Chronic Kidney Disease
For the Primary Care Physician

Monita Poudyal, M.D.
February 6-8, 2020
DISCLOSURES

None

Monita Poudyal, M.D.
February 6-8, 2020
1. Understand the definition of CKD and how to screen for it
2. Implement strategies to decrease the progression of CKD
3. Understand how new diabetes drugs impact CKD care
4. Understand how to manage anemia associated with CKD
5. Know when to refer patients to Nephrology
6. Understand that advanced CKD profoundly affects QOL
7. Understand the effect of dialysis on the elderly and the importance of defining goals of care in this population
What is Chronic Kidney Disease?

Case Study: Mrs. A is a 62 yo Caucasion woman with DM, HTN, gout and osteoarthritis. She takes Celebrex, metformin and hydrochlorothiazide and has a stable serum creatinine of 0.9 mg/dl, eGFR 67ml/min/1.72m2 over the past 6 months.

Does she have chronic kidney disease?
A. Maybe, we need more information
B. No, her creatinine is normal
C. No, her eGFR is normal for her age
D. Both B and C
Kidney damage or GFR <60 mL/min / 1.73 m² for >3 mo

Kidney damage can be either functional or structural

➢ Functional abnormalities
  -- Proteinuria, albuminuria
  -- Electrolyte abnormalities due to tubular disorder
  -- Abnormalities of urinary sediment (dysmorphic red cells, cell casts)

➢ Structural abnormalities
  -- On radiological evaluation
  -- Polycystic kidney disease, reflux nephropathy, etc
  -- On biopsy
  -- Transplanted kidney

# Classifying Chronic Kidney Disease

CKD classification according to GFR

<table>
<thead>
<tr>
<th>GFR stages</th>
<th>GFR (mL/min/1.73 m²)</th>
<th>Terms</th>
</tr>
</thead>
<tbody>
<tr>
<td>G1</td>
<td>≥90</td>
<td>Normal or high</td>
</tr>
<tr>
<td>G2</td>
<td>60 to 89</td>
<td>Mildly decreased</td>
</tr>
<tr>
<td>G3a</td>
<td>45 to 59</td>
<td>Mildly to moderately decreased</td>
</tr>
<tr>
<td>G3b</td>
<td>30 to 44</td>
<td>Moderately to severely decreased</td>
</tr>
<tr>
<td>G4</td>
<td>15 to 29</td>
<td>Severely decreased</td>
</tr>
<tr>
<td>G5</td>
<td>&lt;15</td>
<td>Kidney failure (add D if treated by dialysis)</td>
</tr>
</tbody>
</table>

Chart adapted from UpToDate.com
## Classifying Chronic Kidney Disease

**CKD classification according to Albuminuria**

<table>
<thead>
<tr>
<th>Albuminuria stages</th>
<th>AER (mg/day)</th>
<th>Terms</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1</td>
<td>&lt;30</td>
<td>Normal to mildly increased (may be subdivided for risk prediction)</td>
</tr>
<tr>
<td>A2</td>
<td>30 to 300</td>
<td>Moderately increased</td>
</tr>
<tr>
<td>A3</td>
<td>&gt;300</td>
<td>Severely increased (may be subdivided into nephrotic and nonnephrotic for differential diagnosis, management, and risk prediction)</td>
</tr>
</tbody>
</table>

Chart adapted from UpToDate.com
Classification Corresponds to Severity and Prognosis

<table>
<thead>
<tr>
<th>Prognosis of CKD by GFR and Albuminuria Categories: KDIGO 2012</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Persisting albuminuria categories</strong></td>
</tr>
<tr>
<td><strong>Description and range</strong></td>
</tr>
<tr>
<td><strong>A1</strong></td>
</tr>
<tr>
<td>Normal to mildly increased</td>
</tr>
<tr>
<td>&lt;30 mg/g &lt;3 mg/mmol</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Stage</th>
<th>GFR categories (ml/min/1.73m²)</th>
<th>Description and range</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>G1 Normal or high</td>
<td>≥90</td>
<td>55%</td>
</tr>
<tr>
<td>2</td>
<td>G2 Mildly decreased</td>
<td>60-89</td>
<td>33%</td>
</tr>
<tr>
<td>3a</td>
<td>G3a Mildly to moderately decreased</td>
<td>45-59</td>
<td>12%</td>
</tr>
<tr>
<td>3b</td>
<td>G3b Moderately to severely decreased</td>
<td>30-44</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>G4 Severely decreased</td>
<td>15-29</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>G5 Kidney failure</td>
<td>&lt;15</td>
<td></td>
</tr>
</tbody>
</table>

