Diagnostic Challenge

Hemodynamically stable wide-complex tachycardia in a young patient

Sameer Shaikh, MD*; Teresa Chan, MD*; Saeed Darvish-Kazem, MD†; Craig Ainsworth, MD†

Wide-complex tachycardia (WCT) is a condition that requires prompt recognition and management in the emergency department. Advanced Cardiovascular Life Support (ACLS) guidelines offer a systematic approach to managing patients with such arrhythmias. We describe the case of a young man with hemodynamically stable WCT unresponsive to standard ACLS interventions. Based on his electrocardiographic features, the patient was felt to have Belhassen or fascicular ventricular tachycardia (VT), a calcium channel blocker–sensitive VT, and was successfully cardioverted with intravenous verapamil. In the rare setting of refractory WCT, early cardiologist consultation should be sought and deviation from ACLS algorithms may be warranted.

CASE HISTORY

A 38-year-old man presented to the emergency department by ambulance with a chief complaint of acute palpitations. The palpitations began at rest, approximately 30 minutes prior to his presentation, and were continuous. The patient denied any associated chest pain, dyspnea, nausea, diaphoresis, presyncope, or syncope. He had no significant past medical history and denied having previous episodes of palpitations. He was taking no medications, and there was no history of recent recreational drug use.

On examination, the patient was alert and oriented, had no signs of respiratory distress, and was afebrile. His heart rate was 215 beats/min, respiratory rate was 20 breaths/min, and oxygen saturation was 100%. Cardiovascular examination revealed normal heart sounds, with no extra sounds or murmurs. His respiratory, peripheral vascular, and neurologic examinations were unremarkable.

A 12-lead electrocardiogram is shown in Figure 1. Subsequently obtained laboratory investigations revealed a normal complete blood count, renal function, and electrolytes. Extended electrolytes and thyroid-stimulating hormone were normal. Initial troponin I was negative at 0.01 μg/L.

Because of the patient's hemodynamic stability and lack of end-organ dysfunction, a discussion was initiated with him regarding options for cardioversion. The patient opted for an attempt at chemical cardioversion. Procainamide was chosen; however, there was no effect from an appropriately dosed intravenous infusion. The decision was then made to attempt electrical cardioversion under procedural sedation. Midazolam and fentanyl were administered, and biphasic synchronized cardioversion with 100 J was administered, without success. Incremental doses of 150 and 200 J were subsequently administered but also failed to result in cardioversion.

QUESTION

What is the most likely diagnosis?

a. Right ventricular outflow tract ventricular tachycardia
b. Supraventricular tachycardia with aberrancy

