Pediatr Nephrol (2000) 14:1022–1029

ORIGINAL ARTICLE

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Evaluation and long-term outcome of pediatric renovascular hypertension

Received: 27 July 1999 / Revised: 23 February 2000 / Accepted: 28 February 2000

Abstract Seventeen children with renovascular hypertension were managed at the Royal Children's Hospital, Melbourne, over the 20-year period from 1975 to 1996. The age at presentation ranged from 10 days to 18 years. All children presented with severe hypertension with mean systolic blood pressure 7 standard deviations above age-matched averages and mean diastolic blood pressure 5.5 standard deviations above age-matched averages. Neurofibromatosis was the most common etiology (58% of patients) and there were no cases of Takayasu's arteritis. Patients underwent a variety of biochemical and imaging investigations but in all cases renal angiography was necessary for definitive diagnosis and for planning therapy. Ten of the 17 patients had surgical procedures performed. Percutaneous transluminal angioplasty was performed in four patients but led to cure in only one patient following thrombosis of the affected artery producing segmental renal infarction. Other vascular reconstructive procedures, including the use of autologous or synthetic bypass grafts and autotransplantation, produced cure of hypertension in 50% of children with improvement in a further 30%. The long-term outlook for children treated with surgical reconstructive procedures was excellent. One patient underwent surgery for avulsion of an arterial graft following a pubertal growth spurt. No other patient originally cured by surgery has required reoperation with no cases of restenosis at a mean follow-up of 11 years 3 months.

Key words Renovascular hypertension · Renal artery stenosis · Neurofibromatosis

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Introduction

Three to 10% of all children and adolescents referred to pediatric centers for evaluation of severe hypertension are subsequently found to have clinically significant renovascular lesions [1, 2, 3]. Renal artery stenosis (RAS) is a heterogeneous group comprising intrinsic lesions of the renal arteries and, rarely, extrinsic compressive lesions. Renovascular stenotic lesions are also frequently accompanied by involvement of the aorta and its visceral branches. The most commonly reported cause of RAS is fibromuscular dysplasia [1].

Seventeen children with a diagnosis of intrinsic RAS have been treated at the Royal Children's Hospital, Melbourne, over a 20-year period. We present here our experience in the investigation, management and long-term follow-up of these children.

Materials and methods

The individual records of all children with angiographically proven RAS evaluated at the Royal Children's Hospital, Melbourne, between 1975 and 1996 were examined retrospectively. Children with extrinsic compressive lesions or stenoses of transplanted renal arteries were excluded. Patient case records were reviewed noting clinical features, investigations, treatment and status at last follow-up. Follow-up of those children no longer being treated at the Royal Children's Hospital was obtained by questionnaire sent to their current physician.

The etiology of the RAS was established by clinical and radiographic examination. A clinical diagnosis of neurofibromatosis was made according to standard criteria [4]. Mid-aortic syndrome was diagnosed when the criteria for neurofibromatosis were absent and the arteriogram showed stenosis of the abdominal aorta with narrowing of the renal and visceral arteries [5, 6].

The outcome of surgical treatment was evaluated in terms of: (a) cure – normalization of blood pressure to below the 95th percentile for age without requirement for antihypertensive medication; (b) improvement – normalization of blood pressure while on drug therapy, or decrease in diastolic pressure by more than 15% compared with preoperative levels; and (c) failure – persistent hypertension on antihypertensive therapy [7]. The outcome of medical management was assessed as: (a) improvement – normalization of blood pressure on drug therapy or a decrease in antihyper-

tensive medication over time and (b) failure – persistent hypertension on antihypertensive therapy.

Results

Patients

During the 20-year period from 1975 to 1996 there were 17 children with angiographically proven renal artery stenosis. Patient characteristics at the time of presentation are shown in Table 1. There were ten male and seven females with a median age at presentation of 6.2 years (range 10 days to 18 years). Follow-up ranged from 5 days to 20 years (mean 8.5 years) with long-term follow-up data available on 15 patients. All patients except for one child with neurofibromatosis presented with a systolic blood pressure greater than the 99th percentile for patient age and gender (*Z*-score >3.0) according to the Report of the Second Task Force on Hypertension [8].