Risk of ESRD, AKI, CV, or Death
- Green: Low
- Yellow: Moderate
- Orange: High
- Red: Very high

Back to Mrs. A

62 yo Caucasian woman with DM, HTN, OA, gout

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCr</td>
<td>0.9, eGFR 67</td>
</tr>
<tr>
<td>UACR</td>
<td>550mg/g</td>
</tr>
<tr>
<td>UA</td>
<td>2+ prot, no cells, +gluc</td>
</tr>
</tbody>
</table>

Diagnosis: Diabetic Kidney Disease
### Screen those at greatest risk

<table>
<thead>
<tr>
<th>Genetic Factors</th>
<th>Sociodemographic Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Diabetes mellitus</td>
<td>➢ Older age</td>
</tr>
<tr>
<td>➢ Hypertension</td>
<td>➢ Black race</td>
</tr>
<tr>
<td>➢ Cardiovascular disease</td>
<td>➢ Smoking</td>
</tr>
<tr>
<td>➢ Urological disorders, esp obstructive uropathies</td>
<td>➢ Heavy alcohol use</td>
</tr>
<tr>
<td>➢ FMHx of polycystic kidney dz</td>
<td>➢ Obesity</td>
</tr>
<tr>
<td>➢ Autoimmune diseases</td>
<td>➢ NSAIDs</td>
</tr>
</tbody>
</table>
Screening tools for CKD

Screening: estimate GFR and test for kidney damage markers

- Serum creatinine and estimated GFR
- Urinalysis for leukocytes and dysmorphic red blood cells and casts
- Urine albumin-to-creatinine ratio
- Renal ultrasound
Case Study: Mrs. A is a 62yo Caucasian woman with DM, HTN, OA, gout. EGFR 67 ml/min/1.73m2, UACR 550mg/g. She is confirmed to have CKD stage G2A3.

For Mrs. A, there is data that prevention of CKD progression might include all of the following EXCEPT:

A. Treating hypertension
B. Using ACEi or ARB
C. Improving diabetic control
D. Correcting metabolic acidosis
E. Treating hyperuricemia
Hypertension is a significant risk factor for development of end-stage renal disease.
Aggressive BP control is more effective in delaying progression in proteinuric CKD. Jafar et al. Annals of Internal Medicine 2003; 139: 244-252.
KDIGO Guidelines for Blood pressure control¹

- ACR <30 mg/g: ≤140/90 mm Hg
- ACR 30-300 mg/g: ≤130/80 mm Hg (or <140/90)*
- ACR >300 mg/g: ≤130/80 mm Hg
- Individualize BP targets and meds according to age, coexistent CVD, and other comorbidities

*Reasonable to select a goal of 140/90 mm Hg, especially for moderate albuminuria (ACR 30-300 mg/g).*²

ACEi decreases risk of ESRD/doubling creatinine, but only for proteinuria >500mg/day

KDIGO Guidelines: Use ACEi or ARBs

➢ Diabetics with ACR 30-300 mg/g
➢ Diabetics and nondiabetics with ACR >300 mg/g
➢ Combination of ACEi and ARB is not recommended (high risk of impaired kidney function and hyperkalemia)

Additional thoughts on HTN management:

➢ Low salt diet (2gm/day) and diuretics can augment ACEi/ARBs effectiveness in controlling BP and proteinuria

➢ Diuretics are typically necessary for BP and volume in CKD
  -- Thiazides are effective in CKD G1-3
  -- Loop diuretics may be needed in CKD G4 or greater

➢ Nondihydropyridine calcium channel blockers (diltiazem, verapamil) can help in proteinuria reduction

➢ For the elderly:
  -- If ambulatory and healthy, same general BP targets
  -- If fragile, tailor medications and BP targets to the individual
Case Study: Mrs. A with CKD has 550mg albuminuria. She takes metformin, HCTZ and Celebrex. You add Lisinopril and maintain BP < 130/80. One month later, her creatinine is 1.2 (baseline 0.9).

