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Concomitant coronary and renal revascularization improves left ventricular hypertrophy more than coronary stenting alone in patients with ischemic heart and renal disease*

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Abstract: Percutaneous transluminal renal artery stenting (PTRAS) has been proved to have no more benefit than medication alone in treating atherosclerotic renal artery stenosis (ARAS). Whether PTRAS could improve left ventricular hypertrophy (LVH) and reduce adverse events when based on percutaneous coronary intervention (PCI) for patients with coronary artery disease (CAD) and ARAS is still unclear. A retrospective study was conducted, which explored the effect of concomitant PCI and PTRAS versus PCI alone for patients with CAD and ARAS complicated by heart failure with preserved ejection fraction (HFpEF). A total of 228 patients meeting inclusion criteria were divided into two groups: (1) the HFpEF-I group, with PCI and PTRAS; (2) the HFpEF-II group, with PCI alone. Both groups had a two-year follow-up. The left ventricular mass index (LVMI) and other clinical characteristics were compared between groups. During the follow-up period, a substantial decrease in systolic blood pressure (SBP) was observed in the HFpEF-I group, but not in the HFpEF-II group. There was marked decrease in LVMI in both groups, but the HFpEF-I group showed a greater decrease than the HFpEF-II group. Regression analysis demonstrated that PTRAS was significantly associated with LVMI reduction and fewer adverse events after adjusting for other factors. In HFpEF patients with both CAD and ARAS, concomitant PCI and PTRAS can improve LVH and decrease the incidence of adverse events more than PCI alone. This study highlights the beneficial effect of ARAS revascularization, as a new and more aggressive revascularization strategy for such high-risk patients.

Key words: Coronary artery disease (CAD), Heart failure with preserved ejection fraction (HFpEF), Percutaneous transluminal renal artery stenting (PTRAS), Renal artery stenosis
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1 Introduction

Atherosclerotic renal artery stenosis (ARAS) and coronary artery disease (CAD) are independently associated with major cardiovascular events and confer a high risk of death in patients with CAD (Park *et al.*, 2004; Kane *et al.*, 2010). Previous studies have shown that about 40% of patients with renovascular stenosis are complicated with cardiac diastolic dysfunction, such as heart failure with preserved ejection fraction (HFpEF) (Little and Brucks, 2005; Wright *et al.*, 2005; Asrar ul Haq *et al.*, 2014). The progression of HFpEF reportedly is accelerated by myocardial ischemia (Middleton *et al.*, 2001; Ronco and di Lullo, 2014). Revascularization of coronary arteries for CAD or renal arteries for ARAS can relieve left ventricular hypertrophy (LVH), which is considered a

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strong indicator for death and cardiovascular complications. LVH has been the ideal surrogate for HFpEF assessment. Although previous studies supported a neutral recommendation for percutaneous transluminal renal artery stenting (PTRAS) versus medication in the general RAS population, there is little evidence as to whether concomitant percutaneous coronary intervention (PCI) and PTRAS, compared with PCI and medication alone, can better improve LVH in patients with both CAD and ARAS. As renal vascular intervention was not mentioned in guidelines for treatment of heart failure (McMurray *et al.*, 2012), the purpose of the present study is to evaluate the effect of concomitant PCI and PTRAS versus that of PCI and medication alone for patients with both CAD and ARAS complicated by HFpEF, and to determine whether PTRAS could improve the long-term prognosis of these high-risk patients.

