CASE REPORT

Spleno-renal artery transposition in a solitary functioning kidney for treatment-resistant hypertension and acute kidney injury

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SUMMARY

Renal Artery Stenosis (RAS) is an important cause of treatment-resistant hypertension. Uncontrolled hypertension with RAS can cause progressive chronic kidney disease (CKD) leading to end-stage kidney disease. Therapeutic revascularisation can be helpful in appropriate circumstances where pharmaceutical intervention has failed and significant renovascular disease contributes to resistant hypertension. We present an interesting case of a Caucasian male with peripheral vasculopathy, abdominal aortic aneurysm (AAA), single functioning kidney and ostial RAS caused by stent struts from an endovascular AAA stent graft. He had escalating medications requirement, with repeated failed attempts at percutaneous radiological intervention that led to an episode of contrast-induced acute kidney injury (AKI), before undergoing successful surgical revascularisation by a splenic artery transposition graft to the left renal artery that was performed to improve kidney function and the blood pressure. This report highlights the challenges faced with regard to the management of severe hypertension and progressive CKD.

BACKGROUND

Renal Artery Stenosis (RAS) is an important cause of treatment resistant hypertension in chronic kidney disease (CKD). Progressive CKD in a single functioning kidney with resistant hypertension due to RAS poses a difficult but not unusual clinical challenge. This case highlights the issues faced during the management of a complex clinical problem with a patient who had co-existing widespread arteriosclerotic vascular disease treated with arterial bypass grafts and endovascular stents, occlusive ostial renal artery stenosis, treatment resistant hypertension and declining kidney function. The authors share important learning points from this case.

CASE PRESENTATION

A 76-year-old Caucasian man was referred by his general practitioner (GP) to the local nephrology services for asymptomatic decline in kidney function and difficult to treat hypertension since 2001. Review of the background revealed that his kidney function was recorded as normal between October 2003 and December 2005, with a serum creatinine (SCr) ranging from 100 to 111 mmol/L and

an estimated glomerular filtration rate (eGFR) of >60 mL/min/1.73 m². He was diagnosed with an infrarenal abdominal aortic aneurysm (AAA) in August 2004 and was under regular follow-up with interval ultrasonography (US). He also suffered from symptomatic peripheral vascular disease (PVD) with claudication in the right lower limb. In 2005, he underwent uncomplicated right femoral-popliteal bypass surgery for his PVD. An interval US scan at that time did not reveal any involvement of his renal arteries within the AAA. In 2007, he underwent an elective endovascular repair and graft stent insertion for his AAA. His kidney function at the time of AAA repair showed an eGFR of 55 mL/min/1.73 m². He had type 2 diabetes mellitus since 2010 that was managed with metformin. In September 2012, a routine check by the GP showed that he had developed chronic kidney disease (CKD) stage three with an eGFR of 32 mL/min/1.73 m². His blood pressure (BP) also became more difficult to control, despite treatment with four agents. He also had a history of transient ischaemic attack (TIA). Osteoarthritis was managed without the use of non-steroidal anti-inflammatory drugs. His kidney function remained stable during and after a total knee replacement in early 2013.

In March 2013, by the time of first contact with nephrology, he had drug-resistant hypertension and renal impairment, with eGFR 28 mL/min/1.73 m². He was on metformin 500 mg two times a day, clopidogrel 75 mg once a day, indapamide 2.5 mg once a day, candesartan 8 mg once a day, bisoprolol 10 mg once a day, doxazosin 4 mg two times a day and simvastatin 20 mg once a day. There was no family history of kidney disease. He had two healthy children, drank 1–2 glasses of wine a day and was an ex-smoker, having stopped 10–12 years ago (40 pack-years history). Blood pressure readings at home ranged from 165/80 to 190/110 mm Hg. On examination, he appeared euvoelaemic with a BP of 178/88 mm Hg and had a full complement of peripheral pulses without radio-radial or radio-femoral delay. Urine albumin-to-creatinine ratio was 0.5 mg/mmol. US of his kidneys excluded urine outflow obstruction and showed kidney sizes of 11 cm (left) and 7 cm (right). ECG showed left ventricular hypertrophy. In view of the suspicion of significant renovascular disease, candesartan was stopped, doxazosin was increased to 8 mg two

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Figure 1 This CT figure is a cross-sectional image that shows the presence of the graft stent struts at the origin left renal artery from the abdominal aorta complicating the existing left ostial renal artery stenosis.

