

## ✓ CKD

According to the K/DOQI (Kidney Disease Outcomes Quality Initiative): CKD =

- Kidney damage for **≥3 months** defined by:
  - Structural or functional abnormalities
  - With OR without decreased GFR

Damage is diagnosed by either:

- Pathological abnormalities** (biopsy)
- Markers of kidney damage:**
  - Blood tests (e.g., ↑urea, ↑creatinine, ↓Hb)
  - Urine tests (e.g., proteinuria, hematuria)
  - Imaging abnormalities (e.g., small echogenic kidneys)

OR:

- GFR ≤60 mL/min/1.73m<sup>2</sup> for ≥3 months (with or without above markers)

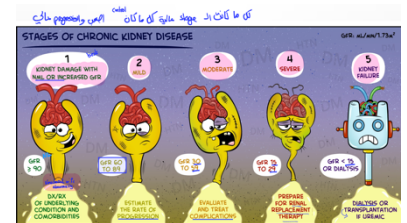
Albuminuria stages, description and range				
A1				
Normal to mildly increased				
A2				
Moderately increased				
A3				
Severely increased				
<3 mg/100ml				
3-29 mg/100ml				
≥30 mg/100ml				

GFR	Stage 1 (G1)	Stage 2 (G2)	Stage 3 (G3a)	Stage 3 (G3b)	Stage 4 (G4)	Stage 5 (G5)
Normal or high	≥90					
Mildly decreased		60-89				
Mildly to moderately decreased			45-59			
Moderately to severely decreased				30-44		
Severely decreased					15-29	
Kidney failure						<15

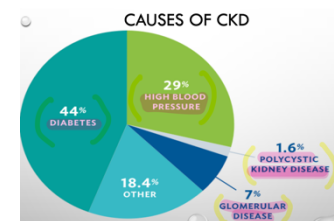
## STAGES OF CKD:

Stage	GFR (mL/min/1.73m <sup>2</sup> )	Notes
Stage 1	≥90	Normal GFR + persistent hematuria/proteinuria
Stage 2	60–89	Mild ↓GFR + persistent markers
Stage 3	30–59	Moderate GFR ↓
Stage 4	15–29	Severe GFR ↓
Stage 5	<15 or on dialysis	End-Stage Renal Disease (ESRD)



## CAUSES OF CKD:

- Diabetes mellitus (MC cause)
- Hypertension
- Glomerulonephritis (e.g., SLE, IgA)
- Polycystic kidney disease
- Chronic interstitial nephritis (e.g., NSAID-induced)
- Obstructive uropathy



## PROGRESSION OF CKD:

**Monitoring GFR decline helps predict the time to (ESRD) and evaluate treatment effectiveness.**

- GFR declines **progressively over time**
- Must estimate GFR decline to:
  - Predict when ESRD will occur
  - Assess effectiveness of therapy to slow GFR

How?

- Track **serum creatinine (past and ongoing)**
- Identify **risk factors** for fast progression (e.g., proteinuria, hypertension)
- Consider interventions in **all** CKD patients

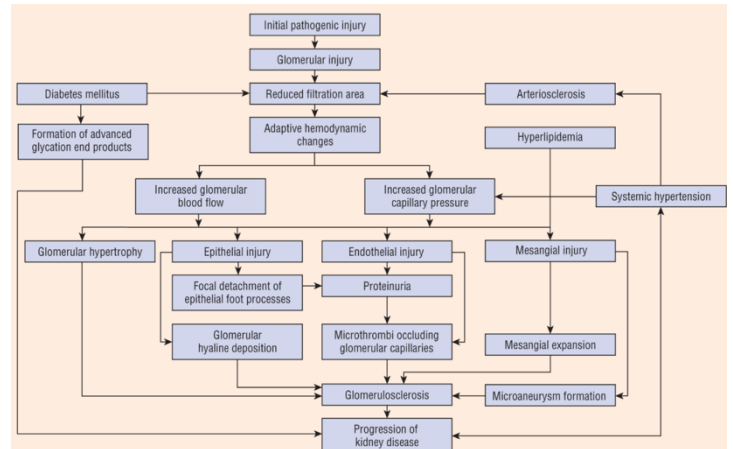
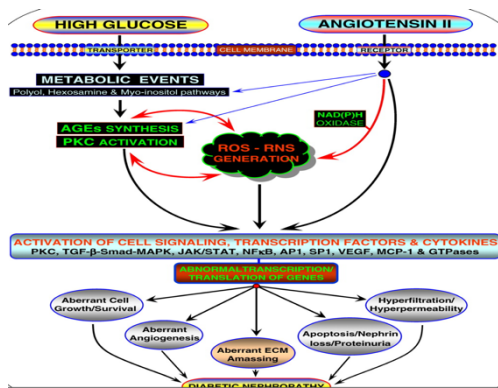
Creatinine should be checked **yearly** at minimum, or more frequently if:

