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Addition of a Temporary Portocaval Shunt Does Not Reduce Acute Kidney Injury in Caval-Sparing Liver Transplantation

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ABSTRACT

Acute kidney injury (AKI) is a common complication following liver transplantation (LT), with multifactorial etiology. It is believed that perioperative hemodynamic instability could lead to AKI. A temporary portocaval shunt (TPCS) could possibly prevent this, but its beneficial effect is still controversial, especially in caval-sparing LT. Therefore, the aim of this study was to evaluate whether the use of a TPCS during hepatectomy reduces the incidence and severity of post-LT AKI in caval-sparing LT, defined according to AKIN criteria. Between January 2005 and August 2023, all orthotopic LTs performed in a single center were retrospectively analyzed and were divided into a TPCS group (n = 134) and a no-TPCS group (n = 260). Serum creatinine was collected right before LT and daily during the first week post-LT. In multivariate analysis, TPCS was not related to AKI, while diabetes mellitus (p = 0.01) and LabMELD (p = 0.02) were. When comparing TPCS and no-TPCS groups, no differences were seen in median increase of serum creatinine post-LT (TPCS; 12 μmol/L (-4–52) versus no-TPCS; 14 μmol/L ± (-3–52) (p = 0.94)), number of post-LT AKI (TPCS; 31% versus no-TPCS; 33% (p = 0.57)), or severity of post-LT AKI (p = 0.90). In conclusion, the application of a TPCS during hepatectomy is not associated with less post-LT AKI or less severe post-LT AKI when using a caval-sparing LT technique.

1 | Introduction

Acute kidney injury (AKI) is a common complication following orthotopic liver transplantation (LT), with a reported incidence of 20%–78% [1–8]. About 15% of recipients require transient renal replacement therapy (RRT) immediately after LT [9, 10]. AKI has an unfavorable effect on the prognosis of LT recipients by impairing short-term survival, while it is also associated with reduced long-term survival due to an increased risk of developing chronic kidney disease (CKD) [1–3, 6, 7, 11]. The etiology of post-LT AKI is not completely understood, but it is most likely multifactorial, including donor and recipient factors (e.g., high model for end-stage liver disease [MELD] score), perioperative factors (e.g., hypotension and acute blood loss), as well as post-transplant factors (e.g., use of nephrotoxic agents, including a calcineurin inhibitor) [12].

Abbreviations: AKI, acute kidney injury; BAR, balance of risk; DBD, donation after brain death; DCD, donation after circulatory death; DRM, donor-recipient model; ET-DRI, Eurotransplant donor risk index; LabMELD, laboratory model for end-stage liver disease; LT, liver transplantation; LUMC, Leiden University Medical Center; MELD, model for end-stage liver disease; RRT, renal replacement therapy; sRRi, simplified recipient risk index; TPCS, temporary portocaval shunt.

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Reperfusion of the transplanted liver graft by unclamping of the portal vein is often followed by hemodynamic instability [13, 14]. This hemodynamic instability can trigger a cascade of effects, ultimately resulting in renal tubular injury, increasing the risk of AKI. The vena cava-sparing technique has been introduced as a surgical technique to preserve caval flow during the entire procedure, thereby maintaining venous return to the right chamber and reducing hemodynamic instability [15]. Additionally, a temporary portocaval shunt (TPCS) during hepatectomy has been introduced to decrease portal pressure, thereby preventing gut edema [16]. The beneficial effect of a TPCS to prevent post-LT AKI is still under debate. Several relatively small, retrospective studies have reported conflicting results in post-LT creatinine clearance [17–20], making the beneficial effect of a TPCS in LT still controversial.

Therefore, the primary aim of this study was to evaluate whether the use of a TPCS during hepatectomy reduces the incidence and severity of post-LT AKI when using a caval-sparing LT technique. The secondary aim of this study was to evaluate whether the use of a TPCS during hepatectomy improves 1-year patient survival or 1-year graft survival.

2 | Materials and Methods

2.1 | Patients

All consecutive LTs at the Leiden University Medical Center (LUMC), Leiden, the Netherlands, performed between January 2005 and August 2023 were included. Recipients who received dialysis before LT, combined liver-kidney transplantation, use of machine-perfused liver techniques, domino, split, auxiliary, or who received urgent LT for acute liver failure were excluded from analysis. Recipient baseline characteristics at the time of admission were collected. Clinical information was obtained from a prospectively collected database and completed with information from electronic patient files. Covariates included donor demographics, recipient demographics, pre-transplant information, intraoperative data, and postoperative outcome.

