Imaging and Diagnostic Testing

A simple prediction rule for significant renal artery stenosis in patients undergoing cardiac catheterization

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Background Renal artery stenosis (RAS) is a potentially reversible cause of hypertension and renal insufficiency and is associated with poor prognosis.

Methods We aimed to identify simple predictors of significant RAS among patients undergoing coronary angiography. Prospective data were collected on 843 consecutive patients who underwent cardiac catheterization and abdominal aortography. Stenoses ≥75% were considered significant. Multivariable logistic regression was used to assess the relationship between baseline characteristics and coronary anatomy with significant RAS. A simple risk score was derived from the model.

Results The prevalence of RAS ≥75% was 11.7%. Independent predictors of significant RAS were older age, higher creatinine levels, peripheral vascular disease, number of cardiovascular drugs, hypertension, female sex, and 3-vessel coronary artery disease or previous coronary artery bypass graft. The concordance index of the model was 0.802. These variables were used to develop a simple predictive score of significant RAS for patients undergoing cardiac catheterization. The prevalence of RAS increased stepwise with increasing score values: 0.6% for a score ≤5, 1.5% for 6 to 7, 6.1% for 8 to 9, 12.2% for 10 to 11, 18.7% for 12 to 14, 35.7% for 15 to 17, and 62.1% for ≥18 ($P < .001$). Approximately one third of the patients had a score ≥11, which yielded a sensitivity of 76% and a specificity of 71%.

Conclusions Renal artery stenosis is a relatively common finding among patients referred for coronary angiography. A simple score can predict the presence of significant RAS among patients referred for cardiac catheterization. (Am Heart J 2005;150:1204-11)

Renal artery stenosis (RAS) is a potentially correctable cause of hypertension, ischemic nephropathy, and loss of renal mass. It is estimated that 5% of patients with hypertension and up to 14% of patients initiating hemodialysis have RAS.[1-3] Several investigations have reported a prevalence of significant RAS ranging between 4% and 59%, depending on the population studied.[4-13]

It is well established that RAS is a progressive condition. Duplex scanning and angiography studies have shown rates of progression to severe stenosis or occlusion ranging from 11% to 46% at long-term follow-up.[6,9,14-17] A recent report showed that the presence and degree of RAS diagnosed during cardiac catheterization independently correlated with increased all-cause mortality at 4 years.[18] Furthermore, the subset of hemodialysis patients with atherosclerotic renal disease have a significantly shorter survival compared with other etiologies.[3,19]

Current endovascular therapies for RAS achieve high procedural success rates with excellent long-term patency rates, preservation of renal function, and more manageable hypertension.[20-24] However, controversy remains on which patient population should be screened for RAS and which screening method provides the best balance between accuracy, safety, and cost.[25] A recent review article advocates systematic RAS screening during diagnostic angiography for coronary artery or peripheral vascular disease (PVD) in asymptomatic patients with clinical findings suggestive of RAS.[26] However, there are no definitive data to validate this approach.

We aimed to examine the prevalence of RAS in patients referred for cardiac catheterization and to identify simple clinical variables that when combined in a predictive score could provide an estimation of

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Table I. Patient demographics

Variable	All patients (N = 843)	RAS <50% (n = 689)	RAS ≥50% (n = 154)	RAS ≥75% (n = 99)	P
Age (y)	64 (55-73)	63 (55-71)	70 (61-76)	71 (64-76)	<.0001
Female sex	252 (29.9)	199 (28.9)	53 (34.4)	33 (33.3)	.19
Diabetes	133 (15.8)	107 (15.5)	26 (16.9)	14 (14.1)	.77
Hypertension	549 (65.1)	428 (62.1)	121 (78.6)	81 (81.8)	.0001
Hypercholesterolemia	545 (64.7)	441 (64.0)	104 (67.5)	65 (65.7)	.45
Current smoking	181 (21.5)	146 (21.2)	35 (22.7)	22 (22.2)	.69
Angina	626 (74.3)	523 (75.9)	103 (66.9)	69 (69.7)	.028
CHF	94 (11.2)	80 (11.6)	14 (9.1)	6 (6.1)	.29
Previous MI	141 (16.7)	116 (16.8)	25 (16.2)	14 (14.1)	.79
PVD	96 (11.4)	64 (9.3)	32 (20.8)	25 (25.2)	<.0001
Renal insufficiency	40 (4.7)	14 (2.0)	26 (16.9)	20 (20.2)	<.0001
Previous PCI	93 (11.0)	76 (11.0)	17 (11.0)	12 (12.1)	.95
Previous CABG	49 (5.8)	34 (4.9)	15 (9.7)	9 (9.1)	.025

