Myocardial Infarction: What's New?

Kirk Magee, MD, MSc, FRCPC
Presented at Dalhousie University's 33rd Annual February Refresher: Emergency Medicine, February 2007.

The saying that "time is muscle" is the long familiar mantra for physicians treating patients with acute MI (AMI). Most senior medical students will have little difficulty recognizing the diagnosis and quickly realize the necessity for rapid medical intervention. The greater challenge in the face of such time constraints is the ability to recognize those patients that we historically miss or in whom we delay treatment.

Who do we miss? (Table 1)

Patients in their 20s or 30s presenting with chest pain are often quickly dismissed as having costochondritis or chest pain not yet diagnosed. However, the possibility of a cardiac etiology should always be seriously considered in patients who have a strong family history of MIs under the age of 40 years or who use cocaine or amphetamines.

At the other end of the age spectrum, clinicians are often slow to recognize the diagnosis of AMI in the elderly. Remember, the most common presentation of AMI in the elderly is shortness of breath, not chest pain.

Table 1

Who do we miss?

- Young patients
- Elderly patients
- Female patients
- Patients with diabetes
- Ethnic patients

Meet Shawn

Shawn, 47, presents to the ED with a 30 minute history of chest pain. He states that he was working on his new cottage when he developed severe crushing chest pain which radiated to his left shoulder and jaw and was associated with nausea and shortness of breath.

Shawn is a smoker and has a past medical history of hypertension.

Examination

On exam, Shawn is pale and diaphoretic with stable vital signs. His heart sounds are normal and his lung fields are clear. His initial ECG shows normal sinus rhythm with no evidence of ST-segment abnormalities.

Questions

1. How would you treat Shawn?
2. What further investigations are necessary?

Diagnosis

Shawn is put on a cardiac monitor and an IV line is inserted. Oxygen, acetylsalicylic acid, nitroglycerin and morphine are administered. Serial ECGs show ST-segment elevation in leads II, III and AVF with reciprocal depression in leads I and AVL. Shawn is diagnosed with an inferior STEMI.

As the nearest catheter laboratory is > 90 minutes away and Shawn has no contraindications to thrombolysis, he is given a bolus dose of tenecteplase with a complete resolution of his symptoms and the normalization of his ECG.

As with patients who have diabetes mellitus, the elderly may also present atypically—weakness, nausea and vomiting, or an altered level of consciousness are not uncommon presentations.

After the age of menopause, the risk for AMI in women rapidly approaches that for men. Yet surprisingly, studies have consistently shown that women are thrombolyzed later and receive less aggressive treatment than their male counterparts.

Finally, in our increasingly multicultural country, we must be careful to consider our patients in their cultural context. "Crushing retrosternal chest pain" is a Western European description that might not translate exactly. While working in an inner city ED which served a large native population with a high risk of cardiovascular disease (CVD), I was often surprised to hear patients describe their AMI as a "hard" chest pain.

The challenging ECG

The first decision node in the treatment pathway for AMI is centered on the ECG. Is there evidence of ST-segment elevation? Again, few senior medical students would have problems recognizing that our patient, Shawn, is a candidate for urgent reperfusion therapy, but in other situations, interpretation of the ECG is more challenging.

Clinicians should not be falsely reassured by the presence of a normal ECG. MI is a dynamic disease process and this is reflected in the ECG (Table 2). A significant proportion of initial ECGs are reported as normal for patients who eventually have a confirmed diagnosis of AMI. In the setting of an acutely ill patient presenting with symptoms of MI, the patient should either have serial ECGs or dynamic ST-segment monitoring.

Dr. Magee is an Associate Professor and Program Director, Royal College Residency, Department of Emergency Medicine, Dalhousie University, Halifax, Nova Scotia.

The possibility of a cardiac etiology should always be seriously considered in patients who have a strong family history of MIs under the age of 40 years or who use cocaine or amphetamines.

Diagnosing STEMI in the setting of left bundle branch block (LBBB) is difficult because ST-segment elevation is often masked. Comparison with old ECGs may be helpful. Looking for concordant (in the same direction as the dominant RS complex) ST-changes > 1 mm or an ST-segment elevation ≥ 5 mm in V1 to V3 is suggestive of AMI. However, the absence of these changes should not be assumed to rule out the diagnosis as these guidelines are specific rather than sensitive. Real-time consultation with a cardiologist is recommended in those cases where the diagnosis is not clear.