2 Materials and methods

2.1 Subjects

Patients with CAD and ARAS who had undergone concomitant PCI and/or PTRAS were retrospectively enrolled. ARAS was defined as a ≥50% luminal stenosis of a renoartery confirmed by catheter selective renoarterial angiography. Coronary angiography was carried out using the Judkins technique. CAD was defined as coronary artery lesions graded as a >40% narrowing of the luminal diameter in the left main coronary artery (LMCA) or >70% in the left anterior descending artery (LAD), left circumflex coronary artery (LCX), right coronary artery (RCA), or the main branches. Significant stenosis in any vessel of the LAD, LCX, or RCA was defined as single, double, or triple vessel disease. Stenosis in the LMCA was considered equal to double vessel disease. The indications of renoarterial angiography included: a history of severe hypertension before 30 or after 55 years of age; progressive deterioration of renal function after angiotensin converting enzyme inhibitor (ACEI) or angiotensin receptor blocker (ARB) initiation; flush pulmonary edema; unexplained angina or heart failure; refractory and severe hypertension, and coexisted with multiple coronary disease. HFpEF was defined as a left ventricular ejection fraction (LVEF) of >50% and N-terminal pro-brain natriuretic peptide (NT pro-BNP) of >220 pg/ml, as well as the typical symptoms (e.g. breathlessness, orthopnoea, paroxysmal nocturnal dyspnea, reduced exercise tolerance, ankle swelling, and fatigue) and signs (e.g. elevated jugular venous pressure, hepatojugular reflux, gallop rhythm, pulmonary crackles, and displaced apex beat) (McMurray *et al.*, 2012). Normal cardiac function was defined as an LVEF of >50% and NT pro-BNP of <220 pg/ml. Patients were divided into two groups according to interventional procedures: (1) the HFpEF-I group, undergoing concomitant PCI and PTRAS; and (2) the HFpEF-II group, undergoing PCI alone. Baseline characteristics of the patients including blood pressure (BP), cardiac and renal function, and serum lipid profile were compared with those measured after the two-year follow-up, within and between groups (Table 1). The exclusion criteria were as follows: patients with CAD or ARAS only, or patients with acute myocardial infarction (AMI), chronic renal failure with routine hemodialysis, functioning kidney with a serum creatinine (SCr) of >4 mg/dl, size of targeted kidney <8 cm, coronary or renal artery lesion anatomy not suitable for stenting, renal artery stenosis secondary to fibromuscular dysplasia (FMD), or vasculitis.

2.2 Data collection

Eligible patients were prescribed antiplatelet drugs (aspirin plus plavix or ticlopidine), statin, ACEI or ARB, and β-blockers, calcium channel blocker, nitrates or diuretics. BP was measured three times consecutively at resting status during an interval of at least 5 min (Chobanian *et al.*, 2003). Left ventricular systolic function was assessed by LVEF using modified Simpson's method (Folland *et al.*, 1979). The left ventricular mass index (LVMI) was used for evaluating cardiac diastolic function by normalizing left ventricular mass (LVM) over the body surface area (BSA) (Levy *et al.*, 1990). The formulas used were as follows: $LVM=1.04\times((LVEDd+IVSd+LVPWd)^3-LVEDd^3)-13.6$, where LVEDd is left ventricular end diastolic dimension, IVSd is interventncular septal thickness at diastole, and LVPWd is left ventricular posterior wall at diastole; LVMI=LVM/BSA.

The estimated glomerular filtration rate (eGFR, ml/min per 1.73 m^2) was estimated by the Cockroft-Gault formula incorporating age (A), sex, body weight (BW), and SCr concentration (c_{SCr}, mg/dl) as follows: $eGFR=((140-A)\times BW)/(c_{SCr}\times 72)\times(0.85$, if women). An eGFR of <60 ml/min per 1.73 m^2 was

defined as moderate to severe renal dysfunction (Patel *et al.*, 2002). Diagnostic criteria for contrast-induced nephropathy (CIN) were as follows: an SCr increase of 44.2 μmol/L or 25% of baseline value and an SCr of ≥110 μmol/L within 48 h of undergoing iodinated contrast injection.

Major adverse clinical events (MACEs) included all-cause mortality, AMI, unstable angina pectoris, congestive heart failure, resuscitation from cardiac arrest, and stroke.

2.3 Intervention

PTRA and PCI were performed using standard techniques by a femoral or radial artery approach. Heparin was infused to achieve an activated clotting time of at least 200 s during stent implantation. Coronary and renal artery stenosis was measured as the percentage of the decrease in luminal diameter. Coronary stenting was performed for significant narrowing lesions (>40% narrowing of the LMCA and >70% narrowing of other branches). Renoarterial stenting was performed if there was a ≥50% organic stenosis of the luminal diameter or a systolic pressure gradient of at least 20 mmHg across the stenotic lesion. Pre- or post-dilation of the target lesions was carried out if necessary. Technical success was defined as a <20% residual stenosis of the luminal diameter.

2.4 Statistical analysis

Statistical analyses were performed using SPSS 13.0 (SPSS Inc., Chicago, IL, USA). Continuous variables with a normal distribution are expressed as mean±standard deviation (SD). Means were compared by independent Student's *t*-tests. Categorical variables are presented as percentages and were analyzed by χ^2 test. The significance level of this study was set at two-tailed α=0.05. Multivariate logistic regression and linear analysis were used to estimate MACEs and LVMI variation during the two-year follow-up.

