ENFERMEDAD RENAL CRÓNICA

GUÍAS KDIGO 2024

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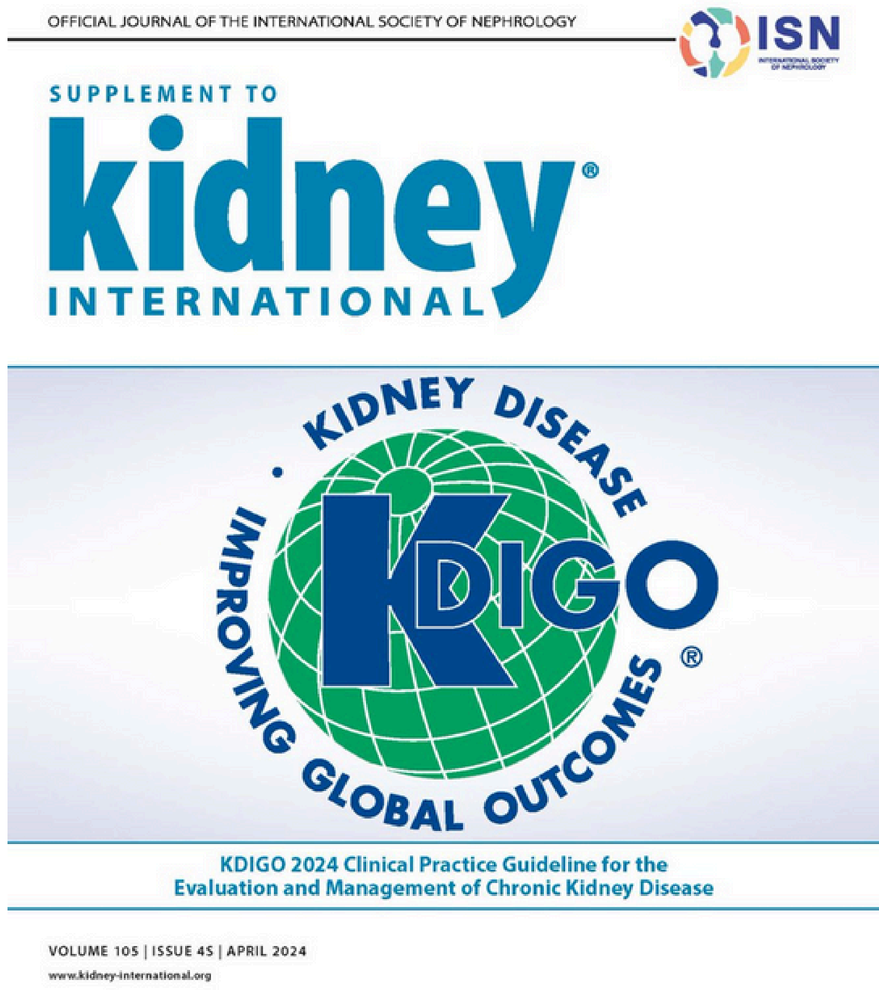
MIR 4º año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KDIGO®

KDIGO 2024 Clinical Practice Guideline for the
Evaluation and Management of Chronic Kidney Disease

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- CLASIFICACIÓN
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- TRATAMIENTO
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DEFINICIÓN

La ERC se define como el daño estructural o funcional del riñón mantenido más de 3 meses:

MARCADORES DAÑO RENAL

- **Albuminuria (albumina/creatinina > 30 mg/g [>3 mg/mmol])**
- **Alteraciones en el sedimento urinario**
- **Hematuria**
- **Alteraciones tubulares con anormalidades electrolíticas**
- **Alteraciones histológicas**
- **Alteraciones estructurales detectadas con imagen (poliquistosis renal..)**
- **Antecedentes de transplante renal**

DISMINUCIÓN FGE

FGE < 60 mL/min/1.73m2

Prevalencia global de ERC 9,1%
¡Conciencia de enfermedad < 10 %!

DETECCIÓN ERC

CRIBADO A **PERSONAS DE RIESGO**

Hipertensión arterial
Diabetes
Enfermedad CV
Obesidad
Antecedentes de IRA
Antecedentes familiares
Comorbilidades
(nefrotóxicos, lupus...)

Medir:
1. Tas
2. Coc
3. Otr

Revisión de **mediciones previas alteradas**

Hallazgos de imagen como la reducción del tamaño renal o el grosor cortical

Hallazgos histológicos de fibrosis y atrofia

Condiciones clínicas que lo sugieran (HTA o DM de larga evolución mal controlados)

FGE < 60 mL/min/1.73 m2 +/- CAC > 30 mg/g +/- presenci
de otros marcadores de daño renal **durante 3 meses**
evidencia de cronicidad

Clasificación
Establecer la causa
Calcular riesgo de progresión
Iniciar tratamiento

0 mL/min/1.73 m2 +/- CAC mg/g agudo

Daño renal agudo

CLASIFICACIÓN

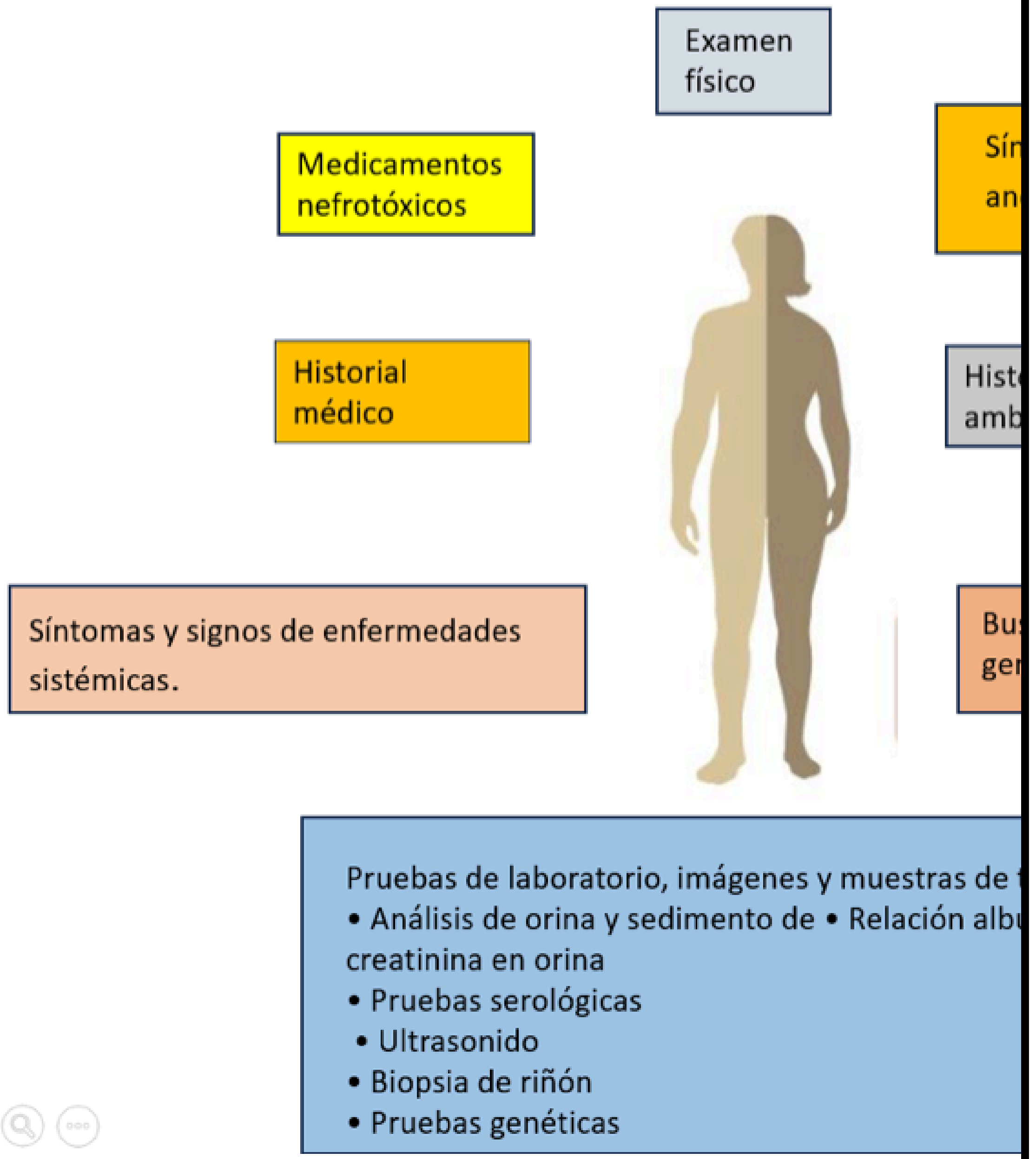
Sistema CGA:

- Causa
- FGE
- Albuminuria

Esta clasificación determina la **severidad y el riesgo** de:

PROGRESIÓN
EVENTOS CV
MORTALIDAD

CAUSAS



Examen físico

Medicamentos nefrotóxicos

Sín
an

Historial médico

Hist
amb

Síntomas y signos de enfermedades sistémicas.

