Cystatin C: Its Utility as an Alternative for Creatine-based eGFR

Panelists
Josef Coresh, MD, PhD
Amy Karger, MD, PhD
Michael Shlipak, MD, MPH
Michelle Estrella, MD, MHS

Moderator
Silas Norman, MD, MPH

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  • We will answer questions out loud during the Q&A portion of the presentation.
Silas Norman, MD, MPH

University of Michigan

- Transplant nephrologist and Co-Medical Director of Kidney and Pancreas Transplantation
- Research is focused on reducing health disparities and evaluation of medical frailty
- Member of AKF Board of Trustees and incoming Vice-Chair
Learning Objectives

• Demonstrate benefits of cystatin C related to diagnosis, risk classification and racial bias
• Describe the epidemiological argument for measuring cystatin C
• Examine illustrative case examples
• Identify practical barriers to ordering the cystatin C test
Josef Coresh, MD, PhD
Johns Hopkins University

- International expert in kidney and cardiovascular disease epidemiology
- Helped to develop the chronic kidney disease definition, staging and estimated kidney function equation
- Co-leads the CKD Prognosis Consortium (CKD-PC)
Importance of Estimated GFR
Background: Importance of Estimated GFR

- Included participants
- Not included

www.ckdpcrisk.org

2002

2013
Outline

• CKD definition and staging
• GFR estimation
• Cystatin C complements serum creatinine
CKD Staging (KDIGO 2012)

- Cause
- GFR (<60 ml/min/1.73m²)
- Albuminuria (> 30 mg/g)

<table>
<thead>
<tr>
<th>Albuminuria Categories, Description and Range</th>
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<tr>
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|     | A2                                                      |
|     | <30 mg/g                                                |
|     | 30-299 mg/g                                             |
|     | ≥300 mg/g                                               |

|     | A3                                                      |
|     | <3 mg/mmol                                              |
|     | 3-29 mg/mmol                                            |
|     | ≥30 mg/mmol                                             |

<table>
<thead>
<tr>
<th>GFR Categories, Description and Range (mL/min/1.73 m²)</th>
</tr>
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<tbody>
<tr>
<td>G1</td>
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<tr>
<td>G2</td>
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<tr>
<td>G3a</td>
</tr>
<tr>
<td>G3b</td>
</tr>
<tr>
<td>G4</td>
</tr>
<tr>
<td>G5</td>
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</table>
CKD-PC Risk Summary:

Adjusted Hazard ratios
Meta-Analysis of GP Cohorts

Very Strong Associations with eGFR and ACR (<30, 30-299, 300+ mg/g)


KDIGO Conference Report.
CKD Staging by eGFR and ACR

• Very strong risk relationships across:
  • Many outcomes:
    • Concurrent complications¹ – anemia, acidosis, functional status …
    • Future risk – CKD progression², acute kidney injury³, CVD⁴, mortality⁴, hospitalization
  • Many settings
    • ~80 cohorts, >40 countries, >1 Million participants
    • Subgroups by diabetes⁵, hypertension⁶, sex⁷, age⁸ (larger attributable risk despite smaller relative risk for mortality), race⁹
  • Risk prediction (ckdpcrisk.org – 7 equations including KFRE)
  • Clinical trial surrogate outcomes (40% eGFR decline, eGFR slopes, ACR change)¹⁰,¹¹
  • Different measurement techniques
    • Albuminuria (ACR) preferred but proteinuria (PCR) and dipstick show risk¹²
    • eGFR – CKD-EPI preferred but MDRD and other equations work²
    • eGFRcys shows stronger risk gradients for CVD and mortality but creatinine works¹³
    • [Strive to improve but don’t let seeking perfection be the enemy of the good]
GFR Estimation – Powerful Despite Imperfections

- Serum Creatinine vs. mGFR in the MDRD Study
- Lower GFR → higher serum creatinine (true for all filtration markers)
- Non-filtration influence → Different serum creatinine for the same mGFR
  - mGFR=60 → higher creatinine for men and black participants
- eGFR equations use regression to adjust for the average effect of non-filtration factors using surrogates
  - Age, sex and race for average creatinine generation & tubular secretion
# Performance of CKD-EPI eGFR Equations Compared to measured GFR (mGFR) in the Validation Dataset (N=1119)

