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Journal

Clinical Transplantation, 30(9)

ISSN

0902-0063

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Publication Date

2016-09-01

DOI

10.1111/ctr.12800

Peer reviewed

HHS Public Access

Author manuscript

Clin Transplant. Author manuscript; available in PMC 2017 February 01.

Published in final edited form as:

Clin Transplant. 2016 September ; 30(9): 1140–1145. doi:10.1111/ctr.12800.

Key donor factors associated with graft loss among liver transplant recipients with human immunodeficiency virus

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Abstract

Background—Human immunodeficiency virus (HIV)-infected liver transplant (LT) recipients have higher risk of graft loss than HIV-uninfected recipients. As the original donor risk index excluded HIV-positive patients, donor factors associated with graft loss in HIV-positive recipients are unknown.

Methods—Identifying all HIV-positive patients in the Scientific Registry of Transplant Recipients, supplemented by all HIV-infected patients in the solid organ transplantation in HIV: Multi-Site Study (HIV-TR), we evaluated donor factors associated with graft loss among HIV-positive recipients transplanted between March 2002 and August 2012.

Results—A total of 249 HIV-positive LT recipients were followed for median 2.4 (interquartile range [IQR]: 0.8–4.9) years. In univariate analysis, donor diabetes (HR=2.09; *P*=.002) and donor hypertension (HR=1.43; *P*=.048) were significantly associated with graft loss, and African-American (AA) recipient:non-AA donor race mismatch (HR=1.60; *P*=.07), other cause of donor death compared to trauma (HR=2.02; *P*=.09), and donor age 30 years or older (HR=1.53; *P*=.05) were of borderline significance. In multivariate analysis, donor diabetes (HR=2.12; 95% CI: 1.33–3.38; *P*=.002) was the only significant predictor of graft loss.

Conclusion—In HIV-positive LT recipients, risk of graft loss is strongly influenced by donor diabetes. This information may be useful to transplant physicians seeking to optimize overall graft survival in their HIV-positive LT recipients.

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CONFLICT OF INTEREST

NAT: Institutional Grant support: Gilead, Eisai, Biotest, AbbVie, and P30 DK026743 (UCSF Liver Center). Consulting: BMS, Merck, Pfizer, Achillion, Cocrystal Pharma.

AUTHORS' C ONTRIBUTIONS

Isabel Campos-Varela: The primary author of the article, participated in the design of the study and data analysis; Jennifer L. Dodge: The primary data analyst for the study and participated in editing the article and designing the study; Peter G. Stock: Participated in writing and editing the article and designing the study; Norah A. Terrault participated in study design, data analysis, and writing/editing the article.

Keywords

diabetes; donor age; donor risk index; race mismatch; survival

1 | INTRODUCTION

For patients infected with human immunodeficiency virus (HIV) who have complications of cirrhosis including small hepatocellular carcinoma (HCC), liver transplantation (LT) can be the only life-saving treatment option.[1] Overall survival rates are comparable to those achieved with older LT recipients without HIV infection, with 1-and 3-year liver graft survival rates of 82% and 64%, respectively.[2,3] However, the 3-and 5-year patient survival rates of 62% and 54% are lower among HIV–HCV-coinfected LT recipients compared to their HCV-positive, HIV-uninfected counterparts.[4–6]

Identifying means of improving outcomes is of critical importance. To date, efforts to optimize outcomes have focused on patient selection and better management of post-transplantation immunosuppression. Acute rejection has been reported to occur at a significantly higher rate in HIV–HCV-coinfected patients than HCV-infected patients without HIV, potentially due to overly cautious use of the immunosuppressant drugs in the early post-transplant period or difficulties in achieving adequate immunosuppression due to drug–drug interactions.[4,5] Recipient factors associated with poorer outcomes post-LT identified in prior studies include HCV genotype 1, higher Model for End-Stage Liver Disease (MELD) at LT, transplant center experience, lower body mass index (BMI), and combined kidney–liver transplantation.[4,5,7–9] However, these studies were limited to HIV-infected patients with HCV coinfection.

