had restrictive cardiomyopathy with cardiogenic shock and DCM secondary to cobalt poisoning. Classification of degree of heart failure prior to LVAD was done as per INTERMACS score and 7 patients were classified at INTERMACS category 2, 3 patients in INTERMACS category 3 and one in INTERMACS 4. 2 patients were on prior ECMO support and 8 were on inotropes prior to LVAD implant. 3 patients had high creatinine more than 2 and 3 had high bilirubin and altered LIVER function. In all patients ECHO derived RV function, RV stroke works index and pulmonary vascular resistance were measures carefully. RV function parameters include TAPSE, mean area change of RV and S' by tissue Doppler. PVR calculated by cardiac catheterization with cardiac output measured by thermodilution catheter.

Results: 9 patients were discharged successfully from the hospital. There was one hospital mortality due to sepsis and intracerebral hemorrhage. And another due to severe right heart failure and liver failure. All patients were put on oral anticoagulants (Warfarin). All the 9 discharged patients were assessed every 2 months post LVAD with pump interrogation, echocardiography and clinician's judgment of Quality of life (Minnesota QOLI) and PT/INR. All patients reported a good to excellent improvement. 7 patients are doing well with good pump parameters and no signs of heart failure after a minimum of 4 months and maximum of 3 years follow-up There was one late mortality 18 months post LVAD due to fulminant hepatic failure. There was no incidence of CVA or significant aortic leak.

Conclusion: LV assist devices can be safely implanted in terminally ill heart failure patients. Good patient selection, adequate patient optimization before implant and timing of intervention are the key to successful outcomes. Meticulous assessment and management of RV function and strict anticoagulation regimen plays a crucial role.

"Big Heart Syndrome": A not uncommon problem associated with pediatric heart transplantation

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Peadatric heart transplantation poses huge challenges especially in developing country like India. A common challenge for sick children with severe, end stage heart failure is paucity of donors of similar weight, because child heart donors are rare in India. Because of this problem some of the strategies employed by transplant teams is usage of oversized donors up to 2 times body weight and also utilizing ABO incompatible donors. Here we report our unique experience in 2 children aged 13 years (HK, weight = 37 kg) and 10 years (BJ, weight = 25 kg), both of whom needed oversized donors.

We report 2 children suffering from end stage heart failure due to dilated cardiomyopathy. Preoperatively, they were in INTERMACS Grade 3–4 CHF and on intermittant IV milrinone infusion. Creatinine levels 1.8–2 mg/dl and bilirubin levels 2 mg and 2.7 mg/dl. Cardiac cath showed PVR of 4 and 4.2 Wood units respectively. They were transplanted with donor hearts of 65 kg and 50 kg respectively. On the 2–3rd postoperative day, both patients had seizures (right focal in one and generalized in other) followed by reduced consciousness levels. BP was noted to be persistently above 150/90 in the first 2 days although they were on one inotrope (isoprenaline). The problem was recognized as Big Heart Syndrome and appropriate action take to reduce cerebral hyperperfusion and reactive vasoconstriction with BP lowering using small doses of nifedipine and NTG and slow tapering of inotrope. Both patients recovered completely over next 8–10 days.

Big Heart Syndrome, a well described entity in countries with large transplant programmes but not so commonly known in India. This syndrome has been described when donor hearts are oversized. This procedure is deemed suboptimal but becomes extremely essential when recipient is sick because of heart failure and same size donors are unavailable. This is a common problem in countries with *Inadequate Organ Sharing and Donation programmes* in children with end stage heart failure below body weight of 35 kg.

Modified immunosuppressive regime for heart transplant patients with preoperative renal dysfunction

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We studied 36 patients who underwent heart transplant in our center between 2011 and 2014 who had elevated creatinine more than 1. 8 mg/dl and creatinine clearance less than 40 ml/min and who were selected for a induction therapy using modified immunosuppressive regime with the CD 25 inhibitor basilixmab (simulect) – different from International protocols using antithymocyte globulin or OK T3.

Patient profile – Age range 10–66 years, weight range – 34–70 kg, creatinine range 1.8–2.6 mg/dl. All patients were NYHA class 3 and above, 24 were in NYHA class 4 (severe CHF) and 16 were in INTERMACS grade 3 and below.

Almost all had raised JVP with pedal edema, 20 had ascites and 26 had albumin less than 3 mg/dl.

LV EF 12–30%, 34 had moderate to severe RV dysfunction.

Cardiac cath done in all – RA mean = 18–24 mmHg, PA mean = 28–40 mmHg PVR range = 2.1–4.5 Wood units. A total of 5 patients had PVR more than 4 (labile with milrinone and nitric oxide).

All patients were put on IV milrinone infusion for 5–7 days before heart transplant which reduced RA mean by average of 8 mmHg, PVR by 2.3 WU, improved NYHA class by 1 grade and improved urine output and Appetite. A total of 2 patients were tested with inhaled NO with up to 18 mm fall in PA mean pressure.

Just before heart transplant, all patients received 10–20 mg of basiliximab (dose as per body weight and general condition) single dose. Post operatively in 26 patients creatinine levels normalized by day 5, allowing initiation of tacrolimus. In other 10 patients tacrolimus could be started only at day 10 with no ill effects or rejection episodes. In all patients CD 25 level was checked on day 3 and second dose of simulect given only of CD 25 more than 3%. Post op no patient had sustained neutropenia. Only 2 patients needed therapy for azotemia with post operative dialysis. A total of 1 patient died of severe RF and sepsis. Advantages of induction therapy using basiliximab will be discussed along with pathophysiology of azotemia in severe CHF and reversal of cardiorenal syndrome using milrinone.

Clinical significance of cysteine cathepsins in human dilated cardiomyopathy

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