EP CASE REPORT

Atrial arrhythmia triggering electromechanical dissociation and ventricular fibrillation in a patient with atrial switch operation

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A 26-year-old man with D-transposition of the great arteries (D-TGA) and previous Mustard atrial switch surgery was referred for catheter ablation of a recurrent symptomatic paroxysmal atrial flutter. The arrhythmia was easily inducible (*Figure 1A*, cycle length 310 ms, rate 194 b.p.m.), with rapid conduction to the ventricles. While mapping flutter, simultaneous recording of endocardial signals and invasive blood pressure monitoring showed haemodynamic deterioration with intermittent electromechanical dissociation (*Figure 1B*) and then pulseless electrical activity (*Figure 1C*). After initiation of cardiopulmonary resuscitation, several electric shocks failed to restore sinus rhythm and atrial flutter transitioned a few minutes later into ventricular tachycardia and then ventricular fibrillation (*Figure 1D*); this was ultimately terminated by defibrillation after an adrenaline bolus.

In patients with D-TGA and previous atrial switch equipped with an implant-

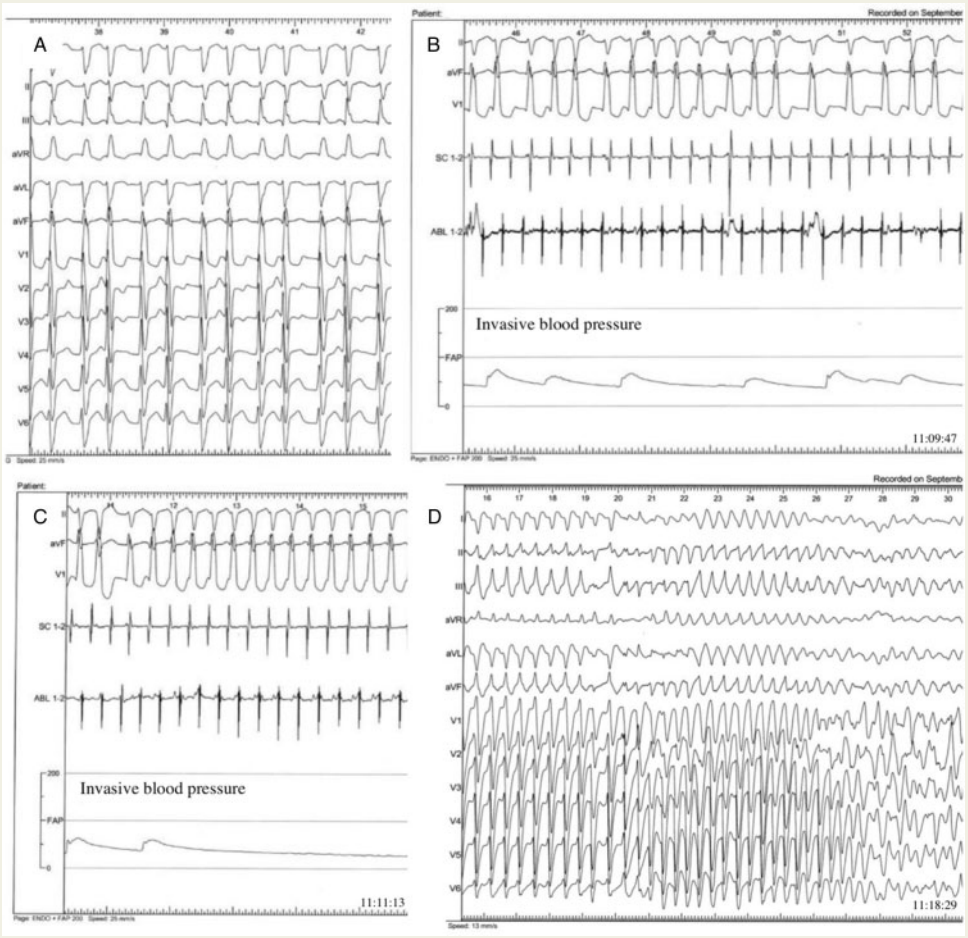

Figure 1 (*A*) Atrial flutter on 12-lead ECG induced by atrial bursts (240 ms), average ventricular response rate 140 b.p.m. (*B*) Atrial flutter recorded by endocavitary catheters (SC 1, 2 and ABL 1, 2) with electromechanical dissociation on invasive blood pressure curve. (*C*) Pulseless electrical activity. (*D*) Atrial flutter degenerating into ventricular tachycardia and then ventricular fibrillation. Invasive blood pressure was measured by an arterial line.

able cardioverter-defibrillator (ICD), a high rate of atrial arrhythmias has been reported before the onset of ventricular arrhythmias, suggesting that the former may trigger the latter.[1] Invasive haemodynamic studies have shown that in these patients, load-independent indexes of right ventricular function were preserved during exercise test or dobutamine infusion, but that stroke volume was reduced due to failure to increase right ventricular filling during tachycardia.[2] Impaired blood flow from atria to ventricles due to abnormal pathways is the most likely explanation, which may be amplified by coexisting baffle stenosis or ventricular disease. Ventricular arrhythmias may also be triggered by myocardial ischaemia due to rapid heart rates.[3] In atrial switch patients, beta-blockers are associated with fewer appropriate ICD therapies: this may depend on reducing primary ventricular arrhythmias, but also probably on reducing atrial arrhythmias and increasing diastolic filling time with slower ventricular rates. This case perfectly illustrates how atrial arrhythmias can trigger sudden cardiac death in these patients, for whom aggressive management with early catheter ablation is encouraged.

Conflict of interest: none declared.

References

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