Values are expressed as median (range) or n (%). *CHF*, congestive heart failure; *MI*, myocardial infarction; *PCI*, percutaneous coronary intervention.

the probability of severe RAS of an individual in this population.

Methods

Patients

During the year 2000, screening abdominal aortography became part of the cardiac catheterization procedure at Hospital Italiano de Buenos Aires, when the additional contrast load was deemed safe. Between September 2000 and May 2002, a total of 1556 cardiac catheterizations were performed. The 843 (54%) patients who underwent abdominal aortography represent our study population.

Clinical and procedural data for patients undergoing abdominal aortography were prospectively collected and entered into a database specially designed for the present study. Hypertension was defined as systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg or current therapy with antihypertensive agents for previously diagnosed hypertension. Hypercholesterolemia was defined as cholesterol levels ≥200 mg/dL or current therapy with lipid-lowering agents. Peripheral vascular disease was defined as the presence of intermittent claudication, previous stroke, or history of peripheral vascular revascularization procedures. Angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers, β-blockers, calcium-channel antagonists, and diuretics were classified as cardiovascular drugs.

Exclusions and angiographic procedure

The decision not to perform abdominal aortography was left at the discretion of the physician performing the procedure. In brief, reasons for not performing abdominal aortography were increased left ventricular end diastolic pressure, excessive use of contrast dye during the diagnostic procedure, subsequent coronary intervention, and/or elderly patients with increased risk of volume overload. Arterial access in most cases was obtained via the femoral artery with 6F sheaths using the modified Seldinger technique. After coronary angiography and left ventriculography, the pigtail catheter was positioned in the abdominal aorta approximately 1 cm above the origin of the renal arteries. The images were obtained in the anterior-posterior view in a 9-in field. A volume of contrast not >40 mL was injected at a rate of 20 mL/s using a power injector in all cases. The contrast bolus was followed downstream to visualize the iliac arteries. Then, the angiography table was rapidly moved back to the level of the kidneys to assess the symmetry of the renal silhouettes during the nephrographic contrast phase. Images suggestive of significant RAS were further assessed with selective renal angiography.

Table II. Prevalence of RAS according to degree of stenosis

Prevalence		Cumulative prevalence	
Degree of stenosis (%)	n (%)	Degree of stenosis (%)	n (%)
0-24	627 (74.4)	≥0	843 (100)
25-49	62 (7.4)	≥25	216 (25.6)
50-74	55 (6.5)	≥50	154 (18.3)
75-94	45 (5.3)	≥75	99 (11.7)
95-99	37 (4.4)	≥95	54 (6.4)
100	17 (2.0)	100	17 (2.0)

All angiograms were independently reviewed by an experienced angiographer. Lesion severity in the coronary tree and the peripheral vasculature was assessed by visual estimation. Renal artery disease severity was graded in categories from 0% to 24%, 25% to 49%, 50% to 74%, 75% to 94%, 95% to 99%, and 100%. Renal artery stenoses ≥75% were considered significant. Coronary lesions with >70% stenosis were considered significant, except for those located in the left main trunk that were considered significant if the degree of stenosis was >50%. Aortic atheromatosis was subjectively categorized as mild, moderate, or severe based on the presence, appearance, luminal compromise, and complexity of atherosclerotic disease in the abdominal aorta.

