Chronic Kidney Disease

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Overview

- Chronic kidney disease
  - Definition
  - Epidemiology
  - Risk factors
  - Pathophysiology
    - Most common primary diseases
    - Adaptive response by the kidney to the loss of nephrons
  - Stages of CKD
  - Treatment to slow progression
  - CKD impact on morbidity and mortality
  - Treatment to prevent and manage comorbidities
  - Preparation for renal replacement therapy and transplant
  - Cases

Overview of Kidney Function

- Homeostasis
  - Elimination of waste products
  - Adjusts water and electrolyte balance via tubular reabsorption or secretion
- Hormone secretion
  - Systemic and renal hemodynamic
    - Renin, prostaglandins, bradykinin
  - Red blood cell production
    - erythropoietin
  - Calcium, phosphorus, and bone metabolism
    - 1,25-dihydroxyvitamin D3 (calcitriol)

Definition of Chronic Kidney Disease

- The presence of markers of kidney damage for ≥3 months, as defined by structural or functional abnormalities of the kidney with or without decreased glomerular filtration rate (GFR), that can lead to decreased GFR, manifest by either pathological abnormalities or other markers of kidney damage, including abnormalities in the composition of blood or urine, or abnormalities in imaging tests.

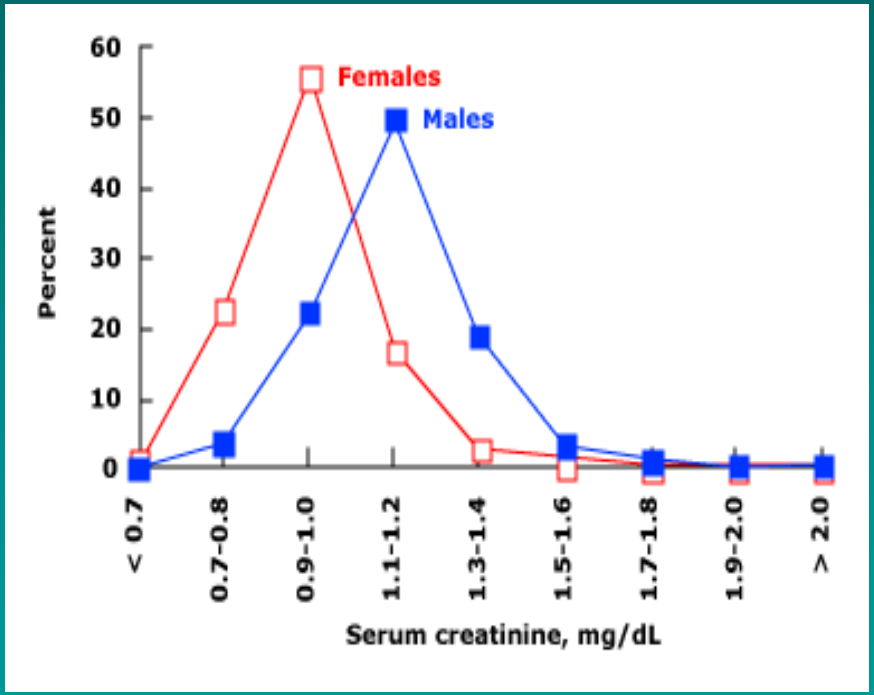
OR

- The presence of GFR <60 mL/min/1.73 m2 for ≥3 months, with or without other signs of kidney damage as described above.

Normal Serum Creatinine

Survey of individuals without hypertension or diabetes in the United States: the mean values for men 1.13mg/dL, women 0.93mg/dL

Ethnic group	Men	Women
Non-hispanic black	1.25	1.01
Non-hispanic white	1.16	0.97
Mexican-American	1.07	0.86



Jones, CA, et al. *Am J Kidney Dis* 1998 Dec;32(6):992-9.

Prevalence of Chronic Kidney Disease

- 14% of adults in the U.S. have chronic kidney disease (31million people)
- 44% of people over age 65 have chronic kidney disease

Progression to CKD

- Framingham Offspring Study
  - 1223 men and 1362 women initially free of preexisting kidney disease
  - 18.5 year mean follow-up
  - 9.4% developed CKD (GFR <64 men, <59 women)

Risk Factors for Susceptibility to Chronic Kidney Disease

Clinical Factors

- Diabetes
- Hypertension
- Autoimmune diseases
- Systemic infections
- Urinary tract infections
- Urinary stones
- Neoplasia
- Family Hx of CKD
- Recovery from ARF
- Reduction in kidney mass
- Exposure to nephrotoxic drugs
- Low birth weight

Sociodemographic factors

- US ethnic minority status:
  - African American
  - American Indian
  - Hispanic
  - Asian
  - Pacific Islander
- Exposure to certain chemical and environmental conditions
- Low income/education

Most Common Diseases

- Secondary
  - Diabetic nephropathy
  - Hypertensive nephrosclerosis
- Primary
  - IgA nephropathy
  - Focal Segmental Glomerulosclerosis (FSGS)
  - Membranous Nephropathy

Goals of Care

- Identify cause
- Provide treatment to correct the cause or slow progression
- Address cardiovascular risk factors
- Address metabolic abnormalities
- Education

CKD: Mechanisms of Progression

- Primary Disease Progression
- Secondary Adaptations by the kidney to the loss of nephrons
  - Hemodynamic changes
  - Structural adaptations

Slowing Primary Disease Progression

- Glycemic control
- Blood pressure control
- Immunosuppressants for secondary immune-modulated diseases

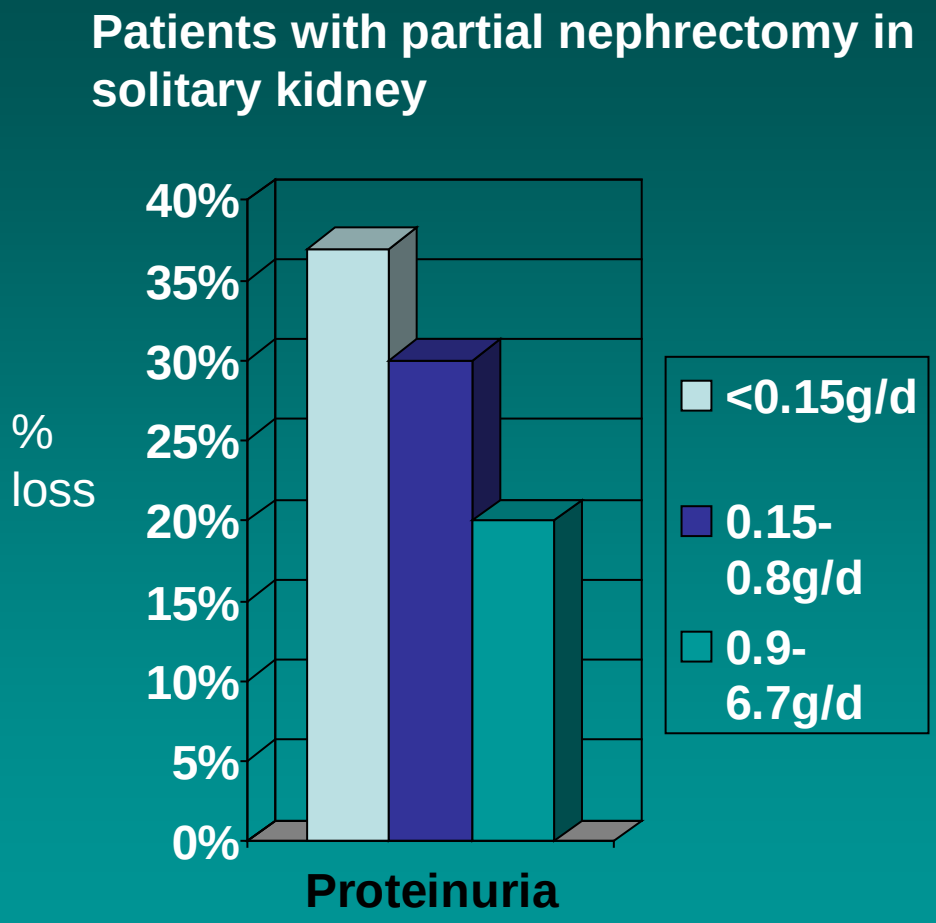
Treating Damage from Secondary Adaptations

