Acute ST Elevation Myocardial Infarction

(See also *Harrison's Principles of Internal Medicine*, 17th Edition, Chapter 239)

Definition

- Acute ST-elevation myocardial infarction (STEMI)
  - Characterized by clinical findings of acute infarction and ST-segment elevation on 12-lead electrocardiogram (ECG)
  - Generally occurs when coronary blood flow decreases abruptly after a thrombotic occlusion of a coronary artery
  - Early identification and reperfusion are critical for reducing morbidity and mortality.
- See Unstable Angina and Acute Non–ST-Elevation Myocardial Infarction.

Epidemiology

- Incidence
  - One of the most common diagnoses in hospitalized patients in industrialized countries
  - In the U.S.
    - ~650,000 patients have a new acute MI each year.
      - ~370,000 are STEMI, and the remainder are non–ST-elevation myocardial infarction (NSTEMI).
    - 450,000 patients have a recurrent acute MI each year.
- Age
  - Incidence increases with advancing age.
- Sex
  - More common in men

Risk Factors

- Common
  - Age
    - Men ≥50 years
    - Women ≥60 years
    - Incidence increases with age in both sexes.
  - Cigarette smoking
  - Hypertension
    - Blood pressure ≥140/90 mmHg or using antihypertensive medication
  - Low high-density lipoprotein cholesterol level
    - <40 mg/dL
  - High low-density lipoprotein cholesterol level
    - >130 mg/dL
  - Diabetes mellitus
  - Metabolic syndrome
  - Family history of premature coronary heart disease (CHD)
    - CHD in male first-degree relative <55 years
    - CHD in female first-degree relative <65 years

    - Lifestyle risk factors
        - Obesity (body mass index ≥30 kg/m^2)
        - Physical inactivity
    - Unstable angina
    - Prinzmetal's variant angina
- Less common
    - Hypercoagulability
    - Collagen vascular disease
    - Cocaine abuse
    - Intracardiac thrombi or masses that can produce coronary emboli
- Emerging risk factors
    - Lipoprotein(a)
    - Prothrombotic factors
    - Proinflammatory factors, as reflected in elevated C-reactive protein level
    - Impaired glucose tolerance

Etiology

- STEMI usually occurs when coronary blood flow ceases or decreases abruptly, after thrombotic occlusion of a coronary artery previously affected by atherosclerosis.
    - In most cases, infarction occurs when an atherosclerotic plaque ruptures or fissures, and when conditions (local or systemic) favor thrombogenesis.
        - Mural thrombus forms at the site of rupture and leads to coronary occlusion.
    - Histologic studies indicate that the coronary plaques prone to rupture are those with a rich lipid core and a thin fibrous cap.
- In rare cases, STEMI may be due to coronary artery occlusion caused by:
    - Coronary emboli
    - Congenital abnormalities
    - Coronary spasm
    - Wide variety of systemic—particularly inflammatory—diseases
- The amount of myocardial damage caused by coronary occlusion depends on:
    - The territory supplied by the affected vessel
    - Whether or not the vessel becomes totally occluded
    - The duration of coronary occlusion
    - The quantity of blood supplied by collateral vessels to the affected tissue
    - The demand for oxygen of the myocardium, whose blood supply has been suddenly limited
    - Native factors that can produce early spontaneous lysis of the occlusive thrombus
    - The adequacy of myocardial perfusion in the infarct zone when flow is restored in the occluded epicardial coronary artery
- In up to half of cases, precipitating factors such as the following are implicated in STEMI.
    - Vigorous physical exercise
    - Emotional stress
    - Medical or surgical illness
    - Cocaine abuse (rare)

Symptoms & Signs

Symptoms

- STEMI may commence at any time of the day or night.
    - Circadian variations have been reported, with clusters seen in the morning within a few hours of awakening.
- Chest pain
    - Most common presenting symptom

