CKD – What’s New?

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Clinical Assistant Professor of Nephrology, UCONN
Chair, Medical Advisory Board, NKF serving Connecticut
Objectives

- When and who to refer?
- What are the means of coordinating care?
- How to monitor?
- What to treat and why?
- When and why to worry about medication safety?
- How do we transition from CKD to beyond?
Patient 1

* 31 y/o WM lawyer seeks second opinion for CKD.
* Type 1 DM diagnosed at the age of 18; + retinopathy
* HTN x 4-5 years; presented with HTN urgency to a suburban ER a year prior. Admitted and evaluated by Nephrology; was told his S Cr was 2.4 mg/dL
* Was on Benicar, Cartia and Insulin; occ Advil use
* Quit smoking a year prior; no EtOH/drug abuse
* No family history of kidney disease
Patient 1

- BP 182/96; no significant arm variation
- Ht 5’ 9” Wt 213 lbs
- +S4 gallop. No bruits. 1+ LE edema
- Over next few years, BP controlled better. Referred and listed for a kidney transplant.
- Over the last 2 years, lost his job and insurance. Ran out of meds including Insulin.

<table>
<thead>
<tr>
<th>Year</th>
<th>2007</th>
<th>2009</th>
<th>2010</th>
<th>2013</th>
<th>2014</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2.4</td>
<td>3.0</td>
<td>3.2</td>
<td>4.0</td>
<td>6.0</td>
</tr>
</tbody>
</table>
Patient 2

- 68 y/o BF referred for worsening renal function - ?DN.
- S Cr of 1.3 mg/dL in 07/08; was 2.9 in 08/09.
- No retinopathy on history.
- Asymptomatic otherwise.
- BP 150/80. P/E unremarkable otherwise
- UA + large blood and 2 g/d protein; +dysmorphic RBCs
- Serologies + p-ANCA and anti-MPO in significant titers
Patient 2

* Renal biopsy with crescentic, pauci-immune GN
* Risks/benefits of immunosuppressive Rx discussed
* Received Cyclophosphamide IV and Pred PO; doing well until she had syncopal episode in casino.
* Found to have Hb of 3.5 g/dL and bleeding peptic ulcer
* Re-bled in hospital and required extensive laparotomy
* All immunosuppressives discontinued; focus on BP control and proteinuria reduction. Hyperkalemia (recurrent) with ACEI/ARB.
Patient 2

* AVF placed in 04/2014 after PD and transplant ruled out
* BP under control
* Anemia – on iron/Epo
* MBD – on Calcitriol

<table>
<thead>
<tr>
<th></th>
<th>2010</th>
<th>2011</th>
<th>2012</th>
<th>2013</th>
<th>2014</th>
</tr>
</thead>
<tbody>
<tr>
<td>Value</td>
<td>2.0</td>
<td>2.4</td>
<td>3.2</td>
<td>4.4</td>
<td>5.6</td>
</tr>
</tbody>
</table>
CKD - Staging

Stage 5 (<15)  N=372,000
Stage 4 (15-29)  N=700,000
Stage 3 (30-59)  N=15,500,000
Stage 2 (60-89)  N=6,500,000
Stage 1 (>90)  N=3,600,000

CV death
CKD – the new paradigm

<table>
<thead>
<tr>
<th>GFR categories (mL/min/1.73 m²)</th>
<th>Description and range</th>
<th>A1</th>
<th>A2</th>
<th>A3</th>
</tr>
</thead>
<tbody>
<tr>
<td>G1</td>
<td>Normal or high</td>
<td>≥90</td>
<td>Monitor</td>
<td>Refer*</td>
</tr>
<tr>
<td>G2</td>
<td>Mildly decreased</td>
<td>60–89</td>
<td>Monitor</td>
<td>Refer*</td>
</tr>
<tr>
<td>G3a</td>
<td>Mildly to moderately decreased</td>
<td>45–59</td>
<td>Monitor</td>
<td>Monitor</td>
</tr>
<tr>
<td>G3b</td>
<td>Moderately to severely decreased</td>
<td>30–44</td>
<td>Monitor</td>
<td>Monitor</td>
</tr>
<tr>
<td>G4</td>
<td>Severely decreased</td>
<td>15–29</td>
<td>Refer*</td>
<td>Refer*</td>
</tr>
<tr>
<td>G5</td>
<td>Kidney failure</td>
<td>&lt;15</td>
<td>Refer</td>
<td>Refer</td>
</tr>
</tbody>
</table>

