Heart transplantation in patients with extreme right ventricular failure

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Abstract

Objective: Donor shortage and improved medical treatment of heart failure increase the prevalence of patients with extreme right ventricular failure and refractory ascites to heart transplantation. The clinical outcome of heart transplantation in these patients has rarely been reported. Here, we sought to evaluate the clinical outcome of heart transplantation in patients with extreme right ventricular failure and refractory ascites.

Methods: Data were collected by retrospective chart review.

Results: Between 1993 and 2005, 12 patients with extreme right ventricular failure and refractory ascites underwent orthotopic heart transplantation at the authors’ hospital. The causes of heart failure were congenital heart disease in four patients, dilated cardiomyopathy in two patients, rheumatic heart disease in two patients, coronary artery disease in two patients, and restrictive cardiomyopathy and transplant coronary artery disease each in one patient. Eight of 12 patients had previous cardiac operation. The findings of preoperative abdominal sonography were massive ascites in all patients, congestive liver in 11 patients, and probably cardiac cirrhosis in 1 patient. One patient underwent combined heart and kidney transplantations. There were six in-hospital deaths: bleeding in three patients and multiple organ failure in three patients. Major postoperative complications occurred in 10 patients: renal failure requiring dialysis in 9, bleeding requiring reoperation in 8 patients. Patients with previous cardiac operation had a higher mortality rate (5/7 vs 1/5).

Conclusions: Heart transplantation in patients with extreme right ventricular failure and refractory ascites was associated with high mortality and morbidity. The presence of previous cardiac operation implied even poor prognosis.

Keywords: Heart transplantation; Right ventricular failure; Ascites

1. Introduction

Right ventricular failure and ascites occur as late complications of end-stage heart failure, and their development usually reduces patient survival [1—3]. In recent decades, survival after heart transplantation is gradually improving, and there is increasing patient risk profiles before transplantation. The increasing patient risk profiles include previous cardiac operations, mechanical assist devices, diabetes mellitus, critically ill recipients, high pulmonary vascular resistance, prior sensitization, long allograft ischemic times, and nonstandard or marginal donors [4]. Both donor shortage and improved medical treatment of severe heart failure will increase the prevalence of patients with extreme right ventricular failure to heart transplantation. Cardiac ascites and cirrhosis might develop in patients with a long history of right ventricular failure and systemic venous hypertension [5,6]. The clinical outcome of heart transplantation in patients with extreme right ventricular failure was reported infrequently in the literature [7]. Performing heart transplantation with cardiopulmonary bypass on these critically ill patients with cardiac cirrhosis has a low success rate [7]. Here, we sought to evaluate the clinical outcome of heart transplantation in patients with extremely right ventricular failure and refractory ascites.

2. Patients and methods

2.1. Patients

A total of 231 consecutive patients underwent heart transplantation from June 1993 through July 2005 at National Taiwan University Hospital. Twelve patients with extremely right ventricular failure and refractory ascites before heart transplantation were included in this study. Patients with high pulmonary vascular resistance (>6 Woods unit) at the initial evaluation would have re-assessment with pharmacological manipulation before accepting them as transplant candidates.

2.2. Definitions

The extreme right ventricular failure was defined as a combination of a persistent high right atrial pressure...
(>16 mmHg) and massive ascites, which was refractory to forced diuresis and required therapeutic paracentesis to relieve respiratory distress. The diagnosis of cardiac ascites was based on physical examination and findings of abdominal sonography. Patients with liver cirrhosis secondary to hepatitis virus infection or alcoholism were excluded. The diagnosis of cirrhosis was based on ultrasonographic findings, which was characterized by a coarsened heterogeneous echo pattern, increased parenchymal echogenicity, and nodularity of the liver surface [8]. Data on age, sex, diagnosis of heart disease, renal and liver function tests, hemodynamics, allograft ischemic time, and clinical outcome were recorded. Data of right atrial pressure, transpulmonary gradient, and pulmonary vascular resistance were derived from cardiac catheterization. Data of left ventricular ejection fraction were obtained from echocardiography, cardiac catheterization, or radioisotope angiography. Data of right ventricular ejection fraction were obtained from radionuclide angiography.

