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Arteriovenous Fistula Creation and Estimated Glomerular Filtration Rate Decline in Advanced Chronic Kidney Disease: a Matched Cohort Study

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ABSTRACT

Background

Kidney failure is associated with a high burden of morbidity and mortality. Previous studies have raised the possibility that arteriovenous fistula creation (AVF) may attenuate estimated glomerular filtration rate (eGFR) decline. This study aimed to compare eGFR decline in predialysis patients with AVF, matched to patients oriented towards peritoneal dialysis (PD).

Methods

Predialysis patients with AVF and those oriented toward PD were retrospectively matched using a propensity score. Time zero was defined as 'AVF creation date' for the AVF group and 'date when eGFR was closest to the matched patient's eGFR at AVF creation' for the PD group. Crude and predicted eGFR decline in AVF and PD groups were compared before and after time zero using mixed effect linear regressions.

Results

Sixty-one pairs were matched. Crude annual eGFR decline pre AVF creation / time zero was -4.1 ml/min/m^2/year in the AVF group versus -5.3 ml/min/m^2/year in the PD group (p=0.75) and after time zero, -2.5 ml/min/m^2/year in the AVF group versus -4.5 ml/min/m^2/year in the PD group (p=0.02). The predicted annual decline decreased from -5.1 ml/min/m^2/year in the AVF group before AVF creation to -2.8 ml/min/m^2/year after (p<0.01) while there was no difference in the PD group (-5.5 versus -5.1 ml/min/m^2/year respectively, p=0.41).

Conclusions

In this matched study, AVF creation was associated with a deceleration of kidney function decline compared to a control PD-oriented group. Prospective studies are needed to assess potential mechanisms between vascular access creation and eGFR slope attenuation.

Introduction

The incidence of chronic kidney disease (CKD) has increased in the recent years. More than 124,000 patients reached end-stage kidney disease (ESKD) in the United States in 2017 and the vast majority started hemodialysis (HD) [1]. Patients on HD are at higher risk of mortality and have lower quality of life compared to CKD patients [2, 3]. Moreover, hemodialysis is associated with a high burden on health care economics [4]. Known risk factors associated with faster decline in estimated glomerular filtration rate (eGFR) included young age, proteinuria and high blood pressure [5, 6]. In contrast, use of angiotensin II receptor blockers (ARB) or angiotensin-converting enzyme (ACE) inhibitors, lowering of high blood pressure, glycemic control and acidosis correction have been shown to attenuate eGFR decline [7]. Timely reference for vascular access creation is a cornerstone in predialysis care with guidelines suggesting patients should be referred for arteriovenous fistula (AVF) creation when their eGFR reach 15-20 ml/min/1.73m^2 [8].

Recent studies suggested that AVF creation could slow eGFR decline[9-12]. The major limitation of these studies was the absence of control group (or one that was poorly comparable to the AVF group) and the inability to adjust for confounding factors. Thus, it remains unclear if the attenuation in eGFR slope is due to beneficial effect of AVF, the natural history of late CKD, or an artefact caused by the equations used to evaluate eGFR that performs less well at low kidney function level. This study aimed to evaluate kidney function decline before and after AVF creation compared to a control group of patients oriented towards peritoneal dialysis (PD). It was hypothesized that patients in the AVF group would have a slower eGFR decline after AVF creation than matched PD patients.

Material and methods

Study design and population

All patients included in this matched observational single-center study were followed in a tertiary predialysis care clinic between January 2002 and July 2019. Patients were included in the AVF group if they were older than 18 years old, had a patent native AVF created during predialysis follow-up and at least two documented eGFR values in the 6-month period before and after AVF creation. AVF patency was defined as presence of a thrill reported by a nephrologist or successful use of AVF at hemodialysis initiation. Patients were included in the PD group if they were older than 18 years old, and had a peritoneal catheter installed during predialysis follow-up. In both groups, exclusion criteria were history of any other access creation in the 6-month period before AVF or PD catheter installation and a previous kidney transplantation. This study adhered to the Declaration of Helsinki and was approved by the Research Ethic Board from Maisonneuve-Rosemont Hospital (#15094), without requirement for individual patient consent considering the retrospective observation design.