Neurofibromatosis was the most commonly diagnosed cause (58% of patients) followed by mid-aortic syndrome. There were no cases of arteritis and, specifically, no patient diagnosed with Takayasu's arteritis. The child who could not be classified presented at 10 days of age with cardiac failure, severe hypertension, and microangiopathic hemolytic anemia and died at 21 days of age. She was found to have bilateral RAS at the origins of the renal vessels and stenotic superior and inferior mesenteric arteries.

The manifestations of hypertension at presentation are shown in Table 2. Patients whose hypertension was discovered incidentally were spread throughout the 20-year period of this study, indicating that the current recommendation [8] for regular checking of blood pressure in children has not led to an increase in diagnosis of RAS.

Investigations

All patients had normal serum urea, creatinine and electrolyte levels. Ten of the patients had assessment of peripheral plasma renin activity (PRA). The PRA was normal in seven patients and elevated in three patients (Table 3). In five children, selective samplings from the renal veins were performed, with two patients having a renal vein renin ratio (RVRR = renal vein renin activity from a kidney with a stenotic renal artery divided by the renin activity from the other renal artery or, if bilateral RAS, the aorta) greater than 1.5. In one patient this correctly localized the side of the stenosis; the other patient had bilateral disease.

The results of radiological investigations are summarized in Table 3. Renal ultrasound examinations were performed in eight children. In five children the kidneys were reported as normal; another two had unilateral small kidneys, and one child had increased echogenicity of both kidneys. Doppler flow studies were performed in seven patients. In two patients the examination was reported as normal and in another four patients the examinations were reported as unsatisfactory, with inability to visualize the renal arteries. One study accurately identified renal artery stenosis in a patient who was later shown to have stenosis by angiography. Diaminotetrapentoic acid (DTPA) scans were performed in

Table 1 Patient characteristics at initial evaluation

Diagnosis	Number	Sex (M/F)	Age range	BP (systolic/diastolic)		BP (*Z*-score: systolic/diastolic)[a]	
				Mean	Range	Mean	Range
Neurofibromatosis	10	5/5	4 months–18 years	178/116	130–250/70–210	6.6/5.1	2.3–13/0.5–15.1
Mid-aortic syndrome	3	3/0	7 months–8 years	173/100	140–240/80–130	7.6/5.8	4.2–9.8/3.4–7.7
Fibromuscular dysplasia	2	1/1	15 months–15 years	193/138	155–230/120–180	7.1/8.3	3.4–10.7/2.6–13.9
Williams syndrome	1	1/0	17 years	–	165/97	3.9/2.6	–
Idiopathic	1	0/1	10 days	–	210/120	10.7/11.5	–
Total	17	10/7	10 days–18 years	180/115	130–250/70–210	6.9/5.5	2.3–13/0.5–13.9

[a]*Z*-scores calculated from [8]

Table 2 End organ dysfunction at presentation

Cause	Cardiac murmur	Left ventricular hypertrophy	Congestive cardiac failure	Encephal-opathy	Hematuria or proteinuria	No end organ dysfunction
Neurofibromatosis	0	1	0	2	3	4
Mid-aortic syndrome	2[a]	1[a]	0	0	0	1
Fibromuscular dysplasia	0	1	0	0	0	1
Williams syndrome	0	0	0	0	0	1
Idiopathic	0	0	1	0	0	0
All causes	2	2[a]	1	2	3	7

[a]One patient presented with cardiac murmur and left ventricular hypertrophy

Table 3 Summary of plasma renin activity levels and radiological investigations (*L* left, *R* right, *RA* renal artery/arteries, *DMSA* dimercaptosuccinic acid, *DTPA* diaminotetrapentoic acid, *SMA* superior mesenteric artery, *IMA* inferior mesenteric artery)