What would be the LEAST appropriate next step?

A. As long as K+ is ok, continue Lisinopril
B. Stop Celebrex and recheck creatinine in 1 month
C. Stop HCTZ and recheck creatinine in 1 month
D. Stop Lisinopril and check for renal artery stenosis

Do not ignore a rise in creatinine ≥ 25% after initiation of ACEi/ARB
Case Study: Mr. B is a 72 yo AA man with stage G5 (eGFR 17) CKD and is being prepared for dialysis. He has ischemic cardiomyopathy and diabetes. Meds: Lisinopril, Metoprolol, Insulin, Torsemide, Rosuvastatin, and ASA.

What is the data on stopping RAAS inhibitors in advanced CKD?

A. RAAS inhibitors have significant cardiovascular protective benefits in advanced CKD, so discontinuation is not advised
B. Discontinuation of RAAS inhibitors may allow for improved GFR and postponement of dialysis
C. Discontinuation of RAAS inhibitors might accelerate kidney damage, so should be avoided
ACEi/ARBs in advanced CKD

ACEi discontinuation improves eGFR in advanced CKD: a small observational study


N= 52
Avg eGFR 16 ml/min
The decision to continue or discontinue ACE inhibitor/ARB use at CKD stage 4 or 5 is controversial.
CKD causes acidosis which in turn perpetuates renal injury. Mechanisms:
Retrospective Cohort Study: Bicarbonate level ≤22 mmol/L associated with progression of kidney disease, independent of baseline eGFR.

Risk for Progression

<table>
<thead>
<tr>
<th>Bicarbonate Quartiles (meq/L)</th>
<th>Hazard Risk</th>
</tr>
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<tbody>
<tr>
<td>≤ 22</td>
<td>2.4</td>
</tr>
<tr>
<td>23-24</td>
<td>2.2</td>
</tr>
<tr>
<td>25-26</td>
<td>2.0</td>
</tr>
<tr>
<td>≥ 27</td>
<td>1.8</td>
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</tbody>
</table>

- * p<0.001
- ** p=0.006

N= 5422

Key Points on Metabolic acidosis:

- Seldom significant until GFR <30 mL/min
- Contributes to CKD progression, insulin resistance, decreased cardiorespiratory fitness, altered bone metabolism
- Meta-analysis of small RCTs: Bicarbonate supplementation in CKD improves kidney function\(^a\)
- Use alkali therapy with serum bicarbonate <22 mmol/L to maintain serum bicarbonate levels >22\(^b\) and within normal range\(^c\)
- Typical prescription: 650mg sodium bicarbonate bid, up to 1300mg bid (watch for volume overload!)

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Slowing progression of CKD: Diabetes Mellitus

➢ Good glycemic control reduces:
  ➢ Progression of CKD
  ➢ Incidence proteinuria

➢ KDIGO CKD guidelines recommend a goal A1c level ~7% \(^1\)

➢ ADA recommends goal A1c level <8% for patients at risk for hypoglycemia (advanced age, vascular disease, CKD, multiple comorbidities) \(^2\)

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Kidney’s Role in Glucose Homeostasis:\(^1\):
1. Gluconeogenesis
2. Glucose resorption from filtrate

As GFR declines, you get:
1. ↓ capacity for renal gluconeogenesis
2. ↓ elimination of glucose lowering drugs.
3. ↓ peripheral degradation of insulin
4. Uremia, anorexia, low glycogen stores

Hypoglycemia is a real threat in CKD, with or without diabetes\(^2\)

\(^1\) J Am Soc Nephrol 28: 2263–2274, 2017
\(^2\) Moen, et al. CJASN June 2009, 4 (6) 1121-1127
Decrease CKD progression by:

- **Treating hypertension**
  Goal BP $\leq 140/90$ if no albuminuria, BP $\leq 130/80$ if albuminuria is present
  Goal for the frail and elderly should be customized

- **Using ACEi/ARBs for HTN and albuminuria**
  ACEi/ARBs dramatically affect glomerular hemodynamics
  Withdrawal of ACEi/ARBs may improve eGFR, delay dialysis in stage 4-5 CKD
  CV benefits of RAAS inhibition in pre-dialysis CKD is not well-defined

