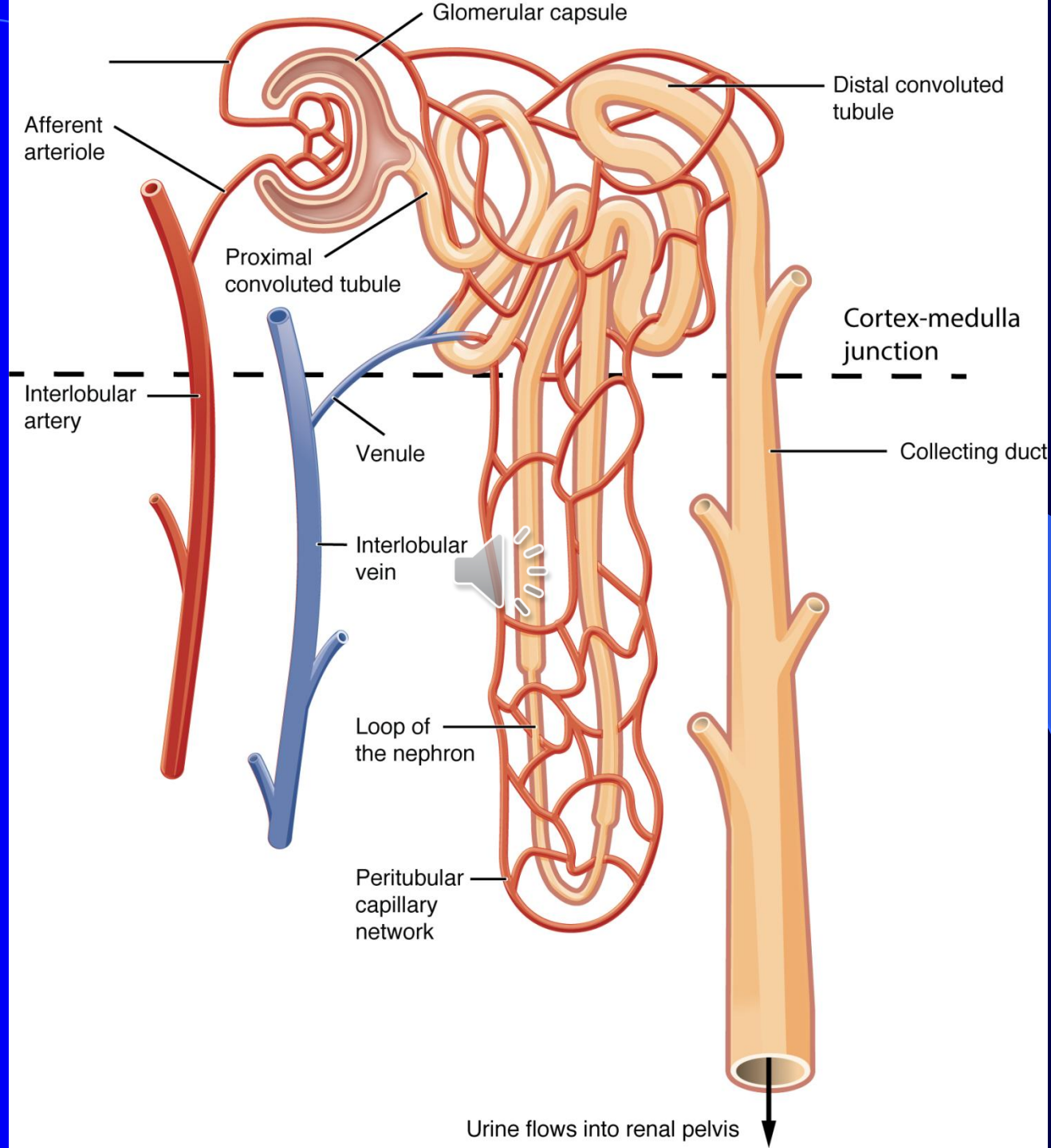
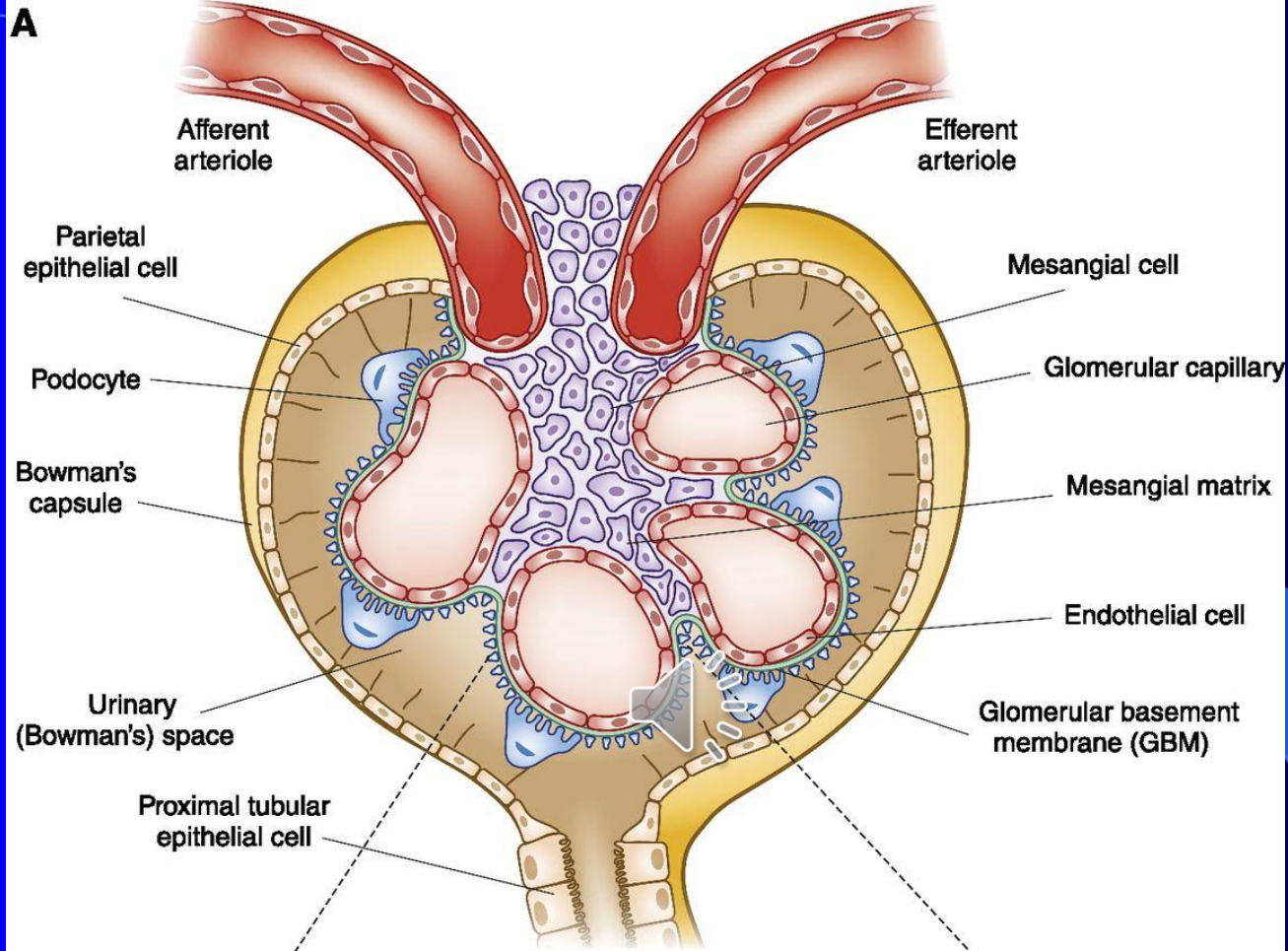
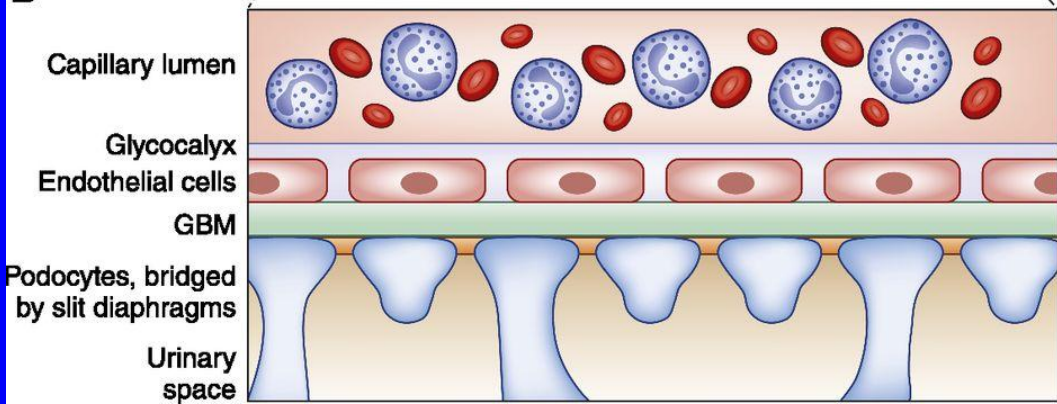


