Some notes on Myocardial Infarction

Myocardial infarction (MI) is a coagulative type of necrosis of cardiac muscle and is due to prolonged severe ischemia.

Risk Factors

- Race: Any race can be affected; whites and blacks are affected equally. Indians are also having high-risk of IHD.
- Age: Its frequency rises progressively with age and peak is between 40 to 65 years of age. It can develop at younger age in patients with major risk factors of atherosclerosis (hyperlipidemia, hypertension, diabetes and cigarette smoking).
- Sex: Males have significantly higher risk than females mainly during the reproductive period. However, after menopause the risk is similar to that of males. The protective effect may be due to estrogen.
- Other risk factors: Risk factors for atherosclerosis

A. Modifiable major risk factors

- Hyperlipidemia
- Hypertension
- Cigarette smoking
- Diabetes mellitus

B. Nonmodifiable (constitutional) risk factors

- Genetic abnormalities
- Family history
- Increasing age
- Male gender

C. Additional risk factors

- Inflammation
- CRP level
- Hyperhomocystinemia
- Metabolic syndrome
- Lipoprotein (a)
- Raised procoagulant levels
- Inadequate physical activity
- Stressful lifestyle
- Obesity
- Alcohol

Pathogenesis of MI

In coronary arteries with pre-existing (fixed) atherosclerotic occlusion, inadequate coronary perfusion may occur due to a new superimposed thrombosis and/or coronary vasospasm. *Thrombus formation is due to acute plaque change.*

Sequence of events in a typical case of MI is as follows:

1. Acute plaque change: It is a sudden change/event occurring in an atheromatous plaque where the initial is sudden change in the atheromatous plaques and convert partially occlusive atherosclerotic plaque **to produce sudden ischemia**. These changes are divided **into three categories**:

1. Rupture, fissuring of plaque → exposes highlythrombogenic plaque constituents→ sudden thrombus formation→ sudden occlusion of lumen.
2. Erosion/ulceration of plaque
   - Exposes highly thrombogenic subendothelial basement membrane
   - Sudden thrombus formation
   - Sudden occlusion of lumen.
3. Hemorrhage into the central core of plaque→ increases the plaque size→ sudden occlusion of lumen.

Factors that trigger acute plaque change:

– **Intrinsic**: Plaque composition and structure (namely vulnerable plaque).

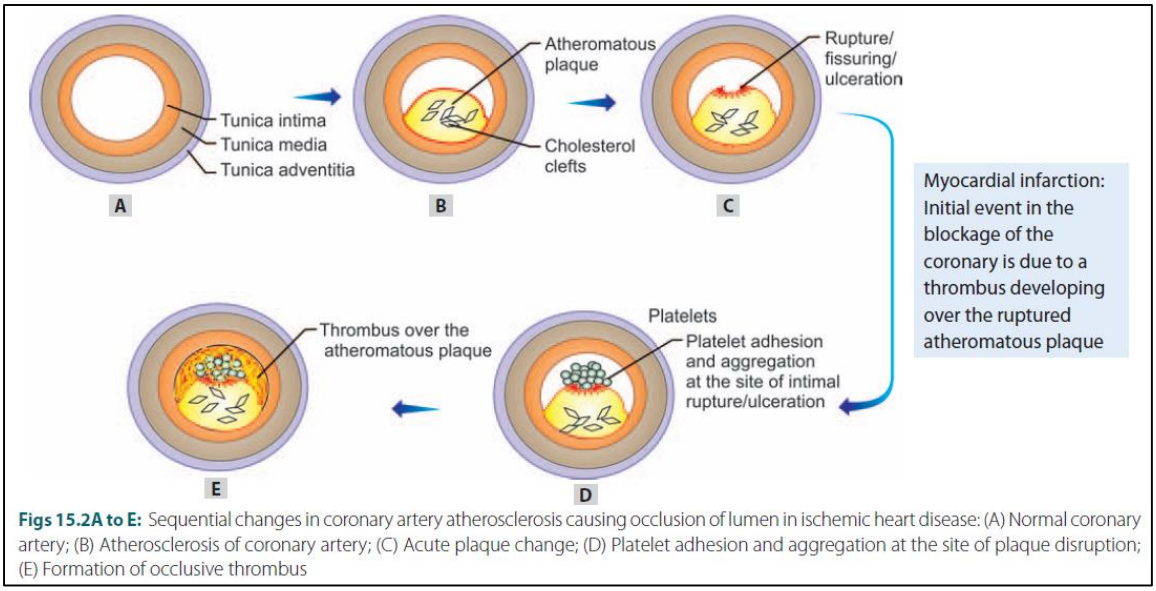
– **Extrinsic**: Blood pressure and platelet reactivity which induces total thrombotic occlusion of already narrowed coronary artery by atheromatous plaque.

2. Formation of microthrombi: Acute plaque changes exposes thrombogenic subendothelial collagen→ platelets adhere to the site→ platelet activation and aggregation→ formation of microthrombi on the atheromatous plaque→ partial or complete occlusion
of the affected coronary artery.

3. Vasospasm: Activated platelets, endothelial cell and inflammatory cells release mediators→ cause vasospasm at the sites of atheroma→ further narrowing of the lumen.

4. Activation of the coagulation pathway: Tissue factor released at the site of acute plaque change→ activates coagulation system→ increase the size of the thrombus.

5. Complete occlusion of vessel: Within minutes, the thrombus may completely occlude the lumen of the vessel.
6. Myocardial necrosis: Complete occlusion→ results in ischemic coagulative necrosis of the area supplied by the particular coronary artery. The anatomic area supplied by that artery is called as the area at risk.



Figs 15.2A to E: Sequential changes in coronary artery atherosclerosis causing occlusion of lumen in ischemic heart disease: (A) Normal coronary artery; (B) Atherosclerosis of coronary artery; (C) Acute plaque change; (D) Platelet adhesion and aggregation at the site of plaque disruption; (E) Formation of occlusive thrombus

