



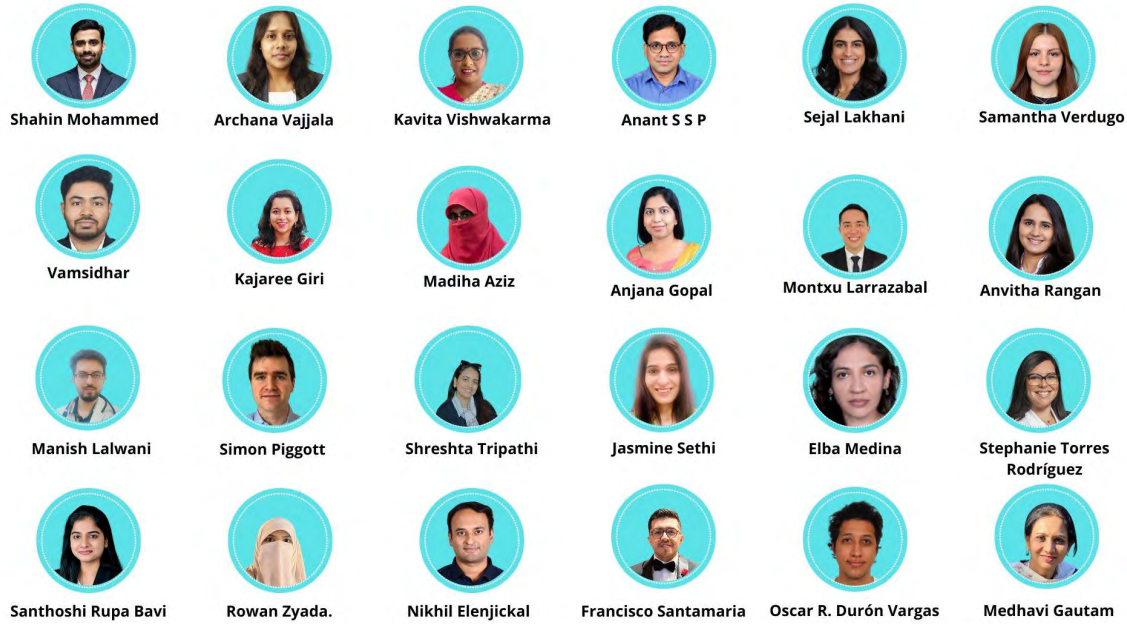
KEY TAKEAWAYS FROM THE KDIGO 2024 CLINICAL PRACTICE GUIDELINE FOR THE EVALUATION AND MANAGEMENT OF CKD

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Medhavi Gautam



**TOP 10 TAKEAWAYS ON
EVALUATION OF PEOPLE WITH
OR AT RISK OF CKD**

Top 10 Takeaways for Nephrologists on Evaluation of People with or at Risk of CKD

1

CKD definition

Abnormalities of kidney structure or function for more than 3 months. Classified according to **CGA** to establish severity.

Cause
GFR
ACR

6

Accuracy and reliability

Understand the variability and limitations of GFR and urine albumin when determining whether a change is a true change.



2

Distinguish between AKD and CKD

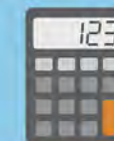
Chronicity can be established using past GFRs, past albuminuria, imaging, pathological findings of fibrosis, medical history, and repeat measurements within and beyond 3 months.



7

Use a validated GFR estimating equation

Use a validated GFR estimating equation to derive GFR from serum filtration markers and use the same equation within geographical regions.



3

CKD care across the lifespan

Use a personalized approach to diagnosis, risk assessment, and management that considers age, sex, and gender.



8

Point-of-care tests (POCT)

Use quality-assured POCT for creatinine and urine albumin measurement where access to a laboratory is limited.



4

Diagnosis of CKD in older adults

Epidemiological population data support retaining the threshold GFR of 60 ml/min/1.73m², given the increasing relative risk of adverse outcomes below this threshold.



9

Use validated risk assessment tools

Use validated risk assessment tools to aid in decision-making and timing of multidisciplinary care. For cardiac events and mortality risk, use tools that included people with CKD.



5

Improving accuracy of GFR assessment

Estimating GFR from a combination of creatinine and cystatin C (eGFR_{cr-cys}) improves accuracy and strengthens risk relationships.

Cys-C

10

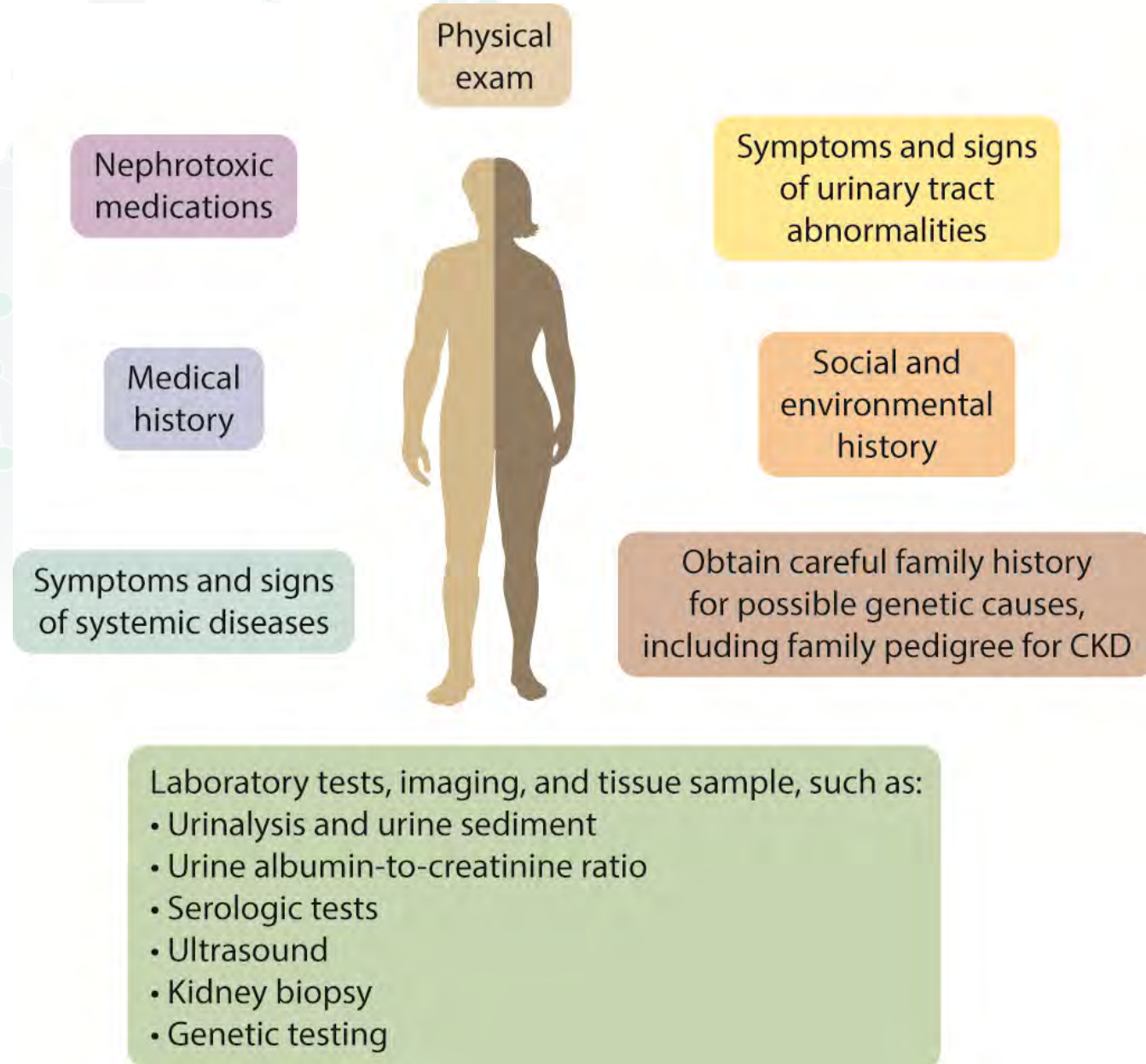
Timing assessment and reevaluation

Use validated risk prediction tools and clinical evaluation when determining timing of follow up and reassessment.



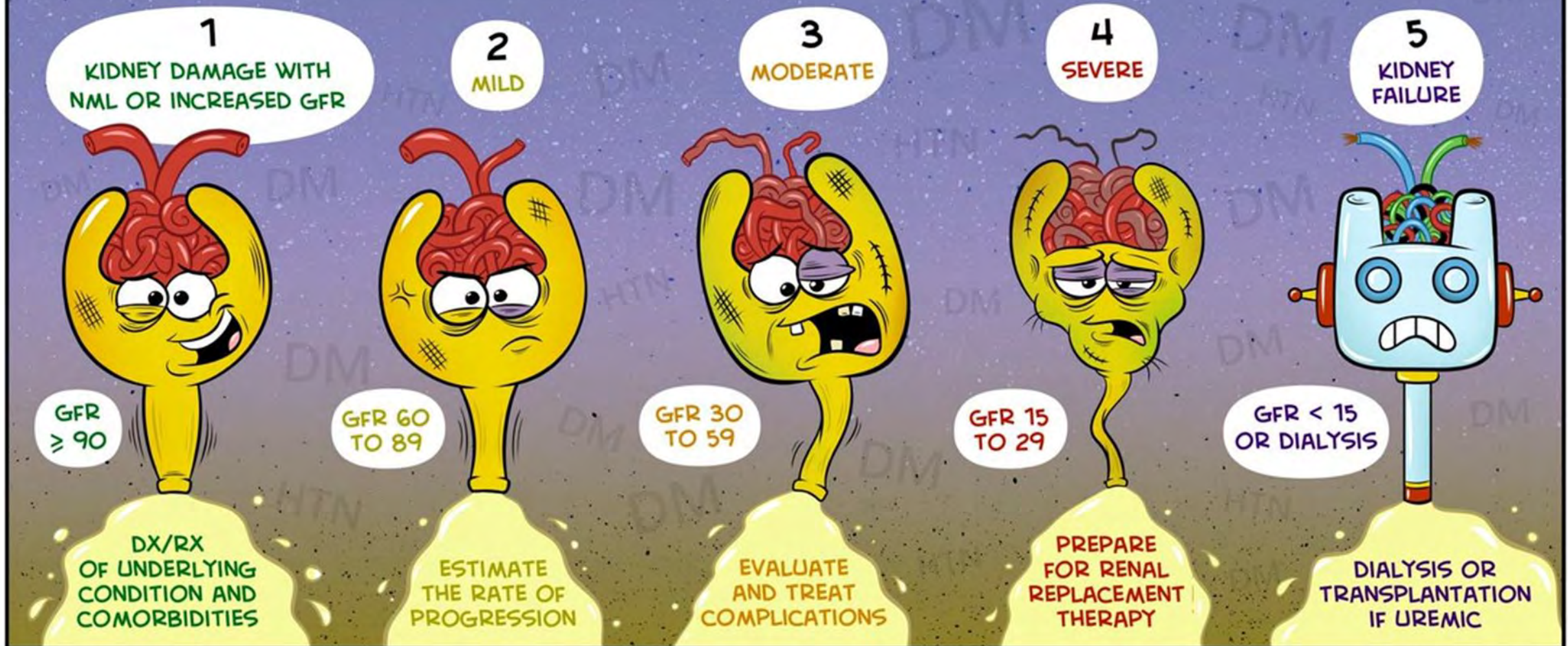
EVALUATION – CKD DEFINITION

CKD is defined as abnormalities of kidney structure or function, present for >3 months, with implications for health.

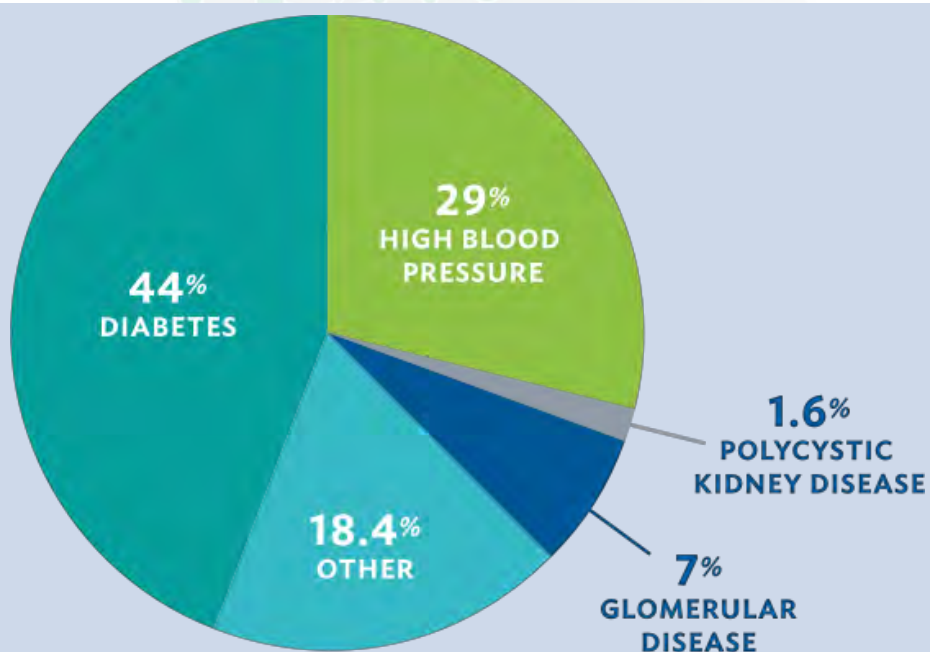


STAGES OF CHRONIC KIDNEY DISEASE

GFR: mL/MIN/1.73M²



Cause (s) of CKD



KDIGO: Prognosis of CKD by GFR and albuminuria categories

				Persistent albuminuria categories		
				Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–300 mg/g 3–30 mg/mmol	>300 mg/g >30 mg/mmol
GFR categories (ml/min/1.73 m ²) Description and range	G1	Normal or high	≥90	Green	Yellow	Orange
	G2	Mildly decreased	60–89	Green	Yellow	Orange
	G3a	Mildly to moderately decreased	45–59	Yellow	Orange	Red
	G3b	Moderately to severely decreased	30–44	Orange	Red	Red
	G4	Severely decreased	15–29	Red	Red	Red
	G5	Kidney failure	<15	Red	Red	Red

Green: low risk (if no other markers of kidney disease, no CKD); Yellow: moderately increased risk; Orange: high risk; Red: very high risk. GFR, glomerular filtration rate.

EVALUATION – CKD DEFINITION

CKD is defined as abnormalities of kidney structure or function, present for >3 months, with implications for health.

The definition includes many different markers of kidney damage, not just decreased GFR and ACR and the cause of CKD should be actively sought.

CKD is classified according to **C**ause, **G**FR, and **A**CR to establish severity and guide the type and timing of interventions.

