# MEDICINE

## 432 Team

15

# Glomerular Diseases


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**COLOR GUIDE:** • Females' Notes • Males' Notes • Important • Additional

# Objectives

1- To understand the pathophysiology of primary Glomerular Diseases.

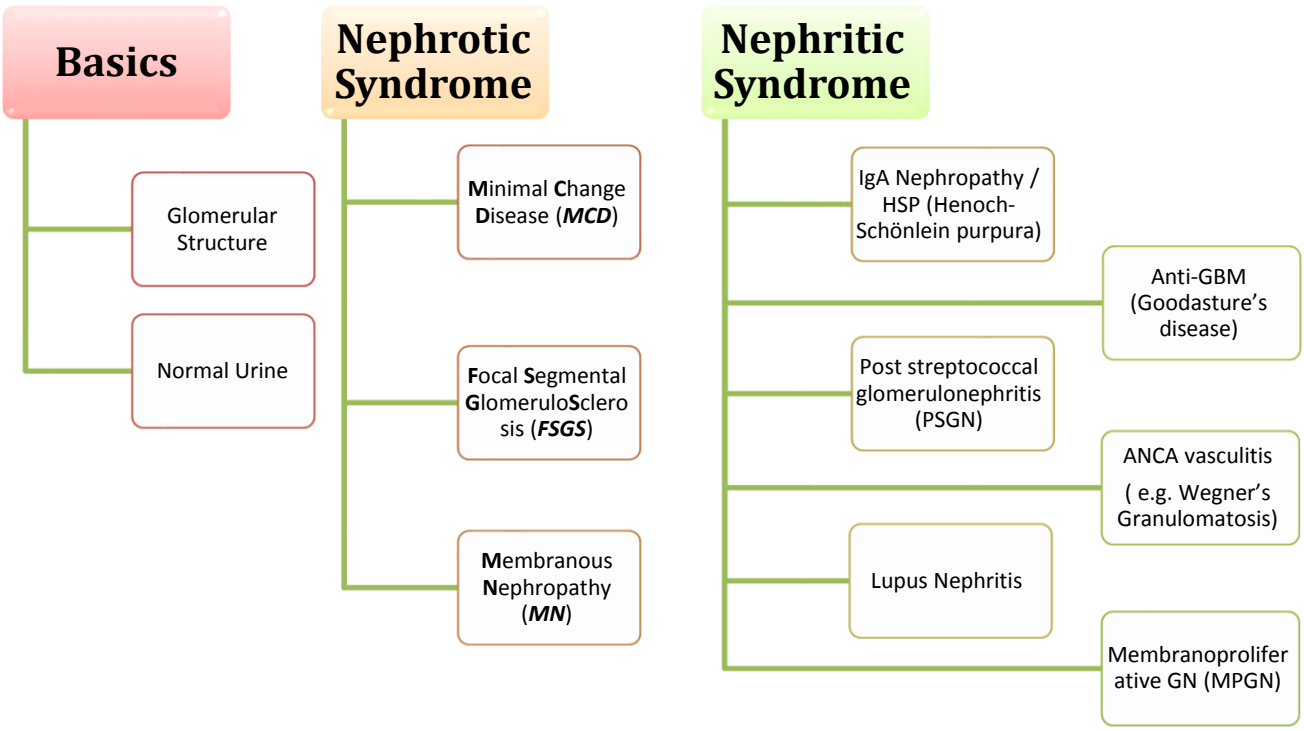
2- To correlate the clinical findings with the underlying renal pathology

3- To recognize the important features of Nephrotic syndrome.

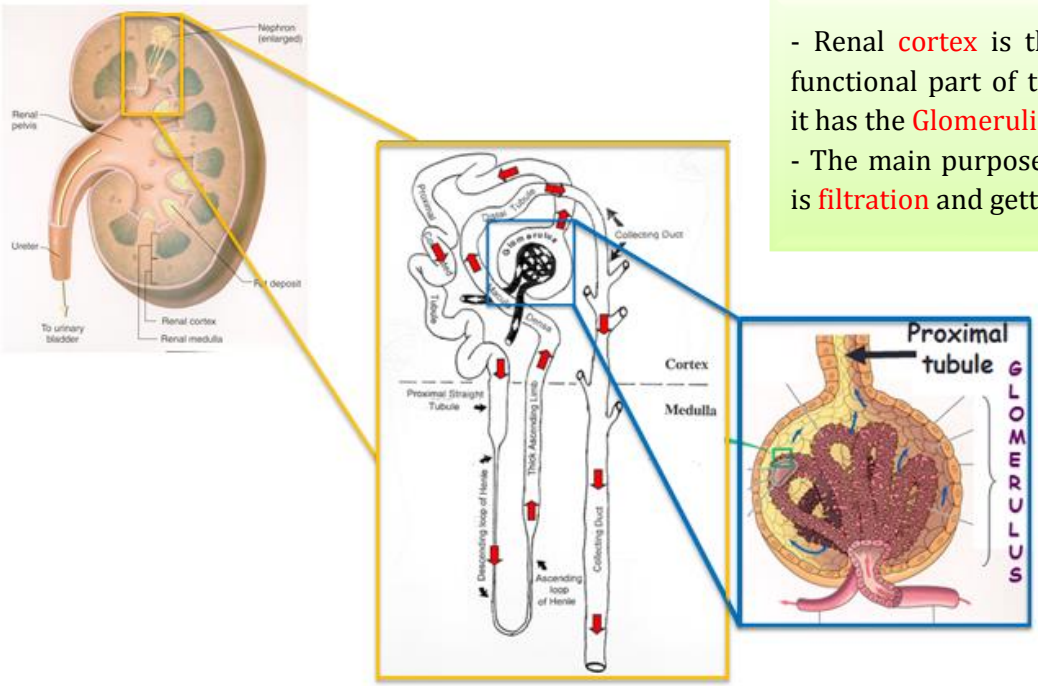
4- Learn the most common causes of NS in adults.

5- To recognize the most important Glomerular diseases that cause Nephritic (Glomerulonephritis) pattern of clinical presentation.

