CASE REPORT

Wide QRS complex tachycardia with a mysterious mask—Tricuspid isthmus-dependent atrial flutter with preexcitation syndrome and dual atrioventricular nodal pathway conduction

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Key Clinical Message

Atrial flutter (AFL) and supraventricular tachycardia (SVT) are common arrhythmias in clinic. However, some AFL cases may present additional complexities, such as both accessory pathways (AP) and dual atrioventricular node pathways, putting on a mysterious mask and making it challenging to distinguish on electrocardiograms (ECGs).

Abstract

A 60-year-old male patient had a sudden syncope, and an ECG showed wide QRS complex tachycardia. This diagnostic ambiguity is further compounded by the fact that SVT via AP conduction can exhibit wide QRS complex tachycardia characteristics resembling ventricular tachycardia (VT). Consequently, a definitive diagnosis through electrophysiological (EP) examination becomes imperative, as it dictates subsequent ablation strategies. In this article, we present a rare case involving three distinct arrhythmias including AFL, AP, and dual atrioventricular node pathways, and successfully treated through ablation.

K E Y W O R D S

ablation, accessory pathways, atrial flutter, supra ventricular tachycardia, ventricular tachycardia

1 | INTRODUCTION

Atrial flutter (AFL) and supraventricular tachycardia (SVT) are widely common arrhythmias in clinical practice.¹ However, some patients of AFL are combined with an accessory pathway (AP) or dual atrioventricular node pathway conduction, which increases the difficulty of distinguishing the electrocardiogram (ECG), owing to the truth that SVT through AP conduction may have the same wide QRS complex tachycardia characteristics compared with ventricular tachycardia (VT). Therefore, a definitive diagnosis by electrophysiological (EP) examination is

Chao Liu, Changjin Li and Xiaonan Xu are the co-first authors of this paper.

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critical, which will determine the further ablation strategy.² In this article, we report a rare case with three kinds of arrhythmia with successful ablation.

2 | CASE PRESENTATION

A 60-year-old male patient had a sudden syncope and fell in the bathroom in the morning. He regained consciousness 10s later. Upon arrival at the hospital, the emergency ECG showed wide QRS complex tachycardia (HR = 300 bpm). Subsequently, syncope occurred again with blood pressure (BP) of 80/40 mmHg. The 200J simultaneous electric energy was immediately given to turn into the sinus rhythm. But soon enough, the tachyarrhythmia was awakened again, which obtained the same ECG as before. The propafenone was used continuously to convert the sinus rhythm, and the patient was transferred to the cardiology department. This patient had a history of hypertension and diabetes with poor control. Radiofrequency ablation was performed in 2010 due to preexcitation syndrome. Physical examination: clear consciousness, clear breath sounds in both lungs, BP 85/57 mmHg, HR 120 bpm with regular sinus rhythm. For this patient, the ECG with sinus rhythm showed the preexcitation syndrome (Figure 1), and the tachycardia showed the rapid wide QRS complex (Figure 1B). Thus, VT or AFL combined with preexcitation was initially considered, and further cardiac EP examination should be performed to confirm the diagnosis.

Femoral veins were all punctured under local anesthesia, and the 10-polar catheter of the coronary sinus (CS), the ventricular electrode, and the high right atrial (HRA) electrode catheter were respectively delivered to the corresponding position (Figure 2). Intracardiac ECG showed AFL with 2:1 conduction, HRA leading. Entertaining the distal catheter of CS12 was not ideal, and when towing the CS 90, PPI-TCL=18 ms, EP diagnosis: tricuspid isthmus-dependent AF (Figure 1C). Subsequently, linear ablation was performed from the tricuspid annulus to the inferior vena cava (IVC) to terminate the AFL, obtaining



FIGURE 1 (A) Emergency ECG: wide QRS complex tachycardia (HR = 300 bpm). (B) The ECG showed preexcitation under sinus rhythm, with a positive delta wave in V1 lead and negative delta wave in III lead, the left posterior septal AP should be considered. (C) EP diagnosis: tricuspid isthmus-dependent AFL, PPI-TCL = 18 ms; (D) RAS₁S₁ 400 ms stimulation, CS34 leading, EP diagnosis: left AP; (E) A and V waves were immediately separated, and the AP forward conduction was blocked; (F) RVS₁S₁ 500 ms stimulation, CS56 leading, the retrograde conduction of AP existed; (G) VA separation by the ventricle pacing after ablation, AP retrograde conduction was blocked (blue and red arrows indicate V and A waves); (H) RA S₁S₁ 250 ms stimulation, A-V jump and induce AVNRT.

the bidirectional block of the cavotricuspid isthmus (CTI). At this point, the intracardiac ECG suggested that the V wave was fully preexcited by RAS1S1 400 ms stimulation, CS34 leading, EP diagnosis: left AP (Figure 1D). After the atrial septal puncture, small A and large V waves fused were mapped near CS34 and discharged at the earliest position. A and V waves were immediately separated within 3s (Figure 1E). Continued to be ablated until 114s, and the forward conduction of the AP was blocked successfully. The RV S1S1 500 ms was stimulated again, and the retrograde conduction still existed, with CS56 leading (Figure 1F). Ablation was performed again and successfully blocked near CS 56 within 10s, VA separation was observed by the ventricle pacing (Figure 1G). During the observation period, EP showed an A-V jump in RA S1S2 500/300 ms stimulation, and RA S1S1 250 ms can directly



FIGURE 2 The X-ray image during ablation (White arrow indicate the RV electrode catheter, the sequence of CS electrode is shown as CS12–CS90 for distal to proximal).

