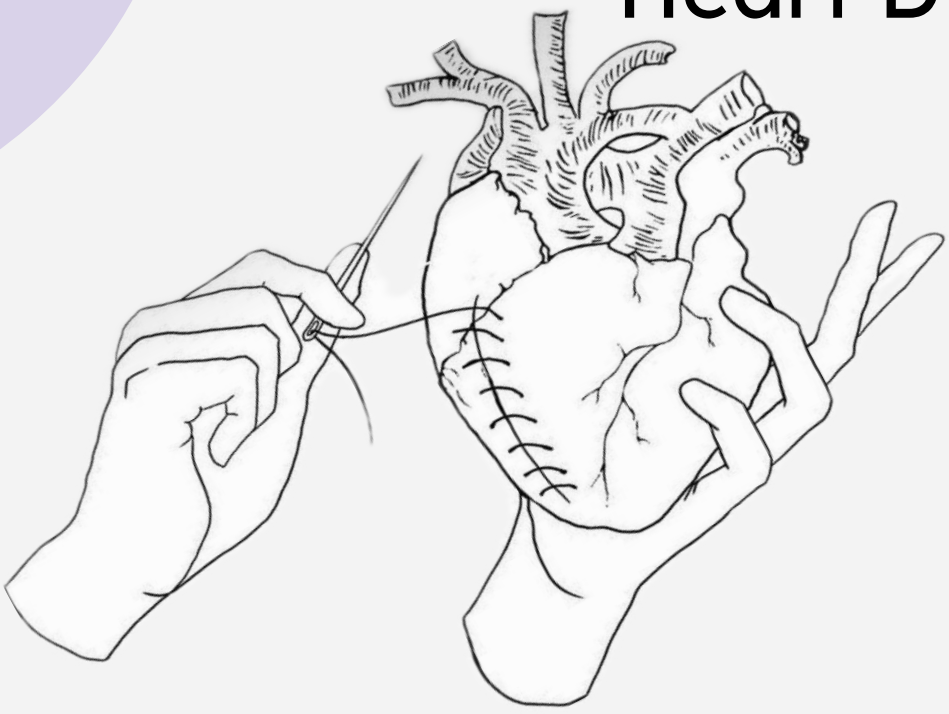
Pathology &

Pathogenesis Of Ischemic

Heart Diseases



Editing file:

COLOR INDEX:

MAIN TEXT (BLACK)

FEMALE SLIDES (PINK)

MALE SLIDES (BLUE)

IMPORTANT (RED)

DR'S NOTE (GREEN)

EXTRA INFO (GREY)

Objectives

- Understand the pathogenesis and clinical consequences of atherosclerosis
- 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 diseases.

Key principles to be discussed:

- Risk factors of atherosclerosis.
- Pathogenesis of the fibro lipid atherosclerotic plaque.
- Clinical complications of atherosclerosis.
- Commonest sites for the clinically significant coronary atherosclerosis
- Macroscopic and microscopic changes in myocardial infarction.
- Biochemical markers of myocardial infarction.
- Complications of myocardial infarction: immediate and late.

If you want to read the lecture from Robbins click here

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Ischemic Heart Disease

Definition

- IHD: a group of closely related condition/syndromes caused by an imbalance between the myocardial oxygen demand and blood supply. (myocardial ischemia)
Usually caused by decreased coronary artery blood flow (“coronary artery disease”)

- The most common cause if IHD is coronary artery atherosclerosis, Less commonly it is due to vasospasm and vasculitis.

- Is it exactly the same as coronary artery disease (CAD)? – Frequently yes

Four syndromes

Female’s slides

- Angina pectoris (chest pain)
- Acute myocardial infarction
- Sudden cardiac death
- Chronic ischemic heart disease with congestive heart failure

Female’s slides

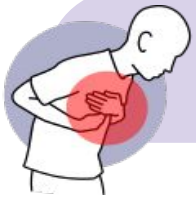
EPIDEMIOLOGY

Peak incidence: 60y for males and 70y for females.

Men are more affected than women.

Contributing factors are same as that of atherosclerosis e.g.

- Hypertension
- Diabetes mellitus
- Smoking
- High levels of LDL
- Genetic factors (direct or indirect)
- Lack of exercise



Pathogenesis of IHD

Female's Slides

Pathogenesis of IHD

1. Role of Critical stenosis or obstruction
2. Role of Acute Plaque Change
3. Role of Coronary Thrombus
4. Role of Vasoconstriction
5. Role of Inflammation

Deep Focus Question

Thx 443

A patient has non-ischemic chest pain and a friction rub the day after myocardial infarction. What characteristics of pain are expected?

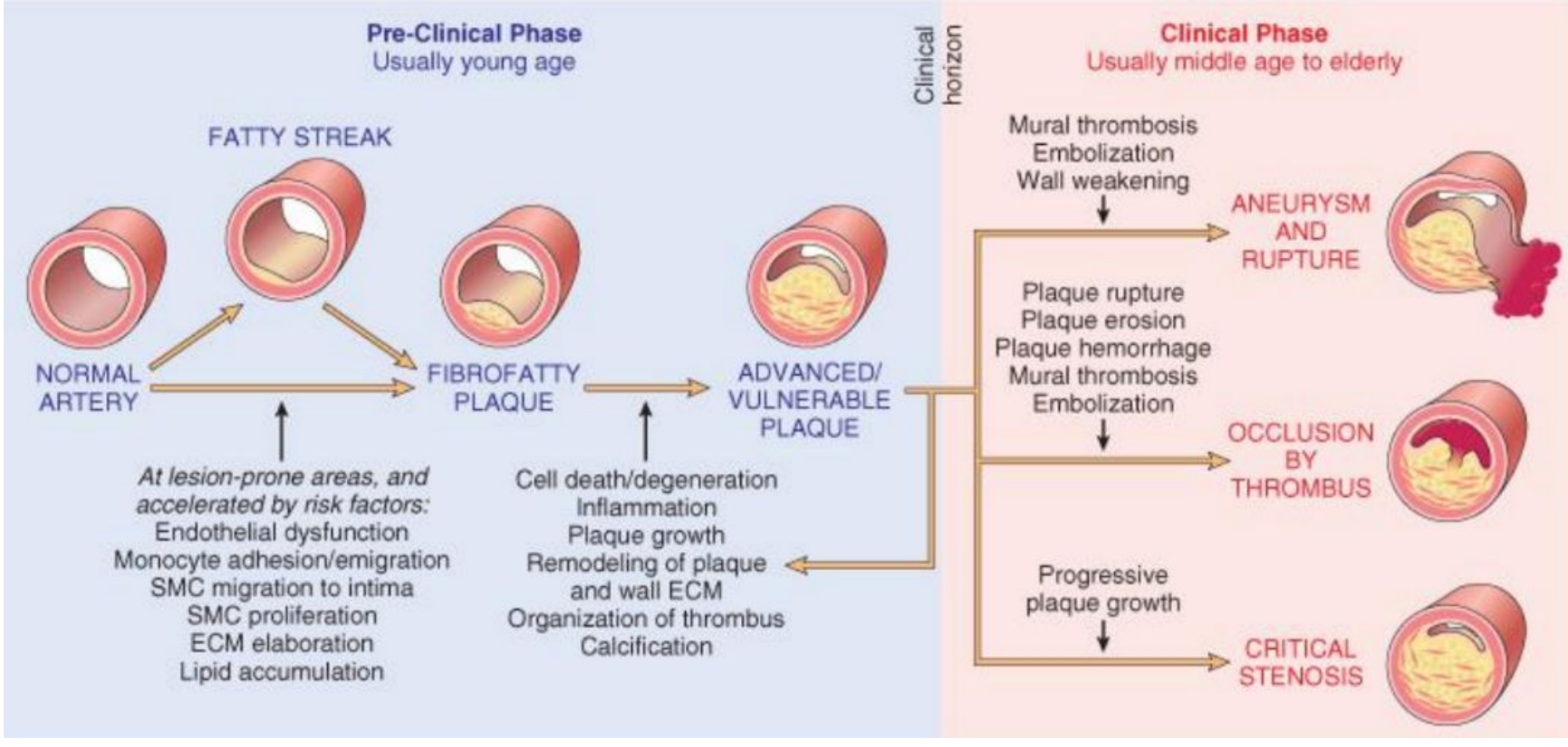
A. Improved by lying supine
B. Tender to palpation
C. Better with deep inspiration
D. Worse with sitting up and leaning forward
E. Improved by sitting up and leaning forward
Answer: E
Non ischemic -> first lecture

Pathogenesis of IHD

Female's Slides

1 Role of Critical stenosis or obstruction

(>=75% of the lumen of one or more coronary arteries by atherosclerotic plaque).