Calculated MELD scores were included in the recipient analysis. The Eurotransplant donor risk index (ET-DRI), simplified recipient risk index (sRRI), combined donor-recipient model (DRM), and balance of risk (BAR) score were calculated as described previously [21–23].

2.2 | Operative Techniques Recipient Surgery

Briefly, standard incision and exposure was followed by the dissection of the hepatoduodenal ligament and by liver mobilization. Since May 2010, a change in center protocol was incorporated, which consisted of the use of a TPCS prior to mobilization and removal of the native liver. After hilar dissection, a TPCS was created by an end-to-side anastomosis of the recipient's portal vein (splanchnic side) to the inferior vena cava at the level of the renal veins. After insertion of the liver graft, a side-to-side caval anastomosis was performed as described previously [16]. If a TPCS was used, it was divided just before portal reconstruction using a vascular endo-GIA stapling device (Medtronic, Minneapolis, MN, USA), after which a standard end-to-end portal anastomosis was performed. Reperfusion was induced after either initial portal or arterial anastomosis. Finally, biliary reconstruction was performed, preferably with a duct-to-duct anastomosis.

2.3 | AKI

Baseline serum creatinine levels were collected right before operation. Postoperative serum creatinine levels were collected daily in the first week after LT. AKI was defined according to AKIN criteria [24]: Stage 1, ≥1.5 times baseline serum creatinine level or an increase of 26.1 μmol/L above baseline Stage 2, >2 times baseline level; and Stage 3, >3 times baseline level or requirement of RRT, all within the first 7 days after LT. Postoperative AKI was divided into mild AKI (AKIN Stage 1) and severe AKI (AKIN Stages 2 and 3) as described previously [25]. Graft and patient survival, up to 1 year, were documented. All major postoperative complications during hospitalization, defined as grade ≥ 3a by the Clavien-Dindo classification [26], were documented.

2.4 | Immunosuppression Regime

Immunosuppression was provided per standard protocol and included tacrolimus initiated on Days 2 to 5 and, if required, mycophenolate mofetil. In the anhepatic phase and on postoperative Day 4, basiliximab was given. Methylprednisolone in the anhepatic phase, followed by prednisolone orally from Day 1, tapered out between 3 and 6 months after transplantation. Since 2009, sirolimus and everolimus, have been used more often in combination with tacrolimus. Tacrolimus trough levels were 5–15 ng/mL in the first 3 months and thereafter 4–8 ng/mL for monotherapy or—in the case of combination therapy—3–5 ng/mL.

2.5 | Statistical Analysis

Continuous variables were presented as mean and standard deviation (SD), or median and interquartile range (IQR), whereas categorical variables were presented as number and percentage. Univariate analysis between the groups was performed using Student's *t*-test, Chi-square test, one-way analysis of variance (ANOVA), or Mann–Whitney *U* test, where applicable. A multivariate binary logistic regression analysis (Wald) in the comparison between recipients who developed AKI after LT and recipients who did not was conducted. Graft and patient survival were estimated using the Kaplan–Meier method and compared using the log-rank test. A p value below 0.05 was considered significant. Statistical analyses were performed using SPSS software version 27.0 for Windows (SPSS Inc., Chicago, IL, USA). The local medical ethics committee approved the protocol and waived the need for consent from participants. The Declaration of Helsinki was adhered to.

3 | Results

In total, 580 recipients received an LT in the period from January 2005 through August 2023. Of these, 186 recipients were excluded due to either preoperative RRT (continuous veno-venous hemofiltration, dialysis) ($n = 34$), receiving a combined liver-kidney transplant ($n = 18$), receiving a split or auxiliary LT ($n = 25$), high-urgency listing for LT ($n = 74$), or due to the use of machine perfusion techniques (D-HOPE, NMP, NRP) ($n = 65$). Multiple exclusion criteria may apply to a single transplantation procedure. Of the 394 recipients included in this study, 134 recipients received a perioperative TPCS (34%), and 260 recipients (66%) did not receive a TPCS.

