

An unusual property of the fasciculoventricular pathway with conduction block



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Introduction

Fasciculoventricular pathways (FVPs) are known to be rare variants of pre-excitation syndrome without any observed clinical tachycardia. They are accessory connections from the bundle of His or the fascicles (Figure 1A), inserted into the ventricular myocardium, and they conduct whenever the bundle of His is engaged.¹ Patients with FVPs exhibit ventricular pre-excitation on electrocardiogram (ECG) but do not experience reciprocating tachycardias or sudden cardiac death. This is unlike Wolff-Parkinson-White syndrome when individuals with electrocardiographic pre-excitation experience symptoms like palpitations or presyncope.¹ ECG characteristics of an FVP consist of a fixed, minimal pre-excitation pattern with a normal QRS frontal plane axis, and the degree of pre-excitation does not change even with variable PR interval. Conduction through the bundle of His with a block at the FVP has not been reported.²

Case report

A 42-year-old male patient with a long-standing history of palpitations, Wolff-Parkinson-White syndrome, and supraventricular tachycardia was referred after multiple unsuccessful ablation attempts. He also had a history of atrial fibrillation with an embolic stroke to the occipital area, without significant deficit. After the patient had been seen and examined, he underwent a comprehensive electrophysiological study with catheter locations as shown in Figure 1B.

The surface ECG revealed minimal ventricular pre-excitation in lead V₂ (Figure 1C), and during the study, an HV interval of 45 ms (Figure 2A) was noted. Baseline intervals were recorded as follows: PR, 147 ms; QRS, 92 ms; QT, 36 ms; AH, 67 ms; HV, 45 ms. Differential pacing from multiple locations consistently showed a fixed pre-excitation and a fixed HV interval. During atrial decremental pacing, the AH interval increased without significant changes in pre-

KEY TEACHING POINTS

- Fasciculoventricular pathways are very uncommon accessory pathways connecting the bundle of His or the fascicles, inserted into the ventricular myocardium with minimal pre-excitation on the electrocardiogram.
- Fasciculoventricular pathways do not give rise to reciprocating tachycardia or clinical tachycardia and hence, ablation of the pathway is not indicated.
- The conduction block in the fasciculoventricular pathway has never been described before.

excitation or HV interval. However, with further pacing reduction, pre-excitation decreased. At an atrioventricular (AV) Wenckebach cycle length of 250, the beat before the AV block showed no pre-excitation, and minimal pre-excitation returned after the conduction block without HV interval variation (Figure 2B). During adenosine-induced AV block, there was no AV conduction (Figure 3A).

Post adenosine administration, following the AV block, a premature atrial beat from the region of the superior atrium initiated atrial fibrillation (Figure 3B). Pre-excited R-R intervals had the longest R-R interval of 500 ms during atrial fibrillation, consistently displaying pre-excitation with a fixed HV interval regardless of the preceding R-R interval. The patient developed a left bundle branch block (LBBB) aberrancy with hemodynamic instability requiring synchronized cardioversion. Interestingly, the hemodynamic instability induced by LBBB had an HV interval of 75 ms, which was observed every time the patient had atrial fibrillation with LBBB aberrancy.

Ventricular-atrial (VA) conduction was evaluated using ventricular decremental pacing, revealing a concentric atrial activation pattern. Retrograde conduction was all AV nodal as suggested by VA increase with VH increase without HA change. The ventricular effective refractory period was determined to be 250 ms with a drive train duration of 600 ms. Intravenous administration of adenosine induced VA block, triggering atrial fibrillation with a premature atrial complex and earliest local activation in the superior atrium.