Bus
ger

Pruebas de laboratorio, imágenes y muestras de t
- Análisis de orina y sedimento de • Relación albu creatinina en orina
- Pruebas serológicas
- Ultrasonido
- Biopsia de riñón
- Pruebas genéticas

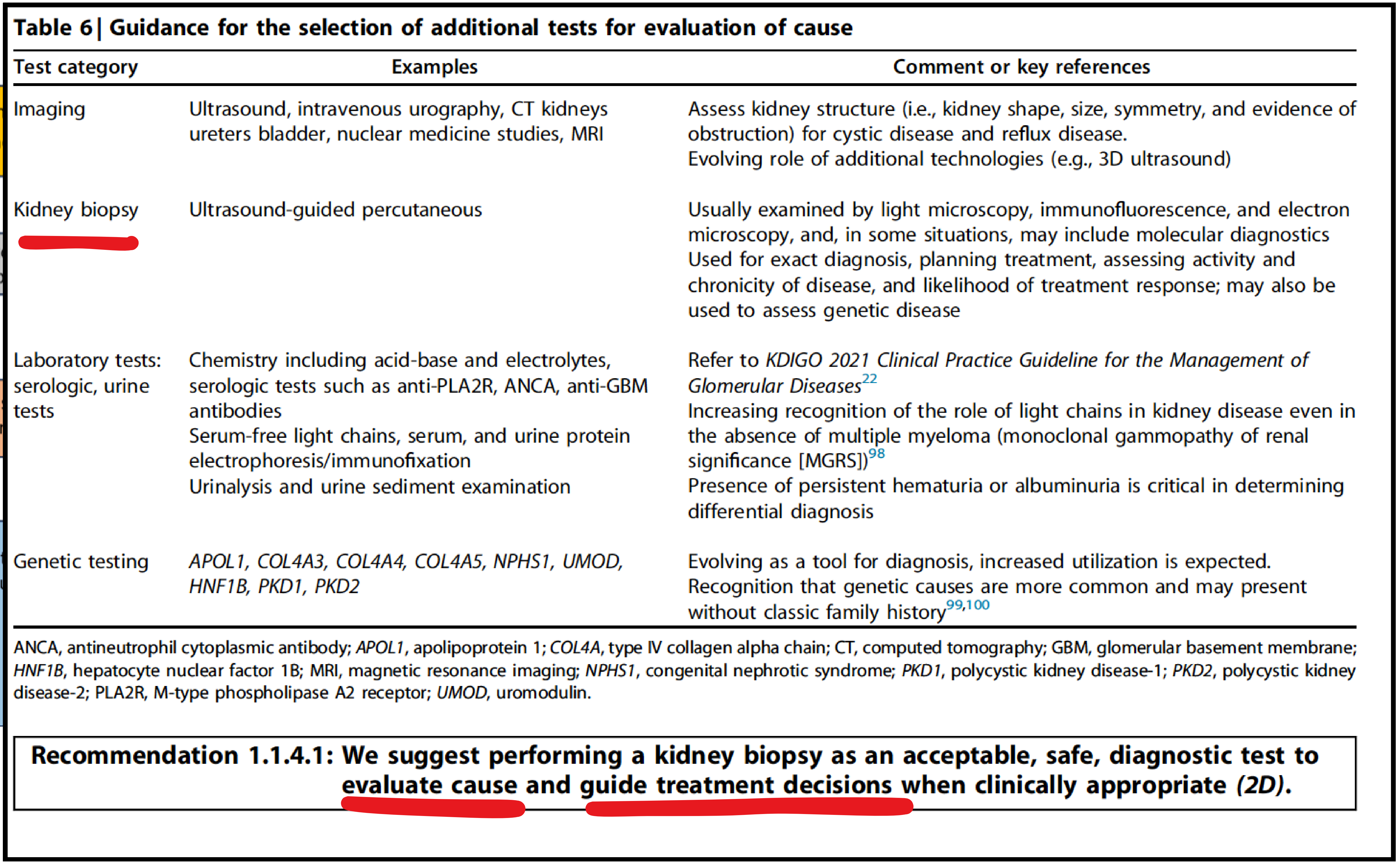


Table 6| Guidance for the selection of additional tests for evaluation of cause

Test category	Examples	Comment or key references
Imaging	Ultrasound, intravenous urography, CT kidneys ureters bladder, nuclear medicine studies, MRI	Assess kidney structure (i.e., kidney shape, size, symmetry, and evidence of obstruction) for cystic disease and reflux disease. Evolving role of additional technologies (e.g., 3D ultrasound)
Kidney biopsy	Ultrasound-guided percutaneous	Usually examined by light microscopy, immunofluorescence, and electron microscopy, and, in some situations, may include molecular diagnostics Used for exact diagnosis, planning treatment, assessing activity and chronicity of disease, and likelihood of treatment response; may also be used to assess genetic disease
Laboratory tests: serologic, urine tests	Chemistry including acid-base and electrolytes, serologic tests such as anti-PLA2R, ANCA, anti-GBM antibodies Serum-free light chains, serum, and urine protein electrophoresis/immunofixation Urinalysis and urine sediment examination	Refer to *KDIGO 2021 Clinical Practice Guideline for the Management of Glomerular Diseases*[22] Increasing recognition of the role of light chains in kidney disease even in the absence of multiple myeloma (monoclonal gammopathy of renal significance [MGRS])[98] Presence of persistent hematuria or albuminuria is critical in determining differential diagnosis
Genetic testing	*APOL1*, *COL4A3*, *COL4A4*, *COL4A5*, *NPHS1*, *UMOD*, *HNF1B*, *PKD1*, *PKD2*	Evolving as a tool for diagnosis, increased utilization is expected. Recognition that genetic causes are more common and may present without classic family history[99,100]

ANCA, antineutrophil cytoplasmic antibody; *APOL1*, apolipoprotein 1; *COL4A*, type IV collagen alpha chain; CT, computed tomography; GBM, glomerular basement membrane; *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.

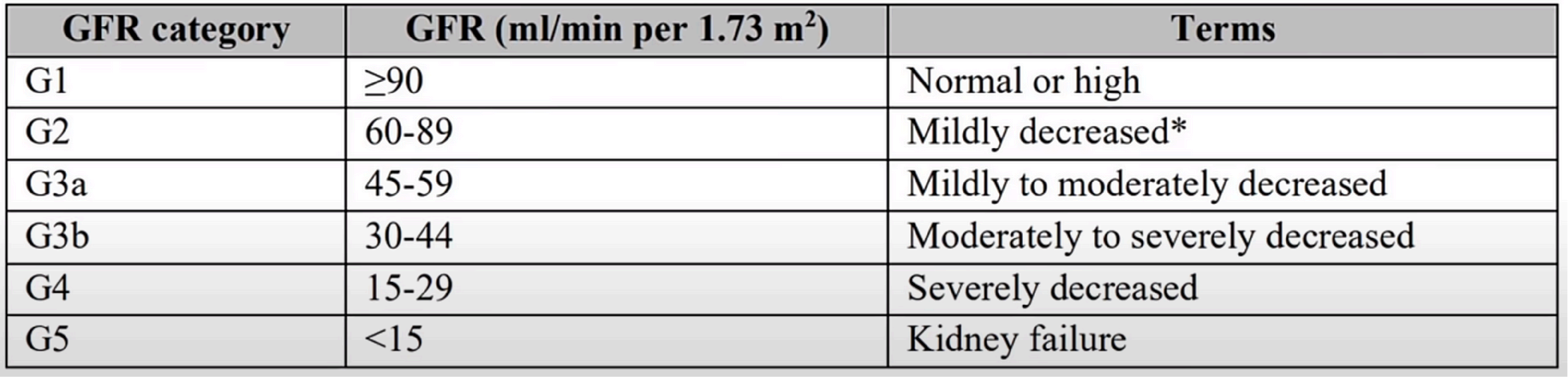
Recommendation 1.1.4.1: We suggest performing a kidney biopsy as an acceptable, safe, diagnostic test to evaluate cause and guide treatment decisions when clinically appropriate *(2D)*.

