

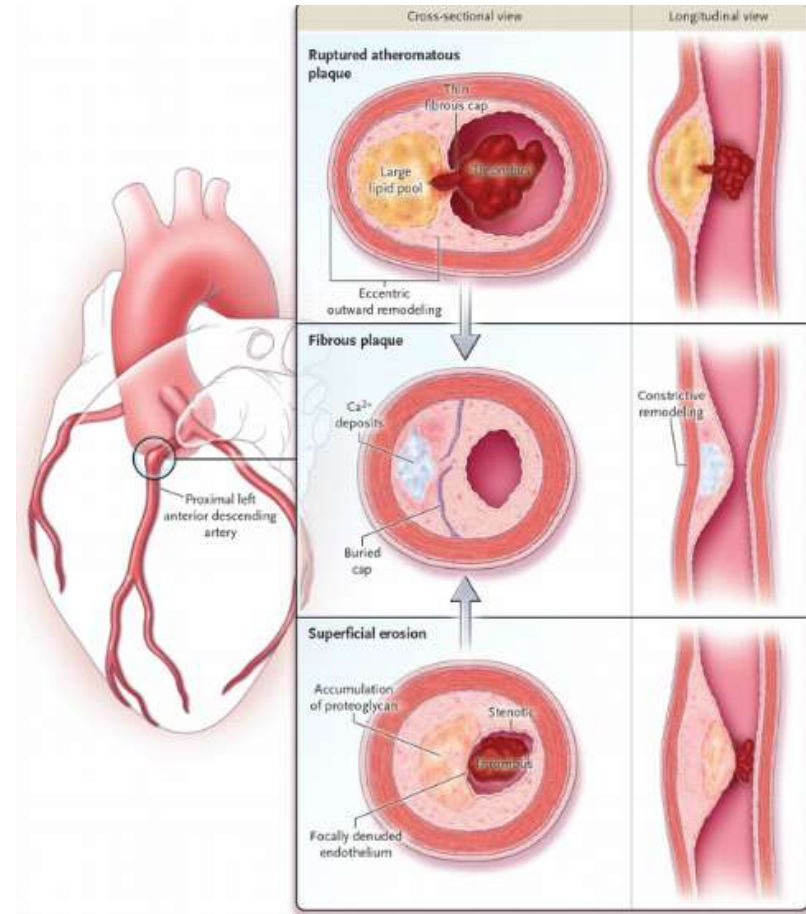
Acute coronary syndrome

LECTURE IN INTERNAL MEDICINE FOR V COURSE STUDENTS

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Plan of the Lecture

- Definition
- Epidemiology
- Risk factors
- Etiology
- Mechanisms
- Classification
- Clinical investigation
- Diagnosis
- Treatment
- Lifestyle modifications and cardiac rehabilitation
- Prognosis
- Prophylaxis
- Abbreviations
- Diagnostic and treatment guidelines



Definition

An acute coronary syndrome (ACS) has evolved as a useful operational term that refers to a spectrum of conditions compatible with acute myocardial ischemia and/or infarction (MI) that are usually due to an abrupt reduction in coronary blood flow ranging clinically from those for ST-segment elevation myocardial infarction (STEMI) to presentations found in non-ST-segment elevation myocardial infarction (NSTEMI) or in unstable angina (UA).

Most patients with STEMI develop QMI, and a few develop NQMI. Most of those without ST-elevation develop NQMI and only a few may develop QMI.

The ACS associated with high healthcare costs, frequent recurrences and hospitalizations, and high risks of sudden death and short-term mortality

Definition

(Unstable Angina)

- UA is defined as myocardial ischemia at rest or minimal exertion in the absence of cardiomyocyte necrosis.
- Among patients presenting with suspected NSTEMI-ACS, the introduction of high-sensitivity cardiac troponin measurements in place of standard troponin assays resulted in an increase in the detection of MI and a reciprocal decrease in the diagnosis of UA.
- Compared with NSTEMI patients, individuals with UA do not experience myocardial necrosis, have a substantially lower risk of death and appear to derive less benefit from intensified antiplatelet therapy as well as early invasive strategy.

Definition

(Definitions of Myocardial Infarction)

- Universal: MI defines cardiomyocyte necrosis in a clinical setting consistent with acute myocardial ischemia.
- My: Acute myocardial infarction is a disease or a clinical syndrome accompanying other diseases, which is represented by acute coronarogenous aseptic inflammation of the part of a heart wall, and clinically correlates with stress reactions of body control systems and is determined by the extent, localization, nature, and stage of structural transformations in the infarction zone, as well as circulation changes, and their consequences.

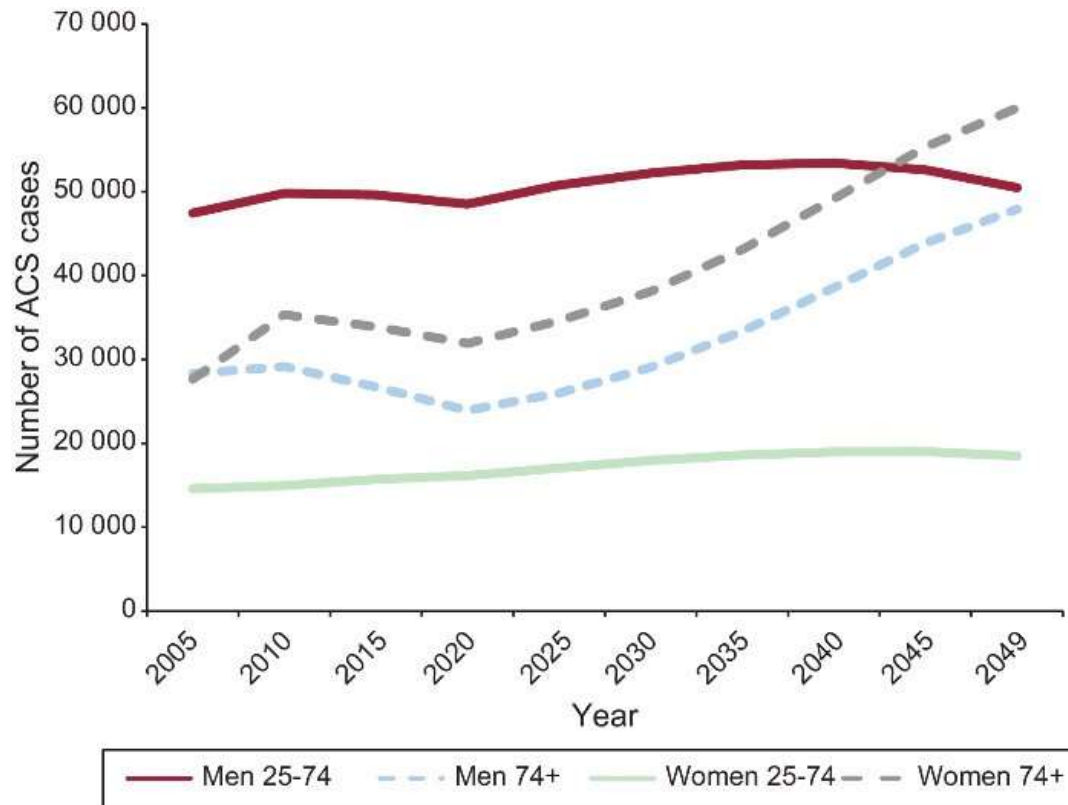
Epidemiology

(Acute Coronary Syndrome around the World)

- Worldwide, coronary artery disease (CAD) is the single most frequent cause of death.
- Over seven million people every year die from CAD, accounting for 12.8% of all deaths.
- Every sixth man and every seventh woman in Europe will die from myocardial infarction.
- The incidence of hospital admissions for myocardial infarction varies among countries.
- The most comprehensive STEMI registry is probably in Sweden, where the incidence is 66 STEMI/100 000/year.

Epidemiology

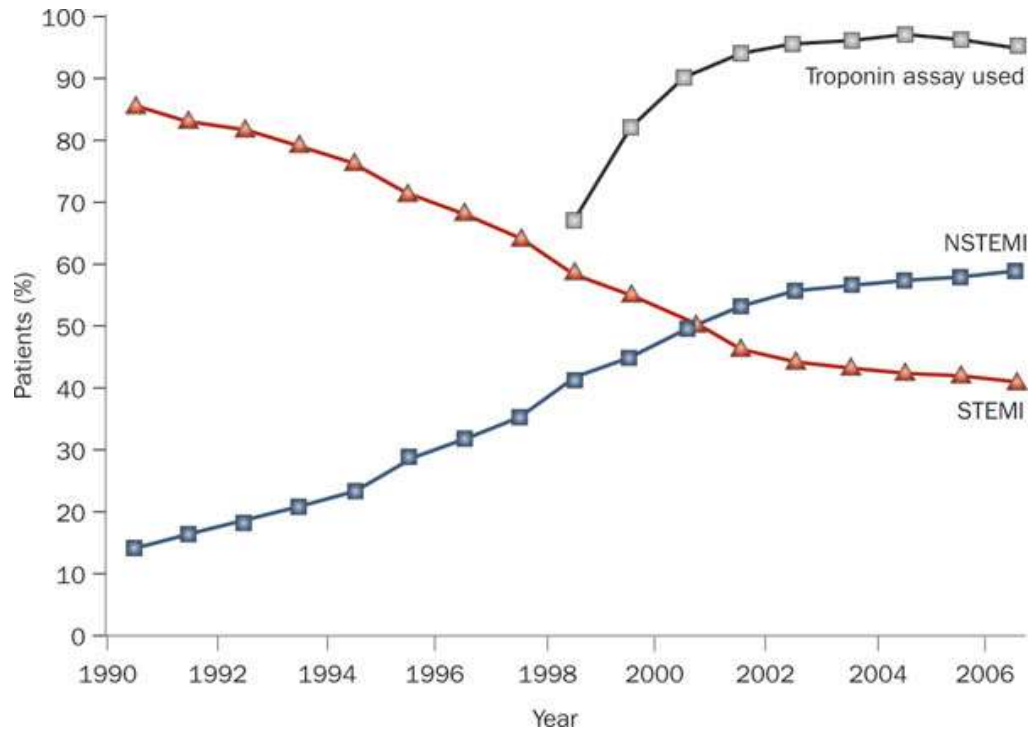
(Acute Coronary Syndrome in the Spanish Population)



Number of acute coronary syndrome cases, trend from 2005 to 2049 by sex and age group in the Spanish population. ACS, acute coronary syndromes.

