VASCULAR DISORDERS

Asst. Prof. Dr. Ozan AHLAT
oahlat@ankara.edu.tr

Anemia

Hyperemia

Edema

Hemorrhage

Thrombosis

Shock

ANEMIA
OLIGEMIA
ISCHEMIA

Anemia

- It is a decrease in blood volume, number of erythrocytes, amount of hemoglobin (hematocrit value).
- It is not a disease in itself, but a finding (symptom) that occurs as a result of various diseases.
- In hypochromic anemia, which occurs as a result of underproduction of erythrocytes by the bone marrow or low hemoglobin content of erythrocytes, the mucous membranes are pale.
- In hypoxia cases that develop depending on the severity of anemia, degenerative findings are formed in the organs.
- In hemolytic anemia, signs of both anemia and jaundice and hemosiderosis occur in the organs.

Anemia

Regenerative	Nonregenerative
• **Hemorrhage** (trauma, neoplasia, parasitism etc.) • **Hemolysis** (immun mediated, infections, toxicity etc.)	• **Primary bone marrow disease** • **Extramarrow disease**

Anemia

- Bone marrow failure (aplastic anemia)
- Iron, copper, protein deficiency (nutritional anemia)
- Decrease in both blood volume and erythrocyte count due to blood loss (hemorrhagic anemia).
- Lysis and reduction of erythrocytes for various reasons (hemolytic anemia)
- Infectious (such as equine anemia infectious, leptospirosis, blood parasites), isoimmune (such as blood group incompatibility) and autoimmune, toxic, hereditary hemolytic anemias

Anemia

Acute

- Severe hemorrhagic anemias result in hypovolemic shock.

Chronic

- Cardiac hypertrophy, dilatation, respiratory distress, pulmonary edema, edema
- Pallor of the mucous membranes and changes in the spleen.

Anemia

Size of RBCs	• Macrocytic • Microcytic • Normocytic
Amount of hemoglobin	• Hypochromic anemia

Anemia

I. Related to erythrocyte loss;

A. Posthemorrhagic

a. **Primary** Trauma

b. **Secondary** Aortic aneurism; spleen and liver rupture after amyloidosis; lead toxicity

B. Hemolytic anemias

- Toxic-infectious-immunhemolytic-idiopathic-secondary

II. Decreased erythrocyte production

- Nutritional-Aplastic

Anemia findings

- **Mucosae** are pale
- **Lungs** are pale, edematous, foamy fluid in bronchi and trachea
- If leukocytes increase while erythrocytes decrease; sedimentation increases, blood does not clot well.
- **Heart:** Hypertrophic, dilated. Pale color and crunchy consistency after fatty degeneration of myocardium in chronic cases.

Anemia findings

- Parenchymatous organs such as **liver and kidney** become pale and brittle after parenchymatous and fatty degeneration.
- **Muscle:** pale due to loss of myoglobin
- **Edema:** It occurs in the body ventral and legs as a result of protein loss in chronic events.
- **The blood** differs according to the type of anemia.

Local anemia (Oligemia and ischemia)

Oligemia

- It is the event that less blood goes to a tissue as a result of a disorder in the veins.

Ischemia

- It is the event that no blood goes to the tissue due to vascular disorder.
- Tissue necrosis (infarction)

Local anemia

Compression anemia

Obturation anemia

Spastic anemia

Reflex anemia (collateral anemia)

Local anemia findings

- **Anemic** organs are **pale** and cool.
- **Degenerative-necrotic changes** and **dysfunctions** develop in organs.
- While **connective tissue resists anemia**, parenchymatous organs, especially the brain, are quickly affected.
- **In compensating anemia;**
  - Whether there is a **collateral connection of the feeding vessels** in the tissue
  - It is also very important that anemia occurs **suddenly or slowly.**

HYPEREMIA

Hyperemia

- It is the presence of more blood than normal in the vessels of organs or tissues in a part of the body.
- It occurs in one or more organs, tissues, and does not include the entire body.
- When more than normal blood rushes to the veins in that area and collects, the organs and tissues in that area appear bloody.
- If the permeability of the vessel is impaired, liquor-leukodiapedesis may develop, but the erythrocytes do not go out of the vessel.

