revealed sinus rhythm, normal atrioventricular conduction and subepicardial ischaemia in the lateral region. The 2-D echocardiogram revealed a multitude of hydatid cyst lodges, situated around the right atrium, the lateral walls of the left and right ventricles and the left atrium. Hydatid cysts were also seen in the pericardium.

NMR (Figs 1 and 2) revealed multiple circular formations on the intermediary signal in the T₁ sequence (in regard to the cardiac cavities' signal), and in the hypersignal in the dense proton. These formations, visible on the cardiac mass at the T₂ sequence, are between 2 to 5 mm in size, and appear on the periphery with an intramural and peripheral topography. Extrinsinc compression signs are located on different cavities in relation to adjacent cystic formation, in particular at the level of the inferior vena cava, both atria and both ventricles. The cyst walls are outside the heart. The walls of the heart and the periphery of the myocardium are fluid, possibly formed by hydatid cysts.

Diagnosis of Echinococcosis was confirmed by a strongly positive haemagglutination test (titre = 1/5120), a positive ELISA reaction at 0:574 and positive immunofluorescence at 1/50. NMR exploration ruled out the presence of the disease in the rest of the body. Surgery was not considered since it could threaten the rest of the heart. Albendazole was administered. The patient was stable after 3 months of follow-up.

This case shows the value of NMR in the anatomical and topographical diagnosis of cardiac Echinococcosis. Advantages of NMR are numerous. No radiation is produced. It has the ability to explore multiple cut section of the heart in different planes⁵⁻⁶. No contrast injection is necessary. Blood flow creates a natural contrast between blood and cardiovascular structures⁵⁻⁶. 

Our observation shows the value of NMR in diagnosing the incidence and spread of disease. It permits the diagnosis of bi-atrial, bi-ventricular and intra-pericardial cysts. It can exclude the presence of hydatidosis in preferential sites, such as liver and chest. NMR exploration would appear to be the gold standard for diagnosing cardiac hydatidosis and its complications.

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Long-term survival following pericardiectomy for Staphylococcus aureus pericarditis in an HIV-positive drug user

Pericardial effusion occurs in up to 10% of HIV-positive patients and although the majority are small and clinically insignificant⁴¹, reports of cardiac tamponade are becoming more common, particularly in association with opportunistic infection or pericardial malignancy⁴¹. In 1989 we treated a 22-year-old male, HIV-positive, intravenous drug user who had developed Staphylococcus aureus pericarditis by pericardiocentesis followed by partial pericardiectomy. His subsequent long-term survival has highlighted the importance of appropriate treatment for this condition.

The patient gave a 3-week history of pleuritic chest pain and dyspnoea on exertion and had been using intravenous buprenorphine regularly. He was known to be HIV-positive through contact tracing 18 months previously but had not satisfied criteria for a diagnosis of AIDS.

On examination he was thin and unwell with a pyrexia of 38.2°C. His JVP was not elevated initially and his heart sounds and chest examination were normal. A chest X-ray showed a small right pleural effusion.
and cardiac enlargement. The ECG was characteristic of acute pericarditis. His condition deteriorated 3 days later when he became hypotensive 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 mm$^{-3}$ to 86 cells 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 pericardiectomy 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[3-6] and has been treated effectively with pericardiocentesis and pericardiectomy[3]. Although a recent study suggested that surgical intervention is not beneficial to AIDS patients with pericardial effusion[4], 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‡

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References


Severe mitral regurgitation following Inoue balloon valvuloplasty

Severe mitral regurgitation is an infrequent complication of Inoue balloon mitral commissurotomyny[1]. It may result from chordal rupture or tearing of the leaflets. The scoring system developed by Wilkins et al. based on analysis of echocardiographic variables was developed to predict procedural outcome, not post-procedural mitral regurgitation[5]. This scoring system, adopted by the NHLBI Balloon Valvuloplasty Registry (BVR score) assesses valve pliability, calcification, thickening and subvalvular apparatus (each score 1–4, total score 4–16). The relationship of BVR score to the subsequent development of severe mitral regurgitation remains controversial[1-3,4].

A 58-year-old female with severe rheumatic mitral valve stenosis (mitral valve area=0.7 cm$^2$, transmitral pressure gradient=24 mmHg) underwent percutaneous balloon mitral valvuloplasty at our institution using the Inoue technique (26 mm balloon, patient height=164 cm). Pre-procedural transthoracic echocardiography demonstrated marked doming of the anterior leaflet. The BVR score was 7 (pliability=1, calcification=2, thickening=3, subvalvular apparatus=1). The first and only balloon inflation was complicated by severe mitral regurgitation which necessitated surgical replacement with a mitral valve prosthesis. At operation the mitral valve commissures were noted to be fused. There was a large tear in the posterolateral leaflet and torn chordae tendineae from the posteromedial papillary muscle group (Fig. 1).

There is very little published graphical information on the valve pathology associated with severe mitral regurgitation following Inoue valvuloplasty. This case illustrates the unpredictability of this potentially serious complication and the associated pathological correlations. It also highlights the fact that the echocardiographic BVR score predicts widening of the valve following valvuloplasty but not necessarily the development of complications. The pre-procedure morphological predictors of severe mitral regurgitation remain to be determined.

Morbidity and mortality with percutaneous balloon mitral valvuloplasty is low and similar to that seen with surgical commissurotomy. The development of severe mitral regurgitation (≥3+ angiographic grade) has been reported as low as 0% and as high as 19%. Usually the risk is of the order of 2-4%[3]. Splitting of fused