Focus on ECGs: Case #20

Tall R Waves in Precordial Electrocardiogram Leads

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A 55-year-old man with a history of coronary artery bypass grafting and severe biventricular failure presented with several weeks of shortness of breath and associated chest pain. He needed an Impella® heart pump (ABIOMED, Inc.) as mechanical circulatory support for volume overload and cardiogenic shock. His mildly elevated cardiac troponin I level (1.35 ng/mL on admission) was attributed to demand ischemia from decompensated heart failure; the level decreased after diuresis. He was referred for advanced heart failure evaluation. Figure 1 shows his electrocardiogram (ECG) on presentation.

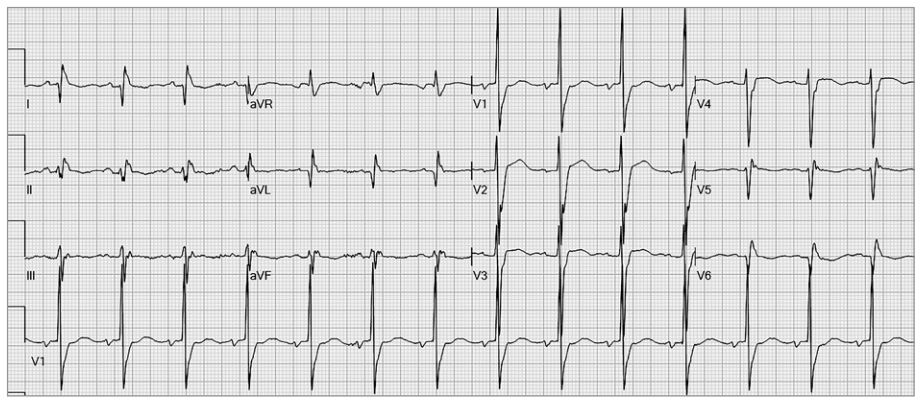

Fig. 1

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The ECG shows sinus rhythm with a premature atrial contraction, a premature ventricular contraction, fragmented QRS complexes in all leads, prominent R waves in the right precordial leads (V_1 through V_2), and Q waves in the lateral leads (I, aVL, and V_6).

What is the associated diagnosis?

A) Left septal fascicular block
B) Inferolateral infarction
C) Right ventricular hypertrophy
D) Duchenne muscular dystrophy
E) Hypertrophic cardiomyopathy

See next page for the answer, as well as a link to the Focus on ECGs *blog, where you can participate in a moderated discussion.*

Focus on ECGs: Answer #20

Answer

B) Inferolateral infarction

The broad differential diagnosis for tall R waves in the right precordial leads includes right ventricular (RV) hypertrophy, right bundle branch block, inferolateral wall infarction, hypertrophic cardiomyopathy, Duchenne muscular dystrophy, Wolff-Parkinson-White (WPW) syndrome, dextrocardia, left septal fascicular block (LSFB), rightward displacement of the heart, misplaced precordial leads, technical errors (such as inadequate high-pass and low-pass filters), and normal variant.[1-4]

The ECG shows no evidence of right-axis deviation, right atrial enlargement, or RV strain pattern (that is, ST-segment depression or T-wave inversion in the right precordial or inferior leads) to support a diagnosis of RV hypertrophy.[1] The absence of a short PR interval and delta waves makes WPW syndrome unlikely. The diagnosis is not LSFB, because the pertinent ECG criteria include the loss of Q waves with an R pattern in the left-sided leads (I, aVL, V_5, and V_6), minimal QRS prolongation (<120 ms), and a prominent R pattern in V_1 through V_2 after other causes of prominent anterior forces had been excluded.[2] Of importance, the diffuse fragmented QRS complexes, defined as the presence of an R' wave or notching of the R or S wave in a narrow QRS, suggest heterogeneous depolarization of the ventricular myocardium, indicating ischemia or a scar.

Clinically, the patient exhibited no features of muscular dystrophy on examination. His chest radiograph showed no dextrocardia or displacement of the heart. Of note, an echocardiogram showed a severely reduced left ventricular ejection fraction of 0.25 to 0.29, akinesis and thinning of the inferolateral wall, and hypokinesis and normal thickness of the other walls. The RV was mildly enlarged and hypokinetic, although without hypertrophy.

The combination of Q waves in the lateral leads, diffuse fragmented QRS complexes, remarkably tall R waves in leads V_1 through V_2, and the patient's history of coronary artery disease is most consistent with a chronic inferolateral infarction. However, in the absence of chronic ischemic heart disease, the diagnosis of hypertrophic cardiomyopathy should be considered, because a tall R pattern in V_1 through V_2 can also result from septal depolarization related to prominent asymmetric septal hypertrophy.

References

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