Pathology - Topic 5 - Hemodynamic disorders

Oedema

- **Oedema** is excess fluid within the interstitial tissues.

Oedema, Effusions and Distribution

- Fluid may also accumulate in body cavities, producing a pleural effusion, a pericardial effusion or ascites.
- **Anasarca** is severe generalised oedema, whereas localised oedema affects a limited region.
    - The distribution helps identify whether the main problem is systemic or local.

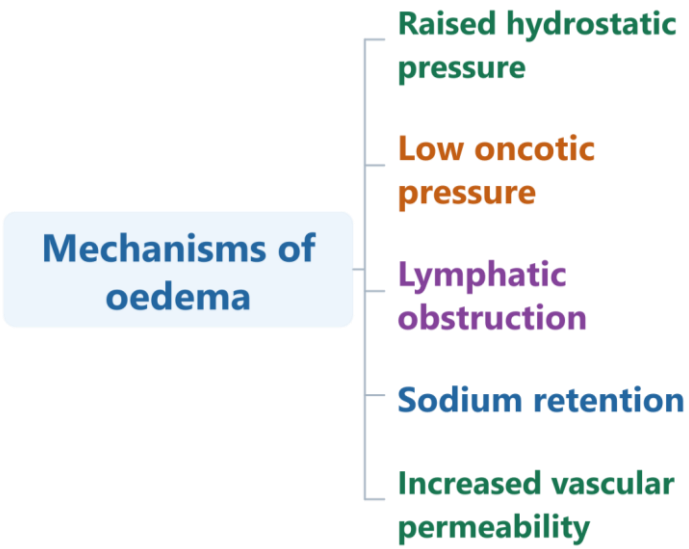
Normal Fluid Balance and Its Disturbance

- Normal fluid balance depends on vascular pressures, plasma proteins, endothelial integrity and lymphatic drainage.
    - A disturbance in any of these factors can increase tissue fluid.

Transudates, Exudates and Appropriate Criteria

- A transudate is usually relatively protein-poor fluid produced by hydrostatic or oncotic imbalance, whereas an exudate reflects increased permeability and is relatively rich in protein and cells.
- Classification of a sampled effusion uses criteria appropriate to that body cavity rather than its appearance alone.

Mechanisms of Oedema



1. Increased Hydrostatic Pressure

- Raised venous pressure increases fluid movement from small vessels into the interstitium.
    - Heart failure can produce widespread venous congestion, while a venous thrombosis produces more local swelling.

Copyright by Engacy Learning Private Limited. All rights reserved. Reproduction or distribution without written consent is strictly prohibited.

- Dependent ankle oedema is often prominent in ambulant patients, while sacral oedema may be more apparent in a person lying in bed.

2. Reduced Plasma Oncotic Pressure

- Low plasma albumin reduces the force that normally helps retain fluid within the circulation.
    - Causes include urinary protein loss in nephrotic syndrome, reduced synthesis in severe liver disease and severe protein deficiency.
- Loss of effective circulating volume can activate sodium-retaining mechanisms and worsen oedema.

3. Lymphatic Obstruction

- Blocked lymphatic drainage prevents removal of interstitial fluid and proteins.
    - Tumour infiltration, surgical lymph-node removal, fibrosis and filarial infection are possible causes.
- Long-standing lymphoedema can become firm and less pitting because fibrosis develops.

4. Sodium and Water Retention

- Renal sodium retention expands extracellular fluid volume and can increase hydrostatic pressure.
    - It occurs in renal disease and as a secondary response to reduced effective circulation in some cardiac and hepatic disorders.

5. Increased Vascular Permeability

- Inflammation allows proteins and fluid to escape through the vascular barrier.
    - This produces a relatively protein-rich exudate and may accompany redness, warmth and inflammatory cells.

Morphology and Consequences

1. Subcutaneous Tissue

- Subcutaneous oedema separates connective-tissue elements with clear fluid.
    - Pressure may leave a temporary indentation called pitting, although not every form of oedema pits.

2. Lungs

- Oedematous lungs are heavy and wet, and fluid may escape from the cut surface.
    - Microscopy shows pale fluid within alveolar spaces and congestion of capillaries.
- Pulmonary oedema increases the diffusion distance for gases and can seriously impair oxygenation.

Copyright by Engacy Learning Private Limited. All rights reserved. Reproduction or distribution without written consent is strictly prohibited.

3. Brain

- Cerebral oedema increases brain volume within the fixed space of the skull.
  - Swollen gyri and narrowed sulci may be present, and severe swelling can produce dangerous brain displacement or herniation.

Hyperaemia and Congestion

- Hyperaemia and congestion both increase the amount of blood in a tissue, but hyperaemia reflects increased inflow and congestion reflects impaired outflow.

Active and Passive Increases in Blood Volume

1. Hyperaemia

- **Hyperaemia** is an active increase in tissue blood flow caused by arteriolar dilation.
  - The tissue is usually red and warm because it contains more oxygenated blood.
- Exercise in skeletal muscle and acute inflammation are common examples.

2. Congestion

- **Congestion** is a passive increase in tissue blood volume caused by impaired venous outflow.
  - The tissue may appear blue-red because it contains more deoxygenated blood.
- Persistent congestion causes hypoxia, oedema, small haemorrhages and eventual fibrosis.

