Kidney Disease 101

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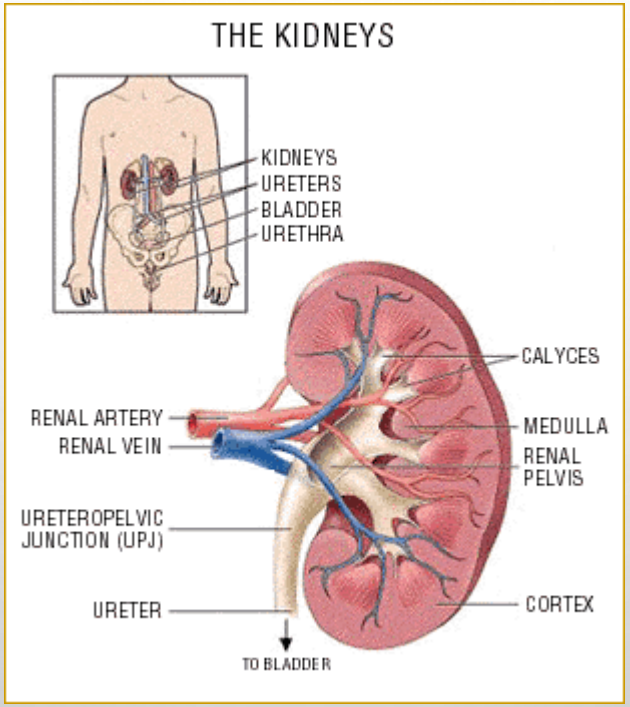
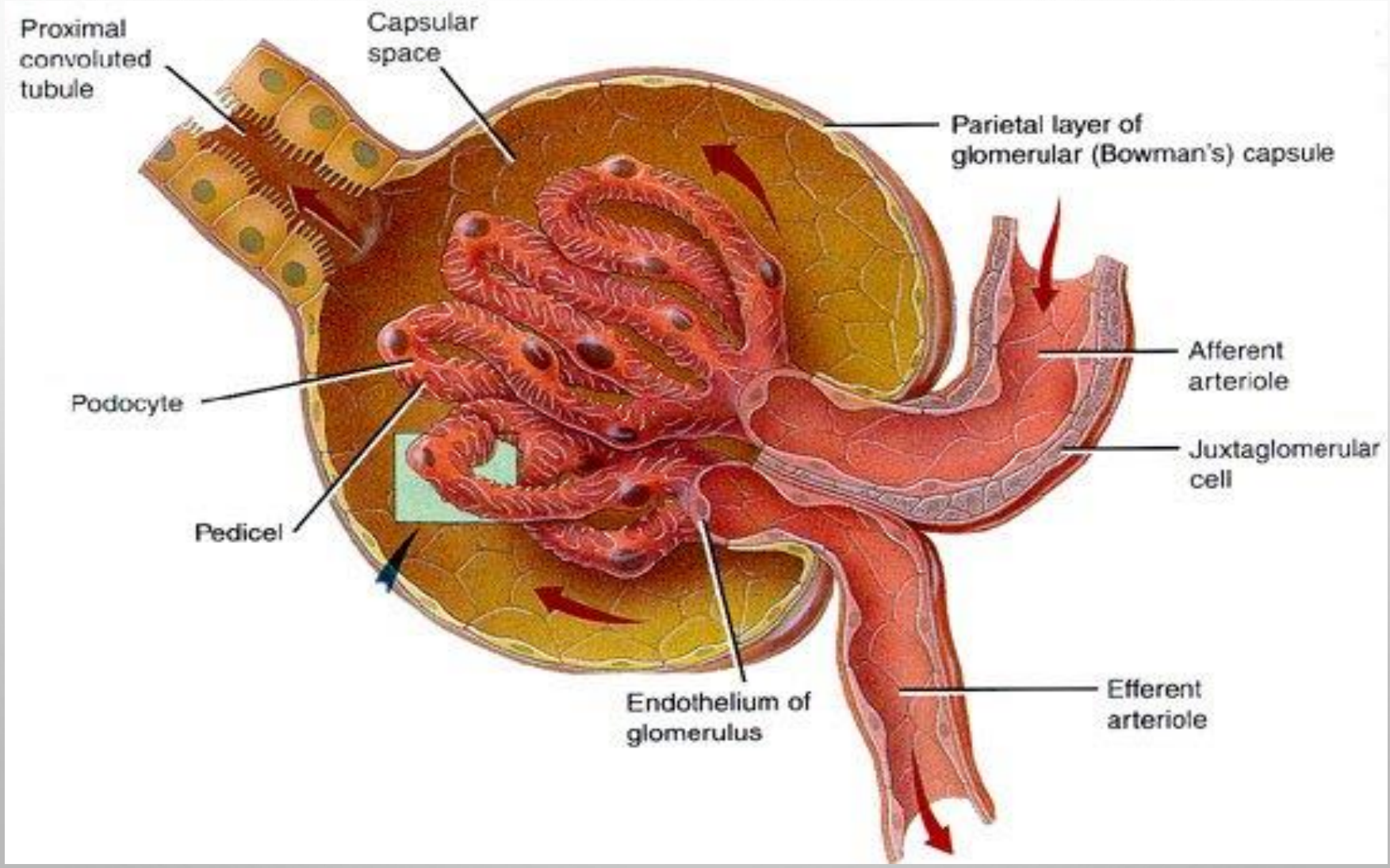
Objectives

- 1. Renal Anatomy and Physiology
- 2. Key Renal Concepts
- 3. Diagnostic Tests
- 4. Acute Kidney Injury
- 5. Chronic Kidney Disease
- 6. End Stage Renal Disease
- 7. Medications for kidney preservation

Anatomy


THE KIDNEYS
KIDNEYS
URETERS
BLADDER
URETHRA
CALYCES
RENAL ARTERY
RENAL VEIN
MEDULLA
RENAL PELVIS
URETEROPELVIC JUNCTION (UPJ)
URETER
CORTEX
TO BLADDER

Glomerulus:


Proximal convoluted tubule
Capsular space
Parietal layer of glomerular (Bowman's) capsule
Afferent arteriole
Podocyte
Juxtaglomerular cell
Pedicel
Endothelium of glomerulus
Efferent arteriole

The various functions of the kidneys

- Electrolyte Balance
  - Potassium, Sodium, Phosphorus, Calcium, magnesium
- Excretion of Body Waste
  - Blood Urea Nitrogen (BUN), Creatinine (Cr), Drugs
- Acid/Base Balance
- Endocrine Organ- various hormone functions
  - PTH (parathyroid hormone), Vitamin D, Erythropoietin

Key labs related to the kidneys

Blood Urea Nitrogen (BUN)

- Reflection of catabolism & protein ingestion
- Exists in a 10:1 relationship with Creatinine
- Normal value 6-20 mg/dl
- Less specific indicator of kidney function
- Elevated in certain states
  - ➢ Steroid, TPN, GI Bleed, Dehydration

Creatinine (Cr)

- Reflection of muscle metabolism therefore various depending on muscle mass and body size
- Freely filtered @ the glomerulus
- Excellent indicator of kidney function
- Normal level 0.5-1.2 mg/dl

Creatinine Clearance

- Indirect measurement of Glomerular Filtration Rate (GFR)
- Estimated with various equations

Name	Equation characteristics
Cockcroft-Gault	• Tends to overestimate true GFR • Less accurate at GFR≥60 ml/min/1.73 m^2
MDRD	• Tends to underestimate true GFR at GFR≥60 ml/min/1.73 m^2 • Less accurate at GFR≥60 ml/min/1.73 m^2
CKD-Epi	• Preferred equation for estimating GFR • Tends to underestimate true GFR at GFR≥60 ml/min/1.73 m^2 (but less so than MDRD equation) • Less accurate at GFR≥60 ml/min/1.73 m^2

Glomerular Filtration Rate (GFR)

- Healthy adult has a GFR of 100-125 ml/min
- Decreases by one mL/min/year after the age of 40
- Abrupt decrease in GFR will show an increase in Cr
- Gradual decrease in GFR with aging will show little change in Cr…why?

Creatinine and GFR

- Is a Cr of 1.2 in a 20 year old 100 kg male the same as a Cr of 1.2 in an 80 year old 50 kg female?

