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Częstoskurcz przedsionkowy czy „atypowe” trzepotanie przedsionków? Dylematy klinicysty rozstrzygnięte przez elektrofizjologa.

Autorzy: Paweł Wałek, Ireneusz Domański-Giec, Przemysław Dąbkowski, Maciej Młodnicki, Beata Wożakowska-Kapłon

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CASE REPORT

Atrial tachycardia or atypical atrial flutter? Clinician's dilemmas resolved by electrophysiologist

Paweł Wałek[1, 2], Ireneusz Domański-Giec[3], Przemysław Dąbkowski[2], Maciej Młodnicki[2], Beata Wożakowska-Kapłon[1, 3]

[1]*Collegium Medicum*, The Jan Kochanowski University, Kielce, Poland
[2]Laboratory of Electrophysiology, Świętokrzyskie Center of Cardiology, Provincial Hospital in Kielce, Poland
[3]1st Clinic of Cardiology and Electrotherapy, Swietokrzyskie Cardiology Center, Kielce, Poland

Address for correspondence: Paweł Wałek MD, PhD, *Collegium Medicum* Uniwersytetu Jana Kochanowskiego w Kielcach; Pracownia Elektrofizjologii, Świętokrzyskie Centrum Kardiologii, Wojewódzki Szpital Zespolony, ul. Grunwaldzka 45, 25–736 Kielce, Poland, e-mail: pawel.walek@o2.pl

Abstract

We present a case report of a patient who underwent ablation of "atypical" atrial flutter. In the surface electrocardiogram, atrial tachycardia was initially diagnosed. As a result of electrophysiological mapping using the CARTOPRIME module, the diagnosis was verified and "atypical" atrial flutter was diagnosed, whose propagation wave in the right atrium met the criteria for the diagnosis of typical atrial flutter.

Key words: arrhythmia, atrial flutter, ablation, electrophysiological examination

Case report

A 60-year-old patient with post-myocardial infarction heart failure, after multiple myocardial infarctions, following coronary artery bypass surgery in 2005, after implantation of a cardioverter-defibrillator for the primary prevention of sudden cardiac death in 2017, and with paroxysmal atrial fibrillation, was admitted to the Department of Cardiology for worsening

symptoms of circulatory failure in the course of persistent supraventricular arrhythmia, which was initially classified as atrial tachycardia. A 12-lead surface electrocardiogram (ECG) recorded atrial arrhythmia with a cycle length of 580 ms with negative P-waves in leads II, III and aVF, positive ones in aVR and aVL, and a flat P-wave in lead I, which indicated a right atrial focus of the arrhythmia. The ECG recordings showed an isoelectric line between the P waves. The ECG also recorded Q-waves in leads II, III, aVF indicative of a history of inferior wall myocardial infarction (MI) (Figure 1) . During outpatient treatment, the patient's dose of beta-blockers (metoprolol) was escalated, achieving the slowing of ventricular rhythm without affecting the atrial arrhythmia cycle length. Echocardiography revealed enlargement of the right atrium (right atrial volume index — 72 mL/m^2), left atrium (left atrial volume index — 62 mL/m^2) and right ventricle, left ventricular segmental wall motion abnormalities in the inferior, inferolateral and interventricular septum walls, hypokinesis of the remaining left ventricular walls with an ejection fraction of 26%. At the Department of Cardiology, the patient was scheduled for an electrophysiology study and an attempt to ablate the arrhythmia. A transesophageal echocardiogram was performed before the ablation procedure, ruling out the presence of thrombi in the cardiac chambers. An electrophysiological study was performed using a ten-field electrode, which was inserted into the coronary sinus, and a PentaRay electrode (Biosense Webster, Inc., Diamond Bar, CA, USA). Moreover, an electroanatomical map of the right atrium was performed using the CARTO system (Biosense Webster, Inc., Diamond Bar, CA, USA). Due to the risk of arrhythmia termination due to irritation of the right atrial wall with a mapping electrode, the right atrial roof was not mapped after a full cycle length of arrhythmia was achieved. Then, using the Coherent CARTOPRIME™ module of the CARTO system, the extensive low-voltage zone was localized on the lateral wall of the right atrium and the arrhythmia cycle length was mapped (Figure 2A and 2B). Figure 2 shows the propagation waves of re-entry arrhythmia propagating counterclockwise around the tricuspid annulus. Very slow conduction of the arrhythmia wave was recorded on the lateral wall in the low-voltage zone. After the analysis of arrhythmia wave propagation around the tricuspid valve annulus, the patient was diagnosed with atrial flutter (AFl) and arrhythmia wave propagation that is specific to typical atrial flutter. Subsequently, a Smarttouch SF electrode (Biosense Webster, Inc., Diamond Bar, CA, USA) was inserted and an application was made at the site of slowed conduction on the lateral wall of the right atrium, obtaining termination of the arrhythmia and the return of sinus rhythm (Figure 3 and 4). Then, several consolidation applications were made to connect the free conduction zone to the tricuspid valve annulus. Currently, the patient is under the

