Results in liver transplantation using grafts from donors after controlled circulatory death: A single-center experience comparing donor grafts harvested after controlled circulatory death to those harvested after brain death

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Abstract

Background. In recent years, interest in donation after cardiac death (DCD) has increased. Although DCD liver transplantation (LT) has demonstrated satisfactory long-term outcomes, different studies have shown poorer patient and graft survival after DCD than after donation after brain death (DBD). This study aimed to evaluate the results of LT using controlled DCD (cDCD) donors, specifically the incidence of primary non-function and ischemic cholangiopathy (IC), and to compare these results with those of LT using DBD in the same time period.

Methods. Between June 2012 and July 2018, we performed 66 transplants using cDCD and 258 with DBD grafts.

Results. The incidence of IC was similar in both groups (2% in DBD, 1.5% in DCD; $P = .999$). No significant differences were found for overall graft and patient survival rates between the groups at 1 and 2 years post-transplantation.

Conclusions. This study provided evidence that cDCD donors exhibit excellent graft and patient survival outcomes. When the warm ischemia time is <30 minutes and cold ischemia time is <6 hours, the graft and patient survival rates and the incidence of IC in DCD are similar to those in DBD, even when using donors without age restrictions.

Keywords
Donors and donation, donation after brain death, donors and donation: donation after circulatory death, immunosuppressant, liver disease, primary nonfunction

1 INTRODUCTION

Liver transplantation (LT) is a highly successful, life-saving modality for treating end-stage liver disease with 1- and 5-year patient survival rates up to 80% and 70%, respectively.[1] This success, however, has resulted in an increasing demand for this procedure, and the discrepancy between supply and demand has resulted in significant morbidity and mortality for patients awaiting LT. To overcome this shortage, significant efforts have been made to find alternatives and offer LT to more patients. The expansion of the donor pool involves the use of extended criteria donors (ECDs), and these donors, although not formally characterized, represent a wide spectrum of donors who may contain some unfavorable characteristics historically associated with poorer graft and patient survival outcomes. Characteristics of this heterogeneous group of donors include advanced age, significant macrovesicular steatosis, hypernatremia, and donation after cardiac death (DCD). DCD donors represent a specific type of ECD for whom death is declared on the basis of cardiopulmonary criteria rather than cessation of whole brain function. In recent years, interest in DCD liver grafts has grown in parallel with the successful results observed after kidney transplants using DCD grafts. Although DCD-LT has shown satisfactory long-term outcomes, different studies have demonstrated poorer patient and graft survival outcomes than those obtained by donation after brain death (DBD).[1, 2] Nevertheless, recent studies have displayed similar graft and recipient survival outcomes following DCD-LT compared with livers transplanted from DBD donors.[3-6]

Several studies have been designed to identify risk factors in DCD-LT. Different donor factors, such as age, weight, or body mass index (BMI), procedure-related factors such as cold ischemia time (CIT) or warm ischemia time (WIT), or recipient model for end-stage liver disease (MELD) scores have been significantly associated with graft failure after DCD-LT.[7, 8] A published meta-analysis from the United States[9] demonstrated poorer

outcomes after controlled DCD-LT than after DBD-LT, mainly due to higher rates of biliary complications. Biliary complications, including ischemic cholangiopathy (IC), are a major source of morbidity after LT.[8-11] IC is defined as strictures, irregularities, or dilatations of the intrahepatic or extra-hepatic bile ducts of the liver graft, excluding isolated strictures at the bile-duct anastomosis.[12] IC is difficult to predict because the pathophysiology is poorly understood. It has been attributed to prolonged donor warm ischemic times leading to microcirculatory impairment or thrombosis,[13] the solitary hepatic artery supply of the peribiliary capillary plexus,[4] sensitivity of biliary epithelium to ischemia reperfusion injury,[14] failure of biliary epithelium to regenerate,[15] and the composition of bile, particularly bile-salt toxicity contributing to bile-duct injury.[16] IC is often refractory to treatment, and it leads to the requirement of retransplantation in a significant proportion of patients. Although not all patients with IC require retransplantation, this complication can result in considerable patient morbidity, including biliary sepsis, prolonged antibiotic therapy, and multiple endoscopic or percutaneous biliary procedures.[8] Single-center studies have found that donor weight over 100 kg,[10] advanced donor age,[8, 17] and CIT >8 hours[8] significantly increase the risk of IC. In previous reports, our group published our experience with the DCD Maastricht Category II (Uncontrolled DCD; uDCD), showing low graft survival and a high rate of biliary complications.[18, 19] Since 2012, we have implemented the DCD Maastricht Category III (Controlled DCD; cDCD) program for liver transplants at our center. The legal basis for the use of controlled DCD in Spain is established by Royal Decree 1723/2012.[20]

This study aimed to evaluate the results of LT using cDCD donors, specifically examining the incidence of primary non-function and ischemic cholangiopathy, and to compare these results using DBD during the same time period. This protocol has been approved by the institutional ethics committee from our center, and all patients provided informed consent.

