Transplant International

Transplant International ISSN 0934-0874

ORIGINAL ARTICLE

Results of kidney transplantation from controlled donors after cardio-circulatory death: a single center experience

Hieu Ledinh,[1] Laurent Weekers,[2] Catherine Bonvoisin,[2] Jean-Marie Krzesinski,[2] Josée Monard,[1] Arnaud de Roover,[1] Jean Paul Squifflet,[1] Michel Meurisse[1] and Olivier Detry[1]

1 Department of Abdominal Surgery and Transplantation, University Hospital of Liège, University of Liège, Liège, Belgium
2 Department of Nephrology, University Hospital of Liège, University of Liège, Liège, Belgium

Keywords
brain death, organ preservation, primary graft dysfunction, risk assessment, treatment outcome, warm ischemia.

Correspondence
Hieu Ledinh, Department of Abdominal Surgery and Transplantation, University Hospital of Liège, University of Liège, Sart Tilman B35, 4000 Liège, Belgium.
Tel.: +32 4 366 72 16; fax: +32 4 366 70 69;
e-mail: ledinhhieu@pnt.edu.vn

Conflicts of Interest
The authors have declared no conflicts of interest.

Received: 25 July 2011
Revision requested: 12 August 2011
Accepted: 14 November 2011
Published online: 23 December 2011

doi:10.1111/j.1432-2277.2011.01402.x

Summary

The aim of this study was to determine results of kidney transplantation (KT) from controlled donation after cardio-circulatory death (DCD). Primary end-points were graft and patient survival, and post-transplant complications. The influence of delayed graft function (DGF) on graft survival and DGF risk factors were analyzed as secondary end-points. This is a retrospective mono-center review of a consecutive series of 59 DCD-KT performed between 2005 and 2010. Overall graft survival was 96.6%, 94.6%, and 90.7% at 3 months, 1 and 3 years, respectively. Main cause of graft loss was patient's death with a functioning graft. No primary nonfunction grafts. Renal graft function was suboptimal at hospital discharge, but nearly normalized at 3 months. DGF was observed in 45.6% of all DCD-KT. DGF significantly increased postoperative length of hospitalization, but had no deleterious impact on graft function or survival. Donor body mass index ≥30 was the only donor factor that was found to significantly increase the risk of DGF ($P < 0.05$). Despite a higher rate of DGF, controlled DCD-KT offers a valuable contribution to the pool of deceased donor kidney grafts, with comparable mid-term results to those procured after brain death.

Introduction

Confronted with the universal critical organ shortage, many transplant centers have started the use of donation after cardio-circulatory death (DCD) as an alternative donor source. Results of kidney transplantation (KT) from DCD over the past 30 years showed comparable results with those from donation after brain death (DBD) [1–7]. These results of DCD-KT have led Belgian transplant centers to revisit this option and urged the Belgian National Council of Physicians on organ procurement from DCD [8]. The first DCD-KT was performed in Belgium in 2000, and up to now all seven Belgian transplant centers have active DCD-KT programs [9,10]. In 2009, there were 60 DCD procurements [21.7% of the deceased donor (DD) pool] and 74 DCD-KT (17.3% of the DD kidney pool) in comparison with 9 DCD procurements (3.8%) and 14 DCD-KT (3.9%) in 2005. A preliminary report over 44 DCD-KT in Belgium during the 2003–2005 period showed a delayed graft function (DGF) rate of 20.5% and a primary nonfunction (PNF) rate of 9.1%. DCD kidneys preserved by machine perfusion had a significant lower rate of DGF than cold-stored kidneys (25% vs. 42%) and the risk of graft loss of 3% [8].

The University Hospital of Liège initiated a program of controlled DCD-KT in 2005 [11]. This study was aimed at evaluating results of DCD-KT at our institute with regard to short- and mid-term graft function, graft and patient survival, rejection and surgical complications. The influence of DGF on graft function and survival as well as the potential DGF risk factors were also analyzed as secondary end-points.

Patients and methods

This study is a retrospective review of the experience of the Department of Abdominal Surgery and Transplantation at the University Hospital of Liège with controlled DCD-KT from 2005 to 2010. Kidneys procured from DCD donors were distributed within the Eurotransplant organization according to the same allocation rules as DBD kidneys (except Germany and Croatia where organ procurement and transplantation activity from DCD are prohibited by Law). The rate of local, national, and international sharing was 47.5%, 44.1%, and 8.5%, respectively, in this series. The acceptance criteria for DCD kidneys were as follows: donor age less than 65 years; no history of renal disease, uncontrolled hypertension, complicated diabetes mellitus, systemic sepsis or malignancy; warm ischemia time (WIT) less than 45 min (from cardio-circulatory arrest to aortic cold perfusion) or less than 60 min (from withdrawal of life-support to aortic cold perfusion) [12] and terminal serum creatinine <20 mg/l. Donor characteristics are presented in Table 1.

