Heart failure: be aware of reversible causes

Sir,

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia. There is wide recognition that AF is an important risk factor for thromboembolic events, particularly stroke. A less well-recognized (but nonetheless important) consequence of prolonged and usually unrecognized AF is left ventricular dysfunction, which resolves with treatment of the atrial fibrillation.

A 50-year-old man developed breathlessness on exertion, and a cough productive of streaky haemoptysis. He had just returned from a recent business trip to the Middle East. Admission to hospital was precipitated by acute severe breathlessness. On examination, he looked unwell, with jaundice, ascites and gross oedema. He was in fast atrial fibrillation at 150 bpm, with a blood pressure of 92/60 mmHg. There were no murmurs. Liver and renal function tests were abnormal: creatinine 163 μmol/l; bilirubin 71 μmol/l. He was treated with intravenous dobutamine, nitrate, diuretics and amiodarone. Echocardiography revealed dilated cardiac chambers (left ventricle 6.8 cm in diastole and 5.6 cm in systole) with globally and severely impaired left and right ventricular function. Left ventricular ejection fraction was 20%. A coronary angiogram showed normal coronary arteries. A cardiac MRI study was performed, which revealed no evidence of myocarditis or infarction. A myocardial biopsy was taken, which was reported as normal. Initially, he was thought to be a possible candidate for heart transplantation but after treatment with ACE inhibitors, beta blockers and diuretics, there was good clinical improvement with reduction in the patient’s heart rate. After 10 days he reverted to sinus rhythm. On discharge from hospital he was asymptomatic. On follow-up, the patient remained in sinus rhythm and denied any limiting symptoms. A repeat echocardiogram, 7 months after presentation, demonstrated marked improvement in left ventricular function (ejection fraction 60%; left ventricular diastolic dimension 5.3 cm and systolic dimension 4.0 cm). In addition, there was a notable reduction in cardiac size on follow-up chest X-ray (Figures 1 and 2).

Left ventricular dysfunction in the presence of AF is believed to be a consequence of the rapid ventricular response to the atrial arrhythmia causing a tachycardia-induced cardiomyopathy. This was clearly described in 1913 by Gossage and Braxton Hicks in one of the first monographs on atrial fibrillation.

This case illustrates the importance of identifying patients in whom atrial fibrillation is the cause rather than the consequence of left ventricular dysfunction. Pointers include the presence of AF with a rapid ventricular rate, mild rather than severe left ventricular dilatation, the lack of any other apparent cause of the heart failure (normal coronary arteries, normal thyroid function, no history of drug or alcohol abuse). As has been demonstrated in the above case, treatment of the underlying tachycardia results in positive left ventricular remodelling with improvement.
resolution of ventricular dilatation, normalization of systolic function and complete resolution of symptoms of heart failure. Tachycardia-induced cardiomyopathy is one of the few reversible causes of heart failure.2,4 Recovery of ventricular function can be variable and depends on the duration of the tachycardia, as well as co-existent heart disease. Ventricular improvement occurs most quickly in the first weeks after correction of the tachycardia, with a period of slow improvement for up to 6–8 months.5 Furthermore, recurrent tachycardia can cause rapid decline in left ventricular function and development of heart failure in patients with previously treated tachycardia-induced cardiomyopathy. Sudden death has also been reported in such cases.6 It is thus important not only to recognize and treat this condition, but also to prevent recurrence.

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Evaluating broad-spectrum antimicrobials

Sir,

In a recent QJM paper, investigators at the Shaare Zedek Medical Center, Jerusalem studying the prospective utility of cefepime, piperacillin-tazobactam and meropenem recommended formal infectious disease consultation for their appropriate usage.1 Ideally, empirical treatment regimens with broad-spectrum and/or costly antimicrobials would be based on unit-specific data. Nevertheless, any retrospective data on the in-vitro susceptibility profile of bacteria encountered in local intensive care could help guide selection of an appropriate approach. Therapeutic intervention is immediate and cannot be linked with the in-vitro susceptibility profile of the isolates. Background information on the local prevalence of extended spectrum β-lactamase producers (ESBL) and methicillin-resistant S. aureus (MRSA) would thus be an asset.

In the Sant Parmanand Hospital, a 140-bed, tertiary-care, private-sector hospital in Delhi, antimicrobial susceptibility of isolates encountered from clinical material is primarily computed for 28 antimicrobials. Retrospective data from October 2004 onwards are used to recommend antibiotics with susceptibility exceeding 75% against organisms encountered in urinary tract, blood and pyogenic foci.2 Initial screens would exclude cefepime, piperacillin-tazobactam and meropenem. However, from December 2005, a two-tier susceptibility protocol has been used on isolates from patients in the medical and surgical intensive care units, nursery, or patients with aggressive infections. They are tested for in-vitro susceptibility for cefepime, piperacillin-tazobactam and meropenem. Among 114 isolates up until May 2005, there were 46 Klebsiella strains, 30 E. coli, 10 Proteus, 8 Pseudomonas, and 20 S. aureus. The respective cumulative susceptibilities to cefepime,