

MINI-FOCUS ISSUE ON ELECTROPHYSIOLOGY AND PACING

BEGINNER

CASE REPORT: CLINICAL CASE

Tachycardiomyopathy Caused by a Pseudo-Ventricular Tachycardia



The Role of Imaging and Ablation in Diagnosis and Treatment

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ABSTRACT

A 50-year-old man presented with general fatigue on exertion. Investigations revealed tachycardia-induced cardiomyopathy induced by Wolff-Parkinson-White syndrome and atrial fibrillation. He was successfully treated with catheter ablation. Cardiac magnetic resonance imaging revealed that delayed enhancement throughout the left ventricle disappeared within 2 months after ablation. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2020;2:572-6) © 2020 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

The prevalence of accessory pathways (AP) is 0.1% to 0.3% in the general population. Tachyarrhythmia is observed in 10% to 30% of Wolff-Parkinson-White (WPW) pattern cases of which 70% are of atrioventricular re-entrant tachycardia and 10% to 38% are of atrial fibrillation (AF) (1).

AF complicated with WPW syndrome, “fast, broad, and irregular (FBI) tachycardia,” is clinically important because it leads to tachycardia-induced cardiomyopathy (TIC) (2) and sudden cardiac death (3). However, TIC caused by FBI tachycardia is rare. We

report a case of reversible delayed enhancement of cardiac magnetic resonance (CMR) imaging.

HISTORY OF PRESENTATION

A 50-year-old man presented to a primary care doctor with edema and general fatigue on exertion. An electrocardiogram revealed AF with rapid ventricular response, which was accompanied by WPW pattern (Figure 1A). The minimum RR interval was 160 ms. Digoxin and edoxaban were administered for 1 month to control AF and heart failure without improvement. The patient was referred to a cardiologist. Echocardiography revealed that left ventricular (LV) ejection fraction (EF) was markedly reduced. Amiodarone was administered intravenously and then orally; however, it was ineffective. Despite electrical cardioversion, AF recurred. Catecholamine was necessary to control heart failure. He was subsequently transferred to our hospital for recurrent AF treatment by catheter ablation (CA).

LEARNING OBJECTIVES

- To determine the efficacy of CA in treating TIC caused by simultaneous AF and WPW.
- To demonstrate the role of treatment in reversing delayed-enhancement in CMR imaging in a case of TIC.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, or patient consent where appropriate. For more information, visit the *JACC: Case Reports* [author instructions page](#).

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PAST MEDICAL HISTORY

The patient's medical history was unremarkable, and he was taking no medications.

INVESTIGATION

Brain natriuretic peptide (690 pg/ml) and creatinine (1.31 mg/dl) levels were elevated. A chest radiography revealed cardiomegaly (cardiothoracic ratio: 60%) with pulmonary congestion. Echocardiography revealed dilatation of LV and left atrium, LV systolic dysfunction (EF: 31.3%), moderate mitral regurgitation due to tethering, and moderate tricuspid regurgitation. We performed screening tests to detect any underlying diseases causing LV dysfunction; however, coronary angiogram, cardiac biopsy, and gallium scintigraphy were inconclusive. He was diagnosed with TIC secondary to FBI tachycardia. CMR imaging performed during sinus rhythm revealed delayed enhancement throughout the LV circumference (Figure 2A). T₂-weighted images showed a slightly higher signal intensity.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis included dilated cardiomyopathy, TIC, myocarditis, and cardiac sarcoidosis. Dilated cardiomyopathy and cardiac sarcoidosis were ruled out because LVEF improved in a relatively short time. Regarding myocarditis, no symptoms suggestive of viral infection, inflammatory responses, or any abnormal findings other than WPW pattern and AF in

electrocardiogram were observed. Therefore, myocarditis was unlikely. TIC was diagnosed based on EF improvement shortly after the termination of tachycardia.