Withdrawal of life support occurred in the operating room. Heparine was injected intravenously prior to withdrawal of both ventilator and cardiac support in most DCD donors. Vital signs (blood pressure, heart rate, respiratory rate, and trans-cutaneous oxygen saturation) were monitored after discontinuation of treatment until cardio-circulatory arrest took place. Cardio-circulatory arrest was defined by femoral mean arterial pressure less than 30 mmHg without arterial pulse. A 5 min no-touch period was respected after cardio-circulatory arrest, then cardio-circulatory death was declared. Rapid laparotomy with direct aortic cannulation technique was utilized to *in situ* perfuse organs. HTK was the most common used preservation solution (84.7%) and kidneys were cold-stored in most cases (83.1%). Ten kidney allografts were preserved by the hypothermic machine perfusion (HMP) technique in the context of a Eurotransplant randomized controlled trial about the efficacy of HMP over static cold storage (SCS) [13]. Mean total WIT was 20.1 ± 7.2 min (range: 8–39). This time period comprised the withdrawal phase (from treatment discontinuation to cardio-circulatory arrest, mean: 9.4 ± 5.5 min, range: 2–30) and the acirculatory phase (from cardio-circulatory arrest to initiation of aortic cold perfusion, mean: 10.6 ± 4.8 min, range: 5–27). Mean cold ischemia time (CIT), defined as the time interval from aortic cold perfusion until removal of the kidney graft out of the cold preservation solution for implantation, was 731.3 ± 267.5 min (range: 207–1255). Mean vascular anastomosis suture time was 35.1 ± 9.7 min (range: 18–60).

Table 1. Donor characteristics.

Donor characteristics	Mean ± SD or n (%)	Range
Age (years)	45 ± 12.9	3–68
Gender		
Male	35 (59.3)	
Female	24 (40.7)	
BMI (kg/m^2)	25.4 ± 3.2	20–31.4
Hypertension		
Yes	9 (15.3)	
No	38 (64.4)	
Unknown	12 (20.3)	
Diabetes		
Yes	2 (3.4)	
No	43 (72.9)	
Unknown	14 (23.7)	
Donor cause of death		
Head trauma	16 (27.1)	
Cerebral vascular accident	22 (37.3)	
Anoxia	19 (32.2)	
Euthanasia	2 (3.4)	
Length of stay in ICU (days)	7.1 ± 6.5	0–24*
Terminal serum creatinine (mg/l)	7.5 ± 3.1	2.3–17.2
24 h diuresis (ml)	2841.6 ± 1312.2	1270–5940
Last hour diuresis prior to procurement (ml)	144.2 ± 125.3	10–600

*Euthanasia donors did not stay in the ICU.
BMI, body mass index; ICU, intensive care unit.

Recipient variables are summarized in Table 2. Mean recipient age was 54.9 ± 13.5 years (range: 21–76). Recipients older than 65 years received kidneys from older donors in the context of Eurotransplant Senior Program [14]. Mean panel reactive antibodies (PRA) at transplant was 5.2% ± 15.2% (range: 0–75). Mean number of HLA (human leukocyte antigens) mismatches was 2.8 ± 1.0 (range: 0–4). The frequency of 0, 1, 2, 3, and 4 HLA mismatches was 1.7%, 8.5%, 28.8%, 32.2%, and 28.8%, respectively. Ureteral double J catheter was utilized in half of the patients (49.2%), largely depending on the surgeon's preference and experience. All recipients received induction therapy with anti-CD25 monoclonal antibody (basiliximab) and a standard triple therapy with tacrolimus or cyclosporin, mycophenolate mofetil or mycophenolic acid and steroids. Anti-infective prophylaxis comprised sulfamethoxazole/trimethoprim for pneumocystis and urinary tract infection for at least 6–12 months, valganciclovir for cytomegalovirus (CMV) depending on donor and recipient CMV serologic status (if D+/R−: valganciclovir for 3 months, other cases: acyclovir for herpes virus for 3 months). Diagnosis of renal allograft rejection was suggested by an unexplained rise in serum creatinine level of >0.3 mg/dl or a 25% increase from baseline level and confirmed by ultrasound-guided per-cutaneous

Table 2. Recipient characteristics.

Recipient characteristics	Mean ± SD or *n* (%)	Range
Age (years)	54.9 ± 13.5	21–76
Gender		
Male	37 (62.7)	
Female	22 (37.3)	
BMI (kg/m^2)	26.8 ± 5.3	15.9–38.2
ESRD etiology		
Primary glomerulo-nephritis	8 (13.6)	
Hypertension	7 (11.9)	
Diabetes	7 (11.9)	
Lupus	2 (3.4)	
Tubulo-interstitial nephropathy	4 (6.8)	
HIV nephropathy	1 (1.7)	
Hemolytic uremic syndrome	1 (1.7)	
Hepato-renal polycystosis	12 (20.3)	
Uropathy	5 (8.5)	
Unknown causes	12 (20.3)	
Time on waiting list (days)	535.7 ± 498.5	3–2160
Duration of pretransplant dialysis (days)	933.2 ± 617.1	0–2425*
Residual diuresis (ml)	650.4 ± 748.9	0–2520
Previous transplants		
First transplant	55 (93.2)	
Re-transplant	4 (6.8)	
Peak PRA (%)	11.5 ± 18.7	0–70
PRA at transplant (%)	5.2 ± 15.2	0–75
Number of HLA mismatches		
A locus	0.8 ± 0.7	0–2
B locus	1.1 ± 0.4	0–2
DR locus	0.8 ± 0.4	0–2

*One pre-emptive kidney transplant in the context of combined liver-kidney transplantation.

BMI, body mass index; ESRD, end-stage renal disease; PRA, panel reactive antibody; HLA, human leukocyte antigens; HIV, human immunodeficiency virus.

biopsy. Renal biopsy was also routinely done for all grafts at 3 months post-transplant for the purpose of deciding to withdraw steroids or not. Given the importance of subclinical rejection as a risk factor for interstitial fibrosis and tubular atrophy as well as worse glomerular filtration rate (GFR) and graft survival [15], they were all treated with bolus of steroids. Donor specific HLA antibody was checked periodically at the hospital discharge, 3 months and every year post-transplant, simultaneously at the time of graft biopsy and after a sensitizing event. Doppler ultrasound was systemically done at hospital discharge, 3 months and every year post-transplant or at any change of renal allograft function without clear explanation.

