Recognizing and Treating Chronic Kidney Disease (CKD)

Nephrology Certification Review Course
June 1, 2014

Objectives

- Identify patients who have or are at risk for chronic kidney disease (CKD).
- Describe strategies to slow progression to End-Stage Renal Disease (ESRD).
- Discuss occurrence, prevention, and treatment of complications and co-morbid conditions of CKD.
- Develop strategies for collaborative treatment of CKD.

Take Home Messages

“You can’t find what you’re not looking for!”

“If you wait, it’s too late!”

“Don’t get caught with your pants down!”
More than 8 million Americans have substantial kidney impairment, and 10 million more have albuminuria.

Problem is bigger than nephrology

1) 7.6 million people with GFR 30-60 ml/min/1.73m²
2) About 4,500 full-time nephrologists
3) Nearly 2,000 new patients per nephrologist

Therefore, 7 new patients per day per nephrologist
**Classification tree, with significant interactions**

USRDS 2008

**What’s the most common sign or symptom of early kidney disease?**

Asymptomatic

**Definition of CKD**

Kidney damage (structural or functional)

OR

GFR < 60 ml/min/1.73 m² for greater than 3 months

**Stages of CKD**

<table>
<thead>
<tr>
<th>Stage</th>
<th>GFR</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>&gt;120</td>
<td>At risk Kidney Damage</td>
</tr>
<tr>
<td>1</td>
<td>90</td>
<td>Mild Kidney Failure</td>
</tr>
<tr>
<td>2</td>
<td>60</td>
<td>Moderate Kidney Failure</td>
</tr>
<tr>
<td>3</td>
<td>30</td>
<td>Severe Kidney Failure</td>
</tr>
<tr>
<td>4</td>
<td>15</td>
<td>Kidney Failure</td>
</tr>
</tbody>
</table>

GFR: mL/min/1.73 m²

**NKF-K/DOQI Staging Classification of CKD**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
<th>GFR/Focus of Care</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Chronic kidney damage with normal or ↑ GFR</td>
<td>&gt;90 ml/min Screen for CKD &amp; ↓ Risk</td>
</tr>
<tr>
<td>2</td>
<td>Mild ↓ GFR</td>
<td>60–89 * Diagnose &amp; Treat to slow progression &amp; reduce risk</td>
</tr>
<tr>
<td>3</td>
<td>Moderate ↓ GFR</td>
<td>30–59 Neph/Tx referral</td>
</tr>
<tr>
<td>4</td>
<td>Severe ↓ GFR</td>
<td>15–29 Prepare KRT</td>
</tr>
<tr>
<td>5</td>
<td>Kidney failure</td>
<td>&lt;15 Dialysis</td>
</tr>
</tbody>
</table>

GFR: mL/min/1.73 m²

*May be normal for age


**High Risk Patients**

- Diabetes
- Hypertension
- Aging (>50 years old)
- Racial-ethnic background
  - (African American, Native American, Asian-American, Pacific Islander, Latin American, Hispanic)
- Family History of kidney disease

Primary Diagnoses for Patients Who Start Dialysis

Diabetes 50%
Hypertension 27%
Glomerulonephritis 13%
Other 10%

United States Renal Data System (USRDS), 2000. www.hypertensiononline.org

Risk Factors for CKD Early Detection
- Male gender
- Tobacco use
- Low Income/education
- UTI, urinary stones, lower UT obstruction
- Autoimmune disease
- AKI – Recovery
- Neoplasia
- Anemia
- High-protein diet
- Hyperlipidemia
- Atherosclerosis
- Obesity
- Exposure to nephrotoxic drugs
  - NSAIDS, Cox 2
  - Contrast dye
- Neoplasia
- Anemia


Recommended Screening Tests For Patients at Risk for CKD

Screening is the beginning of a complex management process for CKD.
- Serum creatinine (SCr) – Use prediction equation
- Blood pressure – Early factor
- Glucose – Early factor
- Urinalysis
- Microalbuminuria/proteinuria – Diabetics

Measures for Defining CKD

- Glomerular filtration rate (GFR)
  - Best indicator, usually estimated
- Serum creatinine (SCr)
  - Women ≥1.2 mg/dl
  - Men ≥ 1.4 mg/dl
  - Over 65 years old >1.2 mg/dl
- Creatinine clearance (CrCl) <60 mL/min
  - 24-hour specimen may be required