2 MATERIALS AND METHODS

This is a retrospective single-center study. Between June 2012 and July 2018, we performed a total of 326 liver transplants in 304 patients. Sixty-six of these transplants were performed using cDCD grafts (study group); two of these were performed with

uDCD and 258 with DBD (control group). The cDCD organs were allocated in our own hospital (n = 20) or sent from other hospitals without cDCD liver programs (n = 46). Each hospital performed organ recovery using normothermic regional perfusion (NRP) (n = 42) or super rapid recovery (SRR) (n = 24). The long-term outcomes after cDCD were analyzed. Exclusion criteria were recipients with DCD grafts in Maastricht category II (uDCD, n = 2) and patients with a follow-up of <1 month (one patient after DCD liver transplant and six patients after DBD liver transplants). Finally, the study group included 65 DCD-LT (24 SRR and 41 NRP), and the control group included 252 DBD-LT. We also analyzed the results in the DCD group comparing the two techniques for organ recovery: NRP or SRR.

There were no differences in surgical technique for DCD, nor were there differences in duct management. T-tubes were used in all patients for duct-to-duct biliary anastomosis. The management of biliary strictures was performed via ERCP, and if this was not possible or failed, surgery was the alternative.

2.1 DCD donor organ procurement

Withdrawal support occurred either in the intensive care unit (ICU) or in the induction room according to the donor center policy. The death certification was performed by a physician independent from the transplant team. The process for organ recovery varies according to center preferences, and each individual donor hospital determines the process by which cDCD organs are recovered. These options include NRP with pre-mortem vessel cannulation or super rapid recovery (SRR).[21]

2.1.1 SRR

Once death has been declared, heparin is administered (if it has not been given previously), and the surgical team enters to perform rapid vessel cannulation and cold perfusion of the abdominal organs. The abdomen is incised through a midline laparotomy, the distal abdominal aorta is cannulated, and the supraceliac aorta is clamped. The cold perfusion solution is flushed and vented through the inferior vena cava, and crushed ice is placed in the abdomen to cool the organs topically.[21]

2.1.2 NRP with pre-mortem vessel cannulation

Prior to cannulation, a bolus of heparin is administered to the potential donor (300 IU/kg, IV.). Cannulation of the femoral vessels may be performed open or percutaneously. The contralateral femoral artery is cannulated with a Fogarty balloon catheter, which is advanced into the supraceliac aorta under radiographic control. Once the cannulation procedure is completed, the ventilatory support is withdrawn. The arterial blood pressure is continuously monitored, and the time at which the systolic blood pressure drops below 60 mm Hg is recorded, marking the start of functional WIT. Death is declared after a 5-minute period of respiratory and circulatory arrest in accordance with Spanish legislation. Once death has been declared, the Fogarty balloon catheter is inflated, and the NRP circuit is initiated.[21]

2.2 DCD donor selection

There were no absolute criteria for DCD graft selection. In general, age was not a limiting factor. Donor WIT acceptance was limited to 30 minutes, and the graft appearance after cold perfusion was assessed subjectively by surgeons. Only organs with adequate perfusion were considered valid for graft selection.

2.3 Recipient selection of DCD grafts

The candidates for DCD grafts were similar to those for DBD, and no restrictive criteria were applied for recipient selection. In general, recipients with primary sclerosing cholangitis (an indication marginally represented in our population) or retransplants are not transplanted with DCD livers. When a cDCD graft was available, it was offered to the sickest patient according to the MELD scale.

2.4 Endpoints

The endpoints of the study were the evaluation of incidence of primary non-function, IC, and graft and patient survival. Primary non-function is defined as immediate graft failure resulting in either emergent retransplantation or death. As it has been described in the literature, IC was defined in our study as “diffuse intrahepatic strictures without the

presence of concomitant hepatic artery thrombosis" (HAT); thus, we did not include patients with IC secondary to arterial thrombosis or arterial stenosis. The diagnosis of IC was made either by magnetic resonance cholangiography or endoscopic retrograde cholangiography. The biliary stricture was differentiated as anastomotic or non-anastomotic. A non-anastomotic biliary stricture was defined as a stricture more than 1 cm above the biliary anastomosis, requiring endoscopic or radiological procedures.

We also analyzed the occurrence of postoperative acute kidney injury (AKI), length of hospital stay, vascular complications (hepatic artery stenosis and HAT), biliary complications, and acute cellular rejection.

AKI was defined according to the RIFLE criteria for acute kidney injury: peak serum creatinine ≥2 times the baseline level.[22]

2.5 General variables

The donor and graft variables included the following: age, gender, body mass index (BMI), cause of death, donor WIT, CIT, and UK risk score.[23] An allograft biopsy was performed immediately after reperfusion, and the steatosis level was graded. A pre-biopsy of the liver is a not a routine procedure for DCDs. The graft appearance after cold perfusion was assessed subjectively by surgeons, and only organs with adequate perfusion were considered valid for graft selection.

Recipient information included age, gender, etiology of liver disease, and MELD score.

