Acute Myocardial Infarction

SoM-340

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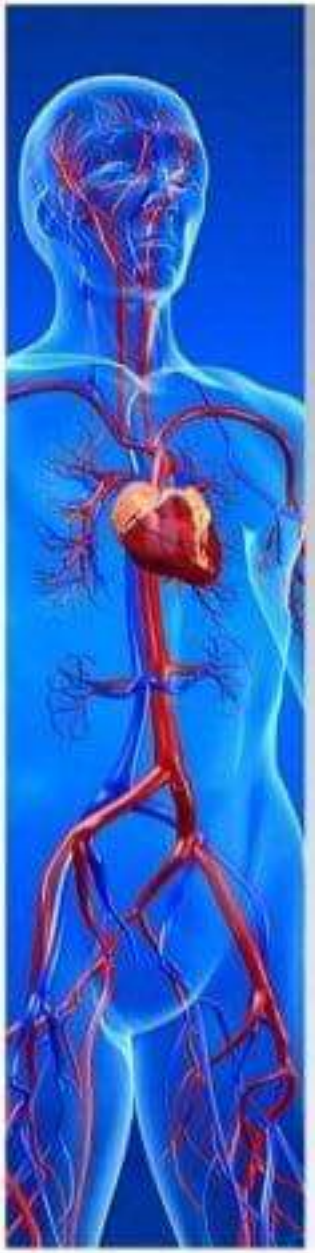
Outline

- Introduction
- Etiology and risk factors
- Pathogenesis
- Classification
- Diagnosis
- Management



Definition

- **Acute myocardial infarction (MI)** is a clinical syndrome that results from occlusion of a coronary artery, with resultant death of cardiac myocytes in the region supplied by that artery.
- Удаан хугацааны миокардын ишемийн улмаас үхжлийн голомт үүсэхийг ЗЦШ гэнэ

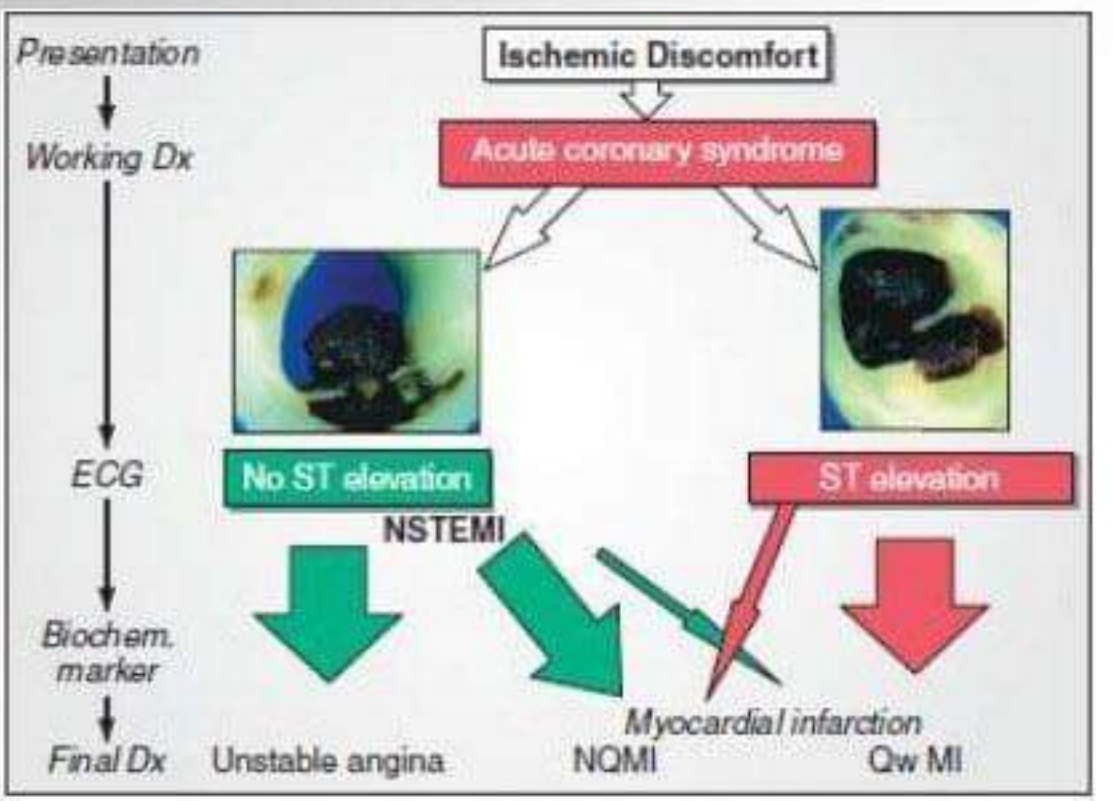
Defined by "Current diagnosis and treatment in Cardiology - 2013"

Definition

- **Acute Coronary Syndrome** is following disruption of a vulnerable plaque, patients experience ischemic discomfort resulting from a reduction of flow through the affected epicardial coronary artery.
  - STEMI
  - NSTEMI
  - Unstable angina

- Defined by: Harrison's Cardiovascular medicine

Acute Coronary Syndrome



Resource: Harrison's Cardiovascular medicine

Causes of Acute Coronary Syndromes

- Atherosclerotic plaque rupture with superimposed thrombus/95%/
- Vasculitic syndromes
- Coronary embolism (e.g., from endocarditis, artificial heart valves)
- Congenital anomalies of the coronary arteries
- Coronary trauma or aneurysm
- Severe coronary artery spasm (primary or cocaine-induced)
- Increased blood viscosity (e.g., polycythemia vera, thrombocytosis)
- Spontaneous coronary artery dissection
- Markedly increased myocardial oxygen demand (e.g., severe aortic stenosis)

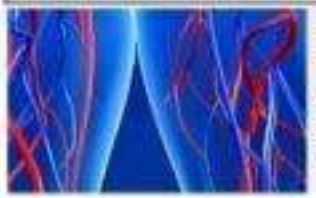
Risk factors

Major independent risk factors	Predisposing risk factors	Possible risk factors
Cigarette smoking	Physical inactivity*	Fibrinogen
Hypertension	Obesity*	C-reactive protein
Elevated total and LDL cholesterol	Family history of premature coronary disease	Homocysteine
Low HDL cholesterol	Ethnicity	Elevated Lp(a)
Diabetes mellitus	Psychosocial factors	
Older age		

- American Heart Association guide to risk factors for coronary artery disease.
- Resource: "Cardiology explained"