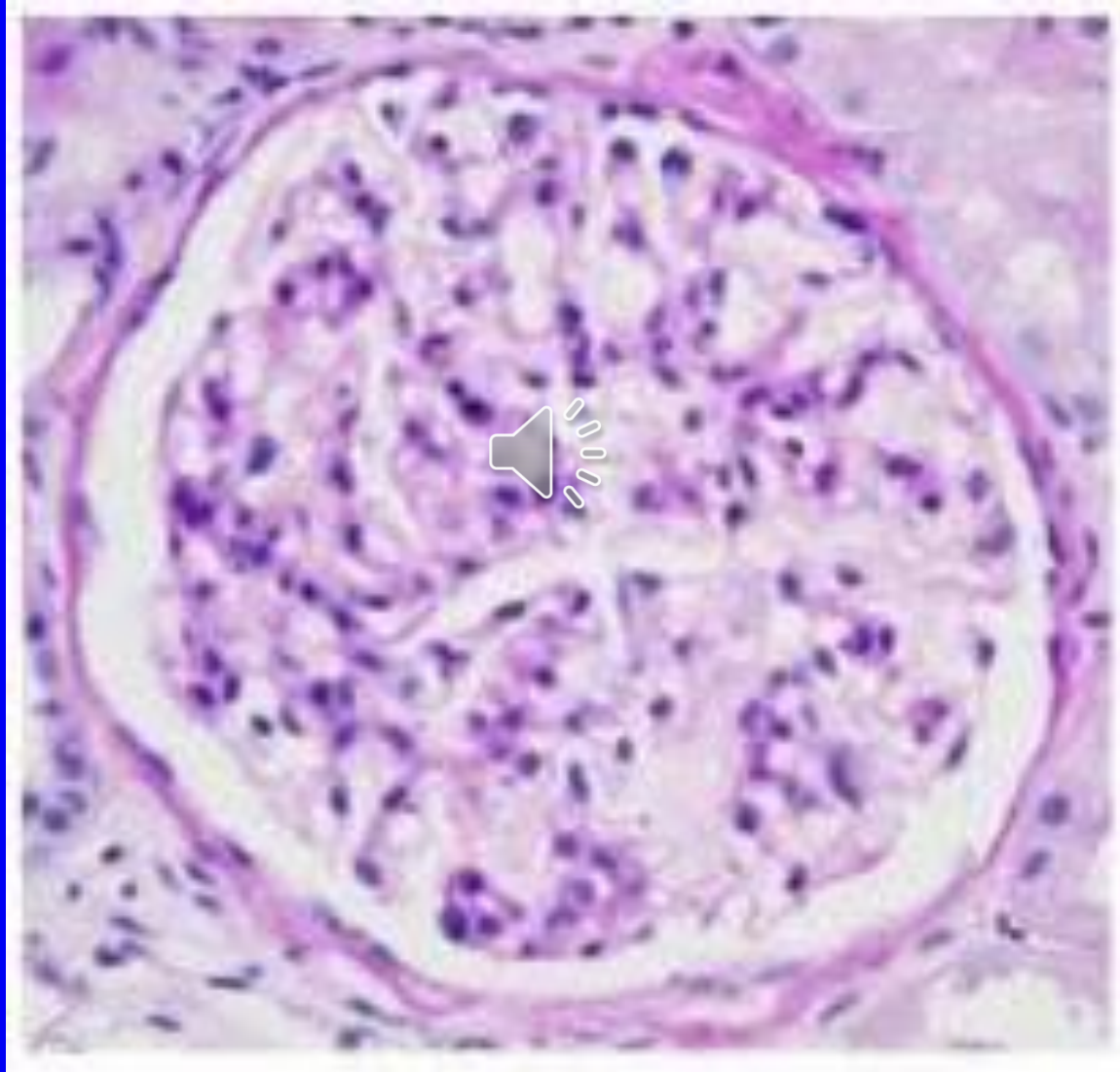
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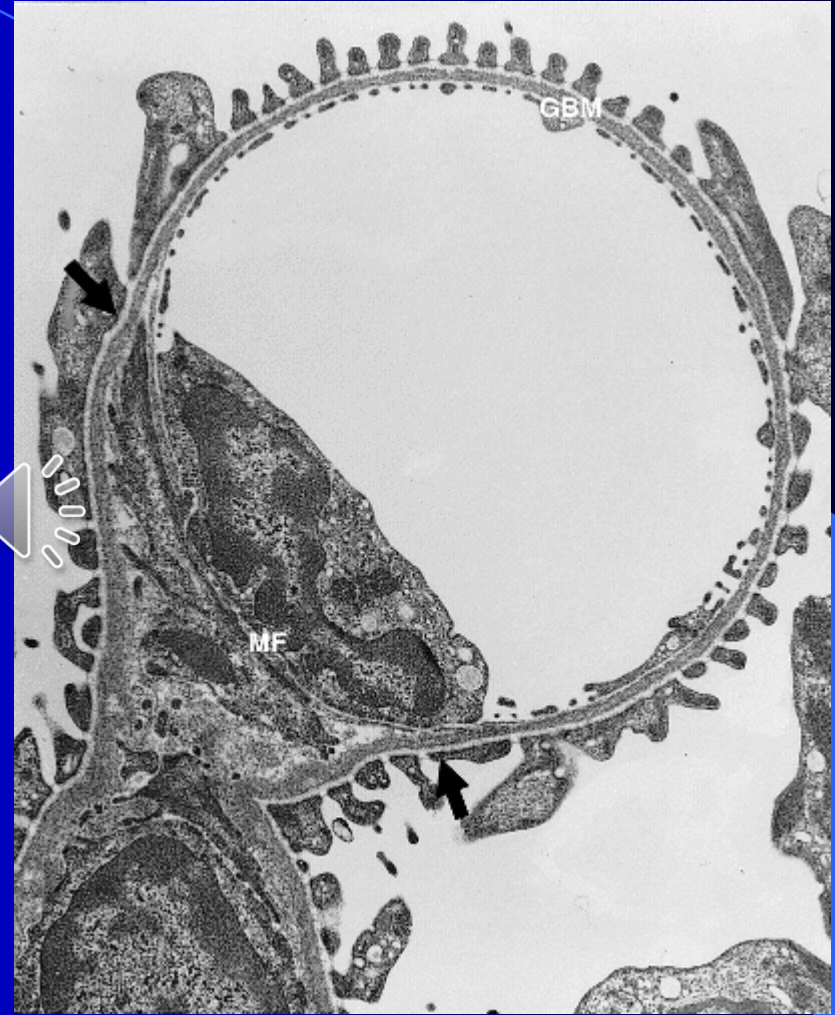
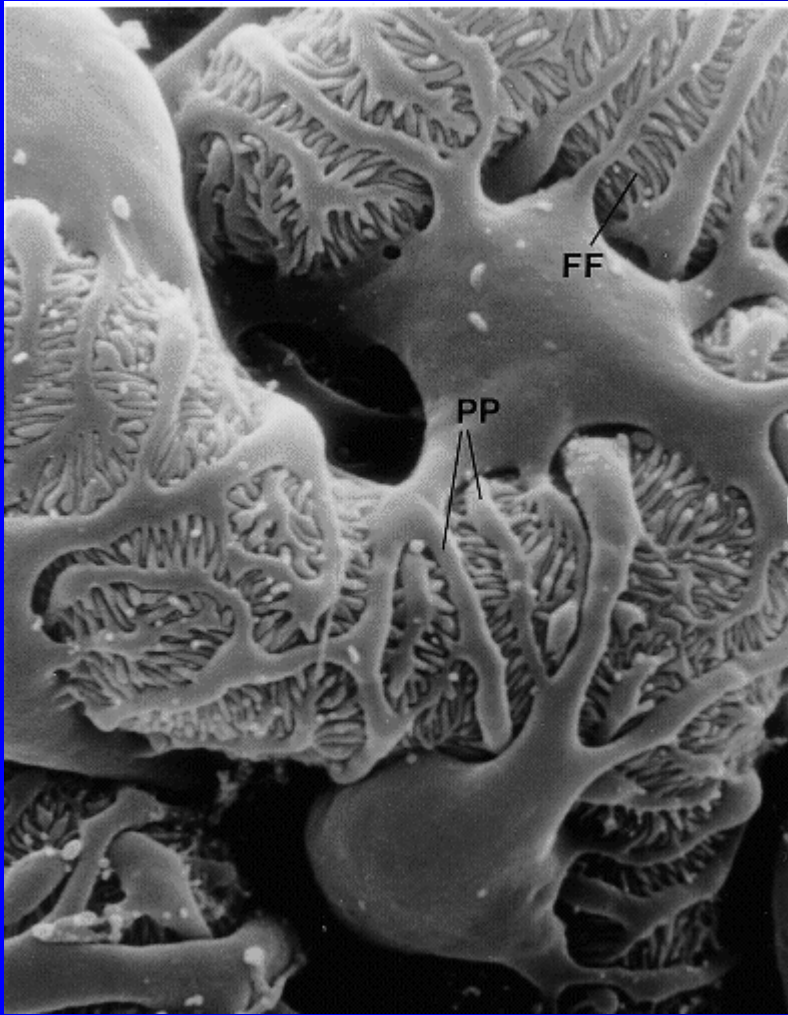
Δημήτριος Β. Βλαχάκος
Καθηγητής Παθολογίας-Νεφρολογίας
Υπεύθυνος Νεφρολογικής Μονάδας
Β΄ Προπαιδευτική Παθολογική Κλινική
Πανεπιστημιακό Γενικό Νοσοκομείο «ΑΤΤΙΚΟΝ»

