

# Intravascular ultrasound-guided shockwave lithotripsy of heavily calcified bilateral renal artery stenosis: a case report

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Background	Calcified lesions represent a hard obstacle to overcome in renal arteries, particularly when renal angioplasty repre- sents the only feasible course of action in the setting of high-risk bilateral renal artery stenosis (RAS) with refrac- tory systemic hypertension and recurrent flash pulmonary oedema.	
Case summary	We herein report a case of symptomatic bilateral severely calcified RAS, treated successfully with intravascular ultrasound (IVUS)-guided coronary and peripheral intravascular shockwave lithotripsy systems and stenting.	
Discussion	Intravascular shockwave lithotripsy is an attractive modality for the treatment of challenging, heavily calcified renal arteries that combines the calcium-disrupting capability of lithotripsy with the familiarity of balloon catheters to facilitate proper stent deployment.	
Keywords	Renal artery stenosis • Intravascular shockwave lithotripsy • Intravascular ultrasound • Angioplasty • Pulmonary oedema • Refractory hypertension • Case report	

#### Learning points

- We described a case of high-risk Pickering syndrome with recurrent flash pulmonary oedema due to heavily calcified bilateral renal artery stenosis (RAS) successfully treated with intravascular ultrasound-guided intravascular shockwave lithotripsy (IVL) and stenting with dramatic normalization of renal function and blood pressure 2 weeks after the procedure.
- Intravascular shockwave lithotripsy is a novel technology with a unique mechanism of action that powerfully cracks calcium in renal arteries to optimize a percutaneous intervention for complex calcified RAS.
- Transradial renal artery stenting carry less risk of local complications than the femoral approach and allows multipurpose guide catheter to be engaged coaxially into the renal artery, minimizing the risk of guide-induced ostial dissection. Furthermore, radial access will allow early ambulation and discharge after an uncomplicated procedure.
- Randomized trials are warranted to study the safety and efficacy of IVL in treating calcified RAS.

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#### Introduction

Percutaneous intervention for renal artery stenosis (RAS) may represent the only feasible course of action in the setting refractory systemic hypertension; flash pulmonary oedema (FPE) and speedy worsening of renal function, although evidences from major studies such as The Angioplasty and Stenting for Renal Artery Lesions 'ASTRAL trial' and Cardiovascular Outcomes in Renal Atherosclerotic Lesions 'CORAL trial' were unfavourable.<sup>1,2</sup>

The conclusion extracted from those trials are considered inconclusive, and the current guidelines have not yet been changed and recommended renal angioplasty as a sensible choice for patients with high-risk bilateral or solitary RAS.<sup>3</sup>

Despite the substantial progress in the endovascular interventions, undoubtedly, debulking of calcified renal artery lesions is a major challenge but remains to be a crucial step before stent deployment to avoid the negative impact of calcifications on stent expansion and drug elution.<sup>3</sup> Recently, the use of intravascular shockwave lithotripsy (IVL) for modification of severely calcified coronary and peripheral atherosclerotic plaques showed pronounced success in terms of feasibility and safety.<sup>4</sup>

Here, we present a case of recurrent episodes of hypertensive FPE due to heavily calcified bilateral RAS successfully treated with intravascular ultrasound (IVUS)-guided IVL and stenting.

#### Timeline

Eight months before admission Two months before admission	Refractory systemic hypertension on Amlodipine/ Valsartan/hydrochlorothiazide combination therapy 10 mg/160 mg/12.5 mg o.d., Carvedilol 25 mg b.i.d., and Aldactone 25 mg o.d. First episode of unexplained flash pulmonary oe- dema with mild renal impairment (Creatinine 120 μmol/L, reference range 44–88 μmol/L)
One month	- Recurrent presentations to the emergency de-
before	partment with flash pulmonary oedema, uncon-
admission Day 1	<ul> <li>trolled hypertension, oliguria, and progressive worsening of renal function (Creatinine 165 µmol/L, reference range 44–88 µmol/L).</li> <li>Amlodipine/Valsartan/hydrochlorothiazide combination therapy was changed to amlodipine 10 mg o.d., hydralazine 25 mg TDS, and furosemide 60 mg b.i.d. along with Carvedilol 25 mg b.i.d. and Aldactone 25 mg o.d.</li> <li>Referred to our cardiac centre for evaluation</li> <li>On admission: serum Creatinine 225 µmol/L (reference range 44–88 µmol/L), serum potassium 4.4 (reference range 3.6–5.2), PH 7.36 (reference range 7.35–7.45).</li> <li>Coronary angiogram showed no flow-limiting disease</li> <li>Renal angiogram showed bilateral heavily calcified high-grade ostioproximal renal artery stenosis</li> <li>After the procedure, she had acute pulmonary oedema and anuria, which necessitated haemodialysis</li> </ul>

