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# Title page

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# Abstract page

Complete title: Pre-transplant dialysis modality does not influence short- or long-term outcome in kidney transplant recipients: Analysis of paired kidneys from the same deceased donor.

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**Keywords:** kidney transplantation; pre-transplant dialysis modality; hemodialysis; peritoneal dialysis; outcomes.

Abstract (200 words): Previous studies have reported contradictory results regarding the effect of pretransplant dialysis modality on the outcomes after kidney transplantation (KT). To minimize the confounding effect of donor-related variables, we performed a donor-matched retrospective comparison of 160 patients that received only one modality of pre-transplant dialysis (peritoneal dialysis [PD] and hemodialysis [HD] in 80 patients each) and that subsequently underwent KT at our center between January 1990 and December 2007. Cox regression models were used to evaluate the association between pre-transplant dialysis modality and primary study outcomes (death-censored graft survival and patient survival). To control for imbalances in recipient-related baseline characteristics, we performed additional adjustments for the propensity score (PS) for receiving pre-transplant PD (versus HD). There were no significant differences according to pre-transplant dialysis modality in death-censored graft survival (PS-adjusted hazard ratio [aHR]: 0.65; 95% confidence interval [95% CI]: 0.25-1.68) or patient survival (aHR: 0.58; 95% CI: 0.13-2.68). There were no differences in 10-year graft function or in the incidence of post-transplant complications either, except for a higher risk of lymphocele in patients undergoing PD (odds ratio: 4.31; 95% CI: 1.15-16.21). In conclusion, pre-transplant dialysis modality in KT recipients does not impact short- or long-term graft outcomes or patient survival.

## Introduction

A key factor in the choice between peritoneal dialysis (PD) and hemodialysis (HD) in patients with end-stage renal disease (ESRD) is the potential impact exerted by dialysis modality on the success of an eventual kidney transplant (KT) procedure. Unfortunately, conflicting results have been reported on the effect of pre-transplant dialysis modality on graft and patient survival. Several studies have reported an association

between the use of PD and poorer outcomes due to an increased incidence of graft thrombosis (1-7), new-onset diabetes after transplantation (NODAT) (8), infection (9) or acute graft rejection (10-12), leading to a lower short-term graft survival (5,10). In contrast, other studies have found better outcomes in patients receiving PD, including lower rates of delayed graft function (DGF) (5,12-19) or shorter time on dialysis (13,15,20,21). Finally, some authors have reported no significant differences between both dialysis modalities in terms of the risk of DGF (22-24), acute tubular necrosis (22,24-26), graft rejection (22-25), infection (22), thrombosis (22), NODAT (27), or graft and patient survival (18,22-26,28-31).

These inconsistent results may be due to factors that are unrelated to dialysis modality, such as donor characteristics or differences in methodological design (i.e., registry-based or single-center studies). In all of them the patients compared according to the dialysis modality had received grafts from different donors, thus hampering an accurate adjustment for imbalances in donor-related characteristics. In addition, some authors included patients that had received both PD and HD in a sequential manner. To date, no study has compared outcomes in pairs of patients with ESRD treated with different, single pre-transplant dialysis modalities who subsequently received kidney grafts from the same donors.

We hypothesized that dialysis modality could influence the short- and long-term post-transplant outcomes. In order to precisely control for donor-related variables, the present study was aimed at analyzing the impact of pre-transplant dialysis modality (PD or HD) by using a large cohort of donor-matched KT recipients.

#### Methods

Study population and design

This retrospective cohort study was performed at the University Hospital "12 de Octubre" (Madrid, Spain). Overall, 2,081 patients underwent KT at our institution between January 1990 and December 2007. The key inclusion criterion was that one of the two grafts from a given donor was transplanted into a patient who had received only PD during the pre-transplant period and that the second graft was transplanted into a patient who had received only HD. Patients who sequentially underwent both dialysis modalities were excluded, as well as those receiving a double KT, a double-organ transplantation or an organ from a living donor. On the basis of these criteria, and through a computer-aided search into our institutional transplant database, we selected 160 donor-matched patients: 80 received only pre-transplant PD (PD group) and 80 received only pre-transplant HD (HD group). The modality of pre-transplant dialysis was not formally accounted for in the manual allocation process. The functions of Transplant Coordinator were performed by the same person throughout the entire study period (A.A.). In addition, the same surgical team performed both types of procedures. A number of pre-transplant, peri-operative and post-transplant variables were retrospectively

recorded for each patient by using a dedicated data collection form. The need for specific informed consent was waived by the Institutional Review Board due to the retrospective and non-interventional nature of the investigation.

Study outcomes and definitions

The *primary study outcomes* were death-censored graft survival and patient survival. Graft loss was defined as return to dialysis or retransplantation. *Secondary outcomes* included long-term graft function (as assessed by estimated glomerular filtrate rate [eGFR] through different post-transplant points), DGF (defined as the need for dialysis within the first week after transplantation) and primary graft non-function (defined as permanent absence of graft function starting immediately post-transplant). Other post-transplant complications were also analyzed: biopsy-proven acute graft rejection, infection (only episodes requiring hospitalization and intravenous therapy were considered), surgical reintervention, vascular complications (including arterial or venous graft thrombosis and renal artery stenosis), urinary complications (including symptomatic urinary leakage and ureteral stricture), NODAT, cardiovascular events (including coronary artery disease, stroke and other forms of cerebrovascular disease, and peripheral arterial disease leading to critical ischemia of the lower limbs), lymphocele (defined as the presence of a peri-graft fluid collection with a diameter >5 cm diagnosed beyond the first week after transplantation), and *de novo* malignancy. Dyslipidemia was defined as low-density lipoprotein cholesterol >100 mg/dL, high-density lipoprotein cholesterol <40 mg/dL in men or <45 mg/dL in women, and/or triglycerides >150 mg/dL (32). The eGFR was estimated through the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation (33).

