L1: Acute Coronary Syndrome

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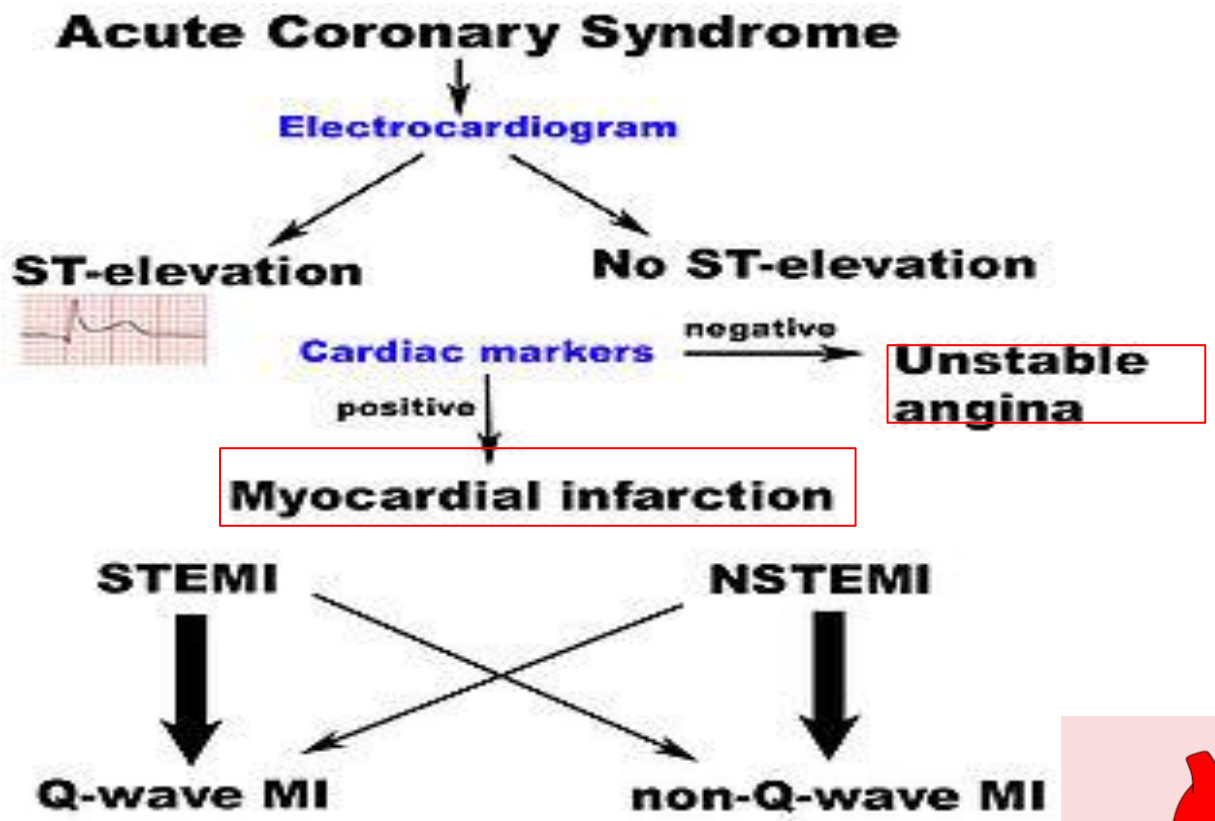
MEDICINE 433

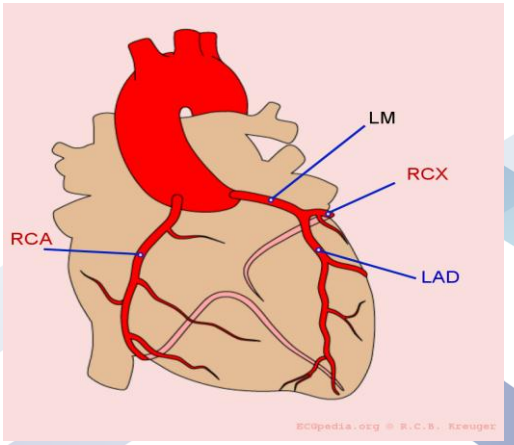
objectives

1. Know the major risk factors for atherosclerosis
2. Understand the pathophysiology of atherosclerosis
3. Know the classification of acute coronary syndrome
4. General approach to assessing an ischemic chest pain
5. General approach to the diagnostic work up of ACS
6. Know the universal definition of myocardial infarction
7. General lines of management of ACS, with emphasis on reperfusion therapy of STEMI including the indication of administrating fibrinolytic therapy, and knowing the absolute contraindication for its use.
8. General understanding of the immediate and late complications of STEMI

Color index: Step up to medicine , slide , Female's note , Male's note , Davidson

Acute coronary syndrome





unstable angina (USA), oxygen demand is unchanged. Supply is decreased secondary to reduced resting coronary flow

MI is due to necrosis of myocardium

Coronary blood flow to a region of the myocardium may be reduced by a mechanical obstruction that is due to:

- Atheroma
- Thrombosis
- Spasm
- Embolus
- Coronary ostial stenosis
- Coronary arteritis (e.g. in SLE).