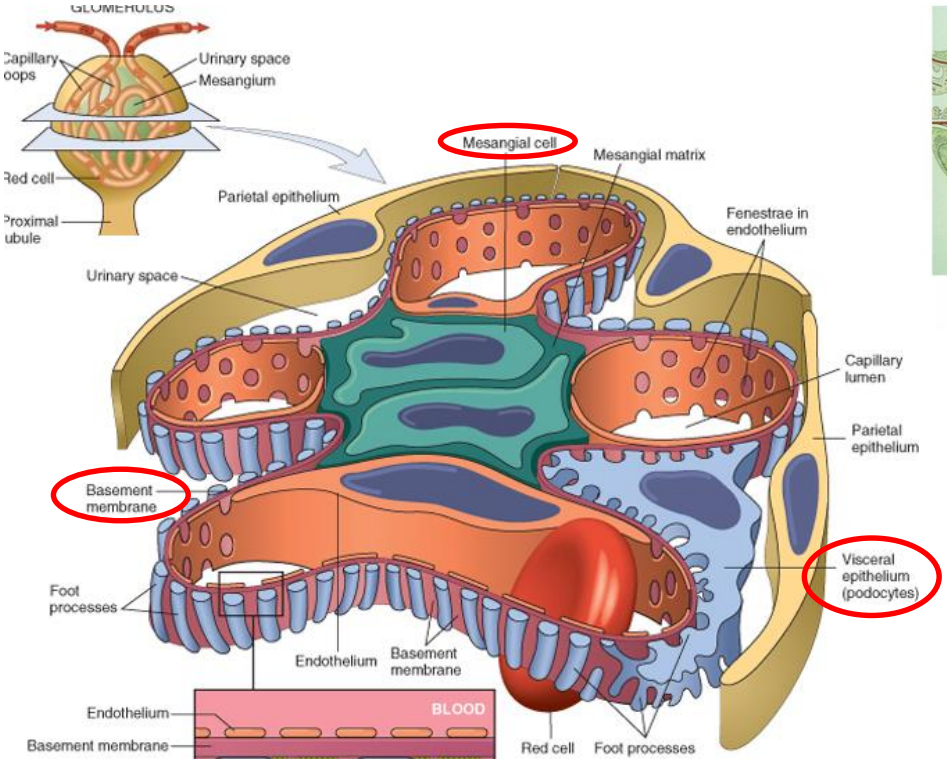
# Mind Map

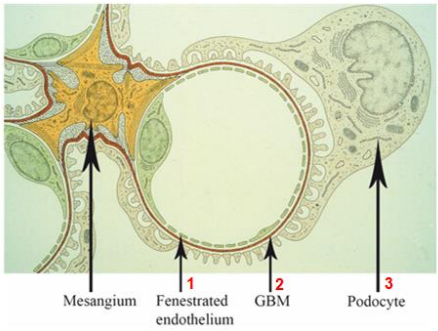


# Let's review some basics!




- Renal cortex is the most important functional part of the kidney, because it has the Glomeruli.
- The main purpose of the glomerulus is filtration and getting rid of toxins.

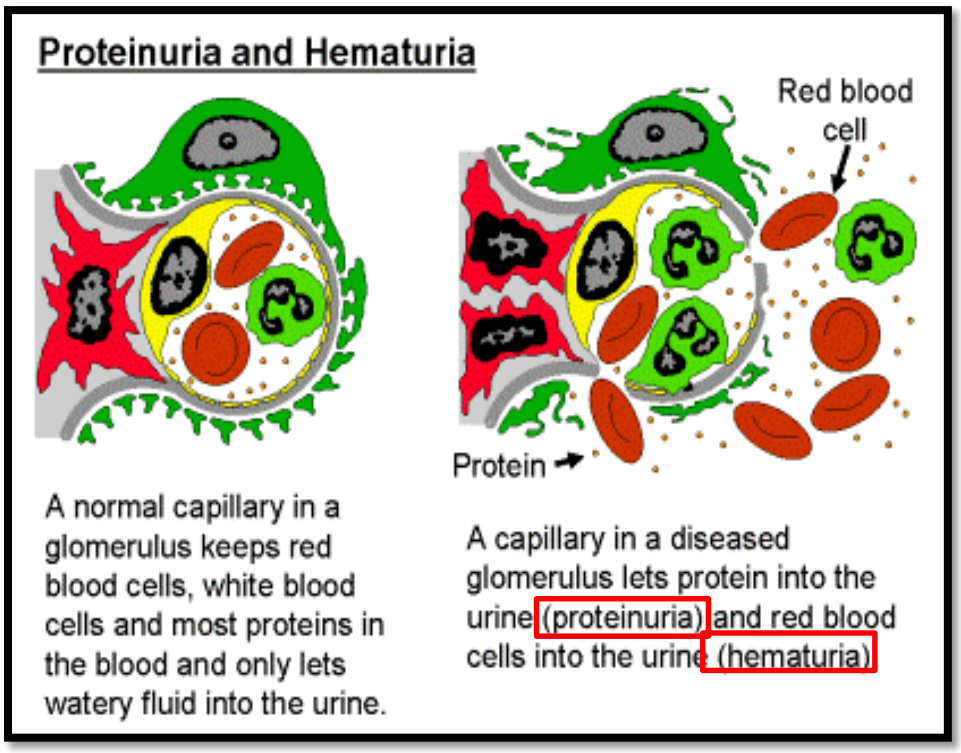


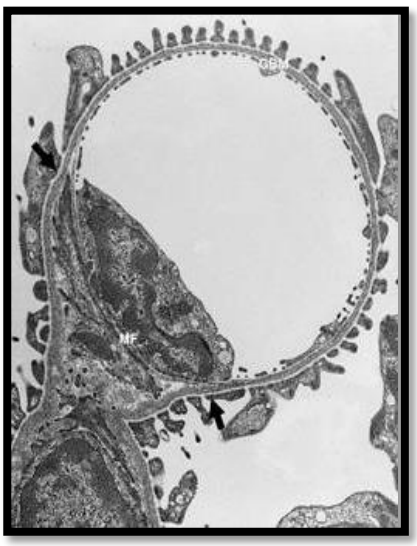




**The Glomerular Capillary wall has 3 layers, through which filtration occurs:**
1- Endothelial cells.
2- Basement membrane.
3- Podocytes, outside the basement membrane.