Recognition of Wellen's syndrome is of particular importance to the primary care physician. In the setting of a recent history of chest pain with negative cardiac markers, biphasic or deep symmetrically inverted T-waves in V2 to V5 (Figure 1) is suggestive of a proximal left anterior descending artery lesion at high risk for imminent occlusion. These patients require rapid referral to a cardiologist.

Table 2

ECGs of which to be wary

- Normal ECG in setting of the acutely symptomatic patient
- Left bundle branch block
- Wellen's syndrome

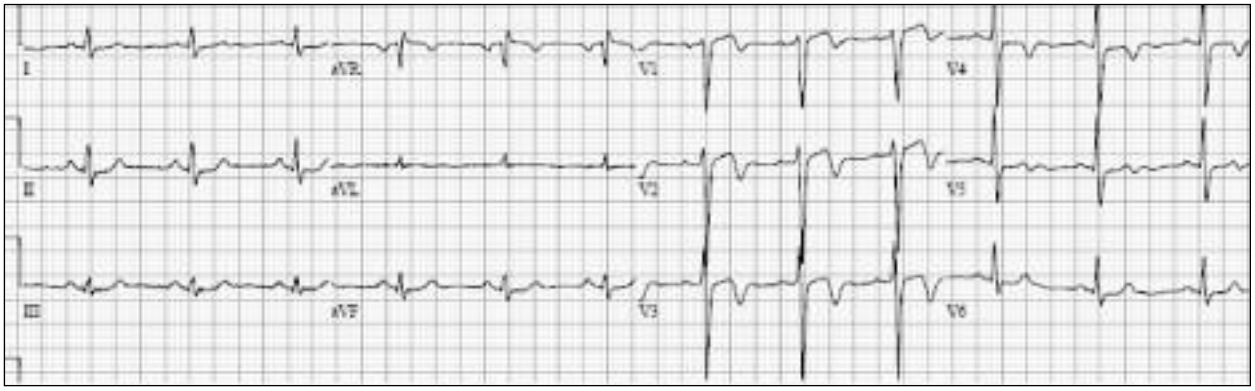

Figure 1. Wellen's syndrome.

Thrombolysis vs. PCI

Having diagnosed STEMI, the clinician is then faced with the choice of primary percutaneous coronary intervention (PCI) or medical thrombolyis (Table 3). In the absence of rapid access to primary PCI, 24 hours, seven days a week, the decision to call in the catheter team or to transport the patient to an alternative institution must be weighed against the benefit of immediate thrombolysis. Though PCI has consistently been shown to be superior, this benefit is time-dependant. In the absence of contraindications to thrombolysis (Table 4), current guidelines discourage the use of primary PCI if the door-to-balloon time minus the door-to-needle time is > 60 minutes. Furthermore, in those emergency medical service (EMS) systems so-equipped, pre-hospital thrombolysis should be considered if the delay from EMS to needle time is > 30 minutes.

Table 3

Indications for invasive strategy

- Skilled percutaneous coronary intervention lab with surgical back-up
  - Delay of < 60 minutes from when fibrinolytic could be administered
- High risk STEMI
  - Cardiogenic shock
- Contraindications to fibrinolysis
- Diagnosis of STEMI is unclear

Table 4

Absolute contraindications for thrombolysis therapy

- Prior intracranial hemorrhage
- Known structural cerebral vascular lesion or neoplasm
- Active bleeding or bleeding diathesis
- Suspected aortic dissection
- Severe persistent hypertension despite treatment
- Recent head trauma, cerebral vascular accident in past 3 months

Adjunct medications

Ultimately, the pathophysiology of AMI is a result of an imbalance between myocardial oxygen demand vs. supply. Treatment with oxygen, acetylsalicylic acid, nitroglycerin, morphine and β-blockers is important to shift this equilibrium in favour of oxygen delivery. Those patients that are candidates for primary PCI should receive additional pharmacologic support with unfractionated heparin, clopidogrel and an early glycoprotein IIb/IIIa inhibitor.

cme

Resource

1. Antman EM, Anbe DT, Armstrong PW, et al: ACC/AHA Guidelines for the Management of Patients with ST-Elevation Myocardial Infarction: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of Patients with Acute Myocardial Infarction). Circulation 2004; 110(9):e82-292.