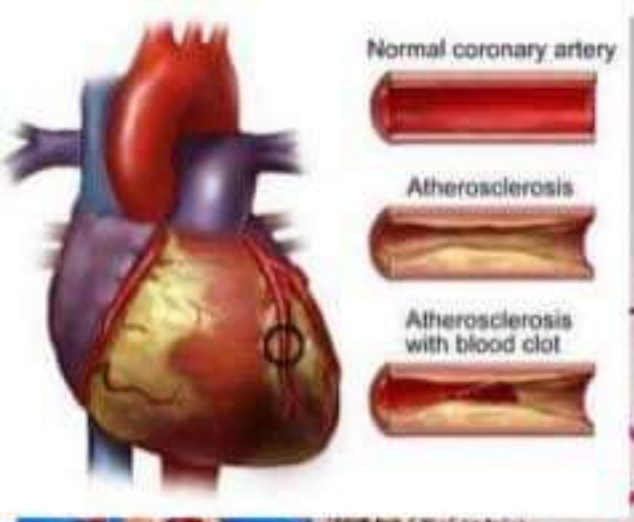
Risk factors

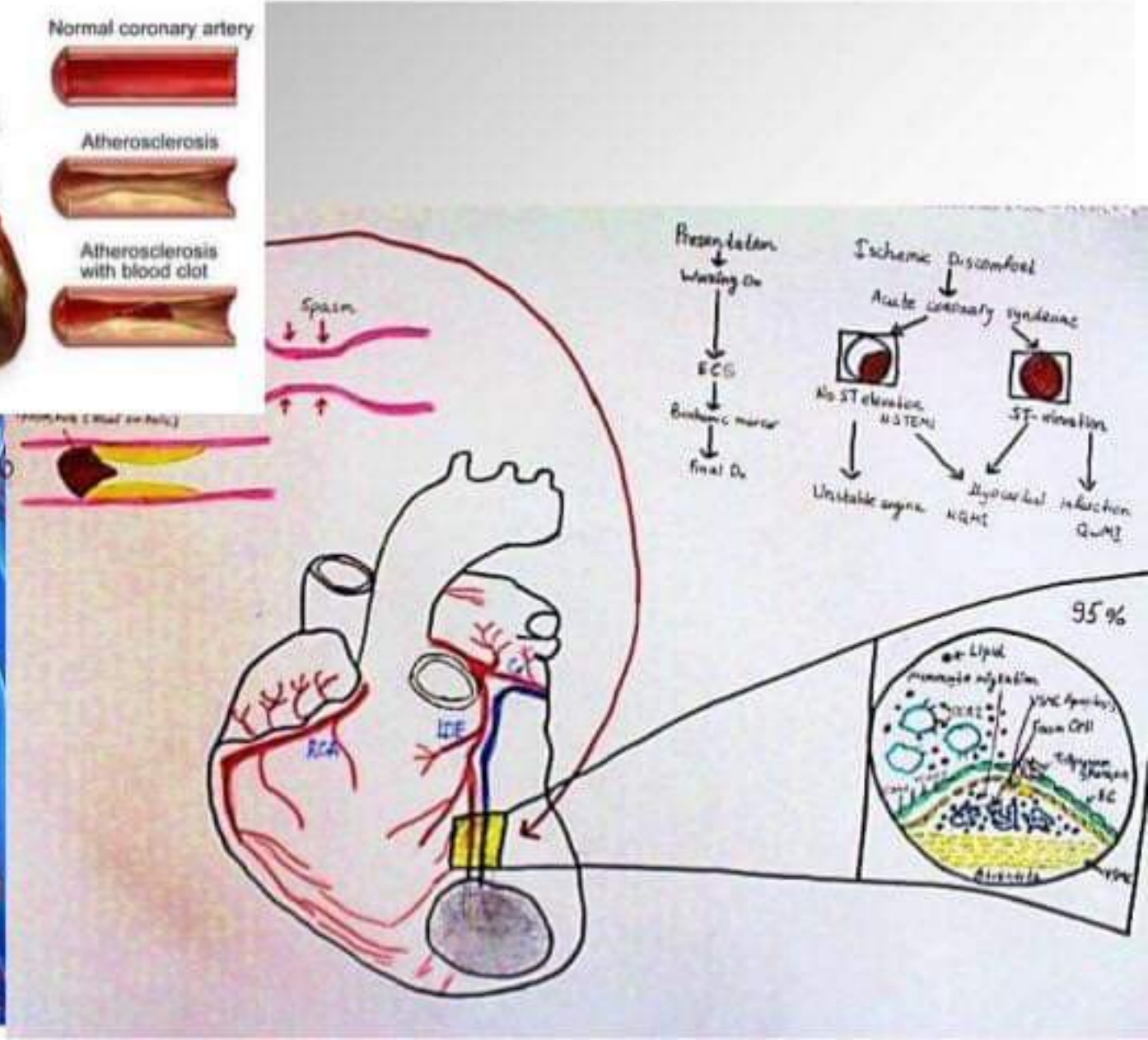
Lifestyle	Biochemical or physiological characteristics (modifiable)	Personal characteristics (nonmodifiable)
Diet high in saturated fat, cholesterol, and calories	Elevated blood pressure	Older age
Tobacco smoking	Elevated plasma total cholesterol (LDL cholesterol)	Male gender
Excess alcohol consumption	Low plasma HDL cholesterol	Family history of CHD or other atherosclerotic vascular disease at early age (men <55 years, women <65 years)
Physical inactivity	Elevated plasma triglycerides	Personal history of CHD or other atherosclerotic vascular disease
	Hyperglycemia/diabetes	
	Obesity	
	Thrombogenic factors	

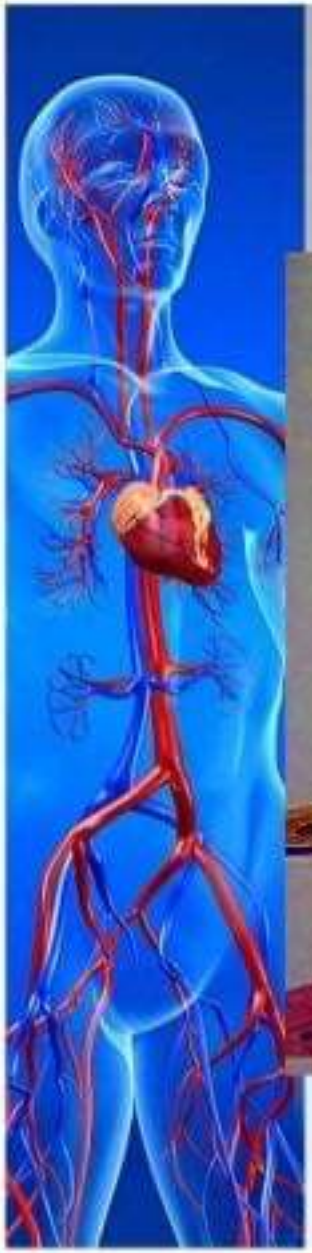


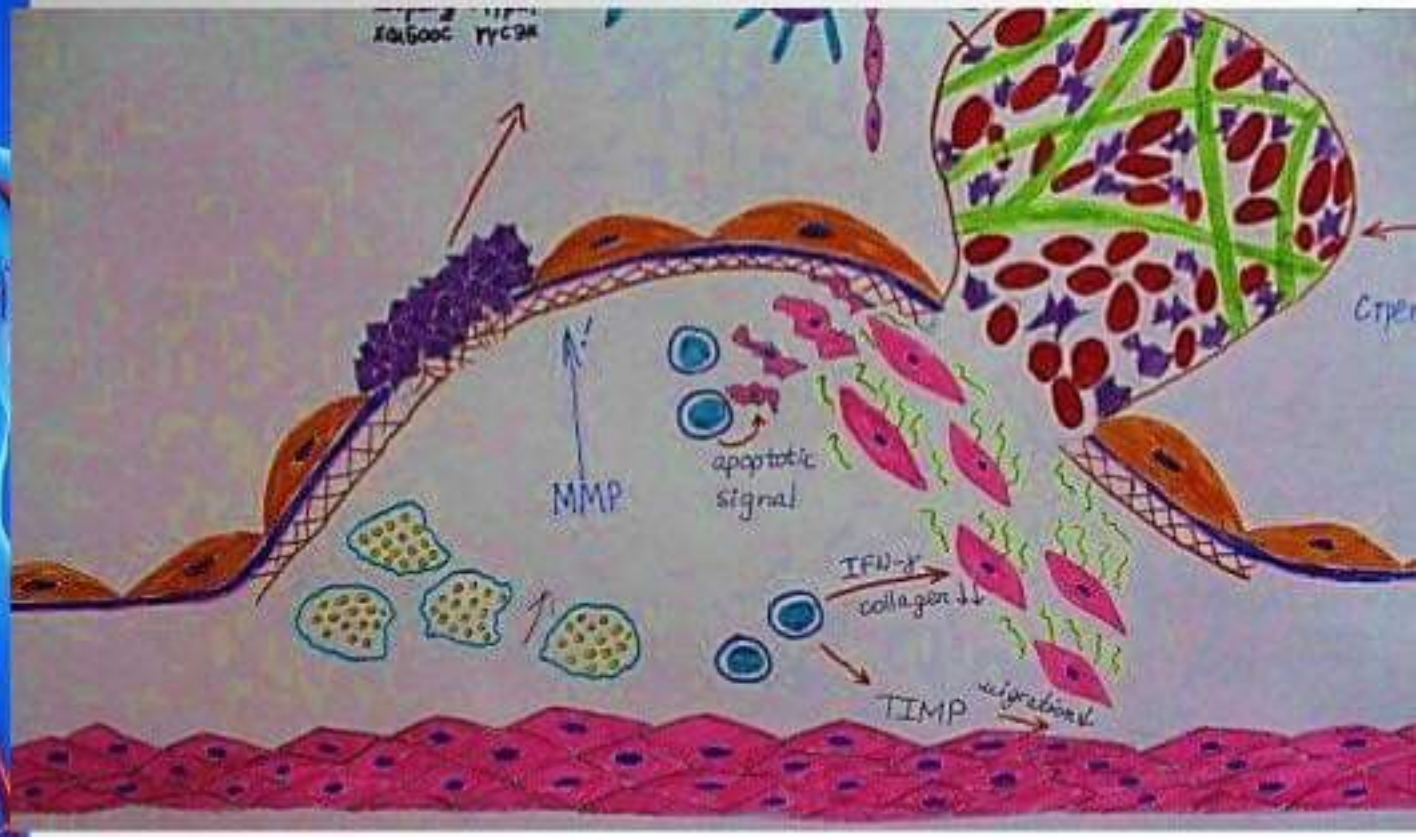
European Society of Cardiology table of lifestyles and characteristics associated with an increased risk of a future coronary heart disease event.
Resource: "Cardiology explained"

