Impact of Donor Liver Macrovesicular Steatosis on Deceased Donor Yield and Posttransplant Outcome

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Background. The Scientific Registry of Transplant Recipients (SRTR) had not traditionally considered biopsy results in risk-adjustment models, yet biopsy results may influence outcomes and thus decisions regarding organ acceptance. **Methods.** Using SRTR data, which includes data on all donors, waitlisted candidates, and transplant recipients in the United States, we assessed (1) the impact of macrovesicular steatosis on deceased donor yield (defined as number of livers transplanted per donor) and 1-y posttransplant graft failure and (2) the effect of incorporating this variable into existing SRTR risk-adjustment models. **Results.** There were 21 559 donors with any recovered organ and 17 801 liver transplant recipients included for analysis. Increasing levels of macrovesicular steatosis on donor liver biopsy predicted lower organ yield: ≥31% macrovesicular steatosis on liver biopsy was associated with 87% to 95% lower odds of utilization, with 55% of these livers being discarded. The hazard ratio for graft failure with these livers was 1.53, compared with those with no pretransplant liver biopsy and 0% to 10% steatosis. There was minimal change on organ procurement organization–specific deceased donor yield or program-specific posttransplant outcome assessments when macrovesicular steatosis was added to the risk-adjustment models. **Conclusions.** Donor livers with macrovesicular steatosis are disproportionately not transplanted relative to their risk for graft failure. To avoid undue risk aversion, SRTR now accounts for macrovesicular steatosis in the SRTR risk-adjustment models to help facilitate use of these higher-risk organs. Increased recognition of this variable may also encourage further efforts to standardize the reporting of liver biopsy results.

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INTRODUCTION

The Scientific Registry of Transplant Recipients (SRTR) publicly releases organ procurement organization (OPO)–specific reports (OSRs) and program-specific reports (PSRs), which include assessments of organ utilization and posttransplant outcomes, respectively. The SRTR has not traditionally included donor liver biopsy results (ie, those obtained at time of organ procurement) in posttransplant

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The data that support the findings of this study are available from the Scientific Registry of Transplant Recipients. Restrictions apply to the availability of these data, which were used under license for this study, and so are not publicly available.

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risk-adjustment because they are not always available and they may not be interpreted or reported consistently. Despite these concerns, biopsy results may influence outcomes and thus decisions regarding organ acceptance.[1]

Macrovesicular steatosis in the donor liver has been identified as a predictor of graft failure, and so these livers are often considered extended criteria or marginal livers.[2] Previous studies have demonstrated an association between ≥30% macrovesicular steatosis and lower 1-y graft survival, as well as primary graft nonfunction and early allograft dysfunction.[3–5] Hepatic steatosis in donor livers is thought to contribute to graft failure by decreased hepatic blood flow and microcirculation and increased intrahepatic vascular resistance at the time of transplantation, leading to ischemia/reperfusion injury.[6,7] This study evaluates (1) the effect of donor macrovesicular steatosis on liver yield and graft outcome and (2) the impact of incorporating this variable into SRTR risk-adjustment models for OPO-specific deceased donor yield and program-specific graft outcomes for liver transplantation.

MATERIALS AND METHODS

This study used data from SRTR. The SRTR data system includes data on all donors, waitlisted candidates, and transplant recipients in the United States, submitted by the members of the Organ Procurement and Transplantation Network, and has been described elsewhere.[8] The Health Resources and Services Administration, US Department of Health and Human Services, provides oversight of the activities of the Organ Procurement and Transplantation Network and SRTR contractors. The study was exempt from institutional review board approval, as the data are deidentified and publicly available.

