GENITOURINARY SYSTEM

SUBJECT : Pathology

LEC NO. : Two

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وَقُل رَّبِّ زِدْنِي عِلْمًا

GENITOURINARY SYSTEM

Objectives

- Clinical manifestation of kidney disease
- understand the terminology of Renal diseases
- Discussion of Glomerular disease
- Nephrotic syndrome
- Nephritic syndrome
- Disease of blood vessels
- Urinary tract infection
- Analgesic nephropathy
- Acute Tubular Necrosis
- Hemolytic Uremic Syndrome
- Urolithiasis and hydronephrosis
- Renal Tumours RCC
- Bladder Tumours

موضوع محاضرة اليوم

يعطيكم العافية وريد ❤️ قبل ما نبدأ المحاضرة حابة اشرح الكم كم شي ❤️ نتوفة فسيو مع باثوفسيو، عشان لما ننتقل للسلايدات نكون فاهمين كل حرف ان شاء الله ❤️
تذكير اي شي بالاحمر فهو شرح خارجي، اي شي بالاخضر فهو ملاحظات الدكتورة❤️

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ال filtration barrier بتكون من ثلاث أقسام

Capillary endothelial

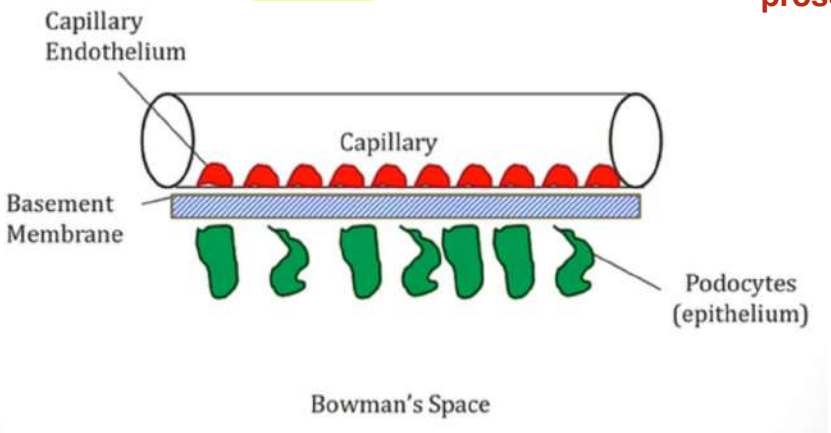
-fenestrated, has small openings
-repels RBCs, WBCs, Platelets.

Capillary basement membrane

-negatively charged
-contain type 4 collagen and heparan sulfate
-repels negative molecules like albumin.

Podocyte = epithelial cells

-called epithelial cells
-has foot processes
-wrap capillaries
-slits between foot prosesses filter blood



**Albumin
-Small (~3.6nm)
-Can fit through all size barriers
-Negatively charged
-Repelled by GBM charge barrier.

*فلو صار عندي مشكلة بال basement membrane حنلاحظ دخول الalbumin و البروتينات الى ال nephron و بالتالي بلاقيه بالبول.

*و لو صار في مشكلة بالendothelial حنلاحظ وجود cells مثل RBC,WBC,Platelets.

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Glomerular Diseases

*Breakdown of components of filtration barrier

*Things in urine that shouldn't be there like RBCs and Protein (especially albumin)

Hematuria

*Many, many causes

*Gross: abnormal color to urine from blood ->
ممكن اشوف بالعين المجردة اختلاف بلون البول

*Microscopic: Incidental finding on urinalysis ->
او ممكن ما يكون في تغير بالبول بس اثناء الفحص لاحظت وجود RBCs

*Can occur after exercise

*Common causes: UTI and Kidney stones

*Feared cause: bladder cancer

*Glomerular disease is rare cause

Glomerular Bleeding

*Red cell casts

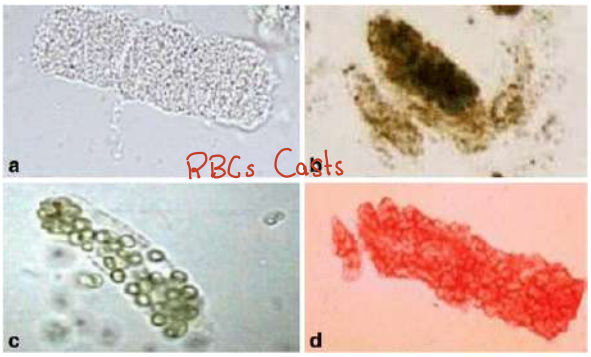
*Dysmorphic red blood cells

*Acanthocytes

*Proteinuria

*Red, smoky brown or "coca cola"

*Clots generally not seen → We usually see clots in kidney stones and bladder tumors



Proteinuria

*Color change indicates amount of protein

*Primarily detects albumin (good for glomerular disease!) -> 1+, 2+, 3+, 4+

هدول عبارة عن درجات لكمية ال albumin و غالبا ال Glomerular disease بكون +4

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Glomerular Diseases

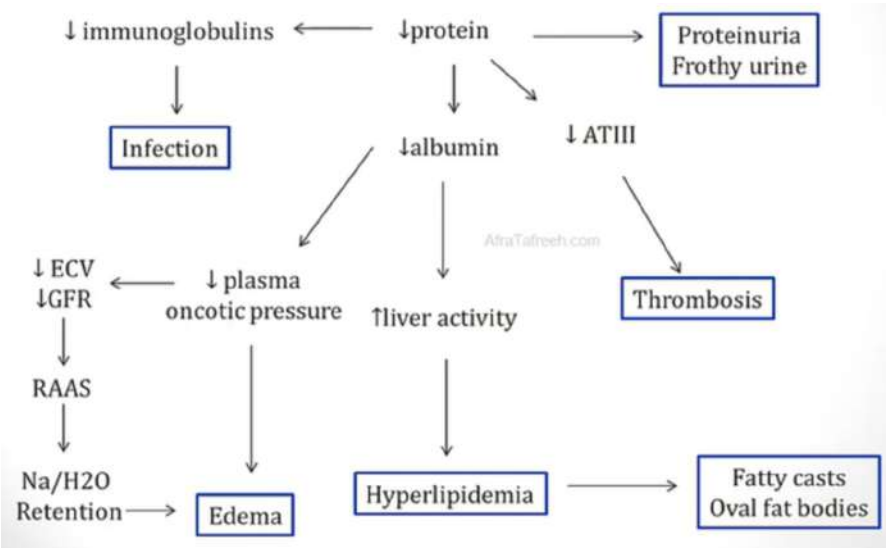
Spectrum

Nephritic Syndrome
1.RBC casts
2.Mild proteinuria
3.Renal Failure
*loosing both RBCs and Proteins

Nephrotic Syndrome
1.Massive proteinuria
2.Hyperlipidemia
*loosing lots of albumin

Nephrotic Syndrome

*Lost in filtration barrier to protein -> Massive proteinuria (+4,>3.5g/day)
*RBC filtration barrier remains intact.
*Triggers cascade of pathology



هاي الرسمة كثير حلوة، ركزوا معي 🤓
نقصان البروتين حيؤدي الى 5 أمور :

