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Coronary anatomy predicts presence or absence of renal artery stenosis

A prospective study in patients undergoing cardiac catheterization for suspected coronary artery disease

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Aims This study aimed to determine the prevalence of renal artery stenosis (RAS) and associated risk factors in patients undergoing cardiac catheterization for suspected coronary artery disease (CAD).

Methods One hundred and seventy-seven consecutive patients (62 females) with a serum creatinine concentration $<2{\cdot}0$ mg . dl^{-1} were studied. Abdominal aortography followed cardiac catheterization to screen for RAS.

Results In 110 patients (62%) CAD and in 19 patients (11%) significant RAS (luminal narrowing of $\geq 50\%$) were detected, 12 of whom had high grade ($\geq 70\%$) RAS, and two subjects had significant RAS without CAD. Patients with RAS were older (67 ± 8 vs 61 ± 11 years, mean $\pm$ SD; $P=0{\cdot}004$), had higher systolic blood pressure (150 ± 15 vs 138 ± 20 mmHg; $P=0{\cdot}005$), a lower glomerular filtration rate (GFR; 61 ± 16 vs 80 ± 22 ml . min^{-1}, $P<0{\cdot}001$) and more often diabetes mellitus (69% vs 30%; $P=0{\cdot}004$). In multivariate analysis a low GFR and the extent of CAD were independent predictors of RAS. The presence of >2 significant coronary lesions predicted RAS (sensitivity 0·84, specificity 0·77, positive predictive value 0·30, negative predictive value 0·98).

Conclusion Screening for RAS in patients with >2 diseased coronary segments has a high diagnostic yield, which is even greater in the presence of a reduced GFR, diabetes mellitus, and elevated systolic blood pressure.
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Key Words: Renal artery stenosis, coronary artery disease, cardiac catheterization, ROC analysis. on this article

Introduction

Renal artery stenosis (RAS) is a possibly curable cause of arterial hypertension and of renal insufficiency. The prevalence of RAS in the general population is not known, but from 5 to 10%[1] in hypertensive subjects and 17% in patients with type 2 diabetes mellitus and coexistent hypertension[2]. The prevalence of RAS was found to be in the range of 22–44% in patients referred for evaluation of peripheral vascular disease or abdominal aortic disease[3], and may be even higher in patients with impaired renal function and coexistent atherosclerotic vascular disease in other arterial territories[4]. RAS may account for 7% of all end stage renal disease (ESRD), and up to 20% of cases in white patients 50 years of age or older[5].

The prevalence of RAS in patients undergoing routine cardiac catheterization has been estimated to be in the range of 11 to 23%[6,7]. In a large cohort of patients, age, severity of coronary artery disease (CAD), congestive heart failure, female gender and peripheral vascular disease (PVD) were risk factors associated with RAS[6]. The presence or absence of significant RAS may serve as a prognostic marker. Recently, in a cohort of 3987 patients undergoing abdominal aortography immediately following coronary angiography, the presence of significant RAS (in this study defined as $\geq 75\%$ narrowing in the luminal diameter) was a strong independent predictor of mortality[8].

The current study was undertaken to examine in detail the prevalence of RAS in patients with undergoing cardiac catheterization for suspected CAD, in terms of

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renal function, lipids, blood glucose and glycosylated haemoglobin, all factors possibly associated with RAS. A secondary goal was to define variables which may help decide in which group of patients abdominal aortography could be done in tandem with cardiac catheterization.

Methods

Study population

A total of 177 patients referred for cardiac catheterization because of suspected CAD were studied. Symptoms of coronary artery disease were recorded and classified in accordance with the Canadian Cardiovascular Society Classification (CCS)[9], symptoms of congestive heart failure were classified according to the New York Heart Association (NYHA)[10] criteria. Blood and urine samples were taken before the cardiac catheterization after an overnight fast. The reported blood pressure is the mean of at least three measurements taken the day before angiography. The study was approved by the ethics committee of the Medical Faculty, Karl Franzens University Graz, and written informed consent was obtained from each patient.

Angiography

Coronary angiography was performed following puncture of the femoral artery. For the purpose of this study a coronary artery stenosis $\geq 50\%$ was considered to be significant[11]. After left ventriculography, the pigtail catheter was withdrawn into the abdominal aorta and positioned a few centimetres cranial of the renal arteries. Aortography was performed in the anterior–posterior projection with Omnipaque 350 injected at a rate of 20 ml . s^{-1} to a total volume of 30 ml. The injection was recorded with a 35–mm cine film at 30 frames per second. An angiographically significant RAS was defined by a narrowing of the lumen $\geq 50\%$[6], a high grade RAS was defined as a narrowing $\geq 70\%$.