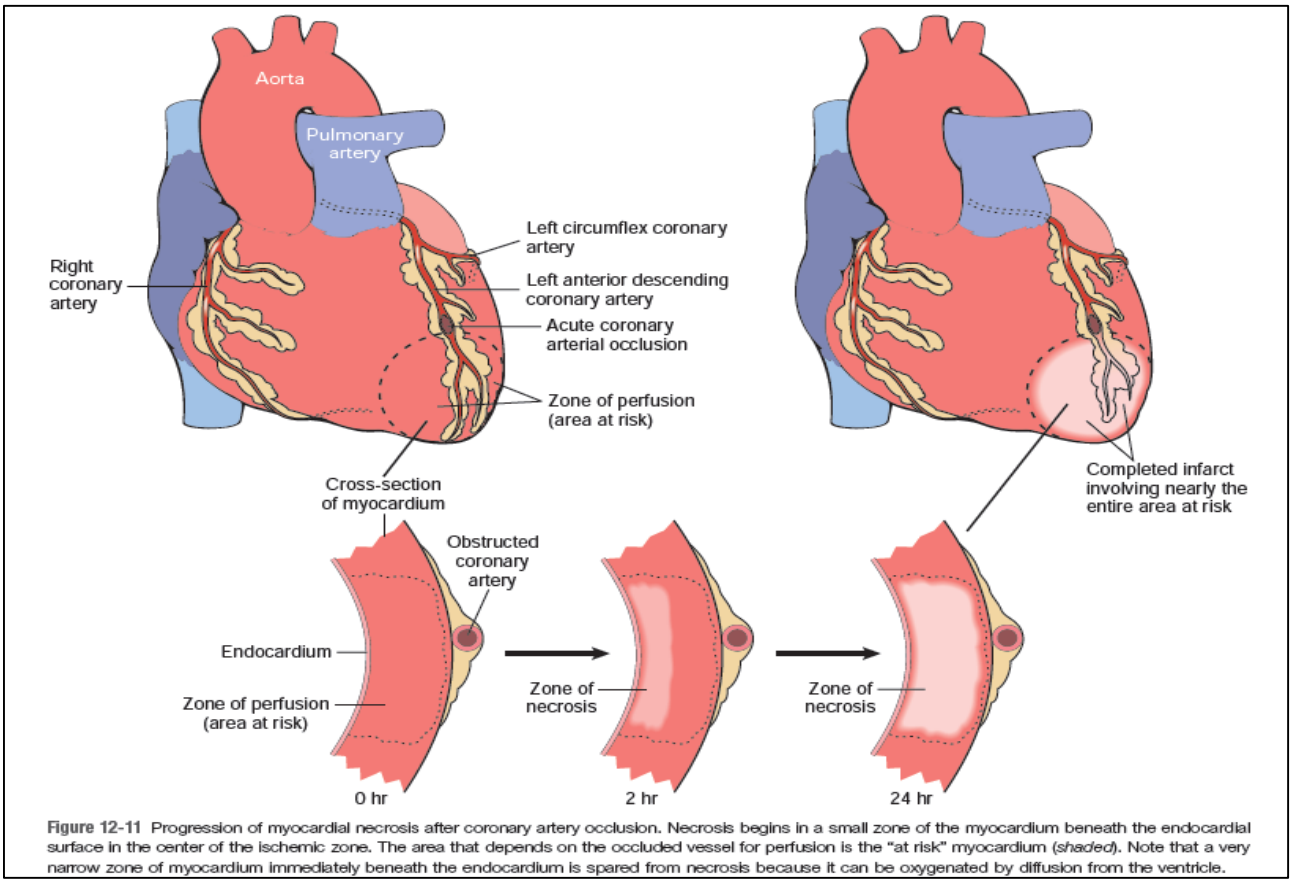


Figure 12-11 Progression of myocardial necrosis after coronary artery occlusion. Necrosis begins in a small zone of the myocardium beneath the endocardial surface in the center of the ischemic zone. The area that depends on the occluded vessel for perfusion is the "at risk" myocardium (*shaded*). Note that a very narrow zone of myocardium immediately beneath the endocardium is spared from necrosis because it can be oxygenated by diffusion from the ventricle.

Consequence of Myocardial Ischemia

These include functional, biochemical and morphological changes. Morphological changes can be divided into reversible and irreversible damage/injury.

A. **Reversible injury:** These changes are potentially reversible and include:

1. Biochemical changes: Cessation of aerobic glycolysis occurs within seconds of myocardial ischemia→decreased production of ATP (adenosine triphosphate)→accumulation of potentially toxic metabolites (such as lactic acid).
2. Functional disturbances: Loss of contractility within 60 seconds→can precipitate acute heart failure.
3. Morphological changes: They are seen at ultrastructural level such as mitochondrial swelling, glycogen depletion and myofibrillar relaxation. They also develop within a few minutes.

B. Irreversible injury: It develops only after prolonged, severe myocardial ischemia of more than 20–40 minutes.

1. Biochemical changes: They cause leakage of cytoplasmic proteins into the blood. In the early phases of myocardial cell necrosis, there is breakdown of the sarcolemmal membrane→leakage of intracellular proteins (such as myoglobin, LDH, CK, and troponins I and T) into the blood. The levels of these leaked myocardial proteins in the blood is used for the diagnosis as well as management of MI.
2. Functional disturbances: Arrhythmias.
3. Morphological changes: Coagulative necrosis of cardiac muscle fibers usually complete within 6 hoursof the onset of myocardial ischemia.

Zones damaged: First necrosis in the subendocardial zone→ later transmural infarct.

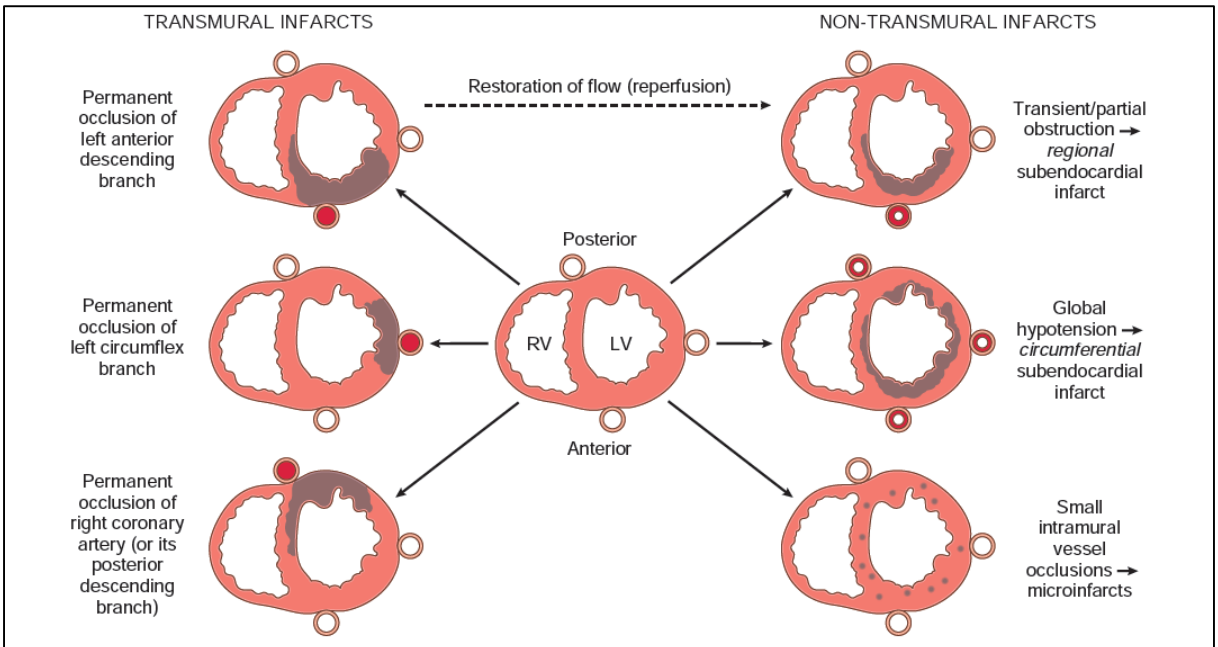


Figure 12-12 Distribution of myocardial ischemic necrosis correlates with the location and nature of decreased perfusion. *Left,* The positions of transmural acute infarcts resulting from occlusions of the major coronary arteries; *top to bottom,* left anterior descending, left circumflex, and right coronary arteries. *Right,* The types of infarcts that result from a partial or transient occlusion, global hypotension, or intramural small vessel occlusions.

