A successful second living renal transplant in a child with severe mitral stenosis

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Introduction
Dramatic advancements in renal transplantation in children have been made possible by recent developments in transplantation techniques and intra- and postoperative management of the cardiovascular system [1,2]. Nevertheless, cardiovascular complications in low body weight children pose serious problems because successful grafting of the transplanted kidney depends upon adequate blood flow to the kidney.

We report a case of a successful partial renal transplantation for a child with mitral stenosis (MS).

Case report
An 8-year-old girl weighing 13 kg with chronic renal failure and severe MS was scheduled to have a second, ABO-incompatible, living renal transplant from her father. Her family history revealed that her elder sister died of renal and heart failure at the age of 6. The patient's first living renal transplant from her mother at age 6 was unsuccessful: no urine output was obtained from the immediate postoperative period, and cortical necrosis of the implanted kidney was confirmed 1 month postoperatively.

The present renal transplant, which followed 2 years after the first one, was planned with her father as the donor. Immediately prior to the renal transplant, under sedation, a percutaneous balloon valvectomy was performed for her MS, which reduced the left atrial pressure from 23 to 11 mmHg. In addition, a pulmonary artery catheter (Swan-Ganz catheter) was inserted to determine the most responsive vasoactive agent to administer and to estimate the limit of the fluid load. We needed to determine an effective vasoactive agent which caused minimal elevation of pulmonary arterial pressure (PAP) because high PAP causes pulmonary congestion and acute heart failure together with a decrease in renal blood flow. The results of the vasopressor loading tests are shown in Table 1. Metaraminol alone was found to elevate the blood pressure with minimal changes in the PAP.

The purpose of the fluid loading test was to determine the maximum amount of fluid allowable without causing pulmonary congestion. In our previous nine cases of renal transplantation for children weighing less than 15 kg, the rate of fluid loading sufficient to obtain urinary output was 20–25 ml·kg⁻¹·hr⁻¹. Based on this, the rate of 300 ml·hr⁻¹ for this child was considered to be sufficient. A mixture of saline and albumin was infused intravenously for an hour at the rate of 300 ml·hr⁻¹ during the fluid loading test. At this rate, we found no sign of pulmonary congestion.

The preoperative chest X-ray revealed an enlargement of the left atrium and hilar congestion. Blood gases while breathing room air were PaO₂ 60.7 mmHg, PaCO₂ 33.8 mmHg and pH 7.45.

Anesthesia was induced with thiopental, and maintained with 0.3%–1% isoflurane, nitrous oxide, oxygen, and a total of 200 μg fentanyl. A total of 6.5 mg vecuronium was used for muscle relaxation. Although blood, albumin and saline were infused at the rate of 260 ml·hr⁻¹ under the guidance of the PAP before declamping of the recipient's renal artery, the PAP unexpectedly and rapidly increased to 120/64 mmHg, and the blood pressure increased to 140/80 mmHg. Prostaglandin E₁ was administered at the rate of 0.02–0.1 μg·kg⁻¹·min⁻¹ to lower the PAP. The PAP decreased to 60/20 mmHg and the blood pressure dropped to 100/68 mmHg after declamping the recipient's renal artery.
Table 1. Results of vasopressor loading tests

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose (bolus)</th>
<th>Before loading PAP</th>
<th>After loading PAP</th>
<th>Before loading BP</th>
<th>After loading BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>0.13 mg</td>
<td>44/22</td>
<td>40/18</td>
<td>85/60</td>
<td>90/50</td>
</tr>
<tr>
<td>Dopamine</td>
<td>1.3 mg</td>
<td>44/19</td>
<td>58/20</td>
<td>102/57</td>
<td>137/73</td>
</tr>
<tr>
<td>Metaraminol</td>
<td>50 µg</td>
<td>44/22</td>
<td>54/22</td>
<td>85/60</td>
<td>150/85</td>
</tr>
<tr>
<td>Noradrenaline</td>
<td>25 µg</td>
<td>45/20</td>
<td>70/30</td>
<td>100/60</td>
<td>167/90</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>50 µg</td>
<td>48/22</td>
<td>50/20</td>
<td>110/60</td>
<td>130/70</td>
</tr>
<tr>
<td>Ephedrine</td>
<td>4 mg</td>
<td>46/22</td>
<td>68/27</td>
<td>110/65</td>
<td>120/68</td>
</tr>
</tbody>
</table>

PAP, pulmonary arterial pressure; BP, blood pressure.