- **Treating acidemia, when bicarb $<22$ mEq/L**

- **Treating Diabetes**
  Goal HgA1c $\sim 7$ but $<8$ for those at risk for hypoglycemia
Sodium-glucose Transport Protein-2 Inhibitors:

- Inhibit SGLT-2, cause glucosuria
- Reduce cardiovascular risk
- Reduce blood pressure
- Reduce proteinuria and progression of CKD
- Adverse effects: UTIs, genital mycotic infection, osmotic diuresis, fractures
- Weak glucose lowering agents: decrease A1C by 0.5%–0.7%
- Costly: 2\textsuperscript{nd}/3\textsuperscript{rd} line agent
New diabetic drugs and CKD: SGLT-2 inhibitors

Meta-Analysis: SGLT2 inhibitors reduce the risk of dialysis, transplantation, AKI, or death due to kidney disease in DM T2 (vs PBO).

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Relative risk (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dialysis, transplantation or death due to kidney disease</td>
<td>0.67 (0.51-0.8)</td>
</tr>
<tr>
<td>ESKD</td>
<td>0.65 (0.53-0.81)</td>
</tr>
<tr>
<td>Substantial loss of kidney function, ESKD, death due to CV disease or kidney disease</td>
<td>0.71 (0.63-0.82)</td>
</tr>
<tr>
<td>AKI</td>
<td>0.75 (0.66-0.85)</td>
</tr>
</tbody>
</table>

SGLT2 inhibitors studied: [canagliflozin](#), [dapagliflozin](#), [empagliflozin](#)

4 RCTs
N=38,723

Neuen et al. *Lancet Diabet Endocrin* 2019 Sep 5. Epub online
<table>
<thead>
<tr>
<th>New diabetic drugs and CKD: Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Decrease in A1C with Monothx</strong></td>
</tr>
<tr>
<td>------------------------------------</td>
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<tr>
<td><strong>SGLT-2 Inhibitors</strong></td>
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<tr>
<td><strong>GLP-1 Agonists</strong></td>
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<tr>
<td><strong>DPP-4 Inhibitors</strong></td>
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</tbody>
</table>

* Others require dose adjustment according to eGFR

1. Neuen et al. *Lancet Diabet Endocrin* 2019 Sep 5. Epub online
The Prevalence of Anemia Rises with CKD Stage

Anemia is a Mortality Risk Multiplier

CKD and Anemia

Herzog, et al. Journal of Cardiac Failure Vol. 10 No. 6, p467-472, 2004
Anemia has severe consequences in CKD:

- Reduced total oxygen delivery
- Reduced quality of life:
  - Fatigue, reduced exercise capacity
  - Reduced cognitive function
- Increased compensatory cardiac output
- Progressive cardiac and therefore renal damage
- Left ventricular hypertrophy
Mechanisms:

a) Erythropoietin deficiency---Epo enhances maturation of erythroblastic cells and prevents apoptosis of marrow cells.

b) Iron and Functional iron deficiency

c) B12, Folate deficiency

d) Blood loss---phlebotomy and blood trapping in dialysis circuit

e) Shortened RBC survival

f) Uremia induced inhibition of erythropoiesis

g) Hyperparathyroidism-- bone marrow fibrosis, PTH-inhibited Epo synthesis
Case study: Mr. D is a 55 yo man with DMT2, stage 3 CKD, and chronic uninfected non-healing wounds. Labs: Hgb 10, ferritin 400 and TSAT 19%. He complains of fatigue.

What would be the LEAST appropriate next step in management?

A. Start ESA injections
B. Stool testing for occult blood
C. Check B12 and Folate levels
D. Check absolute reticulocyte count
E. Start iron supplementation
Hepcidin and Epo are central to hematopoietic balance

Figure adapted from: JASN October 2012, 23 (10) 1631-1634
Anemia and CKD: pathophysiology

CKD disrupts hematopoietic balance

Figure adapted from: JASN October 2012, 23 (10) 1631-1634
Male: Hgb < 13 g/dl or Female: Hgb < 12 g/dl

Rule out Non-Renal Cause of Anemia
- CBC, Abs Retic, Ferritin, TSAT, Vit B12, Folate, Stool testing for blood

TSAT < 30% and Ferritin < 500 ng/ml?

