

Hemodynamic Disorders, Thromboembolism and Shock

LEC.3

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Clinical significance of venous & arterial thrombosis

- Thrombi are significant because they (1) can obstruct vessels and (2) can embolize;
- The relative importance depends on the site. Thus, although venous thrombi can cause distal congestion and edema, embolization is more clinically significant (e.g. embolize from deep leg vein to lung and cause death). Conversely, although arterial thrombi can embolize, vascular obstruction (e.g., causing myocardial or cerebral infarctions) is much more important.

Venous Thrombosis (Phlebothrombosis)

- Most venous thrombi occur in the superficial or the deep veins of the leg.
- ❖ **Superficial venous thrombi:**
 - Usually arise in the **saphenous system, particularly in the setting of varicosities.**
 - Can cause; Local pain, congestion and swelling from impaired venous outflow.
 - Predisposing to infection & ulceration of overlying skin (varicose ulcers).
 - **Rarely embolize.**
- ❖ **Deep venous thrombi:**
 - Occur in large leg veins at or above the level of knee joint (popliteal, femoral & iliac veins).
 - **More serious because they are prone to embolize to the lungs** (pulmonary embolization).
 - Can result in pain and edema.
 - Venous obstruction is usually offset by collateral flow, and deep vein thromboses are asymptomatic in approximately 50% of patients, being recognized only after embolization.

Risk conditions for deep venous thrombosis (DVT):

1. **Advanced age, bed rest, or immobilization**, thereby diminishing the milking action of muscles in the lower leg and slowing venous return
2. **Congestive heart failure (CHF)** (cause stasis..... DVT).
3. **Trauma, surgery, & burns** (reduced physical activity, injury to blood vessels, & release prothrombotic factors & decrease antithrombotic factors..... DVT).
4. **Late pregnancy & postpartum period** (hypercoagulability state).
5. **Disseminated cancer** (Tumor-associated procoagulant release) (migratory thrombophlebitis or Trousseau syndrome)
6. **Advanced age** also increases the risk of DVT.

Arterial and Cardiac Thrombosis

- **Causes:**
 1. **Atherosclerosis** is the major cause of arterial thrombi due to abnormal flow and endothelial damage
 2. **Myocardial infarction** with dyskinetic and endocardial damage can cause mural thrombi.

3. **Rheumatic valvular disease** resulting in mitral valve scarring and stenosis, with left atrial dilation, predisposes to atrial thrombus formation; concurrent atrial fibrillation augments the blood stasis and propensity to thrombose.
- Cardiac and aortic mural thrombi can embolize peripherally; brain, kidneys, and spleen are main targets.

Embolism

- **An embolus:** is a detached intravascular solid, liquid, or gaseous mass that is carried by the blood to a distant site, where it causes tissue dysfunction or infarction.
- **Types of emboli according to the constituents:**
 1. **Solid emboli: e.g.**
 - a. Fragments detached from a thrombus, this is called thromboembolism. (The vast majority of emboli).
 - b. Fragments of an ulcerated atherosclerotic plaque. (cholesterol emboli).
 - c. Fragments of tumor.
 - d. Fat droplets.
 - e. Bits of bone marrow
 2. **Fluid emboli: e.g.** Amniotic fluid embolus.
 3. **Gaseous emboli: e.g.** Air embolism.
 - The primary consequence of systemic embolization is ischemic necrosis (infarction) of downstream tissues resulting from partial or complete vascular occlusion; Whereas embolization in the pulmonary circulation leads to hypoxia, hypotension, and right-sided heart failure.

❖ **Clinical effects of embolism are divided according to the site where they are lodged into:**

Pulmonary thromboembolism or Systemic thromboembolism.

❖ **Pulmonary thromboembolism:**

- The incidence of pulmonary embolism (PE) is 2 to 4 per 1000 hospitalized patients.
- In more than 95% of cases, emboli originate from thrombi within deep leg veins proximal to the popliteal fossa; embolization from lower leg thrombi is uncommon.
- Fragmented thrombi from DVTs are carried through progressively larger channels and usually pass through the right side of the heart before arresting in the pulmonary vasculature. Depending on size, a PE can occlude the main pulmonary artery, impact across the bifurcation (saddle embolus), or pass into smaller arterioles.
- Multiple emboli can occur, either sequentially or as a shower of small emboli from a single large mass; in general, one PE puts a patient at risk for more.
- Rarely, emboli pass through atrial or ventricular defects into the systemic circulation (paradoxical embolism).
- The clinical and pathological features of pulmonary embolism depend on the **size of embolus:**
 1. Most PE (60% to 80%) are small and clinically silent. They eventually organize and get incorporated into the vessel wall or leave a delicate, bridging fibrous web.
 2. A large embolus that blocks a major pulmonary artery can cause sudden death.