2.3. Heart transplantation

All of the procedures of heart transplantation were performed through a median sternotomy. The techniques of cardiopulmonary bypass were described previously [9]. In brief, cardiopulmonary bypass was instituted with an ascending aortic and separate caval venous cannulation. The operative techniques of heart transplantation in patients with prior cardiac operation were similar to that in patients receiving transplantation as a first cardiac procedure.

Postoperative management in intensive care unit was the same as that in routine patients receiving cardiac surgery. Blood components were given whenever needed and no aprotinin was used in our patients because of non-availability. Pulmonary artery pressure and cardiac output were continuously monitored. Patients with postoperative allograft dysfunction or right ventricular failure were treated with intravenous inotropes and inhaled nitric oxide. Extracorporeal membrane oxygenation was used in cases with refractory shock. Major postoperative complications were classified as neurological (consciousness disturbance, seizure, or stroke), pulmonary (prolonged ventilator support for more than 48 h), infectious (wound infection, bacteremia, pneumonia, or urinary tract infection), gastrointestinal, renal (acute renal failure or need of new dialysis), bleeding (profuse chest tube drainage in need of reoperation), and hepatic decompensation (hepatic function deterioration or hepatic failure).

2.4. Immunosuppression

All patients received triple-drug immunosuppressive therapy according to our heart transplantation protocol previously described [10,11]. Rabbit antithymocyte globulin (1.5—2.5 mg/kg/day) was given after transplantation for 5 days. Cyclosporine was started orally within 5 days after transplantation or after the recovery of renal function. Azathioprine was given at 1—2 mg/kg/day after transplantation, with the dose adjusted to maintain a white blood cell count 4000—6000/mm³. Prednisone (0.5 mg/kg/day) was started on the second postoperative day and tapered to 0.2 mg/kg/day by the first month after transplantation. Tacrolimus (FK-506) and mycophenolate mofetil (Cellcept) were used for recurrent rejection or severe adverse reactions to cyclosporine and azathioprine. Since 2004, we started to use mycophenolate mofetil for primary immunosuppression instead of azathioprine.

All patients were followed monthly at special cardiac transplantation clinic. Standard chest roentgenogram, blood tests, electrocardiogram, and physical examinations were routinely performed at regular intervals.

3. Results

3.1. Patient characteristics

From 1993 through 2005, 12 patients with extremely right ventricular failure and refractory ascites underwent orthotopic heart transplantation. There were eight men and four women, and the median age was 33 years (range, 12—58 years). The causes of heart failure were congenital heart disease in four patients, dilated cardiomyopathy in two patients, rheumatic heart disease in two patients, coronary artery disease in two patients, and restrictive cardiomyopathy and transplant coronary artery disease each in one patient. Patient demographics and laboratory data before transplantation are listed in Table 1. Eight of 12 patients (75%) had previous cardiac operation including 2 multiple valve replacements, 1 coronary artery bypass surgery, 1 total cavopulmonary connection, 1 Fontan operation, 1 Rastelli operation, 1 heart transplantation, and 1 Senning operation. The median level of serum total bilirubin was 1.37 mg/dl (range, 0.5—5.7); serum albumin, 3.2 g/dl (range, 1.4—4.2); serum blood urea nitrogen, 42 mg/dl (range, 21.7—79.1); serum creatinine, 1.8 mg/dl (range, 0.6—6.0); serum aspartate aminotransferase, 34.5 U/l (range, 16—81), and serum alanine aminotransferase, 15 U/l (range, 7—85). Three patients had the prothrombin time international ratio over 1.5. The findings of preoperative abdominal sonography were massive ascites in all patients, congestive liver in 11 patients and probably cardiac cirrhosis in 1 patient. No patient received liver biopsy. Before transplantation, three patients had mechanical support with extracorporeal membrane oxygenation, and three patients required dialysis treatment because of anuria.

