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The Clinical Picture

Wide QRS complex rhythm with pulseless electrical activity

Ventricular rate	156	BPM	Undetermined rhythm
PR interval	*		Right bundle branch block
QRS duration	208	ms	Inferior infarct, age undetermined
QT/QTc	204/328	ms	Abnormal ECG
P-R-T axes	* 255	0	No previous ECGs available

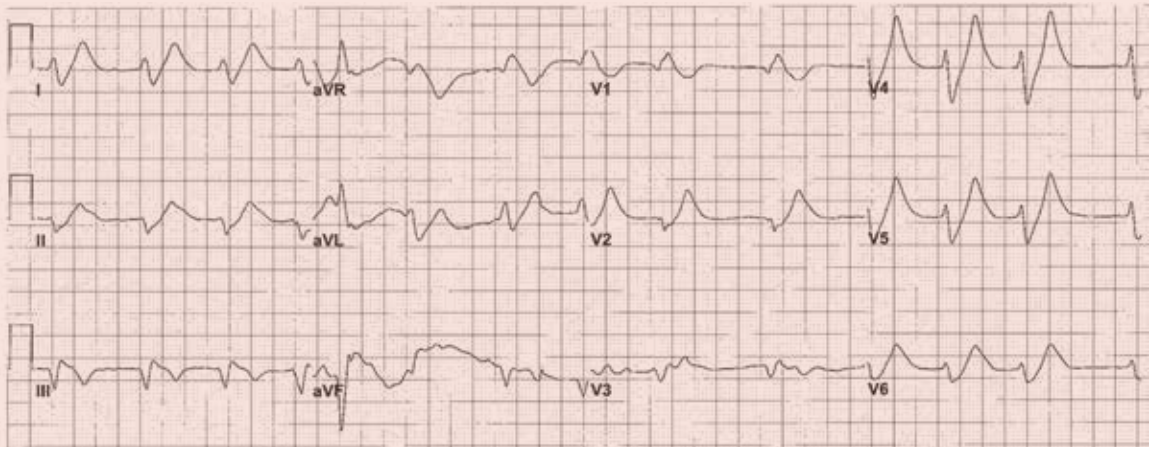

FIGURE 1. At presentation, the standard 12-lead electrocardiogram demonstrated an irregular wide-QRS-complex rhythm with features of right-superior-axis deviation and right bundle branch block. Although the interpretation software indicated a ventricular rate of 156 bpm, inspection reveals the rate to be only half that (ie, "double counting" of the heart rate). The serum potassium concentration at the time of this test was 8.9 mmol/L.

A 64-YEAR-OLD MAN with chronic kidney disease and recent upper gastrointestinal hemorrhage suffered pulseless electrical activity and cardiac arrest. Cardiopulmonary resuscitation was started, with three attempted but failed electrical cardioversions. Return of spontaneous circulation required prolonged resuscitation efforts, including multiple rounds of epinephrine, calcium, and sodium bicarbonate. The standard 12-lead electrocardiogram (**FIGURE 1**) showed an irregular wide-QRS-complex rhythm, with right bundle branch block and right-superior-axis deviation.

What was the cause of the pulseless electrical activity and the features on the electrocardiogram?

The presentation of cardiac arrest with pulseless electrical activity usually has a grave prognosis, and in the acute setting, the cause may be difficult to establish. However, several conditions that cause this presentation have treatments that, applied immediately, can lead to quick and sustained recovery.[1]

Electrocardiography can be a powerful tool in the urgent evaluation of pulseless electrical activity.[2,3] Narrow-QRS-complex pulseless electrical activity is often

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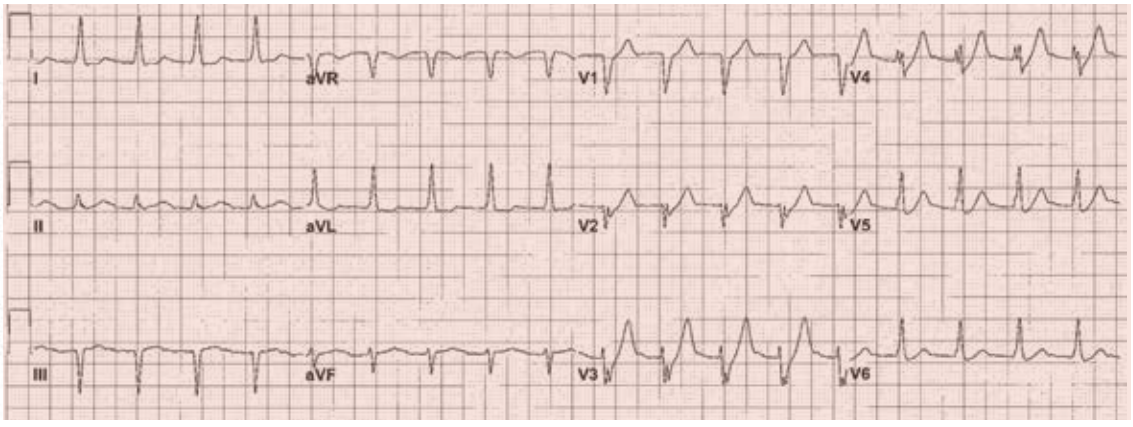

FIGURE 2. After aggressive treatment of the hyperkalemia, the QRS complexes narrowed and the QRS axis normalized, but the chest leads demonstrated J-point depression followed by upsloping ST segments, culminating in tall, peaked T waves. This pattern is also occasionally seen in acute occlusion of the left anterior descending coronary artery. The serum potassium at this time was 7.1 mmol/L.

caused by mechanical factors such as cardiac tamponade, tension pneumothorax, pulmonary embolism, and major hemorrhage.[3] Pulseless electrical activity associated with a wide QRS complex and marked axis deviation, as in this patient, is usually the result of a metabolic abnormality, most often hyperkalemia[3]; additional indicators of severe hyperkalemia include ST-segment elevation in the anterior chest leads (including the Brugada pattern[4]) and, as in this patient, "double counting" of the heart rate by the interpretation software (FIGURE 1).[5,6]

ECG can be a powerful tool in the urgent evaluation of pulseless electrical activity

Based on the suspicion of a metabolic cause, the serum potassium was tested and was 8.9 mmol/L (reference range 3.5–5.0). The patient was given intravenous calcium, sodium bicarbonate, glucose, and insulin, and 2 hours later the serum potassium had decreased to 7.1 mmol/L. At that time, the electrocardiogram (FIGURE 2) showed a regular rhythm with ectopic P waves, probably an ectopic atrial tachycardia. There were now narrow QRS complexes with J-point depression, upsloping ST segments, and tall, hyperacute T waves in the chest leads—a pattern recently described in proximal left anterior descending coronary artery occlusion.[7] The electrocardiographic similarities in hyperkalemia and acute myocardial infarction are probably the result of potassium accumulation in the ischemic myocardium associated with acute coronary occlusion.[7]

The patient had a full recovery, both clinically and on electrocardiography. ■

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