PATHOLOGY TEAM
439

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1957

Nephrotic/ Nephritic Syndrome

Nephrotic syndrome-pathoma
Nephritic syndrome- pathoma
Saudi nephrologist explained these syndrome perfectly,you have time so waste it in a good thing🤪=البيديا لعبتي

Objectives:

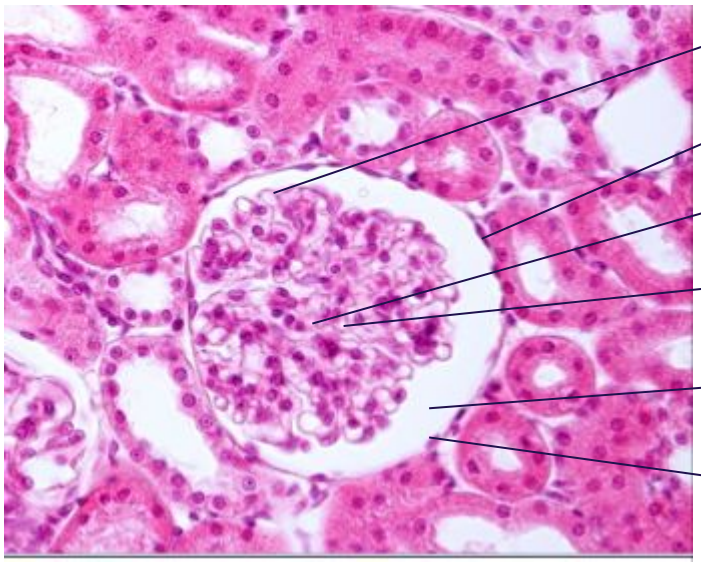
At the end of the activity (2 lectures) the students will be able to:

- Recognize the five major renal glomerular syndromes.
- Describe the main differential pathological diagnosis for each syndrome.
- Perform a clinico-pathological correlation.
- Describe the patterns of injury of each syndrome.

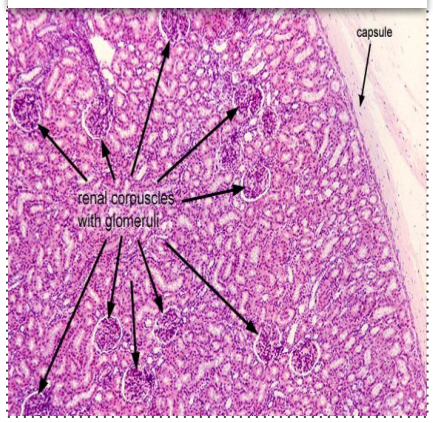
Index:
Important
NOTES
Extra Information

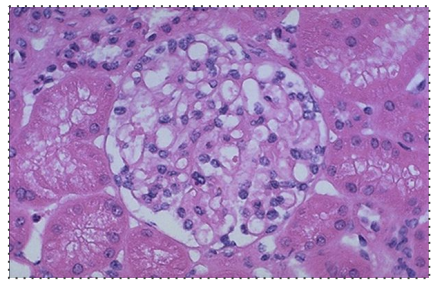
Histology of kidney

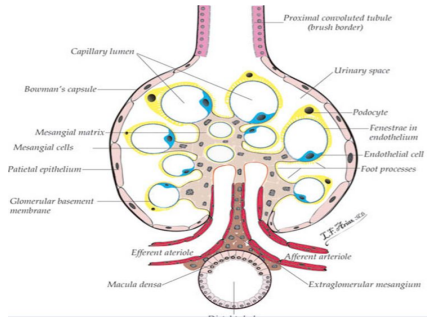
Normal Glomerulus:



High power showing the capillary loops and glomerular basement membrane clearly



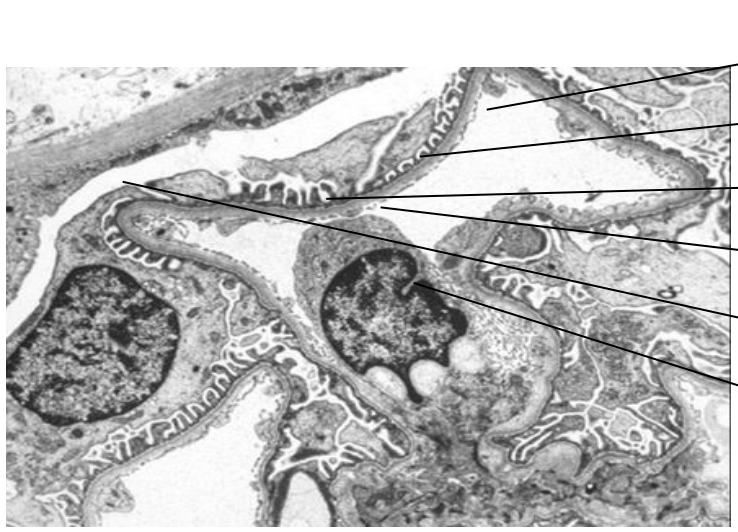


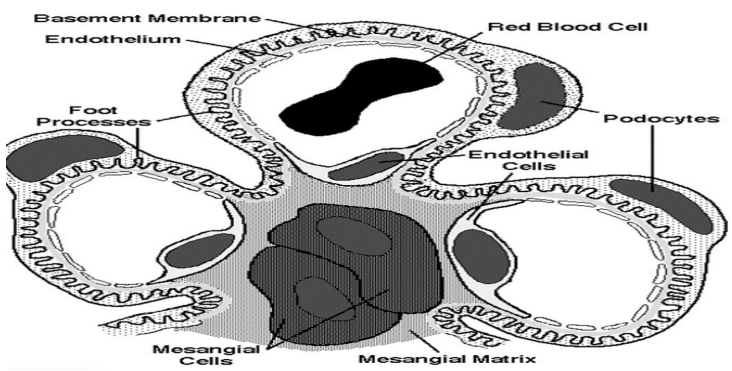


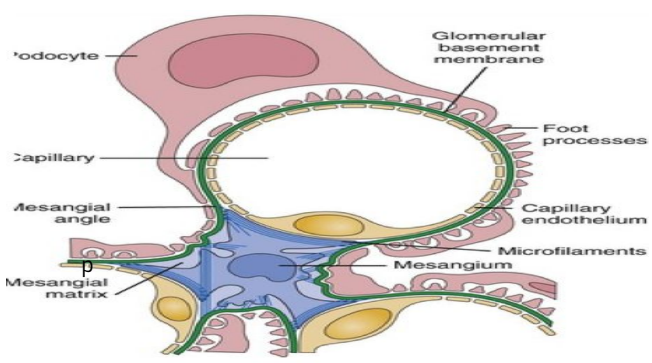
Note That:

- The glomerular basement membrane (GBM) is a crucial structure formed by podocytes of epithelial cells (mainly) and podocytes of endothelial cells as well
- Podocytes lie outside the GBM with their foot processes
- The mesangium (mesangial cells) act like a glue to hold the structure together
- In cases of pathology these structures will be effaced or damaged and the podocyte or foot processes will not be seen, instead a thicker membrane of damaged podocytes is seen.
- In the capillary loops lies blood which contains plasma, proteins, RBCs and inflammatory cells.

Glomerulus on Transmission Electron Microscope

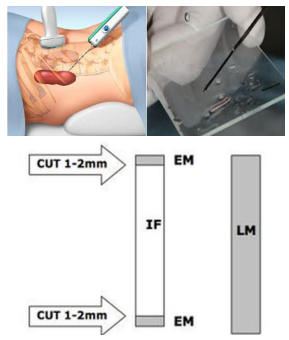






Kidney Biopsy

A biopsy of the kidney is taken on ultras guidance by passing a tube through the back and extracting a renal tissue.



Then, the sample is divided into 3 parts allowing us to collectively reach a diagnosis:

1- Light microscope (using silver stain),

2- Immunofluorescence

3- Electron microscope.

1

Light Microscopy (LM)
To study renal histology.

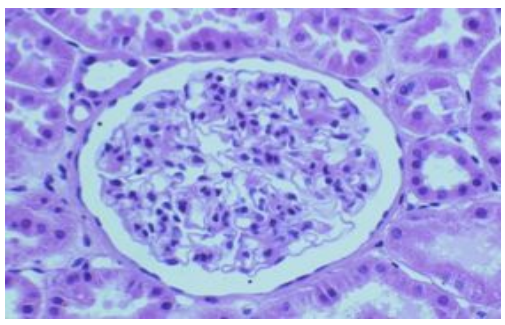
2

Immunofluorescence (IF)
The study is used to detect for The presence of immunoglobulins (IgA, IgG, IgM) and complements (C3 and C1q) in the glomeruli (in the mesangium or in the glomerular capillary loop wall.