3.1 | Donor, Transplant, and Recipient Characteristics

Table 1 shows the baseline donor, transplant, and recipient characteristics of both groups. The warm ischemic period (WIP) was significantly longer in the TPCS group (35 min (30–41) vs. 32 (28–37); $p < 0.001$). Thirty-five percent of the recipients in the no-TPCS group received a donation after circulatory death LT (DCD-LT), versus 47% in the TPCS group ($p = 0.03$). The median number of perioperative distributed blood products in the no-TPCS group was significantly higher compared to the TPCS group (4 (1–8) vs. 3 (0–6); $p = 0.02$). The median number of perioperative distributed fresh frozen plasma (FFP) (no-TPCS; 6 units (2–10) vs. TPCS; 7 units (4–11); $p = 0.15$) and the median volume of cell saver return (no-TPCS; 1079 mL (660–1833) vs. TPCS; 1200 mL (784–2077); $p = 0.05$) did not significantly differ between both groups. We conducted a multivariate regression with the aim of adjusting for confounding variables and thereby avoiding potential bias in the comparison between recipients who did develop AKI after LT and recipients who did not. Based on the total number of inclusions in our database, 11 preoperative variables were included: donor age, donor BMI, recipient age, recipient sex, hypertension, diabetes mellitus, LabMELD, type of donor liver (DCD/DBD), cold ischemic period, recipient warm ischemic period, and distributed packs of red blood cells. Of these included factors, diabetes mellitus ($p = 0.01$) and LabMELD ($p = 0.02$) were significantly associated with developing AKI post-LT (Table 2).

3.2 | Postoperative AKI

The median increase between preoperative creatine level and highest creatine level during the first week post-LT in the TPCS group was 12 μmol/L (-4 - 52), versus 14 μmol/L (-3 - 42) in the no-TPCS group ($p = 0.0.94$) (Figure 1). In total, 128 (32%) of all recipients developed post-LT AKI, of which 41 recipients (31%) were in the TPCS group, versus 87 (33%) in the no-TPCS group ($p = 0.57$) (Table 2). There was no significant difference in the number of recipients that received post-LT RRT ($p = 0.61$) (Table 3) or in severity of post-LT AKI between both groups ($p = 0.90$) (Table 4). When comparing postoperative complications, no statistical difference was seen in Clavien-Dindo complication grade ≥ 3a between both groups (39% vs. 36%; $p = 0.64$).

3.3 | Graft and Patient Survival

Figure 2 shows the 1-year graft survival in both groups. There was no significant difference in 1-year graft survival between both groups ($p = 0.19$). Also, there was no significant difference in 1-year patient survival between both groups ($p = 0.41$) (Figure 3).

4 | Discussion

This cohort study demonstrates that a perioperative TPCS is not associated with less post-LT AKI in LT when using a side-to-side caval anastomosis. Previously, several small, retrospective studies have reported conflicting results regarding the effect of TPCS on post-LT creatinine clearance [17–20]. Pratschke et al. [17] previously described no significant difference with versus without TPCS in serum creatinine levels measured on the first, second, and seventh postoperative days. Whereas Pratschke et al. only included donation after brain death donors, the current study also includes donation after circulatory death donors, thereby giving a good representation of current practice in LT. Arzu et al. [19] also previously described no significant difference in creatinine values 1 and 3 days postoperative. In the study by Arzu et al., a significantly lower mean creatinine level at discharge was found in recipients who received a TPCS. However, the median number of days until discharge is not mentioned.

Studies by De Cenarruzabeitia et al. and Ghinolfi et al. previously describe a significant beneficial effect of TPCS on post-LT AKI [18, 20]. Whereas the study by De Cenarruzabeitia et al. described a significant difference in postoperative urea level when comparing an initial portal flow above 800 mL/min in favor of the TPCS group, there were no significant differences in postoperative creatinine levels. Furthermore, they do not specify between mild or severe post-LT AKI. Ghinolfi et al. described a significantly higher creatinine clearance on postoperative Day 3 in the TPCS group. However, there was no established policy for using TPCS, and the final decision was made on a case-by-case basis by the attending surgeon according to the individual preferences of the case, thereby introducing selection bias.

