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ORIGINAL ARTICLE

Prevalence and predictors of renal artery stenosis in hypertensive patients undergoing coronary angiography

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Renal artery stenosis (RAS) is a major comorbid condition in patients with coronary artery disease (CAD). Although the reported prevalence of significant RAS among patients undergoing coronary angiography varies from 6.2 to 28% in Western countries, little information is available on the prevalence and predictors of RAS in Middle Eastern countries. From April 2007 to May 2008, 732 hypertensive patients with suspected CAD immediately after selective coronary angiography underwent abdominal aortography with or without selective renal angiography. Coronary angiography revealed stenosis of $\geqslant$50% in at least one of the main coronary vessels in 434 (59.3%) patients, including 100 (13.7%) cases with single-vessel CAD, 114 (15.6%) with two-vessel CAD and 220 (30.1%) with three-vessel CAD. Significant RAS was present in 87 patients (11.9%), severe ($\geqslant$75%) RAS in 35 patients (4.8%) and bilateral RAS in 37 patients (5.1%). Higher serum creatinine level, severity of CAD, history of coronary artery bypass graft surgery (CABG), congestive heart failure, and pulmonary edema together with atrial fibrillation were the most powerful predictors of significant RAS. In multivariate logistic regression analysis, three-vessel CAD (odds ratio 1.61, 95% confidence interval (1.36–1.91), $P<0.001$), history of CABG (odds ratio 4.40, 95% confidence interval (1.17–16.5), $P=0.028$) and serum creatinine level of >1.2 mg per 100 ml (odds ratio 2.12, 95% confidence interval (1.09–4.12), $P=0.026$) were the most powerful predictors of significant RAS. The prevalence of RAS in our patients was similar to that reported in the Western countries. The presence of multi-vessel CAD or a history of CABG along with a higher serum creatinine level in hypertensive patients undergoing coronary angiography was found to be a risk factor for RAS.
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INTRODUCTION

Hypertension and ischemic nephropathy are the most important consequences of renal artery stenosis (RAS).[1] Ischemic nephropathy progresses to end-stage renal disease (ESRD) in 6–17% of patients.[2–4] RAS is the most common potentially reversible disorder leading to renal replacement therapy.[5,6] Moreover, the presence of RAS has been independently associated with increased mortality, particularly in patients with coronary artery disease (CAD) and ESRD.[7–9] The reported prevalence of significant RAS ($\geqslant$50%) among patients undergoing coronary angiography ranges from 6.2% to as high as 28%.[2,8–18] A positive association between the presence and severity of RAS and CAD has been described,[11–13,16,18,19] and the presence of RAS has been associated with poor cardiovascular outcome.[9,20–22] There is growing evidence that a high percentage of significant atherosclerotic RAS is clinically indolent.[13–16] In recent years, efforts have been made to determine the prevalence of RAS and its predictors among patients with peripheral and coronary artery disease.[20,23–25] Attention has also been paid to the role of ethnicity in the prevalence of RAS.[26–28] This study aims to assess the prevalence of RAS in patients with hypertension and suspected CAD who are candidates for coronary angiography. This research further aims to clarify RAS predictors and to estimate prevalence based on the number of risk factors in patients.

METHODS

Study design

This is a multicenter cross-sectional descriptive-analytical study on the prevalence and predictors of RAS in hypertensive patients undergoing coronary angiography between April 2007 and May 2008 at three hospitals in Tabriz, Iran. The ethics committee of the Tabriz University of Medical Sciences approved the study, and written informed consent was obtained from each patient after thorough explanation of the study to patients and their families before enrollment.

Diagnostic criteria

Patients with angina or angina-like chest pain or their equivalents and with a positive result on exercise tolerance test and/or myocardial stress scan and/or dobutamine stress echocardiography were considered suspected CAD patients and approached for enrollment in the study. Hypertension was defined as blood pressure of $\geqslant$140/90 mm Hg recorded at least two times or current antihypertensive therapy. Diabetes was defined as fasting plasma glucose of

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>126 mg per 100 ml for at least two measurements or current antidiabetic therapy as defined by the World Health Organization. Hyperlipidemia was defined as total cholesterol of >200 mg per 100 ml or a history of elevated serum total cholesterol during the previous 6 months resulting in lipid-lowering agent prescription. Current smokers or those who had stopped smoking in the previous 3 years were considered smokers. Peripheral arterial disease was defined as showing intermittent claudication or a history of lower extremity revascularization or the presence of peripheral occlusive disease during the performance of an abdominal aortography procedure. Patients with a history of ischemic stroke were classified as a separate group in the analyses. Patients with compensated congestive heart failure were classified in a separate group from those with flash pulmonary edema. Echocardiography was used to estimate left ventricular ejection fraction with Simpson's method. More than moderate mitral regurgitation was considered significant.