Epidemiology

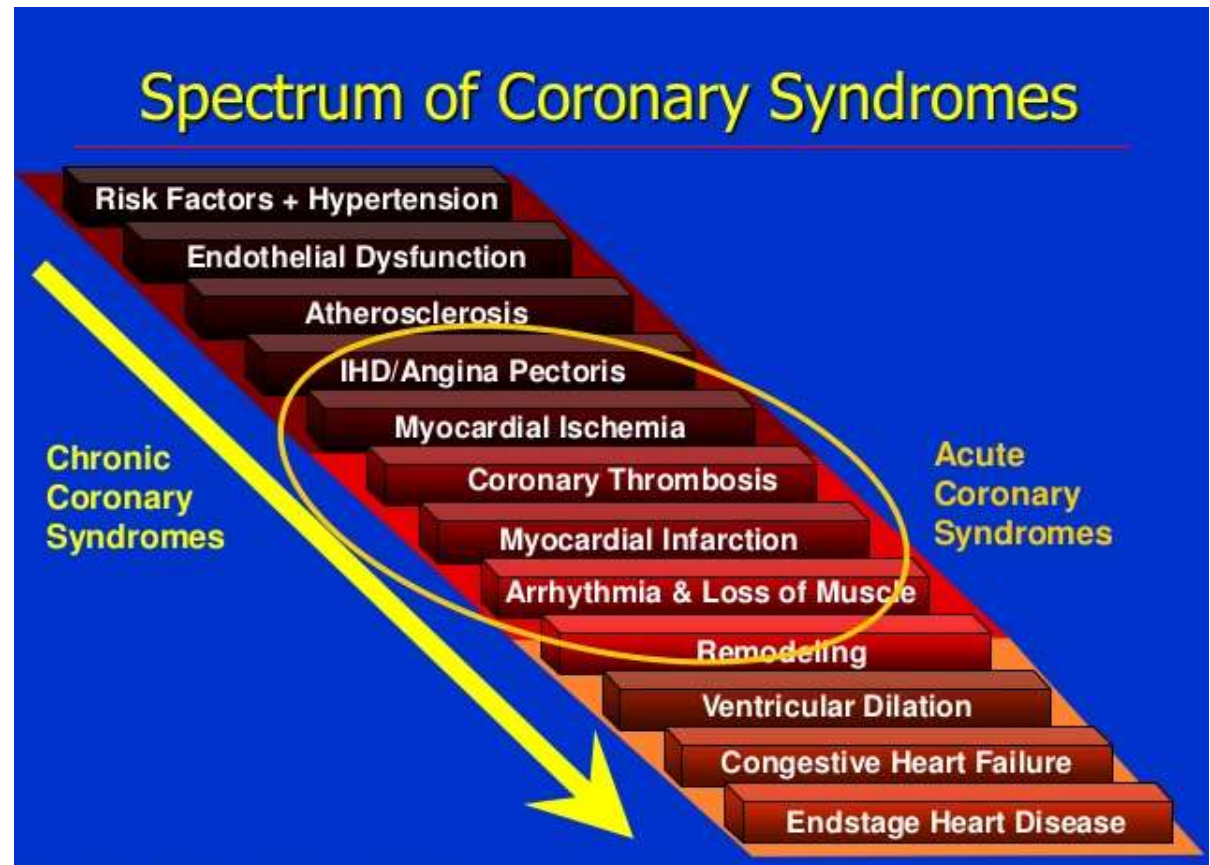
(Troponin and Diagnosis of ST-elevated and non-ST-elevated Acute Myocardial Infarction)



Quality of a diagnosis depends strictly from diagnostic procedures

Risk Factors

The risk factors for an ACS are the same as those for other types of clinical atherosclerosis including heart disease.



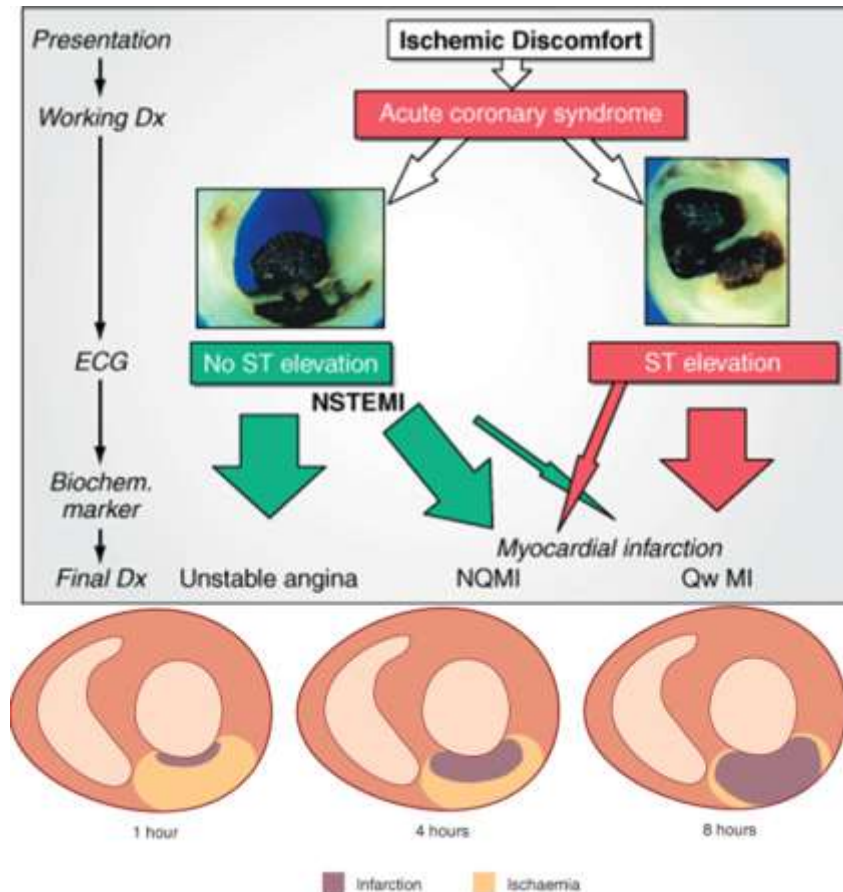
Etiology

- The hallmark of an ACS is the sudden imbalance between myocardial oxygen consumption (MVO_2) and demand, which is usually the result of coronary artery obstruction.
- The imbalance may also be caused by other conditions: excessive myocardial oxygen demand in the setting of a stable flow-limiting lesion; acute coronary insufficiency due to other causes (e.g., vasospastic [Prinzmetal] angina, coronary embolism, coronary arteritis); noncoronary causes (e.g., hypotension, severe anemia, hypertension, tachycardia, hypertrophic cardiomyopathy, severe aortic stenosis); nonischemic myocardial injury (e.g., myocarditis, cardiac contusion, cardiotoxic drugs); and multifactorial causes (e.g., stress [Takotsubo] cardiomyopathy, pulmonary embolism, severe heart failure [HF], sepsis).

Mechanisms

(In Reversible and Irreversible Ischemia Terms)

- UA develops, until local coronary circulation disturbances cross the time of reversible ischemia.
- MI develops, when local coronary circulation disturbances cross the time threshold and make the ischemia irreversible.



Dx - abbreviation for diagnosis.

Mechanisms (Key Moments)

- Mechanisms of an ACS were selected by evolution and consist at systemic and local levels.
- These mechanisms are aimed at providing most favourable of possible the ACS courses.
- Their disturbances build complications of the ACS.

Mechanisms

(Systemic Level: 1)

- The response of body systems to an ACS is realized through stress and manifests itself as brain mediated sympathetic activation and increased functional activity of a hypothalamo-pituitary an adrenal systems with the change of functions of all target organs.
- For a favourable course and outcome of the ACS, all other conditions being equal, an adequate organization of stress (eustress) becomes of primary importance.
- For STEMI leukocyte reactions are important for the further development of the process:
 - These reactions are triggered by the ejection of leukocytes from the depot to the systemic blood flow.

Mechanisms

(Systemic Level: 2)

- Since the depot mainly contains neutrophils, leukocytosis appears as the shift in cell count, neutrophils are activated and migrate to the infarction zone by positive chemotaxis. Infarction zone products getting in the blood flow play the role of attractants for them.
- The activation of neutrophils appears as hyperenzymemia, higher contents of eicosanoids, leukotrienes in particular, protein carbohydrate complexes, and other biologically active agents.
- Stress is changing as the process develops and leukocytosis declines with the leukogram changes. Granulocyte counts decrease, and their functional activity is suppressed, while agranulocyte counts and their activity increase.

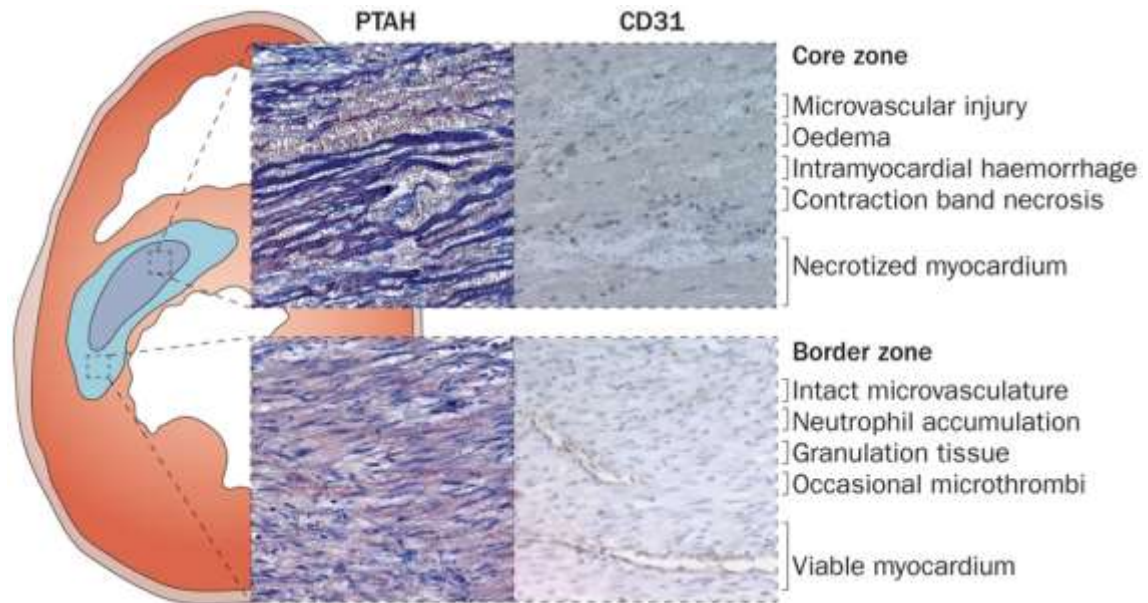
Mechanisms

(Systemic Level: 3)

- The structural change of the leukogram is the result of controlling effects of infarction zone products getting in the blood flow: neutrophil decay products from the infarction zone are repellents for neutrophils and attractants for agranulocytes.
- As result an enzyme level in blood falls, while the proteins and protein-carbohydrate complexes content grows.
- These are the systemic manifestations of an inflammation process in the infarction zone.
- With the termination of an acute myocardial infarction phase, regulation problems disappear and not a trace remains of the stress.

Mechanisms (Local Level)

- The local level is the heart.
- The components of a pathologic process are not only changes in the infarction zone and recovery of heart shape and size but also adaptive changes of heart biomechanics.



The top panels show the infarction zone and the bottom panels show the border (periinfarction) zone

Mechanisms

(Infarction Zone: 1)

- All that happens is inflammation: special, aseptic, alternative but still inflammation.
- The first one is ischemia, reversible myocardial changes (NSTEMI).
- The transfer to irreversible changes (STEMI) marks the onset of necrosis, and with the transfer from ischemic changes in the infarction zone to the necrotic ones, the inflammation starts in accordance with its traditional scheme.
- The necrotized myocardium undergoes destruction, and decay products are removed through the peri-infarction zone.