Diagnosis Sex/age	Plasma renin activity[a]	Doppler ultrasound	Nuclear medicine	Renal arteriography	Aortogram
Neurofibromatosis					
F/2 years	1.5	–	–	L RA stenosis	Normal
M/11 months	21.0	Small (L) kidney	–	R upper pole RA stenosis, L ostial stenosis	Normal
M/7 years	3.7	–	DTPA: L 65%, R 35%	Occluded R RA, L RA stenosis	Occluded origin of SMA and IMA. Mild stenosis of L subclavian artery
F/18 years		–	–	R RA stenosis	SMA stenosis
F/4 months	13.6	–	–	R and L ostial stenosis	SMA stenosis. Narrowing of mid and distal abdominal aorta
M/7 years	3.1[b]	–	DTPA: L 28%, R 72%	R and L ostial stenosis	Stenosis of celiac axis. SMA totally occluded at its origin
F/5 years	0.3	Small L kidney, RA not visualized	DTPA: L 37%, R 63%	R and L ostial stenoses	Aorta irregularly narrowed. Stenosis of SMA and celiac axis
F/11 years	1.0	R RA normal, L RA not visualized	Captopril DTPA normal	R RA stenosis	Normal
M/3 years		Kidneys normal, RA not visualized	Normal DTPA	R segmental stenosis	Normal
M/3 years		Increased echo-genicity of kidneys Doppler normal	DTPA: R 36%, L 64%	R and L stenoses	Normal
Mid-aortic syndrome					
M/8 years		–	–	R and L ostial stenoses	Hypoplasia of the thoracic and abdominal aorta. Stenosis of splenic and L subclavian artery. Total occlusion of the origin of the SMA
M/5 years		–	–	R and L ostial stenoses	Tubular coarctation of abdominal aorta. Severe stenosis of celiac artery. Stenosis SMA. Bilateral femoral stenoses
M/7 months	1.0	Kidneys normal, RA not visualized	DMSA: small R kidney; DTPA: R 44%, L 56%	R RA occluded; L ostial stenosis	Aortic narrowing from celiac axis to IMA
Fibromuscular hyperplasia					
M/15 years	1.3[b]	–	–	L RA stenosis	Stenosis at origin of celiac artery
F/15 months		Doppler normal	DMSA: small R kidney; DTPA: R 36%, L 64%	R RA stenosis	Normal

Table 3 (continued)

Diagnosis Sex/age	Plasma renin activity[a]	Doppler ultrasound	Nuclear medicine	Renal arteriography	Aortogram
Williams					
M/17 years		Kidneys normal; Doppler: flat waves	–	R and L ostial stenoses	Marked stenosis of origin of celiac axis. Mild narrowing of upper abdominal aorta
Idiopathic					
F/10 days	8.0	–	–	R and L stenosis	Stenosis of SMA and IMA

[a]Reference range for plasma renin activity 1–4 ng/ml/h
[b]Renal vein renin ratio >1.5

seven patients with six demonstrating abnormal differential function. However, only one of these studies correctly localized the side with stenosis. The other five patients who had abnormal differential function suggesting localization to one side were shown by angiography to have bilateral disease. Captopril renography performed in one patient revealed no evidence of a significant response.

All patients had renal angiography (Table 3). Of the ten patients with neurofibromatosis, six patients had bilateral disease with a mixture of ostial, renal artery and branch renal artery stenosis. A variety of additional vascular lesions were found in the children with neurofibromatosis and are summarized in Table 3. In addition to RAS all patients with mid-aortic syndrome showed narrowing of the abdominal aorta. Two of these patients also demonstrated narrowing of the origin of the superior mesenteric artery, and a further two showed narrowing of celiac axis, extending, in one case, to the splenic artery. The patient with Williams syndrome had arteriogram appearances resembling the mid-aortic syndrome with narrowing of the aorta from the level of the celiac axis and narrowing at the ostium and proximal third of the renal arteries.

Treatment and outcome

Long-term follow-up data were available on 15 patients (88%). Patient treatment and follow-up is summarized in Table 4. Ten of the 17 patients underwent at least one surgical procedure, with three of the ten patients requiring further surgical intervention. The cure rate for those children treated surgically was 50% with another 30% improved. Except for one patient who died at 3 weeks of age, all children treated medically were normotensive on a single antihypertensive agent.

Histology was available for two patients with neurofibromatosis who underwent surgery. In one patient the tissue external to the internal elastic lamina was dysplastic, consisting of mainly collagenous fibrous tissue. One other patient had histology of the renal artery performed at autopsy. The lesion in this section was described as medial fibrous dysplasia with extensive secondary changes including calcification.