Pathogenesis of IHD

Female's Slides

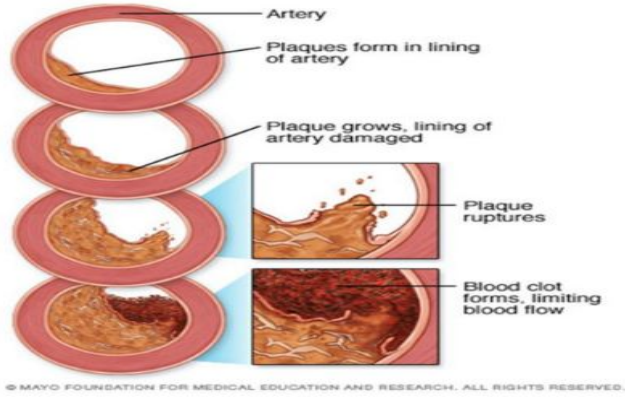
2 Role of Acute Plaque change

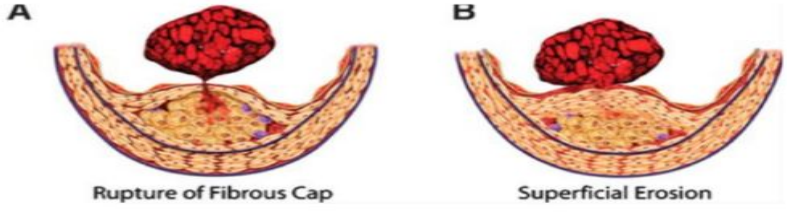
Disruption of a mildly stenosing plaque leading to rupture/ ulceration.
This can lead to:

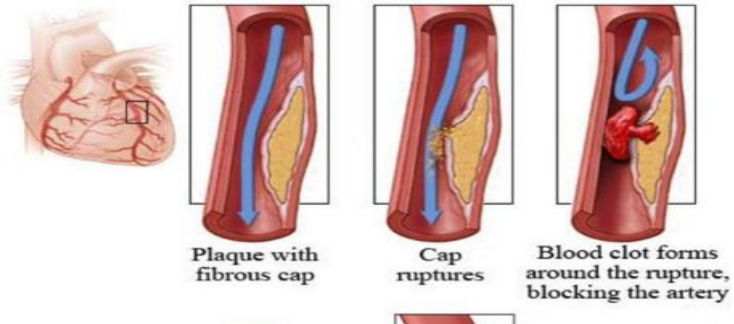
- hemorrhage into the atheroma which will expand in volume.
- exposure of the pro-thrombogenic basement membrane just below the endothelial cells thrombosis the formed thrombus will further block the lumen of the blood vessel.

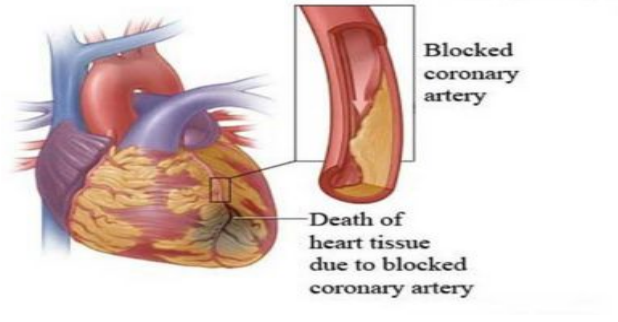
So :
Acute plaque change can cause myocardial ischemia in the form of

- unstable angina
- acute myocardial infarction.
- and sudden cardiac death.









Pathogenesis of IHD

Female's Slides

3 Role of Acute Plaque change

thrombus superimposed on a disrupted partially occluding plaque can convert the plaque to either:

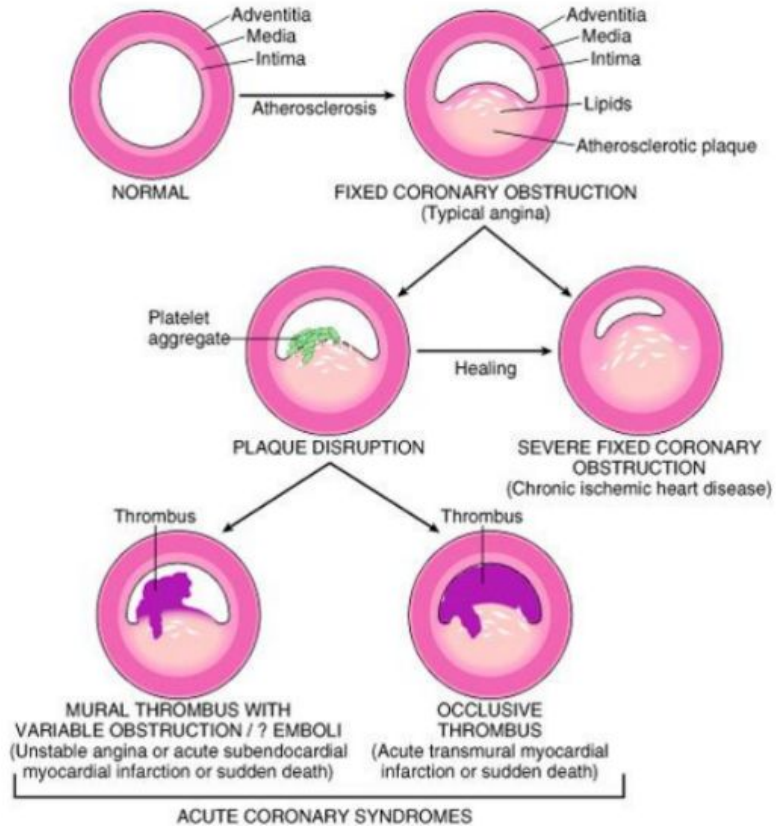
- A total occlusion leading to acute transmural MI or sudden death.
- a partial/incomplete/subtotal occlusion leading to unstable angina, acute subendocardial infarction, or sudden death.
- Thrombus in coronary artery can also embolize.

4 Role of vasoconstriction

Vasoconstriction reduces lumen size and can therefore potentiate plaque disruption.

