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Flash pulmonary edema and resistant hypertension due to renal artery in-stent restenosis: a case report

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Abstract

Renal artery in-stent restenosis is an uncommon but serious cause of resistant hypertension and flash pulmonary edema. We present the case of a 75-year-old woman with extensive multidistrict atherosclerosis and a history of multiple endovascular procedures, who presented to the emergency department with recurrent pulmonary edema due to uncontrolled hypertension. Despite maximal antihypertensive therapy, her blood pressure remained severely elevated, with evidence of acute kidney injury. Doppler ultrasound revealed restenosis of the right renal artery stent, characterized by a tardus-parvus waveform, along with an atrophic left kidney. The patient underwent successful right renal artery lithotripsy and stenting, resulting in restoration of renal perfusion and subsequent improvement in blood pressure control and pulmonary congestion. In patients with resistant hypertension and recurrent flash pulmonary edema, prompt recognition of renal artery stenosis, the so-called *Pickering syndrome*, or in-stent restenosis as in the present case, and timely endovascular reintervention can be lifesaving.

Introduction

Renal Artery Stenosis (RAS) is a well-recognized cause of secondary hypertension and heart failure. Atherosclerotic RAS induces activation of the Renin–Angiotensin–Aldosterone System (RAAS), leading to sodium retention, volume overload, and resistant hypertension.[1,2]
In-Stent Restenosis (ISR) represents a therapeutic challenge and may precipitate recurrent flash pulmonary edema, also known as Pickering syndrome.[3,4]
Endovascular revascularization can provide clinical benefit in selected patients.[5] We report the case of severe, refractory hypertension and acute pulmonary edema secondary to in-stent restenosis of the right renal artery in a patient with a solitary functioning right kidney, successfully treated with shockwave lithotripsy and stent implantation.

Case Report

A 75-year-old woman with severe atherosclerotic disease presented to the emergency department on October 19, 2025, with acute dyspnea and hypertensive pulmonary edema.
Her medical history included left renal artery Percutaneous Transluminal Angioplasty (PTA) in 2013, infrarenal aortic stenting in 2014, right renal artery PTA in 2021, bilateral iliac kissing stents in 2021, and coronary stenting in 2022 and 2025. Comorbidities included atrial fibrillation, severe aortic stenosis treated with Transcatheter Aortic Valve Implantation (TAVI), Chronic Obstructive Pulmonary Disease (COPD), and dyslipidemia.
Since July 2025, the patient had experienced three hospitalizations for hypertensive pulmonary edema, during which her heart failure was managed and antihypertensive therapy repeatedly adjusted; she was discharged after the last hospitalization on four antihypertensive medications. During these admissions, renal artery Doppler ultrasound and renal scintigraphy had been performed.
Doppler ultrasound revealed restenosis of the right renal artery stent (peak velocity >3 m/s, tardus-parvus waveform, resistive index 0.60–0.65), while the left kidney appeared atrophic. Captopril-enhanced renal scintigraphy demonstrated limited right-sided renin activation, likely attributable to both renal artery stenosis and pre-existing hypertensive microangiopathy, supporting a conservative management approach at that time.
Upon arrival at the emergency department, she was alert with a Glasgow Coma Scale (GCS) score of 15, hypertensive with a blood pressure (BP) of 200/100 mmHg, tachydyspneic with an oxygen

saturation (SpO_2) of 89% and a Respiratory Rate (RR) of 28 breaths/min, and apyretic. Arterial blood gas analysis on room air showed: pH 7.52, PCO_2 31 mmHg, PO_2 46 mmHg, and HCO_3^- 26.8 mmol/L. Point-of-care ultrasound demonstrated pulmonary B-lines with bilateral pleural effusions (~2 intercostal spaces). Echocardiography revealed concentric left ventricular hypertrophy without regional wall motion abnormalities and evidence of diastolic dysfunction. The left atrium was dilated (area 28 cm^2) with mild mitral regurgitation, while the aortic prosthetic valve appeared normally functioning. The inferior vena cava measured 23 mm in diameter, with respiratory collapsibility <50%.