The importance of donor factors in post-transplant graft and patient survival was first highlighted in 2006 with the development of the donor risk index (DRI).[10] Seven donor factors were identified as predictive of post-LT graft failure. While DRI has been crucial in highlighting the impact of donor factors in post-LT outcomes, some patients subgroups, notably HIV-infected patients, were excluded. Several studies have shown that DRI may have limited predictive accuracy among subgroups, such those transplanted by HCV infection, or African Americans (AA).[11,12] Regarding HIV infection, the donor factors suggested as being of potential importance in graft survival in HIV–HCV-coinfected patients were older donor age and receipt of an organ from a HCV-positive donor but no prior study has examined the impact of donor factors on graft survival among all HIV-positive patients.[4,5] Thus, we aimed to evaluate the donor factors having the greatest effect on post-transplant outcomes in HIV-positive liver transplant recipients.

2 | MATERIALS AND METHODS

HIV-positive transplant recipients were identified from two sources. The primary source was all HIV-positive patients in the Scientific Registry of Transplant Recipients (SRTR), as submitted by members of the Organ Procurement and Transplantation Network. This was supplemented with all HIV-infected patients in the solid organ transplantation in HIV: Multi-Site Study (HIV-TR), the largest US multicenter study of transplantation in HIV.[4] Of note,

55 (22%) patients known to have HIV infection based on enrollment in the HIV-TR study were not identified as HIV positive in SRTR. These patients were instead classified as negative (11) undetermined (4), unknown (36), or missing (4) regarding their HIV serostatus. By combining patients from both databases, we were able to identify and include a maximum number of HIV-infected patients in our study. All adult HIV-infected recipients of deceased donor liver transplants from March 1, 2002 to August 31, 2012 (MELD-era) were included. Patients with acute liver failure (status 1), multiple organ transplantation (other than liver–kidney), or retransplantation were excluded. Additionally, due to small numbers limiting evaluation, partial liver transplants (four splits and two living donors) were excluded. Data for donor, recipient, transplant variables, and outcomes were obtained from the SRTR.

Recipient and donor factors were described with frequency distributions and medians with interquartile ranges (IQRs). Donor covariates were evaluated as our primary predictor of interest and included age, sex, sex match, race, race match, height, weight, BMI, antihypertensive, and vasodilators within 24 hours of donor aortic cross-clamp, HCV antibody status, inotropic support, diabetes, history of hypertension, creatinine, aspartate aminotransferase, bilirubin, donation after cardiac death, cause of death (COD), Centers for Disease Control (CDC) high-risk donors, cold ischemic time (CIT), and regional and national organ sharing and center. We imputed missing CIT (4% of patients) and out-of-range CIT (defined as CIT less than 2 hours or greater than 20 hours (1% of patients) with the median CIT value for the patient's UNOS transplant region by organ share type (local, regional, or national organ donation). Center experience with transplantation of HIV-positive patients (categorized as <1 or ≥1 HIV-positive transplant per year) and transplant year was evaluated as covariates. Thirty years of age was the donor age cutoff that offered the strongest association with outcomes, compared to 40, 50 and 60 years of age, and corrected issues with non-proportional hazards. To evaluate the independent association between donor factors and allograft outcomes, recipient covariates were evaluated and included for adjustment. Recipient factors considered were age, sex, ethnicity, height, weight, BMI, diabetes, life support at transplant, transplantation region, previous abdominal surgery, dialysis prior to transplantation, hospitalization at transplantation, portal vein thrombosis, HCV infection, HCC, simultaneous kidney transplantation, and laboratory values at transplantation (MELD, bilirubin, INR, albumin, sodium, glomerular filtration rate).