CAUSAS

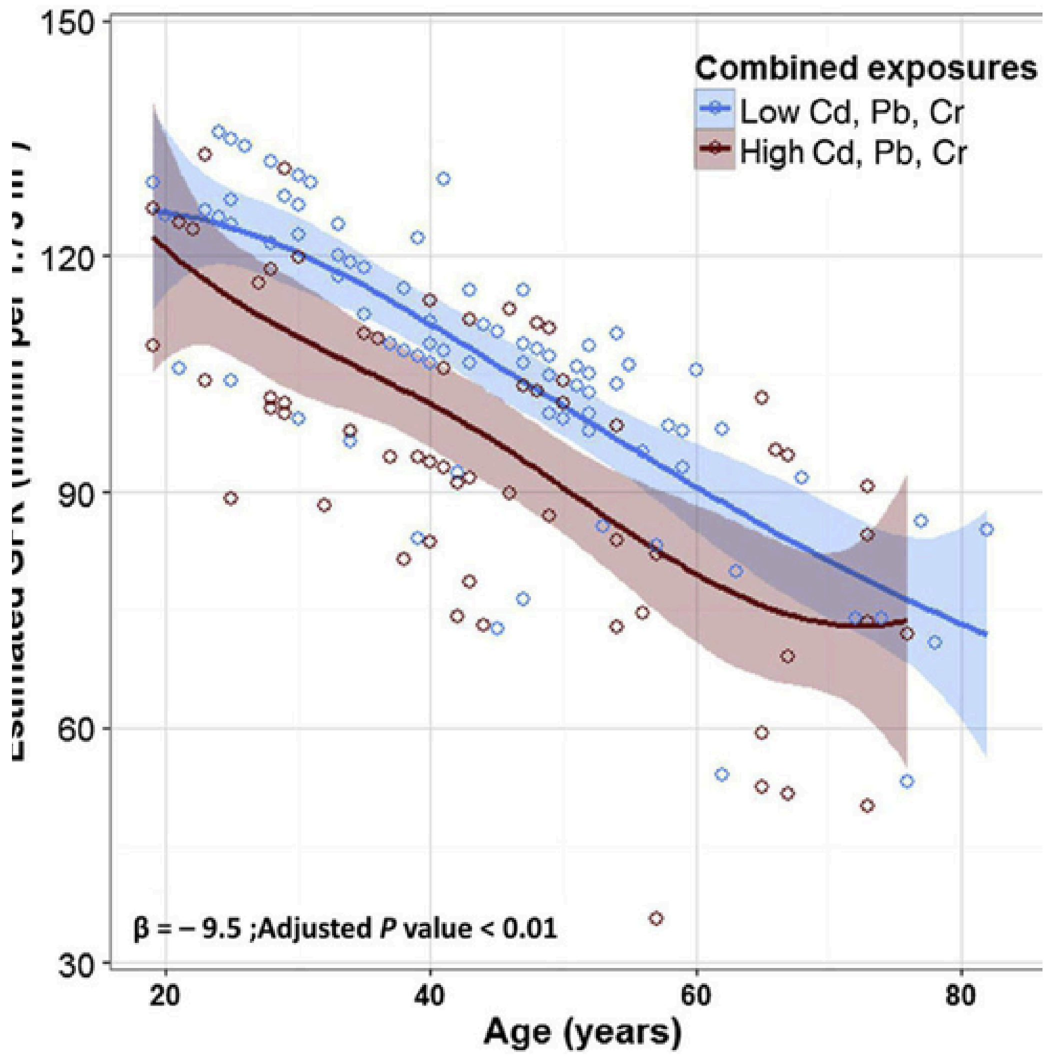
50%

ENFERMEDAD GLOMERULAR	Diabetes, enfermedades autoinmunes, medicaciones, neoplasias (incluye amiloidosis),...
ENFERMEDAD TUBULOINTERSTICIAL	Infecciones sistémicas, enfermedades autoinmunes, sarcoidosis, medicaciones, neoplasias (mieloma),...
ENFERMEDAD VASCULAR	Ateroesclerosis, hipertensión, isquemia, vasculitis,...
ENFERMEDAD QUÍSTICA CONGÉNITA	Enfermedad renal poliquística, enfermedad de Fabry, síndrome de Alport...

EVALUACIÓN DE TFG



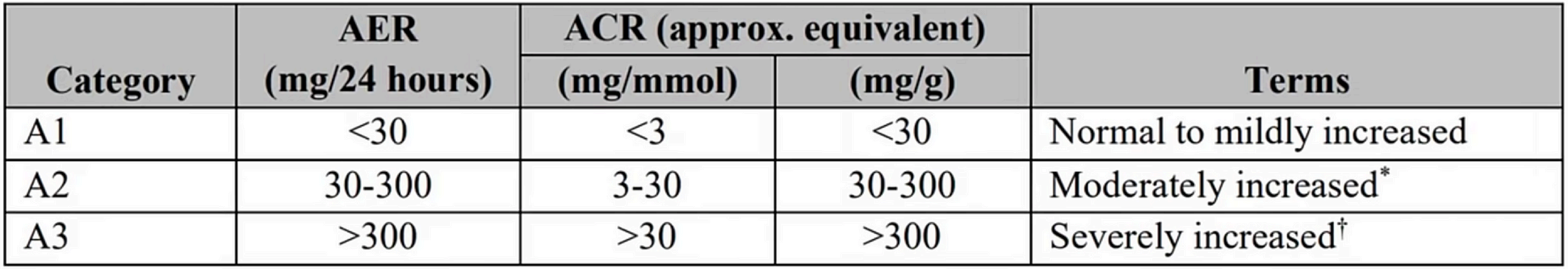
GFR category	GFR (ml/min per 1.73 m^2)	Terms
G1	≥90	Normal or high
G2	60-89	Mildly decreased*
G3a	45-59	Mildly to moderately decreased
G3b	30-44	Moderately to severely decreased
G4	15-29	Severely decreased
G5	<15	Kidney failure



FG ≤ 60 ML/MIN/1.73 M2

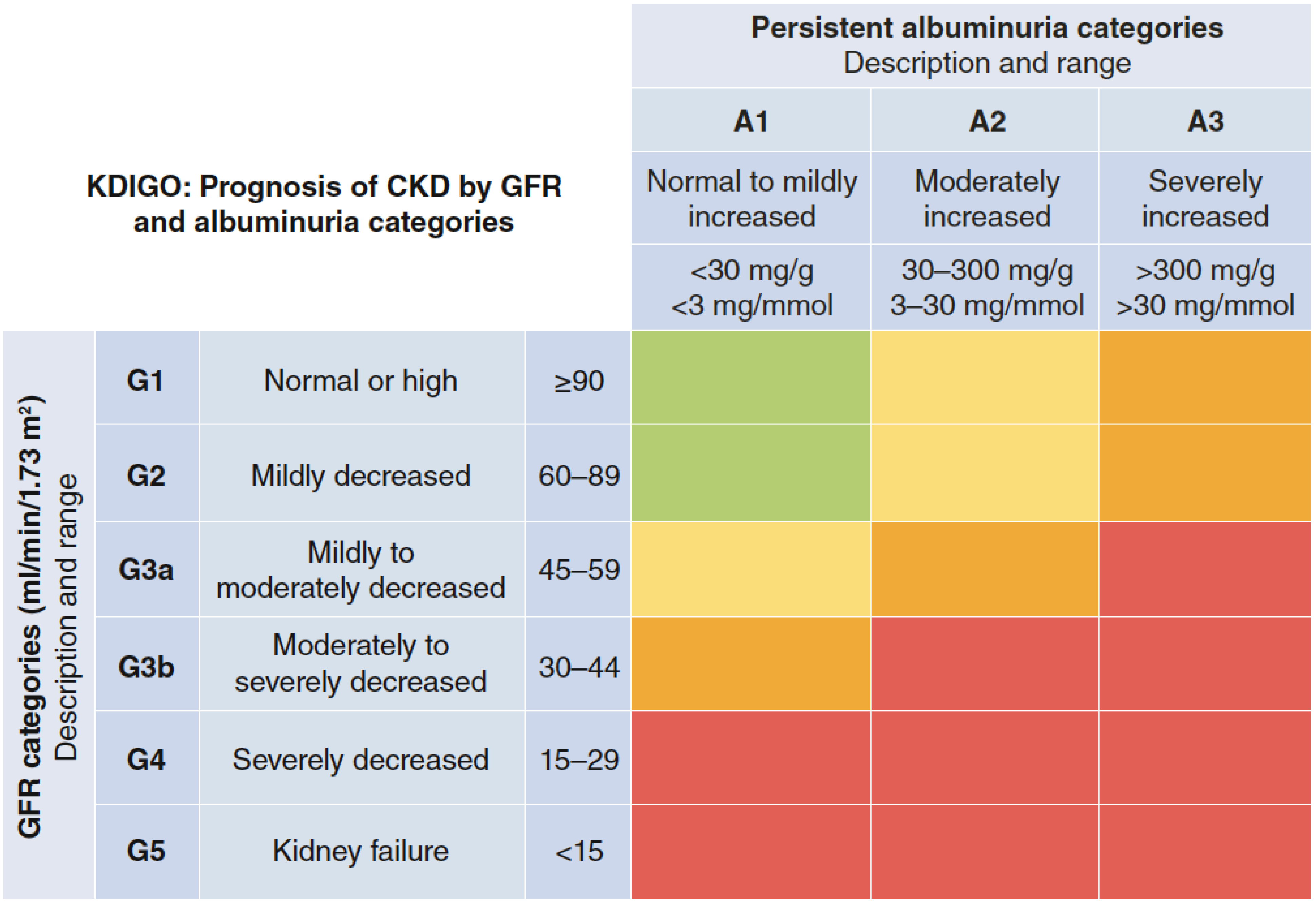
CAÍDA FGE DE 0.6 - 0.8 ML AÑO

EVALUACIÓN DE ALBUMINURIA



Category	AER (mg/24 hours)	ACR (approx. equivalent)		Terms
		(mg/mmol)	(mg/g)	
A1	<30	<3	<30	Normal to mildly increased
A2	30-300	3-30	30-300	Moderately increased[*]
A3	>300	>30	>300	Severely increased[†]

