Do You See the Acute MI? Which Artery Is Occluded? And Where?

Jerry W. Jones, MD FACEP FAAEM

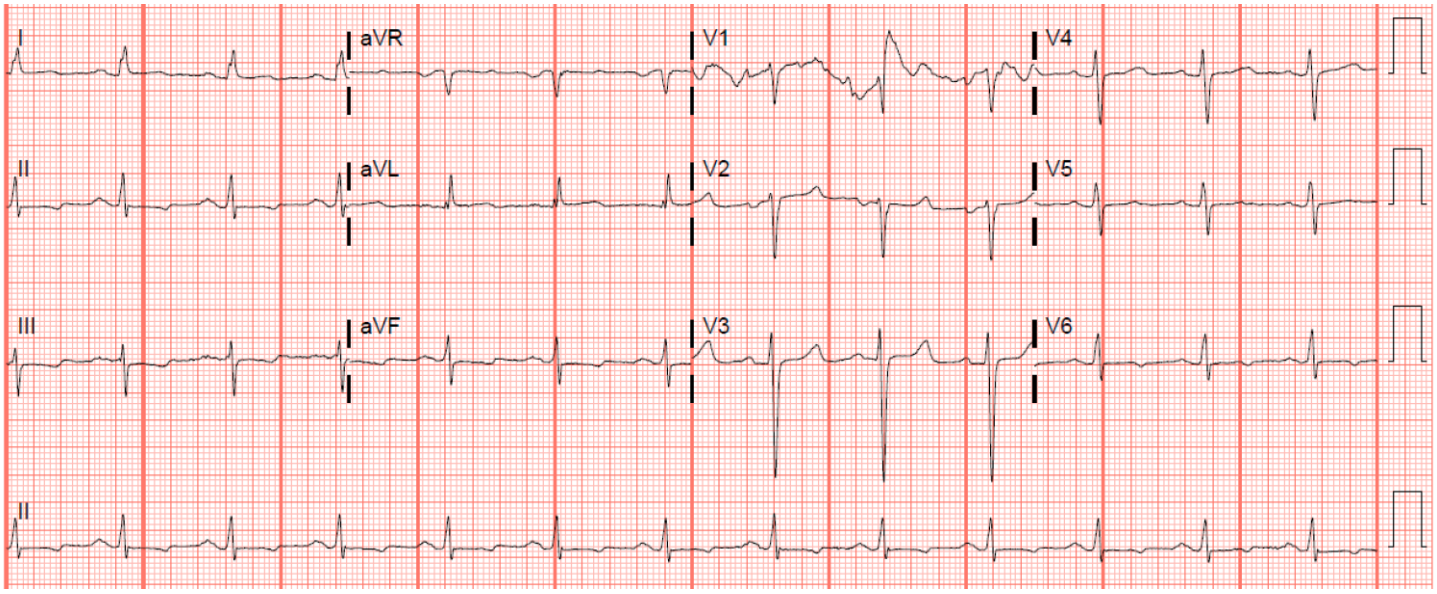

Figure 1

This is the arrival ECG of a 61 year old male, overweight, heavy smoker and Type 2 diabetic. He has been having central, crushing chest pain off and on for an hour but it became continuous about 30 minutes prior to arrival in the ER. It radiates to his right arm and jaw. He is pale, diaphoretic and feels nauseous. He also has a past history of hypertension but is generally non-compliant with his diet and medication.

Occlusion Myocardial Infarction (OMI)

While the STEMI/NSTEMI paradigm was a tremendous improvement over previous methods, it has served its purpose and there is now a "new sheriff in town" – the OMI/NOMI paradigm.

When I was a resident in internal medicine many years ago, we had no way to intervene in an acute transmural ischemia. Our job was to keep the patient alive until the infarction was completed. The initiation of thrombolytic therapies followed by the revelation that PCI was not only life-saving but myocardial-saving as well has brought us to where we are today. None of that changes with the OMI protocol.