99.2/min/1.73m2

45.9mL/min/1.73m2

ASN TASK FORCE RECOMMENDATION FOR ESTIMATED GFR

1. ASN TASK FORCE RECOMMENDED THE IMMEDIATE ADOPTION OF THE 2021 CKD-EPI CREATININE EQUATION THAT ESTIMATES KIDNEY FUNCTION **WITHOUT** A RACE VARIABLE.
2. RECOMMENDED INCREASED USE OF CYSTATIN C COMBINED WITH SERUM CREATININE TO ESTIMATE GFR WHEREVER POSSIBLE
3. COMBINING BOTH THE SERUM CREATININE AND CYSTATIN C INTO A SINGLE EQUATION APPEARS TO CONSISTENTLY PROVIDE MORE PRECISE EGFR THAN EQUATIONS THAT USE EITHER CREATININE OR CYSTATIN C ALONE
4. IF UNSURE A 24 HOUR CREATININE CLEARANCE IS A TRUE MEASUREMENT NOT AN ESTIMATION
5. CERTAIN INSTITUTIONS OFFER NUCLEAR MEDICINE CLEARANCE

Blood Abnormalities

- CBC/Hemoglobin
- Sodium 135-145 mEq/L
- Potassium 3.5-5.0 mEq/dl
- Chloride 95-112 mEq/L
- Bicarbonate 22-30 mEq/L
- Phosphorus 3.0-5.0 mEq/dl
- Calcium 8.5-10.0 mEq/dl
- BUN/Cr/GFR

Urinary Assessment

- Random or “spot” sample adequate
- Sediment
  - Casts, red cells, white cells

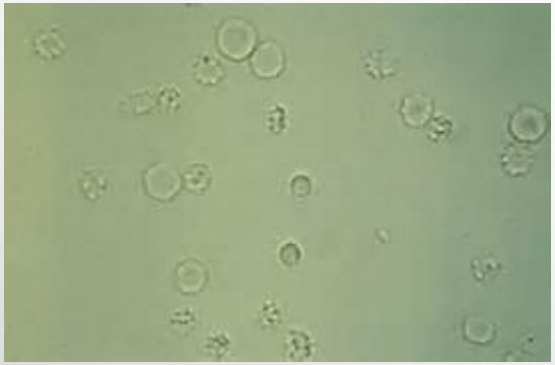
- Proteinuria
  - Urinary excretion of albumin & other proteins
  - Albumin specific dipstick for detecting albuminuria
  - Early and sensitive marker of kidney disease

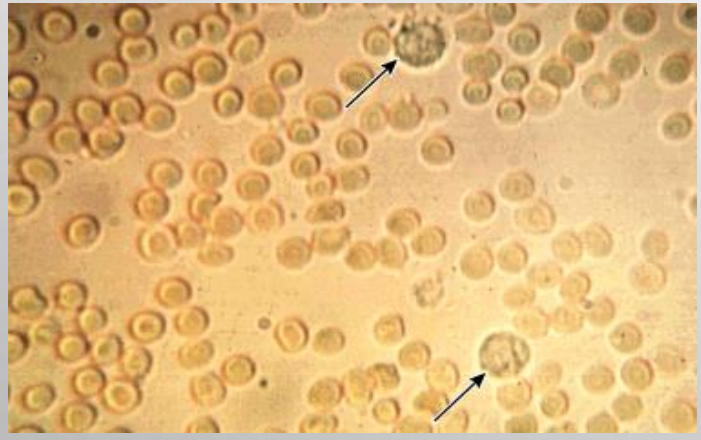
Urinary Microanalysis

- Erythrocytes (RBCS) > 2-3 per HPF
  - Originating in renal parenchyma, dysmorphic
  - Orginating in collecting system, retain shape
- Leukocytes (PMNs)
  - Indicate urinary tract inflammation
  - Upper or lower tract UTI
- Renal Tubular Epithelial Cells
  - May be seen in normal urine sample
  - More commonly indicate tubular damage or damage from ATN or interstitial nephritis

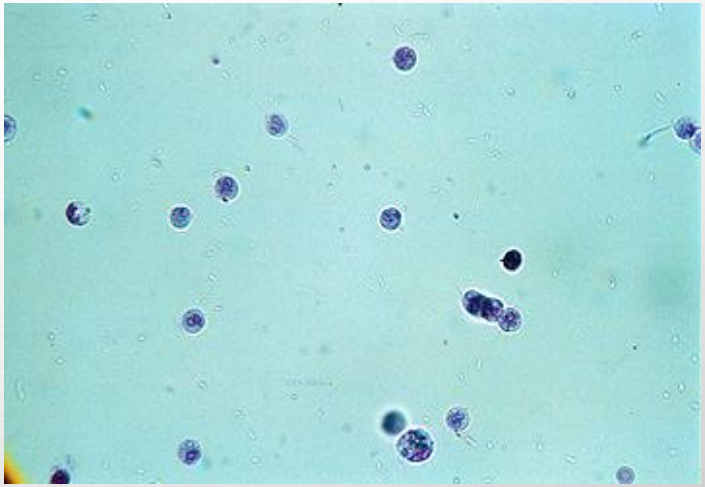
Urine Microanalysis

- Red Blood Cells





- White Blood Cells



UA Casts

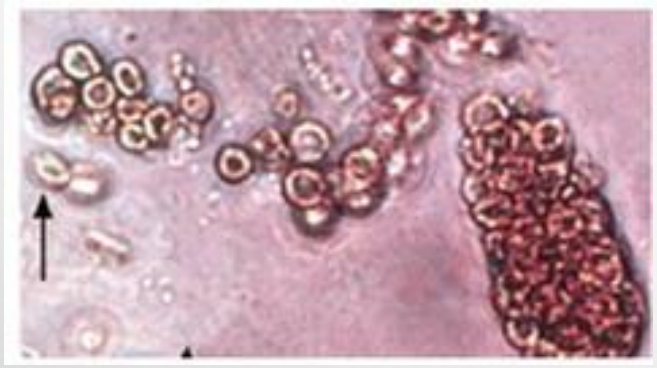
- Hyaline Casts
  - Protein alone
  - Nonspecific, can be seen in concentrated urine as well as pathologic conditions
- Granular Casts
  - Fine or coarse granulated material
  - Nonspecific but usually pathologic
  - May be seen after exercise, with simple volume depletion, ATN, GN, tubulointerstitial disease
- Waxy Casts
  - Forms in tubules that have become dilated and atrophis due to chronic parenchymal disease

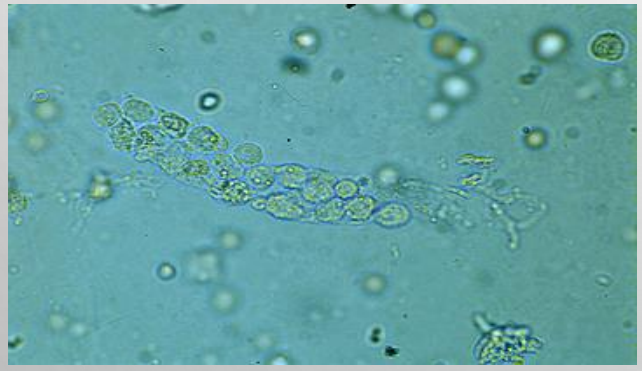
UA Casts

- RBC Casts
  - Interparenchymal bleeding
  - Hallmark if GN, less frequently in tubulointerstitial disease
- WBC Casts
  - WBC in protein matrix
  - Characteristic of pyelonephritis
  - May be seen in interstitial nephritis and other tubulointerstitial disorders
- Tubular Cell Casts
  - Sloughing tubular cells
  - Concentrated urine
  - More charecteristic of sloughing tubular cells occurring in ATN

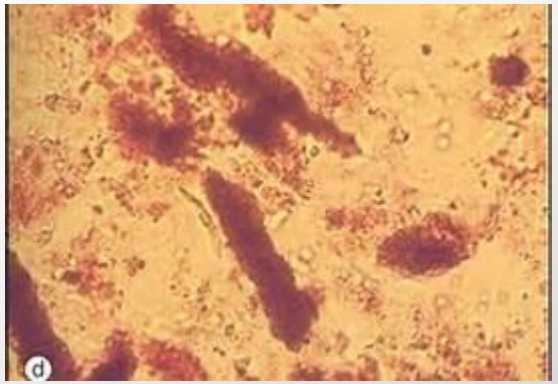
CASTS

- Red and White Cell





- Granular



Urine Protein

- Most consistent results from 1st AM voided specimen
- Abnormal albumin/creatinine ratio >30mg/g
- Abnormal proteinuria from:
  - Glomerular pathology
  - Tubular pathology
  - Overflow of an abnormal plasma protein
  - Pathologic protein secretion from urinary tract (hemoglobin, myoglobin or monoclonal gammopathies)