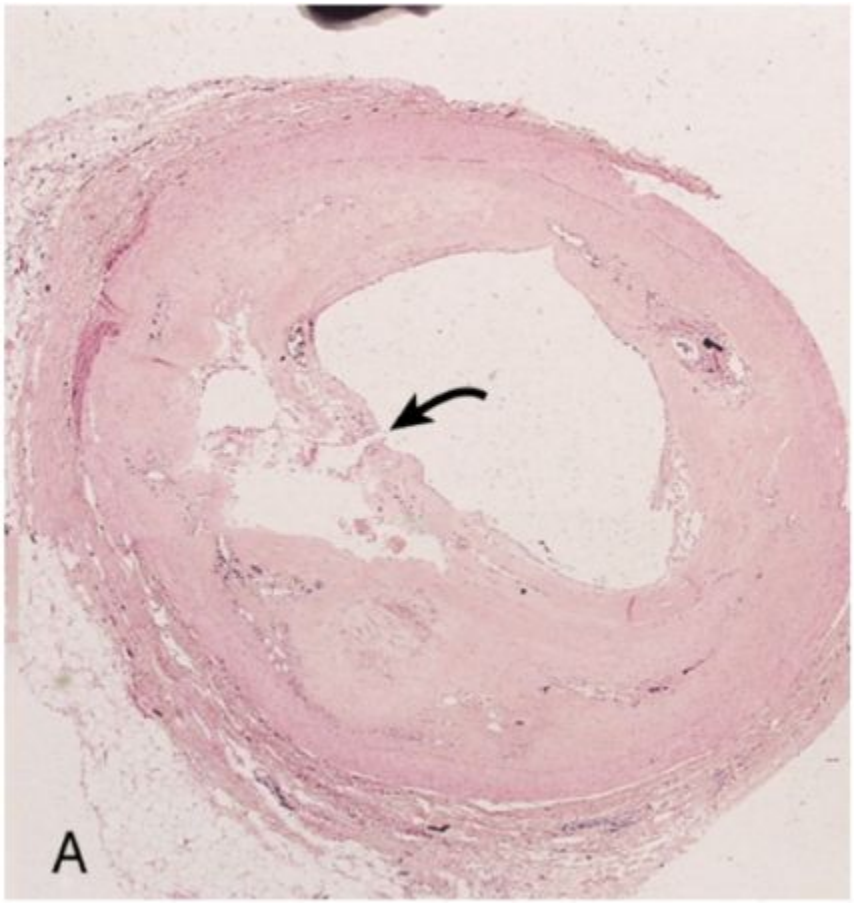
5 Role of inflammation

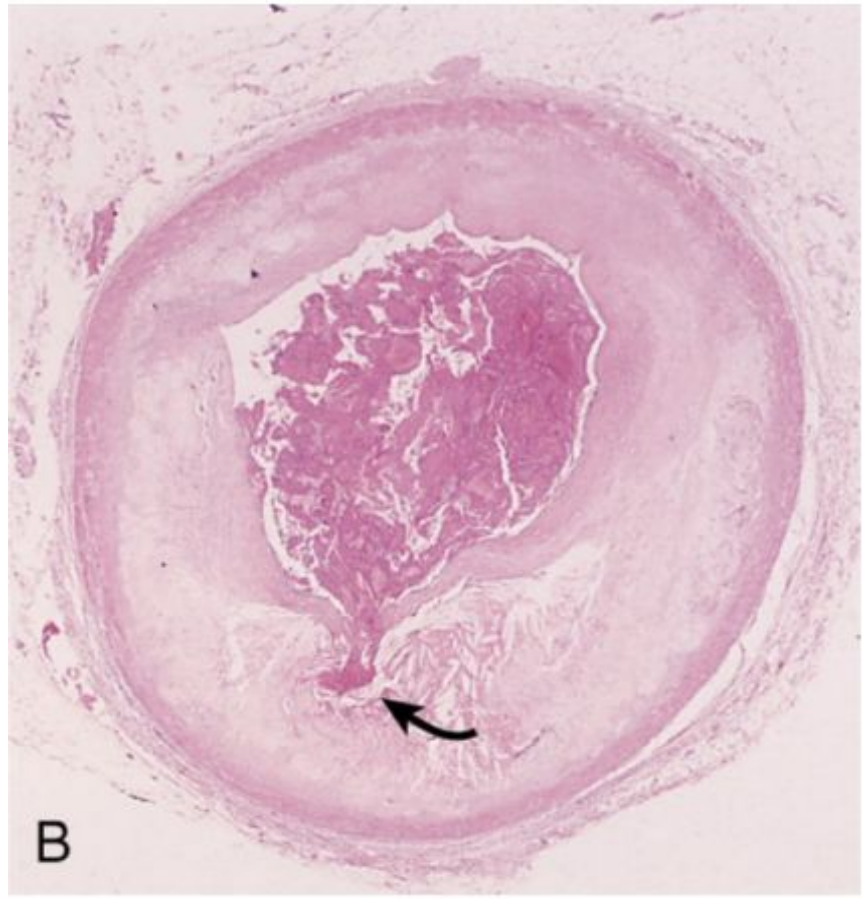
Inflammatory processes play important roles at all stages of atherosclerosis .



Morphology

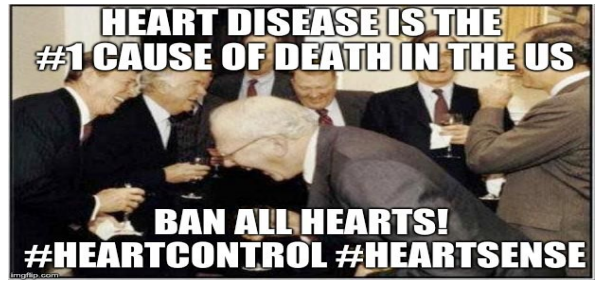
Female's Slides





-Plaque rupture without superimposed thrombus in a patient who died suddenly.

Acute coronary thrombosis superimposed on an atherosclerotic plaque with focal disruption of the fibrous cap, triggering fatal myocardial infarction.



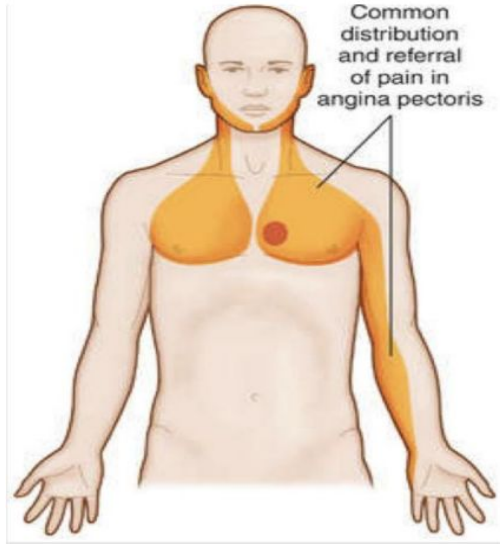
Angina Pectoris

Definition

Angina pectoris is a type of IHD characterized by paroxysmal(sudden&violent) and usually recurrent attacks of substernal or precordial chest discomfort, described as constricting, crushing, squeezing, choking, or knifelike pain.
The pain may radiate down the left shoulder, left arm, neck or left jaw (called as referred pain) .

Causes

Angina pectoris is due to inadequate (lack of) perfusion and is caused by transient (15 seconds to 15 minutes) myocardial ischemia that falls short of inducing necrosis i.e. duration and severity is not sufficient to cause infarction(irreversible injury)



Types

- Stable or typical angina
- Unstable or crescendo angina
- Prinzmetal or variant angina

Types of angina pectoris

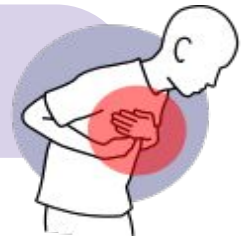
Stable Angina(Typical Angina)

- Is the most common form of angina
- Occurring reliably after certain levels of exertion.
- Is due to a fixed stenosis, The chest pain is episodic.
- Is caused by atherosclerotic disease with usually ≥70% / 75% narrowing of lumen (i.e. fixed stable critical stenosis).
- This reduction (due to ≥ 70% stenosis) of blood flow in coronary vessels makes the heart vulnerable, so whenever there is increased demand e.g. physical activity, emotional excitement, or any other cause of increased cardiac workload, there is angina pain.
- Relieved by rest (i.e. decreasing demand) or with vasodilators like sublingual nitroglycerin.

Unstable or Crescendo Angina

- It is an unstable and progressive condition.
- Pain occurs with progressively increasing frequency, and is precipitated with progressively less exertion or even at rest, and tends to be of more prolonged duration.
- It is induced by disruption or rupture / small fissure of an atheroma plaque (acute plaque change)(atherosclerotic plaque) triggering : -Platelet aggregation -Vasoconstriction -Formation of a mural thrombus that may not be occlusive.
- with superimposed thrombosis and partial occlusion of a coronary vessels.
- Unstable angina is often the precursor of subsequent acute MI. Thus also called as **preinfarction angina.**

Types of angina pectoris



Prinzmetal or Variant angina

- uncommon pattern of episodic angina that occurs at rest and is due to coronary artery spasm.
- Not related to atherosclerotic disease .
- The etiology is not clear .
- Prinzmetal angina generally responds promptly to vasodilators, such as nitroglycerin and calcium channel blockers.

