- 9 **Sogaard P**, Egeblad H, Kim WY, *et al*. Tissue Doppler imaging predicts improved systolic performance and reversed left ventricular remodeling during long-term cardiac resynchronization therapy. *J Am Coll Cardiol* 2002;**40**:723–30.
- 10 **Gorscan J III**, Kanzaki H, Bazaz R, *et al*. Usefulness of echocardiographic tissue synchronization imaging to predict acute response to cardiac resynchronization therapy. *Am J Cardiol* 2004;**93**:1178–81.
- 11 **Yu CM**, Zhang Q, Wing-Hong Fung J, *et al*. A novel tool to assess systolic asynchrony and identify responders of cardiac resynchronization therapy by tissue synchronization imaging. *J Am Coll Cardiol* 2005;**45**:677–84.
- 12 **Rector TS**, Cohn JN. Assessment of patient outcome with the Minnesota Living with Heart Failure questionnaire: reliability and validity during a randomized, double-blind, placebo-controlled trial of pimobendan. Pimobendan Multicenter Research Group. *Am Heart J* 1992;**124**:1017–25.
- 13 **Refsgaard J**. This is a walking test not a talking test: the six minute walking test in congestive heart failure. *Eur Heart J* 2005;**26**:749–50.
- 14 **Bland JM**, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986;**327**:307–10.
- 15 **Bleeker GB**, Kaandorp TA, Lamb HJ, *et al*. Effect of posterolateral scar tissue on clinical and echocardiographic improvement after cardiac resynchronization therapy. *Circulation* 2006;**113**:969–76.
- 16 **Murphy RT**, Sigurdsson G, Mullanalla S, *et al*. Tissue synchronization imaging and optimal left ventricular pacing site in cardiac resynchronization therapy. *Am J Cardiol* 2006;**97**:1615–21.

IMAGES IN CARDIOLOGY

doi: 10.1136/hrt.2006.099465

Four-valve endocarditis caused by group G Streptococci

A 41-year-old man, a known intravenous drug user with positive hepatitis C serology, was admitted with a 4-day history of malaise, rigors and night sweats.

Clinical examination demonstrated a pyrexia (39.4°C), sinus tachycardia (120 bpm) and hypotension (blood pressure 85/55 mm Hg). Auscultation disclosed pansystolic and early diastolic murmurs. There were no peripheral stigmata of endocarditis. Initial blood tests showed a haemoglobin of 105 g/l; white cells $20.3 \times 10^9/l$; neutrophils $18.5 \times 10^9/l$; C reactive protein 250 mg/l; urea 17.3 mmol/l and creatinine 178 mmol/l.

Transthoracic and transoesophageal echocardiograms showed a non-dilated hyperdynamic left ventricle with small vegetations on the mitral valve leaflets (arrows, panel A) and chordae tendinae and mild mitral regurgitation. There was a vegetation on a flail aortic left coronary cusp (arrow, panel B) that prolapsed into the left ventricular outflow tract together with severe aortic regurgitation (panel C). The tricuspid valve had a large vegetation on the septal leaflet (panel A) with moderate tricuspid regurgitation. A trans-thoracic subcostal view also revealed a small vegetation on the pulmonary valve (arrow, panel D)

Infective endocarditis was diagnosed and treatment was started with empirical antibiotics and inotropic support. Blood

cultures grew group G Streptococci. Intravenous benzylpenicillin and gentamicin were started.

Emergency aortic valve surgery with a homograft and tricuspid valve vegetectomy were performed 10 days after admission. He was discharged after a further 6 weeks of antibiotic treatment.

Most echocardiographically demonstrated endocarditis occurs on a single valve; the involvement of two valves

occurs much less frequently, and triple- or quadruple-valve involvement is extremely rare.¹

J P Fernandez, D B McKenzie, P R Roberts
 juan.fernandez@doctors.org.uk

Reference

- 1 **Kim N**, Lazar JM, Cunha BA, *et al*. Multi-valvular endocarditis. *Clin Microbiol Infect* 2000;**6**:207–12.