Acute Myocardial Infarction: Pre-hospital Issues 2 CEUs
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INTRODUCTION

An in depth review of the pathophysiology, symptomatology, and treatment modalities for acute MI. It covers most of the major literature support for each currently recommended treatment.

Objectives

By the end of this lecture, the participant should be able to...

1. Describe coronary artery anatomy
2. List risk factors for coronary artery disease
3. Explain pathophysiology of angina and acute myocardial infarction (AMI)
4. Recognize signs and symptoms of unstable angina and the acute MI
5. Explain pre-hospital management of myocardial infarction
6. Review studies of 12-lead ECG's in the field
7. Describe latest treatment of the acute MI

Cardiovascular Statistics

- According to current estimates, 60,800,000 Americans have one or more types of cardiovascular disease (CVD). This includes coronary artery disease, hypertension, angina, stroke, prior MI, congestive heart failure and congenital cardiovascular defects.

- Since 1900, CVD has been the number 1 killer in the United States every year except 1918. More than 2,600 Americans die of CVD each day! CVD claims almost 10 thousand more lives each year that the next 6 leading causes of death combined!

- It is what you will see most in practice and what you need to know thoroughly.

References:

Pathophysiology of Coronary Artery Disease (CAD)

- It can be argued that much of CAD comes from evolutions poor design of the heart. Here is the vital organ delivering oxygenated blood to the entire body and the heart itself has only 3 major vessels providing it's blood supply! A better design would have at least three times that number of vessels so a blockage in one would not cause such devastating
consequences. But since that is not the case we need to understand how blockages and MI's occur so this can be prevented.

- The heart has 4 chambers.
- Two thin walled atriums and two thick walled ventricles.
- The ventricles are thicker as they do the major pumping work.
- Blood returns from the body via veins to the right atrium where it passes through the tricuspid valve to the right ventricle and gets ejected through the pulmonic valve into the pulmonary artery.
- Blood then enters the pulmonary circulation in the lungs where carbon dioxide is exchanged for oxygen.
- Blood then travels back through the pulmonary veins to the left atrium where oxygenated blood flows through the mitral valve to the left ventricle and is pumped out of the aorta to the entire body.

**Coronary Anatomy**

- The coronary arteries are what distribute blood to the myocardium (heart muscles cells). There are 3 main arteries that supply the heart and get blood from a small branch off the aorta. The left anterior descending coronary artery, right main, and left circumflex are the major arteries supplying the heart.
- The left main coronary artery bifurcates within a few centimeters of its start to form 2 of 3 main vessels, the left anterior descending and left circumflex arteries. The LAD or left anterior descending coronary artery proceeds down the heart between the two ventricles. The left circumflex coronary artery travels around the side of the heart between the left atrium and left ventricle.
- The right coronary artery travels in a groove between the right atrium and ventricle and 90 percent of the time supplies the AV node.
- Blood to the sinus node is from a branch of the right coronary artery a little more than half the time. Otherwise it comes from a branch of the circumflex.
- Atherosclerotic plaque can build up in any or all of these coronary arteries. Blockage in any one of these main arteries or their branches results in myocardial cell death, which is the definition of myocardial infarction or heart attack.
- You can imagine that depending on which vessel is blocked there will be a different part of the heart muscle affected and different clinical and ECG findings.

What are the risk factors for developing coronary artery disease?

- Age
- Male sex
- Elevated LDL cholesterol
- Low HDL cholesterol
- Smoking
- Hypertension
- Diabetes
- Elevated homocysteine levels
- Stress, anger, depression and physical inactivity have also been shown to be risk factors for cardiovascular events through various mechanisms.

**Acute Coronary Syndromes**

- Unstable angina, Q wave MI, non-Q wave MI all represent acute coronary syndromes. It is important to recognize that these three terms simply represent various degrees of the same physiologic process.
- When a person has high cholesterol, smokes, has diabetes or any or all of the discussed risk factors atherosclerotic plaques develop. When a sufficient degree of narrowing has occurred a person will experience symptoms with a certain level of exertion. For example, every time John climbs 2 flights of stairs he gets chest tightness. He is fine if he avoids stairs or climbs only 1 flight but at 2 flights the degree of oxygen demanded by his heart muscle cannot be met by his diseases coronary arteries and he gets chest pain. This is stable angina. John may go on for years avoiding stairs, taking the elevator and in general exerting himself less.
- Now if John begins to get chest pain at rest, this is unstable angina. Something has changed. The atherosclerotic plaques have progressed or there has been some plaque rupture and subsequent thrombus formation. Now one morning John wakes up at 6am diaphoretic, nauseous, with chest tightness and shortness of breath. John is having an acute myocardial infarction. There has been a rupture or fissure of one of the atherosclerotic plaques and platelets are activated, thrombin generated and a cascade of factors that result in complete blockage of one of John's vessels. Heart muscle cells supplied by that vessel are dying. "Time is muscle."
- Unfortunately John does not call 911 right away. When looking at where the longest delay to thrombolytics occurs it is in patients accessing EMS quickly.
- Once called, EMS quickly responds. John has the following signs and symptoms:
  - Chest tightness/heaviness
  - Jaw tightness
  - Pain radiating down his left arm
  - Nausea
  - Palpitations
  - Lightheadedness (pre syncope)
  - Shortness of breath
- On exam he is:
  - Diaphoretic
  - Hypotensive BP 90/40
  - Bradycardia HR 50
  - Pulse irregular at times
  - Lungs clear
  - Jugular venous distention
- You assess from the signs and symptoms that John is having an acute MI
Right! Even before you get the ECG you assume this is an acute inferior myocardial infarction.