Summary

Intermittent chest pain caused by transient, reversible ischemia

Stable Angina(Typical Angina)	Unstable or Crescendo Angina	Prinzmetal or Variant angina
• pain on exertion • fixed narrowing of coronary artery	• increasing pain with less exertion • plaque disruption and thrombosis	• pain at rest • coronary artery spasm of unknown etiology

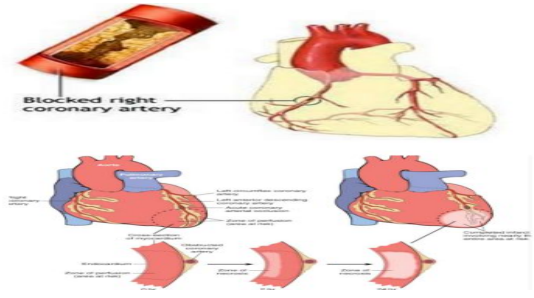
Myocardial Infarction

Definition

The death of cardiac muscle (coagulative necrosis) resulting from ischemia.
The severity or duration of ischemia is enough to cause cardiac muscle death
Typically results from acute thromboses that follow plaque disruption.

Risk Factors

Are the same as those of coronary atherosclerosis.
➢ Atherosclerosis + age + male gender.
➢ Females are more affected after menopause due to decreased estrogen production



1. Long standing atherosclerotic plaque
2. Acute plaque change
3. Disruption of the plaque
4. Thrombus formation
5. Ischemia
6. Hypoxia
7. Irreversible cell Injury -unlike angina-
8. cardiac muscle necrosis

note: the thrombus usually evolves to completely occlude the lumen of the coronary vessel within minutes).

Myocardial Infarction

- The commonly affected coronary vessel in MI: In persons with right dominant coronary artery heart (90% of population) the commonly affected blood vessels are:

1

2 cm proximal Left anterior descending artery.(40-50%) : Most commonly affected artery :

- anterior left ventricle
- anterior septum
- apex circumferentially

2

1st and last thirds of Right coronary artery (30-40%) :

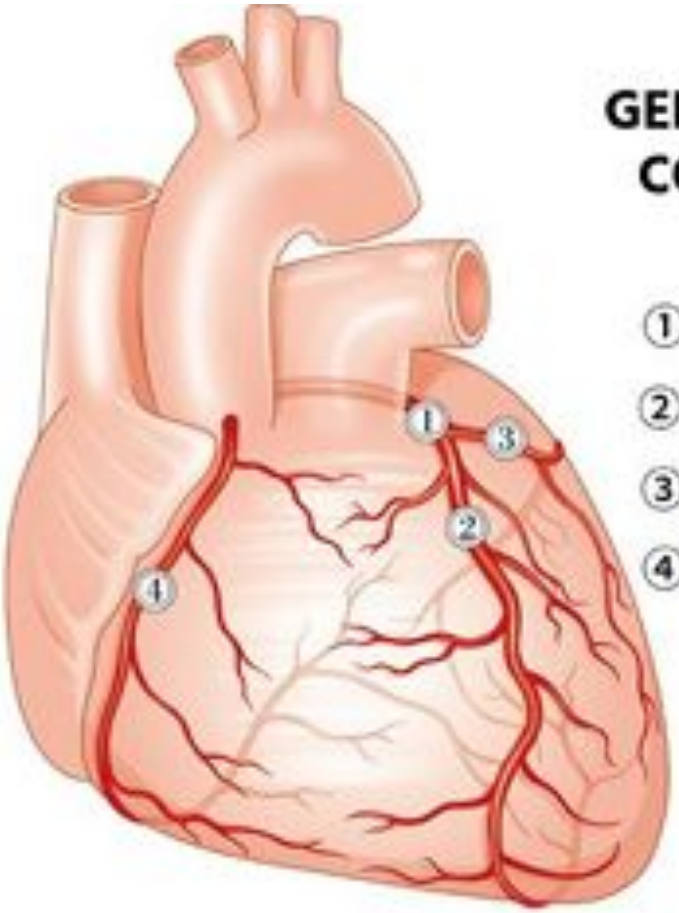
- posterior left ventricle
- posterior septum
- right ventricular free wall, sometime

3

Left circumflex artery (about 20%):

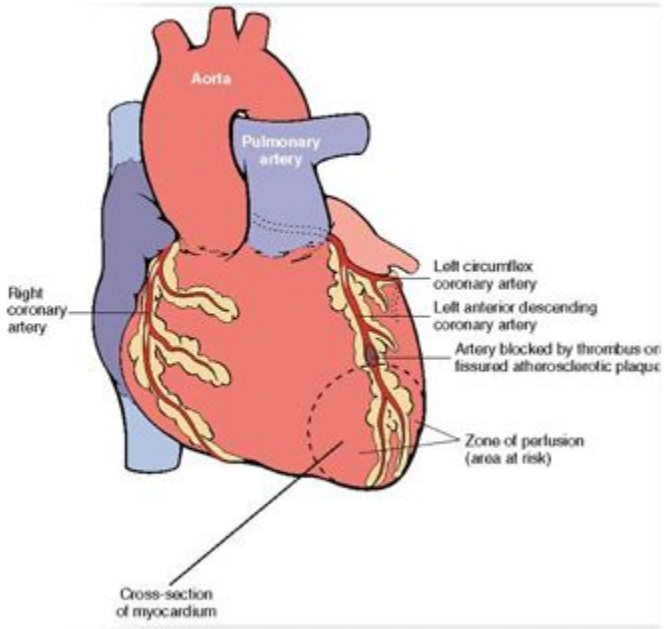
infract involves lateral left ventricle except the apex

Myocardial Infarction



GENERAL ANATOMY OF CORONARY ARTERIES

① left main trunk
② left anterior descending artery
③ left circumflex artery
④ right coronary artery.



Myocardial Infarction

pathogenesis

Most common cause is thrombosis on a preexisting disrupted atherosclerotic plaque. In the typical case of MI, the following sequence of events usually occur:

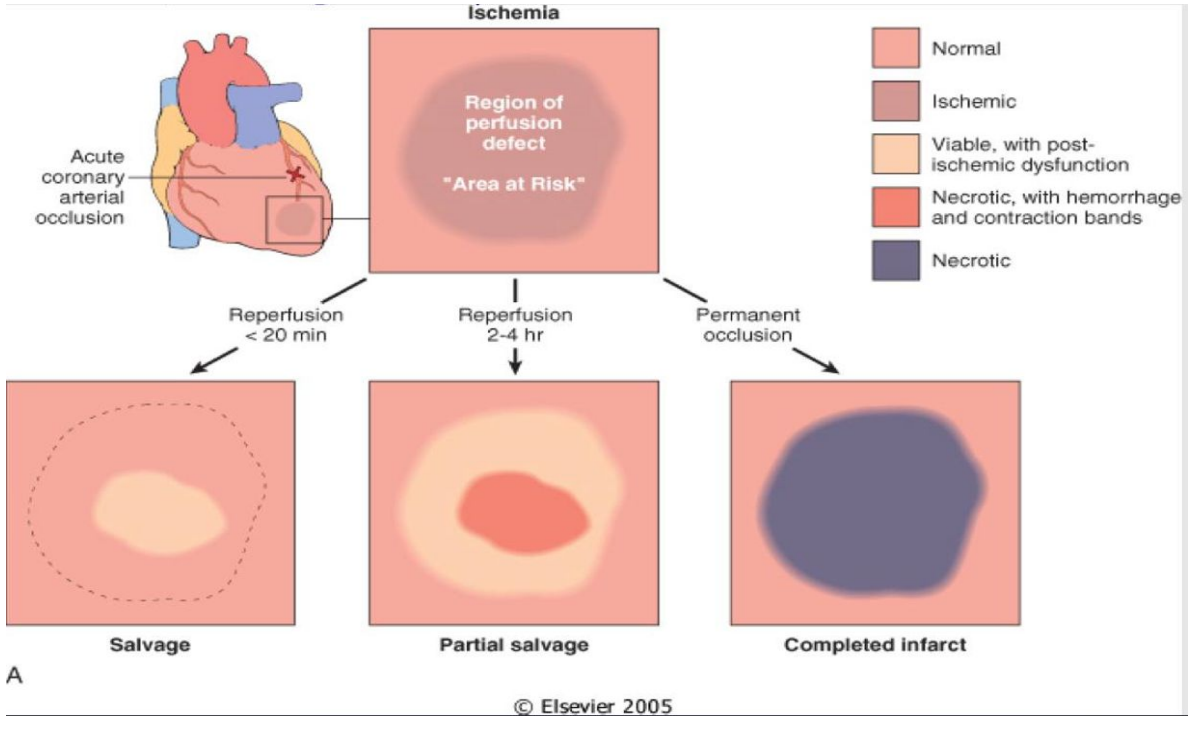
Acute Plaque change	(sudden change in the structure of an atheromatous plaque e.g. disruption, ulceration, rupture or intraplaque hemorrhage).
Thrombus formation	Exposure of the thrombogenic subendothelial basement membrane resulting in thrombus formation
Occlusion of the lumen	Exposure of the thrombogenic subendothelial basement membrane resulting in thrombus formation

Pathogenesis

vere ischemia lasting at least 20 to 40 minutes causes irreversible injury and myocardial necrosis on the ultrastructural level (on electron microscopy).

Myocardial necrosis mostly starts in the sub-endocardial region (because it is less perfused and has high intramural pressure)

The full size of the infarct is usually determined within 3-6 hours of the onset of severe myocardial ischemia. During this period, lysis of the thrombus by treatment with streptokinase or tissue plasminogen activator, may limit the size of the infarct. So any intervention in this time frame can potentially limit the final extent of necrosis.



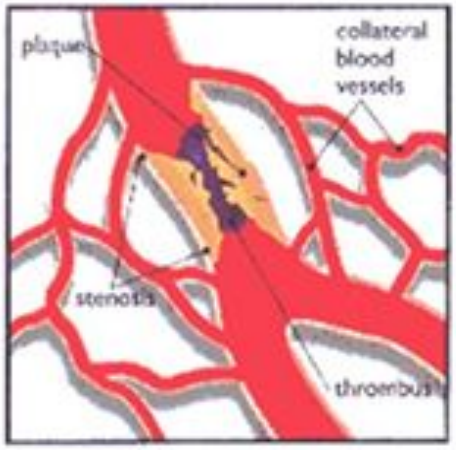
Myocardial Infarction

Female's Slides

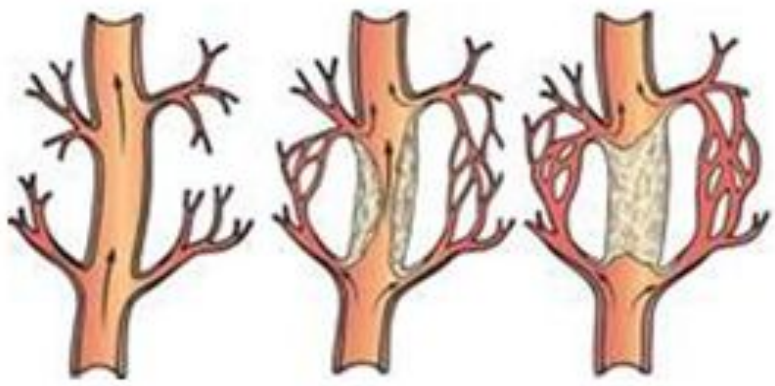
The precise location, size, and specific morphologic features of an acute myocardial infarct depend on:

1. The location, severity, and rate of development of coronary atherosclerotic obstructions
2. The size of the area supplied by the obstructed vessels
3. The duration of the occlusion
4. The oxygen needs of the myocardium at risk
5. The extent of collateral blood vessels
6. Other factors, such as blood vessel spasm, alterations in blood pressure, heart rate, and cardiac rhythm.
7. In addition reperfusion may limit the size of the infarct.

Myocardial Infarction



further explanation



The body adapt to atherosclerosis plaque by forming collateral blood vessels allowing alternative pathway to blood flow this process happen with people with chronic atherosclerotice diseases , but someone with acute change in the plaque there will be no time to form collateral circulation

- Ischemia to myocardium rapidly (minutes) leads to loss of function and causes necrosis after 20 to 40 minutes

Myocardial Infarction

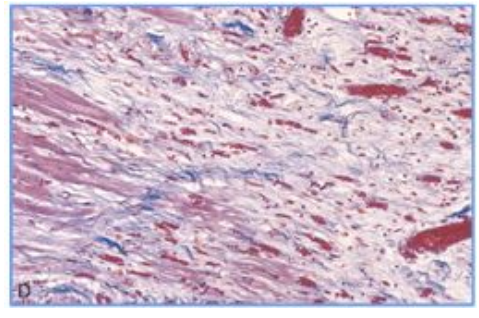
MI Types

- **Transmural**
  - Full thickness (>50% of the wall)
- **Subendocardial**
  - Inner 1/3 of myocardium

- The infarction as usual will start distal to the vessel but will stop due to :

Two mechanisms:

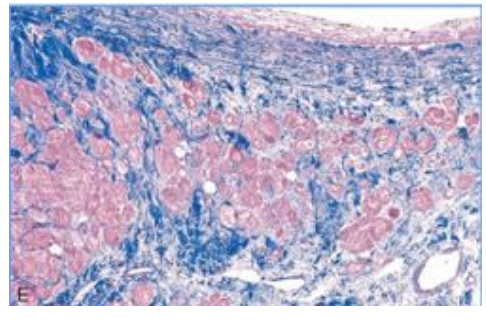
- Fixed atherosclerosis but with increased demand,vasospasm or hypotension
- Evolving transmural with relieve of the obstruction (often multifocal)



Morphology

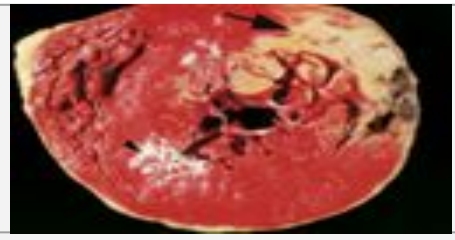
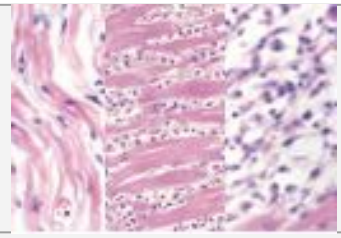
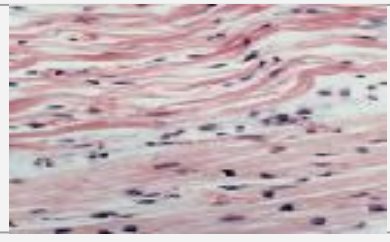
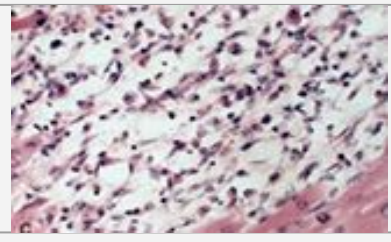
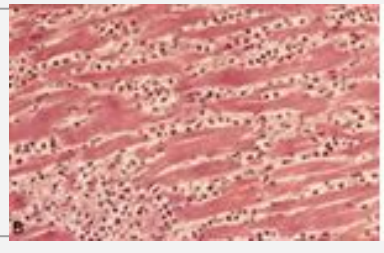
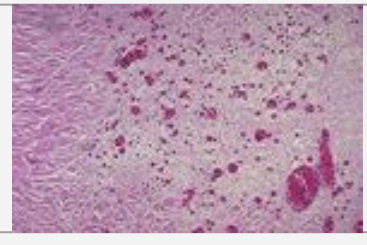
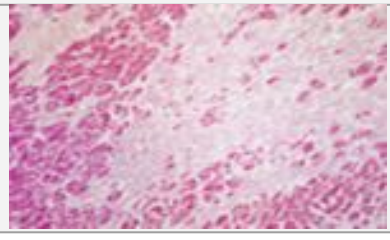
Begins with coagulative necrosis and inflammation (initially mainly neutrophils and later macrophages).