EVALUAR EL RIESGO



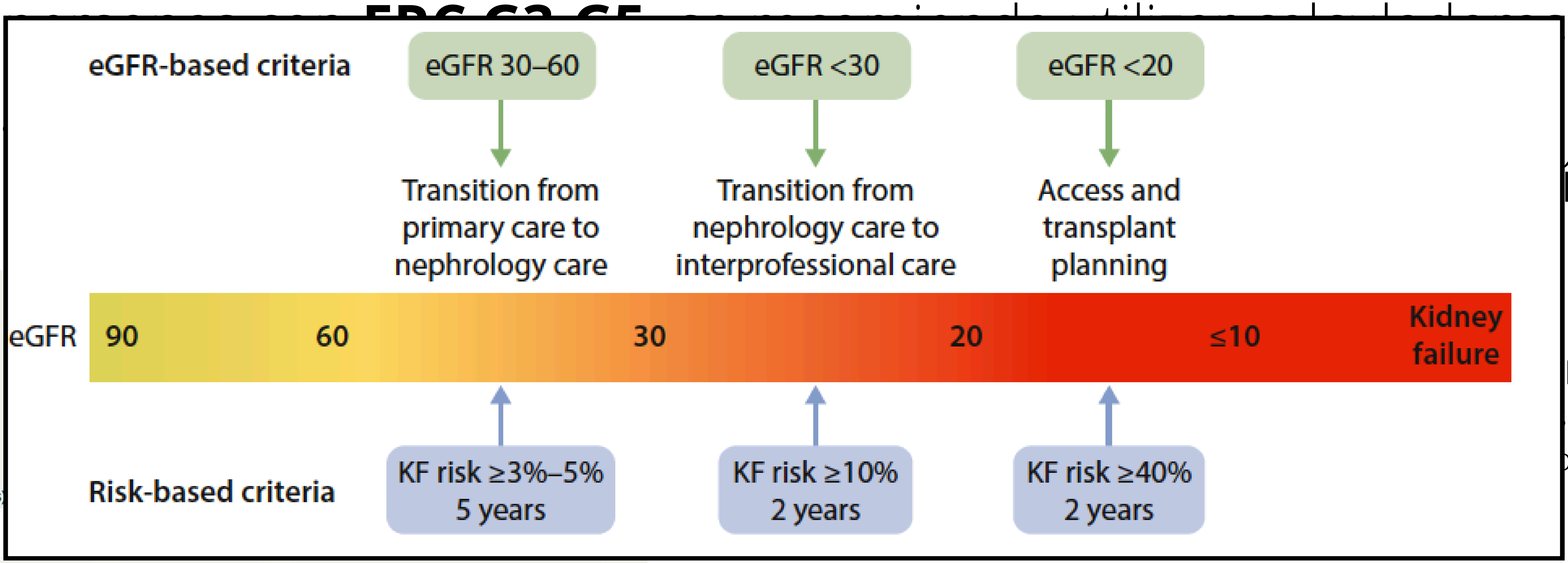
KDIGO: Prognosis of CKD by GFR and albuminuria categories				Persistent albuminuria categories Description and range		
				A1	**A2**	**A3**
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–300 mg/g 3–30 mg/mmol	>300 mg/g >30 mg/mmol
GFR categories (ml/min/1.73 m^2) Description and range	**G1**	Normal or high	≥90	🟩	🟨	🟧
	G2	Mildly decreased	60–89	🟩	🟨	🟧
	G3a	Mildly to moderately decreased	45–59	🟨	🟧	🟥
	G3b	Moderately to severely decreased	30–44	🟧	🟥	🟥
	G4	Severely decreased	15–29	🟥	🟥	🟥
	G5	Kidney failure	<15	🟥	🟥	🟥

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.

- **PROGRESIÓN**
- **EVENTOS CV**
- **MORTALIDAD**

EVALUAR EL RIESGO

- El riesgo se aproxima con el mapa pero no es absoluto.
- En de
  ries
  - ía IgA)...



eGFR-based criteria

eGFR 30–60 → Transition from primary care to nephrology care

eGFR <30 → Transition from nephrology care to interprofessional care

eGFR <20 → Access and transplant planning

eGFR: 90 | 60 | 30 | 20 | ≤10 | Kidney failure

Risk-based criteria

KF risk ≥3%–5% 5 years

KF risk ≥10% 2 years

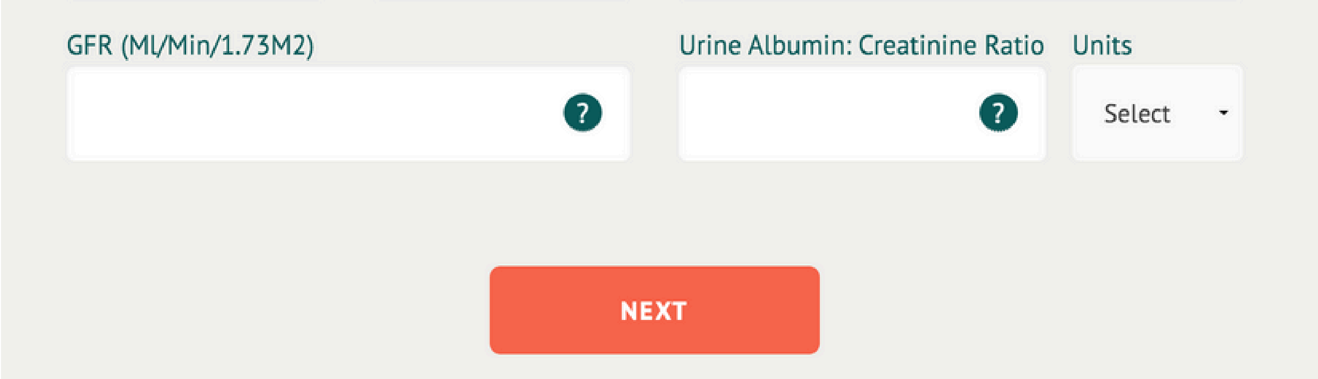
KF risk ≥40% 2 years

logo.

ciplinario.

ara trasplante y

Age (Yrs)



GFR (Ml/Min/1.73M2)

Urine Albumin: Creatinine Ratio

Units

Select

RETRASO DE LA PROGRESIÓN

MANEJO NO FARMACOLÓGICO

CESE TABÁQUICO

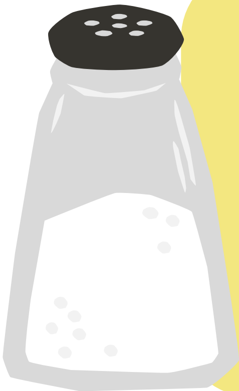
NUTRIPLATO

DIETA

Dieta variada y saludable
Aumentar consumo de alimentos de origen vegetal
Evitar ultraprocesados

EJERCICIO FÍSICO

150 minutos a la semana



Ingesta de proteinas de 0,8 g/kg/día

*No restringir en niños, pacientes frágiles, sarcopénicos..

Ingesta de sodio <2g (<5g sal)

MANEJO FARMACOLÓGICO

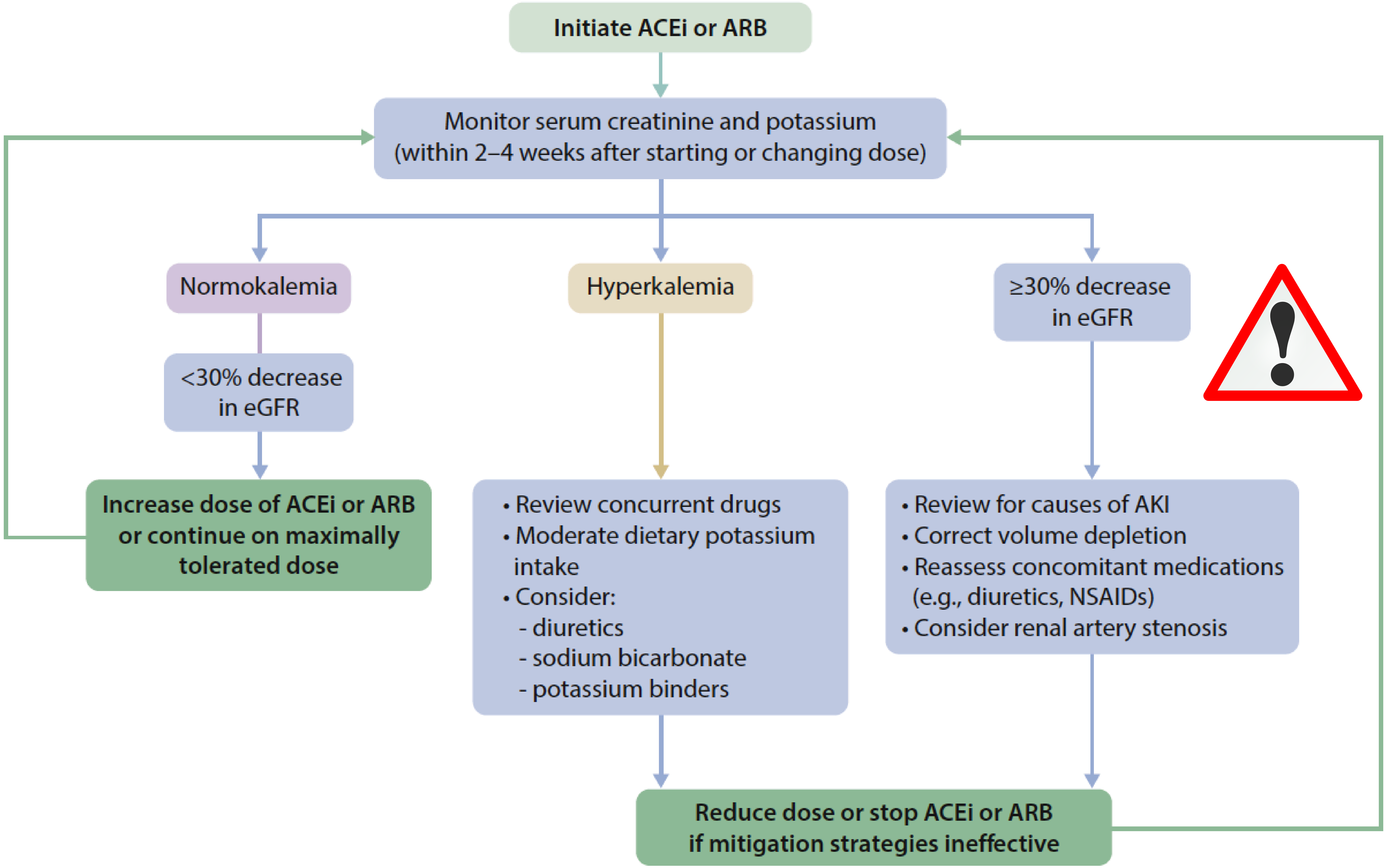
Inhibidores del sistema renina-angiotensina (IECA ó ARA II)

