CASE REPORT

Cardiac rupture caused by *Staphylococcus aureus* septicaemia and pericarditis: an incidental finding

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Abstract

A 35 year old woman with a long history of intravenous drug abuse presented to a local hospital with severe anaemia, fever, raised markers of inflammation, and positive blood cultures for *Staphylococcus aureus*. She responded to treatment with antibiotics with improvement in her symptoms and markers of inflammation. Four weeks later a "routine" echocardiogram showed a rupture of her left ventricular apex and a large pseudoaneurysm. There had been no deterioration in her symptoms or haemodynamic status to herald this new development. It was successfully repaired surgically and the patient made a good recovery.

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Keywords: ventricular rupture; pseudoaneurysm; staphylococcal septicaemia

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Cardiac rupture and pseudoaneurysm of the left ventricular free wall is a well recognised complication of myocardial infarction, cardiac trauma or cardiac surgery.[1 2] There are a few reported associations with endocarditis, septicaemia or tumour infiltration.[3–6] These reported cases are all associated with a sudden or progressive haemodynamic deterioration. We report a case of cardiac rupture and pseudoaneurysm of the left ventricular free wall found on "routine" echocardiography in a patient with *Staphylococcus aureus* septicaemia.

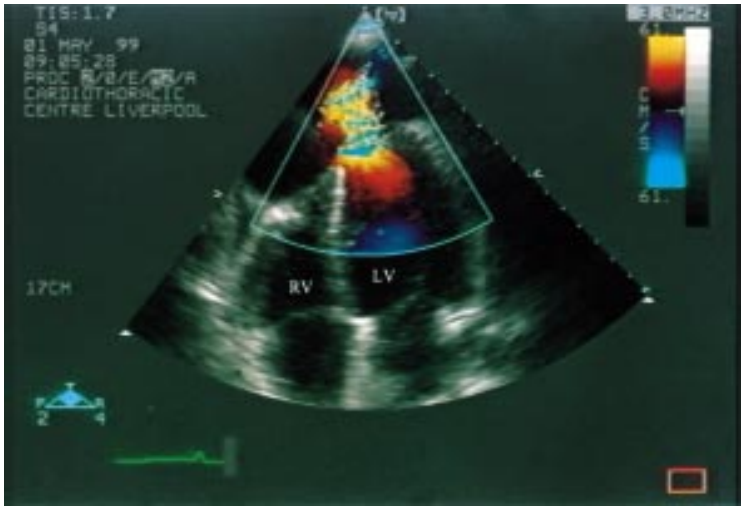

Figure 1 Apical four chamber view with colour flow Doppler, showing the point of cardiac rupture and false aneurysm. LV, left ventricle; RV, right ventricle.

Case report

A 35 year old female intravenous drug abuser was referred to a local hospital with a three week history of malaise, lethargy, and weight loss. She had lost 10 kg in three weeks and complained of left sided pleuritic chest pains.

She had a long history of intravenous drug misuse and had been on a methadone withdrawal programme for two months, although she admitted to the concomitant use of heroine. In 1992 and 1997 she was admitted to the same hospital with a left proximal deep vein thrombosis and right groin abscess respectively caused by injecting heroin into her femoral vein. A systolic murmur had been heard and an echocardiogram booked, but she had defaulted.

On examination she looked ill and had a low grade pyrexia. She had a tachycardia of 100 beats/min but was normotensive. There were no cutaneous stigmata of endocarditis and the only positive finding was a systolic murmur suggestive of mitral regurgitation.

Investigations revealed a microcytic anaemia with a haemoglobin of 3.5 mmol/l (5.7 g/d1) and a white cell count of 10.7 $\times$ 10^9/l. Inflammatory markers were raised (C reactive protein 130 mg/l and erythrocyte sedimentation rate 54 mm/hour), and two sets of blood cultures grew *S aureus*. Her chest *x* ray was normal. The ECG showed non-specific T wave inversion affecting the inferiolateral leads. Her echocardiogram revealed normal left ventricular systolic function and a left ventricular band near the apex. There was mild mitral and tricuspid regurgitation but no vegetations were seen. The aortic valve was normal. A small pericardial effusion was noted. A diagnosis of bacterial endocarditis was made and she was treated with appropriate antibiotics (flucloxacillin and gentamicin) and given a transfusion of 4 units of blood. Her temperature settled and markers of inflammation improved.

