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CASE REPORT
Arrhythmias / electrophysiology

Atrioventricular 2:1-conduction via an accessory pathway during left atrial flutter unmasking WPW syndrome: a case report

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Background Implantable cardioverter defibrillators (ICDs) are most effective in treating sudden cardiac death. However, accurate diagnostic workup of broad complex tachycardia is crucial to ensure correct indication for ICD treatment and to avoid unnecessary invasive treatment and device-associated morbidity.

Case summary We present a case of atypical atrial flutter with 2:1 atrioventricular (AV) conduction via a left-posterior accessory pathway (AP), leading to the diagnosis of Wolff-Parkinson-White (WPW) syndrome. Upon admission, the 72-year-old patient showed a regular broad complex tachycardia with superior axis and positive concordance in precordial leads, suggestive of either ventricular tachycardia (VT), antidromic AV re-entrant tachycardia (AVRT), or supraventricular tachycardia with antegrade conduction via a left-posterior AP. Interrogation of the two-chamber ICD, which was very likely implanted unjustified in a peripheral clinic before, revealed atrial flutter with 2:1 AV conduction. Remarkably, after the restoration of sinus rhythm, no classic echocardiogram (ECG) criteria for preexcitation syndrome were detected. An invasive electrophysiological study proved the diagnosis of a bidirectionally conducting, left-posterior AP, which was successfully ablated.

Discussion Differential diagnosis of broad complex tachycardia with superior axis and positive concordance of chest leads consists of i) VT with a left ventricular exit at the posterior mitral annulus, ii) antidromic AVRT involving a left-posterior AP, and iii) supraventricular tachycardia predominantly conducted via a left-posterior AP. The absence of classic ECG criteria for preexcitation syndrome does not rule out AP sufficiently, highlighting the importance of minimal surface-ECG preexcitation criteria. In the case of detection of minimal surface-ECG preexcitation criteria, administration of adenosine rules out or proves the existence of an AP noninvasively and cost-effectively.

Keywords WPW syndrome • Left-posterior accessory pathway • Atypical atrial flutter • 2:1 AV conduction • Broad complex tachycardia • Case report

ESC Curriculum 5.10 Implantable cardioverter defibrillators • 5.6 Ventricular arrhythmia • 5.5 Supraventricular tachycardia

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Learning points

- Differential diagnosis of a broad complex tachycardia with superior axis and positive concordance of chest leads consists of i) ventricular tachycardia (VT) with a left ventricular exit at the posterior mitral annulus, ii) an antidromic AV re-entrant tachycardia (AVRT) involving a left-posterior accessory pathway (AP), and iii) a supraventricular tachycardia predominantly conducted via a left-posterior AP.
- In case of ambiguities concerning a documented broad complex tachycardia, application of adenosine and/or performing an electrophysiological study should be indicated generously to rule out an AP and to differentiate VT from SVT.

Introduction

Implantable cardioverter defibrillators (ICDs) are the most effective treatment to prevent sudden cardiac death.[1] More than 50% of patients carrying an ICD for secondary prevention receive appropriate therapy for ventricular arrhythmias (VA) within five years.[2] However, given the perioperative risks and potential device-associated morbidity, establishing correct indication for ICD treatment is crucial. Not every broad complex tachycardia constitutes ventricular tachycardia (VT) and not all VTs require ICD implantation. Antidromic atrioventricular re-entrant tachycardia (AVRT) may mimic VT, and the majority of idiopathic VTs can be cured by ablation therapy. Recognition of a respective situation in the context of broad complex tachycardia may avoid unnecessary invasive treatment and morbidity.

