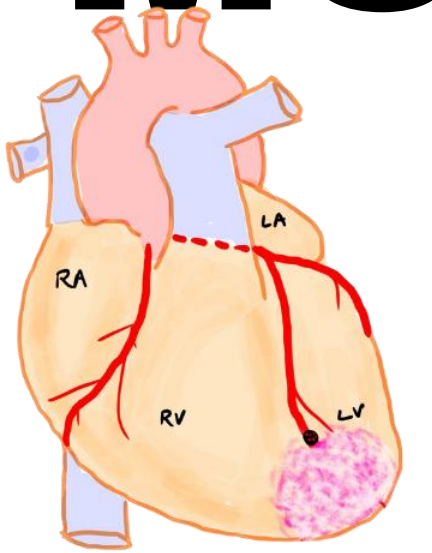
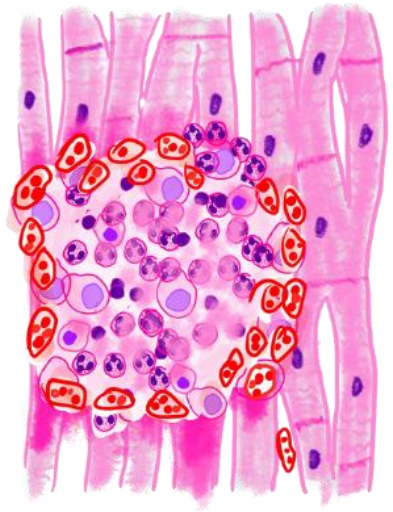
MYOCARDIAL INFARCTION

MORPHOLOGY



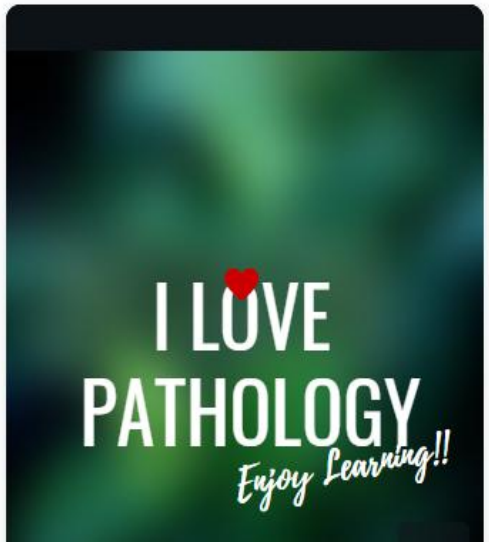


WHY PATHOLOGY?

CATEGORIES

Select Category

ILOVEPATHOLOGY

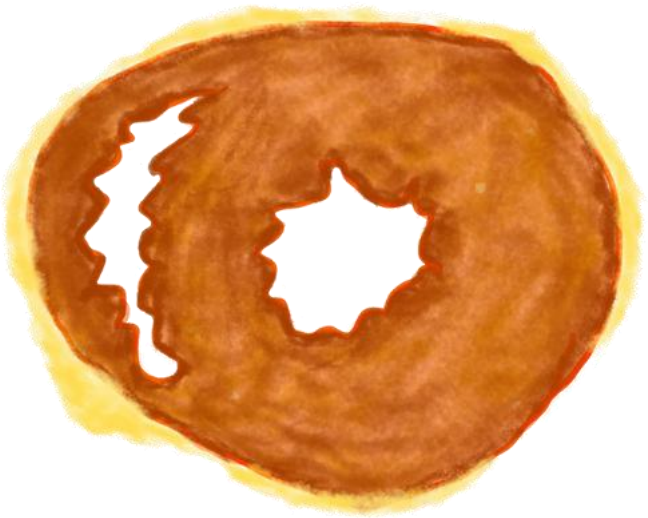


Overview

- Gross
- Microscopic findings

GENERAL FEATURES

Nearly all transmural infarcts



Involve a portion of the left ventricle (LV), including the free wall and ventricular septum.

Infarcts cover almost the entire perfusion zone except for a narrow subendocardial rim (~0.1 mm)

Preserved by diffusion of oxygen/nutrients.

Arterial Involvement and Associated Infarct Sites

Left Anterior Descending (40%-50%)

