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Clinical Investigation

Incidence, Predictors, and Outcomes of New-Onset Left Ventricular Systolic Dysfunction After Orthotopic Liver Transplantation

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ABSTRACT

Background: Adverse cardiovascular events after liver transplantation (LT) are relatively common and are a significant source of early mortality. Although new-onset systolic dysfunction after LT is a reported phenomenon, there is little data regarding its incidence, risk factors, and outcomes.

Methods and Results: This single-center retrospective study included all adult patients from January 2002 to March 2015 with deceased-donor LT and available preoperative transthoracic echocardiograms (TTEs). In total, 1,760 patients were included in the study, 602 (34.2%) of whom had a postoperative TTE. The primary end point was development of new-onset cardiomyopathy, defined as a new left ventricular ejection fraction (LVEF) of <40% within 180 days of transplant. Sixty-nine (11.4%) of the patients who received post-LT TTE had a reduction in LVEF to <40% within 6 months. Clinical parameters of donor and recipient did not show significant impact on development of post-LT LV systolic dysfunction (LVSD). Presence of wall motion abnormalities ($P = .004$) on preoperative TTE was predictive of development of post-LT LVSD. These patients did not have longer hospitalizations, but they had worse survival.

Conclusions: Post-LT LV systolic dysfunction occurs at higher rates than previously suspected and may develop more frequently in patients with underlying cardiac structural abnormalities, which appear to adversely affect post-LT survival. (*J Cardiac Fail 2019;25:166–172*)

Key Words: Left ventricular systolic dysfunction, liver transplant, heart failure.

More than 600,000 individuals in the United States are living with cirrhosis[1] due to viral hepatitis, alcohol abuse, and nonalcoholic fatty liver disease, with chronic liver disease being the eighth leading cause of death.[2] When cirrhosis progresses to end-stage liver disease, the only definitive treatment is liver transplantation (LT). According to the United Network for Organ Sharing, more than 14,000 people are listed for a liver transplant,[3] the second most common solid organ transplant, but owing to the limited supply of suitable organs, up to 20% of patients will die before receiving an offer for a high-quality organ.[4] Physicians evaluating patients for LT must ensure that they are likely to tolerate a high-risk operation which has significant operative time, blood loss,[5] metabolic derangements,[6] and hemodynamic instability[7] and that recipients have the greatest likelihood for a long-term survival without significant other complications. Patients undergoing LT are increasingly older and have risk factors for cardiac dysfunction, including hypertension, obesity, and diabetes mellitus,[8] and patients with established coronary artery disease (CAD), arrhythmias, or structural heart disease have worse outcomes after liver transplantation.[9–11] Multiple studies have shown that cardiovascular events, including heart failure, arrhythmias, and myocardial ischemia, are a leading cause of post-LT morbidity and mortality, with incidence ranging from 11% to 26%.[9,12,13] Several studies have reported on post-LT heart failure and LV systolic

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dysfunction (LVSD), although most studies have been small in both the number of transplants and the number of cardiac events.[14–16] One larger study examined heart failure based only on symptoms and supportive findings and included events that occurred years after the transplantation.[17] The goal of the present study was to describe the incidence of post-LT LVSD, here defined as a new-onset left ventricular ejection fraction (LVEF) of <40% within 180 days of transplantation. In addition, we examined associated preoperative risk factors for the development of post-LT LVSD and explored its impact on long-term survival.