2.6 Immunosuppression

The immunosuppressive regimen contained a calcineurin inhibitor (mainly tacrolimus to maintain a trough concentration in the range 8-10 ng/mL until month 3 of transplantation, after which, the target range was decreased to 4-8 ng/mL), mycophenolate, and prednisone, tapered to achieve elimination at the start of month 4. In patients with renal dysfunction (defined as GFR <60 mL/min for more than 3 months), a renal-sparing immunosuppressive protocol was used. This regimen combines an interleukin (IL-2) receptor antagonist for induction, half dose of tacrolimus (to maintain a trough level in the range 5-8 ng/mL), and mycophenolate.

2.7 Statistical analysis

A descriptive analysis was performed for all the studied variables. Quantitative data are expressed as mean ± standard deviation (SD) or median and range, and qualitative variables are expressed in frequencies and percentages.

Donor and receptor characteristics and prognoses after transplantation were compared between the DBD and DCD groups. Chi-squared or Fisher's exact tests were used for categorical variables, and Student's *t* or Mann-Whitney tests were used for quantitative ones. Normality was tested by the Kolmogorov-Smirnov test.

Patient survival was analyzed with Kaplan-Meier survival curves, which were compared between the DBD and DCD groups using the log-rank test. A competing risk approach was used to estimate graft survival. Death with a functioning graft was considered a competing risk event. The death-adjusted cumulative incidence of the marginal probability of graft loss was obtained, and the cumulative incidences in the competing risk data were compared using the modified log-rank test.[24]

Data analysis was performed using SPSS ver 21.0 for Windows. The cumulative incidence in competing risk analyses was calculated using the software package R. A two-sided *P* value <.05 was considered statistically significant.

3 RESULTS

3.1 Liver transplantation with organs from DCD vs DBD

A total of 317 liver transplants between June 2012 and July 2018 were included in the analysis. The study group included 65 DCD-LT, and the control group included 252 DBD-LT. All patients had at least 6 months of follow-up. The mean donor age was 59.7 ± 12.9 years in DCD and 58.9 ± 15.3 in DBD. In the DCD group, 60% of the donors were male, and the mean BMI of the donor was 27.2. In the DBD group, 54.4% of the donors were male, and the mean BMI of the donor was 26.8. The mean recipient age was 57.6 ± 7.1 years in the DCD group and 57.5 ± 8.9 years in the DBD group. The most common causes of chronic disease were alcohol-related cirrhosis, viral hepatitis cirrhosis, and cholestatic disease, while 41.6% of the patients in DCD group and 36.1% in DBD group had a hepatocellular carcinoma. None of these variables achieved significance. The mean

MELD score was 12.9 ± 6.6 in the DCD group and 14.3 ± 6.3 in the DBD group (*P* = .043). The most common donor cause of death was cerebrovascular accident (CVA), in both the DCD and DBD groups (Table **1**).

As previously mentioned, an allograft biopsy was performed immediately after reperfusion. Data of the post-reperfusion biopsies in DCD were normal: 9.2%; steatosis <30%: 50.8%; steatosis >30%: 4.6%; mild ischemia reperfusion injury: 60%; moderate ischemia reperfusion injury: 3.02%; mild centrozonal necrosis: 9.2%; and moderate centrozonal necrosis: 6.1%.

Primary non-function was present in two grafts (3%) in the DCD group with zero incidence in the DBD group. The incidence of IC was similar in the two groups (1.5% in DCD, 2% in DBD; *P* = .999). No significant differences were found for overall graft and patient survival rates between the groups at 1 and 2 years post-transplantation (Figures **1** and **2**). Three patients died in the DCD group of separate causes: cancer (Pleural mesothelioma), mesenteric ischemia, and a traffic accident. In the DBD group, 32 patients died. The most common causes of death in the DBD group were sepsis and multiorgan failure, “de novo” cancer, and the recurrence of previous disease, mainly hepatocellular carcinoma.

As shown in Table **2**, the incidence of postoperative AKI, length of intensive care stay, length of hospital stay, vascular complications (hepatic artery stenosis and HAT), biliary complications, and acute cellular rejection were similar in the two groups. Peaks of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were significantly higher in the DCD group. Remarkably, no difference was observed with respect to alkaline phosphatase or bilirubin levels at 1-year post-transplantation between both groups.

3.2 Liver transplantation with organs from DCD: comparing the methods for graft recovery

We analyzed the results of the DCD group, comparing the two techniques for organ recovery (NRP or SRR), examining the incidence of biliary complications, IC, vascular complications, and graft survival (Table **3**). No differences were detected between the groups. Although the variance was not significant, a higher proportion of grafts recovered with the super rapid technique was lost after LT 16.7% (n = 4) vs 4.9% (n = 2) of those

recovered with NRP (*P* = .183). The causes of graft loss with the super rapid technique were due to PNF (n = 1), HAT (n = 1), and biliary complications (n = 2). We lost 2 grafts with NRP due to artery thrombosis and PNF (Figure **3**).