At most recent follow-up (mean 9 years 10 months, range 5 days–20 years), 12 of the 15 patients available for follow-up were alive. Two patients died due to complications of hypertension, one at 21 years of age following a cerebrovascular accident and another at 3 weeks of age due to congestive heart failure. The 21-year-old patient had severe ongoing hypertension despite previous left nephrectomy. In addition to her left renal artery stenosis, the angiogram had shown irregularity of the right renal artery, but with no stenosis. It is possible that her persistent hypertension was related either to asynchronous development of a critical stenosis of the right renal artery [37] (repeat angiography was not performed) or secondary to intrarenal changes as a consequence of pre-

Table 4 Patient treatment and follow-up (*FMD* fibromuscular dysplasia, *PTA* percutaneous transluminal angioplasty, *R* right, *L* left, *RA* renal artery)

Sex/age	Diagnosis	Initial treatment	Length of follow-up (years.months)	Outcome
Surgical treatment				
F/2 years	Neurofibromatosis	L nephrectomy	18.0	Failure; died
F/18 years	Neurofibromatosis	PTA unsuccessful	2.9	Improved[a]
F/4 months	Neurofibromatosis	R and L autotransplants	20.4	Cure
M/7 years	Neurofibromatosis	Bilateral RA bypass grafts	15.10	Cure
F/5 years	Neurofibromatosis	Bilateral RA bypass grafts	9.8	Cure
M/7 years	Neurofibromatosis	Bilateral RA bypass grafts. PTA for restenosis R RA. Reanastomosis R graft	14.3	Failure; died
M/8 years	Mid-aortic syndrome	Thoracoabdominal reconstruction	18.3	Improved[a]
M/5 years	Mid-aortic syndrome	PTA unsuccessful. Aortic bypass graft. Bilateral RA bypass grafts	10.3	Improved[a]
F/15 months	FMD	PTA unsuccessful	3.0	Cure
M/15 years	FMD	PTA unsuccessful. L RA bypass graft	0.1	Cure
Medical treatment				
M/11 months	Neurofibromatosis	Multiple antihypertensives	12.8	Improved
F/11 years	Neurofibromatosis	No treatment	9.0	Unchanged
M/3 years	Neurofibromatosis	Single antihypertensive	1.1	Improved
M/3 years	Neurofibromatosis	Single antihypertensive	5.0	Improved
M/7 months	Mid-aortic syndrome	Single antihypertensive	3.1	Improved
M/17 years	Williams	Single antihypertensive	7.5	Improved
F/10 days	Idiopathic	Multiple antihypertensives	5 days	Failure; died

[a]Blood pressure within normal limits on antihypertensive treatment

vious and ongoing hypertension. One other patient died following an unrelated illness. Only one patient has hypertension requiring multiple antihypertensive therapy.

Discussion

Renovascular disease (RVD) is an important cause of hypertension in children, with a reported incidence of 3–10% compared with a 1% incidence in adults. In contrast to adult patients, where RVD is most commonly due to atherosclerosis, 75–95% of children with RAS have some form of arterial dysplasia [1, 9, 10]. This is a heterogeneous group with varying histological characteristics including medial hyperplasia (commonly referred to as "fibromuscular dysplasia"), medial fibroplasia, intimal fibroplasia, perimedial fibroplasia and periarterial fibroplasia [11, 12].

In contrast to most series that report fibromuscular dysplasia (FMD) as the most common cause of RAS (summarized in [1]), vascular lesions associated with neurofibromatosis were the most common cause of RVD in our population (58% of patients). This may reflect our aggressive evaluation of children with neurofibromatosis referred for hypertension. There may also be differences in classification, with some studies previously classifying RAS associated with neurofibromatosis as FMD [13, 20]. Published series that separate these two entities have reported a 10–25% incidence of neurofibromatosis in children with renovascular hypertension [2, 9, 14, 15]. The wide range of reported incidences might also reflect the difficulty of classifying children who have RAS but without other clinical stigmata of neurofibromatosis. Although well-defined diagnostic criteria for neurofibromatosis are available, the diagnosis remains problematical in young children, who may not manifest clinical features such as café-au-lait spots, Lisch nodules and cutaneous neurofibromas until late childhood [11, 16]. O'Regan et al. [14] reported that, of five children with RAS secondary to neurofibromatosis, three children had only one other clinical feature of neurofibromatosis at presentation with hypertension. In addition, in this series the radiological appearance of vascular neurofibromatosis was often identical to the appearance of either isolated FMD or mid-aortic syndrome and therefore was unhelpful in classifying children without other features of neurofibromatosis. In a review of pathological specimens from children with RAS, Blackburn [17] found that many of the lesions that had previously been labeled as FMD were actually secondary to vascular neurofibromatosis. The histology of the renal artery lesions in two of our patients with neurofibromatosis was non-specific. Devaney et al [42] reported that the renal artery lesions in patients with neurofibromatosis did not differ significantly from those in patients who did not have neurofibromatosis. Therefore neither radiological nor histologi-

cal features can reliably differentiate children with neurofibromatosis from children with fibromuscular dysplasia or mid-aortic syndrome. Thus the true prevalence of vascular neurofibromatosis may have been underestimated in previous studies.