Immunosuppression and prophylaxis regimens and dialysis catheter management

All recipients of organs from donors after circulatory death underwent induction therapy with intravenous (IV) rabbit antithymocyte globulin (ATG) (ATG-Fresenius, 1.25 mg/Kg daily for 5-7 days), with the delayed introduction of the calcineurin inhibitor (CNI) from day 6 post-transplant due to the high incidence of delayed graft function among these patients. Recipients at high immunological risk —peak panel-reactive antibody (PRA) level >50%, second KT in case the first graft was lost due to rejection within the previous two years, or those receiving a third or fourth kidney graft— also received ATG induction at the same dose for 1-3 days with the early initiation of the CNI from day 0. Induction therapy with basiliximab (20 mg on days 0 and 4) was used in patients at high risk of CNI-related nephrotoxicity due to advanced age or certain pre-transplant comorbidities (i.e., diabetes), with the delayed introduction of the CNI from day 5 post-transplant. If a patient also fulfilled the criteria for high immunological risk, induction therapy was based on ATG for 3 days.

Maintenance immunosuppression consisted of cyclosporine A (2.5 mg/Kg daily, adjusted to a target trough level of 200-300 ng/mL during the first month and 100-150 ng/mL thereafter) or tacrolimus (0.1 mg/Kg daily, adjusted to a target trough level of 10-15 ng/mL for the first month and 5-10 ng/mL thereafter); mycophenolate mofetil (MMF) (1,000 mg twice daily) or mycophenolic acid (MPA) (720 mg twice daily); and prednisone (1 mg/Kg daily with progressive tapering). MMF/MPA was replaced with azathioprine (1.5 mg/Kg/day) in patients with impaired wound healing or early occurrence of urinary leakage. Conversion from CNI- to mammalian target of rapamycin (mTOR) inhibitor-based immunosuppression was reserved to patients developing with post-transplant *de novo* malignancy, severe cytomegalovirus (CMV) disease, or unacceptable CNI-related toxicity.

All patients received a single dose of IV cefazolin as peri-operative prophylaxis. Prophylaxis for *Pneumocystis jirovecii* pneumonia with trimethoprim-sulfamethoxazole (160/800 mg three times weekly) was given for 9 months. In patients at high-risk for CMV disease (donor/recipient mismatch or induction therapy with ATG), either IV ganciclovir (5 mg/Kg daily) or oral valganciclovir (900 mg daily) were administered for 3 months.

The catheter used for pre-transplant PD was removed at the end of the transplant procedure, in an attempt to decrease the risk of catheter-related peritonitis. If required due to the development of DGF, these patients received HD through a newly inserted central venous catheter. In those patients in which no arteriovenous fistula could be created or the fistula was non-functioning, the catheter used for pre-transplant HD was usually removed during the first weeks post-transplantation, once the graft function had stabilized.

# Statistical analysis

Quantitative data were shown as the mean  $\pm$  standard deviation (SD) or the median with interquartile ranges (IQR). Qualitative variables were expressed as absolute and relative frequencies. Categorical variables were compared using the  $\chi^2$  test, whereas Student's T test or U Mann-Whitney test were applied for continuous variables, as appropriate. Paired Student's T test was used for comparison between paired grafts obtained from the same donor. Correlations were analyzed by Pearson's correlation coefficient. Survival curves were plotted by the Kaplan-Meier method and differences between groups were compared with the log-rank test. Multivariate Cox regression models with backward stepwise variable selection were used to evaluate the association between the pre-transplant dialysis modality and study outcomes. Some clinically relevant factors (i.e., recipient and donor age, peak PRA or pre-transplant dialysis vintage) were also forced into the multivariate models irrespective of their univariate statistical significance. Cox regression models were stratified by matched pairs. Results were expressed as hazard ratios (HRs) with 95% confidence intervals

(Cls).

To partially overcome the limitation posed by the non-randomized design of our study, we calculated the propensity score (PS) for receiving PD as pre-transplant dialysis modality (versus HD) according to a given patient's features. The PS-based methodology takes into account the predicted probability of treatment assignment conditional on observed baseline characteristics, aiming at analyzing an observational study in a way that mimics to some extent the nature of a randomized controlled trial (34). Conditional on the PS derived from the fitted regression model, the distribution of baseline covariates should be similar between patients treated with PD or HD. The PS was based on the covariates selected by a conditional logistic regression analysis that included those baseline (pre-transplant) variables that differed between PD and HD groups with univariate P-values <0.1. The fit of the resulting model was assessed by means of the Hosmer-Lemeshow test and the area under the receiving operator characteristics curve (auROC). The estimated PS was then entered in the different multivariate models in which the remaining explanatory covariates were retained in order to adjust for potential confounding due to factors associated with the choice of the type of dialysis (34). The impact of the pre-transplant dialysis modality on the risk of specific post-transplant complications (secondary outcomes) was assessed through logistic regression, with results expressed as odds ratios (ORs) with 95% CIs. All the significance tests were two-tailed. Statistical analysis was performed with SPSS v. 15.0 (Statistical Package for Social Sciences Inc., Chicago, IL), and graphics were generated with Prism v. 6.0 (GraphPad Software Inc., La Jolla, CA).