Chronic Congestion in Important Organs

1. Lungs

- Left-sided heart failure can produce pulmonary venous congestion.
  - Capillaries within alveolar septa become distended, and red cells may enter alveolar spaces.
- Macrophages ingest red-cell breakdown products and become haemosiderin-laden **heart-failure cells**.
  - Long-standing congestion and fibrosis may produce a firm brown lung, historically called brown induration.

2. Liver

Gross and Microscopic Appearance

- Impaired venous drainage causes congestion around central veins in the liver.
- The dark congested centrilobular zones contrast with paler surrounding tissue, producing a nutmeg appearance.

Copyright by Engacy Learning Private Limited. All rights reserved. Reproduction or distribution without written consent is strictly prohibited.

- Severe or prolonged congestion can cause centrilobular hypoxic injury and fibrosis.
- The resulting pattern reflects impaired outflow rather than primary inflammation of every hepatic lobule.
- Periportal areas may appear paler or fatty beside the congested centrilobular zones.

Hypoxic Injury and Fibrosis

- Centrilobular hepatocytes are especially vulnerable because their oxygen supply is already relatively low.
- Prolonged severe cardiac congestion may produce central-to-central fibrosis and advanced cardiac-associated cirrhosis.

Haemorrhage

- **Haemorrhage** is escape of blood from the cardiovascular system.

Sites and Patterns of Bleeding

- It may be external, enter a body cavity or remain within tissue as a haematoma.
- **Petechiae** are tiny pinpoint haemorrhages, while **purpura** refers to larger small haemorrhagic lesions.
  - **Ecchymoses** are larger bruises, although size terminology may vary slightly between clinical settings.

Causes and Bleeding Patterns

- Bleeding may result from vessel injury, platelet disorders, coagulation defects or combinations of these mechanisms.
  - A platelet problem often produces mucocutaneous bleeding, while severe coagulation-factor defects often cause deeper bleeding.

Clinical Consequences and Named Effusions

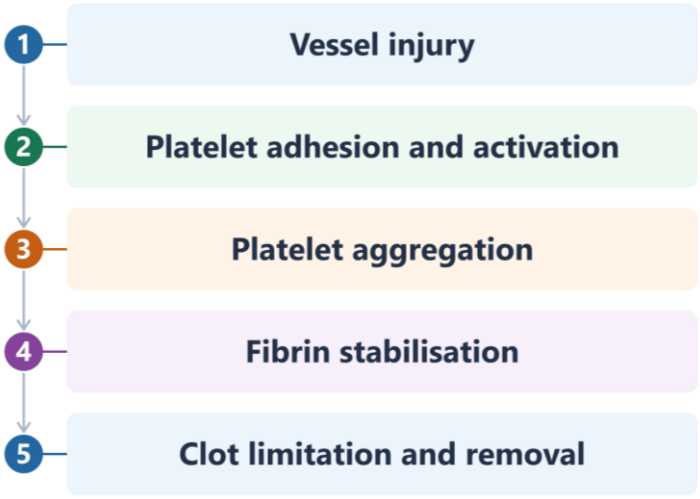
- The effect depends on the amount, rate and anatomical site of blood loss.
  - A small intracranial haemorrhage can be critical, whereas a similar volume in subcutaneous tissue may be minor.
- Rapid substantial blood loss can cause shock, while repeated external blood loss can cause iron deficiency.
  - Iron from an internal haematoma is usually recycled rather than lost from the body.
- Blood in pleural, pericardial and peritoneal cavities is termed haemothorax, haemopericardium and haemoperitoneum respectively.

Copyright by Engacy Learning Private Limited. All rights reserved. Reproduction or distribution without written consent is strictly prohibited.

Normal Haemostasis

- Haemostasis is the regulated process that stops bleeding at an injured vessel while maintaining blood flow elsewhere.

Stages and Regulation of Haemostasis



1. Vascular Response and Platelet Adhesion

- Vascular injury causes a brief local vasoconstrictor response that reduces blood loss.
- Exposure of subendothelial matrix allows platelets to adhere through interactions that include von Willebrand factor and platelet glycoprotein Ib.
    - Adherent platelets change shape and become activated.

2. Platelet Activation and Aggregation

- Activated platelets release mediators such as ADP and produce thromboxane A2, which recruit and activate more platelets.
    - Activated glycoprotein IIb/IIIa receptors bind fibrinogen and link platelets together.
- This primary platelet plug initially provides a relatively fragile seal.

3. Coagulation and Fibrin Stabilisation

- Tissue factor and coagulation reactions generate thrombin at the injured site.
    - Thrombin converts fibrinogen into fibrin and amplifies platelet and coagulation responses.
- Cross-linked fibrin stabilises the plug and traps additional blood cells.
- Activated factor XIII cross-links fibrin and increases the stability of the clot.

4. Restriction and Removal of the Clot

Endothelial Restraint

- Intact endothelium limits platelet activation through nitric oxide and prostacyclin and provides a surface that favours anticoagulant mechanisms.

Copyright by Engacy Learning Private Limited. All rights reserved. Reproduction or distribution without written consent is strictly prohibited.

FREE PREVIEW

Click here to visit our website

MBBS PREMIUM NOTES

Complete MBBS notes for every subject and year are available only in the FirstHope app.

Scan or click here

Click here to get the app >

Available on Google Play

Browse MBBS notes by year

firsthope.co.in/mbbs

Click here to explore MBBS >

Scan or click here

FirstHope Medical on Instagram

@firsthope.medical

Click here to follow

App and notes help

Find answers about access and downloads

Click here for help

YEAR 1 / YEAR 2 / YEAR 3 / YEAR 4