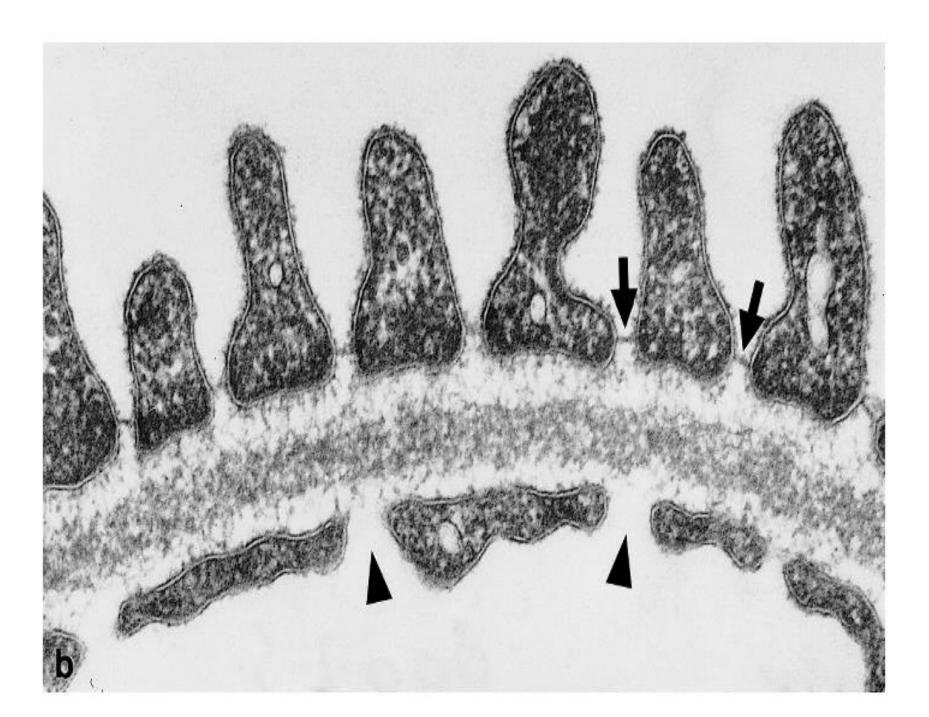
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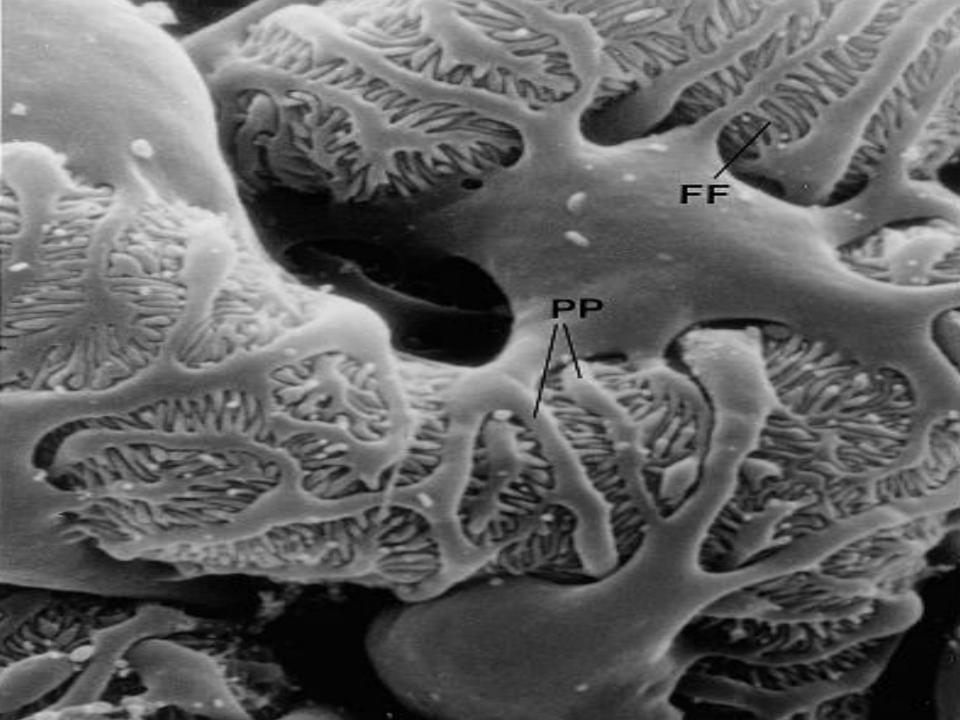
Dr Javid Safa MD