Finally, meta-analyses by Pratschke et al. [27] and Nacif et al. [28] showed conflicting results. Whereas the meta-analysis by Pratschke et al. showed a significant beneficial effect of a TPCS on post-LT AKI, Nacif et al. did not find a similar significant beneficial effect of a TPCS on post-LT AKI, even though both meta-analyses included the same studies. The I^2 values in both studies indicated substantial heterogeneity among the studies ($p < 0.05$), which could bring a potential bias to the results.

A possible explanation for a TPCS to not have a beneficial effect on post-LT AKI could be the use of the caval vein-sparing technique [29]. By preserving the caval vein, this technique leads to improved hemodynamics and better venous drainage of the kidneys than the caval vein insertion technique, thereby better preserving renal function.

In the current study, the number of perioperative distributed blood products in the no-TPCS group was significantly higher than in the TPCS group (4 vs. 3 units). Previous meta-analyses by Pratschke et al. [27] and Nacif et al. [28] did not find the

TABLE 1 | Donor, transplant, and recipient characteristics.

	TPCS ($n = 134$)	No-TPCS ($n = 260$)	p
Donor sex (female%)	47	52	0.32
Donor age (years)	46 ± 16	49 ± 15	0.33
Donor BMI (kg/m^2)	24 ± 4	25 ± 3	0.38
Recipient age (years)	55 ± 11	54 ± 11	0.15
Recipient BMI (kg/m^2)	26 ± 4	27 ± 5	0.33
Recipient sex (female%)	28	25	0.58
Etiology of liver disease (%)			
Metabolic disease	9	7	0.38
Acute etiology	6	4	0.34
Cholestatic liver disease	16	20	0.44
Alcoholic liver disease	22	29	0.15
Malignancy	25	16	0.02
Hepatitis B	3	5	0.28
Hepatitis C	3	4	0.80
Other cirrhosis	9	11	0.57
Other/ unknown	6	6	0.94
Hypertension (%)	53	46	0.32
Diabetes (%)	28	29	0.79
Chronic kidney disease (%)	39	32	0.16
ET-DRI	1.8 ± 0.3	1.8 ± 0.4	0.83
sRRI	2.0 ± 0.7	2.1 ± 0.7	0.51
DRM	1.4 ± 0.1	1.4 ± 0.1	0.44
BAR score	5.8 ± 3.9	6.2 ± 4.0	0.29
LabMELD	15 ± 8	16 ± 8	0.31
Liver grafts (DCD%)	**47**	**35**	**0.03**
Cold ischemic period (median, IQR, minutes)	521 (452–611)	507 (439–609)	0.47
Donor warm ischemic period (median, IQR, minutes)	13 (7–19)	17 (10–24)	0.23
Recipient warm ischemic period (median, IQR, minutes)	**35 (30–41)**	**32 (28–37)**	**< 0.001**
Operative time (median, IQR, minutes)	325 (278–379)	327 (284–390)	0.33
Distributed packs of red blood cells (median, IQR, units)	**3 (0–6)**	**4 (1–8)**	**0.02**
Fresh frozen plasma (median, IQR, units)	7 (4–11)	6 (2–10)	0.15
Cell saver (median, IQR, mL)	1200 (784–2077)	1079 (660–1833)	0.05

Note: Data are presented as mean ± SD, unless specified otherwise.

Abbreviations: BAR score, balance of risk score; DCD, donation after circulatory death; DRM, Donor Risk Model; ET-DRI, Eurotransplant Donor Risk Index; LabMELD, Laboratory Model for End-Stage Liver Disease Score; SD, Standard Deviation, sRRI, simplified recipient risk index; TPCS, temporary portocaval shunt.

same result. To our knowledge, there were no major changes in perioperative transfusion protocol. Furthermore, the number of perioperative distributed FFP transfusions, which have been described as the strongest predictor for AKI [30], did not significantly differ between both groups.

The WIP was significantly longer in the TPCS group. A possible explanation for this finding could be the introduction of initial arterial perfusion of the liver graft during the study period. It is thought that the arterial anastomosis is more difficult than the portal anastomosis, thereby leading to a few (but statistically significant) extra minutes of WIP.

There were significantly more DCD-LTs in the TPCS group. Next to classic risk factors such as perioperative renal function and comorbidities such as diabetes mellitus and hypertension, graft

TABLE 2 | Binary logistic regression to determine possible covariates in developing AKI after LT.