Statistical analysis

Continuous variables are presented as medians with 25th and 75th percentiles. Categorical variables are expressed as frequencies with percentages. Bivariate associations with the

Table III. Pharmacologic therapies at the time of catheterization

Drug	All patients (N = 843)	RAS <50% (n = 689)	RAS ≥50% (n = 154)	RAS ≥75% (n = 99)	*P*
β-Blocker	629 (74.6)	511 (74.2)	118 (76.6)	78 (78.8)	.48
Calcium-channel blocker	135 (16.0)	92 (13.4)	43 (27.9)	30 (30.3)	<.0001
Diuretic	84 (10.0)	54 (7.8)	30 (19.5)	22 (22.2)	<.0001
ACE inhibitor	275 (32.6)	214 (31.1)	61 (39.6)	38 (38.4)	.046
Angiotensin receptor blocker	21 (2.5)	13 (1.9)	8 (5.2)	6 (6.1)	.014
Nitrates	150 (17.8)	113 (16.4)	37 (24)	26 (26.3)	.020
Statin	267 (31.7)	218 (31.6)	49 (31.8)	34 (34.3)	.90
Aspirin	754 (89.4)	617 (89.6)	137 (89.0)	90 (90.9)	.91
Oral antidiabetic agents	46 (5.5)	38 (5.5)	8 (5.2)	4 (4.0)	.81

Values are expressed as n (%).

Table IV. Angiographic characteristics

Variable	All patients (N = 843)	RAS <50% (n = 689)	RAS ≥50% (n = 154)	RAS ≥75% (n = 99)	*P*
Extent of CAD					
Insignificant CAD	336 (39.9)	283 (41.1)	53 (34.4)	33 (33.3)	.12
Left main disease	22 (2.6)	18 (2.6)	4 (2.6)	3 (3.0)	.97
1-Vessel CAD	125 (14.8)	104 (15.1)	21 (13.6)	13 (13.1)	.63
2-Vessel CAD	159 (18.9)	139 (20.2)	20 (13.0)	14 (14.1)	.045
3-Vessel CAD	201 (23.8)	145 (21.0)	56 (36.4)	36 (36.4)	.0001
LV dysfunction	222 (26.3)	174 (25.3)	48 (31.2)	29 (29.3)	.15
Abdominal aorta					<.0001
Normal	503 (59.7)	450 (65.3)	53 (34.4)	31 (31.3)	
Mild atheromatosis	137 (16.3)	104 (15.1)	33 (21.4)	22 (22.2)	
Moderate atheromatosis	103 (12.2)	77 (11.2)	26 (16.9)	14 (14.1)	
Severe atheromatosis	100 (11.9)	58 (8.4)	42 (27.3)	32 (32.3)	
Asymmetric renal size	22 (2.6)	2 (0.3)	20 (13.0)	18 (18.2)	<.0001

Values are expressed as n (%). *LV*, left ventricle.

degree of RAS (<50%, 50%-74%, and ≥75%) were analyzed by the Kendall τ correlation coefficient test for numeric or ordinal variables and the Wilcoxon rank sum test for binary categorical variables. All tests were 2-sided. Results were interpreted as statistically significant when $P < .05$.

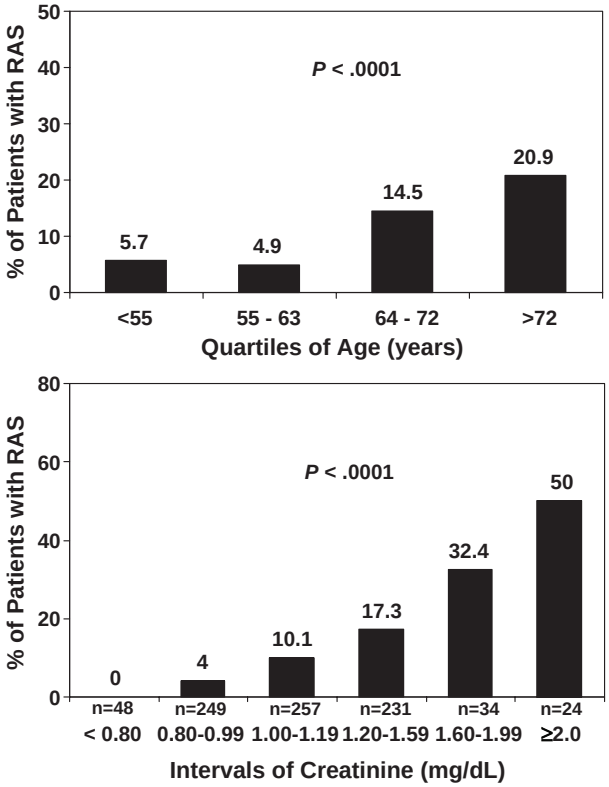
A multivariable logistic regression model was developed to predict the presence of RAS ≥75%. Those variables, including clinical and angiographic characteristics that achieved a significance level of <.20 in the bivariate analysis, were considered as candidates for testing in the multivariable logistic regression with stepwise forward selection. Variables were retained in the final model if they contributed significantly ($P < .05$). Abdominal aortography data were not considered in the model. The predictive performance of the multivariable model was evaluated using the concordance index (C-index), a statistic equivalent to the area under the receiver operating characteristic curve for dichotomous variables.[27] The goodness of fit of the regression model was evaluated using the Hosmer-Lemeshow goodness-of-fit test. The multivariable model was validated internally using the bootstrap algorithm with 400 replications. We developed a simple scoring system for the prediction of RAS based on the independent predictors of the multivariable model. With this purpose, we searched for integer scores for each predictor with similar ratios as the regression coefficients. The predictive score was computed for each patient, summing these integer scores for each predictor present. Statistical analysis was done with S-Plus software (version 6.1; Insightful, Seattle, WA).