Hyperemia (congestion)

Active (arterial) hyperemia

Passive (venous) hyperemia

Active hyperemia

- It is characterized by the increase of blood in the veins as a result of the **acceleration of the blood flow** in the arteries and capillaries, the enlargement of the vessel or the **stagnation of the blood flow.**

Acceleration of blood flow in the arteries:

- In this case, the amount of blood in the vein also increases.
- However, it is not possible for the liquid or cellular part of the blood in the vein to go out of the vein.
- Hyperemic areas are seen in red.
- It occurrs physiologically and this type is called **"functional hyperemia"**.

Active hyperemia

Deceleration of blood flow in the arteries:

- With various effects, especially as a result of contraction of the precapillary sphincter in the terminal arterioles, the blood stagnates in the capillaries.
- Hyperemia that occurs in this way is also called **"prestatic hyperemia".**
- As a result of stagnation, blood accumulates and the veins expand. As the blood slows down in the veins, the circulation is not complete and the oxygen decreases in these parts.
- Therefore, it appears in a darker red color than before.
- In addition, since the capillaries are filled with blood, it draws attention like tree branches in the region (arborization).

Etiopathogenesis

Physiological	Vascular occlusion	Affecting the vascular wall
Chemical effects	Physical effects	Increased blood pressure
Infectious agents	Inflammation	Shock

Etiopathogenesis

Physiological (Functional hyperemia)

- Due to the increased function of the gastrointestinal tract during digestion, arterial (active) hyperemia occurs in the mucosa of these regions.
- It is also shaped by nervous and hormonal stimulation as a result of stress. Psychologically, blushing (in cases of embarrassment or anger) depends on this.
- This type of hyperemia is also called **"reflex hyperemia"** because it is caused by reflex.

Etiopathogenesis

Vascular occlusion

- It is shaped by the increase in the pumping power of the heart, either in general with the increase in blood pressure, or locally as a result of the narrowing and occlusion of the vein in a region.
- For example; When there is a blockage in a vessel with a collateral connection, blood rushes to the lateral connections. **Collateral hyperemia** is seen in these regions.

Etiopathogenesis

Affecting the vascular wall

- **Decreased vascular tone (tension):** In this case, the arteries and capillaries relax and fill with blood. This is also called **"relaxation hyperemia".**
- **Expansion of vessels by the action of nerves:**
  - With the paralysis of vasoconstrictor nerves, vascular tone is relaxed. Hyperemia that develops in this way is called **"neuroparalytic hyperemia".**
  - Hyperemia caused by stimulation of vasodilator nerves is called **"neuroirritative hyperemia".**

Etiopathogenesis

Chemical effects

- Chemical substances generally cause general or local hyperemia by affecting the nerves.
- **Toxic effects**
- **Chemical-irritating substances**

Physical effects

- In general, it causes myogenous hyperemia by acting on the muscle layer of the vessels.
- Mostly local effect occurs; erythema (redness) develops on the skin.
- **Trauma or temperature**

Etiopathogenesis

Increased blood pressure

- Due to the acceleration of blood flowing in the veins, the face turns red in cases of hypertension in humans and also with an increase in body temperature (fever).

Infectious agents

- In general, they cause hyperemia in various organs or only in the area they affect.
- Its effects are diverse.
- For example, bacterial toxins can act on vasomotor nerves.
- Insect stings cause skin erythema.