Among donors, there were nine men and three women, and the median age was 24 years (range, 7—45 years). ABO blood types between donors and recipients were identical in eight cases and compatible in two cases. The body weight ratio between donors and recipients ranged from 0.72 to 1.50. The median duration of allograft ischemic time was 205.5 min (range, 95—260), and the median duration of cardiopulmonary bypass was 230 min (range, 72—426). All patients underwent orthotopic heart transplantation. One patient who had transplant coronary artery disease and severe renal impairment underwent combined heart and kidney transplantsations.

3.2. Clinical outcomes

As shown in Table 2, there were six in-hospital deaths (50%) occurring between 1 and 46 days after transplantation.
The causes of death were bleeding in three patients and sepsis with multiple organ failure in the other three patients. Two patients having complex congenital heart disease, single ventricle physiology and failed previous cardiac operations (one total cavopulmonary connection and one Fontan) died of surgical bleeding shortly after transplantation. A 39-year-old female having pulmonary atresia and previous Rastelli operation also died of surgical bleeding after transplantation. Patients with hospital death had more previous cardiac operation (five of six hospital deaths vs two of six survivors).

Major postoperative complications occurred in 10 patients: renal failure requiring dialysis in 9 patients, profuse postoperative bleeding requiring reoperation in 8 patients, hepatic decompensation in 3 patients, gastrointestinal complications in 3 patients, and sepsis in 3 patients. Among gastrointestinal complications, exploratory laparotomy for intra-abdominal bleeding was required in two patients because of multiple organ failure and severe coagulopathy. Another patient had gangrenous changes of gastrointestinal tracts found at autopsy because of disseminated intravascular coagulation.

As shown in Table 2, among the six hospital survivors, there was one late death. This patient died of heart failure 11 months after combined heart and kidney transplantation. Endomyocardial biopsy showed no evidence of acute cellular or humoral rejection. Two patients developed chronic renal failure and received regular hemodialysis at 27 and 91 months after transplantation. The other three patients were uneventful with follow-up durations of 1, 12, and 42 months.

Autopsy was performed in three patients with hospital death. Pathological examination of the livers in all three patients showed pictures of cardiac cirrhosis with centrilobular necrosis and varying degrees of fibrosis.

Table 1
Patient demographics and laboratory data before heart transplantation

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Diagnosis</th>
<th>UNOS status</th>
<th>Albumin (g/dl)</th>
<th>Total bilirubin (mg/dl)</th>
<th>AST (U/l)</th>
<th>ALT (U/l)</th>
<th>Serum creatinine (mg/dl)</th>
<th>PT INR</th>
<th>Previous cardiac operation</th>
<th>Preoperative dialysis</th>
<th>Preoperative ECMO</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>27</td>
<td>Rheumatic heart disease</td>
<td>1B</td>
<td>4.2</td>
<td>2.8</td>
<td>34</td>
<td>11</td>
<td>1.6</td>
<td>1.45</td>
<td>Aortic and mitral valve replacement</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>17</td>
<td>Complex congenital heart disease</td>
<td>1B</td>
<td>2.0</td>
<td>1.0</td>
<td>43</td>
<td>23</td>
<td>0.6</td>
<td>1.14</td>
<td>Total cavopulmonary connection</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>39</td>
<td>Tetralogy of Fallot</td>
<td>1A</td>
<td>3.2</td>
<td>1.2</td>
<td>31</td>
<td>12</td>
<td>2.0</td>
<td>1.08</td>
<td>Rastelli operation</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>30</td>
<td>Dilated cardiomyopathy</td>
<td>1A</td>
<td>2.5</td>
<td>1.5</td>
<td>58</td>
<td>30</td>
<td>0.7</td>
<td>2.2</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>17</td>
<td>Complex congenital heart disease</td>
<td>1B</td>
<td>2.5+</td>
<td>0.5</td>
<td>16</td>
<td>11</td>
<td>0.7</td>
<td>5.69</td>
<td>Fontan operation</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>54</td>
<td>Coronary artery disease</td>
<td>1B</td>
<td>1.4</td>
<td>0.9</td>
<td>21</td>
<td>23</td>
<td>2.5</td>
<td>1.06</td>
<td>Coronary artery bypass surgery</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>53</td>
<td>Coronary artery disease</td>
<td>1B</td>
<td>3.4</td>
<td>1.5</td>
<td>28</td>
<td>15</td>
<td>6.0</td>
<td>1.41</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>12</td>
<td>Restrictive cardiomyopathy</td>
<td>1B</td>
<td>2.75</td>
<td>1.24</td>
<td>68</td>
<td>22</td>
<td>0.49</td>
<td>1.17</td>
<td>None</td>
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<td>No</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>36</td>
<td>Rheumatic heart disease</td>
<td>1A</td>
<td>3.27</td>
<td>5.19</td>
<td>40</td>
<td>20</td>
<td>1.6</td>
<td>3.87</td>
<td>Heart transplantation</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>41</td>
<td>Transplant coronary artery disease</td>
<td>1A</td>
<td>2.4</td>
<td>2.1</td>
<td>12</td>
<td>7</td>
<td>3.5</td>
<td>1.24</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>12</td>
<td>Transposition of the great arteries</td>
<td>1B</td>
<td>3.4</td>
<td>0.8</td>
<td>39</td>
<td>15</td>
<td>0.5</td>
<td>1.36</td>
<td>Senning operation</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>58</td>
<td>Dilated cardiomyopathy</td>
<td>1A</td>
<td>3.7</td>
<td>5.17</td>
<td>81</td>
<td>85</td>
<td>3.4</td>
<td>1.80</td>
<td>None</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