Mechanisms

(Infarction Zone: 2)

- The necrotized myocardium is specifically destroyed by neutrophil getting by chemotaxis from blood into the infarction zone and producing cathepsins. Their migration rate is rather high, about 2-4 mm per hour and even the largest possible infarction is infiltrated by neutrophils in 6 hours at the most.
- At the same time, fibroblast precursors enter the infarction zone and the recovery process begins.
- It is impossible to separate necrotic and reparative processes, to look at them as the individual ones. They are synchronized and interconnected not only at the level of the infarction zone itself but also at the systemic blood level.

Mechanisms

(Infarction Zone: 3)

- The result of a natural inflammation course in the infarction zone is the formation of a full-fledged scar in the place of necrosis .
- Maturation of a granulation tissue results in its consolidation followed by a decrease in infarction zone sizes. Depending on conditions, they can decrease by 25% or more.
- I should remind those who want to strongly intervene in the infarction zone that the phenomena occurring there (inflammation, compensatory and adaptive responses) are protective reactions originated as the result of evolution.
- We may intervene in these processes but carefully.

Mechanisms

(Periinfarction Zone)

- Systemic mechanisms and the infarction zone are interconnected through the periinfarction zone, first of all, through its microcirculatory bed.
- Wastes from the infarction zone are removed through it, and the products necessary for reparative processes enter there the same way.
- The larger the infarction–periinfarction zone interface, the better the mutual effect of the infarction zone and systemic control.
- The peri-infarction zone cannot be smaller than that required for uncomplicated healing of the infarction.

Mechanisms

(Heart Shape and Size)

- In the case of infarction, a part of a myocardium becomes disabled and its functions should be compensated.
- Hypertrophy of an intact myocardium develops.
- Healing of the infarction zone accompanied with the consolidation of scar leads to a decrease in its size.
- The heart shape is remodeled in such a way that its anatomic proportions are restored to favor its pumping functions.
- In the best case, the traces of infarction are difficult to reveal, even after thorough investigations.

Mechanisms

(Complications: Frequency and Immediate Cause)

- Acute myocardial infarction is a complex and vulnerable process.
- In the most optimistic estimations, its complications are observed somewhere in a quarter of cases, if it is diagnosed and treated correctly.
- The immediate cause of complications is inadequate stress or distress.
- Have little patience, and we would see how distress is realized as complications.

Mechanisms

(Complications: Standard Mechanism)

- The cause of complications is (hyper reactive, hypo reactive or intermittent) distress.
- The mechanism of complications, irrespective of a distress type, is always the same, viz. desynchronization of necrotic and reparative processes.
- Desynchronization of necrosis and reparation leads to the loss of heart wall strength in the infarction zone with cardiac aneurysm or heart rupture outcomes.

Mechanisms

(Complications: Hyperreactive Distress)

- The result of hyperreactive distress is in intensive and rapid migration of polynuclears to the infarction zone with acceleration of necrosis and destruction of infarcted myocardium and lag behind of reparative processes.
- There is the desynchronization.
- Fast destruction of myocardium in the infarction zone has one more consequence, viz. high concentration gradients of all products formed in it along the boundary with the peri-infarct zone with the electronic instability of a heart and as a result, arrhythmia.

Mechanisms

(Complications: Hyporeactive Distress)

- In this the case everything develops slowly.
- Systemic reactions are sluggish or are absent at all.
- Thus, there are problems with the infiltration of polynuclears to the infarction zone and the necrotic phase is slow, which leads to the delay of reparative processes and their slow development.
- There is the desynchronization again.
- Hypo reactive distress with a sluggish disease pattern determines large sizes of aneurysm and as a rule, heart rupture occurs in the thinnest area of aneurysm.

Mechanisms

(Complications: Intermittent Distress)

Intermittent distress means superposition of the above mechanisms by the formula 'out of the frying-pan into the fire' and everything becomes much more dangerous.

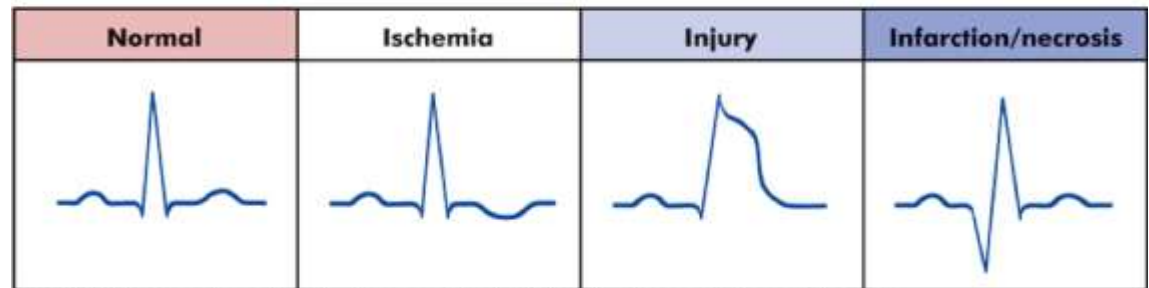
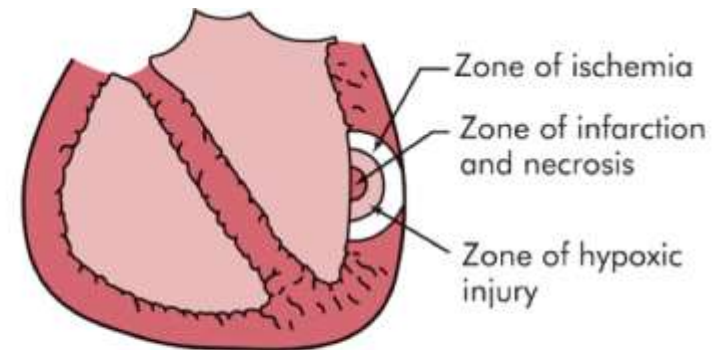


Fig. 23-25. Electrocardiographic Alterations Associated With the Three Zones of Myocardial Infarction

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Classification

(International Classification of Diseases (ICD))

Chapter IX

Diseases of the circulatory system

(I00-I99)

I20-I25 Ischemic heart diseases

I20.0 Unstable angina: Angina (crescendo, de novo effort, worsening effort), Intermediate coronary syndrome, Preinfarction syndrome

I21 ST elevation (STEMI) and non-ST elevation (NSTEMI) myocardial infarction

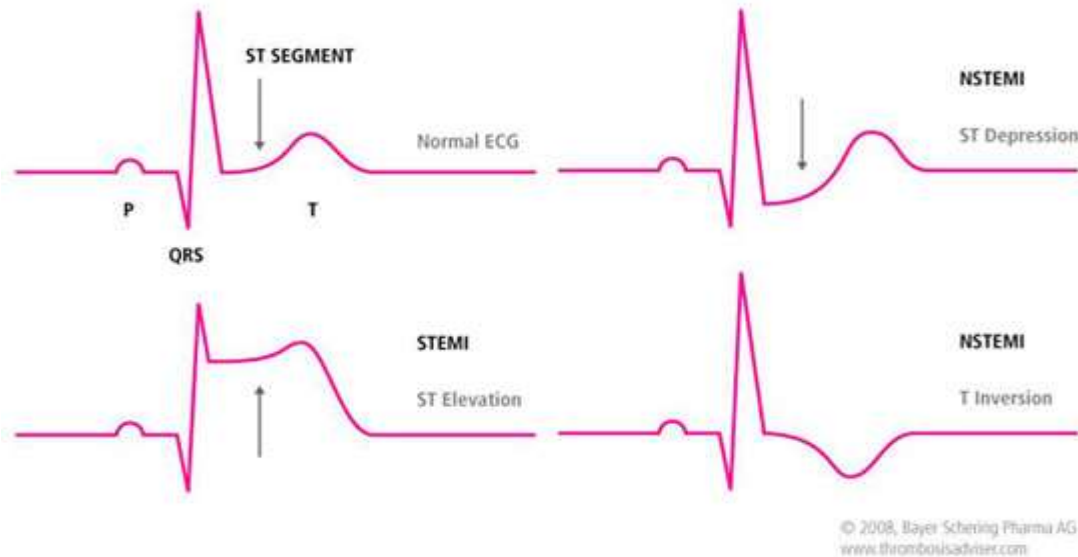
122 Subsequent ST elevation (STEMI) and non-ST elevation (NSTEMI) myocardial infarction

123 Certain current complications following ST elevation (STEMI) and non-ST elevation (NSTEMI) myocardial infarction (within the 28 day period)

124 Other acute ischemic heart diseases

Classification

(Acute Coronary Syndromes Types)



- STEMI - occlusive thrombus - ST elevation (and Q waves) - Cardiac Enzyme elevation - Fibrinolytics beneficial
- NSTEMI - non-occlusive thrombus - NO ST/Q - Cardiac Enzyme elevation present - Fibrinolytics not beneficial
- UA - non-occlusive thrombus - NO ST/Q - Cardiac Enzyme elevation absent - Fibrinolytics not beneficial

Classification

(Myocardial Infarction)

- Anatomic perspective: transmural, nontransmural (subendocardial, subepicardial)
- Localization: anterior, lateral, posterior, septal, inferior, right ventricle
- Clinical periodization: acutest (UA), acute (predominance of necrotic processes), subacute (predominance of reparative processes), postinfarction (consolidation of postinfarction scar)
- Course: noncomplicated, complicated (hyperreactive, hyporeactive)

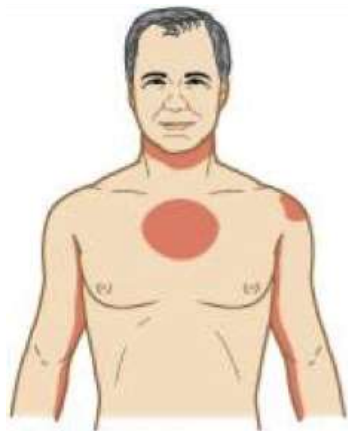
Clinical Investigation

(Symptoms)

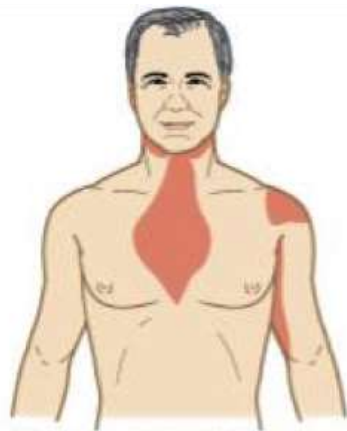
- The cardinal symptom is chest pain, experienced as tightness around the chest and radiating to the left arm and the left angle of the jaw.
- This may be associated with diaphoresis (sweating), nausea and vomiting, as well as shortness of breath.
- In many cases, the sensation is "atypical", with pain experienced in different ways or even being completely absent (which is more likely in female patients and those with diabetes).
- Some may report arrhythmia, anxiety or a sense of impending doom (angor animi) and a feeling of being acutely ill.
- The description of the chest discomfort as a pressure has little utility in aiding a diagnosis as it is not specific for ACS.