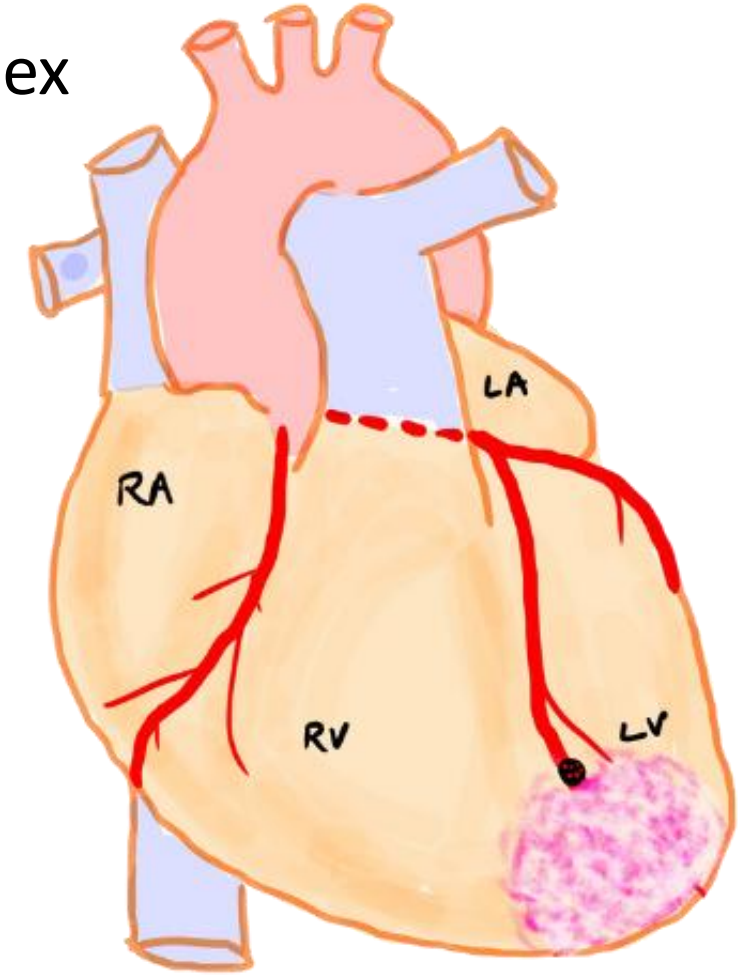
Anterior LV wall (near apex)
Anterior ventricular septum
Apex (circumferentially)

Right Coronary Artery (30%-40%)

Inferior/posterior LV wall
Posterior ventricular septum
Inferior/posterior right ventricular free wall (some cases)

Left Circumflex Artery (15%-20%)

Lateral LV wall (except apex)



OTHER FEATURES

Infarcts may also arise from lesions in

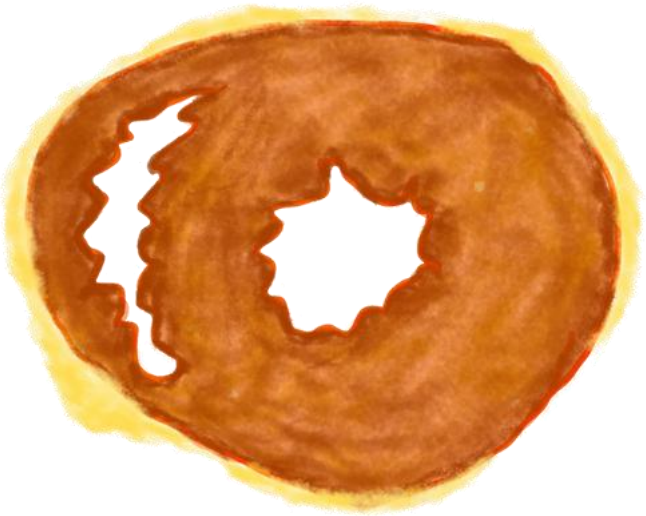
- Left main coronary artery
  - Diagonal branches of LAD
  - Marginal branches of LCX

Isolated right ventricular infarcts are rare (1%-3%).

