SNS COLLEGE OF ALLIED HEALTH SCIENCES

SNS Kalvi Nagar, Coimbatore - 35

Affiliated to Dr MGR Medical University, Chennai

DEPARTMENT OF CARDIOPULMONARY PERFUSION CARE TECHNOLOGY

COURSE NAME: PATHOLOGY II

II YEAR

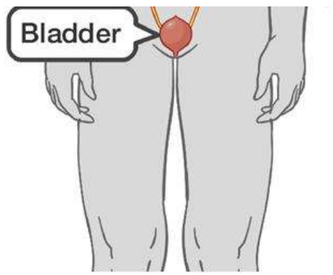
UNIT III : PATHOLOGY OF KIDNEY

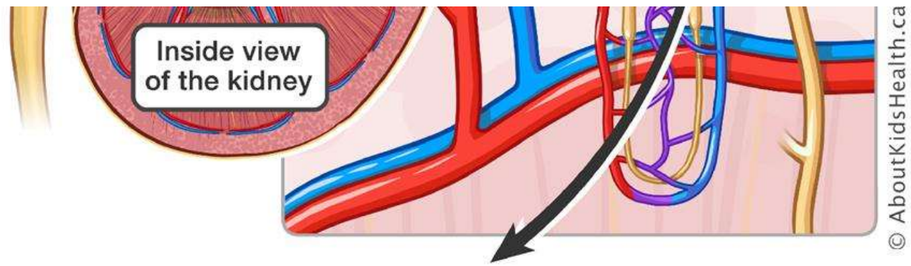
TOPIC 6 : NEPHROTIC SYNDROME

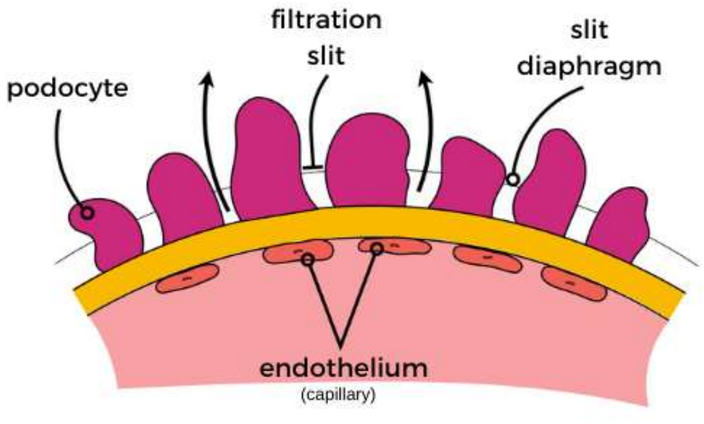
Introduction

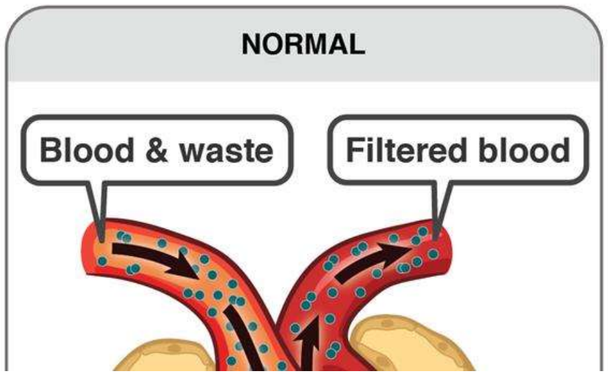
It is glomerular basement membrane disease characterised by

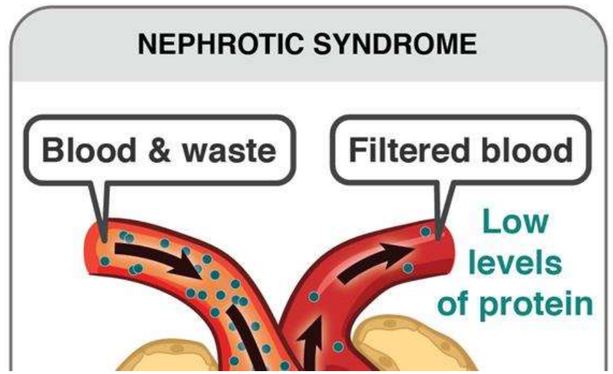
- Massive proteinuria
- Hypoalbuminemia
- Hyperlipidemia and lipiduria
- Generalized edema











Causes

Primary causes

- Membranous nephropathy
- Minimal change disease
- Focal segmental glomerulonephritis
- Membranoproliferative glomerulonephritis
- Other proliferative glomerulonephritis (Ig A nephropathy)