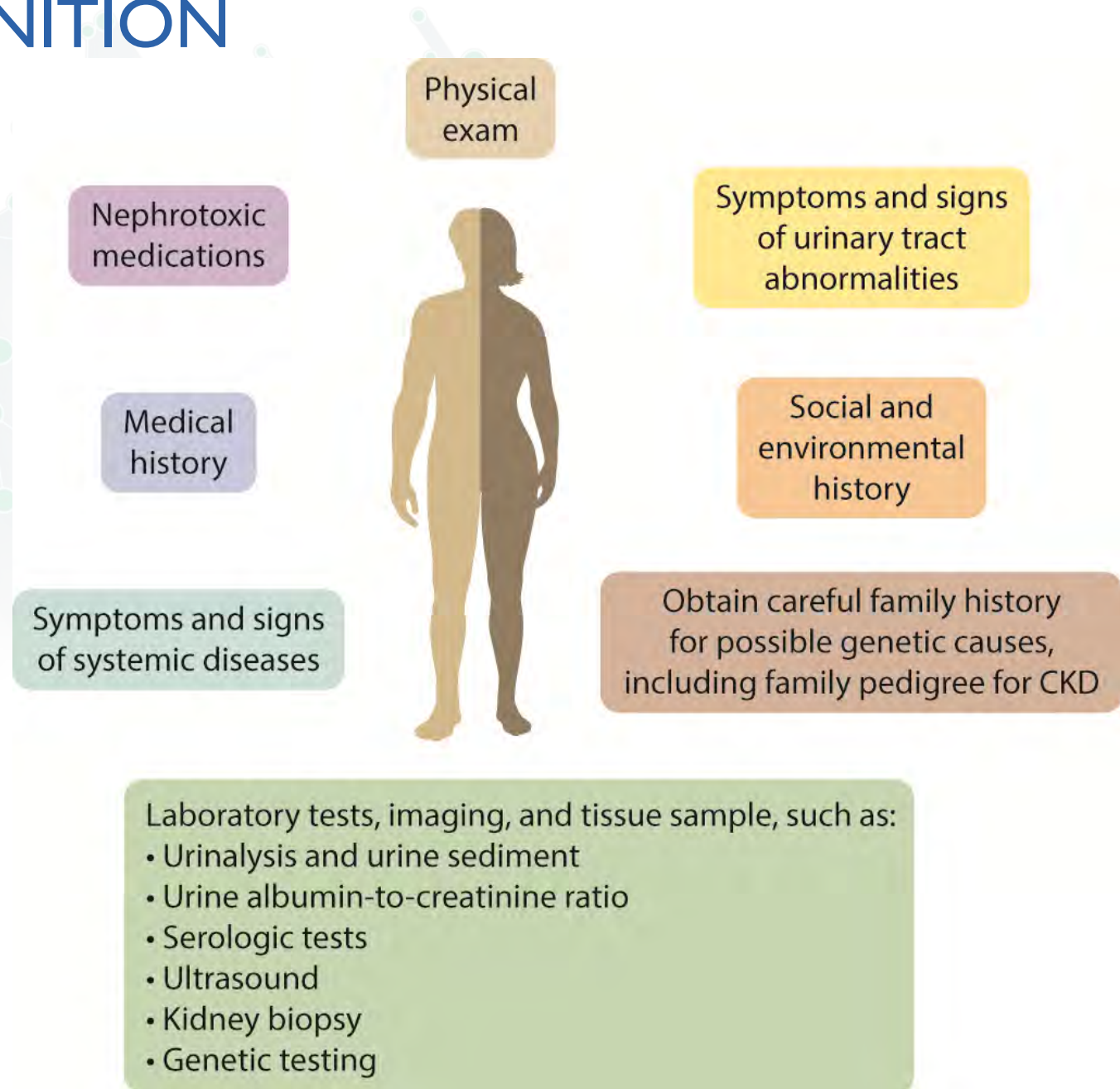















Table 6: Guidance for the Selection of Additional Test for Evaluation of Cause

Test category	Examples	Comment or key references
 <p>Imaging</p>	 <p>Ultrasound, intravenous urography, CT kidneys ureters bladder, nuclear medicine studies, MRI</p>	 <p>Assess kidney structure (i.e., kidney shape, size, symmetry, and evidence of obstruction) for cystic disease and reflux disease. Evolving role of additional technologies (eg, 3D ultrasound).</p>
 <p>Kidney biopsy</p>	 <p>Ultrasound-guided percutaneous</p>	 <p>Examined by light microscopy, immunofluorescence, electron microscopy, and molecular diagnostics (in some situations), help in precise diagnosis, treatment planning, disease activity assessment, and treatment response prediction, including genetic disease evaluation.</p>
 <p>Laboratory tests: serologic, urine tests</p>	 <p>Chemistry including acid-base and electrolytes, serologic tests such as anti-PLA2R, ANCA, anti-GBM antibodies</p> <p>Serum-free light chains, serum, and urine protein electrophoresis/immunofixation</p> <p>Urinalysis and urine sediment examination</p>	 <p>KDIGO 2021 Clinical Practice Guideline for Management of Glomerular Diseases.</p> <p>Light chains' emerging role in kidney disease in absence of multiple myeloma → MGRS</p> <p>Persistent hematuria or albuminuria crucial for diagnosis</p>
 <p>Genetic testing</p>	 <p>APOL1, COL4A3, COL4A4, COL4A5, NPHS1, UMOD, HNF1B, PKD1, PKD2</p>	 <p>Evolving as a diagnostic tool.</p> <p>Genetic causes >10% of CKD, regardless of family history. Increased utilization is expected.</p>
 <p>Practice pearl</p>	<p>Use diagnostic tests based on available resources & pretest probability informed by clinical presentation</p>	

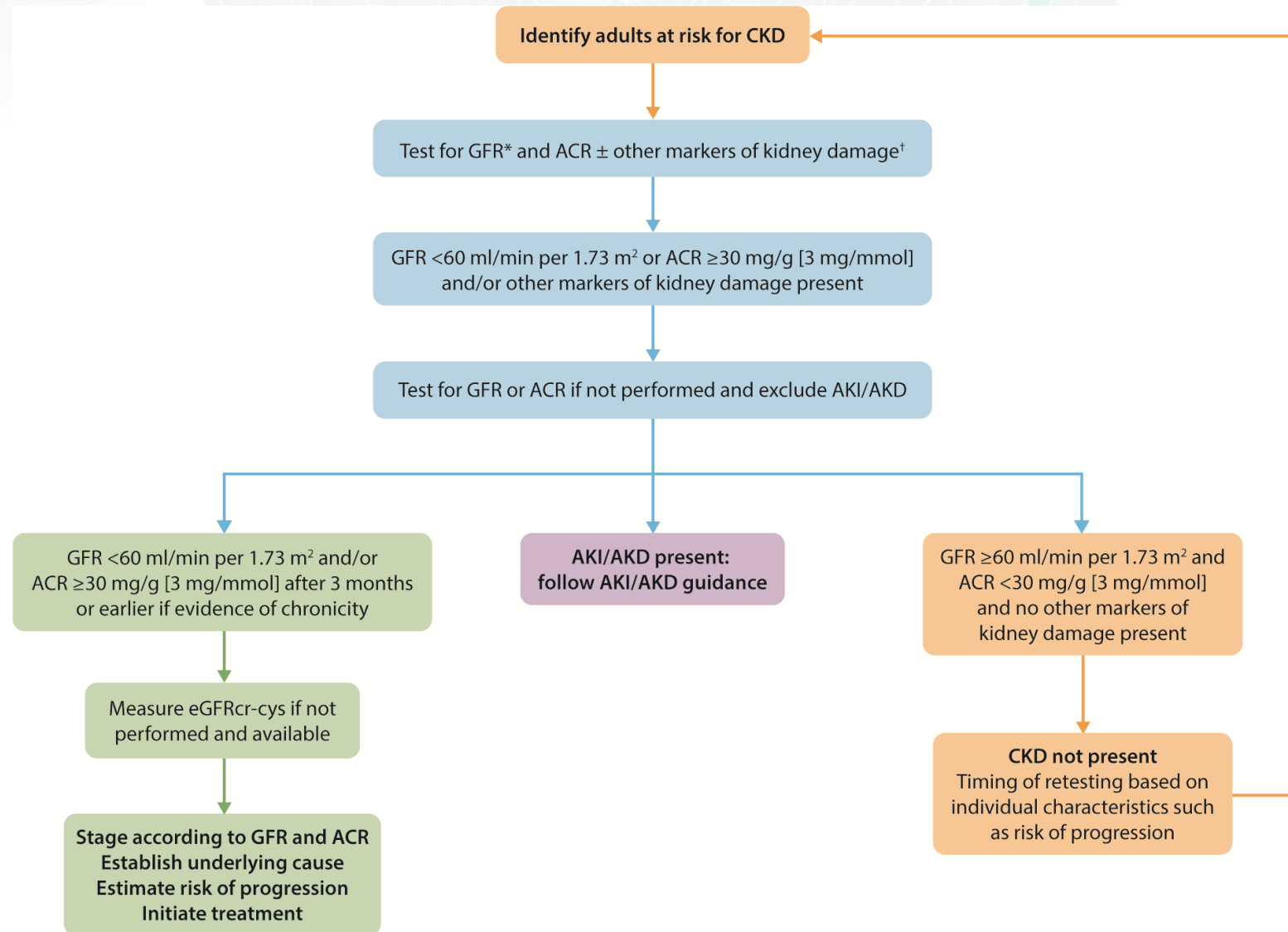
ANCA, antineutrophil cytoplasmic antibody; APOL1, apolipoprotein 1; COL4A, type IV collagen alpha chain; CT, computed tomography; GBM, glomerular basement membrane; MGRS: Monoclonal gammopathy of renal significance; HNF1B, hepatocyte nuclear factor 1B; MRI, magnetic resonance imaging; NPHS1, congenital nephrotic syndrome; PKD1, polycystic kidney disease-1; PKD2, polycystic kidney disease-2; PLA2R, M-type phospholipase A2 receptor; UMOD, uromodulin

EVALUATION – DISTINGUISH BETWEEN AKD AND CKD

It is important to distinguish between AKD and CKD and to establish chronicity.

Practice Point 1.1.3.1: Proof of chronicity (duration of a minimum of 3 months) can be established by:

- (i) review of past measurements/estimations of GFR;
- (ii) review of past measurements of albuminuria or proteinuria and urine microscopic examinations;
- (iii) imaging findings such as reduced kidney size and reduction in cortical thickness;
- (iv) kidney pathological findings such as fibrosis and atrophy;
- (v) medical history, especially conditions known to cause or contribute to CKD;
- (vi) repeat measurements within and beyond the 3-month point.



	AKI	AKD	CKD	NKD
Duration	Within 7 days	≤3 months	>3 months	
Functional criteria	Increase in SCr by 50% within 7 days Or Increase in SCr by 0.3 mg/dl (26.5 μmol/l) within 2 days Or Oliguria for ≥6 hours	AKI Or GFR < 60 ml/min/1.73 m ² Or Decrease in GFR by ≥35% Or Increase in SCr by >50%	GFR < 60 ml/min/1.73 m ²	GFR > 60 ml/min/1.73 m ²
And/or		And/or	And/or	And
Structural criteria	Not defined	Marker of kidney damage (albuminuria, hematuria, or pyuria are most common)	Marker of kidney damage (albuminuria is most common)	No kidney damage

Figure 1 | Functional and structural criteria for kidney diseases and disorders. AKD, acute kidney disease; AKI, acute kidney injury; CKD, chronic kidney disease; GFR, glomerular filtration rate; NKD, no kidney disease; SCr, serum creatinine.

[KDIGO executive conclusions](#)

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Harmonizing acute and chronic kidney disease definition and classification: report of a Kidney Disease: Improving Global Outcomes (KDIGO) Consensus Conference

Norbert H. Lameire^{1,9}, Adeera Levin^{2,9}, John A. Kellum^{3,9}, Michael Cheung⁴, Michel Jadoul⁵, Wolfgang C. Winkelmayer⁶ and Paul E. Stevens^{7,9}; for Conference Participants⁸



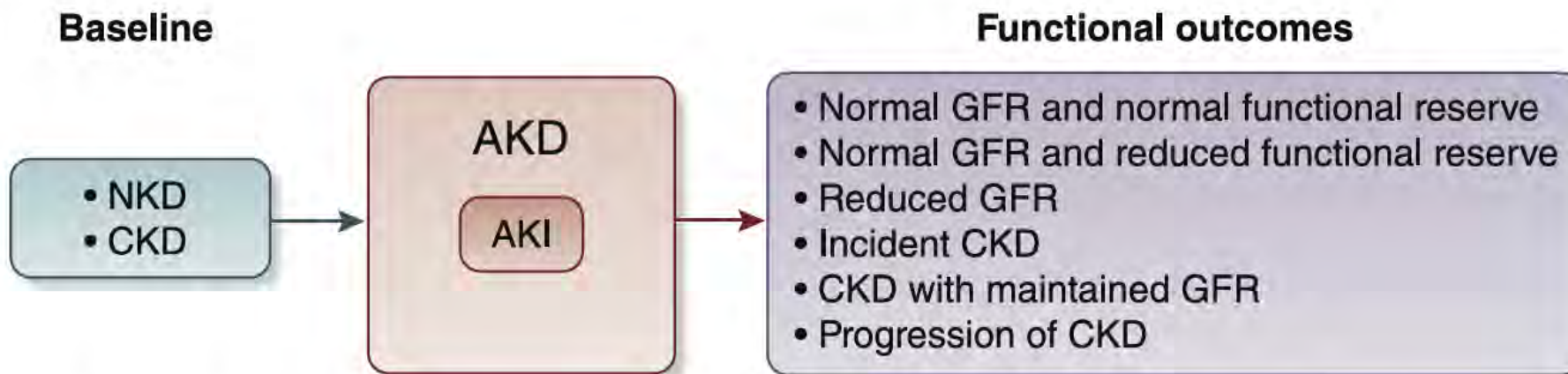


Figure 2 | Acute kidney disease (AKD), acute kidney injury (AKI), and kidney disease outcomes. CKD, chronic kidney disease; GFR, glomerular filtration rate; NKD, no kidney disease.

Harmonizing acute and chronic kidney disease definition and classification: report of a Kidney Disease: Improving Global Outcomes (KDIGO) Consensus Conference

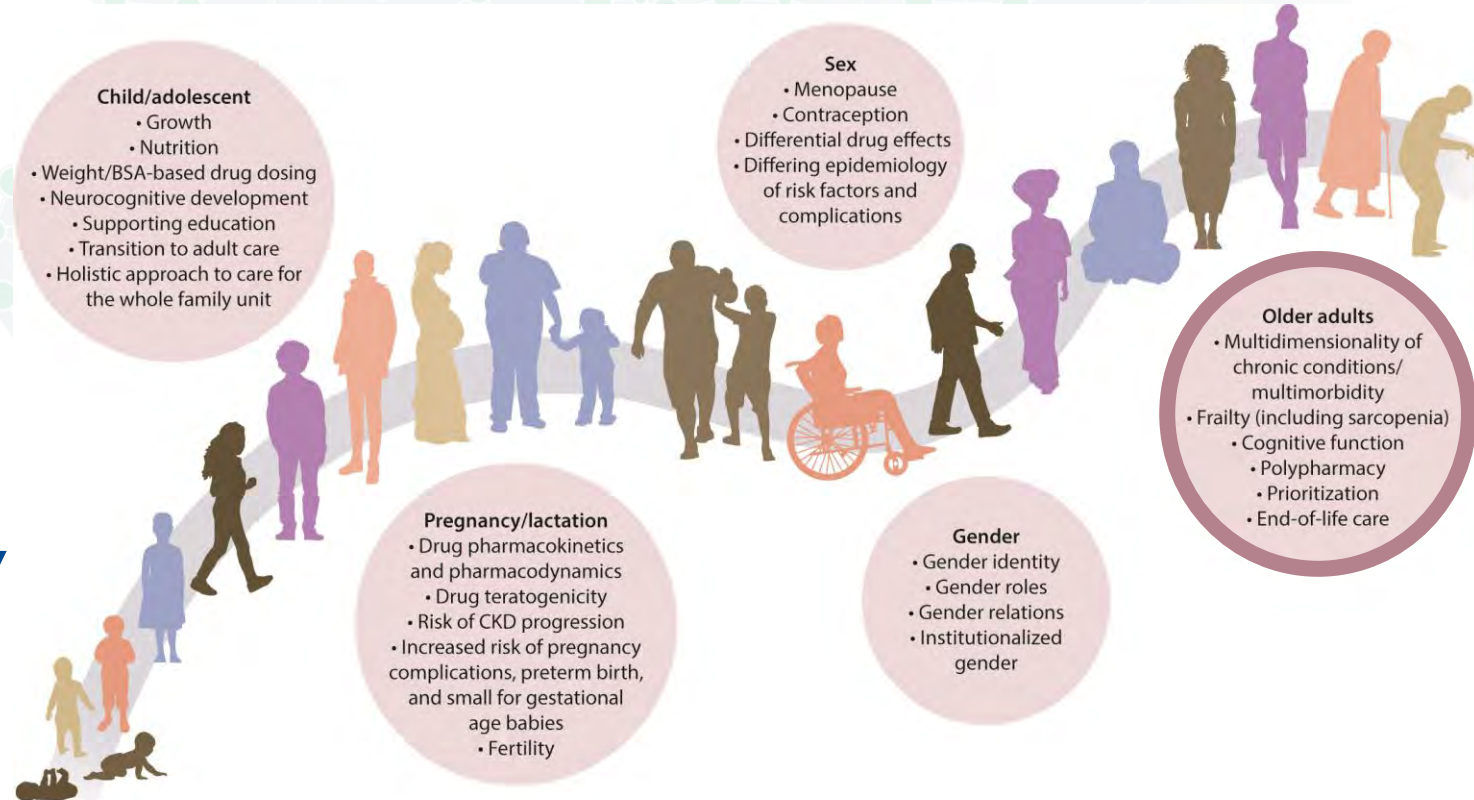
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EVALUATION –CKD CARE ACROSS THE LIFE SPAN

CKD impacts people across the lifespan and as a chronic condition, care is influenced by changes in life circumstances.

Use a personalized approach to diagnosis, risk assessment, and management that considers age, sex, and gender.

