Wolff-Parkinson-White pattern in the setting of antibody-mediated rejection after heart transplant

Luke Chong, MD,* Aneeq Waqar, MBBS,* Ahmed Elkhouly, MBBch,*
Joshua Hermsen, MD,† Maryl Johnson, MD,* Jennifer Wright, MD, FHRS*

*From the *Division of Cardiovascular Medicine, University of Wisconsin Hospital and Clinics, Madison, Wisconsin, and †Division of Cardiothoracic Surgery, University of Wisconsin Hospital and Clinics, Madison, Wisconsin.*

Introduction

Donor heart availability is a major issue that affects heart transplantation worldwide. Inclusion of hearts with minor or treatable conditions expands the pool of available organs. Hearts with known Wolff-Parkinson-White (WPW) syndrome are typically excluded from transplantation. Case reports where WPW syndrome is discovered posttransplant have been reported, and many donor heart recipients ultimately undergo postoperative electrophysiology study (EPS) and ablation. We present a case of manifest preexcitation developing in the setting of antibody-mediated rejection in a patient whose donor heart had no known history of WPW.

Case report

A 40-year-old woman with postpartum cardiomyopathy and no prior arrhythmic history underwent a bicaval orthotopic transplant with a heart procured via normothermic regional perfusion technique in a non-brain-dead donor who suffered a traumatic fall. The recipient's postoperative course was complicated by delayed graft function and antibody-mediated rejection. Two weeks after transplantation, her electrocardiogram (ECG) demonstrated preexcitation (Figure 1A); this was seen on neither her posttransplant ECG (Figure 1B) nor the last known ECG obtained from her donor. Short paroxysms of atrial tachycardia with preexcitation were noted on telemetry during her hospitalization but no atrial fibrillation was observed. Following discussion with the patient and the advanced heart failure service, she was referred for EPS after completing a 2-week course of plasmapheresis for management of antibody-mediated rejection.

KEY TEACHING POINTS

- The emergence of new preexcitation on electrocardiogram following orthotopic heart transplantation warrants careful consideration, especially if the circumstances surrounding the donor's death are unclear.
- Features for stratifying high-risk accessory pathways (AP) during electrophysiology study (EPS) include shortest preexcited R-R interval <250 ms in atrial fibrillation, shortest paced cycle length with preexcitation during atrial pacing ≤ 250 ms, and AP effective refractory period ≤ 250 ms.
- In cases where new preexcitation is discovered following heart transplantation, EPS and AP ablation can be considered at a clinically appropriate time prior to postoperative discharge. Given that significant arrhythmias or sudden cardiac death have been reported in patients without high-risk EPS features, ablation should be considered based on the anatomic location of the AP.

During EPS, atrial pacing with extrastimuli demonstrated loss of preexcitation with an accessory pathway effective refractory period (APERP) recorded at 600/300 ms (Figure 2A). With decremental atrial pacing, loss of preexcitation was seen at 290 ms, and atrioventricular (AV) nodal conduction thereafter was decremental with an AV nodal Wenckebach cycle length of 260 ms. There was no apparent retrograde conduction via the accessory pathway (AP), as ventricular pacing demonstrated that ventriculoatrial conduction was decremental, and atrial activation was noted to be concentric (septum first), consistent with retrograde conduction via the AV node. Atrial pacing was performed to obtain maximal preexcitation to facilitate mapping for the

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Address reprint requests and correspondence: Dr Luke Chong, University of Wisconsin Hospital and Clinics, 600 Highland Ave, Division of Cardiovascular Medicine, CSC H4/583, Madison, WI 53792. E-mail address: luke.y.chong@gmail.com.