KEYWORDS Fasciculoventricular pathway; Conduction block; Atrial fibrillation; Wolff-Parkinson-White syndrome; Ventricular pre-excitation (Heart Rhythm Case Reports 2024;10:29–32)

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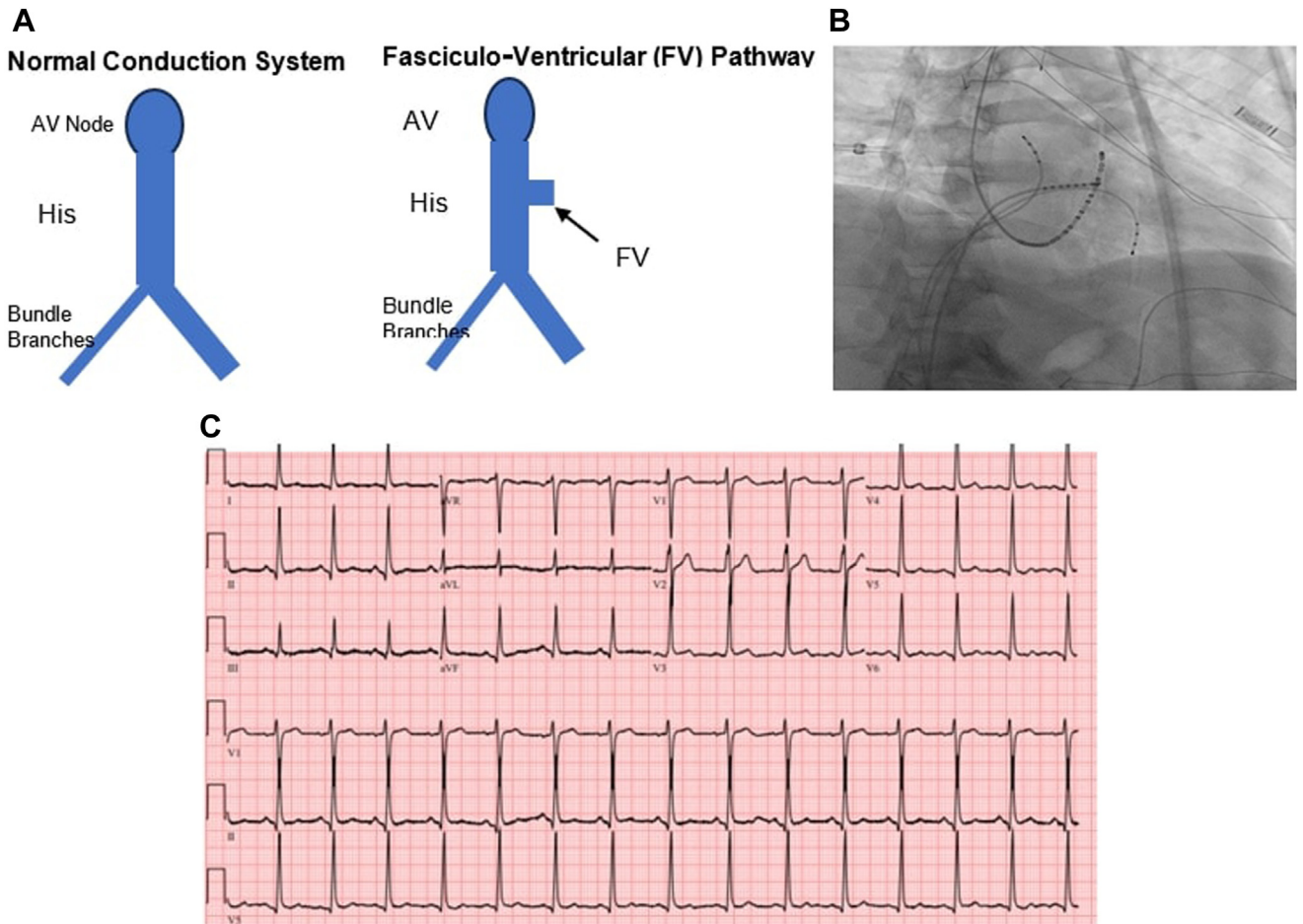


Figure 1 A: Diagram of the proposed location of the fasciculoventricular (FV) pathway with respect to the atrioventricular (AV) node. B: Fluoroscopy image showing location of catheters. C: Electrocardiogram depicting FV pathway.

