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Severe acute kidney injury in a patient with renal artery stenosis of a single-functioning kidney: A case report of rapid normalisation of the renal function after percutaneous transluminal angioplasty with stent placement

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Abstract

Revascularisation of renal arterial stenosis in acute settings, such as uncontrolled arterial hypertension, flash pulmonary oedema and/or acute renal failure, has shown controversial results in observational and prospective studies. Current guidelines do not recommend revascularisation in the occurrence of renal failure as revascularisation and best medical treatment have shown similar long-term outcomes on renal function. We describe a case of acute degradation of the renal function (with oligo-anuria and a peak creatinine of 462 µmol/L) after the re-introduction of an angiotensin-II receptor blocker (irbesartan) in a 66-year-old Caucasian diabetic male patient with bilateral renal stenosis and a right-sided single-functioning kidney, with a rapid improvement of the renal function which normalized 5 days after percutaneous angioplasty and stenting of the right renal artery.

Keywords

Acute renal failure, acute kidney injury, renal artery stenosis, percutaneous transluminal angioplasty with stent placement, single-functioning kidney, angiotensin receptor blocker

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Introduction

Renal arterial stenosis (RAS) is a known cause of uncontrolled arterial hypertension, causing in severe cases, flash pulmonary oedema and/or acute renal failure. Current recommendations[1,2] concerning its management are based on several important prospective/randomised studies and meta-analysis (EMMA/1998, Scottish and Newcastle/1998, Dutch RAS/2000, STAR/2009, ASTRAL/2009, CORAL/2014),[3] showing no benefit of revascularization on the renal function as compared to best medical treatment (BMT).[3–9] It should be noted, however, that some of the above-mentioned studies included a significant proportion of patients with low-grade stenosis and/or with normal or near-normal renal function, focusing therefore on low-risk patients.[9–13] In acute settings with rapidly progressive heart and renal failure, BMT versus revascularisation has been only rarely studied and some observational studies have shown improved outcomes in patients who underwent percutaneous transluminal renal angioplasty (PTRA) and stenting of the RAS.[13–20] Indeed, some subgroups of patients, including patients with a solitary functioning kidney,[20–23] could benefit from this more interventional approach.

We report the case of a patient with a significant renal artery stenosis of a single-functioning kidney who rapidly

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Table 1. Laboratory values at admission, on day of clinical degradation (just before PTRAS) and at discharge.

Laboratory values (reference range)	At admission (Day 0)	Time of degradation (Day 3)	At discharge (Day 9)
Haemoglobin (120–160 g/L)	92	86	88
Leucocytes (4–10 G/L)	16.0	11.1	10.8
Platelets (150–300 G/L)	208	196	206
Urea (2.8–7.0 mmol/L)	14.2	31.0	10.8
Creatinine (50–95 micromol/L)	183	384	88
Sodium (136–146 mmol/L)	139	136	141
Potassium (3.7–5.0 mmol/L)	4.5	7.4	4.4
C-reactive protein (<5 mg/L)	19.6	26.1	
TSH (0.27–4.20 mU/L)	1.16		
NT-pro-BNP (<300 ng/L)	4'3674	10'498	
Troponine T (<5 ng/L)	785	896	
Creatinine kinase (CK) (<170 U/L)	444		
CK- MB (<25 U/L)	32		
pH (7.35–7.45)	7.29	7.10	7.45
p02 (83–108 mmHg)	56	40	63
pC02 (35–48 mmHg)	37	65	41
cHC03- (21–28 mmol/L)	17.5	20.6	26.0
Lactate (0.7–1.8 mmol/L	0.6	2.8	
Proteinuria (<150 mg/L)	131	1620	
Albuminuria (<20 mg/L)	69	1069	
Urine albumin-creatinine ratio (<2.5 mg/mmol)	12.0	80.4	

developed an oligo-anuric renal dysfunction after the re-introduction of an angiotensin-II receptor blocker (irbesartan) and who rapidly normalised his renal function after the successful stenting of the stenosed artery.