The primary outcome was post-transplant graft loss, defined as a recipient death or retransplantation. Time to graft loss was calculated as years from date of LT to the date of first retransplantation or death. Patients alive or lost to follow-up were censored at the date of last follow-up. The Kaplan-Meier method was used to estimate 1-and 3-year observed post-transplant graft survival, with 95% confidence intervals (95% CI) provided. The log-rank test compared survival estimates across categories of donor characteristics. We used Cox proportional hazards regression with Wald sandwich estimators (accounts for within-center clustering) to evaluate donor and recipient factors associated with risk of graft loss. Donor factors with a pre-specified statistical significance of $P<.1$ were analyzed by multivariable Cox regression. Backwards elimination with $P<.05$ was used to select the multivariable donor model. The final model was adjusted for significant recipient variables, although we carefully evaluated all biologically and statistically (univariable $P<.1$) plausible

recipient variables in the modeling process. The proportional hazards assumption was tested, and violations for donor age were resolved by dichotomizing at 30 years. Interactions between donor variables and recipient HCV were evaluated to determine the necessity of subgroup analyses among the HIV–HCV coinfected and HIV monoinfected. Statistical analyses were conducted using SAS v. 9.4 (Cary, NC, USA). This study was approved by the University of California San Francisco Institutional Review Board.

3 | RESULTS

3.1 | Recipient and donor characteristics

A total of 249 MELD-era HIV-positive LT recipients with a median of 2.4 (IQR: 0.8–4.9) years of follow-up were included in the analysis (Table 1). Recipients were 82% male and had median age of 50 (IQR: 45–55) years at LT, 21% were AA race, median MELD at transplantation was 19 (IQR: 13–28), 55% had HCV coinfection, and 38% had HCC. Donors were 57% male, with median age of 41 (IQR: 25–53) years, 20% were AA, and 8% were anti-HCV positive. The cause of donor death was anoxic, cardiovascular, and other in 20%, 38%, and 2%, respectively, and the median cold ischemia time was 6.5 (IQR: 6.0–7.0) hours. The median DRI was 1.28 (IQR: 1.05–1.57) (Table 2).

3.2 | Donor factors associated with graft survival

Overall, 116 patients experienced graft loss during post-transplant follow-up. Graft survival rates at 1 and 3 years for HIV-positive LT recipients were 74% (95% CI: 68–79) and 59% (95% CI: 52–65), respectively. Donor characteristics associated with graft loss in univariate analysis are shown in Table 2.

In univariate analysis, donor diabetes (HR=2.09; 95% CI: 1.31–3.34, P=.002) and history of hypertension (HR=1.43; 95% CI: 1.003–2.04, P=.048) were significantly associated with reduced graft survival. Increasing age was of borderline significance, with an increased risk of graft loss among HIV-infected recipients of donors ≥30 compared to <30 years of age (HR 1.53; 95% CI: 1.00–2.33, P=.05). The 3-and 5-year post-LT graft survival in recipients receiving a donor of 30 years of age or older was significantly lower than those receiving a donor younger than 30 years of age, 54% (95% CI: 46–62) vs 68% (95% CI: 56–78) (log-rank P=.047) and 45% (95% CI: 36–53) vs 64% (95% CI: 51–75) (log-rank P=.02), respectively (Fig. 1A). Donor diabetes was associated with a 3-and 5-year survival of 45% (95% CI: 21–66) and 27% (95% CI: 7–51) compared to 60% (95% CI: 53–66) and 53% (95% CI: 45–60) in donors without diabetes (log-rank P=.17 and P=.06, respectively) (Fig. 1B). AA recipient:non-AA donor race mismatch (vs recipient–donor both non-AA HR=1.60; P=.07) and other COD (vs trauma) (HR=2.02; P=.09) also met the criteria for evaluation in the multivariable analysis.

After adjusting for recipient dialysis, transplant year, and donor age, the only donor variable independently associated with risk of graft loss was donor diabetes (HR=2.12; 95% CI: 1.33–3.38; P=.002). Donor age of 30 years or older was no longer statistically significant (HR 1.40; 95% CI: 0.92–2.13, P=.12) (Table 3).