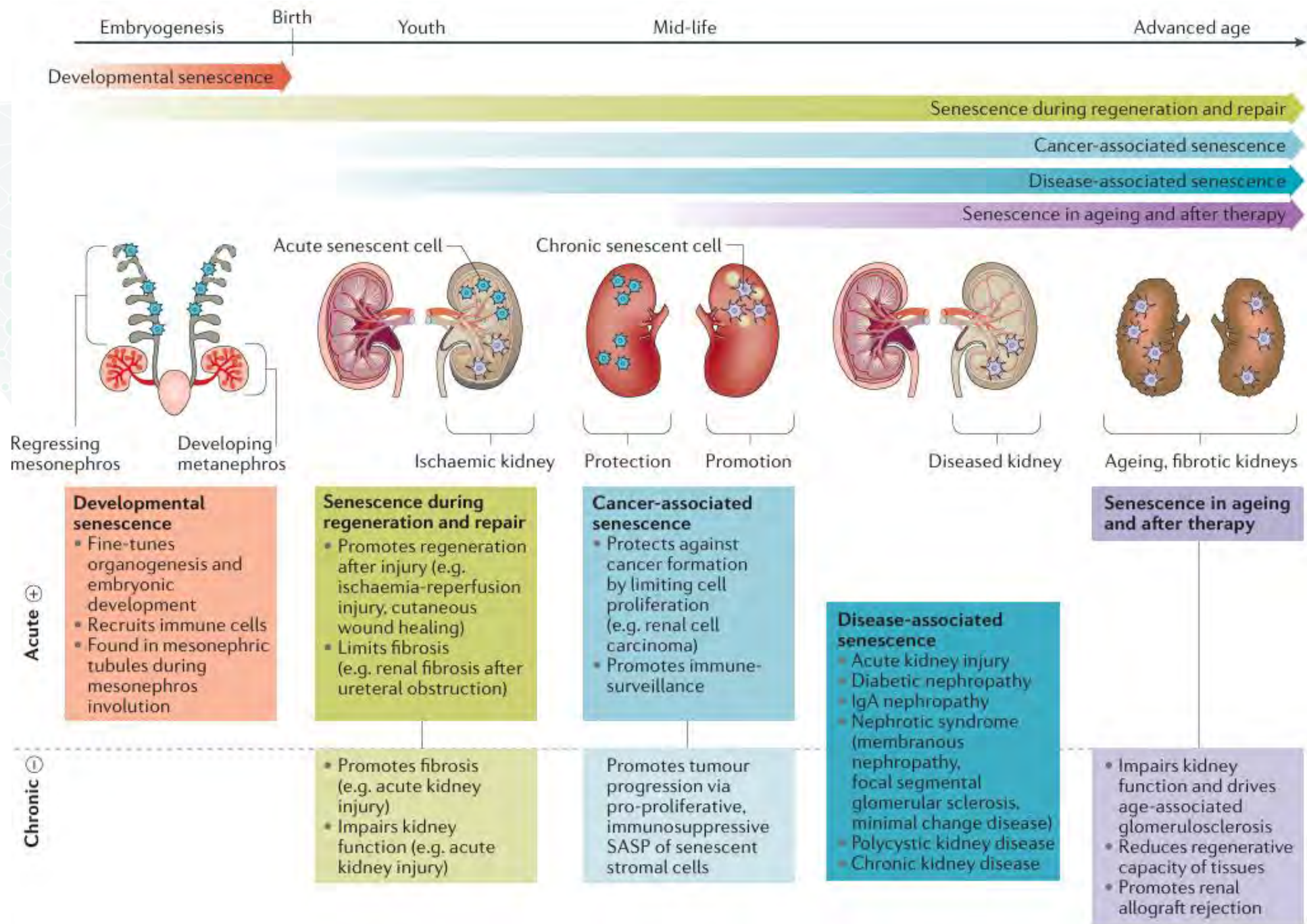
At the extremes of age - ***the very young and the very old*** – diagnostic procedures, treatment aims, treatment modalities, and decision-making differ due to differences in prognosis treatment options, and prioritization.



EVALUATION – DIAGNOSIS OF CKD IN OLDER ADULTS

Epidemiological population data support retaining the **threshold GFR of 60 mL/min/1.73 m² for diagnosis of CKD in older adults**, even in the absence of significant albuminuria, with consistently elevated and increasing relative risk of adverse outcomes below this threshold.

Age 65+	ACR, mg/g				ACR, mg/g			
	<10	10–29	30–299	300+	<10	10–29	30–299	300+
eGFRcr-cys	All-cause mortality				Myocardial infarction			
105+	1.2	1.4	1.9	3.5	0.97	1.4	2.0	19
90–104	ref	1.2	1.4	2.0	ref	1.2	1.1	1.9
60–89	1.2	1.5	1.8	2.3	1.1	1.4	1.5	1.9
45–59	1.6	2.0	2.4	2.9	1.6	1.9	2.3	3.4
30–44	2.0	2.4	3.2	4.1	2.1	2.6	3.1	3.8
<30	3.4	4.1	5.1	6.5	4.9	3.0	5.1	5.0
	Cardiovascular mortality				Stroke			
105+	1.1	1.5	2.0	12	1.2	1.3	1.5	3.3
90–104	ref	1.4	1.4	3.4	ref	1.3	1.3	2.8
60–89	1.2	1.7	2.2	3.1	1.1	1.4	1.8	2.5
45–59	1.7	2.4	3.0	4.3	1.5	1.7	2.0	2.3
30–44	2.4	3.1	4.5	5.8	1.5	2.0	2.1	2.3
<30	5.7	5.2	5.1	7.8	1.7	2.0	2.4	4.8
	Kidney failure replacement therapy				Heart failure			
105+	2.0	1.0	2.1		0.99	1.5	1.7	7.0
90–104	ref	1.9	4.7	10	ref	1.3	1.5	2.2
60–89	1.4	2.6	6.2	19	1.2	1.5	2.0	3.2
45–59	3.7	7.9	16	42	1.6	2.0	2.9	4.1
30–44	14	14	46	137	2.3	2.9	3.5	6.1
<30	87	364	241	406	4.4	4.1	5.5	7.2
	Acute kidney injury				Atrial fibrillation			
105+	0.91	1.1	1.3	1.9	0.95	1.1	1.0	3.7
90–104	ref	1.3	1.4	3.9	ref	1.2	1.3	2.4
60–89	1.5	2.1	2.7	4.7	1.1	1.2	1.5	2.0
45–59	3.6	4.3	5.1	7.3	1.2	1.4	1.7	1.9
30–44	5.7	5.9	7.2	9.8	1.5	1.8	2.0	2.2
<30	10	11	11	22	1.8	1.8	2.2	3.2
	Hospitalization				Peripheral artery disease			
105+	1.0	1.1	1.2	2.2	1.1	2.3	2.9	4.9
90–104	ref	1.1	1.3	1.4	ref	1.3	2.0	4.8
60–89	1.1	1.2	1.3	1.5	1.3	1.6	2.0	3.2
45–59	1.2	1.2	1.4	1.6	2.0	2.8	3.1	3.1
30–44	1.5	1.4	1.6	2.0	3.5	2.8	3.8	5.9
<30	1.9	1.9	2.0	2.6	8.4	4.1	5.9	10



GFR in Healthy Aging

METHODS



RENIS
(n=2946)



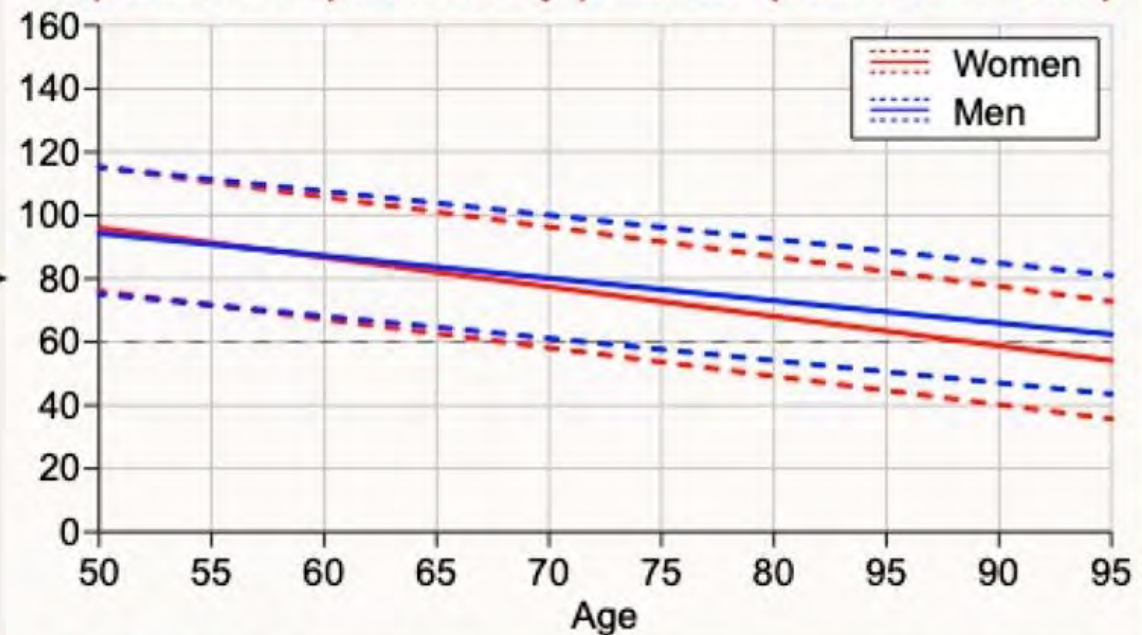
BIS
(n=547)



AGES-Kidney
(n=716)

Iohexol clearance
measured in
population-
based
cohorts

Iohexol clearance (median, 2.5th and 97.5th percentiles) in healthy persons (mL/min/1.73m²)



CONCLUSION: This study suggests that healthy aging is not associated with preserved GFR in old age.

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EVALUATION – IMPROVING ACCURACY OF GFR ASSESSMENT

Estimating GFR from a combination of creatinine and cystatin C (eGFRcr-cys) improves accuracy and strengthens risk relationships.

GFR should be measured where more accurate ascertainment of GFR will impact treatment decisions.

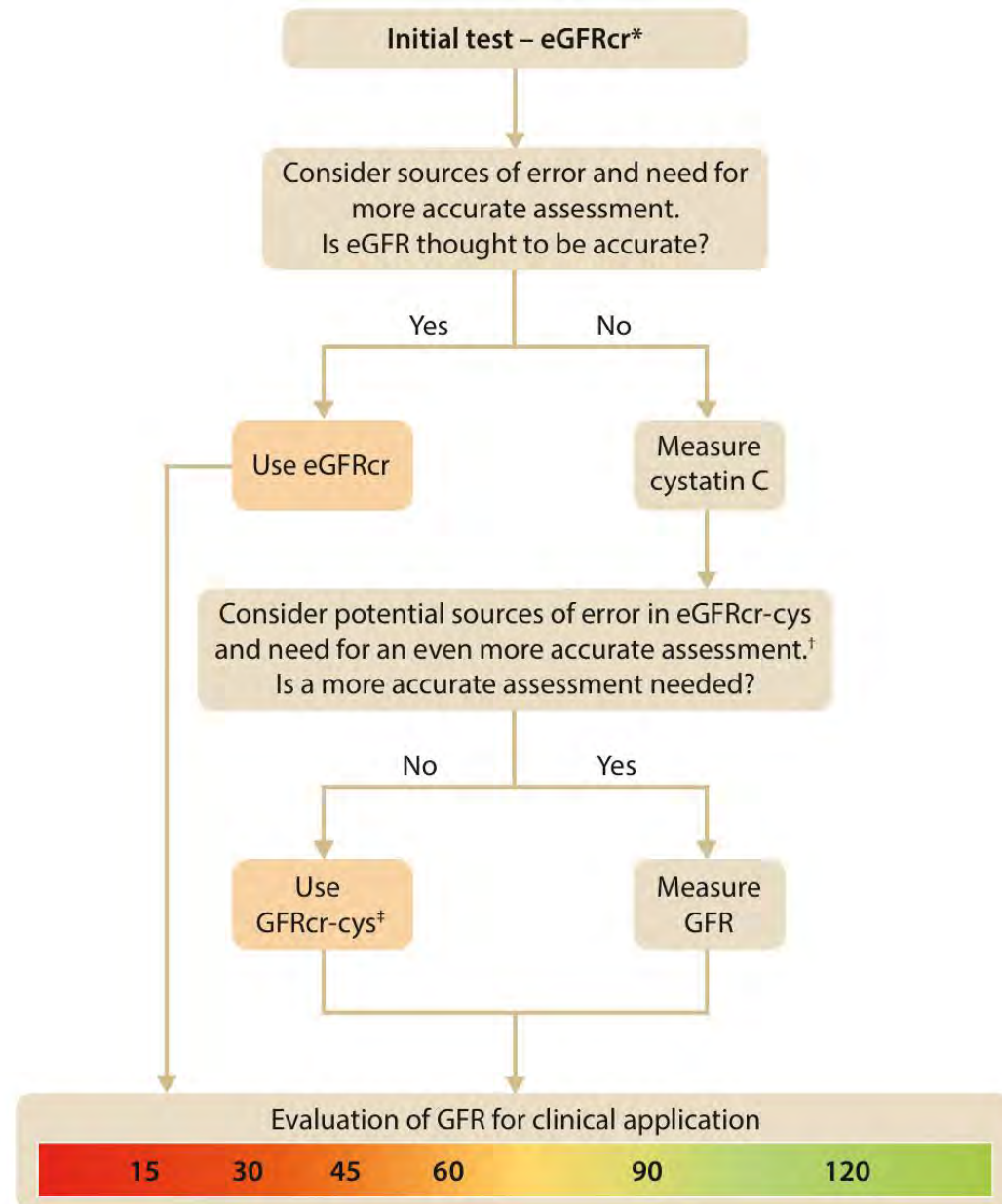














Table 7. Description of Initial and Supportive Tests for the Evaluation of GFR

GFR assessment method	Specific tests	Guidance for use and implementation
 <p>Estimated GFR</p>	 <p>Creatinine (eGFRcr) Cystatin C (eGFRcr-cys, eGFRcys)</p>	 <p>Most used method to assess GFR. In most cases, initial test for the evaluation of GFR. Standardized assay required to decrease between-center analytical variation</p>
 <p>mGFR</p>	 <p>Gold standard. Urinary or plasma clearance of exogenous markers (e.g., iohexol, iothalamate, 51Cr-EDTA, and 99m Tc-DTPA)</p>	 <p>Used in selected circumstances as listed in Table 8. Standardized assay required to decrease between-center analytical variation</p>
 <p>Timed urine clearance</p>	 <p>Creatinine</p>	 <p>Highly prone to errors and recommended only when no other options for supportive tests for GFR evaluation; performance under supervised conditions may decrease error</p>
 <p>Nuclear medicine imaging</p>	 <p>Imaging of the kidneys after injection of tracer cleared by the kidneys (e.g, 99mTc-DTPA scintigraphy)</p>	 <p>Highly prone to errors; not recommended</p>

Cr-EDTA, chromium 51-labeled ethylenediaminetetraacetic acid; 99m Tc-DTPA, technetium 99m-labeled diethylenetriamine pentaacetate; eGFRcr, creatinine-based estimated GFR; eGFRcr-cys, creatinine and cystatin C-based estimated GFR; eGFRcys, cystatin C-estimated GFR; GFR, glomerular filtration rate; mGFR, measured glomerular filtration rate.



Table 9. Comparison of estimated GFR and measured GFR



Estimated GFR by SCr and/or cystatin C	Measured GFR
Inexpensive and easy to implement	More expensive, more time-consuming, and invasive
Widely available and may also be used at point of care, easily repeatable	Only available in certain centers Methods to measure that do not require urine collections are available (i.e., plasma clearance) Most protocols require repeat blood samples potentially over a long duration Microsampling tests by fingerpick enable point-of-care testing. Testing has been described, but not routinely performed
Not sufficiently accurate and precise for all clinical situations	Accurate for GFR in all situations and across the GFR range. Requires individualized protocols
Lags behind changes in GFR	Able to identify early changes in GFR
Subject to non-GFR determinant confounding	Less influenced by non-GFR determinants

GFR, glomerular filtration rate; SCr, serum creatinine.

EVALUATION – ACCURACY AND RELIABILITY

Understand the variability of GFR and urinary albumin and the value and limitations of the methodology of assessment when determining whether a change is a true change.

Implement the requisite laboratory standards of care to ensure accuracy and reliability.