FGE > 15

- **Todo paciente con albuminuria A3 (1B)**
- **Todo paciente con albuminuria A2 + diabetes (1B)**
    - ○ **Considerar en albuminuria A2 sin diabetes (2C)**

Considerar en albuminuria A1 para indicaciones específicas (HTA, IC FEVI disminuida)

- **Utilizar la dosis más alta tolerada**
- **Control de TA, creatinina sérica y potasio en 2-4 semanas**



Initiate ACEi or ARB

Monitor serum creatinine and potassium (within 2–4 weeks after starting or changing dose)

Normokalemia

<30% decrease in eGFR

Increase dose of ACEi or ARB or continue on maximally tolerated dose

Hyperkalemia

- Review concurrent drugs
- Moderate dietary potassium intake
- Consider:
  - diuretics
  - sodium bicarbonate
  - potassium binders

≥30% decrease in eGFR

- Review for causes of AKI
- Correct volume depletion
- Reassess concomitant medications (e.g., diuretics, NSAIDs)
- Consider renal artery stenosis

Reduce dose or stop ACEi or ARB if mitigation strategies ineffective

MANEJO FARMACOLÓGICO

Inhibidores del cotransportador sodio-glucosa-2 (SGLT2i)

FGE > 20

- Todo paciente con ERC + diabetes (1A)
- Todo paciente con ERC + IC (1A)
- Todo paciente con CAC > 200mg/g (1A)
  - Considerar en FGE 20-45 sin albuminuria (2B)

- Continuar si FGe desciende < 20
- Suspender en situaciones de riesgo de cetosis (ayuno, cirugía..)

MANEJO FARMACOLÓGICO

Antagonistas de los receptores de mineralocorticoides (ARM)

Finerenona

FGE > 25

- **Todo paciente con ERC + diabetes + albuminuria A2,A3 + K normal** a pesar de dosis máxima tolerada de IECA/ARAII (2A)

- **¡Vigilar K!**
  - **Suspender si K > 5,5 mmol/l**
- **Se pueden combinar con SGLT2i**

MANEJO DE COMPLICACIONES

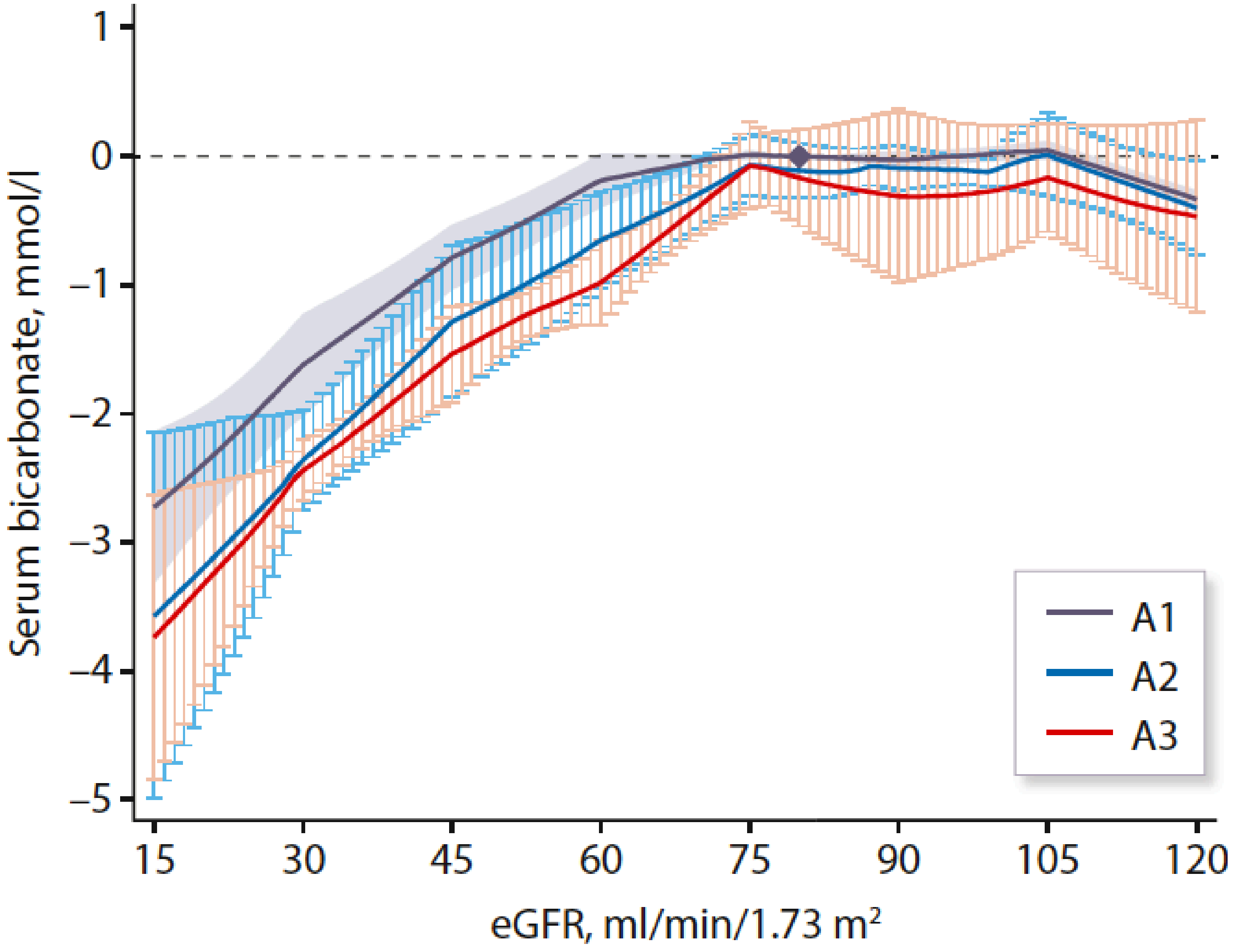
ACIDOSIS METABÓLICA

PREVALENCIA

G3: 7,7 %
G5: 38,3%

BICARBONATO

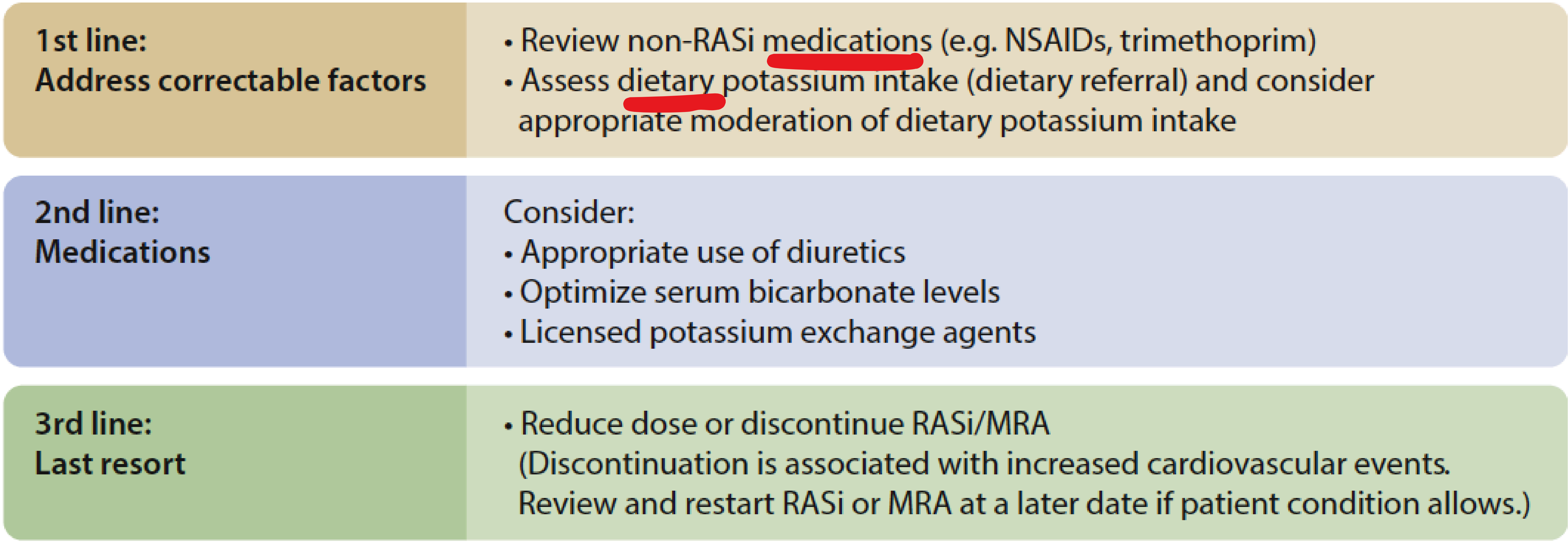
- No hay clara evidencia de su beneficio
- Considerar si HCO3 < 18 mmol/l



HIPERPOTASEMIA

PREVALENCIA

G3: 5-9%.
G5: 24-34%



1st line: **Address correctable factors**	• Review non-RASi medications (e.g. NSAIDs, trimethoprim) • Assess dietary potassium intake (dietary referral) and consider appropriate moderation of dietary potassium intake
2nd line: **Medications**	Consider: • Appropriate use of diuretics • Optimize serum bicarbonate levels • Licensed potassium exchange agents
3rd line: **Last resort**	• Reduce dose or discontinue RASi/MRA (Discontinuation is associated with increased cardiovascular events. Review and restart RASi or MRA at a later date if patient condition allows.)