Remnant Kidney Rat Model

- 1 kidney is removed, and 5/6 of the remaining kidney is removed
- Rats progress to ESRD in 4-6 months
- Pathology is diffuse focal and segmental glomerulosclerosis

Human Response to Nephron Loss

- Human response to nephron loss is similar to the animal model
- Degree of secondary injury depends on amount of renal mass remaining



Mechanism: hemodynamic changes

Afferent > efferent arteriolar vasodilation

↓

Decreased arteriolar resistance

↓

Increased glomerular pressure (more systemic pressure is transmitted to the glomerulus)

↓

Increased renal blood flow

Mechanism: structural changes

Glomerular hypertrophy (to increase filtration surface area) without hyperplasia

↓

Decreased density of cells: cells cannot maintain intact podocytes and slit diaphragms

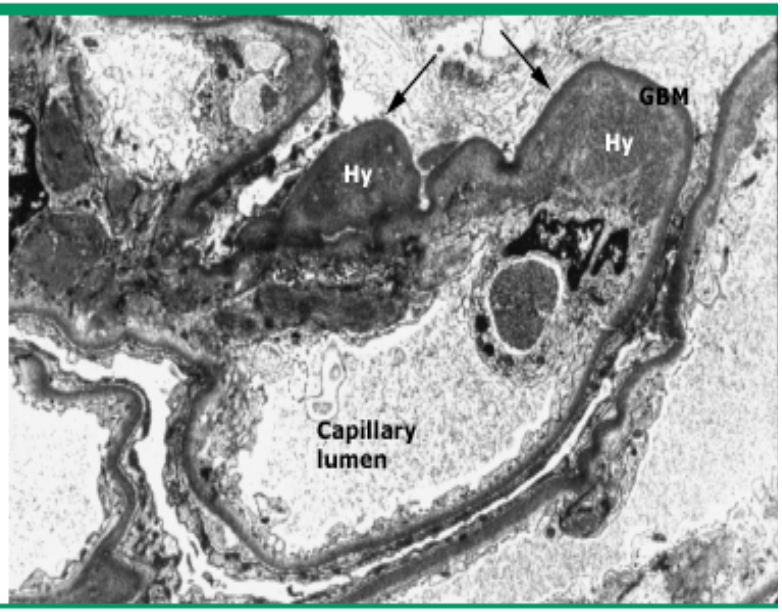
↓

Simplification and fusion of podocytes, denudation of epithelial cells, loss of slit diaphragms

↓

Proteinuria/Focal and segmental glomerulosclerosis

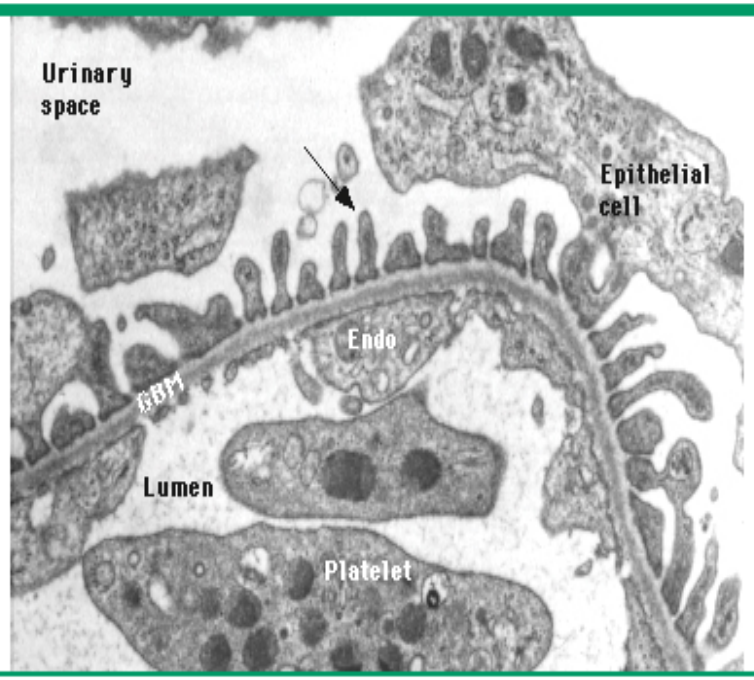
Focal glomerulosclerosis



Electron micrograph in focal segmental glomerulosclerosis shows diffuse epithelial cell foot process fusion with occasional loss of the epithelial cells (arrows). The other major finding is massive subendothelial hyaline deposits (H under the glomerular basement membrane (GBM). These deposits reflect insudation of plasma proteins, not the deposition of immunoglobulins. These deposits contribute narrowing of the capillary lumens.

Courtesy of Helmut Rennke, MD.

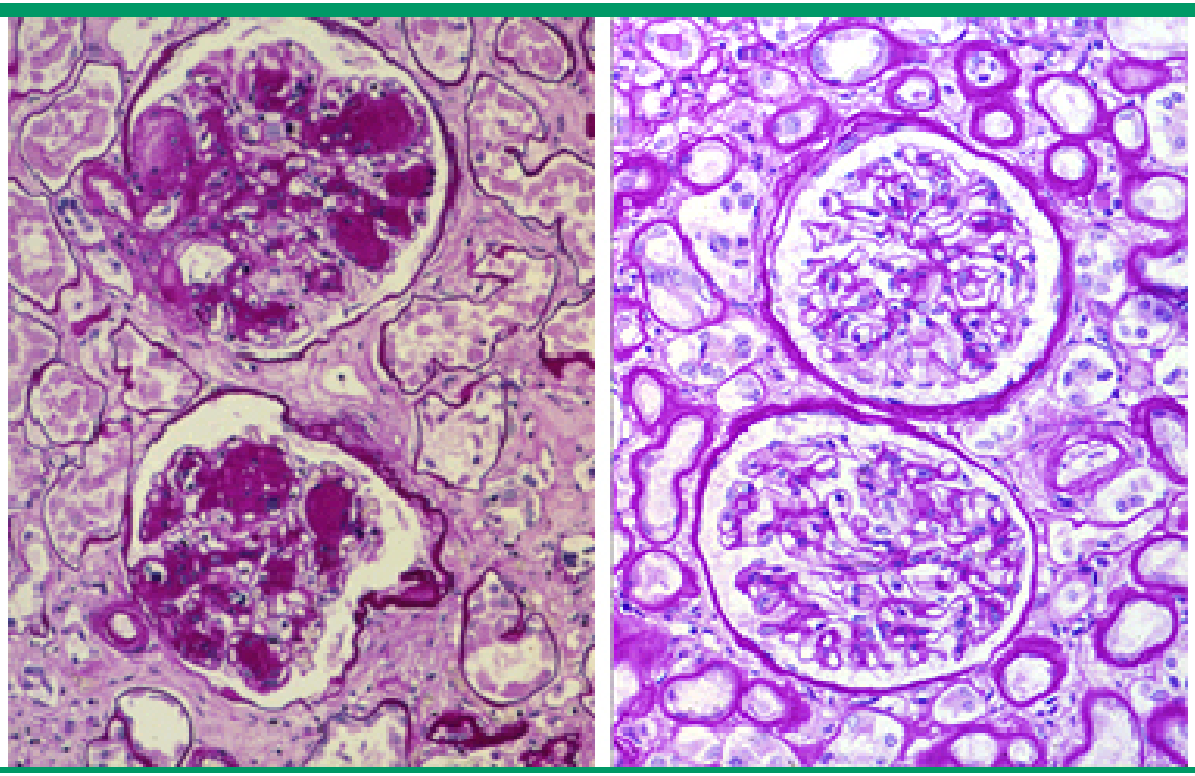
Normal glomerulus



Electron micrograph of a normal glomerular capillary loop showing the fenestrated endothelial cell (Endo), the glomerular basement membrane (GBM), and the epithelial cells with its interdigitating foot processes (arrow). The GBM is thin and no electron dense deposits are present. Two normal platelets are seen in the capillary lumen.

Courtesy of Helmut Rennke, MD.

Unilateral diabetic glomerulosclerosis



Light micrographs from a postmortem examination of a diabetic patient with unilateral renal artery stenosis on the right side. Classic Kimmelstiel-Wilson nodules are seen in the glomeruli in the left kidney (left panel); in contrast, the glomeruli are normal in the "protected" right kidney (right panel).

Courtesy of Helmut Rennke, MD.