CASE

- Case:
  - 86-year-old woman, 66-kg body weight
  - Hospitalized March & April, Hct 16%, GI work-up negative, Transfused
  - May – Admit with weakness, Hct 27.5%, K+ 6.9 mg/dl, Creatinine 1.8 mg/dl
  - Nephrology consult

CASE

- Case:
  - What, if any, level of kidney disease?
    - None
    - Mild (GFR 60-90)
    - Moderate (GFR 30-60)
    - Severe (GFR 15-30)
    - Kidney failure (GFR <15)
**Improving Upon SCr Screening Use Prediction Equation**

Cockcroft-Gault (C-G) Method for Estimating Ccr

\[
Ccr = \frac{(140 - \text{age [y]})(\text{body wt [kg]} \times 0.85^*)}{(72)(\text{SCr [mg/dL]})}
\]

- Example:
  - 86-year-old woman, 66-kg body weight, 1.8 mg/dL SCr
- Formula result: STAGE 4 SEVERE KIDNEY DISEASE
  - \(Ccr = 23 \text{ mL/min}\)

* For woman (x 1.0 for men) Cockcroft, 1976.

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**MDRD calculation**

- Calculation is based on age, gender, race, creatinine
- Ex: 50yo 60kg white female-creatinine 0.6 or 1
- Creatinine 0.6mg/dl = 112ml/min
- Creatinine 1mg/dl = 62ml/min
- MDRD calculator websites – click on GFR calculator
  - [www.kidney.com](http://www.kidney.com) or [www.hdcn.com](http://www.hdcn.com)

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**Serum creatinine is not a good indicator of Kidney Function**

Prevalence of abnormal GFR values (\(\leq 50 \text{ ml/min}\)) by age in pts. with normal serum creatinine (\(\leq 130 \text{ umol/l [1.5mg/dl]}\))

<table>
<thead>
<tr>
<th>Age</th>
<th>ALL</th>
<th>&lt; 40</th>
<th>40-49</th>
<th>50-59</th>
<th>60-69</th>
<th>(&gt; 70)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALL</td>
<td>2343</td>
<td>503</td>
<td>396</td>
<td>500</td>
<td>475</td>
<td>248</td>
</tr>
<tr>
<td>Abn. GFR</td>
<td>387</td>
<td>0</td>
<td>3</td>
<td>5</td>
<td>60</td>
<td>316</td>
</tr>
<tr>
<td>%</td>
<td>15.2</td>
<td>0</td>
<td>0.8</td>
<td>1.6</td>
<td>7.8</td>
<td>47.2</td>
</tr>
</tbody>
</table>

Creatinine Alone Is Not Accurate for Predicting Renal Function

- Need to be in steady state
- Diet – Vegans lower
- Less creatinine in malnutrition
- Less muscle mass children, women, elders
- Cimetidine, Septra®, cephalosporins, ketoacidosis increase creatinine
- ~40% of people with decreased GFR have a serum creatinine in lab’s normal range

Recommended Screening Tests for Renal Complications

- Random, spot urine for albumin/creatinine
- Positive if >30 mg/g* or ratio >0.03
- 30 – 300 mg/day albumin excretion = microalbuminuria; >300 macroalbuminuria
- Repeat 2 to 3 times over 6 months, or confirm with 24-hour collection for microalbumin

Who Should Be Treated for CKD?

- Diabetics with urine albumin/creatinine ratios more than 30mg albumin/1 gram creatinine.
- Non-diabetics with urine albumin/creatinine ratios more than 300mg albumin/1 gram creatinine. OR
- Non-diabetics with estimated GFR less than 60 ml/min/1.73m².

* >30 (mg) albumin/ (g) creatinine

Goals of Treatment

Early Detection of CKD

Delay progression of CKD
BP control
BS control
Avoid nephrotoxins
Lifestyle

Prevent complications
Anemia
Malnutrition
Bone disease
Acidosis

Treat co-morbidities
Cardiac disease
Hypertension
Diabetes

Prepare for RRT
Educate patient
Select RRT modality
Create access and initiate dialysis in a timely fashion

Adapted from Pereira, 2000.

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Adapted from Pereira, 2000.