*Between the capillaries are the mesangial cells "they have an impact on immune system".

* Endothelium and GBM are close to the blood stream while podocytes are slightly away from it.



**Normal Capillary Loop (Electron Microscopy)**

## Normal Glomerular structure is needed to:

- Keeps the glomerular filtration normal, thus maintains normal kidney function.
- Keeps the urine volume maintained; so preventing fluid retention in the body which causes edema and high blood pressure.
- Prevents the blood components (cells, proteins) from leaving the blood stream and appearing in the urine.

### *So normal urine will have:*

- No protein. (if present: proteinuria)
- No red blood cells (accept: <3 RBCs/high power field)
- No heme.
- No cellular casts.
- No fat.
- No sugar.

Microscopy:
- **LM:** 2000x
- **EM:** 10,000,000

# How glomerular diseases start?

- Many times the exact cause is not really clear, but the result of the damage in the glomerulus is telling how immune system is playing an important role.
- Here we are talking about primary glomerular diseases that are mostly caused by **immune system dysfunction**.
- There are two mechanisms: **Auto-antibodies targeting glomerular structure** "local to the glomeruli" or **immune-complexes (antigen-antibody) depositing** and traumatizing the glomerular components "the glomerulus is a very fine filter and it almost filters everything, so it filters the complexes which leads to deposition and triggering more inflammation because the body recognizes them as foreign bodies".

## *Important things to remember*

- The manifestations of a glomerular disease are usually indicative of which component of glomerular capillary wall was affected.
- If **Podocytes** are the main target of the disease process, mainly **proteinuria** will manifest; thus **Nephrotic**.
- If the target is the podocytes "foot process" away for the blood stream, those digitations will disappear "effacement" > dysfunction of podocytes > proteinuria.
- If **endothelial cells, GBM** or **mesangial cells** are affected, mainly **hematuria** and **abnormal renal function** will manifest; thus **Nephritic**. Proteinuria is always present in this kind of injury.
- Mesangium is very sensitive. It starts to proliferate when anything "not part of it" attached to it "e.g. IgA" > disruption of capillaries > protein and RBCs in urine.
- Glomerular diseases are named based on their *histo-pathological characteristics* seen under the microscope. So, almost always a **kidney biopsy** is needed to diagnose a suspected primary glomerular disease.

**Glomerular diseases**

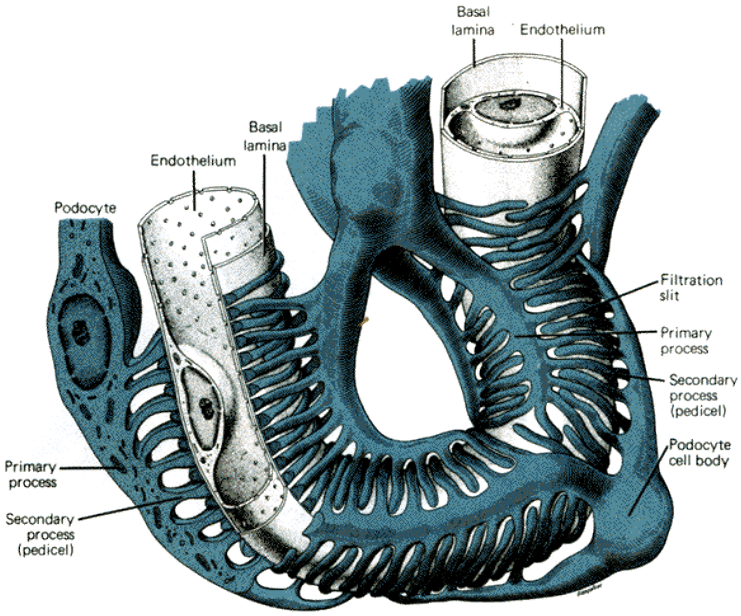
**Two main clinical categories**

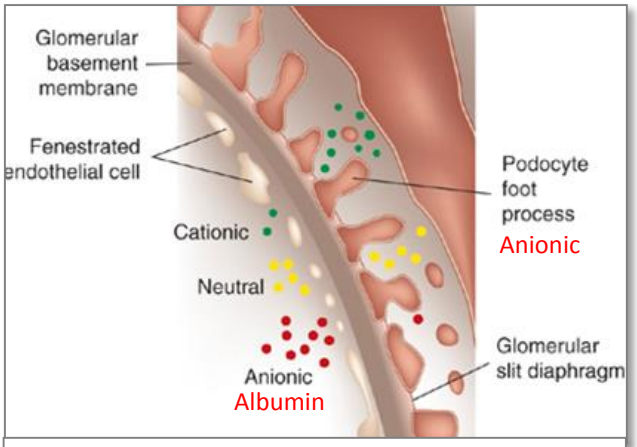
(clinical i.e. the symptoms, signs and laboratory abnormalities)

| | |
|---|---|
| **Nephrotic** | Due to Podocytes dysfunction, so heavy proteinuria will be present. |
| **Nephritic** | Due to glomerular capillaries inflammation; so hematuria, impaired renal function and variable amount of proteinuria will be present. |

# Nephrotic Syndrome (NS):

- Podocytes abnormality is the primary finding in NS.
- Podocytes will sustain a structural dysfunction; making them lose their Foot-processes.
- This will lead to significant amount of protein appearing in the urine (Proteinuria) or (Nephrotic range proteinuria).




Albumin and Podocytes have negative charges so they pull albumin from the glomerulus.

***It refers to a constellation of clinical and laboratory features of renal disease:***

- Hypoalbuminemia (<30 g/L) Normal serum Alb: 35-55g/L.
- Heavy proteinuria (> 3.5 g/24 hours).
- Peripheral or generalized edema. "oncotic pressure is decreased"
- Hyperlipidemia. "Liver is trying to compensate by making more proteins and lipoprotein carriers"

## *Complications:*

- Infections & sepsis. "loss of Ig in the urine"
- Thrombosis. "loss of anticoagulation factors in the urine"
- Acute kidney injury.
- ESRD if heavy proteinuria not going into remission.

