# Presentation of glomerular diseases

1. Nephrotic syndrome
2. Nephritic syndrome.
3. Rapidly progressive glomerulonephritis. ( RPGN).
4. Asymptomatic proteinuria.
5. asymptomatic hematuria.
6. Macroscopic hematuria.
7. Chronic glomerulonephritis.

# Nephrotic syndrome

pathognomonic of glomerular disease

Definition five criteria

1. ( ***proteinuria)*** > 3.5 g protein in 24 h urine = frothy urine. Normal protein in urine 150 mg/24 hour.
2. **Edema** = puffy eyes in the morning decreasing gradually during the day, leg swelling, increasing body weight. Is due to accumulation of fluid in the interstitial space . The most acceptable mechanism is the

under fill theory . (The protein loss in urine lead to decrease albumin in plasma and decreased effective vascular volume with resultant activation of the aldosterone leading to retention of fluid ).the other less acceptable is

the over fill theory which is that the primary glomerular pathology leads to retention due to inflammation and activation of the RAAS .

3- ***hyperlipidemia*** increase serum lipid because the liver compensate for proteinuria by protien synthesis and one of these are lipoproteins.

Lipid abnormality ( increase LDL, IDL, VLDL ,( increase hepatic synthesis), increase total cholesterol ( increase hepatic synthesis), decrease HDL levels ( increase hepatic synthesis and increase urinary clearances), increase atherogenisity due to increase in lipoprotein level and increased oxidized form of the LDL levels.

**4. hypoalbuminemia** : serum albumin < 3.5 g/dl ( normal 3.5-6 ) = white nails

***5- Lipiduria appearance of lipid substances in urine during microscopic exam.***

- Differential diagnosis of generalized oedema

1. Heart failure ( increase jugular venous pressure + EF <55%)
2. Hepatic cirrhosis ( stigmata of chronic liver disease and usually started as ascites)
3. Malnutrition
4. Protein losing  enteropathy
5. Iatrogenic
6. Cyclical edema
7. Static edema

# Complications

I. Hypercoagulability = abnormal spontaneous thrombosis of the blood.

(increase platelets aggregation, immobility, hypovolemia, increase fibrinogen, factors V, VII, von willibrands and ά 1 macroglobulin, increase urinary clearances of anti thrombin III) this leads to venous thrombosis and with accelerated atherosclerosis to arterial thrombosis the later is more in children with minimal change disease. These usually happened when albumin level decrease to less than 2 g/dl. Dysfibrinogenemia leads to increase ESR even more than 100. rena;l vein thrombosis can ocuur ( clinically in 8% ) but when looking for it present in 50 % and if bilateral can leads to AKI ( rare) and when present it present as flank pain and hematuria. Pulmonary embolism

II. malnutrition

III. Hyperlipidemia and lipidurea

IV. decreased Vit D2 level with normal free Vit D level ( due to loss of binding protien ) so frank osteomalacia is rare in nephrotics.

V. Iron, zink, copper deficiency ( binding protiens lost in urine)

VI. Decrease total T-4 but free T-4 & TSH are normal and no clinical consequences.

VII.. Infection : due to oedema fluid which is a good culture media, loss of complement and IgG in urine, fragile skin in nephrotics, dilution of local humoral immunity factors by fluids , impaired neutrophil phagocytosis, loss of transferrin and zink in urine which are required for lymphocytes function

VIII. Acute kidney injury : due to volume depletion, sepsis , transformation of the disease, bilateral renal vein thrombosis, drugs susceptibility ( NSAIDs, ACEIs, ARBs), increased risk of interstitial nephritis due to the drugs including diuretics, nephrosarca ( intra renal oedema with compression of the tubules)

IX. Chronic kidney disease : especially if protienuria > 5 gs /24 h but not usually in minimal change disease.

- Management of nephrotic syndrome

**1.<u>Non specific management</u>**:

- Diuretics for edema (loop diuretic +/- thiazides ).
- Lipid lowering for hyperlipidemia ( statin)
- ACEIs <u>+</u> ARBs with CCBs and spironolacton if needed for proteinuria