١. وجود بروتينات بالبول و حتى بكون frothy يعني مزبد و رغوي

٢. نقصان كمية ال immunoglobulin و بالتالي حدوث infection

٣. نقصان الalbumin و الي يؤدي الى :
- نقصان الplasma oncotic pressure و بالتالي حدوث edema و برضه نقصان الextracellular volume و نقصان glomerular filtration rate و بالتالي تفعيل الrenin angiotensin aldosterone system و بالتالي حدوث retention للصوديوم و الماء و بالتالي حدوث edema
- نقصان الalbumin برضه بحفز عمل الliver و بالتالي بصير عنا hyperlipidemia و زيادة total cholestrol
- برضه نقصان الalbumin يؤدي الى نقصان anti-thrombin 3 و بالتالي حدوث thrombosis و pulmonary embolism

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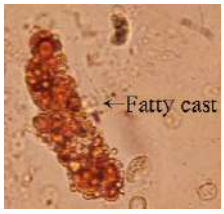

Urine in Nephrotic Syndrome:

*Urinary lipid may be present

*Trapped in casts (fatty casts)

*Enclosed by plasma membrane of degenerative epithelial cells (oval fat bodies)

*Under polarized light fat droplets have appearance of (Maltese cross)

Classic presentation

1-Swelling of ankles

2-Swelling around eyes (periorbital) -> Often mistaken for allergic reaction

3-Proteinuria (>3.5g/day)

Nephritic Syndrome

*Inflammatory process damages entire glomeruli

*Filtration barrier to RBCs and protein is lost

*Glomerular damage: low GFR

***RBCs in urine :**
- Dysmorphic.
- RBC casts.

***Protein in urine :**
- Less than nephrotic syndrome due to lower GFR
- <3.5g/day

***Classic presentation**

1-Dark urine (RBCs)

2-Fatigue (uremia)

3-Proteinuria (<3.5g/day)

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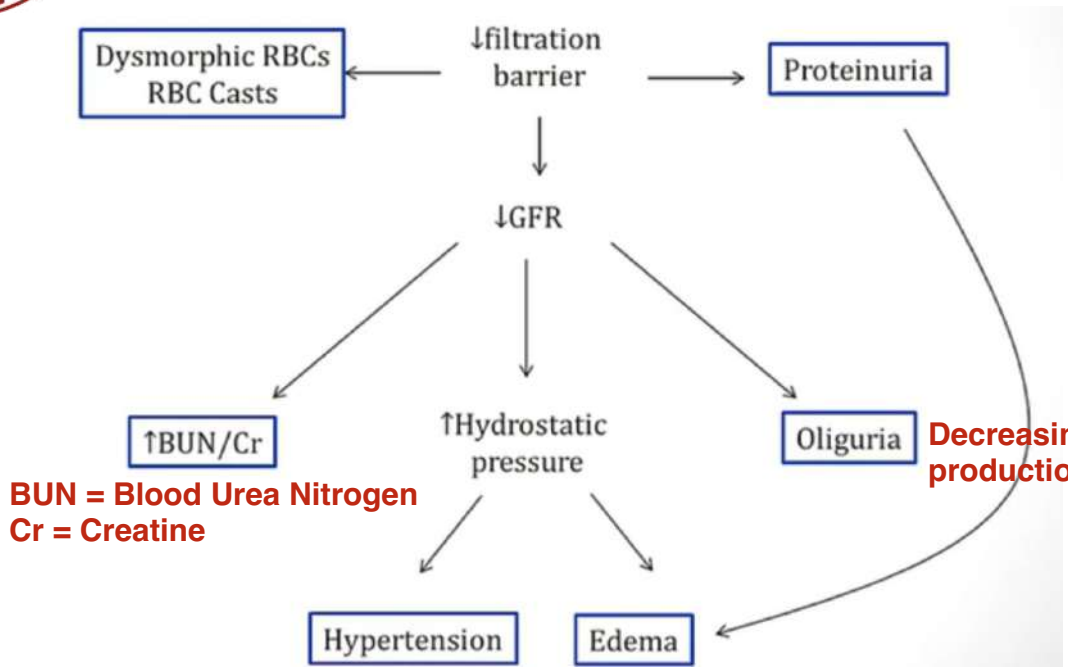
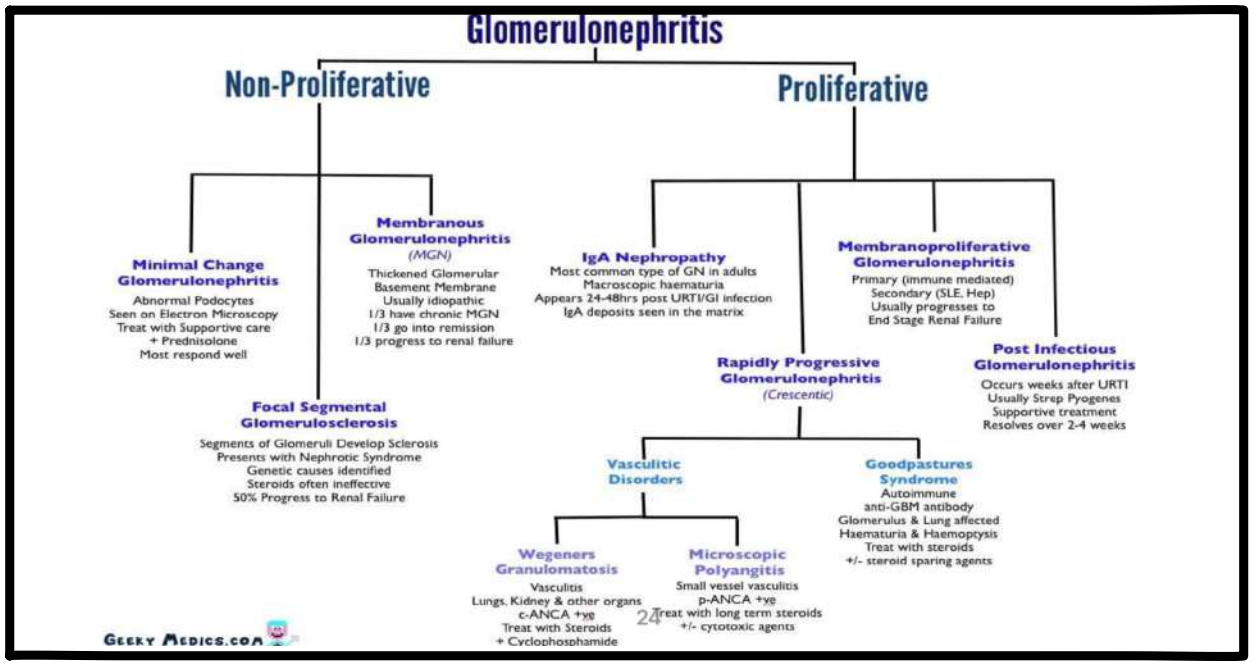


BUN = Blood Urea Nitrogen
Cr = Creatine

Decreasing in urine production

GENITOURINARY SYSTEM

هدول سلايدتين من المحاضرة الماضية، فيهم ملخص لشغلات حنوخدهم لقدام، موجودين بالملف الكبير و مو موجودين بملف المحاضرة، لهيك للاحتياط بدي اذكركم فيهم



	Nephrotic Features	Nephritic Features
Minimal-change nephropathy	++++	–
Membranous nephropathy	++++	+
Diabetic glomerulosclerosis	++++	+
Amyloidosis	++++	+
Focal segmental glomerulosclerosis	+++	++
Mesangioproliferative glomerulonephritis	++	++
Membranoproliferative glomerulonephritis	++	+++
Proliferative glomerulonephritis	++	+++
Acute poststreptococcal glomerulonephritis	+	++++
Crescentic glomerulonephritis[a]	+	++++

[a]Can be immune complex-mediated, antiglomerular basement membrane antibody-mediated, or associated with antineutrophil cytoplasmic autoantibodies.

