TECHNIQUE

Right ventricular outflow stent implantation: an alternative to palliative surgical relief of infundibular pulmonary stenosis

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Abstract

Objective—Preliminary assessment of the use of stents for palliative relief of right ventricular infundibular stenosis as an alternative to palliative surgical ventricular outflow enlargement.

Design—Descriptive clinical study.

Patients—Four patients with right ventricular outflow obstruction, aged between 2 and 15 years. One had had previous palliative surgery for pulmonary atresia, one had hypoplastic pulmonary arteries after palliative surgery for tetralogy of Fallot, one had multiple congenital abnormalities, and one had hypertrophic cardiomyopathy.

Setting—Tertiary paediatric cardiac centre.

Methods—After initial echocardiographic diagnosis the extent of right ventricular outflow obstruction was assessed by angiography. Balloon expandable stainless steel stents (Johnson & Johnson) were deployed in the right ventricular infundibulum.

Main outcome measures—Improvement in right ventricular outflow assessed by ventriculography and change in right ventricular/left ventricular pressure ratio, change in systemic oxygen saturation, freedom from arrhythmias, and sustained improvement in echocardiographic indices of obstruction.

Results—Mean right to left ventricular pressure ratio fell from 0.95 to 0.35 in the three patients with intact ventricular septum. Oxygen saturation increased from 76% to 91% in the patient with tetralogy. No arrhythmias were detected. Improvement was maintained at mean follow up of 9.7 months in three cases, but one patient required stent enlargement 17 months later because of neoendothelial proliferation within the stent.

Conclusion—Stent implantation provides an effective alternative to palliative surgical enlargement of the right ventricular infundibulum. Neoendothelial proliferation causes reduction in lumen in some cases, but this may respond to redilatation.

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Keywords: infundibular pulmonary stenosis; stents

Surgical relief of infundibular pulmonary stenosis is associated with very low risk of mortality and morbidity. Reoperation is rarely required and surgery remains unchallenged as the treatment of choice in patients with isolated right ventricular outflow obstruction. Enlargement of the right ventricular outflow tract also has a clear place in the palliative treatment of infundibular pulmonary stenosis when it is associated with other major anomalies, and it is in such rare situations that a non-surgical form of palliative treatment would be desirable. Stent implantation has been used successfully to relieve obstruction at a variety of intravascular sites but there is little available information on the use of stents in muscular obstruction of the right ventricular infundibulum. This report describes our preliminary experience with infundibular stenting in four patients who required palliative enlargement of the right ventricular infundibulum.

Patients and methods

PATIENTS

The patients were aged between two and 15 (median 10) years and their weights ranged from 6.6 to 42 kg. All four patients had limited exercise tolerance and all had severe muscular infundibular pulmonary stenosis. The infundibular stenosis was associated with chromosome 12 short arm deletion, severe global retardation, and epilepsy in one case; with Noonan's syndrome and right ventricular hypertrophic cardiomyopathy in another; with hypoplasia of the right heart elements following a neonatal pulmonary valvotomy and aortopulmonary shunt in the third; and with tetralogy of Fallot with persistently hypoplastic pulmonary arteries despite an aortopulmonary shunt in the fourth patient. Simple balloon dilatation of the right ventricular outflow tract was carried out before stenting in three of the four cases but with no significant benefit. Informed consent was obtained from the par-

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ents of each patient, but ethical committee approval was not sought as each case presented individual difficulties in management.

