

Pericardial abscess causing right ventricular outflow tract obstruction

Successful surgical correction

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SUMMARY A 67 year old man with a six month history of gradually worsening malaise and dyspnoea presented in cardiac failure. Investigations showed a pericardial abscess leading to right ventricular outflow obstruction and pericardial constriction. Drainage of the abscess and partial pericardiectomy led to full recovery.

Case report

A previously fit 67 year old man developed central chest pain while on holiday in the Channel Islands. Examination disclosed a pyrexia of 39°C and a loud pericardial friction rub. Chest x-ray film showed slight cardiomegaly but no evidence of lung consolidation. Electrocardiographic abnormalities supported the clinical diagnosis of pericarditis and he was treated with tetracycline 250 mg four times a day and indomethacin 25 mg three times a day. Though his pyrexia subsided in the ensuing three weeks, he remained unwell and his erythrocyte sedimentation rate, which was normal on admission, increased to 142 mm/h, (Westergren). Prednisolone, 10 mg three times a day, was given with initial subjective improvement. This improvement was not maintained and six months after his initial presentation with pericarditis he was referred to King's College Hospital.

On examination his jugular venous pressure was raised 10 cm above the sternal angle and he had bilateral ankle oedema. He was in sinus rhythm at 92 beats/minute and his blood pressure was 150/80 mmHg. A grade 3/6 ejection systolic murmur was heard at the left sternal edge. Examination was otherwise unremarkable.

Investigations

The haemoglobin was 13.5 g/dl, and the white cell count was $8.8 \times 10^9/l$ with a normal differential. The erythrocyte sedimentation rate was 40 mm/h. Routine biochemical screening was normal apart from a slight

increase in the gamma glutamyl transpeptidase and alkaline phosphatase and a slight reduction in serum albumin. Autoantibodies and rheumatoid factor were negative. Chest x-ray film showed cardiomegaly (CTR 17:2.9), with upper lobe venous diversion. Echocardiography indicated a restrictive pattern of movement of the right ventricular and posterior left ventricular walls. Electrocardiogram showed sinus rhythm with left axis deviation. The electrocardiographic complexes were less than 0.5 mV in all leads. Cardiac catheterisation showed a gradient of 36 mmHg across the right ventricular outflow tract. Right atrial mean pressure was 16 mmHg. Right ventricular pressure was 60/20 mmHg and left ventricular end-diastolic pressure was 16 mmHg. Right atrial angiography (Fig. 1) showed a band of opacity between the injected contrast and the lung fields, suggesting the presence of pericardial thickening or effusion. A severe right ventricular infundibular stenosis was delineated by right ventricular angiography (Fig. 2). A left ventricular angiogram showed normal systolic function, with impaired diastolic relaxation.

FINDINGS AT OPERATION

At operation (Mr A Macarthur) the pericardium was found to be densely adherent and grossly thickened. A large, anterior, thick walled abscess cavity was opened and 800 ml of yellow pus was aspirated. The cavity was roughly triangular with the apex extending on to the pulmonary artery and the base at the lower border of the right ventricle. On the right it extended to cover the right atrium while on the left it extended to just beyond the left anterior descending coronary

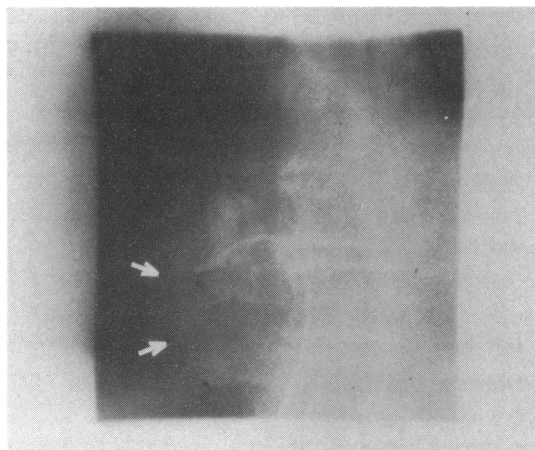


Fig. 1 Contrast injected through a catheter positioned against the lateral wall of the right atrium fails to opacify an area between the catheter tip and the edge of the lung field (arrowed). This suggests either pericardial thickening or the presence of pericardial effusion.

artery. The abscess was drained and a partial pericardiectomy performed. The patient made a full recovery. Fourteen months after operation he was well with no signs of cardiac failure. Right heart catheterisation at this time showed normal right sided pressures with a mean pulmonary artery wedge pressure of 6 mmHg. There was no pulmonary infundibular gradient and angiography showed a normal right ventricular outflow tract (Fig. 2).