constant care of the Cardiac Rhythm Disorder Centre and no recurrence of arrhythmia has been recorded.

Discussion

According to current guidelines, the ECG-based diagnosis of unifocal atrial tachycardia (AT) is made when there are P waves of identical morphology that is different from the morphology of the P waves of sinus rhythm, there is an isoelectric line between P waves in the limb leads, the frequency of P waves is in the range from 100/min. (600 ms cycle length) to 250/min (240 ms cycle length), usually above 140/min. The criteria accept P-wave irregularity. The PQ interval exceeds 100 ms, the frequency of the QRS complexes corresponds to the frequency of the atrial rhythm or is lower due to atrioventricular conduction disorders. The morphology of the QRS complexes remains unchanged except for intraventricular conduction aberrations or intraventricular conduction disorders. AT should be differentiated from typical or atypical AFl. The diagnosis of AT is supported by both the presence of an isoelectric line between P waves and P-wave irregularity [1]. AT should be considered if AFl is suspected, especially if the P-wave or F-wave frequency is in the range of 200–250/min.

In the patient in question, the criteria for the diagnosis of AT were undoubtedly met in the recorded ECG before the ablation procedure: P waves had identical morphology, were positive in aVR and aVL leads, negative in II, III and aVF leads, flat in I lead, and their frequency was approximately 103/min. and did not correspond to the P waves of sinus rhythm, there was an isoelectric line between P waves, and the PQ interval was approximately 160 ms. Despite meeting the criteria for AT on the ECG, the electrophysiological study ruled out such a diagnosis and definitively diagnosed slow AFl.

In the rare clinical situation presented here, the slowed conduction of electrical excitation within the lateral part of the right atrium was likely the result of a history of MI or the result of cannulation of the right atrium during coronary artery bypass surgery.

The case presented here provided significant diagnostic difficulties in differentiating AT from AFl. According to the European Society of Cardiology guidelines, emergency treatment for AT includes pharmacotherapy with adenosine (recommendation class IIa, level B), beta-blockers, non-dihydropyridine calcium channel antagonists (recommendation class IIa, level C). Other antiarrhythmics such as flecainide, propafenone, amiodarone can be considered (recommendation class IIb, level C). In the case of hemodynamic instability or ineffectiveness of pharmacotherapy, electrical cardioversion is recommended

(recommendation class I, level B). In chronic treatment, if tachycardia-induced cardiomyopathy is present, the highest class of recommendations is percutaneous ablation (recommendation class I, level B). The guidelines do not recommend anticoagulant treatment in AT patients [2]. In contrast, the use of thromboprophylaxis in the case of AFl is similar to that in patients with atrial fibrillation due to the risk of formation of thrombotic material, which is reflected in important clinical decisions based on the differentiation of these arrhythmias (recommendation class I, level B) [2]. In patients with heart failure, there is often a clear link between the presence of arrhythmias and cardiovascular decompensation. Therefore, antiarrhythmic treatment — including pharmacotherapy, electrical cardioversion or ablation – should not be delayed [3]. In the case of the coexistence of diagnosed heart failure with reduced ejection fraction, there are important limitations in terms of pharmacotherapy. Heart failure with reduced ejection fraction is a contraindication to the use of diltiazem and verapamil [3].