Proteinuria

- Urinary excretion of protein including albumin and other proteins
- Albuminuria is early and sensitive marker of CKD and cardiovascular mortality

Renal US

- Size of kidneys, presence of pathology
- Obstruction/hydronephrosis
- Renal artery flow
- Stones
- Bladder
- Prostate

Chronic Kidney Disease
CKD

- Classification system developed by National Kidney Foundation
- Allows providers to identify patients at risk and begin treatment
- Ultimate goal to slow progression of disease and delay ESRD
- Healthy People 2020: Reduce new cases of chronic kidney disease (CKD)and its complications, disability, death, and economic costs

Chronic kidney disease classification based upon glomerular filtration rate and albuminuria

GFR stages	GFR (mL/min/1.73 m^2)	Terms
G1	≥90	Normal or high
G2	60 to 89	Mildly decreased
G3a	45 to 59	Mildly to moderately decreased
G3b	30 to 44	Moderately to severely decreased
G4	15 to 29	Severely decreased
G5	<15	Kidney failure (add D if treated by dialysis)
Albuminuria stages	**AER (mg/day)**	**Terms**
A1	<30	Normal to mildly increased (may be subdivided for risk prediction)
A2	30 to 300	Moderately increased
A3	>300	Severely increased (may be subdivided into nephrotic and non-nephrotic for differential diagnosis, management, and risk prediction)

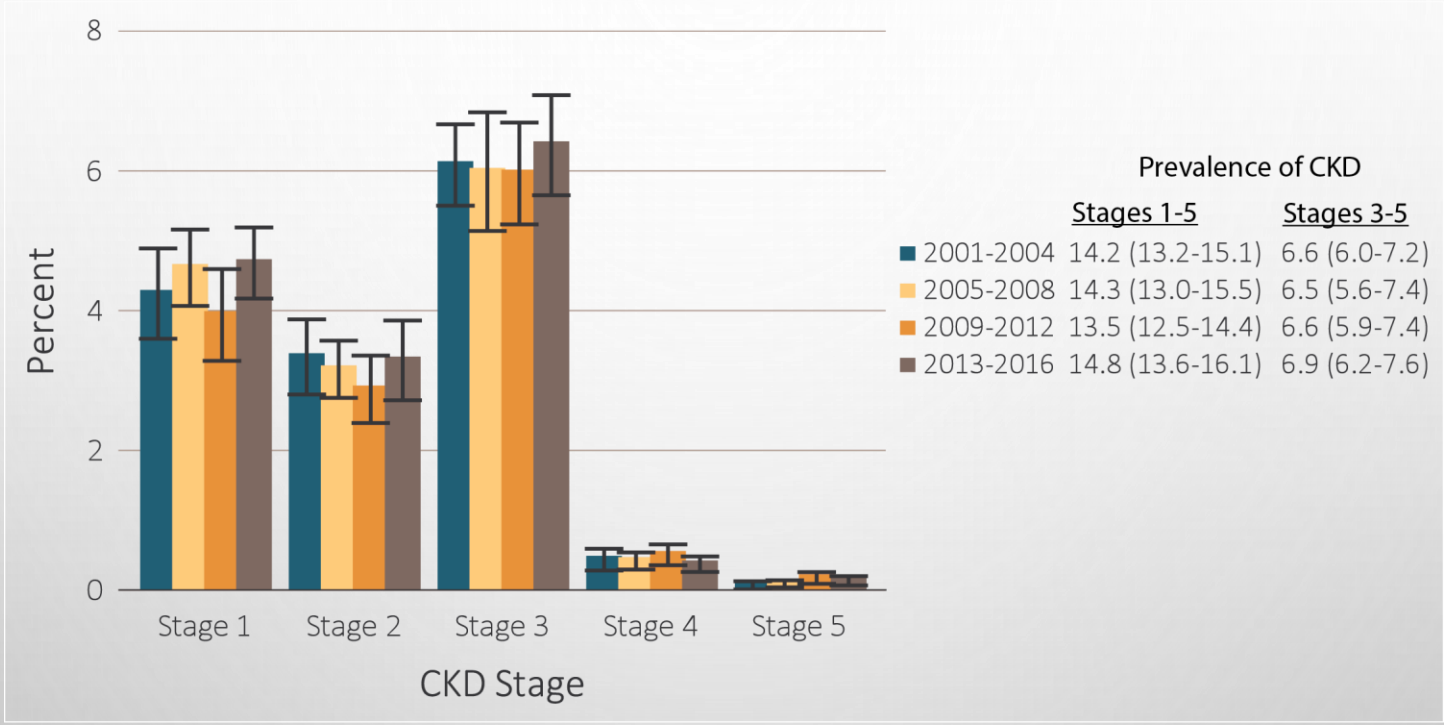
The cause of CKD is also included in the KDIGO revised classification but is not included in this table.

GFR: glomerular filtration rate; AER: albumin excretion rate; CKD: chronic kidney disease; KDIGO: Kidney Disease Improving Global Outcomes.

Data from:

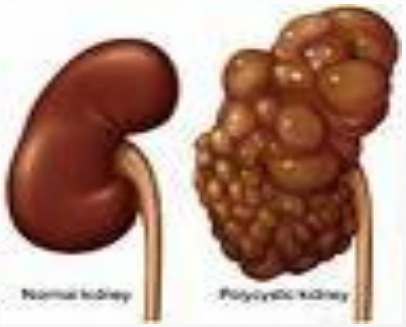
1. *KDIGO. Summary of recommendation statements. Kidney Int 2013; 3 (Suppl):5.*
2. *National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis 2002; 39 (Suppl 1):S1.*

VOL 1 FIGURE 1.1 PREVALENCE OF CKD BY STAGE AMONG NHANES PARTICIPANTS, 2001-2016



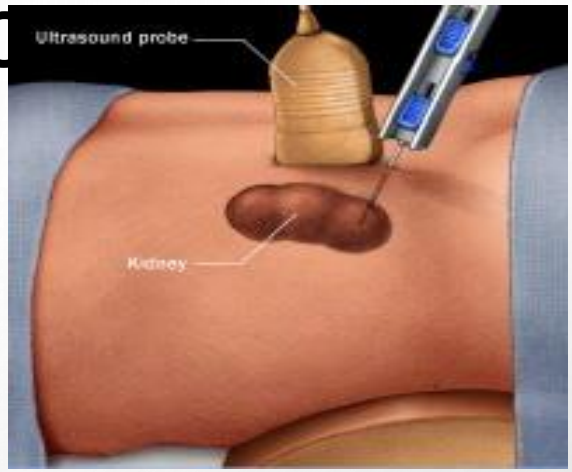
Data Source: National Health and Nutrition Examination Survey (NHANES), 2001-2004, 2005-2008, 2009-2012 & 2013–2016participants aged 20 & older. Whisker lines indicate 95% confidence intervals. Abbreviation: CKD, chronic kidney disease.

Renal Ultrasound



- Simple, noninvasive, low risk, relatively inexpensive
- Identify renal mass, obstruction, size, ureters, bladder and structural abnormalities
- One of the initial tests done in evaluation of renal disease
- More difficult with obese patients

Kidney Bio



- Invasive with risk
- Done when a definitive diagnosis will determine plan of care
- Needle injected into retroperitoneal space, lower pole on inspiration

Outcomes of Early Screening

- Delayed Progression of Kidney Disease
  - Blood pressure control
  - Treat proteinuria
  - Blood sugar control , A1C <7.0%
  - Avoid nephrotoxins
  - Treat dyslipidemia
  - Lifestyle modifications
    - Weight loss
    - Exercise
    - Smoking cessation
    - Treating sleep apnea

Why Treat CKD?

