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A Complex Rhythm Treated Simply: Fascicular Ventricular Tachycardia

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PRESENTATION

A patient with no known history of ischemic or structural heart disease developed a ventricular tachyarrhythmia from a somewhat uncommon cause. The 57-year-old man presented to the emergency department with an initial complaint of several days of abdominal pain. During examination, he was found to have an irregular rapid heart rate. A 12-lead electrocardiogram (ECG) revealed a wide-complex tachycardia at a rate of 186 beats per minute (Figure 1A) with 2:1 ventriculoatrial conduction (Figure 2). The patient denied any active or prior chest pain, palpitations, presyncope, or syncope.

His past medical history was significant for hypertension and a similar arrhythmia 4 years earlier. At that time, he underwent successful electrical cardioversion and was treated subsequently with metoprolol tartrate, 50 mg, twice daily, but he was lost to follow-up after discharge. He denied any use of alcohol, tobacco, or illicit drugs.

ASSESSMENT

During the tachyarrhythmia, the patient had a blood pressure of 85/64 mm Hg. He was alert, oriented, and not in acute distress. A cardiopulmonary examination was negative for decompensated heart failure or valvular disease. He was given 6-mg and 12-mg doses of adenosine intravenously without success. Next, amiodarone, 150 mg, was given intravenously without results. Ultimately, sinus rhythm was achieved (Figure 1B) with 100 joules of external electrical cardioversion.

A transthoracic echocardiogram demonstrated a normal left ventricular ejection fraction of 55-60% and no significant valvular abnormalities. Serial cardiac biomarkers were negative. Coronary angiography did not demonstrate any significant coronary artery disease.

DIAGNOSIS

The patient then underwent an electrophysiology study. With invasive mapping techniques, the tachyarrhythmia focus was found to originate in the left posterior fascicle, confirming that the patient's tachyarrhythmia was consistent with fascicular ventricular tachycardia.

The presentation of wide complex tachycardia can pose a diagnostic dilemma for the clinician, with significant differences in choice of treatment depending on the etiology of the arrhythmia. Most wide complex tachycardias are the result of ischemic heart disease and should be identified and treated according to current guidelines on pharmacologic and therapeutic interventions.[1,2] The differential diagnosis for these arrhythmias also may include supraventricular tachycardia with aberrancy and idiopathic ventricular tachycardia.

Fascicular ventricular tachycardia represents a less common subgroup of these idiopathic ventricular tachycardias. It can be misdiagnosed easily and over-treated with antiarrhythmic drugs and cardioversion in the acute setting, because it is relatively infrequent. This fairly benign rhythm, which originates near and involves 1 of the 2 fascicles of the left ventricular conduction system, occurs in individuals without any evidence of ischemic or structural heart disease. Typically, fascicular ventricular tachycardia is found in young, otherwise healthy individuals at a mean age of 31 years (range, 12-68 years of age) who have a chief complaint of palpitations.[3-5] Belhassen et al first identified that these arrhythmias could be effectively terminated and/or suppressed with verapamil.[6]

In addition to the patient's clinical presentation, fascicular ventricular tachycardia can be identified from its

