INTRODUCTION

Orthotopic heart transplantation has been performed for many years with excellent results and is still the "gold standard" in the management of end-stage heart failure in children. In addition to ABO and HLA compatibility, the size of the donor heart is one important criterion for organ allocation in pediatric heart transplantation. In particular, in children with dilated cardiomyopathy, oversized donor hearts in relation to patients' height and weight are often accepted. Razzouk et al. have defined oversized donor hearts as a donor-recipient weight ratio of >2.5 and Kanani et al. labeled a donor-recipient weight ratio of 3 or greater as an extreme mismatch.

In patients with a big donor-recipient ratio, a trend toward an increased frequency of right ventricular failure was found as well as an association with transient left lung lobar collapse. We report one case of a so far not described postoperative complication with a transient high-grade tricuspid regurgitation in a patient with extreme organ size mismatch.

CASE REPORT

Transient severe tricuspid regurgitation after transplantation of an extremely oversized donor heart in a child—Does size matter? A case report

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Abstract

In pediatric heart transplantation, the size of the donor organ is an important criterion for organ allocation. Oversized donor hearts are often accepted with good results, but some complications in relation to a high donor-recipient ratio have been described. Our patient was transplanted for progressive heart failure in dilated cardiomyopathy. The donor-to-recipient weight ratio was 3 (donor weight 65 kg, recipient weight 22 kg). The intra-operative echocardiography before chest closure showed excellent cardiac function, no tricuspid valve regurgitation, and a normal central venous pressure. After chest closure, central venous pressure increased substantially and echocardiography revealed a severe tricuspid insufficiency. As other reasons for right ventricular dysfunction, that is, myocardial ischemia, pulmonary hypertension, and rejection, were excluded, we assumed that the insufficiency was caused by an alteration of the right ventricular geometry. After 1 week, the valve insufficiency regressed to a minimal degree. In pediatric heart transplant patients with a high donor-recipient weight ratio, the outlined complication may occur. If other reasons for right ventricular heart failure can be ruled out, this entity is most likely caused by an acute and transient alteration of the right ventricular geometry that may disappear over time.

KEYWORDS
organ size mismatch, oversized heart, pediatric heart transplantation, tricuspid regurgitation

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Our patient was an 8-year-old girl with familiar dilated cardiomyopathy in the context of Carvajal syndrome—characterized by palmoplantar keratoderma, curly hair, and dilated cardiomyopathy. Her height was 129 cm and her weight 22 kg. Besides optimal heart failure medication, she needed continuous intravenous inotropic support including milrinone, dobutamine, and levosimendan. Repeat echocardiography revealed normal right ventricular systolic pressures (RVSP<30 mmHg) during the weeks before transplantation and pulmonary hypertension could be excluded based on this information. She was listed for progressive heart failure, and 4 months later, orthotopic heart transplantation was performed. The donor was 35 years old, his height 170 cm, his weight 65 kg. The donor-recipient weight ratio was 3, the donor-recipient body-surface-area ratio was two (Table 1). The overall ischemic time was 196 minutes only and there were no technical problems during organ harvesting or implantation.

