End-stage kidney disease (ESKD) is a significant public health problem, with 678,383 patients reported prevalent in the United States in December 2014. Among the options for kidney replacement therapy, kidney transplantation is preferred because it is associated with better patient outcomes and lower costs. However, only 2.6% of incident patients with ESKD received a preemptive kidney transplant, indicating that the majority of patients must receive dialysis for variable periods of time before they can receive a transplant, with center hemodialysis being the predominant modality in the United States. Hemodialysis requires a vascular access such as an arteriovenous fistula (AVF), arteriovenous graft (AVG), or tunneled central venous catheter (CVC).

Several studies have established AVFs as the preferred access in hemodialysis patients. A recent systematic review and meta-analysis by Ravani et al included 67 studies and showed that patients using CVCs had 53% higher all-cause mortality, more than twice as many fatal infections, and 38% higher risk for cardiovascular events compared with patients with AVFs. The study also reported worse outcomes with hemodialysis catheter access when compared with AVGs, with 38% higher mortality, 49% more fatal infection rates, and 26% higher cardiovascular event rates. Hence, catheters were associated with poorer outcomes compared with both AVFs and AVGs. When directly comparing AVGs with AVFs, grafts were noted to have worse clinical outcomes, with 18% higher all-cause mortality and 36% higher rates for fatal infection, although no difference was reported for cardiovascular outcomes.

When patients on hemodialysis receive a kidney transplant, AVFs and AVGs remain in place, whereas CVCs are usually removed. However, the putative effects of dialysis access type on clinical outcomes after transplantation have not been studied. Such associations could plausibly exist. Studies have found higher levels of inflammatory markers in transplant...
recipients with CVCs and AVGs. The increased cardiac output caused by a patent AVF and AVG may impose greater cardiovascular burden and contribute to poorer outcomes in transplant recipients. Overall, the effect of vascular access pretransplantation on outcomes posttransplantation is not known and needs to be better understood.

We therefore conducted this study to determine whether posttransplantation outcomes of first-time kidney transplant recipients differed by type of vascular access used for the last outpatient hemodialysis treatment before the kidney transplantation surgery.

**METHODS**

This study adheres to the Declaration of Helsinki and was approved by institutional review boards at Stanford University School of Medicine (protocol #17904) and Baylor College of Medicine (protocol #H-36408). The need for informed consent was waived owing to the use of deidentified data. The clinical and research activities being reported are consistent with the principles of the Declaration of Istanbul as outlined in the Declaration of Istanbul on Organ Trafficking and Transplant Tourism.

**Sources of Data**

We used individual-level data from 2 merged databases to conduct this study: (1) the US Renal Data System (USRDS), the national registry of patients with ESKD; and (2) the electronic health records of a large dialysis organization. Following approval by the Institutional Review Board at Stanford University School of Medicine and a Data Use Agreement from the National Institute for Diabetes and Digestive and Kidney Diseases (NIDDK), the databases were cross-linked using a Health Insurance Portability and Accountability Act (HIPAA)-compliant approach.

The USRDS contains detailed information about the timing and modalities used for the treatment of ESKD, information for sociodemographics and comorbid conditions at onset of ESKD treatment, and detailed kidney transplant–related information from the United Network for Organ Sharing. The electronic health records of the large dialysis organization contain detailed information on each dialysis session provided within their facilities, as well as results of laboratory tests conducted in their patients.

**Study Population**

We conducted a retrospective cohort study of hemodialysis patients receiving a first kidney transplant. Patients who underwent their first kidney transplantation between 2006 and 2011 were included in the study if they had received at least 30 days of in-center hemodialysis in the facilities of the large dialysis organization (Fig 1).

**Exposure of Interest**

We identified the hemodialysis access used during the most recent outpatient dialysis treatment before these patients’ transplantation surgeries from the electronic health records, in which this information is documented by clinical staff in each dialysis facility for each patient and each session. We categorized these into AVF, AVG, and CVC.