Stages of CKD (KDOQI)

Stage	GFR	Intervention
1	>90 with kidney damage	Diagnosis and treatment, treat comorbid conditions, slow progression, CVD risk reduction.
2	60-89 with kidney damage	Above + Prevent progression
3	31-60	Prevent progression and manage morbidities assoc with ckd (renal osteodystrophy, anemia, etc.)
4	16-30	Modality and transplant planning
5	0-15	Renal replacement therapy

Stages of CKD (KDIGO)

GFR Stage	GFR	Intervention
G1	>90 with kidney damage	Diagnosis and treatment, treat comorbid conditions, slow progression, CVD risk reduction.
G2	60-89 with kidney damage	Above + Prevent progression
G3a	45-59	Prevent progression and manage morbidities assoc with ckd (renal osteodystrophy, anemia, etc.) ? Nephrology referral – for evaluation and plan
G3b	30-44	Prevent progression and manage morbidities assoc with ckd (renal osteodystrophy, anemia, etc.) ? Nephrology referral – for evaluation and plan
G4	15-29	Nephrology Comanagement: Modality and transplant planning
G5	<15	Renal replacement therapy

Stages of CKD (KDIGO)

AER Stage	AER
A1	<30mg/g creatinine
A2	30-300mg/g creatinine
A3	>300mg/g creatinine

Referral to Nephrologist

- Decreased mortality associated with early referral (CKD 3) (but these patients are less likely to progress to ESRD or develop cardiovascular disease)
- Increased mortality if referred <4 months of starting dialysis

- Recommendation:
  - CKD3 for initial evaluation and treatment plan
  - CKD4 for active management
  - Glomerular disease

Preventing Progression: HTN CONTROL

HTN from Renal Disease: epidemiology

- Most common cause of secondary HTN
- Hypertension eventually occurs in 85-90% of CKD patients

HTN from Renal Disease: mechanism

- 80% - volume expansion is primarily responsible for HTN
- ≅ 20% - increased renin release is important

HTN from Renal Disease: mechanism of renin-mediated HTN

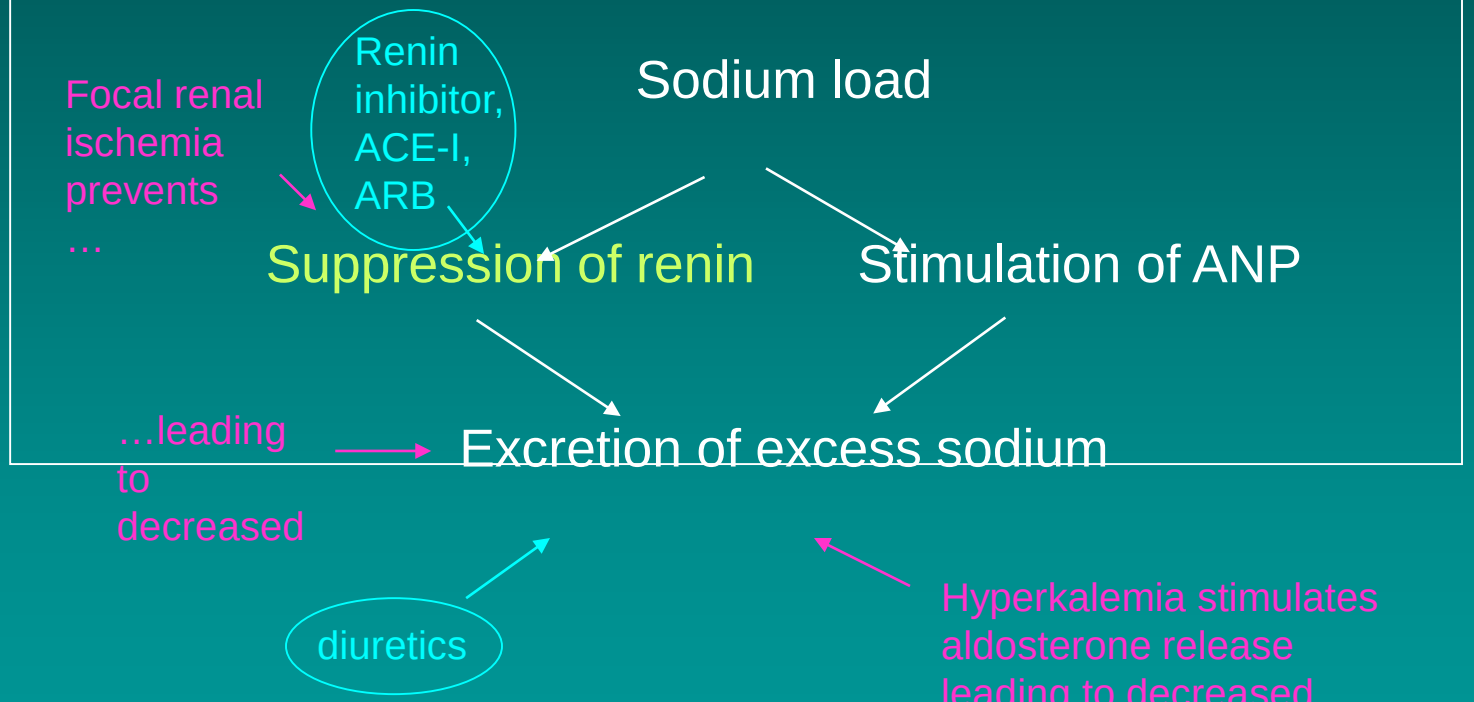
- Primary vascular disease from vasculitis, hypertensive nephrosclerosis, or atherosclerotic renal artery stenosis
- Disordered renal architecture causing focal ischemia leading to increased renin release

Renal Disease: HTN is necessary for maintenance of homeostasis

- Decreased number of nephrons requires that each nephron secrete more sodium
- HTN leads to increased renal perfusion pressure which increases sodium excretion
- Thus, HTN is the “price paid” for preventing sodium accumulation

HTN from Renal Disease: Treatment

Role of Pressure Natriuresis in renal disease:

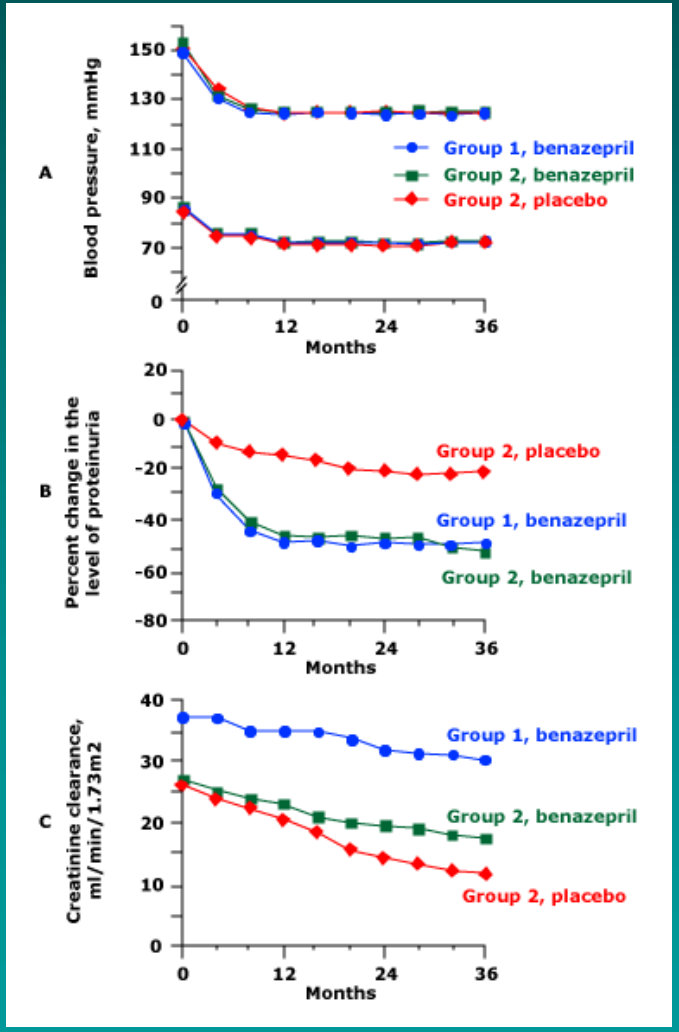


Preventing Progression: HTN control

- ACE-I/ARB
  - ACE-I/ARB is most protective, if proteinuria is present
  - Can continue if the GFR decline over four months is less than 30 percent from baseline value and serum potassium is 5.5 mEq per L
  - Combination ACE-I + ARB (controversial)
    - Can reduce proteinuria more than monotherapy
    - Higher rate of adverse effects: hyperkalemia, worsening renal function
    - Thus – combined use is not recommended

Creatinine clearance over time in mild and advanced CKD

- Group 1 – creatinine 1.5-3.0 all got benazepril
- Group 2 – creatinine 3.1-3.5 got either benazepril or placebo
- Even advanced ckd patients benefit from ACE-I



Hou FF, et al. *N Engl J Med* 2006; 354:131.

