

Arteriovenous Fistula Creation and Estimated Glomerular Filtration Rate Decline in Advanced CKD: 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 (AVF) creation may attenuate eGFR decline. This study aimed to compare eGFR decline in predialysis patients with an AVF, matched to patients oriented toward peritoneal dialysis (PD).

Methods Predialysis patients with an AVF and those oriented toward PD were retrospectively matched using a propensity score. Time zero was defined as the “AVF creation date” for the AVF group and the “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 In total, 61 pairs were matched. Crude annual eGFR decline before AVF creation/time zero was -4.1 ml/min per m^2 per year in the AVF group versus -5.3 ml/min per m^2 per year in the PD group ($P=0.75$) and after time zero, -2.5 ml/min per m^2 per year in the AVF group versus -4.5 ml/min per m^2 per year in the PD group ($P=0.02$). The predicted annual decline decreased from -5.1 ml/min per m^2 per year in the AVF group before AVF creation to -2.8 ml/min per m^2 per year after ($P<0.01$), whereas there was no difference in the PD group (-5.5 versus -5.1 ml/min per m^2 per year respectively, $P=0.41$).

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

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Introduction

The incidence of CKD has increased in recent years. More than 124,000 patients reached 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 with patients with CKD (2,3). Moreover, HD is associated with a high burden on health care economics (4). Known risk factors associated with faster decline in eGFR included young age, proteinuria, and high BP (5,6). In contrast, use of angiotensin II receptor blockers or angiotensin-converting enzyme inhibitors, lowering of high BP, 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 reaches 15–20 ml/min per 1.73 m^2 (2,8).

Recent studies suggested AVF creation could slow eGFR decline (9–12). The major limitation of these studies was the absence of a 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 the beneficial effect of AVF, the natural history of late CKD, or an artifact caused by the equations used to evaluate eGFR that performs less well at a low kidney function level. This study aimed to evaluate the kidney function decline before and after AVF creation, compared with a control group of patients oriented toward 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

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older than 18 years, 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 the presence of a thrill, reported by a nephrologist, or successful use of AVF at HD initiation. Patients were included in the PD group if they were older than 18 years, and had a peritoneal catheter installed during predialysis follow-up. In both groups, the exclusion criteria were a 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 a 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 the 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, and 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 with the PD group.

Statistical Analyses

Baseline characteristics are presented as the numbers and percentages for categorical variables, and median and interquartile ranges (IQR) for continuous variables. Categorical variables were compared using chi-squared test and continuous variables were compared using Mann–Whitney *U* tests. Monthly eGFR was calculated using CKD Epidemiology Collaboration 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 patients who were PD oriented to patients with AVF using a greedy nearest-neighbor matching algorithm 1:1. Time zero was defined by the 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 on the basis of the eGFR at the time of PD catheter installation because 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 per 1.73 m² 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-group differences in crude eGFR annual decline before and after time zero. Systolic and diastolic BP were assessed in the subgroup of patients who remained in predialysis at the end of the 12-months after the 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 postmatch) was estimated using univariate mixed-effect models. Paired *t* tests and Wilcoxon Mann–Whitney tests 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 variables: follow-up time (in months), group (AVF and PD), and period (pre and post-time zero). Two-way and three-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 was 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) was used to conduct all analysis. N.E. performed the analyses.

Results

A total of 181 patients met the inclusion criteria for the AVF group and 79 in the PD group. Initially 75 pairs were matched 1:1, according to the variables already described. Of these, four pairs were excluded due to lack of creatinine measured in the 4-month period preceding AVF creation and ten more pairs were excluded because the closest eGFR at time zero was more than 2 ml/min per 1.73 m² discordant. Finally, 61 pairs were included in the main analysis (Supplemental Figure 1).