Clinical Investigation

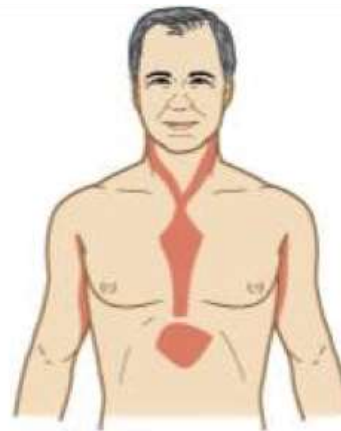
(Common Locations and Patterns of Chest Pain)



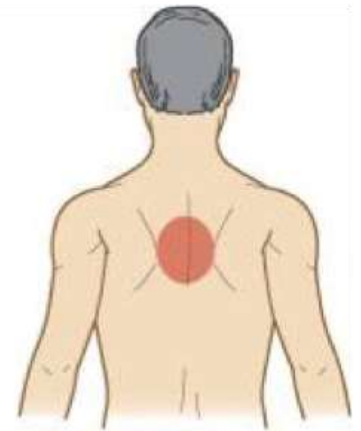
- Mid sternum
- Left shoulder and down both arms
- Neck and arms



- Substernal radiating to neck and jaw
- Substernal radiating down left arm



- Epigastric
- Epigastric radiating to neck, jaw, and arms

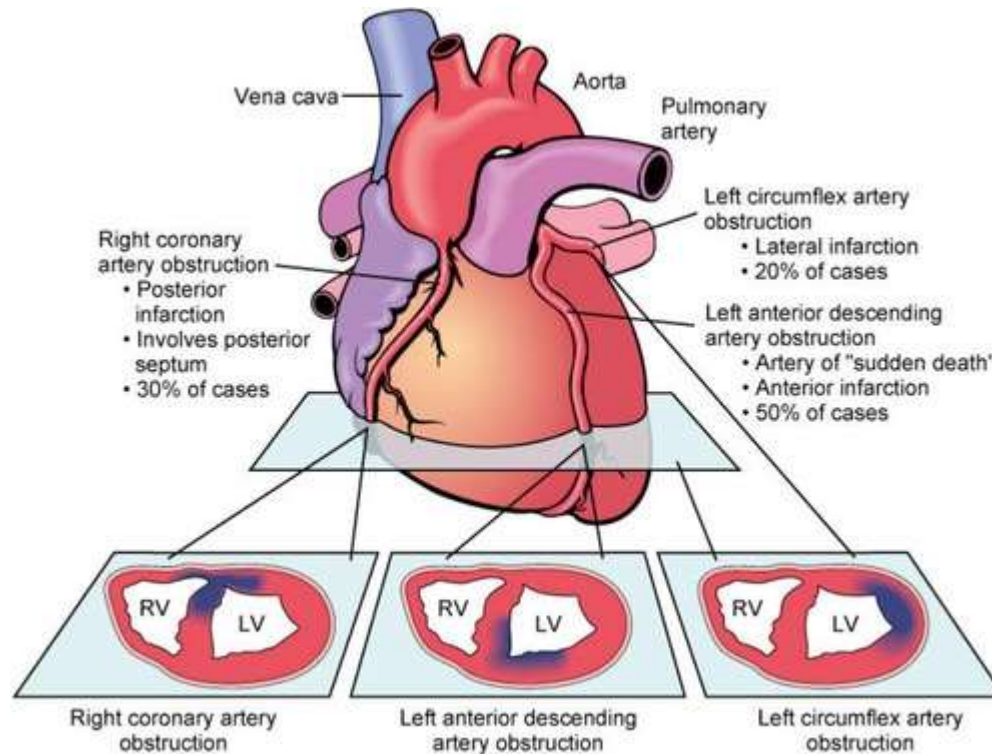


- Intrascapular

The characteristics of discomfort-related to chest pain may be divided into four categories: location, character, duration and relationship to exertion and other exacerbating or relieving factors. The discomfort caused by myocardial ischemia is usually located in the chest, near the sternum, but may be felt anywhere from the epigastrium to the lower jaw or teeth, between the shoulder blades or in either arm to the wrist and fingers.

Clinical Investigation

(Distribution of ACS)



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Clinical Investigation (History)

- The history should include any current symptoms and a complete inventory of comorbid conditions.
- An inventory of cardiac risk factors, and a complete family history are essential components.
- The history should also include information about the character and location of discomfort, radiation of discomfort, associated symptoms, and precipitating, exacerbating, or alleviating factors.
- The importance of the family history should not be underestimated.
- A detailed assessment, particularly of first-degree relatives for the presence of coronary artery disease and age of diagnosis is imperative when evaluating a patient's risk factor profile.

Clinical Investigation

(Physical Examination)

- The results of the physical examination of a patient may be entirely normal.
- The presence of multiple risk factors or atherosclerosis symptoms increases the likelihood that a chest pain is related to myocardial ischemia.
- Evaluation should include measurements of blood pressure, the ankle-brachial index, cardiac and carotid arteries auscultation for bruits.
- Examination of the chest wall, neck, and shoulders for deformities and tenderness may be helpful in diagnosing musculoskeletal chest discomfort.
- The physical examination may help identify comorbid conditions that impact therapeutic risk and decision making.

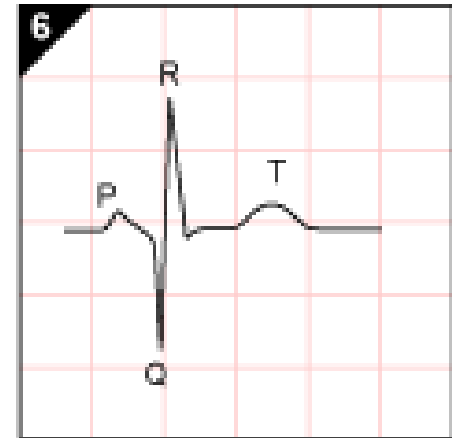
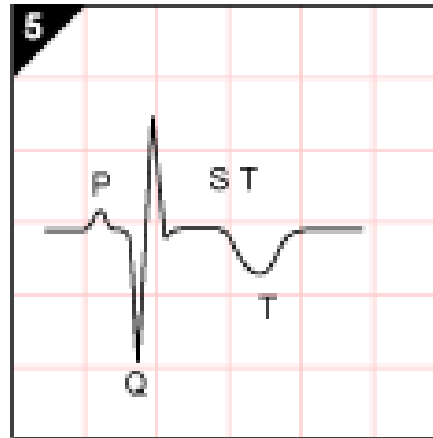
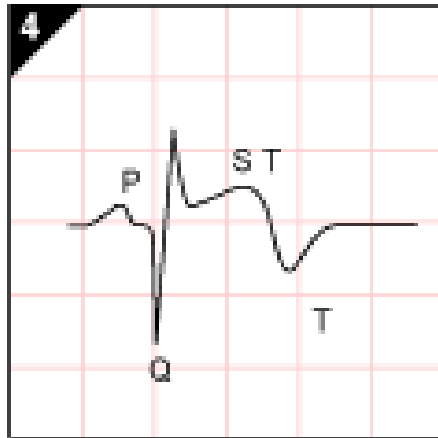
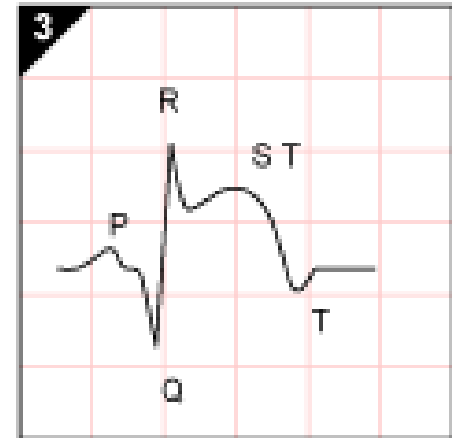
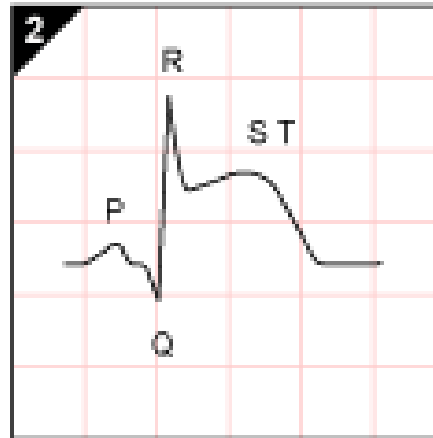
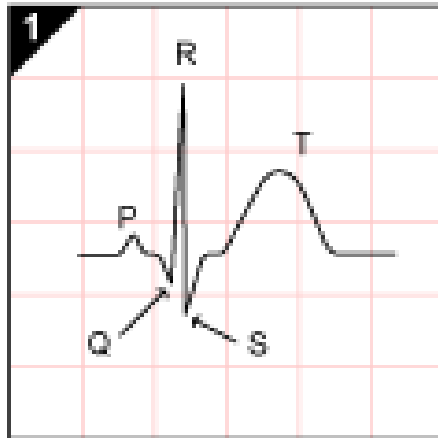
Clinical Investigation

(Myocardial Infarction versus Stable Angina Comparison Chart)

Symptoms	Myocardial Infarction	Stable Angina
Occurrence of pain	Occurs at any time	Occurs due to physical or emotional stress
Modality of pain	With damage to the heart and usually described as severe, steady, and crushing	With no damage to the heart
Outcome	May be fatal	Usually not fatal
Relieving factors of pain	Symptoms persists after 15 min. and not relieved by rest or nitro	Symptoms relieved by rest or nitro within 10-15 min.
Duration of pain	Usually lasts for more than 15 min.	Usually for less than 15 min.; discomfort is transient, lasting 3-5 min.
Serum cardiac marker	Present	Not present

Clinical Investigation

(Sequence of Changes in evolving MI)



Clinical Investigation

(ECG' Localizing of Myocardial Injury)

I LATERAL	aVR	V1 SEPTAL	V4 ANTERIOR
II INFERIOR	aVL LATERAL	V2 SEPTAL	V5 LATERAL
III INFERIOR	aVF INFERIOR	V3 ANTERIOR	V6 LATERAL