Classification of Myocardial Infarct

A. ***Depending on the thickness of myocardium involved****:*

1. **Transmural infarct**:

– Ischemic necrosis of the entire thickness of the ventricular wall. However, a narrow rim (approximately 0.1 mm) of subendocardial myocardium is preserved due to diffusion of oxygen and nutrients from the ventricular lumen.

– Most myocardial infarcts are transmural.

– Usually associated with chronic coronary atherosclerosis, acute plaque change, and superimposed thrombosis.

2. **Subendocardial (nontransmural) infarct**:
– Ischemic necrosis of inner one-third to one-half of the ventricular wall.
– Occurs due to plaque disruption followed by a coronary thrombus, which undergoes lysis or prolonged, severe reduction in systemic blood pressure. For example, shock superimposed on chronic, coronary stenosis.
3. **Multifocal microinfarcts**: Develop with occlusion of small vessel (e.g. vasculitis, embolization) and may not show any changes in ECG.

B. ***Depending on the age of the infarct: Recent (fresh) or old (healed).***

C. ***Depending on the anatomic region involved: Anterior, posterior, lateral, septal and their combination like posterolateral.***

D. ***Depending on the electrocardiographic changes***:

a. ST elevation myocardial infarct (STEMI) found in **transmural infarct**,

b. Non–ST elevation infarct (NSTEMI) found in **subendocardial infarc**t and

c. Electrocardiographically silent with nonspecific changes in **microinfarctions** (depending on the extent and location of the vascular involvement).

TABLE 15.1: Differences between transmural and subendocardial infarct

Characteristics	*Transmural infarcts*	*Subendocardial infarcts*
Nature of lesion	Unifocal and solid	Multifocal and patchy
Distribution	Specific coronary artery	Circumferential
Thrombus in the coronary artery	Common	Rare
Shock	Often causes shock	Often result from hypotension or shock
Pericarditis	Common	Absent
Cardiac aneurysm	May develop	Does not develop.
ECG changes	Elevation of ST segment → ST elevation infarcts (STEMIs)	Non-ST elevation infarcts

Reperfusion injury

In some occasions, restoration of blood flow to the damaged myocardium triggers further ischemic cellular damage, this paradoxical effect is known as reperfusion injury. This process involves a complex interaction between oxygen free radicals and intracellular calcium, leading to acceleration of myocardial damage and death, microvascular dysfunction and fatal arrhythmias. The role of nitric oxide (an endothelium-derived relaxing factor) as a cardioprotective agent against reperfusion injury, has been demonstrated, as nitric oxide works to inactivate oxygen free radicals, therefore, ameliorating the process of reperfusion injury. Despite the improved understanding of the process of reperfusion injury, there are no specific therapies to prevent it.

GROSS AND MICROPATHOLOGY

Gross and microscopic changes develop only hours to days after the onset of ischemia.

Duration	Gross changes	Light microscopic changes
0–½ hour	**No identifiable/apparent gross changes** are seen in the first 12 hours. **Triphenyl tetrazolium chloride (a histochemical stain)** can grossly identify infarct within **2–3 hours after onset.** – **Noninfarcted myocardium appears brick-red** (lactate dehydrogenase activity is preserved). – **Infarcted area** remains **unstained pale** (loss of dehydrogenases). – **Old infarcts appear white and glistening**.	Earliest changes can be detected only by electron microscopy – **Reversible injury (0–1½ hours): Relaxation of myofibrils, loss of glycogen, and mitochondrial swelling**. – **Irreversible injury** (½–6 hours): Develops after 30–60 minutes of ischemia. The changes include **mitochondrial amorphous matrix densities.**
½–6 hours		
6–12 hours		Coagulative necrosis begins and shows edema and hemorrhage. Other changes include: – **Wavy fibers**: They represents noncontractile, stretched, buckled dead myofibrils at the periphery of the infarct. – **Vacuolization of myocardial cell (myocytolysis)**
12–24 hours	Appears **pale reddish-blue area** (due to stagnated, trapped blood) and progressively becomes **sharply defined**, **yellow-tan**, **and soft**.	Coagulative necrosis; pyknotic nuclei; increased eosinophilia of cytoplasm; contraction band necrosis at margins; beginning of neutrophilic infiltrate.
1–3 days	Mottled with a **central pale**, **yellowish**, **necrotic region** with well-demarcated border of **hyperemic zone** (due to granulation tissue).	**Acute inflammatory reaction** characterized by accumulation of PMNLs, **at the periphery/borders of infarct**. **Coagulation necrosis, loss of nuclei and striations; increased interstitial infiltration of neutrophils**
3–7 days		**Appearance of macrophages**: *The polymorphonuclear leukocytes are replaced by macrophages.* **Macrophages phagocytose** and remove the necrotic myocardial cells and neutrophil fragments at the border of infarct. Disintegration of dead myocardial cells, with disintegrating neutrophils
7–10 days	Appears **soft and rimmed by a hyperemic zone** of highly vascularized granulation tissue	**Process of healing starts from its margins** toward its center **which is responsible for most advanced healing at the periphery**. – **Well-established highly vascularized granulation tissue,** which consists of proliferating capillaries and fibroblasts. – **Fibroblasts proliferate** and **collagen deposition** proceeds.
10–14 days		
2–8 weeks	Older, healed infarcts appear **firm**, **pale gray and contracted** and evelops into a **fibrous scar**	Increased collagen deposition and decreased cellularity
>2 months		Dense collagenous scar tissue

Pattern of left ventricular infarcts

1. **Left anterior descending (LAD) coronary artery occlusion** (40–50%):

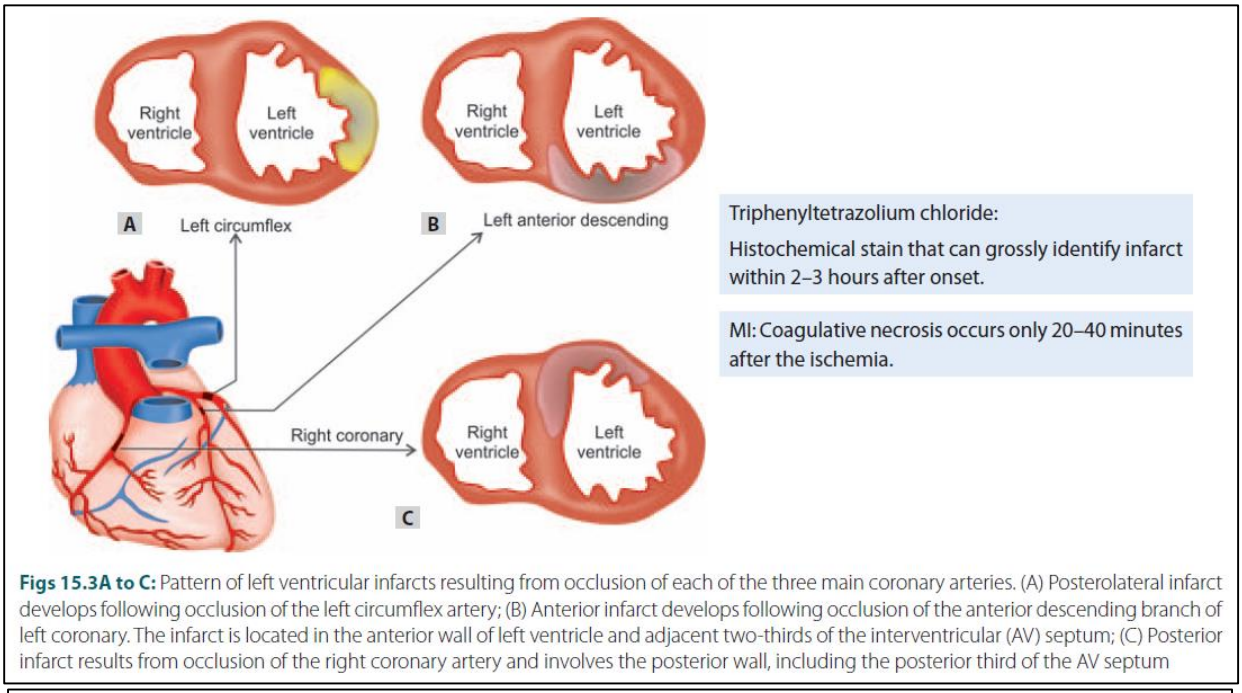
Infarcts involve:
– **Anterior wall of left ventricle near the** apex
– **Anterior portion of ventricular septum**
– Apex circumferentially.