Data collection, measurements and outcomes

Potentially eligible patients were identified by archival personnel using AVF surgical creation (1.KY.76.LA) and peritoneal catheter surgical installation (1.OT.53.DA-TS, 1.OT.53.LA-TS, 1.OT.53.HA-TS) Canadian Classification of Health Interventions codes. All clinical data were obtained from electronic and paper charts. Demographic data including, age, sex, race, CKD etiology, height and comorbidities were identified at time of access creation. Serum creatinine was recorded monthly, as available, from study initiation (up to 12 months before AVF creation) until the end of follow-up, defined as dialysis initiation, death, kidney transplantation or 12 months after access creation, whichever came first. The use of renin-angiotensin-aldosterone system (RAAS)

inhibitors and diuretics was also documented, as well as the amount of albuminuria, when available. The study primary outcome was the difference in crude and predicted eGFR after time zero (AVF creation or matched point) in the AVF compared to PD group.

Statistical analysis

Baseline characteristics are presented as number and percentages for categorical variables, and median and interquartile ranges (IQR) for continuous variables. Categorical variables were compared using $\chi 2$ test and continuous variables were compared using Mann-Whitney U tests. Monthly eGFR was calculated using Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation [13]. Logistic regressions were used to calculate propensity scores for AVF versus PD catheter groups using age, sex, race, diabetes and eGFR. Propensity score matching was then used to match PD-oriented patients to AVF patients using greedy nearest-neighbor matching algorithm 1:1. Time zero was defined by 'AVF creation date' for the AVF group and 'date when eGFR was closest to the matched patient's eGFR at AVF creation' in the PD group. Matching could not be based on eGFRs at time of PD catheter installation since this intervention was performed at lower eGFRs than AVF creation, which may have influenced the subsequent natural history to CKD progression. Matched pairs without an eGFR measure < 4 months before AVF creation or with >2 mL/min/1.73m^2 difference in eGFR values at match point were excluded.

Differences in crude eGFR annual decline before and after AVF creation/time zero were compared in each group using paired t-tests. Wilcoxon-Mann-Whitney tests were used to assess between groups difference in crude eGFR annual decline before and after time zero. Systolic and diastolic blood pressure (BP) were assessed in the subgroup of patients who remained in predialysis at the end of the 12-month post 'time zero' period. Paired t-tests were used to compare BP at the visit immediately before 'time zero' and the 12-month end of study visit.

Mixed effect linear regressions were used to predict eGFR before and after time zero in both groups, with patient-level random intercepts and random slopes for time. Predicted annual decline for each group (AVF and PD) and each period (pre- and post-match) were estimated using univariate mixed effect models. Paired t-tests and Wilcoxon-Mann-Whitney test were respectively used to compare within group and between group differences in predicted eGFR annual decline (pre- / post-time zero). A multivariable mixed effect linear regression was also performed with the following three main variable: follow-up time (in month), group (AVF and PD) and period (pre- and post-time 0). Two-way and 3-way interactions between time, period and group were included to examine their effect on predicted eGFR.

An additional analysis was performed to take into account potential residual confounding factors (despite propensity score matching) with inclusion of covariates with p-value <0.2 in the univariate models in the main mixed-effect model. Sensitivity analysis were performed with an extended follow-up of up to 24 months and with the 75 initially matched patients. SAS version 9.4 (SAS Institute, Cary, NC, USA) was used to conduct all analysis. N.E. performed the analyses.

Results

One hundred and eighty-one patients met inclusion criteria for the AVF group and 79 in the PD group. Seventy-five pairs were initially matched 1:1 according to the variables already described. Of these, four pairs were excluded due to lack of creatinine measured in the four-month period preceding AVF creation and 10 more pairs were excluded because closest eGFR at time zero was more than 2 mL/min/1.73m^2 discordant. Sixty-one pairs were included in the main analysis (**Figure S1**).

