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This EKG is Concerning for Ischemia!

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CASE PRESENTATION

An asymptomatic 45-year-old gentleman with no significant past medical history presented to a primary care physician's (PCP) office for a pre-employment exam. He was noted to have a blood pressure of 127/75 mm Hg and the rest of the vitals were unremarkable as well. An EKG was obtained with findings identical to those in Figure 1.

Questions:

1. Describe the EKG?
2. What is the best next step?

EKG INTERPRETATION

The EKG shows an R wave in aVL (black oval) greater than 1.1 mV in amplitude, consistent with the Sokolow-Lyon criteria for left ventricular hypertrophy (LVH). The sum of the amplitudes of the R wave in aVL (black oval) and the S wave in V3 (blue oval) is greater than 2.8 mV, consistent with the Cornell criteria. Additionally, the EKG shows depressed ST segments followed by asymmetric inverted T waves in leads I, aVL, and V5 - V6 (red ovals), that are consistent with repolarization changes, the so-called strain pattern.

MECHANISM FOR EKG ABNORMALITIES

EKG changes in LVH arise from structural and electrical alterations in the heart, including increased muscle mass and disrupted conduction due to fibrosis. Biochemical changes also affect electrical signaling, leading to characteristic EKG abnormalities like prolonged QRS complexes. Moreover, ST-T changes reflect repolarization issues, the mechanical impact of hypertrophy, and ischemia from higher oxygen demands in the enlarged heart.

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DIAGNOSTIC CRITERIA

Several sets of criteria have been developed to diagnose anatomic LVH based on EKG abnormalities. The Sokolow-Lyon and Cornell voltage criteria are the most widely used criteria. These and other major criteria are summarized in Table 1.

DISCUSSION

1% to 5% of the general population have findings consistent with LVH on EKG. Diagnostic accuracy of EKG criteria to detect structural LVH are highly variable and differ based upon the specific criteria tested, the population studied, and the imaging modality used to determine true LVH (e.g., echocardiography or Cardiac MRI [CMR]). Low sensitivities and high specificities have been reported in most studies². The MESA study demonstrated a sensitivity and specificity of 22.4% and 95.1% respectively for detecting CMR-determined LVH based upon having a positive Sokolow-Lyon or Cornell voltage criterion³. Conditions that increase the amount of body tissue (obesity), air (COPD, pneumothorax), fluid (pericardial or pleural effusion), or fibrous tissue (coronary artery disease, sarcoid or amyloid) between the myocardium and the EKG electrodes decrease the amplitude of the QRS complex causing a decrease in the sensitivity of the voltage criteria for diagnosis of LVH. In contrast, thin body habitus and left mastectomy may increase QRS amplitude, decreasing the specificity of the voltage criteria⁴.

Hypertension is the most common cause of LVH. As many as one-third of patients with hypertension have an EKG consistent with LVH². Hypertension as a side effect of various medications, including commonly used drugs such as steroids (e.g., prednisone) and NSAIDs (e.g., ibuprofen), as well as less common

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FIGURE 1. 12-lead electrocardiogram