**Covariates: Other Patient Characteristics**

We defined each patient’s age (at transplantation), sex, race (white, black, and other), Hispanic ethnicity, and residential Census Division from the USRDS, which also provided information for several comorbid conditions and body mass index at the time of ESKD. We additionally identified several laboratory measurements from the 30 days preceding the transplantation, as well as donor and recipient characteristics commonly used in kidney transplant outcomes research. Table 1 lists all characteristics used for this study.
## Table 1. Patient Characteristics Before First Kidney Transplant, According to Most Recent HD Access Type Used, 2006-2011

| Comorbid conditions                        | AVF | AVG | CVC | \( P \) |
|--------------------------------------------|-----|-----|-----|---------|
| Diabetes (13 missing)                      | 3,217 (53.1\%) | 1,190 (62.9\%) | 689 (52.0\%) | <0.001  |
| Hypertension (<10 missing)                 | 5,989 (98.7\%) | 1,885 (99.6\%) | 1,315 (99.0\%) | 0.008  |
| Heart failure (21 missing)                 | 2,789 (46.1\%) | 968 (51.2\%) | 602 (45.5\%) | <0.001  |
| Arteriosclerotic heart disease (20 missing)| 1,814 (30.0\%) | 674 (35.6\%) | 375 (28.3\%) | <0.001  |
| Cerebrovascular disease (22 missing)       | 784 (12.9\%) | 311 (16.4\%) | 199 (15.0\%) | <0.001  |
| Peripheral vascular disease (22 missing)   | 1,761 (29.1\%) | 772 (40.8\%) | 405 (30.6\%) | <0.001  |
| History of malignancy (22 missing)         | 558 (9.2\%) | 196 (10.4\%) | 125 (9.4\%) | 0.33    |
| BMI, kg/m\(^2\) (429 missing)             | 112 (1.9\%) | 36 (2.0\%) | 43 (3.4\%) | <0.001  |
| Time since ESKD, y (median (IQR); mean (SD)) | 3.8 [2.4-5.4]; 4.2 (2.5) | 4.6 [3.3-6.6]; 5.1 (2.9) | 3.4 [1.6-5.2]; 3.7 (2.7) | <0.001  |
| Donor age, y (138 missing)                | 39.2 (15.9) | 39.4 (16.0) | 38.7 (15.0) | 0.38    |
| Donor male sex                             | 3,421 (56.4\%) | 1,093 (57.7\%) | 750 (56.4\%) | 0.59    |
| Donor race                                 | 0.02 |
| Donor type                                 | <0.001  |
| Deceased                                   | 5,008 (82.5\%) | 1,687 (89.1\%) | 872 (65.6\%) |  |
| Living                                     | 1,060 (17.5\%) | 207 (10.9\%) | 457 (34.4\%) |  |
| Cold ischemia time (1,054 missing)         | <0.001  |
| <12 h                                      | 2,064 (38.1\%) | 553 (32.6\%) | 557 (49.5\%) |  |
| 12-24 h                                    | 2,427 (44.8\%) | 828 (48.8\%) | 418 (37.1\%) |  |
| >24 h                                      | 925 (17.1\%) | 314 (18.5\%) | 151 (13.4\%) |  |
| HLA antigen mismatch (529 missing)         | <0.001  |
| 0                                          | 419 (73\%) | 113 (6.3\%) | 95 (76\%) |  |
| 1-3                                        | 1,320 (23.0\%) | 336 (18.8\%) | 354 (28.5\%) |  |
| 4-6                                        | 3,989 (69.6\%) | 1,342 (74.9\%) | 794 (63.9\%) |  |
| Recipient peak PRA (1,445 missing)         | <0.001  |
| 0%-10%                                     | 3,730 (72.9\%) | 1,084 (65.7\%) | 748 (69.3\%) |  |
| 11%-80%                                    | 1,072 (20.9\%) | 411 (24.9\%) | 235 (21.8\%) |  |
| >80%                                       | 315 (6.2\%) | 155 (9.4\%) | 96 (8.9\%) |  |
| Recipient ABO blood type                   | 0.001  |
| O                                          | 2,975 (49.0\%) | 868 (45.8\%) | 592 (44.5\%) |  |
| A                                          | 1,990 (32.8\%) | 627 (33.1\%) | 472 (35.5\%) |  |
| B                                          | 874 (14.4\%) | 327 (17.3\%) | 197 (14.8\%) |  |
| AB                                         | 229 (3.8\%) | 72 (3.8\%) | 68 (5.1\%) |  |

