12 jornada actualización redGDPS

Sevilla 25-26 oct 2019

Caída brusca del filtrado glomerular

Antonio Rodríguez

Sumario

1. Caso clínico
2. LRA (AKI) Definición
3. CKD (ERC) Definición
4. **Definición e identificación de la progresión rápida de la ERC (CKD)**
5. Factores que pueden contribuir a la progresión rápida de la ERC

Caso clínico. 1

- Hombre de 62 años sin antecedentes de interés y con hábitos de vida saludables.
- Analíticas y síntomas:
  - 03/2014. Cr: 0,94 mg/dL. GFR: 86,5 ml/min/1.73 m^2
  - O1/2015. Cr: 0,81 mg/dL
  - 19/10/2016. Acude a la consulta por malestar general, astenia, anorexia y edemas en EEII de 10 días de evolución. Orina 1,5 a 2 l/día.

    Analítica: Cr: 1,8 mg/dL. GFR: 38,4 ml/min/1.73 m^2
  - 5/11/2016 Acude a urgencias. Cr: 2,09 mg/dl. Se solicitó consulta urgente con nefrología.

Caso clínico. 2

- Consulta con nefrología (12/11/2016).
- Analítica: Cr: 2,39 mg/dL. GFR: 27,2 ml/min/1.73 m^2. Resto de la analítica normal. Ecografía: Hidronefrosis bilateral con dilatación de las vías excretoras. Se coloca sonda uretral hasta RTU.
- 19/12/2016 . Cr: 1,55 mg/dL. GFR: 46 ml/min/1.73 m^2
- 27/12/2016. Cr: 1,14 mg/dL. GFR: 66,6 ml/min/1.73 m^2

AKI Definition

- AKI is defined as any of the following (Not Graded):
  - Increase in SCr by ≥ 0.3 mg/dl (≥ 26.5 lmol/l) within 48 hours; or
  - Increase in SCr to ≥ 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or
  - Urine volume < 0.5 ml/kg/h for 6 hours.

  The cause of AKI should be determined whenever possible. (Not Graded)

Kidney Disease Improving Global Outcomes (KDIGO) Clinical Practice Guideline for Acute Kidney Injury. Kidney Int Suppl. 2012;2:1-138

Causes of AKI and diagnostic tests

Selected causes of AKI requiring immediate diagnosis and specific therapies	Recommended diagnostic tests
Decreased kidney perfusion	Volume status and urinary diagnostic indices
Acute glomerulonephritis, vasculitis, interstitial nephritis, thrombotic microangiopathy	Urine sediment examination, serologic testing and hematologic testing
Urinary tract obstruction	Kidney ultrasound

Kidney Disease Improving Global Outcomes (KDIGO) Clinical Practice Guideline for Acute Kidney Injury.
Kidney Int Suppl. 2012;2:1-138

IDIAP
Jordi Gol

Proyecto de Investigación

Asociación entre la administración de inhibidores de la bomba de protones y la incidencia de enfermedad renal crónica y lesión renal aguda en población general, con el código P17/114, presentado por el investigador Antonio Rodriguez Poncelas.

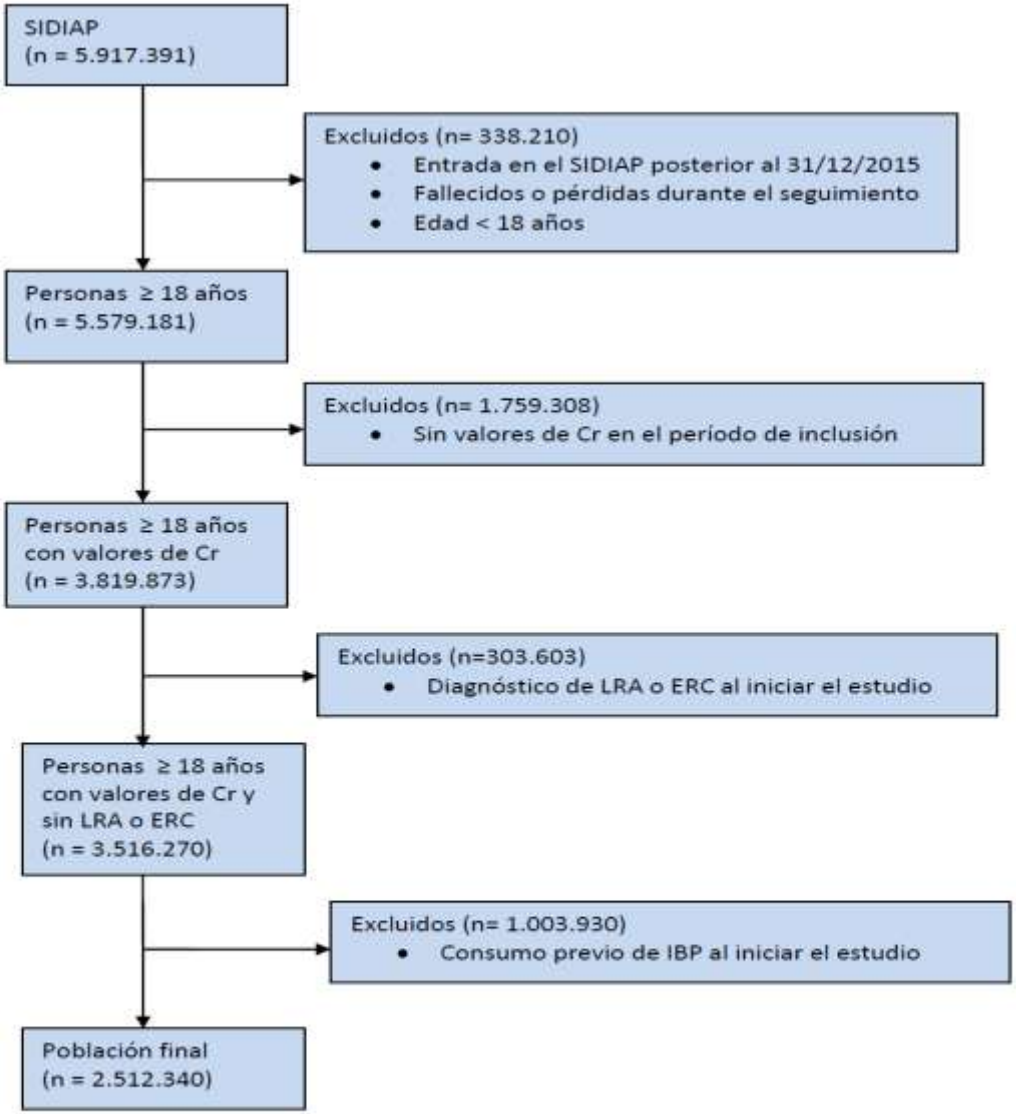

Figura 1. Diagrama de flujo del estudio de cohorte

Rodríguez-Poncelas A et al

Consumo de IBPs y riesgo de incidencia de LRA
(n= 2.512.340 personas; 993.697 consumieron IBP)