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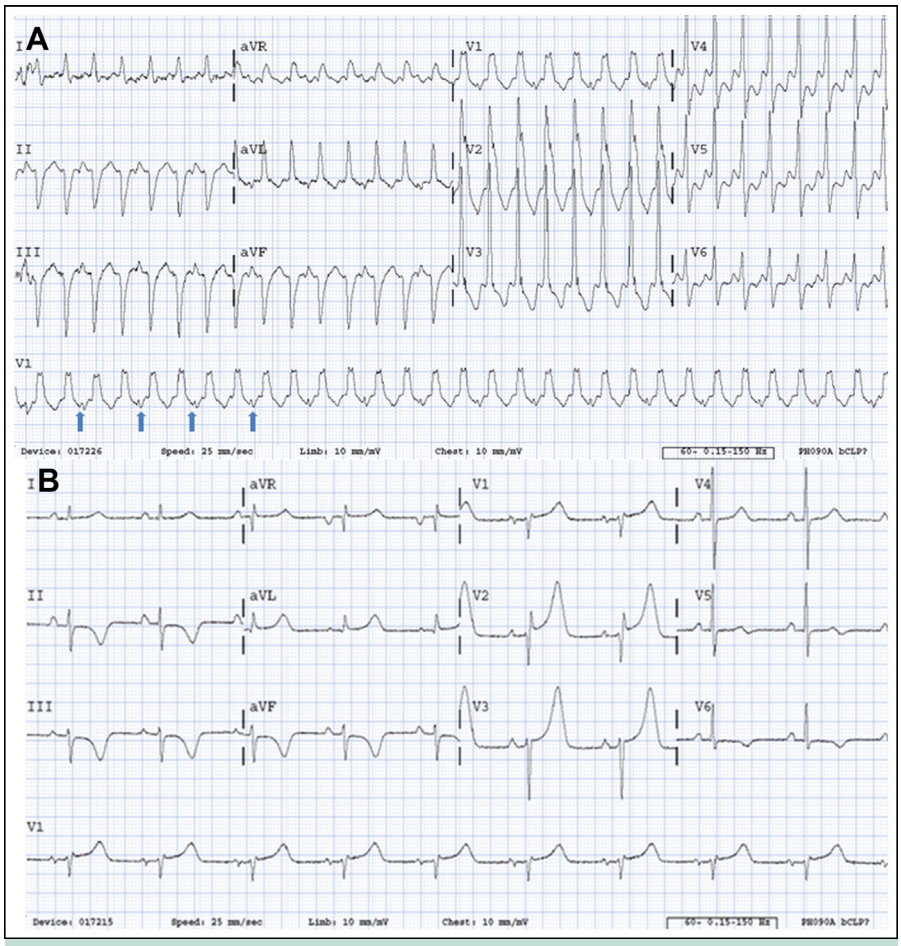

Figure 1 (**A**) A 12-lead electrocardiogram (ECG) was obtained upon the patient's admission. It showed a regular wide complex tachycardia at 186 beats per minute and a QRS interval of 138 msec. The rhythm had typical right bundle branch block (RBBB) and left anterior fascicular block (LAFB) morphology (as seen with left-axis deviation). The conduction vector demonstrated a bifascicular block, suggesting that the origin of the wide complex tachycardia was the left posterior fascicular focus. In addition, 2:1 ventriculoatrial conduction was seen, showing intermittent retrograde conduction from the ventricle to the atrium (arrows). (**B**) The 12-lead ECG ordered after cardioversion showed a sinus rhythm of 56 beats per minute, LAFB, deep T-wave inversions in the inferior leads, and ST-T segment abnormalities in the lateral leads.

characteristic ECG findings (**Table 1**). Left posterior fascicular ventricular tachycardia, occurring in up to 90% of cases, has a QRS complex that resembles a right bundle branch block (RBBB) morphology with left axis deviation, consistent with left anterior fascicular block. In approximately 10% of cases, fascicular ventricular tachycardia arises from the left anterior fascicle and has a QRS complex with a RBBB morphology and right-axis deviation due to concurrent left posterior fascicular block. Rarely, the origin is in the left upper septum, and the ECG shows a narrow QRS configuration and normal or right-axis deviation ($<$ 1% of cases).[8]

Fascicular ventricular tachycardia is believed to result from a macroreentry circuit involving the Purkinje system near the left posterior fascicle that is triggered by an abnormal slow pathway with verapamil sensitivity.[8,10] Because the reentry circuit does not involve the atrioventricular node; administration of adenosine has no effect.[11,12] On a cellular level, fascicular ventricular tachycardia appears to be primarily dependent on slow inward calcium channels in the Purkinje system.[13]

MANAGEMENT

Verapamil is effective in slowing and terminating episodes of fascicular ventricular tachycardia, and it remains the pharmacologic treatment of choice. Adenosine, lidocaine, and propranolol can be ineffective and should not be used if fascicular ventricular tachycardia is suspected.[5,11] Calcium channel blockers have been suggested as first-line

