Infective endocarditis from group C streptococci causing stenosis of both the aortic and mitral valves

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Infective endocarditis (IE) carries a high mortality despite modern antimicrobial therapy. Valvular replacement in the acute phase of IE was first described in 1965¹ and plays an important role in treatment.

Lancefield group C streptococci very rarely cause endocarditis, but when this occurs, the course tends to be acute with gross tissue destruction leading to valvular incompetence.²

It is extremely rare for the vegetations in IE to cause valvular stenosis.³⁻⁵ We report a case of acute infective endocarditis caused by Lancefield group C haemolytic streptococci in which the vegetations produced both aortic and mitral valve stenosis.

Case report

A 38-year-old butcher (BG) was admitted to hospital with a 10-day history of general malaise, fever, progressive shortness of breath, jaundice, and drowsiness. Four days before the onset of symptoms he had cut his finger while at work. He had received trimethoprim/sulphamethoxazole for three days and subsequently tetracycline. There was no history of rheumatic fever.

On examination, he was pyrexial (39°C), jaundiced, drowsy, and short of breath at rest. His pulse showed atrial fibrillation, the pulse rate was 98/min, and his blood pressure was 100/50 mmHg. His jugular venous pressure was raised by 2 cm. Auscultation revealed a third heart sound, an aortic systolic murmur and the murmur of mitral regurgitation. He had bilateral basal crepitations.

His chest radiograph showed cardiomegaly and pulmonary oedema. Serum electrolytes were abnormal (sodium 127 mmol/l, potassium 3.4 mmol/l, urea 21 mmol/l, creatinine 230 µmol/l). Liver function tests were also abnormal: albumin of 21 g/l, SGOT 133 IU/l (normal less than 35 IU/l), SGPT 51 IU/l (normal less than 30 IU/l), LDH 1427 IU/l (normal range: 200-500 IU/l) and the total bilirubin 196 µmol/l (normal less than 17 µmol/l). His haemoglobin was normal and his white cell count was $22.1 \times 10^9/l$ (89% neutrophils). His ASO titre was 480 IU/ml (normal less than 200 IU/ml). The echocardiogram showed a thickened mitral valve which moved normally. No

definite vegetations were seen on either the mitral or the aortic valve. The left ventricular dimensions were normal. A small pericardial effusion was present. His initial blood cultures grew a streptococcus, Lancefield group C, sensitive to penicillin and cefotaxime.

The day after his admission he reverted to sinus rhythm with first degree heart block. He was started on cefotaxime (1 g tds iv) and back titration of his serum revealed bactericidal concentrations. Three days after his admission he developed complete heart block leading to an asystolic cardiac arrest. Resuscitation was successful and a temporary transvenous pacemaker was inserted.

His pyrexia, mental confusion, jaundice, and pulmonary oedema showed very little improvement over three days and his antibiotic therapy was changed to benzylpenicillin (2 mega units four hourly iv) and gentamicin. His haemoglobin fell to 8.8 g/dl. A second echocardiogram now showed vegetations on the aortic valve, the posterior mitral valve cusp and chordae tendineae (fig 1). Despite antibiotic therapy he showed no improvement and at this stage an early diastolic murmur was noted for the first time. A third echocardiogram showed an increase in the vegetations with obstruction to the left ventricular outflow tract (fig 2). He was accepted for urgent surgery.

At operation there was bile-stained pericardial effusion. Granulation tissue was seen on the right side of the aortic root. The aortic valve was explored and was found to be almost completely obstructed by vegetations, the infective process extending down the aortic root anteriorly into the ventricular septum. The necrotic tissue was removed, leaving a ventricular septal defect. The aortic valve was excised, but was too disorganised to permit identification of cusp anatomy.

The mitral valve was explored through the left atrium. Massive vegetations involving the lateral papillary muscle and chordae tendineae were found, extending through the valve orifice and obstructing it. The vegetations and mitral valve were removed. Bjork mitral (31 mm) and aortic (25 mm) valve prostheses were inserted and the ventricular septal defect was closed with a Teflon felt patch. An epicardial pacemaker lead was attached to the right ventricle and connected to a permanent lithium generator. He made a good recovery and was discharged five weeks later taking warfarin only.

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Fig 1 (a) Vegetations related to posterior leaflet of the mitral valve and the chordae tendineae (arrowed). (b) Vegetations on the aortic valve (arrowed).

(a)

(b)

Discussion

Lancefield group C streptococcal infections are usually seen in animals, particularly cattle, horses and poultry, and carrier states are known in these animals. Two of the four recognised species can cause disease in man, commonly minor illnesses such as pharyngitis, and may be isolated from throat swabs. Serious infections such as septicaemia and endocarditis are extremely rare. Mohr *et al*² reported the results of 150 000 blood cultures in a nine-year period in which group C streptococci were isolated in only eight patients.

There are 11 previously reported cases of IE caused by Lancefield group C streptococci,²⁻⁶ five of whom died, four with destructive lesions affecting the heart valves. In two further patients urgent cardiac surgery was required, in the first because of heart failure and septal abscess formation and in the second because of aneurysm of a sinus of Valsalva with a fistula into the right ventricle. This organism thus appears to be highly destructive of cardiac tissue. In our patient, septal abscess formation and complete heart block confirmed again the invasive nature of this organism. In six cases there was no history of pre-existing heart

Fig 2 Vegetations causing obstruction to the left ventricular outflow tract (arrowed).

disease, a feature also seen in our patient. However, the most striking aspect of our case was the obstruction of the mitral and aortic valves by the profuse vegetations. This may be related to rapid growth of the vegetations despite adequate bactericidal concentrations of antibiotics.

Serial echocardiograms showed increasing vegetations related to the aortic and mitral valves. Echoes in the left ventricular outflow tract, confluent with the septum, confirmed our clinical impression of septal abscess formation causing the conduction disturbance. There are few reported cases of single valve stenosis caused by vegetations in IE.³⁻⁵ In the most recent case reported by Copeland *et al*⁵ the diagnosis, made by echocardiography, led to urgent valve replacement. Stenosis of the two valves as illustrated by our case has not previously been described.

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