Laboratory studies

Laboratory parameters were determined by routine standard methods. The glomerular filtration rate (GFR) was estimated from the plasma creatinine concentration (PCr), gender, age, and body weight of the patient, with the Gault–Cockroft formula[12]: GFR = (140-age) * body weight/(PCr*72) in men; multiply*0·85 in women, with GFR in ml . min^{-1}, age in years, body weight in kilograms, and PCr in mg . dl^{-1}.

Statistical analysis

The study population was split into cohorts with and without RAS. Between-group continuous variables were compared by t-test, categorical variables were compared by χ^2 test. A binary logistic regression model with the presence or absence of RAS as the dependent variable was used to identify univariate predictors of RAS. Significant univariate predictors were used to build a multivariate logistic regression model. Multiple group comparisons were done by one-way ANOVA, the *P*-values were adjusted for multiple comparisons by the Bonferroni procedure. These computations were carried out with the Systat for Windows, version 8.0 (SPSS Inc. 1998).

In the context of this study the diagnosis of RAS in the presence of a pre-defined degree of CAD was defined as a true positive result (TP), true negative (TN), false positive (FP), and false negative (FN) results were defined accordingly. Definitions of the diagnostic indices were as follows: sensitivity = TP/(TP + FN); specificity = TN/(TN + FP); positive predictive value = TP/(TP + FP); negative predictive value = TN/(TN + FN). Receiver operation characteristic (ROC) analysis employed the rocfit.exe program in the case of categorical variables (number of coronary artery stenoses) and the labroc1.exe program in the case of continuous variables (age, glomerular filtration rate, systolic blood pressure). The programs were kindly provided by Charles Metz, University of Chicago. The Youden index is defined as (sensitivity + specificity − 1)[13]. The Youden index provides a means to estimate the optimal operating points of diagnostic procedures. The 95% confidence intervals (95% CI) of continuous variables, diagnostic indices (sensitivity, specificity, positive and negative predictive values), and ratios were computed with the Confidence Interval Analysis program, version 2.0[14]. Continuous data are presented as mean ± standard deviation (SD), ratios as percent. The 95% CI are given when appropriate.

Results

A total of 177 patients were studied, 62 females and 115 men. In 158 patients (89·3%) no significant RAS was found (non-RAS group). An insignificant RAS (30–49% luminal narrowing) of one renal artery was found in 25 (14·1%) patients, of whom it was unilateral in 15 and bilateral in 10. Significant unilateral RAS ($\geq 50\%$ luminal narrowing) was identified in 14 (7·9%) patients, and five (2·8%) had a significant bilateral RAS. These 19 patients (10·7%) made up the RAS group. The clinical characteristics of the RAS and the non-RAS groups are shown in Table 1. Twelve patients of the RAS group had one or more high grade ($\geq 70\%$) renal artery lesion (nine unilateral, three bilateral; Table 2).

Patients with significant RAS were older (61 ± 11 vs 67 ± 8 years; mean difference 6 years, 95% CI: 2–11 years, $P = 0{\cdot}004$). The area under the ROC curve of age was 0·67 (95% CI: 0·55–0·79). Diabetes mellitus was more frequent in the RAS group (69 vs 30%; $P = 0{\cdot}004$). Systolic blood pressure was higher in the RAS group