There can be a decrease in the flow of oxygenated blood to the myocardium that is due to:

- Anaemia
- Carboxyhaemoglobulinaemia
- Hypotension causing decreased coronary perfusion pressure

Pathophysiology of Atherosclerosis

Coronary atherosclerosis is a complex inflammatory process characterized by the accumulation of lipid, macrophages and smooth muscle cells in intimal plaques in the large and medium-sized epicardial coronary arteries.

Two different mechanisms are responsible for thrombosis on the plaques :

The ***first process is superficial endothelial injury, which*** involves denudation of the endothelial covering over the plaque.

The ***second process is deep endothelial fissuring,*** which involves an advanced plaque with a lipid core.

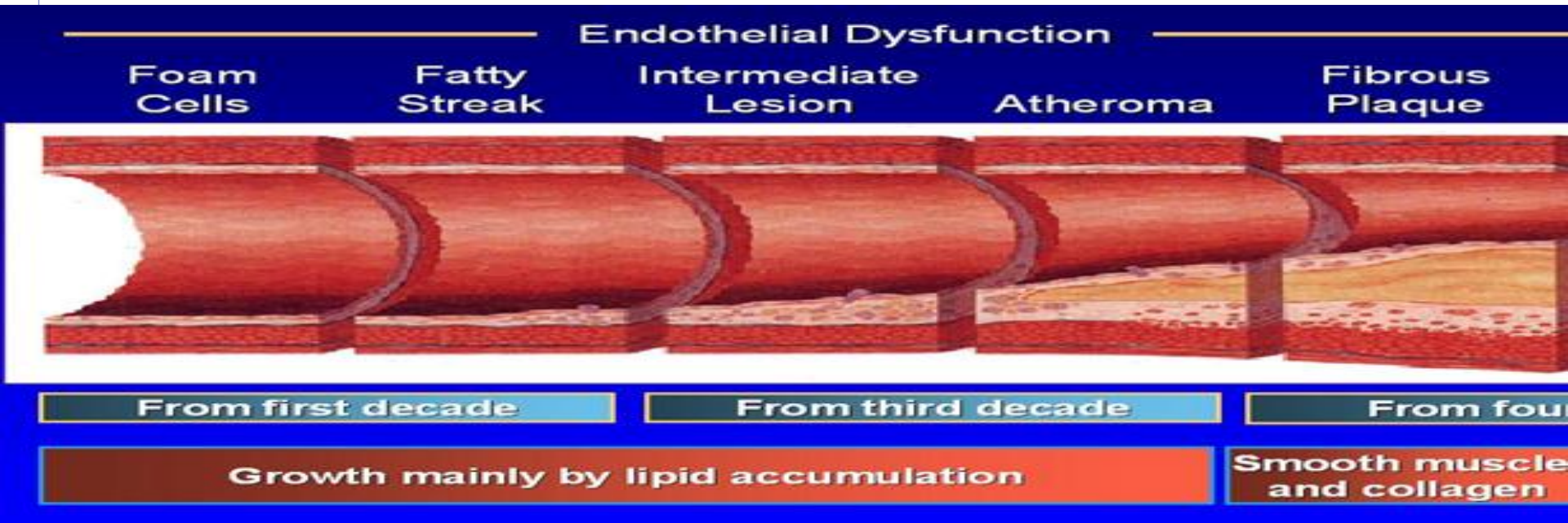
Thrombus forms within the plaque, expanding its volume and distorting its shape.

Thrombosis may then extend into the lumen. A 50% reduction in luminal diameter causes a haemodynamically significant stenosis.

https://www.youtube.com/watch?v=R6QTiBfzULE

Pathophysiology of MI

- The common mechanism to all ACS is rupture or erosion of the fibrous cap of a coronary artery plaque. This leads to platelet aggregation and adhesion, localized thrombosis, vasoconstriction and distal thrombus embolization. The presence of a rich lipid pool within the plaque and a thin fibrous cap are associated with an increased risk of rupture. Thrombus formation and the vasoconstriction produced by platelet release of serotonin and thromboxane A2, results in myocardial ischaemia due to reduction of coronary blood flow.