    - Similar to angina, but more intense and persistent
        - When brought on by exertion, it does not subside with rest.
    - Deep and visceral; commonly described as heavy, squeezing, crushing, and sometimes stabbing or burning
    - Typically involves the central portion of the chest and/or the epigastrium
    - May radiate to:
        - Arms
        - Abdomen
        - Back
        - Lower jaw
        - Neck
    - Not fully relieved by rest or nitroglycerine
    - Not uniformly present in patients with STEMI
        - ~25% of MIs are clinically silent.
        - Proportion of painless STEMIs is greater in patients with diabetes mellitus and increases with age.
- Associated symptoms
    - Weakness
    - Nausea/vomiting
    - Sweating
    - Apprehension, anxiety, sense of impending doom
- Other presentations, with or without pain
    - Sudden-onset breathlessness
        - In elderly patients, may progress to pulmonary edema
    - Sudden loss of consciousness
    - Confusional state
    - Sensation of profound weakness
    - Arrhythmia
    - Evidence of peripheral embolism
    - Unexplained decrease in arterial pressure

Physical examination

- Pallor
- Diaphoresis
- Cool extremities
- Pulse rate and blood pressure
    - Many patients have normal pulse rate and blood pressure within the first hour of STEMI.
    - Patients with large infarctions have hypotension (systolic blood pressure <100 mmHg and/or sinus tachycardia >100/min)
    - Anterior infarction: About one-fourth of patients have manifestations of sympathetic nervous system hyperactivity (tachycardia and/or hypertension).
    - Inferior infarction: Up to half of patients show evidence of parasympathetic hyperactivity (bradycardia and/or hypotension).
- Apical impulse may be difficult to palpate.
- Anterior wall infarction: an abnormal systolic pulsation caused by dyskinetic bulging of infarcted myocardium may develop in the periapical area within the first days of the illness and then may resolve.
- RV infarction: Jugular venous distention is common.
- Signs of ventricular dysfunction
    - Third and fourth heart sounds
    - Decreased intensity of the first heart sound
    - Paradoxical splitting of the second heart sound

- Transient midsystolic or late systolic apical systolic murmur due to dysfunction of the mitral valve apparatus may be present.
- Pericardial friction rub
- Carotid pulse is often decreased in volume, reflecting reduced stroke volume.
- Temperature elevations up to 38 °C may be observed during the first week after STEMI.
- Arterial pressure is variable.
    - In most transmural infarctions, systolic pressure decreases by approximately 10–15 mmHg from the preinfarction state.
- New, loud (≥Gr 3/6) precordial systolic murmur in ruptured ventricular septum and mitral regurgitation

Differential Diagnosis

- Clinical differential diagnosis
    - Acute pericarditis
        - Radiation of discomfort to the trapezius is not seen in patients with STEMI and may be a useful distinguishing feature.
    - Pulmonary embolism
    - Acute aortic dissection
    - Costochondritis
    - GI disorders
        - Gastroesophageal reflux disease
        - Peptic ulcer disease

Diagnostic Approach

- Diagnosis is based on characteristic history, ECG, and serum cardiac markers.
- Early recognition and immediate treatment are essential.

Laboratory Tests

- Serum cardiac biomarkers (see Figure 1)
    - Assess the magnitude of STEMI and should be measured at presentation, 6–9 hours later, and then at 12–24 hours if diagnosis remains uncertain
    - Cardiac-specific troponin T and I
        - Highly specific for myocardial injury
        - Preferred biochemical markers for diagnosis of acute MI
        - Levels of both markers remain elevated for 7–10 days after STEMI.
    - Level of MB isoenzyme of creatine kinase (CK)
        - Increases within 4-8 hours
        - Peaks at 24 hours without reperfusion
        - Returns to normal by 48–72 hours
        - Peaks earlier (about 8 hours) and returns to normal earlier (about 48 hours) after acute reperfusion therapy
        - Considerably more specific than CK; however, cardiac surgery, myocarditis, and electrical cardioversion often result in elevated serum levels of MB isoenzyme
- Ratio of CK-MB mass to CK activity ≥2.5 suggests acute MI.
    - Less useful when levels of total CK are high owing to skeletal muscle injury or when the total CK level is within the normal range but the CK-MB level is elevated
- Myoglobin
    - One of the first serum cardiac markers that increases above the normal range
    - Lacks cardiac specificity
    - Blood levels return to the normal range within 24 hours of the onset of MI.
- Nonspecific reaction to myocardial injury