The admission electrocardiogram showed sinus rhythm at 70 bpm and inverted T waves in the lateral leads, consistent with ventricular hypertrophy.

Chest X-ray confirmed pulmonary congestion (Figure 1).

Laboratory tests showed mild anemia (Hb 11.9 g/dL), sodium 146 mEq/L, potassium 3.0 mEq/L, NT-proBNP>70,000 pg/mL, and elevated creatinine (1.41 mg/dL) with a Glomerular Filtration Rate (GFR) of 27 mL/min consistent with Acute Kidney Injury (AKI).

She received oxygen via Venturi mask (40%), intravenous antihypertensive therapy (clonidine, labetalol, urapidil), and continuous furosemide infusion (10 mg/h), and was subsequently admitted to our high-dependency unit. Despite maximal therapy (IV labetalol up to 60 mg/h, amlodipine 10 mg, doxazosin 4 mg twice daily, nitroglycerin patch, furosemide 20 mg/h), systolic blood pressure remained >180 mmHg, with persistent pulmonary congestion and oliguric AKI (creatinine 1.81 mg/dL, GFR 21 mL/min).

Given the inefficacy of pharmacological treatment, a new vascular surgery consultation was obtained. Considering the inability to achieve adequate blood pressure control despite five different antihypertensive agents, the progressive decline of the only functioning kidney, and the contraindication to renal denervation due to pre-existing stenosis and renal impairment, the vascular team ultimately decided to proceed with a high-risk endovascular revascularization attempt, despite the patient's history of multiple prior revascularization procedures and the substantial risk of complications.

On October 22, 2025, the patient underwent intravascular lithotripsy (Shockwave balloon) and stenting of the right renal artery under CO_2 angiographic guidance. Final angiography demonstrated restored patency of the artery and its branches, with preserved parenchymal perfusion and no evidence of residual stenosis or procedural complications.

Intraoperative CO_2 angiography of the renal artery revealed severe calcified in-stent restenosis of the right renal artery (Figure 2a). The Shockwave coronary balloon was positioned across the calcified lesion at the renal artery ostium, and shockwaves were subsequently delivered to the

lesion (Figure 2b). This resulted in a significant reduction of the stenosis, allowing deployment of a stent at the renal ostium. Final angiography confirmed correct stent positioning with no residual stenosis (Figure 2c).

Doppler ultrasound performed before and after the procedure demonstrated a marked reduction in Peak Systolic Velocity (PSV) and normalization of the waveform (Figure 3).

Post-procedure, the patient's BP rapidly improved, oxygen requirements decreased, and renal function stabilized.

The following table summarizes the changes in BP, Heart Rate (HR), oxygen therapy and SpO_2, urine output, and GFR during the pre- and post-procedural hospital stay (Table 1).

The patient was subsequently transferred to the internal medicine ward, where antihypertensive therapy was gradually tapered and ultimately discontinued in the setting of stable BP (130/70 mmHg). Oxygen therapy was also withdrawn, and renal function and serum electrolyte levels had normalized prior to discharge (creatinine 0.91 mg/dL, sodium 139 mEq/L, potassium 3.6 mEq/L). She was discharged on October 27, 2025.

In February 2026, the patient underwent a follow-up evaluation by the vascular surgery team, including a renal artery Doppler ultrasound, which demonstrated satisfactory renal perfusion. She was normotensive and reported full clinical well-being.

Discussion

Renovascular hypertension results from impaired renal perfusion and subsequent activation of the RAAS. This patient exhibited progressive renal dysfunction, volume overload, and refractory hypertension, consistent with hemodynamically significant RAS.

