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STUDIUM PRZYPADKU / *CLINICAL VIGNETTE*

Cardiac and neurogenic syncope and atrial flutter misdiagnosed as ventricular tachycardia in a patient after myocardial infarction

Omdlenie sercowe i omdlenie neurogenne u pacjenta po przebytym zawale serca z nierozpoznanym pierwotnie napadowym trzepotaniem przedsionków

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A 61-year-old male presented with a history of transient ischaemic cerebral episode (ten years earlier), previous myocardial infarction (MI) treated with percutaneous coronary intervention of the left circumflex artery, and deployment of a drug-eluting stent (four years earlier). Initially, he was admitted to hospital because of retrosternal pain and recurrent syncope that followed a wide QRS complex tachycardia (WCT). The arrhythmia was originally diagnosed as ventricular tachycardia (VT) and reverted to sinus rhythm (SR) using a direct current cardioversion (DCCV) followed by an intravenous amiodarone infusion. Despite the treatment, the same arrhythmia reoccurred and was accompanied by two episodes of syncope and retrosternal pain. Therefore, he was referred to cardiology department as recurrent VT secondary to acute coronary syndrome (troponin I 0.25 ng/mL) was suspected. On admission, he reported fatigue, dizziness, and pre- or syncope episodes that started two days earlier. The electrocardiogram (ECG) revealed WCT (HR = 210 bpm) of left bundle branch block (LBBB) morphology (QRS = 140 ms) (Fig. 1A). Intravenous amiodarone bolus plus a gentle carotid sinus massage (CSM) produced significant vasovagal reaction revealing atrial flutter (AFl) wave (Fig. 1B). Ultimately, DCCV to SR was performed (100 J), but AFl recurred shortly. Echocardiography and coronary angiography documented no abnormalities. The patient was qualified to cavotricuspid isthmus (CTI) radiofrequency ablation. Anticlockwise AFl (CL = 217 ms) with 2:1 atrioventricular conduction was confirmed at invasive procedure. During the manipulation with the coronary sinus catheter introduced via the external jugular vein, accidental tapping of the right carotid sinus region produced a 14-second vasovagal syncope deteriorating to ventricular fibrillation (Fig. 1C). Immediate defibrillation (200 J) restored SR and a successful CTI ablation was completed. No VT was induced with a programmed ventricular stimulation. The patient was sent for thoracic angio-magnetic resonance imaging. As well as fibrotic tissue, it revealed blood flow limitation at the right carotid sinus level. Ultimately, a dual-chamber pacemaker was implanted. No recurrent syncope or arrhythmias were observed during the subsequent 12 months. Syncope can precede cardiac arrest, especially in patients with organic heart disease. A male with previous MI and fast LBBB-WCT resulting in syncope must be suspected of VT requiring eventual implantation of a cardioverter-defibrillator. However, if excluded, it needs further differential diagnosis of WTC. Valsalva manoeuvre and CSM may be helpful in aberrated atrial arrhythmias that can be treated with radiofrequency ablation. In this patient, WCT initially misdiagnosed as VT could contribute to cardiac syncope. Moreover, in our case, the response to mild CSM was enormous, suggesting substantial carotid sinus hypersensitivity leading to neurocardiogenic syncope. The disease is also treatable, and symptoms may be resolved with implantation of a pacemaker. In the presented case the ECG diagnosis of WCT may be quite error-prone.

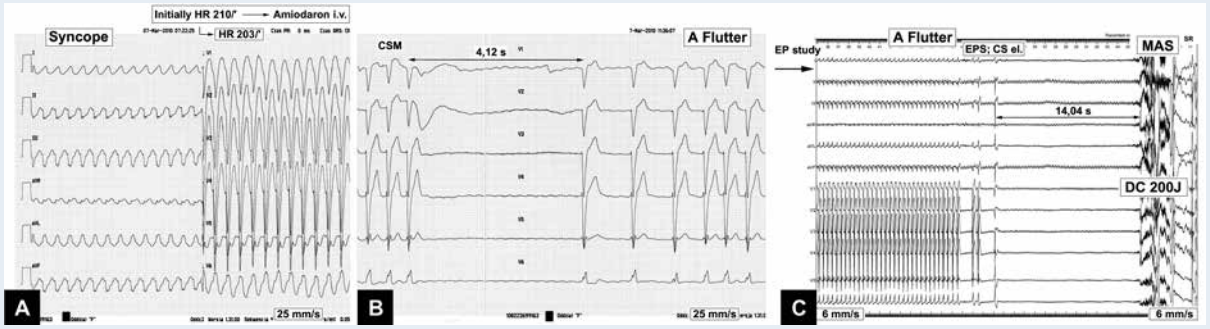

Figure 1. **A**. Atrial flutter (HR 210 bpm) producing syncope; **B**. Right carotid sinus massage documenting atrial flutter; **C**. Morgagni-Adams-Stokes syndrome episode during electrophysiology study due to accidental compression of the carotid sinus area

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