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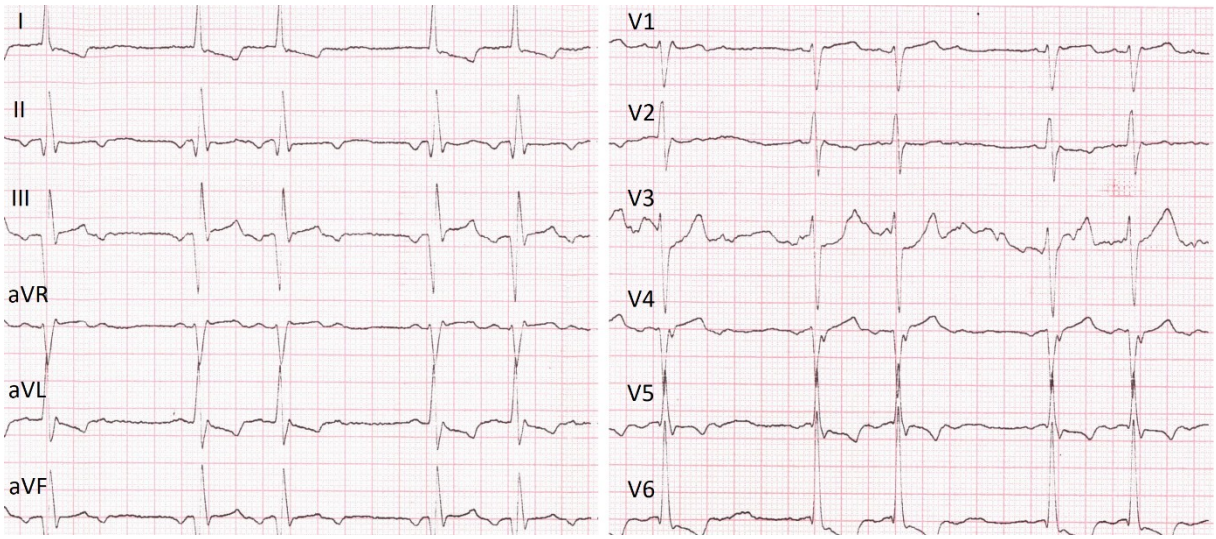

Figure 1. The 12-lead electrocardiographic recording before atrial tachycardia/atrial flutter ablation