4 DISCUSSION

The use of DCD organs has the benefit of increasing the number of potential recipients and reducing waiting list mortality rates. Currently, the use of DCD donors for organ transplantation is heavily weighted toward the use of kidneys, for which it is broadly accepted that the outcomes for DCD donors are equivalent to those obtained from DBD donors. In the liver transplant scenario, this equality has not been uniformly accepted. However, the last decade has witnessed a significant increase in both the absolute number of liver transplants performed with DCD liver allografts and the number of institutions performing DCD liver transplants. There are several recent publications from centers in North America and the United Kingdom describing the successful transplantation of cDCD livers[3-6] without a significant increase in the incidence of PNF or HAT, both of which have been previously reported in the literature as significant causes of graft failure. Our results confirm the findings of former studies and suggest that the rates of PNF and HAT are not significantly higher in DCD grafts than those in DBD grafts, even when using older donors. As De Olivera et al did,[4] we demonstrated a low incidence of IC (1.5%) in the DCD cohort, similar to that observed in the DBD group. We consider this figure to be remarkable, considering the mean age of our donors (59.7 ± 12.9 years), significantly higher than in other research groups. In fact, different studies have identified donor age asone1 of the main risk factors for the occurrence of IC. De Vera et al[17] in a multivariate analysis found that only transplantation of donors aged >60 years (risk ratio =5.61 [1.0-32.0]; *P* = .05) was an independent predictor of the development of biliary complications, and Foley et al[8] found in a multivariate analysis that CIT >8 hours (Hazard ratio = 2.46 [1.0-6.1]; *P* = .05) and donor age >40 (Hazard Ratio = 2.90 [1.1-7.6]; *P* = .02) significantly increased the risk of IC. One possible explanation for the low incidence of IC detected in our patients could be the short WIT and CIT (mean WIT 13.2 ± 5.6 minutes and mean CIT <6 hours). The absence of differences in graft and patient survival after 2 years of follow-up between DBD and DCD is probably due to the low incidence of PNF, HAT, and IC.

One of the limitations for acceptance of DCD grafts for LT is the uncertainty of short-term (because of PNF or HAT) or medium-term survival (because of IC). In a recent publication, A. Schlegel et al[23] developed a new prediction model for graft loss in DCD liver transplantation, the UK DCD Risk Score. This score involves seven clinically relevant risk factors: donor age, donor BMI, functional donor warm ischemia, cold storage, recipient age, recipient laboratory model for end-stage liver disease, and retransplantation. Three risk classes were defined as follows: low risk (≤5 points), high risk (>5 ≤ 10 points), and futile (>10 points). More than 10 points significantly increase the risk for graft loss caused by PNFs and IC. The UK risk score for our patients was less than 10 points (5.8 ± 3.0 points; Figures **4** and **5**), in the lower range of the high risk group, due to the short warm and CIT and the very low incidence of retransplantation with DCD grafts. This figure is in agreement with the low incidence of PNF and IC observed in our study.

It is already known that AST and ALT levels are higher in DCD recipients because of the ischemia reperfusion injury associated with this type of donor. The clinical impact of these parameters is not well defined. Recently, Leithead et al[25] described an increased frequency of AKI after LT from DCD grafts, suggesting that hepatic ischemia reperfusion injury could play a critical role in the pathogenesis of post-transplant renal dysfunction. In this study, the peak of perioperative aspartate aminotransferase, a surrogate marker of hepatic ischemia reperfusion injury, was the only consistent predictor of renal dysfunction after DCD transplantation. In our study, we also observed a significantly higher peak of AST and ALT within the first 48 hours in DCD recipients, but this was not associated with the development of AKI. Only 6.2% of DCD and 11.5% of DBD recipients developed AKI in our study.

One of the endpoints of our study was to analyze the results of cDCD according to the technique used for organ recovery (NRP or SRR). Most centers use these methods, but to the best of our knowledge, there is no information available comparing their outcomes for recovering grafts. We have had the chance to compare the two techniques, but perhaps because of the reduced number of patients, we could not observe significant differences with respect to the incidence of PNF or IC. However, the incidence of graft loss increased from 4.9% to 16.7% when the SRR procedure was used. As Hessheimer et al pointed out,[26] the use of post-mortem NRP could neutralize the risk of using older donors by

returning cDCD livers to their pre-arrest state of viability. To reinforce this hypothesis, the warm ischemic time was significantly lower in the NRP group.

These excellent results encouraged us to increase the number of transplants with DCD donors and could also stimulate other centers to consider this type of donation as an effective policy to increment organ supply. In our center, after the implementation of the cDCD program, the number of transplants using DCD grafts increased from 3.9% in 2012 to 32.7% in 2017.

There are several limitations in the present study related to its observational nature and the non-random distribution of potential cDCD donors; the short follow-up period (24 months) and the number of patients in the DCD group. Since most of the known consequences of DCD grafts develop soon after the transplant, 24 months could be adequate to examine the outcomes of this type of donation. In contrast, the advantage of analyzing results of two different techniques for organ recovery (NRP or SRR) is countered with the low number of patients in each group.

