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Cardiac tamponade due to actinomycosis

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Actinomycosis is an uncommon infection produced by anaerobic Gram positive bacilli normally resident in the mouth. It produces a progressive granulomatous disease with focal or systemic manifestations.[1] The heart is affected in less than 2% of cases,[2] usually by direct spread from a primary thoracic source.[3] Primary pericardial actinomycosis has been reported only once to our knowledge.[4] We present a case of primary actinomycotic purulent pericarditis causing cardiac tamponade and death despite pericardiocentesis, antibiotics, and pericardiectomy.

Case report

A 56 year old white man presented with a six month history of progressive dyspnoea, fatigue, and weight loss. His exercise tolerance had diminished over the previous 10 days, although he had not experienced orthopnoea or nocturnal dyspnoea. He gave a two year history of angina and moderate hypertension.

On examination he was unwell, dyspnoeic at rest, and peripherally cyanosed. He had carious teeth and pyrexia (37·5°C). He had a sinus tachycardia (100 beats min); the blood pressure was 110/70 mm Hg and the jugular venous pressure was raised. There was a gallop rhythm (S_3) and tender hepatomegaly but the lung fields were clear and there was no peripheral oedema.

Laboratory investigations showed: haemoglobin concentration 13·6 g/dl; packed cell volume 0·42; leucocyte count 15·2 × 10^9/l (83% segmented neutrophils); erythrocyte sedimentation rate 45 mm in the first hour; total serum protein concentration 67 g/l; globulin 38 g/l; glucose 12·5 mmol/l (225·2 mg/100 ml); creatinine 157 μmol/l (1·78 mg/100 ml); urea 14·5 mmol/l (87·3 mg/100 ml); total bilirubin 37 μmol/l (2·2 mg/100 ml); aspartate transaminase 59 IU/l; alkaline phosphatase 107 IU/l; electrolytes normal; blood cultures negative. The electrocardiogram showed sinus rhythm, ventricular premature beats, and non-specific T wave abnormalities anterolaterally. The chest radiograph showed cardiomegaly with a widened mediastinum.

Within 48 hours the patient developed atrial fibrillation (140 beats min), arterial hypotension (85/70 mm Hg), a paradoxical pulse, a grossly raised jugular venous pressure, diminished heart sounds, and oliguria and uraemia (20·9 mmol/l (125·9 mg/100 ml)). Echocardiography indicated an anterior pericardial effusion, vigorous ventricular contractility, and paradoxical septal movement (fig 1). Cardiac catheterisation confirmed cardiac tamponade and pericardiocentesis was performed, yielding 150 ml of viscous, pale yellow pus. The systolic blood pressure rose to 95 mm Hg and the mean right atrial pressure fell from 12 to 8 mm Hg, but there was little clinical improvement. Microscopic examination of the aspirate showed many pus cells and Gram positive bacilli but sulphur granules were not identified. Acid-alcohol fast bacilli were not seen.

Treatment consisted of intravenous benzylpenicillin (12 MU daily), cloxacillin (3 g six hourly), gentamicin (80 mg daily), and antituberculous drugs. During the ensuing 24

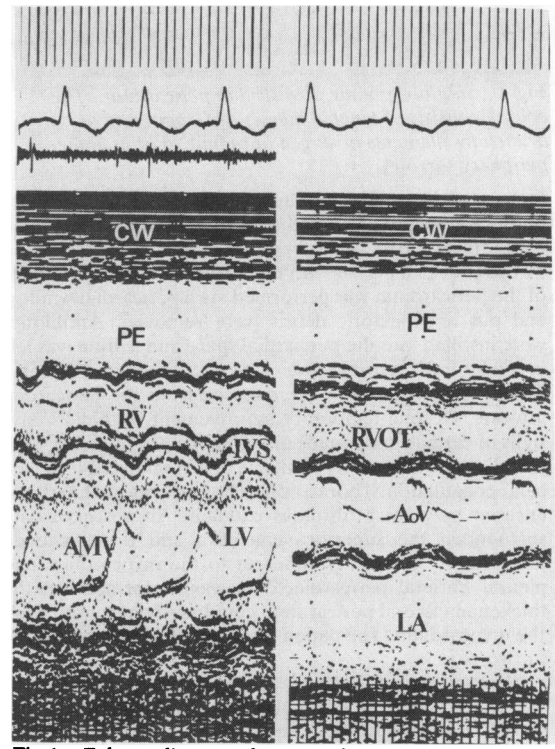

Fig 1 *Echocardiogram showing a large anterior pericardial effusion (PE). CW—chest wall; RV—right ventricle; RVOT—right ventricular outflow tract; IVS—interventricular septum; AMV—anterior mitral valve* leaflet; AoV—*aortic valve; LA, LV—left antrium and ventricle.*

