Bridge-to-recovery strategy using extracorporeal membrane oxygenation for critical pulmonary hypertension complicated with cardiogenic shock

Meng-Ta Tsai, Chih-Hsin Hsu, Chwan-Yau Luo, Yu-Ning Hu and Jun-Neng Roan

INTRODUCTION

Pulmonary hypertension (PH) associated with right ventricular (RV) failure has a dismal prognosis [1]. Despite improving clinical outcomes [2], target therapies for PH are inadequate when RV decompensation or cardiopulmonary failure develops. Moreover, drug-related vasodilatory effects are intolerable during refractory cardiogenic shock or cardiopulmonary-cerebral resuscitation (CPCR) [3]. Consequently, extracorporeal life support (ECLS) is required to stabilize the haemodynamics and to provide a therapeutic window to optimize PH medications and RV function. Various types of ECLS, including right ventricular assist devices (RVADs) [4, 5], veno-arterial (VA) [6–10] or veno-venous (VV) [3, 8, 9, 11] extracorporeal membrane oxygenation (ECMO), and pumpless lung assist devices (LADs) [12–15], in acutely decompenated PH have been studied in recent years.

Current guidelines recommend that patients with an inadequate response to PH therapies be referred for lung transplantation. In the current lung allocation scoring system, PH patients may have low scores at listing, potentiating the prolonged waiting period. Therefore, mechanical support is important in the time between listing and transplant surgery [16]. The potential strategies using ECLS devices for decompenated PH patients include bridge-to-transplant (BTT) and bridge-to-recovery (BTR) approaches. While target PH therapies are optimized by increasing the titration of various medications in the BTR strategy [8], most centres rapidly taper off PH medications and catecholamines in BTT [7, 16].
Although BTT, using either ECMO or pumpless LAD, has been proved to be advantageous in reducing waiting-list mortality and improving post-transplant results [13, 17], these approaches are inevitably limited by donor availability. The BTR strategy, therefore, should be an option despite the perceived difficulty in weaning off of ECLS [6, 8, 9]. We report our experience in the management of critical PH and severe cardiogenic shock with VA ECMO as a BTR strategy. This study aimed to identify the factors determining the success of the BTR strategy and to evaluate the long-term outcomes of survivors.

MATERIALS AND METHODS

Patient characteristics

The ethics committee of our hospital approved this retrospective study and waived the need for informed consent. From 2009 to 2012, 7 consecutive PH patients required VA ECMO support for severe haemodynamic deterioration. The mean age of the patients was 47.6 ± 13.0 years, and there were 3 males and 4 females. The mean body weight was 58.4 ± 10.2 kg. The PH aetiologies included idiopathic pulmonary arterial hypertension (iPAH) in 3 patients, pulmonary fibrosis in 1 patient, systemic lupus erythematosus (SLE) in 2 patients and porto-PH in 1 patient. Of these patients, 3 were weaned off of ECMO and survived to discharge; these patients constituted the survivor group. The other 3 non-survivors included 2 patients for whom the weaning off of ECMO failed and resulted in death due to multiple organ failure and 1 patient who was weaned off of ECMO after 30 days of support but subsequently died of refractory ventricular tachycardia and fibrillation (VT/VF) 1 day after ECMO removal. The single patient who was bridged to a lung transplant after 7 days of VA ECMO support died due to surgical bleeding and was excluded. The baseline characteristics and adjustments to PH medications were compared between the survivor and non-survivor groups.