- GOAL TO PREVENT or SLOW PROGRESSION TO ESRD
- Poor Patient Outcomes
  - Kidney disease one of the top 10 causes of death in the United States
  - Annual mortality rate for ESRD approaches 20%
    - Average life expectancy of a 59 year old is 20.4 years
    - Average life expectancy of the same individual with ESRD is 4.3 years

Why Treat CKD and Prevent ESRD? Survival in ESRD

II 6.b Expected remaining lifetimes (yrs) of the U.S. population & of dialysis & transplant patients, by age, gender, & race general population, 2004, & prevalent dialysis & transplant pts, 2006

	General U.S. population, 2004									ESRD patients, 2006					
	All races			White			African American			Dialysis			Transplant		
	All	M	F	All	M	F	All	M	F	All	M	F	All	M	F
0-14	71.4	68.8	73.9	71.8	69.2	74.3	67.2	63.7	70.3	19.8	20.2	19.4	53.3	52.8	54.1
15-19	61.6	59.1	64.1	62.0	59.5	64.4	57.5	54.0	60.6	16.8	17.7	15.9	41.5	41.0	42.3
20-24	56.9	54.4	59.2	57.2	54.8	59.5	52.7	49.4	55.7	14.5	15.3	13.6	37.8	37.3	38.6
25-29	52.1	49.7	54.4	52.5	50.1	54.7	48.1	44.9	50.9	12.8	13.4	12.1	34.5	34.0	35.3
30-34	47.4	45.1	49.5	47.7	45.4	49.8	43.5	40.5	46.2	11.1	11.5	10.7	30.7	30.2	31.7
35-39	42.7	40.4	44.7	42.9	40.7	45.0	39.0	36.0	41.5	9.6	9.9	9.3	27.1	26.5	28.2
40-44	38.0	35.8	40.0	38.3	36.1	40.2	34.5	31.6	37.0	8.3	8.4	8.1	23.7	23.1	24.8
45-49	33.5	31.4	35.4	33.7	31.7	35.6	30.3	27.5	32.6	7.2	7.2	7.1	20.5	19.9	21.7
50-54	29.2	27.2	30.9	29.3	27.4	31.1	26.3	23.7	28.5	6.3	6.3	6.2	17.6	17.0	18.8
55-59	25.0	23.1	26.5	25.1	23.3	26.6	22.6	20.1	24.5	5.4	5.4	5.4	15.1	14.4	16.2
60-64	21.0	19.3	22.4	21.0	19.4	22.4	19.1	16.9	20.7	4.6	4.6	4.7	12.7	12.1	13.8
65-69	17.2	15.7	18.5	17.3	15.8	18.5	15.9	14.0	17.2	3.9	3.9	4.0	10.6	10.0	11.7
70-74	13.8	12.5	14.8	13.8	12.5	14.8	13.0	11.4	14.0	3.3	3.2	3.4	8.9	8.3	9.9
75-79	10.8	9.7	11.5	10.7	9.6	11.5	10.4	9.1	11.2	2.8	2.7	2.8	7.4	6.8	8.4
80-84	8.2	7.3	8.7	8.1	7.2	8.6	8.3	7.3	8.7	2.3	2.3	2.4			
85+	4.4	3.9	4.6	4.3	3.8	4.5	5.0	4.4	5.1	1.9	1.8	2.0			
Overall*	25.3	23.6	26.7	25.4	23.7	26.8	23.2	21.0	24.9	5.8	5.8	5.7	16.1	15.5	17.2

Complications of CKD

Cardiovascular Disease (CVD)

- CKD an independent risk factor for CVD
- American Heart Association states individuals with CKD are the “highest risk group” for CVD
- Death from CVD is 10-30 times more common in Stage 5 CKD than the general population

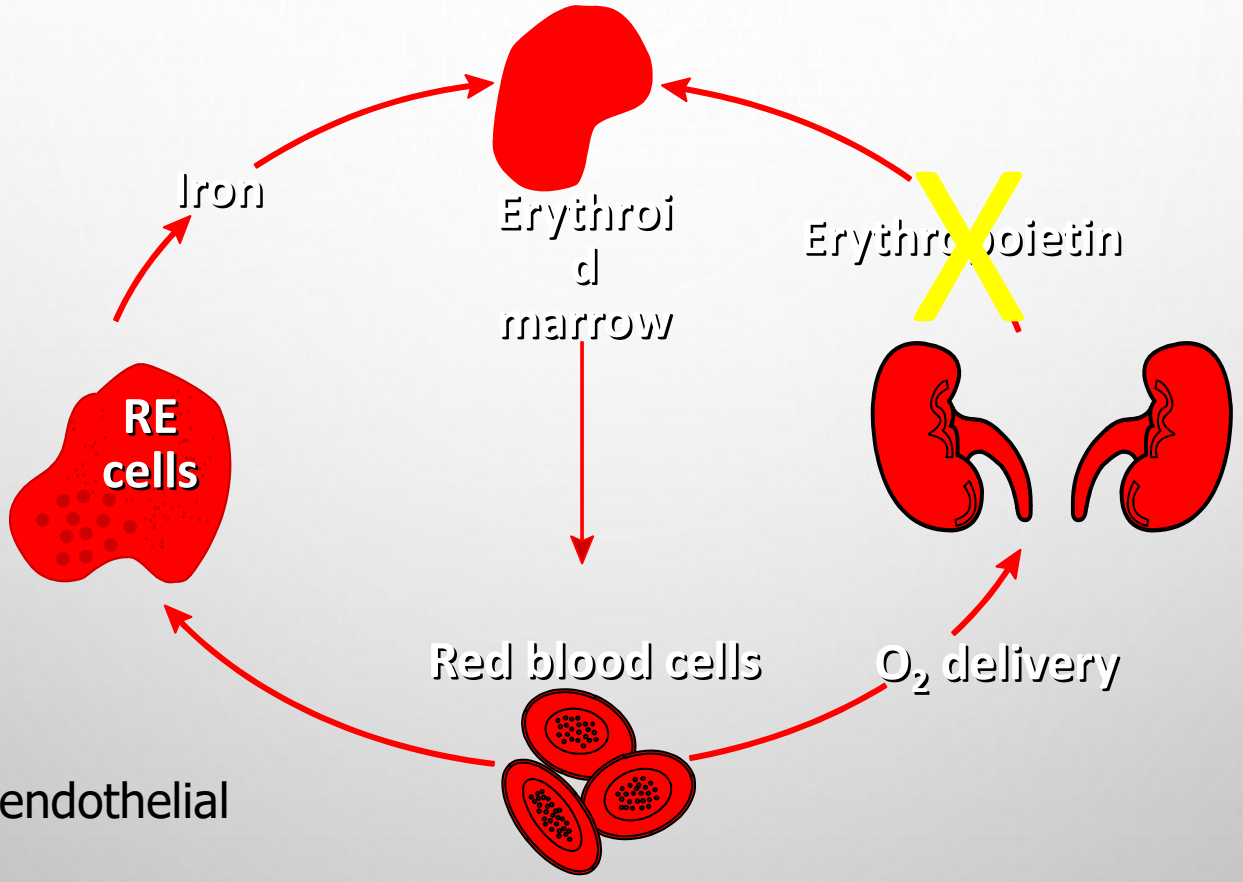
Complications of CKD: Hypertension

- Low salt diet
- Lifestyle modifications
- Medication
  - Angiotensin converting enzyme inhibitors or angiotensin II receptor blockers
  - Diuretics (thiazide early but loop diuretic with advanced CKD)
  - Calcium channel blockers
  - Beta blockers
  - Alpha blockers
  - MINERALOCORTICOID ANTAGONISTS
  - Other (central acting and direct acting)

HTN MANAGEMENT

- INDIVIDUALIZE BP TARGETS AND AGENTS ACCORDING TO AGE, COEXISTENT CARDIOVASCULAR DISEASE AND OTHER COMORBIDITIES, RISK OF PROGRESSION OF CKD
- THE TARGET BP FOR PATIENTS WITH HYPERTENSION AND DIABETES AND/OR CKD IS <130/80 MM HG -2012 ACC/AHA GUIDELINE
- ACEI OR ARB FIRST LINE AGENT IN CKD
- THIAZIDE DIURETICS ARE A CORNERSTONE OF HYPERTENSION TREATMENT BUT LOSE EFFICACY WITH SEVERE CKD.
- LOOP DIURETICS ARE PREFERRED IN PATIENTS WITH CKD WITH AN EGFR <30 ML/MIN/1.73 M