Baseline characteristics of both groups are shown in Table 1. The 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 toward PD had a lower body mass index than AVF patients, 25.4 kg/m² (IQR, 23.1–28.5) versus 28.6 kg/m² (IQR, 25.1–32.1, *P*<0.001) and a trend toward less cardiovascular disease (31% versus 47%, *P*=0.06). There was no difference in use of RAAS inhibitors. The 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 with 9 months (IQR, 3–11) before match and 11 months (IQR, 5.5–12) after in the PD group. Reasons for the end of follow-up are shown in Table 2. In the AVF group, 5% patients died and 39% started dialysis, whereas in the PD group, no patient died and 79% started dialysis within 12 months. The median eGFR at AVF

Table 1. Baseline characteristics for matched patients

Variable	Peritoneal Dialysis Group, n=61	Arteriovenous Fistula Group, n=61	P Value
Age, yr, 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) ^a	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)	
Yr 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 (%) ^b	12 (22)	12 (20)	0.56
Primary kidney disease, n (%)			0.83
Hypertensive disease	15 (25)	15 (25)	
Diabetic nephropathy	14 (23)	19 (31)	
GN	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. IQR, interquartile range; BMI, body mass index; ACEi, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker.
^aSeven and 12 missing for the PD and AVF groups, respectively.
^bSeven missing for the PD group.

creation/time zero was 12.8 (IQR, 10.5–14.0) and 12.3 (IQR, 10.6–14.0) ml/min per 1.73 m² in the AVF and PD groups, respectively. The median number of eGFR observations was six (IQR, 5–7) before versus five (IQR, 4–7) after time zero in the AVF group, and four (IQR, 3–7) before versus five (IQR, 4–7) after in the PD group. Of note, median eGFR at time of PD catheter installation was 9.2 (8.1–10.7) ml/min per 1.73 m². In patients still followed in predialysis at the end of the study period, systolic BP was lower 12 months after AVF creation compared with immediately before (149 mm Hg [IQR, 136–158] before, versus 141 mm Hg [IQR, 128–153] after, $P=0.03$) whereas diastolic BP remained similar (80 mm Hg [IQR, 70–87] before, versus 76 mm Hg [IQR, 69–83] after, $P=0.16$). In contrast, there was no statistically significant difference in systolic BP (142 [IQR, 120–159] before, versus 142 mm Hg [IQR, 132–163] after, $P=0.27$) or diastolic BP (77 mm Hg [IQR, 70–87] before, versus 76 mm Hg [IQR, 70–83] after, $P=0.29$) in the PD-oriented group.

Crude eGFR

Crude annual eGFR decline pre AVF creation/time zero was -4.1 ml/min per 1.73 m² per year in the AVF group

and -5.3 ml/min per 1.73 m² per year in the PD group ($P=0.75$). After time zero, the annual eGFR decline was -2.5 ml/min per 1.73 m² per year in the AVF group and -4.5 ml/min per 1.73 m² per 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 per 1.73 m² per year in the AVF group ($P=0.003$) and 0.9 ml/min per 1.73 m² per year in the PD group ($P=0.87$) (Figure 1A).

Predicted eGFR

Predicted kidney function slopes for each group, pre and postintervention/time zero, are shown in Figure 2. The predicted annual decline decreased from -5.1 ml/min per 1.73 m² per year in the AVF group before AVF creation to -2.8 ml/min per 1.73 m² per year after ($P<0.01$). In contrast, eGFR decline pre and post-time zero was similar in the PD group (-5.5 versus -5.1 ml/min per 1.73 m² per year respectively, $P=0.41$) (Figure 1B).

Results of the mixed-effects model including a three-way interaction term (group, period, and time) are displayed in Table 3. This analysis estimated eGFR changes, taking into

Table 2. Status at the end of follow-up

Status at 12 Mo	Arteriovenous Fistula Group, n (%)	Peritoneal Dialysis Group, n (%)
Death	3 (4.9)	0
Dialysis initiation	24 (39.3)	48 (78.7)
Kidney transplantation	2 (3.3)	0
Predialysis	32 (52.5)	13 (21.3)