(Continued)
Outcomes

Patients were followed up from the date of kidney transplantation until the following outcomes: (1) all-cause mortality; (2) allograft loss from all causes as indicated by return to dialysis, retransplantation, or death; and (3) allograft loss from cause other than death (return to dialysis or retransplantation). All analyses censored patient follow-up at the end of the study period (December 31, 2011). Determination of all outcomes and censoring events was made through standard data fields from the “Patient” file in the USRDS.

Statistical Analyses

We used standard descriptive statistics to characterize the 3 exposure groups by the last known dialysis access before the kidney transplantation. Continuous variables are presented as median with interquartile range or mean (standard deviation), and categorical variables, as count (percentage). Any differences among the vascular access groups were identified using analysis of variance or Kruskal-Wallis tests for continuous variables and Pearson $\chi^2$ or Fisher exact tests for categorical variables. We used cause-specific Cox-proportional hazards regression to challenge the null hypotheses of no differences in study outcomes among the categories of last dialysis access used before kidney transplantation. All models were stratified by calendar year of the transplantation. The adjusted models included demographic characteristics, comorbid conditions, transplant-related variables, and laboratory results. Each of these variable categories was added in incremental adjustment steps with the final model simultaneously accounting for all the factors. For allograft loss from causes other than death, we conducted analyses in 2 ways: (1) using death as a competing risk, and (2) using death as a censoring event. The ensuing results were essentially identical; hence, we only presented results that censored for death. In regression analyses, missing data were addressed with multiple imputation by chained equation using the MICE package in R. A total of 27.4% of patients had at least 1 variable missing, with the percentage of missing ranging from $<0.01\%$ (race) to 15.6% (recipient panel-reactive antibody). There was no reason to believe that the data would be related to unobserved characteristics, Therefore, we assumed the data to be missing at random and performed multiple imputation by
those with AVFs in unadjusted or adjusted models. Rates of all-cause allograft loss that were no different from attenuated and no longer significant in the fully adjusted model (1.31; 95% CI, 1.17-1.48), but the association was attenuated and no longer present in a model adjusted for demographics, comorbid conditions, transplant variables. However, adjustment for laboratory values once again rendered both associations null.

Table 2. Association Between Last Vascular Access Used Pretransplantation and All-Cause Mortality

| Unadjusted | HR  | 95% L | 95% U | P |
|------------|-----|-------|-------|---|
| AVG vs AVF | 1.33 | 1.15 | 1.54 | <0.01 |
| CVC vs AVF | 0.99 | 0.83 | 1.18 | 0.91 |
| Demographics-adjusted | | | | |
| AVG vs AVF | 1.27 | 1.09 | 1.48 | <0.01 |
| CVC vs AVF | 1.07 | 0.89 | 1.28 | 0.48 |
| + Comorbid conditions added | | | | |
| AVG vs AVF | 1.13 | 0.97 | 1.33 | 0.11 |
| CVC vs AVF | 1.05 | 0.88 | 1.26 | 0.60 |
| + Transplant variables added | | | | |
| AVG vs AVF | 1.16 | 0.99 | 1.36 | 0.06 |
| CVC vs AVF | 1.10 | 0.92 | 1.32 | 0.31 |
| + Laboratory results added | | | | |
| AVG vs AVF | 1.13 | 0.97 | 1.33 | 0.12 |
| CVC vs AVF | 1.00 | 0.83 | 1.21 | 0.99 |

Note: HRs estimated using Cox proportional hazards regression models stratified for calendar year of transplantation; results shown are based on multiply imputed data (N = 9,291; m = 32 sets).