Exclusion criteria

Patients with acute coronary syndromes or acute heart failure and those with high left ventricular filling pressure, defined as left ventricular end-diastolic pressure >25 mm HG, or patients on renal replacement therapy were excluded from the study. Patients in whom there was excessive use of a contrast agent (>200 cc) during the diagnostic procedure or subsequent coronary intervention were excluded from the study. Finally, we excluded patients with serum creatinine of >2 mg per 100 ml as a safety measure.

Catheterization procedure

Cardiac catheterization was performed using the femoral approach in 728 (99.3%) patients and through the radial artery in the remaining number of patients. In the first step, coronary angiography was performed and based on visual estimation by two observers. A diameter stenosis ⩾50% for the left main trunk and ⩾60% for other coronary branches was considered as significant stenosis. Patients underwent renal angiography before completion of cardiac catheterization. Renal angiography was performed using a powered injection of 35–40 ml of the contrast agent at the rate of 15–18 ml min^{-1} through a pigtail catheter positioned at the L1 vertebral body level in the straight anteroposterior projection. Selective renal angiography was performed if there was inadequate visualization of renal arteries or if a diagnosis of RAS was in doubt. Omnipaque was used as the contrast agent. To prevent contrast-induced kidney injury, patients were hydrated with 1 ml kg^{-1} h^{-1} saline for 12 h, and diabetic patients or those with serum creatinine of >1.2 mg per 100 ml were treated with *N*-acetylcysteine 600 mg twice a day for 2 days. Any degree of RAS was recorded and a diameter stenosis ⩾50% on the basis of visual estimation was considered significant RAS. Severe RAS was defined as RAS ⩾75%. Bilateral RAS was defined as bilateral RAS with at least one RAS ⩾50%.

Data regarding coronary risk factors, demographics, coronary and renal artery anatomy, relevant past medical history, electrocardiographic results and laboratory and echocardiographic findings were collected for subsequent analysis (Table 1).

Table 1 Baseline characteristics and angiographic findings of 87 patients with and 645 patients without significant RAS

	All patients (n=732)	*RAS ⩾50% (N=87)*	*Normal or RAS <50% (N=645)*	P-*value*
Mean age (year)	59.01±9.7	58.8±8.7	59.5±9.8	0.815
Males	316 (43.2%)	36 (41.4%)	280 (43.4%)	0.720
Diabetes Mellitus	208 (28.4%)	31 (35.6%)	177 (27.4%)	0.112
Hyperlipidemia	374 (51.1%)	45 (51.7%)	329 (51%)	0.900
Smoking	145 (19.8%)	13 (14.9%)	132 (20.5%)	0.225
Medical history				
MI	114 (15.6%)	10 (11.5%)	104 (16.2%)	0.225
CABG	15 (2.1%)	6 (6.9%)	9 (1.4%)	0.005
PTCA	29 (4%)	3 (3.4%)	26 (4%)	0.794
CVA	24 (3.3%)	2 (2.3%)	22 (3.4%)	0.582
CHF	20 (2.7%)	6 (6.9%)	14 (2.2%)	0.011
PAD	21 (2.9%)	6 (6.9%)	15 (2.3%)	0.030
Pulmonary edema	15 (2.1%)	6 (6.9%)	9 (1.4%)	0.005
Cr concentration (mg/dl)	1.0±0.37	1.03±0.34	0.97±0.37	0.040
Cr >1 mg per 100 ml	128 (17.5%)	24 (27.6%)	104 (16.1%)	0.020
Hematocrit (%)	40.5±3.4	39.9±4.5	40.6±3.9	0.149
SBP (mm Hg)	154.2±30.0	157.0±29.3	153.8±30.2	0.357
DBP (mm Hg)	85.5±13.2	85.6±14.3	85.5±13	0.922
Ejection fraction (%)	50.2±9.4	49.0±10.1	50.5±9.2	0.187
Coronary anatomy				0.007
No CAD	169 (23.1%)	6 (6.9%)	163(25.3%)	
Non significant CAD	129 (17.6%)	15 (17.2%)	114 (17.76%)	
One-vessel CAD	100 (13.7%)	13 (14.9%)	87 (13.5%)	
Two-vessel CAD	114 (15.6%)	22 (25.3%)	92 (14.3%)	
Three-vessel CAD	220 (30.1%)	31 (35.6%)	189 (29.3%)	
AF rhythm	12 (1.6%)	6 (6.9%)	6 (0.9%)	0.001
LBBB	22 (3.6%)	4 (4.6%)	18 (3.4%)	0.593
⩾Moderate MR	47 (7.6%)	6 (6.9%)	41 (7.7%)	0.892

