



## Lecture 3

# Ischemic Heart Diseases (IHD)



- [http://youtu.be/afYCN3Upy\\_w](http://youtu.be/afYCN3Upy_w) acute coronary syndrome
- <http://youtu.be/Bnoo5insrUQ> aneurysm

# OBJECTIVES

- **Be able to discuss pathology and complications of ischemic heart diseases with special emphasis on myocardial infarction.**
- **Know how lifestyle modifications can reduce the risk of ischemic heart disease.**
- **Macroscopic and microscopic changes in myocardial infarction.**
- **Biochemical markers of myocardial infarction.**
- **Complications of myocardial infarction: immediate and late.**

## Ischemic Heart Diseases (IHD)

OR

## Coronary artery disease (CAD)

**Definition:** A group of related syndromes resulting from myocardial ischemia ( An imbalance between cardiac blood supply and myocardial oxygen demand).

### Causes of Ischemic Heart Disease:

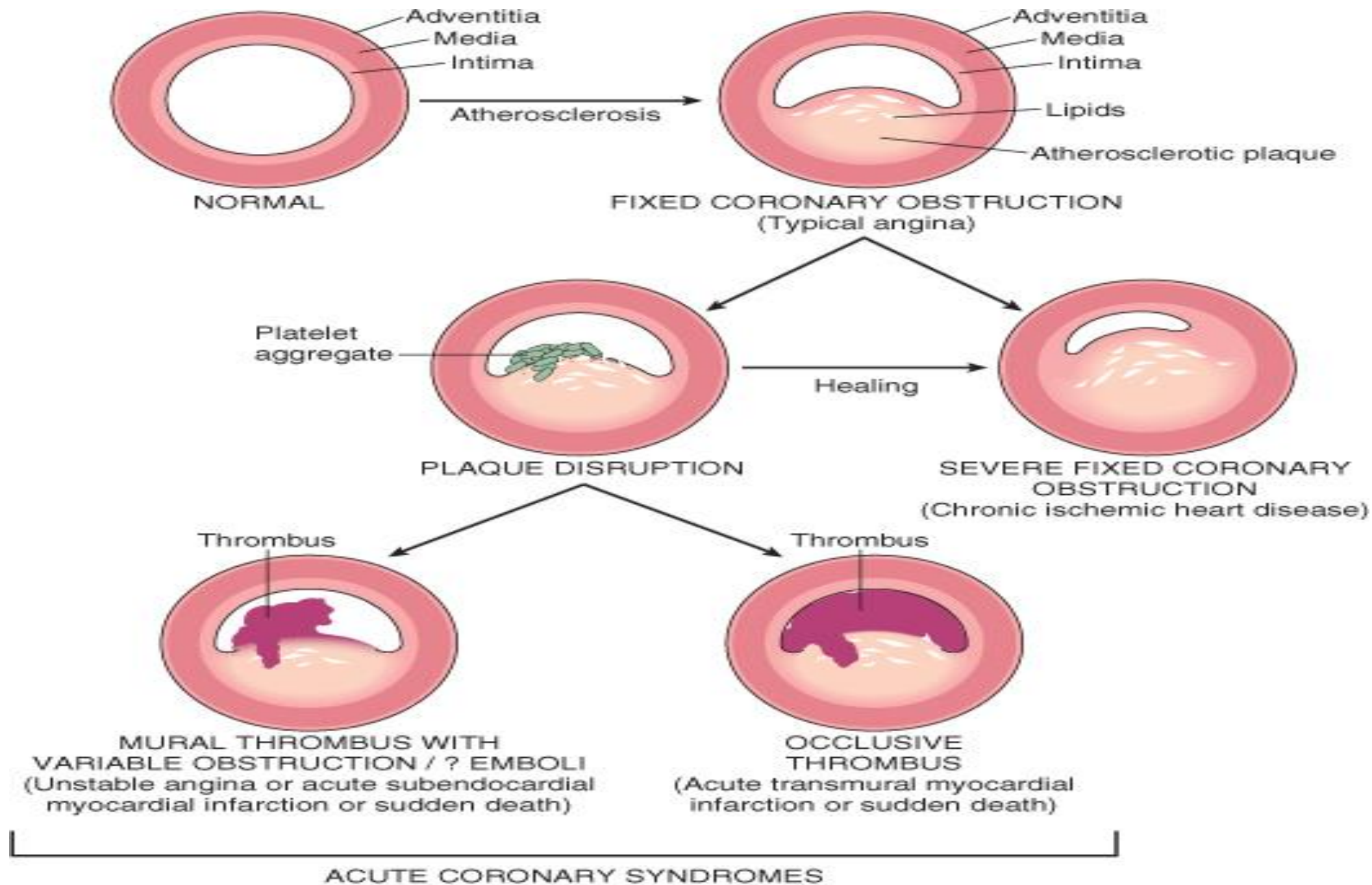
- Coronary artery atherosclerosis (**Major causes**)
- Less frequency Vasospasm and Vasculitis