The cohort was comprised of 137 HIV–HCV-coinfected patients and 112 HIV-monoinfected patients. We evaluated potential interactions between donor factors and recipient HCV status. As no statistically significant interactions were identified and subgroup sample sizes were small, stratified models by HCV status were not developed.

4 | DISCUSSION

The original DRI excluded patients with HIV infection. When applied to HIV-positive population, the original DRI has a limited ability to predict risk of graft failure (*C*-index 0.56). We present the first evaluation of potential donor factors associated with post-LT outcomes in HIV-infected patients. The key donor factor associated with risk of graft failure was diabetes. This finding, encompassing the largest cohort of transplant recipients with HIV, may be helpful to transplant physicians in discussing different donor options with HIV-positive recipients.

The original DRI did not include donor diabetes as a risk factor. This donor variable, likely to be seen with increasing frequency among donors over time, increased the risk of graft loss twofold. Diabetes has been associated with non-alcoholic fatty liver disease and is suggested to be the main risk for chronic liver disease among patients with diabetes.[13,14] Diabetic microvascular complications are well described systemically, but have not been well addressed in the liver. A recent study focused on the association of diabetes with hyaline arteriolosclerosis in the liver.[15] The main feature was hyaline thickening of the hepatic arteriolar walls associated with concomitant sinusoidal fibrosis, pericentral fibrosis, and hyaline appearance of the portal tracts in several diabetic patients. This histological characteristic was present among 45% of the 89 patients with diabetes included in the study, compared to 29% of the controls (P=.298). Biliary changes were associated with hyaline arteriolosclerosis in a 10.6% of the cases. The implication of this small-vessel complication of the liver remains to be further elucidated, but small-vessel damage may be one factor leading to worse outcomes observed in LT with livers from diabetic donors. Of note, other studies have reported diabetes donor as a negative factor for post-LT outcomes in HCV-infected recipients.[16,17] Unfortunately, due to the characteristics of this large database, data regarding hyaline arteriolar changes were not recorded. However, this is an interesting feature for future studies using non-registry data.

Older donor age was associated with a 40% higher risk of graft loss in our HIV-infected cohort, but this failed to reach statistical significance. This association is consistent with other literature in non-HIV transplant recipients, and given the magnitude of the association, donor age was maintained in the final model despite not achieving statistic significance (possibly due to the small sample size). Donor age was the dominant predictor in both DRI and the AA DRI, limited to HCV-infected patients.[10,12] Prior studies in non-HIV-infected patients have shown that the risk of premature graft loss begins with donors older than 40 years for HCV-infected patients, but for HCV-negative recipients, the risk does not increases until the age of 60. Notably, in the HIV transplant cohort, the median donor ages were 39 and 44 years, respectively, among those with and without HCV coinfection. Thus, our cohort was likely limited in being able to tease out the contribution of donor age across the spectrum of HIV-infected patients. However, based on the broader transplant literature, we

believe older donor age remains an important donor risk factor for graft loss. The important remaining issue is what the specific cutoff for “older” donor should be in HIV-infected patients.

In previous studies, the receipt of an anti-HCV-positive donor, especially if an older anti-HCV-positive donor, was associated with an increase in the risk of graft loss; however, we cannot confirm this association.[4,18] The number of recipients receiving anti-HCV-positive donors was only 11.7%, potentially attenuating this association in the face of other donor variables such as donor diabetes.

In this cohort, median MELD at LT was 19, which was lower than the national median, and this fact was related to the proportion of patients with HCC exceptions, with a median laboratory MELD at LT of 11 (IQR 9–15). Laboratory MELD at LT was clearly higher for subjects without HCC exceptions, median 25 (IQR 18–33).