The renal transplant was primary transplant in most cases (93.2%) with one combined liver–kidney transplantation. There were four re-transplant recipients (6.8%), of whom, one was immunized with peak PRA of 61% while the remaining three had no panel reactive antibodies. No patients developed donor specific antibodies that were routinely screened by single antigen Luminex technique. The average number of HLA mismatches was 2.2 ± 1.5 (range: 1–4). Cross-match tests were performed at the procurement center with the recipient's historic sera and repeated again at the transplant center with a recent serum and these tests must be negative prior to graft implantation. For primary transplant recipients who were at low immunological risk, KT was allowed before the result of cross-match test to shorten the CIT.

Primary endpoints of the study were PNF, DGF, graft function at the hospital discharge, 3 months, 1, and 3 years post-transplant, graft and patient survival at 3 months, 1, and 3 years post-transplant. PNF was defined as inadequate renal function after transplantation that necessitates continuation of dialysis, excluding operative technical problems. DGF was defined as the requirement for haemodialysis during the first week post-transplant, with subsequent recovery of renal function, except dialysis treatments to correct hyper-kalemia or volume overload [16]. Graft function was estimated via serum creatinine and GFR according to the abbreviated Modification of Diet in Renal Disease equation [17,18]. Secondary endpoints of the study were the potential risk factors for DGF, the effect of DGF on graft and patient survival, duration of post-transplant haemodialysis, length of patient's hospital stay, acute rejection rate within the first 3 months post-transplant and the occurrence of vascular or urological complications. Acute rejection was diagnosed on the base of the initiation of anti-rejection treatment or renal biopsy result.

Statistical analysis was as follows: continuous variables were presented as mean ± standard deviation (SD) and categorical variables as percentage. Differences between groups were evaluated by nonparametric Mann–Whitney U/Wilcoxon Ranked Sum tests for continuous variables and Fisher's exact test or Chi square test for categorical variables. Survival rates were estimated by the Kaplan–Meier method and compared by the log rank test with graft failure and patient death as events. Multivariate logistic regression analysis was used to identify potential risk factors for DGF. All tests were two-tailed and *P*-values <0.05 were considered as significant. All analyses were performed using the SPSS statistical software, version 11.0 for PC Windows.

Results

During the 6-year period, there were 59 and 215 renal transplants from controlled DCD and DBD donors, respectively. In other words, DCD kidneys made up 21.5% of the DD kidney pool and helped to increase the

activity of KT up to 27.4% without impairing the DBD kidney source. The organ procurement and transplantation activity of the KT program at the University Hospital of Liège from 2005 to 2010 is presented in Fig. 1.

Functional and survival data

Analysis of Kaplan–Meier survival curves showed overall and death-censored graft survival rates were 96.6% and 96.6% at 3 months, 94.6% and 96.6% at 1 year, 90.7% and 92.6% at 3 years, and 84.6% and 92.6% at 4 years, respectively (Fig. 2). Five renal grafts were lost during the post-transplant follow-up, one because of renal vein thrombosis, one secondary to the relapse of HIV infection in the allograft and three others because of patient deaths. Mean follow-up of patients was 26.5 months (range: 0.5–62 months). Patient survival rates at 3 months, 1, 3, and 4 years were 98.3%, 96.3%, 96.3%, and 90.3%, respectively (Fig. 3). Three patients (5.1%) died during follow-up, one because of acute myocardial infarction 24 h postoperatively and other two because of broncho-pneumonitis caused by CMV and Aspergillus infection at 5 and 41 months.

No PNF grafts were observed in this series. Two recipients were excluded from the analysis of DGF rates, because one died 24 h post-transplant and it remain unknown whether the graft was functioning at the time of patient death, the other lost the kidney graft because of renal vein thrombosis. Twenty-six of 57 patients (45.6%) experienced DGF. The occurrence of DGF did not adversely influence graft survival, as overall graft survival rates were 100%, 95%, 95%, and 83.1% for patients with DGF compared with 100%, 100%, 91.7%, and 91.7% for patients without DGF at 3 months, 1, 3, and 4 years, respectively ($P = 0.52$, Fig. 4). In addition, DGF did not increase the risk of acute rejection or surgical complications: among 26 recipients with DGF, 8 (30.7%) developed acute rejection compared with 8 (25.8%) recipients without DGF ($P = 0.67$). The rate of all surgical complications was 34.6% and 25.8% in recipients with and without DGF, respectively ($P = 0.46$).

The use of HMP ($n = 10$) was associated with a non-statistically significant lower rate of DGF in comparison to that of SCS (30% versus 48.5%, respectively, $P = 0.31$). Likewise, donor age (≥60 years), donor terminal serum creatinine (≥15 mg/l), recipient age (≥60 years), recipient BMI (BMI ≥ 30), kidney allocation policy (national or international sharing), WIT (≥45 min), suture time (≥45 min) as well as CIT (≥18 h) had no apparent effect on the risk for DGF (P = NS, both in univariate and multivariate logistic regression analysis, Table 3). Donor body mass index (BMI), in contrast, had an impact on DGF in multivariate model (not in univariate analysis). Kidneys from donors with BMI ≥ 30 compared with ones with BMI < 30 was 17 times more likely to have DGF ($P = 0.03$).