Table 16: Factors causing biological variation in urine albumin or urine protein








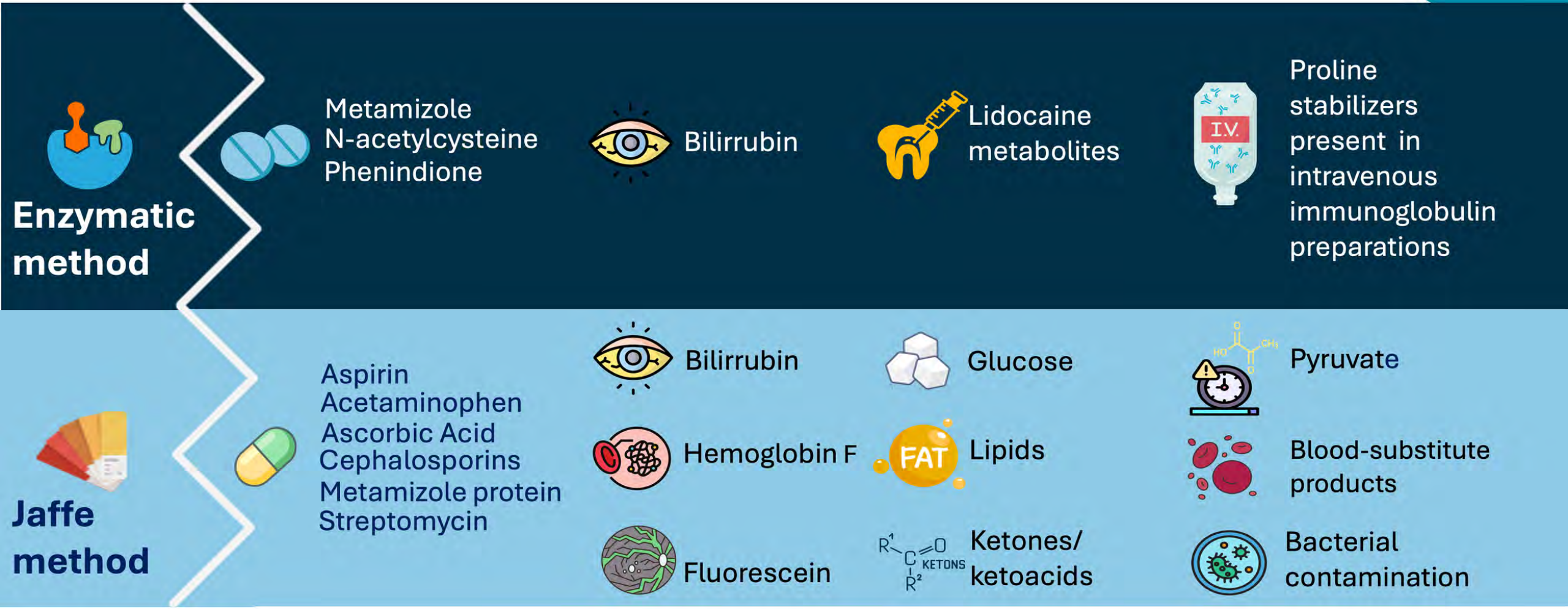
Factor	Falsely elevated ACR or PCR	False decrease in ACR or PCR
Variability in urine albumin or protein		
 Hematuria Menstruation	Increases albumin and protein in the urine	
 Exercise	Increases albumin and protein in the urine	
 Infection	Symptomatic urinary infection can cause production of protein from the organism	
 Nonalbumin proteins		Other proteins may be missed by albumin reagent strips
Variability in urinary creatinine concentration		
 Biological sex	Females have lower urinary creatinine excretion, therefore higher ACR and PCR	Males have higher urinary creatinine excretion, therefore lower ACR and PCR
 Weight	Low urinary creatinine excretion consistent with low weight can cause high ACR or PCR relative to timed excretion	High urinary creatinine excretion consistent with high weight can cause low ACR or PCR relative to timed excretion
 Changes in creatinine excretion	Lower urinary creatinine excretion with AKI or low-protein intake	High urinary creatinine excretion with high-protein intake or exercise

Table 12. Reported examples of substances that may cause analytical interferences in creatinine assays



EVALUATION – USE A VALIDATED GFR ESTIMATING EQUATION

Use a validated GFR estimating equation to derive GFR from serum filtration markers (eGFR) and use the same equation within geographical regions recognizing that these equations may differ for adults and children.

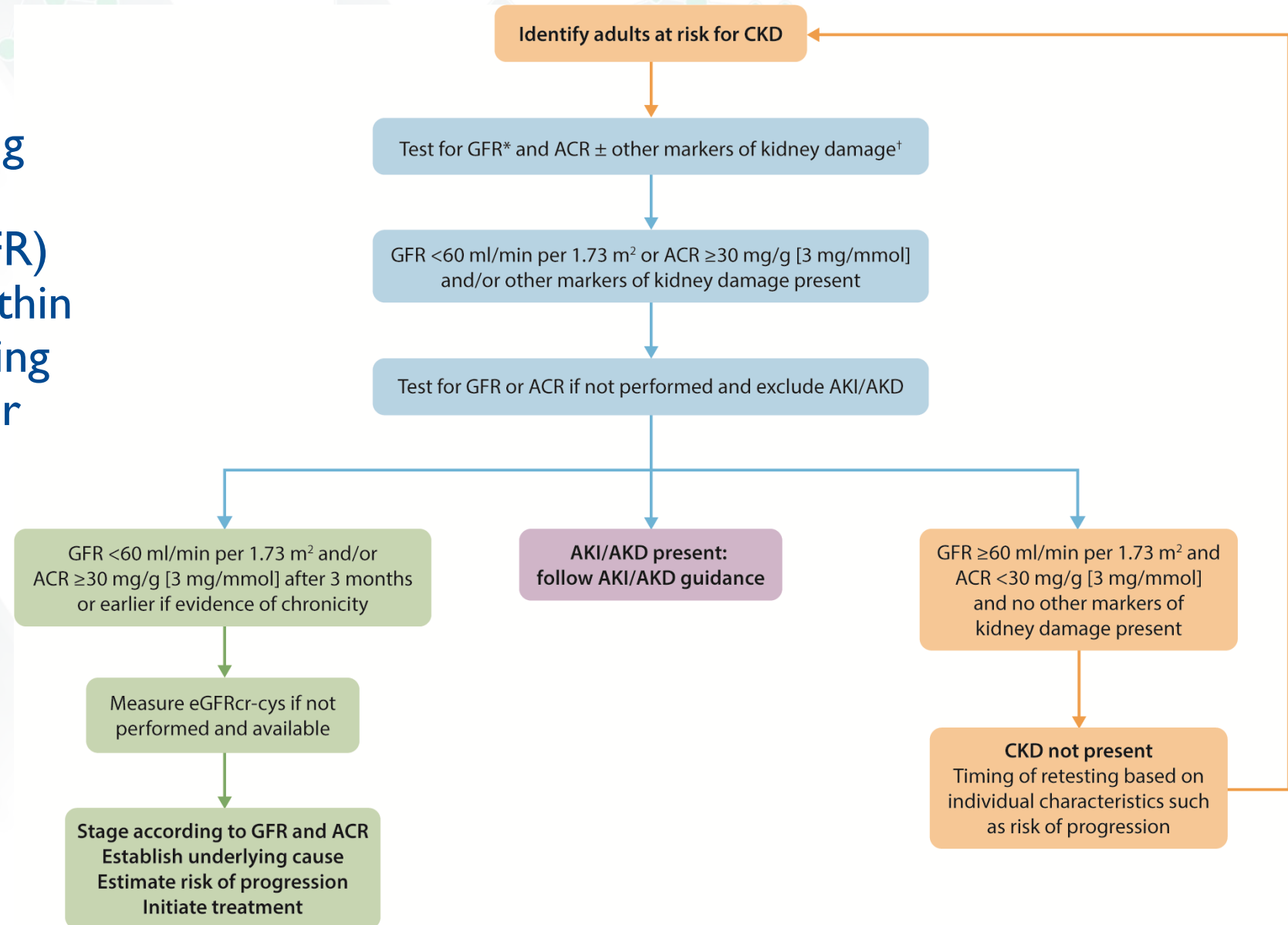










Table 14 | Validated GFR estimating equations

Marker	Equation name and year	Age	Variables	Development populations
Creatinine	 CKD-EPI 2009 ²³⁸	≥18; modification CKD-EPI 40 for pediatric available	Developed using A, S, R but reported not using the Black race coefficient, A, S, R (NB)	8254 Black and NB individuals from 10 studies in the United States and Europe ^a
	 CKiD U25 2021 ²³⁹	1–25	A, S, height	928 children with CKD in the United States and Canada
	 CKD-EPI 2021 ¹⁴⁷	≥18	A, S	8254 Black and NB individuals from 10 studies in the United States and Europe ^a
	 EKFC 2021 ²⁴⁰	2–100	A, S, European Black and NB specific Q-value; separate Q-values for Africa vs. Europe	mGFR vs. SCr (11,251 participants in 7 studies in Europe and 1 study from the United States) Normal GFR from 5482 participants in 12 studies of kidney donor candidates (100% Caucasian) European NB Q from 83,157 laboratory samples (age 2–40 years) in 3 European hospital clinical laboratories; European Black Q-value (N = 90 living kidney donors from Paris); African Black Q-value (N = 470 healthy individuals from République Démocratique de Congo); All Q-values developed in cohorts independent for EKFC development and validation
 Lund Malmö Revised 2014 ²⁴¹		A, S		3495 GFR examinations from 2847 adults from Sweden referred for measurement of GFR
 CKD-EPI 2009 Modified for China 2014 ^{b,242}	≥18	A, S		589 people with diabetes from the Third Affiliated Hospital of Sun Yat-sen University, China
 CKD-EPI 2009 Modified for Japan 2016 ^{b,83}	≥18	A, S		413 hospitalized Japanese people in 80 medical centers
 CKD-EPI 2009 Modified for Pakistan 2013 ^{b,235}	≥18	A, S		542 randomly selected low- to middle-income communities in Karachi and 39 people from the kidney clinic

(CKD-EPI) equation

GFR(mL/min/1.73 m²)

$$= 141 * \min(S_{Cr}/K, 1)^\alpha$$

$$* \max(S_{Cr}/K, 1)^{-1.209} * 0.993^{Age}$$

$$* 1.018[\text{if female}]$$

$$* 1.159 [\text{if African American}]$$

where

S_{Cr} is serum creatinine in mg/dL,

K is 0.7 for females and 0.9 for males

α is -0.329 for females and -0.411 for males,

min indicates the minimum of S_{Cr}/K or 1

max indicates the maximum of S_{Cr}/K or 1

- It is used to estimate GFR in adult patients with chronic kidney disease
- Considered more accurate than the MDRD Study equation for a GFR > 60 mL/min/1.73 m².

A Unifying Approach for GFR Estimation: Recommendations of the NKF-ASN Task Force on Reassessing the Inclusion of Race in Diagnosing Kidney Disease



Recommend immediate implementation of the **CKD-EPI creatinine equation refit without the race variable** in all laboratories in the U.S.

The equation refit excludes race in the calculation and reporting, includes diversity in its development, is immediately available to all labs in the U.S. and has acceptable performance characteristics and potential consequences that do not disproportionately affect any one group of individuals.



Recommend national efforts to facilitate increased, routine, and timely use of cystatin C, especially to confirm eGFR in clinical decision-making



Encourage and fund research on GFR estimation with new endogenous filtration markers and on interventions to eliminate racial and ethnic disparities



The Task Force gathered input from diverse stakeholders and carefully reviewed the evidence to create these recommendations

Cynthia Delgado, Mukta Baweja, Deidra C. Crews, et al. *A Unifying Approach for GFR Estimation: Recommendations of the NKF-ASN Task Force on Reassessing the Inclusion of Race in Diagnosing Kidney Disease.* *AJKD* DOI: 10.1053/j.ajkd.2021.08.003, *JASN* DOI: 10.1681/ASN.2021070988

Visual Graphic by Edgar Lerma, MD, FASN



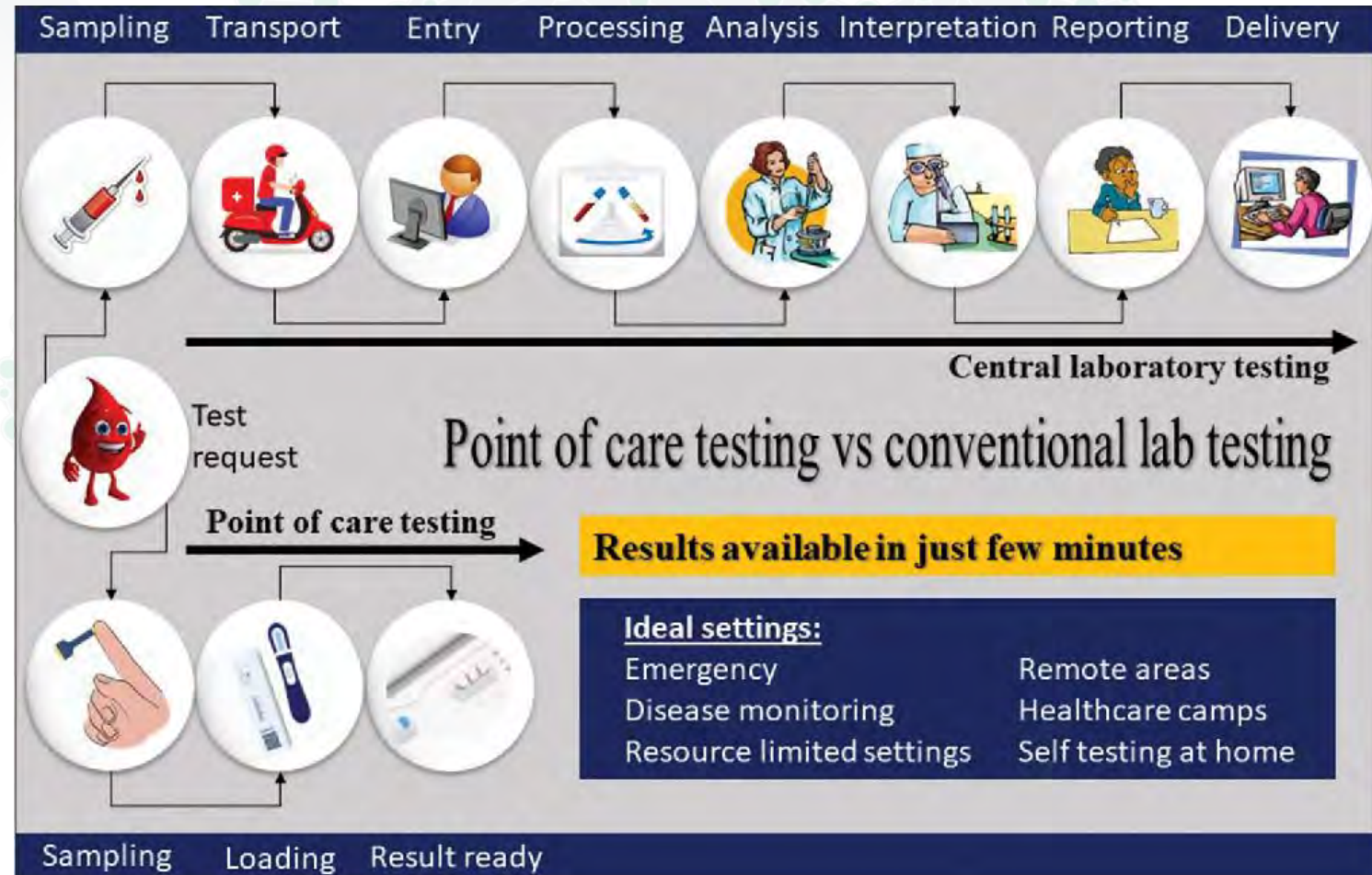
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EVALUATION – POINT-OF-CARE TESTS

Point-of-care tests (POCT) for creatinine (blood and saliva) and urine albumin measurement are available, and if adequately quality-assured, are accurate enough to facilitate the clinical pathway where access to a laboratory is limited.



Top 10 Takeaways for Nephrologists on Evaluation of People with or at Risk of CKD

1

CKD definition

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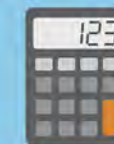
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8

Point-of-care tests (POCT)

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4

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5

Improving accuracy of GFR assessment

Estimating GFR from a combination of creatinine and cystatin C (eGFR_{cr-cys}) improves accuracy and strengthens risk relationships.

Cys-C

10

Timing assessment and reevaluation

Use validated risk prediction tools and clinical evaluation when determining timing of follow up and reassessment.



EVALUATION – USE VALIDATED RISK ASSESSMENT TOOLS

Use validated risk assessment tools to aid in decision-making and timing of multidisciplinary care.

Choose the appropriate tool for the event of interest: kidney failure treatment, cardiac events, or mortality.

