

Abnormal Electrocardiogram in a Man Presenting With Dyspnea and Chest Pain

Daniel W. Nelson, MD¹; Ryley McPeters, MD¹; Atul Singla, MD¹

¹Tulane University School of Medicine and Southeast Louisiana Veterans Health Care System, New Orleans, Louisiana

A 70-year-old man with a history of smoking presented at the emergency department with dyspnea and a 24-hour history of intermittent chest pain that had acutely worsened. His heart rate was 128 beats/min; blood pressure, 114/81 mmHg; respiratory rate, 24 breaths/min; and oxygen saturation, 85% on 4 L of oxygen. He had bibasilar crackles and a grade 3/6 holosystolic murmur over the left sternal border. A basic metabolic panel showed nothing unusual. A coronavirus nasopharyngeal swab test was negative. The patient's N-terminal pro-brain-type natriuretic peptide level was 1,583 pg/mL (normal, <100 pg/mL), and his cardiac troponin I level was 39 ng/mL (normal, ≤0.04 ng/mL). He became hypoxic and needed intubation. He was started on intravenous vasopressors and was taken to the cardiac catheterization laboratory, where an intra-aortic balloon pump was placed. His presenting electrocardiogram (ECG) is shown below (Fig. 1).

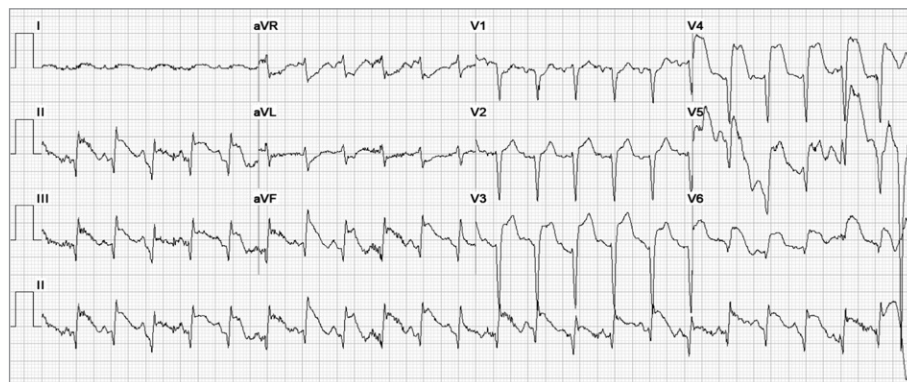


Fig. 1

In addition to ST-segment-elevation myocardial infarction (STEMI), what diagnosis should be suspected from this ECG?

- A) Papillary muscle rupture
- B) Ventricular septal rupture
- C) Free wall rupture
- D) Aortic dissection

See next page for the answer.

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Corresponding author:

Daniel W. Nelson, MD, Tulane University School of Medicine, 5977 General Haig St., New Orleans, LA 70124

E-mail:

dnelson8@tulane.edu

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FOCUS on ECGs: Answer #29

B) Ventricular septal rupture

In cases of delayed STEMI, mechanical complications can occur. The differential diagnosis in acute myocardial infarction (AMI) complicated by a holosystolic murmur includes papillary muscle rupture (PMR) and ventricular septal rupture (VSR). Free wall rupture would not present with a holosystolic murmur, and aortic dissection resulting in aortic regurgitation would cause a diastolic murmur. Patients with acute PMR typically present with shock and acute pulmonary edema. In acute PMR, the systolic murmur may be soft or absent, given pressure equalization between the left ventricle and left atrium; conversely, in VSR, there is hemodynamic compromise and a harsh systolic precordial murmur.

Inferior and anterior STE with inferior Q waves, such as in our patient's ECG, should raise suspicion for VSR.¹ Hayashi and colleagues² observed STE in the inferior and anterior leads in 42.9% of patients who had VSR, but in only 3.6% of patients without VSR. Moreover, abnormal Q waves appeared in all inferior leads (II, III, and aVF) in 44.4% of patients with VSR but in only 4% of those without.² Whereas the ventricular septum receives a dual blood supply from the left anterior descending coronary artery (LAD) and right coronary artery, in a dominant "wrap-around" LAD, the ventricular apex is supplied only by the LAD. Occlusion of the LAD (Fig. 2, arrow) results in necrosis of the anterior and inferior walls, leading to STE in the anterior and inferior leads, and a risk of VSR (Fig. 3, arrow) if reperfusion is delayed.³ This case highlights that, in AMI, an ECG showing STE in the anterior and inferior leads with inferior Q waves should raise suspicion for VSR.



Fig. 2

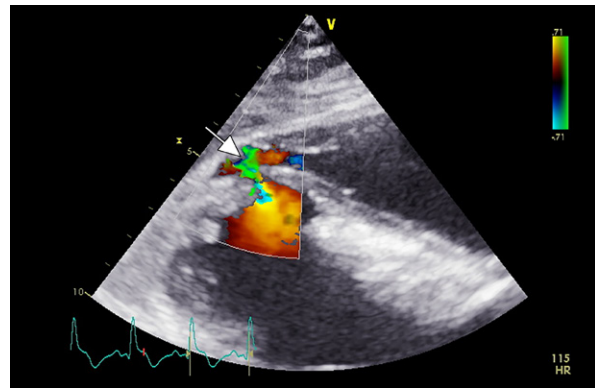


Fig. 3

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