Last, we studied the association between the last vascular access used pretransplantation and allograft loss from causes other than death (Table 4). AVG use was associated with a 30% higher rate of allograft loss (HR, 1.30; 95% CI, 1.10-1.53), and CVC use was associated with a 22% higher rate of allograft loss (HR, 1.22; 95% CI, 1.01-1.47), both compared with AVFs in the unadjusted model. The associations were maintained, albeit attenuated, after adjustment for demographic, comorbid condition, and transplant variables. However, adjustment for laboratory values once again rendered both associations null.

Figure 2. Actuarial cumulative incidence of all-cause mortality and all-cause allograft loss. Solid lines: all-cause allograft loss; dotted lines: mortality. Abbreviations: AV, arteriovenous; CVC, central venous catheter.
compared with those with AVFs. Similar evidence was based partly on the established association of grafts otherwise similar patients who had AVFs. This expectation was partly on the established association of grafts versus fistulas in the hemodialysis population, but more importantly on studies demonstrating that the presence of AVGs was associated with markers of systemic inflammation.22

Our main a priori hypothesis was that kidney transplant recipients with AVGs would have worse outcomes than otherwise similar patients who had AVFs. This expectation was based partly on the established association of grafts versus fistulas in the hemodialysis population, but more importantly on studies demonstrating that the presence of AVGs was associated with markers of systemic inflammation.22

A study by Wasse et al8 of 91 patients undergoing hemodialysis showed that patients with retained AVGs had higher concentrations of inflammatory markers, namely C-reactive protein, interleukin 6, and tumor necrosis factor. These patients lacked clinical evidence of previous infection or inflammation and hence retained AVGs were considered to be the source of the inflammatory markers in these patients. Additionally, the study also reported an association between elevated C-reactive protein levels and erythropoietin resistance (P = 0.003), which associates with cardiovascular morbidity in these patients.

Other studies have found similar results. Banerjee et al7 analyzed participants in the CHOICE (Choices of Healthy Outcomes in Caring for End-Stage Renal Disease) prospective cohort study and found that the presence of an AVG was associated with a significant 30% increase in C-reactive protein levels. Thus, a similar phenomenon was hypothesized to occur post-transplantation when an AVG left in place could possibly lead to elevated inflammation and contribute to worse outcomes in these patients.

Another explanation for the possibility of worse outcomes in patients with AVGs relates to the presence of subclinical vascular graft infections in these patients post-transplantation. This has been reported in prospective studies and case series of hemodialysis patients and kidney transplant recipients and is particularly concerning in immunocompromised patients after kidney transplantation. We did not study episodes of unexplained infections in our cohort and were unable to study inflammatory markers other than those routinely measured in hemodialysis clinics, namely, white blood cell count, platelet count, and albumin and ferritin levels. Among the

### Table 3. Association Between Last Vascular Access Used Pretransplantation and Allograft Loss From All Causes

|               | Unadjusted HR 95% L 95% U P | Demographics-adjusted HR 95% L 95% U P | Comorbid conditions added HR 95% L 95% U P | Transplant variables added HR 95% L 95% U P | Laboratory results added HR 95% L 95% U P |
|---------------|-------------------------------|----------------------------------------|------------------------------------------|-------------------------------------------|------------------------------------------|
| AVG vs AVF    | 1.31 1.17 1.48 <0.01          | 1.34 1.10 1.62 <0.01                  | 1.21 1.00 1.46 0.05                      | 1.19 1.00 1.37 0.02                      | 1.13 1.00 1.28 0.05                     |
| CVC vs AVF    | 1.09 0.95 1.25 0.23           | 1.20 0.99 1.50 0.04                   | 1.21 1.00 1.46 0.05                      | 1.21 1.00 1.37 0.02                      | 1.17 1.00 1.31 0.02                     |