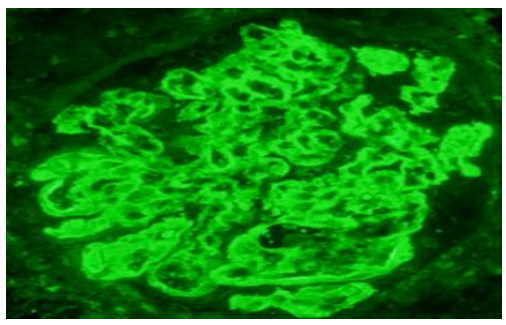
3

Electron Microscopy (EM)
The study is used to detect for the presence or absence of:
1) Effacement of the epithelial cell (**podocytes**) foot processes.
2) Electron dense **immune complex deposits** (Antigen-antibody complexes which settle down in the glomerulus)
3) And if **deposits** are present then it's used to identify the location of the deposits in the glomeruli (mesangial/paramesangial, subepithelial, subendothelial)

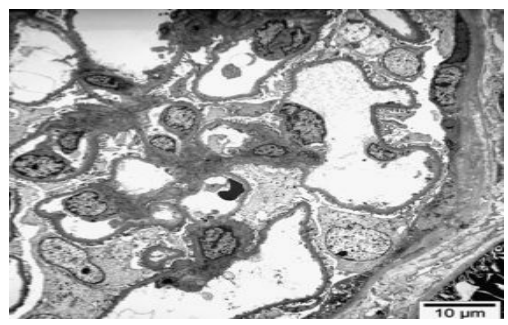
Light Microscopy (Normal state)



Immunofluorescence

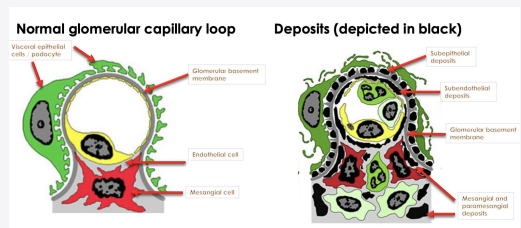


Electron Microscopy



Note that:

- Normally the glomerulus shouldn't have immunoglobulins, which are only present in pathologies and each disease leads to the release of certain Immunoglobulins and complements which are characteristic to it and which will fluorescence in the IF test, and so based on the combination Igs and complements seen in the IF test the disease causing them can be identified.
- The effacement of the epithelial cell (podocytes) foot processes Is only distinguished in electron microscopy.
- Mesangial: deposits that lie in the mesangium.
- Paramesangium: deposits that lie close to the loops in the mesangium.
- Subepithelial: deposits **outside** the glomerular basement membrane.
- Subendothelial: deposits **inside** the glomerular basement membrane.
- For the normal state the epithelial cells, endothelial cells and mesangial cells will be apparent on the light microscopic image.

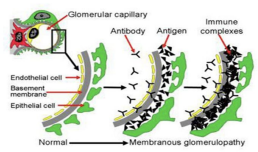
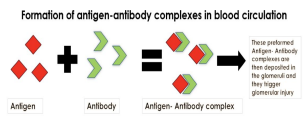
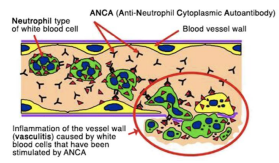


Pathogenesis of glomerular injury

Glomerulonephritis (GN) is frequently caused by immunologic mechanisms. Both antibody-mediated (**mainly**) and cell-mediated types of immunity play roles in the production of glomerular inflammation.

1. Antibody-mediated immune GN

There are 3 major mechanisms of antibody-mediated inflammation in most forms of GN, all 3 initiate glomerular inflammatory injury by the attraction and activation of leukocytes. These are:

In situ (formed at site) **immune complex formation**	Certain circulating antibodies react with certain antigens that are already present in glomeruli leading to the formation of immune complexes in the glomeruli. These deposits then attract leukocytes and activate complement leading to a glomerular injury	
Deposition of pre-formed circulating immune complexes in the glomeruli	Antigen-antibody reaction take place in circulation and these preformed circulating immune complexes are deposited in the glomeruli, these deposits trigger glomerular injury (by attracting inflammatory cells and activate complements). (the antigen can be a bacteria, virus etc.)	
Antineutrophil cytoplasmic autoantibodies (ANCAs)	they cause a severe type of GN in which the patients have circulating autoantibodies against antigens present in neutrophil cytoplasm. This interaction leads to activation and adhesion of the neutrophils to endothelial cells lining the capillaries especially the glomerular capillaries. The neutrophils release injurious products that promote endothelial injury, vascular inflammation (vasculitis) and GN.	