Results

Study patients

Between September 2000 and May 2002, a total of 843 patients underwent abdominal aortography as part of their cardiac catheterization procedure at Hospital Italiano de Buenos Aires. The major indications for cardiac catheterization were stable angina in 37% of the cases, unstable angina in 33%, valvular heart disease in 7%, silent ischemia in 5%, and acute myocardial infarction in 3%, among others. Baseline characteristics are displayed in Table I.

Figure 1



Relationship of age and creatinine with the prevalence of significant RAS.

Table V. Independent predictors of significant RAS after coronary angiography

Variable	β Coefficient	χ^2	*P*	OR (95% CI)
Creatinine value (mg/dL)*	0.231	47.7	<.0001	1.26 (1.18-1.35)
Age (y)		17.3	.0002	
<64	0			1
64-72	0.777			2.17 (1.16-4.08)
≥73	1.243			3.47 (1.89-6.34)
PVD	1.095	12.1	.0005	2.99 (1.65-5.42)
No. of cardiovascular drugs		11.3	.010	
None	0			1
Treated with 1 drug	0.192			1.21 (0.43-3.41)
Treated with 2 drugs	0.808			2.24 (0.80-6.27)
Treated with ≥3 drugs	1.324			3.76 (1.15-12.3)
Hypertension	0.750	6.4	.011	2.12 (1.16-3.87)
Female sex	0.645	5.3	.022	1.91 (1.10-3.29)
3-Vessel CAD or previous CABG	0.552	4.2	.040	1.74 (1.03-2.92)

χ^2 and *P* value correspond to the likelihood ratio test. Intercept, −7.085; C-index, 0.822; Hosmer-Lemeshow test, *P* = .65. *OR*, Odds ratio.
*Per 0.10 mg/dL increments.

Prevalence of RAS

The prevalence of RAS in our study population was 11.7%, with 99 patients with ≥75% stenoses in one or both renal arteries. A total of 84 (9.9%) patients had unilateral disease and 15 (1.8%) had bilateral renal disease. Table II summarizes the cumulative prevalence of RAS according to angiographic severity.

Patients with RAS were older, more likely to have hypertension, PVD, angina, and previous coronary artery bypass graft (CABG) as depicted in Table I. Median (interquartile range) creatinine baseline level was 1.3 (1.0-1.5) for patients with RAS ≥75%, 1.2 (1.0-1.5) for patients with RAS ≥50%, and 1.0 (0.9-1.2) for patients with RAS <50% (*P* < .0001). Patients with RAS were more likely to be on calcium-channel blockers, diuretics, ACE inhibitors, angiotensin receptor blockers, and nitrates (Table III). The number of cardiovascular drugs was significantly greater in patients with RAS (*P* < .0001). Although the presence of coronary artery disease (CAD) did not differ between groups, patients with RAS were more likely to have more extensive CAD with higher prevalence of 3-vessel disease and less prevalence of 1- or 2-vessel disease than patients with RAS <50% (Table IV). There were no significant differences in the proportion of patients with left ventricular dysfunction in each group (*P* = .15).