Variable	HR (95% CI)	Valor de p	HR (95% CI) tras ajustar por datos perdidos o eliminados	Valor de p
Consumidores IBP	2,42 (2,36-2,48)	< 0,001	2,57 (2,50-2,64)	< 0,001
Mujer Hombre	1,00 0,60 (0,59-0,61)	< 0,001	0,56 (0,55-0,57)	< 0,001
Diabetes	1,58 (1,54-1,62)	< 0,001	1,45 (1,41-1,49)	< 0,001
HTA	1,34 (1,31-1,37)	< 0,001	1,16 (1,12-1,17)	< 0,001
Obesidad	1,05 (1,03-1,08)	< 0,001	1,01 (0,99-1,03)	< 0,001
ECV	1,43 (1,39-1,47)	<0,001	1,36 (1,31-1,40)	< 0,001
Consumo AINEs	1,12 (1,10-1,14)	< 0,001	1,11 (1,09-1,13)	< 0,001
Consumo diuréticos	1,27 (1,10-1,15)	< 0,001	1,10 (1,08-1,13)	< 0,001
Consumo IECA/ARA2	1,13 (1,62-1,76)	< 0,001	1,70 (1,63-1,76)	< 0,001

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Consumo de IBPs y riesgo de incidencia de ERC (n= 2.512.340 personas; 993.697 consumieron IBP)

Variable	HR (95% CI)	Valor de p	HR (95% CI) tras ajustar por datos perdidos o eliminados	Valor de p
Consumidores IBP	1,37 (1,35-1,39)	< 0,001	1,35(1,33-1,37)	< 0,001
Mujer Hombre	1,00 1,14 (1,12-1,15)	< 0,001	1,13 (1,12-1,15)	< 0,001
Diabetes	1,98 (1,95-1,77)	< 0,001	1,75 (1,72-1,77)	< 0,001
HTA	2,17 (2,13-2,21)	< 0,001	2,16 (2,12-2,20)	< 0,001
Obesidad	1,25 (1,23-1,27)	< 0,001	1,25 (1,24-1,27)	< 0,001
ECV	1,15 (1,13-1,17)	<0,001	1,15 (1,13-1,17)	< 0,001
Consumo AINEs	O,99 (0,97-1,00)		O,99 (0,97-1,00)	
Consumo diuréticos	1,22 (1,20-1,24)	< 0,001	1,21 (1,20-1,23)	< 0,001
Consumo IECA/ARA2	1,37 (1,35-1,38)	< 0,001	1,37 (1,35-1,39)	< 0,001

Rodríguez-Poncelas A et al

Definition of CKD

• **CKD is defined as abnormalities of kidney structure or function, present for ≥3 months, with implications for health. (Not Graded)**

Criteria for CKD (either of the following present for >3 months)

Markers of kidney damage (one or more)	Albuminuria (AER ≥30 mg/24 hours; ACR ≥30 mg/g [≥3 mg/mmol]) Urine sediment abnormalities Electrolyte and other abnormalities due to tubular disorders Abnormalities detected by histology Structural abnormalities detected by imaging History of kidney transplantation
Decreased GFR	GFR <60 ml/min/1.73 m^2 (GFR categories G3a-G5)

Lesley A. Inker et al. Am J Kidney Dis.2014; 63(5):713-735

Valores del filtrado glomerular según la edad y el sexo en la cohorte del PREDADPS

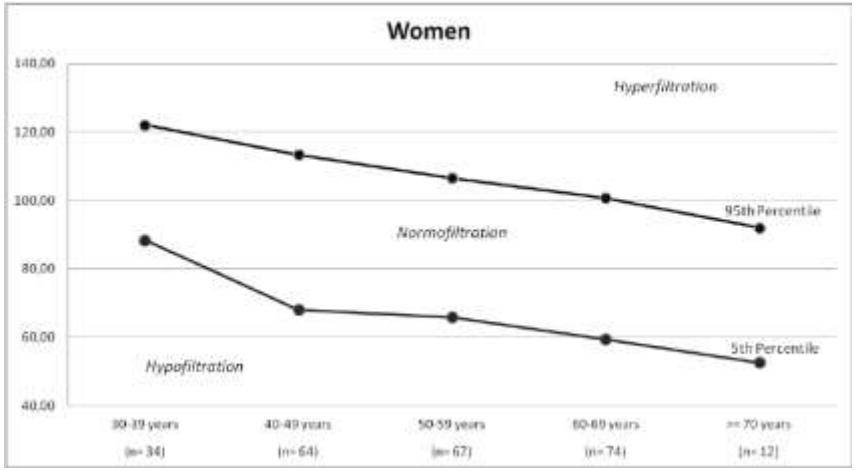
Table 1. Estimated glomerular filtration rate (eGFR) according to age and sex of all participants at baseline by glucose metabolism status.

