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Progression-related bias in the monitoring of kidney function in patients with diabetes and chronic kidney disease

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Abstract

The Cockcroft and Gault (CG) and Modification of Diet in Renal Disease (MDRD) equations underestimate the glomerular filtration rate (GFR) decline in diabetes. Do this decline and the albumin excretion rate (AER) influence their validity ? In 161 diabetic patients, isotopically determined GFR (i-GFR) (51Cr-EDTA) was compared with estimated GFR (e-GFR) by the CG, MDRD, and the new Mayo Clinic Quadratic (MCQ) equations. We searched for a relation between the error in e-GFR and the AER. An influence of the AER outcome on the e-GFR decline was evaluated in 63 subjects followed up over 3 years. The MDRD and the MCQ were more precise and accurate than the CG, but they were biased. The error increased with AER for the CG ($r = 0.25$, $P = .001$) and the MDRD ($r = 0.20$, $P = .009$), but not for the MCQ. For the 63 patients followed up, the e-GFR declines by the 3 estimations were related to the initial AER, whereas no relation with arterial blood pressure, hemoglobin A_{1C}, hemoglobin, and blood lipids emerged. The MCQ declines were more pronounced: $-10.5\% \pm 8.9\%$ in the macroalbuminuric group ($P < .05$ vs both microalbuminuric [$-2.6\% \pm 10.1\%$] and normoalbuminuric [$-0.1\% \pm 6.6\%$] groups), and were related to the outcome of the AER ($r = 0.33$, $P < .05$). As chronic kidney disease progresses in diabetes, the declining GFR and rising AER influence the estimation of GFR by the CG and MDRD equations, underestimating the GFR decline and the benefit of reducing the AER. The less affected MCQ evidences a slower e-GFR decline with AER control.

1. Introduction

About one third of patients with diabetes are affected by diabetic nephropathy [1], the first cause of end-stage renal disease in most countries [2]. According to the recent recommendations of the American Diabetes Association and the National Kidney Foundation, 2 practically accessible parameters are critical for the detection [3] and follow-up [4] of chronic kidney disease (CKD) in patients with diabetes: the albumin excretion rate (AER) and the glomerular filtration rate (GFR) as estimated (e-GFR) by the Cockcroft and Gault formula (CG) or the Modification of Diet in Renal Disease (MDRD) equation.

These recommended GFR-predictive equations are far from perfect. Because it is more accurate [5], not biased by body weight [6], and more precise when glucose control is poor [7], the MDRD seems preferable. However, it underestimates normal to high GFR levels [8]; and better results have been reported with the CG in recent-onset type 1 [9] and type 2 [10] diabetes mellitus. The inaccuracy of the CG and the MDRD bias according to the GFR level may be of particular importance when monitoring GFR decline is the objective: both the CG and the MDRD underestimate it [11,12], which has even been qualified as “unacceptable” [13]. To our knowledge, the less biased Mayo Clinic Quadratic (MCQ) equation [14] has not been tested for the purpose of monitoring kidney

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function. A possible interaction between the AER and the estimation of GFR is also poorly documented, although Cockcroft and Gault [15] themselves mentioned that heavy proteinuria was associated with a GFR overestimation due to increased tubular secretion of creatinine: such an effect may become relevant as the AER becomes a target to retard the GFR decline [14,16,17].

In 161 patients with diabetes, we determined isotopic GFR (i-GFR) by 51Cr-EDTA clearance; and we compared the results to the creatinine-based estimations (e-GFR: CG, MDRD, MCQ). Regression analyses were performed to detect an influence of the AER on the error of e-GFR. Sixty-three of the patients who had e-GFR less than 60 were then followed up for 3 years to study the influence of AER, its progression, and other recognized predictors (arterial pressure, hemoglobin A_{1C} [HbA_{1C}], blood lipids, hemoglobin) on e-GFR decline.