3 Results

3.1 Patients and baseline characteristics

A total of 1112 patients with concomitant coronary and renoarterial selective angiography between January 2003 and December 2011 in our hospital were screened; 228 patients with both ARAS and CAD were enrolled in the study, and 805 patients with only CAD and 32 patients with only ARAS were excluded. Thirty-one patients with AMI and 16 patients with FMD were also excluded. The percentages of single-, double-, and triple-vessel coronary artery lesions in the study population were 18.9%, 30.7%, and 50.4%, respectively (Table 1). The baseline LVMI value and systolic blood pressure (SBP) level were significantly higher in the HFpEF-I group than in the HFpEF-II group. The indications for undergoing PTRAS procedures included refractory hypertension (53.8%, 84/156), progressive renal dysfunction (16.0%, 25/156), flash pulmonary edema (7.1%, 11/156), and silent but severe renal artery stenosis (23.1%, 36/156). There were no significant differences in age, sex, history of hypertension and diabetes mellitus, the distribution of ARAS type, angiographic severity of coronary lesions, LVEF, or renal function between the HFpEF-I and the HFpEF-II groups (Table 1). The proportion of patients who received ACEI/ARB therapy was significantly lower in the HFpEF-II group than in the HFpEF-I group (Table 1).

3.2 Relationship between stent numbers, contrast volume, and CIN prevalence

The average accumulated volume of contrast for simultaneous coronary and renal stenting was (172.7±49.4) ml, and the CIN occurrence rate was 18.4% among all patients. The contrast volume and CIN occurrence and the average number of coronary stents were not significantly different between the HFpEF-I and HFpEF-II groups (Table 2).

3.3 Clinical outcomes after interventional procedures

Patients were followed up for at least two years (the mean duration of follow-up was (28.7±16.0) months) after the revascularization procedures. Within groups, BP, eGFR, LVEF, and LVMI were compared before and two years after the revascularization procedures were performed. There was a significant decrease in SBP. A greater decrease in SBP was observed in the HFpEF-I group than in the HFpEF-II group (Δ=(21.60±5.24) mmHg vs. Δ=(7.49±3.13) mmHg, P<0.001). The LVEF within and between groups did not change significantly in the post-intervention period (Fig. 1c). There was a significant decrease in LVMI in the HFpEF-I group (from (198.93±36.81) g/m^2 to (160.55± 36.39) g/m^2, P<0.001). Although there was

Table 1 Clinical characteristics of the study population at baseline

Characteristics	HFpEF-I (n=156)	HFpEF-II (n=72)	Total (n=228)	$F/Z/t/\chi^2$	P-value
Age (year)	71 (64, 76)	70 (67, 75)	69.24±8.69	2.347	0.309
Male, n (%)	103 (66.0%)	49 (68.1%)	152 (66.7%)	1.533	0.444
Coronary vessel disease, n (%)					
Single	25 (16.0%)	18 (25.0%)	43 (18.9%)	1.836	0.518
Double	54 (34.6%)	16 (22.2%)	70 (30.7%)	1.745	0.472
Triple	77 (49.4%)	38 (52.8%)	115 (50.4%)	2.168	0.843
RAS type, n (%)					
Unilateral	112 (71.8%)	56 (77.8%)	168 (73.7%)	6.536	0.759
Bilateral*	44 (28.2%)	16 (22.2%)	60 (26.3%)	5.712	0.637
Stenosis rate of renal artery (%)					
Left	84.63±11.99	76.92±8.99	81.36±11.02	1.554	0.215
Right	82.74±10.70	74.76±10.84	79.80±10.41	1.160	0.316
eGFR (ml/min per 1.73 m^2)	41.18±19.42	43.72±15.51	44.61±19.40	2.264	0.183
Chronic renal dysfunction (eGFR <60 ml/min per 1.73 m^2), n (%)	129 (82.7%)	63 (87.5%)	192 (84.2%)	8.391	0.114
Kidney size					
Left	10.88±3.07	10.69±2.58	10.21±2.57	7.292	0.873
Right	9.75±2.98	9.88±1.61	9.34±1.82	6.381	0.759
Stroke, n (%)	22 (14.1%)	11 (15.3%)	33 (14.5%)	6.770	0.235
Hypertension, n (%)	153 (98.1%)	65 (90.3%)	218 (95.6%)	5.064	0.053
SBP (mmHg)	156.34±27.90	143.09±24.89	151.43±27.37	4.710	0.000
DBP (mmHg)	79.77±13.78	80.88±13.12	80.89±13.38	2.984	0.124
Diabetes mellitus, n (%)	53 (34.0%)	31 (43.1%)	84 (36.8%)	2.305	0.330
LDL-C (mmol/L)	2.58 (2.16, 3.13)	2.31 (1.94, 2.89)	2.66±0.91	6.325	0.142
ACEI/ARB therapy, n (%)	127 (81.4%)	49 (68.1%)	176 (77.2%)	5.923	0.039
LVEF (%)	64.00 (58.00, 69.50)	61.00 (52.00, 65.00)	58.93±12.88	6.802	0.204
LVMI (g/m^2)	198.93±36.81	169.15±47.24	178.95±44.06	4.657	0.002
NT-pro BNP (pg/ml)	2865.00 (819.70, 4510.00)	1514.50 (510.50, 3020.50)	1450.73±423.49	158.320	0.105