#### Results

Baseline characteristics and post-transplant immunosuppression

The demographics and clinical characteristics of the 80 donors providing the 80 graft pairs are depicted in **Table 1**. Most of them were young males with brain death following head trauma that required vasoactive drug support. On the other hand, **Table 2** shows the baseline characteristics of the 160 recipients analyzed. Most variables were well balanced between both groups. However, as compared to those who received HD, patients in the PD group had shorter dialysis vintage, higher prevalence of pre-transplant dyslipidemia, lower number of pre-transplant blood product transfusions, higher residual urine output, lower immunological risk—as reflected by both peak and current PRA levels— and lower baseline values of hemoglobin and serum albumin. After entering these eight variables into the conditional logistic regression model, the following covariates were selected to construct the PS: pre-transplant dyslipidemia, residual urine output, and baseline serum albumin (**Table S1**). The resulting PS showed a goodness-of-fit to the data (Hosmer-Lemeshow test *P*-value = 0.377; auROC: 0.871; 95% CI: 0.812-0.930).

There were no significant differences between both groups regarding peri-transplant variables, type of induction therapy, or primary or maintenance immunosuppression (**Table 3**). The cold ischemia times were roughly equal between the paired grafts from the same donor  $(1,222.0 \pm 269.4 \text{ versus } 1,235.5 \pm 261.5 \text{ minutes for kidney #1 and kidney #2, respectively; } P-value = 0.499) and the correlation between both variables was high (Pearson's <math>r = 0.635$ ; P-value = 0.000). There were no significant differences in the duration of immediate post-transplant oliguria (i.e. total daily urine output <500 mL) between both grafts either (mean duration: 3.6 versus 3.2 days for kidney #1 and kidney #2; P-value = 0.111).

#### Post-transplant outcomes

The median follow-up for the overall cohort was 73.0 months (IQR: 36-99.8). Death-censored graft loss occurred in 13 (16.2%) and 15 (18.8%) patients in the PD and HD groups, respectively. There were no differences in death-censored graft survival between both groups, with 1-, 5- and 10-year rates of 94%, 84% and 78% in patients receiving PD compared to 92%, 82% and 77% in those receiving HD (log-rank test *P*-value = 0.722) (**Figure 1**). We found no significant differences in patient survival either (1-, 5- and 10-year survival rates of 97%, 87% and 74% in the PD group compared to 97%, 92% and 67% in the HD group; log-rank test *P*-value = 0.954) (**Figure 2**). This lack of association persisted after adjusting by Cox regression for clinical covariates (patient and donor age, pre-transplant comorbidities, type of underlying ESRD, peak PRA, pre-transplant dialysis vintage, occurrence of DGF or acute graft rejection), as well as after adding the PS for receiving pre-transplant PD into these models (**Table 4**).

There were no significant differences in the rate of primary graft non-function or DGF between patients receiving PD or HD, as detailed in **Table 5**. Long-term graft function, as measured by eGFR at different points throughout the first 10 post-transplant years, was also similar in both groups (**Figure 3**).

# Other post-transplant complications

Finally, we found no differences in the occurrence of early (i.e., during the post-transplant hospitalization period) or long-term post-transplant complications according to the pre-transplant dialysis modality (**Table 5**), except for the higher incidence of lymphocele in the PD group compared to the HD group (17.3% [13/80] versus 3.9% [3/80], respectively; *P*-value = 0.007). The independent impact of pre-transplant PD on the development of lymphocele persisted in a logistic regression model adjusted for recipient age, dialysis vintage, serum albumin level at transplantation and induction with ATG (OR: 4.31; 95% CI: 1.15-16.21; *P*-value = 0.031).

#### Discussion

In order to assess whether pre-transplant dialysis modality plays a role on the expected benefits derived from KT, we have analyzed the short- and long-term outcomes in two groups of recipients undergoing only PD or HD who received paired grafts from the same deceased donors. In addition, in view of the differences in some recipient-related baseline variables between both groups, we performed a PS-based multivariate analysis that provided further adjustment for confounding bias. By using these approaches we found no significant differences in either the primary —death-censored graft survival and all-cause mortality— or secondary outcomes —which included the 10-year evolution of the graft function. The effect of pre-transplant PD only became evident for the risk of developing lymphocele but not for other post-transplant complications.

Previous studies analyzing the influence of pre-transplant dialysis modalities on short- and long-term kidney graft outcome have yielded controversial results (summarized in **Table 6**). This may be due to differences in study design, including dissimilar donor-related characteristics or unequal numbers of patients analyzed within each group (18,23,26,30,35). In addition, several studies included patients who had received both PD and HD in a sequential manner (3,5,19,21) that were further categorized according to the modality used for the longest period (21) or at the time of transplantation (5). Repeated thrombosis of the vascular access is one of the most common reasons for changing from HD to PD, a circumstance that could partially account for the higher incidence of graft thrombosis associated to pre-transplant PD in some studies (3). Interestingly, by including patients who had received only one pre-transplant dialysis modality, we found no differences in the occurrence of graft thrombosis between both groups.