MANAGEMENT

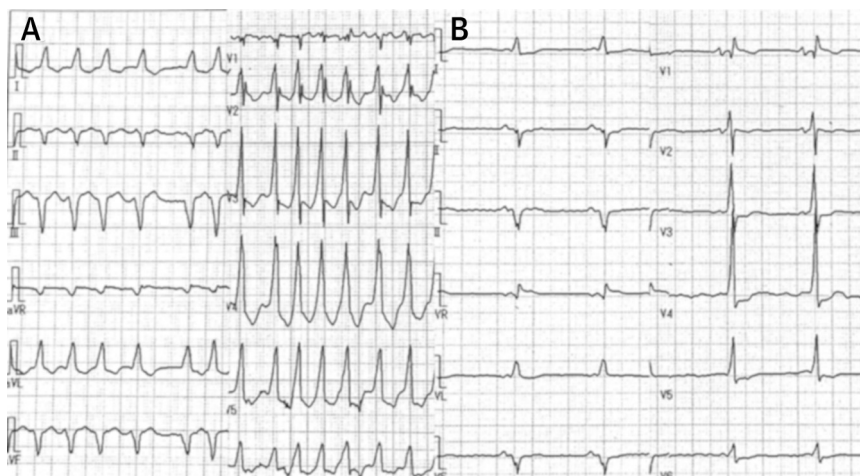
On admission to our hospital, in addition to amiodarone infusion, the patient received continuous dobutamine infusion and furosemide to control heart failure. However, an electrocardiogram revealed AF with a rapid ventricular response and wide QRS complexes. His heart rate was 155 beats/min under amiodarone infusion, and his body weight had increased by 10 kg due to general edema. We administered landiolol, which was ineffective in controlling AF rate. Although electrical cardioversion was applied several times, AF recurred repeatedly. Therefore, CA was attempted to eliminate AP conduction and control heart rate; however, it failed because the pathway was located adjacent to the His bundle, and it was difficult to recognize the earliest point of ventricular activation during rapid AF. Amiodarone was infused rapidly again, and following several days of continuous infusion, sinus rhythm gradually resumed, with an improvement of heart failure symptoms (Figure 1B).

On the 18th day, a second CA session was performed successfully, with cryoballoon pulmonary vein isolation and radiofrequency ablation of the AP (Figure 3). The patient was discharged on the 30th day.