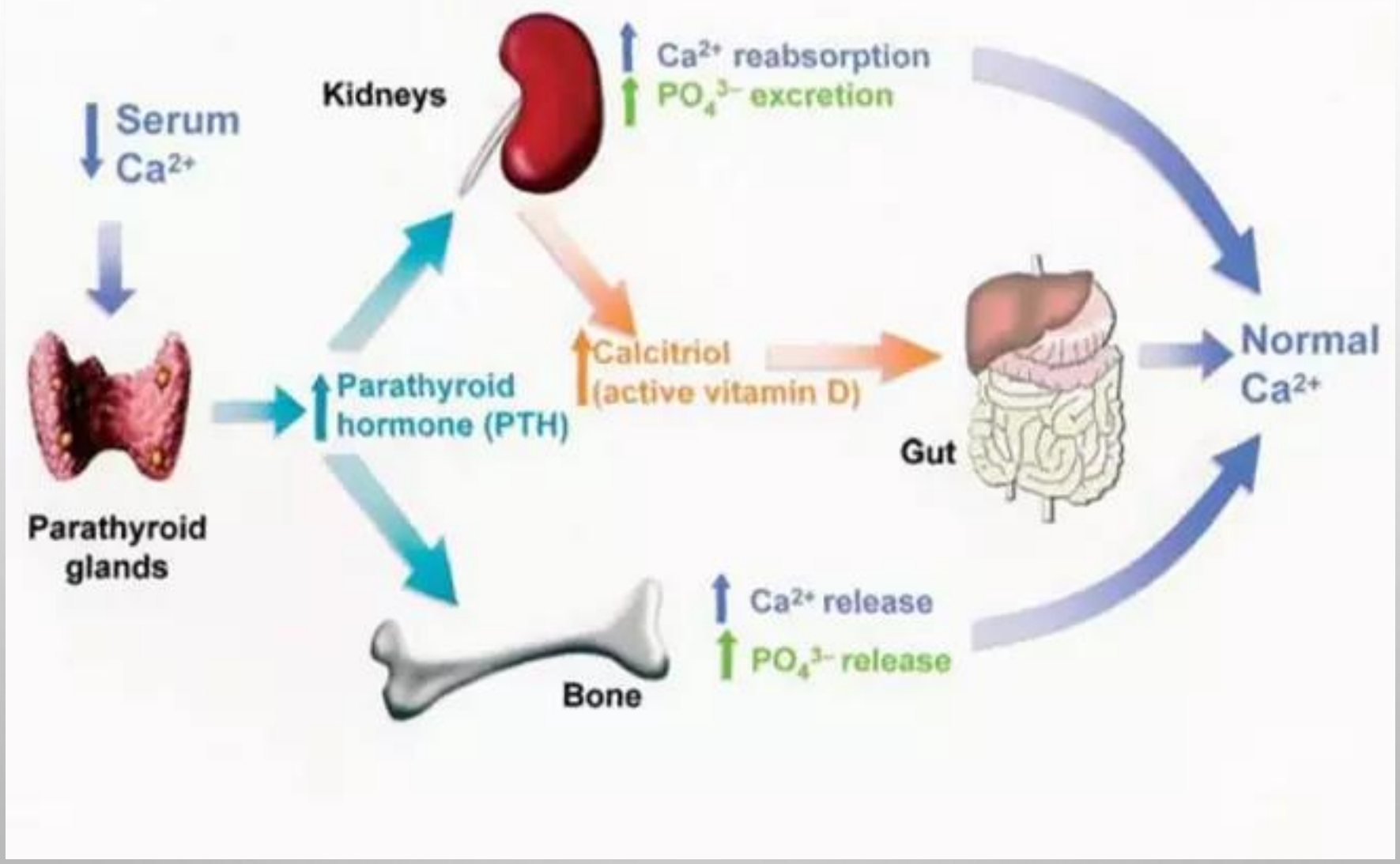
Complications of CKD: Anemia



RE=Reticuloendothelial

RE=reticuloendothelial

Complications of CKD: BMD



SECONDARY HYPERPARATHYROIDISM

- RESULT OF IMPROPER RENAL HANDLING OF PHOSPHOROUS, CALCIUM AND VITAMIN D
- PTH >600, HIGHER RISK FOR ALL-CAUSE AND CARDIOVASCULAR MORTALITY, AND CV HOSPITALIZATIONS
- MEDICATIONS:
  - PO CALCITRIOL, PARICALCITOL AND DOXERCALCIFEROL
  - PO CINACALCET
  - IV PARSABIV
- POSSIBLE PARATHYROIDECTOMY IF PTH UNCONTROLLED.

Common Meds to Treat Hyperphosphatemia in CKD

- Phosphate Lowering Therapy
  - Calcium Acetate (Phos Lo)
  - Calcium carbonate (Tums)
  - Sevelamer (Renvela/Renagel)
  - Lanthanum (Fosrenol)
  - SUCROFERRIC OXYHYDROXIDE (VELPHORO)
  - FERRIC CITRATE (AURYXIA)

HYPERKALEMIA

- PATIROMER (VALTESSA)
  - POTASSIUM BINDER
  - TREAT HIGH LEVELS OF POTASSIUM
  - BINDS TO OTHER MEDICATIONS IN GI TRACT
    - 3 HOURS BEFORE OR AFTER OTHER MEDICATIONS

- SODIUM ZIRCONIUM CYCLOSILICATE (LOKELMA)
  - POTASSIUM BINDER 10G ONCE A DAY
  - TREAT HIGH LEVELS OF POTASSIUM
  - TASTELESS AND ODORLESS
  - ADMINISTER OTHER MEDS 2 HRS BEFORE OR AFTER
  - AE MILD TO MODERATE EDEMA

Glycemic Control

- American Diabetes Association Recommends
  - A_1C< 7%
  - Preprandial glucose level 90-130 mg/dL
  - Peak postprandial glucose level <180 mg/dL

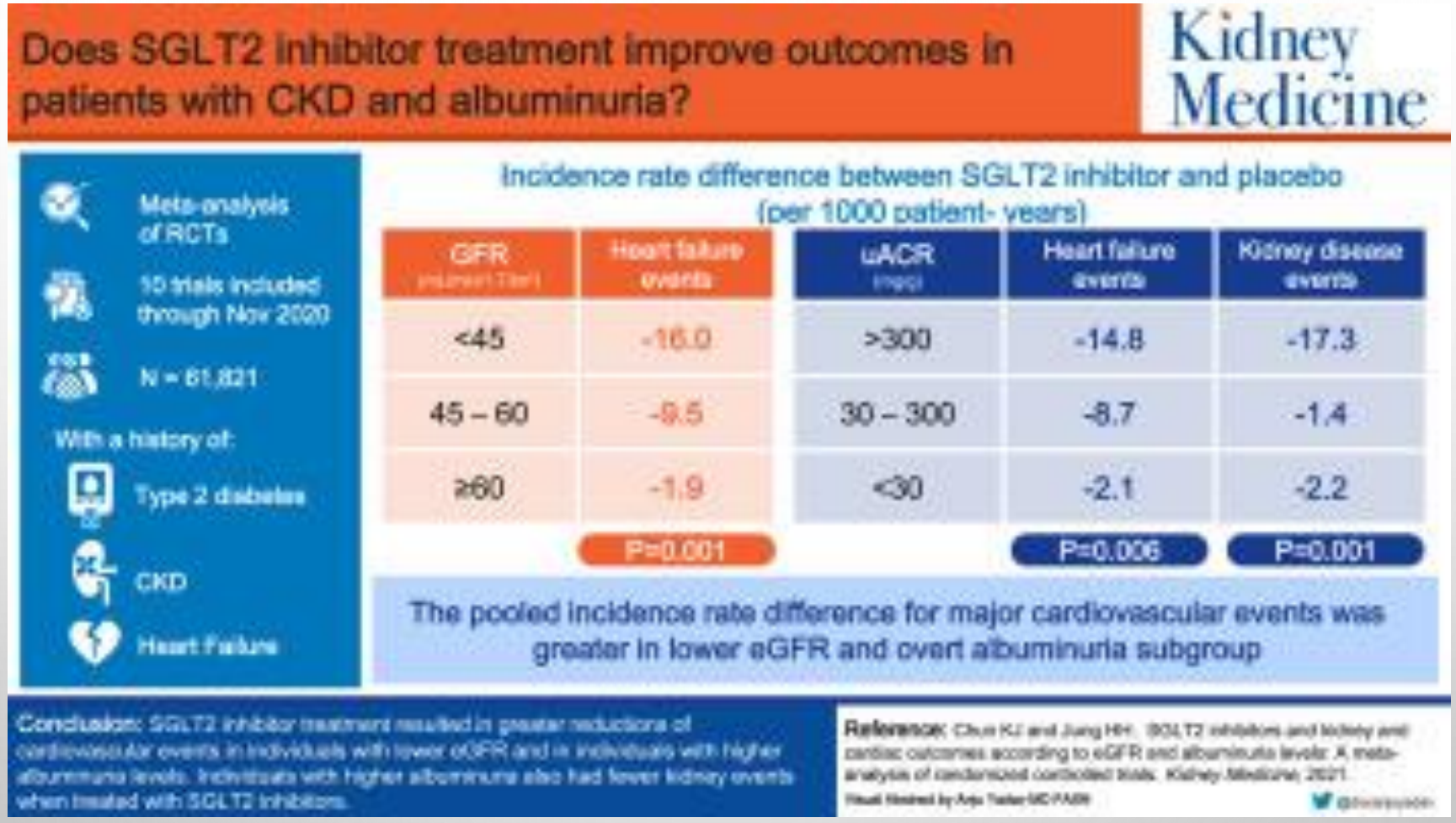
- Treatment
  - Modification in diet and exercise
  - Self monitoring of blood glucose
  - Medication
    - Metformin okay when eGFR >45
      - Caution when eGFR 30-44
      - Not use when eGFR <30
  - Decreasing renal function increases the risk of hypoglycemia from insulin therapy