Bilateral critical renal artery stenosis or critical stenosis of a solitary functioning kidney may trigger recurrent flash pulmonary edema, also known as Pickering syndrome. Since Pickering syndrome was first described in *Lancet* in 1988, 16 case reports, 2 case series, and 7 clinical studies have been published, collectively including 87 patients presenting with flash pulmonary edema and bilateral renal artery stenosis.[3,4]

Pickering syndrome is considered a rare condition, largely because renal artery stenosis often remains underdiagnosed. However, renovascular hypertension represents the most common cause of secondary hypertension, and renal artery stenosis accounts for a variable proportion of cases, occurring in approximately 10–14% of patients with coronary artery disease and 24–35% of those with peripheral arterial disease.[6]

Key warning signs suggesting Pickering syndrome include a history of poorly controlled hypertension (often requiring multiple antihypertensive agents), recurrent episodes of flash

pulmonary edema characterized by rapid onset of symptoms, oligoanuria, and acute worsening of renal function, often associated with hypokalemia.

Echocardiography typically demonstrates concentric left ventricular hypertrophy and diastolic dysfunction, occasionally accompanied by moderate-to-severe mitral regurgitation. When afterload increases due to uncontrolled hypertension, the left ventricle compensates by increasing end-diastolic volume, which, in the presence of diastolic dysfunction, leads to a marked rise in left ventricular end-diastolic pressure. This pressure is transmitted to the left atrium and pulmonary venous circulation, resulting in post-capillary pulmonary hypertension.[7,8] The increase in left atrial end-diastolic pressure may be associated with dilation of the mitral annulus, resulting in acute mitral regurgitation or worsening of pre-existing valvular disease, which further exacerbates post-capillary pulmonary hypertension and consequently pulmonary edema.

At this stage, rather than merely managing hypertension and pulmonary congestion along with their complications, it is crucial to investigate the underlying causes of arterial hypertension, particularly to determine why sudden pulmonary edema recurs and why progressive renal dysfunction develops, often in the presence of hypokalemia.

Since renovascular disease is the most frequent cause of secondary hypertension, evaluation of renal morphology and vascularization should be an integral part of the patient's diagnostic work-up.

Renal ultrasound allows assessment of kidney length, while color Doppler imaging provides information on renal artery perfusion through measurement of PSV. In addition, intraparenchymal resistance indices and the renal-to-aortic PSV ratio (RAR) can be calculated. Criteria suggestive of significant renal artery stenosis include a longitudinal kidney diameter <8 cm, renal artery PSV >180 cm/s, RAR >3.5, and an intraparenchymal tardus-parvus waveform pattern.

In the absence of access to renal artery ultrasound, direct abdominal Computed Tomography (CT) may be sufficient to identify aortic atherosclerosis at the origin of the renal arteries.[7] Alternatively, as recommended by current guidelines, the diagnostic work-up may include CT Angiography (CTA), with efforts to minimize contrast volume, or Magnetic Resonance Imaging (MRI).[9] Once clinical suspicion is raised using these indirect techniques, the recommended approach is to proceed with selective angiography, using the minimal required contrast volume (approximately 15 mL),[7] or perform carbon dioxide angiography, as in our case.

When hypertension is suspected to be caused by bilateral renal artery stenosis or stenosis of a solitary functioning kidney, RAAS inhibitors should be avoided, as they can suppress compensatory mechanisms that preserve renal perfusion, potentially leading to acute deterioration of kidney

function.

Scintigraphy has the limitation of potentially being non-diagnostic in the presence of microvascular damage, such that current guidelines do not recommend its routine use.[9] In our patient, scintigraphy performed in July did not clearly identify the cause of reduced renin activation in the functioning kidney, which could reflect either renal artery stenosis or parenchymal microangiopathy. Given the periprocedural risk in a patient with severe vasculopathy and a history of stent restenosis, an initial conservative approach was chosen. However, recurrent episodes of flash pulmonary edema associated with uncontrolled hypertension and renal dysfunction ultimately raised suspicion of Pickering syndrome, prompting definitive angiographic evaluation.[10]

Intravascular Lithotripsy (IVL) is emerging as an effective modality for modifying calcified lesions and improving stent expansion.[11] Endovascular revascularization may restore renal perfusion, improve BP control, and prevent further episodes of pulmonary edema in appropriately selected patients.[12]

Conclusions

This case underscores the importance of recognizing the link between uncontrolled hypertension, recurrent flash pulmonary edema and renal insufficiency, as these conditions may all arise from a single, treatable cause. In patients with prior renal artery stenting, in-stent restenosis should be considered and promptly identified. Intravascular lithotripsy–assisted renal artery revascularization is an effective option to restore renal perfusion and stabilize clinical status when medical therapy fails.

