Country cardiograms case 46: Answer

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Figure 1 (on page 26) shows normal sinus rhythm at a rate of 90 beats/min. PR interval is normal. Compared with the previous tracing, left atrial abnormality is present, and QRS duration has increased to 0.12 seconds. The axis is 0°. ST segment depression is seen in leads V2 through V6, I and aVL. ST segment elevation is present in leads III and aVF.

A QRS duration of 0.12 seconds or more in the presence of a deep, wide S wave or QS complex in lead V1 suggests a left bundle branch block (LBBB). However, caution is appropriate in diagnosing this. Because the duration is "borderline," myocardial infarction may lead to slightly wider QRS complexes, and the QRS morphology in the 2 tracings is reasonably similar (other than in width).

This is important because LBBB that is known to be of new onset can be used as a criterion for thrombolysis if the clinical setting strongly suggests myocardial infarction. The changes of acute ST elevation myocardial infarction (STEMI) can be difficult to identify in the presence of LBBB, and the opportunity for early thrombolysis might thus be missed in such cases. Furthermore, patients with acute myocardial infarction and new LBBB have a higher mortality rate and may derive substantial benefit from thrombolysis. Against this benefit must be weighed the risks of thrombolysis in those who receive it unnecessarily.

Some clinicians may be hesitant to use the "new LBBB" criterion in this case. Can the ST–T changes be used to bolster the case for thrombolysis, even though LBBB may be present?

Indeed they can, and it is a common misconception that LBBB inevitably obscures the changes of an acute STEMI. It is necessary, then, to know the typical ST–T changes associated with LBBB to recognize what is abnormal. Such ST–T changes are directed away from the QRS forces. The tall, wide, often notched positive QRS complexes typically seen in LBBB in leads I, aVL, V5 and V6, for example, are followed by down-sloping, depressed ST segments and inverted T waves. The ST segment elevation of an acute STEMI may be "cancelled out" by such ST segment depression, resulting in an ST segment that is close to the baseline. If ST segment elevation is seen in these leads, that would therefore be a significant abnormality.

By contrast, the typical deep S wave seen with LBBB in V1 and V2 is typically followed by ST elevation and a tall T wave. However, this elevation is "concave up," in other words, not coved or "tombstone" in appearance. A coved, tombstone appearance in these leads would also be abnormal. Alternatively, in these leads if there is MI-induced ST segment elevation, it would be added to the LBBB-induced ST segment elevation. In the right clinical context, extreme ST segment elevation in these leads would therefore also suggest an anteroseptal STEMI pattern. ST segment depression in these leads would definitely be abnormal.

In Figure 1, considerable ST segment depression is present in leads V2 and V3. This clearly is an abnormal finding, which cannot be related to LBBB. It may represent an anterior ischemic pattern or posterior STEMI. In lead V3 the ST segment depression has a horizontal appearance, with an abrupt

transition to the onset of the T wave; this further strengthens suspicion of an ischemic process. By contrast, the ST–T changes seen in leads V5, V6, and leads I and aVL, appear to be consistent with LBBB.

The inferior leads display significant ST segment elevation in III and aVF, with a coved appearance that is more apparent in aVF. The reciprocal changes of ST segment depression seen in leads I and aVL are impressive but, as noted above, they may be secondary changes to LBBB and are therefore not of much use. A 15-lead electrocardiogram would be of further use and should always be obtained when inferior myocardial infarction is considered.

These abnormal ST segment changes in leads III, aVF, V2 and V3 were considered substantial enough, combined with the clinical scenario, to proceed with thrombolysis. Follow-up testing of troponin T showed the level was significantly increased at 0.45 μg/L (> 0.10 μg/L is consistent with myocardial infarction).

For the question, see page 26.

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