## Proteinuria:

- How many mgs of proteins are normally secreted in the urine per-day?
  < 150 mg/day of all kinds of proteins including on average 4-7mg/day of Albumin that are secreted in the urine.

## Urine Analysis in Nephrotic Syndrome:

- Heavy protein (Proteinuria) or called Nephrotic range proteinuria.
- No RBCs (few are occasionally seen).
- No RBCs casts.
- Lots of fat (Lipiduria) (Fatty casts, oval fat bodies & fat droplets).
- No WBCs (few may be seen).

Pathological terms in glomerular disease: The most commonly used terms are:

- *Focal:* some, but not all, glomeruli show the lesion.
- *Diffuse (global):* most of the glomeruli (>75%) contain the lesion.
- *Segmental:* only a part of the glomerulus is affected (most focal lesions are also segmental, e.g. focal segmental glomerulosclerosis).
- *Proliferative:* an increase in cell numbers due to hyperplasia of one or more of the resident glomerular cells with or without inflammation.
- *Membrane alterations:* capillary wall thickening due to deposition of immune deposits or alterations in basement membrane.
- *Crescent formation:* epithelial cell proliferation with mononuclear cell infiltration in Bowman's space.

## Clinical Presentation:

- Generalized Edema "also called anasarca" due to:
  1- Low serum Albumin (Low oncotic pressure).
  2- Increase Renal sodium retention, because of uncontrolled activation of the epithelial sodium channels (ENaC channels in the renal tubules) > water will retain.
- Fatigue
- Frothy urine (froth persists for long time after voiding)
- Anorexia
- Nausea & vomiting
- Abdominal pain
- Weight gain due to fluid retention
- Shortness of breath if having pleural effusion
- Signs & symptoms of DVT, PE

Other **important causes** of Nephrotic syndrome in adults:

- **Diabetes Mellitus**
- **Amyloidosis**
- IgA Nephropathy
- MPGN

## Causes of Nephrotic Syndrome:

1- **M**inimal **C**hange **D**isease (***MCD***)
2- **F**ocal **S**egmental **G**lomerulo**S**clerosis (***FSGS***)
3- **M**embranous **N**ephropathy (***MN***)

# 1- Minimal Change Disease (MCD):

- ***Called*** **minimal** because: **Light microscopy** is typically showing normal glomeruli so called: nil disease. But Electron microscopy shows **diffuse effacement** of the epithelial cells' foot processes.
  - Therefore, the most important difference between MCD and the FSGS is the presence of **glomerular sclerosis** in FSGS.

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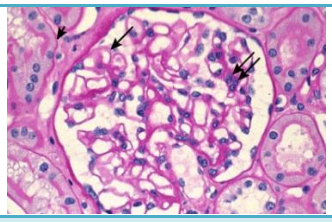
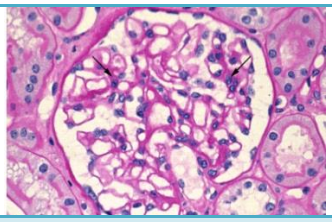
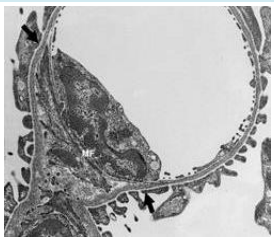
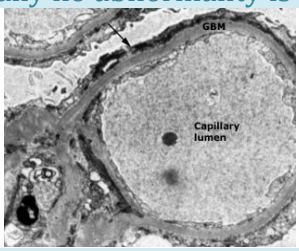
| **LM** |  |  |
| | Normal Glomerulus | MCD, basically no abnormality is seen on LM |
| **EM** |  |   |
| | Normal Glomerulus | MCD, EM shows the diffuse foot process effacement |

- It is the ***Main Cause*** of Nephrotic syndrome in **children**:
  - 90 % of cases in children < 10 years old.
  - > 50 % of cases in older children.
  - In children; typically is *corticosteroid responsive in > 90%*, thus *kidney biopsy* is commonly not done and treatment is given empirically for such cases.
- ***It Causes***: **10-25 %** of Nephrotic syndrome in **adults.**
- ***Can be :*** Primary (Idiopathic "like in children") **or** Secondary:
  - *Drugs* (**NSAIDs**, Lithium, Sulfasalazine, Pamidronate, D-penicillamine, some antibiotics)
  - *Neoplasm* (**Hodgkin Lymphoma**, non-Hodgkin lymphoma, and leukemia)
  - *Infections* (TB, syphilis)
  - *Allergy*.
- ***Clinical presentation:***
  - Typically has **a sudden onset Edema**. "within a week"
  - BP may be normal or slightly elevated.
  - Hypoalbuminemia (usually very low serum Albumin).
  - Heavy proteinuria (Nephrotic range).
  - Hyperlipidemia and Lipiduria.
  - Creatinine is always within the normal range or slightly elevated.
- ***Diagnosis:*** Must do **kidney biopsy** in adult patients with this presentation
- ***Treatment:*** **"only for Primary MCD" , "if Secondary: look for the underlying cause"**
  - *First line*: **Corticosteroids**, given x 3-4 months then taper over 6 months.
  - *Second line*: oral Cyclophosphamide, Cyclosporin. "immunosuppressive"

# 2- Focal Segmental GlomeruloSclerosis (FSGS)

- The primary variant on **light microscopy**:
  - Focal: some glomeruli are affected (the rest look normal).
  - Segmental: only a segment of the affected glomerulus is **sclerosed.** **"Same as MCD with Sclerosis on top of that".**
- A common cause of Nephrotic syndrome in **adults** (12 – 35 %) "Specially African American".

All glomeruli (the affected by sclerosis and not affected one) will have a diffuse foot processes effacement; like what is seen in MCD.