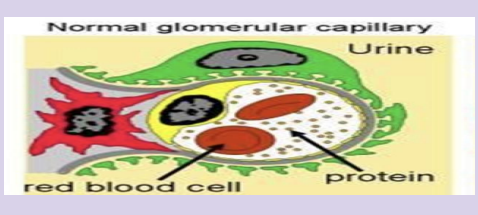
2. Cell mediated immune GN:

Sensitized T cells can also cause glomerular injury. (Less common)

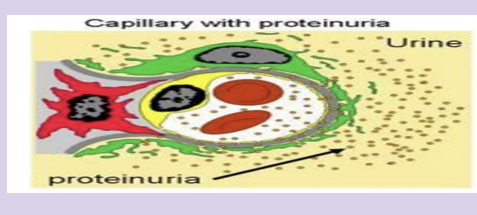
Common markers of renal diseases

Markers

- **In the blood**
  - **BUN**: blood-urea- nitrogen amount of nitrogen in your blood that comes from the urea.
  - **Serum creatinine**
- **In the urine**
  - **Hematuria**: rbcs in urine (smoky brown urine). as a result of glomerular injury (not specific) and rupture of the glomerular capillaries with resultant bleeding into the Bowman's/ urinary space. It could be microscopic.
  - **Proteinuria**: abnormal quantities of protein in the urine
  - **Casts**: rbcs mix with proteinaceous material in the tubules and forms rbc casts (which are found in the urine).



Capillary loop in a normal glomerulus retains the RBCs , wbcs and most of the protein in the blood that is passing through and the ultrafiltrate is mainly watery fluid going into the urinary space



Protein molecules spill out into the urinary space due to filtration abnormality in the glomerular capillary wall

In certain types of glomerular injury the injured capillary loop has abnormal permeability and ruptured GBM which allows rbcs (hematuria) and proteins (proteinuria) to leak into the urinary space. these RBCs in the tubule might mix with other materials to form casts

Clinical manifestations of kidney disease

Nephritic syndrome	Results from glomerular injury leading to acute onset of hematuria (rbcs in urine), mild to moderate proteinuria, azotemia, edema & hypertension.
Nephrotic syndrome	heavy proteinuria (excretion of more than 3.5 g of protein/day in urine), hypoalbuminemia, severe edema, hyperlipidemia, and lipiduria.
Asymptomatic hematuria &/or non-nephrotic proteinuria	A sign of mild glomerular abnormalities e.g. IgA nephropathy.
Rapidly progressive glomerulonephritis	Results from severe glomerular injury leading to loss of renal function within days or weeks → hematuria, dysmorphic rbcs, rbc casts in urine, mild to moderate proteinuria.
Acute kidney injury	oliguria or anuria with recent onset of azotemia; can result from glomerular injury (e.g. crescentic glomerulonephritis), interstitial injury, vascular injury (e.g. TMA) or acute tubular injury/necrosis.
Chronic kidney disease	Any chronic renal diseases that progresses to end stage kidney requiring dialysis and transplantation
Urinary tract infection	Affect the kidney (pyelonephritis) or the bladder (cystitis) → bacteriuria and pyuria (bacteria and leukocytes in urine).
Nephrolithiasis (renal stones)	Renal colic, hematuria (without rbc casts).

Terminology

1. **DIFFUSE**: majority of the glomeruli are involved. (more than 50%)
2. **SEGMENTAL**: only part of a glomerular tuft is involved
3. **FOCAL**: some of the glomeruli are involved
4. **GLOBAL**: involving the total glomerular tuft

NEPHROTIC SYNDROME

A group of clinical features* that include the following:

01 **Massive proteinuria:**
is the loss in the urine of >3.5 g of protein/day.
This is due abnormal permeability of the glomerular capillary wall.

02 **Hypoproteinemia or hypoalbuminemia:**
plasma albumin levels <3g/dL (this due to the loss of protein in the urine)

03 **Odema**:
Hypoproteinemia causes reduced plasma colloid osmotic pressure, salt water retention & odema.

04 **Hyperlipidemia and lipiduria:**
hypoalbuminemia causes compensatory increase in lipoprotein secretion by the liver leading to hyperlipidemia. At the same time there is increased loss of lipid in the urine, lipiduria (due to abnormal permeability of the GBM).

Note: in the beginning there is little or no azotemia, hematuria, or hypertension (not part of definition of nephrotic syndrome).