Risk Factors

Major risk factors:

- Age (men >45 years; women >55 years)
- Hypertension (defined as a systolic blood pressure of ≥140 mmHg,or a diastolic blood pressure of ≥90 mmHg)
- Hyperlipidemia—elevated low-density lipoprotein (LDL)
- Low levels of high-density lipoprotein (HDL)
- Diabetes mellitus
- Cigarette smoking
- Family history of coronary artery disease (CAD) or myocardial infarction (MI)
- Type A personality
- After stroke

Minor risk factors: (less clear significance) include obesity, sedentary lifestyle (lack of physical activity), stress, excess alcohol use

ACS classifications

- 1- ST elevated Myocardial infarction (STEMI)
- 2- Non ST elevated Myocardial infarction (NONSTEMI)
- 3- Unstable angina

Signs & Symptoms of ACS

Symptoms	Physical sign
Prolonged cardiac pain	Sympathetic activation=pallor, sweating
Anxiety	Vagal activation= vomiting, bradycardia
Nausea and vomiting	Impaired myocardial function=hypotension, raised JVP
Breathlessness and collapse	Tissue damage= fever
	Complication= preicarditis, mitral regurgitation

definition of Myocardial Infarction

Typical rise in cardiac troponin T or I , CK-MB with at least one of the following:

1. Ischemic symptoms

2. Pathological Q wave on ECG

3. Ischemic ECG changes (e.g ST elevation or depression, new LBBB)

4. Imaging evidence of new loss of viable myocardium or a new WMA

5. Identification of an intracoronary thrombus by angiography or autopsy.

definition of Myocardial Infarction

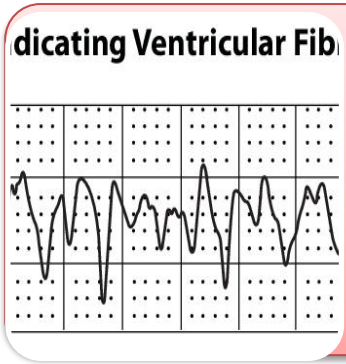
- Myocardial infarction: is when cardiac myocytes die due to prolonged myocardial ischaemia.
- MI is associated with a 30% mortality rate; half of the deaths are prehospital.
- Most patients with MI have history of angina, risk factors for CAD, or history of arrhythmias.

Location of MI	Leads Affected	Vessel Involved	ECG Changes
Anterior wall	V_2 to V_4	• Left Anterior Descending artery (LAD) - Diagonal branch	• Poor R-wave progression • ST-segment elevation • T-wave inversion
Septal wall	V_1 and V_2	• Left Anterior Descending artery (LAD) - Septal branch	• R wave disappears • ST-segment rises • T-wave inverts
Lateral wall	I, aV_L, V_5, V_6	• Left Coronary Artery (LCA) - Circumflex branch	• ST-segment elevation
Inferior wall	II, III, aV_F	• Right coronary artery (RCA) - Posterior descending branch	• T-wave inversion • ST-segment elevation
Posterior wall	V_1 to V_4	• Left Coronary Artery (LCA) - Circumflex branch • Right Coronary Artery (RCA) - Posterior descending branch	• Tall R waves • ST-segment depression • Upright T waves

Signs & Symptoms of MI

Chest pain ,substernal pressure sensation described as (crushing) and elephant standing on my chest

- **Radiation** to neck, jaw or back ,commonly to the left side
- Similar to angina in character and distribution **but much more sever and lasts longer and does not respond to nitroglycerin**



Can be **asymptomatic** in up to one third of patients : painless in postoperative patients,elderly,diabetic patient and women

- **Sudden cardiac death** due to ventricular fibrillation
- Thready pulse, significant hypotension and tachycardia or bradycardia

Other symptom :

dyspnea,diaphoresis,weakness,fatigue,nausea,

vomiting,syncope

How To Diagnose

1-ECG

Markers for ischemia/infarction include:

- **Peaked T waves**: Occur very early and may be missed
- **ST segment elevation** indicates transmural injury and can be diagnostic of an acute infarct.
- **Q waves**: Evidence for necrosis (specific)—Q waves are usually seen late; typically not seen acutely
- **T wave inversion** is sensitive but not specific.
- **ST segment depression**: Subendocardial injury

Categories of infarcts :

- ST segment elevation infarct= transmural involves entire thickness of wall
- non ST segment elevation infarct= subendocardial involves inner one half of the wall