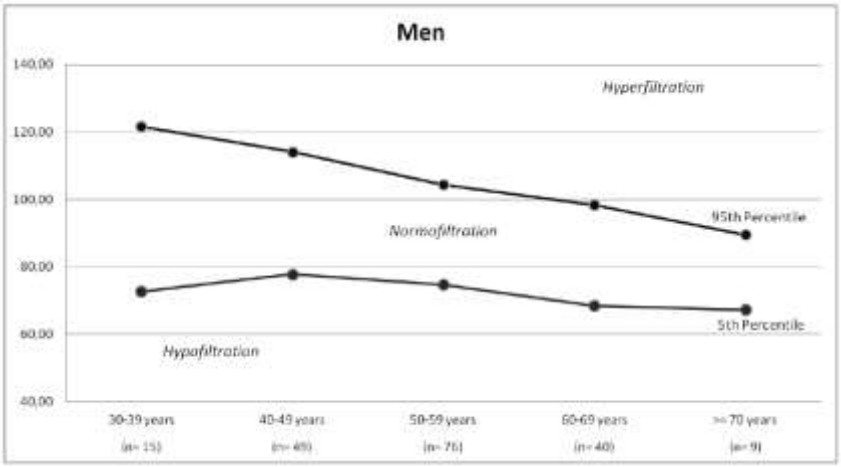
	Prediabetes					Normoglycemia				
	30–39 y N = 39	40–49 y N = 147	50–59 y N = 361	60–69 y N = 473	≥ 70 y N = 162	30–39 y N = 60	40–49 y N = 141	50–59 y N = 257	60–69 y N = 297	≥ 70 y N = 83
eGFR (Total)	106.0 (15.4)	98.2 (13.8)	90.7 (14.7)	83.6 (13.0)	77.7 (13.8)	104.4 (13.7)	97.9 (13.2)	90.9 (11.9)	84.4 (12.9)	75.5 (13.2)
eGFR (Women)	109.2 (12.9)	100.1 (15.2)	92.0 (15.5)	84.5 (12.3)	78.8 (13.9)	104.2 (12.1)	96.5 (14.1)	90.9 (12.6)	84.4 (12.8)	74.1 (13.4)
eGFR (Men)	101.8 (17.7)	96.7 (12.4)	89.3 (13.8)	82.7 (13.6)	76.6 (13.8)	104.9 (16.2)	99.7 (11.9)	91.0 (11.3)	84.4 (12.9)	77.4 (12.8)

Data are mean and (standard deviation) of mL/min per 1.73 m2

Rodríguez-Poncelas A et al. PLoS One. 2019 Sep 19;14(9):e0222848.

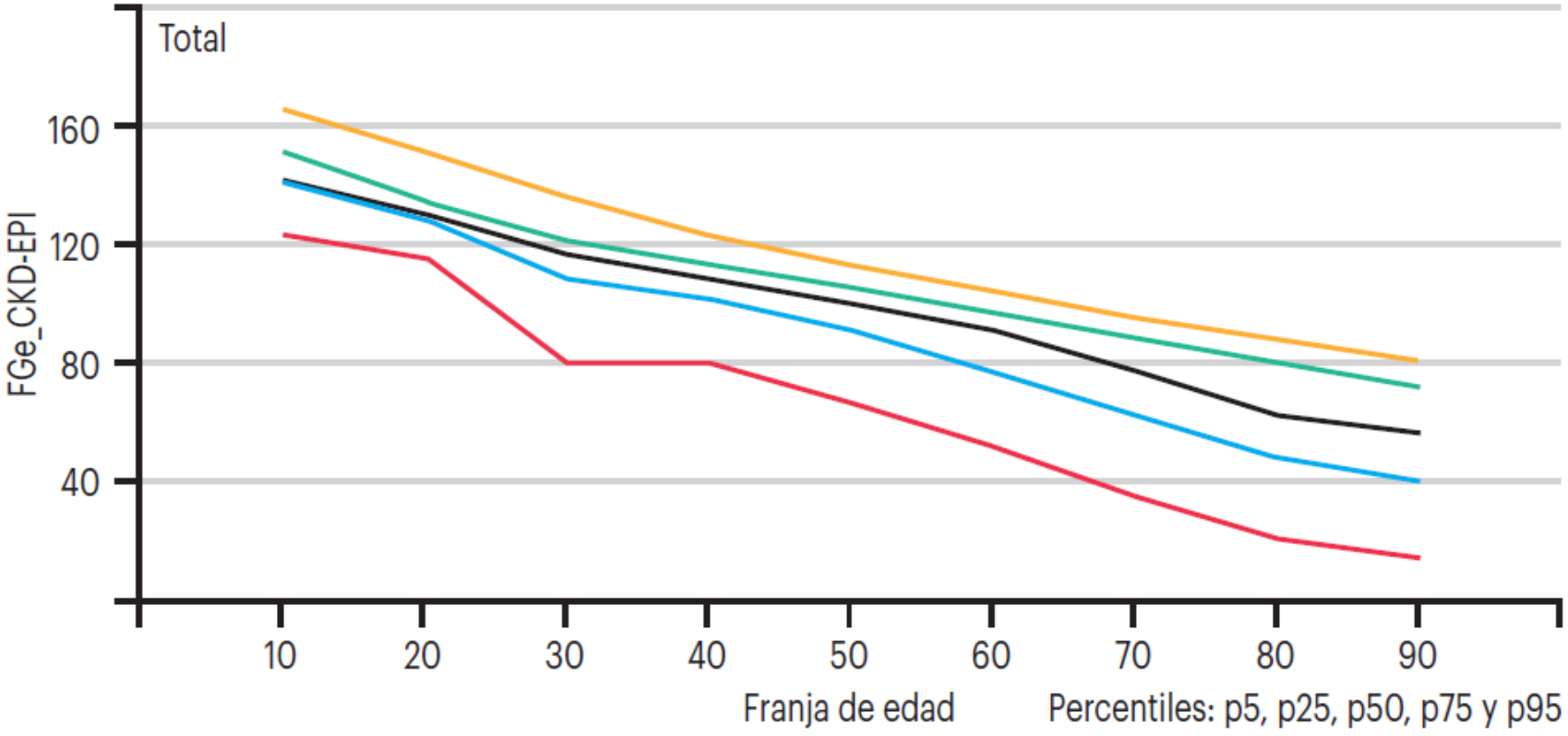
Valores del filtrado glomerular según la edad en la cohorte del PREDADPS