Methods

Study Design

This was a retrospective cohort study of all consecutive adult (≥18 years old at the time of transplant) patients undergoing liver transplantation at the University of California—Los Angeles (UCLA) Medical Center from January 1, 2002, to March 1, 2015, with at least 1 year of follow-up. Per institutional protocol, all patients undergo annual preoperative echocardiography. Patients with known CAD or significant diabetes mellitus (history >5 years or requiring insulin) and those >45 years old are evaluated with a preoperative stress test or coronary angiography as clinically appropriate, and those with obstructive CAD are revascularized before liver transplantation according to protocol. Inclusion criteria were all patients with pre-LT LVEF ≥50%. Patients who underwent simultaneous liver-kidney transplantation was also included. For patients who underwent more than one liver transplantation during the study period, only the first event was included. If a patient was receiving a second liver transplant, but it was the first transplantation in the study period, this was included and the patient was noted to be receiving a "re-do" transplant. Exclusion criteria included patients who underwent simultaneous or previous heart transplantation or living-related liver transplantation, those whose preoperative transthoracic echocardiogram (TTE) showed LVEF <50%, and those whose TTE imaging was unavailable owing to limitations in electronic medical record retrieval. The center's Institutional Review Board approved the study. All patients that met the inclusion criteria underwent chart review, including their preoperative TTE and all inpatient and outpatient postoperative TTEs, at the transplanting institution up to 180 days after transplantation.

Data Collection

Donor, recipient, and peritransplantation variables of interest were obtained from the institution's transplant database and electronic medical record. Recipient variables were age, sex, hypertension, CAD (defined as previous myocardial infarction, revascularization, and both documented obstructive and nonobstructive coronary atherosclerosis), diabetes, hepatitis C, previous liver transplantation, etiology of liver disease, hepatic malignancy, and laboratory Model for End-Stage Liver Disease (MELD) score. The laboratory MELD score was calculated based on a previously described formula[13] before the use of sodium in the model and excluded exception points. Donor variables included in this analysis were age, sex, and donation after cardiac death. Peritransplantation variables included hospitalization at time of transplantation, need for vasopressors, mechanical ventilation, dialysis, cold ischemia time, and simultaneous kidney transplantation. Echocardiographic variables were obtained through review of the echocardiography reports and included LVEF as a measure of systolic function, presence of wall motion abnormalities, grade of valvular regurgitation for all valves, presence of aortic stenosis, right ventricular (RV) dysfunction, left atrial (LA) enlargement (LAE), elevated RV systolic pressure (RVSP), and presence of a pericardial effusion. When available, elevated RVSP was defined as >35 mm Hg, otherwise the subjective report of normal or elevated RVSP was used. When available, LA enlargement was defined as an indexed LA volume >34 mL/m,^{2} otherwise when unindexed LA volume was available it was defined as >68 mL, and when neither was available the subjective report of normal or enlarged LA was used. In patients who demonstrated a drop in LVEF, additional variables were obtained, including peak cardiac protein (troponin and CK-MB, if available), pre-and postoperative ischemia assessments, use of cardioprotective and antiarrhythmic medications after surgery, and arrhythmic events. We then divided the patients who developed LVSD into groups based on LVEF <40% and the presence or absence of recovery at 1 month. A 30-day recovery time was selected from literature review because of previously reported variable rates of recovery for stress-induced cardiomyopathies.[18–21]

Data Analysis

The primary outcome assessed was new-onset LVSD defined as the development of a new LVEF <40% within 180 days of transplantation. Descriptive statistics were calculated and presented for the cohorts. Continuous variables are presented as mean ± SD if normally distributed and compared by means of an independent-samples *t* test. Those with skewed distributions are presented as median (interquartile range [IQR]) and compared by means of the Wilcoxon rank sum test or Kruskal-Wallis test. Categoric variables are presented as proportions and compared by means of Fisher exact test. Time-to-event data, including survival after transplantation, were displayed with the use of a Kaplan-Meier curve. To examine predictors of new-onset LVSD, a logistic regression model was created. Given its exploratory nature, the model was developed in a backward stepwise fashion in which all candidate variables were included and variables with $P > .15$ were removed sequentially as the model was rerun until no further variables met criteria for removal.