Since the kidney to be transplanted was considered to exceed the child's cardiac loading capacity, one-third of the kidney was resected. The resulting warm ischemia time of the transplanted kidney was 5 min. The total ischemia time of the transplanted kidney (from donor's renal arterial clamping to the declamping of the recipient's renal artery) was 190 min. After transplantation, the systolic pressure decreased from 140 mmHg to 100 mmHg after declamping the recipient's renal artery, and metaraminol was infused intravenously at the rate of 0.5–2 µg·kg⁻¹·min⁻¹, and dopamine was subsequently infused intravenously at the rate of 3–5 µg·kg⁻¹·min⁻¹.

Urine output was confirmed 12 min after declamping the recipient's renal artery with 40 mg of furosemide, 100 ml of mannitol, and 10000 units of ulinastatin [3]. Subsequently, the urinary output decreased temporarily, which recovered by the next day. Perioperatively, the following immunosuppressive agents were administered [4,5]: 8 mg·kg⁻¹ cyclosporin, 2 mg·kg⁻¹ azathiopurine, 250 mg·kg⁻¹ methylprednisolone, 30 mg·kg⁻¹ prophylactic equine antilymphocyte globulin for 14 days, and 5 mg·kg⁻¹ deoxyspergualin for 5 days. The intraoperative total fluid balance was +11.9 ml·kg⁻¹·hr⁻¹. Pulmonary edema was not noted during the perioperative period.

Two years later, the grafted kidney was functioning at a serum creatinine level of 0.6 mg·dl⁻¹.

Discussion

To ensure the success of the second renal transplantation, we designed a thorough management plan of the cardiovascular function that emphasized the following three points: (1) preoperative management of MS with a dilatation of the mitral valve, (2) performance of a fluid loading test to simulate the declamping of the recipient's renal artery during the renal transplantation, and (3) selection of the most effective vasopressor after declamping the recipient's renal artery during the renal transplantation procedure.

Preoperative management of MS was indispensable for this operation. Open heart surgery, however, had to be ruled out considering the child's physical condition. A percutaneous dilation of the mitral valve was performed instead. The etiology of the mitral stenosis of this child was not of a congenital or infectious origin, but was believed to be strongly related to fibrosis at the root of the mitral valve due to repeated hemodialysis and uremia [6,7]. Insertion of a balloon percutaneously is minimally invasive, and was particularly effective in this case.

Assessment of the amount of water to be loaded was necessary to ensure adequate cardiac output and renal blood flow. After the management of mitral stenosis, we maintained the child in a good condition at the water loading of 300 ml·hr⁻¹ preoperatively. We assumed this value to be sufficient for the resumption of the renal blood flow based on our experiences described above. In her first transplantation, the rate of the fluid load was only 10.5 ml·kg⁻¹·hr⁻¹ because of her cardiac dysfunction caused by MS. This could have been one reason for the failure of the first transplantation. In her second transplantation, the rate was increased to approximately twice the previous value, and subsequently urine output was obtained. Preoperative dilatation of the mitral valve permitted the additional fluid loading.

The selection of the most effective vasopressor to be used after declamping the recipient's renal artery during the transplantation procedure was necessary to maintain adequate urinary output. During the first renal transplantation the blood pressure did not increase sufficiently despite the administration of dopamine. Thus, we tested the effect of the vasoactive drugs listed in Table 1 prior to her second operation, and found metaraminol to be the best. It is well known that metaraminol causes contraction of the peripheral blood vessels by stimulating selectively the α-receptors without affecting cardiac functions. Consequently, sufficient elevation of blood pressure with minimal elevation in PAP was expected by metaraminol even when dopamine was ineffective. Avoiding an increase in PAP was