HEMODYNAMIC DISORDERS

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Edema

Learning Objectives

Definition

Mechanism of formation

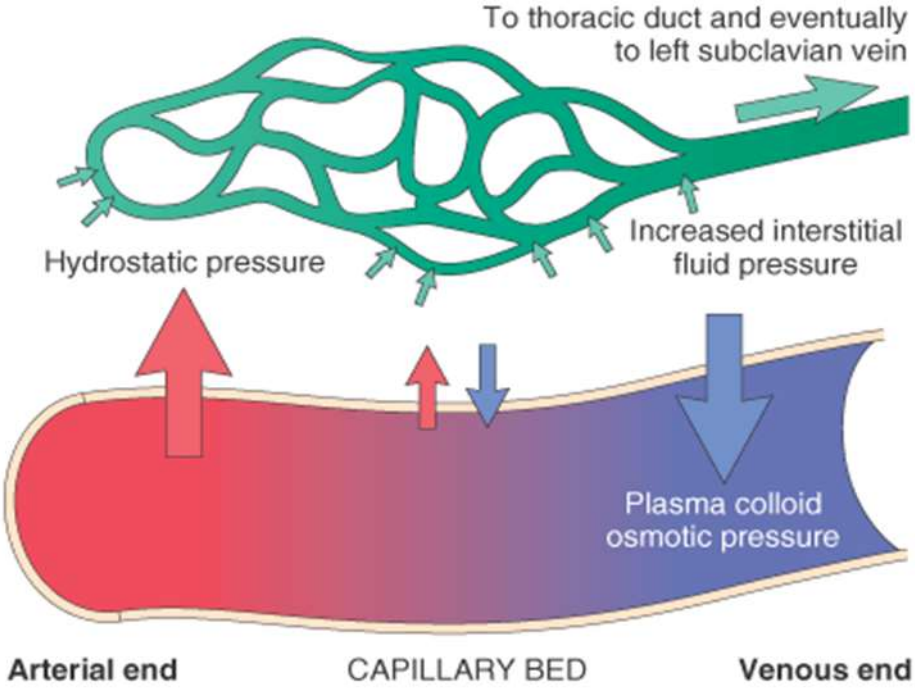
Simple point on their pathogenesis

morphology

Definition: edema is an accumulation of interstitial fluid within tissues.
Extravascular fluid can also collect in body cavities such as:
a) **Hydrothorax** – fluid accumulation in pleural cavity in a pathologic amount.
b) **Hydropericardium** – pathologic amount of fluid accumulated in the pericardial cavity.
c) **Hydroperitoneum (ascites)** – fluid accumulation in peritoneal cavity.
d) **Anasarca** – is a severe & generalized edema of the body with profound subcutaneous tissue swelling and accumulation of fluid in body cavities.

Mechanism of edema formation:
Approximately 60% of lean body weight is water, two thirds of which is intracellular. Most of the remaining water is found in extracellular compartments in the form of interstitial fluid; only 5% of the body's water is in blood plasma.
The capillary endothelium acts as a semi permeable membrane and highly permeable to water & to almost all solutes in plasma with an exception of **proteins**. Proteins in plasma and interstial fluid are especially important in controlling plasma & interstitial fluid volume.
Normally, any outflow of fluid into the interstitium from the arteriolar end of the microcirculation is nearly balanced by inflow at the venular end. Therefore, normally, there is very little fluid in

the interstitium. Tissue lymphatics drain much of the excess fluid back to the circulation by way of thoracic duct.



So causes of Edema are:

1) Increased Hydrostatic pressure
2) Decrease plasma Oncotic pressure
3) Increased vascular permeability
4) Lymphatic channels obstruction
5) Sodium retention

We will discuss each of the above sequentially.

1) Increased Hydrostatic pressure

Increases in hydrostatic pressure are mainly caused by disorders that impair venous return.

- **Local increases** in intravascular pressure caused, for example, by deep venous thrombosis in the lower extremity can cause edema restricted to the distal portion of the affected leg.
- **Generalized increases** in venous pressure, with resultant systemic edema, occur most commonly in congestive heart failure

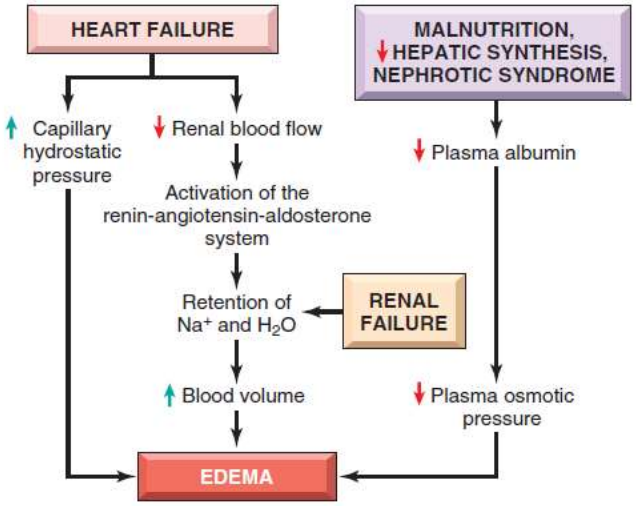
Several factors increase venous hydrostatic pressure in patients with congestive heart failure:

- The reduced cardiac output leads to systemic venous congestion and resultant increase in capillary hydrostatic pressure.