2. **Right coronary artery occlusion (30–40%):**

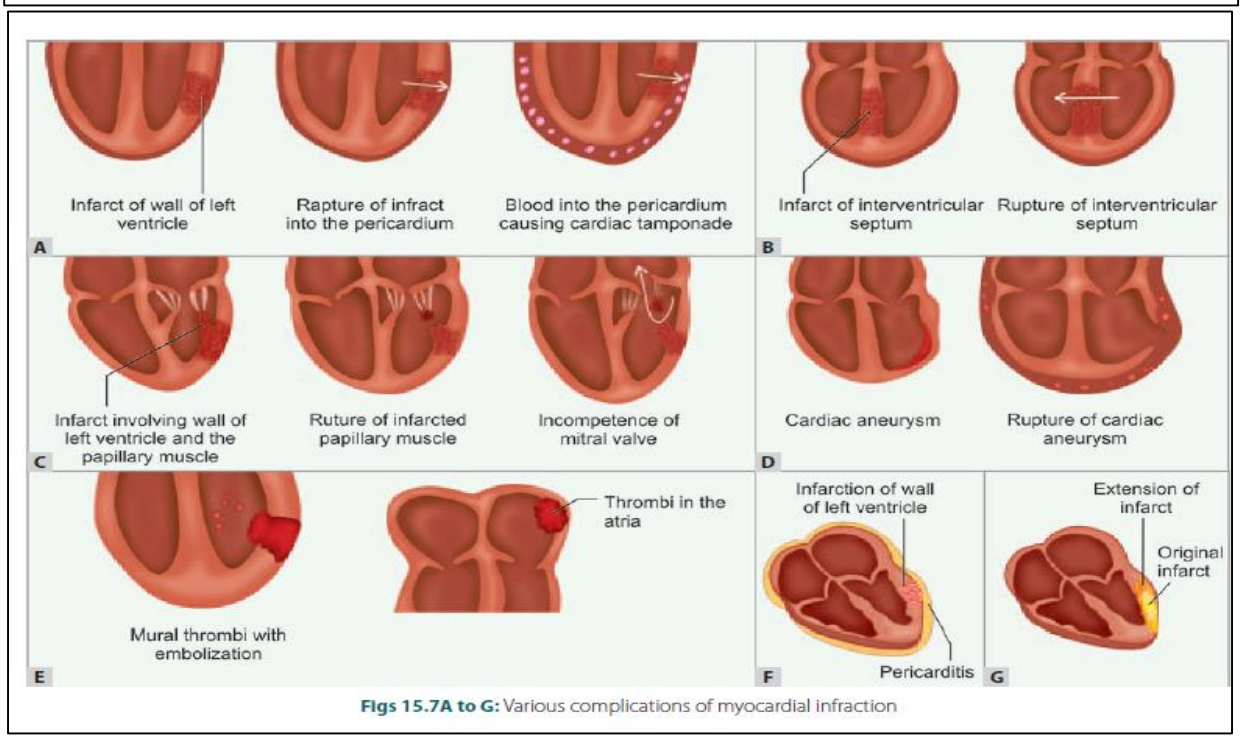
Infarcts involves:
– **Region of the inferior/posterior wall of left ventricle**
– **Posterior portion of interventricular** septum (inferior infarct)
– Inferior/posterior right ventricular free wall (in some).

3. **Left circumflex coronary artery occlusion** (15–20%):

Infarcts involve the **lateral wall of left ventricle** except at the apex.



Figs 15.3A to C: Pattern of left ventricular infarcts resulting from occlusion of each of the three main coronary arteries. (A) Posterolateral infarct develops following occlusion of the left circumflex artery; (B) Anterior infarct develops following occlusion of the anterior descending branch of left coronary. The infarct is located in the anterior wall of left ventricle and adjacent two-thirds of the interventricular (AV) septum; (C) Posterior infarct results from occlusion of the right coronary artery and involves the posterior wall, including the posterior third of the AV septum



Figs 15.7A to G: Various complications of myocardial infraction

Complications of Myocardial Infarction

The risk of complications depends on: (1) infarct size,(2) location, and (3) thickness of myocardium involved(subendocardial or transmural).

1. **Left ventricular failure and cardiogenic shock:** MI produces contractile functional abnormalities of left ventricle and is roughly proportional to the size of infarct. Mild dysfunction → left ventricular failure→pulmonary edema.

Cardiogenic shock: Severe pump failure occurs with a large infarct (>40% of the left ventricle) → cardiogenic shock→fatal.

2. **Arrhythmias**: Almost most MI patients develop abnormal cardiac rhythm→myocardial irritability and/or conduction disturbances→arrhythmias
These include sinus bradycardia, tachycardia, ventricular tachycardia, and ventricular fibrillation. In inferoseptal infarcts→partial or complete heart block.
MI: Heart is maximally soft during 3–7 days; risk of myocardial rupture more during this period.

3. **Myocardial rupture**: Necrosis and neutrophilic infiltration →causes softening and weakening of myocardium→lead to cardiac rupture. Most frequent during 3‑7 days after transmural infarcts.

a. Rupture of the ventricular free wall: It is most common → result in hemopericardium and cardiac tamponade.
b. Rupture of the ventricular septum : It is less common → lead to an acute VSD and left-to- rightshunt.
c. Rupture of papillary muscle: It is least common→ leads to acute severe mitral regurgitation.
d. Posteriomedial papillay muscle rupture:
    1. Due to thrombosis of RCA
    2. Causes mitral regurgitation.

4. Dilatation of ventricular chamber: Area of infarct being weak region may be disproportionately stretched→ dilation of the infarct region (especially with anteroseptal infarcts) not severe enough to cause aneurysm.

5. Ventricular aneurysm: After acute transmural infarction, the affected ventricular wall may bulge outward during systole resulting ventricular aneurysm. It develops as a late complication of large transmural infarcts.