2. Methods

2.1. Subjects

One hundred sixty-one diabetic patients (93 men; 52 type 1; mean age, 62 ± 14 years [19-83]; body mass index [BMI], 27.5 ± 4.9 [15.6-48.9]; HbA_{1C}, 8.6% ± 1.7% [5.2%-15%]) were recruited from the Nutrition-Diabetology and Nephrology departments of the Centre Hospitalier Universitaire de Bordeaux.

Sixty-three patients (32 men; 16 type 1; mean age, 64 ± 12 years; BMI, 27.4 ± 4.4; HbA_{1C}, 8.7% ± 1.7%.) were followed up for 38 ± 11 months because they presented CKD according to an MDRD-estimated GFR (MDRD e-GFR) of less than 60 mL/min per 1.73 m^2, not requiring renal replacement therapy on inclusion. Thirty-seven were on angiotensin-converting enzyme inhibitors and 10 were on angiotensin receptor blockers on inclusion.

The patients gave written informed consent to participate to the study, which was approved by the ethical committee of our institution. This study was supported by a clinical research program in the Bordeaux University Hospital.

2.2. Analytical methods

The AER was determined on one 24-hour urine collection during a short hospitalization with an immunonephelometric analyzer (Behring Nephelometer 2; Dade Behring, Marburg, Germany) using an appropriate kit (Nantiserum VO human albumin, Dade Behring). Serum creatinine was determined on a multiparameter analyzer (Olympus AU 640; Olympus Optical, Tokyo, Japan) using the Jaffé method, with bichromatic measurements according to the manufacturer's specifications and daily calibration of the analyzer. This procedure did not change in our laboratory during the study. Clearance of the radionuclide marker was measured after intravenous injection of 51Cr-EDTA (Cis Industries, Gif/Yvette, France). All patients were studied in the morning at 9:00 AM after a light breakfast. After a single bolus of 100 μCi (3.7 MBq) of 51Cr-EDTA, 4 venous blood samples were drawn at 75, 105, 135, and 165 minutes; and urinary samples were collected at 90, 120, 150, and 180 minutes, as previously described [18]. The final result (i-GFR) was the mean of the 4 clearance values. If for one period the urine flow was too low or if a clearance value was not within ±20% of the mean of the 3 others, this value was excluded; and the mean was calculated for the other 3 clearances. Less than 5% of the values were excluded this way. The coefficient of variation in the i-GFR measurement was 13.7% ± 7.4%. The 51Cr-EDTA radioactivity was measured in a gamma counter (COBRA 2, model 05003; Packard Instruments, Meriden, CT).

2.3. Follow-up, care

This prospective study began on June 2001. It was based on a cooperative follow-up between diabetologists and nephrologists with the establishment of a joint medical file for each patient. This cooperative follow-up was complementary and included 1 visit with the diabetologist every 4 months and 1 visit with the nephrologist every year if 40 < MDRD e-GFR ≤ 60 mL/min per 1.73 m^2, every 4 months if 20 < MDRD e-GFR ≤ 40, or every 1 or 2 months if MDRD e-GFR ≤20. Mean duration of the follow-up was 39 ± 9 months.

The care program objectives included glycemic control according to the French 1999 recommendations [19]: HbA_{1C} <8.0%, if possible 6.5% without severe hypoglycemia in type 2 and <7.0% in type 1, but also control of associated factors such as hypertension (objective: <130/80 mm Hg) and dyslipidemia (objective: low-density lipoprotein cholesterol <1.3 g/L). We prescribed 0.8 g protein per kilogram per day according to the National Kidney Foundation recommendations [20], except for patients with clinical signs of undernutrition or who were 65 years and older.

2.4. GFR estimations (e-GFR)

Single serum creatinine measurements were performed the day before the isotopic measurement of GFR (i-GFR) to calculate:

1. CG formula [15]:

$$\mathrm{CG} = \frac{(140 - \text{age [years]}) \times \text{body weight [kilograms]} \times (0.85 \text{ if female})}{72 \times \text{serum creatinine [milligrams per deciliter]}}$$

As this equation was originally designed to estimate creatinine clearance expressed in milliliters per minute, the results were adjusted to body surface area calculated by the formula of Dubois and Dubois [21]. For the following MDRD, MCQ, and composite estimations of GFR, the

results are directly expressed as milliliters per minute per 1.73 m^2.