We began recognizing a few years ago that using a fast, "cookbook" determination with fixed criteria for deciding whether to intervene in an acute MI or to manage the patient medically had significant flaws. We were missing about 30% of acute coronary occlusions for several reasons – but the two main reasons were that 1) complete 100% occlusions

were being designated “non-STEMIs” and 2) the interpretation skills of many of those reading and interpreting the ECGs needed improvement. Even to this day, most medical schools and residency programs in the US do not provide a formal, structured course in electrocardiography. In my Masterclasses, I find that non-physician medical professionals (PAs in particular) are often much better equipped to understand and benefit from the course than some of the physicians.

So – obviously – we need to apprach the problem from *two different perspectives*: first, we need to change to a diagnostic paradigm that is much more likely to recognize acute coronary vascular occlusions and 2) we need to start educating our medical students and residents with formal classes in electrocardiography. Twelve-lead ECGs are among the most ordered tests in the US and – unlike many of the other tests – they demand *immediate* review and interpretation.

The ECG in Figure 1 does not meet STEMI criteria. In many medical centers this patient may have been relegated to medical management. Now please understand me – in many cases, medical management is indeed the appropriate form of management. The problem is this: too many patients for whom medical management would be very inappropriate are being denied the acute intervention they require.

This is one of those patients. However, medical management in the ER did not alleviate the chest pain and he began to experience more disturbing symptoms. The ER doctor advocated for him to the cardiologist on-call who immediately took the patient to the cath lab and found a complete coronary occlusion which was re-opened and stented with very good results.

For more on OMI, visit “Dr. Smith’s ECG Blog” (https://drsmithsecgblog.com/).

Which vessel was occluded? Did you decide?

One of the main tenets of the STEMI protocol is that there must be a *fixed, absolute amount of ST elevation* (or depression in the case of Leads V1 – V3) before a patient can be considered for the cath lab. Or, as in our patient here, if they fail medical management.

This is where the OMI paradigm steps in. First of all, everything you learned about the STEMI/NSTEMI paradigm still applies to the OMI paradigm. But there’s much, much more.

The main difference is that there is no fixed amount of ST elevation or depression required. The STEMI paradigm is basing the decision to go to the cath lab on the amount of ST elevation. The OMI paradiagm bases that decision on the likelihood that a coronary vascular occlusion has occurred. A;though the ECG in Figure 1 does NOT meet the STEMI

protocol, there IS evidence of ACUTE ischemia occurring. But you need to sharpen your skill at ECG interpretation to see it.

What was found in the cath lab? A complete occlusion of the left circumflex artery – which was successfully stented. Is there anything on this ECG that would point to that? Yes, there is.