	B	S.E.	Wald	Sig.	Exp(B)
Donor age	−0.15	0.01	1.86	0.17	0.99
Donor BMI	−0.001	0.05	0.00	0.99	1.00
Recipient age	0.03	0.02	0.03	0.87	1.00
Recipient sex	0.63	0.39	2.66	0.10	1.88
Hypertension	0.18	0.33	0.30	0.58	1.20
Diabetes	**0.96**	**0.35**	**7.58**	**0.01**	**2.62**
LabMELD	**0.05**	**0.02**	**5.66**	**0.02**	**1.05**
Donor liver type (DBD vs. DCD)	−0.56	0.35	2.57	0.11	0.57
Cold ischemic period	0.00	0.00	3.13	0.08	1.00
Recipient warm ischemic period	0.00	0.02	0.00	1.00	1.00
Distributed packs of red blood cells	−0.02	0.03	0.22	0.64	0.99
Constant	−5.27	1.71	9.49	0.002	0.01

Abbreviations: AKI, acute kidney injury; DBD, donation after brain death; DCD, donation after circulatory death; LT, liver transplantation; LabMELD, Laboratory Model for End-Stage Liver Disease Score.

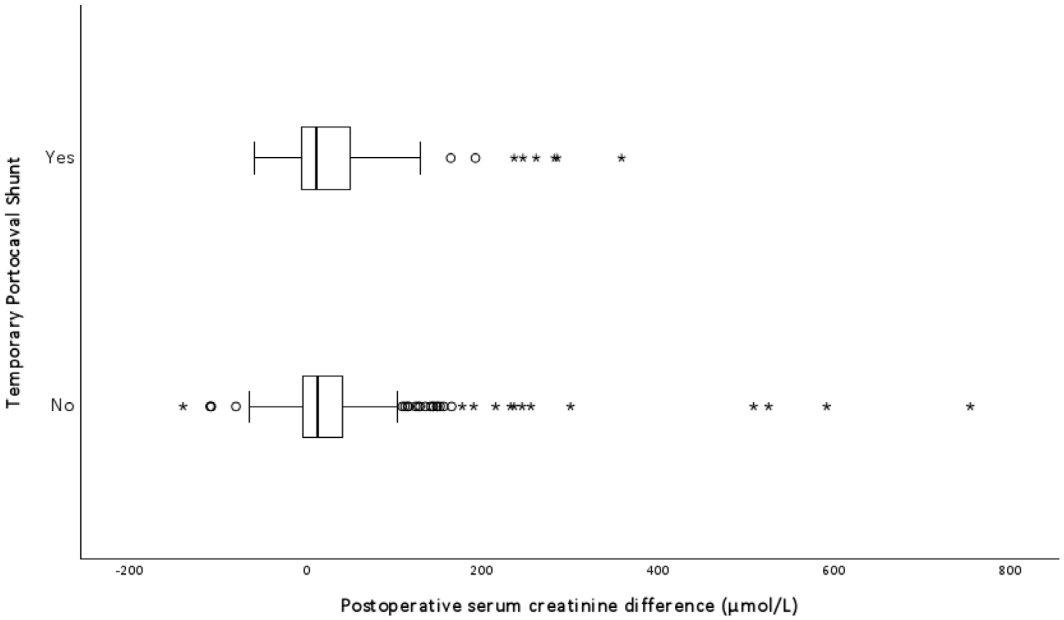

FIGURE 1 | Boxplot showing the median increase between preoperative creatine level and highest creatine level during the first week post-LT in the TPCS group versus the no-TPCS group.

TABLE 3 | Incidence of post-LT AKI.

		Temporary portocaval shunt		Total
		No	Yes	
Acute kidney injury	No	173 (67%)	93 (70%)	266
	Yes	87 (33%)	41 (31%)	128
Post-LT renal replacement therapy	No	243 (93%)	127 (95%)	370
	Yes	17 (7%)	7 (5%)	24
Total		260	134	394

AKI: $p = 0.57$.
Renal replacement therapy: $p = 0.61$.

TABLE 4 | Severity of post LT-AKI.

		Temporary portocaval shunt		Total
		No	Yes	
Acute kidney injury stage	None	173 (67%)	93 (70%)	266
	Stage 1	51 (20%)	22 (24%)	73
	Stage 2	11 (4%)	6 (4%)	17
	Stage 3	25 (10%)	13 (10%)	38
Total		260	134	394

AKI stage: $p = 0.90$.