0 – ½ HOUR

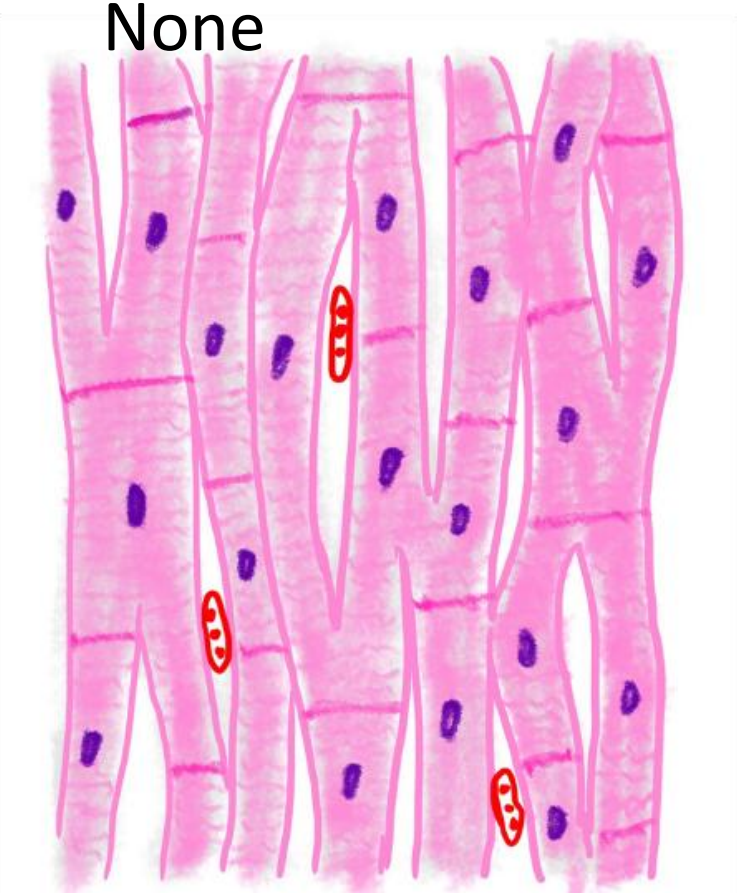
GROSS

None



MICROSCOPY

None



E M

Relaxation of myofibrils

Glycogen Loss

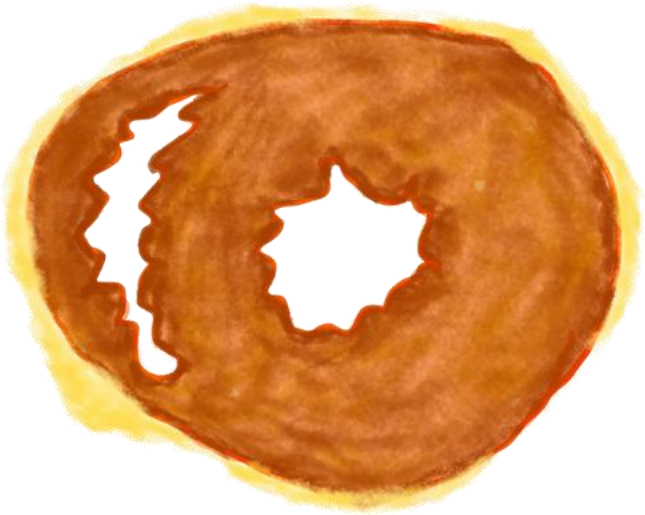
Mitochondrial swelling

REVERSIBLE

½ – 4 hours

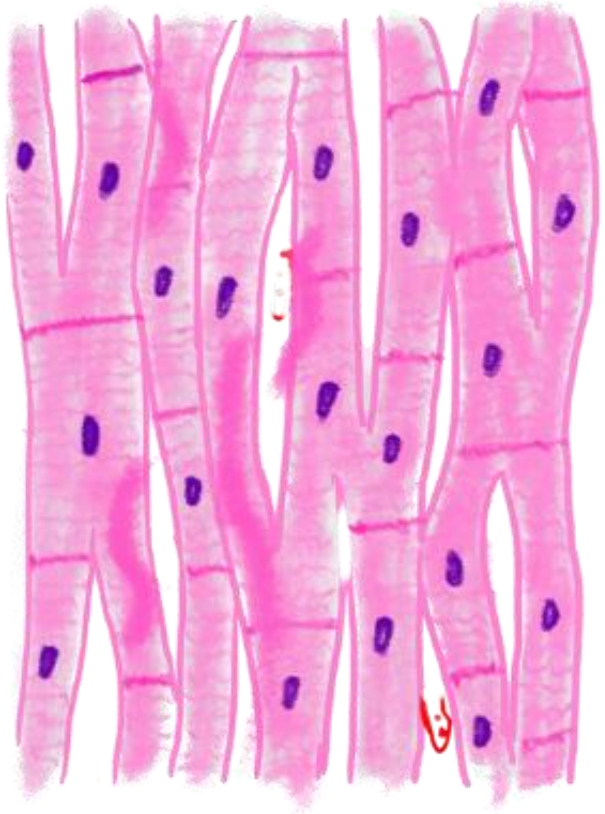
GROSS

None



MICROSCOPY

None



E M

Sarcolemmal disruption

mitochondrial amorphous densities

2 – 3 hours

Area of necrosis can still be highlighted by **special techniques**

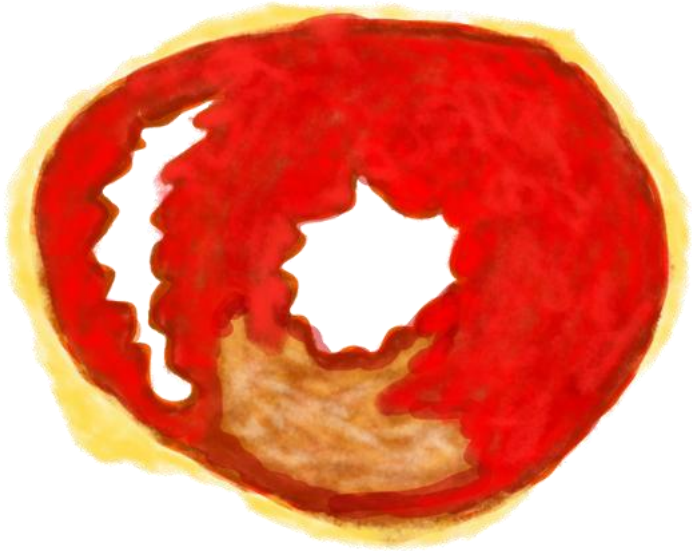
immersion of tissue slices

Triphenyltetrazolium chloride solution

Intact, noninfarcted myocardium

Preserved lactate dehydrogenase activity

BRICK RED COLOR



Infarcted myocardium
Dead cells

No dehydrogenase (leaked out)