Secondary causes

- Diabetes Mellitus
- Amyloidosis, SLE
- Drugs (NSAID, Pencillamine, heroine)
- Infections (malaria, syphilis, hepatitis B and C, HIV)
- Malignant disease (carcinoma, lymphoma)
- Miscellaneous (Bee-sting allergy, hereditary nephritis)

Pathophysiology

Massive proteinuria

- Daily loss of 3.5g or more of protein/24hrs
- Normal a small amount of protein i.e. 20 to 150 mg/day passes through the glomerular filtration barriers and is reabsorbed by the tubules.
- ***If excess of protein is filtered exceeding the capacity of tubules for reabsorption, these proteins appear in urine.***
- Highly selective proteinuria consists of low molecular weight proteins (albumin, 70kd, transferrin, 76kd molecular weight)
- Poorly selective proteinuria – high molecular weight globulins in addition to albumin
- In nephrotic syndrome there is loss of albumin (molecular weight 66,000)

Hypoalbuminemia

- The normal range is **3.4 to 5.4 g/dL**
- With plasma albumin levels less than 3g/dl
- These may be due to

  Increased loss of albumin

  Increased renal catabolism

  Inadequate hepatic synthesis of protein

Hyperlipidemia

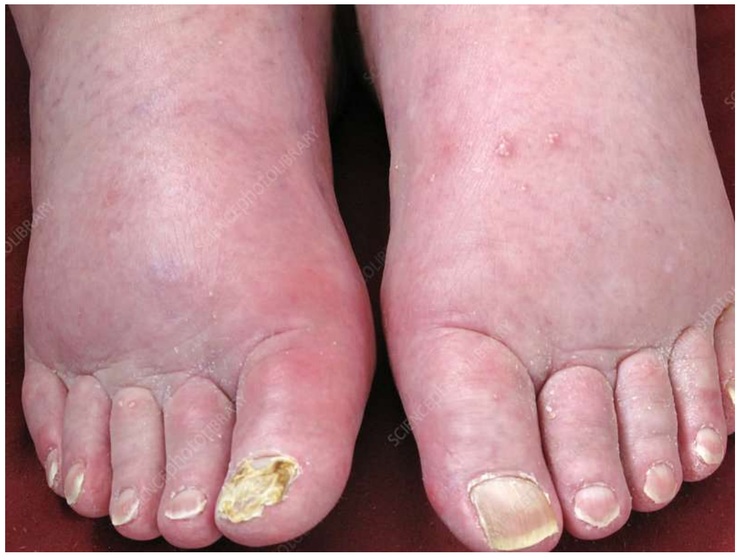
- Exact mechanism is not known but may be due to stress on liver for synthesis of proteins to compensate proteinuria
- This causes increased synthesis of lipoproteins and there is increased blood levels of total lipids, cholesterol, triglycerides, VLDL and LDL but decrease in HDL
- Increased lipoproteins may also be due to abnormal transport of circulating lipid particles and increased lipid catabolism
- Lipidemia is followed by lipiduria