<table>
<thead>
<tr>
<th>Equations</th>
<th>Bias (P30)</th>
<th>Precision (IQR)</th>
<th>Accuracy (P30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatinine (CKD-EPI)</td>
<td>3.7</td>
<td>15.4</td>
<td>87.2</td>
</tr>
<tr>
<td>Cystatin C</td>
<td>3.4</td>
<td>16.4</td>
<td>85.9</td>
</tr>
<tr>
<td>Creatinine-cystatin C</td>
<td>3.9</td>
<td>13.4*</td>
<td>91.5*</td>
</tr>
<tr>
<td>Average of creatinine and cystatin C</td>
<td>3.5</td>
<td>13.9</td>
<td>91.8</td>
</tr>
</tbody>
</table>

*\(P \leq 0.001\) compared to creatinine alone or cystatin C alone

- Bias – small
- Precision & Accuracy – better with 2 markers than either marker alone
- Cystatin provides a useful alternative when serum creatinine is thought to be unreliable (altered muscle mass)
eGFR Equations (ASR vs. NB) & Risk

Kidney Failure Replacement Therapy (KFRT) Hazard Ratio Adjusted for Age & Sex

- eGFRcr (ASR-NB)
  - No change for non-black individuals (orange)
  - “Dropping” the race term in CKD-EPI - lowers eGFR by 16% for black individuals compared to current equation (green)
- ~1,000-fold relative hazard gradient for eGFR and KFRT
  - Hugely informative
  - For all equations
eGFR Equations (ASR vs. NB) & Risk

Kidney Failure Replacement Therapy (KFRT) Hazard Ratio Adjusted for Age & Sex

- ~1,000-fold relative hazard gradient for eGFR and KFRT
  - Hugely informative
  - For all equations
- eGFRcr (ASR-NB)
  - No change for non-black individuals
  - Lowers eGFR by 16% for black individuals compared to current equation
  - Mathematically “explains” much of the excess risk by assigning black individuals a lower eGFR (based on no data)
eGFRcys Equations (ASR vs. AS) & Risk

Kidney Failure Replacement Therapy (KFRT) Hazard Ratio Adjusted for Age & Sex

- eGFRcys -- Didn’t need a race term (NEJM 2012)

- Cystatin may be biased by other things but not race (~5% vs. ~16% for Cr)

- Substantial race inequality in risk is found with eGFRcys
  - Concern about any eGFRcr measure of disparities but particularly NB which “explains away risk disparities” as low eGFR
Race Term in eGFR Can Influence Measures of Population Health Disparities KFRT & CKD-EPI eGFRcr, cys, cr-cys Risk Example

- eGFRcr-ASR vs eGFRcr-NB
- eGFRcys
- eGFRcr-cys vs. eGFRcr-cys-NB

eGFRcr-cys is more robust to the way race is modeled
Summary

- GFR is a powerful measure of kidney function
  - Other functions exist but CKD staging relies on eGFR and ACR
- CKD staging is a powerful risk factor for many outcomes
  - Despite limitations of eGFR and ACR
  - The fundamentals work!
  - We should improve: awareness of CKD, albuminuria measurement rates, treatment of CKD
  - Better GFR estimation and new risk markers are helpful too
- Cystatin C provides a useful complement to creatinine
  - KDIGO recommends
    - Using cystatin C when serum creatinine is not reliable
    - Higher precision in eGFR is needed – average eGFRcr and eGFRcys
  - Stronger risk information about CVD
  - Less sensitive to changes in muscle mass, diet or ethnic origin
Acknowledgements:

- CKD-EPI Collaboration (eGFR)
- CKD Prognosis Consortium (NKF & KDIGO)
  - Steering committee: J Coresh (Chair, co-PI), M Grams (co-PI), K Matsushita, S Ballew, A Levey, R Gansevoort, K Polkinghorne, E Schaffner, O Gutierrez, T Konta
  - Analysis leaders: Y Sang, A Surapaneni
- Johns Hopkins co-investigators & staff

Thank you!
Q & A

If you have a question for Dr. Coresh:
Please type it into the Q&A box in your control panel.
The Epidemiology Argument for Cystatin C
Michael Shlipak, MD, MPH
San Francisco VA Health Care System and University of California - San Francisco

• Co-Founder and Scientific Director of the Kidney Health Research Center at UCSF

• Research missions include the prevention of chronic kidney disease and its complications, and the development of investigators in clinical research

• Chaired the 2019 KDIGO Controversies Conference on CKD Detection, Risk Stratification and Treatment
How can we compare creatinine and cystatin C in the general population of “real world” patients?