Continued	
Day 2	<ul> <li>Successful intravascular ultrasound-guided shock- wave lithotripsy and stenting of bilateral renal artery stenosis</li> <li>Second haemodialysis session to enhance renal recovery</li> </ul>
Day 3	<ul> <li>Dramatic improvement of urine output and renal parameters without further haemodialysis sessions</li> </ul>
Day 4	Good post-procedural recovery and successful discharge to home on Amlodipine 10 mg o.d., furosemide 40 mg o.d., and Carvedilol 12.5 mg b.i.d. (systolic blood pressure < 140 mmHg).
Two weeks after discharge	She was asymptomatic. Renal parameters almost normalized (Creatinine 96 µmol/L, reference range 44–88 µmol/L) with adequate blood pres- sure control.
3 months after discharge	Complete recovery of renal function (Creatinine 81 μmol/, reference range 44–88 μmol/L) with good blood pressure control on amlodipine 10 mg o.d. only.

#### **Case presentation**

A 72-year-old Bahraini female known to have type 2 diabetes mellitus, resistant systemic hypertension, hyperlipidaemia, and peripheral artery disease (PAD) with a history of stenting distal abdominal aorta (12 mm  $\times$  40 mm S.M.A.R.T control self-expanding stent) and right common iliac artery (7 mm  $\times$  17 mm Express LD) in 2014 was referred to our cardiac centre for evaluation of repeated episodes of FPE.

On arrival, she was in mild dyspnoea and oliguric. Her blood pressure was 182/105 mmHg, and her heart rate was 109 b.p.m. Physical examination has revealed bilateral basal crackle with no audible cardiac murmur and periumbilical, high-pitched bruits. Her electrocardiogram showed QRS voltage criteria for left ventricular hypertrophy, which was confirmed with a transthoracic echocardiogram that revealed concentric left ventricular hypertrophy with Grade II diastolic dysfunction and normal left ventricular size and systolic function with no regional wall motion abnormality. Baseline Creatinine was 225  $\mu$ mol/L (reference range 44–88  $\mu$ mol/L) and hence we performed the coronary angiogram with minimal contrast (18 cc only) to rule out underlying coronary artery disease in view of multiple risk factors and troponinaemia, which showed coronary calcifications but no flow-limiting disease in the epicardial coronaries. Since the patient had resistant systemic hypertension with abdominal

bruits, non-selective renal angiography was conducted and showed bilateral heavily calcified high-grade ostioproximal RAS. While we were sorting out a strategy for *ad hoc* intervention for the tight renal arteries she desaturated, her blood pressure raised to 240/130 mmHg, her heart rate increased to 130 b.p.m., and her respiration rate was 26/min with diffuse bilateral fine crackles. Considering acute florid pulmonary oedema and being anuric despite large dose of diuretics, we aborted the procedure and timely inserted the right internal jugular dialysis line and transferred her to the cardiac care unit for urgent haemodialysis.

The next day, after full stabilization, we brought her back to the Cath lab for renal angioplasty. We decided for the left transradial approach for two reasons: the left radial artery has a shorter distance to the renal arteries than the right radial artery and the angles of the renal arteries off the aorta favour a radial approach than a retrograde femoral access.

Under local anaesthesia, the left radial artery access was achieved with a 7-Fr sheath. We managed to navigate a 90 cm sheath across an unexpected left subclavian stenosis with balloon dilatation (*Figure 1A* and *B*) and slide it subsequently across a tortuous descending aorta using NaviCross microcatheter and Terumo Glidewire Advantage, which was exchanged to Hi-Torque supracore guidewire thereafter to land the sheath into the abdominal aorta.

A 7-Fr multipurpose guide catheter engaged the right renal artery, and a selective renal angiogram confirmed the presence of critical calcified stenosis at the proximal part of the right renal artery (*Figure 2A*).

The lesion crossed with Hi-Torque BMW 0.014  $\times$  300 cm guidewire. Intravascular ultrasound pullback showed a very high superficial and deep calcium burden in the proximal segment of the right renal artery with a 5.2 cm reference vessel diameter (*Figure 2B*). Considering severe calcifications, we decided to proceed with shockwave lithotripsy using a 4.0 mm  $\times$  12 mm, 138 cm shockwave C2 IVL balloon with a total of 80 pulses. Fair expansion of the 4.0 mm IVL balloon at 6 atmospheres was achieved (*Figure 2C* and *D*). Now, without further balloon dilation, a 5.0 mm  $\times$  18 mm Resolute Onyx stent was deployed at 14 atmospheres with a good final angiographic result (*Figure 2E*).