Delay Progression: Hypertension and Proteinuria

GOALS:

With Proteinuria
125/75 or lower

Proteinuria
< 500 - 1000 mg/g

Without Proteinuria
130/80 mm Hg
DM/CKD
(JNC VII, NKF)

National Institutes of Health; National Heart, Lung, and Blood Institute; National High Blood Pressure Program, 1997.
Blood Pressure Is Poorly Controlled in CKD

Hypertension Affects 50 million in US 1 Billion worldwide

Effect of Blood Pressure on Progression of Nephropathy*

*Summary of trials using ACE inhibitors to achieve target BP

Multiple Agents Usually Required to Achieve BP Goals in Diabetic Patients

Number of Agents Needed

Adapted with permission from Diabetes Care 1999;30:640-661

The American Nephrology Nurses’ Association (ANNA)
Chronic Renal Disease: Initial Treatment Recommendations

- CKD
  - Clcr < 60 mL/min
  - Crser > 1.4 mg/dL
- Microalbuminuria (only Abnormality)
- Proteinuria
- Diabetes Mellitus

ACE Inhibitor (or ARB) Start And Titrate To Maximum Tolerable Dose

* For women, Cr > 1.2 mg/dL

Delay Progression: Treat Hypertension and Proteinuria

<table>
<thead>
<tr>
<th>Agent</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE Inhibitors *</td>
<td>↓ Proteinuria, ↓ Decline of GFR, CHF, CAD, Post MI, Stroke Pre.</td>
</tr>
<tr>
<td>ARBs*</td>
<td>↓ Proteinuria, ↓ Decline of GFR, CHF</td>
</tr>
<tr>
<td>Diuretic Thiazide/Loop</td>
<td>Enhance antiproteinuric effect, CHF, CAD, Stroke Pre.</td>
</tr>
<tr>
<td>Aldosterone Receptor Blockers</td>
<td>CHF, Post MI</td>
</tr>
</tbody>
</table>

* Monitor K and Cr.

Delay Progression: Treat Hypertension and Proteinuria

<table>
<thead>
<tr>
<th>Agent</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>CCBs Non-dihydropyridine</td>
<td>↓ Proteinuria, CAD, Arrhythmias</td>
</tr>
<tr>
<td>CCBs Dihydropyridine</td>
<td>Very effective in lowering BP Don't use alone</td>
</tr>
<tr>
<td>Beta Blockers</td>
<td>↓ Proteinuria, CAD Post MI, CHF, arrhythmia</td>
</tr>
</tbody>
</table>

JNC VII, JAMA, 2003
Flack, Prem. On KD, 1998

www.hypertensiononline.org

*for women, Cr > 1.2 mg/dL
Delay Progression: Treat Hypertension and Proteinuria

<table>
<thead>
<tr>
<th>Agent</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central alpha- and Beta-blockers</td>
<td>CHF, Anti-oxidants</td>
</tr>
<tr>
<td>Others: Alpha blockers</td>
<td>Lower BP</td>
</tr>
<tr>
<td>Central alpha2-agonist</td>
<td>BPH, + lipoprotein fractions</td>
</tr>
<tr>
<td>Vasodilators</td>
<td>Useful in BP emergencies</td>
</tr>
<tr>
<td>Combinations</td>
<td>Effective in severe hypertension</td>
</tr>
<tr>
<td></td>
<td>Decrease number of Meds</td>
</tr>
</tbody>
</table>

JNC VII, JAMA, 2003
Flack, Pract. On KD, 1998

Monitoring GFR w/ BP meds

<table>
<thead>
<tr>
<th>Baseline GFR (mL/min/1.73 m²)</th>
<th>After initiation or changes in dose of antihypertensive therapy</th>
<th>After blood pressure is at goal and dose is stable</th>
</tr>
</thead>
<tbody>
<tr>
<td>GFR ≥60</td>
<td>4-12 weeks</td>
<td>6-12 months</td>
</tr>
<tr>
<td>GFR 30-50</td>
<td>2-4 weeks</td>
<td>3-6 months</td>
</tr>
<tr>
<td>GFR &lt;20</td>
<td>1-2 weeks</td>
<td>1-3 months</td>
</tr>
</tbody>
</table>

NKF-K/DOQI guidelines

GFR monitoring w/ ACE/ARB

<table>
<thead>
<tr>
<th>Early decrease in estimated GFR (%)</th>
<th>0-15%</th>
<th>15-30%</th>
<th>30-50%</th>
<th>&gt;50%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dosage adjustment for ACE and ARB</td>
<td>None</td>
<td>None</td>
<td>Reduce</td>
<td>Discontinue</td>
</tr>
<tr>
<td>Recommended interval for monitoring GFR</td>
<td>As per GFR (previous table)</td>
<td>Once et al 10-14 days until GFR is within 15-30% of baseline value, then resume monitoring schedule as per GFR (previous table)</td>
<td>Every 5-7 days until GFR is within 30% of baseline value</td>
<td>Every 5-7 days until GFR is within 15% of baseline value</td>
</tr>
<tr>
<td>Evaluate for causes of decreased GFR (including consideration of RAS, see Guideline 4)</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>
ACE/ARB monitoring intervals

