Atrioventricular nodal re-entrant tachycardia mimicking ventricular tachycardia on the surface electrocardiogram

Częstoskurcz nawrotny w węźle przedsionkowo-komorowym naśladujący częstoskurcz komorowy w 12-odprowadzeniowym elektrokardiogramie

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A 40-year-old obese male without any previous medical history was admitted to our Department due to palpitations for several hours. In electrocardiogram (ECG), wide QRS complex tachycardia was recorded (Fig. 1). Sinus rhythm was restored with amiodarone infusion. No abnormalities in physical examination and transthoracic echocardiography (TTE) and no signs of coronary artery disease in coronary angiography were observed. ECG analysis suggested that recorded arrhythmia may be ventricular tachycardia. According to the Brugada algorithm, ventricular origin of arrhythmia was indicated by the presence of fusion QRS complexes, negative concordance in precordial leads, time from the beginning of the QRS complex to S-wave nadir more than 100 ms (“Brugada sign”), and the fact that QRS morphology did not meet the left or right His bundle branch block criteria. Similar conclusions were made using Vereckei’s algorithm. Because of many ambiguities, to establish the final diagnosis an electrophysiological study was performed. Incremental atrial pacing showed normal decremental atrioventricular conduction without any evidence of pre-excitation. With programmed atrial stimulation, repetitive induction of clinical arrhythmia (cycle length 370 ms) with AH “jump” was observed (Fig. 2). Using several electrophysiological manoeuvres, atrioventricular nodal re-entrant tachycardia (AVNRT) was confirmed. After successful ablation of the atrioventricular node slow pathway, the previously observed arrhythmia was no longer inducible. In daily clinical practice, several algorithms to distinguish the origin of wide QRS complex tachycardias could be utilised. The two most often used are Brugada’s and Vereckei’s. The sensitivity and specificity of the Brugada algorithm are 98% and 96%, respectively, while that of Vereckei are 97% and 75%, respectively. In the presented clinical situation, a lack of criticism in evaluation of results and symptoms could have lead to inappropriate diagnosis and treatment. If diagnosis were not verified, it could be followed by incorrect therapy, e.g. cardioverter-defibrillator implantation for primary prevention of sudden cardiac death. In this ambiguous case, performing an electrophysiological study allowed us to establish the diagnosis of AVNRT, which was easily treated with ablation.

Figure 1. 12-lead electrocardiogram showing wide QRS complex tachycardia

Figure 2. Induction of clinical arrhythmia with AH “jump” during programmed atrial stimulation

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