Baseline characteristics of both groups are shown in **Table 1.** Median age was 64 years old (IQR 53-75) in the AVF group and 61 years old (IQR 54-70) in the PD group (p=0.47). Patients oriented towards PD had a lower body mass index (BMI) than the AVF patients, 25.4 kg/m^2 (IQR 23.1-28.5) versus 28.6 kg/m^2 (IQR 25.1-32.1, p<0.001) and a trend towards less cardiovascular disease (31% vs. 47%, p=0.06). There was no difference in use of RAAS inhibitors. Median duration of follow-up was 11 months (IQR 10-12) before AVF creation and 10 months (IQR 8-12) after in the AVF group, compared to 9 months (IQR 3-11) before match and 11 months (IQR 5.5-12) after in the PD group. Reasons for end of follow-up are shown in **Table 2**. In the AVF group, 5% patients died and 39% started dialysis while in the PD group, no patient died and 79% started dialysis within 12 months. Median eGFR at AVF creation/time zero was 12.8 (IQR 10.5-14.0) and 12.3 (IQR 10.6-14.0) ml/min/1.73m^2 in AVF and PD groups, respectively. Median number of eGFR observations was 6 (IQR 5-7) pre versus 5 (IQR 4-7) post time zero in the AVF group, and 4 (IQR 3-7) pre versus 5 (IQR 4-7) post in the PD group. Of note, median eGFR at time of PD catheter installation was 9.2 (8.1-10.7) ml/min/1.73m^2. In patients still followed in predialysis at the end of the study period, systolic BP was lower 12 months after AVF creation compared to immediately before (149 mmHg [IQR 136-158] pre, versus 141 mmHg [IQR 128-153] post, p=0.03) while diastolic BP remained similar (80 mmHg [IQR 70-87] pre, versus 76 mmHg [IQR 69-83] post, p=0.16). In contrast, there was no statistically significant difference in systolic BP (142 [IQR 120-159] pre, versus 142 mmHg [IQR 132-163] post, p=0.27) or diastolic BP (77 mmHg [70-87] pre, versus 76 mmHg [70-83] post, p=0.29) in the PD-oriented group.

Crude eGFR

Crude annual eGFR decline pre AVF creation/'time zero' was -4.1 ml/min/1.73m^2/year in the AVF group and -5.3 ml/min/1.73m^2/year in the PD group (p=0.75). After time zero, the annual eGFR

decline was -2.5 ml/min/1.73m^2/year in the AVF group and -4.5 ml/min/1.73m^2/year in the PD group (p=0.02)**.** The mean difference in annual eGFR decline between pre- and post-time AVF creation/time zero was 1.7 ml/min/1.73m^2/year in the AVF group (p=0.003) and 0.9 ml/min/1.73m^2/year in the PD group (p=0.87) (**Figure 1a)**.

Predicted eGFR

Predicted kidney function slopes for each group, pre- and post-intervention/time zero, are shown in **Figure 2**. The predicted annual decline decreased from -5.1 ml/min/1.73m^2/year in the AVF group before AVF creation to -2.8 ml/min/1.73m^2/year after (p<0.01). In contrast, eGFR decline pre- and post-time zero was similar in the PD group (-5.5 ml/min/1.73m^2/year versus -5.1 ml/min/1.73m^2/year respectively, p=0.41) (**Figure 1b).**

Results of the mixed effect model including a 3-way interaction term (group, period and time) are displayed in **Table 3**. This analysis estimated eGFR changes, taking into account the group (AVF or PD), period (before/after time zero) and follow-up time (in months). It showed that the attenuation of eGFR decline was statistically significant in the AVF group after the intervention, compared to before and to the PD group, with an estimated sparing of 0.14 ml/min/1.73m^2 each month after time zero (p=0.02), corresponding to 1.72 ml/min/1.73m^2 per year. More concretely, predicted monthly eGFR decline was -0.37 ml/min/1.73m^2/month before AVF creation and -0.22 ml/min/1.73m^2/month after, while predicted pre- and post-decline were respectively -0.41 ml/min/1.73m^2/month and -0.41 ml/min/1.73m^2/month in the PD group. Results of predicted eGFR mixed effect models, stratified by AVF and PD groups are presented in **Table 4.**

Sensitivity analysis

To account for potential residual imbalances after propensity score matching, an additional analysis was performed with further adjusted for sex, age, RAAS inhibitors use and furosemide use at time AVF creation /match. Results were consistent and are shown in supplementary **Table S1** and **Figure S2**. Additionally, analyses using up to 24-month follow-up and the 75 initially matched pair, also showed similar associations for crude and predicted eGFR decline (Supplementary **Tables S2-S3** and **Figures S3 a-c.**

