Right Ventricular Failure Following LVAD Insertion: What's New in RV Support?
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Disclosures: This presenter has no financial relationships with commercial interests.

Stem Case and Key Questions Content
The patient is a 35-year-old male with a history of viral cardiomyopathy who presents for insertion of a HeartWare LVAD as a bridge to cardiac transplantation. He has no other medical issues. He is allergic to angiotensin converting enzyme inhibitors, which make him cough. Medications include metoprolol, losartan, amiodarone, and furosemide. His baseline vital signs are height 180 cm, weight 85 kg, HR 100 bpm, BP 90/45, respiratory rate 16 bpm, oxygen saturation on room air of 95%. He is awake but not talkative. His airway is Mallampati II with a thyromental distance of 6 cm, full dentition and cervical range of motion. His heart is regular and his lungs are clear. He has pitting edema in his lower extremities. His TEE revealed severe LV dysfunction with global hypokinesis and an LVEF of 15%, dilated mildly hypokinetic RV dysfunction, severe mitral regurgitation, trace tricuspid regurgitation, normal aortic valve and mild pulmonary hypertension. Baseline labs include: Na 128, K 3.4, Cl 108, CO2 40, BUN 26, creatinine 1.4, glucose 154, HCT 29, PT 16.1, PTT 45.8, INR 1.8, HCT 35, platelet 100,000.

1. What is a HeartWare and how does it work?

2. What concerns do you have about this patient?

3. What further information would you obtain?

4. What risk factors does this patient have for RV failure after LVAD placement?

The patient is brought into the OR and standard monitors and oxygen are placed. His vital signs are HR 98%, BP 92/40, oxygen saturation 100% on 6L face mask. Defibrillation pads and noninvasive near infrared spectroscopy (NIRS) cerebral oximetry pads are placed on the patient. Baseline cerebral oximetry readings are R 52, L 55.
5. What are your plans for invasive monitoring?

6. Where and when would you place them?

7. What is your plan for induction?
After premedication with 1 mg of midazolam, a 20 gauge left radial arterial catheter and a 16g peripheral IV are placed. After preoxygenation, 10 mg of etomidate, 150 ug of fentanyl, and 50mg of rocuronium are administered. After intubation, the patient’s vital signs are now HR 102, blood pressure 69/32, oxygen saturation of 100%.

8. How would you treat this blood pressure?
Epinephrine boluses of 20 ug and an infusion of 0.1 ug/kg/min are administered with an increase of blood pressure to 95/52. A 9.0F introducer with an SVO2/CCO pulmonary artery catheter and a 16g femoral arterial line are placed. The surgeon also asks for a 5F catheter to be placed in the right internal jugular vein. A transesophageal echo probe is inserted. Vitals are now BP 120/70, HR 105, CVP 18, PA 56/35, SVO2 56, and CO 2.9. The initial blood gas is 7.25/41/250/14/-6 HCT 28 K 3.5 and the baseline TEG is shown in Fig 1.

9. How would you optimize this patient prior to cardiopulmonary bypass?

10. What type of blood products should this patient receive?

11. What information from the TEE exam does the surgeon need to know before implanting an LVAD?
0.25ug/kg/min of milrinone is started. The chest is opened and the vitals are now BP 122/72, HR 100, CVP 15, PA 50/28, SVO2 63, and CO 3.5. Even with the inotropic support, the RV remains mildly hypokinetic with a TAPSE of 0.9 mm. The surgeon opens the pericardium and 25,000 units of intravenous heparin is administered. After 3 minutes, the activated clotting time is 295 sec. 10g of e-aminocaproic acid is administered and a 1g/h infusion started.

12. How would you treat the low ACT?
Two units of fresh frozen plasma and another 5000 units of heparin are administered. The repeat ACT is 421. Aortic and venous cannulas are placed and the patient is placed on cardiopulmonary bypass. The initial MAP is 52 mmHg and the cerebral oximetry is reading R 43, L 48. The initial blood gas on CPB is 7.32/56/395/16/-4, HCT 26, K 3.6, glucose 248.

13. How will you manage this patient while on CPB?
Five minutes later, the MAP is now 35 mmHg and the cerebral oximeters are reading R 21, L 24. The MAP does not increase despite boluses and infusions of phenylephrine, norepinephrine, and
vasopressin. At this time, the surgeon is implanting the left ventricular inflow cannula.

14. What is the mechanism of this hypotension and how would you treat it?
170mg of methylene blue is administered with an increase in MAP to 55. The surgeon finishes placing the aortic outflow cannula and is getting ready to deair the device. A heparinase TEG is performed and the results are in figure 2.