NEWER THERAPIES IN CKD

- SGLT2I
- SELECTIVE MRA
- GLP-1

SGLT2 Inhibitors

Mechanism of action	• Inhibits SGLT2 (sodium/glucose cotransporter 2) in the proximal tubule, blocking reabsorption of filtered glucose (leading to osmotic diuresis)
Examples (_ gliflozin)	• Empagliflozin (Jardiance®) - Best risk/benefit ratio of the three • Dapagliflozin (Forxiga®) • Canagliflozin (Invokana®)
Major advantages	• Weight loss (~2-3kg) • Empagliflozin and canagliflozin ↓ CV mortality in high risk patients with T2D + atherosclerotic heart disease • All 3 ↓ heart failure hospitalizations and progression of nephropathy
Contraindications	eGFR < 30 mL/minute/1.73 m^2 (for first initiation of use)
Common side effects and important toxicities	• AKI (likely from hypovolemia) • GU infections (e.g. UTIs, vulvovaginal candidiasis) • Euglycemic diabetic ketoacidosis (DKA) • Canagliflozin ↑ risk of lower limb amputation and bone fractures



Chun K, Jung H

SGLT2 Inhibitors and Kidney and Cardiac Outcomes According to Estimated GFR and Albuminuria Levels: A Meta-analysis of Randomized Controlled Trials

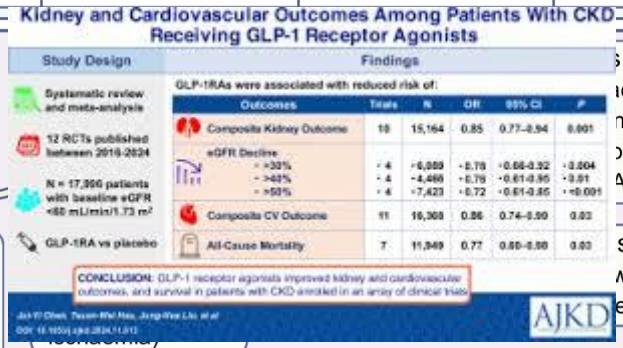
Kidney Medicine, 2021; 3, 732-744.e1

Distinct properties from steroidal MRA

	Steroidal MRA		Non-steroidal MRA
	Spironolactone	Eplerenone	Finerenone
MR selectivity	+	++	+++
MR potency	+++	+	+++
MR cofactor recruitment	Partial agonist	Partial agonist	Inverse agonist
Tissue distribution	>>	>>	==
$t_{1/2}$	>20 hours	4–6 hours	2–3 hours
Structure	Flat	Flat	Bulky

vs eplerenone:
- ↓Macrophage invasion
- ↓Cardiac fibrosis
- ↑Longitudinal strain

(Rat model of HF)



s eplerenone:
- ıc remodelling
- nuria
- oBNP
- A-challenged rats)

spironolactone:
- wn adipose tissue
- ese mice)

Key randomised clinical trials

	ARTS-HF Phase II | *n*=1066	ARTS-DN Phase II | *n*=823	FIDELIO-DKD Phase III | *n*=5734	FIGARO-DKD Phase III | *n*=7437
Comparison	Finerenone vs eplerenone	Finerenone vs placebo	Finerenone vs placebo	Finerenone vs placebo
Patients	HFrEF + CKD/T2DM	T2DM + CKD	T2DM + CKD	T2DM + CKD
Primary finding	Similar reduction in NT-proBNP	↓ UACR	↓ Kidney failure, eGFR reduction or renal death	↓ CV death, nonfatal MI, nonfatal stroke or HF hospitalisation

Abbreviations: CKD, chronic kidney disease; CV, cardiovascular; DOCA, deoxycorticosterone acetate; HF, heart failure; HFrEF, heart failure with reduced ejection fraction; MI, myocardial infarction; MR, mineralocorticoid receptor; MRA, mineralocorticoid receptor antagonist; NT-proBNP, N-terminal pro-B-type natriuretic peptide; T2DM, type 2 diabetes mellitus; UACR, urinary albumin/creatinine ratio

Popular GLP-1 Medications

Semaglutide

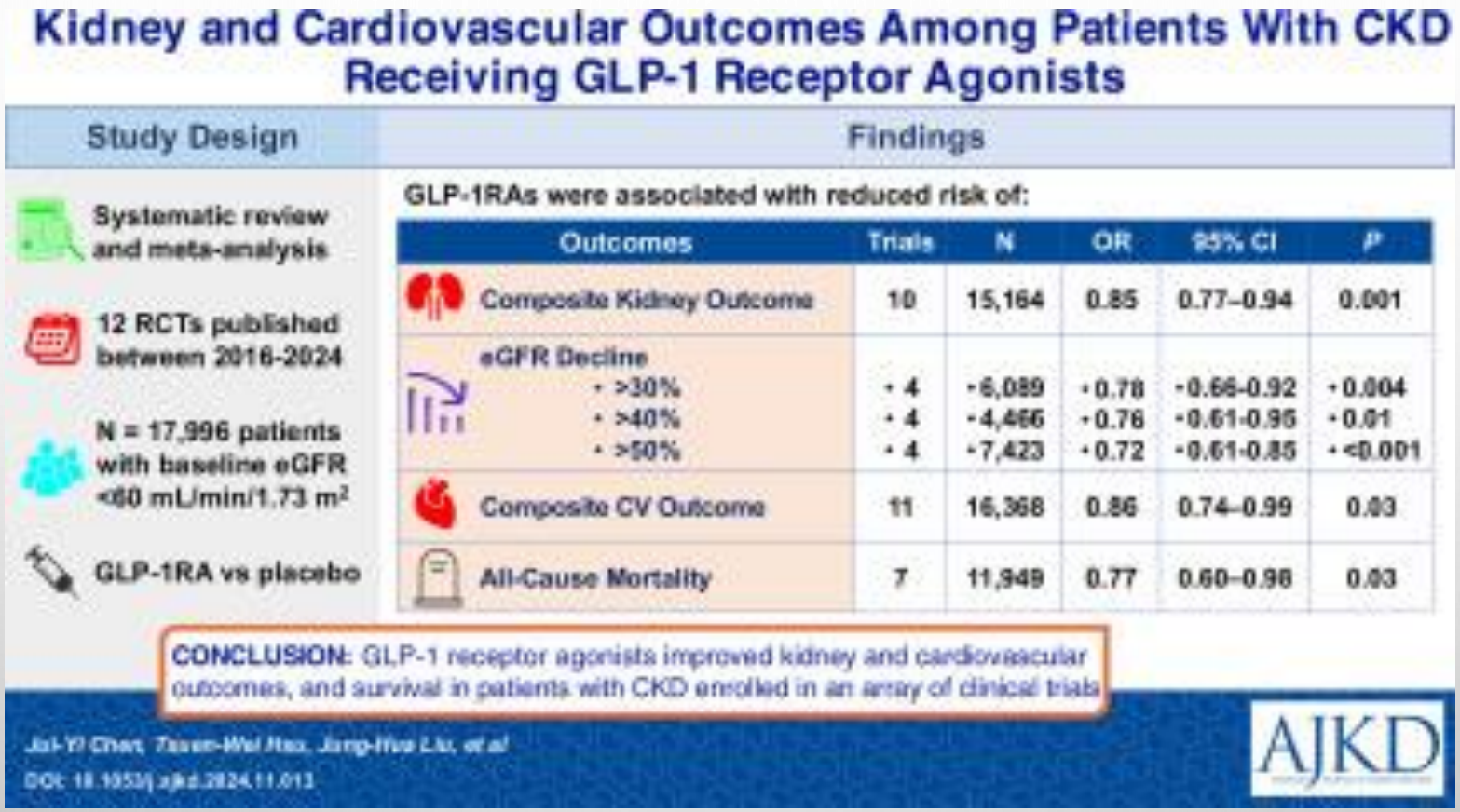
- **Ozempic:** Weekly injectable medication to treat type 2 diabetes
- **Rybelsus:** Daily oral tablet to treat type 2 diabetes
- **Wegovy:** Weekly injectable weight loss medication