Discussion

In this propensity-scored matched predialysis cohort study, kidney function decline was statistically significantly attenuated in patients with AVF creation compared to the matched PD-oriented group. Crude yearly eGFR decline decreased by 1.7ml/min/1.73m^2 (p=0.003) after access creation in the AVF patients (compared to before) and only by 0.9 ml/min/1.73m^2 (p=0.87) in PD-oriented patients. There results were consistent across several statistical analyses, using crude eGFR and predicted eGFR estimations.

These results are concordant with other recently published observational studies showing association between AVF creation and eGFR decline. Golper and colleagues first observed this association in 123 CKD patients with AVF creation. They reported a -5.9 ml/min/1.73m^2/month and a –0.46 ml/min/1.73m^2/month eGFR decline pre and post AVF creation respectively [9]. The slope of the eGFR decline was statistically different before and after AVF creation (p=0.001), although unadjusted for other potential confounders. A large cohort study, published by Sumida and colleagues, compared over 6,000 veteran patients with AVF/arteriovenous graft (AVG) creation or tunneled central venous catheter (CVC) prior to hemodialysis initiation. In their adjusted models, the median eGFR slope for the AVF/AVG group before and after the surgical procedure was -18.1 and -8.3 ml/min/1.73m^2/year respectively. In the CVC group, eGFR slopes

before and after the index date (6 months prior hemodialysis initiation) were -20.6 and -58.8 ml/min/1.73m^2/year respectively [10]. The presence of a control group strengthens the observed association between the access creation and the eGFR decline attenuation, although one could argue that patients who start hemodialysis with a CVC are inherently sicker, explaining the observed steeper decline in eGFR.

Our group recently published a similar study with data adjusted for key confounding factors (RAAS inhibition, age and comorbidities), further confirming the observed association between AVF creation and CKD decline attenuation. This single-center study included 146 patients with AVF creation during their predialysis follow-up. The eGFR decline decreased from -3.6 ml/min/1.73m^2 pre-intervention to -2.28 ml/min/1.73m^2 post-intervention [11]. In the mixed effect model, eGFR decline was more attenuated each month after AVF creation. However, the lack of control group remained a major limitation for these findings.

In contrast to the three prior studies, Lundström and colleagues reported similar eGFR decline in patients with AVF creation compared to those with PD catheter installation [12]. Alike the present study, this Swedish study had the advantage of a 'healthy' control group mitigating potential confounding that might have been present in the study from Sumida and colleagues where comparator was CVC patients. In the Swedish study, kidney function decline was less steep after AVF creation than before (-5.6 vs. -1.6 ml/min/1.73m^2/year, $p<0.01$). However, the same observation was made in the PD catheter group where the decline went from -6.7 to -2.17 ml/min/1.73m^2/year before and after the surgery respectively ($p<0.01$). This is in contrast to the present study findings. However, two notable differences between the two studies could explain the inconsistencies in results. First, index time eGFR was drastically lower in the Swedish cohort: 8.1 ml/min/1.73m^2 in the AVF group and 7.0 ml/min/1.73m^2 in the PD group, compared to 12.3

ml/min/1.73m^2 in both groups in the present study. If there is truly a physiological benefit to AVF creation, it is possible that it can only occur before the disease is too advanced. Second, the follow-up was more than thrice longer in the present study (12 months vs. 100 days) and might have allowed finding an association that was not observed with the shorter follow-up in the study by Lundström and colleagues. On the other hand, it remains possible that the current study overestimates the association between AVF creation and eGFR decline attenuation due to a smaller cohort size.

Kidney transplant recipient is another relevant population to assess the effect of AVF on cardiovascular and renal outcomes. Weekers and colleagues reported outcomes of 285 kidney transplant recipients, of whom 114 had an AVF ligature and where slopes of eGFR decline were steeper after AVF closure [14]. However, this study contrasts other studies that suggest a neutral effect of AVF ligation on cardiac parameters (left ventricular ejection fraction, left ventricular hypertrophy and cardiac index) [15, 16], and others showing deleterious associations between AVF creation and kidney graft function [17].