Values are expressed as mean±SD, number of patients (percentage), or median (Q1, Q3). Chi-square for categorical variables. * Bilateral disease was defined as stenosis of 50% or more of the diameter of at least one artery supplying each kidney. RAS: renal artery stenosis; eGFR: estimated glomerular filtration rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; LDL-C: low density lipoprotein-C; ACEI: angiotensin converting enzyme inhibitor; ARB: angiotensin receptor blocker; LVEF: left ventricular ejection fraction; LVMI: left ventricular mass index; NT-pro BNP: N-terminal pro-brain natriuretic peptide; $F/Z/t/\chi^2$: statistical value for ANOVA (F)/Mann-Whiney U test (Z)/Student's t-test (t)/chi-square test (χ^2)

Table 2 Comparison of contrast volume, CIN prevalence, and average number of coronary stents in two groups

Group	Contrast volume (ml)	Coronary stent, n	CIN (%)
HFpEF-I (n=156)	174.6±54.5	1.4±1.5	19.20
HFpEF-II (n=72)	171.0±46.8	1.6±1.4	16.70
Total (n=228)	172.7±49.4	1.5±1.2	18.40
F/χ^2	0.192	0.279	0.808
P-value	0.826	0.757	0.668

CIN: contrast-induced nephropathy

also a significant decrease in LVMI in the HFpEF-II group (from (169.15±47.24) g/m^2 to (149.40±33.10) g/m^2, P<0.001), the HFpEF-I group showed a more significant improvement in LVMI than the HFpEF-II group (Δ=(32.80±12.62) g/m^2 vs. Δ=(18.52±8.17) g/m^2, P<0.001) (Fig. 1d). Linear analysis found that PTRAS and better BP control contributed significantly to LVMI improvement (Table 3). In both groups, there was no significant change in eGFR from before undergoing the revascularization procedures until two years after treatment (P>0.05) (Fig. 1e).