Case report

A 66-year-old Caucasian male patient, with a history of diabetes, peripheral arterial disease, uncontrolled arterial hypertension and chronic renal insufficiency (baseline creatinine 150 µmol/L) was admitted for the second time in 1 month in a district hospital for hypertensive crisis with an acute pulmonary oedema and an acute-on-chronic renal dysfunction. Clinical examination at admission showed a patient in respiratory distress with bi-basal hypoventilation and crackles on both lung fields. Vital parameters showed a blood pressure of 230/110 mmHg, heart rate 94 bpm regular, respiratory rate 27/min, peripheral oxygen saturation 91% and temperature 36°C. Medical treatment included aspirin 100 mg/day, atorvastatin 20 mg/day, irbesartan 300 mg/day, metoprolol 50 mg/day, amlodipine 10 mg/day and a transdermic patch of nitroglycerin 10 mg/day. Laboratory values at admission (detailed in Table 1) showed an acute-on-chronic renal insufficiency (serum creatinine 183 µmol/L, urine albumin/creatinine ratio12 mg/mmol), a marked metabolic acidosis (pH 7.29, p02 56 mmHg, pC02 37 mmHg, cHC03 17.5 mmol/L), a moderate inflammatory state (C-reactive protein (CRP) 19.6 mmol/L, leucocytes 16.0 G/l) and signs of decompensated heart failure (NT-proBNP 4'3674 ng/L, high-sensitive cardiac troponin-T 785 ng/L). The electrocardiogram showed a regular sinus rhythm without evidence of conduction or repolarisation troubles, and the chest X-ray showed bilateral pleural effusion with vascular redistribution on pulmonary upper lobes. The urinalysis suggested the presence of a urinary infection (leukocyturia 15–20, erythrocyturia 2–4, nitrite positive) and the urine culture was positive for *Citrobacter Freundii*.

Supportive treatment with intravenous diuretic therapy and non-invasive ventilation was started, showing initial improvement. Antibacterial medication with single-dose of 2 g of intravenous ceftriaxone switched to oral ciprofloxacin 500 mg/day was administrated for the treatment of the urinary tract infection. Coronary angiography was delayed because of the worsening renal function, the absence of specific cardiac symptoms and the suspicion of a secondary Type 2 NSTEMI (non-ST elevation myocardial infarction) in the setting of acute heart failure. Irbesartan was initially stopped for 48 h and then re-introduced. Three days after admission, the clinical and the laboratory situation deteriorated with recurrent flash pulmonary oedema, necessitating orotracheal intubation, and oligo-anuria motivating the transfer to our hospital for emergency dialysis.

At time of transfer on day 3, laboratory values showed a rapidly worsening renal failure (serum creatinine 384 µmol/L) with severe hyperkalaemia and respiratory acidosis (see Table 1). Further investigations with duplex ultrasound of the renal arteries showed haemodynamically significant bilateral arterial stenosis (>80%), with a left atrophic kidney (7 cm length with a thinned cortex and several cysts) and a single-functioning right kidney having a normal morphology