UNSTAINED/ PALE ZONE

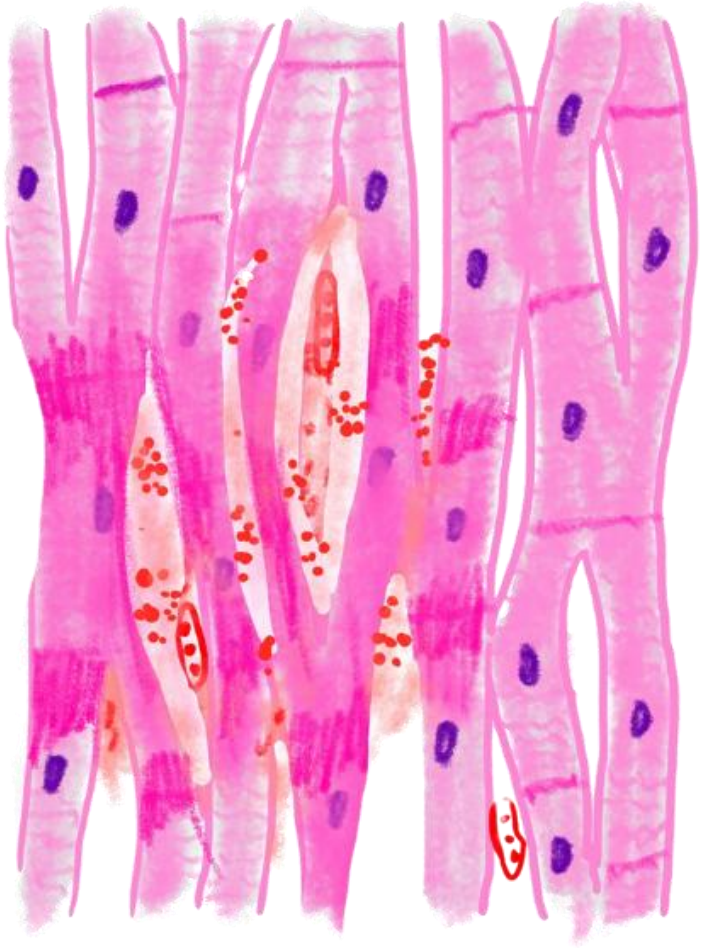
4- 12 hours

GROSS

Dark mottling

Occasionally

MICROSCOPY



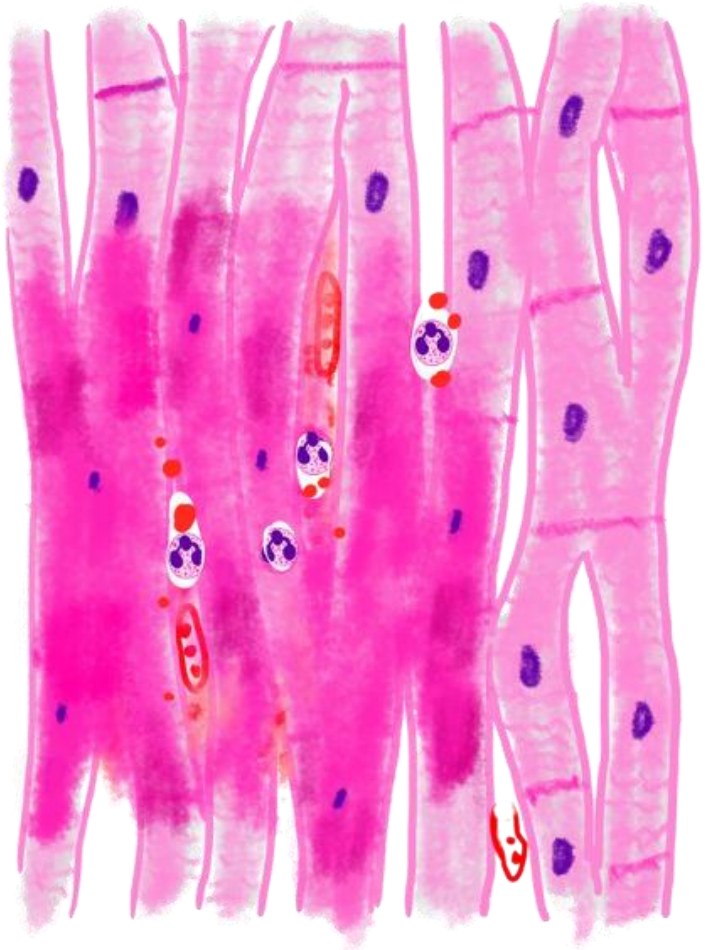
Early coagulative necrosis, edema, hemorrhage