Etiopathogenesis

Inflammation

- It is formed as a result of the acceleration of the blood in the arteries, and then its stagnation.
- As a result of the **contraction of the precapillary sphincters**, blood accumulates, especially in the capillaries, the **capillaries expand and stasis occurs.**
- The erythrocytes gather in the middle and pile up on each other like coins and become invisible by forming a column in the vein.
- **Vascular permeability** is also impaired, **liquor and leukodiapedesis** are formed, which is not functional hyperemia caused by acceleration of blood flow, but real **active hyperemia** due to slowing and stasis of blood in the arteries.
- In this case, blood stagnation also occurs in the veins of the venous system.

Etiopathogenesis

Shock

- As a result of severe effects, severe contraction occurs in the precapillaries depending on the nervous reflex, the vessels narrow, but it is short-lived.
- Immediately after this, paralysis occurs and the vessels dilate **(paralytic dilatation)** and fill with blood.

Morphological findings

Grossly

- In physiological state, it is slightly red. When it occurs in areas such as the face, the temperature of the area also increases due to the acceleration of blood.
- In active hyperemia due to stagnation of blood, the area is swollen and hot, as the vessels are also disrupted.
- Erythema (redness) is more pronounced (dark red) due to stagnation of blood and clumping of erythrocytes.

Morphological findings

Microscopically

- Excess erythrocytes are found in the veins.
- If there is stagnation of blood, erythrocytes are piled up like coins in the capillaries.
- Other signs of permeability disorder develop.

The end of active hyperemia

- It varies according to the organ in which it is located, the severity, shape and duration of the hyperemia.
- When it is in **the brain**, disorders such as fainting, bleeding and edema occur due to the pressure exerted by the blood vessels on the nerve cells.
- **Inflammatory** hyperemia is followed by other disorders.
- **Physiological** ones are temporary.

Passive hyperemia

- It is characterized by the prevention of blood flow in the veins and the accumulation of blood in the veins as a result of slowing and stopping of the blood.
- It is called **passive hyperemia** because it is caused by other disorders other than veins, such as occlusion of the arteries, the effect of pressure on the veins (such as suffocation), heart disorders, the inability of the heart to pump blood into the veins, and the inability to draw blood from the veins.
- It is also called **"congestion"** because of the collection of blood in the veins of organs and tissues.
- When congestion is mentioned, venous or passive hyperemia is understood.

Passive hyperemia

- Tissues appear blue-red (violet) and cyanotic.
- Because the erythrocytes in the veins are rich in carbon dioxide.
- Moreover, their numbers have increased due to blood stagnation.
- The appearance of organs and tissues in such a violet color is defined as cyanosis.
- Cyanotic appearance of tissues is the most important indicator of venous hyperemia.

Etiopathogenesis

Cardiac causes

Peripheral causes

Cardiac causes

- **Left heart failure:** It is mainly related to the disorder in the lungs. Since the circulation in the blood vessels is blocked, the lungs appear cyanotic, edema forms over time, and edema fluid collects in the alveoli and bronchi.

- **In acute cases,** the lungs are macroscopically cyanotic, increase in volume, have a paste-like consistency when pressed with a finger (palpation), thin foamy liquid comes from the cross-section.

- **In chronic cases**, besides color change, edema, peribronchiol interalveolar connective tissue also increases, that is, fibrosis is formed. The consistency of the lung is harder than normal (induration). heart failure cells

Cardiac causes

- **Right heart failure:** Since the general circulation is impaired, it shows its effect in organs in other parts of the body.
- Mucosa have a cyanotic appearance.
- **Spleen; stomach; liver; kidneys**

Cardiac causes

- **Intravital hypostasis:** It is a special form of cardiac passive hyperemia. It is mainly observed in agony. Since the activity of the heart decreases, the blood stagnates in the veins, especially in the veins of the organs on the side where the body lies. The relevant color change occurs in these areas.

- Another similar but unrelated change to the heart and circulation is **postmortal hypostasis**. It is characterized by the collection of blood in the veins on the side of the cadaver. The cause is circulatory cessation after death, not related to circulatory failure.