Table 1 Baseline characteristics of the study population

	No RAS		With RAS		*P* value
	Mean ± SD	95% CI	Mean ± SD	95% CI	
Number	158		19		
Age (years)	61 ± 11	59–62	67 ± 8	63–71	0·004
Gender	54 females		8 females		
Hypertension (%)	65	58–72	94	67–97	0·009
Cerebrovascular accident (%)	2·5	1–6	6·3	1–25	
Diabetes mellitus (%)	30	23–37	69	46–85	0·004
Hyperlipidaemia (%)	61	53–68	81	57–92	
Renal insufficiency (%)	4·4	2–9	6·2	1–25	
Smoking (%)	18	13–24	25	12–49	
Adiposity (%)	58	50–66	31	15–54	
Digitalis (%)	7	4–12	13	6–38	
Nitrates (%)	13	9–19	38	19–59	0·011
Diuretics (%)	11	7–17	31	15–54	0·025
ACEI (%)	35	28–43	31	15–54	
CCI (%)	12	8–18	19	7–37	
Statins (%)	61	53–68	63	41–81	
NYHA class	2·6 ± 0·8	2·5–2·7	3·0 ± 1·0	2·5–3·5	
CCS class	3·2 ± 0·9	3·0–3·3	3·3 ± 0·6	3·0–3·6	
Prior myocardial infarction (%)	24	18–31	44	23–64	
PTCA/stent (%)	16	11–22	19	9–43	
ACBG (%)	1·3	0–5	6·3	1–25	
Height (cm)	169 ± 9	168–170	169 ± 7	166–172	
Weight (kg)	79 ± 13	77–81	75 ± 9	71–79	
BMI (kg . m^2)	27·5 ± 3·7	27–28	26·3 ± 2·7	25–27·6	

ACEI=angiotensin converting enzyme inhibitor; CCI=calcium channel blocker; ACBG=aortocoronary bypass graft; BMI=body mass index.

(150 ± 15 vs 138 ± 20 mmHg; mean difference 12 mmHg, 95% CI: 4–21 mmHg; P=0·005; Table 3). The area under the ROC curve of systolic blood pressure was 0·65 (95% CI: 0·55–0·76). Pulse pressure was increased in the RAS group (72 ± 16·2 vs 55 ± 14·7 mmHg, mean difference 17 mmHg; 95% CI: 10–24 mmHg; P<0·001).

Serum concentrations of urea and creatinine were higher in the RAS group (50 ± 17 vs 41 ± 11 mg . dl^{-1}, mean difference 9 mg . dl^{-1}, 95% CI: 0·5–18 mg . dl^{-1}, P=0·04, and 1·3 ± 0·5 vs 1·1 ± 0·2 mg . dl^{-1}; mean difference 0·3 mg . dl^{-1}; 95% CI: 0·1–0·4 mg . dl^{-1}, P=0·049, respectively). Glomerular filtration rate was lower in the RAS group (61 ± 16 vs 80 ± 22 ml . min^{-1}, mean difference 19 ml . min^{-1}, 95% CI: 10–28 ml . min^{-1}, P<0·001). The area under the ROC curve of GFR was 0·75 (95% CI: 0·64–0·87).

Table 2 Renal artery stenosis according to side and extent of stenosis

	% stenosis left renal artery			
	0	1–49	50–69	≥70
% stenosis right renal artery				
0	133	3	4	**4**
1–49	12	10	0	**2**
50–69	1	1	1	**1**
≥70	**1**	**1**	**0**	**3**

In 12 patients (bold numbers) one or more high grade stenoses (≥70%) were detected.

In the non-RAS group diuretics and nitrates were used less frequently at baseline (Table 1). Significantly more stenotic coronary segments were diagnosed in the RAS group (Tables 1 and 3). The reduction of GFR paralleled the number of significant coronary artery lesions (Fig. 1, Table 4) so that in subjects without CAD the GFR was significantly higher as compared to subjects with four affected coronary segments.

Stenosis of the LAD, CX and RCA were more frequent in patients with significant RAS (Table 3; P<0·002). The frequency of RAS increased with the number of stenotic coronary segments (χ^2=30·6, P<0·001, 4 d.f.) and, in turn, more stenotic coronary arteries were observed in the RAS group (3·8 ± 1·2 vs 2·3 ± 1·3; P<0·001).

The two groups did not differ with respect to gender, CCS and NYHA scores, cardiovascular risk factors such as smoking, lipids, parameters of glucose metabolism and coagulation (Tables 1 and 3).

Receiver operating characteristics analysis was performed to analyse the predictive value of the coronary artery status on the presence or absence of significant renal artery stenoses (Table 5; Fig. 2). The Youden index was maximal in the presence of >2 stenotic coronary segments (Youden index: 0·61). Applying the presence of >2 significant coronary lesions as a test criterion for the presence or absence of RAS, a

Table 3 Comparison of the non-RAS and RAS group. Data are shown as mean ± SD, when appropriate