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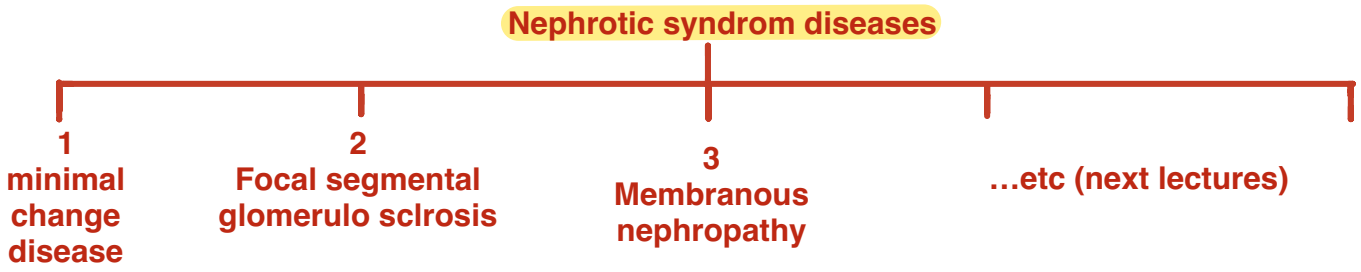
موضوعنا اليوم هو Nephrotic syndrom و نفس الشي بالاول بدي اشرح الكم بعدين ننتقل للسلايدات لنفهمهم

شرح الموضوع على باثوما :

https://mega.nz/folder/cEMQELzL#FLrqg-PXmBMjoNNpOwK2SQ/file/NAVnCl5l

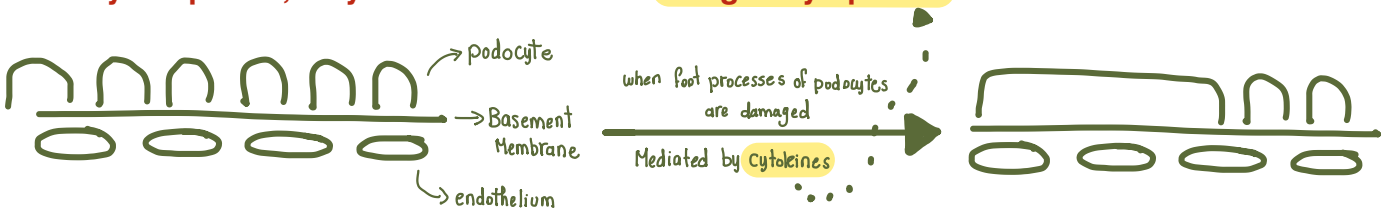
-Nephrotic Syndrom is a Glomerular disorders with proteinuria (> 3.5 g/day)
-Hypoalbuminemia -> decrease oncotic pressure -> edema
-Hypogammaglobulinemia
-Hypercoagulable state -> loss of antithrombin 3
-Hyperlipidemia and hypercholesterolemia



1- Minimal Change Disease (MCD):

- Most common cause of nephrotic syndrome in children

- Usually idiopathic; may be associated with Hodgkin lymphoma



- Normal glomeruli on H&E stain, lipid may be seen in proximal tubule cells

- Effacement of foot processes on EM

- No immune complex deposits; negative IF -> no deposition of immunoglobulin.

- Selective proteinuria (loss of albumin, but not immunoglobulin}

- Excellent response to steroids (damage is mediated by cytokines from T cells)

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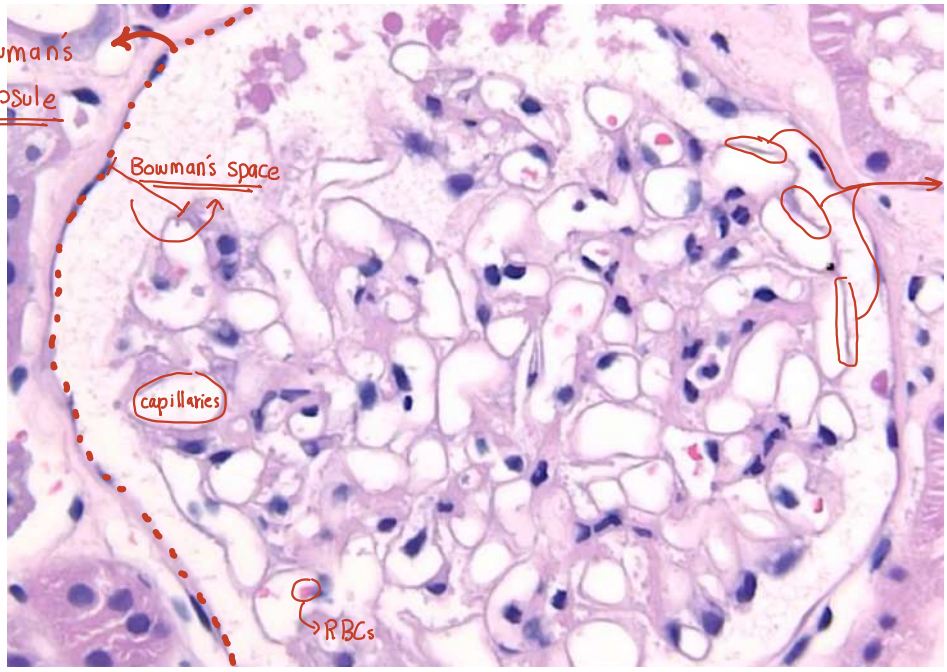
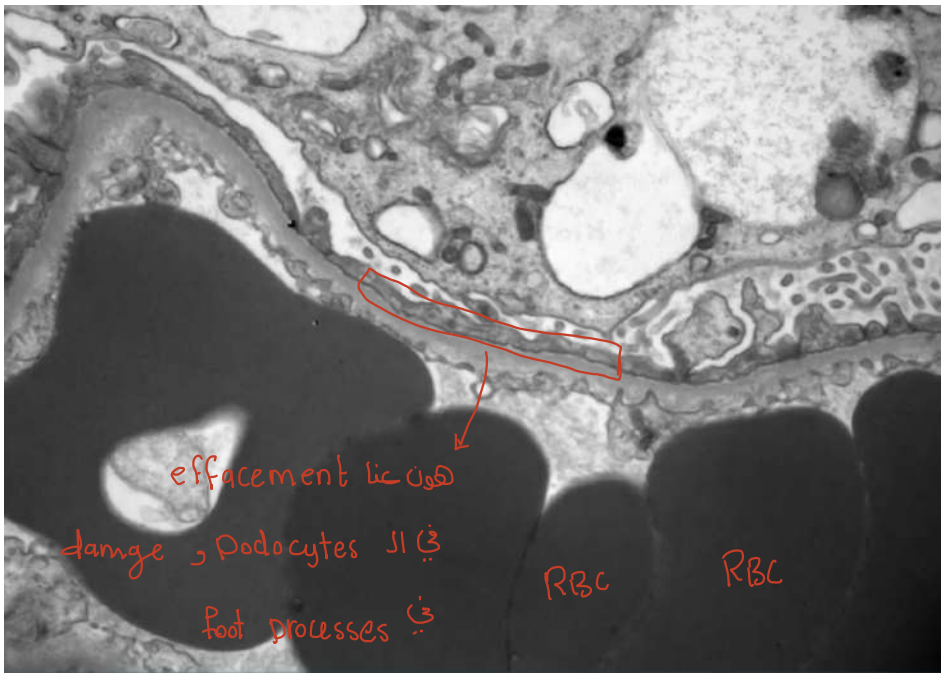
normal glomeruli under light microscope بكون عندهم





ال diagnose بصير عن طريق الelectron microscope, لاحظوا كيف الpodocyte عاملة

و يلا ننتقل عالسلايدات ...