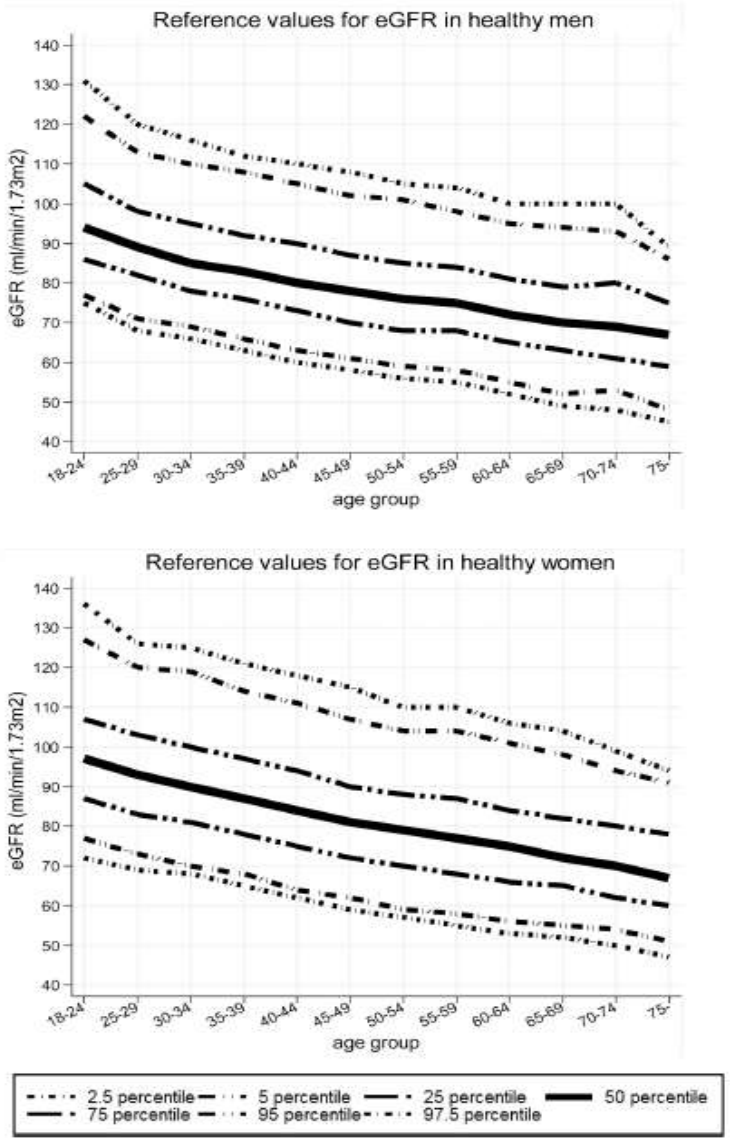
Rodríguez-Poncelas A et al. PLoS One. 2019 Sep 19;14(9):e0222848.

Valores del filtrado glomerular estimado (FGe) en una cohorte de 13.376 personas con DM2 en Girona.



Rodríguez-Poncelas A et al. Datos no publicados (2016)

Longitudinal Study of the Decline in Renal Function in Healthy Subject



Baba M et al. PLoS One 2015;10:e0129036

Definition and Identification of CKD Progression

1. Assess GFR and albuminuria at least annually in people with CKD. Assess GFR and albuminuria more often for individuals at higher risk of progression, and/or where measurement will impact therapeutic decisions. (Not Graded)
2. Recognize that small fluctuations in GFR are common and are not necessarily indicative of progression. (Not Graded)
3. Define CKD progression based on one of more of the following (Not Graded):
   - Decline in GFR category (≥ 90 [G1], 60-89 [G2], 45-59 [G3a], 30-44 [G3b], 15-29 [G4], 15 [G5] ml/min/1.73 m^2). A certain drop in eGFR is defined as **a drop in GFR category accompanied by a 25% or greater drop in eGFR from baseline**.
   - **Rapid progression is defined as a sustained decline in eGFR of more than 5 ml/min/1.73 m^2/yr.**
   - The confidence in assessing progression is increased with increasing number of serum creatinine measurements and duration of follow-up.
4. In people with CKD progression, review current management, examine for reversible causes of progression, and consider referral to a specialist. (Not Graded)

Lesley A. Inker et al. Am J Kidney Dis.2014; 63(5):713-735

- There are few therapies that slow or prevent CKD progression, particularly in early-stage CKD. New interventions must be developed and tested in randomized, controlled trials. Unfortunately, clinical trials in persons with earlier stages of CKD can be impractical and costly due to the necessary large sample size and long duration of follow-up, because **established clinical end points, such as ESKD and doubling of serum creatinine, are uncommon and occur late in the disease process**.

- In March of 2018, the National Kidney Foundation (NKF), Food and Drug Administration (FDA), and European Medicines Agency (EMA) cosponsored a scientific workshop, “Change in Albuminuria and GFR as End Points for Clinical Trials in Early Stages of CKD,” to evaluate surrogate end points for trials of kidney disease progression and to improve understanding of **changes in albuminuria and GFR as measures of kidney disease progression in early stages of CKD**.

Performance of GFR Slope as a Surrogate End Point for Kidney Disease Progression in Clinical Trials: A Statistical Simulation

Tom Greene,[1] Jian Ying,[1] Edward F. Vonesh,[2] Hocine Tighiouart,[3,4] Andrew S. Levey,[5] Josef Coresh,[6] Jennifer S. Herrick,[1] Enyu Imai,[7] Tazeen H. Jafar,[8,9] Bart D. Maes,[10] Ronald D. Perrone,[5] Lucia del Vecchio,[11] Jack F.M. Wetzels,[12] Hiddo J.L. Heerspink,[13] and Lesley A. Inker[5]

Due to the number of contributing authors, the affiliations are listed at the end of this article.

ABSTRACT

Background Randomized trials of CKD treatments traditionally use clinical events late in CKD progression as end points. This requires costly studies with large sample sizes and long follow-up. Surrogate end points like GFR slope may speed up the evaluation of new therapies by enabling smaller studies with shorter follow-up.

Methods We used statistical simulations to identify trial situations where GFR slope provides increased statistical power compared with the clinical end point of doubling of serum creatinine or kidney failure. We simulated GFR trajectories based on data from 47 randomized treatment comparisons. We evaluated the sample size required for adequate statistical power based on GFR slopes calculated from baseline and from 3 months follow-up.

Results In most scenarios where the treatment has no acute effect, analyses of GFR slope provided similar or improved statistical power compared with the clinical end point, often allowing investigators to shorten follow-up by at least half while simultaneously reducing sample size. When patients' GFRs are higher, the power advantages of GFR slope increase. However, acute treatment effects within several months of randomization can increase the risk of false conclusions about therapies based on GFR slope. Care is needed in study design and analysis to avoid such false conclusions.