12 – 24 hours

GROSS

Dark mottling

MICROSCOPY



Ongoing coagulative necrosis;

Pyknosis of nuclei;
Myocyte hypereosinophilia;

Marginal contraction band necrosis

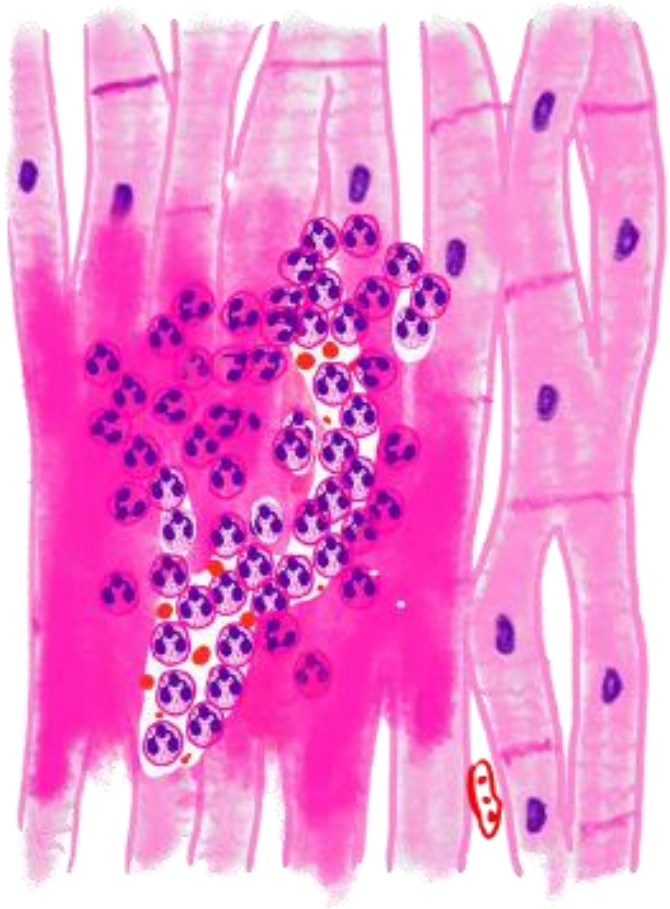
Early neutrophilic infiltrate

24 – 72 hours/ 1-3 days

GROSS

Mottling with yellow-tan infarct center

MICROSCOPY



coagulative necrosis;

with loss of nuclei and striations

brisk interstitial infiltrate of neutrophils

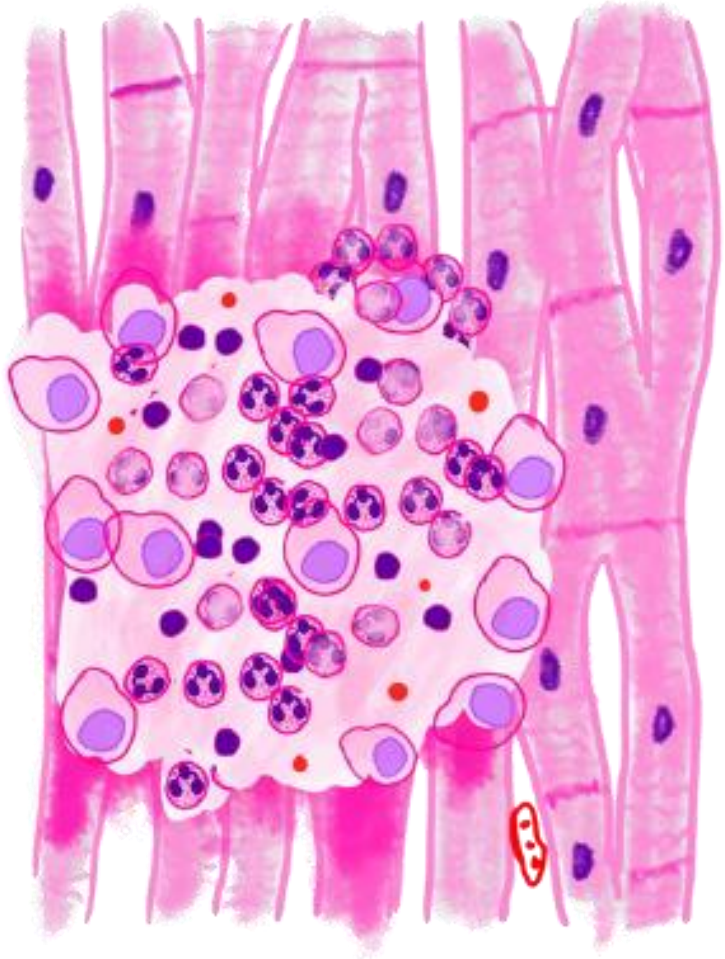
3 – 7 days

GROSS

Hyperemic border;

