and cardiac enlargement. The ECG was characteristic of acute pericarditis. His condition deteriorated 3 days later when he became hypertensive with an elevated JVP and pulsus paradoxus. A repeat chest X-ray revealed an increase in the cardiac diameter and an echocardiogram demonstrated a large, fibrinous pericardial effusion with right ventricular diastolic collapse (Fig. 1). Nine hundred millilitres of green pus was drained following the insertion of a pericardial catheter and culture of this confirmed *Staphylococcus aureus* infection for which he was treated with flucloxacillin, gentamicin and vancomycin. After 4 days the drainage catheter occluded and therefore we proceeded to create a pericardial window under general anaesthesia during which further pus was easily removed.

During this illness his CD4 count fell from 147 cells \( \text{mm}^{-3} \) to 86 cells \( \text{mm}^{-3} \), but he made a full recovery following a prolonged course of antibiotics and remains well several years later despite this. An echocardiogram performed 5 years after the pericardietomy was normal with no evidence of residual pericardial disease.

Cardiac tamponade has occurred in two of the other six reported cases of *Staphylococcus aureus* pericarditis in HIV-positive patients[3,4] and has been treated effectively with pericardiocentesis and pericardiectomy[5]. Although a recent study suggested that surgical intervention is not beneficial to AIDS patients with pericardial effusion[6], there are no data on the long term outcome of such measures in patients at an earlier stage of HIV infection. Our case confirms that aggressive surgical and medical management is appropriate because their prognosis may be much better than some authorities have suggested.

P. F. CURRIE*  
R. A. WRIGHT*  
C. CAMPANELLA†  
J. GRAY†  
N. A. BOON*

*Department of Cardiology and  
†Department of Cardiothoracic Surgery,  
Royal Infirmary of Edinburgh;  
‡Regional Infection Unit,  
City Hospital,  
Edinburgh, U.K.

References

commissures has been documented to be the mechanism by which mitral valve area increases following balloon valvuloplasty. This same mechanism is responsible for mild increases in mitral regurgitation[6].

Outcome following balloon valvuloplasty has been related to the BVR score[7]. Only one study has been able to demonstrate a correlation between valvular morphology and the development of mitral regurgitation[8]. From an echocardiographic point of view the patient described had a favourable valve for mitral commissurotomy. The severe mitral regurgitation that resulted was due to non-commissural tearing of the posterolateral leaflet. This complication is in keeping with the findings of Essop[9], who speculates that 'excessively pliable valves may constitute a risk factor for leaflet rupture and abrupt mitral regurgitation'. Though outcome was better for patients with a lower BVR score in the NHLBI outcome was better for patients with abrupt mitral regurgitation. Though outcome was better for patients with a lower BVR score in the NHLBI study[3], valve morphology did not predict the development of mitral regurgitation in this or other studies[10].

Late in-stent restenosis in coronary arteries and in grafts

Intracoronary stents were at first limited to the treatment of restenosis, angioplasty-related acute coronary closure and graft lesions. Their use has greatly widened to include new onset lesions in native vessels, following two recent trials showing less restenosis in patients treated with stents compared to conventional angioplasty[11,12]. No long-term prospective data are available on stenting. One report suggests that the time course of restenosis after intracoronary stenting is similar to that after conventional angioplasty[11,12].

We report on four male patients aged 50 to 70 who underwent conventional balloon angioplasty for stenosis ≥50% measured by quantitative coronary angiographic analysis. Stents were implanted for restenosis in one native right coronary artery, for angioplasty-induced acute closure in one venous graft and one native right coronary artery, and for suboptimal results after angioplasty in a venous graft. A total of six self-expandable Wallstents (Schneider AG, Zurich, Switzerland) were implanted using the standard technique[13]. All patients were treated with anticoagulation for 6 months and antiplatelet therapy. Six months following the procedure, a control coronary angiogram was performed.

Figure 1 Excised mitral valve specimen. View is from the atrial side. Note the large tear in the posterolateral leaflet [1].

References


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