### Table 4. Association Between Last Vascular Access Used Pretransplantation and Allograft Loss From Causes Other Than Death

|               | Unadjusted HR 95% L 95% U P | Demographics-adjusted HR 95% L 95% U P | Comorbid conditions added HR 95% L 95% U P | Transplant variables added HR 95% L 95% U P | Laboratory results added HR 95% L 95% U P |
|---------------|-------------------------------|----------------------------------------|------------------------------------------|-------------------------------------------|------------------------------------------|
| AVG vs AVF    | 1.30 1.10 1.53 <0.01          | 1.22 1.01 1.47 0.04                   | 1.21 1.00 1.46 0.05                      | 1.19 1.00 1.37 0.02                      | 1.17 1.00 1.31 0.02                     |
| CVC vs AVF    | 1.09 0.95 1.25 0.23           | 1.20 0.99 1.50 0.04                   | 1.21 1.00 1.46 0.05                      | 1.21 1.00 1.37 0.02                      | 1.17 1.00 1.31 0.02                     |

**DISCUSSION**

Using a large cohort of first-time kidney transplant recipients who had previously received hemodialysis, we examined whether outcomes differed by type of vascular access used for hemodialysis. We did not find any compelling evidence that all-cause mortality or allograft survival differed among the 3 groups of hemodialysis vascular access after we accounted for differences in patient characteristics.

This study was motivated by established evidence on differences in outcomes across access types in patients undergoing hemodialysis. The associations of type of vascular access used in hemodialysis patients with important patient outcomes are well established. Several studies have shown higher mortality and worse cardiovascular outcomes in patients with CVCs and AVGs compared with those with AVFs. Similar evidence for any differences in the outcomes of new kidney transplant recipients was unavailable. Patients undergoing kidney transplantation usually retain their peripheral vascular access (CVCs are usually removed after the transplant is considered functional) and these remain patent for variable periods and may induce chronic inflammation or impose long-term cardiovascular burden in these recipients.

Higher concentrations of inflammatory markers, namely C-reactive protein, interleukin 6, and tumor necrosis factor. These patients lacked clinical evidence of previous infection or inflammation and hence retained AVGs were considered to be the source of the inflammatory markers in these patients. Additionally, the study also reported an association between elevated C-reactive protein levels and erythropoietin resistance (P = 0.003), which associates with cardiovascular morbidity in these patients.

Other studies have found similar results. Banerjee et al analyzed participants in the CHOICE (Choices of Healthy Outcomes in Caring for End-Stage Renal Disease) prospective cohort study and found that the presence of an AVG was associated with a significant 30% increase in C-reactive protein levels. Thus, a similar phenomenon was hypothesized to occur post-transplantation when an AVG left in place could possibly lead to elevated inflammation and contribute to worse outcomes in these patients.

Another explanation for the possibility of worse outcomes in patients with AVGs relates to the presence of subclinical vascular graft infections in these patients post-transplantation. This has been reported in prospective studies and case series of hemodialysis patients and kidney transplant recipients and is particularly concerning in immunocompromised patients after kidney transplantation. We did not study episodes of unexplained infections in our cohort and were unable to study inflammatory markers other than those routinely measured in hemodialysis clinics, namely, white blood cell count, platelet count, and albumin and ferritin levels. Among the
limited markers available, there was no difference in white blood cell counts or the acute-phase protein ferritin.

