**Case reports**

*A commentary on the following three case reports appears on page 1016.*

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**Successful stenting of a pulmonary arterial stenosis after a single lung transplant**

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**Abstract**

A 44 year old patient with end stage idiopathic pulmonary fibrosis underwent single left lung transplantation. A perfusion lung scan performed 13 days after transplantation revealed deficient perfusion in the transplanted lung. Pulmonary angiography showed severe pulmonary artery anastomotic stenosis. Percutaneous insertion of a balloon expandable stent improved both pulmonary perfusion and respiratory function. (Thorax 1995;50:1011-1012)

Keywords: lung transplantation, pulmonary artery stenosis, stent.

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Pulmonary arterial anastomotic stenosis is rarely reported following lung transplantation.1,2 The diagnosis may be suspected in the presence of persistent pulmonary hypertension or from a lung perfusion scan, and confirmed by pulmonary angiography. Surgical treatment of this stenosis is usually required.2,3 We report the case of a patient in whom a single lung transplant was performed who developed severe pulmonary arterial anastomotic stenosis suspected after a lung perfusion scan and confirmed by pulmonary angiography. He was not well enough to undergo any further surgery, so perfusion was restored with a balloon expandable stent.

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**Case report**

A 44 year old man noted the onset of exertion dyspnoea in the middle of 1992. The first arterial blood gas analysis with the patient breathing air showed hypoxaemia (Po2 9.4 kPa; PCO2 4.8 kPa; pH 7.41). Investigations revealed a restrictive defect with forced expiratory volume in one second (FEV1) 2.57 l (68% predicted), forced vital capacity (FVC) 3.24 l (69% predicted), FEV1/FVC 79%, carbon monoxide transfer factor (TLCo) 75% predicted. These changes were attributed to interstitial fibrosis confirmed by chest radiography and a thoracic computed tomographic (CT) scan. No cause for the interstitial fibrosis could be found from the occupational and drug history and various investigations including open lung biopsy. Treatment with prednisolone and cyclophosphamide was started.

Despite treatment, the patient’s pulmonary status quickly worsened. A transtracheal catheter was inserted to administer 5 l/min of oxygen. An increase in the dose of corticosteroids had no effect on the impaired lung function. Blood gas measurements at that time were: Po2 9 kPa (on 5 litres O2); PCO2 5.7 kPa; FEV1, 1.79 l (47% predicted); FVC, 1.99 l (43% predicted), FEV1/FVC 88%. A lung transplant became an obvious necessity and a single left lung transplant was carried out.3

The postoperative period was marked by an immediate phrenic palsy which necessitated mechanical ventilation for more than 15 days. Despite the palsy having totally subsided, as shown by fluoroscopy, the patient could not be weaned off mechanical ventilation and he remained mildly hypoxic and breathless despite endotracheal oxygen. A scintigraphic perfusion scan showed hypoperfusion of the transplanted lung which was only receiving 28% of the pulmonary flow, and overperfusion of the native lung. Pulmonary angiography revealed an anastomotic stenosis associated with kinking of the donor pulmonary artery (fig 1). Pressure measurement showed a trans-stenotic gradient of 40 mm Hg (prestenotic systolic pressure 60 mm Hg, post-stenotic systolic pressure 20 mm Hg).

It was decided to treat this stenosis percutaneously. At first, an attempt was made to dilate it by means of balloon angioplasty using a 12 mm balloon (Medi-Tech, Boston Scientific Corporation, Watertown, Massachusetts, USA). However, there was no improvement in either the angiographic morphology or the trans-stenotic pressure gradient. A Strecker bal-
Arterial blood gases before and after pulmonary artery stenting

<table>
<thead>
<tr>
<th>Days</th>
<th>$D_1$</th>
<th>$D_2$</th>
<th>$D_3$</th>
<th>$D_4$</th>
<th>$D_5$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_{aO_2}$ (kPa)</td>
<td>9.79</td>
<td>10.49</td>
<td>11.57</td>
<td>15.65</td>
<td>9.35</td>
</tr>
<tr>
<td>$F_{iO_2}$ (%)</td>
<td>40</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>$P_{aco_2}$ (kPa)</td>
<td>23</td>
<td>32.6</td>
<td>35</td>
<td>53.2</td>
<td>—</td>
</tr>
<tr>
<td>Perfusion (%)</td>
<td>3.9</td>
<td>3.9</td>
<td>2.7</td>
<td>2.7</td>
<td>5.47</td>
</tr>
</tbody>
</table>

$D_1$ = day stenting was performed; $D_2$ = one day after stenting; $D_3$ = patient breathing spontaneously without any intratracheal oxygen.

* Before stenting.
** After stenting.
† $F_{iO_2}$ delivered to patient under mechanical ventilation.
‡ Perfusion of transplanted lung on perfusion scan. Results are given in percentage of perfusion of both lungs.

Discussion

Single lung transplantation is an effective treatment for end stage pulmonary fibrosis, although the postoperative course may be difficult.1 Pulmonary artery anastomosis stenosis is a rare but serious complication defined as a minimal anastomotic diameter of less than 75% of that of the neighbouring vessels.1 The stenosis may be worsened by kinking of the donor pulmonary artery distal to the anastomosis. It can be suspected from persistent pulmonary hypertension in the early post-operative period.1 An anastomotic malfunction should also be sought when perfusion to the transplanted lung is less than 50%.2 Transoesophageal echocardiography is useful in the detection of stenosis,1 but pulmonary angiography remains the preferred method for examination of the pulmonary arterial morphology.1,2

Various techniques can be used to restore lung perfusion. In congenital or postoperative branch pulmonary stenosis, balloon angioplasty is an effective and safe method of treating the stenosis, and thereby decreasing the systolic trans-stenotic pressure gradient.3 Balloon expandable stent implantation in pulmonary arteries has been used with success in dogs,4 pigs,5 and humans.6 In the latter, a self expandable stent was inserted after unsuccessful balloon angioplasty into a woman with life threatening congenital pulmonary artery stenosis. The stent remained open, becoming covered with neointima in the medium term.3

In our patient an arterial stenosis was suspected because hypoxaemia persisted, despite complete recovery from the pneumonic palsy, and because there was pronounced hypoperfusion of the transplanted lung.2 The need for surgical treatment was obvious but, because of prolonged arterial clamping and bronchial devascularisation, there was a high risk of bronchial and graft necrosis. Perfusion techniques were therefore preferred.

Failure to dilate a pulmonary artery stenosis with a balloon has been described, and is due to excessive elasticity of the artery when stretched.7 Although our balloon had a smaller diameter than the stent, there was no waisting impression on its surface while it was totally occluded. In contrast, our balloon expandable stent was opened up very easily. This, in our view, suggests kinking of the artery.

To our knowledge this is the first report of such a procedure in a patient undergoing transplantation. Although the long term outcome in our patient remains uncertain, the percutaneous insertion of a stent restored adequate perfusion of the transplanted lung, thus avoiding the risks of further surgery.

References