6. Mural thrombus: Infarct → causes local abnormality in myocardial contractility (causing stasis) and endocardial damage (creating a thrombogenic surface)→favor mural thrombosis →left-sided thromboembolism. They may also develop within ventricular aneurysms.
MI: Mural thrombus on the infarcted site is likely source of emboli.

7. Pericarditis:
Early—due to acute inflammation
Late—autoimmune mechanism.

8. Extension of infarct:
New areas of repeated necrosis can occur adjacent to an existing myocardial infarct causing extension of infarct.

In extended infarct, healing is more advanced in the central zone than the periphery of the infarct. This is in contrast with that simple infarct, in which thehealing is more advanced at the periphery.

Cause of extension: It may be due to retrograde propagation of a thrombus or proximal vasospasm, microemboli, or an arrhythmia.

9. Infarction of right ventricle: It is unusual. However, part of right ventricular myocardium may be involved with infarction of adjacent posterior left ventricle and ventricular septum→venous circulation pooling→systemic hypotension.

10. Progressive late heart failure (chronic IHD): Usually develops due to the functional decompensation of hypertrophied noninfarcted myocardium.

Myocardial Infarction Workup

History

The patient's history is critical in diagnosing myocardial infarction (MI) and sometimes may provide the only clues that lead to the diagnosis in the initial phases of the patient presentation.
Patients with typical acute MI usually present with chest pain and may have prodromal symptoms of fatigue, chest discomfort, or malaise in the days preceding the event; alternatively, typical ST-elevation MI (STEMI) may occur suddenly without warning.
The typical chest pain of acute MI usually is intense and unremitting for 30-60 minutes. It is retrosternal and often radiates up to the neck, shoulder, and jaws, and down to the left arm. The chest pain is usually described as a substernal pressure sensation that is also perceived as squeezing, aching, burning, or even sharp. In some patients, the symptom is epigastric, with a feeling of indigestion or of fullness and gas.

Other symptoms of myocardial infarction include the following:

- Anxiety, commonly described as a sense of impending doom
- Pain or discomfort in areas of the body, including the arms, left shoulder, back, neck, jaw, or stomach
- Lightheadedness, with or without syncope
- Cough
- Profuse sweating
- Shortness of breath
- Wheezing

Physical Examination

Physical examination findings for myocardial infarction (MI) can vary; one patient may be comfortable in bed, with normal examination results, whereas another patient may be in severe pain, with significant respiratory distress and a need for ventilatory support.
Patients with ongoing symptoms usually lie quietly in bed and appear pale and diaphoretic.

Vital signs

Heart rate
The patient's heart rate is often increased (tachycardia secondary to sympathoadrenal discharge). The pulse may be irregular because of ventricular ectopy, an accelerated idioventricular rhythm, ventricular tachycardia, atrial fibrillation or flutter, or other supraventricular arrhythmias.

Blood pressure
In general, the patient's blood pressure is initially elevated (hypertension because of peripheral arterial vasoconstriction resulting from an adrenergic response to pain, anxiety, and ventricular dysfunction). However, it is not uncommon to have increased blood pressure as the precipitant of acute MI.
Alternatively, hypotension can also be seen. Usually this indicates either right ventricular MI or severe left ventricular dysfunction due to a large infarct area or impaired global cardiac contractility.

Respiratory rate
The respiratory rate may be increased in response to pulmonary congestion or anxiety.

Temperature
Fever is usually present within 24-48 hours, with the temperature curve generally parallel to the time course of elevations of creatine kinase (CK) levels in the blood. Body temperature may occasionally exceed 102°F.

Neck veins

In patients with acute inferior-wall MI with right ventricular involvement, distention of neck veins is commonly described as a sign of failure of the right ventricle. Impaired right ventricular function also leads to systemic venous hypertension, edema, and hepatomegaly.

Heart

On palpation - lateral displacement of the apical impulse.
On Auscultation, **Paradoxical splitting of S_2 may reflect the presence of left bundle-branch block or prolongation of the pre-ejection period with delayed closure of the aortic valve, despite decreased stroke volume.**

A new mitral regurgitation murmur (typically holosystolic near the apex) indicates papillary muscle dysfunction or rupture, or mitral annular dilatation; it may be audible even when cardiac output is substantially decreased.

A holosystolic murmur that radiates to the midsternal border and not to the back, possibly with a palpable thrill, suggests a ventricular septal rupture; such a rupture may occur as a complication in some patients with full-thickness MIs.
With resistive flow and an enlarged pressure difference, the ventricular septal defect murmur becomes harsher, louder, and higher in pitch than before.
A pericardial friction rub may be audible as a to-and-fro rasping sound; it is produced through sliding contact of inflammation-roughened surfaces.

Chest

Rales or wheezes may be auscultated; these occur secondary to pulmonary venous hypertension, which is associated with extensive acute left ventricular MI. Unilateral or bilateral pleural effusions may produce egophony at the lung bases.

Abdomen

Patients frequently develop tricuspid incompetence; hepatojugular reflux may be elicited even when hepatomegaly is not marked.
A pulsatile abdominal mass may suggest an abdominal aortic aneurysm.

Extremities

Peripheral cyanosis, edema, pallor, diminished pulse volume, delayed rise, and delayed capillary refill may indicate vasoconstriction, diminished cardiac output, and right ventricular dysfunction or failure. Pulse and neck-vein patterns may reveal other associated abnormalities, as previously discussed.

LABORATORY DIAGNOSIS

The objectives of laboratory testing and imaging include the following:

- *To determine the presence or absence of myocardial infarction (MI) for diagnosis and differential diagnosis (point–of-care testing and testing in central laboratory of cardiac troponin levels)*
- *To characterize the locus, nature (ST-elevation MI [STEMI] or non–ST-elevation MI [NSTEMI]), and extent of MI (ie, to estimate infarct size)*
- *To detect recurrent ischemia or MI (extension of MI)*
- *To detect early and late complications of MI*
- *To estimate the patient's prognosis*

Electrocardiography

The electrocardiogram (ECG) is the most important tool in the initial evaluation and triage of patients in whom an acute coronary syndrome (ACS) is suspected.

Different ECG abnormalities

ECG is an effective tool to distinguish between acute MI and the myocardial ischemia that usually precedes it, as not all patients with myocardial ischemia will develop MI.
In STEMI, typical ST-segment elevation persists for hours and is followed by inversion of T waves during the first few days and by the development of Q waves.
High-probability ECG features of MI are the following:

- ST-segment elevation greater than 1 mm in two anatomically contiguous leads
- The presence of new Q waves

Intermediate-probability ECG features of MI are the following:

- ST-segment depression
- T-wave inversion
- Other nonspecific ST-T wave abnormalities

However, normal or nonspecific findings on ECGs do not exclude the possibility of MI.

Localization of the involved myocardium based on distribution of ECG abnormalities in MI is as follows:

- Inferior wall - II, III, aVF
- Lateral wall - I, aVL, V_4 through V_6
- Anteroseptal - V_1 through V_3
- Anterolateral - V_1 through V_6
- Right ventricular - RV_4, RV_5
- Posterior wall - R/S ratio greater than 1 in V_1 and V_2, and T-wave changes in V_1, V_8, and V_9

To summarize, non-ischemic causes of ST-segment elevation include left ventricular hypertrophy, pericarditis, ventricular-paced rhythms, hypothermia, hyperkalemia and other electrolyte imbalances, and left ventricular aneurysm.