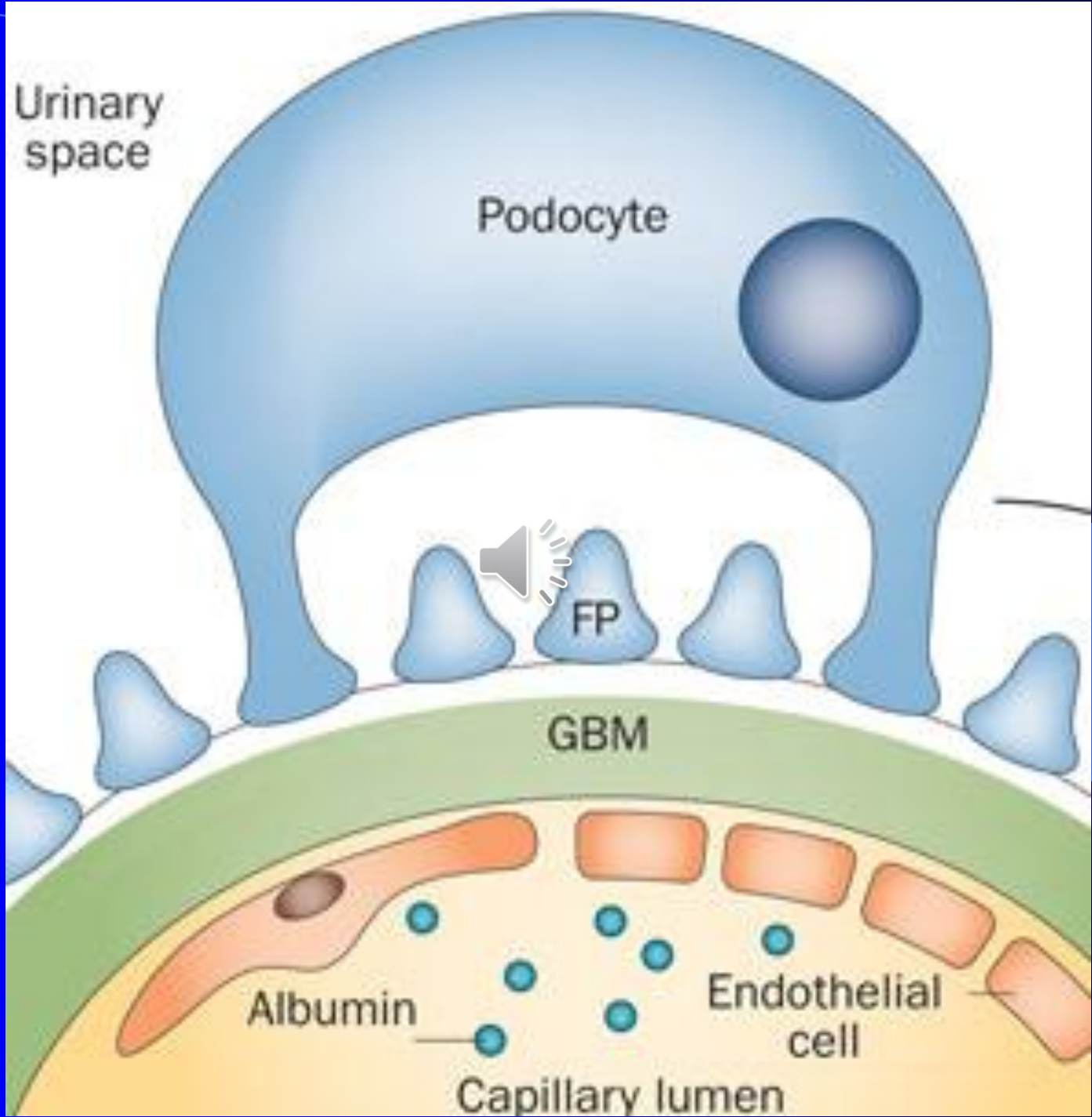


A**B**

Φυσιολογικό σπείραμα

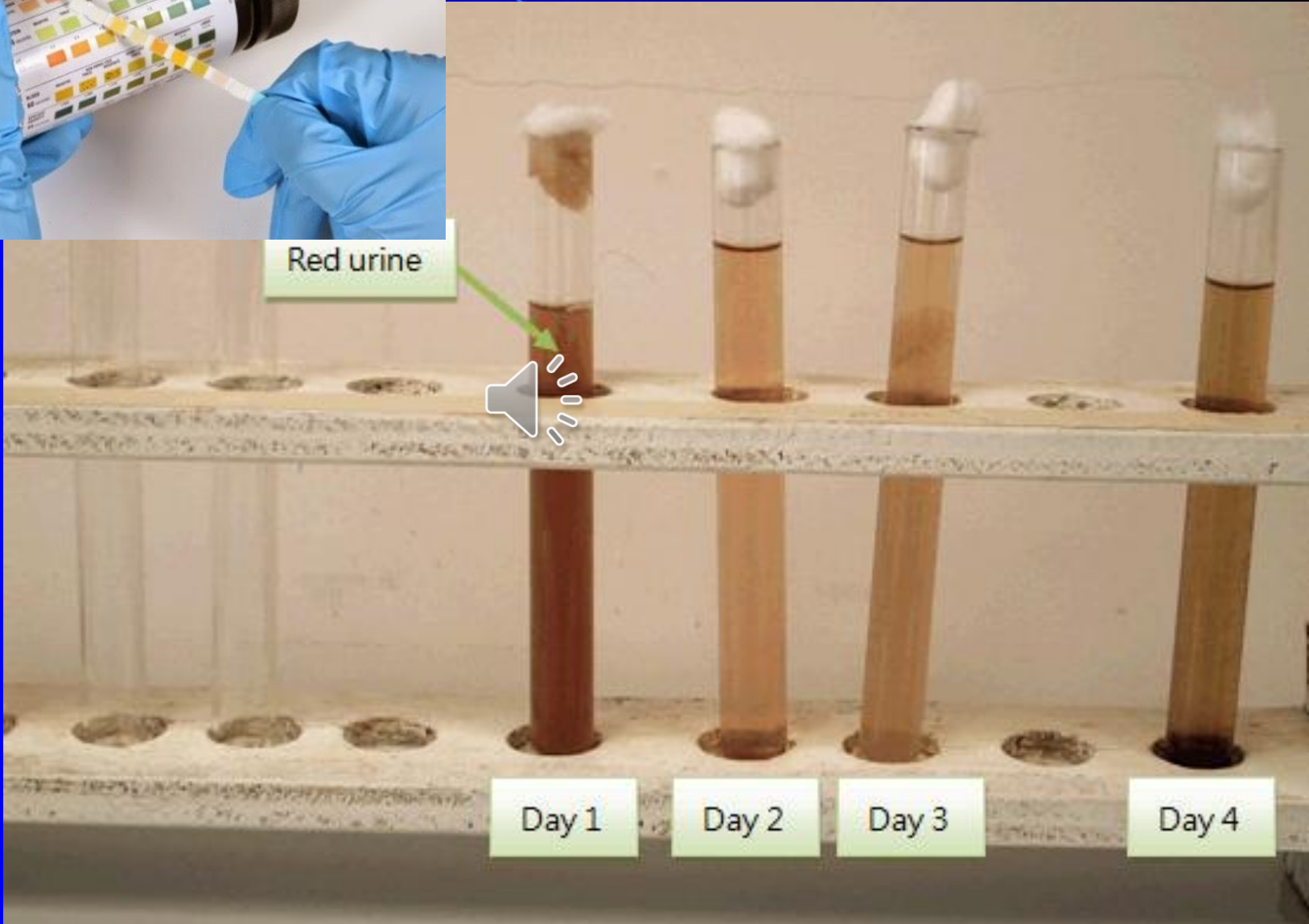








Red urine

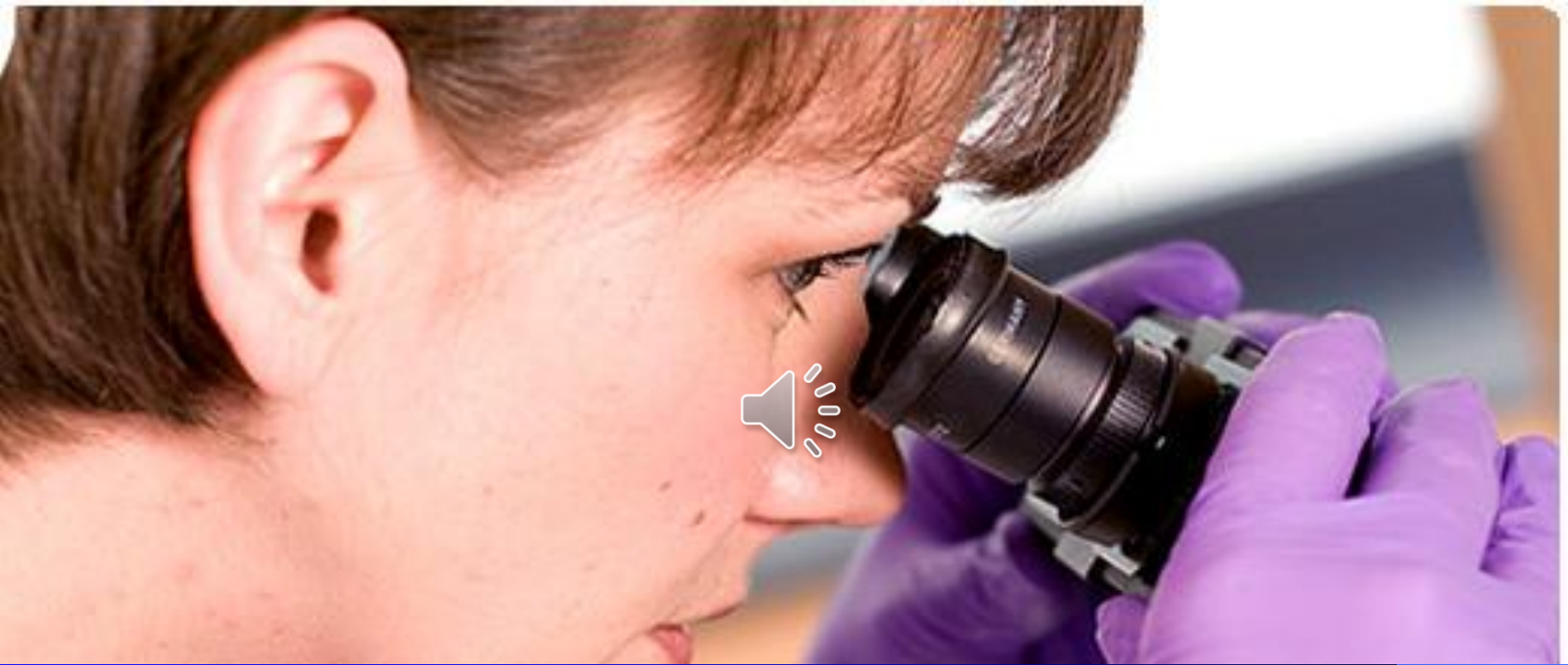


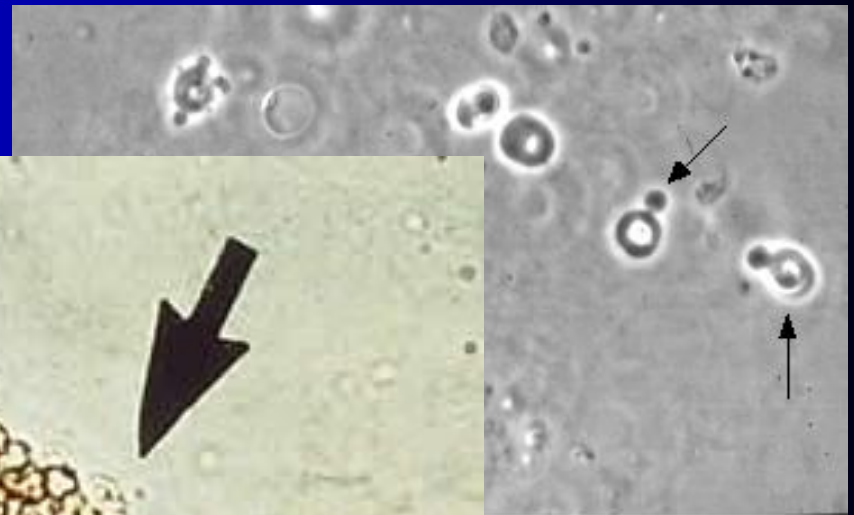
Day 1

Day 2

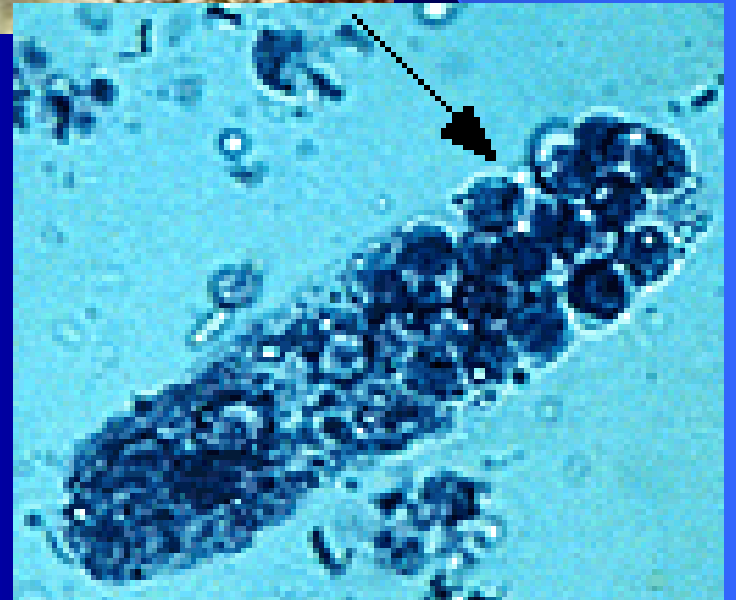