Physiological plausibility underlying potential benefit of AVF creation have already been described.[9, 11, 18] Briefly, ischemic preconditioning effect and change in cardiovascular hemodynamics may be involved. Ischemic preconditioning comes from the finding that short periods of upper or lower limb ischemia can induce remote protection of other organs, such as the myocardium [19, 20]. More recently, ischemic preconditioning was shown to reduce acute kidney injury in patients undergoing cardiac surgery[21-23]. In patient with AVF, vasodilatation in the contralateral arm through non-endothelial pathways has been described[24], suggesting a systemic vasodilatory effect of AVF creation. In this regard, a recent meta-analysis shows a significant reduction of BP following AVF creation in ESKD patients[25]. AVF may also alter kidney function

through hemodynamics effects, as an AVF is considered a high flow, low-resistance and high-compliance compartment added to the cardiovascular system[26]. Conflicted data on cardiovascular hemodynamics following AVF creation exists. It has been shown that AVF creation reduces arterial stiffness and blood pressure, while also increasing left ventricular ejection fraction, which are all beneficial to the kidney function[27, 28]. Additionally, the resultant increase in venous return has been proposed to promote blood flow in underperfused lung areas, which may increase blood oxygen delivery to the kidneys, decreasing the renal chemoreflex responsible for vasoconstriction through central sympathetic activation.[18]

AVF creation has also been linked with an increased myocardial oxygen demand[29] and left ventricular hypertrophy[30], and an increase in excess pressure integral, in turn associated with adverse cardiovascular outcomes[31]. However, these changes are only described in ESKD patients, where a longstanding abnormal development of the AVF may contribute to adverse hemodynamic changes.

Aside for any potential effect on eGFR, AVF is the preferred vascular access for hemodialysis [8]. Indeed, multiple studies have shown association between CVC and increased mortality from cardiovascular and infectious causes [32-34]. Native fistulas have been associated with fewer hospitalisation from infectious causes [35], septicemia [36] and central vein stenosis [37]. Consequently, the vast majority of predialysis patient with progressive CKD should be referred for vascular access creation when eGFR is between 15-20 ml/min/1.73m^2 [8]. However, in 2001, according to the CHOICE study, only 25% incident patients started hemodialysis with an AVF [32]. In 2017, 16.8% of patients in the United States started dialysis with a matured AVF and an additional 15% started through a catheter with a maturing AVF [1]. These observations concur that although AVF is the preferred hemodialysis access, actual practices often do not follow these guidelines. Knowing that

AVF creation can potentially delay CKD progression is another reason why the nephrology community should make additional efforts to follow guidelines.

In all cases, vascular access choice should be personalize based on each patient's characteristics [8]. Patients with heart failure can be at increased risk of AVF-related adverse events. AVF has been associated with a right ventricular diastolic dysfunction and dilatation, potentially correlated to worsened volume overload with AVF [38]. High out-put cardiac failure can occur especially in association with high flow AVF, generally with an access flow higher than 2000 ml/min[39]. This high left-to-right shunt leads to cardiac volume overload, which in turn leads to high cardiac demand, cardiac hypertrophy and ultimately classical high output heart failure [39]. Ligation or reduction of AVF in patients with high-flow access may lead to reduction in left ventricular end-diastolic diameter [40]. Moreover, AVF is associated with pulmonary hypertension, a progressive and potentially fatal cardiopulmonary condition that is improved by ligation of AVF [41].

This study has important strengths. Patients with AVF were compared to a peritoneal dialysis-oriented control group, often considered the healthiest predialysis population [42, 43]. Using a propensity-score matched model allowed to decrease the confounding effect of age, sex, race and comorbid conditions. Patients also had a median index time eGFR between 10 and 15 ml/min/1.73m^2, reflecting a not too advanced CKD. Finally, results were consistent in crude and adjusted analysis, as well as in various sensitivity models.