Peripheral causes

- It is not related to heart failure.
- It is related to the obstruction of blood flow in peripheral veins and the accumulation of blood in the veins.

Compression

- Tumour, abscess
- Cirrhosis
- Stomach, spleen torsion
- Intestinal, torsion, invagination, volvulus
- Strangulation

Vein obturation

- Emboli
- Varicosis

Morphological findings

- Areas of passive hyperemia are **cyanotic.**
- The organ **increases in volume**, its temperature decreases (it is **cold**), its cross section is bloody.

The end of passive hyperemia

- If the vein has **collateral connections and develops gradually**, the result will not be too bad.
- Conversely, serious consequences occur.
- As a result of stagnation, the movement of blood cells first slows down.
- Erythrocytes move slowly back and forth; this table is called **stragnation,**
- In time, the movement of erythrocytes completely stops, and **stasis** is formed. If there is a collateral connection, blood flow is provided from these parts.

The end of passive hyperemia

- **In acute cases,** when the blood stops suddenly, **necrosis** occurs in the cells of the region.
- For example, **changes in the state of the intestines** are like this.
- **Chronic passive hyperemia** consists of partial blockage of blood flow.
- For example, this is the case in **heart failure.**
- For this reason, although the circulation is partially provided, the blood fluid goes out of the vessel and **stagnation edema** is formed.
- When it becomes more chronic, there is an **increase in connective tissue** in the area.

The end of passive hyperemia

- **Stagnation edema:** With the stagnation of blood and the pressure of arterial blood, the hydrostatic pressure increases, blood fluid goes out.
- Vascular permeability is not impaired as in inflammation (active hyperemia), and therefore, the inflammatory cells do not go out.
- **Fibrosis** occurs when edema persists for a long time.
- For example, in chronic passive hyperemia, the extremities thicken like an elephant's leg due to edema and fibrosis, which is called **"elephantiasis".**

The end of passive hyperemia

- **Hemorrhagic infarction:** It is excessive blood stagnation that occurs when the venous circulation suddenly stops in a tissue.
- For example, it occurs in **pathological changes in the intestines.**
- In this case, there is no arterial circulation.
- Vascular permeability is impaired, **edema** and **hemorrhage** occur in addition to **necrosis** in the region.
- **In infarction**, on the other hand, it is necrosis (coagulation necrosis area) which is formed by the occlusion of the vessel (main arteries) without collateral connection, and whose top is on the side of the occluded vessel.

The end of passive hyperemia

- In chronic passive hyperemia, tissues and organs cannot be fed well, and **atrophy** also occurs.
- This type of atrophy seen in dark color is called **cyanotic atrophy.**
- When it lasts for a long time, there may be **ectasia** (enlargement) in the veins. Such locally observed enlargements are defined as varicosis.
- When varicosis or other conditions take a long time, thrombosis is formed in the veins.

EDEMA

Edema

- It is the accumulation of transudate (water, fluid) in tissue spaces, intercellular (extracellular) regions and body cavities.
- The term **hydrops** is used to indicate the increase in fluid in the body cavities (abdominal, thoracic cavities). For example, the accumulation of fluid in the abdominal cavity is called **ascites.**
- **Inflammatory and non-inflammatory edema** are also mentioned.
- **In inflammatory edema**, there are inflammatory cells, exfoliated tissue cells, albuminous substances (fibrin, globulin, etc.), which is exudate.
- In non-inflammatory edema, only transudate is present.

Etiopathogenesis

- It is caused by the irregularity of fluid exchange between blood and cells.
- High hydrostatic pressure and low osmotic (oncotic) pressure cause blood fluids to rise into tissue spaces.