Who gets to be seen?

<table>
<thead>
<tr>
<th>GFR categories (mL/min/1.73m²)</th>
<th>Description and range</th>
<th>A1</th>
<th>A2</th>
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<td>Normal or high</td>
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<tr>
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<td></td>
</tr>
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<td>Severe decreased</td>
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<td></td>
</tr>
<tr>
<td>G5</td>
<td>Kidney failure</td>
<td>&lt;15</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Persistent albuminuria categories

- **A1**: Normal to mildly increased
- **A2**: Moderately increased
- **A3**: Severely increased

<table>
<thead>
<tr>
<th>&lt;30 mg/g</th>
<th>30-300 mg/g</th>
<th>&gt;300 mg/g</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;3 mg/mmol</td>
<td>3-30 mg/mmol</td>
<td>&gt;30 mg/mmol</td>
</tr>
</tbody>
</table>


- **G1**: 57.9%
- **G2**: 35.4%
- **G3a**: 4.6%
- **G3b**: 1.6%
- **G4**: 0.2%
- **G5**: 0.1%

**Nephrology Consultation 1.6%**
Indications for Nephrology referral for patients with CKD

- AKI or sustained fall in eGFR
- eGFR < 30 ml/min
- Persistent Albuminuria (ACR > 300 mg/g)
- Progression of CKD (25% decline or > 5 ml/min/yr)
- CKD with hypertension refractory to 4 or more agents
- Unexplained red cell casts/need for biopsy
- Recurrent or persistent hyperkalemia
- Hereditary kidney disease
Late nephrology referral before the onset of chronic kidney failure remains common. U.S. data from 2011 reveal 42.1% of new dialysis starts had no prior nephrology care.*

*USRDS 2013 Annual Data Report: Table 1.f (Volume 2) Page 430 Analytical Methods www.usrds.org
Why early vs late?

<table>
<thead>
<tr>
<th>Variable</th>
<th>Early referral mean (SD)</th>
<th>Late referral mean (SD)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall mortality, %</td>
<td>11 (3)</td>
<td>23 (4)</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>1-year mortality, %</td>
<td>13 (4)</td>
<td>29 (5)</td>
<td>0.028</td>
</tr>
<tr>
<td>Hospital length of stay, days</td>
<td>13.5 (2.2)</td>
<td>25.3 (3.8)</td>
<td>0.007</td>
</tr>
<tr>
<td>Serum albumin at RRT start, g/dl [g/l]</td>
<td>3.62 (0.05) [36.2 (0.5)]</td>
<td>3.40 (0.03) [34.0 (0.3)]</td>
<td>0.001</td>
</tr>
<tr>
<td>Hematocrit at RRT start, %</td>
<td>30.54 (0.18)</td>
<td>29.71 (0.10)</td>
<td>0.013</td>
</tr>
</tbody>
</table>

Abbreviation: RRT, renal replacement therapy.

Who should be responsible for patient safety in CKD?

