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STUDIUM PRZYPADKU / *CLINICAL VIGNETTE*

Lethal arrhythmia in a 58-year-old cyclist with early repolarisation in electrocardiogram and left main coronary artery disease

Śmiertelna arytmia u 58-letniego kolarza z obrazem wczesnej repolaryzacji i chorobą pnia lewej tętnicy wieńcowej

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A 58-year-old active cyclist, who rode a bicycle about 60 km a day, without previous history of cardiovascular diseases, was admitted to our department after syncope. The episode occurred during recovery after cycling and was not preceded by any symptoms. A similar event had occurred a week before, at rest in a sitting position, and was associated with apnoea period. Moreover, his wife admitted that several nights before he had had a short period of agonal breathing during sleep. The patient denied any chest pain or other complaints, and the physical examination was normal. The resting electrocardiogram (ECG) showed sinus bradycardia and early repolarisation (ER) pattern, i.e. J-waves with horizontal ST-segment elevations in inferior and lateral leads (Fig. 1A). On echocardiography, the left ventricular function was excellent with moderate hypertrophy of the posterior wall. Laboratory tests were within normal limits except for a slight increase in cardiac markers, but without typical dynamicity seen in myocardial infarction. Exercise test yielded a negative result and confirmed his good physical fitness (12.7 METs). On another day, during ECG Holter monitoring, cardiac arrest occurred due to polymorphic ventricular tachycardia followed by ventricular fibrillation requiring defibrillation (Fig. 1B). After successful resuscitation there was no clear sign of ischaemia, but J-waves were still present. Immediate coronary angiography showed a borderline left main coronary artery stenosis, which was judged as insignificant in the context of the negative exercise test (Fig. 2). Magnetic resonance imaging presented neither features of myocarditis nor ischaemia-originated changes. In order to definitely exclude significant left main coronary artery disease, fractional flow reserve (FFR) was measured; however, the narrowing within the left main turned out to be significant (FFR: 0.63). Consequently, the man underwent coronary artery by-pass grafting. He was also proposed an implantable cardioverter-defibrillator for sudden cardiac death prevention, but he refused it. During the next 6 months no serious event occurred, and both the exercise test and Holter monitoring showed no serious abnormalities — of note, the ECG still presented an ER pattern. This case demonstrates that ER substrate may potentially increase arrhythmic risk in the context of ischaemic heart disease. Given the prevalence of the ER pattern, ER may be viewed as one of arrhythmogenic factors that is rarely solely responsible for clinical events; however, it may increase arrhythmic risk in the event of ischaemia. Although ischaemia undoubtedly matters in the arrhythmogenesis in such patients, the association of ER with arrhythmia is typically at rest or during sleep (i.e. during parasympathetic activation) and not during physical activity, as can be seen in our case.

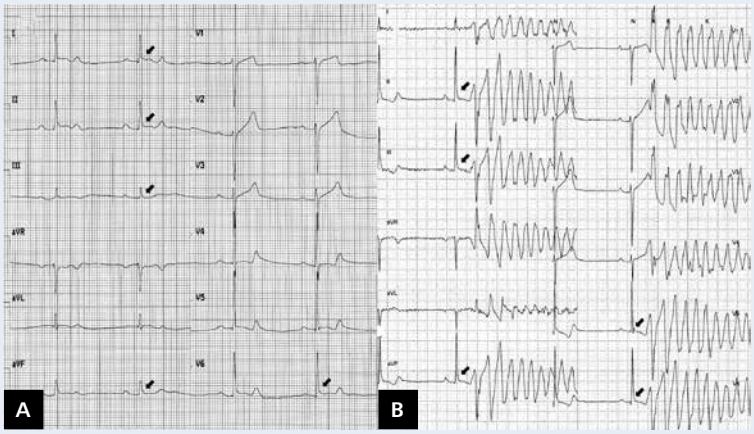

Figure 1. **A**. Electrocardiogram at admission, J-waves (arrows) and horizontal ST-segment elevations in inferior and lateral leads (such features constitute a high risk ER pattern); **B**. Holter monitoring at rest, the onset of polymorphic ventricular tachycardia. J-waves (arrows) and horizontal ST-segment elevations just before the cardiac arrest

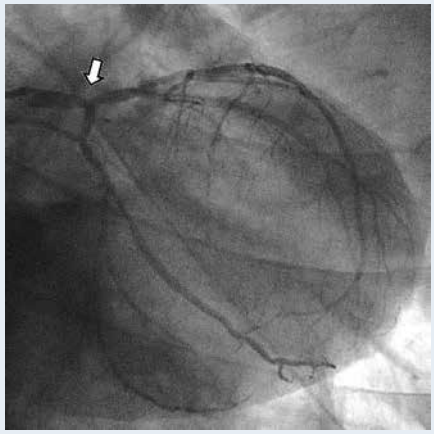

Figure 2. Coronary angiography, the borderline left main coronary artery stenosis (arrow)

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