Hemodynamic Disorders, Thrombosis, and Shock

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Edema

- The accumulation of abnormal amounts of fluid in intercellular spaces of body cavities.
- Inflammation and release of mediators (*exudate*: contains inflammatory cells).
- Alterations in hemodynamic forces (*transudate*: consists of fluid without cells).
Lobar Pneumonia with Inflammatory Response
Microscopic: Pneumonia with Inflammatory Response
Edema
Hemodynamic Mechanisms

- Increased hydrostatic pressure
- Loss of plasma colloid
- Increased vascular permeability
- Impaired lymphatic drainage
- Salt and water retention
Increased Hydrostatic Pressure

• Generalized
  – Cardiac failure
  – Renal failure

• Localized
  – Venous stasis
  – Ascites
Dilated Heart: Congestive Failure
Left Ventricular Hypertrophy
Alveolar Spaces and Bronchiole
Pulmonary Edema
Fluid Droplets in Trachea/Bronchi
Pitting Edema
Surface of Cirrhotic Liver
Abdominal Ascites
Normal Brain
Edematous Brain
Hyperemia and Congestion

• An increased volume of blood in an affected tissue or part
• Hyperemia: active process
• Congestion: passive process
Hyperemic Lungs
Pulmonary Congestion
“Heart Failure Cells” in Alveoli
Hemosiderin
Hyperemic Enlarged Spleen
Hemorrhage

• Rupture of blood vessels with loss of blood
• Acute
• Chronic (compensatory mechanisms)
Berry Aneurysms in Circle of Willis
Subarachnoid Hemorrhage
Intracerebral Hemorrhage
Pericardial Hemorrhage
Ruptured Spleen
Hemostasis

Normal hemostatic mechanisms that maintain the fluidity of the blood and yet allow the rapid formation of a solid plug to close a defect in a vascular channel.
Thrombosis

A pathologic process that denotes the formation of a clotted mass of blood within a non-interrupted vascular system.
Hemostasis and Thrombosis: Dependent on Three Factors

- Vascular endothelium
- Platelets
- Coagulation system
Endothelial Cells: Antithrombotic Properties

- Antiplatelet effects
- Anticoagulant properties
- Fibrinolytic properties
Endothelial Cells: Prothrombotic Properties

• Adhesion of platelets
• Synthesis of von Willebrand’s factor (VWF)
• Synthesis of tissue factor (TF)
Platelets

- Recognize sites of endothelial injury
- Adhere to subendothelial collagen and become activated
- Release chemicals stored within granules (ADP, Thromboxane A2)
- These molecules recruit additional platelets (primary hemostasis)
Coagulation Cascade

• Release of tissue factor from injured endothelial cells initiates coagulation cascade.
• Ultimately forms a more stable plug (secondary hemostasis).
Coagulation cascade

1. Tissue damage
   - TF (Tissue Factor)

2. Extrinsic Pathway
   - VIIa
   - VII

3. Intrinsic Pathway
   - XII → XIIa
   - XI → XIa
   - X

4. Fibrinogen to Fibrin
   - II (Prothrombin)
   - Ila (Thrombin)
   - Xa
   - Va
   - Va

Additives:
- VIIIa
- PL
- Ca++

Stages:
- XII → XIIa
- XI → XIa
- IX → IXa
- X → Xa
- II (Prothrombin)
- Ila (Thrombin)
- Fibrinogen → Fibrin
Anticoagulants

- Antithrombins inhibit serine protease factors
- Proteins C and S inactivate factors Va and VIIIa
- Plasminogen-plasmin system results in fibrinolysis
Thrombus

- A mass of blood constituents, platelets, red cells, fibrin, and white cells formed in the circulating blood stream
Thrombosis
Predisposing Factors

- Endothelial injury
- Alterations to normal blood flow
- Hypercoagulability states
- (Stasis of blood flow)
Thrombi: Arterial

- Often attached to an atherosclerotic lesion
- Most occur in coronary, cerebral, and femoral arteries.
- Occlusive and alter blood flow
Mural Thrombus
Coronary Artery Occlusion
Abdominal Aortic Aneurysm Thrombus
Thrombus in Vessel: Lines of Zahn
Thrombus Potential Sequelae

• Propagate
• Embolize
• Dissolve
• Recanalize
Deep Vein Thrombosis (DVT)
Thrombi: Venous

• Occlusive cast of the vessel
• 90% in veins of lower extremities
  – Femoral
  – Popliteal
  – Iliac
Plaque with Recent Thrombus
Early Organizing Thrombus
Embolus

• A detached solid, liquid, or gaseous mass that is carried by the blood stream to a site distal from its point of origin.
Emboli

- 90% originate from thrombi
- Either arterial or venous
  - Arterial: 85% arise from heart
  - Venous: Majority arise from leg veins
- Occlude vessels resulting in varying pathology
R Ventricle Embolus from Leg Vein
Pulmonary Embolus (Saddle)
Distal Pulmonary Embolus
Pulmonary Infarction
Coagulative Necrosis in Pulm Inf
Atheromatous Embolus
Tumor Embolus
Fat Embolus to Lung
Fat Embolus to Kidney
Fat Embolus to Brain: Macro
Fat Embolus to Brain: Micro
Infarct

- An area of ischemic necrosis within a tissue or an organ that is produced by occlusion of either its arterial supply or its venous drainage
Infarction

Etiologies

- Occlusive thrombi or emboli (99%)
- Compromised venous drainage
- Decreased blood flow
Infarction
Factors Influencing Development

• Rapidity of vascular inadequacy
• Availability of collateral flow
• Duration of occlusion
• Susceptibility of tissue to anoxia
Infarction

• Pale (anemic)
  – Arterial occlusion
  – Solid tissues

• Red (hemorrhagic)
  – Venous occlusion
  – Loose tissues
  – Dual or extensive collateral blood supply
Kidney: Pale Infarct
Pale Infarct (Wedge) of Spleen
Pulmonary Infarction
Pulmonary Infarction
Pulmonary Infarction
Small Intestine Infarction
Kidney Infarct, Old
Temporal-Frontal Infarct, Old
Shock

• Widespread hypoxia of tissues caused by the ineffective circulation of blood
• Hypovolemia
• Impaired cardiac function
• Trauma
• Severe infections
• Generalized hypersensitivity reactions
Morphologic Features of Shock

- Brain: ischemic encephalopathy
- Heart: subendocardial hemorrhages and necrosis
- Kidneys: acute tubular necrosis or diffuse cortical necrosis
- Gastrointestinal tract: patchy hemorrhages and necrosis
- Liver: fatty change or central hemorrhagic necrosis
Kidney: Acute Tubular Necrosis
Kidney: Pale Cortex in Shock
Ischemic Necrosis of Liver
Liver: Central Hemorrhagic Necrosis
Septic Shock

• High mortality rate (25-50%; critically ill)
• Systemic release of endotoxins
  – Gram negative bacterial cell walls
  – Lipopolysaccharides (LPS)
• Hypotension, dec myocardial contractility, endothelial injury, disseminated intravascular coagulation (DIC), fibrinolysis (plasmin)
• Bleeding due to consumption of coagulation factors and activation of fibrinolysis