

MWLCEMS SYSTEM
Continuing Education Packet
Management of the Acute MI Patient

In this CE we will discuss the patient presenting with an acute ST-Elevation Myocardial Infarction (STEMI)

Definition:

Myocardial injury (as evidenced by ST-segment elevation) that progresses to irreversible necrosis (as evidenced by the rise and fall in Troponin or CK-MB). STEMI patients require reperfusion therapy (PCI-percutaneous coronary intervention, fibrinolytic therapy, or CABG-coronary artery bypass graft).

Facts:

- Frequency of thrombotic events is highest from 6am to 12 noon.
- 20 – 30% have no chest pain. The average STEMI patient does not seek medical care for about 2 hours after the onset of pain.
- 1/3 of STEMI patients die within 24 hours, usually from V-Fib.
- Mortality increases with the number of ECG leads with ST elevation.
- 20% of AMI patients will have heart failure within 6 years.

Pathophysiology:

Coronary Thrombus

Coronary thrombus is the primary cause of ST-elevated MIs. Injury to the coronary artery endothelium attracts platelets, white blood cells, fibrin and lipids. Macrophages infiltrate the damaged lining and engorge with substances, then die. A fibrous scar (or cap) forms which narrows the lumen and secretes procoagulant factors. When the fibrous cap ruptures (due to physical or hemodynamic substances) the GP IIb/IIIa receptors on the platelet surface are activated and bind to fibrinogen, which forms a bridge between platelets, collecting them in a growing mass.

Twenty to forty minutes of occlusion leads causes irreversible myocardial damage. Six to eight hours of occlusion leads to death of most of the distal myocardium.

Catecholamines cause an increase in heart rate, blood pressure, systemic vascular resistance and blood volume which in turn increases myocardial oxygen demand.

With death of cells there is release of intracellular contents (including potassium, CK, CK-MB, lactate) which causes ventricular arrhythmias. Ischemia of the SA, AV node or the conducting system will also cause arrhythmias.

Other causes of STEMI

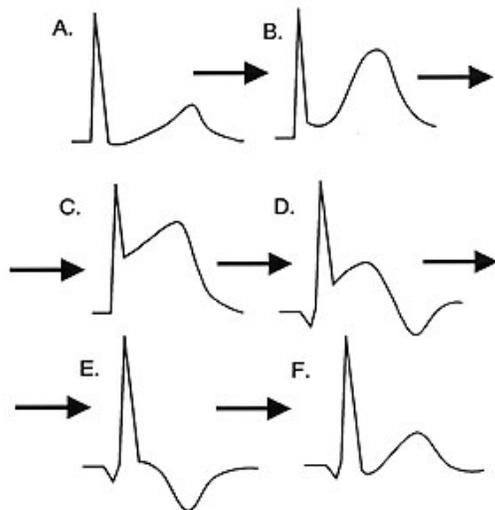
Hypotension, hypoxemia and chemical toxins.

The ECG

For a STEMI to be present there must be ST-elevation of 1mm in at least 2 continuous leads.

- In the very early phase of a STEMI, hyperacute T waves may precede ST elevation.
- Subendocardial MI (multifocal areas of necrosis confined to the inner 1/3-1/2 of the left ventricular wall) does not generate Q waves, but rather ST depression or inverted T waves, and is treated as NSTEMI (non-ST-elevated MI)
- ST-segment depression in the precordial leads represents posterior wall insult and is also considered NSTEMI.
- Q waves indicate transmural infarction (involving the entire thickness of the left ventricular wall from endocardium to epicardium) to the epicardium, but may take hours to develop (1 – 20% of Q waves are false and do not mean MI).

Sequence of changes in acute MI



Evolution of Acute MI

A) Shows the normal QRS complex in a lead.

B & C) Within **hours** of the clinical onset of an MI, there is **ST segment elevation**. At this stage no QRS or T wave changes have occurred. This indicates myocardial damage only, not definitive evidence of infarction.

D) Within **days**, the R wave voltage falls and abnormal Q waves appear. This is sufficient evidence of an infarction. In addition, T wave inversion will also have appeared but the ST segment elevation may be less obvious than before.

E) Within **one or more weeks**, the ST segment changes revert completely to normal. The R wave voltage remains low and the abnormal Q waves persist. Deep, symmetrical T wave inversion may develop at this stage.

F) **Months** after the MI, the T waves may gradually return to normal. The abnormal Q waves and reduced R wave voltage persist.

Occasionally, all evidence of infarction may be lost with the passing of time; this is due to shrinkage of scar tissue.

Lab Values

In the presence of a non-diagnostic ECG, lab markers can be diagnostic in AMI. They include the serum isoenzyme creatine kinase and the serum muscle protein troponin.

CK-MB – Creatine kinase, myocardial band (CK-MB) immunochemical testing is possible within several hours after onset of AMI. AT 3 hours, sensitivity and specificity are greater than 90 percent, and at 10 to 12 hours there is almost 100 percent sensitivity. If the value of CK-MB is elevated and the ratio of CK-MB to total CK (relative index) is more than 2.5 to 3, it is likely that myocardium damaged. A high CK with a relative index below this value suggests that skeletal muscle was damaged.

Troponin – Troponin is a family of proteins found in skeletal and cardiac muscle, though the test used for AMI detect only the troponin released from damaged myocardium into the blood, Troponin T. Troponin T has a 98% diagnostic accuracy for AMI.