John has JVD (jugular venous distention) and he is hypotensive. Hypotension can occur in a large LV infarct causing cardiogenic shock but then you would likely here rales, signs of heart failure, on lung exam. Also sinus bradycardia often occurs early in an acute inferior myocardial infarction. This is related to the RCA (right coronary artery) supplying the SA node. Heart block is common on ECG as the RCA also usually supplies the AV node.

You conclude acute inferior myocardial infarction caused by plaque rupture and thrombus formation in the right coronary artery.

This case seems straightforward but often the presentation is not so clear. Here are other cardiac and non-cardiac causes of chest pain.

**Differential Diagnosis of Chest Pain**

- Unstable angina - not acute MI
- Pericarditis
- Dissecting aortic aneurysm
- Pulmonary edema
- Pneumonia
- Spontaneous pneumothorax
- Herpes zoster
- Costochondritis
- Esophageal reflux
- Peptic ulcer disease
- Cholecystitis

**Remember:** many patients present with atypical symptoms and are having an AMI. This is particularly true with women, the elderly and the diabetic patient. Always assume the worst diagnosis!

References:

The Acute Myocardial Infarction: Pre-hospital Issues in Management

**Now you begin to treat John:**
1. "Time is muscle" Don't waist time before transport.
2. Minimize anxiety. Place him at rest in a comfortable position. AMI patients are anxious and the catecholamine surge only makes things worse.
3. Administer high flow oxygen. Get as much oxygen as you can to those heart muscle cells that need it.
4. Establish an IV either at the seen if it will not delay you or in route. Avoid too many attempts, as this patient may be a candidate for thrombolysis.
5. Start IV fluids. NS as this patient is hyposensitive.
6. If possible do an ECG or 3-lead ECG tracing. This is important because the patient may be pain free on arrival to the ER and the tracing may be normal.
7. Measure any ST segment change and communicate with receiving facility.
8. Assume all patients are candidates for thrombolytics so you do not waste time.
9. Treat with nitroglycerin sublingual either tablet or spray if blood pressure allows. The patient may need to get some IV fluid first if hypotensive.
10. Administer medication per written protocols or by medical director.
11. Ask first about allergies to medications.
12. If no contraindications, give aspirin 160-325 mg PO.
13. Administer morphine sulfate for pain MSO4 2-4 mg IV Q 5 min x3 doses PRN chest pain and systolic BP >100 mmHg.
14. β blockers are beginning to hit the scene pre-hospital. Patients experiencing symptoms of Acute MI or unstable angina should receive early beta-blockers unless contraindicated. The dose is Metoprolol 5mg IV Q 5 min x3. Hold for SBP less than 100, HR less than 60.

**Contraindications for beta-blockers**

Contraindications for beta-blockers:
- Hypotension: SBP less than 100 mmHg
- Bradycardia: HR less than 60 beats/min
- Presence of heart block 1st, 2nd or 3rd degree AV block
- Strong history of asthma. Beta-blockers cause bronchoconstriction
- Intolerance or history of allergy to beta-blocker use in the past

- Our patient John would not receive metoprolol, as he is bradycardic.

**Out-of-hospital ECG and Thrombolytics**

*What does the data tell us?*

- More and more 12-lead ECG's are becoming available for pre-hospital personnel. Let us examine what studies demonstrate regarding how good out-of-hospital ECG's are for evaluating patients with suspected Acute Myocardial Infarction (AMI) and Acute Cardiac Ischemia (ACI). Many studies have been done since the report from the National Heart Attack Alert Program was issued.
- These trials were recently analyzed by meta-analysis and published in the Annals of Emergency Medicine in May 2001.
- Based on 11 studies with 7,508 patients, as expected out-of-hospital ECG has excellent diagnostisic value performance for AMI and good performance for ACI.
- Out-of-hospital ECG does not appear to prolong in field time and if used with thrombolytics about ¾ to 1 hour is saved. Thrombolytics pre-hospital achieves time savings and improved short-term mortality. Long-term benefits are less clear.

**References:**

Thrombolytics (clot busting)

- Your area may have a protocol for pre-hospital use of thrombolytics or may be developing one. You need to know what you are using, the indications and contraindications for use.

Thrombolytic Therapy

The recommendations are summarized in treatment guidelines.

Patient Selection Criteria

1. Chest pain typical of MI > 30 minutes
2. Elapsed time from onset of worst pain to evaluation
3. ECG evidence of acute MI:
   - **ST Segment elevation greater or equal to 1 mm in contiguous leads:**
     - 2 Inferior leads
     - 2 Precordial leads
     - 2 Lateral leads
     - New LBBB

Strong contraindications

- The Cardiology Service would generally not recommend thrombolytic therapy under the following circumstances:
  1. Significant surgical procedure within 2 weeks
  2. Recent GI bleed
  3. Hypertension: systolic BP > 180 mmHg or diastolic > 100 mmHg
  4. History of stroke
  5. Prolonged CPR
  6. Recent severe trauma
  7. Cerebral AVM, tumor or intracranial surgery

Related Contraindications

1. Any past history of GI bleed
2. History of CNS structural lesion
3. Pregnancy
4. Unsuccessful central venous puncture
5. Any severe trauma within 6 months
6. Onset of severe chest pain > 12 hours

References:

1. Baystate Medical Center. Springfield, MA