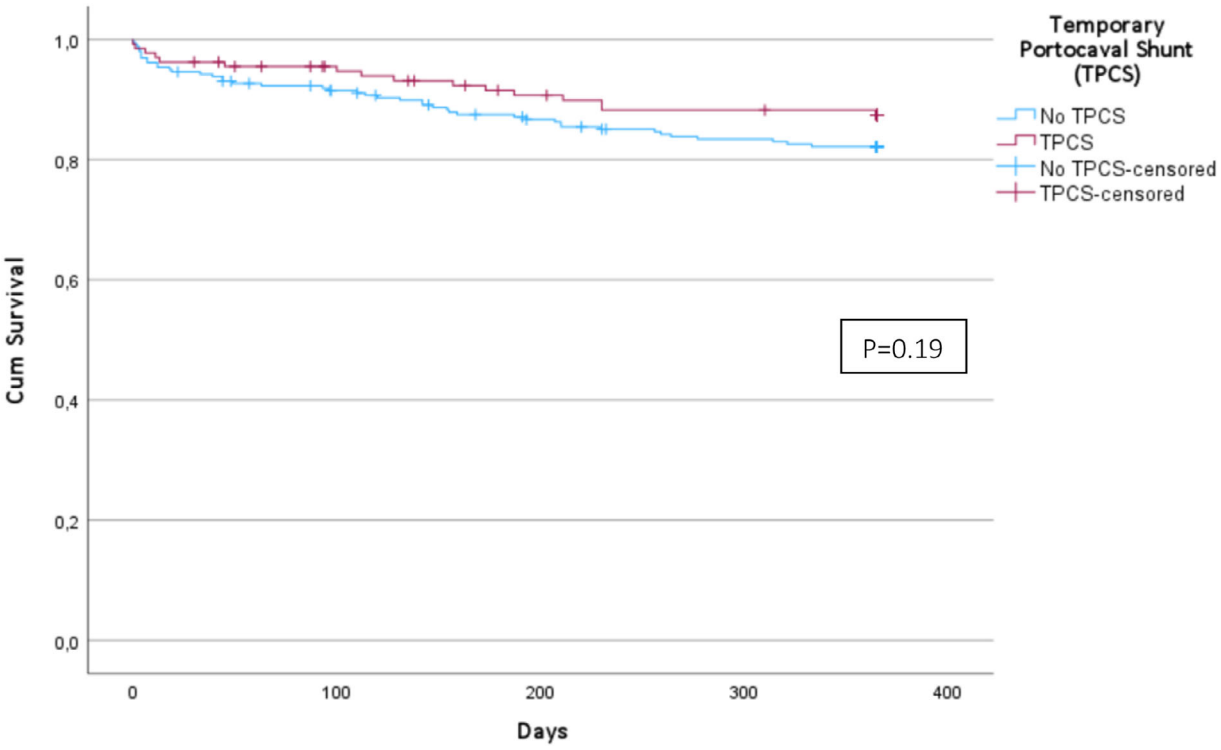

FIGURE 2 | One-year graft survival curve noncensored for death and retransplantation.

characteristics are increasingly being recognized as contributors to the development of post-LT AKI [31]. Even though there were significantly more DCD-LT in the TPCS group, there was no significant difference in the number of cases and severity of post-LT AKI. Also, when analyzing DCD-LTs separately, there was no significant difference between both groups ($p = 0.51$).

Currently, it is up to the surgeon's discretion whether to perform a TPCS during LT. The TPCS deviates portal flow and subsequently resolves portal hypertension, decompresses other portosystemic shunts, and prevents splanchnic compression. In our experience, it may therefore be beneficial in liver mobilization and removal, especially when the liver is very large and/or spontaneous shunts are around the liver itself. Interestingly, in our series there were less RBC transfusions in the TPCS group as compared to the control group.