Controversy of ACE-I/ARB in Advanced CKD

- Stopping ACE-I or ARB in advanced CKD (GFR 16) resulted in improved GFR and delayed onset or renal replacement therapy

- Treating ESRD patient with ACE-I/ARB resulted in prolonged residual renal function

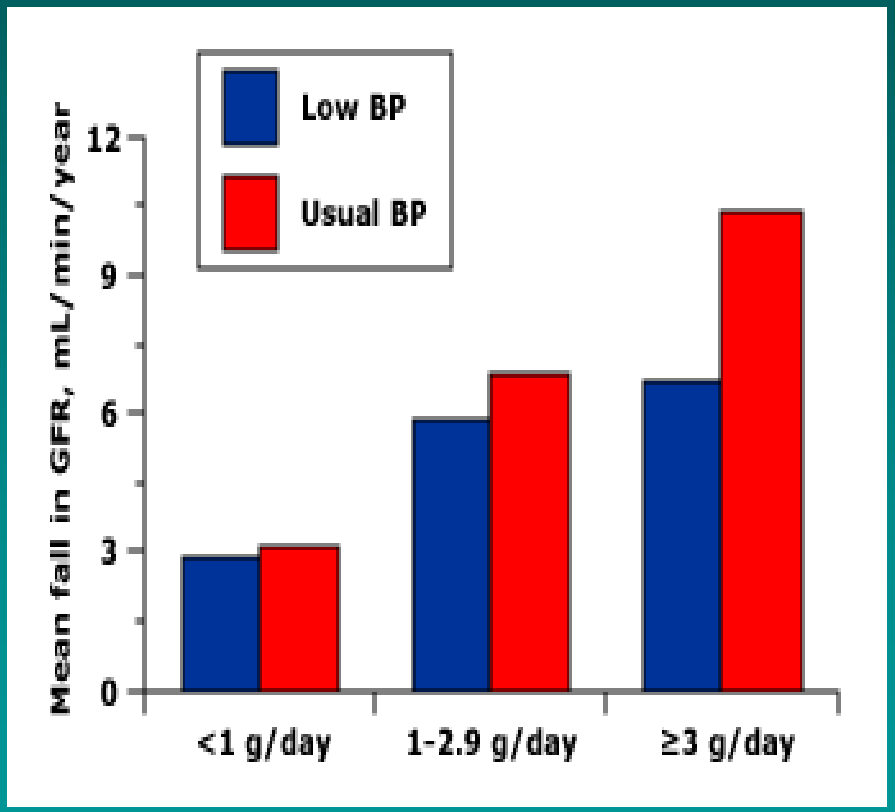
Preventing Progression: HTN control

- 2009 Cochrane review of patients with CKD found no reduction in cardiovascular events, stroke, end-stage renal disease, or total mortality in those with lower (135/85 mm Hg or less) versus standard (140 to 160/90 to 100 mm Hg or less) blood pressure goals
- Review of nondiabetic patients with CKD suggested that lower targets might benefit patients with proteinuria greater than 300 mg per day.
- Optimal BP 110-129 to prevent CKD progression
  - RR 1.83 SBP 130-139
  - RR 3.14 SBP >160

Arguedas JA et al. *Cochrane Database Syst Rev*. 2009;(3)
.Upadhyay A,et al. *Ann Intern Med*. 2011;154(8):541-548
Jafar TH et al. *Ann Int Med*. 2003; 139:244-52.

Aggressive BP control in proteinuric patients

- Usual BP control
  - <130/80
- Aggressive BP control
  - 4.7mmHg lower than usual BP
- Aggressive BP control only benefited pts. with >1g/day proteinuria



Klahr S, et al. *N Engl J Med* 1994;330:877.

Preventing Progression: HTN control

KDIGO guidelines 2013:

- CKD without proteinuria <140/90
- CKD with proteinuria (>500mg/day) <130/80
- RAS blockade in all patients with albuminuria regardless of CKD
- If edema: start both loop diuretic and ACE-I/ARB
- If no edema: RAS blockade, then either non-dihydropyridine CCB or loop diuretic

Most guidelines recommend relaxed blood pressure control <150/90 in older patients (>70) regardless of CKD or proteinuria

Preventing Progression: Proteinuria remission

- Reduction of proteinuria
  - Reduce protein excretion to <500mg/day, or at least a reduction of 60% baseline values

- ACE-I/ARB
- Nondihydropyridine calcium channel blockers
  - Diltiazem, verapamil

Preventing Progression: glycemic control

- K/DOQI guidelines recommend Hgba1c <7 in diabetic patients
- A few RCT's have shown that A1C<7 preserves GFR except in those with proteinuria Several RCTs in high risk patients have not shown benefit of A1C<7, and showed higher rate of complications (severe hypoglycemia and death) with more aggressive control
- Thus – individualized goals should be pursued

Preventing Progression: metabolic acidosis

- Each surviving nephron must make more ammonia to handle daily acid load
  - Activates alternative complement pathway which increases renal inflammation
- Bicarbonate level <23 associated with higher risk of worsening function
- Correcting bicarbonate to >22 led to 80% lower rate of progression to ESRD
  - No increase in CHF, edema or BP

DeBrito et al. JASN. 2009; 20:2075-84

Preventing Progression: treatment of dyslipidemia

- no prospective evidence that treating dyslipidemia prevents the progression of CKD or diabetic nephropathy

- BUT, it lowers cardiovascular risk in thise patients

Preventing Progression: Contrast-Induced Nephropathy

- Serum creatinine increase > 25% from baseline or an absolute increase > 0.5 mg/dL within the first few days after receipt of intravenous contrast.
- Risk factors
  - CHF (class III/IV, or hx of pulm edema)
  - Hypotension (<80 for 1 hour or need for inotropes)
  - IABP
  - Age >75
  - DM
  - Contrast volume >100ml
  - Creatinine >1.5, or GFR <60

Mehran R, et al. *J Am Coll Cardiol*. 2004;44(7):1393-1399

Preventing Progression: Contrast-Induced Nephropathy

- IV fluids with isotonic saline or bicarbonate
  - Conflicting data on which is better
- Minimizing contrast volume and using low or iso-osmolal nonionic contrast agents
- NAC
  - Meta-analysis suggests that high dose NAC may be beneficial
- Statins
  - Meta-analysis of 3 RCT – no significant benefit; 7 non-randomized studies – marginally significant benefit (OR=0.60)
  - RCT statin-naïve individuals with NSTEMI, rosuvastatin vs placebo. CIN 6.7% rosuvastatin vs 15.5% controls

Pappy R, et al. International Journal of Cardiology 151 (2011) 348–353
Leoncini M, et al. J Am Coll Cardiol 2014;63:71–9

Preventing Comorbidities

CKD impact on Morbidity

- Retrospective study – 259pts with CKD (females creatinine >1.5, males >2.0)
  - 87% HTN
  - 35% DM
  - 40% CV disease
  - 14% peripheral vascular disease
  - 47% were hospitalized over the following year

Modification of Diet in Renal Disease Study

CKD impact on Morbidity & Mortality

- Rates of hospitalization 3 times higher than in the general population
  - Risk of hospitalization increases as GFR declines.
- CKD 3 patients are 20 times more likely to die of a cardiovascular event than to reach ESRD
- 2/3 of CKD patients have metabolic syndrome

CKD impact on Cardiovascular Risk

- Degree of renal dysfunction

  5 year death rates by stage:

  2 - 19.5%

  3 – 24.3%

  4 – 45.7%
- Hyperphosphatemia
- Proteinuria

Managing Comorbidities: HTN & DM

- HTN:
  - <130/80 proteinuria and diabetes
  - <140/90 no proteinuria
  - <150/90 age >70
- Glycemic control: large RCTs have not shown reduction in cardiovascular events or death from renal disease with aggressive glycemic control (A1C <7); risks outweigh benefits