PATHOGENESIS

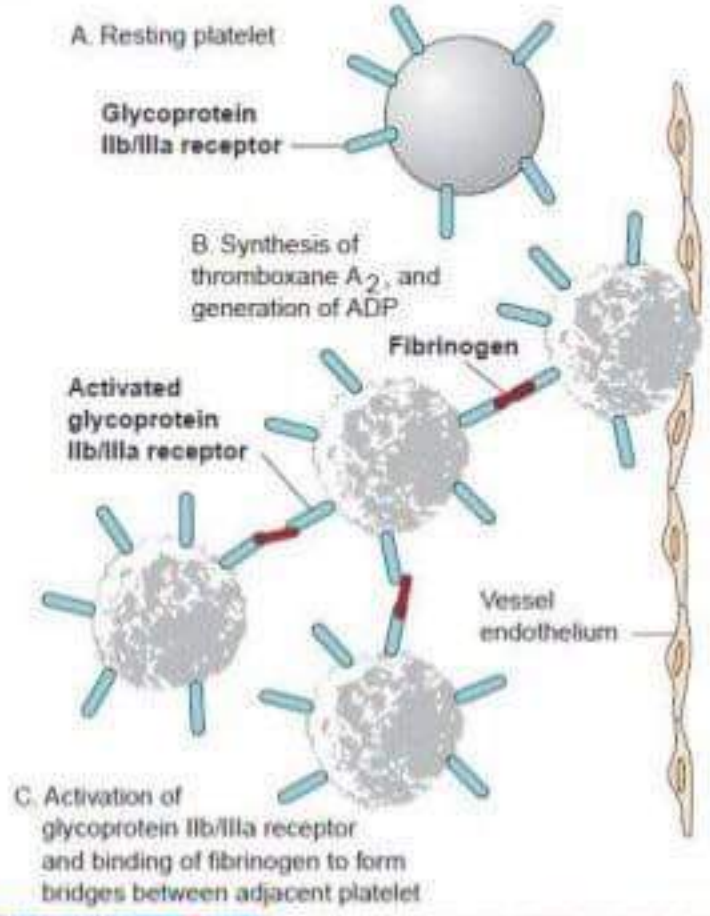

Normal coronary artery
Atherosclerosis
Atherosclerosis with blood clot

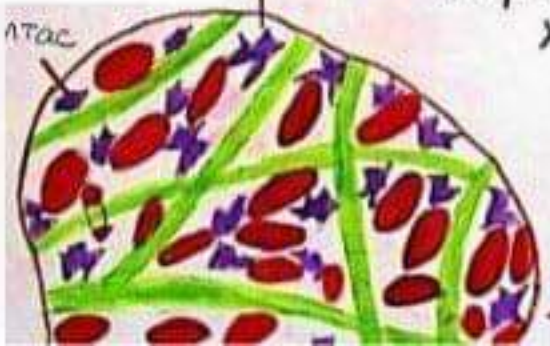

Spasm
Ischemic Discomfort
Acute coronary syndrome
ECG
Biochemic marker
Unstable angina
ST-elevation
95%
Lipid
RCA
LCX
LCE