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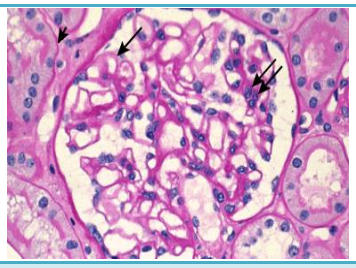
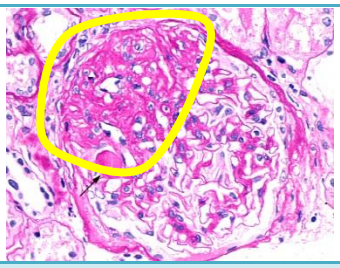
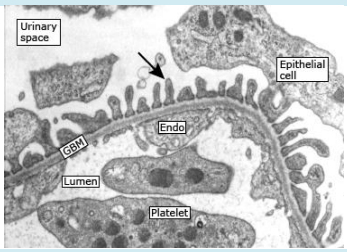
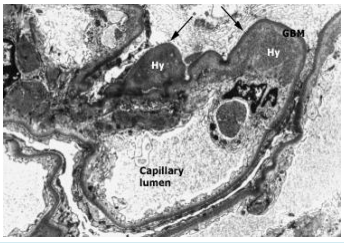
| **LM** |  |  |
| | Normal Glomerulus | FSGS |
| **EM** |   |   |
| | Normal Glomerulus | FSGS, like minimal change disease, diffuse foot process effacement but with segmental sclerosis |

- ***Types:***

| | ***Primary FSGS*** | ***Secondary FSGS*** |
|---|---|---|
| ***Can be*** | - Sudden onset of **heavy proteinuria** and other manifestations of nephrotic syndrome.<br>- Usually treated with corticosteroids and other immunosuppressive. | - Proteinuria is **less heavy** than other causes of nephrotic syndrome.<br>- Serum Albumin is not very low like the primary type<br>**- Renal impairment is commonly seen with the secondary FSGS and "not a good prognostic sign"** |
| ***Causes*** | | - Massive **obesity**<br>- **Nephron loss** ( > 75% of renal mass)<br>- **Reflux nephropathy** "they work more to compensate"<br>- Renal agenesis<br>- Healing of prior GN (IgA, Lupus)<br>- **Anabolic steroid abuse**<br>- Severe preeclampsia<br>- Drugs: Interferon, Pamidronate, **Heroin**<br>- Infections: **HIV** |
| ***Treatment*** | **Immunosuppressive therapy** is indicated in most patients with **primary FSGS:**<br>- First line: corticosteroids<br>- Second line: cyclosporine | Not typically treated with Immunosuppression, **treat** the **primary cause** and add supportive measures to protect the kidneys, e.g. keeping blood pressure well controlled. |

## 3- Membranous Nephropathy (MN):

- Most common cause of nephrotic syndrome in **adults** (15% and 33%).
- Mostly secondary in children (hepatitis B antigenemia). "secondary to infections"
- ***Presentation***: slowly developing nephrotic syndrome.

| | | |
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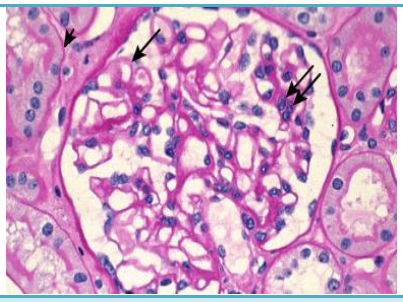
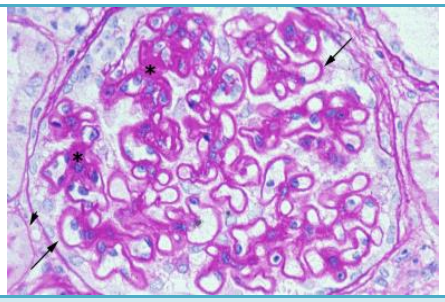
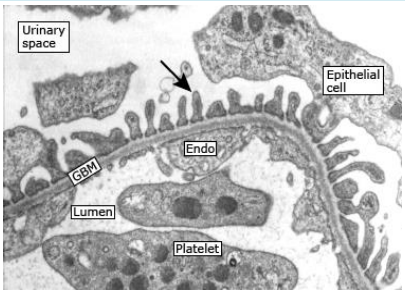
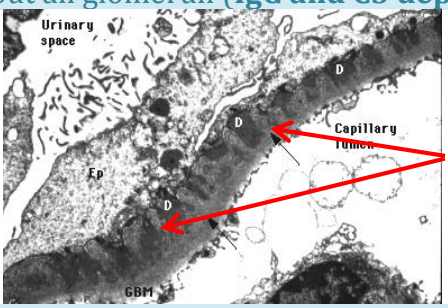
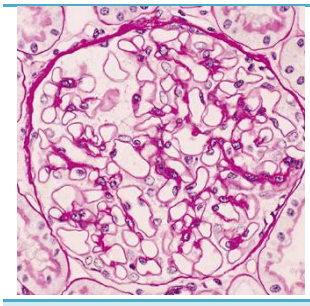
| **LM** |  |  |
| | Normal Glomerulus | Diffuse **thickening** of the glomerular capillary **wall** throughout all glomeruli (**IgG and C3 deposition**) |
| **EM** |  |  |
| | Normal Glomerulus | MN |

- ***Etiology:***

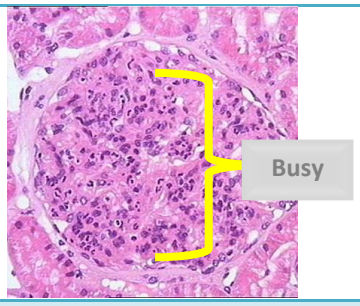
| | ***Primary*** | ***Secondary*** |
|---|---|---|
| ***Causes*** | (Idiopathic), approximately 75% of cases in **adults**. | - Systemic lupus erythematosus **(SLE)**, Class V Lupus Nephritis (10-20%). "deposition and loss of process"<br>- Drugs: penicillamine, gold, high dose Captopril, and NSAIDs, Anti-TNF<br>- Infections: Hepatitis B, Hepatitis C, syphilis<br>- Malignancy: **solid tumors** prostate, lung, or GI track |
| ***Treatment*** | Corticosteroids + (Cyclophosphamide or cyclosporine)<br><br>- May be Rituximab | Mainly target the **primary disease** that caused MN, and treat the Nephrotic syndrome manifestations |