Timeline

10/2015	Implantation of dual-chamber ICD for secondary prevention due to symptomatic broad complex tachycardia in the context of structural heart disease (ischaemic cardiomyopathy, septal-basal, and inferior-basal akinesia, LVEF 48%) in a peripheral hospital
2017–2018	Recurrent ICD therapies. Outpatient initiation of long-term therapy with amiodarone
05/11/2021	Persistent broad complex tachycardia. Admission to emergency department
	Interrogation of ICD. Diagnosis of atypical atrial flutter with 2:1 AV conduction. Suspected accessory pathway in the context of broad complex tachycardia
	Restoration of normal sinus rhythm (cardioversion). Discontinuation of amiodarone therapy
05/17/2021	Electrophysiological study. Diagnosis of a bidirectionally conducting, left-posterior accessory pathway
09/16/2021	Re-confirmation and ablation of left-posterior accessory pathway

Case presentation

A 72-year-old male Caucasian patient with a past medical history of ischaemic cardiomyopathy, carrying a two-chamber ICD for secondary prevention, was admitted to the emergency department due to persistent tachycardia. For several days, he had noticed an accelerated heartbeat. Associated symptoms such as angina pectoris or syncope were denied. Apart from a regular tachycardia, physical examination remained unremarkable. Since 2017 he was on long-term drug therapy with amiodarone (200 mg once daily) and bisoprolol (5 mg twice daily). The electrocardiogram (ECG) performed upon arrival revealed a regular broad complex tachycardia with a ventricular frequency of 117/min, superior axis, positive concordance in precordial leads (V1–V6) and a deflection time >100 ms in lead V1 compatible with VT or antidromic AVRT involving a left-posterior accessory pathway (AP). Remarkably, monophasic positive P-waves in leads V1–V6 with an atrial rate of ~235/min were registered, suggestive of atypical atrial flutter with 2:1 conduction. The ECG is displayed in *Figure 1A*. Interrogation of the ICD, which had been implanted in another hospital in 2015 for secondary prevention, clearly showed atrial flutter (cycle length, 260 ms) with 2:1 atrioventricular (AV) conduction (*Figure 1B*). Device settings are shown in Table 1. Electrical cardioversion (eCV) was performed successfully after atrial overstimulation remained ineffective. The ECG after eCV showed normal sinus rhythm (SR) with positive P-waves in inferior leads and biphasic P-waves (+/–) in lead V1, a PQ-interval of 160 ms, a narrow QRS complex in all 12 leads, and preterminal T-wave inversions in inferior leads (*Figure 2A*). The absence of initial positive deflection (septal r-wave) in lead aVR and absence of an initial negative deflection (septal q-wave) in lead V6 was observed.[3,4] A comparison of central ECG features of i) broad compex tachycardia and ii) normal SR is provided in the Supplementary material section. Echocardiography after the restoration of SR revealed a reduced left ventricular ejection fraction (LVEF) of 48% due to septal-basal and inferior-basal akinesia, which may additionally have triggered the misdiagnosis of this broad complex tachycardia as VT before. The electrophysiological study, performed in normal SR, revealed a bidirectionally conducting, left-posterior AP. Right ventricular (RV) pacing induced a C-shaped activation sequence of the 10-pole coronary sinus (CS) catheter (*Figure 2B*) and atrial pacing with decreasing cycle length led to increasing preexcitation of the QRS complex (*Figure 2C and D*); 12-lead ECG-morphology of the pre-excited QRS complex was identical to the broad complex tachycardia upon admission (*Figure 1A*). The refractory period of the AP was 360 ms in the context of long-term drug therapy with amiodarone and no administration of catecholamines. Importantly, subsequent re-evaluation of the broad complex tachycardia leading to ICD-implantation in 2015 showed i) irregular RR-intervals and discrete beat to beat variations of the QRS-complexes suggestive of a fast-broad-irregular (FBI) tachycardia and ii) a nearly identical QRS morphology compared to the pre-excited QRS complex induced