- reduction in cardiac output results in hypoperfusion of the kidneys, triggering the renin-angiotensin-aldosterone axis and inducing sodium and water retention (secondary hyperaldosteronism) leads to increase blood volume and worsening of edema .

a vicious circle of fluid retention, increased venous hydrostatic pressures, and worsening edema ensues.

Unless cardiac output is restored or renal water retention is reduced (e.g., by salt restriction or treatment with diuretics or aldosterone antagonists), this downward spiral continues.



Renal failure also cause edema because of retention of Na and water leads to increase blood volume (increase hydrostatic pressre).

2) Decrease plasma Oncotic pressure

Reduction of plasma albumin concentrations leads to decreased colloid osmotic pressure of the blood and loss of fluid from the circulation.

Under normal circumstances, albumin accounts for almost half of the total plasma protein.

Common causes of reduced plasma osmotic pressure.

- Albumin lost from the circulation.
- Or albumin synthesized in inadequate amounts.

Nephrotic syndrome is the most important cause of albumin loss from the blood. the glomerular capillaries become leaky, leading

to the loss of albumin (and other plasma proteins) in the urine and the development of generalized edema.

Reduced albumin synthesis occurs in the setting of ***severe liver disease (e.g., cirrhosis) and protein malnutrition.***
low albumin levels lead in a stepwise fashion to edema, reduced intravascular volume, renal hypoperfusion, and secondary hyperaldosteronism.
Increased salt and water retention by the kidney not only fails to correct the plasma volume deficit but also exacerbates the edema, because the primary defect—low serum protein—persists.

3) Increased Vascular permeability:

Increased vascular permeability usually occurs due to acute inflammation.
In inflammation, chemical mediators are produced. Some of these mediators cause increased vascular permeability which leads to loss of fluid & high molecular weight albumin and globulin into the interstitium.
Such edema (i.e. that caused by increased vascular permeability) is called **inflammatory edema**.
Inflammatory edema differs from non inflammatory edema by the following features:

a) Inflammatory edema (exudate)

- Due to inflammation-induced increased permeability and leakage of plasma proteins.
- Forms an exudate [protein rich]
- Specific gravity > 1.012

b) Non-inflammatory oedema (transudate)

- A type of edema occurring in hemodynamic derangement (i.e. increased plasma hydrostatic pressure & decreased plasma oncotic pressure.)
- Formed transudate [protein poor]
- Specific gravity < 1.012.

4) Lymphatic channels obstruction

Edema may result from lymphatic obstruction that compromises resorption of fluid from interstitial spaces. Impaired lymphatic drainage and consequent **lymphedema**

It usually results from a localized obstruction caused by an inflammatory or neoplastic condition.

For example,

- the parasitic infection **filariasis** can cause massive edema of the lower extremity and external genitalia (so-called "elephantiasis") by producing inguinal lymphatic and lymph node fibrosis.
- Infiltration and obstruction of superficial lymphatics by breast cancer may cause edema of the overlying skin; the characteristic finely pitted appearance of the skin of the affected breast is called **peau d'orange** (orange peel).
- Lymphedema also may occur as a complication of **therapy.** in women with breast cancer who undergo axillary lymph node **resection** and/or **irradiation**, both of which can disrupt and obstruct lymphatic drainage, resulting in severe **lymphedema of the arm.**

5) Sodium and water retention:

Excessive retention of salt (and its obligate associated water) can lead to edema by **increasing hydrostatic pressure** (because of expansion of the intravascular volume) and **reducing plasma osmotic pressure.** Excessive salt and water retention are seen in a wide variety of diseases that compromise renal function, including poststreptococcal glomerulonephritis and acute renal failure

Causes of sodium and water retention:

1- excessive salt intake with renal insufficiency.
2- increased tubular reabsorption of sodium
3-Renal hypo perfusion
4-Increased renin- angiotensin- aldosteron secretion.

Morphology of edema

Gross examination:

clearing and separation of the extracellular matrix (ECM) elements. edema most commonly encountered in subcutaneous tissues, lungs, and brain.

Subcutaneous edema can be diffuse but usually accumulates preferentially in parts of the body where hydrostatic pressures are highest. Thus, edema typically is most pronounced in the legs with **dependent edema**. Finger pressure over edematous subcutaneous tissue displaces the interstitial fluid, leaving a

finger-shaped depression; this appearance is called **pitting edema.**

Edema resulting from renal dysfunction or nephrotic syndrome often manifests first in **loose connective tissues (e.g., the eyelids, causing periorbital edema).**

Pulmonary edema, the lungs often are two to three times their normal weight, and sectioning shows frothy, sometimes blood-tinged fluid consisting of a mixture of air, edema fluid, and extravasated red cells.

Brain edema can be localized (e.g., because of abscess or tumor) or generalized, depending on the nature and extent of the pathologic process or injury.

With generalized edema, the sulci are narrowed as the gyri swell and become flattened against the skull.