In conclusion, this study provides evidence that controlled DCD donors offer excellent medium-term graft and patient survival rates. When using short WIT and CIT, graft and patient survival and the incidence of IC are similar to those in DBD grafts, even when using donors without any age restriction.

CONFLICT OF INTEREST

The authors of the present manuscript do not have any conflict of interest to disclose.

AUTHORS' CONTRIBUTIONS

Alejandra Otero, M. Angeles Vázquez, and Francisco Suárez designed the study, participated in the acquisition of data, analyzed and interpreted the data, drafted and critically reviewed the manuscript, and approved the final version for publication. Sonia Pértega ran the statistical analysis, and Jose Ignacio Rivas, Fernando Mosteiro, and Manuel Gómez participated in the acquisition of data, critically reviewed the manuscript, and approved the final version for publication.

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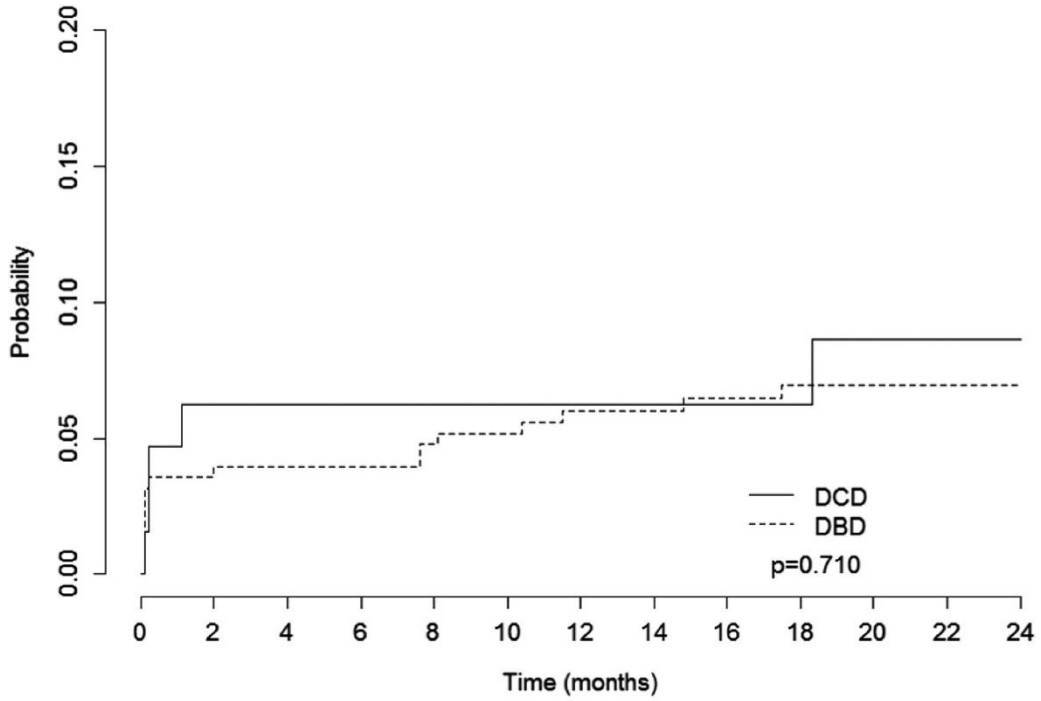
Table 1. Characteristics of donors and LT recipients in DBD group and in DCD group

	DBD	DCD	*P*
Donor age (y) (mean)	58.9 (15.3)	59.7 (12.9)	.825
Receptor age (y) (mean)	57.5 (8.9)	57.6 (7.1)	.981
Donor sex (male %)	137 (54.4%)	39 (60%)	.415
Receptor sex (male %)	182 (79.5%)	53 (82.8%)	.554
Cause of chronic disease (n/%)			
Alcoholic cirrhosis	126 (50%)	38 (58.5%)	-
Viral cirrhosis	60 (23.8%)	14 (21.6%)	-
Cholestatic disease	9 (3.6%)	1 (1.5%)	-
Other	57 (22.7%)	12 (18.4%)	-
Hepatocellular carcinoma (n/%)	91 (36.1%)	27 (41.6%)	.505
Meld (mean)	14.3 (6.3)	12.9 (6.6)	.043
Donor BMI (mean)	26.8 (4.0)	27.2 (4.4)	.317
UK risk score (mean)		5.8 (3.0)	
Donor cause of death (n/%)			
CVA	183 (72.9%)	41 (63%)	-
Anoxic brain injury	25 (10%)	10 (15.4%)	-
Traumatic brain injury	30 (12%)	7 (10.8%)	-
Other	13 (5.2%)	7 (10.8%)	-
Donor functional warm ischemic time (min)		13.2 (5.6)	-
CIT (min) (mean)	349.4 (89.2)	359 (84)	.312
Steatosis (n/%)			
None	105 (42%)	29 (44.6%)	-
<30%	124 (49.6%)	33 (50.8%)	-
>30%	21 (8.4%)	3 (4.6%)	-

Note.