AST = asparate aminotransferase; ALT = alanine aminotransferase; PT INR = prothrombin time international ratio; ECMO = extracorporeal membrane oxygenation.

Table 2
Hemodynamics, operative data, and clinical outcomes

<table>
<thead>
<tr>
<th>No.</th>
<th>RA pressure</th>
<th>TPG or PVR</th>
<th>LVEF (%)</th>
<th>RVEF (%)</th>
<th>Abdominal sonography</th>
<th>Allograft ischemic time (min)</th>
<th>Hospital outcome</th>
<th>Postoperative complications*</th>
<th>Late outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>30</td>
<td>3 WU</td>
<td>13</td>
<td>No data</td>
<td>Congestive liver</td>
<td>104</td>
<td>Alive</td>
<td>B</td>
<td>Alive 124 months</td>
</tr>
<tr>
<td>2</td>
<td>18</td>
<td>7 mmHg</td>
<td>15</td>
<td>No data</td>
<td>Congestive liver</td>
<td>240</td>
<td>Died bleeding</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>25</td>
<td>2 WU</td>
<td>51</td>
<td>31</td>
<td>Congestive liver</td>
<td>210</td>
<td>Died bleeding</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>25</td>
<td>1 WU</td>
<td>43</td>
<td>29</td>
<td>Congestive liver</td>
<td>221</td>
<td>Died sepsis</td>
<td>B, Gl, H, I, N, P, R</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>17</td>
<td>No data</td>
<td>25</td>
<td>8</td>
<td>Congestive liver</td>
<td>205</td>
<td>Died sepsis</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>16</td>
<td>2 WU</td>
<td>21</td>
<td>12</td>
<td>Congestive liver</td>
<td>117</td>
<td>Died sepsis</td>
<td>B, Gl, H, I, N, P, R</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>22</td>
<td>1 WU</td>
<td>13</td>
<td>28</td>
<td>Probably cirrhosis</td>
<td>95</td>
<td>Alive</td>
<td>B, H, R</td>
<td>Alive 58 months</td>
</tr>
<tr>
<td>8</td>
<td>21</td>
<td>18 mmHg</td>
<td>17</td>
<td>42</td>
<td>Congestive liver</td>
<td>260</td>
<td>Alive</td>
<td>None</td>
<td>Alive 42 months</td>
</tr>
<tr>
<td>9</td>
<td>21</td>
<td>4.7 WU</td>
<td>17.8</td>
<td>27.6</td>
<td>Congestive liver</td>
<td>206</td>
<td>Died sepsis</td>
<td>B, Gl, H, I, N, P, R</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>28</td>
<td>No data</td>
<td>39</td>
<td>No data</td>
<td>Congestive liver</td>
<td>172</td>
<td>Alive</td>
<td>R</td>
<td>Died 11 months Heart failure</td>
</tr>
<tr>
<td>11</td>
<td>23</td>
<td>No data</td>
<td>No data</td>
<td>No data</td>
<td>Congestive liver</td>
<td>118</td>
<td>Alive</td>
<td>R</td>
<td>Alive 12 months</td>
</tr>
<tr>
<td>12</td>
<td>23</td>
<td>2.3 WU</td>
<td>14.2</td>
<td>46.7</td>
<td>Congestive liver</td>
<td>220</td>
<td>Alive</td>
<td>None</td>
<td></td>
</tr>
</tbody>
</table>