2. MDRD study equation:
We used the simplified equation [22]:

$$\begin{aligned}\text{MDRD} = 186 &\times (\text{serum creatinine [milligrams per deciliter]})^{-1.154} \\ &\times (\text{age [years]})^{-0.203} \times (0.742 \text{ if female}) \\ &\times (1.210 \text{ if African American})\end{aligned}$$

3. MCQ equation [5]:

$$\begin{aligned}\text{MCQ} = \exp(&1.911 + 5.249/\text{SCr [milligrams per deciliter]} \\ &- 2.114/\text{sCr [milligrams per deciliter]}^2 \\ &- 0.00686 \times \text{age [years]} - 0.205 \text{ if female})\end{aligned}$$

sCr indicates serum creatinine.

2.5. Statistical analysis

The performances of the predictive equations (CG, MDRD, MCQ) were assessed: bias (Bland and Altman procedure and percentage of difference with isotopic GFR), precision (r and interquartile range of the bias), and accuracy (absolute percentage of difference with isotopic GFR and percentage of estimations within ±30% of isotopic GFR). The relation between the AER and the error of GFR estimation (e-GFR less i-GFR) was studied by regression analysis. Analysis of variance for repeated measurements was used to compare the renal parameters at inclusion and at the end of the follow-up. The e-GFR declines were expressed as percentage per year; their relations to the AER and the AER progression were studied by regression analysis. The e-GFR errors and e-GFR declines were compared between the normoalbuminuric, microalbuminuric, and macroalbuminuric groups by analysis of variance, with a Bonferroni correction. The calculations were performed with SPSS (Chicago, IL) software, version 10.0. Results are presented as mean ± SD, and $P < .05$ was considered significant.

Table 1
The performances of the e-GFRs by the 3 creatinine-based equations in 161 patients with diabetes; i-GFR: 60.2 ± 35.6 mL/min per 1.73 m^2

	Cockcroft	MDRD	MCQ
e-GFR (mL/min per 1.73 m^2)	63.8 ± 36.0	53.9 ± 24.6	64.2 ± 31.5
Correlation to i-GFR (r)	0.74	0.81	0.83
Median % difference with i-GFR	+5.2%	−6.9%	+8.9%
Interquartile range	55%	41%	41%
% Absolute difference with i-GFR	43% ± 55%	30% ± 36%	32% ± 46%
% e-GFR within i-GFR ±30%	57%	69%	65%
Bland and Altman 2 SD	51.6	42.4	39.6
r	0.03	−0.54	−0.21
P	NS	<.001	<.01

3. Results

3.1. GFR and its estimations at inclusion (N = 161)

The i-GFR was 60.2 ± 35.6 mL/min per 1.73 m^2, well correlated with each of the e-GFR. The CG did not differ significantly from i-GFR, whereas the MDRD underestimated and the MCQ overestimated GFR. The CG was not biased according to the Bland and Altman procedure, whereas the MCQ and more dramatically the MDRD underestimated high GFR. Their accuracies, as reflected by mean absolute differences as percentage, differed: the MDRD was the most accurate (not significant [NS] vs MCQ, $P < .001$ vs CG), followed by the MCQ ($P < .01$ vs CG), as shown in Table 1.

3.2. Influence of the AER on the error of e-GFR (N = 161)

The relative error of e-GFR (e-GFR minus i-GFR/i-GFR) was correlated with the AER for the CG ($r = 0.25$, $P = .001$) and the MDRD ($r = 0.20$, $P = .009$), but not for the MCQ ($r = 0.03$, NS). Because differing AERs are associated with differing GFR levels, these analyses were repeated after inclusion of the mean of e- and i-GFR (as in the Bland and Altman procedure) in the regression model. The effect of the AER on the relative error was still significant for the CG ($P < .01$), and a bias according to GFR level was detectable for this estimation ($P < .05$). The influence of the AER was no longer significant for the MDRD and did not reach significance for the MCQ after accounting for the GFR level. The error by the CG was also correlated with BMI ($r = 0.41$, $P < .001$; for the MDRD $r = 0.007$; for the MCQ $r = 0.04$, both NS).