Conclusions Use of GFR slope can substantially increase statistical power compared with the clinical end point, particularly when baseline GFR is high and there is no acute effect. The optimum GFR-based end point depends on multiple factors including the rate of GFR decline, type of treatment effect and study design.

JASN 30: 1756–1769, 2019. doi: https://doi.org/10.1681/ASN.2019010009

When patients' GFRs are higher, the power advantages of GFR slope increase. However, acute treatment effects within several months of randomization can increase the risk of false conclusions about therapies based on GFR slope.

GFR Slope as a Surrogate End Point for Kidney Disease Progression in Clinical Trials: A Meta-Analysis of Treatment Effects of Randomized Controlled Trials

Lesley A. Inker,[1] Hiddo J. L. Heerspink,[2] Hocine Tighiouart,[3,4] Andrew S. Levey,[1] Josef Coresh,[5] Ron T. Gansevoort,[6] Andrew L. Simon,[1] Jian Ying,[7] Gerald J. Beck,[8] Christoph Wanner,[9] Jürgen Floege,[10] Philip Kam-Tao Li,[11] Vlado Perkovic,[12] Edward F. Vonesh,[13] and Tom Greene[7]

Due to the number of contributing authors, the affiliations are listed at the end of this article.

ABSTRACT

Background Surrogate end points are needed to assess whether treatments are effective in the early stages of CKD. GFR decline leads to kidney failure, but regulators have not approved using differences in the change in GFR from the beginning to the end of a randomized, controlled trial as an end point in CKD because it is not clear whether small changes in the GFR slope will translate to clinical benefits.

Methods To assess the use of GFR slope as a surrogate end point for CKD progression, we performed a meta-analysis of 47 RCTs that tested 12 interventions in 60,620 subjects. We estimated treatment effects on GFR slope (mean difference in GFR slope between the randomized groups), for the total slope starting at baseline, chronic slope starting at 3 months after randomization, and on the clinical end point (doubling of serum creatinine, GFR<15 ml/min per 1.73 m^2, or ESKD) for each study. We used Bayesian mixed-effects analyses to describe the association of treatment effects on GFR slope with the clinical end point and to test how well the GFR slope predicts a treatment's effect on the clinical end point.

Results Across all studies, the treatment effect on 3-year total GFR slope (median R^2=0.97; 95% Bayesian credible interval [BCI], 0.78 to 1.00) and on the chronic slope (R^2 0.96; 95% BCI, 0.63 to 1.00) accurately predicted treatment effects on the clinical end point. With a sufficient sample size, a treatment effect of 0.75 ml/min per 1.73 m^2/yr or greater on total slope over 3 years or chronic slope predicts a clinical benefit on CKD progress with at least 96% probability.

Conclusions With large enough sample sizes, GFR slope may be a viable surrogate for clinical end points in CKD RCTs.

JASN 30: 1735–1745, 2019. doi: https://doi.org/10.1681/ASN.2019010007

META-ANALYSIS www.jasn.org

Evaluating Glomerular Filtration Rate Slope as a Surrogate End Point for ESKD in Clinical Trials: An Individual Participant Meta-Analysis of Observational Data

Morgan E. Grams,[1] Yingying Sang,[1] Shoshana H. Ballew,[1] Kunihiro Matsushita,[1] Brad C. Astor,[2,3] Juan Jesus Carrero,[4] Alex R. Chang,[5] Lesley A. Inker,[6] Timothy Kenealy,[7,8] Csaba P. Kovesdy,[9,10] Brian J. Lee,[11] Adeera Levin,[12] David Naimark,[13] Michelle J. Pena,[14] Jesse D. Schold,[15,16] Varda Shalev,[17] Jack F. M. Wetzels,[18] Mark Woodward,[1,19,20] Ron T. Gansevoort,[21] Andrew S. Levey,[6] and Josef Coresh[1]

Due to the number of contributing authors, the affiliations are listed at the end of this article.

ABSTRACT

Background Decline in eGFR is a biologically plausible surrogate end point for the progression of CKD in clinical trials. However, it must first be tested to ensure strong associations with clinical outcomes in diverse populations, including patients with higher eGFR.

Methods To investigate the association between 1-, 2-, and 3-year changes in eGFR (slope) with clinical outcomes over the long term, we conducted a random effects meta-analysis of 3,758,551 participants with baseline eGFR≥60 ml/min per 1.73 m^2 and 122,664 participants with eGFR<60 ml/min per 1.73 m^2 from 14 cohorts followed for an average of 4.2 years.

Results Slower eGFR decline by 0.75 ml/min per 1.73 m^2 per year over 2 years was associated with lower risk of ESKD in participants with baseline eGFR≥60 ml/min per 1.73 m^2 (adjusted hazard ratio, 0.70; 95% CI, 0.68 to 0.72) and eGFR<60 ml/min per 1.73 m^2 (0.71; 95% CI, 0.68 to 0.74). The relationship was stronger with 3-year slope. For a rapidly progressing population with predicted 5-year risk of ESKD of 8.3%, an intervention that reduced eGFR decline by 0.75 ml/min per 1.73 m^2 per year over 2 years would reduce the ESKD risk by 1.6%. For a hypothetical low-risk population with a predicted 5-year ESKD risk of 0.58%, the same intervention would reduce the risk by only 0.13%.

Conclusions Slower decline in eGFR was associated with lower risk of subsequent ESKD, even in participants with eGFR≥60 ml/min per 1.73 m^2, but those with the highest risk would be expected to benefit the most.

JASN 30: 1746–1755, 2019. doi: https://doi.org/10.1681/ASN.2019010008

Slower decline in eGFR was associated with lower risk of subsequent ESKD, even in participants with eGFR ≥60 ml/min per 1.73 m^2, but those with the highest risk would be expected to benefit the most.