Results

During the study period, there were 2205 patients considered for inclusion (Fig. 1). A total of 443 patients (20.1%)

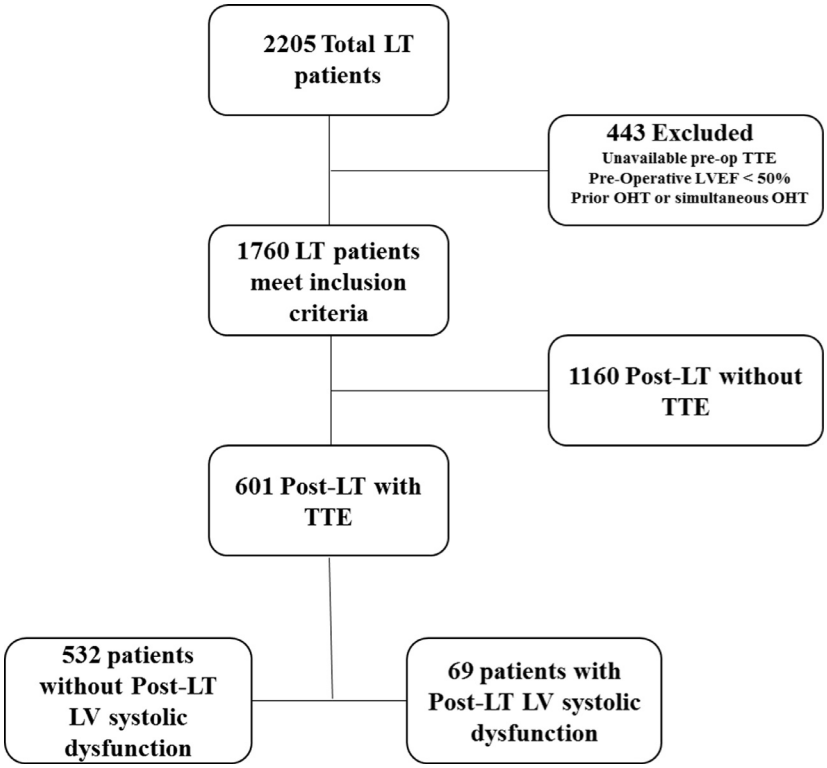

Fig. 1. Study design and screening. A proportion of patients were excluded from the study because of either inaccessible preoperative TTE, a preoperative TTE with LVEF <50%, or planned simultaneous or previous OHT. Median time between transplantation and postoperative TTE was 14 days. Sixty-nine post-LT patients were found to have LVEF <40% and were diagnosed with post-LT LV systolic dysfunction. LT, liver transplantation; LV, left ventricular; LVEF, left ventricular ejection fraction; OHT, orthotopic heart transplantation; TTE, transthoracic echocardiography.

were excluded, for reasons including an inaccessible preoperative echocardiography report (n = 310), a preoperative LVEF <50% (n = 6), a retransplantation during the same study period (n = 124), or simultaneous or previous heart transplantation (n = 3), leaving 1,760 patients for the study with a median of 68 days between preoperative TTE and the LT. Of the entire cohort, 601 (34.1%) had a postoperative TTE within 6 months of the procedure and were included in the analysis. The median time between transplantation and postoperative TTE was 14 days, and 74% of the patients had the TTE before discharge from the transplantation hospitalization. Of the patients with a postoperative TTE, 69 (11.4%) were noted to have an LVEF of fewer than 40%, discovered at a median of 7 days after transplant. These patients had a median of 69 days between preoperative TTE and LT. Of the entire cohort of patients undergoing transplantation, this represents an incidence of 3.9%. The degree of systolic dysfunction was stratified in patients with post-LT LVSD: 20 patients (20.4%) had LVEF ≤20%, 21 (21.4%) had LVEF 21%−30%, and 28 (28.6%) had LVEF 31%−39%. Although not protocolized, all 69 patients (100%) received a follow-up TTE after discovery of post-LT LVSD. Of patients with new-onset LVSD, 56 (81.1%) had troponin checked after transplantation at the discovery of the newly reduced LVEF. The median peak troponin elevation was 0.24 ng/mL (upper normal range 0.04 ng/mL), and 42 patients (61%) had a troponin <1 ng/mL. Postoperative arrhythmias were relatively common in these patients, with 23 (33%) experiencing atrial fibrillation or flutter after transplantation and an additional 7 (10.1%) suffering from ventricular arrhythmias. Mechanical circulatory support, defined as extracorporeal membrane oxygenation or intra-aortic balloon pump, was required in 8 patients (11.6%), and ionotropic blood pressure support was required in 29 (42.0%). After discovery of post-LT LVSD, 29 patients (42.0%) were treated with beta-blockers, 11 (16.0%) with angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers, 7 (10.1%) with aldosterone antagonists, and 7 (10.1%) with digoxin.