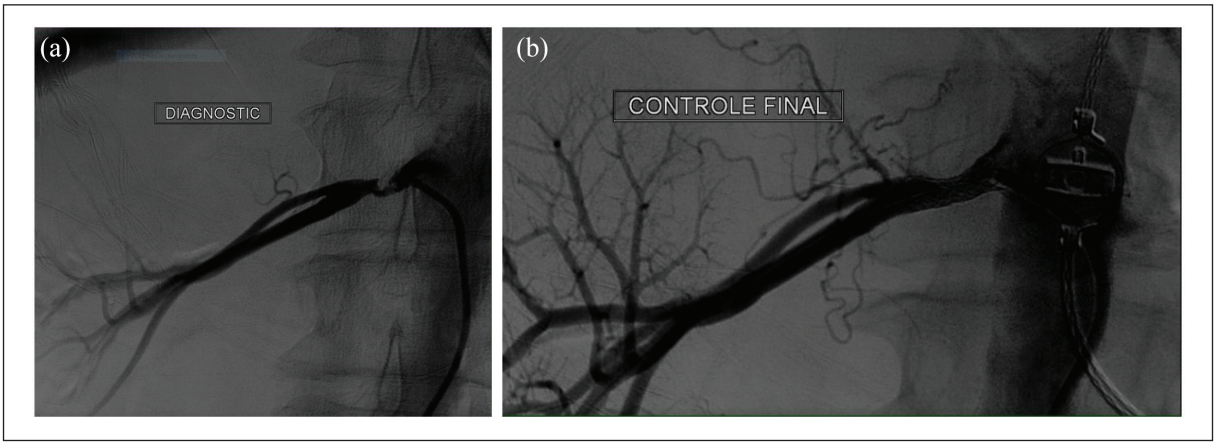

Figure 1. (a) Diagnostic arteriography of the right renal arteria showing an 80%–90% stenosis in its proximal part. (b) Result after percutaneous transluminal renal angioplasty and stent placement.

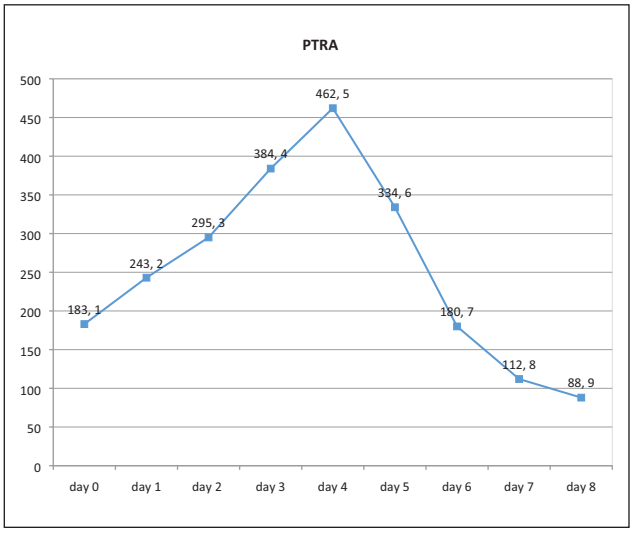

Figure 2. Serum creatinine levels time course during hospitalisation.

and whose length was 10 cm. A transthoracic echocardiogram showed a diastolic dysfunction and concentric hypertrophy with left ventricular ejection fraction of 65% and a haemodynamically non-relevant pericardial effusion.

Because of the rapid degradation of renal function with oligo-anuria, metabolic and respiratory acidosis, hyperkalaemia and uncontrolled blood pressure with systolic BP > 200 mmHg despite a maximal medical therapy (invasive ventilation, insulin-glucose, calcium gluconate, salbutamol, iv labetalol), it was decided to perform a renal arteriography. This exam showed a severe ostial stenosis of the right arteria (Figure 1(a)), successfully stented (Dynamic Renal 6 × 15 mm, Figure 1(b)). After PTRAS the patient showed a rapid clinical improvement without need of haemodialysis, with extubation the next day and a rapid normalisation of renal function (see Figure 2) and blood pressure as well as metabolic and electrolyte disturbances within 5 days. Notably, the creatinine level at discharge was much lower (88 µmol/L) than the pre-hospitalisation baseline levels (150 µmol/L), indicating that the pre-existing chronic renal insufficiency was already related to ischemic nephropathy. Blood pressure at discharge was well controlled with a monotherapy of lisinopril 5 mg/day and all other blood pressure medications could be discontinued.

Discussion

We present the case of recurrent acute pulmonary oedema, uncontrolled hypertension and rapidly progressive oligo-anuric renal failure in a patient with significant bilateral stenosis of the renal arteries with a right-sided single-functioning kidney. PTRA with stent placement of the stenosed right renal artery permitted a remarkably fast normalisation of the renal function, blood pressure and electrolyte disturbances.

Haemodynamically significant RAS is described as a narrowing of at least 70% of the lumen of one or both renal arteries, being responsible for 2%–5% of the cases of refractory hypertension.[24] Aetiology includes mostly atheromatous disease (90%), most of the time involving the proximal third of the renal arteria.[8,24] While angiotensin-converting enzyme inhibitors and angiotensin receptor blockers have been shown to be very effective in treating hypertension associated with unilateral RAS, they may induce a rapid degradation of the renal function or even anuria in patients with a significant bilateral stenosis or a single-functioning kidney,[25–27] as was the case in our patient.