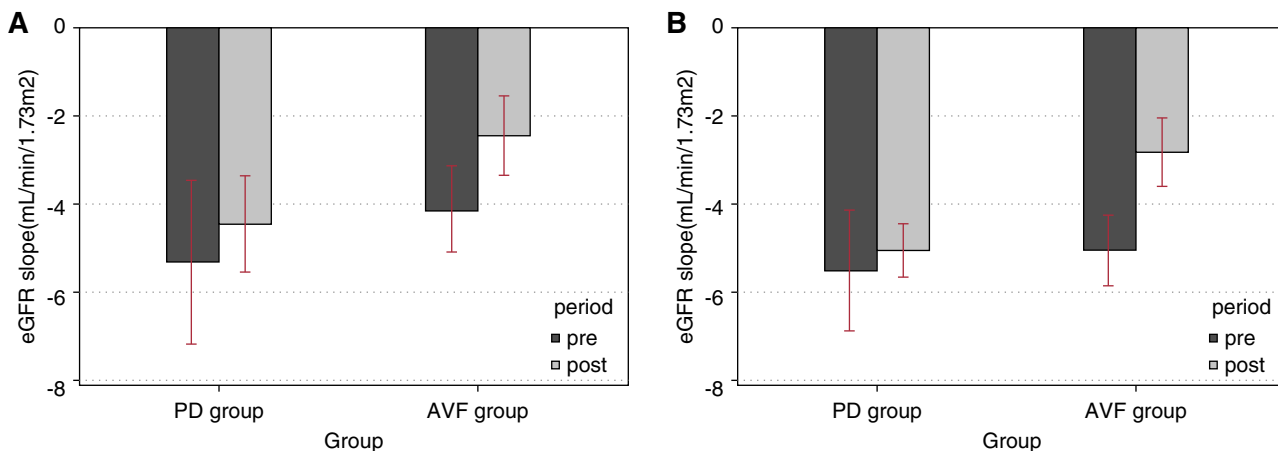


Figure 1. | Differences in yearly kidney function decline according to group and time using (A) crude eGFR and (B) predicted eGFR. PD, peritoneal dialysis; AVF, arteriovenous fistula.

account the group (AVF or PD), period (before/after time zero), and follow-up time (in months). It showed the attenuation of eGFR decline was statistically significant in the AVF group after the intervention, compared with before and to the PD group, with an estimated sparing of 0.14 ml/min per 1.73 m² each month after time zero ($P=0.02$), corresponding to 1.72 ml/min per 1.73 m² per year. More concretely, predicted monthly eGFR decline was -0.37 ml/min per 1.73 m² per month before AVF creation and -0.22 ml/min per 1.73 m² per month after, whereas predicted before and after decline were -0.41 ml/min per 1.73 m² per month and -0.41 ml/min per 1.73 m² per month, respectively, in the PD group. Results of the 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, additional analysis was performed, further adjusted for sex, age, RAAS inhibitor use, and

furosemide use at the time of AVF creation/match. The results were consistent and are shown in Supplemental Figure 2 and Supplemental Table 1. Additionally, analyses using up to 24-month follow-up and the 75 initially matched pairs also showed similar associations for crude and predicted eGFR decline (Supplemental Figures 3, A–C, Supplemental Tables 2 and 3).

Discussion

In this propensity-scored matched predialysis cohort study, kidney function decline was statistically significantly attenuated in patients with AVF creation compared with the matched PD-oriented group. Crude yearly eGFR decline decreased by 1.7 ml/min per 1.73 m² ($P=0.003$) after access creation in the AVF patients (compared with before) and only by 0.9 ml/min per 1.73 m² ($P=0.87$) in PD-oriented patients. These 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 patients who had CKD with AVF creation. They reported a -5.9 ml/min per 1.73 m² per month and a -0.46 ml/min per 1.73 m² per 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 6000 veteran patients with AVF/arteriovenous graft creation or tunneled central venous catheter (CVC) before HD initiation. In their adjusted models, the median eGFR slope for the AVF/arteriovenous graft group before and after the surgical procedure was -18.1 and -8.3 ml/min per 1.73 m² per year respectively. In the CVC group, eGFR slopes before and after the index date (6 months before HD initiation) were -20.6 and -58.8 ml/min per 1.73 m² per year, respectively (10). The presence of a control group strengthens the observed association between the access creation and the

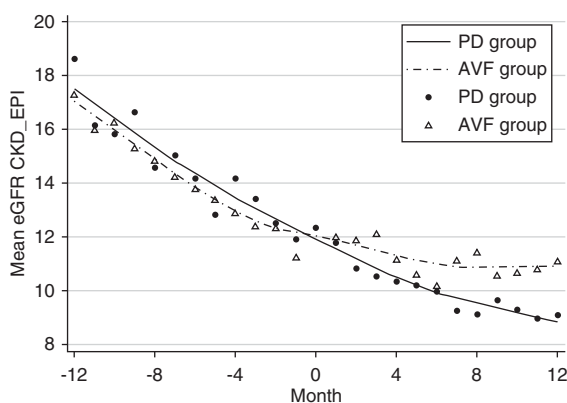


Figure 2. | Crude (dots) and predicted (lines) eGFR slopes in patients with AVF and patients who were PD oriented before and after time zero. PD, peritoneal dialysis; AVF, arteriovenous fistula; CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration.