Arteritis was reported as the second most common cause of RAS in one series [15], and Takayasu's arteritis in particular has been the subject of a number of case reports [18, 19]. However, our series and a number of others reported in the literature [2, 10, 20] would suggest that arteritis is an uncommon cause of RAS in Caucasian children.

The accurate diagnosis of RAS in hypertensive children remains difficult and has been the subject of a number of recent reviews [21, 22, 23]. Although a wide variety of specific diagnostic procedures are available, variable results have been reported on the ability of diagnostic tests to predict the presence and location of RAS. In contrast to previous reports in which the majority of children with renovascular hypertension had elevated peripheral venous plasma renin activity (PRA) levels [24], PRA levels were elevated in only 30% of the patients in our series (Table 3). Interpretation of PRA in this population is complicated by the high incidence of bilateral disease, which may be associated with normal PRA activity [1]. In our series, four of seven patients with bilateral disease had a normal PRA. Measurement of the renal vein renin ratio (RVRR) was also unhelpful, being informative in only one patient out of the five who had the test performed. Accurate determination of plasma renin levels is dependent upon sampling conditions and a number of other factors, including posture, sodium intake and concurrent antihypertensive therapy [1]. At the time of sampling, three patients with normal RVRR were taking β-adrenergic-blocking drugs, which are known to decrease renin release [43]. While the low diagnostic yield in our series may have been related to the lack of standard conditions at the time of sampling, the strict protocols advocated for adults [25] are unlikely to be feasible for young children. Although the usefulness of PRA in detecting significant RAS appears to be limited, peripheral PRA should still be considered as part of the initial evaluation of children with hypertension as it identifies a small group of children with mineralocorticoid-induced refractory hypertension [26].

In agreement with other series, we found no reliable non-invasive screening test for RAS. While Doppler ultrasound has been suggested as a useful technique in the investigation of hypertensive children with neurofibromatosis [27], up to 20% of examinations may be technically unsatisfactory, even in experienced hands [22]. In our series, differences in size, shape or echotexture were noted in only two out of eight affected patients and Doppler ultrasound was suggestive of RAS in only one out of eight examinations (three technically unsatisfactory). These figures are similar to those reported by Ellis et al. and may reflect the fact that Doppler ultrasound is less effective in bilateral or branch disease [21]. In our patient series, a number of the unsatisfactory examinations were performed prior to the availability of high-resolution scanners. More recently, the availability of technically sophisticated equipment along with the use of multiple views has been shown to lead to improved sensitivity and specificity of this technique [28]. Echo-enhanced duplex ultrasound using a galactose-based contrast agent is a new technique that has been shown in adults to produce better images than conventional color Doppler [29]. The fact that this technique significantly reduced mean examination time makes it an attractive investigation for use in children, and further evaluation of this technique is awaited.

Nuclear medicine has become more widely used in the preliminary investigation of children with suspected renovascular disease [30]. Mann et al. [31] reported that captopril renography, using criteria of asymmetry of DTPA uptake, time to peak uptake, or retention seen on a single post-captopril renogram, was highly sensitive and specific for detecting unilateral RAS in adults. However, the same study found that a positive result could not reliably differentiate renovascular disease from unilateral parenchymal disease and that renography did not distinguish unilateral from bilateral disease. Gauthier et al. [32] studied hypertensive children with non-diagnostic ultrasound and concluded that the captopril challenge test was inadequate for diagnosing RAS in this population. The low sensitivity and specificity (59% and 68%, respectively) of isotope scanning has also been confirmed by Ng and colleagues [33]. DTPA scanning pre- and post-captopril was performed in only one patient in our study and failed to detect a clinically significant unilateral RAS. Non-captopril DTPA scans were performed more frequently in our population and also failed to differentiate unilateral from bilateral disease. Because of these limitations, renography, either with or without captopril, cannot be recommended as a routine investigation for suspected RAS.