- **Stage 1**: GFR 90
- **Stage 2**: GFR 60
- **Stage 3**: GFR 30
- **Stage 4**: GFR 15

Primary Care Practitioner

Nephrologist

Patient safety
Impact of primary care CKD detection with a patient safety approach
Drug safety in CKD

* AVOID
  * NSAIDs
  * Iodinated contrast
  * Oral Phosphates
  * Gadolinium
  * Glyburide

* CAUTION
  * Metformin
  * Gabapentin
  * Aminoglycosides
  * Digoxin
  * ACEI/ARB
AKI and CKD integration

![Graph showing the proportion surviving over months of follow-up from baseline. The x-axis represents months of follow-up from baseline, ranging from 0 to 100. The y-axis represents the proportion surviving, ranging from 0 to 1. The graph includes lines for different AKI events: No AKI, 1 AKI, 2 AKIs, and 3+ AKIs. The p-value is given as P<0.0001.](image)
How are we following up?

* VA study – 8% of patients saw nephrologist after AKI

* USRDS data – only 13% of patients saw nephrologist within 3 months after AKI. Those with recurrent AKI were seen in only 18% of cases

* Only 75% saw their PCP after hospitalization for AKI and < 50% had repeat S Cr measured within 3 months after AKI

* KDIGO AKI guidelines – “evaluate patients within 3 months of AKI for resolution, new onset or worsening of pre-existing CKD”
How are we following up?

- **AKI Survivors Following Discharge within 30 days**
  - 11.9% Nephrology follow up
  - 29.5% Cardiology follow up
  - 74.5% Primary care visit

- **AKI Requiring Dialysis Survivors Following Discharge**
  - 33% Nephrology visit within 30 days
  - 48.6% Nephrology visit within 1 year

- **Acute Myocardial Infarction Survivors After Discharge**
  - 76% Cardiology Consultation within 30 days
What to expect when we are expecting (to see CKD patients) ?

- Serum creatinine (caveats: equation and changes in range)
- Blood pressure
- Urinalysis including an ACR or spot P/C ratio
- CBC, BMP, extended ‘lytes, albumin and iPTH levels
- Renal imaging
- Depending on H & P:
  - Light chain assay, IFE/SPEP, UPEP
  - HIV and Hepatitis studies
  - Complements and serologies (limited value)
Definitions: Albuminuria and Proteinuria

- **Normal to Moderately Increased Albuminuria**
  - Albumin:creatinine ratio < 30 mg/g creatinine

- **Moderately Increased Albuminuria**
  - Albumin:creatinine ratio 30-300 mg/g creatinine
  - 24-hour urine albumin 30-300 mg/d

- ** Severely Increased Albuminuria**
  - Albumin:creatinine ratio ≥ 300 mg albumin/g creatinine
  - 24-hour urine albumin > 300 mg/d

- **Proteinuria**
  - (+) urine dipstick at > 30 mg/dl
  - > 200 mg protein/g creatinine
  - 24-hour urine protein > 300 mg/d
Potentially modifiable CKD progression risk factors

- Hypertension
- Diabetes/Glycemic control
- Albuminuria/Proteinuria
- Metabolic acidosis
- Obesity ?
- Hyperuricemia ?
- Smoking ?
- Sedentary lifestyle ?
- Dietary Protein Intake ?
CKD as a risk factor for CVD
- Hypertension
- Bone and mineral disorder
- Dyslipidemia
- Sympathetic overactivity
- Salt- and volume overload
- Anemia
- Uremic toxins
- “Undertreatment”
- Immunosuppressants

CVD as a risk factor for CKD
- Hypertension
- Obesity
- Dyslipidemia
- Diabetes
- Acute cardiac events
- CHF/ CAD
  - Underperfusion
  - Toxicity from Dye
  - Cholesterol emboli

Cardiovascular disease
Decline in GFR not always linear!
BP and CKD progression

* Control of BP more important than which agents are used
  * Avoidance of side-effects is important
* With proteinuria: diuretic + ACEi or ARB
* No proteinuria: no clear drug preference
  * ACEi or ARB ok to use
Goals for Renoprotection