Figure 32 | Actions to manage hyperkalemia (potassium >5.5 mmol/l) in chronic kidney disease. MRA, mineralocorticoid receptor antagonists; NSAID, nonsteroidal anti-inflammatory drug; RASi, renin-angiotensin system inhibitors.

HIPERPOTASEMIA

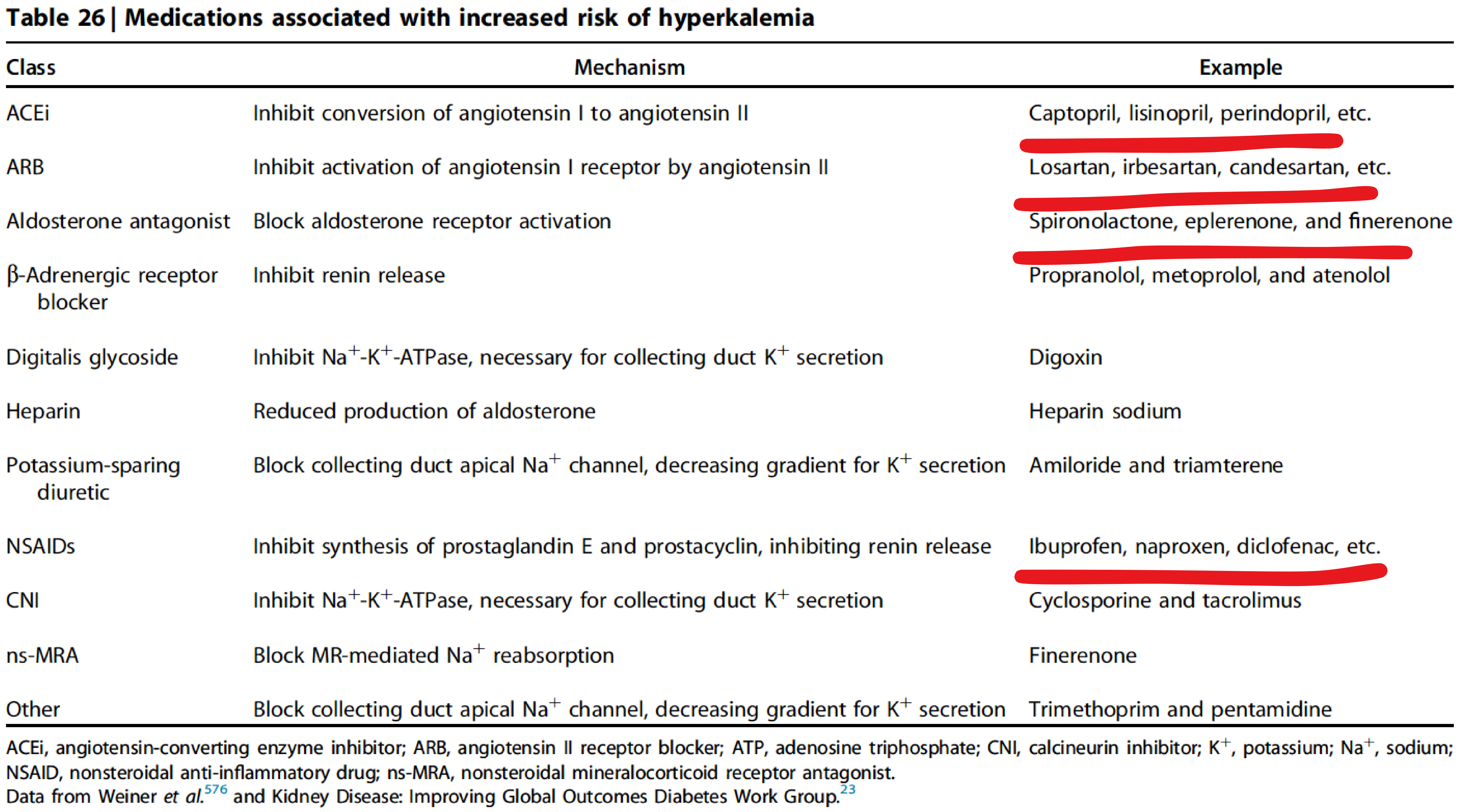
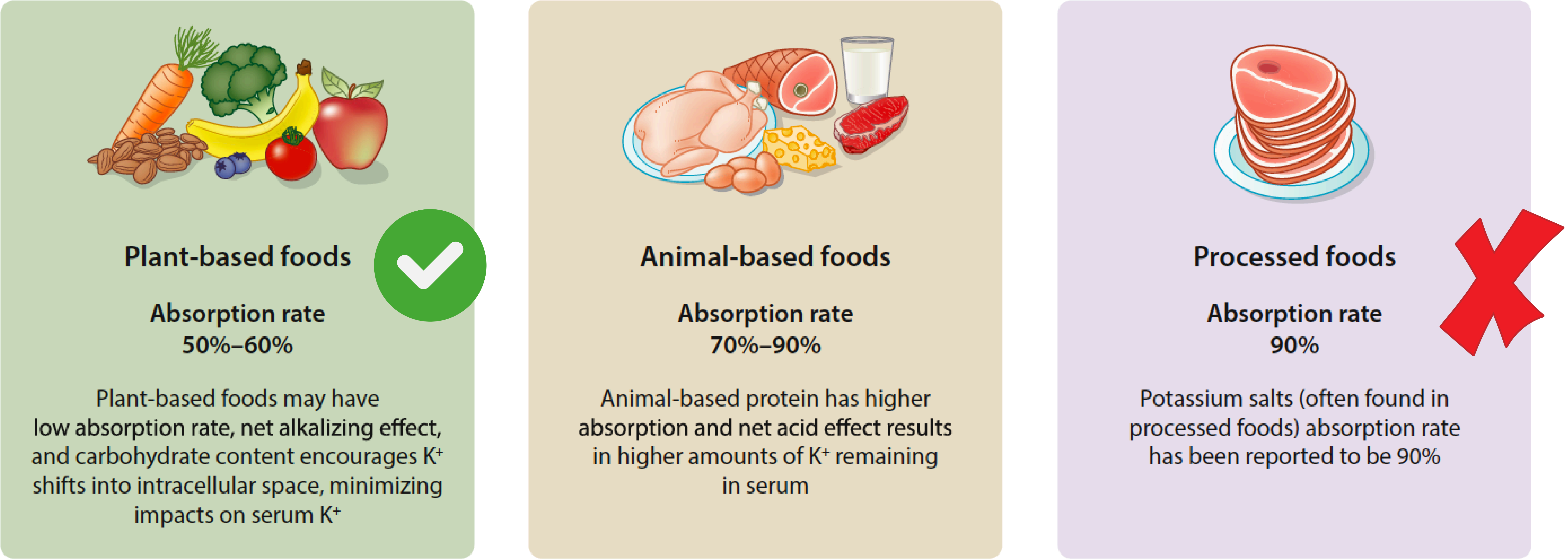


Table 26 | Medications associated with increased risk of hyperkalemia

Class	Mechanism	Example
ACEi	Inhibit conversion of angiotensin I to angiotensin II	Captopril, lisinopril, perindopril, etc.
ARB	Inhibit activation of angiotensin I receptor by angiotensin II	Losartan, irbesartan, candesartan, etc.
Aldosterone antagonist	Block aldosterone receptor activation	Spironolactone, eplerenone, and finerenone
β-Adrenergic receptor blocker	Inhibit renin release	Propranolol, metoprolol, and atenolol
Digitalis glycoside	Inhibit Na$^+$-K$^+$-ATPase, necessary for collecting duct K$^+$ secretion	Digoxin
Heparin	Reduced production of aldosterone	Heparin sodium
Potassium-sparing diuretic	Block collecting duct apical Na$^+$ channel, decreasing gradient for K$^+$ secretion	Amiloride and triamterene
NSAIDs	Inhibit synthesis of prostaglandin E and prostacyclin, inhibiting renin release	Ibuprofen, naproxen, diclofenac, etc.
CNI	Inhibit Na$^+$-K$^+$-ATPase, necessary for collecting duct K$^+$ secretion	Cyclosporine and tacrolimus
ns-MRA	Block MR-mediated Na$^+$ reabsorption	Finerenone
Other	Block collecting duct apical Na$^+$ channel, decreasing gradient for K$^+$ secretion	Trimethoprim and pentamidine

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; ATP, adenosine triphosphate; CNI, calcineurin inhibitor; K$^+$, potassium; Na$^+$, sodium; NSAID, nonsteroidal anti-inflammatory drug; ns-MRA, nonsteroidal mineralocorticoid receptor antagonist.
Data from Weiner *et al.*[576] and Kidney Disease: Improving Global Outcomes Diabetes Work Group.[23]

HIPERPOTASEMIA



Plant-based foods ✓

Absorption rate
50%–60%

Plant-based foods may have low absorption rate, net alkalizing effect, and carbohydrate content encourages K^+ shifts into intracellular space, minimizing impacts on serum K^+

Animal-based foods

Absorption rate
70%–90%

Animal-based protein has higher absorption and net acid effect results in higher amounts of K^+ remaining in serum

Processed foods ✗

Absorption rate
90%

Potassium salts (often found in processed foods) absorption rate has been reported to be 90%

Figure 33 | Potassium absorption rates of plant-based, animal-based, and processed foods. Data from Picard K, Griffiths M, Mager DR, Richard C. Handouts for low-potassium diets disproportionately restrict fruits and vegetables. *J Ren Nutr*. 2021;31:210–214.[592]

ANEMIA

PREVALENCIA

G3: 14,9 %
G5: 60,7 %

- **No** atribuir a ERC. Hacer un **adecuado abordaje diagnóstico**.
- Si finalmente es anemia renal, valorar Eritropoyetina (si Hb<10 g/dL)

ENFERMEDAD MINERAL-ÓSEA

- Alteraciones en fósforo, calcio, PTH, vitamina D y FGF23.
- Condiciona múltiples enfermedades óseas.