Our series confirms that contrast angiography is currently the only reliable way to diagnose renal artery stenosis.

Revascularization, either surgically or by percutaneous transluminal angioplasty (PTA), is recommended for children with RAS to help preserve renal function, prevent injury to other organs and obviate or decrease the need for long-term antihypertensive medication [23]. PTA has been advocated as a safe and effective therapy for RVD, especially in patients with fibromuscular hyperplasia [34, 35]. However, PTA was less successful in our series. Dilatation of primary RAS was attempted in four patients, with no child cured of their hypertension despite technically satisfactory procedures. The stenosis in one patient with FMD (M/15 years, Table 4) was unable to be dilated despite three balloon inflations at maximal pressure, with a fourth attempt leading to balloon rupture. At surgery, a rigid-walled stenosis of the renal artery was found suggesting a true hypoplastic vessel, a lesion that may not be amenable to PTA [37]. In the other patient with FMD (F/15 months, Table 4), attempted PTA of a segmental renal artery led to thrombosis of the

vessel and infarction of the mid-zone of the kidney. Paradoxically this led to cure of hypertension in this patient, an outcome previously reported [36]. PTA for restenosis of a bypass graft was performed in one patient and was also unsuccessful. Stanley et al. [37] have reported vessel fracture and apparent success with balloon inflation followed by reappearance of the stenosis as deflation occurs due to excess elastin of these arteries as the two most common outcomes following PTA. This led them to conclude that PTA has a very limited role in the treatment of pediatric renovascular hypertension. Other studies [20, 23, 38] have also reported a poorer outcome for PTA in children with isolated ostial lesions or syndromes with aortorenal involvement such as neurofibromatosis and mid-aortic syndrome. Of the four children in our series on whom PTA was attempted, one patient had neurofibromatosis and one had mid-aortic syndrome. This may explain the failure of PTA in these patients and confirms previous recommendations for a surgical approach to these patients [20, 23].

Surgical reconstructive procedures for RVD involve either angioplastic reconstruction to bypass the stenosis with synthetic or autologous grafts, or autotransplantation of one or both kidneys. Nephrectomy may also be considered as an alternative to revascularization if an affected kidney contributes less than 10% of renal function. In a review of published surgical series of pediatric patients, five of six series examined reported postoperative rates of cure or improvement of over 90% [7]. Two more recent very large long-term series of surgically treated children with RAS reported cure rates of 70% [39] and 79% [37], with improvement in a further 26% and 19% respectively. Our series had worse cure rates (50%) but the number of patients was small in comparison to other studies and many patients in our series had complex lesions involving both the renal arteries and the vascular tree. Our figures are in close agreement with those of Ellis et al. [23], who summarized a 10-year experience in 52 children with complex bilateral lesions reporting a 52% cure rate. Of note, 5 of the 27 cured patients (18%) required more than one surgical procedure, and follow-up in all studies was relatively short. Our patients treated surgically have been followed for a mean of 11 years 3 months. During this time no patient who was initially cured following surgical intervention has had recurrent disease. One child required emergency reoperation following avulsion of an autologous graft from the aorta during a pubertal growth spurt. This child survived and remains normotensive on no antihypertensive medication.

In summary all children with hypertension should be fully assessed for the possibility of a correctable renal arterial lesion. In particular, young children that do not have the full diagnostic criteria for neurofibromatosis may have significant renal lesions. While angiography remains the gold standard for diagnosis of renovascular disease, newer imaging modalities such as gadolinium-enhanced MRI [40] and spiral CT angiography [41] are becoming more widely available and have the potential to provide relatively non-invasive diagnosis although their validity in pediatrics remains to be determined. In addition, the increasing availability of high-resolution Doppler ultrasound is likely to lead to improvements in the diagnostic accuracy of this modality. Optimal treatment remains highly individualized and requires careful discussion between nephrologist, radiologist and vascular surgeon. The current role for PTA in these children is controversial and dependent on the nature of the lesion and the experience of the operator. PTA appears to be suitable for children with discrete lesions of the renal artery secondary to fibromuscular hyperplasia. However, in children with ostial lesions, especially those associated with neurofibromatosis, or more extensive renal lesions with involvement of intrarenal vessels, PTA cannot be recommended. The long-term outcome after surgical revascularization remains excellent with a low rate of restenosis.

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