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CASE REPORT

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Transient complete heart block following catheter ablation of a left lateral accessory pathway

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Abstract

A 16-year-old female with symptomatic Wolff-Parkinson-White (WPW) syndrome underwent catheter ablation of a left-sided lateral accessory pathway. The accessory pathway was eliminated with the first ablation lesion; however, the patient immediately developed complete heart block (CHB). At first, complete heart block was thought to be due to ablation of left atrial extension of the AV node, and pacemaker therapy was considered. However, careful ECG analysis revealed that the development of CHB was in fact due to bump injury to the AV node during transseptal catheterization. Conservative management allowed resolution of AV nodal conduction without need for a permanent pacemaker.

KEYWORDS

accessory atrioventricular bundle, atrioventricular node, catheter ablation, heart block, Wolff-Parkinson-White syndrome

1 | INTRODUCTION

Complete heart block (CHB) is a rare, but well-known complication of catheter ablation procedures involving areas in proximity to the atrioventricular (AV) node.[1] We present a unique case of CHB that occurred during ablation of a left-sided lateral accessory pathway, remote from the AV node, in a patient with orthodromic atrioventricular tachycardia (ORT).

2 | CASE REPORT

A 16-year-old female with symptomatic Wolff-Parkinson-White (WPW) syndrome (Figure 1A) was referred for catheter ablation of her accessory pathway. ORT (Figure 1C) was induced with single programmed extrastimuli from the atrium, confirmed with ventricular overdrive pacing, and localized to a left lateral pathway with earliest retrograde activation at CS 5-6. Transseptal puncture, guided by intracardiac echocardiography (ICE) and fluoroscopy, was performed. A solid tip ablation catheter was used to ablate the accessory pathway at the left lateral mitral annulus, away from the AV node, as seen on multiple imaging modalities including fluoroscopy, ICE, and 3D electroanatomic mapping (Figures 1B,D and 2C). The accessory pathway was eliminated within 5 seconds of radiofrequency energy application (i.e. 50 W, 55°C). However with elimination of the pathway, the patient simultaneously developed CHB with a junctional rhythm (Figure 2A).

The initial thought was that CHB developed due to ablation of left atrial extensions of the AV node.[2] Immediate pacemaker implantation was considered, but after careful review of the electrograms leading up to CHB, it was noted that maximal pre-excitation occurred during transseptal puncture (Figure 2B) and persisted 30 minutes later until CHB occurred after the first ablation lesion, as demonstrated by the similar pre-excited QRS morphology in Figure 2A,B. It was therefore hypothesized that the AV node was blocked due to bump catheter injury from either the ICE catheter or stretching of