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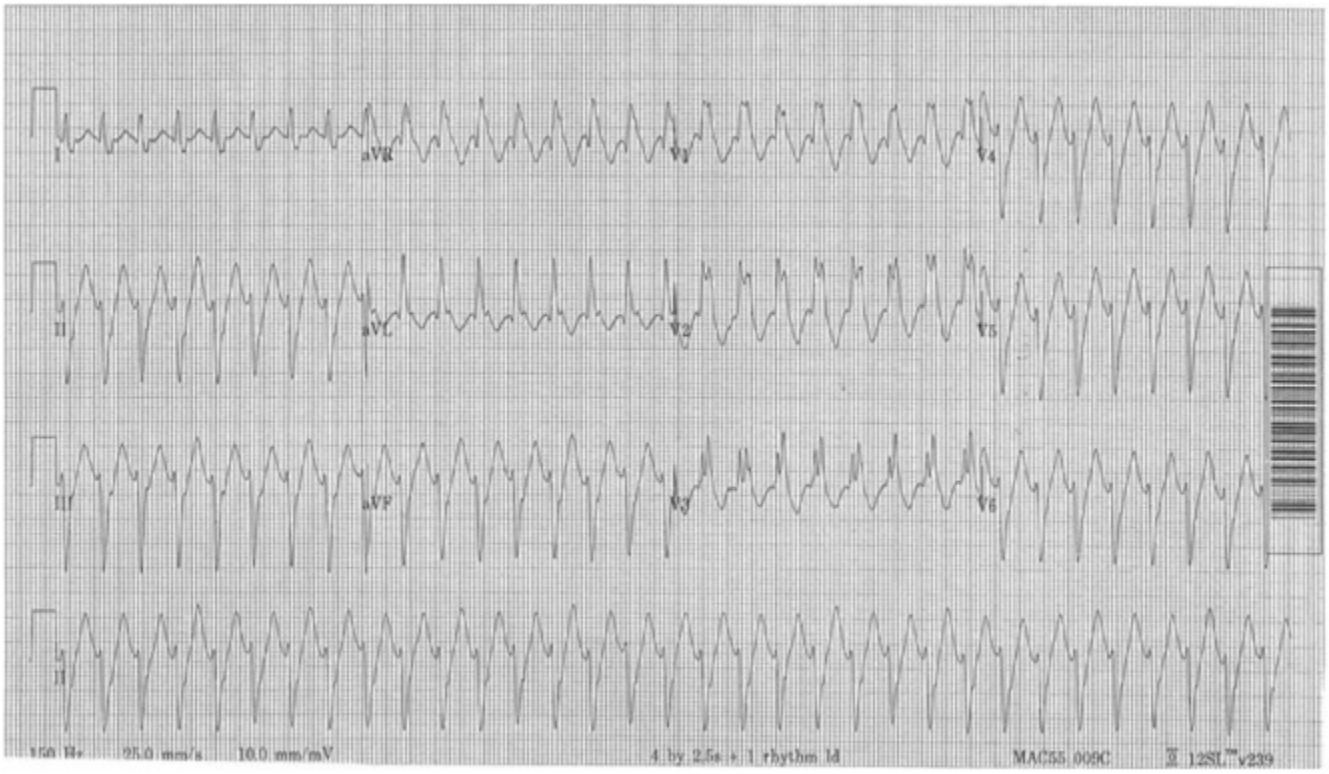

Figure 1. Initial 12-lead electrocardiogram showing wide-complex tachycardia.

c. Supraventricular tachycardia with pre-excitation
d. Fascicular ventricular tachycardia
e. Scar-mediated ventricular tachycardia

For the answer to this challenge, see next page.

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ANSWER AND DISCUSSION

The correct answer in this case is d) fascicular ventricular tachycardia (VT).

Patients with wide-complex tachycardia (WCT) often present a diagnostic dilemma for emergency physicians. According to Advanced Cardiovascular Life Support (ACLS) guidelines, WCT should be divided based on clinical stability. Unstable patients (those with hypotension [systolic blood pressure < 90 mm Hg], ischemic chest pain, heart failure, shock, or altered mental status) should undergo immediate synchronized electrical cardioversion, whereas stable patients can be initially treated with intravenous medications. The four recommended medications for stable, regular WCT are adenosine, amiodarone, sotalol, and procainamide (Table 1).[1] In patients not responding to first-line medications, alternate causes should be considered and cardiologist consultation is advised. Medications such as intravenous calcium channel blockers are generally avoided in WCT because patients with VT should be presumed to have structural heart disease. In such patients, medications with a negative inotropic effect, such as calcium channel blockers, may precipitate hemodynamic compromise and occasionally lead to death.[1]

Belhassen-type VT

Belhassen or fascicular VT is a unique type of WCT that was first described in 1981 by Belhassen and colleagues.[2] Although most cases of VT are associated with structural heart disease, up to 10% of patients presenting with VT have no evidence of structural heart disease on electrocardiogram, exercise stress test, and echocardiogram.[3] Belhassen VT is the most common form of idiopathic VT originating in the left ventricle and represents up to 15% of all idiopathic VTs.[4] Patients with this form of VT are generally young, healthy men between the ages of 15 and 40 and are usually hemodynamically stable during the episodes.[5]