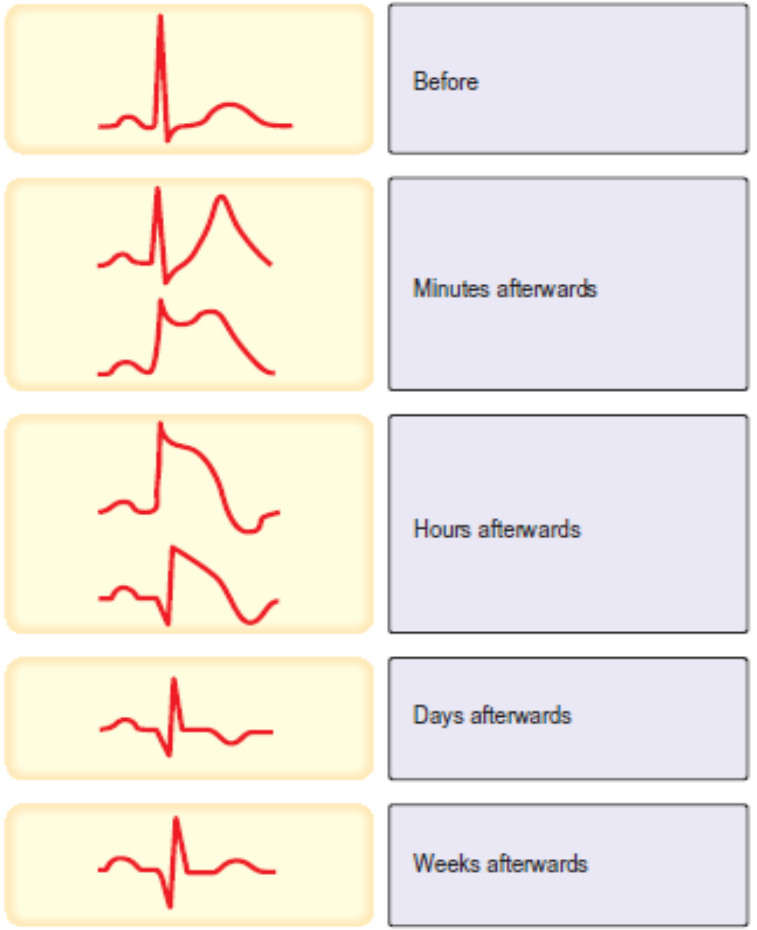


Figure 14.67 Electrocardiographic evolution of myocardial infarction (STEMI). After the first few minutes the T waves become tall, pointed and upright and there is ST segment elevation. After the first few hours the T waves invert, the R wave voltage is decreased and Q waves develop. After a few days the ST segment returns to normal. After weeks or months the T wave may return to

How To Diagnose

2-Cardiac enzymes- gold standard for myocardial injury	3-CBC	4-Chest X-ray	5-Echocardiography
1-Troponins (Troponin I and T)—most important enzyme test to order • Increases within 3 to 5 hours and returns to normal in 5 to 14 days; reaches a peak in 24 to 48 hours • Greater sensitivity and specificity than creatine kinase-MB (CK-MB) for myocardial Injury If the initial troponin assay is negative, then it should be repeated 6–12 h after admission. • **Troponin I** can be falsely elevated in patients **with renal**	**Elevation of** • Leucocystosis • Erythrocyte sedimentation rate (ESR) • C reactive protein (CRP)	**We can find** • Pulmonary oedema that is not evident on clinical examinations • Cardiomegaly due to pre existing myocardial damage	**This is useful for** • assessing ventricular function and for detection important complications
2-CK-MB—less commonly used • Increases within 4 to 8 hours and returns to normal in 48 to 72 hours*; reaches a peak in 24 hours ***used in recurrent infarction*** • When measured within 24 to 36 hours of onset of chest pain, has greater than 95% sensitivity and specificity			

Note

*ischemic pain does NOT change with breathing nor with body position. Also, patients with ischemic pain do not have chest wall tenderness. If any of these are present, the pain is not likely to be due to ischemia

*The combination of substernal chest pain persisting for longer than 30 minutes and diaphoresis strongly suggests **acute MI**

***Right** ventricular infarct will present with inferior ECG changes, hypotension, elevated jugular venous pressure, hepatomegaly, and **clear lungs**

***left** ventricular infarct (tachycardia, third heart sound, **crackles at the lung bases** and elevated jugular venous pressure)

*the distinction between unstable angina and NSTEMI is based entirely **on cardiac enzymes** because both of them lack ST elevation.

- ***Variant (Prinzmetal's) angina* refers to an angina that** occurs without provocation, usually at rest, as a result of coronary artery spasm. It occurs more frequently in women.
- ***Cardiac syndrome X* refers to those patients with a good** history of angina, a positive exercise test and angiographically normal coronary arteries.