Causes of nephrotic syndrome

PRIMARY CAUSES	SECONDARY CAUSES
• Minimal change disease (well explained in the next slide) • Membranous GN • Focal segmental glomerulosclerosis (FSGS) • Membranoproliferative GN (can also present as nephritic syndrome) • Others	• Diabetes mellitus (most common systemic causes) • Amyloidosis • Systemic lupus erythematosus (it can also present as nephritic syndrome) • Drugs (gold, penicillamine, "street heroin") • Others

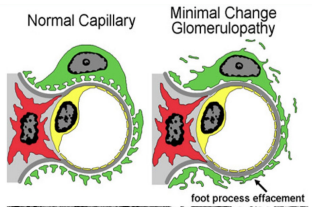
NOTE: In children the most common cause of nephrotic syndrome is minimal-change disease. In adults the most common primary glomerular diseases that causes nephrotic syndrome are membranous glomerulopathy in Caucasians and Asians, and FSGS is the most common etiology in American blacks.

Nephrotic Syndrome

Minimal change disease / glomerulopathy

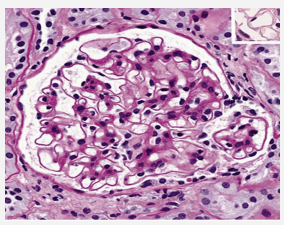
Definition

- Minimal change disease (MCD) is also known as lipoid nephrosis.
- It causes nephrotic syndrome.
- It is characterized by effacement of the foot processes of the visceral epithelial cells (podocytes).



Morphology

Light microscopy

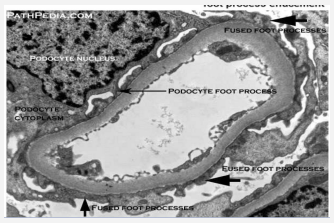


- Glomeruli : look **normal**.
- The proximal convoluted tubular cells are heavily laden with protein droplets and lipids.
- There is no tubular atrophy or interstitial fibrosis.

Immunofluorescence (IF)

- **Negative** for immunoglobulins and complement.

Electron microscopy(EM)



- MCD is **characterized by diffuse fusion or effacement** (loss of face, it's a sign of proteinuria) **of the epithelial cell (podocyte) foot processes.**
- This effacement is due to the retraction of the foot processes as a result of extensive cell swelling.

NOTE: effacement occurs in virtually all cases of nephrotic proteinuria; it is not specific for minimal-change glomerulopathy. Therefore to make an accurate diagnosis we have to combine the different microscopy techniques (LM & IF are normal, whereas EM is abnormal).

Pathogenesis

Unknown, usually idiopathic.

Clinical features

Nephrotic syndrome

Treatment and Prognosis

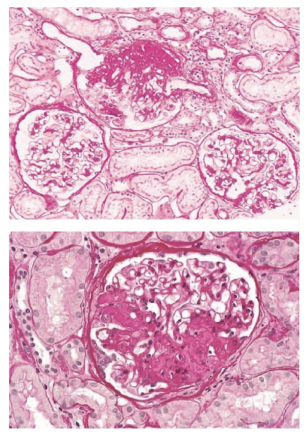
- Over 90% of children and few adults have complete remission with corticosteroid therapy.
- Prognosis: good, especially in children.
- Some patients become steroid dependent i.e. they relapse each time corticosteroid treatment is stopped. Causing incomplete remission, they have to take steroids or they'd relapse.
- Less than 5% develop chronic renal failure

Nephrotic Syndrome

Focal And Segmental Glomerulosclerosis (FSGS)

- Occurs in older children and adults.
- It is characterized by sclerosis of some (but not all) glomeruli that involves only a part of each affected glomerulus.
- It is important to distinguish FSGS from minimal-change disease. Both are associated with nephrotic syndrome, but the incidence of hematuria and hypertension is higher in individuals with FSGS.

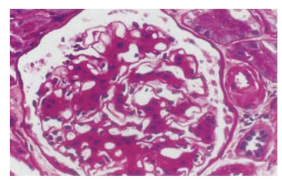
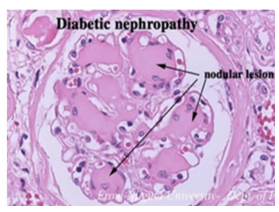
Morphology

Light microscopy (LM)	1- Glomeruli at the corticomedullary junction are commonly affected. 2- Only some of the glomeruli are involved (i.e. focal). 3- There is focal and segmental sclerosis (fibrosis) of the glomeruli (i.e. some glomeruli show sclerosis in a segment of the glomerular tuft). 4-Adhesions and hyalinosis +/- 
Immunofluorescence (IF)	Usually negative. Sometimes **IgM** is positive.
Electron microscopy (EM)	There is patchy/focal effacement (unlike MCD which had diffuse effacement) of podocyte foot processes.

Diabetic Nephropathy (DM)

- Long standing poorly controlled DM leads to kidney disease.
- A common cause of secondary nephrotic syndrome.

