Percutaneous transluminal coronary angioplasty of focal coronary lesions after cardiac transplantation

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Summary. Transplant coronary artery disease is the greatest impediment to long-term survival beyond the first year after cardiac transplantation. Transplant coronary artery disease shows a heterogeneous angiographic appearance, but focal stenoses can occur alone or at least predominate. Based on an angiographic indication 35 critical focal lesions causing narrowing by 75% or more were treated by PTCA during 23 procedures in seven patients 18-84 months after cardiac transplantation. Three patients each underwent only one procedure and four underwent repeated procedures [2, 3, 4 and 11, respectively]. Primary success was achieved without any complication in 35 of 35 lesions (100%). The mean degree of stenosis was reduced from 86±9% to 28±17% (P<0.001). The rate of restenosis was 18/29 (62%) at a mean of 4 months after angioplasty. Four patients are alive and free of adverse effects (symptoms, myocardial infarction, repeated percutaneous transluminal coronary angioplasty, retransplantation) 16±10 months after their last angioplasty. One patient underwent a successful second heart transplantation 26 months after the first angioplasty. Two patients died, 1 and 31 months after the last angioplasty. In conclusion, percutaneous transluminal coronary angioplasty can be performed safely with an excellent primary success rate in critical focal transplant coronary artery disease. The rate of restenosis is higher than in native coronary artery disease. Long-term follow-up depends on the individually variable accelerated nature of graft atherosclerosis.

Key words: Cardiac transplantation – Transplant coronary artery disease – PTCA

The myocardial function of the cardiac allograft is well preserved in the long-term course after transplantation, and the vast majority of recipients gain an almost normal quality of life [19, 21, 27]. However, the development of transplant coronary artery disease (TxCAD) is a serious and frustrating problem after transplantation and accounts for most deaths beyond the 1 year [8, 9, 11, 14, 21, 25, 27]. The prevalence of TxCAD is 40–50% by 5 years after transplantation [4, 16, 26, 27]. In our heart transplant population, the prevalence of critical stenoses with narrowing by 75% or more is about 16% 4 years after heart transplantation (HTX) [27]. TxCAD is considered to be a chronic vascular rejection leading to diffuse intimal injury and subsequent myointimal proliferation [1, 8–10, 14, 29]. This specific angiopathy may progress in excess of 10 times as rapidly as native coronary atherosclerosis [14], thus representing a specific form of accelerated atherosclerosis [9]. The histopathologic and angiographic appearance of TxCAD is not uniform [4, 11, 12, 18, 26, 27]. Diffuse concentric obliteration with rarification of the vascular tree due to marked intimal thickening or isolated or multiple focal lesions, comparable to native coronary atherosclerosis, may predominate [4, 11, 12, 18, 26, 27]. No effective preventive or therapeutic medical treatment of TxCAD is known [8, 10]. Retransplantation is associated with a markedly less favorable outcome than initial transplantation [5]. Recently, a first multicenter study of percutaneous transluminal coronary angioplasty (PTCA) in TxCAD has been published [6]. The present study reports our experience with PTCA of 35 critical focal lesions in seven heart transplant recipients.

Patients and methods

From August 1981 to February 1992, a total of 240 HTXs were performed in the Department of Cardiac Surgery, Großhadern Clinic, University of Munich. As a part of the surveillance program, all patients underwent annual cardiac catheterization including coronary arteriography. Early expe-
Table 1. Patient and lesion profile

- 7 male patients, mean age 45 ± 4 years (range 38-51 years)
- Indication for HTX:
  - Dilated CMP n = 6
  - Ischemic CMP n = 1
- Time from HTX to first PTCA: 43 ± 16 months
- Ejection fraction at time of first PTCA: 67 ± 8%
- 35 lesions ≥ 75% (17 primary lesions, 18 restenoses)
- 23 procedures (1 procedure n = 3; ≥ 1 procedure n = 4)
- Location:
  - LAD 18 (p 5, m 12, d 1)
  - Diagonal branch 2
  - Septal branch 1
  - CFX 1
  - RCA 13 (p 2, m 11)

CMP, cardiomyopathy; LAD, left anterior descending artery; CFX, circumflex artery; RCA, right coronary artery; p, proximal; m, mid; d, distal vessel segment; n, number of patients

Results

Primary results

The patients' clinical profiles, with ejection fraction and numbers and locations of lesions dilated, are listed in Table 1. Myocardial function in terms of ejection fraction was normal in each patient, with a mean ejection fraction of 67 ± 8%. Accordingly, no patient suffered from signs of heart failure. In PTCA patients compared to heart transplant recipients with normal coronary arteries similar findings were obtained for total cholesterol levels (265 ± 58 mg/dl vs 249 ± 53 mg/dl; difference not significant by Student's t-test for unpaired samples) and mean aortic pressure under antihypertensive therapy (108 ± 17 mmHg vs 112 ± 15 mmHg, n.s.). No patient suffered from diabetes mellitus. All were nonsmokers. Results of PTCA are listed in Table 2. In the seven heart transplant recipients 35 critical focal stenoses causing 75% narrowing or more were dilated during 23 procedures. The 35 lesions included 17 that were primary lesions, while 18 were restenoses. Three patients each underwent only one procedure, while four patients underwent repeated procedures (2, 3, 4 and 11). In 1 primary lesion angioplasty was combined with directional atherectomy. Additional coronary lesions in non-PTCA segments were present in 18 of 23 procedures (78%). Primary success, i.e., post-PTCA stenosis 50% or better [20], was achieved with no complications in all 35 lesions (100%); see Table 2. A typical example is shown in Fig. 1. The mean degree of stenosis was reduced from 86 ± 9% to 28 ± 17% (P < 0.001, Student's t-test for paired samples). Angina-like pain during angioplasty was reported by two patients, one of whom had his first PTCA 84 months after HTX, while the other was undergoing his 11th PTCA after 10 painless procedures previously.

Table 2. Primary results of PTCA in TxCAD, early and late follow-up

<table>
<thead>
<tr>
<th>Follow-up</th>
<th>Early</th>
<th>Late</th>
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<tbody>
<tr>
<td>Number of lesions</td>
<td>29</td>
<td>8</td>
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<tr>
<td>Time since PTCA (months)</td>
<td>3.9 ± 2.4</td>
<td>23 ± 4</td>
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<tr>
<td>Mean % stenosis before PTCA</td>
<td>71 ± 25%</td>
<td>55 ± 25%</td>
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<tr>
<td>Mean % stenosis, no restenosis</td>
<td>43 ± 13%</td>
<td>38 ± 11</td>
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<tr>
<td>Mean % stenosis, restenosis present</td>
<td>88 ± 9%</td>
<td>83 ± 10</td>
</tr>
<tr>
<td>Rate of restenosis</td>
<td>18/29 (62%)</td>
<td>3/8 (38%)</td>
</tr>
<tr>
<td>Patients with restenosis</td>
<td>3/7 (43%)</td>
<td>2/5 (40%)</td>
</tr>
<tr>
<td>Progression in non-PTCA-segments</td>
<td>21/29 (72%)</td>
<td>5/8 (63%)</td>
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n, No. of lesions