The Cardio-VASCULAR System

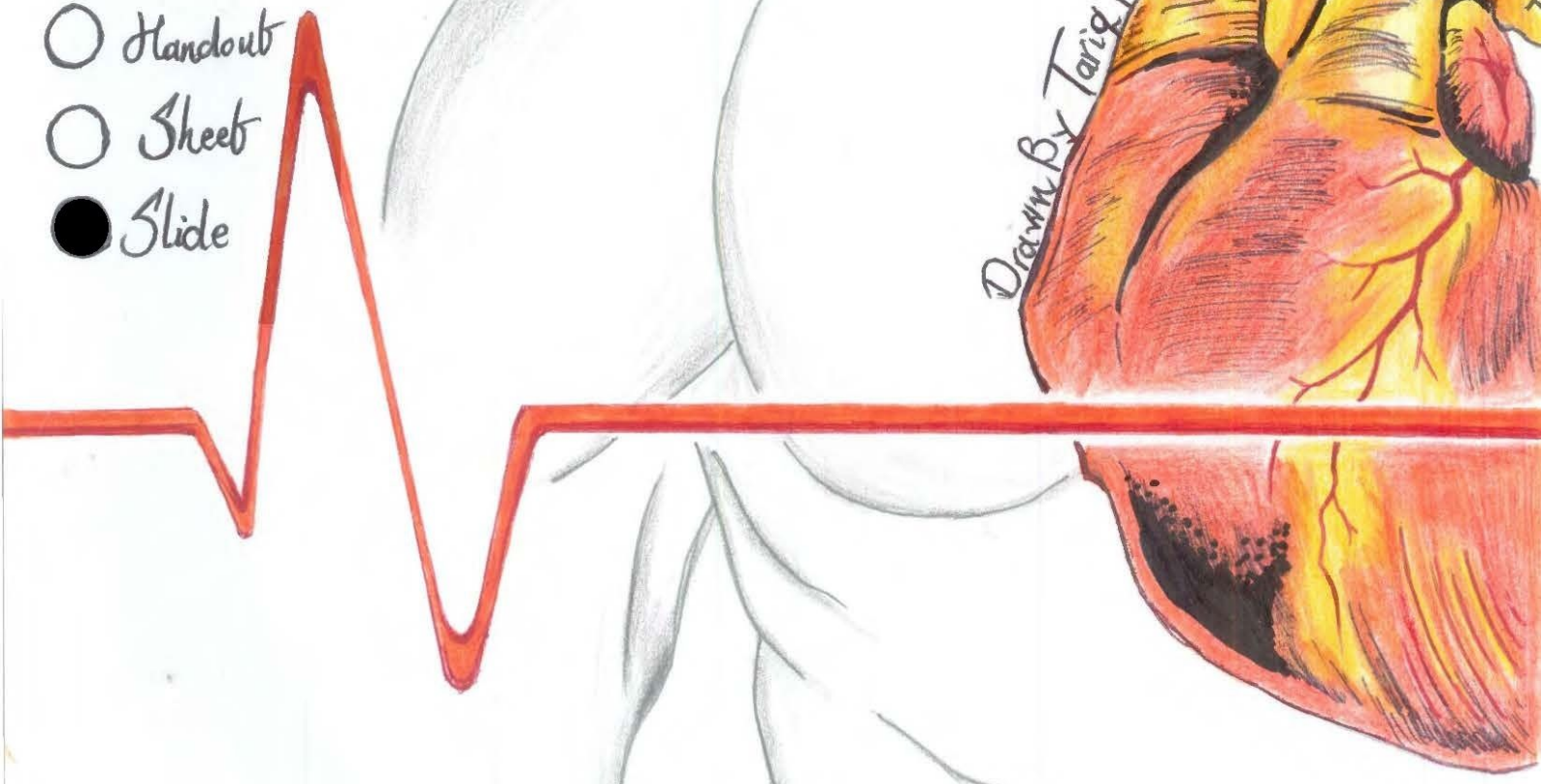
- ○ Anatomy
- ○ Histology
- ● Pathology
- ○ Pharmacology
- ○ Physiology
- ○ Microbiology

Lec #: 2

Dr. Name: Dr.Nisreen

Done By:

- ○ Handout
- ○ Sheet
- ● Slide



EMBOLISM:

- An embolus is a detached intravascular solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin

- *99% due to dislodged thrombus*

- *Types:*

1. *Thromboembolism*
2. *Fat embolism*
3. *Air embolism*
4. *Nitrogen embolism*
5. *Cholesterol embolism*
6. *Amniotic fluid embolism*

- Emboli result in partial or complete vascular occlusion.
- The consequences of thromboembolism include ischemic necrosis (*infarction*) of downstream tissue

Embolus derived from a lower extremity deep venous thrombosis and now impacted in a pulmonary artery branch

Pulmonary thromboembolism

- *95% originate from deep veins of L.L*
- *Saddle embolus: at bifurcation of Pulmonary artery*
- *Paradoxical embolus: Passage of an embolus from venous to systemic circulation through IAD, IVD*

Clinical consequence of Pulmonary thromboembolism :

- Most pulmonary emboli (60% to 80%) are clinically silent because they are small

a. ***Organization: 60 – 80 %***
b. ***Pulmonary infarction***
c. ***Sudden death, Right ventricle failure, CV collapse when more than 60 % of pulmonary vessels are obstructed.***
d. ***Pumonary hemorrhage: obstruction of medium sized arteries.***
e. ***Pulmonary Hypertension and right ventricular failure due to multiple emboli over a long time.***

Systemic thromboembolism

- Emboli traveling within the arterial circulation
- 80% due to intracardiac mural thrombi

2/3 Lt. ventricular failure

¼ Lt. atrial dilatation

Ulcerated atherosclerotic plaque,

Aortic aneurysm

valvular regetation

- The major targets are:

1. *Lower limbs 75%*
2. *Brain 10%*
3. *Intestine*
4. *Kidneys*
5. *Spleen*

- **Fat embolism**

- Causes

1. *Skeletal injury (*fractures of long bones)
2. *Adipose tissue Injury*

- Mechanical obstruction is exacerbated by free fatty acid release from the fat globules, causing local toxic injury to endothelium.

- In skeletal injury, fat embolism occurs in 90% of cases, but only 10% or less have clinical findings

- ***Fat embolism syndrome*** *is characterized by*

A. *Pulmonary Insufficiency*
B. *Neurologic symptoms*
C. *Anemia*
D. *Thrombocytopenia*
E. *Death in 10% of the case*

- Symptoms appears 1-3 days after injury

Tachypnea, Dyspnea, Tachycardia and Neurological symptoms

- **Air Embolism**
- causes:

1. *Obstetric procedures*
2. *Chest wall injury*
3. *Decompression sickness: in* Scuba and deep-sea divers ((nitrogen))

- *More then 100ml of air is required to produce clinical effect.*
- Clinical consequence

1. ***Painful joints**: due to rapid formation of gas bubbles within Sk. Muscles and supporting tissues.*
2. *Focal **ischemia in brain** and heart*
3. ***Lung edema**, Hemorrhage, **atelectasis**, **emphysema**, which all lead to Respiratory distress. (chokes)*
4. ***caisson disease**:* gas emboli in the bones leads to multiple foci of ischemic necrosis, usually the heads of the femurs, tibias, and humeri

Amniotic fluid embolism

- Mortality Rate = 20%-40%
- Uncommon complication of labor
- due to infusion of amniotic fluid into maternal circulation via tears in placental membranes and rupture of uterine veins.
- sudden severe dyspnea, cyanosis, and hypotensive shock, followed by seizures, DIC and coma
- Finding:

  Squamous cells, languo hair, fat, mucin .....etc within the pulmonary microcirculation

INFARCTION

- An infarct is an area of ischemic necrosis caused by occlusion of either the arterial supply or the venous drainage in a particular tissue
- Nearly 99% of all infarcts result from thrombotic or embolic events
- other mechanisms include: local vasospasm, expansion of an atheroma, extrinsic compression of a vessel (e.g., by tumor); vessel twisting (e.g., in testicular torsion or bowel volvulus; and traumatic vessel rupture

Morphology of infarcts

- infarcts may be either red (hemorrhagic) or white (anemic) and may be either septic or bland
- All infarcts tend to be wedge shaped, with the occluded vessel at the apex and the periphery of the organ forming the base
- The margins of both types of infarcts tend to become better defined with time
- The dominant histologic characteristic of infarction is **ischemic coagulative necrosis**
- most infarcts are ultimately replaced by scar. The brain is an exception, it results in liquefactive necrosis