Morphology

Light microscopy (LM) LM shows 2 types of lesions: Both show diffuse thickening of the glomerular basement membrane	1- **Diffuse glomerulosclerosis:** all the glomeruli show increase in mesangial matrix ending in sclerosis. 
	2- **Nodular glomerulosclerosis (Kimmelstiel Wilson nodules)**: there are nodules in the mesangium. These nodules are spherical and eosinophilic, with a central acellular area. It is pathognomonic of diabetic nephropathy. 
Immunofluorescence (IF)	Negative
Electron microscopy (EM)	There is diffuse increase in the thickness of the glomerular basement membrane without deposition.

Nephrotic Syndrome

Membranous glomerulopathy/ glomerulonephritis (GN) nephropathy

Etiology

- **Primary / idiopathic** — About 80% of cases.
  - in primary MN there are autoantibodies **against** phospholipase A2 receptor 1 (**PLA2R1***) antigen. (***MCQs**)
- **Secondary** — Should be investigated for secondary causes
  and treat the underlying cause
  1. Autoimmune disease (SLE)
  2. infectious disease (hepatitis B)
  3. Therapeutic agents (penicillamine)
  4. Neoplasms (lung cancer)

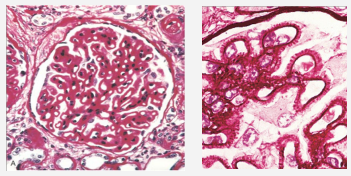
Definition

- It is an immune complex disease. The antigen-antibody immune complexes are formed either in situ in the glomeruli or are performed in circulation and then deposited in the glomeruli.

- It is characterized by **deposition of immune complexes** in the subepithelial area in the glomeruli (between the podocytes and the GBM).

Morphology

Light microscopy

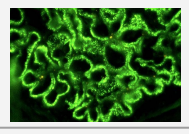


- The capillary walls of the glomeruli are diffusely thickened (due to the subepithelial deposits seen on EM).

- In silver stain: The deposits are separated from each other by protrusions of GBM matrix called spikes (right picture: lines sticking out are spikes, whereas dark spots are immune deposits).

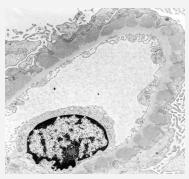
- As the disease progresses (slowly) there is glomerular sclerosis and interstitial fibrosis.

Immunofluorescence (IF)



- Granular positivity of immunoglobulin **IgG** and complement **C3** along the GBM.

Electron microscopy(EM)



- The immune complex appear in capillary walls as electron-dense deposits in the subepithelial space.

- There is diffuse effacement of epithelial cell foot process also.

Clinical features

1. A frequent cause of Nephrotic syndrome
2. Commonly 30 to 50 years of age
3. Proteinuria persists in about half the patients
4. The proteinuria does not usually respond to corticosteroid therapy
5. Some case progress to renal failure

Treatment and Prognosis

- It is a slowly progressive disease.
- If not treated → fibrosis of the kidneys (glomerular sclerosis, atrophy of tubules and interstitial fibrosis) and end stage disease/ renal failure.
- 10% to 30% have a more benign course with good prognosis.

NEPHRITIC SYNDROME

- Nephritic syndrome is a clinical complex caused by inflammatory lesions of glomeruli characterized by acute onset of:

1 **Hematuria**

(smoky brown urine)due to oxidation of hemoglobin to methemoglobin as a result of glomerular injury and inflammatory rupture of the glomerular capillaries with resultant bleeding into the Bowman's space. The rbcs pass into the tubules and mix with proteinaceous material in the tubules and forms rbc casts which are passed in the urine. The hemodynamic changes caused by the rupture lead to a reduction in the glomerular filtration rate (GFR.)

1 **Oliguria**

is a result of the reduced GFR.

1 **Azotemia**

increased blood urea nitrogen and creatinine. as a result of the reduced GFR.

1 **Hypertension**

it is a result of the fluid retention and renin release from the ischemic kidneys.

★ NOTE: There may be mild proteinuria and edema.

Examples of nephritic syndrome include:

Post-infectious glomerulonephritis:

it is the most classical example.

Lupus nephritis:

can also present as nephrotic syndrome

Nephritic Syndrome

Post-infectious glomerulonephritis (PIGN)

Overview

- It is a type of acute diffuse proliferative GN. caused by glomerular deposition of immune complexes resulting in proliferation of and damage to glomerular cells and infiltration of leukocytes, especially neutrophils.
- Usually there is a latent period (pathogenesis takes place) between the exposure and the occurrence of glomerulonephritis.
- Classically PIGN develops in children 1 to 4 weeks after streptococcal infection of:
  1. the pharynx (pharyngitis)
  2. tonsils (tonsillitis)
  3. the skin (impetigo or infected insect bite).