Clinical and transplant characteristics of the patients are presented in Table 1, stratified by the presence or absence of new post-LT LVSD defined as LVEF <40%. The groups did not statistically vary in their clinical characteristics. Simultaneous liver-kidney transplantation and post-LT length of stay trended toward but did not reach statistical significance: 2.9% vs 9.6% ($P = .071$) and 44 days vs 34 days ($P = .088$), respectively. The rates of CAD, diabetes, and hypertension and the MELD scores did not differ between groups. Perioperative parameters, including preoperative dialysis needs, mechanical ventilation, vasopressor use, intraoperative packed red blood cell use, incidence of major reperfusion syndrome, and re-do liver transplantation were similar between groups. Donor characteristics, including donor age, donor sex, and cardiac versus brain death of

Table 1. Clinical and Transplant Recipient Characteristics

Characteristic	No Post-LT LVSD (n = 532)	Post-op LVEF <40% (n = 69)	*P* Value*
Age (y)	58 (51−63)	57 (50−63)	.388
Male	338 (63.5%)	40 (58%)	.427
CAD	61 (11.5%)	10 (14.5%)	.432
Diabetes	160 (30.1%)	14 (20.3%)	.12
Hypertension	190 (35.8%)	18 (26.1%)	.139
Lab MELD score	35 (28−40)	34 (28−39)	.533
Requiring dialysis	244 (45.9%)	34 (49.3%)	.61
Mechanical ventilation	149 (28%)	23 (33.3%)	.396
Requiring vasopressors	112 (21.1%)	11 (15.9%)	.427
Donation via cardiac death	26 (4.9%)	4 (5.8%)	.767
Male donor	298 (63.4%)	43 (65.2%)	.891
Donor age (y)	40 (25−53)	36 (23.8−56)	.729
Cold ischemia time (h)	6.9 (5.2−8.6)	6.6 (4.6−8.0)	.186
Intraoperative PRBC (units)	16 (10−26)	18 (11−25)	.456
Major reperfusion syndrome	38 (10.8%)	6 (12.5%)	.805
Re-do liver transplant	25 (4.7%)	4 (5.8%)	.763
Simultaneous kidney transplant	51 (9.6%)	2 (2.9%)	.071
Post-LT length of stay (d)	34 (18−64)	44 (21−88)	.088

Values are presented as median (interquartile range) or n (%). CAD, coronary artery disease; MELD, Model for End-Stage Liver Disease; PRBC, packed red blood cells; LT, liver transplantation; LVEF, left ventricular ejection fraction; LVSD, left ventricular systolic dysfunction.

*Kruskal-Wallis test for continuous, Fisher exact test for categoric variables.

the donor, as well as cold ischemia time did not vary between groups. The preoperative echocardiography characteristics are presented in Table 2. LVEF was quantitatively and qualitatively assessed as a categoric variable: low-normal (50%−55%), normal-high (56%−74%), and hyperdynamic (≥75%). The post-LT LVSD group had higher rates of preoperative low-normal LVEF (14.5% vs 6.0%; *P* = .025) and more wall motion abnormalities (8.7% vs 2.6%; *P* = .02). Moderate or severe aortic or mitral insufficiency was rare and did not differ between groups before transplantation. LAE, a marker of increased left atrial pressure and diastolic dysfunction, did not differ between groups. Preoperative echocardiograms were similar in patients with and without post-LT systolic dysfunction in terms of abnormal RV function, RVSP, and pericardial effusions.

