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Concomitant Wolff-Parkinson-White and Atrioventricular Nodal Reentrant Tachycardia: Which Pathway to Ablate?

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
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Conflict of interest: None declared

Patient: Male, 54
Final Diagnosis: WPW and AVNRT
Symptoms: Palpitations • shorthness of breath
Medication: —
Clinical Procedure: EP Study/Radiofrequency Ablation
Specialty: Cardiology

Objective: Rare co-existance of disease or pathology

Background: Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common form of supraventricular tachycardia. In contrast, Wolff-Parkinson-White (WPW) pattern consists of an accessory pathway, which may result in the development of ventricular arrhythmias. Frequent tachycardia caused by AVNRT and accessory pathways may play a role in left ventricular systolic dysfunction.

Case Report: A 54-year-old man presented with palpitations and acute decompensated congestive heart failure. His baseline EKG showed Wolff-Parkinson-White (WPW) pattern. While hospitalized, he had an episode of atrioventricular nodal reentrant tachycardia (AVNRT). He underwent radiofrequency catheter ablation for AVNRT, and his accessory pathway was also ablated even though its conduction was found to be weak. He was clinically doing well on follow-up visit, with resolution of his heart failure symptoms and normalization of left ventricular function on echocardiography.

Conclusions: This case raises the question whether the accessory pathway plays a role in the development of systolic dysfunction, and if there is any role for ablation in patients with asymptomatic WPW pattern.

MeSH Keywords: Heart Failure • Pre-Excitation Syndromes • Tachycardia, Supraventricula

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/894647

Background

Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common form of supraventricular tachycardia. In contrast, Wolff-Parkinson-White (WPW) pattern consists of an accessory pathway which predisposes to the development of ventricular arrhythmias if conduction takes place through this pathway. WPW in asymptomatic patients is less understood. Little is known about the role of AVNRT and accessory pathways in the development of left ventricular dysfunction.

Case Report

A 54-year-old man with hypertension, dyslipidemia, and diabetes presented to the hospital complaining of several weeks' history of palpitations and 1-week history of progressive shortness of breath, orthopnea, paroxysmal nocturnal dyspnea, bilateral leg swelling, and weight gain. He also complained of intermittent palpitations. On examination, his blood pressure was 166/88 mm Hg and his heart rate was 102 beats per minute (bpm). He had an elevated jugular venous pressure (JVP), clear lungs, normal S1/S2 with no added sounds, and 2+ pitting edema bilaterally. His EKG demonstrated sinus tachycardia, left ventricular hypertrophy and WPW pattern (Figure 1). CXR showed subsegmental atelectasis. His creatinine was 1.4 (baseline 1.06), and pro-BNP was 3732 pg/ml. He was started on intravenous furosemide and continued on metoprolol and lisinopril. The following morning, he had abrupt onset of palpitations and a 12-lead EKG revealed narrow complex tachycardia with a short RP interval at a heart rate of 180 bpm, suggestive of AVNRT (Figure 2). Carotid massage was attempted without success, but rapid intravenous administration of adenosine 6 mg converted him back to sinus rhythm. A 2-D echocardiogram showed severely reduced systolic function with ejection fraction (EF) of 25–30% (LV function was normal 6 months prior), mild left ventricular hypertrophy with borderline enlarged left ventricle, left atrial enlargement, and a small-to-moderate pericardial effusion without hemodynamic effect. Because of the presence of unexplained cardiomyopathy as well as preexcitation in this relatively young patient, a blood sample was obtained to rule out Fabry's disease. The test was performed at the Mayo Clinic, but was negative. An electrophysiologic study (EPS) confirmed typical AVNRT, and the patient underwent a slow pathway ablation. In addition, the EPS revealed a bystander right anterolateral accessory pathway with weak anterograde conduction and no retrograde conduction. The pathway's effective refractory period (ERP) from the coronary sinus os was 360 ms, and from the high right atrium it was 390 ms. The AH interval was 69 ms and the HV interval was –31 ms. During mapping there appeared to be evidence of a pathway potential and it was felt the benefits of ablation outweighed the risks. There was no evidence of preexcitation post-ablation (Figure 3). Coronary angiography revealed no evidence of coronary artery disease. The patient was discharged home on optimal heart failure medications and was clinically compensated and palpitation-free at follow-up. A repeat echocardiogram done 6 months later showed improved EF of 60–65%.

Discussion

Atrioventricular nodal reentrant tachycardia (AVNRT) is a narrow complex tachycardia characterized by the presence of dual electrical pathways near or in the AV node. In contrast, Wolff-Parkinson-White (WPW) pattern is diagnosed by the presence of short PR interval, delta waves, and widened QRS complex. Once symptomatic arrhythmias develop, it is termed WPW syndrome [1]. Its prevalence is estimated to be 1–4.5 per 1000 [2]. The risk of sudden cardiac death is approximately 0.3% when conduction through the accessory pathway predisposes to the development of ventricular fibrillation, especially in the presence of atrial fibrillation [3].

Our patient presented with intermittent palpitations, new-onset symptomatic systolic dysfunction, and an EKG with preexcitation, suggestive of a right free-wall origin. His palpitations during the hospital stay were caused by a narrow complex tachycardia highly suggestive of (AVNRT) given the location of the retrograde p wave in the terminal portion of the QRS, making atrioventricular reentrant tachycardia (AVRT) unlikely since the participation of a right-sided free-wall accessory pathway would demonstrate a longer RP interval. AVNRT was confirmed during the EPS by a combination of: (1) short septal VA time of 50 ms, (2) dissociating the ventricle from the atrium during ventricular pacing, and (3) consistent termination of the tachycardia without advancing the atrial electrogram.