Etiology

- It is caused by deposition of immune complexes in glomeruli. (just like membranous GN)
  (it is not clear if immune complexes are formed mainly in the circulation or in situ)
- The most common cause of post-infectious glomerulonephritis is infection with group A, beta-hemolytic streptococci and is therefore also called poststreptococcal glomerulonephritis
- Other infections include:
  1. pneumococcal and staphylococcal infections
  2. viral diseases (mumps, measles, chickenpox).

Epidemiology

This disease was more common in the past because now we have antibiotics to prevent immune mediated response to take a place, but it is still one of the common childhood renal diseases.

Pathogenesis

01 The immune complexes are deposited in the glomeruli in the subepithelial area at outer surface of glomerular BM and are composed of **IgG** and **C3** appear in IF

02 The deposits initiate inflammation by:

I. activating complements: this way the complements get used up leading to development of hypocomplementemia (serum C3 levels are low during the acute phase of disease).
II. activating various other inflammatory mediators

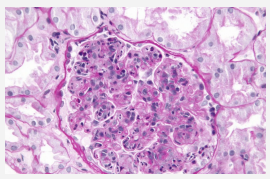
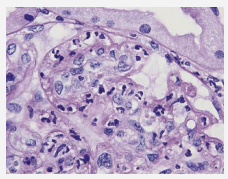
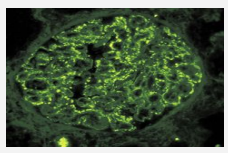
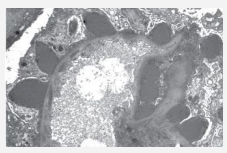
03 All these inflammatory mediators:

I. attracts and activate neutrophils and monocytes
II. stimulates proliferation of mesangial and endothelial cell proliferation.

04 marked glomerular **hypercellularity** the presence of an abnormal excess of cells **result in diffuse proliferative glomerulonephritis .**

Nephritic Syndrome

Post-infectious glomerulonephritis (PIGN), contd..

Morphology	**Light Microscopy**  	- There is diffuse enlargement and **hypercellularity** of glomeruli due to : 1. Proliferation of endothelial cells and mesangial cells 2. Infiltration of neutrophils and monocytes. - Crescents proliferation within the urinary space & Glomerular necrosis in response to the severe injury maybe present. - All histologic changes resolve completely in most patients after several months.
	Immunofluorescence 	- Coarse granular **lumpy bumpy** - IgG and C3 are positive along the capillary walls
	Electron Microscopy 	- Characteristic subepithelial electron dense immune deposits. - They look like dome shaped humps called **subepithelial humps** - The deposits clear up and disappear in a few months.
	Grossly	- Multiple punctate hemorrhagic spots on the kidney surface.
Clinical features	❖ The onset of kidney disease is sudden. ❖ Present as nephritic syndrome (oliguria, hematuria, hypertension and azotemia). ❖ Serum C3 levels are low during the acute phase. why? because it has been used up.	
Diagnosis	❖ Depends on serologic evidence of a rise in antibody titers to streptococcal products e.g. ASO Anti-Streptolysin an antibody targeted against streptolysin O, a toxic enzyme produced by group A Streptococcus bacteria titre is positive. ❖ bacterial culture will be negative (ASO) will be the only evidence.	
Prognosis	❖ Resolves in over 90% of patients. ❖ Rarely patients (usually adults) develop progressive renal failure.	

Nephritic Syndrome

Lupus nephropathy / nephritis (LN)

- Patients with the systemic lupus erythematosus (SLE) autoimmune disease, tend to have renal involvement and it is known as lupus nephritis (LN)
- LN is an immune complex mediated disease in which there is the deposition of antigen antibody complexes in the glomeruli
- It can present as **nephrotic or nephritic** syndrome
- LN can be active or chronic or a combination of both.
- The LN lesions have been classified into 6 classes by the International Society of Nephrology/Renal Pathology Society (ISN/RPS). This classification helps give information regarding the activity, chronicity and the prognosis of the disease.

LN Pathogenesis:

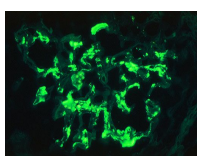
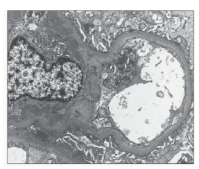
Deposition of antigen antibody complexes in the glomeruli.

The deposits trigger an inflammatory response

which triggers Proliferation of the epithelial, endothelial and mesangial cells of the glomeruli

It can even lead to glomerular necrosis.