### Clinical presentation of IHD may include one or more of the following cardiac syndrome:

- 1- Angina pectoris
- 2- Myocardial infarction (**MI**)
- 3- Chronic IHD with CHF
- 4- Sudden cardiac death (**SCD**)

\* Weight loss, quit smoking, aerobic exercises, decrease stress, and avoiding the consumption of unsaturated fats, can reduce risk of IHD

**Note:** Estrogen protects women against IHD, so the men are more affected than women



# 1- Angina pectoris

**Definition:** An intermittent chest pain caused by transient, reversible myocardial ischemia, the pain Can radiate down the left arm or to the left jaw.

## Types:

<b>Stable angina</b> (typical angina)	<ul style="list-style-type: none"><li>- Occurring after certain levels of <b>exertion</b>.</li><li>- Due to <u>atherosclerotic disease</u> with (<b>critical stenosis</b>) fixed chronic stable stenosis.</li><li>- Is usually relived by rest , thereby decreasing demand , nitroglycerin or a strong vasodilator.</li></ul>
<b>Unstable angina</b> (crescendo angina)	<ul style="list-style-type: none"><li>- Often is harbinger of <b>MI</b>.</li><li>- Occurring with less exertion or even <b>at rest</b> with <b>more prolonged duration</b>.</li><li>- Associated with plaque disruption triggering by (Platelet aggregation, Vasoconstriction, Formation of mural thrombus).</li></ul>
<b>Variant angina</b> (Prinzmetal angina)	<ul style="list-style-type: none"><li>- Occurs <b>at rest</b>, and caused by <b>coronary artery spasm</b>. (the aetiology is not clear)</li><li>- <b><u>Not related to atherosclerosis</u></b> .</li><li>- Responds promptly to vasodilators, such as (<b>nitroglycerin and Ca channel blockers</b>).</li></ul>

## 2- Myocardial infarction (Heart attack)

**Definition:** Necrosis of heart muscle resulting from ischemia, usually results from acute thrombosis that follow plaque disruption.

**Risk factors of MI :**

Are the same of atherosclerosis

**Types:**

1- Transmural	Myocardial necrosis involves the entire ventricular wall.
2- Subendocardial	Inner 1/3 of ventricular wall

**Note**

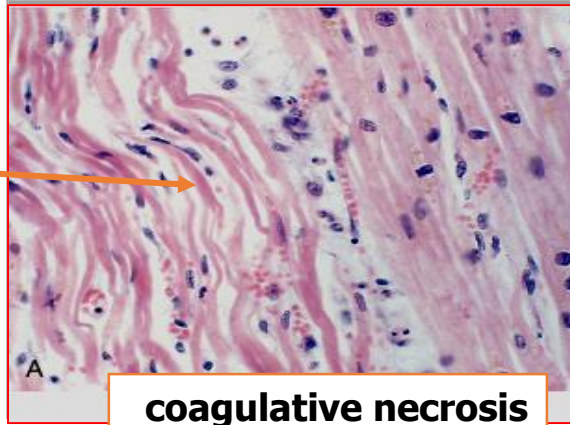
Ischemia to myocardium rapidly (20 – 30 minutes) leads to loss of function and causes necrosis after 20 to 40 minutes and become irreversible.

\*50% of MI involve the left anterior descending (LAD) artery.

The next most common site for MI is the right coronary artery (RCA), followed by the left circumflex (LCX).