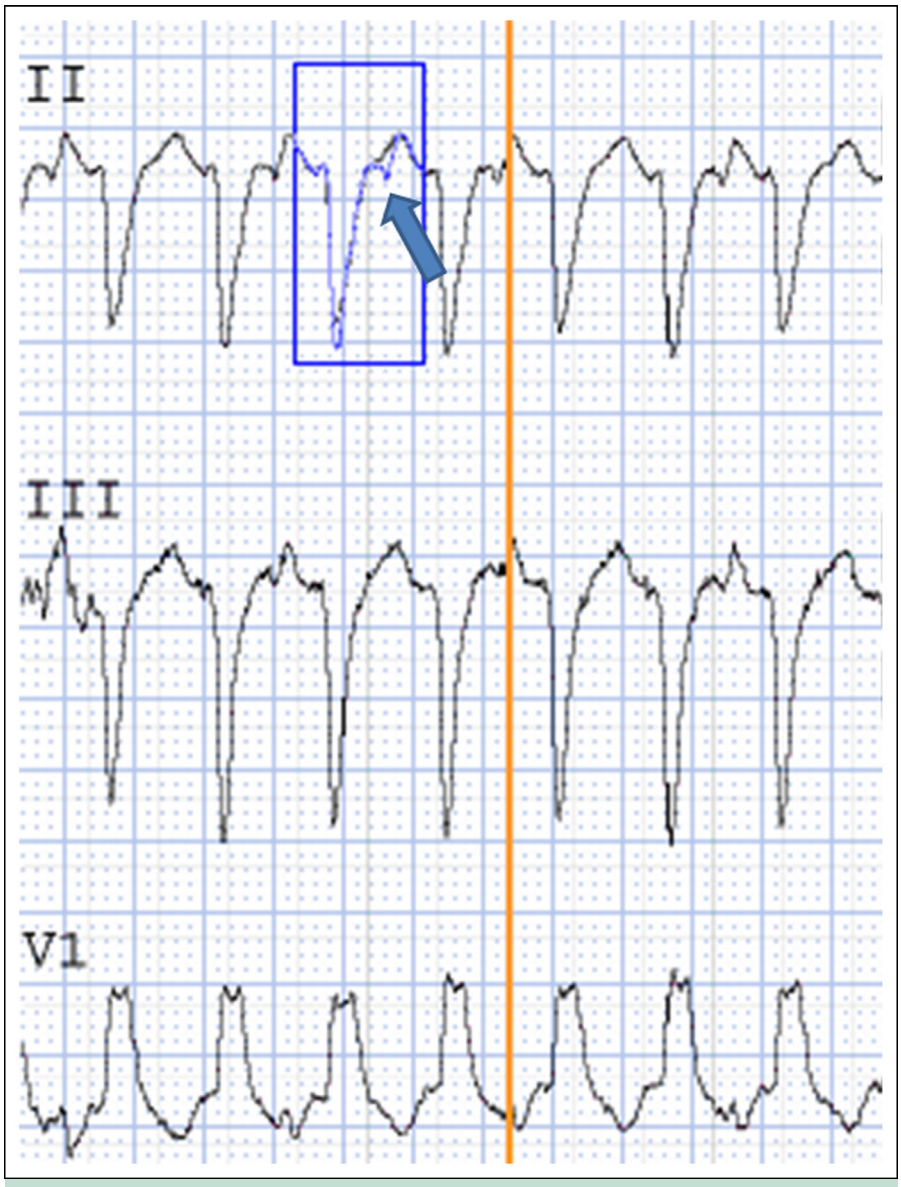

Figure 2 This magnified section of **Figure 1** concentrates on leads II, III, and V1 and demonstrates 2:1 ventriculoatrial conduction. An orange line is used to simultaneously align the retrograde p waves in the leads displayed. A QRS complex and a p wave are superimposed (in blue) on a QRS complex without retrograde atrial conduction to show the negative vector of the p wave (arrow) in lead II that is embedded within the up-sloping portion of the ST segment.

treatment for any hemodynamically stable patient without underlying ischemic heart disease who presents with broad complex tachycardia and RBBB.[14] Still, caution should be exercised in patients—especially in older patients—who present with an unknown diagnosis, as calcium channel blockers are contraindicated in ischemia-related ventricular tachycardia; deletrious hemodynamic effects can be provoked.[15] Cardiology consultation should be considered.