central yellow-tan softening

MICROSCOPY



Beginning disintegration of dead myofibers,

Dying neutrophils

early phagocytosis of dead cells by macrophages at infarct border

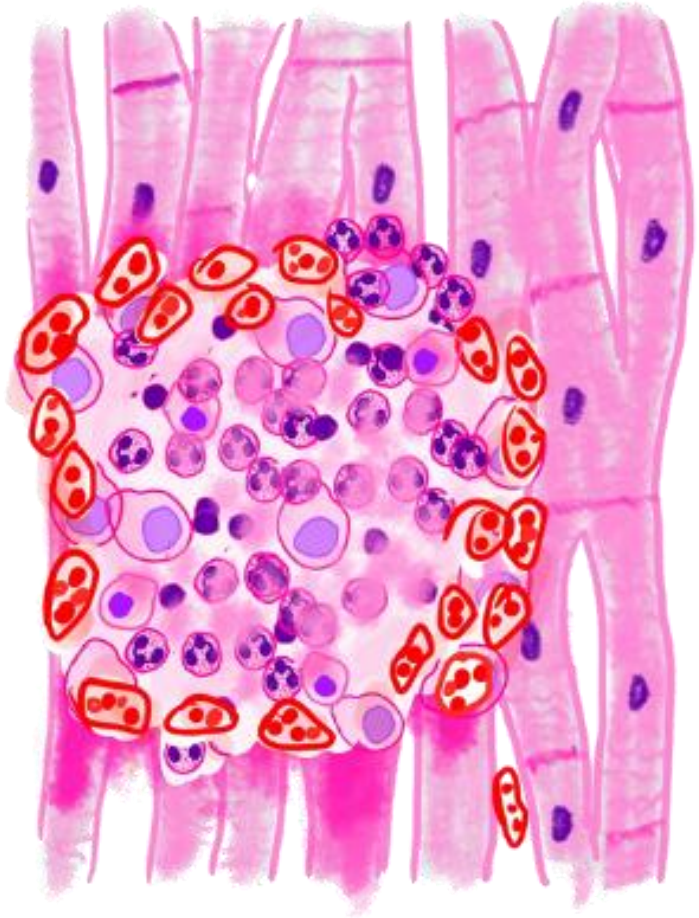
7 – 10 days

GROSS

Maximally yellow-tan and soft,

Depressed red-tan margins

MICROSCOPY



Well-developed phagocytosis of dead cells;

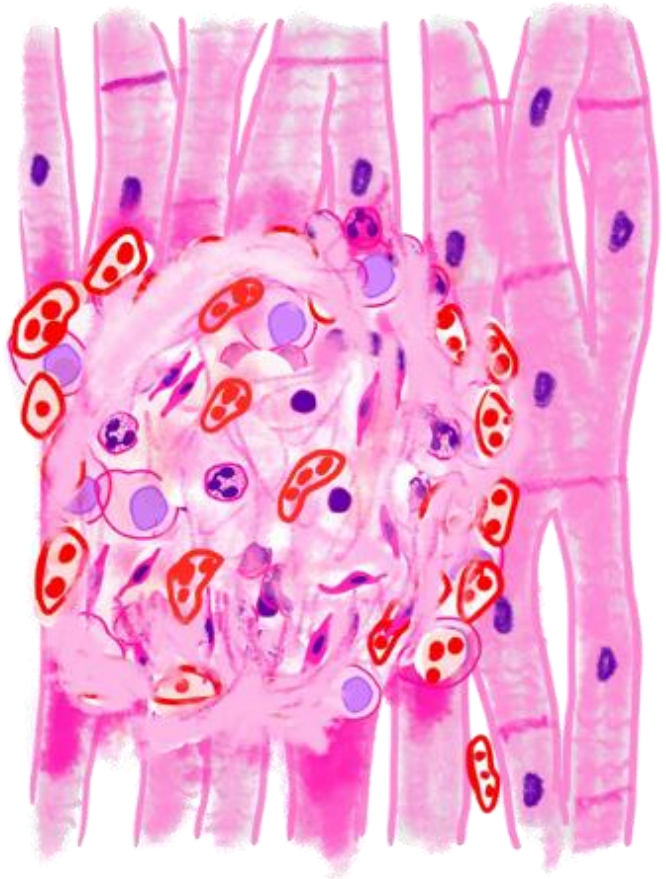
Granulation tissue at margins

10 – 14 days

GROSS

Red-gray depressed infarct borders

MICROSCOPY



Well-established granulation tissue with new blood vessels and collagen deposition