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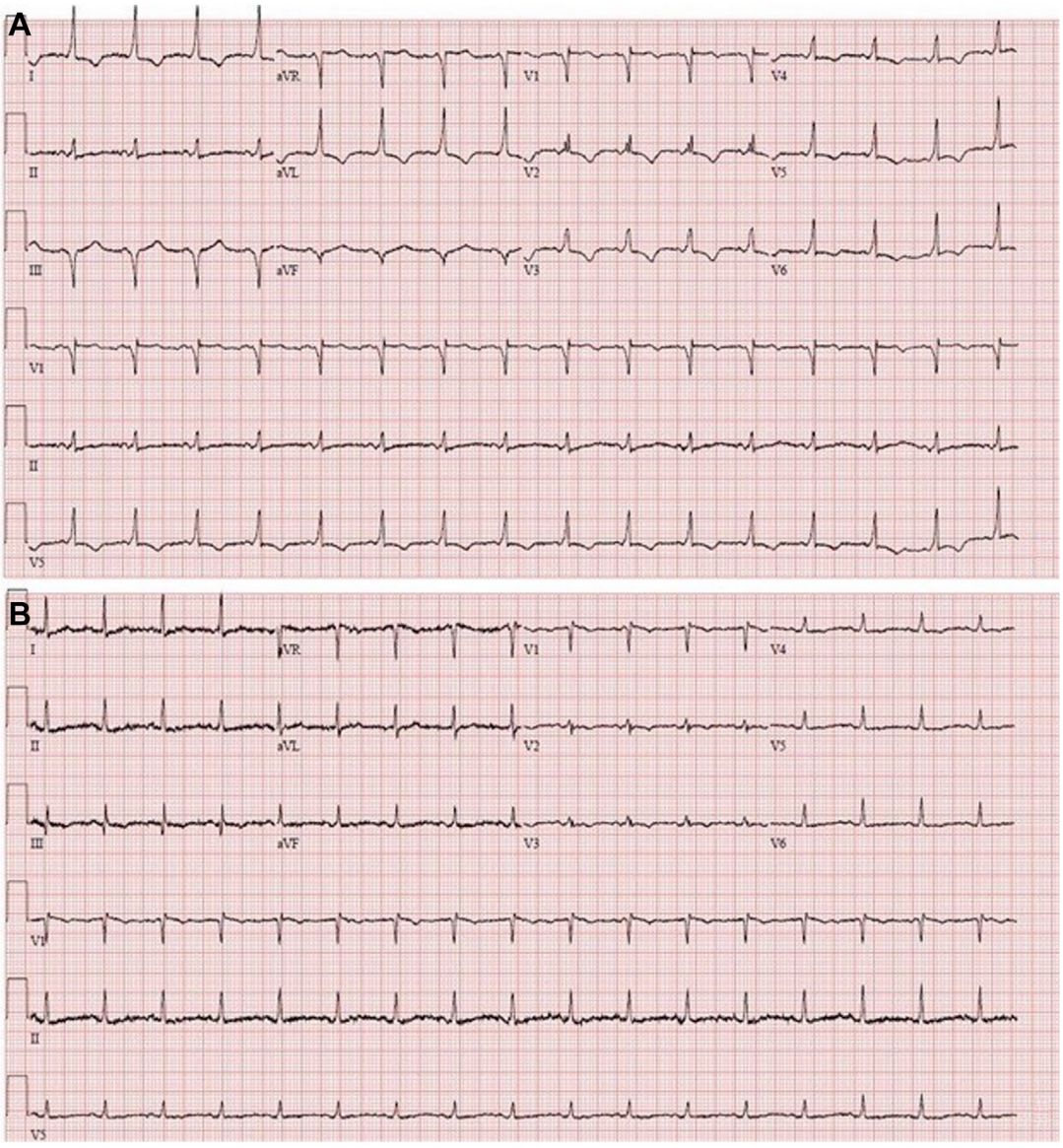

Figure 1 **A:** Electrocardiogram (ECG) 2 weeks posttransplant demonstrating preexcitation. **B:** ECG 1 week posttransplant showing sinus tachycardia with narrow QRS complexes.

ventricular insertion of the AP. Mapping around the triangle of Koch in the vicinity of the slow pathway and the coronary sinus os demonstrated that the site of earliest activation was located at the middle third of the anterior lip of the coronary sinus os, consistent with a posteroseptal AP. Locations with pathway potentials were annotated as putative sites for ablation. A series of ablations were delivered, and on the final lesion, loss of preexcitation was noted to occur within seconds (Figure 2B). Following ablation, programmed stimulation was repeated. Para-Hisian pacing demonstrated a nodal response. No recovery of AP conduction was noted. Her postprocedure ECG demonstrated a narrow QRS morphology without preexcitation (Figure 3). Following ablation, the patient was discharged from hospital several days later. No recovery of preexcitation was seen on ECGs done on several visits with the transplant cardiology and advanced heart failure clinic following discharge.