Abbreviations: AF, atrial fibrillation; CABG, coronary artery bypass graft surgery; CAD, coronary artery disease; CHF, congestive heart failure; Cr, creatinine; CVA, cerebrovascular accident; DBP, diastolic blood pressure; LBBB, left bundle branch block; MI, myocardial infarction; MR, mitral regurgitation; PAD, peripheral arterial disease; PTCA, percutaneous transluminal coronary angioplasty; RAS, renal artery stenosis; SBP, systolic blood pressure.

RESULTS

Population

Over a period of 13 months between April 2007 and May 2008, 2500 patients underwent coronary angiography in the three hospitals. Of these patients, 732 (29.3%) met our criteria and were enrolled in the study. The average age of the patients was 59±9.7 years. Males accounted for 316 patients (43.2%). Of those with coronary risk factors, 208 patients (28.4% of the sample) were diabetic, 374 (51.1%) had hyperlipidemia and 145 patients (19.8%) were current smokers.

Clinical and laboratory variables

A history of myocardial infarction was present in 114 (15.6%) patients. There was a previous history of coronary artery bypass graft surgery (CABG) in 15 (2.1%) patients, percutaneous angioplasty in 29 (4%), cerebrovascular accident in 24 (3.3%) and symptomatic peripheral artery disease (PAD) in 21 (2.9%) patients. Compensated congestive heart failure (CHF) at the time of coronary angiography was seen in 20 (2.7%) patients and history of pulmonary edema without clinical CHF at the time of cardiac catheterization was positive in 15 (2.1%) patients. Among 732 patients 87 (11.9%) had significant RAS. The mean creatinine level was significantly higher in patients with significant RAS (1.03±0.34 vs 0.97±0.37, P=0.040). There was no significant difference in ejection fraction between the two groups with and without significant RAS (49.0±10.1 vs 50.5±9.2%, P=0.187). Atrial fibrillation (AF) (6.9 vs 0.9%, P=0.001) was more common in patients with severe RAS. Table 1 compares the prevalence of these parameters between groups with and without significant RAS. A similar analysis was carried out to determine the

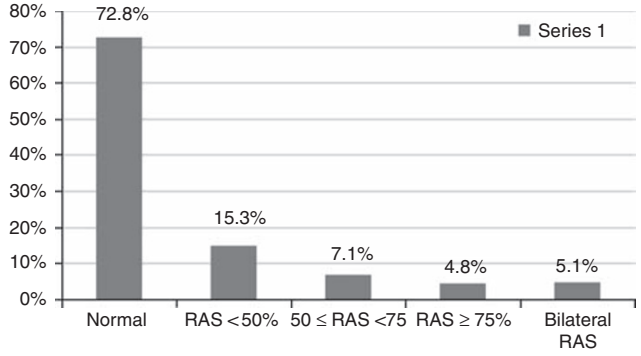

Figure 1 Prevalence of degrees of renal artery stenosis among 732 hypertensive patients undergoing coronary angiography.

