Challenges in Clinical Electrocardiography

Chest Pain and Wide QRS Tachycardia

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Case Presentation

A patient in their 70s with diabetes presented with 3 hours of chest pain. The patient had a history of acute myocardial infarction 5 years before, with stenting of the right coronary artery and left anterior descending coronary artery. At the current hospital admission, the patient denied any dyspnea but felt dizzy while in the sitting position. The patient's supine blood pressure measured 100/55 mm Hg. Laboratory test results revealed a 83 ng/L serum troponin T level (upper limit of normal [ULN], 14 ng/L; to convert to µg/L, multiply by 1) and 76 U/L serum creatine kinase level (ULN, 170 U/L; to convert to µkat/L, multiply by 0.0167).

The electrocardiogram (ECG) on admission showed a regular tachycardia at a rate of 135 beats per minute. There was an unusual right bundle branch block morphology with right axis deviation (**Figure**, A). A repeated ECG about 10 minutes later showed an identical heart rate, with similar QRS morphology in the chest leads. However, the frontal plane QRS axis had changed substantially (Figure, B).

Questions: What was the cause of the wide QRS complex tachycardia and abrupt change in the QRS axis? What were the best treatment options for the patient?

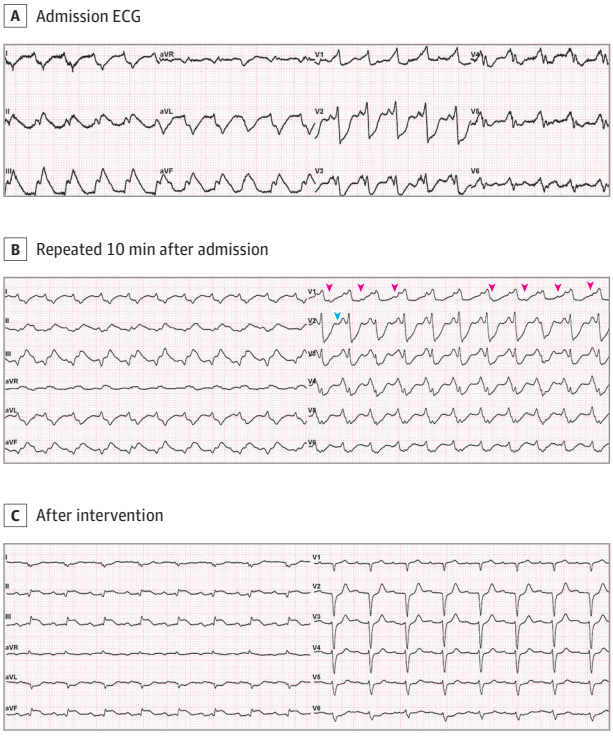
Interpretation

The ECG on presentation showed a regular wide QRS complex tachycardia with QRS duration of more than 160 milliseconds and a rate of 135 beats per minute. The ECG performed by the emergency department physician was initially interpreted as supraventricular/sinus tachycardia with right bundle branch block and precordial ST segment depression. However, on closer inspection, there were at least 2 important ECG features that suggested ventricular tachycardia. In the first ECG (Figure, A), the apparent P waves, the small bumps seen in leads V_2 and V_3, occurred simultaneously with the onset of the QRS complexes in the V_1 lead, indicating that these positive waves were part of the wide and notched QRS complexes rather than separate P waves. A second ECG (Figure, B) demonstrated a wide QRS complex tachycardia with deep ST depression in the precordial leads. In this ECG, the lead V_1 hid regular small upright P waves (see arrowheads) at a rate of 100 beats per minute, dissociated from the wide QRS complexes. Atrioventricular (AV) dissociation is a defining characteristic for ventricular tachycardia. The identical rate and near-identical horizontal plane QRS morphology but changing QRS axis suggested ventricular tachycardias with different exit points.

Clinical Course

Given the stable blood pressure, the patient received lidocaine, 100 mg, which was administered intravenously over 2 minutes. Within minutes, the tachycardia terminated, but the chest pain did not resolve. The ECG now depicted sinus rhythm with first-degree AV block and inferior ST segment elevation myocardial infarction (Figure, C). Treatment with intravenous heparin was initiated, and the patient was transferred to the cardiac catheterization laboratory, where urgent coronary angiography results showed an occluded right coronary artery distal to the previously implanted stent. The peak creatine kinase level was 3700 U/L (UNL, 170 U/L). The left ventricular ejection fraction decreased to 10%. Because of progressive heart failure and concomitant kidney failure, the patient died on the fifth hospital day.

Figure. Serial Electrocardiograms (ECGs) of a Patient Who Presented With Prolonged Chest Pain



A, The admission ECG showed an unusual regular wide QRS tachycardia at a rate of 135 beats per minute. B, Repeated ECG 10 minutes later revealed an identical heart rate but different morphology in the frontal plane (QRS axis, −30° to −60°; QRS duration, 220 milliseconds). Pink arrowheads indicate dissociated P waves. The blue arrowhead indicates the onset of QRS in lead V_2. After this arrowhead, the positive little bump is not a P wave but part of the QRS. C, After 1 treatment with intravenous lidocaine, 100 mg, the ECG demonstrated sinus rhythm with first-degree atrioventricular block, abnormal Q waves in leads I and aVL, and ST segment elevation in the inferior leads.

Discussion

Several studies have shown that wide-complex tachycardia identification algorithms perform poorly at the bedside and are frequently associated with misdiagnosis and inappropriate treatment.[1,2] There are several common scenarios associated with misdiagnosis. First is not recognizing AV dissociation, a crucial clue for ventricular tachycardia that is present in up to 50% of all wide-complex tachycardias.[2] Frequently, there is no attempt to scrutinize for dissociated P waves or, as in the case presented, parts of the QRS complexes or ST segments are incorrectly identified as P waves.

Second, the cause of the wide-complex tachycardia is incorrectly deduced from the patient's relatively benign clinical and hemodynamic status. In patients with structural heart disease, regardless of the clinical status, most wide-complex tachycardia is ventricular tachycardia.[2] Third, because ventricular tachycardia in acute coronary syndrome is usually polymorphic,[3] a ventricular origin of monomorphic tachycardia with normal QT duration is not even considered. Although rare, the SWEDE HEART registry reported early monomorphic ventricular tachycardia during ST elevation myocardial infarction in 1.5% of the cases. These were high-risk patients with high all-cause mortality during 8 years of follow-up.[4] In ventricular tachycardia with hemodynamic instability, direct current electrical shock followed by intravenous amiodarone is the most effective treatment.[5] However, in stable ventricular tachycardia that is associated with an acute ischemic syndrome, intravenous lidocaine can also be effective.[6]

Take-home Points

- In a case of wide QRS tachycardia, a ventricular origin should always be considered if the patient has known structural heart disease.
- Patients with ventricular tachycardia can be clinically stable even in the setting of acute coronary syndrome.
- Atrioventricular dissociation is the most reliable sign in identifying ventricular tachycardia. If you are not sure, repeat the ECG with a longer rhythm strip.
- Although polymorphic ventricular arrhythmias are most often seen in acute ischemia, monomorphic ventricular tachycardia can also occur. This is especially true if there is scar tissue present from a previous event.
- During wide QRS tachycardia, it is difficult to interpret repolarization abnormalities. A significant ST-T change occurring at similar heart rates usually indicates acute ischemic origin of the chest pain.

ARTICLE INFORMATION

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