We evaluated all donors referred for procurement between July 1, 2017, and June 30, 2019. Liver yield was defined as the number of transplanted livers from donors from whom any organ was recovered. Donor factors analyzed included demographics, laboratory values, comorbidities, cause of donor death, and type of donor (donation after circulatory death [DCD] and brain death [DBD]), as well as biopsy data. Liver biopsy results were as reported by the OPO, performed predonation or at the time of donation and interpreted by a pathologist. Grading of microvesicular and macrovasicular steatosis is reported on a continuous scale, ranging from 0% to 100%. Other features including inflammation, necrosis, and fibrosis may also be reported and available to centers for review but not in a standardized format. For this analysis, levels of macrovesicular steatosis were categorized as (1) 0% to 10%, (2) 11% to 30%, (3) 31% to 50%, (4) ≥51%, and (5) not available. Microvesicular steatosis was not considered, as this has not been associated with graft outcome. For adult deceased donor liver transplants performed between June 1, 2016, and June 30, 2018, posttransplant outcome was assessed by 1-y graft failure, defined as the time from transplant to death or retransplantation. Covariates considered for this study aligned with the SRTR risk-adjustment models for organ yield and posttransplant outcome. Because this was a retrospective study and macrovesicular steatosis was not included in the SRTR risk-adjustment models during the study period, the observed outcomes are reflective of OPO and center practices in that context.

The association between macrovesicular steatosis and deceased donor yield was assessed using multivariable logistic regression. Donor age and DCD total warm ischemic time (minutes between withdrawal of support and cross-clamp) had an interaction; that is, the effect of donor age depended on DCD total warm ischemic time. Based on clinical insights, we anticipated macrovesicular steatosis would also have an interaction with DCD status; as such, the model included an overall effect for macrovesicular steatosis and separate effect for only DCD donors.

LASSO (least absolute shrinkage and selection operator)-penalized Cox proportional hazards regression models were used to evaluate the association between macrovesicular steatosis and 1-y posttransplant graft failure, adjusted for recipient and donor factors.[9,10] The primary outcome was 1-y graft failure, defined as the time from liver transplant to death or retransplantation, with 1-y posttransplant mortality as a secondary outcome. Patients were censored if these events had not occurred at 1 y or by the date of last follow-up.

The impact of adding this variable to OPO-specific deceased donor yield estimates and program-specific posttransplant outcome assessments was assessed by comparing the multivariable models with and without macrovesicular steatosis. The publicly available SRTR risk-adjustment models released during the study period had not included macrovesicular steatosis.

RESULTS

Organ Yield

There were 21 559 donors with any recovered organ during the study period, with a mean age of 41 (SD = 17) y. The most common cause of death was anoxia (43.2%) , and 20.1% were designated as DCD. Liver biopsy data were available for 34.2%–23.6% who were graded as 0% to 10% macrovesicular steatosis, 5.9% as 11% to 30%, and 4.6% as ≥31% (Table 1).

Organ yield decreased with increasing levels of macrovesicular steatosis at a rate of 0.50 for livers with 31% to 50% steatosis and 0.31 for livers with ≥51% steatosis among DBD donors (Figure 1). Fifty-five percent of livers with ≥31% macrovesicular steatosis were discarded. DCD status was associated with lower organ yield at all levels at a rate of 0.17 for livers with 31% to 50% steatosis and 0.02 for livers with ≥51% steatosis. In the multivariable logistic regression, livers were less likely to be utilized with increasing levels of steatosis (among DBD donors: OR = 0.72 for 11%–30% steatosis, OR = 0.13 for 31%–50% steatosis, and OR = 0.05 for ≥51% steatosis); in addition, the DCD-specific effect was observed (**Table S1, SDC,** http://links.lww.com/TP/C531).

Posttransplant Outcomes

There were 17 801 liver transplant recipients during the study period, with a mean age of 58 (SD = 11) y (Table 1). The most common indication was cirrhosis or chronic liver disease (65.5%), followed by malignant neoplasms (17.7%). The mean biochemical Model for End-stage Liver Disease (MELD) at transplant was 21. Of the livers they received, 6.8% were designated as DCD, the mean cold ischemia time was 5.8 h (SD 2.0), and 2.0% had ≥31% macrovesicular steatosis.

TABLE 1.