times a day, spironolactone 12.5 mg once a day was commenced and a salt-restricted diet was advised. Metformin was stopped and diabetes mellitus was adequately managed by diet control alone. Subsequently, his kidney function appeared to improve (creatinine 148 mmol/L and eGFR 40 mL/min/1.73 m²). A CT angiogram with an arterial contrast phase of the kidneys showed severe atrophy of the right kidney measuring 8.4 cm with a right ostial renal artery stenosis (RAS); the left kidney measured 12.1 cm and there was a bifurcated aortic stent graft with suprarenal fixation (figures 1 and 2). There were no visible aortic endoleaks, but a left RAS and the presence of AAA stent graft struts were noted at the origin of left renal artery. Comparison with previous imaging showed that the small right kidney predated the stent graft insertion for the AAA. His BP remained poorly controlled with both home and clinical readings of 180–210/90–100 mm Hg.

Compliance with the medications was good. Spironolactone, however, was not tolerated and it was switched to amiloride and bumetanide. Hydralazine was later introduced with dose titration. His BP improved only marginally and remained around

Figure 2 The CT renal angiogram in an arterial phase shows the presence of an aortic stent strut graft. There is a left renal ostial stenosis.

Figure 3 This is a catheter renal angiogram, which shows aortic graft stent in situ with contrast in the aorta and left renal artery. There is left renal ostial stenosis, but the presence of graft stent struts at the origin of left renal artery prevents the catheter to move forward into the left renal artery for revascularisation.

160–180/80–90 mm Hg after several months of outpatient follow-up.

At this stage, an elective left renal artery catheter angiogram was performed with a 6-French sheath that was placed in the right common femoral artery and a catheter flush angiogram confirmed a left ostial RAS. The guidewire was able to pass into the left renal artery origin, but the catheter would not advance further due to the presence of metal struts of stent graft in the abdominal aorta (figure 3). The attempt at revascularisation was abandoned. Prehydration before catheter angiogram prevented contrast-induced nephropathy and renal function remained stable.

BP control in the clinic remained a significant challenge. Urine sodium excretion was 130 mmol/24 hours (salt intake of approximately 7–8 g/day) and he was again advised to reduce salt intake. Aldosterone levels were 1266 pmol/L (100–800 pmol/L) with plasma renin activity of 97.1 mU/L (5.4–60.0 mU/L) and the ratio of aldosterone:PRA was 13 (<80 ratio). At this time, he was on seven antihypertensive agents (indapamide 2.5 mg once a day, hydralazine 50 mg two times a day, lacidipine 6 mg, bisoprolol 10 mg, doxazosin 8 mg, minoxidil 5 mg, amiloride 5 mg once a day and bumetanide 2 mg once a day). A few months later, he developed an episode of flash pulmonary oedema and an elevated N-terminal pro-brain natriuretic peptide of 2384 units, hypokalaemia and a stable eGFR of 45 mL/min/1.73 m². Minoxidil and indapamide were discontinued. Amiloride was continued and the dose of bumetanide was increased. In January 2015, he underwent a second attempt of revascularisation of the left RAS via the percutaneous radial artery approach, which again was unsuccessful due to the presence of the graft stent struts. On this occasion, however, there was a contrast-induced nephropathy leading to an acute kidney injury (AKI) with SCr 468 mmol/L and eGFR 11 mL/min/1.73 m². Over the next few weeks, the AKI recovered and SCr stabilised to 140 mmol/L and eGFR 45 mL/min/1.73 m² with titration of the diuretics and hydration. He remained asymptomatic, although he was starting to complain of side effects relating to the high pill burden.

In April 2015, he developed exertional chest pain and another episode of flash pulmonary oedema. A myocardial perfusion scan excluded coronary artery disease. A month later, he developed an acute deterioration in his vision. A specialist

Figure 4 The chronology of the events with respect to the serum creatinine (SCr) levels in mmol/L and the number of antihypertensive drugs (single numbers in the plot area) the patient is on at that time.

ophthalmology clinic assessment revealed a left anterior ischaemic optic neuropathy secondary to hypertension. In view of treatment-resistant hypertension (BP of 155–190/70–90 mm Hg) with seven drugs, widespread target end-organ damage, including a TIA, LVH, hypertensive retinopathy, AKI on CKD, and recurrent episodes of flash pulmonary oedema and failure to manage his BP adequately by means of pharmacological and radiological interventions, surgical options were then explored. The option of autotransplantation of the left kidney into the iliac vessels was raised and explored with a CT of the aorta and iliac arteries. This demonstrated an aortic stent graft with supra renal fixation extending bilaterally to the distal common iliac arteries, with an aortic aneurysmal sac of 4.6 cm with further aneurysmal dilatation of the right common iliac artery around the distal end of the right limb of the graft measuring 3.8 cm. Bilateral external iliac arteries were calcified with a diameter of 1 cm. A multidisciplinary team, comprising nephrologists, interventional radiologists and vascular surgeons, decided in favour of surgical revascularisation of his left renal artery.

