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thrombosis

- the primary causes of thrombosis
  - endothelial injury.
    - lead to platelet activation
      - severe endothelial injury may trigger thrombosis by exposing VWF and tissue factor
    - cardiac and arterial clots are typically rich in platelets
    - caused by
      - physical injury.
      - infectious agents.
      - inflammatory mediators.
      - metabolic abnormalities, such as hypercholesterolemia or homocystinemia,
      - toxins absorbed from cigarette smoking.
  - stasis or turbulent blood flow.
    - is a major factor in the development of venous thrombus.
    - effects:
      - Stasis allows platelets and leukocytes to come into contact with the endothelium when the flow is sluggish.
      - Stasis also slows the washout of activated clotting factors and impedes the inflow of clotting factor inhibitors.
    - turbulant flow is caused by:
      - change in vessel diameter
      - increase in velocity
      - low blood viscosity
    - contributes to thrombosis in a number of clinical setting
      - Ulcerated atherosclerotic plaques.
      - aortic aneurysms
      - Hyperviscosity syndromes (such as polycythemia vera)increase resistance to flow and cause small vessel stasis.
      - sickle cell anemia: The deformed red cells in cause vascular occlusions
  - hypercoagulability of the blood
    - typically caused by alterations in coagulation factors
    - an importnat risk factor for venous thrombosis
    - the alteration in the coagulation pathways can be divided into:
      - Primary (genetic):
        - mutations in the factor V .
        - elevated levels of homocysteine
          - due to thioester linkages formed between homocysteine metabolites and a variety of proteins, including fibrinogen
      - secondary (acquired) disorders
        - oral contraceptive.
        - Pregnancy**.
          - hyperestrogenic state leads to hepaic synthesis of coagulation factors and reduced synthesis of anti-thrombin III
        - Cancers
          - release of procoagulant tumor products (e.g., mucin from adenocarcinoma)
- thrombophilic states
  - Heparin-induced thrombocytopenia (HIT) syndrome
    - occurs in patients treated with unfractionated heparin.
    - It is marked by the development of autoantibodies that bind complexes of heparin and platelet membrane protein (platelet factor-4).
    - Its resulting in platelet activation, aggregation, and consumption (hence thrombocytopenia), as well as causing endothelial cell injury.
  - Anti-phospholipid antibody syndrome:
    - Acquired antibodies against phospholipid - protein complexes.
    - Suspected antibody targets include β2-glycoprotein I, a plasma protein that associates with the surfaces of endothelial cells, trophoblasts, and prothrombin
    - symptoms
      - recurrent thromboses.
      - repeated miscarriages.
      - cardiac valve vegetations.

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- **thrombosis**
  - **the clinical presentations can include:**
    - **pulmonary embolism (following lower extremity venous thrombosis).**
    - **pulmonary hypertension (from recurrent subclinical pulmonary emboli)**
    - **Stroke.**
    - **bowel infarction.**
    - **renovascular hypertension.**
  - **Fate of the Thrombus**
    - **1. Propagation: The thrombus enlargment.**
    - **2. Embolization: transported in the vasculature.**
    - **3. Dissolution: shrinkage and complete dissolution.**
    - **4. Organization**
      - **ingrowth of endothelial cells, smooth muscle cells, and fibroblasts.**
    - **5. Recanalization:**
      - **capillary channels are formed create canal along the length of the thrombus, thereby reestablishing the continuity of the original lumen.**
  - **common examples**
    - **Venous Thrombosis (Phlebothrombosis)**
      - **clinical features**
        - **Pain.**
        - **local congestion and swelling from impaired venous outflow.**
        - **varicose ulcers.**
        - **rarely embolize.**
      - **they frequently propagate some distance toward the heart, forming a long cast within the vessel lumen that is prone to give rise to emboli.**
      - **they tend to contain more red cells.**
    - **Mural thrombi:**
      - **Thrombi occurring in heart chambers or in the aortic lumen**
      - **Both cardiac and aortic mural thrombi are prone to embolization. .**
        - **The brain, kidneys, and spleen are particularly likely targets because of their rich blood supply.**
    - **Vegetations:**
      - **Thrombi on heart valves , divided into :**
        - **infective endocarditis: Infective thrombotic masses**
        - **nonbacterial thrombotic endocarditis: Sterile vegetations.**
        - **LibmanSacks endocarditis:**
          - **Sterile, occur in the systemic lupus erythematosus**
    - **Arterial thrombi**
      - **are frequently occlusive. They are typically rich in platelets**
      - **is mostly caused by Atherosclerosis because it is associated with the loss of endothelial integrity and with abnormal blood flow.**
    - **Disseminated Intravascular Coagulation (DIC)**
      - **is widespread thrombosis within the microcirculation that may be of sudden or insidious onset.**
      - **= widespread microvascular thrombosis + fibrinolytic mechanisms activation.**
      - **consumes platelets and coagulation proteins (hence the synonym consumptive coagulopathy).**