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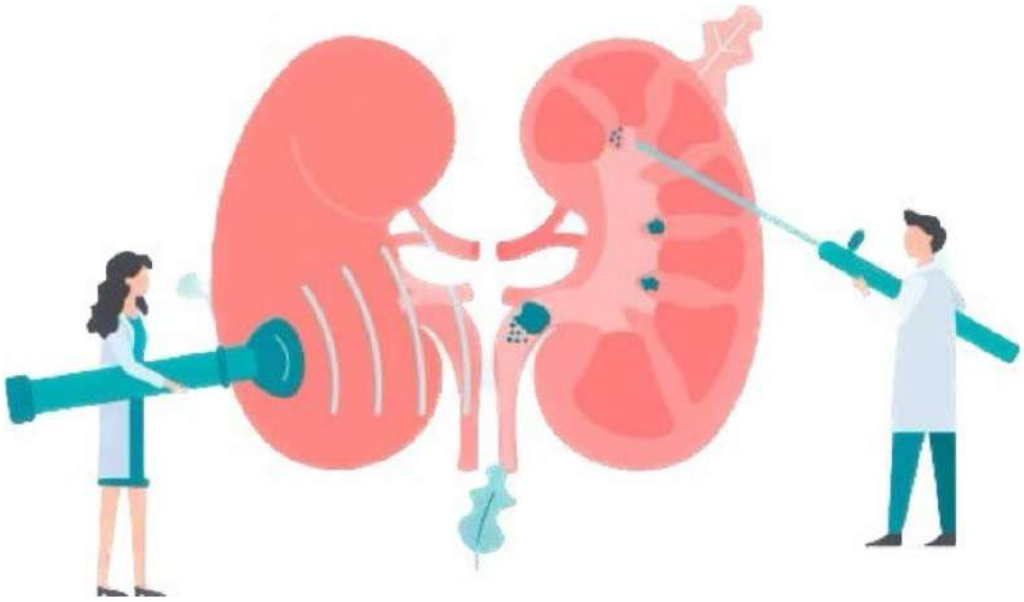
MEDICINE

NEPHROLOGY

By

Dr.Priyansh Jain



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MEDUHUB_FMGE

Meduhub - FMGE

- MBBS (Gold Medalist)
- MD (General Medicine) — JLN Medical college
- Consultant Physician & Assistant Professor
- USMLE/MRCP qualified
- 7 International Publications
- National Level Faculty - NEET-PG, FMGE/NExT
- President's Award (Scouts)
- Selected for Research and Training at NIH, USA (sponsored by USA Government)
- Delivered lectures in International and National Medical Colleges

Dr. Priyansh Jain

INDEX OF NEPHROLOGY WORKBOOK

CHAPTER 1
RENAL TUBULAR ACIDOSIS

Renal Tubular Acidosis

Defect in renal tubules leading to Acidosis

Type I → defect in _____ cell in _____ tubules

↓

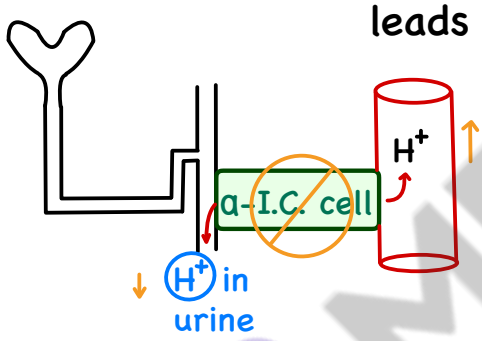
so unable to excrete ____ in urine

↓

leads to ____ buildup in blood

↓

Acidosis



a/c/a - _____ RTA

- Urine pH →
- Associated with → _____ → _____
- Etiology → Sjogren syndrome/cirrhosis/Amphotericin B/Sarcoidosis
  → Hyper Pth.
- Investigation →

EXTRA POINT

- NH_4Cl → is excreted in distal tubules by α-intercalated cell
  ↓
  So in distal RTA → pH will (NOT) change

- Check urine pH → Infusion of NH_4Cl [mild acid] → again check urine pH

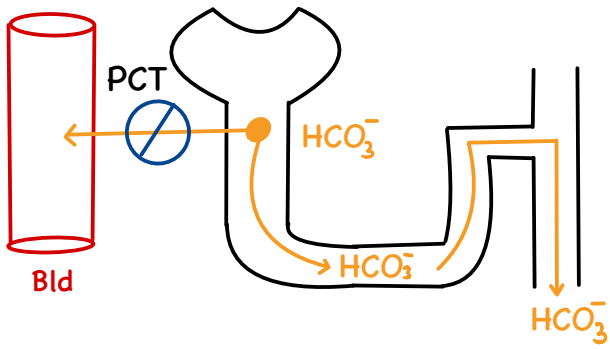
Type II RTA – Defect in _____ tubule

↓

Unable to absorb _____

↓

So leads to _____ of blood



- a/c/a → _____ RTA
- Associated with → _____ but there is also ⇧ in _____
  So, ____ RENAL STONE
- Urine pH = variable
- Etiology - Fanconi Synd./Amyloidosis/M.myeloma/Carbonic anhydrase#
  [_____]
  ↓
  _____
- Investigation → _____

Type IV [MC type]