Discussion

The use of donor hearts from patients with WPW syndrome has been previously reported.[1–4] In many cases, preexcitation was not initially manifest around the time of organ procurement and transplant. Intermittent preexcitation and long antegrade refractory periods are several postulated mechanisms to account for the appearance of normal baseline donor ECGs when a preexcitation pattern emerges only following transplant. Cellular rejection has previously been reported to contribute to first-degree AV block.[4] Given that our patient exhibited preexcitation in the setting of

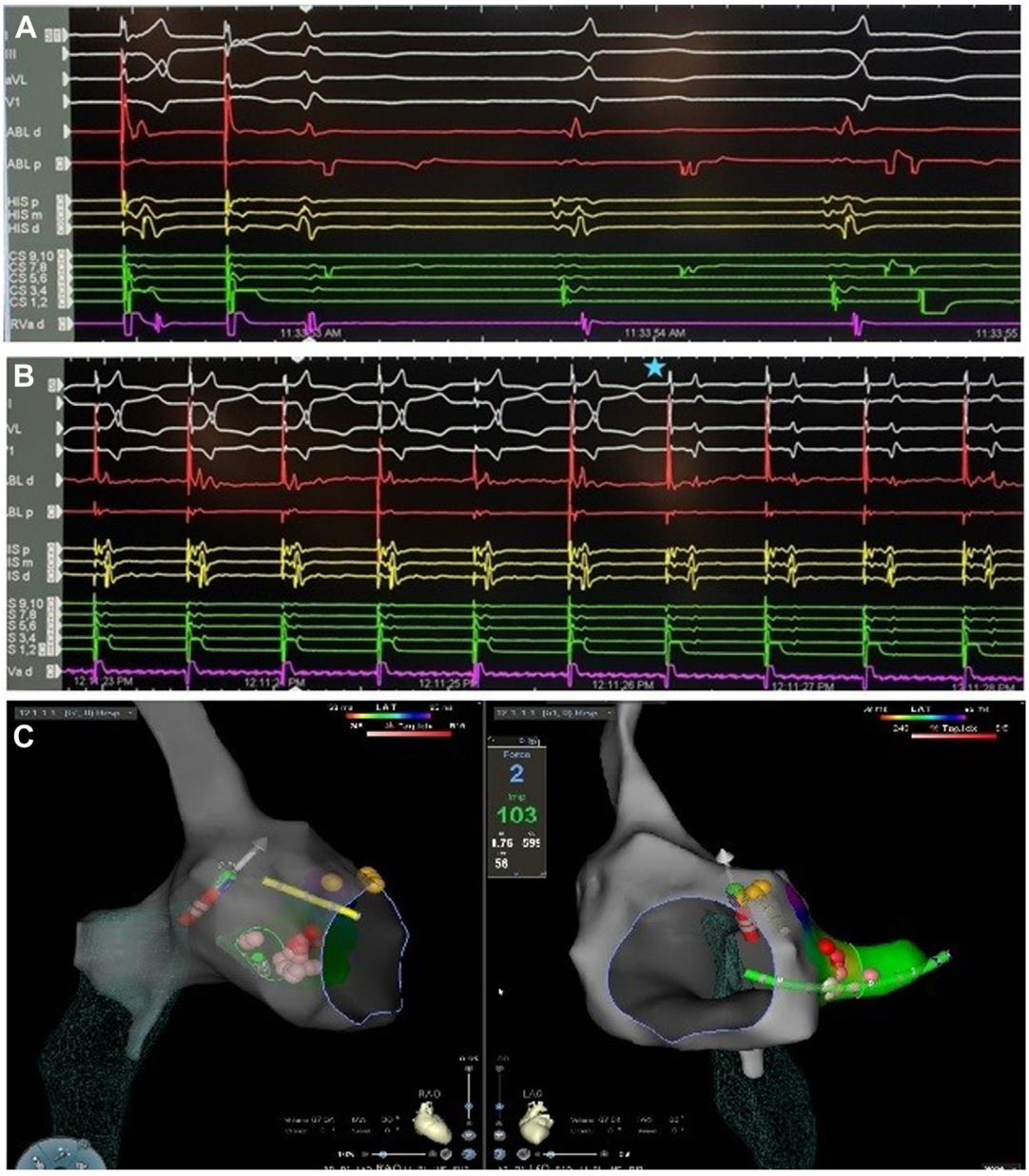

Figure 2 **A:** Atrial pacing with extrastimuli demonstrates the loss of preexcitation and conduction via the AV node following the extrastimulus. **B:** Loss of preexcitation (*star*) was noted within seconds following the final ablation lesion. **C:** Electroanatomic map with red dots corresponding to ablation points along the posteroseptal accessory pathway. Yellow dots denote areas where His electrograms were recorded.

antibody-mediated rejection but not immediately in the postoperative setting, we postulate that antibody-mediated rejection, via a similar mechanism, may contribute to conduction abnormalities impacting on AV nodal conduction that favor the emergence of preexcitation. Processes such as inflammatory cell infiltration into AV nodal tissue in the setting of rejection also seem to have an impact on relative conduction time or AV nodal refractoriness.[5,6]

