

# Improvement of left ventricular filling and pulmonary artery pressure following unilateral renal artery total occlusion stenting in a patient with recurrent congestive heart failure complicated by renovascular hypertension and renal failure

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## Abstract

Recurrent congestive heart failure related to renal artery disease is an important clinical entity that is typically observed in bilateral renal artery stenosis or solitary functioning kidney. However, the relationship between heart failure and unilateral renal artery disease, especially that with total occlusion, remains unclear. We report a successful management by unilateral renal artery total occlusion stenting with an evidence of improvement of left ventricular filling and pulmonary artery pressure in case of a patient suffering from medical therapy resistant recurrent congestive heart failure with preserved ejection fraction.

**Keywords** HFpEF; Unilateral renal artery disease; Total occlusion; Stent; Left ventricular filling; Pulmonary artery pressure

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## Introduction

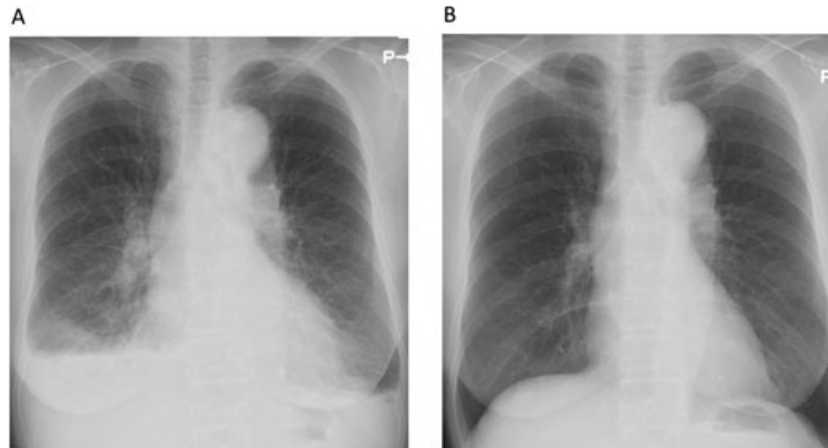
Recurrent congestive heart failure because of renal artery disease can be caused by volume overload or peripheral arterial vasoconstriction. This important clinical entity is typically observed in bilateral renal artery stenosis or solitary functioning kidney.<sup>1–3</sup> However, amid a broad spectrum of clinical features in patients with renal artery disease, the relationship between heart failure and unilateral renal artery disease, especially that with total occlusion, remains unclear. We report a successful management by unilateral renal artery total occlusion stenting with an evidence of improvement of left ventricular filling and pulmonary artery pressure in case of a patient suffering from medical therapy resistant recurrent congestive heart failure with preserved ejection fraction.

## Case report

A 63-year-old female with a history of twice flash pulmonary edema requiring hospitalization during the last 2 months was

referred to our unit for the treatment of recurrent congestive heart failure (Figure 1A). Right renal artery total occlusion was documented at the first hospitalization. She was also complicated by resistant hypertension (171/99 mmHg on four medications), progressive renal failure (fluctuation in creatinine between 1.74 and 3.21 mg/dL), diabetes, and dyslipidemia, and was intolerant of angiotensin II receptor blocker (Table 1). Transthoracic echocardiography demonstrated an elevated left ventricular filling (E/e' 19.5) and pulmonary hypertension (tricuspid regurgitation peak gradient; TRPG 44 mmHg) with a preserved ejection fraction (Table 1). On cardiac scintigraphy, no evidence of myocardial ischemia was observed. Renal ultrasonography demonstrated bilateral preservation of kidney size (right kidney 10.5 cm, left kidney 10.8 cm), whereas the resistive index measured in the renal artery was normal on the right side and high on the left (right kidney 0.59, left kidney 0.87). Colour signal was absent in the right renal artery, and a peak systolic velocity in the left renal artery was within normal limit (45 cm/s). Multidisciplinary discussion led to the decision to attempt endovascular therapy for unilateral renal artery total occlusion because of

**Figure 1** (A) Chest X-ray in a standing position taken before renal artery stenting reveals congestive heart failure with pleural effusion. (B) Chest X-ray in a standing position after renal artery stenting reveals disappearance of congestive heart failure.