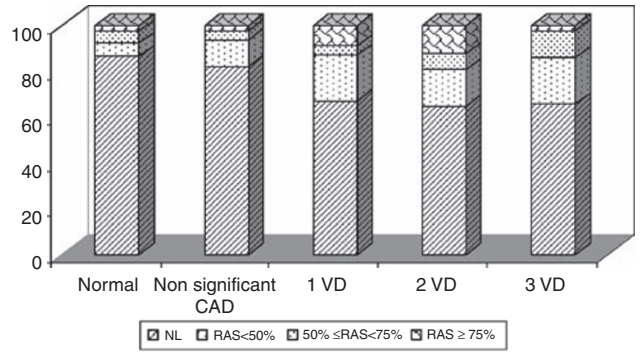

Figure 2 Prevalence of degrees of renal artery stenosis by severity of coronary artery disease.

predictors of any degree of RAS. RAS of any degree was found in 199 patients (27.1%) and angiographic appearance of the renal artery was normal in the remaining 533 patients (78.9%). History of CABG (8 (4.0%) in patients with RAS vs 7 (1.3%) in those without RAS, P=0.022), coronary anatomy (P=0.001), AF (9 (4.5%) in patients with RAS vs 3 (0.5%) in those without RAS, P=0.001) and more than moderate mitral regurgitation (6 (3.1%) in patients with RAS vs 41 (7.7%) in those without RAS, P=0.048) were strong predictors of patients with any degree of RAS compared with patients without RAS.

Results of coronary and renal angiography

Coronary angiography revealed stenosis of ⩾50% in at least one of the main coronary vessels in 434 (59.3%) patients, including 100 (13.7%) cases with single-vessel CAD, 114 (15.6%) with two-vessel CAD and 220 (30.1%) with three-vessel CAD. Atherosclerotic lesions with <50% stenosis were found in 129 (17.6%) patients and coronary angiography was normal in 169 (23.1%) patients.

Selective renal artery injection was necessary in 65 (8.9%) patients. Significant RAS, defined as stenosis of >50% diameter was present in 87 (11.9%) patients, of whom 52 (7.1% of total study population) had stenosis of 50–75%; 35 (4.8% of total study population) had severe (⩾75%) RAS, including two patients with complete occlusion of one renal artery. Bilateral RAS was seen in 37 (5.1%) patients (Figure 1). RAS prevalence in patients with CAD was 15.2% (66/434). Figure 2 shows the distribution of RAS among CAD groups. There is a significant association between the frequency of significant RAS and severity of CAD (P<0.007).

Table 2 Independent predictors of significant RAS based on multivariate analysis

	Odds ratio (95% CI)	P-*value*
Atrial fibrillation	0.83 (0.50–1.38)	0.482
Age	1.02 (0.99–1.04)	0.192
Coronary anatomy	1.61 (1.36–1.91)	<0.001
Hx of CABG	4.40 (1.17–16.5)	0.028
CHF	2.16 (0.89–9.66)	0.313
Creatinine concentration	2.12 (1.09–4.12)	0.026
⩾Moderate MR	0.38 (0.14–1.08)	0.065
PAD	2.57 (0.72–9.21)	0.147

Abbreviations: CABG, coronary artery bypass graft surgery; CHF, congestive heart failure; CI, confidence interval; MR, mitral regurgitation; PAD, peripheral arterial disease; RAS, renal artery stenosis.

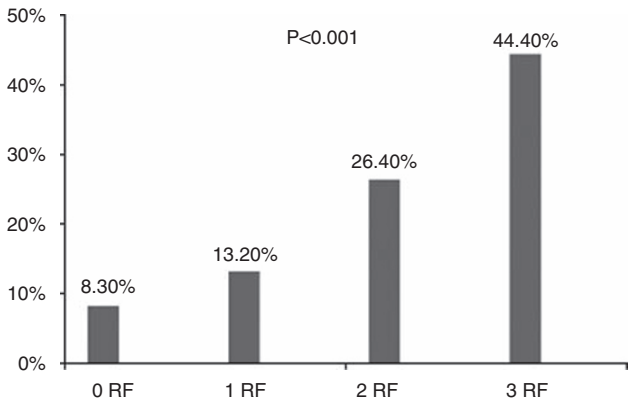

Figure 3 Prevalence of significant renal artery stenosis by the number of primary risk factors (3-VD, atrial fibrillation rhythm, creatinine >1 mg per 100 ml, Hx of coronary artery bypass graft surgery (CABG), peripheral artery disease (PAD), congestive heart failure (CHF)).

No patients showed cholesterol emboli, and no patient with renal artery or femoral artery traumatization required surgical intervention. No patient with contrast-induced renal insufficiency required dialysis.