- Leukocyte count
  - Nonspecific polymorphonuclear leukocytosis appears within a few hours after the onset of pain and persists for 3–7 days.
  - Leukocyte count often reaches 12,000–15,000/μL.
- Erythrocyte sedimentation rate
  - Increases more slowly than leukocyte count, peaking during the first week and sometimes remaining elevated for 1 or 2 weeks

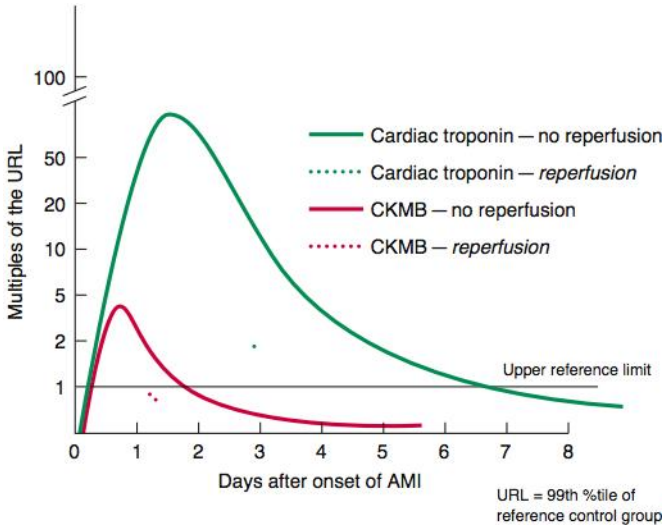

Figure 1: Typical cardiac biomarkers that are used to evaluate patients with STEMI include the MB isoenzyme of CK (CKMB) and cardiac-specific troponins. The white horizontal line depicts the upper reference limit (URL) for the cardiac biomarker in the clinical chemistry laboratory. The kinetics of release of CKMB and cardiac tropon in in patients who do not undergo reperfusion are shown in the solid green and red curves as multiples of the URL. When patients with STEMI undergo reperfusion, as depicted in the dashed green and red curves, the cardiac biomarkers are detected sooner, rise to a higher peak value, but decline more rapidly, resulting in a smaller area under the curve and limitation of infarct size. (*Adapted from JS Alpert et al: J Am Coll Cardiol 36:959, 2000, and AH Wu et al: Clin Chem 45:1104, 1999*.)

Imaging

- 2-dimensional echocardiography
  - Abnormalities of wall motion are almost universally present.
  - Echocardiography cannot distinguish acute STEMI from an old myocardial scar or from acute severe ischemia, but ease and safety make it useful as a screening tool to aid in management decisions.
  - Estimation of left ventricular (LV) function is useful prognostically.
  - May identify the presence of right ventricular (RV) infarction, ventricular aneurysm, pericardial effusion, and LV thrombus
- Doppler echocardiography
  - Useful in detection and quantitation of a ventricular septal defect and mitral regurgitation
- Myocardial perfusion imaging (^{201}thallium or ^{99m}technetium sestamibi)
  - Sensitive for regions of decreased perfusion, but not specific for acute MI
- Cardiac magnetic resonance imaging
  - Demonstrates hyperenhancement with myocardial necrosis

Diagnostic Procedures

- ECG (see Figure 2)
  - Typical STEMI: Q-wave MI develops
    - Initial ST elevation
    - Followed by T-wave inversion
    - Followed by Q-wave development over several hours
  - STEMI leading to non–Q-wave MI
    - Small proportion of STEMI patients
    - Initial ST elevation
    - No development of Q waves
  - If no ST-segment elevation is seen
    - Initially considered to be experiencing either unstable angina or NSTEMI
      - NSTEMI is diagnosed when cardiac biomarkers are elevated.
    - A minority of patients may develop a Q-wave MI.