HIPERURICEMIA

- Hiperuricemia sintomática (tras primer episodio de gota): tratamiento preferiblemente con Alopurinol.
- Evitar AINES; mejor Colchicina o corticoides.

ENFERMEDAD CARDIOVASCULAR

- La **enfermedad c** **mortalidad** en los pacientes con
- Objetivo de PAS <

Pacientes con ERC

ERC sin diabetes o EA

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a/

Riesgo alto
LDL < 70

Riesgo
muy alto
LDL < 55

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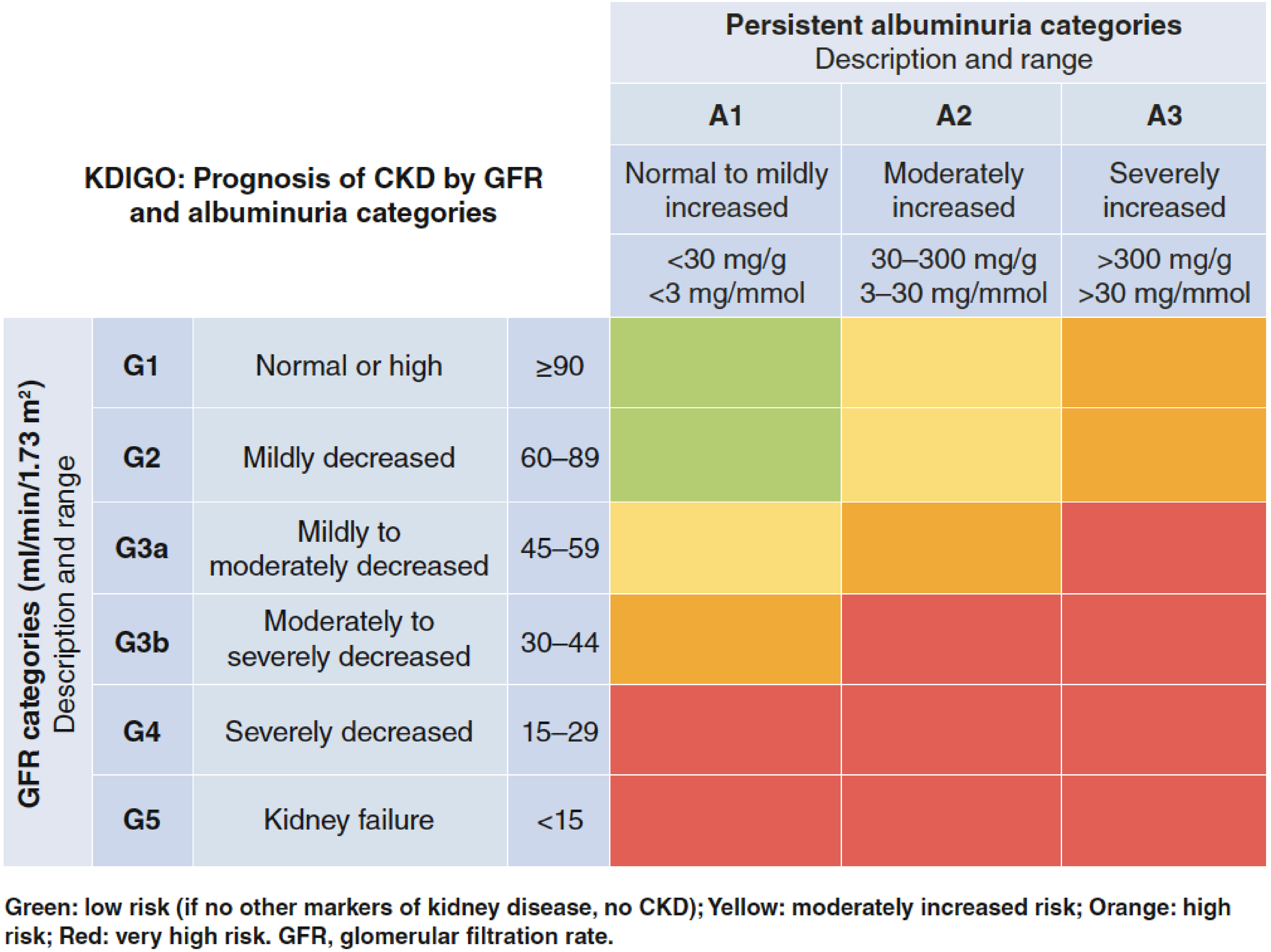

KDIGO: Prognosis of CKD by GFR and albuminuria categories

				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 m²) Description and range	**G1**	Normal or high	≥90	🟩	🟨	🟧
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	G3a	Mildly to moderately decreased	45–59	🟨	🟧	🟥
	G3b	Moderately to severely decreased	30–44	🟧	🟥	🟥
	G4	Severely decreased	15–29	🟥	🟥	🟥
	G5	Kidney failure	<15	🟥	🟥	🟥

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.

FIBRILACIÓN AURICULAR

- La prevalencia de FA es mayor en ERC. Las complicaciones son mayores (ACV).

- Se recomiendan los **anticoagulantes orales no antagonistas de la vitamina K (ACODs)** frente a los antagonistas de la vitamina K (warfarina).
  - *AJustar dosis por función renal.*

- En diálisis no hay clara evidencia del beneficio de la anticoagulación.

