Cardiovascular Innovations and Applications

Vol. 4 No. 4 (2020) 287–289
ISSN 2009-8618
DOI 10.15212/CVIA.2019.0019

COMMENTARY

Some Issues Related to STEMI and NSTEMI

C. Richard Conti, MD[1]

[1]*University of Florida Medical School, Gainesville, FL, USA*

Diagnosis of Myocardial Infarction

The diagnosis of myocardial infarction is based on clinical story and troponin leak. Troponin levels can be normal or low due to very early symptoms or high due to a large infarction or due to spontaneous reperfusion with washout of viable ischemic tissue. If there is ST segment elevation, there is a strong suggestion that a coronary artery is occluded or nearly occluded, but that needs confirmation by angiography and ventriculography.

STEMI is not a Diagnosis

The term STEMI is developing into a mildly confusing term for ED and CV physicians. STEMI is not a diagnosis. It is a description of the ECG in patients with acute myocardial infarction and suggests that an epicardial artery is occluded and ischemia is ongoing. Myocardial infarction is the diagnosis and the ECG is a lab test. Currently this abnormal laboratory test initiates the strategy for immediate transfer to the catheterization laboratory for PCI/Stent, if appropriate.

Two Infarctions at the Same Time

Recently I was asked how I would handle the situation, in which two patients with acute myocardial infarction and ST elevation appeared in the ED at the same time. My response is that someone senior in the ED has to make a decision, i.e. triage the patients, since both patients have had a myocardial infarction. The critical issue is related to when the infarction occurred. If I were the person triaging I would urge transfer of the patient with an initial normal or near normal troponin, coupled with a good story and ST elevation to the cath lab. This patient probably has had an infarction in the recent past few hours i.e. less than 4–6 hours and may have more salvageable peri-infarction ischemic myocardium, as reported in animal experiments by Reimer and Jennings [1]. In contrast, the patient with an initial abnormally high troponin leak probably had the infarction more than 4–6 hours prior to arrival in the ED and may have completed the infarction with less salvageable peri-infarction, ischemic tissue. In the first example, a short Door 2 B time is critical, and in the second case the D2B time may not be as critical. It's not a perfect solution, but in my opinion, it's the best we have.

Difference between NSTEMI and STEMI Patients

I think there is a fine line between STEMI and NSTEMi.

ST segment elevation is considered by most as being compatible with an occluded coronary artery and the occlusion of the coronary artery is suspected to be the cause of the infarction in the patient with ST elevation on the ECG. Patients with myocardial infarction who do not show ST segment elevation may still have evidence for myocardial ischemia manifested as ST depression T wave abnormalities

Correspondence: **C. Richard Conti**, MD, University of Florida Medical School, Gainesville, FL, USA,
E-mail: conticr@medicine.ufl.edu

or nonspecific St-t wave abnormalities etc. Despite the absence of ST segment elevation, these patients may have a coronary occlusion but with collateral blood flow to the distal circulation of the infarct related artery.

Why should we assume that the patient without ST segment elevation did not occlude an epicardial artery but developed the myocardial infarction due to marked myocardial oxygen demand? Admittedly this patient could have embolized to the microcirculation from a disrupted plaque or a plaque that had thrombus attached. If either of these latter clinical scenarios are occurring and the epicardial coronary artery is widely patent, then epicardial coronary artery PCI/Stent will not be useful but medical therapy may be. However, that will not be obvious until after coronary angiography is done.

Concerns about the Etiology of Myocardial Infarction

My concern is that the patient that does not demonstrate ST segment elevation, who is now leaking enzymes may be doing so after spontaneous opening of the epicardial artery secondary to their own tissue plasminogen activator. Some of these patients still have myocardial ischemia and will be left with a high-grade stenosis of the infarct related coronary artery. It should be considered a possibility that the epicardial artery in question could have been occluded prior to the patient appearing in the ED or in the emergency vehicle and prior to the first ECG taken. Thus, the patient might have demonstrated ST segment elevation had the patient been evaluated with ECG at an earlier time.

Urgent PCI/Stent vs Medical Therapy for Acute Myocardial Infarction

Some will argue that if the epicardial vessel is now patent, there is no need for PCI. I agree with that point but only if the vessel is widely patent as it would be after PCI/stent (that can only be determined at coronary angiography). I will not accept the argument about patency, if the vessel remains patent but nearly occluded. FFR may solve the problem.

Another Clinical Issue; Myocardial Infarction but no ECG Changes

Another clinical issue that must be considered by first responders, especially ED personnel, relates to an area of the heart that is infarcting but may not demonstrate changes on the first 12 lead ECG (even if vessels are occluded). This occurs in the lateral wall of the left ventricle, which is supplied by the obtuse marginal branches of the circumflex coronary artery. In this instance the initial 12 lead ECG may be normal despite a good story and troponin elevation. ECG leads, V7-9 may be necessary to detect any ECG abnormalies, i.e. ST segment elevation or ST segment depression.

Still Another Clinical Issue (LBBB and Myocardial Infarction)

It is common practice to take patients with recent onset LBBB, especially if a previous ECG was normal, (and the patient had a Troponin leak and good story,) for coronary angiography and possible PCI/Stent. Why not do the same for patients with a good story for a myocardial infarction and troponin leak, who do not have ST elevation on the ECG?

Current Opinion

Currently, I share the opinion that all patients with acute myocardial infarction are at higher risk than those without an acute myocardial infarction. I think all patients with acute myocardial infarction should be considered for left heart catheterization and possible PCI/stent. If the vessel is not widely patent, make it so with PCI/Stent. Some have the impression that NSTEMI is benign compared to STEMI and thus need not be considered for urgent revascularization. I do not agree with that statement as do many others.

The Literature

The message I am getting from the literature is that there is some evidence to accept the use of coronary angiography and subsequent PCI/Stent in the NSTEMI patient [2]. In this cohort from

Duke 2413 patients with STEMI were compared to 1974 NSTEMI patients. STEMI patients had a higher short term mortality than NSTEMI patients but NSTEMI patients had a higher risk of long term mortality. Early revascularization was associated with a similar improvement in long term outcomes for both STEMI and NSTEMI patients. The results of this report suggest that efforts should be made to find out what is going on in the coronary circulation. It may be a surprise to find occlusion when it was not expected.

Conclusion

We need to rethink what should be done in each case and not generalize what should be done for myocardial infarction patients presenting with ST elevation or NST elevation on the ECG.

REFERENCES

1. Reimer KA, Jennings RB. The "wavefront phenomenon" of myocardial ischemic cell death. II. Transmural progression of necrosis within the framework of ischemic bed size (myocardium at risk) and collateral flow. Lab Invest 1979;40(6):633–44.
2. Chan MY, Sun JL, Newby LK, Shaw LK, Lin M, Peterson ED, et al. Long term mortality of patients undergoing cardiac catheterization for ST elevation and non ST elevation myocardial infarction. Circulation 2009;119(24):3110–7.