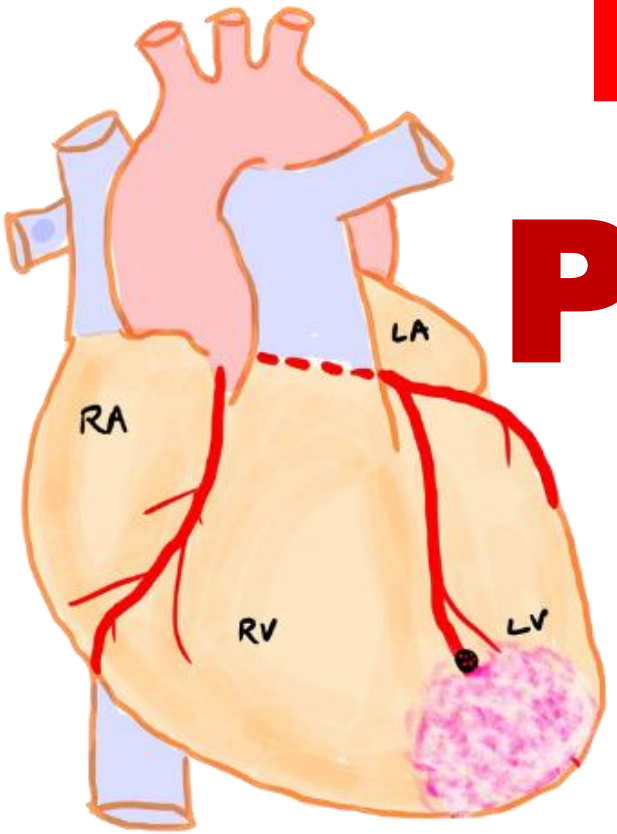
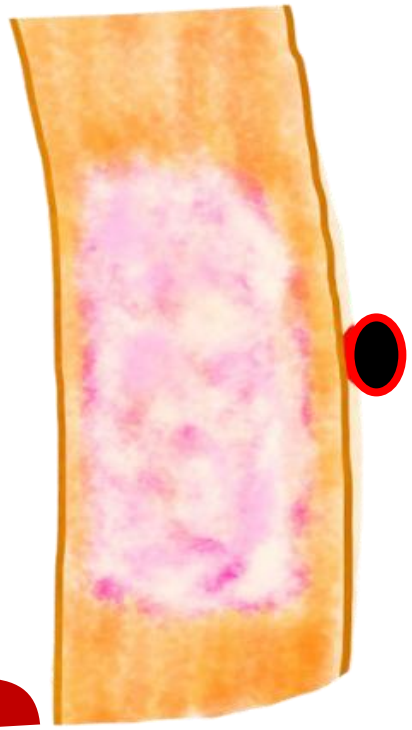
ISCHEMIC HEART DISEASE

Part - 2

MYOCARDIAL INFARCTION

PATHOGENESIS



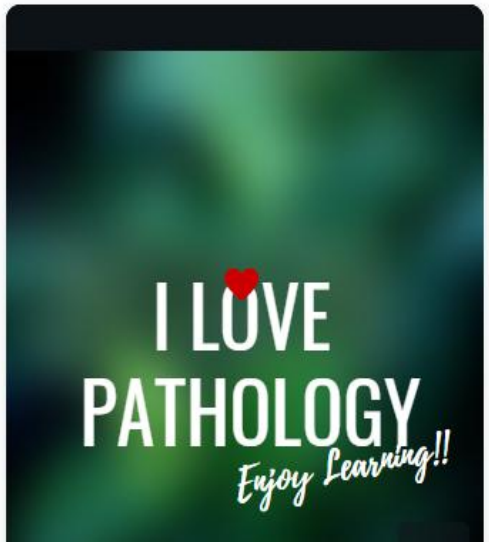


WHY PATHOLOGY?

CATEGORIES

Select Category

ILOVEPATHOLOGY



MYOCARDIAL INFARCTION

PATHOGENESIS

Overview

- Definition
- Epidemiology
- Pathogenesis
- Myocardial injury pattern

MYOCARDIAL INFARCTION

Death of cardiac muscle due to prolonged ischemia.

EPIDEMIOLOGY OF MYOCARDIAL INFARCTION

Occur at virtually any age

10% of MIs occur in people younger than 40 years of age

45% occur in people younger than 65 years of age.