Three weeks into treatment she developed acute left sided pleuritic chest pain with further T wave inversion across her inferior and anterolateral leads. A ventilation/perfusion scan was performed which excluded a pulmonary embolus and a repeat echocardiogram was unchanged. She remained haemodynamically stable and her pain settled spontaneously. A week later a "routine"

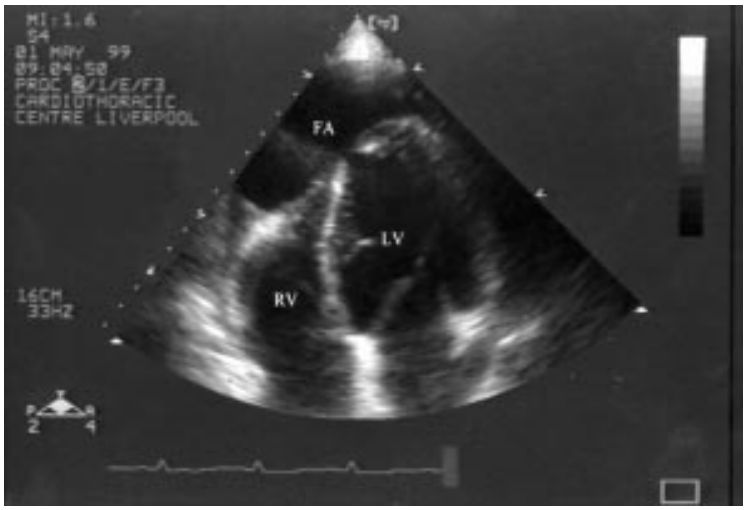

Figure 2 Apical four chamber view showing the point of free wall rupture. FA, false aneurysm.

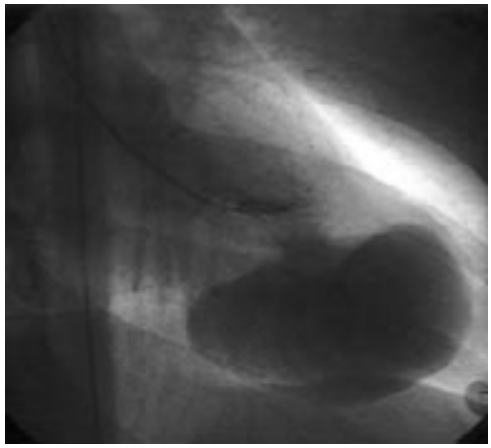

Figure 3 Left ventricular angiogram showing false aneurysm.

echocardiogram revealed a rupture of the left ventricular apex and a large pseudoaneurysm (figs 1 and 2). Left ventricular systolic function remained normal with no change in the degree of mitral or tricuspid regurgitation. No vegetations were identified and the aortic valve was normal. She was transferred to the regional cardiothoracic centre where she underwent cardiac catheter studies which confirmed the presence of an apical pseudoaneurysm of the left ventricle (fig 3) and normal coronary arteries. The patient underwent emergency cardiac surgery, which revealed a false aneurysm arising from the free wall of the left ventricle. There was a 1 cm punch hole rupture contained by pericardial adhesions, which was successfully repaired. Postoperatively, intravenous antibiotic treatment was continued and the patient made a good recovery.

Discussion

Cardiac rupture and pseudoaneurysm of the left ventricular free wall associated with endocarditis or *S aureus* septicaemia and pericarditis is exceedingly rare.[4 5] In all reported cases there was a major deterioration in the patients' well being. Our patient was stable and the diagnosis was made by a "routine echocardiogram". The mechanism of rupture with infective endocarditis is caused by either septic embolisation of aortic valve vegetations resulting in a mycotic abscess, or myocardial infarction.[3] Spread of infection to the surrounding myocardium and rupture of an abscess remote from the affected valve have been reported.[7 8] Myocardial abscesses have been associated with purulent pericarditis which can lead to rupture.[4 5]

In this case the diagnosis of endocarditis was not well established. A mitral murmur was noticed two years earlier and the degree of regurgitation did not change during this admission. Blood cultures confirmed *S aureus* septicaemia, and the ECG changes and symptoms of chest pains were suggestive of pericarditis, which could account for her life saving pericardial adhesions.

Rupture of the left ventricular free wall is a potentially fatal complication of septicaemia with pericarditis or endocarditis. The onset, as in this case, may not be heralded by symptoms or haemodynamic deterioration. The diagnosis can easily be made by echocardiography. There is clearly a role for echocardiographic screening at regular planned intervals in patients with endocarditis or septicaemia with cardiac involvement. Had the initial diagnosis not been made by routine repeat echocardiography the patient would have been at high risk of sudden death.

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