- Followed by formation of granulation tissue.
- Heals by formation of a fibrous scar



note: for clearer pictures its better to check the slides

Myocardial Infarction

Morphology		
Description	Acute Myocardial Infarction	DAY1 DAY2 DAY3 Myocardial Infarction
Morphology		
Description	One-day-old infarct showing coagulative necrosis with few neutrophils, wavy fibers with elongation, and narrowing, compared with adjacent normal fibers (lower right).	Nearly complete removal of necrotic myocytes by phagocytosis (approximately 7 to 10 days).
Morphology		
Description	Dense neutrophilic infiltrate in an area of acute myocardial infarction of 3 to 4 days' duration.	Granulation tissue approximately 3 weeks post MI
Morphology & Description		Healed MI with replacement of the necrotic fibers by dense collagenous scar. Residual cardiac muscle cells are present

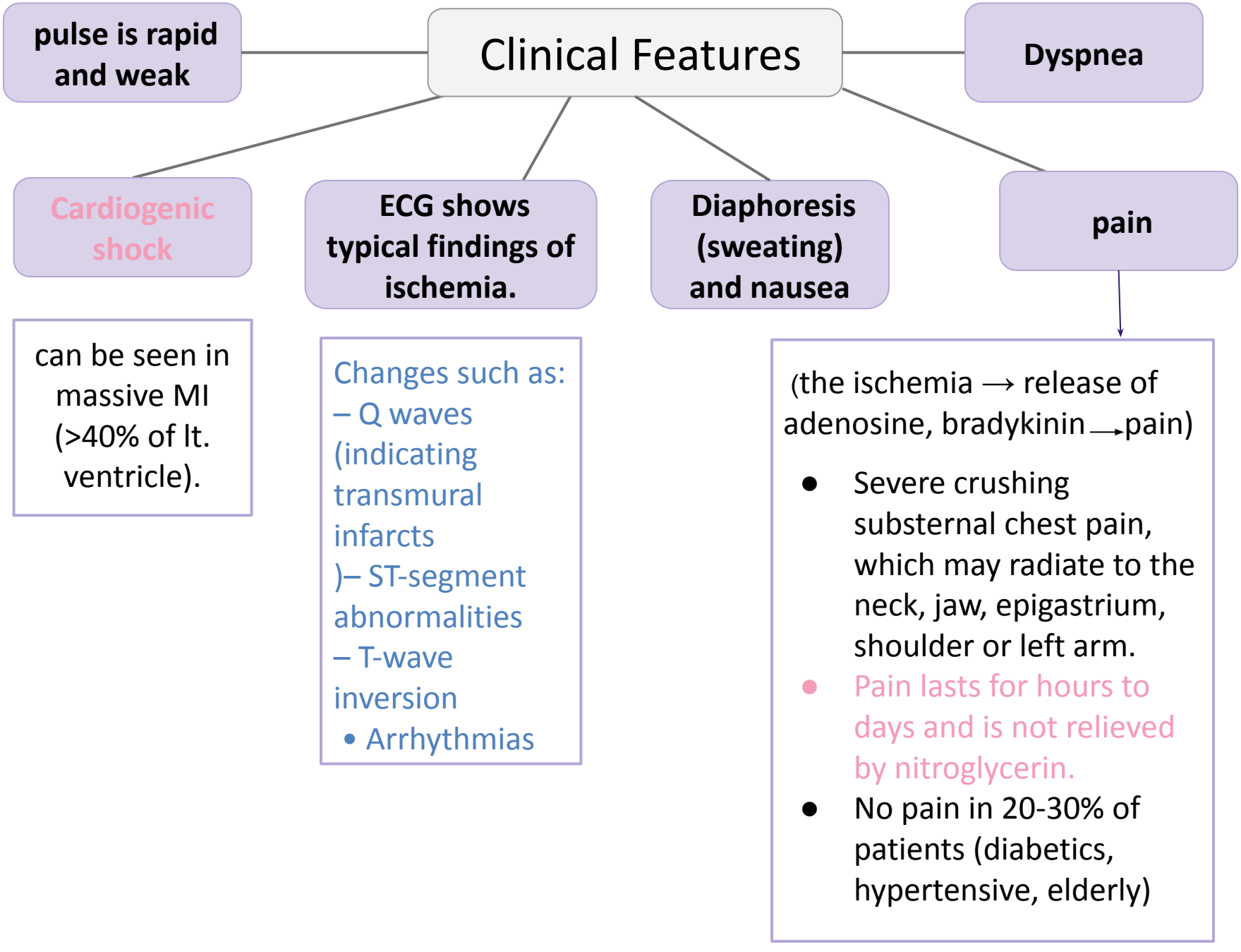
Myocardial Infarction

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

reperfusion injury can incite greater local damage than the damage might have occurred without rapid restoration of blood flow. reperfusion injury is mediated in part by oxygen free radicals generated by the increased number of infiltrating leukocytes facilitated by reperfusion. Reperfusion-induced microvascular injury causes not only hemorrhage but also endothelial swelling that occludes capillaries and may prevent local blood flow (called no-reflow).

A reperfused infarct usually has hemorrhage because the vasculature injured during the period of ischemia is leaky after flow is restored

Myocardial Infarction



MI : Laboratory Elevation

IMPORTANT

Troponins

best marker, TnT, TnI (more specific).
1)TnI and TnT are not normally detectable in the circulation 2)

After acute MI both troponins become detectable after 2 to 4 hours, peaks at 48 hours. Their levels remain elevated for 7 to 10 days

CK-MB

second best marker ,Creatine kinase (CK, and more specific the myocardial specific isoform, CK-MB)
: It begins to rise within 2 to 4 hours of MI, peaks at 24 hours and returns to normal within approximately 72 hours.