Red infarcts:

- occur in

(1) venous occlusions (such as in ovarian torsion)

(2) loose tissues (like lung) that allow blood to collect in the infarcted zone;

(3) tissues with dual circulations (lung and small intestine),

(4) previously congested tissues because of sluggish venous outflow

(5) when flow is re-established to a site of previous arterial occlusion and necrosis

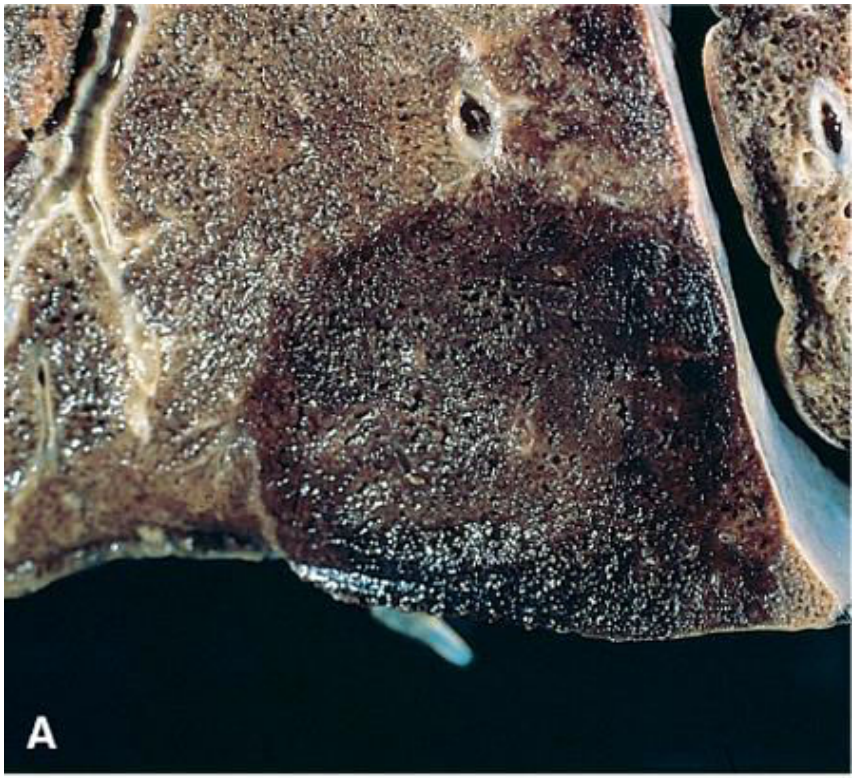
White infarcts

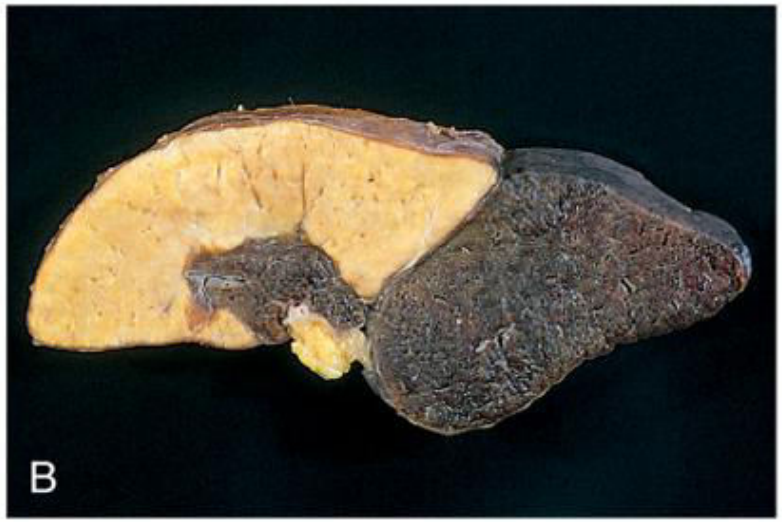
- occur with:

1) arterial occlusions
2) solid organs (such as heart, spleen, and kidney).

