HAEMODYNAMIC DISORDERS, THROMBOEMBOLIC DISEASE, and SHOCK

TOPIC 1 EDEMA & EFFUSION

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References:

- Robbins & Cotran Pathologic Basis of Disease- 9th edition
- Davidson's Principles and Practice of Medicine-23rd edition
- IMAGES- Above mentioned books & internet







- 1. EDEMA & EFFUSION
- 2. THROMBOSIS
- 3. EMBOLISM
- 4. INFARCTION
- 5. SHOCK
- 6. HYPEREMIA & CONGESTION
- 7. HEAMORRHAGE
- 8. DISSEMINATED INTRAVASCULAR COAGULATION (DIC)





HAEMODYNAMIC DISORDERS



- Haemo- related to blood
- Dynamics- concerned with the motion of bodies under the action of forces.
- Haemodynamics -Is the study of dynamics of blood flow or the circulation



HAEMODYNAMIC DISORDERS

- The health and well being of cells and tissues depends not only on the circulation of blood which delivers oxygen but also on normal fluid balance
- Alteration of any of these results in some disorders known as haemodynamic disorders





- **Hyperemia & Congestion**
- Shock

Disorders of abnormal bleeding and clotting

HAEMODYNAMIC DISORDERS,

- Hemorrhage
- Thrombosis
- Embolism
- Infarction





EDEMA & EFFUSION





TOPICS

- Definition
- Pathophysiology
- Types and examples
- Morphology
- Clinical significance
- Renal edema
- Cardiac edema
- Hepatic edema
- Pulmonary edema
- Cerebral edema





EDEMA- DEFINITION

Accumulation of fluid in the interstitial tissue spaces



Interstitial spaces

Interstitial Tissue Space

At venous end of capillary: predominant movement of fluid is from interstitial spaces into bloodstream.

At arterial end of capillary: predominant movement of fluid is from bloodstream into interstitial spaces. Excess fluid and escaped protein drain into lymphatic vessels.





EFFUSION DEFINITION

Accumulation of fluid in the body cavities





EFFUSION

Hydrothorax (pleural effusion) Hydropericardium Hydroperitoneum (ascites)



Pleural effusion



This is a right sided pleural effusion (in a baby)The fluid is clear, pale yellow in appearance (serous effusion)





Pleural effusions



Bilateral pleural effusions. The fluid appears reddish, because there has been hemorrhage into the effusion





ASCITES

 It is clinically detectable when at least 500 mL have accumulated

CAUSES:

- Malignant disease
- Cardiac failure
- Hepatic cirrhosis







Three major fluid compartments

Intravascular

Body fluid compartments

- Interstitial and
- Intracellular
- <u>60%</u> of body weight is water (in a healthy adult)
- 2/3rd (40%) is *INTRACELLULAR*
- 15% is *INTERSTITIAL*
- Only 5% is *INTRA-VASCULAR* (plasma)







MECHANISM OF NORMAL CONTROL IN SYSTEMIC CIRCULATION





Starling's Hypothesis

- states that the fluid movement across the capillary wall is dependent on the balance between
- the hydrostatic pressure gradient and
- the oncotic pressure gradient across the capillary







Factors affecting fluid balance across capillary wall

INTERSTITIAL SPACES













Increased Hydrostatic Pressure

Reduced Plasma Osmotic Pressure (Hypoproteinemia) Lymphatic Obstruction Sodium Retention Inflammation





I. Increased Hydrostatic Pressure

A. Impaired venous return

i.