- **<u>2-Specific treatment</u>** according to the cause ( coming lectures)

**<u>3-Treatment of complications</u>** as antibiotics for infections and anticoagulants for thrombotic events.

# causes of nephrotic

1. minimal change disease usually in children ( no changes in LM,
2. membranous GN( thikining and spikes in GBM with IgG deposition in the glomeruli only,
3. focal segmental glomeruloseclerosis( fibrosis and adhesion or synechiae of the bowman capsule and now regarded the most common cause of nephrotic syndrone in adults)
4. diabetes mellitus ( diabetic nephropathy, nodular seclerosis )
5. amyloidosis ( deposition of abnormal protien)
6. Membranoproliferative GN ( type I.II, cryoglobulinemic)

# nephritic syndrome

Definition:

1. hypertension ( normal blood pressure in nephrotic),
2. less oedema than nephrotic,
3. less protienuria, i.e non nephrotic range that is less than 3.5 g / 24 hour
4. very rapid onset ( usually insidious in nephrotics except in minimal),
5. Hematuria with RBCs cast ( for academic purposes we will regard as not present in nephrotic)

RBC cast = proteinatious substances embedded in it RBCs and these RBCs are usually destructed RBCs ( dysmorphic RBCs) coming up from the inflamed glomerulus . (hematuria + proteinuria ) is called active urine sediment ( absent in nephrotic in which urine sediment is called bland),

1. Oliguria ( may happened in nephrotics with severe oliguria )
2. normal to slightly reduced serum albumin ( severe in nephrotic),
3. elevated jugular venous pressure ( normal or low in nephrotic)

# Causes of nephritic syndrome.

1. Post streptococcal GN ( typically 2 weeks after pharyngitis, 3 weeks after impetigo .), elevated ASOT , tea color urine , decrease urine output = oliguria= < 300ml/24 h or anuria = <100 ml /day , hypertension )
2. IgAN typically few days… 3-5 days after URTI or Gastroenteritis
3. Systemic lupus ( SLE) = (lupus nephritis). SLE is a connective tissue disease of unknown etiology (4 of 11 diagnostic criteria) with positive antinuclear Ab, Anti double strand DNA Ab, decreasesd C3 & C4 complement level
4. After endocarditis, abcesses. In both blood culture is helpful in diagnosis.
5. Vasculitis ( small vessel vasculitis ( wegners granulomatosis = granulomatous polyangitis) , microscopic polyangitis , churg – strauss syndrome.

Days to weeks renal function will deteriorate with features of nephritic

But Usually blood pressure is normal, may have vasculitic features as petichial rash in skin,

Causes

1. Goodpasture syndrome ( renal failure + pulmonary hemorrhage .. Hemoptysis positive anti glomerular basement membrane Abs.
2. Vasculitis;

wegner ( upper.. sinusitis & lower RT…. lung nodules, hemoptysis, lung cavitation with renal failure and positive C- ANCA)

microscopic polyangitis.. Multisystem involvement with positive P ANCA

pauci immue crescentic GN .. Renal involvement only with positive P ANCA

3. PSCGN
4. IgAN/ Henoch Schonlein purpura
5. endocarditis

Asymptomatic Protienuria (150 mg- 3000mg / 24 hours)

Asymptomatic Hematuria > 2 RBCS/ HPF or > 10 milion / liter and are usually dysmorphic RBCs

Brown red painless no clots coincide with infection

And between attacks there is microscopic hematuria +/- protienuria

Presents as Renal insuffiecncy ( increase blood urea and serum creatinine) , hypertension, protienuria often > 3 grams, shrunken smooth kidney by U/S < 9 cm length and renal biopsy is contraindicated.

- oliguria < 500ml in 24 hour
- Anuria < 100 ml/ 24 hour