Abdominal aortography showed more severe aortic atheromatosis in patients with RAS compared with patients with RAS <50%. In addition, the kidneys of patients with RAS were more likely to be asymmetric in size (Table IV).

Predictors of significant RAS and predictive score

We explored the relationship of the continuous variables age and creatinine with significant RAS to categorize these variables accordingly (Figure 1). There were no significant differences in the prevalence of RAS below the age of 64 years. For the logistic regression analysis, age was categorized in 3 groups using the median and the third quartile as cutoff points. The relation between the logit of the prevalence of significant RAS and serum creatinine was approximately linear up to a value of 2.2 mg/dL, becoming flat beyond this point. Only 17 patients had creatinine values >2.2 mg/dL.

Table VI. Nomogram to calculate the predictive score for significant RAS from baseline characteristics and coronary anatomy

Predictive factor	Factor score
Age (y)	
<64	0
64-72	2
≥73	4
Female sex	2
Hypertension	2
No. of cardiovascular drugs	
None	0
Treated with 1 drug	1
Treated with 2 drugs	3
Treated with ≥3 drugs	4
PVD	4
Creatinine (mg/dL)	
<0.80	0
0.80-0.99	1
1.00-1.19	3
1.20-1.39	4
1.40-1.59	6
1.60-1.79	7
1.80-1.99	9
2.00-2.19	10
≥2.20	12
3-Vessel CAD or previous CABG	2

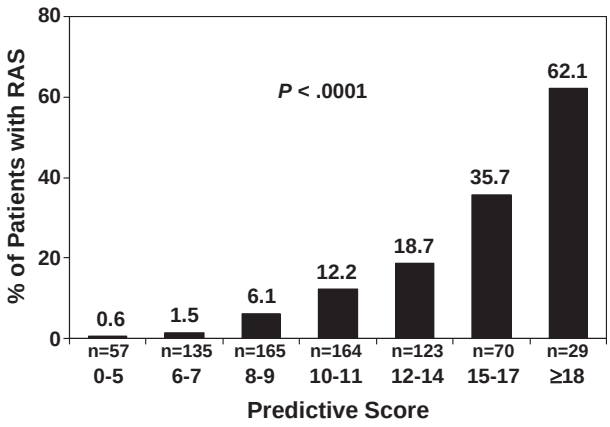
The results of the multivariable logistic regression analysis are shown in Table V. Independent predictors associated with significant RAS were baseline creatinine level, older age, PVD, number of cardiovascular drugs, hypertension, female sex, and 3-vessel CAD or previous CABG. The C-index of the multivariable model was 0.822, indicating an adequate ability for the prediction of RAS in this population referred for cardiac catheterization. After correction by the bootstrap technique, there was only minimal alteration in this result with a C-index of 0.802.

A simple score derived from the multivariable model was used to estimate the probability of RAS for individual patients (Table VI). As depicted in Figure 2, the probability of RAS increased significantly as the score increased ($P < .0001$).

Using the nomogram in Table VI, the predictive score for an individual patient can be calculated by summing the points for each predictor. Then, the probability of significant RAS can be determined using a formula accompanying Figure 3 or the simplified bar graph shown in Figure 2. Thus, a 56-year-old man with hypertension, treated with 2 cardiovascular drugs, a creatinine level of 1.40 mg/dL, 3-vessel disease, and no PVD has a predictive score of 13 (0 [for age], 0 [for sex], 2 [for hypertension], 3 [for cardiovascular drugs], 0 [for PVD], 6 [for creatinine], and 2 [for 3-vessel disease]) (Table VI). The probability of RAS in this patient can be estimated in 19.2% using the formula (Figure 3) or 18.7% using the bar graph (Figure 2).