The study by Kramer et al (30), which included more than 29000 patients from 16 European registries, reported opposing results depending on the statistical analysis performed. In an attempt to decrease the risk of confounding by indication the authors applied an instrumental variable method, which is used to approximate randomization in observational studies. Although standard Cox regression model showed a potential benefit from pre-transplant PD in terms of graft and patient survival, such association was not confirmed in the instrumental analysis (30). The findings of the present study, although based on an alternative methodological approach (PS-adjusted analysis), confirm those reported by Kramer et al.

In previous studies the grafts transplanted into patients receiving pre-transplant PD or HD had been obtained from different donors, which may also influence the inconsistent findings in the literature. In line to other authors (36-42), we designed our study to minimize the effect of donor-related characteristics by means of a donor-matched approach that ensured that graft pairs were extracted and preserved under identical

conditions and transplanted by the same surgical team with comparable surgical procedures and postoperative care management. In addition, the single-center nature of our study obviates any potential intercenter confounding. Thus, we were able to adjust for the quality of the transplanted graft and to focus exclusively on graft- and patient-specific functional outcomes.

In our experience, patients receiving pre-transplant PD spent shorter time on dialysis than those who underwent HD. Such finding has been also reported in previous studies (5,13) and is not totally unexpected, since in many centers PD constitutes the modality preferentially offered to the best transplant candidates whereas HD is usually reserved to those patients who are deemed less suitable for KT (5). Accordingly, there were no significant differences in other baseline characteristics—such as the number of pre-transplant comorbidities—that could account for the between-group imbalance observed in the dialysis vintage.

Hypervolemia, better blood pressure control, shorter cold ischemia time and larger residual urine output have been proposed as protective factors associated with a shorter time to the recovery of graft function in patients previously receiving PD as compared to HD (12,13,16). We have found no significant differences in cold ischemia time or baseline mean blood pressure between both groups whereas, as expected, the residual urine output was higher among patients treated with PD. Although this variable has been shown to be beneficial in other studies (12,13,16), in our experience it was not associated to a better graft outcome in the PD group, as evidenced by the similar rates of primary graft non-function and DGF and the comparable numbers of days of oliguria and of post-transplant dialysis sessions. Not surprisingly, the number of blood product transfusions was higher among patients undergoing HD, since blood losses are more common with this modality than with PD. In accordance to this, the immunological risk was higher in the HD group, a finding that may be also explained by the numerically higher rate of second transplants in these patients. Nevertheless, the incidence of acute graft rejection was similar irrespective of the pre-transplant dialysis modality, likely due to the more frequent use of ATG as induction therapy in the HD group.

We found no apparent impact of the modality of dialysis on the incidence of early (i.e., during the post-transplant hospitalization period) or long-term complications either. The most common difference reported in previous studies was a higher risk of graft thrombosis in patients receiving PD (1-6), an association that has been explained by the presence of hypercoagulability states (43,44) and obesity (45) in this group. Our study did not confirm this finding and, in addition, we found no significant differences in baseline body mass index (BMI) between patients receiving PD or HD. Therefore, the association observed by other authors may have been due to certain donor characteristics or to the presence of undiagnosed prothrombotic conditions in the recipient, which would have in turn led to a change from pre-transplant HD to PD following vascular access

thrombosis. Although it has been reported a higher incidence of post-transplant infection in KT recipients receiving pre-transplant PD (9), in our experience there were no differences in the occurrence of such event. Lymphocele constitutes the only post-transplant complication whose incidence was found to be significantly higher in patients who received PD. Lymphocele —a collection of lymph fluid in a cavity that is not covered by an epithelial membrane—occurs in 0.6% to 49% of KT recipients, mainly due to the surgical injury to the iliac or renal hilar lymphatic vessels. Differences in diagnostic criteria underlie the broad variability in the reporting frequency of this complication across different series (21,46-48). A higher incidence of lymphocele and peri-graft fluid collection has been correlated by some authors with the presence of acute graft rejection and obesity (46) and with the type of immunosuppression used, mainly those including mTOR inhibitors (46-48). Two studies have evaluated the occurrence of lymphocele after KT according to the pre-transplant dialysis modality, with conflicting results (22,49). Caliskan et al. did not find significant differences between patients receiving PD or HD; however, it was not clear whether the definition of lymphocele included only the presence of lymphatic fluid or also other forms of peri-graft fluid collection (22). On the other hand, Kitada et al. reported a higher occurrence of ascites among living-donor KT recipients who had received PD as compared to those who underwent HD (49). Of note, the association found in our study between pretransplant PD and lymphocele was independent of the baseline BMI, the use of mTOR inhibitors or the occurrence of acute graft rejection. It could be hypothesized the role of changes in the lymphatic circulation due to pre-transplant peritoneal stress —such as that associated to PD— or post-transplant alterations in mesothelial cells due to the intra-operative administration of large volumes of fluids (49).