PATHOLOGICAL FINDINGS

Microscopy of the abscess contents disclosed Gram positive cocci and numerous pus cells. Histological examination of the pericardium showed vascular fibrous tissue with changes of chronic inflammation but no evidence of malignancy. Mycobacteria were not found by specific staining or culture techniques. The only organism isolated was *Staphylococcus aureus*, and this only from enrichment culture media.

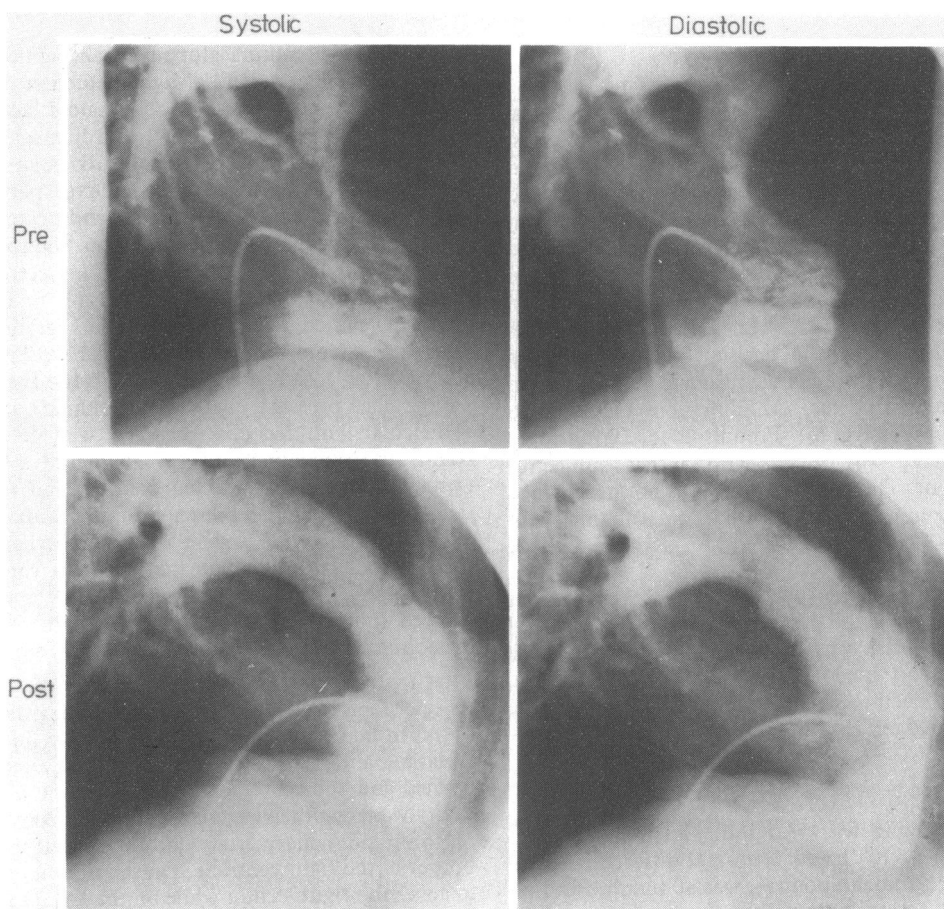


Fig. 2 Systolic and diastolic frames of right ventricular angiogram in lateral position showing right ventricular outflow obstruction which was relieved by operation.

Discussion

We believe this case represents the first description of right ventricular outflow tract obstruction caused by pericardial abscess. Infundibular pulmonary stenosis resulting from pericardial band formation has been reported but is rare without a history of pericardial surgery.¹⁻³

In our case the presence of pericardial constriction was suggested by the clinical picture^{4,5} and the abnormal movement of the ventricular wall detected by echocardiography.⁶ The clinical diagnosis was supported by the findings at cardiac catheterisation^{5,7} when right ventricular outflow obstruction was also demonstrated. The history and subsequent findings suggest that abscess formation and pericardial constriction followed acute pericarditis. Since the advent of the antibiotic era purulent pericarditis has been an uncommon diagnosis^{8,9} and when it does occur it is usually in the setting of a debilitating disease, chest trauma, or severe infection elsewhere.¹⁰⁻¹³ It is of interest that this patient had no predisposing illness, and has enjoyed uninterrupted good health since recovering from surgery.

Though the value of steroid treatment in selected cases of "benign" or viral pericarditis is well recognised, there are clearly dangers to such treatment.^{8,12,14} In this case we feel that the steroids and a bacterostatic antibiotic may have played a part in producing the unusual clinical picture, either by altering the course of a purulent pericarditis or by allowing a secondary infection after a viral illness.

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