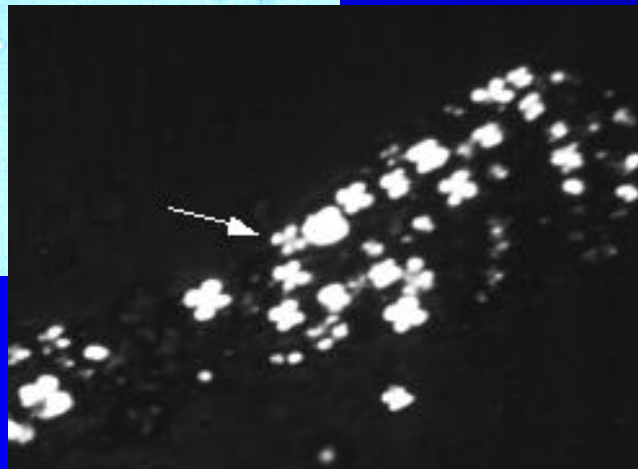
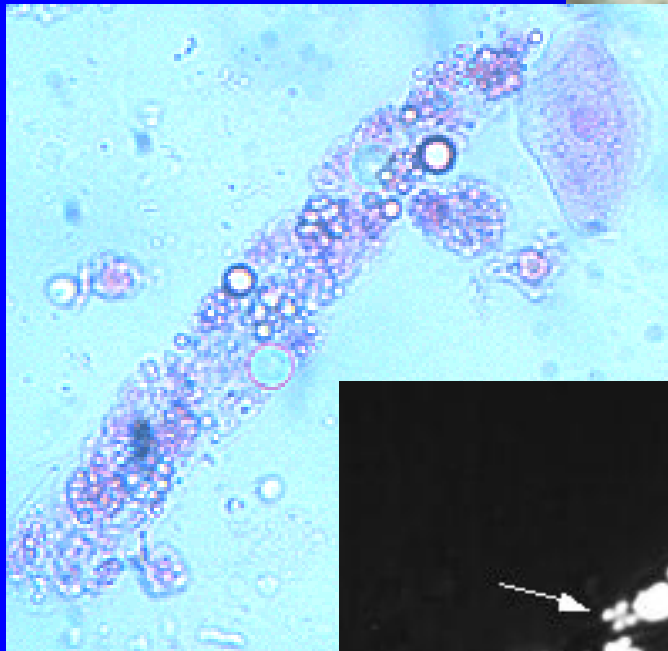
Day 3

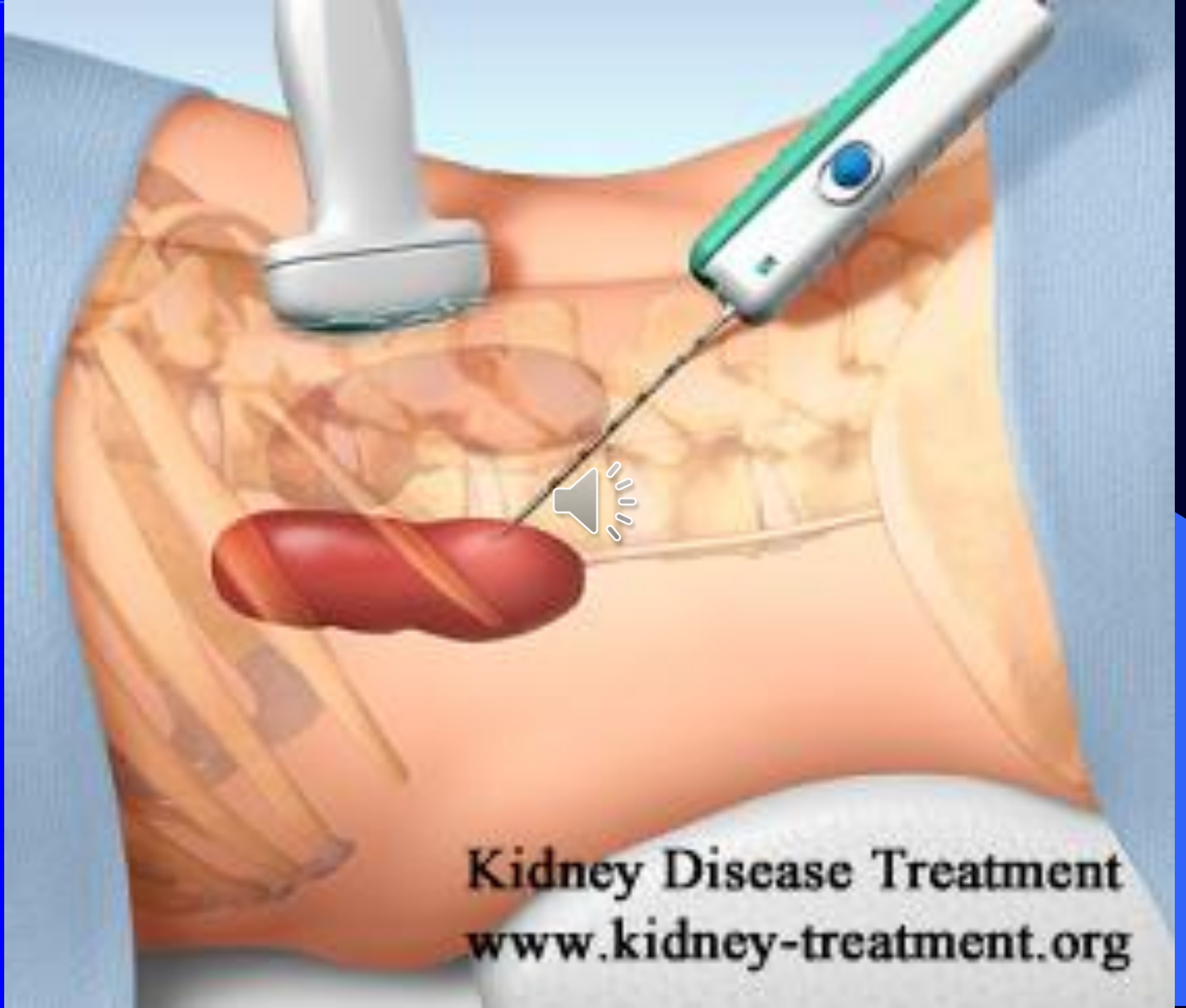
Day 4





contrast microscopy





Kidney Disease Treatment
www.kidney-treatment.org

MNEMONIC “ANNURIC”)