Prevalence increases with age

EPIDEMIOLOGY OF MYOCARDIAL INFARCTION

Middle age: men> women

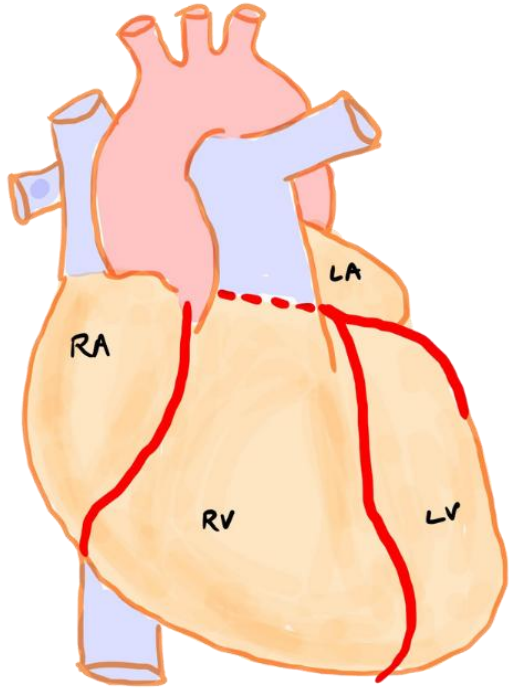
Women are protected during reproductive years

Not in postmenopausal women

Causes of myocardial ischemia

Obstructive atherosclerotic lesions in the epicardial coronary arteries

>90% of cases



PATHOGENESIS of myocardial Infarction

Erosion of atherosclerotic plaque

endothelial injury, intraplaque hemorrhage, or mechanical forces

↓

Exposure of sub endothelial collagen & necrotic plaque content to blood

↓

Adherence, aggregation & activation of platelets

→

Releases TXA2, ADP & seratonin

↑

further platelet aggregation and vasospasm

→

Coagulation activation by tissue factor

→

Growth of thrombus

→

Lumen occlusion

↓

Reduced blood supply

↓

Myocardial necrosis

Causes of myocardial Infarction

Other causes – 10% of cases

Coronary emboli	From left atrium in association with AF
	Mural thrombus, vegetations, prosthetic material etc
Vascular spasm	Drug induced . Eg - cocaine., ephedrine
Myocardial vessel inflammation	Vasculitis
Others	Amyloidosis, sickle cell disease, shock,. dissection etc…
	Congenital anomalous origin of a coronary artery

MYOCARDIAL RESPONSE

Myocardial ischemia

Limits tissue oxygenation

↓

Reduced ATP generation

Earliest biochemical change

Reduces the removal of metabolic wastes → Lactic acid accumulation

Reduces the availability of nutrients & oxygen → Decreased myocardial contractility

Systolic dysfunction

Much before myocardial death

Ultrastructural changes

Myofibrillar relaxation

Glycogen depletion,

Within Few minutes!

Cell and mitochondrial swelling

Reversible!

Severe ischemia (blood flow less than 10% of normal

20 to 30 minutes or longer

→

Irreversible changes (Myocardial Necrosis)

TIME IS MYOCARDIUM!

Rapid diagnosis

Time	Feature	Explanation
Seconds	**Onset of ATP depletion**	ATP levels drop rapidly due to lack of oxygen for energy production.
<2 minutes	**Loss of contractility**	Myocytes lose their ability to contract, leading to impaired heart function.
10 minutes	**ATP reduced to 50% of normal**	Significant drop in ATP levels affecting cell metabolism.
40 minutes	**ATP reduced to 10% of normal**	Severe energy depletion, increasing the risk of irreversible damage.
20–40 minutes	**Irreversible cell injury**	Cell membrane damage occurs, leading to leakage of cellular contents.
>1 hour	**Microvascular injury**	Damage to small blood vessels, worsening ischemia.