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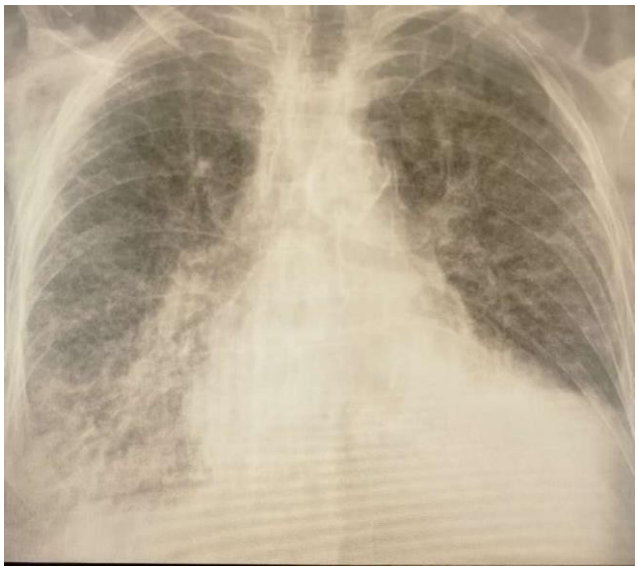

Figure 1. Pulmonary congestion on chest X-ray.

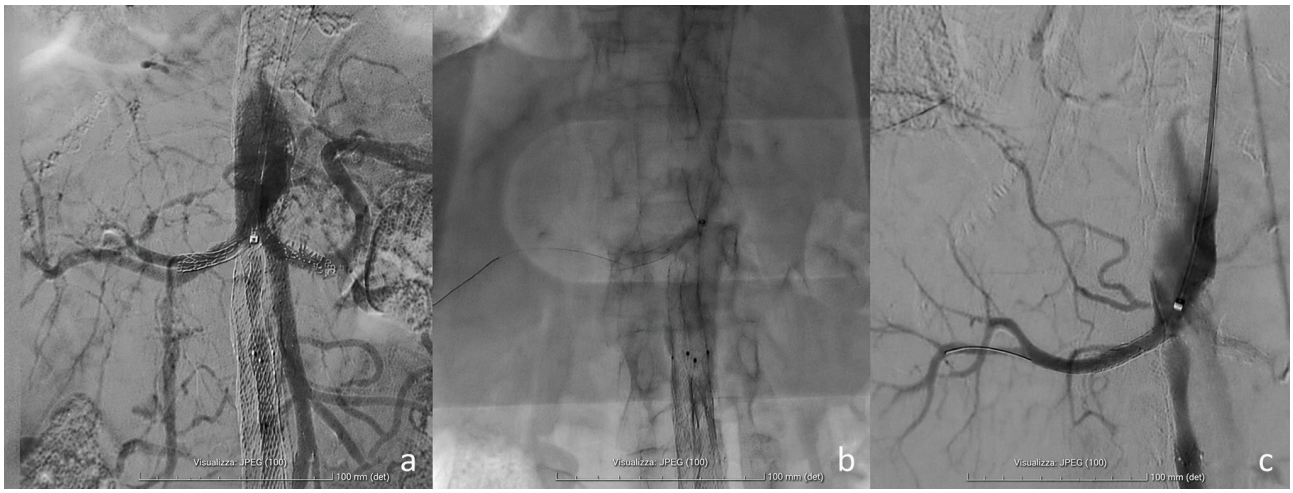

Figure 2. Intraoperative CO_2 angiography of the right renal artery. a) Severe calcified in-stent restenosis of the right renal artery; b) Intravascular lithotripsy with Shockwave coronary balloon; c) Final angiography after stent implantation showing no residual stenosis.