- Congestive heart failure
- ii. Constrictive pericarditis
- iii. Ascites (liver cirrhosis)
- iv. Venous obstruction or compression
 - a. Thrombosis
 - b. External pressure (e.g., mass)
 - c. Lower extremity inactivity with prolonged dependency
- B. Arteriolar dilation
 - i. Heat
 - ii. Neurohumoral dysregulation



II. Reduced Plasma Osmotic Pressure (Hypoproteinemia)

- A. Protein-losing glomerulopathies (nephrotic syndrome)
- B. Liver cirrhosis (ascites)
- C. Malnutrition
- D. Protein -losing gastroenteropathy



III. Lymphatic Obstruction

- A. Inflammatory
- **B. Neoplastic**
- C. Postsurgical
- **D.Post irradiation**



IV. Sodium Retention

- A. Excessive salt intake with renal insufficiency
- B. Increased tubular reabsorption of sodium
 - i. Renal Hypoperfusion
 - ii. Increased secretion of
 - renin- angiotensin- aldosterone



V. Inflammation

- A. Acute inflammation
- B. Chronic inflammation
- C. Angiogenesis





I. PathophysiologyII. Extent of involvementIII. Clinical basis





I. On the basis of pathophysiology

- INFLAMMATORY Due to increased vascular permeability <u>(exudate)</u>
- NON-INFLAMMATORY Due to alterations in haemodynamic forces across the capillary wall <u>(transudate)</u>





II. On the basis of extent of involvement LOCAL – pulmonary, cerebral, effusions GENERALISED- anasarca





III. On clinical basis

- PITTING
- NON PITTING

Comparison of an exudate with a transudate

| | EXUDATE | TRANSUDATE |
|--------------------------|------------------------------------|--|
| Total protein content | High, as in plasma>3g/dl | Low, <3 g/dl |
| Distribution of protein | As in plasma | Nearly all albumin |
| Fibrinogen | Present, as it clots spontaneously | Not present. No tendency to coagulate |
| Specific gravity | High (over 1.018) | Low (about 1.012) |
| Inflammatory cells | Plentiful; (poly,lympho) | Few present; nearly all mesothelial cell |





Pitting edema of the leg

A. In a patient with congestive heart failure, severe edema of the leg is demonstrated by applying pressure with a finger.

B. The resulting "pitting" reflects the inelasticity of the fluid-filled tissue.







Non pitting edema (Pretibial myxedema)







MORPHOLOGY OF EDEMA

Edema is most easily recognized grossly Microscopically, clearing and separation of the extracellular matrix

elements.

 Normal appendix



Appendicitis






MORPHOLOGY OF EDEMA

- Although any organ or tissue in the body may be involved,
- Most commonly encountered in
- subcutaneous tissues
- the lungs and
- the brain





MORPHOLOGY OF EDEMA

Subcutaneous

Variable distribution Diffuse Dependent : Cardiac failure (legs and sacral area) Periorbital :

Nephrotic syndrome



Dependent edema







MORPHOLOGY OF EDEMA

Severe, generalized edema is called anasarca





CLINICAL FEATURES

Merely annoying to fatal

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CLINICAL FEATURES

Subcutaneous edema

In cardiac & renal failure subcutaneous
edema signals underlying disease
May impair wound healing &
clearance of infection





CLINICAL FEATURES Pulmonary edema

- Interferes with normal ventilatory function
- Favours bacterial infection



CLINICAL FEATURES Brain edema

- Can be rapidly *fatal*
- Brain substance can herniate through foramen magnum
- Brain stem vascular supply can be compressed
- Both the conditions can injure medullary centre & cause death





Laryngeal edema

Fatal



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LOCAL EDEMA

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Local edema- causes

Acute Inflammation

Hypersensitivity reaction

Venous obstruction

Lymphatic obstruction





Acute inflammation acute appendicitis (local edema)





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Acute inflammation

acute cholecystitis

(local edema)





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Local edema (Cont)

- Vascular permeability seen in
- type l
 - type III &

Hypersensitivity edema

type IV hypersensitivity reaction

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Local edema

Edema due to venous obstruction Examples

- Venous thrombosis
- A tumour / growth compressing a vein Increased hydrostatic pressure proximal to the obstruction





Local edema (Cont)