The same procedure was applied for the left renal artery after confirmation of tight osteoproximal lesion with selective injection (Figure 3A). This time, the IVUS study showed a larger vessel diameter of 7.1 cm with severe semi-circumferential calcium arc (Figure 3B) that we overcame successfully with 6.0 mm  $\times$  60 mm, 110 cm shockwave M5 IVL balloon (10 rounds each of 30 pulses) at 6 atm (Figure 3C and D). Once the lithotripsy treatment is completed, a 7.0  $\text{mm}\times 17\,\text{mm}$  Express SD Renal stent was deployed at 10 bar. Final angiography demonstrated an excellent position of the stent and confirmed patency of the left renal artery (Figure 3E). The patient tolerated the procedure well and was transferred to the cardiology ward for further monitoring. A few hours later, we noticed a dramatic improvement of blood pressure measurements and urine output. She required one more haemodialysis session to enhance renal recovery and minimize the risk of contrast induced-nephropathy and was successfully discharged to home 48 h post-procedure.

In the 2-week of follow-up, she was asymptomatic, serum creatinine almost normalized (96  $\mu$ mol/L, reference range 44–88  $\mu$ mol/L)



**Figure 1** (*A*, *B*) Balloon dilatation of left subclavian artery stenosis to enable the passage of a 7-Fr guidcatheter via left transradial access for renal angioplasty.

with adequate blood pressure control. Three months after discharge, renal function recovered completely and return to normal (Creatinine 81  $\mu$ mol/L, reference range 44–88  $\mu$ mol/L) with good blood pressure control on amlodipine 10 mg o.d. only.

In our institution, there are no standards for routine duplex or imaging follow-up after renal angioplasty unless mandated clinically.

#### Discussion

Transradial renal artery angioplasty and stenting are very practical and carry less risk of local complications than the femoral approach.<sup>5</sup> Because of the angle of renal artery take-off (*Figures 2A* and 3A), a



**Figure 2** Intravascular ultrasound-guided intravascular shockwave lithotripsy for right renal artery stenosis. (A) Selective right renal angiogram showed critical calcified stenosis at the proximal part of the right renal artery. (B) Intravascular ultrasound pullback confirmed very high superficial and deep calcium burden. (C,D) Fair expansion of the 4.0 mm  $\times$  12 mm intravascular shockwave lithotripsy balloon. (E) Good post-stenting angiographic result.

craniocaudal approach was another attractive reason to go radial, which allows multipurpose guide catheter to be engaged coaxially into the renal artery, minimizing the risk of guide-induced ostial dissection. Furthermore, radial access will allow early ambulation and discharge after an uncomplicated procedure.

Knowledge of anthropometric measurements and proportions of the human body is essential; thus, the left radial access is preferable to the right radial access, because the former saves 10–15 cm of travelling distance across the aortic arch.<sup>6</sup> In most cases, a 110 cm guide catheter can reach the renal arteries from the left radial access regardless of length and tortuosity at the left radial and subclavian arteries and the aorta.<sup>7</sup>

Different clinical trials such as DISRUPT coronary artery disease, DISRUPT PAD, and DISRUPT Below-the-Knee demonstrated good results of IVL in terms of safety and procedural success in the coronary, popliteal, and femoral arteries, but none has included renal arteries.  $^{\rm 8,9}$ 

#### Conclusion

Intravascular shockwave lithotripsy is an attractive modality for the treatment of challenging, severely calcified renal artery that combines the calcium-disrupting capability of lithotripsy with the familiarity of balloon catheters to facilitate proper stent deployment. Future trials will enhance our discernment into the safety, effectiveness, and position of IVL in the algorithms for the management of heavily calcified symptomatic bilateral RAS.



**Figure 3** Intravascular ultrasound-guided shockwave lithotripsy for left renal artery stenosis. (A) Selective left renal angiogram showed critical calcified stenosis at the proximal part of the left renal artery. (B) Intravascular ultrasound pullback confirmed sever semi-circumferential calcium arc. (C,D) Good expansion of the 6.0 mm  $\times$  60 mm intravascular shockwave lithotripsy balloon. (E) Excellent post-stenting angiographic result.

## Lead author biography



Dr Husam A. Noor is the head of cardiac catheterization laboratories at Mohammed bin Khalifa Cardiac Centre (MKCC) in the Kingdom of Bahrain and a consultant interventional cardiologist as well as a senior lecturer at the royal college of surgeon Ireland-medical university of Bahrain. He completed cardiology fellowship at Loma Linda University, California followed by interventional cardiology fellowship at University of Massachusetts. In addition, he had advanced training in the field of peripheral endovascular interventions at Arizona Heart Hospital. He has established the chronic total occlusion, peripheral angioplasty and Transcatheter aortic valve replacement programs at MKCC.

### Supplementary material

Supplementary material is available at *European Heart Journal - Case* Reports online.

**Slide sets:** A fully edited slide set detailing these cases and suitable for local presentation is available online as Supplementary data.

**Consent:** The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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