showed decreased extent of interstitial infiltration (Figure 1E) and ITI progressively increased (Figure 1A). Pulmonary function test also revealed significant improvement.

Discussion

Two hypotheses have been put forward for the mechanisms of amiodarone-induced AFA, including a direct cytotoxic injury and an indirect immunologic reaction.2 Clinical diagnosis of amiodarone-induced AFA requires the exclusion of other diagnostic possibilities, such as autoimmune or infectious disease-related AFA.2 In this patient, amiodarone-induced AFA was supported by full-filling four of the seven diagnostic criteria proposed by Martin et al.2

Current evidence suggests that amiodarone (200–400 mg/day) should be added to a β-blocker once an ICD recipient experiences recurrent VT or ICD shocks. However, 5–7% of the patients taking long-term amiodarone might insidiously develop pulmonary toxicity, such as amiodarone-induced AFA.2 To the best of our knowledge, this is the first case in the literature demonstrating that amiodarone-induced AFA (decreased ITI) mimicking HF can be antecedently detected by OptiVol > 3 months prior to clinical symptoms. Interestingly, the pre-clinical interval (from triggering of the OptiVol alarm to the occurrence of clinical symptoms) in this particular case with amiodarone-induced AFA (> 3 months) was much longer than that in HF patients (an average of 15.3 ± 10.6 days).1 In such a scenario, physicians might reduce/discontinue amiodarone dosage and add/switch to prednisolone, rather than adding/ increasing diuretics.

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CASE REPORT

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What is the time course of reversal of tachycardia-induced cardiomyopathy?

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Although the reversal of tachycardia-induced cardiomyopathy has been described previously, the time course of improvement in biventricular systolic function associated with tachycardia-induced cardiomyopathy has not been described. We present the case of a tachycardia-induced cardiomyopathy associated with atrial fibrillation. A transthoracic and transoesophageal echocardiogram performed the day prior to ablation revealed severe right and left ventricular (LV) systolic dysfunction. New York Heart Association Class IV symptoms and LV function were noted to improve 12 h after a successful catheter ablation. However, right ventricular (RV) systolic function remained depressed. A transthoracic echocardiogram demonstrated improvement of RV systolic function 5 months later.

A 59-year-old male with a history of symptomatic paroxysmal atrial fibrillation (AF) refractory to dofetilide was referred for catheter ablation. Despite medical therapy, the patient had episodes of AF associated with New York Heart Association (NYHA) Class IV symptoms. Prior to ablation, a transthoracic echocardiogram (TTE) revealed a left ventricular ejection fraction (LVEF) of 25–30%. The patient underwent a successful wide area circumferential ablation of his pulmonary veins with confirmation of entrance and exit block and no inducible arrhythmias. On discharge the next day, a TTE revealed a normal LVEF.

Two and a half years after his first ablation, the patient developed recurrent AF associated with NYHA Class IV symptoms of shortness of breath, palpitations, and fatigue. The patient decided to undergo a repeat AF ablation. On the day of his ablation, a TTE revealed biventricular dysfunction with an LVEF of 25–30% [Supplementary material online, Video 1a (parasternal long axis) and b (apical four chamber)]. He was in AF for 48 h prior to his repeat ablation with a mean heart rate of 110 bpm and a blood
pressure of 108/73 mmHg. Three of his four pulmonary veins had reconnected and were successfully isolated with ablation. There were no inducible arrhythmias on conclusion of the procedure.

A TTE performed 12 h later showed the LVEF improved to 55% with a mean heart rate of 93 bpm and a blood pressure of 110/65 mmHg [Supplementary material online, Video 2a (parasternal long axis) and b (apical four chamber)]. However, moderate right ventricular (RV) dilatation and moderately depressed RV function were noted.

A TTE performed 5 months after ablation (sinus rhythm, mean heart rate of 55 bpm) demonstrated a normal RV systolic function in addition to the sustained LVEF improvement [Supplementary material online, Video 3a (parasternal long axis) and b (apical four chamber); Figure 1A, tricuspid valve annular plane peak systolic velocity = 12.8 cm/s (normal >11.5 cm/s), and B, tricuspid valve annular plane systolic excursion = 2.2 cm (normal >1.5 cm)].

Discussion
This case is unusual in demonstrating a complete reversal of tachycardia-induced cardiomyopathy with improvement of LV systolic function within 24 h of AF ablation and the restoration of sinus rhythm. In addition, RV systolic function was restored 5 months after treatment.
after AF ablation. Other groups have demonstrated an improvement in LV systolic function after the restoration of sinus rhythm after AF ablation over a 6-month follow-up. This case is unique for the dramatic improvement in LV systolic function and symptoms of congestive heart failure <24 h after AF ablation with evidence of RV remodelling and improved RV systolic function 5 months after ablation. Although it is possible that the irregular and rapid heart rate during AF may have impaired the estimation of ejection fraction, the reproducible improvement of systolic function almost immediately after the second ablation suggests that the restoration of atrioventricular synchrony and atrial transport function can improve preload and cardiac output in patients with a tachycardia-induced cardiomyopathy. The improved systolic function with a lower heart rate suggests that improved lusitropic function may have contributed to our patient’s improved systolic function both acutely and with short-term follow-up. This correlates with the findings of Gentleski et al. who have noted that AF ablation can improve LV systolic function with the restoration of sinus rhythm despite satisfactory rate control pre-ablation.

This case report also highlights several variables that can be assessed to stratify a patient’s risk of tachycardia-induced cardiomyopathy. In a retrospective review of tachycardia-mediated cardiomyopathy secondary to focal atrial tachycardia, Medi et al. identified several variables predisposing to cardiomyopathy—an incessant tachycardia, male gender, a mean ventricular rate of 117 ± 21 bpm, and an origin of the tachycardia from the appendages or the pulmonary veins. Dandamudi et al. found that in patients whose LVEF normalized 14 months after treatment for a tachycardia-induced cardiomyopathy, negative remodelling of the ventricles was still apparent by echocardiography with an elevated LV end-systolic and end-diastolic volume index compared with age-, gender-, and ejection fraction-matched controls without tachycardia-induced cardiomyopathy.

This case illustrates how rapid and marked improvement in LV systolic function with additional RV remodelling and improvement in RV function can occur with the restoration of sinus rhythm immediately after radiofrequency ablation of AF.

Supplementary material
Supplementary material is available at Europace online.

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References

CASE REPORT

A completely subcutaneous implantable cardioverter defibrillator system functioning simultaneously with an endocardial implantable cardioverter defibrillator programmed as pacemaker

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Because of multiple ventricular lead fractures with inappropriate shocks, a 31-year-old male received a completely subcutaneous implantable cardioverter defibrillator (ICD) system with the already existing ‘endocardial’ ICD functioning as an atrial pacemaker.

Case report
A 31-year-old male received an implantable cardioverter defibrillator (ICD) because of documented polymorphic ventricular tachycardias and collapse during exercise. He was also equipped with an atrial lead because his atrial rate repeatedly dropped below 40 beats per minute due to the use of calcium antagonists and beta blockers to suppress his ventricular arrhythmias.