• Nephrology research overly influenced by measured GFR

• GFR is not a perfect Gold Standard – imprecise, rarely available clinically, and not measured in very sick or hospitalized patients

• CKD elevates risk for: death, CVD, heart failure

• Our strategy – compare associations of creatinine vs cystatin C with clinical outcomes relevant to CKD
Chronic Kidney Disease Prognosis Consortium Meta-Analysis: eGFR by creatinine vs. cystatin C

16 cohorts; 90,000 participants

Cystatin C Can Impact Clinical Decision Making by Reclassifying CKD Stages

Adjusted for age, gender, race, smoking, systolic blood pressure, total cholesterol, diabetes, history of cardiovascular disease, body mass index, and albuminuria.

GFR Staging Impacts Medication Eligibility and Dosing
Meta-Analysis had Direct Impact on 2012 KDIGO International CKD Guidelines

• Confirm CKD with Cystatin C if based only on creatinine:
  • If cystatin C eGFR <60: patient has CKD
  • If cystatin C eGFR >60: patient does NOT have CKD

• To dose potentially toxic medications, do not rely on creatinine only:
  • Use cystatin C or direct measures of GFR

Accurate Kidney Function Estimation is Critical for Medication Dosing

• ~2/3 of medications are primarily eliminated by the kidneys
• ~23% of medications in hospital are potentially nephrotoxic.
• ~¼ of hospitalized AKI related to medication toxicity
• Accurate prediction of drug clearance and medication dosage - critically important to patient safety and treatment efficacy.
Does Cystatin C change Metformin Eligibility?

- Cross-sectional study – adding cystatin C to routine labs
- Participants: Veterans with DM2 in primary care (n=550)
- Findings: 20% were re-classified to new eGFR stage
  - eGFRcr 30-45: 40% <30 by cystatin C

Conclusion: Cystatin C identifies persons who should not be on metformin

Systematic Review on Medication Dosing Comparing Cystatin C vs. Creatinine

• Reviewed 28 articles on 16 different medications (N= 3,455 patients)
• Outcomes of drug clearance, target blood level treatment, and toxicity
• Overall, cystatin C-based eGFR was more predictive of drug levels and drug clearance than creatinine-eGFR

Barreto et al; Mayo Clinic Proceedings 2019
Clinical Trial Demonstrates that Vancomycin Dosing Improved by Cystatin C Algorithm

• Mayo Clinic – Critical Care setting

• Q.I. Intervention for Vanc dosing:
  o 22 months of creatinine only (N=264)
  o 18 months of creatinine/cystatin C combined eGFR (N=135)

• Therapeutic trough concentrations improved: 50% vs. 28%
  o OR=2.79; 1.76-4.44

• Next step – demonstrate impact of cystatin C-guided dosing and clinical outcomes.”

How Do I Interpret 2 Different GFR Estimates?
What if eGFR_{cys} \neq eGFR_{cr} ?

• If the 2 measures always agreed, then would be pointless to use cystatin C
• Our team has developed (+/-) 15 mL/min rule for eGFR to simplify
• If both eGFR’s are within 15 ml/min – we average them, which approximates the combined eGFR_{cr}/cys
• If \geq 15 point eGFR difference – usually the cystatin C is more accurate
• Younger populations – the cystatin C GFR often is higher (Cr biased in muscular persons)
• Older or chronically ill – cystatin C eGFR usually lower (Cr biased by frailty)
eGFR Diff > 15mL/min in older adults: The Systolic Blood Pressure Intervention Trial (SPRINT)

AJKD July 2020; Potok et al

Cystatin C eGFR better

Higher risk for: frailty, falls, CVD, and mortality

Cystatin C eGFR worse

13%

15%

Ref.

eGFR\text{cys} \ll eGFR\text{cr} \rightarrow \text{patient likely to be frail}
When Should you Suspect that the Creatinine is too Low?