GESTIÓN DE LA MEDICACIÓN Y ADMINISTRACIÓN DE FÁRMACOS

43 %

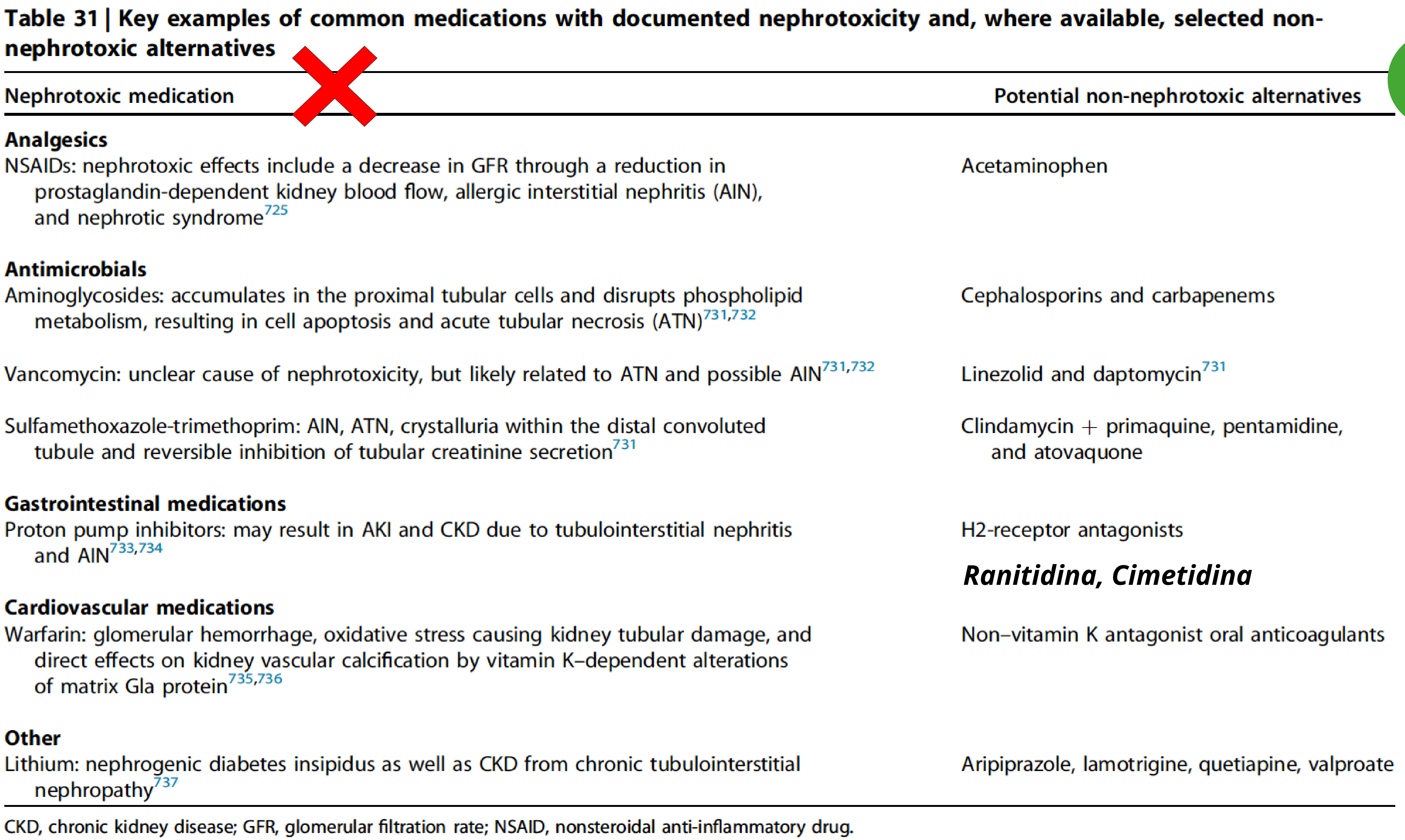
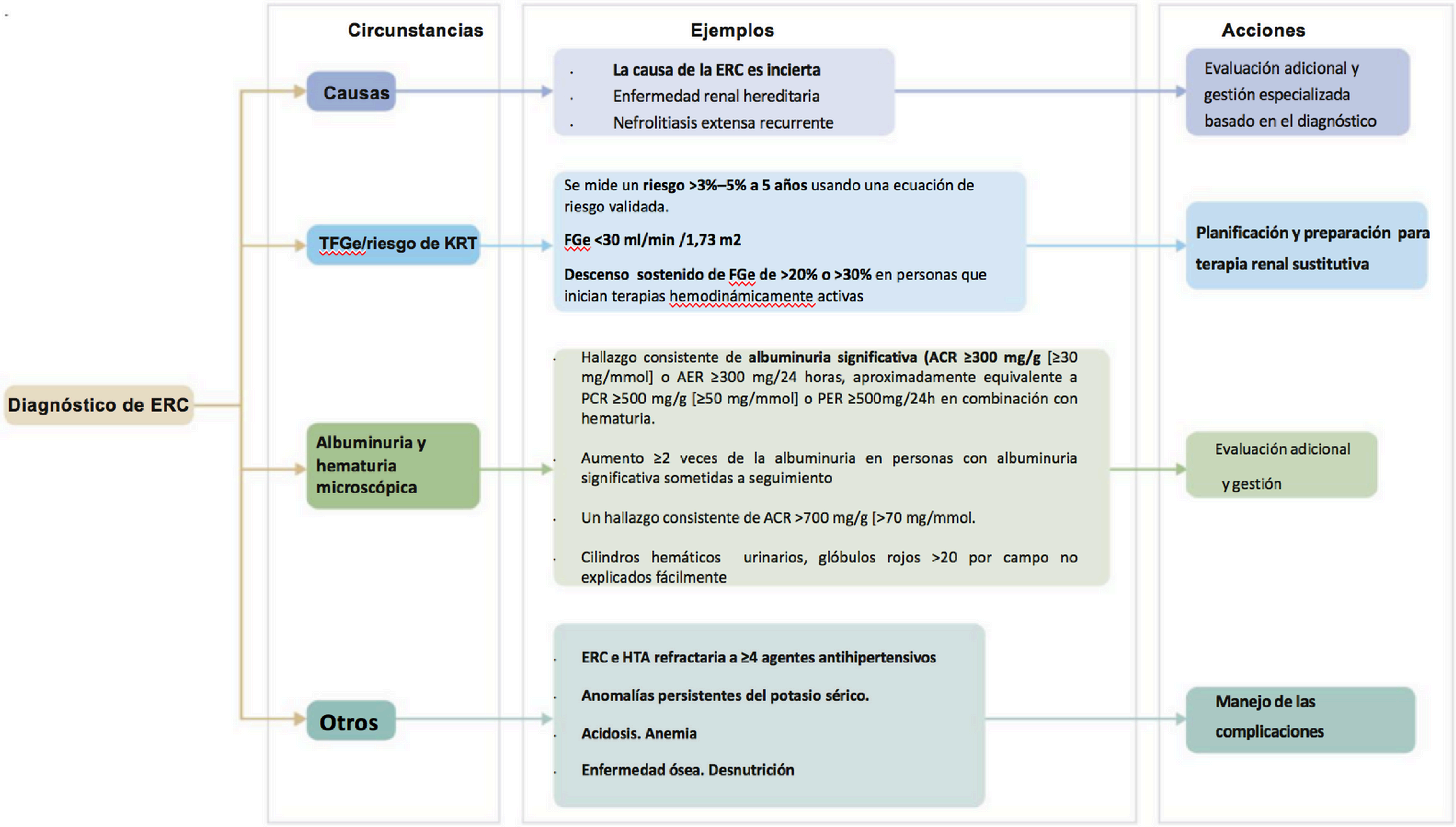


Table 31 | Key examples of common medications with documented nephrotoxicity and, where available, selected non-nephrotoxic alternatives

Nephrotoxic medication	Potential non-nephrotoxic alternatives
Analgesics	
NSAIDs: nephrotoxic effects include a decrease in GFR through a reduction in prostaglandin-dependent kidney blood flow, allergic interstitial nephritis (AIN), and nephrotic syndrome[725]	Acetaminophen
Antimicrobials	
Aminoglycosides: accumulates in the proximal tubular cells and disrupts phospholipid metabolism, resulting in cell apoptosis and acute tubular necrosis (ATN)[731,732]	Cephalosporins and carbapenems
Vancomycin: unclear cause of nephrotoxicity, but likely related to ATN and possible AIN[731,732]	Linezolid and daptomycin[731]
Sulfamethoxazole-trimethoprim: AIN, ATN, crystalluria within the distal convoluted tubule and reversible inhibition of tubular creatinine secretion[731]	Clindamycin + primaquine, pentamidine, and atovaquone
Gastrointestinal medications	
Proton pump inhibitors: may result in AKI and CKD due to tubulointerstitial nephritis and AIN[733,734]	H2-receptor antagonists ***Ranitidina, Cimetidina***
Cardiovascular medications	
Warfarin: glomerular hemorrhage, oxidative stress causing kidney tubular damage, and direct effects on kidney vascular calcification by vitamin K–dependent alterations of matrix Gla protein[735,736]	Non–vitamin K antagonist oral anticoagulants
Other	
Lithium: nephrogenic diabetes insipidus as well as CKD from chronic tubulointerstitial nephropathy[737]	Aripiprazole, lamotrigine, quetiapine, valproate

CKD, chronic kidney disease; GFR, glomerular filtration rate; NSAID, nonsteroidal anti-inflammatory drug.

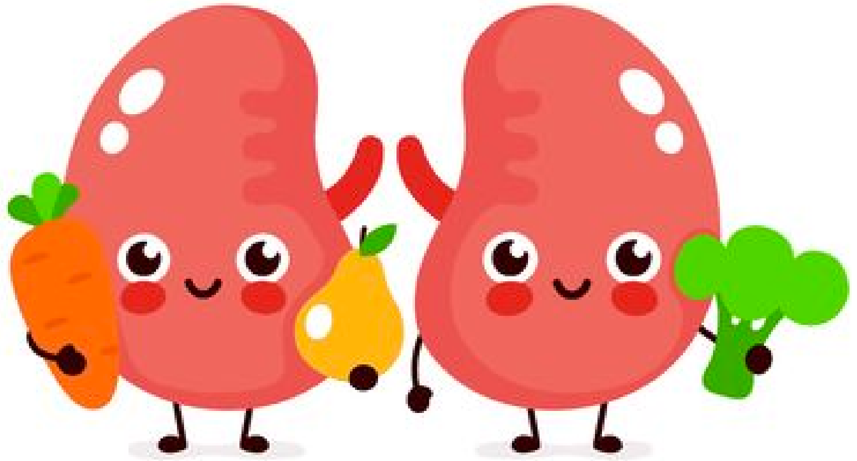
REMITIR A NEFROLOGÍA



Diagnóstico de ERC

Circunstancias	Ejemplos	Acciones
Causas	- **La causa de la ERC es incierta** - Enfermedad renal hereditaria - Nefrolitiasis extensa recurrente	Evaluación adicional y gestión especializada basado en el diagnóstico
TFGe/riesgo de KRT	Se mide un **riesgo >3%–5% a 5 años** usando una ecuación de riesgo validada. **FGe <30 ml/min /1,73 m2** **Descenso sostenido de FGe de >20% o >30%** en personas que inician terapias hemodinámicamente activas	**Planificación y preparación para terapia renal sustitutiva**
Albuminuria y hematuria microscópica	- Hallazgo consistente de **albuminuria significativa (ACR ≥300 mg/g** [≥30 mg/mmol] o AER ≥300 mg/24 horas, aproximadamente equivalente a PCR ≥500 mg/g [≥50 mg/mmol] o PER ≥500mg/24h en combinación con hematuria. - Aumento ≥2 veces de la albuminuria en personas con albuminuria significativa sometidas a seguimiento - Un hallazgo consistente de ACR >700 mg/g [>70 mg/mmol. - Cilindros hemáticos urinarios, glóbulos rojos >20 por campo no explicados fácilmente	Evaluación adicional y gestión
Otros	- **ERC e HTA refractaria a ≥4 agentes antihipertensivos** - **Anomalías persistentes del potasio sérico.** - **Acidosis. Anemia** - **Enfermedad ósea. Desnutrición**	**Manejo de las complicaciones**

CONCLUSIONES



- La ERC es una patología muy prevalente pero silente.
- Es posible tener ERC con una tasa de filtración glomerular normal.
- Importante estimar el riesgo de progresión, eventos CV y mortalidad.
- Tratamiento con IECAS si albuminuria.
- Tratamiento con SGLT2i si diabetes, albuminuria o FG < 45.
- Finerenona como tratamiento novedoso cuando no se consiguen objetivos con IECAS.

BIBLIOGRAFÍA

- KDIGO 2024: Clinical Practice Guideline for the Management of Chronic Kidney Disease

- 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk

¡MUCHAS GRACIAS!