While hypertension and diabetes could have contributed to the development of his cardiomyopathy, the presence of WPW pattern as the sole or partial contributor to possibly explain his cardiomyopathy cannot be definitely proven. Interestingly, it has been reported that the location of the accessory pathway may lead to left ventricular dysfunction in asymptomatic WPW syndrome [4,5]. The postulated mechanism is that right-sided accessory pathways can lead to dyssynchronous LV contraction with subsequent remodeling and depressed function [6]. In addition, incessant tachycardia from AVNRT may play a role in the development of systolic dysfunction [7].

There remains a controversy on how to approach asymptomatic patients with WPW pattern, given the low annual incidence of sudden cardiac death (up to 0.39%) and the relatively low sensitivity and specificity of non-invasive testing used for risk stratification, (e.g., exercise or pharmacological stress

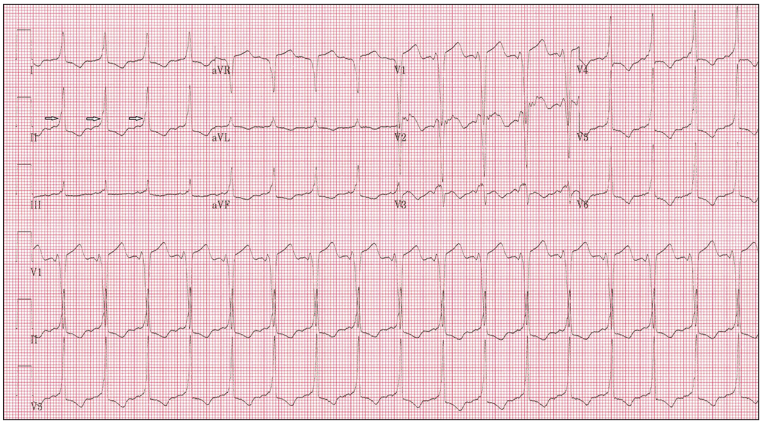

Figure 1. Pre-excitation with delta waves (arrows).

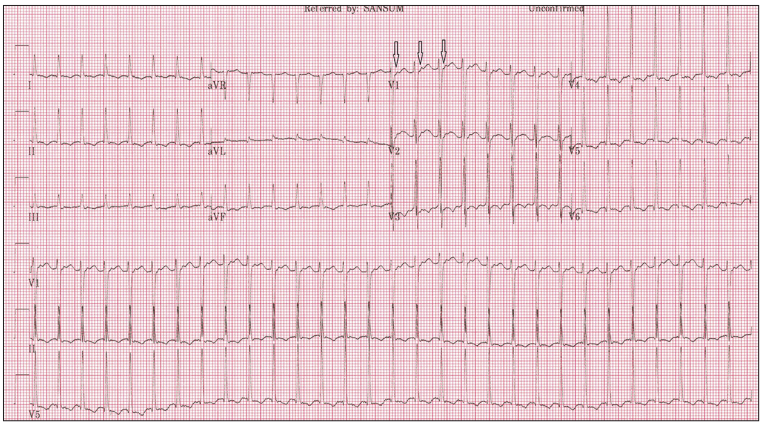

Figure 2. AV nodal reentry tachycardia. The P waves can be seen immediately following the QRS complex (arrows).

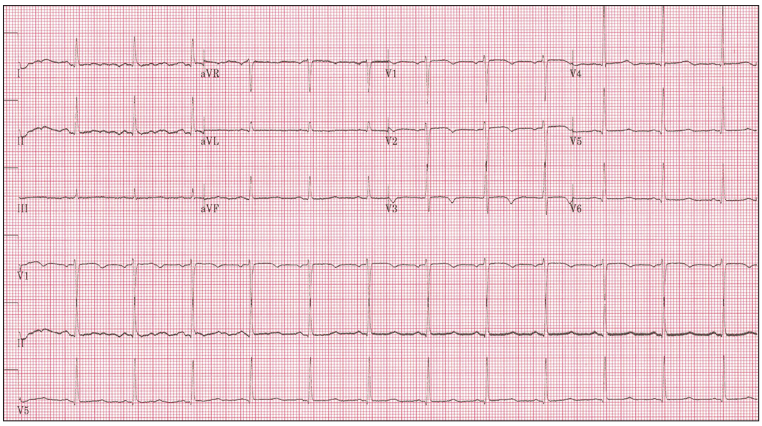

Figure 3. Normal sinus rhythm.

test) [8]. The current American College of Cardiology (ACC) and American Heart Association (AHA) guidelines for asymptomatic patients with WPW restricts catheter ablation to high-risk occupations [9].

Our patient was undergoing radiofrequency catheter ablation for AVNRT, and, given the possible association of LV dysfunction associated with an accessory pathway and the known risk (although low) of sudden cardiac death, we felt that ablation of the accessory pathway in the same setting was a reasonable therapeutic approach, despite current guidelines.

Conclusions

While the presence of delta waves may be an incidental finding, it may play a significant role in patients with newly diagnosed congestive heart failure. Comorbid conditions, risk of sudden cardiac death, and left ventricular dysfunction should all be taken into consideration when physicians evaluate asymptomatic WPW patients who may be considered for catheter ablation. In selected cases, a judicious and well-balanced clinical decision may be more beneficial and patient-oriented than rigid adherence to guidelines.

Conflicts of interest

Authors report no conflicts of interest.

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