Quantitative variables are shown as mean (standard deviation).

Qualitative variables are described as number (percentages).



	Probability				
	1 month	**3 months**	**6 months**	**12 months**	**24 months**
DCD					
Death with functioning graft	0%	0%	3.1%	3.1%	3.1%
Graft loss	4.7%	6.2%	6.2%	6.2%	8.6%
Alive with functioning graft	95.30%	93.80%	90.70%	90.70%	88.30%
DBD					
Death with functioning graft	0.0%	1.6%	2.8%	4.4%	7.1%
Graft loss	3.6%	4.0%	4.0%	6.0%	6.9%
Alive with functioning graft	96.40%	94.40%	93.20%	89.60%	86.00%

Figure 1. Probability of graft loss

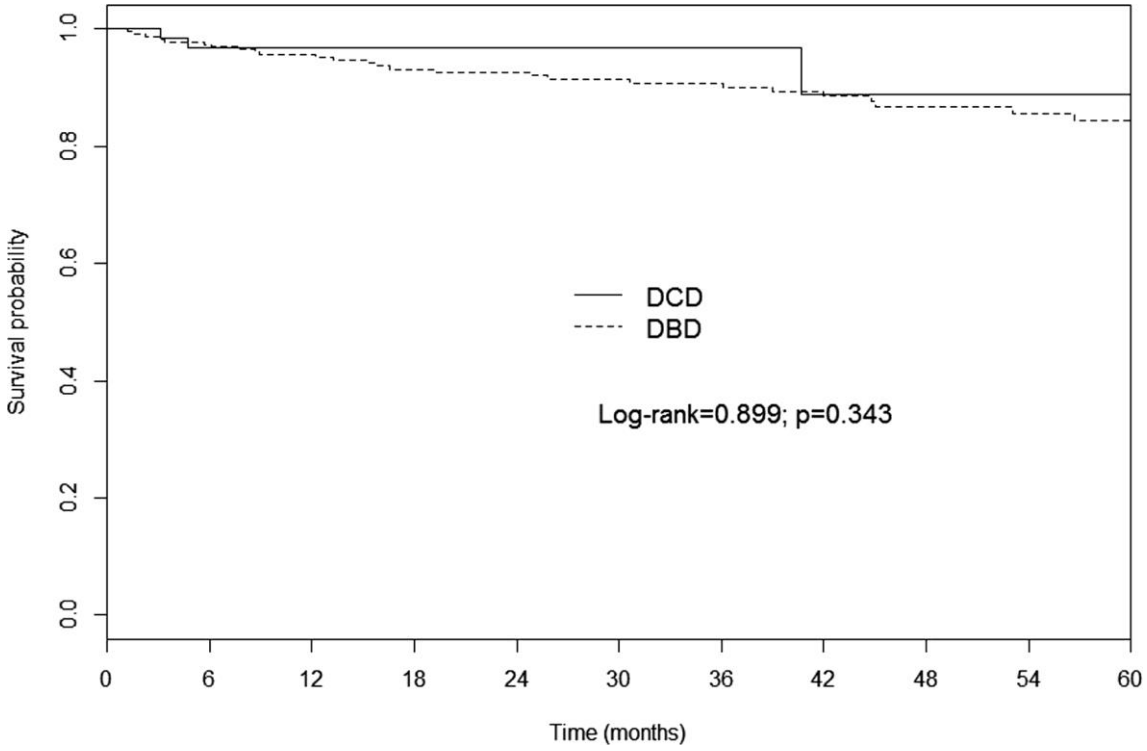


Figure 2. Patient survival

Table 2. Operative characteristics and complications by donor type

	DBD	DCD	*P*
Peak AST (UI/L) (median)	1128 (2662)	1798 (1771)	.001
Peak ALT (UI/L) (median)	1080 (1838)	1551 (1435)	.002
Bilirubin at 1 y (mg/dL) (median)	0.9 (1.0)	0.7 (0.6)	.220
Alkaline phosphatase at 1 y (mg/dL) (median)	246 (235)	198 (145)	.196
INR (median)	1.4 (0.3)	1.3 (0.2)	.544
Primary non-function (n/%)	0	2 (3.1%)	-
Biliary complications (n/%)	80 (32.3%)	13 (23.6%)	.083
IC	5 (2%)	1 (1.5%)	.999
Bile leak	10 (12.5%)	0	-
Anastomotic stricture	45 (56.3%)	6 (46.2%)	-
AKI (n/%)	28 (11.5%)	4 (6.2%)	.211
Hepatic artery thrombosis (n/%)	17 (6.7%)	3 (4.6%)	.775
Hepatic artery stenosis (n/%)	24 (9.5%)	4 (6.2%)	.393
Acute cellular rejection (n/%)	27 (10.8%)	4 (6.3%)	.280
Hospital stay (days) (median)	25 (17)	20 (14)	.010
Intensive care stay (days) (median)	5.9 (8)	4.6 (3.7)	.169

Note

Quantitative variables are shown as mean (standard deviation).