# Nephritic Syndrome (NS):

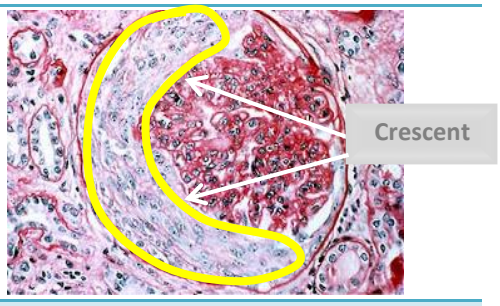
- When we say Nephritic; it means a clinical pattern of presentation for a group of GNs, and not a syndrome like what we saw in Nephrotic causes.
- The Nephritic pattern is always indicative of underlying **inflammatory process in the glomeruli**; causing inflammatory modulators attraction, cellular proliferation and eventually glomerular permanent dysfunction if left untreated.
- The **Glomerular mesangium, endothelium and GBM** components of the Glomerulus are likely going to be targeted because of their proximity to blood circulation.



**Normal Glomerulus**

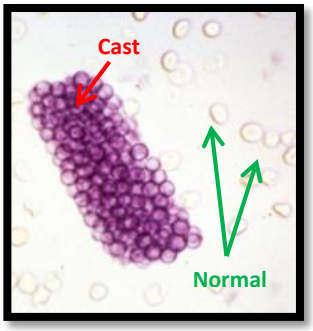



Glom. with proliferative (inflammatory) GN




Glom. with Crescent: Indicates **severe inflammation** & **worse outcome** if not treated rapidly

## *Nephritic urine analysis shows:*




- Red Blood Cells (RBCs)
- **RBCs casts** *, or cellular casts. "formed by naturally occurring ***Tamm-Horsfall mucoprotein*** *in the distal tubules & collecting ducts when they become loaded with RBCs coming from the Glomerulus (due to GN)."*
- **Dysmorphic RBCs** (RBCs lose their smooth surface).
- Protein (at variable amount).
- They are called **Active Urinary Sediments**. "Active = is indicative of underlying glomerular inflammatory process; requiring urgent medical attention".

## *Nephritic Clinical Manifestations:*

- **AKI** (Acute Kidney Injury) = Acute Renal impairment or Failure= elevated Creatinine.
- **Decreased Urine output.**
- **Edema.**
- **High Blood Pressure.** "always high in *nephritic*, normal or slightly high in *nephrotic*"
- May have other manifestations of systemic vasculitis since some GN types are actually vasculitis (e.g. skin rash, pulmonary hemorrhage, etc.).

- Positive immune markers: ANA, Anti-DNA, low complements, +ve ANCA (depends on the cause).

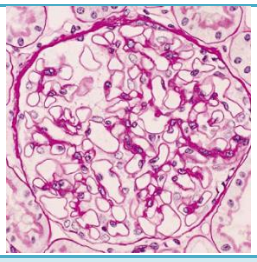
## Glomerulonephritis (GN):

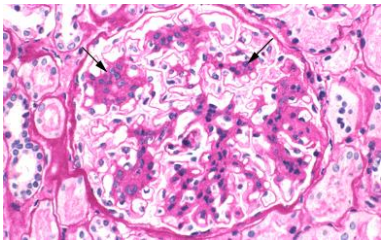
**Glomerulonephritis (GN): Renal Diseases that can present with Nephritic picture**

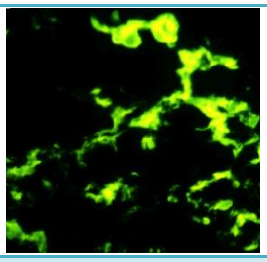
1- IgA Nephropathy / HSP (Henoch-Schönlein purpura)
2- Post streptococcal glomerulonephritis (PSGN)
3- Lupus Nephritis
4- Anti-GBM (Goodasture's disease)
5- ANCA vasculitis ( e.g. Wegner's Granulomatosis)
6- Membranoproliferative GN (MPGN)

### 1- IgA Nephropathy: (Henoch-Schönlein purpura)

- **Most common type** of Primary GN in developed countries.
- Can present as **dark urine 1-3 days after upper respiratory tract infection** (< one week of URT infection).
- A lot of times it gets picked up incidentally by finding abnormal urine analysis (Hematuria+/- Proteinuria) done for other reasons with no symptoms.
- It has a **chronic course** that can progress to ESRD.
- Needs **kidney biopsy** to reach the diagnosis.
- The diagnosis is made by finding abnormal deposition of IgA immunoglobulins in the Glomeruli, and that elicit a local inflammatory response in the Glom mesangium (mesangial expansion).
- It is thought to be secondary to altered mucosal immunity that leads to excessive IgA synthesis upon exposure to environmental antigens. And they eventually deposit in the Gloms may be because of altered structure.
- There is really no effective immunosuppressing therapy except in severe cases where it can be tried.
- Most important treatment is to **control the blood pressure** which decreases proteinuria.
- **HSP** (is a **systemic vasculitis** caused by immune deposition of IgA in different organs; typically skin, bowel and kidneys). (It causes Skin rash, joint pain abdominal pain and involvement of the kidney).


Normal Glomerulus on LM


IgA Nephropathy on LM


IgA IF

## 2- Post streptococcal glomerulonephritis (PSGN):

- Typically caused by throat infection with Gram-positive cocci (Streptococcus).
- But also can be caused by Staphylococcus soft tissue or bone infection in adults.
- Bacterial Antigen **cross react with Glom antigens**, or may be an **immune-complex (Antigen-antibody)** response that is responsible.
- Patients present with frank hematuria usually **after one week and up to 3 weeks** from the start of infection. "IgA nephropathy is within a week !"
- Serum will show positive Antistreptolysin (ASO) titer. Low C3, Normal C4. May have positive throat culture.
- **Children have better and faster recovery than adults**.
- Treatment is usually supportive= wait and see.