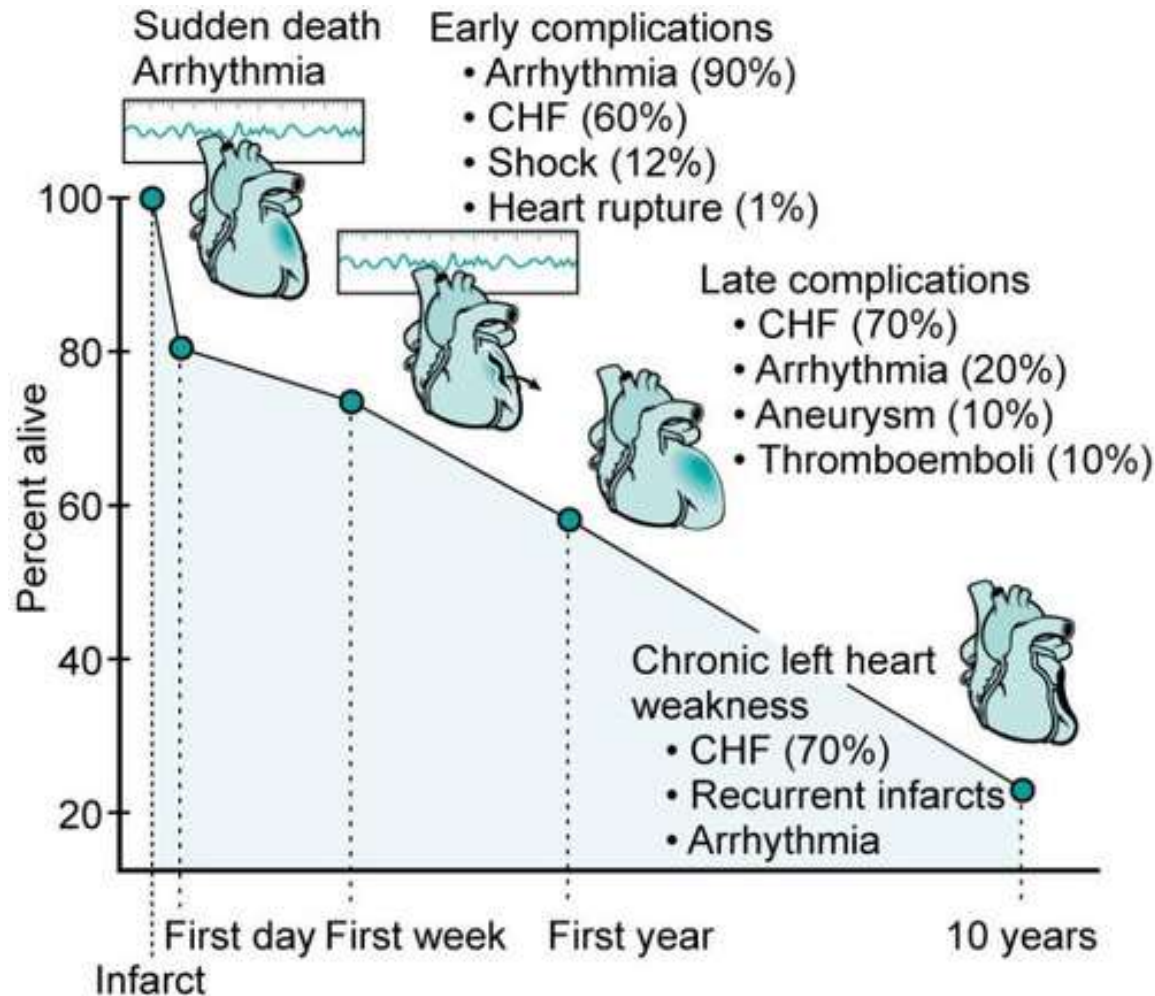
Clinical Investigation

(Complications)

- Complications may occur immediately following the heart attack (in the acute phase), or may need time to develop (a chronic problem).
- Acute complications may include heart failure if the damaged heart is no longer able to pump blood adequately around the body; aneurysm of the left ventricle myocardium; ventricular septal rupture or free wall rupture; mitral regurgitation, in particular if the infarction causes dysfunction of the papillary muscle; Dressler's syndrome; and abnormal heart rhythms, such as ventricular fibrillation, ventricular tachycardia, atrial fibrillation, and heart block.
- Longer-term complications include heart failure, atrial fibrillation, and an increased risk of a second MI.

Clinical Investigation

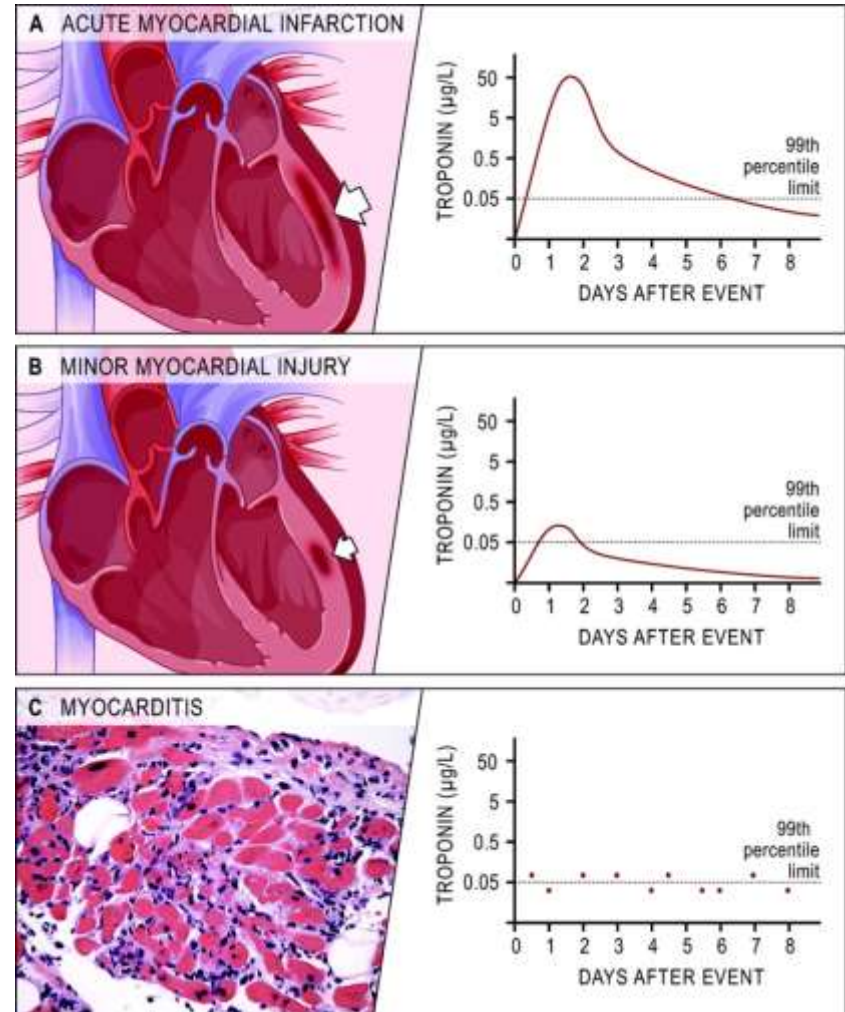
(Outcome of ACS)



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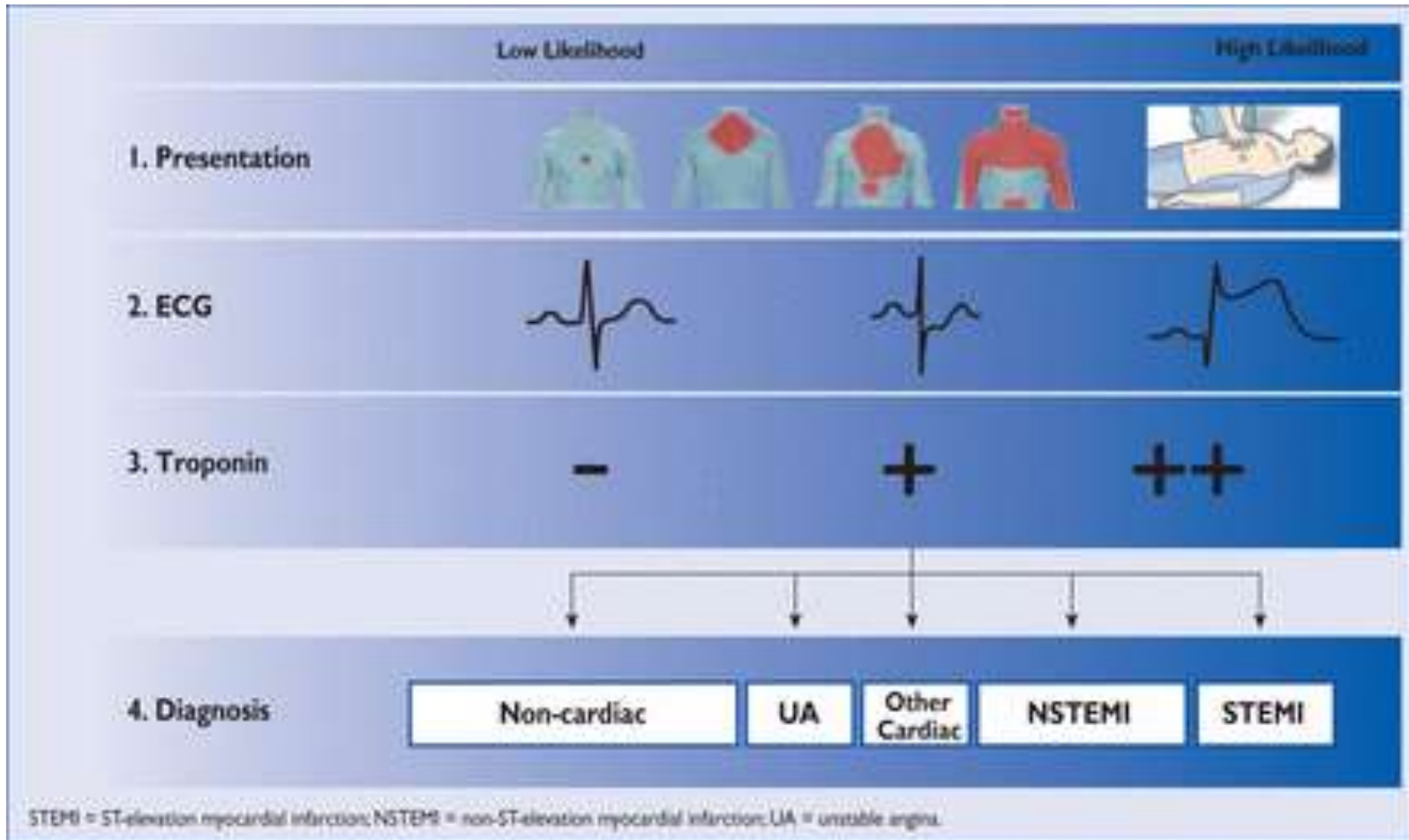
Diagnosis

- Blood tests: cardiac biomarkers; WBC; cholesterol and C-reactive protein levels.
- Electrocardiogram (ECG).
- B-mode, Doppler and intravascular ultrasound.
- Vascular catheterization and angiogram.
- Other imaging tests.