Generalised edema

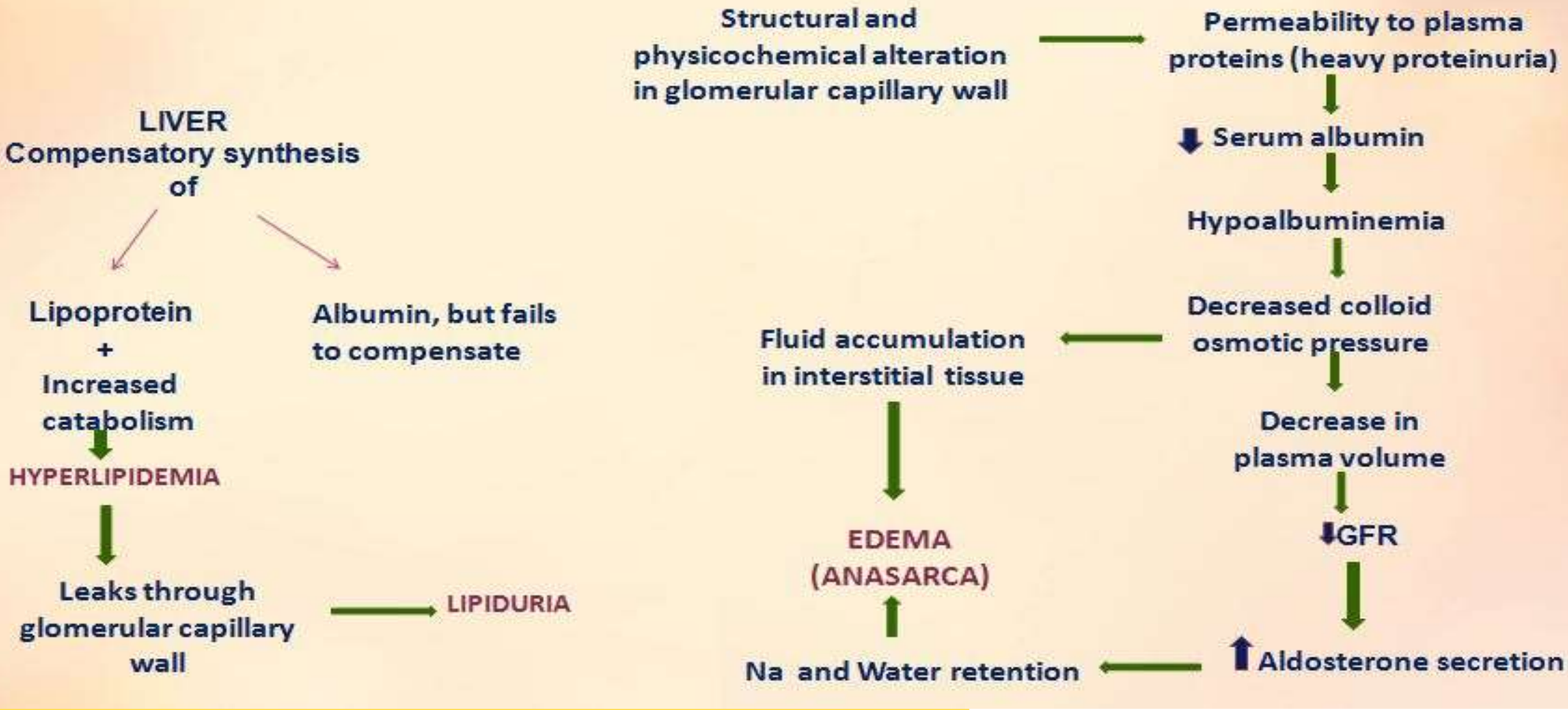
- Due to fall in colloid osmotic pressure consequent upon hypoalbuminemia

- Na and H2O retention further contribute to oedema

- Na and water retention may due to compensatory secretion of aldosterone mediated by the hypovolemia enhanced renin secretion, stimulation of sympathetic system and a reduction in the secretion of natriuretic factors such as atrial peptides