ECMO implantation and haemodynamic monitoring

VA ECMO implantation was considered when the cardiogenic shock was refractory to baseline PH medications with inotropic equivalent scores >50 (defined as μg/kg/min = dopamine + dobutamine + epinephrine × 100 + norepinephrine × 100 + isoproterenol × 100 + milrinone × 15) [18] or when cardiopulmonary collapse occurred. All of the patients received peripheral VA ECMO (Rotaflow centrifugal pump: Maquet, Hirrlingen, Germany; Hillite 7000 LT Oxygenator: Medos, Stolberg, Germany). The femoral artery was cut down at bedside under local anaesthesia or sedation when necessary. A 5 or 6 Fr. distal perfusion catheter was routinely introduced into the superficial femoral artery for distal perfusion. The ipsilateral or contralateral femoral vein was cannulated with the Seldinger technique. For patients with unfavourable femoral or iliac artery anatomies and in cases in which retrograde femoral perfusion led to cerebral or coronary hypoxaemia, right axillary artery cannulations via 8 mm side-arm grafts were used (1 of the 3 survivors). The ECMO flows were initially set to 40–50 ml/kg/min with comparable gas flows. Anticoagulation was achieved using Carmeda-coated systems (Carmeda BioActive Surface, Minneapolis, OH, USA) and heparin infusions to achieve activated clotting times of 180–220 s. The ECMO settings were adjusted to achieve mean arterial pressures (MAPs) >60 mmHg, central venous oxygen saturations (SvO₂) >65%, and adequate end-organ perfusions, as monitored by urine outputs, renal and hepatic biochemistry, and serum lactate levels. Serial systolic pulmonary artery pressures (sPAPs) and right heart morphologies were also monitored via echocardiography. The between-group differences in ECMO parameters, haemodynamic data and complications were analysed.

Bridge-to-recovery strategy during ECMO support

We preferred the strategy of primary recovery, rather than bridging for pulmonary transplantation, considering organ shortage. Our BTR strategy adopted an aggressive medical optimization of RV function during ECMO support, which included the identification and correction of predisposing factors for RV failure (e.g. infection, arrhythmia, anaemia or acute pulmonary embolism etc.), optimization of fluid status, inotropes to increase RV contractility, avoidance of RV ischaemia and individualized optimization of targeted PH therapy, including an up-titration and combination of multiple target PH therapies, such as inhaled nitric oxide (iNO), milrinone, iloprost, sildenafil or bosentan, according to their availability and patients’ tolerance for side-effects. A balance between tapering off inotropic agents and the up-titration of PH medications was important to minimize side-effects. Optimization of fluid status was achieved with diuretics or continuous renal replacement therapy according to serial central venous pressure measurement and the daily echocardiographic monitoring of the adequacy of RV decompression. The identified predisposing factors and adjustment of PH medications were compared between the two groups.

Statistical analyses

All continuous variables are presented as the mean ± the standard deviation and were examined with Student’s t-tests. Categorical variables were analysed with χ² tests. P-values <0.1 were considered statistically significant. SPSS 17.0.0 (SPSS, Inc., Chicago, IL, USA) for Windows was used for all statistical analyses.

RESULTS

Patient demographics

The mean age of the survival group was 52.67 ± 4.73 years, and the male-to-female ratio of this group was 2.1. The non-survival group was younger (40.67 ± 19.09) and entirely female. The body weight (kg) was 60.78 ± 13.35 for the survivors versus 53.90 ± 8.45 for the non-survivors, and the body surface area (m², Dubois) was comparable between the two groups (1.59 ± 0.17 vs 1.54 ± 0.10). The baseline creatinine level (mg/dl) was 2.12 ± 1.10 vs 0.90 ± 0.35. iPAH was present in 1 of the 3 patients in each group. The other aetiologies included pulmonary fibrosis and porto-PH in the survivors, and 2 cases of SLE in the non-survivors (Table 1). None of the patients had hypertension, diabetes or dyslipidaemia. All of the patients had one identifiable triggering factor for RV failure. Generally, the triggering factors (general anaesthesia, fluid overloading and anaemia) of the survivors seemed to be more quickly