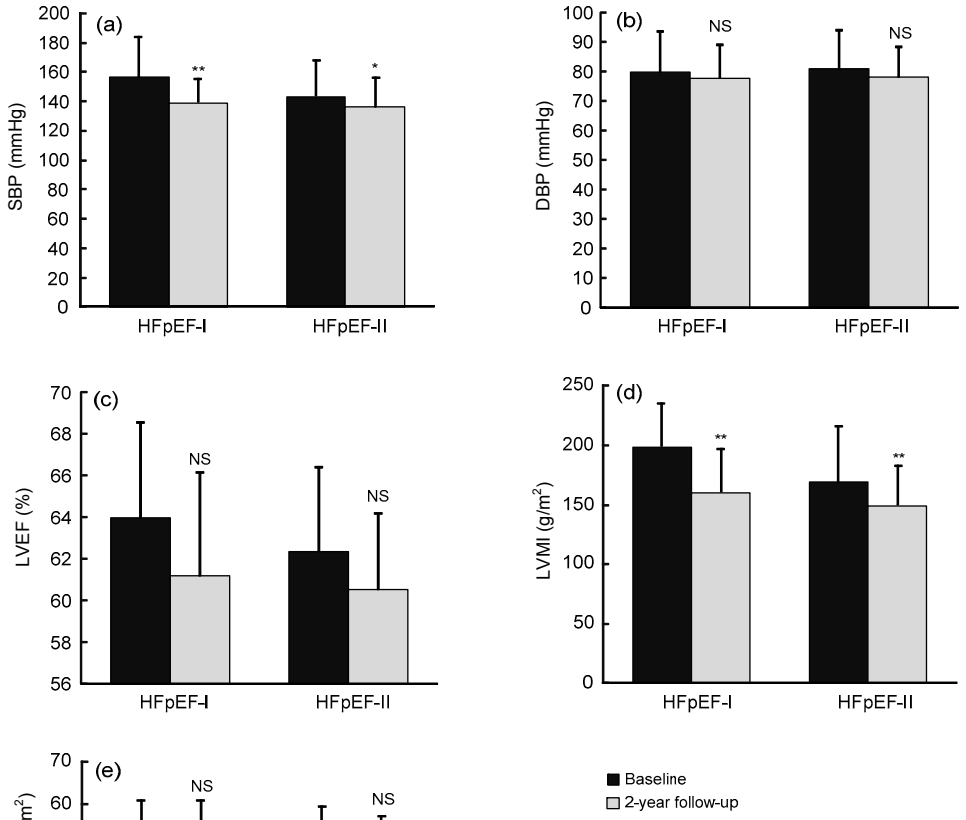

Fig. 1 Modifications of blood pressure, LVEF, LVMI, and eGFR in the HFpEF-I and HFpEF-II groups two years after coronary and renal artery stenting

* $P<0.05$, ** $P<0.01$ vs. baseline. SBP: systolic blood pressure; DBP: diastolic blood pressure; LVEF: left ventricular ejection fraction; LVMI: left ventricular mass index; eGFR: estimated glomerular filtration rate; NS: no significance

Table 3 Linear regression analysis for ΔLVMI

Impact factor	β	SE	P value
PTRAS	0.563	0.102	0.038
ACEI/ARB therapy	−1.091	10.627	0.918
ΔSBP	0.261	0.112	0.044
Baseline LVMI	0.452	8.277	0.745

PTRAS: percutaneous transluminal renal artery stenting; ACEI: angiotensin converting enzyme inhibitor; ARB: angiotensin receptor blocker; LVMI: left ventricular mass index; SBP: systolic blood pressure; ΔLVMI: "baseline LVMI value" minus "follow-up LVMI value"; ΔSBP: "baseline SBP value" minus "follow-up SBP value"

3.4 MACEs

After the revascularization procedures, 7 patients died during hospitalization and 29 died during the two-year follow-up period. Perioperative deaths were attributed to: cardiac causes (n=5), sepsis shock (n=1), and cerebral hemorrhage (n=1). Deaths during the two-year follow-up were attributed to cardiac causes (n=14), renal failure and multiple organ dysfunction syndrome (n=4), pneumonia or other causes of respiratory failure (n=4), stroke (n=2), cancer (n=3), and cataclasis (n=2). There was no significant difference in death rate between the HFpEF-I and HFpEF-II groups during follow-up, but the rate of MACEs in the HFpEF-I group was significantly lower than that in the HFpEF-II group (31.4% vs. 48.6%, P=0.004; Table 4). The HFpEF-II group had a significantly higher incidence of unstable angina pectoris (7.7% vs. 18.1%, P<0.001) and congestive heart failure (7.1% vs. 11.1%, P=0.019) than the HFpEF-I group during follow-up. Multivariable logistic regression showed that PTRAS was significantly associated with fewer MACEs (P=0.031; Table 5).

Table 4 Comparison of MACEs and hospitalization days in different groups

Group	Death	MACE	Hospitalization day
HFpEF-I (n=156)	19 (12.1%)[a]	49 (31.4%)	11 (6, 18)[b]
HFpEF-II (n=72)	10 (13.9%)	35 (48.6%)	8 (5, 16)
χ^2/H value	2.673	15.581	1.388
P value	0.276	0.004	0.500

Data are expressed as number of patients (percentage)[a] or median (Q1, Q3)[b]. MACE: major adverse clinical event

4 Discussion

Although prior trials indicated that PTRAS was no better than medication in the general renal artery stenting (RAS) population (Simon, 2010), only one study considered CAD and RAS together, and that study lacked details of the coronary intervention (Marcantoni *et al*., 2012). In our study, we found that PTRAS could further relieve LVH based on PCI, and reduce the incidence of MACEs. As ARAS has been recognized as a common and significant independent risk factor for survival in patients with cardiovascular disease (Hirsch *et al*., 2006), our results indicate that revascularization for ARAS in addition to PCI, is a potentially beneficial treatment for such high-risk patients with CAD and ARAS.

