Left ventricular dysfunction resulting from frequent unifocal ventricular ectopics with resolution following radiofrequency ablation

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A case is presented, in which asymptomatic but persistent right ventricular outflow tract (RVOT) ectopics resulted in left ventricular (LV) dilatation and systolic dysfunction. The patient underwent extensive investigation with no other cause for the cardiomyopathy being found. Successful ablation of the RVOT ectopic focus resulted in normalization of LV size and function. This case suggests that frequent ventricular ectopy should be considered as a potentially remediable cause of LV dysfunction.

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Introduction

Repetitive or sustained tachyarrhythmia is a well-recognized and potentially reversible cause of cardiomyopathy[1,2]. The possibility that frequent premature ventricular complexes (PVCs), without tachycardia, might similarly result in a reversible cardiomyopathy has been proposed[3] and a single case has been reported, in which a dilated cardiomyopathy, associated with frequent PVCs, resolved after radiofrequency (RF) ablation of the PVC focus[4]. We report the case of an asymptomatic young man in whom frequent monomorphic ventricular ectopics, arising in the right ventricular outflow tract (RVOT), resulted in left ventricular (LV) dysfunction that resolved following successful RF ablation of the ectopic focus.

Case report

A 33-year-old male commercial airline pilot presented in March 1997 with ventricular ectopy on a routine 12-lead resting ECG. The ectopics had left bundle branch block morphology with an inferior axis (Fig. 1), suggesting an origin in the RVOT. The patient had no symptoms and no significant past medical history or family history. He did not smoke and drank alcohol socially in small amounts. There was no history of recreational drug use and he was on no regular medication.

A 24-h Holter recording demonstrated frequent monomorphic ventricular ectopics (approximately 31,000 in 24h). A treadmill exercise test showed no evidence of myocardial ischaemia but there were persistent ectopics throughout the test. A transthoracic echocardiogram (October 1997) was reported to be normal although no
objective measurements were made, given the degree of ectopy. In view of the limitations of echocardiography in this context, magnetic resonance imaging (MRI) was performed in December 1997. This showed mild LV dilatation with an LV end diastolic diameter (LVEDD) of 5.9 cm (Table 1). As a result of this, the patient’s medical certification to fly was restricted, permitting him only to fly as or with a qualified co-pilot. He continued to work uneventfully with this restriction until 12 months later, when a repeat echocardiogram suggested LV dilatation with mild to moderate impairment of LV systolic function. Further Holter recording documented 27,000 ventricular ectopics in 24 h. The patient remained asymptomatic but because of the apparent cardiac abnormality, his medical certification to fly was suspended and his future career threatened. A repeat MRI scan was performed in August 1999. Images were acquired during a bigeminal rhythm, gated on normal sinus beats, the same technique as was used during the previous MRI study. The dynamic images showed hypokinesis of the anterior and septal walls with a slightly increased LVEDD of 6.0 cm, a significant increase in LV volume and a reduced LV ejection fraction of 39.4% (Table 1). Proportionally more dilatation at the apex was noted and considered to be abnormal despite the bigeminal rhythm. Right ventricular size and function on this and the previous MRI scan appeared normal. Cardiac catheterization demonstrated LV enlargement with some anterior dyskinesia and evidence of systolic LV dysfunction. The epicardial vessels were angiographically normal. Laboratory investigations and chest X-ray were normal.

In view of the fact that the ectopy appeared to have preceded the onset of cardiomyopathy and in the absence of any other evident aetiology, ablation of the ectopic focus was performed in November 1999. Venous access was obtained via right femoral vein puncture. A diagnostic quadripolar catheter (6 French, Josephson fixed curve, Cordis) was positioned at the right ventricular apex and an ablation catheter (7 French, 4 mm tip, Blazer II, Boston Scientific) was advanced to the RVOT. Baseline electrophysiological studies confirmed frequent ectopics arising from the RVOT. A full diagnostic electrophysiological study was not performed and no attempt was made to induce tachycardia. Pace mapping was performed with the ectopic focus being localized in the high RVOT, below the pulmonary valve, posteriorly. Pacing at this site reproduced the morphology of the ectopics in 12 of 12-ECG leads (Fig. 2) and RF energy was delivered. After three separate energy deliveries, totalling 90 s, complete cessation of ectopic activity was observed for 30 min and this was judged a satisfactory end-point.

