

## SHORT REPORT

# Selective renal embolisation for renovascular hypertension?

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*Arch Dis Child* 2002;**86**:127–129

An 11 year old girl developed hypertensive encephalopathy and renal failure from reflux nephropathy. Resection of her shrunken left kidney did not control her hypertension. Two selective arterial embolisations of the scarred right lower pole produced only transient benefit, but a heminephrectomy gave good control. Embolisation may delay definitive treatment.

Hypertension is uncommon in childhood,<sup>1</sup> and is often diagnosed late, despite recommendations to measure blood pressure in all unwell children.<sup>2</sup> It may be severe, leading to encephalopathy, retinopathy, and even death.<sup>1</sup> The commonest cause is renal scarring from reflux nephropathy,<sup>1</sup> which may occur after one urine infection in the young.<sup>3</sup> Management sometimes includes resection or ablation of scarred renal tissue. The case we present provides important lessons.

## CASE REPORT

A toddler was repeatedly treated for urine infections, but not investigated. At 10.5 years she developed severe headaches and vomiting; her brain computerised tomogram was normal, and pizotifen treatment was ineffective. Her blood pressure was repeatedly recorded as normal by an automatic oscillometric device. She then developed a further urine infection and was imaged with ultrasound, a dimercaptosuccinic acid scan, and a cystogram. Her left kidney was small, had vesicoureteric reflux, and virtually no function. Her right upper pole was hypertrophied and functioned well; the lower pole was small, functioned poorly, and contained a calculus. There was no vesicoureteric reflux on the right. The raised plasma creatinine concentration of 112  $\mu\text{mol/l}$  (equivalent to a glomerular filtration rate of about 55  $\text{ml/min/1.73 m}^2$ )<sup>4</sup> was not appreciated because the laboratory quoted an adult upper limit of normal (120  $\mu\text{mol/l}$ ).

Six months later, when she had visual impairment and weight loss, and was too ill to attend school, the automatic device failed to register her blood pressure at all. Her systolic pressure was found to be 230 mm Hg, using a sphygmomanometer with a large cuff and Doppler. Her creatinine had risen to 291  $\mu\text{mol/l}$ , and she was transferred to our unit. She had left ventricular hypertrophy on echocardiography. Fundoscopy revealed papilloedema, exudates, and haemorrhages. Her visual acuity was 6/18 bilaterally. She began hallucinating. Treatment with intravenous labetalol 4 mg/kg/h, oral nifedipine 1.6 mg/kg/day, atenolol 6 mg/kg/day, and frusemide 3 mg/kg/day failed to control her blood pressure. Adding intravenous sodium nitroprusside 1.8  $\mu\text{g/kg/min}$  was effective, but had to be withdrawn because her red blood cell cyanide concentration was significantly increased at 72  $\mu\text{mol/l}$ . We therefore considered resecting her kidney scars.

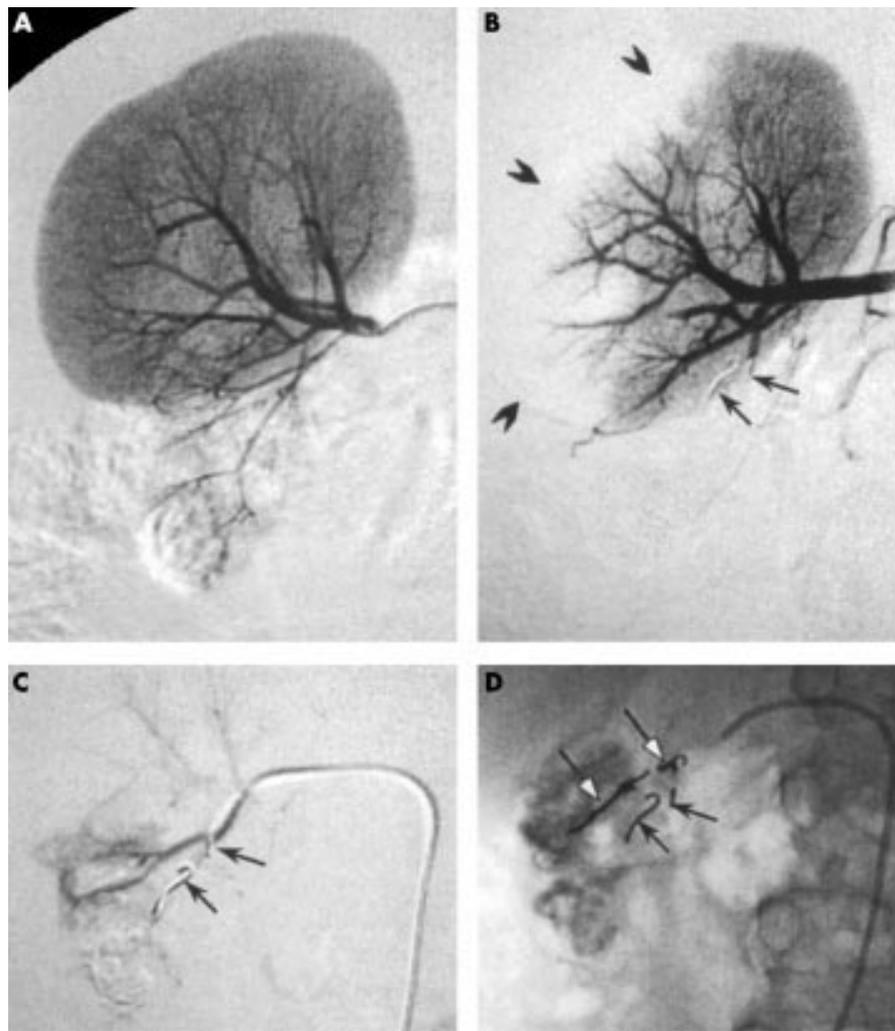
Unfortunately, left nephrectomy produced no improvement. Histology revealed end stage pyelonephritis and vascular hypertensive changes. To avoid the risk of damaging her upper pole during right lower pole partial nephrectomy, we embolised two small arteries with microcoils and gelfoam obstructing all blood flow to the lower pole (fig 1A and B). This caused immediate but transient, patchy impairment of perfusion to the upper pole, followed by three days of pyrexia and pain. There was a brief rise in plasma creatinine, but her blood pressure control improved. However, a month later her blood pressure had risen again, and we repeated her arteriogram. Two small arteries had revascularised the lower pole. These were embolised in a second procedure, resulting in no blood flow through them (fig 1C and D). She followed a similar postoperative course with only a transient improvement in blood pressure control. She therefore had a right lower pole heminephrectomy. The kidney was found to be duplex, and the surgery uncomplicated.