* ST segment elevation indicates an infarction 75% of the time. ST segment depression indicates an infarction only 25% of the time.

Management

1.Admit patient to a cardiac monitored floor (CCU) and establish IV access. Give supplemental oxygen and analgesics

2. Medical therapy

3. Reperfusion therapy

a. Fibrinolytic therapy
b. Primary Percutanous coronary intervention (PCI)

4. Revascularization

5. Thrombolytic Therapy

6. Rehabilitation

a. Cardiac rehabilitation is a physician-supervised regimen of exercise and risk factor reduction after MI.
b. Shown to reduce symptoms and prolong survival

2-Medical therapy

Heparin is used for unstable angina and MI (both NSTEMI and STEMI). It is **NOT** used for stable angina

Heparin (enoxaparin)

Prevent progression of thromus

***have been shown decrease the risk of another MI**

Nitrate

*Dilate coronary arteries

*Vasodilator

*Reduce chest pain

Morphine sulfate

analgesia

Statins

Reduce risk of further coronary event

Stabilize plaques and lower cholesterol

ACE inhibitor

*Have been shown to reduce mortality

*Have great benefit with HF patient

B blocker

Block stimulation of HR and contractility

***Have been shown to reduce mortality**

Aspirin (antiplatelet)

Inhibit platelet aggregation

*Have been shown to reduce mortality

1-Aspirin + clopidogrel

2-B blocker

3-ACE inhibitor

Can be used as secondary prevention therapy

*In combination with aspirin we use clopidogrel

*Remember that patients can have a recurrent infarction on same hospital admission

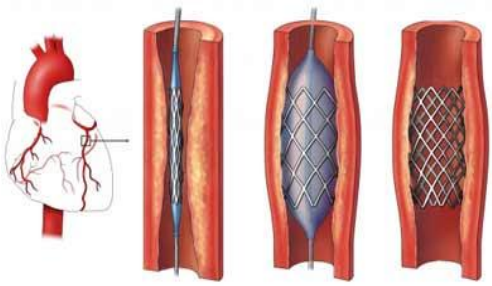
*Aspirin, β-blockers, and nitrates are used for stable angina, unstable angina, STEMI, and NSTEMI

3- Reperfusion therapy

We preferred to use in case of

1-STEMI	2-NSTEMI
-Primary Percutanous coronary intervention (PCI)	**-Primary Percutanous coronary intervention (PCI)**
-Fibrinolytics • Reduces short and long term mortality • Should be given during a 12hr window, and given ASAP. • 2 types of fibrinolytics: 1. Non Fibrin specific (Streptokinase) 2. Fibrin specific Tenecteplase: given as bolus injection, which is easier and has a less margin of dosage error, to dissolve clots)	**We don't use fibrinolytic or thrombolytic therapy here it may be harmful**

4-Revascularization



- Benefit highest when performed early
- Should be considered in all patients
- Revascularization options include 1-PCI, 2-thrombolysis or 3-CABG

1-Percutaneous Coronary Intervention

This is the preferred treatment for STEMI

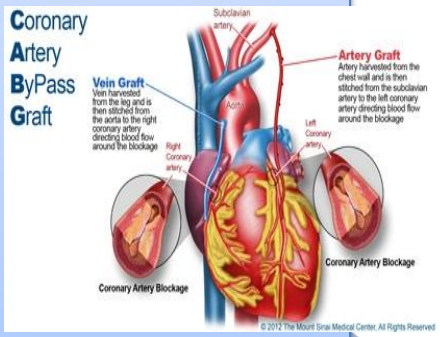
Also preferred in patients with contraindications for thrombolytic therapy; no risk of intracranial hemorrhage

2-Thrombolytic Therapy

Indications: **ST segment elevation** in two contiguous ECG leads in patients with pain onset within 6 hours who have been refractory to nitroglycerin

Absolute **Contraindications** to Thrombolytic Therapy

- Trauma: Recent head trauma or traumatic CPR
- Previous stroke
- Recent invasive procedure or surgery
- Dissecting aortic aneurysm
- Active bleeding or bleeding diathesis



3-Coronary Artery Bypass Grafting

Less often used than the other two in the acute setting.

It remains the **procedure of choice** in patients with severe multivessel disease and complex coronary anatomy.

5-Thrombolytic Therapy

- **remains an important treatment modality** since PCI is still available only at specialized centers. It is useful for patients who present later, and for those in whom PCI is contraindicated.

• Early treatment is crucial to salvage as much of the myocardium as possible. Administer as soon as possible up to 24 hours after the onset of chest pain. Outcome is best if given within the first 6 hours.