One patient was transplanted because of HIV nephropathy and lost quite rapidly her renal allograft (29 months post-transplant) secondary to the relapse of HIV infection in the allograft. This was a rare indication of transplantation and this patient was excluded in the assessment of renal allograft function. Mean serum creatinine level at hospital discharge was 22.1 ± 11.7 mg/l (range: 6.8–56.6). The percentage of patients with serum creatinine level at hospital discharge <20, 20–40, and >40 mg/l was 61.1%, 25.9%, and 13%, respectively. Renal graft function continued to improve up to 3 months post-transplant and nearly stabilized over the following 4 years (Fig. 5). The mean GFR at hospital discharge, 3 months, 1, and 3 years was 37.1 ± 16.6, 50.7 ± 11.7, 50.9 ± 11.3, and 49.2 ± 11.2 ml/min, respectively. Among four recipients who underwent retransplantation, two developed DGF. However, the four kidney grafts functioned well during the study period.

Postoperative evolution and complications

The average number of haemodialysis post-transplant in case of DGF was 4.96 ± 6.01 sessions (range: 1–32). Mean duration of haemodialysis was 10.6 ± 17.1 days (median: 7, range: 1–90). Mean hospital stay was 17.8 ± 5.7 days

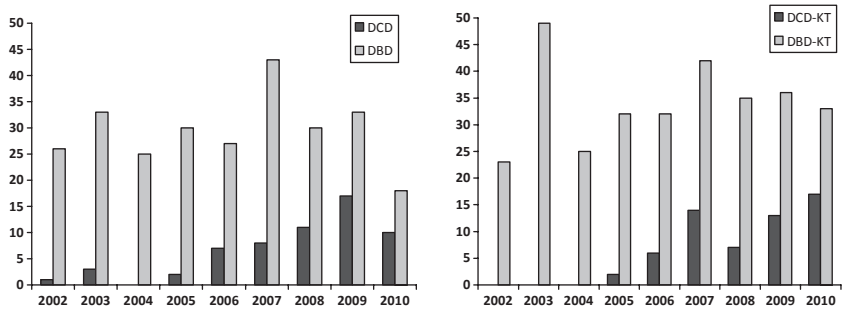

Figure 1 Organ donation and kidney transplantation activity in Liège over time. The number of DCD-KT increased without impairing the number of DBD-KT. DCD: donors after cardiac death. DBD: donors after brain death. KT: kidney transplants.

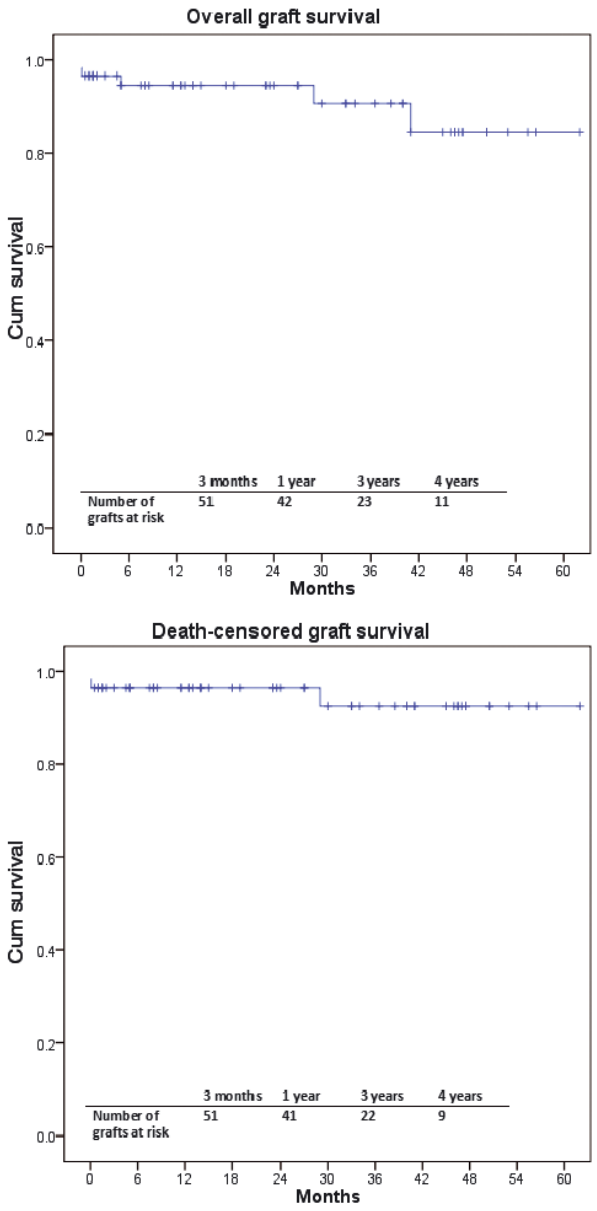

Figure 2 Overall and death-censored graft survival after DCD-KT ($n = 59$). Overall and actuarial graft survival rates were 96.6% and 96.6% at 3 months, 94.6% and 96.6% at 1 year, 90.7% and 92.6% at 3 years, and 84.6% and 92.6% at 4 years, respectively.

(range: 2–32). There was a significant difference in length of hospitalization between DGF and IGF (immediate graft function) groups (19.3 ± 5.3 vs. 13.4 ± 3.9 days, $P < 0.001$).

Sixteen of 59 patients (27.1%) experienced graft rejection during the first 3 months post-transplant, making up 17 rejection episodes. Rejection might be either clinically suspected without graft biopsy (10.1%) or biopsy-proven at the time of rejection suspicion (8.5%) or diagnosed only at 3 month protocol biopsy (8.5%).