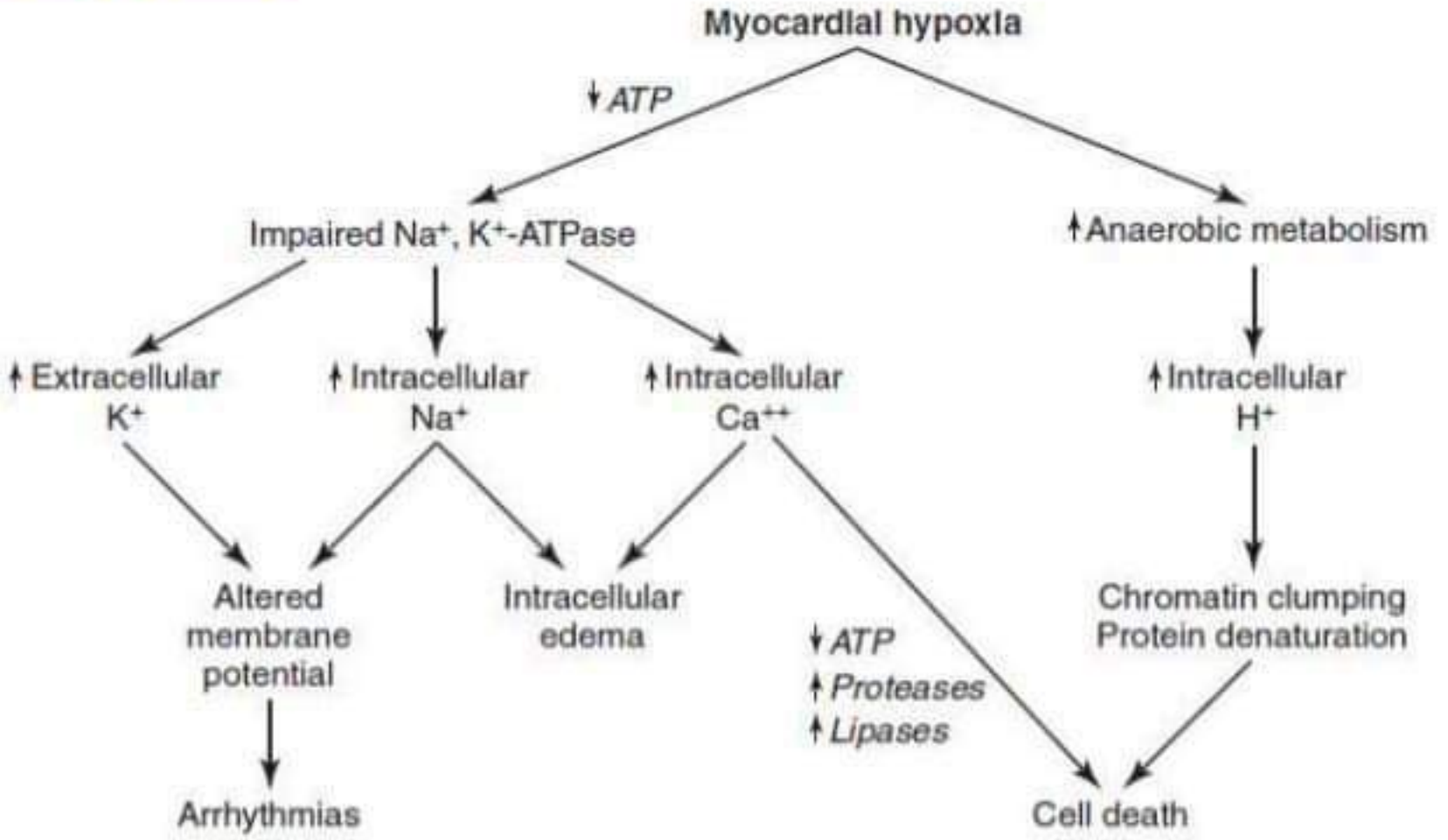



MMP
apoptotic signal
IFN-γ
collagen ↓↓
TIMP

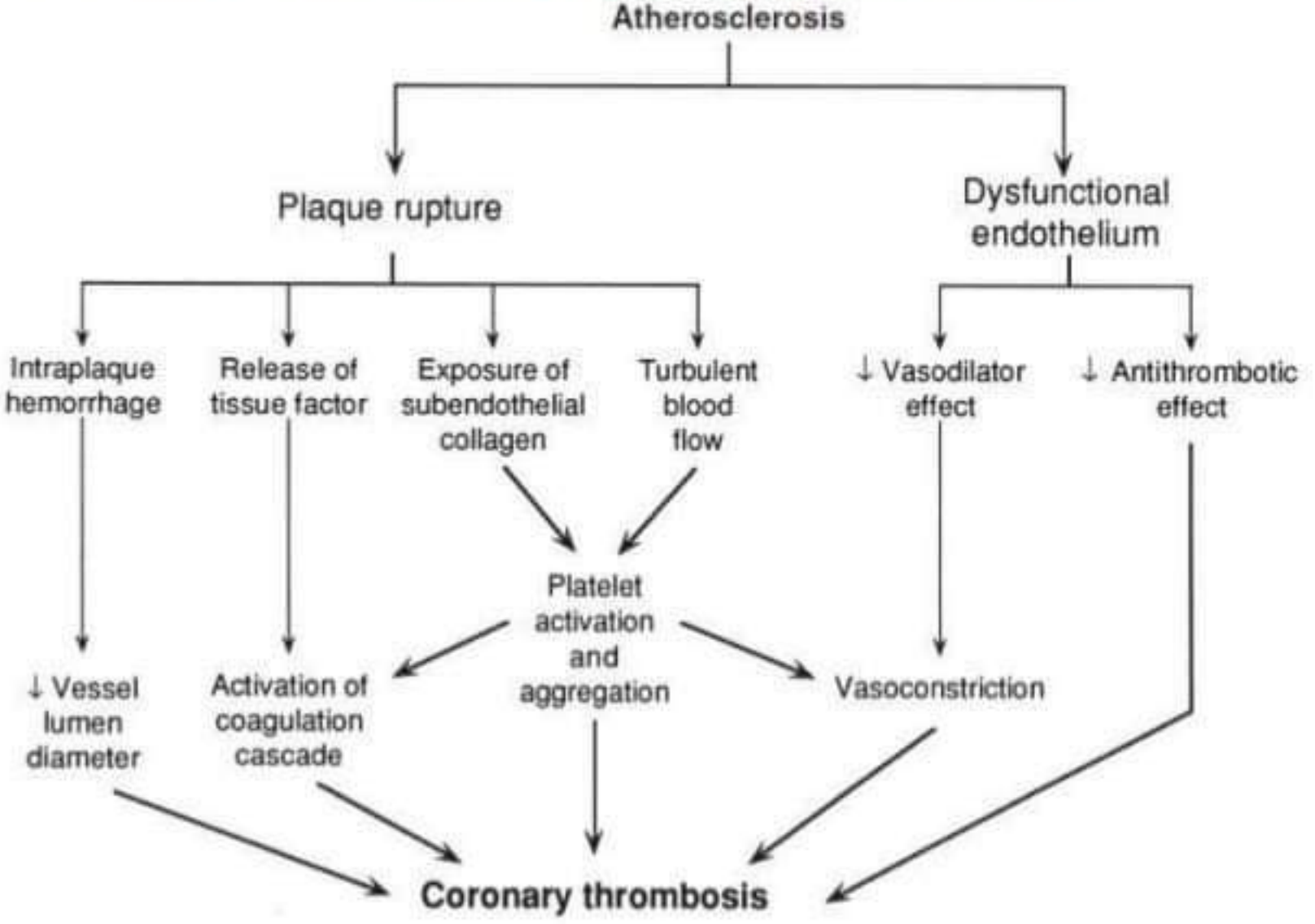
Formation of thrombus






Myocardial hypoxia
↓ ATP
Impaired Na+, K+-ATPase
↑ Anaerobic metabolism
↑ Extracellular K+
↑ Intracellular Na+
↑ Intracellular Ca++
↑ Intracellular H+
Altered membrane potential
Intracellular edema
↓ ATP
↑ Proteases
↑ Lipases
Chromatin clumping
Protein denaturation
Arrhythmias
Cell death

Mechanism of coronary thrombus



CLASSIFICATION

Main

- STEMI
- NSTEMI/Unstable angina

By necrotic area:

- **Micro size** of MI
- **Small size** of MI (LV muscle damage <10%)
- **Moderate size** of MI (LV muscle damaged 10-30%)
- **Large size** of MI (LV muscle >30%)

By progress:

- Initial period: (<6 hours)
- Acute period: (6h – 7 days)
- Recovering period: (7-28 days)
- Convalescence period: (≥29 days)

DIAGNOSIS

- Anamnesis
- Signs and symptoms
- ECG
- Serum analyze
- Echocardiography

Anamnesis:

- Complains
- Onset of disease
- Time
  - initial of disease - calling 103
  - Initial of disease – arrive of a Doctor
- Risks/HTN, Smoking, Diabetes, stress, hereditary/
- Whether having MI, AP, coronary artery by pass graft before that

Symptoms

- By CHEST PAIN:
  - ➢ Typical or classical type of MI
  - ➢ Atypical type of MI
    - Asthmatic
    - Abdominal
    - Low blood pressure
    - Arrhythmatic
    - Brain's

Chest pain of MI

Pain indicators	Description
Location	Behind side of sternum, left side of chest
Radiation	Left arm, jaw, neck
Characterizes	Pressure, dull, squeezing, aching, crushing, burning **/elephant sitting in the chest/**
Duration	>10-20mins
Relieving factor	No abatement of nitroglycerin, relieved with analgesic/morphine/
Associated symptoms	Weakness, dyspnea, fainting fit/syncope/ Cold sweat, apprehensive. Dyspnea, orthopnea, cough, wheezing, nausea and vomiting, or abdominal bloating

Occurs at rest, more commonly in the early morning

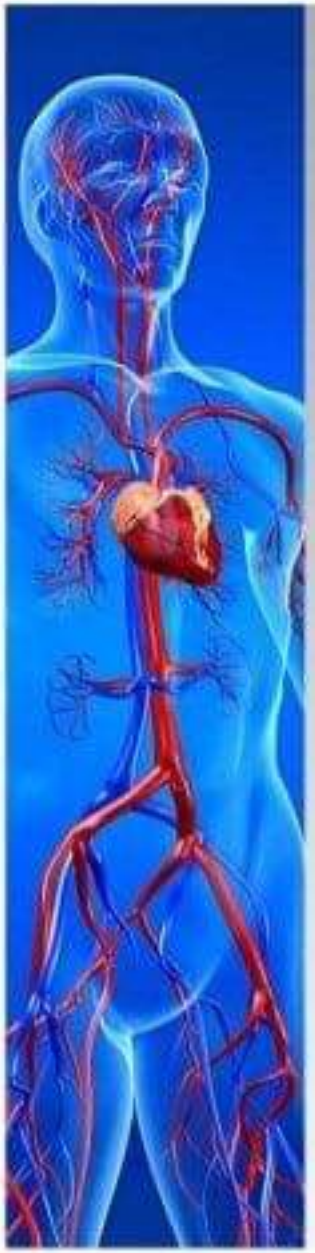
Painless infarction

- One-third of patients with acute myocardial infarction present without chest pain, and these patients tend to be undertreated and have poor outcomes.