Demographic characteristics of donors and recipients

Characteristic	Value	Characteristic	Value
Donors (N = 21 559)		Recipients (N = 17 801)	
Age, mean (SD), y	41 (17)	Age, mean (SD), y	58 (11)
Sex, no. (%)		Sex, no. (%)	
Female	8507 (39.5)	Female	11731 (65.9)
Male	13 052 (60.5)	Male	6070 (34.1)
Race, no. (%)		Race, no. (%)	
Asian	529 (2.5)	Asian	15193 (85.3)
Black	3502 (16.2)	Black	1580 (8.9)
White	17207 (79.8)	White	725 (4.1)
Other	321 (1.5)	Other	303 (1.7)
Height, mean (SD), cm	168 (19)	Height, mean (SD), cm	173 (10)
Weight, mean (SD), kg	81 (26)	Weight, mean (SD), kg	83 (20)
Cause of death, no. (%)		Diagnosis, no. (%)	
Anoxia	9319 (43.2)	Acute hepatic necrosis	680 (3.8)
Trauma	5865 (27.2)	Malignant neoplasms	3151 (17.7)
CVA/stroke	5717 (26.5)	Noncholestatic cirrhosis	11656 (65.5)
Other	658 (3.1)	Other	2314 (13.0)
History of heavy alcohol use, no. (%)	4046 (18.8)	Biochemical MELD, mean (SD)	21 (10)
Diabetes, no. (%)	2759 (12.8)	Bilirubin, mean (SD), mg/dL	3.6 (10.7)
Hypertension, no. (%)	7623 (35.4)	INR, mean (SD)	0.5 (0.4)
HCV antibody positive, no. (%)	1197 (5.6)	Serum creatinine, mean (SD), mg/dL[a]	1.1 (1.3)
HIV positive, no. (%)	61 (0.3)	Serum sodium, mean (SD), mmol/L	137 (5)
Blood type, no. (%)		Albumin, mean (SD), g/dL	3.1 (0.7)
A	7908 (36.7)	Encephalopathy, no. (%)	11177 (62.8)
B	2592 (12.0)	Diabetes, no. (%)	5305 (29.8)
O	10341 (48.0)	Life support, no. (%)	1632 (9.2)
AB	717 (3.3)		
Donation after circulatory death, no. (%)	4329 (20.1)	Life support: ventilator, no. (%)	830 (4.7)
PHS increased infectious risk, no. (%)	5838 (27.1)	Prior liver transplant, no. (%)	778 (4.4)
		Portal vein thrombosis, no. (%)	2670 (15.0)
		Cold ischemia time, mean (SD), h	5.8 (2.0)

	Overall	DBD	DCD	
Macrovesicular steatosis, no. (%)				Medical condition at transplant (%)
0–10%	5096 (23.6)	4779 (27.7)	317 (7.3)	Not hospitalized 11752 (66.0)
11–30%	1269 (5.9)	1194 (6.9)	75 (1.7)	Hospitalized 3393 (19.1)
31–50%	721 (3.3)	681 (4.0)	40 (0.9)	In ICU 2656 (14.9)
≥50%	288 (1.3)	267 (1.5)	21 (0.5)	
Not available	14185 (65.8)	10309 (59.8)	3876 (89.5)	

[a]Creatinine defaulted to 4 mg/dL if recipient had dialysis twice, or 24 h of continuous venovenous hemodialysis, within a week before the serum creatinine test.
CVA, cerebrovascular accident; DBD, donation after brain death; DCD, donation after circulatory death; HCV, hepatitis C virus; ICU, intensive care unit; INR, international normalized ratio; MELD, Model for End-stage Liver Disease; PHS, Public Health Service.

In the Cox regression analysis for 1-y posttransplant graft failure, increased hazard of graft failure was observed among recipients of grafts with 11%–30% (HR = 1.25) and ≥31% macrovesicular steatosis (HR = 1.53) (Figure 2). There was no increased risk of graft failure with 0% to 10% macrovesicular steatosis compared with those without liver biopsy. Similar results were seen in the secondary analysis for patient death, although the effect was less pronounced because of the possibility of retransplantation, with an HR of 1.17 for recipient of grafts with 11% to 30% macrovesicular steatosis and with an HR = 1.22 for recipients of grafts with ≥31% macrovesicular steatosis.

