Diagnose and Discuss
The Discussion
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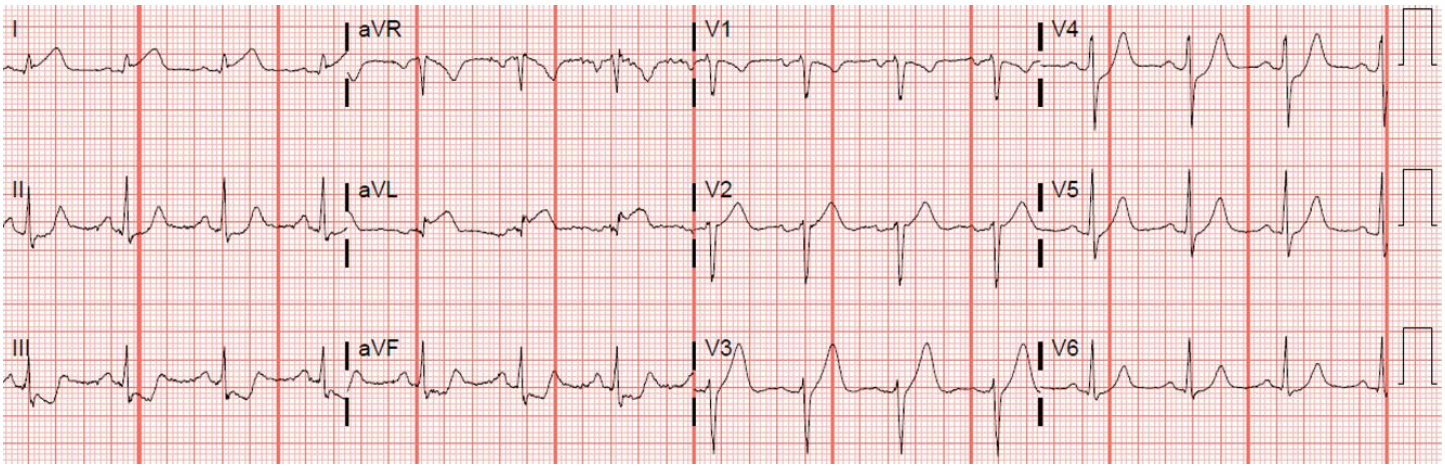

Figure 1

I want to know…

1. Which coronary artery is the culprit?
2. There are two findings on here that quickly rule out two of the coronary arteries as the culprit artery. Which two arteries are ruled out and what was the finding that eliminated each artery?
3. Why is there ST depression in Leads V4 – V6?
4. There are reciprocal changes on this ECG but there is something not quite "right" about them. What is it?

The Discussion

1. The left anterior descending artery (LAD) is the culprit artery. While there is a possibility that this may represent an isolated occlusion of the D1 branch, we just don't have enough conclusive evidence of that; therefore, it is best to call this an LAD occlusion that is *proximal* to the D1 branch. An *occlusion of the LAD* with *ST depression in the inferior leads* places the site of occlusion *proximal to D1*. NOTE: there is no rule that states exactly what a proximal occlusion of an LAD is actually proximal *to*…! Proximal to S1 or proximal to D1? Personally, I always assume that a "proximal LAD occlusion " is proximal to D1. Remember: *sometimes D1 is proximal*

to S1. The loss of blood supply to the D1 branch will cause an acute ischemia to the basolateral (formerly "high lateral") area of the left ventricle which, in turn, will cause ST elevation in Leads I and aVL in the frontal plane. The ST depression in the inferior leads is the reciprocal change to the ST elevation of Leads I and aVL (but mostly Lead aVL).

2. An occlusion of the RCA *never* produces ST elevation in Leads I and aVL, therefore, the RCA is eliminated. The left circumflex artery (LCx) *never* produces ischemic changes in Leads V1 – V3 (the ischemic change here is represented by the hyperacute T waves in Leads V2 and V3); therefore, the LCx is eliminated. Bear in mind that an occlusion of the LCx can definitely produce *ST depression* in Leads V1 – V3, but *those aren't primary ischemic changes* – those are *reciprocal changes* secondary to a primary acute transmural ischemia on the opposite side of the heart!