Qualitative variables are described as number (percentages)

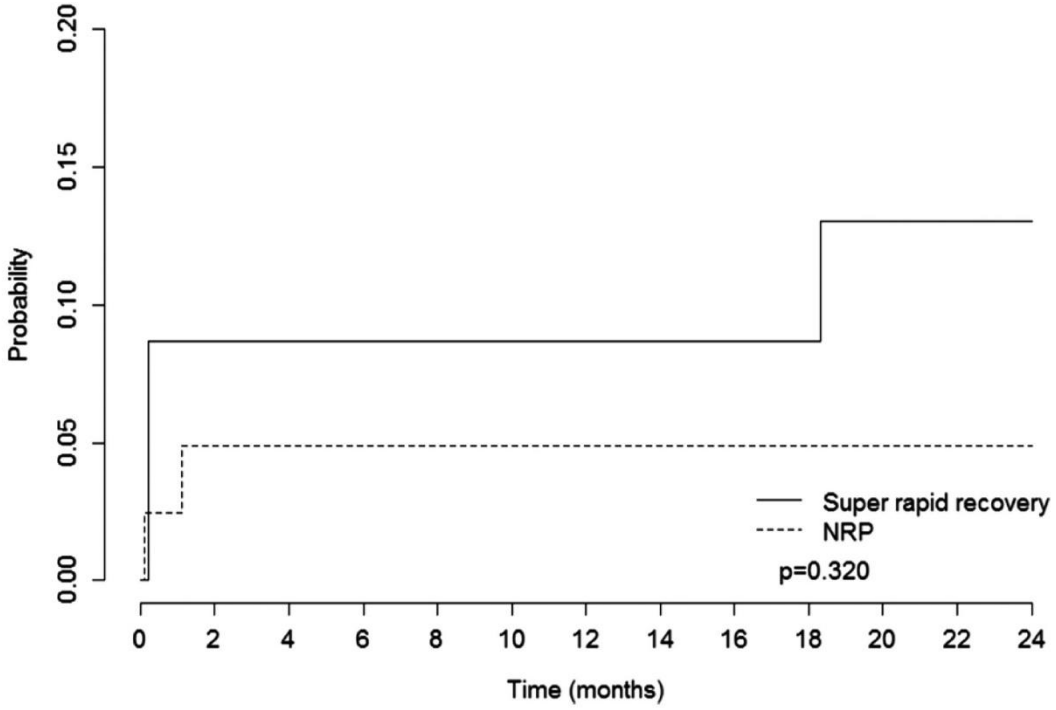
Table 3. Complications in DCD by technique for organ recovery

	NRP	Super rapid recovery	*P*
Biliary complications (n/%)	6 (15.4%)	7 (30.4%)	.203
IC (n/%)	0	1 (4.2%)	.369
Hepatic artery thrombosis (n/%)	2 (4.9%)	1 (4.2%)	.999
Hepatic artery stenosis (n/%)	2 (4.9%)	2 (8.3%)	.622
Primary non-function (n/%)	1 (2.4%)	1 (4.1%)	-
Warm ischemic time (min) (mean)	11.3 (5.1)	16.3 (5.2)	.001
UK donor risk index (DRI) (mean)	4.8	6.7	-

Note

Quantitative variables are shown as mean (standard deviation).

Qualitative variables are described as number (percentages).



		Probability			
	1 month	3 months	6 months	12 months	24 months
NRP					
Death with functioning graft	0%	0%	0%	0%	0%
Graft loss	2.4%	4.9%	4.9%	4.9%	4.9%
Alive with functioning graft	97.60%	95.10%	95.10%	95.10%	95.10%
Super rapid recovery					
Death with functioning graft	0%	0%	8.7%	8.7%	8.7%
Graft loss	8.7%	8.7%	8.7%	8.7%	13.0%
Alive with functioning graft	91.30%	9.30%	82.60%	82.60%	78.30%