الي بهم الدكتورة بكل مرض، تعريفه، اسبابه، الpathogenesis، الlight microscope، ال electon
microscope, و برضه الimmunoflurecense

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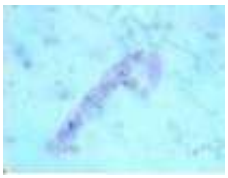
The Nephrotic Syndrome

❑a clinical complex resulting from glomerular disease & includes the following:

- (1) massive proteinuria (3.5 gm /day in adults).
- (2) hypoalbuminemia (≤ 3 gm/dL).
- (3) generalized edema
- (4) hyperlipidemia and lipiduria.
- (5) little or no azotemia, hematuria, or hypertension.



هم عبارة عن كمشة امراض و ال labratory findings تبعوهم مشروحين فوق و فهمناكم اياهم بالشرح فوق بس ضيفوا عليها hyaline cast و fatty cast ✅ الدكتورة ضافتها

Causes of Nephrotic Syndrome

Primary Diseases that Present Mostly with Nephrotic Syndrome

1-Minimal-change disease

2-Focal segmental glomerulosclerosis(FSGS).

3-Membranous nephropathy

4-membranoproliferativeGN type 1 (usually a combination of nephrotic/ nephritic syndrome)

Systemic Diseases with Renal Manifestations:

1. Diabetes mellitus.
2. Amyloidosis
3. Systemic lupus erythematosus
4. drugs (gold, penicillamine, "street heroin").
5. Infections (malaria, syphilis, hepatitis B, HIV).
6. Malignancy (carcinoma, melanoma).
7. Miscellaneous (e.g. bee-sting allergy)

مو بس الkidney بتكون involved

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Minimal Change Disease (Lipoid Nephrosis),nil disease, and foot process disease

➢ **Essential Features :**

- MCG is the most frequent (about 65%) cause of the nephrotic syndrome in children.
- Although it may develop at any age, MCD is most common between ages 1 and 7 years.
- It is characterized by **G** that have a normal appearance by light microscopy, but when viewed with the **EM it** shows:

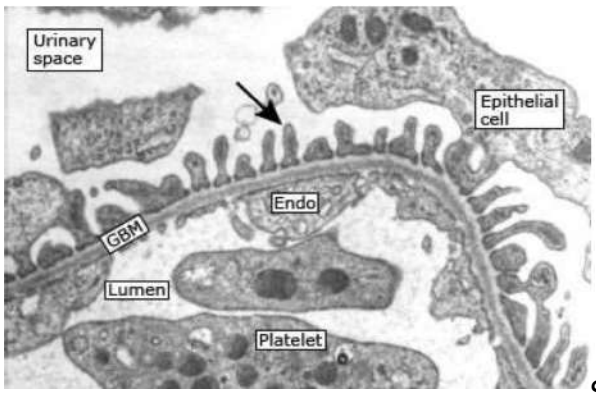
(1)diffuse effacement of podocyte foot processes

(2) Without antibody deposits.

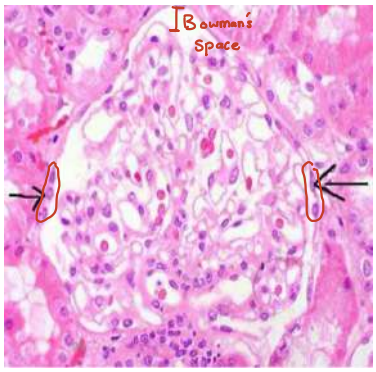
- **Pathogenesis:** The pathogenesis of podocyte injury, which is the underlying mechanism of proteinuria in MCD is **unknown** & it may be the result of nonimmune causes.

و ممكن نعتبر انه وحدة من اسبابها هو Dysfunction في T-cell فال cytokines حتعمل injury

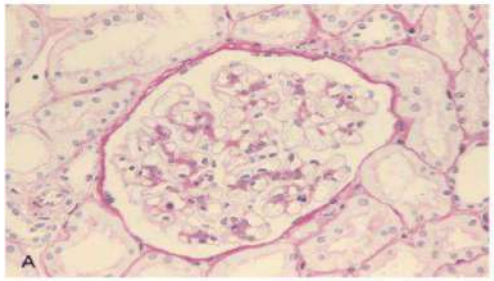
Normal Glomerulus by electron microscope

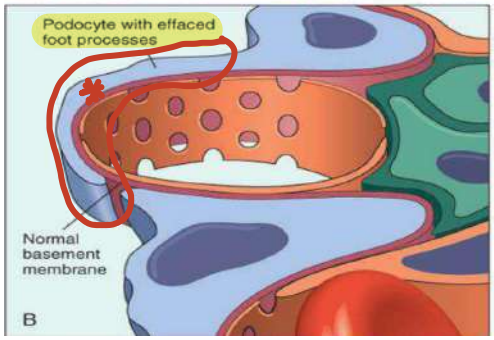


Normal Glomerulus



See the cellularity of the glomerular tuft. The arrows indicate nuclei of parietal epithelial cells covering the Bowman's capsule. In vivo the Bowman's space is narrower than seen in conventionally





Minimal change disease.

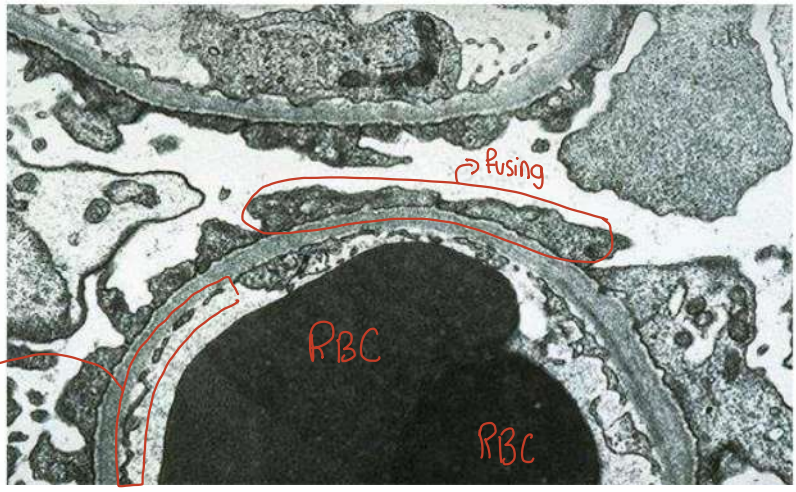
A
Glomerulus appears normal, with a delicate basement membrane
B
diffuse effacement of foot processes of podocyteswith no immune deposits.