Management

All interventions used in managing an AMI patient are done to achieve one of the four goals needed to attain a positive outcome. The goals of management are:

- Increase myocardial oxygen supply
- Decrease myocardial oxygen demand
- Correct disturbances to the heart rate and rhythm
- Reduce pain and relieve stress

Increase myocardial oxygen supply

This is accomplished by administering supplemental oxygen via nasal cannula or mask. Oxygen should be administered to maintain oxygen saturations of 94 – 99%. Improving coronary blood flow further increases myocardial oxygen supply. Nitrates and calcium channel blocker improve coronary blood flow via direct smooth muscle relaxation. The goal of transferring the patient to a center with a cardiac cath lab is to perform PTCA to open up the involved vessel thereby increasing the oxygen supply to the muscle.

Decrease myocardial oxygen demand.

Nitrates, beta-blockers, and calcium channel blockers decrease myocardial oxygen demand. Nitrates accomplish this by reducing preload and afterload. Beta-blockers inhibit beta-adrenergic receptors, thus decreasing heart rate and contractility. Calcium channel blockers decrease myocardial contractility. They also vasodilate peripheral arteries, thus reducing afterload and thereby decreasing myocardial oxygen demand.

Correct disturbances to the heart rate and rhythm.

During the management of an acute MI cardiac rate and rhythm disturbances may present and must be corrected immediately. Many serious arrhythmias develop before hospitalization. At least 75% of patients with MI have an arrhythmia during the peri-infarct period. Both atrial and ventricular arrhythmias can be seen during and after the acute phase of STEMI. These include atrial fibrillation or flutter, which can cause symptomatic hypoperfusion due to a rapid rate, and life-threatening ventricular tachycardia or ventricular fibrillation. Sinus bradycardia can occur in patients with STEMI, especially when the inferior wall is involved. Atrioventricular nodal and intraventricular conduction abnormalities also may be seen in STEMI, particularly of the anterior wall. Heart rate and rhythm must be monitored constantly during transfer and dysrhythmias treated per protocols.

Reduce pain and relieve stress

Both of these interventions will contribute to the lessening workload placed on the heart by heightened sympathetic tone from the stress response. The goal for pain reduction is "0." That means the goal is to continue to relieve pain until the patient states their pain is a "0" on a scale of "1 to 10." This is achieved with the use of oxygen, nitroglycerin and pain medications. The relief of stress should be done by constant verbal reassurance to ease the mind of the patient and keeping them in a comfortable position during transport.

Heparin:

Heparin increases the ability of antithrombin to inactivate circulating thrombin, preventing thrombus formation and progressive thrombus development.

The dose should be reduced for the elderly or females

Dose should be increased for diabetics, smokers and the obese.

Heparin infusion should be continued for a minimum of 48 hours. (ED may bolus the patient for short transfers so heparin drip and be discontinued during transfer and restarted at receiving facility)

Complications of Myocardial Infarction:

Complication	Clinical Indications	Prevention/Treatment
Dysrhythmias and conduction system defects	<ul style="list-style-type: none"> • Change in rhythm or conduction on rhythm strip or 12 Lead • Indications of hypoperfusion may be evident 	<ul style="list-style-type: none"> • Close monitoring for changes in rhythm or conduction • Beta-blocker as a cardioprotective agent • Correct electrolyte imbalances • Antidysrhythmic agents as indicated and prescribed • Application of external pacemaker if indicated • Cardioversion or defibrillation as indicated
Heart Failure	<ul style="list-style-type: none"> • Tachycardia, tachypnea • Clinical indications of Left Ventricular Failure (dyspnea, orthopnea, cough, crackles in lung bases) • Clinical indications of Right Ventricular Failure (JVD, hepatomegaly, peripheral edema) • Chest X-ray shows pulmonary venous congestion 	<ul style="list-style-type: none"> • Oxygen • Sodium and fluid restriction • ACE inhibitors • Beta-blockers • Diuretics • Vasodilators • Inotropic agents
Cardiogenic Shock	<ul style="list-style-type: none"> • Tachycardia, tachypnea, hypotension • Clinical indications of LVF • Clinical indications of RVF • Urine output less than 0.5ml/kg/hr • Cool to cold skin • Lethargy to confusion to coma 	<ul style="list-style-type: none"> • Oxygen • Sodium and fluid restriction • ACE inhibitors • Inotropic agents (dobutamine, dopamine)
Cardiac Rupture	<ul style="list-style-type: none"> • Clinical indications of hypoperfusion • Clinical indications of cardiac tamponade • Sinus tachycardia or PEA • Eventual cardiopulmonary arrest 	<ul style="list-style-type: none"> • CPR if indicated • Pericardiocentesis by physician
Sudden Cardiac Death	<ul style="list-style-type: none"> • Cardiopulmonary arrest 	<ul style="list-style-type: none"> • CPR and ACLS procedures

7. The goal for pain management is _____ on a 1 to 10 scale.

8. Explain why heparin is used in the management of an acute MI

9. Explain why it is important to check the patient electrolytes prior to transfer.

10. You are transferring a STEMI patient from CH-W to CH-M cath lab. Patient suddenly becomes unresponsive and your monitor shows V-Fib. Explain in detail your plan of care.

If you are NOT a member of the McHenry Western Lake County EMS System, Please include your address on each optional quiz turned into our office. Our mailing address is: Northwestern Medicine – McHenry Hospital EMS, 4201 Medical Center Drive, McHenry, Illinois 60050. We will forward to your home address verification of your continuing education hours.

If you ARE a member of our EMS System, your credit will be added to your Image Trend record. Please refer to Image Trend to see your current list of continuing education credits. Any questions regarding this can be addressed to Cindy Tabert at 224-654-0160. Please fax your quiz to Cindy Tabert at 224-654-0165.