MCC

JG cell ← ___ ←

↓

Renin

↓

Angiotensin

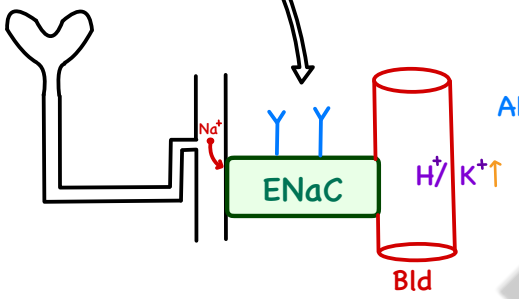
↓

Aldosteron

Adrenal pathology

↓

leads to met. Acidosis with _____ Q



Aldosterone receptor resistance

Can be seen → with the drugs which _____

↓

- Eg - _____

→ HIV / SCA

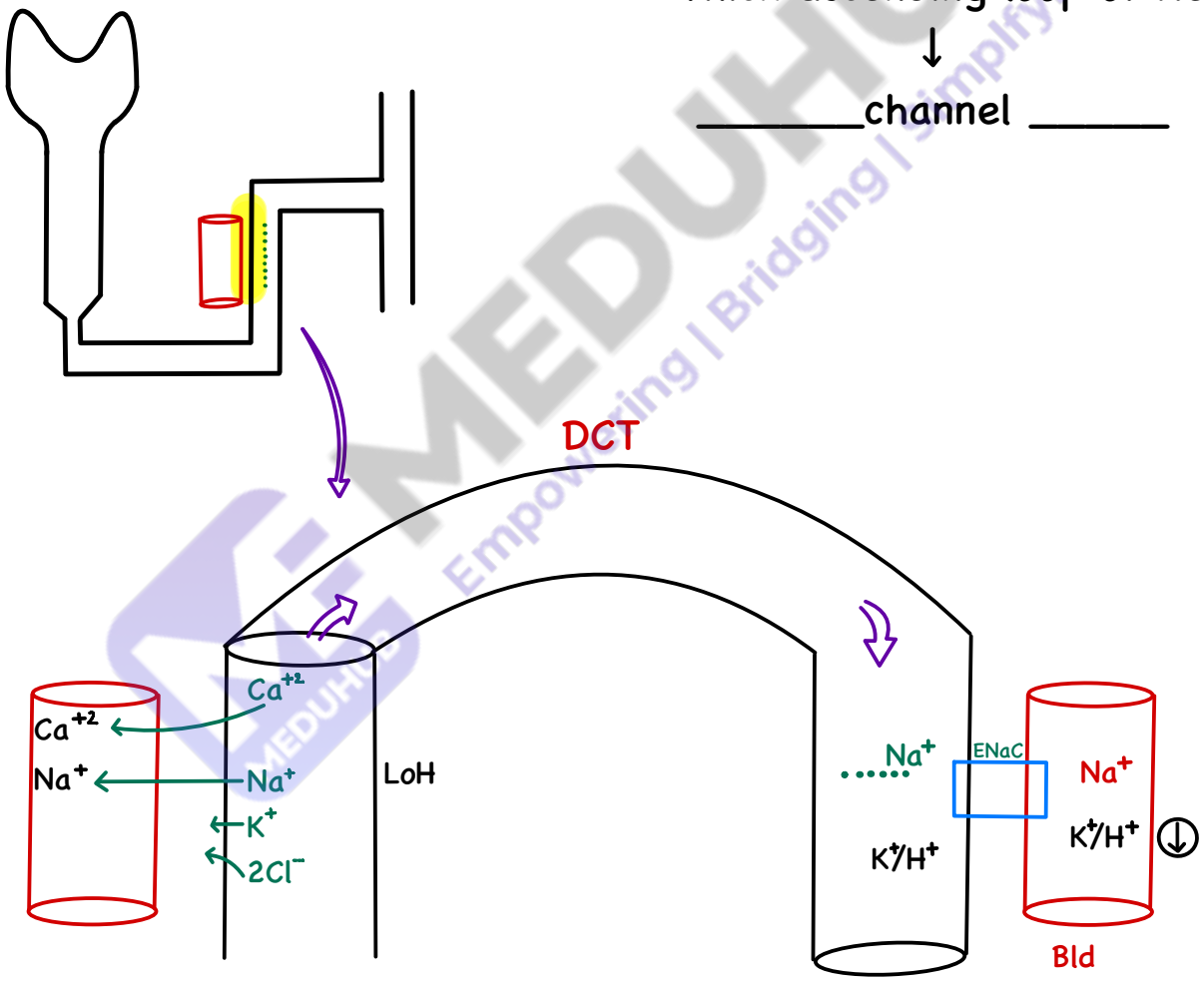
SUMMARY

RTA	TYPE I	TYPE II	TYPE IV
	Distal	Proximal	Hypoaldo. OR Aldo. Resistance
Mechanism	Unable to excrete	Unable to absorb	Unable to excrete
Hyperkalemia			
Renal stone	[$CaPO_4$]		
Investigation			

CHAPTER 2
BARTTER SYNDROME/GITTLEMEN SYNDROME/ LIDDLE SYNDROME

BARTTER SYNDROME

- Thick ascending loop of Henle
  ↓
  _____channel _____



- Loss of Na^+ in urine
  - → Hyponatremia → _____
  - → water loss → _____
- $K^+\downarrow$ → _____
- Met. _____
- Hypercalciuria → _____

Rx
- → K^+ & Na^+ supplement
- → NSAID → GFR → loss of Na^+ & K^+/ water [by PG]

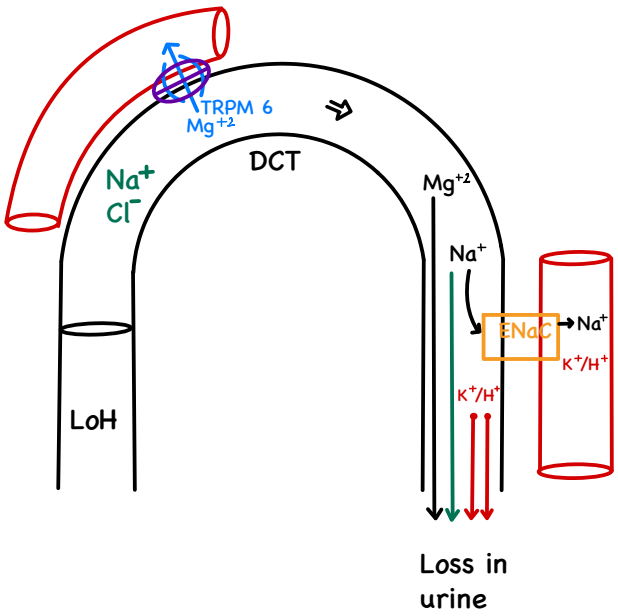
GITLEMAN SYNDROME

→ Defect in NaCl channel & TRPM6 in DCT

↓

[Na^+ K^+ Mg^{+2}]

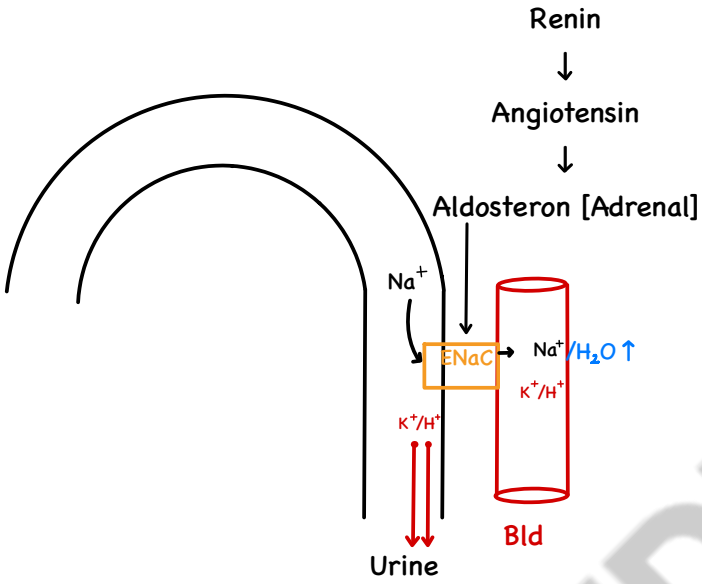
(No) renal stone



SUMMARY

	BARTER	GITELMAN
	_____ defect In _____	_____ _____defect in _____
Bld. Na^+ K^+ H^+		
Additional loss Ca^{+2} or Mg^{+2}		
BP	ⓝ / ↓	ⓝ / ↓
Similar to chronic use of		

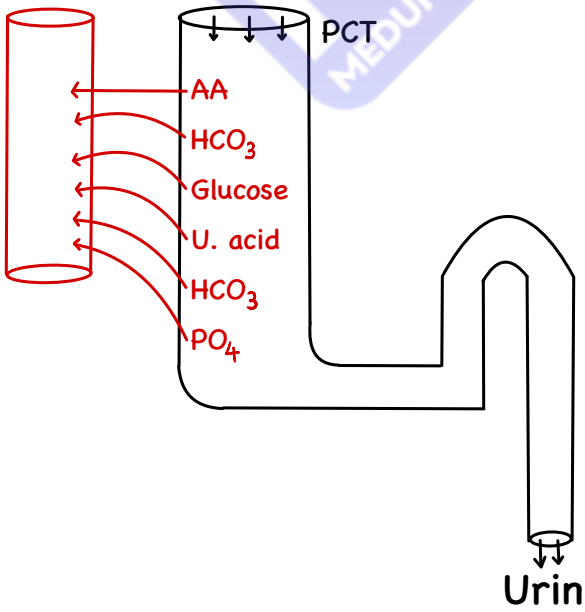
LIDDLE SYNDROME



Defect
↓
Gain of function mutation of ENaC
↓
Bld. → ↑ Na^+ / ↑ H_2O → HTN
⊖ feedback will ↓ Renin
↓
Low Renin HTN

Rx → ENaC # → Amiloride

FANCONI SYNDROME – defect of ____ [d/t Wilson disease/Tyrosinemia/ GSD/Lead poisoning]