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<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Gender</th>
<th>PH aetiology</th>
<th>Predisposing factor for RV failure</th>
<th>Transplant candidacy (contraindication)</th>
<th>sPAP baseline</th>
<th>PH therapy baseline</th>
<th>PH therapy before ECMO</th>
<th>PH therapy during ECMO</th>
<th>PH therapy post-ECMO explant</th>
<th>sPAP post-ECMO explant</th>
<th>ECMO days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Survivor 1</td>
<td>51</td>
<td>M</td>
<td>Pulmonary fibrosis</td>
<td>General anaesthesia(^a)</td>
<td>Yes</td>
<td>60</td>
<td>Iloprost</td>
<td>Iloprost</td>
<td>iNO milrinone Iloprost</td>
<td>Milrinone Iloprost sildenafil</td>
<td>Sildenafil</td>
<td>70</td>
</tr>
<tr>
<td>Survivor 2</td>
<td>58</td>
<td>M</td>
<td>iPAH</td>
<td>Fluid overload</td>
<td>No (renal failure)(^b)</td>
<td>X</td>
<td>Treatment-naive(^d)</td>
<td>Milrinone Iloprost</td>
<td>Sildenafil iNO milrinone Iloprost</td>
<td>sildenafil</td>
<td>Milrinone Iloprost sildenafil</td>
<td>Sildenafil</td>
</tr>
<tr>
<td>Survivor 3</td>
<td>49</td>
<td>F</td>
<td>Porto-pulmonary hypertension</td>
<td>Anaemia</td>
<td>No (cirrhosis)</td>
<td>X</td>
<td>Treatment-naive</td>
<td>Treatment-naive</td>
<td>Milrinone Iloprost</td>
<td>sildenafil</td>
<td>Milrinone Iloprost sildenafil</td>
<td>Sildenafil</td>
</tr>
<tr>
<td>Non-survivor 1</td>
<td>48</td>
<td>F</td>
<td>SLE CTEPH(^c)</td>
<td>SLE flare-up</td>
<td>Yes</td>
<td>97</td>
<td>Sildenafil iloprost</td>
<td>Sildenafil iloprost</td>
<td>Milrinone Iloprost</td>
<td>sildenafil</td>
<td>Milrinone Iloprost sildenafil</td>
<td>Sildenafil</td>
</tr>
<tr>
<td>Non-survivor 2</td>
<td>55</td>
<td>F</td>
<td>iPAH</td>
<td>Pneumonia</td>
<td>No (hypoxic encephalopathy)(^b)</td>
<td>X</td>
<td>Treatment-naive</td>
<td>Bosentan</td>
<td>Milrinone Iloprost</td>
<td>sildenafil</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Non-survivor 3</td>
<td>19</td>
<td>F</td>
<td>SLE</td>
<td>SLE flare-up</td>
<td>No (sepsis/leucopenia)(^b)</td>
<td>83</td>
<td>Sildenafil</td>
<td>Sildenafil milrinone Iloprost</td>
<td>sildenafil</td>
<td>X</td>
<td>X</td>
<td>16</td>
</tr>
</tbody>
</table>

\(^a\)For planned lung transplant: the operation was cancelled due to malignancy in the donor.  
\(^b\)Complications after ECMO or CPCR.  
\(^c\)Considered inoperable.  
\(^d\)without any PH medication.

sPAP: systolic pulmonary artery pressure; iPAH: idiopathic pulmonary arterial hypertension; SLE: systemic lupus erythematosus; CTEPH: chronic thromboembolic pulmonary hypertension; M: male; F: female.
correctable than the triggering factors of the non-survivors (pneumonia and SLE flare-up) (Table 1).

Peri-ECMO titration of pulmonary hypertension therapies

Two of the 3 survivors and 1 of the 3 non-survivors were first diagnosed with PH and were treatment-naïve for PH before this admission (Table 1). The other 3 patients received baseline PH therapies and were all maintained in New York Heart Association (NYHA) functional class III in the outpatient department for 2–3 years. Two patients (one from each group) were on the transplant waiting list. The other 4 patients were considered to be contraindicated for transplant either due to their underlying comorbidities or irreversible complications after ECMO (Table 1).

Generally, all of the patients received at least 3 types of targeted PH therapies to facilitate weaning off ECMO. The non-survivors tended to receive more PH medications prior to ECMO implantation. As a result, they had less reserve for additional PH therapies after ECMO (Table 1).

Haemodynamic data prior to and after ECMO

Given the profound cardiogenic shock with high inotropic-equivalent requirements and low PaO₂/FiO₂ (Table 2), intubation with ventilatory support was performed for all of the patients.

All of the non-survivors suffered cardiac arrest and received CPRC prior to ECMO implantation. All of the CPRC durations were within 30 min. In contrast, none of the 3 survivors received CPRC prior to the ECMO run (0 vs 100% in the survivors and non-survivors, respectively, P = 0.100; Table 2).

The non-survival group presented with a higher baseline sPAP than the survivors (127.67 ± 25.81 vs 67.67 ± 24.83 mmHg, respectively, P = 0.044; Table 2). The significantly higher pre-ECMO sPAP in the non-survival group decreased to 74.67 ± 33.50 mmHg on post-ECMO day 1 compared with 39.67 ± 0.58 mmHg in the survival group (P = 0.145, Table 2). The non-survivors also exhibited greater inotropic-equivalent requirements, lower MAPs, and higher lactate levels prior to ECMO implantation. These findings are indicative of the consequences of more profound cardiogenic shock and delayed ECMO intervention. However, the post-ECMO inotropic-equivalent doses, MAPs and PaO₂/FiO₂ improved to similar levels in both groups. The ScvO₂ (92.00 ± 4.58% vs 90.00 ± 2.83%) and serum lactate levels (3.87 ± 2.15 vs 4.40 ± 2.70 mmol/l) were not significantly different after the ECMO runs.