Several limitations must be outlined. First, the relatively small sample size limited the study power and the ability to assess specific subgroups who might benefit more (or less) from AVF creation. Importantly, patients in the PD group may have been subjected to a survivor bias considering they had to be alive with CKD until PD catheter installation. The retrospective design precludes any

causality assumption between the intervention and eGFR decline attenuation. Of note, it is possible that creation of AVF increased patient's awareness on the gravity of their CKD condition, while this realization only came later at time of catheter installation in the PD group. There was also residual imbalance between the study groups, including higher BMI in the AVF group, which could have introduced a bias. Nonetheless, the main finding remained similar in a model with multivariable adjustment. Finally, generalization of the results could be questioned due to the single-center design although other studies led to similar conclusion, suggesting benefits might be extend to different populations.

In conclusion, this study found that predialysis patients in this cohort had a slower kidney decline after AVF creation than matched PD-oriented patients, pointing towards a potential association between AVF creation and preservation of kidney function. Considering the limitations of observational data, multicentre trials should be performed to further assess this hypothesis, considering the psychosocial, economic and clinical benefits of residual kidney function preservation in advanced CKD.

Disclosures

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TABLES

Table 1. Baseline characteristics for matched patients

Variable	PD group n=61	AVF group n=61	p-value
Age, years - median (IQR)	61 (54 - 70)	64 (53 - 75)	0.47
Sex (male) - n (%)	37 (61)	37 (61)	1.00
BMI, kg/m^2 - median (IQR)*	25.4 (23.1 - 28.5)	28.6 (25.1 - 32.1)	<0.001
Race - n (%)			1.00
Black	7 (12)	7 (12)	
Caucasian / Other	54 (88)	54 (88)	
Year of 'Time zero'			0.10
2002-2011	24 (39)	33 (54)	
2012-2019	37 (61)	28 (46)	
Comorbidities - n (%)			
Hypertension	59 (97)	57 (93)	0.68
Diabetes	24 (39)	25 (41)	0.85
Cardiovascular disease	19 (31)	29 (47)	0.06
Chronic obstructive pulmonary disease	6 (10)	10 (16)	0.28
Heart failure	9 (15)	6 (10)	0.40
Active smoking - n (%)**	12 (22)	12 (20)	0.56
Primary Kidney disease - n (%)			0.83
Hypertensive disease	15 (25)	15 (25)	
Diabetic nephropathy	14 (23)	19 (31)	
Glomerulonephritis	16 (26)	14 (23)	
Others	16 (26)	13 (21)	
Furosemide use - n (%)	28 (46)	30 (49)	0.72
ACEi/ARB use - n (%)	44 (72)	44 (72)	1.00

All variables defined at time of AVF creation or match time, * 7 and 12 missing for PD and AVF groups respectively, ** 7 missing for PD group, PD: peritoneal dialysis, AVF: arteriovenous fistula, IQR: interquartile range, BMI: body mass index, ACEi: angiotensin converting enzyme inhibitor, ARB: angiotensin receptor blocker

Table 2 – Status at end of follow-up

Status at 12 months	AVF group, n (%)	PD group, n (%)
Death	3 (4.9)	0
Dialysis initiation	24 (39.3)	48 (78.7)
Kidney transplantation	2 (3.3)	0
Pre dialysis	32 (52.5)	13 (21.3)

Table 3. Adjusted mixed effect linear regression model adjusted for follow-up time, group and follow-up period

Variables	***Beta estimate***	***95% CI***	**p-value**
Intercept	12.29	11.51 ; 13.08	<0.001
Follow-up time (*per month*)	-0.41	-0.52 ; -0.31	<0.001
Group			
PD	0		
AVF	-0.55	-1.62 ; 0.51	0.31
Period			
Pre match	0		
Post match	-0.34	-0.83 ; 0.15	0.17
Group * follow-up time (*per month*)			
PD	0		
AVF (*per month*)	0.05	-0.09 ; - 0.18	0.49
Period * follow-up time (*per month*)			
Pre match	0		
Post match	0.006	-0.09 ; 0.10	0.9
Period * group			
Pre match – PD	0		
Pre match – AFV	0		
Post match – PD	0		
Post match – AVF (*per month*)	0.94	0.26 ; 1.61	0.006
Period * group * follow-up time (*per month*)			
Pre match – PD	0		
Pre match – AVF	0		
Post match – PD	0		
Post match – AVF (*per month*)	0.14	0.03 ; 0.26	0.02

PD: peritoneal dialysis, AVF: arteriovenous fistula.

