Out of the frying-pan into the fire: a supraventricular tachycardia triggering a ventricular tachycardia in a patient with ischaemic cardiomyopathy and an implantable-cardioverter defibrillator

Antonio Sorgente1,2*, Carlo de Asmundis1, Gian Battista Chierchia1, and Pedro Brugada1

1Heart Rhythm Management Center, UZ Brussel-VUB, Laarbeeklaan 101, 1090 Brussels, Belgium and 2Department of Cardiology, University of L’Aquila, L’Aquila, Italy
* Corresponding author. Tel: +32 2 4763038, Fax: +32 2 4776840, Email: sorgente.antonio@gmail.com

A 67-year-old man, known for an ischaemic cardiomyopathy and an implantation of a dual-chamber implantable-cardioverter defibrillator (ICD), was admitted to the emergency department of our hospital because of several internal shocks of the ICD. Upon admission, device interrogation was performed. Results of the interrogation are shown in the figure. In each panel, the first row represents atrial electrograms (AEGMs), the second row displays the leadless ECG, while the third shows the ventricular electrograms (VEGMs) obtained directly from the defibrillation lead positioned in the apex of the right ventricle. Furthermore, in the bottom of each panel, pacemaker intracardiac markers are shown together with the beat-to-beat cycle length in milliseconds. In Panel A, it is present a tachycardia with a cycle length (CL) between 440 and 450 ms, with 1:1 relation between VEGMs and AEGMs and with a VA interval close to 0 ms. Abruptly, as shown in Panel B, VA dissociation occurs and a shortening of the CL of the tachycardia from 453 to 230–240 ms is documented, through two intermediate beats characterized, respectively, by a CL of 309 and 250 ms. Interestingly, although the morphology of the VEGMs of these two beats were identical, an evident variation in the QRS aspect occurred in the leadless ECG (see asterisks), testifying a possible fusion between a ventricular and a supraventricular beat. In Panel C, a failure of the first attempt of defibrillation is documented and consequently followed by two further attempts, the latter of which, showed in Panel D, obtained the restoration of sinus rhythm. Based on these observations, a supraventricular tachycardia triggering a fast ventricular tachycardia was suspected. Thus, after a progression of coronary atherosclerosis was excluded by coronary angiography, an electrophysiological study was performed. During the electrophysiological study, no ventricular arrhythmias were inducible with the programmed stimulation from the apex of the right ventricle. Dual atrioventricular nodal physiology was instead demonstrated and an atrioventricular nodal re-entrant tachycardia was repeatedly induced during atrial programmed stimulation. Radiofrequency ablation of the slow pathway was then performed successfully and no supraventricular tachycardias were more inducible after ablation, even after intravenous infusion of isoproterenol. At 1-year follow-up, patient outcome was uneventful.