↓

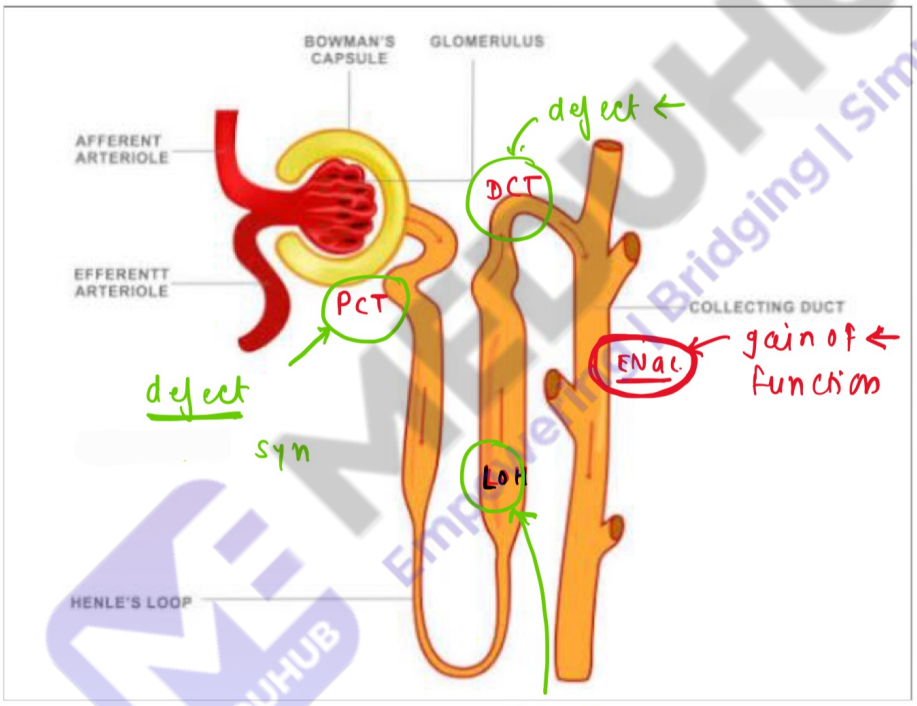
_____ absorb $HCO_3^{\ominus}$ /AA/U.acid/ Glucose/$PO_4^{\ominus}$ in PCT

↓

_____ urine

- AA = growth _____
- $HCO_3^{\ominus}$ loss → _____ RTA [Type___] in

SUMMARY


BOWMAN'S CAPSULE
GLOMERULUS
AFFERENT ARTERIOLE
EFFERENTT ARTERIOLE
PCT
DCT
COLLECTING DUCT
ENaC
HENLE'S LOOP
LOH
defect
syn
defect
gain of function
syn
defect
syn
syn

CHAPTER 3
INVESTIGATIONS IN NEPHROLOGY

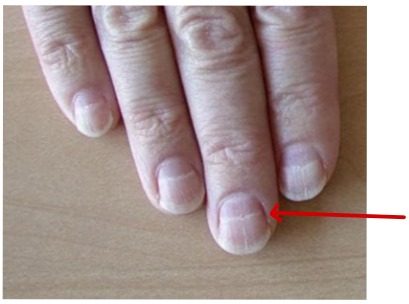
INVESTIGATION IN NEPHROLOGY

1) Urine Analysis

- pH =ⓝ=
- Proteinuria
  - ↳ⓝ =
- Albuminuria →ⓝ→ mg/day

  Microalbuminuria → mg/day

  Macroalbuminuria → mg/day



- Difficult to collect 24 hr urine

  ↓

  So for spot analysis → used →

ACR

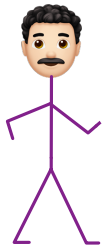
A1 = <30 mg/gm

A2 = 30-300 mg/gm

A3 = >300 mg/gm

- Nephrotic Range → Protienuria → gm/day
  → Albuminuria → gm/day

- Orthostatic Protienuria



→ U. Protien – up to gm

Long standing

↓

C/a. Protienuria

↓

To confirm- collect U. Sample after

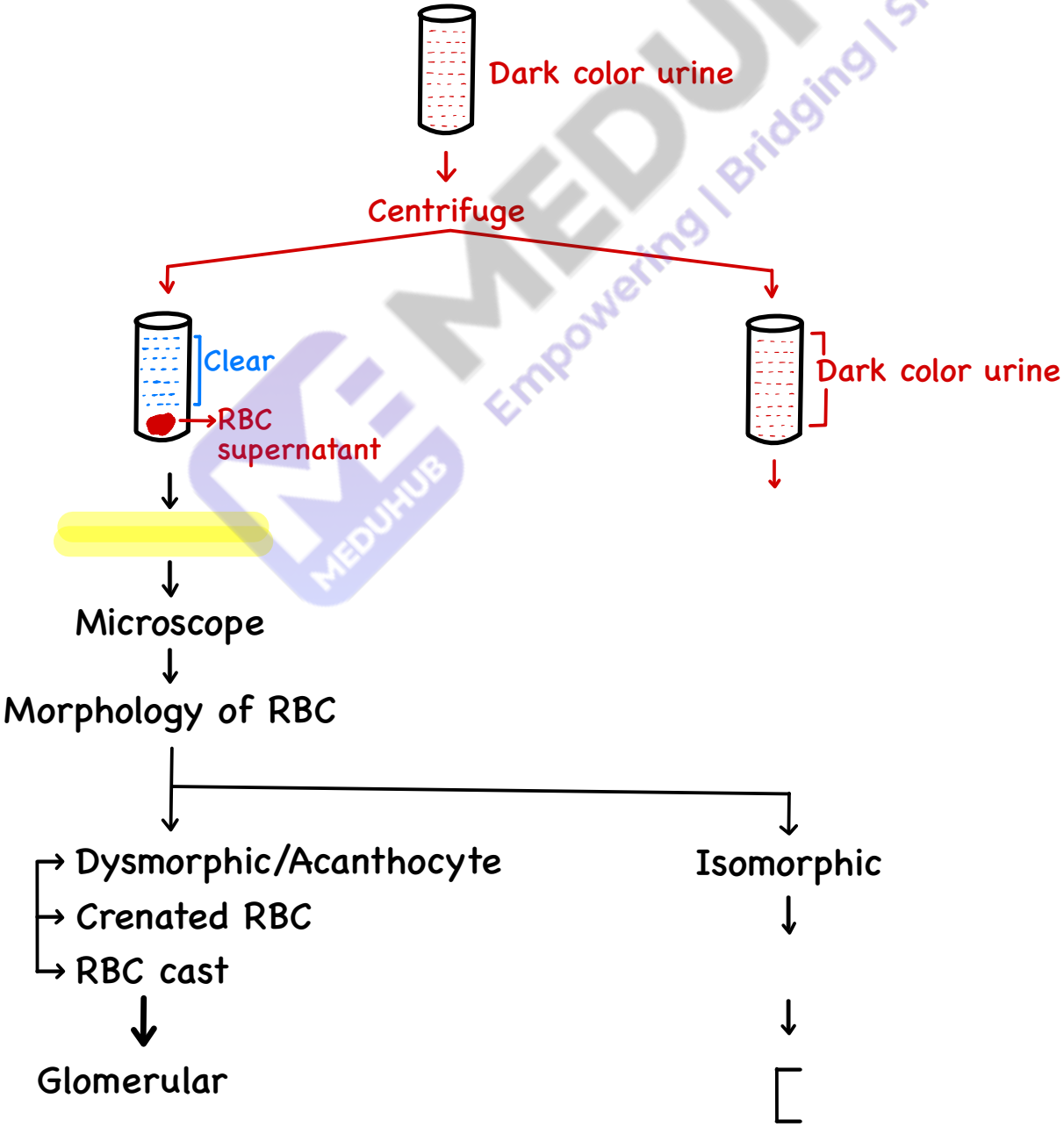
↓

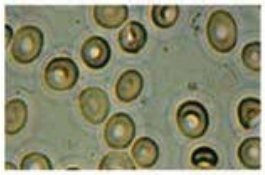
c/a –

↓

Rx –

- Hematuria → ≥3 RBC/HPF[Q]