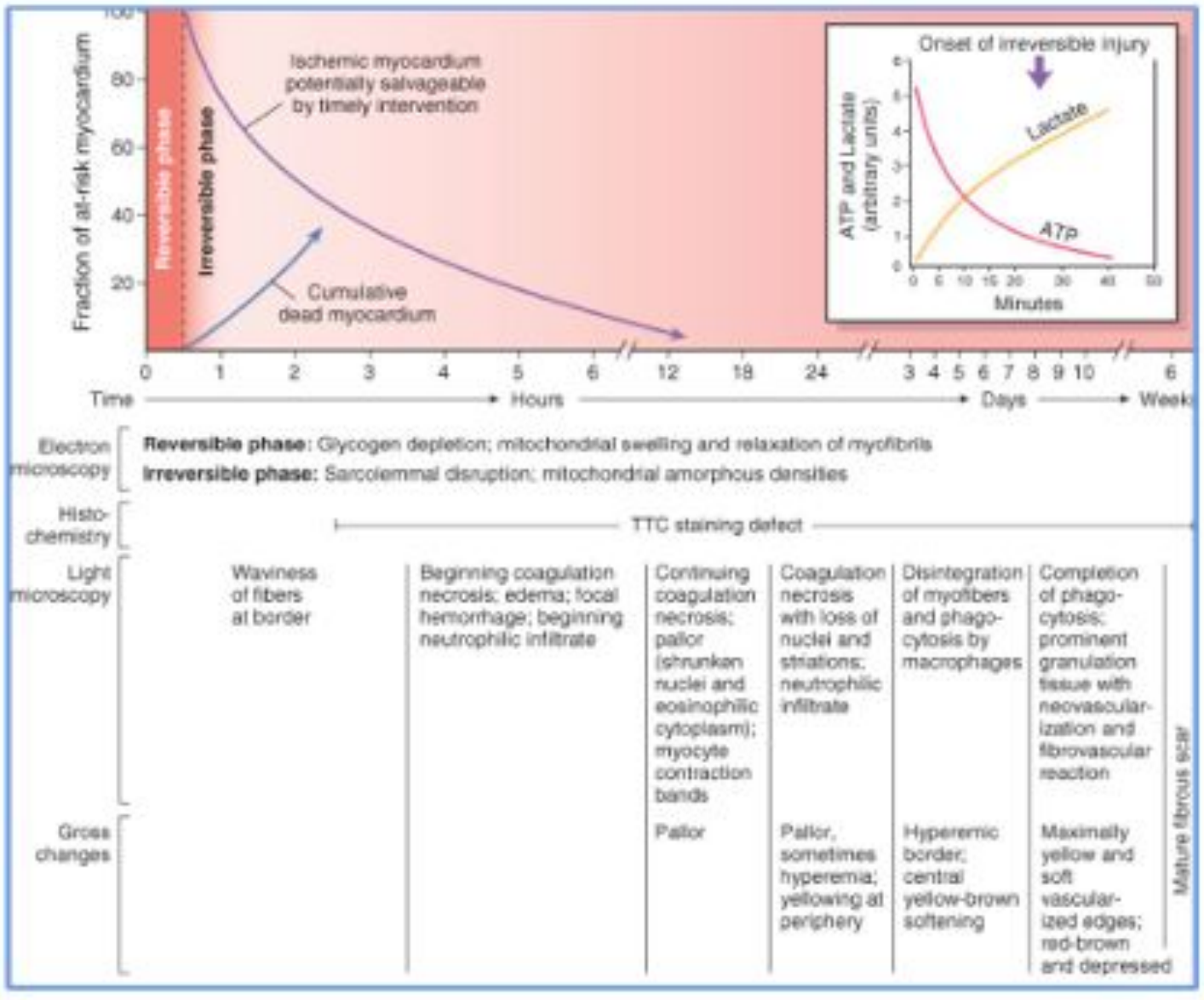
Myoglobins

Myoglobin is a protein found in muscle tissue that is responsible for storing and transporting oxygen. It is not specific for myocardial infarction it could be also seen in skeletal muscle necrosis

Lactate dehydrogenase

LD : Rise 24 hrs, peaks 72 hrs, gradually disappears in 5 to 14 days

MI : Laboratory Elevation



- nitroglycerin works on angina, so, you can differentiate between the two by the patient's response to the drug

MI: COMPLICATIONS

Complication

- No complications in 10-20%.
- 25% die, presumably due to arrhythmia.
- 10% of the rest will die within a month
- Overall 30% die in the 1st year and then 10% per year.

80-90% experience one or more of the following complications:

MI lead to sudden death in some cases, even before reaching the hospital

1 Cardiac arrhythmia a (75-90%)

patients have conduction disturbances which can cause sudden death especially in ventricular arrhythmia.
(The most common cause of death in patients with acute MI)

2 Left ventricular failure with pulmonary edema (60%)
Congestive heart failure (CHF)

Associated with mild to severe pulmonary edema

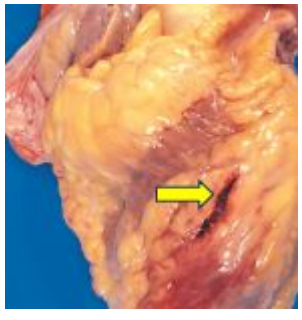
MI: COMPLICATIONS

3	Cardiogenic shock(10%) (>40% infarct of LV)	condition in which your heart suddenly can't pump enough blood
4	Myocardial rupture	• Rupture of free wall • Septum • Papillary muscle (which will lead to blood regurgitation) or Mitral valve incompetence
5	Thromboembolism occurs in 15-49% of patients (mural thrombus) potentially source of emboli	Combination of contractility abnormality in contractility (causing stasis), and endocardial damage (due to exposure of underlying thrombogenic basement membrane) can lead to cardiac thrombosis and embolism
6	Pericarditis	Inflammation of the pericardium
7	Ventricular aneurysm formation	(ventricle is dilated and wall is thinned out)

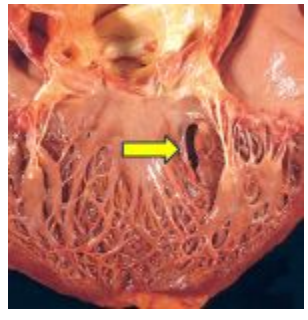
MI: COMPLICATIONS

8	External rupture of the infarct	Associated with bleeding into the pericardial space (hemopericardium).
9	Progressive late heart failure in the form of chronic IHD	-
10	Infarct extension and expansion	-

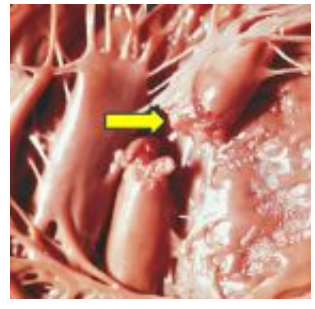
Ventricular wall rupture



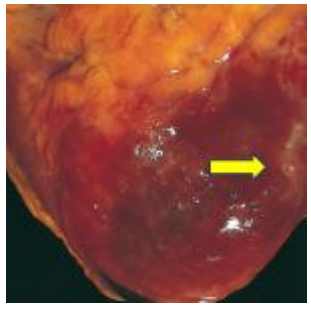
Septal rupture



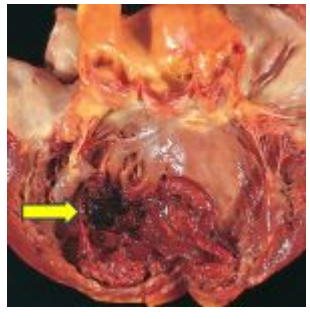
Papillary muscle rupture



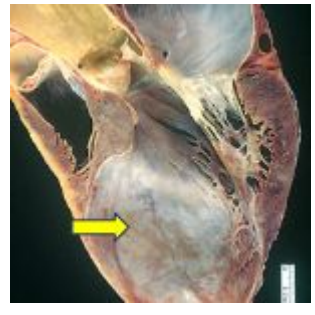
Fibrinous pericarditis



Mural thrombus



Aneurysm



Summary

Necrosis of heart muscle caused by ischemia	
Mostly due to acute coronary artery thrombosi	● sudden plaque disruption ● platelets adhere ● coagulation cascade activated ● thrombus occludes lumen within minutes ● irreversible injury/cell death in 20-40 minutes
Clinical features	● Severe, crushing chest pain ± radiation ● Not relieved by nitroglycerin, rest ● Sweating, nausea, dyspnea ● Sometimes no symptoms
Laboratory evaluation	● Troponins increase within 2-4 hours, remain elevated for a week ● . CK-MB increases within 2-4 hours, returns to normal within 72 hours
Complications	● contractile dysfunction ● arrhythmias ● rupture ● chronic progressive heart failure
Prognosis	● depends on remaining function and perfusion ● Prompt reperfusion can salvage myocardium

Ischemic Heart Disease

Chronic ischemic heart disease

Progressive heart failure due to ischemic injury, either from:
1) prior infarction(s) (most common)
2) or chronic low-grade ischemia

Sudden cardiac death

Definition: Unexpected death from cardiac causes either without symptoms or within 1 to 24 hours of symptom onset Results from a fatal arrhythmia, most commonly in patients with severe coronary artery disease

Acute coronary syndrome

MALES SLIDES

is applied to three catastrophic manifestations of IHD:
- Unstable angina
- Acute MI
- Sudden cardiac death

