

### (1) OVERVIEW OF THE ECG

This is a very unusual and bizarre-looking ECG, especially in a 20-year-old (no history given). It is always unwise to jump to conclusions or make snap decisions when analysing an ECG, and this is particularly so in this case. Before coming to any conclusion as to the mechanism of the abnormalities or the likely underlying pathology, it is worth describing the tracing systematically.

#### More detailed analysis of the ECG

The rate is 60bpm. The rhythm is irregular, in that the P-P intervals gradually increase, probably due to sinus arrhythmia, with the exception of the 7th QRS complex, which is premature. The QRS complexes are wide, about 150ms, and each is preceded by a P wave. The premature complex also seems to have a premature P wave at its onset, but with an extremely short PR interval.

The P wave axis is normal, about +50°, but the Ps are broad (130ms) and bifid in I and II, with a slurred terminal negative component in VI, suggesting left atrial enlargement. The PR interval is normal (180ms).

The wide QRS does not fit the pattern of either right or left bundle branch block. The QRS axis is in the north-west quadrant at +210°. Broad (70ms) Q waves are present in I, II, III, aVF and V5-6. There is a tiny initial r in V4, followed by a predominantly negative complex.

There is no tall R in V5 or V6, and so Sokolow-Lyon criteria for LVH are not met. However, the S in V3 + R in aVL = 31mm, thus meeting Cornell's criteria, particularly if the QRS width is taken into account.<sup>(1)</sup> The R wave in VI is abnormally tall.

The ST segments are elevated 2mm in V2-3 in the presence of deep S waves and a wide QRS.

The T waves are inverted in the inferior leads and are biphasic in V6.

The QTc interval is about 450ms, but the QRS is wide.

The premature complex (no. 7) appears to closely follow a premature P wave, but this may be part of the QRS.

#### In summary:

- Normal axis P waves, consistent with sinus rhythm, but left atrial enlargement.
- Wide QRS - non-specific intraventricular conduction delay.
- LVH on Cornell criteria.
- Deep, broad Q waves in the inferior and lateral leads, not readily explicable on the basis only of the intraventricular conduction delay.
- Prominent R in VI, followed by an upright T wave.
- Premature complex, probably ventricular (the PR is too short for a conducted premature atrial complex). The "P" wave may be part of the QRS.

Two of the ECG diagnoses can be discarded immediately. Lead misplacement should affect the P wave axis as much as the QRS, which is not the case here. One would also have to postulate simultaneous misplacement of the chest leads. Similarly, mirror-image dextrocardia affects the P wave axis as well as the QRS. The QRS pattern is not that of RBBB.

**The most complete description is therefore (c): Sinus rhythm, PVC, pathological Q waves, North-West axis, LVH.**

### (2) LIKELY PATHOLOGICAL DIAGNOSIS

The steps to diagnose the possible underlying pathology should follow analysis of the abnormalities and decisions as to their likely mechanisms. Many ECG features which have similar mechanisms, such as scar, may be due to a variety of different aetiologies. While Q waves commonly are due to myocardial infarction, any pathology which causes extensive fibrous replacement of myocardium may result in pathological Q waves<sup>(2,3)</sup> (Table I).

#### Dilated cardiomyopathy with non-specific intraventricular conduction abnormality

This is the least likely diagnosis. Severe dilated cardiomyopathy can cause bundle branch block or non-specific intraventricular delay (IVCD). The QRS pattern in this case would be difficult