CKD is classified based on:

- Cause (C)
- GFR (G)
- Albuminuria (A)

				Albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–299 mg/g 3–29 mg/mmol	≥300 mg/g ≥30 mg/mmol
GFR categories (ml/min/1.73 m ²) Description and range	G1	Normal or high	≥90	Screen 1	Treat 1	Treat 3
	G2	Mildly decreased	60–89	Screen 1	Treat 1	Treat 3
	G3a	Mildly to moderately decreased	45–59	Treat 1	Treat 2	Treat 3
	G3b	Moderately to severely decreased	30–44	Treat 2	Treat 3	Treat 3
	G4	Severely decreased	15–29	Treat* 3	Treat* 3	Treat 4+
	G5	Kidney failure	<15	Treat 4+	Treat 4+	Treat 4+

■ Low risk (if no other markers of kidney disease, no CKD)
■ Moderately increased risk

■ High risk
■ Very high risk





Table 19. Externally validated risk equations for Predicting kidney failure in the general (G3-G5) population



Equation	Variable	Population	Outcome (time horizon)	Discrimination and calibration	Usability
KFRE ^{9,10,407,408} www.kidneyfailurerisk.com www.ckdpc.org/risk-models.html	Age, sex, eGFR, ACR (4 variable) + calcium, phosphate, bicarbonate, and albumin (8 variables)	>1 million patients, >100,000 events from more than 30 countries	Treated kidney failure (2–5 yr)	0.88–0.91/+	+
KPNW ⁴¹⁰	Age, sex, eGFR, albuminuria, systolic BP, antihypertensive use, diabetes, and diabetes complications	39,013 patients, 1097 events from the Kaiser Permanente Health System (United States)	Kidney failure (5 yr)	0.95/+	+
Landray <i>et al.</i> ⁴¹¹	Sex, SCr, albuminuria, and phosphate	595 patients, >190 events from the CRIB and East Kent cohorts in the United Kingdom	Kidney failure	0.91/+	–
Z6 score ⁴⁰⁹	SCr, albumin, cystatin C, urea, hemoglobin, and ACR	7978 patients, 870 events—developed in the German CKD study, validated in 3 additional European cohorts	Kidney failure (5 yr)	0.89–0.92/+	–


ACR, albumin-to-creatinine ratio; BP, blood pressure; CKD, chronic kidney disease; CRIB, chronic renal impairment in Birmingham; eGFR, estimated glomerular filtration rate; KFRE, Kidney Failure Risk Equation; KPNW, Kaiser Permanente Northwest; SCr, serum creatinine.


Understanding the

Kidney Failure Risk Equation (KFRE)

What is the KFRE?

A simple formula to help doctors predict whether your kidneys will fail in the next two to five years. Using just **four pieces of information**, the formula calculates the likelihood of kidney failure requiring dialysis or transplant.


 ***Glomerular filtration rate:** How well your kidneys are filtering


 ***Urine protein test:** Ratio of creatinine to albumin



Why is the KFRE important?

Only a small minority of people with kidney disease will progress to kidney failure. The risk equation helps ensure everyone with kidney disease receives the most appropriate care based on their individual risk.

 Low-risk individuals avoid unnecessary anxiety due to fear of kidney failure and the "chronic disease" label

 High-risk individuals get faster referral to specialist care that can help delay or prevent progression

10% of Canadians are living with chronic kidney disease



Only 3% of people with CKD experience kidney failure

Who uses the equation?

The equation is widely used around the world* by kidney specialists. Can-SOLVE CKD researchers are working to develop interactive tools that will help primary care doctors in Canada use the equation to make individualized treatment.

*Validated in more than 700,000 patients in 30+ countries

Learn more: kidneyfailurerisk.com

THE KIDNEY FAILURE RISK EQUATION

Find out your real risk of kidney failure



KIDNEY FAILURE RISK CALCULATOR

Risk Assessment



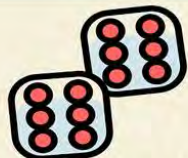
Assess urine albumin/ proteinuria and GFR
1/year in CKD



More frequently in those at higher risk



Fall in eGFR > 20% on subsequent tests or >30%
in those on active therapies needs urgent
evaluation



Doubling of serum creatinine requires urgent
evaluation

Risk Prediction

Kidney failure risk

Externally validated risk equation tool in CKD
G3-G5 is recommended



eGFR 30-60 → 5 year KF risk 3-5% → Nephrology referral
eGFR < 30 → 2 year KF risk > 40% → Interprofessional care
eGFR < 20 → 2 year KF risk > 40% → Transplant planning



Risk prediction equation G3-G5 not same as G1-G2



Use of disease-specific, externally validated equations in
IgA nephropathy and ADPKD



Cardiovascular risk

Use of externally validated models that are developed in
CKD populations or that incorporate eGFR and albuminuria



Use of externally validated models that predict all-cause
mortality in CKD population

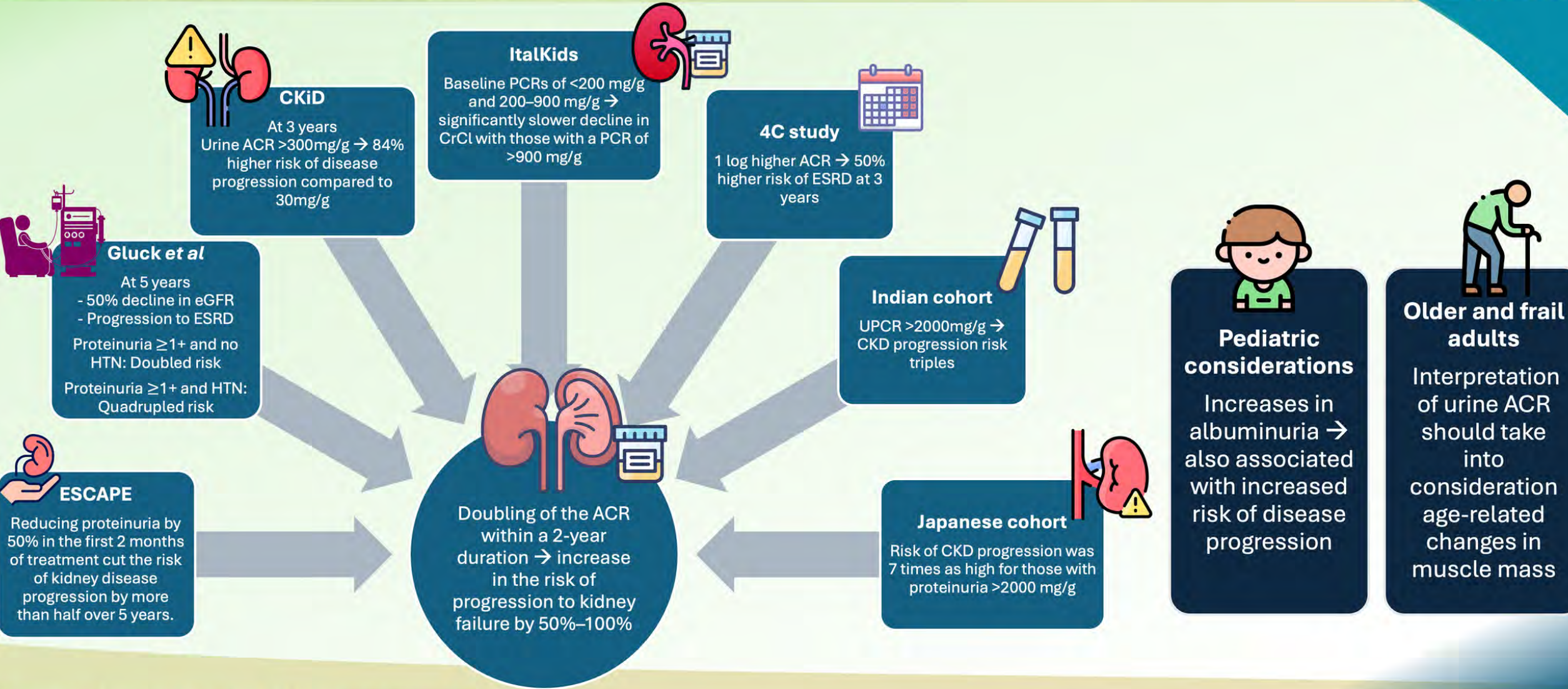




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Monitoring proteinuria:

For albuminuria monitoring of people with CKD, a doubling of the ACR on a subsequent test exceeds laboratory variability and warrants evaluation.



EVALUATION – TIMING ASSESSMENT AND REEVALUATION

Timing of follow up and reassessment using validated risk prediction tools and clinical evaluation, together with education, may inform better selection of targets of care to support people and families living with CKD.

This approach is part of longitudinal care.

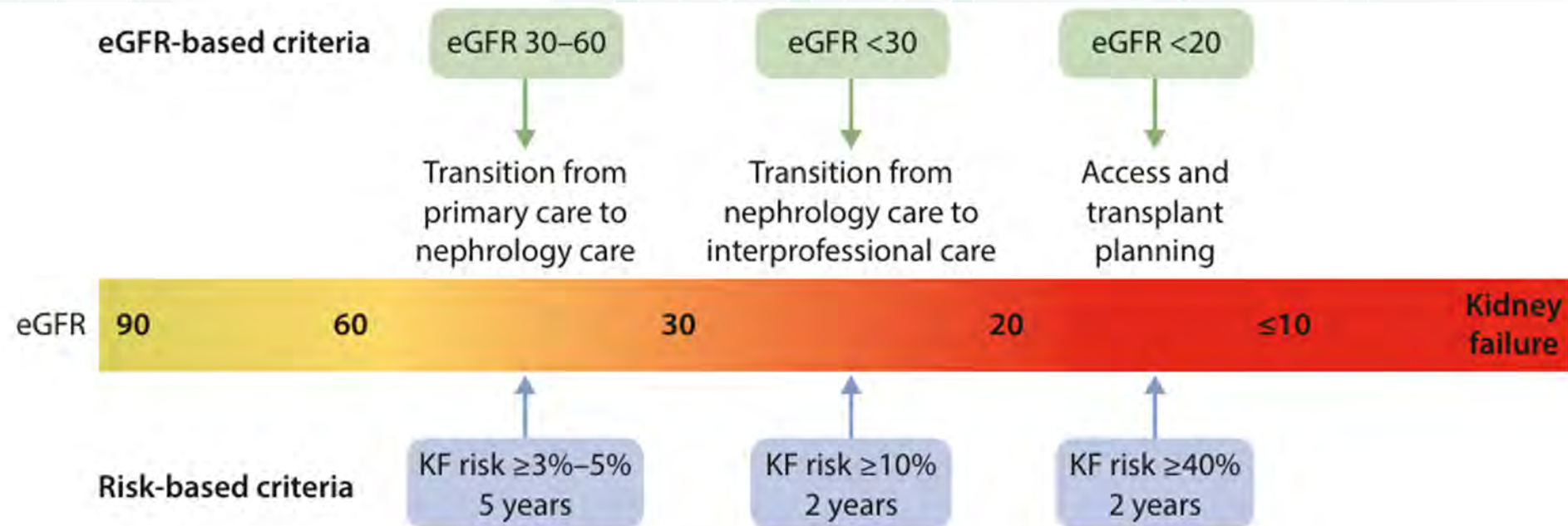


Figure 15 | Transition from an estimated glomerular filtration rate (eGFR)-based to a risk-based approach to chronic kidney disease care. KF, kidney failure.

KDIGO Recommendations on CKD Evaluation



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Method for CKD Staging



Creatinine based estimated GFR (eGFRcr)

If cystatin C available



AVAILABLE

Combination of creatinine and cystatin C based estimated GFR (eGFRcr-cys)

Strength 1B

Evaluation of CKD



To evaluate the CKD cause and guide treatment decision



Suggest kidney biopsy as a safe/acceptable diagnostic test

Strength 2D

Other



Use eGFRcr-cys in situations when eGFR is less accurate



Use validated GFR estimating equation



Use Point of Care (POC) test for creatinine & urine albumin testing with limited lab access

Strength 1C, 1D, 2C



Practice Points for CKD Evaluation

CKD Detection

Test people at risk



- Urine albumin
- GFR



If abnormal- repeat test to confirm

CKD Staging

Method



-Creatinine based eGFR



AVAILABLE

-If cystatin C available-creatinine and cystatin based eGFR estimation

Evaluate chronicity



- Records/GFR
- Imaging-shrunken kidney size
- Pathology (fibrosis/atrophy)



- Repeat testing within/beyond 3 months
- Medical history/Comorbid conditions

GFR estimation



- Use validated eGFR equation
- Use same equation within geographical region
- Don't use race in eGFR computation



CKD Cause



- ✓ Lab (serology / urine)
- ✓ Imaging
- ✓ Biopsy
- ✓ Genetics



Kidney biopsy is an acceptable/safe diagnostic test for CKD evaluation

Pediatric



- Use enzymatic creatinine assay
- eGFR_{cr} <90- low

Albuminuria test



-First void morning urine



- Urine ACR
- Reagent strip



-Use Point of care test (POCT) if lab access limited

GFR evaluation



Use eGFR_{cr}-cys in situations when eGFR_{cr} less accurate



**TOP 10 TAKEAWAYS ON
MANAGEMENT OF PEOPLE
WITH OR AT RISK OF CKD**

Top 10 Takeaways for Nephrologists on the Management of People with CKD

1 Comprehensive treatment strategy



Lifestyle: Healthy diet and Physical activity



Stop tobacco products

6 Cardiovascular disease



Estimate 10-year CVD risk using validated tool → preventive strategy



Imaging/ invasive strategies using contrast media: not contraindicated in all

2 Adopting a healthy and diverse diet



Prefer plant-based foods over animal-based foods



Avoid ultra-processed foods

7 Thorough medication review



Validated eGFR equations using serum creatinine for drug dosing



Limits over-the counter and herbal remedies

3 Individualize BP control



Target office BP: SBP <120mmHg



Less intensive strategy in frailty, high risk of falls, low life expectancy

8 Discontinuation and restart of medications



Clear plan of restarting SGLT2i/RASi while discontinuing



Re-initiation of SGLT2i/RASi: Required to avoid harm

4 Renin-angiotensin-system Inhibitors (RASi) and SGLT2i



In all CKD with moderate and severe albuminuria with/without diabetes



CKD and heart failure – SGLT2i irrespective of albuminuria

9 Symptom control in CKD



Patient-centered management: Symptom assessment and redirecting treatment



Effective communication and shared decision making

5 Acute changes in eGFR after RASi



Initial dips in eGFR expected after initiation of RAS1/SGLT2i



An eGFR fall of ≥30%: Warrants evaluation

10 Advanced care planning



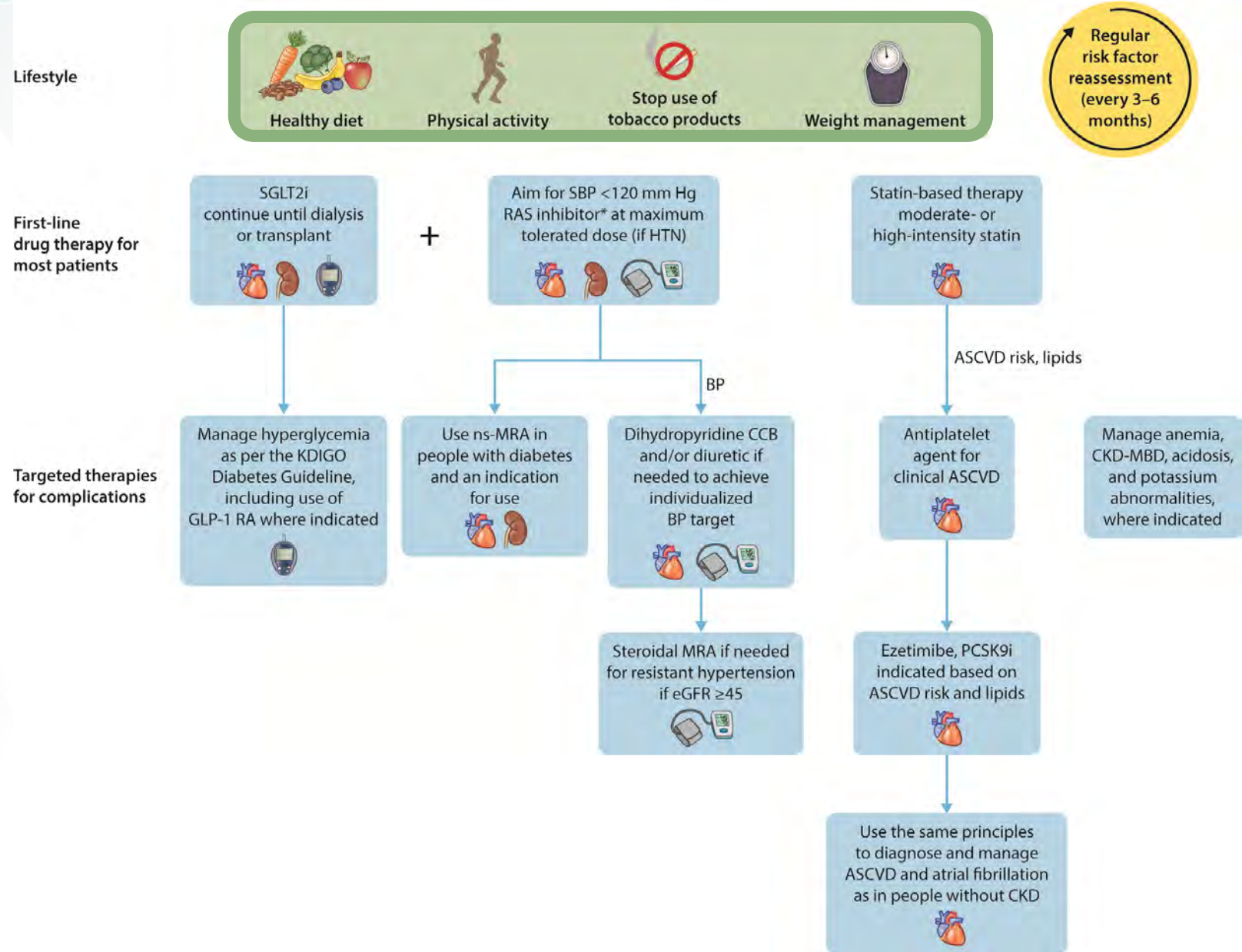
Future health care plans: Discussed and jointly agreed with patient and caregivers



Appropriate supportive care

MANAGEMENT – COMPREHENSIVE TREATMENT STRATEGY

Treat people with CKD with a comprehensive treatment strategy to reduce risks of progression of CKD and its associated complications encompassing education, lifestyle, exercise, smoking cessation, diet, and medications, where indicated.