2- 8 weeks

GROSS

Gray-white scar,

Progressive from Border toward core of infarct

MICROSCOPY

Increased collagen deposition,

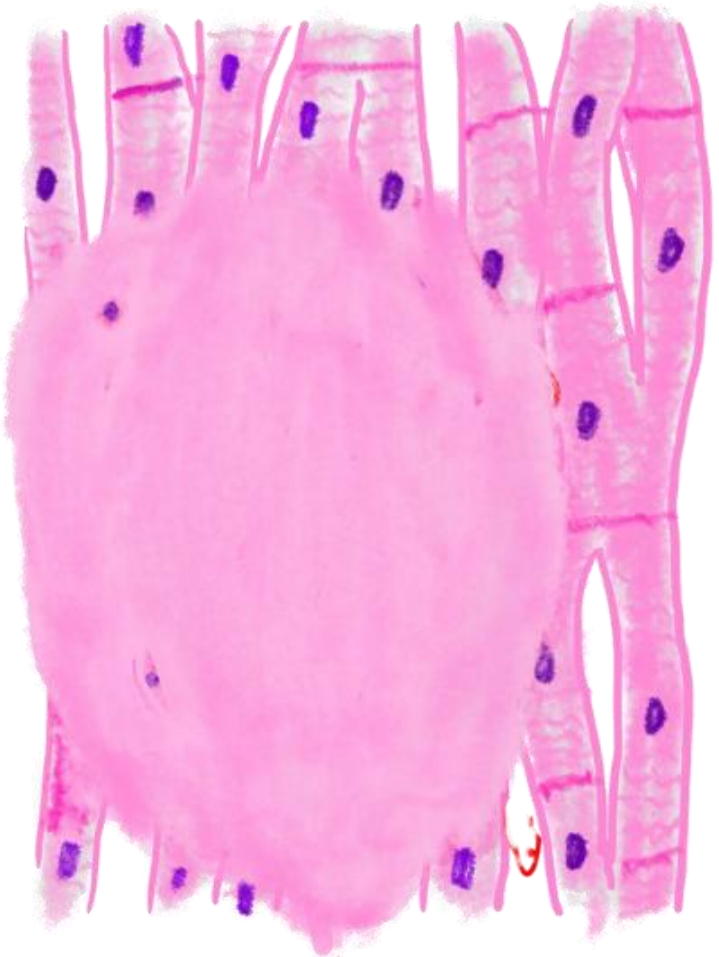
with decreased cellularity

>8 weeks/ >2 months

GROSS

Scarring complete

MICROSCOPY



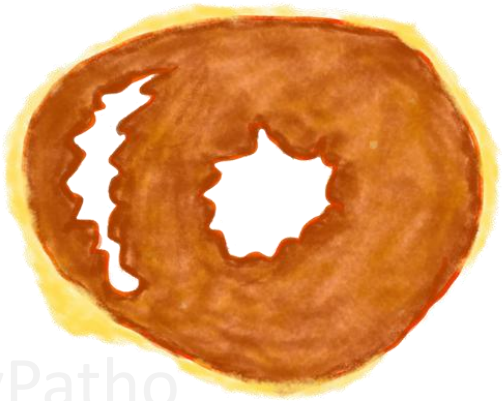
Dense collagenous scar

Morphologic Changes in Myocardial Infarction: **GROSS**

0– 1/2 hour

None

1/2 –4 hours



None

4–12 hours

Dark mottling

12–24 hours

Dark mottling

1–3 days

Mottling with yellow-tan infarct center

3–7 days

Hyperemic border; central yellow-tan softening

7–10 days

Maximally yellow-tan and soft, with depressed red-tan margins

10–14 days

Red-gray depressed infarct borders

2–8 weeks

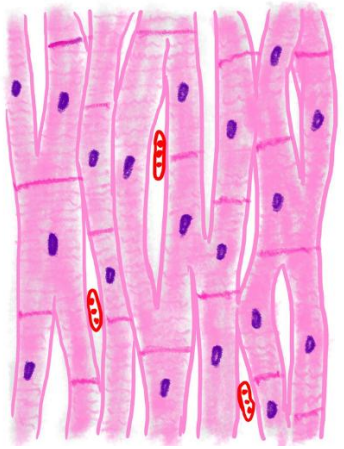
Gray-white scar, progressive from border toward core of infarct