* Blood pressure—STILL CONTROVERSIAL
  * $\leq 130-140/80-90$ mmHg if urinary albumin $\geq 30$ mg/d
  * $\leq 140/90$ mmHg if normal urinary albumin

* Proteinuria
  * ACEi or ARB in diabetic with Ualb $\geq 30$ mg/d
  * ACEi or ARB in non-diabetic with Ualb $\geq 300$ mg/d
Multiple agents needed for BP control

Number of Agents Needed

- AASK
- HDT
- MDRD
- ABCD
- UKPDS
- IDNT
- IRMA 2
- RENAAL

Number of Agents Needed: 0, 1, 2, 3, 4
Other Goals of CKD Management

* Target HgbA1C ~ 7%; higher if significant comorbidities or limited life expectancy

* Limit sodium intake to < 90 mmol (2 gm of Na or 5 gm salt) per day

* CVD management: lipids, ASA (secondary prevention), etc
Managing complications
Prevalence of CKD related complications
Anemia

Anemia – to ESA or not?

- Look for and treat iron deficiency: TSAT of < 20%
- Rule out other causes of anemia – B12/Folate/SPEP
- Epogen/Procrit and Darbepoetin or Aranesp
- Cannot start unless Hb < 10 g/dL
- Treat to goal of 11-11.5 g/dL
- Hold for Hb > 12 g/dL
SHPT or ROD or MBD

* Secondary hyperparathyroidism seemed to focus only on the parathyroid, calcium, phosphorus and its problems

* Renal osteodystrophy appeared to give the impression that these abnormalities affected only the bones

* Mineral and bone disorders (MBD) is an all-encompassing term that has been added to the renal landscape in the last decade
## Elevated iPTH – D/Dx

<table>
<thead>
<tr>
<th>Calcium</th>
<th>Phos</th>
<th>iPTH</th>
<th>Suggested Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal or ↓</td>
<td>Normal or ↑</td>
<td>↑</td>
<td>Secondary hyperparathyroidism due to CKD</td>
</tr>
<tr>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>Secondary hyperparathyroidism due to vitamin D deficiency</td>
</tr>
<tr>
<td>↑</td>
<td>↑</td>
<td>↑↑↑↑</td>
<td>Tertiary hyperparathyroidism in advanced CKD</td>
</tr>
<tr>
<td>High-normal or ↑</td>
<td>Low-normal or ↓</td>
<td>High-normal or ↑</td>
<td>Primary hyperparathyroidism or familial hypocaliuric hypercalcemia</td>
</tr>
<tr>
<td>↑</td>
<td>Variable</td>
<td>↓</td>
<td>Non-iPTH related process (e.g. vitamin D toxicity, PTH-rp)</td>
</tr>
</tbody>
</table>

Adapted from Estrella M, Sisson S. CKD Module. Internet Learning Center, 2014.
Mineral Bone Disease KDIGO
Treatment Goals

- Bone density testing (DEXA) does not predict fracture risk in stage 3-5 CKD.

- **Goals**
  - Maintain calcium and phosphorus levels in normal reference ranges
  - Maintain iPTH
    - High-normal (~55 pg/mL) for Stage 3 & 4 (eGFRs 15-59 mL/min)
    - 2-9x normal for Stage 5 (eGFRs <15 mL/min)

Calcium and 25-OH Vitamin D in Stage 3-4 CKD

- Keep corrected serum calcium within normal range preferably toward the lower end (8.4 to 9.5 mg/dL)
- Vitamin D2 if serum 25-OH vit D level <30 ng/mL
  - Cholecalciferol 800 IU daily
- Treat with active oral vitamin D if serum 25(OH) vitamin D >30 ng/mL and iPTH is above target range
  - Calcitriol: 0.25 mcg 3x/wk-daily
  - Doxercalciferol: 2 mcg 3x/wk-daily
  - Paricalcitol: 2 mcg 3x/ wk-daily
Bisphosphonates for osteoporosis

- Safety and efficacy unclear in CKD

- Treat as in the general population (w/ dose adjustment) if:
  - Stages 1-2 CKD
  - Stage 3 CKD w/ normal iPTH

- Exclude other potential forms of bone disease in those w/ Stages 4-5.
When and what to test?