It has been reported that about one third of patients with CAD have coexisting ARAS, with or without HFpEF (Przewlocki *et al.*, 2008). Mechanisms by which coexistence of CAD and ARAS is associated with HFpEF are under investigation (Groban and Kitzman, 2010; Wang and Shi, 2014). The progressive increase in cardiac diastolic dysfunction leading to a worsening of CAD and ARAS is partly explained by factors such as increased afterload, renal hypertension resulting from ARAS, myocardial ischemia, overactivation of RAAS, and inflammation (Ding *et al*., 2014). Risks that increase with coexisting CAD and ARAS complicated by HFpEF include oxidative stress, de-arrangements in calcium-phosphate homeostasis, and conditions promoting coagulation, all of which share similar pathogenic factors associated with accelerated atherosclerosis and endothelial dysfunction (de Silva *et al.*, 2005; Liu *et al*., 2015). The Angioplasty and Stent for Renal Artery Lesions (ASTRAL) trial and the Cardiovascular Outcomes in Renal Atherosclerotic Lesions (CORAL) studies showed no significant difference in the ability of PTRAS or medicine alone to lower BP and CV (Wheatley *et al.*, 2009; Cooper *et al.*, 2014). The Renal Artery Stenosis in Coronary Artery Disease (RASCAD) study also drew a negative conclusion but lacked details of the coronary intervention, which may have affected the results from PTRAS (Marcantoni *et al.*, 2012). The intervention may have included controversial inclusion criteria or neglected the effects of coronary atherosclerosis or cardiorenal interaction. In the present study, we considered systemic atherosclerosis and the potential interaction between CAD and ARAS, and confirmed the diagnosis by selective artery angiography to exclude false positive or negative stenosis by echo or computed tomographic arteriography (CTA). Coronary stenosis was resolved to guarantee myocardium perfusion.

Table 5 Multivariable logistic regression analysis for overall MACEs

Impact factor	β	SE	Wald	HR (95% CI)	P value
Baseline eGFR (ml/min per 1.73 m^2)	0.004	0.005	0.467	1.004 (0.993–1.014)	0.494
Coronary complete revascularization	−0.080	0.173	0.215	0.923 (0.657–1.296)	0.643
PTRAS	−0.548	0.396	1.913	0.780 (0.596–0.964)	0.031
Baseline LVEF (%)	0.010	0.011	0.782	1.010 (0.988–1.033)	0.376
Diabetes mellitus	0.011	0.171	0.004	1.011 (0.723–1.413)	0.950
Hypertension	−0.357	0.384	0.867	0.699 (0.330–1.484)	0.352
Sex	0.269	0.197	1.873	1.309 (0.890–1.924)	0.171
Stroke	−0.002	0.224	0.000	0.998 (0.644–1.547)	0.992
Age (year)	0.004	0.011	0.140	1.004 (0.983–1.025)	0.708
LDL-C (mmol/L)	−0.088	0.970	0.819	0.916 (0.757–1.108)	0.365

MACE: major adverse clinical event; eGFR: estimated glomerular filtration rate; PTRAS: percutaneous transluminal renal artery stenting; LVEF: left ventricular ejection fraction; LDL-C: low density lipoprotein-C; β: regression coefficient; SE: standard error; Wald=$(\beta/\mathrm{SE})^2$; HR (95% CI): hazard ratio (95% confidence interval)