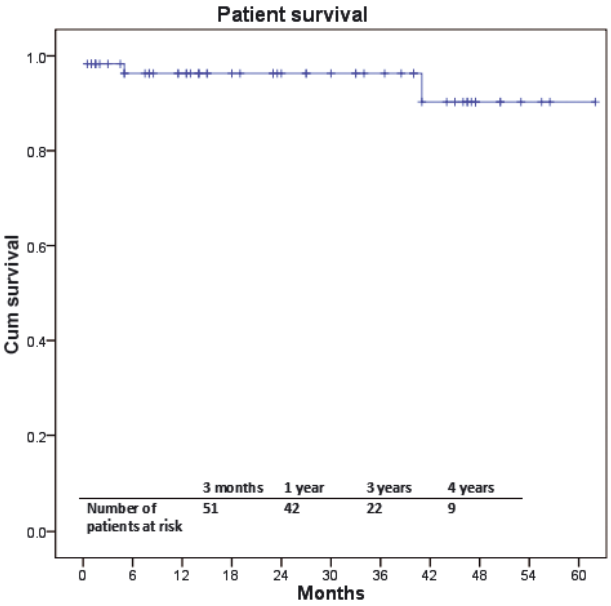

Figure 3 Overall patient survival after DCD-KT. Patient survival rates at 3 months, 1, 3, and 4 years were 98.3%, 96.3%, 96.3%, and 90.3%, respectively.

Early postoperative complications are presented in Table 4. After hospital discharge, renal artery stenosis was detected in two patients (3.4%) and stenting was necessary in one of them. Peripheral artery disease developed in two patients and all of them were stented at the level of iliac arteries. Infectious complications included pulmonary tuberculosis (one patient) and urinary tract infection (11 patients). Urologic exploration was performed in one patient because of repeated urinary infection, but no urinary anomaly was found. Peri-renal lymphocele occurred in one patient and was treated by puncture aspiration technique. One patient became pregnant 20 months post-transplant and gave birth of a healthy boy at 33rd amenorrheal week because of pre-eclampsia. No urinary leakage or ureteral obstruction was observed during the study period.

Discussion

This study showed excellent results of controlled DCD-KT, which were comparable to those from DBD in the literature although the use of DCD kidneys led to an elevated rate of DGF because of the unavoidable WIT between the withdrawal of life-support and the initiation of cold preservation. DGF increased significantly the length of hospitalization, nevertheless had no deleterious impact on post-transplant DCD kidney outcomes as demonstrated in several other studies [19,20]. A recent meta-analysis in studies with controlled DCD donors showed no difference in PNF rate between two groups of DBD

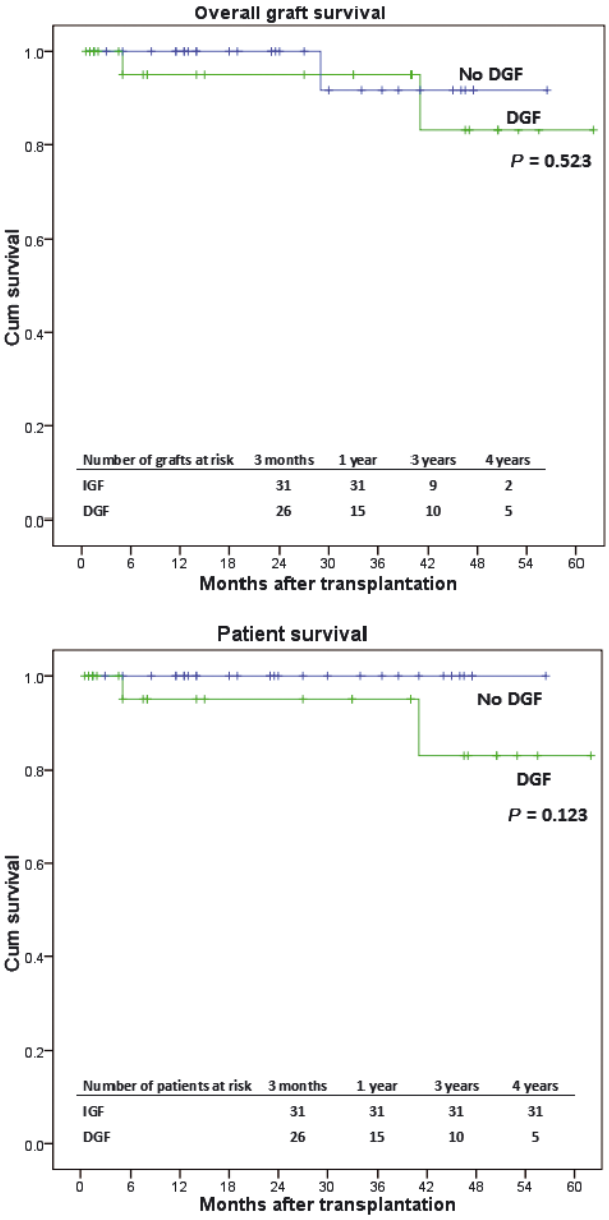

Figure 4 Graft and patient survival between DGF and no DGF groups. The presence of DGF did not adversely influence graft and patient survival (P = NS).

Table 3. Multivariate logistic regression analysis between the risk of DGF and different factors linked to the donor, recipient or transplantation procedure.

Factors	Odds ratio	95% CI	*P*-value
Donor age ≥50 years	0.902	0.235–3.465	0.881
Donor BMI ≥30	17.415	1.258–241.179	0.033
Donor serum creatinine ≥15 mg/l	0.000	0.000	1.000
Recipient age ≥60 years	3.249	0.776–13.610	0.107
Recipient BMI ≥30	3.505	0.872–14.088	0.077
Kidney allocation policy (national or international sharing)	0.801	0.221–2.907	0.736
WIT ≥30 min	1.982	0.239–16.457	0.527
Suture time ≥45 min	2.276	0.380–23.650	0.368
CIT ≥12 h	2.886	0.572–14.556	0.199
CIT ≥18 h	3.252	0.210–50.358	0.399
Preservation method (HMP)	0.462	0.058–3.647	0.463

DGF, delayed graft function; BMI, body mass index; WIT, warm ischemia time; CIT, cold ischemia time; HMP, hypothermic machine perfusion.