Managing Comorbidities: Hyperlipidemia

- 2009 Cochrane meta-analysis – statins decreased all-cause and cardiovascular mortality in CKD patients
- Large RCT in mod/adv CKD showed simvastatin/ezetimibe reduced major CV events by 17%

Managing Comorbidities: Hyperlipidemia

- Dialysis patients: 2 large RCTs showed no significant benefit to statin therapy in lowering cardiovascular events in dialysis patients
- RCT of simvastatin and zetia vs placebo in dialysis and non-dialysis patients showed 17% decrease in major atherosclerotic events
- Meta-analysis of all 3 trials could be helpful to clarify the issue

- Goal LDL should be <100

4D study, AURORA study, SHARP study

Managing Comorbidities: Renal bone mineral disorder

- Changing trends over past 20 years: previously predominantly high bone turnover disease, now 40-70% low turnover

Renal bone mineral disorder: causes

- Decreased 1,25 dihydroxy vitamin D production
- Increased Fibroblast Growth Factor 23
  - Produced by osteoblasts and osteocytes
  - Increases renal phosphate excretion
  - Decreases 1,25 dihydroxy vitamin D levels
  - FGF-23 levels rise earlier than PTH levels

Treating Hyperphosphatemia

- Phos >3.5-4 is associated with increased mortality
- Goal phosphorus normal (2.5-4.5mg/dL)
- Role of assessing total phosphorus burden (FGF-23)
- Low phosphorus diet
- Phosphate binders
  - Sevelamer
  - Lanthanum
  - Velphoro (sucroferric oxyhydroxide)
  - Calcium acetate
- Restrict calcium-based binder if hypercalcemic, presence of arterial calcifications, or adynamic bone disease

Treating Secondary Hyperparathyroidism

- Replace 25 hydroxy vitamin D
- Control phosphorus
- Then, if PTH still elevated,
  - Nephrology referral
  - 1,25 dihydroxy vitamin D replacement
    - Calcitriol
    - Paricalcitol
    - Doxercalciferol
  - Calcium mimetics
    - cinacalcet

Treating Secondary Hyperparathyroidism: PTH goals

- K/DOQI guidelines (2003)
  - Stage 3 35-70
  - Stage 4 70-110
  - Stage 5/ESRD 150-300
- KDIGO guidelines (2009)
  - Stage 3-5: correct hyperphosphatemia, hypocalcemia and vitamin D deficiency first; add calcitriol if PTH above upper limit normal
  - ESRD: 2-9 x upper limit normal

Managing Comorbidities: Anemia

- Cochrane meta-analysis of 22 trials: either no difference or higher mortality with hgb >13.3 vs <12 target groups
- 2 RCTs showed higher hemoglobin target groups had higher rates of death, adverse cardiovascular events, and dialysis
- RCT with diabetic CKD patients: darbopoetin had higher stroke risk, no improvement in other CV risk
- FDA recommends not initiating ESA agents until Hgb <9

Singh AK et al.; CHOIR Investigators. *N Engl J Med*. 2006;355(20):2085-2098.
Drüeke TB et al.; CREATE Investigators. *N Engl J Med*. 2006;355(20):2071-2084.
Pfeffer MA, et al.; TREAT Investigators. *N Engl J Med*. 2009;361(21):2019-2032

Treating Anemia

- Erythropoetin replacement
- Iron replacement

Treating Anemia: goals

- CKD:
  - assess for correctable causes of anemia
  - do not initiate if Hgb >10
- ESRD:
  - avoid Hgb falling below 9.0
  - start ESA when hgb is between 9-10
- All patients: not recommended to maintain hgb >11.5, and especially not >13

What if kidney disease continues to progress?

- Dialysis
  - Hemodialysis
  - Peritoneal dialysis
- Transplant

Clinical Manifestations of CKD

- Most people have no symptoms until kidney function is very low
  - Problem is discovered on blood or urine tests done for other reasons
- Even in advanced kidney failure, many people still make a normal amount of urine

Clinical Manifestations of CKD

- In advanced kidney failure, people may develop symptoms of uremia
  - Nausea or vomiting
  - Taste disturbances
  - Poor appetite
  - Confusion, drowsiness
  - Fluid buildup and inflammation around the heart (pericarditis)
- Fluid retention
  - Leg swelling
  - Shortness of breath

Clinical Manifestations of CKD

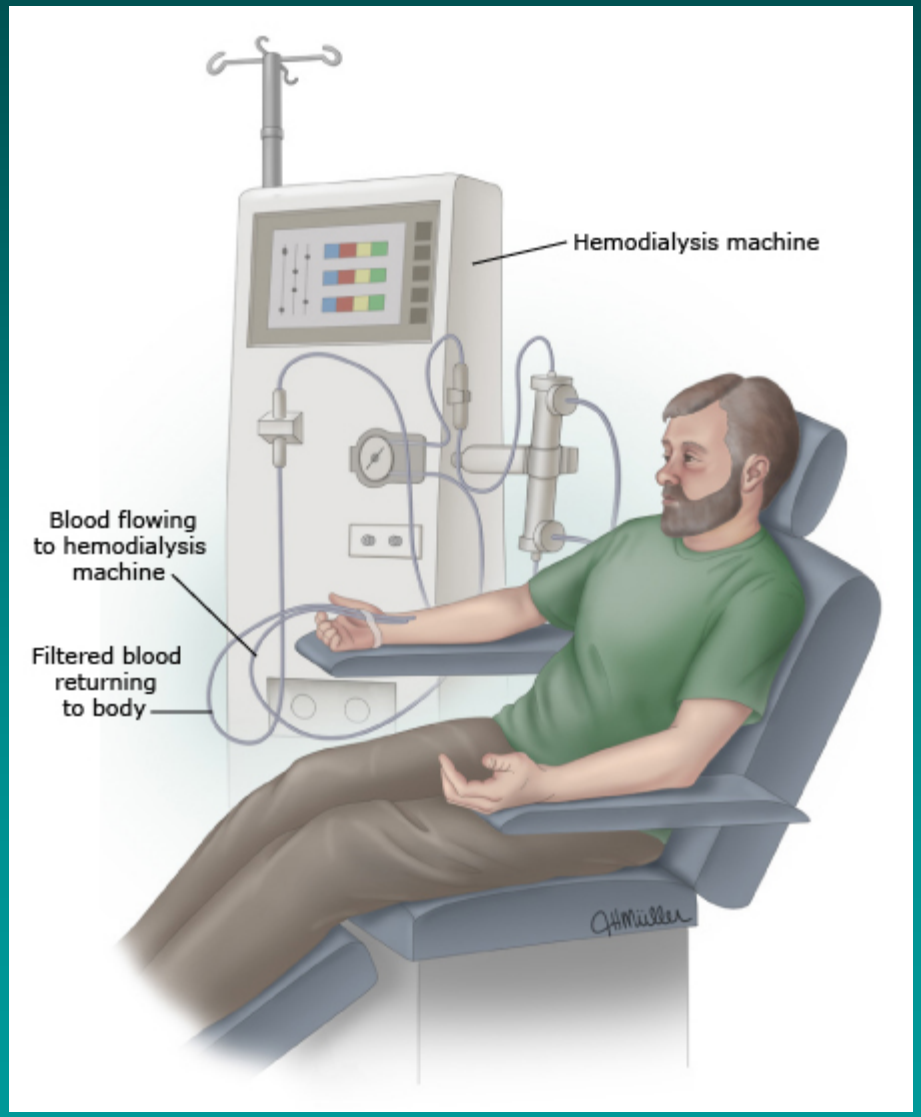
- In less-severe kidney disease patients may not have symptoms, but can have other complications
  - High blood pressure
  - Anemia
  - Bone disease from vitamin D deficiency and secondary hyperparathyroidism
  - Blood chemistry abnormalities (high potassium, high phosphorus)
  - Cardiovascular disease (from high phosphorus, secondary hyperparathyroidism, hypertension)