Earliest detectable feature of myocyte necrosis

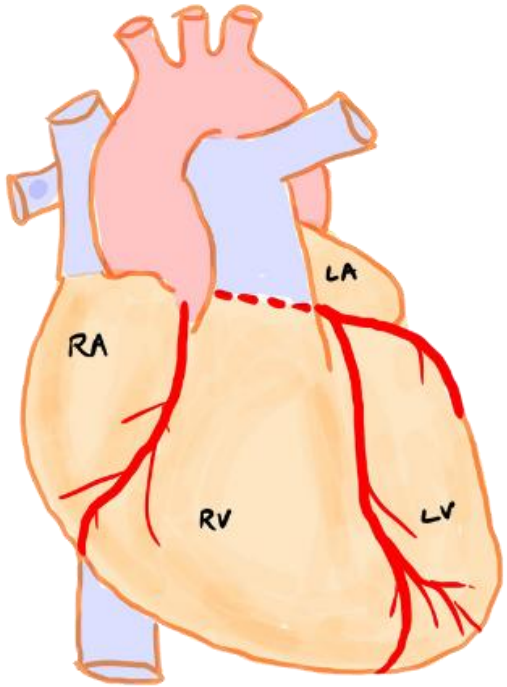
Integrity of the sarcolemmal membrane **DISRUPTED**

Leak of intracellular macromolecules (troponin and CK-MB) into interstitium

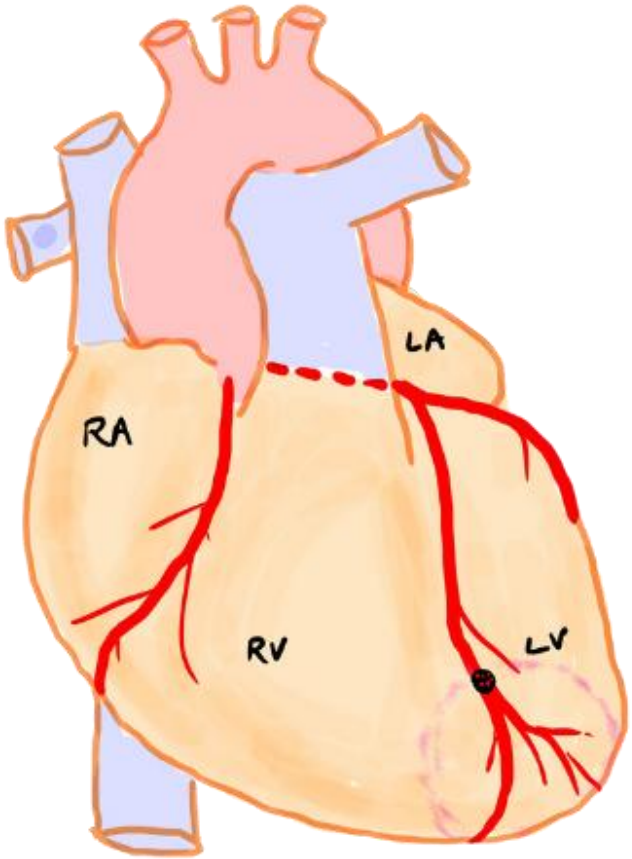
Microvasculature and lymphatics.

Early diagnosis of MI

Detection of irreversible myocardial damage

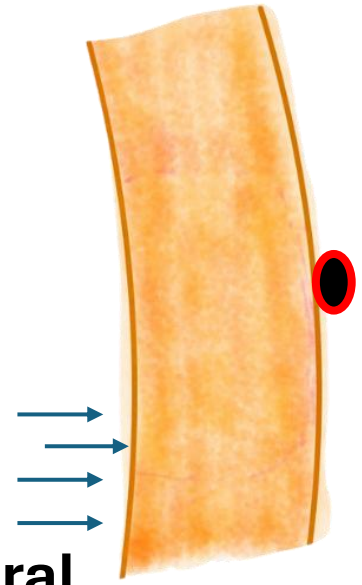

LA
RA
RV
LV

LOCATION OF INFARCT



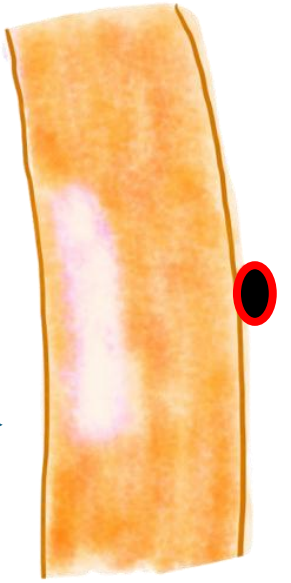
Sub endocardium

First to suffer irreversible damage



High intramural pressure *(which restricts blood flow).*

Delayed blood supply *(last to receive blood from epicardial vessels).*



Subendocardial Infarct

PROGRESSION

Reactive Oxygen Species (ROS)