• Alteplase has been shown to have the best outcomes amongst thrombolytic medications, and is the first choice in many centers, despite its high costs. Alternatives include streptokinase, tenecteplase, reteplase, lanoteplase, and urokinase.

•the main reason to initiate therapy with thrombolytics/angioplasty is whether there **is ST segment elevation on ECG.**

- **Note:** in STEMI, The pain does not usually respond to sublingual GTN, and opiate analgesia is required (morphin)

The main adverse effect "risk" of Thrombolytic therapy is **bleeding** and the worst bleeding is in the brain

Relative contraindications to thrombolytic: potential candidates for PCI

- Active internal bleeding or intracerebral hemorrhage
- Recent surgery or trauma
- Uncontrolled hypertension or pregnancy
- Active peptic ulcer

Complications

- Pump failure (CHF)
- Arrhythmias

1- ventricular fibrillation: the major cause of death and defibrillation restores sinus rhythm.

2- atrial fibrillation: if it causes rapid ventricular rate with hypotension prompt cardioversion synchronised DC shock and it is often a feature of impending left ventricular failure.

3- bradycardia

- Acute circulatory failure
- Recurrent infarction
- ischaemia
- Mechanical complications

1- rupture of the papillary muscle can cause acute pulmonary oedema and shock due to sever mitral regurgitation.

2- rupture of interventricular septum causes left to right shunting and patient may develop right heart failure

