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Commentary

The Importance of Pulmonary Hypertension in Heart Transplant Patients

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DESCRIPTION

Due to a greater probability of recipient heart failure, fixed pulmonary hypertension is a contraindication for cardiac transplantation. To ensure safe cardiac transplantation, examination of left ventricular assist devices reduce fixed pulmonary hypertension in cardiac transplant candidates. A risk factor for both early and late mortality following heart transplantation is Pulmonary Hypertension (PH). The unacceptably high chance of abrupt right-sided donor heart failure occurring soon after implantation is the cause of this. There is widespread agreement that individuals with Pulmonary Vascular Resistance (PVR) higher than 2.5 Wood Units (WU), if PVR cannot be reduced by pharmacologic therapies (fixed PH), have an increased risk of mortality after receiving a heart transplant.

The ability of several pharmacologic treatments to lower PVR in these patients has been evaluated. Tested pharmaceuticals include levosimendan, phosphodiesterase inhibitors, urapidil, sodium nitroprusside, inhaled nitric oxide, phosphodiesterase inhibitors, and sodium nitroprusside. However, many of these individuals' PH cannot be considerably reduced even when various pharmacologic treatments are combined. Patients with end-stage heart failure with fixed severe PH cannot currently get a safe and effective therapy. In patients with advanced heart failure, PH has been shown to be decreased with Left Ventricular Assist Devices (LVADs). This is the first study to compare pulsatile and continuous flow LVADS and to conduct a prospective study on this subject. Due to a higher likelihood of postoperative recipient heart failure, severe fixed PH is an indication against cardiac transplantation. Candidates for cardiac transplantation who use LVADs have lower fixed PH and can get orthotopic heart transplantation. A frequent side effect of severe, chronic heart failure is pulmonary vascular hypertension. The percentage of people with PH who are qualified for cardiac transplantation and have terminal heart failure is around 72%. The complex process that leads to left ventricular failure is primarily responsible for the pathophysiology of PH in individuals with terminal heart failure. Increased post capillary pressure in the pulmonary circulation results from the left ventricular failure and the left atrial hypertension. By reducing the availability of nitric oxide and prostacyclin and increasing the synthesis of thromboxane A2 and endothelin-1, this worsens pulmonary endothelial dysfunction. In the sub endothelium, serine elastase activity is upregulated, resulting in the deposition of glycoproteins and the hypertrophy and hyperplasia of smooth muscle cells. The pulmonary vascular system is stimulated to contract by intermittent hypoxia. Platelet fibrin micro thrombi form as a result of variations in von Will brand factor expression. Additionally, this mechanism is linked to the hypertrophy and hyperplasia of smooth muscle cells. The pulmonary vascular tree undergoes modification depending on how long these processes persist. When high PVR cannot be considerably (>20%) lowered by pharmacological therapies, fixed PH is thought to be present. For the following reasons, most transplant centres will not give heart transplantation to patients with PVR higher than 3 to 4 WU, despite the fact that there is no international consensus. In the case of cardiac transplantation, an unusually large PVR impairs the right ventricle of the organ donor, which has relatively thin valves. This involves a significant risk of causing right ventricular failure by displacing the recipient's heart's right ventricle. High inotrope dosages in this situation are ineffective, and neither is mechanical support of the right ventricle. Both short- and long-term follow-ups show that post-transplant survival in patients with fixed PH is much poorer than in individuals with normal PVR.

LVADs reduced fixed PH in the current trial during the course of a 6-week support period. At least in part, higher left atrial filling pressures brought on by compromised left ventricular systolic performance are the cause of PH in patients with terminal heart failure. Theoretically, LVADs could work to stop this progression by continually emptying the left ventricle. If LVAD support causes a reverse-remodeling of the pulmonary vascular tree, this issue needs more research. In both pulsatile and continuous blood flow LVADs, PH reduced during the course of a 6-week support period. It was previously emphasized that continuous blood flow devices perform left ventricular unloading more effectively. The theoretical premise behind this was that continuous blood flow devices empty the left ventricle during the whole cardiac cycle. Based on this study, the kind of LVAD chosen for the cardiac transplant candidates with fixed PH

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should only be determined by the patient's requirements. The flow produced by continuous flow LVADs, particularly in big individuals, may be insufficient to improve exercise tolerance and facilitate patient recovery. The combined method of providing LVAD assistance and afterwards undergoing cardiac transplantation appears promising in comparison to the significant risk of donor heart failure in patients with fixed PH and orthotopic cardiac transplantation. In patients without PH, successful bridging rates have been reported to be between 65% and 70%. 69% of patients were successfully transitioned to secure cardiac transplantation. Following cardiac transplantation, post-transplant survival rates at 30-day (95%) and 12-month (95%) follow-ups were equivalent to those observed in patients without PH.

Patients with severe PH have previously been advised to consider heterotopic cardiac transplantation as an alternative to orthotopic cardiac transplantation as well as right ventricle-sparing transplant procedures. In these rare circumstances, the donor heart functions as the natural left ventricles or both ventricles' biological assist device. The reported survival rates at 12-month follow-ups varied from 83% to 59%. The lack of a suitable donor, technical challenges during implantation, and, in particular, late interactions between the donor and recipient hearts, are the main drawbacks of heterotopic transplantation. Comparing these alternative methods to the LVAD implantation and subsequent orthotopic heart transplantation, survival rates with these other methods are significantly lower. The provided method is linked with improved post-transplant survival and considerably lowers the incidence of right-sided heart failure to less than 1% following cardiac transplantation.

Nevertheless, because 51% of all patients showed a serious adverse event, patient morbidity while on LVAD support continues to be a significant issue. The most frequent occurrences were neurological ones, which also significantly increased patient death while receiving LVAD assistance. Blood loss and other problems included. There were no issues linked to the gadget. The frequency of unfavourable incidents in the series is comparable to what other people have described. Adverse occurrences were the same for pulsatile and continuous flow LVADs. To minimize LVAD-related issues, it is essential to conduct ongoing research, make improvements to devices, and choose patients with care. The nonrandomized design is the main disadvantage of this particular study. This approach was chosen since cardiac orthotopic transplantation is contraindicated in the presence of fixed PH. Therefore, it would be unethical to compare cardiac transplantation before and after LVAD installation. LVADs help cardiac transplant candidates who have fixed PH overcome it as a barrier to receiving a heart transplant. LVADs have to be taken into account in all cardiac transplant candidates with fixed PH.