Cardiac Biomarkers

In the past, different cardiac biomarkers have been used to evaluate patients with suspected acute myocardial infarction (MI) (acute coronary syndrome [ACS] and ST-elevation MI [STEMI]). The cardiac-specific troponins I and T, creatine kinase (CK), the MB isoenzyme of creatine kinase (CK-MB), and myoglobin have been used as surrogates for myocardial necrosis.

Cardiac troponins: These are proteins involved in heart muscle contraction. **Increased plasma levels establish the diagnosis of myocardial infarction. Cardiac-specific proteins are of two types: Troponins I (TnI) and T (TnT)**. They are **most sensitive and specific markers** of myocardial infarction. Levels begin to **rise at 2–4 hours** and **peaks at 48 hours**. The elevated troponin levels may **remain for 7–10 days** after acute MI.

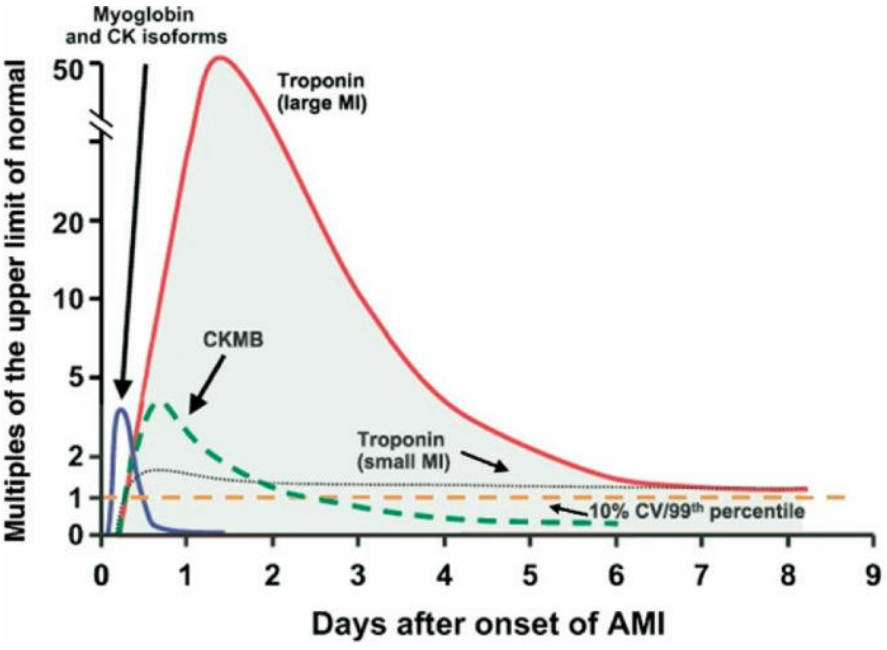
***Cardiac creatine phosphokinase (CK)*:** It is a **nonspecific enzyme marker** and it is present in brain,myocardium, and skeletal muscle. It has two isoformsdesignated "M" and "B". **MB heterodimers chiefly in cardiac** muscle (lesser amounts in skeletal muscle). **MB form of creatine kinase (CK-MB)** is **sensitive but not specific**, because it is also raised with skeletal muscle injury. CK-MB levels **rise within 2–4 hours** of the onset of MI, **peaks at 24 hours**, and returns to **normal within72 hours**.

Lactate dehydrogenase (LDH): It not specific marker.It starts rising after 24–48 hours. It remains for many days and returns to normal in 7–14 days.

Myoglobin: It is an oxygen-carrying respiratory protein found only in skeletal and cardiac muscle. It is an earliest marker of MI, the level rises within 1–3 hours, peaks in about 8–12 hours and return to normal in about 24–36 hours

TABLE 15.3: Laboratory markers of myocardial infarction`

Marker	*Onset*	*Peak*	*Normalization*	*Significance*
Troponins T & I	2–4 hours	48 hours	7–10 days	Most sensitive and specific marker
CK-MB	2–4 hours	24 hours	72 hours	Sensitive but not specific
LDH	24–48 hours	Many days	7–14 days	Not specific marker
Myoglobin	1–3 hours	8–12 hours	24–36 hours	Earliest marker



Other Laboratory Studies

Complete blood cell count

Obtain to rule out anemia as a cause of decreased oxygen supply and prior to giving thrombolytic agents. Leukocytosis is also common, but not universal, in the setting of acute myocardial infarction.
A platelet count is necessary if a IIb/IIIa agent is considered; also, the patient's white blood cell (WBC) count may be modestly elevated in the setting of MI, signifying an acute inflammatory state. The platelet count may become dangerously low after the use of heparin because of heparin-induced thrombocytopenia (HIT).

Chemistry profile (comprehensive metabolic panel)

In the setting of MI, monitoring of potassium and magnesium levels is important. Blood glucose levels are important to measure, as many patients are first diagnosed with diabetes when they present with MI.
The erythrocyte sedimentation rate (ESR) rises above reference range values within 3 days and may remain elevated for weeks.
The serum lactate dehydrogenase (LDH) level rises above the reference range within 24 hours of MI, reaches a peak within 3-6 days, and returns to the baseline within 8-12 days.
Blood oxygenation should be checked and repeatedly corrected if any clinical findings suggest hypoxemia. Fingertip oximetry may be adequate.

Lipid profile
A lipid profile may be helpful if obtained upon presentation. However, regardless of the lipid profile results, initiation of a high-intensity statin is recommended in all patients with acute coronary syndrome.

Cardiac Imaging

Coronary angiography can be performed; this procedure can be used to definitively diagnose or rule out CAD. Based on the angiographic result and patient comorbidities, subsequent treatment recommendations can be made, which may include medical therapy, percutaneous coronary intervention (PCI), or coronary artery bypass graft (CABG) surgery.
Echocardiography is highly recommended and is required to evaluate ventricular function and wall-motion abnormalities. It is also used to identify pericardial effusion, ischemic mitral regurgitation, and cardiac tamponade that may complicate acute MI.

Myocardial Infarction Treatment & Management

The first goal for healthcare professionals in management of acute myocardial infarction (MI) is to diagnose the condition in a very rapid manner.

As a general rule, initial therapy for acute MI is directed toward restoration of perfusion as soon as possible to salvage as much of the jeopardized myocardium as possible. This may be accomplished through medical or mechanical means, such as percutaneous coronary intervention (PCI), or coronary artery bypass graft (CABG) surgery.

Although the initial treatment of the different types of acute coronary syndrome (ACS) may appear to be similar, it is very important to distinguish whether the patient is having an ST-elevation MI (STEMI) or a non–STEMI (NSTEMI), because definitive therapies differ between these two types of MI. Particular considerations and differences involve the urgency of therapy and the degree of evidence regarding different pharmacologic options.