Diabetologia
https://doi.org/10.1007/s00125-019-4948-4

ARTICLE

The relationship between eGFR slope and subsequent risk of vascular outcomes and all-cause mortality in type 2 diabetes: the ADVANCE-ON study

Megumi Oshima[1,2] · Min Jun[1] · Toshiaki Ohkuma[1] · Tadashi Toyama[1,2] · Takashi Wada[2] · Mark E. Cooper[3] · Samy Hadjadj[4] · Pavel Hamet[5] · Stephen Harrap[6] · Giuseppe Mancia[7] · Michel Marre[8] · Bryan Williams[9] · John Chalmers[1] · Mark Woodward[1,10,11] · Vlado Perkovic[1] · on behalf of the ADVANCE Collaborative Group

Received: 4 April 2019 / Accepted: 5 June 2019

Abstract

Aims/hypothesis Some studies have reported that annual change in eGFR (eGFR slope) is associated with the future risk of end-stage kidney disease, cardiovascular disease and death in general or chronic kidney disease cohorts. However, the benefits of using eGFR slopes for prediction of major clinical outcomes in diabetes are unclear.

Methods We used data from the Action in Diabetes and Vascular Disease: Preterax and Diamicron MR Controlled Evaluation (ADVANCE) trial and the ADVANCE Post-Trial Observational Study (ADVANCE-ON). After excluding the first 4 months during which an acute fall in eGFR was induced by the initiation of an ACE inhibitor and diuretic combination agent, eGFR slopes were estimated by linear mixed models, using three measurements of eGFR at 4, 12 and 24 months after randomisation over 20 months, and categorised according to quartiles. Cox regression models were used to evaluate adjusted HRs for the study's primary outcome, a composite of major renal events, major macrovascular events and all-cause mortality during the subsequent follow-up from 24 months after randomisation.

Results A total of 8,879 participants (80%) were included in this cohort. The mean age was 65.6 years (SD 6.3), the mean eGFR was 75 ml min^{-1} (1.73 m)$^{-2}$ (SD 17) and the median urinary albumin/creatinine ratio was 14 μg/mg (interquartile range 7–38). The mean eGFR slope was −0.63 ml min^{-1} (1.73 m)$^{-2}$ $year^{-1}$ (SD 1.75). Over a median follow-up of 7.6 years following the 20-month eGFR slope ascertainment period, 2,221 participants (25%) met the primary outcome. An annual substantial decrease in eGFR (lowest 25%, <−1.63 ml min^{-1} [1.73 m]$^{-2}$ $year^{-1}$) was significantly associated with the subsequent risk of the primary outcome (HR 1.30 [95% CI 1.17, 1.43]) compared with a stable change in eGFR (middle 50%, −1.63 to 0.33). An annual

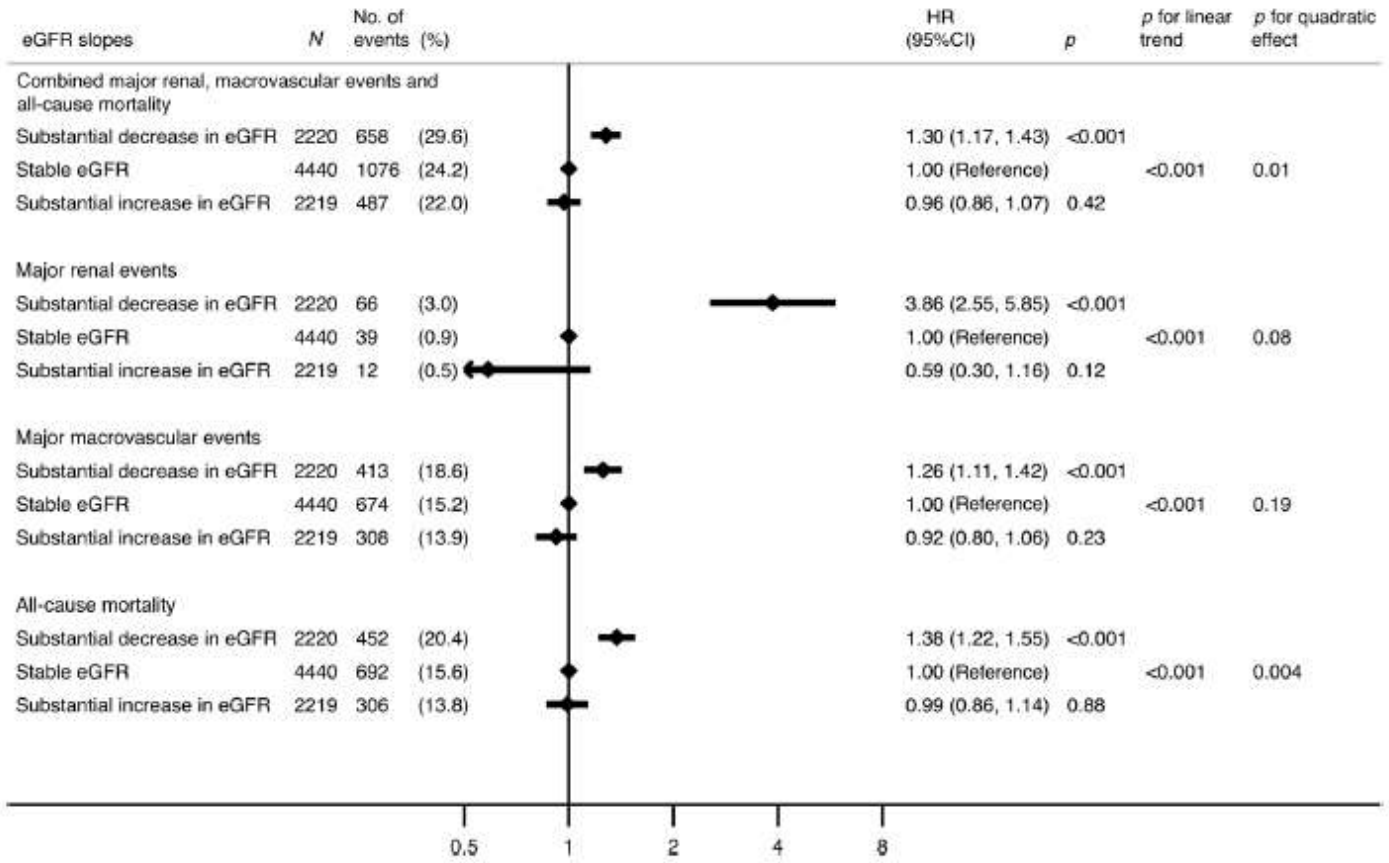


Fig. 2 Adjusted HRs for study outcomes according to categories of eGFR slope over the 20-month eGFR slope ascertainment period. Covariates: registration values of age, sex, region of residence, duration of diabetes, log-transformed UACR, systolic BP, diastolic BP, a history of macrovascular disease, smoking, drinking, treated hypertension, HbA_{1c}, HDL-cholesterol, LDL-cholesterol, log-transformed triacylglycerol and BMI, 4-month eGFR and randomised treatment allocation (BP and glucose treatment)

An annual substantial decrease in eGFR over 20 months was strongly associated with the future risk of renal and cardiovascular events and all-cause mortality in type 2 diabetes, supporting the potential for using eGFR slope as a predictor for major clinical outcomes.