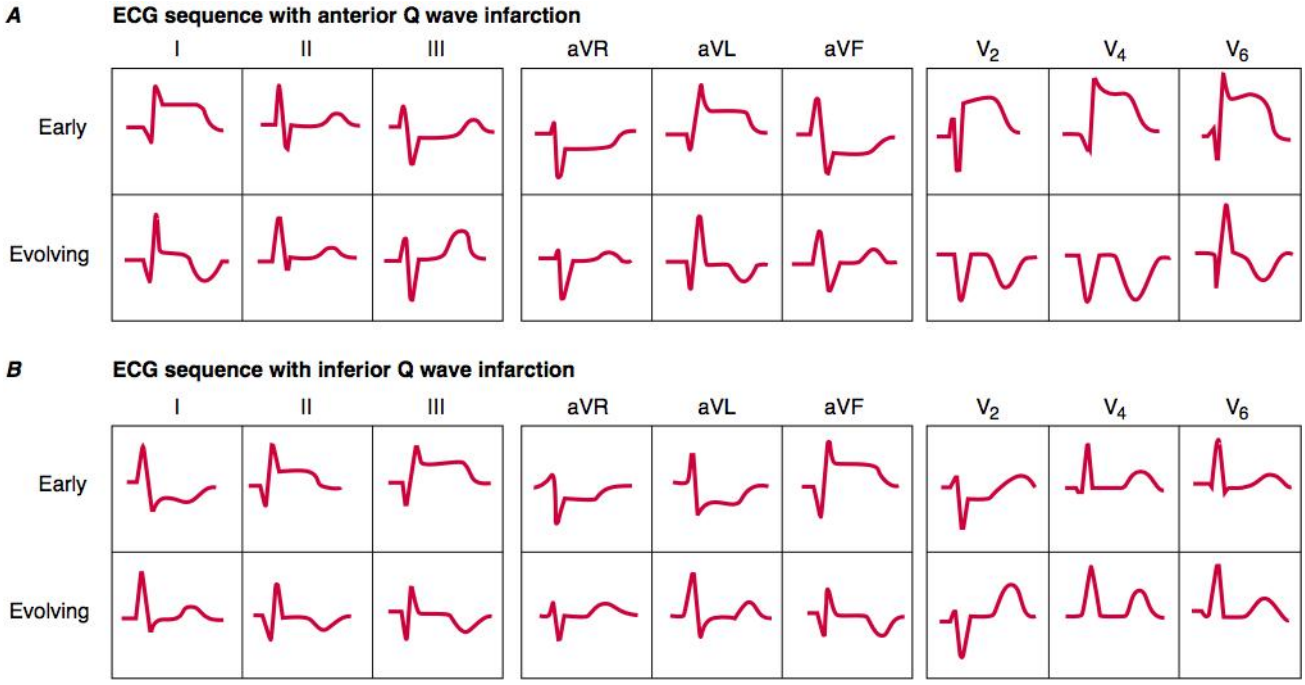

Figure 2: Sequence of depolarization and repolarization changes with (*A*) acute anterior and (*B*) acute inferior wall Q-wave infarctions. With anterior infarcts, ST elevation in leads I, aVL, and the precordial leads may be accompanied by reciprocal ST depressions in leads II, III, and aVF. Conversely, acute inferior (or posterior) infarcts may be associated with reciprocal ST depressions in leads V_1 to V_3. (*After Goldberger, 1999*.)

Treatment Approach

- Initial goals of therapy are to:
  - Quickly identify via 12-lead ECG whether patient has ST elevation and therefore is a candidate for immediate reperfusion therapy
  - Relieve pain
  - Prevent/treat arrhythmias and mechanical complications
- Prehospital care
  - Recognition of symptoms by the patient and prompt seeking of medical attention

    - Rapid deployment of an emergency medical team capable of performing resuscitative maneuvers, including defibrillation
    - Expeditious transportation of the patient to an appropriately staffed hospital facility
- Management in the emergency department
    - Aspirin
    - Supplemental oxygen
    - Control of pain
        - Nitroglycerin
        - Morphine
        - Beta blockers
    - Expeditious implementation of reperfusion therapy
        - Transfer to catheterization laboratory for primary percutaneous coronary intervention (PCI) **or**
        - Immediate fibrinolysis in the emergency department

Specific Treatments

Initial therapy

Aspirin

- Administer aspirin immediately, unless the patient is aspirin intolerant.
- Dosage: 81-162 mg chewed at presentation, then 162–325 mg PO qd

Reperfusion: general considerations

- PCI or intravenous fibrinolytic agent
- Reduces infarct size, LV dysfunction, and mortality
- Identify candidates for reperfusion.
    - First, use 12-lead ECG to identify STEMI.
    - ST-segment elevation of ≥2 mm in 2 contiguous precordial leads and 1 mm in 2 limb leads
    - In the absence of ST-segment elevation, fibrinolysis is not helpful and may be harmful.