Septic infarctions

- occur when **bacterial vegetations** from a heart valve embolize or when **microbes seed an area of necrotic tissue.**
- the infarct is converted into an **abscess**, with a correspondingly greater inflammatory response

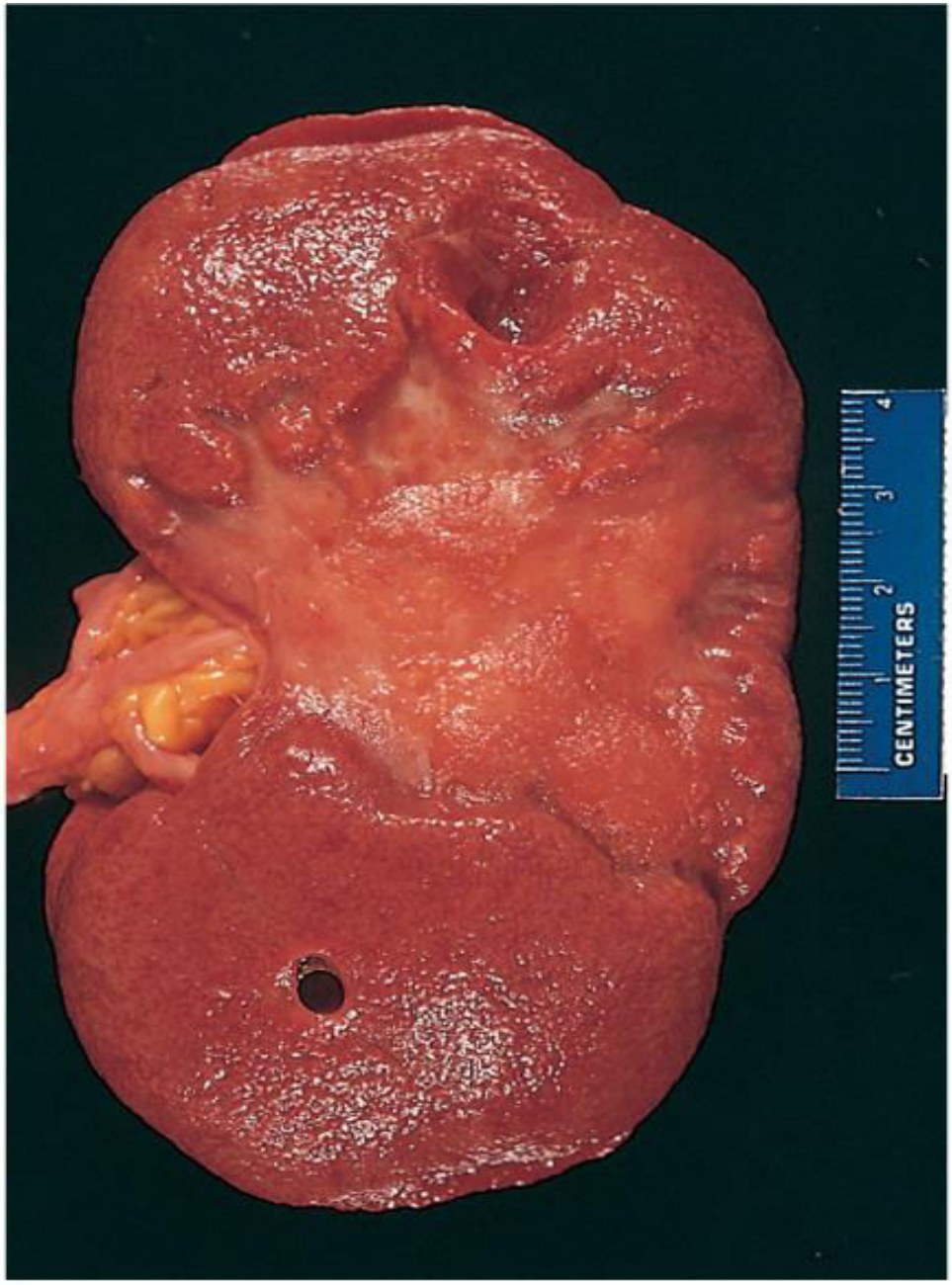




Red and white infarcts.

A→lung

B→spleen



kidney infarct, now replaced by a large fibrotic scar

Factors That Influence Development of an Infarct

- ***nature of the vascular supply***
- ***rate of development of the occlusion*** ***(***collateral circulation)
- ***vulnerability to hypoxia***

- Neurons undergo irreversible damage → 3 to 4 minutes of ischemia.
- Myocardial cells die after only 20 to 30 minutes of ischemia

- ***the oxygen content of blood***