The availability of direct-acting antivirals (DAAs) for treatment of HCV is changing the landscape for transplant recipients, including those with HIV.[19] In this new era of therapeutics, the impact of recipient and donor variables on post-LT survival is likely to be diminished. However, the magnitude and rapidity of improvements remains to be seen, particularly given the difficulties with accessing new therapies in some countries. Moreover, it is unlikely that eradication of HCV by DAAs will eliminate the association of donor factors with graft outcomes. Also, it must be mentioned that for many centers, transplantation of HIV-infected patients, especially with HIV–HCV coinfection, is still regarded as high risk and LT is not universally offered. Hence, information from this study may provide centers with means of maximizing patient and graft survival until the benefits of new HCV therapies are fully established.

Although limitations are inherent in the use of registry data, the registry provides the opportunity to evaluate the largest possible cohort of HIV-infected LT recipients, a strength given this population accounts for a small proportion (0.3%) of all liver transplants in the United States.[20] A further strength of this study is the use of supplemental data from the HIV-TR cohort, a large prospective multicenter US study, allowing a maximal sample size to be reached. Nonetheless, we acknowledge the limitation of modest sample size, despite our best efforts. Ultimately, the smaller simple size may have limited our power to identify additional donor variables that may have a less strong association with graft loss. Furthermore, we did not have sufficient sample size to perform validation studies.

In summary, in HIV-positive LT recipients, donor diabetes is the key determinant of graft loss. This information may be used to guide donor choices for HIV recipients to optimize overall graft survival.

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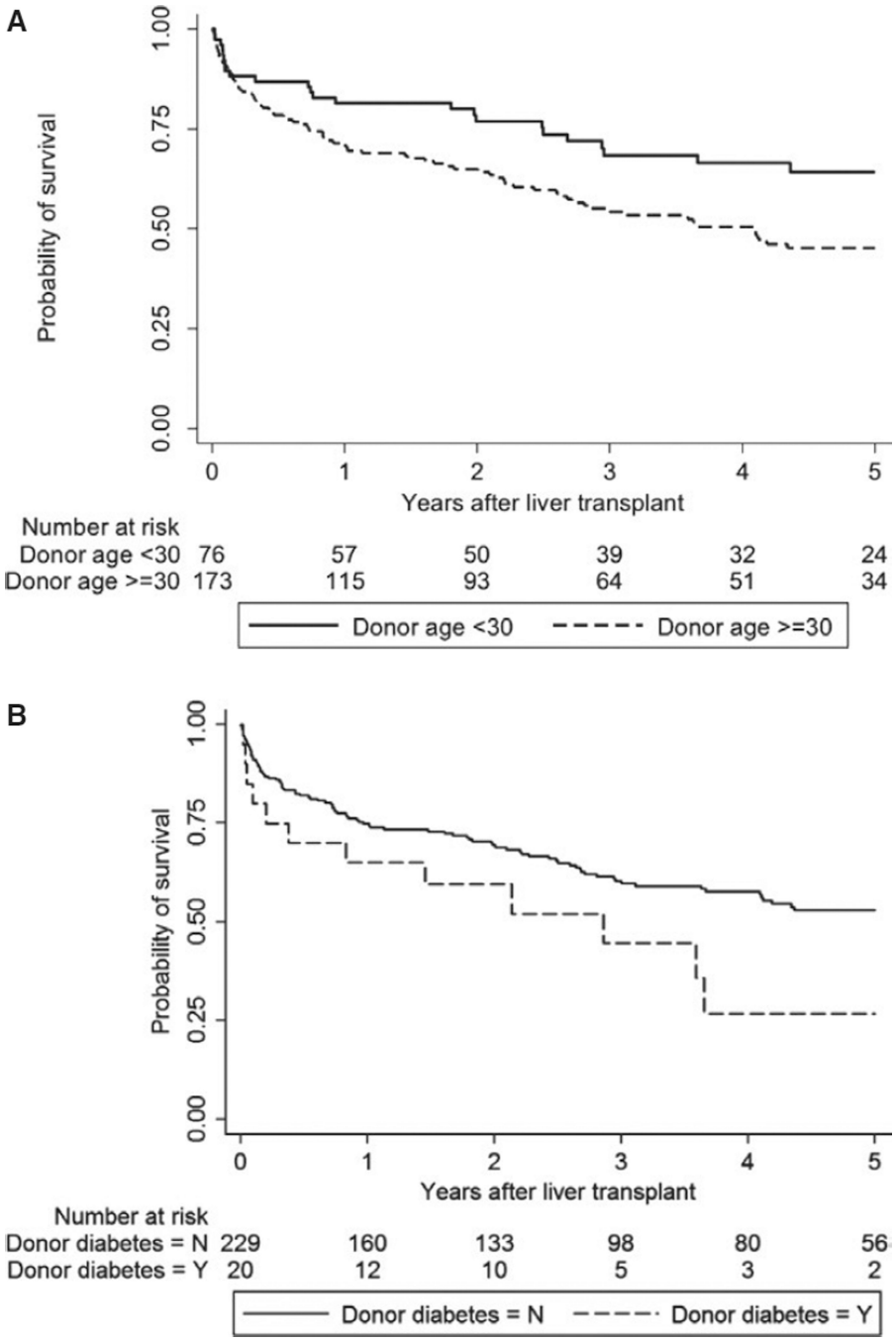