	No RAS	With RAS	*P*-value
Systolic BP (mmHg)	138 ± 19·9	150 ± 15·4	0·005
Diastolic BP (mmHg)	83 ± 17·8	79 ± 12·8	
Pulse pressure (mmHg)	55 ± 14·7	72 ± 16·2	<0·001
Heart rate ($l \cdot min^{-1}$)	68 ± 15	69 ± 16	
LAD stenosis (number of patients)	66	17	<0·001
RCA stenosis (number of patients)	65	16	0·002
CX stenosis (number of patients)	44	15	<0·001
LCA stenosis (number of patients)	24	5	
No CAD (number of patients)	65	2	
1 stenotic coronary segment (n)	31	1	
Two stenotic coronary segments (n)	25	0	
Three stenotic coronary segments (n)	30	12	
Four stenotic coronary segments (n)	7	4	<0·001
Coronary stenosis per patient (n)	2·3 ± 1·3	3·8 ± 1·2	<0·001
Ejection fraction (%)	66 ± 14	61 ± 12	
CK ($U \cdot l^{-1}$)	36 ± 54	37 ± 22	
Serum creatinine ($mg \cdot dl^{-1}$)	1·1 ± 0·2	1·3 ± 0·5	0·049
Creatinine clearance ($ml \cdot min^{-1}$)	80 ± 22	61 ± 16	<0·001
Serum urea ($mg \cdot dl^{-1}$)	41 ± 11	50 ± 17	0·040
Uric acid ($mg \cdot dl^{-1}$)	6·4 ± 1·5	7·1 ± 1·5	
Leukocytes ($k \cdot \mu l^{-1}$)	6·6 ± 1·6	7·1 ± 1·7	
Haemoglobin ($g \cdot dl^{-1}$)	14·1 ± 1·4	14·2 ± 1·1	
Platelets ($K \cdot \mu l^{-1}$)	227 ± 64	230 ± 57	
Prothrombine time (s)	102 ± 16	106 ± 23	
APTT (s)	35 ± 29	38 ± 26	
Fibrinogen ($mg \cdot dl^{-1}$)	379 ± 136	450 ± 154	
ATIII (%)	132 ± 15	98 ± 14	
D-dimer ($\mu g \cdot l^{-1}$)	3·8 ± 13·5	5 ± 2·8	
Serum Na^+ ($mmol \cdot l^{-1}$)	141 ± 2·5	142 ± 2·2	
Serum K^+ ($mmol \cdot l^{-1}$)	4·1 ± 0·5	4·1 ± 0·3	
Serum Cl^- ($mmol \cdot l^{-1}$)	103 ± 2·7	103 ± 2·9	
Total serum Ca^{++} ($mmol \cdot l^{-1}$)	2·3 ± 0·1	2·3 ± 0·1	
Serum protein ($g \cdot dl^{-1}$)	7·4 ± 0·6	7·7 ± 0·6	
Serum albumin ($g \cdot dl^{-1}$)	4·1 ± 0·3	4·2 ± 0·2	
CRP ($mg \cdot l^{-1}$)	9·8 ± 22	7·9 ± 15	
Urinary albumin ($mg \cdot l^{-1}$)	69 ± 177	26 ± 10	
Serum cholesterol ($mg \cdot dl^{-1}$)	202 ± 55	209 ± 35	
Serum triglyceride ($mg \cdot dl^{-1}$)	135 ± 72	117 ± 51	
HDL ($mg \cdot dl^{-1}$)	49 ± 15	59 ± 13	
LDL ($mg \cdot dl^{-1}$)	126 ± 44	135 ± 31	
ApoA1 ($mg \cdot dl^{-1}$)	250 ± 123	153 ± 29	
ApoB ($mg \cdot dl^{-1}$)	108 ± 34	115 ± 30	
ApoA1/B	1·2 ± 3·0	0·7 ± 0·4	
Lp(a) ($mg \cdot dl^{-1}$)	32 ± 38	36 ± 40	
Fasting blood glucose ($mg \cdot dl^{-1}$)	104 ± 28	106 ± 19	
HbA1c (%)	5·8 ± 1·2	6·2 ± 0·8	

sensitivity of 0·84 (95% CI: 0·62–0·95), specificity of 0·77 (95% CI: 0·69–0·83), predictive positive value of 0·30 (95% CI: 0·20–0·44), and a negative predictive value of 0·98 (95% CI: 0·93–0·99) for the diagnosis of RAS. The area under that respective ROC curve was 0·79 (95% CI 0·70–0·89; Fig. 2).