Complications of ECMO support and the outcomes

The differences in the incidences of ECMO-related complications were not significantly different due to the limited number of cases. However, the non-survivors were more likely to suffer from renal failure, hepatic failure or sepsis compared with the survivors (66.7 vs 0%, 33.3 vs 0% and 66.7 vs 0%, respectively; P = 0.400, 1.000 1.000 and 0.000, respectively), which were the main causes of mortality (Table 3). The implantation of the peripheral ECMO resulted in minor bleeding at the cannulation site in 1 patient in each group. All 3 survivors suffered from pneumonia, and one of them required a subsequent tracheostomy, which was possibly a result of intubation. The duration of ECMO support was 7.67 ± 4.93 days in the survivors. In contrast, the non-survivors expired after a mean duration of ECMO support of 19.33 ± 9.45 days (P = 0.131). Among the non-survivors,
one was successfully weaned off of ECMO on post-ECMO day 30 but expired 1 day after ECMO removal due to refractory VT/VF.

Outcomes of the survivors

The average hospital stay of the 3 patients was 62.33 ± 23.12 days. Survivor 1 received a lung transplantation on post-ECMO day 22. His sPAP was 70 mmHg before transplant (Table 1). He was discharged on post-transplant day 59 but died of sepsis and cytomegalovirus infection on Day 207.

Survivor 2 had well-controlled sPAP with sildenafil after ECMO explantation (Table 1). He was discharged on Day 65, but died of pneumonia and septic shock on Day 694.

Survivor 3 was regularly followed (for more than 4 years thus far) with an NYHA functional class II and sPAP of 50 mmHg (Table 1) after discharge. The distances of her 6-min walking tests were 253 m 1 week post-ECMO and 387 m 1 month post-ECMO.

DISCUSSION

This study summarized our experiences with peripheral VA ECMO using the BTR strategy for critical PH with cardiogenic shock. Four (66.7%) of the 6 patients survived to weaning off of ECMO and 3 (50%) survived to discharge. The major determinants of mortality were CPCR and high sPAPs prior to ECMO set-up. The factors triggering RV failure and baseline PH therapies also affected the success of BTR. A poor pre-ECMO haemodynamic status in the non-survivors indicated that a delay in ECMO intervention, which resulted in more pulmonary and end-organ complications, jeopardized recoveries and precluded their candidacy for lung transplant [19]. Pneumonia was not uncommon among the survivors and was the major cause of late death during the long-term follow-up.

The vasodilatory effects of PH therapies were typically intolerable in critical PH patients with RV failure and cardiogenic shock. Various types of ECLS have been used to stabilize the haemodynamic status, to offer a therapeutic window and to bridge these patients to definitive therapies. Forcing RV blood flow into the pressurized pulmonary circulation via an RVAD has not been shown to be effective [4, 5]. VA ECMO decompresses the preload of a distended RV and bypasses the high-pressure pulmonary vascular bed. It can be rapidly implanted peripherally and is effective in various degrees of RV decompensation with low cardiac output or CPCR [6, 9]. When a right-to-left shunt, such as patent foramen ovale or atrial septal defect, exists, VV ECMO partially bypasses the hypertensive pulmonary vascular bed with oxygenated blood and represents an alternative option [3, 8, 9, 11]. The novel pumpless LAD Novalung, which connects the pulmonary artery to the left atrium, efficiently reduces RV afterload but requires general anaesthesia and a sternotomy in the operation theatre [10]

The evidence supporting both VV ECMO and pumpless LAD as primary choices for decompressed critical PH is limited, bridging via initial VA ECMO has been well documented [12, 14, 15].