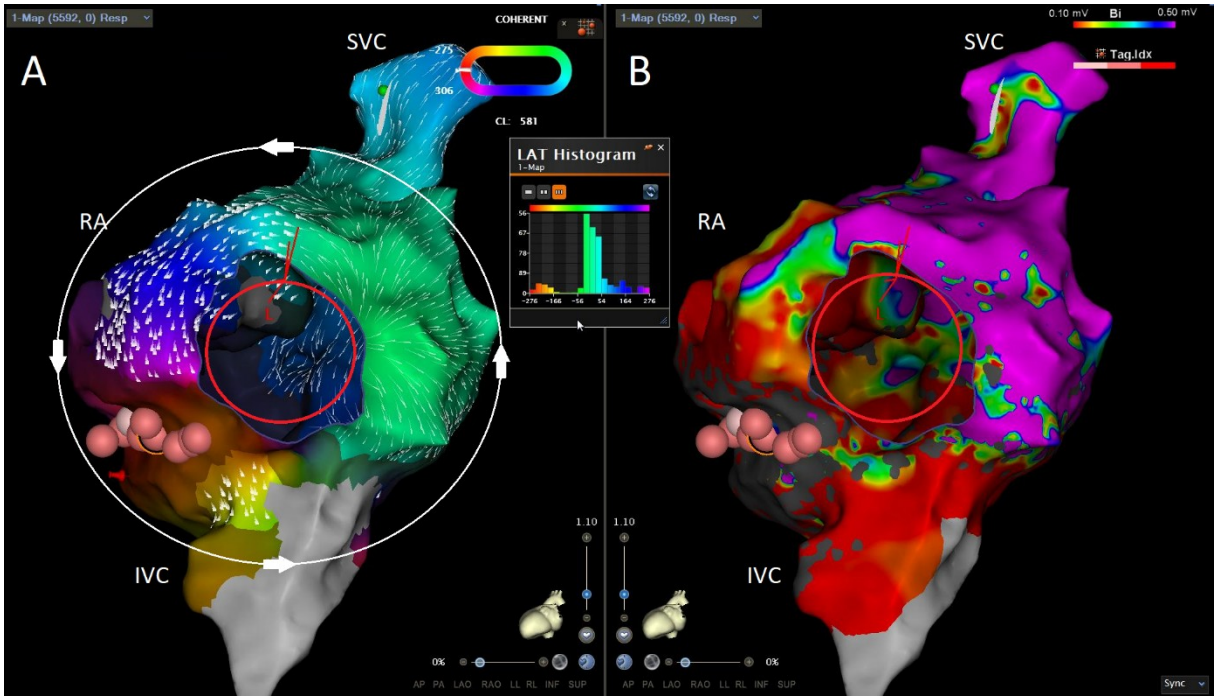

Figure 2. The electroanatomic map of the right atrium in the left oblique projection. In the activation map (**A**), the electrical excitation propagation waveform (white circle) revolves around the tricuspid valve annulus (red circle). On the potential map (**B**), red and gray mark the zones of slowed conduction of electrical excitation; IVC — inferior vena cava; RA — right atrium; SVC — superior vena cava