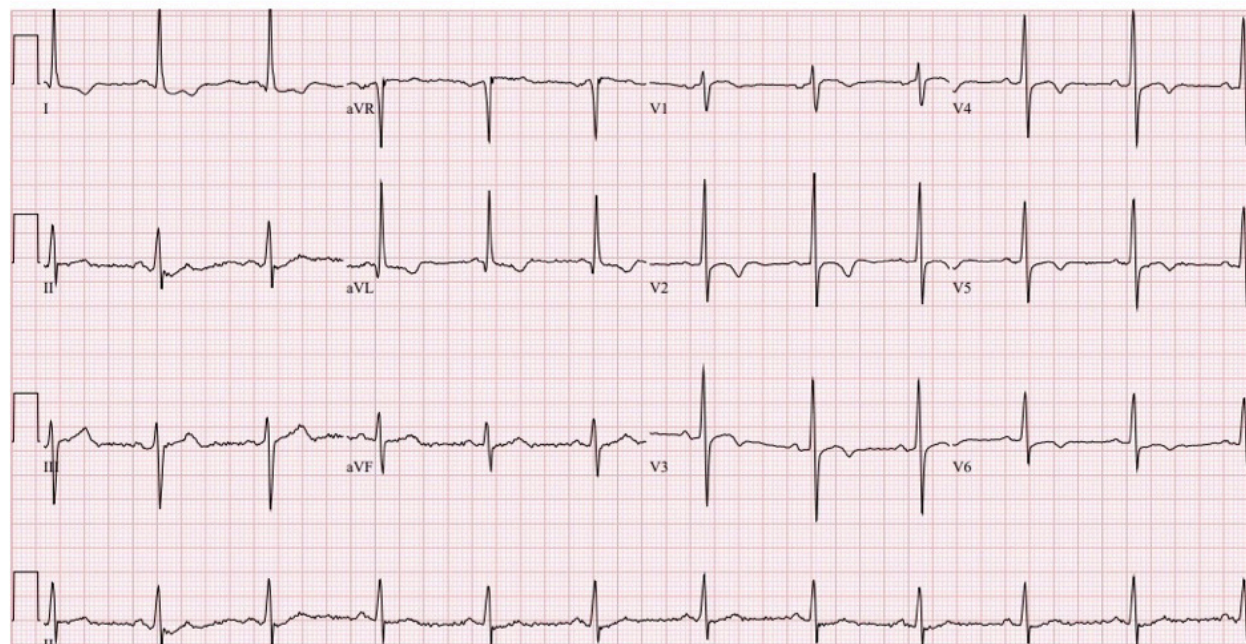


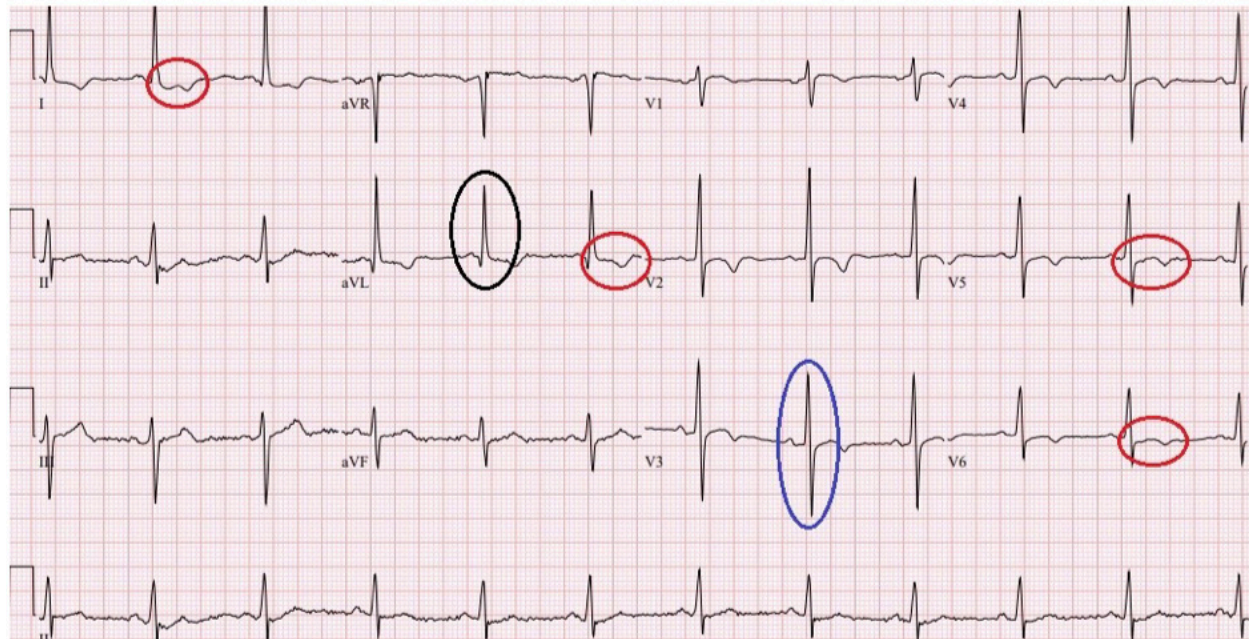
TABLE 1. Common Diagnostic Criteria for Left Ventricular Hypertrophy¹

Measurement	Criteria
Sokolow-Lyon voltages	SV1 + RV5 >3.5 mV RaVL >1.1 mV
Romhilt-Estes point score system*	Any limb lead R wave or S wave >2.0 mV (3 points) Or SV1 or SV2 ≥3.0 mV (3 points) Or RV5 to RV6 ≥3.0 mV (3 points) ST-T wave abnormality, no digitalis therapy (3 point) ST-T wave abnormality, digitalis therapy (1 point) Left atrial abnormality (3 points) Left axis deviation ≥-30 degrees (2 points) QRS duration ≥90 msec (1 point) Intrinsicoid deflection in V5 or V6 ≥50 msec (1 point)
Cornell voltage criteria	SV3 + RaVL >2.8 mV (for men) SV3 + RaVL >2.0 mV (for women)
Cornell voltage duration measurement	QRS duration x Cornell voltage > 2436 mm.ms+

*Probable LVH is diagnosed with totals of 4 points, and definite LVH is diagnosed with totals of 5 or more points
+ For women, add 8 mm

ones like immune suppressants (e.g., calcineurin inhibitors) and targeted therapies (e.g., RET inhibitors), should also be considered⁵⁻⁷. Other causes of LVH include aortic stenosis, aortic regurgitation, mitral regurgitation, and hypertrophic cardiomyopathy (HCM). A focused history and physical exam should be obtained to evaluate for these etiologies, the findings of which will help guide further workup. The repolarization changes secondary to LVH that are detailed in the

“EKG Interpretation” section can mimic ischemia or infarction and are important to recognize to avoid unnecessary testing. It is crucial to exclude hypertension as the etiology with ambulatory blood pressure monitoring as patients with ‘masked hypertension’ will have normal blood pressure readings at office visits. A transthoracic echocardiogram should be obtained to confirm LVH and to rule out the various valvular abnormalities and HCM as possible etiologies.

FIGURE 2. Annotated 12-lead electrocardiogram

CLINICAL COURSE

The patient was referred to cardiology for “concern for ischemia on EKG”. As the patient was asymptomatic and the EKG changes were repolarization changes secondary to LVH, no ischemic workup was obtained. Instead, an echocardiogram was obtained that confirmed moderate concentric LVH and ambulatory blood pressure monitoring revealed readings in the range of 130s-140s/90s mmHg confirming masked hypertension. Patient was initiated on appropriate antihypertensive medications and recommended routine follow-up with PCP for continued management of hypertension.

TAKE-HOME POINTS

- Patients without a diagnosis of hypertension who have normal office blood pressure readings and LVH on EKG should undergo ambulatory blood pressure monitoring to screen for masked hypertension.
- Transthoracic echocardiogram should be obtained in patients with LVH on EKG to confirm the diagnosis and rule out potential etiologies.
- Repolarization changes secondary to LVH can mimic ischemia or infarction.

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