Impact on Deceased Donor Yield and Posttransplant Outcome Assessments

For each OPO and transplant center in the United States, SRTR provides performance indicators via OSRs and PSRs every 6 mo. The OSRs report on OPO performance in terms of the number of deaths and donor conversion rates, and PSRs report on organ-specific program performance for each transplant center in terms of candidates waiting for transplant, transplant recipients, and outcomes on the waiting list and after transplant. An HR of 1 indicates that a program is performing as expected, whereas a higher HR suggests more events than expected.[11] There was minimal impact on OPO-specific deceased donor yield estimates or program-specific posttransplant outcome assessments when the previous model was compared with an updated model with the addition of macrovesicular steatosis; that is, based on retrospective data, these risk-adjusted performance indicators were similar whether or not macrovesicular steatosis was included in the models and did not appear to differentially affect OPOs or programs (**Figure S1, SDC,** http://links.lww.com/TP/C531).

DISCUSSION

The study results show that macrovesicular steatosis on donor liver biopsy is associated with lower organ yield and reduced graft survival, consistent with findings from previous studies.[5] We observe that the hazard of graft failure observed with steatotic livers is lower than that of other extended-criteria livers, whereas their discard rate is higher. For example, the risk for graft failure for DCD livers compared with DBD livers has been estimated at 1.65 to 1.73, or a 65% to 73%

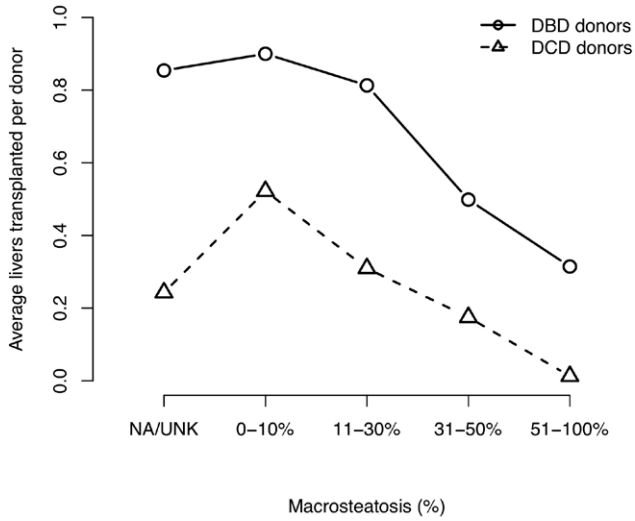

FIGURE 1. Average livers transplanted per donor by degree of macrovesicular steatosis, stratified by DCD status. DCD, donation after circulatory death; DBD, donation after brain death; NA/UNK, not available or unknown.

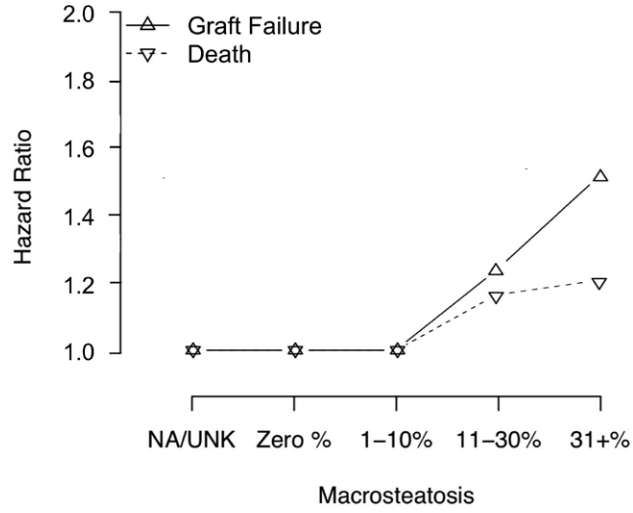

FIGURE 2. 1-y posttransplant graft failure and mortality by degree of macrovesicular steatosis. NA/UNK, not available or unknown.

increased risk compared with DBD livers.[12,13] Based on single-center reports, the graft failure risk with DCD donors may be lower in experienced programs.[14,15] In our analysis, even among those with no biopsy available, DCD livers had over 70% lower odds of organ yield. By comparison, the risk of graft failure for donor livers with ≥31% steatosis was 1.53 but with 90% lower odds of organ yield, suggesting that steatotic livers are disproportionately not transplanted relative to their risk for graft failure.