Predictors of RAS

Factors more common in patients with significant RAS are mentioned in Table 1. Higher serum creatinine level, severe CAD, history of previous CABG, CHF, and pulmonary edema together with AF were the strongest predictors of significant RAS. Predictors of any degree of RAS were severe CAD, history of previous CABG, AF and more than moderate mitral regurgitation.

Table 2 shows the results of multivariate logistic regression analysis to identify factors more common in patients with significant RAS. Among these factors, three-vessel CAD (3-VD), history of CABG and high serum creatinine level were the most powerful predictors. There was a nonsignificant trend toward higher prevalence of significant RAS among patients with PAD and CHF.

Finally, we evaluated the likelihood of significant RAS on the basis of the count of six main predictors (3-VD, AF rhythm, creatinine >1 mg per 100ml, history of CABG, PAD and CHF) (Figure 3). The percentage of patients not showing any of these factors but with significant RAS was 8.3%, increasing to 44.4% in patients with three risk factors (P<0.001).

Table 3 Summary of studies screening for RAS during coronary angiography

	Number of cases	*Inclusion criteria*	*RAS ⩾50% (%)*	*RAS >70–75% (%)*	*Predictors of significant RAS (multivariate analysis)*
Rihal *et al.*[10]	297	Suspected CAD+HTN	19.2	7	Systolic BP, Hx of TIA or stroke, cancer
Ollivier *et al.*[11]	650	Documented CAD	14.5	NA	Male sex, severity of CAD, HTN, RI
Dizelinska *et al.*[12]	333	Documented CAD+HTN	12	7.5	Carotid IMT, severity of CAD, Cr concentration, BMI, no. of anti-HTN drugs
Park *et al.*[13]	1459	Suspected CAD	10.8	NA	CAS, PAD, RI, HTN, age >60 years, hypercholesterolemia, severity of CAD
Tumelero *et al.*[14]	1656	Suspected CAD	13.8	3.5	NA
Zhang *et al.*[15]	1200	Suspected CAD	9.7	NA	Older age, >10-year Hx of HTN, hypercholesterolemia, severity of CAD, proteinuria, Cr concentration
Przewłocki *et al.*[16]	1036	Suspected CAD	6.2	2.5	Aortic arch branch stenosis, severe CAD, female gender, Cr concentration and smoking
Aqel *et al.*[1]	90	Suspected CAD+HTN	28	16	Age >65 years, Cr >1 mg per 100 ml
Buller *et al.*[17]	837	Suspected CAD+one of the high-risk criteria[a]	14.3	7.3	Age, female gender, clinically apparent CAS, PAD, kidney dysfunction and systolic hypertension
Weber-Mzell *et al.*[18]	177	Suspected CAD	11	6.8	Low GFR and the extent of CAD
Harding *et al.*[8]	1235	Suspected CAD	15	7.4	Age, severity of CAD, CHF, female gender, PAD
Cohen *et al.*[19]	843	Suspected CAD	NA	1.8	Older age, Cr concentration, PAD, HTN, female sex, and severe CAD or previous CABG, number of cardiovascular drugs
Our study	732	Suspected CAD+HTN	11.9	4.8	Severe CAD, history of CABG, serum Cr level

Abbreviations: BMI, body mass index; BP, blood pressure; CABG, coronary artery bypass graft surgery; CAD, coronary artery disease; CAS, carotid artery stenosis; CHF, congestive heart failure; Cr, creatinine; GFR, glomerular filtration rate; HTN, hypertension; IMT, intimo medial thickness; NA, not available; PAD, peripheral arterial disease; RAS, renal artery stenosis; RI, renal insufficiency; TIA, transient ischemic attack.
[a]Severe hypertension or unexplained renal dysfunction or acute pulmonary edema with hypertension or severe atherosclerosis.
Results are presented as percentages.

