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Mycobacterium kansasii pericarditis

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Mycobacterium kansasii was thought to exist primarily as a saprophyte until two cases of clinical infection were reported by Buhler and Pollak in 1953.[1] Although *M kansasii* is known to cause pulmonary and extrapulmonary infections, we are unable to find a previous reported case of pericarditis caused by this organism. We here describe a patient with pericarditis, pericardial effusion, and mediastinal lymphadenitis caused by *M kansasii*.

Case report

A 75 year old black man was admitted to hospital with a one year history of intermittent cough, mucoid sputum, fever, chills, and night sweats, together with gradual weight loss and effort related fatigue. He was found to have a pericardial effusion and anaemia. After 14 days no cause could be determined and he was referred to St Elizabeth Hospital Medical Center for further evaluation and treatment. He had no previous history of exposure to tuberculosis or of haemoptysis and he had never smoked.

On examination his temperature was 38·3°C, pulse rate 100/min, blood pressure 118/66 mm Hg, and respiratory rate 20/min. The patient was alert and not in any acute distress. There was a loud biphasic pericardial friction rub, which seemed to intensify with deep respiration. There were no gallops or murmurs. There was no pulsus paradoxus and the jugular venous pressure was normal. Breath sounds were decreased at the left lower base. The liver was slightly enlarged. Investigations showed: haemoglobin concentration 7·1 g/100 dl; packed cell volume 0·24; white blood cell count 7·3 × 10^9/l (83% polymorphonuclear leucocytes, 14% lymphocytes, 3% monocytes. The blood urea nitrogen concentration was 17 mg/dl. Arterial oxygen and carbon dioxide tensions during the breathing of room air were: Pa_{O_2} 60 mm Hg (8 kPa), Pa_{CO_2} 29 mm Hg (3·87 kPa); the pH was 7·56. The serum albumin concentration was 29 g/l. The erythrocyte sedimentation rate was 39 mm in one hour. Urine analysis showed: protein 0·3 g/l, 6–10 white blood cells per high power field, and a few hyaline and many granular casts. The chest radiograph showed a moderately enlarged heart and blunting of both costophrenic angles. Two dimensional echocardiography showed pericardial fluid with normal left ventricular function. Sputum was examined for acid fast bacilli and cultures of *Mycobacterium tuberculosis* were initiated. Three blood cultures yielded no growth.

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On the third hospital day the electrocardiogram (ECG) showed atrial fibrillation with a fast ventricular response. The patient was treated with digoxin and verapamil. Bone marrow aspiration and biopsy showed no evidence of tumour or granulomatous processes. On the seventh hospital day the patient underwent pericardectomy and mediastinal lymph node biopsy. Histological examination of the lymph node and pericardial tissue showed features of chronic granulomatous inflammation. Pericardial tissue and fluid cultures yielded no growth. On the eighth hospital day daily treatment with isoniazid 300 mg orally, rifampicin 600 mg orally, and streptomycin 1 g intramuscularly was started. The ECG now showed normal sinus rhythm and one the 13th hospital day the friction rub was no longer audible. Streptomycin was later discontinued because the patient complained of decrease in hearing and ethambutol 1·2 g daily was added. The patient was discharged after 36 days in hospital.

Bone marrow cultures yielded no growth. Sputum cultures did not yield fungus or mycobacterium. Material from the mediastinl node grew *M kansasii* susceptible to rifampicin, ethambutol, and ethionamide but partially resistant to streptomycin, aminosalicylic acid, and isoniazid. Culture of pericardial tissue and fluid failed to yield the mycobacterium. Ethionamide 250 mg four times a day and pyridoxine 50 mg twice daily were added to the regimen of 300 mg isoniazid, 600 mg rifampicin, and 1000 mg ethambutol a day. It is intended that this treatment will be continued for two years. The patient continued to improve clinically after discharge and there has been no reaccumulation of pericardial fluid.

Discussion

M kansasii is known to cause pulmonary disease, bone and joint infections, soft tissue and tendon infections, and disseminated infections.[2] To the best of our knowledge, however, no cases have been reported of pericarditis caused by *M kansasii*.

M tuberculosis is the cause of 7–10% of all cases of pericarditis and the mortality rate of tuberculous pericarditis is as high as 40% despite advances in treatment—in contrast to a mortality of only 3% for patients with pulmonary tuberculosis in the United States.[3] The mortality is related to the cardiovascular complications. Rooney and his associates support the use of corticosteroids in tuberculous pericarditis to prevent the constrictive complications of the inflammatory exudate.[3] The steroids used in combination with antituberculosis drugs appear to limit the amount of exudate while at the same time causing a more rapid resolution. If, however, there is persistent enlarge-

ment of the heart or progressive heart failure, pericardectomy should be performed. In our patient pericardectomy and biopsy were performed to establish the diagnosis. Tuberculous pericarditis was suspected and the patient was started on antituberculosis treatment. The histological examination of biopsied tissue cannot differentiate infection caused by *M tuberculosis* from that caused by other mycobacteria.[4 5]

Most studies agree that *M kansasii* infection is best treated with combination treatment.[6 7] Wolinsky found that the combination of isoniazid and streptomycin was more effective than either alone.[8] In the treatment of pulmonary infections caused by *M kansasii,* Harris and his associates found bacteriological conversion in 85% of patients treated with isoniazid, ethambutol, and streptomycin.[9] They recommended an alternative regimen including rifampicin if cultures remain positive after four to six months of treatment. On the other hand, Banks and associates reported 100% sputum conversion with regimens containing rifampicin in the treatment of *M kansasii* pulmonary infection.[10] In our patient rifampicin was used because of the excellent results with rifampicin containing regimens. Ethambutol and ethionamide were used because of the organism's in vitro susceptibility, and isoniazid because of its excellent mycobactericidal activity despite partial resistance in vitro.

M kansasii infections will usually respond well to proper combination drug treatment based on in vitro susceptibility testing. Regimens containing ethionamide or viomycin have been useful in patients who fail to respond to standard drug treatment.[11] The prognosis of *M kansasii* pericarditis is not known because of lack of clinical experience.

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