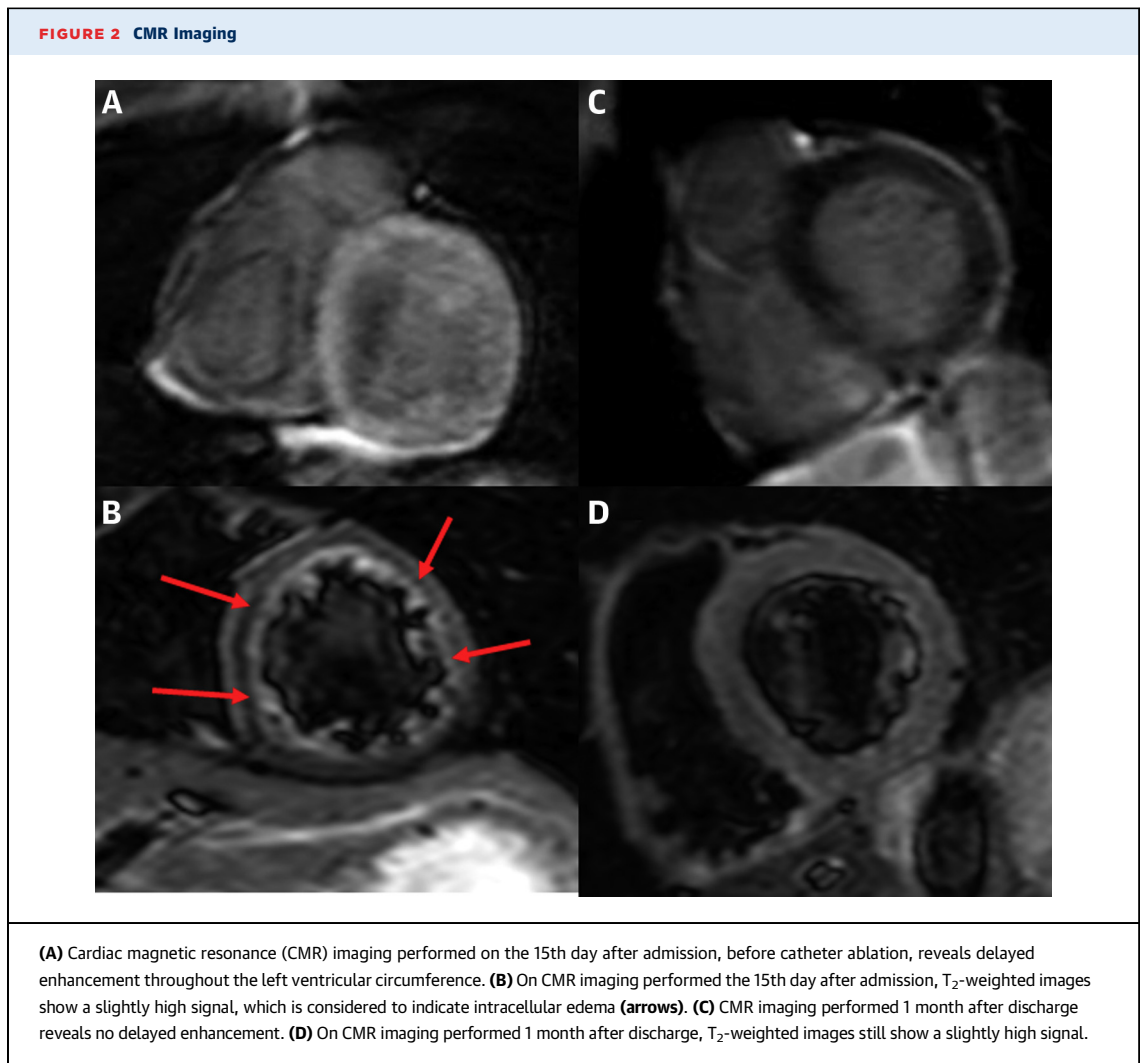
ABBREVIATIONS AND ACRONYMS

- AF = atrial fibrillation
- AP = accessory pathway
- CA = catheter ablation
- CMR = cardiac magnetic resonance
- EF = ejection fraction
- FBI = fast, broad, and irregular
- LV = left ventricle
- TIC = tachycardia-induced cardiomyopathy
- WPW = Wolff-Parkinson-White

FIGURE 1 A 12-Lead Electrocardiogram During AF and Sinus Rhythm



(A) Atrial fibrillation (AF) with rapid ventricular response and wide QRS interval. (B) Sinus rhythm and delta-wave with the same configuration as in A during AF.