METHODS

All procedures were performed under general anaesthesia, using femoral venous access, and the severity and extent of obstruction were assessed angiographically. The right ventricular outflow tract was crossed using a hydrophilic guidewire (Terumo, Terumo Corporation, Tokyo, Japan) which was then exchanged for a long, stiff, 0.035 inch guidewire (Amplatz superstiff, Medi-tech Boston Scientific Corporation, USA) allowing a long sheath (F8 in the youngest child, F11 or F12 in the older patients) to be positioned in the pulmonary trunk. Balloon mounted stainless steel stents (Johnson & Johnson) were used in each case. In the 6.6 kg child with hypoplasia of the right heart a 15 mm long stent (P154) was delivered on a 7 mm diameter balloon (Opta 5, Cordis, UK); in the patient with hypertrophic cardiomyopathy a 40 mm long stent (P4014) was delivered on a 14 mm diameter balloon (Olbert, Meadox, UK); and in the remaining two cases a 30 mm long stent (P308) was mounted on either a 7 mm diameter (Blue Max, Medi-tech Boston Scientific Corporation, USA) or a 15 mm diameter balloon (Merck, UK). After delivery of the mounted stent to the infundibulum, the sheath was withdrawn to leave the stent uncovered, the fine positioning of the stent was adjusted with the aid of hand injections of contrast through the sheath, the balloon was inflated, and then right ventriculography was repeated. Prophylactic antibiotic treatment (a cephalosporin) was given for 48 hours. Ambulatory ECG recordings were performed for the first 24 hours after the procedure and the patients were discharged from hospital 24 hours after stent implantation. All the patients were treated with low dose aspirin on an indefinite basis.

Results

Stent implantation was successful in all four cases and no complications were encountered. The haemodynamic results are summarised in the table. The mean systolic right ventricular pressure fell from 105 mm Hg to 39 mm Hg, the ratios of right and left ventricular systolic pressures falling from 0.95 to 0.35 in the three patients with an intact ventricular septum. The oxygen saturation rose from 76% to 91% in the patient with tetralogy of Fallot. Ambulatory ECG recordings showed stable sinus rhythm in all four patients.

FOLLOW UP

Follow up was available for a mean period of 12.3 (range 5 to 20) months. Change in clinical symptoms was not objectively assessed, but two of the four patients felt some subjective improvement in their exercise capacity, there being no evident change in the patient with hypoplasia of the right ventricle nor the patient with hypertrophic cardiomyopathy. The most dramatic improvement in exercise tolerance occurred in the patient with tetralogy of Fallot. This was maintained until 15 months after the procedure, when he noticed a gradual deterioration in his symptoms and his oxygen saturation had fallen from 91% to 80% at rest. Repeat cardiac catheterisation showed that the 7 mm diameter stent was in a stable position but that its lumen was narrowed by intimal hyperplasia (figure). The stent was redilated with a 12 mm diameter balloon with an excellent result and the oxygen saturation rose to 98%. He remained well palliated four months after the redilatation.

The patient with hypoplasia of the right ventricle was electively recatheterised one year after stent implantation to reassess the degree of ventricular hypoplasia and the suitability for further surgery. Disappointingly, her right ventricle did not show any significant increase in size. The stent appeared undistorted and fully deployed. There was some localised muscle hypertrophy at the proximal end of the stent, but there was no visible intimal hyperplasia in this case. The right ventricular pressure had risen from 40 mm Hg (its level immediately after stent implantation) to 75 mm Hg, representing an increase in right to left ventricular systolic pressure ratio from 0.37 to 0.68. She remained well palliated by her aortopulmonary shunt but died suddenly and unexpectedly during an episode of gastroenteritis 16 months after stent implantation. Necropsy confirmed marked hypoplasia of the right ventricle and tricuspid valve, showed the stent to be patent and endothelialised, and showed acute thrombosis of her modified Blalock shunt.

In the remaining two patients follow up was principally by echocardiography. Right ventricular outflow velocities fell from 4.5 m/s to 3.5 m/s in one case and 5 m/s to 3.8 m/s in the other. These improvements were maintained at follow up of eight months and five months respectively. There was no evidence of left ventricular outflow obstruction either before or after stent implantation in the patient with hypertrophic cardiomyopathy.

The stents extended into the proximal pulmonary trunk in the child with pulmonary atresia and in the child with tetralogy of Fallot,

Changes in ventricular pressures or oxygen saturation after right ventricular infundibular stenting

Patient	Age (y)	Pre-stent			Post-stent		
		Po ₂ %	LV	RV	Po ₂ %	LV	RV
Tetralogy of Fallot	13	76	80	94	91	—	—
Pulmonary atresia	2	—	106	116	—	108	40
Infundibular stenosis	9	—	108	100	—	105	48
Hypertrophic cardiomyopathy	10	—	116	98	—	116	30

LV, left ventricular systolic pressure; RV, right ventricular systolic pressure.