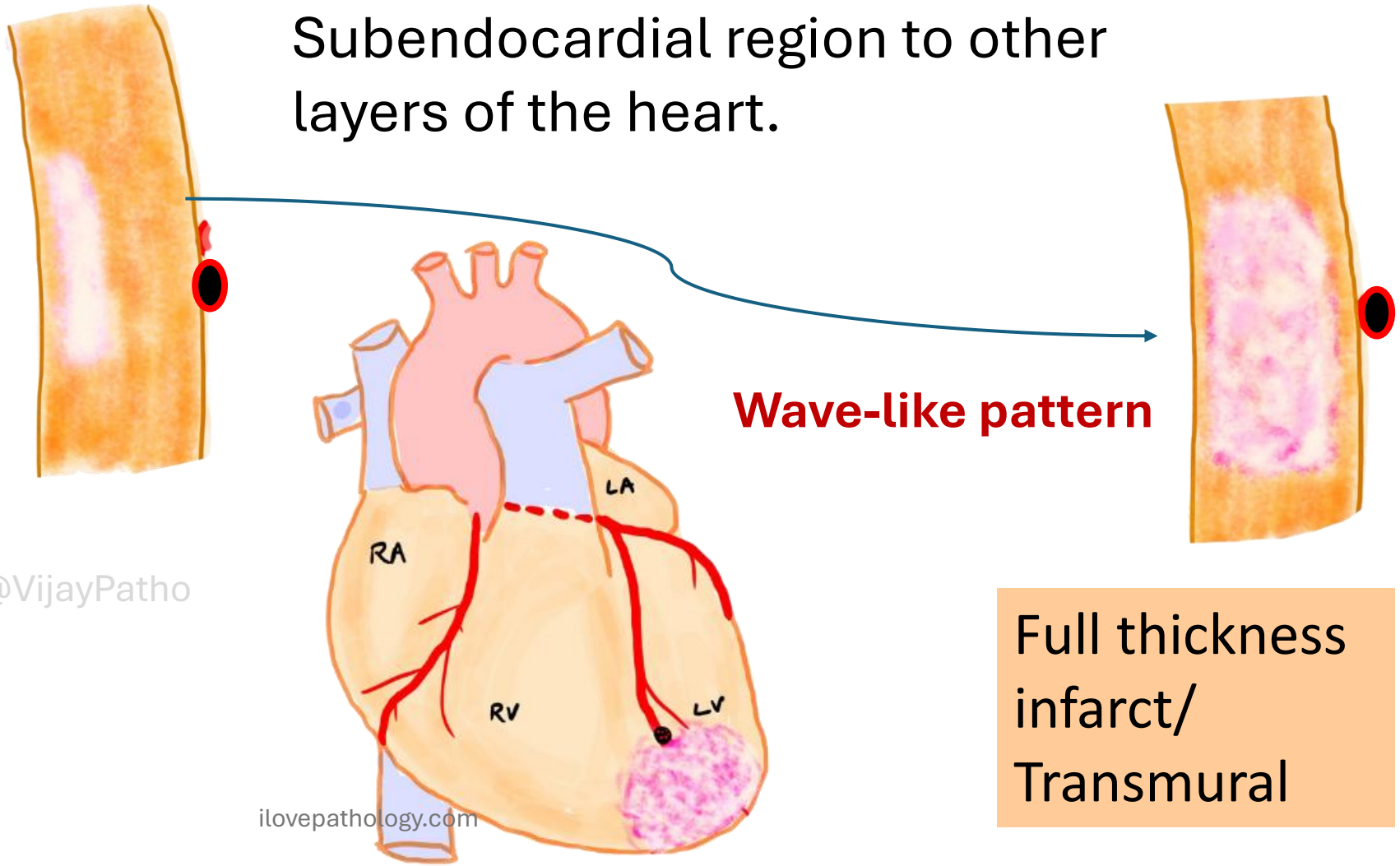
Tissue Edema:

Inflammatory Mediators

3- 6hrs

Cell death spreads **OUTWARD**

Subendocardial region to other layers of the heart.

