ECG Quiz 34

CASSA
CARDIAC ARRHYTHMIA SOCIETY OF SOUTHERN AFRICA
A SPECIAL INTEREST GROUP OF SA HEART ASSOCIATION

ANSWER

(1) OVERVIEW OF THE ECG

The ventricular rate is slow, around 46/minute. The QRS complexes are wide, with a pattern resembling left bundle branch block, but atypical in respect of the deep S-waves in V6. Each QRS is preceded by a P-wave.

MORE DETAILED ANALYSIS OF THE ECG

The P-waves are probably of sinus origin, being positive in II and negative in aVR. They are slightly more than 120ms wide and have a prominent negative component in V1, suggesting left atrial enlargement. The PR interval is 160ms.

Small notches are present on the T-waves (arrows, Figure1) which are P-waves which fail to conduct to the ventricles. They are premature, occurring 440ms from the preceding sinus P.

The QRS complexes are very wide (160ms). The pattern in V1 to V3 suggests LBBB, but there is no initial R-wave. The R-wave only appears in V4, where it is sharp and narrow, but the amplitude does not progress. The S-waves from V2 to V5 are very deep. There is marked left axis deviation of -80°, and there are Q-waves in the inferior leads.

The ST segments and T-waves are consistent with the abnormal QRS complexes.

INTERPRETATION

Rhythm

The apparent sinus bradycardia is due to non-conducted premature atrial complexes (PACs) which either reset the sinus node, delaying the onset of the next P-wave, or block the exit of the next impulse from the sinus node. While it is possible that there is underlying sinus bradycardia which is uninfluenced by the PACs, this is less likely. An ECG done the following day showed a sinus rate of 85/minute.

Insofar as only every second P-wave conducts to the ventricles, there is 2:1 AV block. However, the PACs occur only 440ms after the previous conducted sinus P. This is within the normal AV node refractory period and is therefore not pathological.

Early PACs may be difficult to see and give the false impression of sinus arrest or sino-atrial block (arrows, Figure 2 – different patient).

QRS morphology

The QRS complexes are very abnormal. Their width indicates considerable intraventricular conduction delay. The pattern is not typical for LBBB, but the delay is clearly in the left ventricle. The marked left axis deviation together with a left bundle branch block pattern suggests diffuse left ventricular damage, rather than primary conduction disease.

The absence of transition in the chest leads could be due to loss of anterior forces due to anterior infarction, but I think it is more likely that there is clockwise rotation due to right ventricular dilatation.

While the wide QRS cannot be ascribed to left ventricular hypertrophy alone, the very deep S-waves are compatible with LVH. Voltage criteria for LVH may still be applied in the presence of LBBB.[1] An ECG done 8 months before showed LVH with repolarisation changes (Figure 3).

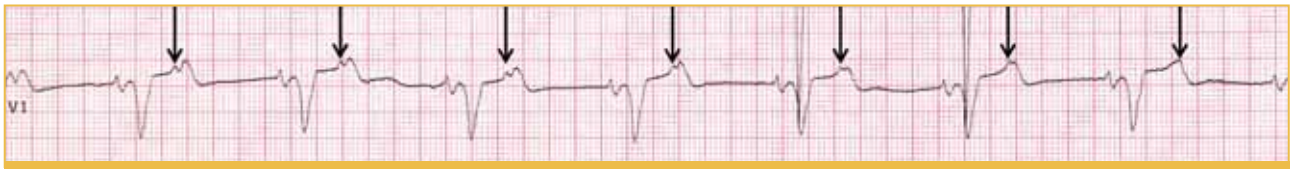

FIGURE 1: The arrows indicate the non-conducted atrial premature complexes.

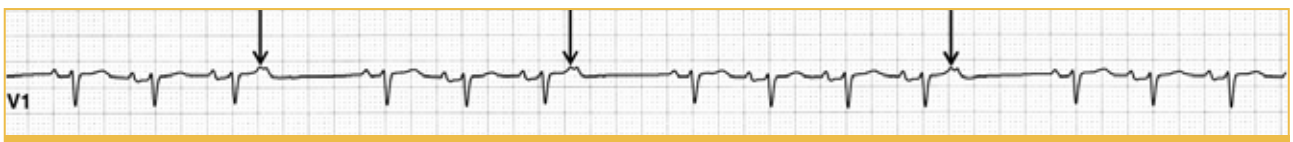

FIGURE 2: The arrows indicate the non-conducted atrial premature complexes.

