IMAGES IN CARDIOLOGY

A diagnostic pitfall in a patient with an implantable cardioverter-defibrillator

A patient with dilated cardiomyopathy and aborted sudden death was admitted for repeated shocks from his Guidant Ventak Prizm dual chamber implantable cardioverter-defibrillator (ICD). Interrogation of stored events over the last six months revealed 46 episodes at a rate of 140/min, interpreted as ventricular tachycardia (VT). These were usually interrupted by programmed overdrive ventricular pacing, but sometimes required shocks. During monitoring, rhythm strips showed repeated episodes of narrow complex tachycardia at 136/min, initiated by an atrial premature beat (APB) with a prolonged PR interval (*), without visible P waves during tachycardia (A). These episodes set off antitachycardia ventricular pacing by the ICD with return to sinus rhythm (B). Interrogation of stored events of the ICD revealed similar episodes initiated by an APB (●), with inconsistent atrial sensing during tachycardia (AS 2033), despite evident 1:1 atrial activity on the electrogram. This was due to the very short ventriculoatrial (VA) interval, with atrial activity falling within the blanking period of 85 ms (during which the atrial channel is "blinded" to avoid far-field sensing of ventricular activity). Thus apparent atrioventricular (AV) dissociation led to misdiagnosis of VT by the device. AV nodal re-entrant tachycardia was suspected and subsequently confirmed by an electrophysiological study, leading to successful catheter radiofrequency ablation of the slow pathway. At eight months' follow up, interrogation of the ICD showed no relapse of tachycardia. This case illustrates how ICDs may be tricked by a relatively common supraventricular arrhythmia despite dual chamber technology, resulting in inappropriate shocks.

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Neonatal ECG changes caused by supratherapeutic flecainide following treatment for fetal supraventricular tachycardia

Below are the ECGs of a newborn infant (39 weeks of gestational age) at three hours (left) and three days of life (right). Fetal supraventricular tachycardia was not controlled with maternal digoxin, but was well controlled with flecainide. Four maternal flecainide concentrations were obtained at 32–35 weeks' gestation and ranged from 380–470 µg/l. Last concentration was 440 µg/l (therapeutic range 200–700 µg/l). Maternal ECGs were normal throughout. Following delivery at 39 weeks' gestation, the baby's flecainide concentration was very high (1030 µg/l) and the ECG showed a prolonged QRS duration (below left). By day 3 the QRS duration was normal (below right). The newborn was

commenced on digoxin and there was no recurrence of the arrhythmia. There is no evidence that flecainide is concentrated in the fetus and the most likely explanation of the high neonatal flecainide was that the maternal concentration had increased very late in gestation. We strongly recommend that maternal flecainide concentrations need to be checked up to term, to avoid potential maternal and fetal toxicity.

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