Some limitations to the present study should be noted, including its retrospective nature and relatively small sample size, which might compromise the stability of the multivariate analyses. It is conventionally assumed that Cox models should be used with a minimum of 10 events per predictor variable, as the results are subject to increasing bias and variability, unreliable confidence interval coverage and problems with model convergence when this ratio declines below such threshold (50). Moreover, it should not be ruled out that the observed absence of differences in outcome might reflect a type 2 statistical error, particularly if attempts to extrapolate our results to specific subpopulations of patients (i.e., those with pre-transplant diabetes) are made. Although the analysis of paired kidneys constitutes a well-established methodological approach in the literature to reduce the confounding effect of donor-related characteristics (36-42), we cannot guarantee that the functions of both grafts harvested from the same donor were strictly comparable. In an attempt to control for certain imbalances found in the baseline characteristics between patients undergoing different pre-transplant dialysis modalities (i.e., dialysis vintage or immunological risk), we performed an additional

adjustment through a PS-based analysis. However, propensity analysis can only adjust for known measured variables, so we are unable to exclude the potential effect of other confounders. In that sense, there was a non-significant trend towards an increased presence of dyslipidemia at baseline —a cardiovascular risk factor that was found to be associated to mortality— within the PD group. However, about half of the patients in both groups were given statins, suggesting the implementation of an intensive lipid-lowering strategy during the post-transplant period that was even more evident among patients undergoing HD, likely due to their higher prevalence of established cardiovascular disease. Testing for donor-specific antibodies (DSA) was not systematically performed during the study period, thus preventing us from assessing the impact of this variable on graft outcome. Finally, the generalizability of our results to the contemporary transplant practices may be limited due to the long time period spanned by the study (i.e., our patients had mean age and BMI values at transplantation lower than those usually reported in more recent series).

In conclusion, our study of donor-matched KT recipients that underwent only one modality of pre-transplant dialysis did not find significant differences in either short- or long-term graft or patient outcomes, including death-censored graft survival or all-cause mortality. These findings suggest that both PD and HD are equally valid options for patients with ESRD on the waiting list for KT, as it is likely that donor- and graft-related variables may well play a larger role than the pre-transplant dialysis modality in determining post-transplant outcomes. Future multicenter studies with a similar donor-matched and PS-adjusted design that includes post-transplant DSA monitoring should be carried out to corroborate these results in a larger and more contemporary cohort of patients.

### **Supporting Information**

Additional Supporting Information may be found in the online version of this article:

• **Table S1:** Selection of the baseline (pre-transplant) covariates included in the PS for receiving pre-transplant PD (conditional logistic regression).

# **Author contributions**

T.D., M.F.R., M.P. and A.A. designed research; T.D., M.P., N.P., E.G., E.G.S., E.G. and A.A. performed research; T.D., M.F.R., M.P. and A.A. analyzed data; T.D. and M.F.R. wrote the paper; and M.P. and A.A. revised the final draft of the manuscript.

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#### Tables.

**Table 1.** Demographics and clinical characteristics of the 80 deceased donors that provided the 80 graft pairs.

Variable	
Age, years [mean ± SD]	39.2 ± 18.1
Gender (male) [n (%)]	59 (73.8)
Type of donor [n (%)]	
Donation after brain death	74 (92.5)
Donation after circulatory death	6 (7.5)
Cause of death [n (%)]	
Trauma	43 (53.8)
Hemorrhagic stroke	22 (27.5)
Ischemic stroke	2 (2.5)
Cardiac arrest	5 (6.3)
Others	8 (10.0)
Serum creatinine, mg/dL [mean ± SD]	$0.9 \pm 0.3$
Requirement for vasoactive drugs [n (%)] <sup>a</sup>	66 (86.8)
Pre-transplant kidney biopsy [n (%)]	13 (16.3)

Table 2. Baseline and clinical characteristics according to pre-transplant dialysis modality.

Variable	<b>PD group</b> (n = 80)	<b>HD group</b> (n = 80)	<i>P-</i> value
Age of recipient, years [mean ± SD]	43.2 ± 13.9	46.6 ± 15.1	0.150
Gender (male) [n (%)]	54 (67.5)	53 (66.2)	0.867
BMI, $Kg/m^2$ [mean $\pm$ SD] <sup>a</sup>	$25.3 \pm 5.4$	$24.8 \pm 5.6$	0.737
Mean blood pressure, mmHg [mean ± SD]	102.1 ± 14.1	102.5 ± 12.4	0.870
Pre-transplant comorbidities [n (%)]			
Hypertension	60 (75.0)	51 (63.7)	0.123
Diabetes mellitus	12 (15.0)	13 (16.2)	0.828
Dyslipidemia	12 (15.0)	5 (6.2)	0.073
Smoking history	12 (15.0)	7 (8.8)	0.222
Cardiovascular disease	5 (6.2)	11 (13.8)	0.114
Stroke	3 (3.8)	3 (3.8)	1.000
Peripheral arterial disease	2 (2.5)	4 (5.0)	0.681
Malignancy	4 (5.0)	2 (2.5)	0.681
Positive anti-HCV serostatus [n (%)] <sup>b</sup>	4 (5.2)	6 (7.7)	0.746
Previous KT [n (%)]	5 (6.2)	9 (11.2)	0.263
Etiology of underlying ESRD [n (%)]			
Glomerulonephritis	17 (21.2)	15 (18.8)	0.693
Diabetic nephropathy	10 (12.5)	6 (7.5)	0.292
Nephroangiosclerosis	15 (18.8)	16 (20.0)	0.841
Policystosis	10 (12.5)	8 (10.0)	0.617
Reflux nephropathy	7 (8.8)	9 (11.4)	0.580
Unknown	5 (6.2)	9 (11.2)	0.263
Other	16 (20.0)	17 (21.2)	0.845
Vascular dialysis access [n (%)]			
Arteriovenous fistula	NA	72 (91.1)	NA
Central venous catheter	NA	7 (8.9)	NA
Dialysis vintage, months [median (IQR)]	15.0 (8.3-28.0)	25.5 (11.3-52.5)	0.002
Number of pre-transplant transfusions [median (IQR)]	0.0 (0.0-1.5)	0.0 (0.0-4.0)	0.011
Peak PRA >10% [n (%)] <sup>c</sup>	8 (10.7)	21 (28.0)	0.007
Current PRA >10% [n (%)]°	1 (1.3)	7 (9.3)	0.063
Residual urine output in 24 hours, cc [median (IQR)] <sup>d</sup>	800.0 (200.0- 1,500.0)	250.0 (0.8-675.0)	0.000
Number of HLA mismatches [median (IQR)]	3.0 (3.0-4.0)	3.0 (3.0-4.0)	0.868
Pre-transplant laboratory values [mean ± SD]			
Hemoglobin, g/dL	12.6 ± 1.7	$13.4 \pm 1.6$	0.006