Primary PCI (angioplasty or stenting)

- Effective in restoring perfusion in STEMI when carried out on an emergency basis in the first few hours of STEMI
- More effective than fibrinolysis in opening occluded coronary arteries
- Better short-term and long-term clinical outcomes compared to fibrinolysis when performed:
    - By experienced operators (≥75 PCI cases per year)
    - In dedicated medical centers (≥36 primary PCI cases per year)
- Compared with fibrinolysis, primary PCI is generally preferred when:
    - Diagnosis is in doubt
    - Cardiogenic shock is present
    - Bleeding risk is increased
    - Symptoms have been present for >2–3 hours (when the clot is more mature and less easily lysed by fibrinolytic drugs)
- Glycoprotein inhibitors and clopidogrel appear useful for preventing thrombotic complications in patients undergoing PCI.

Fibrinolysis

- If PCI is not available or if logistics would delay PCI > 1 hours longer, fibrinolysis could be initiated.
- Preferable if symptoms to needle time <2–3 hours, but can be useful up to 12 hours if chest pain is persistent or ST remains elevated in leads that have not developed new Q waves
- Door-to-needle time should be <30 minutes for maximum benefit.
- Absolute contraindications
    - History of cerebrovascular hemorrhage at any time
    - Nonhemorrhagic stroke or other cerebrovascular event within the past year
    - Marked hypertension (a reliably determined systolic arterial pressure > 180 mmHg and/or diastolic pressure >110 mmHg) at any time during the acute presentation
    - Suspicion of aortic dissection
    - Active internal bleeding (excluding menses)
- Relative contraindications
    - Current use of anticoagulants (international normalized ratio ≥ 2)
    - Recent (<2 weeks) invasive or surgical procedure or prolonged (>10 minutes) cardiopulmonary resuscitation
    - Known bleeding diathesis
    - Pregnancy
    - Hemorrhagic ophthalmic condition (e.g., hemorrhagic diabetic retinopathy)
    - Active peptic ulcer disease
    - History of severe hypertension that is currently adequately controlled
- Complications
    - Hemorrhage/bleeding
        - Hemorrhagic stroke in 0.5–0.9% of patients; rate increases with advancing age
    - Reperfusion arrhythmias
    - Streptokinase
        - Allergic reactions (2% of patients)
        - Minor degree of hypotension in 4–10% of patients
- Agents of reperfusion approved by the U.S. Food and Drug Administration
    - Direct plasminogen activators
        - Tenecteplase
            - Single weight-based intravenous bolus of 0.53 mg/kg over 10 seconds
        - Reteplase
            - Double-bolus regimen consisting of a 10-million U bolus given over 2–3 min, followed by a second 10-million U bolus 30 minutes later
        - Tissue plasminogen activator
            - 15-mg bolus
            - Followed by 50 mg IV over the first 30 minutes
            - Followed by 35 mg over the next 60 minutes
    - Indirect plasminogen activators
        - Streptokinase
            - 1.5 million U IV over 1 hour
- Clopidogrel: A 300-mg loading dose followed by a 75-mg/d maintenance dosage is useful for fibrinolysis-enhanced patency.
- If chest pain or ST elevation persists >90 minutes after fibrinolysis:
    - Consider referral for rescue PCI.
- Later coronary angiography after fibrinolysis generally reserved for patients with recurrent angina or positive stress test

Antiplatelet and antithrombotic drugs

- Unfractionated heparin
  - 60 U/kg (maximum, 4,000 U), then 12 (U/kg) per hour (maximum, 1,000 U/h)
  - Maintain activated partial thromboplastin time at 1.5–2.0 times control values (~50–70 seconds).
  - Should be initiated with fibrinolytic agents other than streptokinase
  - Elective use with streptokinase