Go et al. BMC Nephrology (2018) 19:146
https://doi.org/10.1186/s12882-018-0942-1

BMC Nephrology

RESEARCH ARTICLE Open Access

Contemporary rates and predictors of fast progression of chronic kidney disease in adults with and without diabetes mellitus

Alan S. Go[1,2,3*], Jingrong Yang[1], Thida C. Tan[1], Claudia S. Cabrera[4], Bergur V. Stefansson[4], Peter J. Greasley[4], Juan D. Ordonez[5] and for the Kaiser Permanente Northern California CKD Outcomes Study

Abstract

Background: Chronic kidney disease (CKD) is highly prevalent but identification of patients at high risk for fast CKD progression before reaching end-stage renal disease in the short-term has been challenging. Whether factors associated with fast progression vary by diabetes status is also not well understood. We examined a large community-based cohort of adults with CKD to identify predictors of fast progression during the first 2 years of follow-up in the presence or absence of diabetes mellitus.

Methods: Within a large integrated healthcare delivery system in northern California, we identified adults with estimated glomerular filtration rate (eGFR) 30–59 ml/min/1.73 m^2 by CKD-EPI equation between 2008 and 2010 who had no previous dialysis or renal transplant, who had outpatient serum creatinine values spaced 10–14 months apart and who did not initiate renal replacement therapy, die or disenroll during the first 2 years of follow-up. Through 2012, we calculated the annual rate of change in eGFR and classified patients as fast progressors if they lost > 4 ml/min/1.73 m^2 per year. We used multivariable logistic regression to identify patient characteristics that were independently associated with fast CKD progression stratified by diabetes status.

Results: We identified 36,195 eligible adults with eGFR 30–59 ml/min/1.73 m^2 and mean age 73 years, 55% women, 11% black, 12% Asian/Pacific Islander and 36% with diabetes mellitus. During 24-month follow-up, fast progression of CKD occurred in 23.0% of patients with diabetes vs. 15.3% of patients without diabetes. Multivariable predictors of fast CKD progression that were similar by diabetes status included proteinuria, age ≥ 80 years, heart failure, anemia and higher systolic blood pressure. Age 70–79 years, prior ischemic stroke, current or former smoking and lower HDL cholesterol level were also predictive in patients without diabetes, while age 18–49 years was additionally predictive in those with diabetes.

Conclusions: In a large, contemporary population of adults with eGFR 30–59 ml/min/1.73 m^2, accelerated progression of kidney dysfunction within 2 years affected ~ 1 in 4 patients with diabetes and ~ 1 in 7 without diabetes. Regardless of diabetes status, the strongest independent predictors of fast CKD progression included proteinuria, elevated systolic blood pressure, heart failure and anemia.

Keywords: Diabetes, Chronic kidney disease, Progression, Risk factors, Proteinuria, Anemia, Blood pressure

Risk of fast progression of chronic kidney disease in adults with and without diabetes mellitus

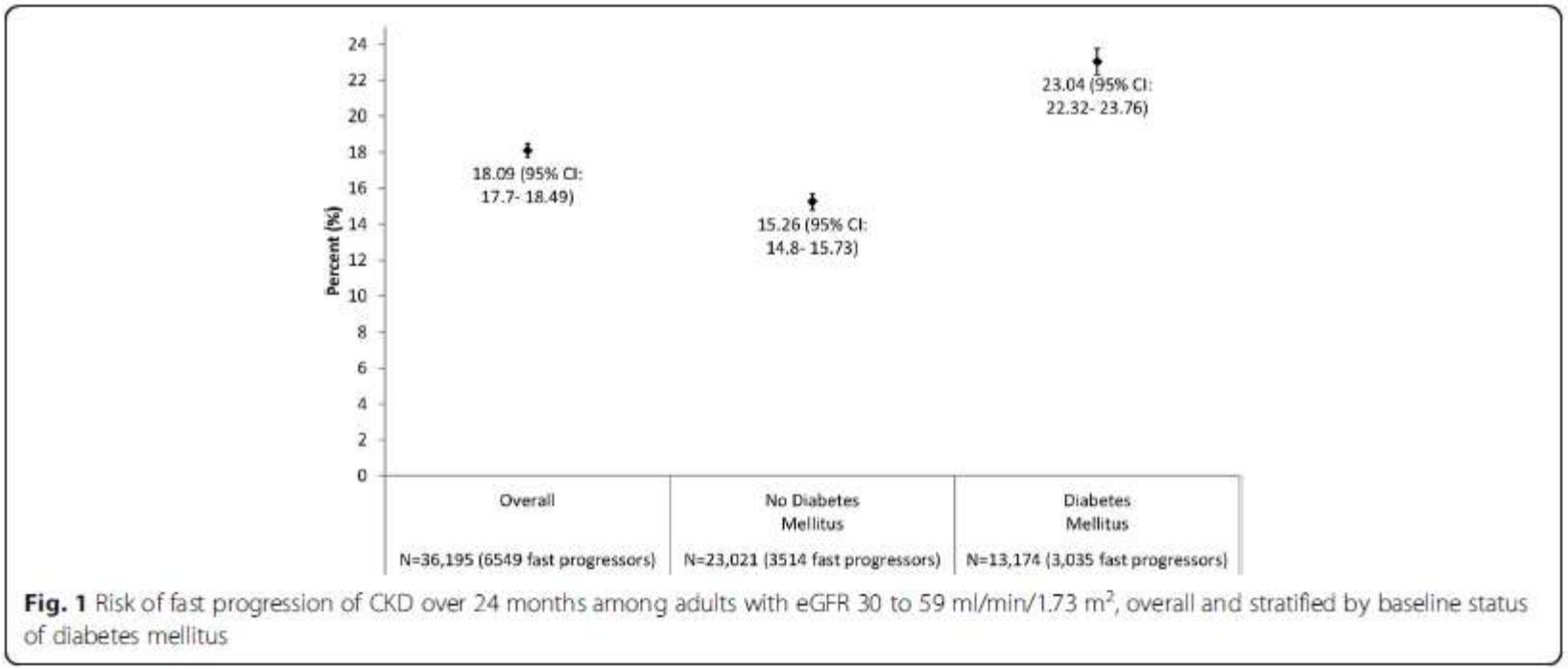


Fig. 1 Risk of fast progression of CKD over 24 months among adults with eGFR 30 to 59 ml/min/1.73 m^2, overall and stratified by baseline status of diabetes mellitus