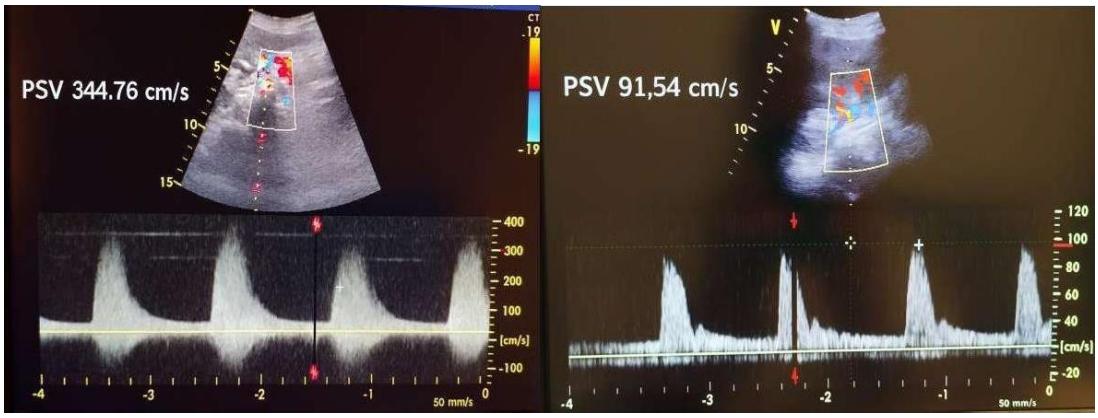

Figure 3. Doppler pathological flow with a hemodynamically significant stenosis of the right renal artery (on the left) and normalized flow after correction of the renal stenosis (on the right).

Table 1. Hemodynamic and renal function parameters pre- and post-procedure. SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; HR, Heart Rate; FiO_2, Fraction of Inspired Oxygen, SpO_2, peripheral oxygen saturation; VM, Venturi Mask; HFNC, High Flow Nasal Cannula; LFNC, low-flow nasal cannula; GFR, Glomerular Filtration Rate.

	T0h pre-procedure	**T24 h pre-procedure**	**T 48 hpre-procedure**	**T0 after-procedure**	**T24 after-procedure**
SBP	220	180	170	124	98
DBP	120	60	70	60	60
HR	68	65	60	60	60
FiO_2 Oxygen flow	VM 60% 15 l/min	HFNC 60% 60l/min	HFNC 40% 60l/min	LFNC 4l/min	LFNC 2l/min
SpO_2	95%	96%	96%	99%	99%
GFR	27 ml/min	21 ml/min	23 ml/min	34 ml/min	39 ml/min
urine output	60 ml/h	50 ml/h	20 ml/h	100 ml/h	120 ml/h

Contributions: Rosa Di Donato, patient management, clinical data collection, and drafting of the case report; Sara Speziali, endovascular management of the patient and drafting of the section related to the interventional procedure; Francesca Caldi, clinical management and data collection. Francesca Innocenti, clinical management and contribution to manuscript drafting; Lorenzo Pelagatti, clinical management and literature review; Raffaele Pulli, critical revision and final approval of the vascular management section; Simone Vanni, clinical management, literature review, critical revision, and final approval of the manuscript.

Conflict of interest: the authors declare no potential conflict of interest, and all authors confirm accuracy.

Ethics approval and consent to participate: no ethical committee approval was required for this case report by the Department, because this article does not contain any studies with human participants or animals. Informed consent was obtained from the patient included in this study.

Patient consent for publication: the patient gave her written consent to use her personal data for the publication of this case report and any accompanying images.

Availability of data and materials: all data underlying the findings are fully available.

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