In July 2015, he underwent a complex but successful splenic artery to left renal artery transposition graft. The procedure was performed in the decubitus position under general anaesthesia. Following a transverse laparotomy, a medio visceral rotation manoeuvre anterior to the left kidney was performed. The splenic artery was dissected on the superior aspect of the pancreas from its origin on the coeliac trunk to its end in the splenic hilum. The left renal artery was dissected below the pancreas and just against the abdominal aorta. A tunnel was created behind the pancreas, once both the arteries were dissected and safely slung. An injection of 5000 IU unfractionated heparin was given and the splenic artery was clamped just after its origin and was then cut next to the spleen. The artery was then mobilised behind the pancreas, through the tunnel and positioned next to the left renal artery that was clamped and cut just after the stenosis (1 cm from its origin). An end-to-end anastomosis was performed by a continuous suture with a 5/0 prolene. Both the arteries were unclamped and haemostasis was achieved. Closure was achieved layer-by-layer and a drain was left in situ for few days. BP improved postoperatively (138/72 mm Hg), recovery was uneventful and he was discharged after 8 days.

By September 2015, there was an improvement in his kidney function, with an eGFR of 55 mL/min/1.73 m² (SCr 113 mmol/L)

and he now required only four drugs (candesartan 8 mg once a day, bisoprolol 10 mg once a day, amlodipine 10 mg once a day and hydralazine 25 mg once a day) to control his BP (130–150/70–80 mm Hg) (figure 4).

OUTCOME AND FOLLOW-UP

Since December 2016, his hypertension has been managed with three drugs (candesartan 12 mg once a day, bisoprolol 10 mg once a day and amlodipine 10 mg once a day) and his kidney function has remained stable (SCr 110–125 mmol/L and eGFR 51–59 mL/min/1.73 m²). He remains under regular long-term follow-up. By May 2017, his eGFR continued to remain stable and his BP control is excellent (120–140/70–80 mm Hg).

DISCUSSION

Hypertension is one of the leading causes of CKD.^{1 2} Resistant hypertension is defined as BP that remains above goal despite the concurrent use of three antihypertensive agents of different classes, one of which should be a diuretic.³ Treatment-resistant hypertension is not an infrequent problem in nephrology clinics, with a 40.2% prevalence of apparent treatment-resistant hypertension according to the Chronic Renal Insufficiency Cohort Study.⁴ Incomplete concordance, high-salt diet and undiagnosed secondary cause of hypertension all have to be considered when assessing such a problem. Patients often end up on multiple antihypertensive drugs. Hypertension can cause chronic kidney injury due to ischaemic nephropathy and when CKD progresses, hypertension is compounded in a vicious cycle.² Atherosclerosis is the leading cause of RAS, which is one of the important causes of secondary hypertension and CKD. Most patients with RAS have asymptomatic, non-critical disease with hypertension, CKD or both and they are predominantly managed with pharmacological intervention. Diagnosis of RAS is suspected when there is one or more of the following: drug refractory hypertension (>3 drugs) with a history of flash pulmonary oedema, history of PVD, radiological imaging showing more than 1.5 cm disparity in the size of the kidneys and declining eGFR. RAS can be diagnosed with by CT renal angiogram or magnetic resonance angiogram of the renal arteries. The gold standard for confirmation of this diagnosis is by performing a catheter renal intra-arterial digital subtraction angiogram. The Angioplasty and Stent for Renal Artery Lesions (ASTRAL) trial showed that there was no evidence of significant clinical benefit from percutaneous revascularisation in patients with asymptomatic atherosclerotic renovascular disease when compared with those who were treated pharmacologically.⁵ Similar outcomes were found in the CORAL study.⁶ However, evidence from these studies does not necessarily apply to all the patients with renovascular disease, particularly in those clinical situations that were not likely to have been represented in the ASTRAL and CORAL studies, such as those with a solitary functioning kidney with RAS and declining renal function in whom an intervention to revascularise may be beneficial.