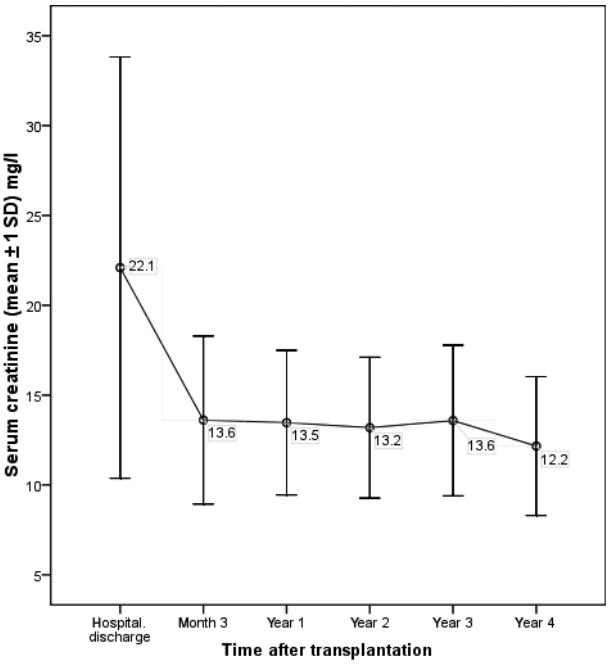

Figure 5 Sequential serum creatinine levels over time.

and DCD kidneys. The only significant difference was the DGF rate [21]. In our series, we did not experience any PNF and found a DGF rate of 45.6%. However, this high rate of DGF was not associated with an increased graft loss. When evaluating risk factors for DGF, only donor BMI ≥ 30 was significantly associated with an increased rate of DGF in multivariate logistic regression model. The significance of this finding remains unclear.

The DCD kidneys recovered their function slowly and in majority of cases failed to optimize their function at the time of hospital discharge. However, their function continued to improve and nearly normalized at 3 months post-transplant. Afterward renal allograft function stabilized over the following 4 years. By examining outcomes of DCD KTs that functioned for at least 1 year and had a follow-up of 2–5 years, Chapman found that the rate of graft loss at 5 years was similar between DCD and DBD

Table 4. Early postoperative complications.

Complications	*n*	Treatment
Renal vein thrombosis	1	Transplantectomy
Peri-graft hematoma	5	Conservative treatment (4 patients) Surgical re-intervention (1 patient)
Hematuria	5	Bladder irrigation
Hydronephrosis	2	Resolving spontaneously without urologic intervention
Abdominal wall bleeding	1	Surgical re-intervention
Rupture of drainage catheter	1	Surgical re-intervention
Urethral stenosis and BPH	3	Urethrotomy (1 patient), TURP (2 patients)
Acute myocardial infarction	2	Coronary artery stenting (1 patient death)
Cardiac rhythmic disorders	2	CPR (1 patient) Cardiac pace-maker placement (1 patient)
Anemia	11	Blood transfusion

BPH, benign prostatic hypertrophy; TURP, trans-urethral resection of prostate; CPR, cardio-pulmonary resuscitation.

grafts (approximately 3%) and both groups showed similar declines in GFR after 1 year (−1.3 ml/min for the DCD group vs. −1.4 ml/min for the DBD group). This means that DCD kidneys might have a reduced functioning glomerular mass because of the initial ischemic damage, but once transplanted there was no evidence of accelerate deterioration [22].

Graft survival rates in this study were favorably comparable to other reported series [1,4,23,24]. The major cause of graft loss was patient death with a functioning graft. Although DCD kidneys experienced worse early transplant outcomes than those coming from DBD donors, they did provide real survival benefit to patients [25]. Patients who were willing to accept a standard-criteria DCD kidney had a 56% reduction in mortality risk compared with those remaining on dialysis or awaiting a standard-criteria DBD kidney. This reduction in mortality translates into 2.4 months additional expected lifetime during the first 4 years after transplantation for recipients of DCD kidneys in comparison with patients who wait for a DBD kidney [26].

The rate of clinical and subclinical rejection in our study was similar to that reported in many studies, either single-center reports [4,27,28], national databases [2,29] or a recent meta-analysis [21]. DCD kidneys, despite experiencing greater DGF rates, do not display a greater incidence of acute allograft rejection episodes (10–19%) compared with DBD kidneys (9–18%). Similarly, in a recent publication, Saeb-Parsy did not find any difference in the rate of major urological complications (urinary leak and ureteral stenosis) between DCD and DBD kidney grafts (3.5% versus 1.7%, $P = 0.28$) [30]. Inversely, Droupy found that the risk of ureteral stenosis and fistula was significantly higher for DCD than DBD kidneys (15% vs. 7%, $P = 0.04$) [31]. In 76 controlled DCD-KT performed at Leiden University Medical Centre, Khairoun reported one urinary leakage because of ureteral necrosis and two ureteral obstructions (one after removal of the double J stent and the other because of blood clot) [32]. The rate of renal artery stenosis in this study was 3.4%. Although the incidence of transplant renal artery stenosis is expected to be higher in DCD kidneys because of the exposure to an excessive ischemic injury, many published series, as ours, also did not find any significant difference between DCD and DBD kidneys [33].