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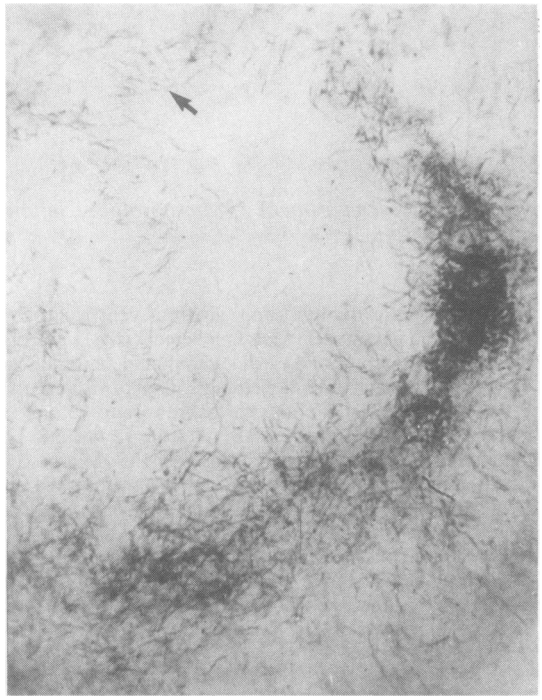

Fig 2 *Sulphur granule identified in pericardium. The colony consists of tangled masses of Gram positive branching filaments arranged in radiate fashion at the periphery (arrow).*

hours, however, the systolic blood pressure and urinary output decreased again and the blood urea and creatinine concentration rose to 31·9 mmol/l (192·2 mg/100 ml) and 321 μmol/l (3·6 mg/100 ml) respectively. Surgical drainage of the pericardium was performed via a pericardial window and pus and necrotic debris were removed. Antibiotics were instilled into the pericardial space and a drain was left in situ. Oral metronidazole was started when anaerobic Gram negative bacilli were cultured from the pericardial aspirate. Despite temporary improvement, within 12 days signs of cardiac tamponade and renal failure returned, with jaundice and laboratory evidence of disseminated intravascular coagulation. Thoracotomy showed that the heart was encased by a thick fibrinous exudate. An abscess cavity surrounded the superior vena cava and the thickened pericardium was firmly adherent to the myocardium and pleura. Parietal pericardiectomy was performed but the thickened visceral pericardium could not be removed from the myocardium. The patient deteriorated further and died in two days.

Microbiological culture of the pericardial aspirate yielded Gram positive and negative anaerobic bacilli; both were extremely fastidious and neither was identified. Histological examination of the pericardium, however, showed typical "sulphur granules" among fibrous and chronic inflammatory tissue. The colonies consisted of tangled masses of Gram positive branching filaments arranged in radiate fashion at the periphery, characteristic of the genus *Actinomyces* (fig 2).

Discussion

As a cause of purulent pericarditis, actinomycosis is rare.[5] Two cases, however, have been reported, one of which appeared to be a primary infection.[4 6] A similar bloodborne pathogenesis originating from *Actinomyces* in the mouth may have occurred in our patient, who had many carious teeth.

The genus *Actinomyces* consists of anaerobic or microaerophilic organisms and failure to collect and culture specimens under strict anaerobic conditions may be responsible for the high incidence (40%) of negative cultures in proved cases of actinomycosis affecting other organs.[7] Histopathological study may, however, be diagnostic.

Holm[8] found associated bacteria in more than 95% of specimens of pus from cases of human actinomycotic infection. It is thought that by lowering the oxygen tension of affected tissues their presence may improve conditions of anaerobic growth for the actinomycetes. Furthermore, it has been shown that such normally penicillin resistant organisms can be eradicated, together with the actinomycetes, by penicillin treatment.

The clinical and echocardiographic findings in pericardial effusion and tamponade are well recognised, but purulent pericarditis can be diagnosed only by pericardiocentesis. Although fewer than 30% of cases are actually diagnosed during life the need for prompt diagnosis and aggressive treatment is emphasised by the high mortality rates of 40% and 100% reported for treated and untreated individuals.[9 10] Thoracotomy and surgical drainage are preferable to repeated pericardiocentesis and should be performed to obtain histological specimens if aspiration yields pus but no microbiological diagnosis. Acute pericardial constriction or tamponade demand prompt surgical relief and adequate cardiac decompression may require extensive stripping of the epicardial surface of the heart.

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