The use of donor hearts with known preexisting WPW is limited. Some investigators have suggested that they should not be used, given the risk of life-threatening supraventricular arrhythmias during the postoperative phase.[2] However, others support careful consideration to increase the availability of the transplant pool, given the potential for AP ablation to address clinically significant arrhythmias.[1] Medical therapy, surgical ablation at the time of transplant, or catheter-based ablation posttransplant have been used to manage donor hearts when a WPW pattern is identified.[1,2,4,7] In contrast to previously reported cases of posttransplant WPW where APs were discovered to be left lateral, our patient's donor heart was found to have a posteroseptal AP. In addition, to the best of our knowledge, this is the first reported case of WPW becoming manifest in the setting of antibody-mediated rejection. The mechanism by which

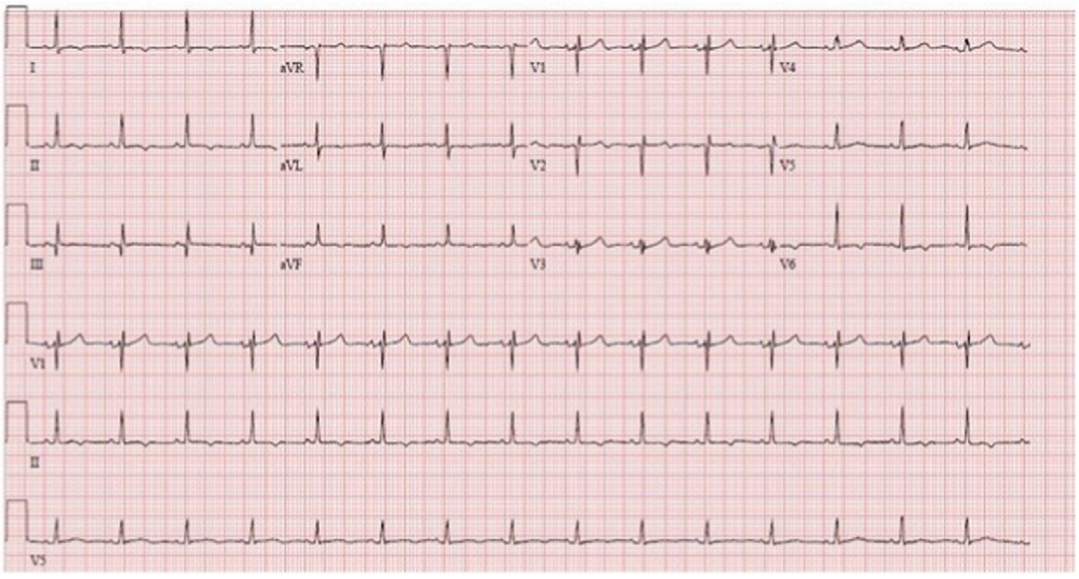

Figure 3 Postablation electrocardiogram showing the absence of preexcitation following ablation of the posteroseptal accessory pathway.

antibody-mediated rejection might contribute to preexcitation is not well understood, but additional sympathetic drive may play a role in addition to the aforementioned factors.

The decision to pursue EPS and ablation was driven by the observation of nonsustained atrial tachycardia during hospitalization, concern for a high-risk AP given the unclear etiology of the traumatic fall contributing to the donor's death, and our patient's preference to pursue a definitive solution to eliminate preexcitation in case she should develop future sustained atrial arrhythmias including atrial fibrillation. In circumstances where patients prefer to avoid EPS and ablation, or if ablation cannot be performed in the acute posttransplant setting, ambulatory monitoring or exercise testing to evaluate for the abrupt loss of preexcitation can be considered. As for other cases of WPW outside the posttransplant setting who undergo EPS, features such as the shortest preexcited R-R interval (SPERRI) $<$250 ms in atrial fibrillation, shortest paced cycle length with preexcitation during atrial pacing $\leq$250 ms, or APERP $\leq$250 ms have historically been used for risk stratification of high-risk pathways.[8] However, given that significant arrhythmias and aborted sudden death have been reported in patients who do not demonstrate high-risk EPS features, and taking into account that SPERRI, shortest paced cycle length with preexcitation, and APERP recorded in the electrophysiology lab setting may not correlate well with the SPERRI recorded during a clinically recorded arrhythmia, ablation of an AP should be considered based on the anatomic location.[9,10]

Conclusion

As this case demonstrates, manifest AP conduction may be revealed in the setting of acute antibody-mediated rejection, and successful mapping and ablation can be performed in this setting. In carefully selected cases, inclusion of donor hearts with known WPW could be considered to increase donor heart availability as long as a plan for EPS and ablation prior to postoperative discharge can be established.

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