Hemodynamic Disorders

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LEC 1

Hemodynamic Disorders, Thromboembolic Disease, and Shock

- Edema
- Hyperemia and congestion
- Hemorrhage
- Hemostasis and thrombosis
- Embolism
- Infarction
- Shock

EDEMA

- Approximately 60% of body weight is water.
- Two thirds of the body's water is intracellular, and the remainder is in extracellular compartments, mostly as interstitial fluid
- only about 5% of total body water is **INTRA-vascular** (blood plasma)
- **Edema: is abnormal increase in interstitial fluid within tissues**
- EDEMA is SHIFT to the INTERSTITIAL SPACE FROM EITHER DIRECTION
  CELL→ECF← VASC
- Collections of fluid in the different body cavities are variously designated as
  Hydrothorax ……in pleura
  Hydropericardium …….in pericardium
  Hydroperitoneum (ascites)….. in peritoneum
- **Anasarca** is a severe and generalized edema with widespread subcutaneous tissue swelling, occur in cirrhosis, heart and renal failure ….

Pathophysiology of edema:

- The movement of water and low molecular weight solutes such as salts between the intravascular and interstitial spaces is controlled primarily by the opposing effect of
  1- Vascular hydrostatic pressure
  2- Plasma colloid osmotic pressure.
- Normally the outflow of fluid from the arteriolar end of the microcirculation into the interstitium is nearly balanced by inflow at the venular end; a small residual amount of fluid may be left in the interstitium and is drained by the lymphatic vessels, which return to the bloodstream via the thoracic duct.

Types of edema fluid

1- **Transudates** developed in hydrodynamic disturbances with low protein content and specific gravity of **1.012** and less.
2- **Exudate** is inflammatory edema which is protein rich fluid due to increased vascular permeability with specific gravity of **1.020** and more.

Pathophysiological classification of edema:

A- Inflammatory Edema

B- Non-Inflammatory Edema

1. **Increased Hydrostatic Pressure**
2. **Reduced Plasma Osmotic Pressure**
3. **Lymphatic Obstruction**
4. **Sodium Retention**

Increased hydrostatic pressure:

1- **Localized increase** in venous pressure e.g (**deep venous thrombosis DVT**) with edema of the affected limb.

2- **Generalized increase** in venous pressure with systemic edema occur in **congestive heart failure** in which reduced cardiac output causes reduced renal perfusion & trigger of rennin angiotensin aldosterone axis causing sodium & water retention by kidney in order to increase intravascular volume & improve cardiac output & renal perfusion .

- This extra fluid load only increased venous pressure & edema
- .Unless cardiac output restored or renal fluid retention reduced (e.g salt restriction, diuretics &/or aldosterone antagonists), repeated cycles of renal fluid retention & worsening edema result.

Reduced plasma osmotic pressure: (reduced plasma proteins)

Result from:

- Increased loss of albumin as in **nephrotic syndrome**.
- Reduced protein synthesis as in **cirrhosis, malnutrition**.

Lymphatic obstruction: Usually localized:

Causes:

(1) Inflammatory obstruction e.g filariasis which causes lymphatic obstruction& lymph node fibrosis in inguinal region leading to edema of genitalia& lower limb (elephantiasis).

(2) Cancer of breast treated by surgery or irradiation with resection of lymphatic drainage of upper limb & scarring; there is edema of the arm.

(3) In CA breast, infiltration & obstruction of superficial lymphatics will cause edema of breast skin (peau-de-orange) due depression of the skin at site of hair follicles.

Morphology of edema:

Grossly: Edema result in increased in size of affected organ microscopically, it is appreciated as clearing and separation of the extracellular matrix and subtle cell swelling.

Any organ or tissue can be involved, but edema is most commonly seen in subcutaneous tissues, the lungs, and the brain.

Subcutaneous edema can be diffuse or more in regions with high hydrostatic pressures. In most cases the distribution is influenced by gravity and is termed **dependent edema** (e.g., the legs when standing, the sacrum when recumbent).

Finger pressure over edematous subcutaneous tissue displaces the interstitial fluid and leaves a depression, a sign called **pitting edema.**
Edema as a result of renal dysfunction can affect all parts of the body. It often initially manifests in tissues with loose connective tissue matrix, such as the eyelids; periorbital edema is thus a characteristic finding in severe renal disease.
With **pulmonary edema**, the lung is 2-3 times their normal weight, cut section frothy, blood tinged fluid represent mixture of air, edematous fluid & extravasated RBCs.