<table>
<thead>
<tr>
<th>CKD Stage</th>
<th>Calcium, Phosphorus</th>
<th>PTH</th>
<th>25(OH)D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 3</td>
<td>Every 6-12 months</td>
<td>Once then based on CKD progression</td>
<td></td>
</tr>
<tr>
<td>Stage 4</td>
<td>Every 3-6 months</td>
<td>Every 6-12 months</td>
<td></td>
</tr>
<tr>
<td>Stage 5</td>
<td>Every 1-3 months</td>
<td>Every 3-6 months</td>
<td></td>
</tr>
</tbody>
</table>
Phosphate binders

- Aluminum hydroxide
- Calcium carbonate
- Calcium acetate (PhosLo)
- Sevelamer carbonate (Renvela)
- Fosrenol (Lanthanum)
- Velphoro (Sucroferric oxyhydroxide)

Least Expensive

Most Expensive
Other MBD issues

- Goal iPTH unclear – when to start and what number to treat
- Do NOT use calcimimetics
- Unclear role of DEXA scans with eGFR < 45 ml/min/1.73m²
- Use bisphosphonates as in general population if eGFR > 30 ml/min/1.73m²
  - Avoid in most patients with lower eGFR
Metabolic acidosis

- Often becomes apparent at GFR < 25-30 ml/min
  - More severe with higher protein intake
- May contribute to bone disease, protein catabolism, and progression of CKD

Adults with CKD (eGFR 15-30 ml/min/1.73m²) with bicarbonate 16-20 mmol/L; treated with sodium bicarbonate for 2 years to normalize serum bicarbonate concentration
What’s bad about acid?

Bicarb – the good and bad

![Graph showing dialysis free survival over months with intervention and control groups.]

**Table 3. Adverse events during the study period**

<table>
<thead>
<tr>
<th>Adverse Events</th>
<th>Control (% of Patients)</th>
<th>Bicarbonate (% of Patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospitalization for CHF</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Worsening hypertension requiring increase in therapy</td>
<td>48</td>
<td>61</td>
</tr>
<tr>
<td>Worsening edema requiring increase in loop diuretics</td>
<td>30</td>
<td>39</td>
</tr>
<tr>
<td>Bad taste requiring switch to powder form of sodium bicarbonate</td>
<td>N/A</td>
<td>6.5</td>
</tr>
</tbody>
</table>
Dietary acid load

PRAL = Potential renal acid load

Metabolic acidosis

* Maintain serum bicarbonate ≥ 22 mmol/L
  * Start with 0.5-1 mEq/kg per day
  * Sodium bicarbonate tablets
    * 325mg, 625 mg tablets; 1 g = 12 mEq
  * Sodium citrate solution
    * 1 mEq/ml
  * Avoid if on aluminum phosphate binders
  * Baking soda
    * 54 mmol/level tsp
## Hyperkalemia – who is at risk?

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Odds Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female vs. male</td>
<td>0.61</td>
<td>0.57, 0.66</td>
</tr>
<tr>
<td>Black vs. white</td>
<td>1.29</td>
<td>1.25, 1.32</td>
</tr>
<tr>
<td>Either ACEi/ ARB</td>
<td>1.41</td>
<td>1.37, 1.44</td>
</tr>
<tr>
<td>Both ACEi/ ARB</td>
<td>1.67</td>
<td>1.55, 1.80</td>
</tr>
<tr>
<td>Cancer</td>
<td>1.16</td>
<td>1.13, 1.19</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1.51</td>
<td>1.47, 1.55</td>
</tr>
<tr>
<td>CVD</td>
<td>1.14</td>
<td>1.12, 1.17</td>
</tr>
<tr>
<td>CKD Stage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>2.24</td>
<td>2.17, 2.30</td>
</tr>
<tr>
<td>4</td>
<td>5.91</td>
<td>5.63, 6.20</td>
</tr>
<tr>
<td>5</td>
<td>11.00</td>
<td>10.34, 11.69</td>
</tr>
</tbody>
</table>