• Hospitalized patients
  – Low albumin (<3.5 mg/dL), low hemoglobin, high BUN, proteinuria
  – Duration of hospitalization, as Cr production declines steadily

• Ambulatory patients
  – Likely to have CKD – diabetes, HTN, CVD, heart failure
  – Likely to under-produce Cr – HIV, cirrhosis, frail elders, malignancy

• Measuring Cystatin C eGFR – as a 2nd opinion – can have a substantial impact on patient safety
Q & A

If you have a question for Dr. Shlipak:
Please type it into the Q&A box in your control panel.
Clinical Case Examples
Michelle Estrella, MD, MHS
San Francisco VA Health Care System and University of California - San Francisco

• Renal Section Chief at the San Francisco VA Health Care System

• Research encompasses identification of markers of kidney injury that will lead to earlier detection and management of kidney disease and development of strategies that leverage care coordination and health technology to improve clinical outcomes in patients at risk and with kidney disease.

• Serves as the Executive Director of the Kidney Health Research Collaborative
Two common clinical questions regarding cystatin C

• In which clinic patients should I order cystatin C?
• How do I reconcile differences in eGFR between creatinine and cystatin C?
**CASE 1**

80 yo with multiple morbidities with hyperkalemia and eGFRSCr ≥60

- Significant history:
  - “Persistent hyperkalemia despite holding ACE inhibitor without other evidence of kidney dysfunction”
  - Poor exercise tolerance in the setting of deconditioning

**Most recent labs:**

<table>
<thead>
<tr>
<th>eGFR_scR</th>
<th>SCr</th>
<th>A1c</th>
</tr>
</thead>
<tbody>
<tr>
<td>138</td>
<td>107</td>
<td>5.9</td>
</tr>
<tr>
<td>20</td>
<td>22</td>
<td>1.11</td>
</tr>
<tr>
<td>105</td>
<td>7.9</td>
<td>11.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>160</td>
</tr>
</tbody>
</table>

**eGFR_scR trend:** 64 → 57 → 64 mL/min per 1.73 m²

A1c = 6.1%
80 yo with multiple morbidities with hyperkalemia and eGFRSCr ≥60

Which of the following is most correct regarding this patient’s kidney tests?

A. This patient does not have CKD since his eGFR has generally been ≥60 mL/min per 1.73 m².

B. Cystatin C would help determine whether the patient has CKD.

C. The patient has well-controlled diabetes so a microalbumin is not indicated.

D. This patient is at low risk of progression because his eGFR_{SCr} is ≥60 mL/min per 1.73 m².
A. This patient does not have CKD since his eGFR has generally been $\geq 60$ mL/min per 1.73 m$^2$.

Lab trends over the past year:

- **Serum K+ range:** $4.9 - 5.9$ mEq/L
- **Serum bicarb range:** $20 - 23$ mEq/L
- **Hemoglobin range:** $11 - 12$ g/dL

These lab results are consistent with moderately advanced CKD.
80 yo with multiple morbidities with hyperkalemia and eGFRSCr ≥60

Trends in eGFR

<table>
<thead>
<tr>
<th>Time</th>
<th>eGFRScr</th>
<th>eGFRCysc</th>
</tr>
</thead>
<tbody>
<tr>
<td>DEC-19</td>
<td>64</td>
<td>40</td>
</tr>
<tr>
<td>JUN-20</td>
<td>64</td>
<td>40</td>
</tr>
<tr>
<td>JUL-20</td>
<td>63</td>
<td>43</td>
</tr>
<tr>
<td>JAN-21</td>
<td>51</td>
<td>34</td>
</tr>
</tbody>
</table>

eGFR, mL/min per 1.73 m²
Cystatin C can be used to confirm/ detect CKD

2012 CKD Guidelines

Confirm CKD with CysC if based only on SCr:

• If eGFR < CYSC < 60: patient has CKD
• If eGFR CYSC ≥60: patient does not have CKD

2019 CKD Screening Controversies Conference

Conclusion 5. Accurate GFR estimation includes both SCr and CysC for initial diagnosis and staging.

Kidney Int. 2013.