Lifestyle Modification in CKD

Physical activity should consider age/ ethnic background/presence of comorbidities and access to resources



People with CKD should be advised to avoid sedentary behavior



Encourage people with obesity and CKD to lose weight



People with CKD are advised to undertake moderate intensity physical activity for cumulative duration of at least **150 minute/ week**

Strength 1D



Avoid use of tobacco products



For people at higher risk of falls, healthcare providers should provide advice on the intensity and type of physical activity



Encourage children with CKD to undertake physical activity and achieve a healthy weight



- ✓ 1-5 years-≥**180 min daily**
- ✓ 5-17 years-≥**60 min daily**
- ✓ Limit sedentary/screen time

MANAGEMENT – HEALTHY AND DIVERSE DIET

Adopting a healthy and diverse diet with a higher consumption of plant-based foods compared to animal-based foods and a lower consumption of ultra-processed foods has the potential to benefit complications related to progressive CKD such as acidosis, hyperkalemia, and hyperphosphatemia with less risk of protein energy-wasting.

Lifestyle



Healthy diet



Physical activity



Stop use of tobacco products



Weight management

Regular risk factor reassessment (every 3–6 months)

Animal proteins



Meat, poultry, fish, seafood, eggs:

28 g (1 oz) = 6–8 g protein

1 egg = 6–8 g protein

Dairy, milk, yogurt, cheese:

250 ml (8 oz) = 8–10 g protein

28 g (1 oz) cheese = 6–8 g protein

Plant proteins



Legumes, dried beans, nuts, seeds:

100 g (0.5 cup) cooked = 7–10 g protein

Whole grains, cereals:

100 g (0.5 cup) cooked = 3–6 g protein

Starchy vegetables, breads:

2–4 g protein

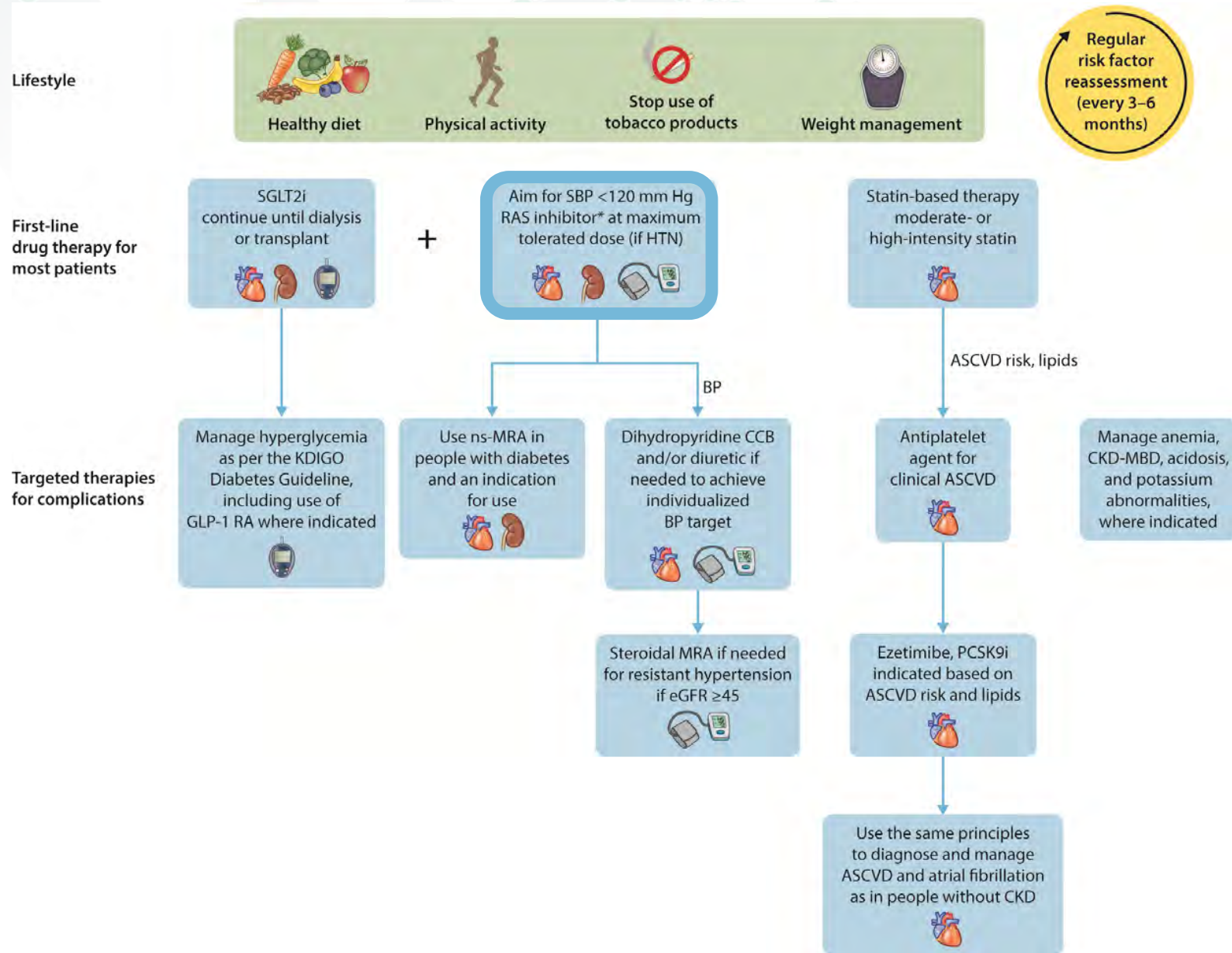
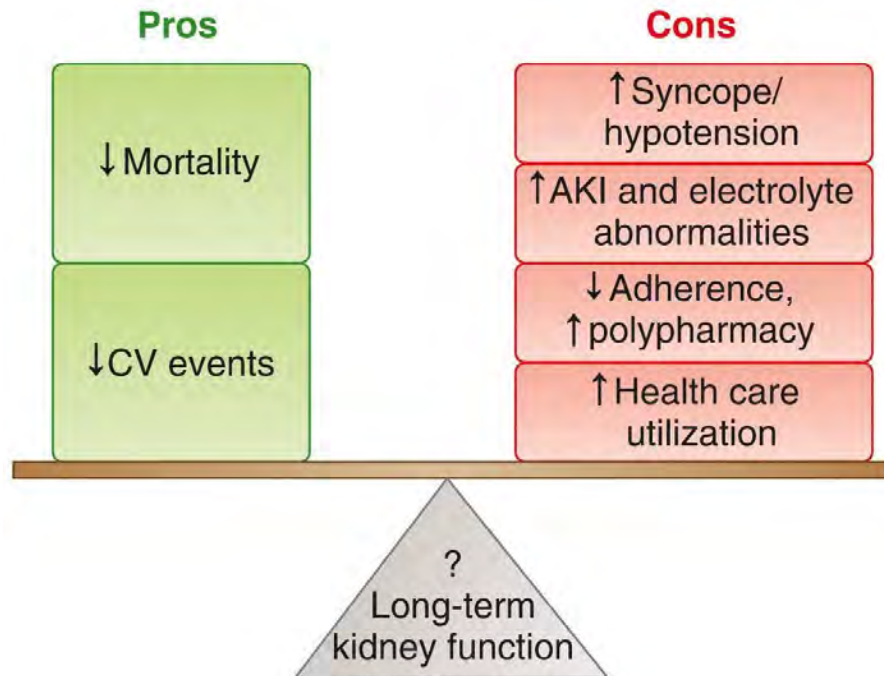
Table 21 | Impact of plant-based foods in people with CKD

Study (N); study design	CKD stage or GFR	Intervention (follow-up)	Outcome
CRIC ⁴⁶⁷ (N = 2403); observational	20–70 ml/min per 1.73 m ²	High DASH vs. low DASH (14 yr)	CKD progression: HR: 0.83; 95% CI: 0.69–0.99 Mortality: HR: 0.75; 95% CI: 0.62–0.90
NHANES ⁴⁶⁸ (N = 1110); observational	30–59 ml/min per 1.73 m ²	DASH by quintiles (7.8 yr)	Kidney failure relative hazard (RH) compared with quintile 5: quintile 1: RH: 1.7; 95% CI: 1.1–2.7; quintile 2: RH: 2.2; 95% CI: 1.1–4.1
CORDIOPREV ⁴⁶⁶ (N = 53); RCT	<60 ml/min per 1.73 m ²	Mediterranean diet vs. low-fat diet (5 yr)	Decline in GFR –3.72 ml/min per 1.73 m ² vs. –5.4 ml/min per 1.73 m ² , <i>P</i> = 0.03
CKD QLD ⁴⁶⁹ (N = 145); observational	CKD G3–G4	High vegetable and nut intake (median 36 mo)	Composite all-cause mortality, kidney failure, or doubling of SCr: HR: 0.61, 95% CI: 0.39–0.94
REGARDS ⁴⁷⁰ (N = 3972); observational	<60 ml/min per 1.73 m ²	Plant-based diet (6 yr)	All-cause mortality: HR: 0.77; 95% CI: 0.61–0.97
NHANES III ⁴⁶⁵ (N = 5346); observational	<60 ml/min per 1.73 m ²	Increasing plant-to-protein ratio (8.4 yr)	All-cause mortality for every 33% increase: HR: 0.77, 95% CI: 0.61–0.96
Longitudinal study of aging women ⁴⁶⁴ (N = 1374); observational	Baseline 65.6 ± 13.1 ml/min per 1.73 m ²	Higher vs. lower intake of plant-based protein (10 yr)	Each 10 g higher intake of plant-based protein reduced a decline in GFR by 0.12 ml/min per 1.73 m ² per year

CI, confidence interval; CKD, chronic kidney disease; CKD QLD, Chronic Kidney Disease in Queensland; CORDIOPREV, CORonary Diet Intervention with Olive oil and cardiovascular PREvention study; CRIC, Chronic Renal Insufficiency Cohort; DASH, Dietary Approaches to Stop Hypertension; GFR, glomerular filtration rate; HR, hazard ratio; NHANES, National Health and Nutrition Examination Survey; RCT, randomized controlled trial; REGARDS, Reasons for Geographic and Racial Differences in Stroke; SCr, serum creatinine.

MANAGEMENT – INDIVIDUALIZE BP CONTROL

Individualize BP-lowering therapy and treatment targets in people with frailty, high risk of falls, very limited life expectancy, or symptomatic postural hypotension.



Intensive versus Standard Blood-Pressure Control

RANDOMIZED, CONTROLLED, OPEN-LABEL, MULTI-CENTER CONTROLLED TRIAL



Age 50y or greater, SBP of 130-180 mmHg ASCVD risk >10%, No Diabetes, No Stroke

Intensive Treatment

Targetted Systolic Blood Pressure:
<120 mmHg Systolic

N = 4678



Standard Treatment

Targetted Systolic Blood Pressure:
<140 mmHg Systolic

N = 4683



5.2%

FIRST OCCURENCE OF MI, ACS, STROKE,
HF OR DEATH FROM CVS CAUSE
HR 0.75; 95% CI, 0.64 to 0.89; P<0.001






6.8%

3.3%

DEATH FROM ANY CAUSE
HR 0.73; 95% CI, 0.60 to 0.90; P=0.003

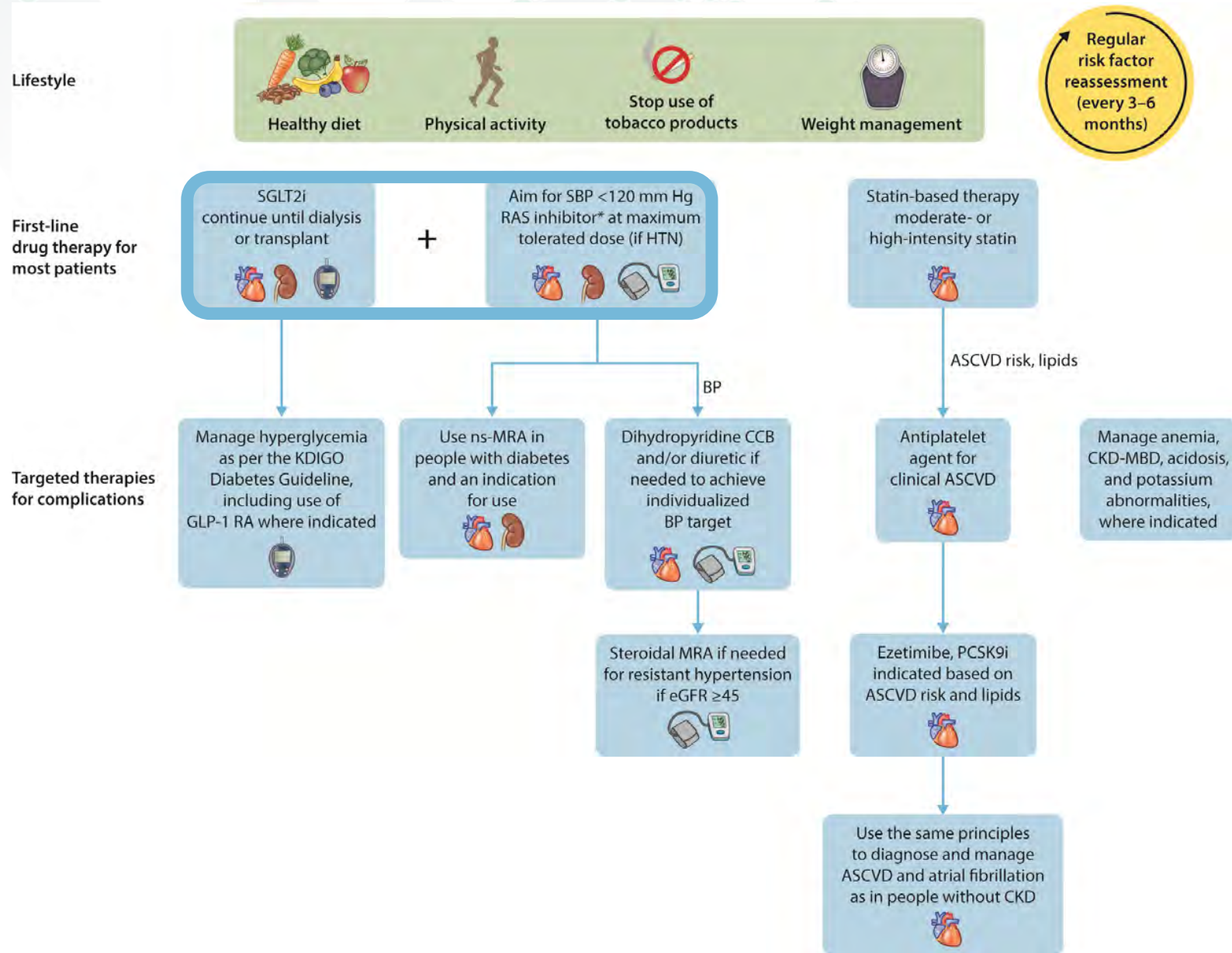
4.5%

CHOICE OF ANTIHYPERTENSIVE IN CKD G1-4

	NON DIABETIC			DIABETIC		
Albuminuria	A1	A2	A3	A1	A2	A3
First-line agent (Grade of recommendation)	ACEi/ ARB (Practice Point)	ACEi/ ARB (2C)	ACEi/ ARB (1B)	ACEi/ ARB (Practice Point)	ACEi/ ARB (1B)	ACEi/ ARB (1B)
Key trials 	HOPE (vs placebo, pre-specified CKD subgroup)	HOPE (vs placebo, pre-specified CKD subgroup)	REIN (Ramipril vs placebo)	HOPE (vs placebo, pre-specified CKD subgroup, 1/3 DM)	CKD G1-3 Micro-HOPE (ACEi vs placebo)	CKD G3-4 IDNT* (Irbesartan vs CCB vs placebo) RENAAL (Losartan vs placebo)
Outcome 	No clear benefits, (low baseline risk)	 ✓ AASK- Improved	✓ Improved, x2Cr, ESKD	No clear benefits, (low baseline risk)	No studies powered for hard renal outcomes	✓ Improved with RASi (ESKD, x2Cr and all cause mortality)
Comments 	✓ Reduced stroke and all-cause mortality (but lowering BP was not the aim, 1/3 DM, 1/2 HTN)	✓ HOPE Reduced stroke and all-cause mortality (but lowering BP was not the aim, 1/3 DM, 1/2 HTN)	✓ Improved (Greater benefit in higher albuminuria)	✓ Reduced stroke and all-cause mortality (but lowering BP was not the aim, 1/3 DM, 1/2 HTN)	✓ Improved (baseline high CV risk)	
Comments 	Other agents are as appropriate	Limited studies with other agents	Limited evidence for other agents. ROAD- Benazepril vs losartan (similar)	Other agents are as appropriate	ACEi may lower progression to higher grades of albuminuria	Renoprotection stronger for lower GFR and higher albuminuria

MANAGEMENT – INDIVIDUALIZE BP CONTROL

Individualize BP-lowering therapy and treatment targets in people with frailty, high risk of falls, very limited life expectancy, or symptomatic postural hypotension.