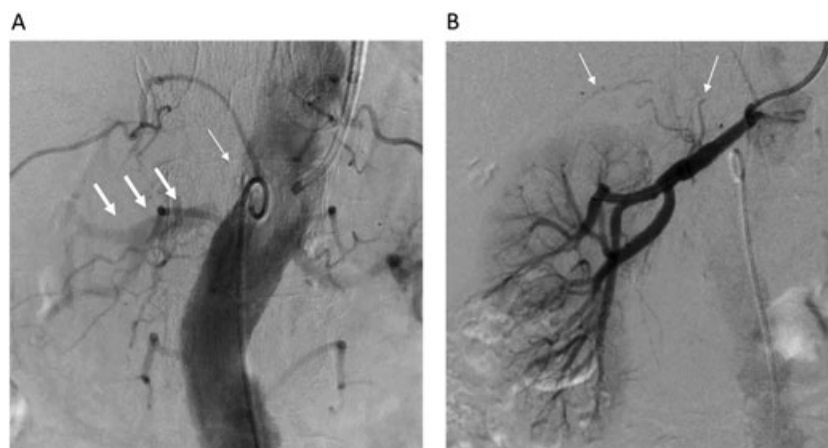
**Table 1** Clinical and echocardiographic parameters; pre and post stenting for unilateral renal artery total occlusion

	Pre	1 day	1 month	6 month
Body weight (kg)	47.8	44.8	43	44
Brain natriuretic peptide (pg/mL)	223.1	227.6	20.1	37
NYHA class	IV	I	I	I
Heart rate (bpm)	72	65	64	61
Ejection fraction (%)	67	60	61	65
E/e'	19.5	15.2	12	10.6
TRPG (mmHg)	44	26	20	18
Left atrial dimension (mm)	43	42	36	37
Left ventricular end-diastolic dimension (mm)	49	46	44	43
Left ventricular end-systolic dimension (mm)	31	30	28	27
Systolic blood pressure (mmHg)	171	104	114	118
Diastolic blood pressure (mmHg)	99	62	79	76
Mean blood pressure (mmHg)	123	76	91	90
Number of antihypertensive agent	4	3	1	1
Breakdown of antihypertensive agents	Nifedipine 80 mg Amlodipine 10 mg Azosemide 30 mg Doxazosin 2 mg	Amlodipine 10 mg Azosemide 30 mg Doxazosin 2 mg	Amlodipine 10 mg	Amlodipine 10 mg
Serum creatinine (mg/dL)	2.03	1.35	1.1	1
eGFR (mL/min/1.73 m <sup>2</sup> )	20.1	31.4	39.3	43.7
U-albumine (mg/L)	2359	84	NA	NA
Serum potassium (mEq/L)	4.3	4.4	4.3	4.2
Renin (ng/mL/h)	23.1	3.9	NA	4.4
Aldosterone (pg/mL)	33.2	174	NA	153

failed medical management and inability to wean from intravenous atrial natriuretic peptide and furosemide. Baseline angiography demonstrated a flush occlusion of the right ostial renal artery with a reconstituted distal segment and a patent left renal artery (*Figure 2A*). Immediately after successful implantation of a balloon expandable stent (5 × 15 mm), final angiography showed excellent results, with a preserved intrarenal artery and retrograde filling of collateral vessels (*Figure 2B*). During the next 24 h, she had 6800 mL of diuresis. The following day, the patient's dyspnea dramatically disappeared with the improvement of hypertension (104/62 mmHg on three medications) and renal failure (creatinine 1.35 mg/

dL). Also, E/e' and TRPG decreased to 15.2 and 26 mmHg, respectively, suggesting improvement of left ventricular filling and pulmonary artery pressure (*Table 1*). Three days later, she lost 3 kg in weight, and pulmonary congestion and hypertension improved significantly with reduction of the number of antihypertensive agents (four agents to one agent) (*Figure 1B*). She was uneventfully discharged 4 days after the procedure. Brain natriuretic peptide decreased from 223 to 18 pg/mL and serum creatinine from 2.0 to 1.1 mg/dL even within 14 days after the procedure. Echocardiography at 1 month revealed further improvement of E/e' and TRPG with reduced size of left atrium and ventricle (*Table 1*). At a

**Figure 2** (A) Aortography showing ostial total occlusion (small arrow) and reconstituted distal segment (large arrows) in the right renal artery and a patent left renal artery. (B) Final angiography after stenting demonstrates excellent revascularisation with a preserved intrarenal artery and the retrograde filling of collateral vessels (arrows).



6 month follow-up, no recurrence of congestive heart failure was observed with the sustained benefits on echocardiographic parameters as well as blood pressure and renal function (Table 1).

## Discussion

Bilateral renal artery disease or solitary functioning kidney is thought likely to be behind recurrent congestive heart failure.<sup>1–4</sup> Indeed, the effect of intravascular volume reduction was first reported in patients with renal artery stenosis in the solitary functioning kidney or bilateral renal artery stenosis in the era of balloon angioplasty.<sup>5,6</sup> In the era of stenting, improvement of left ventricular filling following renal artery stenting was observed in patients with not only bilateral renal artery stenosis or solitary functioning kidney but also

unilateral renal artery stenosis<sup>7,8</sup>; Kawarada *et al.* reported that  $E/e'$  in patients with cardiac symptom decreased from  $17.1 \pm 6.5$  at baseline to  $12.7 \pm 4.1$  at a mean follow-up period of  $7 \pm 4$  months.<sup>8</sup> However, few data are available regarding the effects of revascularization of renal artery ‘total occlusion’ on cardiac function.