Table 4. **Adjusted mixed effect linear regression model stratify by group (AVF and PD) and adjusted for follow-up time and follow-up period**

	PD		AVF	
Variables	*Beta estimate*	p-value	*Beta estimate*	p -value
Intercept	12.30	<0.001	11.74	<0.001
Follow-up time (per month)	-0.41	<0.001	-0.37	<0.001
Period				
Pre match	0		0	
Post match	-0.34	0.17	0.59	0.02
Period * follow-up time (per month)				
Pre match	0		0	
Post match (per month)	-0.001	0.98	0.152	<0.001

PD, peritoneal dialysis; AVF, arteriovenous fistula

Figure 1.

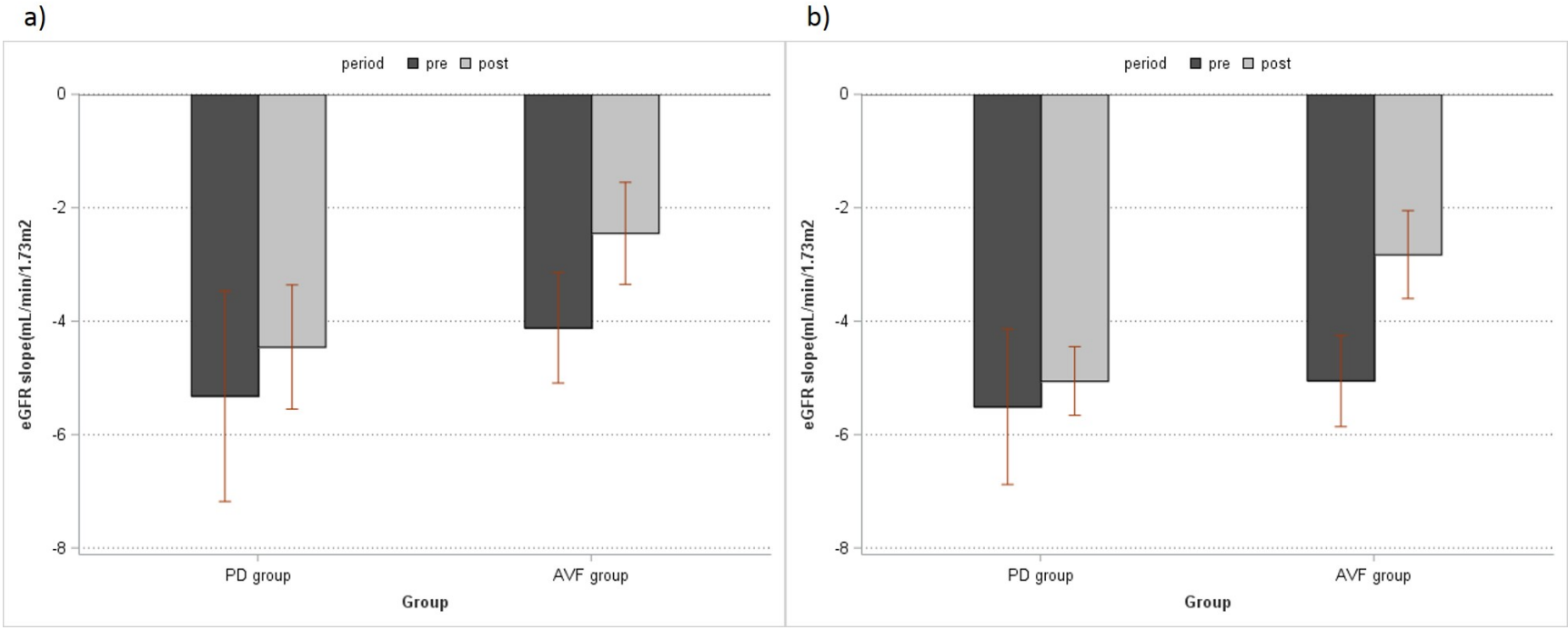

Figure 1. Differences in yearly kidney function decline according to group and time using (a) crude estimated glomerular filtration rate and (b) predicted estimated glomerular filtration rate. PD: peritoneal dialysis; AVF: arteriovenous fistula.