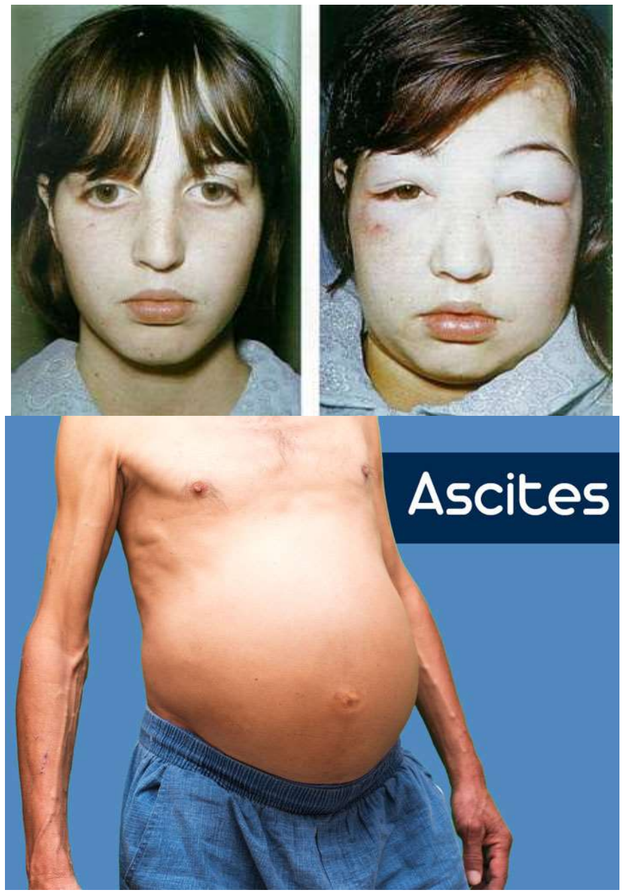


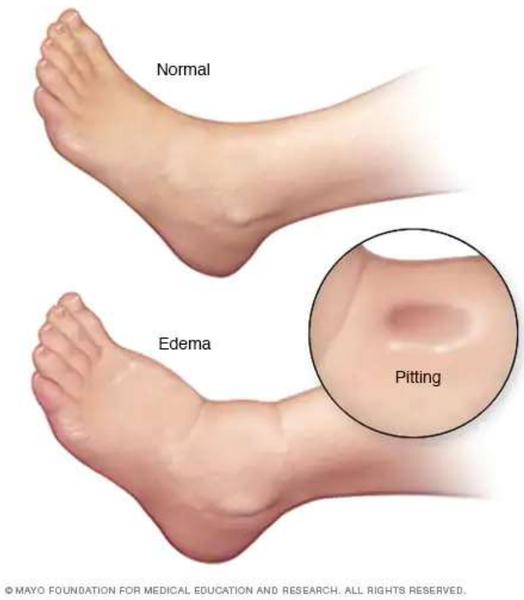
PATHOPHYSIOLOGY OF NEPHROTIC SYNDROME



Clinical features

- Puffiness around the eyes
- Pitting edema over the legs
- Pleural effusion and pulmonary edema
- Ascites
- Hypertension
- Anemia
- Dyspnea
- Anorexia
- Fatique
- Abdominal pain
- Diarrhoea

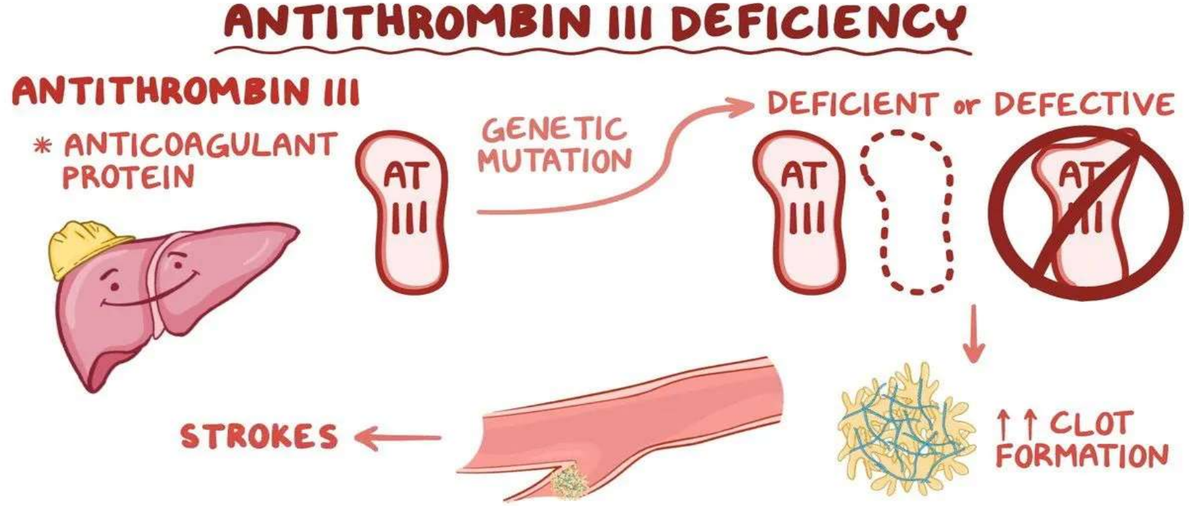




Clinical Manifestations

Patients develop arterial and venous thrombosis due to

- Increases urinary loss of anti-thrombin –III
- Hyper-fibrinogenemia from increased synthesis in liver
- Decrease in fibrinolysis
- Increases platelet aggregation
- Altered levels of proteins C



Complications

Nephrotic syndrome patients are vulnerable for

- **Infection** – especially staphylococcal and pneumococcal probably due to loss of immunoglobulin in urine

- **Thrombotic and thromboembolic complication** due to loss of endogenous anticoagulants (Antithrombin III) in urine

- **Renal vein thrombosis** seen in membranous nephropathy as a consequence of hypercoagulable state.

Difference between nephrotic and nephritic

Nephrotic Syndrome	Nephritic Syndrome
Massive proteinuria > 3.5 gms / day	Hematuria and RBC Casts
Hypoalbuminemia	Low grade proteinuria
Hyperlipidemia	Hypertension
Non inflammatory process	Inflammatory process
Immune complex deposition	Retraction of epithelial foot process (podocyte)
Lipiduria	Oliguria
Oedema (peripherally)	Oedema

Reference

- Harsh mohan. Text book of Pathology.8th edition.2019
- A.K.Mandal, Dr. Sharmana Choudhary. Textbook of Pathology for MBBS. Vol II. Second edition 2017.

THANK YOU