The performances of the 3 equations in the normo-, micro-, and macroalbuminuric groups are shown in Table 2: the CG significantly more overestimated GFR in macro- as compared with normoalbuminuric patients ($P = .02$), and there were also tendencies for overestimations in the macro- as compared with the microalbuminuric patients by the CG ($P = .08$) and MDRD ($P = .06$) after the Bonferroni correction. By contrast, the MCQ overestimated GFR in the 3 groups to a similar extent. As shown in Table 2, the greater overestimations of GFR with growing AER also seemed to be present in subjects with i-GFR less than 60; but this was not significant in their lower effective.

3.3. Outcome (n = 63)

The i-GFR of the 63 patients followed up was 46.4 ± 29.1 mL/min per 1.73 m^2 on inclusion. As shown in Table 3, their arterial pressure and HbA_{1C} improved during the follow-up; and blood lipids were controlled. Chronic kidney disease progressed during the follow-up, as reflected by increased serum creatinine (initial, 146 ± 7 μmol/L; final, 186 ± 15; $P = .001$) and a tendency for increased AER (initial, 401 ± 520 mg/24 h; final, 647 ± 1041; $P = .08$). The e-GFR

Table 2
The performances of the e-GFRs by the 3 creatinine-based equations in 161 patients with diabetes, categorized by their AERs (normoalbuminuric, <30 mg/24 h; microalbuminuric, 30-300 mg/24 h; macroalbuminuric, >300 mg/24 h)

	Normoalbuminuric	Microalbuminuric	Macroalbuminuric
n (with i-GFR <60)	*47 (21)*	*56 (23)*	*58 (42)*
AER (mg/24 h)	19 ± 6	133 ± 90	1276 ± 809
i-GFR (mL/min per 1.73 m^2)	64 ± 28	72 ± 37	45 ± 34
CG			
Correlation to i-GFR (*r*)	0.79	0.67	0.73
Median % difference with i-GFR	−1.7%	+1.6%	+21.1%
Interquartile range	30%	46%	84%
% Absolute difference with i-GFR	27% ± 41%	40% ± 47%	57% ± 68%
% e-GFR within i-GFR ±30%	76%	46%	51%
Median % difference with i-GFR	*+3%*	*+42%*	*+46%*
Interquartile range if i-GFR<60	*53%*	*82%*	*86%*
MDRD equation			
Correlation to i-GFR (*r*)	0.80	0.71	0.88
Median % difference with i-GFR	−7.8%	−9.6%	+7.5%
Interquartile range	29%	40%	55%
% Absolute difference with i-GFR	27% ± 43%	26% ± 19%	35% ± 43%
% e-GFR within i-GFR ±30%	80%	67%	62%
Median % difference with i-GFR	*+6%*	*+15%*	*+18%*
Interquartile range if i-GFR<60	*23%*	*30%*	*51%*
MCQ			
Correlation to i-GFR (*r*)	0.83	0.75	0.87
Median % difference with i-GFR	+13.9%	+6.0%	+8.5%
Interquartile range	28%	49%	48%
% Absolute difference with i-GFR	32% ± 56%	30% ± 29%	35% ± 52%
% e-GFR within i-GFR ±30%	78%	57%	62%
Median % difference with i-GFR	*+21%*	*+37%*	*+16%*
Interquartile range if i-GFR<60	*55%*	*28%*	*49%*

The results limited to the 86 subjects with i-GFRs less than 60 mL/min per 1.73 m^2 are given in italics.

declined to 41.6 ± 17.5 mL/min per 1.73 m^2 with the CG (P = .002 vs initial), 38.4 ± 17.2 with the MDRD (P = .052 vs initial), and 43.7 ± 23.2 with the MCQ (P = .03 vs initial).