- YES: Iron/Functional Iron Deficiency
  - Provide po/IV iron
  - Target Hemoglobin 9-11.5

- NO: Epo Deficiency
  - Consider ESA if Hgb < 10

### TREAT rct: Using ESA in CKD to achieve target Hgb 13 increases risk of stroke

<table>
<thead>
<tr>
<th></th>
<th>Darbo, Target Hgb 13</th>
<th>PBO, Darbo if Hgb&lt;9</th>
</tr>
</thead>
<tbody>
<tr>
<td>Achieved Hgb</td>
<td>12.5</td>
<td>10.6</td>
</tr>
<tr>
<td>HR for CV events</td>
<td>1.05 (p=0.4)</td>
<td></td>
</tr>
<tr>
<td>HR for Death</td>
<td>1.05 (p=0.48)</td>
<td></td>
</tr>
<tr>
<td>HR for ESRD</td>
<td>1.06 (p=0.29)</td>
<td></td>
</tr>
<tr>
<td>HR for Stroke</td>
<td>1.92 (p &lt;0.001)</td>
<td></td>
</tr>
</tbody>
</table>

N=4038  
Type 2 diabetic, egfr 20-60

Anemia is **modifiable** risk factor for CV morbidity, mortality and poor quality of life

1. **Target Hgb 9-11.5 g/dL**
   BUT individualize targets for younger, active, or symptomatic patients
2. Rule out iron/functional iron deficiency **first**
3. Iron can raise Hgb, delay/prevent the need for ESAs, or lower ESA doses
4. Avoid IV iron in setting of systemic infection
5. Adjust ESA dose according to symptoms, **use lowest possible ESA**
6. If iron stores are adequate, start ESA once Hgb <10
7. **Do not use ESAs to target Hgb > 13; risk for stroke, HTN, CV events***
8. Use caution with ESAs in active malignancy, if cure is anticipated

All of the below should seek nephrology evaluation EXCEPT:

A. Initial visit: eGFR 25 & 3 months later: eGFR 28

B. Initial visit: eGFR 50 & 3 months later: eGFR 50, both dates the ACR < 30 mg/g

C. Initial visit: eGFR 54, & 3 months later: eGFR 44 confirmed with repeat eGFR 45

D. Initial visit: ACR 450 & 3 months later: ACR 400, eGFR 90

E. Initial visit: eGFR 90 & 3 months later: eGFR 90 with recurrent extensive nephrolithiasis
### KDIGO 2012 Guidelines for Referral to Nephrology

- AKI or abrupt sustained fall in GFR (>25%)
- GFR <30 ml/min/1.73 m² (GFR categories G4-G5)
- Significant albuminuria (ACR >300 mg/g, PCR >500 mg/g)
- Progression of CKD (a sustained decline in eGFR of >5 ml/year)
- Urinary red cell casts not readily explained
- Persistent abnormalities of serum potassium
- CKD and HTN refractory to treatment with 4 or more antihypertensive agents
- Recurrent or extensive nephrolithiasis
- Hereditary kidney disease

How does CKD feel?

- Pain
- Pruritis
- Depression/Anxiety
- Sleep Disorders
- Impaired Physical Functioning
- Loss of Energy
- Sexual Dysfunction
- Cognitive Dysfunction
- Anorexia/Nausea
- Constipation/Diarrhea
- Chest pain
- DOE
- Edema
- Impact of dialysis: cramps, access pain, lightheadedness, fatigue, missed meals, travel logistics......

Data shows that QOL scores decrease as CKD progresses.*

*Mujais et al. CJASN August 2009, 4 (8) 1293-1301
## CKD, QOL, possible therapies