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induce AVNRT (170 bpm) (Figure 1H). The His bundle was mapped under the Ensite 3D-mapping system and ablated for 114s at the lower 1/3 of the His to the CS ostium (Figure 3). A stable junctional rhythm was observed during ablation. After ablation, the EP examination showed that the A-V jump disappeared, and the tachycardia could not be induced after repeated RA and RV stimulation with isoproterenol added. The patient returned to the ward after 30 min of observation.

3 | DISCUSSION

The main mechanisms of wide QRS complex tachycardia include VT (80%), SVT with aberrant conduction (15%), and SVT through AP (5%).³ Accurate diagnosis of VT is crucial; the use of inappropriate antiarrhythmic drugs may cause further harm to patients. It is essential to emphasize that the default diagnosis of ventricular tachycardia should be made for all wide QRS complex tachycardias until proven otherwise, and then we proceeded to gradually identify the arrhythmia in this case: (1) Atrioventricular dissociation⁴: atrioventricular dissociation is critical to the diagnosis of VT. However, in Figure 1, there is a regular reversed P' wave after every QRS wave in limb leads and no obvious changes in chest leads; (2) the same tropism of QRS complex in the chest leads⁵: when the negative tropism in the chest leads, VT can be diagnosed, while in positive tropism, SVT through left AP conduction should be further identified. In this case, the positive tropism in the chest leads are still not diagnosed; (3) QRS complex duration⁶: the QRS duration should be more than 160 ms when VT with LBBBlike form, with RBBB-like form>140 ms, and more than 200 ms is mostly diagnosed VT. In this case, the QRS complex duration of tachycardia is 210 ms in Figure 1. However, type A pre-excitation is shown in Figure 1B;



FIGURE 3 Three-dimensional mapping of ablation targets: tricuspid isthmus line (red and white mark lines), AP target (green marks), slow pathway target (red mark), his bundle (yellow mark).

therefore, SVT combined with AP conduction should be considered. (4) The morphology of QRS complex in V1 lead⁶: when the like-RBBB form is present, the onset of QRS complex in V1 lead would appear small "r" wave owing to the high ventricular septal excited, showed rSR ', rSr 'or rR' type. However, during the VT, the activation sequence of the ventricle is mostly conducted from the left ventricle to the right; therefore, the right chest leads would show a more significant R wave (monophasic R wave, Rsr', or bidirectional qR). The QRS complex in V1 lead during some episodes of VT shows a bimodal R wave (similar to the "M" form), but it also tends to be suggestive of VT if the left peak is higher than right peak of QRS complex, which is consistent with Figure 1. In conclusion, although SVT with AP conduction could be considered in this case, VT is still not excluded. When hemodynamic disturbance occurs, we should immediately perform emergency electrical cardioversion to restore sinus rhythm.^{7,8}

During subsequent EP examination, tricuspid isthmus-dependent AFL was first induced. After linear ablation through the tricuspid annulus to the IVC, EP showed left AP with bidirectional conduction and atrioventricular nodal dual pathways again, which induced AVNRT. Finally, all kinds of arrhythmias obtained successful ablation.

Combined with the EP examination, we analyzed that emergency ECG showed the 1:1 of AFL with forward AP conduction is more likely. The AP has a short refractory period, and the AFL could obtain 1:1 conduction by AP to reach the 300 bpm ventricular rate. This case illustrates again the importance of differential diagnosis of wide QRS complex tachycardia, especially tachycardia with AP conduction. Inappropriate choice of antiarrhythmic drugs may cause ventricular fibrillation.^{9,10} When necessary, emergency electrical cardioversion is crucial.¹¹ When the patient's condition is stable, an EP examination should be performed immediately to confirm the diagnosis and terminate the tachycardia by catheter ablation.

4 | CONCLUSION

The differential diagnosis of wide QRS complex tachycardia has always been a key topic in ECG, owing to the challenges in identification and vital clinical significance. This case is rare to have multiple arrhythmias at the same time, hoping to further expand the thinking of our electrophysiologists. When it is difficult to identify whether type of wide QRS complex tachycardia promptly, it should be treated as VT first.

AUTHOR CONTRIBUTIONS

Chao Liu: Conceptualization; writing-original draft; writing – review and editing. **Changjin Li:** Conceptualization; writing – original draft; writing – review and editing. **Xiaonan Xu:** Conceptualization; writing – original draft. **Mingyao Zhou:** Conceptualization; writing – original draft. **Xinmiao Huang:** Writing – review and editing. **Bingyan Zhou:** Writing – review and editing. **Jiang Cao:** Writing – review and editing. **Songqun Huang:** Conceptualization; writing – original draft. **Zhifu Guo:** Conceptualization; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors state that they have no conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

This manuscript conforms to the provisions of the Declaration of Helsinki in 1995 (as revised in Brazil 2013).

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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