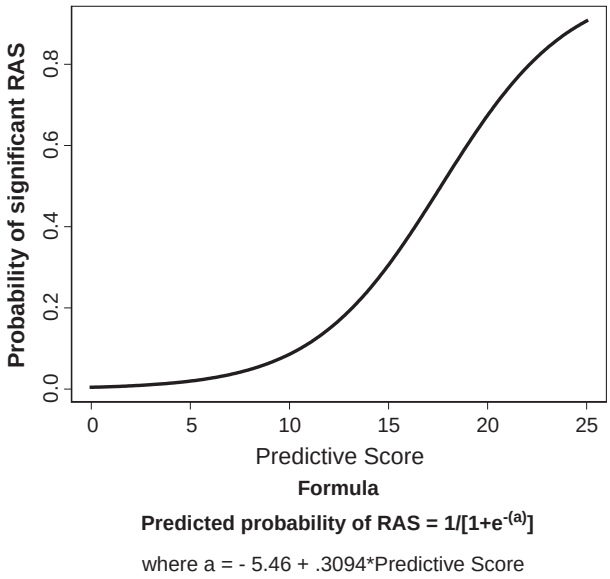
When applying the score in clinical practice, the probability of RAS according to the prediction rule can be used to select patients for angiography. Table VII displays sensitivity and specificity for different cutoff levels for the predicted probability of RAS. A score value

Figure 2



Prevalence of significant RAS according to the predictive score.

Figure 3



Formula

Predicted probability of RAS = $1/[1+e^{-(a)}]$

where a = - 5.46 + .3094*Predictive Score

Estimated probability of significant RAS as a function of the predictive score.

Table VII. Application of the score in clinical practice

Score	Patients with score (%)	Predicted probability of RAS (%)	Sensitivity (%)	Specificity (%)
≥5	86	≥2	100	16
≥6	81	≥3	99	21
≥7	70	≥4	98	33
≥8	65	≥5	97	39
≥9	54	≥6	94	52
≥10	46	≥9	87	60
≥11	34	≥11	76	71
≥12	26	≥15	67	79
≥13	20	≥19	60	85
≥14	16	≥24	49	88
≥15	12	≥31	43	92
≥16	8	≥37	29	95
≥17	6	≥45	26	97
≥18	3	≥53	18	99

11 corresponds to the point nearest the top left-hand corner in the receiver operating characteristic curve and maximizes the sum of the sensitivity and specificity. Using this cutoff value in clinical practice could reduce the proportion of patients receiving abdominal aortography to 34%.

Discussion

Our study results show a prevalence of unilateral RAS of 9.9% and bilateral RAS of 1.8% in a patient population referred for cardiac catheterization. In addition, we developed a simple predictive score for estimating the probability of significant RAS using clinical variables with independent predictive information.

The prevalence of RAS varies widely, depending on the population studied, the definition of significant stenosis, and the diagnostic modality. Autopsy studies have shown a prevalence of 4% in diabetic patients, 10% in patients with stroke, and 12% in patients with myocardial infarction.[5,8,10] Angiographic studies have shown that the prevalence of RAS is highest in patients with concomitant PVD (24%-59%),[28-34] intermediate in patients with abdominal aortic aneurysm (19%-24%),[35,36] and lower in patients with CAD (6%-19%).[7,9,11-13,37] In these studies, the cutoff point used for the definition of significant RAS varied between 50% and 75%. To better describe our study population, we opted to provide the characteristics of patients with RAS ≥50% and ≥75%. Because of the potential limitations of visual assessment to estimate stenosis severity, the model was built to predict RAS ≥75%. The presence of significant RAS in 11.7% of our study patients is within the previously reported range of prevalence in patients undergoing coronary angiography.

Our study showed that patients with significant RAS were older and more likely to be hypertensive, female, to have higher creatinine levels, to have significant atherosclerotic disease in other vascular territories, and to be treated with more cardiovascular agents compared with patients with insignificant RAS. A report from the Duke Databank found similar associations in 14 152 patients undergoing abdominal aortography with their cardiac catheterization.[9] The strong relationship of RAS and lower extremity vascular disease in our study was an expected finding considering the high prevalence of RAS in patients undergoing angiographic procedures for PVD.[28-34] We also found an independent association between 3-vessel CAD or previous CABG and RAS. This association has been previously described in a study with 110 patients where the presence of ≥2 coronary lesions had a sensitivity of 0.84 and a specificity of 0.71 to predict significant RAS.[38] Previous CABG, possibly a surrogate for extensive CAD, was associated with significant RAS in our study. This may allow the calculation of the predictive score in patients before coronary angiography. Although we found a bivariate relationship between RAS and degree of aortic atheromatosis and asymmetric renal size, we decided not to include these variables in the model because our purpose was to predict RAS before performing abdominal aortography.

