Biventricular Assist Devices for Acute Heart Failure After Orthotopic Liver Transplantation

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SUMMARY
Background: Medical comorbidities augment surgical risk of liver transplantation. This is a report of immediate post-operative biventricular failure following liver transplant requiring venoarterial extracorporeal membrane oxygenation (VA-ECMO) and subsequent conversion to minimally invasive biventricular assist devices (BIVAD) for cardiac recovery and liver graft preservation.

Case Report: 66-year-old male decompensated alcoholic cirrhotic with pre-operative stress echocardiogram (ECHO) showing no significant valvular or coronary disease and a left ventricular ejection fraction (LVEF) of 65% underwent liver transplantation. Transesophageal echocardiogram at conclusion of case demonstrated a LVEF of 10% with biventricular dysfunction and severe mitral regurgitation requiring four pressors. VA-ECMO was initiated for temporary stabilization with subsequent transition to biventricular support using an Impella® 5.5 left ventricular device (VAD) via axillary artery graft and a Protek-Duo percutaneous right VAD via the right internal jugular vein, both placed peripherally through a minimally invasive approach. Serial echocardiograms showed recovery of myocardial function. BIVAD were removed on day 8 and day 13. Excellent liver function was maintained.

Conclusion: This is the first report of minimally invasive BIVAD used for acute cardiogenic shock after liver transplantation. A multidisciplinary team approach to prompt mechanical support ensured preservation of liver graft while allowing for cardiac recovery.

Abbreviations:
LT – Liver transplantation
TCM – Takotsubo cardiomyopathy
CAD – Coronary artery disease
ECMO – Extracorporeal membrane oxygenation
DLCO – Diffusing capacity of carbon monoxide
LVEF – Left ventricular ejection fraction
LV – Left ventricle
MELD-NA – Model for end-stage liver disease - sodium
ECHO – Echocardiogram
POD – Postoperative day
VA-ECMO – Venoarterial extracorporeal membrane oxygenation
LVAD – Left ventricular assist device
RVAD – Right ventricular assist device
TEE – Transesophageal echocardiogram
BIVAD – Biventricular assist device

Background
Liver transplantation (LT) is the optimal treatment for end stage liver disease. Comorbidities such as coronary artery disease (CAD), age >50, smoking, hypertension, hyperlipidemia, and diabetes can contribute to increased operative risk in patients with liver failure [1,2]. Cardiac disease is the leading cause of post LT mortality [1-4]. Additionally, the stress of LT can lead to transient reversible cardiopulmonary pathophysiologic changes such as increased systemic vascular resistance, decreased cardiac output, and increased pulmonary capillary wedge pressure resulting in increased myocardial stress and oxygen demands [2]. Anticipation and prompt management of these cardiopulmonary changes is important in management of post-operative LT patients.

Baseline cardiomyopathy is generally part of the pre-operative assessment in all LT patients, but specific algorithms vary by institution. Cardiac assessment can include a combination of: history, physical exam, electrocardiogram, transthoracic echocardiogram, functional testing, invasive coronary testing, and coronary computed tomography angiogram [1-4]. Despite comprehensive pre-operative testing, patients still can sustain adverse cardiac events during LT. For example, Takotsubo Cardiomyopathy (TCM) is reversible stress-induced cardiomyopathy in absence of CAD,
Case Report
A 66-year-old male, MELD-NA 31, was listed for LT due to alcoholic cirrhosis (abstinent from alcohol 11 years, tobacco 16 years). His past medical history included Crohn’s disease and hypertension. Dobutamine stress echocardiogram two months prior to LT demonstrated normal biventricular function, left ventricular ejection fraction (LVEF) of 65%, trace aortic insufficiency, trace mitral regurgitation (MR), trace tricuspid regurgitation (TR), and no evidence of inducible ischemia. The patient underwent LT from a brain-dead donor with standard anatomy and venovenous bypass per our standard institutional protocol. Intraoperative transfusions included: 7 units packed red blood cells, 7 units fresh frozen plasma, 1000 mL autologous transfusion via cell saver, 2000 mL crystalloids, and 500 mL 5% albumin. Induction immunosuppression was solumedrol and mycophenolate.