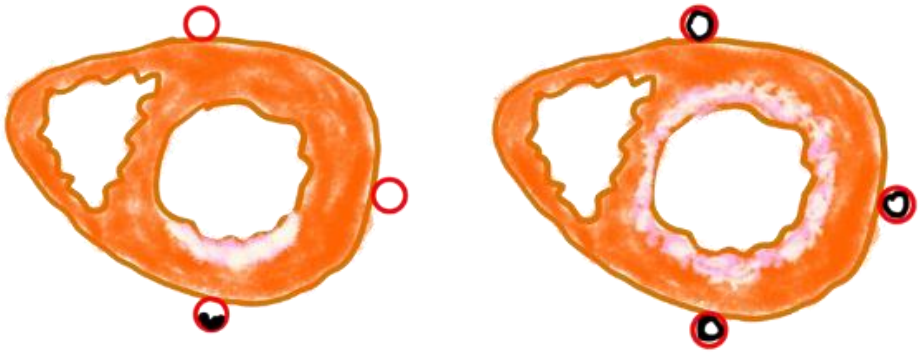


Wave-like pattern

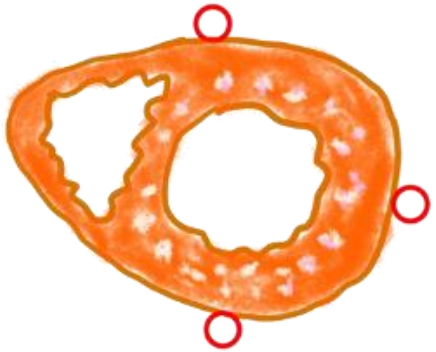
Full thickness infarct/ Transmural

SUB ENDOCARDIAL INFARCT

Patchy, Can be multifocal or circumferential



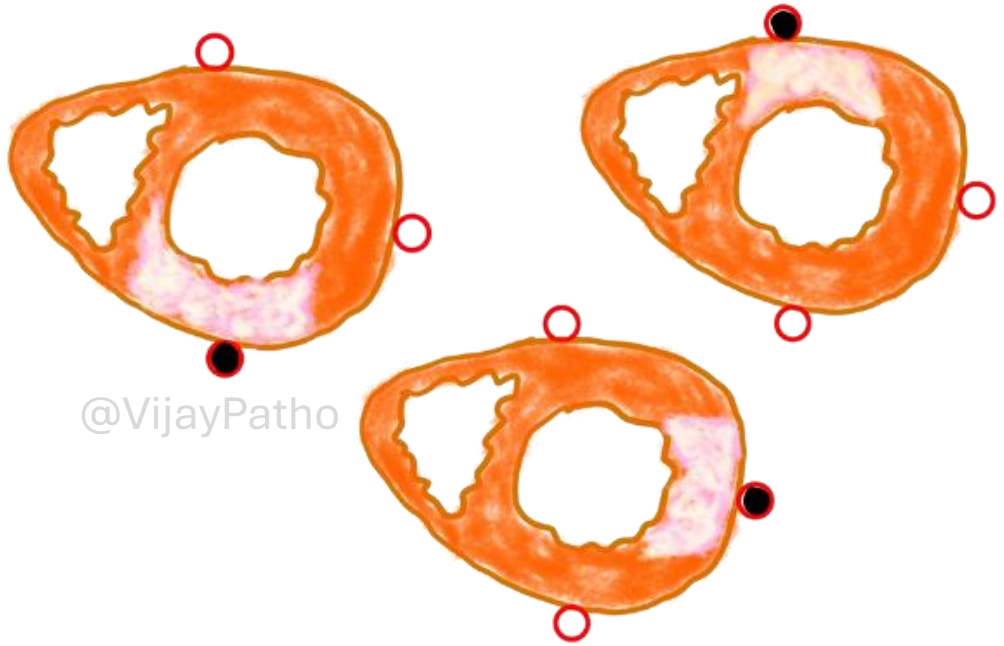
INTRAMURAL INFARCT



NON TRANSMURAL

TRANSMURAL INFARCT

Solid, unifocal and in the distribution of specific coronary artery



SUB ENDOCARDIAL INFARCT

Often due to hypotension or shock

No weakening of wall, so Do not form aneurysms or lead to ventricular rupture

TRANSMURAL INFARCT

Often causes shock

May cause aneurysms or lead to ventricular rupture

GROSS & MICROSCOPIC FEATURES

Next Video.....

Summary

- Definition
- Epidemiology
- Pathogenesis
- Myocardial injury pattern

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