- Older patients, women, and patients with diabetes mellitus are more likely to present without classic chest pain. As many as 25% of infarctions are detected on routine ECG without any recallable acute episode.

Signs

- General
- Chest
- Heart
- Extremities

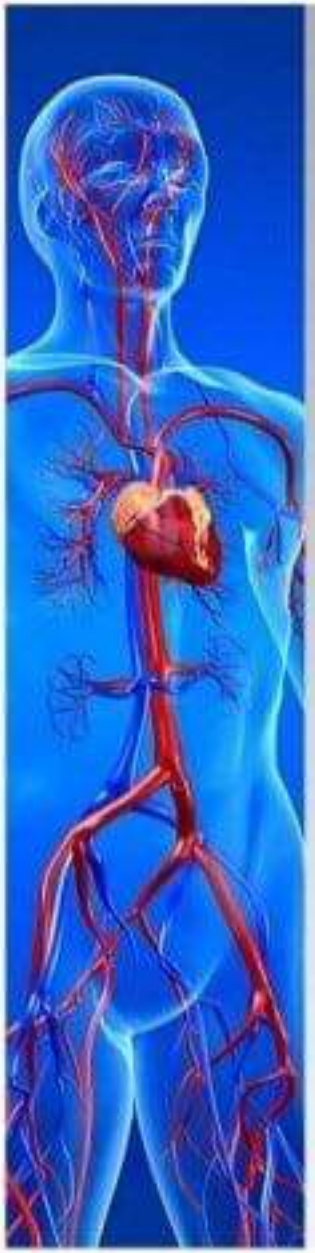


General signs

- Patients may appear **anxious** and sometimes are **sweating profusely**.
- The heart rate may range from marked bradycardia (most commonly in inferior infarction) to tachycardia, **low cardiac output**, or **arrhythmia**.
- The **BP may be high**, especially in former hypertensive patients, or **low** in patients with shock.
- **Respiratory distress** usually indicates heart failure.
- **Fever**, usually low grade, may appear after 12 hours and persist for several days.

Chest

- *The **Killip classification** is the standard way to classify heart failure in patients with acute myocardial infarction and has powerful prognostic value.*
- **Killip class I** is absence of rales and S3
- **Class II** is rales that do not clear with coughing over one-third or less of the lung fields or presence of an S3
- **Class III** is rales that do not clear with coughing over more than one-third of the lung fields
- **Class IV** is cardiogenic shock (rales, hypotension, and signs of hypoperfusion).



Heart

- Jugular venous distention reflects RA hypertension, and a Kussmaul sign (failure of decrease of jugular venous pressure with inspiration) is suggestive of RV infarction. Soft heart sounds may indicate LV dysfunction.
- Atrial gallops (S4) are the rule, whereas ventricular gallops (S3) are less common and indicate significant LV dysfunction. Mitral regurgitation murmurs are not uncommon and may indicate papillary muscle dysfunction or, rarely, rupture. Pericardial friction rubs are uncommon in the first 24 hours but may appear later.

ECG

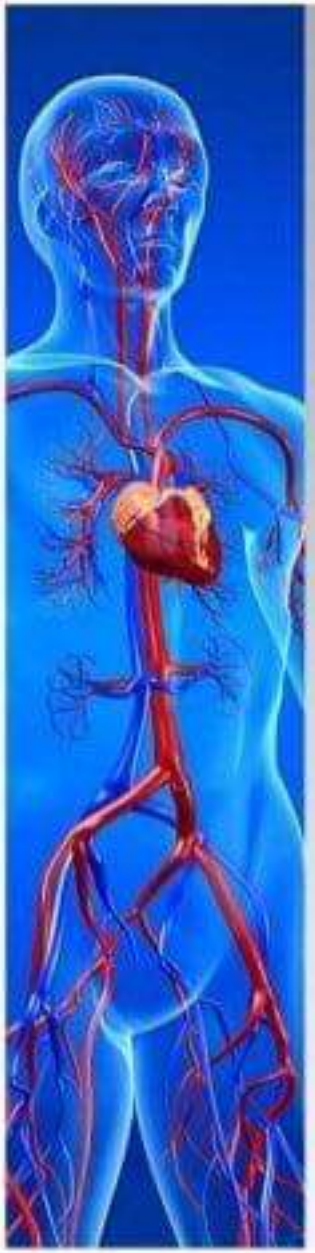
- It should be performed as soon as possible, preferably within 10 minutes, after the patient's arrival in the emergency department or clinician's office, since the presence or absence of ST elevation determines the preferred management strategy.

ECG early changes

- Presence of MI
- QRS complex, ST segment, T waves are changed
- Tall T wave in contiguous 2 or more leads

MI's **specific** changes

- Pathological Q wave present in ≥2 leads
- Pathological Q wave indicate cellular necrosis
- It is generated after beginning of infarction in 8-6 hours
- ST segment's elevation is formed after beginning of infarction in 4-2 hours



Ischemic changes in ECG

- ***ST segment elevation***
- ST segment's elevation (J-point): **elevated in**
  - V2-V3: for men ≥2 mm, for women ≥1,5 mm or
  - contiguous 2 leads for another leads

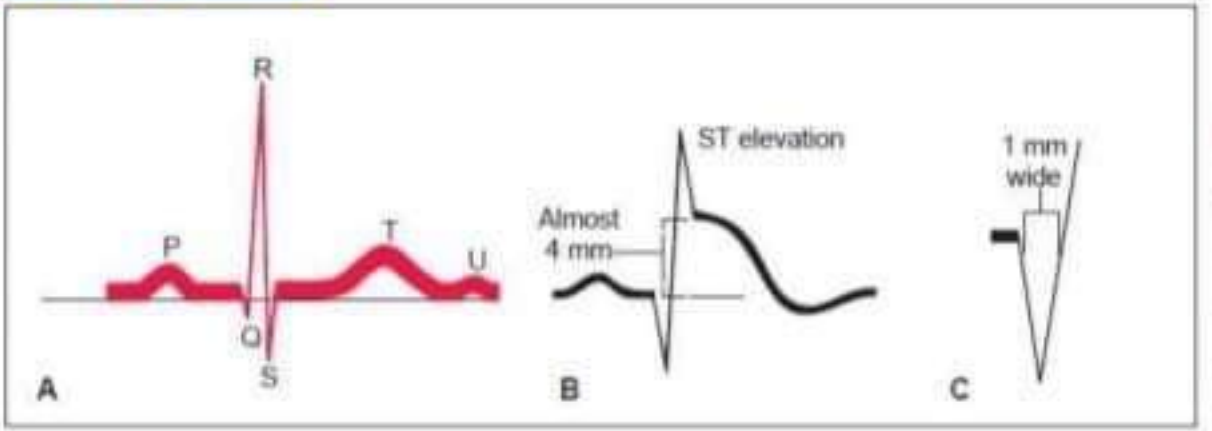
- ***ST segment depression and T wave changes***
- ST segment depressed by horizontal or downward in contiguous 2 leads ≥0,5 cm depressed from isoelectric line, inverted and negative poled(≥1 mm) T wave, more elevated R wave or R/S ratio be >1

- It is positive without LV hypertrophy and LBBB

Goal of ECG during MI

- Confirm or deny DS
- Identify location of infarction
- Identify size of infarction
- Identify time of infarction
- Control outcome of treatment