In adults with eGFR 30–59 ml/min/1.73 m^2, accelerated progression of kidney dysfunction within 2 years affected ~ 1 in 4 patients with diabetes and ~ 1 in 7 without diabetes. Regardless of diabetes status, the strongest independent predictors of fast CKD progression included proteinuria, elevated systolic blood pressure, heart failure and anemia.

Go et al. BMC Nephrology (2018) 19:146

Cambios en el filtrado glomerular desde la visita basal hasta el final del seguimiento en la cohorte del PREDADPS

Mean changes of estimated Glomerular Filtration Rate (eGFR) from baseline to the end of the 5-years follow-up and p value for difference in change in eGFR between prediabetes cohort with respect no prediabets cohort

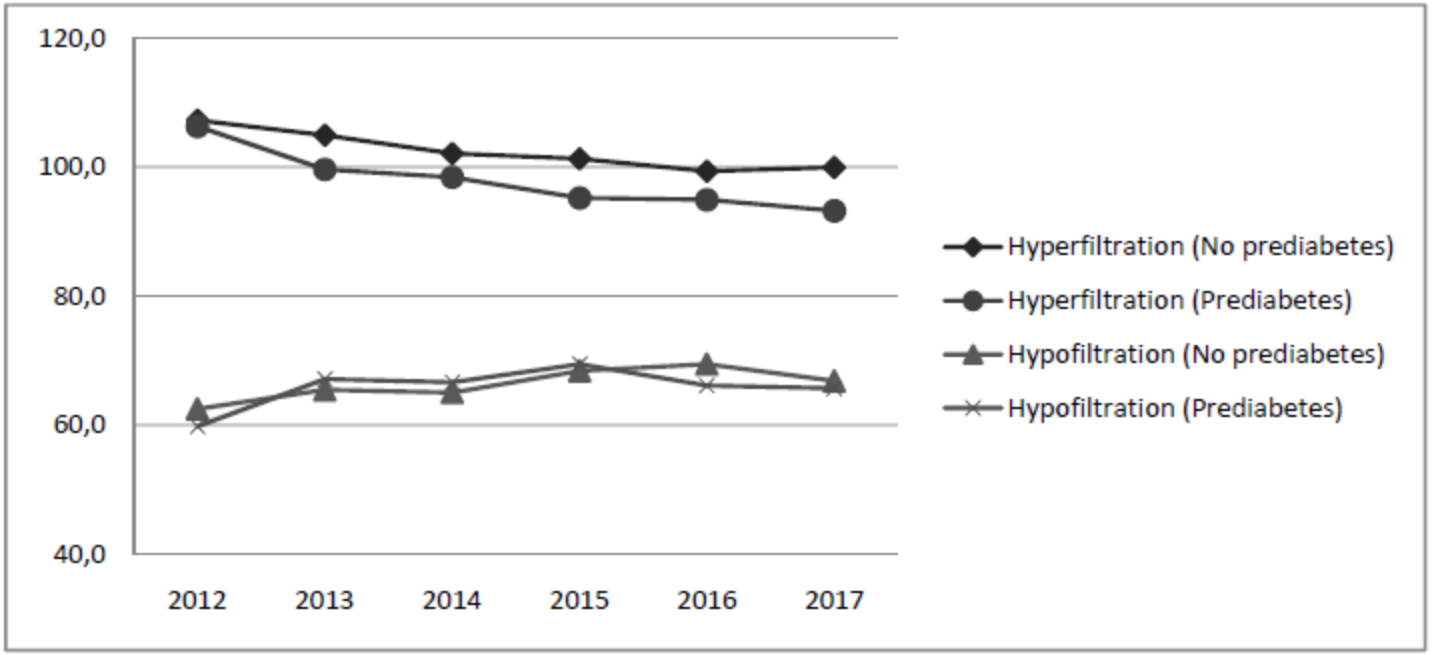
	n	eGFR[$] at baseline	Change in eGFR[$] (1st year)	Change in eGFR[$] (2nd year)	Change in eGFR[$] (3rd year)	Change in eGFR[$] (4th year)	Change in eGFR[$] (5th year)
Prediabetes cohort							
Hypofiltration	72	59.75	+ 7.32*	+ 6.82	+ 9.64	+ 6.35	+5.91
Normalfiltration	601	88.34	-1.13	-1.20	-0.23	-1.69	-2.60
Hyperfiltration	61	106.26	-6.63*	-7.88	-11.10*	-11.37	-13.08*
No prediabetes cohort							
Hypofiltration	41	62.48	+ 2.95	+ 2.51	+ 5.90	+ 6.96	+ 4.36
Normalfiltration	465	88.73	-1.56	-0.71	-1.15	-2.68	-3.21
Hyperfiltration	38	107.24	-2.33	-5.13	-5.98	-7.90	-7.30

[$] mL/min per 1.73 m^2

* $p < 0.05$.

Rodríguez-Poncelas A et al. Datos no publicados

Cambios en el filtrado glomerular desde la visita basal hasta el final del seguimiento en la cohorte del PREDADPS



Rodríguez-Poncelas A et al. Datos no publicados

Conclusiones

- Identificar las caídas bruscas del FG, diagnosticar la causa y corregirla siempre que sea posible.
- A partir de los 40 años se produce una disminución del FG (0,75-1 ml/min/1.73 m^2/año) relacionado con la edad.
- Descensos del FG mayores de lo considerado normal se asocian con un aumento de episodios renales, cardiovasculares y mortalidad total en la diabetes.
- Controlar los factores de riesgo de la progresión rápida de la ERC.