Morphology

- **LM**
- the glomeruli appear normal.
- **IF**
- negative
- **EM**
- **uniform and diffuse effacement of the foot processes of the podocytes.**
- No immune deposits

اذا بال**EM** صار عنا **effacement** بال **podocytes** و **fusing**

MCD-EM the capillary loop in the lower half contains two electron dense RBC's. Fenestrated endothelium is present and the BM is normal. The overlying epithelial cell foot processes are fused (arrows).



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MCD Clinical Course

- nephrotic syndrome in an otherwise healthy child.
- no hypertension
- renal function preserved
- selective proteinuria (albumin)
- prognosis is good .
- Treatment : corticosteroids 90 % of cases
- < 5 % develop chronic renal failure after 25 years
- In Adults with minimal change disease the response is slower and relapses are more common

غالبا بكون طفل عنده edema و انتفاخ بالرجلين ، ضغطه طبيعي، و حنلاحظ انه الprotein urea حتكون selective لل albumin، و لما اطلب kidney function test حنلاحظ انه كويس فال renal function بتكون preserved
و اعرفوا كمان انه الprognosis بالاطفال افضل من الadults

2- Focal Segmental Glomerulo Sclerosis (FSGS):

- Most common cause of nephrotic syndrome in Hispanics and African Americans

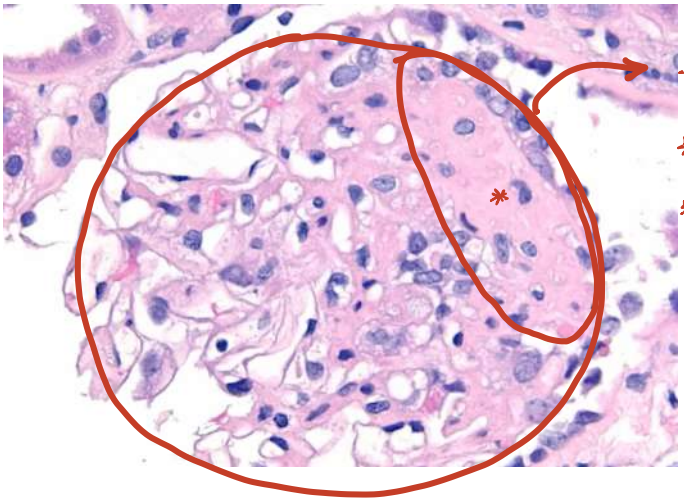
- Usually idiopathic; may be associated with HIV, heroin use, and sickle cell disease

- Focal and segmental sclerosis on H&E ->
ركزوا على كلمة focal، يعني مش diffuse يعني منطقة معينة من الglomeruli، كلمة sclerosis يعني scaring حيصر في الtissue

- Effacement of foot processes on EM

- No immune complex deposits; negative IF

- Poor response to steroids; progresses to chronic renal failure



Focal and Segmental Glomerulosclerosis (FSGS)

- Essential features
- **Glomerular** lesion characterized histologically, by;

A. sclerosis affecting some, but, not all **G** (**focal** involvement) & involving only some (**segments**) of each affected **G**

B. **often** associated with the **nephrotic syndrome**, can occur:

(1) in **association** with other known conditions, e.g., **HIV nephropathy, heroin nephropathy**;
(2) As a secondary event in other forms of GN(e.g., **[IgA] nephropathy**);
(3) as a **maladaptation after nephron loss.**
(4) in **inherited or congenital** forms resulting from mutations affecting cytoskeletal or related proteins expressed in podocytes (e.g., nephrin), i.e. nonimmune cause;

***FSGS is a serious condition, can lead to kidney failure, which can only be treated with dialysis or kidney transplant.**

***Nephrin is a protein crucial for maintaining the structure and function of the glomerular filtration barrier. In FSGS, there is often a disruption in nephrin expression or function, contributing to increased permeability of the glomerular barrier and protein leakage into the urine, a hallmark of the disease.**

- **(Nephrin a transmembrane glycoprotein, is the major component of the slit diaphragms between adjacent foot processes)**
- (5) as an **primary or idiopathic** FSGS, which accounts for **20% to 30%** of all cases of the nephrotic syndrome.
- It is becoming an **increasingly common** cause of nephrotic syndrome in **adults (35%)** & remains a frequent cause in children.

Very important

- In children it is important to distinguish **FSGS** cause of the nephrotic syndrome from **MCD**, because the clinical courses and prognosis are markedly different:
- **Unlike MCD, patients with FSGS have**

(1)Nonselective proteinuria,
(2) Higher incidence of hematuria & hypertension
(3) Generally, a poor response to corticosteroid therapy, with 50% of cases developing RF within 10 years of diagnosis. Adults in general feel even less well than children.

مهم جدا نقارن بينهم و محور الاختلاف هو الlight microscope

لو اجاك سؤال، طفل عمره سبع سنوات، عنده edema، normal glomeruli in light microscope
بدي افكر بال MCD
طيب طفل عنده heavy protein urea و هي non selective و الطفل عنده nephrotic syndrom و بالLM
ظهر انه some of G is effected و فيها lesions
لهاي الحالة بدي افكر ب FSGS

Pathogenesis

- The pathogenesis of primary FSGS **is unknown**.
- In any case, nonimmune injury to the podocytes is thought to represent the initiating event of primary FSGS **(as** with MCD)& is the underlying mechanism of proteinuria.
- The **permeability-increasing factors produced by lymphocytes** have been proposed in both MCD & FSGS.

- هو اكثر خطورة

- المشكلة هي dysfunction و dysregulation في الT-lymphocyte

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- The recurrence of proteinuria in some persons with FSGS, who receive **renal allografts**, sometimes within 24 hours of transplantation, supports the idea that a **circulating mediators is the cause of the damage to podocytes.**
- The deposition of **hyaline masses** in the **G** in FSGS represents the entrapment of plasma proteins & lipids in foci of injury where sclerosis develops.
- IgM & complement proteins commonly seen in the lesion are also believed to result from nonspecific entrapment in damaged **G**.

- A renal allograft refers to a transplanted kidney. The term "allograft" specifically denotes that the kidney comes from a genetically non-identical donor.

Morphology

- **Microscopically** :

FSGS is characterized by both focal & segmental lesions occurring in

1) some segments within a G & sparing of the others (hence the term "segmental"),

2) the disease first affects only some of the G (hence the term "focal").

- **The affected G exhibit**

(a)Increase mesangial matrix,

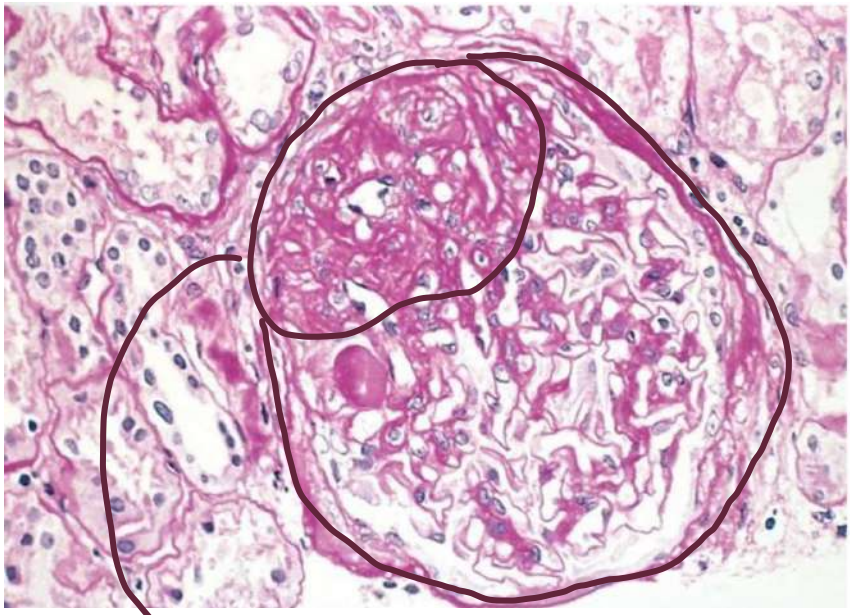
(b) deposition of hyaline masses (hyalinosis) & lipid droplets in the affected G (PAS+, trichrome red, silver negative) and endocapillary foam cells or lipoid droplets in focal glomeruli, , causing....