Table 130. Summary of Recommended Intervals to Monitor for Side Effects of ACE inhibitor or ARB Therapy after Blood Pressure is at Goal and Dose is Stable, According to Baseline Values

<table>
<thead>
<tr>
<th>Baseline Value</th>
<th>SBP (mm Hg)</th>
<th>DBP (mm Hg)</th>
<th>GFR (mL/min/1.73 m²) Early GFR Decline (%)</th>
<th>Potassium (mEq/L)</th>
<th>Interval (Months)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>120-129</td>
<td>110-119</td>
<td>&gt;50</td>
<td>&lt;15</td>
<td>6-12</td>
</tr>
<tr>
<td></td>
<td>100-109</td>
<td>80-89</td>
<td>30-50</td>
<td>&lt;15</td>
<td>3-6</td>
</tr>
<tr>
<td></td>
<td>&lt;100</td>
<td>&lt;80</td>
<td>&lt;30</td>
<td>&gt;15</td>
<td>1-3</td>
</tr>
</tbody>
</table>

Delay Progression: Diabetes

- CKD – Peripheral insulin resistance, acidosis, decreased insulin excretion in uremia
- Tight control (HgbA1C < 7%) slows progression of renal disease in Type I and II
- Test for microalbuminuria at time of diagnosis and annually
- Suspect nephropathy if retinopathy is present
- Treatment cautions:
  - No Metformin if <40ml/min – Lactic acidosis
  - Sulfonylureas accumulate in renal failure
  - Insulin degraded by the kidney
  - Glitazones aggravate edema and CHF

Delay Progression: Treat Hyperlipidemia

- Meta analysis of 12 studies showed lipid lowering agents slowed GFR decline
- Monitor for Dyslipidemia (TG>150, LDL>100, HDL<40)
- High risk – Goal: LDL < 100, Non-HDL < 130
- Diet
- Statins appear safe
- Caution fibrin acid derivatives

* Fried, Orchard, Kasiske, Ki, 2001
Delay Progression: Avoidance of Nephrotoxic Substances

- NSAIDs (COX-2 inhibitors, ibuprofen) are potentially nephrotoxic.
- Avoid other nephrotoxic substances (intravenous dye, aminoglycosides, amphotericin B, cyclosporin, tacrolimus, lithium, cisplatin, gold).
- ACE inhibitors/ARBs.


Delay Progression: Lifestyle/Nutritional Restrictions

- Exercise
- Cessation of smoking (increased rate of progression of CKD)
- Sodium restriction
  - Hypertensive/nephropathy patients ≤ 2000 mg/day
- Fluid Restriction 1,000 ml/day plus output
- Potassium – Low-K diet, diuretics, treat acidosis
- Protein (controversial)
  - Microalbuminuria: 0.8 g/kg/day
  - Decreasing GFR: 0.6 g/kg/day

American Diabetes Association, 2001b.
CKD patients receiving an albumin test in the two years prior to ESRD, by age & race/ethnicity

Delay Progression: Treat Hypertension and Proteinuria

Lifestyle modifications

<table>
<thead>
<tr>
<th>Weight Loss</th>
<th>10 kg loss</th>
<th>5-20 mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>DASH Diet</td>
<td>Diet rich in fruit vegetables</td>
<td>8-14 mm Hg</td>
</tr>
<tr>
<td>Low-Sodium Diet</td>
<td>Restrict sodium intake</td>
<td>2-8 mm Hg</td>
</tr>
<tr>
<td>Physical Activity</td>
<td>30 minutes/day most days</td>
<td>4-9 mm Hg</td>
</tr>
<tr>
<td>Mod. Alcohol Consumption</td>
<td>2 drinks/day men 1 drink/day/women</td>
<td>2-4 mmHg</td>
</tr>
</tbody>
</table>

Goals of Treatment

Early Detection of CKD

Delay progression

Prevent complications

Treat co-morbidities

Prepare for RRT

Adapted from Pereira, 2000.
Treat Complications and Co-morbidities Associated With CKD