RA = right atrial; TPG = transpulmonary gradient; PVR = pulmonary vascular resistance; LVEF = left ventricular ejection fraction; RVEF = right ventricular ejection fraction; WU = Woods units.

* Postoperative complications: B = profuse postoperative bleeding; Gl = gastrointestinal; H = hepatic; I = infectious; N = neurological; P = pulmonary; R = renal.
4. Discussion

4.1. Right ventricular failure

Peak oxygen consumption is the most commonly used prognostic parameter to determine the timing and suitability of heart transplantation in patients with end-stage heart failure [12]. The prognostic value of the right ventricular failure has been also demonstrated in end-stage heart failure leading to heart transplantation [1—3]. Right ventricular failure may result from primary insult to the right ventricle or from the inability of the right ventricle to properly function in the presence of prolonged increasing afterload. The occurrence of signs and symptoms of right ventricular failure (ascites and high right atrial pressure) is a well-known poor prognostic sign in patients with end-stage heart failure. In a previous study, patients with a right atrial pressure over 12 mmHg had a 47% 1-year survival as compared with the 68% survival for those with a right atrial pressure less than 12 mmHg [13]. In the current series, all patients had high right atrial pressure for long period of time. Half of patients were in critically UNOS 1A conditions and were expected to die soon if without urgent heart transplantation.

4.2. Cardiac cirrhosis

Elevated right atrial pressure results from right ventricular failure and is associated with a significantly increased risk of early death after heart transplantation [14]. Prolonged right ventricular failure and systemic venous hypertension will lead to cardiac cirrhosis. Severe bleeding and infection were usually the terminal events. Cardiac cirrhosis was found at autopsy in 75% of the early deaths of heart—lung transplant recipients with right ventricular failure and hyperbilirubinemia [15].

The spectrum of heart diseases affecting the liver includes three forms of clinical presentation: mild alterations of liver function tests, ischemic hepatitis, and congestive liver fibrosis or cardiac cirrhosis [5,6]. Hyperbilirubinemia occurs in one third of these patients, it increases with prolonged and repeated bouts of congestive heart failure [6]. Preoperative hepatic insufficiency with prolonged prothrombin time or elevated serum levels of liver enzymes was an independent predictor of early death after heart transplantation [16]. Cardiac cirrhosis is a clinically silent disorder characterized by a spectrum of morphological alterations in the liver ranging from mild deposition of sinusoidal collagen to emergence of broad fibrous septa [17,18]. Focal cirrhosis, incomplete cirrhosis, and complete cirrhosis are variants of cardiac cirrhosis. Laboratory tests have a little role in the diagnosis of cardiac cirrhosis. In the majority of patients with cardiac cirrhosis, serum levels of liver enzymes, bilirubin, and albumin are within the normal range [6]. Occurrence of cardiac ascites is the hallmark of cardiac cirrhosis. Empirically, cardiac ascites is treated with diuretics, along with paracentesis for refractory ascites. However, prolonged course of systemic venous hypertension and increasing use of diuretics lead to deteriorating renal and liver functions, and these patients sometimes are rejected as candidates for heart transplantation. In the current series, hypoalbuminemia was present in 9 of 12 patients and hyperbilirubinemia (serum total bilirubin >2.0 mg/dl) in 5 of 12 patients. None of our patients had ischemic hepatitis with a significant elevation of liver enzymes. The prolonged and critical conditions of our patients indicated that cardiac cirrhosis might exist silently. Although the findings of preoperative abdominal sonography in the majority of patients showed congestive liver, congestive liver fibrosis (cardiac cirrhosis) was confirmed in three patients on postmortem pathological examination.

