Wide-complex tachycardia in a patient with old myocardial infarction

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A 79-year-old male patient presented with complaints of sudden-onset palpitations to the emergency department. There was no history of syncope or seizure. The patient was hypertensive, had a history of anterior wall myocardial infarction, and had undergone percutaneous coronary intervention 15 years back. The patient had dyspnea on exertion (New York Heart Association class II) with an ejection fraction of 40%-45%. The electrocardiogram (ECG) available revealed a wide complex tachycardia (WCT) which terminated spontaneously. The patient remained hemodynamically stable, and a cardiology referral was sought for the need of antiarrhythmic therapy and subsequent implantable cardioverter defibrillator (ICD). His serum electrolytes levels were normal. He had been on medical therapy for heart failure and coronary artery disease including beta-blockers, angiotensin receptor blockers, and statins.

As per the ECG provided (Figure 1), what is the diagnosis and what should be the management?

1 | COMMENTARY

The most common WCT in patients with underlying structural heart disease is ventricular tachycardia (VT). This assumption may be correct for the acute management, but differentiation of VT from supraventricular tachycardia (SVT) with aberrancy has important implications on the prognosis and long-term management. The initial ECG available at presentation shows a WCT with LBBB morphology in V1, normal QRS axis, normal R-wave progression in precordial leads, and negative QRS in lead aVR (Figure 1A). These features suggest the diagnosis of SVT over VT. Careful examination shows variability in the RR intervals with the difference between the first and the fourth RR interval being 64 milliseconds (supporting information). No P waves are seen between the QRS complexes. A wide complex irregular tachycardia without obvious P waves has a limited differential diagnosis with the foremost being atrial fibrillation (AF) with a coexistent conduction abnormality. This conduction abnormality can be an underlying bundle branch block, functional bundle branch block occurring at fast or extremely slow rates (right bundle branch block in nearly 85% cases), pre-excitation over an accessory pathway, delayed conduction secondary to drugs (class IC and class III anti-arrhythmic agents), and electrolyte disturbances such as hyperkalemia. Monomorphic VTs may have a small variability in the RR intervals as observed during electrophysiology studies. However, obvious RR variability is rare and a variation greater than 50-60 milliseconds has good accuracy for differentiating AF from VT. Most ICDs employ similar rate stability criteria.

The second ECG supports the diagnosis (Figure 1B). The rhythm is irregular with an undulating baseline and no distinct P waves. Wide QRS complexes occur irregularly and the intermittent narrow QRS complexes are normally conducted QRS complexes which can be mistaken with fusion beats. In patients with AF, aberrant conduction is occasionally seen after a long cycle described as the Ashman’s phenomenon. It occurs because of the time dependence of refractoriness on the preceding cycle length and often manifests as RBBB as the right bundle has a longer effective refractory period. However, in this case the aberrant conduction appears intermittently without dependence on the previous cycle. This phenomenon should be differentiated from the Ashman’s phenomenon and rate-dependent aberrancy occurring in normal hearts during tachycardia. It is independent of preceding cycle length, appears at comparatively slow rates, usually of LBBB morphology, may occur without significant changes in cycle length, and almost always occurs in patients who have underlying heart disease.

The tachycardia terminated spontaneously, and subsequent ECG showed normal sinus rhythm with narrow QRS complexes (Figure 1C). A 24-hour Holter study was done and showed intermittent LBBB during sinus rhythm (Figure 2). The final diagnosis was paroxysmal AF with aberrant conduction because of an underlying diseased left bundle. The
CHADS2VASc score was 4, and the patient was prescribed oral anticoagulants. Misdiagnosis as VT would have resulted in an altogether different management strategy, possibly omitting oral anticoagulation and predisposing the patient to a higher risk of thromboembolic events.

Anti-arrhythmic drugs can be used for paroxysmal AF either as a pill-in-the-pocket strategy or to maintain sinus rhythm after cardioversion.

Class IC anti-arrhythmic agents, such as flecainide and propafenone, are the most commonly used drugs but are contraindicated in patients with structural heart disease, ischemic heart disease, or heart failure. These agents produce use-dependent QRS prolongation and QRS duration should be monitored in patients while on therapy. They should be used with caution in patients with underlying conduction disturbances.
including sinus node disorders, atrioventricular nodal disease, and infranodal or intraventricular conduction abnormalities as they may produce advanced heart blocks in such conditions. These drugs should not be used in patients with intermittent LBBB, like in the present case, as they may not only worsen the conduction disturbance but also make differentiation of the ECG abnormality from drug toxicity difficult.

**FIGURE 2** 24-hour Holter study. Tracings taken at different times from the 24-hour Holter recording showing intermittent left bundle branch block occurring during normal sinus rhythm (NSR), independent of RR interval, suggestive of a diseased conduction system

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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