- Anemia
- Malnutrition
- Bone disease
- Metabolic acidosis

- Cardiovascular disease
- Hypertension
- Diabetes

Cumulative percentage of patients receiving hemoglobin testing in the 12 months prior to ESRD

USRDS 2008

Erythropoiesis in CKD

Adapted from Hillman, 1998.
**Anemia Starts Early in CKD and Worsens With Disease Progression**

![Graph showing prevalence of anemia at different serum creatinine levels](image)

- *Kausz, Steinberg, Neeman, & Pereira, 2000.*
- *Obrador, Ruthazer, Arora, Kausz, & Pereira, 1999.*

<table>
<thead>
<tr>
<th>Serum Creatinine Level (mg/dL)</th>
<th>Prevalence of Anemia (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 2</td>
<td>5%</td>
</tr>
<tr>
<td>2–2.9</td>
<td>17%</td>
</tr>
<tr>
<td>3–3.9</td>
<td>15%</td>
</tr>
<tr>
<td>&gt; 4</td>
<td>2%</td>
</tr>
<tr>
<td>Start of Dialysis (n = 131,484)</td>
<td>67.5%</td>
</tr>
</tbody>
</table>

**How Anemia Might Cause Cardiovascular Disease**

- ↑ Cardiac output
- Limited myocardial O₂ supply
- Decreased peripheral resistance
- Volume overload
- Left-ventricular dysfunction (eg, LVH)
- CHF

![Relative Risk Chart](image)

**Anemia Is a Mortality Multiplier**

**Medicare 5% Sample 1996-1997 2-Year Follow-up, Adjusted for Age, Gender, and Race**

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Relative Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1.5</td>
</tr>
<tr>
<td>Anemia</td>
<td>2</td>
</tr>
<tr>
<td>CKD</td>
<td>2</td>
</tr>
<tr>
<td>CCF</td>
<td>2.9</td>
</tr>
<tr>
<td>CHF</td>
<td>3.7</td>
</tr>
<tr>
<td>DM/CHF only</td>
<td>3.7</td>
</tr>
<tr>
<td>CKD</td>
<td>4</td>
</tr>
<tr>
<td>CHF/Anemia</td>
<td>7.3</td>
</tr>
</tbody>
</table>

Analysis performed by Minneapolis Medical Research Foundation.
Anemia NKF KDOQI guidelines
www.kidney.org/professionals/kdoqi/guidelines

• ↓ Hemoglobin (<11-12gm/dl)
• ↓ Hematocrit (<33-37%)
• Iron deficiency: Normocytic, normochromic
• ↓ Ferritin (<100ng/ml) [<12 absolute iron deficiency]
• ↓ % Saturation (<20%) [<16% absolute iron deficiency]

Anemia Evaluation

• Iron stores
• Folate or vitamin B deficiency
• Potential sources of blood loss (gi, menses)
• Infection/inflammation (diabetes, systemic lupus, rheumatoid arthritis)

Erythropoietin Therapy

• H&H <10gm/dl and <32
• Symptoms: SOB, fatigue, tachycardia, cold intolerance
• Subcutaneous – Weekly or bimonthly
• Monitoring
  – B/P
  – Labs
  – seizures

The American Nephrology Nurses’ Association (ANNA)
Treat Anemia

- Work up for anemia
- Treat with erythropoietin stimulating agent (ESA)
- Similar side effect profile
- Dosing varies by ESA and patient response
- Monitor BP


Cumulative percentage of patients receiving EPO/DPO in the 12 months prior to ESRD

USRDS 2008

LVH in CKD

- LVH is an independent predictor of cardiac death.
- Hypertension, anemia, and diabetes are modifiable predictors of LVH.
  - Blood pressure increase of 5 mm Hg is associated with 3% increase in LVH risk.
  - Hb decrease of 1 g/dL is associated with 6% increase in LVH risk.

Unadjusted survival probabilities in patients with or without CKD & walking disabilities (Medicare 65+yo)

Bone Disease
- Begins in stage 3
- Phosphorus retention (nl 3.5-5.5 mg/dl)
- Inadequate vitamin D conversion
- Decreased gi calcium absorption (nl 8.4-9.5 mg/dl)
- 2nd hyperparathyroidism develops (intact PTH target goals) 35-70 pg/ml stage 3; 70-110 stage 4; 150-300 stage 5

Renal Osteodystrophy
- Accurate diagnosis with bone biopsy but rarely done
- Goal to prevent high or low bone turnover dz
- Estimate when GFR <60ml/min (stage 3)
  - ↑ Intact PTH
  - ↑ Phosphorus
  - ↓ Calcium
  - Calcium X phosphorus product <55 is goal
Therapy Goals for Renal Osteodystrophy

