Circulatory disorders

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**Edema**

Approximately 60% of lean body weight is water; **two thirds** of this water is **intracellular**, and the remainder is found in the **extracellular space** (mostly as **interstitial fluid**) and only about **5%** of total body water is in **blood plasma**.

**Edema:** Is pathological accumulation of excess fluid in the interstitial tissue spaces.

**Mechanism of edema formation and causes:**

There are four primary forces that determine fluid movement across the capillary membrane. These four primary forces are known as Starling forces & they are:

**a. The capillary hydrostatic pressure.** This pressure tends to force fluid outward from the intravascular space through the capillary membrane to the interstitium.

**b. The interstial fluid hydrostatic pressure**. This pressure tends to force fluid from the interstitial space to the intravascular space.

**c. The plasma colloid osmotic (oncotic) pressure**. This pressure tends to cause osmosis of fluid inward through the capillary membrane from the interstitium. The plasma oncotic pressure is caused by the presence of plasma proteins.

**d. The interstial colloid osmotic (oncotic) pressure**. This pressure tends to cause osmosis of fluid outward through the capillary membrane to the interstitium.

The mean blood hydrostatic pressure at the arteriolar end of capillaries is about 30mmHg while the colloid osmotic pressure of plasma (also called oncotic pressure) is 20-25 mmHg. The net difference of the two forces causes fluid to escape from within the vessels (intravascular compartment) into the tissue spaces (interstitial compartment).

On the other hand the mean blood hydrostatic pressure at the venous end of the capillaries is lower than that of oncotic pressure so the higher oncotic pressure returns the fluid back to the vessels. In other word fluid return to the intravascular compartment at the venular end of the microcirculation mainly because of osmotic (oncotic) pressure of the blood. Small quantities of tissue fluids which contain substances of large molecules drain along the lymphatic vessels, which ultimately reaches the venous circulation. **From this brief outline of fluid movement in the body we can conclude the pathogenesis of edema:**

1. increase hydrostatic pressure: which could be
2. **generalized increase in** **hydrostatic pressure which seen in:**
3. **Cardiac**: right ventricular failure or combined right and left ventricular failure: because of insufficient pumping power of the heart there will be hypoperfusion to the vital organ and increase in venous blood pressure.
4. **Renal**: decrease renal blood flow ( for any cauase) which stimulate juxta glomerular apparatus to **activate the rennin-angiotensin-aldosterone system** that result in reabsorption of sodium (Na) and water by the renal tubules.
5. **localized increase in hydroststic pressure which seen in** : left ventricular failure (pulmonary edema), thrombosis of major veins, pressure of gravid uterus, pressure of tumor on the surrounding vessels or lymphatic tumor metastasis and incompetent venous valve (varicose vein).
6. decrease in colloid oncotic pressure of plasma (protein loss diseases) : seen in:

a-nephrotic syndrome (protein loss in urine)

b- liver disease (decrease protein synthesis)

1. malnutrition (decrease protein intake)
2. protein loss gastroenteropathy
3. **Lymphatic obstruction**: could be occurring due to tumor, post-surgical, post radiation, parasitic infection (filariasis) that causes lymphatic obstruction.
4. **increased capillary permeability** as in inflammation, sever allergy and anaphylactic shock

**Types (classification) of edema:**

**1.according to pathophysiological mechanism (composition of the accumulated fluid):**

1. **transudate (low protein content)**
2. **exudate (high protein content)**

**2.according to the location:**

1. **generalized**
2. **localize**

**3.according to clinical finding:**

**a. pitting edema**

**b. non pitting edema**

**According to the composition of the accumulated fluid edema divided to:**

**Transudate**:

* Accumulation of thin serous protein poor fluid.
* It is usually related to increase in hydrostatic pressure (congestion) within intravascular compartment.
* It has specific gravity below 1.012 with protein concentration less than 2.5 gm/dl.
* This type of edema can be encountered in heart failure and nephrotic syndrome.

**Exudate**: this differs from transudate in:

* Protein rich: > than 2.5 gm/dl
* Encountered with inflammation
* Resulting from increased vascular permeability that lead to escape of intravascular proteins especially albumin.
* it has specific gravity above 1.012
* seen in inflammation and allergic reaction

**According to the location:**

* **Generalized:** edema all over the body as in heart failure, renal failure
* **Localized:** edema occurs in limited area due to impaired venous return

1. Acute left ventricular failure (pulmonary edema)
2. Thrombosis of major veins
3. Valve incompetence
4. Tumor pressure on the adjacent vessels

**According to clinical finding:**

* Pitting edema: fluid can easily mover when pressing the affected par (usually occurs in transudate including the causes of generalized and localized edema)
* Non pitting edema: fluid cannot easily removed when pressing the affected part (usually occurs in exudate including inflammation, allergic reaction and lymphatic obstruction)

The most important clinical conditions of edema

**pulmonary edema**: due to left ventricular failure associated with sever dyspnea and anoxia it interfere with normal ventilation.

**Brain edema**: it is serious and rapidly fatal. It leads to increase intracranial pressure . cerebral thromboembolism is one of the major cause

**Laryngeal edema**: serious may cause suffocation.

**Anasarca**: is a severe and generalized edema with profound subcutaneous tissue swelling.

**Hydrothorax** (pleural effusion): accumulation of fluid in the pleural cavity.

**Hydropericardium** (pericardial effusion) fluid accumulated in the pericardium

**hydroperitoneum** (ascites) fluid accumulated in the peritoneum.

**Hyperemia**

**Hyperemia** is an active increase blood volume in arterial and arteriolar blood vessels (arterial and arteriolar dilatation) which can occur in:

1. exercise and heat exposure
2. inflammation and release of vasoactive substances
3. sympathetic neurogenic mechanism

**Congestion**

**Congestion:** is a passive increase in blood volume in venous part of blood vessles (impaired venous drainage). Congestion could be localized venous congestion or generalized venous congestion.

**Haemorrhage**

Hemorrhage: is extravasation of blood outside the blood vessel. Causes:

1. ruptured blood vessels wall: Physical trauma – Stabbing - Stick injury - Gunshot - RTA (road traffic accident)
2. Inadequacies in blood clotting factors which can be due to:

**A**.. qualitative & quantitative defect of platelets

**B.** Missing or low amount of clotting factors E.g. Low levels of prothrombin, fibrinogen & other precursors.

**C.** Inadequate vitamin K leads to clotting factor deficiency because this vitamin is important in the synthesis of the clotting factors by the liver.

Terminology:

1. Petechial: minute 1-2 mm hemorrhages occurring in the skin, mucosal membrane, or serosal surface.
2. Purpura: slightly > 3mm hemorrhage occurring in the skin.
3. Ecchymosis (bruises): larger than 1-2cm subcutaneous hemorrhage. It is typical after trauma.
4. Hematoma: hemorrhage enclosed within a tissue or a cavity.

Effects of hemorrhage: depend on the rate and amount of blood loss:

1. If > 20% the total blood volume is rapidly lost from the body, it may lead to hypovolemic shock & death.
2. Chronic loss of blood it leads to anemia.