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

Variable	Beta Estimate	95% Confidence Interval	P Value
Intercept	12.29	11.51 to 13.08	<0.001
Follow-up time (per mo)	-0.41	-0.52 to -0.31	<0.001
Group			
PD	0		
AVF	-0.55	-1.62 to 0.51	0.31
Period			
Prematch	0		
Postmatch	-0.34	-0.83 to 0.15	0.17
Group * follow-up time (per mo)			
PD	0		
AVF (per mo)	0.05	-0.09 to -0.18	0.49
Period * follow-up time (per mo)			
Prematch	0		
Postmatch	0.006	-0.09 to 0.10	0.9
Period * group			
Prematch PD	0		
Prematch AVF	0		
Postmatch PD	0		
Postmatch AVF (per mo)	0.94	0.26 to 1.61	0.006
Period * group * follow-up time (per mo)			
Prematch PD	0		
Prematch AVF	0		
Postmatch PD	0		
Postmatch AVF (per mo)	0.14	0.03 to 0.26	0.02

PD, peritoneal dialysis; AVF, arteriovenous fistula.

eGFR decline attenuation, although one could argue that patients who start HD 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 per 1.73 m² preintervention to -2.28 ml/min per 1.73 m² postintervention (11). In the mixed-effects 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 *et al.* (12) reported a similar eGFR decline in patients with AVF creation compared with those with PD catheter installation. As with this study, the Swedish study had the advantage of

a healthy control group, mitigating potential confounding that might have been present in the study from Sumida *et al.* where the comparator was patients with CVC. In the Swedish study, kidney function decline was less steep after AVF creation than before (-5.6 versus -1.6 ml/min per 1.73 m² per 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 per 1.73 m² per year before and after the surgery, respectively ($P < 0.01$). This is in contrast to this study's findings. However, two notable differences between the studies could explain the inconsistencies in results. First, the index time eGFR was drastically lower in the Swedish cohort: 8.1 ml/min per 1.73 m² in the AVF group and 7.0 ml/min per 1.73 m² in the PD group, compared with 12.3 ml/min per 1.73 m² in both groups in this study. If there is truly a physiologic benefit to AVF creation, it is possible it can only occur before the disease is too advanced. Second, the follow-up was more than three times

Table 4. Adjusted mixed effect linear regression model stratify by group (arteriovenous fistula and peritoneal dialysis) and adjusted for follow-up time and follow-up period

Variable	Peritoneal Dialysis		Arteriovenous Fistula	
	Beta Estimate	P Value	Beta Estimate	P Value
Intercept	12.30	<0.001	11.74	<0.001
Follow-up time (per mo)	-0.41	<0.001	-0.37	<0.001
Period				
Prematch	0		0	
Postmatch	-0.34	0.17	0.59	0.02
Period * follow-up time (per mo)				
Prematch	0		0	
Postmatch (per mo)	-0.001	0.98	0.152	<0.001

longer in this study (12 months versus 100 days) and might have allowed the finding of an association that was not observed with the shorter follow-up in the study by Lundström *et al.* In contrast, it remains possible that this study overestimates the association between AVF creation and eGFR decline attenuation due to a smaller cohort size.

Kidney transplant recipients are another relevant population to assess the effect of AVF on cardiovascular and renal outcomes. Weekers *et al.* (14) reported outcomes of 285 kidney transplant recipients, of whom 114 had an AVF ligation and where slopes of eGFR decline were steeper after AVF closure. 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).

Physiologic plausibility underlying potential benefit of AVF creation has 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 AKI in patients undergoing cardiac surgery (21–23). In patients with AVF, vasodilation in the contralateral arm through nonendothelial 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 after AVF creation in patients with ESKD (25). AVF may also alter kidney function through hemodynamic effects, as an AVF is considered a high-flow, low-resistance, and high-compliance compartment added to the cardiovascular system (18). Conflicted data on cardiovascular hemodynamics after AVF creation exists. It has been shown that AVF creation reduces arterial stiffness and BP, while also increasing left ventricular ejection fraction, which are all beneficial to the kidney function (26,27). 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 (28) and left ventricular hypertrophy (29), and an increase in the excess pressure integral, in turn associated with adverse cardiovascular outcomes (30). However, these changes are only described in patients with ESKD, where a longstanding abnormal development of the AVF may contribute to adverse hemodynamic changes.