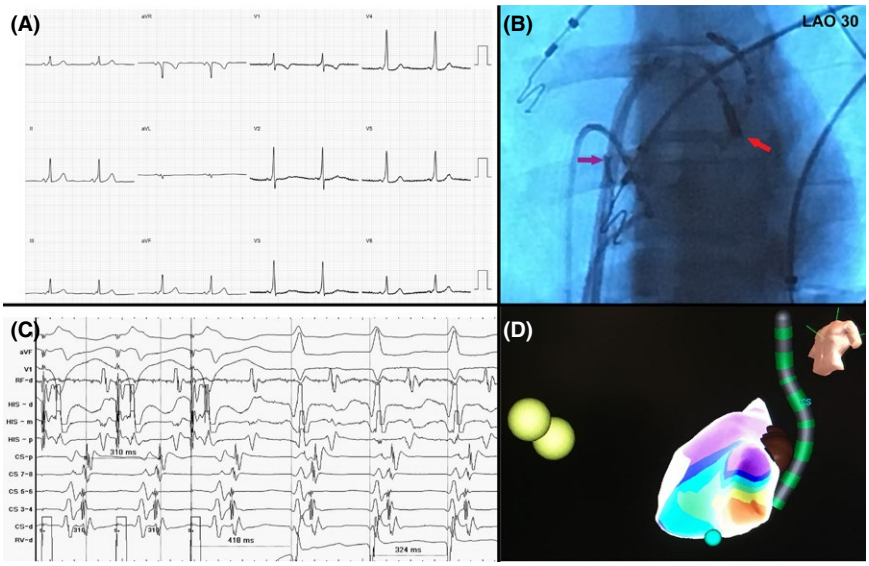

FIGURE 1 Orthodromic atrioventricular reentrant tachycardia localized to the lateral mitral annulus. A, Baseline 12-lead electrocardiogram showing pre-excited QRS complexes with delta waves consistent with a left-sided accessory pathway. B, Fluoroscopy in LAO 30π projection shows CS catheter positioned far into CS with CS5-6 near lateral mitral annulus. Ablation catheter (red arrow) positioned in the left atrium while mapping. ICE catheter (purple arrow) positioned in the anteroseptal right atrium. C, Orthodromic atrioventricular reentrant tachycardia was induced with single extrastimuli and retrograde atrial activation was earliest at the CS5-6 electrode pair. Antegrade ventricular activation was earliest at the HIS, consistent with an intact anteroseptal compact AV node. ORT was confirmed with ventricular overdrive pacing in the circuit with PPI-TCL = 94. D, Electroanatomic map showing ablation lesions (brown balls) far away from the HIS (yellow balls)

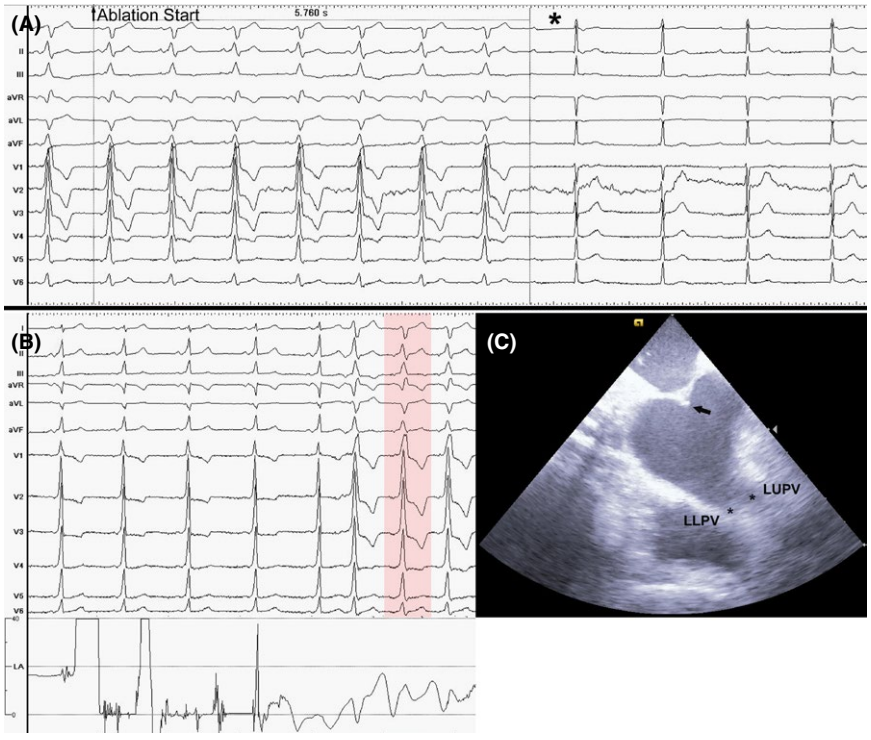

FIGURE 2 Maximal pre-excitation occurs during transseptal puncture and persists until ablation of accessory pathway with simultaneous complete heart block. A, A 12 lead ECG of maximal pre-excitation before ablation of accessory pathway (vertical arrow) and development of CHB (asterisk) simultaneously with elimination of accessory pathway. B, Careful review of electrograms revealed the development of maximal pre-excitation (pink highlight) during transseptal puncture (pressure waveforms at bottom). C, Transseptal puncture guided by intracardiac echocardiography shows standard transseptal puncture with BRK needle in the mid-fossa ovalis pointed posteriorly towards the left pulmonary veins and away from the anterior aortic valve and AV node

the atrial septum during transseptal catheterization, and antegrade AV conduction was shifted entirely over to the accessory pathway; ablation of the accessory pathway thus resulted in CHB.