A subsequent 24-h Holter recording performed in December 1999 demonstrated only a single ventricular ectopic. A further 24-h Holter recording in March 2000

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<th>Table 1 Serial MRI scan measurements</th>
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<td>Ejection fraction (%)</td>
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<td>LVEDD (cm)</td>
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LVEDD, left ventricular end diastolic diameter; LVEDV, left ventricular end diastolic volume; LVESV, left ventricular end systolic volume.
was normal and a repeat MRI scan showed normal LV volume and dimensions with an LVEDD of 5.3 cm and an ejection fraction of 54% (Table 1). The patient subsequently regained restricted medical certification and returned to flying. After a further 12 months and two further 24-h Holter recordings, both of which were unremarkable, the restriction was removed from his medical certificate. He remains well and under annual review with no evidence of further ectopic activity to date.

**Discussion**

Tachycardia-induced cardiomyopathy, with resolution following successful ablation of the arrhythmia substrate, has been described in patients with a wide variety of tachyarrhythmias, including ectopic atrial tachycardia[5], permanent junctional reciprocating tachycardia[6], atrial flutter[7] and RVOT tachycardia[6]. Only recently has it been suggested that frequent ventricular ectopy, without tachycardia, may be responsible for LV impairment in patients with presumed idiopathic dilated cardiomyopathy[2] and a single case has been reported in which cardiomyopathy resolved after RF ablation of the ectopic focus[4]. The case presented is only the second such report and the first in an asymptomatic subject.

RF ablation of ventricular ectopics arising from the RVOT is a safe and effective procedure but it is usually reserved for severely symptomatic patients who fail to respond to medical therapy[9,10]. However, in the case presented, notwithstanding the lack of symptoms, it was felt that the evidence of progressive LV dysfunction on serial MRI scans, and the implications for the patient’s career as a pilot, justified proceeding to ablation. In addition to providing a curative treatment, the favourable response to ablation also served a diagnostic purpose in that it supported the clinical, laboratory and imaging evidence that there was no primary cardiac disease and the presumption, based on the temporal sequence of events, that the arrhythmia was the cause of the LV dysfunction.

The causal mechanisms for this reversible cardiomyopathy are a matter of speculation. Correlates and postulated mechanisms in other forms of tachycardia-related cardiomyopathy include neurohormonal, ultrastructural and metabolic changes, involving abnormal calcium handling and depletion of myocardial energy stores[1,2,5]. Altered myocardial perfusion has also been proposed, which would be supported in this case by the presence of a regional wall motion abnormality. A further possibility in the present case might be a phasic alteration of baroreceptor function and autonomic tone by persistent bigeminy. Alternatively, the prolonged coupling interval between ectopic and subsequent sinus beats might increase ventricular filling with an adverse effect on ventricular dynamics. Repetitive eccentric ventricular activation with asynchronous contraction is somewhat analogous to the situation during chronic right ventricular apical pacing, which has been shown to result in remodelling of the left ventricle with systolic and diastolic dysfunction in previously normal hearts[11].

This case serves to emphasize that frequent ventricular ectopy may cause reversible LV dysfunction. In patients presenting with frequent ventricular ectopy, even in the absence of symptoms or structural heart disease, regular
assessment of LV function is advisable. Transthoracic echocardiography may be difficult to interpret and gated MRI may provide a more reliable indication of early LV dysfunction. If LV dysfunction does occur, antiarrhythmic treatment should be considered, including RF ablation, which may offer the prospect of a permanent cure.

References