NON ST ELEVATED MI – ACUTE CORONARY SYNDROME

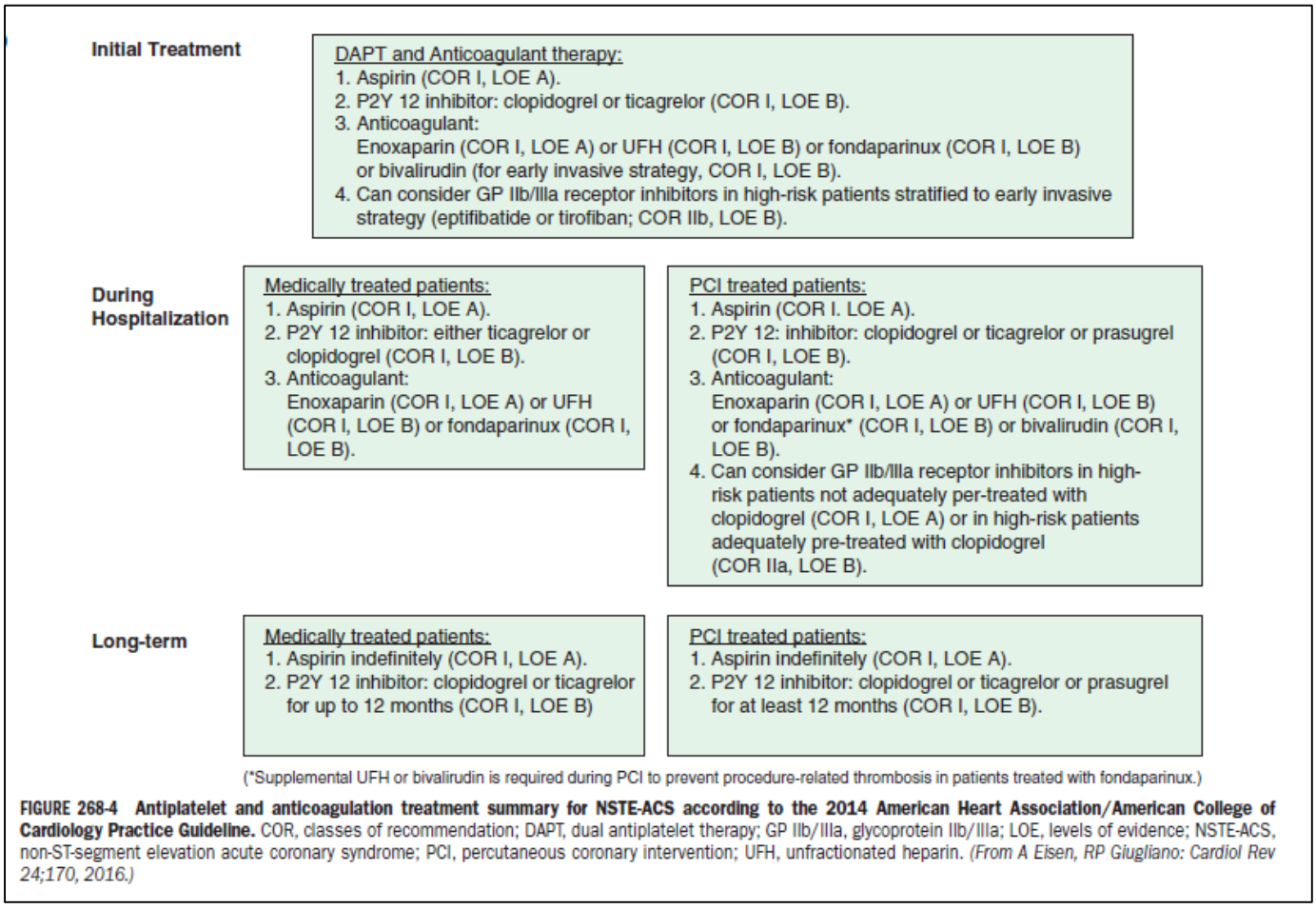


FIGURE 268-4 Antiplatelet and anticoagulation treatment summary for NSTE-ACS according to the 2014 American Heart Association/American College of Cardiology Practice Guideline. COR, classes of recommendation; DAPT, dual antiplatelet therapy; GP IIb/IIIa, glycoprotein IIb/IIIa; LOE, levels of evidence; NSTE-ACS, non-ST-segment elevation acute coronary syndrome; PCI, percutaneous coronary intervention; UFH, unfractionated heparin. *(From A Eisen, RP Giugliano: Cardiol Rev 24;170, 2016.)*

TABLE 268-3 Clinical Use of Antithrombotic Therapy

Oral Antiplatelet Therapy	
Aspirin	Initial dose of 325 mg nonenteric formulation followed by 75–100 mg/d of an enteric or a nonenteric formulation
Clopidogrel	Loading dose of 300–600 mg followed by 75 mg/d
Prasugrel	Pre-PCI: Loading dose 60 mg followed by 10 mg/d
Ticagrelor	Loading dose of 180 mg followed by 90 mg twice daily
Intravenous Antiplatelet Therapy	
Abciximab	0.25 mg/kg bolus followed by infusion of 0.125 μg/kg per min (maximum 10 μg/min) for 12–24 h
Eptifibatide	180 μg/kg bolus followed 10 min later by second bolus of 180 μg with infusion of 2.0 μg/kg per min for 72–96 h following first bolus
Tirofiban	25 μg/kg per min followed by infusion of 0.15 μg/kg per min for 48–96 h
Cangrelor	30 μg/kg bolus followed immediately by a 4 μg/kg per min infusion
Anticoagulants[a]	
Unfractionated heparin (UFH)	[b]Bolus 70–100 U/kg (maximum 5000 U) IV followed by infusion of 12–15 U/kg per h (initial maximum 1000 U/h) titrated to ACT 250–300 s
Enoxaparin	1 mg/kg SC every 12 h; the first dose may be preceded by a 30-mg IV bolus; renal adjustment to 1 mg/kg once daily if creatine clearance <30 mL/min
Fondaparinux	2.5 mg SC qd
Bivalirudin	Initial IV bolus of 0.75 mg/kg and an infusion of 1.75 mg/kg per h

[a]Other low-molecular-weight heparins have been studied other than enoxaparin; however there are less data to support their use. [b]If no glycoprotein IIb/IIIa inhibitor planned.

Abbreviations: ACT, activated clotting time for HemoTec; IV, intravenous; SC, subcutaneous.

Source: Modified from J Anderson et al: J Am Coll Cardiol 61:e179, 2013.

