VITEBSK STATE MEDICAL UNIVERSITY

CHAIR OF THE FACULTY THERAPY

**FACULTY THERAPY**

(Methodics work out for foreign students, studying the course of faculty therapy)

Part 1

Vitebsk – 2005

**MYOCARDIAL INFARCTION**

Synonyms and related keywords: MI, acute myocardial infarction, AMI, heart attack

Myocardial infarction (MI) is irreversible necrosis of heart muscle secondary to prolonged ischemia. This usually results from an imbalance of oxygen supply and demand. The appearance of cardiac enzymes in the circulation generally indicates myocardial necrosis. MI now is considered, more appropriately, part of a spectrum referred to as acute coronary syndromes (ACSs), which also includes unstable angina and non–Q-wave MI (NQWMI).

Patients with ischemic discomfort may or may not have ST-segment elevation. The majority of those with ST-segment elevation will develop Q waves. Those without ST elevation ultimately will be diagnosed with unstable angina or NQWMI on the basis of the presence of cardiac enzymes.

MI may lead to impairment of systolic or diastolic function and to increased predisposition to arrhythmias and other long-term complications.

**Frequency**

Cardiovascular diseases cause 12 million deaths throughout the world each year, according to the third monitoring report of the World Health Organization, 1991-93. They cause half of all deaths in several developed countries and are one of the main causes of death in many developing countries; they are the major cause of death in adults everywhere.

Ischemic heart disease is the leading cause of death worldwide.

Approximately 6.3 million deaths due to heart disease occurred in 1990 worldwide, which represents 29% of all deaths. The prevalence of coronary artery disease (CAD) is increasing rapidly in nonindustrialized countries.

Male predilection exists in persons aged 40-70 years. In persons older than 70 years, no sex predilection exists. Acute MI occurs most frequently in persons older than 45 years.

Certain subpopulations younger than 45 years are at risk, particularly cocaine users, insulin-dependent diabetics, patients with hypercholesterolemia, and those with a positive family history for early coronary disease.

A positive family history includes any first-degree male relative aged 45 years or younger or any first-degree female relative aged 55 years or younger who experienced a myocardial infarction.

**Pathophysiology**

 Atherosclerosis is the disease primarily responsible for the majority of ACS cases. Approximately 90% of MIs result from an acute thrombus that obstructs an atherosclerotic coronary artery. Plaque rupture is considered to be the major trigger of coronary thrombosis. Following plaque rupture, platelet activation and aggregation, coagulation pathway activation, and endothelial vasoconstriction occur and lead to coronary thrombosis and occlusion.

Consider nonatherosclerotic causes of acute MIs in younger patients or if no evidence of atherosclerosis is noted. Such causes include coronary emboli from sources such as an infected cardiac valve, coronary occlusion secondary to vasculitis, primary coronary vasospasm (variant angina), cocaine use, or other factors leading to mismatch of oxygen supply and demand, as may occur with a significant GI bleed.

Risk factors for atherosclerotic plaque formation include the following:

* Age
* Being male and younger than 70 years
* Smoking
* Hypercholesterolemia and hypertriglyceridemia
* Diabetes mellitus
* Poorly controlled hypertension
* Type A personality
* Family history
* Sedentary lifestyle

**CLINICAL**

**History**

* Chest pain, usually across the anterior precordium, is described as tightness, pressure, or squeezing.
* Pain may radiate to the jaw, neck, arms, back, and epigastrium. The left arm is affected more frequently than the right arm.
* Dyspnea, which may accompany chest pain or occur as an isolated complaint, indicates poor ventricular compliance in the setting of acute ischemia.
* Nausea and/or abdominal pain often are present in infarcts involving the inferior wall.
* Anxiety
* Lightheadedness and syncope
* Cough
* Nausea and vomiting
* Diaphoresis
* Wheezing
* Elderly patients and those with diabetes may have particularly subtle presentations and may complain of fatigue, syncope, or weakness.

**Physical**

Frequently, physical examination findings are normal.

Patients with ongoing symptoms usually will lie quietly in bed and appear pale and diaphoretic.

Hypertension may precipitate AMI, or it may reflect elevated catecholamines due to anxiety, pain, or exogenous sympathomimetics.

Hypotension indicates ventricular dysfunction due to ischemia. It usually indicates a large infarct and may be observed with a right ventricular infarct.

**Lab Studies**

* Creatine kinase–MB (CK-MB)

This is the criterion standard for detection of myocardial necrosis.

Levels begin to rise within 4 hours after injury, **peak at 18-24 hours**, and subside over 3-4 days.

Normal range values for CK-MB is 0-20 ed.