15. What is your plan for weaning from cardiopulmonary bypass?
0.1 ug/kg/min of epinephrine, 0.06 ug/kg/min of vasopressin, and 0.25 ug/kg/min of milrinone are started. The MAP is 54 mmHg. The pump is deaired, the patient is weaned off of CBP, and the HeartWare is started at 2400 rpm. The vital signs are now MAP 45 mmHg, HR 67, oxygen saturation of 100%, CVP 27, PA 45/24, SVO2 56, and CCO 3.1. The TEE shows this (Fig 3).

16. What is the cause of the hypotension?

17. What are the determinants of RV supply and demand?

18. How would you treat the hemodynamics?
The epinephrine was increased to 0.15 ug/kg/min, and the milrinone is discontinued. 0.15 ug/kg/min of norepinephrine and 40 ppm of nitric oxide are added. The surgeon places epicardial pacing wires on the right atrium and ventricle and the patient is paced at DDD at 98 beats per minute. Despite all of these interventions, the MAP is 40 mmHg, HR 98, oxygen saturation 100%, CVP 26, PA 39/20, SVO2 48, CCO 2.7.

19. What is the next step in the management of this patient?

20. What devices are available for short-term mechanical support of this patient?
The patient is placed back on CPB. A decision is made to implant a temporary TandemHeart into the patient.

21. What is a TandemHeart and how does it function?

22. How does the management of a patient with a biventricular assist device differ than that of a patient with an LVAD?
A wire is placed in the 5F IJ catheter inserted after induction and threaded into the pulmonary artery under fluoroscopy. An outflow cannula is inserted into the pulmonary artery and the inflow cannula is placed in the right atrium. The pump is started and the patient is weaned off of CPB. Vitals are MAP 100 mmHg, HR AV paced at 98 bpm, oxygen saturation 100%, CVP 10, PA 32/18, SVO2 70, CCO
8.5L/m.

23. How would you manage the patient’s hemodynamics?
The norepinephrine and vasopressin infusions and nitric oxide are weaned off, the epinephrine infusion is decreased to 0.025 ug/kg/min and a milrinone infusion at 0.25 ug/kg/min is started. The pacemaker is disconnected and the underlying HR is now 88 bpm. Vitals are now MAP 78, HR 88, oxygen saturation is 100%, CVP 8, PA 30/16, SVO2 75, CCO 6.1. The surgeon asks for protamine. After 250 mg of protamine is administered, the surgeon complains that there is no clot in the field. A coagulation panel is sent and the results are PT 18.9, PTT 52, INR 2.1, platelets 56,000. Arterial blood gas results 7.36/40/329/17/-1 HCT 25, K 4.0, glucose 136.

24. How would you treat this coagulopathy?
6 units of fresh frozen plasma, 8 units of cryoprecipitate, 3 units of packed red blood cells and 8 units of pooled platelets were administered. The bleeding was controlled, the chest closed and the patient brought to the ICU in stable condition.
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Model Discussion Content

The indications for LVAD placement include bridge to recovery or to a definitive procedure, bridge to transplantation, and destination therapy. The HeartWare is a centrifugal LVAD called the HVAD pump. The pump is implanted in the pericardial space and has only one moving part, the impella, making it more durable than the Heartmate II. When the impeller spins, blood flows across the surface of the blades, separating the impeller from the pump housing. The pump is attached to a driveline cable, which delivers power to the impeller. The HeartWare operates between 2400-3200 rpm and can generate a pump flow rate of up to 10L/min, but the maximum output that the pump can generate is dependent on the RV output. This pump has a flat pump head curve, which means that the pump can generate a wide range of flows for a small change in pressure. The advantage of the HeartWare over the Heartmate II, which is an axial pump, is that it has more reliable flow estimations from power and speed, it has a lower preload sensitivity, that prevents LV suck-down, and a higher afterload sensitivity and can pump against a higher SVR. It also can drop the pump speed when the pulsatility index, a measure of the LV contribution to pump filling, decreases, preventing suction events. The preoperative assessment includes the indication for the VAD, the ventricle(s) involved, and previous treatments including CIEDs, either a pacemaker, AICD or CRT, and surgical, including previous valve repair or replacements or coronary artery bypass grafting. Most of these patients arrive with an AICD and it is important to determine the type, how often and the last time the patient was shocked. The rest of the preoperative assessment focuses on evaluating RV function and determining the presence of any end-organ dysfunction, especially renal or hepatic dysfunction, which are
associated with altering the metabolism and excretion of drugs and increasing the incidence of post-bypass coagulopathy.