C. The patient has well-controlled diabetes so a microalbumin is not indicated.

D. This patient is at low risk of progression because his eGFR_{Scr} is ≥60 mL/min per 1.73 m².

2012 CKD Guidelines

CKD is classified based on cause, GFR category and albuminuria category

2019 CKD Screening Controversies Conference

Conclusion 4. CKD screening and risk stratification must consist of a dual assessment eGFR and albuminuria.
UACR trends over the past year:

88 → 100 → 293 mg/g
60 yo referred for discrepant eGFRScr and eGFRCysC

- Significant history:
  - Described in notes as having “failure to thrive”
  - History of alcohol use disorder and chronic hepatitis C infection
- Exam:
  - Ambulated with 4-wheel walker
  - Weight 118 pounds

**Most recent labs:**

<table>
<thead>
<tr>
<th>eGFRScr = 37</th>
<th>eGFRCysC = 18</th>
</tr>
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<tbody>
<tr>
<td>135</td>
<td>102</td>
</tr>
<tr>
<td>25</td>
<td>19</td>
</tr>
<tr>
<td>100</td>
<td>19</td>
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Δ = 19
Which of the following is most correct regarding cystatin C?

A. Cystatin C always reclassifies patients into lower CKD stages.
B. Unlike SCr levels, cystatin C levels are unaffected by medications.
C. Unlike SCr, cystatin C is not influenced by muscle mass.
D. Cystatin C yields no additional prognostic information beyond SCr.
A. Cystatin C always reclassifies patients into lower CKD stages.

CysC reclassifies many individuals to either more or less advanced CKD stages.

Adjusted for age, gender, race, smoking, systolic blood pressure, total cholesterol, diabetes, history of cardiovascular disease, body mass index, and albuminuria.

Reclassifications largely stem from the effect of muscle mass and activity on creatinine.

Cystatin C is not biased by muscle mass or activity.
Clinical contexts in which CysC may yield more accurate estimates of GFR

**SCr GENERATION IS LOW**
- Elderly
- Inactivity
- Malignancy
- Veggie diet
- HIV
- Cirrhosis

**SCr GENERATION IS HIGH**
- Weight-lifting
- Meat diet
- Protein supplements

**Meds that elevate SCr**
- Trimethoprim
- Fenofibrate
- H2-blockers
- Dolutegravir/Cobicistat
- Tyrosine kinase inhibitors
60 yo referred for discrepant eGFRSCr and eGFRCysC

B. Unlike SCr levels, cystatin C levels are unaffected by medications.

EXTRA-RENAI FACTORS THAT AFFECT CYSTATIN C

**HIGHER CYSC**
- Body mass index
- Hyperthyroidism
- Inflammation

**LOWER CYSC**
- Hypothyroidism
- Corticosteroids

Among those with eGFR$_{Cr}$ ≥60

D. Cystatin C yields no additional prognostic information beyond SCr.

Association of CKD Definition with All-Cause Mortality

Among those with eGFR$_{Cr}$ <60

On which eGFR should we base our clinical decisions?

- Make clinical decisions based on eGFR\textsubscript{CysC}
- Clinical picture points to an issue of non-renal factors affecting SCr level

When clinical picture is less clear, consider combined SCr & CysC CKD-Epi equation

If eGFR\textsuperscript{SCR} & eGFR\textsuperscript{CysC} are largely discrepant, consider measured GFR study (e.g. iothalamate or iohexol clearance)
Take-Home Points

• Cystatin C has fewer non-renal determinants than serum creatinine.
• Consider checking cystatin C in patients with:
  • eGFRSCr stage G3a/b (eGFR 30-59) to confirm or “un-confirm” CKD
  • Conditions that may render SCr insensitive/ inaccurate for detecting CKD
  • Significant risk factors for CKD (e.g. diabetes, hypertension, CVD, heart failure)
  • A “triple marker” approach with SCr, CysC and albuminuria is the most informative for CKD detection and risk stratification.
Q & A

If you have a question for Dr. Estrella:
Please type it into the Q&A box in your control panel.
Barriers to widespread use of cystatin C and how to overcome them
Amy Karger, MD, PhD
University of Minnesota

- Recognized leader in the field of laboratory medicine, currently serving in leadership roles with both the College of American Pathologists and the American Association of Clinical Chemistry

- Serves as the director of the Central Laboratory for the CKD-EPI research group

- Expertise on the measurement of filtration markers, including creatinine, cystatin C, beta-2 microglobulin, and beta-trace protein
Evolution of cystatin C assay availability