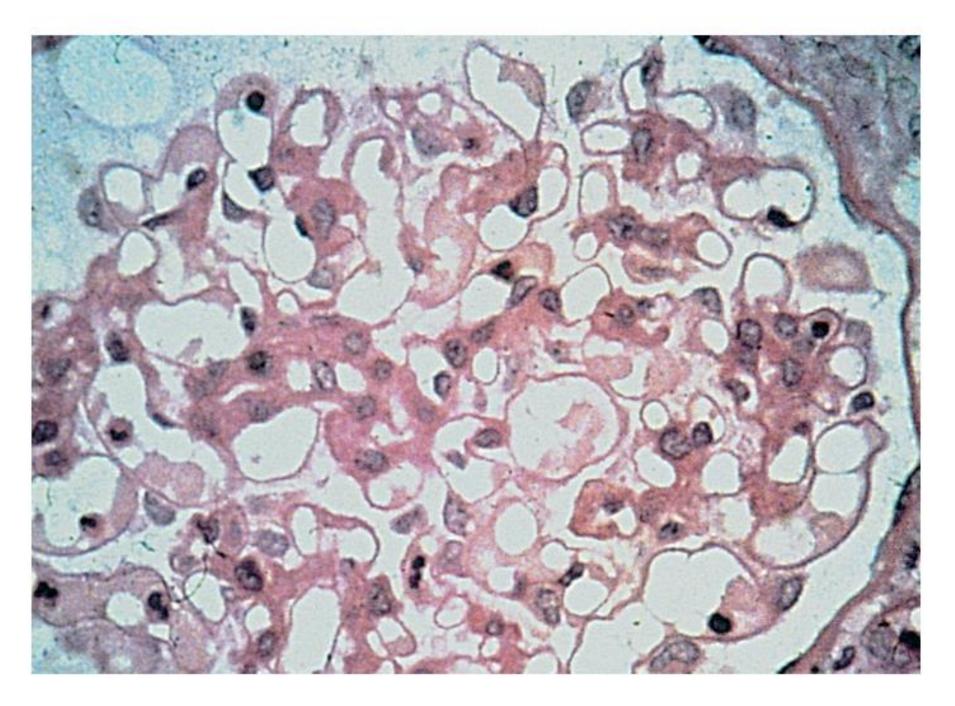
 Internist-Nephrologist
 Tabriz University of Medical Sciences

FILTRATION MEMBRANE

1-Epithelial cell with foot processes and structural proteins.
2-GBM (lamina rara externa,lamina densa,lamina rara interna).
3-Endothelial cells.







PROTEINURIA

Less than 150mg/day is in normal range

Microalbuminuria:30-300mg/day

Urine protein to urine Cr ratio > 0.1-0.15

MECHANISMS OF PROTEINURIA

1-FUNCTIONAL PROTEINURIA: Transient Absence of renal disease Fever, Emotions, Acute medical illness Orthostatic proteinuria due to hemodynamic change (2-5% of teenagers but rare in adults >30 years)

MECHANIMS OF ...

2-OVER-PRODUCTION

High plasma concentration of some proteins such as light chains, heavy chains, and other fragments of Ig.