When to Start Dialysis

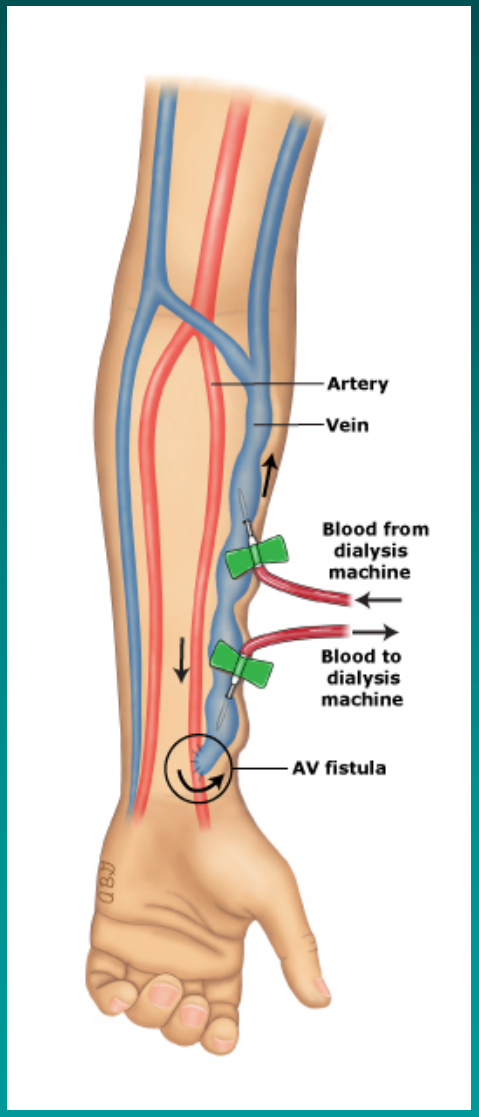
- No benefit to starting dialysis at GFR 10-14 vs 5-7ml/min
- Decision should be made based on quality of life and uremic symptoms

Hemodialysis

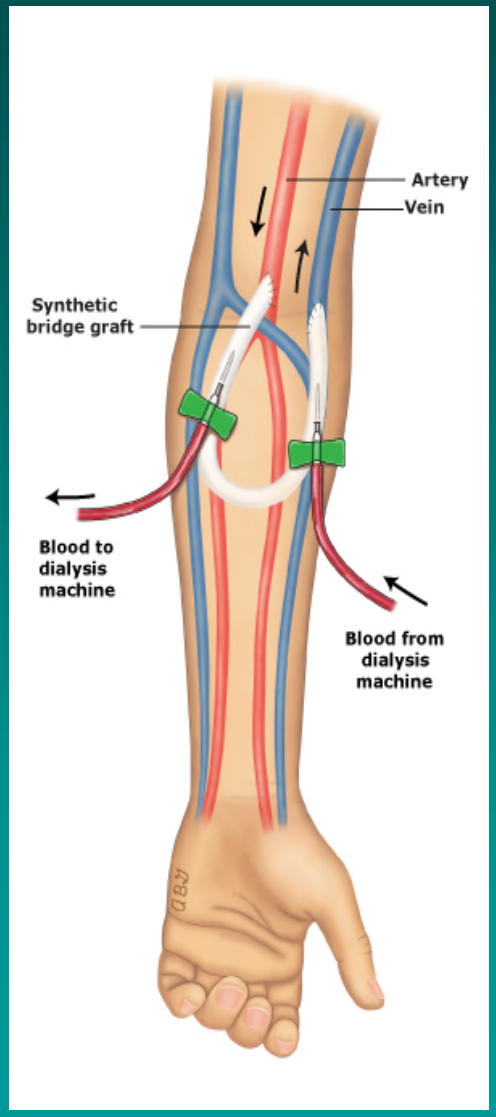
- 2 needles are placed in an “access” in the arm
- Blood flows to the dialysis machine, is filtered and is returned to the body
- 3-5hours, 3-7 times per week