Using those variables which differed significantly between the RAS and non-RAS groups in the univariate analysis a binary multiple logistic regression model was developed (Table 6). In the multivariate analysis the extent of CAD and the glomerular filtration rate were significant predictors of renal artery stenosis.

In 71 patients one or more serum creatinine concentrations in the 72 h following cardiac catheterization were available. Serum creatinine concentrations before intervention and afterwards (peak values) did not differ (mean difference $-0{\cdot}01\ mg \cdot dl^{-1}$; 95% CI: −0·1–0·1; ns). In two patients a transient maximal rise in serum creatinine concentration of $0{\cdot}4\ mg \cdot dl^{-1}$ was observed.

Discussion

In the present study of 177 patients undergoing cardiac catheterization the prevalence of significant RAS was 11% (95% CI: 7–16%), which is in agreement with the previously reported RAS frequencies[6,15,16].

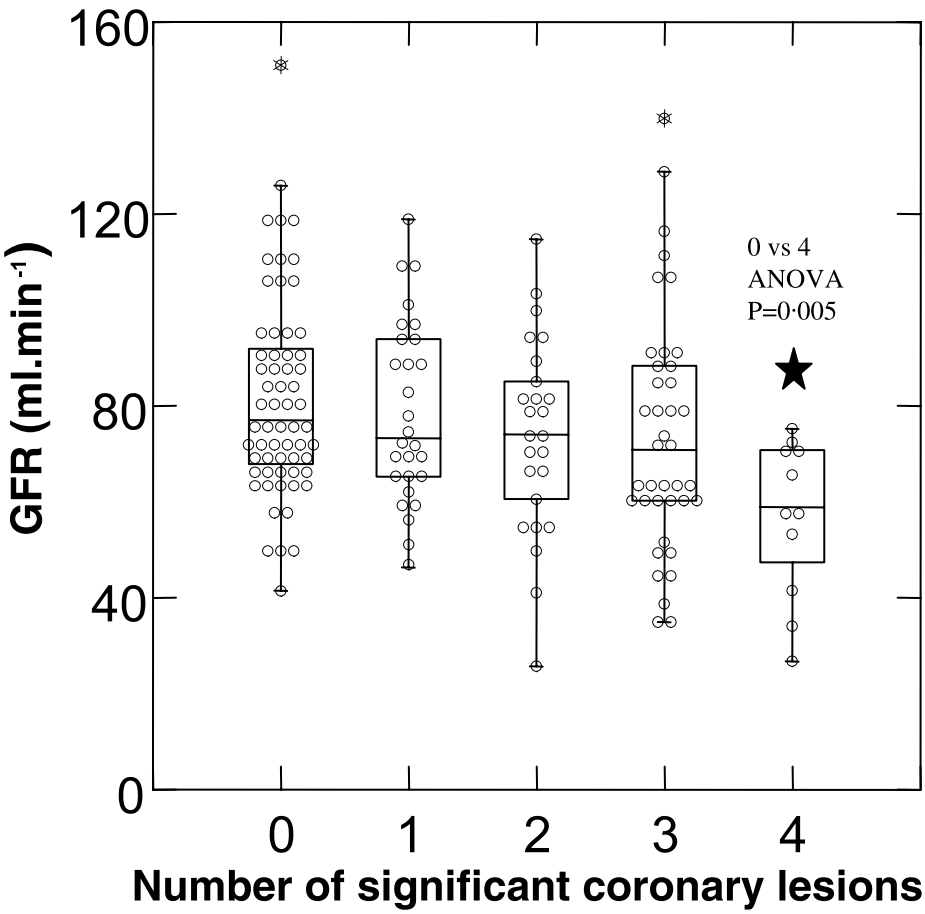

Figure 1 **The relationship between the number of significant stenosed coronary arteries and glomerular filtration rate (GFR) is shown. There is a negative relationship between the extent of CAD and the GFR.**

Table 4 The relation between number of significant coronary segment stenoses and glomerular filtration rate is shown. The groups were compared by ANOVA, the P-values adjusted for multiple comparisons (Bonferroni). In group 4 stenoses of LAD, RCA, CX, and LCA were observed

Number of significant coronary artery lesions	Glomerular filtration rate (ml . min $^{-1}$); mean ± SD (range); n	*P* value
0 (group 0)	83 ± 23 (41–169); n=67	0·005 vs group 4
1 (group 1)	79 ± 19 (46–119); n=32	0·06 vs group 4
2 (group 2)	75 ± 20 (26–115); n=25	
3 (group 3)	75 ± 25 (36–140); n=42	
4 (group 4)	57 ± 17 (27–76); n=11	