An intra-operatively performed transesophageal echocardiography immediately after transplantation of the donor organ and before chest closure showed good left and right ventricular function, no tricuspid regurgitation, and normal levels of central venous pressure (5-8 mm Hg). During transplantation, the total fluid input was 795 mL and the total fluid output was 1300 mL. Therefore, primary chest closure was performed without hemodynamic compromise. No additional volume or change in catecholamine support was required for chest closure. The CVP increased acutely, and this change, however, was judged to be related to the maneuvers during transfer from theater to the pediatric cardiac intensive care unit. Postoperative central venous pressure—levels 1 hour after admission to the pediatric cardiac intensive care unit—rose up to 15-25 mm Hg, again no additional volume boluses were given nor changes in catecholamine support or sedation were performed. The monitoring of the central venous pressure curve showed a typical RV-pressure curve (Figure 1), while the central venous pressure line was placed securely within the superior vena cava. Repeat echocardiography revealed a severe tricuspid insufficiency despite an excellent right ventricular function (tricuspid annular plane systolic excursion minimal 18 mm) (Figure 2). Systolic pressure in the right ventricle estimated by echocardiography was not elevated. Tamponade, volume overload could be excluded (no edema, excellent urine output, no volume boluses, negative volume balance, etc.). The patient showed continuous sinus rhythm at a relatively slow rate of 65/min. To improve cardiac output, we initialized AV sequential pacing using various different AV intervals. As the application of inhalative NO as well as inhalative Ilomedine did not affect the severe tricuspid regurgitation, this therapy was stopped and the patient was extubated on day 2 after transplantation. Inotropic support (dobutamine max. 5 μg/kg/min, milrinone max. 0.5 μg/kg/ min) was continued for 5 days. Based on the investigations performed during this week and after other etiologies such as tamponade, acute rejection, reperfusion injury due to prolonged ischemia time, volume overload or right heart ischemia could be excluded, the alteration of the geometry of the right ventricle caused by the chest closure and perhaps direct compression of the right heart was thought to be causative. Severe tricuspid regurgitation persisted for 1 week, then rapidly decreased and was trivial at 10 days after transplantation (Figure 3).

The patient was discharged from pediatric cardiac intensive care unit on day 14 and from the hospital on day 30 after transplantation. The troponin level decreased rapidly after transplantation and was

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Recipient and donor body measurements</th>
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<tbody>
<tr>
<td></td>
<td>Age (years)</td>
</tr>
<tr>
<td>Recipient</td>
<td>8</td>
</tr>
<tr>
<td>Donor</td>
<td>35</td>
</tr>
<tr>
<td>Donor-recipient ratio</td>
<td>1.3</td>
</tr>
</tbody>
</table>

BMI, body mass index; BSA, body surface area.
in this cohort, one short-term problem after transplantation was a higher rate of postoperative ECMO support in a small subgroup of patients with an extreme donor-recipient mismatch of 3. However, again no overall difference in mortality, duration of intubation, and length of stay in ICU were observed. As this subgroup was very small (n=9), these results were not statistically conclusive.

Another short-term complication associated with high donor-to-recipient weight ratio was described by Fullerton already in 1992: In 27 of 69 patients with a D-R ratio >1.6, a transient lobar or complete lung collapse was observed. Despite this problem, the ventilation time and use of inotropic drugs were not elevated. The authors concluded that overall, large size mismatches appear to be very well tolerated in pediatric heart transplantation.

Based on the encouraging literature and facing a big shortage of donor hearts, our policy in pediatric heart transplantation consists in accepting oversized organs up to a donor–recipient mismatch of 3. In the present case, we report a complication which to our knowledge has not been described previously: Our patient developed a high-grade tricuspid insufficiency without other signs of RV dysfunction.

As intra-operative TEE post-transplantation but before chest closure still showed a completely competent tricuspid valve and a normal CVP, we interpreted that the insufficiency was due to an alteration of the geometry of the right ventricle caused by the chest closure and perhaps direct compression of the right heart. No signs of myocardial ischemia and no pulmonary hypertension as possible other causes of right ventricular dysfunction were detected. In the course of the following 10 days, valve insufficiency completely regressed as geometry of the ventricle turned back to normal. Prophylactic short time application of inhalative pulmonary vasodilators was performed but did not show any clinical effect in the context of normal pulmonary artery pressure. In our observation, left ventricular function was not affected by the problem described. This might be due to the fact that the left ventricular anatomy is different from the right, with a thicker muscular wall, a backward position in the chest, and a round form.

In pediatric heart transplant patients with a high donor-to-recipient weight ratio, this unusual complication may occur. If other reasons for right ventricular heart failure (ie, pulmonary hypertension, myocardial ischemia, acute rejection) are ruled out, this entity is most likely caused by an alteration of the right ventricular geometry and may disappear over time.

DISCLOSURE
The authors of this manuscript have no conflicts of interest to disclose as described by Pediatric Transplantation.

REFERENCES


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