- Prevent/control 2nd hyperparathyroidism
- Phosphorus control w/ diet and/or calcium supplements
- Active vitamin D either as calcitriol or an analog paricalcitol or doxercalciferol
- Cinacalcet alters Ca sensing PTH cells
- Calcium replacement
- Must monitor labs with therapy

Phosphorus and GFR

NKF-K/DOQI guidelines

Metabolic Acidosis

- Inadequate excretion of hydrogen and ammonium
- Inadequate production of bicarbonate
- Consequences include increased serum potassium
Acid/Base Balance

- **Endogenous H+ Production**: 70 mEq/day
- **Renal NH₄⁺ Excretion**: 40 mEq/day
- **Renal T.A. Excretion**: 30 mEq/day
- **Renal Net Acid Excretion**: 70 mEq/day

Normal Acid/Base Balance: [HCO₃⁻] = 24 mEq/L

TA=Treatable acid

Consequences of Metabolic Acidosis

- Abnormal renal handling of ions:
  - ↓ tubular-phosphate reabsorption
  - ↑ filtered load of calcium and phosphate
  - ↓ tubular-calcium reabsorption
- Increased resorption of bone
- Increased muscle catabolism

Treatment of Metabolic Acidosis in CKD

- **Goal**:
  - Serum HCO₃⁻ > 20 mEq/L
  - pH > 7.35
- **Agents**
  - Sodium bicarbonate tablets
    - (650 mg = ~8 mEq HCO₃⁻)
  - Sodium citrate (Shohl’s solution)
- **Dose of HCO₃⁻**:
  - 1.0-1.5 mEq/kg/day
  - Dependent upon initial serum HCO₃⁻ and degree of renal insufficiency
Adjusted hazard ratio of mortality: Medicare patients

Medicare-only, point prevalent general Medicare patients entering Medicare before January 1, 2004, after age 65 or older on December 31. Patients enrolled in an HMO, with Medicare as secondary payor, diagnosed with ESRD during the year, or enrolled in Medicaid during the period are excluded. CKD, diabetes, & CHF defined during each period; comorbidity groups are mutually exclusive; patients are followed one year from January 1, to December 31, of the next year.

Cardiovascular Disease: Prevalence in CKD

Framingham Heart Study

% Male Patients

CVD CHD CHF LVH

Normal SCr (n=2591)

Elevated SCr (1.5–3.0 mg/dL, n=246)

Predictors of mortality in the Medicare-only population

Figure 3.17 (Volume 1)

Medicare-only, point prevalent general Medicare patients entering Medicare before January 1, 2004, after age 65 or older on December 31. Patients enrolled in an HMO, with Medicare as secondary payor, diagnosed with ESRD during the year, or enrolled in Medicaid during the period are excluded. CKD, diabetes, & CHF defined during each period; comorbidity groups are mutually exclusive; patients are followed one year from January 1, to December 31, of the next year.
Cardiovascular Mortality

Risk Factors for Cardiac Disease in CKD

Traditional
- Hypertension
- Diabetes
- Age
- Smoking
- Dyslipidemia
- Obesity
- Inactivity
- GFR < 60 ml/min
- Family history

Nontraditional
- Anemia
- Inflammation
- Oxidative stress
- Hyperhomocysteinemia
- Ca/Phos metabolism
- Fluid overload
- Hypoalbumin
- Uremic toxins

Likelihood of death vs. ESRD in the Medicare population

The American Nephrology Nurses’ Association (ANNA)
Why Treat CKD and prevent ESRD?
Survival in ESRD

ESRD patients lose approximately 80% of remaining years that the general population is expected to live.

Cost of ESRD Care – $20 Billion

Expected Years Remaining*

* Based on adult, age 59 years

Definable Target Treatments

- Blood pressure < 130/80
- Proteinuria < 500mg – 1gm/day
- Anemia Hgb 11-12
- Ca, Phosphate, iPTH – Normal values
- Nutrition – HCO3 = Normal
- Sequential measurement of kidney function
- Predict progression
- Education and preparation

Benefits of Early Intervention in the Management of CKD

- Delayed progression of CKD
- Decreased complications and co-morbid conditions
- Improved dialysis outcomes
- Better educated and prepared patients

Obrador et al., 1999.
USRDS, 1999.


References


References


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References


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References


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