



The High Risk Recipient

Under Pressure: Pulmonary Hypertension in Heart Transplant Recipients

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Patients with chronic heart failure commonly develop pulmonary hypertension. This condition is due to direct backward transmission of elevated left atrial pressure to the lungs, as well as “reactive” pulmonary vasoconstriction. The exact time course for development of pulmonary hypertension in patients with heart failure is unknown, but it usually takes several years.

Once a diagnosis of pulmonary hypertension has been made in a potential heart transplant recipient, it is important to determine if the elevated pulmonary vascular resistance (PVR) is reversible or fixed. This determination is clinically important in that patients with fixed pulmonary hypertension are at high risk for acute right heart failure after transplantation. The normal donor right ventricle cannot adjust to the elevated and fixed afterload and is subject to failure. To assess the reversibility of PVR, pulmonary vasodilator agents such as sodium nitroprusside, adenosine, prostacyclin, or nitric oxide can be administered during right heart catheterization. Although the threshold hemodynamic values are not definitively known, it is generally accepted that patients with transpulmonary gradient (TPG) > 15 (TPG = mean pulmonary arterial pressure – post-capillary wedge pressure) or PVR > 4 wood units (PVR = TPG/Cardiac output) have fixed pulmonary hypertension.

Most transplant centers offer heart transplantation to patients who have reversible pulmonary hypertension. With improvements in intra-operative RV management, the incidence of acute right heart failure is low. Strategies to improve intra-operative RV function include: 1) Maximizing coronary perfusion by maintaining aortic pressure (ie. IABP), 2) Decreasing RV afterload (pulmonary vasodilator therapy, optimizing oxygenation, and acid-base status), and 3) Optimizing RV function (adequate preload, optimal inotropic support, and stable rhythm). Until recently, most candidates with fixed pulmonary hypertension were rejected for heart transplantation, or offered heart-lung transplantation in selected cases.

A promising and evolving alternative therapy for patients with fixed pulmonary hypertension is chronic pulmonary vasodilator therapy. Heart transplant candidates with fixed pulmonary hypertension have been treated with sildenafil for 2 months with a significant decline in PVR, which allowed successful heart transplantation. A randomized cross-over study of sildenafil in heart transplant candidates with fixed pulmonary hypertension is currently underway to better define the role of this class of agents.

Another promising strategy to treat patients with fixed pulmonary hypertension is to lower left atrial pressure (and lower PA pressure) by a left ventricular assist device (LVAD). Several groups have implanted pulsatile and nonpulsatile LVAD's in heart transplant candidates with fixed pulmonary hypertension and have shown a significant decline in PVR overtime, followed by successful heart transplantation.

Despite advances in peri-operative management of heart transplant candidates, fixed pulmonary hypertension remains a significant risk factor for early graft failure and is usually considered a contraindication to heart transplantation at many centers. Chronic pulmonary vasodilator therapy or implantation of LVAD are 2 promising strategies to lower PVR and allow these patients to undergo successful heart transplantation.

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