Because of the extreme donor shortage and long waiting times for lung transplants in Taiwan, mechanical supports, such as ECMO, play important roles in haemodynamically decompen-sated PH. However, the perceived difficulty in weaning these patients off of ECMO has led to an intended delay of ECMO intervention, which was clearly reflected by the higher inotropic equivalent than our regular threshold of ECMO intervention for cardiogenic shock (e.g. inotropic equivalent scores >40–50 µg/kg/min) and the disastrous pre-ECMO haemodynamic status in our non-survivors. The greater delay in the ECMO intervention caused more irreversible end-organ damage, which not only complicated patients’ recoveries but also precluded their transplant candidacy.

Although the post-ECMO haemodynamic data suggested that the circulatory support was comparable between both groups, the higher sPAPs of the non-survivors reflected their baseline PH severities and poorer responses to titrations of PH therapies, and medical treatment for RV failure. The survivor group received fewer types of PH medications prior to RV decompensation and had more readily correctable predisposing factors of RV failure. Thus, there were more choices for optimizing a target PH therapy, greater potential for RV recovery and an increased probability of weaning off ECMO. Therefore, prompt ECMO intervention, careful patient selection and more aggressive medical optimization of RV function during ECMO support, including identification and correction of triggering factors, optimization of fluid status, and individualized titration of target PH therapies and inotropes, offer opportunities for BTR rather than simply waiting for BTT [20].

Belohlavek et al. [6] reported 4 patients who received percutaneous VA ECMO for RV failure that resulted from PH. BTR strategies were applied to 3 of those 4 patients (1 idiopathic PH, 1 familiar PH and 1 ASD with Eisenmenger’s syndrome) (Table 4). Two of these 3 patients expired on post-ECMO days 12 and 13 due to multiple organ failure and bleeding complications. Only 1 patient survived ECMO decannulation after 16 days on ECMO. The patient survived 3 months after discharge until sudden cardiac death. Srivastava et al. [3] and Javidfar et al. [11] reported successful BTR with VV ECMO in PAH patients with PFO or ASD.

The New York Presbyterian group [21] described their algorithm for deciding between BTT and BTR strategies for PH patients who require ECMO support. Patients are considered BTR candidates when they are not eligible for organ transplant, and present with obviously reversible aetiologies due to RV decompensation, or suboptimal PH medications. Targeted PH therapies, including intravenous prostanooids, were escalated prior to ECMO weaning. Three of the 5 BTR patients (3 idiopathic and 2 with congenital heart disease) suffered cardiac arrest prior to the ECMO runs (Table 4). For a mean duration of 12 ± 7 days, 60% (3/5) survived on ECMO support. Three (60%) patients survived to discharge and 2 of these patients died of pneumonia within 3 months post-ECMO [8, 9, 21]. Except for the eligibility of organ transplant, their selection criteria for BTR are compatible with the conditions of our survivors, which highlight the importance of patient selection in the BTR strategy. However, how the underlying aetiologies of PH affect the response to BTR is uncertain. Both the SLE patients in our cohort died, but the effect of specific PH aetiologies on patient selection for BTR remains inconclusive from our limited experiences as well as the highly heterogeneous patient characteristics in previous studies (Table 4).

On the other hand, some reports considered lung transplantation an obligatory treatment for survival. Wiklund et al. [10] reported successful BTT after 12 days of VA ECMO support in a patient with porto-PH. BTT with VV ECMO in a patient with ASD and Eisenmenger’s syndrome has also been reported [11]. In 2010, Olsson et al. [7] demonstrated the feasibility of non-intubated awake ECMO support as a BTT strategy in PH cases with cardio-pulmonary failure. Three of the 5 cases reported by these authors survived to transplant after 18–35 days of awake VA ECMO support, which obviated the need for sedation and permitted further physiotherapy prior to lung transplant. None of these
patients experienced serious infections. In 2012, Fuehner et al. [17] reported superior postoperative and midterm results after lung transplant in an awake ECMO group compared with traditional mechanical ventilation. Using the BTT strategy, ECMO patients who required mechanical ventilation exhibited inferior long-term survival after bridging to transplantation. The evolving trend of non-intubated or extubated ambulatory strategies has also been applied to pumpless LAD for BTT [12, 13]. In contrast, experiences with these strategies are limited in BTR (Table 4). The potential benefits of these non-intubated or extubated ambulatory strategies on BTR are worth further investigation.