Isomorphic RBC

Dysmorphic RBC

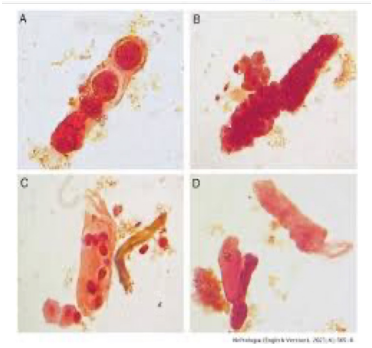
RBC cast

RBC

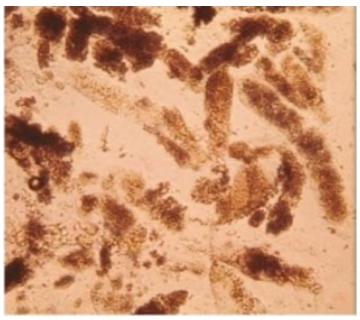
- Other color of Urine

Yellow to Orange	
Orange	
White	
Red – sun exposure ↓ Dark	
ⓝ→ after sometime ↓ Dark	

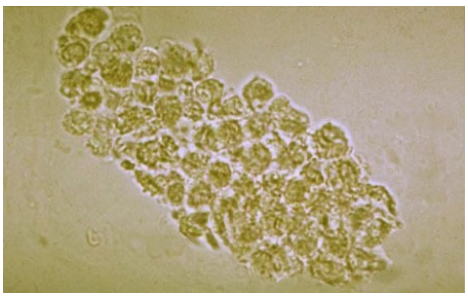
- Cast



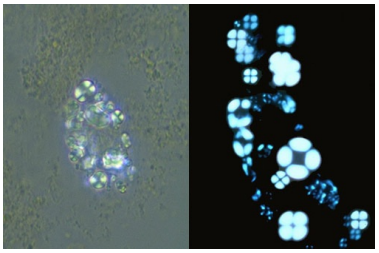
RBC cast
↓



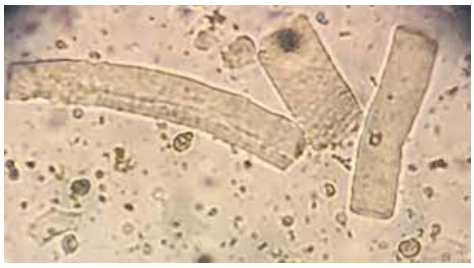
Muddy Brown
↓



WBC cast
↓



Fatty Cast
[Maltose cross]
↓



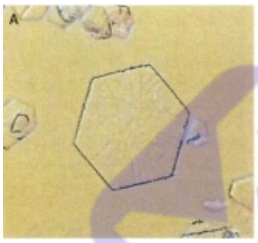
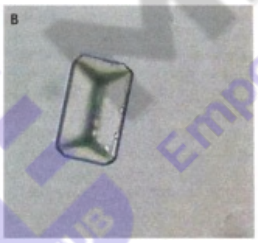
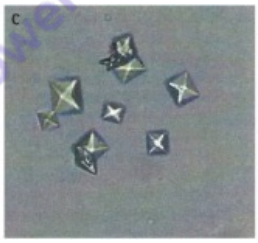
Broad Waxy Cast
↓

- Pyuria → ≥5 WBC/HIF
  → seen in

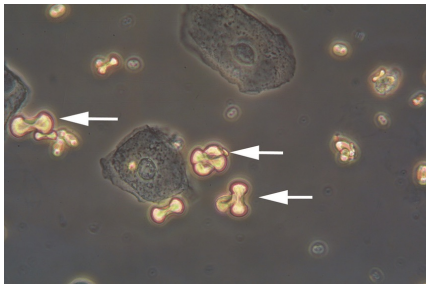
Sterile Pyuria → WBC But Bacteria
↓
Seen in

- Leucocyte ESTERase test — to detect in urine
- Nitrate test — to detect in urine

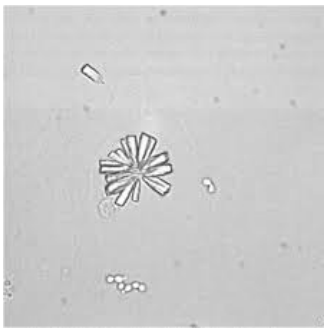
- Urinary Crystal


Hexagonal ↓ | Coffin lid ↓ | Envelope ↓ | Rhomboid ↓



Dumbbell shape
↓



Star shaped
↓

[Renal stone leads to gastric symptom c/a - Reflex]

- Specific gravity of urine = 1.020-1.030
- ⓝ→ S.G. of urine > S.G. of plasma [1.010]

If S.G. of urine = S.G. of plasma
↓
c/a -
↓
Seen in

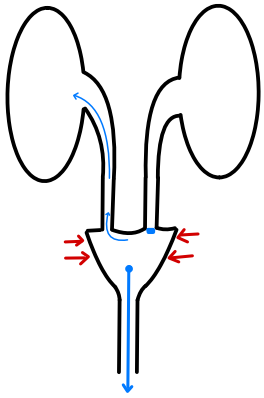
GFR ESTIMATION

ⓝ→ [>90 ml/min/1.73m^2]

formula
clearance [BEST]
clearance
↓
During decline in kidney function, tubular secretion of creatinine occurs — which may. GFR.

- Polyuria = >3 lit./day or >50 ml/kg/day
- Oligouria = <400 ml/day
- Anuria = <100 ml/day

DMSA SCAN — to see — of kidney



Vesicular-ureteric Reflex

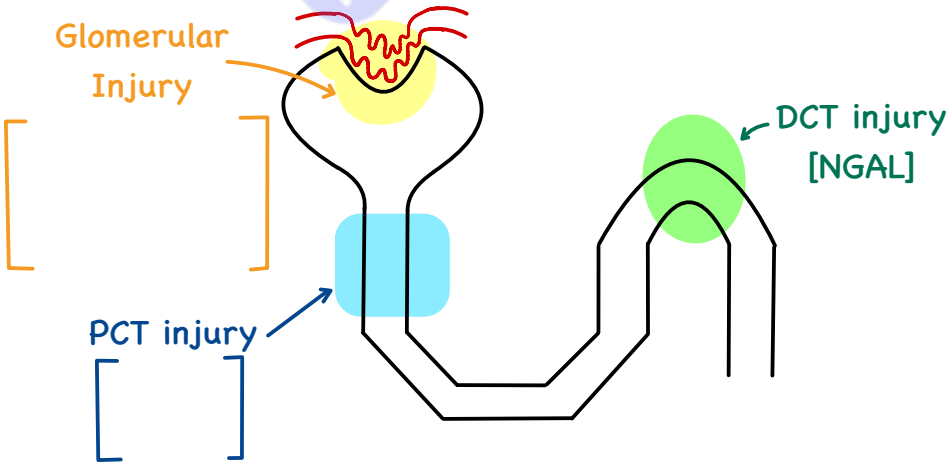
↓

↓

↓

DTPA — to assess of Kidney

MARKER OF KIDNEY INJURY



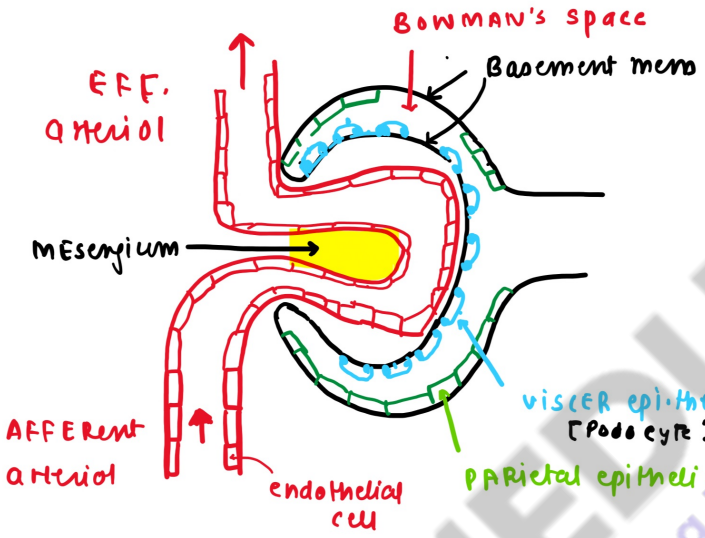
Kidney injury marker - I
Neutrophil gelatinase associated lipocalin