Aside from any potential effect on eGFR, AVF is the preferred vascular access for HD (8). Indeed, multiple studies have shown an association between CVC and increased mortality from cardiovascular and infectious causes (31–33). Native fistulas have been associated with fewer hospitalizations from infectious causes (34), septicemia (35), and central vein stenosis (36). Consequently, the vast majority of patients who are predialysis and have progressive CKD should be referred for vascular access creation when eGFR is between 15 and 20 ml/min per 1.73 m² (2,8). However, in

2001, according to the Choices for Healthy Outcomes in Caring for End-Stage Renal Disease study, only 25% of incident patients started HD with an AVF (31). 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 HD 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 patients, vascular access choice should be personalized on the basis of 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 dilation, potentially correlated to worsened volume overload with AVF (37). High output cardiac failure can occur, especially in association with high-flow AVF, generally with an access flow higher than 2000 ml/min (38). This high left-to-right shunt leads to cardiac volume overload, which in turn leads to high cardiac demand, cardiac hypertrophy, and ultimately classic high output heart failure (38). Ligation or reduction of AVF in patients with high-flow access may lead to a reduction in the left ventricular end-diastolic diameter (39). Moreover, AVF is associated with pulmonary hypertension, a progressive and potentially fatal cardiopulmonary condition that is improved by ligation of the AVF (40).

This study has important strengths. Patients with AVF were compared with a PD-oriented control group, often considered the healthiest predialysis population (41,42). Using a propensity-score-matched model allowed a decrease in the confounding effect of age, sex, race, and comorbid conditions. Patients also had a median index time eGFR between 10 and 15 ml/min per 1.73 m², reflecting CKD that was not too advanced. Finally, results were consistent in crude and adjusted analysis, and 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 creating the AVF increased the patients' awareness of the gravity of their CKD condition, whereas this realization only came at the time of catheter installation in the PD group. There was also a residual imbalance between the study groups, including a higher body mass index in the AVF group, which could have introduced a bias. Nonetheless, the main finding remained similar in a model with multivariable adjustment. Finally, a generalization of the results could be questioned due to the single-center design, although other studies led to similar conclusions, suggesting benefits might be extended to different populations.

In conclusion, this study found that patients who were predialysis in this cohort had a slower kidney decline after AVF creation than matched patients who were PD oriented, pointing toward a potential association between AVF

creation and preservation of kidney function. Considering the limitations of observational data, multicenter 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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Author Contributions

A.-C. Nadeau-Fredette was responsible for the conceptualization, methodology, supervision, and writing review and editing; J.-P. Lafrance, R. Goupil, and V. Pichette were responsible for the conceptualization and writing review and editing; L.-P. Laurin was responsible for the visualization and writing review and editing; M. Pichette and V. Bénard were responsible for the data curation; M.-È. Dupuis curated the data and wrote the original draft; N. Elftouh was responsible for the formal analysis and methodology; and all authors approved the final manuscript.

Supplemental Material

This article contains the following supplemental material online at <http://kidney360.asnjournals.org/lookup/suppl/doi:10.34067/KID.0005072020/-/DCSupplemental>.

Supplemental Table 1. Yearly predicted eGFR decline adjusted for RAASi, age, sex and furosemide use with 12 month follow-up

Supplemental Table 2. Comparison of yearly decline using crude eGFR, unadjusted mixed model predicted eGFR and adjusted mixed model predicted eGFR, with a 24 months follow-up.

Supplemental Table 3. Comparison of yearly decline using crude eGFR, unadjusted mixed model predicted eGFR and adjusted mixed model predicted eGFR, with all 72 marched pairs.

Supplemental Figure 1. Study flow chart.

Supplemental Figure 2. Yearly predicted eGFR decline in PD and AVF groups, adjusted for RAASi, age, sex and furosemide use with 12-month follow-up.

Supplemental Figure 3. Comparison of yearly decline using (a) crude eGFR, (b) unadjusted mixed model predicted eGFR and (c) adjusted mixed model predicted eGFR, with a 24-month follow-up.

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