DISCUSSION

In our study of hypertensive patients with suspected CAD, the estimated prevalence of RAS was 11.9%, and severe RAS was present in 4.8% of the cases. The previously reported prevalence of significant RAS among patients with suspected or documented CAD undergoing diagnostic coronary angiography varied from 6.2 to 28% (Table 3). Although this discrepancy may result from differences in the study population, including age, risk factors and social or environmental determinants, other contributing factors may be involved. For example, in the study of Aqel *et al.*[1] in which RAS prevalence was 28%, >80% of patients had severe angina or acute coronary syndrome, and the prevalence of significant CAD and PAD was 89 and 35.6%, respectively. Vetrovec *et al.*[29] reported an incidence of 23%, perhaps because their study included patients with serum creatinine levels of >2 mg per 100 ml. In one study, race/ethnicity (Hispanic vs African American) was an independent predictor of lower RAS prevalence in the second group.[27] Yamashita *et al.*[28] reported that significant RAS among Japanese patients with suspected CAD had a prevalence of 7%. Our study shows that the prevalence of significant RAS in Iranian patients is very similar to that reported in other countries (Table 3). Whether there is a racial/ethnic difference in the prevalence of RAS, as has been suggested for peripheral and coronary artery disease, warrants further study.[30]

In our study, 59.3% of the patients had significant CAD. The percentage increased to 75.9% among patients with significant RAS. The prevalence of RAS ranged from 14.9% in patients with 1-VD to 25.3% in those with 2-VD and 35.6% in those with three-vessel involvement. Among severe RAS patients with differentiated vessel involvement, the prevalence was 16.6, 20.1 and 38.2%, respectively. In the study of Park *et al.*,[13] the prevalence of significant RAS ranged from 19.6% in patients with 1-VD to 41.8% in those with 3-VD. Similar differences in groups have been found in other studies.[11–19] The higher rate of RAS among patients with a history of CABG in our study and that of Cohen *et al.*[19] reflects a higher prevalence among patients with multi-vessel CAD. This finding may certify that RAS is a manifestation of diffuse atherosclerosis and has a higher prevalence in those with CAD and PAD.

Although there was no significant difference between serum creatinine concentrations of patients with and without RAS, the group with significant RAS did show a higher serum creatinine concentration. We found that serum creatinine concentration was an important predictor of significant RAS in multivariate logistic regression analysis. Atherosclerotic RAS results in loss of renal function or parenchyma; this condition is known as ischemic nephropathy.[31] Furthermore, loss of renal parenchyma is directly related to increasing levels of serum creatinine.[32] Buller *et al.*[17] found that creatinine clearance showed an independent and continuous relationship with the frequency of RAS. In a recent prospective study, a lower rate of RAS progression was reported in elderly patients;[33] however, most studies point to a progressive worsening of RAS as a typical course in the natural history of RAS. In a study by Zierler *et al.*,[34] 42% of patients with RAS showed progression of RAS over the 2-year follow-up period, and 11% eventually progressed to occlusion. It has been estimated that the rates of lesion progression, renal atrophy and artery occlusion are approximately 20, 10 and 5% per year, respectively.[32,35] As RAS is the most common potentially reversible disorder leading to renal replacement therapy, and noting the grave natural history of untreated RAS noted above, early detection and treatment are ideal. Whether subsequent revascularization of renal arteries will affect the natural history of the disease remains controversial; in particular, the results of the recently reported ASTRAL (angioplasty and stent for renal artery lesions) trial offers new insights into this issue.[36]

There is growing evidence that the physiologic consequences of RAS may themselves drive atherosclerosis[1] and lead to higher cardiovascular mortality. Despite permitting the safe use of ACEIs, there is some evidence for the potential benefit of RAS correction in preventing crescendo angina, flash pulmonary edema and CHF.[37,38]

Owing to ethical concerns, mostly considering the possibility of contrast-induced nephropathy, our study included only hypertensive patients. Table 3 shows that, except for Aqel *et al*.'s [1] study, which included a high-risk group and reported an incidence of 28% for RAS in hypertensive patients with suspected CAD, the prevalence of RAS in hypertensive patients varied from 12% in the study of Dizelinska *et al*.[12] to 19.2% in the study of Rihal *et al*.;[10] the prevalence was 11.9% in our study. Studies that did not use hypertension as an inclusion criterion reported RAS incidence up to 15%. This small difference may be explained by noting that a significant number of patients with hypertension and RAS do not have renovascular hypertension. Our patients included those with renovascular hypertension and those with essential hypertension and incidentally found RAS. Among 199 patients with RAS, only 87 (11.9%) had significant RAS; severe RAS, more closely associated with renovascular hypertension, was present in only 35 patients (4.8%). A low incidence of renovascular hypertension, $<10\%$ in hypertensive patients, may explain the modest difference in the prevalence of RAS between studies that used hypertension as an inclusion criterion and those that did not. Furthermore, there are many patients with RAS who do not have hypertension; therefore, hypertension should not be an obligatory inclusion criterion in screening for RAS.