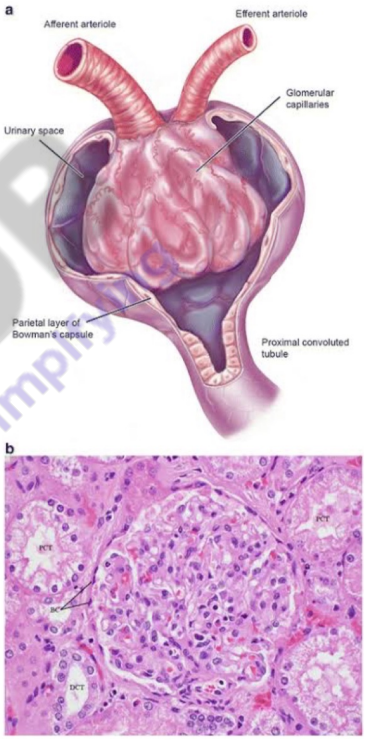
SUMMARY

CHAPTER 4
NEPHROTIC SYNDROME

GLOMERULAR DISEASE

- Nephrotic Syndrome
- Nephritic Syndrome





NEPHROTIC SYNDROME

↳ Protienuria: /day

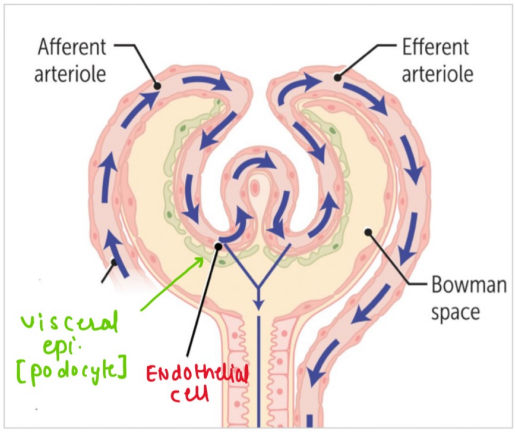
- Loss of Albumin [albuminuria gm/day]
  ↓
  Hypo-albuminemia →
- Loss of protein © & antithrombin III — ⇧ Risk of
- Loss of IgG - ⇧ Risk of →
  ↓
- Compensatory ⇧ synthesis → Lipid →
  by liver
  → ↑ Fibrinogen →

Etiology → Renal → MC - Child —
→ MC - Adult —
→
→
→ Systemic → [overall MCC of Nephrotic syndrome]

MINIMAL CHANGE DISEASE/LIPOID NEPHROSIS

Etiology → 1° — Idiopathic → Sometime d/t mutation of
c/a - type
→ 2° — Drug —
— SLE/HIV/Hodgkin lymphoma

Pathology — Loss of foot process of. → →



- Hematuria, HTN, S.creatinine —

- Light microscopy →
- Electron microscopy →

Also seen in — membranous nephropathy
— FSGS

Rx → → if resistant → →
→ ⊕ Standard Rx of Nephrotic syndrome
→ To ↓ Proteinuria -
→ Avoid excessive in diet
→ Reduce to ↓ edema
→ [For Hyperlipidemia]

Complication → can convert in

MEMBRANOUS NEPHROPATHY

→ Etiology → 1° → Idiopathic
→ 2° → Hep. B/(F. malaria) → can lead to DPGN
→ Autoimmune disease — SLE/RA/Hashimoto's
→ Drug — NSAID
→ Malignancy[Q] — GI/Lung

Pathology — M-PLA2 [Membrane associated phospho-lipase A2]

of podocytes becomes.

↓

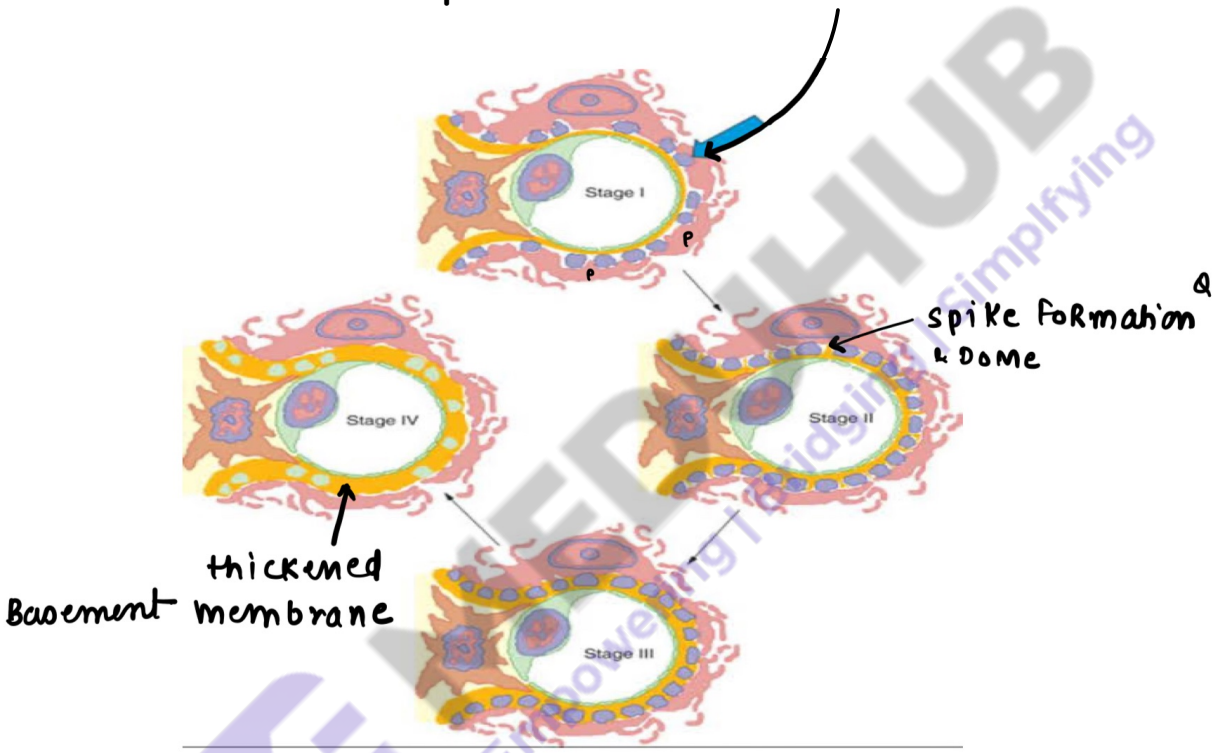
Formation of

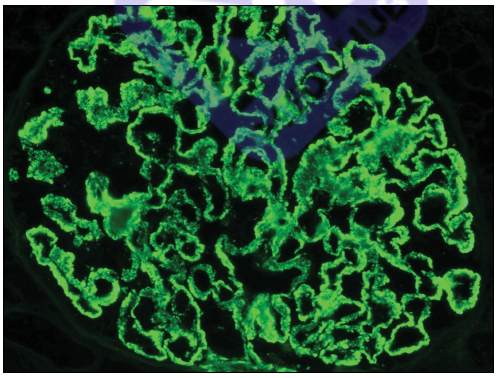
↓

Formation of [M-PLA2 Ag + IgG-4 + Complement]