Right ventricular failure post VAD placement is associated with a higher incidence of failure to wean from CPB, increased organ dysfunction due to decreased perfusion, increased coagulopathy, and decreased rate of transplantation due to increased mortality. Planned biventricular assist device placement has less complications than LVAD implantation followed by RVAD implantation. The incidence of post LVAD RV failure is difficult to ascertain because there is no set definition for RV failure post LVAD and it has been reported to occur in approximately 20-50% of patients undergoing LVAD insertion.

Risk factors for post VAD RV failure include destination therapy, non-ischemic cardiomyopathy, reoperation, female sex, the presence of preoperative circulatory failure requiring vasopressors such as phenylephrine or vasopressin and antiarrhythmics, such as amiodarone, preoperative IABP, or preoperative cardiac arrest, the presence of end-organ dysfunction including ventilator support, elevated liver enzymes, including INR, AST, and total bilirubin, thrombocytopenia, elevated BUN or creatinine, or evidence of malnutrition such as hypoalbuminemia, the presence of severe RV systolic function manifested as a RVEDD > 35mm, RVEF 50 mm, and severe TR, and hemodynamic parameters, such as a CVP ≥ 15 mmHg, a CVP/PCWP ≥ 0.63, RVSWI 4.0 cm, either a tricuspid annuloplasty or a De Vega stitch may be placed. If there is > 2+ aortic regurgitation, the aortic valve may need to be oversewn or replaced. If the aortic valve is to be replaced, a bioprosthetic valve is chosen to prevent thromboembolism. Measurements of RV function that may indicate pre-existing RV failure include a TAPSE < 1.6mm, RV FAC < 35%, and an S’ < 10mm. TAPSE is a very specific but not a sensitive measure for RV failure post LVAD. Puwanant, et al, found that in these patients, a TAPSE of 43mm, an RV/LV diameter ratio of >.72%, and moderate severe to severe tricuspid regurgitation.

Prebypass management includes optimizing RV function to prevent RV failure after LVAD placement. This includes limiting intravenous fluids, preventing RV ischemia, and correcting any metabolic, hematologic, or electrolyte abnormalities including hyperglycemia, anemia, and hypokalemia. To prevent increases in pulmonary artery pressures, which may precipitate RV failure, hypoxia, hypercarbia and acidosis should be avoided. Since the RV is perfused in both systole and diastole, hypotension should be avoided. The optimal CVP is dependent on the extent of RHV but is kept between 10-16 mmHg. Fluid management is important. The failing RV is preload dependent but too much volume will increase RVEDP, stretch the TV annulus causing TR and ischemia. Blood products should be leukocyte reduced, especially if the patient becomes a candidate for transplantation. Patients with a previous sternotomy, should have blood available, defibrillator pads should be placed on the patient, and large bore IV access obtained. Defibrillator pads should also be placed if the patient has had an AICD that has been turned off. Patients who have undergone multiple procedures
and who have been exposed to heparin may be antithrombin III deficient and may require exogenous antithrombin III or FFP.

The goals of managing these patients on bypass are to remove as much excess volume as possible, either through the use of diuretics or ultrafiltration by the bypass pump, to correct coagulopathy, to treat electrolyte and glucose abnormalities, and to maintain an adequate MAP to perfuse the brain. Vasoplegia, may develop in patients with decompensated heart failure or in those taking ACE inhibitors or amiodarone and may require treatment with vasopressin, norepinephrine, or methylene blue, an inhibitor of NO. The dose of methylene blue is 2mg/kg boluses until a total of 10 mg/kg is reached. Near the end of CBP, an inotropic regimen to wean off of CPB should be initiated. A common regimen is a combination of epinephrine and milrinone infusions. Milrinone is effective in patients on beta-blockers or with down-regulated beta-receptors and has the added benefit of decreasing both pulmonary artery pressures and the dose of catecholamines. Other inotropic agents that have been used include dopamine, dobutamine, and levosimendin, a calcium sensitizer. Dobutamine has the advantage of increasing heart rate and decreasing PVR, both of which are advantageous in patients with RV dysfunction. If using an inotropic agent such as epinephrine or dopamine that does not have intrinsic pulmonary artery vasodilatory properties, pulmonary artery vasodilation with inhaled NO or prostacyclins, or intravenous nitroglycerin can be considered.

After the LVAD cannulas are placed and the pump is deaired, the patient is weaned off of CPB, volume is administered to prevent air entrainment, and the VAD is started, first at 2300 rpm and then slowly increased until the LV is decompressed and the interventricular septum is midline. The monitor measures the rotor speed in rpm, the power, which is constant unless there is a thrombus or low flow, and the pulsatility index (PI), which is a measure of the intrinsic contractility of the left ventricle and how much it is contributing to pump filling, and the flow.