Belhassen VT is a monomorphic tachycardia that originates in the fascicles of the left ventricle. Electrocardiographic findings include a right bundle branch block (RBBB) pattern, a relatively narrow QRS duration (130–160 ms) and RS interval (60–80 ms) when compared to other forms of VT, and axis deviation depending on the anatomic site of re-entry.[6] In patients with left-axis deviation, the re-entry location is usually the left posterior fascicle, whereas in patients with right-axis deviation, the location is usually the left anterior fascicle.[7]

A specific hallmark of Belhassen VT is that it usually does not revert spontaneously to normal sinus rhythm. Belhassen VT is most frequently localized to a small region of re-entry located in the posteroinferior left ventricle close to the posterior fascicle of the left bundle branch. Because of its rapid response to intravenous verapamil, it has been suggested that this arrhythmia is dependent on slow inward calcium channels in the ventricular Purkinje fibers.[4,6] Although some evidence suggests that vagal maneuvers and other antiarrhythmics, such as sotalol and amiodarone, may also be effective, verapamil is the hallmark of therapy.[8,9]

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Table 1. ACLS-recommended medications for stable wide-complex tachycardias

Medication	Recommended dose	Mechanism of action	Adverse effects
Adenosine*	6 or 12 mg IV push	Blocks AV node	Flushing, presyncope, metallic taste, transient asystole
Amiodarone	150 mg IV over 10 min, then 1 mg/min infusion for first 6 h	Class III antiarrhythmic	Hypotension Long-term effects on multiple systems
Procainamide	20–50 mg/min, with maximum dose of 15–17 mg/kg IV load over 30–60 min, then 1–4 mg/min IV infusion	Class IA antiarrhythmic	Hypotension QRS widening
Sotalol†	1.5 mg/kg IV over 5 min	Class III antiarrhythmic	Prolonged QT

ACLS =Advanced Cardiovascular Life Support; AV = atrioventricular; IV = intravenous.
*Only recommended in regular, monomorphic wide-complex tachycardia.
†Not available in Canada.

Back to the case

The patient was referred to the in-hospital cardiology service and was administered 6, 12, and 12 mg of intravenous adenosine with no response. On transfer to the cardiac care unit, the patient was given 10 mg of intravenous verapamil over 2 minutes, which resulted in immediate conversion to normal sinus rhythm at a rate of 96 beats/min.

In this case, the patient's electrocardiogram had findings highly suggestive of Belhassen VT, with an RBBB, relatively narrow QRS duration (138 ms), and left-axis deviation (Figure 2). A postcardioversion