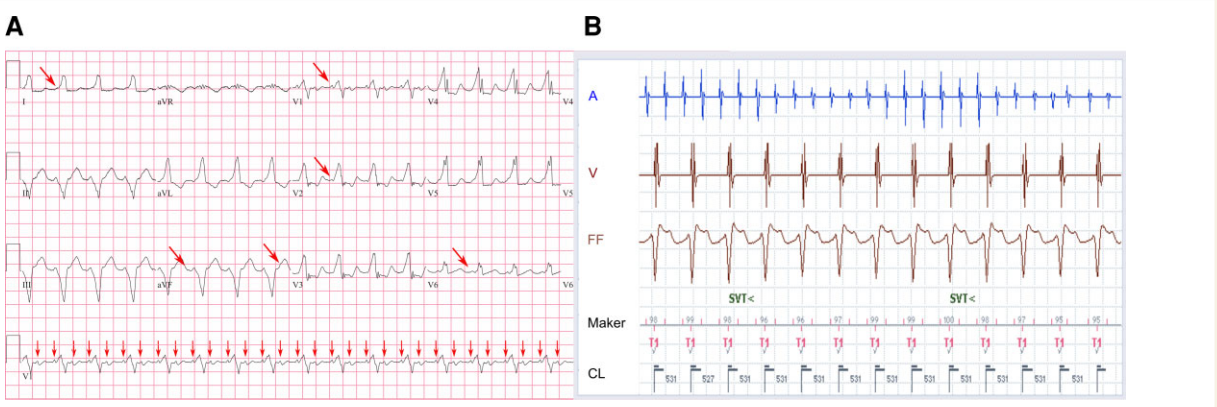

Figure 1 (*A*) Electrocardiogram (ECG) upon arrival: 25 mm/s, 10 mm/mV, and 100 Hz. Broad complex tachycardia, ventricular heart rate, 117/min; QRS interval, 170 ms, superior axis, positive concordance V1–V6, R-S-interval > 100 ms. T-wave inversion in aVL, biphasic T-waves in V2 and V3. Monophasic positive P-waves in leads V1–V6, atrial frequency ~235/min. (*B*) Interrogation of implantable cardioverter defibrillator. Intracardiac electrogram (EGM) determining atrial flutter (cycle length, 260 ms) with 2:1 AV conduction. Atrial EGMs (A), ventricular EGMs (V), far-field ECG, marker channels (marker), and cycle length.

during the electrophysiological study (*Figure 3A and B*). The patient underwent AP ablation. The left atrium was accessed via a transseptal approach. RV pacing revealed the earliest atrial activation at the posterior mitral annulus (CS 5/6). An electro-anatomical activation map was acquired during RV-stimulation, and the AP could be effectively ablated slightly septal from CS 5/6 as annotated (*Figure 4*). No recurrence of the AP was registered during the 30-minute waiting time after ablation. Amiodarone therapy was stopped. As the underlying electrograms (EGMs) of ICD therapies in 2017 and 2018 were deleted beforehand, retrospective differentiation of supraventricular tachycardia (SVT) involving the AP (atrial fibrillation, atrial flutter) or antidromic AVRT from VT was not possible anymore. Therefore, and in the context of structural heart disease (ischaemic cardiomyopathy, LVEF 48%, septal-basal and inferior-basal akinesia), the ICD was left in situ and the patient was discharged. Three months after catheter ablation the patient was examined routinely in the outpatient clinic. He reported a good physical state and denied any form of palpitations, and device interrogation revealed no arrhythmias since catheter ablation.

Table 1 Device settings of dual-chamber implantable cardioverter defibrillator. St. Jude Medical. Ellipse™ DR 2377-36QC implantable cardioverter defibrillator

Pacing mode	DDD		
Lower rate	60 bppm		
Upper rate	120 bpm		
Atrial output (bipolar)	1.5 V/0.5 ms		
Atrial sensitivity (bipolar)	Auto		
RV output (bipolar)	1.0 V (auto)/0.5 ms		
RV sensitivity (bipolar)	Auto		
Zone	VT1	VT2	VF
Rate (bpm/ms)	162/370	181/330	230/260
Detection intervals	18	16	16
Therapy	Monitor	ATP + shocks	ATP + shocks
Discrimination algorithm (dual chamber)	A:V ratio, onset, stability, morphology		