ECG abnormalities



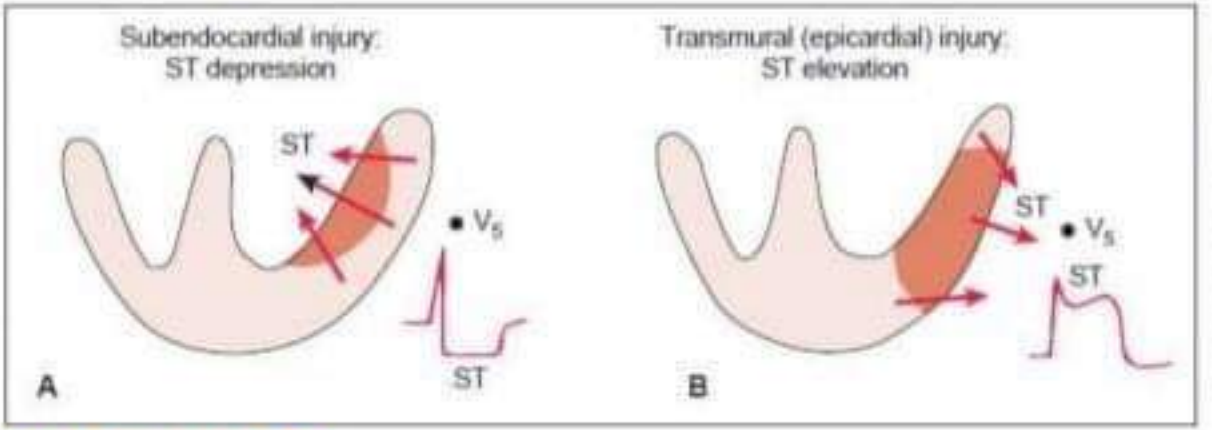


FIGURE 26-10 (**Top**) (**A**) ECG tracing showing normal P, Q,R,S, and T waves. (**B**) ST elevation with acute ischemia. (**C**) Q wave with acute myocardial infarction. (**Bottom**) Current-of-injury patterns with acute ischemia. With predominant subendocardial ischemia (**A**), the resultant ST segment is directed toward the inner layer of the affected ventricle and the ventricular cavity. Overlying leads therefore record ST-segment depression. With ischemia involving the outer ventricular layer (**B**) (transmural or epicardial injury), the ST vector is directed outward. Overlying leads record ST-segment elevation.
(*Bottom* adapted from Braunwald E., Zipes D.P., Libby P. (2002). *Heart disease: A textbook of cardiovascular medicine* (6th ed., p. 108). Philadelphia: W.B. Saunders).

Unstable angina/ NSTEMI

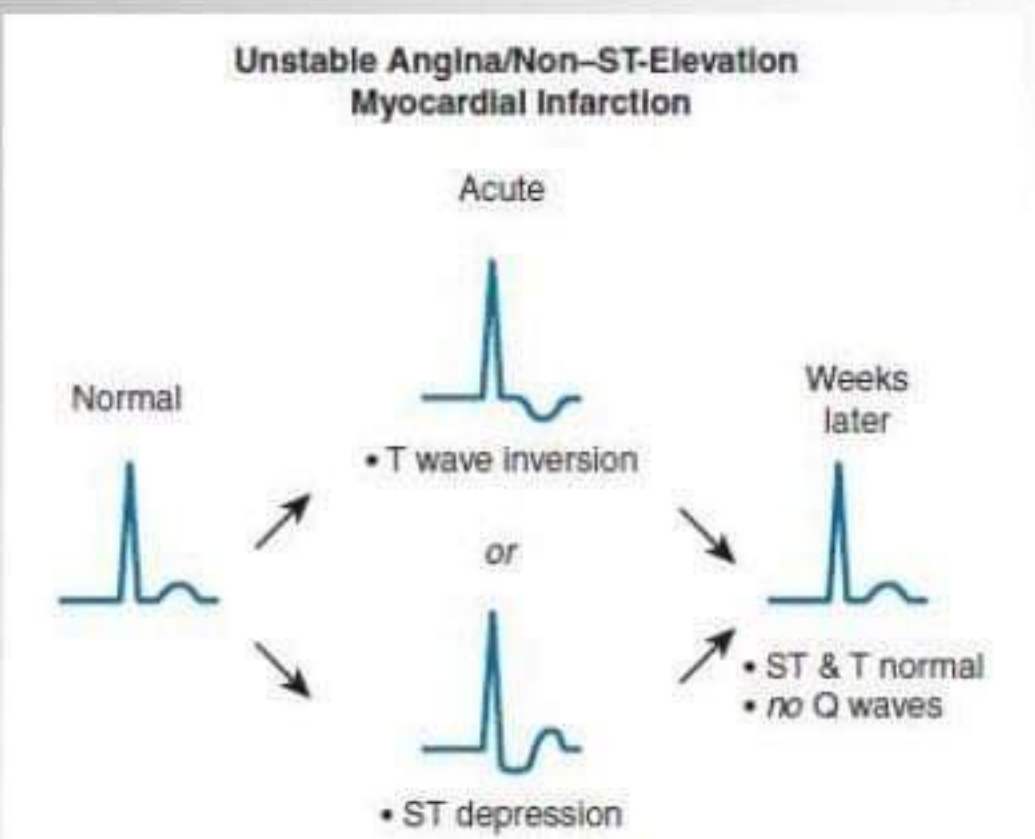


Figure 7.7. ECG abnormalities in unstable angina and non–ST-elevation myocardial infarction.

STEMI

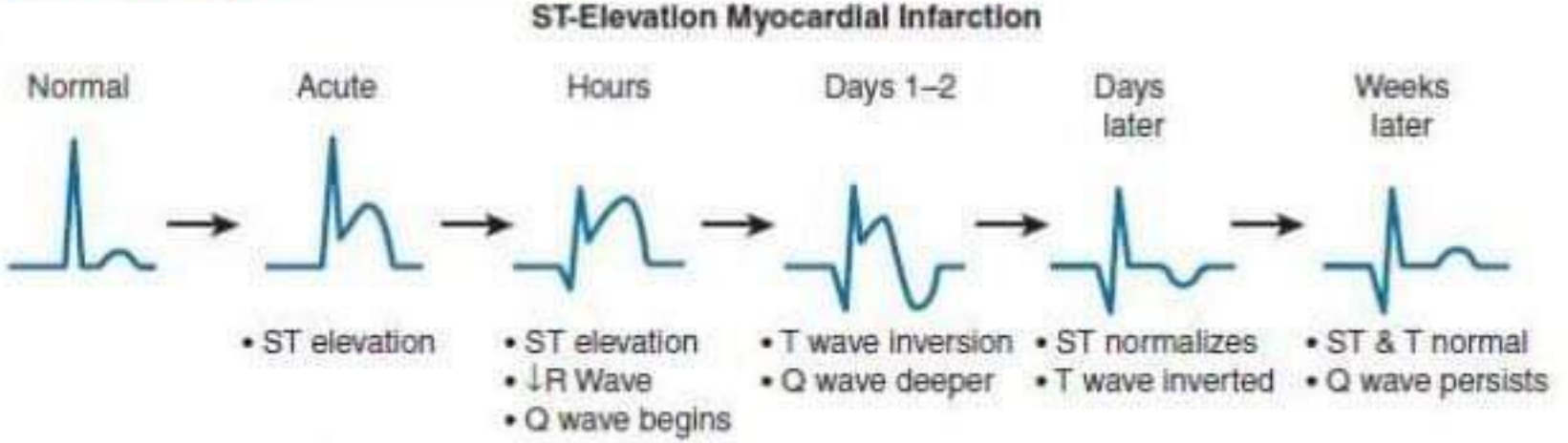
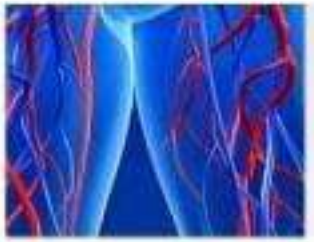


Figure 7.8. ECG evolution during ST-elevation myocardial infarction.