**TABLE I: Causes of Q waves not due to infarction.**

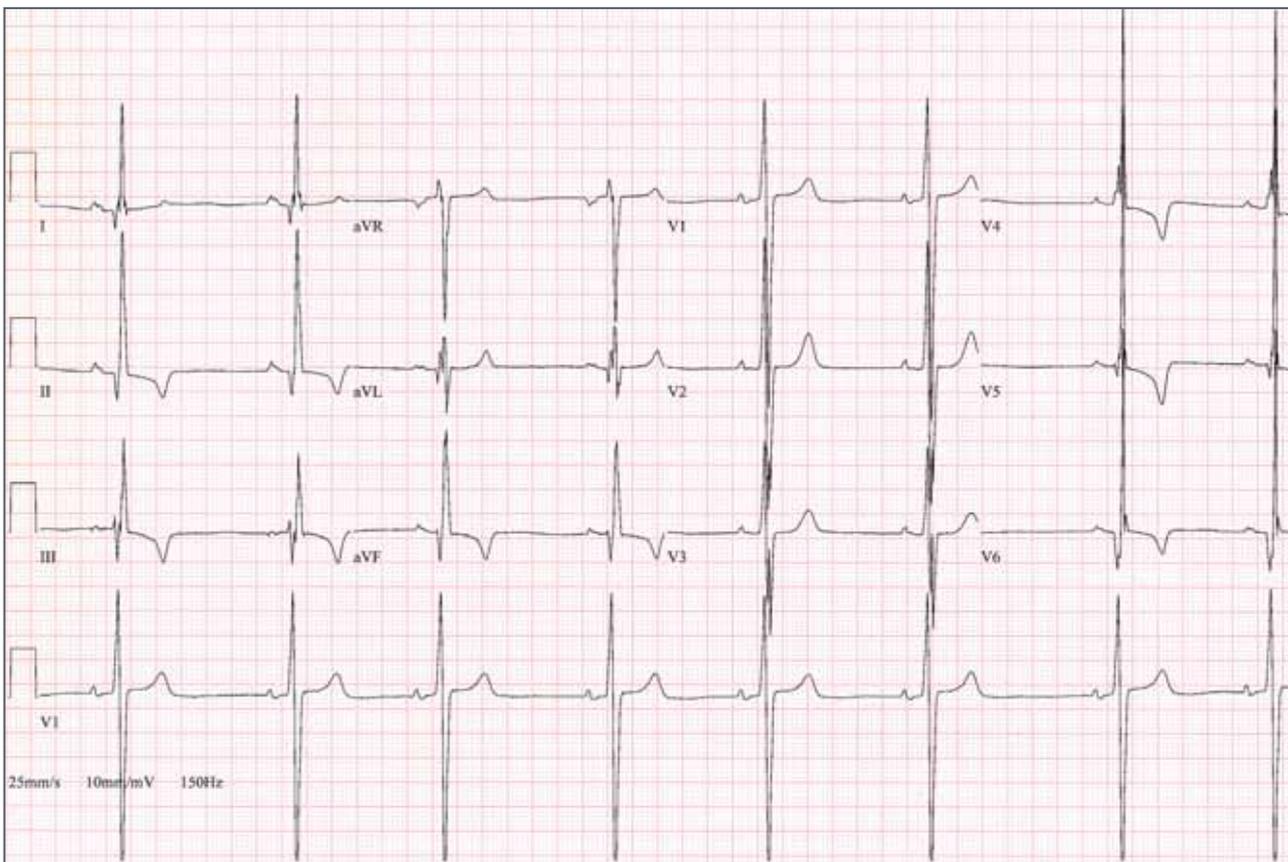
Normal septal Q waves I, aVL, V5-6
Normal variant Q wave VI (may be high lead position)
Normal variant Q waves III and aVF
Left pneumothorax
Myocarditis
Cardiomyopathy <ul style="list-style-type: none"> <li>Hypertrophic</li> <li>Dilated</li> <li>Infiltrative (e.g. amyloid, sarcoid, Chagas disease)</li> </ul>
Wolff-Parkinson-White patterns
Non-specific intraventricular conduction delay
Ventricular rhythms
Hyperkalaemia

to explain on this basis. While IVCD may cause QS complexes in the lateral chest leads, it is unlikely to explain the deep Q waves in the limb leads, and while Q waves may occur in idiopathic dilated cardiomyopathies, they are not a prominent feature.

Anomalous origin of the left coronary artery from the pulmonary artery may present as a dilated cardiomyopathy with Q waves (occasionally in adults), but these do not occur in the inferior leads.<sup>(4)</sup>

**Inferior, posterior and lateral myocardial infarction**

While this could produce this pattern, it would be most unusual, particularly in a 20-year-old.



**FIGURE I: ECG of the same patient taken 5 years before.** It is more typical of HCM, but has changed profoundly over that time. This tracing shows marked left ventricular hypertrophy with repolarisation changes. There is a prominent R wave in V1 and the Q waves in I, II and V6 are smaller, but still pathological (>40ms).

### Duchenne's muscular dystrophy

A 20-year-old male could be in the last stages of Duchenne's cardiomyopathy. The tall R and upright T in V1 occur in this condition because of fibrous replacement of the posterior ventricular wall. Q waves do occur in Duchenne's, but tend to be narrow and not as deep as in this case.<sup>(5)</sup>

### Granulomatous myocardial disease

Sarcoidosis and Wegener's granulomatosis are examples of granulomatous myocardial disease.<sup>(6)</sup> The hallmark of these conditions is conduction disease, including bundle branch block and varying degrees of AV block, as well as ventricular arrhythmias. While there is IVCD, the PR is normal. Extensive Q waves are not usual.

### Hypertrophic cardiomyopathy (HCM)

While atypical in the earlier stages of presentation of hypertrophic cardiomyopathy, this pattern of QRS widening and pathological Q waves may develop later. A clue on this ECG is the presence of LVH, still diagnosable by Cornell criteria, even in the absence of tall R waves in the lateral chest leads. This was the proven diagnosis in this patient. An ECG done 5 years before showed large left ventricular voltages with repolarisation changes and developing Q waves (Figure 1). The mechanism of Q waves in HCM may be due to loss of electrical forces because of myocardial fibrosis. In the absence of significant fibrosis, it appears to be due to alteration of the initial QRS vector from the increased electrical forces of disproportionate hypertrophy of the basal septal and/or ventricular free wall, unopposed by apical forces.<sup>(7)</sup>

**The answer to (2) is Hypertrophic cardiomyopathy.**

### LESSONS AND CONCLUSIONS

- Approach a difficult ECG systematically, not by snap recognition of a pattern.
- Assess the mechanisms of the observed abnormalities.
- Only then consider the most likely underlying pathologies.
- While the most common, healed myocardial infarction is not the only cause of pathological Q waves.
- The ECG in HCM may change significantly over time.

**Conflict of interest: none declared.**

### REFERENCES

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