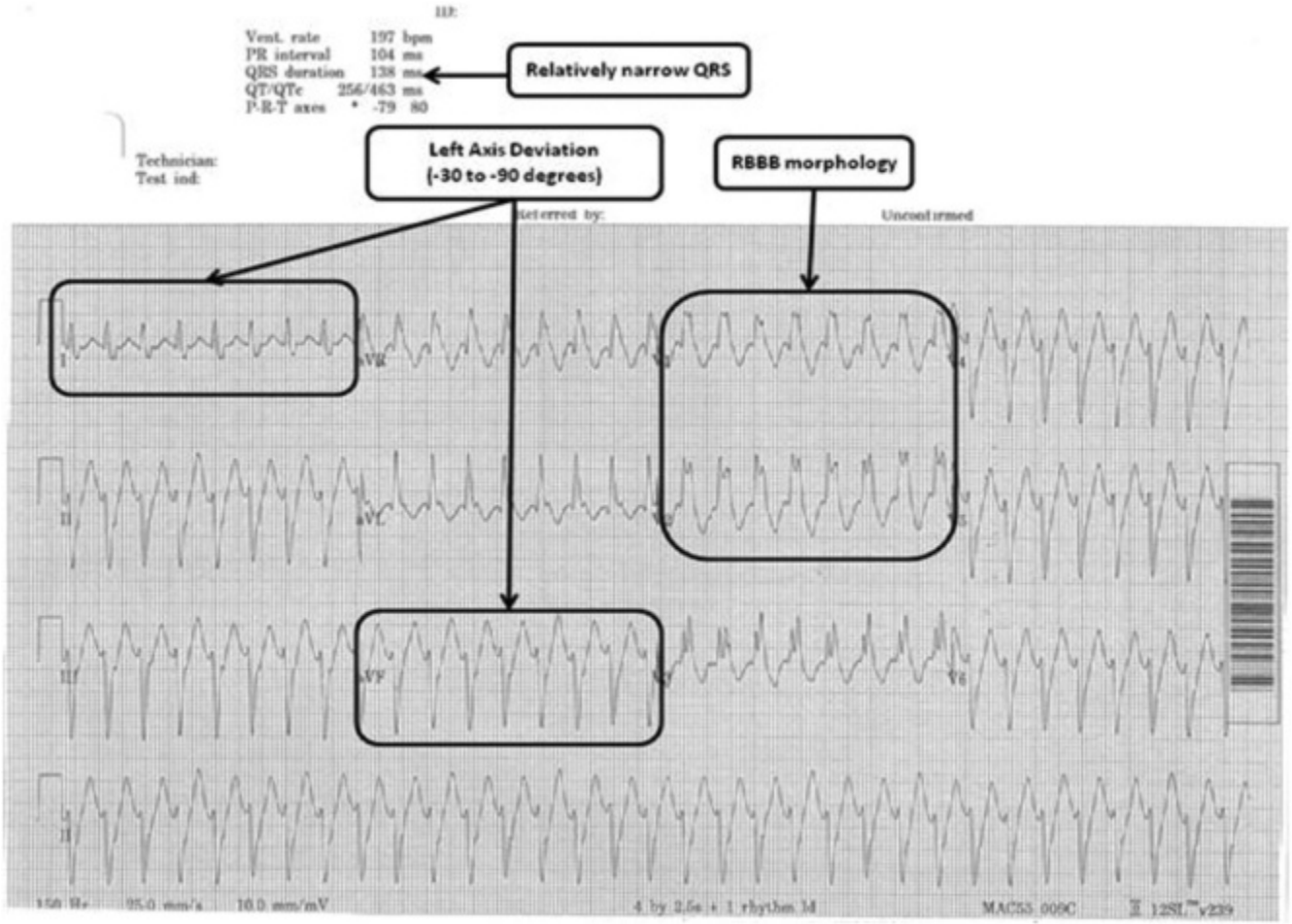

Figure 2. The patient's initial electrocardiogram shows signs of Belhassen ventricular tachycardia with an right bundle branch block (RBBB), relatively narrow QRS duration (138 ms), and left-axis deviation.

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echocardiogram was normal, other than a slightly decreased ejection fraction of 45 to 50%, which subsequently normalized, suggesting that it was transient tachycardia-mediated dysfunction. The patient was discharged home on oral verapamil with an appointment for follow-up with an electrophysiologist. Ablation, which targets the re-entry tract in the fascicles, is the only definitive therapy for this condition.[10]

CONCLUSION

WCT is a condition that requires prompt management in the emergency department. In patients unresponsive to standard ACLS guidelines, early cardiologist consultation is warranted to assess for uncommon causes, such as Belhassen VT. Belhassen VT is a monomorphic VT originating in the fascicles of the left ventricle that usually presents with hemodynamic stability. Hallmark electrocardiogram findings of the disease include RBBB, a relatively narrow QRS and RS interval, and axis deviation. In patients with this condition, the most effective acute therapy is intravenous verapamil. Successful management of such patients in the emergency department may require cautious deviation from ACLS guidelines.

Competing interests: None declared.

Keywords: arrhythmia, cardiology, continuing education, electrocardiogram

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