Figure 3. Probability of graft loss in DCD comparing two methods for recovering

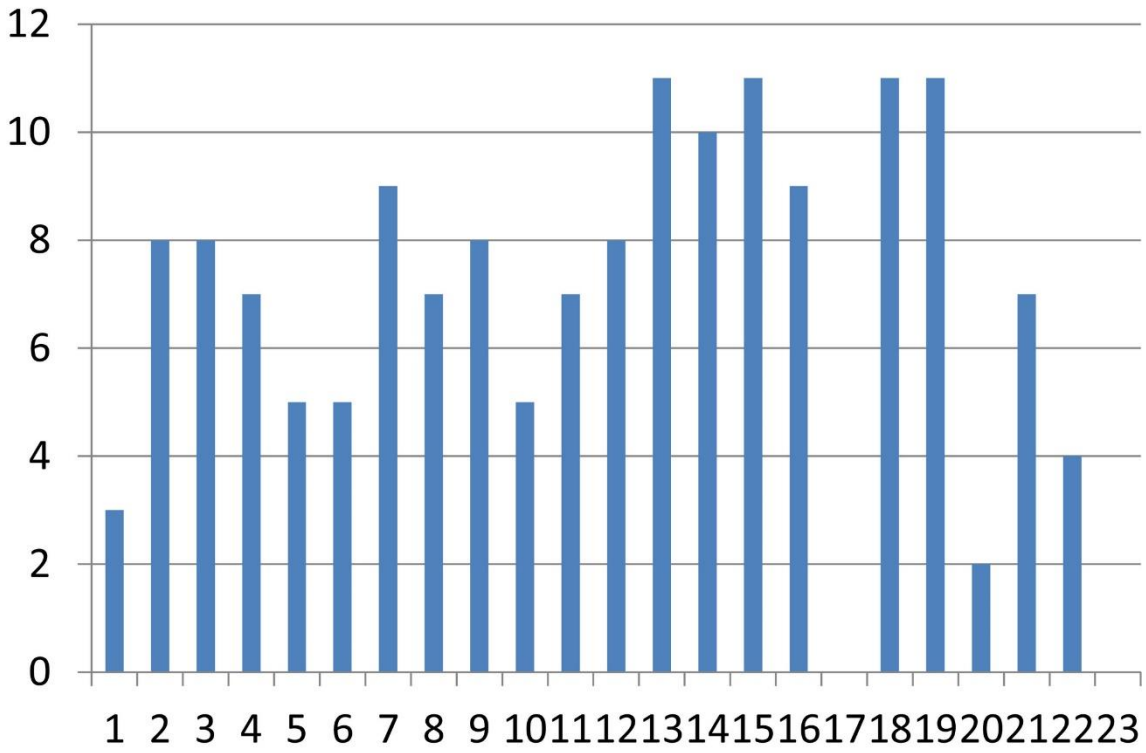


Figure 4. Histogram of the UK DCD risk score in super rapid recovery (3,8,8,7,5,5,9,7,8,5,7,8,11,10,11,9,0,11,11,2,7,4,0)

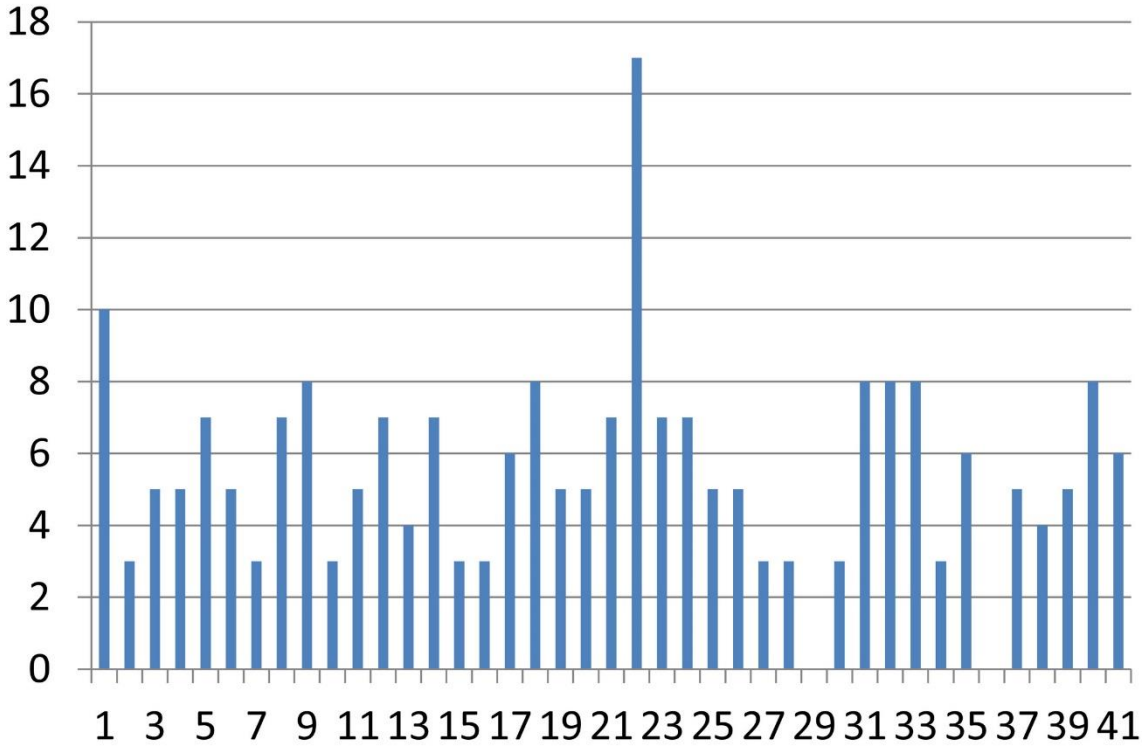


Figure 5. Histogram of the UK DCD risk score in NRP

(10,3,5,5,7,5,3,7,8,3,5,7,4,7,3,3,6,8,5,5,7,17,7,7,5,5,3,3,0,3,8,8,8,3,6,0,5,4,5,8,6)