FIGURE 1.

(A) Unadjusted probability of graft survival by donor age in HIV-infected liver transplant recipients. The unadjusted probability of overall graft survival at 3-and 5-year post-LT was 54% (95% CI: 46–62) and 45% (95% CI: 36–53) for patients receiving an organ from a donor older than 30 years of age and 68% (95% CI: 56–78) and 64% (95% CI: 51–75) for patients receiving an organ from a donor younger than 30 years of age (log-rank *P*=.047 and *P*=.02, respectively). (B) Unadjusted probability of graft survival by donor diabetes in HIV-infected liver transplant recipients. The unadjusted probability of overall graft survival at 3-

and 5-year post-LT was 45% (95% CI: 21–66) and 27% (95% CI: 7–51) for patients receiving an organ from a diabetic donor and 60% (95% CI: 53–66) and 53% (95% CI: 45–60) for patients receiving an organ from a donor without diabetes (log-rank P=.17 and P=.06, respectively).

TABLE 1

Recipient characteristics and univariate association with graft loss

Recipient characteristic	Value	HR (95% CI)	*P*-value
Male, n (%)	205 (82)	1.02 (0.59–1.79)	.93
Age (y), median (IQR)	50 (45–55)	1.00 (0.97–1.02)[a]	.84
Height (cm), median (IQR)	175.3 (170.2–182.9)	0.99 (0.97–1.02)[b]	.58
BMI (kg/m^2), median (IQR)	24.9 (22.8–30.0)	1.00 (0.96–1.03)	.82
Diabetes, n (%)	43 (17)	1.09 (0.63–1.89)	.75
Bilirubin at transplant (ln), median (IQR)	3.7 (1.6–11.6)	1.00 (0.98–1.02)	.97
Albumin at transplant (g/dL), median (IQR)	3.0 (2.5–3.6)	0.92 (0.78–1.08)	.32
GFR (mL/min), median (IQR)	69 (42–93)	1.00 (0.99–1.00	.13
MELD at LT, median (IQR)	19 (13–28)	1.01 (0.99–1.02)	.57
HCV positive, n (%)	137 (55)	1.21 (0.89–1.64)	.23
Hepatocellular carcinoma, n (%)	95 (38)	0.90 (0.64–1.26)	.53
Simultaneous liver–kidney transplant, n (%)	16 (6)	1.51 (0.72–3.14)	.28
Life support at LT, n (%)	11 (4)	1.90 (0.86–4.17)	.11
Dialysis before LT, n (%)	19 (8)	2.41 (1.27–4.58)	.007
Previous abdominal surgery, n (%)	68 (27)	1.19 (0.75–1.88)	.47

BMI, body mass index; GFR, glomerular filtration rate; HCV, hepatitis C virus; LT, liver ransplantation; IQR, interquartile range; MELD, Model for End-Stage Liver Disease.