Drug-Induced Hyperkalemia in CKD

<table>
<thead>
<tr>
<th>Mechanisms</th>
<th>Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Impaired RAS function</td>
<td>ACEi/ ARBs, β-blockers, heparin, NSAIDs, COX-2 inhibitors</td>
</tr>
<tr>
<td>Altered K+ distribution</td>
<td>Insulin antagonists, hypertonic solutions, digoxin, β-blockers</td>
</tr>
<tr>
<td>Increased K+ load</td>
<td>K+ supplements, herbal supplements, PRBC infusions</td>
</tr>
<tr>
<td>Reduced K+ excretion</td>
<td>K+ sparing diuretics, calcineurin inhibitors, TMP-SMX, pentamidine, lithium</td>
</tr>
</tbody>
</table>

K/DOQI Guidelines on Hypertension and Antihypertensive Agents in CKD
# Acute Management of Hyperkalemia

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Expected serum K+ ↓</th>
<th>Peak effect</th>
<th>Duration</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV Calcium chloride</td>
<td>None</td>
<td>Instant</td>
<td>Transient</td>
<td>Stabilize myocardium</td>
</tr>
<tr>
<td>Insulin + dextrose</td>
<td>0.5-1 mEq/L</td>
<td>30-60 mins</td>
<td>4-6 hrs</td>
<td>Cellular shift</td>
</tr>
<tr>
<td>B2-adrenergic agonists</td>
<td>0.5-1 mEq/L</td>
<td>30 mins</td>
<td>2 hrs</td>
<td>Cellular shift</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>Variable depending on acidosis</td>
<td>4h</td>
<td></td>
<td>Cellular shift</td>
</tr>
<tr>
<td>Loop/ thiazide diuretics</td>
<td>Hours</td>
<td></td>
<td>↑ renal K+ excretion</td>
<td></td>
</tr>
</tbody>
</table>
Chronic Management of Hyperkalemia

- Loop or thiazide diuretics

- Laxatives
  - As effective as cation exchange resins in sorbitol
  - Those that induce secretory diarrhea may be more effective (e.g. bisacodyl)

- Cation exchange resins
  - Sodium polysterene sulfonate (SPS®, Kayexalate®)
  - Mechanism
    - Theoretical: Bound Na+ exchanged for K+ in colonic/rectal lumen
    - Likely: Accompanying sorbitol induces diarrhea
  - Usually requires multiple doses
  - Risk of bowel necrosis or perforation
SPS-Associated Colonic Necrosis

- Initial cases reported in post-op or ICU pts who received enemas
- More recent cases received oral form in non-post-op patients
- Secondary to sorbitol or crystalization of resin within colonic mucosa
- Avoid in post-op patients, those with ileus or bowel obstruction

Managing the CKD patient

1. Assess GFR
2. Determine etiology; consider renal biopsy, etc.
3. Identify reversible factors
   Workup: complete H&P; cbc, electrolytes, bicarbonate, calcium, phosphate, albumin, urinalysis, SPEP/UPEP, assess proteinuria, renal ultrasound

Progressive CKD
1. Reduce progression
2. Manage comorbid conditions
3. Manage complications of CKD
   Treat BP, use ACEI/ARB, evaluate and treat hyperlipidemia, calcium/phosphate/iPTH, anemia; smoking cessation; preserve vessels; patient education

Approaching ESRD
1. Patient/family education
2. Chose RRT modality
3. Referral for transplant evaluation
4. Dialysis access

Serum creatinine

Years
Why see the nephrologist?