Additional standard treatment (with / without reperfusion therapy)

- Hospitalize in the critical care unit with continuous ECG monitoring.
- Intravenous line for emergency arrhythmia treatment
- Oxygen
  - 2–4 L/min by nasal cannula (maintain oxygen saturation > 90%)
- Activity
  - Bed rest for first 12 hours
  - In the absence of complications
    - Ambulating in room by second to third day
    - By day 3: increase ambulation progressively to a goal of 185 m (600 ft) at least 3 times daily
- Pain control
  - Morphine sulfate
    - 2–4 mg IV every 5–10 minutes until pain is relieved or side effects develop
    - Side effects
      - Nausea
      - Vomiting
      - Respiratory depression (treat with naloxone, 0.4–1.2 mg IV)
      - Hypotension (if bradycardic, treat with atropine, 0.5 mg IV; otherwise, use careful volume infusion)
  - Nitroglycerin
    - Sublingual: 0.3–0.4 mg every 5 minutes, up to 3 doses
      - If systolic blood pressure > 100 mmHg
    - Intravenous: Begin at 10 μg/min and titrate upward to a maximum of 100 μg/min, monitoring blood pressure closely.
    - Avoid when there is clinical suspicion of RV infarction.
    - Do not administer nitrates within 24 hours of sildenafil use or within 48 hours of tadalafil use (used for erectile dysfunction)
  - β-adrenergic antagonists
    - Reduce myocardial oxygen consumption, limit infarct size, and reduce mortality
    - Especially useful in patients with hypertension, tachycardia, or persistent ischemic pain
    - Dosage
      - Intravenous: metoprolol, 5 mg every 2–10 minutes to a total dose of 15 mg
      - Followed by oral regimen: metoprolol, 50 mg every 6 hours for 48 hours, followed by 100 mg every 12 hours
    - Contraindications: active congestive heart failure (CHF), systolic blood pressure <95 mmHg, heart rate <50 beats/min, atrioventricular (AV) block, or history of bronchospasm
- Mild sedation
  - Diazepam: 5 mg PO tid to qid
  - Oxazepam: 15–30 mg tid to qid
  - Lorazepam: 0.5–2 mg tid to qid

- Diet
  - Nothing by mouth or clear liquids for first 4–12 hours
  - Soft diet
- Stool softeners
  - Docusate sodium, 100–200 mg/d
- Strict control of blood glucose in diabetic patients
- Anticoagulation/antiplatelet agents
  - Continue aspirin, 75–162 mg/d.
  - Clopidogrel: 300-mg loading dose followed by 75 mg/d in aspirin-intolerant patients
  - Continue clopidogrel maintenance for at least 6 months in patients who have undergone PCI with drug-eluting stents and at least 1 month in patients with bare metal stents.
  - For deep venous thrombosis prevention in the absence of fibrinolytic therapy, administer:
    - Aspirin, 75-162 mg qd, **and**
    - Low-dose heparin, 5,000 U SC every 12 hours
  - Patients with anterior location of the infarction, severe LV dysfunction, CHF, a history of embolism, 2-dimensional echocardiographic evidence of mural thrombus, or atrial fibrillation
    - Full-dose IV heparin (partial thromboplastin time 1.5–2 times control values) **or**
    - Low-molecular-weight heparin (e.g., enoxaparin, 1 mg/kg SC every 12 hours)
    - Followed by 3–6 months of warfarin therapy with INR = 2-3 x normal.
- Angiotensin-converting enzyme (ACE) inhibitors
  - Reduce the mortality rate after STEMI, and the mortality benefits are additive to those achieved with aspirin and beta blockers
  - Captopril: 6.25 mg PO test dose gradually advanced to 50 mg PO tid
  - Should be prescribed within 24 hours of hospitalization for patients with STEMI, to reduce mortality
  - Continue indefinitely after discharge in patients with CHF or those with asymptomatic LV dysfunction (ejection fraction ≤40%)
  - If the patient is ACE inhibitor intolerant, use angiotensin receptor blocker (e.g., valsartan or candesartan).
- Serum magnesium level repleted if necessary to reduce risk of arrhythmias
- Calcium antagonists are not recommended.