Etiopathogenesis

High hydrostatic pressure	Low osmotic (oncotic) pressure	Na deprivation
Increased capillary permeability	Occlusion of lymphatics	Neural-hormonal effects

High hydrostatic pressure

- **Heart disorders** (such as congestive heart failure)
- **Venous obstruction** (occlusion), **compression** (pressure)
- **Arterial dilation, heart failure, or neuromuscular causes**

Low osmotic (oncotic) pressure

- It is formed due to **hypoproteinemia.**
- For example, in cases of **glomerulonephritis, malnutrition, starvation and cachexia, general edema** occurs due to protein deficiency.

Na deprivation

- **Renal failure** (reabsorption and excretion of sodium from the tubules),
- As a result of disruption of the balance of **renin-angiotensin-aldosterone** secretion

Occlusion of lymphatics

- **Lymphedema** is formed.
- For example, it occurs as a result of external compression (pressure) such as **tumor, abscess**, or internal obstruction of lymphatic vessels **(lymphangitis).**

Neural-hormonal effects

- The water balance in the body is kept under control by the **Antidiuretic hormone (ADH)** and the **Renin angiotensin and aldosterone system.**
- **ADH**; It is secreted from the posterior lobe of the pituitary. It keeps the intracellular and extracellular water in the body under control.
- If the osmotic pressure of the extracellular water is high (if it is watery), it has a stimulating effect and ADH is released from the pituitary.
- It acts on the distal tubules of the kidneys, especially the tubulus contortus II and collecting ducts, and ensures the removal of water from the kidney.
- If the osmotic pressure of the extracellular water is low, the release of ADH stops.

Renin-angiotensin-aldosterone system (RAAS)

- **Renin** is secreted in relation to blood pressure (when **blood pressure decreases**) from the juxtaglomerular cells, where afferent arterioles are located in the kidneys.
- This stimulates the production of angiotensin II in the blood, which controls vascular pressure.
- The increase in angiotensin II in the blood stimulates the release of aldosterone from the adrenal glands.

Renin-angiotensin-aldosterone system (RAAS)

- **Aldosterone** is a mineralocorticoid hormone released from the zona glomerulosa cells of the adrenocortex. It is referred to as the **renin-angiotensin-aldosterone system** because it is related to **renin-angiotensin.**
- Aldosterone secretion increases Na+ reabsorption from the kidney and facilitates K+ and H+ excretion.
- The reabsorption of sodium also causes the reabsorption of water.
- The blood volume and therefore blood pressure increase.
- An increase in blood pressure reduces the release of renin from the kidneys, and this mechanism works in reverse.

Types of edema

Systemic edema

- **Heart disorders** (inhibition of general circulation),
- **hypoproteinemia,**
- parasites common to foods, especially **gastrointestinal parasites,**
- Causes such as **kidney diseases that cause sodium and protein loss** cause edema formation in most parts of the body.

Types of edema

Local edema

- It depends on the circulatory disorder of regional vessels (blood, lymph).
- In this case, hydrops occurs in the thoracic or abdominal cavity.
- For example, if the V. porta is under pressure in liver cirrhosis, hydrops and ascites occur in the abdominal cavity.
- However, the same event may occur with general edema in right heart failure.

Types of edema

- **Edema related to venous stagnation:** It occurs in right heart failure in the form of general edema. It is especially shaped in the subcutaneous and extremities. This is called cardiac edema.
- **Renal edema:** Occurs in kidney diseases.
- **Chemical-toxic, allergic edema:** Various chemical-toxic substances that impair vascular permeability; bee, insect stings, bacterial toxins
- **Inflammatory edema:**

Types of edema

- **Hepatic edema:** It develops in cirrhosis related to both the pressure on the V. porta and the decrease in protein production.
- **Agonal edema:** The weakening of the heart occurs with the deterioration of the general circulation. It is formed especially in the lungs. Fluid accumulates in the abdominal and thoracic cavities.
- **Hypoproteinemic edema:** It occurs in starvation, parasite infestation, kidney diseases. It is systemic and occurs with general findings.