## 3- Lupus Nephritis:

- LUPUS: The Disease with a **Thousand Faces.**
- Kidneys can be affected by SLE like other organs.
- The degree of involvement can be mild (or even not visible to the physician) to a very severe one causing ESRD in few months.
- Most important in dealing with these cases is having high suspicion of its presence and to start immediate workup & referral for diagnosis and treatment.
- **Kidney biopsy** is mandatory to make the diagnosis.
- **Low complements (C3, C4) level** in the blood "they get deposited in the kidney" along with the positive Lupus markers, abnormal urine analysis & abnormal renal function should make you think of its presence.
- Lupus Nephritis treatment depends on the findings in renal biopsy.
- It usually involves high degree of immunosuppressing medications.

## 4- ANCA vasculitis: "Anti-neutrophil cytoplasmic antibody"

- Autoimmune disease that involves the presence of Neutrophils adhesion enhancing molecule.
- ***Two types of ANCA:***

  1- **C-ANCA**= **C**ytoplasmic type, more commonly causing Granulomatous Polyangiitis = old name Wegner's Granulomatosis. "A granuloma forming disease".

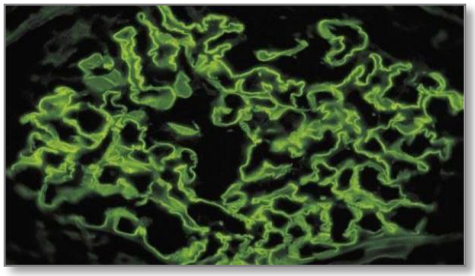
  2- **P-ANCA**= **P**erinuclear type, more commonly associated with Microscopic Polyangiitis & Churg-Strauss syndrome.

  Angiitis: means small vessels vasculitis

- **Upper airways and lung involvement** is common and patients can present with renal and pulmonary manifestations (GN+Pulmonary hemorrhage: hemoptysis).

- ***Diagnosis*** is made by **kidney biopsy and positive ANCA titer in the serum**.
- It is usually an aggressive disease that should be treated with potent immunosuppressing medications.

## 5- *Anti-GBM antibody disease:*



**Linear** **Anti-GBM** staining by Immunofluorescence is a *Diagnostic test.* ***Linear*** *means taking the same shape of smooth capillary walls.*

- Due to **autoantibody against (alpha-3 chain) of type IV Collagen**; found in Glomerular & alveolar basement membrane.
- ***The manifestations will be:***
  - GN (can be the only presenting finding).
  - **Pulmonary hemorrhage** causing hemoptysis (if with GN; it is called: **Goodpasture's disease**). "With smoking"
  - Positive test for Anti-GBM antibodies in the serum.
  - Kidney biopsy shows the diagnostic Immunofluorescence pattern: **Linear stain of IgG and C3**.
- ***Treatment*** is always started immediately to remove the antibodies by **Plasmapheresis** (a process of removing the plasma from the blood which has the autoantibodies), and also preventing further antibodies production by giving **heavy immunosuppression** that includes corticosteroids and cyclophosphamide.

## 6- *Membranoproliferative GN (MPGN):*

- It is a pathological description & has multiple causes.
- It may present with Nephritic picture or Nephrotic syndrome.
- The primary (idiopathic) MPGN is mainly seen in children.
- The secondary type is seen in adults due to:
  - **Hepatitis B and C** "always think about it when the patient has renal manifestations"
  - Endocarditis
  - Lupus and Sjogren's syndrome
  - Cancer
  - Complement deficiency

# Summary

- Primary glomerular diseases mostly caused by immune system dysfunction.
- If Podocytes are the main target >> proteinuria >> Nephrotic.
- If endothelial cells, GBM or mesangial cells are affected >> hematuria and abnormal renal function >> Nephritic.
- Almost always a **kidney biopsy** is needed to diagnose a suspected primary glomerular disease.
- **Important Clinical Presentation of Nephrotic syndrome**: Generalized Edema, Frothy urine, Weight gain due to fluid retention.
- **Nephritic urine analysis shows:** RBCs casts, Dysmorphic RBCs.
- **Nephritic Clinical Manifestations:** AKI (Acute Kidney Injury) = elevated Creatinine, Decreased Urine output, Edema, High Blood Pressure. "always high in nephritic, normal or slightly high in nephrotic".
- **Glomerulonephritis (GN):** Renal Diseases that can present with nephritic picture.

| | Disease | Important Note |
|---|---|---|
| **Nephrotic** | 1- Minimal Change Disease (*MCD*) | • Electron microscopy shows **diffuse effacement** of the epithelial cells' foot processes.<br>• The most important difference between MCD and the **FSGS is the presence of glomerular sclerosis in FSGS.**<br>• It is the Main Cause of Nephrotic syndrome **in children**.<br>• Corticosteroid responsive in > 90%. |
| | 2- Focal Segmental GlomeruloSclerosis (*FSGS*) | • **Focal**: some glomeruli are affected.<br>• **Segmental**: only a segment of the affected glomerulus is sclerosed.<br>• Renal impairment is commonly seen with the secondary FSGS and "not a good prognostic sign" |
| | 3- Membranous Nephropathy (*MN*) | • **Most common cause of nephrotic syndrome in adults** (15% and 33%).<br>• Mostly secondary in children (hepatitis B antigenemia).<br>• Systemic lupus erythematosus (SLE) is an important Secondary etiology**.** |
| **Nephritic** | 1- IgA Nephropathy / HSP (Henoch-Schönlein purpura) | • Most common type of Primary GN in developed countries.<br>• Can present as dark urine 1-3 days after upper respiratory tract infection (< one week of URT infection).<br>• No symptoms. |

| | |
|---|---|
| | • Most important treatment is to control the blood pressure which decreases proteinuria. |
| 2- Anti-GBM (Good pasture's disease) | • Due to autoantibody against (alpha-3 chain) of type IV Collagen.<br>• Pulmonary hemorrhage causing hemoptysis (if with GN; it is called: Goodpasture's disease). "With smoking"<br>• Treatment: Plasmapheresis and heavy immunosuppression. |
| 3- Post streptococcal glomerulonephritis (PSGN) | • Typically caused by throat infection with Gram-positive cocci (Streptococcus).<br>• Patients present with frank hematuria usually after one week and up to 3 weeks from the start of infection |
| 4- ANCA vasculitis ( e.g. Wegner's Granulomatosis) | • Two types of ANCA:<br>1. **C-ANCA= Cytoplasmic type**, more commonly causing **Granulomatous Polyangiitis = old name Wegner's Granulomatosis.** "a granuloma forming disease".<br>2. **P-ANCA= Perinuclear type,** more commonly associated with **Microscopic Polyangiitis & Churg-Strauss syndrome**.<br>• Upper airways and lung involvement is common**.** |
| 5- Lupus Nephritis | • Low complements (C3, C4) level in the blood "they get deposited in the kidney" along with the positive Lupus markers, abnormal urine analysis & abnormal renal function should make you think of its presence. |
| 6- Membranoproliferative GN (MPGN) | • It may present with nephritic picture or nephrotic syndrome.<br>• Hepatitis B and C can cause secondary MPGN. |