There was still a significant percentage of patients who died while waiting despite encouraging reports of successful BTT with ECLS [19]. The 14-year Toronto experience revealed that among 100 PH patients listed for lung transplantation, seven required ECLS bridging, 18 died on the waiting list and the mean duration on the waiting list was 197 days [1]. The mean waiting time for lung transplants in our hospital, however, was 386 days. The benefit of the BTT strategy is inevitably limited. With careful patient selection, aggressive targeted PH therapies [2, 22] and intensive care for RV failure [20], BTR still has a role in the management of severely decompensated PH patients. However, given the poor long-term results of survivors in the literature and our report, transplant should still be considered to be the definite therapy for PH patients who are successfully weaned from ECMO support using a BTR strategy.

Our study was limited by the small number of patients and its retrospective nature. However, these experiences provide valuable information on the selection of patients who are suitable for BTR. This strategy could be particularly important in countries where the waiting times for lung transplants are much longer.

Table 4: Bridge-to-recovery (BTR) strategies of ECMO support for PH patients

<table>
<thead>
<tr>
<th>Author/group</th>
<th>Age</th>
<th>Gender</th>
<th>PH classification</th>
<th>ECMO types</th>
<th>ECMO days</th>
<th>Non-intubation or extubation before weaning off ECMO</th>
<th>Follow-up period after discharge</th>
<th>Final outcome</th>
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<tr>
<td>Srivastava [3]</td>
<td>35</td>
<td>F</td>
<td>IPAH with PFO</td>
<td>VV</td>
<td>10</td>
<td>○</td>
<td>2 months</td>
<td>Subarachnoid haemorrhage during ECMO run</td>
</tr>
<tr>
<td>Javidfar [11]</td>
<td>41</td>
<td>F</td>
<td>ASD Eisenmenger</td>
<td>VV</td>
<td>23</td>
<td>×</td>
<td>4 months</td>
<td>Mortality after 4 m due to pneumonia</td>
</tr>
<tr>
<td>Belohlavek [6]</td>
<td>42</td>
<td>M</td>
<td>IPAH</td>
<td>VA</td>
<td>16</td>
<td>×</td>
<td>3 months</td>
<td>Mortality after 3 m due to sudden cardiac death</td>
</tr>
<tr>
<td>New York Presbyterian</td>
<td>20</td>
<td>F</td>
<td>VSD</td>
<td>VA</td>
<td>15</td>
<td>○</td>
<td>NA</td>
<td>In-hospital mortality</td>
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<tr>
<td></td>
<td>22</td>
<td>F</td>
<td>IPAH, TGA repaired, LPV stent</td>
<td>VA</td>
<td>8</td>
<td>○</td>
<td>21 months</td>
<td>Survival</td>
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<tr>
<td></td>
<td>48</td>
<td>F</td>
<td>IPAH</td>
<td>VA</td>
<td>6</td>
<td>×</td>
<td>2 months</td>
<td>Mortality due to pneumonia</td>
</tr>
<tr>
<td></td>
<td>41</td>
<td>F</td>
<td>APAH-CHD</td>
<td>VV</td>
<td>23</td>
<td>×</td>
<td>3 months</td>
<td>Mortality due to pneumonia</td>
</tr>
<tr>
<td></td>
<td>21</td>
<td>F</td>
<td>HPAH</td>
<td>VAV</td>
<td>10</td>
<td>×</td>
<td>NA</td>
<td>In-hospital mortality, ECMO withdrawn due to hypoxic brain injury</td>
</tr>
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</table>

IPAH: idiopathic pulmonary arterial hypertension; PFO: patent foramen ovale; ASD: atrial septal defect; VSD: ventricular septal defect; HPAH: hereditary pulmonary arterial hypertension; TGA: transposition of great arteries; LPV: left pulmonary vein; APAH-CHD: associated pulmonary arterial hypertension with congenital heart disease; VAV: venoarterial-venous.

*Using an upper body configuration with subclavian artery cannulation; M: male; F: female.

CONCLUSION

ECMO provides a therapeutic window for medically stabilizing critically decompensated PH patients. Given the extreme donor shortage, BTR is an acceptable alternative to BTT for critical PH patients who require ECMO support. Careful patient selection, prompt ECMO intervention prior to profound cardiogenic shock and CPCR, aggressive treatment of RV failure and titration of PH medications are the mainstays of a successful BTR strategy. High systolic pulmonary pressure could be an important predictor for mortality. Pneumonia is not uncommon and is the main cause of late mortality for those who survive to discharge.

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REFERENCES