Delaying CKD progression with SGLT2i



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Who should be on SGLT2i

RECOMMENDATIONS



Type 2 diabetes with
CKD (eGFR ≥ 20 ml/min/1.73m² (1A))



CKD (eGFR ≥ 20 ml/min/1.73 m²)
with
Urine ACR ≥ 200 mg/g
OR
Heart failure, irrespective of level of
albuminuria (1A)



eGFR 20 to 45 ml/min per 1.73 m²
with
Urine ACR < 200 mg/g (2B)

PRACTICE POINTS



Continue once initiated
even if eGFR falls < 20 ml/min/1.73 m²
unless

- ⊗ Not tolerated OR
- ⊗ KRT initiated



Withhold during

- ⊗ Prolonged fasting
- ⊗ Surgery
- ⊗ Critical medical illness



eGFR Decline after SGLT2 Inhibitor Initiation: The Tortoise and the Hare Reimagined

Alejandro Y. Meraz-Muñoz¹, Jordan Weinstein,¹ and Ron Wald^{1,2}
 KIDNEY360 2: 1042-1047, 2021. doi: <https://doi.org/10.34067/KID.0001172021>

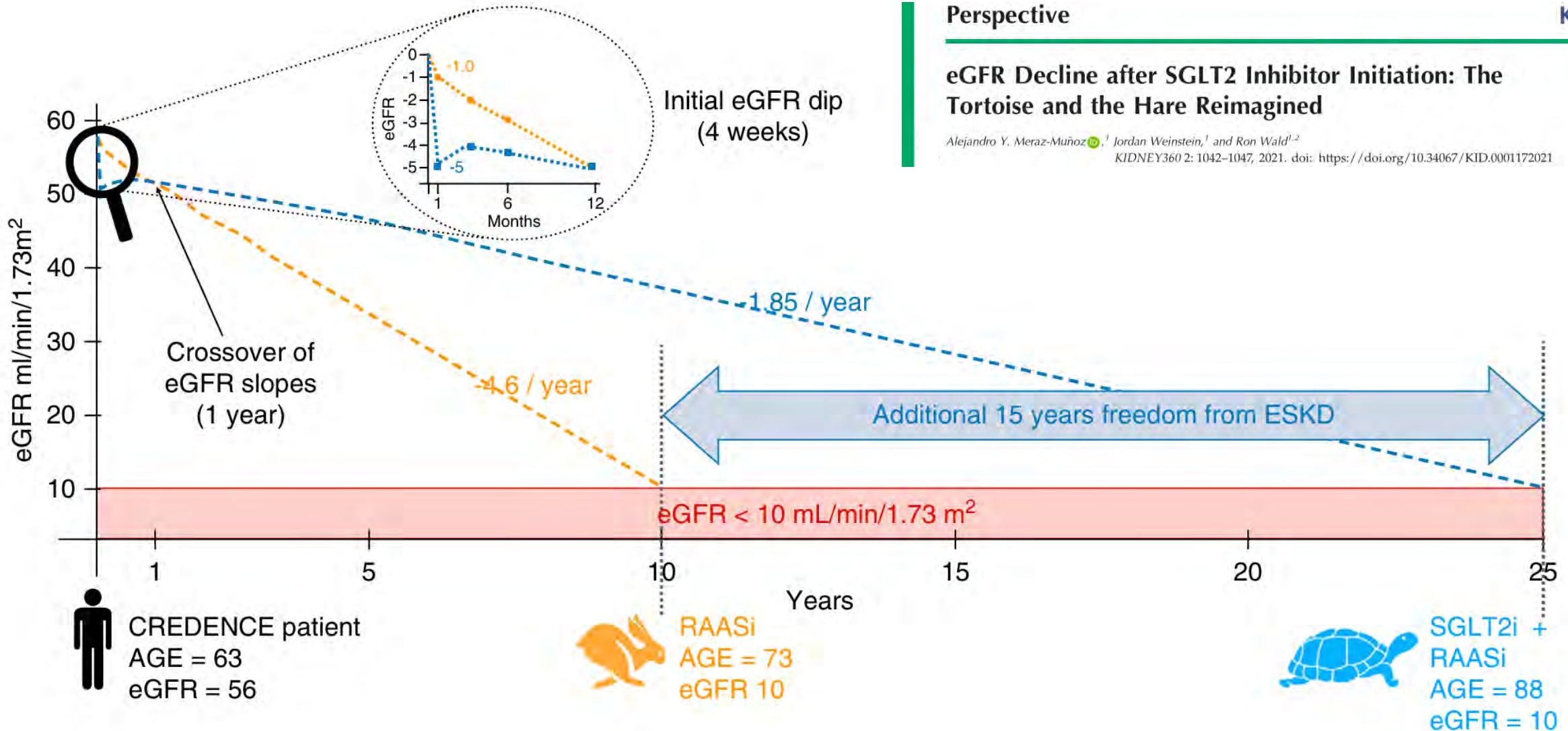
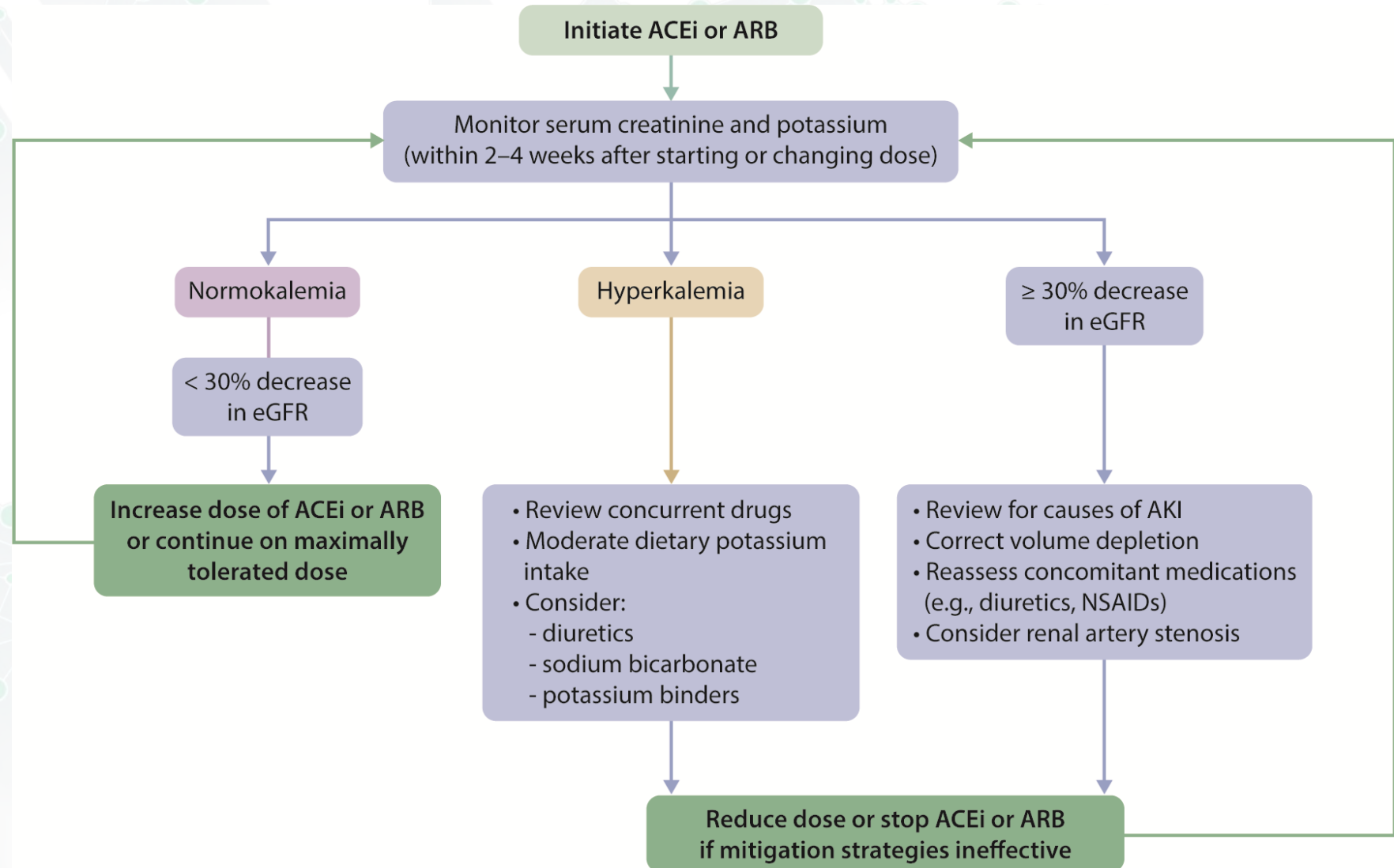


Figure 1. | SGLT2is may delay ESKD by 15 years. A typical patient included in CREDESCENCE would lose 4.6 ml/min per year of eGFR if treated with RAASi only, reaching ESKD in 10 years. However, if canagliflozin is added to his treatment, he would only lose 1.85 ml/min per year of eGFR, delaying ESKD by 15 years. RAASi, renin-angiotensin-aldosterone system inhibitors; SGLT2i, sodium-glucose cotransporter 2 inhibitor.

MANAGEMENT – ACUTE CHANGES IN eGFR

Initial dips in eGFR are expected following initiation of hemodynamically active therapies, including both RASI and SGLT2i.

GFR reductions of $\geq 30\%$ from baseline exceed the expected variability and warrant evaluation.



MANAGEMENT – CARDIOVASCULAR DISEASE AND IMAGING

Estimate 10-year cardiovascular risk using a validated risk tool that incorporates CKD to guide treatment for prevention of cardiovascular disease.

CKD is not a contraindication to an invasive strategy for people with acute or unstable heart disease.

Imaging studies are not necessarily contraindicated in people with CKD and the risks and benefits should be determined on an individual basis.

Strategies to mitigate risks from imaging studies using contrast media are easily

Overall eGFRcr	ACR (mg/g)				
	<10	10–29	30–299	300–999	1000+
105+	1.6	2.2	2.9	4.3	5.8
90–104	Ref	1.3	1.8	2.6	3.1
60–89	1.0	1.3	1.7	2.2	2.8
45–59	1.3	1.6	2.0	2.4	3.1
30–44	1.8	2.0	2.5	3.2	3.9
15–29	2.8	2.8	3.3	4.1	5.6
<15	4.6	5.0	5.3	6.0	7.0

All-cause mortality: 82 cohorts
Study size = 26,444,384; events = 2,604,028

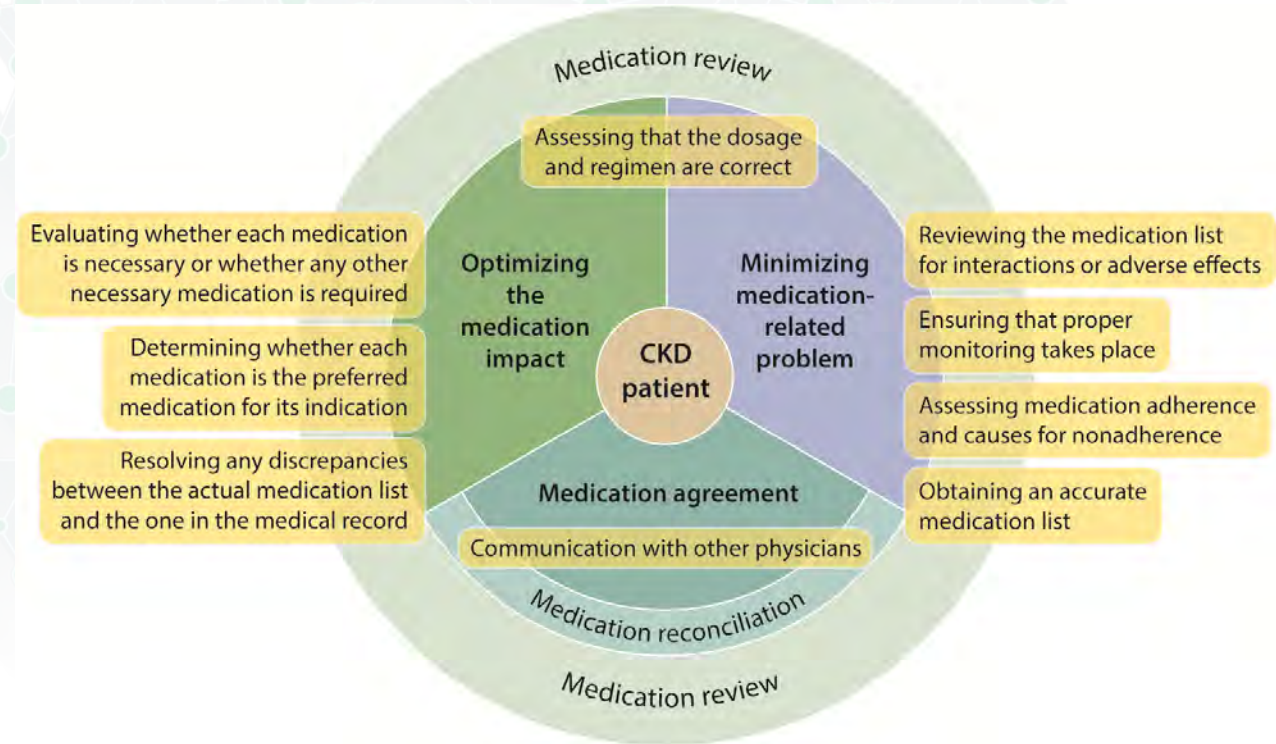
Overall eGFRcr	ACR (mg/g)				
	<10	10–29	30–299	300–999	1000+
105+	1.4	2.0	3.0	4.1	5.4
90–104	Ref	1.3	1.9	2.7	3.6
60–89	1.0	1.4	1.7	2.4	3.2
45–59	1.4	1.7	2.2	2.8	3.8
30–44	2.0	2.3	2.8	3.7	4.6
15–29	3.2	3.1	3.5	5.0	6.5
<15	6.1	6.4	6.4	7.3	8.2

Cardiovascular mortality: 76 cohorts
Study size = 26,022,346; events = 776,441






MANAGEMENT – PERFORM THOROUGH MEDICATION REVIEW

Perform thorough medication review periodically and at transitions of care to assess adherence, continued indication, and potential drug interactions because people with CKD often have complex medication regimens and are seen by multiple specialists.

Review and limit the use of over-the-counter medicines, dietary, or herbal remedies that may be harmful for people with CKD. For most people and clinical settings, validated eGFR equations using SCr are appropriate for drug-dosing. Remember, a validated measured GFR is most accurate.