Brain edema can be **localized**: due to abscess, neoplasm &trauma.
Or **generalized**: due to encephalitis, hypertension crises & trauma
Grossly: Swollen with narrowed sulci & distended gyri.

Clinical correlation of edema:
a. Subcutaneous edema in cardiac failure & renal failure can impair wound healing or clearance of infection.
b. Pulmonary edema can cause death by interfering with normal ventilatory function by: Fluid collects in alveolar space impair oxygen diffusion and represent a favorable environment for bacterial infection.
c. Brain edema if severe may cause death due to brain herniated through foramen magnum which causes compression of vital centers & also cause compression of vascular supply of brain.

Hyperemia & Congestion

Hyperemia and congestion both result from **locally increased blood volumes.**

Hyperemia

- It is an active process in which arteriolar dilation leads to increased blood flow.
- Affected tissues turn red (erythema) because of the engorgement of vessels with oxygenated blood.
- It is divided into:

1- Local 2- General 3- Physiological 4- Pathological

Localized hyperemia: could be
A. physiological: e.g exercise, after meal.
B. Pathological: e.g site of inflammation.

Generalized hyperemia: could be
A. Physiological: e.g hot weather.
B. Pathological: e.g: Fever, Hyperthyroidism.

Congestion

It is a passive process resulting from reduced outflow of blood from a tissue.
It can be **systemic**, as in cardiac failure, or **local**, as in isolated venous obstruction.
Congested tissues take on a dusky reddish-blue color (cyanosis) due to red cell stasis and the accumulation of deoxygenated hemoglobin.

Liver:

Chronic venous congestion of liver resulting from **right sided heart failure**
Grossly: mottled appearance similar to the nut meg hence the name **Nut meg liver**.
Microscopically:
Congestion of the central venule, necrosis of the surrounding hepatocytes because of pressure &hypoxia.
The peripheral liver cells are normal or show fatty change.

Lung:

Pulmonary venous congestion: e.g left ventricular failure where there is accumulation of blood in the surrounding dilated capillaries due to congestion capillaries, RBCs will escape to the alveolar space & engulfed by macrophages resulting in hemosiderin laden macrophages called (heart failure cells) which cause cough with blood stained sputum.

HEMORRHAGE

It is extravasation of blood into the extravascular space due to rupture of blood vessel

Causes of Hemorrhage:

1- Trauma: e.g penetrating wounds to the heart & large vessels----- rapid blood loss.

2- Abnormalities of blood vessel wall like:
a- Inflammatory lesions may lead to weakening of arteries &may cause aneurysmal dilatation &rupture.
b- Neoplastic invasion e.g. carcinoma of tongue with invasion of lingual arteries.
c- Other vascular diseases e.g atheroma, aneurysm.

3- High pressure within blood vessel e.g systemic HT leading to hemorrhage at sites of arterial weakness or increase venous pressure in varicose veins e.g. legs, esophageal hemorrhage.

Patterns of hemorrhage with its clinical implications:

- Hemorrhage may be external or contained within a tissue; any accumulation is called a **hematoma**. Hematomas may be relatively insignificant or so massive that cause death.
- Minute 1- to 2-mm hemorrhages into skin, mucous membranes, or serosal surfaces are called **petechiae.** These are most commonly associated with locally increased intravascular pressure, low platelet counts (thrombocytopenia), or defective platelet function (as in uremia).
- Larger (≥3 mm) hemorrhages are called **purpura**. These may be associated with many of the same disorders that cause petechiae or can be secondary to trauma, vascular inflammation (vasculitis).

- Larger (>1 to 2 cm) subcutaneous hematomas (i.e., bruises) are called **ecchymoses**. The red cells in these lesions are degraded and phagocytized by macrophages; the hemoglobin (red-blue color) is then enzymatically converted into bilirubin (blue-green color) and eventually into hemosiderin (gold-brown color), accounting for the characteristic color changes in a bruise.
- Depending on the location, a large accumulation of blood in a body cavity is denoted as a **hemothorax, hemopericardium, hemoperitoneum, or hemarthrosis** (in joints).

Effects of acute hemorrhage:

Depend on volume &rate of blood loss: as following

1- The effect is of impact in healthy adults; if less than 20% of blood volume is lost, while sudden loss of 33% of blood volume leads to death.

2- Gradual loss (within 24 hour) of more than 50% blood volume is not necessarily fatal but it is serious.

In human 60-70% of blood volume contained in veins &venules, so constriction of these vessels will increase venous return to the heart.
Arteriolar constriction is selective i.e there is decrease blood flow to skin, salivary glands, liver, spleen & kidneys while blood is preserved to the brain, heart, skeletal muscles &diaphragm.