Diagnosis

(Initial Assessment of Patients with Suspected ACS)

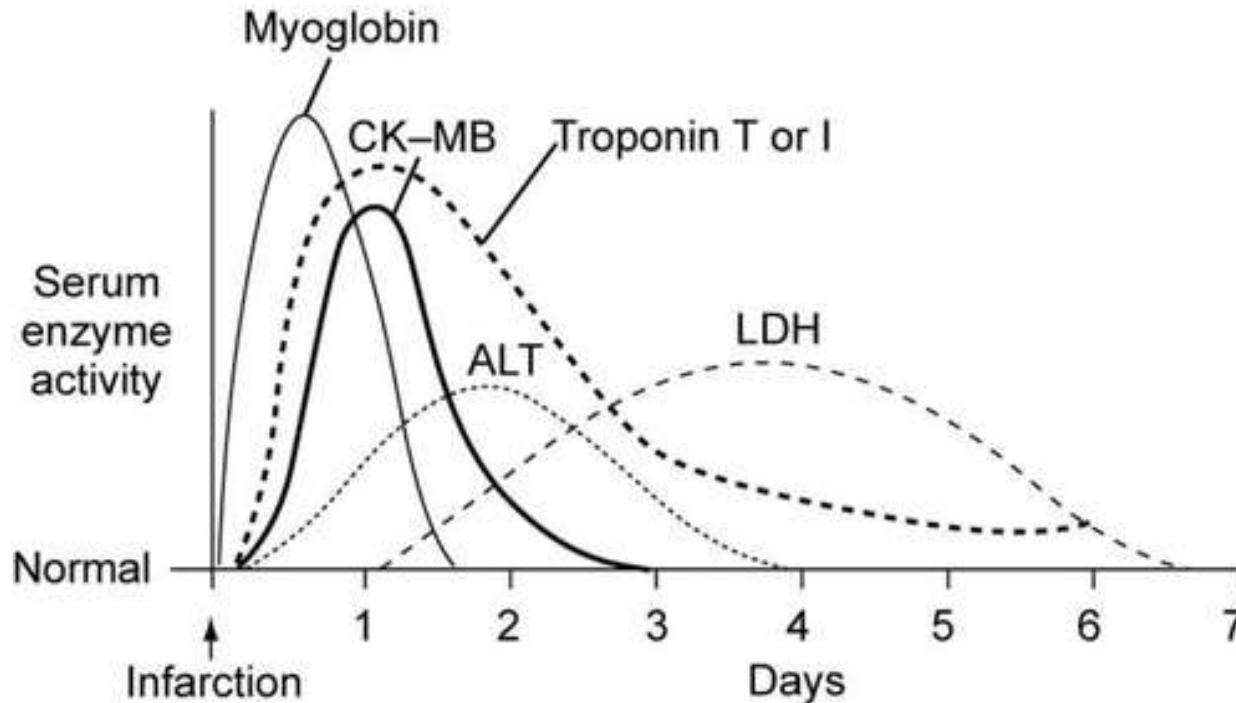


Diagnosis

(Criteria for Diagnosis of MI)

A combination of criteria is required to meet the diagnosis of MI, namely the detection of an increase and/or decrease of a cardiac biomarker, preferably high-sensitivity cardiac troponin, with at least one value above the 99th percentile of the upper reference limit and at least one of the following: symptoms of ischemia; new or presumed new significant ST-T wave changes or left bundle branch block on 12-lead ECG; development of pathological Q waves on ECG; imaging evidence of new or presumed new loss of viable myocardium or regional wall motion abnormality; intracoronary thrombus detected on angiography or autopsy.

Diagnosis (Cardiac Biomarkers)



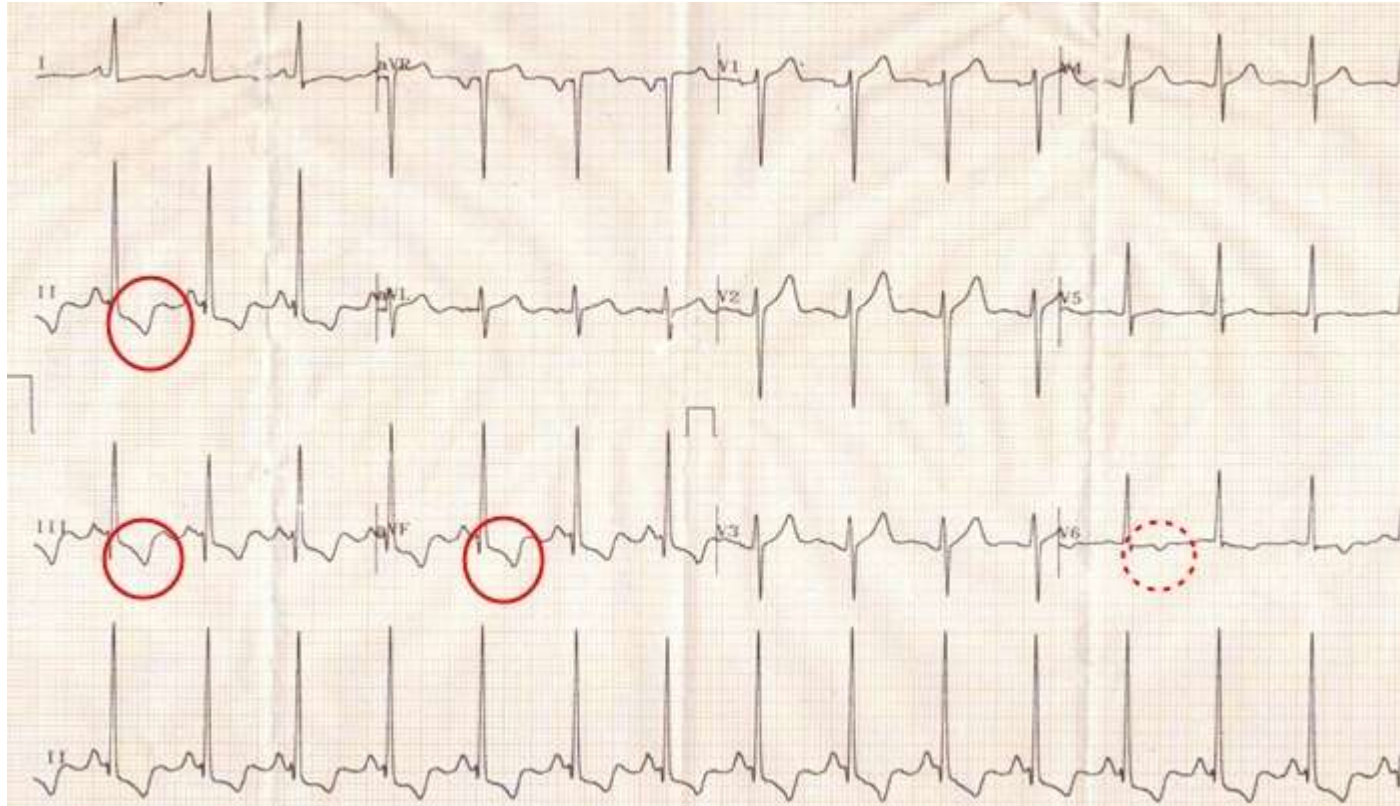
Biomarkers related to necrotic processes in myocardial infarction zone

Diagnosis

(Indications for Measurement of Troponins)

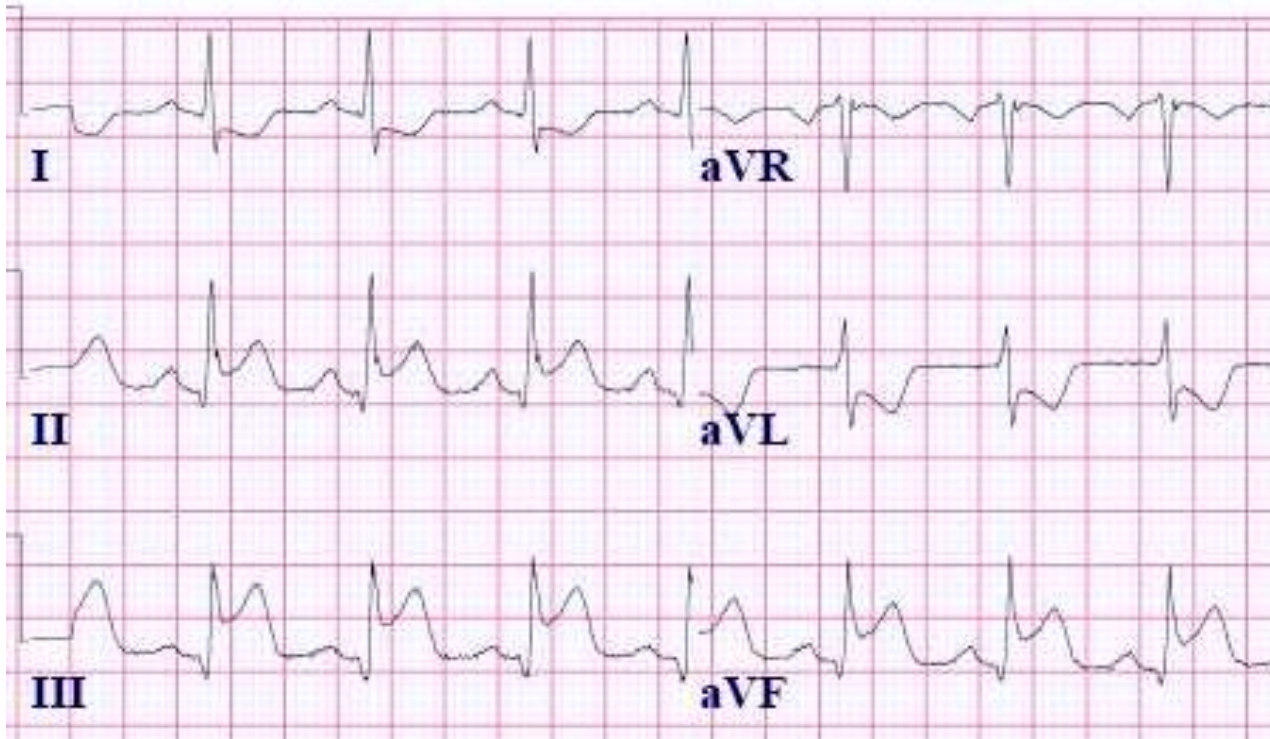
- Cardiac troponin I and T have displaced myoglobin and creatine kinase-MB as the preferred markers of myocardial injury.
- Serum levels increase within 3-12 hours from the onset of chest pain, peak at 24-48 hours, and return to baseline over 5-14 days.
- Troponin levels may not be detectable for six hours after the onset of myocardial cell injury. The most sensitive *early marker* for myocardial infarction is myoglobin.
- Troponin levels should be measured at presentation and again 10-12 hours after the onset of symptoms. When there is uncertainty regarding the time of symptom onset, troponin should be measured at twelve hours after the presentation.
- The risk of death from an ACS is directly related to troponin level and patients with no detectable troponins have a good short-term prognosis.
- Elevated troponin levels can occur in patients without an ACS and are associated with adverse outcomes in many other clinical situations, including congestive heart failure, sepsis, acute pulmonary embolism and chronic kidney disease. Other cardiac causes include myocarditis and aortic dissection.