The percentage of e-GFR declines and their relation to the initial AER and its progression during the follow-up (final minus initial AER) are shown in Table 4. The percentage of MDRD-estimated e-GFR decline was significantly lower than as estimated by the CG and the MCQ (both Ps < .05). All GFR estimates detected a correlation between GFR decline with the initial AER, whereas no significant relation with other usual predictors (arterial blood pressure, HbA_{1C}, hemoglobin, blood lipids) emerged: the MCQ declined significantly more in the macroalbuminuric group (overall, P = .002; P < .01 vs microalbuminuric and vs normoalbuminuric groups after Bonferroni correction). The differences between these groups were far from significant using the CG (P = .24) and the MDRD (P = .29). Only the MCQ detected a significant relation between the GFR decline and the progression of the AER, whereas that evidenced by the MDRD did not reach significance.

Table 3
The outcome of the main progression factors during the 3 years (39 ± 9 months) of follow-up in 63 patients

	Initial	Final	*P*
Body weight (kg)	74.5 ± 12.7	74.9 ± 14.4	NS
Arterial pressure (mm Hg)			
Systolic	147 ± 18	136 ± 19	<.001
Diastolic	81 ± 9	74 ± 10	
No. of distinct antihypertensive drugs	2.4 ± 1.3	3.2 ± 1.3	<.001
HbA_{1C} (%)	8.7 ± 1.7	7.8 ± 1.4	<.001
Cholesterol (g/L)	2.07 ± 0.49	1.95 ± 0.44	NS
Triglycerides (g/L)	1.87 ± 1.43	1.63 ± 1.17	NS
HDL cholesterol (g/L)	0.55 ± 0.18	0.56 ± 0.20	NS
LDL cholesterol (g/L)	1.15 ± 0.36	1.07 ± 0.30	NS

HDL indicates high-density lipoprotein; LDL, low-density lipoprotein.

4. Discussion

Table 4
The e-GFR changes at 3 years (reported to the initial e-GFR, as percentage) by the 3 creatinine-based equations in 63 patients with diabetes and CKD

	Cockroft	MDRD	MCQ
% e-GFR decline			
Total	−3.9% ± 9.3%	−2.1% ± 12.8%	−5.2% ± 10.0%
Normoalbuminuric	−1.1% ± 4.5%	+0.2% ± 4.4%	−0.1% ± 6.6%
Microalbuminuric	−3.1% ± 7.7%	−1.2% ± 8.6%	−2.6% ± 10.1%
Macroalbuminuric	−6.4% ± 12.2%	−6.1% ± 18.6%	−10.5% ± 8.9%
Correlation with initial AER			
r	0.36	0.32	0.45
P	.004	.01	.001
Correlation with evolution of AER			
r	0.12	0.30	0.33
P	.44	.051	.03

The evolution of the AERs was calculated as final AER less initial AER.

Most studies on the validation of GFR-predictive equations are in line with a GFR-dependent bias. In nondiabetic subjects, Froissard et al [23] reported an overestimation of low GFR, mainly by the CG, and an underestimation of high GFR, mainly by the MDRD, also observed in Chinese subjects [24]. Recent reports also demonstrated the underestimated high MDRD in diabetic patients [9-11,13,25], whereas the CG tended to overestimate GFR in the 249 renal-insufficient diabetic patients studied by Poggio et al [26]. The CG formula is in fact affected by multiple biases: overestimation with high BMI [16], imprecision with poor glucose control [17], and the influence of the AER as noted here, all contributing to a marked inaccuracy. A lower underestimate (MDRD) or a greater overestimate (CG) of low GFRs inevitably leads to an underestimate of GFR decline [11-13], although this is masked by the CG because it has a larger drop off in e-GFR for every year of age than the other equations. The new MCQ was less affected by this tendency, as expected, because this equation was derived from the results of subjects with and without CKD [27].