(C) obliteration of the capillary lumens

leads to

- **immunofluorescence M often reveals nonspecific trapping of immunoglobulins, usually IgM, & complement, in the areas of hyalinosis.**
- **Focal tubular atrophy with interstitial fibrosis, hyaline thickening of afferent arterioles**
- **Note: the defining glomerular lesions may not be sampled in needle core biopsy due to their focal nature**

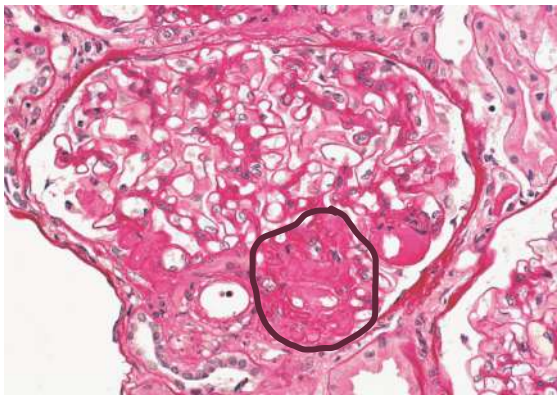
Very important

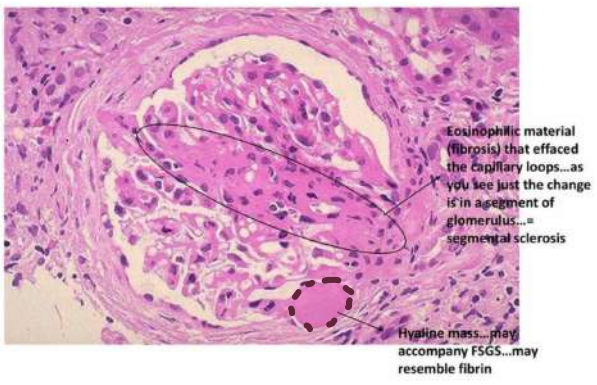


HP view of **focal & segmental glomerulosclerosis (FSGS),** seen as a mass of **scarred, obliterated capillary lumens with accumulations of matrix material,** that has replaced a portion of the glomerulus.

↑ mesangeal matrix
→ Segmental
↓ capillary Lumen

Perihilar





In contrast to minimal change disease, patients with FSGS are more likely to have non-selective, proteinuria, hematuria, progression to chronic renal failure, and poor response to corticosteroid therapy.

- On EM, as in MCD, the podocytes exhibit **effacement of foot processes,**
- Clinically, there is **little tendency for spontaneous remission** of idiopathic FSGS, & responses to corticosteroid therapy are poor.
- **Progression** of FSGS, with time, leads to **global sclerosis** of the **G** with pronounced tubular atrophy & interstitial fibrosis, a picture difficult to differentiate from other forms of chronic **G** disease, with progression to RF occurring in 50% of FSGS patients after 10 years.

renal failure

	MCG	FSGN
Hematuria	–	+
Hypertension	–	+
Proteinuria	Selective	Non-selective
Respond to corticosteroid therapy	Good	Poor

Columbia University classification: perihilar, cellular, tip lesion, collapsing and not otherwise specified ; correlates with prognosis

Collapsing glomerulopathy

Essential features

- A morphologic type of FSGS.
- At least 1 glomerulus with capillary loop collapse and prominence of overlying podocytes or parietal epithelial cells
- Worse prognosis than other variants of focal segmental glomerulosclerosis; supersedes other variants if others present in biopsy.
- May be idiopathic or associated with viruses, genetics, drugs, vascular injury and autoimmune diseases
- CC with Nephrotic range proteinuria
- Elevated serum creatinine at presentation

في عنا انواع للFSGS اخطرهم هو الcollapsing

هاي سلايد اضافية مني لو بتحبوا تتعرفوا عالانواع

Type	Key histologic feature	Possible prognostic implication
FSGS NOS	Segmental sclerosis	Typical course
Collapsing FSGS	Collapse of tuft, podocyte hyperplasia	Poor prognosis
Cellular FSGS	Endocapillary proliferation, often podocyte hyperplasia	?Early-stage lesion
Tip lesion	Sclerosis/adhesion at proximal tubule pole	?Better prognosis
Perihilar variant	Sclerosis and hyalinosis at vascular pole	?May reflect a secondary type of FSGS

NOS, not otherwise specified.

3- Membranous nephropathy :

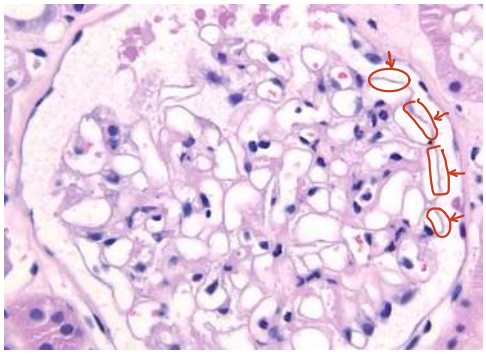
- Most common cause of nephrotic syndrome in Caucasian adults

- Usually idiopathic; may be associated with hepatitis B or C, solid tumors, SLE, or drugs (e.g., NSAIDs and penicillamine)

موضوع علاقتها بالSLE مهم ليش ؟ اهم سبب لموت الناس الي بصير معهم SLE هو renal failure و اكثر سبب اله و اله علاقة بالSLE هو ال membranous nephropathy هاي معلومة كتكوتة الكم ❤️

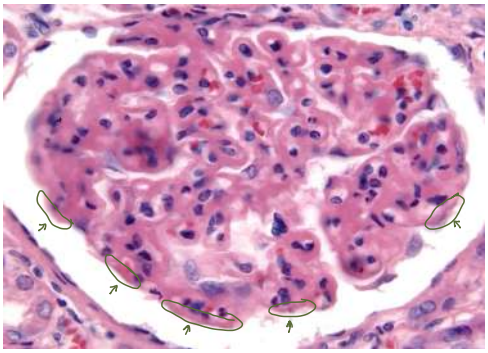
- Thick glomerular basement membrane on H&E

أي مرض فيه كلمة membrane معناته في thickening بالموضوع



"Normal"

لاحظوا كيف ال BM صارت thin

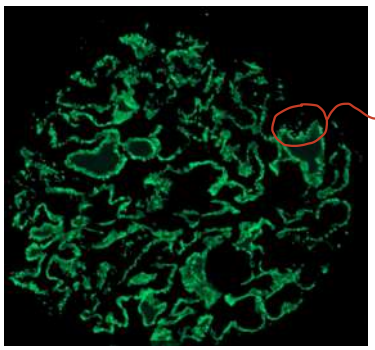


"membrano nephropathy"

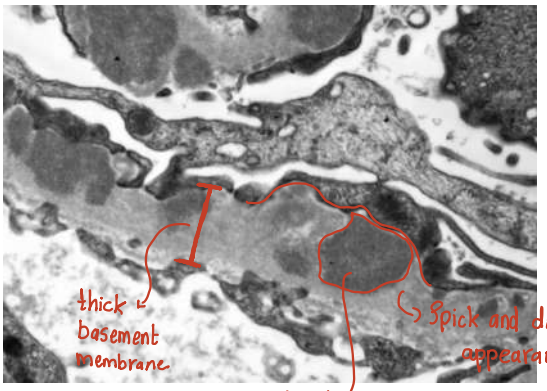
لاحظوا كيف صار ال BM ← thick.