<sup>&</sup>lt;sup>a</sup> Data on the use of vasoactive drugs were not available for 4 donors.

Serum albumin, g/dL	$3.9 \pm 0.5$	$4.4 \pm 0.5$	0.000
Serum creatinine, mg/dL	$8.6 \pm 2.9$	$8.2 \pm 2.9$	0.472

BMI: body mass index; ESRD: end-stage renal disease; HCV: hepatitis C virus; HD: hemodialysis; HLA: human leukocyte antigen; IQR: interquartile range; KT: kidney transplantation; NA: not applicable; PD: peritoneal dialysis; PRA: panel reactive antibody; SD: standard deviation.

**Table 3.** Peri-transplant variables and immunosuppressive regimens.

Variable	<b>PD group</b> (n = 80)	<b>HD group</b> (n = 80)	<i>P-</i> value
Cold ischemia time, minutes [mean ± SD]	1,214.2 ± 283.8	1,243.2 ± 246.2	0.492
Intra-operative use of vasoactive drugs [n (%)]	1 (1.3)	2 (2.5)	1.000
Duration of oliguria, days [median (IQR)]	0.0 (0.0-2.0)	0.0 (0.0-6.0)	0.115
Induction therapy [n (%)]			
ATG	7 (8.8)	14 (17.5)	0.101
Basiliximab	15 (18.8)	9 (11.2)	0.184
None	58 (72.5)	57 (71.2)	0.660
Primary immunosuppression [n (%)]			
Steroids	80 (100.0)	80 (100.0)	1.000
Calcineurin inhibitor			0.871
Tacrolimus	50 (62.5)	49 (61.3)	-
Cyclosporine	30 (37.5)	31 (38.8)	-
Anti-proliferative drug			0.685
MMF / MPA	64 (80.0)	66 (82.5)	-
Azathioprine	16 (20.0)	14 (17.5)	-
Maintenance immunosuppression [n (%)] <sup>a</sup>			
Steroids	57 (76.0)	59 (76.6)	0.928
Tacrolimus	51 (68.0)	49 (63.6)	0.571
Cyclosporine	14 (18.7)	17 (22.1)	0.602
MMF / MPA	47 (62.7)	56 (72.7)	0.185
Azathioprine	8 (10.7)	9 (11.7)	0.842
mTOR inhibitor	12 (16.0)	13 (16.9)	0.883
Concomitant medications [n (%)] <sup>a</sup>			
Statin	33 (44.6)	40 (51.9)	0.366
ACE-inhibitor / ARB	26 (35.1)	33 (42.9)	0.331
Other antihypertensive drugs <sup>b</sup>	14 (17.5)	11 (13.8)	0.664

<sup>&</sup>lt;sup>a</sup> Data on BMI values were not available for 77 patients.

<sup>&</sup>lt;sup>b</sup> Data on the HCV serostatus were not available for 5 patients.

<sup>&</sup>lt;sup>c</sup> Data on the PRA values were not available for 10 patients.

<sup>&</sup>lt;sup>d</sup> Data on the residual urine output were not available for 3 patients.

ACE-inhibitor / ARB: angiotensin-converting-enzyme inhibitor / angiotensin II receptor blocker; ATG: antithymocyte globulin; HD: hemodialysis; IQR: interquartile range; MMF / MPA: mycophenolate mofetil / mycophenolic acid; mTOR: mammalian target of rapamycin; PD: peritoneal dialysis; SD: standard deviation.

Table 4. Risk factors for the occurrence of primary study outcomes (follow-up truncated at 10 years).