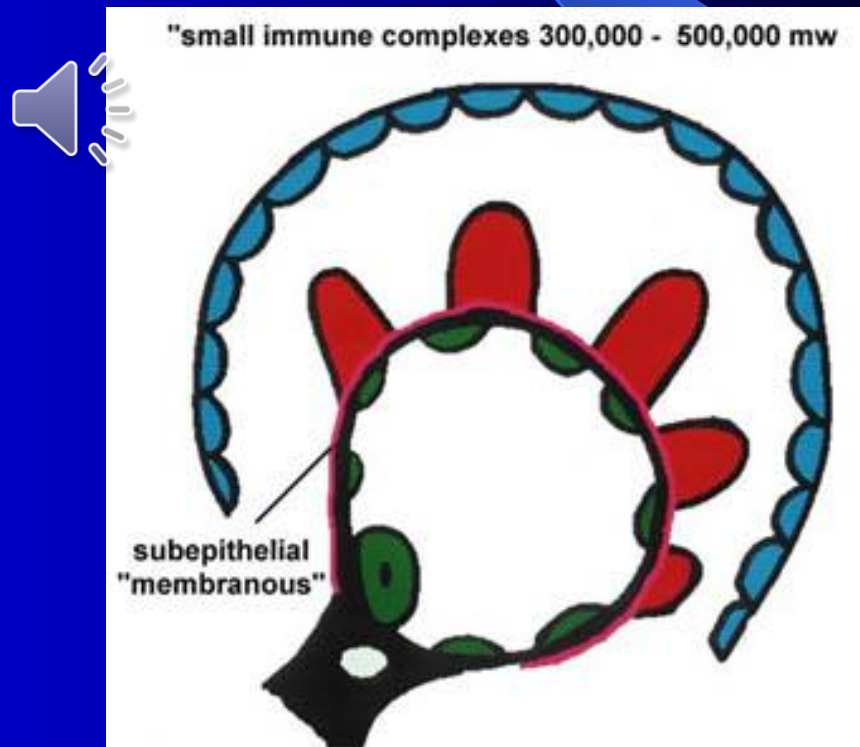
- A Asymptomatic hematuria/proteinuria
- N Nephrotic syndrome
- N Nephritic syndrome
- U Urolithiasis
- R Rapidly progressive glomerulonephritis
- I Interstitial and tubular diseases
- C Chronic renal disease

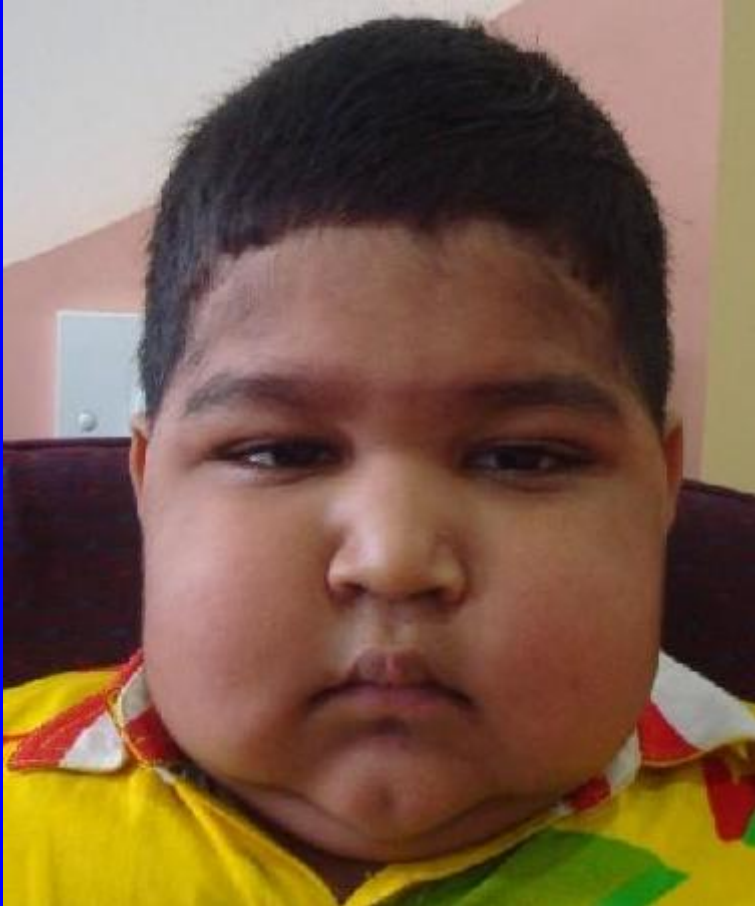
MNEMONIC “ANNURIC”)

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- N Nephrotic syndrome
- N Nephritic syndrome
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- R Rapidly progressive glomerulonephritis
- I Interstitial and tubular diseases
- C Chronic renal disease

Νεφρωσικό Σύνδρομο

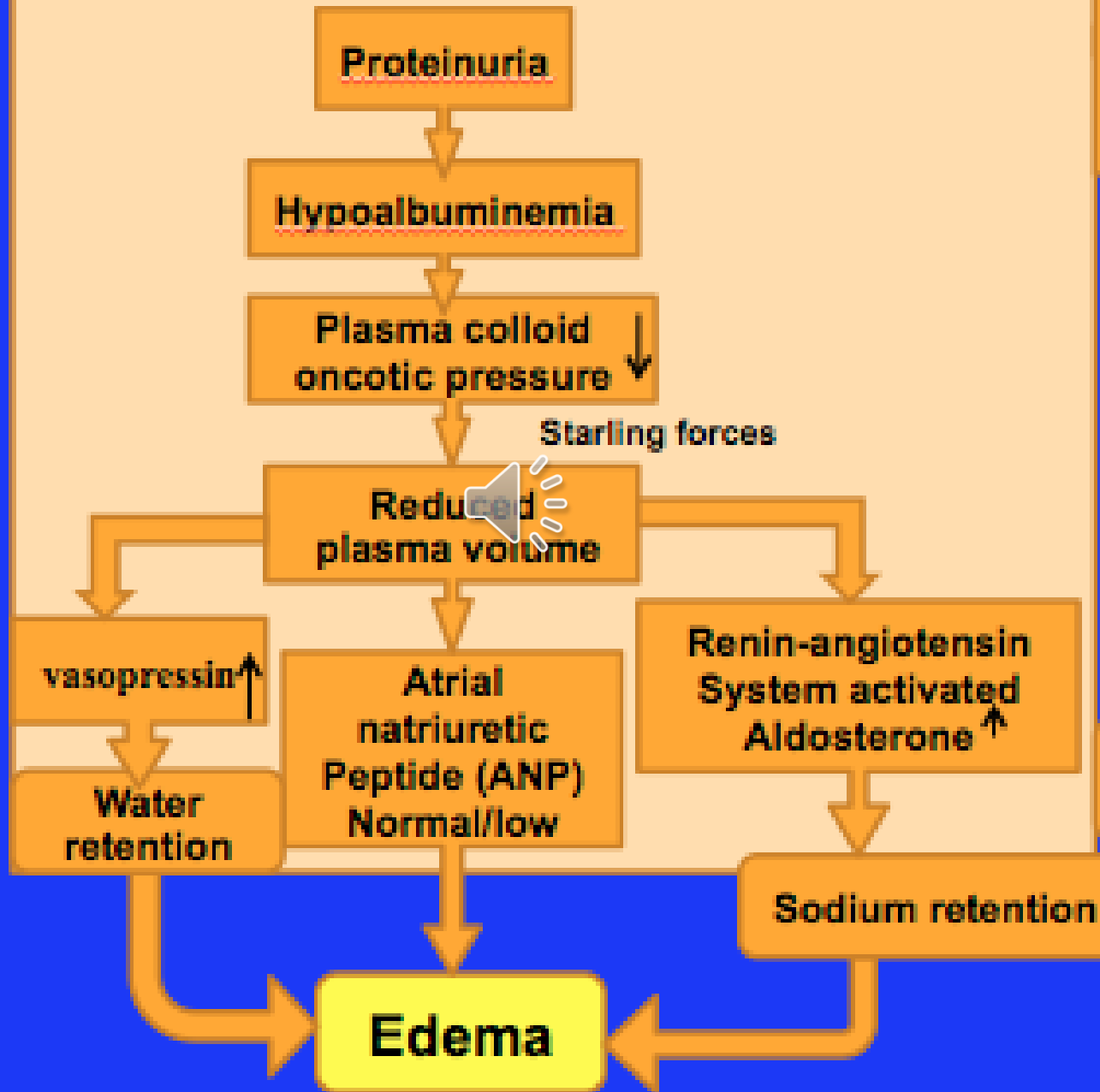
- Πρωτεϊνουρία $> 3\text{g} / 24\text{ωρο}$
- Υπολευκωματαιμία ($< 3 \text{ g/dl}$)
- Υπερλιπιδαιμία
- Λιπιδουρία
- Οίδημα





