6. Fluid and hemodynamic disorders

Background

Total Body Water [Fig. 6-1]
- Human body is 60% fluid (water) by weight
  - Total Body Water (TBW) = 42 liters (70kg M)
- Body has two major compartments (inside cell or outside)
  - 2/3 of TBW is located inside cells
    - intracellular fluid compartment [28 l]
  - 1/3 of TBW is located outside cells
    - extra-cellular fluid compartment [14 l]
      - 1/4 ECF is located inside blood vessels (intra-vascular) [3.5 l]
      - 3/4 ECF is located in extra-vascular (interstitial) space [9.5 l]

Movement of fluid
- Distribution of water between ICF and ECF compartments is determined by distribution of electrolytes
- Distribution of water within the ECF between the intra-vascular and interstitial space is determined by proteins
- Fluid constantly moves between compartments
  - fluid moves out of capillaries due to hydrostatic pressure in the capillary and osmotic pressure in ECF
  - fluid moves into capillaries due to oncotic pressure in the vessel and hydrostatic pressure in the ECF
- Lymphatics remove excess fluid not returned to vessels

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Edema
- Edema is the accumulation of excess fluid in ECF space
  - edema may be localized or systemic
  - edema fluid may be a Transudate or an Exudate
- Exudate
  - an exudate has a high protein content and lots of white blood cells
  - an exudate forms due to inflammation
- Transudate
  - a transudate has a low protein content and few white blood cells
  - a transudate forms due to imbalance of forces across vessel walls
- The cause of edema is often multifactorial
- Terminology
  - anasarca is severe generalized edema
  - ascites is excess fluid in abdominal cavity
  - hydrothorax is excess fluid in pleural cavity
  - hydrocardia is excess fluid in pericardial cavity
- Edema may have serious consequences
  - cerebral edema may result in herniation of the brain and death
  - pulmonary edema may result in impaired air exchange and death
Fluid and hemodynamic disorders

Edema

pathogenesis [Fig. 6-2]

- Presence of edema is an important sign of disease
  - edema may be first indication of problem with an organ
- Cardiac disease (congestive heart failure) [Fig. 6-3]
  - edema fluid may occur in CHF, the cause is often multifactorial
    - increased pressure in veins because heart not pumping effectively
    - anoxia in venous system results in increased permeability
    - Na retention results in water retention
- Renal disease
  - edema fluid may occur in renal disease
    - plasma proteins lost in urine due to renal diseases
    - relative increase in osmotic (protein) pressure of ECF

Hyperemia

- Hyperemia is an increased volume of blood in a tissue
  - active hyperemia occurs due to dilation of arterioles & capillaries
    - exercise, inflammation
  - passive hyperemia (congestion) occurs due to increased venous pressure that occurs with impaired outflow of blood from the area
- Cyanosis is a bluish discoloration of the lips and skin indicating a lack of oxygen

Hemorrhage

- Hemorrhage is the loss of blood [Fig. 6-5]
- In external hemorrhage blood exits the body
- In internal hemorrhage blood remains in the body
  - blood may enter a body cavity
    - hemothorax is blood in thoracic cavity
    - hemoperitoneum is blood in peritoneal cavity
    - hemopericardium is blood in pericardial cavity
  - a hematoma is coagulated blood in tissue (bruise)
    - petechiae is a pinpoint hemorrhage due to rupture of a capillary
    - purpura is a bruise >3mm in size
    - echymoses are larger hemorrhagic spots on skin and mucosa
- Terminology [Fig. 6-5]
  - hemoptysis refers to coughing up blood from lungs
  - hematemesis refers to vomiting blood from upper GI tract
  - hematochezia refers to passing bright red blood per rectum
  - melena refers to passage of dark (black) stools (UGI bleed)
  - hematuria refers to passage of blood in urine
  - metrorrhagia refers to excessive menses
- Symptoms depend on amount, site, duration of blood loss
  - rapid loss of less than 20 % of blood volume is compensatable
  - massive loss (>1500 ml) results in hypovolemia and shock
  - chronic loss results in anemia
  - hemorrhage into brain may result in herniation of the brain
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Thrombosis (clot formation)

- Blood clots in order to prevent loss of blood [Fig. 6-6]
  - if endothelium is damaged then a “plug” is made to fill hole
    - vessel constriction, platelet plug, reinforced by fibrin
- Clotting requires platelets, endothelium, plasma proteins
  - normally there is a balance of clot formation and clot lysis
- Thrombus is formation of clot within vessel during life
  - formation of a thrombus may cause complications [Fig. 6-7]
  - certain factors predispose to thrombus formation (Virchow’s triad)
    - stasis of blood (CHF, dehydration)
    - hypercoaguable states (inherited, malignancy)
    - endothelial injury (thrombogenic surface revealed)
- A thrombus has 4 major fates [Fig. 6-9]
  - lysis of the thrombus removal of thrombus by fibrinolysis
  - organization and recanalization replacement of the thrombus by granulation tissue and creation of new channels through thrombus
  - propagation is complete occlusion of a vessel with extension of the thrombus proximal in vein
  - embolus formation is detachment of a thrombus and impaction lodge distally
- Types of thrombus
  - venous
    - deep vein thrombosis
  - arterial

Emboli

- An embolus is a thrombus or other movable intravascular mass that may cause obstruction of a vessel
- Types of emboli
  - thromboemboli (99%)
  - fat emboli
  - gas emboli
  - solid emboli
  - liquid emboli
- An embolus causes ischemia to organs distal to the site of embolus impaction
- Pulmonary emboli [Fig. 6-10]
  - thrombus may form in deep veins of legs
  - the thrombus may dislodge and enter the inferior vena cava
  - thromboembolus passes through heart and impacts in vessels in lung
    - saddle embolus
    - pulmonary infarcts
- Arterial thromboemboli [Fig. 6-10]
  - cerebral infarcts may result from arterial thrombi in carotids dislodging and impacting in vessel feeding area of the brain
  - infarct of distal extremity may result from arterial thrombus in the aorta dislodging and impacting in vessel supplying distal extremity
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Infarction

• Infarction is irreversible ischemic necrosis of cells usually due to occlusion of arterial supply

• Factors influencing outcome of vessel occlusion include
  – pattern of vascular supply (presence of dual blood supply)
  – rate of development of occlusion
  – vulnerability of tissue to hypoxia
  – oxygen content of blood

• End result of an infarct depends on tissue’s ability to repair
  – heart heals an infarct by fibrosis
  – liver is able to replace damaged tissue over time
  – brain is unable to regenerate or create a scar and forms

Shock

• Shock is inadequate perfusion (blood supply) to tissue

• Normal tissue perfusion requires a functioning pump (heart), intact pipes (vessels) and adequate fluid (blood)

• Causes of shock
  – S septic shock
  – H hypovolemic shock
  – O obstructive shock
  – C cardiogenic shock
  – K anaphylactic shock
  – S spinal/neurogenic shock

pathophysiology [Fig. 6-14]

• Cardiogenic shock
  – results from heart not pumping adequately
    • myocardial infarction
    • arrhythmia

• Hypovolemic shock
  – results from loss of blood volume
    • hemorrhage
    • water loss (burn)

• Hypotonic shock
  – results from pooling of blood in the periphery
    • anaphylaxis
    • sepsis

clinical

• Compensated (nonprogressive) phase
  – compensatory mechanisms able to maintain perfusion

• Decompensated (progressive) phase
  – compensatory mechanisms unable to maintain perfusion
    – hypotension, oliguria, acidosis, short of breath

• Irreversible shock
  – circulatory collapse, hypoperfusion of vital organs, loss of vital functions