[a]Per 1 year increase.

[b]Per 10 cm increase.

TABLE 2

Donor and transplant characteristics and univariate association with graft loss

Donor characteristic	Value	HR (95% CI)	*P*-value
Male, n (%)	141 (57)	0.91 (0.67–1.24)	.55
Age (y), median (IQR)	41 (25–53)	1.01 (1.00–1.03)[a]	.049
Age ≥30 y, n (%)	173 (70)	1.53 (1.00–2.33)	.05
Non-AA donor:non-AA recipient, n (%)	161 (65)	Ref	
Non-AA donor:AA recipient	37 (15)	1.60 (0.96–2.66)	.07
AA donor:non-AA recipient	36 (14)	1.26 (0.63–2.54)	.51
AA donor:AA recipient	15 (6)	1.20 (0.68–2.12)	.53
Height (cm), median (IQR)	170 (165–178)	1.11 (0.93–1.33)[b]	.24
BMI (kg/m^2), median (IQR)	25.8 (22.8–30.0)	1.00 (0.97–1.04)	.91
Diabetes, n (%)	20 (8)	2.09 (1.31–3.34)	.002
History of hypertension, n (%)	83 (33)	1.43 (1.00–2.04)	.048
HCV positive, n (%)	20 (8)	1.40 (0.72–2.74)	.32
COD Trauma, n (%)	98 (40)	Ref	
COD Anoxic	51 (20)	0.85 (0.51–1.42)	.53
COD Stroke	94 (38)	1.45 (0.91–2.31)	.12
COD Other	6 (2)	2.02 (0.90–4.54)	.09
Bilirubin at transplant (ln), median (IQR)	0.7 (0.4–1.2)	0.97 (0.89–1.06)	.56
Creatinine at transplant (mg/dL), median (IQR)	1.1 (0.8–1.9)	1.00 (0.92–1.08)	.95
AST U/L, median (IQR)	47 (25–87)	1.00 (1.00–1.00)	.97
Cold ischemic time (per hour >8), median (IQR)	6.5 (6.0–7.0)	0.96 (0.80–1.16)	.67
CDC high risk, n (%)	31 (12)	0.67 (0.33–1.37)	.27
Donation after cardiac death, n (%)	14 (6)	1.03 (0.48–2.23)	.93
Regional sharing (vs Local), n (%)	65 (26)	1.05 (0.71–1.56)	.80
National sharing (vs Local)	19 (8)	0.89 (0.49–1.64)	.71
Vasodilators, n (%)	31 (12)	0.88 (0.54–1.44)	.61
Year of transplantation, median (IQR)	2007 (2006–2010)	0.93 (0.87–0.99)	.03
Center with <1 LT in HIV-infected patients/y, n (%)	91 (36)	0.97 (0.63–1.49)	.88
Donor Risk Index,	1.28 (1.05–1.57)	1.77 (1.09–2.88)	.02

Donor characteristic	Value	HR (95% CI)	*P*-value
median (IQR)			

AA, African-American; AST, aspartate aminotransferase; COD, cause of death; CDC, Centers for Disease Control; LT, liver transplantation; IQR, interquartile range.

[a] Per 1 year increase.

[b] Per 10 cm increase.

TABLE 3

Adjusted independent variables included in model predicting risk of graft loss

	HR	95% CI	*P*-value
Donor diabetes	2.12	1.33–3.38	.001
Donor age >30	1.40	0.92–2.13	.12
Transplant year	0.91	0.84–0.99	.02
Recipient dialysis	2.71	1.46–5.03	.001

[a] Adjusted for center effect.