Dulaglutide

- **Trulicity:** Weekly injectable medication to reduce the risk of cardiovascular events (heart attack, stroke, etc.) in type 2 diabetics

Tirzepatide

- **Mounjaro:** Weekly injectable medication to treat type 2 diabetes.
- **Zepbound:** Weekly injectable weight loss medication



Glucagon-like Peptide-1 Receptor Agonists: New Evidence of Kidney and Cardiovascular Protection From the FLOW and SELECT Trials

American Journal of Kidney Diseases, 2024; 85, 115-118

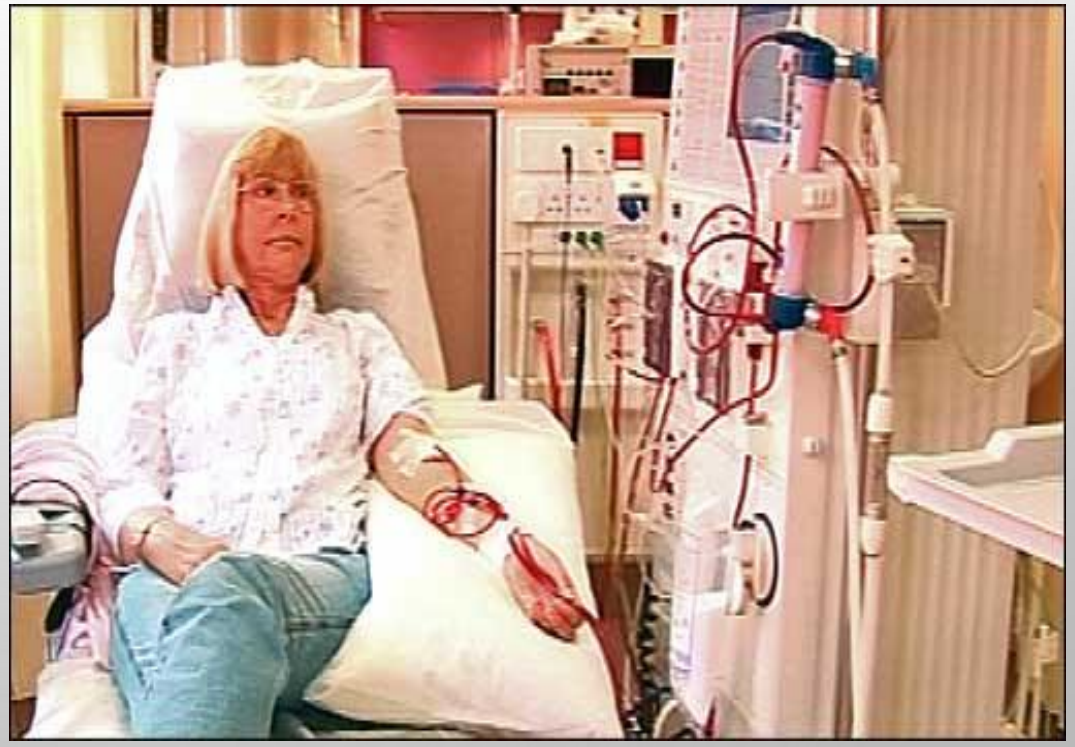
End Stage Renal Disease

- AS OF EARLY 2025, AROUND 516,837 PATIENTS WERE ON DIALYSIS (MOSTLY IN-CENTER HEMODIALYSIS) AND 316,873 PATIENTS WERE LIVING WITH A FUNCTIONING KIDNEY TRANSPLANT.
- 20% annual mortality
  - Cardiovascular complications major cause of death
- Diagnosis leading to ESRD
  - HTN, DM

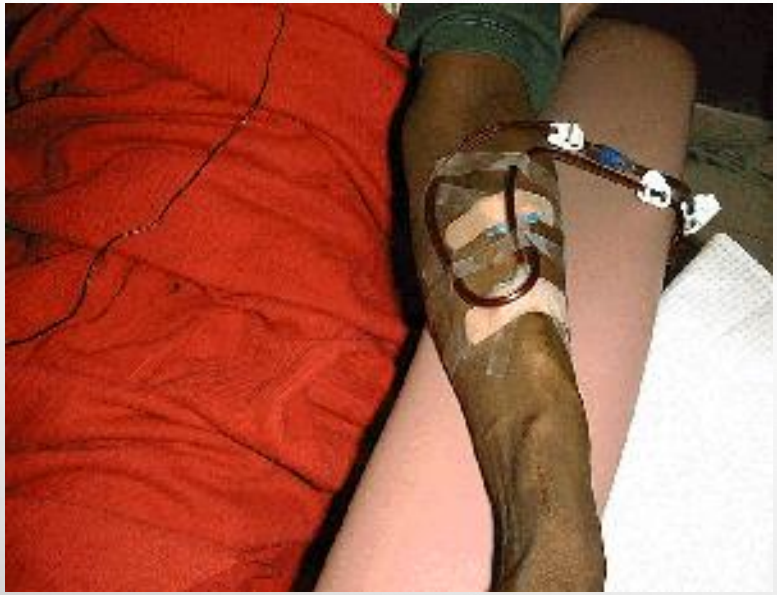
Cause	U.S. Prevalence in Incident ESRD Population (2007)	Diagnosis
Diabetes (Types 1 & 2)	44.0%	• Fasting glucose >126 mg/dl on 2 occasions • Symptoms of diabetes and random blood sugar >200 mg/dl • HgbA1C≥6.5% on 2 occasions
Hypertension	27.6%	• BP>140/90
Glomerulonephritis	7.3%	• RBC casts • Proteinuria • Renal biopsy
Cystic diseases of the kidney	2.4%	• Imaging studies • Ultrasonography • CT Scanning
Other causes	14.6%	• Urinalysis • Imaging studies • Renal biopsy

Hemodialysis

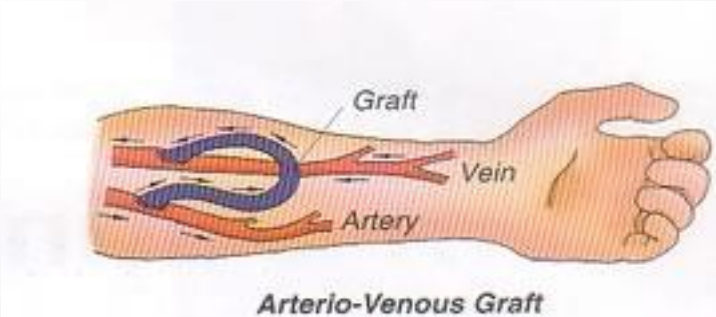
- Can be done at home or in center
- Need for vascular access