To assess the probability of significant RAS, we developed a simple predictive score using multivariable statistical techniques. Previous studies have reported multivariable predictive models for RAS in different patient populations. Krijnen et al[39] developed a predictive score for renovascular hypertension based on 477 patients with drug-resistant hypertension or increased serum creatinine secondary to ACE inhibitors. Independent predictors were older age, female sex, serum creatinine levels, presence of an abdominal bruit, recent onset of hypertension, and a lower body mass index. The model had an excellent predictive ability with a C-index of 0.84 and a sensitivity and specificity that were comparable with that of renal scintigraphy.[39] Despite its simplicity and good predictive ability, this score probably applies to a minority of patients undergoing cardiac catheterization. Crowley et al[9] studied 14 152 patients undergoing routine abdominal aortography as part of their cardiac catheterization and developed a multivariable model that identified older age, CAD, creatinine level, PVD, female sex, cerebrovascular disease, and family history of CAD as independent predictors for the presence of incidental RAS during cardiac catheterization. However, in this study, the continuous predictor variables were not categorized and the intercept of the logistic regression function was not reported, complicating the applicability of this model in clinical practice.[40] Our multivariable model identified similar risk factors for incidental RAS, but we provided clinicians with a simple scoring system that includes clinical variables that are readily available in

cardiac catheterization laboratories or routine clinic visits. The C-index of 0.802 of our model indicated an adequate predictive ability to discriminate patients with and without significant RAS in clinical practice.[41] Furthermore, our scoring system was successful in allocating patients into low- and high-probability categories and may help select patients for abdominal aortography. A low score (≤5) correlated with a probability of significant RAS of 0.6%, whereas a high score (≥18) correlated with a probability of 62.1%.

The significance and therapeutic implications of RAS detected during vascular imaging for other reasons are still under debate. Proponents of systematic screening support their position on the progressive nature of atherosclerotic RAS. In fact, patients with severe RAS are at significant risk of renal atrophy and progression to end-stage renal disease.[42] Renal length may decrease by ≥1 cm in a third of kidneys with >60% RAS severity.[43] Moreover, the presence of RAS ≥75% in patients undergoing cardiac catheterization is independently associated with increased long-term mortality.[18] In addition, the diagnosis of renovascular hypertension is oftentimes made in retrospect because noninvasive screening tests have a limited value in predicting response after successful revascularization, particularly in the presence of renal insufficiency or bilateral disease.[44] As a consequence, patients with RAS may miss a therapeutic window for improving renal function or blood pressure if intervention is excessively delayed.[45]

On the other hand, the consequence of systematic RAS screening is the exposure of clinically asymptomatic patients to interventional procedures that are not without complications and may not provide a substantial clinical benefit. In high-risk subsets, the possibility of renal function deterioration can be as high as 26%.[46,47]

Renal artery stenosis is a marker of increased cardiovascular risk but its optimal management will remain uncertain until the results of adequately powered, randomized, controlled clinical trials assessing therapeutic strategies become available. In the meantime, management decisions will have to be made on case-by-case basis, with close surveillance of patients with significant RAS and a careful assessment of risks and benefits when deciding to perform renal revascularization.

Limitations

Because of the cross-sectional study design, we could not assess clinical impact of incidental RAS on long-term prognosis.

Our study was performed in a single tertiary center and was therefore subject to referral bias. Another source of selection bias was the fact that abdominal aortography was done in only half of the patients undergoing cardiac catheterization during the study period. Information on patients who did not have abdominal aortography was not collected. In our institution, abdominal aortography is done after diagnostic coronary angiography and was deferred in most patients undergoing immediate interventional procedures. This possibly accounted for an underrepresentation of patients with 1- or 2-vessel CAD.

Finally, renal artery anatomy was assessed with a single-view abdominal aortography that may be less sensitive than selective injection. It is possible that discrete ostial lesions could have been missed because of overlapping with the aortic silhouette or the mesenteric vessels.[6]

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