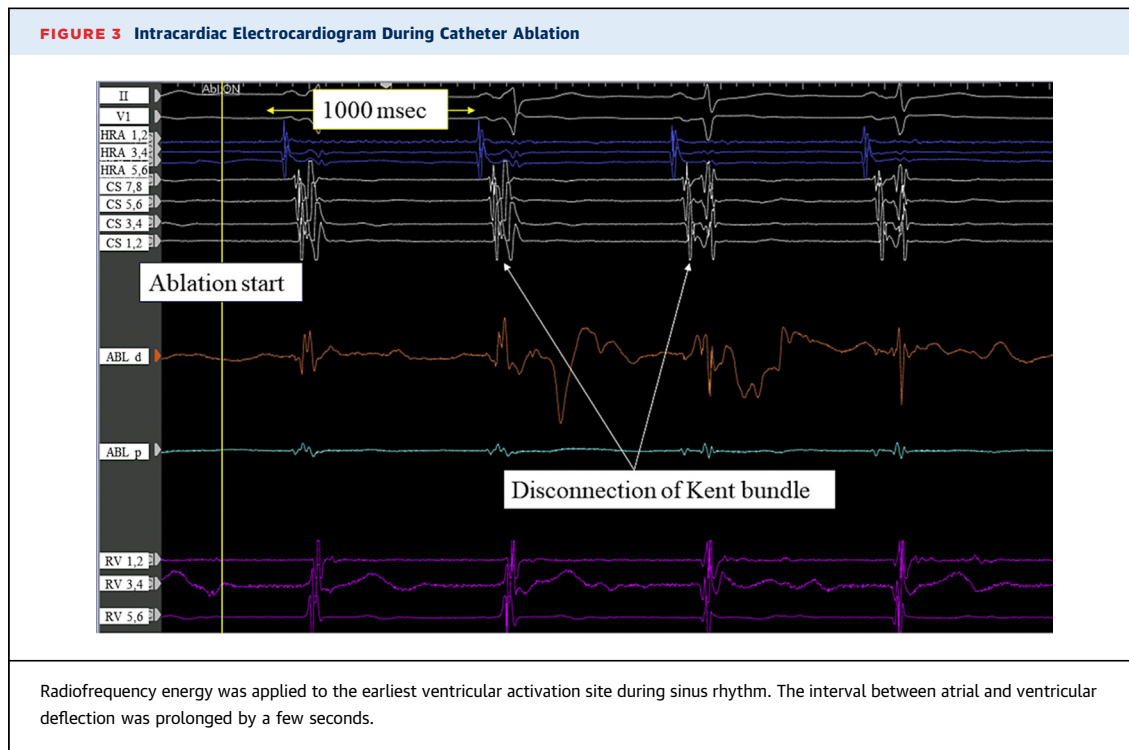
DISCUSSION

Patients with tachycardia such as AF, atrial flutter, or atrial and ventricular tachycardia have a high risk of TIC (4). FBI tachycardia may lead to ventricular fibrillation in cases where AP has a shorter refractory period. However, it is rarely associated with TIC (3). Various factors have been proposed to cause cardiac dysfunction in TIC, including exhaustion of cardiac muscle energy, decreased density and responsiveness of beta receptors, decreased coronary blood flow reserve due to a short diastolic phase, and abnormal calcium dynamics (5).

Reports suggest that LV dysfunction and dilatation are ameliorated by the treatment of tachycardia (5). The period of LVEF recovery varies from 1.5 to 6 months, and it is often difficult to distinguish TIC from dilated cardiomyopathy (6). However, our patient showed a marked improvement in LVEF within

2 months from the treatment of tachycardia. This finding was compatible with the diagnosis of TIC. Reports on WPW-complicated TIC are scarce. Değirmencioglu et al. (7) reported that TIC was treated with medications to control heart rate.

To our knowledge, this is the first report on CMR findings of TIC. Regarding the delayed enhancement in TIC, there is only 1 report on TIC caused by frequent premature ventricular complexes. In this report, 1 of 19 cases showed partially delayed enhancement in TIC. LV contractility was transiently depressed with idiopathic ventricular arrhythmias (8). In contrast, our case showed delayed enhancement in the entire LV. The mechanism of delayed enhancement in CMR includes an increase in the interstitium, accompanied by infarction or fibrosis, decrease in blood flow, and prolonged washout. In this case, transient factors such as myocardial edema, decreased blood flow, or prolonged washout could



have influenced the enhancement because it disappeared after 2 months.

The prognosis of TIC is generally good. However, there are some reports of TIC accompanied by sudden death. Nerheim et al. (9) reported 3 deaths in 24 cases of TIC.

FOLLOW-UP

Echocardiography performed 2 months after discharge revealed that LVEF had normalized to 57%. CMR imaging showed no delayed enhancement at that time (Figure 2C). At the time of discharge, the patient received a beta-blocker, an anticoagulant, and an angiotensin-converting enzyme inhibitor. During follow-up, AF and heart failure did not recur. Medications were gradually reduced and discontinued

9 months after CA. He has been followed for more than 1 year without symptoms. He will continue to receive follow-up to monitor any recurrence of AF.

CONCLUSIONS

We report a rare case of TIC; it is difficult to determine whether TIC was related to the presence of pre-excitation, a rapid ventricular rate, or both. CA resulted in remarkable improvement of cardiac dysfunction.

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KEY WORDS ablation, atrial fibrillation, cardiac magnetic resonance imaging, tachycardia-induced cardiomyopathy, Wolff-Parkinson-White syndrome