Edema (swelling) of the ankles and feet

Underfill



Nephrotic Syndrome

↑ PCSK9 & IDOL
↓ LDLR deficiency

↑ Hepatic ACAT-2

↓ Hepatic uptake of LDL

↓ Intracellular free chol
HMG-CoA activation

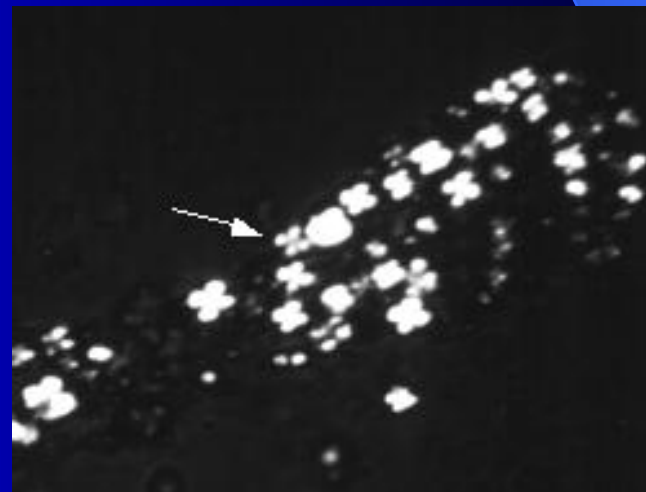
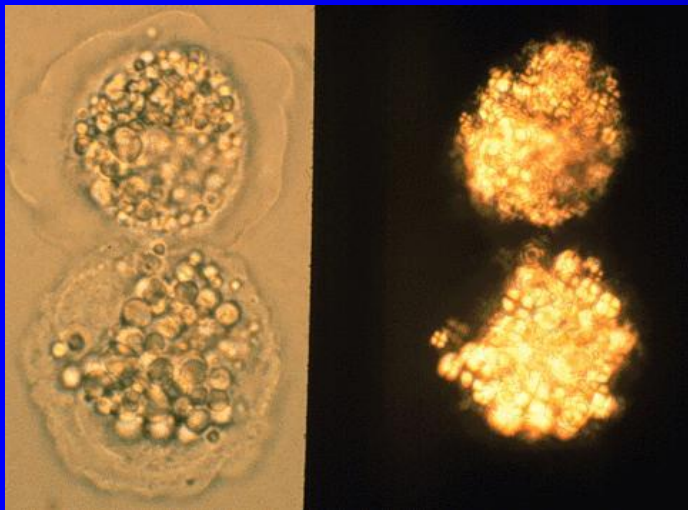
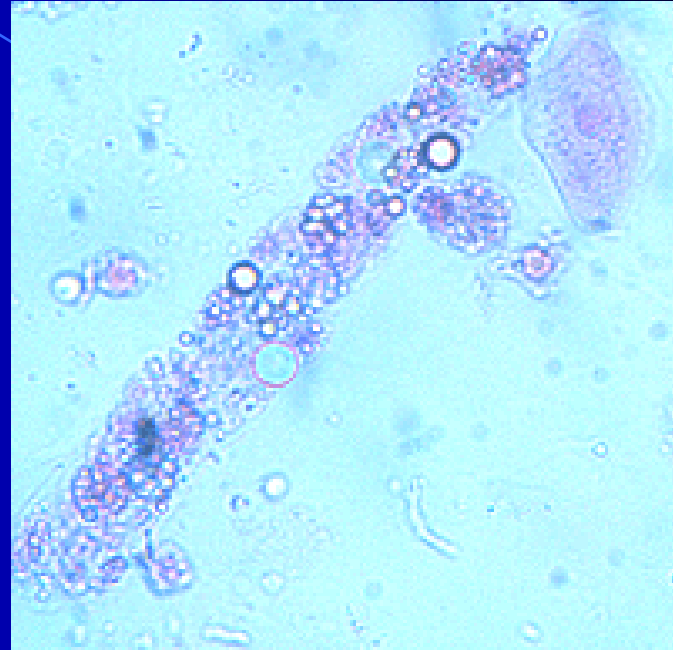
↑ Serum LDL

↑ chol production

Increased serum LDL cholesterol



ΙΖΗΜΑ ΟΥΡΩΝ ΣΕ ΝΕΦΡΩΣΙΚΟ ΣΥΝΔΡΟΜΟ



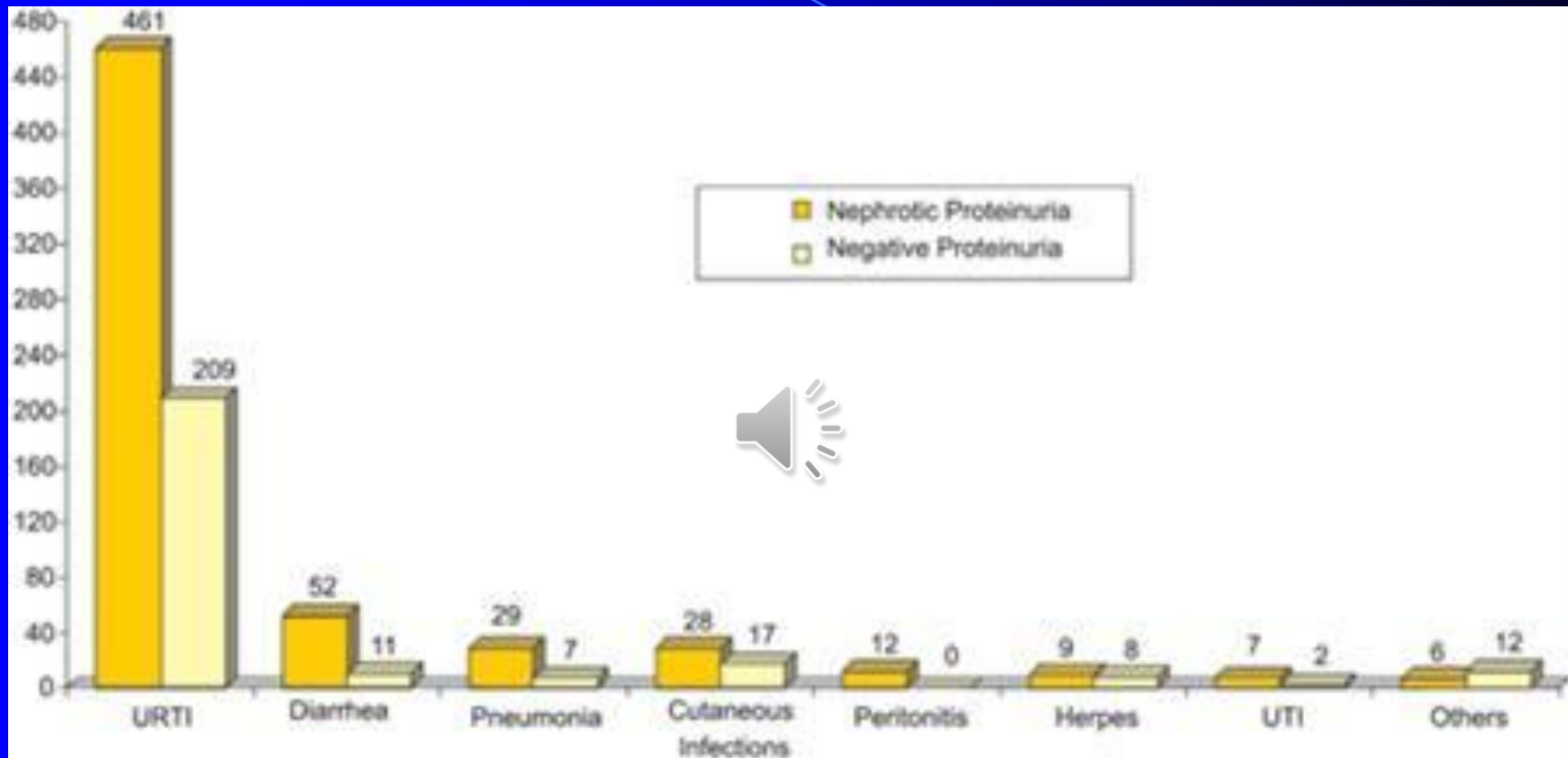


Figure 1 - Number and type of infection of 92 patients during the period with nephrotic proteinuria (604 infections/1140 months) and of the 89 patients during the period with negative proteinuria (266 infections /6822 months).

Hypercoagulation in NS

Low zymogen factors; factors IX, factor XI

Increased procoagulatory cofactors. factor V, factor VIII

Increased fibrinogen levels

Decreased coagulation inhibitors; antithrombin III (but protein C and Protein S increased)

Altered fibrinolytic system (α₂-antiplasmin increased, plasminogen decreased)