The use of revascularisation with PTRA in patients with renal dysfunction is still controversial as BMT shows similar long-term outcomes, even though most studies have not been done in acute settings of decompensation. Some studies have shown that angioplasty, compared to BMT, may have a significant impact on blood pressure and permits a reduction of antihypertensive medication. Nonetheless, according to the

results of previous studies and meta-analysis, long-term outcomes appear to show no significant differences, especially on renal function, even with severe stenosis (i.e. >70% stenosis).[3–9] A recently published systematic review also showed no difference in mortality, cardiovascular events, blood pressure control or progression of renal dysfunction in PTRA versus BMT.[8] However, some subsets of patients may have a clear benefit of revascularisation. An analysis from a single centre showed that high-risk patients presenting with flash pulmonary oedema or both refractory hypertension and declining renal function who underwent revascularization had a reduced risk of death versus those medically treated (HR 0.4).[13] In this study, patients who only had a decline in kidney function or refractory hypertension did not show a similar benefit.

In current recommendations from the American College of Cardiology and the American Heart Association, the consensus statement concludes that all patients with RAS benefit from optimal medical therapy citing level 1A evidence.[1,2] In patients who fail to improve with medical therapy and continue to have refractory hypertension, worsening kidney function, or intractable heart failure, PTRA can be considered (as it was in the case of our patient).[1,3] The Society for Cardiovascular Angiography and Intervention consider appropriate revascularization by severe renal failure and global renal ischemia (Class IIB).[28] At present time, there are no recommendations concerning the indication of PTRA in the setting of acute renal dysfunction.

When revascularization is considered, first-line treatment is angioplasty with stent placement, as angioplasty alone shows higher prevalence of restenosis with similar outcomes.[1,21,24,28,29] Major complications of stenting are specialist- and lesion-dependant; they occur in less than 5% of the cases and include bleeding, pseudoaneurysms, distal embolisation, acute kidney injury and acute renal artery occlusion or dissection, sometimes resulting in terminal renal replacement.[7,9,21,30]

Interestingly, a systematic literature review made by Ma et al.[21] concerning patients with a solitary functioning kidney concluded that up to 77% of the patients with a single-functioning kidney showed an improvement or stabilisation of their renal function after PTRA. They report a particularly good outcome by a functioning kidney size of >80 mm.[21] This specific subgroup of patients with a single-functioning kidney could therefore particularly benefit from PTRA to preserve or improve renal function.

In an acute setting such as our patient, including rapidly progressive acute renal injury, severe hypertension and flash pulmonary oedema, revascularization versus BMT has been only marginally studied. According to observational studies and several case reports, a high proportion of patients in an acute setting improves after PTRA, with possible normalisation of renal function.[13–20] Furthermore, the rapidity of decline in renal function – which suggests the absence of an irreversible chronic parenchymal damage – is reported to be a good prognostic factor after PTRA.[10,30,31] A low urine albumin/creatinine ratio (<22.5 mg/g or <2.25 mg/mmol) has also been reported to be a good prognostic factor after PTRA[32] (note, however, that our patient, who was diabetic, had a much higher UACR). In such cases therefore, improvement of renal function can be expected, and may occur even months after PTRA. The remarkably fast normalisation of the renal function (5 days) of our patient has not yet been described.

Conclusion

In summary, present guidelines recommend PTRA essentially for patients having a severe renal artery stenosis and presenting with uncontrolled or refractory hypertension but not for those with renal dysfunction. Some observational studies however suggest a benefit of PTRA in specific subgroups of patients with renal dysfunction, such as those with acute progression of kidney dysfunction and/or those with a single-functioning kidney, with good chances of improvement or even normalisation of the renal function after PTRA. Further studies focusing on these subgroups of patients are therefore warranted in order to establish more specific guidelines. In the meantime, stenting of the renal arteria should be considered in patients with acute kidney injury related to RAS and in those with RAS of a single-functioning kidney, as outcomes can be remarkably improved.

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Author contributions

C.S. performed the literature research, wrote the draft and was involved in the patient's care. O.M.H. reviewed the literature and the draft. D.P. was involved in the patient care and revised the draft. E.D. was involved in the patient's care and revised the draft critically for important intellectual content.

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Informed consent

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