Macrovesicular steatosis, when added to the models, does not significantly change OPO-specific deceased donor yield estimates or program-specific posttransplant outcome assessments. This is overall reassuring that macrovesicular steatosis, despite not being previously accounted for in the SRTR risk-adjustment models, has not been a major contributor to overall OPO or center performance—albeit in the context of selective utilization and high discard rates. Observations from this study might be influenced by active donor–recipient matching, wherein surgeons may decline a steatotic liver for a critically ill patient but accept it for a lower acuity patient (eg, a patient with hepatocellular carcinoma). Indeed, it has been shown that in "preferred" recipients (ie, first-time recipients with MELD scores of 15–34, without primary biliary cholangitis, and not on life support), steatotic livers are not associated with increased risk of mortality or graft loss.[16] The rates of success historically described may be, in part, because of appropriate selectivity, and observed outcomes may differ if these thresholds change.

The persistent disparity between the number of patients on the waiting list and the number of available donor livers pressures transplant programs to make use of less-than-ideal grafts, such as those with steatosis.[17] With the increasing prevalence of obesity and fatty liver disease, OPOs and transplant programs increasingly face decisions regarding steatotic donor livers. Recent evidence suggests that, for certain populations, these organs may be used with acceptable outcomes and can confer a survival benefit compared with waiting on the list, for example, those with lower MELD and no previous liver transplant.[16,18] Within the past year, SRTR has added donor macrovesicular steatosis as a variable to the publicly available risk-adjustment models to reduce disincentives in the use of these organs, minimize discard of potentially useful livers, and protect against potential negative effects from increased acceptance of these organs—acknowledging that outcomes may vary with expanded use of steatotic grafts, depending on center or provider experience, recipient selection, or other unmeasured donor characteristics. Use of steatotic livers is associated with not only graft failure but also an increased risk of postreperfusion syndrome, early allograft dysfunction, and acute kidney injury, so these grafts do need to be used with caution and judicious donor–recipient matching.[19]

Limitations of this variable in the SRTR database should be acknowledged. Currently, the decision to perform liver biopsy and the recorded data are not standardized, and fewer than half of donors had liver biopsies performed, which could be a source of bias. Accuracy and availability of liver biopsy remain a concern because histologic assessment of steatosis by donor liver biopsy can be cumbersome and prone to sampling error and interobserver variability.[20,21] If there is clear evidence of steatosis on imaging, donors might not undergo liver biopsy and thus would not be accounted for in the organ yield model. For now, histology remains the most reliable method by which to assess hepatic steatosis in potential donor organs.[22] With donor macrovesicular steatosis having been added to the SRTR risk-adjustment models, efforts to make donor liver biopsies more accessible and interpreted by a specialized pathologist or development of novel technologies for more accurate assessment of steatosis may accelerate.[23,24] Normothermic perfusion and techniques such as "defatting" may also impact the utilization and outcomes of steatotic livers and deserve further attention as they become more widely used.[25,26] In addition, the number of biopsies performed may increase as a result of this change, which could independently influence organ discard rates. Notably, among DCD donors with 0% to 10% macrovesicular steatosis, there was increased liver yield compared with those without biopsy, suggesting that the biopsy may in some cases be reassuring.

Before this analysis, biopsy results had not been included in the publicly available SRTR posttransplant risk-adjustment models. Our study shows that macrovesicular steatosis on liver biopsy clearly influences decisions regarding organ acceptance. These organs, although less than ideal, can be utilized in certain situations and benefit many patients on the liver transplant waitlist; however, programs may avoid these riskier transplants if that risk is not adjusted for in their evaluations. To avoid undue risk aversion, SRTR now accounts for macrovesicular steatosis as a categorical variable in the risk-adjustment models for organ yield and posttransplant outcome to facilitate use of these higher-risk organs. Model coefficients, including macrovesicular steatosis, are updated biannually with each OSR and PSR release and can be viewed online through the SRTR interactive tools at https://www.srtr.org/tools/deceased-donor-yield/ and https://www.srtr.org/tools/posttransplant-outcomes/. Utilization of these organs, supported by careful patient selection, appropriate transplant techniques, and greater standardization in the assessment of steatosis, could lead to an increase in the number of transplanted organs in the United States.

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