Table 2. Preoperative Echocardiographic Characteristics, n (%)

Characteristic	No Post-LT LVSD (n = 532)	Post-LT LVEF <40% (n = 69)	*P* Value*
LVEF range			.025
50%−55%	32 (6%)	10 (14.5%)	
56%−74%	474 (89.1%)	58 (84.1%)	
≥75%	26 (4.9%)	1 (1.5%)	
Wall motion abnormalities	14 (2.6%)	6 (8.7%)	.020
Abnormal RV function	7 (1.3%)	0 (0%)	1.000
Aortic regurgitation (>2+)	7 (1.3%)	1 (1.45%)	1.000
Mitral regurgitation (>2+)	17 (3.2%)	2 (2.9%)	1.000
Left atrial enlargement	163 (31.8%)	27 (39.7%)	.216
Elevated RVSP	127 (28.9%)	16 (28.6%)	1.000
Pericardial effusion			.598
None	502 (94.9%)	68 (98.6%)	
Small	23 (4.4%)	1 (1.5%)	
Moderate or large	4 (1%)	0 (0%)	

RV, right ventricular; RVSP, right ventricular systolic pressure; TTE, transthoracic echocardiography; other abbreviations as in Table 1.

*Kruskal-Wallis test for continuous, Fisher exact test for categoric variables.

Indications for TTE as given by the ordering physician were reviewed in the post-LT LVSD group and were categorized into 5 groups: heart failure signs and symptoms (n = 38; 38.4%), concern for acute coronary syndrome (n = 29; 29.7%), arrhythmias (n = 23; 23.5%), possible endocarditis or valvular disease (n = ; 5.1%), and post−cardiac arrest (n = 4; 4.1%).

To examine the comorbidities associated with post-LT LVSD, a logistic regression model was created through backward stepwise selection of all possible variables (Table 3). In this model, abnormalities within the preoperative echocardiogram had the greatest association with post-LT LVSD, including preoperative LVEF 50%−55% (odds ratio [OR] 2.4, confidence interval [CI] 0.9−6.1), preoperative systolic wall motion abnormalities (OR 4.0, CI 1.20−13.5), and simultaneous kidney-liver transplant (OR 0.16, CI 0.02−1.23).

In terms of recovery, 63.1% had a documented recovery to an LVEF >50%, with a median time to recovery of 11 days, ranging from 1 day to 4.76 years. Of those that experienced recovery, 68.0% had recovery before discharge from the index hospitalization and 97.7% experienced recovery before 1 year. In an attempt to differentiate LVSD in the setting of liver cirrhosis from stress-induced cardiomyopathy associated with LT, we stratified patients by the presence of recovery within 30 days based on literature suggesting that cardiomyopathies that only have a stress component have variable rates of recovery.[18−21] Of the 69 patients with new-onset LVSD, 34 (49.3%) demonstrated recovery within 30 days. Conversely, 35 (50.7%) did not demonstrate recovery within 30 days. Although the limited number of patients precluded statistical analysis, the data may be useful in an exploratory manner. Patients who failed to demonstrate LVEF recovery within 30 days did not exhibit differences in age, sex, rates of diabetes, hypertension, or known CAD from those that did demonstrate recovery within 30 days (Table 4). Markers of critical illness before transplantation did not vary between groups and included MELD scores, preoperative LVEF, hemodialysis, mechanical ventilation, and vasopressor requirements.

Table 3. Models for New-Onset Post-LT LVSD

Model	Odds Ratio (95% CI)	*P* Value*
Preoperative LVEF 50%−55%	2.4 (0.9−6.1)	.069
Preoperative SWMA	4.0 (1.2−13.5)	.026
Simultaneous kidney-liver transplant	0.16 (0.02−1.23)	.083

CI, confidence interval; SWMA, systolic wall motion abnormality; other abbreviations as in Table 1.

*P < .15 only.