ST ELEVATED MI

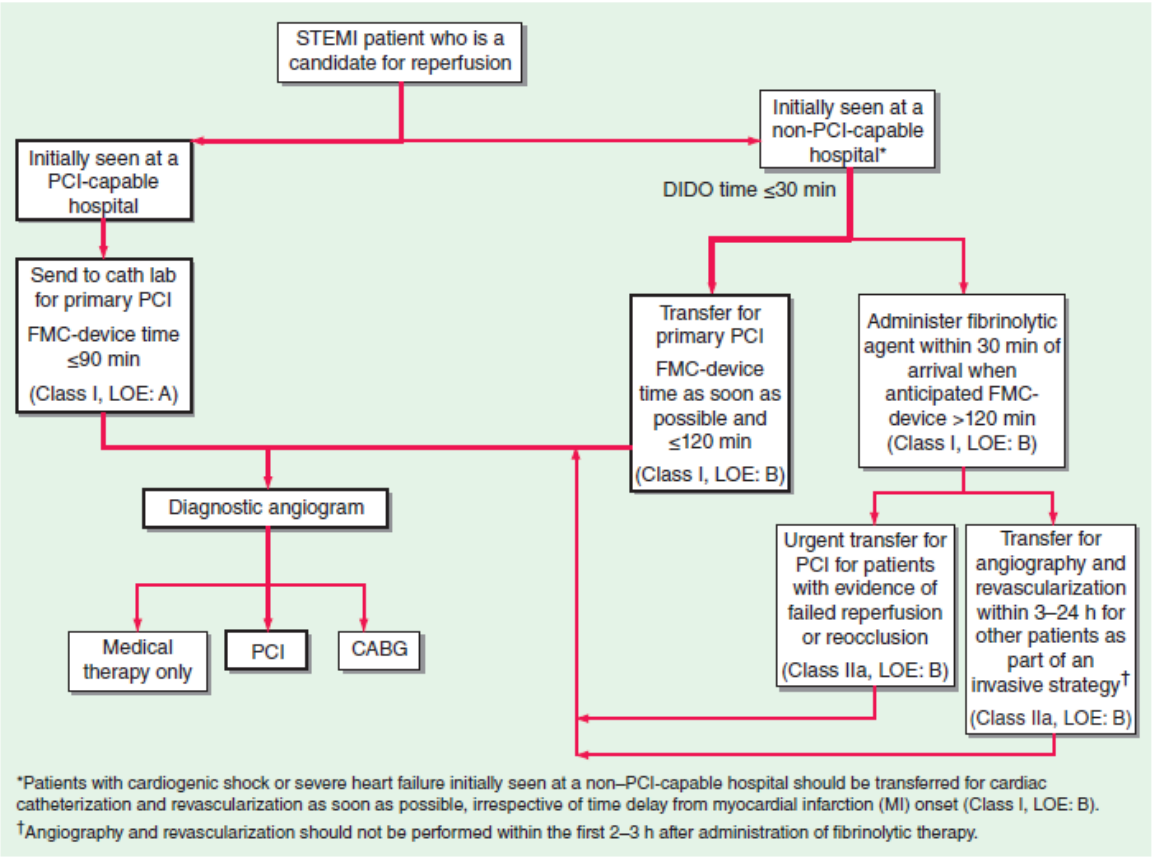


FIGURE 269-4 Reperfusion therapy for patients with ST-segment elevation myocardial infarction (STEMI). The *bold arrows* and *boxes* are the preferred strategies. Performance of percutaneous coronary intervention (PCI) is dictated by an anatomically appropriate culprit stenosis. CABG, coronary artery bypass graft; DIDO, door-in–door-out; FMC, first medical contact; LOE, level of evidence; STEMI, ST-elevation myocardial infarction. *(Adapted with permission from P O'Gara et al: Circulation 127:e362, 2013.)*

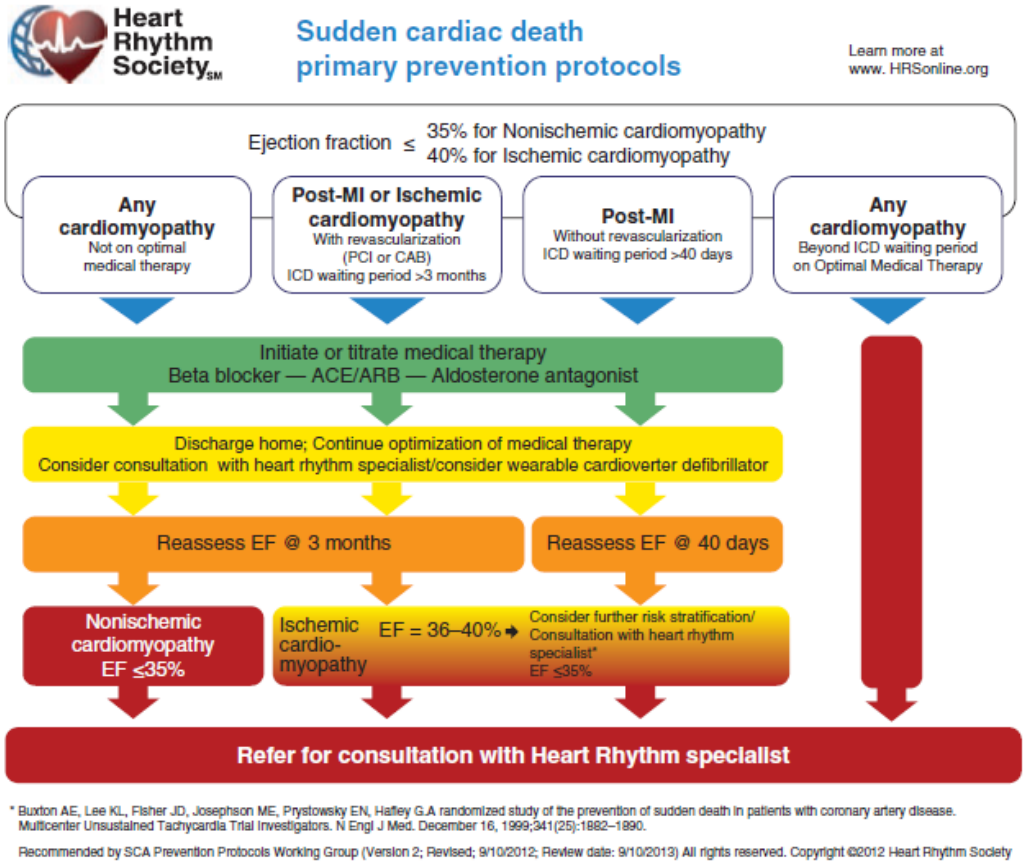


FIGURE 269-5 Algorithm for assessment of need for implantation of a cardioverter-defibrillator. The appropriate management is selected based on measurement of left ventricular ejection fraction, the timing following infarction, and whether revascularization has been performed. *(Reproduced from data at www.hrsonline.org.)*

PHARMACOTHERAPY OF ACUTE MI (STEMI) - In Emergency Setting

1. Admit the patient. Start moist O_2 inhalation.
2. ASPIRIN 325 mg to be chewed stat and CLOPIDOGREL 300 mg to be chewed stat along with ATORVASTATIN 80 mg stat.
3. Sublingual NITROGLYCERIN 0.4mg upto 3 doses at 5 minutes interval; if pain still perists, IV NITROGLYCERIN 5-10 µg/minute.
4. MORPHINE 2-4 mg IV every 5minutes – as an analgesic, anxiolytic.
5. METOPROLOL 5 mg IV every 5 minutes, with maximum of 3 doses only, if heart rate is > 60bpm and Systolic Blood pressure >100 mm Hg and no Acute LVF has been diagnosed.
6. **Fibrinolysis** must be done within **30 minutes of presentation, either FENECTEPLASE IV 0.53 mg/kg body weight over 10 seconds or IV STREPTOKINASE 1.5 million units over 1 hour.**
7. ENOXAPRIN 1 mg/kg body weight subcutanoeusly given, every 12 hours with a preceding bolus dose of 30 mg.

N.B. **Primary Subcutaneous Coronary Intervention (PCI)** is preferred for restoring perfusion in STEMI.