all, the diagnosis is sometimes not made because the jugular venous pressure may be so grossly elevated that the venous pulsations are missed. Such patients present with pleural effusion, hepatomegaly and ascites and hence liver cirrhosis is often mistakenly diagnosed. Other forms of heart disease, e.g. Amyloidosis, may mimic constrictive pericarditis but the presence of pericardial calcification as in Case No. 5, is extremely helpful in the diagnosis.

In the management of pericardial effusion, it is important to remember that the effusion may resolve completely without giving rise to constriction as exemplified by Case No. 1 and 2. In the patients who develop constrictive disease following pericardial effusion, surgery can safely be deferred up to six months, because the constrictive phase with tamponade may pass away completely with antituberculous drugs alone (Schrire 1967). However, if constriction persists, as in Case No. 3, pericardiectomy is indicated.

SUMMARY

Five cases of tuberculous pericardial disease as seen in T.T.S.H. are described. The various clinical syndromes encountered in T.B. pericardial disease, their pathogenesis, prognosis and management are discussed.

ACKNOWLEDGEMENTS

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