

Case Report

Idiopathic Right Ventricular Outflow Tract Tachycardia Induced during Acute Episode of Atrial Fibrillation

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Dual tachycardia, right ventricular outflow tract tachycardia, atrial fibrillation.

Tachycardia-induced tachycardia is a rare condition. We report a case in which an episode of paroxysmal atrial fibrillation led to right ventricular outflow tract tachycardia. We also analyse all potential mechanisms that might contribute to the coexistence of these two different arrhythmias in the same patient.

Tachycardia-induced tachycardia, or so called “dual tachycardia”, is a condition that has only rarely been reported in the literature. Episodes of ventricular tachycardia (VT) or ventricular fibrillation during a paroxysm of supra-ventricular tachycardia are not infrequent in patients with left ventricular dysfunction.¹ The incidence, however, of dual tachycardia in patients without structural heart disease is particularly low. An association between atrioventricular nodal reentry tachycardia and right ventricular outflow tract tachycardia (RVOT) has been reported in a few cases.^{2,3} To our knowledge, however, there is only one previous report describing the induction of RVOT as a result of paroxysmal atrial fibrillation (AF).⁴

Case presentation

An obese 72-year-old female patient was admitted to our hospital through the emergency department for palpitations and mild dizziness. She did not mention any episode of complete loss of consciousness. She had been treated for hypertension with ACE-inhibitors for many years, but had no other medical problems. She had never experienced palpitations, syncope or presyncope

in the past. On admission, the patient was haemodynamically stable. The ECG revealed fast AF with long runs of regular, broad complex tachycardia (left bundle branch block morphology with right axis) at a rate of 185 bpm (Figure 1a). Adenosine administration resulted in transient termination (Figure 1b) and an intravenous β -blocker (esmolol) in complete abolition of the broad complex tachycardia, while the basic rhythm remained AF.

Further investigation with a thallium scan did not reveal ischaemia, while an echo scan showed a moderately dilated left atrium (45 mm) with normal left and right ventricular systolic function. The patient refused to undergo electrical cardioversion and an attempt at chemical cardioversion with ibutilide failed. An electrophysiological study was performed two weeks later, after β -blocker and coumadine treatment had been discontinued for three days before the procedure.

Quadripolar catheters were placed in the His position, right ventricular apex and right ventricular outflow tract. The basic intervals were measured within normal limits. Adenosine administration (12 mg) caused complete atrioventricular block without evidence of existing pre-excitation. Right

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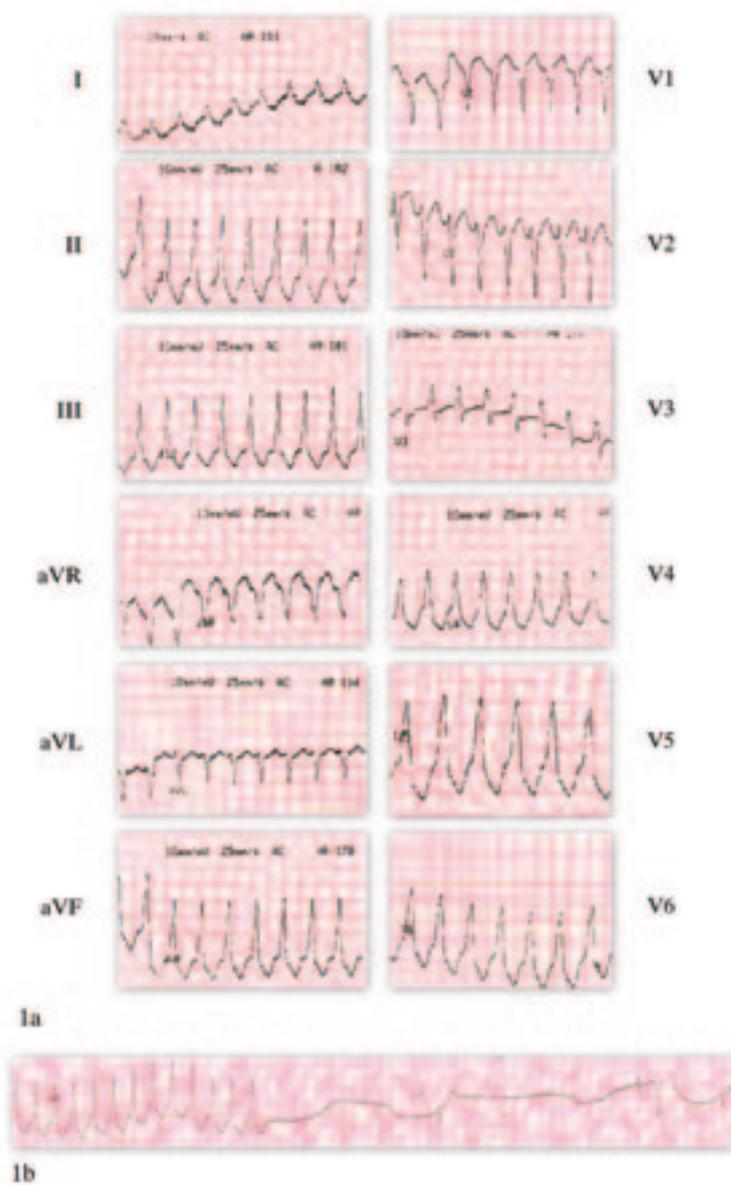