Increased platelet reactivity

Thrombocytosis

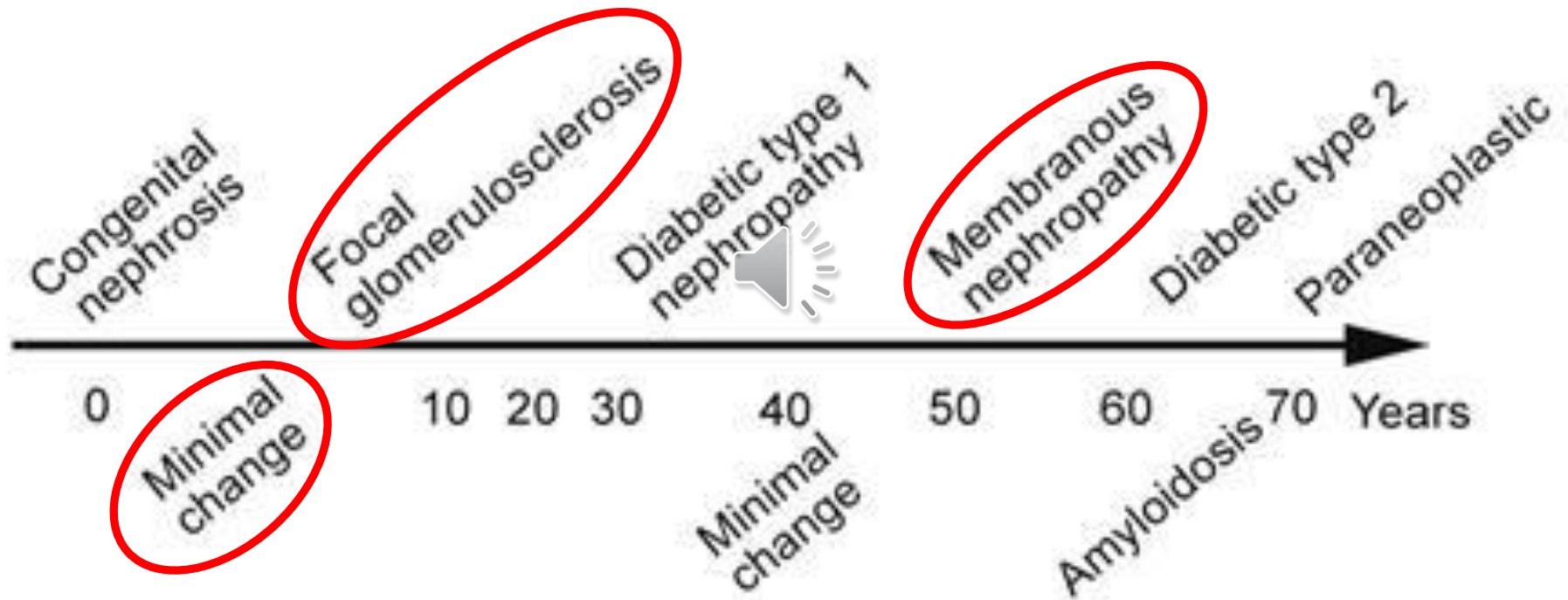
Increased release reaction *in vitro* (adenosine diphosphate; thrombin, collagen, arachidonic acid, epinephrine)

Increased factor IV and b-thromboglobulin *in vivo*

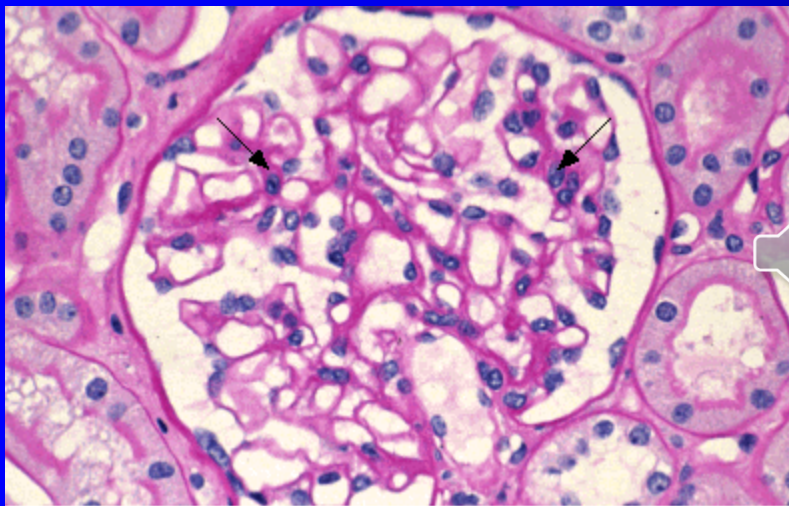
Altered endothelial-cell function

average ages of types of nephrotic syndrome

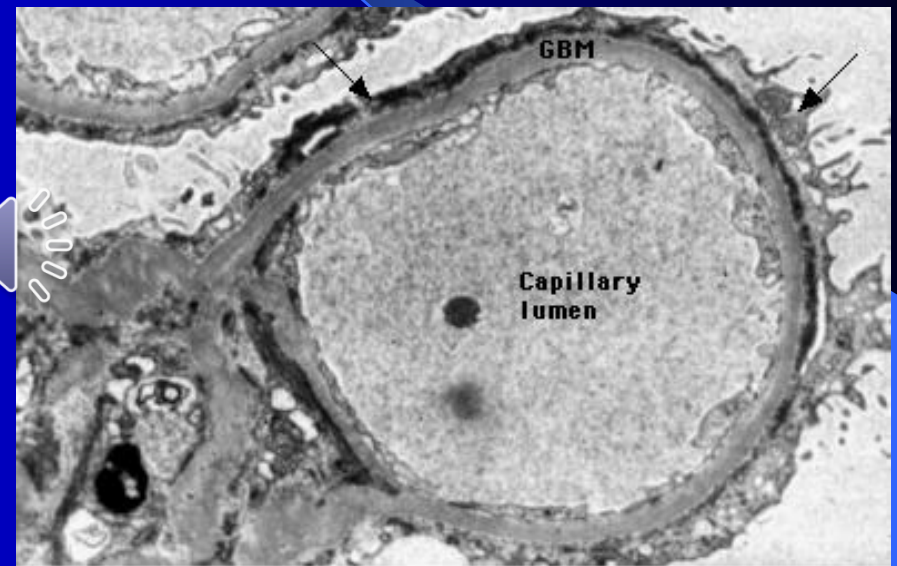
timeline not to scale



Νόσος Ελαχίστων Αλλοιώσεων



Minimal change disease Light micrograph of an essentially normal glomerulus in minimal change disease. There are only 1 or 2 cells per capillary tuft, the capillary lumens are open, the thickness of the glomerular capillary walls is normal, and there is neither expansion nor hypercellularity in the mesangial areas in the central or stalk regions of the tuft (arrows). Courtesy of Helmut G Rennke.



Minimal change disease Electron micrograph in minimal change disease showing a normal glomerular basement membrane (GBM), no immune deposits, and the characteristic widespread fusion of the epithelial cell foot processes (arrows). Courtesy of Helmut Rennke, MD.

MINIMAL CHANGE GLOMERULOPATHY

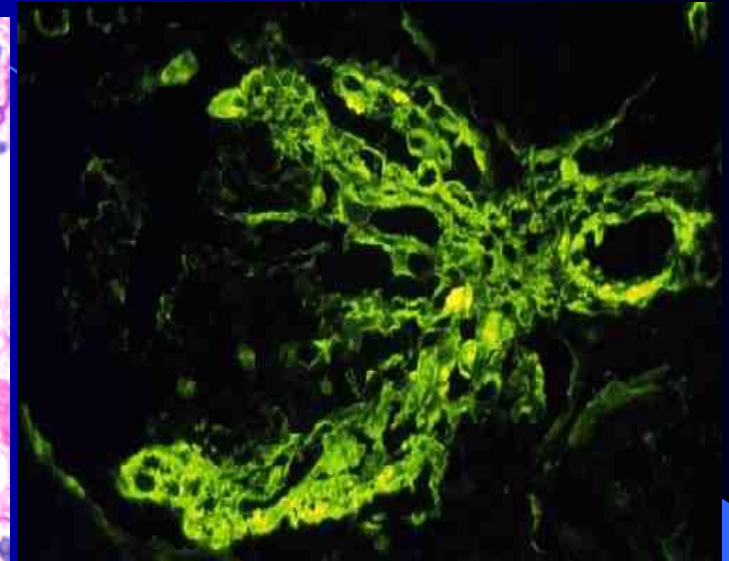
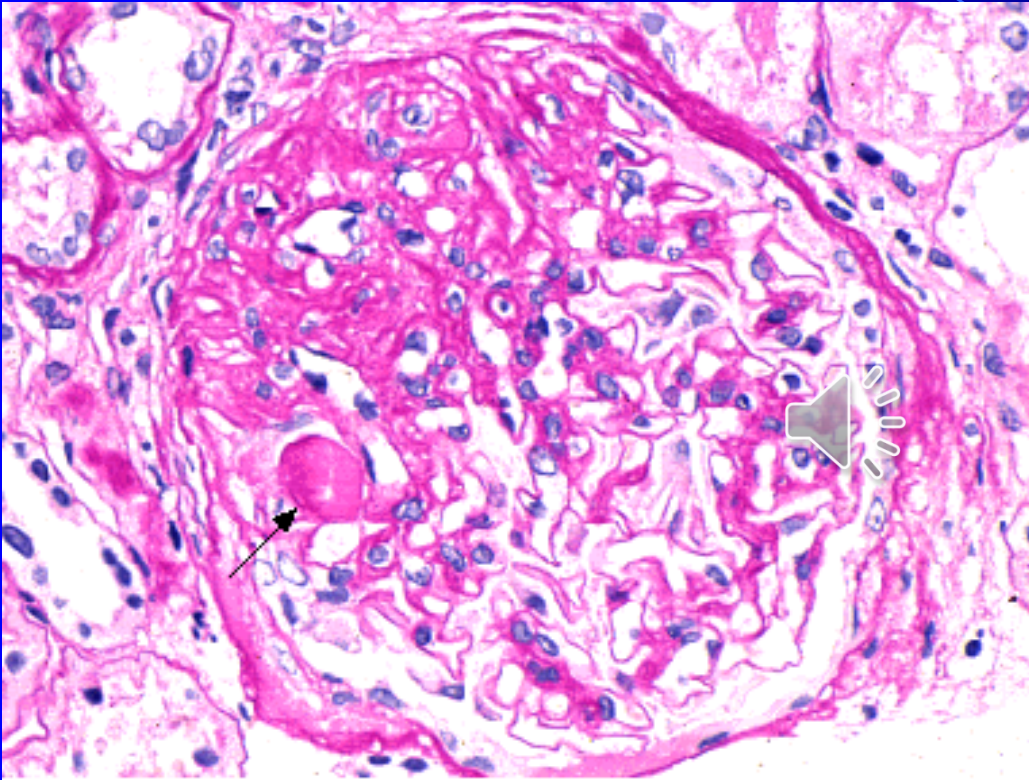
CL: Most common cause of nephrotic syndrome in children

E/P: Remission can be induced by measles, occurs more frequently in Hodgkin lymphoma, cured by glucocorticoids, cyclophosphamide or rituximab, the permeability factor seems to be **IL-13**.