- First cystatin C assay was FDA-approved in 2001
- Initial cystatin C assays were run on specialized immunoassay instruments
  - Immunoassay instruments are typically only purchased by larger laboratories or reference laboratories
- In recent years, instrument manufacturers have made cystatin C available on traditional clinical chemistry instrumentation
  - Clinical chemistry instruments are more widely available in small and large laboratories since they run “routine” tests that require rapid turnaround time, i.e. CMP
- Not yet available on commonly used point-of-care instruments for bedside, rapid testing or small tabletop instrumentation used in small physician office laboratories
Current status of cystatin C testing options

- Five major clinical chemistry instrument manufacturers that represent the majority of the chemistry instrument market in the US
  - 2 of 5 have their own FDA-approved cystatin C assays available for use on small and large laboratory instruments
- Additionally there are two diagnostic companies that manufacture reagents for FDA-approved cystatin C assays
  - These assays can be run on the other 3 major clinical chemistry instruments in an “open channel” configuration
- Therefore running cystatin C as an in-house method should be an option for the majority of clinic and hospital laboratories with clinical chemistry instruments
Current status of cystatin C assay standardization

• Standardization of cystatin C methods is critical for establishing comparable results across methods, which allows for the accurate use of a single GFR estimating equation across different methods
  • Achieved through establishing traceability of methods to reference measurement procedures and/or reference materials
• Certified reference material (ERM-DA471/IFCC) available since 2010
  • Allows manufacturers to calibrate their methods with the same reference material, to facilitate standardization
• Currently no certified reference method for cystatin C
• All major FDA-approved cystatin C manufacturers now have methods traceable to the reference material
  • Last manufacturer established FDA-approved traceability in 2018
    • Users of older instrumentation still are relying on non-traceable reagents
Improvement in between-method agreement with standardization

Data provided by the College of American Pathologists
Current barriers to more widespread cystatin C testing

- Despite availability of standardized assays on all commonly used clinical chemistry platforms, a recent CAP survey of laboratories indicated that only 7% of laboratories offer cystatin C testing in-house, with 93% sending testing out to a reference laboratory.

- Low test utilization is a financial disincentive to bringing testing in-house:
  - Fixed one-time and continuous costs associated with bringing and maintaining an assay in-house
  - One-time costs include building IT infrastructure, method verification studies
  - Continuous costs include reagents, daily QC, requirements for calibration verification and proficiency testing
  - If test volume is low, reagents may expire resulting in waste

- Slow turnaround time relative to creatinine:
  - With the majority of laboratories sending testing out to a reference laboratory, slower turnaround time for cystatin C (vs. creatinine) prevents results being concurrently available

- Current guidelines recommend use of cystatin C only in very limited circumstances:
  - 2012 KDIGO guidelines only recommend cystatin C use for confirmatory testing "in specific circumstances when eGFR based on serum creatinine is less accurate"
Current barriers to more widespread cystatin C testing

• Reimbursement and cost to patient is a concern

• Example of direct cost for testing within our health system:
  • Creatinine: $2.50
  • Cystatin C: $10.60

• Medicare reimbursement rates:
  • Creatinine: $5.12
  • Cystatin C: $18.52

• Unclear how government and non-government third party payers would reimburse if used more routinely, particularly if performed concurrently with creatinine
  • One cystatin C assay manufacturer had plans to include cystatin C on every renal panel, but the concept was rejected due to feedback that it would not be reimbursed
Steps to expand access and use of cystatin C

- Update clinical practice guidelines to provide broadened rationale for use of cystatin C
  - Important step to increase testing volumes and justify reimbursement
- Nephrologists should proactively engage their clinical laboratory directors about options for bringing testing in-house
  - Will result in assay turnaround time comparable to current rapid turnaround time for creatinine results
- In concert, nephrologists should work with clinical labs to educate primary care and other providers within their affiliated clinics and health system about how to use cystatin C, to encourage more widespread use
Summary

• Standardized cystatin C assays are now available on the most commonly used clinical chemistry instrument platforms found in clinic and hospital laboratories.

• Primary barriers to more widespread use include cost/reimbursement concerns, and that the majority of cystatin C testing is currently performed in reference laboratories which impacts result turnaround time.

• Nephrologists can play a key role in advocating for updated clinical practice guidelines broadening rationale for cystatin C use, and can advocate for in-house implementation of cystatin C testing within their own health systems.
Q & A

If you have a question for Dr. Karger or any of the speakers: Please type it into the Q&A box in your control panel.
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