Edema due to lymphatic obstruction

Examples :-

- (a) Filariasis
- (b) Recurrent bacterial lymphangitis
- (c) Lymphatic edema of arm & peau d' orange appearance of the breast in breast carcinoma





Filariasis



- W. bancrofti parasites are mainly transmitted by Culex quinquefasciatus mosquitoes and some species of Anopheles
- Brugia parasites are mainly transmitted by Mansonia mosquitoes



Edema secondary to lymphatic obstruction. Massive edema of the right lower extremity (elephantiasis) in a patient with obstruction of lymphatic drainage.





Peau d' orange appearance in breast carcinoma

 Peau d Orange is a French term, which means "skin of an orange."







GENERALIZED EDEMA





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Generalized edema

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Nephrotic syndrome

CAUSES:

Heart failure

Renal failure

Malnutrition

Cirrhosis of liver

Mechanism of systemic edema in heart failure, renal failure, malnutrition, hepatic failure and nephrotic syndrome **HEART FAILURE** MALNUTRITION, HEPATIC SYNTHESIS, NEPHROTIC SYNDROME Renal blood flow ↑ Capillary Plasma albumin hydrostatic pressure Activation of the renin-angiotensin system RENAL **Retention of** Na⁺ and H₂O FAILURE ▲ Blood volume Plasma osmotic pressure **EDEMA Tamanna Choudhury**



Renin Angiotensin Aldosterone axis





Nephrotic Syndrome

- Massive proteinuria (daily loss of 3.5gm or more of protein)
- Hypoalbuminaemia- plasma albumin level less than 3gm/L
- Generalized edema
- Hyperlipidemia and lipiduria



Edema in Nephrotic Syndrome

 Nephrotic syndrome is caused by a derangement in glomerular capillary walls resulting in increased permeability to plasma proteins



NEPHROTIC SYNDROMEpathogenesis

Leaky glomerular capillary wall

Heavy proteinuria

Hypoproteinaemia

Decreased colloidal osmotic pressure

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EDEMA





Edema in Nephrotic Syndrome





EDEMA IN CIRRHOSIS OF LIVER





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EDEMA (ascites) IN LIVER CIRRHOSIS

- Increased hydrostatic pressure: elevated pressure in the veins running through the liver (sinusoidal hypertension)
- Percolation of hepatic lymph into the peritoneal cavity
- Splanchnic vasodilation and hyperdynamic circulation
- Hypoalbuminemia: a decrease in liver function caused by scarring of the liver, i.e., cirrhosis.
- Failure in hepatic inactivation of aldosterone





Splanchnic circulation

Systemic, portal, hepatic circulation







EDEMA IN CIRRHOSIS OF LIVER

- 1. Increased hydrostatic pressure
- 2. Percolation of hepatic lymph into the peritoneal cavity
- 3. Splanchnic vasodilation and hyperdynamic circulation
- 4. Hypoalbuminemia
- 5. Failure in hepatic inactivation of aldosterone



Ref: Davidson's Principle & Practice of Medicine. 23rd ed. Chapter 22





PULMONARY EDEMA



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CLASSIFICATION OF PULMONARY OEDEMA

A. HEMODYNAMIC EDEMA

B. EDEMA DUE TO MICROVASCULAR INJURY

C. EDEMA OF UNDETERMINED ORIGIN





CAUSES OF PULMONARY OEDEMA (Cont)

EDEMA DUE TO HAEMODYNAMIC CAUSE Increased Hydrostatic pressure

- Left heart failure
- Volume overload

Decreased oncotic pressure

- Nephrotic syndrome
- Liver disease

Lymphatic obstruction





CAUSES OF PULMONARY OEDEMA(Cont)

EDEMA DUE TO MICROVASCULAR INJURY

- Infectious agents : viruses, mycoplasma, other
- Inhaled gases : oxygen, sulfur dioxide, cyanates, smoke
- Liquid aspiration : gastric contents, neardrowning
- Drugs and chemicals:



CAUSES OF PULMONARY OEDEMA(Cont)

EDEMA OF UNDETERMINED ORIGIN

High altitudeNeurogenic



Pulmonary edema

Grossly: Weight Frothy, blood tinged fluid – on section M/E : Alveolar capillaries engorged Intra-alveolar precipitate Alveolar microhaemorrhage Hemosiderin laden macrophages (heart failure cell)



Fluid is expressed from the cut surfaces of both








CEREBRAL EDEMA

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Cerebral edema- causes

- Primary or metastatic tumour
- Infarction
- Contusion
- Abscess
- Encephalitis



MORPHOLOGY OF CEREBRAL EDEMA

Cerebral edema:

- Swollen
- Softer
- > Gyri Flattened
- Sulci Narrowed
- Ventricular cavities – compressed





What we have learnt in this class

- Definition- edema, effusion
- Pathophysiological categories of edema
- Types of edema
- Mechanism of edema- heart failure, renal failure, nephrotic syndrome, hepatic cause (Edema in cirrhosis)
- Morphology of edema
- Local edema- causes & examples
- Pulmonary edema
- Cerebral edema

Practice questions

- 1. Define edema.
- 2. What are the **types** of edema ? What is **anasarca**?
- 3. What are the **pathophysiological categories** of edema?
- 4. What are the causes of **generalized edema**?
- 5. What are the causes of **localized edema**? Give few examples of localized edema.
- 6. What are the differences between **exudates & transudates**?
- 7. What is the **clinical significance** of edema?
- 8. Discuss the mechanism of edema in heart failure.
- 9. Discuss the mechanism of edema in **nephrotic syndrome**.
- 10. What is the mechanism of edema in liver cirrhosis?

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- 11. What are the causes of **pulmonary edema**? What is heart failure cell?
- 12. Mention few causes of **cerebral** edema.



Edema occurs due to

- a) Decreased hydrostatic pressure
- b) Lymphatic obstruction
- c) Increased plasma colloidal osmotic pressure
- d) Sodium retention
- e) inflammation



Edema occurs due to

- a) Decreased hydrostatic pressure F
- b) Lymphatic obstruction
- c) Increased plasma colloidal osmotic pressure F
- d) Sodium retention
- e) Inflammation



Exudate is associated with

- a) Low protein content
- b) Inflammation
- c) Low specific gravity
- d) Increased tendency to clot
- e) High cellular content



F

Exudate is associated with

- a) Low protein content F
- b) Inflammation
- c) Low specific gravity
- d) Increased tendency to clot T
- e) High cellular content T



Local edema occurs in

- a) Inflammation
- b) Neoplasia
- c) Lymphatic obstruction
- d) Hypersensitivity reaction
- e) Malnutrition



Local edema occurs in

- a) Inflammation
 b) Neoplasia
 c) Lymphatic obstruction
 d) Hypersensitivity reaction
- e) Malnutrition

F

Т

F



Edema due to sodium retention occurs in

- a) Congestive cardiac failure
- b) Nephrotic syndrome
- c) Cirrhosis of liver
- d) Filariasis
- e) Increased tubular reabsorption of sodium



Edema due to sodium retention occurs in

- a) Congestive cardiac failure
- b) Nephrotic syndrome F
- c) Cirrhosis of liver
- d) Filariasis F
- e) Increased tubular reabsorption of sodium

F

F





Hypoproteinemia causes edema in

- a) Congestive cardiac failure
- b) Liver cirrhosis
- c) Nephrotic syndrome
- d) Protein losing enteropathy
- e) Filariasis



Hypoproteinemia causes edema in

- a) Congestive cardiac failure
- b) Liver cirrhosis
- c) Nephrotic syndrome
- d) Protein losing enteropathy
- e) Filariasis

F

F



