Pathology

Hemodynamic disorders - 2, Thrombosis

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Thrombosis

- Objectives:
- To define thrombosis.
- To explain how this process occurs.
- To describe its histological changes and clinical significance.
Thrombosis

- It is a process by which a thrombus is formed.
- A thrombus is a solid mass of blood constituents which develops in artery or vein.
Thrombosis

Pathogenesis:

Virchow's triad:
Endothelial injury

Endothelial cell loss:
1. Ulcerated atherosclerotic arterial plaques.
2. Traumatic vascular injury
3. Vasculitis
Endothelial dysfunction:
1. Hypertension
2. Hypercholesterolemia
3. Products absorbed from cigarette smoke
4. Turbulent flow over deformed cardiac valves
5. Bacterial toxins
Alterations in Normal Blood Flow

- *Turbulence*
  Contributes to arterial and cardiac thrombosis by causing endothelial injury or dysfunction, as well as by forming countercurrents and local pockets of stasis.
- *Stasis*
  Is a major contributor to the development of venous thrombi.
Stasis and turbulence effect:

1. Disrupt laminar flow and bring platelets into contact with the endothelium.
2. Prevent dilution of activated clotting factors by fresh-flowing blood.
3. Retard the inflow of clotting factor inhibitors and permit the buildup of thrombi.
4. Promote endothelial cell activation, resulting in local thrombosis, leukocyte adhesion, etc.
Hypercoagulable States

1. **Primary (Genetic):**
   - Mutation in factor V gene
   - Mutation in prothrombin gene
   - Antithrombin III deficiency
   - Protein C deficiency
   - Protein S deficiency
   - Fibrinolysis defects
2. Secondary (Acquired) Hypercoagulable States

- A. High risk for thrombosis:
  - Prolonged bed rest or immobilization
  - Myocardial infarction,
  - Atrial fibrillation
  - Tissue damage (surgery, fracture, burns)
  - Cancer
  - Prosthetic cardiac valves
  - Disseminated intravascular coagulation
B. Lower risk for thrombosis

- Cardiomyopathy,
- Nephrotic syndrome,
- Hyperestrogenic states (pregnancy),
- Oral contraceptive use,
- Sickle cell anemia,
- Smoking.
- Obesity
- Advancing age.
**Morphology of thrombus**

- Thrombi may develop anywhere in the cardiovascular system: the cardiac chambers, valve cusps, arteries, veins, or capillaries.
- They vary in size and shape, depending on the site of origin.
• Arterial or cardiac thrombi usually begin at a site of endothelial injury (e.g., atherosclerotic plaque) or turbulence (vessel bifurcation)

• Venous thrombi characteristically occur in sites of stasis.
Thrombi (cont.)

- Thrombi may have grossly (and microscopically) apparent laminations, called lines of Zahn;
- These are produced by alternating pale layers of platelets admixed with some fibrin and darker layers containing more red cells.
When arterial thrombi arise in heart chambers or in the aortic lumen, they usually adhere to the wall of the underlying structure and are termed mural thrombi.
Fate of the Thrombus
Clinical Consequences

- Thrombi are significant because they cause obstruction of arteries and veins, and are sources of emboli, which effect depends on the site of the thrombosis.
- Venous thrombi can cause congestion and edema in vascular beds distal to an obstruction, but they are far more worrisome for their capacity to embolize to the lungs and cause death.
Conversely, although arterial thrombi can embolize and cause downstream infarctions, a thrombotic occlusion at a critical site (e.g., a coronary artery) can have more serious clinical consequences.
Summary

• Thrombus is a solid mass of blood constituents.
• Pathogenesis of thrombus include endothelial injury, abnormal blood flow and hypercoagulability.
• Venous thrombi are worrisome for embolization.
• Arterial thrombi may cause thrombotic occlusion at a critical site (e.g. coronary arteries).
THANK YOU SO MUCH FOR YOUR ATTENDANCE AND ATTENTION