**Morphology:** Microscopic feature

**One-day-old infarct**



**wavy fibers**

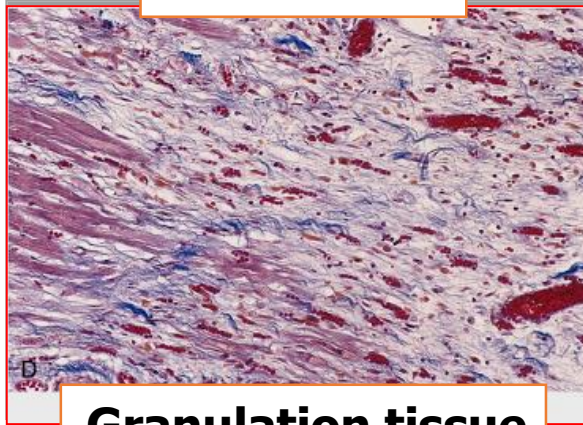
**coagulative necrosis**

**Up to 3 days duration**



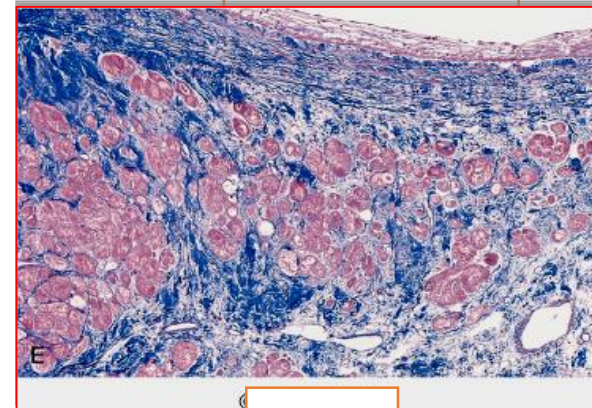
**Neutrophilic infiltrate**

**1 - 2 weeks**



**Granulation tissue**

**>3 weeks**



**Scar**

\*MI less than 12h old usually are not grossly apparent

# Morphologic Changes in Myocardial Infarction

- Coagulative necrosis and inflammation.
- Formation of granulation tissue.
- Organization of the necrotic tissue to form a fibrous scar.

Time	Gross changes	Microscopic changes
0-4h	None	None
4-12h	Mottling	Coagulation necrosis
12-24h	Mottling	More coagulation necrosis; neutrophils come in
1-7 d	Yellow infarct center	Neutrophils die, macrophages come to eat dead cells
1-2 w	Yellow center, red borders	Granulation tissue
2-8 w	Scar	Collagen



# Clinical presentation of MI

1. Severe, crushing substernal chest **pain** or discomfort that can radiate to the neck, jaw, epigastrium, or left arm.

**2. Diaphoresis**  
(Excessive sweating)

**3. Nausea**

**4. Dyspnea**

**5. Can be silent** (DM, old age)

## **6. ECG changes:**

- ✓ Q waves (indicating transmural infarcts).
- ✓ ST-segment abnormalities.
- ✓ T-wave inversion.
- ✓ Arrhythmias.

## Laboratory investigations:

- ✓ Cardiac **troponins** T and I (TnT, TnI).
- ✓ Creatine kinase (CK, and more specifically the myocardial-specific isoform **CK-MB**).
- ✓ Lactate dehydrogenase.

“Troponins and CK-MB have high specificity and sensitivity for myocardial damage”

	<b>Troponins</b>	<b>CK-MB</b>
Notes:	<ul style="list-style-type: none"><li>✓ The <b>best</b> marker for MI.</li><li>✓ TnI and TnT <b>are not</b> normally detectable in the circulation.</li></ul>	✓ <b>2<sup>nd</sup> best</b> marker of MI.
Become detectable:	after 2 to 4 hours.	within 2 to 4 hours of MI.
Peak at:	48 hours.	24 to 48 hours.
Duration:	Their levels remain elevated for 7 to 10 days.	Returns to normal within approximately 72 hours.

## Complications of MI:

At least **80%** will suffer:

- ✓ Cardiogenic shock (> 40% infarct of L.V).
- ✓ Congestive heart failure (**CHF**).
- ✓ Arrhythmia.
- ✓ Rupture of ventricle, free wall, septum, or papillary muscle
- ✓ left ventricular failure.
- ✓ Aneurysm formation.
- ✓ Mural thrombus, potentially source of emboli.
- ✓ Pericarditis.
- ✓ Thromboembolism
- ✓ External rupture of the infarct with associated bleeding into the pericardial space (**hemopericardium**).

## MI death and complications rates:

- 80-90% will develop complications.
- 10% of the rest will die within a month.
- 25% die, presumably due to arrhythmia.
- Overall 30% die in the 1<sup>st</sup> year and then 10% per year.

### 3- Chronic ischemic heart disease

**Definition:** Progressive heart failure due to ischemic injury, either from:

- ✓ prior infarction (**most common**).
- ✓ chronic low-grade ischemia.

### 4- Sudden cardiac death

**Definition:** Unexpected death from cardiac causes either without symptoms or within 1 to 24 hours of symptom onset.

- results from a lethal arrhythmia without myocyte necrosis.
- most commonly in patients with severe **coronary artery disease**.