Our patient had two attempts at percutaneous revascularisation of the solitary left renal artery, but attempts at passing the arterial intervention catheter into the left renal artery failed due to the presence of stent struts from the AAA graft. Surgical options were considered in view of treatment-resistant hypertension with significant target organ damage including declining kidney function. Anatomical considerations and vascular disease burden permitted only a few options for the approach to revascularise the left renal artery. Aortorenal and iliorenal options were rejected in view of the presence of graft stents in the aorta

and the iliac vessels. A hepatorenal approach was thought to be unsuitable for anatomical reasons in our case. A splenic artery diversion was considered the best option and was achieved by mobilising the vessel through a retropancreatic approach to graft the left renal artery.^{7,8} A literature search for splenorenal bypass graft for critical atherosclerotic RAS (ARAS), with or without simultaneous aortic disease, has revealed case reports^{8–10} and a few retrospective studies.^{11–15} Rescuing an arterial supply to a single functioning kidney is challenging and as such outcomes from retrospective analyses are variable. In one early pioneering study from the 1970s, 32 patients with renovascular hypertension underwent splenorenal bypass, out of which 17 patients were 'cured' of hypertension, 9 patients had improved BP readings and 6 failed to improve BP.¹² There were two postoperative deaths and three cases of stenosis of the splenic artery graft as a long-term complication.

Comprehensive review of the literature on the outcomes of revascularisation of renal arteries in a solitary functioning kidney has revealed predominantly retrospective reporting. In one study, 35 patients underwent extra-anatomic surgical revascularisation with 91% patients recovering kidney function and 85% of them having improved BP control.¹³ Another group published data that were collected over an 18-month period on 11 patients who underwent bypass grafting for symptomatic ARAS and hypertension with AKI. Eight patients with ARAS and azotemia were treated with six hepatorenal and five splenorenal artery bypasses. SCr improved in all patients but only four of the five patients with severe hypertension had improved BP outcomes.¹⁴ Splenorenal bypass is associated with a high risk of splenic and pancreatic injury while trying to expose the splenic artery. The risk of splenic injury is reduced when the splenic artery is approached through the retroperitoneum at the base of the transverse mesocolon, especially when the spleen and the splenic flexure of the colon are mobilised to expose the splenic artery.^{14–16} Although studies have shown that splenorenal bypass grafting is a relatively safe procedure, there have been reports of serious complications such as splenic artery infarction.¹⁷ Darling *et al* published on 600 different vascular reconstructive procedures for renal arteries over a period of 12 years and reported 31 (5.5%) surgical deaths.¹⁵ In a prospective study in 1993, 58 patients with RAS were randomised to undergo percutaneous transluminal renal angioplasty (PTRA) versus surgical angioplasty for the treatment of hypertension and showed no difference in the improvement of BP and kidney function.¹⁸ Balzar *et al* published their 10-year single-centre experience in 2012 with surgical intervention following a failed PTRA. Of the 696 cases, 60 who underwent PTRA required

surgical intervention, with improved BP in 22.2% with ARAS and 54% with fibromuscular dysplasia (FMD) and restenosis that occurred in 18% with ARAS and 24% with FMD.¹⁹ Both these studies suggest that the PTRA should be the first non-medical interventional approach for RAS.

Surgical intervention for ARAS and poorly controlled hypertension should not be the first line of management. Carefully selected cases, in which medical therapy and percutaneous revascularisations have either failed or are not appropriate, should be considered for an arterial reconstructive surgical intervention. In these instances, patients should be adequately informed of the possible outcomes, including risk and benefits. It is likely that a multidisciplinary approach will optimise outcomes in this group of patients. Our case attempts to highlight the difficulties and the complexity of managing such patients with significant vascular disease and difficult to treat hypertension.

Contributors SS prepared, edited and researched the manuscript and managed the care of the patient. RJS edited and researched the manuscript and managed the care of the patient. EC operated on the patient and is involved in the editing of the manuscript. FEH managed the patient, edited and researched the manuscript. PS managed the care of the patient, edited and researched the manuscript.

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Learning points

- Atherosclerotic RAS (ARAS) is an important cause of hypertension in adults with significant peripheral vascular disease.
- Abdominal aortic aneurysm (AAA) can cause renal dysfunction and hypertension by causing occlusive renal artery stenosis.
- Endovascular repair of AAA with stent grafts may complicate occlusive renovascular lesions and prevent percutaneous revascularisation.
- Surgical revascularisation of ARAS in carefully selected cases can be a treatment option for hypertension and preservation of renal function.

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