Common Medications with Documented Nephrotoxicity and Non-Nephrotoxic Alternatives

	Medication	Nephrotoxic mechanism/effect	Alternative
 Analgesics	Nonsteroidal anti-inflammatory drug (NSAIDs)	<p>GFR through reduction in prostaglandin-dependent kidney blood flow</p> <p>Allergic interstitial nephritis (AIN)</p> <p>Nephrotic syndrome</p>	Acetaminophen
 Antimicrobials	<p>Aminoglycosides</p> <p>Vancomycin</p> <p>Sulfamethoxazole-trimethoprim</p>	<p>Accumulates in the proximal tubular cells resulting in acute tubular necrosis (ATN)</p> <p>Unclear cause, but likely ATN and possible AIN</p> <p>AIN, ATN, crystalluria within distal convoluted tubule and reversible inhibition of tubular creatinine secretion</p>	<p>Cephalosporins and carbapenems</p> <p>Linezolid and daptomycin</p> <p>Clindamycin + primaquine, pentamidine, atovaquone</p>
 Gastrointestinal medications	Proton pump inhibitors	May result in AKI and CKD due to tubulointerstitial nephritis and AIN	H2-receptor antagonists
 Cardiovascular medications	Warfarin	Glomerular hemorrhage, kidney tubular damage, and direct effects on kidney vascular calcification	Non-vitamin K antagonist oral anticoagulants
 Other	Lithium	Nephrogenic diabetes insipidus as well as CKD from chronic tubulointerstitial nephropathy	Aripiprazole, lamotrigine, quetiapine, valproate

Medication Management in CKD



Medication choices



Dose adjustment by GFR



Polypharmacy



Imaging studies



When prescribing nephrotoxic medications always consider benefits versus harms



Consider GFR when dosing medications cleared by the kidneys



Perform medication review periodically to assess adherence, continued indication/ potential drug interactions



Consider indication for imaging studies in accordance with general population indications



Monitor eGFR, electrolytes, and therapeutic medication levels whenever indicated



Validated eGFR equations using SCr are appropriate for drug dosing



If medications are discontinued during an acute illness, communicate a clear plan of when to restart



Assess the risk for AKI in people with CKD receiving intra-arterial contrast using validated tools



Limit the use of over-the-counter medicines and dietary/herbal remedies



In people with extremes of body weight, eGFR nonindexed for BSA may be indicated



Consider planned discontinuation of medications (such as metformin, ACEi, ARBs, and SGLT2i) 48–72 hours prior to elective surgery



Intravenous administration of radiocontrast media can be managed as per consensus statements from the radiology societies in people with AKI or GFR <60 ml/min per 1.73 m²



Review Teratogenic potential while prescribing medications to people with CKD of child bearing age



Adapt drug dosing in people where GFR, non-GFR determinants of the filtration markers are not in a steady state

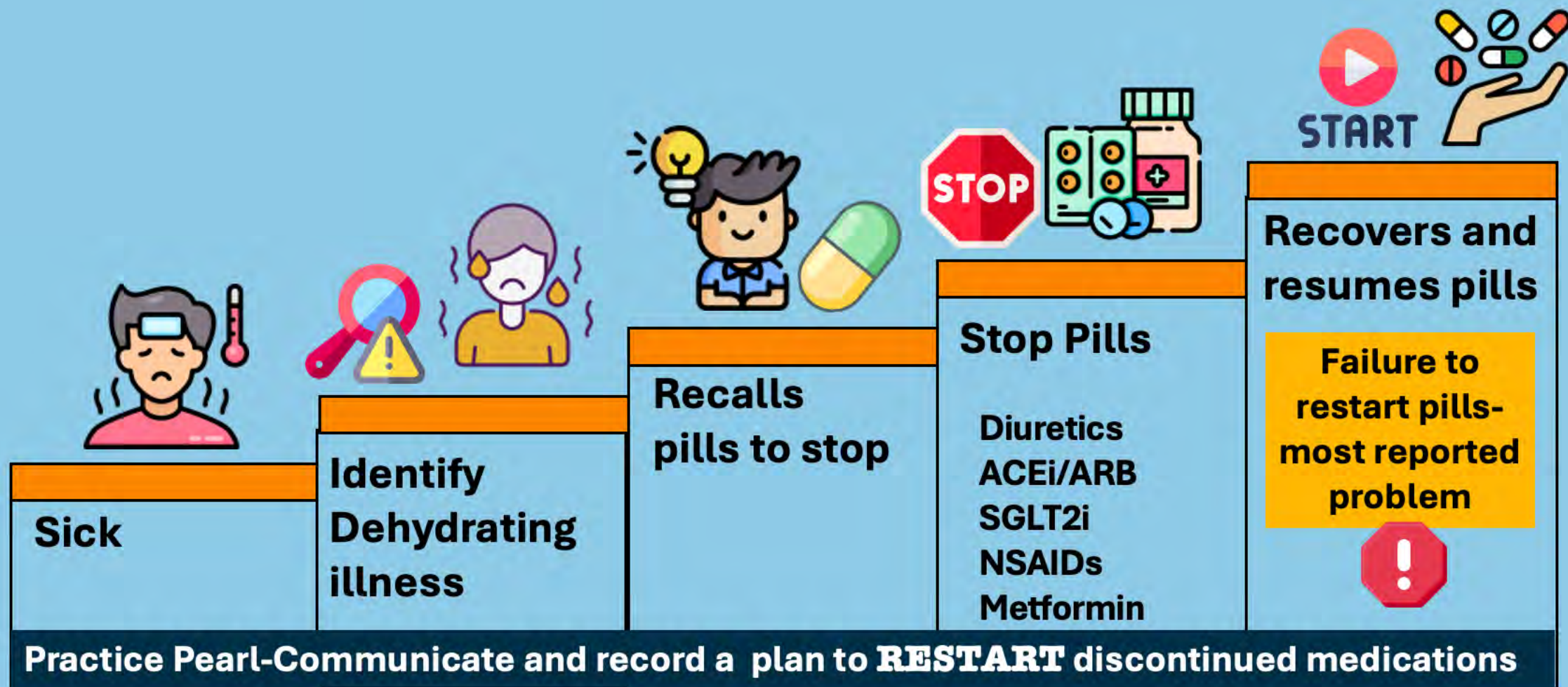


Preferentially use group II and III gadolinium-based contrast agents people with GFR <30 ml/min per 1.73 m²

Sick Day Medication Rule in CKD



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MANAGEMENT – DISCONTINUATION AND RESTART OF MEDICATIONS

If medications are discontinued during an acute illness, communicate a clear plan of when to restart the discontinued medications to the affected person and healthcare providers, and ensure documentation in the medical record.

Failure to restart these medications may lead to unintentional harm.



Stopping renin-angiotensin system inhibitors in patients with advanced CKD and risk of adverse outcomes: a nationwide cohort study

JASN

JOURNAL OF THE AMERICAN SOCIETY OF NEPHROLOGY

METHODS



10.254 patients with eGFR<30 ml/min/1.73 m²



Stopping within 6 months vs. continuing RASi



5 year follow-up

Analytical approach:

Target trial emulation based on cloning, censoring and weighting

OUTCOME

5-year risk	Continuing	Stopping	Difference
 Mortality	40.9% (38.9, 42.8)	54.5% (48.5, 61.2)	13.6% (7.0, 20.3)
 MACE	47.6% (45.9, 49.4)	59.5% (53.8, 66.1)	11.9% (5.7, 18.6)
 KRT	36.1% (34.7, 37.7)	27.9% (23.5, 32.5)	-8.3% (-12.8, -3.6)

Conclusion

In this nationwide study of people with advanced CKD, stopping RASi was associated with a higher absolute risk of mortality and MACE, but a lower absolute risk of KRT.

doi: 10.1681/ASN.2020050682

Does restarting Renin-Angiotensin Inhibitors improve clinical outcomes in those who have previously discontinued it ?

Target Trial Emulation Study



Osaka Consortium for Kidney Disease Research (OCKR)



eGFR 10-60 mL/min/1.73 m²



2005-2021



Discontinued Renin-Angiotensin System inhibitors (RASi)



n=6065



66 years Mean



62% Male



eGFR 40 mL/min/1.73 m²



37% Restarted RASi within 1 year

Patients were followed up for a maximum of 5 years after RASi discontinuation

Restarting RASi within a year after Discontinuation (versus not restarting RASi)



PRIMARY OUTCOME

Composite outcome (initiation of KRT, ≥50% decline in eGFR, or kidney failure)

0.85

(95% CI 0.78-0.93)



SECONDARY OUTCOME

All-cause death

0.70

(95% CI 0.61-0.80)



Incidence of hyperkalemia (between the 2 strategies)

HR 1.11

(95% CI 0.96-1.27)

* KRT, Kidney Replacement Therapy; Kidney Failure, eGFR <5 mL/min/1.73 m²

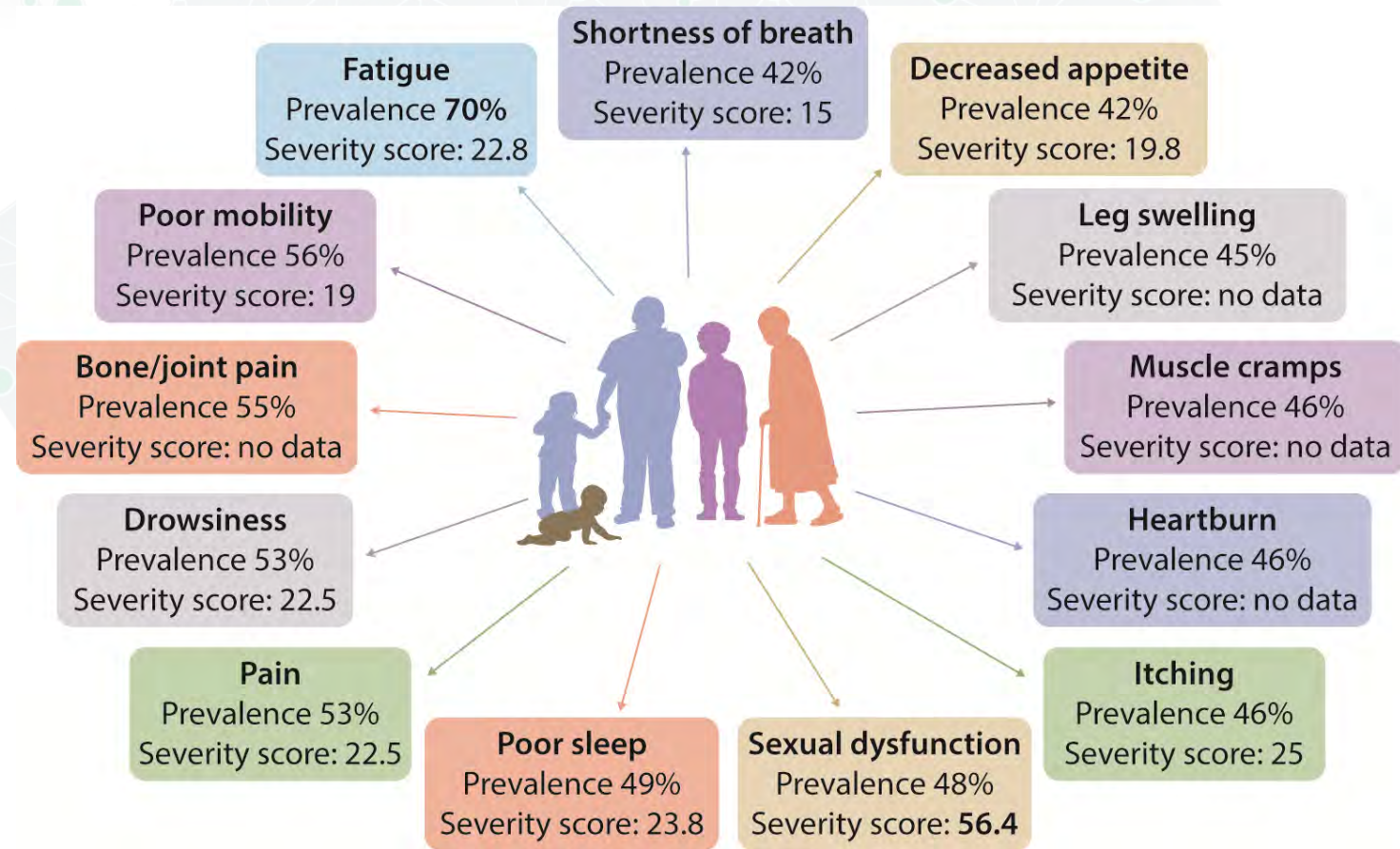
Conclusions: Restarting RASi after discontinuation was associated with a lower risk of kidney outcomes and mortality but not related to the incidence of hyperkalemia.

Koki Hattori, Tatsufumi Oka, Yuta Asahina, et al. *Estimated Effect of Restarting Renin-Angiotensin Inhibitors after Discontinuation on kidney Outcomes and Mortality.* J Am Soc Nephrol
Visual Abstract by Edgar Lerma, MD, FASN

MANAGEMENT – SYMPTOM CONTROL IN CKD

The identification and assessment of symptoms in people with progressive CKD is important for highlighting changes in clinical management, redirecting treatment toward patient-centered management, and may lead to discussion about appropriate supportive care options.

Effective communication and shared decision-making should be key principles between healthcare providers and the people they treat, allowing them to work in partnership to identify symptom burden, possible treatment strategies and person-centered solutions.



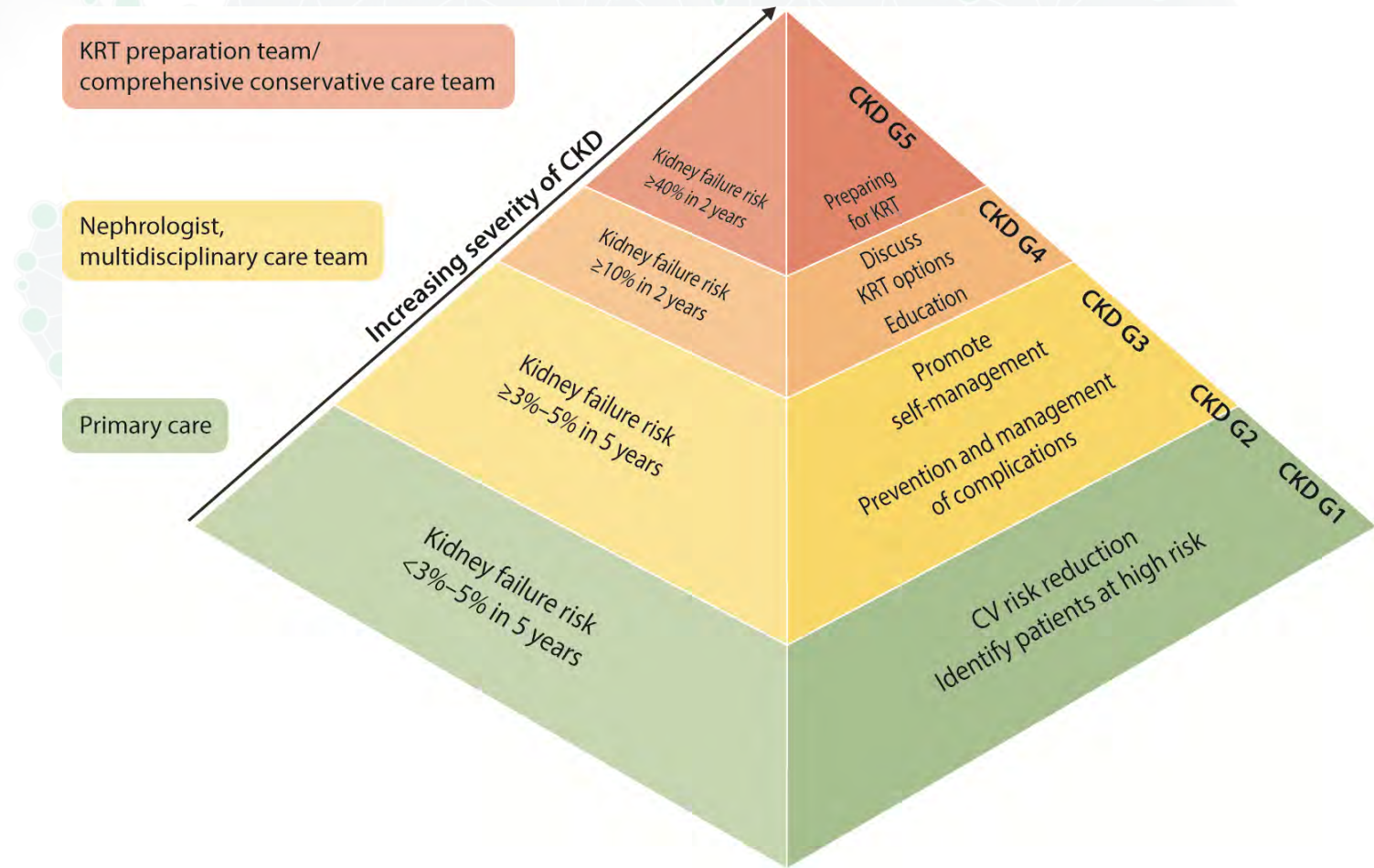
Symptom Management in CKD

Pain	Sleep issue	Restless leg	Depression	Pruritis	Anorexia
<p>Physiotherapy exercise/ massage therapy Acupuncture</p>	<p>Sleep hygiene, exercise, optimal positioning when sleeping/removal of stimulants/CBT</p>	<p>Correction of factors such as hyperphosphatemia and iron deficiency anemia</p>	<p>Exercise/ acupuncture/ CBT</p>	<p>Acupuncture UV-B therapy</p>	<p>Exercise Address contributing factors Dietary assessment</p>
<p>Analgesic ladder taking CKD into account</p>	<p>Melatonin/simple sedative</p>	<p>Trials with levodopa, nonergot dopamine antagonists/ low- dose gabapentinoids</p>	<p>TCA/SRIs/SNRI/ atypical antidepressant</p>	<p>Gabapentinoids Topical- capsicum Rehydrating emollients</p>	<p>No data to support appetite stimulants in people with CKD not on KRT</p>

MANAGEMENT – ADVANCED CARE PLANNING

Plans addressing future health care states should be jointly agreed with people with CKD and their families/carers and known to all.

Advanced care planning for those choosing supportive care is particularly important.





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Rowan Zyada.



Nikhil Elenjickal



Francisco Santamaria



Oscar R. Durón Vargas



Medhavi Gautam



THANK YOU