Figure 2.

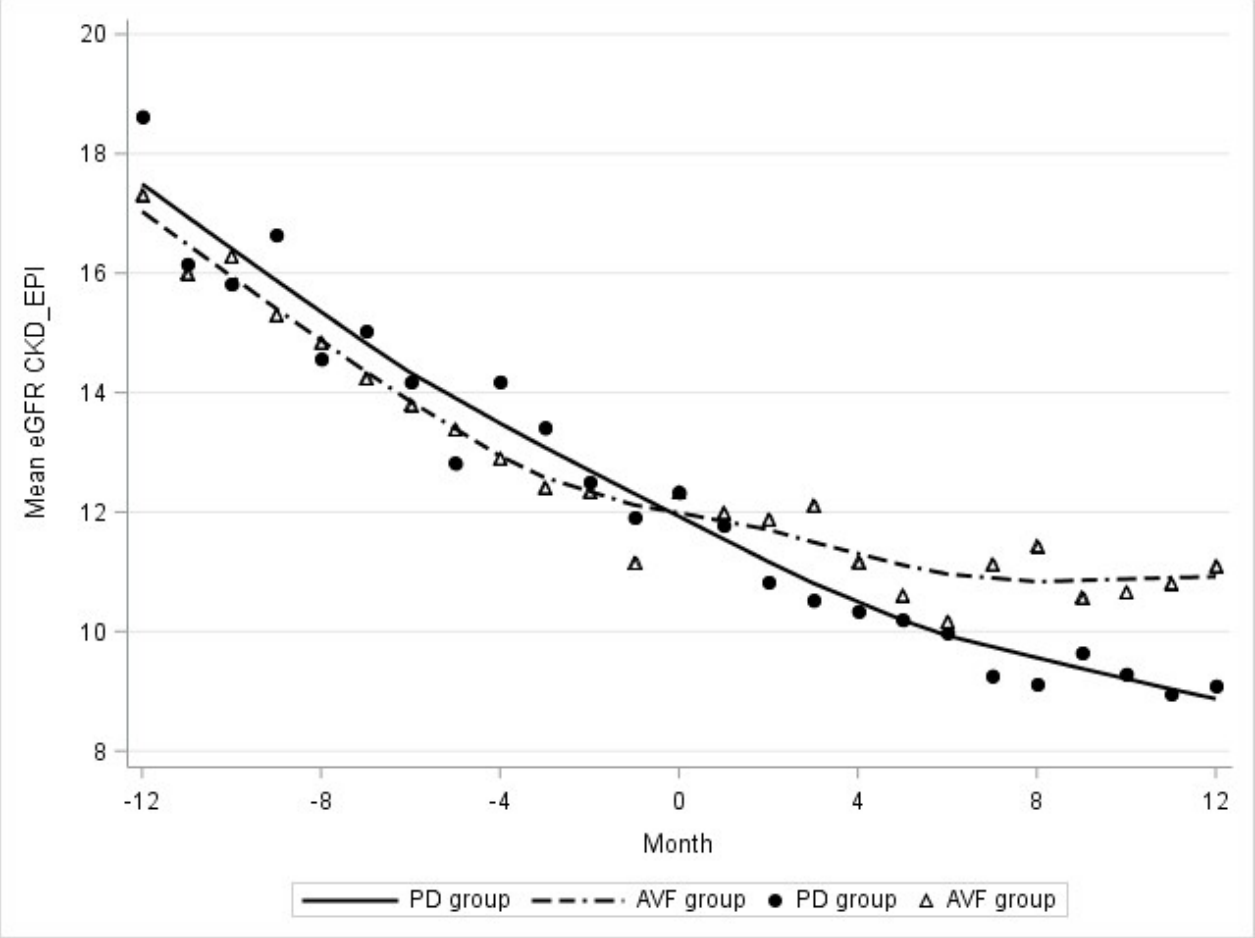

Figure 2. Crude (dots) and predicted (lines) eGFR slopes in AVF and PD-oriented patients before and after 'Time zero'.