- 30 **Ruf A**, Pick M, Deutsch V, et al. In-vivo platelet activation correlates with red cell anionic phospholipid exposure in patients with beta-thalassaemia major. *Br J Haematol* 1997;**98**:51–6.
- 31 **Tavazzi D**, Duca L, Graziadei G, et al. Membrane-bound iron contributes to oxidative damage of beta-thalassaemia intermedia erythrocytes. *Br J Haematol* 2001;**112**:48–50.
- 32 **Tavazzi D**, Comino A, Turrini F, et al. Indices of membrane alterations in beta-thalassaemic erythrocytes. *Hemoglobin* 1998;**22**:483–92.
- 33 **Garozzo G**, Distefano R, Miceli A, et al. Detection of ICAM-1, ICAM-2, ICAM-3, PECAM-1 and VCAM-1, evaluation of hypercoagulable state and platelet aggregation in hemoglobinopathy patients with erythroblasts. *Haematologica* 2001;**86**:778–9.
- 34 **Atichartakarn V**, Chuncharunee S, Chandanamatta P, et al. Correction of hypercoagulability and amelioration of pulmonary arterial hypertension by chronic blood transfusion in an asplenic hemoglobin E/beta-thalassemia patient. *Blood* 2004;**103**:2844–6.

IMAGES IN CARDIOLOGY

doi: 10.1136/hrt.2005.083865

Multiple intracardiac thrombi in a patient with dilated cardiomyopathy

Two dimensional transthoracic echocardiography of a modified apical four chamber view showing multiple mobile clots in both the left and right ventricles on admission (A) and complete resolution of the thrombi in left ventricle with only a small residual thrombus in the right ventricle after 10 days of anticoagulation treatment (B).

The occurrence of multiple biventricular thrombi in patients with dilated cardiomyopathy is extremely rare. A 47-year-old man with a history of 25 years of cocaine and amphetamine use presented with recent onset of weakness, palpitations, persistent dry cough and shortness of breath on minimal exertion. At admission, physical examination revealed sinus tachycardia with pulse 100/min, blood pressure 94/64 mm Hg, elevated jugular venous pressure, bilateral basal rales, S3 gallop, 2/6 midsystolic murmur at the apex, and mild hepatomegaly. Relevant laboratory tests were normal (including troponin I) except for an elevated natriuretic peptide (578 pg/ml, normal < 100 pg/ml).

Chest x-ray showed cardiomegaly, patchy bilateral pulmonary infiltrates (pulmonary congestion with suspected superimposed pneumonia) and a small pleural effusion. Abdominal ultrasound revealed mild hepatomegaly with dilatation of hepatic veins and inferior vena cava. Blood cultures and serology for HIV and hepatitis were negative. The ECG showed sinus rhythm and poor R wave progression in leads V1–V4. Echocardiography demonstrated a dilated left ventricle with severely depressed left ventricular function (ejection fraction –15%) with global biventricular hypokinesis, right ventricle dysfunction, severe mitral and severe tricuspid valve regurgitation and mobile multiple clots in both the left and right ventricles (to view video footage visit the *Heart* website—<http://www.heartjnl.com/supplemental>). Adenosine single photon emission computed tomography (SPECT) was performed to exclude obstructive coronary artery disease and was negative for ischaemia. The evaluation for hypercoagulable disorder was negative. The working diagnosis was dilated cardiomyopathy possibly related to amphetamine and/or cocaine use with recent onset of congestive heart failure, multiple intracardiac thrombi and bilateral pneumonia. Conventional treatment for congestive heart failure was started. Antibiotics were given for bilateral pneumonia. Intravenous heparin was administered for the multiple intracardiac thrombi.

After seven days of heparin the patient was put on warfarin and the echocardiogram on the 10th day of treatment showed resolution of the clots in the left ventricle and only a small residual clot in the right ventricle. There was no evidence of embolism on physical examination or on chest and brain CT. The patient's general condition stabilised and the symptoms of heart failure improved. Biventricular pacing and automatic implantable cardioverter-defibrillator implantation are planned after complete clot resolution.

In this patient the severe biventricular dysfunction most likely led to the formation of multiple biventricular thrombi and the thrombi resolved on anticoagulation treatment.

To view video footage visit the *Heart* website—<http://www.heartjnl.com/supplemental>

S Golland
T Luo
R J Siegel
siegel@cshs.org