Monitoring

- Continuous ECG monitoring
- Continuous hemodynamic monitoring in selected patients
- Uncomplicated STEMI
  - Usual duration of hospitalization is 4–5 days.
  - On return home from hospital
    - First 1–2 weeks
      - Increase activity indoors and outdoors.
    - After 2 weeks
      - Coordinate level of activity with patient on the basis of exercise tolerance.
      - May resume normal sexual activity
    - Most patients can return to work within 2–4 weeks.

Complications

CHF

- Mortality correlates with severity of pump failure.
- Often classified by Killip class
  - Class I: no signs of pulmonary or venous congestion
  - Class II: moderate heart failure, as evidenced by rales at the lung bases, S3 gallop, tachypnea, or signs of failure of the right side of the heart, including venous and hepatic congestion
  - Class III: severe heart failure, pulmonary edema
  - Class IV: shock with systolic pressure < 90 mmHg and evidence of peripheral vasoconstriction, peripheral cyanosis, mental confusion, and oliguria
- Primary therapy
  - Nitrates, in the absence of hypotension
  - Loop diuretics
  - ACE inhibitors
  - Benefits of digitalis are unimpressive.
- See Congestive Heart Failure.

Cardiogenic shock

- Incidence about 7%
- Patients usually have severe multivessel coronary artery disease.
- Only 10% of patients with this condition present with it on admission.
- 90% develop it during hospitalization.
- Mortality rate is approximately 60%.
- Immediate revascularization is treatment of choice.

Arrhythmias

- Ventricular tachycardia
- Ventricular fibrillation
- Accelerated idioventricular rhythm
- Supraventricular arrhythmias, including atrial fibrillation
- Bradyarrhythmias and AV block

Pericarditis

- Frequently encountered in patients with transmural STEMI
- Usually managed with aspirin, 650 mg qid, or another NSAID
- Anticoagulants can potentially cause tamponade in the presence of acute pericarditis.
  - Therefore, they should not be used unless there is a compelling indication.
- See Pericarditis.

Thromboembolism

- Complicates STEMI in 10% of cases
- Occurs in association with large infarcts
  - Arterial emboli originate from LV mural thrombi.
  - If identified on echocardiography, patient should receive systemic anticoagulation for 3–6 months

- Most pulmonary emboli arise in the leg veins.
  - Prophylaxis should be given.
- See Thromboembolism.

Ventricular aneurysm

- Complications occur weeks to months after STEMI.
  - Include CHF, arterial embolism, and ventricular arrhythmias

Recurrent angina

- Develops in first 2 weeks in ~25% of patients hospitalized for STEMI
- Often heralds extension of the original infarct or reinfarction in a new myocardial zone
- Associated with a doubling of risk after STEMI
- Patients should be referred for prompt coronary arteriography and mechanical revascularization.

Mitral regurgitation

- Presents with new, loud holosystolic murmur, hypotension, and heart failure
- Occurs most commonly on first day
- Diagnosis confirmed by echo Doppler
- Rapid stabilization with intra-aortic balloon counterpulsation followed by surgery

Ruptured ventricular septum

- Like mitral regurgitation, presents with new, loud holosystolic murmur, hypotension, and heart failure
- Diagnostic approach and treatment similar to that of mitral regurgitation

Prognosis

- Natural history of MI
  - Temporal stages
    - Acute (first few hours to 7 days)
    - Healing (7–28 days)
    - Healed (≥29 days)
- The prognosis in STEMI is largely related to the occurrence of complications.
  - Electrical complications (arrhythmias)
  - Mechanical complications ("pump failure")
- 30-day mortality rate from acute MI is ~20%.
  - >50% of deaths occur before the patient reaches the hospital.
  - Most out-of-hospital deaths are due to sudden development of ventricular fibrillation.
    - Most deaths due to ventricular fibrillation occur within the first 24 hours of the onset of symptoms, and, of these, over half occur in the first hour.
- Mortality rate after admission for acute MI has decreased by ~30% over the past 2 decades.
  - Approximately 1 of every 25 patients who survives initial hospitalization dies in the first year after acute MI.
  - Survival is markedly reduced in elderly patients (>75 years).
- Factors associated with increased cardiovascular risk after recovery from STEMI
  - Persistent ischemia (spontaneous or provoked)
  - Depressed LV ejection fraction (<40%)
  - Rales above the lung bases on physical examination or congestion on chest radiography