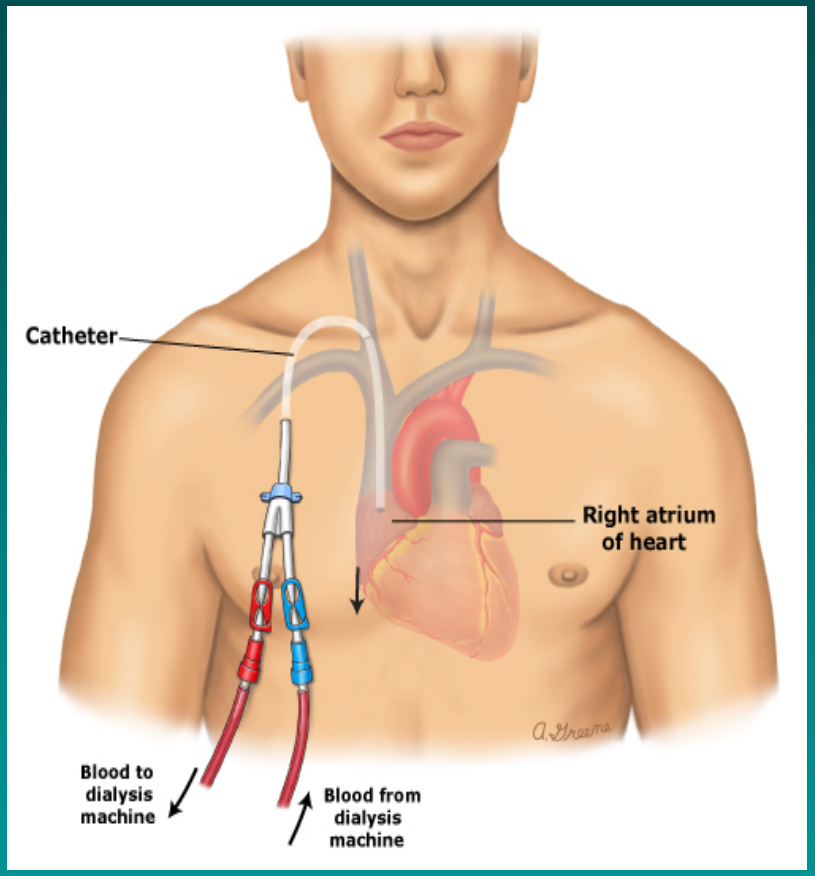
AV fistula



AV graft

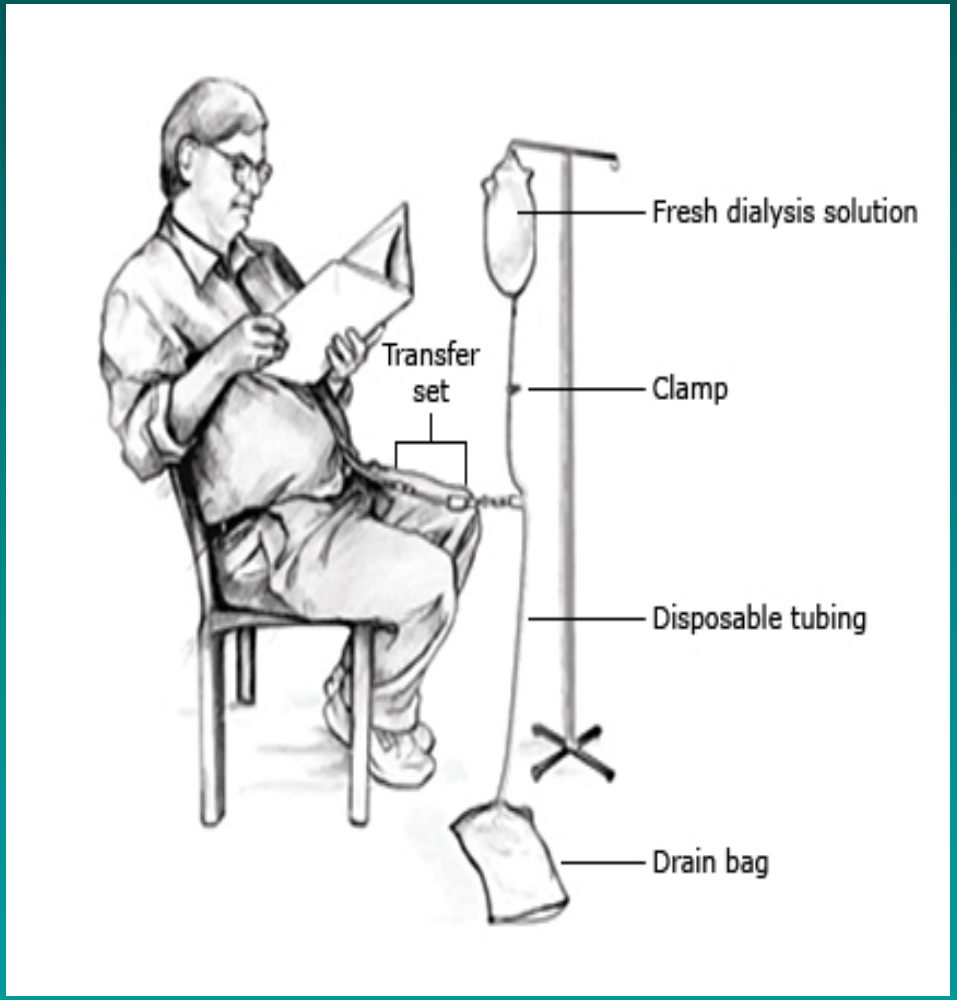


Central Venous Catheter



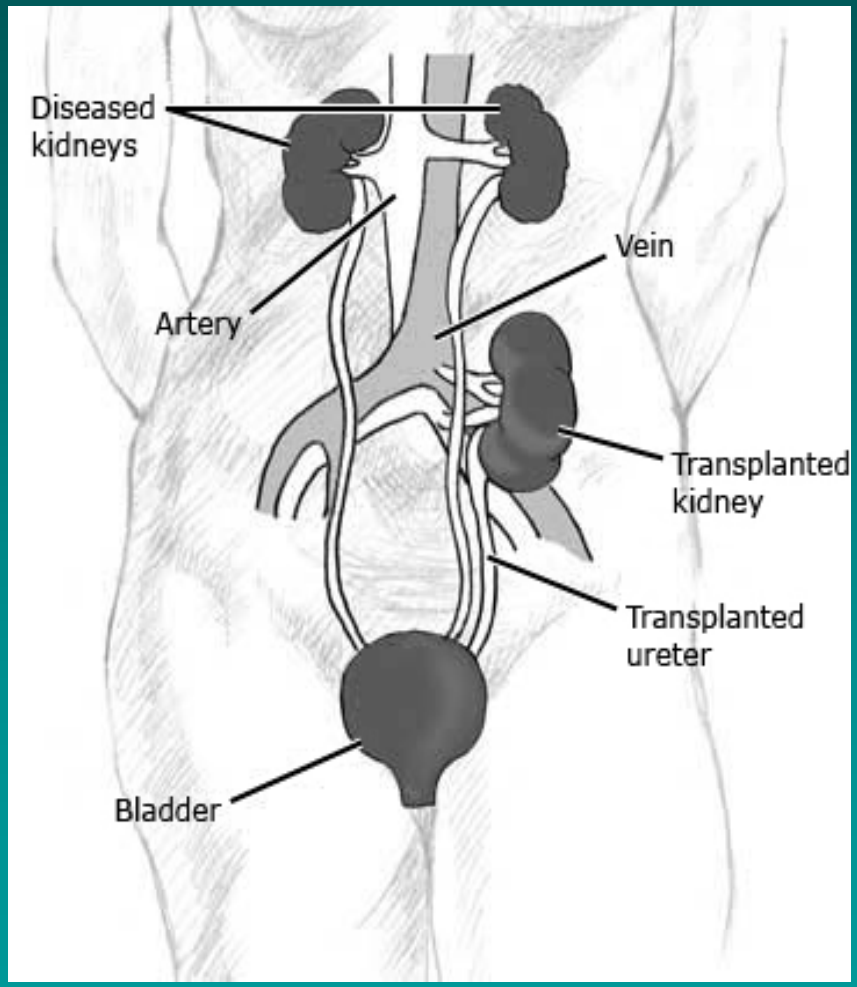
Peritoneal Dialysis

- Fresh dialysis solution flows into the person's belly
- It stays there for a period of time and then is drained into a drain bag
- A transfer set connects the catheter in the person's belly to the dialysis equipment



Transplant

- New kidney is put in the lower part of the belly
- Ureter is hooked up to the bladder
- Usually, the old kidneys are left in place

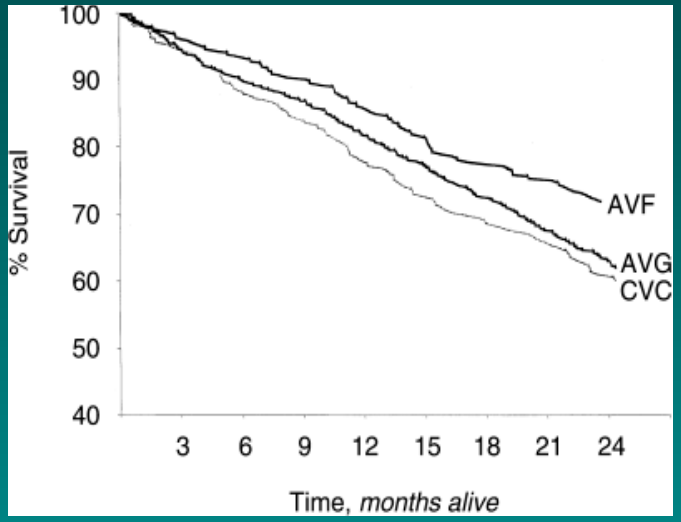


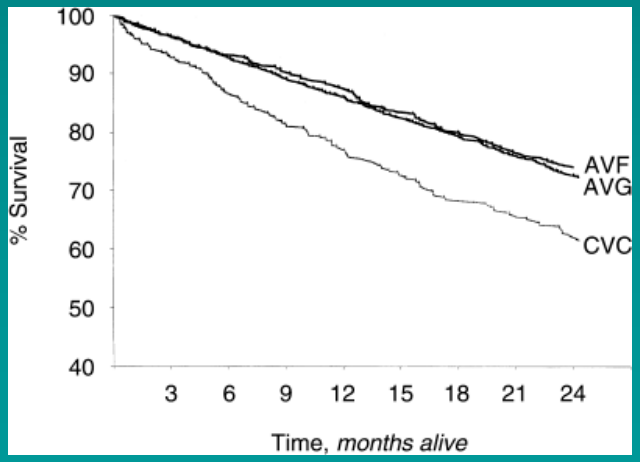
The benefits and downsides of kidney transplant, peritoneal dialysis, and hemodialysis

	Benefits	Downsides
Kidney transplant	• People who get a kidney transplant usually live longer and have a better quality of life than people who are treated with dialysis	• A kidney transplant is major surgery • It can be hard to find a donor kidney • People with certain medical conditions can't have a kidney transplant • After surgery, you need to take medicines every day for the rest of your life. These medicines could cause you to get severe infections or cancer. • After surgery, your body might "reject" your new kidney
Peritoneal dialysis	• You can do it at home, which gives you more freedom and control over your life • It doesn't involve needles • You can do it overnight while you sleep • You don't need anyone to help you if you learn how to do it yourself • You don't need to limit your diet as much as with hemodialysis	• It can be hard to learn how to do it • It increases the chance that you will get an infection in your belly
Hemodialysis	• You usually have it in a dialysis center, where doctors and nurses can watch you closely • You don't have to learn to do it yourself • You do it fewer days each week than peritoneal dialysis	• You need to travel to the center and back three times (or more) every week • You will have needles put in your arm each time • Your access can get infected or stop working • During dialysis, you might feel lightheaded or have trouble breathing

