Alternating and gradually changing narrow QRS complex tachycardia in a patient with heart failure: What is the mechanism?

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Abstract

A 23-year-old woman with palpitations for 9 years was referred for catheter ablation. ECG showed an irregular narrow complex tachycardia with alternating and gradually changing QRS morphologies after alternating and changing RR intervals, with a clear pattern of 2 alternating QRS complexes. An electrophysiology study was performed and confirmed that the mechanism of tachycardia was an automatic left-side His-Purkinje system (HPS) ventricular tachycardia. The gradually changing type-2 QRS complexes was the conduction delayed in the left anterior fascicle due to the short RR interval or the short left-side HH interval. Nine months after the index electrophysiology study, the patient encounter a progressive of heart failure with increased heart rate to 130-150 bpm during rest. Radiofrequency ablation was performed at the upper-septum for eliminating the tachycardia and resulted in complete atroventricular block. A permanent pacemaker with left bundle branch pacing was implanted. Twelve months after the ablation, the enlarged heart shrink to normal with normal left ventricular ejection fraction. In conclusion, careful interpretation of the ECG can identify the sinus P waves followed by irregular narrow complexes, thus avoiding misdiagnosis and unnecessary treatment. Unifocal HPS tachycardia could present with alternating and gradually changing narrow QRS complexes tachycardia and lead to tachycardia cardiomyopathy. Electrophysiology study and catheter ablation were useful for the diagnosis and treatment of HPS tachycardia but with high risk of atrioventricular block. However, successfully elimination the tachycardia would resolve and reverse the enlarged heart and deteriorative heart function.

Keywords
alternating QRS, ventricular tachycardia
1 | CASE PRESENTATION

A 23-year-old woman with palpitations for 9 years was referred for catheter ablation. Previous Holter showed a sustained tachycardia without normal sinus rhythm, with a total of 185,315 beats/24 hr, and an average heart rate of 130 bpm. Her tachycardia was refractory to several medications (including metoprolol, verapamil, diltiazem, and amiodarone, among others) and resulted in global enlargement of the heart with heart failure. At the hospital, her heart rate was 112 bpm and her blood pressure was 135/75 mmHg. The echocardiography data were as follows: left atrial diameter, 46.0 mm; left ventricular end-diastolic/systolic diameter, 60.0/50.0 mm; interventricular septum thickness, 9.0 mm; main pulmonary artery inner diameter, 26.6 mm; right ventricular diameter 24.6 mm; right atrial diameter, 57.9*44.7 mm; left ventricular ejection fraction, 34.3%; fraction shortening, 16.6%; systolic volume, 61.7 ml; cardiac index, 4.1 L/min*m²; moderate to severe mitral regurgitation; and minor tricuspid regurgitation. Laboratory workup revealed a NT-Pro BNP level of 1,267 pg/ml. Her 12-lead ECG on admission was shown in Figure 1.

What is the most likely mechanism underlying this rhythm?

2 | INTERPRETATION

ECG (Figure 2) showed an irregular narrow complex tachycardia with alternating and gradually changing QRS morphologies after alternating and changing RR intervals, with a clear pattern of 2 alternating QRS complexes. One QRS complex (type-1, red arrows), after a longer RR interval (446–545 ms), was relatively constant in axis. The other QRS complexes (type-2, purple arrows), after a shorter RR interval (377–388 ms), gradually changed in axis (shifted to left and superior when compared to type-1 QRS complex). Despite the irregular narrow complex tachycardia, sinus node originated P waves (stars, positive in the inferior leads and lead V1) occurred at relatively regular intervals (811–946 ms). The dissociation between the P waves and irregular QRS complexes ruled out supra-ventricular tachycardias (atrial fibrillation, atrioventricular nodal nonreentrant/reentrant tachycardia, and so on). No presentation of retrograde P waves making junctional tachycardia unlikely. One possible diagnose remained: His-Purkinje system (HPS) tachycardia with aberrant conduction but without retrograde conduction to atrium.

An electrophysiology study was performed. The difference in HV interval during spontaneous ectopias and atrial pacing ruled out junctional tachycardia (Figure 3a). The right-side His was retrogradely activated by the left-side His and resulted in short right-side HV interval during a short VV interval (Figure 3b). However, the left-side HV intervals were constant. Furthermore, the changes in left-side HH intervals preceded the changes in VV intervals. The absence of an entrainment phenomenon in response to overdrive pacing during sustained ventricular tachycardia ruled out the re-entry mechanism. The mechanism of this tachycardia appears to be an automatic left-side HPS ventricular tachycardia. Moreover, it was confirmed that delayed conduction occurred within the HPS due to the short left-side HH interval. Therefore, although no observation the type-2 QRS during electrophysiology study, it was reasonable to presume that the most likely explanation for the gradually changing type-2 QRS complexes was the conduction delayed in the left anterior fascicle due to the short RR interval or the short left-side HH interval. The ladder diagram of this tachycardia is illustrated at the bottom of Figure 2. Activation mapping confirmed that the tachycardia originated from the left-side His. However, due to the high risk for complete atrioventricular block and pacemaker implantation, the patient refused further ablation and was discharged after optimization of medical therapy (Entresto, amiodarone, carvedilol).