Serum analyze

Indicators:

- Troponin T and I (cTnT, cTnI)
- CK-MB (creatinine phosphokinase myocardial bound)
- Myoglobin
- LDH (Lactate dehydro)

Goal:

- Confirm or deny DS
- Identify size of MI
- Assess result of fibrinolytic treatment
- Diagnosing relapse of MI

Troponin using method for DS of MI

- Result must be ready within 1 hour
- Make second analysis after 6-12 hours if first result is negative
- Negative first result is not enough to reject MI
- Shouldn't make MI diagnose with only Troponin (+)

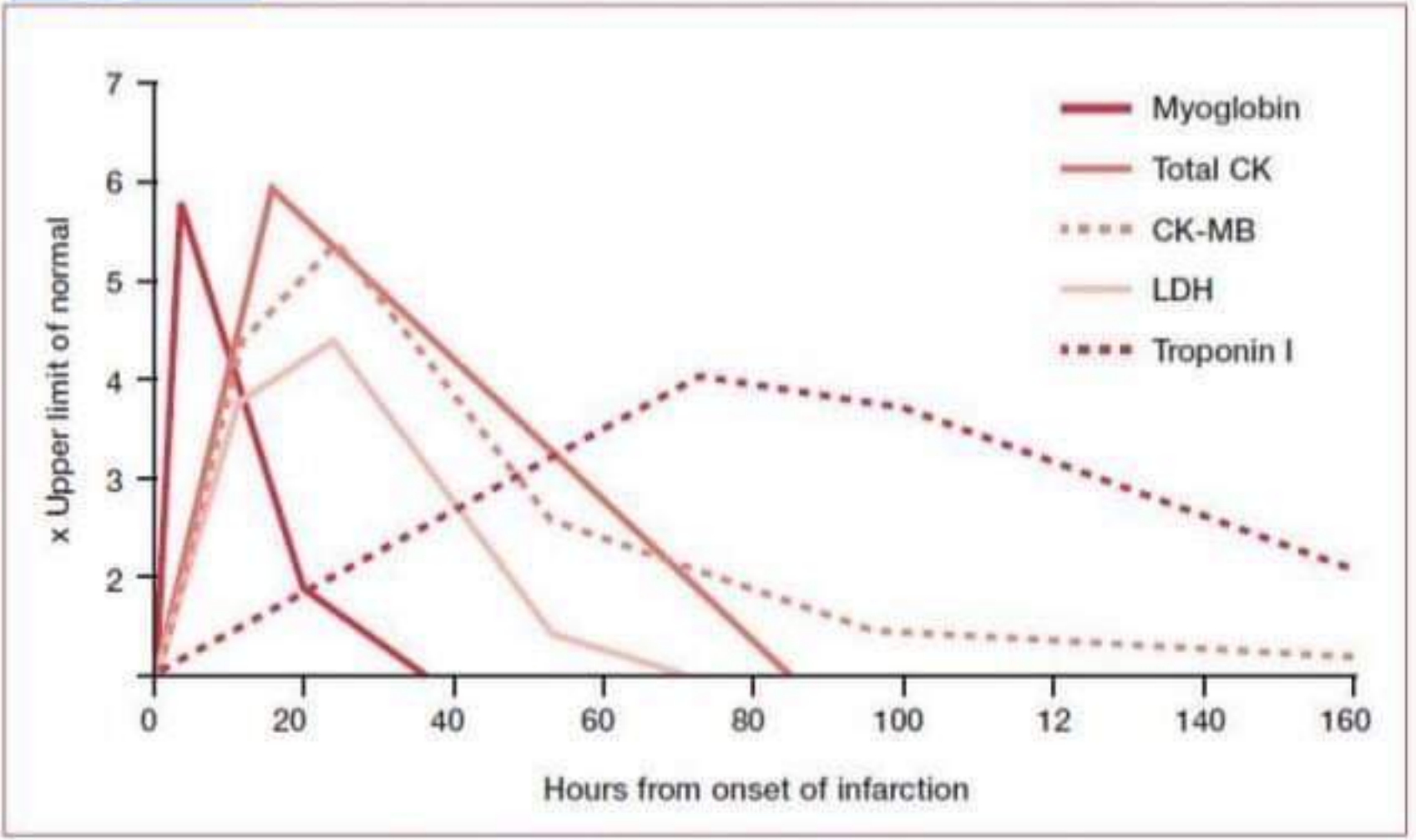


Figure 10. Appearance of cardiac markers in the blood after onset of symptoms. CK: creatine kinase; CK-MB: creatine kinase myocardial band fraction; LDH: lactate dehydrogenase.

Reference intervals

Current metric units × Conversion factor = SI units
SI units ÷ Conversion factor = Current metric units

Test	Specimen	Conventional Units	Conversion Factor	SI Units[2]	Collection[3]
Creatine kinase (CK)	Serum or plasma	32–267 units/L (method-dependent)	0.02	0.53–4.45 mckat/L (method-dependent)	SST, PPT (green)
Creatine kinase MB (CKMB)	Serum or plasma	< 16 units/L or < 4% of total CK (laboratory-specific) Mass units: 0–7 mcg/L	0.04	< 0.27 mckat/L	SST, PPT (green)
Lactate dehydrogenase (LDH)	Serum or plasma	88–230 units/L (laboratory-specific)	0.02	1.46–3.82 mckat/L (laboratory-specific)	SST, PPT (green)
Troponin-I (cTnI)	Plasma	< 0.1 ng/mL (method-dependent)	1.0	< 0.1 mcg/L (method-dependent)	Lavender

Criteria of MI

- Troponin or CK-MB increased with one of these:
  - Chest pain
  - Positive ischemic change in ECG (ST segment and T wave's changes)
  - Pathological Q wave presence in ECG newly
  - Patient had CABG
  - Detected pathological changes in autopsy

Formulation of DS

- Diagnosis must be including **location** of infarction, **type** and **complication**.
- DS: Anterior lateral wall of LV's transmural MI. Pulmonary edema.

General principle of DS

- We should take DS as early as possible. It directly related to treatment result and prognosis.
- Use Diagnosis criteria!
- DS must be including infarction's location, type and complication

Assessment of risk

/patient with MI/

- ✓ Age ≥65 years old
- ✓ * ≥3 risk factors for coronary disease
- ✓ Known coronary stenosis of ≥50% by pre-sentation
- ✓ At least 2 anginal episodes in prior 24 hours
- ✓ Use of aspirin in prior 7 days (i.e., implying resistance to aspirin's effect)
- ✓ Elevated serum Troponin or CK-MB