The prevalence of PAD in our study was significantly higher in the presence of significant RAS. This is in concordance with the findings of other studies; in most of these studies, the association between RAS and PAD is even stronger than with CAD.[8,13,19] The lower prevalence of RAS in our sample may reflect the definition that we used to determine PAD.

Among patients with CHF, a higher rate of significant RAS has been reported in the literature. In a study by MacDowall *et al*.,[39] magnetic resonance angiography was used to identify RAS in 53 elderly patients with chronic heart failure. Significant RAS was found in 34% of patients. de Silva *et al*. suggested that a combination of CHF with an estimated GFR $>60\,\mathrm{ml\,min^{-1}}$ may characterize a group of patients with high probability of significant RAS.[40] RAS is associated with an increase in morbidity and mortality in patients with chronic heart failure.[40] RAS may result in renal ischemia and greater disturbance in neuroendocrine, cytokine and hemopoietic factors that may also cause progressive heart failure. Another possibility is that RAS is a marker of more extensive vascular disease and vascular events leading to death.[40] A higher prevalence of significant RAS among patients with CHF or pulmonary edema in our study is also consistent with these findings.

Atrial fibrillation was more common among patients with significant RAS, but it was not an independent risk factor in multivariate logistic regression. This may be related to the higher prevalence and severity of CAD in patients with significant RAS. A higher prevalence of more than moderate mitral regurgitation in patients with severe RAS may also reflect a higher prevalence of severe multi-vessel CAD in this group.

In multivariate logistic regression, three-vessel CAD, history of CABG and higher serum creatinine concentration were independent predictors of RAS in our study. Using six important parameters (3-VD, AF rhythm, creatinine >1 mg per 100 ml, history of CABG, PAD and CHF) associated with significant RAS in our patients, we evaluated the probability of significant RAS in different counts of these risk factors. Figure 3 shows that the percentage of significant RAS in hypertensive patients with suspected CAD and without any of the noted risk factors is 8.3%, increasing to 44.4% in the presence of three of these risk factors.

A higher prevalence of RAS in patients with CAD and a poor prognosis independently associated with RAS suggest the importance of early diagnosis and screening. However, at a recent consensus conference, screening renal angiography was recommended only in the presence of predefined indications for revascularization.[41,42] Currently, the American College of Cardiology (ACC)/American Heart Association (AHA) guidelines for the management of patients with peripheral arterial disease state that the only Class I indications for atherosclerotic renal artery revascularization are recurrent, unexplained CHF or sudden, unexplained pulmonary edema in the presence of hemodynamically significant RAS.[42] Two ongoing randomized trials, STAR and CORAL (STAR trial, STent placement for Atherosclerotic ostial Renal artery stenosis; CORAL trial, Cardiovascular Outcomes in Renal Atherosclerotic Lesions), will help to clarify the benefits of renal artery revascularization for cardiovascular and renal end points.[36] The results of these trials may help identify more relevant indications for RAS screening.

Study limitations

We did not examine atherosclerotic changes in carotid arteries, aorta or peripheral arteries, and the definitions of PAD and cerebrovascular involvement in our study were primarily based on clinical presentation. We excluded patients with a creatinine concentration of >2 mg per 100 ml; therefore, evaluation of the presence of RAS in that group was not achieved. Finally, we excluded patients with high ventricular filling pressures, which may underestimate the prevalence of RAS in patients with CHF.

Conclusion

The prevalence of significant RAS in our patients is similar to that reported in Western countries and is about 11.9% in hypertensive patients referred for coronary angiography. We found that the prevalence of RAS increases with the severity of CAD. Flash pulmonary edema, congestive heart failure, AF and peripheral arterial disease are more common in patients with significant RAS. In multivariate logistic analysis, history of CABG and severity of CAD and serum creatinine concentration were independent predictors of RAS. The results of upcoming randomized trials will determine appropriate candidates for RAS screening and suggest who will benefit from revascularization.

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