A 48-year-old man with hypertension was being evaluated for a noncardiac issue (progressive multifocal leukoencephalopathy). He had been an active runner and did not have any cardiovascular symptoms at the time. The electrocardiogram (ECG) shown in Figure 1 was a routine study done as a part of that evaluation. His cardiovascular examination was unremarkable, without murmur, S3, or S4. His pulse was regular at 72 beats per minute, and his blood pressure was 112/76 mm Hg.

Q: Which of the following electrocardiographic findings suggest left ventricular hypertrophy?

- Sum of the S wave in V1 and the R wave in V6 ≥ 35 mm
- Sum of the S wave in V3 and the R wave in aVL > 28 mm (men)
- Sum of the S wave in V3 and the R wave in aVL > 20 mm (women)
- All of the above

A: The correct answer is all of the above.1,2

Our patient’s ECG shows sinus bradycardia and left ventricular hypertrophy, suggested by LVH with giant T waves of this quality should raise the suspicion of hypertrophic cardiomyopathy with apical hypertrophy.
prominent voltage (sum of S in V1 and R in V6 ≥ 35 mm) and supported by ST-segment and T-wave changes in the lateral and midprecordial leads. Classic changes of left ventricular hypertrophy often include increased voltage and downsloping ST-segment depression with negative T waves in V5 and V6 (secondary repolarization changes or “strain” pattern).

Notable on this tracing are the large, asymmetric negative T waves in leads V3 through V6. Giant T waves are defined as negative T waves with voltage greater than 10 mm.3 Although there is no specific pattern of ventricular hypertrophy on an ECG that establishes the diagnosis of hypertrophic cardiomyopathy, left ventricular hypertrophy with T waves of this quality suggest the possibility of hypertrophic cardiomyopathy with apical hypertrophy.

**Q:** What are the other causes of giant negative T waves?
- Subarachnoid hemorrhage
- Complete heart block
- Non-Q-wave myocardial infarction
- All of the above

**A:** The correct answer is all of the above. Additional causes of dramatic T-wave inversion are listed in **TABLE 1**. Clinically, non-Q-wave myocardial infarction with T-wave changes and acute central nervous system injury are probably the most commonly seen.4

Echocardiography in this patient revealed severe apical hypertrophy of the ventricle with distal cavity obliteration. The left ventricular outflow-tract gradient was normal. The mitral valve appeared normal, and there was no resting systolic anterior motion.

Cardiac magnetic resonance imaging showed the apical variant of hypertrophic cardiomyopathy but no evidence of left ventricular noncompaction, which is a differential diagnosis of apical hypertrophic obstructive cardiomyopathy. This disease was first described in Japan by Yamaguchi et al5 and Sakamoto et al6 and is regarded as a subgroup of nonobstructive hypertrophic cardiomyopathy. The prognosis of apical hypertrophic cardiomyopathy with regard to sudden cardiac death is believed to be better than that of other forms of hypertrophic cardiomyopathy.3

**REFERENCES**


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**TABLE 1**

**Causes of giant T-wave inversion**

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apical hypertrophic obstructive cardiomyopathy</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
</tr>
<tr>
<td>Cocaine abuse</td>
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<tr>
<td>Non-Q-wave myocardial infarction</td>
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<tr>
<td>Acute abdomen (eg, acute pancreatitis)</td>
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<tr>
<td>Complete heart block</td>
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<tr>
<td>Severe right ventricular hypertrophy</td>
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<tr>
<td>Elevated intracranial pressure</td>
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<tr>
<td>Post-pacemaker syndrome</td>
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<tr>
<td>Wolff-Parkinson-White syndrome</td>
</tr>
</tbody>
</table>

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Echo revealed severe apical hypertrophy of the ventricle with distal cavity obliteration.