Based upon this reasoning and the reversible cause of AV block, pacemaker implantation was deferred to allow recovery of AV nodal conduction. The patient was closely monitored in the intensive care unit where she had a stable junctional escape at 55 beats per minute. She was given IV methylprednisolone 125 mg. CHB persisted for 48 hours, after which she had return of AV conduction with 2nd degree 2:1 AV block with narrow QRS complex. After a total

of 72 hours, the patient had return of normal 1:1 conduction with a normal PR interval. The site of AV block was thought to be at the level of the AV node as the patient did not reveal any evidence of distal conduction disease and remained in junctional escape rhythm. She had normal chronotropic response with walking and was discharged without any further complications. She has had no symptomatic bradycardia in 1.5 years follow-up.

3 | DISCUSSION

This case was instructive for several reasons. First, the presence of an intact compact AV node at baseline was confirmed due to inducibility of ORT in which antegrade ventricular activation was earliest at the HIS bundle catheter (Figure 1C). Secondly, multiple imaging modalities gave assurance that the ablation catheter was distant from the typical location of the AV node, making ablation of the AV node unlikely. Thirdly, careful review of electrograms was critical to formulate an explanation of reversible AV block, and avoided unnecessary pacemaker implantation in this young patient.

Two other cases have been reported of transient CHB (a few minutes) in the setting of catheter ablation of a lateral accessory pathway using a retrograde aortic approach.[3,4] However, this is the first case reported of prolonged CHB (2 days) that occurred during ablation of an accessory pathway with a transseptal approach. The etiology of CHB in the previous cases was hypothesized to be caused by trauma from the diagnostic catheters resulting in inflammation of the tissue surrounding the AV node.[3] These cases reported resumption of sinus rhythm within minutes of the episode; however, our case illustrates a much longer duration of CHB that is still resolved without need for a pacemaker.

Given that the CHB persisted beyond the duration previously described in the case reports, we treated our patient with methylprednisolone in the event that inflammation was aggravating the block. We hypothesized that the corticosteroid therapy would reduce the inflammation around the AV node, and prior case reports have suggested response to steroid therapy in the setting of CHB following ablation of the slow pathway.[5] Our patient had subsequent resolution of AV node block over the next three days, but it is unclear how much corticosteroids played a role. Additional research is required to better define the role of steroid therapy in catheter-based trauma to AV nodal conduction.

4 | CONCLUSIONS

This is the first reported case of prolonged, reversible CHB caused by bump injury to the AV node during transseptal catheterization. This case highlights the critical role of detailed electrogram analysis to evaluate unexpected AV block during cases such as this in which ablation of the AV node is felt to be unlikely due to careful mapping of the accessory pathway far away from the compact AV node as seen on multiple imaging modalities. Further, this case illustrates that standard transseptal catheterization may cause prolonged suppression of the AV node, and may eventually recover after 72 hours. Conservative management was able to avoid unnecessary permanent pacemaker implantation in this young individual, who exhibits normal AV nodal conduction at 1.5 year follow-up.

DISCLOSURES

Dr. Ho has received fellowship support from Boston Scientific, Biotronik and Medtronic and owns equity in Vektor Inc. Dr. Krummen owns equity in Vektor Inc, and has served as a consultant to Abbott Laboratories, has equity in Vektor Medical, and has received fellowship program support from Abbott, Biosense-Webster, Biotronik, Boston Scientific, and Medtronic. Dr. Feld receives CCEP Fellowship Training Program stipends from Medtronic, Boston Scientific, St. Jude, Biotronik, and Biosense-Webster; has ownership equity in Perminova; and received research grants from Catheter Precision and Circa Scientific. Drs. Schweis, Hoffmayer and Green have no disclosures.

CONFLICT OF INTEREST

The authors declare no relevant conflicts of interest for this article.

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