Split Accessory Pathway Potentials in a Patient with Antidromic AVRT

Weizhuo Liu[1], Wentao Gu[2], Xinping Luo[2], Jian Li[2], and Nanqing Xiong[2]

[1]Shanghai Chest Hospital, Shanghai Jiaotong University
[2]Huashan Hospital Fudan University

November 21, 2021

Abstract

A 27-year-old female presenting palpitation without ECG documentation underwent electrophysiology study. EP study revealed atrioventricular accessory pathway with poor and unidirectional pathway conduction, and a fasciculoventricular pathway. During isoproterenol infusion, delta wave promptly became prominent, after which an antidromic AV reentrant tachycardia was induced. When the pathway was mapped, widely split double pathway potentials were observed at 12 o'clock site of tricuspid annulus during mild preexcitation, demonstrating an example of intra-pathway conduction delay, which can be reversed by isoproterenol. Ablation at the site caused accelerated pathway rhythm and eliminated the pathway, rendering the tachycardia non-inducible.

Split Accessory Pathway Potentials in a Patient with Antidromic AVRT

Weizhuo Liu MD[1,2], Wentao Gu MD[2] , Xinping Luo MD[2], Jian Li MD[2], Nanqing Xiong MD[2*]

1 Department of Critical Care Medicine, Shanghai Chest Hospital, Shanghai Jiaotong University, 241 West Huaihai Road, Shanghai 200030, China

2 Department of Cardiology, Huashan Hospital Fudan University, 12 Wulumuqizhong Road, Shanghai 200040, China

*Address for Correspondence: Nanqing Xiong MD

Department of Cardiology

Huashan Hospital Fudan University

12 Wulumuqizhong Road, Shanghai 200040, China

E-mail: huashancardio@163.com

Tel: (86)21-52887065

Key words: antidromic AVRT, split pathway potentials, intra-pathway delay, catecholamine-dependent preexcitation, pathway automaticity

Disclosures: None

Funding: (None)

Introduction

Antidromic atrioventricular reentrant tachycardia (AVRT) usually requires an accessory pathway (AP) with good conduction to serve as the anterograde limb of the circuit[1-3]. Pathways demonstrating intermittent

or unstable conduction properties are infrequently seen in antidromic tachycardia[1,3,4]. When mapping a pathway, presence of AP potential often suggests a highly effective target for ablation. Here we report a phenomenon of widely split AP potentials due to significantly slow conduction within an atrioventricular pathway in a patient with antidromic AVRT.

Case Report

A 27-year-old woman having frequent exercise-induced palpitation without ECG documentation underwent electrophysiology study. Slightly shortened H-V interval (21ms) with mild preexcitation was seen at baseline (Figure 1A). Programmed atrial extrastimuli and atrial pacing were associated with inapparent surface delta wave, while intracardiac ventricular signals were still preexcited given the activation at basal right ventricle (HIS) remaining earlier than apex (Figure 1B). The H-V interval was constant (32ms) when A-H prolonged during programmed atrial extrastimuli, consistent with an extra fasciculoventricular AP. Ventricular pacing and extrastimuli showed poor retrograde conduction solely over the AV node. When high-dose isoproterenol was given, the preexcitation progressively became prominent (Figure 2A). A wide QRS tachycardia demonstrating 1:1 V-A relationship was then initiated by ventricular pacing without visible His before QRS. Instead, a presumable retrograde His could be observed after ventricular electrogram on HISd with an H-A interval of around 100ms. Entrainment from atrium was associated with unchanged QRS morphology and an A-V-A response following overdrive cessation, which excluded ventricular tachycardia and confirmed antidromic AVRT utilizing an isoproterenol-sensitive atrioventricular AP (Figure 2B).

Mapping of the pathway was performed during sinus rhythm with low isoproterenol infusion rate to avoid catheter instability. Mild preexcitation caused by atrioventricular pathway conduction was present in this setting. When the ablation catheter was positioned near the earliest ventricular activation site at 12 o'clock site of tricuspid annulus, 15 millimeters away from the nearest His, split potentials with an interval of 30ms were recorded by the proximal electrode pair (ABLp) between atrial and ventricular signals (Figure 3A). The potentials were significantly later than all atrial signals which made it highly unlikely to be an atrial component. The second one was also visible on distal ABL according to the nearly identical timing, followed by an early local ventricular electrogram with QS unipolar pattern. The interval between the double potentials and QRS onset/local V was variable with spontaneous change of preexcitation (Figure 3B), suggesting that the potentials were also irrelevant to the ventricle, and thus could both be considered as AP potentials, demonstrating an example of intra-pathway delay. Gently withdrawing the catheter made the split potentials shifting to ABLd (Figure 3C) and caused frequent ectopy showing QRS with maximal preexcitation pattern identical to tachycardia, indicating pathway automaticity. The atrial signals were not followed by the split potential when V-A dissociation occurred, further confirming the identity AP potentials. Radiofrequency application at this site was also associated with accelerated pathway rhythm (Supplemental Figure).

The H-V interval was back to 32ms after ablation, which could not be changed by atrial pacing or isoproterenol infusion, suggesting the block of atrioventricular AP conduction. The patient has been free of arrhythmia for 6 months.

