CARDIAC ARRHYTHMIA SPOT LIGHT

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Unmasking latent preexcitation of a right-sided accessory pathway with intravenous adenosine after unexplained sudden cardiac arrest.

Fang Shawn Foo MBChB¹ | Martin K. Stiles MBChB, PhD^{1,2} | David Heaven MBChB^{3,4}

¹Department of Cardiology, Waikato Hospital, Hamilton, New Zealand ²Department of Medicine, University of Auckland, Auckland, New Zealand ³Department of Cardiology, Middlemore Hospital, Auckland, New Zealand ⁴Green Lane Cardiovascular Service, Auckland City Hospital, Auckland, New Zealand

Correspondence

Fang Shawn Foo, Department of Cardiology, Waikato District Health Board, Pembroke Street, Hamilton 3240, New Zealand. Email: shawnfoo@icloud.com

Wolff-Parkinson-White syndrome is a rare cause of sudden cardiac death, when atrial fibrillation conducting rapidly over an accessory pathway (AP) degenerates into ventricular fibrillation. However, manifest preexcitation is not always present on serial electrocardiograms (ECG). "Latent preexcitation" has been used to describe the absence of ECG evidence of preexcitation of an AP during sinus rhythm due to a number of causes. This is opposed to intermittent preexcitation, when there is true intermittent antegrade conduction through the AP. Intravenous adenosine is a simple diagnostic test that can uncover latent preexcitation via an AP.

We present the case of a 17-year old male with a cardiac arrest due to ventricular fibrillation, necessitating resuscitation with multiple shocks and adrenaline. He later reported recurrent palpitations with presyncope for a year but was otherwise well without comorbidities or family history of sudden cardiac death. Investigations including transthoracic echocardiogram, cardiac magnetic resonance imaging and computed tomography showed a structurally normal heart with normal coronary origins. Initial review of ECGs in hospital were thought to be benign. There was no evidence of preexcitation or tachyarrhythmias on telemetry. Ajmaline challenge and exercise tolerance test did not show any evidence of Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia or long QT syndrome. An intravenous adenosine test (6mg) revealed manifest preexcitation without PR shortening, suggestive of a right posterior AP with slow antegrade conduction through the pathway (Figure 1). In retrospect, subtle evidence of manifest preexcitation was discovered on two of more than 20 ECGs.

An invasive electrophysiology study showed no evidence of preexcitation at baseline (H-V = 37 milliseconds). However, there was manifest preexcitation with coronary sinus (CS) pacing (H-V = -55 milliseconds). There was evidence of bidirectional AP conduction with a short antegrade effective refractory period (ERP) (<260 milliseconds at baseline, <240 milliseconds with low dose isoprenaline) as well as retrograde ERP (<300 milliseconds) via the pathway. There was minimal antegrade or retrograde decremental conduction, suggesting a "typical" AP rather than a nodo-ventricular or nodo-fascicular pathway. The AP location was mapped with an irrigated ablation catheter (Biosense Webster D-curve) during proximal CS pacing. The earliest ventricular activation was 35 milliseconds ahead of the delta wave at the right posterior location on the tricuspid annulus where the local A-V time was 85 milliseconds in sinus rhythm and 35 milliseconds with CS pacing (Figure 2). With initial radiofrequency (RF) application there was transient loss of pathway conduction but with a second application the pathway conduction was eliminated at 5.5 seconds. Total RF time: 2.7 minutes at 35 W; number of RF lesions: 3; fluoroscopy time: 9.3 minutes. There was no evidence of antegrade or retrograde AP conduction at 30 minutes post-ablation. Atrioventricular nodal ERP was 400 milliseconds antegradely and 340 milliseconds retrogradely. Parahisian pacing showed stimulus to atrial time of 117 milliseconds during ventricular-only capture and 65 milliseconds during His-only capture. An adenosine test the following day and at 8 weeks post-ablation showed no ECG evidence of preexcitation. Eighteen months following the ablation, he remains asymptomatic with no ECG evidence of preexcitation.

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FIGURE 1 Latent preexcitation without PR shortening unmasked with 6mg intravenous adenosine, suggesting a right posterior accessory pathway with slow antegrade conduction through the pathway.

This case illustrates the utility of intravenous adenosine in uncovering latent preexcitation of a previously unrecognized right posterior AP in a patient resuscitated from unexplained sudden cardiac arrest. Successful ablation of the AP negated the need for a lifelong implantable cardioverter-defibrillator. APs with latent preexcitation are typically located on the left free wall, due to the distance from the sinus node rendering the pathway "concealed" during sinus rhythm. In our patient, the AP was in the right posterior position, but there was longer local A-V time in sinus rhythm than with CS pacing, suggesting an oblique course through the atrioventricular groove. This would have contributed to the absence of manifest preexcitation in sinus rhythm despite being a right-sided AP. To the authors' knowledge, this is the second reported case of latent preexcitation in a typical right-sided AP (excluding nodo-ventricular APs), but the first reported case to have resulted in a cardiac arrest.

Intermittent ECG evidence of preexcitation is generally considered a marker of low, but not absent, risk for sudden death, with only rare exceptions reported. However, latent or subtle preexcitation does not correlate with the antegrade conduction capacity of an AP, which may still be malignant and explains the cause of sudden cardiac arrest in our patient. High dose isoprenaline or induction of atrial fibrillation/ ventricular arrhythmias was not performed at the operator's discretion, primarily due to the limited number of clinical staff. An electrophysiology study with a view to ablation is recommended in patients with a diagnosis of Wolff-Parkinson-White syndrome resuscitated from a sudden cardiac arrest. However, in patients without manifest preexcitation on ECG, international guidelines do not specifically recommend the use of intravenous adenosine to uncover latent preexcitation. In New Zealand, our local heart rhythm society and cardiac inherited disease group have incorporated adenosine testing into our investigation of resuscitated sudden cardiac death. However, we are aware that this diagnostic test is performed variably internationally. Without the use of adenosine to unmask the AP in this particular case, our patient might have received an implantable cardioverter defibrillator, with the associated peri-procedural and long-term issues of a defibrillator implant at such a young age.

In conclusion, intravenous adenosine is a simple test that can uncover latent preexcitation via an AP and is useful in the diagnostic workup of sudden cardiac arrest survivors without an identifiable cause. Latent preexcitation is usually associated with left free wall pathways but may also occur in right-sided APs with slow antegrade conduction. Although intermittent ECG evidence of preexcitation is generally considered a marker of low risk for sudden death, if this is due to an AP with long AV conduction time and short ERP (rather than true intermittent conduction via the pathway), the FIGURE 2 Local A-V time of accessory pathway with ablation catheter (A) during sinus rhythm, A-V=85ms; (B) during coronary sinus pacing, A-V = 35 ms. Schematic representation of right posterior accessory pathway with oblique course through the atrioventricular groove (tricuspid and mitral valve annuli in left anterior oblique view), illustrating the change in local A-V time with change of atrial depolarisation wavefront in sinus rhythm and coronary sinus pacing. This would contribute to the absence of manifest preexcitation in sinus rhythm. ABL, ablation; AP, accessory pathway; AV, atrioventricular; CS, coronary sinus; RV, right ventricle.



risk for sudden death may be equal to those APs with manifest preexcitation.

CONFLICT OF INTEREST

The authors declare no conflict of interests for this article.

ORCID

Fang Shawn Foo D https://orcid.org/0000-0002-3546-4863

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