**\*Acute coronary syndrome:** Refers to any group of symptoms attributed to obstruction of the coronary arteries.

**It is applied to three catastrophic manifestations of IHD:**

- ✓ Unstable angina.
- ✓ Acute MI.
- ✓ Sudden cardiac death.

1- The most common cause of IHD is:

- A. vasospasm
- B. vasculitis
- C. atherosclerosis

2- An imbalance between cardiac blood supply and oxygen demand of cardiac tissues is:

- A. Arrhythmia
- B. Atherosclerosis
- C. Ischemic heart disease

3- Stable angina typically occurs due to atherosclerotic disease with:

- A.  $\geq 75$  fixed chronic stable stenosis
- B.  $< 75$  fixed chronic stable stenosis
- C.  $< 50$  fixed chronic stable stenosis

4- Type of necrosis in MI:

- A. liquefactive
- B. Cuagulative
- C. Caseous

5- The other name of ischemic heart disease :

- A. Arrhythmia
- B. Atherosclerosis
- C. Coronary artery disease

Answers:

1-C

2-C

3-A

4-B

5-C

6- Which of the following is the most sensitive blood test to detect acute myocardial infarction?

- A. Creatine phosphokinase
- B. Cardiac troponins
- C. Lactate dehydrogenase

7- When CK-MB returns to its normal level ?

- A. within 72 hours
- B. within 24 hours
- C. within 48 hours

8- Which hormone protects human against IHD:

- A. Prolactin
- B. Testosterone
- C. Estrogen

9- When the troponins become detectable in MI ?

- A. after 8 hours
- B. after one hours
- C. after 2-4 hours

10- VARIANT ANGINA related to atherosclerosis :

- A. True
- B. False

Answers:

6-B

7-A

8-C

9-C

10-B

11- When does Myocardial Infarction become irreversible:

- A. 20-40 sec
- B. 20-40 min
- C. After 40 min

12- Most site of MI:

- A. left circumflex
- B. right coronary artery
- C. left anterior descending

13- In MI the change in T-wave in ECG is :

- A. Disappear
- B. Invert
- C. Prolong

14- The 2<sup>nd</sup> best marker of MI:

- A. Troponin
- B. Lactate dehydrogenase
- C. CK-MB

15- Which marker is not normally detectable :

- A. Troponin
- B. Lactate dehydrogenase
- C. CK-MB

Answers:

11- B

12-C

13-B

14-C

15-A

16- VARIANT ANGINA caused by:

- A. Coronary artery spasm
- B. Vasculitis
- C. Atherosclerosis

17- Most of people with MI:

- A. Develop complications
- B. Sudden death
- C. Nothing happen

18- Troponins reach their peak at :

- A. 24 hors
- B. 72 hours
- C. 48 hours

19- The most specific biomarker for diagnosis of acute coronary syndrome

- A. Troponin
- B. Myoglobin
- C. LDH
- D. Leucocytosis

20- All of the following are major risk factors of coronary artery disease except

- A. Hypercholesterolemia
- B. Hypertension
- C. Smoking
- D. Hyperuricemia

21- Clinical features of myocardial ischemia can be all except

- A. Crushing
- B. diaphoresis
- C. dyspnea
- D. Burning

Answers:

16- A

17-A

18-C

19-A

20-D

21-D



1- What are the types of angina ?

Stable, unstable, variant

2- **cardiac syndrome:**

1- Angina pectoris, 2- Myocardial infarction (MI), 3- Chronic IHD with CHF, 4- Sudden cardiac death (SCD)

3- List the complication of MI :

Cardiogenic shock, CHF, arrhythmias, aneurysm, pericarditis, mural thrombus, rupture of ventricle wall or papillary muscles.

4- What are the clinical presentations of MI ?

crushing substernal chest pain, diaphoresis, nausea, dyspnea.

5- How can we reduce risk of IHD?

Weight loss, quit smoking, aerobic exercises, decrease stress, and avoiding unsaturated fats.

6- What are Morphologic Changes in Myocardial Infarction?

Coagulative necrosis and inflammation, Formation of granulation tissue, Organization of the necrotic tissue to form a fibrous scar.

7- What are ECG changes in MI?

Q waves (indicating transmural infarcts), ST-segment abnormalities, T-wave inversion, Arrhythmias.

# Team's members:

Contact us:  
 [Pathology433@gmail.com](mailto:Pathology433@gmail.com)  
 @pathology433

- MAHA ALZEHEARY
- ABDULRAHMAN ALTHAQIB
- Aisha maher alsafi
- Abdullah Al-Zahrani
- Khawla dayel alshahrani
- Fahad Alotaibi