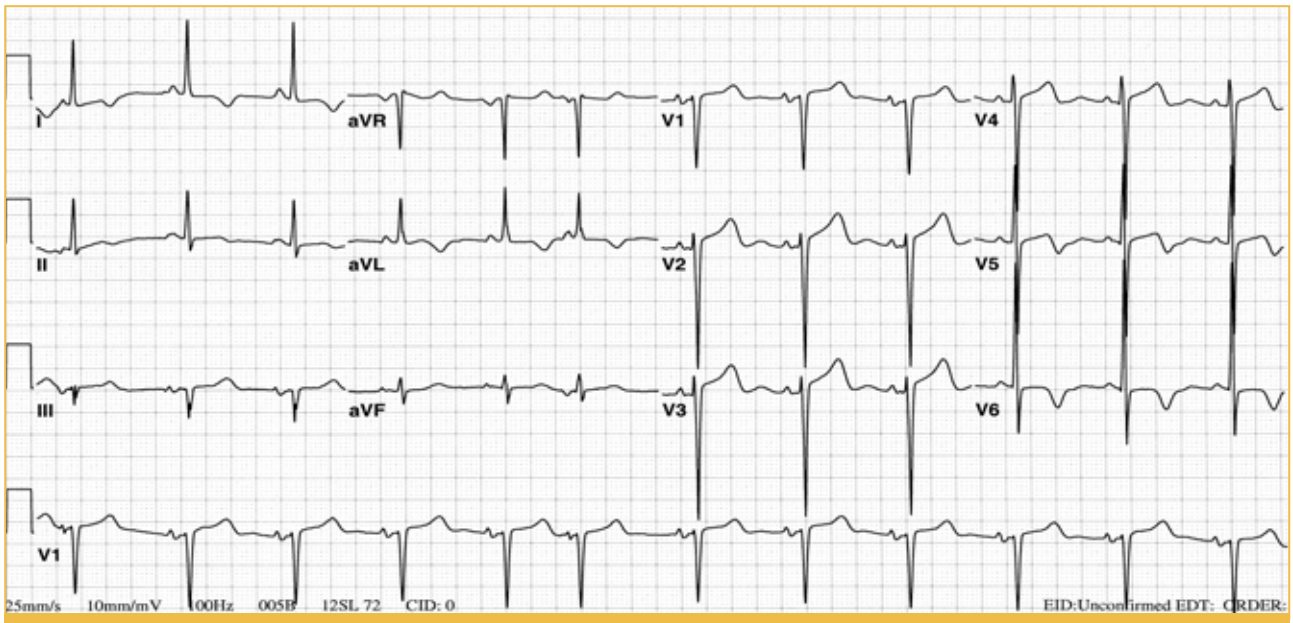

FIGURE 3: An ECG done 8 months before showed LVH with repolarisation changes.

While the inferior Q-waves may well be due to inferior myocardial infarction, this cannot be diagnosed with certainty in the presence of the conduction delay.[2] There are no ST-T changes to support this diagnosis.

The correct answer is therefore (d): Atrial bigeminy with block and atypical LBBB.

(2) MANAGEMENT

As the non-conducted premature atrial complexes most likely represent physiological AV block, there is no compelling reason to implant a pacemaker. As discussed above, atrial depolarisations occurring within the normal AV node refractory period will fail to conduct and do not indicate AV nodal pathology. He does, of course, have evidence of diffuse intraventricular conduction pathology, but this is not sufficient reason to recommend implantation of a pacemaker.

Resynchronisation of the ventricles by means of biventricular pacing may well be justified. He meets the ECG criteria for cardiac resynchronisation therapy (CRT) (the QRS duration is about 150ms, with a LBBB pattern), but his left ventricular function is unknown. Provided his LV ejection fraction is 35% or less and he remains symptomatic on full medical therapy, he would qualify for CRT.[3]

A 24 hour Holter monitor may be useful to see if his symptoms correlate with slow heart rates. It may also reveal evidence of pathological AV block, which would mandate permanent pacing.

LESSONS AND CONCLUSIONS

- What appears to be sinus bradycardia or sinus arrest may be due to non-conducted atrial premature complexes.
- Premature P-waves may block because they fall within the normal AV nodal refractory period.
- Voltage criteria for LVH may still apply in the presence of LBBB.
- This ECG is insufficient, on its own, to recommend a permanent pacemaker.

ACKNOWLEDGEMENT

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REFERENCES

1. Cokkinos DV, et al. Electrocardiographic criteria of left ventricular hypertrophy in left bundle-branch block. British Heart Journal 1978;40:320-324.
2. De Kock J, Schamroth L. Left Bundle-Branch Block Associated with acute inferior wall myocardial infarction. SAfr Med J 1975;49:397-398.
3. 2013 ESC Guidelines on cardiac pacing and cardiac resynchronisation therapy. European Heart Journal 2013;34: 2281-2329.

Conflict of interest: none declared.

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