3- rupture of the ventricle may lead to cardiac tamponade

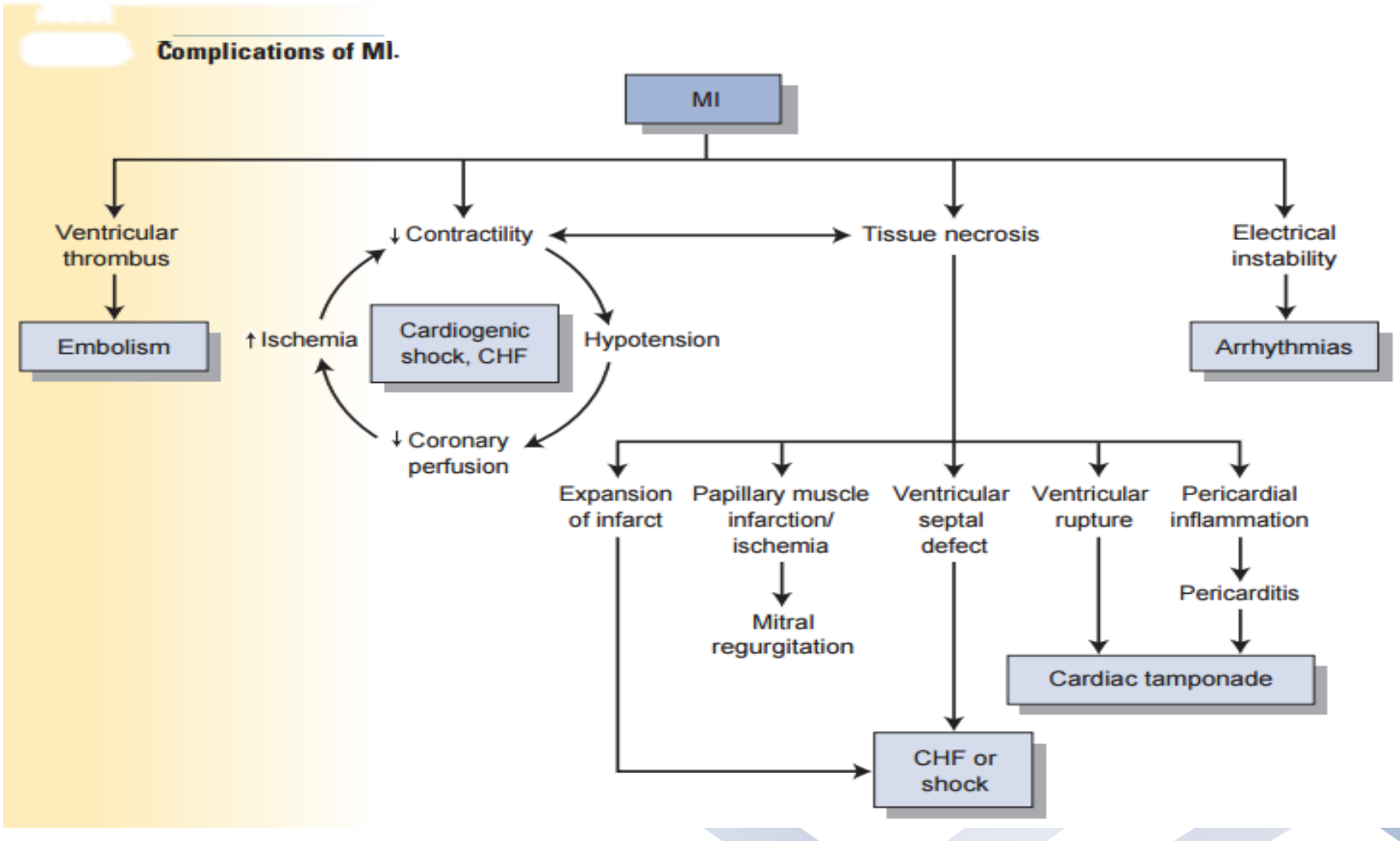
- Acute pericarditis
- Embolism

Forms on endocardial surface and causes stroke, ischemic limb, venous thrombosis and pulmonary embolism.

- Dressler's syndrome

("post myocardial infarction syndrome") a. Immunologically based syndrome consisting of fever, malaise, pericarditis, leukocytosis, and pleuritis, occurring weeks to months after an MI. The treatment is: aspirine,NSAIDs and corticosteroids

Complications



Prognosis

- In almost ¼ of all cases of MI death occurs within **few minutes without medical care** and ½ of death occur within 24 hours of the onset of symptoms.
- Patients with **unstable angina** have a mortality of approximately half that of those patients with **MI.**
- Early deaths usually due to an **arrhythmia.**
- **The** prognosis **is worse** for anterior than for inferior

MCQs

1- Your 60-year-old male patient, followed for chronic stable angina on aspirin, nitrates, and a beta blocker, presents to the ER with history of two to three episodes of more severe and long- lasting angina chest pain each day over the past three days. His ECG and cardiac enzymes are normal. The best course of action of the following is to

A. Admit the patient and begin intra- venous digoxin
B. Admit the patient and begin intra- venous heparin
C. Admit the patient and give prophylactic thrombolytic therapy
D. Admit the patient for observation with no change in medication
E. Discharge the patient from the ER with increases in nitrates and beta blockers

2-The classic presentation of acute myocardial infarction (MI) involves heavy or crushing sub sternal chest pain. The type of patient most likely to present with pain-less, or silent, MI is the

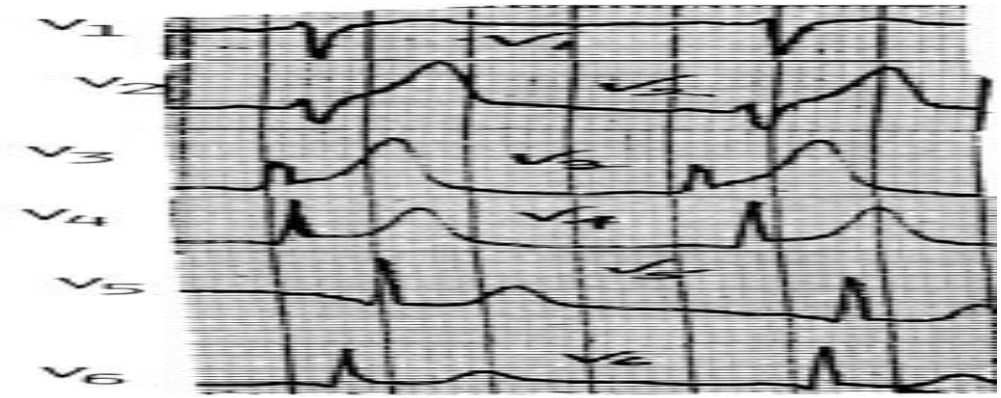
A. Advanced coronary artery disease patient with unstable angina
B. Elderly diabetic
C. Premenopausal female
D. Inferior MI patient
E. MI patient with PVCs