Repeated adenosine administration was required to trigger atrial fibrillation, and mapping showed that the right superior pulmonary vein premature atrial contractions (PAC) triggered atrial fibrillation. Right-sided wide area circumferential ablation was initially performed. Subsequently, left-sided wide area circumferential ablation was performed owing to

adenosine-triggered PACs inducing nonsustained atrial tachycardia (without sustained atrial fibrillation) from the left superior pulmonary vein.

After completion of the ablation sets, adenosine 24 mg administration did not induce atrial fibrillation on 3 occasions. The patient is symptom free 7 months postablation,

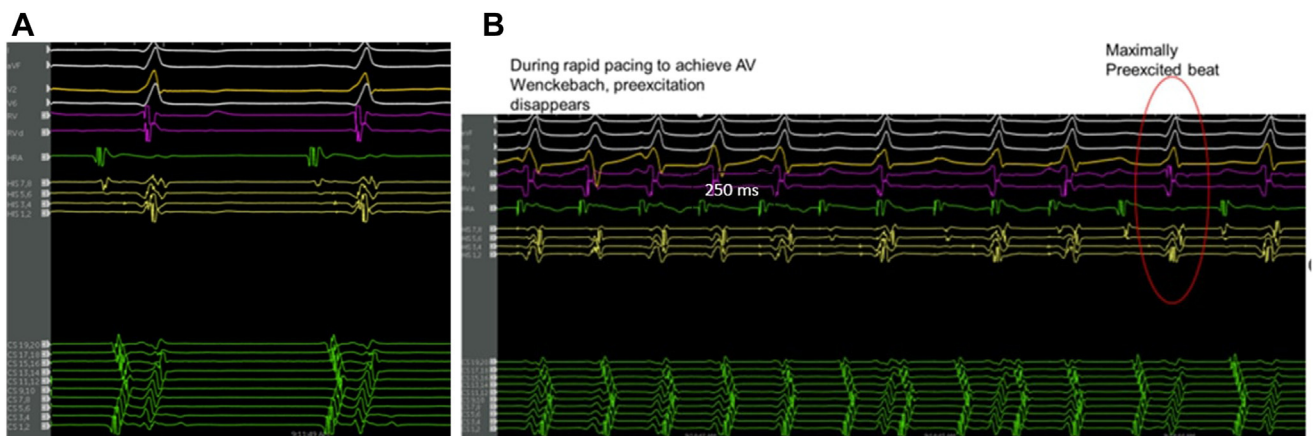


Figure 2 A: Surface electrocardiogram demonstrating pre-excitation (most prominent in lead V₂) with intracardiac recording demonstrating HV interval (measured 45 ms). B: Decremental pacing from high right atrium at the Wenckebach cycle length demonstrating no pre-excitation just before the atrioventricular block along with return of minimal pre-excitation in the subsequent beat without HV interval variation.

