

IMAGING VIGNETTE

ADVANCED

ECG CHALLENGE

An Ominous ECG



What Is the Mechanism?

Specific Subtype of Ventricular Separation

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ABSTRACT

Homologous ventricular electrical separation is a rare electrocardiogram manifestation; it is defined as a specific subtype of ventricular separation caused by excitation that originates from the supraventricular rhythm and travels through the His-Pukenje system, resulting in severe asynchrony of biventricular depolarization and production of 2 QRS complexes. It always indicates irreversible cardiac impairment and electrical instability. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2023;23:102006) © 2023 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

A 73-year-old male patient was urgently admitted to hospital after experiencing chest pain for 15 h with dyspnea. The patient had a history of hypertension. On arrival, he was drowsy with a heart rate of 68 beats/min, blood pressure of 86/42 mm Hg, and respiratory rate of 20 breaths/min with an SpO₂ of 95% in room air. Laboratory tests showed that troponin I levels were markedly increased to 53.947 ng/mL (reference value: 0-0.026 ng/mL). The electrocardiogram (ECG) on admission is shown in **Figure 1A**. Fifteen minutes after admission, he suddenly lost consciousness and the second ECG was obtained after cardiopulmonary resuscitation (**Figure 1B**). Although aggressive treatment was provided, the patient's blood pressure and heart rate continued to decrease and the patient eventually died.

What is the diagnosis of the ECG (**Figure 1B**)?

- A) Homologous ventricular electrical separation
- B) Ischemic J-wave
- C) Junctional rhythm with complete right bundle branch block
- D) Accelerated idioventricular rhythm

The answer is A: homologous ventricular electrical separation.

DISCUSSION

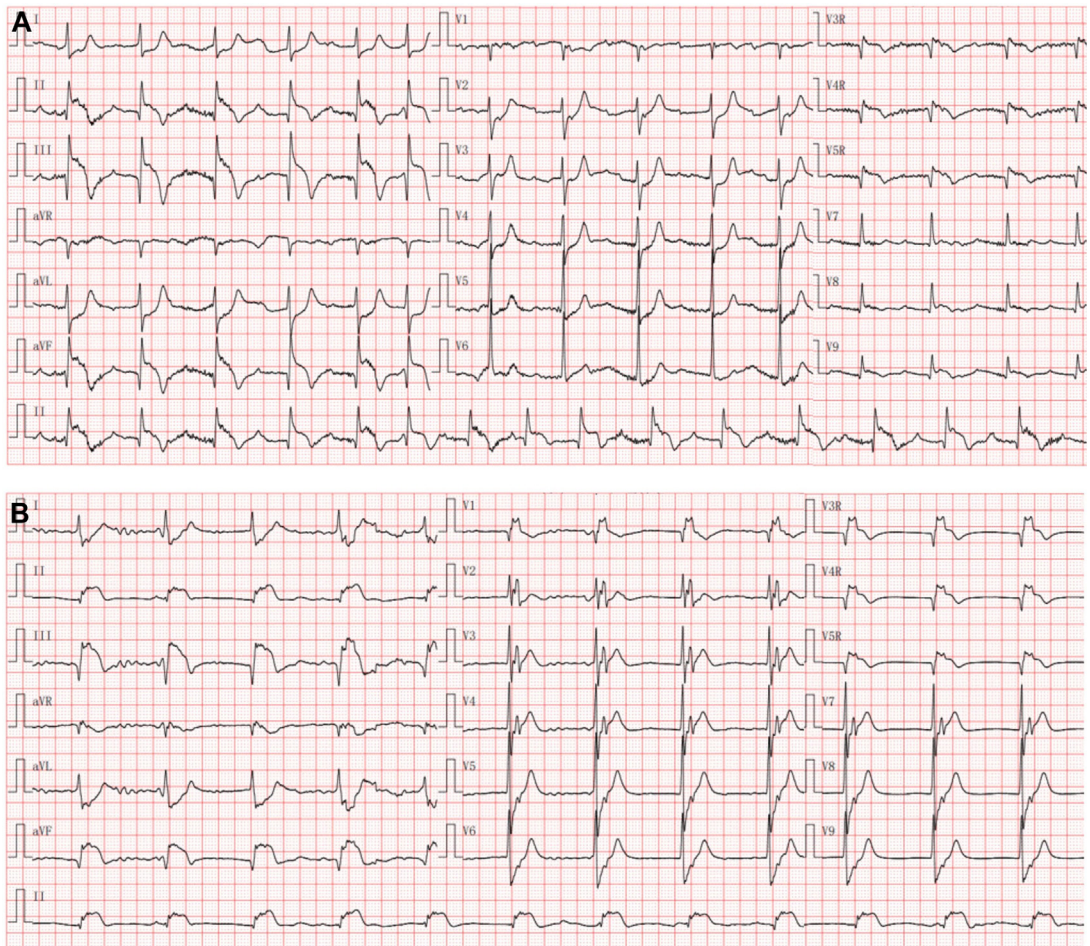
The rhythm strip in **Figure 1A** shows the sinus rhythm with second-degree atrioventricular block (mostly in a 2:1 pattern). The QRS morphology in inferior, right precordial, and posterior leads are QR, Qr, and qR patterns, respectively, was accompanied with ST-segment elevation, and the presumed culprit vessel was the right coronary artery. The rhythm strip in **Figure 1B** shows junctional rhythm. A typical tombstone-like pattern in

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**ABBREVIATIONS
AND ACRONYMS****ECG** = electrocardiogram**HVES** = homologous
ventricular electrical separation

inferior leads indicates the rapid progression of transmural injury to complete myocardial infarction. A thorough analysis of the ECG demonstrated an extremely wide QRS complex composed of 2 separated waves in leads V₂, V₃, V₄, and V₇, the amplitude of the first partial is higher than the second partial, and the terminal of the QRS complex widened with a notch in the remaining leads. Given the fact that the patient had no intracardiac device or electrophysiology catheter to document the intracardiac activation, we can only speculate that the 2 deflections of the QRS complexes visible in the precordial leads represent the depolarization of each of the ventricles occurring in parallel. Because the entire ventricle is excited from the supraventricular rhythm that travels through the His-Pukenje system, resulting in severe asynchrony of biventricular depolarization and the production of 2 QRS complexes, this particular type of ventricular separation is known as homologous ventricular electrical separation (HVES). The underlying mechanism of HVES includes severe ischemia, fibrosis, and drug toxicity. It always indicates irreversible cardiac impairment and severe electrical instability, together with an extremely poor prognosis.

FIGURE 1 Electrocardiographic Findings

12-lead electrocardiogram obtained at (A) time of admission and (B) after cardiopulmonary resuscitation.

A review of the literature showed that this case represented a rare arrhythmia that has only been sporadically reported. The first case of HVES was caused by overdose of procainamide.¹ Other causes of HVES include deterioration of heart failure² and extensive substrate modification of ventricular tachycardia.³ In the present case, acute occlusion of the right coronary artery led to ischemia in its innervation, specifically the conduction system of the heart, manifested by sinus arrest, atrioventricular block, intraventricular conduction disturbances, and massive myocardial ischemia. This resulted in severe asynchrony in the contraction of the 2 ventricles.

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