CASSA

CARDIAC ARRHYTHMIA SOCIETY OF SOUTHERN AFRICA
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Answer to ECG Quiz 9

Observations

There are 2 striking observations on examining this ECG: the QRS voltages and the widespread deeply inverted T waves.

Systematic analysis of the ECG reveals a sinus rhythm at 66 bpm and no obvious or significant atrial abnormality except for a slight notch of the P waves in Lead I. The PR interval is normal (160ms). The QRS complexes have enormous amplitudes both in the limb leads (with R wave in Lead I of 23mm or 2.3mV) and in the chest leads (R wave in V4 of 37mm or 3.7mV). The QRS axis is 15 degrees. The QRS duration is almost exactly 2 small squares, i.e. 80ms. The T waves are most abnormal: in the chest leads, the T wave is biphasic in V2 and deeply inverted all the way from V3 to V6, with deepest T in V4 and shallower but still inverted T waves in Lead I and aVL. With such abnormal T waves it is not surprising to find ST segments dragged into the T waves, upsloping or downsloping in the direction of the T wave, and also the secondary lengthening of the QT interval, which is 11.5 small blocks wide or 460ms.

Diagnosis and discussion

Are these ECG findings consistent with left ventricular hypertrophy (LVH) found in longstanding hypertension or aortic stenosis? The voltages certainly meet the various criteria used for diagnosis of LVH. There are even supporting criteria for LVH: ST segment depression and T wave inversion in the left chest leads.

However, some of the observations in this patient's ECG do not fit with the common type of reactive LVH as seen in hypertension or aortic stenosis:

- Additional evidence for this type of LVH is an increase in the width of the QRS. In fact, the QRS duration is an important component of some of the most sensitive diagnostic criteria, e.g. Cornell (product of QRS duration and voltage). Often this may be subtle and may be limited to the beginning of the QRS complex: from the start of the QRS to the peak of the R wave, which is called the intrinsicoid deflection or ventricular activation time. In LVH, intrinsicoid deflection in V5 or V6 is typically > 50ms (1¼ small blocks). In this ECG, there is no such widening and the whole QRS is exceedingly narrow and the activation time is < 30ms.

- The most unusual finding in this patient's ECG that cannot be attributed to standard LVH is the widespread T wave abnormality. i) In the usual type of LVH the T waves may be inverted, but they are relatively shallow and less sharp. Yet, the T waves on this ECG are spectacularly deep: up to 16mm in V4. ii) In standard LVH, T wave inversion is seen in the lateral chest leads, whereas in this ECG, T wave inversion is seen not only in V5 and V6 but as early as V3. iii) The usual progression of the depth of T wave inversion in reactive LVH is a progressive deepening the further left one goes. However, in this ECG, the T wave abnormalities, which start much too soon, as seen in V2 overlying the septum, the worst profile is in V4 with a subsequent reduction in V5 and V6.

- A left axis of the QRS is a common finding of standard LVH. Whilst this is not a prerequisite for LVH, and it is a poor sign of LVH when used alone, it is supportive. It is not present in this ECG.

So, the conclusion is that, although superficially the ECG may look like that of common LVH, closer inspection reveals a collection of signs that suggest an alternative diagnosis. Thus, this is not the ECG picture that one would expect to find with LVH related to aortic stenosis (answer a) or hypertension (answer b). Well established LVH due to longstanding uncontrolled systemic hypertension with echocardiographic evidence of additional LV dilatation would surely also show some widening of these 'skinny' QRS complexes and 'delay' in the intrinsicoid deflection.

Mitral regurgitation (answer e) is also incorrect. Firstly, there is little evidence of left atrial enlargement. More importantly, the ECG in isolated mitral regurgitation, even if severe, normally does not show evidence of any degree of LVH and especially this pattern of exaggerated morphology. In fact, ECG evidence of LVH in a patient with mitral regurgitation is an excellent clue that there is some additional other cardiac involvement.

This ECG with marked LV voltages and giant T wave negativity (defined as > 10mm, or > 1mV) is evidence of myocardial or muscle disease. Such large LV amplitudes are seen in Hypertrophic Cardiomyopathy (HCMO), myocyte storage diseases, such as Pompe's Disease and LV Non-Compaction.

In these myocardial diseases, if the heart is not enlarged and intraventricular conduction is preserved, and the abnormality is limited to LV wall thickening, the QRS duration remains entirely normal as it is in this ECG. The P waves often show left atrial enlargement but not always. The majority of these patients have a normal QRS axis.

The deeply inverted T waves as seen in this ECG are very suggestive of HCMO. The giant negativity together with the pattern or progression of the T wave inversion in the chest leads, with the deepest T waves in the mid-chest leads and not in V6 or laterally, are characteristic of a particular type of HCMO: not the commoner asymmetrical septal HCMO associated with LV outflow obstruction, but the non-obstructive concentric apical hypertrophic cardiomyopathy. This apical type of HCMO, first described in Japan, has a more benign course than some obstructive HCMO, from which it must be differentiated as it is not amenable to alcohol septal ablation. The correct answer is d.

Conclusion

The combination of marked LV voltage, together with deep or giant T wave inversion is indicative of a cardiac muscle disorder, most commonly hypertrophic cardiomyopathy.

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