The major findings of this study were as follows: (a) Older age and a higher systolic blood pressure were the main clinical characteristics of patients with significant RAS. (b) Estimated GFR was significantly lower in the RAS group. (c) Despite the fact that these variables differ significantly between the RAS and non-RAS groups, it is evident from the areas under the respective ROC curve, that these variables provide limited help in deciding which patient should undergo abdominal aortography following left ventriculography. (d) The pre-test likelihood for the presence of significant RAS is particularly high in patients with more than two coronary lesions. These findings are in good agreement with data provided by one of the largest studies in the field[6]. In a patient with three or more significantly narrowed coronary segments the sensitivity and specificity of diagnosing one or two significant renal artery stenoses is 0·84 and 0·77, respectively. Therefore, it is sensible to rest the decision to perform abdominal aortography on the

Table 5 Sensitivity, specificity, predictive values and Youden index for the detection of RAS by abdominal aortography in relation the extent of coronary artery disease (95% CI of the estimates are given in brackets)

Coronary lesions number ≥50%	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Youden index
>0	0·90 (0·69 to 0·97)	0·41 (0·34 to 0·49)	0·16 (0·10 to 0·23)	0·97 (0·90 to 0·99)	0·31
>1	0·84 (0·62 to 0·95)	0·61 (0·53 to 0·68)	0·21 (0·13 to 0·31)	0·97 (0·92 to 0·99)	0·45
>2	0·84 (0·62 to 0·95)	0·77 (0·69 to 0·83)	0·30 (0·20 to 0·44)	0·98 (0·93 to 0·99)	0·61
>3	0·21 (0·09 to 0·43)	0·96 (0·91 to 0·98)	0·36 (0·15 to 0·65)	0·91 (0·86 to 0·94)	0·17