4.3. Cardiac surgery in cirrhosis

The clinical outcome of cardiac surgery in patients with liver cirrhosis was reported infrequently [9,19—22]. Patients with mild liver cirrhosis can well tolerate cardiac surgical procedures. But major postoperative complications will develop in more than 80% of patients with advanced liver cirrhosis, and the in-hospital mortality rate will be up to 50—100% [19—22]. Major complications included infection, bleeding, and hepatic failure. Advanced liver cirrhosis was thought as an absolute contraindication to cardiac surgery with the use of cardiopulmonary bypass [19]. The surgical risks of heart transplantation are expected to be high in patients with severe heart failure and cardiac cirrhosis [7]. In the current series, the operative mortality rate was high (50%). Bleeding and sepsis accounted for all the causes of early death. Reoperation for bleeding was required in nearly 70% of patients. Our result of heart transplantation in patients with extremely right ventricular failure was similar to that of non-transplant cardiac surgery in patients with post-hepatitis or alcoholic liver cirrhosis. Before the conduct of heart transplantation, further detailed evaluation of liver reserve is necessary in patients with extremely right ventricular failure.

The diagnosis of alcoholic or post-hepatitis liver cirrhosis is easily made by clinical history and physical stigmata consistent with cirrhosis and findings of abdominal sonography. But the clinical picture of cardiac cirrhosis is usually masked by symptoms and signs of right ventricular failure. Ascites is a known manifestation of congestive heart failure and reflects longstanding systemic venous hypertension. Here, we recommend that when a heart transplant candidate has right ventricular failure and massive ascites, a liver needle biopsy be required to evaluate the presence and severity of cardiac cirrhosis.

4.4. Congenital heart disease

Neonatal palliation and staged reconstruction towards a Fontan type circulation is currently feasible in most patients with complex congenital heart diseases and single ventricle physiology. Acute or late Fontan failure represents a growing indication for heart transplantation. Heart transplantation can be performed successfully in patients with end-stage congenital heart disease after a Glenn or Fontan operation [23]. The survival was inferior in heart transplantation following failing Fontan than in heart transplantation following failing Glenn [24]. In the current series, two patients having complex congenital heart disease with single ventricle physiology and failed Fontan type operations died of surgical bleeding after transplantation. Heart transplan-
tation should be considered early in the course of failing Fontan or in high-risk patients with bidirectional Glenn shunt.

4.5. Study limitation

This study was limited by small case number and retrospective study. In addition, the duration of right ventricular failure was unknown and cardiac cirrhosis was not confirmed by liver biopsy. The benefits of inhaled nitric oxide and ventricular assist device were also not assessed. However, among our patients, only one patient had evidence of high pulmonary vascular resistance (Table 2). Inhaled nitric oxide is not considered to be beneficial in our patients. Besides, the implantation of left ventricular assist device was also associated with poor survival in patients with preoperative severe right ventricular failure [25]. Although our results were preliminary, this study is one of the largest series of heart transplantation in patients with extremely right ventricular failure. Patients should be carefully selected for heart transplantation, especially in patients with severe right ventricular failure, refractory ascites and previous cardiac operation. A prospective study with liver biopsy and scoring scale before transplantation are required for further evaluation. Liver biopsy remains a pivotal role in evaluation of the grade of inflammation and stage of cirrhosis before transplantation. The presence of extensive liver fibrosis would disqualify patients from transplantation and the only viable option for these patients with cardiac cirrhosis would be combined heart and liver transplantations.

4.6. Conclusions

Heart transplantation in patients with extreme right ventricular failure and refractory ascites was associated with high mortality and morbidity. The presence of previous cardiac operation implied even poor prognosis.

References