	Univariate analysis		Multivariate analysis <sup>b</sup>			PS-adjusted model <sup>b,c</sup>			
	HR	95% CI	<i>P-</i> value	HR	95% CI	<i>P</i> -value	HR	95% CI	<i>P</i> -value
Death-censored graft loss (n = 28)									
PD as pre-transplant dialysis modality (versus HD)	0.87	0.40 - 1.88	0.870	0.60	0.26 - 1.47	0.263	0.65	0.25 - 1.68	0.378
Dialysis vintage, months <sup>a</sup>	0.99	0.98 - 1.01	0.769	-	-	-	-	-	-
Recipient age, years <sup>a</sup>	1.01	0.98 - 1.04	0.399	-	-	-	-	-	-
Pre-transplant cardiovascular disease	2.86	1.14 - 7.14	0.025	2.99	1.17 - 7.60	0.022	3.01	1.16 - 7.81	0.024
Peak PRA, % <sup>a</sup>	1.00	0.99 - 1.02	0.582	-	-	-	-	-	-
Donor age, years <sup>a</sup>	1.01	0.99 - 1.03	0.265	-	-	-	-	-	-
Biopsy-proven acute rejection	4.56	1.97 - 10.55	0.000	4.74	1.99 - 11.30	0.000	5.29	2.12 - 13.27	0.000
All-cause mortality (n = 22)									
PD as pre-transplant dialysis modality (versus HD)	1.02	0.43 - 2.46	0.959	0.72	0.23 - 2.26	0.572	0.58	0.13 - 2.68	0.486
Dialysis vintage, months <sup>a</sup>	1.01	0.99 - 1.01	0.266	-	-	-	-	-	-
Recipient age, years <sup>a</sup>	1.06	1.02 - 1.09	0.002	-	-	-	-	-	-
Pre-transplant dyslipidemia	3.45	1.13 - 10.58	0.030	3.58	1.12 - 11.44	0.031	-	-	-
Nephroangiosclerosis as underlying ESRD	5.55	2.29 - 13.43	0.000	3.39	1.19 - 9.64	0.022	4.29	1.36 - 13.83	0.014
Donor age, years <sup>a</sup>	1.05	1.03 - 1.08	0.000	1.05	1.02 - 1.08	0.004	1.04	1.00 - 1.07	0.039
Delayed graft function	4.20	1.66 - 10.85	0.003	3.52	1.34 - 9.23	0.011	2.79	0.99 - 7.84	0.051

CI: confidence interval; ESRD: end-stage renal disease; HD: hemodialysis; HR: hazard ratio; PD: peritoneal dialysis; PRA: panel reactive antibody; PS: propensity score.

<sup>&</sup>lt;sup>a</sup> Percentages calculated after excluding 8 patients with primary graft non-function.

<sup>&</sup>lt;sup>b</sup> Includes calcium-channel blockers and alpha- and beta-blockers.

<sup>&</sup>lt;sup>a</sup> Per unitary increment.

<sup>&</sup>lt;sup>b</sup> Cox models were stratified by matched pairs.

<sup>&</sup>lt;sup>c</sup> The propensity score accounted for the probability of receiving PD as pre-transplant dialysis modality according to a given patient's baseline characteristics. The remaining explanatory covariates were also retained in the Cox regression models.

**Table 5.** Ten-year cumulative incidence of post-transplant complications according to pre-transplant dialysis modality.

Variable	<b>PD group</b> (n = 80)	<b>HD group</b> (n = 80)	<i>P-</i> value <sup>a</sup>
Primary graft non-function [n (%)]	5 (6.2)	3 (3.8)	0.719
Delayed graft function [n (%)]	24 (32.4)	30 (39.5)	0.369
Number of dialysis sessions [mean (IQR)]	0.0 (0.0-1.0)	0.0 (0.0-2.0)	0.248
Biopsy-proven acute graft rejection [n (%)]	10 (12.5)	9 (11.2)	0.807
T-cell-mediated	5 (6.2)	3 (3.8)	0.719
Humoral	3 (3.8)	4 (5.0)	1.000
Borderline	2 (2.5)	2 (2.5)	1.000
Early complications [n (%)] <sup>b</sup>			
Infection	16 (20.0)	12 (15.0)	0.405
Surgical reintervention	10 (12.5)	10 (12.5)	1.000
Vascular complication	6 (7.5)	8 (10.0)	0.576
Arterial or venous graft thrombosis	4 (5.0)	5 (5.0)	1.000
Urinary complication	5 (6.2)	2 (2.5)	0.443
Long-term complications [n (%)] <sup>c</sup>			
Infection	33 (44.0)	30 (40.0)	0.620
NODAT	14 (18.7)	23 (29.9)	0.108
Cardiovascular event	21 (28.0)	19 (25.3)	0.712
Urinary complication	15 (20.0)	10 (13.0)	0.244
Lymphocele	13 (17.3)	3 (3.9)	0.007
De novo malignancy	8 (10.7)	12 (16.0)	0.337
Death-censored graft loss	13 (16.2)	15 (18.8)	0.677
All-cause mortality	11 (13.8)	11 (13.8)	1.000

HD: hemodialysis; IQR: interquartile range; NODAT: new-onset diabetes mellitus after transplantation; PD: peritoneal dialysis.

 $<sup>^{\</sup>rm a}_{_{\rm c}}\chi^2$  test and U Mann-Whitney test were used for comparing categorical and continuous variables.

<sup>&</sup>lt;sup>b</sup> During the post-transplant hospitalization period.

<sup>&</sup>lt;sup>c</sup> Percentages calculated after excluding 8 patients with primary graft non-function.

**Table 6.** Review of previous studies evaluating outcomes in KT recipients according to pre-transplant dialysis modality.