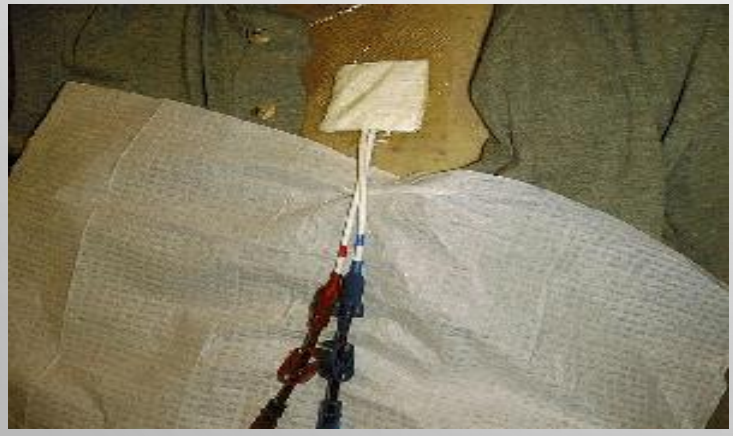


Vascular Accesses



AV Fistula



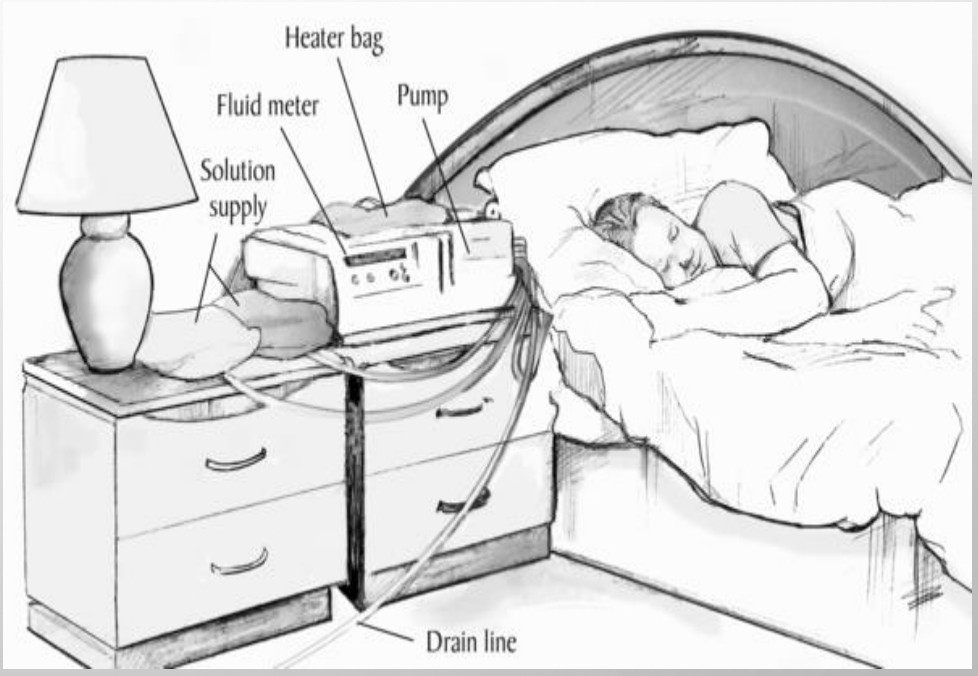


Double Lumen Catheter

HOME HEMODIALYSIS

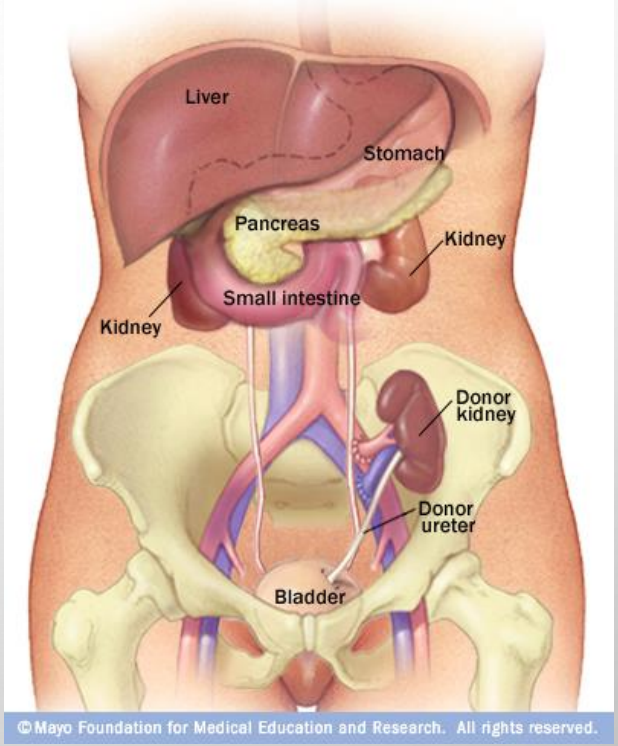
Peritoneal Dialysis

- Done at home
- No assistance needed



Transplant

- Living donor or deceased donor
- Life expectancy increased
- Lower cost ($29,000 annually after first year)



USRDS 2011 Report

CONSERVATIVE MANAGEMENT

- MANAGEMENT OF SYMPTOMS
- ADVANCE-CARE PLANNING
- PROVISION OF APPROPRIATE PALLIATIVE CARE

INDICATIONS FOR REFERRAL TO A NEPHROLOGIST

- Acute kidney injury or sudden sustained drop in GFR
- G4-G5 GFR category
- Persistent significant albuminuria or proteinuria (ACR ≥300 mg/g or PCR ≥500 mg/g)[1]
- CKD progression (≥25% drop in GFR from baseline or >5 ml/min/1.73 m^2 annual decline)
- Unexplained persistent hematuria (>20 RBCs/ hpf) or urinary red blood cell casts
- CKD and hypertension refractory to treatment with 4 or more antihypertensive medications
- Persistent electrolyte abnormalities
- Recurrent/ extensive nephrolithiasis
- Hereditary kidney disease

1: Albumin/creatinine ratio; protein/creatinine ratio

1 Don’t perform routine cancer screening for dialysis patients with limited life expectancies without signs or symptoms.

Due to high mortality among end-stage renal disease (ESRD) patients, routine cancer screening—including mammography, colonoscopy, prostate-specific antigen (PSA) and Pap smears—in dialysis patients with limited life expectancy, such as those who are not transplant candidates, is not cost effective and does not improve survival. False-positive tests can cause harm: unnecessary procedures, overtreatment, misdiagnosis and increased stress. An individualized approach to cancer screening incorporating patients’ cancer risk factors, expected survival and transplant status is required.

2 Don’t administer erythropoiesis-stimulating agents (ESAs) to chronic kidney disease (CKD) patients with hemoglobin levels greater than or equal to 10 g/dL without symptoms of anemia.

Administering ESAs to CKD patients with the goal of normalizing hemoglobin levels has no demonstrated survival or cardiovascular disease benefit, and may be harmful in comparison to a treatment regimen that delays ESA administration or sets relatively conservative targets (9–11 g/dL). ESAs should be prescribed to maintain hemoglobin at the lowest level that both minimizes transfusions and best meets individual patient needs.

3 Avoid nonsteroidal anti-inflammatory drugs (NSAIDS) in individuals with hypertension or heart failure or CKD of all causes, including diabetes.

The use of NSAIDS, including cyclo-oxygenase type 2 (COX-2) inhibitors, for the pharmacological treatment of musculoskeletal pain can elevate blood pressure, make antihypertensive drugs less effective, cause fluid retention and worsen kidney function in these individuals. Other agents such as acetaminophen, tramadol or short-term use of narcotic analgesics may be safer than and as effective as NSAIDs.

4 Don’t place peripherally inserted central catheters (PICC) in stage III–V CKD patients without consulting nephrology.

Venous preservation is critical for stage III–V CKD patients. Arteriovenous fistulas (AVF) are the best hemodialysis access, with fewer complications and lower patient mortality, versus grafts or catheters. Excessive venous puncture damages veins, destroying potential AVF sites. PICC lines and subclavian vein puncture can cause venous thrombosis and central vein stenosis. Early nephrology consultation increases AVF use at hemodialysis initiation and may avoid unnecessary PICC lines or central/peripheral vein puncture.

5 Don’t initiate chronic dialysis without ensuring a shared decision-making process between patients, their families, and their physicians.

The decision to initiate chronic dialysis should be part of an individualized, shared decision-making process between patients, their families, and their physicians. This process includes eliciting individual patient goals and preferences and providing information on prognosis and expected benefits and harms of dialysis within the context of these goals and preferences. Limited observational data suggest that survival may not differ substantially for older adults with a high burden of comorbidity who initiate chronic dialysis versus those managed conservatively.