Estimates suggested that the potential increase in the number of DCD kidneys might be 2–4.5 times that of DBD kidneys [34]. However, in practice, single-center reports usually described a 20–40% proportion of DCD KTs among the DD kidney pool [1,24,35,36]. Exceptionally, a few transplant centers have obtained 50–70%, such as in Maastricht [37] or Madrid [38,39]. Recently several transplant centers in the Netherlands [40], the United Kingdom (UK) [41] and the United States (US) [42] have observed a remarkable increase in the number of DCD donors with a concomitant decrease in DBD donors, resulting in no significant change in the DD pool, some kind of redistribution of donor types within the pool. We have not yet observed such a trend in our experience.

No significant difference in the rate of DGF between ice-stored and machine-perfused DCD kidneys was noted in this study, although the DGF rate was lower among machine-perfused grafts. A recent multi-centric randomized controlled trial, in which 164 DCD kidney pairs were split and one allocated to each preservation modality, convincingly demonstrated that HMP produced less frequent and less severe DGF compared with SCS group (54% versus 70%) [13]. In a study design similar to Moers's study, Watson in the UK found no benefit of HMP over SCS for DCD kidneys. Nevertheless, the author emphasized on the ischemia time as an important factor for the differences between the two trials [43]. A meta-analysis undertaken by Wright [44] and studies in the US using the national database [3,45] all confirmed the advantage of HMP over SCS in DCD kidneys.

Conclusion

The use of controlled DCD kidneys might be an effective way to increase the number of kidneys available for transplantation because of good transplant outcomes and

acceptable postoperative complications. Despite a higher rate of DGF with longer hospitalization, DGF had no harmful effect on the graft future in this series. By using this donor source, transplant centers could help optimize the quality of life and minimize the mortality of end-stage kidney disease patients on the waiting list.

Authorship

HL: designed the study, collected and analyzed the data and wrote the article. LW, CB, and JMK: were responsible for the selection and follow-up of the patients reported in the study; they analyzed the data and corrected the manuscript. JM: collected the data. ADR, J-PS, MM, and OD: performed the procurements and transplantations reported in the study; they analyzed the data and corrected the manuscript. OD: supervised the study.

Funding

No funding was required for this study.

References

1. Akoh JA, Denton MD, Bradshaw SB, Rana TA, Walker MB. Early results of a controlled non-heart-beating kidney donor programme. *Nephrol Dial Transplant* 2009; **24**: 1992.
2. Doshi MD, Hunsicker LG. Short- and long-term outcomes with the use of kidneys and livers donated after cardiac death. *Am J Transplant* 2007; **7**: 122.
3. Locke JE, Segev DL, Warren DS, Dominici F, Simpkins CE, Montgomery RA. Outcomes of kidneys from donors after cardiac death: implications for allocation and preservation. *Am J Transplant* 2007; **7**: 1797.
4. Sudhindran S, Pettigrew GJ, Drain A, *et al.* Outcome of transplantation using kidneys from controlled (Maastricht category 3) non-heart-beating donors. *Clin Transplant* 2003; **17**: 93.
5. Ledinh H, Bonvoisin C, Weekers L, *et al.* Results of kidney transplantation from donors after cardiac death. *Transplant Proc* 2010; **42**: 2407.
6. Moers C, Leuvenink HG, Ploeg RJ. Donation after cardiac death: evaluation of revisiting an important donor source. *Nephrol Dial Transplant* 2010; **25**: 666.
7. Snoeijs MG, Winkens B, Heemskerk MB, *et al.* Kidney transplantation from donors after cardiac death: a 25-year experience. *Transplantation* 2010; **90**: 1106.
8. Squifflet JP. Why did it take so long to start a non-heart-beating donor program in Belgium? *Acta Chir Belg* 2006; **106**: 485.
9. Van Gelder F, Delbouille MH, Vandervennet M, *et al.* An 11-Year overview of the Belgian donor and transplant statistics based on a consecutive yearly data follow-up and comparing two periods: 1997 to 2005 versus 2006 to 2007. *Transplant Proc* 2009; **41**: 569.
10. Van Gelder F, Delbouille MH, Vandervennet M, *et al.* Overview of the Belgian donor and transplant statistics 2006: results of consecutive yearly data follow-up by the Belgian Section of Transplant Coordinators. *Transplant Proc* 2007; **39**: 2637.
11. Ledinh H, Meurisse N, Delbouille MH, *et al.* Contribution of donors after cardiac death to the deceased donor pool: 2002 to 2009 University of Liege experience. *Transplant Proc* 2010; **42**: 4369.
12. Bernat JL, D'Alessandro AM, Port FK, *et al.* Report of a National Conference on Donation after cardiac death. *Am J Transplant* 2006; **6**: 281.
13. Moers C, Smits JM, Maathuis MH, *et al.* Machine perfusion or cold storage in deceased-donor kidney transplantation. *N Engl J Med* 2009; **360**: 7.
14. Smits JM, Persijn GG, Van Houwelingen HC, Claas FH, Frei U. Evaluation of the Eurotransplant Senior Program The results of the first year. *Am J Transplant* 2002; **2**: 664.
15. El-Amm JM, Gruber SA. The significance of subclinical rejection. *Clin Transplant* 2009; **23**: 150.
16. Yarlagadda SG, Coca SG, Garg AX, *et al.* Marked variation in the definition and diagnosis of delayed graft function: a systematic review. *Nephrol Dial Transplant* 2008; **23**: 2995.
17. Houillier P, Froissart M. Elevated serum creatinine. *Rev Prat* 2005; **55**: 91.
18. Flamant M, Boulanger H, Azar H, Vrtovsnik F. Plasma creatinine, Cockcroft and MDRD: validity and limitations for evaluation of renal function in chronic kidney disease. *Presse Med* 2010; **39**: 303.
19. Brook NR, White SA, Waller JR, Veitch PS, Nicholson ML. Non-heart beating donor kidneys with delayed graft function have superior graft survival compared with conventional heart-beating donor kidneys that develop delayed graft function. *Am J Transplant* 2003; **3**: 614.
20. Renkens JJ, Rouflart MM, Christiaans MH, van den Berg-Loonen EM, van Hooff JP, van Heurn LW. Outcome of nonheart-beating donor kidneys with prolonged delayed graft function after transplantation. *Am J Transplant* 2005; **5**: 2704.
21. Kokkinos C, Antcliffe D, Nanidis T, Darzi AW, Tekkis P, Papalois V. Outcome of kidney transplantation from non-heart-beating versus heart-beating cadaveric donors. *Transplantation* 2007; **83**: 1193.
22. Chapman J, Bock A, Dussol B, *et al.* Follow-up after renal transplantation with organs from donors after cardiac death. *Transpl Int* 2006; **19**: 715.
23. Keizer KM, de Fijter JW, Haase-Kromwijk BJ, Weimar W. Non-heart-beating donor kidneys in the Netherlands: allocation and outcome of transplantation. *Transplantation* 2005; **79**: 1195.
24. Brook NR, Waller JR, Richardson AC, *et al.* A report on the activity and clinical outcomes of renal non-heart