• 20-50% lower overall mortality
• More likely to have AV fistula for access
• Fewer dialysis catheters, less bacteremia
• Better control of BP, anemia and MBD
• Shorter hospital LOS if admitted to start dialysis
• Lower costs of care
Collaborative care agreements

* Defines responsibilities of primary care
  - Provide pertinent clinical information to inform the consultation prior to the scheduled visit.
  - Initiate a phone call if the condition is emergent
  - Provide timely referrals with adequate number of visits to treat the condition.

* Defines responsibilities of nephrologist

  Timely communication of consultation (7 days routine & 48 hours emergent) – fax if no electronic information sharing
  - No consultation to other specialist initiated without primary care input
Why Are Patients Referred Late?

Some patient- or disease-related factors cannot be controlled by the clinician, but there are physician-related factors that can be improved:

- CKD often unrecognized (most is asymptomatic)
- Underestimated value of pre-dialysis nephrology care
  - Nephrologist not just dialysis provider
    - Plays major role in caring for CKD
- Family or primary care physician unsure of indications for referral
What Is Late Referral for CKD?

● “Common” definition:
  ○ < 3 months before dialysis

● Better (for patient) definition:
  ○ When management could have been improved by earlier contact with renal services

Consider: any patient with ≥ stage 3 CKD may benefit from early referral
PPPML infectious hosp. costs during the transition to ESRD, by dataset, 2007

Incident Medicare (age 67 & older) & MarketScan (younger than 65) ESRD patients, 2007.
PPPM CVD hospitalization costs during the transition to ESRD, by dataset, 2007.

Incident Medicare (age 67 & older) & MarketScan (younger than 65) ESRD patients, 2007.
PPPM inpatient costs during the transition to ESRD, by dataset, 2007

Incident Medicare (age 67 & older) & MarketScan (younger than 65) ESRD patients, 2007.
Overall PPPM costs during the transition to ESRD, by dataset, 2007

Incident Medicare (age 67 & older) & MarketScan (younger than 65) ESRD patients, 2007.
Referral = Collaboration with Nephrologist

Early Referral ≠ Loss of Patient
= Co-operative care
  - May keep patient healthier longer
  - May delay dialysis
  - Patient may live longer
= Improved patient outcomes
= Savings to health care system
Nephrologists – near extinct?

- Number of practicing nephrologists in the US: 6,891 (1 per 43,300 population) – 2007 data
- Percentage of nephrologists > 55 yrs: 30%
- Number of graduating nephrology fellows each year in the US: 350
- Number of those entering clinical practice: 200
- Number of retiring nephrologists each year: 300
- Number of CKD III patients currently: 12 million
- Number of CKD patients (exp) in 2025: 30 million
- Number of ESRD patients now: 500,000
Is earlier start any better?

- RCT of > 800 patients with eGFR 10-15 mL/min
  - Start HD early or wait until eGFR 5-7 mL/min
  - 76% of late start group started with higher eGFR due to symptoms or other indication

- No difference in survival, other outcomes, QOL

- Conclusion: OK to delay dialysis until GFR < 7 mL/min or other specific clinical indicators for the initiation of dialysis are present such as uremic symptoms, declining nutritional status
Renal function in the elderly

Per decade decline:

• 19 mL/min in men
• 15 mL/min in women
• Many elderly have low GFR without albuminuria and are at very low progression risk
Approach to older adults with CKD

- Maintain independence
- Relieve suffering
- Spend time with family
- Prevent disease progression
- Improve survival

Kidney damage, ↓ GFR

Functional Status

Geriatric Syndromes

Symptoms

Social Support

Outcomes
- Individualized and based on patient preferences
- Not determined by specific disease
- Outcomes including, but not limited to, kidney failure and death
Risk of ESRD vs death

Risk of ESRD > Risk of Death
Thank you for the opportunity!