Right ventriculography in the patient with tetralogy of Fallot. (A) Before stent implantation the infundibulum (arrowed) was so severely stenosed that it was almost atretic. (B) After deployment of a stent (7 mm diameter, 30 mm long) in the infundibulum, forward blood flow to the hypoplastic pulmonary arteries was greatly improved. (C) 17 months after stent implantation the stent was uniformly deployed and remained undistorted, but the lumen within it was narrowed by irregular intimal hyperplasia (arrowed). (D) After redilatation of the stent with a 12 mm diameter balloon flow to the lungs was much improved.

inevitably causing some degree of pulmonary regurgitation (as would have occurred after surgical patching of the outflow tract in these cases). No haemodynamic deterioration appeared to be caused by the pulmonary incompetence in these two patients; in the remaining two cases the stents did not extend across the valve and there were no clinical or echocardiographic signs of pathological regurgitation.

Discussion

Balloon expandable stents have been used successfully for treating a wide variety of intravascular stenoses that are resistant to simple balloon dilatation. There are rare patients in whom surgical relief of infundibular stenosis is palliative and does not provide definitive treatment and it is this group of patients who might benefit from non-surgical improvement in right ventricular outflow obstruction. Little is known of stent implantation in this setting, but one can imagine a potential for complications such as stent migration, ventricular arrhythmias, collapse or fracture of the stent, or recurrent stenosis.

In this preliminary study we found the stent position to be stable once the stent was fully

deployed and we found no evidence of arrhythmias on ambulatory ECG recordings performed the day after stent implantation. We have been unable to detect any stent movement, collapse, or distortion during follow up (mean 12.3 months) in our patients. Hausdorf *et al*¹ found that stenting provided good palliation in a patient with infundibular stenosis after radiofrequency valvotomy for pulmonary atresia, whereas O'Laughlin and colleagues² described fracture of a stent into three pieces after implantation into an obstructed conduit between the right ventricle and the pulmonary trunk. The stent was positioned partly within fixed obstruction in the conduit and partly within muscular stenosis at the proximal end of the stent. The stent failed to expand symmetrically and later review showed the stent to have fractured and partly embolised to the pulmonary artery with no untoward sequelae. A second stent was implanted with good effect. Stent fracture does not appear to be restricted to muscular compression however; Nakanishi³ reported stent compression and fracture one day after surgical placement of a stent within an intracardiac polyester conduit. At reoperation the stent was split longitudinally into two equal and otherwise undistorted halves. These

reports of stent fracture seem to be associated with imperfect stent expansion at initial implantation; it seems likely that radial strength and consequent resistance to stress fracture may be compromised if symmetrical stent expansion is not achieved at the time of implantation.

Though we have not encountered restenosis caused by external compression of the stent, two of our patients have mild to moderate residual stenosis because the device was slightly too short to stent the complete length of the stenosis, and in the patient with hypoplasia of the right heart this residual obstruction at the proximal end of the stent seemed to progress to some extent over the next year. Nonetheless, considerable improvement in right ventricular outflow obstruction was maintained in all four cases. Intimal hyperplasia sufficient to cause stenosis within the stent was seen in our patient with tetralogy of Fallot. In retrospect the stent was originally deployed at an overcautious diameter of 7 mm and the stenosis was relieved along with dramatic improvement in pulmonary blood flow by later redilatation to 12 mm. Stenosis caused by intimal hyperplasia within pulmonary artery stents is known to occur, the reported incidence ranging from 3%⁴ to 36%⁵;

good responses to repeat balloon dilatation have also been reported in this setting.^{4,5}

Our mean follow up of just over a year is still short and continued monitoring will be necessary to exclude the possibility of late stent compression or fracture. It seems wise to implant the largest size of stent in any given patient to maximise the radial strength of the deployed stent and to allow for redilatation if required to compensate for intimal hyperplasia or somatic growth of the child. These preliminary results suggest that stent implantation may offer an effective alternative to palliative surgical enlargement of the right ventricular infundibulum in selected cases.

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