- GFR <60
- Past GFR decline >4 mL/min/year (past)
- Exposure to risk factors for acute GFR decline and for faster progression
- On therapy to slow progression

## CKD PATHOPHYSIOLOGY:

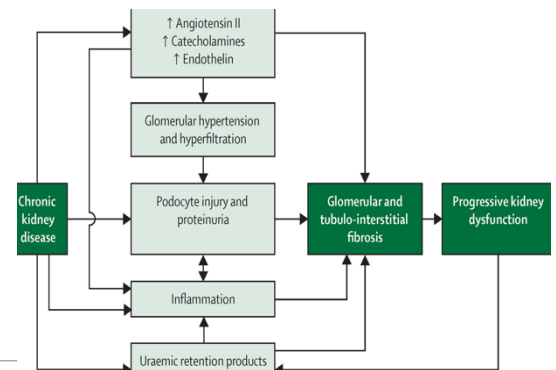
- Initial Injury: e.g. GN, ATN, AIN, PCKD
- ➡ Loss of nephrons (parenchyma) ➡ Compensatory hyperfiltration ( ↑ PGC (Glomerular pressure) ➡ ↑ SNGFR ➡ ↑ Wall stress ➡ ↑ TGF-β
- Long-term damage to remaining nephrons

#### 4. Proteinuria → progressive renal insufficiency



#### HEMODYNAMIC ADAPTATION:

- Intraglomerular hypertension ( $\uparrow$ PGC) = compensation (to nephron loss, maintain GFR)
- Due to:
  - Afferent vasodilation ( $\uparrow$  BF)
  - Efferent vasoconstriction ( $\downarrow$  blood outflow)
- Fall in GFR is minimized by  $\uparrow P_{GC}$
- Response?  $\downarrow$  flow to the macula densa (due  $\downarrow$  nephron fx) → trigger: activation of TGF



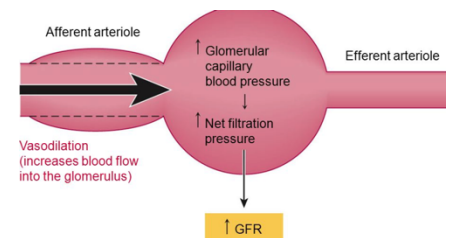
#### MEASUREMENT OF GFR:

##### Common Methods:

- Serum creatinine
- eGFR (MDRD or Cockcroft-Gault)
- Creatinine clearance ( $\pm$  cimetidine)
- Inulin clearance (gold standard) → research
- Radionuclide markers
- Cystatin C

##### Ideal GFR Marker Should Be:

- Constantly produced
- Safe and convenient
- Rapidly diffusable in ECF
- Not protein-bound, freely filterable
- Not reabsorbed or secreted
- No extrarenal elimination or degradation
- Minimal interference with other compounds
- Cheap and accurate



(c) Arteriolar vasodilation increases the GFR

#### EQUATIONS:

##### Cockcroft-Gault

##### MDRD:

#### CYSTATIN C:

- 13 kDa protein made by all nucleated cells

- expensive
- Constantly produced
- Filtered by glomerulus
- Metabolized in PCT
- **Unaffected** by muscle mass or diet
- Still investigational

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#### CKD INVESTIGATION:

- Urinalysis → proteinuria, hematuria, RBC casts <active urine>
- US → kidney size (small = chronic) // large = AKI , polycystic
- Hb, mineral metabolism (check for anemia , common in CKD due to ↓EPO production )
- Past Cr values
- Follow – up serum cr

#### Who Should Get a Biopsy?

- Young > Old (HEREDITARY NEPHRITIS, FABRY ETC.)
- Early > Late
- Hematuria > bland urine
- Heavy proteinuria > none
- Normal kidney size > small
- No clear etiology > DM/HTN/ vascular disease

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#### FACTORS THAT ACCELERATE CKD:

- Hypertension
- Proteinuria
- Hyperlipidemia
- High dietary protein
- Angiotensin II / Aldosterone
- Metabolic acidosis
- Hyperphosphatemia
- Hyperuricemia


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#### GENERAL CKD MANAGEMENT:

1. Treat reversible causes
2. Prevent / slowing progression
3. Treat complications
4. Educate and prepare for RRT


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#### HOW TO SLOW PROGRESSION:

- ↓ Proteinuria (goal <0.5–1 g/day) (PCR < 60)
  - Start ACEi or ARB (combine if needed)
  - SE for combination ? AKI / hyperkalemia
- ↓ BP (target <130/80)
  - Start ACEi or ARB
  - Add diuretic or other meds if needed
- ↓ Dietary protein (0.8–1 g/kg/day)
- Manage lipids, metabolic acidosis ( $\text{HCO}_3^- = 22$ )
- STOP smoking 

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#### CKD COMPLICATIONS:

-  Volume overload

- Na restriction, diuretics

#### Hyperkalemia

- Low K diet, diuretics, Kayexalate (→ bowel perforation → ischemia)

#### Acidosis

- HCO<sub>3</sub> goal = 22 mmol/L
- NaHCO<sub>3</sub> (0.5–1g PO BID–TID) // ps: don't initiate before controlling na + BP

#### Hyperphosphatemia

- Stage 3 CKD onward
- Target PO<sub>4</sub> = 0.87–1.49 mmol/L
- Low diet
- PO<sub>4</sub> binders: CaCO<sub>3</sub>, Sevelamer

#### renal osteodystrophy

- ↑PTH from Stage 2–3 CKD
- Target depends on GFR → Avoid oversuppression (→ adynamic bone disease)
- Vit D3 drops with stage 3
- Calcitriol 0.25 mcg/day
- PO<sub>4</sub> binders: CaCO<sub>3</sub>, Sevelamer

#### Anemia

- Stage 3 CKD (common)
- Exclude non renal causes
- Treat with EPO + iron

#### HTN

- Target <130/80
- ACEi/ARB ± loop, non-dihydropyridine CCB

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### Why early referral is important:

- Helps choose the right **dialysis method** 🇩🇪
- Allows time to place **proper dialysis access** (like a fistula) 📌
- Dialysis can start **calmly, not in an emergency** 🚫📞
- **Fewer complications** and better recovery 🧠💪
- **Shorter and fewer hospital stays** 🏥
- **Lower healthcare costs** 💰
- **Better survival rates** ❤️

💡 Bottom line: Early referral = smoother treatment, healthier patient, happier kidneys.

#### WHEN TO REFER TO NEPHROLOGY:

- AKI
- eGFR <30
- Rapid decline in function
- Proteinuria >500 mg/day (PCR >60) // PRESENT ON 2 OF 3 SAMPLES
- BP not at target
- Unable to use renoprotective meds
- Education for dialysis/transplant

⚠️ Late Referral = seen within 1–6 months before needing RRT (bad outcomes!)

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#### 17 PREP FOR DIALYSIS: Start at **Stage 4 CKD (eGFR ≤30)**

- Begin 1 year before expected start
- Educate about modalities (PD, HD, transplant)
- Promote autonomy & home options

#### Access Placement:

- HD: AV fistula ~3 months before

- PD: PD catheter ~1 week before

#### 🕒 WHEN TO INITIATE DIALYSIS:

- Refractory volume overload
- Hyperkalemia ( $K > 6$ ) not controlled by meds(keyexalate) /diet
- Uremia signs (e.g., pericarditis, confusion)
- Metabolic Acidosis unresponsive to  $\text{NaHCO}_3$
- Not based only on eGFR!

#### 💡 STAGE 5 OPTIONS:

- Pre-emptive living donor transplant
- Home options:
  - Peritoneal dialysis
  - Nocturnal home HD
- In-center HD
- Self-care

#### 📌 HEMODIALYSIS:

- Conventional: 4h, 3x/week
- Short daily: 2–3h, 4–6x/week
- Nocturnal: 6–8h, 3–6x/week

#### 📌 Access:

- AV fistula (best)
- AV graft
- Internal jugular cuffed catheter (temp) → UC cath

#### 💧 PERITONEAL DIALYSIS:

- CAPD: 4–5 manual exchanges daily 2-2.5L/D
- CCPD: Automated overnight + daytime dwell

### APPROACH TO DIAGNOSIS AND TREATMENT AT THREE STAGES