Discussion

In this case report, we have described a phenomenon of split AP potentials in a patient with a slowly-conducting atrioventricular pathway. The tachycardia can be clearly diagnosed as antidromic AVRT based on the intracardiac electrograms and atrial entrainment.

Usually, APs with stable excellent anterograde conduction are necessary for the initiation and maintenance of antidromic AVRTs. Free wall bypass tracts, including those with decremental conduction, are most commonly seen in antidromic AVRT [4,5]. Based on the findings above, our patient had a short atrioventricular pathway with baseline slow conduction in a rare (para-His) region. The evidence of decremental property was yet insufficient given the AP block during atrial pacing at long cycle lengths.

Recording of pathway potentials is useful in guiding AP ablation, which is usually manifested as a single

sharp spike 10 to 30 ms before the onset of the delta wave during anterograde conduction[1]. However, two discrete potentials between A and V were recorded near the earliest ventricular activation site in our case, which were considered as AP potentials by careful analysis of electrograms during different levels of preexcitation. This pattern of double pathway potentials is similar to the split His potentials in the setting of intra-His conduction delay, which reflected the conduction disturbance within the slow atrioventricular pathway. Although mapping during tachycardia was not attempted, we believe that the conduction could be significantly improved by isoproterenol, as evidenced by the rapidly altered QRS configuration to a near maximal preexcitation pattern.

Catecholamine-dependent preexcitation and antidromic tachycardia was not uncommon[6,7]. In our patient, both of the anterograde AP and retrograde AV nodal conduction was simultaneously improved by isoproterenol, which served as the prerequisite of the tachycardia. To our knowledge, this is the first report of split AP potentials reflecting the slow conduction over the pathway in a case of antidromic tachycardia.

In addition, the fixed short H-V interval despite decremental conduction in AV node showed the characteristic of an extra fasciculoventricular AP[8], which would not participate in AV reentry and did not require ablation.

Informed consent was obtained from the patient.

Declarations of interest

None.

References

[1] Callans D. Mark E. Josephson's Clinical Cardiac Electrophysiology. Philadelphia: Wolters Kluwer; 2021.

[2] Atié J, Brugada P, Brugada J et al. Clinical and electrophysiologic characteristics of patients with antidromic circus movement tachycardia in the Wolff-Parkinson-White syndrome. Am J Cardiol. 1990;66:1082-1091.

[3] Brembilla-Perrot B, Pauriah M, Sellal JM, et al. Incidence and prognostic significance of spontaneous and inducible antidromic tachycardia. Europace. 2013;15:871-876.

[4] Packer DL, Gallagher JJ, Prystowsky EN. Physiological substrate for antidromic reciprocating tachycardia. Prerequisite characteristics of the accessory pathway and atrioventricular conduction system. Circulation. 1992;85:574-588.

[5] Goldberger JJ, Pederson DN, Damle RS, et al. Antidromic tachycardia utilizing decremental, latent accessory atrioventricular fibers: differentiation from adenosine-sensitive ventricular tachycardia. J Am Coll Cardiol. 1994;24:732-738.

[6] Sandhu A, Tzou WS, Borne RT, et al. Uncovering a unique path: Antidromic AVRT utilizing a left anteroseptal Mahaim-like accessory pathway. Pacing Clin Electrophysiol. 2021;44:185-188.

[7] Yamamoto T, Yeh SJ, Lin FC, Wu DL. Effects of isoproterenol on accessory pathway conduction in intermittent or concealed Wolff-Parkinson-White syndrome. Am J Cardiol. 1990;65:1438-1442.

[8] Sternick EB, Gerken LM, Vrandecic MO, Wellens HJ. Fasciculoventricular pathways: clinical and electrophysiologic characteristics of a variant of preexcitation. J Cardiovasc Electrophysiol. 2003;14:1057-1063.

Figure legend

Figure 1 A: Mild preexcitation with slightly shortened H-V interval (21ms) was observed at baseline. RVA was preceded by HIS. B: Atrial pacing and the extrastimulus resulted in minimal preexcitation and a fixed H-V interval during A-H prolongation, with activation of RVA remaining later than HIS. AP=accessory pathway; CS=coronary sinus; RVA=right ventricular apex.

Figure 2 A: After isoproterenol infusion, prominent delta wave was present with an H-V interval of -12ms. B: Wide complex tachycardia without anterograde His was induced, during which atrial overdrive pacing entrai-

ned the ventricles with identical QRS configuration and an A-V-A response following cessation. CS=coronary sinus; RVA=right ventricular apex.

Figure 3 A: 2 discrete AP potentials were recorded by ABLp. B: The interval from split pathway potentials to local V increased when the atrioventricular AP blocked spontaneously. C: Pulling back the catheter made both signals appearing on distal ABL. See text for discussion. ABL=ablation catheter; AP=accessory pathway; CS=coronary sinus; RVA=right ventricular apex.

Supplemental Figure: Accelerated rhythm with the identical QRS configuration to tachycardia was present due to enhanced pathway automaticity during radiofrequency. ABL=ablation catheter; CS=coronary sinus; RVA=right ventricular apex.