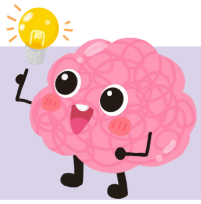
Summary

IHD = a group of closely related conditions/syndromes caused by an imbalance between the myocardial oxygen demand and blood supply (resulting from myocardial ischemia). Usually caused by decreased coronary artery blood flow (“coronary artery disease”)

Type		Description
Angina pectoris	a type of IHD characterized by paroxysmal and usually recurrent attacks of substernal or precordial chest discomfort, described as constricting, Definition crushing, squeezing, , or knifelike pain , due to inad who in inadequate perfusion & transient myocardial infraction .	
	Stable angina	-most common form of angina , due to a fixed stenosis Is caused by atherosclerotic disease with usually ≥70-75% narrowing of lumen (angina / fixed stable critical stenosis). Relieved by rest & vasodilators like sublingual nitroglycerin
	Unstable angina	-unstable and progressive angina condition & Pain occurs with progressively increasing frequency, and is precipitated with progressively less exertion, (even at rest), and tends to be of more Unstable / prolonged duration. induced by disruption or rupture of an atheroma plaque & Formation of a mural thrombus
	Prinzmetal angina	-uncommon pattern of episodic angina that occurs at rest and is due to coronary artery spasm. & responds promptly to vasodilators, such as nitroglycerin and calcium channel blockers , only type that is not related to atherosclerosis

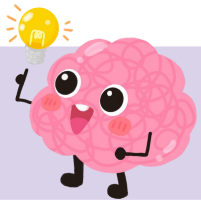
Summary

Type		Description
Myocardial infarction	Definition	The death of cardiac muscle (coagulative necrosis) resulting from ischemia.
	Risk factors	1) Atherosclerosis + age + male gender. 2) Females are more affected after menopause due to decreased estrogen production
	Sequence of events	Long standing atherosclerotic plaque > acute plaque change > distruption of the plaque > thrombus formation > ischemia > hypoxia > irreversible cell injury > cardiac muscle necrosis
	Types	1. Transmural : Full thickness (>50% of the wall) 2. Subendocardial : Inner 1/3 of myocardium.
	Symptoms	•Diaphoresis •Dyspnea • ECG shows typical findings of ischemia •pain • cardiac shock • Rapid and weak pulse
	Laboratory evaluation	• Troponins : best marker •CK-MB : 2nd best marker •Lactate dehydrogenase (LD) • Myoglobins
Chronic ischemic heart disease	Progressive heart failure due to ischemic injury, either from: ● Prior infarction(s) (most common) ● Or chronic low-grade ischemia	
sudden cardiac death(S DC)	● Unexpected death from cardiac causes either without symptoms or within 1 to 24 hours of symptom onset (different authors use different time points) ● Results from a fatal arrhythmia, most commonly in patients with severe coronary artery disease	



KEYWORDS

Angina pectoris	● Substernal pain ● Radiate to left arm , shoulder , jaw
Stable Angina (Typical Angina)	● fixed critical stenosis. ● Occurring reliably after certain levels of exertion
Unstable or crescendo Angina	● More pain with less exertion. ● acute plaque change.
Variant (prinzmetal) Angina	● uncommon pattern of episodic angina that occurs at rest.



KEYWORDS

Myocardial infarction	● ECG changes ● Troponin , CK-MB findings ● Myocardial rupture ● Ventricular aneurysm ● Doesn’t’ respond to nitroglycerin
Chronic ischemic heart disease	Due to : ● infection ● chronic low-grade ischemia
sudden cardiac death	● no symptoms usually ● Results from a fatal arrhythmia

Answers:
1- B 2-A 3- A 4- D

MCQ

1- **A 55 year old male comes to the clinic complaining of chest pain that arises when even while resting, there is no previous history of lung diseases and testing for atherosclerosis was negative which of the following could be the diagnosis:**			
A)Stable angina	B)Prinzmetal angina	C)Myocardial infarction	D)Thrombosis
2-what is the main cause of stable angina?			
A)atherosclerosis	B)hypertension	C)hyperthyroidism	D)diabetes mellitus
3- which one of the following is most related to Stable(Typical) angina?			
A)critical stenosis	B)Acute Plaque change	C)vasoconstriction	D)Emboli
4- **Which one of the following conditions isn't a common complication of MI**			
A)Pericarditis	B)Thromboembolism	C) Ventricular aneurysm	D)Right ventricular failure

Answers:
5-D. 6-c. 7-D 8-A

MCQ

5-The fate of a thrombus may include all of the following except:			
A)Propagation	B)Embolization	C)-Lysis	D) Neoplastic transformation
6-Which of the following microscopic findings can be seen in MI day 7-14?			
A)Granuloma	B)-Fibrosis	C)-Granulation tissue	D)-Neutrophils
7-are more prone to be affected by myocardial infarction:			
A)Pre-puberty boys	B)Young adults	C)-children	D)-Postmenopausal women
8- Which of the following types of angina is not related to atherosclerosis?			
A)variant angina	B)T-crescendo angina	C) -stable angina	D)-unstable angina

Answers:
1-c 2- c 3-a 4-c

Cases

Thxx 443 for these amazing cases <\3

1.A 65-year-old man is brought to the emergency department due to chest pain that started 12 hours ago while watching television. The patient describes the pain as “heavy pressure” located in the center of the chest that radiates up to the neck and jaw. He states that the pain has waxed and waned, although he has felt some relief from taking sublingual nitroglycerin. Medical history is significant for coronary artery disease, stable angina, hyperlipidemia, and type 2 diabetes mellitus. The patient’s temperature is 36.7° C (98.0°F), pulse is 110/min, respirations are 24/min, and blood pressure is 135/93 mmHg. Pulse oximetry on room air shows an oxygen saturation of 97%. Chest x-ray shows no abnormalities, cardiac enzymes are negative, and ECG shows ST-segment depression in the anterior leads. Which of the following is the most likely diagnosis?			
A)Stable angina	B)Prinzmetal angina	C)Unstable angina	D)Diffuse esophageal spasm
2.A 44-year-old man presents to the emergency room with acute chest pain. The ECG is normal. Analysis of which pair of serum markers given below would be most helpful in excluding a diagnosis of acute myocardial infarction in this patient?			
A)Cardiac troponin-I and myoglobin	B)CK-BB and myoglobin	C)CK-MB and cardiac troponin-I	D)Myoglobin and CK-BB
3.A 68-year-old obese woman (BMI = 32 kg/m2) presents with substernal chest pain and a history of recurrent angina pectoris and intermittent claudication. The following day, she develops a fever of 38°C (101°F). Results of laboratory studies include an elevated WBC count (13,000/µL), CK-MB of 6.6 ng/mL, and troponin-I of 2.5 ng/mL. ECG confirms a myocardial infarction of the left ventricular wall. Which of the following mechanisms is most likely responsible for the myocardial infarction in this patient?			
A)A.Coronary artery thrombosis	B)B.Coronary artery vasospasm	C).Decreased collateral blood flow	D)Paradoxical embolism
4.A 58-year-old man with a history of angina was brought to the emergency department after an acute onset of severe chest pain that started 40 minutes prior. Unlike previous episodes of chest pain, this one was unresponsive to nitroglycerin. His medical history is significant for hypertension, type 2 diabetes mellitus, and hyperlipidemia. His current medications include lisinopril, metformin, and simvastatin. His vital signs were: temperature 36.6°C (97.8°F), heart rate 88/min, respiratory rate 25/min, and blood pressure is 130/80 mm Hg. An ECG showed ST-segment elevation in leads V1-V3. He was treated with aspirin, oxygen, and morphine; additionally, myocardial reperfusion was performed. He was discharged within 1 week. He comes back 3 weeks later for a follow-up visit. Which of the following gross findings are expected to be found in patients myocardium?			
A)coagulation necrosis	B)Red granulation tissue	C)White scar tissue	D)D. Yellow infarct area

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