Hematuria

Overview	❖ IgA nephropathy (IgAN) is one of the most common type of primary glomerulonephritis that presents as hematuria ❖ When it occurs in combination with vasculitis (leukocytoclastic vasculitis) and multiorgan involvement then is referred to as **Henoch-Schonlein purpura**. *leukocytoclastic: neutrophils broken into pieces found in vessel
Etiology	Characterized by the deposition of IgA immunoglobulin in the mesangium/ paramesangium of glomeruli.
Clinical Features	1. Usually present as hematuria only **2. Sometimes as nephritic syndrome**
Morphology	**LM:** is very variable may or may not show a mesangial hypercellularity, endocapillary hypercellularity, glomerular sclerosis, tubulointerstitial scarring. **IF:** dominant **IgA** stain positivity in the mesangium.  **EM:** immune complex deposits positive in the mesangium and paramesangial area. 

Summary

	Disease	LM	EM	IF
Nephrotic Syndrome	**Minimal change disease**	Normal	Effacement of foot process	Negative
	(FSGS)	Focal & Segmental sclerosis of the glomeruli	**Patchy** effacement of foot process	Negative
	(DM)	Diffuse thickening of GBM: **1. Diffuse glomerulosclerosis:** increase mesangial matrix & mesangial cell proliferation. **2. Nodular glomerulosclerosis** (Kimmelstiel Wilson nodules): Spherical & eosinophilic, with a central acellular area nodules in the mesangium.	Diffuse increase in the thickness of the glomerular basement membrane	Negative
	Membranous glomerulopathy (GN)	- Walls of glomeruli are diffusely **thickened**. - **Spikes** between the deposits & GBM matrix	- Electron-dense deposits in the subepithelial space. - Effacement of foot process.	**Granular** positivity of IgG & C3
Nephritic Syndrome	**PIGN**	- Diffuse enlargement and **hypercellularity** of glomeruli. - Also, Crescents & necrosis.	**Subepithelial humps**	- Coarse granular **lumpy bumpy.** **-** IgG and C3 along the capillary walls.
	LN	Deposition of antigen antibody complexes in the glomeruli		
	Hematuria	Variable; might show: - Mesangial hypercellularity, - Endocapillary hypercellularity, - Glomerular sclerosis, - Tubulointerstitial scarring.	Immune complex deposits in the mesangium and paramesangial area.	Dominant **IgA** in the mesangium.

Quiz

1- 14- year-old child comes with nephritic syndrome with abnormal renal function tests showing azotemia and hematuria. IF is positive for IgG and C3 showing lumpy, bumpy deposits. EM is showing subepithelial hump. He has a history of sore throat, what is the most likely diagnosis?			
a-Diabetic nephropathy	b-Post-infectious glomerulonephritis	c- lupus nephritis	d-Hematuria
2- Which of the following diseases shown mesangial deposit?			
a-Lupus nephritis	b-Diabetic nephropathy	c-IgA nephropathy	d-Post-infectious glomerulonephritis
3- 10- year-old child with nephrotic syndrome, proteinuria, LM and IF are normal, EM showing diffuse effacement. What is your diagnosis?			
a-MCD	b-Post-infectious glomerulonephritis	c-lupus nephritis	d-SLE
4- A patient with nephritic syndrome and PIGN followed impetigo caused by Staph.A, kidney biopsy is done for the patient. What kind of deposits will you see? or what will IF/EM look like?			
a-liner deposits	b-bump-humb	c-granular	d-looks normal
5- 50- year-old male with Nephrotic Syndrome, IF is positive for IgG and C3 along the capillary loop, LM Shows diffuse thickening of the capillary loops, EM shows subepithelial deposits. What is your diagnosis?			
a-MGN	b-lupus nephritis	c-MCD	d-Diabetic nephropathy
6-A 52-year-old woman presents with swelling of her ankles of 6 weeks in duration. Physical examination reveals an obese woman (BMI = 32 kg/m2) with pitting edema of the lower extremities and periorbital edema. Laboratory studies show hyperlipidemia and hypoalbuminemia. Urinalysis discloses 3+ proteinuria and 3+ glycosuria but no evidence of inflammatory cells or RBCs. A kidney biopsy displays a prominent increase in the mesangial matrix, forming nodular lesions, and thickening of capillary basement membranes. Which of the following is the most likely pathologic diagnosis?			
A-Membranoproliferative glomerulonephritis	Diabetic glomerulosclerosis	c-Acute glomerulonephritis	d-MCD

1-B , 2-C ,3-A , 4-B,5-A 6-B

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Editing File