1-B 2-B

MCQs

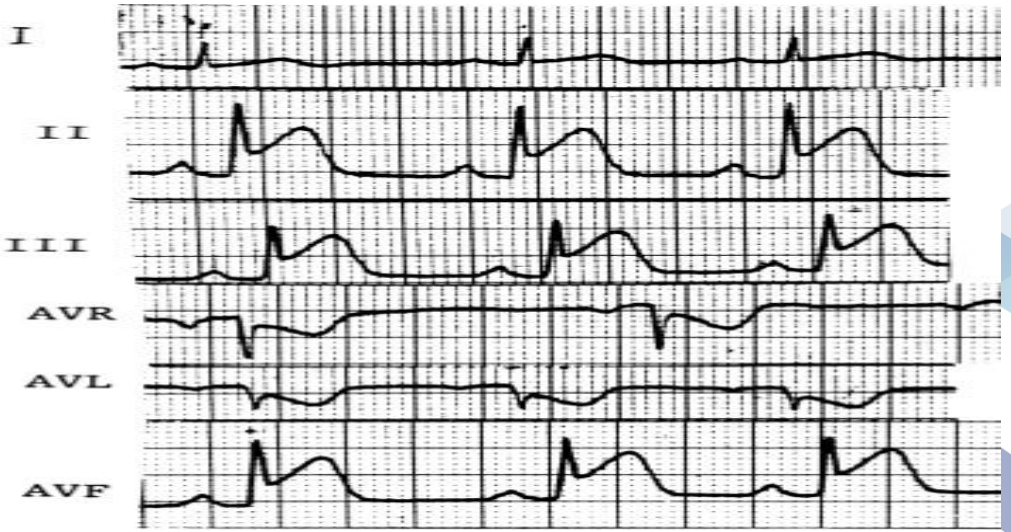
3-A 48-year-old male with a history of hypercholesterolemia presents to the ER after one hour of sub sternal chest pain, nausea, and sweating. His ECG is shown at right. There is no history of hyper- tension, stroke, or any other serious illness. Which of the following therapies would be inappropriate?

A. Aspirin
B. Beta blocker
C. Morphine
D. Digoxin
E. Nitroglycerin
F. Thrombolytic agent



4-A 55-year-old obese woman develops pressure-like sub sternal chest pain of one-hour duration. Her ECG is shown at right. The most likely diagnosis is

A. Acute anterior myocardial infarction
B. Acute inferior myocardial infarction
C. Pericarditis
D. Esophageal reflux
E. Cholecystitis



3-D 4-B

MCQs

5-Two-hour history of chest pain; acute ST-segment elevation in leads 2, 3, and AVF: sinus bradycardia at rate of 40 with hypotension. Which of the following is a best for management

A. Digoxin
B. Propranolol
C. Calcium channel blocker
D. Atropine
E. Heparin

6-You have been asked to evaluate a 42-year-old white male smoker who presented to the emergency department with sudden onset of crushing sub sternal chest pain nausea, diaphoresis, and shortness of breath His initial ECG revealed ST segment elevation in the anteroseptal leads. Cardiac enzymes were normal. The patient underwent emergent cardiac catheterization, which revealed only a 25% stenosis of the left anterior descending (LAD) artery No percutaneous intervention was performed Which of the following interventions would most likely reduce his risk of similar episodes in the future?

A. placement of a percutaneous drug eluting coronary artery stent
B. Placement of a percutaneous non-drug eluting coronary artery stent
C. Beginning therapy with an ACE inhibitor
D. Beginning therapy with a beta blocker
E. Beginning therapy with a calcium channel blocker

5-D 6-E

MCQs

7- yesterday you admited a 55 year old man to the hospital for an episode of chest pain. The patient has past medical history of COPD ,peripheral vascular disease with claudication hypertension and hypercholesterolemia .On admission his BMI 40 there is bilateral wheezing and cardiac examination reveals a grade 1/6 early Systolic murmur at upper left sternal border without radiation . Blood pressure readings have consistently been 140/90 to 150/100. Cardiac enzyme are normal a resting ECG shows left ventricular hypertrophy with secondary ST-T wave change (LVH with strain) you decide to do a cardiac stress test on this patient which cardiac stress test would be most appropriate for him?

A. Exercise EKG stress test
B. Exercise nuclear stress test
C. Pharmacologic nuclear stress test with adenosine
D. Pharmacologic nuclear stress test with dipyridamole
E. Pharmacologic echo stress test with dobutamine

A70 year old man with a history of coronary artery disease presents to emergency department with 2 hours of substernal pressure ,diaphoresis and nausea. He reports difficulty "catching his breath". An ECG shows septal T wave inversion .The patient is given 325 mg aspirin and sublingual nitroglycerin while awaiting the result of his blood work .his troponin I is 0.65 ng/ml(normal <0.04 ng/ml) . The physician in the ER start the patient on low-molecular weight heparin. His pain is 3/10. Blood pressure is currently 154/78 and heart rate is72 . You are asked to assume care of this patient. What is the best rate next step in management?

A. Arrange for emergent cardiac catheterization.
B. Begin intravenous thrombolytic therapy.
C. Admit the patient to a monitored cardiac bed and repeat cardiac enzymes and ECG in 6 hours.
D. Begin intravenous beta-blocker therapy.
E. Begin clopidogrel 75 mg po each day

7-D 8-D

DONE BY

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Medicine is a science of uncertainty and an art of probability