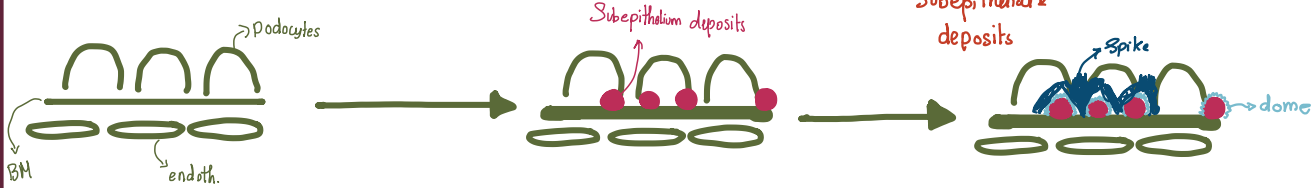
- Due to immune complex deposition (granular IF)

- Subepithelial deposits with 'spike and dome' appearance on EM



Circular immune complex deposition





وقل رب زدني علماً

GENITOURINARY SYSTEM

Membranous GN(MGN)=Membranous Nephropathy MN)

- A slowly progressive disease, most common in the 30-50 years age group, characterized by the presence of:

(I)Diffuse thickening of the capillary wall,
(II)subepithelial immunoglobulin-containing deposits.

مرض بصيب الadults و بكون diffuse زي MCD و فيه شي مميز الي هو ال thickening
و مهم نكون عارفين انه الdeposition بصير تحت الpodocytes

- **Pathogenesis**
- MGN is a form of **chronic immune complex nephritis**.
- Although circulating complexes of known exogenous (e.g., hepatitis B virus) or endogenous (DNA in SLE) Ag can cause MGN,
- it is now thought that most idiopathic MGN are induced by Abs reacting in situ to endogenous, or, planted **G** Ags.

in cases of idiopathic membranous glomerulonephritis (MGN), it is currently believed that the condition is triggered by antibodies (Abs) reacting within the kidney to either self-produced or introduced (planted) foreign substances known as glomerular antigens (G Ags).

Types of Membranous glomerulonephritis :

1-Idiopathic (85% of cases):

- **Most common cause of nephrotic syndrome in nondiabetic adults**
- Idiopathic autoimmune glomerular disease characterized by diffuse subepithelial immune complex deposition with nephrotic range proteinuria, without known systemic cause.
- Thickening of glomerular basement membrane and subepithelial deposition of immune complexes (silver stain, spike)
- Anti-PLA2R autoantibodies **= phospholipase A 2 receptor**
- Circulating autoantibodies bind to an autoantigen on the surface of the podocytes resulting in in situ immune complex formation that activates the lectin complement pathway and causes podocyte injury and proteinuria
- 2 major target antigens are now firmly recognized: the M type phospholipase A2 receptor 1 (PLA2R) (~70%) and the thrombospondin type 1 domain containing 7A (THSD7A) (2 - 5%)

احفظوهم ممكن يجوا ك hint بالامتحان

وقل رب زدني علماً

2-Secondary membraneous nephropathy

- (1) infections (HBV, syphilis, schistosomiasis, malaria).
- (2) malignant tumors (lung, colon and melanoma).
- (3) autoimmune diseases as SLE .
- (4) inorganic salts exposure (gold, mercury).
- (5) drugs (penicillamine, captopril,NSAID).

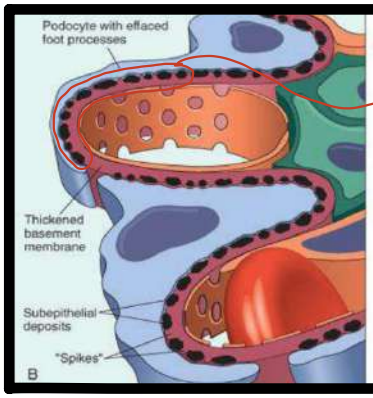
موضوع الdrugs كثير مهم هون خصوصا البنسلين و بتكرر بالامتحانات

Morphology

- LM
- **diffuse thickening of the GBM**. -> It is not focal
- IF
- **deposits** of immunoglobulins and complement along the GBM (IgG)
- By EM

(1) the podocytes show effacement of foot processes, &
(2) the diffuse thickening of the GBM is caused in part by subepithelial dome deposits that nestle against the GBM & are separated from each other by small, spike like protrusions of GBM matrix that form in reaction to the dome deposits, resulting in a (spike & dome pattern).
(3)As the disease progresses, these spikes close over the deposits, incorporating them into the GBM.

الbasement membrane عمل reaction فصار عنا spike and dome pattern

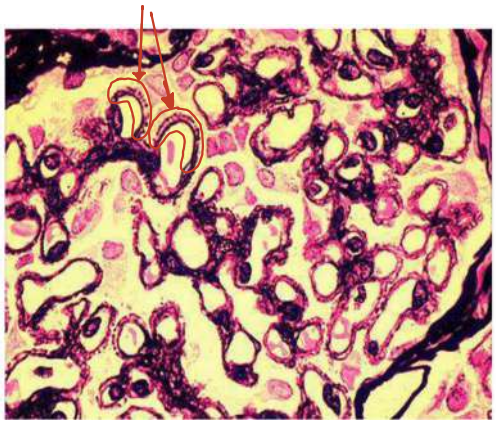


Membranous nephropathy.
Sub epithelial deposits and the presence of "spikes" of basement membrane material between the immune deposits.

