Tricuspid valve excision without replacement in a case of endocarditis secondary to drug abuse

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Summary A patient is presented in whom urgent surgical intervention was necessary to alleviate the consequences of tricuspid valve endocarditis. Six months after operation she remains well, without a valve prosthesis.

Tricuspid valve endocarditis is a well-recognised complication of drug addiction (Arbulu et al., 1973; Curtis et al., 1974). In heroin addiction there is evidence that the most likely causative organism is the staphylococcus (Norro et al., 1974; Ramsey et al., 1970). In most cases, however, staphylococcal endocarditis responds to intensive antibiotic treatment (Arbulu et al., 1973). We report here the case of a young drug addict in whom, despite antibiotic therapy, removal of the tricuspid valve with evacuation of the pericardium and débridement of infected tissue were required to alleviate the haemodynamic consequences of her endocarditis.

Case history

A 27-year-old woman was admitted to this hospital in October 1975 with a pyrexia which had failed to respond to antibiotic therapy. Previously she had pneumonia at the age of 2 years, hepatitis at the age of 17, and glandular fever at 18. She also admitted to heroin addiction 7 years previously but denied recent drug abuse. In March 1975 she developed a fever and investigation at another hospital showed albuminuria, raised blood urea (20 mmol/l), persistent anaemia, and, later, abnormal liver function tests with negative tests for hepatitis B antigen. Abdominal x-ray and intravenous urography confirmed mild hepatomegaly and disclosed normal kidneys. A laparotomy carried out for abdominal pain confirmed the hepatomegaly but no other abnormality was found. At this time she was also noted to have an apical systolic murmur and coagulase positive staphylococci were obtained from blood and bone-marrow cultures. She received 12 courses of antibiotics over a 6-month period, including fucidic acid and cephalaxin for 4 weeks before her transfer to this hospital, without effect on her fever.

On arrival at our hospital she was noted to be pale. The heart rate was 110 per minute and there was a left ventricular heave, with an apical ejection systolic murmur, and a soft third heart sound. The liver was enlarged and there was mild ascites. One week after admission, while under investigation, her condition rapidly deteriorated, her venous pressure rising to +13 cm H₂O, while the chest x-ray film showed an increase in heart size. Aspiration of the pericardium yielded a blood-stained effusion. Staphylococci resistant to fucidic acid were grown from her blood and she was treated with flucloxacillin and gentamicin.

Though satisfactory bactericidal blood levels of both drugs were achieved for 3 weeks she remained pyrexial. She then complained of bilateral pleuritic chest pain and developed increased ascites and peripheral oedema. Investigations at this time showed that she had a Hb 7·7 g/dl, WBC 7000, ESR 87 mm in the first hour, serum complement C₃, 47, and creatinine clearance 18 ml per minute. Antinuclear factor was positive 1:250. The x-ray film and electrocardiogram were consistent with a diagnosis of pericardial effusion. Cardiac catheterisation confirmed severe tricuspid regurgitation, probably secondary to acute bacterial endocarditis, with impaired left ventricular function as shown by an ejection fraction of 0·39 and a raised left ventricular end-diastolic pressure of 10 mmHg. There was also a pericardial effusion (Fig.). Surgery was immediately proceeded to on cardiopulmonary bypass.

At operation there was a tense organising suppurative pericardial effusion which appeared to be producing significant cardiac tamponade. The tricuspid valve was grossly regurgitant because of total destruction of the septal leaflet and dilatation of the tricuspid annulus. There were organising vegetations at the site of the septal leaflet. The organising pericardial exudate was evacuated, the tricuspid valve completely excised, and both were
sterile on bacteriological examination. Postoperatively she recovered well and her cardiac failure was easily controlled with frusemide and digoxin. Her renal function remained satisfactory with a serum creatinine of 100 μmol/L. One year later she remains well with tricuspid regurgitation and hepatomegaly while controlled on 80 mg frusemide and 250 μg digoxin daily. She has, in addition, reverted to oral methadone on occasion but there is no firm evidence of intravenous drug abuse.

Discussion

The onset of congestive cardiac failure in the course of infective endocarditis carries a grave prognosis (Manhas et al., 1972). In our patient increasing cardiac failure was evidently induced by the development of a pericardial effusion and aggravated by tricuspid valve regurgitation. At operation both valve and pericardial fluid were found to be sterile, and bactericidal antibiotic blood levels had been obtained for 18 days before operation. In view of a total of 7 weeks' antibiotic therapy it seems unlikely that active staphylococcal infection was the cause of the patient's sudden rapid deterioration necessitating surgical intervention. Our experience suggests that the clinical improvement in this patient was primarily because of the relief of her suppurative tamponade together with the débridement of all infected tissue. The absence of gross haemodynamic complications postoperatively supports the argument against prosthetic valve replacement (Manhas et al., 1972; Arbulu et al., 1972, 1973) at the time of operation, which in our patient is reinforced by her evident continued susceptibility to drug abuse.

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References


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