Table 4. Characteristics by Recovery Time of ≤30 Days or ≥31 Days After Liver Transplantation

Characteristic	Recovery ≤30 Days (n = 34)	Recovery >30 Days (n = 35)	*P* value*
Age (y)	55.5 (48−62)	57 (51−63)	.394
Male	19 (55.9%)	21 (60%)	.809
CAD	6 (17.7%)	4 (11.4%)	.513
Diabetes	6 (17.7%)	8 (22.9%)	.766
Hypertension	8 (23.5%)	10 (28.6%)	.785
Lab MELD score	34 (27−39)	33 (29−38)	.976
Requiring dialysis	17 (50%)	17 (48.6%)	1
Mechanical ventilation	9 (26.5%)	14 (40%)	.309
Requiring vasopressors	6 (17.7%)	5 (14.3%)	.752
Donation via cardiac death	2 (5.9%)	2 (5.7%)	1
Male donor	20 (64.5%)	23 (65.7%)	1
Donor age (y)	47 (25−57.3)	33.6 (23−48)	.169
Cold ischemia time (h)	6.9 (4.9−8.8)	6.4 (4.1−7.4)	.165
Intraoperative PRBC (units)	18 (11−30)	18 (12−25)	.938
Major reperfusion syndrome	2 (11.8%)	4 (12.9%)	1
Re-do liver transplant	0 (0%)	4 (11.4%)	.114
Simultaneous kidney transplant	0 (0%)	2 (5.7%)	.493
Pre-LT LVEF >65%	6 (17.7%)	7 (20%)	1
Pre-LT LVEF <55%	4 (11.8%)	6 (17.1%)	.734
Wall motion abnormalities	2 (5.9%)	4 (11.4%)	.673
Abnormal RV function	0 (0%)	0 (0%)	N/A
Aortic regurgitation (>2 +)	0 (0%)	1 (2.9%)	1
Mitral regurgitation (>2 +)	1 (2.9%)	1 (2.9%)	1
Left atrial enlargement	10 (29.4%)	17 (50%)	.136
Elevated RVSP	5 (17.2%)	11 (40.7%)	.076
Nadir LVEF			.332
31%−40%	12 (35.3%)	17 (48.6%)	
≤30%	21 (61.8%)	18 (51.4%)	

Abbreviations as in Tables 1 and 2.
*Kruskal-Wallis test for continuous, Fisher exact for categoric variables.

Perioperative characteristics, including cold ischemia time, intraoperative blood transfusion requirements, and incidence of major reperfusion syndrome, were also similar.

For those patients who demonstrated recovery after 30 days, re-do liver transplantations appeared to be more common in the delayed-recovery group (11.4% vs 0%; $P = .114$), which group also had a greater number of simultaneous kidney transplants (5.7% vs 0%; $P = .493$). The groups had similar rates of LAE (50.0% vs 29.4%; $P = .136$) and elevated RVSP (40.7% vs 17.2%; $P = .076$). Nadir LVEF ≤30% in the 30-day recovery group was 61.8% versus 51.4% in the delayed-recover group, and nadir LVEF 31%−40% in the 30-day recovery group was 38.2% versus 48.6% in the delayed-recover group, but these were not statistically significant.

To assess the impact of post-LT systolic dysfunction on survival, Kaplan-Meier curves up to 1,000 days after transplantation were created and are shown in Fig. 2. Survival was significantly worse for patients who required postoperative echocardiography, even when normal, and patients with post-LT systolic dysfunction compared with the remaining patients, as demonstrated by the log-rank test of survival function ($P < .0001$). Through graphic representation, patients with post-LT LVSD appeared to have worse survival; however, this did not meet statistical significance in the log-rank test ($P = .348$).