The maximum cardiac output that the pump can produce is based on the maximal RV output. Once the LVAD is started, there will be a shifting of blood from the central circulation to the peripheral circulation, decreasing the RV afterload but increasing RV preload. Other factors that contribute to RV failure post VAD include pre-existing RV dysfunction, ischemia due to prolonged CPB time, increased RVEDP due to multiple transfusions, and alterations in RV geometry due to septal shifting from the LVAD, decreasing RV function. Besides maintaining RV contractility, it is important to maintain sinus rhythm. Any arrhythmias should be treated and bradycardia may require atrioventricular pacing. Postbypass echocardiography includes evaluating RV function and cannula placement, and assessing for residual MR, TR, aorta for dissections, and determining the position of the interventricular septum. The IVS is a guide to determining the presence of hypovolemia, RV function, and LVAD flow. For optimal function of the LVAD, the septum should be midline. A septal shift to the left with a dilated, hypokinetic RV is indicative of RV failure, and should be treated with increasing inotropic agents or mechanical support and a shift to the right is indicative of excessive LV volume and the VAD speed should be increased.
Based on the CVP and the PI, a Yale algorithm has been described to diagnose the etiology of low flows. In this algorithm, a normal CVP is between 10-12 mmHg and the PI is between 4-5. A CVP < 10 with a PI of < 3 is consistent with hypovolemia. A CVP 5.5 is consistent with hypertension, and a CVP > 12 and a PI < 2 is indicative of right ventricular failure.

Patients with hepatic congestion and renal dysfunction have a higher incidence of post bypass coagulopathy. The problem of administering large doses of blood products to patients with pre-existing RV function is that the barely compensating pre-bypass RV is now receiving added volume from the blood products that can precipitate RV failure. Closing the chest can further exacerbate RV failure. This can be treated with either higher doses of inotropic agents, pulmonary vasodilators, leaving the chest open for 24 hours, or a placing a temporary RVAD, such as the Impella, Tandem Heart, or Centrimag. If the RV does not recover, the only permanent FDA approved is the Thoratec PVAD and if there are any difficulties in oxygenation, the patient would require veno-venous ECMO. The Tandem Heart is an extracorporeal centrifugal pump with a magnetically driven impeller and a three-phase motor that can provide up to 4L/min of cardiac output and can be implanted either percutaneously or surgically for up to 14 days, allowing the native RV to recover. The inflow cannula is placed in the right atrium and the outflow cannula is placed in the pulmonary artery. The pump is less thrombogenic due to a fluid infusion system that lubricates the impeller and localized anticoagulation. (Fig 1.)

The Impella Recover RVAD is a small paracardiac pump, used to provide RV support for up to 10 days. The system consists of a microaxial pump, located in the right atrial inflow cannula and connected to the outflow cannula in the pulmonary artery by a flexible vascular prosthesis, a console, connected to the pump by a drive line, and a purger, that continuously delivers heparinized rinsing fluid to the pump, reducing the need for systemic anticoagulation. It has a maximal speed of 32,000 rpm and can deliver a flow rate of 5 L/min. (Fig 2.)

The Levotronix Centrimag is a centrifugal pump that consists of a pump, motor, console, a flow probe, and cannulae. The pump contains an impeller that is electromagnetically levitated to reduce friction in the pump, resulting in minimal heat and wear. The inflow cannula is placed in the right atrium and the outflow cannula is placed in the pulmonary artery. It is FDA approved for 30 days of right ventricular support and can be used as part of an ECMO circuit. The pump has a 31 ml priming volume, has a maximum pump speed of 5500 rpm and can generate up to 9.9 L/min of flow. (Fig 3.)

The Thoratec pVAD is a pulsatile VAD that can provide right, left, and biventricular support. It is a paracorporeal pump consisting of a polyurethane sac encased a rigid plastic case, surrounded by pneumatically driven pusher plates that are controlled by a console, which determines the pump rate and flow, driving pressure, % systole, and vacuum pressure. Blood is directed via unidirectional valves and it can deliver a cardiac output of 7 L/m. This VAD requires anticoagulation because the
pump does not eject until the volume in the polyurethane sac reaches 65 ml, leading to stasis and increasing the risk of stroke. (Fig 4.)
References
4. Catena E, et.al; Echocardiography and cardiac assist devices; Minerva Cardioangiol 2007 55(2) 247-65.