النادي الطبي

GENITOURINARY SYSTEM

SUBJECT : Pathology

LEC NO. : Lec 1,2

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وَقُل رَّبِّ زِدْنِي عِلْمًا

GENITOURINARY SYSTEM

*most of renal pathology is clinically oriented

*Kidney disease can effect glomeruli, tubules, blood vessel, interstitium.
-glomeruli: a blood vessels lined by endothelial cells (fenestrated) and below it BM, and below the BM is the epithelium of boman capsule
*The disease can occur in any part & it is possible to damage mesangial cells

-the diseases of glomeruli usually immune mediated

*if there is problem with epithelium the foot processes are fused and leak for protein

there are number mesangial cells (its function is filtration of residual vesicle also helping control of GFR
*GBM consists of collagen (mostly type IV), laminin, proteoglycans, fibronectin, & several other glycoproteins
the urea gathers in bowman space (urinary space) then to the tubules

*tubular & interstitial disorders are more likely to be caused by toxic or infectious agents
**Glomerular Diseases is the one of most causes of renal failure (chronic renal failure treated with steroid

-Types of Glomerular Diseases :

1-Primary Glomerular Diseases: only kidney involved
- Minimal-change disease (MCD)
- Focal and segmental glomerulosclerosis (FSGS)
- Membranous GN = Membranous nephropathy (MN)
- Membranoproliferative GN (MPGN)
- Acute postinfectious GN
- IgA nephropathy
- Chronic GN

**The first 4 disease make Nephrotic syndrom (massive proteinuria 3.5 gm)

GENITOURINARY SYSTEM

2-Secondary to Systemic Diseases:
•Lupus (SLE) nephritis (Ab against the DNA)
•Diabetic nephropathy
•Goodpasture syndrome: Ab against BM that attack lung and may effect kidney
•Microscopic polyangiitis
•Wegener's granulomatosis
•Henoch-Schönlein purpura
•Thrombotic microangiopathy
•Amyloidosis
•Bacterial endocarditis-related GN
•GN secondary to extrarenal infection
•GN secondary to lymphoplasmacytic disorder

**Pathogenisis:1- Nephrotic: Heavy proteinuria >3.5 with hyaline PAS\firstly periorbital edema then generalized edema\ hyperlipidemia(hypercholestrol)\ little or no azotemia, hematuria, or hypertension\ hypoalbuminemia
the urine is foamy because it contain protein & we see hyline cast
**2-Nephritis: inflammation at glomeruli --> hypercellular
**the hematuria smokey (tea like) \ dark urin maay come from bilirubin
case: patient with hematuria or smokey urine you should exclude nephrotic also come with azotemia, oliguria and hypertention

*Nephrortic may come from infection like streptococcal ,pharyngitis or any infection make ag-ab complex
A-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)
B-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)

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1)Nephrotic (minimal change):No immune complex mainly child comes with: periorbital edema, generalized edema,heavy protein urea, it may comes from T-cell dysfunction which will give the permeability for protein.
It is not a requirement that its immune
**normal appearance by LM and IF (-) --->effacement of podocyte foot processes and no ab by EM its only way to diagnosis
**it has selective protein urea (good prognosis) and respons to treatment and 75% recovered
**non selective protein urea seen at Membranous nephropathy

2)Focal and segmental glomerulosclerosis(FSGS): No immune complex

*most common disease which make nephrotic syndrom at adult, after it the membranous Nephropath, it may come primary and secondary
**its not diffuse (you can find normal part of glomeruli)
-it can come from IgA nephropathy
*Idiopathic 20-30% of cases and may effect children

**clinically: its non selective to protein,HT,hematuria,excrete lipid and make hyaline

Collapsing glomerulopathy is developed FSGS

*we see Mesengial matrix which make scar --->IgM but the most cause is T-cell dysfunction, poor response
-if the problem systemic and we do renal allograft the protein urea consist

*scarred, obliterated capillary lumens with accumulations of matrix material which will reduce perfusion then HT

**The permeability-increasing factors produced by lymphocytes have been proposed in both MCD & FSGS

3)Membranous GN(MGN)=Membranous Nephropathy MN):
-slowley progress --->adult people--->diffuse effect---> subepithelial deposit (immune complex)-may come from secondry (SLE,HBV) or any reason for GNF
*85% idiopathic ---> second cause of NS
*it has Anti-PLA2R autoantibodies(Phospholipase A2 receptor)u-;

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-the kidney is big+pale+dense capsule+smooth outer surface
**LM: thick BM
**IF: IgG (PAS silver) and C complement its appear granular not linear
**EM:thick subepithelium it codense gather and make spike+dome shape

**clinically: idiopathic---> make NS, proteinurea(non selective) poor response to cortocosteroid
-60% of patient persist proteinurea, 40% renal failure, 30% remession

-Nephritis: 1) Membranoproliferative GN:

*problem with BM + hypercellular of endothelial+mesingeal cell

**the patient come with Hypocomplementemia

it has 3 types:
1) type 1: subendothelial deposit+Mesingeal--> immune complex or activation of complement, IgG make stablization the C3 and it still active
2) type 2: deposition at BM intramembranous due to just excessive complement activation lead to Hypocomplementemia , linked with ab against C3 convertase called C3 nephritic factor

* >50% with nephrotic syndrome
*10 -20% with acute nephritis syndrome

***the difference between 1 and 2 :
type 1: IF-> you see IgG,C3,C4
type 2:IF-> you see C3 intramembrance of

Morphology we see the same feature:
large,proliferation of mesangial,subendothelial and endothelial cells+infiltration of leukocyte make hypercellular

**the key word of this disease is tram track appearance caused by "splitting" of the GBM Forming double contour / tram track appearance (PAS or silver stain)
**50% of MPGN cases presented with nephrotic syndrome,20% nephritic
*Crescents(rapidly progression) in ~20% cases
*poor prognosis

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