This study has some limitations. First, the non-randomized character could bring a potential bias. Second, due to strict inclusion criteria, there was a relatively high number of excluded recipients. Even though the current study still includes a large cohort analyzing the effect of TPCS on post-LT AKI. Third, due to the large inclusion period, it is possible that small changes in anesthetic technique were implemented (e.g., use of different intraoperative and postoperative vasopressors). To our knowledge, no changes have been made in minimum values for mean blood pressure during transplantation, therefore not influencing perioperative and postoperative hemodynamic stability. Finally, during the study period, some small changes were made in operative technique. Initial arterial perfusion of the liver graft was implemented, thereby giving a possible bias to the study. Also, even though the use of machine preservation is changing the landscape of LT, in order to retain a homogenous group, we decided to exclude this group in the current study. In conclusion, this study shows that the application of a TPCS is not associated with less post-LT AKI or less severe post-LT AKI when using a caval-sparing LT technique, nor does it improve 1-year patient survival or 1-year graft survival.

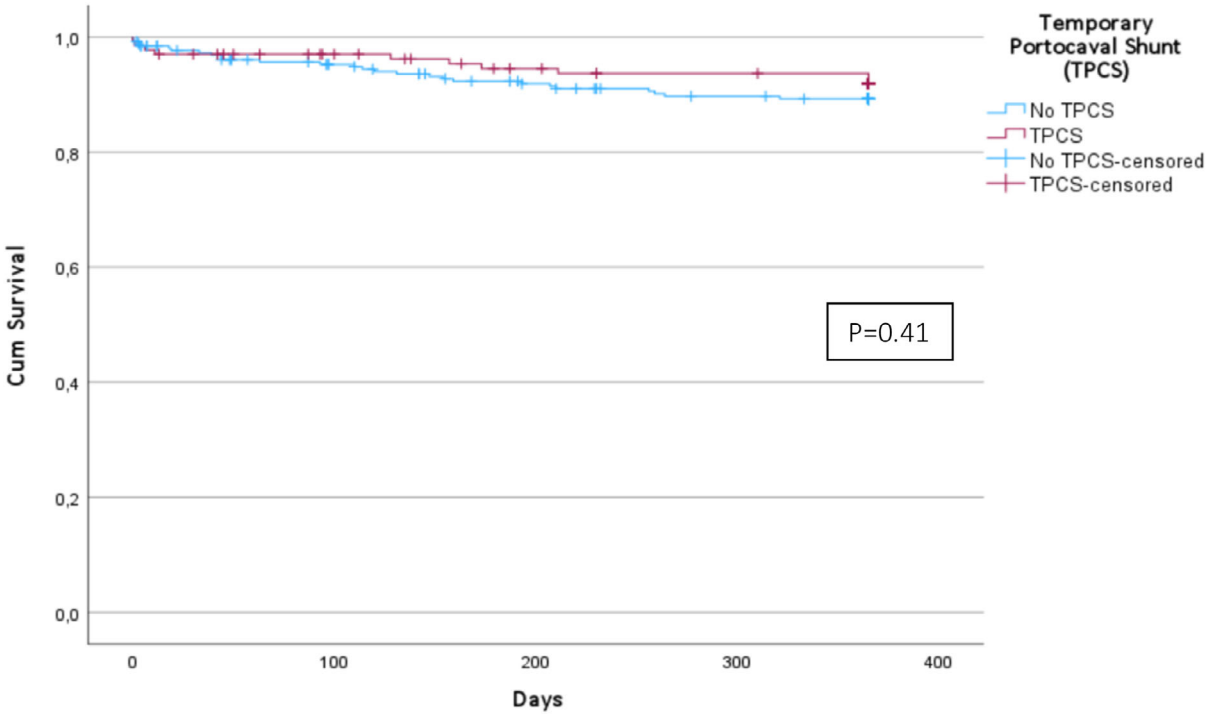

FIGURE 3 | One-year patient survival curve noncensored for retransplantation.

Author Contributions

Lars C. Pietersen: concept/design, data analysis/interpretation, drafting article, critical revision of article, approval of article, statistics. **Niels Broekman**: data analysis/interpretation, drafting article, critical revision of article, approval of article. **Marije Reekers**: drafting article, critical revision of article, approval of article. **Hein Putter**: data analysis/interpretation, critical revision of article, approval of article, statistics. **Maarten E. Tushuizen**: data analysis/interpretation, drafting article, critical revision of article, approval of article. **Ian P. J. Alwayn**: critical revision of article, approval of article. **Bart van Hoek**: concept/design, data interpretation, drafting article, critical revision of article, approval of article. **Andries E. Braat**: concept/design, data interpretation, drafting article, critical revision of article, approval of article.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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