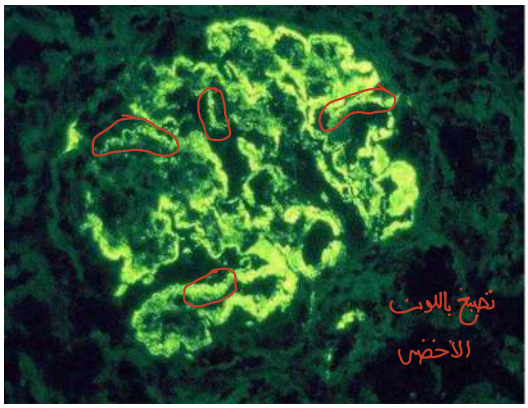
وقل رب زدني علماً

GENITOURINARY SYSTEM

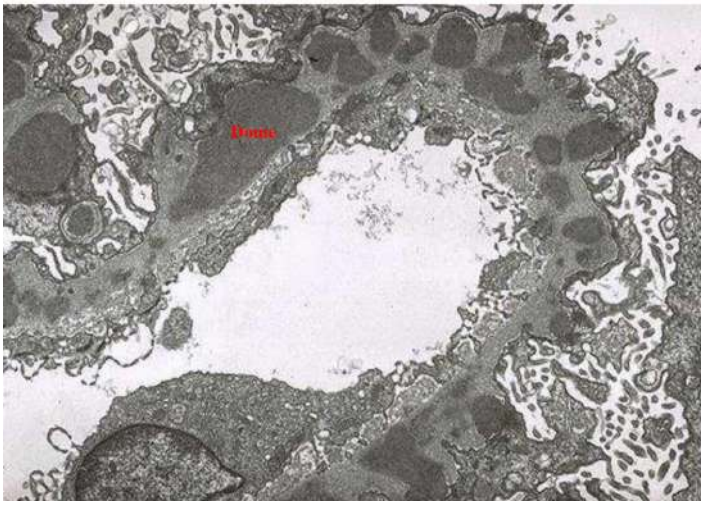
Very important



A silver stain (black). Characteristic "spikes" seen with membranous glomerulonephritis as projections around the capillary loops.

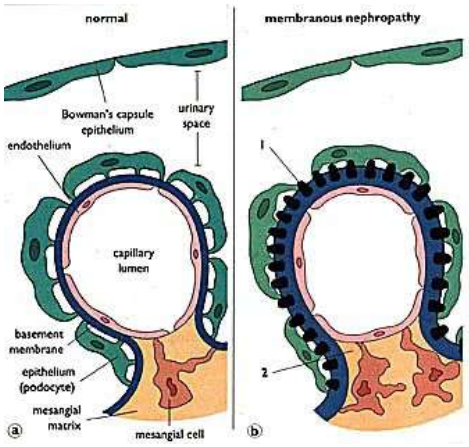


Finely granular staining for gG, predominately IgG4, presents uniformly in a subepithelial distribution in all glomeruli



EM- ("spike and dome"pattern)

Very important



وقل رب زدني علماً

Clinical Course

- Clinically, idiopathic MGN characterized by **insidious development of the nephrotic syndrome**, usually without antecedent illness.
- **In contrast to MCD,**

(I) the proteinuria is nonselective,

(II) does not usually respond to corticosteroid therapy((**poor response to corticosteroid therapy**)).

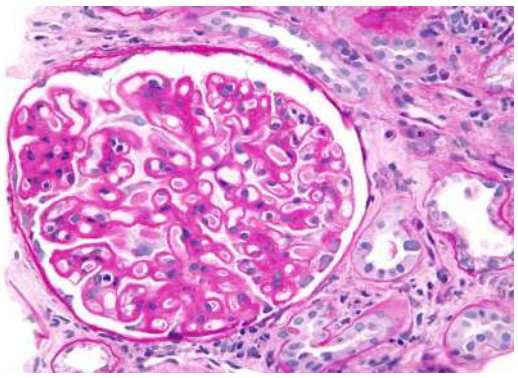
- **Secondary causes of MGN should be ruled out.**

Prognosis:

60% من المرضى حتى لو اخدوا علاج حتضل الproteinuria موجودة

- **60% of cases → proteinuria persists**
- **About 40%→progressive disease and renal failure 2 to 20 yr.**
- **30% →partial / complete remission of proteinuria.**

A 52-year-old female comes to the primary care physician due to worsening weight gain. She has gained approximately 20 lbs over the past six weeks. In addition, the patient has been experiencing lower extremity edema and has noticed her urine appears more cloudy than usual. Past medical history is notable for hypertension and hyperlipidemia. Temperature is 37.3°C (99.1°F), pulse is 76/min, respirations are 18/min, blood pressure is 137/84 mmHg, and O2 saturation is 97% on room air. Physical exam is notable for bilateral pitting edema of the lower extremities. A urinalysis is notable for 4+ protein and oval fat bodies but no hematuria. A renal biopsy is performed, and the following is visualized on light microscopy,Which of the following is most suggestive of this patient's underlying diagnosis?



A- IgG autoantibody that stabilizes C3 convertase

B- Positive glomerular immunostaining for IgA

C- Decreased serum levels of C3

D- IgG4 antibodies against phospholipase A2 receptor

E- Mutation in type IV collagen

GENITOURINARY SYSTEM

Answer those question

Q1:Which of the following is true about primary membranous nephropathy?

- Active periglomerular inflammation and rupture of Bowman capsule
- Little or no immunoglobulin or complement deposits by immunofluorescence
- Most common cause of idiopathic nephrotic syndrome in nondiabetic adults worldwide
- Significant mesangial or endocapillary hypercellularity

Q2:Which of the following is true about minimal change glomerulopathy?

- Interstitial inflammation and fibrosis are usually absent
- It is the most common type of nephrotic syndrome in adults
- Monoclonal antibody therapy should be the first line therapy
- Pretreatment biopsy is always done

Q3:Which of the following signs and symptoms is common in minimal change disease?

- Azotemia
- Hypertension
- Macrohematuria
- Selective proteinuria

Q4 : An 8-year-old boy presents to the primary care physician with facial puffiness. The patient's parent states, "He seems like he's gained some weight recently, and he's always complaining about his feet hurting now." She reports the patient had a "sore throat" 2-4 weeks ago. The patient's temperature is 37.0°C (98.6°F), pulse is 80/min, respirations are 22/min, blood pressure is 100/64 mmHg, and O2 saturation is 96% on room air. On physical exam, a comfortable appearing boy is noted to have 2+ pitting edema in the lower extremities. The posterior pharynx is without erythema or exudate. Cardiopulmonary exam demonstrates normal heart sounds and clear lung fields bilaterally. The patient is asked to provide a urine sample, and it is noted to appear frothy. Which of the following pathophysiologic processes is most likely contributing to this patient's clinical presentation?

A-Increased aldosterone and ADH secretion

B-Decreased lipoprotein synthesis

C-Poor cardiac ejection fraction

D-Hypercellular and inflamed glomeruli

E-Increased oncotic pressure

وقل رب زدني علماً

Q5 : A 13-year-old male with a history of non-Hodgkin lymphoma presents to the primary care physician with leg swelling. He states that "My shoes won't fit anymore, and I feel like I've gained weight too." The patient last received treatment for non-Hodgkin lymphoma 1.5 weeks ago. Temperature is 37.0°C (98.6°F), pulse is 80/min, respirations are 18/min, and blood pressure is 105/64 mmHg. On physical exam he is noted to have bilateral periorbital edema and 2+ pitting edema of the lower extremities. Urinalysis demonstrates 4+ protein and is negative for blood. The patient subsequently undergoes a kidney biopsy. Which of the following findings is most likely to be observed on electron microscopy?

A- Glomerular basement membrane with a lamellated basket weave appearance

B- Podocyte foot effacement

C- Thickened basement membrane with a spike and dome appearance

D- Mesangial immune complex deposition

E- Subepithelial immune complex humps

طولت عليكم بعدد الصفحات ❤️ بس ان شاء الله هيك
تكونوا فهمتوا و استوعبتوا الثلاث امراض الي حكيناها
لو في اي ملاحظات حأبلغكم بالكومنتات ❤️

وقل رب زدني علماً