EMBOLISM AND INFARCTION

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**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.

**Types (according to composition of emboli):**

1. **Thromboembolism:** 99% (from dislodged thrombus)
2. **Fat embolism**
3. **Air / Nitrogen embolism**
4. **Amniotic fluid embolism**

1%
2 TYPES / SIDES OF CIRCULATION: VENOUS & ARTERIAL (SYSTEMIC)

Figure 9-1 Regional vascular specializations. Although all vessels share the same general constituents, the thickness and composition of the various layers differ as a function of hemodynamic forces and tissue requirements.
Origin of most venous thrombi = lower limbs

Target of most venous thrombi = lungs

Figure 9-1 Regional vascular specializations. Although all vessels share the same general constituents, the thickness and composition of the various layers differ as a function of hemodynamic forces and tissue requirements.
Origin of most arterial thrombi = heart chambers

Target of most arterial thrombi = lower limbs (75%)
- Types according to site of origin: **venous** and **arterial** (systemic) emboli

- Emboli result in partial or complete vascular occlusion.

- The consequences of thromboembolism: ischemic necrosis (**infarction**) of downstream tissue
VENOUS THROMBESIS

1. A piece of a blood clot (embolus) breaks free
2. Embolus travels through the vein
3. Embolus gets stuck in a blood vessel of the lung

Tissue not getting blood
PULMONARY THROMBOEMBOLISM

- 95% originate from deep veins of Lower Limbs (DVT)

- Special terms:

1- **Saddle embolus**: large embolus occluding the bifurcation of pulmonary artery trunk (fatal)

2- **Paradoxical embolus**: Passage of embolus from venous to systemic circulation through ASD or VSD
Embolus derived from a lower extremity deep venous thrombosis and now impacted in a pulmonary artery branch.
CLINICAL CONSEQUENCE OF PULMONARY THROMBOEMBOLISM:

- 60%-80% pulmonary emboli are clinically asymptomatic (small) → Organization

- If large → Pulmonary infarction

- >60% of pulmonary vessels are obstructed → RVF, CV collapse → Sudden death

- Obstruction of medium sized arteries → Pulmonary hemorrhage

- If multiple emboli (showers of emboli) over a long time → Pulmonary Hypertension and right ventricular failure
ARTERIAL EMBOLI

2. Blood clot travels in the bloodstream

3. Clot blocks an artery in the brain, causing stroke
Systemic (arterial) thromboembolism

- Emboli traveling within the arterial circulation
- 80% due to intracardiac mural thrombi (origin)
  - causes: -2/3 Lt. ventricular failure
    - ¼ Lt. atrial dilatation
    - Ulcerated atherosclerotic plaque
    - Aortic aneurysm
    - valve vegetation …etc
- The major targets are:

  Lower limbs 75%; Brain 10%; Intestine; Kidneys; Spleen; etc… (any organ that has arterial supply!)
- **Fat embolism**
  
  - **Causes:**
    1. *Skeletal injury* (fractures of long bones)
    2. *Adipose tissue Injury* (massive fat necrosis like acute pancreatitis, etc...)

- **Results:**
  1. Mechanical obstruction of vessels
  2. free fatty acid release from fat globules → 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
FAT EMBOLUS = FAT GLOBULES + HEMATOPOIETIC CELLS
Fat embolism ‘syndrome’ is characterized by:

- **Pulmonary Insufficiency** (rapid breathing; shortness of breath)
- **Neurologic symptoms** (mental confusion; lethargy; coma)
- **Petechial rash** (pinpoint rash, found on chest, head, and neck area due to bleeding under skin)
- **Fever**
- **Anemia**
- **Thrombocytopenia**
- **Death in 10% of the case**

- Symptoms appears 1-3 days after injury
Air Embolism

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

Note: More than 100ml of air is required to produce clinical effect!
DECOMPRESSION SICKNESS

The Bends

0 metres
Pressure = 1 atm

A slow return to the surface lets the nitrogen return to the lungs where it is breathed out

10 metres
Pressure = 2 atm

Swimming up too quickly doesn’t give the nitrogen enough time to leave the blood - instead it can form painful bubbles

Nitrogen moves from high pressure in the lungs into the blood (low pressure)
AIR EMBOLISM - CLINICAL CONSEQUENCE

1. Painful joints: rapid formation of gas bubbles within Skeletal Muscles and supporting tissues.

2. Focal ischemia in brain and heart

3. Respiratory distress (chokes) $\rightarrow$ Lung edema, hemorrhage, atelectasis, emphysema

4. Caisson disease: in scuba divers; gas emboli in the bones leads to multiple foci of ischemic necrosis, usually the heads of the femurs, tibias, and humeri
Amniotic fluid embolism

- High Mortality Rate = 20%-40%
- Very rare complication of labor
- infusion of amniotic fluid into maternal circulation via tears in placental membranes and rupture of uterine veins.

- Symptoms: sudden severe dyspnea, cyanosis, ARDS, and hypotensive shock, followed by seizures, DIC and coma

- Microscopic Findings upon autopsy:
  fetal squamous cells, languo hair, fat, mucin .....etc within the maternal pulmonary microcirculation
Amniotic fluid embolism (AFE) is a rare but lifethreatening condition. Amniotic fluid escapes the uterus, enters the uterine vasculature, and travels through the veins to the heart and lungs. The fluid can cause pulmonary microvascular obstruction, leading to decreased oxygen exchange. This results in pulmonary edema,ARDS, and low oxygen saturation. The condition affects up to 1 in 2000 pregnancies in the United States and can be triggered by various factors such as cesarean section, labor, and delivery. Early recognition and treatment are crucial to improve outcomes.
AMNIOTIC FLUID EMBOLUS: KERATIN AND FETAL SQUAMOUS CELLS IN PULMONARY ARTERIOLES
INFARCTION

- infarct = an area of **ischemic necrosis** caused by occlusion of arterial supply or venous drainage in a tissue
- 99% of 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
- tend to be wedge shaped (occluded vessel at the apex and the periphery of the organ forming the base)
- margins of infarcts tend to become better defined with time
- histologic hallmark of infarction is ischemic coagulative necrosis
- note: The brain is an exception (liquefactive necrosis)
- most infarcts are ultimately replaced by scar
**Red infarcts:**

- **occur in any of the following scenarios:**
  1. **venous** occlusions (e.g. ovarian torsion)
  2. **loose** tissues (e.g. lung) that allow blood to collect in the infarcted zone
  3. tissues with **dual** circulations (e.g. 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).

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**Septic infarctions:**

- occur when infarct is superimposed by infection;
- examples:
  1) **infected vegetations**
  2) microbes seed an area of necrotic tissue
- infarct is converted into **abscess** with a greater inflammatory response
Red and white infarcts.

A → lung
B → spleen
Kidney infarct replaced by a large fibrotic scar
Factors That Influence Development of an Infarct

- nature of vascular supply
- rate of occlusion development (collateral circulation)
- tissue vulnerability to hypoxia
  - Neurons undergo irreversible damage $\Rightarrow$ 3 to 4 minutes of ischemia.
  - Myocardial cells die after only 20 to 30 minutes of ischemia
- oxygen content of blood