* Myoglobin

This is a very sensitive early marker of acute myocardial necrosis, but it is not specific for myocardial cell necrosis.

* Troponin I

This is a contractile protein that normally is not found in serum. It is released only when myonecrosis occurs.

For early detection of myocardial necrosis, sensitivity of this study is superior to that of the CK-MB. Troponin I is detectable in serum 3-6 hours after an AMI, and its level remains elevated for 14 days.

Troponin T has similar release kinetics and specificity for myocardial necrosis, but it is slightly less sensitive than troponin I within the first 6 hours.

* Complete blood count

Leukocytosis may be observed within several hours after an AMI. It peaks in 2-4 days and returns levels within the reference range within 1 week.

CBC may be useful if anemia is suspected as a precipitant. Transfusion with packed red blood cells and supplemental oxygen may be the only treatment modalities in cases where anemia is considered the only precipitant.

Erythrocyte sedimentation rate (ESR) rises above reference range values within 3 days and may remain elevated for several weeks.

The serum lactase dehydrogenase (LDH) level rises above the reference range within 24 hours of an AMI, reaches a peak within 3-6 days, and returns to the baseline within 8-12 days.

**Imaging Studies**

* **Electrocardiogram**

An electrocardiogram (ECG) should be obtained as soon as possible after presentation to the emergency depatment. The ECG is the most important tool in the initial evaluation and triage of patients in whom an ACS is suspected. It is confirmatory of the diagnosis in approximately 80% of cases. Approximately one half of patients have diagnostic changes on their initial ECG.

Obtain an ECG immediately if MI is considered or suspected.

Convex ST-segment elevation with upright or inverted T waves generally is indicative of MI in the appropriate clinical setting.

ST depression and T-wave changes also may indicate evolution of NQWMI.

Results indicating high probability of AMI are ST-segment elevation greater than 1 mm in 2 contiguous leads and new Q waves.

Results indicating intermediate probability of AMI are ST-segment depression or T-wave inversion, Q waves, and ST-T wave abnormalities that are known to be old.

Results indicating low probability of AMI are normal findings on ECG; however, normal or nonspecific findings on ECG do not exclude the possibility of AMI.

Localization of AMI based on distribution of ECG abnormalities is as follows:

Inferior wall - II, III, aVF

Lateral wall - I, aVL, V4-V6

Anteroseptal - V1-V3

Anterolateral - V1-V6

Right ventricular - RV4, RV5

Posterior wall - R/S ratio >1 in V1 and V2; T-wave changes (ie, upright) in V1, V8, and V9

* Chest radiography

Chest radiography may provide clues to an alternative or complicating diagnosis (eg, aortic dissection, pneumothorax).

Chest radiography also reveals complications of AMI, particularly pulmonary edema, and CHF.

* Echocardiography

Use 2-dimensional and M-mode echocardiography when evaluating wall motion abnormalities and overall ventricular function.

This also can identify complications of AMI (eg, valvular insufficiency, ventricular dysfunction, pericardial effusion).

* Technetium-99m sestamibi scan

Technetium-99m is a radioisotope that is taken up by the myocardium in proportion to the blood flow and is redistributed minimally after injection. This allows for time delay between injection and imaging.

It has potential use in identifying infarct in patients with atypical presentations or uninterpretable ECGs.

Normal scan findings are associated with an extremely low risk of subsequent cardiac events.

Thallium scanning: Thallium accumulates in the viable myocardium.

**TREATMENT**

**Prehospital Care**

All patients being transported for chest pain should be managed as if the pain were ischemic in origin unless clear evidence to the contrary exists.

If available, an ALS unit should transport patients with hemodynamic instability or respiratory difficulty.

Prehospital notification by Emergency Medical Services (EMS) personnel should alert ED staff to the possibility of an AMI. EMS personnel should receive online medical advice for a patient with high-risk presentation.

Specific prehospital care includes the following:

* IV access, supplemental oxygen, pulse oximetry
* Immediate administration of aspirin en route
* Nitroglycerin for active chest pain, given sublingually (SL) or by spray
* Prehospital thrombolysis allows eligible patients to receive thrombolysis 30-60 minutes sooner than if treatment was given in the ED; however, prehospital thrombolysis is still under investigation (Thrombolysis in Myocardial Infarction [TIMI] and other trials).
* Prehospital ECG, if available.

**Emergency Department Care**

Initial therapy for acute MI is directed toward restoration of perfusion in order to salvage as much of the jeopardized myocardium as possible. This may be accomplished through medical or mechanical means, such as angioplasty or coronary artery bypass grafting.

Further treatment is based on

(1) restoration of the balance between the oxygen supply and demand to prevent further ischemia,

(2) pain relief,

(3) prevention and treatment of any complications that may arise.