Types of edema

- **Hydrops congenitus:** Congenital.
- **Hydrops amnii:** It is hydrops of the amniotic sac in pregnancy.
- **Hydrocele:** It is local edema that occurs in the scrotum.
- **Oedema ex vacuo:** It occurs as a result of cachexia, especially the deprivation of fat of the adipose tissue around the kidney and heart, and the influx of water into the interstitial areas where collagen is present.

Types of edema

- **Brain edema:** It occurs due to various reasons, including trauma, heat stroke (hit), impaired venous circulation or congenital.
- **Hydrocephalus externus:** Fluid collection in the meninges outside the brain.
- **Hydrocephalus internus:** It is more fluid than normal in the ventricles, it is congenital or occurs later.
- **Hydranencephaly:** A large, fluid-filled cavity in the area normally occupied by CNS tissue of the cerebral hemispheres resulting from abnormal development.

Types of edema

- **Pulmonary edema:** The lungs are heavy, non-collapsing, pale, and fluid oozes from the cross-section. There is also a thin foamy fluid in the trachea and bronchi. It occurs mainly in **left ventricular failure.**

- **Skin edema:** It is the most common form of edema. It is formed by local and systemic reasons. The edematous skin parts are swollen and tense; When it is palpated, it is in the consistency of dough and fingerprints remain. When the section is made, fluid comes out or is moist depending on the severity of the edema. If severe, subcutaneous mushy, watery, gelatinous appearance.

- If there is widespread edema on the skin and it is severe, it is called **anasarca,** it is mainly congenital.

Types of edema

- **Hydropericardium;** in the heart sac
- **Hydrothorax;** in the thoracic cavity
- **Hydroperitoneum, hydrops ascites;** in the abdominal cavity
- **Hydrarthrosis;** is the accumulation of fluid in the joint cavity.

The end of edema

- It creates **dysfunction** according to its location.
- As a result of **brain edema**, life centers are affected, unconsciousness and death occur.
- In **pulmonary edema**, breathing is impaired and results in death. In mild cases, bacteria grow in the edema fluid and pneumonia occurs. (especially in heart failure, pneumonia-related deaths are common).

The end of edema

- It becomes **chronic.**
- Connective tissue collagen increases. When it is in the lung, the connective tissue collagen increases as well as edema.
- Connective tissue cells and collagen fibers increase when it is formed due to circulatory disorders, especially in the extremities and often in regional vessels. **Elephantiasis** occurs in the leg.

DEHYDRATION

Dehydration

- The loss of water from the body is the decrease of body water below normal.
- Along with water, electrolyte loss also occurs and the body's fluid-electrolyte balance is disrupted.
- As a result of the loss of fluid, **exsiccosis** occurs in the body.
- **Exsiccosis** (drying): It is characterized by drying of tissues, increase in blood viscosity, decrease in body secrets and excreta such as saliva, sweat, decrease in hormone production (due to thirst), inability to excrete toxic metabolic products such as urea, and ultimately deterioration of metabolism and death.

Dehydration

- **Primary dehydration** is caused by avoiding drinking water and taking less water.
  - The amount of water in the blood decreases and anhydremia occurs.
  - Water is drawn from tissues to maintain blood volume.
- **Secondary dehydration** occurs when excess water is removed from the body.
  - In this case, electrolyte loss is also formed, and the viscosity of the blood also increases.
  - It occurs in conditions such as diarrhea, vomiting, diabetes, excessive sweating, blood loss, kidney diseases.

ACIDOSIS and ALKALOSIS

- It is caused by the deterioration of the acid-base balance in the body. There is a loss of electrolytes.

Metabolic acidosis

- It is formed as a result of sodium and bicarbonate loss.
- Diarrhea, kidney failure, diabetes, ketosis, starvation, shock are the main causes.

Metabolic alkalosis

- With the effect of adrenal steroids, excessive sodium absorption from the kidneys, H+ and potassium excretion, respiratory disorder develops if CO^2 excretion is insufficient.