Diagnosis (Electrocardiogram)



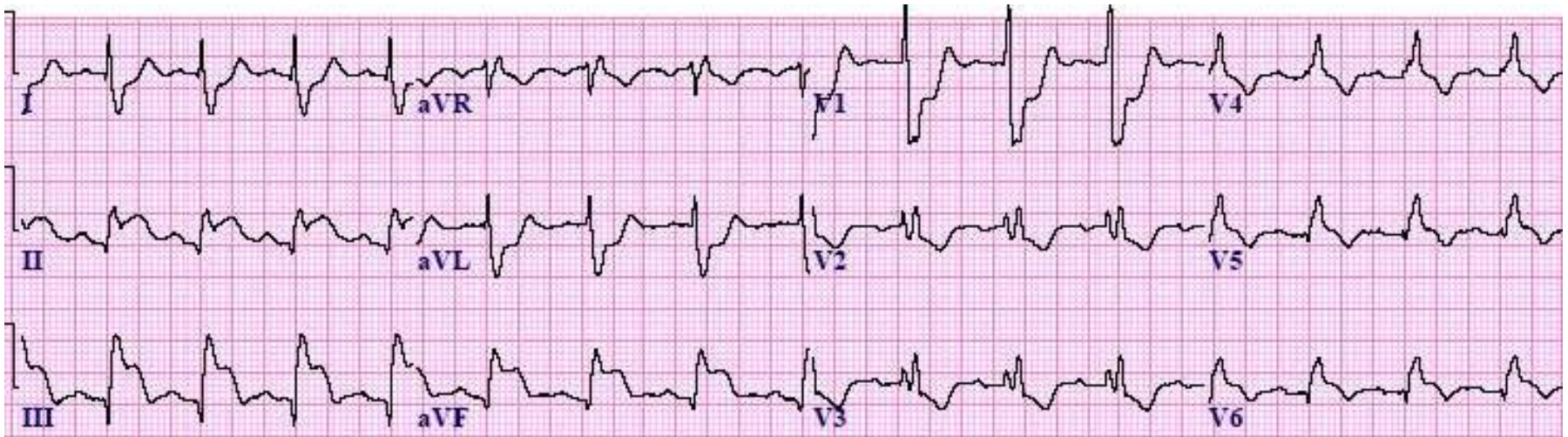
There are ST and T changes in leads II, III, aVF (solid red circles) – it is a suspicion of ischemia of the inferior myocardial wall. Similar change is outlined in the lead V6 (dashed circle).

Diagnosis (Electrocardiogram)



Inferior STEMI in acutest phase

Diagnosis (Electrocardiogram)



An inferior STEMI with a right bundle branch block on the ECG.
The reciprocal depression in lead I and aVL.

Diagnosis

(Criteria of Differentiation Noncomplicated and Complicated MI)

Criterion	Type of acute myocardial infarction		
	Noncomplicated with eureactive stress	Complicated with hyperreactive distress	Complicated with hyporeactive distress
Blood leukocytes, E 9 1/l.: 1 st day results and further dynamics	7-11, rapid decrease	>11, slow decrease	<7, increase, then slow decrease
Shift of leukocytes, arb. units: 1 st day results and further dynamics	3.0-6.5, rapid decrease	>6.5, slow decrease	<3.0, increase, then slow decrease
Time of maximum activity of enzymes, hrs	16-22, rapid decrease	>16, slow decrease	>22, slow decrease
Initial period of protein-carbohydrate complex growth, days	1-2	>2	>2
Period of reaching maximum protein-carbohydrate complex concentrations, days	4-5	>5	>5
Interval between maxima of enzymes and protein-carbohydrate complexes, days	>4	>6	>6
Return of the ST segment on ECG to the isoline, hrs	>36	>36	>36

Treatment

(Prehospital Care and Initial Management)

- All patients should be managed as if the pain is ischemic in origin, unless clear evidence to the contrary is established.
- Specific care includes intravenous access; supplemental oxygen; pulse oximetry; immediate administration of aspirin; nitroglycerin for active chest pain; telemetry and prehospital ECG, if available.
- Additional objectives include adequate analgesia (generally achieved with morphine); pharmacologic reduction of excessive sympathoadrenal and vagal stimulation; treatment of ventricular arrhythmias; support of cardiac output, and respiration.
- Prehospital fibrinolytic therapy by the administration of tissue-type plasminogen activator , aspirin, and heparin may be given by paramedics, as guided by electrocardiographic findings, within 90 minutes of the onset of symptoms.

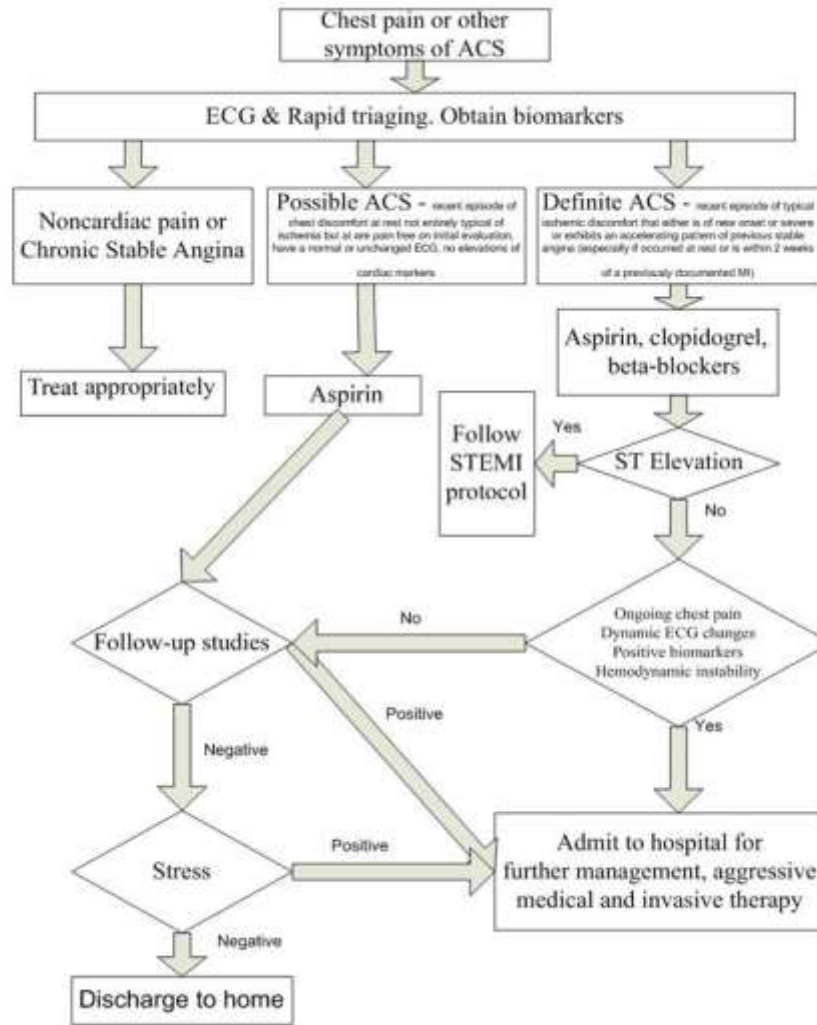
Treatment

(Emergency Department Care and In-Hospital Management)

- Triage and evaluation: because ACS is a spectrum of conditions, initial evaluation to establish a working diagnosis is crucial, as this will dictate management owing to some differences in management steps and timelines for each component of the ACS spectrum.
- Initial management: restoration of the balance between oxygen supply and demand to prevent further ischemia; pain relief, prevention and treatment of complications.

Treatment

(Suggested Algorithm for Triageing Patients with Chest Pain)



Treatment

(Presumed ACS and NSTEMI)

- The accepted management is empirical treatment with aspirin, a second platelet inhibitor such as clopidogrel, and heparin (usually a low-molecular weight heparin such as enoxaparin), with intravenous glyceryl trinitrate and opioids if the pain persists.
- A blood test is generally performed for cardiac troponins twelve hours after onset of the pain and if this is positive, coronary angiography is typically performed on an urgent basis.
- If there is no evidence of ST segment elevation on the electrocardiogram, delaying urgent angioplasty until the next morning is not inferior to doing so immediately.

Treatment (STEMI)

- If the ECG confirms changes suggestive of MI, thrombolytics may be administered or primary coronary angioplasty may be performed.
- The time frame for door-to-needle thrombolytic administration should be within 30 minutes, whereas the door-to-balloon Percutaneous Coronary Intervention (PCI) time should be less than 90 minutes.

Treatment

(Absolute and Relative Contraindications to Fibrinolytic Therapy)

Absolute Contraindications	Relative Contraindications
<p>Prior intracranial hemorrhage</p> <p>Structural cerebral vascular lesion</p> <p>Intracranial neoplasm</p> <p>Ischemic stroke within the past 3 months (except for acute stroke within 4.5 hours)</p> <p>Suspected aortic dissection</p> <p>Active bleeding or bleeding diathesis (excluding menses)</p> <p>Significant closed-head or facial trauma within 3 months</p> <p>Intracranial or intraspinal surgery within 2 months</p> <p>Severe uncontrolled hypertension (unresponsive to emergency therapy)</p> <p>For streptokinase (no longer marketed in the US): Prior treatment within previous 6 months</p>	<p>Chronic, severe, poorly controlled hypertension</p> <p>Systolic pressure >180 mm Hg or diastolic pressure >110 mm Hg</p> <p>History of prior ischemic stroke >3 months</p> <p>Dementia</p> <p>Intracranial pathology</p> <p>Traumatic or prolonged CPR (>10 minutes)</p> <p>Recent (within 2-4 weeks) internal bleeding</p> <p>Noncompressible vascular punctures</p> <p>Pregnancy</p> <p>Active peptic ulcer disease</p> <p>Current use of anticoagulants</p> <p>For streptokinase: prior exposure (>5 days previously) or prior allergic reaction to these agents</p>

CPR = cardiopulmonary resuscitation

Treatment

(Absolute and Relative Contraindications to Fibrinolytic Therapy)

Fibrinolytic Agent	Dose	Fibrin Specificity	Antigenic	Patency Rate
Streptokinase (no longer marketed in the US)	1.5 million units IV given over 30–60 min	No	Yes	60%–68%
Tenecteplase (TNK-tPA)	30 mg for weight <60 kg 35 mg for 60–69 kg 40 mg for 70–79 kg 45 mg for 80–89 kg 50 mg for >90 kg	++++	No	85%
Retepase (rPA)	10-U IV boluses given 30 min apart	++	No	84%
Alteplase (tPA)	Bolus 15 mg followed by infusion 0.75 mg/kg for 30 min (maximum 50 mg), then 0.5 mg/kg (maximum 35 mg) over the next 60 min; total dose not to exceed 100 mg.	++	No	73%-84%

IV = intravenous; rPA = recombinant human tissue plasminogen activator; STEMI = ST-elevation myocardial infarction; tPA = tissue plasminogen activator; US = United States of America.