In the three years since surgery, she has remained well with excellent blood pressure control on oral nifedipine 1.5 mg/kg, atenolol 1.6 mg/kg, frusemide 1.2 mg/kg, and propranolol 1.2 mg/kg per day. Her glomerular filtration rate is approximately 50  $\text{ml/min/1.73 m}^2$ . Her visual acuity has returned to normal, and she is on the 50th centile for both height and weight. The left ventricular hypertrophy has completely resolved.

## DISCUSSION

If this girl had been imaged as a young child to investigate her urine infections, as recommended,<sup>3</sup> reflux nephropathy would have been identified. The left ventricular hypertrophy indicated that her hypertension had been long standing at detection. Had she had blood pressure screening implemented because of her scarring, the hypertension would almost certainly have been detected long before she developed a life threatening encephalopathy, or worsening of her renal function. Except when it is identified as a result of screening for a renal condition, hypertension is rarely diagnosed promptly in childhood, perhaps because it is seldom considered as it is relatively uncommon, and because blood pressure measurement is not always easy in small children. In this case, further delay occurred because an automatic measurement device failed to detect it. There is more potential for a complicated instrument such as an oscillometer to develop unrecognised faults than a simple sphygmomanometer, ideally used with a Doppler to detect radial flow at the wrist.<sup>1</sup> All blood pressure measuring equipment should be regularly maintained and calibrated.

The choice between surgical excision or embolic ablation of renal scars is primarily related to the potential risk of damaging adjacent normal kidney tissue. Surgical removal of this child's globally scarred and virtually non-functioning left kidney carried no such risk, and was thus undertaken early. By contrast, we felt that the risk of removing her right lower pole carried a risk of damaging the functioning upper pole. Though



**Figure 1** Subtraction right renal arteriograms in a hypertensive girl with reflux nephropathy: (A) before first embolisation; (B) after first embolisation. Coils are indicated by black arrows; areas of transiently reduced perfusion due to spillage of gelfoam are indicated by arrowheads. (C) Lower pole before second embolisation; previously placed coils shown with black arrows; newly placed coils indicated with white arrows.

heminephrectomy is a relatively straightforward procedure if the kidney is duplex and has a clear plane of division, it is more likely to harm the remaining pole if it is not. Because none of the imaging investigations were able to determine this, we initially chose embolisation over surgery.

We considered measuring her selective renal vein renin activities to pinpoint precisely the anatomical source of the renin drive before planning surgery. However, this technique does not always lateralise the renin source reliably,<sup>5,6</sup> and would have required another procedure under general anaesthetic which was unlikely to have a major impact on the final management. If it had pointed to the right lower pole as the prime source of renin, we would almost certainly have also removed her functionally useless left kidney anyway because of its future risk of causing hypertension.

Though selective renal embolisation has been used successfully to treat hypertension in adults<sup>7</sup> and children,<sup>8</sup> it is not always successful, partly because of the capacity of the kidney to develop collateral vessels,<sup>7</sup> as in this patient. Embolisation causes significant pain post procedure,<sup>7,8</sup> which in our patient was as severe as after her nephrectomy. Spillage of gelfoam into healthy tissue is also a potential hazard. In this case, although the two embolisations were technically successful, they failed to improve blood pressure control. The post-procedure symptoms were unpleasant and effective management was delayed.

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Accepted for publication 28 August 2001

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