However, 9 months after the index electrophysiology study, the patient encounter a progressive of heart failure with increased heart rate to 130-150 bpm during rest. Therefore, she was referred to our hospital and finally underwent the catheter ablation. The tachycardia could be suppressed by ablation from the non-coronary cusp but could not be eliminated completely and recurred soon (Figure 4). Ablation performed at the upper-septum eliminated the tachycardia and resulted in complete atrioventricular block. A permanent
FIGURE 2  Twelve-lead ECG showing tachycardia with annotation and associated ladder diagram. Twelve leads are shown in the upper panel. The P waves, positive in inferior leads and lead V1, are marked with blue stars. The PP intervals and RR interval were annotated with ms units. The red and purple arrows indicate type-1 and type-2 QRS complexes, respectively. Note the alternation between type-1 and type-2 QRS complex after the changes in RR interval. Type-2 QRS complexes changed gradually (notably in the limb leads). The bottom panel shows the ladder diagram of the tachycardia. The green dots indicate the originating foci. The curves indicate delayed conduction within the His-Purkinje system (HPS). AVN, atrioventricular node

FIGURE 3  Surface 12 leads ECG and intracardiac electrograms with annotations. All numbers were annotated with ms units. Panel A. Intracardiac electrograms, including His recorded at the right side (His P, M, and D) and coronary sinus (CS). The spontaneous atrial electrograms (As) were recorded and dissociated with the His and QRS. Note the changes in HV intervals and VV intervals during spontaneous ectopias. Panel B. Intracardiac electrograms, including CS and left-side His (MAP P and D) and right-side His (His P and D). The left-side HV (LHV) was constant but the right-side HV (RHV) changed. The last QRS complex presented with right bundle branch block, as a result of delayed conduction from left-side His to right-side His.
pacemaker with left bundle branch pacing was implanted. Twelve months after the ablation, the enlarged heart shrink to normal with normal LVEF (60%).

3 | DISCUSSION

The differential diagnoses of irregular narrow QRS complexes tachycardia included atrial fibrillation, atrial flutter with variable block, sinus rhythm with premature supra-ventricular complexes, atrioventricular nodal reentry tachycardia with upper common pathway block, multifocal atrial tachycardia, and dual atrioventricular nodal nonreentrant tachycardia, and so on (Fadahunsi et al., 2019). However, in this case, the dissociation between the sinus P waves and irregular QRS complexes ruled out supra-ventricular tachycardias. Furthermore, no presentation of retrograde P waves making junctional tachycardia unlikely. Therefore, the diagnoses of this narrow complex tachycardia with alternative QRS morphology should be an HPS tachycardia, which was confirmed by the electrophysiology study.

According to the electrophysiology study, the mechanism of this HPS tachycardia should be unifocal tachycardia with abnormal automaticity, but not multifocal tachycardia (Itoh & Yamada, 2018). In general, unifocal HPS tachycardia presented with monomorphic tachycardia and was classified as a propranolol-sensitive automatic ventricular tachycardia (Nogami, 2011). However, as indicated in this rare case, her tachycardia presented with alternative QRS morphologies and was refractory to several medications, including metoprolol, verapamil, diltiazem, and amiodarone. Therefore, it would be difficult to control some unifocal HPS tachycardia by medicine, although with the mechanism of abnormal automaticity.

The catheter ablation for HPS tachycardia would be challenge in clinical practice, especially when the original site near the His bundle (Haissaguerre et al., 2016). Although new approach (Chen et al., 2020) (ablation from coronary cusp) was tried but failed, this patient suffered with completely ativoventricular block during ablation. However, after the elimination of tachycardia and the pacemaker implantation, the enlarged heart and heart failure were both dramatically improved. Therefore, the dilated heart and heart failure was the result of tachycardia cardiomyopathy (Towbin et al., 2019).

In conclusion, careful interpretation of the ECG can identify the sinus P waves followed by irregular narrow complexes, thus avoiding misdiagnosis and unnecessary treatment. Unifocal HPS tachycardia could present with alternating and gradually changing narrow QRS complexes tachycardia and lead to tachycardia cardiomyopathy. Electrophysiology study and catheter ablation were useful for the diagnosis and treatment of HPS tachycardia but with high risk of atrioventricular block. However, successfully elimination of the tachycardia would resolve and reverse the enlarged heart and deteriorative heart function.

CONFLICT OF INTEREST

None.

DATA AVAILABILITY STATEMENT

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

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