Figure 1. (a) Twelve lead ECG (25 mm/s) during broad complex tachycardia and (b) its termination by adenosine administration.

ventricular stimulation from its apex and outflow tract, introducing single, double and triple extrastimuli during two paced cycle lengths (500 and 400 ms), did not induce ventricular tachycardia. Stimulation with concurrent infusion of isoproterenol (up to 10 $\mu\text{g}/\text{min}$) produced a few ventricular ectopic beats originating from the right ventricular outflow tract, with a morphology very similar to that of clinical tachycardia. Pace mapping from a site localised in the septal and rather superior area of the right ventricular outflow tract produced QRS complexes very similar to those of broad complex tachycardia (Figure 2). Ablation was not attempted since attempts to induce even short runs of VT failed.

Discussion

It is generally accepted that the mechanism of idiopathic RVOT is c-AMP-mediated, triggered activity caused by delayed afterdepolarisations.⁵ In our case, it is possible that AF played the triggering role for VT initiation.

Several possible mechanisms may be involved. Irregular RR intervals during AF could induce VT by increasing the pause-dependent afterdepolarisation above a critical threshold. This is consistent with the rather repetitive pattern of VT initiation in our case, following a long-short RR sequence (Figure 3). A similar initiation sequence has been also reported by others.⁶

A reflex increase in sympathetic tone due to the haemodynamic effects of atrial tachyarrhythmia could