↓

Deposit in





Complication — Highest risk of among all causes of nephrotic syndrome

Rx → Standard Rx of nephrotic →

→ Modified Ponticeli Regime

↓

_____ [cyclophosphamide]

FOCAL SEGMENTAL GLOMERULOSCLEROSIS

glomerulus is involved

glomerulus is involved

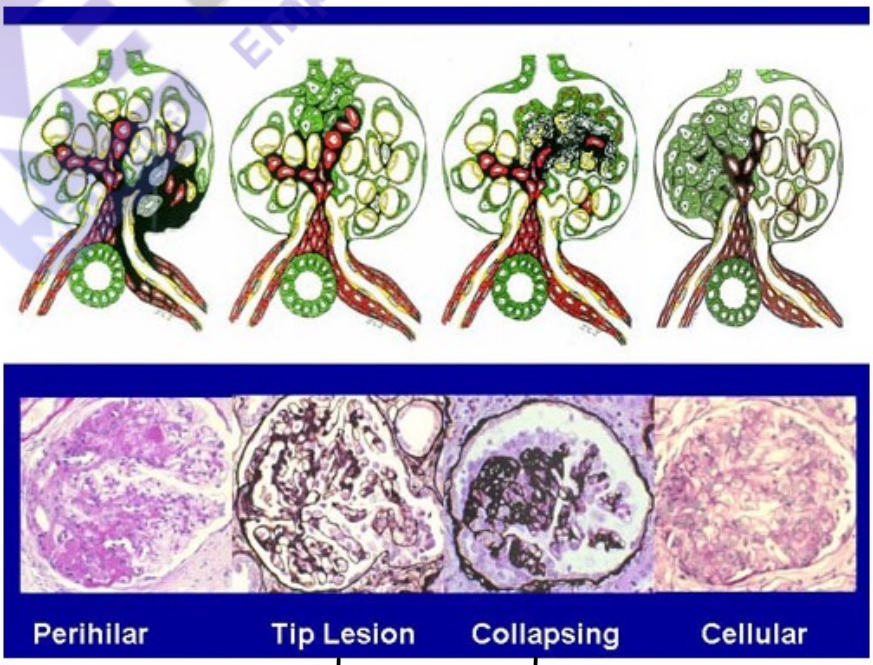
Mainly at cortico-medullary junction

Kidney Bx — min. 10 glomerulus

Etiology → 1 → Idiopathic → sudden onset → More severe

→ 2 → HIV/DM/Reflux nephropathy/obesity/post nephrectomy → C/a. Hep C → Insidious onset → Less severe

MCC of nephrotic syndrome in



Prognosis Prognosis

SUMMARY

CHAPTER 5
NEPHRITIC SYNDROME

NEPHRITIC SYNDROME

- → Proteinuria
- → Hematuria →
- → HTN ⊕ / Oligouria ⊕

IgA NEPHROPATHY / BERGER NEPHROPATHY

Pathogenesis: Defect in. antibody [galactose deficit]

↓

So act as foreign particle

↓

So antibody is formed c/a antibody

↓

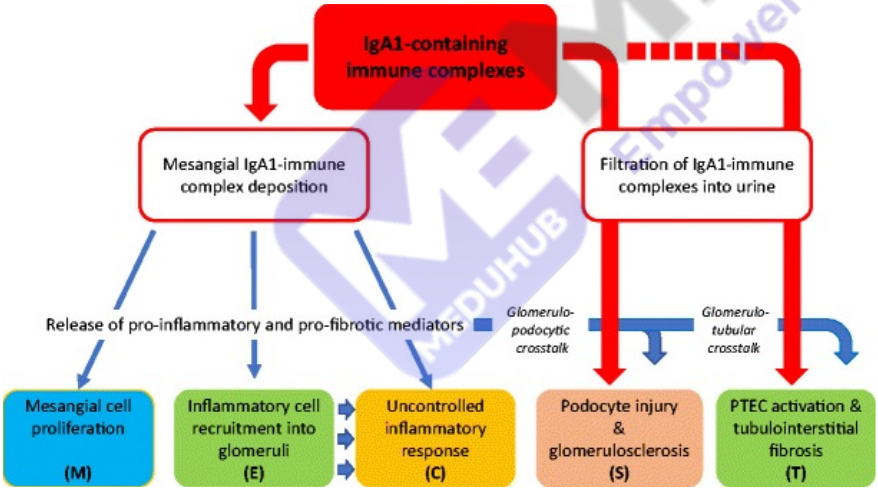
So, defective IgA + anti-glycan antibody → Immune complex

↓

Deposit in

↓

proliferation



- — Nephritic syndrome
- — Bimodal age → Young →. Hematuria
  → Old →. Hematuria
- — Can progress to

C/F — H/O. infection F/B

— Associated with - disease
- disease
— If. deposit
— Oxford classification —. score

Rx → To ↓ Proteinuria -
→ HTN -
→ Fish oil [omega-3] →
→ If Proteinuria [>1g/day] →

ACUTE POST-STREPTOCOCCAL G.N.

Pathogenesis

Streptococcus [1/4/12/49]
Antigen — exotoxin - B
— plasminogen

days

→ Formation → deposit

Anti-streptolysin-ab
Anti-DNAase-ab
Anti-hyaluronidase-ab

GN

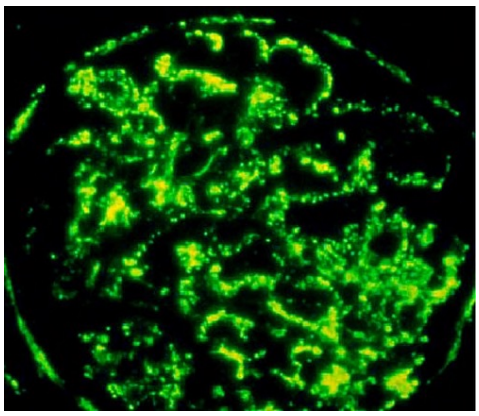
Gross Hematuria [Cola]

Skin impetigo Or Pyoderma

months

- C3 level

Bx
- If → appearance



- electron microscope →. appearance

Rx
- Antibiotic –
- Low protein diet
- BP control –

SUMMARY

IgA Nephropathy	PSGN
• Sore throat ⟶ Hematuria days • C3 level – • If —	• Sore throat ⟶ Hematuria days • C3 level – • If —

MEMBRANO-PROLIFERATIVE GN [MPGN]
OR
LOBAR GN

Pathogenesis:

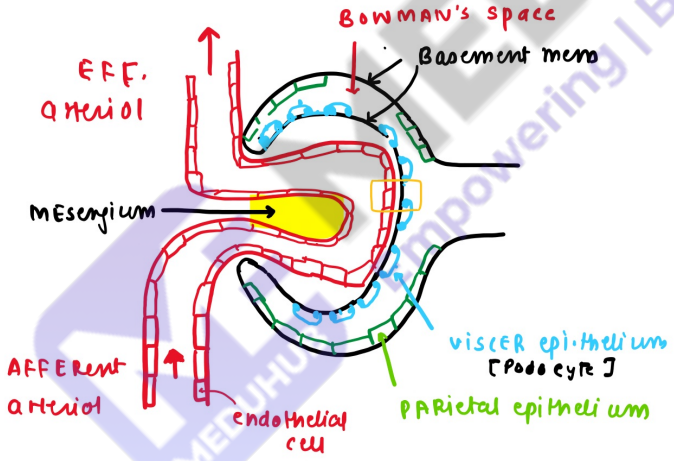
Ch. illness [malignancy Autoimmune dis.] → Ch. deposition of