- 1 score given each question

* - *MI's hereditary anamnesis, HTN, DM, Smoking, Dyslipidemia*

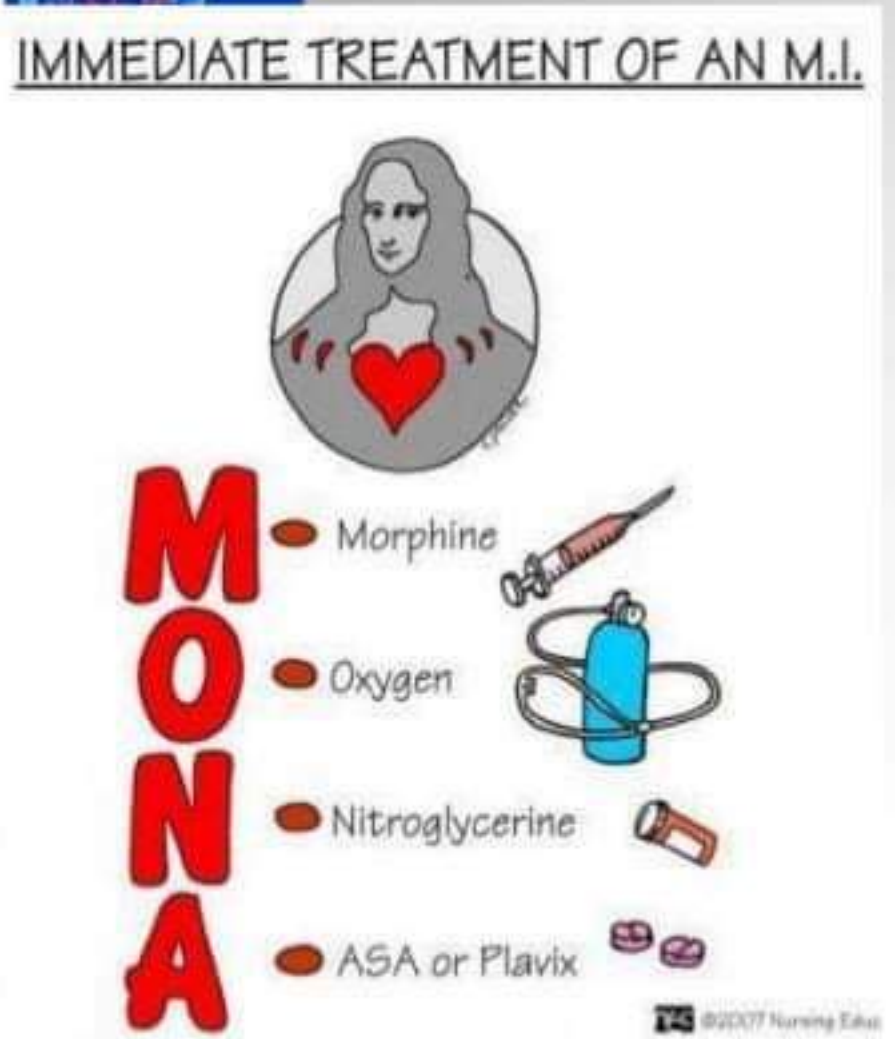
Assessment of risk

- Total score is TIMI /Thrombolysis in myocardial infarction/ assessment's score
- Total scores:
  - 2-1: Low risk
  - 4-3: Moderate risk
  - 7-5: High risk
- TIMI is important for deciding esp. NSTEMI prognosis

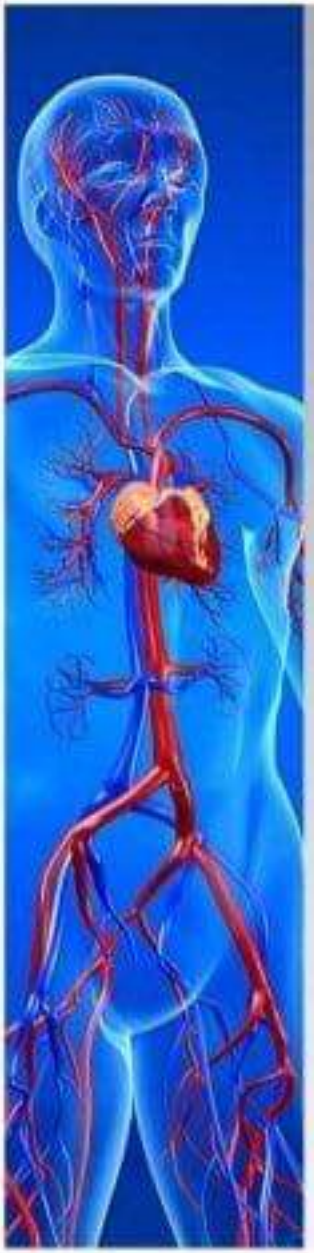
MANAGEMENT

- ✓ It should be successively staged
  - ✓ Pre-hospital management
  - ✓ Emergency department therapy
  - ✓ Post discharge
- ✓ As quickly as begin treatment
- ✓ Correctly choose treatment method
- ✓ Correctly combine treatment methods

Pre-Hospital management



- Chest pain
- Dyspnea
- Restless



Morphine

- Analgesic
- Sedative
- Reducing fear
- Dilate venous → reduce heart burden (important in pul.edema)
- Reduce sympathetic tonus
- 4-8 mg by IV
- Repeat 2-4 mg by IV in 5-15 mins

- Vomiting – Metoclopramide /5-10 mg by IV/
- Hypotension, bradycardia – Atropine /0,5-1 mg by IV/
- Naloxone /0,1-0,2 mg by IV/
- The highest dose of Morphine: 20 mg

Improving heart blood supply

OXYGEN

- 2-8 liter per minute by nasotubule
- Saturation: >90%

NITROGLYCERIN

- Dilating coronary artery
- 0,3-0,6 mg by sublingual or spray
- Repeat 2 times in 5 minute

Pre-Hospital care

Aspirin /ASA/

- 162-325 mg by chew
- If contraindicated:

  Clopidogrel

Fibrinolytic therapy

- Anistreplase/Streptokinase/ Urokinase
- Slowly ejecting by IV />5 minutes/
- If Carrying to hospital requires >30 minutes

Table 5-4. Contraindications for Fibrinolysis Use in STEMI.

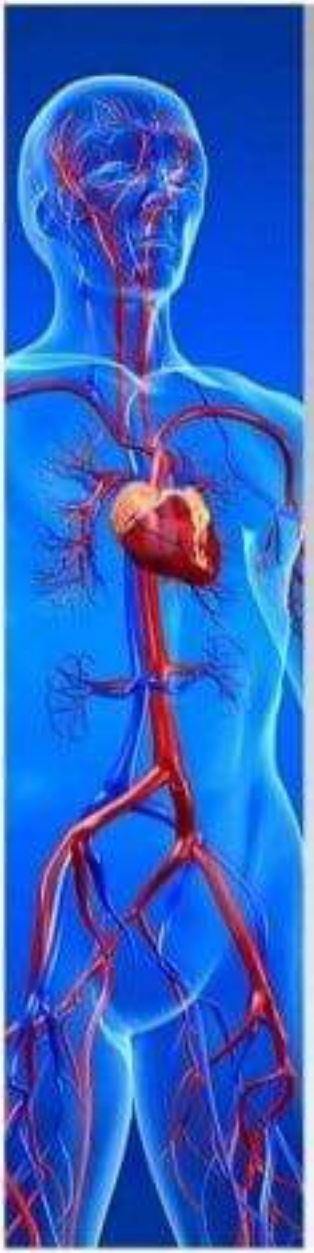
Absolute contraindications
Any prior ICH
Known structural cerebral vascular lesion (eg, AVM)
Known malignant intracranial neoplasm (primary or metastatic)
Ischemic stroke within previous 3 months
Suspected aortic dissection
Active bleeding or bleeding diathesis (excluding menses)
Significant closed head or facial trauma within 3 months
Severe uncontrolled hypertension (SBP > 180 mm Hg and/or DBP > 110 mm Hg)
Relative contraindications
History of prior ischemic stroke greater than 3 months, dementia, or known intracranial pathology not covered in contraindications
Traumatic or prolonged (greater than 10 minutes) CPR or major surgery in previous 3 weeks
Recent internal bleeding (within 4 weeks)
Noncompressible vascular punctures
For streptokinase/anistreplase: prior exposure (more than 5 days ago) or prior allergic reaction to these agents
Pregnancy
Active peptic ulcer
Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

AVM, arteriovenous malformation; CPR, cardiopulmonary resuscitation; DBP, diastolic blood pressure; ICH, intracranial hemorrhage; INR, international normalized ratio; SBP, systolic blood pressure; STEMI, ST elevation myocardial infarction.

Table 5-2. Overview of Management of Acute MI.

Pre-hospital management
Aspirin
Call 911
Continuous cardiac monitoring
Consider pre-hospital 12-lead ECG
Emergency department treatment
Intravenous access
Continuous cardiac monitoring
12-lead ECG
Aspirin
Oxygen
Nitroglycerin
Morphine
Heparin
β-Blocker
Reperfusion strategies
Primary PCI vs fibrinolysis for STEMI
Glycoprotein IIb/IIIa for NSTEMI, followed by elective PCI
In-hospital management
Initial bedrest
Continuous cardiac monitoring
Oxygen for hypoxemia
Nitroglycerin for ongoing pain
ACE inhibitor, β-blocker, aspirin, clopidogrel, statin
Post-discharge
Prognostic indicators
Cardiac rehabilitation
Aggressive secondary prevention with smoking cessation, therapeutic lifestyle changes, and medications

ACE, angiotensin-converting enzyme; ECG, electrocardiogram; NSTEMI, non-ST elevation myocardial infarction; PCI, percutaneous coronary intervention; STEMI, ST elevation myocardial infarction.



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