Observational studies on clinical outcomes by vascular access type are prone to selection bias. In patients undergoing hemodialysis, it has been shown that patients with AVFds are relatively less sick compared with those with AVGs. It has been shown throughout these studies that patients with CVCs and, to a lesser degree, AVGs have a higher burden of comorbid conditions, and adjusted associations were usually attenuated compared with unadjusted findings. Thus, one must assume that the well-described associations in the hemodialysis population are only partly causal and that there is (perhaps considerable) residual confounding present by the inability to perfectly measure, quantitatively and qualitatively, all relevant comorbidities and conditions. In kidney transplant recipients, for whom eligibility for this procedure serves as an “equalizer” and restricts the range of comorbid conditions acceptable, one would expect less confounding by comorbid conditions. However, our findings illustrate that there were still considerable differences in comorbidity burden across vascular access groups, with patients in the AVG group generally being sicker than patients in the other 2 groups. The average time since ESKD was also longer in patients with AVGs (5.1 years) compared with those with AVFs (4.2 years), which makes sense given that patients are more likely to use up their native vessel options as duration of hemodialysis treatment increases. The impact of these differences is illustrated in the sequentially adjusted models, in which most of the confounding for the comparison of AVGs versus AVFs was driven by adjustment for comorbid conditions, with almost no changes after adjustment for transplant-related factors and laboratory measurements.

Interestingly, patients using CVCs had the shortest time since ESKD incidence, 3.7 years. It is possible that patients who expect to receive a transplant quickly, for example, from a living donor, opt to have a more temporary access solution rather than to have a surgically created fistula or graft. Some might also have run out of options for vascular access, prompting them to actively look out for live kidney donors. The proportion of living donors was much greater in patients using a hemodialysis CVC (34.4%) than in AVF or AVG users (17.5% and 10.9%, respectively). Patients using CVCs pretransplantation had similar rates of all-cause mortality compared with patients with AVFs, an unexpected outcome because patients’ CVCs are usually removed soon after kidney transplantation, typically within a week, after graft function is recovered. By contrast, our study identified an association of CVC use with increased risk for allograft loss from causes other than death compared with patients using AVFs. This finding was unexpected and cannot easily be explained. It is possible that this association is spurious or by chance. Future research needs to refute or corroborate this specific association.

Certain limitations of our study require discussion. In this retrospective analysis of routinely collected data from mandated reporting to the Centers for Medicare & Medicaid Services and the electronic health records of a national dialysis provider, the associations identified may be residually confounded. Patients were not randomly assigned to different access types and so confounding by indication, information bias, and ascertainment bias are possible due to the nature of data collection. We have accounted for the potential confounders in the statistical model, though residual confounding by unobserved characteristics, or imperfectly measured characteristics, could still be present. Because laboratory parameters measured pretransplantation could be a downstream consequence of vascular access choice, it would have been possible that adjustment for these laboratory markers as potential mediators would lead to potential over-adjustment. However, the absence of any major changes in estimated associations indicates that laboratory factors did not confound the associations of interest. We also lacked more specific data for inflammatory markers, such as C-reactive protein and interleukin 6, which were used in the prior studies. Finally, the generalizability of our findings to other countries with different vascular access and transplantation practices is also uncertain.

In conclusion, contrary to studies in the hemodialysis population in which type of vascular access has been shown to be associated with important health outcomes, the present study does not provide convincing support for the hypothesis that type of vascular access pretransplantation is a strong determinant of post-transplantation outcomes.

ARTICLE INFORMATION

Authors’ Full Names and Academic Degrees: Medha Airy, MD, MPH, Colin R. Lenihan, MD, PhD, Victoria Y. Ding, MSc, Maria E. Montez-Rath, PhD, Jizhong Cheng, MD, Sankar D. Navaneethan, MD, MS, MPH, Haimanot Wasse, MD, MPH, and Wolfgang C. Winkelmayer, MD, MPH, ScD.

Authors’ Affiliations: Seizman Institute for Kidney Health, Section of Nephrology, Department of Medicine, Baylor College of Medicine, Houston, TX (MA, JZ, SDN, WCW); Division of Nephrology, Department of Medicine (CRL, MEM-R), and Center for Biomedical Informatics Research (VYD), Stanford University School of Medicine, Palo Alto, CA; and Division of Nephrology, Department of Medicine, Rush University School of Medicine, Chicago, IL (HW).