Multiple myeloma

MECHANISMS OF

3-TUBULAR:

- Impaired tubular reabsorption of normally filtered proteins.
- Hereditary diseases (Wilson)
- Toxic injuries (Cadmium, Lead, Genetamicin)
 Metabolic (hypokalemia)
 CTIN

Fanconi syndrome
Diuretic phase of ATN
Heparan sulfate, Tom-Horsefal protein loss

MECHANISMS OF 4-GLOMERULAR due to: A-Loss of the negative charge of GBM B-An increase in effective pore size or number due to direct damage C-Disease related changes in glomerular homodynamic. D-Abnormality in the synthesis, composition, and negative charge density of GBM.

PROTEINURIA IS GLOMERULAR IF:

A-amount >2 g/day

B-protein is >70% albumin.

GLOMERULAR SYNDROMES

1-Asymptomatic proteinuria 2-Asymptomatic hematuria 3-Recurrent gross hematuria 4-Acute nephritis 5-Nephrotic syndrome 6-Nephritic syndrome

 7-Pulmonary renal syndrome (vasculitic syndromes)
 8-RPGN
 9-CRF(chronic sclerosing GN)

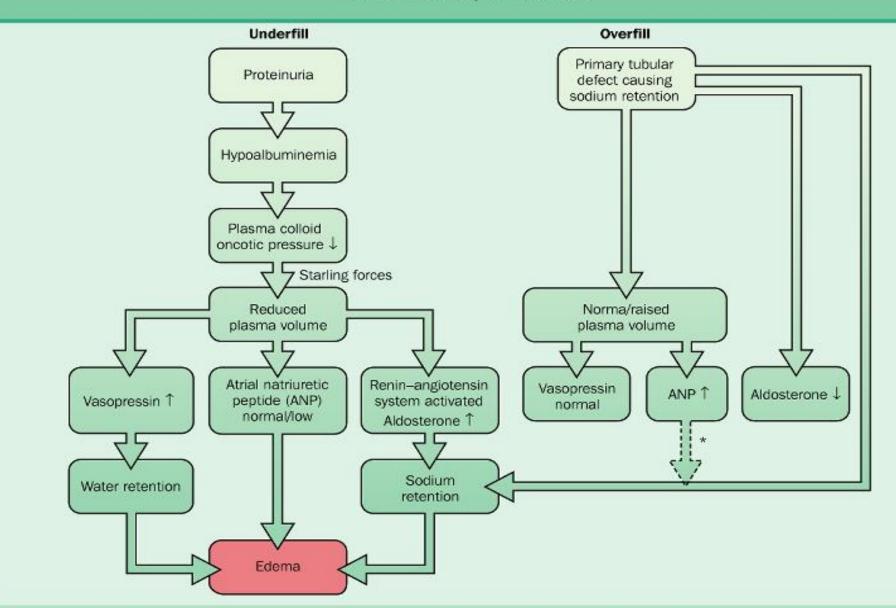
NEPHROTIC SYNDROME

DEFINITION: Urinary protein loss>3.5 g/day/1.73m2 of body surface

ETHIOLOGY

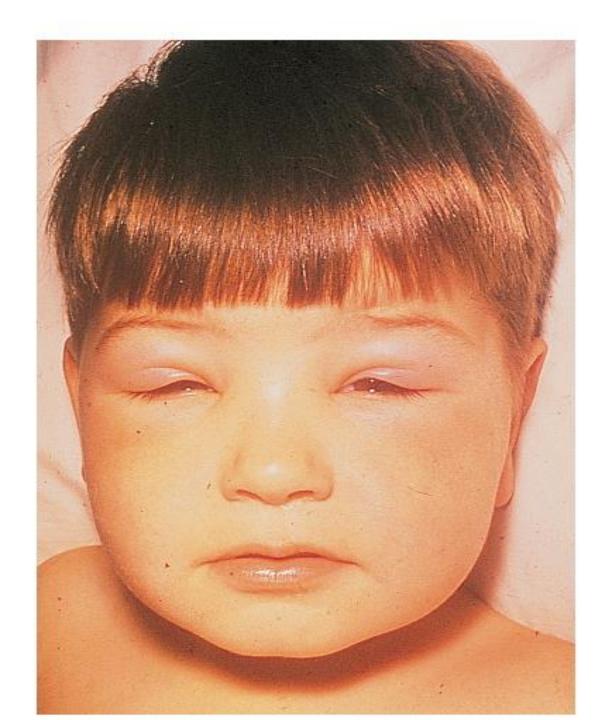
1-Infectious (bacterial, viral, parasitic) 2-Metabolic 3-Collagen vascular 4-Toxins and drugs 5-Malignancies 6-Hereditary and familial 7-Idiopathic