For long-term management of fascicular ventricular tachycardia, oral verapamil has been shown to be effective in mild to moderate cases, but it may be ineffective in patients with severe symptoms.[3,16] For those intolerant of or noncompliant with oral therapy or who have refractory symptoms, catheter-based ablation has been found to be an effective and safe option. While there is concern for tachycardia-induced cardiomyopathy in severe cases, the overall long-term prognosis of fascicular ventricular tachycardia is excellent with a very low risk for lethal ventricular arrhythmias and/or sudden cardiac death.[16]

The patient underwent successful radiofrequency ablation of the focus of the fascicular ventricular tachycardia,

Table 1 Characteristic ECG Findings[4,7-9]

	Fascicular VT	Ischemic VT
Axis	Left axis deviation (most cases)	Extreme axis deviation
QRS Duration	100-140ms	>140 ms
RS Interval	<80 ms	>100 ms

and was discharged on extended released diltiazem 120 mg daily. He has had no further recurrences.

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References

1. Trappe HJ, Brugada P, Talajic M, et al. Prognosis of patients with ventricular tachycardia and ventricular fibrillation: role of the underlying etiology. *J Am Coll Cardiol*. 1988;12:166-174.
2. Steinman RT, Herrera C, Schuger CD, Lehmann MH. Wide QRS tachycardia in the conscious adult: ventricular tachycardia is the most frequent cause. *JAMA*. 1989;261:1013-1016.
3. Ohe T, Shimomura K, Aihara N, et al. Idiopathic sustained left ventricular tachycardia: clinical and electrophysiologic characteristics. *Circulation*. 1988;77:560-568.
4. Andrade FR, Eslami M, Elias J, et al. Diagnostic clues from the surface ECG to identify idiopathic (fascicular) ventricular tachycardia: correlation with electrophysiologic findings. *J Cardiovasc Electrophysiol*. 1996;7:2-8.
5. Lin FC, Finley CD, Rahimtoola SH, Wu D. Idiopathic paroxysmal ventricular tachycardia with a QRS pattern of right bundle branch block and left axis deviation: a unique clinical entity with specific properties. *Am J Cardiol*. 1983;52:95-100.
6. Belhassen B, Rotmensch HH, Laniado S. Response of recurrent sustained ventricular tachycardia to verapamil. *Br Heart J*. 1981;46:679-682.
7. Brugada P, Brugada J, Mont L, Smeets J, Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation*. 1991;83:1649-1659.
8. Nogami A. Purkinje-related arrhythmias part I: monomorphic ventricular tachycardias. *Pacing Clin Electrophysiol*. 2011;34:624-650.
9. Chew HC, Lim SH. Verapamil for ventricular tachycardia. *Am J Emerg Med*. 2007;25:572-575.
10. Maruyama M, Tadera T, Miyamoto S, Ino T. Demonstration of the reentrant circuit of verapamil-sensitive idiopathic left ventricular tachycardia: direct evidence for macroreentry as the underlying mechanism. *J Cardiovasc Electrophysiol*. 2001;12:968-972.
11. Elswick BD, Niemann JT. Fascicular ventricular tachycardia: an uncommon but distinctive form of ventricular tachycardia. *Ann Emerg Med*. 1998;31:406-409.
12. Griffith MJ, Garratt CJ, Rowland E, Ward DE, Camm AJ. Effects of intravenous adenosine on verapamil-sensitive "idiopathic" ventricular tachycardia. *Am J Cardiol*. 1994;73:759-764.
13. Lerman BB, Stein KM, Markowitz SM. Mechanisms of idiopathic left ventricular tachycardia. *J Cardiovasc Electrophysiol*. 1997;8:571-583.
14. Wang JC, Lim SH, Teo WS, Anantharaman V. Calcium channel blockers as first line treatment for broad complex tachycardia with right bundle branch block: ingenuity or folly? *Resuscitation*. 2002;52:175-182.
15. Buxton AE, Marchlinski FE, Doherty JU, Flores B, Josephson ME. Hazards of intravenous verapamil for sustained ventricular tachycardia. *Am J Cardiol*. 1987;59:1107-1110.
16. Ohe T, Aihara N, Kamakura S, Kurita T, Shimizu W, Shimomura K. Long-term outcome of verapamil-sensitive sustained left ventricular tachycardia in patients without structural heart disease. *J Am Coll Cardiol*. 1995;25:54-58.