The patient tolerated reperfusion with minimal hemodynamic changes and only mild elevation of pulmonary artery pressures that improved with diuresis; however, his vasopressor requirement continued to increase during the neohepatic phase. Transesophageal echocardiogram (TEE) demonstrated a LVEF of 10% with biventricular dysfunction, severe MR and TR as compared to his normal TEE at the beginning of the operation. He required inotropic support with epinephrine in addition to vasopressor support with norepinephrine and vasopressin upon transfer to the intensive care unit. Over the next day, additional inotropic support with dobutamine was added to augment cardiac output; however, he continued to have a low cardiac index, elevated left-sided filling pressures, and low urine output.

After consultation with cardiac intensive care and surgery teams, the patient was urgently placed on mechanical support with VA-ECMO for temporary stabilization. Adequate systemic perfusion pressures were achieved with peripheral VA-ECMO; however, he developed LV distension leading to pulmonary edema, pulmonary hypertension, and worsening MR and right-sided failure risking both liver and cardiac function. Transesophageal echocardiogram (TEE) demonstrated normal biventricular function, left ventricular ejection fraction (LVEF) of 65%, trace aortic insufficiency, trace mitral regurgitation (MR), trace tricuspid regurgitation (TR), and no evidence of inducible ischemia. The patient underwent LT from a brain-dead donor with standard anatomy and venovenous bypass per our standard institutional protocol. Intraoperative transfusions included: 7 units packed red blood cells, 7 units fresh frozen plasma, 1000 mL autologous transfusion via cell saver, 2000 mL crystalloids, and 500 mL 5% albumin. Induction immunosuppression was solumedrol and mycophenolate.

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Over the next three days, hemodynamics improved considerably and the patient was weaned off vasopressor support. Close monitoring of liver function through serial ultrasounds and laboratory tests showed no evidence of graft dysfunction. No major bleeding occurred despite anticoagulation. Pulmonary edema and elevated central venous pressures were treated via diuresis with furosemide. Repeat transesophageal echo 5 and 7 days after liver transplant (days 3 & 5 on BIVAD) showed improvement in LVEF to 13% and 38% and decreased MR/TR. Given significant right ventricular recovery, the Protek Duo was removed at bedside 6 days after placement (day 8 from LT) with continued LVAD wean and extubation. Five days later the LVAD was successfully removed in the operating room (Figure 2).

The patient now maintains cardiac recovery along with a functioning liver graft and had resolution of his acute kidney injury. An outpatient left and right cardiac catheterization...
approximately 4 months after transplant revealed no significant CAD and normal LVEF.

Discussion
This report highlights a unique and successful management of a rare post LT phenomenon, non-ischemic stress cardiomyopathy (TCM). The prevalence of TCM causing transient acute left ventricular dysfunction is between 1-7% in post LT patients [8]. It is postulated that the history of alcohol use and afterload stress on left ventricular can contribute to left sided dysfunction [9]. However, the underlying preventable cause is not well defined as the pre-operative coronary angiogram in these patients is usually normal. Prompt recognition and supportive treatment of LV failure is important to decrease morbidity and mortality. Untreated left sided heart strain can progress to right-sided dysfunction leading to hepatic congestion and graft failure.

Acute biventricular failure is a rare finding post reperfusion. While outcomes of left sided heart failure post LT have been studied, little data exists to predict outcomes following biventricular failure [10,11]. The only report on BIVAD following organ transplantation is in the pediatric heart transplant population [12]. To our knowledge, this is the first report of BIVAD for acute cardiogenic shock without underlying CAD after LT. Prior case reports of RV or LV failure have been reported with right heart decompensation due to intracardiac thrombus or pulmonary hypertension and left heart failure due to undiagnosed coronary artery disease or underlying cardiomyopathy [13]. In addition, our case is the first to report the novel use of minimally invasive techniques for VAD placement after LT. Our approach decreased risk of post-operative bleeding and infection in an immunocompromised patient by avoiding traditional median sternotomy for temporary BIVAD placement. Our case describes successful management of TCM post-LT with mechanical support via ECMO and BIVAD. Prompt escalation of mechanical support allowed for preservation of function in the new liver graft as well as preventing impending renal failure.

Conclusion
Close intraoperative and post-operative cardiac monitoring with high index of suspicion for myocardial dysfunction in patients undergoing LT with steadily increasing vasopressor requirements is imperative. Prompt recognition of cardiac failure and initiation of cardiac support is necessary to optimize outcomes. A multidisciplinary collaborative approach with transplant and cardiac surgery, critical-care anesthesia, and the cardiac ICU team is required to ensure cardiac recovery and preservation of function of the newly transplanted liver.

References

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