3. There is an occlusion of the LAD proximal to D1 but there is ST depression in the apicolateral leads (Leads V4 – V6). Why is there ST depression? Why not ST elevation?

   The apicolateral area which is viewed by Leads V4 – V6 is – under normal circumstances – perhaps the best perfused area of the heart. The first and second diagonal branches of the LAD provide circulation to that area as does the posterolateral branch of the RCA and the obtuse marginal and posterolateral branches of the LCx. In about 30% of people, the ramus intermedius also supplies blood. The LAD, however, is typically the largest contributor. The fact that there is a proximal occlusion of the LAD – yet no ST elevation or even hyperacute T waves in the apicolateral area – strongly suggests the presence of other sources of circulation preventing the area from developing a transmural ischemia and infarcting… but obviously not enough to prevent some subendocardial ischemia!

   Could Leads V4 – V6 represent a DeWinter pattern? Possibly. The reason I say *possibly* is because most of the DeWinter patterns I've seen have been characterized by deeper J point depressions and larger T waves. They also represent a proximal occlusion of the LAD and are typically accompanied by ST elevation in Lead aVR, which indicates an occlusion *proximal to S1*. But remember: an occlusion can be *proximal to S1* yet still be *distal to D1*. So, based on the subtle difference in morphology and lack of ST elevation in Lead aVR, I would think it *less* likely that these leads represent a DeWinter pattern – but I cannot say that I am 100% certain.

   Many people think that *any* ST depression followed by a tall T wave represents a

DeWinter pattern – but that's not correct! DeWinter et al. were *very specific* that the ST depression was upsloping from the J point. They did NOT include flat ST depressions or ST depressions that are initially downsloping and then turn upward. The ST depressions in Leads V4 – V6 are all purely upsloping depressions beginning at the J point which *would* comply with the morphological pattern of a DeWinter T wave.

Perhaps this ECG was recorded too early for the repolarization changes in Leads V4 – V6 to have fully developed.

4. The primary ST deviation in the frontal plane leads is the ST elevation in Leads I and aVL. Look closely at the repolarization in those leads. There is ST elevation PLUS an *upright* T wave. This is what one would expect to see DURING the acute ischemic episode. Pay special attention to those T waves: T waves do NOT invert DURING the ischemia. They invert *after* the ischemic episode is over and following resolution of the O_2 supply/demand imbalance.

   Now look at the repolarizations in Leads II, III and aVF that represent the reciprocal changes. In theory, the reciprocal to *ST elevation with an upright T wave* should be represented by *ST depression with an inverted T wave*, which would be the *opposite* of the ischemic changes.

   But that isn't the finding here!

   The reciprocal changes on this ECG are manifested by ST depression with *upright* T waves (T waves are categorized by the polarity of the terminal deflection – a negative/positive T wave would be categorized as an upright T wave). I have not found any articles with a reasonable explanation.

   While classic reciprocal changes include ST depression *with T wave inversion*, it's just not always that textbook. Here are some *possible* reasons why you might still see an upright T wave in leads reciprocal to an acute transmural ischemia:

- **Timing of the Ischemic Event**: Early in an MI, the T wave may not have inverted yet. T wave inversion often follows ST changes as ischemia progresses.

- **Partial Reciprocity**: Reciprocal changes are not always perfect mirror images. The ST segment may reflect the injury, but the T wave can behave differently due to regional differences in repolarization.

- **Lead Orientation and Vector Influence**: The electrical vectors seen by each lead vary. A lead might show ST depression due to reciprocal change but still pick up a positive repolarization vector, resulting in an upright T wave.

- **Concurrent Conditions**: Conditions like left ventricular hypertrophy, electrolyte imbalances, or early repolarization can influence T wave morphology independently of ischemia.

Let me assure you, you *will* see this frequently. It still represents a reciprocal change, but *not* what is often described as the *classic findings of reciprocity*.