In our study, significant differences in the extent of the reduction in SBP between the HFpEF-I and HFpEF-II groups reflected an effective role of PTRAS in controlling SBP for HFpEF patients. The results could further explain why concomitant PCI and PTRAS led to a greater reduction in the LVMI than PCI alone. This effect may be due to the blocking of the vicious circle of the renin-angiotensin-aldosterone system (RAAS) as well as the inhibition of vasoconstriction by angiotensin and water-sodium retention. As the regression of LVMI may have been influenced by BP control and RAAS inhibitors, we performed a linear analysis of ΔLVMI (difference between baseline and follow-up LVMI values) and found that the effect of PTRAS on LVMI improvement remained significant even after adjusting for the above factors. LVMI has usually been used as an ideal indicator of cardiovascular events. PTRAS was found to be associated with a reduction in the LVMI, suggesting that concomitant revascularization of coronary and renal arteries was associated with a greater improvement in cardiac diastolic function than coronary revascularization alone. Hypothetical mechanisms by which coronary and renal revascularization might have increased cardioprotection in our study may include a superior control of hypertension, better perfusion to the myocardium, and relief of RAAS overactivity. Improved coronary circulation could contribute to the balance between myocardial energy requirements and oxygen supply, and relief of myocardial stiffness. A previous cohort study had demonstrated that diastolic function was significantly and independently associated with BP response and follow-up survival in patients undergoing open renal revascularization (Ghanami *et al.*, 2011). The extent of systolic BP improvement in our study was more significant in the HFpEF-I group than in the HFpEF-II group. Moreover, the prescription rate of ACEI/ARB was also much higher in the HFpEF-I group than in the HFpEF-II group (81.4% vs. 68.1%). This was probably due to revascularization of RAS giving physicians much more confidence to prescribe RAAS inhibitors, which were the core drugs for RAS treatment.

Similar to previous studies (Dean *et al.*, 1981), we found insignificant changes in eGFR during the follow-up period whether or not PTRAS was performed. However, whether the patient received PTRAS or not, the CIN rate in our study was not significantly different between groups, and was comparable with the rates reported in other studies (Chábová *et al.*, 2000; Su *et al.*, 2013). In our study, additional PTRAS was significantly associated with a lower incidence of overall MACEs. This may be explained by better control of BP, the decrease in the LVMI, and more prescription of ACEI/ARB. However, the comparison of HFpEF-I with HFpEF-II in death rate was negative, suggesting that PTRAS still could not improve the long-term survival of patients in this condition.

As a retrospective study, our study has inevitable limitations of sample size and matching of baseline data. A future intervention study will be needed to verify our findings. In conclusion, PTRAS could further relieve LVH based on PCI, and reduce MACEs to some extent. This highlights ARAS as a potential therapeutic target and provides evidence supporting a more aggressive strategy of RAS intervention in CAD and RAS with HFpEF, when PCI is performed.

Compliance with ethics guidelines

Hao-jian DONG, Cheng HUANG, De-mou LUO, Jing-guang YE, Jun-qing YANG, Guang LI, Jian-fang LUO, and Ying-ling ZHOU declare that they have no conflict of interest.

All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008 (5). Informed consent was obtained from all patients for being included in the study.

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中文概要

题　目：冠脉合并肾动脉狭窄同期血运重建术比单纯冠脉介入更能减轻左室肥厚

目　的：研究经皮肾动脉支架术（PTRAS）能否在冠脉介入（PCI）基础上进一步改善冠心病合并肾动脉狭窄患者的左室肥厚（LVH）及减少主要心血管不良事件的发生。

创新点：本研究对集中入选全身动脉粥样硬化这类高危患者（冠心病合并肾动脉狭窄（CAD & ARAS））临床诊治进行研究，有别于既往对单纯的肾动脉狭窄（RAS）人群的研究，且入选标准使用选择性动脉造影以排除其他诊断手段可能带来的假阴性或假阳性，并对冠脉狭窄进行血运重建以解决心肌灌注问题，再对 PTRAS 进行评价；有别于既往对 PTRAS 较为保守的建议，本研究发现对于 CAD & ARAS 患者，肾动脉狭窄的血运重建应该更加积极，RAS 的介入治疗可能是该类患者一个重要的治疗靶点。

方　法：将入选的 228 名 CAD & ARAS 患者，分为收缩功能保留性心衰-I（HFpEF-I）组（PCI & PTRAS）以及 HFpEF-II 组（单纯 PCI），术后随访至少两年。随访发现，两组的左室重量指数（LVMI）均较基线明显下降，且 HFpEF-I 组下降幅达大于 HFpEF-II 组（Δ=(32.80±12.62) g/m^2 vs. Δ=(18.52±8.17) g/m^2, P<0.001），回归分析发现 PTRAS 与 LVMI 的下降及不良事件的发生减少密切相关。

结　论：对于 CAD & ARAS 并 HFpEF 患者，同期行 PCI 及 PTRAS 可较单纯 PCI 进一步减轻 LVH 及降低心血管不良事件发生。对该类高危患者，可予以积极的肾动脉狭窄血运重建治疗。

关键词：经皮肾动脉支架术；冠心病；肾动脉狭窄