↓

Inflammation

↓

Deposition of immune complex &/or complement



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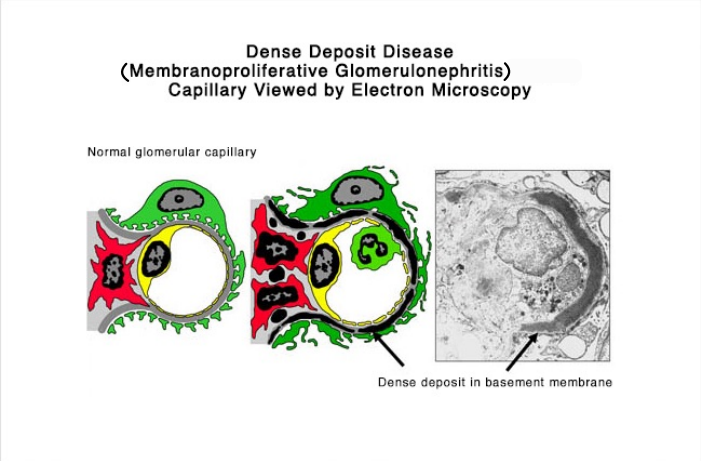
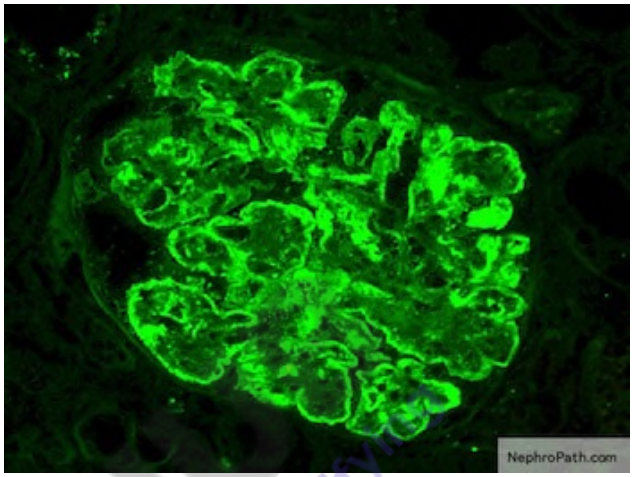
Types of MPGN

- Only immune-complex deposition → Steroid
- Immune complex + complement deposition → steroid

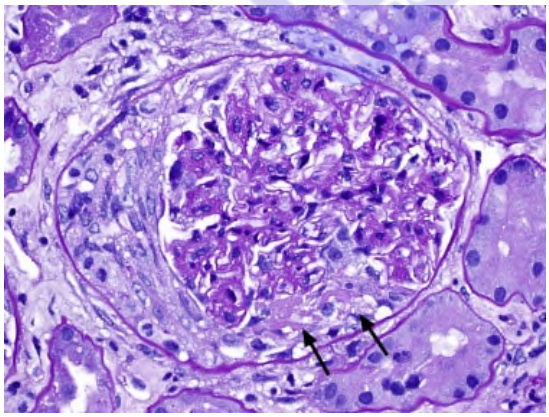


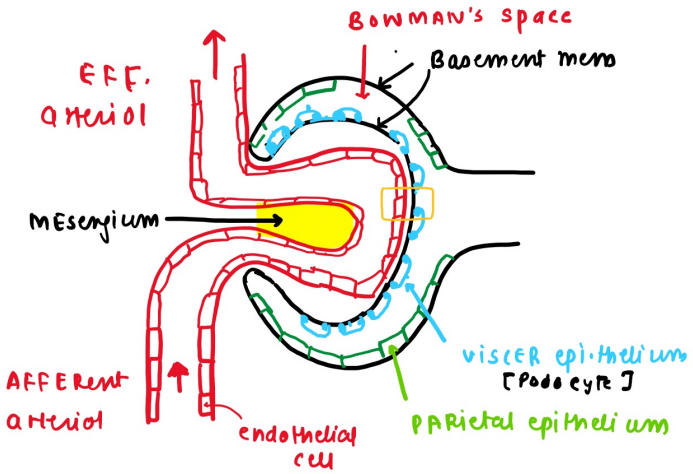


RPGN

Pathology of RPGN
- → Rupture of
- → Proliferation of

↳ Classification
- → Type I → G. Pasteur's syndrome/anti-GBM disease
- → Type II → PSGN/IgA nephropathy/SLE
- → Type III → ANCA + GN [Wegner/C. Strauss]
- → Type IV → Mixed Variety [anti-GBM dis. + one more type]





SUMMARY

CHAPTER 6
ACUTE KIDNEY DISEASE

ACUTE KIDNEY INJURY [AKI]

Definition:
- ↑ in S. creatinine ≥0.3 mg/dl in 48hrs
- OR
- ↑ in S. creatinine 1.5 times from baseline in 7 days
- OR
- U. Output <0.5ml/kg/hr for 6hrs

↳ If

RIFLE Classification:

R - Risk — U. Output <0.5 ml/kg/hr x

I - Injury — U. Output <0.5 ml/kg/hr x

F - Failure — U. Output <0.5 ml/kg/hr x

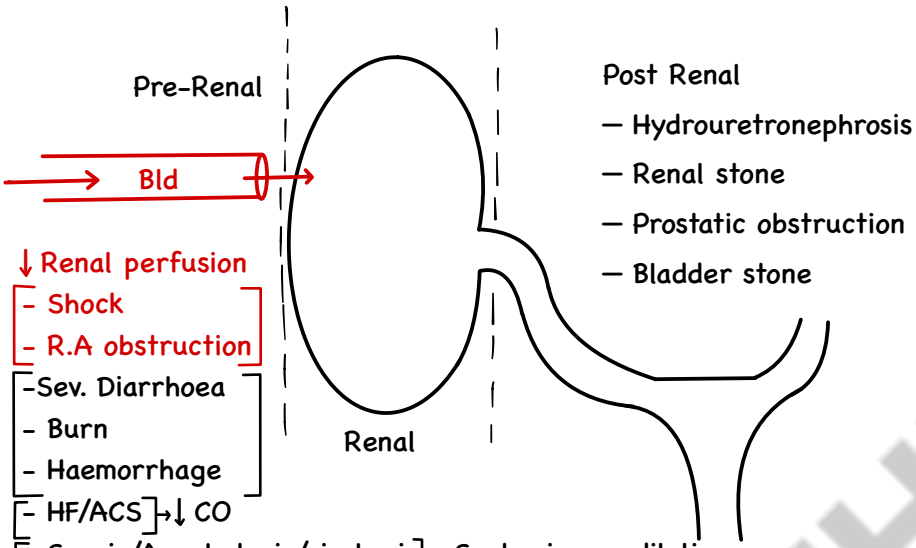
L - loss of kidney function — Dialysis required for

E - ESRD — Dialysis require for

KDIGO Classification

	U. Output.	S. creatinine
Stage I	<0.5 ml/kg/hr x	>0.3 mg/dl within
Stage II	<0.5 ml/kg/hr x	2-2.9 times from baseline
Stage III	<0.3 ml/kg/hr x Or Andria >12 hours	>3 time or dialysis req.