CLINICAL COMPLICATION OF NEPHROTIC SYNDROME ■ 1-HYPOALBUMINEMIA: a-Edema B-Increased hepatic lipoprotein synthesis so hyperlipoproteinemia. Increased platelets aggregation



Formation of nephrotic edema

*The kidney is relatively resistant to ANP in this setting, so it has little effect in countering sodium retention



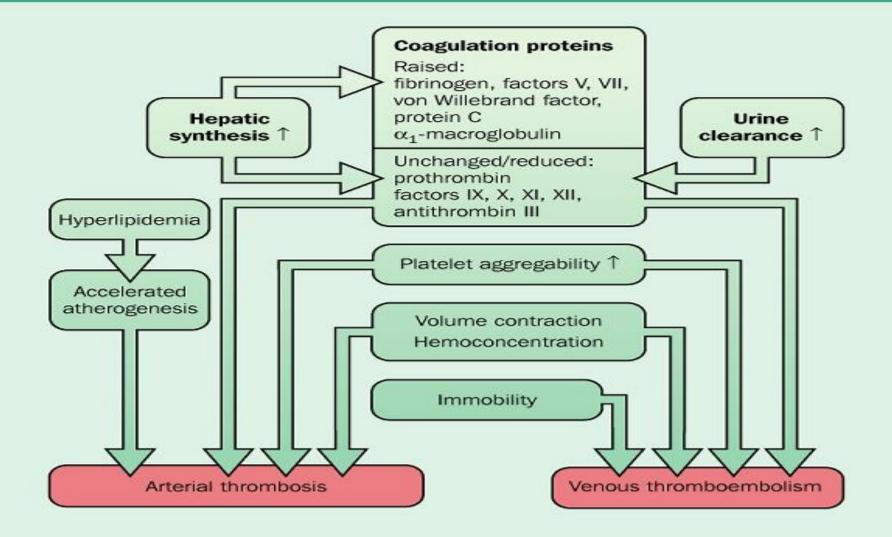


COMPLICATIONS OF....

- 2-INCREASED TUBULAR PROTEIN REABSORPTION:
- a-Tubular dysfunction
- b-Tubular damage
- 3-LOSS OF PROTEINS CARRYING VITAMINS, HORMONS, AND MINERALS.
 4-LOSS OF Ig
- a-Reduced cellular immunityB-Susceptibility to infection

- 5-ALTERATION IN COAGULATION FACTORS
- DVT,PTE, RVT
- 6-NEGATIVE NITROGEN BALANCE→ Malnutrition
- 7-ALTRATION IN DRUGS METABOLISM (binding proteins)
- 8-DIURETIC RESISTANCE

Coagulation abnormalities in nephrotic syndrome



PATHOLOGIC TYPES		
	nephrotic	nephritic
MCD	++++	
MGN	++++	+
Diabetic GN	++++	+
FSGS	+++	++
MPGN	++	+++
Acute GN	+	++++
RPGN	+	++++

TREATEMENT

 1-Treatement of underlying disease
 2-Specific treatment (corticosteroids, cyclosporine, MMF, plasmapheresis, cytotoxics,....)

3-Nonspecific treatment a-Dietary protein restriction (reducing both GFR & Pgc) (0.6g/kg+urinary Pr loss) b-ACE-I & ARB c-NSAIDS in high dose d-Statins e-Soy protein f-Anticoagulants

THE END