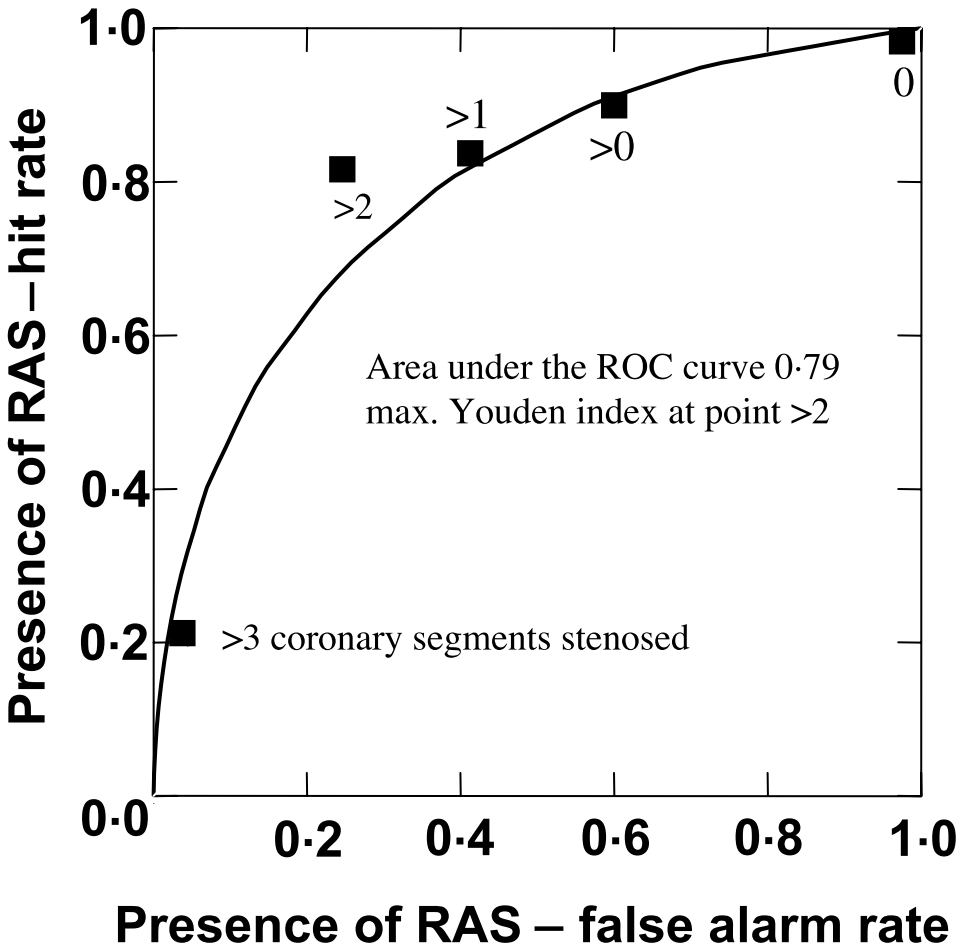

Figure 2 **Receiver operating characteristic (ROC) analysis of the diagnostic relationship between number of significant stenosed coronary arteries and the presence of renal artery stenosis (RAS) is shown. An optimal diagnostic threshold is the presence of >2 stenotic coronary artery segments (for details see text).**

number of significantly narrowed coronary arteries revealed by cardiac catheterization.

The possibility of acute renal failure due to contrast nephropathy has to be considered especially when a contrast dose >100 ml is used, and in diabetics with a creatinine clearance $<50\ ml\ .\ min^{-1}$ and $<40\ ml\ .\ min^{-1}$ in non-diabetics[17]. Adequate hydration prior to the contrast study is of paramount importance to prevent contrast nephropathy[18]. In none of the patients studied did acute renal failure develop.

The question arises, whether or not the diagnosis of significant RAS is of any value for patient management. In the majority of patients with arterial hypertension refractory to medical therapy or in those with renal insufficiency likely to be attributed to significant RAS, intervention (e.g. percutaneous balloon angioplasty (PTA) with or without stenting) will be offered to the patient[19]. The outcome of interventional therapy for renal artery stenosis is more favourable if the intrarenal resistance index obtained by Doppler ultrasonography

Table 6 Multivariate logistic regression of univariate significant predictors of RAS

	Regression coefficient	Odds ratio	95% CI	*P*-value
Extent of CAD	0·801	2·227	1·204 to 4·119	0·011
GFR	− 0·040	0·961	0·925 to 0·998	0·038
Systolic blood pressure	0·025	1·026	0·996 to 1·057	0·078
Age	− 0·010	0·990	0·917 to 1·069	0·802
Diabetes mellitus	0·091	1·095	0·570 to 2·071	0·781

from segmental arteries is <0·80[20]. Recurrent pulmonary flash oedema in the presence of significant RAS constitutes another widely accepted indication for an intervention[21].

The case is far from being clear in patients with significant RAS but without signs and symptoms attributable to the presence of RAS. There is general agreement that the presence of significant RAS in the absence of a clinical correlate should not result in an intervention, since the natural history of asymptomatic RAS may be favourable in many patients. We believe that this group of patients should be enrolled in a follow-up programme with a high clinical index of suspicion to possible consequences of RAS. The blood pressure and the evolution of renal function and renal size over time deserves special attention. Initiation and intensification of drugs blocking components of the renin–angiotensin system should be conducted in a particularly careful way to avoid renal side effects. It has been demonstrated recently, that statins may ameliorate the progression of renal failure[22]. Aggressive lipid management may favourably influence the progression of renal artery lesions, although data from controlled trials in this field are lacking[23]. A multidisciplinary approach, including nephrologists, radiologists and interventionalists, with careful evaluation of risks and benefits of intervention is particularly needed in patients with clinically silent RAS. Recent evidence suggests that renal arterial stenting may be most rewarding in those patients with ostial RAS whose renal function declined prior to the intervention[24]. Unfortunately unambiguous predictors of progressive renal artery disease in silent RAS are not available. The risk of clinically significant complications following percutaneous balloon angioplasty of renal arteries is in the range of 5–15%, including most prominently the consequences of atheroembolism[25]. Hopefully the advent of distal protection devices may reduce this particular risk of angioplasty in the future.

In conclusion, renal artery stenosis is prevalent in a significant proportion of patients undergoing cardiac catheterization for suspected coronary artery disease. The number of coronary arteries with stenotic lesions is the most powerful predictor of significant renal artery stenosis. The likelihood of having significant renal artery lesions is increased in subjects with systolic hypertension and reduced glomerular filtration rate. The other investigated biochemical parameters did not predict the presence or absence of RAS. Abdominal aortography should be considered particularly in subjects with >2 significantly narrowed coronary segments and also in the absence of hypertension and/or renal retention, and should be mandatory in markedly hypertensive patients or in those with impaired renal function.

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