Author [reference]	Number of patients	Median follow- up	Post-transplant outcomes
Bleyer et al (13)	N = 9291	1 year	Shorter time on dialysis and lower incidence of DGF in the PD group
Butani et al (29)	N = 3606 (pediatric patients) [PD: 1226, HD: 1370, predialysis: 1010]	NA	No differences in graft survival among deceased donors, lower graft survival in the HD group among living donors
Caliskan et al (22)	N = 88 [PD: 44, HD: 44]	6.5 years	No differences in the incidence of DGF, lymphocele, or short- and long-term complications, no differences in graft survival
Chalem et a (20)	N = 6240 [PD: 647, HD: 5190, predialysis: 583]	2 years	Shorter time on the waiting list, shorter ischemia time
Courivaud et al (27)	N = 2010 [PD: 332, HD: 1564, predialysis: 114]	6 months	No differences in the incidence of NODAT
Freitas et al (17)	N = 306 [PD: 38, HD: 268]	3 years	Lower incidence of primary graft failure and DGF in the PD group, no differences in the incidence of acute rejection and survival
Goldfarb- Rumyantzev et al (21)	N = 92844	11 years	Shorter time on dialysis, better graft and patient survival in the PD group
Guillou et al (10)	N = 121	1 years	Higher incidence of acute rejection i the PD group
Helal et al (25)	N = 78 [PD: 39, HD: 39]	10 years	No differences in the incidences of acute rejection and ATN, and survival
Joseph et al (15)	N = 325 [PD: 183, HD: 117, both: 25]	5 years	Shorter time on the waiting list, lower incidence of DGF
Kitada et al (49)	N = 58 (living donors) [PD: 14, HD: 36, predialysis: 8]	6 months	Higher incidence of ascites in the PI group
Kramer et al (30)	N = 29088 [PD: 10135, HD: 18953]	5 years	No differences in graft and patient survival in the instrumental variable analysis
Lim et al (19)	N = 6701 [PD: 1244, HD: 4241, both: 1216]	NA	Higher incidence of DGF in the HD group, lower graft and patient survival in patients consecutively receiving HD followed by PD
Madziarska et al (8)	N = 308 [PD: 48, HD: 260]	1 years	Higher incidence of NODAT in the PD group
Maiorca et al (28)	N = 72 [PD 34; HD 38]	5 years	Lower incidence of DGF in the PD group, similar graft survival
Malyszko et al (43)	N = 47 [PD: 23, HD: 24]	NA	Higher presence of

			hypercoagulability state in the PD group
McDonald et al (1)	N = 7247 (pediatric patients) [PD: 2924, HD: 2070, both: 439, predialysis: 1808]	NA	Higher incidence of graft thrombosis in the PD group
Molnar et al (31)	N = 14508 [PD: 2092, HD: 12416]	717 days	No differences in the incidence of DGF or patient survival in the multivariate analyses
Murphy et al (2)	N = 202	16 days	Higher incidence of graft thrombosis in the PD group
Ojo et al (3)	N = 2223 [PD: 502, HD: 1489, predialysis: 232]	30 days	Higher incidence of graft thrombosis in the PD group
Palomar et al (4)	N = 189 [PD: 44, HD: 115, predialysis: 30]	1 year	Higher incidence of graft thrombosis in the PD group
Passalacqua et al (9)	N = 156 [PD: 32, HD: 103, both: 21]	30 days	Higher incidence of infection and longer length of stay in the PD group
Perez Fontán et al (16)	N = 114 (39 patients received paired grafts from the same donors) [PD: 56, HD: 58]	3 years	Lower incidence of DGF and transfusion requirements in the PD group
Prasad et al (24)	N = 90 (living donor recipients) [PD: 45, HD: 45]	8 years	No differences in the incidence of DGF, acute rejection, incidence of surgical complications, or graft and patient survival
Ramos-Sánchez et al (26)	N = 1006 [PD: 37, HD: 969]	NA	No differences in the incidence of ATN, levels of proteinuria, or graft and patient survival
Resende et al (23)	N = 421 [PD: 47, HD: 374]	8 years	No differences in the incidence of DGF and acute rejection, graft and patient survival, and graft function
Satoh et al (11)	N = 38 [PD: 16, HD: 22]	30 months	Higher incidence of acute rejection in the PD group
Snyder et al (5)	N = 252402 [PD: 33162, HD: 219240]	5 years	Higher probability of receiving KT transplantation, lower incidence of DGF, and higher incidence of sort-term graft failure and graft thrombosis in the PD group
Tomura et al (44)	N = 34 [PD: 17, HD: 17]	NA	Higher presence of hypercoagulability state and higher incidence of cardiovascular events in the PD group
Van Biesen et al (14)	N = 119 [PD: 40, HD: 79]	NA	Lower incidence of DGF and acute renal failure in the PD group
van der Vliet et al (6)	N = 915 [PD: 303, HD: 612]	NA	Higher incidence of graft thrombosis in the PD group
Vanholder et al (12)	N = 234 [PD: 117, HD: 117]	6 months	Higher incidence of acute graft rejection and lower incidence of DGF in the PD group

ATN: acute tubular necrosis; DGF: delayed graft function; HD: hemodialysis; NA: not available; NODAT: new-onset diabetes mellitus after transplantation; PD: peritoneal dialysis.

# Figure legends

- **Figure 1.** Kaplan-Meier death-censored graft survival curves according to pre-transplant dialysis modality with follow-up truncated at 10 years (log-rank test *P*-value = 0.722) (PD: peritoneal dialysis; HD: hemodialysis).
- **Figure 2.** Kaplan-Meier patient survival curves according to pre-transplant dialysis modality with follow-up truncated at 10 years (log-rank test *P*-value = 0.954) (PD: peritoneal dialysis; HD: hemodialysis).
  - **Figure 3.** Long-term evolution of graft function according to pre-transplant dialysis modality. Dots and squares represent the mean value in each group, whereas error bars denote the standard deviation (eGFR: CKD-EPI equation-estimated glomerular filtration rate; PD: peritoneal dialysis; HD: hemodialysis).