Causes of AKI



[NSAID/Amphotericin B/Contrast induced] → AFF constriction

[RAAS # → ↓ AT II] → EFF dilation

Renal causes

- a) Glomerular →
- b) Vascular → HUS
- c) Tubular →
  - → Blockage →
- d) Drug →

Phases —. phase → from trigger to onset of effect on kidney

—. phase → C/F of AKI → Fluid retention ↓ Edema/ ↑ BUN

OR. [2 days - 2 weeks]

→ Renal replacement

—. phase —→unable to. ↓ urine
(2 - 3 week)

— Recovery phase →

SUMMARY

	Per-Renal	Post-Renal
S.creatinine -		
[</>] U. Na -	20 mEq/lit	20 mEq/lit
[</>] Fe Na -	1%	2%
[</>] Fe urea -	35%	35%
[</>]U. Osmol. -	[500]	[400]
[</>]S. Gravity -	1.018	1.018
	cast	_____ cast

CHAPTER 7
CHRONIC KIDNEY DISEASE

CHRONIC KIDNEY DISEASE

Risk factor
- → DM [mcc]
- → HTN/Family history
- → Recurrent UTI
- → Premature birth [↓ Nephron reserve]

Stage of CKD	GFR	Albuminuria
I	>90 ml/min	A1 - <30 mg/dl
II	60-89 ml/min	A2 - 30-300 mg/dl
IIIa	45-59 ml/min	A3 - >300 mg/dl
IIIb	30-44 ml/min	
IV	15-29 ml/min	
Renal failure/V	0-14 ml/min	

Apparent ↓ in GFR → II

formula

C/F
- Fluid retention -
- BP —
- Sign of azotemia
- Impotence.
- CKD →... Ca^{+2} & active vit.D, ……

↓

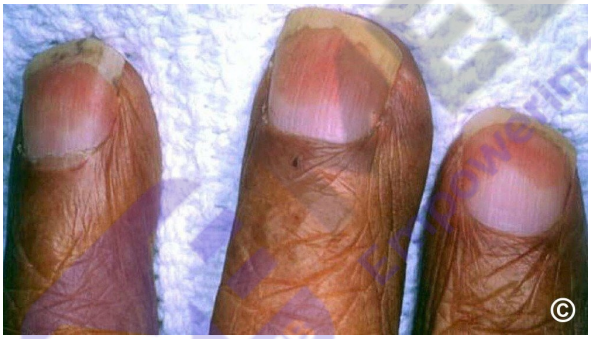
PTH stimulation

PTH ——→ osteoclast

↓

Renal

O/E —



Rx

Inv. — BP / BS — Control BS →

— S. creatinine. — RAAS# to ↓ Proteinuria

— Hb. ——→ — Ist correct —. level

— S. Ca^{+2} S. PO_4 IInd - agent

→ Calcimimetic —

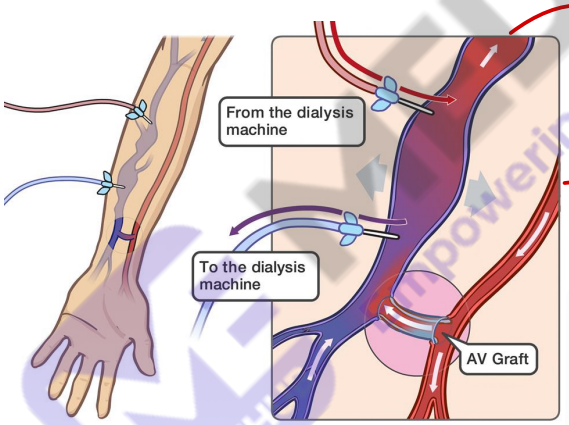
— Vit. D3. ——→ →PO binder

K^+ → K^+ intake
→ K^+ binders →

For acute Hyperkalemia →

Dialysis — Indication — A - [pH < 7.1]
E - [refractory hyper K^+]
I -
O -
U -

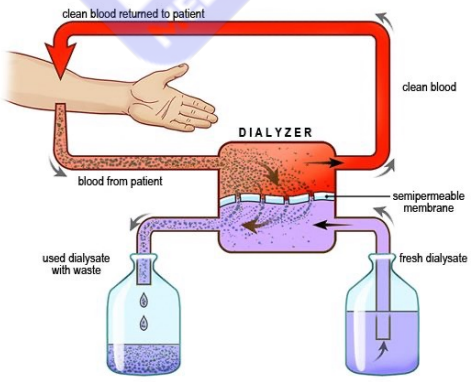
HEMODIALYSIS —— AV fistula — Arterialization of vein

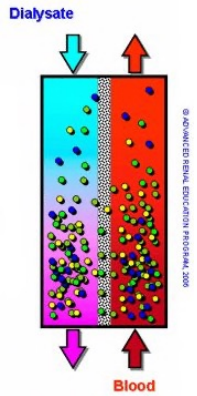


Cephalic vein

Radial artery

- AV fistula — patency check -





- MCC of death on Ch. hemodialysis —
- Ch. hemodialysis — aluminium toxicity — (from diasylate)

SUMMARY

CHAPTER 8
RENAL TRANSPLANT & RENAL CYSTIC DISEASE

RENAL TRANSPLANT

- From donor → Kidney
- Recipient →
- HLA & Blood group matching
- MC infection after transplant –

CYSTIC KIDNEY DISEASE

- Autosomal dominant poly cystic kidney disease [ADPKD]
  - d/t → PKD 1 gene → (Polycystin - 1) →
  - d/t → PKD 2 gene → → (Polycystin - 2)

△ → cyst in one kidney & in another

C/F
- ↑ Renal size -
- Hematuria, HTN
- Recurrent UTI
- Extra renal →
  - →
  - →

  [(NO) involvement]
- Can convert into CKD →

Rx →
→
[Risk of malignancy] Q

AUTOSOMAL RECESSIVE POLYCYSTIC KIDNEY DISEASE

- Ch - defect
- C/F → kidney palpable at birth
  → Hepatic fibrosis
  → Pul. Hypoplasia

JUVENILE NEPHROPATHIES

↓

Defect in

↓

Cyst +

J. NEPHROPATHIES + R. PIGMENTOSA

↓

Senior-Loken syndrome

- Medullary cystic disease → d/t. defect
  → cyst + Renal stone
- Medullary sponge disease → cyst + Renal stone

ALPORT SYNDROME

↓

chain of defect

↓ [type IV]

(IR) thinning of basement membrane

↓

GOOD - PASTUER SYNDROME

Pathogenesis:

HLA-DR-B1-15 Defect ⟶ autoantibody against

- → if only Renal —›
  involvement
- → Renal+lung Involvement —›

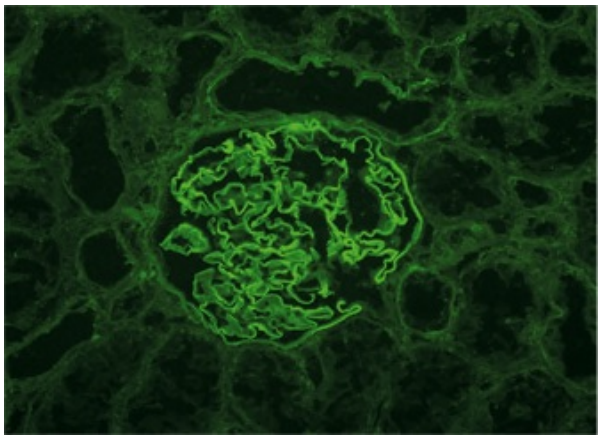
M:F
6:1

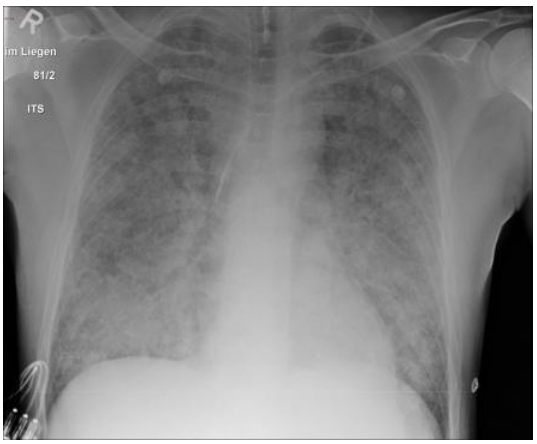
C/F →
- →
- →
- →

Patho. → Diffuse proliferative G.N. → Crescent Formation →

IF — linear deposit along basement membrane

- ANCA
- CxR -





Rx →
- →
- →

SUMMARY