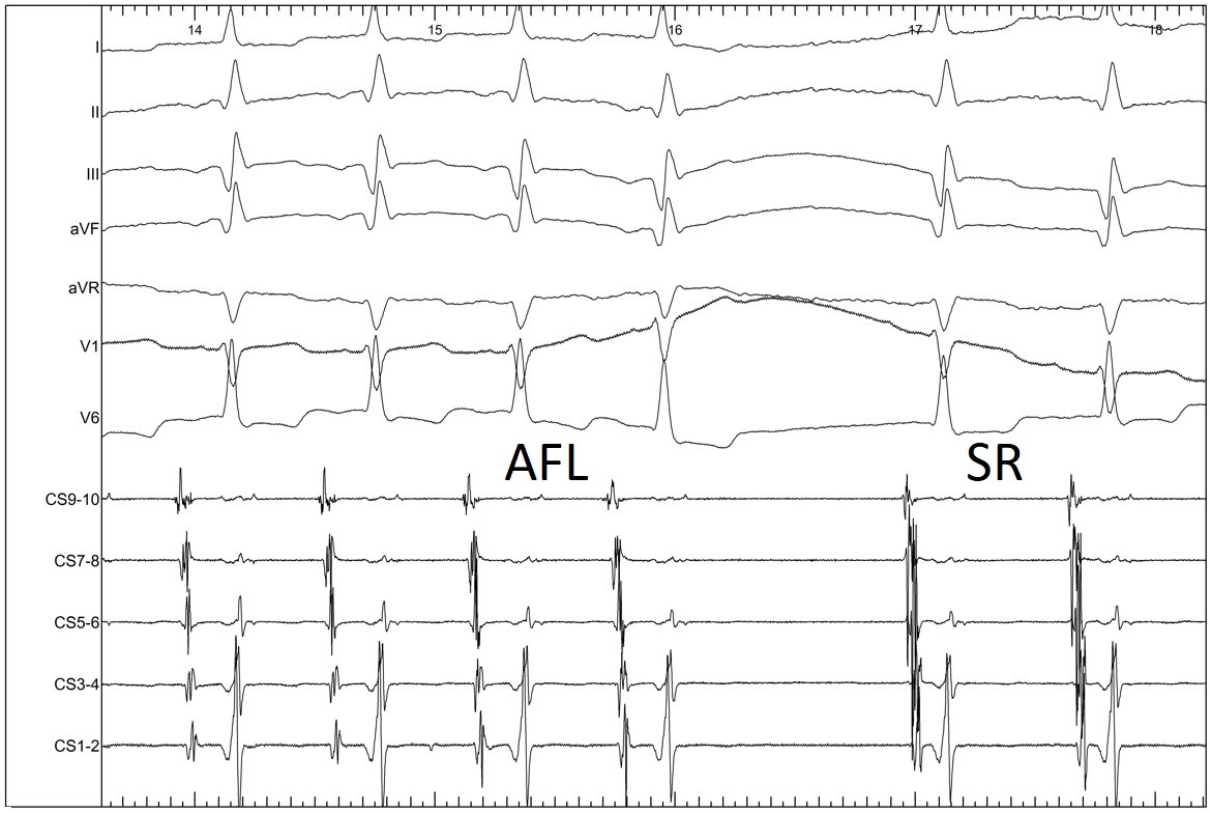

Figure 3. The surface and intracardiac electrocardiographic recordings from the coronary sinus. The recorded moment of termination of atrial flutter (AFL) and return of sinus rhythm (SR)

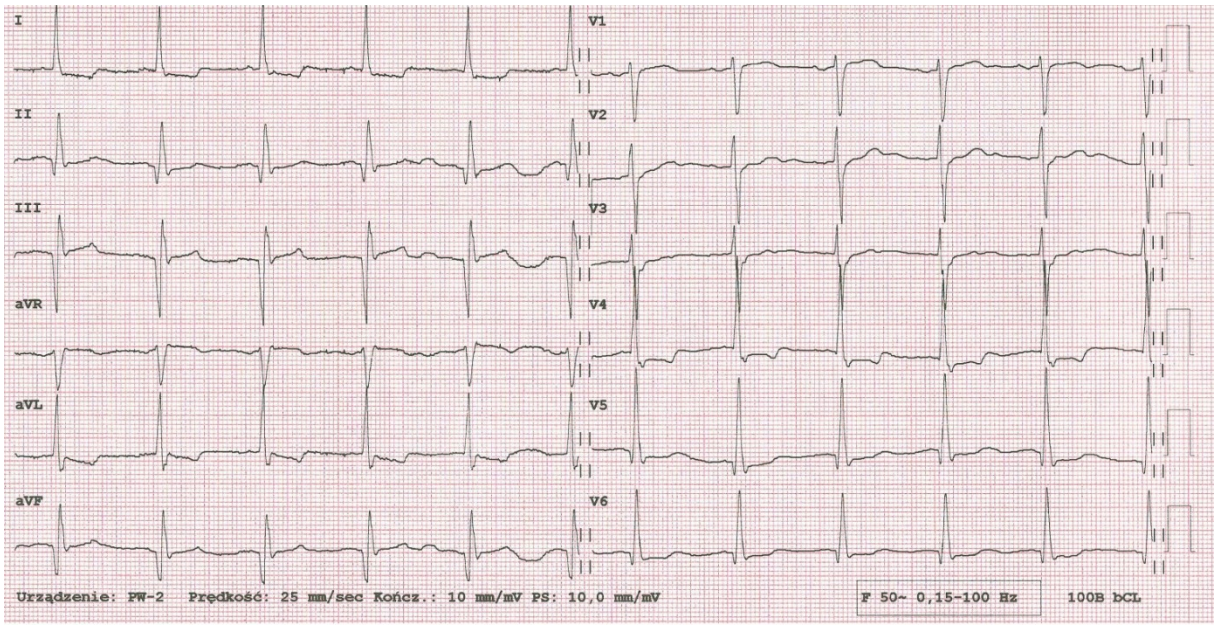

Figure 4. The 12-lead surface electrocardiographic recording. Sinus rhythm after ablation. Attention should be drawn to the low voltage P waves