<table>
<thead>
<tr>
<th>Selected QOL Elements</th>
<th>Treatment examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression/Anxiety</td>
<td>CBT, Pharmacotherapy (SSRI)</td>
</tr>
<tr>
<td>Pain</td>
<td>Pain management, Exercise, Massage therapy, Acupuncture, Topical analgesics, CKD-MBD treatment</td>
</tr>
<tr>
<td>Sexual Dysfunction</td>
<td>PDE-5 inhibitor (minimal data in ESRD)</td>
</tr>
<tr>
<td>Fatigue</td>
<td>Evaluate for: OSA, Anemia, Malnutrition</td>
</tr>
<tr>
<td>Pruritis</td>
<td>Emollients, UBV phototherapy, Gabapentin, CKD-MBD</td>
</tr>
<tr>
<td>Sleep Disturbance</td>
<td>Sleep hygiene, sleep study, RLS treatment (Ropinirole, Gabapentin), melatonin, CKD-MBD treatment</td>
</tr>
<tr>
<td>Nausea/anorexia</td>
<td>Evaluate for: uremia, gastroparesis</td>
</tr>
</tbody>
</table>

**Bottom line:** You won’t know unless you ask.  
 Treatment takes an interdisciplinary effort.
Final Case: SS is a 75 yo AA woman with hx DM Type II, dementia, CVA and CKD stage V who lives independently. She has had repeated hospitalizations due to weight loss, falls and difficulty completing her ADLs. Patient’s family is adamant that she start dialysis as soon as is necessary.

You, her PCP, are asked to attend the family meeting. What do you advise with regard to ESRD and starting dialysis?

A. Let’s leave it to the Nephrologist and take their lead either way.
B. This is mostly uremia, and dialysis should improve her overall functional status.
C. The patient has a grim prognosis and dialysis may not improve her quality of life
D. A and B
Problems with hemodialysis in the elderly:

- Fistulas that do not mature
- Increased Hospitalizations
- Lower albumin
- Increased risk for depression
- Increased risk for adynamic bone disease
- Increased sensitivity to volume shifts: sub-endocardial ischemia
- Greater time to “recover” from the dialysis treatment
Life expectancy on dialysis depends on your frailty phenotype
Dialysis Does not Prevent Functional Decline in Nursing Home Patients

Change in Functional Status after Initiation of Dialysis in Nursing Home Patients

- Months since Initiation of Dialysis:
  - 3 months: 61% decreased, 39% maintained
  - 6 months: 52% decreased, 48% maintained
  - 9 months: 87% decreased, 13% maintained
  - 12 months: 87% decreased, 13% maintained

N: 3702
Avg Age: 73

Maximum conservative therapy may lead to comparable number of hospital free days as hemodialysis in the elderly with high comorbidity (Observ. study)

Distribution of days survived: MCM vs HD

<table>
<thead>
<tr>
<th>Survival (months)</th>
<th>Hospital-free days</th>
<th>Outpatient Hemodialysis days</th>
<th>Hospital Inpatient days</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-6 months</td>
<td>MCM pts n = 29</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7-18 months</td>
<td>All HD-only pts n = 112</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

N 202
Age ≥70

Carson, et al. CJASN October 2009, 4 (10) 1611-1619
Non-Dialysis Maximum Conservative Management is active treatment with:

- Advanced care planning, implementing patient’s goals
- Management of anemia, bone disease, fluid balance, acidosis, MBD
- Discontinue Acei
- Low protein diet (0.8g/kg)
- Candidates
  - stage V CKD
  - typically older than 75 years
  - have high level of comorbidity
  - significantly impaired functional status
  - serum albumin <2.5 g/dl
1. Progression of CKD can be delayed by:
   a. Management of HTN
   b. Use of ACEi/ARBs
   c. Treatment of Acidemia
   d. Control of Diabetes

2. New diabetes drugs (SGLT-2 inhibitors, GLP-1 agonists, DPP-4 inhibitors) demonstrate some promising renoprotective benefits but are still 2nd/3rd line agents due to cost and safety concerns.

3. Anemia in CKD is multifactorial, but address iron deficiency/functional iron deficiency before initiating ESA therapy.
4. Advanced CKD and ESRD have a comparable and profound effect on QOL. Many symptoms like depression, pain and sexual dysfunction can be treated with interdisciplinary effort.

5. The elderly (>65yo) is the fastest growing ESRD population and the most susceptible to adverse effects of dialysis. Longevity on dialysis is a function of frailty.

6. Frail elderly ESRD patients may not substantially benefit from dialysis in terms of QOL and longevity. Non-dialysis “maximum conservative therapy” should be offered. PCP involvement in defining goals of care is critical.
Thank you!