To date, some investigators resolutely reported the clinical improvement of pulmonary edema after renal artery stenting in the extent of bilateral renal artery disease with total occlusion<sup>9–13</sup> (Table 2). In the context of unilateral renal artery total occlusion, only hypertension or renal failure was the indication of renal artery stenting<sup>14,15</sup> (Table 2). In the present case, we found a dramatic reduction of  $E/e'$  and TRPG, or improvement of left ventricular filling and pulmonary hypertension following unilateral renal artery total occlusion stenting in a patient with recurrent congestive heart failure with preserved ejection fraction. These cardiac benefits might be because of the direct

**Table 2** Reported cases of stenting for renal artery total occlusion

Extent of renal artery disease	Author, year (reference)	Age	Sex	Lesion	Main reason for renal revascularization	Stented site
Bilateral	Rehan <i>et al.</i> , 2007 <sup>9</sup>	25	Female	Right occlusion, left occlusion	Pulmonary edema, hemodialysis	Unilateral (left)
	Wykrzykowska <i>et al.</i> , 2008 <sup>10</sup>	81	Female	Right 50% stenosis, left occlusion	Pulmonary edema, renal failure	Unilateral (left)
	Islam <i>et al.</i> , 2009 <sup>11</sup>	60	Female	Right occlusion, left occlusion	Pulmonary edema, hemodialysis	Bilateral
	Kanamori <i>et al.</i> , 2009 <sup>12</sup>	72	Female	Right occlusion, left 90% stenosis	Pulmonary edema, hemodialysis	Bilateral
	Nasser <i>et al.</i> , 2013 <sup>13</sup>	66	Male	Right occlusion, left >90% stenosis	Hemodialysis	Bilateral
Unilateral	Nagata <i>et al.</i> , 2010 <sup>14</sup>	57	Male	Left occlusion	Resistant hypertension, renal failure	Unilateral (left)
	Chandra <i>et al.</i> , 2011 <sup>15</sup>	57	Male	Right occlusion	Resistant hypertension	Unilateral (right)
	Present case	63	Female	Right occlusion	Recurrent congestive heart failure	Unilateral (right)

effect of improvement of severe hypertension after renal artery stenting. On the other hand, according to a previous study that demonstrated the control of heart failure after renal artery stenting,<sup>16</sup> one-third of patients did not present with poor blood pressure control before stenting. Another study suggested that renal artery stenosis patients with heart failure are exposed to elevated left ventricular filling pressure, regardless of blood pressure.<sup>8,17</sup> According to a review article,<sup>1</sup> multifactorial disorders, including volume retention, activation of the renin–angiotensin–aldosterone system, and stimulation of the sympathetic nervous system, are speculated to precipitate heart failure. Also, renal failure can be associated with the development of heart failure. Therefore, there is a possibility that heart failure may be controlled not only by the standalone effect of blood pressure reduction but also by the correction of complex pathophysiological disorders after renal artery stenting. An increase in serum aldosterone levels despite a downward trend in serum renin levels (as shown in *Table 1*) might be because of a reduction of antihypertensive agents and to excessive diuresis after renal artery stenting.

Of great interest, an asymmetric resistive index was observed; there was a normal resistive index (0.59) on the right side, where the renal artery was totally occluded, and a higher resistive index (0.87) on the left side, where the renal artery was patent. The reasons for this paradox are possibly because the blockage of the right renal artery can protect

the ipsilateral kidney by reducing intra-glomerular pressure and the left kidney with patent renal artery can be damaged by direct exposure to hypertension.

The normal value of resistive index, and the presence of collateral vessels and preserved intrarenal artery identified following revascularization implies that ipsilateral kidney is viable and ischemic, and potentially could develop pathophysiological disorders including renin–angiotensin–aldosterone system, sympathetic nervous system, and sodium and fluid retention.<sup>1</sup> Furthermore, given that contralateral kidney's resistive index was high (over 0.80), it can be speculated that contralateral kidney with parenchymal disease could fail to compensate unilateral renal artery total occlusion. Hence, renal artery total occlusion can be considered as a treatable cause to precipitate recurrent congestive heart failure even if the extent of renal artery disease is unilateral.

In this particular case, renal artery stenting could be translated to an improved patients' care in the clinical practice of heart failure management even if concomitant renal artery disease is unilateral total occlusion.

## Conflict of interest

All authors declare that they have no conflict of interest.

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