>2 months

Scarring complete

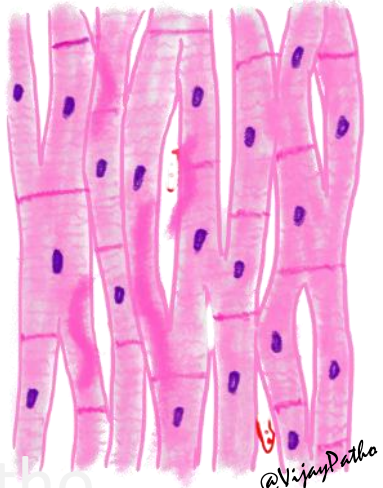
Morphologic Changes in Myocardial Infarction: **MICROSCOPY**

0– 1/2 hour



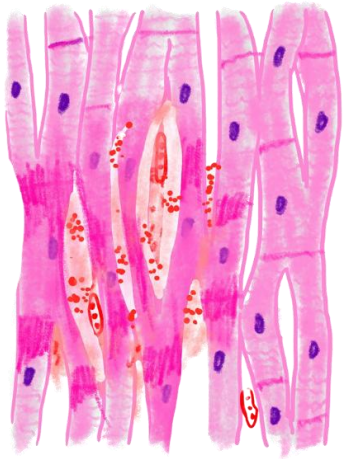
None

1/2 –4 hours



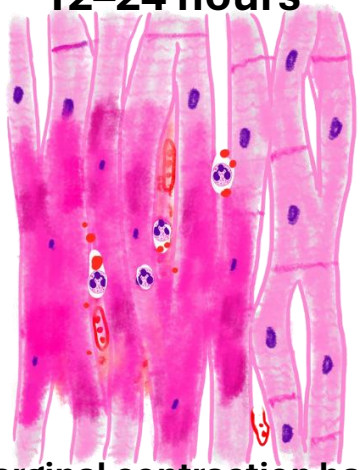
None; variable waviness

4–12 hours



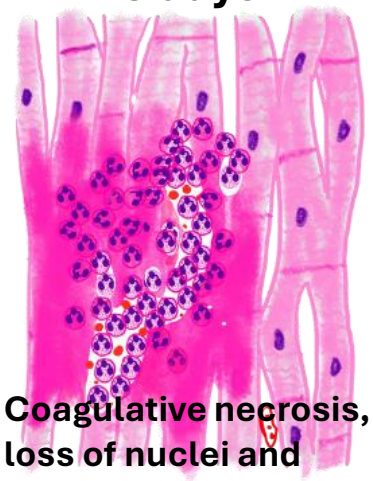
Early coagulative necrosis; edema; hemorrhage

12–24 hours



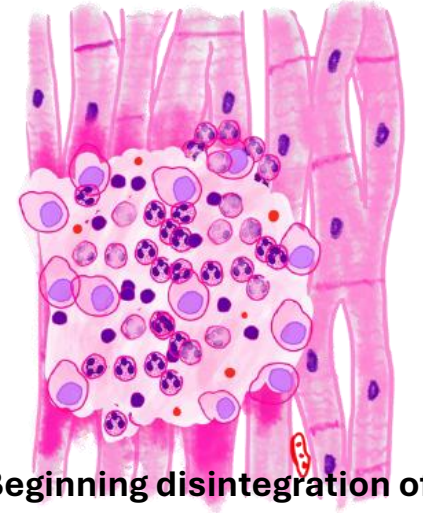
Marginal contraction band necrosis; early neutrophilic infiltrate

1–3 days



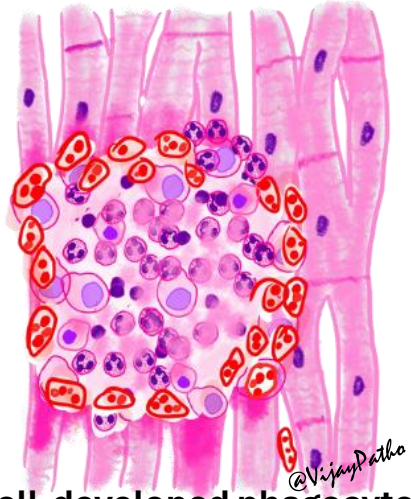
Coagulative necrosis, with loss of nuclei and striations; brisk interstitial infiltrate of neutrophils

3–7 days



Beginning disintegration of dead myofibers, with dying neutrophils; Phagocytosis (macrophages at border)

7–10 days



Well-developed phagocytosis of dead cells; granulation tissue at margins

10–14 days

Well-established granulation tissue with new blood vessels and collagen deposition

2–8 weeks

Increased collagen deposition, with decreased cellularity

>2 months

Dense collagenous scar

Why Understanding Gross and Microscopic Changes in MI is Clinically Important ?

MI progresses in predictable histological stages.

Each stage correlates with clinical complications (e.g., neutrophils = pericarditis, macrophages = rupture risk

Helps us understand the disease process, complications, and outcomes.

ISCHEMIA-REPERFUSION INJURY

why treating an MI can sometimes make things worse!

Next Video.....

Summary

- Gross
- Microscopic findings

http://ilovepathology.com

@VijayPatho

ilovepathology

ilovepathology