Discussion

Classic surface-ECG criteria for APs comprise i) shortened PR interval, ii) slurred upstroke of the QRS complex (delta wave), and iii) prolonged QRS duration. These ECG-changes occur due to the fusion of simultaneous activation of the ventricles via the AP and via the AV node. The varying degree of preexcitation, including no apparent preexcitation, depends on both the anatomical localization and the conduction properties of the AP as well as the conduction properties of the AV node and the His-Purkinje-system.

In our case, no classic ECG criteria for preexcitation syndrome were present in SR, whereas maximal preexcitation occurred during atypical atrial flutter. The pathophysiologic explanation for this phenomenon lies in i) primary activation of the left atrium during left atrial flutter/left atrial focal tachycardia and ii) the refractoriness and delayed conduction of the AV node when exposed to high atrial frequencies. During normal SR, primary excitation occurs at the high right atrium, resulting in comparatively late excitation of a left-sided AP. However, primary left atrial excitation represents a temporal conduction advantage of a left-sided AP over the AV node, resulting in more prominent preexcitation. Furthermore, the atrial rate of ~235/min induces increasing refractoriness of the AV node, especially in the context of co-medication with beta-blockers, so that AP-conduction becomes more prominent.

Differential diagnosis of broad complex tachycardia with superior axis and positive concordance of chest leads—as observed in our case—consists of VT with a left ventricular exit at the posterior mitral annulus, an antidromic AVRT involving a left-posterior AP as well as an SVT conducted via a left-posterior AP. Detection of an AP in a