beating donor transplantation in the United Kingdom. *Clin Transplant* 2004; **18**: 627.
25. Ojo AO, Hanson JA, Meier-Kriesche H, *et al.* Survival in recipients of marginal cadaveric donor kidneys compared with other recipients and wait-listed transplant candidates. *J Am Soc Nephrol* 2001; **12**: 589.
26. Snoeijs MG, Schaubel DE, Hene R, *et al.* Kidneys from donors after cardiac death provide survival benefit. *J Am Soc Nephrol* 2010; **21**: 1015.
27. Chudzinski RE, Khwaja K, Teune P, *et al.* Successful DCD kidney transplantation using early corticosteroid withdrawal. *Am J Transplant* 2010; **10**: 115.
28. Barlow AD, Metcalfe MS, Johari Y, Elwell R, Veitch PS, Nicholson ML. Case-matched comparison of long-term results of non-heart beating and heart-beating donor renal transplants. *Br J Surg* 2009; **96**: 685.
29. Rudich SM, Kaplan B, Magee JC, *et al.* Renal transplantations performed using non-heart-beating organ donors: going back to the future? *Transplantation* 2002; **74**: 1715.
30. Saeb-Parsy K, Kosmoliaptsis V, Sharples LD, *et al.* Donor type does not influence the incidence of major urologic complications after kidney transplantation. *Transplantation* 2010; **90**: 1085.
31. Droupy S, Blanchet P, Eschwege P, *et al.* Long-term results of renal transplantation using kidneys harvested from non-heartbeating donors: a 15-year experience. *J Urol* 2003; **169**: 28.
32. Khairoun M, Baranski AG, van der Boog PJ, Haasnoot A, Mallat MJ, Marang-van de Mheen PJ. Urological complications and their impact on survival after kidney transplantation from deceased cardiac death donors. *Transpl Int* 2009; **22**: 192.
33. Nicholson ML, Metcalf MS, White SA, *et al.* Comparison of the results of renal transplantation from non-heartbeating, conventional, cadaveric and living donors. *Kidney Int* 2000; **58**: 2585.
34. Kootstra G. The asystolic, or non-heartbeating, donor. *Transplantation* 1997; **63**: 917.
35. Wells AC, Rushworth L, Thiru S, *et al.* Donor kidney disease and transplant outcome for kidneys donated after cardiac death. *Br J Surg* 2009; **96**: 299.
36. D'Alessandro AM, Fernandez LA, Chin LT, *et al.* Donation after cardiac death: the University of Wisconsin experience. *Ann Transplant* 2004; **9**: 68.
37. Kootstra G, van Heurn E. Non-heartbeating donation of kidneys for transplantation. *Nat Clin Pract Nephrol* 2007; **3**: 154.
38. Sanchez-Fructuoso AI, Marques M, Prats D, *et al.* Victims of cardiac arrest occurring outside the hospital: a source of transplantable kidneys. *Ann Intern Med* 2006; **145**: 157.
39. Sanchez-Fructuoso AI, Giorgi M, Barrientos A. Kidney transplantation from non–heart-beating donors: a Spanish view. *Transplant Rev* 2007; **21**: 249.
40. Cohen B, Smits JM, Haase B, Persijn G, Vanrenterghem Y, Frei U. Expanding the donor pool to increase renal transplantation. *Nephrol Dial Transplant* 2005; **20**: 34.
41. Brook NR, Nicholson ML. Kidney transplantation from non heart-beating donors. *Surgeon* 2003; **1**: 311.
42. Saidi RF, Bradley J, Greer D, *et al.* Changing pattern of organ donation at a single center: are potential brain dead donors being lost to donation after cardiac death? *Am J Transplant* 2010; **10**: 2536.
43. Watson CJE, Wells AC, Roberts RJ, *et al.* Cold machine perfusion versus static cold storage of kidneys donated after cardiac death: a UK multicenter randomized controlled trial. *Am J Transplant* 2010; **10**: 1991.
44. Wight JP, Chilcott JB, Holmes MW, Brewer N. Pulsatile machine perfusion vs. cold storage of kidneys for transplantation: a rapid and systematic review. *Clin Transplant* 2003; **17**: 293.
45. Schold JD, Kaplan B, Howard RJ, Reed AI, Foley DP, Meier-Kriesche HU. Are we frozen in time? Analysis of the utilization and efficacy of pulsatile perfusion in renal transplantation. *Am J Transplant* 2005; **5**: 1681.