Address for Correspondence: Sankar D. Navaneethan, MD, MPH, Section of Nephrology, Baylor College of Medicine, One Baylor Plaza, Houston, TX 77030. E-mail: sankar.navaneethan@bcm.edu

Authors’ Contributions: Research idea and study design: MA, HW, WCW; data acquisition: WCW; data analysis/interpretation: MA, CRL, VYD, MEM-R, SDN, HW, WCW; statistical analysis: VYD, MEM-R; supervision or mentorship: WCW. Each author contributed important intellectual content during manuscript drafting or revision and accepts accountability for the overall work by ensuring that questions pertaining to the accuracy or integrity of any portion of the work are appropriately investigated and resolved.
Support: Data acquisition was supported by grants R01 DK090181 and R01 DK095024. Dr Cheng was supported by grant R01 DK095867.

Financial Disclosure: Dr Winkelmayer reports having served as a scientific advisor to Akebia, Amgen, AstraZeneca, Bayer, Daichii Sankyo, Relypsa, and Vifor Fresenius Medical Care Renal Pharma. Outside the submitted work, Dr Navaneethan has served on an independent event adjudication committee for clinical trials sponsored by Bayer and Boehringer Ingelheim, served as a consultant to Tricida and Reata pharmaceuticals, and received investigator-initiated research support from Keryx Biopharmaceuticals. The remaining authors declare that they have no relevant financial interests.

Disclaimer: This study was conducted under data use agreements between WCW and the NIDDK and DaVita Inc, respectively. Data reported here were supplied by the USRDS. Interpretation and reporting of these data are the responsibility of the authors and in no way should be seen as official policy or interpretation of the US government. An NIDDK peer reviewer, with direct editorial input from the Statistical Editor, Dr Winkelmayer reports having served as a consultant to Akebia and Reata Pharmaceuticals. Financial Disclosure: Dr Winkelmayer reports having served as a consultant to Tricida and Reata Pharmaceuticals. Outside the submitted work, Dr Navaneethan has served on an independent event adjudication committee for clinical trials sponsored by Bayer and Boehringer Ingelheim, served as a consultant to Tricida and Reata Pharmaceuticals, and received investigator-initiated research support from Keryx Biopharmaceuticals. The remaining authors declare that they have no relevant financial interests.

Peer Review: Received February 9, 2019. Evaluated by 1 external peer reviewer, with direct editorial input from the Statistical Editor, an Associate Editor, and the Editor-in-Chief. Accepted in revised form August 20, 2019.