Although less documented, the influence of the AER on the GFR prediction error could in fact be expected: creatinine clearance is known to overestimate GFR in highly proteinuric CKD as in the nephrotic syndrome [28,29], and also in type 2 diabetes mellitus patients with less pronounced levels of proteinuria [30]. In these studies, the overestimation also concerned the CG [30] and the MDRD [28] and was attributed to changes in the tubular secretion of creatinine: indeed, Kemperman et al [30] showed that the creatinine clearance to GFR ratio (and the CG/GFR ratio) was reduced with cimetidine administration, which blocks this tubular secretion; and the reduction tended to be more pronounced in micro- and macroalbuminuric patients. Our results suggest that this effect also concerns more advanced stages of CKD (GFR 90 mL/min per 1.73 m^2 in the report of Kemperman et al vs 60 in our patients), where the monitoring of kidney function becomes crucial.

Overestimating GFR when the AER is high has practical consequences in a condition involving a highly variable and treatment-sensitive degree of albuminuria, which is the case for diabetic nephropathy. The natural history of diabetic nephropathy associates a decline in GFR with a rise in AER: the AER-dependent overestimation of GFR will produce an underestimate of the decline in GFR with both the CG and MDRD [11-13]. On the other hand, if an active therapeutic approach reduces AER, the estimation of GFR will be improved, leading to less overestimated final GFR estimations, which might preclude detection of a benefit on the GFR decline. This effect may be of particular importance on regression of nephrotic proteinuria, as reported in 22% of patients with type 1 diabetes mellitus, with a half-reduced isotopic GFR decline [16], and in 25% of patients with type 2 diabetes mellitus [17]. These specific studies did not report how the remission altered the e-GFR decline; however, the Steno group [13] has later reported that the CG and MDRD "unacceptably" underestimated the GFR decline. In our study, both the CG and MDRD, which overestimated GFR according to the AER, failed to detect a relation between the progression of the AER and the decline in GFR.

By contrast, the MCQ estimation, which was not affected by the AER-related bias (and less by the GFR-related bias), could detect a relation between the AER progression and the e-GFR decline in our patients. This effect is important: None of the other potential progression predictors (arterial pressure, HbA_{1C}, hemoglobin, blood lipids) could be related to the e-GFR decline in our patients. The primary importance of the AER and its outcome on the e-GFR decline has been emphasized by Perkins et al [31], who used 100/cystatin C to estimate the GFR decline in patients with type 1 diabetes mellitus. Interestingly, this group had shown that cystatin C was more sensitive in detecting the GFR decline of microalbuminuric type 2 diabetes mellitus Pima Indian subjects than the MDRD or the CG [32]. We did not include a cystatin C follow-up, but the less expensive MCQ estimation may be of value for monitoring kidney function. Although a little less accurate than the MDRD, it was less biased according to the GFR level; and the MCQ overestimation was not influenced by the AER. We have previously reported that the MCQ was more strongly (negatively) correlated with the AER [33]. These characteristics can contribute to a better detection of the influence of the AER on the e-GFR decline as we observed.

Several limitations of our study should be noted. We did not perform a final isotopic GFR determination that would have further validated the MCQ-estimated decline. The effect of the AER outcome on the MDRD decline almost reached significance in our patients, although we used the 4-variable abbreviated MDRD version. The original 6-variable MDRD includes the serum albumin level; its better accuracy may allow to detect the influence of the AER outcome on the e-GFR decline. Creatinine calibration also improves the performance of the MDRD equation [34], although this does not abolish the underestimation of high GFR [23,24,26,27,34]. A cystatin C–based outcome study might have led to more accurate results than the MCQ: cystatin-based equations are less biased according to GFR than are creatinine-based predictive equations in diabetic patients [35], and we found no correlation between any of these equations' errors and the AER in 99 of our patients (unpublished result). However, we do not have a second cystatin C determination to evaluate the outcome.

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