- Symptomatic ventricular arrhythmias
- History of previous MI
- Age >75 years
- Diabetes mellitus
- Prolonged sinus tachycardia
- Hypotension
- ST-segment changes at rest without angina ("silent ischemia")
- Abnormal signal-averaged ECG
- Nonpatency of the infarct-related coronary artery on angiography
- Persistent advanced heart block or new intraventricular conduction abnormality on ECG

Prevention

Postinfarction prevention

- Strategies to reduce risk
  - Submaximal exercise stress testing before hospital discharge
    - To detect residual ischemia and ventricular ectopy
    - To provide exercise guideline in early recovery period
      - Builds patient confidence
  - Evaluation of LV function
    - To identify patients to receive ACE inhibitors
  - Patient education before discharge ("teachable moment")
- Cardiac catheterization with coronary angiography and/or invasive electrophysiologic evaluation is advised for patients at high risk for recurrent MI or death from arrhythmia.
  - Angina induced at relatively low workload
  - Large reversible defect on perfusion imaging or a depressed ejection fraction
  - Demonstrable ischemia
  - Symptomatic ventricular arrhythmia provoked by exercise

Secondary prevention

- Antiplatelet agents
  - Long-term treatment with an antiplatelet agent (usually aspirin) after STEMI is associated with a 25% reduction in death.
  - In patients taking aspirin long term, STEMIs tend to be smaller and are more likely to be non–Q wave in nature.
  - Clopidogrel (75 mg/d PO) may be used in patients intolerant of aspirin and should be used with aspirin for at least 1 month in patients with bare metal stents and for at least 6 months in patients with drug-eluting stents.
  - For details see Antiplatelet Therapy
- ACE inhibitors
  - Should be used indefinitely by patients with clinically evident heart failure, moderate decrease in global ejection fraction, or a large regional wall motion abnormality
- β-blockers
  - Long-term routine use of oral β-blockers for at least 2 years after STEMI reduces mortality, sudden death, and, in some instances, reinfarction.
- Warfarin may be added for patients at increased risk of embolism.
  - In patients <75 years, low-dose aspirin (75–81 mg/d) plus warfarin administered to achieve an international normalized ratio >2.0 is more effective than aspirin alone for preventing recurrent MI and embolic cerebrovascular accident.
  - Complication of combination therapy: increased risk of bleeding
- Risk factors for atherosclerosis should be favorably modified.
  - Smoking cessation

    - Control of hypertension and hyperlipidemia (reduce low-density lipoprotein cholesterol level <100 mg/dL)
    - Regular physical exercise
    - Reduction of emotional stress
- Hormone replacement therapy
    - Should not be given de novo to postmenopausal women after STEMI
    - Postmenopausal women already taking estrogen plus progestin at the time of STEMI may continue that therapy.

ICD-9-CM

- 410.__ Acute myocardial infarction, (anatomic location of myocardial infarction specified by fourth digit; episode of care specified by fifth digit)
- 410.90 Acute myocardial infarction, Unspecified site, episode of care unspecified

See Also

- Antiplatelet Therapy
- Chronic Stable Angina
- Fibrinolytic Therapy
- Unstable Angina and Non–ST-Elevation Myocardial Infarction

Internet Sites

- Professionals
    - Clinical Statements / Guidelines
      American College of Cardiology
- Patients
    - Heart attack
      MedlinePlus

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PEARLS

- After coronary occlusion, a "wavefront" of myocardial necrosis begins in the subendocardium and advances to the epicardium, as described by Reimer and Jennings.
- In STEMI, infarct size can be limited and prognosis improved by early reperfusion: "Time is muscle."
- A history of chest pain and sweating suggests presence of a large STEMI.