3. Embolic obstruction of medium-sized arteries can cause pulmonary hemorrhage but usually not pulmonary infarction due to collateral bronchial artery flow; however, with left-sided cardiac failure (and diminished bronchial circulation), infarcts can result.
4. Embolism to small end-arteriolar pulmonary branches usually causes infarction. (**chest pain, dyspnea & hemoptysis**).
5. Multiple emboli occurring through time can cause pulmonary hypertension and right ventricular failure (cor pulmonale).

❖ **Systemic thromboembolism:**

- This refers to emboli in the arterial circulation.
- 80% arise from intra-cardiac mural thrombi; Two-thirds of these are associated with left ventricular infarcts 25% with dilated left atria.
- The remainder originate from aortic aneurysms, thrombi overlying ulcerated atherosclerotic plaques, fragmented valvular vegetations or paradoxical emboli (venous emboli that pass through an atrial or ventricular septal defect, including patent foramen ovale).
- 10% to 15% of systemic emboli are of unknown origin.
- Major sites for arteriolar embolization are the lower extremities (75%) and brain (10%); intestines, kidneys, spleen, and upper extremities are less frequent.
- The consequences of embolization depend on the caliber of the occluded vessel, the collateral supply, and the affected tissue's vulnerability to anoxia.
- Most arterial emboli cause tissue infarction.

❖ **Fat embolism**

- It means presence of microscopic fat globules in the circulation.
- occurs after fractures of long bones or, rarely, after burns or soft tissue trauma.
- Fat embolism occurs in 90% of severe skeletal injuries; Only less than 10% of patient show clinical features.

➤ **Pathogenesis:**

- 1- Fat microemboli cause occlusion of cerebral & pulmonary microvasculature.
- 2- Free fatty acids released from fat globules leading to local toxic injury to endothelium, platelet activation and granulocyte recruitment along with release of injurious free radical, protease and eicosanoid.

- Edema and hemorrhage (and pulmonary hyaline membranes) can be seen microscopically.

➤ **Clinical features:**

- Symptoms start within 1 to 3 days after injury as the sudden onset of pulmonary insufficiency (tachypnea, dyspnea, tachycardia), neurological symptoms (irritability and restlessness that can progress to delirium or coma), anemia, thrombocytopenia, a diffuse petechial rash and is fatal in about 10% of cases.
- **Bone marrow embolus in the pulmonary circulation.**
- The embolus is composed of hematopoietic marrow and marrow fat cells (*clear spaces*) attached to a thrombus.

❖ **Amniotic Fluid Embolism**

- Develops when amniotic fluid enter the maternal pulmonary circulation; The entry occurs through tears in the placental membranes and/or uterine vein rupture.

- It is a serious (mortality rate 80%) but uncommon maternal complication of labor and the immediate postpartum period.
- **The syndrome** is characterized by sudden severe dyspnea, cyanosis, and hypotensive shock, followed by seizures and coma. If the patient survives the initial crisis, pulmonary edema develops with disseminated intravascular coagulation secondary to release of thrombogenic substances from amniotic fluid.
- **Classic histologic findings** include fetal squamous cells, mucin, lanugo hair, and fat from vernix caseosa in the maternal pulmonary microcirculation. Other findings include marked pulmonary edema, diffuse alveolar damage and systemic fibrin thrombi generated by disseminated intravascular coagulation.
- It is thought that morbidity and mortality in such cases results not from mechanical obstruction of pulmonary vessels but from biochemical activation of the coagulation system and the innate immune system caused by substances in the amniotic fluid.

❖ **Air Embolism**

- Gas bubbles within the circulation can coalesce and obstruct vascular flow and cause distal ischemic injury.
- **Causes:**
 1. **Trauma/injury:** Air may enter the venous circulation through **neck wounds and chest wall injury**.
 2. **Surgery/invasive procedures:** These include invasive surgical procedures such as **thoracocentesis**, punctures of the great veins during **obstetric or laparoscopic procedures**, into the **coronary artery during bypass surgery**, cerebral circulation by neurosurgery in the “sitting position”, or hemodialysis.
- Generally, **more than 100ml of air are required to produce a clinical effect**.
- **Decompression sickness** is a special form of air embolism caused by sudden changes in atmospheric pressure; deep-sea divers and individuals in unpressurized aircraft during rapid ascent are at risk.
- When air is breathed at high pressure causes increasing amounts of gas (particularly nitrogen) to be dissolved in blood and tissues.
- If the diver then ascends (depressurizes) too rapidly, the nitrogen expands in the tissues and bubbles out of solution in the blood to form gas emboli, which cause tissue.
- Formation of gas bubbles in skeletal muscles and joints causes painful bends. In lungs, edema, hemorrhage, and focal emphysema lead to respiratory distress, or chokes. Gas emboli may also cause focal ischemia in a number of tissues, including brain and heart.
- A more chronic form of decompression sickness is **caisson disease**; persistent gas emboli in poorly vascularized portions of the skeleton (heads of the femurs, tibia, and humeri) lead to ischemic necrosis.