Discussion

This retrospective single-center study describes the incidence and clinical predictors for development of post-LT LVSD. To our knowledge, it represents the largest and longest analysis of post-LT LVSD in patients undergoing liver transplantation. This study's population was composed of severely advanced cirrhotic patients, evidenced by median MELD scores of 34 and 35 in the post-LT systolic dysfunction and LT groups, respectively. A higher MELD score represents a poorer prognosis and signifies more critical illness with higher likelihood of requiring dialysis, mechanical ventilation, and intraoperative blood product use; however, incidence did not differ between groups. We found that 69 transplant recipients developed post-LT LVSD within 6 months, a conservative estimate of 3.9%, but it may be as high as 11.4% when analyzing all patients who received post-LT TTE. Our estimate is in line with the 3.4%−6.9% reported in smaller studies within the MELD scoring system era.[17] Qureshi et al categorized heart failure as systolic, diastolic, or mixed-type and diagnosed new cardiomyopathy by identifying 2 new symptoms of heart failure, with confirmatory laboratory testing, and LVEF <50%.[17] Our study focused on development of post-LT LVSD regardless of the type and limited subjective clinical interpretation. Yataco et al defined LVSD as the presence of LV dilation and decreased LVEF without mentioning severity and excluded delayed post-LT LVSD patients, reporting an incidence of 1.2%.[22] Incidence of post-LT LVSD in our study was more prevalent than expected with median time of post-LT TTE occurring in 14 days. Previous studies show mean time of onset of LVSD occurring 2−5 days after LT.[22,23] In the present study, there was no impact of donor or recipient preoperative clinical parameters on development of post-LT LVSD. Preoperative TTE with evidence of systolic wall motion abnormalities was predictive of post-LT LVSD. In addition, patients with post-LT LVSD had significantly worse survival. We found that 63.1% of patients who developed LVSD had documented recovery to LVEF >50% with a mean time of recovery of 11 days. Thirty-four patients (49.3%) demonstrated recovery within 30 days, which is consistent with the time course of recovery seen in stress-induced cardiomyopathy. We suggest that the most susceptible patients are those with underlying cirrhotic cardiomyopathy, a chronic condition defined as reduced cardiac contractility and diminished responsiveness to stress, with or without diastolic dysfunction and electrophysiologic abnormalities, in the absence of known cardiac disease.[24] Portal hypertension and cirrhosis lead to vascular dysfunction, hormonal irregularities, and electrolyte imbalances, resulting in

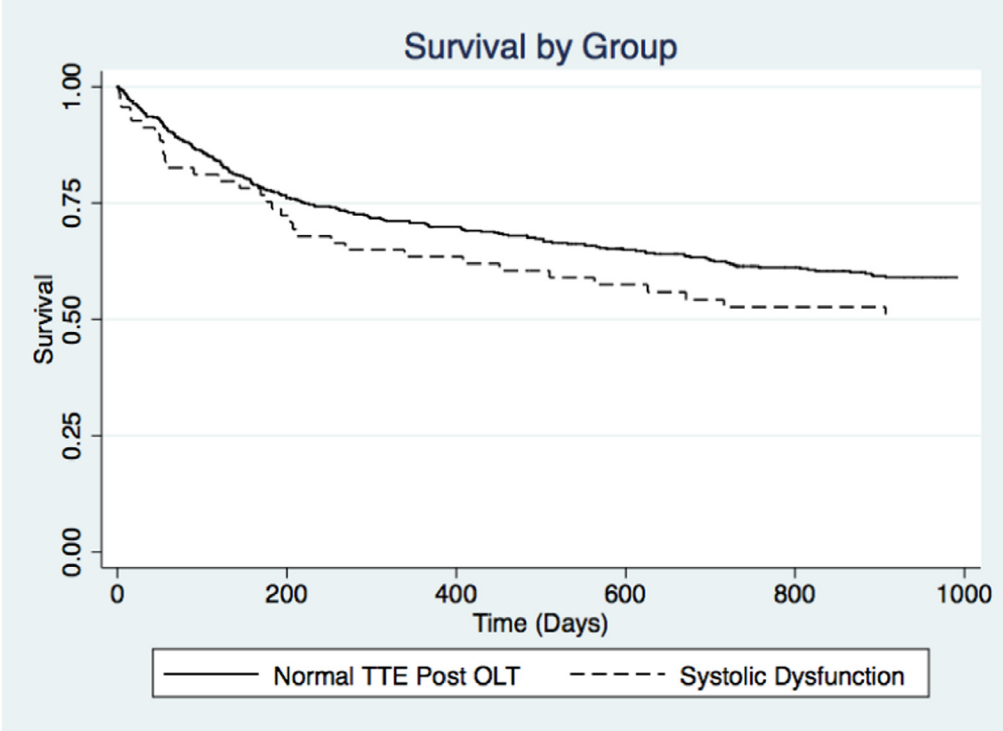

Fig. 2. Post-LT LV systolic dysfunction survival. The impact of post-LT left ventricular systolic dysfunction (LVEF <40%) on survival. Patients followed up to 1,000 days after transplantation. LT, liver transplantation; LVEF, left ventricular ejection fraction.