Thrombolytic therapy has been shown to improve survival rates in acute MI if administered in a timely fashion in the appropriate group of patients. The optimal approach is to administer thrombolytics within 12 hours of onset of symptoms in patients with ST-segment elevation greater than 0.1 mV in two or more contiguous ECG leads, new left bundle-branch block (LBBB), or anterior ST depression consistent with posterior infarction. Tissue plasminogen activator (t-PA) is superior to streptokinase in achieving a higher rate of coronary artery patency; however, the key to efficacy lies in the speed of delivery of therapy. Recent trials show an even greater patency rate if a IIb/IIIa receptor antagonist, such as abciximab, is combined with a half dose of a thrombolytic agent as the initial reperfusion strategy. The reduced dose of a thrombolytic agent combined with a potent platelet inhibitor may prove to be the preferred method for medical reperfusion, but larger clinical trials are pending.

Aspirin and/or antiplatelet therapy

Aspirin has been shown to decrease mortality and re-infarction rates after MI. Administer aspirin immediately, which the patient should chew if possible upon presentation. Continue aspirin indefinitely unless an obvious contraindication, such as a bleeding tendency or an allergy, is present. Clopidogrel may be used as an alternative to aspirin in cases of aspirin resistance or allergy.

Administer a platelet glycoprotein (GP) IIb/IIIa-receptor antagonist, in addition to acetylsalicylic acid and unfractionated heparin (UFH), to patients with continuing ischemia or with other high-risk features and to patients in whom a percutaneous coronary intervention (PCI) is planned. Eptifibatide and tirofiban are approved for this use. Abciximab also can be used for 12-24 hours in patients with unstable angina or non–ST-segment elevation MI in whom a PCI is planned within the next 24 hours.

Beta-blockers reduce the rates of reinfarction and recurrent ischemia and possibly reduce mortality rate if administered within 12 hours after MI. Administer routinely to all patients with MI unless a contraindication is present.

Heparin (and other anticoagulant agents) has an established role as an adjunctive agent in patients receiving t-PA but not with streptokinase. Heparin also is indicated in patients undergoing primary angioplasty. Little data exist with regard to efficacy in patients not receiving thrombolytic therapy in the setting of acute MI. Low–molecular-weight heparins (LMWHs) have been shown to be superior to UFHs in patients with unstable angina or NQWMI.

Nitrates have no apparent impact on mortality rate in patients with ischemic syndromes. Their utility is in symptomatic relief and preload reduction. Administer to all patients with acute MI within the first 48 hours of presentation, unless contraindicated (ie, in RV infarction).

ACE inhibitors reduce mortality rates after MI. Administer ACE inhibitors as soon as possible as long as the patient has no contraindications and remains in stable condition. ACE inhibitors have the greatest benefit in patients with ventricular dysfunction. Continue ACE inhibitors indefinitely after MI. Angiotensin-receptor blockers may be used as an alternative in patients who develop adverse effects, such as a persistent cough, although initial trials need to be confirmed.

**Surgical Care**

Primary angioplasty

Primary percutaneous transluminal coronary angioplasty (PTCA) is an attractive alternative to thrombolytic therapy. PTCA provides greater coronary patency (>96% thrombolysis in myocardial infarction [TIMI] 3 flow), lower risk of bleeding, and instant knowledge about the extent of the underlying disease. Some studies have shown that primary PTCA has a mortality benefit over thrombolytics; however, the data are limited, and long-term benefit remains in question. The widespread use of stenting and adjunctive IIb/IIIa therapy are improving the results of primary PTCA. A recently published trial showed that, in patients with acute MI, coronary stenting plus abciximab leads to a greater degree of myocardial salvage and a better clinical outcome than does fibrinolysis with a t-PA. Improvement of long- and short-term outcomes, however, depends highly on the speed with which reperfusion is achieved. However, a body of evidence is accumulating in support of primary angioplasty as a more effective therapy than thrombolytics.

Cardiac catheterization and angioplasty are indicated in patients who do not fit the above-mentioned criteria for thrombolytic therapy or have persistent ischemia. Primary angioplasty also is the treatment of choice in patients with cardiogenic shock, patients in whom thrombolysis failed, and those with high risk of bleeding or contraindications to thrombolytic therapy.

Only an experienced operator should perform primary PTCA, and PTCA should be performed only where the appropriate facilities are available. Operators should have at least 75 cases per year, while the center should perform at least 200 cases per year as per the recommendations of the ACC.

Emergent or urgent coronary artery graft bypass surgery is indicated in patients in whom angioplasty fails and in patients who develop mechanical complications such as a VSD, LV rupture, or a papillary muscle rupture.