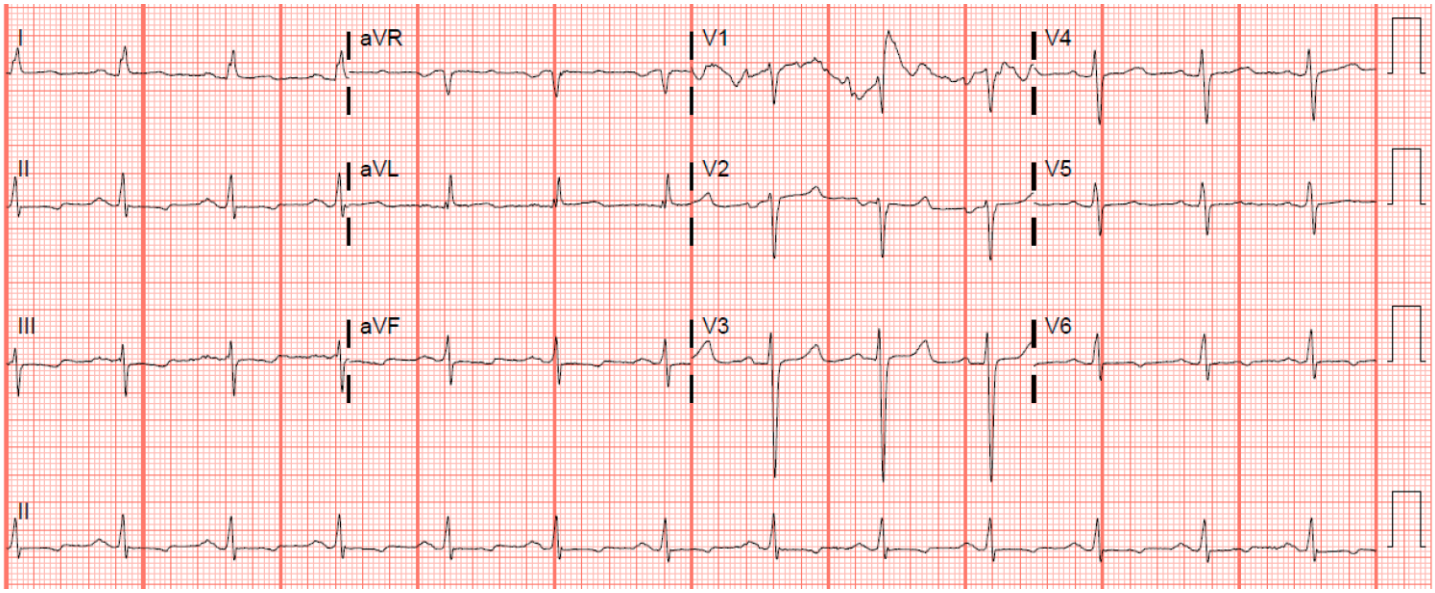

Figure 1 (repeated)

Regarding ST elevation – essentially, there is none. And there is no ST depression in Leads V1 – V3. How can we diagnose a *complete coronary occlusion* without the required amount of ST elevation. Well, using the STEMI protocol – we don't. We put the patient in the hospital and a few days later we discover – to our SURPRISE(!) – that the patient's ECG now shows deep, wide Q waves and the T wave inversions of reperfusion. That wasn't what we expected!

The answer to this quandry lies in **JONES'S RULE**:

> Any ST depression on the ECG of a patient having chest pain compatible with an acute coronary syndrome should be considered a reciprocal change to an acute transmural ischemia occurring in another part of the heart.

OK, where comes the part pertinent to this ECG:

> **COROLLARY #1:** The ST *depression* of the reciprocal change may appear BEFORE the ST *elevation* of the ischemia.

And then there is

> **COROLLARY #2:** Even when BOTH are present, the ST depression of the reciprocal change may remain much more impressive than the primary ST elevation of the ischemia.

Look closely at the inferior leads (II, III and aVF). Now look even closer. There is ST depression in those leads. You're right! It's less than 1 mm. But the old STEMI protocol doesn't apply here. We are using the OMI paradigm. There are no absolute amounts of ST deviation to be adhered to.

This is a *proximal* occlusion of the left circumflex artery (LCx).

"OK," you say, "why isn't there any ST elevation in Leads I and aVL, or ST elevation in Leads II, III and aVF, or ST depression in Leads V1 – V3, or ST elevation in Leads V5 and V6?

Maybe there are.

A proximal occlusion of a *dominant* LCx very frequently presents with little or no ST segment changes – yet it is a very dangerous MI, risking about the same amount of myocardium as a proimal occlusion of the left anterior descending artery (LAD). This could be such an occlusion, though it probably isn't.

More likely, this is an occlusion of a proximal *non-dominant* LCx. What? Did you think that non-dominant LCx occlusions didn't happen… or weren't serious enough to consider? Even though the LCx is non-dominant 80 – 85% of the time – they can still be dangerous! Granted, the area at risk is much less and mortality is (thanfully) lower.

What makes me think this is a ***non-dominant*** LCx occlusion rather than an occlusion of a ***dominant*** LCx?

Join me on June 28, 2026 to find out more about the LCx and its occlusions.

If you are going to miss an acute MI – THIS is where it will happen!

Just don't let it happen to YOU!