myocyte dysfunction which manifests as a progression from a resting high cardiac output state to one with diminished systolic and diastolic function, electrical conduction disturbance, and chronotropic incompetence.[24] Despite a paucity of data, it is estimated that up to 50% of cirrhotic patients undergoing transplant evaluation may have underlying cardiac dysfunction or cirrhotic cardiomyopathy.[24] This maladaptive state becomes exposed during the correction of a previously low systemic vascular resistance by LT, which results in elevated LV filling pressures, inadequate cardiac output, and cardiogenic shock.[25]

Based on our model, preoperative TTE can help to predict development of post-LT LVSD through identification of wall motion abnormalities. Early recognition of at-risk LVSD patients in the quiescent pre-LT phase is challenging but may allow for therapeutic optimization. We think that post-LT patients that did not show LVEF recovery represent a susceptible population with maladaptive cardiac remodeling. Other causes for a lack of systolic recovery include ischemia and genetic predilection to cardiomyopathy. The combination of systemic vasodilation and high cardiac output in liver cirrhosis led to normal-high or hyperdynamic LVEF in 84.1%−89.1% and 1.5%−4.9% of study patients, respectively. In contrast, a low-normal LVEF (LVEF 50%−55%) was observed 6.0%−14.5% of the time and may signify underlying loss of beta-receptors in cirrhotic cardiomyopathy, ventricular remodeling, or a combination of both. Thus, cardiac remodeling with chronic liver cirrhotic physiology may be a predictor of poor recoverability. We think that different levels of systolic and diastolic dysfunction exist in our post-LT LVSD study population and, although not statistically significant, may determine recovery of post-LT LVSD within 30 days. Preoperative systolic wall motion abnormalities did not predict recovery of LVEF. However, the presence of preoperative wall motion abnormalities predicted development of post-LT LVSD and may be a precursor to cardiac remodeling within a chronically vasodilated vascular system. Within the post-LT systolic dysfunction group, there was no difference in clinical characteristics between those that recovered within 30 days and those that did not, which supports a spectrum of disease severity.

Future directions of study include evaluation for both systolic and diastolic LV dysfunction in post-LT patients and establishing postoperative TTE intervals in patients with LVSD. We also propose the use of advanced imaging techniques, including myocardial strain analysis, and measurement of 3-dimensional volumes with the use of TTE and cardiac magnetic resonance to further characterize who may be at higher risk to develop post-LT LVSD.

Limitations of this study include its retrospective single-center design, limited medication reconciliation data collection after discovery of post-LT LVSD, and absence of a post-LT surveillance protocol to reevaluate LVEF. Nonrandom performance of TTE in only 34% of post-LT patients represents selection bias based on clinician judgement and may underestimate subclinical incidence of systolic dysfunction. Further risk stratification in the post-LT LVSD group that did not show LVEF recovery was not collected and may limit interpretation of data.

Conclusion

In summary, patients who developed post-LT LVSD had more preoperative wall motion abnormalities and higher rates of preoperative low-normal LVEF, but these findings did not influence LVSD recovery. Survival was worse for patients that developed post-LT LVSD, although post-LT LVSD did not affect length of hospitalization. The pathophysiologic mechanism behind development of post-LT LVSD is likely multifactorial. Post-LT LVSD with recovery in ≤30 days likely represented stress-induced cardiomyopathy, whereas longer recovery times may represent an occult cirrhotic cardiomyopathy population. Although post-LT LVSD is an uncommon condition and no societal guidelines exist for nontraditional risk factor assessment in the pre-LT setting, newer recommendations and considerations have emerged to guide clinicians in preoperative risk stratification and risk reduction.[26] Early recognition of susceptible individuals will allow clinicians to carefully decipher transplant candidacy, medically optimize for surgery, and manage postoperative complications in a more effective manner, hopefully leading to reduction in the development of post-LT LVSD.

Disclosures

None.

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