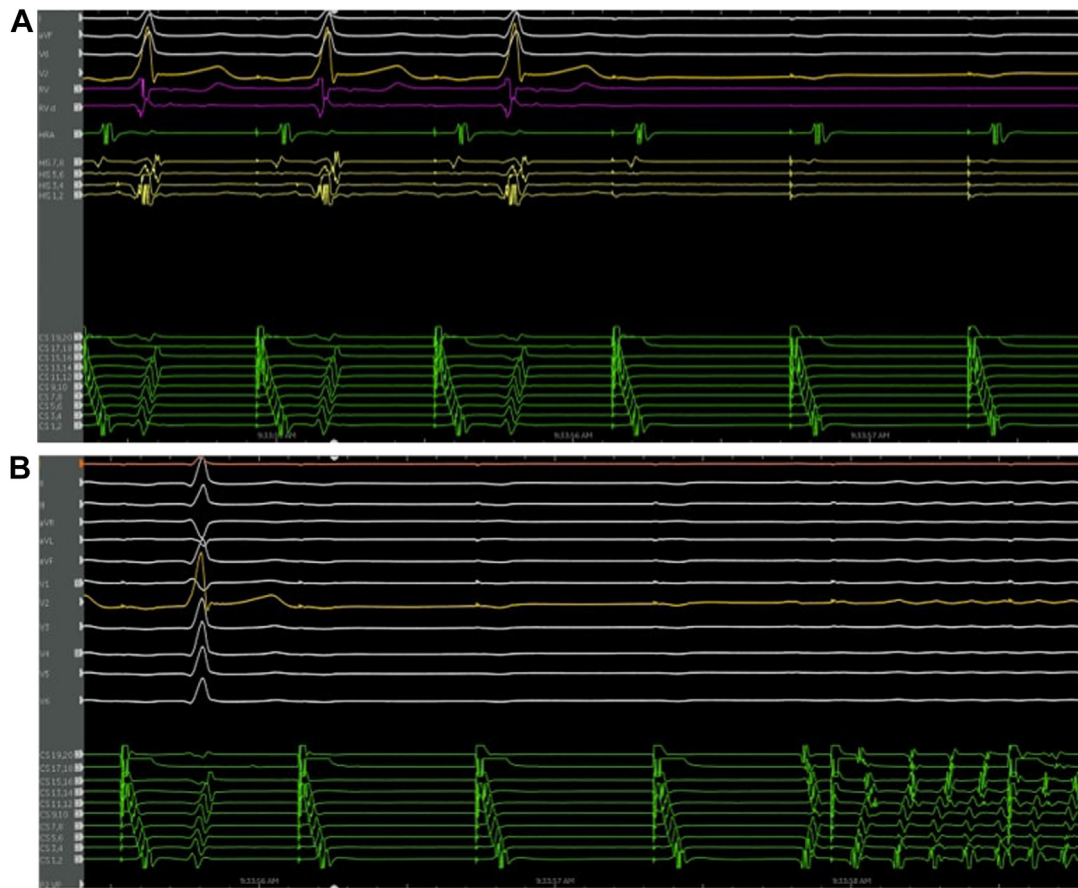


Figure 3 A: Suprahisian atrioventricular block after adenosine administration demonstrating loss of pre-excitation, implying that the pre-excitation is beyond the His. B: Atrioventricular block post-adenosine administration followed by a premature atrial beat and initiation of atrial fibrillation.

and extended monitoring for 30 days at 6 months did not reveal any arrhythmia.

Discussion

FVPs are the least common form of pre-excitation, occurring in only 1.2%–5.1% of all pre-excitation cases. These pathways connect the His bundle or bundle branches to the ventricular septum.³ In a conventional accessory pathway, the presence of AV nodal Wenckebach is not detected, since it is concealed by conduction through the accessory pathway. Similarly, the absence of blocked P waves occurs owing to the availability of an alternative pathway for ventricular conduction.¹ FVPs are distinguished by a small delta wave, a normal AH interval, and a shortened HV interval during sinus rhythm.³ In this case, differential pacing from multiple locations during the electrophysiological study consistently showed a fixed pre-excitation, suggesting the presence of an FVP. It is important to note that the degree of pre-excitation remains unaffected by multisite or incremental atrial pacing.³

In this case, the novel finding was the cycle length-dependent change in pre-excitation with a lack of discernible pre-excitation at shorter cycles and recovery of observable pre-excitation at slower cycle lengths. The induced tachycardia is unrelated to the pre-excitation itself.

No evidence suggests that FVPs are involved in supraventricular tachycardias resulting from a reentrant circuit. When other supraventricular tachycardias occur with normal AV node function, FVPs exist as passive bystanders without any increased risk of sudden cardiac death associated with their presence.¹ The FVP is anatomically connected to the His bundle and consistently conducts the impulse when it engages the His bundle.

In our case, intravenous adenosine was administered to confirm the location of the accessory pathway further, resulting in a complete heart block. This suggests that the pre-excitation extends beyond the level of the AV node and is within the conduction system rather than at the annulus with independent atrial and ventricular insertion, characteristic of an accessory pathway. To our knowledge,

there have been no reported cases where the His bundle conducts, yet the impulse loses pre-excitation owing to a block in the FVP. This unique finding was observed in our case.

Conclusion

FVPs represent the rarest form of pre-excitation, establishing a connection between the His bundle or bundle branches and the ventricular septum. This case highlights the uncommon conduction block and physiological features of an FVP.

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