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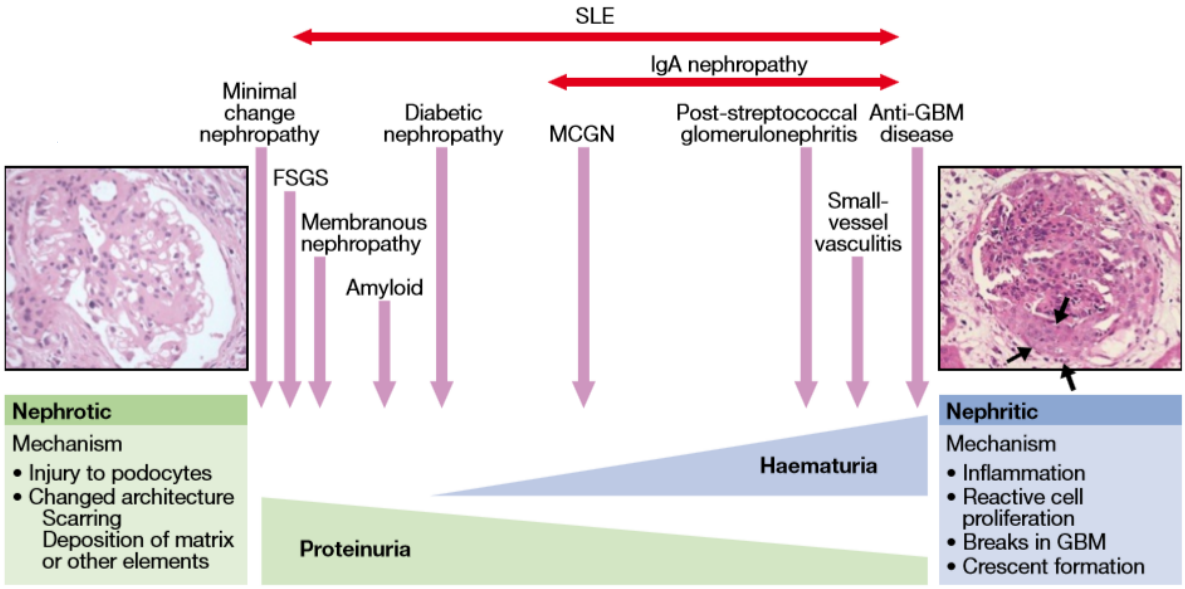


**Fig. 17.27 Spectrum of glomerular diseases.** At one extreme, specific injury to podocytes, or structural alteration of the glomerulus affecting podocyte function (for example, by scarring or deposition of excess matrix or other material), causes proteinuria and nephrotic syndrome (see Box 17.19, p. 479). The histology to the left shows diabetic nephropathy. At the other end of the spectrum, inflammation leads to cell damage and proliferation, breaks form in the GBM and blood leaks into urine. In its extreme form, with acute sodium retention and hypertension, such disease is labelled nephritic syndrome (FSGS = focal and segmental glomerulosclerosis; MCGN = mesangiocapillary glomerulonephritis). The histology to the right shows a glomerulus with many extra nuclei from proliferating intrinsic cells and influx of inflammatory cells shows crescent formation (arrows) in response to severe post-infectious glomerulonephritis.

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**TABLE 7-5 Nephritic Versus Nephrotic Syndrome**

| | Nephritic Syndrome | Nephrotic Syndrome |
|---|---|---|
| **Pathogenesis** | Inflammation of glomeruli due to any of the causes of glomerulonephritis | Abnormal glomerular permeability due to a number of conditions |
| **Causes** | Poststreptococcal glomerulonephritis is the most common cause, but may be due to any of the causes of glomerulonephritis | Many conditions. Membranous glomerulonephritis is the most common cause in adults. Other causes include diabetes, SLE, drugs, infection, glomerulonephritis (focal segmental and others)<br>Minimal change disease is the most common cause in children |
| **Laboratory findings** | Hematuria<br>AKI—azotemia, oliguria<br>Proteinuria, if present, is mild and not in nephrotic range | Urine protein excretion rate >3.5 g/24 hr<br>Hypoalbuminemia<br>Hyperlipidemia, fatty casts in urine |
| **Clinical findings** | HTN<br>Edema | Edema<br>Hypercoagulable state<br>Increased risk of infection |

# Questions

1) Which glomerular disease would you suspect most in a patient with the following findings? Anti-GBM antibodies, hematuria, hemoptysis:
   a) Post streptococcal glomerulonephritis (PSGN)
   b) Lupus nephritis
   c) Wegner's Granulomatosis
   d) Goodpasture syndrome

2) Which of the following is the most common nephrotic syndrome in children?
   a) Focal segmental glomeulosclerosi
   b) Minimal change disease
   c) Henoch-Schonlein purpura
   d) Membranous glomerulonephritis

3) Which glomerular disease would you suspect most in a patient with the following findings? Nephrotic syndrome associated with hepatitis B:
   a) Membranoproliferative glomerulonephritis.
   b) Lupus nephritis
   c) Henoch-Schonlein purpura
   d) Goodpasture syndrome

4) Which glomerular disease would you suspect most in a patient with the following findings? Nephrotic syndrome associated with HIV:
   a) Membranoproliferative glomerulonephritis.
   b) Focal segmental glomeulosclerosis
   c) Post streptococcal glomerulonephritis (PSGN)
   d) Membranous glomerulonephritis

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**Answers:**

1. **D**
2. **B**
3. **A**
4. **B**