REFERENCES

1. US Renal Data System. USRDS 2016 Annual Data Report: epidemiology of kidney disease in the United States. Am J Kidney Dis. 2017;69(3)(suppl 1):S1-S668.
2. Port FK, Wolfe RA, Mauger EA, Berling DP, Jiang K. Comparison of survival probabilities for dialysis patients vs cadaveric renal transplant recipients. JAMA. 1993;270(11):1339-1343.
3. Wolfe RA, Ashby VB, Milford EL, et al. Comparison of mortality in all patients on dialysis, patients on dialysis awaiting transplantation, and recipients of a first cadaveric transplant. N Engl J Med. 1999;341(23):1725-1730.
4. Ravani P, Palmer SC, Oliver MJ, et al. Associations between hemodialysis access type and clinical outcomes: a systematic review. J Am Soc Nephrol. 2013;24(3):465-473.
5. Almasri J, Alsawas M, Mainou M, et al. Outcomes of vascular access for hemodialysis: a systematic review and meta-analysis. J Vasc Surg. 2016;64(1):236-243.
6. Murad MH, Elamin MB, Sidaway AN, et al. Autogenous versus prosthetic vascular access for hemodialysis: a systematic review. J Vasc Surg. 2008;48(5)(suppl):S4-S75.
7. Dhingra RK, Young EW, Hulbert-Shearon TE, Leafey SF, Port FK. Type of vascular access and mortality in U.S. hemodialysis patients. Kidney Int. 2001;60(4):1443-1451.
8. Wasse H, Cardarelli F, De Staercke C, Hooper WC, Long Q. Accumulation of retained nonfunctional arteriovenous grafts correlates with severity of inflammation in asymptomatic ESRD patients. Nephrol Dial Transplant. 2013;28(4):991-997.
9. Banerjee T, Kim SJ, Astor B, Shafi T, Coresh J, Powe NR. Vascular access type, inflammatory markers, and mortality in incident hemodialysis patients: the Choices for Healthy Outcomes in Caring for End-Stage Renal Disease (CHOICE) study. Am J Kidney Dis. 2014;64(6):954-961.
10. How good are the data? USRDS data validation special study. Am J Kidney Dis. 1992;20(5)(suppl 2):68-88.
11. Montez-Rath ME, Winkelmayer WC, Desai M. Addressing missing data in clinical studies of kidney diseases. Clin J Am Soc Nephrol. 2014;9(7):1328-1335.
12. Pastan S, Soucie JM, McClellan WM. Vascular access and increased risk of death among hemodialysis patients. Kidney Int. 2002;62(2):620-626.
13. Ng LJ, Chen F, Pisoni RL, et al. Hospitalization risks related to vascular access type among incident US hemodialysis patients. Nephrol Dial Transplant. 2011;26(11):3659-3666.
14. Wasse H, Speckman RA, McClellan WM. Arteriovenous fistula use is associated with lower cardiovascular mortality compared with catheter use among ESRD patients. Semin Dial. 2008;21(5):483-489.
15. Xue H, Lacson E Jr, Wang W, Curhan GC, Brunelli SM. Choice of vascular access among incident hemodialysis patients: a decision and cost-utility analysis. Clin J Am Soc Nephrol. 2010;5(12):2289-2296.
16. Feldman HI, Held PJ, Hutchinson JT, Stoiber E, Hartigan MF, Berlin JA. Hemodialysis vascular access morbidity in the United States. Kidney Int. 1993;43(5):1091-1096.
17. Lacson E Jr, Wang W, Lazarus JM, Hakim RM. Change in vascular access and mortality in maintenance hemodialysis patients. Am J Kidney Dis. 2009;54(5):912-921.
18. Leake AE, Yuo TH, Wu T, et al. Arteriovenous grafts are associated with earlier catheter removal and fewer catheter days in the United States Renal Data System population. J Vasc Surg. 2015;62(1):123-127.
19. Basile C, Vernaglione L, Casucci F, et al. The impact of haemodialysis arteriovenous fistula on haemodynamic parameters of the cardiovascular system. Clin Kidney J. 2016;9(5):729-734.
20. Ori Y, Korzets A, Katz M, et al. The contribution of an arteriovenous access for hemodialysis to left ventricular hypertrophy. Am J Kidney Dis. 2002;40(4):745-752.
21. Iwashima Y, Horio T, Takami Y, et al. Effects of the creation of arteriovenous fistula on cardiac function and natriuretic peptide levels in CRF. Am J Kidney Dis. 2002;40(5):974-982.
22. Aching SG, Ayus JC. Inflammation from dialysis, can it be removed? Nephrol Dial Transplant. 2013;28(4):770-773.
23. Canaud B, Senecal L, Leray-Moragues H, et al. Vascular access, an underestimated source of inflammation in dialysis patients. Nephrology. 2003;24(7):353-358.
24. Goldstein SL, Ikizler TA, Zappitelli M, Silverstein DM, Ayus JC. Non-infected hemodialysis catheters are associated with increased inflammation compared to arteriovenous fistulas. Kidney Int. 2009;76(10):1063-1069.
25. Nassar GM, Ayus JC. Infectious complications of old nonfunctioning arteriovenous grafts in renal transplant recipients: a case series. Am J Kidney Dis. 2002;40(4):832-836.
26. Nassar GM, Fishbane S, Ayus JC. Occult infection of old nonfunctioning arteriovenous grafts: a novel cause of erythropoietin resistance and chronic inflammation in hemodialysis patients. Kidney Int Suppl. 2002;60:49-54.