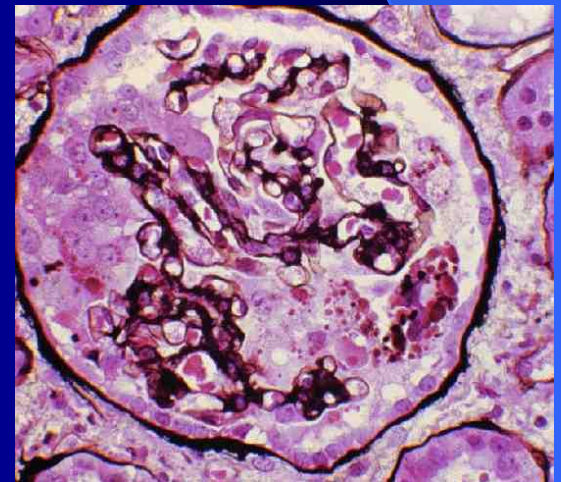
Path: normal by LM and IF

EM: fusion of foot processes

ΕΣΤΙΑΚΗ ΣΠΕΙΡΑΜΑΤΟΣΚΛΗΡΥΝΣΗ



Moderate FGS Light micrograph in focal segmental glomerulosclerosis shows a moderately large segmental area of sclerosis with capillary collapse on the upper left side of the glomerular tuft; the lower right segment is relatively normal. Focal deposition of hyaline material (arrow) is also seen. Courtesy of Helmut Rennke, MD.



FOCAL SEGMENTAL GLOMERULOSCLEROSIS

Def: 15% of all nephrotic syndromes; heterogenous group of diseases (primary vs secondary)

E/P: Increased circulating levels of soluble urokinase receptor (**suPAR**). In HIV, IV drug abuse, CHD, obesity, sickle-cell disease



Path: focal and segmental glomerular hyalinosis

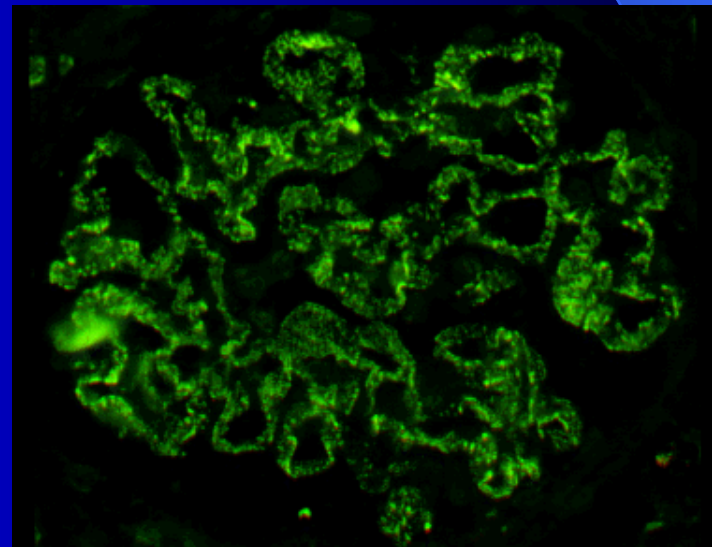
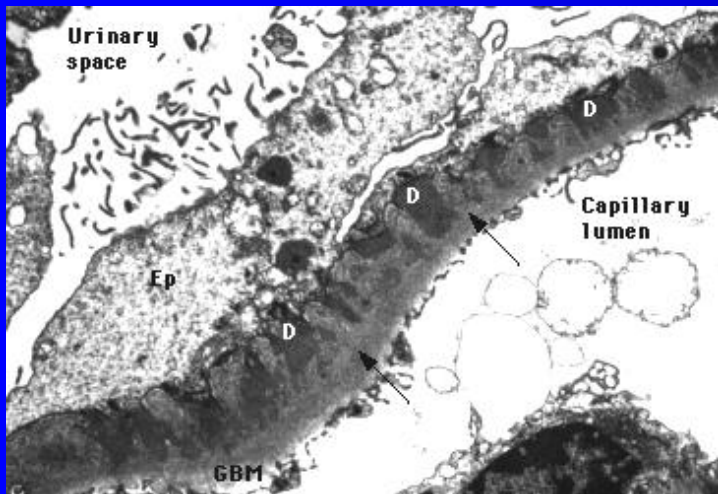
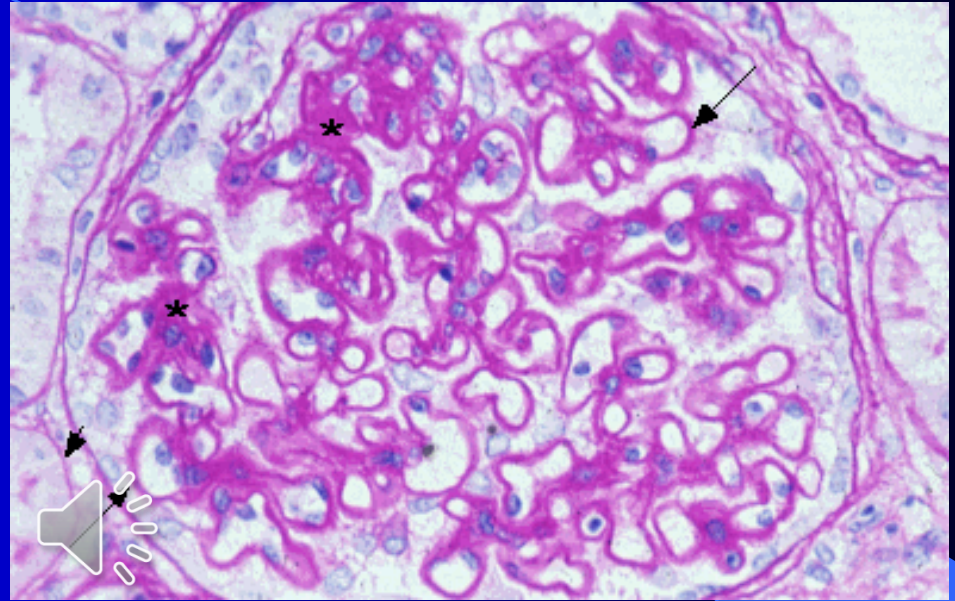
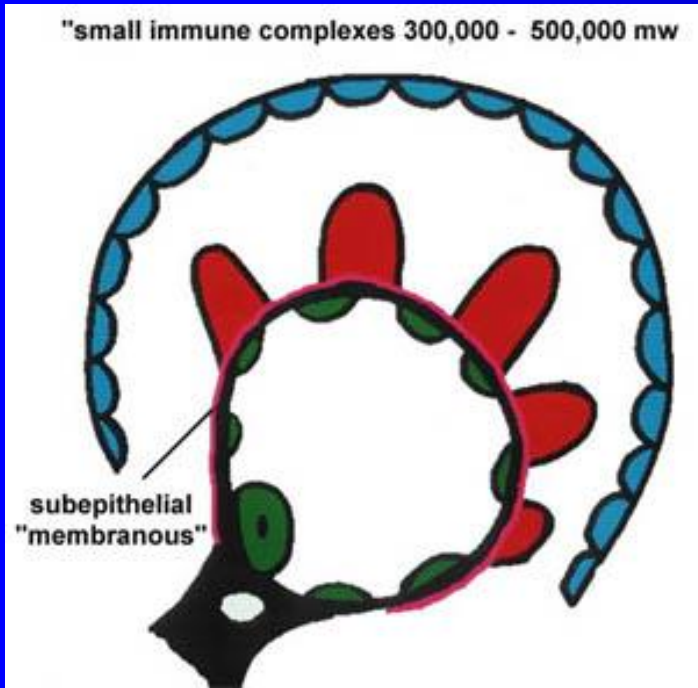
“Collapsing” pattern (e.g. HIV)

Trapping of serum proteins (IF and EM)

Clin: Nephrotic syndrome → ESRD (5-20y); In HIV related FSGS → ESRD (1 year)

Therapy: Glycocorticosteroids +/- calcineurin inhibitors

ΜΕΜΒΡΑΝΩΔΗΣ ΣΠΕΙΡΑΜΑΤΟΠΑΘΕΙΑ



MEMBRANOUS NEPHROPATHY

Def: Most common cause of nephrotic syndrome in adults (40%)

M-Type **Phospholipase A₂ Receptor** as Target Antigen in Idiopathic Membranous Nephropathy -
> Immune complex → BM thickening

E/P: Primary



Secondary (SLE, HBV, drugs, cancer)

Path: Subepithelial deposits of immune complex

CL: Nephrotic syndrome

(25% recover, 50% persist, 25% progress)

Therapy: Glycocorticoids and cytotoxic therapy

Αίτια Μεμβρανώδους Σπειραματοπάθειας

Major Causes of Membranous Nephropathy

Idiopathic, may represent autoantibody against
glomerular epithelial cell antigens

Malignancy, primarily solid tumors

Systemic lupus erythematosus

Rheumatoid arthritis

Drugs:

Penicillamine

Gold

Tiopronin

Hepatitis B virus

Syphilis – congenital and secondary

Chronic renal transplant rejection

Hepatitis C virus

Hepatosplenic schistosomiasis

Other glomerular diseases

Rare:

Sarcoidosis

Captopril

Formaldehyde



ΕΥΧΑΡΙΣΤΩ ΓΙΑ ΤΗΝ
ΠΡΟΣΟΧΗ ΣΑΣ

