What Can You See on This ECG?

Discussion

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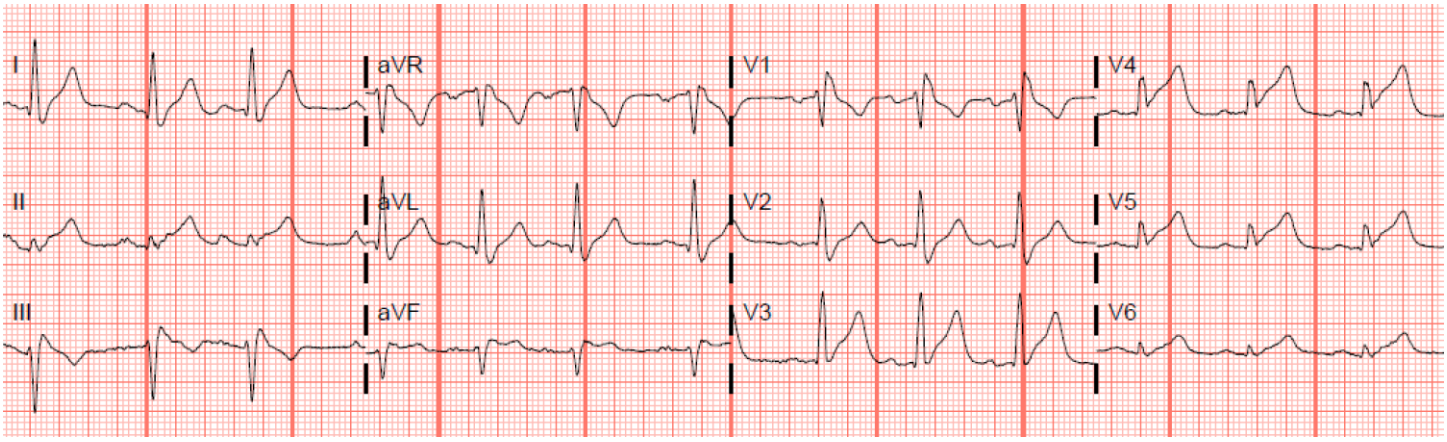

Figure 1

This is the first ER ECG of a male patient – 59 years of age – who has been having central, pressure-like chest pain for about 30 minutes which radiates to his left arm and jaw. He feels clammy.

Sinus rhythm, rate approximately 80 and regular. There are some blocks present and there are also significant ST-T changes on this ECG.

This ECG is filled with hyperacute T waves (Leads I, II, aVL, V3 – V6), so let's discuss them a bit.

First – **HYPERACUTE** refers to that period of time between the *onset of the ischemia* (likely a sudden occlusion of a coronary artery) and the *appearance of ST elevation on the ECG*.

The hyperacute T wave is a ***subendocardial*** phenomenon. Since **ALL ischemia begins in the subendocardium**, the hyperacute T wave reflects the ischemia as it moves toward the subepicardium. Once the ischemia extends from *endocardium to epicardium* it is said to be **transmural** – encompassing the entire thickness of the ventricular wall.

If you could describe these hyperacute T waves using just one word – what would it be?

WIDE – they are ALL very wide and that is the most reliable characteristic of hyperacute T waves. Unfortunately, most people are told they are TALL and large. Leads I, V3 and V4

have the tallest T waves on this tracing, but as hyperacute T waves – they certainly aren't the tallest or the largest I've ever seen! But they are all wide. If you labor under the assumption that they are all TALL – you are not only fooling yourself – you are also missing a lot of hyperacute T waves! That being said – many of them are indeed tall – they just don't *have* to be tall.

There is a **complete right bundle branch block (cRBBB)** present and a borderline **first degree AV block**. I'll have more to say about that in a moment.

Do you have any idea which coronary artery is the culprit? We are going to determine this by using the **Jones Method of Diagnostic Elimination**. We aren't going to try to decide *what this could be* when it is likely much faster to decide *what it cannot be*.

The ischemic changes (the hyperacute T waves) in Leads I and aVL rule out the **right coronary artery (RCA)** as the culprit.

The ischemic change in Lead V3 is a bit too proximal for the **left circumflex artery (LCx)**. Plus, if we consider the cRBBB to be acute, everything points to a very proximal occlusion of the **left anterior descending artery (LAD)** as the culprit since an occlusion of the LCx cannot cause a cRBBB.

Now here is a distinction you should remember: regarding the **RCA** and the **LCx** – whichever is dominant will control the artery to the AV node and may result in ischemia of the node. That will result in various blocks (first degree, Mobitz I second degree or even third degree AV block). However, occlusions of those vessels NEVER cause bundle branch blocks. That is due to occlusions of the LAD only.

On the other hand, the LAD does NOT provide significant circulation to the AV node, so the first degree AV block cannot be due to an occlusion of the LAD…

…or CAN it?

If there is disease of the left bundle branch resulting in slow conduction and now the right bundle branch is blocked – a first degree AV block CAN appear following an occlusion of the LAD – even though there is nothing wrong with the AV node itself. What you must understand here is that the term "AV" (atrioventricular) does not specifically refer to the AV NODE but rather *the entire route from the SA node to the first activated myocyte in the left ventricular septum*.

There are two other interesting discoveries in this tracing that need to be explained:

1. Lead II has a hyperacute T wave even though it's an inferior lead.

2. Lead III has an RBBB morphology even though it, too, is an inferior lead.

Lead II is not just an *inferior* lead – it's a *left-sided* inferior lead and may reflect perturbations in the left-sided circulation (such as occlusions of the LAD).

Lead III is not just an *inferior* lead – it's a *right-sided* lead and may reflect right-sided conduction disturbances (such as RBBB).

So, remember:

1. Hyperacute T waves occur in the subendocardium – they do NOT represent a transmural ischemia… yet!
2. Sometimes it is more expedient to decide – from the ECG – what could NOT have happened rather than what possibly DID happen (**Jones Method of Diagnostic Elimination**).
3. A cRBBB appearing during an occlusion of the LAD indicates a *very proximal occlusion* with a *very large area at risk*. ***These are very dangerous occlusions***.
4. A first degree AV block may occur below the AV node in a bundle branch. It does NOT have to occur in the AV node proper.