Mortality & Vascular Access

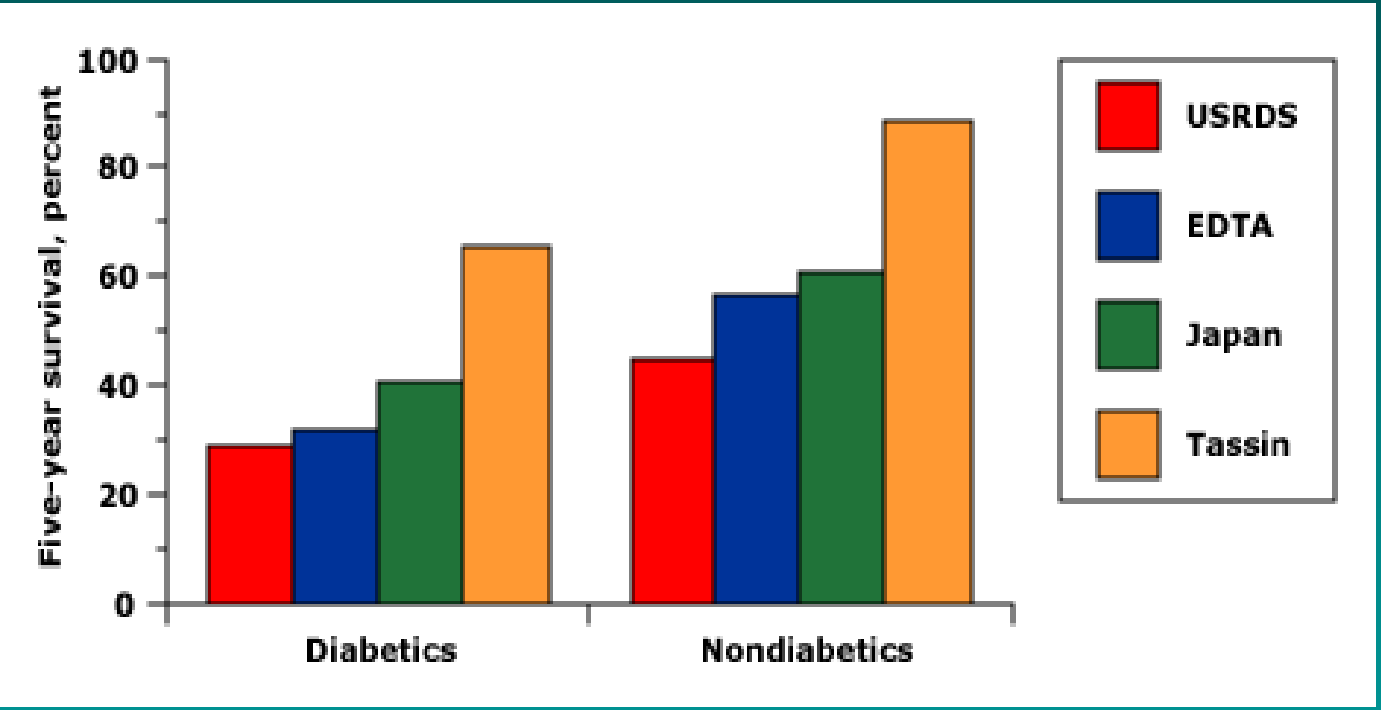
- % Survival in diabetics (top) and non-diabetics (bottom)
- AVF has significantly less mortality





Dhingra, RK et al. Kidney International (2001) ***60****, 1443–1451*

Mortality in ESRD



Hull, AR, Parker, TF III, Am J Kidney Dis 1990; 15:375, and Charra, B, Calemard, E, Ruffet, M, et al, Kidney Int 1992; 41:1286.

Mortality in ESRD

- Cardiovascular diseases 50%
- Infection 15-20%

Vaccines

- Hepatis B series with periodic monitoring to ensure immunity
- Annual flu shot
- Pneumococcal vaccines
  - Age <65 with CKD, PCV-13 first, followed by PPSV-23 no sooner than 8 weeks later
  - Age >65
    - Vaccine naïve PCV 13, then PPSV-23
    - If PPSV-23 after age 65, then PCV 13
    - If PPSV-23 before age 65, then PCV 13 followed by PPSV-23

Case 1

- A 52-year-old woman with type 2 diabetes mellitus and hypertension comes for a routine office visit.
- She has a 30-pack-year history of cigarette smoking. Her mother had diabetes and was on hemodialysis.
- Medications are insulin; metoprolol, 100 mg/d; fosinopril, 40 mg/d; hydrochlorothiazide, 50 mg/d; atorvastatin, 40 mg/d; and aspirin, 81 mg/d.
- On physical examination, blood pressure is 165/95 mm Hg. There are retinal microaneurysms. Cardiac examination reveals a regular rhythm with an S4. The lungs are clear to auscultation. There is no jugular venous distention. There is 1+ pedal edema. The distal pulses are absent in both feet.
- **Laboratory Studies**Hemoglobin A1C 7.2%, Glucose 180 mg/dL, Creatinine 1.2 mg/dL, 24-Hour urinary protein excretion 1.8 g/24 h.

Case 1 (cont.)

Which of the following factors is most likely to cause this patient's chronic kidney disease to rapidly progress to end-stage renal disease?

A Poorly controlled diabetes

B Family history

C Poorly controlled hypertension

D Proteinuria

E Cigarette smoking

Case 2

- A 47-year-old woman with type 2 diabetes mellitus and hypertension comes for a routine office visit. Her mother had diabetes and was on hemodialysis.
- Medications are insulin, hydrochlorothiazide, simvastatin, and aspirin.
- Her blood pressure is 140/90 mm Hg, but physical exam is otherwise normal.
- Laboratory studies: hemoglobin A1C 7.9%, creatinine 1.3mg/dL, potassium 4.0mg/dL, glucose 170mg/dL, 24-hour urinary protein 2.1g/24 hours.

Case 2…

- Which of the following medications would have the most beneficial effect on slowing the progression of her kidney disease?
- Atenolol
- Amlodipine
- Lisinopril
- Hydralazine

Case 3

- A 60-year-old woman with hypertension, type 2 diabetes mellitus, obesity, and chronic kidney disease comes for a routine office visit. She follows a renal diet.
- Medications are glipizide, 10 mg twice daily; pioglitazone, 30 mg/d; atenolol, 100 mg/d; benazepril, 80 mg/d; furosemide, 40 mg twice daily; simvastatin, 40 mg/d; and aspirin, 81 mg/d.

- Laboratory Studies: Creatinine 2.3 mg/dL, Sodium 140 meq/L, Potassium 5.0 meq/L, Chloride 106 meq/L, Bicarbonate 23 meq/L, Calcium 9.7 mg/dL, Phosphorus 6.9 mg/dL, Albumin 4 g/dL, Hgb 12.5, Parathyroid Hormone 50

Case 3…

Which of the following should be started to most appropriately treat this patient?

A. Sevelamer (phosphate binder)
B. Erythropoetin
C. Calcitriol (1,25 dihydroxy vitamin D)
D. Hemodialysis

Case 4

- A 60-year-old woman with a history of stage IV chronic kidney disease is anticipated to require dialysis therapy within the next year.
- She has a history of longstanding hypertension and Crohn's disease. She has undergone multiple bowel resections and has had one small-bowel obstruction.
- Medications are enalapril, 10 mg/d; furosemide, 60 mg/d; aspirin, 81 mg/d; and sevelamer, 800 mg three times daily.
- On physical examination, pulse rate is 80/min and blood pressure is 130/80 mm Hg. Cardiac and pulmonary examinations are normal. Radial, femoral, dorsalis pedis, and posterior tibial pulses are 2+. Cephalic veins measuring approximately 5 mm in diameter are present in both forearms. There is trace pretibial edema.

Case 4…

Which of the following is the most appropriate management for this patient?

A Placement of a tunneled central venous catheter when dialysis is needed

B Placement of a polytetrafluoroethylene graft now

C Creation of an arteriovenous fistula now

D Placement of a peritoneal dialysis catheter when dialysis is needed

CASE 5

- 75 yo white man with a history of CKD presents for evaluation of resistant HTN. Average home BP is 170/94. His BP medications are: losartan 100mg qd, amlodipine 10mg qd, chlorthalidone 25mg qd, and carvedilol 25mg bid. On exam, his BMI is 23, BP 174/96. He has hypertensive retinopathy and a S4 gallop. No edema. Labs show creatinine of 2.1 (eGFR 1.73)

Which of the following is the most likely to be associated with his resistant hypertension?

A. His race

B. His age

C. Underlying CKD

D. His BMI

Summary

- Primary disease should be treated
  - blood pressure control
  - glycemic control
  - immunosuppression if warranted
- Regardless of the cause of chronic kidney disease, treatment must focus on slowing the secondary adaptive hemodynamic and structural changes in the kidney
  - blood pressure control
  - ACE-Inhibitor/ARB
  - low protein diet

Summary (cont.)

- Measures must also be taken to prevent comorbidities
  - **BP CONTROL**
  - Glycemic control
  - Phosphorus lowering
  - Vitamin D therapy for secondary hyperparathyroidism
  - Lipid lowering
  - Anemia management

Summary (cont.)

- Once GFR is less than 30…
  - Dialysis access planning
  - Evaluation for transplantation