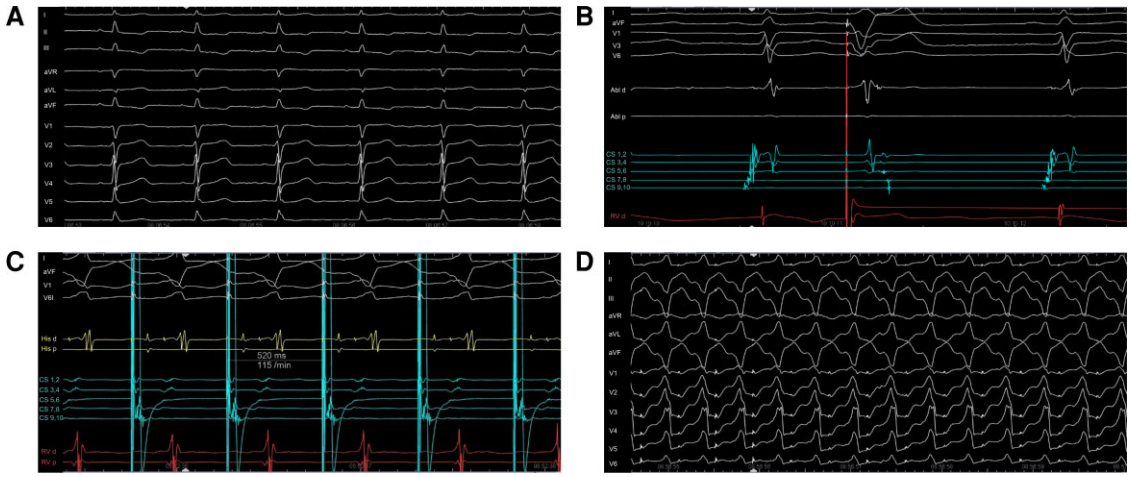

Figure 2 (*A*) ECG after electrical cardioversion: 50 mm/s, 10 mm/mV, and 100 Hz. Sinus rhythm, heart rate 74/min, normal axis, PR-interval 160 ms, P-waves with inferior axis (II, III, and aVF), biphasic P-wave (+/–) in V1, QRS-duration 106 ms, and R-wave transition V3/V4. Preterminal T-wave inversion in II, III, and aVF. QT 450, QTc 503 ms. Red arrows indicate P-waves. (*B*) Right ventricular pacing inducing a C-shaped activation sequence of the 10-pole CS catheter. Surface ECG and intracardiac electrograms (EGMs) registered at 100 mm/s. (*C*) Atrial pacing with decreasing cycle length leading to increasing preexcitation of the QRS complex. Surface ECG and intracardiac EGMs registered at 100 mm/s. (*D*) 12 lead surface ECG (50 mm/s, 10 mm/mV) during atrial pacing leading to maximum preexcitation of QRS complex.

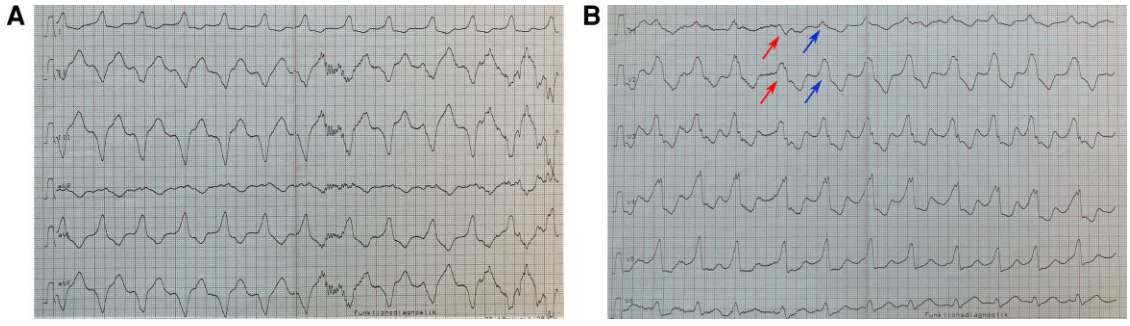

Figure 3 ECG of broad complex tachycardia recorded at an external clinic in 2015 prior to implantation of implantable cardioverter defibrillator: 50 mm/s, 10 mm/mV, and 100 Hz. Ventricular heart rate, 147/min; QRS interval, 162 ms. Superior axis, positive concordance V1–V6, superior axis, positive concordance V1–V6. (*A*) Limb leads. (*B*) Chest leads V1–V6. Red and blue arrows displaying QRS-complexes with discrete variations of QRS morphology.

respective situation is particularly important as ICD-implantation is not indicated and catheter ablation offers a curative treatment.[5]

The absence of classic preexcitation ECG criteria does not rule out AP. A retrospective analysis of 238 patients with successfully ablated APs revealed that 10–15% of the study population showed a PR interval >120 ms respectively no obvious delta wave and a narrow QRS complex.[3] This is especially true for left-lateral APs as atrial activation in this area is late, and thus, ventricular preexcitation is often limited. Of note, however, additional ECG criteria exist to identify minimal preexcitation:[3,4] In case of a left-sided, non-septal, antegrade conducting AP, initial ventricular excitation via the AP precedes septal activation via the AV node and thus obscures the physiological r-wave in lead aVR and q-wave in lead V6. The absence of the physiological r-wave (aVR) and q-wave (V6) therefore depicts minimal ECG preexcitation criteria in patients with a suspected AP (*Figure 2A*).[3,4]

Nonetheless, an AP cannot be ruled out with certainty based on the surface-ECG. Therefore, if intermittent preexcitation is suspected or minimal preexcitation criteria are detected, administration of a selective AV node blocking agent such as adenosine helps to rule out or prove an antegrade conducting AP noninvasively. In any case of persisting ambiguities concerning a documented broad complex tachycardia, an electrophysiological study should be performed to differentiate VT from SVT before considering ICD implantation.