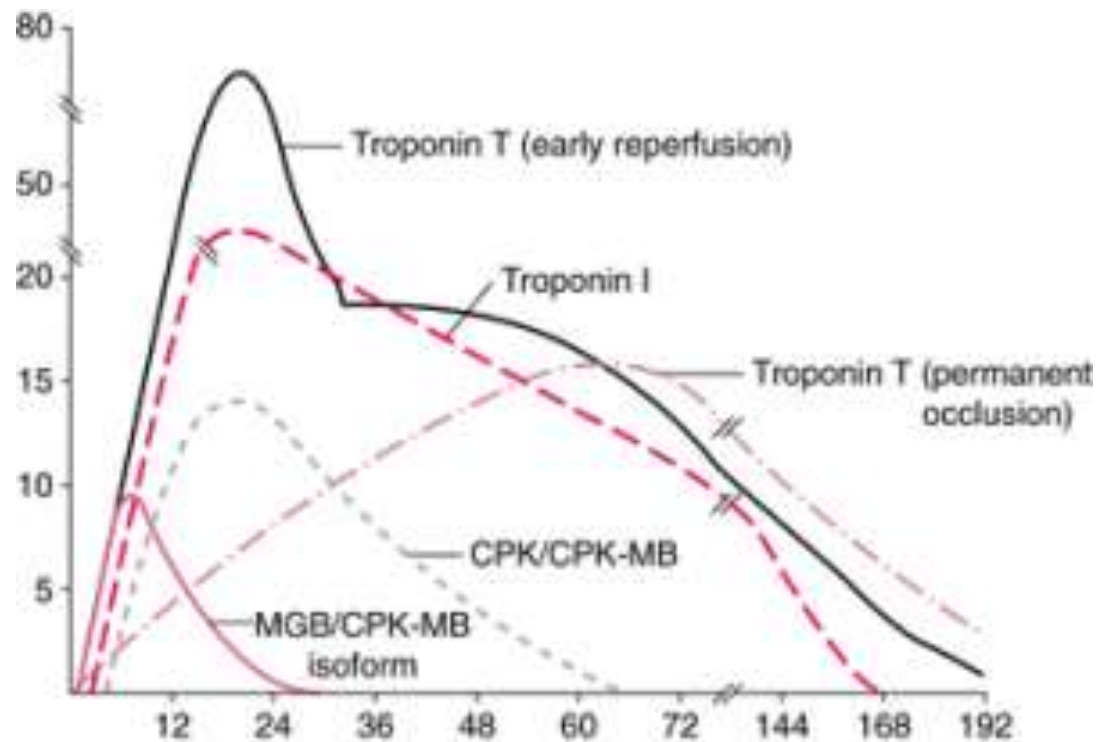
Treatment

(Cardioprotective Medications)

- Inhibitors of the renin-angiotensin-aldosterone (RAA) system
- Beta blockers
- Statins

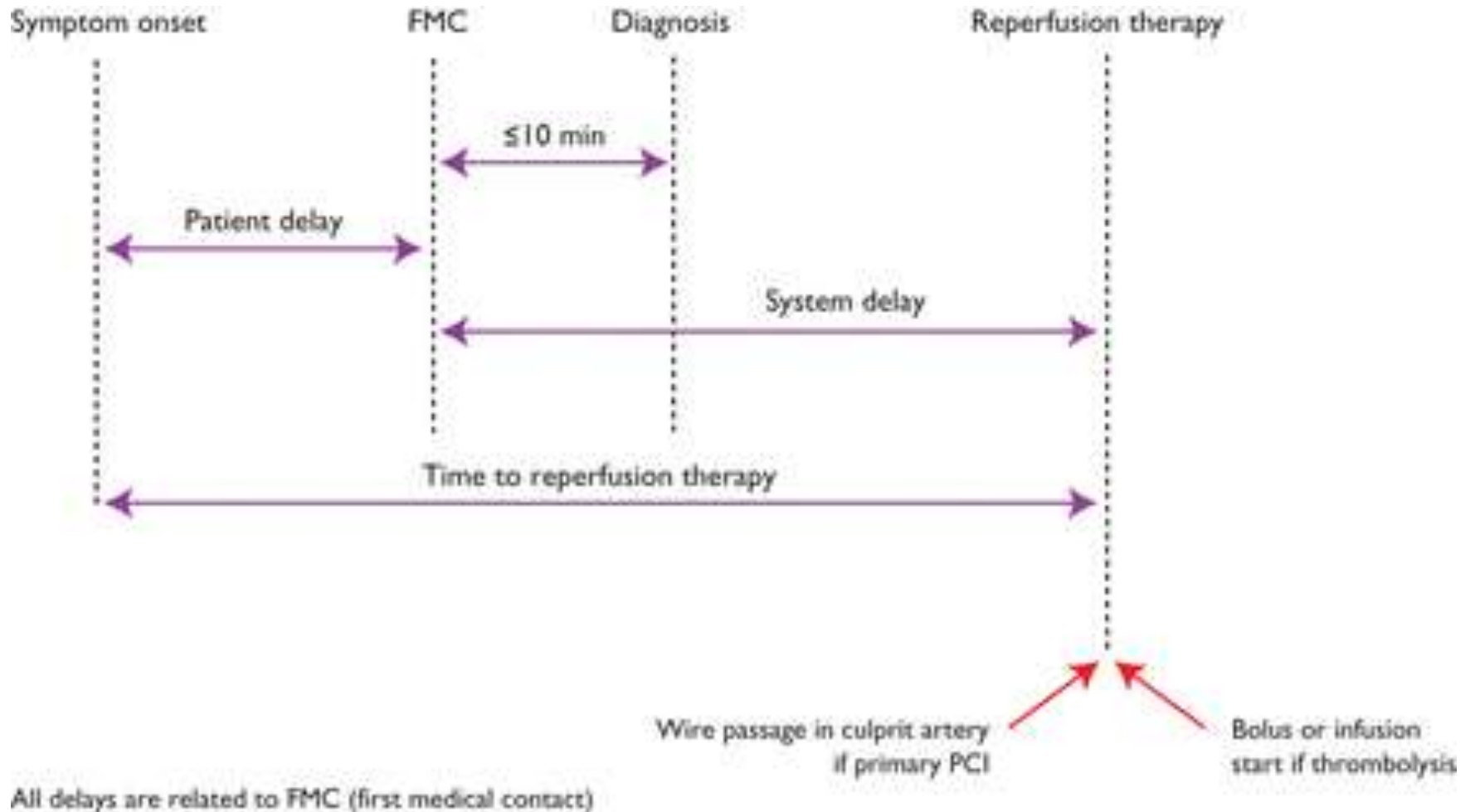
Treatment

(Troponin in MI reperfusion)

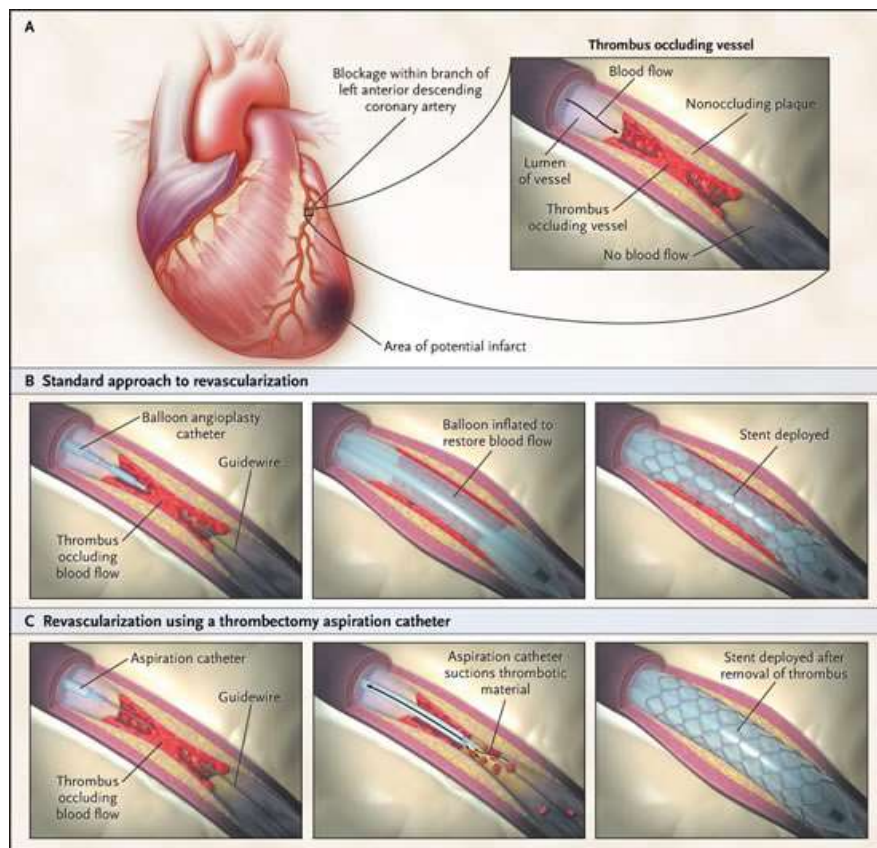


Treatment

(Components of Delay in ACS and Ideal Time Intervals for Intervention)



Treatment (Revascularization)



- Percutaneous coronary intervention
- Coronary artery bypass surgery
- Revascularization vs. medical therapy

Treatment

(Stress' Optimization)

- Upon hyperreactive distress and accelerated necrotic processes, our aim is to decrease the former and to retard the latter, using beta blockaders and anti-inflammatory drugs administered enterally and/or parenterally.
- Hyporeactive distress and retarded necrotic processes require the prescription of alpha -and beta- adrenostimulators, bacterial and leukocytic pyrogens, leukopoiesis stimulants, using several ways of administration.
- Upon intermittent distress, the attitude should be even more careful to prevent wave recurrence of necrotic processes.

Lifestyle Modifications and Cardiac Rehabilitation

- Extensive patient education that includes providing easily understood and culturally sensitive written and verbal instructions about symptoms of ACS, as well as how and when to seek emergency care, in addition to providing instructions about medication types, purposes, doses, frequency, and side effects.
- Dietary changes that adopt a low-fat and low-salt diet with dietary counseling, smoking cessation, up-to-date vaccination, and an increase in physical activity and exercise.
- Aerobic exercise training within a cardiac rehabilitation programs, with the need for an evaluation of both exercise capacity and exercise-associated risk.

Prognosis

- Six-month mortality rates in the Global Registry of Acute Coronary Events (GRACE) were 13% for patients with NSTEMI ACS and 8% for those with unstable angina.
- The prognosis varies greatly depending on a person's health, the extent of the heart damage, and the treatment given.

Prophylaxis

- Lifestyle recommendations include the adoption of a Mediterranean-type diet, maintaining alcohol intake within recommended limits, exercising to the point of mild breathlessness for 20–30 minutes every day, stopping smoking, and trying to achieve a healthy weight.
- Exercise is both safe and effective even if people have had stents or heart failure.
- People are usually started on several long-term medications: aspirin, warfarin, beta blockers, ACE inhibitors, angiotensin II receptor antagonist, statins, aldosterone antagonists.
- Previous studies suggested a benefit from omega-3 fatty acid supplementation but this has not been confirmed.

Abbreviations

- ACE - angiotensin converting enzyme
- ACS - acute coronary syndrome
- ALT - alanine aminotransferase
- ECG – electrocardiography
- ICD - International Classification of Diseases
- LDH - lactate dehydrogenase
- NQMI non-Q-wave myocardial infarction
- NSTEMI - non–ST-segment elevation myocardial infarction
- MI – acute myocardial infarction
- MVO_2 - myocardial oxygen consumption
- STEMI - ST-segment elevation myocardial infarction
- QMI Q-wave myocardial infarction

Diagnostic and treatment guidelines

Europe

- [2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation](#)
- [2014 ESC/EACTS Guidelines on myocardial revascularization](#)
- [ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation](#)

North America

- [2014 AHA/ACC Guideline for the Management of Patients With Non–ST-Elevation Acute Coronary Syndromes](#)
- [Recommendations for best-practice STEMI management in Ontario](#)