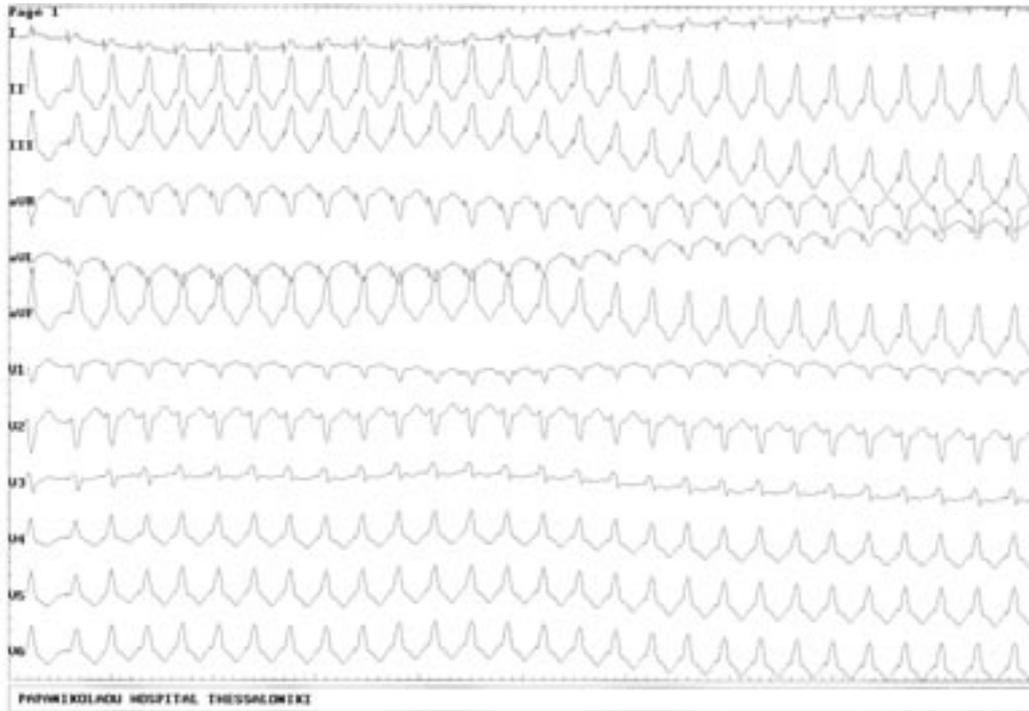


Figure 2. Twelve lead ECG (25 mm/s) during pace mapping. The QRS morphology during pacing in the septal and superior region of the right ventricular outflow tract is almost identical to the clinical tachycardia.

be an additional reason. Transient oscillations in sympathetic tone increase intracellular c-AMP and calcium, which can induce VT. This is in keeping with ventricular tachycardia termination after the administration of esmolol and adenosine, which both decrease

the sympathetic tone and catecholamine dependent stimulated levels of c-AMP.

Although the assumption that AF begets VT seems reasonable, we cannot exclude an inverse mechanism. Ventricular extrasystoles with retrograde

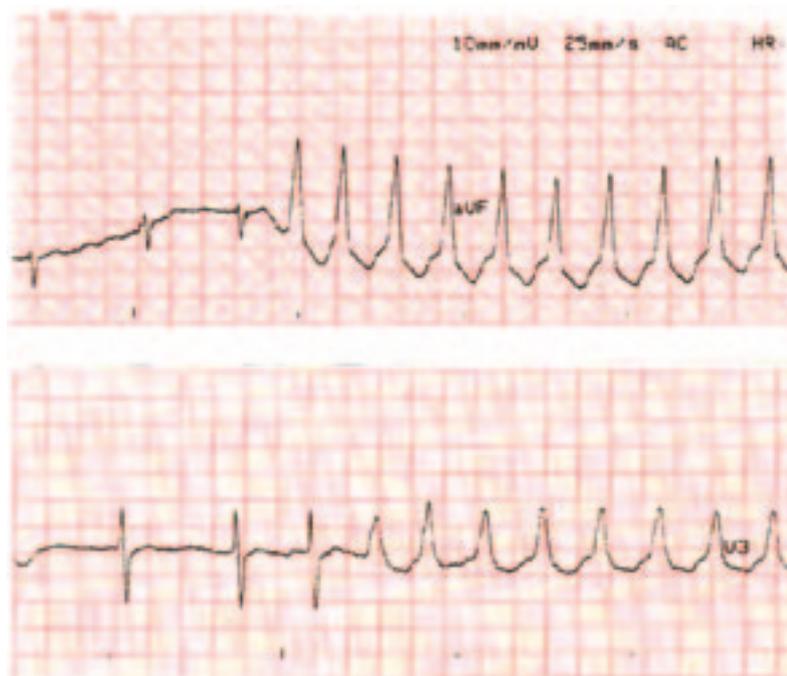


Figure 3. Examples showing initiation of the ventricular tachycardia during atrial fibrillation. Note the long– short RR cycle length sequence during atrial fibrillation preceding the initiation of ventricular tachycardia.

conduction could induce AF through a premature atrial depolarisation. This has not been tested, however, as the patient was in AF during the electrophysiological study.

We cannot explain adequately our failure to reproduce the tachycardia during programmed stimulation from different sites of the RV, despite the use of isoprenaline in high doses. A narrow range of cycle lengths for tachycardia induction may explain this failure. Experimental data have shown a decrease in the amplitude of delayed afterdepolarisations at cycle lengths above and below a specific induction window in tachycardias due to triggered activity.^{7,8} Alteration of the sympathovagal balance could also affect VT induction. A decrease in parasympathetic activity, rather than an enhanced sympathetic tone, has been related to RVOT initiation according to some authors.⁹

In conclusion we report a case of two tachycardias with apparently separate mechanisms of origin, AF and RVOT, in which one tachycardia has probably led to the induction of the other. We hypothesise that increased heart rate and ventricular cycle length variability due to AF have both acted as triggers for RVOT initiation.

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