Misinterpretation of maximum preexcitation as VT exposes the patient to unnecessary invasive treatment and in the case of ICD-implantation to potential short- and long-term device-

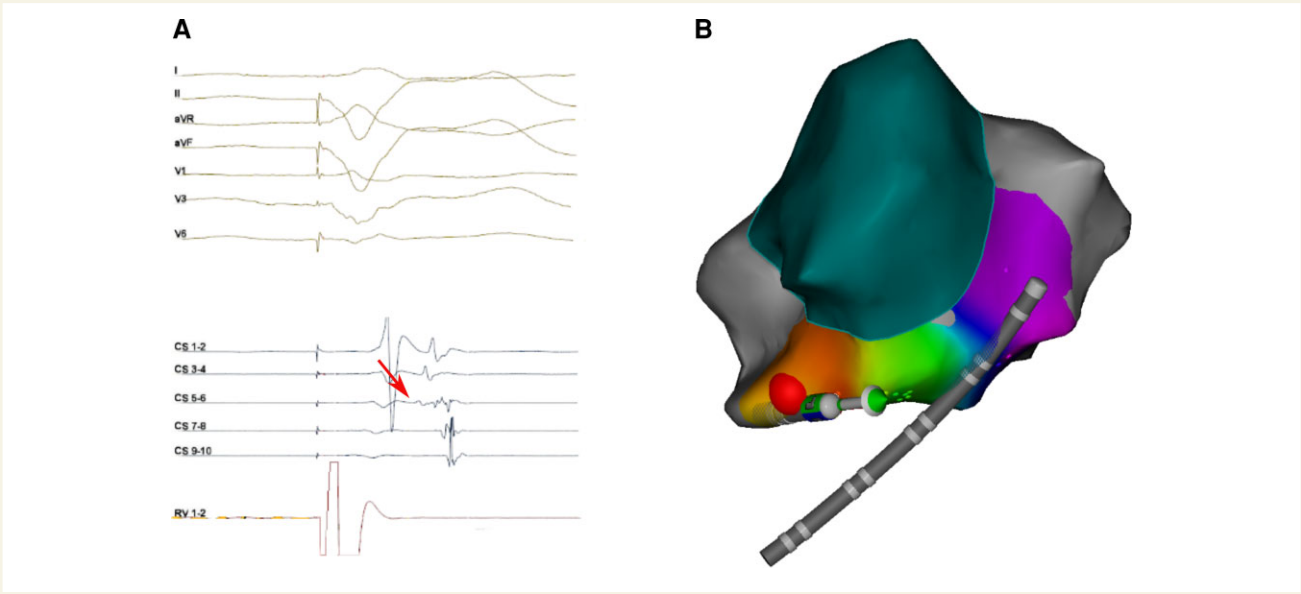

Figure 4 (*A*) Surface ECG and intracardiac ECG at site of successful ablation of the accessory pathway during right ventricular pacing. (*B*) Electro-anatomical map (local activation time) displaying the ablation—as well as coronary sinus catheter and visualizing the site of ablation of the accessory pathway (red point).

associated complications. Hence, every patient without structural heart disease and incorrect indication for ICD treatment should be evaluated for ICD removal following successful AP ablation. However, an ICD should be left in situ in patients with either documented additional secondary prophylactic indication or structural heart disease and primary prophylactic indication for ICD treatment. In the context of structural heart disease, not fulfilling the criteria for primary prophylactic ICD implantation, the chances and risks of ICD explanation versus maintaining ICD therapy should be discussed with the individual patient. Here, the time interval since the leads have been implanted, the likelihood for further progression of structural heart disease and the morbidity of the patient must be considered.

Lead author biography

After studies at the universities of Tuebingen, Bristol and Brown Medical School, M. T. Huttelmaier graduated from medical school at the University of Tübingen in 2013 and completed training in internal medicine (University Clinic Wuerzburg) in 2019. Currently, he is working as a fellow of interventional electrophysiology in the department of internal medicine at the University Clinic of Wuerzburg.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

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Anonymized.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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